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Intravenous Fluid Therapy in Calves

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Oral and intravenous fluid therapy is aimed at restoring circulating blood volume as well as correcting specific acid-base, electrolyte, and energy deficits. Solutions for parenteral use should be selected on their ability to correct specific volume and electrolyte deficits and, if possible, ability to correct acid-base and blood glucose abnormalities. In general, solutions intended for replacement of losses should have a composition similar to that of the fluid or electrolyte lost by the animal.^{45,47} For example, solutions that contain electrolytes in preparations similar to extracellular fluid (ECF) are best used to restore losses of ECF incurred through diarrhea.^{44,45,47} It would be inappropriate to correct diarrheic losses with isotonic dextrose since diarrhea-induced deficits in electrolytes are not addressed with isotonic dextrose therapy alone. Selection of the appropriate parenteral solution is dependent on a knowledge of the properties of commonly used parenteral solutions and an understanding of the pathophysiology of fluid and electrolyte abnormalities associated with common conditions in calves.

TYPES OF PARENTERAL SOLUTIONS

Solutions intended for parenteral use can be grouped into two broad categories: crystalloid fluids and colloid fluids. *Crystalloid fluids* are aqueous solutions of electrolytes and/or low molecular weight organic compounds such as dextrose, sodium lactate, sodium acetate, and sodium bicarbonate. Crystalloid solutions can be subdivided into replacement, maintenance, or special-use solutions, depending on their composition.

Replacement solutions have an electrolyte composition similar to that of extracellular fluid. Since sodium concentration is the major

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determinant of extracellular fluid volume, replacement fluids have sodium concentrations at or near physiologic concentrations (Table 1). Concentrations of other major ions may vary widely but are generally similar to plasma concentrations.

Maintenance solutions are characterized by having relatively low sodium concentration, and many are relatively rich in potassium. Maintenance fluids are formulated predominantly to replace insensible losses, which are primarily water of low sodium concentration. In calves, insensible loss is 15-20 ml/kg/24 hours.^{35,58} Insensible losses are independent from other losses and are similar in diarrheic and normal calves. Specific maintenance solutions are not widely used in calves. In most cases, the same solution used for replacement is also used for maintenance.

Special-use fluids are prepared for use in correction of specific fluid, electrolyte, or energy abnormalities. Examples are sodium bicarbonate, dextrose, or hypertonic saline solutions. Isotonic (1.3%) or hypertonic solutions of sodium bicarbonate are used to correct metabolic acidosis as discussed below. Isotonic (5%) or hypertonic dextrose solutions are used to provide metabolizable energy but also provide a convenient source of free water in excess of sodium after metabolism of the dextrose. Dextrose solutions should not be considered adequate to meet complete nutritional needs, although they may be used to provide caloric requirements in calves.² Hypertonic saline solutions (5.2-(7.2%) administered in small volumes (4-5 ml/kg) have proven useful as adjuncts to replacement therapy in cases of cardiovascular collapse from hemorrhage or hyponatremic, hypoosmolal fluid loss.^{16,23} Infusion of hypertonic saline solutions results in improvement in cardiac performance by increasing myocardial contractility and decreasing vascular resistance in pulmonary, systemic, and coronary circulations.^{16,23,60}

Colloidal fluids are solutions containing high molecular weight, nondiffusible compounds. Examples are plasma and dextrans in crystalloid solution. Nondiffusible osmotically active colloidal compounds do not freely traverse the vascular endothelium, assuring that the colloid remains within the vascular compartment. The resultant colloid osmotic pressure or oncotic pressure retains water within the intravascular space. Therapeutic use of colloids reduces the volume of crystalloid necessary to restore losses of circulating blood volume. For general use in replacement therapy, colloidal solutions have not shown consistent advantages over crystalloid solutions.²³ In calves, use of plasma can provide a valuable source of immunoglobulin for passive immunity.

ESTIMATION OF VOLUME REPLACEMENT REQUIREMENTS

The quantity of fluid necessary to restore blood volume is determined by first estimating the degree of dehydration on the basis of clinical signs.^{4,7,63} Dehydration of less than 5-6% of body weight does not result in specific clinical signs. The animal will, however, have a history of decreased fluid intake or signs of increased losses such as

		Table 1.	Selected Pan	renteral Soluti	ons for Use in	Calves		
							HCO ₃	
NOILATOS	туре†	Na (mEq/L)	K (mEq/L)	Ca (mEq/L)	Mg (mEq/L)	Cl (mEq/L)	EQUIVALENT (mEq/L)	(mosm/L)
Plasma*		135-144	3.9-5.6	2.6-3.2	0.8-1.3	93-104	22-26	279-298
Ringer's solution	R	147	4	ŭ	I	156	I	310
Lactated Ringer's‡§	В	130	4	£	-	109	28 Lactate	274
McSherry's solution	Я	140	10	ũ	ю	103	50 Acetate	308
Plasmalyte A‡	R	140	ы	ļ	n	98	27 Acetate 23 Gluconate	294
Multisol-R§	R	140	ы	I	က	98	27 Acetate 23 Gluconate	294
0.9% NaCl	R	154	ł	Ι	I	154		308
5% NaCl	SU	856	Ι	I	ł	856	I	1712
Acidifying Solution	R,M	77	36	ł	1	113	I	289
5% Dextrose	SU	I	Ι	Ι	I	I		250
1.3% NaHCO ₃	SU	156	I	I	I	Ι	156	312
5% NaHCO ₃	SU	595	l	-		Ι	595	1190
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R = replacement solution; M = maintenance solution; SU = special use solution
Baxter Healthcare, Deerfield, IL
Ceva Laboratories, Overland Park, KS
10.45% NaCl, 0.275% KCl, 1.25% dextrose

INTRAVENOUS FLUID THERAPY IN CALVES

fecal staining of the perineal area. As loss approaches 6-8% of body weight, clinical signs become apparent. Eves become slightly sunken, as evidenced by appearance of a noticeable gap between the globe and bony orbit (enophthalmia). In addition, there is loss of skin turgor, reflected in persistence of a skin tent produced on the upper evelid or side of the neck for 3-5 seconds. At 8-10% loss in body weight, eyes become more substantially sunken, and the persistence of a skin tent extends up to 10 seconds. Mucous membranes are dry and tacky at this stage. Calves are often depressed and unwilling to suckle. Maximal clinical dehydration occurs with loss of 10-14% of body weight. Such animals have markedly reduced peripheral perfusion with cool extremities and poor peripheral pulse quality. They are severely depressed and may be comatose. Eyes are deeply sunken in the orbit and a skin tent usually persists indefinitely. Clinical evaluation of dehydration should include a rapid but thorough physical examination to identify other abnormalities that will influence choice of therapy or prognosis. Particularly important are signs of injury, respiratory disease, septicemia, or localized sepsis such as omphalitis, arthritis, and uveitis.

If dehydration has occurred rapidly, loss from the ECF space may be more severe than indicated by clinical signs. Neonatal animals may rapidly lose large quantities of extracellular fluid and electrolytes into the gastrointestinal tract. These losses occur from the entire extracellular space but are most severe from the intravascular compartment.⁵⁵

Loss of body weight can be an unreliable indicator of severity of dehydration. Rarely is weight recorded prior to fluid loss to serve for comparison. Even in cases in which pre-loss weight is known, extracellular fluid loss may be underestimated if a substantial quantity of fluid is sequestered in a body space such as the gastrointestinal tract or, less commonly, the peritoneal or pleural cavity. Sequestered fluid contributes to total body weight while not contributing to extracellular or intracellular fluid volumes.

Packed cell volume (PCV) and total serum protein concentration vary widely among normal, hydrated calves,^{61,62} which effectively limits their usefulness for assessment of degree of dehydration. Packed cell volume may be used as a rough guide to assign calves to appropriate fluid treatment groups or as an aid in defining prognosis. Calves with PCV greater than 40% may be best treated with intravenous fluids, whereas calves with lower PCV values may be managed without intravenous fluid administration.⁷⁶ Marked elevations in PCV are associated with a poor to guarded prognosis for recovery.^{15,76} Serial PCV determinations are useful in monitoring responses to fluid administration (see below).

Once the degree of clinical dehydration has been estimated, the quantity of fluids necessary to correct the deficit can be calculated by multiplying the estimated dehydration by body weight. An error is created when dehydrated body weight rather than an estimated predehydration weight is used, but this error is not large enough to be significant in calves. In the absence of complicating circumstances such as unexplained depression or the presence of sepsis, calves do not require intravenous fluid therapy until dehydration is 8% or more.^{47,55,63} Calves that are less than 8% dehydrated can usually be treated most practically with oral fluid administration. In experimental and naturally occurring diarrhea, the value of intravenous fluids for treatment of dehydrated calves has been demonstrated.^{19,20}

Once the volume required to correct the deficit has been determined, a parenteral solution appropriate for the particular case must be chosen. Selection of fluid for replacement is guided by knowledge of the type of fluid and electrolyte loss that has occurred. In most cases, the appropriate fluid for initial therapy contains electrolytes at concentrations similar to that of ECF (see Table 1). Ringer's solution and similar products approximate ECF in their composition of major ions. As a first choice, these solutions are appropriate for correction of volume deficits.

Although lactated Ringer's solution is used to replace volume and electrolyte deficits, it will not usually correct the metabolic acidosis in calves with diarrhea. Lactated Ringer's solution contains lactate as an alkalizing agent. Dehydrated diarrheic calves have a reduced ability to metabolize L-lactate.⁸ Even healthy calves utilize D-lactate poorly. As a result, the racemic mixture of D- and L-lactate present in lactated Ringer's solution is expected to be poorly metabolized in dehydrated calves. Neonatal calves with diarrhea have metabolic acidosis characterized by elevated blood levels of L-lactate consistent with decreased utilization of endogenously generated lactate.⁴⁹ Because of this decreased utilization as well as the low concentration of L-lactate in lactated Ringer's solution, it may be used to replace water and electrolyte losses in diarrheic calves, but it has limited value in correction of metabolic acidosis.³⁰ However, if L-lactate is provided in greater concentration (50 mmol/L), it can be used to effectively correct metabolic acidosis. Ringer's solution and acetated or lactated Ringer's solutions may be purchased from several sources or can be prepared in concentrated form, using readily available chemicals.^{11,32}

Other parenteral fluids with compositions similar to that of ECF are available commercially or can be produced (see Table 1). A balanced electrolyte solution, McSherry's solution,⁴³ provides sodium, chloride, calcium, and magnesium at concentrations similar to that of plasma. Potassium is present at higher concentrations than in plasma, and acetate is present as alkalizing agent. McSherry's solution does not have sufficient alkalizing agent to restore normal acid-base balance in most diarrheic calves. This solution is routinely used for replacement therapy in calves admitted to the Veterinary Teaching Hospital, Ontario Veterinary College.

Acceptable solutions for parenteral replacement therapy can be produced using physiologic saline (0.9% NaCl) as a base solution.^{55,63} Potassium and dextrose should be added to improve value as a replacement solution. Potassium may be added at 5-20 mEq/L, with dextrose

included to give a concentration of 5% in the final solution (i.e., 50 gm/L). This solution has no alkalizing potential, but sodium bicarbonate may be added at 5 g/L⁶³ or administered separately as an isotonic (1.3%) or hypertonic solution.

CORRECTION OF SPECIFIC ELECTROLYTE DEFICITS

In addition to blood volume restoration, intravenous fluid therapy should address specific abnormalities associated with the primary cause of dehydration. This requires knowledge of the pathogenesis of the primary condition responsible for the dehydration. In the evaluation of specific deficits, consideration should be given to the major electrolytes, sodium, potassium, and chloride, as well as to acid-base and energy balance.

The most common cause of severe dehydration in calves is enteritis due to bacterial, viral, and/or protozoal agents.^{33,55,73} In addition to dehydration, calves with diarrhea experience substantial alterations in sodium, potassium, and chloride balance from electrolyte loss into the gastrointestinal tract.^{35,36,58} Sodium is lost predominantly from the extracellular space, although sodium loss also occurs from the intracellular space.³⁶ Since sodium is the major determinant of the size of the ECF space, its loss is accompanied by reduction in ECF volume.⁴⁴ When sodium and water are lost in similar proportions, the concentration of sodium in plasma may be within normal range in the face of substantial whole-body sodium depletion. If blood volume is restored with solutions of low-sodium concentration or if the dehydrated calf consumes large volumes of water, plasma sodium concentration may be markedly reduced as a result of the disproportionate restoration of the ECF volume by sodium-free fluid.44-47 Fluid mixtures with sodium concentrations similar to those in plasma are indicated to replace both volume and sodium deficits. In cases of severe sodium depletion, isotonic or hypertonic saline will provide additional sodium, although saline does not correct deficits in other electrolytes and may exacerbate a preexisting acidosis.^{23,47}

Calves may experience dehydration with hypernatremia in situations of reduced water intake or when water is lost without simultaneous sodium loss, as occurs with uncorrected insensible losses. Severely dehydrated diarrheic calves may also exhibit some degree of hypernatremia.⁷³ Calves may suffer hypernatremia secondary to consumption of improperly diluted oral electrolyte solutions.⁵⁹ In cases of hypernatremic dehydration from decreased water intake or excess free water loss, sodium chloride (0.3%)/dextrose (3.3%) may be slowly infused as a replacement fluid. Free water will be provided by an initially isotonic solution following metabolism of the dextrose. In the presence of extreme hypernatremia, treatment is aimed at slowly correcting dehydration, while simultaneously reducing plasma sodium concentration. Overly aggressive rehydration will result in development of neurological signs secondary to cerebral swelling.⁵⁹ Extreme hypernatremia from consumption of concentrated sodium-containing solutions carries a guarded to poor prognosis, even with treatment.

Chloride is the major extracellular anion. In diarrhea, changes in chloride parallel changes in sodium. Similar to those of sodium, plasma chloride concentrations are relatively unchanged, although substantial quantities of chloride are lost in diarrheic feces.^{35,36,73} Correction of chloride deficits is usually achieved with correction of sodium deficits.

Hypochloremia is observed in adult cattle following obstruction of duodenal outflow by abomasal displacement or volvulus.^{1,67} This hypochloremia is accompanied by hypokalemia and metabolic alkalosis. In severe cases that progress to cardiovascular collapse, metabolic acidosis develops and is superimposed over the preexisting alkalosis. As in adults, calves with experimentally induced gastric outflow obstructions develop hypochloremia with hypokalemia and metabolic alkalosis.^{12,21} Unfortunately, there is insufficient published information to allow prediction of the electrolyte and acid-base status of a calf with naturally occurring abomasal outflow obstruction. In one case of naturally occurring abomasal dilation in a calf, there was no alteration in plasma electrolyte values, and mixed respiratory and metabolic acidosis was observed.¹⁷ Similar to dogs with gastric dilation-volvulus, calves with abomasal volvulus may be expected to experience a range of electrolyte and acid-base abnormalities that cannot be predicted and can only be assessed by measurement of electrolyte and acid-base values.^{48,78} In situations in which abomasal outflow is impaired, balanced electrolyte solutions present the optimal first choice for volume replacement, unless specific electrolyte abnormalities can be identified and treated. When hypochloremia is present, isotonic saline (0.9%) or half-strength saline (0.45%) with KCl (0.275%) and dextrose (1.25%) may be administered. This latter fluid contains potassium at 36 mEq/L and, therefore, should not be rapidly infused.

Diarrheic calves lose large quantities of potassium in feces.^{35,36,55} Simultaneously, potassium moves from the intracellular to the extracellular compartments, resulting in plasma potassium concentration at or above normal values.^{36,55} The mechanism of intracellular to extracellular potassium movement is complex, but significant changes in intracellular potassium are assumed to occur secondary to the rise in extracellular hydrogen ion concentration from the metabolic acidosis that occurs in diarrheic calves. As the extracellular hydrogen ion concentration rises, intracellular hydrogen concentration rises, and intracellular potassium concentration falls. This shift of intracellular potassium leads to hyperkalemia despite whole-body potassium depletion from diarrheic loss.

Severe hyperkalemia will alter the resting membrane potential.⁵ Alterations in membrane potential may be expressed clinically as skeletal muscle weakness or in electrocardiographic (EKG) abnormalities. Hyperkalemia-associated cardiovascular changes include development of relative bradycardia, with heart rate decreasing by 8 ± 2 beats per minute with each 1 mmol/L increase in plasma potassium.³⁶ Bradycardia is often not sufficiently severe to be a useful clinical sign in an individual calf. As plasma potassium increases, the amplitude of the P wave on EKG decreases and may be absent at potassium concentrations greater than 6.5 mmol/L. In addition, the T wave increases in amplitude and spikes, while the QRS complex broadens.

In severely dehydrated diarrheic calves, the presence of hyperkalemia should be assumed when selecting fluids for volume replacement. Treatment with fluids containing high potassium concentration should be avoided until steps have been taken to reduce plasma potassium levels. Correction of acidosis and infusion of glucose promotes uptake of potassium by cells and reduces plasma potassium.⁴⁵ Slow infusion of one liter of a potassium-rich alkalizing solution (K,23 mmol/ L; sodium bicarbonate, 82 mEq/L; glucose, 68 g/L) resulted in a reduction in plasma potassium concentration and an increase in intracellular potassium concentration.^{56,57} This serves to demonstrate the safety of administering high potassium solutions if acidosis is corrected simultaneously. Commercially available parenteral solutions for replacement contain potassium at much lower concentrations and will not significantly exacerbate hyperkalemia. Although these fluids are unlikely to exacerbate hyperkalemia, they cannot replace the whole-body potassium deficit in diarrheic calves.^{47,55} If supplemental potassium has been added to a solution at more than 5-8 mEq/L, the solution should not be rapidly infused until acidosis has been corrected.

Reversal of hyperkalemia can occur quickly, but replacement of whole-body deficit is usually completed more slowly, since active transport of potassium into cells occurs relatively slowly.^{45,47} In adult animals, it is recommended that potassium infusion not exceed 0.5 mEq/kg/hr.⁴⁵ At a replacement volume administration rate of 30 ml/kg/hr, parenteral solutions can contain K at approximately 17 mEq/L and not exceed the recommended potassium replacement rate. To achieve this potassium concentration in most commercial parenteral fluid solutions, supplemental KCl should be added. Dextrose should also be added to facilitate cellular uptake of potassium. If plasma potassium concentration is known, severely hypokalemic animals can be treated with solutions with potassium concentration greater than 20 mEq/L.²² As a general rule, however, whole-body potassium deficits may best be corrected by oral potassium supplementation alone or combined with the intravenous route.^{47,55}

Acid-Base Balance

Calves with diarrhea develop metabolic acidosis that increases in severity with duration of diarrhea.^{36,43,46,49,56,73} Origin of the acidosis is multifactorial. It is partially related to gastrointestinal loss of bicarbonate^{35,73} and to decreased renal excretion of hydrogen ion⁷³; in addition, calves under 8 days of age have acidosis associated with accumulation of organic acids in circulation, as indicated by significantly increased blood lactate values.⁴⁹ Older calves may have a similar degree of acidosis but have normal or near-normal blood lactate values.⁴⁹ Some diarrheic calves may suffer metabolic acidosis in the absence of significant clinical signs of dehydration.^{29,31,53} The mechanism of acidosis in these calves is unclear, but it is accompanied by hyperchloremia, elevated blood urea nitrogen, and increased anion gap. The increased anion gap suggests accumulation of unidentified anions, although plasma lactate and ketoacids were within normal limits.³¹

Correction of acidosis is beneficial in decreasing the severity of hyperkalemia. In addition, acidosis contributes significantly to depression in diarrheic calves, and the restoration of normal acid-base status often dramatically improves clinical signs.^{30,31}

Although the presence of metabolic acidosis is well recognized, it is difficult to estimate the magnitude of acidosis in any individual animal. Guidelines have been proposed to estimate deficit on the basis of clinical dehydration.^{4,63} Unfortunately the association between severity of acidosis and degree of dehydration has not proven to be consistent.^{18,31,49} This is particularly true in calves that have received oral fluids to fully or partially correct fluid and electrolyte losses without appropriate correction of acidosis.⁵⁰ To overcome the difficulty in estimating acid-base status, determination of total CO₂ in serum⁵⁰ and whole venous blood¹⁸ has been advocated as feasible in practice or field situations.

Balanced electrolyte parenteral solutions contain alkalizing agents (see Table 1), but these agents are usually present at concentrations insufficient to correct the degree of acidosis observed in many diarrheic calves.^{49,50} Racemic mixtures of sodium lactate present in lactated Ringer's solution are only partially metabolized in acidotic dehydrated calves,^{8,51,56} further limiting their alkalizing ability. Sodium L-lactate and sodium acetate added to polyionic solutions at 50 mmol/L during volume restoration have significant alkalizing ability.³⁰ Acetate in balanced polyionic solutions (see Table 1) also contributes alkalizing potential. Solutions containing acetate often contain gluconate, but the gluconate makes no contribution to the alkalizing ability of these solutions because it is poorly metabolized in calves.⁵¹ Propionate has alkalizing ability similar to that of L-lactate and acetate in healthy calves⁵¹ but is not incorporated into polyionic solutions. In parenteral solutions, sodium L-lactate and sodium acetate have comparable alkalizing ability, although the alkalizing effect of sodium bicarbonate is more rapid and of greater magnitude than that of either lactate or acetate.³⁰

Isotonic (1.3%) or hypertonic (3-5%) aqueous solutions of sodium bicarbonate are indicated for correction of metabolic acidosis. Bicarbonate solutions may be purchased premixed or prepared by dissolving sodium bicarbonate in sterile distilled water immediately prior to use. Solutions of sodium bicarbonate are not stable during autoclaving. Each gram of sodium bicarbonate provides 12 mEq of sodium and 12 mEq of bicarbonate.

The alkalizing ability of balanced electrolyte solutions used for rehydration can be increased by the addition of sodium bicarbonate.^{30,63} When adding sodium bicarbonate powder to parenteral solutions, the powder should first be completely dissolved in a small volume of the solution, and this smaller volume is then added to the larger container. This ensures that the powder is completely dissolved prior to infusion of the fluid. Sodium bicarbonate should not be added to Ringer's solution or to similar solutions containing calcium, as calcium carbonate precipitate will form. Precipitate formation may not be grossly visible initially, but even microscopic precipitate of calcium carbonate may pose a risk from intravenous infusion of particulate matter. In addition, precipitate formation reduces the alkalizing potential of the solution.³²

Disorders of Blood Glucose

Diarrheic calves are often normoglycemic but experience a negative energy balance as a result of decreased dietary intake and decreased intestinal absorptive ability combined with increased metabolic demands associated with fever, sepsis, and other intercurrent abnormalities.^{2,55} Dextrose (5-7%) in the polyionic replacement solutions cannot meet the maintenance nutrition requirements of diarrheic calves,^{2,55} but dextrose added to parenteral solutions will provide a source of energy and facilitate reversal of hypoglycemia. Dextrose will also aid in correction of hyperkalemia by promoting movement of potassium into cells.

Hypoglycemia is present in diarrheic calves, particularly severely dehydrated calves.^{38,43,72,73} Hypoglycemia and lactic acidosis develop in concert as a consequence of impaired hepatic gluconeogenesis believed to result from the action of endotoxin absorbed through the gastrointestinal tract.^{8,55,56} Hypoglycemia (<3 mmol/L or 54 mg/dl) indicates a poor prognosis in severely dehydrated calves, although calves with low blood glucose may recover with prompt therapy.²⁰

ADMINISTRATION RATES FOR REPLACEMENT AND MAINTENANCE

Once the necessary replacement volume has been determined and replacement solution selected, deficit correction should be initiated promptly. There is little experimental evidence in calves, but fluid administration rates of 30-40 ml/kg/hr are reported to be appropriate in dehydrated calves.^{37,63} Using these administration rates, a 45-kg calf that is 10% dehydrated will receive 4.5 L of replacement solution within 2.5-3.5 hours. At a rate of 30-40 ml/kg/hr, the parenteral fluid should not contain potassium at more than 13-17 mEq/L in order not to exceed a potassium administration of 0.5 mEq/kg/hr.^{22,45} Caution is also encouraged to prevent too-rapid administration of any parenteral solutions.^{45,47} Administration of fluid to dehydrated calves at a rate of 80 ml/Kg/hr resulted in elevation in central venous pressure consistent with volume overload, although there were no other clinical signs of volume overload.³⁰ Periods of such high-flow rate should be maintained for only brief periods of time.

As an alternative to a uniform administration rate for all cases, it has been recommended that rate vary with estimated severity of clinical dehydration.^{45,57} In one scheme, the goal is to replace one half of the calculated deficit within 6 hours, three quarters within 24 hours, and complete replacement within 48 hours. Using this system, an initial administration rate for a 45-kg calf that is 10% dehydrated would be

approximately 8.3 ml/kg/hr. Such a rate is often impractical and probably unnecessarily slow for most practice situations. In confirmed or suspected cases of oliguric renal failure, however, exuberant fluid administration should be avoided until some measure of response to therapy has been considered.^{28,60}

If rehydration over a prolonged period of time or long-term maintenance on intravenous fluids is undertaken, some consideration should be given in the fluid administration plan to replacement of insensible losses and continuing losses. Obligatory insensible water losses for diarrheic and normal calves are similar, at 15-20 ml/kg body weight (BW)/day. Normal urinary losses require inclusion of at least an additional 30-35 ml/kg BW/day. It is usually difficult to accurately estimate ongoing losses from diarrhea. In diarrheic calves, fecal water losses can approach 130 ml/kg BW/day, but average values are approximately 75 ml/kg BW/day.^{35,58} In summary, calves receiving long-term intravenous therapy require at least 50 ml/kg/day (5% of BW/day) to meet insensible and urinary losses plus additional fluid to replace continued loss.

It is often difficult to maintain consistent rates of fluid administration by gravity flow for active calves. This is a particular problem at low-drip rates necessary for delivery of small fluid volumes. Active calves substantially alter flow rates by alternating between periods of standing and recumbency. They also may periodically obstruct fluid flow by kinking the catheter and administration set tubing through their movements. It may also be difficult to maintain catheter patency under these conditions. As a result, active calves should be maintained on oral fluids or, if necessary, on intravenous fluids only under careful supervision.

MONITORING RESPONSE TO FLUID THERAPY

Although it is convenient to simply administer the estimated volume of replacement fluid, attention should be given to monitoring the clinical responses to therapy. The value of clinical signs for monitoring will depend on the severity of the initial dehydration. In severely dehydrated patients, return of a palpable peripheral pulse, reduction in capillary refill time, and improvement in mucous membrane color indicate improvement in cardiovascular performance. Skin turgor and enophthalmia will not return to normal for several hours, since these signs are dependent on replacement of interstitial fluid losses.^{28,30}

Observation of urination is a valuable monitoring sign. Although difficulty in placing a urinary catheter in calves limits the capability of monitoring the volume of urine produced, an attempt should be made to observe urination. Placing the patient on an absorbent surface such as a blanket or towel may facilitate observation of urination. Urinalysis with determination of specific gravity is valuable and easily accomplished.

Respiratory signs such as coughing or development of moist rales may develop with pulmonary edema owing to overhydration,⁶⁰ but the value of these signs in calves is poorly defined. Improvement in mental attitude has limited value as a monitor of rehydration, since degree of depression is also influenced by other factors such as the presence of acidosis.³⁰ If the patient continues to be depressed after substantial volume replacement, other causes of depression should be investigated.

Serial determination of packed cell volume (PCV) and plasma protein is a simple and useful method of assessing restoration of plasma volume.^{43,73} As extracellular volume is restored, the PCV initially falls, then maintains a constant level when volume replacement is complete.³¹

Serial determination of body weight may be used to monitor hydration and to assess longer-term rate of weight gain. During volume replacement, initial increases in weight may be due to pooling in the gastrointestinal tract or other third space rather than restoration of extracellular fluid.⁴⁹

In complicated cases, more invasive monitoring may be necessary. Available techniques include measurement of central venous pressure (CVP) and direct measurement of arterial blood pressure.²⁸

FLUID DELIVERY SYSTEMS

Administration Sets

Administration of intravenous fluids over a prolonged time requires a delivery system and secure venous access. The delivery system or administration set serves to transport parenteral fluid solutions from a container to an indwelling needle or catheter, while allowing regulation and monitoring of the rate of flow. The administration set should be long enough to provide elevation of the fluid container to approximately 1 meter to facilitate gravity flow. It must also be long enough to permit some freedom of movement to an active patient.

Commercially available administration sets offer the assurance of consistent quality manufacture and sterility, but they are usually too short for most applications unless a calf's movements are severely restricted. A coil of sterilized air hose (external diameter, 10-15 mm) may be inserted between the fluid container and a standard administra-



Figure 1. Fluid administration set, composed of a commercial administration set coupled to coiled air hose.



Figure 2. Fluid administration set, fabricated from latex tubing and several coils of rigid nylon tubing. Note the adjustable metal clamp and glass drip chamber, used to adjust and monitor flow rate.

tion set to extend the length of commercially available administration sets³² (Fig. 1). Redundant administration set may then be tethered, using several elastic bands chained together.^{63,77}

Serviceable fluid administration sets (Fig. 2) can also be readily fabricated in most practices from latex rubber tubing and rigid plastic tubing. These sets may carry a high initial cost, but they are easily resterilized by autoclaving for repeated use. Latex rubber components deteriorate with repeated autoclaving, although they may be resterilized up to eight to ten times. The basic components of the administration set are adaptors for connection to the fluid container and to the intravenous catheter or transfer set as well as a valve to regulate flow and a monitor or drip chamber to assess rate of administration. Such a device has been described⁷⁷ (see Fig. 2). Components are available from veterinary and scientific supply houses (CDVM Inc, St-Hyacinthe, Quebec, Canada).

In humans, it is recommended that administration sets be changed every 24-48 hours to reduce risk of catheter-associated sepsis.^{6,40} Most calves will not require intravenous fluid therapy beyond 24-48hours, but in cases of prolonged therapy, consideration should be given to regular replacement of administration sets.

Extension sets (see Fig. 6C) may be placed between intravenous catheters and administration sets. Extensions with injection ports reduce the necessity of manipulating the catheter during administration of antibiotics and other drugs or during replacement of administration sets.³

Catheters and Needles

Long-term administration of parenteral fluids requires a secure venous access through an intravenous cannula. Cannulas are available in two forms: metal needles and plastic catheters.⁶⁹

Metal needles, either plain or wing-tipped butterfly needles, are more easily placed in the vein than plastic catheters. They also are associated with significantly less cannula-associated thrombophlebitis, but, because they are unstable in the vein, their use is associated with high rates of perivascular infiltration.⁷⁵ As a result, needles can only be recommended for short-term intravenous use and, even then, only in the presence of constant supervision.

Plastic cannulas are available in two general forms: over-theneedle (ON) catheters and inside-the-needle (IN) catheters. The ON catheter is constructed as a flexible plastic cannula fitted tightly over a metal needle (Fig. 3). The needle tip extends beyond the cannula tip to protect the plastic tip during insertion. After placement, the metal needle is withdrawn, and the plastic cannula remains in the vein with the hub secured to the skin.

Over-the-needle catheters allow greater mobility of the patient than plain metal needles. The blunt plastic tip limits trauma to the vessel wall and lessens the risk of infiltration. Unlike a metal needle, plastic cannulas have limited internal strength and are prone to kinking. Kinking may occur anywhere along the length of the catheter with resultant interruption of fluid flow. Repeated kinking at the hub may result in fatigue failure, with potential for leakage or catheter embolism from complete separation at the hub.

The large size of many plastic catheters allows rapid fluid administration, but large size increases difficulty of insertion, particularly insertion through thick skin that has lost turgor from dehydration. The plastic tip of ON catheters may split and fray during insertion. A small skin incision made prior to insertion will reduce risk of splitting the tip and facilitate insertion by reducing skin resistance.

Catheter emboli may occur from shearing of plastic catheters by the needle during insertion. Care should be taken to avoid pulling the plastic cannula back over the needle once the cannula has been advanced into the vein, as this action can result in shearing. The plastic cannula should only be withdrawn in concert with the needle or only after the needle has been completely withdrawn.

The IN or through-the-needle catheter consists of a metal needle and long plastic catheter protected by an outer plastic sleeve (Fig. 4). The needle is first inserted into the vein and withdrawn after the plastic catheter has been advanced through it. In some products, the needle can be completely removed from the catheter after insertion; in others,



Figure 3. A,B, Examples of two over-the-needle cannulae, with metal insertion needles in place.



Figure 4. A,B, Examples of two through-the-needle cannulae. For clarity, outer plastic sleeves that protect the plastic catheter during insertion have been removed.

the needle is withdrawn then left in place over the plastic catheter but covered with a plastic shield.

Inside-the-needle catheters are useful for long-term placement and as central venous lines to facilitate assessment of cardiovascular pressures.⁶⁹ They are useful for administration of parenteral nutrition solutions.²

Similar to ON catheters, IN catheters are more difficult to place than plain metal needles and may be prone to kinking. Catheter embolism from shearing may also occur, with particular care required for catheters in which the insertion needle is shielded rather than removed after placement.

The insertion needle of the inside-the-needle catheter produces a puncture site larger than the indwelling plastic cannula. Bleeding around the cannula may result following withdrawal of the needle. This can be minimized by applying pressure to the puncture site as the needle is withdrawn. Residual blood should be removed to reduce the potential for bacterial growth.

An IN catheter can be fashioned from a 20- to 30-cm length of polyvinyl chloride (Becton Dickinson Co, Rutherford, NJ), polyethylene (Clay-Adams, Parsippany, NJ), or siliconized (Dow-Corning, Midland, MI) tubing. Sterile tubing is inserted through a standard 12- to 14-gauge needle that has been placed in the vein percutaneously.^{13,63,66,77} A Touhy-Borst adaptor or blunt needle (Becton Dickinson Co, Parsippany, NJ) fitted into the tubing can be used to allow connection to a standard administration set (Fig. 5).

Catheter Selection

Catheter size is designated by a gauge number on the basis of external diameter.⁶⁹ The larger the external diameter, the lower the gauge designation. There may be marked differences in the internal diameter of catheters with identical external diameter and gauge values.³³ Since flow through the catheter is proportional to the fourth power of the internal diameter, small differences in diameter can have a marked influence on flow rates.

Commonly available 18-gauge catheters will allow flow rates of 80



Figure 5. Catheter constructed from a length of sterile polyethylene tubing and a Touhy-Borst adaptor (Becton-Dickinson Co, Parsippany, NJ).

to 100 ml/min of water flowing with gravity from a water head of 1 meter. Flow rates in this range are adequate for rehydration therapy in calves but may be inadequate for larger animals.^{45,47} Increasing catheter size to 16 gauge will approximately double flow rates to 160-210 ml/min.

As risk of catheter-associated thrombophlebitis increases with increased catheter size, selection of catheters of unwarranted diameter should be avoided.^{13,34} Larger catheters may also be more difficult to place in dehydrated animals. As a result, catheters of 16- to 18-gauge are the most appropriate choices for use in the jugular veins of calves.

Catheter length should be chosen to provide secure venous access. Catheters that are too short may be dislodged from the vein by movement of the calf. Catheters that are excessively long may be difficult to insert into a severely dehydrated patient. For use in the jugular vein, catheters should be at least 6-8 cm in length to provide sufficient catheter length within the vein lumen. Length should not exceed 30 cm, as there is a risk of inducing ventricular fibrillation with longer catheters.³⁷

Polymers used in catheter construction have an influence on the frequency of catheter-related complications (see below). For shortterm use in calves, choice of catheter material among commercially available catheters need not be the major consideration in catheter selection.

Venous Site Selection and Catheter Placement (Fig. 6)

The most accessible veins in the calf are the jugular veins. The jugular vein is readily distinguished, except in the most severely dehydrated calves, by occluding flow with digital pressure in the jugular groove near the thoracic inlet. The vein can be distended by gentle massage in the jugular groove with strokes moving from the mandible toward the thoracic inlet. Elevation of the hind end and lowering of the head may facilitate distention and visualization of the jugular. Once the vein has been identified, a site approximately one third of the distance from the head to the thorax should be selected and clipped. A complete surgical preparation should be performed; then a small stab incision may be made through the skin at the insertion site. The dermis should be incised, but the vein should be avoided, since a hematoma will result if it is punctured. The final skin preparation should be repeated. With care taken not to handle the plastic indwelling portion, the catheter is introduced into the vein and directed toward the thoracic inlet. The catheter hub is sutured to the skin with nonabsorbable suture. An adhesive tape "butterfly" may be first applied to the catheter hub and affixed to the skin.⁶³ Fluids may be administered or heparin-saline (1000-10,000 U/L) flushed through the catheter to reduce risk of thrombosis as the catheter is sutured in place. A gauze dressing with or without antibiotic cream may be placed over the catheter and held in place by a nonrestrictive bandage.^{41,69}

Alternate sites of venous access include the saphenous vein on the medial aspect of the hind limb. In veins with low flow rates such as the saphenous vein, infusion of hypertonic or irritant solutions will often result in the rapid development of thrombosis and thrombophlebitis.³⁴ These veins should be reserved for use with isotonic, nonirritant solutions. If it is necessary to give an irritant drug, it should first be diluted in several volumes of saline or 5% dextrose; then the total volume should be administered slowly.

In severely dehydrated calves, percutaneous catheterization of the jugular veins may be difficult because the vein is often collapsed. In such cases, a cutdown procedure may be necessary and may be the most rapid method of placement.

Catheter Complications

Use of intravenous catheters is associated with several complications that reflect the invasive nature of intravenous placement. Thrombosis with thrombophlebitis is the most frequent complication.^{3,13,27,52,71,74,75} Additional and potentially more serious complications are embolus formation, perivascular infiltration, catheter sepsis, perivascular cellulitis, and septicemia.

A thrombus begins to form within 20 minutes of catheter placement.⁶⁸ Although most catheter materials are biologically inert, catheters induce thrombus formation directly as a foreign material by activating the clotting cascade or indirectly by promoting platelet adhesion, activation, and degranulation.³ In addition, thrombosis formation is promoted by trauma to the vascular intima incurred during catheter insertion, by intimal damage secondary to catheter tip movement during infusion of fluids, or by intimal inflammation associated with the infusion of irritant fluids or drugs.³⁴

Thrombus formation is influenced by catheter gauge and length. In general, thrombus size is related to catheter size,^{24,26,54} with small catheters inducing small thrombi that are less likely to occlude the vein.²⁴ The type of plastic used in catheter construction and the method of construction also has an influence on the tendency to induce



Figure 6. Placement of an over-the-needle catheter in a jugular vein. The area has been clipped and prepared as for surgery. An incision has been made through the dermis. A, The catheter is inserted at an acute angle and should enter the vein immediately after traversing the skin. Note that the catheter is held by the hub only and is directed toward the thoracic inlet. B, The catheter may be flushed with heparin-saline to assess proper placement and prevent thrombosis. An extension set, previously filled with heparin-saline, has been attached to the catheter. D, Nonabsorbable suture is secured around the catheter hub. Illustration continued on opposite page

thrombosis. Catheters constructed of polyvinyl chloride and polyethylene cause more local irritation than do fluorethylene propylene (Teflon) or tetra-fluoroethylene resins.¹⁴ Ethylene acrylic acid catheters were less frequently associated with thrombus formation then Teflon, polyvinyl chloride,⁷¹ polypropylene, or polyethylene catheters.⁶⁴ Polyurethane is less thrombogenic than either polyvinyl chloride or silicone.⁶⁸

A major determinant of the variation in the tendency to induce thrombus formation between different types of polymers is roughness of the catheter surface. Catheters constructed of polymers with rough surfaces promote greater thrombus formation than those with smoother surfaces.^{25,26} Manufacturing technique also influences surface roughness such that catheters made of identical polymers can have marked



differences in surface roughness and thrombogenicity as a result of differences in manufacturing conditions.²⁴

The risk of catheter complications increases with increasing duration of catheter placement.^{6,9,34,71} In humans, thrombophlebitis most often develops within 70–75 hours of placement,^{27,52} which has led to the recommendation that catheters should be removed within 24–48 hours of insertion. Conscientious removal of a catheter does not completely alleviate risk, however, as thrombophlebitis may still develop up to 48 hours after removal.^{27,52}

The chemical properties of solutions infused have an important influence on development of thrombophlebitis. Solutions that are hypertonic (e.g., 50% dextrose and some total parenteral nutrition solutions) are prone to production of chemical injury of the intima and ultimately to thrombophlebitis. Such solutions should only be administered in veins with high blood flow rates, such as the jugular veins, or via central venous catheters.^{3,34} Solutions and drugs that are strongly acidic or alkaline also induce injury to the intima. Dextrose solutions and normal saline with pH values ranging from 4.3 to $5^{3,34}$ may predispose to thrombophlebitis. Irritating drugs such as thiobarbiturates, phenylbutazone, and the aminoglycosides are also associated with chemical damage to vessels.^{3,52}

The degree of tissue trauma during insertion also influences risk of subsequent thrombosis.³⁴ Catheter insertion by specially trained or experienced personnel reduces the incidence of thrombosis.^{3,34,75} Risk of postinsertional complications appears to be reduced as well by increased experience in catheter monitoring.⁷⁴

Although thrombophlebitis that develops following catheter placement is usually nonseptic inflammation, presence of a thrombus on the catheter or in the vein lumen predisposes to development of septic thrombophlebitis, perivascular cellulitis, and septicemia.^{3,9,13,34,71} Bacteria may also adhere directly to the tip of some types of catheters, even in the absence of a thrombus.⁶⁵ In humans, the majority of organisms isolated from catheter tips are bacteria ubiquitous in the hospital environment.^{9,40} The infecting bacteria in catheter sepsis may be introduced during catheter insertion or subsequent to insertion by manipulation of the catheter or improper handing of administration lines and intravenous medications. Bacteria may also colonize from a remote site following hematogenous spread.

Diagnosis and Treatment of Catheter Complications

Thrombophlebitis presents with signs of induration; the affected area is warm, swollen, and painful to palpation or manipulation, although in some cases the animal may only be febrile before any other signs develop. The affected vein may be hard and "corded," depending on the extent of thrombosis.^{3,13,70} Blood flow may be partially or completely obstructed. In complete occlusion, there may be swelling of the drained area. With bilateral jugular vein obstruction, the head may become severely swollen.

Infection within the vein or perivascular tissues may be difficult to detect. The first sign of catheter-related sepsis may be an unexplained fever, appearance of an inflammatory leukogram, or unexplained clinical deterioration.^{3,13,70} Infection may be evident by suppuration from the catheter site. After catheter removal, gentle manipulation of the area or manual distention of the vein may aid in demonstrating purulent exudate.³ Confirmation of sepsis may only be obtained by positive catheter tip culture with or with positive blood culture.

When thrombophlebitis or catheter-related sepsis is recognized or suspected, the catheter should be removed immediately^{3,13} and the catheter tip cultured, if possible, with a semiquantitative technique.⁴² A Gram's stain preparation of the catheter tip may demonstrate bacteria and serve as a guide for antibiotic selection.¹⁰ Another catheter may be placed in an alternate site if necessary. The affected area may be treated with local applications of hot or cold packs. Clinical signs of thrombophlebitis often improve within 24 hours of catheter removal. In cases of suspected catheter-related sepsis, local treatment should be combined with systemic broad-spectrum antibiotic therapy or with antibiotic therapy guided by culture and sensitivity tests. Cases of deep-seated infections involving the vein or surrounding tissues may require surgical drainage.¹³

In cases of perivascular infusion with irritating medication, the tissues should be infiltrated with a large volume of saline to dilute the medicant and promote its dispersal. The area may be treated with hot compresses and hydrotherapy.¹³

Catheter Care Recommendations

Risk of thrombophlebitis and catheter-related sepsis can be reduced by careful catheter placement and management.^{3,13,69,70} The use of prophylactic systemic antibiotics does not lessen the risk of thrombophlebitis or catheter-related sepsis.^{34,40} The catheter should be inserted using aseptic technique after full surgical preparation of the skin. The catheter should be inserted as atraumatically as possible to reduce damage to the intima. The catheter should be securely anchored in place. Use of an extension set will reduce likelihood of bacterial colonization by reducing catheter manipulation.^{3,39} A sterile dressing with or without antiseptic or antibiotic ointment may reduce risk of infection.^{3,13,41,70} The dressing should be kept clean and replaced every 24 hours or more frequently if it becomes soiled. The insertion site should be cleaned and redressed each time the bandage is changed.

Continuous fluid flow should be maintained through the catheter.³ If this is not possible, the catheter should be flushed with heparinsaline (1000-10,000 U/L) every 4-6 hours. Heparin-saline for flushing should not be retained for more than 24-48 hours to reduce the potential for bacterial contamination.³ During any manipulation of the catheter, extension set, administration set, or fluid container, attention must be given to aseptic technique. Injection ports should be wiped with alcohol before inserting a needle.

In the absence of complications, a catheter should be removed within 48-72 hours.^{3,13,40,70} A new catheter may be placed at another site if continued venous access is desired. Any catheter should be removed unless its continued use is necessary. If possible, the catheter site should be monitored for 1-2 days after catheter removal.²⁷

SUMMARY

Intravenous fluid therapy is valuable as primary or ancillary therapy for many conditions in calves. The first step in developing an intravenous fluid plan is determination of the volume needed to replace the fluid deficit, estimated on the basis of clinical signs. The parenteral solution to be used is chosen to be similar in electrolyte composition to the fluid lost. Abnormalities of specific electrolytes, acid-base balance, and energy metabolism can be addressed during volume replacement; this requires an understanding of the pathophysiology of the primary clinical abnormality. The fluid delivery system is composed of an administration set and an intravenous catheter. The administration set allows sterile delivery and regulation of the rate of administration. An intravenous catheter is selected, inserted, and maintained to minimize the potential for catheter-related complications.

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