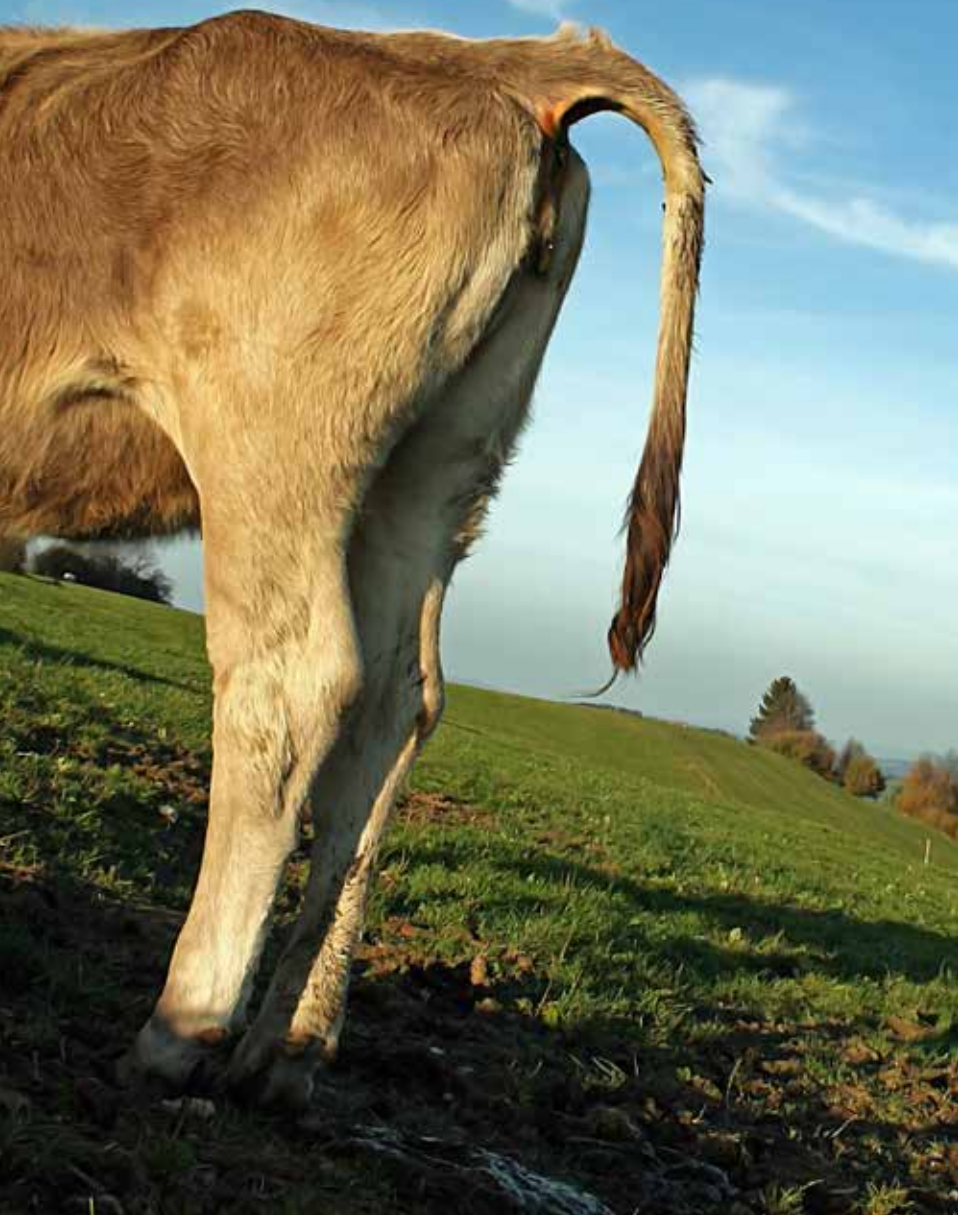
Calf diarrhoea

Sandra Forsyth, of SVS Laboratories, stresses the importance of understanding the fluid, electrolyte and acid-base requirements of calves with diarrhoea to ensure the correct therapy is given.



THE TIME OF the year when calf diarrhoea can become a major issue on dairy farms is upon us. While diarrhoea reduces growth rate at best, and causes death at worst, blood-work is rarely assessed in these cases. Therefore, understanding the biochemical abnormalities that are likely to occur is important in determining disease severity and establishing a treatment plan.

DEHYDRATION

Diarrhoea-associated dehydration, if undertreated, can reduce tissue perfusion and lead to hypovolaemic shock. Fluid deficits are usually determined by clinical observations, and, except for serum total protein (TP), laboratory tests have limited utility. TP usually increases in cases of dehydration, unless intestinal damage is severe (eg, haemorrhagic diarrhoea), when protein loss may reduce the apparent severity of the dehydration.

Measuring serum TP is more useful for detecting failure of passive transfer, which may be contributing to the diarrhoea. While direct measurement of immunoglobulin (IgG) is ideal, TP is a relatively good proxy, and better than gamma glutamyl transferase (GGT) to assess failure of passive transfer. GGT measures the volume of ingested colostrum, rather than quality. A TP >52g/L reflects adequate IgG, provided that the calf is well hydrated. As TP increases with dehydration, it must be interpreted with caution when used to estimate IgG in calves with diarrhoea.

ELECTROLYTE LOSSES

Sodium, chloride and potassium are lost in moderate to large quantities in diarrhoea, exacerbating weakness, depression and ileus. Although serum potassium may be normal or high, a whole-body potassium deficiency is present due to GI loss. Low or normal serum potassium is seen with dehydration, whereas elevated potassium typically occurs when there is a fall in glomerular filtration rate accompanying hypovolaemia. Hyperkalaemic calves show bradycardia, arrhythmias,

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TABLE 1:

Base deficit[1] as an estimate of acidosis in calves of different ages

Clinical signs	Estimated base deficit (mmol/L)	
	<8 days of age	>8 days of age
BAR, strong suckle reflex, warm	0	5
Standing or lying, moves if disturbed, weak suckle reflex, slightly cold	5	10
Depressed, sternally recumbent, unwilling to rise, no suckle reflex, cold	10	15
Very depressed, laterally recumbent, no suckle reflex, very cold	10	20

[1]Base deficit is calculated: Base Deficit (BD) = Normal [HCO_3^-] – Patient [HCO_3^-]

DLA is excreted through the kidneys. With appropriate fluid therapy the concentration falls within hours, and is normal by 24-48 hours.

CALCULATING BICARBONATE REQUIREMENTS

A 40kg calf with a 20mmol/L base deficit (BD) needs 480mmol HCO_3^- to replace the deficit and counteract the effect of lactate accumulation.

Bicarbonate requirement = (0.6L/kg) x (body weight, kg) x (BD, mmol/L)

= 0.6 x 40 x 20 = 480mmol

0.6 is the volume of distribution of bicarbonate (a constant).

recumbency and shock, and require urgent treatment. Conversely, severe hypokalaemia causes ileus and muscle weakness, prolonging diarrhoea and recumbency, respectively.

HYPOGLYCAEMIA

Calves have very low energy stores, and anorexia and nutrient malabsorption readily result in a negative energy balance and hypoglycaemia, causing lethargy, hypothermia, seizures and coma.

ACIDOSIS

Acidosis is extremely common in calves with moderate to severe diarrhoea. It results from intestinal loss of bicarbonate, accumulation of L-lactic acid secondary to hypovolaemia and formation of D-lactic acid (DLA) from abnormal feed fermentation.

D-lactic acid: A special type!

Intestinal villus atrophy occurs with most infectious causes of diarrhoea, which allows undigested milk to reach the colon, where bacteria ferment lactose to DLA. Serum DLA may increase further in calves, with concurrent oesophageal groove dysfunction (OGD), which lets milk enter the rumen, where it is fermented to DLA. Factors that predispose to OGD include pain, feeding cold milk, force-feeding, irregular feeding times and poor-quality milk replacer. Calves with ruminal acidosis combined with diarrhoea have a poor prognosis.

Determining severity of acidosis

The severity of L-lactic acidosis is related to the severity of hypovolaemia, but D-lactic acidosis is not. While tachycardia, poor pulses and weakness accompany L-lactic acidosis, D-lactic acidosis produces clinical signs of inebriation ranging from ataxia to coma. The severity of acidosis is best determined by assessing serum [HCO_3^-] because clinical signs are inconsistent between individuals. Despite the variability in clinical response to acidosis, clinical signs remain common for estimating [HCO_3^-] deficits (Table 1). Urine pH has been touted as being useful in the field, but it is difficult to get a urine sample from a hypovolaemic calf. Regarding Table 1, there is nothing magical about being eight days of age, but the base deficit estimates were established from a study in which young calves with enterotoxigenic *E. coli* that doesn't produce significant acidosis were compared to older calves with diarrhoea of other aetiologies.

FLUID THERAPY

Fluids can be administered via the oral, intravenous, intraperitoneal and intraosseous routes. Subcutaneous administration has slow uptake, and solutions with dextrose may cause sterile abscess formation and consequently are not recommended.

Calves with <8% dehydration can be given oral replacement solutions (ORS), whereas those with greater dehydration, acidosis, or poor suckle reflexes need parenteral fluids.

Oral electrolyte solutions (ORS)

There is considerable variability in the composition of ORS, and it is necessary to put some thought into the solutions that you recommend to ensure they provide adequate but not excessive electrolyte, energy and alkalinising agent (Table 2). Kristina Mueller (2017) evaluated ORS and found few that are perfect. Electrolyte powders must be added to the correct volume of fluid to address dehydration and to prevent administration of electrolytes in toxic concentrations.

Sodium

Sodium is the skeleton of the extracellular fluid compartment, and it must be present in adequate quantities to retain water. Products containing less than 90mmol/L sodium do not correct dehydration, and products with high sodium content delay abomasal emptying.

TABLE 2:

Recommended concentrations for electrolytes in ORS

	Sodium	Potassium	Chloride	Strong Ion Difference (SID)*	Alkalinising Agent (acetate, propionate)	Osmolality
Recommended values	90-130mmol/L	10-30mmol/L	40-80mmol/L	60-80meq/L	50-80mmol/L	400-600mOsml/L

SID = ([Na+] + [K+]) – [Cl-]

Potassium

ORS must contain sufficient potassium to replace the whole-body depletion and to return gut activity and muscle function to normal.

Other components

Intestinal sodium absorption is passive and associated with active absorption of glucose and neutral amino acids such as alanine, glutamate and glycine. Too little glucose and insufficient amino acids reduce sodium uptake from the GI tract. Acetate and proprionate (volatile fatty acids) stimulate sodium uptake by a different, but poorly understood, mechanism, and add to sodium and water absorption.

Osmolality

ORS range from isotonic (280-300mOsm/L) to hypertonic (700-800mOsm/L), depending on the amount of glucose and sodium present. While glucose in ORS improves nutritional support, milk replacer is far better, but it does not supply the needed electrolytes and alkalinising agents. Administration of both provides the best resolution. Hypertonic solutions >700mOsm/L cause ileus, increasing the risk of bloat, and should be avoided, whereas solutions that are ~400-600mOsm/L are considered ideal.

Alkalinising agents

Acetate, bicarbonate, citrate and proprionate are alkalising agents. Bicarbonate immediately corrects acidosis, whereas other agents must be metabolised by the liver before they can do so. This might suggest that

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HCO_3^- is best; however, it alkalinises the abomasum, which facilitates bacterial growth, increasing the severity and duration of diarrhoea.

Intravenous fluids

IV fluids are given when calves are too weak to suckle, can't stand, have an absent palpebral response or are severely acidotic. DLA may be present in any sick calf with or without diarrhoea that has slow or absent palpebral or suckle reflexes and is ataxic, listless, recumbent or comatose.

Solutions

Hartmanns (LRS) is expensive because large volumes must be given. Also, it contains both L- and D-lactate, potentially exacerbating DLA. Acetated Ringers solution is an alternative, but is not commonly available in New Zealand.

Isotonic sodium bicarbonate is very efficient in correcting acidosis, but does not address other electrolyte deficiencies, and like LRS is expensive because of the need for an IV catheter, ongoing monitoring and relatively large volumes.

Hypertonic saline (HS) rapidly expands the vascular volume in severely dehydrated animals. When small volumes are given with ORS it is as effective as large volumes of LRS and can be given rapidly. However, it does not correct acidaemia.

Hypertonic sodium bicarbonate (HSB) in combination with ORS effectively expands vascular volume while also counteracting acidosis. Calves require about 10ml/kg of an 8.4% HSB solution (84g/L, 1mmol/ml) combined with 8-10ml/kg of an ORS.

SUMMARY

In a calf with diarrhoea, it is important to estimate fluid, electrolyte and acid-base requirements accurately so that the correct therapy is given. There are differences between various preparations, and choosing the right product is an important component of the decision-making process. Before the season starts, decide which ORS and fluids you will use for each situation. VS

REFERENCE:

Mueller, K. Comparison of commercial oral electrolyte solutions for scouring calves. *The Dairy Cattle Veterinarian* 35(10), 18–19, 2017