Treatment of Calf Diarrhea: Oral Fluid Therapy

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KEYWORDS

• Calf diarrhea • Acid-base • Fluid therapy • Neonatology
• Acidosis • Dehydration • Oral electrolyte

Neonatal diarrhea remains the most common cause of death in 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. According to the World Health Organization (WHO), the development of oral rehydration therapy was one of the most significant advances in human medicine of twentieth century. Oral rehydration also continues to serve as the backbone of treatment protocols for diarrhea in neonatal calves. This article provides an overview of oral electrolyte therapy in calves, emphasizing when they should be used, how they should be used, and what practitioners should be looking for when choosing a product.

A complete review of the pathophysiology of diarrhea is beyond the scope of this article and is covered elsewhere in this issue. Some pathogens cause secretory diarrhea, causing small intestinal enterocytes to switch from net absorption of fluid to 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). Regardless of the pathogen or the mechanism involved, diarrhea increases the loss of electrolytes and water in the feces of calves and decreases milk intake. This process 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). Diarrhea is by far the most common indication for fluid therapy in neonatal calves. Oral electrolyte solutions have classically been used to replace fluid losses, correct acid–base and electrolyte abnormalities, and provide nutritional support, because they are cheap and easy to administer on-farm.

The goals of oral fluid therapy are to replace fluid, acid–base, and electrolyte deficits and to provide nutritional support. They are indicated in any diarrheic calf that has at

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least a partially functional gastrointestinal tract. If oral electrolytes are administered to a calf that has ileus, the fluid pools in the rumen resulting in bloat and rumen acidosis. In general, a calf that has any sort of suckle reflex or that demonstrates any ''chewing'' action can be considered to safely tolerate oral fluids.

ASSESSING DEHYDRATION IN CALVES

Dehydration in calves that have diarrhea is accompanied by large decreases in extracellular fluid volumes along with small increases in intracellular fluid volumes.[1–3] The intestinal loss of electrolytes in these calves results in hypoosmotic extracellular (plasma and interstitial) fluid, which causes free water to move from the extracellular fluid (ECF) to the intracellular fluid (ICF) space (thereby increasing ICF space). The practitioner must therefore attempt to clinically estimate the degree of ECF loss in dehydrated calves during physical examination.

Attempts to estimate dehydration based on physical examination findings have been around for more than 40 years. In 1965, Watt[4] evaluated hydration status by assessing the attitude of the calf, eyeball position, skin elasticity, mucous membrane appearance, capillary refill time, and urine production and classified dehydration as mild, moderate, or severe. It was later recognized, however, that these guidelines were certainly subject to error.[5] One of the more accurate predictors of acute dehydration is monitoring change in body weight. Using this principle, Bywater[6] took the three established categories of severity and assigned weight losses of 1% to 5% for mild dehydration, 6% to 8% for moderate dehydration, and 9% to 11% for severe dehydration. These categories were likely developed based on data that indicated most calf deaths occurred when weight loss was between 12.7% and 13.4% of body weight.[7,8]

A study by Constable and colleagues[1] has provided more accurate data for estimating hydration status in calves in the field. This study used an experimental model that produced severe, acute diarrhea.[9] Several clinical and laboratory parameters were monitored throughout the duration of the study and compared with actual percent dehydration of each calf. The results of this study indicated that the most accurate methods for assessing dehydration in calves are eyeball recession into orbit (degree of enophthalmos), skin tent duration in the neck region, and plasma protein concentration. All other methods of assessment are inferior to these. The degree of enophthalmos is estimated by gently everting the lower eyelid and estimating the recession of the globe into the orbit (**Fig. 1**). Skin elasticity is best measured on the lateral side of the midcervical area by pinching a fold of skin, rotating it 90 degrees, and measuring the time for the skinfold to disappear. The data from this study provide

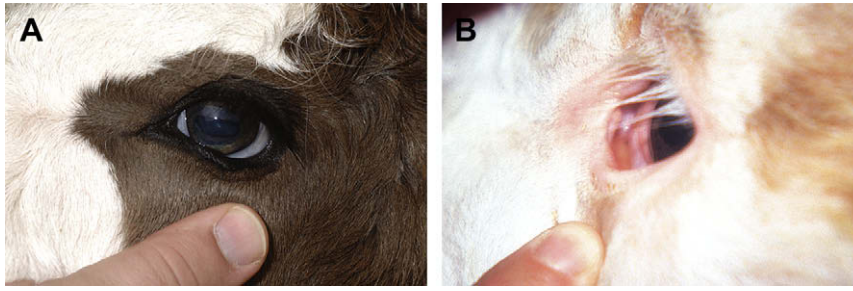

Fig. 1. Calf on the left (*A*) has a normal hydration status. There is no space between the eyelid and the eyeball. The calf on the right (*B*) is severely dehydrated. The eye is sunken at least 7 to 8 mm into the orbit. (*Courtesy of* Peter Constable, BVSc, MS, PhD, MRCVS, West Lafayette, IN.)

the most practical and accurate method for predicting hydration status in calves that have diarrhea (**Table 1**).

Eyeball recession may not be as accurate in chronic cachexia. Because the position of the eye depends partially on body fat stores, it is possible that eyeball recession is of limited value to predict hydration status in calves that have chronic diarrhea. In these calves it is likely that skin elasticity over the neck region or thorax may be a better indicator.[1] Using these clinical parameters can be somewhat subjective and initial assessments may occasionally be inaccurate. Eyeball recession and skin tent duration in the neck region provide more accurate clinical indicators of dehydration than any other parameter that can be easily measured during the physical examination process.

CHOOSING AN ORAL ELECTROLYTE PRODUCT

Oral electrolyte solutions were originally developed in human medicine for treatment of diarrhea associated with cholera infection and have been credited as being one of the most significant medical advances of the twentieth century. The original WHO electrolyte formulation was based on the following main principles:[10]

- It was an isotonic solution that contained an approximately equimolar mixture of sodium (90 mmol/L) and glucose (2%).
- It contained potassium because of the severe potassium depletion associated with diarrhea and anorexia.
- It contained glycine to facilitate absorption of sodium, glucose, and water.
- It contained bicarbonate to correct the metabolic acidosis associated with diarrhea.

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

Considerable variability exists in the quality of commercial oral electrolyte solutions available today and practitioners must put some thought into the product they choose to use in practice (**Table 2**). As was eloquently stated in a previous article by Dr. Robert Michell,[11] simply recommending oral electrolyte rehydration in this decade is as imprecise as advocating antibiotics 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 oral electrolyte solution must satisfy the following four requirements: (1) supply sufficient sodium to normalize the ECF 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 metabolic acidosis usually present in calves that have diarrhea; and (4) provide energy, because most calves that have diarrhea

Table 1
Guidelines for assessment of hydration status in calves with diarrhea

Dehydration	Demeanor	Eyeball Recession	Skin Tent Duration (s)
<5%	Normal	None	<1
6%–8% (mild)	Slightly depressed	2–4 mm	1–2
8%–10% (moderate)	Depressed	4–6 mm	2–5
10%–12% (severe)	Comatose	6–8 mm	5–10
>12%	Comatose/dead	8–12 mm	>10

Table 2
Comparison of oral electrolyte products available in North America

	Sodium (mmol/L)	Potassium (mmol/L)	Chloride (mmol/L)	Strong Ion Difference	Alkalinizing Agent	Total Osmolality (mOsm/L)
Advance Arrest (MS Specialty Nutrition)[a]	46	7	30	23	Bicarbonate (12 mmol/L)	245
Biolyte (Pfizer)	142	24	80	86	Bicarbonate (86 mmol/L)	732
Bounce Back (Manna Pro)[a]	136	10	112	34	Bicarbonate (48 mmol/L)	
Blue Ribbon Calf Electrolytes (Merrick)[a]	144	20	75	89	None	390
Bovine Bluelite C (Techmix)	59	24	56	27	None	269
Calf-Lyte II (Vetoquinol)	112	15	43	84	Acetate (80 mmol/L)	428
Calf-Lyte II HE (Vetoquinol)	112	15	43	84	Acetate (80 mmol/L)	726
Calf Quencher (Vedco)	142	24	80	86	Bicarbonate (86 mmol/L)	731
Deliver (Agri-Labs)[a]	67	16	49	34	Bicarbonate (36 mmol/L)	305
Diaque (Boehringer Ingelheim)	90	15	55	50	Bicarbonate (25 mmol/L) and acetate (12 mmol/L)	377
Entrolyte HE (Pfizer)	106	26	51	81	Bicarbonate (80 mmol/L)	739
Epic calf electrolyte (Bioniche)	92	30	45	77	Acetate (52 mmol/L)	360
Hydrafeed (A&L Laboratories)	110	10	40	80	Bicarbonate (80 mmol/L)	380
Hydralyte (Vet-A-Mix)	90	30	45	75	Acetate (60 mmol/L)	614
Hysorb (Bimeda)	120	10	70	60	Bicarbonate (40 mmol/L)	360
OneBetter calf electrolyte (Felton)	124	24	63	85	Bicarbonate (12 mmol/L)	440
Resorb (Pfizer)	75	25	80	20	None	315
Revibe (Wyeth)	120	20	50	90	Acetate (80 mmol/L)	466
Revitilyte (Vets Plus Inc.)	110	50	20	140	Bicarbonate (90 mmol/L)	577
VitaLyte (Vita Plus Corp.)	150	31	45	136	Bicarbonate (80 mmol/L)	527

This listing does not include every product available in North America. No discrimination or specific endorsement of any product is intended.

[a] Signifies data were 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 not be completely accurate.

are in a state of negative energy balance.[12] Factors to consider when choosing an oral electrolyte solution include the following.

Sodium Concentration

Sodium is the osmotic skeleton of the extracellular fluid and therefore of plasma. Because sodium is the principal determinant 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 lower concentrations are not able to adequately correct dehydration. For example, one study compared the ability of three different commercially available oral electrolyte solutions to resuscitate calves using an enterotoxigenic *Escherichia coli* diarrhea model.[11] The three electrolyte products had sodium concentrations of 120, 73, and 50 mmol/L. Results of this study showed that the product containing sodium at 120 mmol/L was able to restore extracellular fluid volume and correct dehydration, whereas the other two products containing lower sodium concentrations were not. Oral electrolyte products with very high sodium concentrations might be expected to cause hypernatremia; however, there is not a lot of research to demonstrate what sodium concentration would be too much. Results from studies that have fed oral electrolyte products for multiple days containing either 120 or 134 mmol/L of sodium have not resulted in hypernatremia;[11,13] however, in the author's opinion, products with sodium concentrations much higher than 130 mmol/L should be avoided. Very high sodium concentrations have also been shown to delay abomasal emptying rates because of increased osmolality and may cause ileus, thus predisposing to abomasal bloat and other gastrointestinal disorders.[14]

Chloride Concentration

Although calves lose chloride during diarrhea, this loss does not occur nearly to the same degree as sodium.[15] A general guideline has been that oral electrolyte products should contain chloride in concentrations between 40 and 80 mEq/L. When considering the importance of strong ion difference (SID) in correcting metabolic acidosis (see more thorough discussion later under alkalinizing agents), it may be advisable to use products with chloride concentrations toward the low end of the above range to increase the SID (see later discussion).

Potassium Concentration

Like sodium and chloride, potassium is lost in the feces of calves that have diarrhea. All calves that have diarrhea therefore have a total body deficit of potassium.[8] In acute cases of diarrhea, however, calves may have elevated blood potassium concentrations (hyperkalemia). This paradoxical situation arises in response to metabolic acidosis. The Na^+-K^+-ATPase pump functions optimally at physiologic pH ranges. During acidemia, the pump starts to fail, causing intracellular Na^+ ions to increase (they are not pumped out of the cell) and extracellular K^+ ions to increase. The ECF (which usually contains only about 5% of the total body potassium) therefore has greater-than-normal potassium concentrations, which can result in hyperkalemia. Because of increased fecal loss, however, ICF and total body potassium concentrations are decreased.

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. In chronic cases of diarrhea, therefore, calves can have profound depletion

of body potassium stores and generally have low serum concentrations of K^+. 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 that have diarrhea contain potassium concentrations between 10 and 30 mmol/L. Higher K^+ concentrations might theoretically be beneficial in calves with chronic diarrhea that have extreme depletion of total body potassium; however, there is no research available to support this recommendation nor is the author aware of any commercially available products contain levels of K^+ significantly higher than 30 mmol/L.

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.[16] One of the earliest mechanisms of intestinal sodium absorption discovered was linked with sugar.[17] Glucose can be cotransported with sodium from the intestinal lumen to the inside of the enterocyte at the brush border membrane. At the basolateral membrane, the Na^+-K^+-ATPase actively pumps Na^+ ions out of the cell thus raising the intercellular osmolality.[16] Any increase in sodium influx at the brush border must be compensated for by an increase in sodium efflux from the enterocyte at the base of the cell. This increase in intercellular osmolality then draws more water from the intestinal lumen through the tight junctions between cells, thus expanding extracellular fluid volume and rehydrating the calf. Because this mechanism was well understood by the 1960s, 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.[16] Whether amino acids are needed in addition to glucose in oral electrolyte solutions is not well understood; however, the addition of glycine does seem to further improve water absorption in the intestine. In addition, volatile fatty acids, such as acetate or propionate, have been shown to facilitate sodium absorption in the gut.[18,19] In studies using isolated loops of small intestine from calves, electrolyte solutions containing acetate showed markedly enhanced sodium absorption when compared with formulations with other solutes.[18] The mechanism by which volatile fatty acids stimulate sodium absorption in the intestine seems to be different from that of glucose or amino acids. Acetate therefore seems to have an additive effect to glucose and amino acids, meaning you can expect a significant increase in intestinal sodium absorption in electrolyte products containing volatile fatty acids, even when they already contain high concentrations of glucose or glycine.

Glucose-To-Sodium Ratio

Glucose is present in various concentrations in virtually all commercially available oral electrolyte solutions. It is necessary to facilitate sodium absorption and to provide an energy source for the calf. The ratio of glucose to sodium present in an oral electrolyte solution should also be considered, however. This ratio can be calculated by adding the mmol/L of dextrose in a product (along with glycine if present) and dividing by the mmol/L concentration of sodium. This ratio should fall somewhere between 1:1 and 3:1.[20] Products that have a glucose-to-sodium ratio less than 1:1 do not contain adequate solute to facilitate sodium absorption (unless perhaps the product also contained significant levels of acetate or propionate). Conversely, products that have

a glucose-to-sodium ratio greater than 3:1 are likely to increase the risk for osmotic diarrhea.

Osmolality

Commercially available oral electrolyte products in North America can range from isotonic (280–300 mOsm/L) to extremely hypertonic (700–800 mOsm/L). The primary difference in most of these products is the amount of glucose that is added. Because of a countercurrent exchange mechanism in the small intestine, the effective osmolality at the tip of the intestinal villus is about 600 mOsm/L.[21] We can therefore take advantage of hypertonic solutions that have higher energy levels. On the other hand, low osmolality fluids (<350 mOsm/L) generally have inadequate energy content because they have insufficient glucose. Hypertonic solutions provide greater nutritional support to calves relative to isotonic products and have not been shown to cause detrimental effects, particularly in relation to maintaining hydration status, intestinal osmolality, serum glucose concentrations, and intestinal flow rate.[22] Research has demonstrated that milk replacer is better able to maintain normal serum glucose concentration than either hypertonic or isotonic oral electrolyte solutions.[12] As expected, however, 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.[12] Multiple studies have demonstrated that hypertonic oral electrolyte solutions maintain higher serum glucose and lower β-OH butyrate (ketone) concentrations when compared with isotonic electrolyte solutions.[12,23] Previous research has also shown that when calves were deprived of milk, those fed isotonic oral electrolyte solutions had significantly greater weight loss as compared with calves fed hypertonic oral electrolytes.[24]

With the principle that hypertonic oral electrolytes supply more energy to calves as compared with isotonic products, the next question becomes at what osmolality might we start to see deleterious effects? The physiologic effect of higher-than-normal intestinal glucose concentrations in calves that have diarrhea is not completely understood; however, the addition of glucose to facilitate intestinal sodium and water absorption increases the risk for osmotic diarrhea if the glucose is not absorbed. Although the research available to date does not provide a good answer to that question, there are certainly indications that electrolyte solutions with extremely high osmolalities (>700–750 mOsm/L) and glucose concentrations might cause problems. To begin with, a product with an osmolality greater than what is already present in the intestinal lumen could worsen diarrhea. Most calves that have enteric pathogens already have hypersecretion of electrolytes and water into the small intestinal lumen, which could be exacerbated with the feeding of extremely hypertonic solutions (electrolyte or milk replacer). Raising the intraluminal tonicity would serve to increase the secretion of water and electrolytes into the intestine, thus increasing the severity of diarrhea. This effect would likely be magnified with severe villus damage, which is often present in diarrheic calves.

The primary energy source in an oral electrolyte solution is glucose, which is provided in most oral electrolyte solutions between 2 to 3 g of glucose per kg of body weight. The small intestine of the healthy calf has been shown to absorb all glucose when fed at 2.5 g/kg of body weight.[22] In anesthetized calves, glucose was absorbed in both healthy and diarrheic calves at a rate of 2.4 to 7.2 mg/cm of small intestinal segment per hour.[25] Based on a mean small intestinal length of 15.8 to 18.6 m in 1- to 2-week-old Holstein calves,[26] the total glucose absorption rate in the small intestine is estimated to range from 3.8 to 13.4 g per hour.[14] Assuming twice a day feeding, calves should be able to absorb up to 161 g of glucose per feeding. This

calculation suggests that for a 45-kg calf that has normal plasma glucose concentration and gastrointestinal motility, the upper limit of glucose in an oral electrolyte solution should about 3.6 g/kg.[14] Higher concentrations may allow unabsorbed glucose to carry over into the large intestine, where it may be fermented to short-chain volatile fatty acids, exacerbate fecal water loss, and worsen diarrhea.

Hypertonic oral electrolyte solutions have also been shown to slow abomasal emptying rates as compared with isotonic products.[14,27] Calves fed an oral electrolyte solution with a total osmolality of 360 mOsm/L had a significantly faster abomasal emptying rate as compared with calves fed a solution with an osmolality of 717 mOsm/L.[27] This finding suggests that electrolyte products with a high osmolality (or high glucose concentrations) would be likely to induce abomasal ileus, thus increasing the risk for bloat or abomasitis. Abomasal bloat is a syndrome in young calves characterized by anorexia, abdominal distension, bloat, and often death in 6 to 48 hours. This condition occurs most commonly in dairy calves and seems to have a sporadic occurrence with some farms having multiple outbreaks at times. Recently the abomasal bloat syndrome was experimentally reproduced by drenching young Holstein calves with a carbohydrate mixture containing milk replacer, corn starch, and glucose mixed in water.[28] The authors of this study proposed that the pathophysiology of abomasal bloat is primarily excess fermentation of high-energy gastrointestinal contents. Gas-producing bacteria, such as *Clostridium perfringens*, *Sarcina ventriculi*, or *Lactobacillus* species have also been believed to play a role in this syndrome.[28,29] Although the exact pathogenesis of abomasal bloat is not completely understood, the disease is likely to be multifactorial in origin. Having large amounts of fermentable carbohydrate present in the abomasum (from milk, milk replacer, or high-energy oral electrolyte solutions) along with the presence of fermentative enzymes (produced by bacteria) would likely lead to gas production and bloat. This process would be exacerbated by anything that slowed abomasal emptying or caused gastrointestinal ileus. In fact, feeding high-osmolality electrolyte products or milk replacers has been noted to be a risk factor on some farms for the development of abomasal bloat in calves (Geof W. Smith, DVM, MS, PhD, unpublished data, 2008).

Although the ideal osmolality of an oral electrolyte solution for calves is not completely understood, a hypertonic oral electrolyte solution (500–600 mOsm/L) would be ideal in dairy calves or in beef calves that have been separated from the dam. Certainly if milk were to be withheld for any length of time, a hypertonic oral electrolyte solution would be indicated to provide energy to the calf. Isotonic solutions might still be appropriate, however, for beef calves that are still suckling or in conjunction with milk replacer in dairy calves that maintain a good appetite. The author recommends avoiding extremely hypertonic oral electrolyte product (>700 mOsm/L) for the reasons stated previously.

Alkalinizing Ability

Acidemia and metabolic acidosis occur in almost all cases of calf diarrhea. This finding was originally attributed to bicarbonate loss in the feces along with a decrease glomerular filtration rate in response to severe dehydration.[7,15,30] More recent data have indicated that metabolic acidosis in calves that have diarrhea actually results from differences in strong ion balance (described in more detail later).[15] We must therefore attempt to correct this strong ion acidosis when using oral fluid therapy. Research examining intravenous fluid therapy protocols has indicated that severely acidemic calves are unable to correct their metabolic acidosis, even when rehydrated with nonalkalinizing fluids.[31] It is imperative, therefore, that either oral or intravenous fluid therapy protocols be able to increase blood pH. Classically this has been done by adding

alkalinizing agents (ie, bicarbonate, acetate, or propionate) to oral electrolyte mixtures. More recently, there has been growing interest in looking at the 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 and warrant discussion.

Alkalinizing agents

Acetate, propionate, and bicarbonate are all considered alkalinizing agents and are frequently present in commercial oral electrolyte solutions. Bicarbonate-containing fluids are effective at correcting a severe acidosis, because 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.[32–34] Acetate and propionate are only effective alkalinizing agents when they are metabolized by the liver; a process that forms water and creates hydrogen ions. This metabolic process seems to still function efficiently in calves that have severe diarrhea because the alkalinizing ability of the acetate has been shown to be as effective as bicarbonate.[33] Acetate and propionate have several advantages over bicarbonate:

- Acetate and propionate facilitate sodium and water absorption in the calf small intestine, whereas bicarbonate does not.
- Acetate and propionate produce energy when metabolized, whereas bicarbonate does not.
- Acetate and propionate do not alkalinize the abomasum, whereas bicarbonate does; low abomasal pH is a natural defense mechanism against bacterial proliferation.
- Acetate and propionate do not interfere with milk clotting in calves, whereas bicarbonate may potentially cause some disturbance of the normal digestive process.

Alkalinization of the abomasum Gastric acidity is a well-accepted barrier to colonization and infection of the gastrointestinal tract by bacteria, and is a primary defense mechanism against pathogens that are ingested orally.[35] Bacteria, such as *E coli* and *Salmonella*, are killed at gastric pH between 2.5 and 3.0, whereas they multiply at pH greater than 5.0.[36,37] Maintaining a low abomasal pH is therefore critical to avoid colonization of the intestinal tract with pathogenic bacteria in calves. An example of the importance of abomasal acidity is provided by enterotoxigenic *E coli* (ETEC). ETEC is an important cause of diarrhea in calves 1 to 2 days of age, but does not cause disease in older calves. In colostrum-fed calves older than 24 hours of age, oral administration of $NaHCO_3$ (4–10 g in 60–150 mL of water), followed immediately by an inoculum of viable ETEC bacteria, can produce clinical disease. Alkalinization of the abomasum with bicarbonate is thus necessary to produce ETEC in older calves. This modified protocol has been successful in producing an experimental model of diarrhea in calves up to 14 days of age.[7,25,38,39] Oral administration of the bacteria without $NaHCO_3$ does not produce any clinical diarrhea, however.

The feeding of oral electrolyte products containing bicarbonate has been shown to alkalinize the abomasum in calves.[40–43] Suckling of bicarbonate-containing oral electrolyte solutions can cause a large and sustained increase in abomasal pH (**Fig. 2**). A similar effect is not seen with acetate-based products.[41,42] Abomasal acidity provides a natural barrier to ingested bacteria, and maintaining a low abomasal pH decreases the number of viable coliform bacteria that reach the small intestine. This process increases nonspecific resistance to intestinal colonization. The increase in abomasal pH

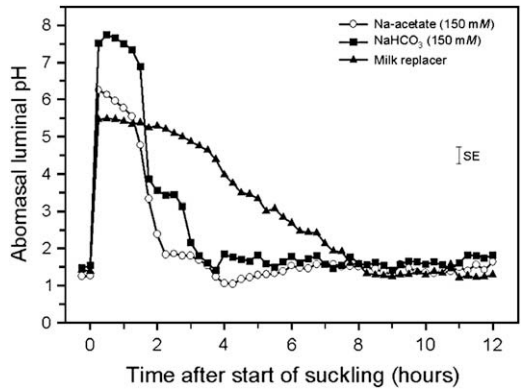

Fig. 2. Mean 12-hour abomasal luminal pH (least square mean ± SEM values) in five Holstein calves fed 2 L of milk replacer or 150 mmol/L solutions of sodium acetate or sodium bicarbonate. (*Courtesy of* Peter Constable, BVSc, MS, PhD, MRCVS, West Lafayette, IN; and *Modified from* Marshall TS, Constable PD, Crochik SS, et al. Effect of suckling an isotonic solution of sodium acetate, sodium bicarbonate, or sodium chloride on abomasal emptying rate and luminal pH in calves. Am J Vet Res 2008;69:824–31; with permission.)

seen with electrolyte products that contain high concentrations of bicarbonate may therefore facilitate growth of bacterial diarrheal pathogens and thus increase the severity, duration, and mortality rate associated with diarrhea in calves.

Interference with normal digestion During the normal digestion process, milk clots in the abomasum as casein coagulates under the influence of renin and pepsin. The whey fraction then passes quickly into the small intestine, whereas digestion of the abomasal clot continues for up to 12 hours.[44] In the early 1990s, in vitro experiments demonstrated that bicarbonate-containing oral electrolyte products inhibited the clotting of milk in the abomasum.[45] A further study examining 50 different commercially available oral electrolyte products formulated for calf oral rehydration therapy showed that those that contained bicarbonate consistently inhibited milk clotting, whereas those that contained acetate or propionate did not.[46] The conclusion of this research was that bicarbonate-based oral electrolyte solutions would upset the normal digestive process if fed together with milk or milk replacer. The recommendation to separate the feeding of milk and electrolytes by 2 to 4 hours has been common for the last 15 years. In fact, the labels of many electrolyte products still contain statements warning against concurrent feeding with milk for this reason. A more recent study demonstrated that an oral electrolyte product containing low concentrations of bicarbonate (25 mmol/L) and citrate (12 mmol/L) did not inhibit clot formation in calves.[47]

The importance of abomasal clot formation in milk digestion has since been questioned. Early milk replacers in the United States were formulated with casein-containing protein sources. Currently the milk replacer industry uses protein sources that are nonclotting, such as whey and soy protein, to formulate milk replacers.[48] Although first judged as inferior to casein, many of these nonclotting milk replacers can produce growth rates far superior to products that were originally used. Eventually it was determined that factors other than clot formation were responsible for calf performance and this process may not be as important as was originally believed.[48] Although the significance of bicarbonate inhibiting abomasal clot formation is not well understood, it is possible that these products do somehow interfere with the normal digestive

process. It has been shown that electrolyte solutions containing bicarbonate reduce growth rates in calves when fed simultaneously with milk.[49] Although the exact cause or clinical importance of this reduction in growth rate is not well understood, some experts still recommend that bicarbonate-based oral electrolytes not be fed together with milk or milk replacer. Products containing acetate or propionate as an alkalinizing agent would not have similar concerns and are well tolerated when fed with milk.

Strong ion difference

Strong ion theory is a different approach to looking at acid–base abnormalities. Traditionally, veterinarians have been taught to use the Henderson-Hasselbalch equation that uses measured pH and pCO_2 values along with calculated HCO_3 concentrations to characterize acid–base disturbances. This approach has several limitations that make it less than ideal for clinical use in sick animals.[50,51] The strong ion model reduces chemical reactions in plasma to that of simple ions in solution.[50] Strong ions are nonbuffer ions, meaning they are fully dissociated at physiologic pH values and do not participate in chemical reactions, yet exert an electrical effect. According to the strong ion model, the plasma SID is the primary determinant of acid–base balance in vivo. The primary strong cations are sodium (Na^+) and potassium (K^+) with minor contributions from calcium (Ca^{2+}) and magnesium (Mg^{2+}), and the primary strong anions are chloride (Cl^-), D- and L-lactate, and organic acids. Using the strong ion approach, the relationship between these ions becomes the primary factor that determines the acid–base status of an animal. Calves that have diarrhea have a tremendous loss of cations (Na^+ and K^+) relative to normal or increased strong anion concentrations, which creates a strong ion (metabolic) acidosis.[15] A significant part of the increase in strong anions is from D-lactic acid, which comes from bacterial fermentation of malabsorbed nutrients in calves that have diarrhea.[52] Calves that have diarrhea have a significantly higher serum concentration of D-lactic acid as compared with normal calves[52,53] and intravenous administration of D-lactate to normal calves has been demonstrated to induce many of the adverse clinical signs traditionally associated with metabolic acidosis.[54]

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^-). It has therefore been advocated to consider the SID of an oral electrolyte solution when choosing a product.[55] The SID 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 60 to 80 mEq/L would be recommended in a calf that has diarrhea. A case example may provide a better understanding of this concept. Suppose you are treating a calf that has diarrhea that can still stand but is lethargic and has a weak suckle reflex. We can assume this calf has a moderate metabolic acidosis with a base deficit somewhere around 8 mmol/L. Assuming the calf weighs 40 kg we can calculate a total base deficit as follows:

$$40\ \text{kg} \times 8\text{mmol/L} \times 0.6\ (\%\text{ECF}) = 192\ \text{mmol}$$

If we then administer 2 L of an oral electrolyte solution that has an SID of 80 we have corrected 160 mmol of this calf's base deficit. The metabolic acidosis would not be fully resolved; however, the pH would move much closer to the normal range. In contrast, if we fed 2 L of an oral electrolyte product that has an SID of 30, we have only corrected the base deficit by 60 mmol and the calf will continue to have a significant metabolic acidosis. This acidosis is likely to increase in severity as the calf has ongoing

electrolyte losses associated with diarrhea. From this example the practitioner can appreciate the importance of administering a solution rich in strong cations (high SID value) in calves that have diarrhea. Another important point is that oral fluid therapy in calves that have severe metabolic acidosis is not practical. These calves often have base deficit values greater than 20 mmol/L and much larger total body deficits (40 kg calf $\times$ 20 mmol/L $\times$ 0.6 = 480 mmol). Even with good quality electrolyte products, it becomes impractical or impossible to correct a deficit this severe with oral fluid therapy.

There are two different ways of thinking about alkalinizing ability when considering oral electrolyte products in calves. Is it more important to choose a product with an alkalinizing agent, or one with a high SID? In reality there is not a good answer to this question; however, to achieve optimal results, both factors are important. Studies with intravenous fluid therapy have demonstrated that the metabolic acidosis in calves does not resolve just by rehydrating the animal.[31] Several studies in diarrheic calves have shown that oral electrolytes without alkalinizing agents do not correct metabolic acidosis; in fact, they can have a mild acidifying effect.[11,33,34,56] Recovery rates are always higher and mortality always lower in studies that compare an oral electrolyte solution with an alkalinizing agent to one without.[33,56,57] It has generally been accepted, therefore, that oral electrolyte products should contain 50 to 80 mmol/L of an alkalinizing agent.[44]

When thinking about the strong ion approach, it would certainly be possible to correct metabolic acidosis in a calf without an alkalinizing agent by choosing a product with a high SID. Studies comparing different oral electrolyte solutions that do not contain any alkalinizing agent consistently show that products with higher SIDs have a greater alkalinizing effect than products with lower SIDs.[11,38] To achieve maximum alkalinizing ability out of an oral electrolyte product, however, the author believes that both elements are important. For example, one study showed there was no difference in the alkalinizing ability of an electrolyte solution that contained 80 mmol/L of acetate and had an SID of 90 when compared with a product that contained 80 mmol/L of bicarbonate and had an SID of 88.[33] Both products were far superior to an oral electrolyte solution that contained no alkalinizing agent and had an SID of 15, however. The ideal electrolyte solution for use in calves that have diarrhea should contain at least 50 mmol/L of an alkalinizing agent (preferably acetate or propionate) and have a SID of at least 60 to 80. Unfortunately, products without alkalinizing agents and with low SIDs are commonly available in North America and should be avoided in calves that have diarrhea.

Psyllium

It has been hypothesized that adding dietary fiber (mucilage) in the form of psyllium to oral electrolyte solutions would enhance nutrient absorption from the digestive tract and improve glucose absorption by slowing gastric emptying.[58] In addition, fiber may help reduce the severity of diarrhea. It is able to pass undigested through the gastrointestinal tract and give a more formed appearance to the feces, which some practitioners and producers have correlated with increased efficacy of the oral electrolyte product. This improvement in fecal consistency is due to the gelling of liquid and should not be mistaken for a real improvement in the calf's overall condition. Research from multiple clinical trials has shown that the addition of psyllium to oral electrolyte solutions does not improve glucose absorption in calves that have diarrhea.[59,60] In fact, one study showed that calves fed oral electrolytes containing psyllium had significantly lower glucose concentrations after feeding as compared with the same oral electrolyte formulation without psyllium.[59] Although supplemental dietary fiber may

generate slight improvements in fecal consistency, it seems to impair glucose absorption in the small intestine and is not recommended for inclusion in oral electrolyte products intended for calves.

ADMINISTRATION OF ORAL ELECTROLYTES

In general, oral electrolytes should be fed as an extra meal to calves that have diarrhea. For example, if calves are normally being fed twice a day (morning and evening), then oral electrolytes can be fed in the middle of the day. If the additional labor required for the extra feeding is not available, then electrolytes can be fed along with milk (particularly those products that contain acetate or very low concentrations of bicarbonate). Some farms prefer to offer diarrheic calves constant access to low-osmolality electrolytes throughout the day. Regardless of the feeding schedule for electrolytes, it is best to continue milk or milk replacer in these calves.

Some experts have recommended a ''rest the gut'' approach to treating calf diarrhea, suggesting that continued milk feeding worsens the diarrhea. This concept is based on the principle that milk supplies nutrients in the intestines that the bacteria could use as an energy source. This process would lead to further maldigestion of nutrients and increased excretion of fluids (thus more diarrhea). Other arguments for withholding milk in calves that have diarrhea include a faster healing of the intestines, less opportunity for overgrowth of the intestines with harmful bacteria, and impaired digestion and use of milk or milk replacer. Despite these ideas, research has shown milk feeding does not prolong or worsen diarrhea, nor does it speed healing of the intestines. In a study by Garthwaite and colleagues,[61] 42 calves that had naturally occurring diarrhea were divided into three groups. In one group milk was withheld and calves were fed only oral electrolytes, followed by a gradual return to milk after 2 days. In the second group there was partial removal of milk; calves were fed only a small amount (2.5% of body weight for 2 days followed by 5% of body weight for 2 days) along with oral electrolytes. In the third group calves were continued on their full allotment of milk (10% of body weight per day) along with electrolytes. There was no difference in the severity or duration of diarrhea between any of the groups during the study. The calves that had diarrhea that were fed both milk and oral electrolytes gained more weight than did calves from which milk was withheld for 1 to 2 days. The calves that continued to receive milk actually gained weight during the study period, whereas calves in the other two groups lost weight. Weight loss in calves limited to only oral electrolyte solutions has been reported in other studies also.[24]

Another study used an experimentally induced model of diarrhea in calves fed either milk (2 L every 12 hours), an isotonic oral electrolyte solution (85 mmol glucose), or a hypertonic oral electrolyte solution (330 mmol glucose) over a 48-hour period. Serum glucose concentrations were unchanged over the 48-hour period in the calves fed milk, but steadily declined throughout the study in both groups fed only oral electrolytes.[12] Calves fed only electrolytes developed significant increases in β-OH butyrate and nonesterified fatty acid concentrations over the 48-hour period, indicating these calves were in a profound negative energy balance (**Fig. 3**). These studies indicate that even hypertonic oral electrolyte products with very high glucose concentrations do not provide significant energy to meet the maintenance and growth requirements of a calf. The recommendation to temporarily discontinue milk feeding in calves that have diarrhea is therefore inappropriate. Calves should be maintained on their full milk diet plus oral electrolytes when possible. If calves are depressed and refuse to suckle, milk can be withheld for one feeding (12 hours) and a hypertonic oral electrolyte product substituted. Milk feeding should always be resumed within 12 hours.

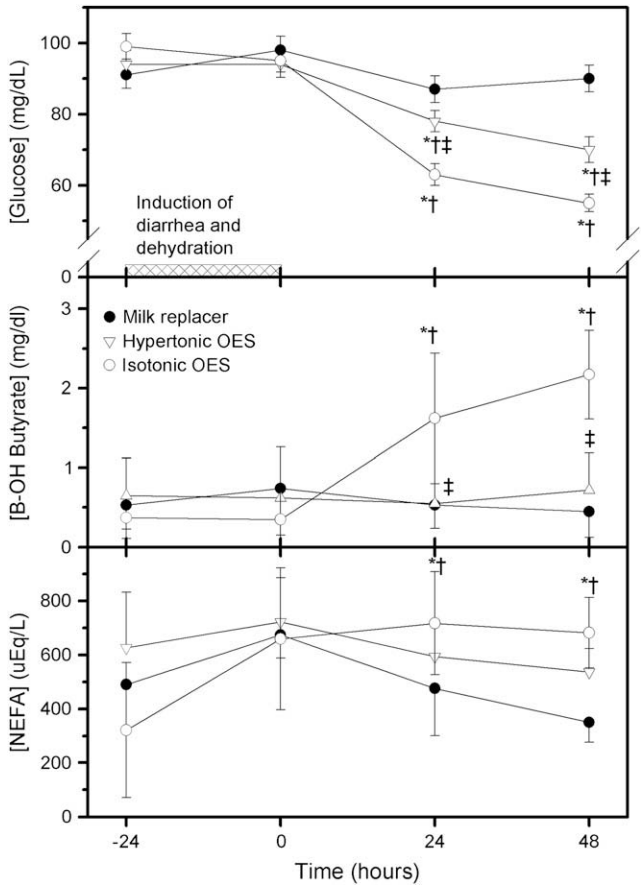

Fig. 3. Serum glucose, β-OH butyrate, and nonesterified fatty acid concentrations in neonatal calves that had experimentally induced diarrhea and dehydration. Calves were administered milk replacer, hypertonic oral electrolyte solution, or isotonic oral electrolyte solution. Values are expressed as mean ± SD. *P<.05; compared with time = 0 value; †P<.05 compared with milk replacer group at the same time interval; ‡P<.05 compared with the isotonic group at the same time interval. (*Modified from* Constable PD, Thomas E, Boisrame B. Comparison of two oral electrolyte solutions for the treatment of dehydrated calves with experimentally-induced diarrhea. Vet J 2001;162:129–40; with permission.)

SUMMARY

In summary, oral electrolytes continue to be the hallmark of routine therapy for treating neonatal calf diarrhea. It is important that practitioners are able to assess dehydration accurately and understand how and when to use oral electrolyte products. There are tremendous differences in the formulation of commercially available electrolyte products found in North America and around the world. All products are not created equally and choosing which of these products to use in practice is an important decision. Practitioners should focus on selecting oral electrolyte solutions that satisfy the following four requirements: (1) supply sufficient sodium to normalize the ECF volume, (2) provide agents that facilitate absorption of sodium and water from the intestine,

(3) correct the metabolic acidosis usually present in calves with diarrhea, and (4) provide energy. Additionally, the oral electrolyte should not cause any deleterious effects (such as abomasal bloat). Because veterinarians are often not directly involved with the administration of oral electrolytes to calves, it is important that they examine the electrolyte product being used in their clients' herds and make recommendations when appropriate.

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