Nutritional Diseases of Cattle

BY LOUIS L. MADSEN

A DISCUSSION of the symptoms, causes, prevention, and treatment of disorders due to mineral deficiencies, vitamin deficiencies, and certain other conditions associated with nutrition in cattle.

FAULTY NUTRITION is often responsible, directly or indirectly, for large economic losses in the cattle industry. Recognition of this has stimulated research in this field, and much is being accomplished. It is becoming evident that the so-called nutritional diseases that occur under practical conditions on the farm or range are not always fatal. Undoubtedly, greater losses result from decrease in rate of growth and economy of production, reproductive failures, and increased susceptibility to certain infectious and parasitic diseases than from death. The vital role of nutrition in maintaining health in cattle and other farm animals has been recognized for many years, and this accounts for the careful attention given by livestock producers and veterinarians to providing an adequate diet as a part of the program for the treatment or prevention of many diseases.

In feeding studies much attention is given to comparing the rate of gain or the economy of production of milk or butterfat in animals fed certain feeds or feed combinations. Extensive tables of feed values are being compiled, and it is becoming increasingly evident that the value of a feed for any given purpose depends on its content of essential nutrients, which can now be determined to a large extent by chemical analysis. With this knowledge at hand it is possible to supplement deficient rations with the necessary essential nutrients—minerals, vitamins, or protein—and obtain good results with feeds which, when fed alone, have a relatively low value or even cause nutritional diseases. Knowledge of the essential nutrients has also

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led to improved methods of growing, harvesting, storing, and processing feeds in order to increase or preserve nutritive values. Recognition of these facts has often made it possible for a thriving cattle industry to be built up in sections where cattle feeding for either dairy or beef purposes was not entirely successful because of some rather obscure factors that limited the growth or production or affected the health of the animals.

Some nutritional diseases are due to a deficiency of essential nutrients and others to overfeeding or dietary imbalance or the presence of toxic substances in the feed. Characteristic symptoms of many nutritional diseases of cattle are now well established, and by study and observation livestock men and others concerned can learn to recognize these conditions and overcome them before serious losses result. Prevention, however, is the most successful means of control.

Before the common nutritional diseases are considered, it is important to emphasize that in many cases the results obtained in overcoming them have been so dramatic that some minerals and vitamins have come to be popularly regarded as almost in the nature of stimulating drugs or medicines. This is a mistaken point of view and has often led to unnecessary expense and disappointment when more of the essential factors are added to an already adequate ration. Results from increasing the intake of essential substances are obtained only when the ration is deficient in them. Furthermore, when a ration is deficient in several essential nutrients, all of them must be supplied for optimum results.

THE MINERAL-DEFICIENCY DISEASES

The minerals which have received the most attention in cattle-nutrition studies are phosphorus, calcium, magnesium, sodium and chlorine, iodine, iron, copper, and cobalt. Other minerals such as fluorine and selenium have been investigated largely from the standpoint of their toxicity. The mineral-deficiency diseases and the conditions caused by toxic minerals usually produce fairly typical symptoms and gross alterations in the body tissues. Blood and tissue analyses as well as microscopic examination of tissues are further aids to diagnosis.

CALCIUM AND PHOSPHORUS DEFICIENCY

The first symptom usually noted in cattle receiving too little phosphorus or calcium is a depraved appetite (pica), particularly evident in phosphorus deficiency (51). Phosphorus-deficient cattle frequently chew and eat bones (osteophagia) or other material such as wood, hair, putrid flesh, soil, rocks, leather, feathers, etc., in an effort to get additional phosphorus (fig. 1). Consumption of putrid bones, flesh, and other carcass material may lead to toxic symptoms and death from botulism if the material is infected with Clostridium botulinum, as is often the case. Loin disease of cattle in Texas and lamsiekte of sheep and cattle in South Africa are

*Italic numbers in parenthesis refer to Literature Cited, p. 670.
caused by toxin obtained in putrid material consumed in an effort to satisfy the craving for phosphorus. As the deficiency progresses, there are marked loss of appetite, emaciation, lowered efficiency of food utilization, weak bones, stiff joints (creeps), decreased milk flow, failure to come in heat normally, small calf crop, and a generally unthrifty appearance.

When laboratory facilities are available, phosphorus deficiency can be diagnosed either by analyzing the forage accessible to the cattle or by blood analysis for phosphorus. A large decrease in total

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_Figure 1._—Phosphorus-deficient cattle often chew bones and other nonfood material in an effort to remedy the deficiency.

inorganic phosphorus in the blood is the first indication of phosphorus deficiency.

Calcium deficiency in cattle, including lactating dairy cows, is very rare, especially when the animals are fed rations containing liberal amounts of roughage. Isolated cases of calcium deficiency may occur, however, under unusual feeding conditions. Becker, Neal, and Shealy (9) report that dairy cows on low calcium rations frequently suffered bone fractures and gave less milk than when adequate calcium supplements were fed. The calcium-deficient cows remained in good condition, however, and did not show lethargy and stiffness, which is characteristic of advanced phosphorus deficiency. Adult cattle usually show only a slight decrease in blood calcium when they are on calcium-deficient rations; younger animals show a greater
decrease, but it is practically never as great as the decrease in blood phosphorus in phosphorus deficiency.

An inadequate phosphorus or calcium intake over a long period leads to a gradual decrease in the body reserves of these elements. The largest reserve of calcium and phosphorus is in the bones, and if abnormal demands are made on this storage depot to meet the requirements of the body, the deficiency symptoms described may gradually develop. In general, during periods of rapid growth and heavy lactation cattle use more calcium and phosphorus than at any other time. Dairy cows frequently secrete more calcium and phosphorus in their milk during periods of heavy lactation than is obtained from the feed consumed. The extra calcium and phosphorus come from the bones. Any deficit is usually made good later when the milk flow declines or during the succeeding dry period.

Phosphorus deficiency is much more common in cattle than calcium deficiency. Cattle grazing in range areas or on farm pastures may develop phosphorus deficiency during periods of drought, especially in regions where the available soil phosphorus is rather low. When the feed supply is restricted owing to drought, overgrazing, or other causes, there may be a phosphorus shortage that would not ordinarily develop if ample forage was available.

In some sections of the country many dairy and beef cattle are fed primarily on roughage, such as alfalfa, with little or no grain to supplement it. Alfalfa is not usually rich enough in phosphorus to be depended upon as the sole source of this mineral, particularly if it is grown under conditions of relatively low soil fertility and low soil moisture. Most hays are even lower in phosphorus than alfalfa. In sugar-beet-producing areas, beet pulp is fed in addition to hay. Any beet byproduct is very low in phosphorus, as is corn silage; so a combination of either one with hay is apt to lead to a deficiency of this mineral. Wintering animals in stubble fields with free access only to cereal straw, corn fodder, prairie hay, or other similar low-phosphorus feeds may also lead to phosphorus deficiency unless some cereal grains, protein concentrates, or a phosphorus-rich mineral mixture is supplied. Pregnant and lactating cows or young growing animals are more susceptible to phosphorus deficiency than mature stock cattle.

**Prevention and Treatment**

Calcium and phosphorus deficiency can usually be avoided or overcome by giving animals an adequate supply of these minerals through the use of feeds that are good sources, the feeding of calcium- and phosphorus-rich mineral supplements, or increasing the mineral content and yield of available forage by fertilizing the soil on which the crops are grown.

Investigations of the Bureau of Animal Industry in cooperation with the Texas Agricultural Experiment Station and the King Ranch in south Texas have shown that many cattle in the Gulf coast area graze on phosphorus-deficient or semi-phosphorus-deficient ranges. Preliminary results obtained in these experiments by hand-feeding a mineral mixture rich in phosphorus to beef cattle on phosphorus-
deficient range are given in table 1. The phosphorus supplements consisted of bonemeal and disodium phosphate. They were fed individually so as to supply the dry cows with 6.5 grams of phosphorus and the lactating cows with 14.3 grams of phosphorus per head daily, in addition to that received from range forage. It is obvious from the data that feeding a phosphorus supplement costing $2.60 per cow per year raised the calf crop considerably, improved the growth rate of calves, and increased the value of the calves at weaning time by an average of $10.44 and that of the yearling heifers by an average of $12.19. It is obvious also that adequate feeding of existing herds is a rapid and economical means of increasing beef production.

<table>
<thead>
<tr>
<th>Item</th>
<th>Cattle fed phosphorus supplement in addition to the range forage</th>
<th>Cattle on range fed no additional phosphorus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calf crop (2-year average).</td>
<td>83</td>
<td>58</td>
</tr>
<tr>
<td>Cows producing calves for 2 consecutive years. do</td>
<td>72</td>
<td>21</td>
</tr>
<tr>
<td>Average weaning weight of calves. pounds</td>
<td>499</td>
<td>421</td>
</tr>
<tr>
<td>Average weaning weight of calves prorated to all cows... do</td>
<td>448</td>
<td>330</td>
</tr>
<tr>
<td>Value of calf at weaning time 2</td>
<td>39.92</td>
<td>29.35</td>
</tr>
<tr>
<td>Weight of heifers at 1 year of age. pounds</td>
<td>541</td>
<td>441</td>
</tr>
<tr>
<td>Value of yearling heifers less cost of supplement fed from weaning to 1 year 3</td>
<td>43.06</td>
<td>30.87</td>
</tr>
<tr>
<td>Annual cost of phosphorus supplement per cow... do</td>
<td>2.60</td>
<td></td>
</tr>
</tbody>
</table>

1 Data obtained by Bureau of Animal Industry, Texas Agricultural Experiment Station, and the King Ranch, Kingsville, Tex., cooperating.
2 Calves from cows fed phosphorus supplement were valued at 8 cents a pound and those from the cows that received no additional phosphorus were valued at 7 cents a pound.

Table 1.—Preliminary comparisons of gains and other production data on cows and calves fed a phosphorus supplement and on similar cattle receiving only range vegetation

Under practical range conditions, phosphorus supplements are usually fed free choice. Soluble phosphorus salts such as disodium phosphate, may be given in the drinking water if the source of water can be controlled. The experience of H. Welch, of the Montana Agricultural Experiment Station, in supplying range cattle with phosphorus supplements indicates some of the problems involved. He writes:

We have been using bonemeal and other phosphorus-containing supplements for approximately 20 years in the Montana phosphorus-deficient areas. Feeding bonemeal without salt has 2 principal drawbacks. One is that when put out in a box for range cattle, it blows out badly in windy areas: salt helps to bind it together and prevents this blowing. Second, in any bunch of bone-chewing cattle there are several individuals that refuse to eat bonemeal at all unless mixed with salt. Another objection to pure bonemeal is that after it becomes damp the flies will soon deposit eggs, and the bonemeal will become a mass of fly larvae. However, some cattlemen do use the bonemeal in separate compartments of the salt box.

Extensive experiments with disodium phosphate, either straight or mixed with salt, in our hands, have not been as satisfactory as with bonemeal. As far as we can tell, the disodium phosphate is tasteless, but the cattle almost universally found it unpalatable. The same was true of monocalcium phosphate, of

3 Communication to the Animal Nutrition Division, Bureau of Animal Industry.
phosphate rock origin, containing up to 45 percent P₂O₅, which proved entirely unpalatable to cattle on account of its acid taste.

I mention all these things because it seems to the casual observer that an existing condition of phosphorosis [phosphorus deficiency] can be remedied by the use of bonemeal, and that that is all there is to it. In actual fact, however, there are some difficulties in entirely remedying the situation, because of the difficulties encountered in getting an adequate dose of the supplement into the cattle.

Mineral mixtures fed on the range or in farm pastures should be placed in well-constructed boxes that protect the contents as much as possible from blowing out or being wet. A satisfactory mineral box is shown in a recent publication from the Florida Agricultural Experiment Station (48).

The phosphorus requirement of lactating dairy cows needs special attention, particularly in areas where there is a deficiency of this mineral in the soil and consequently a low content in the pasture grass and harvested forage. If the cows are fed a liberal amount of grain, including a protein-rich concentrate such as wheat bran, cottonseed, linseed, or soybean meal, and a roughage, their phosphorus and calcium intake is usually adequate. For very heavily producing dairy cows, a mineral mixture of bonemeal and salt or some other calcium-phosphorus supplement may be added to the grain mixture at the rate of 1 to 2 percent when necessary. There is little experimental evidence indicating that increased amounts of calcium in the form of mineral supplements have proved beneficial for either growing or lactating cattle when the animals were receiving liberal quantities of feeds of normal composition.

Lactating animals fed roughage with little or no grain should have access to a mineral mixture such as bonemeal alone or 1 or 2 parts of bonemeal to 1 of common salt, according to the appetites of the animals, in order to insure an adequate phosphorus intake. Ground rock phosphate, either raw or as a superphosphate, is likely to contain too much fluorine and arsenic and should not be used in livestock feeding. Purified phosphate salts are available. Dicalcium phosphate and disodium phosphate are probably the most widely used forms of phosphate salts, but they have often proved to be rather unpalatable to cattle and are therefore most successfully used mixed with salt or as a constituent of a grain mixture. Bonemeal and the other calcium-containing salts mentioned also provide an abundance of calcium, and when these supplements are used it is not necessary to add additional calcium in the form of calcium carbonate. If a ration needs the addition of calcium only, a good grade of ground calcium carbonate or oystershell is usually the cheapest source of this mineral.

**Phosphorus Deficiency and Parturient Hemoglobinemia of Dairy Cows**

Madsen and Nielsen (25, 26) have described a disease of high-producing dairy cows which most frequently occurs 2 to 3 weeks after the birth of the third to the sixth calf and is characterized by anemia (decrease of red blood cells), hemoglobinemia (hemoglobin
Nutritional Diseases of Cattle

651

or red blood pigment in solution in the serum), hemoglobinuria (hemoglobin in the urine), and general weakness. The disease is common in the intermountain areas of the United States and has been reported from Norway, Sweden, Denmark, Finland, Austria, Ireland, and Scotland. It may be confused with other conditions causing hemoglobinuria.

Madsen and Nielsen show a definite relationship between the occurrence of the disease and the ration fed. The disease is most common during the winter, especially when the cows are fed on alfalfa and sugar-beet pulp. No cases have been observed in beef cows. Other characteristic symptoms are loss of appetite, decrease in milk production, rapid pulse, cold and blanched teats, drooping ears, gaunt appearance, weakness, staggering gait, constipation or diarrhea, dehydrated appetite, increased water consumption, an odor of acetone on the breath, and, in some cases, milk tinted with hemoglobin. Cows that do not die of the disease usually start to recover after the fifth day of illness, and there is partial or complete recovery in a period varying between a week and several months. Cows fed liberal amounts of grain usually recover faster than those kept on their original ration of alfalfa and beet pulp.

The principal blood findings in this disease are a marked reduction in red blood cells and a very marked drop in inorganic phosphorus content. As already noted, the blood plasma is often tinged with hemoglobin. Treatment consisting of intravenous injection of dibasic sodium phosphate followed by drenches with the same material or with bonemeal have been quite successful. No cases have been observed by these authors where cows were liberally fed with grain during the lactation period. Regular feeding of a mineral supplement such as bonemeal is also indicated, particularly with low-phosphorous feeds such as beet pulp.

MAGNESIUM DEFICIENCY IN CALVES

When young calves are fed on milk without forage or a grain supplement for more than a few weeks, they invariably develop a severe disorder characterized by irritability, nervousness, and loss of appetite. In later stages the animals have convulsions. Duncan and associates (14) in describing this symptom state:

The animal, apparently temporarily blinded, will run into obstacles, or becoming confused will turn in circles until its balance is completely destroyed. As the convulsions become more violent, the calf may fall on its side with the legs rigid and alternately extended and contracted; there is frothing at the mouth and profuse salivation. These attacks may last continuously for several minutes or, intermittently, for a longer time. Young calves seem to be able to withstand several such convulsions but older calves usually succumb to the first attack.

Such symptoms are associated with low blood magnesium. Moore and coworkers (31), in connection with the Michigan work on magnesium, have described the lesions (tissue changes) in calves fed rations low in magnesium. The principal findings were calcification of the yellow elastic fibers of the endocardium (membrane lining the interior of the heart), in the walls of the larger veins and arteries,
on the surface of the diaphragm, and in the trabeculae (fibrous bands extending into the interior) and capsule of the spleen. Degeneration and calcification were also found in the Purkinje system (conducting fibers) of the heart. Kidney and liver damage was also found in some cases. These workers suggest a possible relationship between diets low in magnesium and arteriosclerosis of human beings. (Typical blood-vessel lesions found in calves suffering from magnesium deficiency are shown in fig. 2.)

Ordinary diets of forage and grains contain enough magnesium that disorders due to magnesium deficiency are not likely to occur in calves under practical conditions unless they are fed too long on milk without access to supplementary feeds. Feeds rich in magnesium
are legume and grass hays, cottonseed, linseed and soybean meal, wheat bran, wheat middlings, beet pulp, and hominy feed. Huffman and Duncan (22) report that 15 to 20 milligrams of magnesium as magnesium carbonate (MgCO₃) or magnesium oxide (MgO) per pound of body weight maintained plasma magnesium values and prevented the manifestations of magnesium deficiency. Under the same conditions 8 to 10 milligrams of magnesium from natural feeds per pound of body weight was equally effective, indicating that the magnesium of natural feeds is more readily utilized by calves.

**GRASS TETANY**

Grass tetany is a highly fatal disease of cattle characterized biochemically by lowered blood magnesium and calcium. It occurs most often within 2 weeks after the animals have been turned out to good pasture. The disease has received more attention in Holland, Great Britain, and New Zealand than elsewhere, although it is known to occur also in the United States (28, 45). A somewhat similar condition has been described in sheep and horses.

SjoUema (49) describes this condition vividly:

Usually the initial signs are nervousness, restlessness, grazing apart from the herd, lack of appetite, muscle-twitching, unsteady gait, spreading of the hind limbs, grasing of the teeth, rolling of the eyes, an anxious or wild look resulting from abnormal eye muscle contraction, frothing at the mouth and abundant salivation. Some animals low continuously. The ears stand erect, and certain muscles, e.g. those of the tail, are in tetanic contraction. Abnormal contraction of the neck muscles causes a backward thrust of the head and similar contraction of the masticatory muscles results in trismus [lockjaw] . . . clonic-tonic convulsions [alternate rigidity and relaxation] appear, and the animal strikes heavily with all four limbs. During these cramps the animal is in a state of intense excitement, and alternately falls down and gets up with a jerk, the attempt to rise being often unsuccessful. The animals also run against walls, into ditches, etc., violent cramps being easily induced. The excitement is often followed by a paretic [paralytic] or comatose state of depression.

The cause of this condition is still obscure, but it is probably not a simple magnesium deficiency, since pasture grass is a good source of this mineral. Young, fast-growing pasture grass on well-fertilized pastures is suspected by some to contain an unbalanced content of minerals or toxic products which cause the disease. SjoUema (49) believes that heavy grain feeding in winter, together with limited roughage consumption, predisposes cattle to this disease, since such rations are usually low in calcium and high in phosphorus. A condition called grass tetany is reported by Quin (45) to occur in Kansas, Nebraska, and Oklahoma among cattle turned out to young lush pastures of rye, wheat, or barley; no blood studies are reported. Metzger (28) states that, with few exceptions, the cases reported in Kentucky occur in cows grazing on young, rapidly growing grass, and the record rainfall during the spring of 1935 caused a marked increase in the number of cases.

Since the disease is often rapidly fatal, there may not be sufficient time for treatment. Intravenous injection of a solution containing calcium chloride and magnesium chloride, which of course requires
the services of a qualified veterinarian, has given good results (49). After the injection, it is advisable to keep the animal quiet and feed it only dry hay. Taking cattle off pasture at night for the first 2 weeks or so and giving them a feeding of hay has been recommended as a preventive measure.

**SALT (SODIUM CHLORIDE) DEFICIENCY**

Such common feeds as forages, grains, and grain byproducts ordinarily do not contain enough sodium and chlorine to meet the needs of farm animals. Milk production, hot weather, and hard work with excessive sweating increase the salt requirement. Symptoms of salt deficiency in dairy cows are loss of appetite, salt hunger, rough hair coat, and a rapid decline in condition and milk yield. Special attention should be given to providing an adequate supplement of salt, which may be supplied in granulated, flake, rock, or block form. Animals will usually consume more in the granulated or flake than in the solid form. Potassium or sodium iodide when needed, bone-meal, and ground limestone are frequently mixed together with salt and fed free choice or included in the grain mixture. On the range, boxes containing salt or general mineral mixtures should be protected from rain as much as possible, and enough boxes should be supplied so that even the very timid animals will have ample opportunity to obtain some. Animals that have been deprived of salt for a considerable period should not be given all they will eat at once, but should first be hand-fed limited amounts for several days, or harmful results may follow.

**POTASSIUM DEFICIENCY IN CALVES**

Salts of potassium are also required by the body for normal nutrition, but any practical diet usually contains an ample quantity of this mineral. Experimental semipurified rations containing 0.10 to 0.12 percent of potassium have been fed to calves, beginning at 160 days of age, by Sykes and Alfredson (50). They report finding lowered serum potassium, which promptly increased when additional potassium was fed, while the calcium, phosphorus, and magnesium values were not altered from normal. A striking result of their studies was the finding that three of the four calves used developed marked changes in the heart, determined by the electrocardiograph. Histological (cell) changes were also found in the Purkinje system (conducting tissue) of the right and left ventricles.

**THE TRACE MINERALS IN CATTLE NUTRITION**

*Iodine Deficiency and Goiter*

Iodine deficiency in cattle nutrition, as in that of other animals, is largely a sectional problem. The areas of the greatest iodine deficiency are in the Northwest and in the North Central States around the Great Lakes. The symptoms and prevention of this nutritional disease in cattle and other livestock are discussed in the article on Nutritional Diseases of Farm Animals, page 332.
Nutritional Anemia (Iron, Copper, and Cobalt Deficiency)

For many years a condition occurring in cattle in Florida, known as salt sick, and a similar or identical malady in Michigan, called Grand Traverse, or lake-shore disease, have caused considerable loss to the cattle industry in certain areas of these States. Affected animals gradually lose their appetites and become emaciated and weak. Some have a depraved appetite. Diarrhea or severe constipation may be present. The blood is pale, owing to its low content of hemoglobin (red blood pigment). Young cattle fail to grow normally and if they survive do not reach normal adult size. Sexual maturity is delayed, and reproduction and lactation are very unsatisfactory. After death the liver, kidneys, and other tissues are pale, and the heart muscle is flabby. The disease is most prevalent in young cattle over 6 months of age and in heifers calving for the first time. Cattle of all ages and both sexes may be affected when the animals are restricted to deficient ranges.

Recent studies indicate that salt-sick animals are frequently suffering from a deficiency of one or more minerals, and possibly other factors also. Copper deficiency and cobalt deficiency may occur separately or overlap in some areas. In Michigan the condition is due essentially to cobalt deficiency.

Neal and Ahmann (23), of Florida, describe the symptoms of cobalt deficiency in calves (fig. 3) fed a ration of natal grass hay, shelled corn, and dried skim milk as follows: Affected cattle usually

Figure 3.—These two purebred Jersey bulls were fed a ration of natal grass hay and shelled corn from cobalt-deficient land, with skim milk. The addition of 5 milligrams of cobalt daily to the ration of the animal on the left resulted in its weighing 550 pounds at 15 months of age, while the maximum weight of the animal on the right at 16 months of age was 350 pounds. (Courtesy of W. M. Neal, Florida Agricultural Experiment Station.)
show a long rough coat of hair, scaliness of the skin, listlessness, retarded development of sexual characteristics, gauntness due to loss of appetite (much less marked when liquid skim milk is a part of the ration), and muscular atrophy. The erythrocyte (red blood corpuscle) count may be above average, and the hemoglobin concentration equal to or above that in animals receiving cobalt and making normal growth. The amount of hemoglobin per erythrocyte, or per volume of erythrocytes, is reduced. The condition would be classified as a microcytic hypochromic anemia (an anemia characterized by undersized red blood corpuscles and too little hemoglobin). The spleen is shriveled and fibrous and the heart of normal size but very flabby.

Copper deficiency also causes nutritional anemia, loss of appetite, slow growth, lowered fertility, and occasionally bleaching of the hair coat in cattle. Further studies will undoubtedly yield additional information on how deficiencies of copper and cobalt, and possibly of other trace minerals, may be differentiated.

Workers at the Florida experiment station have demonstrated that the forage and soil are usually lower in iron, calcium, magnesium, and phosphorus in areas where salt sick occurs than in areas where cattle are not affected with nutritional anemia (35). Iron deficiency has been thought to be a cause of this disease, but recent studies tend to give more weight to the possibility of a deficiency of cobalt or copper or both. In Michigan the cobalt content of the hay grown in affected areas is considerably lower than that of hay from farms where the disease does not develop.

Considerable success has been attained in treating and preventing anemia in cattle by supplying iron, cobalt, or copper salts along with common salt for use as a lick. Advanced cases have also been successfully treated by drenching animals with these salts in solution. There is some indication that the responses obtained from feeding iron salts are due at least in part to the cobalt impurity which these salts frequently contain. Cases of Grand Traverse disease in Michigan respond well to a cobalt supplement alone (3).

In Florida supplements of 5 to 10 milligrams of cobalt a day markedly improved the growth of calves on a cobalt-deficient ration. Calves fed supplements of ferric ammonium sulfate and copper sulfate developed deficiency symptoms sooner than individuals on the basal ration only, while those receiving supplements of cobalt or of iron, copper, and cobalt appeared normal (34). Cobalt deficiency in the feeds of Florida is associated with certain soil types (Norfolk series) low in cobalt; it may also be associated with copper deficiency.

Whenever salt-sick cattle fail to respond to the usual supplement of iron and copper, it has been recommended by the Florida Agricultural Experiment Station that cobalt be added to the mineral mixture.

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*Communication to the Animal Nutrition Division from W. M. Neal, of the Florida Agricultural Experiment Station.

For advanced cases the following treatment is recommended:

Dissolve 10 grams of cobalt chloride or cobalt sulfate in one gallon of water. Give mature animals 6 ounces as a drench once weekly for three or four weeks. Calves should be given 3 ounces, and other animals in proportion.

For prevention of the disease:

Add one pound of cobalt chloride or cobalt sulfate to each ton of the regular salt sick mixture (100 lbs. of salt, 25 lbs. of red oxide of iron, and 1 lb. of powdered copper sulfate). A recommended manner of making this addition is to dissolve 22 grams of the cobalt salt in a small amount of water and spray it over 100 lbs. of the salt sick mineral with a fly sprayer or atomizer. It should be mixed thoroughly so that animals taking it will secure a uniform amount. This should be offered in protected mineral boxes at all times.

**TOXIC MINERALS**

**Selenium and Fluorine Poisoning**

Losses due to consumption of plants containing toxic amounts of selenium occur among livestock on the ranges and ranch lands of the Great Plains, particularly in South Dakota, Wyoming, and Nebraska. Other States that undoubtedly have a more or less serious selenium problem are North Dakota, Kansas, Oklahoma, Texas, New Mexico, Arizona, Colorado, Utah, and Montana.

Symptoms of chronic poisoning are described by Moxon (33) and also in the article on Nutritional Diseases of Farm Animals, page 323. An acute attack may lead to so-called blind staggers, as commonly reported from Wyoming. This name for the disease is misleading, since the animals do not always become blind or stagger, but they do have a tendency to stray, stumble over objects in their way, and bump into objects and attempt to push them over rather than walk around them. In the last stages paralysis may set in and the animal dies with symptoms of abdominal pain, grinding of the teeth, and respiratory failure.

Prevention of selenium poisoning involves keeping animals away from areas that produce toxic plants, not always an easy matter. Treatment of alkali disease, or blind staggers, is not entirely successful, since the injury caused by selenium is often permanent. The Wyoming station (4) reports success in treating blind staggers in the early stages by hypodermic injections of strychnine and drenching with warm water. The treatment should be carried out by a competent veterinarian. Changing the ration of the animal to selenium-free grain, a protein-rich concentrate, and good hay will do much to favor recovery if the damage to the internal organs has not been too great. Recent work at the South Dakota experiment station indicates that small amounts of arsenic (12 to 15 parts per million) in the feed or drinking water are effective in treating selenium poisoning in cattle as well as in dogs and chickens.

Fluorine poisoning in cattle is most frequently the result of feeding such products as raw phosphatic limestone as a mineral supplement. A purified product low in fluorine is now available for livestock feeding. Areas of excessive fluorine are mentioned in the article on Nutritional Diseases of Farm Animals, page 338.
Fluorine poisoning as seen in cattle is usually of the chronic type caused by the consumption of a small amount of fluorine over a considerable period. In mineral-feeding investigations with dairy cattle, Reed and Huffman (46), of Michigan, and Phillips, Hart, and Bohstedt (42), of Wisconsin, have observed the harmful effects of feeding raw rock phosphate in amounts ranging from 0.625 percent to 2.5 percent of the grain mixture. In the Michigan studies the health of the animals declined, and their teeth became badly worn. The condition of the teeth was first discovered when the animals were about 2½ years of age. At this time the animals began to lap cold water when drinking; if the water was warmed to body temperature they would drink freely. The jawbones and leg bones became abnormally thick and rough. Other symptoms noted were loss of appetite, progressive emaciation, decreased milk yield, delay of estrum after parturition, and lowered birth weight of calves. These may have been secondary effects due to the poor condition of the animals.

Animals usually recover to some extent if the period of fluorine feeding has not been too long and damage to the body is not too severe. The source of excessive fluorine in the ration should be removed. Cattle with excessively worn and defective teeth have difficulty in eating dried roughage but many improve considerably if turned out to pasture.

VITAMIN-DEFICIENCY DISEASES OF CATTLE

Cattle and other ruminants are unique among animals in their vitamin requirements. According to present knowledge, vitamin A or its precursor, carotene, and vitamin D are the only vitamins that must be furnished in order to prevent characteristic nutritional diseases. Nearly all of the recognized vitamin B factors are known to be synthesized by bacteria in the digestive tract of these animals, but information is still lacking on whether this source is entirely adequate under all conditions. Vitamin C is also synthesized in the body by cattle. Recent studies have indicated that under certain conditions the formation of vitamin C may be inadequate, but under such circumstances a dietary source of vitamin C is apparently of no benefit, and injection of the vitamin appears necessary to maintain successful reproduction. Most cattle feeds are rich in vitamin E, but there is no conclusive evidence that this vitamin is essential. Fortunately most cattle feeds such as green pasture grass, hay, silage of good quality, and cereal grains are rich in the known vitamins. The essential vitamins should be furnished to cattle in ordinary feeds whenever possible. An understanding of the conditions under which vitamin deficiencies are likely to occur and prompt recognition of early symptoms of deficiency are essential to eliminate the large losses caused by these diseases.

Vitamin A Deficiency

Vitamin A deficiency is the most common vitamin-deficiency disease of cattle (18). It has been reported from the Southern States, where cottonseed meal and hulls are fed, from the West and Southwest.
where large numbers of cattle are grazed on ranges that become
deficient in carotene-rich forage because of drought or overstocking,
and from the Midwest and sections where corn or cereal grains, beet
byproducts, cereal hay and straw, or old hay or poorly cured forage
are used to fatten cattle. The practice of wintering cattle in stubble-
fields with access to a straw stack without a supplement of well-
cured hay which has retained a good green color may also lead to a
deficiency of this vitamin, particularly in young, growing cattle or
pregnant animals.

Symptoms of vitamin A deficiency appear in cattle of all ages
after they have been for an extended period on a ration containing
too little carotene or vitamin A. One of the first symptoms of the
deficiency that can be detected in fattening cattle or adult animals
is night blindness, or an inability of the animals to see well in dim
light, as during the late evening. This can be detected by driving
the cattle about in a corral and noting whether they bump into the
fence or each other or stumble over objects placed in their way.
Several stages or degrees of night blindness can be recognized, and
the condition may progress to total blindness unless the deficiency is
corrected.

Night blindness results from inability of the animal to regenerate
sufficient visual purple, a light-sensitive pigment of the retina, which
is a compound of vitamin A and a protein. In young animals night
blindness may progress rapidly to permanent blindness owing to in-
jury to the optic nerve. Simple night blindness can usually be cured,
but injury to the optic nerve is likely to result in permanent damage.
In the advanced stages of vitamin A deficiency the eye is very sensitive
to bright light. Animals are somewhat dazzled, blinking abnormally
or keeping their eyes closed when exposed to bright sunlight. An
excessive secretion of tears, which run out over the face and jaw, is
often seen. In total blindness the pupil is fully dilated, and in some
cases the cornea may ulcerate or become opaque.

Swelling of the legs and forequarters (commonly called anasarca)
is often found in cattle showing symptoms of vitamin A deficiency
(fig. 4). This condition has been observed in a large number of fat
cattle coming from the Corn Belt in the Middle West. Other symptoms
noted in this condition are rapid respiration, stiffness or lameness,
night blindness, total blindness, convulsions, loss of appetite, and loss
of weight. Death occurs in advanced cases. When such animals are
slaughtered, an extensive edema (waterlogged condition) of the fat
and muscle tissues is found, a condition that occurs in dry-lot-fed
cattle that have been on low-carotene rations for 10 to 18 months or
longer. The animals recover within a few weeks if well-cured alfalfa
hay is added to the ration (11).

Cows and bulls may lose their reproductive ability in advanced
vitamin A deficiency, but the interruption of sexual activity or damage
to the sexual organs is not always permanent. The usual course is
for the cows to conceive and later to either abort or give birth to weak
or blind calves, which often fail to survive. Cows that have deficient
calves may remain apparently normal themselves, even though they are
receiving an insufficient amount of carotene for normal reproduction.
Hart and Guilbert (19) report that retention of the placenta is common in vitamin-A-deficient cows.

Calves suffer from vitamin A deficiency within a few weeks after birth unless their ration is adequate in this factor. Calves deficient at birth are often blind and weak, slow to get up, and very wobbly on their legs. They may have convulsions shortly after birth, and these often continue intermittently with increasing severity until death (fig. 5). Such calves frequently suffer from diarrhea (or occasionally constipation) and may develop respiratory disorders such as pneumonia. Blindness usually develops in the later stages. As the deficiency progresses, the coat becomes rough, the appetite is poor, and growth is slow, or there is loss in weight. Examination of calves that die from vitamin A deficiency usually reveals that blindness is due to stenosis of the optic nerve (29), while a cystic pituitary with considerable destruction of glandular tissue and white spotted kidneys (30) may also be found.

**Cause, Prevention, and Treatment**

An inadequate carotene intake over a period long enough to permit almost complete exhaustion of the body stores of preformed vitamin A and carotene is the principal cause of vitamin A deficiency in cattle.

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Since forage, either fresh or cured, is the chief source of carotene for cattle, the kind, quality, and amount of forage consumed are the first factors to investigate if vitamin A deficiency is suspected.

During extended periods of drought, pasture and range herbage becomes scarce and is also lower than usual in carotene, protein, and phosphorus. Consequently cattle grazing under such conditions are subjected to a shortage of carotene and other essential factors. Carotene deficiency is an important economic problem in the West, South, and Southwest, where large numbers of beef cattle are grazed on ranges which may become deficient in carotene-containing forage because of insufficient rainfall. Feeder cattle raised under these conditions may show symptoms of vitamin A deficiency or have such a low body storage of vitamin A and carotene that they develop this deficiency later during the fattening period.

In work of the Bureau of Animal Industry in cooperation with the Texas Agricultural Experiment Station it was found that the time required for the development of night blindness in cattle fed a carotene-deficient ration

![Image of cattle and calf]

**Figure 5.**—If a cow's ration is low in carotene during the gestation period, her calf may be blind and weak at birth. *A*, This cow is in good condition and shows no evidence of vitamin A deficiency other than low blood carotene and vitamin A. Her calf was normal in size and condition but was unable to stand alone and had frequent convulsions. *B* shows the characteristic attitude during a convolution. *C*, Abnormal position of front legs. This calf could not stand alone until 4 days of age and survived only 4 weeks.
varied considerably, depending on the age of the animals and their previous nutritional history. Older animals on carotene-rich feeds accumulate a larger supply of carotene than younger animals, and consequently deficiency symptoms appear first in the young animals on a vitamin-A-deficient diet. During grazing seasons of abundant rainfall all animals have a larger storage of carotene than during periods of drought. In this work, reported by Riggs (47), yearling steer calves showed symptoms of vitamin A depletion within 128 to 266 days, with an average of 178 ± 37 days, while younger calves (3 to 5 months old) developed night blindness within 46 to 61 days during 1939, and steers of the same age developed these symptoms within 65 to 131 days during 1939-40. In work of the Bureau of Animal Industry at Beltsville, Md., symptoms of vitamin A deficiency became evident in a group of yearling Hereford heifers as early as 60 days after they came off good pasture and were given a carotene-deficient ration.

Carotene requirements for fattening beef cattle have been investigated at the Texas Agricultural Experiment Station in cooperation with the Bureau of Animal Industry. Steer calves used in the experiments were first largely depleted of their vitamin A reserves and were then divided into five groups and fed sufficient alfalfa-leaf meal at five levels of carotene intake so as to provide 1,250 to 5,000 micrograms of carotene per 100 pounds of body weight daily. No significant differences were found in rate of gain, carcass weight, or carcass grade after a 140-day fattening period. Steers fed the 1,250- and 1,500-microgram level remained completely night blind, and night blindness persisted in some of the steers fed the 2,500- and 3,000-microgram levels. Steers fed 5,000 micrograms of carotene per 100 pounds of body weight became essentially normal.

The condition of fattening animals is not necessarily an indication of inadequate carotene intake, since animals may be critically low in this factor and yet appear to be in good condition. This deficiency never occurs in cattle fattened on good pasture, but it should be guarded against in lot-fed cattle receiving heavy rations of grain with roughages of relatively low carotene content. Vitamin A deficiency can be avoided or corrected in fattening cattle by feeding at least 2 to 3 pounds of new green, leafy (preferably legume) hay daily to each animal.

Carotene or vitamin A requirements for normal reproduction are much higher than for the prevention of night blindness (see the article Nutritional Diseases of Farm Animals, p. 342) or other common symptoms of vitamin A deficiency. For dairy cattle, Converse and Meigs (3) found that normal calvings resulted when timothy, clover, or alfalfa hay was fed without pasture if the hay was of high quality and rich in carotene. In a limited number of experiments a carotene intake of 80 to 100 milligrams appeared sufficient, and a daily intake of 50 to 60 milligrams resulted in the birth of many weak or dead calves. They point out that in order for clover and timothy hay to be graded U. S. No. 1 the hay must have 45 percent

Nutritional Diseases of Cattle 663

or more of green color. Carotene analyses made during these studies at Beltsville on clover and timothy hay with 50 percent of green color (slightly more than is required for U. S. No. 1) resulted in values of 18 to 20 milligrams of carotene per kilogram (2.2 pounds) of hay. Under the conditions of the experiment, it is estimated that 4½ kilograms (about 10 pounds) of this high-quality hay daily would supply sufficient carotene for normal reproduction.

In some preliminary studies by Davis and Madsen (12a), 60 micrograms of carotene per kilogram of body weight (about 2,727 micrograms per 100 pounds of body weight) was found to be sufficient for beef heifers to produce calves which were apparently normal at birth. Calves born to beef heifers receiving 30 and 45 micrograms of carotene per kilogram of body weight were weak or blind at birth and had other symptoms of vitamin A deficiency.

Work in the Bureau of Dairy Industry at Beltsville, Md., has also shown that the minimum amount of carotene and vitamin A needed for the cure of night blindness in cattle does not furnish enough for normal growth and health in young calves. The vitamin A needs of calves are summarized by Converse and Meigs, as follows:

The experiment shows: (1) That when skim milk is fed even after a month on whole milk, calves should receive some well-cured green hay as a source of vitamin A unless cod-liver oil or some other supplement is furnished; (2) that too much dependence should not be placed on whole milk feeding to supply vitamin A unless the milk comes from cows on pasture; (3) that milk does vary widely enough in its vitamin A content to indicate needed caution in its selection for calf feeding; (4) that whole milk in the calf ration is needed more for its vitamin A content than for its fat or energy; and (5) that 20 to 25 cubic centimeters of animal-feeding cod-liver oil or 15 milligrams of carotene roughly equals the vitamin A requirement of growing calves during the milk-feeding age.

Feeds low in carotene, which should be supplemented with carotene-rich feeds if fed to cattle over an extended period, are cottonseed meal and hulls, linseed and soybean meals, wheat, oats, barley, corn (white or yellow), sorghum grains, beet pulp and molasses, cereal straw, corn stover, low-grade legume or grass hays, range herbage during extended periods of drought, and hays held in storage more than 1 year. Feeds rich in carotene are green pasture (grass, legume, or browse), high-grade green, leafy hay (legume, grass, or cereal) of the current year's crop, well-made silage (corn, grass, or legume), and new high-grade alfalfa meal.

The Vitamin B Complex in Bovine Nutrition

Interest concerning the role of the vitamin B complex in the nutrition of cattle dates back to the early work of Theiler and associates in South Africa (52), reported in 1915. They suggested after some rice-feeding experiments in which small amounts of roughage were also fed that:

It may be that cattle are capable of synthesizing their own vitamins in virtue of the extensive bacterial flora of their intestinal tract—a sort of commensal symbiosis.

This hypothesis was investigated later by workers at the Pennsylvania experiment station (5) and was shown to be true for what was then called vitamin B. The workers were successful in obtaining apparently normal growth, gestation, and parturition in dairy heifers on a ration that was low in vitamin B as determined by rat growth. Successful lactation was never obtained for more than several weeks, however. The reason for failure in lactation has never been satisfactorily explained. The work was carried out before the complex nature or chemical properties of the vitamin B group were known. Recent work has confirmed the original observation of vitamin synthesis in the digestive tract of ruminants, and considerable information is now available on the formation of the individual members of the B complex. (See the article on Nutritional Diseases of Farm Animals, p. 344.)

The question whether synthesis of the vitamin B complex in the digestive tract of cattle is sufficient to meet their needs under all circumstances has not been solved in all details. The report from the Kenya Colony (12) that "sweating sickness" in calves, characterized by clinical symptoms similar to those of blacktongue in dogs and pellagra in human beings, is interesting in this regard. Rapid recovery from this disease follows treatment with bakers' yeast or liver extract. Newman and Savage (36) have noted improved results by including dried brewers' yeast and cereal-yeast feed in a dry calf starter fed to young calves that received a limited amount of milk. Baker (2) describes beneficial results from feeding yeast to calves that were in an advanced state of malnutrition and debility as a result of diarrhea, followed by constipation and loss of appetite due to gastrointestinal parasitism (trichostrongylosis). After the animals had gained in appetite and strength, anthelmintic (worm) treatment was successfully administered, and the animals made remarkable recoveries.

Fortunately most feeds of good quality commonly used in cattle feeding are usually good sources of most of the individual vitamins of the B complex. Circumstances may upset their intake however, and other conditions such as infectious diseases, heavy gastrointestinal parasitism, or other disturbances may have a modifying influence on the synthesis of essential factors in the digestive tract. More research on this subject is necessary.

VITAMIN C (ASCORBIC ACID) AND BREEDING EFFICIENCY

It has been the belief for a long time that farm animals as a group, including poultry, usually synthesize enough vitamin C to meet their requirements, but recent studies indicate that this is not always true. Phillips and coworkers (44), at the Wisconsin station, have shown that calves on a carotene-deficient ration do not maintain normal levels of vitamin C in their blood plasma or urine. Later work (43) indicated that the breeding efficiency of some bulls with low fertility could be markedly improved by subcutaneous injections of ascorbic acid. Directions for ascorbic acid therapy of slow-breeding bulls have recently been published (41). The average
dose recommended is 5 milligrams of ascorbic acid per kilogram (2.2 pounds) of body weight or 1 to 2 grams per 1,000 pounds of body weight injected at 3- to 4-day intervals for 5 or 6 weeks or until the bull shows improvement. Preliminary work also indicates that vitamin C treatment of cows that fail to conceive may also be beneficial in some cases (1).

**VITAMIN D DEFICIENCY**

Vitamin D is essential for normal calcium and phosphorus metabolism in both young and adult cattle. Vitamin D deficiency is most apt to occur during the winter season, when the sun's rays are less potent in ultraviolet light, or during periods of barn feeding when the animals do not have access to either sunshine or sun-cured roughage. Bone deformities are probably the most easily recognized feature of the nutritional disease; however, vitamin D deficiency leads to important changes in the body fluids and other tissues also. Vitamin D deficiency in cattle is very rare except under experimental conditions.

The symptoms of rickets and of osteomalacia have been described in considerable detail in the article on Nutritional Diseases of Farm Animals, page 325.

Vitamin D in any form is suitable for the cure or prevention of rickets in calves. Duncan and Huffman (13, 21) have demonstrated the effectiveness of sunshine and sun-cured roughage in curing and preventing rickets experimentally produced in calves. One to two pounds of sun-cured alfalfa a day prevented rickets in calves up to 195 days of age. However, in work at the Pennsylvania experiment station (7) 1 pound of sun-cured alfalfa did not have sufficient antirachitic potency to produce a well-calcified skeleton in young calves kept away from direct sunshine over a 6-month feeding period. Two pounds of good sun-cured timothy a day was found to prevent rickets up to 1 year of age, while 3 pounds of hay cured rickets in an animal at 9 months of age. Bechtel and associates (8) found that calves would not eat enough corn silage to prevent rickets when an experimental basal ration low in vitamin D was fed, but that in older animals consuming 15 to 20 pounds of silage rachitic symptoms were alleviated in 3 out of 4 cases. Bechdel and coworkers (6, 7) found that 3 to 5 pounds of oat straw or 2½ pounds of sun-cured alfalfa prevented rickets, while up to 2½ pounds of night-harvested, machine-dried alfalfa or alfalfa machine-dried immediately after cutting in the daytime was ineffective. Ultraviolet irradiation of the basal ration or irradiation of the calves themselves cured rickets, while feeding cod-liver-oil concentrate or irradiated yeast at the rate of 300 U. S. P. units of vitamin D per 100 pounds of body weight or more per day prevented rickets in calves from birth to 7 months of age. Long, Huffman, and Duncan (23) found that only 0.3 to 0.4 U. S. P. units of vitamin D per pound of body weight was necessary to prevent rickets in the growing calf when winter and early spring milk was the sole source of vitamin D, if normal plasma magnesium values were maintained.
Daily exposure to the sun during the summer protects calves against rickets, but during cloudy weather in the late fall and winter extra vitamin D may be needed if roughage of low quality is fed and if the calves are kept inside for long periods because of bad weather. The vitamin D requirements of adult cattle can be readily satisfied by regular exposure to sunshine, together with a liberal amount of sun-cured roughage as normally fed in practical rations.

**Vitamin E and Sterility in Cattle**

Benefits from wheat-germ oil (rich in vitamin E) in the treatment of noninfectious barrenness of cattle has been reported particularly under certain feeding conditions found in Denmark and elsewhere (see the article on Nutritional Diseases of Farm Animals, p. 346), but supporting data are rather meager and often conflicting. Under conditions in the United States, wheat-germ-oil treatment for sterility in cows has produced some confusing results. The Oregon experiment station reports (10):

> Extensive experiments have been made to test the value of giving cows injections of the antisterility vitamin E which is being extensively advertised as somewhat of a cure-all for difficult breeding. Although earlier trials have indicated these might have some value, further and more extensive tests, using herds at the state hospitals in Salem and Pendleton, have given results too indefinite for conclusions to be drawn.

Research workers have not been able to establish satisfactorily that vitamin E is a dietary essential of calves, cows, or bulls. The technical difficulties and expense involved in preparing vitamin-E-free experimental diets in the quantities needed have limited the studies with cattle.

Since most cattle feeds are good sources of vitamin E, a natural deficiency of the vitamin in balanced rations would, of course, normally be rare (20, 40).

Determination of the need of including vitamin-E-rich concentrates in cattle rations in order to insure fertility and overcome certain forms of sterility awaits further work.

**MISCELLANEOUS NUTRITIONAL DISEASES AND DISORDERS OF CATTLE**

**Urinary Calculi (Urolithiasis)**

Cattle frequently suffer from the formation of crystalline deposits or stonelike concretions in the urinary tract. The size, number, and location of the calculi mainly determine whether or not symptoms of the condition become evident.

Newsom (37) summarizes some of the causes of urinary calculi and discusses them under the headings of water, vitamin A, mineral unbalance, reaction, hyperparathyroidism (overactivity of the parathyroid gland), infection, and urinary irritation. It is evident from the list of factors that may influence calculi formation that the problem is complex. Evidence that dietary factors are concerned is defi-
nite, but it should be kept in mind that other factors may be involved, and also that there are many different types and kinds of calculi.

Newsom points out that calculi are more common in winter, when water is limited or lacking because of freezing weather. The case for vitamin A deficiency as a cause of calculi formation in cattle is not complete, although some rations that tend to favor calculi formation may be low in this factor. An unbalanced mineral intake, particularly of calcium, phosphorus, and magnesium, is believed to influence calculi formation in cattle to a considerable degree.

In cooperative experiments of the Bureau of Animal Industry and the Texas Agricultural Experiment Station conducted at Big Spring, Tex., a serious calculi problem has developed in steers fattened on milo grain, cottonseed meal, and sorgo silage. Such rations are naturally high in magnesium. Steers given high levels of calcium, furnished either as pulverized oystershell or bonemeal had fewer calculi than those receiving less calcium and more magnesium. The work is being continued, with special emphasis on the ratio and quantity of calcium, phosphorus, and magnesium in the diet.

Symptoms and treatment are discussed in the article on Nutritional Diseases of Farm Animals, page 347.

**Bloat**

Bloat is a troublesome disorder of cattle that is often the cause of heavy losses. The disease is usually of a sporadic nature; when due to overeating or consuming spoiled feeds, it may be considered as nutritional; but other conditions, such as occlusion of the openings of the forestomachs with hair balls or other foreign bodies, and factors that interfere with normal rumination and intestinal movements, may also be involved. Bloating may occur suddenly or may appear in some individuals more or less regularly after each feeding.

Bloat is due to an excessive accumulation in the rumen of gases probably formed to a large extent by bacterial action. The conditions that favor the kind and amount of gas produced in the rumen and the factors that inhibit its normal removal are not completely understood. Recent studies have indicated that the pressure of the gas in the rumen, which results in a distention of the organ, causing pressure on other vital organs such as the heart and lungs, is not the sole cause of death in bloated animals. Adsorption of toxic gases from the rumen also produces distressing symptoms and may cause death (88).

A number of precautions for controlling or preventing bloat have been advocated, but practically none have proved to be of much value under experimental tests. It is generally agreed, however, that legume pastures (alfalfa, clovers, etc.) are more likely to cause bloating than is grass. A young, lush growth of legumes appears to be more dangerous than the mature forage. Pasture that is wet with dew is often said to be particularly dangerous. Before turning hungry animals on legume pasture, it may be worth while to give them a good feed of dry hay so they will not gorge themselves on green forage.
It is possible that soil conditions under which a feed is grown may also influence its tendency to produce bloat. McIntosh (24) states that—

The farmers who have the least trouble with bloating are those whose alfalfa fields are in a high state of fertility, while those who have the greatest amount of trouble are farmers whose pasture land is in a worn-out condition.

His theory has not been critically tested, but it is no doubt worthy of careful consideration. The determination of the chemical characteristics of feeds that cause bloat and a more accurate definition of the environmental and physiological factors concerned in this disease are important problems for further study.

Fattening cattle or heavy-milking cows that receive large quantities of grain also have a tendency to bloat. In cattle-fattening experiments, Osland and coworkers (39) at the Colorado experiment station report that—

* * * barley, when fed alone in a grain, cottonseed cake, and hay ration, is not a safe feed for calves because of its tendency to cause bloat, even though it is fed at the rate of only 1 percent of their live weight or less. The addition of cheap, bulky carbonaceous feeds such as wet beet pulp, silage, or potatoes has proved effective in checking digestive troubles but has failed to prevent bloats entirely.

Similar results were obtained by the Bureau of Animal Industry in cooperation with the Montana experiment station. A large proportion of steer calves fattened on barley and alfalfa were found to suffer from bloat or other digestive disturbances that caused scouring or going off feed. Feeding a grain ration consisting of an equal amount of barley and dried molasses-beet pulp, however, resulted in no serious cases of bloat or other digestive disorders. In these experiments bloat was usually treated by drenching the animals with as much as 12 ounces of raw linseed oil.

In severe cases of bloat, puncturing the paunch with a trocar may be necessary. This operation may injure the animal if not done properly, and it should be performed by a qualified veterinarian whenever possible. Further treatment to stop fermentation in the paunch and restore normal gastrointestinal activity may also be prescribed.

**Ketosis**

The metabolic disorder of cattle in which there is an abnormal quantity of ketone bodies (acetone, acetoacetic acid, and beta-hydroxybutyric acid) in the blood, urine, and milk is an important problem in the dairy industry. The disease causes a loss of condition in affected animals, a marked decline in milk flow, and the production of a characteristic off-flavored milk. Duncan, Huffman, and Tobin (15) studied this condition in a herd of Jersey cows under farm conditions, many of which were seriously affected with the disease. The disorder was apparently associated with feeding a ration of poor-quality soybean hay, corn silage, and a small amount of grain during the fall and winter. The disease was described as follows:
The occurrence of the disturbance in the health and milk production of the cows became most pronounced within two to six weeks following a normal parturition. It was the owner's opinion that the cows were in good nutritional condition before calving and that all had begun to produce large quantities of milk. He also observed that the cows lost appetite gradually, rapidly decreased in milk production, became constipated, hide-bound, listless, and emaciated in appearance. He reported that the barn had a peculiar odor at times.

Cows in the most critical condition were given chloral hydrate (1 dram in 12 ounces of warm water 3 times a day for 1 day), and this was repeated later in severe cases. The ration of all the animals was changed. Liberal quantities of alfalfa hay of medium quality and cornstalks were fed instead of the soybean hay and silage previously used. Severely affected cows were given 1 to 2 pounds of corn sugar or molasses and the grain was increased to 5 pounds a day. The new feeding schedule resulted in marked improvement within 2 weeks in most cases, as shown by improvement in appetite and general condition and by a decrease of the ketone bodies in the blood, urine, and milk as determined by chemical analyses.

Duncan and coworkers and others have reached the general conclusion that ketosis may be reduced in stall-fed animals by feeding hay of a good quality together with a liberal supply of grain. In addition, high-producing individuals should have some feed that is a source of quickly available sugar, such as molasses. Turning animals to pasture also resulted in recovery. An extract from the anterior lobe of the pituitary, and even vitamin B₁, have been reported to be of use in the treatment of this disease.

**Overeating and Undernutrition**

Cattle, like other farm animals, are adversely affected by overeating. (See the article on Nutritional Diseases of Farm Animals, p. 348.) When this occurs, the regular ration, except water, should be withheld for a day or two, and they should be slowly brought back on feed, a small amount of palatable roughage being given first, with gradual introduction of the grain mixture as the appetite returns. If there is severe constipation after an attack of diarrhea, it should be treated as soon as possible with Epsom salts, raw linseed oil or pure mineral oil. The advice and help of a qualified veterinarian may prevent serious consequences if it is obtained early enough.

Lactating dairy cows are occasionally overfed when record-breaking production is desired. Symptoms of overeating are loss of appetite, dullness, diarrhea, distention of the rumen (bloat), and a marked drop in the milk yield. Milk fever (see p. 533) occurs most often in heavily fed, high-producing animals. In an effort to force apparently normal animals to maximum performance or above, drugs are occasionally prescribed to stimulate appetite, a practice that should be discouraged. The record may be attained, but the productive life and reproductive ability of the animal may thereby be permanently injured. Losses due to overeating can best be controlled or prevented by careful feeding, with strict attention to the appetites of the animals.
Underfeeding may be harmful to the animal body and is frequently uneconomical (27). Undernutrition may come from either quantitative or qualitative deficiencies in the ration or from both. (See Nutritional Diseases of Farm Animals, p. 349.)

Losses from livestock consuming poisonous plants on the range are usually largest during periods of drought or when the range is overgrazed. Shortage of water is as serious or more so than a lack of feed. Forsling (16) discusses the dangers of overstocking.

Hart (17) points out that “profitable livestock management does not include allowing animals to border on starvation or failure to supply sufficient quantities of essential substances until symptoms appear.”

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