

# Fluid Therapy

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## OUTLINE

Body Fluid Balance, 52	Body Response to Hypovolemia, 59
Steady State and the Concept of Maintenance, 52	General Considerations for Fluid Therapy, 59
Body Fluid Compartments, 53	Fluid Types, 59
Perfusion, 55	Routes of Administration, 69
Salt Balance: Disorders of ECF Volume, 55	Fluid Therapy Plans and Monitoring, 73
Water Balance: Disorders of Sodium Concentration, 56	Intravenous Fluids During Anesthesia and Surgery, 82
Understanding Fluid Losses, 57	Specific Disease Conditions, 82

Fluid therapy should be approached with the same attention to detail as drug therapy, and the foundation for this approach is an understanding of body fluid balance and perfusion. Without understanding these concepts, the clinician risks taking a “cookbook” or one-size-fits-all approach to fluid therapy. Potential adverse effects of oversimplified approaches to fluid therapy include persistent dehydration, fluid overload, hypoperfusion, acid–base imbalance, and electrolyte disorders, all of which have profound effects on morbidity in patients.

## BODY FLUID BALANCE

Body fluid balance depends on both salt and water balance and the relationship between them. When referring to salt balance, we primarily consider the sodium ion ( $\text{Na}^+$ ) because it is the principal extracellular cation. *Water balance* refers to the amount of  $\text{Na}^+$  present relative to water. The concepts of salt and water balance are challenging, but they are essential for understanding the types, amounts, and rates of fluids to administer. It is perhaps counterintuitive that disordered salt balance does not cause abnormalities of serum sodium concentration but rather results in abnormalities of extracellular fluid (ECF) volume. Disorders of sodium concentration

result from abnormalities in water balance. Salt and water balance will be discussed in more depth in later sections.

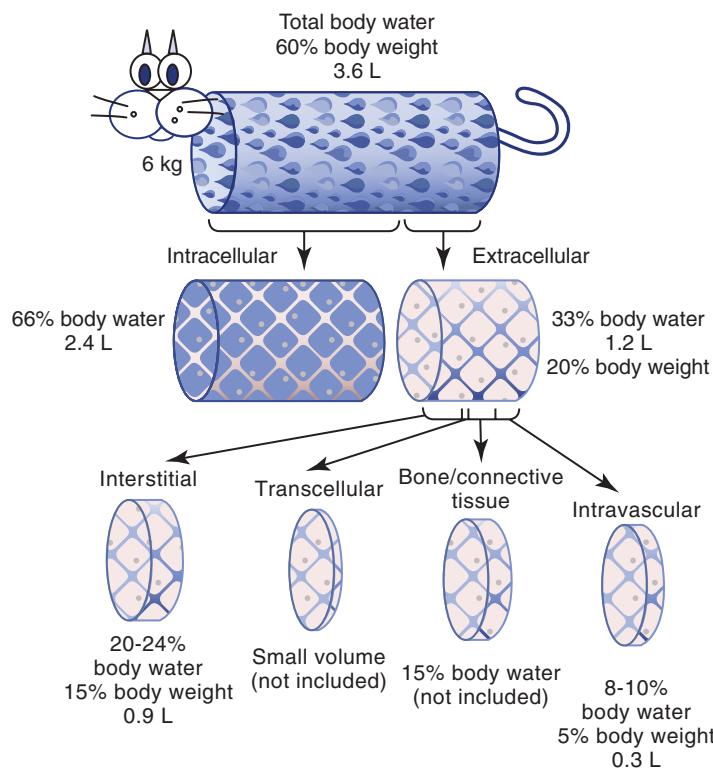
## STEADY STATE AND THE CONCEPT OF MAINTENANCE

With the exception of small, steady changes during growth, the amount of water coming into the body each day must equal the amount of water eliminated from the body over the same period. If not, the cat will have either a net water gain or a net water loss. Cats take in water by drinking, eating (food contains some water), and metabolizing nutrients to  $\text{CO}_2$  and water. Physiologic water losses result from the following:

- Obligate urinary loss
- Fecal loss
- Salivary loss
- Evaporation from the respiratory tract and the skin surface (insensible losses)

Pathologic loss of water can result from the following:

- Vomiting or regurgitation
- Diarrhea



**FIGURE 5-1** Body fluid compartments of the cat. The body's two main fluid compartments are the intracellular fluid (ICF) and the extracellular fluid (ECF). Approximately 66% of functional total body water is located within the ICF compartment, and 33% is in the ECF compartment.

- Bleeding
- Loss from wounds, burns, or drains
- Excessive urinary loss
- Excessive respiratory loss
- Excessive salivary loss

Electrolytes must also be consumed and eliminated in equal quantities on an approximately daily basis to maintain homeostasis. These continual losses that must be replaced promptly and nearly continually underlie the concept of "maintenance fluids." Maintenance needs, normally met by eating food and drinking water, are largely dependent on the cat's lean body mass. Sick animals that are no longer eating or drinking will continue to have daily obligatory fluid and electrolyte losses that can be addressed by fluid therapy to prevent negative fluid and electrolyte balance.

## BODY FLUID COMPARTMENTS

Water is a major contributor to a cat's body weight. In healthy animals approximately 60% of body weight is water. This value can change slightly depending on age, lean body mass, degree of leanness or obesity, and gender. For example, neonatal and young kittens have a

relatively higher percentage of water in their bodies than do adults.

Figure 5-1 depicts the cat's body fluid compartments. The body's two main fluid compartments are the intracellular fluid (ICF) and the ECF. Approximately 66% of functional total body water is located within the ICF compartment, and 33% is in the ECF compartment.

The ICF is, of course, not a single compartment but rather a conceptualization of the result of combining the very small volumes of a body's trillions of cells as one. This is useful for understanding physiology because of commonalities of ICF composition and behavior. The fluid inside cells is high in potassium ( $K^+$ ) and magnesium ( $Mg^{++}$ ) and low in  $Na^+$  and chloride ( $Cl^-$ ) ions. Additionally, fluid inside all cells will respond similarly to tonicity changes in the ECF.

The ECF space is composed of four main subcompartments: interstitial, intravascular, transcellular, and bone and dense connective tissue. The intravascular fluid is that which is contained within blood vessels; it contributes only 8% to 10% of total body water (5% of body weight) and has been estimated to be approximately 45 mL/kg in cats.<sup>10</sup>

The *interstitial compartment* refers to that portion of the ECF located outside of the vascular space. Like the ICF, this is not a single space but rather a conceptualization,

or “virtual space,” that would be created if all the interstitial fluid spaces were to be combined. It contributes approximately 22% to 24% to total body water (15% of total body weight).

The fluid of bone and dense connective tissue provides about 15% of the total body water. However, this fluid is mobilized very slowly, decreasing its importance when considering the effects of acute fluid interventions. Transcellular fluid is a normally small compartment that represents all those body fluids that are formed from the transport activities of cells. It is contained within epithelium-lined spaces. It includes cerebrospinal fluid (CSF), gastrointestinal fluids, urine in the bladder, aqueous humor, and joint fluid. The electrolyte compositions of the various transcellular fluids are dissimilar, but they are small in aggregate volume. However, fluid fluxes involving gastrointestinal fluid can be significant in disease.

The water in bone and dense connective tissue and the transcellular fluids, because of their slow mobilization, are subtracted from the total ECF volume to yield the functional ECF.

It is important to note that when excess fluid builds up in transcellular or interstitial compartments in which fluid volume are normally small, the process is termed *third spacing*. Excess fluid in the peritoneal space, pleural space, or gastrointestinal tract can add considerably to body weight, while diminishing the effective ECF volume.

## Fluid Movement in the Extracellular Fluid Compartment

The water in the body's ECF compartments is in a constant state of flux. Fluid moves across the capillary membrane, which is composed of endothelial cells that contain gap junctions through which fluid and solutes can flow. Solutes dissolved in fluid move from an area of higher to lower concentration along concentration gradients by the process of passive diffusion. The factors that regulate this transport of fluid and the electrolytes and other molecules it contains are called *Starling forces* (Box 5-1). The key factors are the hydrostatic and colloid oncotic pressure gradients between the intravascular and extravascular spaces. The hydrostatic pressure is greater in the capillary than in the interstitium, and the gradient favors fluid movement (filtration) out of the capillary. The colloid oncotic pressure, determined by protein concentration, is also greater in the capillary, and this tends to draw fluid into the capillary. Simplistically, at the beginning of the capillary the high hydrostatic pressure results in fluid egress into interstitium. As the fluid leaves along the length of the capillary, the hydrostatic pressure falls and the colloid oncotic pressure increases, resulting in fluid reentry into the capillary lumen toward the end of the capillary.

### BOX 5-1

#### Starling Forces

The Starling forces are defined by the equation  $J_v = K_f([P_c - P_i] - \sigma[\pi_c - \pi_i])$  relating to the following six variables: capillary hydrostatic pressure ( $P_c$ ), interstitial hydrostatic pressure ( $P_i$ ), capillary oncotic pressure ( $\pi_c$ ), interstitial oncotic pressure ( $\pi_i$ ), filtration coefficient ( $K_f$ ), reflection coefficient ( $\sigma$ ).

In the equation,  $([P_c - P_i] - \sigma[\pi_c - \pi_i])$  is the net driving force,  $K_f$  is the proportionality constant, and  $J_v$  is the net fluid movement between compartments.

By convention, outward force is defined as positive, and inward force is defined as negative. The solution to the equation is known as the net filtration or net fluid movement ( $J_v$ ). If positive, fluid will tend to leave the capillary (filtration). If negative, fluid will tend to enter the capillary (absorption). This equation has a number of important physiologic implications, especially when pathologic processes grossly alter one or more of the variables.

Fluid also leaves the interstitial compartment by way of the lymphatics.

Fluids in the ECF move continuously between the vascular space and the interstitial space across the capillary endothelium to achieve tissue perfusion. Edema results when the balance of the hydrostatic and colloid oncotic pressure gradients shifts such that fluid egress from the capillary is favored. All of the following promote edema formation: (1) decreased plasma oncotic pressure, (2) increased capillary hydrostatic pressure, (3) increased capillary permeability, and (4) lymphatic obstruction. The other key requirement for edema formation is  $\text{Na}^+$  retention: an increase in the ECF  $\text{Na}^+$  content.

## Fluid Movement Between the Intracellular Fluid and Extracellular Fluid Compartments

The ICF and ECF are separated by cellular membranes. The protein components of these membranes give them substantial and rapid permeability to water while carefully controlling their permeability to solutes such as ions. Cell membranes are also flexible. Thus, when water flows into or out of cells, those cells expand or shrink, respectively. Hydrostatic pressure, therefore, does not play a significant role in fluid movement between ECF and ICF compartments, because osmosis results in water flow rather than the development of pressure. Osmotic water flow occurs wherever there is a gradient of impermeable solute (such as  $\text{Na}^+$ ) across a water-permeable membrane (the body cell membranes).

In the body the ECF and ICF compartments are always in osmotic equilibrium, even though the composition of the fluids within them is very different. Water flows

into or out of cells and changes their volume when an osmotic gradient exists between the ICF and ECF compartments.

## PERFUSION

*Perfusion* refers to the process in which blood carries oxygen and nutrients to body tissues and organs and transports waste products of cellular metabolism away. Perfusion is optimized when an animal is in a state of normal fluid balance. Oxygen delivery is a critical part of perfusion and is dependent on the animal's cardiac output and the oxygen-carrying capacity of the blood. Cardiac output is a function of heart rate and stroke volume. Stroke volume depends on ventricular preload, ventricular afterload, and contractility. The amount of blood that enters the ventricle, causing the ventricular wall to stretch, thus affects the ventricular preload because the amount of wall stretch is directly proportional to the force of contraction. When there is adequate circulating volume, cardiac preload on the healthy ventricle results in a contraction of appropriate force. By contrast, in a cat with hypovolemia cardiac preload will be diminished, thus decreasing the force of ventricular contraction. Intravenous fluid therapy can affect cardiac preload by replenishing intravascular fluid volume in a hypovolemic animal.

## SALT BALANCE: DISORDERS OF ECF VOLUME

### Sodium Content

The  $\text{Na}^+$  content of the body determines the volume of the ECF and total body fluid volume. It does this because the osmolality of body fluids is regulated within a very narrow range. If  $\text{Na}^+$  ions (always with an accompanying anion) are added to the ECF space, more water molecules must be added to ECF space in the same proportion or the osmolality will increase beyond the relatively narrow range compatible with health and normal cellular function. Thus increasing the number of  $\text{Na}^+$  ions in the ECF (the  $\text{Na}^+$  content) increases the ECF volume. Similarly, if  $\text{Na}^+$  ions are removed from the ECF space, water molecules must leave in proportion, resulting in a decreased ECF volume, or the fluid's osmolality would decrease beyond that which the body's regulatory mechanisms will allow in health. In that sense, the  $\text{Na}^+$  content of the ECF fluid space determines its volume.

### Regulation of Sodium Balance

Regulatory mechanisms exist to control both  $\text{Na}^+$  content and  $\text{Na}^+$  concentration ( $[\text{Na}^+]$ ), and they are interrelated.

Body  $\text{Na}^+$  content is regulated by mechanisms that control the renal excretion of  $\text{Na}^+$  and which operate in response to body fluid volume and not  $[\text{Na}^+]$ .<sup>9</sup> Control of  $[\text{Na}^+]$  is determined by the osmoregulatory control mechanisms.

Evolutionarily, it appears that salt was a scarce commodity. Thus the kidney has evolved mechanisms to conserve salt.  $\text{Na}^+$  excretion in the urine can vary over 500-fold depending on  $\text{Na}^+$  intake and body need. The homeostatic mechanisms that control  $\text{Na}^+$  content are poorly understood. Regulation is generally a comparatively slow process. For example, many hours will pass before excesses in  $\text{Na}^+$  content (e.g., when isotonic saline is infused) are corrected by increased renal  $\text{Na}^+$  excretion. In contrast, excesses or deficiencies of water relative to  $\text{Na}^+$  (changes in  $[\text{Na}^+]$ ) activate the osmoregulatory mechanisms and are dealt with very rapidly. Many physiologists believe that a set point for  $\text{Na}^+$  regulation does not exist. Rather,  $\text{Na}^+$  is retained in low volume states until the volume deficit is corrected.<sup>12</sup>

Sodium excess results in augmented ECF volume, which increases urinary  $\text{Na}^+$  excretion. A useful analogy is a bucket with a hole in its side: When volume is at or below the hole,  $\text{Na}^+$  excretion is minimal while the bucket fills to the level of the hole. Once there, the inflow equals the outflow. When the volume of the bucket is above the hole (ECF expansion), the pressure of fluid in the bucket drives the fluid to flow out of the hole more quickly. For example, when dietary  $\text{Na}^+$  intake is increased, it takes several days to reach a new steady state of neutral  $\text{Na}^+$  balance.

The following factors are known to affect renal  $\text{Na}^+$  excretion:

1. Arterial hypertension
2. Tubuloglomerular feedback
3. Circadian rhythm

It is suspected that others also exist. The sensors (afferent signals) in the regulation of  $\text{Na}^+$  excretion are thought to include intrathoracic volume receptors, atrial pressure receptors, arterial baroreceptors, intrarenal baroreceptors, the macula densa, hepatic volume receptors, CSF volume receptors, and possibly tissue receptors. The mediators of  $\text{Na}^+$  conservation or excretion include the sympathetic nervous system, the renin-angiotensin-aldosterone system (RAAS), vasopressin, atrial natriuretic peptide (ANP), renal prostaglandins, the kallikrein-kinin system, nitric oxide, and renal pressure and flow phenomena (glomerular filtration rate [GFR], renal blood flow, and arterial pressure).

Salt excess states include congestive heart failure, nephrotic syndrome, hepatic cirrhosis, hyperaldosteronism,  $\text{Na}^+$  channel defects, and pregnancy. In the pathogenesis of certain salt-retaining states, reference is made to the *effective plasma volume*. This is not a measurable quantity, and the concept lacks a precise definition.

It refers to the “fullness” of the vascular volume. It is the portion of the vascular volume that is being sensed by those mechanisms that regulate body fluid volume. An inadequate effective circulating volume is inferred when salt-retaining mechanisms are activated.

Salt-deficient states are secondary to a number of disease conditions that result in losses of  $\text{Na}^+$  or inadequate intake. Extrarenal losses are localized to diseases of the gastrointestinal tract, skin, respiratory tract, and third space losses. Renal salt loss may occur with the following:

1. Diuresis (e.g., diuretic phase of acute tubular necrosis, postobstructive diuresis, or the use of diuretic medications)
2. Intrinsic renal disease (e.g., chronic renal failure, Fanconi syndrome, Bartter syndrome)
3. Defects in the RAAS system (e.g., hypoadrenocorticism, hyporeninemic hypoaldosteronism)
4. Disorders resulting in excesses of ANP

The use of high-sodium, isotonic fluid types in fluid therapy is thus primarily for the administration of  $\text{Na}^+$ . They augment the total ECF  $\text{Na}^+$  content and thus expand the functional ECF volume, both vascular and interstitial.

## WATER BALANCE: DISORDERS OF SODIUM CONCENTRATION

### Permeant and Impermeant Solutes

The next important concept to be understood is that of water balance. Cells must be in osmotic equilibrium with the fluid that surrounds them, insofar as their membranes are permeable to water. Although extracellular and intracellular fluids have very different compositions, they must have the same total solute concentrations because of the free movement of water. Looking at this concept “in reverse,” the water concentration of the ECF and the ICF must be the same. Inequalities of water concentrations in body fluid compartments can exist only transiently because water movement occurs rapidly to correct these inequalities. This basic concept underlies an understanding of fluid movement between intracellular and extracellular compartments during intravenous fluid therapy.

The concentration of solutes in fluid defines the solution’s osmolality. Because cell membranes are water permeable and water movement will occur until solutions on either side of a membrane are iso-osmolar, the osmolality of plasma reflects the osmolality of the body fluid in total. It is important to distinguish between permeant and impermeant solutes. Permeant solutes (e.g., urea)

move freely across cellular membranes and thus do not induce net water movement across cell membranes when they are introduced into a solution; they are termed *ineffective osmoles*. Impermeant solutes (e.g.,  $\text{Na}^+$ ) do not freely move across cell membranes and do induce water movement when introduced into a solution; thus they are effective osmoles.

### Tonicity

The term *tonicity* refers to the effect a solution has on cellular volume. Hypertonicity results when impermeant solutes are added to the ECF; this promotes cellular dehydration. Hypotonicity results from a decrease in the concentration of impermeant solutions; this results in water movement into cells and cellular swelling. Hypertonic solutions are always hyperosmolar. The reverse is not always the case: Hyperosmolar solutions are not necessarily hypertonic because ineffective osmoles contribute to osmolality but not tonicity.

Plasma  $[\text{Na}^+]$  is the key determinant of the osmolality of body fluids. Glucose and urea make minor contributions under normal circumstances. Plasma osmolality may be calculated using the following equation:

$$2[\text{Na}^+](\text{mEq/l}) + [\text{glucose}](\text{mg/dL})/18 + [\text{BUN}](\text{mg/dL})/2.8$$

The preceding equation is a simplification because it does not take into account the fact that plasma is only 93% water; that sodium salts are not completely dissociated in solution; or that calcium, magnesium, and potassium salts also contribute. However, these factors appear to cancel out because experimental evidence demonstrates that calculated osmolality and measured osmolality are in close agreement in normal patients.

Plasma  $[\text{Na}^+]$  reflects the plasma tonicity very well in normal patients. Urea is an ineffective osmole because it equilibrates freely across cell membranes and does not induce fluid shifts. Glucose normally can move across cell membranes in normal patients in the presence of insulin; therefore it is usually an ineffective osmole, similar to urea. In diabetic patients lacking insulin, it becomes an effective osmole. Thus plasma  $[\text{Na}^+]$  predicts plasma tonicity when the glucose concentration is known. In hyperglycemia water leaves the cells because of the hypertonicity of the ECF. This serves to dilute the  $\text{Na}^+$ , and hyponatremia is observed.

It is important to remember that serum  $[\text{Na}^+]$  does not reflect body salt balance. Salt balance determines ECF volume. Serum  $[\text{Na}^+]$ , instead, reflects the state of water balance. The term *osmoregulation* refers to the control of body fluid tonicity. By stabilizing body fluid tonicity, osmoregulation thus controls cell volume. Osmoreceptors are, in fact, hypothalamic cells that sense their own

cell volume. Changes in plasma osmolality sensed by these cells affect secretion of arginine vasopressin (antidiuretic hormone; ADH). ADH is the primary regulator of renal water excretion. Changes in plasma osmolality also strongly affect the thirst mechanism. This is why patients with central diabetes insipidus, who thus lack ADH, are able to maintain a relatively normal osmolality provided they have access to water and the ability to drink.

## Regulation of Water Balance

In contrast to salt balance, which is controlled by many factors and has a relatively slow response to changes in effective plasma volume, plasma osmolality is very tightly regulated. When plasma osmolality is altered, changes in thirst and ADH secretion, and the resulting renal response, are brisk.

In addition to plasma osmolality, hypotension and hypovolemia also stimulate ADH release and thus this is a point where regulation of salt and water balance are interrelated. ADH release is not as sensitive to hemodynamic stimuli as it is to changes in osmolality; however, when the hemodynamic stimulus is sufficiently strong, the ADH response will be of higher magnitude. In the presence of a significant volume deficit, decreased water excretion by the kidney will in fact act to increase volume at the expense of a decrease in plasma osmolality.

The primary function of ADH is to increase the water permeability of the luminal membrane of the collecting duct of the nephron. ADH, through a second-messenger system, causes water channels called *aquaporins* to be inserted into the cell membrane. Water reabsorption in the collecting duct occurs through these channels, thus allowing the kidney to conserve water.

## UNDERSTANDING FLUID LOSSES

### Sensible and Insensible Fluid Losses

Sensible fluid losses are those that can be measured and include fluid lost in the form of urine, feces, vomitus, body cavity effusions, and wound exudates. It is important in seriously ill patients actually to quantify these losses and incorporate them into the fluid prescription. For example, urine output can be determined by collecting voided urine or by weighing the bedding or litter when dry and again after urination. Similarly, vomitus or diarrhea can be weighed to allow the clinician to estimate fluid loss, given that 1 g is approximately equivalent to 1 mL water. For wounds with large volumes of exudate, an animal's bandage material can be weighed before and after use to create a fluid loss estimate. For patients with drains or chest tubes in place, the amount of fluid produced from these devices can also be measured.

Insensible losses are those that cannot directly be measured. They include largely solute-free water in evaporative respiratory, sweat, and salivary losses. This classical definition of *insensible losses* is sometimes replaced with a clinical definition that includes fecal water loss. This is because the amount of normal daily fecal water loss is small and is rarely measured.<sup>16</sup>

As a gross approximation, sensible fluid losses account for half of a healthy animal's daily fluid requirement and insensible losses account for the other half. However, this partitioning is variable and is species and environment dependent. For example, dogs as a species may have a higher percentage of insensible losses compared with cats because of the greater use of panting for thermoregulation. However, cats may have considerable salivary losses if they have increased grooming or lick their fur to promote evaporative cooling in hot weather.<sup>7</sup>

Insensible fluid losses are generally considered to be solute-free water because respiratory losses are the major contributor in small animal species, including cats. Insensible losses are estimated to be 12 to 30 mL/kg/day, depending on the study and definition.<sup>16</sup>

In contrast to insensible losses, sensible fluid losses do contain solutes. Although a necessary oversimplification, this explains why maintenance fluid types are hypotonic. They replace the solute-free water of insensible losses and the solute-containing water of sensible losses. As a matter of practicality, given these estimates and variable definitions, it is reasonable to assume that half of daily maintenance fluids are to offset normal levels of obligate urine output required for daily solute excretion and are solute-containing fluid losses, and the other half, accounting for everything else, are solute-free water losses. This is important clinically when adjusting the fluid prescription for "ins and outs" because it provides a method to estimate how much of measured hourly urine production is abnormal losses in polyuric patients (who have excessive ongoing urinary fluid loss that must be met by the replacement portion of the fluid prescription) and how much is normal obligate urine (and thus met by maintenance portion of the fluid prescription).

### Body Weight and Fluid Losses

Total body water remains essentially the same day to day in a healthy animal. However in disease, excessive loss of fluid can occur associated with hemorrhage, vomiting, diarrhea, burns, fever, effusions, wound exudates, polyuria, and panting. Because rapid changes in body weight, over the span of hours to a few days, are largely due to changes in total body water, changes in an animal's weight are an invaluable tool in the assessment of an animal's hydration status. Because of the relative small size of cats, weighing animals on scales that can

accurately detect changes of a few ounces or several grams (human pediatric scales) are very important.

### Relationship to Lean Body Mass

Because lean body mass is so important in determining an animal's daily fluid need, resting energy requirement (RER), or daily caloric requirement, is used to calculate an animal's metabolic water requirements: To metabolize 1 kilocalorie of energy, 1 mL of water is consumed. As such, calculation of an animal's RER can be extrapolated to determine the volume of fluid in mL required in a 24-hour period. Several equations may be used to calculate RER and hence water requirement.<sup>5</sup> The following is one of the most commonly used:

$$\text{RER} = 30(\text{BW}_{\text{kg}}) + 70$$

This formula is accurate for animals weighing more than 2 kg and less than 25 kg and thus is applicable to adult domestic cats. Kittens and cats weighing less than 2 kg require the use of a different formula:

$$\text{RER} = 70(\text{BW}_{\text{kg}})^{0.75}$$

### Terminology of Body Fluid Balance

The terminology used to describe body fluid balance is at times unfortunately vague. *Dehydration* refers to a decrease in total body water: loss of fluid from the ICF and ECF compartments. However, the physical examination findings used to assess hydration, such as skin tenting and mucous membrane dryness, are specifically assessments of the ECF volume and subject to significant individual variation and inaccuracy. Thus when clinicians suggest that a patient "appears dehydrated," they are referring specifically to clinical signs of ECF volume depletion. This is to be distinguished from *hypovolemia*, which refers to inadequate circulating intravascular fluid volume. The distinction is important because hypovolemia is a much more time-sensitive condition requiring rapid, aggressive treatment.

### Assessment of Fluid loss

Patients can be assumed to have a decrease in total body water in the presence of known excessive net losses, such as those produced by vomiting, diarrhea, anorexia, and marked polyuria, even without a demonstrable increase in skin tenting and mucous membrane dryness, which are detectable only when 4% to 5% of total body weight has been lost. Humans report headaches with dehydration, and presumably this may reflect some of the general lethargy seen in volume-depleted cats. At 7% total body weight loss, mild tachycardia could also be present. At 10% total body weight loss, the patient might

also have a palpably decreased pulse pressure. Signs of very severe total body water loss are sunken eyes, dry corneas, and altered mentation. Overt hypovolemia will occur with severe fluid loss (>12% body weight), even when chronic. It is important to note at this time that lethargy can be present with both underhydration and overhydration. This is particularly important in cats with oliguria because they are readily susceptible to overhydration.

The aforementioned physical findings that are used to determine an animal's total body fluid status are not used to assess hypovolemia. Peripheral perfusion should instead be assessed by capillary refill time (CRT), mucous membrane color, arterial blood pressure, pulse quality and rate, and temperature of extremities.

The body responds to fluid loss by redistributing the functional ECF volume—that is, by pulling fluid into the intravascular space from the interstitial space to maintain circulating blood volume as the first priority. When the interstitial space can no longer replenish intravascular volume depletion, clinical signs of hypovolemia will result. In decompensated shock, severe hypovolemia results in a marked worsening of perfusion parameters. Hypotension, bradycardia, prolonged CRT, pale pink to gray or cyanotic mucous membranes, hypothermia, decreased central venous pressure, altered mentation, and decreased urine output will be present in decompensated shock. Table 5-1 gives examples of the types of fluid losses that would be expected with selected medical problems.

### Assessment of Fluid Excess

Overhydration, like dehydration, is detrimental to patients and should be avoided in patients on fluid therapy. Human and canine patients with ECF volume excess can have pulmonary edema, ascites, and generalized peripheral edema. Unique to the cat, possibly because of a difference in the anatomy of pulmonary venous drainage, pleural effusion may develop more readily than pulmonary edema or ascites. Cats with preexisting cardiac disease are more susceptible to pleural effusion or pulmonary edema with volume overload, depending on their underlying disease. Cats with oliguria or markedly decreased GFRs are also particularly at risk for overhydration, and measurement of urinary output is essential in such patients. Early signs of overhydration may include the more subtle findings of loss of appetite and mental dullness. An astute clinician will notice tachypnea or crackles on auscultation, insofar as pleural effusion or pulmonary edema develops before the onset of overt dyspnea. Careful and repeated weighing is important for prevention of volume overload, particularly in cats, because of their small size.

**TABLE 5-1** Examples of the Types of Fluid Losses that Would Be Expected with Selected Medical Problems

Condition	Dehydration	Hypovolemia
Blood loss		X
Vomiting	X	X (if severe)
Diarrhea	X	X (if severe)
Sepsis/vasodilation		X
Hypoadrenocorticism		X
Polyuria	X (depending on cause)	X (depending on cause)
Hypodipsia or water deprivation	X	

## BODY RESPONSE TO HYPOVOLEMIA

Intravascular volume status is sensed by baroreceptors in the carotid body and aortic arch. In euvolesmic cats, stimulation of the stretch receptors triggers the vagus nerve to maintain an appropriate heart rate. In hypovolemia the baroreceptors sense a decrease in wall tension, and the sympathetic nervous system is activated. Norepinephrine and epinephrine release results in vasoconstriction, improved cardiac contractility, and an increase in heart rate. These effects are designed to compensate for decreased intravascular fluid volume by improving cardiac output and maintaining systemic blood pressure and, ultimately, perfusion. Hypovolemic shock results when intravascular volume is sufficiently reduced that these compensatory mechanisms are overwhelmed and decreased tissue perfusion results. Perfusion parameters that can be assessed include capillary refill time, blood pressure, heart rate, and temperature of extremities.

Cats are unique in that the vasoconstrictor response to volume loss is blunted in the presence of hypothermia.<sup>13,14</sup> For this reason, cats are more prone than other species to fluid overload when they have been volume resuscitated while hypothermic. Once body temperature returns to normal, the vasoconstrictor response returns and intravascular pressure rises. For this reason, hypothermia in cats is a potentiator of shock, as well as a result of shock. Cautious fluid resuscitation must coincide with aggressive rewarming efforts to prevent volume overload. Specific therapy and therapeutic endpoints for resuscitation are discussed in subsequent sections.

## GENERAL CONSIDERATIONS FOR FLUID THERAPY

Fluid therapy choices are often the product of educated guesswork. They certainly rely on the physiologic abilities of a normally functioning kidney for fine-tuning. Although there are many useful guidelines for selection of fluid types and rates, good fluid management demands careful monitoring of body weight, physical

examination, and electrolyte concentrations. The veterinarian must be prepared to alter the fluid therapy prescription in response to changes in these parameters, and should understand that the initial fluid therapy plan merely provides a starting point. The clinician must also be more vigilant with fluid therapy monitoring in patients with cardiovascular or renal disease. It should be remembered that cats may have cardiac disease in the absence of a detectable murmur.

As fluid bag sizes are not scaled down to the size of feline patients, it is helpful to use fluid pumps, burettes, and other devices to prevent fluid overload and pulmonary edema or pleural effusion (Figure 5-2).

## FLUID TYPES

The two main types of parenteral fluids, crystalloids and colloids, have fundamental differences that affect the way fluid distributes among body fluid compartments. Crystalloids are composed of smaller molecules that diffuse readily; therefore approximately 80% of the fluid infused will leave the intravascular space within 1 hour. Colloids, made of larger molecules, stay within the intravascular space longer, which is an important advantage when managing hypovolemia. The tonicity of fluids determines distribution rates to the intracellular and extracellular spaces. When the  $[Na^+]$  of a fluid approximates that of plasma (145 mEq/L), it will equilibrate rapidly with the interstitial space. Remaining fluid that is not lost in urine or as other ongoing losses will distribute to the ICF in proportion to the normal size of those compartments: two thirds ICF and one third ECF.

Hypotonic fluids, with a  $[Na^+]$  lower than that of plasma, will dilute the plasma and drive water into cells to equilibrate the water concentration inside and outside cells. The decreasing plasma osmolality (pOsm) will also result in a decreased ADH production and, thus, increased water excretion by the kidney. Most of the  $Na^+$ -free fluid thus either enters cells or is excreted. Hypertonic fluids with a  $[Na^+]$  higher than plasma will draw water out of cells and into the ECF, thus increasing



**FIGURE 5-2** Because fluid bag sizes are not scaled down to the size of feline patients, it is helpful to use fluid pumps (A and B), burettes (C), and other devices to prevent fluid overdosing.

the intravascular and interstitial volumes but at the expense of taking water from the ICF compartment. Thus an understanding of which body compartments need to be replenished in any given patient is essential in fluid selection. This is true not only regarding fluid types selected but also in terms of route of administration. For example, fluids instilled into the subcutaneous space cannot be used readily to replenish the intravascular blood volume because they will be absorbed too slowly in a patient with hypovolemia.

### Crystalloids

A crystalloid is a solution that is able to pass through a semipermeable membrane, including the vascular endothelium. The ability of crystalloids to pass through the capillary endothelium allows them to replenish fluid losses both in the intravascular and interstitial compartments, making them ideal for rehydration therapy. All crystalloid fluids are true solutions, meaning that they are homogeneous and transparent, diffuse rapidly, and do not settle. Substances that are dissolved in crystalloids are termed *solute*; these are predominantly electrolytes and dextrose.

Solutes contained in crystalloid fluids move freely from the intravascular space to the interstitial space. Movement of impermeant solutes such as ions and glucose into the intracellular compartment is comparatively slower, occurring by facilitated diffusion or active transport. As parenteral fluid solutions, most crystalloids are formulated with a solute concentration close to that of plasma to avoid osmotic cell damage, particularly red blood cell damage from tonicity-induced osmotic water movement. Some parenteral intravenous solutions, such as 0.45% NaCl and 5% dextrose in water (D5W) are hypotonic and can cause hemolysis if given too rapidly.

The three categories of crystalloids are isotonic high-sodium, hypotonic low-sodium, and hypertonic saline; they differ primarily in their sodium concentrations.

#### ***Isotonic High-Sodium Crystalloids***

##### **GENERAL CHARACTERISTICS AND INDICATIONS FOR ISOTONIC HIGH-SODIUM CRYSTALLOIDS**

Isotonic high-sodium fluids are commonly referred to as *replacement fluids* because they are often used for rapid replacement of ECF volume deficits caused by vomiting

**TABLE 5-2** Composition of Common Crystalloids\*

	0/9% NaCl	Lactated Ringer's Solution	Ringer's Solution	Normosol-R	0.45% NaCl + 2.5% Dextrose	5% Dextrose	Plasma-Lyte 56 + 5% Dextrose
Na <sup>+</sup> (mEq/L)	154	130	147	140	77	0	40
Cl <sup>-</sup> (mEq/L)	154	109	154	98	77	0	40
K <sup>+</sup> (mEq/L)	0	4	4	5	0	0	13
Ca <sup>++</sup> (mEq/L)	0	3	4	0	0	0	0
Mg <sup>++</sup> (mEq/L)	0	0	0	3	0	0	3
Osmolality (mOsm/L)	308	273	309	294	280	252	112
pH	5.6	6.6	5.4	6.6	4.3	4.3	5.5
Buffer (mEq/L)	0	28 (Lactate)	0	27 (Acetate), 23 (Gluconate)	0	0	16 (Acetate)
Dextrose (g/L)	0	0	0	0	25	50	50
Calories (Kcal/L)	0	9	0	15	85	170	175

\*High-sodium "replacement" fluids are in red. Low-sodium "maintenance" fluids are in green.

and diarrhea. They have a [Na<sup>+</sup>] near that of ECF, ranging from approximately 130 mEq/L (e.g., lactated Ringer's solution [LRS]) to a high of 154 mEq/L (e.g., 0.9% saline). Table 5-2 includes additional examples of replacement fluids, highlighted in red.

Isotonic high-sodium fluids are used both for hypovolemia and for less severe ECF volume depletion, such as dehydration. When given rapidly, they can be used to restore the intravascular fluid volume in cats with hypovolemia. They are also used, when administered more slowly, to replace ECF volume in states of isotonic dehydration that are not immediately life threatening, such as occurs in patients with gastrointestinal or urinary fluid losses when oral intake is insufficient to balance losses.

Isotonic high-sodium fluids are not suited for use as maintenance fluids. They lack sufficient solute-free water content to offset ongoing solute-free water loss, such as through respiratory evaporation. When used on a short-term basis, most patients with normal renal function will tolerate the excess Na<sup>+</sup> that these fluids contain when they are being used primarily to compensate normal daily ongoing hypotonic fluid loss. This is particularly true when patients are able to drink some water in addition to their intravenous fluid therapy. Some patients can become hypernatremic after therapy with high-sodium fluid. There are also some patients for whom the use of high-sodium fluids is contraindicated, including those with congestive heart failure, oliguric renal disease, and some edema states.

Isotonic high-sodium fluids are used to maintain patients with ongoing isotonic fluid losses, as, for

example, in vomiting or diarrhea. However, in these patients the fluids are in fact being used for replacement of these losses, rather than for true maintenance. It is critical to understand this distinction. *Maintenance* is a term used to reflect what is needed to replace only normal sensible and insensible losses, and such losses are not isotonic. Isotonic fluids may work to maintain fluid balance in animals with additional pathologic losses because such patients need the additional sodium and chloride. However, such patients need to drink to provide solute-free water; otherwise, hypernatremia will develop. In addition to the relative solute-free water deficit of high-sodium isotonic fluids, all of these fluids are also too low in potassium to be used as true maintenance fluids, unless K<sup>+</sup> is added to the fluids. Patients with continuing ongoing losses for more than 1 to 2 days that stay on high-sodium fluids are likely to need nutritional support, which will also replace their hypotonic maintenance fluid needs.

Some patients with readily corrected deficits and no ongoing losses will need to be transitioned to a maintenance-type solution, such as Normosol-M-D5 or Plasmalyte-56-D5, after their rehydration and electrolyte needs have been corrected and before the start of enteral or parenteral nutrition. The need to change to a true maintenance fluid will be indicated by a progressive increase in serum [Na<sup>+</sup>] in these patients. Changes should be made well in advance of the development of hypernatremia.

Sick cats that have been anorexic for 2 to 3 days or longer should receive nutritional support. Generally, the provision of enteral or parenteral nutrition sufficient to

**TABLE 5-3** Calculation Worksheet for Fluid Therapy\*†

Components of the Fluid Plan		Type of Fluid	Volume of Fluid	
			mL/day	mL/h
Deficits	Isotonic			
	Hypertonic			
Maintenance	Normal ongoing losses (Enteral contribution from feeding)			( ) ( )
	Net normal loss to be provided by fluids			
Abnormal ongoing losses	GI			
	Urinary			
	Other sensible			
	Insensible			
Totals		1. 2. 3.	1. 2. 3.	1. 2. 3.

\*The enteral contribution includes what the cat is eating or drinking on its own plus any provision of food or water through tube feeding. It is indicated in *parentheses* ( ) to signify that this volume is subtracted from the calculated maintenance fluid needs to yield the net that must be provided to the cat as part of intravenous fluid therapy plan.

†For a case example, see Table 5-8.

meet the patient's caloric needs will also provide maintenance fluid needs. Thus the use of additional isotonic, high-sodium fluids in this setting will be to replace excessive isotonic losses, such as those associated with gastrointestinal loss or polyuria. Intravenous fluid rates can be greatly reduced in patients receiving either enteral or parenteral nutrition to a rate sufficient to meet additional ongoing losses only. In other words, the fluid therapy recipe should account for all sources of fluid intake (Table 5-3; also see Table 5-8).

#### ACIDIFYING AND ALKALINIZING FLUIDS

Sick cats requiring fluid therapy may also have acid-base disorders, and fluid therapy can be used to mitigate these disturbances. Restoration of ECF volume will improve tissue perfusion and correct lactic acidosis. The replenishment of water and electrolytes in appropriate concentrations will also improve renal perfusion and normalize renal electrolyte handling, thus promoting an improved acid-base balance. The volume expansion and improved perfusion seen with appropriate fluid therapy will also promote the peripheral utilization of glucose and decrease production of lactate. The end result of this can be the normalization of acid-base balance without the need to resort to the use of sodium bicarbonate, which can have adverse effects, such as hypernatremia and central nervous system acidosis.

High-sodium crystalloids will have a primary effect on the patient's acid-base status, depending on their composition. As such, they can be classified as either acidifying or alkalinizing solutions. High-sodium fluids that contain more Cl<sup>-</sup> than is present in the patient's ECF are acidifying. Although 0.9% saline has a high Na<sup>+</sup> content and thus is frequently used to restore intravascular fluid in hypovolemic patients, it also has a high

Cl<sup>-</sup> content and will be acidifying. This fluid is most appropriate for treatment of patients with hypochloremic metabolic alkalosis because it provides the necessary Cl<sup>-</sup>. A common clinical scenario associated with hypochloremic metabolic acidosis is the vomiting of gastric contents. It is important to point out that although the measured pH of parenteral fluid solutions ranges from about 4 to 6.5, they are extremely weak acids. These low measured in vitro pH values will *not* reflect their effect on pH in the patient because of buffering.

Alkalinating fluids, by contrast, do not have a higher Cl<sup>-</sup> concentration than ECF fluid. Some of the chloride is replaced with another anion such as lactate, acetate, or gluconate. The anions are metabolized by the liver to bicarbonate. One example of a commonly used alkalinizing fluid is LRS.

#### SUPPLEMENTS

In some situations fluids must be supplemented with additional electrolytes; this decision is based on an assessment of the history, physical examination findings, and measured electrolyte values. Commonly added electrolytes are listed in Table 5-4. Electrolyte additives may be appropriate for replacing deficits, providing replacement for normal maintenance losses in anorexic patients, compensation for transcellular movement of ions, or replacement of ongoing gastrointestinal or urinary losses. Potassium and magnesium are found in some low-sodium hypotonic fluids formulated as maintenance fluids. If not, they can be added to fluids to maintain homeostasis in animals that are not depleted.

**POTASSIUM** All of the isotonic high-sodium fluids, apart from 0.9% NaCl, contain 4 or 5 mEq/L of potassium. Although this amount of potassium is within

**TABLE 5-4** Concentration of Common Fluid Additives

Product	Concentration per mL
KCl	2 mEq each
KPO <sub>4</sub>	4.4 mEq K <sup>+</sup> , 3 mM PO <sub>4</sub> <sup>3-</sup>
MgCl	1.97 mEq each
MgSO <sub>4</sub>	4.06 mEq each
Ca gluconate 10%	0.465 mEq Ca <sup>++</sup>
CaCl <sub>2</sub>	1.36 mEq Ca <sup>++</sup>
NaPO <sub>4</sub>	4 mEq Na <sup>+</sup> , 3 mM PO <sub>4</sub> <sup>3-</sup>
Dextrose 50%	500 mg

From Abbott Animal Health Fluid Therapy Module 2, courtesy Dr. Steve Haskins.

the normal range of plasma [K<sup>+</sup>], it is not in fact sufficient for maintenance of the patient. This is because therapy with intravenous isotonic high-sodium fluids typically causes a solute diuresis. The rate of flow of filtrate through the renal tubule is one of the factors regulating renal potassium excretion. As urine flow rate increases in response to intravenous fluid administration, K<sup>+</sup> loss in the urine will also increase. The loss of K<sup>+</sup> from the body will be further compounded by decreased intake in patients that are anorexic or hyporexic and by increased losses of K<sup>+</sup> in gastrointestinal secretions in patients with vomiting or diarrhea. Thus when isotonic high-sodium fluids are used for maintenance of patients that are drinking or for support of patients with ongoing isotonic fluid losses, it is necessary to supplement the fluids with additional K<sup>+</sup>. A common level of supplementation for a cat that is normokalemic is the addition of 20 mEq/L of KCl to the isotonic high-sodium fluid. This amount is typically added to the K<sup>+</sup> already present in the fluids; it is not necessary to subtract the small amount that is already present in the fluid. If K<sup>+</sup> is not added to these fluids when they are used for more than a short time in normokalemic patients, hypokalemia will result.

For patients that are hypokalemic, a sliding scale is used to calculate how much potassium to add to the fluid. One such scale is shown in **Table 5-5**. When using the potassium-containing replacement fluids for fluid resuscitation, it must be remembered that if the K<sup>+</sup> concentration of the fluid exceeds 5 mEq/L, the fluid must not be infused rapidly for intravascular volume restoration because of the risk of hyperkalemia.

Cats with anorexia, gastrointestinal losses, or polyuria are particularly at risk for K<sup>+</sup> depletion. As an alternative to the sliding scale, a constant-rate infusion (CRI) is typically used to give K<sup>+</sup> separately when the patient seems resistant to "normal" amounts of K<sup>+</sup> supplementation, particularly in diabetic ketoacidosis (DKA) (**Figure 5-3**). This allows K<sup>+</sup> to be adjusted separately



**FIGURE 5-3** A constant-rate infusion is typically used to give K<sup>+</sup> separately when the patient seems resistant to "normal" amounts of K<sup>+</sup> supplementation, particularly in diabetic ketoacidosis.

**TABLE 5-5** Sliding Scale for the Amount of KCl Added to Intravenous Fluids Depending on the Serum [K<sup>+</sup>]\*

Measured Serum K <sup>+</sup> (mEq/L)	KCl Added (mEq/L)
>5.5	None
3.6-5.5	20
3.1-3.5	30
2.6-3	40
2-2.5	60
<2	80

\*If the [K<sup>+</sup>] of the fluid exceeds 5 mEq/L, the fluid must *not* be infused rapidly for intravascular volume restoration because of the risk of hyperkalemia.

from the remainder of the fluid prescription. If CRI is used, it must be monitored very carefully. The usual dose range to replace normal ongoing losses of potassium is 0.05 to 0.1 mEq/kg per hour. The dosage for cats with severe whole body potassium depletion, severe symptomatic hypokalemia, or both can be as high as 0.5 mEq/kg per hour, known as KMax. Administration at rates higher than 0.5 mEq/kg per hour can cause serious or fatal cardiac arrhythmias. Administration of undiluted KCl (2 mEq/mL) through a programmable syringe pump is possible, but should only be done with extreme care, reserved for intensive care situations in patients with life-threatening hypokalemia (generally levels below 1.5 mEq/L).

**PHOSPHATE** Hypophosphatemia may develop rapidly during insulin therapy for DKA. For hypophosphatemia a portion or all of the phosphate (PO<sub>4</sub><sup>3-</sup>) can be administered as potassium phosphate (KPO<sub>4</sub>) when the [PO<sub>4</sub><sup>3-</sup>] is less than 2 mEq/L or when serum [PO<sub>4</sub><sup>3-</sup>] is observed to be decreasing rapidly and a drop below that

level is expected. This also provides  $K^+$  for concurrently hypokalemic patients. Hemolysis can occur when the  $[PO_4^-]$  is less than approximately 1.5 mEq/L in cats. Sodium phosphate is used in the unusual case of phosphate-depleted patients that do not require potassium. The usual dose range to replace normal ongoing losses of phosphate is 0.01 to 0.03 mmol/kg per hour. Dose rates as high as 0.12 mmol/kg per hour may be necessary in some patients being treated for DKA.

**MAGNESIUM** Magnesium depletion is common in critically ill patients, particularly those with decreased dietary intake and polyuria, such as patients with diabetes mellitus. Supplementation is generally recommended when serum total magnesium concentration is less than 1.5 mg/dL. Because total serum magnesium concentration does not represent the physiologically active form of the element, measurement of ionized magnesium should be performed when possible; however, this assay is not readily available in most veterinary hospitals.

Replacement doses are 0.03 to 0.04 mEq/kg per hour for severe cases requiring rapid replacement and 0.013 to 0.02 mEq/kg/hour for more mild deficiency.  $MgCl_2$  or  $MgSO_4$  may be given but not mixed with calcium- or bicarbonate-containing fluids.<sup>2</sup> Patients with reduced GFR are at greater risk for hypermagnesemia during supplementation and require more frequent monitoring. Magnesium is a cofactor for potassium homeostasis, and magnesium supplementation should be considered in any patient with refractory hypokalemia.

**CALCIUM** Calcium gluconate 10% is used as a calcium source for animals with symptomatic hypocalcemia, such as that associated with eclampsia, hypoparathyroidism, acute pancreatitis, and renal failure. It may be administered on an emergency basis at a dosage of 0.5 to 1.5 mL/kg, diluted and given over several minutes, or it is added to fluid therapy and given more slowly and at a rate that is titrated to effect. Calcium chloride contains about three times the amount of calcium per mL compared with calcium gluconate and is administered at one third the volume of calcium gluconate. Calcium gluconate is preferred because it is less irritating if inadvertently given perivascularly. Renal failure patients often have hypocalcemia associated with hyperphosphatemia, and thus administration of calcium may lead to the formation of  $CaPO_4$  in tissues. The latter occurs when the product of  $[Ca^{++}]$  and  $[PO_4^-]$  exceeds 70. Ideally, these patients should have their serum phosphate lowered as rapidly as possible to minimize this risk.

**ELECTROLYTE INFUSIONS** Because rapid infusion of any of these electrolytes can cause cardiac arrhythmias and other side effects, they should be added

to fluids provided at a constant rate for maintenance to avoid inadvertent overdose. Delivery systems that guard against fluids being inadvertently left to run "wide open" are strongly recommended. Electrocardiographic monitoring is necessary when intravenous  $Ca^{++}$  or  $Mg^{++}$  are given rapidly.

When using these electrolyte additives, remember that divalent cation salts of phosphate are insoluble. They should not be added to fluids containing  $Ca^{++}$ ,  $Mg^{++}$ , or  $PO_4^-$  to avoid precipitation of  $CaPO_4$  or  $MgPO_4$ .

**GLUCOSE** Hypoglycemia may accompany critical illness and can be treated with dextrose supplementation. There is increasing evidence in humans that tight glycemic control improves outcomes in critically ill patients; whether this holds true in cats is unknown. Typically, 50% dextrose is added to intravenous fluids in concentrations from 2.5% to 5% to maintain blood glucose in the 80 to 120 mg/dL range. Final concentrations that are above 10% should be administered through central venous catheters to reduce the risk of thrombophlebitis. The concentration can be measured in percentage or g/dL. For example, to make 1 L of a 5% solution (5 g/dL or 50 g/L), 100 mL of fluid is removed from the bag and 100 mL (50 g) of 50% dextrose is added. For partial liters the amount of stock dextrose solution (usually 50% dextrose) to add can be calculated by the following means:

$$\frac{\text{Volume remaining in bag (mL)}}{\text{Concentration of stock solution (as a decimal)}} \times \text{desired dextrose concentration (as a decimal)}$$

For example, to make a 3% dextrose solution with 650 mL fluids using 50% dextrose:

$$\frac{650 \text{ mL} \times 0.03}{0.5} = 39 \text{ mL of 50\% dextrose}$$

The 39 mL of dextrose should be added after removing 39 mL of fluid from the bag.

### Hypotonic Low-Sodium Crystalloids

#### MAINTENANCE

Low-sodium crystalloid fluids are indicated for the short-term support of water and electrolyte homeostasis by replacing normal ongoing losses in patients in which oral intake is not appropriate or possible. Thus hypotonic low-sodium fluids have historically been referred to as *maintenance fluids*. These crystalloids have a lower  $[Na^+]$  than the ECF. Given that normal insensible fluid losses (respiratory and other evaporative loss) do not contain  $Na^+$ , these fluids are indicated when the patient needs a supply of solute-free water to replace daily requirements normally met through drinking and metabolism of food. The  $[Na^+]$  concentration of

low-sodium crystalloid fluids ranges from 0 mEq/L in the case of 5% dextrose in water to 77 mEq/L in the case of a half-strength, or 0.45%, saline solution. Solutions with less than 77 mEq/L of  $\text{Na}^+$  contain 2.5 or 5% dextrose to raise the osmolality closer to that of ECF. Nonetheless, maintenance fluids are hypotonic and must be given slowly to allow for equilibration and to prevent hemolysis. It is important to note that dextrose, when present in these fluids, does not provide significant calories. The addition of dextrose is merely a way to raise the osmolality of the fluid (to make it isotonic with plasma) with a readily metabolized solute that will allow sodium-free water to be administered intravenously without causing hemolysis. It should be noted that the potassium content of the hypotonic low-sodium crystalloids is highly variable. As previously discussed, a potassium concentration of 20 mEq/L is the minimum that is usually considered necessary for true maintenance of a normokalemic patient.

**Table 5-2** lists some examples of low-sodium crystalloid fluids in green. The rate of administration of IV fluids used for maintenance is based on the metabolic body size and will only change when patients begin to eat and drink. Low-sodium crystalloids are contraindicated in patients requiring rapid administration, such as for hypovolemia, as doing so will rapidly reduce the ECF  $[\text{Na}^+]$  and cause cell swelling due to rapid reduction in osmolality.

### SUBCUTANEOUS ADMINISTRATION

Fluids administered to replace normal ongoing losses on a long-term basis are generally best provided orally, such as by means of an enteral feeding tube. This also allows for the essential provision of additional calories. These fluids can also be given subcutaneously in animals that are too ill to consistently maintain their hydration orally, such as cats with severe chronic kidney disease (CKD). Half-strength LRS with dextrose is an example of an appropriate fluid type for this type of subcutaneous administration. However the provision of fluids containing dextrose in the home setting may add risk, in that these fluids can support the growth of fungi and bacteria when single bags of fluid are generally reused for multiple days. LRS without dextrose is also an appropriate fluid type for this type of subcutaneous administration. Although classified as a high-sodium isotonic crystalloid, LRS is often used for subcutaneous fluid administration for cats that cannot maintain hydration orally. This fluid is slightly hypotonic to plasma and can provide some solute-free water. Anecdotally, LRS is better tolerated by patients during subcutaneous administration than is 0.9% sodium chloride or Normosol-R. However, LRS is still higher in  $[\text{Na}^+]$  than is optimal for maintenance use and may result in a higher  $\text{Na}^+$  intake than is ideal. This may promote hypertension in

predisposed patients and even hypernatremia when given chronically and at a high volume. An additional advantage of subcutaneous administration of LRS in cats with CKD is that this fluid is alkalinizing, which can be beneficial in managing the acidosis that often occurs in these patients.

### OTHER USES

**INFUSION VEHICLE** Because the rate of administration of the maintenance component of a fluid therapy plan is often constant, these fluids may be used as a vehicle for the continuous administration of drugs provided they are physically compatible with the fluid. Several references exist that provide compatibility information for various medications and fluid compositions. If drugs are administered in this way, clinicians must remember that adjustments in the fluid rate to meet the patient's changing fluid needs will also affect the rate of delivery of medications.

**HYPERTONIC DEHYDRATION** Low-sodium crystalloid fluids are also used to treat hypertonic dehydration, which is loss of water in excess of solute. These patients are hypernatremic. This type of dehydration is comparatively rare in cats; heat stroke (one of the most common causes) is more frequently encountered in dogs. However, hypertonic dehydration may be seen in cats that are inadvertently locked in closets or basements without water or in animals with hypothalamic disease manifesting as hypodipsia. This type of dehydration may also be seen in cats with hyperglycemic hyperosmolar syndrome.

### Combinations of High- and Low-Sodium Crystalloids

There is a common misconception that patients should only need to be given one intravenous fluid solution at any one time. There are many instances when both high- and low-sodium crystalloid fluids should be used concurrently. Patients that are not eating or drinking will need to have an ECF volume deficit replaced or will have ongoing excessive losses in addition to their maintenance needs. Combinations of fluids in these patients are often better able to meet their requirements than are prepackaged "maintenance" fluids.

Thus the cat will require a calculated amount of a high-sodium crystalloid to restore its ECF content back to normal baseline and replace any pathologic ongoing loss of high-sodium fluid, such as gastrointestinal losses or polyuria. The patient should also be given a low-sodium crystalloid to replace its normal daily losses of solute-free water. It is very important to remember that cats with inadequate caloric intake must also have those needs addressed very early in their hospitalization. Provision of nutrition through enteral or parenteral means

will also meet maintenance fluid needs, as mentioned previously.

### Hypertonic Saline Solutions

Hypertonic saline fluids contain sodium at concentrations substantially higher than the ECF; thus they facilitate rapid restoration of the ECF  $\text{Na}^+$  content during treatment of hypovolemic shock. Increasing the  $[\text{Na}^+]$  in the ECF produces a rapid, although transient, osmotically driven movement of water from ICF to ECF, which can occur far more quickly than when infusing isotonic high  $\text{Na}^+$  fluids alone. Because the ECF volume determines plasma volume, this fluid type is thus a rapid intravascular volume expander, at the expense of the ICF fluid volume. Hypertonic saline doses must be followed by infusions of isotonic high-sodium fluids to maintain their effect, insofar as they will rapidly equilibrate from the intravascular space into the ICF. Hypertonic saline will expand the intravascular volume by 2.5 to 3 mL for each mL infused.<sup>15</sup>

Clinical situations in which hypertonic saline may be useful in cats include resuscitation of animals with both hypovolemic shock and preexisting tissue edema, particularly cerebral edema resulting from traumatic brain injury. Hypertonic saline promotes rapid restoration of blood volume to improve blood flow to the brain while simultaneously decreasing cell volume, thus reducing brain edema. It also has positive inotropic effects on the myocardium. There are several contraindications to the use of hypertonic saline, including heart failure, uncontrolled hemorrhage, hypernatremia, and severe dehydration. Hypertonic saline is not appropriate as a treatment for chronic hyponatremia because of the risk for severe neurologic side effects that ensue when hypernatremia is corrected faster than 0.5mEq per hour.

Hypertonic saline solutions range in concentration from 3 to 23.4%. Solutions above 7.5% must be diluted before administration because they can cause phlebitis at the injection site. The dose in cats for the 7.5% solution is 2 to 4 mL/kg administered over 5 to 10 minutes.<sup>3</sup> Hypertonic saline may be given in combination with a colloid. One way to accomplish this is to add 1 part of 23.4% saline to 2 parts of a hetastarch; this yields a solution with a final concentration of just above 7.5%.

## Colloids

### General Characteristics of Colloids

Colloids are high-molecular-weight substances contained in a high-sodium solution, usually 0.9% saline. Unlike crystalloids, colloids will not readily diffuse through the vascular endothelium and will thus stay in the intravascular space longer than crystalloids. This effect is beneficial in achieving a sustained increase in intravascular volume when treating hypovolemia. In

states of low oncotic pressure, such as is seen with sepsis, systemic inflammatory response syndrome (SIRS), and hypoalbuminemia, colloids can theoretically plug leaks in capillary endothelium and prevent extravasation of fluids and the resultant edema.

Colloids are often used to replace and maintain intravascular colloid osmotic pressure (COP) and decrease edema that can result from the use of crystalloid fluids. Colloids are rarely used alone, however; they are typically used in conjunction with crystalloid fluids. Because of their efficient volume expansion, colloids can produce volume overload and pulmonary edema at lower volumes than crystalloids.

The crystalloid-versus-colloid controversy for resuscitation of hypovolemic patients has been an ongoing discussion in human and veterinary medicine for many years. Results of studies have failed to show a clear benefit to colloids, despite numerous theoretical advantages. Some studies have concluded that colloids promote decreased mortality, whereas other studies have shown better results with crystalloid therapy. Until more veterinary studies have been undertaken, it is difficult to make specific recommendations. One clinical approach to hypovolemic feline patients would be to begin with crystalloid therapy, reserving colloid therapy for patients who fail to respond. As previously discussed, hypothermic feline patients should be aggressively rewarmed concurrently with fluid therapy. For the markedly hypotensive patient, crystalloids, colloids, and potentially blood products may have to be provided concurrently.<sup>11</sup>

### Natural Colloids and Blood Products

Natural colloids include human albumin, blood products, and hemoglobin-based products. They are generally more expensive than the synthetic colloids, but there are certain situations in which they are preferred. The main use of fresh whole blood (FWB) and packed red blood cells (PRBCs) is in patients with symptomatic anemia. As a very general guideline, blood transfusion should be considered at a packed cell volume (PCV) below 20% (which corresponds to a hemoglobin concentration of 7 g/dL). However, the decision to transfuse should also be based on the patient's clinical signs and not on numeric values alone. More details on blood transfusion medicine are found in Chapter 25.

FWB contains all of the coagulation factors and platelets, so it can be used in patients with coagulation or platelet disorders. However, 6 to 8 hours after donation, platelet viability is markedly diminished. Fresh plasma, which is less than 6 to 8 hours old, has all of the coagulation factors but will not have platelets unless platelet-rich plasma is specifically prepared. Refrigerator storage of whole blood or plasma results in the gradual loss of the unstable or labile coagulation factors (factor VIII and von Willebrand's factor) within about 24 hours and

factor V and factor XI after about 1 week. What remains are the stable coagulation factors: II, VII, IX, X, and XIII. Fresh frozen plasma (FFP) is created by freezing plasma within 4 to 6 hours of collection. Freezing destroys platelets but preserves all coagulation factors. FFP should not be used for thrombocytopenia or thrombocytopathia. Plasma can be frozen for up to 1 year with the unstable coagulation factors preserved. Plasma that has been frozen after 6 hours of collection or kept frozen for longer than 1 year does not provide coagulation factors, only albumin, and is known as frozen plasma (FP).

Plasma or concentrated human albumin can also be used primarily for albumin replacement, although the volumes of plasma needed may be prohibitive. Albumin, although it has many functions, is the principal protein responsible for COP. Fluids containing albumin include concentrated albumin and some blood products.

The discussion of human albumin must be prefaced with a statement that its use in cats is considered controversial by many. Concentrated human albumin solution typically contains 20% or 25% albumin (200 or 250 mg/mL). By contrast, whole blood and plasma contain about 2.5% albumin; thus they will not increase intravascular COP or albumin as effectively and will require higher volumes.

Plasma can be used for albumin replacement and oncotic pressure support at a dosage of 20 to 30 mL/kg, but the volumes needed can risk volume overload and incur considerable expense. Concentrated 25% albumin may be the more appropriate colloid for resuscitation in patients with symptomatic hypoalbuminemia, particularly in postoperative or septic critical care feline patients.

Cats must always be blood typed before any blood product administration (human serum albumin does not require blood typing for cats), and donors must always be typed. A cross-match should ideally be performed for all transfusions but may not always be feasible because of the typically limited number of donors against which to cross-match.

Simply replacing albumin because the measured value is low is not indicated in the absence of clinical signs and exposes the patient to unnecessary risk. If possible, nutritional support by way of enteral or parenteral nutrition is the preferred means for normalizing serum albumin through hepatic synthesis, but this may not always be feasible. It should also be noted that in patients with protein-losing disorders, such as protein-losing enteropathies or nephropathies, albumin that is administered therapeutically will quickly be lost by the same routes as the patient's own albumin.

Albumin dose for transfusion of concentrated albumin solutions can be calculated through the following formula:

1. Calculate the total body plasma volume in deciliters: 4.5% of body weight (kg) × 10.

2. Calculate the total plasma albumin in grams (patient and target value): This is the patient's measured serum albumin level × plasma volume, as well as the desired albumin level × plasma volume (typically a value of 2 g/dL is sufficient for a target level).
3. Calculate the plasma albumin deficit; this is the difference between the patient's albumin level and the target level.
4. Because only 40% of the total body albumin resides in the vascular space (60% is interstitial), divide the plasma albumin deficit by 0.4 to obtain the total body albumin deficit in grams.
5. Calculate the volume of albumin needed, given that 1 mL of 25% albumin contains 250 mg of albumin. This is equivalent to multiplying the albumin deficit in grams by a factor of 4 for 25% and a factor of 5 for 20%.

An example calculation is given in **Box 5-2**.

As a general rule, administration of 1 mL of whole blood per kg body weight will raise the PCV by 1%. Thus for a rise of 10%, one would administer 10 mL/kg whole blood. PRBCs can be dosed at 75% of the whole blood dose because of the higher PCV.

Risks of using concentrated human albumin and blood products in cats are allergic transfusion reactions and volume overload.<sup>11</sup> Signs of mild allergic transfusion reactions may include fever, vomiting, urticaria, and facial swelling. More serious transfusion reactions include acute respiratory difficulty, hypotension, and circulatory collapse. Urine, serum, or plasma can show signs of hemolysis. Fatal reactions to mismatched blood products can and do occur, particularly if a type B cat is administered type A blood. Delayed reactions up to

## BOX 5-2

### Calculation of Albumin Dose for Transfusion of Concentrated Albumin Solution: A Case Example

The patient is a 5-kg cat that is 2 days postsurgery for a septic abdomen caused by a penetrating injury. There is generalized edema and persistent vomiting. The serum albumin is 1.5 g/dL.

1. Plasma volume:  $5 \text{ kg} \times 4.5\% \times 10 = 2.25 \text{ dL}$
2. Total plasma albumin:  $1.5 \text{ g/dL} \times 2.25 \text{ dL} = 3.375 \text{ g}$
3. Plasma albumin deficit using a target value of 2:
  - a. Target:  $2 \text{ g/dL} \times 2.25 \text{ dL} = 4.5 \text{ g albumin}$
  - b. Patient's albumin = 3.375 g
  - c. Deficit is  $4.5 - 3.375 = 1.125 \text{ g albumin}$
4. Total body albumin deficit is  $1.125 \text{ g}/0.4 = 2.8 \text{ g}$
5. Amount of 25% albumin solution to administer:  $2.8 \text{ g}/0.25 \text{ g/mL} = 11.2 \text{ mL}$

several weeks later have also been reported with human albumin administration. Cats should have capillary refill time (CRT), mucous membrane color, temperature, pulse rate, and respiratory rate monitored initially every 15 minutes during the first hour of administration, then hourly until the transfusion is completed.

When side effects do occur, the transfusion should be immediately discontinued, and additional therapy considered, such as diphenhydramine (1 to 2 mg/kg intramuscularly) for mild reactions. Fast-acting corticosteroids, such as dexamethasone sodium phosphate (0.1 to 0.2 mg/kg intramuscularly or intravenously) may be used for severe transfusion reactions but are generally not needed for milder reactions. An assessment should be made to determine if there is a continued need for blood products but at a slower infusion rate. Re-evaluation of donor compatibility through further cross-matching should be considered. Because of the possibility that blood products may be contaminated with bacteria, submission of a sample for bacteriologic testing should be considered. Pretreatment with corticosteroids or antihistamines is not routinely indicated unless a patient has experienced a transfusion reaction in the past.

Once the unit of blood or plasma has been entered ("spiked" with the administration set), the transfusion should be completed within 4 hours to minimize the risk of bacterial contamination. Unused product should be refrigerated immediately and used within 24 hours, then discarded. (For instance, if a kitten were to receive a partial unit of blood, the volume to be administered would be drawn off under sterile conditions and transfused. The remainder of the unit could be refrigerated immediately, stored for 24 hours, and then administered.)

Oxyglobin (bovine hemoglobin glutamer-200, Hemopure) is another natural colloid. It is a hemoglobin-based oxygen-carrying solution used for the treatment of symptomatic anemia, and the dosages are the same as for whole blood. A benefit of Oxyglobin is that it has universal compatibility; therefore no blood typing or cross-matching is necessary.

There are considerable risks with the use of Oxyglobin in cats, and the veterinarian should carefully conduct a risk assessment for each patient before using it. When larger volumes are infused, it produces a dark plasma color, which interferes with enzyme-based chemistry analyzers (e.g., the Vet-Test Chemistry Analyzer, IDEXX Laboratories, Westbrook, Maine); thus these results may not be reliable. Side effects are most common in euvolemic patients, such as patients with immune-mediated hemolytic anemia. It is contraindicated in cats with known or suspected cardiac disease. Side effects include volume overload, pulmonary edema, vomiting, diarrhea, and hypertension. When side effects arise, the veterinarian should administer small quantities slowly

and titrate to effect. Oxyglobin is not currently available commercially.

### **Synthetic Colloids**

The most commonly used colloids are the synthetic hetastarch solutions. Hydroxyethyl starch is a synthetic polymer of glucose, a polysaccharide closely resembling glycogen. Available hydroxyethyl starch products include both hetastarch and pentastarch. 6% Hetastarch is formulated either in 0.9% sodium chloride or in LRS. The difference between the two is that hetastarch with LRS also contains calcium, magnesium, a small amount of dextrose, and a lower  $[Cl^-]$ .

Hetastarch contains very large molecules that must be broken down before they leave the vasculature, which generates a long-lasting colloid osmotic pressure effect. Hetastarch may decrease inflammation in the intravascular space and decrease vascular membrane permeability by plugging leaks in vascular endothelium in cases such as septic shock and SIRS patients.<sup>4</sup>

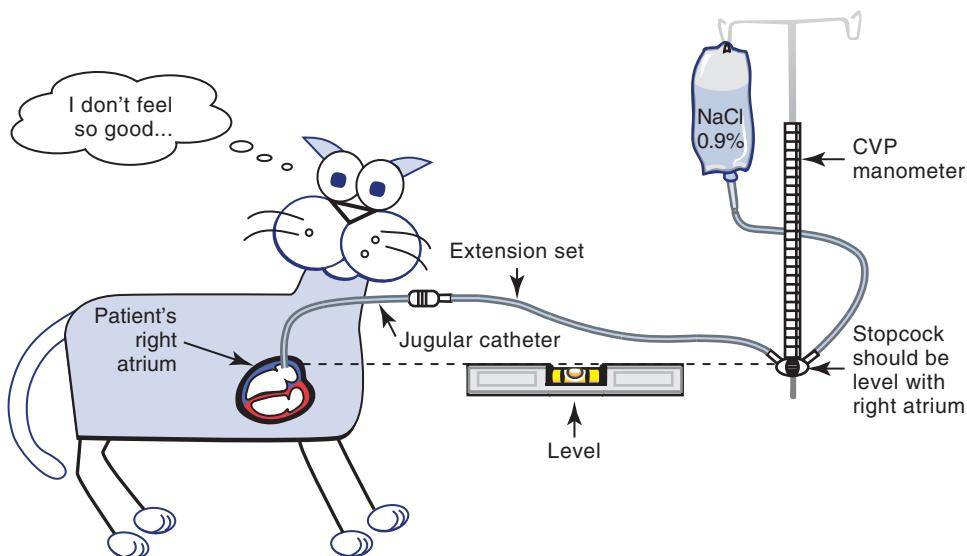
Rapid infusions within 5 to 10 minutes in cats can result in vomiting and transient hypotension. This effect can be mitigated by using hetastarch in small volume increments to effect (on the order of 5 mL/kg) over 10 to 15 minutes. Total dose for use in shock and hypotension is 10 to 20 mL/kg.

Hetastarch can interfere with clotting; however, the presence of calcium in hetastarch with LRS may reduce this effect. Clinical evidence of bleeding has not been reported in patients receiving 6% hetastarch at doses up to 20 mL/kg/day. If the activated clotting time or partial thromboplastin time rises above 50% of the high end of the reference range, concurrent coagulation problems should be investigated. 10% Pentastarch contains a narrower range of hydroxyethyl starch molecule size. Without the largest molecule sizes present in hetastarch, pentastarch breaks down more quickly and is eliminated from the body more rapidly. It has less of an effect on blood coagulation than hetastarch does.

An important objective when using colloids is to infuse the smallest volume needed to achieve resuscitation endpoints, such as normalized heart rate, improved blood pressure, oxygen delivery, and lactate clearance. Cats are very susceptible to volume overload, which can result in serious and disastrous consequences, so careful monitoring is essential.

Because most patients receiving colloids are usually concurrently given isotonic crystalloids, the crystalloid volume for intravascular volume replacement and hydration can be decreased by 40% to 60% of the volume calculated for crystalloids alone.

When more than one type of colloid is indicated, the colloids are usually administered consecutively rather than simultaneously. For example, a patient with hemorrhagic shock caused by severe trauma may receive whole blood to correct hypotension and hypovolemia, then



**FIGURE 5-4** Monitoring of central venous pressure (CVP). A central catheter with the tip in the cranial or caudal vena cava can be used to guide fluid therapy and resuscitation. The normal CVP is 0 to 5 cm H<sub>2</sub>O.

hetastarch for colloid maintenance during acute recovery. Bolus infusions of colloids are titrated in succession, and monitoring for endpoints of resuscitation is performed between boluses.

As previously discussed, cats should be monitored frequently for signs of volume overload, which include serous nasal discharge, jugular distention, chemosis, tachypnea, reduced lung sounds (suggestive of pleural effusion), moist lung sounds (suggestive of pulmonary edema), and subcutaneous edema. As shown in *Figure 5-4*, monitoring of central venous pressure (CVP) through a central catheter with its tip in the cranial or caudal vena cava (see section on vascular access later in this chapter) can also be used to guide fluid therapy and resuscitation. The normal range for CVP is 0 to 5 cm H<sub>2</sub>O; marginal volume overload exists at 5 to 10 cm H<sub>2</sub>O; serious volume overload occurs at amounts greater than 10 cm H<sub>2</sub>O. Because of the considerable variability among operators, consistency in technique and positioning is vital. Trends in CVP are more clinically useful than any single measurement. CVP monitoring will be discussed in greater detail in the section on monitoring therapy for hypovolemia.

Colloids are relatively contraindicated in patients with active, uncontrolled hemorrhage because they may potentially interfere with platelet function and may exacerbate hemorrhage.

## ROUTES OF ADMINISTRATION

### Subcutaneous Fluid Therapy

Cats with mild illness or those requiring fluid therapy at home are candidates for subcutaneous fluid

administration. Subcutaneous fluid therapy is not appropriate for cats with severe dehydration, hypovolemia, hypotension, or critical illness. Isotonic high-sodium crystalloid fluids (e.g., LRS) are most often used. The volume that can be delivered at any one site is limited by the distensibility of the subcutaneous tissue, which is generally favorable in the interscapular region of the upper torso. In most adult cats, fluid pockets of 50 to 150 mL can be accommodated without patient discomfort. Owners should be notified that fluid will settle ventrally with gravity and should disperse within a few hours. Just how much is too much is determined by the development of a painful, firm fluid pocket that leaks fluid from the hole created by the needle.

Although isotonic, high-sodium fluids are preferred, the following broader guidelines may apply to subcutaneous fluids. The osmolality of the fluid being administered should not exceed 450 mOsm/kg and the fluid should contain at least 40 mEq/L of sodium. The maximum concentration of K<sup>+</sup> should not exceed 40 mEq/L, although pain responses have been reported at even lower doses.<sup>6</sup> Fluids containing magnesium have also been observed to cause discomfort.

## Intravenous Fluid Therapy

### Venous Access

Vascular access is indicated for the following clinical scenarios:

- The patient needs fluid or colloid boluses to manage hypotension.
- The patient needs therapy for rehydration, nutrition, or electrolyte imbalances that cannot be met through enteral or subcutaneous means.

- The patient needs hemodynamic monitoring (CVP).
- The patient requires continuous infusions of analgesics or vasoactive substances.
- The patient requires other intravenous medications or transfusion of blood products.
- The patient needs frequent blood sampling (e.g., those with DKA).

If a particular patient does not fit into one of these scenarios, there may not be a need for intravenous access or intravenous fluids. Simply being hospitalized is not an indication for an intravenous catheter, unless the patient's status is uncertain and vascular access may be necessary. Consideration should be given to enteral provision of medications, nutrition, and fluids if the gastrointestinal tract is functional.

There are five main types of intravenous catheters: peripheral catheters, central venous catheters, peripherally inserted central catheters (PICC lines), intraosseous catheters, and winged infusion sets. **Table 5-6** summarizes the features of these catheters.

Catheter gauge reflects the internal diameter of the catheter; the smaller the gauge number, the larger the diameter. If rapid fluid administration is required, it is advisable to choose the largest diameter catheter that fits within the vessel. Intravenous fluids can be administered most quickly through shorter, larger diameter catheters. For cats weighing less than 5 kg, 24- to 22-gauge catheters will be appropriate for routine fluid therapy, but smaller diameter catheters are prone to more technical problems. Most cats in the 5- to 7-kg weight range can accommodate a 22-gauge catheter for routine fluid therapy. Larger (18- to 20-gauge) catheters are useful for resuscitative therapy.

Venous catheters should not be placed in limbs that are traumatized or painful. Animals with severe coagulopathies, including hypercoagulability, should not have catheters placed in the jugular vein, if at all possible. A cat with an aortic thromboembolism should not have a catheter placed in either of the pelvic limbs.

The cephalic and accessory cephalic veins of the thoracic limbs or the lateral saphenous vein of the pelvic limbs are the most common peripheral veins catheterized in feline patients. Alternative vessels include the medial saphenous veins and the femoral veins. The peripheral vessels of hypotensive cats may not be easily observable, although the lateral and medial saphenous veins are often most visible on account of the thin overlying skin in these areas.

If the skin or subcutaneous tissues in the area of the catheter are compromised, that site should be avoided to decrease the risk of infection and thrombosis. If the leg and catheter wrap are likely to be soiled, the risk of a catheter-site infection is increased. Patients with diarrhea, urinary leakage, or vaginal discharge should have

catheters placed in the thoracic limb. Vomiting patients should have the catheter adequately wrapped or have a hind limb catheter. The appropriate choice of vein should minimize the risk of the patient damaging or removing the catheter. Generally, a light wrap will be sufficient to prevent most cats from attempting to remove their catheters; however, some may require an Elizabethan collar to prevent premature removal.

Operator expertise and the temperament of the patient also play a role in deciding where the catheter should be placed. It can sometimes be a challenge to place intravenous catheters in animals that are extremely small, hypovolemic, or challenging to restrain. Newly trained veterinary professionals should place the catheter in a vessel that is readily visible and amenable to catheterization. Experienced veterinary professionals should make the initial catheter attempts when placing larger catheters in critically ill patients, especially if the vessels are difficult to visualize. Animals that are fractious should not have catheters placed in the thoracic limbs because it may be difficult to gain access to the catheter without risk of staff injury.

The overall treatment goal for the patient should be considered when deciding on catheter type and location. Any vein may be used for short-term administration of intravenous fluids. A centrally located catheter or PICC line may be required for the following:

- Long-term (>3 to 5 days) fluid or IV drug administration
- Parenteral nutrition
- Treatment with hypertonic or irritating fluids or drugs
- Concomitant delivery of incompatible drugs or fluids
- Frequent phlebotomy sampling
- CVP monitoring
- Transvenous pacing

### **How to Place an Intravenous Catheter**

The jugular or saphenous veins are most commonly used to gain access to the central venous circulation. Preparation for placement of a catheter consists of several steps: gathering all necessary supplies, clipping the catheter insertion site with a well-lubricated 40 blade, applying a surgical prep to the catheter insertion site, and occluding the vein. When establishing intravenous access, caution should be used in very ill, dyspneic, or compromised patients, for whom the stress of physical restraint could cause an acute decompensation of their condition.

Sites for indwelling catheters should be aseptically scrubbed with sterile gauze squares freshly soaked with antimicrobial solution, alternating with either alcohol or sterile water. A total of three scrubs should be performed, and contact time of the scrub solution with the skin surface should be 3 to 5 minutes. Care should be taken

**TABLE 5-6** Features of Catheters that Are Used to Gain Vascular Access in Cats

Type of Catheter	Indications	Advantages	Disadvantages	Comments
Peripheral	Short-term fluid and/or medication administration	Ease and speed of placement Low complication rate	Tip may fray, damaging vein Often becomes nonpatent May be resented or dislodged by patient	Available as over-the-needle or through-the-needle (former used more commonly)
Central venous	Hyperosmolar solutions (<600 mOsm/L) Parenteral nutrition Long-term fluid or medication administration Caustic drugs Frequent blood sampling CVP monitoring	Large gauge allows blood sampling Patency maintained longer	More difficult and time-consuming to place More expensive	Multiple lumen catheters can facilitate blood sampling and concurrent fluid and parenteral nutrition administration
Peripherally inserted central catheter (PICC)	Coagulopathy Hyperosmolar solutions (<600 mOsm/L) Parenteral nutrition Long-term fluid or medication administration Caustic drugs Frequent blood sampling CVP monitoring	Allows frequent blood sampling Patency maintained longer	More difficult and time-consuming to place More expensive	Inserted into a peripheral vein and terminating in a great vessel Positioning should be verified with radiographs
Intraosseous	Very small patients (e.g., kittens)	Avoid difficult catheter placement in small veins	Cannot use to take blood samples	18-gauge needle Sites include proximal humerus, intertrochanteric fossa of femur, and proximal tibia
Butterfly catheter	Very short-term administration of medications or small volumes of fluid	Ease and speed of placement	Tip may lacerate vein	Should not be bandaged in place or left unattended

CVP, Central venous pressure.

during shaving and scrubbing not to damage the skin surface; this can increase the chance of catheter-site infection.

Appropriate antiseptic agents include chlorhexidine gluconate 4%, chlorhexidine diacetate 2%, or povidone-iodine. The clinician should remove the residual scrub solution from the skin and surrounding hair with gauze sponges soaked in alcohol, sterile water, or sterile saline solution. Residual soap is removed from the skin to avoid irritation. The person placing the catheter should wash his or her hands and apply a germicidal lotion before catheter placement. Clean examination gloves are recommended during catheter placement of peripheral intravenous catheters, and sterile gloves are donned during PICC and central line placement.

For central lines or in patients with thick skin, it may be helpful to make a facilitating incision to ease catheter passage and decrease damage to the tip of the catheter associated with passage through tissue. To create a facilitating incision, the clinician should slide the skin overlying the venipuncture site to either side of the vein, make

an incision about 0.5 mm long using a the bevel of a 20-gauge needle, and then let the skin return to its original position. The incision should now be ideally placed over the vein. The catheter is inserted through the newly created incision.

The final preparation step in catheter placement is to occlude the vein proximal to the anticipated insertion site. The assistant should make sure the vessel is in a position that will be conducive to catheterization. For cephalic catheterization, this is accomplished by rolling the vessel laterally (usually with the thumb that is occluding the vein). The assistant should also ensure that the patient's head is properly restrained so the cat cannot cause harm during the procedure. Trimming nails before catheterization may also help decrease scratch injuries. Some cats require at least two people to properly position and restrain them, whereas others are more compliant with less restraint. Chemical restraint may be needed for some cats. Caution should be used in very ill or compromised patients, for whom the stress of physical restraint could cause an acute decompensation of their condition.



**FIGURE 5-5** Multiple fluid lines can be attached to one intravenous catheter with the use of three-way connectors.

For peripheral catheter insertion, the limb is stabilized and the catheter inserted with the bevel up at an approximately 15-degree angle to the vessel. The catheter is then advanced into the vessel a few millimeters, making sure to stay parallel to the vessel so it does not penetrate the far vessel wall. When blood appears in the hub of the stylet or catheter, the needle and catheter are advanced together approximately 1 to 4 mm to ensure the end of the catheter is entirely inside the lumen of the vessel. The catheter is then advanced off of the stylet and into the vessel. The stylet is removed, and either an injection cap or T-connector is placed on the end of the catheter and flushed with saline. Multiple fluid lines can be attached to one intravenous catheter with the use of three-way connectors (Figure 5-5).

Signs that indicate the catheter is not in the vein include resistance to injection or a visual bleb of fluid forming in the subcutaneous space when a small volume of saline is injected.

Once the catheter has been inserted and the stylet removed, the catheter should be taped in place (for peripheral catheters) or sutured in place (for most central lines). Catheters that will be left in place for more than a few minutes should be covered with a light dressing that protects the catheter from traction, damage, or contamination (Figure 5-6). Addition of antibiotic ointment is not necessary. The catheter wrap should be thick enough to protect the catheter but not so occlusive that moisture will accumulate. The catheter wrap should allow daily visual inspection of the insertion site. For long-term peripheral catheters, typically a layer of cast padding is placed over the tape, followed by stretch gauze, and then adhesive or self-adhesive bandage material. The material should be wrapped snugly but should not occlude venous return or edema will result. Catheters intended for very short-term use (minutes to



**FIGURE 5-6** A properly wrapped jugular catheter will still allow the patient to eat and drink.



**FIGURE 5-7** Swelling distal to a catheter is commonly caused by an excessively tight bandage or tape.

a few hours) can be wrapped with adhesive tape alone, if desired.

Once the catheter has been properly placed and secured, it is necessary to monitor and maintain it to ensure proper function and minimize the risk of infection. Indwelling venous catheters should be unwrapped and the insertion site examined and cleaned every 24 hours or more frequently if the catheter wrap is soiled. Swelling distal to a catheter is commonly caused by an excessively tight bandage or tape (Figure 5-7).

There is no evidence in veterinary patients that the common practice of routine catheter replacement every 72 hours is warranted. If a catheter is functional without any signs of thrombophlebitis, the catheter should be closely monitored and can be left in place. The purpose of catheter monitoring is to identify any of the following signs of thrombophlebitis:

- Thrombosis, or formation of a thrombus on the catheter or vessel wall
- Embolism, or breakage of a portion of the catheter into the circulation
- Subcutaneous fluid infiltration or leakage at the insertion site
- Infection or purulent discharge
- Pain on injection
- Otherwise unexplained fever

Should any of these complications be identified, removal of the catheter should be considered. Culture of the catheter tip is recommended in patients with evidence of catheter site infection or unexplained fever.

Catheters that are used for continuous fluid therapy do not need to be flushed routinely but should still be visually inspected. Catheters that are not in use should be either removed or flushed every 4 to 6 hours using 0.9% NaCl with or without the addition of 1 U/mL of heparin.

To decrease the risk of bacterial contamination, administration sets should be sterile and clean technique, including clean hands and disposable gloves, should be used to attach and detach the system when changing fluid bags. A T-connector should be used to help prevent the catheter from being pulled out from the insertion site and should be clamped whenever fluids are not flowing through the catheter. Needleless connection systems minimize contamination of the catheter and fluid lines by decreasing entry into the system, but these are considerably more expensive than conventional fluid sets.

The fluid administration tubing should be anchored to the catheter bandage with tape to prevent premature dislodgment. If the T-connector is securely taped to the leg, this may not be necessary, but it provides extra security in active patients. If a needleless connector is used, the fluid line should be disconnected before moving a patient, if possible.

Intravenous tubing and fluid bags should be changed every 24 to 72 hours. Injection ports and needleless connectors should be swabbed with 70% isopropyl alcohol before needle puncture or drug administration. All injection caps should be replaced after approximately 20 penetrations or if there is any observed fluid leakage.

## FLUID THERAPY PLANS AND MONITORING

### Management of Hypovolemia

#### **Assessment of Hypovolemia**

Several parameters can be used to estimate the adequacy of circulating blood volume, although it is a physiologic variable that is almost impossible to quantify *in vivo*. It

is important to assess several parameters because no single one can accurately depict overall cardiovascular status. Any of the following clinical signs can be associated with hypovolemia:

- Decreased jugular venous distention
- Poor pulse quality
- Hypotension
- Low CVP
- Hypothermia
- Tachycardia
- Pale mucous membranes
- Prolonged CRT
- Cool extremities
- Oliguria or decreasing urine production
- Metabolic acidosis
- Hyperlactatemia
- Decreased central venous oxygen

Many of these parameters can also be influenced by abnormalities in other body systems and are neither sensitive nor specific for hypovolemia in cats. Tachycardia deserves special mention, insofar as cats can have profound hypovolemia without the tachycardia commonly seen in other species. Important considerations for cats in shock are listed in Box 5-3.

#### **Therapy Options for Hypovolemia**

Correction of hypovolemia and restoration of tissue perfusion are the primary and immediate goals of fluid therapy in any patient with decreasing intravascular volume resulting from blood loss, severe dehydration, or shock. This can be accomplished in different ways depending on the cause and degree of the hypovolemia and the patient's response to therapy. Correction of hypovolemia should be accomplished rapidly and, in cats, should be undertaken in concert with establishment of normothermia and aggressive rewarming, given the link between vasomotor tone and body temperature that is unique to this species.

#### **BOX 5-3**

#### **Important Considerations for Cats in Shock**

- Cats are not small dogs.
- Cats are less tolerant of volume overload than dogs.
- Cats in shock may not be tachycardic (as seen in dogs).
- Blood pressure is more difficult to measure accurately in cats.
- Hypothermic cats cannot regulate blood pressure and are therefore more prone to volume overload; external rewarming should occur before aggressive fluid therapy to avoid volume overload.

Hypovolemia may be treated initially with high-sodium isotonic replacement crystalloids, which are generally available in every practice and very economical. Most crystalloid fluids are redistributed to the interstitial space within 30 to 60 minutes of administration and thus can cause interstitial edema before an acceptable effective circulating blood volume is attained. When edema or hemodilution occur before hypovolemia has been corrected, it may be advisable to add an appropriate colloid or blood product depending on the cause of the hypovolemia.

When intravenous crystalloids redistribute to the interstitial space, the ensuing interstitial edema can result in decreased cellular perfusion. To mitigate this effect, the flow of fluid in the lymphatic system is increased in response to the higher hydrostatic pressure. High concentrations of albumin exist within the lymphatics, and this is rapidly returned to the systemic circulation. This is known as the *protein pump*. The resultant increase in plasma albumin concentration increases intravascular oncotic pressure, which reduces the interstitial fluid volume and increases the intravascular volume through changes in Starling's forces.

The feline blood volume is 50 to 60 mL/kg, which is often cited as both the daily fluid requirement (i.e., maintenance fluids) and the shock dose of fluids. Volumes and rates of fluid administered to treat hypovolemia and dehydration in cats must be considerably lower than those used in dogs because of the risk of fluid overload. To correct hypovolemia, a low dose of a replacement crystalloid is initially given, such as 25% of the shock dose, or roughly 10 to 15 mL/kg. A useful short-cut for calculating 25% of the shock dose of crystalloids for a cat is to add a "0" to the cat's body weight in kg. Thus 25% of the shock dose for a 5-kg cat would be 50 mL. Cardiovascular and physical examination parameters are then reassessed, and additional aliquots of fluid are administered until the cardiovascular status has improved or a full shock dose of fluids has been administered. Because of the risk for fluid overdose in cats, overly aggressive fluid therapy to rapidly achieve normal cardiovascular parameters cannot be recommended. The goal is to ensure resuscitation sufficient that the patient's status does not deteriorate as a result of hypovolemia, as well as prevent organ injury caused by poor perfusion. Any remaining deficit caused by dehydration can be corrected over the next 6 to 24 hours as part of rehydration fluid therapy.

An additional crystalloid that can be used to treat certain types of hypovolemia is hypertonic (usually 7%) saline. Hypertonic saline provides greater blood volume augmentation per volume administered than isotonic replacement crystalloids and has a positive effect on cardiac output. It has similarly transient effects to isotonic crystalloid fluids. It is most efficacious in patients with head injuries and those experiencing arrest because of its tendency to decrease cerebral edema. The efficacy

of hypertonic saline is limited because repeated doses will cause significant hypernatremia, and electrolytes should be closely monitored during its use. Additionally, crystalloid fluids must be concurrently administered to offset the fluid shifts that take place from the interstitial space into the intravascular space. Hypertonic saline can be used in hypovolemia resuscitation at a dosage of 2 to 4 mL/kg, administered over 10 to 15 minutes. It is contraindicated with existing hypernatremia or severe dehydration.

Colloids can also be used to treat hypovolemia, with similar cautions regarding fluid overload in the feline patient as with crystalloid fluids. Colloids have the advantage of mobilizing interstitial fluid into the intravascular space and will actually increase the effective circulating volume by a greater amount than that infused. Additionally, colloids will remain in the intravascular space longer than crystalloids because they must be enzymatically degraded before elimination. For these reasons they are effective for the therapy of hypovolemia, low oncotic pressure, and shock. Total daily dose (typically used for oncotic support in hypoalbuminemic patients or those with vasculitis) as well as shock doses are in the range of 10 to 20 mL/kg. For shock, as for crystalloid therapy, 25% to 33% of the fluid dose (5 to 7 mL/kg) is administered rapidly over the first 5 to 10 minutes, and the patient's physical examination and hemodynamic parameters are reassessed.

### **Monitoring Response to Therapy in Patients with Hypovolemia**

Monitoring is an essential component of fluid therapy and allows for ongoing adjustments in the fluid prescription to meet patient needs. Fluid therapy is a dynamic process requiring active and vigilant monitoring to ensure effectiveness, prevent complications, and meet treatment goals. Endpoints for resuscitation are given in Box 5-4.

### **BLOOD VOLUME STATUS PARAMETERS**

Hypovolemia should be addressed in patients before therapy for dehydration. Volume status is classically divided into preload and forward flow parameters. The Frank-Starling law of the heart dictates that cardiac output is related to end-diastolic volume, a component of cardiac preload; for this reason adequate venous return is vital to ensuring appropriate perfusion. Preload parameters are indicators of the adequacy of venous return to the heart and include venous volume and cardiac chamber diameters. Venous volume cannot be directly measured *in vivo* and must be estimated by assessing the ease of venous distention, central venous pressure, and radiographic diameter of the caudal vena cava.

In a patient with normal blood volume, both jugular and peripheral veins should distend easily when

**BOX 5-4****Resuscitation Endpoints**

- Improved mentation
- Normal blood pressure (>90 mm Hg systolic)
- Normal heart rate
- Warm extremities
- Improved capillary refill time
- Urine output >1-2 mL/kg per hour
- SVO<sub>2</sub> >70% (mixed venous oxygen saturation): the SPO<sub>2</sub> of blood being returned to the heart—a measure of how much oxygen the tissues are extracting. Low SVO<sub>2</sub> indicates either excessive oxygen demand by the tissues or inadequate oxygen supply.

occluded. Lack of venous distention in vessels above the level of the heart may indicate hypovolemia. Obviously, this is a subjective assessment, but it can give some idea of relative volume status.

Venous volume can also be indirectly assessed by measuring CVP. CVP is the hydrostatic pressure of the blood entering the heart, as measured by a catheter with its tip in the right atrium or vena cava. CVP is proportional to the volume of blood in the anterior vena cava and venous tone. This pressure is decreased by hypovolemia or venodilation and is increased by fluid therapy or vasoconstriction. Several other factors can contribute to the accuracy of CVP measurement, such as cardiac or respiratory pathology, making it a somewhat unreliable (but useful) physiologic variable.

CVP can be measured with a column manometer (the most common method) or a direct pressure transducer. The normal range is 0 to 10 cm H<sub>2</sub>O. However, because of variations in venous tone and other technical factors, single CVP values are often difficult to interpret without the aid of other monitoring. Normal and abnormal values can overlap; for example, CVP can range from -5 to +5 cm of H<sub>2</sub>O in hypovolemic animals and from 5 to 15 cm H<sub>2</sub>O in animals with volume overload. Therefore measurements should be considered meaningful to the fluid therapy prescription only if they are below 0 or above 10 cm of water and, more important, if the overall trend in CVP is considered. Taking into account all available parameters, if a patient's CVP is consistently below 0 cm H<sub>2</sub>O, consideration should be given to either a bolus of fluids or an increased rate of fluid administration. If a patient's CVP value is consistently above 10 cm H<sub>2</sub>O, fluid administration should be slowed or discontinued, and diuretic administration should be considered.

CVP measurements are primarily indicated during volume restoration for shock and in patients for whom volume overload is a concern, such as patients in acute renal failure or those with concurrent cardiac disease.

Observation of correct technique for CVP measurement is very important, insofar as there is considerable interoperator variability. For the most accurate and clinically useful results, the patient's position should be recorded in the medical record and the same staff should perform the readings whenever possible.

To measure CVP (see [Figure 5-4](#)), the patient is positioned in right lateral recumbency and the level of the right atrium (near the manubrium—the cranial tip of the sternum) is identified. The clinician should ensure that the stopcock is level with the right atrium (this line is known as the *phlebostatic axis*) using a bubble level. This serves as the reference point and is the "zero" mark on the manometer.

With the stopcock closed towards the patient, the clinician opens the fluid bag line and fills the manometer to about 25 to 30 cm H<sub>2</sub>O. The clinician then opens the stopcock toward the patient (turning it off toward the fluid bag) and allows the fluid in the manometer to run into the patient. At some point the fluid will begin to oscillate with the patient's heartbeat and will stop falling as it equilibrates with the pressure in the vena cava (usually about 25 to 30 seconds); this is the CVP measurement and should be noted in the patient's record.

Venous volume can also be very roughly estimated by evaluating the diameter of the caudal vena cava on a lateral thoracic radiograph. Normal diameter is roughly equivalent to one rib width. A small caudal vena cava diameter suggests hypovolemia, and further fluid administration may be indicated depending on the patient's status. A large caudal vena cava diameter may suggest hypervolemia or heart failure, and the fluid therapy prescription should be re-evaluated.

**PARAMETERS RELATED TO LARGE ARTERIES**

Physiologic parameters related to large arteries include pulse quality and arterial blood pressure. Pulse quality assessment through palpation is a reflection of stroke volume and the difference between systolic arterial pressure and diastolic arterial pressure, not a measure of blood pressure. For this reason, poor pulse quality does not always correlate with hypotension. Poor pulse quality can be associated with small stroke volumes, vasoconstriction, or hypotension. Hypovolemia may be the most common cause of poor pulse quality, but it is also a feature of other conditions, such as poor cardiac contractility, tachycardia, restrictive heart disease, aortic stenosis, and positive pressure ventilation, none of which absolutely requires fluid therapy. If the cause of the poor pulse quality is determined to be hypovolemia by corroborating history, clinical signs, and other measurements, then fluid administration may be indicated.

Mean arterial blood pressure is the average pressure of the pulse pressure waveform. Systolic arterial blood pressure is the highest pressure of the waveform and is primarily determined by stroke volume and arterial wall

compliance. Diastolic arterial blood pressure is the lowest pressure before the next stroke volume and is primarily determined by systemic vascular resistance and heart rate. Arterial blood pressure can be measured indirectly (with a Doppler transducer, occlusion cuff, and sphygmomanometer) or directly (with an arterial catheter by way of a pressure transducer). Normal systolic pressure measurements range between 100 and 160 mm Hg, mean pressure measurements between 80 and 120 mm Hg, and diastolic pressure measurements between 60 and 100 mm Hg.

The determinants of blood pressure (BP) are as follows:

$$\text{BP} = \text{CO} \times \text{SVR}, \text{ where CO is}$$

cardiac output in mL/min and SVR is

systemic vascular resistance in dynes/sec/cm<sup>5</sup>.

Determinants of CO are stroke volume (SV) and heart rate (HR). Taken as a whole, the equation is as follows:

$$\text{BP} = (\text{HR} \times \text{SV}) \times \text{SVR}$$

From this relationship, it can be seen that in order to maintain constant BP (an analog for perfusion) in the face of falling cardiac output (as with hypovolemia), either the heart rate has to increase or the systemic vascular resistance (as mediated by catecholamine release) must rise.

Arterial blood pressure is not a very good measure of circulating blood volume because animals have a great ability to compensate for alterations in blood volume to preserve blood pressure. Eventually, however, severe hypovolemia will result in hypotension as heart rate may be maximized or the systemic vasculature may be maximally constricted.

Hypotension may be caused by hypovolemia, poor cardiac output, or excessive vasodilation, as illustrated by the previous formula. If the cause of the hypotension is hypovolemia, further fluid therapy is indicated. Hypotension resulting from vasodilation, as seen with hypothermia in cats, should be treated with cautious fluid therapy concomitantly with external rewarming. When inappropriate vasodilation is due to vasoplegia, as with sepsis or distributive shock, treatment should consist of fluid therapy coupled with vasoactive substances (pressors or catecholamines) such as phenylephrine or dopamine. It is important to remember that catecholamines should not be administered until the veterinarian is fairly certain that adequate circulating volume has been restored, because their use while the patient is hypovolemic will only worsen ischemia, not improve it. When hypovolemia is due to heart failure, further fluid therapy is not indicated and disease-specific therapy is required.

Another category of forward flow parameters is made up of variables that reflect precapillary arteriolar vasomotor tone. Increased vasomotor tone, or vasoconstriction, commonly occurs in conditions such as

hypovolemia, heart failure, hypothermia, and administration of vasoconstrictors. Decreased vasomotor tone, or vasodilation, commonly occurs in conditions such as septic shock, hypothermia or hyperthermia, and administration of vasodilators and some anesthetics. Arteriolar tone can be assessed by monitoring mucous membrane color and CRT.

Normal mucous membrane color is pink. Vasoconstriction decreases capillary perfusion, causing the color of the mucous membrane to change from pink, to pale pink, and then to white as vasoconstriction progresses in severity. Vasodilation increases capillary perfusion, causing the color of the mucous membrane to change from pink to red as vasodilation progresses in severity. Ambient lighting, visual acuity, and patient pigmentation can make absolute assessment of mucous membrane color problematic, and it should be evaluated in concert with other variables and physiologic parameters. The most common sites to observe mucous membrane color are the unpigmented gingiva, tongue, and conjunctiva.

Deoxygenated hemoglobin causes a bluish discoloration of the capillary beds as cyanosis progresses in severity. Cyanosis is usually caused by hypoxemia, which should prompt an evaluation of pulmonary function, but it occasionally can be caused by sluggish capillary blood flow or diminished cardiac output, which necessitates further evaluation of cardiovascular function.

Anemia causes pale mucous membrane color and may warrant a hemoglobin infusion. Mucous membrane color is determined by the amount of oxygenated hemoglobin in the visible capillary beds. Arteriolar vasomotor tone determines the amount of blood in the observed capillary beds.

CRT is primarily determined by arteriolar vasomotor tone. CRT is assessed by using digital pressure on mucous membranes until they turn white and then noting how long it takes for normal color to return. Normal CRT is 1 to 2 seconds. The most common site to evaluate CRT is on unpigmented gingiva. Vasoconstriction, which prolongs CRT, may be caused by hypovolemia, heart failure, hypothermia, and administration of vasoconstrictors. If the patient's history and other cardiovascular parameters suggest hypovolemia, fluid therapy is indicated. Vasodilation shortens CRT. Vasodilation may be caused by septic shock, hyperthermia, and administration of vasodilators and some anesthetics. Vasodilation may cause hypotension, and blood pressure should be assessed in these patients.

Another forward flow category, tissue perfusion, includes extremity temperature, urine output, lactic acidosis, and central venous partial pressure of oxygen. Urine output can be used in cats with functioning kidneys as an indirect measure of renal perfusion and vital organ perfusion. Hypovolemia and dehydration

reduce renal perfusion and GFR. In both situations there will be avid tubular reabsorption of  $\text{Na}^+$  and glomerular filtrate, thus reducing the urine volume. A decrease in urine volume is thus expected with hypovolemia or dehydration if renal function and urine concentrating ability are normal.

Appendage temperature is a subjective assessment made by palpating the extremities. It can also be assessed by simultaneous measurement of core and toe web temperature. During vasoconstriction the extremities are not as well perfused as vital organs because of shunting of blood away from the periphery. Consequently, appendages cool toward ambient temperature. The normal core appendage temperature gradient is 2 to 4° C. Values in excess of this range suggest poor perfusion, most commonly caused by vasoconstriction or hypovolemia.

Metabolic acidosis has several causes, one of which is poor perfusion. This occurs when lactic acid is generated in poorly oxygenated tissues because of inadequate perfusion and anaerobic metabolism. Acidosis is best assessed by means of a venous blood gas and may be identified by an elevated blood lactate concentration not caused by lactated fluid administration, a decrease in measured bicarbonate or total  $\text{CO}_2$  concentration, or a greater than normal base deficit. Normal blood lactate concentrations range between 0.5 and 2 mM/L. A point-of-care lactate monitor is available for veterinary patients (Accutrend, Roche Diagnostics), and lactate measurement should be considered for all hypotensive patients to help guide resuscitative measures. Rather than overinterpret any single value, normalization of elevated lactate after resuscitation can be used to indicate improved volume status and perfusion. Persistently elevated lactate values after initial fluid resuscitation should prompt a search for possible ischemic tissue (e.g., devitalized bowel) or a need for additional fluid therapy. Clinicians should not be overly surprised when finding elevated lactate in a compromised and hypotensive patient, but failure to normalize lactate should trigger a re-evaluation of the fluid prescription and possibly an evaluation for other causes of poor perfusion, such as cardiac disease or sepsis.

Central venous partial pressure of oxygen ( $\text{CvO}_2$ ) is a measure of the relationship between oxygen delivery and oxygen consumption; alterations in either component will change  $\text{CvO}_2$ . When oxygen delivery is diminished for any reason, including poor perfusion, tissues will continue to extract oxygen to meet metabolic demand. Continued extraction in the face of diminished oxygen delivery results in a greater percentage of oxygen extraction from the blood, resulting in a reduction of  $\text{CvO}_2$ . Increased tissue demand for oxygen, as in a hypermetabolic state, will also decrease  $\text{CvO}_2$  if oxygen delivery does not increase.  $\text{CvO}_2$  can be assessed with point-of-care analyzers (such as the iSTAT, Abbott

Laboratories; IRMA TruPoint, ITC Medical; or Stat Profile, Nova Biomedical) capable of performing blood gas analysis.

Jugular venous blood should be used to assess  $\text{CvO}_2$  because peripheral venous values are highly variable and are considered unpredictable. Normal central venous  $\text{CvO}_2$  values range between 40 and 50 mm Hg. Values between 30 and 40 are common in critically ill patients and have no known adverse consequences. Values between 20 and 30 mm Hg are progressively more worrisome, and values below 20 mm Hg are considered to indicate life-threatening tissue hypoxia.

If the cause of poor oxygen delivery is determined to be anemia, a hemoglobin infusion may be indicated. If the cause is determined to be hypoxemia, further evaluation of pulmonary function is indicated. If the cause is determined to be hypovolemia, further blood volume augmentation may be warranted. If the cause is determined to be heart failure or vasoconstriction, the underlying cause should be investigated and treated appropriately. Central venous  $\text{PO}_2$  values above normal may represent hyperperfusion of the tissues but are more often taken to represent poor oxygen uptake by the tissues secondary to impaired oxygen metabolism, such as occurs in sepsis. No adjustments in fluid therapy are likely to be necessary.

## The Hemodynamically Stable Patient

### ***Development of a Fluid Therapy Plan***

The patient's history, physical examination, laboratory results, and diagnosis are used to develop a fluid therapy plan by completing the following steps:

1. Determine the patient's fluid therapy needs, considering the component of these needs that will be met through the provision of the patient's nutritional requirements. This may occur if the patient is eating and drinking to meet some of its own needs or when parenteral or enteral nutritional support is provided.
2. Identify the ideal crystalloid(s) and/or colloid fluid to administer.
3. Determine the volume and rate of fluid administration for each fluid being administered.
4. Select the appropriate catheter type, size, and insertion site.
5. Develop a monitoring protocol, and make adjustments to the fluid therapy plan according to the patient's response to treatment.

## **HYDRATION STATUS VERSUS HYPOVOLEMIA**

It is important to remember that hydration status is the total body water of the cat. It is reflected in the ECF volume (i.e., the crystalloid volume status of the ECF) and is determined by the ECF  $\text{Na}^+$  content because of the

equality of ICF and ECF osmolality. The vascular fluid compartment is an integral component of the ECF compartment, but the association between the volume of the ECF (and thus total body water) and hypovolemia can vary. Blood volume status and hydration status must be evaluated independently because changes in intravascular volume status can occur independently of hydration. Patients can be hypovolemic without being dehydrated. Patients on therapy can be made hypervolemic without correcting a total body water deficit. Patients with edema may be hypervolemic as a result of fluid overload or hypovolemic as a result of vasculitis or hypoproteinemia.

### CREATING A FLUID PLAN

The fluid plan is generally divided into three categories, evaluated for each patient: deficits, maintenance, and ongoing losses. Within each category the clinician should determine the quantity and type of fluid to administer and also determine the fluid volume and rate for each type of fluid being administered. A worksheet such as that shown in [Table 5-3](#) can be a useful tool for constructing a fluid plan. Although it is often preferable to use a single fluid because of cost considerations, it is not always appropriate to do this for complex cases or seriously ill cats.

### CORRECTION OF EXTRACELLULAR FLUID VOLUME DEPLETION

The first element of the fluid plan should address preexisting dehydration. To determine the degree of total body water depletion (dehydration), the veterinarian should assess skin turgor, mucous membrane moistness, recent changes in body weight, urine output, and blood solute concentrations.

Most dehydrated patients have undergone isotonic fluid loss and thus have too low an ECF  $\text{Na}^+$  content. The magnitude of the deficit is first assessed by changes in skin turgor and moistness of the mucous membranes. The skin along the back or shoulders is lifted into a fold and released. In ECF volume-replete patients, the skin will snap back rapidly to its resting position. If it is detectably slow, the animal is estimated to be about 5% dehydrated. If the skin stands in a fold, the animal has life-threatening ECF volume depletion of about 12% of body weight. Intermediate skin turgor between these two levels of detection is interpolated between 5% and 12% dehydration. These are just rough estimates because of individual variation. Poor skin turgor in emaciated cats may overestimate dehydration, whereas obesity may cause it to be underestimated. Mucous membrane moistness can be assessed to add further support for the initial estimation. Body weight change, especially in the short term, is an excellent way to estimate the percentage of dehydration, provided recent and highly accurate body weights have been recorded. When patients have

a history consistent with the development of dehydration, such as vomiting, diarrhea, or polyuria coupled with poor intake, a 4% of body weight ECF volume depletion should be assumed even when skin turgor appears normal.

The volume deficit is calculated by multiplying the percentage of dehydration by the patient's body weight (in kg). For example, a 5-kg cat that is 5% dehydrated requires replacement of a fluid deficit of 250 mL, because 1 g equals 1 mL. This volume is then divided by the number of hours over which the deficit is to be restored to calculate the mL/hour fluid rate. Often, 24 hours is chosen for restoration of the deficit. However, the presence of azotemia believed to be prerenal or acute renal in origin requires a more rapid restoration of the deficit, such as within 4 to 6 hours. For a less critically ill patient or one with suspected or known heart disease, the clinician may elect to correct the deficit over as long as 36 hours or more. Again, it is important to recognize that these are merely guidelines, and the clinician should be prepared to periodically re-evaluate or change the rate of fluid administration in response to changes in the patient's clinical status.

When selecting the appropriate crystalloid, the clinician should always evaluate the history and clinical signs to understand how the deficit developed. This allows the clinician to make an educated guess as to the composition of the fluid that has been lost. When a serum chemistry panel with electrolytes becomes available, this can be used to confirm the best fluid composition for replacement. For example, fluids without calcium or potassium are indicated for patients with hypercalcemia or hyperkalemia. Fluids with a buffer will be indicated for patients with low serum bicarbonate. [Table 5-7](#) illustrates the types of fluids recommended to replace specific types of losses.

The ideal way to determine the amount of KCl to add to the fluids would be to measure the serum  $[\text{K}^+]$  and then use the sliding scale in [Table 5-5](#). However, if the patient's current serum  $[\text{K}^+]$  is not known, it is generally safe to use the empirical dosage of 20 mEq/L. When KCl is being added to parenteral fluids, it is important to mix the bag to distribute the KCl throughout the fluid.

### CORRECTION OF INTRACELLULAR FLUID VOLUME DEPLETION

Although uncommon, some patients may have hypertonic dehydration. This develops when solute-free water is lost in excess of isotonic fluid loss. It is more commonly observed in dogs because they suffer from heat stroke more commonly than do cats because of the very high respiratory evaporative heat loss required for thermoregulation. However, cats can suffer heat stroke as well, losing water through respiratory and salivary losses. Cats trapped in basements and closets may experience hypertonic dehydration because they have been

**TABLE 5-7** Assessment of Typical Fluid and Electrolyte Losses for Various Clinical Syndromes

Abnormality	Type of Losses	Type of Dehydration	Electrolyte Balance	Acid-Base Status	Fluid Therapy
<b>Simple dehydration</b> (water unavailable, stress, exercise fever)	Free water	Hypertonic Largely intracellular	Normal	Normal	Free water D5W
<b>Heatstroke</b>	Very hypotonic	Hypertonic	K <sup>+</sup> variable Na <sup>+</sup> variable	M Acidosis	0.45% NaCl, followed by balanced electrolyte solution
<b>Anorexia (still drinking)</b>	Hypertonic	Hypotonic	Na <sup>+</sup> lost K <sup>+</sup> lost	M Acidosis	Balanced electrolyte solution w/KCl supplement
<b>Anorexia (unable to drink)</b>	Hypotonic	Hypertonic Largely intracellular	Na <sup>+</sup> lost K <sup>+</sup> lost	M Acidosis	0.45% NaCl w/KCl
<b>Vomiting (gastric contents)</b>	Hypotonic or isotonic	Isotonic or mildly hypertonic	Na <sup>+</sup> lost K <sup>+</sup> lost H <sup>+</sup> lost Cl <sup>-</sup> lost	M Alkalosis	0.9% NaCl w/KCl
<b>Vomiting (duodenal contents)</b>	Hypertonic or isotonic	Isotonic or mildly hypertonic	Na <sup>+</sup> lost Mg <sup>++</sup> lost K <sup>+</sup> lost HCO <sub>3</sub> <sup>-</sup> lost	M Acidosis	Balanced electrolyte solution w/KCl
<b>Diarrhea</b>	Hypotonic or isotonic	Isotonic or hypertonic	HCO <sub>3</sub> <sup>-</sup> lost K <sup>+</sup> lost Mg <sup>++</sup> lost Na <sup>+</sup> lost	M Acidosis	Balanced electrolyte solution w/KCl
<b>Diabetes mellitus</b>	Hypotonic	Hypertonic extracellular & intracellular	Na <sup>+</sup> lost PO <sub>4</sub> <sup>--</sup> lost K <sup>+</sup> lost Mg <sup>++</sup> lost	M Acidosis HAG w/ DKA	Balanced electrolyte solutions w/KCl & Mg <sup>++</sup> upon Rx, some need PO <sub>4</sub> <sup>--</sup>
<b>Hypoadrenocorticism</b>	Hypertonic	Hypotonic	Na <sup>+</sup> lost	M Acidosis	0.9% NaCl initially; if severe, balanced electrolyte solutions
<b>Diabetes insipidus</b>	Free water	Hypertonic largely intracellular	K <sup>+</sup> lost Mg <sup>++</sup> lost	M Acidosis	0.45% NaCl
<b>Hypercalcemia</b>	Hypotonic	Isotonic or hypertonic	Na <sup>+</sup> lost	M Acidosis	0.9% NaCl w/KCl 0.45% NaCl if hypertonic
<b>Chronic renal failure</b>	Isotonic	Isotonic	Variable, depends on GFR H <sup>+</sup> retained	M Acidosis	Balanced electrolyte solutions usually w/KCl
<b>Acute renal failure</b>	Variable, depends on urine output	Variable, depends on urine output	K <sup>+</sup> , H <sup>+</sup> , PO <sub>4</sub> <sup>--</sup> retained Na <sup>+</sup> , Mg <sup>++</sup> variable	M Acidosis	Variable
<b>Urethral obstruction</b>	Isotonic or hypotonic	Isotonic or hypertonic	K <sup>+</sup> , H <sup>+</sup> , PO <sub>4</sub> <sup>--</sup> retained Na <sup>+</sup> , Cl <sup>-</sup> variable	M Acidosis	0.9% NaCl initially Followed by balanced electrolyte solution w/KCl
<b>Congestive heart failure (untreated)</b>	Isotonic gains	Isotonic Overhydration	Na <sup>+</sup> retention	R Alkalosis M Acidosis	D5W for KVO
<b>Congestive heart failure (treated w/furosemide)</b>	Dosage dependent	Variable	K <sup>+</sup> lost Na <sup>+</sup> variable	Variable, possible M alkalosis	Variable
<b>Septic shock/SIRS</b>	Isotonic	Isotonic	Na <sup>+</sup> lost	M Acidosis	0.9% or hypertonic NaCl, balanced electrolyte solutions, colloids
<b>Hemorrhagic shock</b>	Isotonic	Isotonic	Na <sup>+</sup> lost	M Acidosis	0.9% or hypertonic NaCl, balanced electrolyte solutions, colloids, blood

*M*, Metabolic; *R*, respiratory; *HAG*, high anion gap; *DKA*, diabetic ketoacidosis; *Rx*, prescription; *GFR*, glomerular filtration rate; *SIRS*, systemic inflammatory response syndrome.

Modified from Muir WW, DiBartola SP: Fluid therapy. In Kirk RW, editor: *Current veterinary therapy VIII*, Philadelphia, 1983, Saunders, p 31.

deprived of a source of oral water to replace losses. Hypertonic dehydration is detected by the presence of hypernatremia. The volume of a solute-free water deficit can be estimated from the plasma  $[Na^+]$  concentration as follows:

$$\text{Free water deficit (liters)} = 0.6 \times \text{body weight (kg)} \\ \times [( \text{plasma } [Na^+] / 148 ) - 1]^8$$

This type of volume deficit is given a separate line in the worksheet in **Table 5-3** because the type of fluid required to replace the deficit is solute-free water. A water deficit can be replaced enterally or parenterally and is discussed in more detail in the section on fluid therapy for hypernatremia.

### MAINTENANCE

Normal ongoing sensible and insensible losses must be balanced with maintenance fluid intake, provided either through fluid therapy or nutritional support. The cat's maintenance needs are calculated using the formula  $30 \times \text{body weight (in kg)} + 70$ . For example, for a 5-kg cat, this equals 220 mL. This is then divided by 24 hours to calculate a rate of 9 mL/hour. Maintenance fluid needs are replaced using a maintenance fluid type. Patients that are eating and drinking or are receiving enteral or parenteral nutrition support will have their maintenance fluid needs already fully or partially met.

### ONGOING LOSSES

The fluid prescription must also offset any abnormal ongoing losses. The goal is to pick the best crystalloid that will most likely replace the electrolytes lost in the pathologic condition being treated; this is often the same fluid type being used for replacement. Typical losses associated with various clinical conditions are detailed in **Table 5-7**. Gastrointestinal and urinary losses can be measured. However, to prevent underestimating the losses, estimates are often used, based on client information (e.g., the volume and frequency of vomiting) for the first few hours of fluid therapy before such measurements are made.

Some patients also have abnormal insensible losses such as those due to increased respiratory loss or fever. These types of losses are insensible and replaced with solute-free water. Because they cannot be measured, they must be estimated. Allotting an additional one quarter to one third of maintenance rate, depending on severity, is a reasonable starting estimate.

### ACID-BASE

Acid-base status is important in fluid selection because many crystalloid fluids contain buffers. The three most prevalent buffers found in commercial crystalloid solutions are lactate, acetate, and gluconate; they are bicarbonate ( $HCO_3^-$ ) precursors.

The cat's acid-base status may be assessed from a serum total carbon dioxide concentration ( $[TCO_2]$ ), or a venous blood gas.  $[TCO_2]$  provides an estimate of  $[HCO_3^-]$ . Metabolic acidosis occurs when the  $[TCO_2]$  is lower than normal, and metabolic alkalosis occurs when the  $[TCO_2]$  is higher than normal. It must be remembered that any metabolic acid-base derangement may be primary or compensatory. For example, a metabolic alkalosis may exist as a primary disorder owing to vomiting of gastric contents, or it may exist as a compensatory response to respiratory acidosis; thus  $[TCO_2]$  must always be assessed in light of the cat's history and clinical signs. For patients with severe or complex acid-base disorders, a blood gas is necessary for complete assessment.

Buffers are used for prevention and treatment of metabolic acidosis because they replace  $HCO_3^-$  deficit. However, buffered solutions are considered contraindicated in patients with metabolic alkalosis.

Cats rarely need to be treated for metabolic acidosis with  $HCO_3^-$  itself because the kidney is able to correct the acid-base imbalance after fluid replacement and treatment of the primary disease. However, when acidosis is severe ( $pH < 7.1$ ), the mEq of sodium bicarbonate to administer is calculated by multiplying the body weight (in kg) by the base excess obtained from a blood gas sample and multiplying this by a value of 0.3, representing the ECF space to which the  $NaHCO_3$  will redistribute. Only metabolic acidosis, and not respiratory acidosis, should be treated with  $NaHCO_3$ . Respiratory acidosis is treated by improving the cat's ventilation.

Typically, only one third to one half of the aforementioned calculated replacement dose of  $NaHCO_3$  is given intravenously, over 15 to 30 minutes, once the intravascular volume has been restored. After administration and time for equilibration, another blood gas may be sampled to re-evaluate the patient's acid-base status. The goal is not to completely normalize the acidosis but to increase the pH to a value of 7.2.

Although this is not ideal, when blood gas measurement is not available, the  $[TCO_2]$  may be used to infer the severity of metabolic acidosis;  $[TCO_2]$  below 8 mEq/L after rehydration suggests a need for  $NaHCO_3$  therapy.

### DEXTROSE

Dextrose is added to fluids as a method to provide solute-free water isotonically (dextrose moving intracellularly in the presence of insulin) and *not* as an energy source. 5% Dextrose contains 170 kcal/L of solution and will not provide meaningful calorie support. Each mL will contain only 0.17 kcal. Using the formula of  $30 \times \text{body weight (kg)} + 70$ , the resting energy requirement for a 5-kg cat is 220 kcal. The amount of 5% dextrose needed to meet the basal requirements of a 5-kg cat would be 1294 mL, about 6 times the daily maintenance fluid requirement.

### A CASE EXAMPLE

A case example, Table 5-8, illustrates how to develop a fluid plan for a 5-kg cat that is presented with a history of vomiting of 7 days' duration. The vomiting is thought to be due to a flare-up of previously diagnosed inflammatory bowel disease and is not thought to be gastric in origin. The cat is estimated to be 7% dehydrated. Thus the fluid deficit volume would be estimated to be 0.35 kg, which is equivalent to 350 mL. This is likely to be an isotonic loss in which  $\text{Na}^+$ ,  $\text{K}^+$ ,  $\text{Mg}^{++}$ , and  $\text{HCO}_3^-$  are lost. This is entered into the table as either 350 mL total or 14.5 mL/hour. Because the dehydration developed slowly over the past week, the plan is to restore it over 24 hours.

Until the measured electrolyte values are available from the laboratory, the clinician will assume a fluid concentration of 20 mEq/L  $\text{K}^+$  and use a fluid that also contains  $\text{Mg}^{++}$  at approximately 3 mEq/L. Thus a reasonable choice would be Normosol with 15 mEq/L additional KCl added.

The next step is to determine the volume needed for normal ongoing losses for the cat and the most appropriate fluid to use for this purpose. This volume is meant to replace the normal ongoing sensible and insensible fluid losses, *not* including deficits or abnormal ongoing losses from persistent vomiting.

Using the formula  $(30 \times \text{body weight in kg}) + 70$  to estimate the total volume of fluids in mL per 24 hours required by the patient, the clinician determines that a 5-kg cat requires 220 mL/day, or 9 mL/hour. For maintenance a low  $\text{Na}^+$  fluid such as Plasma-Lyte 56 + 5% dextrose is appropriate. It contains a buffer,  $\text{Na}^+$  and  $\text{Mg}^{++}$ , and solute-free water for maintenance. The patient will be transitioned to partial parenteral nutrition to provide for its nutritional needs after approximately the first 24 hours of hospitalization, which will then provide its maintenance fluid needs.

Next, the clinician selects the type and rate type of fluid to administer to replace the abnormal ongoing loss. In talking with the client, the clinician estimates that the cat vomited about 40 mL in the previous 24 hours. The clinician will adjust the fluid plan if the measured volume differs from this estimate while the cat is observed over the next 12 hours. The cat does not have excess urinary or respiratory loss, diarrhea, fever, or known third spacing. Thus at present the only abnormal ongoing loss is vomiting. In anticipation of a similar amount of vomiting over the next 24 hours, an additional 40 mL (approximately 1.5 mL/hour) is added to the fluid plan. This will be provided as an isotonic fluid, such as the Normosol with supplemental potassium, as indicated previously for replacement therapy.

With duodenal vomiting, metabolic acidosis can be expected. However, because the cat will likely be able to correct this itself when fully rehydrated, the clinician will usually wait for the serum chemistry result before considering any specific therapy for an acid-base disorder.

Lastly, to calculate the drops per minute, the clinician takes the desired fluid rate (in mL/hour), multiplies it by the drops/mL designated for the particular fluid administration set, and then divides that number by 60 min/hour.

The completed worksheet represents the fluid prescription for the cat. Next, the prescription should be translated into an administration plan. The fluid types can be administered concurrently using a T-port.

### Patient-Monitoring Recommendations

Repeated measurements of body weight are the best way to assess improving hydration status. Because changes in lean body and fat mass do not occur rapidly, short-term changes in body weight reflect changes in body fluid. Body weight measurements should be

**TABLE 5-8** Case Example Using the Calculation Worksheet for Fluid Therapy

Components of the Fluid Plan		Type of Fluid	Volume of Fluid	
			mL/day	mL/h
1. Deficits	Isotonic	Balanced crystalloid containing 20 mEq/L $\text{K}^+$ and $\text{Mg}^{++}$	350	14.5
	Hypertonic			
2. Maintenance	Normal ongoing losses	Low- $\text{Na}^+$ maintenance fluid	220	9
	(Enteral contribution from feeding)	Will be added on day 2	(0)	(0)
	Net normal loss to be provided by fluids		220	9
3. Abnormal ongoing losses	Gastrointestinal	Balanced crystalloid containing 20 mEq/L $\text{K}^+$ and $\text{Mg}^{++}$	Est 40	1.5
	Urinary		0	0
	Other sensible		0	0
	Insensible		0	0
Totals		1. Normosol + KCL 2. Plasma-Lyte 56 + 5% Dextrose 3. n/a	1. 390 2. 220 3. n/a	1. 16 2. 9 3. n/a

performed every 4 to 12 hours depending on the severity of the cat's condition and rate of fluid being given. It is important to use the same scale each time, and a human pediatric scale is likely to be the most accurate.

Skin turgor can be used serially to assess improvement in hydration status. The skin tenting tendency can also be used to determine the presence of subcutaneous edema. When the skin of a normally hydrated patient is released, it returns rapidly to its resting position. With severe subcutaneous edema, digital pressure to the skin surface will actually cause a transient pit or dimple in the skin surface when the finger is removed. Edematous patients are already overloaded with crystalloids, and further crystalloid therapy is not warranted.

Urine output can also be monitored to evaluate a patient's hydration status. Normal urine output varies between 0.5 and 2 mL/kg/hr. Oliguria (urine output <0.5 mL/kg per hour) associated with dehydration should respond rapidly to rehydration with crystalloid fluids.

Laboratory parameters, such as PCV, total protein, and serum electrolytes, must be monitored serially, often more than once daily depending on the severity of any detected derangements, to allow for necessary adjustments to the fluid therapy plan.

## INTRAVENOUS FLUIDS DURING ANESTHESIA AND SURGERY

Fluid therapy is important during all anesthetic procedures. Cats given appropriate fluid therapy have better outcomes and show fewer adverse side effects. Recovery times and quality are improved after anesthesia. Human patients receiving fluids also report less nausea, dizziness, and thirst. Although some of these parameters are difficult to assess in cats, it is likely that similar benefits occur. Appropriate use of fluids helps maintain intravascular volume and acid-base balance and electrolyte normality, supports organ function, and provides cells with nutrients and oxygen. Maintenance of appropriate liver blood flow also facilitates metabolism of anesthetic drugs, which in turn will result in more rapid patient recovery. In addition to the cardiovascular benefits of fluid therapy during anesthesia, some patients need fluids to correct specific metabolic abnormalities in glucose and acid-base balance. Fluid therapy for surgery and anesthesia will be discussed in greater detail in Chapter 7.

## SPECIFIC DISEASE CONDITIONS

### Hypernatremia

Hypernatremic patients have a free water deficit, or water loss in excess of sodium. When hypernatremia is

present, calculating the dehydration deficit alone, as is done for patients whose sodium is still within normal range, often underestimates the fluid volume needed because the loss is from the ICF compartment. Therefore the free water deficit, or water lost in excess of solute, must be calculated.

The volume of the deficit can be estimated as follows:

$$\begin{aligned} \text{Free water deficit (liters)} &= 0.6 \times \text{body weight (kg)} \\ &\quad \times [(\text{plasma } [\text{Na}^+]/148) - 1]^8 \end{aligned}$$

In cases of chronic hyperosmolality, the rate of replacement should be proportional to the duration of the hyperosmolality. Ideally, serum sodium should not be raised or lowered by more than 0.5 to 1 mEq/hour. For example, to decrease the serum  $[\text{Na}^+]$  from 168 mEq/L to 150 mEq/L, a difference of 18 mEq, divide 18 by 0.5 mEq/hour, which results in 36 hours. Using the example of a 5-kg cat, because the deficit is 405 mL, divide 405 by 36 hours to get a free water administration rate of 11 mL/hour. In patients that are eating on their own or have a feeding tube, this fluid can be given orally as water. When it must be given intravenously, it can be given as D5W.

For patients with hypernatremia, the ECF volume deficit, maintenance requirement, and any abnormal ongoing losses are also calculated as discussed earlier and included in the fluid therapy prescription. The rate at which the serum  $[\text{Na}^+]$  is decreasing should be monitored every 6 to 8 hours.

Optimal therapy for patients with acute salt toxicity ( $\text{Na}^+$  overdose leading to hypervolemic hypernatremia) is unknown, and a more rapid correction may be advantageous. A correction rate of no faster than 1 mEq/hour is generally employed to prevent complications.<sup>1</sup> Furosemide may be given to increase renal  $\text{Na}^+$  excretion. However, acute hypernatremia is unlikely to be encountered in felines because they are not inclined to ingest de-icers or water-softening compounds.

### Azotemia and Renal Disease

#### ***Localization of Azotemia***

Patients with azotemia should always be assessed to determine whether their azotemia is prerenal, renal, postrenal, or some combination thereof. Those patients without preexisting renal disease must have a severe ECF volume depletion to manifest an azotemia that is detectable as outside the normal reference range. By contrast, patients with preexisting functional renal disease (even when nonazotemic in the volume-replete state) may develop azotemia with only mild ECF volume depletion. Both situations warrant rapid restoration of the ECF volume, although total volume required may be different; thus the degree of ECF volume depletion, not

the degree of azotemia, must be used to guide the amount of fluids necessary.

Urethral and ureteral obstructions are the most common causes of postrenal azotemia, although fluid therapy decisions are similar for ruptured urinary tract. The primary difference between urethral and ureteral obstruction is that the former is often acute and complete, whereas the latter is often partial or unilateral and some urine production remains. It should always be remembered that the cat's ability to void urine does not preclude azotemia being postrenal. Postrenal azotemia patients often have accompanying fluid deficits, varying from mild to severe enough to result in hypovolemia. Such deficits should be restored rapidly, even in patients with complete urethral obstruction. Leaving such patients in a state of volume depletion does not protect them from making further urine and does not prevent bladder rupture or kidney damage. Volume should be restored while the urethral obstruction is being corrected or the bladder emptied to remove the pressure preventing renal filtration. Cats that have existing or recently corrected postrenal azotemia need to have their urine output monitored because ongoing urinary loss can be high and unpredictable.

Primary renal azotemia should be suspected when prerenal and postrenal causes of azotemia have been eliminated. The cat with renal azotemia should then be assessed to determine whether the kidney disease is chronic or acute; this assessment is based largely on history. Patients with chronic renal failure typically have polyuria, although exceptions exist with advanced disease. Cats with acute renal failure may be polyuric or oliguric depending on the cause and severity of their disease. Assessment of the hourly rate of urine production is essential in cats with acute renal failure for determination of their fluid therapy plans. Sometimes it is difficult to determine if a patient has acute or chronic renal failure at initial presentation. When this is in doubt, the veterinarian should assume that it is acute and begin measuring urine output promptly. When managing a cat with azotemia, the veterinarian must always consider prerenal, postrenal, and acute renal azotemia, because each of these has the potential to be reversible.

### **Electrolyte Disorders**

Cats with azotemia may have hypokalemia or hyperkalemia. Hypokalemia is most often seen with mild to moderate chronic renal failure. Hypokalemia can cause renal failure in cats, which is termed *hypokalemic nephropathy*.

Hyperkalemia is common with acute renal failure and postrenal azotemia. There may be a temptation to employ K<sup>+</sup>-free solutions, such as 0.9% saline, initially. However, 0.9% saline may contribute to metabolic acidosis, which is also likely to be severe in these patients.

Unfortunately, no studies exist comparing 0.9% saline with buffered balanced electrolyte solutions on the outcome of cats with life-threatening hyperkalemia caused by postrenal azotemia. It is postulated that the dilution provided by a balanced electrolyte solution, even one containing small amounts of K<sup>+</sup>, will lower the cat's serum [K<sup>+</sup>]. Once the obstruction is eliminated and renal filtration is restored, K<sup>+</sup> will be excreted. However, this may not occur rapidly enough in cats that have severe symptomatic hyperkalemia. Such patients will require insulin–dextrose therapy to shift K<sup>+</sup> intracellularly and possibly calcium gluconate if cardiac arrhythmias are present.

Critically ill cats with urethral obstruction and other forms of postrenal azotemia may have severe metabolic acidosis, and the blood pH may be below 7.1. This degree of acidosis can lead to ventricular arrhythmias and poor tissue perfusion. Treatment for metabolic acidosis in such cats requires prompt correction of the problem leading to the postrenal azotemia, fluid diuresis, and bicarbonate therapy, which has been discussed previously.

When the acidosis is not severe, resolution of obstruction and diuresis will allow the return of renal filtration and the kidney should correct the problem without supplemental HCO<sub>3</sub><sup>-</sup>.

Hyperphosphatemia is common whenever the GFR is reduced and is seen with prerenal, renal, and postrenal azotemia. Severe hyperphosphatemia can lead to hypocalcemia, acidosis, and tissue deposition of calcium phosphate salts, potentially causing dysfunction of the kidney, heart, and other organs. In cats that are still eating, phosphate binders can be added to the food. In those that are not yet eating, only improvement in the GFR will rectify the situation. Intractable hyperphosphatemia is one factor that may necessitate dialysis in patients with severely compromised renal function. When hyperkalemia is present with hyperphosphatemia, the associated severe ionized hypocalcemia will accentuate the effect of hyperkalemia on membrane excitability and increase the risk for the development of arrhythmias. Nonetheless, treatment of ionized hypocalcemia with calcium gluconate is reserved for patients that have clinical signs of ionized hypocalcemia, because the additional calcium in the presence of hyperphosphatemia will potentiate tissue mineralization.

### **Diuresis After Resolution of Obstruction and in the Healing Phase of Acute Renal Failure**

There are two clinical situations associated with azotemia in which urine output may be exceptionally high. Postobstructive diuresis is the most common and can be profound—greater than 120 mL/hour in some cats. All postobstructed cats must have their urine output and body weight measured frequently to allow adjustments

in their fluid prescriptions so that the volume in always balances the volume out. Relying on shortcuts for fluid therapy calculations (e.g., twice maintenance, three times maintenance) can markedly underestimate the fluid needs in these patients. Cats that have been obstructed have also suffered a renal insult. During the healing phase, a second insult (e.g., the development of ECF volume depletion and hypoperfusion) will worsen the prognosis for full renal recovery. Hypokalemia may develop during the postobstructive diuresis; therefore serum  $[K^+]$  should also be monitored. Some cats may require  $K^+$  supplementation as soon as a few hours after initial stabilization despite initially presenting with hyperkalemia.

As healing occurs, there is a danger that matching fluid intake with urine output will create a situation in which fluids being administered are driving the increased urine production. To prevent this, once the cat is stable and eating well, fluid rates are adjusted to just below output by approximately 5 mL per hour. If the body weight falls or the urine output does not drop accordingly, the decrease in fluid rate may have been premature. Weaning a cat with postobstructive diuresis off fluids requires careful monitoring and attention to detail.

The other situation in which azotemia may be accompanied by a considerable diuresis is in the healing phase of acute renal failure. Such patients may make large quantities of "bad-quality urine." This state may persist for as long as 1 week, and the considerations for weaning the patient off intravenous fluid therapy discussed for postobstructive diuresis apply. Some of these patients may be left with chronic renal failure as a result of the initial acute insult. Thus it is expected that they will remain polyuric as they are weaned off fluids. But as long as the patient is able to drink and is given the opportunity to do so, increased water intake should balance increased urine output. Fluid therapy should be weaned slowly and carefully so that the clinician can observe if in fact the patient is able to keep up with its increased urinary losses. If it cannot and fluids are weaned too quickly, then recovery from acute renal disease will be compromised.

### **Proteinuric Renal Disease**

Although comparatively rare in cats, patients with primary proteinuric renal disease will generally have increased total body  $Na^+$  content. They may not need fluid therapy, providing additional  $Na^+$ , despite the presence of azotemia. The veterinarian should always assess the volume status of the cat rather than assume that the azotemic patient requires fluid therapy.

### **Oliguric Renal Disease**

Patients with oliguric or anuric renal failure have little or no urine production, respectively. They require careful

monitoring for ECF volume overload. Signs of ECF volume overload were discussed earlier. Cats with oliguric renal failure require intensive nursing care and should have their urine output and body weight monitored very frequently, initially as often as every 1 to 2 hours. Normal urine output for a hydrated animal is 1 to 2 mL/kg/hour. Once interstitial and intravascular fluid deficits have been corrected, patients with oliguria must have their "ins and outs" monitored. That is, the clinician can measure the amount of fluid taken in, orally or by intravenous fluids, and compare it with the amount of fluid lost as urine, vomitus, or diarrhea, worsening third space fluid accumulation, and insensible losses (20 to 30 mL/kg per day). The clinician should adjust the fluid rate to match the fluid coming out in addition to the calculated insensible losses. Because all fluid need calculations are inherently educated guesses, a critical component of the monitoring plan will be to weigh the patient frequently using a highly accurate scale. This will help prevent overhydration and underhydration, both equal enemies of a cat in renal failure.

### **Chronic Renal Disease**

Cats with mild to moderate stable chronic kidney disease are sometimes given prescriptions for subcutaneous fluids as diuresis, described as "flushing out the kidney." There is little justification for this practice. Most cats with mild and moderate stable disease eat and drink enough to offset their own losses, even in the presence of polyuria. Subcutaneous fluid therapy does not result in any sort of dialysis to lessen azotemia. The values for blood urea nitrogen and creatinine may fall slightly, but this is due to the increased  $Na^+$  load increasing the GFR, essentially making the kidney work harder. Subcutaneous fluid therapy should be reserved for situations when the cat cannot keep up with losses, such as an episode of vomiting or diarrhea, and thus fluids are indicated to stave off ECF volume depletion. Cats with more severe chronic kidney disease may have trouble keeping up with losses through oral intake and may benefit from continuous subcutaneous fluid therapy. However, such cats may also not eat enough to meet their calories and may be better served with a feeding tube to allow provision of both fluids and calories.

### **Congestive Heart Failure**

Congestive heart failure (CHF) is a volume overload state, in which the ECF  $Na^+$  content is high, as is the intravascular fluid volume, with increased end-diastolic pressures leading to imbalance of Starling's forces across the capillary endothelium and resulting in edema. Depending upon the underlying pathology, the volume overload and edema can be largely localized to either the systemic or pulmonary circulations or may affect

both. These patients have RAAS activation and high circulating levels of aldosterone, promoting renal Na<sup>+</sup> retention. Consequently, most patients with heart failure do not benefit from the further addition of Na<sup>+</sup> in the form of intravenous fluids. In fact, the clinical aim more often is to remove the excess Na<sup>+</sup> by use of diuretics that increase renal salt loss while simultaneously improving cardiac performance. Patients with pleural or abdominal effusion may need thoracocentesis or abdominocentesis to more rapidly and effectively remove excess Na<sup>+</sup> and fluid.

As a general rule, it is best not to administer significant amounts of intravenous fluids or Na<sup>+</sup>. In most cases of acute left-sided CHF and in cats with significant pleural effusion, it is imperative to focus on treating the CHF and lowering end-diastolic pressures, often with diuretics; it is less important to correct measurable electrolyte or acid-base abnormalities. These can be addressed once the patient is stable.

Stable patients with compensated CHF may require fluid therapy to address acid-base or electrolyte disorders or ongoing excess losses. Some patients with CHF have poor tissue perfusion and metabolic acidosis and may have hypokalemia or hypomagnesemia. Further, they may need intravenous medications and possibly continuous intravenous isotonic dextrose infusions to keep their catheters patent. Additionally, they may need calories; parenteral nutrition may be required if they are unable or unwilling to eat.

If crystalloids are given, they should be low in Na<sup>+</sup>, buffered, and potentially supplemented with K<sup>+</sup> or Mg<sup>++</sup>. Often, improvement of cardiac performance will correct the metabolic acidosis, and thus supplemental sodium bicarbonate administration is rarely needed.

Although patients with CHF typically have volume overload and do not require rehydration therapy, they may have abnormal ongoing losses, such as vomiting or diarrhea, which must be replaced.

Cats with CHF that are receiving diuretics should be monitored: Parameters include urine output, improvement in respiratory character, and ability to rest and sleep. This minimizes the risk of overtreatment, dehydration, and marked decreases in GFR. Mild increases in blood urea nitrogen and creatinine are expected, but induction of renal failure through extreme prerenal lowering of GFR must be avoided. The veterinarian may be tempted to preemptively treat patients in cardiac failure with a conservative dose of intravenous fluids as a form of renal protection. However, this is not medically sound: Diuretics are given to promote renal Na<sup>+</sup> excretion; adding Na<sup>+</sup> in the form of intravenous fluids just counteracts the effect. Having a patient on a diuretic, such as furosemide, concurrently with intravenous fluids is typically counterproductive; it is not possible to simultaneously dehydrate and rehydrate a patient.

One situation in which Na<sup>+</sup>-containing fluids may be needed is when the patient has been grossly dehydrated by overzealous diuretic use and needs to be brought back to euolemia. However, even in these cases, it is often best to allow for slow rehydration by temporarily discontinuing or decreasing the dose of diuretic given while carefully monitoring respiratory rate and other clinical indications of developing pulmonary edema. In some cases oral rehydration (simply by allowing the patient to drink) will suffice.

### **Concurrent Cardiac and Renal Disease**

Cats that have both heart disease and kidney disease pose a unique challenge. It is difficult to prescribe a therapy that will support one organ without harming the other, particularly when organ failure is present for either disease. Expanding the total body fluid will promote better renal perfusion and an improved GFR, but the addition of fluid also increases the risk of CHF. Reducing total body fluid, such as by giving diuretics, alleviates Na<sup>+</sup> and fluid retention in patients with heart failure, but this will lower the GFR and worsen renal failure. When both conditions are severe, the veterinarian aims to address and improve both conditions simultaneously: to improve cardiac function and perform the functions of the kidney through continual renal replacement therapy. However, when disease is relatively mild or advanced therapies such as continuous renal replacement therapy are unavailable, one condition is generally determined to be more severe from a clinical standpoint, on the basis of the physical examination and laboratory data. Identifying the more severe condition is often a straightforward, deliberative process. It is an essential first step for choosing the optimal treatment.

For example, if a cat with cardiomyopathy and chronic kidney disease presents with dyspnea and pulmonary edema, the most immediate problem is CHF; that patient will need a diuretic, regardless of the severity of its azotemia. In contrast, the patient with cardiomyopathy and kidney disease who presents with inappetence, lethargy, vomiting, and dehydration has a dominant problem of renal failure. This patient likely needs fluids or a reduction in the dose of diuretic given. Another consideration is removing ancillary medications such as angiotensin-converting enzyme (ACE) inhibitors. Often, cats will return to eating and drinking when pharmacotherapy is simplified; clinicians should resist the temptation to solve every problem with more medications.

For cats with cardiac disease, it should be pointed out that the urine specific gravity may be inadequately concentrated, even when azotemia is present, if the cat is taking diuretic medications or has hyperthyroidism or other causes of obligate polyuria. Thus azotemia together with isosthenuria (or minimally concentrated urine) is not synonymous with renal failure. Azotemia may, in these cases, be solely prerenal.

When a gallop sound appears for the first time during fluid therapy in a cat with heart disease, it may be a harbinger of fluid overload. Cats can have third heart sounds without being in CHF, so it is the newness of the gallop sound during fluid therapy that should alert the clinician to the impending clinical signs of fluid overload, including dyspnea. As a general rule, a cardiorenal patient that develops dyspnea during fluid therapy should be suspected of having progressed to CHF. Thoracic radiographs can be taken to confirm the condition and rule out other potential causes of dyspnea.

Replacement-type fluids such as LRS should not be given to cardiorenal patients once they are euvolemic because they result in administration of excess of  $\text{Na}^+$  and a deficit of  $\text{K}^+$ . Such patients will either be eating or should be given a maintenance fluid type, although this should be supplanted by the provision of sufficient calories in patients that are not eating.

For cats with chronic renal disease that are being treated with subcutaneous fluids at home, extra vigilance is required when there is concurrent heart disease. Owners of these cats must be advised to watch for the development of dyspnea, often manifested as "belly breathing," because the abdominal effort of many dyspneic cats is apparent to clients.

## Diabetic Ketoacidosis

Poorly regulated diabetic patients lose  $\text{Na}^+$ ,  $\text{Cl}^-$ , and  $\text{K}^+$  in their urine as a result of osmotic diuresis. The presence of ketosis will worsen these losses, and the cat may become unable to compensate through intake, particularly if appetite decreases with the onset of acidosis and dehydration. Electrolyte and fluid losses may be further compounded by vomiting, which can occur in patients with DKA. The components of therapy of DKA, in descending order of importance, are as follows:

1. Correction of volume and hydration deficits
2. Correction and monitoring of electrolyte and phosphorus abnormalities
3. Resolution of ketosis with insulin and dextrose therapy

Although insulin therapy is third on this list, this does not imply that it is not important. It simply serves as a reminder that it is the fluid and electrolyte derangements that are life threatening in these patients. Correction of acid-base abnormalities is also a component of therapy for DKA, but this is usually accomplished through fluid and insulin therapy.

### Correction of Volume and Hydration Deficits

0.9% NaCl is generally the initial fluid of choice for the ketoacidotic diabetic patient with severe ECF volume depletion, insofar as this provides for the rapid

re-expansion of the ECF volume. However, 0.9% NaCl is an acidifying solution and theoretically may contribute further to the acidosis in these patients. Once insulin therapy is initiated, a non-acidifying, buffered fluid such as Normosol-R is preferred. This fluid contains 140 mEq/L of sodium, gluconate, and acetate as buffers and small amounts of potassium and magnesium, both of which are generally depleted in patients with DKA. Fluids that employ lactate as their buffer may, at least in theory, not be the optimal choice for DKA. Metabolism of lactate to bicarbonate occurs in the liver, and cats with DKA may have significant hepatic lipidosis. Despite the acidifying potential of 0.9% NaCl and the concerns about lactate metabolism in cats with DKA, in the clinical situation any isotonic replacement fluid is suitable for initial therapy in these patients, as long as volume status, hydration, electrolytes, and acid-base parameters are closely monitored.

### Correction and Monitoring of Electrolyte and Phosphorus Abnormalities

#### POTASSIUM

Although total body  $\text{K}^+$  depletion exists, serum  $[\text{K}^+]$  may initially be normal or even increased as a result of insulin deficiency and metabolic acidosis in the patient with DKA. Once insulin therapy is initiated, serum  $\text{K}^+$  will move intracellularly and the serum  $[\text{K}^+]$  will often drop precipitously. Thus cats with DKA are expected to need  $\text{K}^+$  supplementation early in their treatment to stave off hypokalemia. Serum  $[\text{K}^+]$  should be monitored frequently during management of DKA; ideally, at least every 4 hours. Table 5-5 can be used to determine the amount of potassium to be added to the fluids for these patients. However, clinicians should anticipate that hypokalemia in these patients can be severe and poorly responsive to "normal" levels of potassium supplementation. In some cases, administration of potassium by CRI, using the KMax value (0.5 mEq/kg per hour) is necessary. However, this should never be undertaken without the ability to frequently monitor serum  $[\text{K}^+]$  values.

#### PHOSPHORUS

Serum phosphorus should be monitored every 8 to 12 hours during initial management of the patient with DKA. Serum phosphorus levels may initially be low or normal or may be increased if the patient's GFR is reduced. However, it is common for phosphorus levels to decrease with the initiation of fluid and insulin therapy. This is due to movement of phosphorus into the cells in the presence of insulin, dilution by intravenous fluid therapy, and improvement in GFR. If phosphorus levels fall below approximately 1.5 mg/dL, the patient is at risk for hemolytic anemia. Hypophosphatemia may also be clinically undetectable or may contribute to

muscle weakness and ataxia. When phosphorus levels approach the low end of the reference range, supplementation is necessary, with ongoing monitoring to allow for dosage adjustments as necessary. Supplementation is provided by the addition of potassium or sodium phosphate to the intravenous fluids. Typical dose rates for phosphorus range from 0.01 to 0.03 mmol/kg per hour. Dose rates as high as 0.12 mmol/kg per hour may be necessary in some patients being treated for DKA. Potassium phosphate is more commonly used, and the amount of potassium provided in this way should be taken into account when calculating potassium supplementation rates in these patients. Some clinicians prefer to provide the patient's calculated potassium needs as a mixture of 50% KCl and 50% potassium phosphate, in anticipation of the development of hypophosphatemia. However, this is a shortcut that may not provide the correct amount of phosphorus in every patient.

## MAGNESIUM

Hypomagnesemia is common in cats with DKA; however, its clinical significance has not been fully elucidated. Supplementation may be considered if serum total [Mg] approaches 1 mg/dL, and supplementation is also indicated when hypokalemia appears to be resistant to potassium supplementation. An initial dose of 0.5 to 1 mEq/kg every 24 hours is used in these situations. This dose may be given on the first day and then followed with half this dose on subsequent days. Magnesium may be provided as the chloride or sulfate. These salts are not compatible with solutions containing calcium or bicarbonate.

### ***Resolution of Ketosis with Insulin and Dextrose Therapy***

In the patient with DKA, the first goal is to restore ECF volume and improve renal perfusion and urine production. These alone will decrease blood glucose values. Insulin therapy in these patients is often not started until ECF volume deficits are being addressed. Insulin may be administered by CRI or intermittent subcutaneous or intramuscular techniques. The details of insulin therapy in DKA are beyond the scope of this chapter; however, the addition of dextrose to the intravenous fluids is an important part of fluid therapy in these patients. In general, the goal is to decrease serum glucose values by approximately 50 to 100 mg/dL per hour. The insulin dose is adjusted to achieve this goal. In addition, glucose is added to the intravenous fluids as the glucose levels fall. This prevents hypoglycemia and provides a substrate for the insulin, thus allowing the body to resume glucose metabolism and resolve the ketosis. Once ketoacidosis has resolved and the patient is eating, food intake will provide an energy source and the addition of dextrose to the fluids can be discontinued.

## ***Acidosis***

Aside from the electrolyte and glucose abnormalities in a patient with ketoacidosis, there is a considerable metabolic acidosis from the circulating ketoacids. In most cases, the acidosis of DKA is rapidly self-correcting. Rapid re-expansion of the ECF volume improves perfusion. Ketones, once their production is halted, will be lost in the urine or converted to bicarbonate. Thus specific therapy for acidosis generally is not required if fluid and insulin therapies are instituted appropriately. When a fluid containing a bicarbonate precursor, such as acetate, is being used, additional bicarbonate will not likely be necessary.

When the venous pH level is below 7.1, the administration of sodium bicarbonate may be considered to promote normal enzymatic functions. In these cases the previously outlined calculation may be used. Again, most clinicians will administer one third to one half of the amount needed over 20 minutes and then reassess the patient's acid-base status. It should be noted that the use of sodium bicarbonate in these patients is the subject of disagreement between clinicians. Potential adverse effects of sodium bicarbonate administration include exacerbation of hypokalemia, increased affinity of hemoglobin for oxygen, paradoxical central nervous system acidosis, delayed development of alkalosis, and sodium overload. Because of these risks, and particularly because of the lack of documented evidence of a clear benefit, many specialists do not advocate the use of sodium bicarbonate therapy in patients with DKA. One conservative approach would be to consider the use of sodium bicarbonate for patients with persistent metabolic acidosis despite fluid therapy for 12 to 24 hours if the pH is 7.1 or below and the  $[HCO_3^-]$  is 12 mEq/L or below.

## ***Monitoring***

Frequent monitoring is crucial for patients with ketoacidosis. Ideally, patients with diabetic ketoacidosis should have their blood glucose monitored every 2 hours and acid-base and electrolyte status evaluated every 4 to 6 hours. Serum phosphorus concentration and urine ketones should be measured twice daily. Serum ketone measurement may also be available in some hospitals. This level of close monitoring is particularly important in the early phases of rehydration and insulin supplementation. If the veterinary clinic is not able to provide this level of care, the feline patient with DKA should be referred to a specialty hospital whenever possible.

## ***Hyperglycemic Hyperosmolar Syndrome***

Hyperglycemic hyperosmolar syndrome (HHS) is an uncommon complication of diabetes mellitus in cats. It is less common than DKA and is often associated with a poorer prognosis. The pathogenesis of HHS is similar to

DKA, but low levels of insulin are present in patients with HHS; thus lipolysis and ketosis do not occur. Hyperglycemia is the predominant abnormality in these patients, with blood glucose levels in excess of 600 mg/dL. Hyperglycemia results in osmotic diuresis, and the patient incurs a significant water deficit. This worsens the hyperglycemia, and eventually the dehydration leads to a reduction in GFR. This is exacerbated by fluid losses caused by vomiting and decreased fluid intake in the sick patient. The decreased GFR then further exacerbates the hyperglycemia.

Patients with HHS are, by definition, hyperosmolar. Osmolality can be measured or calculated. Measurement is typically performed by the freezing point depression method, but this is not readily available to all clinicians. Osmolality is often calculated using the following equation:

$$2[\text{Na}^+](\text{mEq/L}) + [\text{glucose}](\text{mg/dL})/18 \\ + [\text{BUN}](\text{mg/dL})/2.8$$

Serum osmolality in patients with HHS is typically greater than 330 mOsm/kg, largely because of the contribution of the severe hyperglycemia. Normal osmolality in cats is around 300 mOsm/kg.

Cats with HHS may be hypernatremic, normonatremic, or hyponatremic. It is important to remember that these values reflect water balance, and not total body Na levels. The latter are likely to be low in patients with HHS. In the presence of hyperglycemia, water moves into the vasculature along an osmotic gradient, and this will reduce the serum  $[\text{Na}^+]$ . This is called dilutional hyponatremia. To determine if the degree of hyponatremia is appropriate for the patient's level of hyperglycemia, a corrected serum  $[\text{Na}^+]$  can be calculated:

$$[\text{Na}^+]_{\text{Corrected}} = [\text{Na}^+]_{\text{Measured}} \\ + 1.6([\text{measured glucose} - \text{normal glucose}]/100)$$

The equation essentially states that for every 100 mg/dL increase in glucose, there should be a 1.6 mg/dL decrease in  $[\text{Na}^+]$ . Thus if the patient has a normal corrected  $[\text{Na}^+]$ , the measured serum  $[\text{Na}^+]$  should correct as hyperglycemia resolves. However, the patient with HHS may have significant ongoing free water loss, contributing to an increase in serum  $[\text{Na}^+]$ . Thus if a patient with HHS has a normal or elevated uncorrected serum  $[\text{Na}^+]$  in the face of hyperglycemia, the patient has a free water deficit.

Although patients with HHS are hyperosmolar, isotonic fluids are indicated in these cases; 0.9% NaCl is the fluid of choice. This will replenish total body Na, correct volume deficits, improve GFR, promote glycosuria, and reduce hyperglycemia. Potassium supplementation will also be required, depending on

serum levels. As for patients with DKA, it should be expected that aggressive potassium supplementation will be needed once the patient has received adequate fluid therapy and insulin therapy has been initiated.

Fluid therapy in the patient with HHS should be approached conservatively. Rapid correction of long-standing hyperosmolality can lead to fluid shifts that can cause cerebral edema. It is recommended that the fluid deficits be replaced over 24 to 48 hours in these patients, beginning with 0.9% NaCl, as previously indicated. If hypotonic fluids are used initially, the hyperosmolality is likely to be corrected too quickly, and neurologic abnormalities could result. If hypernatremia persists and is not improving after 6 to 12 hours of 0.9% NaCl administration, the patient likely has a significant free water deficit and lower Na fluids such as 0.45% NaCl may be indicated. However, serum  $[\text{Na}^+]$  should be monitored carefully to ensure that the hypernatremia does not resolve too quickly. A decrease in serum  $[\text{Na}^+]$  of approximately 0.5 to 1 mEq per hour is a reasonable goal. Different combinations of 0.9% and 0.45% NaCl may be required to achieve this.

Insulin therapy should be withheld in patients with HHS for at least 4 to 6 hours to allow improvements in hypovolemia and dehydration. During this time the serum glucose will likely fall significantly as a result of dilution and an increase in GFR. Hypokalemia should also be addressed before insulin therapy is initiated. Insulin protocols that are used for cats with DKA can also be used for management of HHS, but the dose of insulin should be reduced by 50%. In the management of HHS the goal should be to decrease serum glucose levels by no more than 50 mg/dL per hour. Again, this is to avoid rapid changes in osmolality with resulting detrimental fluid shifts. As for DKA, dextrose should be added to the fluids as serum glucose levels approach 250 mg/dL.

## Liver Disease

With severe liver disease it is difficult to predict the electrolyte and acid-base abnormalities in an individual patient. Some patients have elevated  $\text{Na}^+$ ,  $\text{Cl}^-$ , and  $\text{HCO}_3^-$  concentrations; others have just the opposite. One consistent finding is that these patients are usually hypokalemic. If in doubt about the electrolyte and acid-base status of a patient with severe liver disease, the clinician should choose a fluid low in  $\text{Na}^+$  and  $\text{Cl}^-$ . A fluid that does not contain lactate is recommended because the liver may not be able to convert the lactate to  $\text{HCO}_3^-$ . It is important not to confuse lactate with lactic acid. The lactate ion in LRS is a precursor of  $\text{HCO}_3^-$  and cannot be converted to lactic acid; 0.45% sodium chloride with 2.5% dextrose fits these

requirements. Because cats with liver disease are generally hypokalemic, additional KCl usually must be added to the fluids. This is particularly important in liver disease because hypokalemia exacerbates the development of hepatic encephalopathy. Because patients with liver disease may also have an obligate polyuria, the clinician should be prepared to adjust the fluid prescription to account for additional urine output. This is a particular concern in patients that do not drink because of debility or hepatic encephalopathy associated with their primary disease.

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