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Nutritional Problems In Cattle

Some common deficiencies

W. E. Peterson, B.S., M.S., Ph.D.*

As the knowledge of the nutritional requirements for farm livestock increases, it becomes increasingly apparent that the practicing veterinarian has a large role in the application of nutrition to his practice. No other factor is more important in economic production of livestock and livestock products than is adequate nutrition. Many cases of uneconomic production have been due to an insufficient quantity of feed, but an increasing number of cases are now showing a deficiency in some of the essential nutritional elements. The latter is becoming more common because of a number of reasons. With time, as the land is farmed, there is apt to be a depletion of soil elements, some of which are essential in nutrition. Livestock feed crops grown thereon suffer from deficiency. Breeding for increased production makes for more exact nutritional requirements; hence greater dangers of deficiencies. Use of more by-products as feeds also increases the danger of certain deficiencies; and lastly, the change in environment and management that accompanies forced production is in many cases a contributing factor.

Major Deficiencies

Adequate and full treatment of nutritional deficiencies would require a volume. In this short article an attempt will be made to consider in a general way the major nutritional deficiencies encountered among cattle. These deficiencies can be classified into the following categories: protein, mineral, and vitamin. Further division may include the deficiency problems of calves and older animals.

Undoubtedly the most serious problem in calf raising is one of nutrition. Faulty nutrition, directly or indirectly, is responsible for the most losses in calves. From recent experimental work, it is apparent that digestive disturbances in calves, while due in many cases to overfeeding of milk, are probably more often due to a lack of vitamins of which vitamin A, ascorbic acid, nicotinic acid, and pantothenic acid are often deficient. It has been known for some time that colostrum was essential to the young calf. The beneficial effects of colostrum were ascribed originally to its laxative effect and its content of antibodies, both of which are important; but of equal importance is its high concentration of vitamins. Vitamin A may be more than 50 times as concentrated in colostrum as in normal milk. This is of extreme importance as the calf has no vitamin A stores at birth, and the vitamin A in milk is proportional to the carotene content of the feed the cow receives. Furthermore, skim milk has a very low vitamin A content and its substitution for colostrum would be expected to produce an avitaminosis A.

It is obvious that artificial administration of the naturally deficient vitamins is indicated in the treatment or prophylaxis of calf scour. There is, however, a shortage of fish liver oils, the customary source of vitamin A; therefore, it is strongly urged that any excess colostrum beyond the needs of the calf be saved and kept in a frozen state until needed. At the first signs of digestive disturbance in a milk-fed calf, up to five pounds of the

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stored colostrum should be given at a feeding.

Protein deficiency is very common especially among dairy cattle who have a high protein requirement because of milk production. Growing cattle with grass hays and silage for roughage and corn for concentrate also suffer from protein deficiency. This is one of the chief reasons why legume hays are superior to grass hays. Dairy cows receiving two pounds of good legume hay per hundred pounds of live weight obtain enough protein for almost any level of production with any kind of concentrate.

The next most widespread deficiencies among cattle are iodine, phosphorus, calcium, cobalt, copper and iron deficiencies found in various parts of the country.

Iodine Deficiency
The chief symptom of iodine deficiency is goitrous new born calves. Treatment of the calf is symptomatic, and includes the administration of iodine in the ration of the herd. With very few exceptions iodized salt is adequate. In some severe iodine deficient areas additional iodine must be administered for best results.

Phosphorus deficiency is found in many parts of the country, and is becoming more extensive as soils become depleted because of continuous cropping. The deficiency is most apt to occur in milking cows that are fed largely on roughage which is normally low in phosphorus content, the grains having a high phosphorous content. The first symptoms of phosphorus deficiency are unthriftiness and decreased milk production accompanied with lower appetite. This is followed by a depraved appetite in which the affected animal shows a particular fondness for chewing bone, although wood and other objects are also vigorously attacked. In phosphorus deficient areas, it is not uncommon to see the mangers and all wood that can be reached gnawed, often necessitating replacement every few years. At this stage the blood phosphorus is usually less than one-half the normal value. Affected animals become stiff and lethargic. Pain in the limb articulations is manifested by the characteristic posture and reluctance to move. The bones become brittle and fractures are not infrequent.

In addition to the lowered milk production, reproduction is interfered with. Affected animals, even though they ovulate normally, may not manifest physiologic heat. It is customary for cows in phosphorus deficient areas to calve on an average of only once every two years. They are dry for about one-half of the time during which the phosphorus stores are replenished.

The treatment is to furnish phosphorus to the affected animals. Bone meal given ad libitum has been shown to be satisfactory as is any other phosphorus containing mineral. In phosphorus deficient areas bone meal, 60 percent and salt, 40 percent should be made available to all animals at all times. Application of phosphate to the soil should also be advocated, as such not only increases the phosphorus content of the crops grown thereon, but also greatly increases the yield.

Calcium deficiency among cattle is not common, as roughages furnish the basis of the ration and are as a rule adequate in their calcium content. Full-fed baby beeves on corn with low grade grass hays for roughage may be deficient in calcium, although not often to the extent that severe symptoms arise unless there is also a shortage of vitamin D. Bone meal or a calcium compound such as slaked lime will correct the deficiency. Here, again, legume roughages serve the purpose as they are rich in calcium.

Other Minerals
Cobalt, iron, or copper deficiencies have been reported in various parts of the country. In all cases the deficiency produces unthriftiness as a result of anemia. Aside from a certain area in Michigan where cobalt has been shown to be deficient, lack of any of these three minerals has not been reported in the northwest.

In a consideration of mineral deficiencies grass tetany must be mentioned. This disease has been reported to occur in Kentucky, Kansas, Nebraska, Oklahoma, and South Dakota after turning cattle

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out on lush pastures of wheat and rye. This has undoubtedly occurred in other places. The disease is highly fatal unless treatment is promptly administered. The first symptoms are nervousness, excitability and tetanic contraction of muscles with tonoclonic spasms. This is followed by coma which resembles parturient paresis, resulting in death unless properly treated. As the symptoms are caused by a lowered blood calcium and magnesium, dramatic recovery is affected by intravenous injection of a solution of calcium gluconate and magnesium chloride. Grass tetany may be prevented by forcing cattle which are on lush pasture to eat some hay every day.

Until recently it was thought that there was little danger of any vitamin deficiency in cattle. More recent investigations, however, have revealed that vitamin deficiency in cattle may be quite extensive and of large economic importance.

Vitamin A

That cattle need vitamin A in the diet has been known for some time, but until recently it was thought that practically all rations contained adequate amounts of provitamin A, the precursor of vitamin A. It has now been shown that cattle may suffer from insufficient amounts of this vitamin in extended areas. The chief sources of provitamin A (carotene) are in green pasture, well-cured hay and silage, and yellow corn. When the hay is bleached or browned, the carotene is destroyed; as a matter of fact, anything that causes a loss of the chlorophyll in the hay will also cause a loss of the carotene. The carotene, however, is destroyed by factors that may not destroy the chlorophyll. With time, the carotene is oxidized in both hay and corn so that even bright colored hay or yellow corn a year old or more is deficient in the provitamin A.

The first symptom of vitamin A deficiency is night blindness. Affected animals will be noted to stumble over objects in their paths during twilight. While vitamin A and carotene is stored in the body, it has been shown that within 60 days after removal from pasture, animals on a carotene-free diet develop night blindness. More advanced stages present keratitis which is often mistaken for infection, but does not respond to symptomatic treatment. Unless treatment is administered, complete and permanent loss of sight occurs. In most cases the early eye complications are accompanied by edema of the legs, brisket, underline, and shoulders. The latter condition has been noted in beef cattle fed old corn and hay low in carotene content. Reports show that carcasses from animals so affected are condemned.

The recent observation that vitamin A deficiency lowers reproductive efficiency in both the male and the female, explains in part the great increase in breeding difficulties toward the end of winter.

Low carotene in the ration is now known to be responsible for the troublesome off-flavor in winter milk known as oxidized or cardboardy flavor.

In prolonged and severe vitamin A deficiency in pregnant cows the calves may be born prematurely or at term but blind, dead, or so weak that they do not survive. In less severe deficiencies the calves may appear normal at birth, but do not survive when placed on their mother's milk. The amount of carotene or vitamin A in milk is dependent upon the provitamin in the feed. With the exception of the complete loss of sight and death of calves at birth all other symptoms of vitamin A deficiency are completely relieved by the administration of vitamin A or hay with a high carotene content.

Vitamin B

Insofar as is known, the ruminant synthesizes all of the vitamin B complex in the rumen. In young calves where the rumen is not functional, the B vitamins must be supplied in the diet and are sometimes deficient as has previously been stated. Reports from Sweden that thiamin (vitamin B1) proved beneficial in the treatment of acetonemia have not
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been confirmed, but suggests the possibility of inadequate vitamin B synthesis in the rumen under certain conditions.

**Vitamin C**

Vitamin C (ascorbic acid) is synthesized in the bovine, but is sometimes deficient, producing sterility in both the male and the female. No other symptoms from vitamin C deficiency have been reported. Recent work indicates that the deficiency in some cases, at least, is associated with inadequate amounts of vitamin A. In such cases the vitamin C content of the blood is restored to normal levels with the oral administration of vitamin A. As ascorbic acid is destroyed in the rumen, oral administration is contra-indicated, and it should be administered parenterally.

**Vitamin D**

The bovine needs vitamin D which is normally obtained from sun-cured roughage during the winter and from direct exposure to the sun’s rays during the pasture season. It is known that grains are devoid of vitamin D and deficiencies of sufficient magnitude to produce marked symptoms are found when large amounts of grain are fed with little or no sun-cured roughage, and also when the amount is not exposed to sunlight. This condition has been observed in steers during the winter when allowed all the grain they wanted and a low quality hay, of which they ate little or none. The first symptom is swollen and painful joints. The soreness rapidly becomes so great that the animal refuses to get up; simultaneously, there is a complete loss of appetite. Erosion of the articular surfaces of the bone is accompanied by infection with pus formation and irreparable damage. Other symptoms are quickly relieved by the administration of vitamin D, including restoration of the appetite.

Recent reports indicate that massive doses of vitamin D (4,000,000 units per day) for 30 days prior to parturition prevents the onset of parturient paresis in susceptible cows. Since milk fever is caused by a hypocalcemia and vitamin D is known to mobilize calcium in the blood, the prophylactic effect of vitamin D seems plausible.

**Vitamin E**

A great deal has been said about the value of vitamin E (alpha-tocopherol) in relation to sterility. There is not as yet a good, well controlled experiment to suggest the value of vitamin E in the treatment of sterility. Experiments now in progress indicate that cattle do not need vitamin E either for their physical well-being or for reproduction.

**AUJESZKY’S DISEASE**

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has presented experimental evidence that swine may serve as the source of infection for cattle. He showed also that fatal infection can be induced in rabbits by bringing their abraded skin in contact with the snouts of infected pigs, and he thought it possible that cattle may become infected in a similar manner. Shope visualized a cycle of infection from rats to pigs (he showed that carcasses of infected rats were eaten voraciously by pigs which as a result became infected), to cattle, and back to rats (the rats and/or pigs feeding on carcasses of dead cattle).

If this mode of transmission is accepted, an explanation of the infection of the cattle in Lot 1 in the case reported is made possible. It is not likely, however, that the deaths of the pigs previous to the infection of the cattle were due to Aujeszky’s disease, since it is rarely fatal to this species except in baby pigs, and animals affected exhibit only a transient depression and inappetence. On the other hand, the pigs may have been affected with the disease and since the virus is present in the nose of a hog it is likely that the cattle lying about the barn lot may have become infected by coming in contact with the noses of the pigs. Cattle lying about a barn lot in which hogs are also kept frequently come in contact with the noses of pigs. Swine under such conditions can be observed to approach a cow and probe

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