Treating cattle with *fodder-beet-farmer-management disorder* (rumen acidosis)

Jim Gibbs, Senior Lecturer in Veterinary and Ruminant Nutrition Research at Lincoln University, outlines some of the traps when it comes to treating cases of rumen acidosis.

THE RAPID EXPANSION of beet crops across New Zealand in the past decade or so has dramatically increased the number of cattle who are winter-fed beet as a primary diet. The critical importance for cow health of the transition to a beet-dominant diet is widely understood by farmers today, and the fact that illness on beet is a result of rumen acidosis dysfunction rather than any toxicity of the plant was the first major discovery that led to widespread beet grazing in New Zealand.

From this research, protocols for transition to ad libitum intakes of beet were developed and thoroughly validated early in the rise of beet hectares in New Zealand (Gibbs, 2011). These protocols are simple and completely effective in preventing acidosis if enacted. Appropriate transition is important for immediate cow health, and also for later management ease, as once cattle are transitioned to ad libitum beet intakes rumen acidosis is not possible, regardless of management procedures.

Nevertheless, each year there are management breakdowns that lead to rumen acidosis cases, and I often get asked what the optimal treatment protocols for these animals are. The unspoken assumption is that treatment is restoring rumen pH to 'normal'. This article outlines why this is not so, why alkalising agents and buffers are therefore commonly ineffective in treating these beet cases, and the approach I use instead.

BACKGROUND ON RUMEN PHYSIOLOGY

'Rumen acidosis' is poorly named because it is the clinical syndrome of a dysfunctional rumen environment in which pH is just one actor in the play – and not even the lead. By terming this syndrome acidosis, pH has been elevated to the primary role, yet 50 years of rumen research has consistently demonstrated that, while pH initiates the disorder, there are more important drivers of rumen function collapse that take over from it.

By strict definition, rumen acidosis should be the pH state due to the excess accumulation of short-chain fatty acids (SCFA: <6 carbon) produced by carbohydrate fermentation, and then

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eventually lactic acid, in the rumen digesta. But the disorder termed rumen acidosis is in most cases independent of pH within 12 hours of the initial pH insult to the rumen environment, with a constellation of physiological, microbiological and tissue dysfunctions driving the disease processes after this time. It is valuable to outline these changes in sequence.

The rumen environment is typically protected from this SCFA accumulation by the steady removal of these fermentation compounds via rumen epithelial transport to the rumen epithelial blood supply (most), by omasal orifice exit (some), by microbe use (even less), and to a limited extent by buffering or alkalising by various diet, digesta or salivary compounds.

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Acid removal is an extremely complex series of events maintained in a careful equilibrium, subject to extensive physiological adaptation in the rumen and in systemic circulation, and it typically takes more than a week to obtain steady state. Part of this complexity is the modulation of rumen removal by firm restrictions on plasma concentrations of water, acid, ammonia and other rumen metabolites. Production of SCFA by rumen microbes, however, is more straightforward, local and quickly achieved, so a large part of SCFA production control is actually via intake behaviours – that is, eating more in a given period is 'loading' the rumen, like shovelling coal into a boiler. So, when removal of SCFA doesn't keep up with production – after a change to a palatable, high-digestibility feed, for example – accumulation can occur.

As the capacity to buffer acids present in the rumen is both limited and subject to fine tuning over a period of several weeks before it is optimised, accumulation becomes acidosis when removal is significantly insufficient. Transition to beet-dominant diets in cattle at its core isn't about the rumen microbes; it is about learnt intake patterns (acid loading) and the rumen and renal physiology changes that enable acid removal from the rumen.

When SCFA accumulation beyond the limits to which the rumen is currently adapted is occurring, several other changes have already taken place. The most important of these is the increase in osmolarity that is in concert with uncontrolled SCFA accumulation. But the earliest and most obvious clinical sign is the change in rumen and abomasal motility. It is first reduced, then absent, and this effect is secondary to the increasing osmolarity and certain acids. Rumen epithelial blood flow is also progressively reduced because of the osmolarity and cellular dysfunction, and these motility and blood-flow changes contribute the most to the trajectory of the animal from that point forward. They also promptly produce declining calcaemia, the distinguishing and very early systemic feature of even mild beet acidosis.

Both high SCFA concentrations (>200mmol/L) and osmolarity (>300mOsm/kg) independently exert influence on microbial activity, reducing normal function in most groups and stimulating production of lactic acid from others, and stop much of the rumen epithelial transport. This is quickly represented in the redox shifts in the rumen. However, SCFA concentrations and osmolarity are not independent of each other, as SCFA and K^+ are the major drivers of osmolarity. Osmolarity, then, becomes dominant in sinking acidosis cases.

It is important to note that although rumen pH is poorly correlated to SCFA concentration (Sun and Gibbs, 2012) until the wheels really fall off, it does broadly represent accumulation. What pH alone does not represent is clinical acidosis. On the contrary, there are normal, healthy periods of temporary SCFA accumulation diurnally even in pasture-fed dairy cows – after evening intake being one example. In these, pH is below 5.5, while osmolarity, redox and rumen motility are within normal ranges, and both microbial function (for example, fibre digestion) and rumen function (for example, digesta passage rates) are strong. Therefore, while every advanced acidosis case will have low pH (4.3–5.4), not every pH under 5.5 is an acidosis case.

Rumen acidosis should, then, be understood as a rumen environment where osmolarity, redox and pH have exceeded boundaries of normal physiological function, have already affected rumen microbes, motility and tissue, and have altered systemic homeostasis.

So, when presented with a severe clinical rumen acidosis cow on beet 24 hours on from the killer meal, I really have about 2L of concentrated acids (in contrast, vinegar is about 5% of full concentration) in 100L of digesta, and more importantly a high and rising osmolarity. This osmolarity:

- has already shut down rumen and abomasal movement, epithelial transport of metabolites (for example, acids) and blood flow, guaranteeing that whatever I now put in the rumen will stay there
- has thrashed the rumen epithelium and the microbiota, promising endotoxins in the systemic circulation should the above be rapidly restored
- is passively drawing water away from the circulation with what little blood flow there is, with no means to replenish it (they won't drink, and at 330mOsm there is zero water flux out from the rumen)

» will keep going up until microbial function and digesta degradation truly cease (some hours after death).

So, all I have to do is 'buffer' this rumen to a higher pH, right, and everything is then okay?

FACT PISTOL

1. If I correct the pH with a static rumen, I don't correct the osmolarity. If the osmolarity is corrected in the rumen (removal), the pH automatically is corrected.

2. So in train-crash acidosis cases, even if I could buffer the SCFA content to a neutral pH (I can't), it won't help me. Why? Because nothing leaves the rumen now – it is static and blood flow has withered – the effect of my 'buffer' is that the osmolarity goes up in concert with how much 'buffer' I use. More buffer, greater osmolarity, more dead cows.

3. SCFA concentrations in severe cases are initially above 250mmol/L (normal is 60–120mmol/L), with an additional lactic acid (20–50+mmol/L; normal is <1mmol/L). On a molar basis, for a 100L rumen content, the $NaHCO_3$ required to raise the pH from 4.5 to above 5.5 is greater than a kilogram. The osmolarity in these train-crash rumens is often above 350mOsm/kg, and the addition of this huge molar dose of $NaHCO_3$ then raises this (via Na^+). Why is this considered an improvement?

4. In the published literature, there is no consistent demonstration of the efficacy of $NaHCO_3$ or MgO to robustly raise pH in rumen acidosis. The use of these 'buffers' or alkalisers is typically based on studies where normal (read: low osmolarity, normal redox) rumen contents were used, and even then the effect on pH is modest. They are used in practice today for clinical acidosis because it seemed logical 50 years ago when the idea and purpose was only to raise pH, and the concept of hyperosmolar damage was unknown.

PRACTICAL TREATMENT OPTIONS

Instead of adding osmoles to the static beet-rumen, hoping against physiology to improve the cow by raising rumen pH, I suggest these alternatives:

1. Wait it out.
2. Get it out.

The art is to decide which option to use.

WAIT IT OUT

In almost all cases where poor management of beet transition has led to significant clinical cases of acidosis, only five percent will present as severe, and another 5–10% as wobbly but walking. In a memorable circumstance many years ago, a management breakdown put a 1,000-cow mob over the wire at the 7–8kg dry matter (DM) intake stage of transition (the death zone). As unpleasant as the five percent casualty rate was, it became an important lesson seen repeatedly thereafter, that in every breakdown most cows in the herd will be normal and do not require either treatment or ration change. The rumen effect is not linear.

Unlike in bloat or nitrate poisoning in cows, most of the herd don't eat themselves to death, although a fraction will. Beet acidosis is not cumulative over eating days, and when they are first affected, cows stop or slow eating, so mild cases don't get worse and aren't aided by being removing from the crop.

Because acidosis only happens with incomplete transition, removing the herd from the crop when some cows are affected has impacts on the farmer in two ways. It increases the costs of wintering, as the supplement use increases and is maintained longer at high levels, and it slows the body condition score gains as the herd is reset to the start of transition to begin again after a break. Because 80% or more of the herd is unaffected, even with severe management breakdowns, and the affected ones won't eat and get worse, this is an extravagant waste that serves no purpose (Gibbs and Saldias, 2014).

So I walk off the clinically affected cows that can walk, and put them on pasture or silage. I don't automatically give parenteral calcium to these, but the odd few will display overt drunkenness and calcium typically corrects that. These will eat very little, which helps the rumen recovery, and won't go back and eat beet again any time soon. So it isn't critical to remove them from the crop, but doing so aids supervision of progress. Set the beet allocation for the remaining mob at 75% of yesterday (whatever it was), and keep it there for two or three days.

The prognosis for the walking cases when they have survived beyond 24 hours post-presentation is good, if no rumen drenches have been given. Giving these walking cases oral drenches of $NaHCO_3$ or MgO to 'buffer' the low pH can speed them

INSTEAD OF ADDING OSMOLES TO THE STATIC BEET RUMEN, HOPING AGAINST PHYSIOLOGY TO IMPROVE THE COW BY RAISING RUMEN PH, I SUGGEST THESE ALTERNATIVES: **WAIT IT OUT; GET IT OUT. THE ART IS TO DECIDE WHEN EITHER OPTION IS WARRANTED.**

into rumen collapse and increase mortality, and there are no cogent physiological arguments for why any of these should improve the rumen environment, except a small pH shift, which after a brief period has a limited effect.

I have encountered this repeatedly in consulting for management breakdowns, and I have known 50% mortality rates over the following week in groups of mildly affected cows who were drenched daily for three days with 500g of MgO. If they are left alone, less than one in 10 of these walking cases at 24 hours will then go down in the next 24–72 hours, although the prognosis for these late-affected cows who do is typically poor.

The cows who are down when presented (typically in the morning) get two bags of calcium borogluconate under the skin and nothing else, although non-steroidal anti-inflammatory drugs for pain relief are positive. Of the cows presented down, approximately half will get up soon after calcium administration. As a rule, if they don't rise by that afternoon, the prognosis is all but hopeless, and in my experience no attention or cocktail of medicants significantly changes that. It does change the farmer's bill. Postmortem of those within 12 hours of this stage typically shows extensive rumen epithelial detachment, beyond simple tissue impact, and I've come to the position that this rumen stage is irrecoverable. The odd cow gets up and recovers after a day or so down after no response to calcium, but this is rare. What I am persuaded of after years of dealing with these events is that rumen drenches of various buffers and alkalisers in these down cows are completely ineffective, and usually counterproductive.

Why does waiting it out work in moderate cases? Because rumen epithelial transport, omasal passage, and microbial use are not truly absent in these cases, and within 24–48 hours rumen removal of both osmoles and solid material improves the rumen environment.

GET IT OUT

Severe over-consumption of beet is 3kg DM or more than the cow ate yesterday, and, as a rule, if the whole mob has had >2kg average increase five percent will be down the next morning because a number will have well exceeded this. If the life and function of the affected cows are of unusual value to the farmer (for example, genetic elites), the rumen should be surgically emptied in any cow who doesn't get up by lunchtime after calcium has been given that morning. With rumen fistulate cattle, my own and other research trials have demonstrated that in early cases rumens can be emptied, have a little silage added with 10L of warm water, and this returns the cattle to clinically normal within days (the rumen takes considerably longer). Clearly, surgical cases have a higher risk, but it is the effective way to stop progressive damage.

Why not tube and lavage? Because beet pieces are often large, and overwhelmingly cows are managed into acidosis within the two-week transition, typically when supplement is more than half the diet. Nearly every beet acidosis postmortem will have a rumen full of supplement, as the amount of supplement plays no role at all in reducing acid loads from fermenting beet plant material. Getting this straw or silage out is impossible. In theory, warm water lavage should remove lots of osmoles and thereby improve the rumen. In practice, it is usually not possible.

What can't typically be judged with any certainty is the post-surgical prognosis of down cows who don't respond to calcium, in order to decide on surgical cases. I suspect the kilogram mass of beet in the rumen is the main factor, and this can't be seen or palpated. I have seen many who were clinically milder and brighter who went on to die without delay, while others sank, stabilised and recovered. So there needs to be a conversation with the farmer at this point. However, waiting a day or so for these – just to 'see' – has closed your window of intervention, as by that stage the rumen epithelium is usually part of the digesta.

In summary, targeting rumen pH correction is typically ineffective treatment in fodder-beet-farmer-management disorder of cows. Here is a checklist of treatment considerations:

- Don't remove the mob. Because most cows will not be affected, leave the mob on the beet, correct to 75% of yesterday's allocation, and proceed.
- Alkalisers like MgO or buffers like $NaHCO_3$ raise the rumen pH a little, and the more dangerous osmolarity a lot. This is counterproductive.
- Walking cases after 24 hours will usually recover with no treatment, and down cows who don't get up within eight hours of calcium usually won't, ever. Neither group benefits from rumen drenches.
- One intervention that can work in down cows is early rumenotomy. But be prepared for losses as some of these candidates look bright but are loaded with beet and sure to die anyway, and you can't easily pick them. VS

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