Treating scouring beef and dairy calves

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Abstract

Diarrhea is the leading cause of calf mortality prior to weaning in both beef and dairy calves. Therefore, both veterinarians and producers should put some effort into designing rational and efficacious protocols for both prevention and treatment of diarrhea. The primary goals of treating calf diarrhea are to 1) correct free water and electrolyte abnormalities; 2) correct acid-base deficits (acidemia); 3) provide nutritional support; and 4) eliminate and/or prevent *E. coli* bacteremia. This presentation will focus on both oral electrolyte and intravenous fluid therapy in calves with diarrhea. Practical approaches to correcting dehydration and metabolic acidosis on-farm will be emphasized.

Key words: bovine, neonatal, diarrhea, fluid therapy

Résumé

La diarrhée demeure la principale cause de mortalité avant sevrage à la fois chez les veaux de boucherie et chez les veaux laitiers. Par conséquent, les vétérinaire et les producteurs devraient faire des efforts pour planifier des protocoles rationnels et efficaces afin de prévenir et traiter la diarrhée. Les buts principaux du traitement de la diarrhée des veaux sont les suivants : 1) rectifier les anomalies électrolytiques et de l'eau libre, 2) rectifier le déséquilibre acide/base (acidémie), 3) apporter un soutien nutritionnel, et 4) éliminer ou prévenir la bactériémie causée par *Escherichia coli*. Cette présentation se portera sur la thérapie de réhydratation orale à base d'électrolytes et la thérapie par voie intraveineuse chez les veaux diarrhéiques. Des approches pratiques pour rectifier la déshydratation et l'acidose métabolique à la ferme seront mises en évidence.

Introduction

Neonatal diarrhea remains the most common cause of death in both beef and dairy calves. Despite significant progress in understanding the pathophysiology of neonatal diarrhea, it continues to be a major cause of economic loss to the cattle industry. A complete review of the pathophysiology of diarrhea is beyond the scope of this chapter, and has recently been covered elsewhere.[11] Some pathogens cause secretory diarrhea, causing the small intestine to move from a net absorption of fluid to a net secretion of chloride, sodium, and water into the intestinal lumen. This increase in secretion overwhelms the absorptive capacity of the large intestine, resulting in diarrhea. Other pathogens damage the small intestinal villi, which results in failure to absorb electrolytes and water (malabsorptive diarrhea). However, regardless of the pathogen or the mechanism involved, diarrhea increases the loss of electrolytes and water in the feces of calves and often decreases milk intake. This results in dehydration, strong ion acidosis, electrolyte abnormalities (usually decreased sodium and increased **OR** decreased potassium), increased D-lactate concentrations, and a negative energy balance (from anorexia and malabsorption of nutrients). Therefore, diarrhea is by far the most common indication for fluid therapy in neonatal calves. The primary goals of treating calf diarrhea are to 1) correct free water and electrolyte abnormalities; 2) correct acid-base deficits (acidemia); 3) provide nutritional support; and 4) eliminate and/or prevent *E. coli* bacteremia. Three of these 4 goals can be met with fluid therapy. The purpose of this article is to provide an overview of fluid therapy in calves with particular emphasis on treating diarrhea. Practical options for fluid therapy that can be performed on the farm will be emphasized.

Oral Electrolyte Therapy

According to the World Health Organization (WHO), the development of oral rehydration therapy was one of the most significant advances in human medicine of the 20th century. Oral electrolyte solutions also continue to serve as the backbone of treatment protocols for diarrhea in neonatal calves because they are cheap and easy to administer on-farm. Oral electrolyte solutions (OES) are indicated in any diarrheic calf that has at least a partially functional gastrointestinal tract. If OES are administered to a calf with ileus, the fluid pools in the forestomach, resulting in bloat and rumen acidosis. In general, a calf with any sort of suckle reflex, or that demonstrates any "chewing" action, can be considered to safely tolerate oral fluids.

Oral electrolyte therapy in calves was thoroughly reviewed in previous manuscripts,[21,22,24] but a brief overview will be given here. Oral electrolyte solutions were originally developed in human medicine for treatment of diarrhea associated with cholera infection, with the original WHO electrolyte formulation based on the following main principles:

1. It was an isotonic solution which contained an approximately equimolar mixture of sodium (90 mM/L) and glucose (2%).
2. It contained potassium because of the severe potassium depletion associated with diarrhea and anorexia.

Table 1. A comparison of oral electrolyte products available in North America.*

	Sodium (mM/L)	Potassium (mM/L)	Chloride (mM/L)	Strong ion difference	Alkalinzing agent	Total osmolality (mOsm/L)
Advance Arrest (Milk Specialties)[†]	46	7	30	23	bicarbonate (12 mM/L)	245
Bounce Back (Manna Pro)[†]	136	10	112	34	bicarbonate (48 mM/L)	
Bovi-Mate ORS (Drench-Mate)	130	20	60	90	acetate (60 mM/L) and propionate (30 mM/L)	600
Bovi-Mate Elyte (Drench-Mate)	124	22	60	87	citrate (30 mM/L)	450
Bluelite Replenish (Techmix)	128	28	80	68	acetate (68 mM/L) and propionate (21 mM/L)	440
Blue Ribbon Calf Electrolytes (Merrick)[†]	144	20	75	89	none	390
Bovine Bluelite C (Techmix)	59	24	56	27	none	269
Calf-Lyte II (Vetquinol)	112	15	43	84	acetate (80 mM/L)	428
Calf-Lyte II HE (Vetquinol)	112	15	43	84	acetate (80 mM/L)	726
Calf Restart One-4 (Techmix)	54	12	70	0	none	
Calf Quencher (Vedco)	142	24	80	86	bicarbonate (86 mM/L)	731
Deliver (Agri-Labs)[†]	67	16	49	34	bicarbonate (36 mM/L)	305
Diaque (Boehringer Ingelheim)	90	15	55	50	bicarbonate (25 mM/L) and acetate (12 mM/L)	377
Electrolyte-F-Calf (Bio Agri Mix, Canada)	92	15	43	64	acetate (60 mM/L)	385
Electrolife Renew (MB Nutritional Sciences)	100	20	50	70	acetate (60 mM/L)	350
Entrolyte HE (Zoetis)	106	26	51	81	bicarbonate (80 mM/L)	739
Epic calf electrolyte (Bioniche)	92	30	45	77	acetate (52 mM/L)	360
Hydrafeed (A&L Laboratories)	110	10	40	80	bicarbonate (80 mM/L)	380
Hydralyte (Vet-A-Mix & AgriLabs)	90	30	45	75	acetate (60 mM/L)	614
Land O Lakes Base + Add Pack (Land O Lakes)	119	23	56	86	acetate (51 mM/L and citrate (10 mM/L)	455
Land O Lakes Complete (Land O Lakes)	121	20	78	64	bicarbonate (38 mM/L)	490
Resorb (Zoetis)	75	25	80	20	none	315
Revibe HE (Zoetis)	120	20	50	90	acetate (80 mM/L)	466
Revitilyte (Vets Plus, Inc.)	110	20	50	80	bicarbonate (80 mM/L)	459
Revitilyte Gelling (Vets Plus, Inc.)	110	20	50	80	bicarbonate (80 mM/L)	417
Sav-A-Caf Electrolytes Plus (Milk Products, LLC)	138	12	123	27	bicarbonate (25 mM/L)	547
TheraCaf Plus (Milk Products, LLC)	128	27	70	80	bicarbonate (44 mM/L) and acetate (6 mM/L)	472
VitaLyte Plus (Vita Plus Corp.)	150	31	45	136	bicarbonate (80 mM/L)	527

*This listing does not include every product available in North America. No discrimination or specific endorsement of any product is intended.
[†]Signifies data was calculated from product label instead of provided by the manufacturer. In some cases there was insufficient information on the label to provide an exact calculation so values may be exact.

3. It contained glycine to facilitate absorption of sodium, glucose, and water.
4. It contained bicarbonate to correct the metabolic acidosis associated with diarrhea.

Although much research has been done on oral fluid therapy since that time, surprisingly we have not moved very far from the original principles of the 1960's.

Considerable variability exists in the quality of commercial oral electrolyte solutions available today (see comparison table of oral electrolytes in reference 4) and the practitioner must put some thought into the product they choose to use in practice. Simply recommending oral electrolyte rehydration in this decade is as imprecise as advocating "antibiotics" would be without considering the drug or condition being treated. There are several important factors to consider when deciding on a product. Current knowledge indicates that an OES must satisfy the following 4 requirements: 1) supply sufficient sodium to normalize the extracellular fluid volume; 2) provide agents (glucose, citrate, acetate, propionate, or glycine) that facilitate absorption of sodium and water from the intestine; 3) provide an alkalinizing agent (acetate, propionate, or bicarbonate) to correct the acidosis usually present in calves with diarrhea; and 4) provide energy, as most calves with diarrhea are in a state of negative energy balance. Factors to consider when choosing an oral electrolyte solution include the following.

1. Sodium concentration – Sodium is the osmotic skeleton of the extracellular fluid, and therefore of plasma. Because sodium is the principal determinant of the volume of the ECF volume, it must be present in an oral electrolyte solution to rapidly correct the losses that have occurred with dehydration and diarrhea. The ideal sodium concentration for oral rehydration therapy in calves is not completely known, however most research would suggest it should be between 90 and 130 mmol/L. Products containing sodium at significantly lower concentrations are not able to adequately correct dehydration. Oral electrolyte products with very high sodium concentrations might be expected to cause hypernatremia, and have also been shown to delay abomasal emptying rates because of increased osmolality.

2. Chloride concentration – Although calves lose chloride during diarrhea, this loss does not occur nearly to the same degree as sodium.[6] A general guideline has been that oral electrolyte products should contain chloride in concentrations between 40 and 80 mmol/L.

3. Potassium concentration – Like sodium and chloride, potassium is lost in the feces of calves with diarrhea. Therefore all calves with diarrhea have a total body deficit of potassium. However, in acute cases of diarrhea calves may have elevated blood potassium concentrations (hyperkalemia).[26] With dehydration, aldosterone is released from the pituitary gland. Aldosterone acts on the kidney to conserve sodium and water at the expense of increased potassium losses. Therefore in chronic cases of diarrhea, calves can have profound depletion of body potassium stores and generally have low serum concentrations of potassium. Clinical signs of hypokalemia include profound muscular weakness, which is often present in calves with chronic diarrhea. General recommendations are that oral electrolyte products used in calves with diarrhea contain potassium concentrations between 10 and 30 mM/L.

4. Sodium absorption – Sodium absorption by the small intestine is a passive process, and is linked to the movement of actively absorbed or secreted solutes. If sodium is present in the lumen of the small intestine without either glucose or amino acid, there is either a small net absorption or no net sodium movement across the jejunum. One of the earliest mechanisms of intestinal sodium absorption discovered was linked with sugar.[10] Glucose can be co-transported with sodium from the intestinal lumen to the inside of the enterocyte at the brush border membrane. Because this mechanism was well understood by the 1960's, almost all early oral electrolyte formulations were mixtures of sodium and glucose. Neutral amino acids such as glycine, alanine or glutamine can also facilitate sodium absorption in the small intestine by a mechanism similar to glucose. In addition, volatile fatty acids, such as acetate or propionate, have also been shown to facilitate sodium absorption in the gut.[24]

5. Osmolality – Commercially available oral electrolyte products in North America can range from roughly isotonic (280 to 300 mOsm/L) to extremely hypertonic (700 to 800 mOsm/L). The primary difference in most of these products is the amount of glucose that is added. High osmolality solutions provide greater nutritional support to calves relative to lower osmolality products; however, milk or milk replacer is better able to maintain normal serum glucose concentration than either hypertonic or isotonic oral electrolyte solutions.[7] However, as expected, oral electrolyte solutions rehydrated calves and prevented the development of metabolic acidosis more effectively than did milk replacer because they have a much higher sodium concentration. Multiple studies have demonstrated that high-osmolality oral electrolyte solutions maintain higher serum glucose and lower β-OH butyrate (ketone) concentrations when compared to lower-osmolality electrolyte solutions.[7] The downsides of high osmolality is 1) it could worsen diarrhea and 2) it will slow abomasal emptying. The first downside was what led most OES designed for children with diarrhea to be formulated with a lower osmolality. Most calves with enteric pathogens already have hypersecretion of electrolytes and water into the small intestinal lumen, which could be exacerbated with the feeding of hypertonic solutions (electrolyte or milk replacer). Raising the intraluminal tonicity would serve to increase the secretion of water and electrolytes into the intestinal lumen, thus increasing the severity of diarrhea. This effect would likely be magnified with severe villus damage, which is often present in diarrheic calves.

Oral electrolyte solutions with extremely high osmolalities have also been shown to slow abomasal emptying rates as compared to isotonic products.[18,20] This suggests that electrolyte products with a very high osmolality (or high glucose concentrations) would be likely to induce abomasal ileus, thus increasing the risk of bloat and/or abomasitis.[4] So although the ideal osmolality of an oral electrolyte solution for calves is not completely understood, a moderate osmolality solution (400 to 500 mOsm/L) would be ideal in dairy calves or in beef calves that have been separated from the dam. Certainly if milk is to be withheld for any length of time, a hypertonic oral electrolyte solution would be indicated to provide energy to the calf. However, lower osmolality solutions might be appropriate for calves that were still suckling or drinking milk. Another consideration is that OES frequently get added to milk or milk replacer for feeding, which further increases osmolality. My recommendation is to avoid OES with extremely high osmolality products. In general I prefer to get the calf's nutrition (glucose) from milk and use OES for improving dehydration, correcting acidosis, and replacing electrolytes.

6. Alkalinizing ability – As discussed above, acidemia and metabolic/strong ion acidosis occur in almost all cases of calf diarrhea. Research has demonstrated that acidotic calves are unable to correct their metabolic acidosis when rehydrated with non-alkalinizing solutions (i.e., milk or OES without alkalinizing agents). Therefore it is imperative that OES to be used in calves with diarrhea be able to increase blood pH. Classically this has been done by adding alkalinizing agents

(i.e., bicarbonate, acetate, or propionate) to oral electrolyte mixtures. More recently, there has been growing interest in looking at the strong ion difference (SID) of electrolytes as they relate to the efficacy of a different product to promote alkalinization. In reality, both (having an alkalinizing agent and a high SID) are likely important. Acetate, propionate, bicarbonate, and citrate are all considered alkalinizing agents and are frequently present in commercial oral electrolyte solutions. Bicarbonate-containing fluids are very effective at correcting a severe acidosis, since bicarbonate reacts directly with H^+ ions to form CO_2 and H_2O. Acetate and propionate are also alkalinizing agents, and have been shown to have alkalinizing effects similar to bicarbonate. Acetate and propionate are only effective alkalinizing agents when they are metabolized; a process which forms water and creates bicarbonate ions (bicarbonate precursors). This metabolic process appears to still function efficiently in calves with severe diarrhea, as the alkalinizing ability of the acetate has been shown to be as effective as bicarbonate.[1] Acetate and propionate have several advantages over bicarbonate:

1. As discussed above, acetate and propionate facilitate sodium and water absorption in the calf intestine and colon, whereas bicarbonate does not.
2. Acetate and propionate produce energy when metabolized, whereas bicarbonate does not.
3. Acetate and propionate do not alkalinize the abomasum, whereas bicarbonate does.[23]

Abomasal acidity provides a natural barrier to ingested bacteria, and maintaining a low abomasal pH will decrease the number of viable coliform bacteria that reach the small intestine. This increases nonspecific resistance to intestinal colonization. Therefore, the increase in abomasal pH seen with electrolyte products that contain high concentrations of bicarbonate may facilitate growth of bacterial diarrheal pathogens and thus increase the severity, duration, and mortality rate associated with diarrhea in calves.

Strong ion theory is a different approach to looking at acid-base abnormalities and is covered in detail elsewhere.[5,14] Based on strong ion theory, it is not necessarily imperative that an electrolyte solution contain an alkalinizing agent to correct metabolic acidosis; rather, the product must deliver an excess of strong cations (Na^+) relative to the concentration of strong anions (Cl^-). Therefore, it has been advocated to consider the strong ion difference (SID) of an oral electrolyte solution when choosing a product. This can be calculated as follows: $[Na^+] + [K^+] – [Cl^-]$ = SID. Although there has not been any definite research to determine the optimal or minimum SID that an oral electrolyte product should contain, a minimum SID of 50 to 80 mEq/L would be recommended in a calf with diarrhea. Recent studies in calves demonstrate SID is a valid approach when formulating oral electrolytes for use in calves with diarrhea and acidosis.[19,25] Ultimately, the ideal electrolyte solution for use in calves with diarrhea should contain at least 50 mM/L of an alkalinizing agent (preferably acetate and/or propionate), and have a SID of at least 50 to 80.

Unfortunately, products without alkalinizing agents and with very low SIDs are commonly available in North America, and should be avoided in calves with diarrhea (Table 1).

Intravenous Fluid Therapy

Assessing the need for IV fluid therapy

The key for bovine practitioners is to be able to decide if IV fluid therapy is necessary in sick calves based on clinical examination rather than on laboratory values. Important clinical parameters to guide decision making on fluid therapy are obtained from the evaluation of hydration status and central-nervous-system (CNS) function. Degree of enophthalmus is the best predictor of dehydration in calves, followed by skin elasticity determined on the neck and thorax.[8,22] In clinically sick calves, it is important to evaluate hydration status along with other clinical signs. These include the ability of the calf to suckle, severity of CNS depression, and whether or not the calf can stand (degree of weakness). These factors in combination are used to determine whether or not IV fluid therapy is indicated.

Blood-gas and acid-base status are ideally determined with a portable blood gas analyzer such as the I-Stat System.[a] However, these laboratory analyzers are expensive and are therefore not used in most practices. Assessment and diagnosis of acidosis on the basis of clinical signs is quite common in bovine practice. The predictive accuracy of the degree of acidosis on the basis of clinical signs has varied between studies. The clinical signs of neurological depression (weakness, ataxia, and decreased menace, suckle and panniculus reflex) are highly correlated with the severity of metabolic acidosis in calves without dehydration. Also in diarrheic calves, signs of CNS depression, ability to stand, and suckling force, all correlated well with metabolic acidosis.[16,17] The degree of enophthalmos and peripheral skin temperature are important, and are obvious signs that determine whether or not IV fluid therapy is indicated, however they do not correlate with the degree of acidosis.

A very important discovery was that metabolic acidosis in diarrheic calves varies during the first weeks of life. Naylor discovered that metabolic acidosis is less severe during the first week of life than in diarrheic calves older than 8 days.[16,17] The base deficit in diarrheic calves older than 1 week was almost twice as high as in calves presented with diarrhea during the first week of life. Subsequent studies confirmed that calves with diarrhea older than 1 week of age usually exhibit a higher base deficit.[12] On the basis of his findings, "depression scoring" charts were developed for predicting the severity of metabolic acidosis based on body position, strength of suckle reflex, and age of the calf, with corresponding values for base deficit and bicarbonate requirements for the treatment of metabolic acidosis in diarrheic calves under or over 8 days of age. These protocols became a popular approach to guide diagnosis and treatment of acidosis in calves with diarrhea, and are presented in common veterinary medical textbooks.

More recent studies have linked the "depression scoring" in calves with elevated D-lactate concentrations instead of the severity of acidosis.[15,27]

In summary, the age of the calf needs to be taken into consideration when assessing the severity of acidosis and determining bicarbonate requirements of diarrheic calves. Calves with diarrhea and dehydration during their first week of life are less acidotic than older calves, and will require less sodium bicarbonate to correct their acidemia. Calves that are unable to stand or have a weak or absent suckle reflex have a more severe acidosis and require intravenous sodium bicarbonate to correct their acidemia. D-lactic acidosis may be present in sick calves with or without diarrhea and dehydration that are recumbent or wobbly, tired, listless or comatose, and with a delayed, incomplete, or absent palpebral reflex.[27] Also if the suckle reflex is absent, weak, or the calf is chewing irregularly instead of suckling normal, D-lactic acidosis may be the underlying disease state.

Solutions for intravenous administration

Thorough reviews of fluid therapy in ruminants and options for fluid therapy in calves with diarrhea have been published previously.[2,24] This section will give a brief overview of intravenous fluid therapy in calves and focus primarily on more recent advances in the treatment of diarrhea. Because acidemia is very common in calves with diarrhea, generally these animals require an alkalinizing fluid type to increase their blood pH. These can include:

1. Lactated Ringer's (LRS) – A traditional isotonic fluid that is sometimes used to correct dehydration and electrolyte abnormalities in neonatal ruminants. Lactate is a metabolizable base and therefore LRS is considered an alkalinizing fluid (that can increase blood pH). However, since the lactate must be metabolized to produce an alkalinizing effect, this fluid type is considered to have weak or slow alkalinizing ability and is not recommended for neonates with severe acidemia. Although LRS can be successfully used to treat dehydration and electrolyte abnormalities in neonates, it is difficult and expensive to administer in the field requiring intravenous catheterization, delivery equipment, animal restraint, large fluid volumes (3 to 5 liters in a calf, depending on size and degree of dehydration), and monitoring. A theoretical disadvantage of commercially available LRS is that the lactate is a racemic equimolar mixture of L-lactate and D-lactate, and its use should be avoided in severely acidemic calves since D-lactate concentrations may already be elevated.[13,15]

2. Acetated Ringer's – Similar to LRS but contains acetate instead of lactate as the metabolizable base. Would also be considered a weak or slow alkalinizing fluid type, and would have to be given in large volumes to correct dehydration in diarrheic calves. Acetated Ringer's solution is theoretically superior to lactated Ringer's solution because acetate is metabolized faster, therefore alkalinization is more rapid. In addition, acetate would not exacerbate the D-lactic acidosis present in the majority of calves with diarrhea.

3. Isotonic sodium bicarbonate – Sodium bicarbonate is often referred to as a "strong" alkalinizing fluid since bicarbonate does not have to be metabolized by the liver to have an alkalinizing effect on the blood. Sodium bicarbonate has proven to be more effective than other metabolizable bases (such as lactate or acetate), bicarbonate precursors, or synthetic bases. Isotonic sodium bicarbonate is often given as a 1.3% solution and can be easily prepared by adding baking soda ($NaHCO_3^-$) to sterile (or distilled) water at 13 grams per liter (155 mEq/L HCO_3^-) and administered via an intravenous catheter. Isotonic sodium bicarbonate has an effective SID of 155 mEq/L and is alkalinizing because it buffers hydrogen ions and increases the SID in blood. The amount of isotonic bicarbonate required to correct an acidemia is usually calculated based on either blood total CO_2 or bicarbonate concentrations, or base excess values, but usually ranges between 2 and 5 liters depending on the calf's weight and severity of acidosis (which can be estimated using depression scores). Base excess values calculated from blood gas analysis or estimated from depression scoring charts are multiplied by body weight, and with a factor that considers the volume of distribution for bicarbonate ions in the body (generally 0.6) according to the following formula: bicarbonate requirement [mEq] = body weight [kg] x base deficit [mEq/L] x 0.6 [L/kg]. Another simple but successful rule of thumb is to administer isotonic sodium bicarbonate solution at approximately 10% body weight over a period of several hours (i.e., 4 liters to a 88 lb (40 kg) calf). The disadvantage of isotonic sodium bicarbonate is that it still requires an intravenous catheter and the administration of a relatively large volume of fluids, which can sometimes be difficult to accomplish under field conditions.

4. Hypertonic saline – Over the past 10 years we have discovered that hypertonic saline (2400 mOsm/L) can be used to rapidly expand plasma volume in a severely dehydrated calf.[2] When combined with oral electrolyte solutions, this therapy can be as effective in resuscitating severely dehydrated calves as large-volume lactated Ringer's administration, and is less expensive and much easier to administer. Hypertonic saline solutions can be purchased commercially in 1000 ml containers and should be given at a rate of 4 to 5 ml/kg, administered slowly over a 4-minute period. Keep in mind, however, that hypertonic saline does not correct an acidemia.

5. Hypertonic sodium bicarbonate – In recent years, the use of hypertonic sodium bicarbonate (HSB) combined with oral electrolytes has gained popularity for the correction of acidosis and dehydration in neonatal ruminants with diarrhea. Generally, HSB is commercially available as an 8.4% solution which contains sodium bicarbonate at 1 mEq/mL of solution. The total osmolality is approximately 2000 mOsm/L. Therefore, the product should theoretically generate an osmotic movement of water and electrolytes from the gastrointestinal tract to the extracellular fluid space similar to hypertonic saline. However, an added benefit would be that it is an alkalinizing fluid and should significantly in-

crease blood pH at the same time. Although there are some theoretical disadvantages to HSB administration, a study in anesthetized calves demonstrated that rapid administration of HSB was safe when administered to anesthetized calves.[3] It was effective in reversing an experimentally-induced acidemia and did not cause cerebrospinal fluid pH to decrease (paradoxical CSF acidosis) as has long been hypothesized. In a German study, 28 calves with naturally occurring diarrhea were divided into 2 groups.[20] One group received hypertonic saline (2.27 mL/lb [5 ml/kg] of body weight over 4 minutes) and the other group received hypertonic (8.4%) sodium bicarbonate (4.55 mL/lb [10 ml/kg] of body weight over 10 minutes). Intravenous fluids were followed by 3 liters of an isotonic oral electrolyte solution. During the 72-hour period following treatment, more calves recovered that had received HSB as compared to hypertonic saline (many calves that received only hypertonic saline required additional fluid therapy). Another study done in Turkey involved 50 calves with diarrhea, dehydration, and strong ion acidosis.[9] Thirty calves in this study received isotonic sodium bicarbonate (30 mL/lb [65 ml/kg] of body weight over 3 hours) while 20 calves received 8.4% HSB (10 ml/kg of body weight over 20 minutes). Although isotonic sodium bicarbonate was able to increase plasma volume to a greater extent than HSB, both fluid types were effective in rapidly increasing venous pH and resuscitating calves. Although more studies are needed to determine the proper dose of HSB for use in calves, this can be an effective fluid type for correcting metabolic acidosis. It can be used in calves with diarrhea when an intravenous catheter or long-term fluid therapy is not practical.

6. Dextrose – This is the only non-alkalinizing fluid type that is generally used in neonatal ruminants. Dextrose is often added to other solutions at 5 to 10% to counteract the negative energy balance in diarrheic calves with or without hypoglycemia. However, in dehydrated calves a plain 5% dextrose solution is not sufficient to correct extracellular fluid deficits because the solution contains no sodium. To provide energy and rehydrate the neonate, 25 to 50 grams of dextrose or 50 to 100 ml of 50% dextrose solution can be added per liter of LRS or isotonic sodium bicarbonate to make a mildly hypertonic solution.

In summary, alkalinizing fluids are the appropriate choice for the intravenous rehydration of calves with diarrhea and dehydration. At present, sodium bicarbonate is the recommended solution for intravenous treatment for diarrheic calves that have lost their suckle reflex, palpebral reflex and are unwilling to stand. Sodium bicarbonate (either hypertonic or isotonic) rapidly corrects both acidosis and dehydration, and will restore normal cellular function. When the calf's suckle reflex is reestablished, further treatment can be given orally.

Administration of intravenous fluids

Many studies have presented various protocols for IV fluid therapy in calves; however, clinical research comparing the effectiveness of different protocols in dehydrated calves with diarrhea is limited. In practice, fluid therapy has to be simple and cost-effective, and must be based on clinical signs that are easily assessed. To determine daily fluid requirements, estimated amounts for replacement, maintenance, and ongoing losses (for diarrhea) must be calculated. The quantity of replacement fluid in liters is calculated by multiplying the estimated dehydration in % with body weight in kg according to the following formula: replacement fluid [L] = dehydration [%] x body weight [kg].

A maximum rate of 80 ml/kg/hour for IV fluid administration has been used without inducing significant over-hydration and hypertension. This rate is equivalent to a maximum fluid volume of 2.8 L per hour for a 77 lb (35 kg) calf, or 1 gallon (3.8 L) per hour for a 104 lb (47 kg) severely dehydrated calf. Higher flow rates are not recommended. Slower infusion rates of 14 to 23 mL/lb (30 to 50 ml/kg)/hour are often used to avoid over-hydration and pulmonary edema. With a rate of 14 to 18 mL/lb (30 to 40 ml/kg)/hour, an 88 lb (40 kg) calf with 10% dehydration can be rehydrated within 3 to 4 hours. In addition, daily maintenance fluid volumes of 37 to 45 mL/lb (80 to 100 ml/kg) and ongoing losses of up to 7 liters per day should be added to calculate the daily fluid requirements. However, if the calf can suckle after initial resuscitation, these fluid requirements can be given orally to reduce costs. Intravenous fluids are generally given via jugular or auricular (ear) vein catheter. Catheterization of the auricular vein in calves has been described in detail previously.[39]

As stated above, practitioners must rely on clinical signs such as ability to stand, suckling intensity, loss of palpebral reflex, and age of diarrheic calves to predict if alkalinizing therapy is indicated and how much isotonic sodium bicarbonate should be administered. Because determining the severity of acidosis on the farm is difficult and costly, buffer administration is commonly done without any laboratory data. Therefore the clinical response of the calf to IV fluid therapy must be monitored. Urination within 30 to 60 minutes, improvement of mental and hydration status, and most importantly restoration of the suckle reflex are monitored as a response to treatment. Recumbent calves should stand within a few hours of IV fluid therapy. If the suckle reflex does not return after IV buffer therapy, other diseases such as septicemia, omphalitis or pneumonia should ruled out.

Endnote

[a]Abbott Point of Care Inc, Princeton, NJ

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