Disease Problems in Cattle Associated With Rations Containing High Levels of Iodide

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Introduction

Iodine compounds are commonly used by veterinarians and livestock producers to treat or prevent diseases, e.g., foot rot, bovine respiratory disease complex, mastitis, infertility, actinomycotic infections and other chronic infectious processes. This use of organic iodide appears to be supported only by reports of clinical observations describing its therapeutic and prophylactic action (2,5,7,15). There are no previous reports in cattle describing situations where the drug may have been detrimental. However, there are reports in man and laboratory animals describing an adverse effect of iodine on infectious processes (6,8,14,17,18,19).

This report describes four field cases in beef and/or dairy cattle in which it appears that feeding of organic iodine interfered with the animals' ability to cope with an infectious or non-infectious insult under situations of additional stress, e.g., transportation, inclement weather, high milk production or parturition. The role that iodine may have in causing the observations will also be discussed and hypotheses for mechanism of action will be offered.

CLINICAL CASES – FIELD CASE 1: Unstressed, market-weight, feedlot cattle receiving a ration containing high levels of organic iodide.

History

This was a feedlot of 310 near-market weight steers. The hay supply had been exhausted and the decision was made to feed an all-concentrate ration for the remaining month or two of the planned feeding period. This consisted of shelled corn and two pounds per day of a commercial supplement designed for such a program as well as free-choice mineral. The mineral contained calcium, phosphorus and ethylenediamine dihydroiodide (EDDI) as a foot rot preventive. These animals had been on the owner's premises for seven to eight months and had been vaccinated for IBR and BVD upon arrival. There had been no observed sickness or death loss during the feeding period.

Shortly after starting on the all-concentrate feeding program, the animals developed a depraved appetite for the mineral and consumed 15 to 16 ounces per day. The owner noticed widespread light coughing. At one point the owner decided to remove the mineral from the ration. The result was that the feed intake dropped by 50% and after two days of this performance the owner replaced the mineral. Within three hours after replacement, the animals ate **one pound** of mineral per head. About three to four weeks later, the owner sought veterinary consultation. The majority of the animals were alert and eating satisfactorily. His concern was based on the following observations:

1. The coughing had increased in frequency to the point where, at any given time, it seemed that one-fourth of the animals were coughing.
2. Most animals showed excessive nasal and lacrimal secretion.
3. Several animals seemed to be breathing heavier than normal and were sluggish.

An animal that was markedly sluggish and breathing heavily was treated by the veterinarian with antibiotics and corticosteroids. The next day the animal had improved to the point where the owner had difficulty identifying it.

Clinical and Necropsy Findings

The most striking overall impression of the feedlot at the time of our visit (M) 1½ months after the mineral had been added to the ration was the continuous coughing of numerous animals that

seemed to be otherwise healthy. They were generally alert, eating vigorously and appeared in good condition. Five animals showing marked signs of coughing and reluctance to move were confined (with minimum excitation) for individual examination. Physical examination of these animals showed increased respiratory rate and temperatures ranging from 102.9-103.9°F.

The owner agreed to slaughter one animal showing the most aggravated signs so that a necropsy could be performed. The gross necropsy examination showed no abnormalities, except perhaps an increased amount of fluid in the bronchial tree of the lung.

The outcome of the case was that four days after the mineral was removed and mineral not containing organic iodine was offered on a restricted basis, the coughing as well as the heavy breathing and excessive nasal and lacrimal secretions had ceased. The animals were marketed uneventfully, shortly thereafter.

Laboratory Findings

Histopathologic examination of tissues showed only a tracheitis of chronic nature in sub-intimal layers.

Total iodine levels of serums of the five animals examined ranged from 103 to 275 micrograms/100 ml. Analyses of three samples taken from normal cattle at the St. Paul Campus ranged from 5.6 to 8.1 micrograms/100 ml. Previous reports of normal values range from 6-8 mg./100 ml. (11,12).

CLINICAL CASES – FIELD CASE 2: Possible exacerabation of chronic lung disease in feedlot yearlings associated with a high level of EDDI in the ration.

History and Clinical Signs

In early January this owner had assembled a group of 450 head of 500-600 pound calves purchased through sale barns. The calves arrived in good condition and began the feedlot experience with relatively little problem. The owner only treated one or two for Bovine Respiratory Disease Complex (BRDC). The calves were vaccinated parenterally for IBR and PI_3 shortly after arrival. About three months after arrival, several animals started to show signs of foot rot and, as the number of cases was mounting, the owner decided to mass-medicate with EDDI rather than continue individual treatment. He mixed 11.5 pounds of a 4.6% mixture into the total daily ration so as to achieve a daily dosage of 380 mg. per head.

After three to four days of feeding the ration containing EDDI, the owner noticed coughing, lacrimation, sluggishness and inappetance in about half of the animals. The number of animals showing such signs increased, and by the eighth day the owner decided to stop the EDDI administration.

Five to ten days after cessation of the EDDI administration, most of the calves returned to full feed. They gradually stopped coughing over the following one to two weeks. However, four calves continued to cough and had poor appetites. They showed almost continuous open mouth breathing. One of these animals died a few days after the EDDI was removed from the ration. Gross necropsy examination revealed severe bronchopneumonia. About a month later another one of these "open-mouth breathers" was slaughtered, since the owner was convinced that this animal would not gain weight. Again the necropsy examination revealed severe bronchopneumonia.

Two months after the EDDI had been removed from the ration, the owner decided to slaughter the two remaining "open-mouth breathers." One of the authors (M) was present at this time. These two animals were estimated to have weighed 500 and 650 pounds, respectively. The other animals of the feedlot, which had gained normally, weighed 800-900 pounds.

Laboratory and Postmortem Findings

The postmortem examination of the slaughtered animals showed severe bronchopneumonia with extensive chronic lung damage.

About 10% of the lung of the smaller animal and 30% of the lung of the larger animal was judged to be in a functional state. Histopathologic examination of the lungs showed severely advanced lesions of airway disease. The small bronchi had their lumens occluded by cylinders of fibrous scar tissue formed by stagnant bronchial exudate which had been organized by invading connective tissue from denuded bronchial epithelium. The trachea had lesions of a subacute tracheitis. Bacteriologic and virologic examinations did not reveal any of the pathogens normally associated with bovine respiratory disease.

CLINCIAL CASES – FIELD CASE 3: Apparent aggravation of acute BRDC in recently shipped 400-500 pound feedlot calves.

History and Epizootiologic Information

An experienced feedlot operator purchased 800 head of 400-500 pound weaned calves through various sale barns. They arrived at the feedlot in late October and all of the animals began to eat a silage and light grain ration readily, with the exception of 100 head which had experienced a longer truck ride than the rest. The 100 head were slower in starting to eat and over the next ten days several of them were treated for BRDC with generally good response. About ten days after

arrival, the owner, in an anticipatory effort to prevent foot rot which in the past had always occurred in his feedlot in February and March, began to feed the animals a 50 gm./lb. mixture of EDDI calculated to achieve a dosage of 125 mg. per head daily. The compound was mixed into a grain supplement which was top dressed on the silage. His problems with what appeared to be simple BRDC became more serious and animals were not responding to treatment as well as those treated previously. By the 15th day the EDDI dosage had been increased to 250 mg. per head daily.

By the 22nd day post arrival, the owner sought veterinary assistance, as about 200 head were being treated and 15 had died. Between the 22nd and the 48th day post arrival, the situation had worsened. A vigorous effort was made to separate sick animals and monitor their response to treatment by daily temperature measurement. Various antibiotics were used at therapeutic dosages and were given for three to five days or longer periods of time. At times some animals seemed to respond, but they eventually would relapse and had to be returned to the sick pen. Mass medication with high levels of antibiotics was properly carried out but yielded no result. The owner increased the EDDI dosage to 312 mg. per head daily on the 30th day post arrival because he observed some lame animals. About the 40th day post arrival the situation became grave, the mortality rate had increased alarmingly, and by the 48th day about 100 head had died. It was clearly evident that many more were going to die soon.

On the 48th day post arrival, the veterinarian became aware, for the first time, that the cattle were being force fed EDDI. Upon his advice, the owner ceased this practice. Also, at this time the entire group was vaccinated with a commercial IBR and PI_3 vaccine and a pasturella bacterin.

The morbidity and mortality rates contined to become more severe, and on the 51st day post arrival we were asked to assist in the problem. We (M and A) visited the farm and examined sick and "well" animals, performed a necropsy and returned with samples for analysis. Our findings are discussed later.

In the days following our visit the mortality rate contined to increase, and by the 59th day, 170 head had died and by the 70th day, 254 of the 800 head had died. Treatment attempts were abandoned as being futile on all except those that looked "fairly bright." As of the 80th day post arrival, 310 animals had died, and the owner estimated that a few more would die and perhaps another 50 head were so chronically affected that an attempt to fatten them was clearly going to be economically unrewarding. By the following March, some four months after our visit, the death loss totaled 420 head and 10-15 animals appeared to be "poor doers." The remainder appeared to be performing normally.

Clinical Signs

The initial animals showing clinical disease had typical signs of BRDC. Later, about five days after the EDDI was added to the ration, excessive coughing, salivation, lacrimation and nasal discharge were observed in a high percentage of the animals regardless of whether they had been depressed or had exhibited signs of sickness previously. Temperatures were measured daily or every other day in 75 of 200 sick animals during the period between the 22nd and 48th days post arrival. These ranged from 105-108° in most animals, until severe depression occurred at which time the temperatures would be lower.

As time passed these signs became more generalized and severe. Open mouth breathing was commonly seen in the later stages and was usually followed by recumbency and death. Response to treatment for BRDC was disappointing at best. The owner reported that three or four calves showed CNS disturbances prior to death. They had a slow staggering gait, threw their heads back and were hyperesthestic. Subsequently they were totally incoordinated and unable to rise. In addition, he reported that one or two calves assumed a dog sitting position shortly before dying and that four or five animals had prolapsed rectums. The veterinarian described one case of an ataxic animal that responded to vitamin B and antibiotics but died three days later.

At the time of our visit on the 51st day post arrival, the animals had been divided into two groups based on general clinical appearance. One group of about 200 head was very depressed and was kept in sheltered pens. Many of these showed severe dyspnes and 30-40 head were recumbent, and some refused to rise. The other group of 600 "well" calves looked dull and had inappetance, coughing, excessive nasal discharge and lacrimation.

Six of the ambulatory sick calves and six of the "well" calves were examined individually. The range of rectal temperatures of the sick calves was 100-105°F, and of the "well" calves was 104-106°F. The sick calves were in poor condition and showed mucoserous nasal discharge, serious lacimation, excessive salivation and open mouth, dyspneic breathing. The oral mucosa was pale in

some. No oral or foot lesions were observed nor was diarrhea seen. The "well" calves were not as depressed and other clinical signs were less marked.

Necropsy Findings

Necropsy examinations had been performed by the local veterinarian on several of the dead calves. He observed varying degrees of pneumonia, fibrinous pleuritis and tracheitis.

On the day of our visit one comatosed animal was euthanatized and necropsied. The tracheal epithelium was hyperemic and contained a large amount of froth mixed with fibrin. The lungs had extensive areas of consolidation and there was a fibrinous pleuritis.

Laboratory Findings

Serum samples were collected at the time of the visit from the 12 animals examined. A convalescent sample was collected from a randomly selected group of 12 calves four months after our visit. BVD virus neutralizing antibodies were found in 11 of the 12 samples at each sampling time (Table 1)

Table 1

Virologic and Serologic Findings of the Two Groups of 12 Calves Examined in Field Case 3

Calf	Serum						Nasal Swabs**	
	BVD		IBR		PI_3			
No.*	Acute	Convalescent	Acute	Convalescent	Acute	Convalescent	BVD	IBR	PI_3
1.a	Abs		Abs Undil.					+	
b		Abs Undil.		No Abs		1:80			
2.a	No Abs		No Abs					+	
b		Abs		1:2		1:80			
3.a	Abs		No Abs					+	
b		Abs		1:2		1:160			
4.a	Abs		1:2					+	
b		Abs		1:128		No Abs			
5.a	Abs		Abs Undil.					+	
b		Abs		No Abs		1:40			
6.a	Abs		Abs Undil.					+	
b		Abs		No Abs		1:10			
7.a	Abs		Abs Undil.					+	
b		Abs		1:32		1:80			
8.a	Abs		No Abs					+	
b		Abs		1:2		1:80			
9.a	Abs		Abs Undil.					+	
b		Abs Undil.		1:128		1:40			
10.a	Abs		Abs Undil.					+	
b		No Abs		1:2		1:20			
11.a	Abs		Abs Undil.					+	
b		Abs		1:128		1:20			
12.a	Abs		1:16					+	
b		Abs		1:8		1:20			

NOT DETERMINED

*Calves numbered 1-6 had marked signs of sickness as compared to calves 7-12 which had less obvious signs.
**Taken at the time the acute serum samples were collected.
NOTE: (1) Samples denoted "a" were taken three days after vaccination on the 51st day after the calves had arrived. (2) Samples denoted "b" were taken from randomly selected calves four months after samples "a" were taken. These calves were not the same as those from which "a" samples were taken.

(1). The passive hemagglutination test was used to determine the presence of IBR antibodies (16). Only one calf had a significant titer against IBR on the initial sampling. Five calves had significant IBR titers (Table 1) at the time of the second sampling. Ten of 12 convalescent samples had antibodies against Parainfluenza (PI_3) as determined by the hemagglutination inhibition (HI) test. The HI test for PI_3 was not conducted on the acute samples.

IBR virus was isolated from all of 12 nasal swabs and from a homogenate of lung, trachea, spleen and liver tissue collected at the time of necropsy. The virus was isolated in bovine turbinate cells (NADL strain). Coverslips were removed from Leighton tubes as soon as typical IBR cytopathogenic effect was evident and were stained with fluorescein conjugated IBR antiserum to confirm the presence of IBR virus (1). *Pasteurella hemolytica* was isolated from two of the 12 nasal swabs collected at the initial visit.

Four serum samples submitted for iodine determination had iodine levels of 207, 396, 412, and 524 μg/100 ml. A sample of the EDDI mineral mixture was analyzed and found to contain EDDI at a concentration of 38.2 gm. per pound.

CLINICAL CASES – FIELD CASE 4: Adult dairy cattle receiving high levels of organic iodide in their ration.

History

This was an 80-cow dairy herd which had been experiencing a chronic mastitis problem. Approximately nine months prior to our visit (J) EDDI had been added to the ration and teat dipping begun in an attempt to reduce the number of cows exhibiting chronic mastitis. On the basis of guaranteed analysis the amount of EDDI being fed was calculated to be 68 mg. per pound of concentrate. The rate of concentrate feeding was 10-25 pounds per head daily. Therefore the total individual daily EDDI intake was 680 mg. to 1700 mg. There had been a definite improvement in the mastitis problem in the herd. However, about six months prior to our observation of the herd, it was noted that a large percentage of the cows had developed a chronic cough and increased nasal discharge. In addition the owner complained that any cow stressed in any way (calving, retained placenta, foot rot, mastitis) developed a high temperature (105-108°) and would show poor response to treatment. During this time eight cows developed an acute septicemic condition either subsequent to parturition or at their peak production one-two months postpartum. This was characterized by high fever (107°F), lung involvement, metritis in some cows, diarrhea in some cows, decreased appetite and unresponsiveness to therapy. Treatment administered by the attending veterinarian included antibiotics, sulfonamides, intratracheally administered enzymes and supportive therapy. The cows failed to respond to therapy even though it was extensive and persistent. Three cows died and the other eventually recovered after a prolonged chronic course. Seven cows added to the herd three months prior to our visit exhibited coughing and excessive nasal discharge 30-60 days after receiving the ration with the high level of EDDI.

Clinical Signs

Four sick cows were examined at the time of our visit and had the following signs:

Cow No. 40 — one of the native cows in the herd, was first observed sick two months prior to our visit. She was still being treated and had not eaten for two-three days. Examination of the lungs by auscultation revealed the right lung was consolidated. Her temperature was 101.8°.

Cow No. 23 — another native cow, had consolidation of the left lung and an inducible cough. Her temperature was 102.0°, pulse rate 140/minute and respiration 60/minute.

Cow No. 63 — one of the purchased cows, had been treated for retained placenta and high fever (107.0°) three weeks earlier. She still had a profuse diarrhea and was not responding to therapy.

Cow No. 66 — another purchased cow, had exhibited a persistent cough for several weeks, but on examination of the lungs by auscultation, no abnormal sounds were evident.

Within two weeks after the iodine was removed from the concentrate the signs of increased nasal discharges and coughing disappeared. Subsequent cases of metritis and mastitis responded favorably to routine antibiotic therapy.

During the time that the cows were on the high levels of EDDI, there was an increased number of repeat breeders. Two cows reabsorbed their feti and there were increased numbers of calves born extremely weak which subsequently died and a few calves were stillborn. Three cows that calved within two months after the EDDI was removed from the ration had calves with contracted tendons.

Laboratory Findings

Two of the four cows examined individually had hemograms which exhibited neutrophilia with a shift to the left (cows no. 99, no. 40); all four appeared to exhibit a degree of dehydration (increased PCV:Hb ratio) and one was anemic (cow no. 40) (Table 2). Samples were collected for serology from 14 cows. There was no evidence of BVD titers in any cows at each of two sampling times. It appeared that IBR virus was present in the herd as titers were found at both sampling times in a number of animals (Table 3).

Table 2

Hemograms of 4 Cows from Field Case 4 Exhibiting Clinical Signs of Disease Characterized by High Fever, Inappetence, General Malaise and Unresponsiveness to Antibiotic Therapy

Cow Number	Age	PCV	Hb	WBC	L	TN	Stabs.	Seg.	E	Mono's	Total Protein
99	A	30	8.1	11,300	27	66	32	34	-	7	6.7
66	A	35	10.4	11,700	49	39	7	32	12	-	7.8
40	A	26	7.2	14,100	15	85	17	68	-	-	10.8
23	A	34	9.9	9,300	42	37	4	33	15	4	8.2

Table 3

Serologic Titers Against IBR and BVD of Cows That Exhibited Respiratory Signs in Herd Being Fed High Levels of Organic Iodide in Field Case 4

		BVD		IBR	
Number	Age	Acute	Convalescent	Acute	Convalescent
16	A	No Abs.	No Abs.	Abs. Undil.	1:2
21	A	No Abs.	No Abs.	1:8	1:16
33	A	No Abs.	No Abs.	1:16	1:32
66	A	No Abs.	No Abs.	1:16	1:4
49	A	No Abs.	No Abs.	1:4	1:4
65	A	No Abs.	No Abs.	1:16	1:32
23	A	No Abs.	No Abs.	1:8	1:4
55	A	No Abs.	No Abs.	1:32	1:16
40	A	No Abs.	No Abs.	1:2	No sample
48	A	No Abs.	No Abs.	1:32	No sample
25	A	No Abs.	No Abs.	1:32	1:8
34	A	No Abs.	No Abs.	1:4	1:8
27	A	No Abs.	No Abs.	1:8	1:4
57	A	No Abs.	No Abs.	1:4	Abs. Undil.

A = Adult.
Abs. Undil. = Antibodies detected in undiluted serum.
No Abs. = No antibodies detected.

Discussion

Aggravation of the clinical condition of animals receiving EDDI appears to be related to the degree of stress, infection, dosage of EDDI and length of time the EDDI had been administered. In each of the four case histories described, the degree of stress was different — ranging from very little stress with animals showing simply signs of typical iodinism (increased coughing, increased lacrimal and nasal secretion) to the situation of recently shipped calves undergoing BRDC and being unable to respond to infectious insult even when treated extensively with antibiotics. The source of stress in the dairy herd appears to be either high production levels or the act of parturition. This combined with the higher levels of EDDI intake appeared to result in cattle being unable to respond normally to infectious insult. In these and similar cases the clinical picture has been confusing to many observers, because the iodine toxicosis is masquerading under a clinical situation which is familiar but which shows a surprisingly aggravated outcome. This puzzlement has led to controversy whether or not iodine is the factor causing the aggravated clinical signs or if other factors, e.g. multiple infectious agents, may be involved.

The argument for iodine being indicated as a source of aggravation to stress in these clinical cases is supported by reports in scientific literature. It has been shown that the administration of iodine to man and animals undergoing acute infectious and non-infectious insults results in the manifestations of a more severe clinical disease picture (6,8,17,18). The experimental evidence shows that iodine administration to animals undergoing chronic types of inflammatory processes may alter these processes detrimentally (14,19,21). Severe bronchopneumonia has been observed in sheep which died after the administration of iodine in the form of iodinated casein. In this work it was also observed that some sheep receiving high levels of

the compound did not die, which suggests the necessity of both infection and iodine being present (4). A similar observation has been made in lambs receiving KI and EDDI (13). There are several reports of large amounts of iodine having been fed to cattle with no observations of toxicity described. These findings were reported in non-stressed adult dairy cattle, calves and beef calves (10,11,12).

Additional toxic effects associated with the feeding of iodide have been reported. Hyperthermia, anorexia and poor weight gain are described in sheep (4,13). The hyperthermia may be due to the action of iodide to uncouple oxidative phosphorylation, resulting in the inability of energy to be captured in the high energy bonds of adenosine triphosphate (ATP) and its dissipation in oxidative metabolism. Two, four-dinitrophenol (DNP) which causes hyperthermia acts in this way (3). The apparent effect of iodide on inflammatory response may be due to this same mechanism, in that a reduction of ATP may be detrimental to any biosynthetic process of the body in a non-specific manner. Another possibility would be some specific interference with some step in the inflammatory process such as complement fixation. These are only reasonable speculations at this point and further research is needed in this area.

In these field cases dosages were calculated on the basis of guaranteed analysis shown on the product description. On several occasions we submitted mineral mixtures for laboratory analysis for EDDI content. The results varied considerably indicating a possible lack of homogeneity of the material, but in general the levels were close to those claimed. This suggests the possibility of different animals eating various amounts of EDDI due to differences in concentration in the mixture. Also there may be differences in intake due to improper blending of the EDDI mixture in the feed itself. The individual variation of clinical observations seen in these cases may in part be due to a difference in intake of EDDI.

A point of interest in Field Case Three related to the virologic studies was the isolation of IBR from all samples tested. This has not been a common finding in previous studies of field outbreaks of disease.

It is impossible to establish whether or not the IBR virus isolated was a field strain or the parenterally administered vaccine strain. However three observations suggest that it may have been the vaccine strain. One is the fact the samples were collected three days after the vaccine had been administered; a time when it is expected that a large percentage of animals may be shedding virus. A second observation was that IBR virus was isolated from the calves considered clinically normal (by observation, but were febrile when examined) as well as the calves in the sick group. Third is the serologic results; if the cause of the illness in the calves which had been evident for eight weeks had been IBR one would expect that a larger number of calves would have had higher IBR antibody titers at the time of the acute sampling; however, this was not evident (Table 1).

Another speculation that should be considered in field case number three is the reason for the virus being isolated from 100 percent of the samples collected. We suggest two possible reasons. One is that the clinical disease was such an overwhelming process that the calves were unable to respond favorably to an additional viral agent. A second reason could be the possible effect of EDDI on the host response mechanism. Further support is brought to this explanation by recent work in which host response was altered by the administration of corticosteroids to cattle infected with IBR virus. One group of workers report that cattle given corticosteroids demonstrated recrudescence of the viral infection as shown by reexcretion of the virus in nasal and conjunctival secretions and by recovery of the IBR virus from a variety of tissues while control animals (not treated with corticosteroids) did not (19). In other work, results indicate the possible reactivation of virus from an infected bull by corticosteroid administration as shown by the ability to isolate IPV (IBR) virus from a bull only after corticosteroids had been administered (9). Therefore it seems that the alteration of an animal's immune response ability by whatever cause could result in the unusual pattern of virus isolation demonstrated.

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75% of the weak calves. Vaccination of the dam with Electroid 7 at the time she was turned dry has reduced the problem where the calf nursed the cow.

Herd No. 5

A 40 cow beef herd began to lose cows in February. Three cows were found dead. Seven were treated. No new cases were found after the bacterin had been given a couple of weeks.

Dirty Sided Cows:

In the winter when a sick cow is found the owner usually has noticed that several other cows have had muddy sides where the cow has been down. Some of the cows that have been found dead had been seen down in mud a couple of times. I think this cow could be a sign that not just the sick cows have been affected.

Salt:

Many cases in small herds became sick a day after having been salted. The only salt some cattle get is used to toll them in to be counted or moved.

Grass Tetany:

In my opinion "grass tetany" has been overworked. I am sure that most of these cases are caused by some other disease.

Whenever the pH of the rumen and etc. change enough, the absorption of necessary elements is hindered. Whenever the pH is changed, perfringens and other undesirable bacterias can also grow.

Nearly all of the dead cows did not have a normal rumen content. Usually there is very little mixing of rumen contents and nearly all of the food recently eaten is still in the reticulum, and that in the rumen isn't even colored by the contents in the reticulum.

At the present time many local farmers are calling for the "7 Way Vaccine" or the stuff another farmer is using. Three thousand doses have been sold since the first of 1971.

Feed changes (lack of feed in some seasons) make a difference in the occurrence.

Dairy herds encounter extreme changes when they turn a cow dry and kick her out on the ridge on dry grass and when the cow has a calf and is brought in to be fed.

Beef cattle in this area are grazed on fescue etc. in winter with poor quality hay. Even 3 to 1 prevents a lot of losses. The only losses where they were using 3&1 were the ones where it was more like 1&1 (three parts cottonseed meal and one part salt).

Downer Cows:

There are several types of downer cows in this area.

1. A cow down that cannot be set up to stay and falls usually to the right. This is Listeriosis. They may live for a couple of weeks or more.
2. A cow down that will sit up and eat usually has pulmonary emphysema and toxic hepatitis.
3. Worms usually cause the cow to die because she is too weak.

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