

Urea (Ammonia) Toxicosis in Cattle

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Introduction

Urea, a source of non-protein nitrogen, is widely used and of value to supply a portion of the protein requirement of cattle. Previous reports have emphasized precautions to use in feeding urea to cattle, such as the need for complete, uniform mixing in concentrates, amounts to use, and feeding programs most suitable for efficient urea utilization.¹⁻³ While precautions have been emphasized for many years, isolated reports continue on cattle losses associated with feeding urea and ammonia. The losses in most instances have been associated with improper urea feed mixtures, feeding pure urea as "top dressing" on cattle rations, accidental consumption of concentrated urea or ammonia feed mixtures and intermittent feeding (24 to 36 hours) of concentrated amounts of urea in pellets or liquid as under range conditions. The losses in most instances have been a few cattle, but since feeds are involved, the disturbances often result in disputes as to responsibilities and even litigations. Toxicities have occurred under a wide variety of field conditions. The evidence for the role of urea and ammonia in cattle losses is at times rather obscure. It requires more evidence than just that these products were being fed. To determine whether cattle losses were due (or not due) to urea or ammonia has been a frustrating problem for veterinary practitioners on many occasions. Experimental data and information on toxicosis of urea and ammonia as they occur in cattle under field conditions are limited.

This report is a review and summary on the nature of urea ammonia toxicosis in cattle as collected from records in cattle losses under field conditions and from experimental work, some of it unpublished.^{4,5}

Urea: a Concentrated Source of Ammonia

When consumed by cattle, urea is rapidly hydrolyzed by the urease in the saliva and rumen to form ammonia and carbon dioxide (Fig. 1). From the formula, 1 unit of urea could theoretically produce 0.57 units of ammonia or 1 gm of urea could produce 570 mg of ammonia. The ammonia is used by the microbial population which in turn is used by the host animal for protein. The microbes also require energy, especially soluble carbohydrates, to promote growth and a high population. The breakdown of urea to ammonia is reported to proceed about 4 times

faster than the uptake of ammonia by microorganisms.⁶ Many products have been investigated and promoted to slow the process of urea hydrolysis or to enhance the utilization of ammonia by microorganisms. When ammonia is not utilized by rumen microorganisms, it is absorbed by the portal circulation and converted by the liver to urea and eliminated in the urine. If the liver is impaired or its capacity to convert ammonia to urea is exceeded, then ammonia is in the blood vascular system and is toxic to the brain in very small amounts.^{7,8} To prevent toxicoses due to the absorption of ammonia and for the efficient utilization of urea (or ammonia) it should be dispersed throughout the feed mixture. The rather large amounts of ammonia that are potentially available from urea in comparison to other feeds are given in Table 1. The 100 grams of urea would supply as much as 290 grams of pure protein or 605 grams (1.34 lbs) of a 48% protein oil meal. This amount would be sufficient to supply 1/3 of the non-protein nitrogen supplement as recommended in some cattle rations.

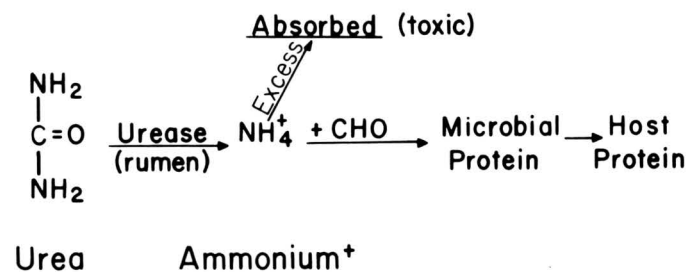


FIGURE 1. Hydrolysis of urea to ammonia.

Acute Urea or Ammonia Toxicosis

Information on acute urea or ammonia toxicosis in cattle was obtained from: 1) information and records in investigating 16 cases of losses associated with urea feeding under practical conditions; 2) toxicoses produced by feeding, drenching or administering urea by stomach tube to 19 steers, heifers and cows, and 3) giving ammonia compounds intravenously to steers.⁵ When fed, the urea was mixed with molasses and ground corn to encourage consumption. Feed was usually withheld 24 hours before

TABLE 1. The potential amount of ammonia that could be formed from 100 grams of urea in comparison to other feed ingredients.

Feed ingredient	Total potential ammonia production in rumen per 100 gm feed (dr. m.) mg	Ruminal ammonia production potential (degradable) %	Realistic ruminal production per 100 gm feed (dr. m.) ¹ mg
Urea ²	57,000	100	57,000
Urea ³	55,800	100	55,800
Soybean meal (48% protein)	9,350	70	6,545
Gr. corn (9% protein)	1,471	70	1,030
Corn silage (7.5% protein)	1,460	60	876
Lush alfalfa pasture (24% protein)	3,922	90	3,530

¹ Total ammonia production potential from feed times ruminal microbial ammonia production equals realistic ruminal ammonia production (on dry matter basis).

² Pure urea (57% NH₃).

³ Feed grade urea (55.8% NH₃).

feeding urea. None of the cattle were previously adapted to urea. The dosage used varied between 0.4 and 0.5 gm/kg body weight. Two steers were drenched with a urea-water mixture, 4 were given urea by stomach tube, 2 readily consumed pure feed grade urea, and 11 were fed the urea molasses mixtures. The trials were conducted with one animal in each experiment. Clinical signs were recorded, blood samples collected for analysis, and necropsies and tissue examination performed on cattle that died.

Clinical Signs

General clinical signs (Fig. 2) were similar when toxic amounts of urea were given by the different methods—yet there were differences. In 10 to 15 minutes after starting to eat a toxic amount, the first signs were observed. These were not eating, head shaking, intermittent belching, kicking at their flank, uneasiness and polyuria. These signs were followed by evidence of impaired vision and a sensitivity to sound and motion, knuckling at the rear fetlocks, a stretched position, excessive salivation, and difficult locomotion. At times the cattle would run wildly within an enclosure and bump into objects and fences. Clonic-tonic convulsions followed with episodes of paralysis and struggling would occur. Bloating became evident and appeared earlier and was more pronounced in cattle with a large rumen—usually individuals being full-

fed or on pasture. Eventually they became prostrate and comatose and died or recovered. Only 1 animal that became comatose recovered. In 24 hours, it appeared normal and was eating. Three that were fed urea recovered during the initial stages of toxicosis and were eating and appeared normal in about 3 hours. Difficult locomotion and wild-crazy running was observed more often in cattle fed urea and convulsive-tetany signs were observed more in cattle drenched or given urea by stomach tube. In urea toxicity under field conditions, cattle owners and veterinarians have all emphasized a "sudden death."

Blood and Rumen Ammonia Values and Lesions

Blood ammonia values varied depending on method of administration, previous diet and rate of consumption. Before giving urea, values were approximately 0.7 to 0.8 mg/100 ml and were usually below 1.0. In toxicoses, values ranged from 1.0 to 2.0 mg/100 ml with clinical manifestations and values of 2.0 to 3.0 mg/100 ml during prostration and in fatal cases. The lowest value recorded in a fatal case of urea toxicity was 1.2 mg/100 ml. This occurred under field conditions.

In 3 fatal cases where cattle were fed urea, ammonia values in the rumen fluid contents were 88,138 and 147 mg/100 ml rumen fluid. Smaller amounts were present in the reticulum and intestine.

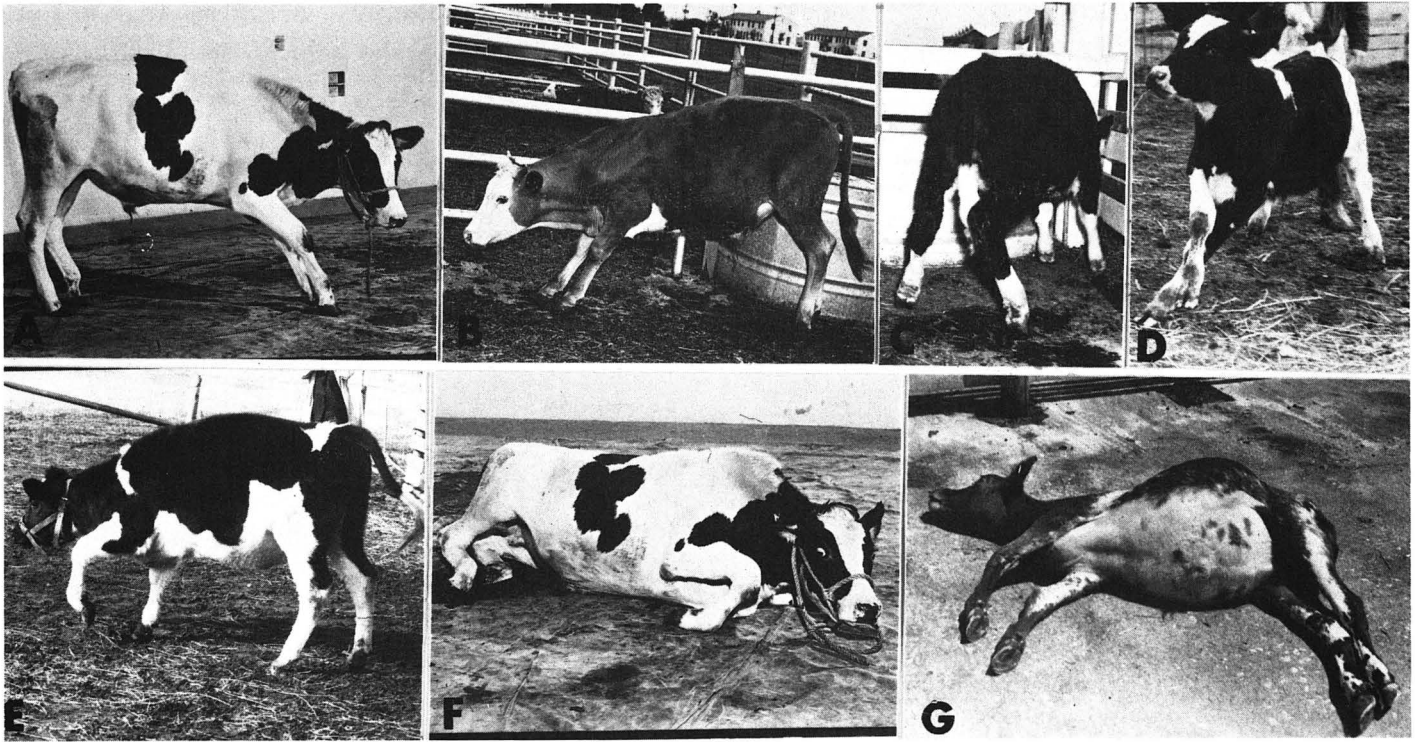


FIGURE 2. Clinical signs of ammonia intoxication in cattle after feeding large amounts of urea.

- A. Starry dilated eyes, uneasiness, and knuckling of rear fetlocks.
- B. Stretched position, elevated tailhead and frequent urination.
- C. Muscular tremors, difficulty standing.
- D. Difficult locomotion and incoordination.
- E. Wild running movements and impaired vision.
- F. Episodes of ataxia, convulsions, tetany, struggling and paralysis.
- G. Coma and bloating.

The most consistent lesion noted in experimentally produced urea toxicosis was edema and congestion in the brain (Fig. 3). In cattle that died from urea toxicosis in less than 1 hour, there was pulmonary edema, but other lesions were not present on gross and microscopic examination. In cattle that died in 1 hour or longer, in addition to pulmonary edema there was petechia; hemorrhages in the heart and catarrhal gastroenteritis.

Effects of Infusing Ammonium Compounds Intravenously

Five steers were used to determine the toxicity of ammonium oxalate, ammonium chloride and ammonium carbonate when infused intravenously in concentrations of 1.2 to 8.0% in 500 ml amounts. All the compounds at 6.0 to 8.0% concentration produced clinical signs as recorded for urea toxicosis. The clinical signs were dependent on the rate of injection and concentration of the compound. Rumen atony and muscular tremors were the initial signs observed and they occurred 5 minutes after the injections were started. Blood plasma ammonia values were correlated with clinical signs. Before, infusion values

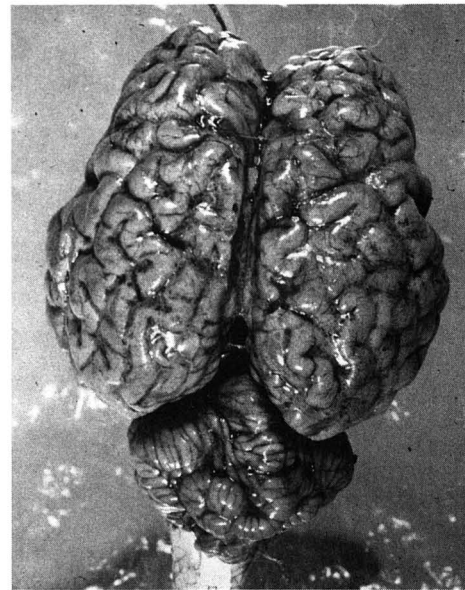


FIGURE 3. Congestion and edema in brain of steer fed fatal amount of urea.

averaged 0.16 mg/100 ml. Clinical signs were manifested when values were above 0.7 mg/100 ml and one steer that died had a plasma level of 1.5 mg/100 ml. The ammonium compounds had no significant effect on the blood pH or the serum sodium, potassium or chloride.⁵

Treatment

Vinegar (1 gallon), acetic acid (5%), and cold water alone have been recommended and used in treatment of urea toxicosis under field conditions, but experimental evidence for reliability where cattle consume toxic amounts is not established. One steer that was fed a urea-molasses mixture and developed clinical signs of intoxication was treated with 500 ml of 5% acetic acid by stomach tube. The pH in the rumen decreased to 7.4 from 7.8 and the steer recovered, but the clinical signs were not significantly improved by the treatment. Untreated steers recovered in the same period of time. The treatment with a weak acid is based on evidence that it will slow the absorption of excess ammonia in the rumen and is dependent on the pH of the rumen contents. At a higher pH, the unionized ammonia is absorbed much faster than the ionized ammonia. Thus, the weak acid administration would slow ammonia absorption. Veterinary practitioners who have used vinegar and cold water alone to dilute the rumen contents were of the opinion they were of value in treating intoxication.

Experimentally Feeding Large Amounts of Urea to Produce a Chronic Toxicosis

For many years uncertainty has existed as to a possible injurious effect of feeding large amounts of urea to cattle over a long period of time. A concern as to an effect on the quality of meat has also been expressed. Since ammonia is quite volatile it is rather rapidly eliminated from tissues or converted to urea by a normally

functioning liver, and any residual effects would not be expected. The small amount of CO₂ resulting from the hydrolysis of urea would not cause any injurious effect.

Experimental

Twelve steers of mixed beef breeding, weighing an average of 300 kg, were used to assess the feeding of large amounts of urea for 140 days. They were divided into 3 equal groups of 4 steers each and individually fed, in tie stalls, one of the following rations: Group 1, control ration of ground corn, molasses and soybean meal in amounts as recommended by the National Research Council for finishing beef cattle. Group 2 was fed the same ration as Group 1 except that urea was mixed in the ration starting at 100 grams per day and gradually increasing for 30 days to 450 grams per day and continued at this amount for the duration of the experiment. Group 3 was fed additional soybean meal, at the expense of corn, to supply the same total nitrogen as the high urea ration fed to Group 2. Steers in all groups were fed twice daily. A mineral-salt mixture and timothy hay were fed free choice in an exercise lot for all steers. Weight gains and blood samples for ammonia and urea analysis were collected at monthly intervals. After 140 days, the steers were slaughtered and tissues collected for gross and microscopic examination. The carcasses were graded, and meat samples were collected for palatability evaluation.

Results

Weight gains for the 140-day period and blood ammonia and urea values at the end of the experiment are summarized in Table 2. Weight gains were slightly less for the steers fed the large amounts of urea. This was attributed to less total feed consumption in these steers. Blood urea values in the steers fed the control ration were about one-half the values of the steers fed the high urea of high protein ration. Except for the initial samples, the

TABLE 2. Average daily weight gains, and urea and ammonia blood values at the beginning and end of the experiment in steers fed high amounts of urea for 140 days.

Group	Ration	No. steers	Avg daily gain (kg)	Blood values avg (mg/100 ml)			
				Initial* Urea	Ammonia	Final Urea	Ammonia
1	Control (NRC)	4	.95	19.4	.73	11.5	.44
2	+ Urea (450 grms daily)	4	.82	18.3	.79	22.2	.48
3	+ SBM same total N as group 2	4	.91	18.9	.63	20.6	.41

* 24 hours after removal from pasture.

values were similar during the feeding trial. Blood ammonia values were about the same for the steers fed the 3 rations. Except for higher values on all steers for the initial samples, the values were similar during the experiment. These steers had been on pasture, and initial blood ammonia values averaged 0.72 mg/100 ml.

When larger amounts of urea were fed to the steers in Group 2, clinical signs of toxicosis were manifested. These were persistent belching (Fig. 4) and coughing, bloating, polyuria and kicking at their flanks as if there was abdominal pain. The urea fed steers would consume their allotted ration in 3 to 4 hours in contrast to 20 to 30 minutes for the steers fed the control or high soybean meal ration. When fed, these steers would eat as usual for about 10 minutes, then quit eating, shake their head, belch, cough, and manifest the clinical signs mentioned, especially bloating. In about 30 minutes, they would start eating again very slowly, nibbling and "nosing" the feed until they had it consumed in 3 or 4 hours. Two of the 4 steers fed the large amounts of urea were classified as chronic bloaters as they remained bloated several hours after they had been turned loose to exercise in a lot with all the steers. At times they were still bloated at their next feeding.



FIGURE 4. Belching and coughing in a bloated steer fed 450 gms urea/day in a high grain fattening ration.

At slaughter, the lateral-dorsal wall of the rumen of the 2 chronic bloaters was edematous with focal hemorrhagic areas. No other lesions were noted on gross or microscopic examination in any of the steers. Rib roasts were prepared from each steer, and on panel palatability tests there were no differences between the roasts from the urea fed steers and steers fed the other 2 rations. The carcasses all graded good to choice. These results were similar to previous studies in that no significant chronic injury could be demonstrated when urea was fed in large amounts for a period of time.⁹ In the previous report, bloating was not a problem, and the same total amount of

urea was fed. In this work the cattle were full-fed while in the earlier study they were fed limited amounts of feed. In pregnant cows, an experimentally produced toxicosis that was treated with acetic acid had no effect on pregnancy.¹⁰

Discussion

A variety of factors influence urea (ammonia) toxicosis, and these factors should be considered in interpretation of absolute values of blood ammonia and amounts that would be toxic. These include the amount consumed, adaptation, type of ration fed, and type of production. The most important single factor conducive to acute urea or ammonia toxicosis is a vigorous appetite in cattle not adapted to urea feeding. It is usually the cow or steer that is first to the feed and anxious to eat at the regular feeding time that is affected, and when allowed to the urea feed mixture it eats rapidly for 5 to 8 minutes. When the cattle are group fed, some dominant individuals will eat more than their share by pushing other cattle away. The palatability of urea and urea-containing rations varies and is unpredictable in cattle. In these acute trials and under practical conditions, some cattle came to a feed bunk and rapidly consumed a fatal amount of pure urea. In other instances, cattle would not eat a concentrated amount of urea even though it was mixed with molasses to "mask" the taste and feed had been withheld for 24 hours. When cattle consume a concentrated urea mixture slowly, they become sick and have mild signs of toxicosis such as belching, polyuria and bloating. They then quit eating and recover. Cattle, even mildly sick from an infectious or metabolic disease, will not usually eat a urea containing ration. The cattle feeder may observe that the steer that was so anxious to eat when fed is found dead near the feed bunk an hour or so later. Ammonia, whether derived from the hydrolysis of urea or other compounds when present in small amounts in the blood circulatory system can be toxic to cattle. Blood ammonia increases of 1.0 mg/100 ml initiate clinical manifestations of toxicosis and increases of 2.0 to 3.0 mg/100 ml are usually fatal, having a toxic effect on the central nervous system. Thus it requires only a small amount of total ammonia to have deleterious effects on cattle health. The blood volume for cattle is given as 57 ml/kg body weight.¹¹ Thus, a 400 kg cow would have a blood volume of 22,800 ml. Theoretically, to increase the ammonia content of blood 2.0 mg/100 ml would require only 456 mg of ammonia to produce clinical signs of toxicosis and death. This amount of ammonia could be derived from 0.8 gm of urea. In previous reports it was concluded that the oral lethal dose of urea for cattle was about 0.5 gm/kg of body weight.^{1,7} This amount was based on toxicity studies in sheep and in cattle when urea was given by drench, stomach tube or through a rumen fistula. Under field conditions, toxicosis occur under different conditions. In experimental methods of administration

more might have been given than necessary to produce a toxicosis as would occur under field conditions. Other reports indicate lower amounts may be toxic when fed.³ Under practical conditions lower amounts are believed toxic, and in one instance a dose of 0.21 gm/kg was fatal to a steer.⁹ In contrast to this low amount and the low amounts believed toxic under field conditions, in the experiment on feeding high levels of urea (almost 1 gm/kg body weight) no neurologic signs or fatalities occurred. Although the amount of urea or ammonia compound fed is important in determining its role in cattle losses, equally or more important is the health of the cattle, how they were fed and whether they were adapted to urea feeding. Since a variety of factors influence the amount of urea that would be toxic to cattle, a range in the amount of urea should be considered. A figure of 0.3 to 0.5 gm/kg body weight seems appropriate. For efficient ammonia utilization, rumen microbes also require readily available source of energy. Otherwise, this source of nitrogen is not only wasted but is a burden to the body to eliminate and may even be toxic.

Feeding Ammoniated Molasses

In the early 1950's, there was an interest in the use of ammoniated molasses to supply a part of the protein in beef cattle wintering and fattening rations.¹²⁻¹⁴ The product most widely investigated was invert cane molasses infused with anhydrous ammonia to supply 15 to 33% protein equivalent. In a large number of cattle where this product was fed, individual animals became what was referred to as "stimulated." Some workers described the cattle as crazy, and they became belligerent and violent, crashing through fences and into buildings. Cattle that were force-fed ammoniated molasses developed clinical signs, especially bloating and convulsions, that appeared very similar to those described for urea toxicosis. Toxicosis due to ammonia was suspected but never established. With the adverse effect of feeding cattle ammoniated molasses and the accompanying publicity, further interest in feeding it discontinued. Recently a similar problem has been experienced in ammoniating large hay bales.

Diagnosis

The history and clinical signs are rather characteristics for urea and ammonia toxicosis. At times the clinical signs may be obscure, especially in beef cattle and to a lesser extent in dairy cattle. In many cases, the initial clinical signs are not observed. Attending veterinarians seldom arrive in time to observe initial, if any, clinical signs. Blood samples for ammonia analysis are specific and valuable if they can be analyzed promptly. At times, blood samples are not available and samples of rumen fluid might be collected for ammonia content. Values of 80 to 100 mg/100 ml rumen fluid may be suggestive of a possible

toxicosis¹⁵ but they do not indicate how much was absorbed and detoxified by the liver. Ammonia rumen values of 120 mg/100 ml have been reported in cattle fed high protein diets and having no signs of toxicosis.¹⁶ Cattle losses due to urea and ammonia toxicosis often end in disputes. Therefore, careful records and documentation should be maintained.

Blood Ammonia Values

While blood ammonia values are important in determining the nature of losses due to urea or ammonia toxicosis, at times problems arise in obtaining an analysis and in the interpretation of the value. Unless very special precautions are taken, blood ammonia values tend to decrease from the time they are taken until they are analyzed.⁵ Thus, if considerable time is involved in getting the samples analyzed, they may not be very accurate. Values depend somewhat on the nature of the diet—cattle on pasture have values about 50% higher than cattle in dry lot.^{5,15,17} Ammonia analysis is considered a specialized procedure in most clinical laboratories costing \$50 to \$60 per sample. It is also prudent to have several samples if possible. Samples from cattle fed the same diet and not having clinical signs as well as cattle fed the same type of diet without the urea or ammonia product would be useful for comparison.

Ammonia as a Cause of Bloat in Cattle

Bloat in cattle pasturing legumes and lush wheat is a problem in many cattle producing operations. It not only causes death losses in cattle but is an impediment to the use of legumes in soil improving programs and in forage production. In acute urea and ammonia toxicosis, bloating and the inability to eructate rumen gases was an early and consistent clinical manifestation. The incidence and severity of bloat is associated with the capacity and fermentation activity of the rumen. Bloating was a major problem in a large cattle feeding operation where toxic amounts of urea were fed over a period of time. On some days over 500 head in a herd of 3,250 were noticeably bloated. Death losses due to bloating and neurological disorders totaled 270. Bloating and death losses ceased when the urea feeding was discontinued. In another herd, under range conditions, that was fed a limited-maintenance type ration containing toxic amounts of urea, bloating was not observed yet 70 head died with neurological disorders. In the experimental work reported, bloating was the most important problem, when the cattle were fed large amounts of urea and had mild clinical signs of intoxication which did not seriously interfere with growth and health. Rumen atony was an early indication of deleterious effect from intravenous infusion of ammonium salts. In an earlier report,¹⁸ rumen extract from cattle dying of bloat while grazing legume pastures inhibited the mobility of segments of the isolated rabbit

intestine, produced bloat in sheep, and inhibited eructation in a cow. The active substance had physical and chemical properties similar to ammonia. The final product in N_2 reduction in alfalfa is the ammonium ion.¹⁹ Ammonia is probably the lowest molecular weight nitrogenous compound with the potential for toxicosis. In ammonia toxicosis and in acute fatal bloat in cattle pasturing legumes, distention of the rumen enough to be fatal was not always present. This would tend to suggest that there are other disturbances such as an impairment of the central nervous system in addition to acute distention of the rumen. Information on the etiology and pathogenesis of bloat in cattle would provide information on methods and products to prevent it and to the greater use of legume pastures in cattle production.

Role of the Liver

The liver has an important role in detoxifying ammonia and other products absorbed by the portal circulation. Small amounts of ammonia not detoxified by the liver may have a profound effect on the central nervous system. In man it is well established that liver impairment and the inability to detoxify ammonia is the cause of an encephalopathy.^{7,8} At times, the increase in blood ammonia is so low it is difficult to detect, and neurologic disturbances are poorly correlated with the degree of hyperammonemia.²⁰ In pigs, liver injury was produced by feeding a low-protein diet or by injections of CCl_4 .²¹ Blood ammonia values were much higher in pigs with liver injury when given intraperitoneal injections of ammonium acetate or a protein hydrolysate, or fed a high protein ration in comparison to control pigs given the same injections or diet. In cattle, the capacity of the liver to detoxify ammonia varies widely.²² In investigating losses in cattle associated with feeding urea, in some herds, while the clinical signs of intoxication were present, rather small amounts of urea were consumed. In these herds there was evidence of protein deficiency in drought areas, prolonged fasting during shipment and insufficient total feed. It was concluded that liver impairment was partially responsible. Enzyme systems in the liver may also need to be adapted to ammonia detoxification.

Summary

Ammonia, whether derived from urea, ammonium compounds or anhydrous ammonia, is toxic to cattle when excess amounts accumulate in the blood. It has a toxic effect on the central nervous system, causing neurological clinical signs and death. Factors modifying toxicosis are rate of consumption, adaptation, diet, and status of the liver. Ammonia causes bloating, indistinguishable from

the type that occurs on lush legume pasture, and it is a consistent clinical sign which may occur independent of the neurological signs. The toxic amounts of urea for cattle are 0.3 to 0.5 gm/kg body weight. While toxic, ammonia is a specific, essential metabolite in the degradation and synthesis of proteins. Urea can be used to supply a portion of the protein requirement of cattle. Care and consideration must be given for its safe and efficient use. This includes proper amounts, thorough mixing in rations, and carefully adapting cattle to consuming it.

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