Clinical Problems Associated with Starvation in Western Beef Herds

Marie S. Bulgin, *D.V.M.*
University of Idaho
Caldwell Veterinary Teaching Center
WOI Regional Program in Veterinary Medicine
Route 8, Box 267
Caldwell, Idaho 83605

The term "starvation", as I refer to it, may cover several situations:

(1) Complete deprivation of food is probably rarely seen by the practitioner and is usually due to severe weather conditions—floods, blizzards, drought, etc. (2) Simple slow starvation (protein and energy deficiency) is seen far more often, particularly in cold climates during late winter and early spring months when animal's nutritional requirements are going up due to late gestation, lactation, and/or cold stress, often in the face of reduced feed. (3) Partial starvation, is the deficiencies of one or more essential nutrient. Other nutrients may be substandard, minimal or adequate. This condition is often confused by the presence of concurrent infectious disease problems. The last situation is one which western bovine practitioners see often but may not recognize.

Beef cow management in the Western United States is influenced enormously by the availability of public lands for grazing. Most large commercial livestock breeders make use of these lands due to the relatively low cost of feed. Producers pay per animal unit month (AUM) used. An animal unit is a mature animal with an offspring under 6 months of age and when left on the range for 6 months would represent 6 AUMs. To make the best use of public lands, the cow needs to be turned out when the calf is as young as possible and left out as long as possible, depending upon the area and the weather conditions. The time livestock spends on public lands varies. In the mountains this may be June through October and lower elevations March through November.

Ranchers living in higher elevations generally bring cows onto private lands in October to graze hay or grain stubble until it is unaccessable due to snow. Feeding hay usually begins in November or early December. Hay raised in these mountainous areas is often wild, brome, bluegrass or timothy grass, as alfalfa doesn't grow well in these climates.

In lower elevations when cattle are taken off public land, they may be left on private desert dry lands, some of which are improved, (sagebrush removed and planted to crested-wheat grass). Cattle may also be grazed on crop aftermath (corn stover, bean straw, grain stubble, etc.). If the weather remains mild the cattle are left on this type of grazing until calving is imminent. Animals then are usually brought in and fed alfalfa hay. If weather permits, cows are often still expected to graze corn, hay or grain stubble with very little supplementation.

In these situations poor quality roughage and cold weather ensures energy as well as protein deficiencies. Mineral deficiencies can be a further complication. Phosphorous, selenium, zinc and copper deficiency are commonly found either together or separately, depending upon the area. The following are several such cases seen by the Caldwell Veterinary Teaching Center (CVTC).

CVTC is part of the Washington-Oregon-Idaho (WOI) veterinary teaching endeavor; a portion of Idaho's contribution to the program. CVTC is a food animal clinic based in Caldwell (SW Idaho), which provides Washington State University senior students with their food animal experience. It operates on referral only and specializes in herd or flock health.

Case 1. In southeastern Idaho (where temperatures commonly dip to 20-30° below 0 in the winter) a beef producer had lost 68 out of 520 head of cows. The signs began with muscular weakness, developed into dorsal recumbancy and although the cows were still alert and appetites were good at this point, they died within 1-3 weeks.

The cows had come off the range in poor condition in late October and gone onto "fall pasture" (grain and alfalfa stubble). After losing 17 head, the owner began supplementing with 25 lbs/hd of 1/3 barley straw and 2/3 hay (half alfalfa-half grass). Deaths continued. On the 1st of January he added 30% non-urea protein and low level terramycin in a molasses based liquid supplement at the rate of 2% /head. Cows were due to calve in April and many apparently were open.

We were consulted in this case April 4 when the owner presented a live animal at our clinic. The owner's statement read, "Three year old cow in good condition until 3 weeks ago when she started losing weight. She's on good feed (25 lbs of hay) and still has good appetite. She's with her second calf which is due shortly. Cow looks bad and will go down in a week or so, then die in another week or so."

The owner also brought 10 blood samples from other "affected cows" and analyses of both his grass and alfalfa hay. He had purchased 2 year old hay which had been sprayed with Disiptan, a herbicide. Disiptan was suspected

to have caused problems in dairy cows in the area, so he was able to get it cheap. It was the owner's belief that the Disiptan was causing the deaths.

Calves of cows which calved were born strong and healthy, according to the owner, and of normal size. However, there were a higher percentage of open cows than were expected (15-20%). The bulls had been cultured earlier for Trichomonas and found negative.

Physical examination of the clinic cow was unremarkable; temperature 101[4], bright-alert, with a voracious appetite, ruminating normally, feces were normal in consistency but she did defecate and urinate frequently. Her mucous membranes were normal and pregnancy status was open. She did appear slightly dehydrated, hair was dry and skin was scaly.

Blood work indicated only a low grade anemia, dehydration, a neutrophil/lymphocyte reversal and a high urine pH. Clinical chemistries showed only a low magnesium and BUN (Table 1). In spite of the owners prediction that she would "go down" within the week, she did not. She ate everything put in front of her and appeared to improve, almost immediately.

TABLE 1. Results of clinical laboratory tests on cow in Clinic — Case No. 1

WBC	10.2 X $10^3/mm^3$	Neut.	68%	6,936
Hgb	13 g/dl	Lymph.	30	3,060
Hct	32%	Mono.	2	204
Pl. Prot.	8.2 g/dl	Urine pH	9.0	
Glucose	102	Protein	8.5	
BUN	8.1	Albumin	3.9	
Calcium	9.8	Magnesium	1.6	
Phosphorus	5.2	Selenium	0.15 ppm	

In instances such as this one, a referral clinic is definitely at a disadvantage as the herd this cow represented was a 6 hour drive away. We depend upon the owner for an accurate history which is very often colored by the owner's prejudices, in this case, his suspicion of the hay and his judgement that the feed the animals were getting was adequate. From his own admission the cows had come in off the range in "poor condition." His own veterinarian had suggested the addition of the protein supplement and he felt that the nutrition was more than adequate. In spite of our suspicions from the cow's condition and the hay analysis, debilitating diseases had to be ruled out. Since this was a herd problem such problems as chronic BVD, parasitism, and deficiencies other than energy (mineral) had to be considered. In this case (Table 2) calcium and selenium were in the normal range; phosphorous and magnesium were slightly low and the serology was interpreted as negative for active viral problems. The analysis of the diet, however, was more rewarding.

Dry range grasses and crested-wheat grass (past ripe) are almost devoid in protein[1] (Table 3). However, since the cows were not in the last 3 months of pregnancy their nutrient requirements were not very high (Table 4). New growth grasses due to fall rains often supplement the less palatable feed. However, this particular fall had been rather dry and the cows reportedly had been in poor condition. According to calculations these cows needed approximately 1.4 kg of TDN and 0.1 Kg digestible protein more per day.[1]

TABLE 2. Serum results of 9 animals showing signs of disease — Case No. 1

CLINICAL CHEMISTRIES

	1	2	3	4	5	6	7	8	9	Normals
Glucose*	35	63	48	38	55	36	28	4.6	41	55-75
BUN	19	9	15	11	14	11	17	14	11	20-30
Calcium	8.5	8.7	8.1	9.4	8.3	9.3	8.8	8.2	7.9	9.7-12.5
Phosphorus	3.1	6.0	5.5	5.5	7.4	5.3	4.6	7.4	6.7	5.6- 6.5
Protein	7.4	7.8	7.3	7.8	6.5	7.4	7.1	7.8	5.9	6.7- 7.5
Albumin	3.0	3.7	2.9	3.6	2.8	2.7	3.4	3.4	2.4	3.1- 3.5
Magnesium	1.6	1.7	1.6	1.8	1.6	1.7	2.1	1.5	1.7	1.8- 2.3

* Serum separated after sitting 12 hours on the cells at 4°C.

TABLE 3.

CRESTED WHEAT GRASS

	DM %	TDN %	Protein %	D.P. %
Early Vegetative	30.8	67	23.6	18.0
Early Bloom	42.5	58	11.0	7.2
Full Bloom	50.0	55	9.8	6.2
Mature	60.0	52	5.7	2.7
Post Ripe	80.0	49	3.1	0.5

BRED IN JUNE—JULY
DIET AUGUST—OCTOBER
Crested Wheat Grass—Dried Range Grasses

	Needed	Got	
DM	7.2	5	—1.8 Kg
CP	0.42	0.21	—
DP	0.20	0.09	—0.1 Kg
TDN	3.9	2.52	—1.4 Kg

TABLE 4.

HAY ANALYSIS

	Grass	Average Bluegrass
Dry Matter (DM)	90.53	93.4
Crude Fiber (CF)	34.91	28.9
Crude Protein (CP)	9.49	11.6
Digestible Protein (DP)	7.45	7.0
TDN	47.93	61.0

	Alfalfa	Average (M.B.)
Dry Matter (DM)	83.82	89%
Crude Fiber (CF)	34.25	30.9%
Crude Protein (CP)	14.08	17.1%
Digestible Protein (DP)	10.08	12.1%
TDN	43.54	55.0%

An important consideration when analyzing diets is feed intake. Obviously an animal cannot take in more food than her rumen can hold. The poorer quality the feed, the longer the processing time—vastly reducing through-put. Thus as crude fiber and legnins go up, intake goes down, no matter how hungry the cow. The rule of thumb is shown on this chart.

FORAGE QUALITY	INTAKE DMB
Poor	.8-1.5% body weight
Medium	1.5-2.0% body weight
High	2.0-2.5% body weight

Therefore you may feed a 1100 lb. cow 25 lbs. of straw which will calculate to be 21 lbs. DMB but she can only eat 8.8 lbs. DMB or 10 lbs. as fed. The same is true when she is foraging on her own except she probably expends more energy looking for it. So this particular animal came off the range deficient in both energy and protein. *(Table 3)* She was then given a diet of 1/3 straw, 1/3 poor quality alfalfa hay and 1/3 average quality grass hay (hay analysis—Table 4). All 3 deficient in Vit A, D & E, we can assume, as it was 2 year old hay. The cows are now going into their 3rd trimester, nutrient requirements are going up and they are still unable to eat but 13.86 lbs. DMB and 15 lbs. as fed of the feed put in front of them *(Table 5)*. Producers will argue, "But Doc, they weren't even cleanin' everything up." In January even with supplement the energy was still not quite adequate *(Table 6)*, lacking 0.3 kg of TDN per cow per day. Cows continued dying, succumbing as the energy deficiency added up *(Table 7)*.

TABLE 5

DIET NOVEMBER—DECEMBER

	Needed	Straw	Alfalfa	Grass	Total	
DM	8.6 Kg	1.3	2.5	2.5	6.3	−2.3
TP	0.51 Kg	0.05	0.35	0.24	0.64	
DP	0.24 kg	0.01	0.25	0.19	0.45	+0.2
TDN	4.5 Kg	0.53	1.09	1.20	2.83	−1.7

	Straw	Alfalfa	Grass	Total
As Fed	8.3	8.3	8.3	25 lbs.
	3.8	3.8	3.8	11.4 Kg
DM	7.3	7.0	7.5	21.8 lb.
	3.4	3.2	3.4	10.0 Kg
Actually	2.9	5.5	5.5	13.9 lb.
Ate (DM)	1.3	2.5	2.5	6.3 Kg

Although the history doesn t reflect it, it's likely that the cows which went down and died were pregnant, since the energy drain on these animals was so much greater. The reason our clinic case responded so quickly is probably because she wasn't pregnant. We are familiar with pregnancy toxemia or ketosis in the sheep and goat, a disease

TABLE 6.

DIET JANUARY 1—APRIL

	Alfalfa	Grass	P.S.*	Total
As Fed	12.5	12.5	2	27 lbs.
	5.68	5.68	0.91	12.3 Kg
DM	10.5	11.3	1.54	23.3 lbs.
	4.8	5.1	0.7	10.6 Kg
Actually	8.9	8.9	1.54	19.3 Kg
Ate (DM)	4.0	4.0	0.7	8.7 lbs.

* Molasses & Protein Supplement

	Needed	Got				
		Alfalfa	Grass	P.S.	Total	
DM	8.6 Kg	4.0	4.0	0.7	8.7	+0.1
CP	0.51 Kg	0.6	0.4	0.2	1.2	
P	0.24 Kg	0.4	0.3	0.2	0.9	+0.67
TDN	4.5 Kg	1.7	1.9	0.6	4.2	−0.3

TABLE 7.

ACCRUED DEFICIENCIES

	Days	Protein		Energy	
Aug.-Oct.	92	− 0.1	− 9.2	− 1.4	− 129
Nov.-Dec.	61	+ 0.2	+ 12.2	− 1.7	− 104
Jan.-April	120	+ 0.7	+ 84	− 0.3	− 36
			+ 87 Kg		− 269 Kg

commonly associated with multiple pregnancies. The ratio of fetal weight to maternal weight is higher by at least a factor of 10 in the smaller ruminants. Thus when the rumen volume is encroached upon by fetal mass, the diet has to be adequate as the smaller ruminant has comparatively little body reserves to call upon. The cow on the other hand can take much more dietary abuse before she will lay down and quit. Like the doe or ewe, however, once she goes down, the prognosis is grave. Clinical tests are not particularly helpful except to rule out other problems.[2] Unlike the ewe, serum glucose is most often normal even in the downer. Treatment of the recumbent starvation case is generally unrewarding probably for the same reason treating ketosis in the ewe is, (whatever they are). The metabolic pathways put into motion when the ruminant hits the end of her reserves are apparently irreversible.

This case represented a severe problem which fortunately is not a common one. This next case represents a far more common one.

Case 2: A rancher from eastern Oregon was referred December, 1981, for a chronic problem with conjunctivitis and diarrhea in hereford feeder calves 6 months and older. Several of 650 animals had died. Several calves had diarrhea and two of those were brought to the CVTC clinic. Many (150) of the others had "pink eye", drooling and some calves were lame. The calves had been previously vaccinated

(November 12) with Syntex IBR, BVD vaccine I.M. BVD vaccine had not been used previously in this herd. The veterinarian suspected a possible BVD vaccine break.

Necropsy of one of the calves revealed severe widespread epithelitis of oral cavity and entire digestive tract which resembled mucosal disease. Isolation of a virus with properties consistent with BVD virus was made. The gross and microscopic changes seen in the second calf were essentially the same. BVD virus was isolated. A consumer complaint was filed with Syntex.

The following month, January, 1982, 9 head of 8-9 month old calves were admitted to the clinic with "pink eye." Conjunctival scrapings were taken and virus with properties consistent with IBR were isolated. It was recommended at that time to use the Jen Sal intranasal IBR vaccine in the calves instilling a drop in the conjunctiva, also.

January, 1984 this client again was referred for "pink eye problems." Although they had not been referred in 1983, this outbreak of "winter pink eye" made the third one in three consecutive years. This particular year calves had been weaned in November and vaccinated with IBR PI_3 and BVD (Norden) two weeks prior to that time. Eight yearling heifers weighing approximately 500 lbs. were presented at the CVTC clinic for examination. They were thin, with shaggy, dull hair coats. These represented the most acute cases in a group of 400, 100 of which were affected.

Each individual was examined. Most had nasal and occular discharges and several were coughing. Blood for CBC, mineral screen, clinical chemistries, viral serology, and feces for parasites and virology isolation were taken. Conjunctival smears were made for Chlamydia, and swab for bacterial and viral isolations were done.

Examination revealed increased respiratory noises in some, erosion of nasal mucosa in some and rectal temperatures ranging from 103° to 106 in all. Eye lesions ranged from healed central scars, or central corneal ulcers with edema to deep vascularization of the limbus.

Laboratory tests revealed that the heifers were slightly anemic, hypoalbuminemic, hypophosphoremic, and had low serum magnesium levels. *(Table 8) Pasturella multocida, Hemophilus* sp. and *Neisseria* had been isolated from eyes. No viruses were found.

The history revealed that:

(1) Heifers' weight had averaged a loss of 1 lb. apiece since weaning in October.
(2) Steers were not affected by this disease problem even though they were kept in an adjacent pen.
(3) Blood tests taken earlier by the referring veterinarian had revealed that calves were deficient in selenium and copper.
(4) Heifers were being fed meadow or wild hay. Steers were being fed purchased alfalfa hay.
(5) Steers had averaged a gain of a pound or more a day.

Historically the animals had come off the range and were fed wild hay. The feeding of purchased alfalfa hay was a new introduction made by the son; he had not been able to justify to his father feeding it to the replacement heifers. They did have trouble getting heifers to breeding size by the following season and the heifers were calved 3-4 months later than the older animals. A 75-80% calving rate was expected.

TABLE 8

CASE 84-85

Calf	1	2	3	4	5	6	7	8	Normals
Hemoglobin	9.4	12.8	10.9	10	11.8	8.3	11	10.9	11-15
Plasma Protein	8.1	6.3	7.3	7.0	8.0	7.1	7.4	7.4	7-9
Fibrinogen	200	100	200	600	300	100	300	300	100-800
Neutrophil	20	27	39	67	36	25	30	30	15-45
Lymphocyte	68	56	55	29	57	62	62	52	45-75
Albumin	2.2	2.3	2.9	1.8	2.6	1.9	2.8	2.2	3-3.7
Calcium	11.0	10.7	11.3	10.8	10.3	8.4	10.3	10.9	9.7-12
Phosphorus	3.7	4.4	3.6	3.9	3.0	3.4	4.3	4.5	5.6-6.5
Magnesium	1.27	1.26	1.44	1.5	1.28	1.25	1.03	1.54	1.8-2.3

Protein and selenium supplementation was attempted using urea, molasses and salt in lick tanks. We were unable to ascertain how much of this supplement was being fed. Evaluation of their diet did indicate that protein supplementation was indicated *(Table 9).* In fact, these heifers needed protein, energy and phosphorus supplementation if they were expected to gain any weight.

Studies with laboratory animals have clearly established the detrimental effects of protein deprivation on resistance to infectious disease. Dogs depleted of protein had greatly reduced capacity to elaborate specific antibodies and were far more susceptible to infection.[3] Mice fed a low protein diet resulted in an all over impairment of phagocytosis.[3] In addition prenatal as well as perinatal malnutrition result in deranged metabolic and clinical response to infectious agents. In experimental animals deficiencies of calories or protein during gestation reduces size, lymphocyte numbers, DNA and protein synthesis in lymphoid organs.[3]

These animals presented for an infectious disease process exemplified a case in which other factors were far more important than an infectious agent. The facts that these calves had actually lost weight over a 3 month period and that the adjacent pen of animals on a better nutritional plane had no or only few cases of disease were major clues.

TABLE 9

GROWING HEIFER 550 LBS.

	Need To		Meadow Hay	Heifers Ate
	Gain 0 lbs/day	Gain 1.5 lbs/day		
DM			92.9%	*6.25 kg
T.P.	.35 kg	.62 kg	9.1%	.57 kg
D. Pr.	.20 kg	.38 kg	2.9%	.18 kg
TDN	2.3 kg	4.1 kg	51.0%	3.2 g
Phosphorus	7 g	15 g	.17%	8.9 g

* 14.8 lbs as fed

Furthermore this had been a chronic problem experienced every year. Note, however, we had seen this man's calves twice before and had not recognized the basic problem.

The referring veterinarian knew the nutritional status was poor and was beginning to suspect the relationship of disease to nutritional deficiency, thus recommending supplementation, but he didn't complete the job. He hadn't determined how much they really needed.

Further, since undernutrition impairs the immune response, the efficacy of prophylactic immunization can be seriously impaired. Agglutination titers and production of secretory IgA have been shown to be reduced in individuals suffering from malnutrition.[3] Historically nutrition was not considered to be in the realm of the veterinarian's professional duties, but for the food animal practitioner, it's difficult to separate (as in this case) nutrition from disease. Perhaps the toughest part of this sort of problem is convincing the owner. The owner of the cows in the first case refused to believe that his problem was lack of nutrition. No doubt it's difficult to accept that one is responsible for starving his animals to death. In the second case the problem consisted of convincing the owner that increasing energy and protein would be economically advantageous.

The particular points that these cases help to emphasize are:

1. Clinical chemistries generally are not helpful in diagnosis of simple starvation cases[2] as shown by our Case #1. Even when animals are in recumbancy, glucose levels are generally normal.[2] Albumin and hemoglobin may be low, particularly if dehydration has been corrected, but this is also seen in chronic disease.

Mineral deficiencies should be ruled out as they may be a complication, at least in our area. Phosphorus, selenium, copper and zinc deficiencies (particularly in young animals) are seen quite often.

Parasitism may or may not be involved and needs to be ruled out also. The presence of infectious disease such as in Case #2, does not rule out nutritional disease and should, on occasion, prompt a practitioner to look further—particularly where vaccine breaks are suspected or an owner is experiencing far more disease problems than he should. Live modified BVD vaccine has a long suspicious history of vaccine breaks. It is well known that it indeed is immunosuppressive particularly in "stressed" animals. Its use should not be recommended in less than "normal" animals.

2. On the subjects of diets—for beef animals, there's no great trick to checking rations. Although in both cases here the animals were protein deficient, the great majority of malnutrition is due to deficiency of energy. In spite of increasing needs for protein during the last trimester and lactation, the cow requires only 10% of the ration as protein.[1]

Protein blocks are an expensive way to provide protein. To provide the necessary protein for a cow on crested-wheat pastures late in the season, one would need to feed 1 block per 12 cows per day. If allowed free access to protein blocks, a cow will generally consume 2-4 lbs. of block per day, so 1 block should last her approximately 12 days. That may provide her, depending on the block, with 0.24-0.8 lbs. of protein a day (Table 10) and cost approximately 40-60 cents/day/cow. If cows do not require that much protein (Recommended feeding is 1-1.5 lbs. per day) then the boss cows eat their 2-4 lbs. and those further down the pecking order don't get any.

TABLE 10

PROTEIN BLOCKS

Block	Urea	Crude Protein	Lbs. Protein	Lbs. Protein + Urea
20%	6.6%	13.4%	6.0 (.12/lb)	.9 (.18/lb)
36%	20.0%	16.0%	7.2 (.14/lb)	5.3 (.2/lb)

FEEDING BLOCKS
Recommended 1-1½ lbs. of Block/Cow/Day

	Recommended	Free Choice*
20% Block-Protein	.12-.18	.24-.48 lbs. Protein
Protein + Urea	.18-.27	.36-.72 lbs. Protein
36% Block-Protein	.14-.21	.28-.56 lbs. Protein
Protein + Urea	.2 - .3	.4 - .8 lbs. Protein
Cow Needs	.92-1.12 lbs Total Protein	

*Cow will eat 2-4 lbs of block free choice
Cost at free choice $.20-.30/cow/day

Urea takes energy to convert to protein.[4] If a ration is energy deficient, such as that in Case 2, urea is of no value. Urea is particularly helpful with high energy, protein deficient diets such as corn. Non Protein Nitrogen (NPN) plus energy nutrients form microbial protein. The utilization of NPN is dependent upon 1) the amount of Nitrogen in the feed that is reduced to NH_3 in the rumen and 2) the amount of digestive nutrients available in the rumen. The relationship between NH_3 and TDN in the rumen is expressed as the urea fermentation potential (UFP). If the available energy in the rumen exceeds the amount required to convert the entire rumen ammonia pool to microbial protein, the UFP is positive (Table 11). UFP is expressed as amount of urea that can be utilized per Kg of material fed. It can be useful when feeding crested-wheat or grain stubble.

TABLE 11

UREA FERMENTATION POTENTIAL

Feed	TDN	Protein	UFP G/kg DM
Alfalfa	61	19.3	– 42.8
Barley	83	13.0	– 1.6
Straw	41	4.1	+ 4.3
Corn	91	10.0	+ 11.8
Beet Pulp	72	10.0	– 1.7
Molasses (Beet)	89	8.7	+ 3.7
Molasses (Cane)	91	4.3	+ 20.1
Corn Stover	59	5.9	+ 6.2

Through-put must be taken into consideration when calculating energy with low quality forages, however, as in Case #1 where the animals were fed 25 lbs. of forage but only utilized 13 lbs.

A nutritionist I know on our staff, claims that the germ theory is after all just a theory. He maintains that most large animal diseases are nutritional. As one becomes more involved with cases of this type and more aware of nutrition, you will find that it does play a large part in the well-being of our large animals. As veterinarians interested in herd health and preventive medicine, we are going to have to become more familiar with basic principles of nutrition.

References

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Questions & Answers:

Question: Have there been treatments that work in the field?

Answer: Yes, if they sedate the calves with something like acepromazine. That's what has been used.

Question: (inaudible)

Answer: Well, I'll cover that. The general summary of the discussion was, the gentleman has had problems with his own cattle, but they respond so well nutrition wise with ammoniated product, that he went ahead and repeated it and is doing it again this year. I think that's the basis. Another point that he brought out had to do with temperature. And certainly temperature and pressure and that's one of the things that I had down here that I was going to go through until 5:00 began to sneak up on us and I didn't want to get anybody in trouble. But certainly the molasses work. Very definitely they had elevated heat and temperature, and, in fact, I received a letter from a gentleman in Australia this week inquiring about ammoniated hay problems. And he, by using high temperature and pressure, and up to 90°C, just utilizing straw, produced humidizols in straw. So I think the higher the temperature the more likely we are to get into trouble to produce compounds and also the higher the pressure. That's very definitely correct. You just do not want to let high moisture hay start through a heat before you ammoniate, because this is going to increase the potential for humidizole content. In fact, I talked with some of the people that did some of the molasses work and they said not to worry about it. With the kind of temperature and pressure that you're doing that dry hay, you're not going to produce these problems. There may be more to this story that we really don't know about today than what I feel we might know 6 months from now.

Question: How about the loss of ammonia?

Answer: They're probably losing most of their ammonia to the atmosphere. They'll greatly improve the ammoniation process and not lose near as much expensive ammonia to the atmosphere if they'll put it under a tarp. Certainly they should put it under a plastic tarp.

Question: If the hay is ammoniated does it last for a long time?

Answer: Yes, it does last for a long time because this is not just a traction kind of a bond. These are actually chemical reactions and chemical bonds taking place, so the ammoniated hay stays as ammoniated hay for a long period of time. Some of the compounds certainly are going to be a little bit more volatile than maybe the hay was itself. And because this thing gets egg shaped and gets flat, you don't ammoniate it, let it set there for three weeks, and pull the tarp off and go ammoniate another stack, if you're in an area where you're going to expect some rainfall. Because you're going to get rain penetrating that hay from top to bottom. So you've got to keep it protected after you ammoniate it until you feed it.

Question: Does any of this reaction take place in the rumen and if you put ice in vinegar in it, a lot of it quits?

Answer: I don't know if it takes place in the rumen. I really don't think that it did. But it may be. Certainly the ice and the vinegar are going to do two things in the rumen we know as far as ammonia is concerned, and that's to go along with the urea toxicity, and that is going to stop the absorption or greatly limit the absorption of ammonia into the blood stream. So in that kind of a situation you think you almost have just a straight ammonia urea type ammonia poisoning rather than humidizole or something else. One of the products that can be produced in the ammoniation process is urea. So you do have some urea produced in this ammoniated hay, although that is probably a whole lot more important when you get into some other starting materials than hay.

Question: Don't some recover spontaneously?

Answer: That is an excellent comment. Most of them get over in about 8 hours if they don't hurt themselves, without any treatment. That's certainly true. That's why I made the statement that they are short-lived kind of a toxin. I don't think it is something that stays around a long time because it does not hang around long. Animals recover themselves. Just like those heifers that they were feeding at night. They were running through the fences and the next morning they appeared all right, except they did run through the fence.

Question: (inaudible)

Answer: So you just gave the Rompun to the bad ones, sedated them, and they recovered. Were these calves or cows? (Two-year-olds) You're a brave individual because a practitioner that I told, and I'm going back to a comment that you wanted to make, it was something that I wanted to get involved in this thing. In this first case I thought we might be dealing with ammonia poisoning. For that we need EDTA blood drawn from the clinically sick animal frozen and sent to the lab for ammonia analysis. So I told this veterinarian I want you to get me a blood sample from that cow that is showing the signs. He said, you're crazy. He said she'll kill you, she'll run over a horse, she'll run into another cow. This

is the kind of syndrome they really get into when they're really going wild. So if you're able to get some that is beginning to show hyper, that's beginning to show a little bit of quivering, get some Rompun or Ace or something to set those cows down, then you can do a good job with them. When they are really full blown I would be afraid to mess with them just as that practitioner was.

Question: (inaudible)

Answer: That's another good comment. Something I wanted to say about one of the cases we dealt with. In that situation he cut the hay, diluted the hay half and half with something else. In some of the cases we've dealt with that's been a little bit difficult for that farmer or rancher to do, because that hay was so blasted palatable that he couldn't have two round bales out here because the boss cow got all the ammoniated hay and we still had problems. You had to limit-feed that hay and feed the ammoniated hay for awhile, remove the cattle from it, and let them eat something else to get their half and half. But certainly that goes along with the early comments about it takes a very high rate of ammoniated hay to cause problems.

Question: (inaudible)

Answer: That would be impossible to dilute and I think that brings up another thing, a good point that may be important in this. He mentioned he had wet hay. If you look at the early pictures how this thing is done you stack up a big old long stack and you put this tube in at one end and blow the stack up, you get a lot more ammonia at one point than you do somewhere else. I think we're going to have some variable toxicities and maybe even the amount of toxin produced in that hay closer to where the end of that pipe is than on the other end. That's just based on some cases we've seen where in one situation it was not a big tall stack but just a real long row. It looked like they had problems about every time they got to the point in the hay where the tube went in.

Question: What about ammoniated corn silage?

Answer: That may be. That brought something else to mind that I mentioned earlier and I'll get to your question or comment. Another disadvantage of ammoniated hay, you mentioned crude protein, and that is we've got some farmers and ranchers that evaluate the quality of their hay, not by the energy level, not by the TDN, but simply by protein. Most of them that's the way they think. They don't compare the protein in this coastal with that canary grass or that sorghum grass, but they compare the protein in sorghum verses sorghum verses sorghum to figure out what quality they have. Many of them are highly mislead and think whenever we jump crude protein by 6-9% with the ammoniation process that we automatically across the board raise that kind of quality. So we've had a few people that may get in trouble simply because they think they're feeding a better product than they are. I sure wanted to mention that because I think that is another negative side to it although it is not toxicity. Ammoniated corn silage, the only case that I have seen where that was involved, it was a poor job of ensiling. It was one where there were a lot of air leaks. It was properly ensiled and it was not acid like it should have been. So to answer the question, with real, proper corn silage I have not seen that cause toxicity problems. Have you seen that?

Answer: You have to know the dry matter content and ammoniate based on dry matter weight rather than bale weight. That's an excellent point and I slipped by that I'm sure, but that very definitely is dry matter weight because the moisture will get you in trouble.