2024 AAHA Fluid Therapy Guidelines for Dogs and Cats

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ABSTRACT

Fluids are drugs used in veterinary patients capable of producing beneficial therapeutic or inadvertent harmful effects within the body's intravascular, interstitial, and intracellular fluid spaces. The individualized design of a fluid therapy plan requires careful patient assessment and targeted selection of proper fluid types, administration routes, and rates, along with adjustments during therapy tailored specifically as per the individual patient's fluid requirement and therapeutic response. Personalized fluid prescriptions and vigilant patient monitoring help avoid patient morbidity from body fluid deficiencies, fluid excess, and electrolyte derangements and support better patient outcomes. These guidelines provide an overview of fluid dynamics within the fluid spaces of the body, describe various types of fluids and their uses, and outline recommendations for fluid administration for resuscitation, rehydration, and maintenance purposes. The guidelines also outline approaches to fluid therapy for anesthetized patients and reiterate the recommendations of reduced fluid rates in this population of patients. Additionally, the guidelines include practical fluid therapy strategies for patients with various common disorders. The goal of these guidelines is to help veterinary professionals safely and effectively prescribe and administer fluid therapy for canine and feline patients. (*J Am Anim Hosp Assoc* 2024; 60:131–163. DOI 10.5326/JAAHA-MS-7444)

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These guidelines were prepared by a task force of experts convened by the American Animal Hospital Association. This document is intended as a guideline only, not an AAHA standard of care. These guidelines and recommendations should not be construed as dictating an exclusive protocol, course of treatment, or procedure. Variations in practice may be warranted based on the needs of the individual patient, resources, and limitations unique to each individual practice setting. Evidence-guided support for specific recommendations has been cited whenever possible and appropriate. Other recommendations are based on practical clinical experience and a consensus of expert opinion. Further research is needed to document some of these recommendations. Drug approvals and labeling are current at the time of writing but may change over time. Because each case is different, veterinarians must base their decisions on the best available scientific evidence in conjunction with their own knowledge and experience.

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AAHA welcomes endorsement of these Guidelines by the American Association of Feline Practitioners (AAFP).

AKI (acute kidney injury); CPP (cryoprecipitate-poor plasma); CPR (cardiopulmonary resuscitation); CRI (continuous rate infusion); CRT (capillary refill time); CSA (canine-specific albumin); FWD (free water deficit); GFR (glomerular filtration rate); IO (intraosseous); KCl (potassium chloride); SC (subcutaneous); TBI (traumatic brain injury); TVI (total volume infused); VTBI (volume to be infused)

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Definitions

Dehydration—A condition in which the body loses more fluids than it takes in, resulting in an imbalance of water and electrolytes.

Euvolemia/euvolemic—Normal balance and distribution of total body water.

Fluid overload/fluid intolerance—A clinical spectrum that spans from hypervolemia to life-threatening edema and cavitary effusions. The guidelines task force has proposed that fluid intolerance may be the more appropriate term for this condition, as this term more accurately describes how the amount of fluid needed to overload a patient is dependent on their tolerance for a given amount of fluids. "Fluid intolerance" thus encompasses both iatrogenic overload and overload due to underlying comorbidities. However, given that "fluid overload" is still widely used and recognized within the veterinary medical profession, it will be the primary term used in these guidelines to refer to this condition.

Hypervolemia/hypervolemic—Increased fluid volume within the vascular space. Hypertension is not usually an indication of hypervolemia (except when renal disease is present).

Hypovolemia/hypovolemic—Decreased fluid volume within the vascular space.

Maintenance fluids—Crystalloid solutions formulated with electrolyte concentrations to meet a patient's daily requirements.

Replacement fluids—Crystalloid fluids intended to replace lost body fluids and electrolytes.

Total body water—The total amount of water contained in three mammalian body compartments: intracellular (67%) and extracellular (33%), which is further divided into interstitial (25%) and vascular (or intravascular) (8%) spaces.

Section 1: Introduction

Fluid therapy is a common aspect of veterinary patient care, required for a wide range of clinical conditions spanning from relatively mild cases (e.g., short-term inadequate voluntary intake from acute gastritis) to more moderate conditions (e.g., chronic kidney disease) to life-threatening emergencies (e.g., substantial volume loss and shock). When prescribed and administered correctly, fluid therapy can be one of the most beneficial treatments available in veterinary medicine. However, achieving desired therapeutic outcomes, rather than administering fluids with no effect or that worsen the problem, requires an understanding of the physiologic factors that influence body fluid movement and electrolyte balance. Refining this knowledge to create tailored therapeutic plans for each patient can be challenging, yet it proves to be a satisfying endeavor in enhancing patient recovery.

These guidelines offer an overview of the body's fluid dynamics and provide practical recommendations for selecting fluids, calculating administration rates, and choosing administration routes in dogs and cats for the purposes of resuscitation, rehydration, and maintenance. The guidelines also cover fluid therapy recommendations for anesthetized patients, patients with common conditions, and those with disorders presenting special fluid therapy challenges. Additionally, these guidelines detail patient monitoring parameters, highlight methods to prevent fluid overload, describe fluid delivery options, and address controversies and misconceptions in fluid therapy. Online resources include case examples and answers to frequently asked questions.

Section 2: General Fluid Therapy Principles

Top Three Takeaways

1. Fluids are drugs and must be prescribed accordingly to achieve the desired therapeutic goals promptly and minimize complications.
2. Each body fluid compartment—intracellular, interstitial, and intravascular—may require a different fluid prescription tailored to a patient's individual needs.
3. Arbitrarily assigning a fluid rate or dose can contribute to patient morbidity and mortality and lead to missed fluid therapy goals.

Overview

Fluid therapy involves administering prescribed fluids to restore a patient's body fluid homeostasis. As with any medication, the pharmacokinetics and pharmacodynamics of each fluid must be considered to attain therapeutic goals and minimize complications. However, fluid therapy alone cannot fix every abnormality, and using a standardized fluid rate for all patients can result in patient morbidity (see Box 1). To prescribe effective fluid therapy, veterinarians must have a basic understanding of the body's fluid compartments and how water is distributed among them.

Box 1: One Fluid Rate Does Not Fit All

A common misconception is that administering fluids at twice the maintenance rate will adequately treat most veterinary patients in need of fluid therapy. However, this approach may be inappropriate or inadequate in several scenarios, as shown in the following examples:

- **Interstitial dehydration.** Using a twice maintenance fluid administration rate would take approximately 33 hr to rehydrate a patient who has 5% interstitial dehydration, far longer than the recommended 12–24 hr.
- **Uremia due to acute kidney injury (AKI).** The primary goal in treating AKI is to ensure adequate renal perfusion and match the kidney's ability to handle fluid volumes. The fluid input rate for normovolemic patients should be determined based on fluid output (see Section 5, Fluid Therapy in Ill Patients).
- **Intoxications.** Administering an increased hourly fluid volume to force diuresis and urine output may not increase toxin excretion and can result in fluid overload. For example, although some animals with nonsteroidal anti-inflammatory drug (NSAID) toxicity may benefit from fluid therapy to treat dehydration or hypovolemia, high fluid rates (also known as forced diuresis) do not accelerate elimination of NSAIDs because most NSAIDs are highly protein-bound.

Fluid Distribution and Flow Among the Three Primary Fluid Compartments

The three main body compartments in mammals contain water. The intracellular and extracellular fluid compartments contain 67% and 33% total body water, respectively (Figure 1), and these sections are separated by cell membranes. The extracellular fluid compartment is further divided into interstitial (25% of the total body water, or 75% of the extracellular body water) and vascular (8% of the total body water, or 25% of the extracellular body water) compartments, and capillary walls separate these spaces (Figure 1).

Fluid intake by any route can affect the body fluid compartments. Administered fluids move between compartments based on:

- Tonicity of the fluid
- Tonicity of the patient's extracellular compartment
- Size of any macromolecules in the administered fluid

Sodium is the most abundant cation in the extracellular fluid compartment and is the most important molecule that supports extracellular tonicity. A fluid given IV that contains a sodium concentration similar to that of the extracellular fluid compartment will redistribute within 45 min based on a compartment's percent total body water; i.e., in a normal animal, 25% of the administered fluid will remain in the intravascular space and 75% will move into the interstitial space. Fluid movement across the endothelial membrane depends on the contents of the administered fluid and the condition of the patient's capillary membrane. The modified Starling hypothesis describes how fluid moves across the capillary membrane (Figure 2). Hydrostatic pressure, colloid osmotic pressure, and vascular permeability influence fluid movement.[1,2]

When increased capillary membrane permeability, elevated intravascular hydrostatic pressure, or decreased plasma colloid osmotic pressure occurs, more isotonic fluid can pass into the interstitium or body cavity and cause tissue edema, effusion, or both.

Administering a hypertonic fluid IV will cause water to move from the interstitial and intracellular spaces into the intravascular space. This can be desirable for rapid intravascular volume resuscitation. However, for this strategy to succeed, the interstitial and intracellular spaces must already be adequately hydrated. When administered IV, a hypotonic fluid will cause water to move from the extracellular space into the intracellular space, which is a suitable approach when treating a solute-free water deficit.

Goal-Directed Fluid Therapy

Goal-directed fluid therapy requires creating a fluid prescription that replaces fluid deficits that may exist in each fluid compartment, using the following steps:

1. Recognize which fluid compartment deficit(s) exists.
2. Understand which fluid type and administration route will best replace each deficit.
3. Calculate the fluid dose and administration rate.
4. Monitor patients for response to therapy and signs of complications.[3–5]

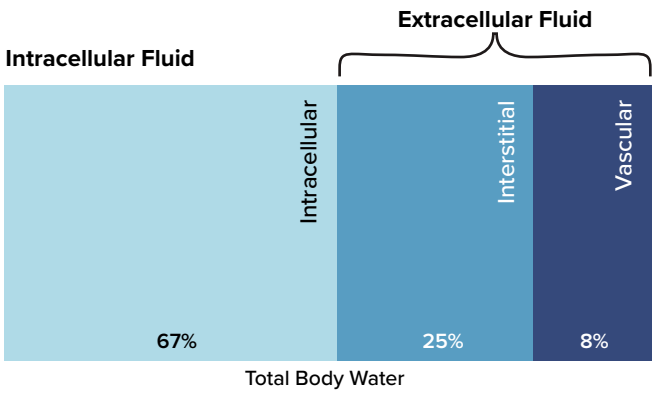

FIGURE 1

Normal distribution of body water

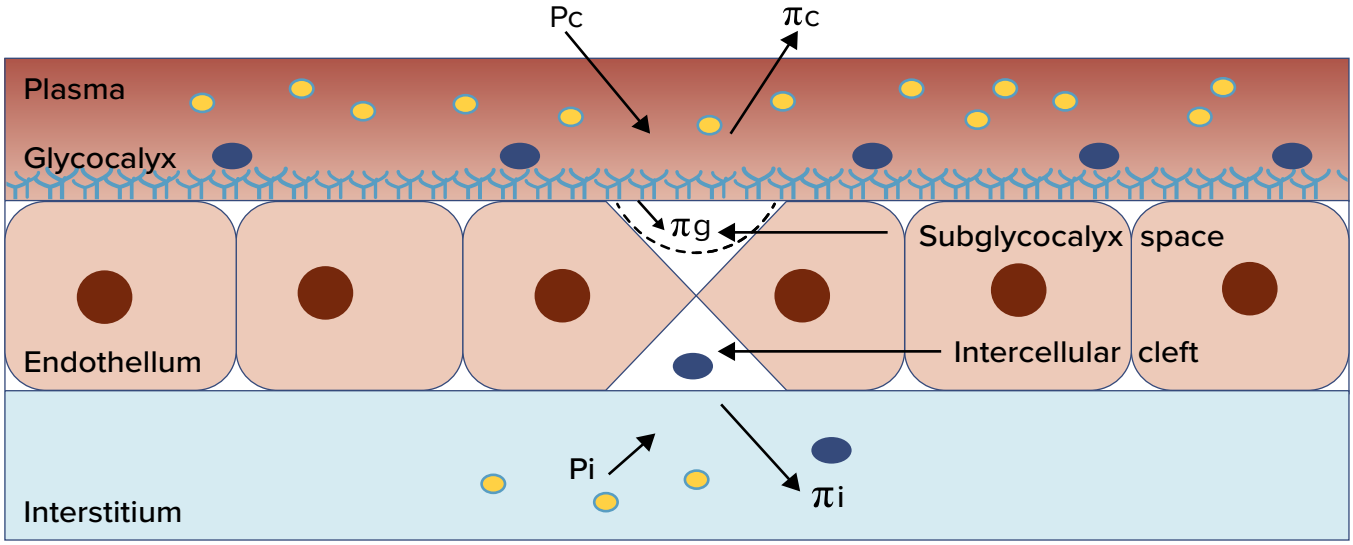

FIGURE 2

Modified Starling hypothesis[a]

Modified Starting hypothesis of fluid flux across the capillary membrane. Filtration force = ([P_c - P_i] - σ [π_p - π_g]). Pc, Capillary hydrostatic pressure; P_i, Interstitial hydrostatic pressure; π_p, plasma oncotic pressure; π_i, interstitial oncotic pressure; π_g, glycocalyx oncotic pressure

[a] Reprinted from Silverstein DC and Hopper K, eds., *Small Animal Critical Care Medicine*, 3rd ed., Waddell L., Colloid osmotic pressure and osmolality, p. 1055, Elsevier (2022), with permission from Elsevier.

Assessing Patients: General Principles Before and During Fluid Therapy

The extracellular fluid compartment (i.e., the vascular and interstitial spaces) must have adequate volume before intracellular fluid compartment deficits can be addressed. Therefore, assess and address alterations in volume homeostasis in the following order:

1. Assess the intravascular fluid space by evaluating:
   - Patient history
   - Perfusion parameters (mentation, heart rate, capillary refill time, mucous membrane color, extremity temperature, skin turgor, and pulse quality)
   - Monitored parameters (blood pressure, electrocardiogram findings)
   - Laboratory test results (Table 1)
   - Diagnostic imaging findings (Tables 1, 2)
2. Assess the interstitial space by evaluating:
   - Patient hydration parameters (Tables 3–5)
3. Assess the intracellular space by evaluating:
   - Patient sodium concentration
   - Solute-free water deficit (FWD) (Box 2)

Replace deficits and monitor response

To replace extracellular fluid space deficits (i.e., vascular and interstitial fluid space deficits):

- Administer a buffered isotonic crystalloid fluid that contains a sodium concentration similar to the patient's.
- For rapid intravascular volume replacement, a hypertonic crystalloid, a colloid solution, or both can also be used.[4]
- Closely monitor patient parameters until fluid homeostasis is achieved and maintained (Tables 1, 3–5).
- Monitoring may also be achieved by assessing the relative variation in the caudal vena cava during one respiratory cycle using ultrasonography and calculating the Caudal Vena Cava Collapsibility Index.[6,7] See Table 6 for some conditions that pose additional challenges in addressing individual fluid compartment needs. For more information on addressing fluid therapy challenges, see Section 5, Fluid Therapy in Ill Patients.

Box 2: Calculating Free Water Deficit

Free Water Deficit (FWD) in Liters (L) = [Patient Na/Desired Na) -1] × (0.6 × Weight [kg])

Na, Sodium

TABLE 1

Intravascular Volume Assessment

Criteria	Hypovolemia	Hypervolemia*
Patient history	Vomiting, diarrhea, decreased water intake, anorexia or hyporexia, respiratory signs, fever, blood loss and hemorrhage	Iatrogenic fluid overload, polydipsia, salt intoxication, osmotic agent administration
Physical examination findings	See Table 2 Can occur with severe dehydration (>12%) May see evidence of hemorrhage (bleeding, epistaxis, etc.)	Bounding pulse quality, new cardiac murmur, wet lung sounds, ocular/nasal discharge, jugular vein distention, peripheral edema
Blood pressure or electrocardiogram findings	Hypotension, arrhythmia	Arrhythmia
Laboratory test results	Hyperlactatemia, metabolic acidosis, acute anemia, hypoproteinemia (may be secondary to hemorrhage)	Hemodilution of packed cell volume, blood urea nitrogen, and electrolytes
Diagnostic imaging results (e.g., radiography, ultrasonography, computed tomography)	Microcardia, small caudal thoracic vena cava, caudal vena cava collapsibility index >27%	Abdominal venous distension, caudal vena cava collapsibility index <27%, pleural effusion, ascites, retroperitoneal effusion, perirenal effusion

*Usually occurs in conjunction with signs of overhydration of the interstitial space (see Tables 4 and 5).

TABLE 2

Stages and Clinical Signs of Hypovolemic Shock

	Heart rate	CRT	MM color	Peripheral pulses	Peripheral blood pressure	Extremities	Body temperature
Compensatory							
Cat	Rarely recognized (seconds to a few minutes in duration)						
Dog	Normal or increased	1-2 s	Normal or red	Bounding	Normal or increased	Normal temperature to touch	Hypothermic, hyperthermic or normothermic
Early decompensatory							
Cat	Normal or decreased	>2 s	Pale	Weak	Low	Cool to touch	Hypothermic
Dog	Increased	>2 s	Pale to white	Weak	Normal or decreased	Cool to touch	Hypothermic, hyperthermic or normothermic
Late decompensatory							
Cat	Decreased	>2 s or absent	White	Absent	Low or unable to obtain	Cool to cold to touch	Hypothermic
Dog	Normal or decreased	>2 s or absent	White	Absent	Low or unable to obtain	Cool to cold to touch	Hypothermic, hyperthermic or normothermic

CRT, capillary refill time; MM, mucous membrane

TABLE 3

Estimated Interstitial Dehydration (%) Based on Physical Examination Findings[a]

Estimated % Dehydration	Physical Examination Finding
<5%	• Not detectable
5–6%	• Some change in skin turgor
6–8%	• Mild decreased skin turgor • Dry mucous membranes*
8–10%	• Obvious decreased skin turgor • Retracted globes within orbits
10–12%	• Persistent skin tent due to complete loss of skin elasticity • Dull corneas** • Evidence of hypovolemia
>12%	• Hypovolemic shock • Death

Note: There is substantial clinical variation in the correlation between clinical signs and degree of dehydration, so this is an estimate only.
[a]Reprinted from Silverstein DC and Hopper K, eds., *Small Animal Critical Care Medicine*, 3rd ed., Rudloff, E, Assessment of hydration, p. 1054-58, Elsevier (2022), with permission from Elsevier.
*Xerostomia can be present in AKI and CKD patients without dehydration.
**Retracted globes may also be present.

TABLE 5

Additional Clinical and Diagnostic Findings That May Indicate Overhydration/Fluid Overload

Acute weight gain

Respiratory signs
- Tachypnea
- Cough
- Moist lung sounds
- Labored breathing
- Diagnostic imaging findings consistent with pleural effusion, ascites, and/or pulmonary edema

Edema
- Chemosis
- Subcutaneous edema
- Organ edema and dysfunction (e.g., gastrointestinal signs, altered mentation, arrhythmia)

Serous nasal discharge

Cavitary effusion

Polyuria in the absence of renal failure

Shivering

Restlessness

TABLE 4

Extracellular Hydration Status Assessment Parameters and Expected Changes from Baseline in Patients Receiving Hypo- or Over-hydration[a]

Parameter	Hypohydration	Overhydration
Skin turgor	↓	↑
Mucous membrane moisture	↓	↑
Packed cell volume	↑	↓
Total protein	↑	↓
Blood urea nitrogen	↑	↓
Urine osmolality	↑	↓
Urine specific gravity	↑	↓

[a] Reprinted from Silverstein DC and Hopper K, eds., *Small Animal Critical Care Medicine*, 3rd ed., Rudloff, E, Assessment of hydration, p. 1054-58, Elsevier (2022), with permission from Elsevier.

TABLE 6

Conditions That Pose Challenges When Addressing Individual Fluid Compartment Needs

Condition	Challenge
Hypovolemic shock in cats	• Cats typically develop bradycardia, hypothermia, and hypotension. • This triad of events makes cats more susceptible to hypervolemia and overhydration compared with dogs, when similar fluid resuscitation strategies are used (see Table 2).
Increased capillary permeability (e.g., due to systemic inflammation, burns, trauma)	• Can result in both hypovolemia and overhydration.
Acute congestive heart failure in a patient receiving diuretics and afterload reducers	• Can result in poor perfusion and signs of shock due to cardiovascular dysfunction.
Osmotic diuretic therapy or uncontrolled hyperglycemia	• Can lead to hypervolemia and reduced interstitial and intracellular water volume.

Section 3: Fluids for Replacement and Maintenance

Top Three Takeaways

1. Calculate fluid requirements based on three main phases of fluid therapy: resuscitation, rehydration, and maintenance. The administration route depends on the severity of the patient's fluid deficit and their ability to take in fluids orally. When possible, enteral routes should be used.
2. Use replacement fluids, also called isotonic crystalloids, to treat hypovolemia and dehydration, keeping in mind that each condition requires different strategies. Hypovolemic patients require immediate intravascular volume replacement delivered as one or more small IV or intraosseous (IO) fluid boluses over 15–30 min. Dehydrated patients require sustained fluid delivery over 12–24 hr.
3. Use maintenance fluids, also referred to as hypotonic crystalloids, to provide daily fluid requirements in patients with inadequate fluid intake. Using isotonic crystalloids to meet maintenance fluid needs can lead to electrolyte derangements in patients.

Overview

When developing a replacement or maintenance fluid therapy plan, tailor the fluid type, volume, administration route, and administration rate to each patient. *Keep in mind that evaluating a patient's fluid balance is not a one-time event.* As the patient's clinical status progresses, adjust the fluid prescription to meet ongoing needs, response to therapy, and the course of the disease.[8]

Hypovolemia and **dehydration** are two related medical conditions that involve a deficiency of fluids in the body and require replacement fluid therapy. Although these conditions share similarities, there are distinct differences between them, and fluid administration strategies differ.

Patients with an intravascular volume deficit, or hypovolemia, require rapid IV fluid replacement. On the other hand, patients with fluid deficits from inadequate fluid intake and ongoing losses, or dehydration, need slow, sustained fluid deficit replacement, allowing the interstitial and intracellular compartments to reabsorb these fluids. Once patients are adequately hydrated and euvolemic, they may only require maintenance fluid therapy if they are unable to maintain fluid homeostasis through oral ingestion.

Managing Hypovolemia

Hypovolemia refers to a decreased volume of circulating blood, which results in reduced tissue perfusion. It occurs when both fluid and electrolytes are lost, leading to a decrease in total blood volume in the circulatory system (Figure 3). Hypovolemia can be caused by various factors, such as excessive bleeding, severe burns, severe diarrhea or vomiting, kidney disease, or inadequate fluid intake. Parameters to detect hypovolemia are listed in Table 2.

Immediate medical attention is crucial for hypovolemic patients because untreated hypovolemia can lead to serious complications. Treatment entails administering IV fluids to restore the blood volume and addressing the underlying cause.

Correct hypovolemia by administering a buffered isotonic fluid bolus of 5–10 mL/kg (cats) and 15–20 mL/kg (dogs) over 15–30 min. The boluses can be repeated if the desired hemodynamic and perfusion goals have not been achieved and the patient remains hypovolemic (Figure 4).

Managing Dehydration

Dehydration is a condition in which the body loses more fluids than it takes in, resulting in an imbalance of water and electrolytes (Figure 5). Inadequate fluid intake, excessive panting in dogs, vomiting, diarrhea, or medical conditions such as diabetes can cause dehydration. Parameters to detect dehydration are listed in Table 3.

Depending on the degree of dehydration, it can usually be managed by replenishing lost fluids through oral rehydration or subcutaneous (SC) fluid administration. IV fluid administration is preferred in severe cases of dehydration or in patients who cannot tolerate oral fluid administration.

Dehydration can be corrected by calculating the fluid deficit (Box 3) based on the degree of dehydration (Table 3) and administering fluid therapy over 12–24 hr (Figure 6).

Box 3: Calculating the Fluid Deficit

Fluid Deficit (mL) = Body Weight (kg) × % Dehydration (as a decimal)

Managing Hypovolemia and Dehydration

In cases in which both hypovolemia and dehydration are present (Figure 7), address hypovolemia first, then rehydration (Figure 8). Both fluid prescriptions should include concrete endpoints to monitor to identify when hypovolemia and dehydration have resolved (Table 7). Additionally, ultrasonographic evaluation of cardiac chambers and the caudal vena cava (Caudal Vena Cava Collapsibility Index) may be used as valid assessments of fluid responsiveness.[9]

Selecting Fluids for IV Administration

To prescribe appropriate IV fluid therapy, many factors must be considered, including a patient's age, current and underlying medical conditions (e.g., renal or cardiac impairment, hypoproteinemia), fluid and electrolyte balance, and other specific needs (see Section 5, Fluid Therapy in Ill Patients). These factors influence the choice of IV fluid type and whether adjustments are needed in the fluid's composition or administration rate.

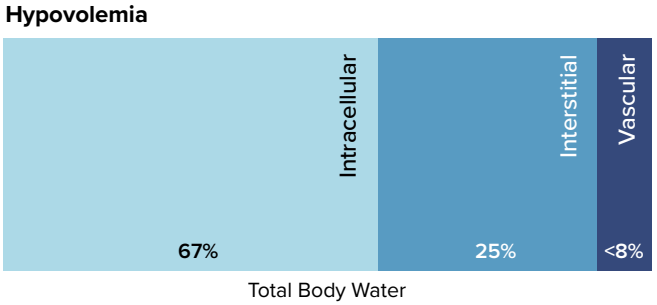

FIGURE 3

Hypovolemia results in a decreased volume within the vascular space. Acute hypovolemia primarily affects this compartment. As the severity and duration of hypovolemia persist, it can affect other compartments as well.

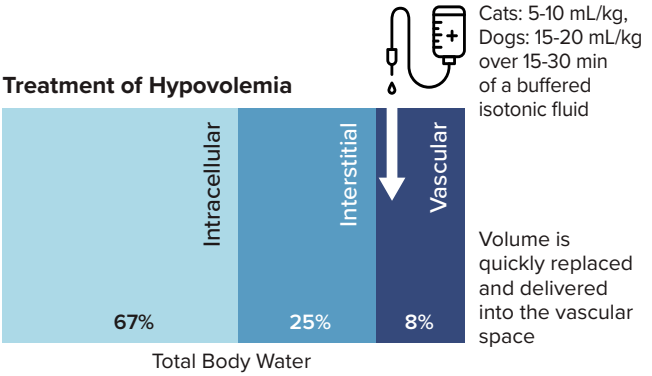

FIGURE 4

Treatment of hypovolemia requires rapidly delivering fluid into the vascular space to restore the effective circulating volume.

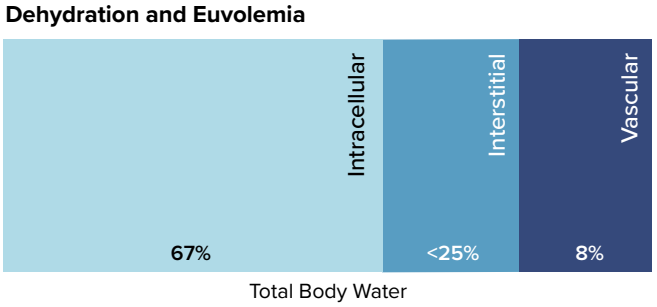

FIGURE 5

Dehydration results in a decreased volume within the interstitial space.

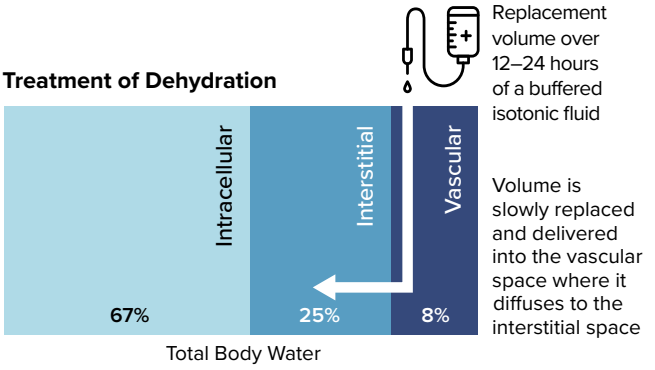

FIGURE 6

Treating dehydration requires slow, sustained delivery of intravascular fluids, which will be slowly absorbed into the interstitial space over 12-24 hours. Subcutaneous and oral routes are not depicted; however, these routes also correct dehydration.

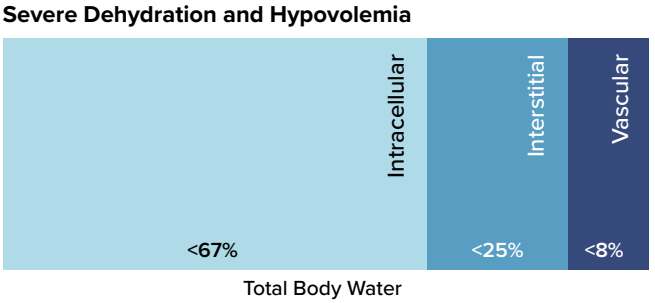

FIGURE 7

Dehydration results in a decreased volume within the interstitial space. As dehydration worsens, it can affect the vascular and intracellular compartments as well, leading to dehydration with concurrent hypovolemia.

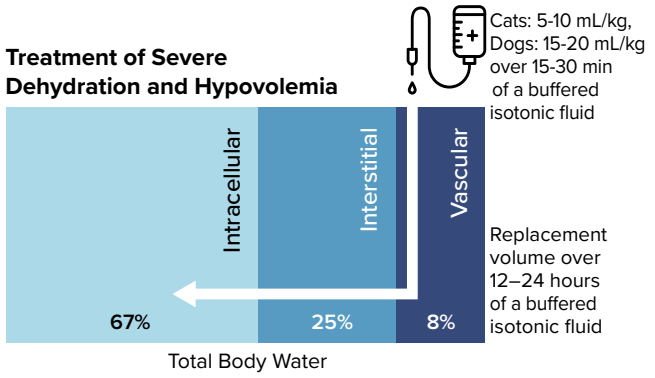

FIGURE 8

Treatment of severe dehydration and hypovolemia requires a two-fold strategy. First, correct hypovolemia by rapidly delivering intravascular fluids and restoring the effective circulating volume. Once the hypovolemia has resolved, address dehydration with the slow and sustained delivery of intravascular fluids administered over 12-24 hours.

TABLE 7

Endpoints to Monitor for Hypovolemia and Dehydration

Fluid Status	Hypovolemia	Dehydration
Initial parameters	(See Table 2)	(See Table 3)
Initial treatment strategy	• 5-10 mL/kg (cat), 15-20 mL/kg (dog) of a buffered isotonic fluid over 15 minutes • Assess perfusion parameters at the end of each bolus.	• Calculate replacement volume and deliver over 12–24 hours. • Assess patient parameters throughout the fluid delivery period with the goal of correcting the full dehydration deficit within 12–24 hours.
End points	• Improvement in heart rate, CRT, blood pressure, and mentation	• Improved skin turgor, mucous membranes, and urine specific gravity and increased body weight and urine output
End point treatment strategy	• If vitals have returned to normal, then assess if dehydration needs to be addressed and continue with a rehydration fluid plan. • If vitals have improved but not normalized, repeat the same or lower-volume bolus and reassess.	• If end points have returned to normal, then assess if oral ingestion is possible. If not, continue with maintenance fluid plan. • If dehydration has not completely resolved, recalculate fluid requirements and deliver over an additional 12–24 hours.

CRT, capillary refill time

Crystalloids

Crystalloid solutions are the most common type of fluid used, and these can be classified as replacement or maintenance solutions. The composition of replacement solutions resembles that of the extracellular fluid (Table 8). Maintenance solutions contain less sodium and more potassium than replacement fluids (Table 8).[10]

Maintenance versus replacement fluids

Using the term "maintenance fluids" to refer to a fluid administration rate is a common misnomer. Instead, the term refers to a classification of crystalloid solutions formulated with different electrolyte concentrations to meet a patient's daily requirements. Replacement fluids (e.g., lactated Ringer's solution) are intended to replace lost body fluids and electrolytes (Table 8). Replacement fluids are frequently used interchangeably to meet replacement and maintenance needs—where clinicians supplement replacement fluids with potassium or dextrose to approximate maintenance requirements. Using replacement fluids long term instead of maintenance fluids may predispose patients to sodium derangements and hypokalemia.[11] Although there is no evidence that using replacement fluids as maintenance fluids has short-term detrimental effects, it is important to refer to these fluids correctly and ensure that patient maintenance needs (electrolyte composition and volume administered) are properly met.

Sodium concentration

Always consider a patient's sodium concentration. Dogs have a lower average sodium concentration (~145 mEq/L) compared with that of cats (~155 mEq/L),[12] and pediatric patients may have slightly lower sodium concentrations than adults. Although point-of-care blood analysis facilitates obtaining quick results, it may not always be available in cases in which immediate fluid resuscitation is required. In situations in which sodium concentration is unknown, the best fluid choice is a buffered isotonic fluid. Once the sodium concentration is obtained, adjust the fluid choice to better reflect the patient's needs (Table 8).

Calculating Fluid Requirements

Divide the fluid therapy plan into resuscitation, rehydration, and maintenance rates (Table 9) as follows:

$$\textit{Total Fluid Requirement} = \textit{resuscitation rate} + \textit{rehydration rate (include ongoing losses)} + \textit{maintenance rate}.$$

Several formulas are available to calculate fluid requirements,[13] and there is no evidence that one is superior to another.[14] Regardless of the formula used, customize the fluid plan to each patient, and adjust it based on patient monitoring findings and ongoing losses.

Selecting Fluid Administration Routes

The choice of fluid administration route depends on the severity of the fluid deficit and the patient's ability to take fluids orally or via a feeding tube. Hypovolemia always requires IV or IO fluid delivery. However, dehydration can be corrected through IV, SC, or enteral fluid administration, or a combination of these routes.

TABLE 8

Composition of Commonly Used Crystalloids[1,2–4]

	Osmolarity (mOsm/L)	pH	Na (mEq/L)	Cl (mEq/L)	K (mEq/L)	Mg (mEq/L)	Ca (mEq/L)	Dextrose (g/L)	Buffers
REPLACEMENT CRYSTALLOID FLUIDS									
Hypertonic Crystalloids									
3% NaCl	1027	5.0	513	513	0	0	0	0	None
5% NaCl	1711	5.0	856	856	0	0	0	0	None
7.5% NaCl	2566	5.0	1293	1293	0	0	0	0	None
23.4% NaCl	8008	5.0	4004	4004	0	0	0	0	None
Isotonic Crystalloids									
0.9% NaCl	308	5.0	154	154	0	0	0	0	None
Plasma-Lyte A	294	7.4	140	98	5	3	0	0	Acetate, Gluconate
Plasma-Lyte 148	294	5.5	140	98	5	3	0	0	Acetate, Gluconate
Normosol R	294	5.5	140	98	5	3	0	0	Acetate, Gluconate
Lactated Ringer's	275	6.5	130	109	4	0	3	0	Lactate
MAINTENANCE CRYSTALLOID FLUIDS									
Hypotonic Crystalloids									
Plasma-Lyte 56 in 5% dextrose	363	3.5-6	40	40	13	3	0	50	Acetate
0.45% NaCl	154	5.6	77	77	0	0	0	0	None
0.45 NaCl in 2.5% dextrose	280	4.5	77	77	0	0	0	25	None
5% dextrose in water	252	4.0	0	0	0	0	0	50	None
Normosol M in 5% dextrose	363	5.0	40	40	13	3	0	50	None

1. Rudloff E, Hopper K. 2021. Crystalloid and colloid compositions and their impact. *Frontiers in Veterinary Science*. 8:639848.
2. Strandvik GF. 2009. Hypertonic saline in critical care: a review of the literature and guidelines for use in hypotensive states and raised intracranial pressure. *Anaesthesia*. 64(9):990-1003
3. Holden D, et al. 2023. Hypertonic saline use in neurocritical care for treating cerebral edema: A review of optimal formulation, dosing, safety, administration and storage. *American Journal of Health-System Pharmacy*. 80(6):331-342.
4. Carr CJ, et al. 2021. An audit and comparison of pH, measured concentration, and particulate matter in mannitol and hypertonic saline solutions. *Frontiers in Neurology*. 12:667842.

Selecting IV and IO Routes

The IV and IO administration routes are preferred in patients with severe fluid deficits, acute fluid losses, and perfusion deficits, as well as in patients with minimal or no oral or enteral fluid intake. These routes allow for rapid dispersion of fluid and electrolytes, ensuring precise dosage and safe delivery of large fluid volumes and hypertonic fluids.

The IO route is typically used in patients for whom IV access is not possible and serves as a bridge for resuscitation until an IV catheter can be placed. However, IO infusion rates are more limited than IV infusion rates; for example, the humerus and femur sites allow IO catheter infusion rates of up to 1 mL/kg/min.[15]

Selecting the SC Route

The SC route is preferred for outpatient fluid therapy or for patients receiving fluids via multiple routes (e.g., IV fluids during day hospitalization and SC fluids during the night). However, evidence-based information is lacking regarding ideal patient selection for SC fluid therapy, the optimal SC infusion volume and treatment frequency, and the possible adverse effects of SC fluids.[13] Table 10 provides empirical recommendations for SC fluid therapy.

To estimate the patient's percent dehydration and calculate maintenance fluid requirements, follow the recommendations outlined above. It is important to avoid prescribing SC fluids for euhydrated patients because there is no evidence that such therapy is beneficial,

TABLE 9

Fluid Therapy Dosing According to Stage of Fluid Requirements

Stage	Formula	Rate of Administration	Comments
Resuscitation	**Cat:** 5–10 mL/kg **Dog:** 15–20 mL/kg	15 min	Assess perfusion parameters after bolus. May repeat bolus as needed.
Rehydration	Total fluid deficit (mL) = Body weight (kg) × % Dehydration (as a decimal)	Over 12–24 hr	Ongoing losses should be assessed through inputs and outputs and incorporated into the fluid plan.
Maintenance	**Dog:** a. 60 mL/kg/day b. 132 × BW (kg)$^{0.75}$ c. 30 × BW (kg) + 70 = mL/kg/day **Cat:** a. 40 mL/kg/day b. 80 × BW (kg)$^{0.75}$ c. 30 × BW (kg) + 70 = mL/kg/day **Pediatric:** Dog: 3 × adult dose Cat: 2.5 × adult dose	Over 24 hr	Also incorporate enteral water, liquid diets, and IV medications into the total volume of the fluid plan.

BW, body weight

TABLE 10

Empirical Subcutaneous Fluid Therapy Recommendations

Subcutaneous Fluid Dose	Frequency	Type of Fluid	Comments
20–30 mL/kg	Once or twice a day	• Lactated Ringer's, Plasma-Lyte, or Normosol R • 0.9% NaCl has a low pH and may be painful. Avoid SC use.	• Deliver to multiple sites depending on volume and skin elasticity. Maximum amount is 10–20 mL/kg per site.

SC, subcutaneous

and it may be detrimental in patients that have challenges in body fluid homeostasis (e.g., underlying cardiac disease and hypoproteinemia). Use a new fluid administration set and fluid bag for each individual patient.

Selecting Enteral Administration

Whenever possible, use the enteral administration route. Enteral fluids are often underused and should be an integral part of the fluid therapy plan if patients can tolerate oral liquids. Placement of feeding tubes can aid in providing enteral fluids in anorexic patients and should be considered as part of an overall fluid therapy plan.

Enteral fluids can be used exclusively to correct mild dehydration in patients with inadequate fluid intake or to supplement parenteral administration routes. First, estimate the patient's percent dehydration and then calculate maintenance fluid requirements as usual. Liquid diets and oral water can be delivered through voluntary intake or feeding tubes (e.g., nasogastric and esophagostomy). Enteral administration may be particularly useful in dehydrated patients who cannot tolerate IV fluids (i.e., patients with cardiac disease or hypoproteinemia). If a feeding tube is present, deliver enteral fluids as intermittent boluses or as a continuous infusion with a fluid pump. Take care to label oral fluids appropriately to avoid connection errors that may lead to accidental administration of water or a liquid diet into a patient's vascular space.

Section 4: Fluid Therapy and Anesthesia

Top Three Takeaways

1. Most healthy animals undergoing elective surgery do not require fluids in the postoperative period. Instead, early return to eating and drinking is recommended.
2. Patients who have not been eating before anesthesia may need postoperative fluid therapy until they can voluntarily consume enough to meet their needs. For patients who are not expected to eat well after surgery, such as geriatric cats, SC fluids can be considered for use at home.
3. In patients with renal disease (specifically, those in International Renal Interest Society stage 3 or 4), do not attempt to rectify hypotension by using excessive fluid infusion rates.

Overview

Fluid therapy is recommended in patients undergoing general anesthesia primarily to counteract the vasodilation and decreased cardiac output induced by inhaled anesthetics, as well as to uphold catheter patency. Before the publication of the 2013 *AAHA/AAFP Fluid Therapy Guidelines for Dogs and Cats*, the recommended fluid administration rate during anesthesia was 10 mL/kg/hr, lacking a foundation of evidence.[16] However, excessively high-volume fluid rates predispose anesthetized patients to an increased risk of volume overload and its associated consequences. The 2013 guidelines recommended reducing fluid rates in anesthetized patients to 5 mL/kg/hr in dogs and 3 mL/kg/hr in cats.[17] Although these reduced administration rates have not undergone formal study, they have been widely accepted and implemented in clinical practice.

Goals for Fluid Therapy in Anesthetized Patients

The following section covers important facets of fluid administration before, during, and after anesthesia, including mechanisms to address hypotension and avoid fluid overload. (For more information on the anesthetized patient, see the 2020 *AAHA Anesthesia and Monitoring Guidelines for Dogs and Cats*, available at aaha.org.)

1. Consider potential fluid deficits (e.g., losses from dehydration, fasting, insensible losses, or anticipated fluid losses during surgery).[18] Whenever feasible, aim to correct 80% of a patient's dehydration deficit within the 24 hr before anesthesia.
2. For most patients, it is unnecessary to withhold water before anesthesia.
3. Place IV catheters in all patients undergoing anesthesia.
4. Administer balanced isotonic crystalloid fluids using the following guidelines:
   a. Initial fluid rate of 5 mL/kg/hr in dogs with normal cardiac and renal function.
   b. Initial fluid rate of 3–5 mL/kg/hr in cats with normal cardiac and renal function.
5. IV fluids can be beneficial for maintaining catheter patency and supporting cardiovascular function. However, euhydrated, euvolemic patients receiving injectable anesthetics for short periods generally do not require IV fluids. Food and water should be offered as soon as possible after recovery, but SC fluids can be given to patients who do not return to eating and drinking immediately. Prioritize the correction of hypovolemia and dehydration before general anesthesia whenever possible.
6. To ensure proper tissue perfusion, maintain a minimum mean arterial blood pressure of 60 mm Hg.

Managing Hypotension

Hypotension is a common complication of general anesthesia involving inhaled anesthetics (Figure 9).[19–21] Both absolute hypovolemia (e.g., due to hemorrhage) and relative hypovolemia (e.g., stemming from trauma, sepsis, or the use of anesthetics and vasodilatory drugs) can also contribute to hypotension. In cases of hypotension during anesthesia, begin by adjusting excessive vaporizer settings and judiciously administering crystalloid fluids.

1. Assess every patient's anesthetic depth carefully. Small reductions in inhalant anesthetic administration (i.e., vaporizer settings) can substantially affect blood pressure and make the difference between normotension and hypotension.
2. Monitor the patient's heart rate. Bradycardia can contribute to hypotension in anesthetized patients. If the heart rate is lower than normal and the patient is hypotensive, consider anticholinergic therapy.
3. Evaluate body temperature. Decreased body temperature or hypothermia can cause hypotension. Open body cavities and breathing in cold gases from the anesthesia machine can both lead to a decrease in body temperature. Increasing body temperature will help improve hypotension.

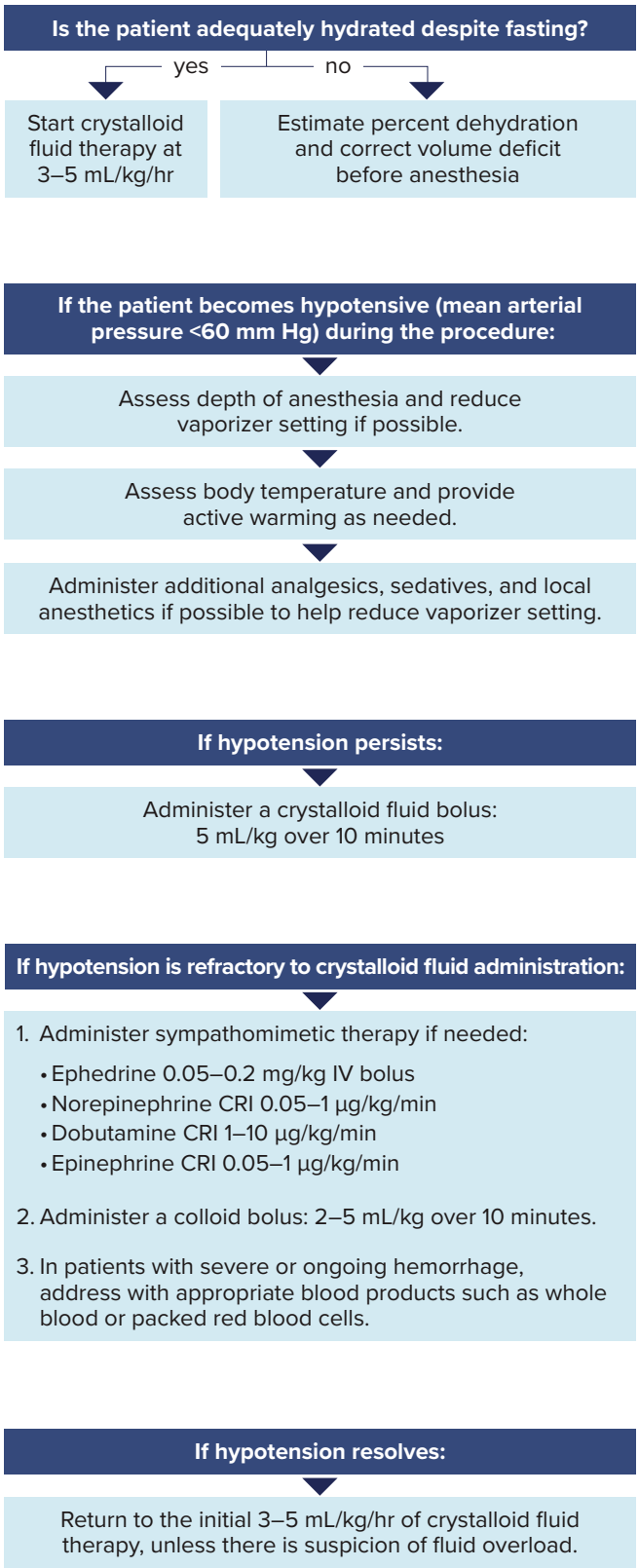

FIGURE 9

Fluid Therapy During Anesthesia

CRI, continuous rate infusion

4. Consider the concurrent administration of additional analgesics, sedatives, or a combination of both to help reduce the requirements for inhalant anesthetics requirements, as well as the use of regional or local anesthetics.
5. Administer balanced isotonic crystalloid fluids when using inhalant anesthetics:
   i. 5 mL/kg/hr for dogs
   ii. 3–5 mL/kg/hr for cats
6. If hypotension persists despite adjusting vaporizer settings and providing crystalloid fluids, then consider sympathomimetic drug therapy with inotropes or vasopressors or colloid fluid therapy.
7. If hypotension is due to severe or ongoing hemorrhage, then transfusion of whole blood or packed red blood cells is necessary to maintain both blood volume and appropriate red blood cell mass to deliver oxygen to tissues.
8. Keep in mind that not all cases of hypotension can be corrected with fluid administration, particularly in pediatric patients and those with cardiac disease, sepsis, etc. (see Section 5, Cardiorenal Disorders, Fluid Therapy in Ill Patients).
9. Remember that both hypovolemia and hypervolemia are detrimental to anesthetized patients.

Monitoring

1. Monitor the duration of anesthesia and total volume of administered fluids closely. If fluid rates surpass 20 mL/kg within a single anesthetic episode, reevaluate both fluid administration rates and the intravascular volume status of the patient. Typically, most healthy animals would not require a maintenance rate of 5 mL/kg/hr for extended periods, unless significant blood loss occurs. To determine the total fluid administration during a single anesthetic event, calculate the daily maintenance rate volume (Table 9).
2. Monitor anesthetized patients carefully to detect any indications of excessive fluid administration. Signs of fluid overload include (see Table 5 for additional clinical and diagnostic signs):
   a. Gallop sound or new murmur (especially in cats)
   b. Edematous tissues and chemosis
   c. Swelling of paws
   d. Clear nasal discharge (nasal edema)
   e. Pulmonary crackles
   f. Low oxygen saturation (SpO_2)
   g. No alteration in blood pressure along with other clinical signs (i.e., patients remain nonhypertensive)
   h. Pleural effusion, ascites
3. Stop fluid administration (or use a minimal volume to maintain catheter patency) if patients have signs of excessive fluid administration. Furosemide (1–2 mg/kg IV) may be needed if patients have signs of pulmonary edema (i.e., audible pulmonary crackles, imaging evidence of pulmonary edema, or low SpO_2) or pleural effusion.
4. For anesthetized patients subjected to positive-pressure mechanical ventilation, consider the use of a pulse pressure variability monitor[22,23] or plethysmographic variability index from advanced pulse oximetry to assess fluid responsiveness.[24,25] The monitor is used in a similar fashion to a pulse oximeter and helps evaluate whether cardiac output increases with volume expansion.
5. Return routine surgical patients to normal eating and drinking as soon as possible after anesthesia.

Patients with Renal Disease

To effectively care for patients with renal disease (specifically, those in International Renal Interest Society stage 3 or 4), it is crucial to correct dehydration before anesthesia, optimize cardiac output by using an appropriate anesthetic protocol that supports cardiovascular function (avoid dexmedetomidine if other alternatives are available), and closely monitor and manage blood pressure. Attempting to rectify hypotension by using excessive fluid infusion rates should be avoided.

Use of synthetic colloids in patients is currently controversial because of safety and efficacy considerations and lack of evidence-based consensus.

For more information on colloids, see Section 7, Questions and Controversies in Fluid Therapy.

Box 4: Special Fluid Therapy Needs During Anesthesia

1. **Hypoglycemia**
   - Add dextrose for pediatric toy dog breeds OR in response to hypoglycemia in dogs or cats.
     - Add 50 mL 50% dextrose to 1 L fluid bag to create a 2.5% dextrose solution.
2. **Acute surgical blood loss**
   - Increase fluid rate (up to 10 mL/kg/hr) while surgically correcting the issue (e.g., dropped pedicle).
   - Consider that it takes three times as much crystalloid fluid volume to replace one volume of blood lost.
   - If blood products are unavailable, and the patient's perfusion is compromised, consider a colloid to aid perfusion.
3. **Hypoproteinemia**
   - Use canine albumin when total protein is <2.0 g/dL (a dose of 450 mg/kg canine albumin will increase the serum albumin by 0.5 g/dL). Start at a rate of 0.5 to 1 mL/kg for 30 min and increase the rate if no adverse effects are noted. Deliver over 3–4 hr. For more information, see Mazzaferro EM, Edwards T. Update on albumin therapy in critical illness. *Vet Clin North Am Small Anim Pract* 2020;50(6):1289–305.
   - Use a colloid with a crystalloid when total protein is <4.0 g/dL (hetastarch 1–5 mL/kg/hr in the anesthetized patient).
   - Use fresh frozen or frozen plasma when available.
     - Consider that it takes ~20–25 mL/kg to raise the albumin by 0.5 g/dL, and for large-breed dogs, this could be cost prohibitive. For specific doses, see Beer KS, Silverstein DC. Controversies in the use of fresh frozen plasma in critically ill small animal patients. *J Vet Emerg Crit Care (San Antonio)* 2015;25(1):101–6.

Section 5: Fluid Therapy in Ill Patients

Top Three Takeaways

1. Do not forget about enteral routes of fluid delivery, including nasogastric, nasoesophageal, or esophageal, when treating sick patients. If tolerated by the patient, water can be mixed with food or administered separately.
2. Do not withhold fluids in patients who are dehydrated or hypovolemic because of concurrent anemia. Monitor them closely to determine whether a transfusion is also indicated.
3. Mitigate electrolyte derangements carefully. Never administer a bolus of fluids supplemented with potassium chloride (KCl). Thoroughly mix the fluids to ensure even dispersion of the KCl before administration. Intentionally manage sodium derangements to avoid potential life-threatening fluid shifts in the brain that can occur with rapid resolution of chronic (>24–48 hr) sodium alterations.

Overview

Fluid therapy in sick patients requires a cautious, balanced approach and the ability to predict problems before they occur. The veterinary team faces complex challenges in fluid therapy when treating patients who present with conditions such as gastrointestinal, renal, or cardiac disease, anemia, electrolyte imbalances, traumatic brain injury (TBI), hypovolemic or vasodilatory shock, edema, thermoregulation disorders, and hypoglycemia.

Nutritional Therapy as a Prompt for Enteral Fluid Therapy

Nutrition is one of the most neglected patient requirements during hospitalization.[26] After 72 hr of anorexia, a patient's metabolism shifts to alternate energy sources, such as ketones and fatty acids, instead of glycogen and glucose.[27] Nasoesophageal, nasogastric, or esophageal feeding tubes facilitate caloric intake,[28] and water can be mixed with food or administered separately.[29] Use a canned diet to increase water intake.

The stomach's capacity is 5–10 mL/kg[30] at the time of starting enteral nutrition, and no consensus on canine and feline gastric emptying times exists (although prolonged emptying times have been reported).[31] Use conservative calculations of fluid and food requirements for enteral nutrition and adjust based on a patient's clinical signs of nausea, regurgitation, and vomiting.

Determine enteral water requirements based on daily maintenance rates and divide this amount between IV and enteral supplementation.[32] It is important to continue to provide free access to water. Given that enteral nutrition rates typically start at one-third the resting energy requirement[33] to avoid refeeding syndrome, enteral water administration may also aid in allowing the stomach capacity to accommodate increased volumes of subsequent enteral nutrition feedings.

Anemia

Do not withhold fluids in anemic patients. If an anemic patient is also dehydrated or hypovolemic, provide fluid therapy while recognizing that patients with a low hematocrit may require blood products. In healthy patients, fluid resuscitation has not been shown to decrease hemoglobin concentrations.[34] Fluid therapy may result in a beneficial increase in microvascular flow and perfusion with an overall increase in oxygen delivery in patients with hypovolemic or distributive shock. However, fluid administration in non–fluid responders or fluid-overloaded patients can lead to a relative, but not absolute, reduction in hemoglobin concentration ("dilutional anemia"), which can cause a paradoxical decrease in oxygen delivery.

Carefully monitor anemic patients and thoroughly assess them for shock, hypovolemia, dehydration, and need for maintenance fluids. These needs should be addressed through an appropriate fluid prescription. These patients may become transfusion dependent when appropriately resuscitated or rehydrated. Transfusion triggers (e.g., heart rate, mucus membrane color, capillary refill time, respiratory rate and effort, pulse quality, blood pressure, mentation, and attitude) should be considered in conjunction with laboratory test results (e.g., packed red blood cell count and trends, hematocrit, hemoglobin levels, and blood lactate) to determine the need for a blood transfusion.[35,36]

Azotemia

Azotemic patients have varying fluid requirements that depend on factors such as their hydration status (including both dehydration and fluid overload), urine production levels, acute versus chronic onset, acid-base and electrolyte status, the underlying cause of the azotemia, and the extent of its severity.[37] These patients present special monitoring challenges as they might experience xerostomia (dry mouth) secondary to uremia,[38] prolonged skin tenting due to reduced skin elasticity associated with aging, concentrated retained urine, and inaccurate relative creatinine levels due to decreased muscle mass.[39] Additionally, there may be a lack of a baseline creatinine concentration or previous body weight for comparison. Anecdotal techniques to assess dehydration include evaluating skin turgor over the rib cage, analyzing weight trends and sodium concentrations, fluid and food intake history, and identifying signs of fluid overload (see Section 6, Fluid Overload).

In cases in which patients are not hypotensive, the fluid prescription should provide a gradual correction of dehydration, whereas patients with significant renal compromise should receive fluids at slower rates. Fluid therapy is not the mainstay of treating azotemic patients. Rather, it is to support the kidneys by correcting treatable abnormalities associated with renal compromise so that the kidneys can heal themselves. Key aspects entail reducing sodium and chloride load,[40] managing blood pressure, treating anemia[41,42] and infections, ensuring adequate short-term nutrition (without protein restriction), and addressing primary conditions that may trigger secondary AKI (e.g., acute pancreatitis)[43] or acute-on-chronic kidney disease presentations. Fluid requirements for patients with chronic kidney disease vary depending on the severity of polyuria and polydipsia along with other clinical signs.[44]

Heart Disease

The most important consideration for fluid therapy in patients with cardiac disease is to prevent the onset of heart failure.[45] For cardiac patients, therapeutic goals include increasing myocardial contractility, decreasing preload and afterload, counteracting the pathological effects of the renin-angiotensin-aldosterone system, improving vasodilation, and optimizing diastolic filling.[46,47]

Cardiac patients may experience dehydration, relative hypovolemia, electrolyte disturbances, moderate to severe azotemia, and metabolic imbalances caused by heart failure and medications. Fluid therapy is frequently avoided in cardiac patients owing to its potential to increase preload in left-sided heart failure, increase afterload, and decrease venous return in right-sided heart failure (especially in the presence of increased intra-abdominal pressure from ascites).[48–50] Whenever possible, fluid intake should be provided enterally, such as through water and a canned diet. When fluid therapy is necessary, administer 0.45% NaCl with 2.5% dextrose IV at half to daily maintenance rates (See Table 9 for maintenance rates), depending on the patient's needs and tolerance of supplemental fluids.[51] Hypotension in patients with congestive heart failure should be addressed by considering positive inotropes.

Cardiorenal Disorders

The cardiorenal axis is an important consideration[51] because a pathological state in either the cardiovascular or renal system has the potential to affect the other.[52] Renal disease treatment focuses on maintaining hydration with enteral water, so less conflict exists with heart disease treatment; however, the administration of additional fluid therapy presents challenges. Cardiac medications may potentially lead to mild azotemia through diuresis (e.g., furosemide)[53] and decreased glomerular filtration rate (GFR) (e.g., enalapril and telmisartan).[54] It is crucial to consistently monitor for fluid overload, severe progressive azotemia, worsening of heart failure, changes in blood pressure, and other relevant indicators to ensure that these patients do not decline as a result of fluid therapy. Even with the best possible therapy, this can be a significant challenge.

Patients with Hypovolemia and Edema

Alterations in Starling's forces (hydrostatic and oncotic pressure) (Figure 2) may contribute to the development of edema in veterinary patients. Common causes of edema include vasculitis, hypoalbuminemia, heart failure, kidney failure, lymphatic obstruction, and

thrombosis.[55] Fluid therapy in hypovolemic, edematous patients presents a therapeutic challenge, and the underlying cause of the edema must be taken into consideration in order to safely provide fluids.

When edema results from hypoalbuminemia, intravascular volume resuscitation and restoration are paramount, and colloids can be used to raise the colloid oncotic pressure. This can be accomplished with rapid administration of synthetic colloids (e.g., hetastarch and tetrastarch [although their use is controversial; see Section 7, Questions and Controversies in Fluid Therapy]), canine-specific albumin (CSA) in dogs, human serum albumin in dogs or cats (which may predispose patients to allergic reactions or anaphylaxis), and veterinary plasma products (e.g., fresh frozen plasma, frozen plasma, and cryoprecipitate-poor plasma [CPP]).

Although plasma products are less prone to induce allergic reactions, large plasma volumes are needed to significantly change albumin concentrations (~22 mL/kg plasma to increase serum albumin by 0.5 g/dL), which results in higher costs of care and risk of volume overload. In one study, there was no difference in mean serum albumin concentrations before and after transfusion with fresh frozen plasma (median dose of 15–18 mL/kg).[56] Thus, although a plasma bolus may be used to treat hypovolemia in edematous patients, its use for this purpose is controversial.[57]

A continuous rate infusion (CRI) of canine CPP, administered at a rate of 1.1–2.2 mL/kg/hr, has been described in a case report for treating hypoalbuminemia.[58] This may be a more reasonable approach to treating hypoalbuminemia because CPP has a higher albumin concentration than other plasma products,[59] although CPP is less widely available.[58]

CSA appears to be a relatively safe alternative to synthetic colloids and increases albumin concentrations more efficiently than plasma products.[60,61] In one study, CSA administration improved the shock index in hypovolemic canine patients.[61] In another study, CSA increased arterial blood pressure within 2 hr of administration in dogs with septic peritonitis.[60]

Giving colloids to patients with edema due to vasculitis may be controversial, as colloids may leak into the interstitial space and worsen interstitial edema. In these patients, judicious colloid use combined with lower volumes of crystalloids is recommended. Overall, regardless of the cause of edema, use crystalloids judiciously in all affected patients.

Low-dose diuretics may be considered in patients with edema, but only in normotensive, normovolemic patients.

Traumatic Brain Injury

The primary fluid therapy goal in treating veterinary patients who have TBI is optimizing cerebral perfusion pressure and mean arterial blood pressure. The Brain Trauma Foundation guidelines for human patients recommend maintaining systolic arterial blood pressure between 100 and 110 mm Hg to reduce mortality and improve outcomes.[62]

Studies that evaluate the ideal fluid choice for veterinary patients with TBI are limited. However, information based on human clinical studies and swine and rodent research studies supports the use of packed red blood cells, plasma, and platelets over crystalloid fluids because of better outcomes in patients with ongoing hemorrhage.[63,64]

Osmotherapy—using osmotic agents (e.g., hypertonic saline or mannitol) to reduce intracranial content volume—is common in treating patients who have TBI (Figure 10). Various studies and a meta-analysis in human medicine suggest that both mannitol and hypertonic saline effectively lower intracranial pressure, but there is no evidence to recommend one over the other.[65–67] Hypertonic saline may have the advantage of avoiding diuresis, increasing cardiac preload, and positively impacting cerebral perfusion.[65] In dogs and cats, mannitol is usually dosed at 0.5–1 g/kg IV given over 15 min with a microfilter, whereas hypertonic saline (NaCl 7.2%) is dosed at 1–6 mL/kg IV given over 15 min. Continuous infusion of hypertonic saline has been described in human patients, but it is not thought to significantly affect outcome.[68]

Electrolyte Disturbances

Electrolyte imbalances are common in ill patients. Proper fluid selection and, if necessary, supplementation of electrolytes aid in restoring balance.

Hypokalemia

Dogs and cats in poor health often experience hypokalemia. This may be due to increased urinary or gastrointestinal loss, prolonged anorexia, alkalemia, an aldosterone-secreting tumor, or treatment with potassium-poor replacement fluids.[69] Clinical signs of hypokalemia may include weakness, cardiac arrhythmias, and respiratory muscle impairment.[70]

Treat hypokalemia with KCl supplementation in IV fluids (Table 11). Determine the KCl dosage based on the patient's serum potassium concentration. For IV KCl administration, calculate the total mEq/kg/hr to be administered, ensuring that the supplementation rate does not exceed 0.5 mEq/kg/hr, as rapid administration can be fatal.[70] Never bolus fluids supplemented with KCl. Before administration, invert the bag and thoroughly mix the fluids to ensure uniform dispersion of the KCl.[71] Exercise caution with highly supplemented fluids or KCl CRIs to prevent inadvertent bolus or over administration to the patient. Use appropriate fluid pump settings or syringe pumps as safeguards and educate staff on the risks of over administration. In cases of persistent hypokalemia even after KCl supplementation, check magnesium levels to determine whether magnesium supplementation is needed.[72]

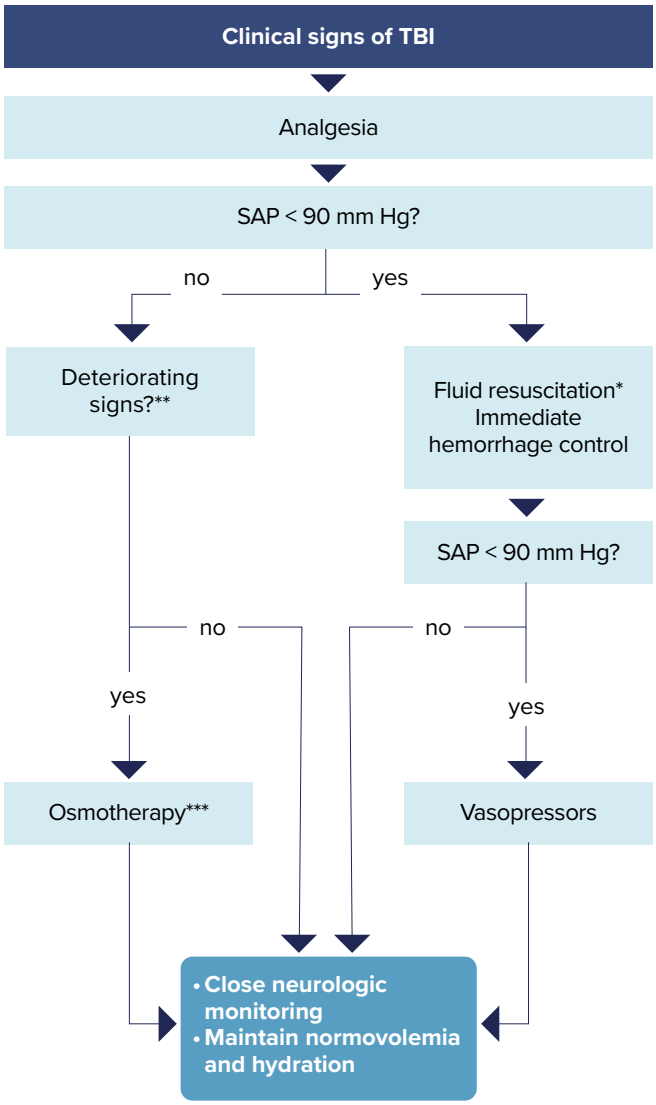

FIGURE 10

Approach to Fluid Therapy for Dogs and Cats with Traumatic Brain Injury

* Fluid resuscitation techniques can be any one of the following or a combination thereof: (1) 10–20 mL/kg crystalloids (Plasma Lyte or Normosol-R) IV rapid infusion up to 60–90 mL/kg. (2) 5–10 mL/kg 6% HES (tetrastarch) IV rapid infusion up to 40–50 mL/kg. (3) 5–10 mL/kg plasma rapid infusion IV up to 20–30 mL/kg. (4) 3–4 mL/kg 7% HTS IV over 10–15 min. (5) Whole blood or pRBC, if indicated.

**Altered level of consciousness with or without bilateral or unilateral miotic pupils; unresponsive mid range pupil(s) or mydriasis; loss of the oculocephalic reflex; bradycardia with hypertension (Cushing reflex); posturing (opisthotonus, decerebellate, decerebrate); alteration of the respiratory pattern.

***1 g/kg mannitol IV up to 3 doses *q* 60–90 min OR 3–4 mL/kg 7% HTS IV.

[d]Reprinted with permission from Pigott A, Rudloff E. Traumatic brain injury–a review of intravenous fluid therapy. *Front Vet Sci.* 2021;8:643800.

Sodium imbalances

Sodium concentration disturbances are common. These cases should be managed with deliberate care to prevent potentially life-threatening fluid shifts in the brain that may occur after rapid correction, faster than 0.5 mEq/kg/hr, of chronic (greater than 24–48 hr) sodium alterations.

Hyponatremia. Patients with acute euvolemic hyponatremia (e.g., primary polydipsia) may present with neurologic signs and should be treated with 2–6 mL/kg of 3–7.5% hypertonic saline over 10–15 min.[73–75] In symptomatic chronic hyponatremia, a similar approach using hypertonic saline should be taken. Once neurological clinical signs resolve, treatment should continue similarly to that in asymptomatic chronic hyponatremia patients. In asymptomatic chronic hyponatremia patients, isotonic crystalloids (Table 12C) that have a sodium content ~10 mEq/L higher than the patient's sodium should be selected. A rate of correction should be calculated that does not exceed 0.5 mEq/L/hr or a maximum sodium increase of 10–12 mEq/L/day to prevent osmotic demyelination syndrome (Tables 12A, 12D).[76]

Patients with hypovolemic hyponatremia should be resuscitated with a crystalloid that contains a similar sodium content to the patient's current sodium concentration.[74,75] Depending on the severity of the hyponatremia, commercial isotonic crystalloids may not be available with similar sodium contents. In these cases, custom fluids can be tailored by adding sterile water to an isotonic crystalloid to achieve the desired sodium content. These fluids should be used exclusively for bolus administration until hypovolemia is resolved. Hypotonic fluids should not be used in patients with hyponatremia.[75]

Hypernatremia. The strategies to treat hypernatremia are similar to those taken with hyponatremia regarding the considerations of chronicity and rate of correction of 0.5 mEq/L/hr. Sodium concentration drops that are faster than this rate in patients with chronic hypernatremia may cause abrupt fluid shifts that lead to cerebral edema.[77] However, patients with acute hypernatremia can undergo rapid sodium concentration correction without the risk of cerebral edema, by using hypotonic IV fluids. Calculate the free water deficit and administer fluids at an appropriate rate while monitoring sodium concentrations frequently to allow for a safe resolution of hypernatremia (Table 13A).

Hypochloremia. Relative shifts in chloride usually occur with shifts in sodium. To assess true chloride disturbances in the face of sodium derangements, chloride should be corrected using the following equation:

$$\text{Corrected Cl} = (\text{normal Na}/\text{measured Na}) \times \text{measured Cl}$$

In patients with true hypochloremia that have developed metabolic alkalosis, 0.9% NaCl has historically been the fluid of choice because of its high chloride concentration (154 mEq/L). Recently, concerns have been raised about the effect of 0.9% NaCl on kidney function owing to the potential risk of causing hyperchloremia, which has been shown to cause renal vasoconstriction and reduced renal blood

TABLE 11

Guidelines for Potassium Supplementation in Fluids

Serum Potassium Concentration	Suggested Potassium Dose	Suggested Potassium Added to Isotonic Crystalloids at 60 mL/kg/Day for a 10-kg Dog (25 mL/hr)
<2.0 mEq/L	0.5 mEq/kg/hr	200 mEq/L
2.0–2.5 mEq/L	0.3–0.4 mEq/kg/hr	120–160 mEq/L
2.6–3 mEq/L	0.2–0.25 mEq/kg/hr	80–100 mEq/L
3.1–3.5 mEq/L	0.1–0.15 mEq/kg/hr	40–60 mEq/L
>3.5 mEq/L	0.05 mEq/kg/hr	20 mEq/L

TABLE 12A

Approach to Fluid Therapy in Hyponatremic Patients

1. Is hyponatremia acute or chronic?

ACUTE	CHRONIC
A. Raise the serum sodium concentration as quickly as possible. B. Administer isotonic crystalloids with a sodium concentration greater than the patient's serum sodium concentration. C. Recheck serum sodium concentrations 2–4 hr after starting therapy to assess therapeutic response, then recheck them every 6–8 hr afterward.	A. It takes 24–48 hr for the brain to compensate for hyponatremia. B. Correct chronic hyponatremia slowly to prevent osmotic demyelination syndrome. C. Increase the serum sodium concentration by no more than 0.5 mEq/L/hr for a maximum total correction of 10–12 mEq/L/day.

2. Does the patient have clinical signs of hyponatremia?

A. Clinical signs include vomiting, disorientation, and seizures secondary to cerebral edema.
B. If symptomatic, treat with 3, 5, or 7.5% hypertonic saline at a recommended dose of 2–6 mL/kg given over 10–15 min.[1]
C. In human patients, serum sodium concentration increases of 4–6 mEq/L are often enough to alleviate clinical signs.[1]

3. Is the patient hypovolemic?

A. Perform fluid resuscitation: 5-10 mL/kg (cats) or 15-20 mL/kg (dogs) given rapidly over 15–30 min with a buffered isotonic solution capable of expanding the intravascular space (Table 12c).[1]
B. Repeat as needed until perfusion parameters are restored.
Maintenance or hypotonic fluids (0.45% NaCl, 5% dextrose in water) have low sodium concentrations and are not indicated to treat hypovolemia.[1]

4. Does the patient have chronic hyponatremia without neurologic signs?

A. Slowly correct the sodium concentration at a maximum rate of 0.5 mEq/L/hr or 10-12 mEq/L/day.
B. Treat asymptomatic patients with mild water restriction and monitor their serum sodium concentrations.
C. Use the Adrogue-Madias formula below to calculate the expected change in sodium concentration when 1 L of a specific fluid type is administered (see Table 12c).[2]

Expected change in serum sodium concentration with 1 L of fluid =
Fluid sodium concentration − serum sodium concentration / (total body water + 1)
Where total body water = body weight in kg × 0.6

1. Adrogue HJ, Tucker BM, Madias NE. Diagnosis and management of hyponatremia: a review. *JAMA.* 2022;328(3):280-91.
2. Heinz J, Cook A. Evaluation and management of the hyponatremia patient. *Today's Veterinary Practice.* 2022;12(2). February 10, 2022. Available at https://todaysveterinarypractice.com/internal-medicine/evaluation-and-management-of-the-hyponatremic-patient/. Accessed January 4, 2024.

TABLE 12B

Common Causes of Acute and Chronic Hyponatremia in Dogs and Cats

Acute	Chronic*
• Consumption of large amounts of fresh water leading to acute water intoxication • Infusion of significant volumes of non replacement fluids (e.g., administering a 5% dextrose in water solution to a dehydrated patient)	• Congestive heart failure • Hypoadrenocorticism • Liver dysfunction • Nephrotic syndrome • Renal and gastrointestinal sodium loss *Consider that patients with vague clinical signs for longer than 24–48 hours likely have chronic hyponatremia.

TABLE 12C

Sodium Concentration of Isotonic Crystalloids

Lactated Ringer's solution	130 mEq/L
Plasma-Lyte A	140 mEq/L
Normosol R	140 mEq/L
0.9% NaCl*	154 mEq/L

*Evidence in human patients suggests that 0.9% NaCl may be detrimental to kidney health.[1,2]

1. Ostermann M, Randolph AG. Resuscitation fluid composition and acute kidney injury in critical illness. *New England Journal of Medicine.* 2022;386(9):888-889.
2. Sigmon J, May CC, Bryant A, Humanez J, Singh V. Assessment of acute kidney injury in neurologically injured patients receiving hypertonic sodium chloride: does chloride load matter? *Annals of Pharmacotherapy.* 2020;54(6):541-546.

TABLE 12D

Calculating Expected Changes in Sodium Concentration

Stevie, a 10 kg, 5-year-old, male neutered Jack Russell terrier, presented for evaluation of vomiting and diarrhea of 72 hours duration. Stevie was mildly lethargic, but all perfusion parameters (heart rate, capillary refill time, blood pressure, pulse quality) were normal, and no other abnormalities were found on physical examination. Stevie's serum sodium concentration was 115 mEq/L.

Stevie is presumed to have chronic hyponatremia because clinical signs have been present for longer than 48 hours. Stevie does not have neurologic signs, so treatment with a hypertonic saline bolus is not indicated. An isotonic crystalloid bolus is also not indicated because Stevie's perfusion parameters are normal.

To correct the hyponatremia, the Adrogue-Madias formula was used with Normosol R as the fluid of choice:

Expected change in serum sodium concentration with 1 L of Normosol R =

$$\frac{140 \text{ mEq/L} - 115 \text{ mEq/L}}{(10 \text{ kg} \times 0.6) + 1} = 3.57 \text{ mEq/L}$$

Therefore, 1 L of Normosol R will change Stevie's sodium concentration by ~3.5 mEq/L.

If Stevie is treated at 25 mL/hr (60 mL/kg/day), ~600 mL of Normosol R will be administered over 24 hours, which will estimate the sodium change at 2.1 mEq/L.

For a faster correction rate, hypertonic saline may be infused into Normosol R to increase fluid sodium concentration using this formula:

Fluid Na = Patient Na + [Target increase in patient's NA over set time x (TBW + Volume of fluids administered over set time)][1]

TBW = body weight in kg × 0.6

1. Heinz J, Cook A. Evaluation and management of the hyponatremia patient. *Today's Veterinary Practice.* 2022;12(2). February 10, 2022 Available at https://todaysveterinarypractice.com/internal-medicine/evaluation-and-management-of-the-hyponatremic-patient/. Accessed January 4, 2024.

TABLE 13A

Approach to Fluid Therapy in Hypernatremic Patients

1. Is hypernatremia acute or chronic?	
ACUTE	**CHRONIC**
• Use hypotonic intravenous fluids to correct. • Can undergo rapid sodium concentration correction without the risk of cerebral edema. • Calculate the free water deficit and administer at an appropriate rate (see #3 below). • Monitor sodium concentrations every 4-6 hours.	• It takes 24–48 hours for the brain to compensate for hypernatremia. • Correct chronic hypernatremia slowly to prevent cerebral edema. • Decrease serum sodium concentration by no more than 0.5 mEq/L/hr for a maximum total correction of 10–12 mEq/L/day (see #3 below).
2. Is the patient hypovolemic?	
• Perform fluid resuscitation with a buffered isotonic solution capable of expanding the intravascular space. • Maintenance or hypotonic fluids (0.45% NaCl, 5% dextrose in water) have low sodium concentrations and are not indicated to treat hypovolemia. • Fluids listed in Table 12c are suitable options to treat hypovolemia (5-10 mL/kg [cat] and 15-20 mL/kg [dog] given over 15–30 minutes and repeated as needed) until perfusion parameters are restored.	
3. Calculations for chronic and acute hypernatremia	
Estimate the amount of water lost (free water deficit). Administer fluids that are relatively dilute compared with plasma. **Free Water Deficit (FWD) in Liters (L) = [(Patient Na/Desired Na) -1] × (0.6 × Weight [kg])**	Modify the calculation of the free water deficit according to whether hypernatremia is acute or chronic, using the subsequent formulas: **FWD replacement time (hr) for acute hypernatremia = Patient Na − Target Na**[1] **FWD replacement time (hr) for chronic hypernatremia = (Patient Na − Target Na) × 2**[1] In general, replace the free water deficit by administering 5% dextrose in water.
4. Is the patient dehydrated?	
• Simultaneously treat by administering a buffered isotonic crystalloid (Table 12c). • Correct dehydration over 12–24 hours to minimize shifts in sodium.[1] • Recheck sodium concentrations every 4–6 hours to prevent dramatic changes. • Limit drinking water until the patient's sodium is close to the target concentration.	

1. Heinz J, Cook A. Evaluation and management of the hyponatremia patient. *Today's Veterinary Practice*. 2022;12(2). February 10, 2022. https://todaysveterinarypractice.com/internal-medicine/evaluation-and-management-of-the-hyponatremic-patient/. Accessed January 4, 2024.

TABLE 13B

Common Causes of Acute and Chronic Hypernatremia in Dogs and Cats

ACUTE	CHRONIC
Intake of large amounts of sodium chloride (ingestion of salt water, homemade playdough, or salt)	Hypotonic fluid losses (diarrhea, peritonitis, vomiting, kidney disease)
	Nephrogenic diabetes insipidus
	Heatstroke
Infusion of replacement fluids or hypertonic fluids may lead to acute or chronic hypernatremia, depending on how often the patient's sodium concentration is rechecked during hospitalization.	

flow.[78,79] To safely use 0.9% NaCl in hypochloremic renal patients, recheck chloride concentrations frequently, and once chloride levels have been corrected, use a buffered isotonic crystalloid (e.g., lactated Ringer's solution or Plasma-Lyte) instead.

Hypocalcemia. Use calcium gluconate to treat patients with clinical signs of hypocalcemia (e.g., weakness, tachycardia, and tremors). Lactated Ringer's solution contains a very small amount of calcium, and it will not resolve hypocalcemia.

Hypercalcemia. Historically, 0.9% NaCl has been recommended for calciuresis in hypercalcemic patients, but its effects are generally mild. Because of concerns regarding kidney injury with 0.9% NaCl, consider using a balanced isotonic crystalloid instead in patients at risk of or with current kidney disease.

Vasodilatory States

Patients may exhibit signs of poor perfusion or shock (such as tachycardia, hypotension, weak peripheral pulses, or elevated lactate concentrations) due to generalized vasodilation, also referred to as vasodilatory or maldistributive shock. In vasodilatory shock, there is excessive dilation of blood vessels, leading to a significant decrease in blood pressure and inadequate perfusion of vital organs.[80,81] Common clinical conditions that can lead to vasodilation include acute pancreatitis, anaphylaxis, sepsis, trauma, and parvoviral enteritis. Historically, vasodilatory shock treatment has revolved around rapid administration of fluids, the administration of broad-spectrum antibiotics, establishing source control, and using pharmacologic interventions to maintain adequate mean arterial blood pressure.[80,82]

Because distinguishing between hypovolemic and vasodilatory shock based solely on physical examination findings is challenging, and because patients with vasodilatory shock may have some degree of hypovolemia, a fluid challenge is a reasonable treatment approach in these situations. Poor or limited response to fluid boluses may suggest vasodilation. In cases in which there is a poor response to fluid therapy, vasopressor therapy should be considered.[82] In patients with suspected anaphylaxis, it is crucial to administer epinephrine, a vasopressor, as soon as possible.[83]

Thermoregulation Disturbances

Hypothermia. Whenever possible, use warm fluids for resuscitation or rehydration in hypothermic patients for active core rewarming. The task force believes warm fluids will at least mitigate worsening hypothermia compared with the administration of room temperature fluids. It has been recommended to warm IV fluids to 40–42°C (104–107.6°F).[84] Fluids can be warmed using numerous methods, including immersion of IV tubing in warm water, microwaving of the fluid bag, in-line fluid warmers, and prewarming of fluids in a convection oven.[85] However, the effect of warm fluids in producing an increase in body temperature is controversial.[86–88]

TABLE 14

How to Formulate Dextrose-Containing Fluids

Amount of 50% Dextrose Solution Added to 1L Bag of Isotonic Crystalloids*	Final Dextrose Concentration
25 mL	1.25%
50 mL	2.5%
100 mL	5%

*Remove an equivalent amount of the isotonic crystalloid fluid from the bag before adding dextrose.

Hypothermia is commonly observed in cats in shock. Aggressive fluid resuscitation with the aim of restoring normal blood pressure in hypotensive, hypothermic cats will often lead to interstitial fluid overload, pulmonary edema, and pleural effusion. Administering conservative fluid volumes (e.g., 5 mL/kg IV boluses at a time) concurrent with active rewarming is an essential part of shock resuscitation therapy in cats.[89]

Hyperthermia. Fluid therapy is important in treating hyperthermia in dogs and cats. Administering room temperature fluids may assist with cooling and maximize volume expansion.[90]

Hypoglycemia

Administer dextrose to patients exhibiting clinical signs related to hypoglycemia, such as lethargy, weakness, ataxia, or seizures. For a bolus, use 0.5–1 mL/kg (0.25–0.5 g/kg) of 50% dextrose, which should be diluted at a ratio of 1:2–1:4 and administered over 2–5 min.[91] In most cases, the bolus should be followed by a CRI of 1.25–5% dextrose (Table 14) until the patient can maintain normoglycemia. The concentration of dextrose instilled in fluids depends on the severity of the patient's hypoglycemia and fluid administration rate. It is advisable to administer dextrose concentrations exceeding 5% through a central line to avoid phlebitis.

Note that in patients with insulinoma, dextrose administration may perpetuate hypoglycemia. Therefore, dextrose should only be given when patients present with clinical indications of hypoglycemia, and the lowest effective concentration should be used to prevent the manifestation of clinical signs.[92]

Section 6: Fluid Overload

Top Three Takeaways

1. First, do no harm. Fluid overload is a potentially life-threatening complication for which no universally effective therapy exists.
2. Excessive iatrogenic fluid administration is the most common cause of fluid overload. Prevention and vigilant monitoring are crucial to mitigate the potential risks associated with fluid therapy.
3. Patients with impaired renal function are at an increased risk of fluid overload because of their kidneys' inability to increase urine output and effectively excrete excess fluid.

Overview

Fluid overload, or fluid intolerance, is a clinical spectrum that spans from hypervolemia to life-threatening edema and cavitary effusions. The guidelines task force has proposed that fluid intolerance may be the more appropriate term for this condition, as this term more accurately describes how the amount of fluid needed to overload a patient is dependent on their tolerance for a given amount of fluids. "Fluid intolerance" also encompasses both iatrogenic overload and overload due to underlying comorbidities. However, given that "fluid overload" is still widely used and recognized within the veterinary medical profession, it will be the primary term used in these guidelines to refer to this condition.

All patients receiving fluid therapy are susceptible to fluid intolerance/fluid overload; however, *excessive administration is the most common cause of fluid overload*. Hypervolemia might be more common than perceived, as early signs of increased body weight and positive fluid balance are difficult to assess. Typically, hypervolemia is recognized when advanced signs such as edema and effusions occur.[93] It is important to understand the harms of fluid overload and the need to prevent this complication, given the limited effectiveness of treatments.

Recognizing and Managing Fluid Overload

As discussed earlier, fluid movement is influenced by hydrostatic pressure, oncotic pressure, and vascular permeability. Patients who receive excess fluids during resuscitation, maintenance, or anesthesia first develop hypervolemia, which causes an increase in hydrostatic pressure. A decrease in oncotic pressure can occur with dilution of plasma proteins associated with excessive fluid administration or protein-losing disease. Hypervolemia, as well as inflammation and injury, can damage the glycocalyx and increase vascular permeability. Together, these changes in fluid dynamics promote movement of fluid out of vessels into the interstitial space. As the interstitial space expands, it becomes more compliant, accommodating larger quantities of interstitial fluid, further exacerbating the problem of fluid overload.

Changes in fluid dynamics favoring accumulation of interstitial fluid are often seen in combination with the body's physiological response to stress. Hypovolemia, illness, and injury lead to water and sodium retention and increased thirst. This response exacerbates the risk of fluid overload as resuscitation fluids are administered to critically ill patients when their bodies are primed to retain fluid.[94]

By the time clinical edema or effusions are noted, internal edema will also be present that can negatively impact organ function (Figure 11).[94] Once edema has occurred, it is very difficult to reverse even with ultrafiltration. Thus, prevention and early recognition are keys to minimizing the morbidity and mortality associated with fluid overload.

Although all patients receiving IV fluids are at risk of fluid overload, those with impaired renal function, heart disease, or liver disease or those receiving large fluid volumes are at highest risk (Box 5). Pets with oligo-anuric renal failure are particularly susceptible to fluid overload because they are unable to excrete excess fluid. Yet these patients often receive aggressive fluid therapy necessitating close monitoring for weight gain, hypertension (in presence of AKI/CKD), or early signs of edema.[93,94,95]

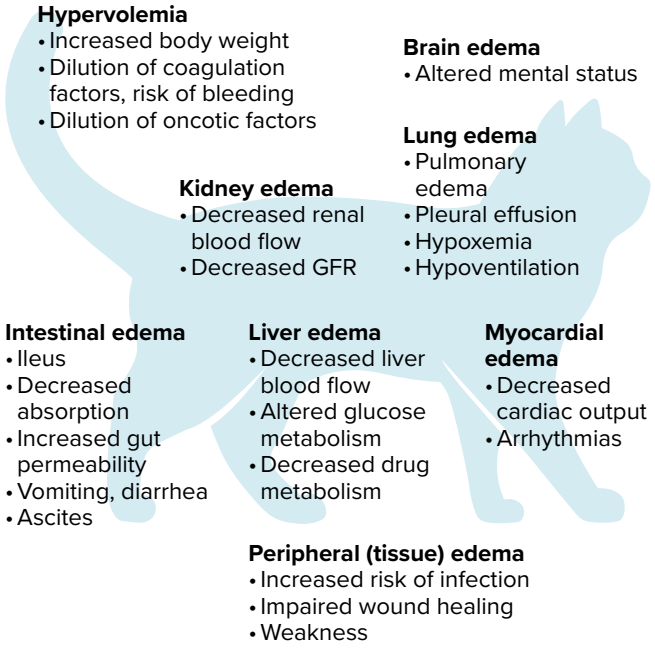

FIGURE 11

Impact of Fluid Overload on Organ Function

Box 5: Common Fluid Overload Case Scenarios

1. ***Continued IV fluid therapy in a feline patient with renal disease***. A cat is dehydrated, uremic, and anuric on presentation. Although IV fluid administration improves hydration status, uremia persists. IV fluids are continued with the misguided goal of improving GFR. However, no effective increase in GFR will occur no matter how much fluid this patient receives.
2. ***IV fluid therapy in a patient anesthetized for a lengthy procedure***. A dog receives 10 mL/kg of fluids throughout a 6 hr procedure and develops respiratory distress during anesthetic recovery.
3. ***SC fluid therapy in a cat with occult or fulminant heart disease***. A cat presents for evaluation of vomiting associated with malaise of congestive heart failure and is given SC fluids despite no evidence of dehydration.

TABLE 15

Clinical, Radiographic, and Ultrasonographic Findings Associated with Fluid Overload

Clinical Findings	Radiographic Findings	Ultrasonographic Findings
• Increased body weight (>10%) • Tissue edema (intermandibular area, limbs, paws, dependent regions, chemosis) • Serous nasal discharge • Serous discharge from endotracheal tube in anesthetized patients • Increased respiratory rate or effort • Reduced SPO_2 • Novel murmur, novel gallop sound • Gastrointestinal signs (abdominal distention, vomiting, diarrhea, inappetence, anorexia) • No change in blood pressure; hypertension rarely associated with fluid overload except in AKI/CKD.[1,2]	• Body wall edema • Pleural effusion • Pulmonary edema • Cardiomegaly • Enlarged pulmonary artery • Enlarged caudal vena cava • Enlarged pulmonary vein • Loss of serosal detail • Distended intestines	• Subcutaneous edema • Pleural effusion • B-lines • Enlarged La:Ao • Enlarged caudal vena cava • Decreased caudal vena cava collapsibility index • Ascites • Intestinal wall thickening • Ileus • Hyperechoic mesentery and pancreas • Hepatic congestion • Gallbladder wall edema • Perirenal edema

AKI, acute kidney injury; Ao, aorta; CKD, chronic kidney disease; La, Left atrium; SpO2, oxygen saturation

1. Cole LP, Jepson R, Dawson C, Humm K. Hypertension, retinopathy, and acute kidney injury in dogs: A prospective study [published correction appears in *J Vet Intern Med.* 2020 Nov;34(6):3168]. *J Vet Intern Med.* 2020;34(5):1940-1947.
2. Park S, Lee CJ, Lee M, et al. Differential effects of arterial stiffness and fluid overload on blood pressure according to renal function in patients at risk for cardiovascular disease. *Hypertens Res.* 2019;42:341–353.

Box 6: Common Misconceptions That May Lead to Fluid Overload

Misconception: IV fluids increase GFR in normally hydrated patients.
Correction: IV fluid administration will not increase GFR in a patient that is hydrated and euvolemic.

Misconception: Hypertension can be used to diagnose hypervolemia.
Correction: Hypertension is rarely associated with hypervolemia or edema. Some patients with renal disease may develop hypertension concomitant with hypervolemia.

Misconception: If a patient is hypotensive, keep giving fluid boluses until their blood pressure improves.
Correction: Some hypotensive patients will not respond to IV fluid therapy. If a patient is not responding to fluid therapy, discontinue fluid boluses and correct any electrolyte or glucose disturbances and consider vasopressor treatment.

Misconception: Hemodialysis is always a backup option for treatment of fluid overload if diuretics do not work.
Correction: Hemodialysis has a very limited therapeutic role in treating edema because the movement of edema fluid into the vascular space is limited.

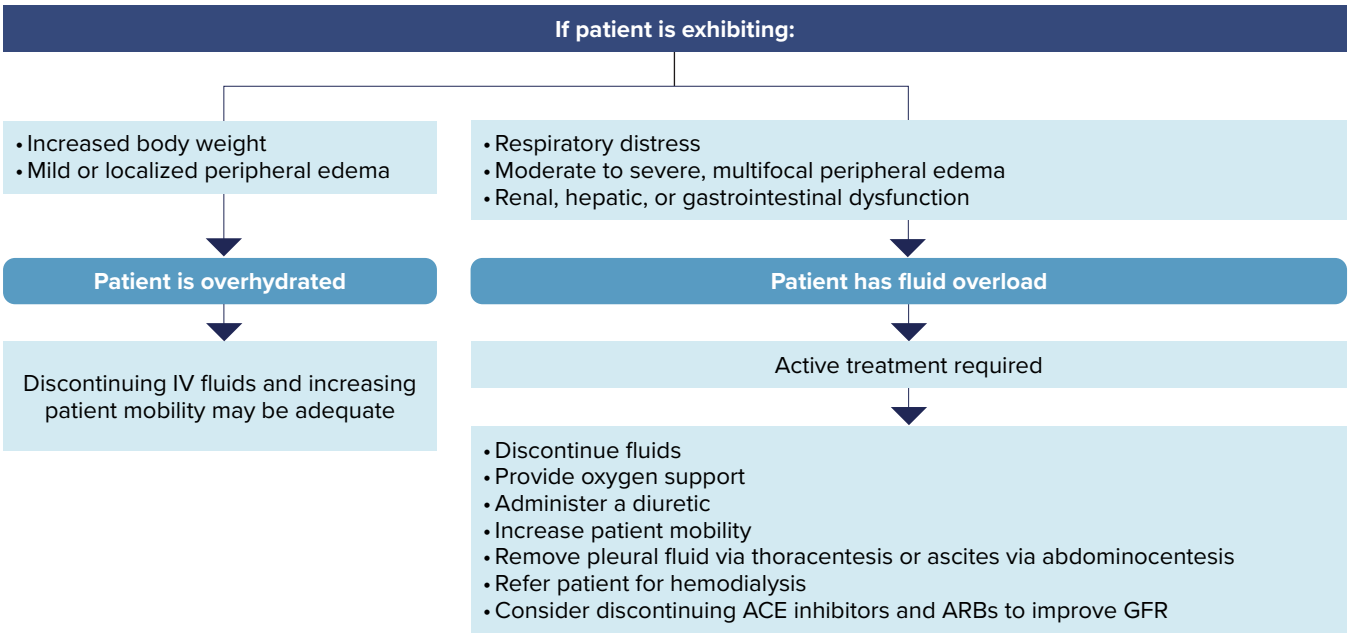

FIGURE 12

Fluid Overload Therapy Algorithm

ACE, angiotensin-converting enzyme; ARBs, angiotensin receptor blockers; GFR, glomerular filtration rate

To reduce the risk of fluid overload:

- Use a low-volume strategy for fluid resuscitation.
- Tailor ongoing fluid therapy to meet each patient's needs.
- Include all fluids delivered as medications, infusions, flushes, and enteral feedings to improve the accuracy of fluid balance calculations. Cats and small dogs with smaller blood volumes can quickly become fluid overloaded if clinicians are not mindful of the volumes administered.
- Limit the total fluid volume delivered in anesthetized patients during lengthy procedures. As a general rule, patients receiving anesthetic fluids should receive no more than the daily recommended total of 20 mL/kg/24 hr.[94]
- Consider enteral water supplementation.

To recognize hypervolemia sooner, weigh patients frequently (every 6–12 hr) to identify a 10% increase in body weight compared with the initial baseline.[93,94] Regularly document all fluid inputs and outputs to help identify positive fluid balance. Additional signs of fluid overload are summarized in Table 15.

Once edema has started, it can become life-threatening and be very difficult to resolve. See Box 6 for common misconceptions that can lead to fluid overload. **There is no definitive therapy.** General therapeutic approaches include sodium and water restriction, discontinuing IV fluids, administering diuretics, and increasing patient mobility (Figure 12). The interventions and success of those interventions are limited. Thus, the best approach is to avoid fluid overload.[93,94]

Section 7: Questions and Controversies in Fluid Therapy

Top Three Takeaways

1. Fluids are drugs with complex interactions in the body and require an understanding of how and when to prescribe them. Clinicians must carefully consider hydration and volume status, electrolyte and acid-base derangements, and underlying comorbidities when prescribing fluid therapy.
2. Questions concerning the safety of synthetic colloids, the efficacy of plasma to treat hypoalbuminemia, whether potassium-containing fluids are safe in treating hyperkalemic patients, and whether IV fluids are indicated during cardiopulmonary resuscitation (CPR) are answered here.
3. The RECOVER Advanced Life Support guidelines for veterinary CPR advocate a careful approach to the fluid prescription in patients with cardiopulmonary arrest, owing to the possibility of reducing oxygen delivery to myocardial and cerebral tissues in patients without hypovolemia or distributive shock.

Overview

The scarcity of large-scale, blinded, prospective studies on fluid therapy in veterinary medicine gives rise to questions and controversies regarding its application in practice. Clinical decisions often must be extrapolated from the existing limited data, further contributing to the ongoing discussions and uncertainties surrounding fluid therapy.

Are Synthetic Colloids Safe?

Colloids rapidly expand the intravascular space and have a longer duration of action than crystalloids. Their use has long been considered "volume-sparing" as compared with the volume of crystalloids required to match their effect. Synthetic colloids (e.g., hydroxyethyl starches [most common], gelatins, and dextrans) are more readily available and less costly than natural colloid alternatives such as blood products or species-specific albumin. Despite their initial attractiveness as resuscitation fluids, the use of synthetic colloids in critically ill small animal patients is currently controversial because of safety and efficacy concerns.[96–100] Complications associated with their use in human patients include the development of AKI and coagulopathy. Colloids are dissolved in NaCl, which delivers a high chloride load to the distal tubule and accounts for some of the renal effects seen in humans. There is limited evidence that these same complications may occur in critically ill dogs and cats, although most studies are retrospective evaluations or small experimental studies.[101–120] A newer understanding of the function of the endothelial surface layer[121] and revision of the Starling hypothesis[122,123] have raised concerns that extravasation of these synthetic macromolecules increases tissue edema and ultimately reduces oxygen delivery in states of increased vascular permeability, such as occurs with systemic inflammatory response syndrome, sepsis, trauma, and other damage to the endothelial surface layer.[124]

There is no evidence-based consensus on the optimal use of synthetic colloids in small animals. Very little evidence exists on whether one synthetic colloid is safer or more effective than another. Clinicians must carefully weigh risk versus benefit for individual patients.

Consider the following before administering synthetic colloids:[100]

- Avoid synthetic colloids if the patient is responsive to crystalloid or alternative therapies.
- Synthetic colloids should not be used as a substitute for albumin. In cases of severe hypoalbuminemia, natural colloids like plasma products or albumin should be considered.
- In complex disease processes, such as sepsis or septic shock, treatment is multimodal and may involve natural colloids and/or vasopressors.
- Current evidence suggests a minimal risk of synthetic colloid-associated AKI in dogs and cats when administered in small doses (<20 mL/kg) for short durations.
- Administering synthetic colloids to hemodynamically stable patients and those with pre-existing azotemia is not advised.
- Synthetic colloids may be considered for short-term need for colloid osmotic pressure support (e.g., during anesthesia for a portosystemic shunt ligation in a patient with hypoalbuminemia).

Current recommendations for synthetic colloid administration include minimizing dose and duration while closely monitoring kidney function (up to 90 days after exposure), coagulation parameters, hematocrit, and platelet count.[100] Synthetic colloids should be avoided or used with caution in patients with pre-existing azotemia, coagulopathy, anemia, and thrombocytopenia.[100] Some veterinary clinicians also consider systemic hypertension and sepsis to be contraindications for synthetic colloid use.[99]

Can Plasma Be Used to Address Hypoalbuminemia?

A serum albumin concentration of <2.0 g/dL is a negative prognostic indicator associated with increased patient morbidity and mortality in small animals due to development of peripheral edema, impaired tissue perfusion and oxygenation, hypercoagulability, altered protein-bound drug metabolism, and poor wound healing. In general, natural colloids (e.g., albumin and plasma) are preferred over synthetic colloids to address hypoalbuminemia, but they remain controversial because of a lack of evidence linking correction of the albumin concentration with improved outcomes in critically ill veterinary patients[56,125,126] Species-specific albumin products are the recommended transfusion products in veterinary patients with severe or worsening hypoalbuminemia.[61,125] Because of the limited availability and cost of species-specific albumin, plasma products (e.g., fresh frozen plasma, frozen plasma, and CPP) are sometimes used to treat hypoalbuminemia in dogs and cats.[127] Although plasma products contain varying amounts of albumin,[59] complications of plasma transfusions appear to be less risky than xenotransfusion with human albumin products.[125,128–131] The volume requirement and cost of plasma to raise serum albumin concentration is a serious limitation in its use for treating hypoalbuminemia. For many patients, nutritional support is more effective, less challenging, less risky, and less costly than attempting to increase serum albumin directly with natural colloids.[132,133]

Are Potassium-Containing "Balanced" Isotonic Crystalloids Harmful in Hyperkalemic Patients?

Hyperkalemia is a common electrolyte disturbance in small animal patients with conditions such as urethral or bilateral ureteral obstruction, ruptured bladder, hypoadrenocorticism (dogs), anuric or oliguric renal failure, and others.[134] Many of these patients require fluid resuscitation, and clinicians must choose between administering potassium-free fluid (e.g., 0.9% NaCl) or balanced isotonic electrolyte solution (e.g., lactated Ringer's solution, Normosol-R, and Plasma-Lyte A).

Balanced isotonic electrolyte solutions for resuscitation contain only a small amount of potassium (4–5 mEq/L) (Table 8). Several studies have concluded that potassium-containing isotonic fluids are not detrimental for fluid resuscitation and rehydration in hyperkalemic cats with urethral obstruction.[135,136] Balanced isotonic fluids correct acid-base imbalances faster than an acidifying fluid such as 0.9% NaCl. Balanced isotonic crystalloids also help prevent excessively rapid sodium correction in hyponatremic patients who have hyperkalemia (e.g., Addisonian crisis).[137] It is key to identify and treat the underlying cause of hyperkalemia. Further management of severe

hyperkalemia is indicated to prevent cardiac conduction disturbances and other life-threatening complications when serum potassium is >7 mmol/L.[138]

Are IV Fluids Indicated During CPR?

Fluid therapy is indicated during CPR if it will help mitigate hypovolemia that led to the cardiopulmonary arrest. IV fluid therapy in the form of crystalloid boluses increases preload and cardiac input in small animal patients with hypovolemia or inappropriate vasodilation (e.g., anaphylaxis, sepsis, and systemic inflammatory response syndrome). Increasing the intravascular volume in euvolemic patients, however, may ultimately decrease perfusion pressures and reduce oxygen delivery to myocardial and cerebral tissues.[139–142] For this reason, the RECOVER Advanced Life Support guidelines for veterinary CPR advocate a careful approach to the fluid prescription in patients with cardiopulmonary arrest. These guidelines recommend fluids for specific indications only, such as patients with or likely to have hypovolemia or distributive shock.[143,144] Experimental studies in rats and pigs suggest there may be a neuroprotective effect associated with hypertonic saline administered during CPR,[145–147] but clinical evidence in dogs and cats with cardiopulmonary arrest is lacking. Synthetic colloids should be avoided in these patients because of potential risks and the absence of a survival benefit to justify the risks.[144] Patients with significant hemorrhage may require blood products. Patients with derangements in potassium, calcium, magnesium, glucose, or acid-base status may benefit from directed therapy to help correct life-threatening abnormalities. Patients with acute lipid-soluble toxicoses may benefit from IV lipid emulsion therapy.

Section 8: Fluid Administration and Monitoring

Top Three Takeaways

1. Using skilled veterinary technicians to administer and monitor fluid therapy allows the veterinarian to focus on the tasks they alone must perform (i.e., diagnosing, prescribing—including the fluid therapy prescription—prognosing, and surgery), leading to increased efficiency and oversight of patients.
2. Monitoring during IV fluid therapy is critical to prevent fluid overload. When patient monitoring or referral to a hospital that provides monitoring is not available, consider an alternate route of delivery.
3. Infusion pumps are frequently used; however, they may not always be the optimal method for fluid administration.

Overview

IV fluids can be administered in a variety of ways. The veterinary nursing care team must be trained in how to use each method to avoid fluid overload in patients and related potential hazards. These factors help determine the best mode for IV fluid delivery:

- Volume to be infused
- Total duration of infusion
- Level of monitoring available during administration
- Type of fluid administered

Fluid Administration

IV infusion pumps are most commonly used but are not always the ideal mode of administration. For example, a large dog who needs a rapid crystalloid infusion may need an infusion rate that exceeds the 1–2 L/hr that standard pumps provide. In this case, a pressure bag would be the preferred method to deliver the large fluid volume in a short time. For animals receiving a small volume of fluids, a syringe pump may be more accurate than a standard infusion pump that does not have micro settings (Table 16).

The IV administration set is another consideration in choosing equipment. Smaller animals may benefit from a micro drip set (60 drops/mL), which provides more precise fluid delivery. This can be especially helpful when using the gravity method to deliver fluids in small patients. Macro drip sets come in a variety of drip factors; those most used in veterinary medicine are 10 drops/mL and 15 drops/mL. Fluid pumps are also usually designed for use with a specific type or size of tubing, so it is important to match these to ensure adequate fluid delivery (Table 16).

Use a new IV line and bag for each patient[148] (see Table 17 for IV catheter care and placement). Prime IV lines with the fluid to be infused before use to avoid air embolism. A T-port can ease medication administration, and a Y-port can be used when administering more than one compatible infusion.

IV fluid bags should not be used for saline flushes because of the risk of contamination. Commercially prefilled syringes are preferred to decrease the chances of introducing bacterial contamination that could lead to infection.[149–151]

If IV access is not achievable, consider IO catheters early in treatment to avoid fluid resuscitation delays. IO devices are able to quickly drill through the bone but require training. When no device is available, needles or peripheral IV catheters can be used (see https://www.youtube.com/watch?v=10twNYP1pB0 for instructions on placing an IO catheter).

Fluid Monitoring

Trained nursing staff should continually monitor IV fluid administration (Table 18). Without adequate monitoring, severe consequences can occur and compromise patient care. Use multiparameter monitoring to gain a broad picture of the patient's volume and hydration status and avoid fluid-related complications (Table 19).

Monitor patients at high risk for fluid overload at least every 2 hr (see Tables 19 and 20 for methods). Weigh these patients 2–3 times a day, check their respiratory rate every 1–2 hr, measure fluid

TABLE 16

Intravenous Fluid Delivery Modes

Method of Delivery	Considerations
Fluid pump	• Limits of very high and, possibly, very low rates of administration • Maximum administration rate can limit ability to rapidly deliver a bolus with large fluid volumes
Syringe pump	• Limited to small volumes • Attach the extension set close to the IV catheter to ensure patient receives the infusion in a timely manner
Gravity drip set	• Need to calculate drip rate: Fluid rate (mL/h) × Drip factor (gtt/mL)/3600 = gtt/s • Patient movement or changes to bag placement can affect drip rate • Close monitoring is essential because there are no alarms
Buretrol	• Used in conjunction with a fluid pump • Prevents delivery of large fluid volume to small patients • Allows for smaller volumes of additives, leading to less waste in smaller patients or in cases of frequent changes to fluid plans
Syringe	• Hand administration of small volumes • Do not leave attached to a patient while unattended
Pressure bag	• Ideal when volume to be infused over given time exceeds the capabilities of a fluid pump and gravity set

gtt, drop; s, second

TABLE 17

Peripheral Intravenous Catheter Placement and Care Checklist

Large bore/ gauge, short cannula	• Larger gauge, short catheters provide less resistance to blood flow than longer, smaller gauge catheters.[1] Ideal to have in place should a need for rapid infusion arise.
Aseptic preparation and care	• See the *AAHA Infection Control, Prevention and Biosecurity Guidelines* protocol for IV catheters at www.aaha.org/resources/2018-aaha-infection-control-prevention-and-biosecurity-guidelines
Secure catheter	• Secure the first piece of tape to the catheter as an anchor. Use the smallest amount of tape possible and tab the tape ends for easy removal. • Use additional bandage material as needed. Be careful not to secure too tight or too loose to avoid swelling or premature dislodgement.
Daily maintenance	• Check catheters at least two times per day. Fully unwrap bandage material covering the tape to examine the catheter site for signs of swelling or thrombophlebitis. Remove the catheter and place another one if indicated. • Evidence in human patients shows that routine catheter replacement does not provide any benefit over replacing peripheral catheters when clinically indicated.[2]
Clean ports when disconnecting	• Wipe ports with isopropyl alcohol. • Needleless injection and connection ports are preferred.

1. Reddick AD, et al. Intravenous fluid resuscitation: was Poiseuille right? *Emerg Med J.* 2011;28(3):201-2.
2. Webster J, Osborne S, Rickard CM, Marsh N. Clinically-indicated replacement versus routine replacement of peripheral venous catheters. *Cochrane Database Syst Rev.* 2019;1(1):CD007798.

TABLE 18

Monitoring Fluid Delivery

Method of Delivery	Monitoring
Fluid pump Buretrol	• Set TVI to 0 at start. Document every 2–4 hours. • Set VTBI for time frame between checks.
Syringe pump	• Set TVI to 0 at start. Document every 2–4 hours. • Set VTBI for time frame between checks.
Gravity drip set Pressure bag	• Mark top of fluids on bag at start and document every 1–2 hours. • Monitor more frequently due to a higher risk of changes in volume delivered (e.g., every five minutes for volumes delivered over 15-20 minutes).
Syringe	• Use only for bolus administration. Do not leave attached to patient.

TVI, total volume infused; VTBI, volume to be infused

TABLE 19

Evaluation and Monitoring Parameters That May Be Used for Patients Receiving Fluid Therapy[a]

- Pulse rate and quality*
- Capillary refill time
- Mucous membrane color
- Respiratory rate and effort
- Lung sounds
- Skin turgor
- Body weight
- Urine output
- Mental status
- Extremity temperature
- Packed cell volume/total solids
- Total protein
- Serum lactate
- Urine specific gravity
- Blood urea nitrogen
- Creatinine
- Electrolytes
- Blood pressure
- Venous or arterial blood gasses
- O_2 saturation

[a]Reprinted from Davis H, Jensen T, Johnson A, et al. 2013 AAHA/AAFP fluid therapy guidelines for dogs and cats. *J Am Anim Hosp Assoc.* 2013;49(3):149-59.

*Including cardiac auscultation to identify new murmurs

inputs and urine output, and watch for signs of edema. If available, perform focused ultrasonography for fluid responsiveness, which includes scanning for cavity effusion and B-lines in the lungs and evaluating the vena cava. For patients at risk of recurrence of hypovolemic shock, evaluating the inferior vena cava may be a better indicator of recurrence than other more commonly monitored parameters such as heart rate and blood pressure.[152]

In smaller patients, especially pediatrics, keep in mind that serial blood draws can decrease blood volume.

Fully utilizing veterinary technicians, who can not only set up and run fluid therapy but also identify the signs of fluid-related complications, allows the veterinarian to delegate this nursing task after prescribing fluid therapy orders. The stronger a veterinary technician's understanding of the "why" of fluid therapy, something that is addressed in veterinary technology and nursing programs, the more they can be optimized to their fullest potential, allowing for higher efficiency of the veterinary team.

Many veterinary practices are either unable to provide 24 hr care or geographically unable to refer patients to a 24 hr facility. When continual patient monitoring is not possible, the task force recommends giving IV fluids during the clinic's open hours and SC fluids before leaving for the night. A slightly higher IV fluid rate can be used during the day unless contraindicated because of the patient's disease state. Patient monitoring must be provided whenever administering IV fluids.

TABLE 20

Methods to Monitor Fluid Outputs and Inputs

Urine output • Urinary catheter with a closed collection system • Absorbent training pads • Non absorbent litter • Free-catch urine collection during walks
Vomit and diarrhea volume estimates
Feeding tube • Amount delivered • Amount of residual content in stomach
Body weight
Drain output

Section 9: Conclusion

Fluid administration is a cornerstone of therapy for many canine and feline patients. Patients who need fluid supplementation require a personalized fluid therapy plan that ensures administration of the proper fluid at the appropriate rate and volume. Relying on a "one-rate-fits-most-patients" strategy in fluid therapy calculations is not only impractical but also potentially harmful. Understanding the differences between fluid types and their effects within the body's fluid compartments is crucial, along with thoughtful, comprehensive patient assessment. Targeted fluid therapy prescriptions address each patient's resuscitation, rehydration, and maintenance requirements. Fluid therapy also involves anticipating the changes in electrolytes and other biologic parameters that will occur as a result of fluid therapy and supplementing fluids or otherwise altering the treatment plan to prevent adverse effects. Diligent patient monitoring is essential to gauge therapeutic response, indicating when changes to fluid therapy plans are needed to optimize patient outcomes.

REFERENCES

1. Waddell L. Colloid osmotic pressure and osmolality. In: Silverstein DC, Hopper K, eds. *Small Animal Critical Care Medicine*. 3rd ed. St. Louis: Elsevier; 2022:1054–58.
2. Woodcock TE, Michel CC. Advances in the Starling principle and microvascular fluid exchange; consequences and implications for fluid therapy. *Front Vet Sci* 2021;8:623671.
3. Boysen SR, Gommeren K. Assessment of volume status and fluid responsiveness in small animals. *Front Vet Sci* 2021;8:630643.
4. Rudloff E, Hopper K. Crystalloid and colloid compositions and their impact. *Front Vet Sci* 2021;8:639848.
5. Rudloff E. Assessment of hydration. In: Silverstein DC, Hopper K, eds. *Small Animal Critical Care Medicine*. 3rd ed. St. Louis: Elsevier; 2022: 373–77.
6. Marshall KA, Thomovsky EJ, Brooks AC, et al. Ultrasound measurements of the caudal vena cava before and after blood donation in 9 greyhound dogs. *Can Vet J* 2018;59(9):973-980.
7. Donati PA, Guevara JM, Ardiles V, et al. Caudal vena cava collapsibility index as a tool to predict fluid responsiveness in dogs. *J Vet Emerg Crit Care (San Antonio)* 2020;30(6):677–86.
8. Tello LH, Pardo MA. Fluid and electrolyte therapy during vomiting and diarrhea. *Vet Clin North Am Small Anim Pract* 2022;52(3):673–88.
9. Harris BA, Hofmeister EH, Gicking JC. A survey of emergency and critical care veterinarians regarding IV fluid bolus therapy and monitoring practices in small animals. *J Vet Emerg Crit Care (San Antonio)* 2021;31(5):564–73.
10. DiBartola SP, Bateman S. Introduction to fluid therapy. In: DiBartola SP, ed. *Fluid, Electrolyte, and Acid-Base Disorders in Small Animal Practice*. 4th ed. St. Louis: Elsevier Saunders; 2012:331–50.
11. Hansen B, Vigani A. Maintenance fluid therapy: isotonic versus hypotonic solutions. *Vet Clin North Am Small Anim Pract* 2017;47(2):383–95.
12. Wellman ML, DiBartola SP, Kohn CW. Applied physiology of body fluids in dogs and cats. In: DiBartola SP, ed. *Fluid, Electrolyte, and Acid-Base Disorders in Small Animal Practice*. 4th ed. St. Louis: Elsevier Saunders; 2012:15.
13. Hopper K, Garcia Rojas A, Barter L. An online survey of small animal veterinarians regarding current fluid therapy practices in dogs and cats. *J Am Vet Med Assoc* 2018;252(5):553–9.
14. Mazzaferro E, Powell LL. Fluid therapy for the emergent small animal patient: crystalloids, colloids, and albumin products. *Vet Clin North Am Small Anim Pract* 2022;52(3):781–96.
15. Lange J, Boysen SR, Bentley A, et al. Intraosseous catheter flow rates and ease of placement at various sites in canine cadavers. *Front Vet Sci* 2019;(19):6:312.
16. Bednarski, RM. Dogs and cats. In: Tranquilli WJ, Thurmon JC, Grimm KA, eds. *Lumb & Jones Veterinary Anesthesia and Analgesia*. 4th ed. Hoboken (NJ): Blackwell Publishing; 2007:705–15.
17. Davis H, Jensen T, Johnson A, et al; American Association of Feline Practicioners; American Animal Hospital Association. 2013 AAHA/AAFP fluid therapy guidelines for dogs and cats. *J Am Anim Hosp Assoc* 2013;49(3):149–59.
18. Muir WW. A new way to monitor & individualize your fluid therapy plan. *Vet Med* 2013;108(2). Available at https://www.dvm360.com/view/new-way-monitor-and-individualize-your-fluid-therapy-plan. Accessed July 13, 2023.
19. Kudnig ST, Mama K. Perioperative fluid therapy. *J Am Vet Med Assoc* 2002;221:1112–21.
20. Gaynor JS, Dunlop CI, Wagner AE, et al. Complications and mortality associated with anesthesia in dogs and cats. *J Am Anim Hosp Assoc* 1999;35:13–7.
21. Redondo JI, Rubio M, Soler G, et al. Normal values and incidence of cardiorespiratory complications in dogs during general anaesthesia. A review of 1281 cases. *J Vet Med A Physiol Pathol Clin Med* 2007;54: 470–77.
22. Fantoni DT, Ida KK, Gimenes AM, et al. Pulse pressure variation as a guide for volume expansion in dogs undergoing orthopedic surgery. *Vet Anaesth Analg* 2017;44:710–8.
23. Celeita-Rodriguez N, Teixeira-Neto FJ, Garofalo NA, et al. Comparison of the diagnostic accuracy of dynamic and static preload indexes to predict fluid responsiveness in mechanically ventilated isoflurane anesthetized dogs. *Vet Anaesth Analg* 2019;46:276–88.
24. Klein AV, Teixeira-Neto FJ, Garofalo NA, et al. Changes in pulse pressure variation and plethysmographic variability index caused by hypotension-inducing hemorrhage followed by volume replacement in isoflurane-anesthetized dogs. *Am J Vet Res* 2016;77(3):280–7.
25. Skouropoulou D, Lacitignola L, Di Bella C, et al. Intraoperative assessment of fluid responsiveness in normotensive dogs under isoflurane anaesthesia. *Vet Sci* 2021;8(2):26.
26. Brunetto MA, Gomes MO, Andre MR, et al. Effects of nutritional support on hospital outcome in dogs and cats. *J Vet Emerg Crit Care (San Antonio)* 2010;20(2):224–31.
27. Kraft MD, Btaiche IF, Sacks GS. Review of the refeeding syndrome. *Nutr Clin Pract* 2005;20(6):625–33.
28. Brunet A, Bouzouraa T, Cadore JL, et al. Use of feeding tubes in 112 cats in an internal medicine referral service (2015–2020). *J Feline Med Surg* 2022;24(10):e338–46.
29. Cooper E. Routes of fluid administration. In: Cooper E, Guillaumin J, Yaxley P, et al. *Small Animal Fluid Therapy*. CABI; 2023:33–40.
30. Kathrani A. Enteral feeding tubes. In: Aronson LR, ed. *Small Animal Surgical Emergencies*. 2nd ed. Hoboken (NJ): Wiley Blackwell; 2022: 251–61.
31. Tolbert MK, Telles NJ, Simon BT, et al. Gastrointestinal transit time is faster in Beagle dogs compared to cats. *J Am Vet Med Assoc*. 2022;260(S3):S8–14.

32. Becvarova I. Tube feeding in small animals: diet selection and preparation. In: Chan DL, ed. *Nutritional Management of Hospitalized Small Animals*. Hoboken (NJ): John Wiley & Sons; 2015:80–91.
33. Chan DL. Nutritional support of the critically ill small animal patient. *Vet Clin North Am Small Anim Pract* 2020;50(6):1411–22.
34. Grathwohl KW, Bruns BJ, LeBrun CJ, et al. Does hemodilution exist? Effects of saline infusion on hematologic parameters in euvolemic subjects. *South Med J* 1996;89(1):51–5.
35. Prittie JE. Triggers for use, optimal dosing, and problems associated with red cell transfusions. *Vet Clin North Am Small Anim Pract* 2003; 33(6):1261–75.
36. Kisielewicz C, Self I, Bell R. Assessment of clinical and laboratory variables as a guide to packed red blood cell transfusion of euvolemic anemic dogs. *J Vet Intern Med* 2014;28(2):576–82.
37. Langston C, Gordon D. Effects of IV fluids in dogs and cats with kidney failure. *Front Vet Sci* 2021;8:659960.
38. Langston C. Managing fluid and electrolyte disorders in renal failure. *Vet Clin North Am Small Anim Pract* 2008;38(3):677–97, xiii.
39. Harison E, Langston C, Palma D, Lamb K. Acute azotemia as a predictor of mortality in dogs and cats. *J Vet Intern Med* 2012;26(5):1093–8.
40. Von Hendy-Willson VE, Pressler BM. An overview of glomerular filtration rate testing in dogs and cats. *Vet J* 2011;188(2):156–65.
41. Fiocchi EH, Cowgill LD, Brown DC, et al. The use of darbepoetin to stimulate erythropoiesis in the treatment of anemia of chronic kidney disease in dogs. *J Vet Intern Med* 2017;31(2):476–85.
42. Chalhoub S, Langston CE, Farrelly J. The use of darbepoetin to stimulate erythropoiesis in anemia of chronic kidney disease in cats: 25 cases. *J Vet Intern Med* 2012;26(2):363–9.
43. Gori E, Lippi I, Guidi G, et al. Acute pancreatitis and acute kidney injury in dogs. *Vet J* 2019;245:77–81.
44. Quimby J. Management of chronic kidney disease. In: Bruyette DS, Bexfield N, Chretin JD, et al., eds. *Clinical Small Animal Internal Medicine*. Hoboken (NJ): Wiley Blackwell; 2020:1165–73.
45. Perkowski SZ, Oyama MA. Pathophysiology and anesthetic management of patients with cardiovascular disease. In: Grimm KA, Lamont LA, Tranquilli WJ, et al., eds. *Veterinary Anesthesia and Analgesia: The Fifth Edition of Lumb and Jones*. Hoboken (NJ): Wiley Blackwell; 2015: 496–510.
46. Keene BW, Atkins CE, Bonagura JD, et al. ACVIM consensus guidelines for the diagnosis and treatment of myxomatous mitral valve disease in dogs. *J Vet Intern Med* 2019;33(3):1127–40.
47. Ferasin L, DeFrancesco T. Management of acute heart failure in cats. *J Vet Cardiol* 2015;17 (suppl 1):S173–89.
48. Boysen SR, Gommeren K. Assessment of volume status and fluid responsiveness in small animals. *Front Vet Sci* 2021;8:630643.
49. Luis Fuentes V, Abbott J, Chetboul V, et al. ACVIM consensus statement guidelines for the classification, diagnosis, and management of cardiomyopathies in cats. *J Vet Intern Med* 2020;34(3):1062–77.
50. Nguyen VQ, Gadiraju TV, Patel H, et al. Intra-abdominal hypertension: an important consideration for diuretic resistance in acute decompensated heart failure. *Clin Cardiol* 2016;39(1):37–40.
51. Pouchelon JL, Atkins CE, Bussadori C, et al. Cardiovascular-renal axis disorders in the domestic dog and cat: a veterinary consensus statement. *J Small Anim Pract* 2015;56(9):537–52.
52. Ronco C, Haapio M, House AA, et al. Cardiorenal syndrome. *J Am Coll Cardiol* 2008;52(19):1527–39.
53. Giorgi ME, Mochel JP, Yuan L, et al. Retrospective evaluation of risk factors for development of kidney injury after parenteral furosemide treatment of left-sided congestive heart failure in dogs. *J Vet Intern Med* 2022;36(6):2042–52.
54. Lefebvre HP, Brown SA, Chetboul V, et al. Angiotensin-converting enzyme inhibitors in veterinary medicine. *Curr Pharm Des* 2007;13(13): 1347–61.
55. Hoorn EJ. Intravenous fluids: balancing solutions [published correction appears in *J Nephrol* 2020;33(2):387]. *J Nephrol* 2017;30(4):485–92.
56. Snow SJ, Ari Jutkowitz L, Brown AJ. Trends in plasma transfusion at a veterinary teaching hospital: 308 patients (1996-1998 and 2006-2008). *J Vet Emerg Crit Care (San Antonio)* 2010;20(4):441–5.
57. Beer KS, Silverstein DC. Controversies in the use of fresh frozen plasma in critically ill small animal patients. *J Vet Emerg Crit Care (San Antonio)* 2015;25(1):101–6.
58. Ropski MK, Guillaumin J, Monnig AA, et al. Use of cryopoor plasma for albumin replacement and continuous antimicrobial infusion for treatment of septic peritonitis in a dog. *J Vet Emerg Crit Care (San Antonio)* 2017;27(3):348–56.
59. Culler CA, Iazbik C, Guillaumin J. Comparison of albumin, colloid osmotic pressure, von Willebrand factor, and coagulation factors in canine cryopoor plasma, cryoprecipitate, and fresh frozen plasma. *J Vet Emerg Crit Care (San Antonio)* 2017;27(6):638–44.
60. Craft EM, Powell LL. The use of canine-specific albumin in dogs with septic peritonitis. *J Vet Emerg Crit Care (San Antonio)* 2012;22(6):631–9.
61. Sahagian MJ, Mastrocco A, Weltman JG, et al. Retrospective analysis of the use of canine-specific albumin in 125 critically ill dogs. *J Vet Emerg Crit Care (San Antonio)* 2023;33(2):192–200.
62. Carney N, Totten AM, O'Reilly C, et al. Guidelines for the management of severe traumatic brain injury, fourth edition. *Neurosurgery* 2017;80(1):6–15.
63. Leibner E, Andreae M, Galvagno SM, et al. Damage control resuscitation. *Clin Exp Emerg Med* 2020;7(1):5–13.
64. Pigott A, Rudloff E. Traumatic brain injury–a review of intravenous fluid therapy. *Front Vet Sci* 2021;8:643800.
65. Shi J, Tan L, Ye J, et al. Hypertonic saline and mannitol in patients with traumatic brain injury: A systematic and meta-analysis. *Medicine (Baltimore)* 2020;99(35):e21655.
66. Li M, Chen T, Cai J, et al. Comparison of equimolar doses of mannitol and hypertonic saline for the treatment of elevated intracranial pressure after traumatic brain injury: a systematic review and meta-analysis. *Medicine (Baltimore)* 2015;94(17):e668.
67. Gu J, Huang H, Huang Y, et al. Hypertonic saline or mannitol for treating elevated intracranial pressure in traumatic brain injury: a meta-analysis of randomized controlled trials. *Neurosurg Rev* 2019;42: 499–509.
68. Roquilly A, Moyer JD, Huet O, et al; Atlanrea Study Group and the Société Française d'Anesthésie Réanimation (SFAR) Research Network. Effect of continuous infusion of hypertonic saline vs standard care on 6-month neurological outcomes in patients with traumatic brain injury: the COBI randomized clinical trial. *JAMA* 2021;325(20):2056–66.
69. Kogika MM, de Morais HA. A quick reference on hypokalemia. *Vet Clin North Am Small Anim Pract* 2017;47(2):229–34.
70. Schaer M. Disorders of serum potassium, sodium, magnesium and chloride. *J Vet Emerg Crit Care (San Antonio)* 1999;9(4):209–17.
71. Hoehne SN, Hopper K, Epstein SE. Accuracy of potassium supplementation of fluids administered intravenously. *J Vet Intern Med* 2015;29(3):834-9.
72. Humphrey S, Kirby R, Rudloff E. Magnesium physiology and clinical therapy in veterinary critical care. *J Vet Emerg Crit Care (San Antonio)* 2015;25(2):210–25.

73. Sterns RH, Silver SM, Hix JK. Treatment of hyponatremia. In: Simon EE, ed. *Hyponatremia: Evaluation and Treatment.* New York: Springer; 2013:221–50.
74. Sahay M, Sahay R. Hyponatremia: A practical approach. *Indian J Endocrinol Metab* 2014;18(6):760–71.
75. Adrogué HJ, Tucker BM, Madias NE. Diagnosis and management of hyponatremia: a review. *JAMA* 2022;328(3):280–91.
76. O'Brien DP, Kroll RA, Johnson GC, et al. Myelinolysis after correction of hyponatremia in two dogs. *J Vet Intern Med* 1994;8(1):40–8.
77. Muhsin SA, Mount DB. Diagnosis and treatment of hypernatremia. *Best Pract Res Clin Endocrinol Metab* 2016;30(2):189–203.
78. Ostermann M, Randolph AG. Resuscitation fluid composition and acute kidney injury in critical illness. *N Engl J Med* 2022;386(9):888–9.
79. Sigmon J, May CC, Bryant A, et al. Assessment of acute kidney injury in neurologically injured patients receiving hypertonic sodium chloride: does chloride load matter? *Ann Pharmacother* 2020;54(6):541–6.
80. Chow JH, Abuelkasem E, Sankova S, et al. Reversal of vasodilatory shock: current perspectives on conventional, rescue, and emerging vasoactive agents for the treatment of shock. *Anesth Analg* 2020;130(1):15–30.
81. Landry DW, Oliver JA. The pathogenesis of vasodilatory shock. *N Engl J Med* 2001;345(8):588–95.
82. Wieruszewski PM, Khanna AK. Vasopressor choice and timing in vasodilatory shock. *Crit Care* 2022;26(1):76.
83. Brown JC, Simons E, Rudders SA. Epinephrine in the management of anaphylaxis. *J Allergy Clin Immunol Pract* 2020;8(4):1186–95.
84. Campbell G, Alderson P, Smith AF, et al. Warming of intravenous and irrigation fluids for preventing inadvertent perioperative hypothermia. *Cochrane Database Syst Rev* 2015;2015(4):CD009891.
85. Brodeur A, Wright A, Cortes Y. Hypothermia and targeted temperature management in cats and dogs. *J Vet Emerg Crit Care (San Antonio)* 2017;27(2):151–63.
86. Jourdan G, Didier C, Chotard E, et al. Heated intravenous fluids alone fail to prevent hypothermia in cats under general anaesthesia. *J Feline Med Surg* 2017;19(12):1249–53.
87. Chiang V, Hopper K, Mellema MS. In vitro evaluation of the efficacy of a veterinary dry heat fluid warmer. *J Vet Emerg Crit Care (San Antonio)* 2011;21(6):639–47.
88. Lee RA, Towle Millard HA, Weil AB, et al. In vitro evaluation of three intravenous fluid line warmers. *J Am Vet Med Assoc* 2014;244(12):1423–8.
89. Oncken AK, Kirby R, Rudloff E. Hypothermia in critically ill dogs and cats. *Compendium* 2001;23(6):506–21.
90. Johnson SI, McMichael M, White G. Heatstroke in small animal medicine: a clinical practice review. *J Vet Emerg Crit Care (San Antonio)* 2006;16(2):112–9.
91. Klein SC, Peterson ME. Canine hypoadrenocorticism: part II. *Can Vet J* 2010;51(2):179.
92. Goutal CM, Brugmann BL, Ryan KA. Insulinoma in dogs: a review. *J Am Anim Hosp Assoc* 2012;48(3):151–63.
93. Thomovsky E, Brooks A, Johnson P. Fluid overload in small animal patients. *Top Companion Anim Med* 2016;31(3):94–9.
94. Hansen B. Fluid overload. *Front Vet Sci* 2021;8:668688.
95. Londono L. Fluid therapy in critical care. *Todays Vet Pract* 2019;9(3). Available at: https://todaysveterinarypractice.com/internal-medicine/fluid-therapy-in-critical-care/. Accessed August 31, 2023.
96. Adamik KN, Yozova ID, Regenscheit N. Controversies in the use of hydroxyethyl starch solutions in small animal emergency and critical care. *J Vet Emerg Crit Care (San Antonio)* 2015;25(1):20–47.
97. Cazzolli D, Prittie J. The crystalloid-colloid debate: Consequences of resuscitation fluid selection in veterinary critical care. *J Vet Emerg Crit Care (San Antonio)* 2015;25(1):6–19.
98. Wong C, Koenig A. The colloid controversy: are colloids bad and what are the options? *Vet Clin North Am Small Anim Pract* 2017;47(2):411–21.
99. Yozova ID, Howard J, Sigrist NE, et al. Current trends in volume replacement therapy and the use of synthetic colloids in small animals–an internet-based survey (2016). *Front Vet Sci* 2017;4:140.
100. Adamik KN, Yozova ID. Colloids yes or no? - a "Gretchen question" answered. *Front Vet Sci* 2021;8:624049.
101. Hayes G, Benedicenti L, Mathews K. Retrospective cohort study on the incidence of acute kidney injury and death following hydroxyethyl starch (HES 10% 250/0.5/5:1) administration in dogs (2007-2010). *J Vet Emerg Crit Care (San Antonio)* 2016;26(1):35–40.
102. Yozova ID, Howard J, Adamik KN. Retrospective evaluation of the effects of administration of tetrastarch (hydroxyethyl starch 130/0.4) on plasma creatinine concentration in dogs (2010-2013): 201 dogs. *J Vet Emerg Crit Care (San Antonio)* 2016;26(4):568–77.
103. Yozova ID, Howard J, Adamik KN. Effect of tetrastarch (hydroxyethyl starch 130/0.4) on plasma creatinine concentration in cats: a retrospective analysis (2010-2015). *J Feline Med Surg* 2017;19(10):1073–9.
104. Sigrist NE, Kälin N, Dreyfus A. Changes in serum creatinine concentration and acute kidney injury (AKI) grade in dogs treated with hydroxyethyl starch 130/0.4 from 2013 to 2015. *J Vet Intern Med* 2017;31(2):434–41.
105. Sigrist NE, Kälin N, Dreyfus A. Effects of hydroxyethyl starch 130/0.4 on serum Creatinine concentration and development of acute kidney injury in nonazotemic cats. *J Vet Intern Med* 2017;31(6):1749–56.
106. Schmid SM, Cianciolo RE, Drobatz KJ, et al. Postmortem evaluation of renal tubular vacuolization in critically ill dogs. *J Vet Emerg Crit Care (San Antonio)* 2019;29(3):279–87.
107. Boyd CJ, Claus MA, Raisis AL, et al. Evaluation of biomarkers of kidney injury following 4% succinylated gelatin and 6% hydroxyethyl starch 130/0.4 administration in a canine hemorrhagic shock model. *J Vet Emerg Crit Care (San Antonio)* 2019;29(2):132–42.
108. Boyd CJ, Sharp CR, Claus MA, et al. Prospective randomized controlled blinded clinical trial evaluating biomarkers of acute kidney injury following 6% hydroxyethyl starch 130/0.4 or Hartmann's solution in dogs. *J Vet Emerg Crit Care (San Antonio)* 2021;31(3):306–14.
109. Adamik KN, Stoffel MH, Tangermann S, et al. Assessment of hydroxyethyl starch (6% HES 130/0.4) kidney storage in critically ill dogs: a post-mortem prospective study. *Front Vet Sci* 2022;8:802507.
110. Classen J, Adamik KN, Weber K, et al. In vitro effect of hydroxyethyl starch 130/0.42 on canine platelet function. *Am J Vet Res* 2012;73(12):1908–12.
111. Falco S, Bruno B, Maurella C, et al. In vitro evaluation of canine hemostasis following dilution with hydroxyethyl starch (130/0.4) via thromboelastometry. *J Vet Emerg Crit Care (San Antonio)* 2012;22(6):640–5.
112. Reuteler A, Axiak-Flammer S, Howard J, et al. Comparison of the effects of a balanced crystalloid-based and a saline-based tetrastarch solution on canine whole blood coagulation and platelet function. *J Vet Emerg Crit Care (San Antonio)* 2017;27(1):23–34.
113. Wurlod VA, Howard J, Francey T, et al. Comparison of the in vitro effects of saline, hypertonic hydroxyethyl starch, hypertonic saline, and two forms of hydroxyethyl starch on whole blood coagulation and platelet function in dogs. *J Vet Emerg Crit Care (San Antonio)* 2015;25(4):474–87.
114. Gauthier V, Holowaychuk MK, Kerr CL, et al. Effect of synthetic colloid administration on coagulation in healthy dogs and dogs with systemic inflammation. *J Vet Intern Med* 2015;29(1):276–85.

115. Albrecht NA, Howard J, Kovacevic A, et al. In vitro effects of 6% hydroxyethyl starch 130/0.42 solution on feline whole blood coagulation measured by rotational thromboelastometry. *BMC Vet Res* 2016;12(1):155.
116. Diniz MS, Teixeira-Neto FJ, Gonçalves DS, et al. Effects of 6% tetrastarch or lactated Ringer's solution on blood coagulation in hemorrhaged dogs [published correction appears in *J Vet Intern Med* 2019;33(2):1110]. *J Vet Intern Med* 2018;32(6):1927–33.
117. McBride D, Hosgood G, Raisis A, et al. Platelet closure time in anesthetized greyhounds with hemorrhagic shock treated with hydroxyethyl starch 130/0.4 or 0.9% sodium chloride infusions. *J Vet Emerg Crit Care (San Antonio)* 2016;26(4):509–15.
118. Boyd CJ, Claus MA, Raisis AL, et al. Hypocoagulability and platelet dysfunction are exacerbated by synthetic colloids in a canine hemorrhagic shock model [published correction appears in *Front Vet Sci* 2020;7:641]. *Front Vet Sci* 2018;5:279.
119. Iannucci C, Dirkmann D, Howard J, et al. A prospective randomized open-label trial on the comparative effects of 6% hydroxyethyl starch 130/0.4 versus polyionic isotonic crystalloids on coagulation parameters in dogs with spontaneous hemoperitoneum. *J Vet Emerg Crit Care (San Antonio)* 2021;31(1):32–42.
120. Smart L, Jandrey KE, Kass PH, et al. The effect of Hetastarch (670/0.75) in vivo on platelet closure time in the dog. *J Vet Emerg Crit Care (San Antonio)* 2009;19(5):444–9.
121. Gaudette S, Hughes D, Boller M. The endothelial glycocalyx: Structure and function in health and critical illness. *J Vet Emerg Crit Care (San Antonio)* 2020;30(2):117–34.
122. Michel CC. Starling: the formulation of his hypothesis of microvascular fluid exchange and its significance after 100 years. *Exp Physiol* 1997;82(1):1–30.
123. Levick JR. Revision of the Starling principle: new views of tissue fluid balance. *J Physiol* 2004;557(Pt 3):704.
124. Becker BF, Chappell D, Bruegger D, et al. Therapeutic strategies targeting the endothelial glycocalyx: acute deficits, but great potential. *Cardiovasc Res* 2010;87(2):300–10.
125. Mazzaferro EM, Edwards T. Update on albumin therapy in critical illness. *Vet Clin North Am Small Anim Pract* 2020;50(6):1289–305.
126. Montealegre F, Lyons BM. Fluid therapy in dogs and cats with sepsis. *Front Vet Sci* 2021;8:622127.
127. Culler CA, Balakrishnan A, Yaxley PE, et al. Clinical use of cryopoor plasma continuous rate infusion in critically ill, hypoalbuminemic dogs. *J Vet Emerg Crit Care (San Antonio)* 2019;29(3):314–20.
128. Francis AH, Martin LG, Haldorson GJ, et al. Adverse reactions suggestive of type III hypersensitivity in six healthy dogs given human albumin. *J Am Vet Med Assoc* 2007;230(6):873–9.
129. Cohn LA, Kerl ME, Lenox CE, et al. Response of healthy dogs to infusions of human serum albumin. *Am J Vet Res* 2007;68(6):657–63.
130. Trow AV, Rozanski EA, Delaforcade AM, et al. Evaluation of use of human albumin in critically ill dogs: 73 cases (2003-2006). *J Am Vet Med Assoc* 2008;233(4):607–12.
131. Horowitz FB, Read RL, Powell LL. A retrospective analysis of 25% human serum albumin supplementation in hypoalbuminemic dogs with septic peritonitis. *Can Vet J* 2015;56(6):591–7.
132. Liu DT, Brown DC, Silverstein DC. Early nutritional support is associated with decreased length of hospitalization in dogs with septic peritonitis: A retrospective study of 45 cases (2000-2009). *J Vet Emerg Crit Care (San Antonio)* 2012;22(4):453–9.
133. Conner BJ. Treating hypoalbuminemia. *Vet Clin North Am Small Anim Pract* 2017;47(2):451–9.
134. Kogika MM, de Morais HA. A quick reference on hyperkalemia. *Vet Clin North Am Small Anim Pract* 2017;47(2):223–8.
135. Drobatz K, Cole S. The influence of crystalloid type on acid-base and electrolyte status of cats with urethral obstruction. *J Vet Emerg Crit Care (San Antonio)* 2008;18(4):355–61.
136. Cunha MG, Freitas GC, Carregaro AB, et al. Renal and cardiorespiratory effects of treatment with lactated Ringer's solution or physiologic saline (0.9% NaCl) solution in cats with experimentally induced urethral obstruction. *Am J Vet Res* 2010;71(7):840–6.
137. Lathan P, Thompson AL. Management of hypoadrenocorticism (Addison's disease) in dogs. *Vet Med (Auckl)* 2018;9:1–10.
138. Jones JM, Burkitt-Creedon JM, Epstein SE. Treatment strategies for hyperkalemia secondary to urethral obstruction in 50 male cats: 2002-2017. *J Feline Med Surg* 2022;24(12):e580-e587.
139. Voorhees WD III, Ralston SH, Kougias C, et al. Fluid loading with whole blood or Ringer's lactate solution during CPR in dogs. *Resuscitation* 1987;15(2):113–23.
140. Ditchey RV, Lindenfeld J. Potential adverse effects of volume loading on perfusion of vital organs during closed-chest resuscitation. *Circulation* 1984;69(1):181–9.
141. Gentile NT, Martin GB, Appleton TJ, et al. Effects of arterial and venous volume infusion on coronary perfusion pressures during canine CPR. *Resuscitation* 1991;22(1):55–63.
142. Yannopoulos D, Zviman M, Castro V, et al. Intra-cardiopulmonary resuscitation hypothermia with and without volume loading in an ischemic model of cardiac arrest. *Circulation* 2009;120(14):1426–35.
143. Fletcher DJ, Boller M, Brainard BM, et al; American College of Veterinary Medicine; Veterinary Emergency and Critical Care Society. RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 7: Clinical guidelines. *J Vet Emerg Crit Care (San Antonio)* 2012;22(suppl 1):S102–31.
144. Fletcher DJ, Boller M. Fluid therapy during cardiopulmonary resuscitation. *Front Vet Sci* 2021;7:625361.
145. Fischer M, Hossmann KA. Volume expansion during cardiopulmonary resuscitation reduces cerebral no-reflow. *Resuscitation* 1996;32(3):227–40.
146. Zhou X, Liu Y, Huang Y, et al. Hypertonic saline infusion suppresses apoptosis of hippocampal cells in a rat model of cardiopulmonary resuscitation. *Sci Rep* 2017;7(1):5783.
147. Breil M, Krep H, Sinn D, et al. Hypertonic saline improves myocardial blood flow during CPR, but is not enhanced further by the addition of hydroxy ethyl starch. *Resuscitation* 2003;56(3):307–17.
148. O'Grady NP, Alexander M, Burns LA, et al; Healthcare Infection Control Practices Advisory Committee (HICPAC). Guidelines for the prevention of intravascular catheter-related infections. *Clin Infect Dis* 2011;52(9):e162–93.
149. Sabino CV, Weese JS. Contamination of multiple-dose vials in a veterinary hospital. *Can Vet J* 2006;47(8):779–82.
150. Gorski LA, Hadaway L, Hagle ME, et al. Infusion therapy standards of practice, eighth edition. *J Infus Nurs* 2021;44(1S suppl 1):S1–224.
151. Guillaumin J, Olp NM, Magnusson KD, et al. Influence of hang time and location on bacterial contamination of intravenous bags in a veterinary emergency and critical care setting. *J Vet Emerg Crit Care (San Antonio)* 2017;27(5):548–54.
152. Yanagawa Y, Sakamoto T, Okada Y. Hypovolemic shock evaluated by sonographic measurement of the inferior vena cava during resuscitation in trauma patients. *J Trauma* 2007;63(6):1245–8.