Nutritional and Management Considerations Associated with Pen Deaths

Keith R. Hansen
Nutrition Service Associates
Hereford, Texas

Introduction

Death of cattle in the feedlot has caused the loss of countless dollars of potential profits annually by cattle feeders. Managers spend large amounts of their time and energy in an effort to decrease these losses in order to save this revenue for the feedyard and their customers. When viewed from an economic standpoint, a feedyard which will annually feed 60,000 head might have an average death loss from various causes of approximately one percent or about 600 head. If these cattle are fed on the average from 725 to 1100 lbs. their median weight would be equal to 912.5 lbs. for a total annual loss of 547,500 lbs. At an average selling price of $72.00 per hundredweight, this would equal a loss in equity of $394,200.00 This is in terms of average **value** of the cattle. Associated reduction in revenue would be the loss of the potential value of finished beef, loss of interest and veterinary expense, loss of potential feed sales and finally the decrease in profit potential by the cattle owner.

Obviously the causes of death in feedlot cattle are numerous and difficult to predict. Realistically, every feedyard has or should have, some type of animal health program which it subscribes to for prevention and treatment, requiring considerable amounts of man-hours and drug expense annually. A large portion of morbidity and mortality in the feedyard is caused by shipping stress. Cattle, which are shipped long distances, have been subjected to excessive handling as well as environments rich in pathogenic organisms in the receiving and handling pens. Other stressors include withdrawal of feed and water, long periods without rest and finally subjection to diesel fumes while on the truck. Physiologically, these animals are tired, weakened and dehydrated, having lost substantial levels of vitamins and electrolytes while in transit. This produces an environment within the animal that is lower in immunological activity than that of nonstressed animals and is therefore perfect for the propagation of pathogens.

This lowered resistance is enhanced by the co-mingling of cattle from various origins and the resulting subjection to infectious agents to which they may not have developed a natural immunity. These cattle are excellent candidates for any of the host of feedlot diseases commonly observed which ultimately result in death losses. Many times the feedyard manager implicates the consulting veterinarian and his health program for **all** death losses in the yard. While the veterinarian does have a large responsibility concerning newly received cattle as well as the overall health program in the yard, there are areas affecting cattle mortality where he has only limited impact.

Appreciation to Mr. Stephen B. Blezinger for his assistance in compiling the literature review.

I. Conditions and Pathenogenesis of Agents in Nutritionally Induced Feedlot Deaths

An area for which veterinarians are often incorrectly held accountable is that of cattle morbidity and mortality of nutritional causes. Clinical and subclinical disorders are more often produced by a number of factors including the nutritional program and management in general. In this situation there is often little the veterinarian can do in terms of prevention with the tools he has at his disposal. Probably the greatest input he can have in these areas is that of proper diagnosis of the causative factors in order that attempts may be made to correct the situation if the source of death is nutritional or management related. In these situations, the consulting nutritionist may be implicated when performance depression or death loss becomes prominent.

Three primary sources of loss are ruminally manifested conditions known respectively as acidosis, bloat and enterotoxemia. All three may be derived from improper management of the rumen microbial population from a nutritional and managerial standpoint. Britton (1981) defined acidosis as a biochemical and physiological stress caused by a rapid production and absorption of ruminal organic acids and endotoxins. He went on to note that it is often caused by the overconsumption of readily fermentable carbohydrates (RFC) such as grains, especially those in an extensively processed form. Corn, grain sorghum (milo), wheat and to a lesser extent, barley, are grains commonly used in growing and finishing cattle and must be processed in some way in order to produce the highest level of performance. By processing, the area of exposed surface is increased and the starch within the seed is made more available to an increased rate of microbial degradation in the rumen. Processes such as rolling or grinding, steam

flaking, exploding and micronizing have all been used to produce a more ruminally degradable product for the feeding of cattle.

A second area of concern in the feedlot is that of bloat. Bloat has been defined by Jensen and Mackie (1979) as a condition characterized by the retention of gas in the rumen or an inability by the animal to eructate and relieve the intraruminal pressure produced by accumulation of microbially synthesized gases. Although several types of bloat exist, the most common types observed in the feedyard are Grain Concentrate Bloat (GCB) and Free-Gas Bloat (FGB). Both conditions appear to partially originate from lower pH levels in the rumen associated with the feeding of RFC. Grains such as corn, barley, alfalfa meal and soybean meal appear to increase the incidence of GCB. Hironka et al. (1973) also found an increased occurrence of GCB when grains were finely ground before feeding. Gas production from finely ground rations was 31.45% higher than that produced from coarsely ground rations. This would indicate a significant role played by ration texture, especially in those rations with high levels of fines resulting from incorrect or over-processing.

The final area to be addressed is that of enterotoxemia (ET). Manifested by the production of endotoxins by *Clostridium perfringes* which are subsequently absorbed in the intestinal regions, ET is characterized by a short course (1-6 hours) and normal body temperature. All strains of C. perfringes (A,B,C, and D) cause ET, only types C and D are thought to produce sudden death losses in feedlot cattle. The bacteria are commonly found in animal intestines and manure and exist on substrates of sugars such as fructose, lactose and starches. The proliferation of the various strains of *C. perfinges* is thought to be initiated following some debilitating event such as fasting followed by a full diet high in RFC (Jensen and Mackie, 1979).

Each of these conditions will be addressed in detail in the sections following.

Acidosis

Acidosis is only one of a number of labels used to describe the acidic condition of the rumen and blood. Other names commonly applied include D- lactic acidosis, overeating, acute impaction, grain engorgement, founder and overloading and describe for the most part the same condition. Overeating disease and founder, however, are often used to describe somewhat different conditions but are thought to originate from acidotic ruminal conditions.

There are fundamentally two types of acidosis recognized to affect feedlot cattle. Acute or clinical acidosis (Dunlop and Hammond, 1965), which is characterized by lowered pH in the rumen, blood and urine, is life threatening and will often result in death in one to several days. This type of acidosis will be the primary focus of this section as it does result in cattle mortality. Subacute or subclinical acidosis (Dirksen, 1970; Koers et al., 1976), while not life threatening, tends to produce greater physiological and economic costs in terms of reduced feed intake and weight gain. Subacute acidosis goes largely undetected until slaughter when effects of rumenitis (a result of the ruminal acidotic condition) and liver abscesses are observed and produce substantial amounts of rumen and liver condemnation (Church, 1989). One observed difference between acute and subacute acidosis is that acute acidosis can produce pH levels as low as 4.0 whereas subacute conditions will rarely exhibit pH levels lower than 5.0 (Britton, 1981). In addition to the two levels of acidosis, Brent (1976) identified related problems existing in the feedlot as well. These conditions include: 1) rumenitis and liver abscesses; 2) laminitis or founder; 3) polioencephalomalacia (PEM). Each of these conditions is brought on in certain situations by ruminal acidosis and vary in degree of severity. With the exception of PEM, they will not normally result in death but will produce severe reductions in performance in stricken animals.

Acidosis finds its origin in an abrupt change in the ruminant's diet. A larger proportion of cattle throughout the world spend the better part of their lives consuming forages and as a result have a microbial population within the rumen that is adapted to this type of diet. This population is high in organisms which ferment cellulose and other structural carbohydrates as well as those microbes capable of using the by-products from this fermentation as a substrate on which to survive. In turn, the animal's body is adapted to those microbical nutrients produced in the ruminal fermentation process.

When cattle are placed in an intensive feeding situation, the objective is for these animals to consume high concentrate, high energy diets with the least amount of roughage possible to maintain the animal's physiological integrity. In order to do this the microbial population and the host animal must be adapted to the new energy and concentrate level, a different type of carbohydrate and a different type of food particle size and configuration. As ration changes take place, a shift in the proportions of the microbial population occurs to best accommodate the new diet. If the dietary change toward RFC is too abrupt, large increases in the proportion of *Steptococcus bovis* are observed. *Streptococcus bovis* is an important producer of lactic acid and in the presence of RFC will synthesize large quantities of this organic acid. Also increasing by vast proportions at this time are the Lactobacillus strains of bacteria which are also large lactate producers. This increase in intraruminal lactate will drive a subsequent reduction in pH. *Streptococcus bovis* appears to grow and reproduce significantly until pH reaches 4.5. At this point if animal death has not already occured, *S. bovis* numbers are reduced due to the acidity of the environment. Lactobacilli, which are tolerant of a very low pH will continue to multiply and generate lactate (Hungate, 1966). Acidosis ap-

pears to be brought in part by this increase in lactate producing bacteria as well as an inability of the lactate utilizers which are normally present in the rumen to increase in numbers sufficient to compensate for the accumulation of the organic acid (Yokoyama and Johnson, 1988).

Another aspect of acidosis relating to the microbial population is the fact that the reduced pH levels brought on by an increase in lactate is the reduction in numbers of gram negative bacteria, largely thought to be highly beneficial in feedlot animals. Destruction of gram negative bacteria results in a release of endotoxins from the dead organisms which have potential pharmacological activity and the shock-like appearance of acutely ill animals may indicate that bacterial endotoxins are a substantial contributing factor in the lactic acidosis syndrome (Huber, 1976).

In addition to bacterial changes, researchers have found that ruminal protozoa concentrations are greatly reduced by the increasing environmental acidity (Hungate, 1966).

The accumulation of ruminal lactate and subsequent absorption producing a systemic acidosis affects a number of associated peripheral functions that serve to compound the overall acidotic condition.

A contributing factor to the acidic condition of the rumen is the reduction of salivation brought on by the acidosis (Slyter, 1976). Under normal conditions, saliva acts as a buffer (largely bicarbonate) to the rumen and with a reduction of salivation a subsequent reduction in buffering capacity is also observed. Bailey (1959) observed that fine grinding or pelleting significantly decreased the salivary output as related to the amount of chewing time which is also decreased with consumption of highly processed feedstuffs. This would indicate that rations must be evaluated on their roughage to concentrate ratios and the subsequent ability of these feeds to maintain an acceptable level of salivary output roughage activity thus retaining buffering capacity (Brent, 1976).

Evidence presented by Svendsen (1973) indicated that increased absorption of volatile fatty acids (VFA) including lactate inhibited rumen motility. Normal movement of the rumen is necessary for the retention and mixing of ingesta for the purpose of microbial digestion. Rumen motility is also necessary for the eructation of the large amount of microbial gases produced (Ruckebusch, 1989). As pH approaches 5.0 amplitude and frequency of rumen contractions progressively diminished with eventual stasis (Huber, 1976). The rumen stasis following rumen acidification may serve as a protective mechanism since lactate absorption from the rumen is dependent on rumen motility (Ahrens, 1967).

Huber (1976) reported a 61% increase in rumen osmolality produced by lactic acid. Such an increase in osmolality would lead to a hypertonic state within the rumen and would draw large amounts of body and blood water concentrations into the ruminal cavity. In addition, large fluid losses via feces coinciding with low rumen motility, suggests a net movement of body water into the intestine. Diarrhea in acidotic cattle is a commonly observed occurrence and coupled with the water losses into the rumen produce a state of dehydration in the animal.

Still another ruminal condition produced by acidosis is that of damage to the rumen epithelium with clumping and necrosis of the papillae in general (Orskov, 1986). This condition, often referred to as rumenitis, leads to decreased performance in less acute cases in terms of obstruction to normal absorptive capacity. Possible factors contributing to rumenitis as listed by Brent (1976) include high organic acid concentrations, the low resulting pH and high osmotic pressure, bacterial endotoxins, and the fact that any one or combination of these factors may render the epithelium susceptible to mechanical injury. Breaks in the rumen wall also provide entry into the portal system for ruminal bacteria, in particular *Sphaerophorus necrophorus. S. necrophorus* are commonly found in liver abscesses as well as ruminal contents and feces of healthy as well as diseased animals. This would lead one to conclude that these organisms gain entry to the portal system from the rumen via the breaks in the ruminal wall caused by sloughing of epithelial cells and papillae which, ultimately, is the source of liver abscesses (Jensen and Mackie, 1979). An interesting observation by Fell (1972) and Orskov (1986) pertained to the level of damage to the epithelium from hair ingested by cattle on high concentrate diets. This is particularly observed in cattle which are in the feedyard in the spring of the year when the winter coat is being shed although a certain amount of hair ingestion is found commonly throughout the year. Hair is taken in by licking after which the hair becomes embedded in the rumen wall and in many instances provides a route for bacteria to enter portal circulation thus contributing to the liver abscess problem.

After the rumen, the next important area of consideration is that of systemic effects of lactic acid on blood pH profiles and metabolites. Acids produced in the rumen are absorbed into the bloodstream, where they accumulate and form the basis for systemic acidosis. Blood pH is lowered and an imbalance of electrolytes results along with an increase of lactate concentrations in the blood. Huntington (1988) reported a decrease in plasma or serum concentrations of minerals, particularly calcium, and an increase in the excretion of calcium.

An important effect of acidosis on the blood is that of alteration of blood acid-base balance and more specifically the effect on the blood buffering system. Under normal circumstances, lactic acid will enter the blood and dissociate. The anion combines with sodium or other cations and the hydrogen combines with bicarbonate forming carbonic acid which in turn dissociates to carbon dioxide and water. The overall effect is an increase in blood CO_2 and a decrease in bicarbonate concentration. According to the

Henderson-Hasselbach equation, the bicarbonate-CO_2 ratio (normally 20:1) should decrease with a decrease in pH. The desired buffer pair ratio is restored through an increased CO_2 tension of the blood along with a decreased pH stimulated respiratory center elimination for the CO_2. With enhanced microbial production of lactate, which lends itself to a much more rapid uptake than is normally observed via intake, the lactate levels in the blood rise rapidly, challenging the blood buffering system. This produces a depression of the respiratory center, decreased blood pressure and a rapid decrease in pH (Huber, 1976).

Concomitant hematological changes also occur due to blood acidosis. Hematocrit and packed cell volume increase significantly due to the net flow of water into the rumen which concentrates the blood and dehydrates the tissues. Because of acute metabolic disturbances, the spleen releases its stored erythrocytes and this along with the dehydration effect produce the increase in hematocrit percentage. Other related effects include stimulated hyperpnea and increased urine pH which gradually compensate the acidosis. Urine volume also decreases which is probably a side effect of the dehydration. Ultimately, death of affected animals probably results from shock and respiratory failure (Jensen and Mackie,1979).

In summary, acidosis is brought on by the rapid fermentation of starches and sugars by the unadapted microbial population. This activity, in acute acidosis produces the following results as reported by Elam (1976):

1. Increased levels of lactic acid in the rumen and blood.
2. Reduction of rumen and blood pH.
3. Increased osmotic pressure in the rumen.
4. Destruction of gram negative bacteria and proliferation of gram positive bacteria in the rumen.
5. Reduction in ruminal protozoal count.
6. Rumenitis and sloughing of rumen epithelium.
7. Rumen stasis.
8. Reduced urine pH.
9. Dehydration and hemoconcentration.

Cumulatively, these conditions produce a very serious physiological effect on the suffering animal and can produce death in one to several days.

Treatment of Acidosis

As with any disease, the most effective treatment is a good prevention program. Prevention of acidosis is most readily accomplished through the conscientious formulation of rations as well as use of carefully planned programs for the increase of energy levels fed to cattle. However, a number of methods and compounds exist which may have an effect on the prevention or treatment of acidosis with efficacies ranging from good to very limited.

Probably the simplest method of prevention of acidosis is to limit the level of concentrate in the ration. This practice, while practical from a physiological standpoint, loses its validity when viewed in an economic sense. Costs per unit of energy is substantially higher for roughages than concentrates and in order to feed cattle as economically as possible, rations must be as high as possible in concentrate.

Currently, the common use of ionophores and other antibiotics in the feedyard has lent itself to the reduction of acidotic effects (Beede and Farlin, 1977; Dennis et al., 1981; Nagaraja, 1982). Nagaraja et al. 1981) conducted a study concerning the efficacy of lasalocid and monensin in the prevention of lactic acidosis. Three experiments consisting of four trials each were designed using two ruminally fistulated steers, three fistulated cows and four fistulated cows respectively. In each experiment, cattle were maintained on an alfalfa hay diet and received either monensin or lasalocid via intraruminal administration at a rate of 1.3 mg/kg of body weight on the day prior to trial initiation. Acidosis was induced by intraruminal administration of glucose (dissolved in water) at 12.5 g/kg of body weight. Finely ground corn was administered in the same manner at a rate of 27.5 g/kg of body weight. Both glucose and corn treatments induced acidotic conditions in control animals. Both lasalocid and monensin effectively prevented acidosis in treated cattle. Ionophore treatments were also shown to increase total VFA concentration in treated cattle.

In a study by Dennis et al. (1981) it was reported that lasalocid and monensin effectively inhibited most of the lactate-producing bacteria including *Butyrivibrio fibrosolvens, Lactobacillus ruminis, L. vitulinus,* and *Streptococcus bovis*. At the same time observations were made that none of the major lactate fermenters including Anaerovibrio, Megasphaera, and Selenomonas were inhibited by the ionophore treatments. The results of these and similar studies indicate that monensin and lasalocid as well as a number of the second generation ionophores appear to have a substantial effect in the reduction of acidosis. The mode of action would seem to be much like that observed in other rumen related ionophoretic affects on the microbial population. The inhibition of certain types of bacteria while other types are not effected produces the desirable effects noted in these studies.

Still another practice that has been identified as a possible hindrance to lactic acid production in the rumen is sulfur supplementation. Whanger and Matrone (1970) reported that sulfur deficiency in the rumen impairs the ability of ruminal microbes to utilize lactic acid as a substrate to the full extent possible. By addition of supplemental sulfur to the diet it may be possible to reduce the accumulation of lactic acid in the rumen after feeding a high concentrate ration. In a three part study by Rumsey (1978), it was shown that addition of sulfur to the diet lowered the peak accumulation of D- and L-lactic acid by 36 and 25% respectively, after feeding when compared to non-supplemented treatments. Lactic acid peaks occurred

6 and 8 hours post feeding for ruminal L- and D-lactic acid respectively, after a 96 hour feed restriction. Based on this data it may be beneficial to add low levels of sulfur to the ration when cattle have gone off feed for a period of time and will be returning to the feedbunk with an increased appetite to aid in prevention of drastic increases in ruminal acid production. Another important point to consider is that it was also shown by this study that addition of higher levels of sulfur to the diet decreased intake significantly when fed for an extended period of time.

Other practices and procedures have been used in the reduction of acidosis in the feedyard but as long as it is economically beneficial to push cattle as hard as possible in terms of feeding of high energy concentrates, acidosis will be a problem at least to a limited extent.

Bloat

A second cause of cattle deaths in the feedyard is bloat. Bloat was first described by a Roman author in 60 A.D. in terms which indicate that the symptoms have not changed over the centuries. The treatment he prescribed included, "pouring sour vinegar through the left nostril and putting two ounces of grease in the jaws." when compared to therapy of the condition used today, it is obvious that little progress has been acheived in almost 2000 years (Essig, 1988). Bloat produces economic losses from a host of factors including death of the animal, substantially reduced performance of recovered cattle, incomplete utilization of bloat-provoking feeds, premature marketing of chronics as well as increased labor required for treatment (Jensen Mackie, 1979). As suggested by the previous statement, this condition will either kill the animal immediately or, if the animal is found in time and treated successfully, the possibility is very good the condition will recur or that the animal will not perform to his potential in the feedyard.

Bloat is caused by extreme intraruminal pressure from gases produced by the microbical population. During this period, a series of pathophysiologic changes occur. The expanded rumen pushes the diaphragm forward, partially collapsing the lungs. The intraabdominal and intrathoracic pressures force blood out of the viscera into peripheral blood vessels and cause more CO_2 to dissolve in the plasma and produce acidosis. This tends to be a contributory factor because in many instances bloat is initiated by a acidotic state in the rumen. The interference to circulation and respiration and the plasma changes rapidly and alarmingly distress the animal and may cause death within a few minutes to a few hours (Jensen and Mackie, 1979).

Bloat develops in all breeds, ages and sexes of cattle with the greatest incidences in the English breeds including Hereford, Angus and Shorthorn. Crosses of these breeds seem to be more resistant as do Brahman crosses such as Santa Gertrudis and Beefmaster. Also more highly susceptible appear to be cattle aged 0.5 to 3 years of age. It should be noted that a great deal of variability is found within breed and age groups.

As discussed previously, there are two major types of bloat commonly observed in the feedlot. Grain Concentrate Bloat (GCB) is thought to be highly dependent on the type and physical form of the ration fed. Free Gas Bloat (FGB), while also dependent upon the type of feed may result from a physiological inability to release the gas pressure built up in the rumen. Both types of bloat are thought to have their origin in an acidic condition of the rumen contents although FGB may be highly correlated with an abnormality in anatomical structure of the cardia of the rumen due to injury or a genetic defect. In almost all situations, however, when bloat has been diagnosed, the pH of the rumen has undergone considerable depression (Hungate, 1966).

Grain Concentrate Bloat

Also referred to as feedlot bloat, the incidence of GCB increases with an increase of high grain diets. Rations high in corn, barley, soybean meal and alfalfa meal have been shown to be especially GBC provocative. *One particular reason for this is similar to the cause of acidosis: the subjection of cattle to a ration high in RFC and utilization of a minimum adaptation period.* As with acidosis, an increased proliferation of *Streptococcus bovis* is observed along with a concomitant increase in *Peptostreptococcus elsdenii,* both of which have been implicated as organisms partly responsible for GCB (Muir and Barreto, 1979; Hungate, 1966). As reviewed previously, *S. bovis* concentrations increase drastically with a sudden change in diet toward concentrates. This may be a partial reason for the increase in bloat during grain adaptation periods in cattle. *P. elsdenii* appears to be an important fermenter of lactate, an important consideration in the observation the P. elsdenii concentrations simultaneously with *S. bovis* which produces large amounts of lactate. Net gas production by *P. elsdenii* is largely in the form of CO_2 and to a lesser extent, H_2.

A second possible bacterial factor is the increase in ethanolprecipitable bacterial slime in the rumen of bloating cattle as observed by Gutierrez et al. (1961). He went on to note that this slime had two to three times the viscosity of water. Analysis of the slime produced results of 34% crude protein, acid-hydrolyzable polysaccharides and nucleic acids. The presence of the nucleic acids may derive its origin from the destruction of ruminal protozoa; a common occurrence due to the increase acidity of the environment. The nucleic acids would serve to greatly increase the viscosity of the fluid. It would appear that the bacterial slime may form a matrix which holds the gas forming a mass which gradually increases in size. Researchers have also found that certain cations, especially calcium, play a roll in the foaming capacity of rumen contents (Rosen et al., 1961; Miltimore et al., 1970). This foaming activity is much

the same as that which occurs in animals consuming a diet of fresh, lush legumes.

A separate contributory factor to that matrix which may serve to trap ruminal gas is the presence of finely ground feeds in the diet. The finely ground particles may contribute to the consistency of the surface tension and thus serve to strengthen the froth matrix (Hironka, 1973). This would also help explain the increased incidence of bloat in cattle given feed containing large percentages of fines. The amount of roughage in a diet is also a contributory factor with roughage levels of less than 10% tending to increase the percentage of bloating cattle. In cattle that bloat after extended periods in the feedyard, bloating may result from the two previously described factors above with greater frequency than any other.

A final factor which may contribute to the tendency of feedlot cattle to bloat is that of reduced levels of anti-foaming mucin from saliva which may facilitate frothing (Jensen and Mackie, 1979). Here again is an effect potentially originating from the acidotic condition common at this time and the subsequent reduction in salivary output.

Free Gas Bloat

Free gas bloat is ruminal overdistension with one or more pockets of gas overlying the ingesta. This form of bloat does not tend to produce the froth observed in feedlot and legume bloat. Retention appears to be caused by an impaired ability of the animal to eructate **normally** produced gas. Jensen and Mackie (1979) observed that this inability may have been caused by one or more factors including: cardial and esophageal obstruction-occlusive and compressive neoplasms, foreign bodies, expanding abscesses, inflammatory swelling, edematous allergic reactions, constrictive scars, enlarged thoracic nodes, diverticula and diphtheria. Other potential causes noted were dysfunctions such as paralysis of the vagus nerve, atrophy of muscle, and disruption of reflexes. All these factors could potentially interfere with the escape of gas and favor its accumulation. This form of bloat tends to repeat in the same animal even after treatment and cure of the immediate case.

Treatment of Bloat

As with acidosis, the best treatment for bloat is prevention. The most obvious methods in reducing the incidence of bloat include refraining from overprocessing of grains which produce high percentages of fines and providing at least 10% roughage in the diet. Processing control may or may not be entirely feasible depending on mill facilities and ability of personnel.

If severely bloated animals are observed before death, the pressure may be relieved by the insertion of a one inch (I.D.) hose via the esophagus into the rumen. This will prove an effective treatment for FGB but not GCB which often is frothy in nature. Situations such as these require the administration of an anti-foaming agent such as mineral oil.

An important consideration in treatment is the handling of bloated animals. Excessive excitement on the part of the animal will only serve to aggravate the matter, placing an additional strain on its already overstressed respiratory system.

Closely tied to acidosis, bloat also will remain a challenge to the cattle feeding industry as long as current feeding practices are maintained and without a product that provides appropriate preventative activity.

Enterotoxemia

Enterotoxemia (ET) is often referred to as sudden death syndrome for the rapid death that occurs in animals stricken with little or no advance warning. While ET is most notably an antagonist of sheep operations, it is commonly found in cattle feeding operations. Enterotoxemia type C and D occur in all breeds, sexes and ages of cattle, but more often in animals under two years of age. Named for the type of endotoxin produced by the *Clostridium perfringens* bacteria, type D appears to be the most prevalent type of ET in feedlot cases although type C is also found on occasion. *Colstridium perfringens* is widely distributed and is found in soil, manure and animal intestines where under normal conditions it exists in such small concentrations and in a state in which little or no endotoxin is produced.

The pathogensis of ET type C is little understood but presumably the majority of this strain is ingested with contaminated feed and water. Other situations conducive to ingestion of spores include mineral deficiencies when animals are known to eat small amounts of soil to attempt to meet these deficiencies and in drought or poor grazing conditions where animals are required to consume the short grass very close to the ground. Under certain provoking conditions, the ingested bacteria metabolize the dietary starch and synthesize toxins. *It has been theorized that conditions that might initiate the production of toxins may very probably be increased acidity of the ruminal environment with increases in the levels of RFC, thus making ET another possible side effect of an acidotic condition.* The synthesized toxins act as an irritant to the gut, produce acute enteritis, increase capillary permeability, and enter the blood. Many affected animals die in a relatively short period of time, possibly from adverse effects on respiration or circulation. Those animals which, for some unknown reason, survive an attack, develop antitoxins and probably are immune to subsequent attacks.

Type D strains differ from C somewhat in that they are commonly found in the small intestine where type C is manifested in the rumen. Type D bacilli metabolize starch particles as well and produce a prototoxin which is activated by trypsin. This toxin acts on the gut wall as well and follows a similar course of events as that with type C. Type

D also further damages and weakens endothelial cells in other organs and causes liver glycolysis and hyperglycemia. Here again, the activity of the *C. perfringens* organism appears to be directly related to the abrupt change in starch content of the diet and the resulting decrease in pH tends to increase population concentration as well as toxin production. Affected cattle with this strain of ET appear healthy and have a normal body temperature. Morbidity from this strain is less than 0.1% while mortality approaches 100%. The course ranges from one to six hours thus lending itself to the name "Sudden Death Syndrome" (Jensen and Mackie, 1979).

Treatment of Enterotoxemia

Due to the rapid manifestation of this condition, treatment is seldom feasible, much less effective. On a parallel note, in many situations due to the low incidence of the disease, preventative measures may not be warranted. However, with the use of multi-strain clostridial immunization commonly practiced in the feedyard, the use of a vaccine including antibodies against *C. perfringens* adds only a minimum expense to the drug cost and provides some degree of effective insurance against the disease.

Other practices which may help in the reduction of the condition include those used to reduce incidence of acidosis and bloat, conditions which seem to stimulate the onset of ET. Loss minimizing procedures include (Jensen and Mackie, 1979):

1. Increase the proportion of dietary roughage.
2. Include in the ration chlortetracycline or oxytetracycline to provide 2 mg/kg feed daily.
3. Include in the ration quaternary ammionum compounds to provide a daily intake of 2 gm/animal.
4. Vaccinate on entrance to the feedlot with a polyvalent bacterin containing several clostridial species including C perfringens types C and D.

II. Effects of Ration Formulation and Management Practies on Nutritionally Related Deaths in the Feedyard

It is necessary to feed cattle a ration which will produce a finished product as rapidly as possible while creating a product that is highly desirable from a marketing standpoint. As a result cattle are fed rations high in energy and concentrates while meeting all other nurtitional needs. This is done to achieve the highest daily weight gains possible with the lowest feed requirement and yet keep ration costs at their absolute minimum.

Most finishing rations contain from 7 to 12% roughage. The considerations are: 1) Produce a feed product with the highest possible energy level while keeping digestive disorders at a minimum; 2) Roughage costs per unit of energy are normally much higher than grain costs per unit; 3) Physical characteristic of the available roughage source. If the second factor is examined, we find that it is true in every situation, despite the changes in commodity costs. If current values for alfalfa hay and steam flaked corn are compared on a dry matter basis, the following data are noted:

Commodity	NEp	$/cwt	$/unit energy
Alfalfa	27.62	$5.68	$.2056
Steam flaked corn	67.47	$5.93	$.0879

From this data it is obvious that per unit of energy, steam flaked corn is much less expensive that alfalfa hay. Nutritionists formulate finishing rations with this cost in mind and also attempting to reach the highest net energy of production value they can achieve in the ration while keeping disorders like those previously described at a minimum. For the most part, finishing rations are formulated to keep cattle "at the edge", just short of falling over into large percentages of acidosis and bloat. A common belief is that a certain amount of digestive disorder resulting in death is to be expected if the cattle feeder is managing his nutritional program correctly, achieving the best gains while holding these disorders to a minimum. One thought which must be kept in mind, however, is that along with the acute cases which result in death losses that can easily be measured from an economic standpoint, subacute cases also exist which are detrimental to performance and reduce the efficiency and overall economic returns which could potentially produce a profit or increase profits. Losses such as these, which cannot be readily identified until the cattle are processed, are not easily measured in an economic sense.

Consideration of the physical characteristics of the roughage source will help to define the exact level of roughage necessary to provide roughage activity (RA). For example, long chopped hay provides more roughage activity than ground cubes which provide more RA than corn silage. These differences determine the buffering activity that may be derived from saliva and the physical stimulation of the rumen to increase motility.

Adaptation of Cattle to Finishing Rations

Elam (1976) observed that five periods exist in the feedyard when cattle are most susceptible to the conditions described in the previous sections. These include:

1. When cattle are starting on feed.
2. When moving cattle from one ration to a ration higher in concentrate and energy.
3. During weather changes.
4. During long periods on a finishing diet.
5. When cattle are extremely hungry or aggressive due to feeding problems.

Each of these times presents an opportunity for cattle to take in large quanities of feed with high levels of readily fermentable carbohydrates (RFC). Consumption of high concentrate diets is in reality an unnatural activity for cat-

tle which for the most part are conditioned to high roughage diets and subsequently possess a microbial population adapted to such a diet. In order to efficiently finish animals as is desired by the consumer, it becomes necessary to adapt cattle to diets high in concentrates. The adaptation process has been reviewed extensively in recent years (Tremere et al., 1968; Fulton et al., Huntington and Britton, 1979; Lofgreen and Kiesling, 1985).

The process normally used starts cattle at a relatively low level of concentrate (approximately 60%) plus availability of long hay to stimulate the ruminal environment, especially in highly stressed cattle which may exhibit reduced rumen motility. Energy and concentrate levels can then be shifted upward from this starting point. Elam (1976) noted that when raising the caloric density of a diet or when increasing concentrate percentage, it is important to make changes gradually. A safe rule has been to increase the net energy by no more than 10% at a time. Another precaution is to never change rations when cattle are hungry due to a lack of feed. In this situation it may be advantageous to feed the lower ration an extra day before changing to the new ration.

Although theories concerning ration change methodology differ significantly, one practice commonly ascribed to has been to split feed rations. In other words the lower ration is fed in the morning and the higher rations is delivered at the afternoon feeding, and following this regime for two or three days to more gradually accustom the ruminant system to the higher energy levels and amounts of RFC.

Another important factor to consider is that of potential causes of feed intake interruption. Anything that may cause cattle to go off feed is a precursor to a potential digestive disorder. A common cause is inclement weather such as snow, heavy rain, etc. When cattle turn away from the feedbunks for a day or more and return with an enhanced appetite the opportunity is good for the reoccurrence of acidosis or bloat. Another deterrent to normal consumption is that of spoiled or moldy feed. Cattle have an acute sense of smell and can readily detect spoiled feed which they will not eat. If this feed is left in the bunk for an extended period of time, cattle will become quite hungry but will not eat the moldy or spoiled feed. Here again, when fresh feed is delivered, the aggressive appetite of the cattle can cause them to overload on the feed and present a serious problem. In these situations it may often be beneficial to drop back to a ration with a lower energy level and more roughage and work the cattle back up instead of feeding the higher ration which can potentially initiate a digestive problem.

A final area which provides potential problems is that of cattle on feed for extended periods of time. The extended subjection to a high concentrate/energy feed tends to enhance subclinical disorders which manifest themselves as depression in intake, and performance in general. A common condition is that of founder or lameness which results in all probability from extended subacute acidosis. This condition restricts the animal's mobility and depresses performance as a result of its inability to get to the feedbunk and waterer as easily as before. In situations where cattle will be on feed for more than 150 days, it may prove beneficial to drop back to the lower energy ration for a short time to give the digestive system a rest and prevent the animal from "burning out".

Ration Formulation

When formulating rations, the nutritionist must keep many of the factors discussed in the previous sections in mind. Factors such as grain source, level of processing, fines produced by processing, and the ability of personnel to manage feedbunks properly are all major considerations when developing a ration system, keeping in mind that no two feeding facilities are the same. Other considerations include type of cattle fed, their point of origin and the type of diet the animals had been accustomed to previously.

Fulton et al. (1979) noted that different grains exhibit different rates of fermentation and extent of fermentation. Wheat has been identified as having the highest rate of fermentation and therefore the greatest capacity to induce acidosis and associated conditions. Britton (1981) rated feed grains in both processed and unprocessed forms by their rate of fermentability in the rumen. The following list ranks the grains from highest to lowest rate of ruminal degradation:

1. Wheat
2. Barley
3. High mositure corn (processed)
4. Steam flaked corn
5. High moisture corn (stored and fed whole)
6. Rolled corn, reconstituted milo, steam flaked milo
7. Whole shelled corn
8. Dry rolled milo

The use of grains with high levels of fermentability should require judicious consideration due to potential threat of acidosis and associated disorders.

Another consideration is the production of fines either by processing method or type of grain and processing method, etc. For example, dry rolled milo will produce a large amount of fines in the feedbunk. Also, mills which require extensive handling of feed will increase the level of fines produced in the ration. Since fines have been implicated in bloat as well as reducing intake, the percentage of this material should be kept at an absolute minimum.

These are a few of the factors which are involved in the role feed formulation plays in management of digestive disorders. Other factors also have an effect on how rations should be balanced.

Bunk Management

Bunk management (BM) can play one of the most important roles in the feedyard in terms of the control of digestive disorders, especially if done poorly. It is essential that the bunk manager maintain a good comprehension of the intake patterns of the cattle as well as have an understanding of factors affecting eating patterns. One primary responsibility of BM, in addition to taking every opportunity to encourage maximum feed consumption yet prevent excess waste of feed, is to ascertain that cattle never become excessively hungry. This means that cattle should never go without feed for an extended period of time; overnight for instance. If cattle are allowed to become exceptionally hungry, when feed finally is delivered, overconsumption may very likely occur, greatly increasing the possibility of severe digestive disorder. The bunk manager must coordinate his reading of bunks closely with his truck drivers in order to expedite the delivery of feed to cattle, especially those which may have completely cleaned up all previously delivered feed. "Slick" bunks are viewed by many as a potential problem and many feedyards will endeavor to prevent slick from occuring in a pen for more than 30 minutes per day.

III. Conclusion

Although digestive disorders are a serious problem in the feedyard, they are a problem which can be managed with a concerted effort between the manager, nutritionist and veterinarian. Every effort should be made to keep disorders of this nature to a minimum without sacrificing animal performance. By the same token, the desire for increased performance should not be so great that increased levels of cattle deaths become common and subclinical cases become widespread. A balance between performance and detrimental conditions needs to be attained at a level most suited to a given feeding operation.

In many of these situations the greatest imput the veterinarian can have is to correctly diagnose cause of death in order that the nutritional program can be fine tuned to compensate for the condition. It cannot be emphasized strongly enough that management of feedlot death is a group effort and as long as this thought is kept in mind, there is a possibility of greatly reducing losses.

References

1. Ahrens, F.A. 1967. Histamine, lactic acid, and hypertoxicity as factors in the development of rumenitis in cattle. Amer. J. Vet. Res. 28:1335. 2. Bailey, C.B. 1959. The rate of secretion of mixed saliva in the cow (Abstr.). Proc. Nutr. Soc. 18:13. 3. Beede, D.K. and S.D. Farlin. 1977. Effects of antibiotics on apparent lactate and volatile fatty acid production: in vitro rumen fermentation studies. J. Anim. Sci. 45:385. 4. Brittion, R.A. 1981. D-lactic acidosis - myth or fact. Univ. of Nebraska. Anim.Sci. Dept. Pub. 5. Brent, B.E. 1976. Relationship of acidosis to other feedlot ailments. J. Anim. Sci. 43:930. 6. Dennis, S.M., T.G. Nagaraja, and E.E. Bartley. 1981. Effects of lasalocid or monensin on lactate-producing or -using rumen bacteria. J. Anim. Sci. 52.418. 7. Dirksen, G. 1970. Acidosis. In: A.T. Phillipson (Ed.) Proceeding of the Third International Symposium on the Physiology of Digestion and Metabolism in the Ruminant. Oriel Press. New Castle, England. p. 612. 8. Dunlop, R.H. and P.B. Hammond. 1964. D-lactic acidosis of ruminants. Annals New York Acad. Sci. 119:1109. 9. Elam, C.J. 1976. Acidosis in feedlot cattle: practical observations. J. Anim. Sci. 43.898. 10. Essig, H.W. 1988. Bloat - nutritional problems related to the gastrointestinal tract. On: D.C. Church (Ed.) The Ruminant Animal - Digestive Physiology and Nutrition. Prentice Hall. Englewood Cliffs, New Jersey. P. 468. 11. Fell, B.F., M. Kay, E.R. Orskov, and R. Boyne. 1972. The role of ingested animal hairs and plant spicules in the pathogenesis of rumenitis. Res. Vet. Sci. 13:30. 12. Fulton, W.R., T.J. Klopfenstein and R.A. Britton. 1979. Adaptation to high concentrate diets by beef cattle. I. adaptation to corn and wheat diets. J. Anim. Sci. 49:775. 13. Gutierrez, J., R.E. Davis and I.L. Lindahl. 1961. Chemical and physical properties of a slime from the rumen of bloating cattle. Appl. Microbiol. 9:209. 14. Hironka, R. 1973. Influence of particle size of concentrate on rumen conditions in feedlot blood. Can, J. Anim. Sci. 53:75. 15. Huber, T. L. 1976. Physiological effects of acidosis on feedlot cattle. J. Anim. Sci. 43:902. 16. Hungate, R.E. 1966. The rumen bacteria. In: The Rumen and Its Microbes. Academic Press. New York, New York. 17. Huntington, G.B. 1988. Acidosis - nutritional problems related to the gastrointestinal tract. In: D.C. Church (Ed.) The Ruminant Animal - Digestive Physiology and Nutrition. Prentice Hall. Englewood Cliffs, New Jersey. P. 474. 18. Huntington, G.B. and R.A. Brittion. 1979. Effect of dietary lactic acid on rumen lactate metabolism and blood acid-base status of lambs switched from low to high concentrate diets. J. Anim. Sci. 49:1559. 19. Jensen, R. and D.R. Mackie. 1979. Bloat. In: Diseases of Feedlot Cattle. Lea and Febiger, Philadelphia, PA. p. 142. 20. Jensen, R. and D.R. Mackie. 1979. Rumenitis-liver abscess complex. In: Diseases of Feedlot Cattle. Lea and Febiger, Philadelphia, PA. p. 80. 21. Koers, W. C., R. Brittion, T.J. Klopfenstein and W.R. Woods. 1976. Ruminal histamine, lactate and animal performance. J. Anim. Sci. 43:684. 22. Lofgreen, G.P. and H.E. Kiesling. 1985. Effects of receiving and growing diets on compensatory gains of stressed calves. J. Anim. Sci. 61:320. 23. Multimore, J.E. 1070. Threshold fraction I (18-S) protein concentration for bloat and relationships between bloat, lipid, tannin, Ca, Mg, and Zn in alfalfa. Can. J. Anim. Sci. 50:61. 24. Muir, L.A. and A. Barreto, Jr. 1979. Sensitivity of streptococcus bovis to various antibiotics. J. Anim. Sci. 48:468. 25. Nagaraja, T.G., T.B. Avery, E.E Bartley, S.J. Galitzer and A.D. Dayton. 1981. Prevention of lactic acidosis in cattle by lasalocid or monensin. J. Anim. Sci. 53:206. 26. Nagaraja, T.G., T.B. Avery, E.E. Bartley, S.K. Roof and A.D. Dayton. 1982. Effect of lasalocid, monensin or thiopeptin on lactic acidosis in cattle. J. Anim. Sci. 54:649. 27. Orskov, E.R. 1986. Starch digestion and utilization in ruminants. J. Anim. Sci. 63:1624. 28. Rosen, W.G., H. Fassel, and R.E. Nichol. 1961. Etiology of legume bloat-nonvolatile acids. Am. J. Vet. Res. 22:117. 29. Ruckebusch, R. 1988. Motility of the gastro-intestinal tract. In: D.C. Church (Ed.) The Ruminant Animal - Digestive Physiology and Nutrition. Prentice Hall. Englewood Cliffs, New Jersey. p. 67. 30. Rumsey T.S. 1978. Effects of dietary sulfur addition and Synovex-S ear implants on feedlot steers fed an all-concentrate finishing diet. J. Anim. Sci. 46:463. 31. Tremere, A.W., W.G. Merril and J.K. Lossli. 1968. Adaptation to high concentrate feeding as related to acidosis and digestive disturbances in dairy heifers. J. Dairy Sci. 51:1065. 32. Slyter, L.L. 1976. Influence of acidosis on rumen function. J. Anim. Sci. 43:910. 33. Svendsen, P. 1973. The effect of volatile fatty acids and lactic acid on rumen motility in sheep. Nord. Vet. Med. 25:226. 34. Whanger, P.D. and G. Matrone. 1970. Effects of sulfur deficiency on metabolism in sheep. Sulfur in Nutrition Syposium. AVI Publ., Westport CT. p. 153. 35. Yokoyama, M.T. and K.A. Johnson. 1988. Microbiology of the rumen and intestine. In: D.C. Church (Ed.) The Ruminant Animal - Digestive Physiology and Nutrition. Prentice Hall. Englewood Cliffs, New Jersey. p. 138.