INTRAVENOUS FLUID THERAPY OF CALVES

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Dehydration in neonatal calves with diarrhea is a common cause of death. Despite progress in understanding the pathophysiology of the disease, calf diarrhea remains a major cause of economic loss to the cattle industry. Good calf-raising management and vaccination to produce hyperimmune colostrum in the dam's mammary gland can reduce the incidence of neonatal diarrhea[22, 103]; however, neonatal diarrhea remains a major cause of financial loss because of treatment costs and the loss of genetically valuable replacement heifers and bulls.

According to the World Health Organization, the development of successful oral rehydration therapy is one of the major advances of human medicine in this century. Oral rehydration therapy has saved numerous human infants and neonatal animals with diarrhea; however, oral rehydration may not rapidly restore the hydration status of moderately to severely dehydrated calves. In these circumstances, intravenous fluid therapy remains the best treatment option for dehydration. The approach to intravenous fluid therapy should be simple and cheap to serve the needs of practitioners and agricultural producers. The purpose of this article is to provide an overview of intravenous fluid therapy in calves using practical on-farm treatment options.

INDICATIONS FOR INTRAVENOUS FLUID ADMINISTRATION

Severe dehydration due to intestinal loss of fluid and electrolytes is the most frequent indication for oral and intravenous fluid therapy in

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calves with diarrhea. Neonatal calf diarrhea occurs in the first 4 weeks of life and usually is caused by mixed infections with viral (rotavirus, coronavirus), bacterial (*Escherichia coli*, *Salmonella* spp), and protozoal (*Cryptosporidium parvum*) pathogens. Neonatal calves have a higher total body water content (~75% body weight) and a higher extracellular fluid volume (~45% body weight) than adult herbivores. Calves are also believed to be more sensitive to fluid losses than adult cattle, and the high proportional water content in calves is not believed to serve as a water reservoir and has no protective effect against dehydration.[43, 89] Fecal fluid losses in profuse watery neonatal diarrhea can reach 13% to 18% body weight per day, and are often underestimated in clinical cases.[22, 89] In extreme cases, calves can lose up to 21% of their body weight in 24 hours when oral fluids are given at the same time.[22] The kidneys can compensate for increased fluid losses by decreasing urine production, but if losses exceed fluid intake, dehydration follows.[45, 84]

Large fecal losses of water, sodium, potassium, chloride, and bicarbonate in neonatal calf diarrhea decrease the extracellular fluid volume, causing decreased plasma volume and venous return, extracellular dehydration, and total body loss of water and electrolytes. The fluid and electrolyte losses in the early stage of neonatal calf diarrhea are primarily of secretory origin and, to a much lesser extent, of osmotic origin.[21] Even in viral infections with rotavirus or coronavirus and in protozoal infections with cryptosporidia, secretory mechanisms are believed to contribute to the diarrhea and dehydration.[21]

Hyponatremia is common in dehydrated calves with diarrhea,[13, 76, 79] but hypernatremia can also occur, particularly when calves are treated with oral electrolyte solutions and do not have access to fresh water.[98, 101] The total body potassium content is decreased in diarrhea, but hyperkalemia is a frequent observation in diarrheic calves with acidemia. Hyperkalemia is the result of complicated and not fully understood mechanisms involving hyponatremia, hypo-osmolality, acidemia, and cellular hypoxia.[76] Neonatal diarrhea usually causes a hypo-osmotic extracellular dehydration with decreased extracellular fluid volume (plasma and interstitial) and a small increase in intracellular fluid volume.[24, 78, 79] Hyperosmotic dehydration may be observed in chronic diarrhea and shortly before death.[44, 45]

The cost-effective use of electrolyte-containing oral rehydration solutions for the treatment of diarrheic calves is popular and widely accepted.[72] Calves with diarrhea presented to veterinary practitioners or teaching hospitals therefore have often been treated with oral fluids of varying compositions. Recent observations found that approximately 50% of diarrheic calves had been treated with oral fluids alone or in combination with other treatments before they were admitted to a referring practice or teaching hospital.[3, 22, 37, 46, 59] Mixing errors of oral rehydration solutions occur frequently, and witholding milk is common practice by some farmers.[80, 98] Therefore, hypernatremia and hyperchloremia can be found in calves with diarrhea, particularly if the calf has not had access to fresh water.[33, 45, 80, 89, 98] Calves often are in poor body condition

when they have been treated with oral fluids for several days without feeding milk.[22, 85] According to these observations, it can be assumed that electrolyte disturbances and the type of dehydration (iso-, hypo-, or hyperosmotic) can vary among individual cases presented for treatment, and are very difficult to predict.

The widely accepted indication for intravenous fluid administration in dehydrated calves is a decrease of 8% body weight, although experimental evidence supporting 8% dehydration as the most appropriate intervention point is currently unavailable. A recent study did demonstrate, however, that calves that were 8% dehydrated required at least 24 hours to be adequately rehydrated by orally administered electrolyte solutions.[11] Recumbent, severely depressed, or comatose calves, and calves without a suckle reflex, also need intravenous fluid resuscitation.[7, 13, 22, 39, 62, 84, 85, 89, 91, 103] Calves that do not fall into these categories but show a rapidly progressing dehydration with consistent profuse watery diarrhea should also be treated intravenously rather than rehydrated by ororuminal intubation.[73] If treatment with oral fluids is not successful and only a weak suckle reflex is present, initial intravenous restoration of fluid and electrolyte deficits is preferred.[85] Collapsed dehydrated calves in severe hypovolemic shock are not able to rapidly resorb sufficient amounts of oral or subcutaneously administered fluids and should be rehydrated intravenously.[7, 11, 27, 62, 73] Resuscitation by intravenous fluid administration will restore oxygen delivery and remove the metabolic products of poorly perfused tissues.[14]

Another indication for intravenous fluid therapy is resuscitation of severely depressed calves that have asphyxia and mixed respiratory-metabolic acidosis.[42, 67, 86] Moreover, severely depressed calves with acidemia but without clinical signs of dehydration also need intravenous alkalinizing fluids to restore the disturbed body functions.[50, 52, 93]

Based on the this, the goals of intravenous fluid therapy in dehydrated calves should be to

  correct extracellular dehydration and restore circulating blood volume;
  correct the acidemia (increase blood pH >7.20);
  correct mental depression;
  restore the suckle reflex;
  correct electrolyte abnormalities;
  correct the energy deficit;
  facilitate repair of damaged intestinal surface.

In summary, neonatal calf diarrhea (independent of the causing agent) induces a similar clinical syndrome of (1) dehydration (most often iso- or hypo-osmotic, later in disease process, may become hyperosmotic) and azotemia leading to hypovolemic shock, (2) metabolic acidosis, (3) electrolyte imbalances (often hyponatremia and hyperkalemia, less frequently hypochloremia and hypernatremia), and (4) negative energy balance with or without hypoglycemia.[51, 89] The risk of hypoglycemia increases with time from last feeding[85] or duration of oral electrolyte

solution administration, particularly when low energy solutions are administered.[12]

ASSESSMENT OF HYDRATION STATUS

Serial measurements of body weight are needed for the exact determination of hydration status and the degree of dehydration in calves. As this is not practical, conventional classification systems for dehydration on the basis of clinical symptoms have been used on an empirical basis to guide calculation of dehydration in diarrheic calves.[47, 51, 87] As fluid losses in diarrheic calves are primarily from the extracellular space, the clinical assessment of existing deficits is based on estimating the loss of extracellular fluid volume.[13] Guidelines to predict fluid losses were first presented by Watt,[105] who classified dehydration as mild, moderate, and severe. These three categories have been widely used and extrapolated to evaluate hydration status in calves.[7, 47, 50–52, 87] These guidelines have included the degree of enophthalmus, skin tent duration, moisture and color of mucus membranes, and capillary refill time. Other factors that have been investigated have included serial determinations of packed cell volume (PCV), hemoglobin and plasma protein concentration, the temperature of extremities (ear and distal limb), and measurement of mean central venous pressure. Several authors have assigned similar percentages of decrease in body weight to three categories of mild, moderate, and severe dehydration without proof on supporting data.[88] A recent study from Constable and coworkers[13] proved the usefulness of various clinical signs of dehydration in calves and verified the

Table 1. EXPERIMENTALLY DETERMINED GUIDELINES FOR THE EVALUATION OF HYDRATION STATUS IN NEONATAL CALVES WITH DIARRHEA.

Dehydration (%)	Clinical Signs
0	No enophthalmos; cervical skin-tent duration, ≤2 s; moist mucous membranes
2	Enophthalmos, 1 mm; cervical skin-tent duration, 3 s; dry mucous membranes
4	Enophthalmos, 2 mm; cervical skin-tent duration, 4 s
6	Enophthalmos, 3 mm; cervical skin-tent duration, 5 s
8	Enophthalmos, 4 mm; cervical skin-tent duration, 6 s; cool extremities
10	Enophthalmos, 6 mm; cervical skin-tent duration, 7 s; cold extremities
12	Enophthalmos, 7 mm; cervical skin-tent duration, >8 s; cold extremities
≥14	Enophthalmos, >8 mm; cervical skin-tent duration, >10 s; cold extremities; white mucous membranes

Adapted from Constable PD, Walker PG, Morin DE, et al: Clinical and laboratory assessment of hydration status of neonatal calves with diarrhea. J Am Vet Med Assoc 212:991–996, 1998; with permission.

preciseness of these predictors for use in clinical practice. In a study with an experimental diarrhea model that induced acute and severe dehydration, the best predictors of the magnitude of dehydration were extent of eyeball recession into the orbit, skin elasticity on the neck and thorax, and plasma protein concentration (Table 1).[13] The degree of enophthalmos was estimated by everting the lower eyelid to its normal position and estimating the recession of the globe into the orbit. Skin elasticity was measured in the lateral midcervical area by pinching a skinfold, rotating it 90 degrees, and measuring the time for the skinfold to disappear.[13]

The new experimentally determined guidelines to assess hydration status in diarrheic calves are reproduced in Table 1. They provide the most practical and accurate method for predicting hydration status in calves with diarrhea, and are therefore useful for determining the need for intravenous fluid therapy. Additionally, Figure 1 shows the relationship between the degree of enophthalmus and the corresponding dehydration in percentage of body weight found in this study.[13] The results of this study indicated that dehydration can be detected at lower values than previously thought, in that dehydration at 2% body weight appeared with skin tent duration of 3 seconds, a slight enophthalmus of 1
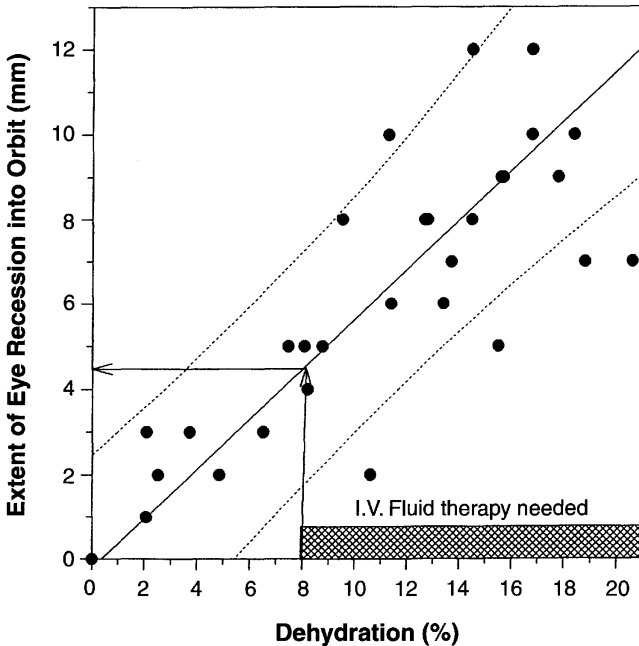

Figure 1. Relationship between enophthalmos (mm) and dehydration (% body weight) with regression line and 95% confidence interval (*Modified from* Constable PD, Walker PG, Morin DE, et al: Clinical and laboratory assessment of hydration status of neonatal calves with diarrhea. J Am Vet Med Assoc 212:991–996, 1998; with permission.)

mm, and with dry mucous membranes. Enophthalmus and cervical skin-tent duration increased linearly with percentage of dehydration (see Fig. 1). At 8% dehydration, enophthalmus was 4 mm, cervical skin-tent duration was 6 seconds, and the extremities felt cool. Very severe and life-threatening dehydration of 12% or 14% or more displayed enophthalmus of 7 or 8 mm or more, skin tent duration of more than 8 or 10 seconds, and cold extremities and white mucous membranes (see Table 1; Fig. 1).[13] These guidelines provide the practitioner a practical and accurate method to hydration status in calves with diarrhea.

Limitations of the guidelines from Table 1 are calves with chronic diarrhea and in calves where fluid losses are into the third space.[13, 102] As the position of the eye within the orbit is also dependent on body fat stores and not only the hydration status, it may therefore be of limited use to determine hydration status in calves with chronic diarrhea and dehydration.[13] In cachectic calves or calves with chronic diarrhea, pliability of the skin of the neck and thorax is the most exact and useful indicator.[13] Because assessment of hydration status with charts and tables will always be subjective and initial estimates may be inaccurate, it is advised to "guess and to reassess" the patient several times during rehydration therapy.[35, 88]

ASSESSMENT OF NEED FOR INTRAVENOUS FLUID ADMINISTRATION

A more advanced but less useful tool to predict the need for intravenous fluid therapy in diarrheic calves is measuring rectal temperature and peripheral temperature at the fetlock with a portable thermistor.[14] A rectal temperature less than 38.0°C (<100°F), indicating hypothermia and a decrease in skin temperature of the extremities (ears and limbs), is commonly observed in calves with diarrhea.[3, 14, 50–52, 71, 101] Hypothermic diarrheic calves exhibit an increase in the difference between temperature of the rectum and the skin of extremities.[14, 23, 78] Because cardiac output is positively correlated with rectal and peripheral temperature as well as hydration status, a reduction in cardiac output below 65% of normal should be suspected if the peripheral skin temperature over the fetlock joint of hind limbs is below 32°C (90°F) or if the temperature difference between the rectum and fetlock is beyond 7°C (13°F).[14] In Figure 2 the relationship between a decrease of peripheral fetlock temperature associated with a decrease of cardiac index below 65% of normal (<170 mL $\times$ min^{-1} $\times$ kg^{-1}) is shown in calves with various degrees of dehydration (Fig. 2). Calves with a cardiac output greater than 65% of normal did not exhibit a decrease in peripheral temperature (see Fig. 2).[14]

It is not known what percentage decrease in cardiac output should be used to guide intravenous fluid therapy in calves, but to preserve satisfactory cardiac output during fluid therapy, a peripheral temperature of more than 32°C (fetlock) and a difference between rectal and
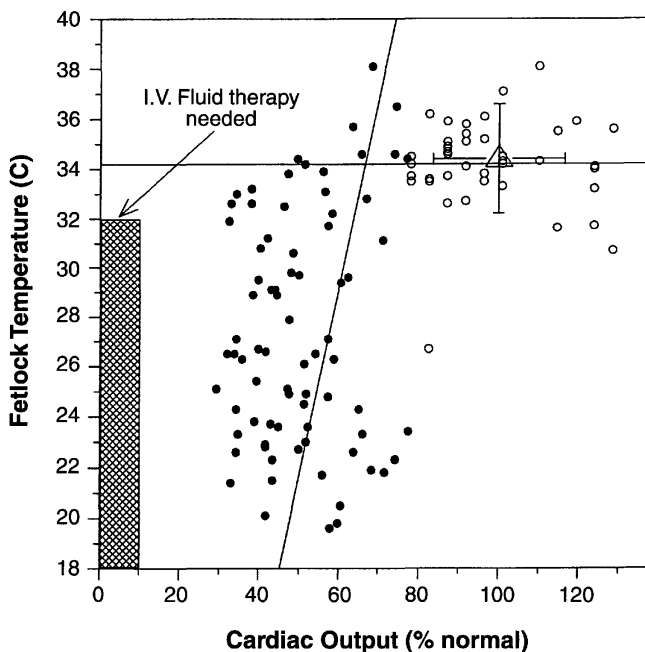
Figure 2. The relation between peripheral temperature and cardiac index in calves with various degrees of dehydration housed in a thermoneutral environment. Linear regression lines for cardiac output <65% normal *(solid circles)* and >65% normal *(open circles)*. Triangle with error bars indicates reference values for healthy neonatal calves. (*Modified from* Constable PD, Walker PG, Morin DE, et al: Use of peripheral temperature and core-temperature difference to predict cardiac output in dehydrated calves housed in a thermoneutral environment. Am J Vet Res 59:874–880, 1998; with permission.)

peripheral temperature of less than 7°C should be maintained for effective fluid therapy if calves are housed in a thermoneutral environment.[14] Assessment of ear temperature is less accurate than fetlock temperature, and hands are much less accurate at measuring fetlock temperature than thermistors[14]; however, peripheral temperature determinations are limited and should not be performed when calves are housed below 10°C (50°F), as physiological peripheral vasoconstriction decreases skin temperature.[14] This greatly limits the utility of peripheral temperature in guiding the need for intravenous fluid therapy.

Heart rate is a poor predictor of the need for intravenous fluid therapy and the presence of hyperkalemia. A study of a large number of calves with naturally aquired diarrhea showed no correlation between heart rate and blood potassium concentration,[53] although another study found that heart rate increased linearly with serum potassium concentration, up to $[K^+] = 8$ mEq/L.[10] Bradycardia, defined as less than 90 beats per minute, is seen in hypothermia, hypoglycemia, and hyperkalemia.[73] Auscultable cardiac arrhythmias in dehydrated diarrheic calves are be-

lieved to indicate the presence of severe hyperkalemia ([K$^+$] >8 mEq/L).[73, 106] Therefore it is recommended that if cardiac arrhythmias *and* bradycardia (<90 beats/min) are present that sodium bicarbonate should immediately be administered intravenously because of the likelihood that life-threatening hyperkalemia is present.[73] Additional stress should be avoided during therapy of hyperkalemic calves, as excessive restraint may cause sudden collapse and death of severely shocked calves.[54]

ASSESSMENT OF ACIDOSIS

Metabolic acidosis is a common finding in calves with diarrhea. Treatment of acidemia with alkalinizing solutions is important for restoration of normal body function and suckle reflex. The gold standard method for determining the severity of metabolic acidosis is blood gas analysis, and the recent availability of portable blood-gas machines has made such analysis more practical. A less expensive hand-held portable pH-meter (Cardy Twin pH meter, Spectrum Technologies, Inc., Plainfield, IL) has also been used to measure blood pH.[64, 65] Other laboratory methods for determining the severity of metabolic acidosis have also been studied. Total CO_2 concentration (TCO$_2$), determined either with the simple Harleco apparatus or with automated laboratory electrolyte analyzers, provides an accurate guide to plasma bicarbonate concentration and can therefore be used to screen for metabolic acidosis.[3, 32, 36, 37, 68] TCO$_2$ determination also enables calculation of the amount of buffer needed to correct a metabolic acidosis[32, 68]; however, clinical prediction of acidemia is much more commonly done in veterinary practice than measurement of acid-base status. Metabolic acidosis in calves with and without dehydration can be predicted from their ability to stand, suckling force, level of depression, and their age with variable success.[3, 30, 50–52, 71] In contrast, the degree of enophthalmos and peripheral temperature should not be used to predict the severity of metabolic acidosis, as acidosis is not correlated with degree of dehydration,[3, 13, 37, 52, 69, 71] and severe metabolic acidosis can be found in calves with depression and ataxia or recumbency but minimal or no diarrhea and dehydration.[1, 37, 50, 52, 93]

In several landmark papers, Naylor[69–71] identified that age differences in the severity of acidemia in diarrheic calves could be used to facilitate calculation of buffer therapy. Diarrheic calves in their first week of life were less acidemic than calves more than 8 days of age, the latter having almost twice the base deficit of younger calves. The finding that diarrheic calves older than 1 week of age had a more severe metabolic acidosis has been confirmed in several other studies.[1, 3, 36, 37] For use in practice, Naylor[70] devised a simple protocol for the treatment of metabolic acidosis in diarrheic calves less than 8 days and more than 8 days of age, which has been adapted and modified by others.[18, 28, 66, 107] Recently, data were obtained from 65 calves with naturally occuring diar-

rhea that permitted estimation of base deficit using either suckling force or ability to stand alone without differentiation for age.[30] When suckle reflex was strong, weak, or absent, diarrheic calves had a mean base deficit of 4.2 mEq/L, 11.4 mEq/L, or 21.5 mEq/L, respectively. Diarrheic calves that were standing strongly, weakly, or were unable to stand had a mean base deficit of 5.2 mEq/L, 7.8 mEq/L, and 19.1 mEq/L, respectively.[30] A modified algorithm for a diagnostic and therapeutic approach of diarrheic calves on the basis of Naylor's original work and several other studies is presented in Figure 3.

It is also important to closely examine diarrheic calves for signs of septicemia (<5 days, extended neck, congested scleral vessels, hypopyon, iridospasm), pneumonia, navel illness, and joint swelling. This will establish a better prognosis of fluid therapy, as diarrheic calves may often be bacteremic showing positive blood culture.[25, 73]

In summary, newly developed guidelines enable practical and accurate assessment of hydration status in dehydrated calves. The best predictors for the percentage of dehydration are degree of enophthalmos, skin tent duration on the neck and thorax, and plasma protein concentration.[13] Age differences should be considered in assessing the severity of metabolic acidosis and for determining the bicarbonate requirements of diarrheic calves.[69] Calves with diarrhea and dehydration in their first week of life are less acidemic than older calves, who require more
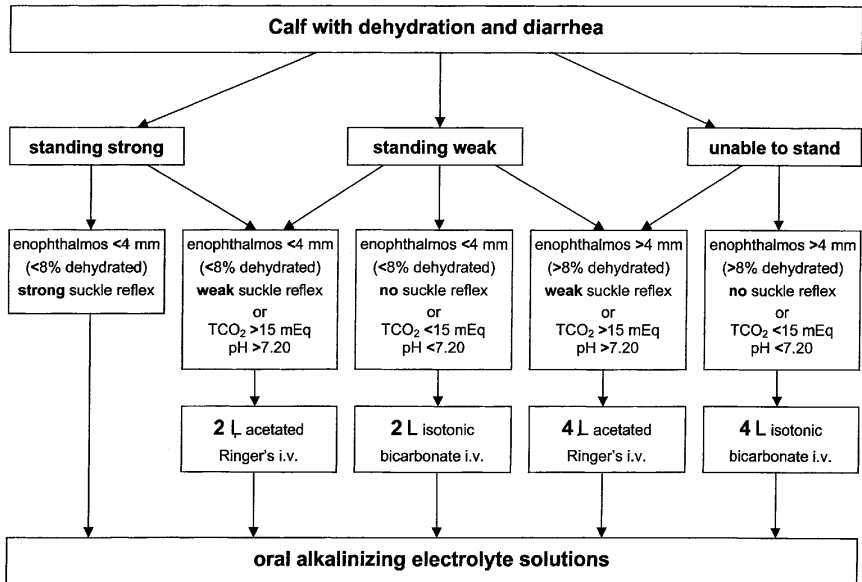

Figure 3. Algorithm for initial on-farm fluid therapy of dehydrated calves with diarrhea. TCO_2 concentration in blood on the farm can be determined with the Harleco-apparatus (American Scientific Products, McGaw Park, IL)[3, 32, 68] and blood-pH with a portable Cardy Twin Waterproof pH meter (Spectrum Technologies, Plainfield, IL).[64, 65]

sodium bicarbonate to correct their metabolic acidosis. Calves that are unable to stand or have a weak or absent suckle reflex have a more severe metabolic acidosis and require intravenous sodium bicarbonate to correct their acidemia.

SOLUTIONS FOR INTRAVENOUS ADMINISTRATION

Preparation

Commercially prepared intravenous solutions are not always readily available and may often be too expensive for routine use in calves.[35] Moreover, compounds necessary for an effective treatment outcome (such as sodium bicarbonate) are rarely available in commercial solutions.[74] Practitioners therefore tend to make their own intravenous solutions for use in calves. Homemade solutions should ideally be prepared using nonpyrogenic sterile water because heat-stable exotoxins, as well as pyrogens, may be present in tap water, and bicarbonate-calcium complexes may form.[16, 56] The use of distilled water and easy-to-sterilize glass containers provides a practical method to make cheap and safe intravenous fluids.[74, 87, 88] The recommended size for containers varies from 1 L to 1 gal.[87] The author prefers the use of 1.0 L hard-plastic or glass bottles because they can be easily sterilized, warmed, transported, and handled. A more detailed description on how to prepare fluids and equipment for intravenous fluid therapy of cattle is given by Corke.[16]

Types of Solutions

In general, solutions are classified as either *crystalloid* or *colloid* and as either *balanced* or *unbalanced* solutions.[20, 102] Balanced solutions resemble the composition of extracellular fluid (e.g., lactated Ringer's), whereas unbalanced solutions are different from extracellular fluid (e.g., 0.9% NaCl).[20] The solutes of crystalloid solutions are substances that form an aqueous or true solution, can be crystallized, and are distributed in all body fluid compartments.[20] The compounds of crystalloid solutions are electrolytes, mainly based on sodium and chloride and organic compounds like dextrose or lactate.[102] Sodium is the backbone of the extracellular fluid. To increase or maintain extracellular volume of the body, isotonic infusion solutions ($\sim$300 mOsm/L) must contain a sodium concentration of approximately 140 mEq/L. Solutions that contain less sodium do not resuscitate dehydrated calves as effectively as 0.9% NaCl, which has a sodium concentration of 154 mEq/L.[34] *Colloids* are substances with a high molecular weight that are too large to pass through a semipermeable membrane. Colloids are restricted to the plasma compartment and thereby provide sustained plasma volume expansion. Examples for *colloid* solutions are whole blood, blood substi-

tutes, plasma, and high molecular glucose polymers, like dextran-70 and hydroxyethyl starch, which are discussed elsewhere.

Alkalinizing Solutions

For fluid therapy in neonatal calves it is important to distinguish between alkalinizing and nonalkalinizing solutions. Because metabolic acidosis is almost always present in calves requiring intravenous administration, calves require administration of alkalinizing buffer substances. The rapid correction of severe base deficits due to fecal bicarbonate losses has the highest priority in the treatment of calves with neonatal diarrhea.[91] Administration of alkalinizing solutions will minimize the deleterious effects of acidemia and restore the suckle reflex.[49, 51, 91] Sodium bicarbonate is the alkalinizing agent of choice and is most often used as a 1.3% isotonic solution (13 g $NaHCO_3$/L). Isotonic sodium bicarbonate is the most important solution for the treatment of metabolic acidosis in calves with and without dehydration. Higher concentrations of sodium bicarbonate solutions (4.2% or 8.4%) have also been recommended, but as they may be deleterious, isotonic bicarbonate is preferred.[3] The tremendous clinical and successful therapeutic effects of isotonic sodium bicarbonate containing 155 mEq/L bicarbonate buffer anions (HCO_3^-) and equal amounts of sodium has been described in a large number of interventional and therapeutic studies. Naylor's group first confirmed the overall advantage of sodium bicarbonate as a rapid alkalinizing and resuscitating agent for intravenous buffer therapy of metabolic acidosis in dehydrated calves.[51, 69, 75] Other metabolizable bases or bicarbonate precursors, such as lactate, and synthetic buffer substances, such as Trisbuffer, have also been used[31, 96]; however, sodium bicarbonate has proven to be more effective. A recent study compared two relatively new and non–CO_2-producing buffer mixtures, Carbicarb and Tribonate, with CO_2-producing sodium bicarbonate for the treatment of metabolic and mixed respiratory-metabolic acidosis in calves[3, 4]; however, the results indicate that further research is needed before Carbicarb can be recommended for the use in practice.

Metabolizable bases do not correct severe metabolic acidosis as fast as sodium bicarbonate because these substances have to be metabolized to induce an alkalinizing effect.[51, 75] For example, lactate showed a delayed effect increasing the blood pH compared to sodium bicarbonate and sodium acetate.[51] Another disadvantage of lactate is that blood lactate concentration in acidemic calves is already increased and lactate metabolization is simultaneously decreased.[8, 51, 93] This means that lactate should not be administered to severely acidemic calves, and it is preferable to use acetate (e.g., Isolyte-E or Plasma-Lyte-R) instead of lactate as an alkalinizing agent in mildly to moderately acidemic calves.[86, 94] Metabolization of acetate is faster than lactate, therefore alkalinization is more rapid with acetate.[51] In general, sodium bicarbonate should be used for the treatment of severe acidemia (pH <7.20, base-deficit >10

mEq/L, or TCO_2 <15 mEq/L), and acetated or lactated Ringer's solution should be used to correct less severe metabolic acidosis.

Combinations of solutions containing potassium in addition to sodium bicarbonate or metabolizable bases have been advocated for the use in hyperkalemic diarrheic calves to decrease blood potassium concentration and correct the coexisting total body deficit of potassium avoiding rebound hypokalemia.[34, 87] Decreasing potentially dangerous hyperkalemia in calves with experimentally induced diarrhea and dehydration was more effective with solutions containing small concentrations of potassium (5 mEq/L) than with higher concentrations (35 mEq/L),[34] but other studies reported no deleterious effect when higher potassium concentration solutions were used[39, 77]; however, infusion of isotonic sodium bicarbonate solution without adding potassium combined with continuous or early feeding of milk and access to fresh water produced cure rates greater than 80%, even without administering oral electrolyte solutions.[29, 57] It is unknown if supplemental administration of oral electrolyte solution will further improve survival rates.

Nonalkalinizing Solutions

Nonalkalinizing solutions are more frequently used in fluid therapy for mature cattle, as adult cattle tend to get alkalemic instead of acidemic.[90] Besides isotonic saline solution (0.9% NaCl), the classical balanced polyionic and isotonic crystalloid standard fluid for adult ruminants is Ringer's solution, which contains nearly physiologic concentrations of sodium, potassium, calcium, and chloride. Of greater importance in calves are nonalkalinizing solutions, such as 5% to 10% dextrose, to counteract negative energy balance in diarrheic calves with or without manifest hypoglycemia.[89] Fifty grams of dextrose added to 1 L of distilled water or a 1-to-9 dilution of 50% dextrose solution with distilled water provide an isotonic 5% dextrose solution. In dehydrated calves a plain 5% dextrose solution is not helpful in correcting extracellular fluid deficits because the solution contains no sodium. After dextrose is metabolized, free water is left that quickly diffuses out of the bloodstream. To provide energy and maintain extracellular fluid volume, 25 to 50 g dextrose or 50 to 100 mL of 50% dextose solution can be added per liter of 0.9% saline or 1.3% sodium bicarbonate solution to make a mildly hypertonic solution. Solutions without an alkalinizing agent do not correct acidemia after successful replacement of fluid deficits and restoration of cardiovascular and renal functions. Either large amounts of isotonic saline or small amounts of hypertonic saline solutions do not significantly alter base deficits in acidemic or in healthy calves.[11, 51, 75, 104] Solutions without an alkalinizing agent are therefore of limited use in calves with diarrhea and metabolic acidosis.[11]

Hypertonic Solutions

In early calf diarrhea, hypo-osmotic dehydration with an increased intracellular fluid volume is present.[17, 24, 44, 79] It is logical, practical, and economic to use hypertonic rather than isotonic solutions to move some intracellular fluid into the extracellular space by creating an osmotic gradient and reversing the hemoconcentration of dehydrated calves.[11, 61, 104] A detailed discussion on the use of hypertonic saline solutions is discussed elsewhere in this issue.

In summary, veterinarians need only four types of intravenous solutions for treating calves: acetated Ringer's, isotonic sodium bicarbonate, hypertonic saline, and dextrose solution. Alkalinizing fluids are required for intravenous rehydration of calves with diarrhea and dehydration. Acetated Ringer's solution should be administered to calves with mild to moderate acidemia. Isotonic sodium bicarbonate (1.3% = 13 g $NaHCO_3$/L = 155 mEq HCO_3^-/L + 155 mEq Na^+/L) at a dose of 1 to 4 L is recommended for intravenous treatment of dehydrated diarrheic calves with metabolic acidosis. Isotonic sodium bicarbonate rapidly corrects metabolic acidosis and dehydration to restore normal cellular function. Once the suckle reflex is re-established, further treatment can be given orally. Correcting dehydration with rapid administration of small volumes of hypertonic saline solutions (4–5 mL/kg body weight, 7.2% NaCl, or 7.2% NaCl in 6% dextran-70) successfully resuscitates the dehydrated calf but does not correct metabolic acidosis. Therefore, administration of hypertonic saline solutions should be accompanied by intravenous sodium bicarbonate infusion in severely acidemic calves or by oral alkalinizing agents (acetate, propionate) in mildly to moderately acidemic calves. Dextrose should be added to the isotonic solutions if blood glucose is less than 60 mg/dL to counteract hypoglycemia and to provide additional energy.

TECHNIQUES FOR INTRAVENOUS CATHETERIZATION OF CALVES

Catheterization of the jugular vein is the most widely used approach for fluid therapy in calves, and the technique has been previously described in detail.[82, 102] Other routes for administration of fluids in cattle used abdominal[100] and coccygeal veins[97] or other parenteral routes, like subcutaneous,[27, 38, 99] intraabdominal,[55] and intrarectal administration. This section focuses on catheterization of auricular veins in calves, which has become increasingly popular in Europe.

Jugular catheterization may be difficult in severely dehydrated calves when the blood vessel is collapsed and not visible, even after a prolonged occlusion.[7, 35, 73] In addition, the tough and dry skin usually present in dehydrated calves can create problems with jugular catheterization.[63] To increase distention of the jugular vein, calves can be raised on their rear legs, thereby increasing blood flow toward the head.[7, 46, 73, 84]

Likewise, an incision of the skin with a scalpel or a punch with a large injection needle enables easier placement of intravenous cannules into the jugular vein.[7, 63, 73] Surgical cutdown of the jugular vein is needed in up to 40% of cases to achieve successful catheterization, particularly in severely dehydrated calves.[35, 103] This approach can be time consuming, increase costs, and may create catheter-related complications if not performed under strict aseptic conditions.[81] Additionally, to make the surgical procedure comfortable, it was recommended to sedate fractious calves for proper preparation and administer antibiotics to reduce the risk of thrombophlebitis.[35] To avoid initial catheterization of the jugular vein in collapsed calves, 0.5 to 1 L of fluids can be infused through a larger injection needle in the jugular vein. The simple injection of a needle is easier than placement of an intravenous catheter, and severely dehydrated calves usually are depressed and do not move during primary fluid administration. After initial resuscitation, the increased venous return makes the jugular vein more visible and it becomes easier to place a catheter.

Auricular Vein Catheterization in Calves

An alternative approach for jugular vein catheterization in adult cattle and calves is catheterization of an auricular vein.[28, 38, 48, 88, 92, 95, 100] Catheterization of the ear vein is currently recommended in Europe as the standard approach for adult cattle because it creates fewer complications than jugular vein catheterization.[41, 100] The initial reports over 40 years ago on fluid administration into auricular veins in calves and adult cattle used a steel needle with a fluid line that was taped securely to the auricular pinna.[38, 48] In 1983, Schmid and Rüsse described a technique of auricular vein catheterization in neonatal calves using a small flexible catheter, and over the last decade the method became popular amongst practitioners and teaching hospitals in Germany.[95] Ear vein catheters have also been used successfully in calves in other countries.[31, 88, 92] Even in severely dehydrated calves, ear vein catheterization is successful, thereby avoiding a surgical cutdown of the jugular vein. Ear catheters allow the application of sufficient amounts of fluids by continuous drip infusion to calves,[3, 28, 57, 58] and are believed to create fewer complications than jugular catheters.[38, 41, 100] A recent report evaluating the use of auricular vein catheters primarily in adult cattle stated that this technique was easy to perform, safe, and less expensive because fewer catheters have to be discarded.[92]

Veterinarians should practice the ear catheterization technique in dehydrated calves because it may require training to place a 22-gauge catheter into an ear vein. For better distention and visualisation of the ear veins, a sponge or gauze soaked with warm water (45°C = 113°F) should be applied for at least 1 minute to increase blood flow to the ear. A 22-gauge, 1-inch long (0.9 × 25 mm) catheter (Vasocan Braunüle, B. Braun, Melsungen, Germany) with a moveable butterfly-shaped wing at

the catheter hub is recommended for ear vein catheterization in calves.[28, 41, 57] In countries where this type of catheter is not available, other 22-gauge winged catheters can be substituted (e.g., Insyte-W, Becton-Dickinson, Sandy, UT). The catheters are sutured to the pinna through preformed holes in the butterfly-shaped wing using a simple injection needle and monofilament rigid suture material (Fig. 4). In catheters without plastic wings (e.g., Angiocath, Becton-Dickinson), a butterfly-shaped wing around the catheter hub can be made of a small strip of tape to permit suturing of the catheter to the ear. Otherwise, the catheter can be attached to the pinna with superglue, a roll of gauze placed inside the ear, and the catheter secured with bandage material.[92]

To avoid arterial puncture or placement of the catheter into the ear artery, the artery should be identified by feeling the pulse wave, which can be difficult to detect in small and severely dehydrated calves. The artery usually is straighter and more prominent and visible than a vein before applying a tourniquet around the base of the ear. Usually the ear artery in most calves runs across the pinna from medial to lateral on the border between the flat, more rigid part (cranial third) and oblique, less rigid part (caudal two thirds) of the pinna (see Fig. 4A). The ear artery palpates harder than a vein and can be rolled under the skin of the pinna with one finger.

Procedure for Auricular Vein Catheterization

If intravenous catheterization is often performed, a case or box with all materials needed for IV catheterization is helpful (see Fig. 4). This "IV-Box" should contain scissors, hemostats, scalpel blades, flashlight, tourniquets, clippers, single-use razors, chlorhexidine antimicrobial scrub, 70% ethyl-alcohol, gauze, catheters, iodine ointment, needles, syringes, local anesthetic, suture material, tape, superglue, bandages, gloves, heparinized saline (10–15 units heparin/ml 0.9% NaCl), and equipment needed to setup a fluid line (extension sets, injection caps, fluid lines, self-retractable dog leash, etc.).

Placement of an Ear Catheter

1. Prepare all materials needed and set up fluid line with (warmed) fluids first.
2. Use right ear if right handed and left ear if left handed (see Fig. 4A).
3. Place tourniquet around base of the ear to distend veins (the longer the better).
4. Clip or shave dorsal pinna carefully and prepare skin surgically (scrub with chlorhexidine solution for 1 minute, then swab with alcohol).
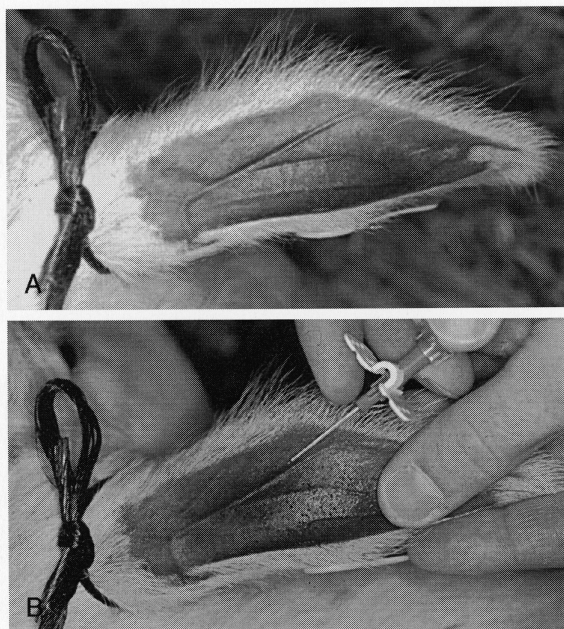5. Identify and select a suitable ear vein; a bifurcation (Y-shaped)

Figure 4. *A,* Cranial third of a right ear in a calf (viewed from the dorsal aspect) showing two ear veins forming a Y, and the lesser visible ear artery below. *B,* Catheter prior to insertion into a straight ear vein holding the catheter with the right hand and the ear with the left hand. Either a straight ear vein or a Y-shaped bifurcation of 2 veins is the preferred site for catheter placement.

of two veins is preferred if distention of a straight vein cannot be achieved (see Fig. 4*A*).

6. If veins are not properly distended, apply a sponge or gauze soaked with warm water (45°C = 113°F) for 1 minute.

7. Prepare 22-gauge, 1-inch catheter by gently moving the steel needle (stylette) of the catheter back and forth one time to allow easier advancement. Form a little butterfly around the hub of the catheter with tape if a butterfly catheter is not available.

8. Hold ear straight with one hand (see Fig. 4*B*) and advance needle tip of catheter into the distended vein at least 0.5 inches (1 cm) (see Fig. 4*C*). Expect the calf to move during this procedure and try to move with the calf.

9. Confirm correct catheter placement in the vein by watching for blood flow into the hub of the catheter before slightly withdrawing the steel needle (see Fig. 4*C*).

10. Advance catheter over the needle gently into the vein; remove the steel needle carefully and remove tourniquet.

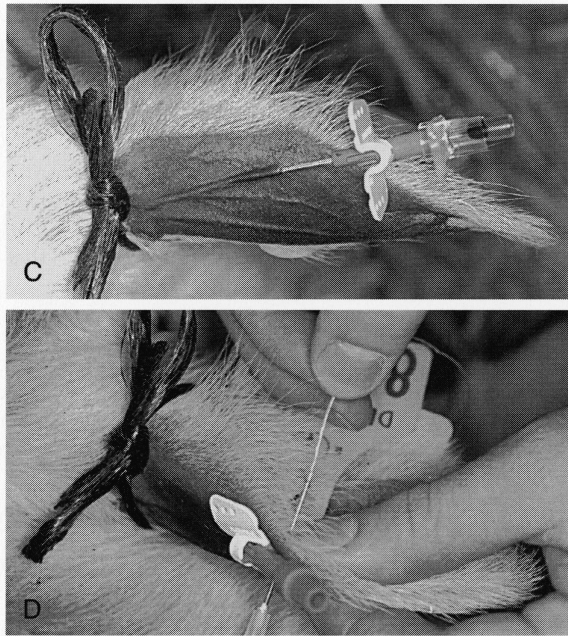11. Check correct catheter placement by examining for very slow

Figure 4 *(Continued)*. *C,* Catheter inserted into a straight ear vein approximately 0.5 inches (1 cm) with blood coming out of the stylet needle. At this point of insertion, the catheter can be fully advanced and the stylet removed. *D,* Suturing of the ear catheter to the auricular pinna using an injection needle and monofilament rigid suture material. Place two stitches on each side of the catheter forming a simple knot.

blood flow out of the catheter hub in severely dehydrated calves or place injection cap on catheter hub and flush catheter carefully.
12. Suture catheter to the ear by quickly sticking an injection needle (18 gauge; 1.2 mm) through the preformed holes or through the tape of the catheter wing and the pinna (expect the calf to move). Use a 10-inch (25 cm)-long suture and advance it from the tip of the needle to the basis. Remove needle *and* hold the catheter in place with other hand. Use a monofilament suture material and place two punctures on each side of the catheter to form a simple stitch on each side of the catheter (see Fig. 4*D*).
13. Remove injection cap, flush the catheter with heparinized saline, and connect the fluid line, preferably including an extension set.
14. Place a small dose of iodine ointment at the site of catheter insertion through the skin.
15. Tape and bandage the catheter with a loop of the fluid line (extension set) to the ear. Place a gauze roll adequate in size inside of the ear to allow better and more forceful taping. Do not tape or bandage in front of the catheter tip.

16. Use coiled tubing. If not available, place a self-retractable dog leash around the neck of the calf and tape infusion line to the leash, allowing free movements of the ear and the calf. Make sure that there is an inlet for air into the fluid infusion system.
17. Avoid stress to the calf. Usually calves with severe dehydration and acidosis are very depressed or comatose. These cases require almost no assistance from another person to place the catheter. If, however, calves are more responsive, it is the author's preference to apply the minimal restraint possible to the calf and try to place a catheter without assistance.

In summary, continuous intravenous fluid therapy in calves is most often accomplished by catheterization of the jugular vein; however, placement of a jugular catheter often creates difficulties if the vein is not distensible, requiring surgical cutdown for successul catheterization. Ear vein catheterization in calves has become increasingly popular in Europe. It is a convenient, safe, and cheap technique that can be used by personnel with limited experience, even in severely dehydrated calves.[92]

METHODS OF ADMINISTRATION

A large number of studies have used different recipes, calculation formulas, and rates of administration for fluid therapy in calves. These recommendations are sometimes too academic and complicated for an economical and practical approach, for which intravenous fluid therapy should be as simple and cheap as possible. Selection of an appropriate electrolyte or fluid therapy should be based on easily obtained clinical signs. Figure 3 shows a simple flow chart with guidelines for the treatment of dehydrated calves with diarrhea; however, for an appropriate determination of daily fluid requirements, estimated amounts for replacement, maintenance, and ongoing losses for diarrhea have to be calculated. The quantity of replacement fluid in liters can be calculated by multiplying the estimated dehydration in percentage with body weight in kilograms according to the following formula:

Replacement fluid [L] = dehydration [%] × body weight [kg].

The rate of fluid administered should not exceed 80 mL/kg/hour.[35, 51, 104] This means that a maximum fluid volume of 2.8 L for a 35 kg (77 lb) or 1 gallon (3.8 L) for a 47 kg (104 lb) for a severly dehydrated calf can be administered per hour; however, to avoid overhydration and pulmonary edema, a slower infusion speed of 30 to 40 mL/kg/hr is more frequently recommended and used. A recent study gave the first 1.0 L within 30 minutes and the subsequent dose of 3.0 L in the next 2.5 hours,[6] which is in agreement with a slower rate of 30 to 40 mL/kg/hr.[87] With an administration speed of 30 to 40 mL/kg/hr, a 10% dehydrated 40-kg calf can be rehydrated within 3 to 4 hours.[87] This can be accomplished through a 22-gauge ear catheter, which permits a flow rate

of approximately 30 mL/minute (1800 mL/hr) when fluids are placed at least 1 m above the calf's head.[26] In addition, daily maintenance fluid of 80 to 100 mL/kg[9] and ongoing losses of up to 7 L per day[22] have to be added to calculate the daily fluid requirements[73]; however, if the calf can suckle after initial resuscitation, the daily fluid requirements should preferably be given orally to reduce costs.

Measurements of buffer needs are based on formulas for extracellular base excess (from blood gas analysis) or plasma TCO_2 concentration. Amounts calculated from blood gas analysis multiply base deficit with body weight and with a factor for the volume of distribution for bicarbonate ions in the body of 0.5 according to the following formula[68, 87]:

$$\text{Bicarbonate needed [mEq]} = \text{body weight [kg]} \times \text{base deficit [mEq/L]} \times 0.5 \text{ [L/kg]}.$$

Another reference uses a higher distribution factor for bicarbonate of 1.0 for the calculation of the bicarbonate requirements in order to consider for the ongoing daily bicarbonate losses in diarrhea,[28] although this figure appears much too high to be used to correct existing deficits. Guidelines developed by Naylor[73] based on standing ability, suckling force, and age of diarrheic calves have been very useful for predicting if alkalinizing therapy is indicated and how much isotonic sodium bicarbonate should be administered; however, depressed calves without diarrhea and dehydration are often severely acidemic and therefore need intravenous buffer administration.[1, 5, 50, 52, 54, 93] Because base deficit or blood pH is normally not determined calf side, the quantity of buffer to be administered is initially estimated and reassessed on the basis of the clinical response of the calf to treatment with sodium bicarbonate. If calves do not respond to buffer therapy or their clinical condition continues to deteriorate, the presence of hypoglycemia or septicemia should be considered as differential diagnoses.[25, 54]

An easy and successful method for the treatment of severely dehydrated diarrheic and acidemic calves is to administer isotonic sodium bicarbonate solution at approximately 10% body weight over a period of 8 to 12 hours.[57] Without exact calculation of base deficit, fluid rate, or maintenance requirements, this study had a success rate of therapy of 91%.[57] A total of 90 calves with a mean base deficit of -19.0 ± 3.8 mEq/L (pH 7.06 ± 0.22) received approximately 10% of their body weight as an isotonic sodium bicarbonate solution (1.26% = 12.6 g/L instead of the more widely used 1.3%). The first half of the calculated dose of isotonic sodium bicarbonate solution was administered rapidly over 2 to 3 hours, which equals an administration rate of approximately 17 to 25 mL/kg/hr. When after 3 hours approximately 5% body weight of isotonic sodium bicarbonate had been infused, 77% of the calves were able to suckle again. In this study, milk replacer was continuously fed, and only water instead of electrolyte solutions was offered to the calves. Further intravenous replacement therapy with 2 to 4 L Ringer's or 0.9% sodium chloride after resuscitation with sodium bicarbonate was given to 16 of 90 calves.[57]

Earlier guidelines for the application of isotonic sodium bicarbonate distinguished between moderate and severe dehydration.[83] Moderately dehydrated calves with an estimated loss of 6% body weight received 50 mL/kg as hydration therapy in the first 4 to 6 hours and 140 mL/kg over the next 24 hours. For more severely dehydrated calves, the dose rate for hydration therapy was doubled to 100 mL/kg in 4 to 6 hours with the same maintenance therapy. A practical instruction for a 45-kg, severely dehydrated and comatose calf is to give 2 L of isotonic sodium bicarbonate rapidly and then change fluids to an isotonic mixture of sodium chloride and sodium bicarbonate. This mixture can be easily achieved mixing isotonic sodium bicarbonate and sodium chloride solutions together.[83] A slightly hypertonic solution of 5% dextrose added to 12.6 g sodium bicarbonate can also be used.[85]

The fluids administered should be warmed if a calf is hypothermic because cold fluids increase energy consumption as they are warmed by the body.[88] This may further decrease cardiac output and even kill a critically ill calf, especially if a rapid infusion rate of 80 mL/kg/hr is used.[73, 107] Warmed fluids cool out very fast with a slow continuous drip administration. In this case the tubing can be looped around a heat lamp or through a bucket with hot water to warm the fluids. Additionally, the calves can be warmed with heat lamps or pads to minimize their energy demand for correcting hypothermia.

Close monitoring of fluid therapy in practice is not possible, and continued treatment should be based on improvement of clinical signs.[35] Besides an improved mental state, criteria for response to treatment are decrease of enophthalmos, returning of suckle reflex, wet mucous membranes, increase of rectal temperature, and urination within 30 to 60 minutes.[35, 84, 88] Recumbent calves should stand within 6 to 24 hours of fluid therapy.[85]

In the past it was recommended to correct the whole amount of calculated dehydration plus maintenance plus substitute for the ongoing daily losses and to rehydrate the patient over a time of 24 to 48 hours. This deficit therapy approach may sometimes be too complicated to calculate and undertake in routine practice. It is therefore recommended to infuse only part of the total fluid requirement intravenously to resuscitate the calf and restore circulation. During a rapid infusion of the first liter of solution the calf can usually be monitored by the veterinarian and the flow rate can be adjusted.[89] In most cases, 1 to 3 L given over 2 to 3 hours partially restore fluid deficits and restore the calf's suckle reflex for further oral rehydration and buffer therapy.[57] If the suckle reflex does not return, one should look for concurrent diseases, such as hypoglycemia, septicemia, omphalitis, or pneumonia.[85]

COMPLICATIONS

Catheter-Related Complications

Reports on complications of intravenous catheterization focus on the problems associated with jugular vein catheterization in cattle.[2, 19, 38,]

[81, 82] Venipuncture damages the wall of the blood vessel and surrounding tissues followed by bacterial contamination of the vessel intima.[82] Common noninfectious failures of intravenous catheterization include phlebitis and perivenous extravasation of fluids or blood with infiltration of surrounding tissue forming hematoma or subcutaneous edema.[41] If the catheter is not correctly placed and securely sutured or lies too short in the vein, movements of the animal may cause paravenous or total displacement of the catheter.[40] If the veins do not occlude from thrombosis, these complications usually heal without further problems.[41] Perivenous infusion of larger amounts of fluids normally creates no serious problems in calves if isotonic solutions are used. Signs of perivenous infusion are slower infusion rate and swelling around the injection site.[40] Further catheter-related complications start with thrombosis, mostly at the catheter insertion site, and local infectious phlebitis as the most frequent complications in adult cattle.[82] This may be followed by embolism and infection of the thrombus, causing thrombophlebitis, periphlebitis, and finally septicemia.[15, 19, 41, 82] Bacterial contamination of jugular catheters occurs mostly from the microflora of the skin forming thrombi at the catheter insertion site.[82] Also, contamination of the catheter during inproper handling prior to insertion and contaminated solutions or infusion tubing results in thrombophlebitis.[2, 15] Clinical findings of infectious thrombophlebitis included a visible, painful, and warm swelling of the jugular vein, increased rectal temperature, reduced feed intake, and abnormal condition and behavior of cows.[81] Jugular thrombophlebitis in adult cattle occurred after catheterization for 72 hours, even after surgical preparation of the skin with an iodine-based antiseptic.[81] If low-dose heparin (120 U/kg q 12 hr) was administered subcutaneously in addition to surgical skin preparation, thrombophlebitis could be avoided during catheterization for 5 days.[82] Jugular catheters in large animals should be removed after 48 hours, according to an older publication, in order to avoid thrombophlebitis.[19]

Auricular ear vein catheterization is believed to result in fewer problems than jugular vein catheterization.[38, 41, 92] To the author's knowledge, a comparative evaluation of ear vein catheterization and other techniques is not available for calves; however, in pigs, ear vein catheterization for 7 to 14 days proved superior to jugular vein catheterization.[60] Jugular vein catheters were more often occluded and produced thrombosis and phlebitis, whereas ear vein catheters showed no reactions.[60] Arterial puncture is a common complication of jugular catheterization with the formation of larger and fast-growing hematomas. With ear vein catheterization, cannulation of an ear artery can be avoided by identifying this vessel as described previously. Duration of ear vein catheterization can be extended for several days without significant complications.[38, 41] A recent study reported no serious complications with ear vein catheterization up to 96 hours.[92] Changing the ear catheter is therefore not needed as frequently as with jugular catheters, but they tend to occlude more frequently than jugular catheters.[92] There are no recommendations when to pull an ear catheter in calves, but in my personal

experience with ear catheters in place for up to 7 days, I noticed only minor swellings and no signs of thrombophlebitis or other serious complications.

In addition, insufficient flow rate, which is the major point of critique with ear catheters in adult cattle, is rarely a problem in calves. In vitro flow rates for 22-gauge catheters range between 28 mL/min (Insyte-W, Becton-Dickinson) and 36 mL/min (Vasocan Braunüle, B. Braun) allowing in vivo flow rates of approximately 1.2 to 2.2 L per hour.[26] Other problems are encountered when attempting to maintain an intravenous catheter for on-farm fluid therapy for several hours or days. Because of lesser observation, the fluids may run out, followed by clotting of the catheter.[54, 86] If the calf starts to move around freely in its stall, the infusion line can be coiled and is often occluded after bending.[63, 86, 104] To reduce the frequency of these complications, either coiled infusion sets or self-retractable dog leashes to which the infusion line is taped should be used.

Overhydration or overinfusion at too rapid an administration rate increases intravascular pressure to an extent that pulmonary edema may develop.[86] Signs of overhydration and pulmonary edema associated with intravenous fluid therapy are nasal discharge, tachypnea, tachycardia, coughing, and wet lung sounds (crackles).[20, 41] Central venous pressure over 12 cm of water, measured at the level of the scapulohumeral joint, indicates that the vascular pressure is too high, and infusion rate has to be reduced or stopped temporarily.[51] With an administration rate of 80 mL/kg/hr, increased central venous pressure was observed in almost one third of the dehydrated calves, but no clinical signs of pulmonary edema were detected.[51] A slower infusion rate than 80 mL/kg/hr should be prefered in practice because body weight often is unknown. This would avoid overhydration and pulmonary and cerebral edema. Anemia and hypoproteinemia may also follow overhydration, leading to hypoxia if the hematocrit falls below 15%. Formation of interstitial edema will occur if total protein concentration is less than 4.0 mg/dL.[41] To the author's knowledge, these complications are not commonly reported for calves.

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