Nitrate and Urea Problems

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The objective of this treatise is to discuss the following questions relative to nitrate poisoning: What is nitrate poisoning? How does it affect animals? What can be done about it?

One of the oldest references to this problem can be found in the Old Testament. II Kings, Chapter 2, verse 19. The people of the city said to Elisha, "You can see how pleasantly our city is situated, but the water is polluted and the country is troubled with miscarriages." The history of veterinary medicine is one of attempting to learn the cause and treatment of disease. The most valuable information during my studies of nitrate problems was given to me by Dr. William Albrecht, an agronomist at the University of Missouri. He said, "Learn to study nature as well as books."

Nitrate poisoning is a misnomer. The actual damage to animals is caused by the oxides of nitrogen: nitrite (NO_2), nitric oxide (NO), nitrous hydroxide (NOH), nitroxyl $(HNO)_2$, and hydroxylamine (NH_2OH). Nitrate poisoning is caused by man. The buffalo which roamed this nation before the advent of domestication were not affected. The basic cause of all disease can be traced to an imbalance of the diet in one of the protein, mineral, or vitamin constituents.

Nitrates are important plant foods in the soil. All plants must utilize nitrogen from nitrates in the manufacture of plant protein. Until man learned to manufacture urea there was no way to get nitrogen into the soil for plant food except by crop rotation. The legume plants will fix nitrogen into the soil by using bacteria.

Four-fifths of the atmosphere is nitrogen. The nitrogen element has eight electrons in its outer orbit. This makes the element one which is capable of a large number of potential combinations. Nitrogen in its natural state is an inert gas. Nitrogen is the active ingredient in gun cotton, TNT, ammonium nitrate and other explosives. All proteins contain nitrogen. The nitrogen in proteins is present as NH_2 or an amino group. All amino acids contain nitrogen.

Amino acid metabolism depends upon a source of NH_2. Plant protein in the diet provides this source. The nitrates taken in by the

plant are reduced to the amino form of nitrogen by using the energy of the sun. This reduction of nitrate to ammonia requires the following factors: 1. Energy, 2. A hydrogen donor, 3. A nicotinamide coenzyme, 4. A flavoprotein, and 5. Mineral catalysts — Copper, iron, molybdenum, manganese, and magnesium. The reduction of nitrate to ammonia was investigated by Nason and Nicholas in 1959. This is the generally accepted pathway.

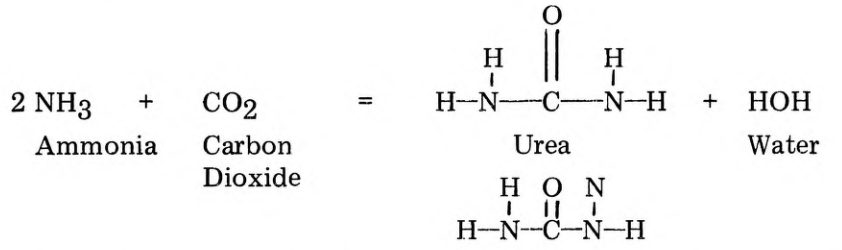
$$NO_3 \xrightarrow{Mb} NO_2 \xrightarrow{Cu} (HNO)_2 \xrightarrow[Fe]{Cu} NH_2OH \xrightarrow[Mg]{Mn} NH_3$$

Nitrate Nitrate Nitroxyl Hydroxylamine Ammonia

Ruminants are capable of reducing nitrates to ammonia in the rumen. The compounds which have a detrimental effect on the animal are absorbed by the animal from the rumen before they have been allowed to complete the reduction to ammonia. All oxides of nitrogen are poisonous.

The reason that urea must be included in a discussion of nitrate problems is the fact that urea is a good source of the NH_2 which the ruminant needs to manufacture amino acids. Urea is such a good source of NH_2 that the rumen will abandon the reduction process of NO_3 because the body wants to use the simplest route to prepare the raw materials for amino acid synthesis. If the animal has ingested nitrates and they have begun the reduction or conversion to ammonia, the intermediate compounds are present in the rumen. The animal which has an adequate nitrogen intake from a source other than nitrate will use the simplest source first and the intermediate or poisonous compounds in nitrate reduction are absorbed into the system by osmosis.

The compound urea is one of the finest examples of recycling. Urea is used by the body for a vehicle of nitrogen transport. The fish can eliminate protein wastes as ammonia. The water environment of the fish immediately dilutes ammonia so that it is not toxic. The land animal must have another system. Urea is simply the combination of two ammonia molecules with a carbon dioxide molecule.



The pathway of urea in the body may be traced. Urea is ingested. It may go from the rumen to the liver and be metabolized into an

amino acid. The amino acid is incorporated in the structure of a muscle. When the amino acid in the muscle is recycled the urea is returned to the liver for further use. If the liver is not in need of urea at that time, urea is sent to the salivary gland where it is once again returned to the rumen. If the salivary gland is incapable of more storage, urea is sent to the kidney where urea becomes the ingredient of urine which gives the ammonia qualities.

The addition of urea to a protein deficient diet is one of the cheapest sources of protein. The body must grow and repair itself in order for an animal to produce. Insufficient protein intake limits production. All veterinarians are familiar with the saying, "If a little bit is good a lot is better." This philosophy has become the thinking of virtually every feed source for our diets. Urea is the simplest and cheapest source of nitrogen for the ruminant. Everyone wants to sell it. It is added to beet pulp, molasses, minerals, protein mixes, grain rations, and all forms of liquid supplements.

When the farmer who listens to the salesmen of these various sources of ingredients for his ration does not understand the problem, he may very well buy urea in his mineral, in his liquid supplement, in his beet pulp, in his protein and combine all of these sources in the diet he feeds his animals.

When the animal is given an adequate nitrogen supplementation in addition to the nitrates present in the feeds and water, the animal begins the reduction of the nitrate in the ration but, because there is adequate NH_2 for metabolism without the nitrogen source of NO_3, the intermediate and poisonous oxides of nitrogen are also absorbed by the animal.

The root causes of many of the perplexing diseases we see in our daily practices can be traced to the stress on the animal by excess nitrogen or nitrate poisoning. The sudden appearance of any of these diseases should make an alert veterinarian look to the nitrate content of the ration.

Diarrhea is an attempt by the system to rid itself of undesirable elements in the ration. The bacteria in this case, as in most other bacterial diseases, are simply opportunists which take advantage of a stressed cell and invade and produce infection. Pneumonia, pinkeye, foot rot, liver abscesses, abortion, retained placenta, mastitis, and rickets are some examples of the type of disease which can have as its root cause the inclusion of nitrates in the ration. This same list of diseases may occur as a result of mineral imbalance and/or vitamin deficiency so that the wise nutritionist must be aware that any one of the three portions of the diet—protein, mineral, and vitamins—need to be considered.

There are four ways that nitrates can impede metabolism. They all happen simultaneously. In the clinical case they are all superimposed, one on the other. This is the basic reason that the nitrate problem is difficult to diagnose. One diet with a higher nitrate than a similar ration

on a different farm may cause no trouble. Several years ago laboratories got into heated arguments about what levels of nitrate were dangerous. No accurate dangerous levels can be accepted as hard and fast rules, because the mineral balance of the ration determines the ability to metabolize nitrates. This superimposing of two seemingly unrelated sets of problems has confused diagnosis and caused arguments.

The four actions of nitrates on metabolism are:

1. Reduction of NO_3 to NH_2 requiring energy.
2. Methemoglobinemia production.
3. Vitamin interference.
4. Impaired antibody production.

Reduction of NO_3

The ruminant is capable of utilizing nitrogen from virtually any source to form protein. The rumen is a reducing media. Reduction is that process which removes oxygen and replaces hydrogen on the nitrogen atom. This reduction process requires energy. In the plant this energy is supplied by the sun. In the animal this reduction is one of the most expensive aspects of the problem. When a cow is fed a ration containing nitrates they must be reduced before the nitrogen has any value to the animal. This reduction requires energy. Energy used in the conversion of NO_3 to NH_2 is lost to the animal for the production of meat or milk. It is actually possible for the reduction of the NO_3 portion of the plant to NH_2 to require more energy than is present in the plant. This can explain how an animal can be knee-deep in grass and starve to death. Some farmers who understand feeding state it another way, "The hay from that farm has no milk in it!"

Methhemoglobinemia

Nitrite or NO_2 combines with hemoglobin to form a stable compound. When hemoglobin is attached to nitrite the red blood cell cannot transfer oxygen to the tissues. The classic picture of nitrate poisoning or oat hay poisoning or cornstalk disease is due to the formation of methemoglobin. The reaction is similar to carbon monoxide poisoning.

Damage to a significant number of red blood cells results in death from anoxia. This syndrome can range from death to virtually no symptoms, depending upon the severity of methemoglobin concentration. The acute form of nitrate poisoning is of little consequence in our practice. Any animal which is killed suddenly from acute nitrate poisoning stops costing money so it is not a continuing liability like a cow which has her potential production ability impaired with a subacute, chronic or marginal form of poisoning.

The herd pattern becomes a reliable method in helping to diagnose the problem. Damaging the oxygen carrying capacity of the blood of a pregnant animal can result in abortion. The disease syndrome of a chronically poisoned animal is difficult to diagnose because some of the symptoms are so slight that they can be overlooked. Many cows will

not abort, but will deliver weak calves about ten days prior to the normal time of termination of pregnancy. These calves will usually not live. There is sometimes a significant absence of colostrum in these cows. The last few days of pregnancy are necessary to prepare the cow for a good lactation, and if the calf comes early, the cow will never milk as well as her genetic capability would allow.

The potential variation in clinical symptoms is infinite, ranging from virtually no visible symptoms to sudden death from anoxia.

Periods of stress on weaning feeder calves when a change in diet from grass and milk to a largely forage diet are very dangerous if the forage has a high nitrate content. Add the hazard of a NO_3 source from water and the problem is worsened. Many cases of "shipping fever" which do not respond to normal medication may, in fact, also be suffering from nitrate poisoning. Pneumonia is seriously complicated by the anoxia from methemoglobinemia.

Interference Sydrome

The third method of damage to an animal is called an interference syndrome.

When nitrates are ingested, regardless of whether they are in the forage or the water, a reaction interferes with the metabolism, first of the fat soluble vitamins, A, D, and E and secondly, diminishes the efficiency of the B vitamins. The interference syndrome is exhibited by symptoms of the vitamin deficiency and these symptoms will occur with varying degrees of severity again ranging from no visible symptoms to those of acute vitamin deficiency.

An example of this interference is the rickets syndrome which we know to be caused by either mineral deficiency or Vitamin D deficiency. It is possible for calves and foals to have rickets with adequate supply of both the minerals and the vitamins present in the diet when the diet also contains excessive nitrate.

The cow and the horse absorb only certain types of carotenoids which are in turn converted into vitamin A. The ability to absorb these carotenoids depends upon a specific protein which will form complexes with the carotenoids. This complex is then easily absorbed. If the complex is not formed because of a delicate "guide like" protein, the vitamin never reaches the necessary areas of metabolism in the body. Nitrogen is the key to all protein, and certainly an imbalance of nitrogen in the system is likely to affect the most delicate protein compounds first. The vitamin interference syndrome is not completely understood but the absence of vitamins which can be utilized by the system suggests a host of disease problems. Any symptoms which suggest a vitamin deficiency may also suggest a potential nitrate problem.

Antibody Impairment

The fourth and most insidious hazard to animals on a ration with a dangerous excess of NO_3 in the forage or water is an impairment of the

reticulo endothelial system, or the antibody manufacturing ability of the animal. All antibodies are very complicated and delicate proteins. Antibodies are very specific in their structure. The animal has no ability to defend itself against the ravages of pathogenic microorganisms and viruses without the action of antibodies. If the antibody manufacture is inefficient in the automatic process of metabolism, the animal is vulnerable to a host of infections not normally dangerous to the animal. Imbalance of nitrogen intake can affect antibody production.

The presence of nitrogen in various forms is known to be a powerful stimulator of bacterial activity. With increased numbers and kinds of bacteria and with poor antibody supplies the animal can succumb to an infinite variety of disease.

Malnutrition is one major cause of poor health. The inclusion of excess nitrates in a ration already well supplied with protein, and further complicated with urea, and drinking water which is polluted with various forms of nitrous oxides, leave no opportunity for the automatic processes of metabolism to defend against the inevitable four hazards of excessive nitrates in the ration mentioned earlier.

Toxic Levels

It is impossible to establish toxic levels and standards because the ability of the animal to metabolize nitrates is influenced by the mineral balance in the diet as well as the availability of the vitamins in the diet.

Any forage which has 0.5% KNO_3 is potentially dangerous. I would like the forage to contain less than 0.3% if it is to be fed to pregnant animals. 0.5% is equivalent to 5000 parts per million.

Water should contain less than 45 parts per million NO_3. Nitrates in water can be especially dangerous. There should be less than one part per million of the nitrite.

There are several things that a veterinarian can do for a client who has a nitrate problem. The first and most important thing is to realize that the problem exists. Find a reliable laboratory to test your feed samples. Learn to interpret the results of the laboratory analysis. Remember that water is also a part of the ration and always include a test of the water. We use the Brookside Laboratory in New Knoxville, Ohio 45871.

Remove as much of the non-protein nitrogen from the ration as possible until the feed analysis allows you to properly balance the ration.

Dilute known high nitrate feed by blending them with feeds of lower nitrate content.

Supply energy for two reasons:

1. Energy helps reduce nitrate.
2. High energy feeds usually have a low nitrate and thus blend or dilute the nitrate.

Balance the ration with adequate minerals and vitamins.