Introduction

It's a common phenomenon in cattle practice for vets to develop their own techniques and theories about diseases that they see commonly, but generally in isolation from other vets. This series of articles demonstrates clearly that there is more than one way to do things...

This installment of the “How I Treat” articles is on the topic of grain overload.

Grain overload – or more correctly carbohydrate overload or rumen acidosis is a not uncommon disease in bovine practice, and it is the author’s observation that every practice has a slightly different approach to it.

For the purposes of this article, as has become customary, and email was sent to a number of ACV members chosen at the whim of the author, asking them to describe their approach to the treatment of grain overload. A summary of the responses is provided here ...

Results

“I divide grain overload cases into 4 groups and treat them differently.

(1) Cow has been observed having access to grain – treat with a dose of 2 cups bicarb or mag-carb and monitor. (2) Cow has diarrhoea and grain in faeces – 2 cups powdered magnesium carbonate 2-3 times daily for 2-3 days. (3) Cow looks drunk or has systemic signs – treat as above but add broad spectrum antibiotics to keep liver abscesses at bay. (4) Cow is down – as above plus 5 litres of isotonic fluids plus a bottle of bicarbonate given IV through a garden spray unit, and stomach tube with 20 litres of fluids with 4 cups mag carb. Mag carb is an alkalisising agent where bicarb is a buffer – Mag Carb acts quickly but it’s possible to overdo it. I would never give hypertonic fluids to an animal that is dehydrated as there is no extracellular fluid to move it’s possible to overdo it. I would never give hypertonic fluids to an animal that is dehydrated as there is no extracellular fluid to move

Endotoxemia from the potentially compromised rumen, and to reduce the production/absorption of lactic acid. Hypertonic saline is a fantastic tool for rehydrating cattle. A couple of litres IV followed by 40 litres orally is arguably as efficacious as running a similar volume of isotonic fluid IV. Dave Van Metre once told me, if you give them plenty of sodium, chloride, and fluid, the cow will sort the rest out itself. When treating an animal with carbohydrate overload, they don’t need additional fluids as the build up of lactic acid has drawn plenty of fluid into the rumen. So… if an animal is recumbent, often with their head in their flank, we will bomb them with at least a litre of hypertonic saline, flunixin meglumine to manage endotoxin, Alamycin 300 to cover them systemically and hopefully reduce/prevent liver abscission. We will then drench them with 5 litres of paraffin followed by mixing up and drenching them with a bucket of 5 litres of water, 60 mls of penicillin (to knock back gram positives like Lactobacilli and Strep. bovis ), some electrolytes (not sure if necessary), and 500 grams of bicarb soda. We will then leave the owner with additional flunixin to treat the animal. We wonder, will a single dose of Metacam cover endotoxin release for a sustained period? Historically, our practice had used Epsom salts as a cathartic, however, we have abandoned its use as we perceived it could exacerbate dehydration? Hopefully, the discussion from others will address these two questions.”

“Old Fashioned medicine” series: Rumen acidosis treatment protocol: (1). IV 5-7L 5% sodium bicarbonate (50g/L =250g sodium bicarb/5L tap water) over 30min for 500kg cow; (2). 25L Water as oral drench (not in first 18 hours of acute acidosis); (3) MgO as oral drench 1g/kg body weight (i.e. 500g per 500kg cow) in 5-10L of warm water. Drench orally and massage rumen; (4). Ancillary drugs: High dose/long course procaine penicillin SQ for 5-7 days. Normal dose NSAID. High dose THIAMINE (20ml B calm for 500kg cow), 5-10 million iu intra ruminal benzyl penicillin. IF COW DOWN, LOW TEMPERATURE, HR 110-130; RUMEN pH < 5 = RUMENOTOMY.

“The initial diagnosis of carbohydrate overload is often straightforward due to history, the number of animals affected and the range of clinical signs. It can be a bit tricky sometimes when there is 1 or 2 affected and a denial by the producer that there was access to grain. Most of our carbohydrate overloads are grain, especially un-buffered. The circumstances under which carbohydrate overload occurs are many and varied, and sometimes you need to think outside the square. For example, if grain is buffered on farm, make sure the mixing is even and complete. I recently had an episode where the buffer was added just before the grain entered the feed auger in the dairy shed. The density of the buffer was greater than the crushed grain and most was deposited in the first few feeders in the dairy. Consequently the cows at the back of the shed were not getting buffer. The reason for the sporadic grain overloads in this herd took a bit of nutting out. Clinical signs vary from depressed; scouring animals to recumbency with nose into flank, groaning. ALWAYS check urine pH! A bright orange indicator is strong supportive evidence – if it’s alkaline explore alternative diagnoses. The rapidity of development of clinical signs gives a fair indication of prognosis. Severe clinical signs and recumbency within 24 hours of ingestion is grave.

Treatment is immediate and vigorous. Treatment consists of I/V fluids...
Carbohydrate overload, typically affecting one to a few animals can be caused by excessive intake of highly fermentable carbohydrates (any number of sources). I have seen this in animals accidentally fed 2 or 3 times the amount of grain/ pellets in the bail (eg going around the rotary multiple times) and quite commonly in herds fed byproducts – potatoes, bread, powdery rolled oats, cereal to name a few. With hobby farmers it is commonly from feeding too much bread and not enough “normal” feed. Diagnosis is made through history of access to a carbohydrate source and by examination of the animal and the rumen fluid. Rumen fluid of an acutely ill animal will have the following characteristics. On farm I will take rumen fluid via rumenocentesis and initially test rumen pH. With the severely affected clinical animals, rumen pH is often < 4.5 (anything < 5.0) is a problem and it looks milky with a distinct smell (lactic acid). This is usually enough to convince the owner. We don’t always have access to a microscope in the field, but when we look at a sample of rumen fluid for signs of - no active protozoa and a fast sedimentation rate. You can gram stain a sample but we don’t usually bother. If the animal dies, we will do a post-mortem exam looking for rumenitis with necrosis of papillae and a low pH of the rumen fluid. In chronic cases, liver abscessation may be present.

Grading/ prognosis - The heart rate in a cow with clinical ruminal acidosis can be used as a prognostic indicator. Cattle with a heart rate > 120 bpm generally have a grave prognosis, despite treatment. I also find that if the animal is down and unable to rise, the prognosis is very poor.

Treatment - Mild cases of acidosis - withhold concentrates and feed hay to stimulate saliva flow. Often I will also give oral antacids (we use sodium bicarb at 1g/kg BW but you can use others) and oral electrolyte solutions. With more severe cases, I give IV fluids (2 L hypertonic saline) as well as drenching with oral fluids (balanced electrolyte solutions not containing lactic acid). Text books describe lavaging the rumen with a wide bore stomach tube however I have never had much success with this technique. I will transfaunate the rumen with rumen fluid from a healthy animal (even 1 L is better than nothing). I also give the following supportive treatments - flunixin meglumine (1mg/kg), antihistamines (not always), thiamine (10mg/kg every 24 to 48 hours for up to three doses), antibiotics (usually oxytetracycline IM) and calcium/ magnesium solutions either IV or SC. Cattle that are successfully treated in the acute stage often show secondary problems such as laminitis. Prevention – pretty simple for the typical carbohydrate overload cow – either don’t feed what has caused the problem or feed in moderation. (prevention on a herd scale requires a whole discussion on the importance of fibre, feed additives etc – read the RAGFAR document)!

“Grain poisoning is mostly just that around here – too much grain rather than pellets. The cases we get are either sporadic dairy cows that presumably get leftovers that a previous cow didn’t eat. Often these are later calving cows where the farmer has already stepped up the level of grain in the lactating cows’ diet. Fortunately with the advent of transition feeding many more cows are at least partly accustomed to some carbohydrate in the diet and we don’t see as many cases of grain poisoning as we used to. On a couple of occasions I have had heifers/beef cows allowed ad lib access to grain in large feeders. Natural selection operates here to thin out the glut-

Most serious overload cases are often in extremis when seen. Bicarb is usually the first line in acute cases; supportive therapy including anti-inflammatories and antibiotics plus anything else off the shelf that may help is used where applicable and practical. In the past, Thiabenzole was a good addition to the treatment regime. Control of bloat is also necessary at times. Parnell’s Diarrhoea Powder or similar medication is useful in the aftermath. Subclinical overload (chronic acidosis) is a bigger worry. This is treated by correction of diet and access to adequate roughage. I have seen a house cow owned by the local baker with a bad case of “bread poisoning”. Also, a feedlot mixing its own rations changed from normal red sorghum to white sorghum without doing the necessary homework; the white sorghum had a higher soluble carbohydrate content and this simple change in ration caused a few death from acute acidosis In the 2002 drought, sorghum became unprocuurable for a local feed miller who was forced to include wheat in rations. Producers were advised and warned of the possible dangers. One client was feeding 700 weaners but had only two self feeder bins; he opened the bins too much "to allow every beast a fair go". The mortality rate was quite high.
tons! Diagnosis is usually a depressed cow with fairly profuse watery diarrhoea and lots of grain in it, acidic urine pH & acidic rumen fluid is the confirmation. If the cow is up and about I treat with oral bicarb 250g BID for 2 days, 60ml oral penicillin and oral propylene glycol. I often also start a systemic course of antibiotics just to keep liver abscesses at bay. If she is spending a lot of time down I would be keen to give IV fluids and usually use hypertonic bicarb followed by oral fluids containing the same mix as previously (bicarb, penicillin & propylene glycol). I would probably also give this cow some dextrose under the skin & toldidine for pain relief. At this point I start to talk about surgery (rumenotomy) as an option. If the cow is likely to have only had a second helping of grain I would probably proceed medically but if she could have had ad lib access I would be much more inclined to operate and empty out her rumen. If the cow is down and grunting I give a poor prognosis but if the farmer wants to take his chances and try I would proceed with rumenotomy, fluids, etc as for the previous cow. That's my 2 cents worth!

“We don’t see a lot of carbohydrate overload here as mostly pasture based systems but when it occurs it has usually been cattle getting into the grain put aside for fattening the sale bulls or dairy cows have broken into the feed shed or eaten the left over grain out of the auger bin. It has invariably been because “somebody” left a gate open so prevention would seem to be giving “somebody” a kick up the date and told not to do it again. Usually we are only dealing with 1 or 2 animals but sometimes more. I tend to do a bit of a reverse triage and treat the healthier looking ones first as the sicker ones can take up a fair bit of time and by that time the better looking ones may start to deteriorate. Diagnosis is usually based on history as the cows are often found with “their hand in the cookie Jar” . Clinical signs vary as it is never known how much may have been inadvertently consumed ( “I think there was only a few kilos”)and will vary with previous grain feeding levels. Animals that are thought to have indulged but are still up, good rumenal movement and cud chewing will get 500gm bicarb B.I.D , Histamill B.I.D. and trimidexel once if not pregnant as well as coverage with penicillin. The rational being to reverse the acidosis in the rumen and stabilise cell membranes elsewhere and to try and avoid the effects of rumenitis with subsequent liver abscessation. Animals that are still up, scouring and dehydrated but with good mucous membrane colour will also get about 20litres of electrolytes orally and/or fluids as well if valuable, preferably after a rumenotomy to try and remove as much grain as possible. Down animals with Diarrhoea will require all of above with guarded prognosis. If no diarrhoea, dehydrated and toxic membranes I don’t even try to treat them. Regular rechecks and ongoing treatment is required so depending on the owner’s budget and severity at presentation as to how far we try and go with these. Distance from clinic and how much drugs I have in my car at the time probably also dictates treatment to some extent. Animals that are up at presentation tend to go reasonably well. Down cows have guarded prognosis. I think I have killed a couple with hypertonic fluids but I do think dosing with cud ( or poop) may be of benefit in trying to re-establish bacteria. Take off grain and try to get them eating good quality hay. Eating is a good prognostic sign.”

“For me, treating downer cows with acute grain poisoning has been very unrewarding. If the cow is down and shooky, I routinely do a left flank approach for a rumenotomy and use about a 1 inch tube as a syphon. I then try to flush and syphon. When satisfied it is done, I then put a 250gm pack of sodium bicarb into a 20l bucket of water and pump it in as well. Cow then get hypertonic saline 2l flush the abdomen as best as possible with 5l hartmans, cover her with penicillin and prayer. If the cows are up and not showing signs of shock, is HR >90 and sunken in the eyes, I try rumen lavage with a stomach tube (but invariably it doesn’t work too well ) . IV hypertonic and again 250gm bicarb in 20l water If they are shaky and I can get them to a crush, I try and convince the client that a rumenotomy is their best bet. I work on heart rate a lot as a prognostic indicator, and rarely do I feel like I have unnecessarily treated a beast.”

“My only advice is with respect to ensuring stock can’t get access to grain dumps/silos. Most of the major problems I have seen is where stock get access to a silo by knocking the chute and getting lots of kilograms. Generally found dead nearby or where they get access to a lick feeder with the lick set too wide. Making sure they don’t have access to the paddock or care taken to ensure can’t be opened by an Houdini cattle!”

“We occasionally see individual cases of acidosis related to an overdose of feed from an accidental cause eg; animals getting into grain accidentally, presumed extra feed as one cow may not have eaten her grain. More commonly we see acidosis at the transition feeding stage, especially when as dry cows they are fed a diet including about 3 kg grain, grass and hay/silage, and then join the milking herd and are fed 6-8 kg of grain and grass as the diet. These cases are less acute than the classical grain overload, but are off feed and milk, have reduced appetite and diarrhoea, with some non-digested grain in it. A standard treatment would include Magnesium hydroxide given as a drench in 20 litres of water, penicillin injection twice daily for 3 days, multivitamin injections and anti-inflammatory for 3 days. More severe cases would be given 5 L of 5% bicarb IV and possibly calcium SQ. A prevention strategy for the transition cows is to have a group of freshly calved cows run separately from the herd for a few days prior to joining the main herd. An adjustment for fibre intake would be made, and Eskalin/Tylan use considered.”

“Fortunately a condition that I seem to diagnose a lot less than 15 years ago. Tend to see it early in the post-partum period when tran- sillon diet has been less than optimal (or non-existent). Otherwise see an odd case after inadvertent access to higher levels of concentrate. A rough rule of thumb for me is: If the cow is standing she is treatable, if she is recumbent then prognosis is poor. After a clinical exam, I simply put an 18 gauge 1 1/2 inch needle on a 3ml syringe and do a ruminal paracentesis at the lowest part of the left paralumbar fossa. I rely on the number that comes up my pH paper to confirm a diagnosis. Any pH around 5 sees me treat with: 1/2kg bicarb and 1/2kg mag oxide orally; 500ml CBG subcutaneous (500ml IV if cow is staggery); 10ml histamill IM; 25ml penicillin IM; 25ml penicillin orally (I did once have a final year German vet student query this on the basis that the penicillin would not work in an acidic environment - his pharmacology may have been a bit better than mine); No access to water for 12 hours’. Provide only fibre of any sort to eat. If the rumen pH is 4 or lower I would still treat the same but with 2L hypertonic as well. Prognosis in this case is poor at best. Most farmers know the importance of quality fibre in the diet, but some cows can be susceptible to subclinical acidosis due to the fact that they simply don’t eat the roughage available. Any herd level issue can be suspected if the butterfat has lowered over a period of time.”

“I think my grain overload treatment is a fairly simple and not really scientific. I do carry pH paper in my sick cow box and find getting a rumen pH sample fairly helpful, often the farmer is sceptical. I re- member treating/necropsing some rams that a farmer had poisoned when he swapped from oats to pellets in his blend. Many had died and most were lame. I couldn’t convince him that it was grain over- load he wanted me to test the pellets for ‘poison’. We calculated how many pellets the rams had, I can’t remember exactly how much it was but it was heaps. I find self feeders are very effective at allowing grain overload, I know they can be used well, but they are quite problematic, especially if they run out over the weekend. Most times I have seen lots of sick/dead cattle/sheep from grain overload it has been when the stock were thought to be used to them and then over-
“Spuds are a common cause in our neck of the woods. I mix 84g bicarb in 1l saline to give an 8.4% hypertonic solution (I read somewhere that these cows were hypernatraemic so thought hypertonic bicarb would be better) I give 1-2l of this IV depending on size of animal and severity, followed by stomach tube with mag hydroxide or calcium carbonate mixed with water. I try to only give 10l rather than 20 as rumen already sequestered a large amount of fluid. I then get the farmer to push on one side of the rumen and myself on the other to mix it all up. Then treat with IV trim-sulph, IV flunixin, 50ml B1, 4 in 1 SC and anthistamines. Farmer to continue with trim-sulph for 5 days, propylene glycol BID if still off feed and gastric stimulants for 1-2 days, 4 in 1 SC if slow, B vits for 3 days (b1 10-20mls) and sometimes I give a shot if AD&E because I also read somewhere it might be useful for mucosal healing/growth. My anecdotal non statistical assessment is I win more often using hypertonic bicarb that I did with hypertonic saline!”

“Treatment generally consists of oral fluids with a good handful of bicarb, iv fluids (hyper- +/- isotonc and always some bicarb iv- dose for this is to fill a 20mL syringe up to the 15mL mark and dissolve that in 1L, and give slowly iv), penicillin and ongoing nsaids (for risk of laminitis). Feed hay afterwards.”

“Regarding the various types of carb overload, I think I have seen them all…. From a group of 8 heifers that ate an entire Pantech truckload of stale McDonalds buns which despite intensive treatment had a 100% mortality rate to a case where a very valuable cow ate about 50kg block of waste chocolate which had melted so that instead of a little lick, she drank the whole lot (and died) , to a herd of beef cows that were turned loose into a frosted wheat crop that obviously had more grain than anticipated again with a virtually 100% mortality rate and a herd of dairy cows fed about 3 tonnes of rotting kiwifruit that resulted in about 35 recumbent cows at 6pm on a Friday night!!! However, by far and away, the main culprit that I see is the self feeder! Most of the significant “outbreaks” that I have seen have been in growing heifers and has resulted from a poorly adjusted self feeder that just runs grain, or in cases where people change from wheat to Triticale to Barley etc without adjusting for the different sized grains or even when farmers get onto some “cheap” pinched or broken grain, which flows too easily and they get an absolute gutful. Usually, the biggest and best heifers in the mob are the worst affected, as they are the greediest/most dominant. Especially in these cases, there is often several days elapsed from the actual engorgement to the symptoms being noticed, and many cases are severely affected by the time we are called out. Depending on the numbers and the value of the heifers determines the response. My treatment can be as simple as dosing with oral bicarb drench, 50 g Eskalin premix pellets mixed into a slurry PO (which we get formulated for us so that there is 250mg VM in 50g pellets), Histamil inj, Metacam inj and a shot of LAP to prevent liver abscesses. For valuable heifers, treatment includes large volume isotonic bicarb infusions (add 130g bicarb soda to a 10L bottle of filtered water from the IGA and give IV), oral Eskalin, Magnesium oxide and for bicarb initially, non responders get ruminotomy, cud transfer, probiotics etc. We have treated as many as 20 or 30 heifers that were found recumbent/moribund surgically with about a 1/3 success rate. Once rehydrated they are also given Metacam, histamil and B-vitamins/aminolyte. The nice thing about the 10L filtered water bottles is that you can easily treat them like 10X 1L units so I may add 5X 9g NaCl and 5X 13g NaHCO3 to make a 0.45% NaCl and 0.65% Bicarb replacement isotonic fluid to follow up a dose of Hypertonic saline at a dose of about 100ml per 50Kg body weight to treat the shock. Lately, I have been covering these cases with Excede rather than LAP.”

“I treat them with 500g MgO orally, penicillin at label dose rates for 3-5 days. B12 and if really crook 5L IV fluids with 42g/L bicarbonate and 45gL NaCl.”

“Thought you might be interested in my sheep protocol!
(1) Mob Treatment: Move off paddock ASAP; Nil water till 24hrs; Exercise mob every 1-2 hours; Provide access to hay.
(2) Sheep that are obviously lethargic and or gut distended: 1gram of Bicarb per kg of Sheep live wt (oral); 500 gram made up to one litre = 40 ml per 20 kg weaner; 1 ml /10 kg of Alamyacin LA 300 intra-muscular inject.
(3) Sheep that are down or cannot travel: Seek urgent veterinary treatment or prompt euthanasia; Will only survive with intensive intravenous fluid therapy (100ml of 5% Bicarb per 10 kg) +/- surgical draining of rumen both of which need a veterinarian. Even with this the chance of recovery is poor.”

Thanks to (in no particular order): Liz Bramley, David Rendell, Debbie Twiss, Enoch Bergman, Ian Gill, Stuart Barber, Andrew Jemmeson, Joe Brady, Phil Sharman, Chris Shirley, Dave Buckle, Amanda Macdonald, Alan Guilfoyle, Liz Pryor, Rob Bonanno, Bruno Ros, Steph Bullen, Dave Beggs, Jakob Malmo, Mic Dhar.

This publication is available free from the AVA website – google “AVA RAGFAR ACIDOSIS”. It is a great textbook on acidosis management.