FOOD AND AGRICULTURE ORGANIZATION OF THE UNITED NATIONS

Nutritional Deficiencies in Livestock

ROME, ITALY

FAO AGRICULTURAL STUDIES

1. *Breeding Livestock Adapted to Unfavorable Environments* — A study of the adaptibility of various breeds of sheep, cattle, horses, pigs, buffaloes, camels, etc., to different climates and altitudes. Well illustrated. In English and Spanish. $1.50 7/6.
2. *Preservation of Grains in Storage :* Papers presented at the International Meeting on Infestation of Foodstuffs, London, 5-12 August 1947. The problems of grain storage discussed by contributors from Canada, USA, Britain, France, Egypt and Australia. In English, French and Spanish. $1.50 7/6.
3. *Using Salty Land* — 49 pages, tables charts and references. In English. $0.50 2/6.
4. *Soil Conservation :* — An international study 96 illustrations, map, charts, references. In English, French and Spanish. $2.00 10/-.
6. *Storing and Drying Grain in Canada, in the United States, in the United Kingdom* — An illustrated discussion of the usual methods. In English, French and Spanish. $0.50 2/6.
7. *Better Utilization of Milk* — A study of current uses of milk and its by-products, and of the possibilities of using them more fully. Written for the technician or government experts. In English, French and Spanish. $0.75 3/9.
8. *Rinderpest Vaccines, Their Production and Use in the Field* — The latest information on the production of recently developed Rinderpest vaccines and their uses (1949). In English and French. $1.00 5/-.
9. *Efficient Use of Fertilizers* — A guide book on fertilizers and their use in crop production. In English, French and Spanish. $2.00 10/- (English edition published by Leonard Hill Ltd., Stratford House, 9 Eden Street, London, N. W. 1.)
10. *Some Important Animal Diseases in Europe :* Papers presented at the Animal Disease Meeting, Warsaw, 1948.
    Papers written by contributors from England, Czechoslovakia, Poland, and the World Health Organization. In English with French summaries. $2.00 10/-.
11. *Consolidation of Fragmented Agricultural Holdings* — Individual studies on the problem of fragmented agricultural holdings in Denmark, France, Ireland and Switzerland. In English, French and Spanish. $1.00 5/-.
12. *Some Aspects of Food Refrigeration and Freezing.* — A well-illustrated compilation of papers written by experts of many nationalities. In English. $2.00 10/-.
13. *Weed Control by Growth-Regulating Substances* — A short practical guide to the use of hormone weed killers. In English, French and Spanish. $0.50 2/6.
14. *Joint WHO/FAO Expert Panel on Brucellosis* — In English and French. $0.25 1/3.
15. *Joint WHO/FAO Expert Group on Zoönoses* — In English and French. $0.30 1/6.
16. *Improving the World's Grasslands* — An international study giving latest information on grassland improvement. Available in English only from Leonard Hill Limited, Stratford House, 9 Eden Street, London, N. W. 1., 10/6.
17. *Communal Land Tenure* — An FAO land tenure study. In English. $0.50 2/6.

Nutritional Deficiencies in Livestock

An FAO Study Prepared by RICHARD T. ALLMAN,
Animal Nutritionist, Agriculture Division,
and
T. S. HAMILTON, Professor of Animal Nutrition,
University of Illinois

Rome, Italy

1st printing, May 1948
2nd printing, August 1949
3rd printing, December 1952

Most of the photographs used in this book were furnished through the good offices of the National Research Council, Washington, U. S. A. Individual prints were obtained from C. E. Aubel, Kansas State College of Agriculture and Applied Science; Charles I. Bray, Louisiana State University; Fred Hale, Agricultural and Mechanical College of Texas; E. H. Hughes, University of California; T. B. Keith, University of Idaho; and H. Welch, Montana State College.

CONTENTS

TABLES

1. The Importance of Feeding Balanced Rations to Livestock

If the people of the world are to be adequately nourished, the total amount of available food must increase in proportion as the population increases. Much more food can be produced on land now used for agricultural purposes—through increasing soil fertility, better management practices, new crops, better plant varieties, etc.—and new areas can be converted to food production.

Correcting dietary deficiencies in livestock rations will do much to increase the world's supply of meat, milk, and eggs.

An immediate increase in the available food for all people throughout the world should be looked upon as essential to their welfare. This booklet is concerned with ways and means of increasing the world's food supply, especially the supply of meat, milk, and eggs.

While it is possible for man to live healthfully on plant foods alone, it is very doubtful that the world's needs can be filled without the maintenance of a balance between foods of plant origin and foods of animal origin. There are three reasons for this: (1) Few of the many dietary essentials —proteins, minerals, and vitamins—are sufficiently concentrated in plant products to make it possible to adequately balance the diet without excess of energy or some other dietary essential; (2) a balanced diet—one containing all dietary essentials needed for healthful maintenance and efficient productiveness without excesses of any nutrient—is more efficiently utilized than any poorly balanced diet; (3) the production of meat, milk, and eggs should be increased because many areas that can be utilized more fully for food production are only suitable for the production of crops—pastures and roughages—and unsuitable for direct consumption by man. Only animals can transform these feeds into human foods.

The chief factors, in order of their probable importance, which are at present limiting the world's production of meat, milk, and eggs, are

1. Insufficient feed;

2. Insufficient concentrations in the ration of one or more dietary essentials;

3. Severe dietary deficiencies.

A wider production of improved pastures would probably do more than any other one thing to alleviate and correct this inadequate feed situation.

The all-too-common practice of feeding rations with insufficient concentrations of one or more dietary essentials is probably the chief factor limiting the production of meat, milk, and eggs in the world. If these rations were devoid of one or more dietary essentials, the animals would probably show some marked evidence that all was not well. Unfortunately, these rations are only partially deficient in one or more dietary essentials. The results of feeding these rations produce only subclinical manifestations of dietary deficiencies such as slow growth, shy breeding, low milk production, susceptibility to certain infectious diseases, inefficient use of feed, etc. Since these symptoms are nonspecific, not characteristic of any individual dietary deficiency, they are not ordinarily traced to inadequate feeding. While they do not point to the specific dietary deficiency, they are very often truly indicative of a poorly balanced inefficient ration. Tremendous quantities of the world's feeds are wasted in this type of feeding, resulting in large losses of human foods.

Under practical conditions, the feeding of truly deficient rations—rations almost devoid of a single dietary essential—is much less common. The feeding of this kind of ration results, usually, rather quickly, in the appearance of either marked generalized indications that the ration is poor or specific and characteristic symptoms of a dietary deficiency. These are usually so obviously unappetizing and poorly balanced that they are quickly changed, if at all possible.

Thus, the feeding of unbalanced rations may produce one or both of two types of symptoms in the animals fed. One type consists of nonspecific symptoms, such as poor growth, rough hair coats, decreased milk yield, etc. The second consists of more or less readily detectable clinical or visible symptoms characteristic or indicative of the particular dietary deficiency. The former almost invariably results from insufficient food, usually when the deficiency is protein or an amino acid. Quite often, too, nonspecific symptoms only result from mineral and vitamin deficiencies. Clear-cut symptoms of single dietary deficiencies are rarely seen under practical conditions. Specific clinical or apparent symptoms of single dietary deficiencies have been recorded under laboratory conditions and

these are most valuable. All livestock men should be familiar with them because they emphasize how important it it to feed well-balanced rations, how drastic may be the results when extremely small amounts of certain mineral elements or vitamins are omitted, and how these deficiencies may be corrected.

The feeding of well-balanced rations to livestock

1. Conserves grains for human use;
2. Stretches available feeds over a larger livestock population;
3. Increases the amounts of meat, milk, and eggs available for human use.

2. Soils and Nutrition

The truth of the statement that "nutrition begins with the soil" has been surmised by observant men for centuries. They noted that livestock would do better in some areas than in others where no apparent differences existed. Through experience they learned not only what localities were best suited to their needs, but that some were definitely undesirable.

The nutritional problems of animals in relation to soil conditions, however, did not become acute nor were they the subject of scientific inquiry until nearly the middle of the nineteenth century; their importance was emphasized as the increase in world population pushed man out into new lands. Many of the new lands were found to be incapable of supporting animal life, particularly when the animals were not permitted to graze large areas and select their food. The need for new lands has continued to create problems of this nature. There are still vast areas in the world that will be suitable for many agricultural purposes when it is learned how to correct their natural shortcomings.

Soil Deficiencies

A deficiency of phosphorus in soils and forage was among the first to be recognized and described. More recently an appreciation of the role of the trace elements, such as cobalt, fluorine, and copper, has developed.

It is evident that a deficiency of phosphorus, next to iodine, is the most widespread of any mineral nutritional trouble in grazing animals. The fact that a nutritional deficiency does exist in an area is sometimes evident from the small size of the animals, failures of reproduction, malformed bones that are easily broken, and a persistent craving by many cattle for bones, dirt, wood, and other materials.

Multiple deficiencies may often be encountered in animals—as calcium and phosphorus, cobalt and phosphorus, cobalt and copper, copper and iron—simultaneously with the same deficiencies in forage crops. Recognition of the possibility of multiple deficiencies may help to explain discrepancies in symptoms found in other areas.

When it is concluded that the crops of a certain area are deficient in a given mineral, corrective measures are required. What these will be depends on the element, the area, and many other factors. Supplemental feeding with the mineral, the use of supplemental feeds from another area, rotation of crops, variety and kind of crop, time of harvesting, irrigation and fertilizers (including liming materials), must all be considered in determining the most economical and practical remedy. A full discussion is beyond the scope of this paper.

The ultimate effect on human food is the basic consideration, for, after all, human food is the primary purpose of agriculture. Many crops are consumed directly by man, so he suffers directly from deficiencies of beneficial minerals and excesses of toxic minerals in the soil. He suffers indirectly from decreased production of crops, livestock, and livestock products.

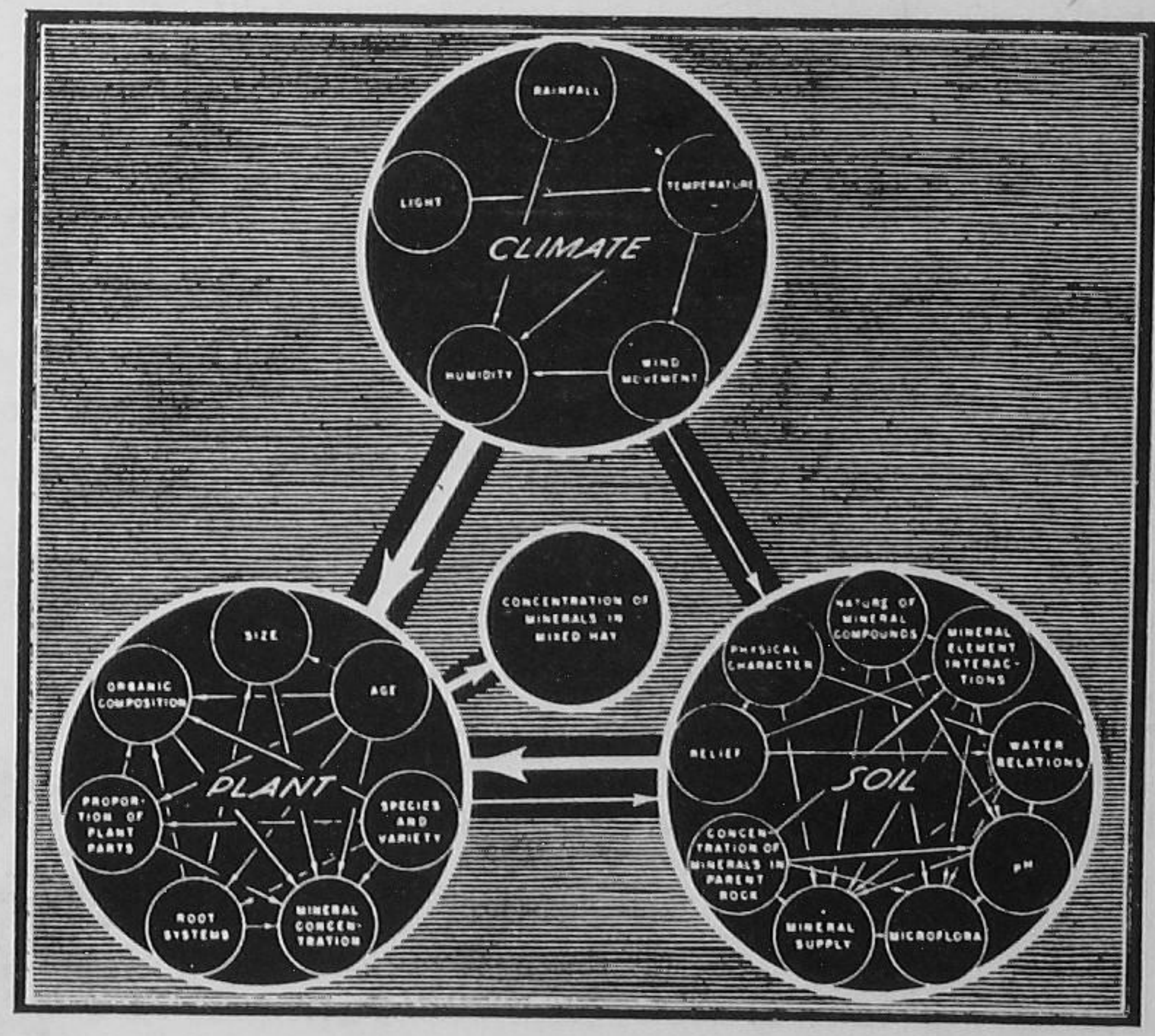


Figure 1. The various factors affecting the mineral content of mixed hay.

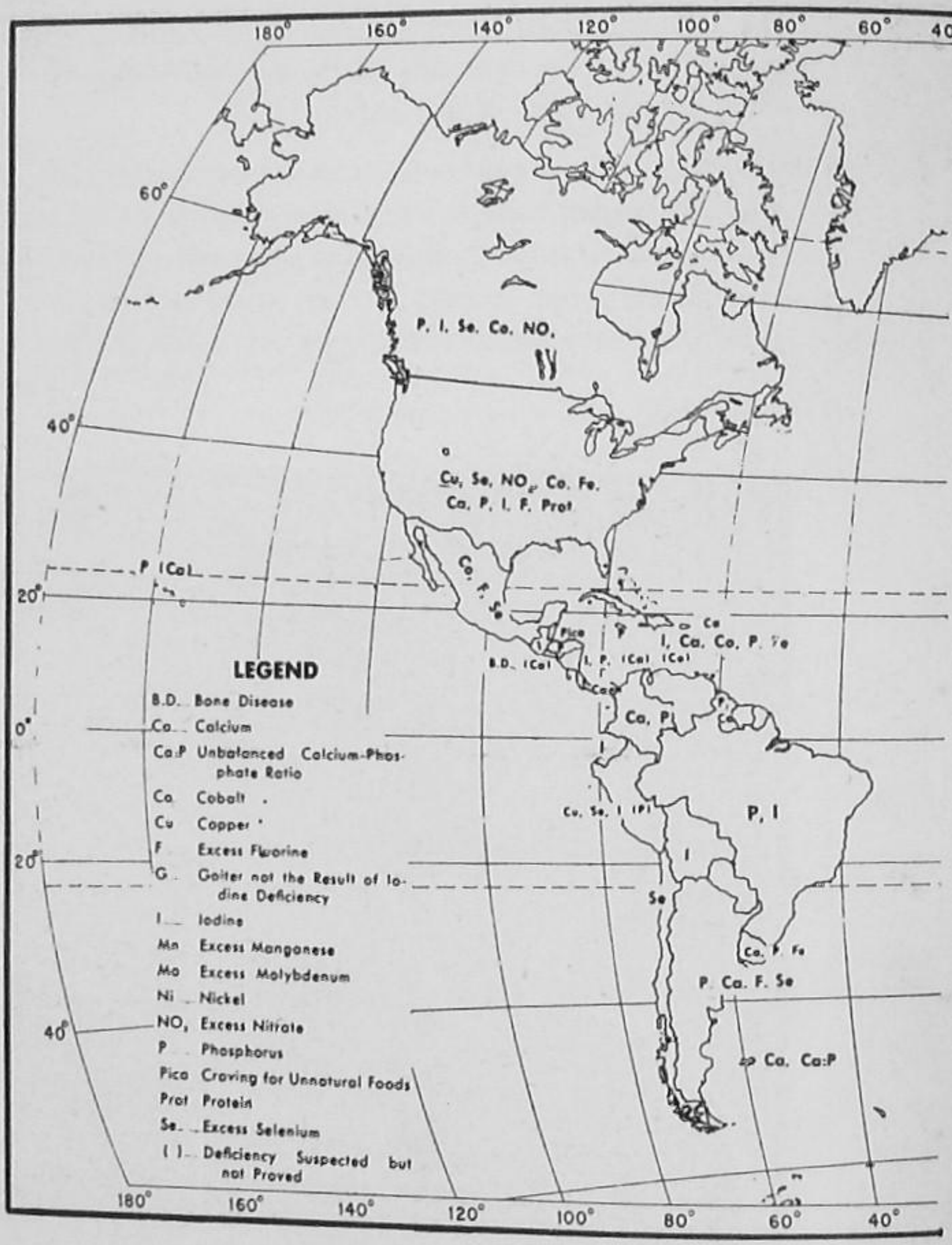
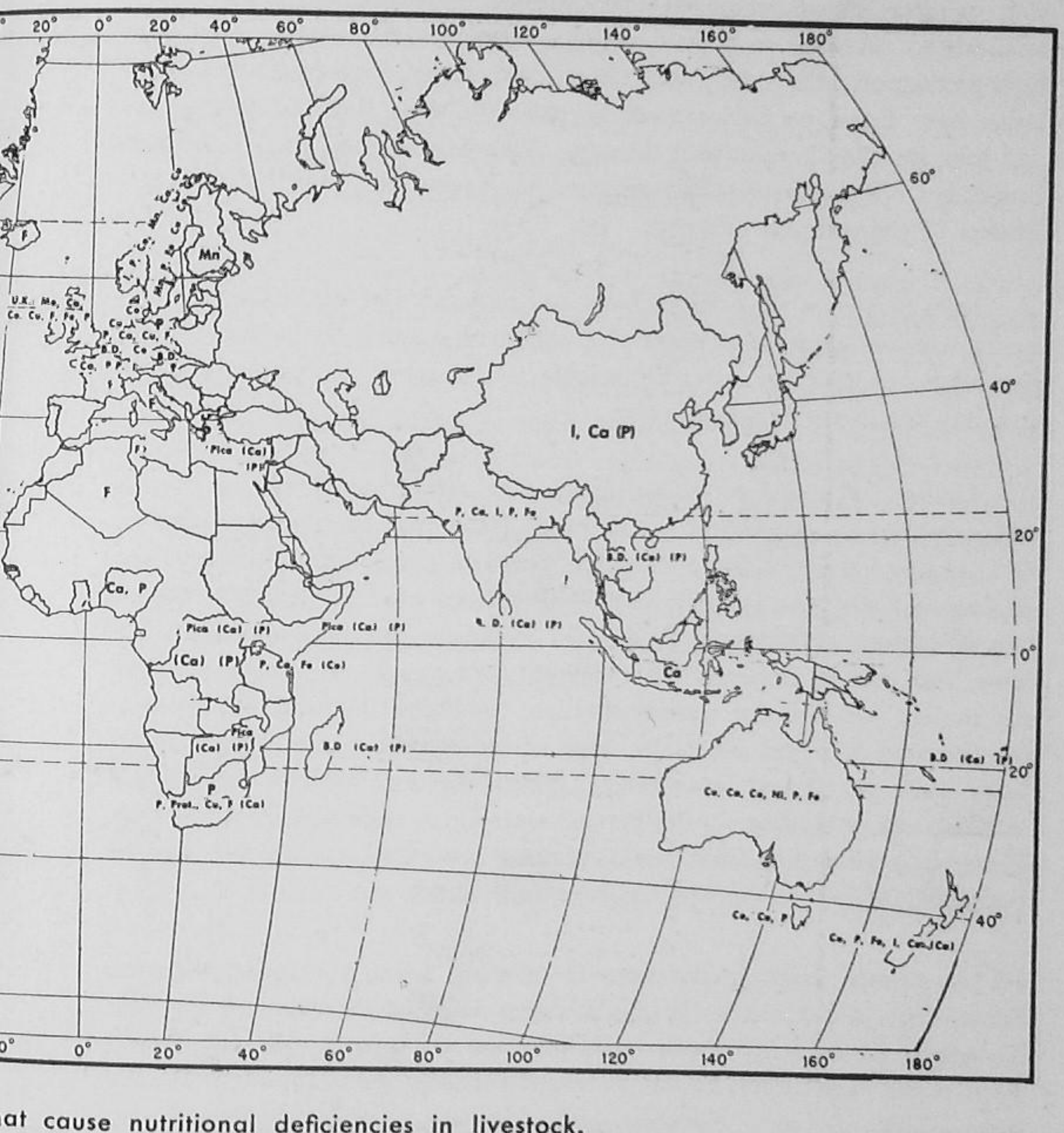


Figure 2. World map showing soil deficiencies



at cause nutritional deficiencies in livestock.

Climate and Soil

The effect of climate, especially on the formation of soils, is one of the basic factors determining the chemical composition of crops. The soils in areas having prolonged heavy rainfall are often low in the soluble minerals so necessary to a good skeleton in cattle and horses. The large, high-production breeds of cattle, sheep, and horses, introduced into these areas, have failed on native feeds to maintain their size and production, and have developed nutritional diseases. This demonstrates that indigenous breeds are often phenologically adjusted to survive and grow on marginal intakes of the essential elements.

Soils that have their origin from soil minerals lacking in calcium naturally will not grow crops containing sufficient calcium for normal animal development. Calcium is readily soluble and is subject to excessive leaching and loss in the drainage water.

Under the abundant rainfall and high temperatures of the tropics and semitropics, weathering forces work faster and carry their influence to a greater extreme. Here there has been no glaciation to interrupt the process and the country rock and regolith have been subjected to drastic decomposition. Hydrolysis, as well as oxidation, has been extremely intense and silicate minerals have quickly succumbed. In many parts of the tropics, wet and dry seasons alternate, which no doubt greatly intensifies chemical activities, especially those of the organic matter. The soluble bases such as calcium, magnesium, potassium, and sodium, are quickly released and, according to the season, are subject to removal by leaching. There is a gradation from these extreme conditions in laterite soils to podsolic soils, where the loss has been more moderate.

In Europe, laterites and lateritic soils are found in southern France, Spain, Italy, and Greece. In the Western Hemisphere, they are typically developed in northern South America, Central America, Mexico, Puerto Rico, Cuba, and other islands in the Caribbean Sea. In India, Burma, Siam, Java, Borneo, Sumatra, and contiguous regions, laterites are common, while on the islands of the Central Pacific red earths are abundant. Hawaii also has lateritic soils. Laterite soils show red or yellow color, especially in the B horizon. However, the surface soil, if uneroded, often is brown or gray. In other cases, red and yellow color dominates the surface soil.

Another disadvantage of the high content of sesquioxides in laterite soils is the unavailability of phosphorus. This element combines with iron and aluminum in very complex compounds, rendering the phosphorus insoluble and unavailable to plants. Thus, much of the calcium and phosphorus deficiency symptoms reported in these countries have their origin in the climate and its effect upon the soil.

Podsolization, especially combined with sheet erosion, can and does develop soils low in calcium, with much of the phosphorus unavailable to the crops, depending on the stage of development. Podsolic weathering is important in the United States, Canada, and in northern and central Europe.

Climatic conditions, especially temperature and rainfall, exert an important influence on the amount of nitrogen found in soils. As progress is made from a warmer to a cooler climate, even though the rainfall may not be exactly the same as to amount and distribution, the nitrogen of soils tends to increase. Rainfall seems to exert a control upon the accumulation of nitrogen in soils as definite, in many respects, as that of temperature. In general, under comparable conditions, the nitrogen content of soils increases as the effective moisture becomes greater. Soils in arid regions are usually low in nitrogen; an exception to this is the black-earth soil (chernozem) of semiarid regions. It may be said, however, that soils low in organic matter are low in nitrogen; the nitrogen, especially in humid regions, is carried almost wholly by the organic matter.

Elements in Soils

The texture of the soil, other factors being constant, seems to influence the nitrogen content. A sandy soil, for example, usually carries less nitrogen than one of a heavier nature, while a virgin soil and its cultivated equivalent differ radically.

The nitrogen content of soils influences the protein content of the crop. The vegetative portion is more affected than the grains. As animal feed is largely derived from the vegetative parts, this effect of the soil is thus more important in animal feed than in human food.

The problem of phosphorus deficiency is not a simple one that can be determined by soil or crop analysis. It is the exception rather than the rule to have aphosphorosis by itself. It is usually complicated by other deficiencies that may be mineral, protein, or the total digestible nutrients available to the animal. The effect of lignification on the availability of the protein and minerals of crops, the interrelationships of rates of growth

and calcium and phosphorus requirements, suitability of the breed of livestock to the climatic conditions, and natural feeds, all must be taken into account. It is not unusual for imported livestock in an area to show deficiency diseases, while the indigenous breeds that are slow-growing, late-maturing, producing small quantities of milk, do not show the deficiencies, at least to the same degree. Imported livestock eventually reverts to the type that is in balance with the environment, unless these deficiencies are corrected.

In certain aridic alkaline soils, the calcium may be rendered unavailable to plants because of the concentration of other basic ions. An increase in pH of the soil solution over 7 also adversely influences the solubility and availability of phosphorus. Soils that have been cultivated contain less calcium than a similar virgin soil, as a result of crop removal and erosion.

There is a high incidence of goiter in certain regions of the world. Soils of such regions are generally quite acid, and the highly leached clay they contain does not possess the power of anion retention, so the small quantities of iodine that were present in the geological strata are leached out by the percolating water. These soils may contain only a few parts per billion of iodine—not enough to grow plants with sufficient iodine to prevent goiter. Other soils are naturally deficient because of their origin. Soils developed from limestone in humid regions and those near the seacoast usually contain sufficient iodine to prevent goiter.

Selenium is present in detectable quantities in all soils, but it reaches toxic concentrations only in soils derived from Cretaceous shales in semi-arid climates.

Fluorine has been found in soils in quantities ranging from a trace up to 0.15 percent. Mica and tourmaline, both of which may contain up to 1 percent, are present in practically all soils. Phosphate rocks may contain from 3 to 4 percent of fluorine; about three-quarters of this amount is retained in the fertilizers made from these sources. Considerable fluorine is therefore added to soils fertilized with commercial fertilizers.

Cobalt occurs in most soils in quantities ranging up to 15 parts per million. In New Zealand, it has been found that sheep suffer from a lack of cobalt in the natural vegetation in soils having less than 2 to 3 parts per million of the element. When cobalt in the soil exceeds 5 to 10 parts per million, the plants take up enough to produce normal growth in the sheep. In general, deficiencies are less likely to occur on geologically older soils, or areas of concentration, than on soils of recent origin.

Most mineral soils contain sufficient copper and iron except some that are very sandy and leachable. Calcareous and alkaline soils may grow crops that are low in iron and copper because the availablity is decreased with increased alkalinity, especially if an excess of phosphates is present. The fixation of iron by phosphate is more likely to occur in sandy soils than in a clay soil. Organic soils such as peat and muck are more likely to have a lower copper content than normal mineral soils, especially if they are alkaline and contain appreciable amounts of ferrous iron.

The fact that mineral requirements of different breeds differ, depending on rate of growth, age of maturity, production and reproduction, has been pointed out, but species difference is also important. Cattle show calcium and phosphorus deficiencies on land that supports sheep, because of their feeding habits and size of skeleton. Sheep, on the other hand, exhibit cobalt deficiency symptoms in four months where horses thrive for generations.

The only conclusion that can be drawn at this time is that the problem is complex, vital and worldwide—and our knowledge is meager.

Selected References

BEESON, K. C. 1941. *The mineral composition of crops with particular reference to the soils in which they are grown. A review and compilation.* U. S. Dept. Agr. Misc. Pub. No. 369. Pp. 164.

——— 1945. "The occurrence of mineral nutritional diseases of plants and animals in the U. S." *Soil Science, 60:* 1, 9-13.

HARTMAN, A. M. 1939. "Deficient and excess minerals in the forage of U. S." U. S. D. A. *Yearbook,* pp. 1027-1044.

HUFFMAN, C. F., and C. W. DUNCAN. 1944. Nutritional deficiencies of farm mammals on natural feeds. Ann. Rev. Biochem. *13:*467-86.

MCMURTREY, J. E., JR., and W. O. ROBINSON. 1938. "Neglected soil constituents that affect plant and animal development." U. S. D. A. *Yearbook,* pp. 807-829.

MADSEN, L. L. 1942. Nutritional diseases of farm animals. U. S. D. A. *Yearbook of Agriculture,* pp. 323-53, 1942A, and pp. 645-72, 1942B.

MARSTON, H. R. 1939. Ruminant nutrition. Ann. Rev. Biochem. *8:*557-78.

MAYNARD, L. A. 1943. Relation of soil and plant deficiencies of toxic constituents in soils to animal nutrition. Ann. Rev. Biochem, *10:*449-70.

MITCHELL, H. H., and F. J. MCCLURE. 1937. Mineral nutrition of farm animals. Nat. Res. Council Bull. 99.

ORR, JOHN BOYD. 1929. *Minerals in Pastures.* London: H. K. Lewis & Co., Ltd.

RUSSELL, F. C. 1944. Minerals in pasture: Deficiencies and excesses in relation to animal health. Imp. Bur. of An. Nutrition Tech. Comm. No. 15.

TABLE 1—SUMMARY OF INVESTIGATIONS OF RELATIONSHIP OF SOIL AND NUTRITIONAL DEFICIENCIES

Disease	Species Affected	Country	Principal Areas Investigated	Blood and Tissues	Response to Feeding Tests	Pasture Characteristics	Effect of Specific Fertilizers on Incidence of Disease	Experimental Production of Disease	Conclusions
Bush-Sickness	Sheep and Cattle	New Zealand	Volcanic soils of N. Island central plateau. Granite soils of S. Island.	Low Co reserve in organs which normally store Co	Co feeding preventative and curative	Low Co content related to incidence of disease: <0.07 p.p.m. D.M.[1] of Co unhealthy for sheep; <0.04 for both sheep and cattle	Co fertilizing effectively controls the disease	(2)	Uncomplicated Co deficiency state due to lack of Co in the pasture
Morton Mains Disease	Sheep, especially lambs	New Zealand	Southland	Ditto	Ditto	Pasture upon which lambs developed the disease, 0.04 to 0.07 p.p.m. D.M. of Co	Ditto		Ditto
Enzootic-Marasmus	Sheep and Cattle	Australia	W. and S. Australia	Ditto	Ditto	Low Co content in affected areas, 0.02 to 0.06, mean 0.04 p.p.m. D.M.			Ditto
Nakuruitis	Cattle	Africa	Nakuru, Kenya		Co feeding beneficial				Probably Co deficiency
Pining or Pine	Sheep	Britain	Ross-shire Scottish Borders		Controlled by Co feeding Ditto	Pasture, 0.08 p.p.m. of Co. Co content 0.05 to 0.20 p.p.m. D.M. No correlation with disease but seasonal variations may obscure this.	Ditto		Probably Co deficiency
			Wales, 1 center Dartmoor		Ditto Co+Mn+Zn beneficial	Affected areas, 0.20 p.p.m. of Co. Recovery areas 0.45 p.p.m.			
Grand Traverse Disease	Cattle	U.S.A.	Michigan		Co feeding curative	"Affected" hay, 0.03 to 0.06 p.p.m. of Co. Healthy hay, 0.12 p.p.m. of Co.			Co deficiency
Coast Disease	Sheep, Cattle, Goats	Australia	Coastal areas of S. and W. Australia and Tasmania	Low Cu reserve in organs which normally store Cu	Co+Cu feeding controls the disease. Neither alone is effective.	Sample of affected pasture (Tasmania), 0.10 p.p.m. D.M. of Co (sample slightly contaminated)	Topdressing with Cu+Co effective in controlling the disease in Tasmania		Probably dual deficiency of Cu+Co

SOURCE: F. C. Russell. Imp. Bur. of An. Nutrition Tech. Comm. No. 15.
See footnotes at end of table.

TABLE 1—SUMMARY OF INVESTIGATIONS OF RELATIONSHIP OF SOIL AND NUTRITIONAL DEFICIENCIES (*Continued*)

Disease	Species Affected	Country	Principal Areas Investigated	Blood and Tissues	Response to Feeding Tests	Pasture Characteristics	Effect of Specific Fertilizers on Incidence of Disease	Experimental Production of Disease	Conclusions
Salt Sick	Cattle, Sheep, Goats	U. S. A.	Florida, sandy soils		In some areas Fe and Cu is effective in controlling the disease: in others Co is also required. The Fe supplement probably contained traces of Co in some areas.	Failure to demonstrate presence of Co spectrographically in either sick or healthy wire grass suggests method inadequate. No significant difference in Cu content. Range 4.7 to 10 p.p.m. D.M.		Feeding experiments with calves indicate that "salt sick" hay may produce Co deficiency uncomplicated by Cu deficiency	Probably Co deficiency usually associated with Cu deficiency. Deficiencies not demonstrated in pasture.
Enzootic Ataxia, associated with Anemia and Stringiness of wool	Lambs Gestating Ewes Adult Sheep	Australia	Coastal and semi-coastal areas of W. Aust., also on calcareous soils of S. Australia	Low Cu reserve in organs which normally store Cu. Low Cu content of ewes' milk.	Feeding Cu to gestating ewes prevents anemia and stringy wool and occurrence of ataxia in the progeny	Values of <3 p.p.m. D.M. of Cu indicate unsound pasture. Between 3 and 5 are marginal and >5 is sound. Some anomalous results.	Topdressing with Cu effectively controls ataxia		Uncomplicated Cu deficiency state in ewes and lambs due to lack of Cu in the pasture eaten by the ewe
Swayback	Lambs	Britain	Derbyshire	Low Cu reserves of ataxic and normal lambs in affected areas	Feeding Cu to gestating ewes or to lambs controls swayback in lambs	Pastures well supplied with Cu, 12 to 27 p.p.m. D.M. High Pb, Ca, Fe and Zn			Cu deficiency state. Possibly the availability of the pasture Cu is affected.
Sudden Death or Falling Disease	Cattle	Australia	South and West Coastal Areas	Low Cu reserve in Liver	Controlled by Cu feeding	Cu usually <2 p.p.m. D.M.			Cu deficiency state associated with lack of Cu in pasture
Scouring Disease	Cattle, Goats	Holland	Sandy soil and reclaimed *polders*	Low Cu reserve in organs. Low blood Cu and low Cu content of hair.	Cu feeding gave very good results	Cu content of pasture mostly >5 p.p.m. D.M. Hay 3.6 to 4.65 p.p.m. and low Mn content. Mo not abnormal. No other obvious abnormality.			Probably Cu deficiency state but no absolute deficiency of Cu demonstrated in the pasture
Scouring Disease ("Teart" pastures)	Cattle, Sheep	Britain	Somerset		Cu feeding preventive and curative	Pasture well supplied with Cu, 11 to 18 p.p.m. D.M. High content of Mo, 20 to 100 p.p.m. D.M., related to incidence of disease.	Manuring healthy herbage with Mo causes scouring in grazing cattle and increase in Mo content of the herbage	The disease has been produced by feeding inorganic salts of Mo to cattle	Direct cause is the abnormal Mo content of the pasture. This may indirectly interfere with Cu metabolism; hence effect of Cu feeding.

See footnotes at end of table.

TABLE 1—SUMMARY OF INVESTIGATIONS OF RELATIONSHIP OF SOIL AND NUTRITIONAL DEFICIENCIES (*Continued*)

Disease	Species Affected	Country	Principal Areas Investigated	Blood and Tissues	Response to Feeding Tests	Pasture Characteristics	Effect of Specific Fertilizers on Incidence of Disease	Experimental Production of Disease	Conclusions
Licking Diseases [1]	Cattle, Sheep, Goats	Holland Sweden	Sandy and moor soils especially reclaimed soil, Gisselas		Cu feeding preventive and curative Controlled by feeding molasses containing 14 to 16 p.p.m. of Cu	Cu in hay usually between 2 and 3 p.p.m. D.M. Cu in hay 0.5 to 5.2 p.p.m. Average 2 p.p.m.			Cu deficiency due to lack of Cu in pasture Probably ditto
Grass Tetany	Cattle, principally lactating cows	Holland		Low blood Mg; often low serum Ca	Cured by injection of Ca+Mg	Protein and KNO_3 high. K: Na wide, sometimes > 50:1. Ca usually rather low. Mg apparently normal		Not produced on diets high in protein or in KNO_3	No conclusion regarding etiology of grass tetany can be drawn from the available data
Grass Tetany	Ditto	New Zealand		Mg content of milk, bones and organs of affected cows within normal limits. Blood low in Mg, often high in inorganic P.	Ca+Mg therapy gave no useful results but feeding of silage with added dolomite is beneficial	Protein high, P high, Ca low, K: Na ratio normal. Mg considered slightly subnormal.			
Ditto	Ditto	Britain	Lincolnshire	Low blood Mg, often low serum Ca, high Mn content of blood		High Mn content. Mg apparently normal.		Feeding Mn in the amounts which occur in these pastures depressed serum Mg but did not produce tetany	
Ditto	Ditto	Ireland		Low blood Mg, often low serum Ca		High Mn content. Mg apparently normal.			
Goiter	Cattle, Sheep, Goats, Horses, Pigs	Worldwide		Low I content of thyroids	Controlled by feeding I except in S. Africa and Sweden	Limited data indicate low I content of pasture and hay, but values considered healthy in some areas may be deemed goiter-producing in others			Iodine deficiency state due to lack of iodine in pasture or deficiency conditioned by unknown factors

See footnotes at end of table.

TABLE 1—SUMMARY OF INVESTIGATIONS OF RELATIONSHIP OF SOIL AND NUTRITIONAL DEFICIENCIES (*Continued*)

Disease	Species Affected	Country	Principal Areas Investigated	Blood and Tissues	Response to Feeding Tests	Pasture Characteristics	Effect of Specific Fertilizers on Incidence of Disease	Experimental Production of Disease	Conclusions
Aphosphorosis	Cattle, and to lesser extent Sheep	World-wide	Principally S. Africa	Low blood inorganic P	Controlled by feeding P	Low P content	P fertilizing beneficial	Can be produced experimentally on diets low in P but otherwise adequate	P deficiency state caused by lack of P in the pasture
Alkali Disease and Blind Staggers	Cattle, Horses, Sheep, and Pigs	U.S.A. and Canada		Se found in tissues which do not normally contain Se and in blood of affected animals only		High content of Se in vegetation. Toxic forage plants 4 to 25 p.p.m. of Se. Toxic weeds several thousand p.p.m. of Se		Similar symptoms and lesions have been produced in rats, pigs, and horses fed inorganic Se	Chronic and acute forms of Se poisoning due to ingestion of fodder containing abnormal concentration of Se and seleniferous weeds
"Forage Anemia," associated with Infectious Anemia	Horses	Scandinavia and Finland	Forest pastures of N. Sweden, Norway, and N. and E. Finland		Administration of yeast prevents appearance of symptoms of "forage anemia"	High Mn content in hay and pasture. Limited data indicate low vitamin B_1 content of hay	Liming, which reduces the availability of soil Mn to the plant, has a beneficial effect	Symptoms of "forage anemia" can be produced experimentally on vitamin B deficient diet and experimental animals show lowered resistance to anemia virus. Administration of Mn, twice the amount in affected hay produced symptoms of "forage anemia" in 1 of 2 horses.	It is concluded that "forage anemia" is a B_1 avitaminosis probably caused by oxidation of B_1 in pasture and hay in presence of large quantities of Mn. An additional effect may be caused by the effect of ingested Mn on vitamin B_1 metabolism. Such a vitamin B_1 deficiency predisposes to infectious anemia
Oat Hay Poisoning	Cattle principally	U.S.A. and Canada		Methaemoglobinaemia		High KNO_3 content of hay, 3 to 7 percent		The condition has been produced in cattle, sheep and horses by feeding KNO_3	The disease is caused by ingestion of large amounts of nitrate which is changed into nitrite in the rumen and causes nitrite poisoning.

[1] P.p.m. D.M.=parts per million of dry matter.
[2] — Signifies no information.
[3] — Licking diseases have also been investigated in Schleswig-Holstein, other parts of Germany, Switzerland, the Dalarna district of Sweden, and the coastal areas of Norway. The etiology has not been established, but there does not appear to be any sure foundation for the views that deficiency of Na or K or excess of K in relation to Na or low alkali-alkalescence have any causal connection with the disease. There is some evidence that in parts of Schleswig-Holstein deficiency of Cu may be a causal or at least a complicating factor.

3. Toxicity of Excess Food Constituents

Certain elements in the soil are assimilated by plants although they are not necessary for normal plant growth. Elements such as iodine, sodium, chlorine, and cobalt are required by animals. However, excesses of certain soil constituents, such as fluorine and selenium, are sometimes accumulated by plants through "luxury consumption." If these are toxic to animals, the plants are definitely injurious to the animals consuming them.

The following are the most important elements that occur in toxic quantities in plants, because of larger quantities than normal in certain agricultural soils.

Fluorine

In large doses fluorine is an acute poison. The harmful effects that are of practical importance in livestock feeding, however, are the result of the continuous ingestion of comparatively minute amounts in fluoride-containing water or in rations supplemented with mineral phosphates high in the element. The most evident effects are on the bones and teeth, in which excess fluorine gradually accumulates.

In hogs and cattle, defects in the enamel are produced and the teeth become soft and worn down until in some cases the pulp cavities are exposed. The teeth become sensitive to cold water and interfere with food consumption. Mottled enamel has also been reported to occur in cattle in areas where the water is high in fluorine.

While injuries to the bones and teeth are the initial or the most evident effects of fluorine, higher intakes or long-continued feeding interferes with food consumption, growth, and milk production. There are generalized toxic effects, however, reflected in degenerative changes in various organs and soft tissues. The form in which fluorine is fed has an influence on its toxicity. Sodium fluoride is more toxic than calcium fluoride and certain other insoluble products. There are also species differences in susceptibility: chickens tolerate a considerably higher level than other farm animals.

Fluorine is a cumulative poison. At first it merely accumulates in the bones and teeth without evident harm, and considerable time

elapses before structural injury becomes evident. The avidity of the bones and teeth for fluorine tends to protect the soft tissues against excessive concentration. As the bones become saturated, however, the greater part of the absorbed fluorine is free to produce its general toxic effects on the organs and soft tissues. While the effects of higher levels are evident much sooner, an intake of rock phosphate which provided approximately 0.008 percent of the total dry matter fed to dairy cows had a marked effect on production and reproduction after only three years of feeding. Thus, the level of fluorine is most critical in connection with long-time feeding of breeding stock and milk-producing animals.

From a study of the literature it appears that the upper limit of safety in the case of cattle, sheep, and hogs is represented by 0.01 percent of the element in the dry matter of the total ration. In the case of chickens, the maximum safe level is around 0.035 percent. Recommended tolerances should be a great deal below these levels to be safe under all conditions. Levels in the total ration of 0.003 percent for cattle, sheep, and swine, and 0.015 percent for chickens, or, for the concentrate mixture of sheep and cattle, 0.006 and 0.004 percent respectively are recommended.

Raw rock phosphates containing between 3.5 and 4 percent fluorine, as well as products such as acid phosphate and dicalcium phosphate, made from rock phosphate, are usually too high in fluorine to be used safely as a phosphorus supplement, except for short-time feeding of animals to be slaughtered. These products can be defluorinated to safe levels, but the defluorinated products are not yet widely available at low costs.

Selenium

This element has recently been found to be responsible for a peculiar disease of livestock which has long been known to exist in certain regions of the world. In the United States of America it occurs in some areas of the plains region, notably South Dakota, and is known locally as "alkali disease" or "blind staggers." Alkali disease was first noted in 1857 in the United States, but the cause was unknown for 75 years. In 1928 it was traced to the consumption of grain and other vegetation grown on definite soil areas, and a few years later the cause was found to be selenium in the grain. In chronic cases there is a loss of hair from the mane and tail in horses, and from the tail in cattle, and a general loss of hair in swine. The hoofs slough off, lameness occurs, feed consumption decreases, and death may occur by starvation. These external symptoms are accompanied by marked pathological changes which are revealed on autopsy.

Any soil that contains more than 0.05 p.p.m. of selenium is potentially dangerous. Plants vary greatly in the amounts they take up, but the concentration in the plant is generally much greater than in the soil. Chronic toxicity is caused by rations containing as little as 8.5 p.p.m. of selenium. Young animals are especially susceptible, and growth is retarded with levels too low to cause other evident symptoms.

No practical method has been found of eliminating selenium injury either by dietary means or soil treatment. In areas where it occurs, toxic levels in the food crops are so general that there is no safe food supply. There seems to be no solution other than to abandon the area. Certainly a livestock industry cannot flourish under these conditions, for the production of food crops constitutes a menace to both animals and humans. When cows and hens are fed rations containing selenium their milk and eggs contain this element.

Molybdenum

A trouble in cattle referred to as "teartness" and known for over a hundred years to be definitely associated with certain pasture areas in England has been established as a molybdenum toxicity. The trouble affects ruminants, particularly calves and cows in milk. The physical symptoms are anemia and extreme diarrhea, with consequent loss in weight and milk yield. The trouble is found where the herbage contains 0.003 percent or more of the element. Copper sulfate is effective in curing the diarrhea. An excess of molybdenum apparently decreases the availability of cobalt, according to investigation of this problem in England.

Nitrites

Cattle, horses, sheep, and swine may suffer from what has been called "oat-hay poisoning." It is really nitrite poisoning, the result of ingesting feed relatively high in potassium nitrate that is reduced to nitrite in the intestinal tract. The nitrites thus formed change the hemoglobin to methemoglobin, resulting in the characteristic physical symptoms of trembling, staggering gait, rapid respiration, and prostration. Oat hay and straw which caused deaths contained 2.2 to 7.3 percent potassium nitrate. Normally, only a trace is present. Other plants, such as certain weeds, wheat, barley, and cane sorghum, sometimes contain enough nitrate to cause the characteristic trouble. It is apparent that the troubles are limited to certain areas, with some evidence that they occur year after year in these

areas. It appears that oat-hay poisoning is of most concern in the case of ruminants because of the special opportunity for chemical changes provided by their digestive tracts. On the basis of present evidence, there appears to be no reason for concern regarding the possibility that nitrate fertilization may produce crops that are toxic.

Saline and Alkaline Waters

The water supplies for animals in certain regions have such high concentrations of various salts as to interfere with growth, lactation, and reproduction. The damage depends more on the total amount of salts present than on any specific one, thus representing an osmotic effect rather than an injury from any particular ion. The maximum concentration of soluble salts which can be safely tolerated appears to be between 1.5 and 1.7 percent. There is, however, a variation in the toxicity of the various salts. Sheep appear to be more resistant than cattle, and cattle more than hogs.

Selected References

BRADLEY, W. B., H. F. EPPSON, and O. A. BEATH. 1940. Livestock poisoning by oat hay and other plants containing nitrate. Wyoming Agr. Expt. Sta. Bull. 241.

HELLER, V. G. 1932. Saline and alkaline drinking waters. Jour. Nutr. *5*:421-429.

——— 1933. The effect of saline and alkaline waters on domestic animals. Okla. Agri. Expt. Sta. Bull. 217. Pp. 23.

MAYNARD, L. A. 1947. *Animal Nutrition.* 2d ed. New York: McGraw-Hill Book Co. Pp. 494.

MITCHELL, H. H. 1943. The fluorine problem in livestock feeding. Fourth report of the Committee on animal nutrition. National Research Council Reprint and Circ. Series 113.

——— and M. EDMAN. 1945. Fluorine in soils, plants and animals. Soil. Sci. *60*:1, 81-90.

MOXON. ALVIN L., and R. MORRIS. 1943. Selenium poisoning. Physiol. Revs. *23*:305-337.

4. Animal Losses Resulting from Improper Feeding

The amount of additional animal products that could be furnished if proper and adequate nutritional practices were carried out is naturally very hard, in fact impossible, to accurately estimate. It is possible, however, to point out specific losses that are avoidable, the result of poor nutrition.

It has already been pointed out that various nutritional diseases occur as the result of deficiencies of protein, minerals, and vitamins. These can either result in the death of the animal, which would be a total loss as far as human food is concerned, or result in an animal that is of poor quality, inefficient, unable to reproduce or supply any animal products such as milk, eggs, or in the case of draft animals, work. Numerous studies have shown that animals suffering from nutritional deficiencies are more susceptible to infection, whereas if the animal were properly nourished it would either resist the infection or enhance the recovery to carry on normal functions. Here again, it is impossible to estimate the losses resulting from nutritional deficiency.

However, the relatively small amounts of meat, milk, and eggs available as food for man in many parts of the world is probably due more often to simply insufficient animal feed or energy than it is to nutrient deficiencies in the rations available.

Losses in Reproduction

A deficiency in the amount of digestible nutrients supplied to breeding animals, as well as an unbalanced ration having a deficient supply of protein, results in a cessation of the estrus cycle, ovulation and sperm formation in extreme cases. Where the deficiency is not great enough to result in the complete cessation, it will at least cause interruptions and delayed breeding, which brings about costly production of the animal products. Notable amongst the vitamins in their direct effect on reproduction is vitamin A. When animals suffering from certain deficiencies have offspring, quite often they are born dead or weak and die in a short while, either from the deficiency or from secondary infection. This may

be the result of protein or calcium, phosphorus or iodine, as examples of mineral deficiencies, or the result of a vitamin A deficiency. In the male, a deficiency of vitamin A causes a very rapid degeneration of the germinal epithelium of the testes with a resultant loss of fertility. In the female, there may be, in the first place, a cessation or irregularity of estrus. This disturbance of the cycle is accompanied by a cornification of the vaginal epithelium. The extent of the deficiency determines whether there is a complete failure to breed or whether, after a delay, estrus occurs. If fertilization actually takes place, an injury to the placenta may occur which results in fetal death and resorption, or in abortion. In fact, reproductive failure more commonly results from placental injury than from cornification.

Milk and Eggs

The vitamin A value of milk is entirely dependent upon the amount present in the feed, and very large variations in vitamin potency may occur. The vitamin A content of milk may be several times as great on high vitamin A feeds as on feeds which contain very little of the vitamin. While the mineral content of eggs, except for iodine, is not influenced by the nature of the diet, there are marked effects in the case of several of the vitamins. This is particularly true of vitamin A and vitamin D. The kind of ration that results in the best production and hatchability is also the kind that provides eggs of the highest nutritive value for human consumption.

Underfeeding dairy cows results in the reduction of milk supply as much as 75 percent, and the length of the milking period is shortened. Animals fed inadequate rations of protein and total digestible nutrients utilize the fat, protein, and minerals of their bodies for production; if the ration is not corrected the animal will go into its next lactation in poorer condition, and the condition is further aggravated. Striking evidence of the effect of inadequate calcium and phosphorus nutrition on production has come from studies in phosphorus-deficient areas. In South Africa, the feeding of bone meal to cows on deficient pasture increased the milk production by 40 percent, while in the state of Minnesota, U.S.A., the addition of phosphorus increased the yield by 50 to 146 percent. The classical work of Theiler further showed that cattle subsisting on phosphorus-deficient forage, besides the lower milk yield, produced weaker calves of lower weight. The animals were stunted and late-maturing, and produced meat of poorer quality than cows that received bone meal

as a mineral supplement. The condition was aggravated by the animals suffering from osteophagia, which caused them to eat putrid and decaying flesh and bones.

Similar effects from severe calcium deficiency have been reported from Florida, U.S.A. Owing to the very low content of this element in roughage, broken hips and ribs were not uncommon in lactating animals. When the calcium intake was raised by the addition of bone meal, the yield per lactation increased by 50 percent and the cows became more persistent producers.

Losses in Meat

In beef production, growth and fattening of cattle depends on the feed eaten in excess of that used for maintenance. Therefore, the more feed consumed over the maintenance requirement, the greater the gain in weight. The edible portion of carcasses where animals have been improperly fed, and especially underfed, is much less and the fat content is less than if they had been adequately fed, skeletal growth being independent of muscle and fat formation.

The use of concentrated feeds enables the feeder to increase the con sumption of digestible nutrients, thus reducing the amount of feed required to raise a steer to slaughter-weight. Feeding at less than the maximum is only justifiable as a mode of salvaging grazing and feeds for which no other use can be found.

Wool and Hides

Recent investigations have shown that the wool and hides are affected by the feeding of the animals. If the animals are underfed the wool is lighter in weight and the length and diameter of the fibers are smaller. There is also a loss from shedding which would not occur if the animal were receiving sufficient digestible nutrients. The hides of lambs adequately fed have been found to have greater strength, tear resistance, and stretch than the hides of underfed lambs.

Work Losses

Draft animals that do not receive sufficient digestible nutrients to carry out the work required of them lose weight, show signs of fatigue more readily than normal, and their efficiency and usefulness is reduced. If, however, the deficiency is one that affects the health of the animal, such as vitamin A, calcium, or phosphorus, lameness results and the animal's usefulness decreases.

This is but a brief outline of the ways improper feeding can decrease the efficiency of yield of animal products for human consumption.

Selected References

BECKER, R. B., W. M. NEAL, and A. A. SHEALY. 1934. Effects of calcium deficient roughages on the milk yield and bone strength of cattle. J Dairy Sci. *17:*1-10.

CRUICKSHANK, E. M. 1941. The effect of diet on the chemical composition, nutritive value and hatchability of the egg. Nutrition Abstracts & Revs. *10:*645-659.

DAVIDSON, H. R. 1930. Reproduction disturbances caused by feeding protein-deficient and calcium-deficient rations to breeding pigs. Jour. Agr. Sci. *20:*233-264.

ECKLES, C. H., T. W. GULLICKSON, and L. S. PALMER. 1932. Phosphorus deficiencies in the rations of cattle. Minn. Agr. Expt. Sta. Tech. Bul. 91.

HART, G. H., and H. R. GUILBERT. 1933. Vitamin A deficiency as related to reproduction in range cattle. Calif. Agr. Expt. Sta. Bul. 560.

IMPERIAL BUREAU OF ANIMAL NUTRITION. 1932. Nutrition in relation to reproduction with special reference to sterility in farm animals. Imp. Bur. Anim. Nutrition Tech. Comm. 2. Pp. 16.

HUGHES, J. S., C. E. AUBEL, and H. F. LEINHARDT. 1928. The importance of vitamin A and vitamin C in the ration of swine. Kansas Agr. Expt. Sta. Tech. Bul. 23.

KEYS, ANCEL. 1943. Physical performance in relation to diet. Federation Proc. *2:*164-187.

MCKEENAN, C. P. 1940, 1941. Growth and development in the pig with particular reference to carcass quality characters. Jour. Agr. Sci. *30:*276-343, 387-436, 511-569; *31:*1-49.

MILLER, R. F., G. H. HART, and H. H. COLE. 1942. Fertility in sheep as affected by nutrition during the breeding season and pregnancy. Calif. Expt. Sta. Bul. 672.

MITCHELL, H. H. 1943. The fluorine problem in livestock feeding. National Res. Coun. Repr. and Circ. Series No. 113.

MEIGS, EDWARD B., and H. T. CONVERSE. 1933. Some effects of different kinds of hay in the ration on the performance of dairy cows. Jour. Dairy Sci. *16:*317-328.

ORR, JOHN BOYD. 1929. *Minerals in Pastures.* London: H. K. Lewis & Co., Ltd.

PHILLIPS, PAUL H. 1942. Nutrition and reproduction of farm animals. Third Rept. of the Committee of Animal Nutrition and Circ. No. 112, Nat. Res. Council (Reprint).

THEILER, A., H. H. GREEN, and P. J. DUTOIT. 1928, 1924. Studies in mineral metabolism. III. Breeding of cattle on phosphorus deficient pastures. Jour. Agr. Sci. *18:*369-371. Phosphorus in the livestock industry. J. Dept. Agr., Union S. Africa, *8:*460-504.

WALLIS, G. C. 1938. Some effects of a vitamin D deficiency on mature dairy cows. Jour. Dairy Sci. *21:*315-333.

WATERS, H. J. 1908. The capacity of animals to grow under adverse conditions. Proc. Soc. Promotion Agr. Sci. (29th Ann. Meeting). "How an animal grows," pp. 71-96.

WATSON, D. M. S. 1943. Beef cattle in peace and war. Empire Jour. Expt. Agr. *11:*191-228.

5. Feed Wastage Caused by Unbalanced Rations

The importance of using properly balanced rations for all classes of livestock has been pointed out from the standpoint of the animals' health, rate of growth, reproduction, and production. However, the fact that proper feeding saves feed, more than would be expected, is more important. Any grain feed that can be saved by efficient feeding can be used to increase the production of animals or animal products, or converted into human food for direct consumption. The magnitude of this loss cannot be thoroughly appreciated—staggering amounts of grain are wasted by improper feeding, even in countries with supposedly efficient agriculture.

Swine

Swine are more apt to suffer from nutritive deficiencies than other farm animals, except poultry. Many rations that were formerly considered satisfactory have been shown by careful experiments to be strikingly inefficient under certain conditions. It is necessary not only to feed "balanced rations" that supply enough digestible crude protein, but also to supply combinations of feeds with protein of the proper quality and ample vitamins and minerals.

Though differing materially in protein content, all of the cereals are low in percentage of this nutrient. While barley, oats, wheat, rye, and kafir contain somewhat more protein than corn, milo, rice, and feterita, none of the cereal grains provides a sufficient amount of protein or protein of proper quality to permit growing and fattening pigs to make rapid and economical gains when they are fed the grain without any protein supplement.

Fully as important as low protein content is the fact that the proteins of the cereals are incomplete in amino-acid composition. To produce pork economically, the deficiency in quality of protein must be corrected. This can be done by the use of such protein supplements as tankage, meat scraps, fish meal, and dairy by-products, which furnish ample amounts of the deficient amino acids. The fact that all the cereal grains are very low in calcium

must be borne in mind in swine feeding. It has been emphasized that suitable mineral supplements should be added to any rations that do not already have enough of these important mineral nutrients. The cereals are also deficient in vitamin D, and none of them, with the single exception of yellow maize, supplies appreciable amounts of vitamin A. All these deficiencies must be corrected in balancing a ration.

No single fact in stock feeding has been more clearly demonstrated by numerous feeding trials than that maize alone gives exceedingly poor results when fed to growing and fattening pigs. A glance at Table 2 should convince any farmer of the folly of feeding such an inefficient ration. The table summarizes the results of seven trials in which maize alone, without pasture, has been fed to young pigs averaging 31.3 kg. in weight, in comparison with a balanced ration of maize and tankage. In these trials, the average daily gain on maize alone was only 268 gm., and 642 kg. of maize were required per 100 kg. gain. This was a poor showing, but the results would have been even worse if the pigs had been started on this inadequate ration when still younger. When maize was balanced with tankage, the gains were doubled, and only 387 kg. maize and 42 kg. tankage were consumed for each 100 kg. gain. Furthermore, at the end of the trials the pigs fed maize alone were usually stunted and averaged only 63.9 kg. in weight, while those fed tankage in addition weighed over 91 kg. and were ready for market. The folly of feeding such an unbalanced ration as maize alone is shown by the fact that in these trials 100 kg. of tankage saved 607 kg. of maize, plus the advantage of more rapid gains.

TABLE 2

Maize Alone vs. Maize and Tankage for Growing and Fattening Pigs

Average	Average length of trial	Daily gain	Feed for 100 kg. gain	
			Maize	Tankage
	Days	*Gm.*	*Kg.*	*Kg.*
Trial with young pigs (31.3 kg.):				
Lot I, maize alone:				
Maize, 1.59 kg.	122	268	642	. . . . .
Lot II, maize and tankage:				
Maize, 2.00 kg.:				
Tankage, 0.22 kg.	122	535	387	42

Although the value of tankage as a supplement to maize has been emphasized in this discussion, tankage is not superior to other protein-rich supplements. Skim milk, buttermilk, fish meal, and combinations of other feeds give fully as good results. The advantage of good pasture must also be stressed.

On barley, wheat, or oats, as the only feeds, or on these grains plus a mineral supplement, the gains may be somewhat better than on maize alone, because these grains supply more protein and protein of somewhat better quality than does maize. However, in the case of pigs not on pasture, the gains are greatly increased by the addition of a sufficient amount of a good protein supplement to balance the ration.

When the feed of pigs in dry lot is restricted they commonly make slow gains and require a large amount of feed per 100 kg. gain. This is because they then need most of their feed for maintaining their bodies. For example, in tests, pigs fed only a half ration from an average weight of 28.1 kg. to market weights of 90 or 100 kg., gained only 295 gm. per head daily and required 428 kg. of feed per 100 kg. gain. Others, full-fed the same well-balanced mixture, gained 630 gm. a day (more than twice as much) and required only 391 kg. of feed per 100 kg. gain. The pigs receiving the half ration took 224 days to reach market weights. The labor was therefore practically doubled by the limited feeding, and the miscellaneous expenses were also increased.

Lambs

Numerous experiments have proved that fattening lambs do not make rapid or economical gains when they are fed only maize and nonlegume roughage, without a protein supplement. For example, in seven tests, lambs fed maize with timothy or prairie hay, without any supplement, gained an average of only 86 gm. per head daily, in comparison with 145 gm. for others fed maize and clover or alfalfa hay. The lambs on the unbalanced ration require 46 percent more maize and 15 percent more hay for each 100 kg. gain than those fed the balanced ration of maize and legume hay.

In four other trials the addition of 91 gm. linseed meal or cottonseed meal to a ration of corn and timothy hay increased the gain from 104 gm. per head daily to 136 gm. and also made a large saving in the amount of feed required per 100 kg. gain. On the average, each 100 kg. of protein supplement saved 186 kg. maize and 173 kg. hay, without considering the advantage of the more rapid gains. In these trials still better results would probably have been secured if a calcium supplement had also been added to the ration of maize and timothy hay.

Dairy Cows

The inadvisability of feeding dairy cows rations too low in protein is shown in recent investigations by the Bureau of Dairy Industry of the

United States Department of Agriculture. Two cows were fed a ration having an abundance of total digestible nutrients but supplying only 1.25 times as much digestible protein as was contained in the milk, in addition to an allowance of only 50 gm. digestible protein daily per 100 kg. live-weight for maintenance. On this ration the cows produced 22 to 50 percent less milk and fat than on a ration containing a liberal amount of protein.

Beef Cattle

Experiments have shown repeatedly that, even for fattening cattle, which do not need large amounts of protein, poor results are secured when the cereal grains are fed with only protein-poor roughages, like timothy or prairie hay, or forages from maize or the sorghums. Such rations that are deficient in protein, are apt to contain insufficient calcium, and may be rather low in phosphorus. An abundance of legume hay of good quality largely or entirely makes good this deficiency of protein and supplies an abundance of calcium for beef cattle.

The value of legume hay for supplementing the grains is well shown by the results of eight experiments. In each experiment one lot of steers, two years or older, was fed a ration consisting of only maize and protein-poor roughage (timothy hay, prairie hay, maize stover, or kafir stover), while another lot was fed maize and good legume hay. The steers fed the well-balanced ration of maize and legume hay gained 1.04 kg. daily on the average, and required only 689 kg. maize and 575 kg. hay for each 100 kg. gain. On the other hand, those fed the unbalanced ration gained only 0.77 kg. a day and consumed 930 kg. maize and 832 kg. hay per 100 kg. gain, thus requiring 36 percent more maize and 44 percent more hay for each 100 kg. of gain.

Similar results were secured in experiments where a protein supplement, such as linseed meal, cottonseed meal, or gluten feed, was added to a ration of only maize and protein-poor roughage. It was found that cattle fed unbalanced rations not only made slow and expensive gains but also they were apt to go off feed and to suffer from digestive disturbances. Even when fed for a long period, they did not reach as good a finish as those receiving balanced rations, and they sold for a considerably lower price.

These great differences occurred with cattle that were two years old or older. With calves or yearlings, the results of feeding unbalanced rations would have been even worse. For example, in a fattening trial, calves gained only 0.68 kg. per head daily on an unbalanced ration of

shelled maize, maize silage, and oat straw (with a small amount of bone meal added to supply calcium and phosphorus). Similar calves gained 1.09 kg. a day on a well-balanced ration, and gave a net return over cost of feed more than double that of those on the poor ration.

Since barley, oats, wheat, rye, and kafir usually contain appreciably more protein than maize, it is not necessary to use so large an amount of protein supplement to balance rations of these grains, fed with nonlegume roughage, as with maize. However, the use of the proper amount of supplement is very important. The amount of supplement needed with the various grains for fattening cattle of the various ages can readily be found by computing balanced rations according to the feeding standards outlined for beef cattle. When no legume hay is included in the ration, care should be taken to add a calcium supplement, except when the roughage has been grown on soils that are well supplied with this mineral.

From these few examples, it can be seen that a farmer, feeding unwisely, can use 50 to 100 per cent more feed to obtain a desired result and fail, considering labor and quality of product. In practice, extremes greater than these are often encountered.

The importance of balanced rations cannot be overstressed in the interest of saving animal feed and producing more human food.

Selected References

MADSEN, L. L. 1939. Factors affecting maintenance nutrition. Feed utilization and health of animals. U. S. D. A. Yearbook, *Food and Life*, pp. 431-449.

MORRISON, F. B. 1945 (c. 1936). *Feeds and Feeding*. 20th ed. Ithaca, N. Y.: Morrison Publishing Co. Pp. 1050.

WATSON, D. M. S. 1943. Beef cattle in peace and war. Empire Jour. Expt. Agr. *11*:191-228.

6. Symptoms of Nutritional Deficiencies in Dairy Cattle

The nutrient requirements of animals producing a large amount of milk differ greatly from the requirements of animals being fattened or of those doing muscular work. For efficient milk production it is essential that dairy cows receive: (1) A liberal amount of total digestible nutrients or net energy; (2) a relatively large amount of protein of the proper quality; (3) at least a certain minimum amount of fat; (4) sufficient phosphorus, calcium, common salt, and other essential minerals; and (5) an ample supply of vitamins A and D.

The amount of nutrients required by any particular milk cow depends on size, the amount of milk produced, and on the fat content. A heifer needs additional nutrients for growth. Pregnancy increases the nutrient requirement for the development of the fetus. This latter requirement is insignificant during the first part of the gestation period, and is not large even during the latter part.

In order to keep the recommendations in feeding standards relatively simple, they are usually based merely on (1) the size of cows and (2) on the amounts and the richness in fat of the milk they are producing. It must therefore be understood that during the last half of the gestation period, and also for heifers, the feed supply should be a little more liberal than is called for by the recommended standards.

The following symptoms are shown by dairy cattle as a result of nutritional deficiencies in the ration.

Insufficient Energy Intake

The symptoms of inadequate energy intake vary with the degree of the deficiency. With milking cows a drop in milk production is first observed. In growing animals there is a stoppage or slowing of the growth rate and varying states of emaciation depending upon the magnitude of the deficiency. The coat tends to be rough. The desire for feed is good unless complicated by other deficiencies.

Protein Deficiency

Little is known about the specific symptoms of protein deficiency. They are similar in part at least to those of insufficient energy intake. Affected animals have a limited appetite for low-protein diets.

Salt (NaCl)

Salt deficiency is manifested by an intense craving for salt, a lack of appetite, a generally haggard appearance, lusterless eyes, and a rough coat. In milking cows there is a rapid loss in live weight and milk production. In high-producing cows, collapse may be sudden and death may rapidly ensue. Salt deficiency in calves is reflected in an unthrifty condition and a harsh coat.

Calcium

The feeding of rations low in calcium over a long period of time may bring about a depletion of calcium and phosphorus in the bones, resulting in fragile bones that are easily fractured. (Figures 3, 4, and 5.) No other clinical symptoms are manifested, although there is some evidence that milk production may decline. The ratio of calcium to phosphorus is an important factor, and with various species wide ratios have been shown to depress the utilization of these elements, as compared with a ratio of 1:1 or 2:1.

Phosphorus

The first evidence of phosphorus deficiency is a decline of inorganic phosphorus in the blood plasma to subnormal levels. The normal values for cows are 4 to 6 mg. per 100 cc. and for calves under one year of age 6 to 8 mg. per 100 cc. Anorexia is the first specific sign of phosphorus deficiency following a drop in plasma inorganic phosphorus. Depraved appetite, the chewing of substances such as bones, wood, hair, rags, etc., may be observed at any stage of the deficiency. Cows, however, may suffer from extreme phosphorus deficiency manifesting depraved appetite (Figure 6). Also, secondary deficiencies, as a result of anorexia, may bring about depraved appetite. In calves, subnormal inorganic phosphorus values may be associated with a deficiency of vitamin D. Under farm conditions, however, older cattle exposed to solar radiation and fed sun-cured hay are not known to suffer from a vitamin D deficiency.

Figure 3. Both hips of this cow have been broken (knocked down) as the result of a low-calcium ration.

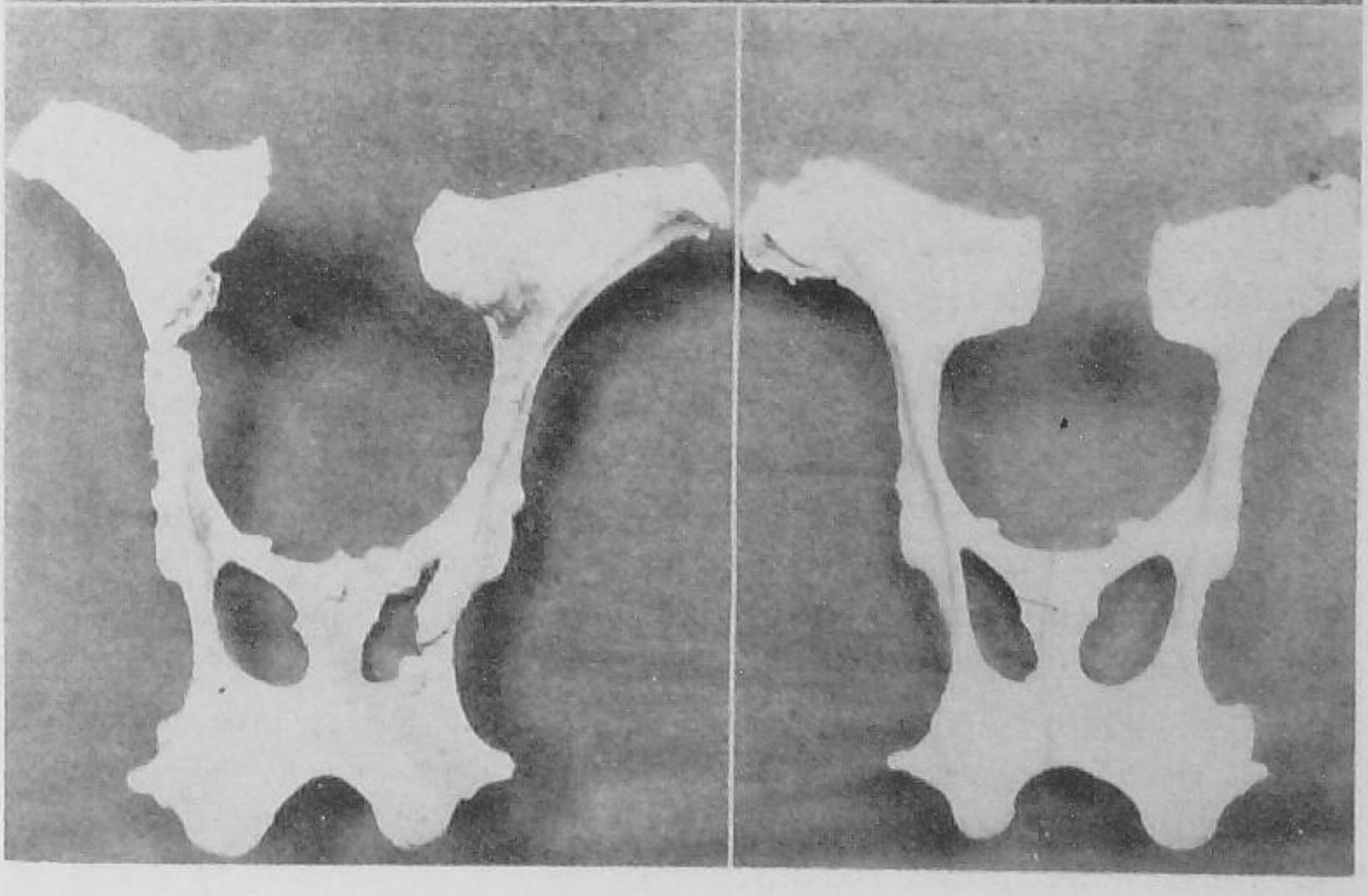


Figure 4. The pelvis of this cow suffered three breaks while the cow received a low-calcium ration.

Figure 5. The pelvis of the cow in Figure 3, showing the breaks involving both hip bones.

In chronic phosphorus deficiency the animals may become stiff in the joints. Upon post-mortem, the articulatory cartilages may appear eroded. In the growing calf, low-phosphorus rickets may develop, affecting the bones in the same manner as in vitamin D deficiency. The bones of cows with phosphorus deficiency become fragile, owing to calcium and phosphorus withdrawal. In phosphorus deficiency produced with low-phosphorus, high-calcium leguminous roughages, fragile bones are not observed. It has been noted that loss of appetite is the most pronounced symptom of phosphorus deficiency; low blood inorganic phosphorus is the usual precursor of anorexia; depraved appetite is not a good criterion in diagnosing phosphorus deficiency when alfalfa is used as the principal source of protein. In this investigation the chewing of wood or hair could not be correlated with low blood plasma inorganic phosphorus or with abnormal appetite for dry matter. As a matter of fact, several cows receiving a phosphorus supplement showed depraved appetite. The possibility of some other deficiency, such as lack of cobalt, cannot be ruled out. In unpublished data from the Michigan Agricultural Experiment Station (U. S. A.) obtained from a study of phosphorus deficiency in cattle under farm conditions, depraved appetite occurred among cattle suffering from mild phosphorus deficiency, but no evidence of depraved appetite was observed among cattle that showed marked evidence of phosphorus deficiency as indicated by very low inorganic plasma phosphorus, stiffness, and fractured bones. More research is needed to clarify the symptoms of uncomplicated phosphorus deficiency.

Iron

Iron-deficiency studies of cattle, as reported in the literature, have been complicated by the possibility of accompanying cobalt deficiency. Therefore, the symptoms of simple iron deficiency in cattle do not usually occur. It can be assumed, however, from work with other animals, that iron deficiency results in anemia. Since iron salts used as supplements usually contain appreciable quantities of cobalt, reliable data on the iron requirements are not available.

Copper

Copper deficiency is manifested by unthriftiness, depraved appetite, and anemia. Temporary sterility, owing to the suppression of estrus, is frequently observed. Young animals show evidence of malnutrition and abnormal development. The pasterns are straight and the calves tend to

stand on their toes. "Falling disease" in cattle occurs because of copper deficiency. The most distinguishing feature of copper deficiency is anemia. Diarrhea may occur. Bleaching of the hair coat has been reported. Post-mortem examination reveals the primary lesion to be starvation atrophy of the myocardium, with replacement fibrosis. Hemosiderin and other pathological changes are observed in the spleen and usually in the kidney and liver.

Cobalt

When the ration contains insufficient cobalt, animals may show a gradual loss of appetite, progressive emaciation, rough coat, scaliness of skin, listlessness, retarded development of sexual characteristics, and anemia (Figures 7 and 8). Anemia is indicated by a decrease in hemoglobin values and in the number of red-blood cells. These changes may not be discernible until late in the course of the disease. There appears to be a reduction in blood volume. In cows, there is a marked decrease in milk production and body weight. Daily intakes of 5-15 mg. have cured the symptoms in cattle. The actual requirement is not known.

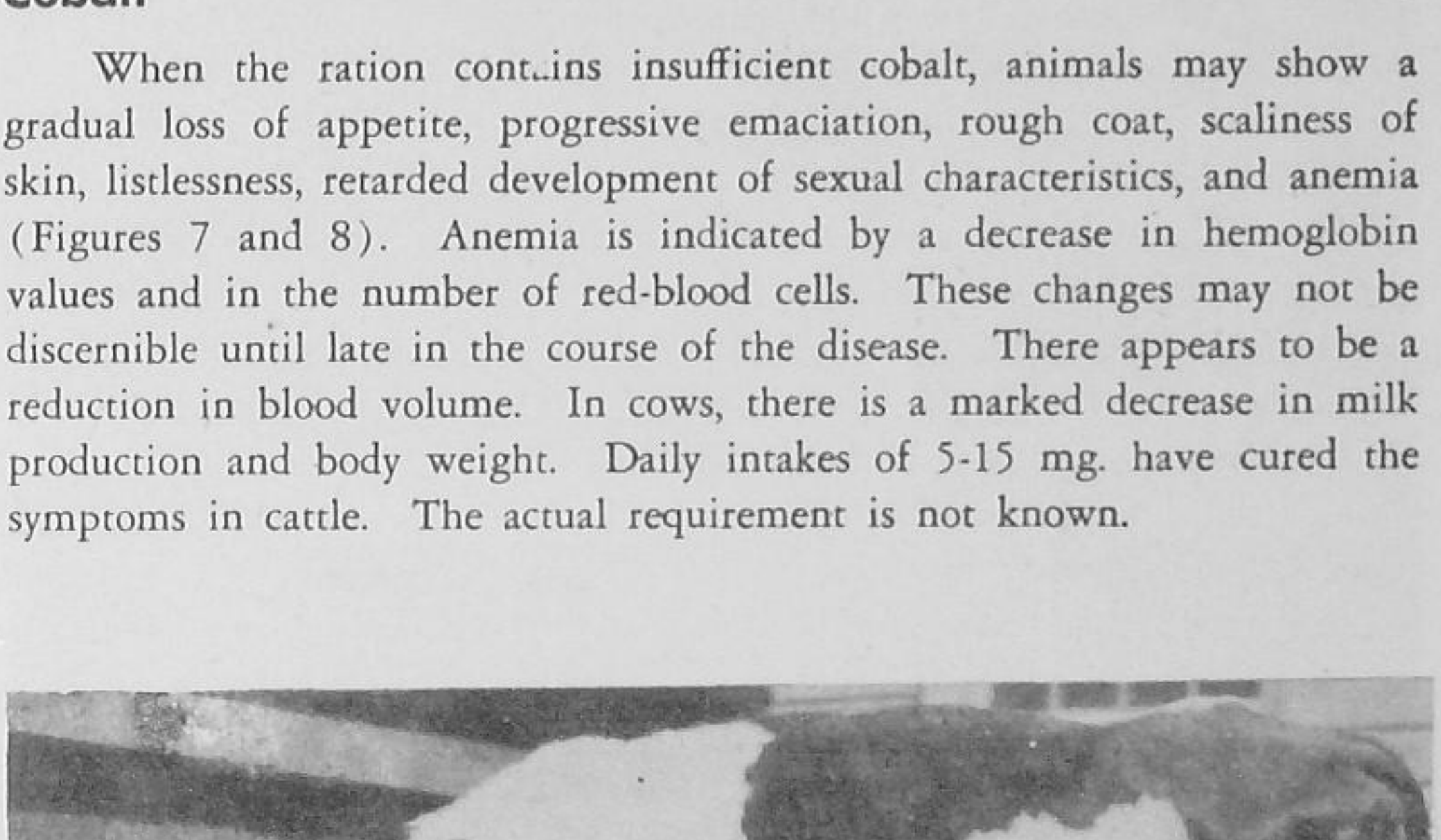


Figure 6. This cow, suffering from phosphorus deficiency, is exhibiting depraved appetite by chewing on a bone.

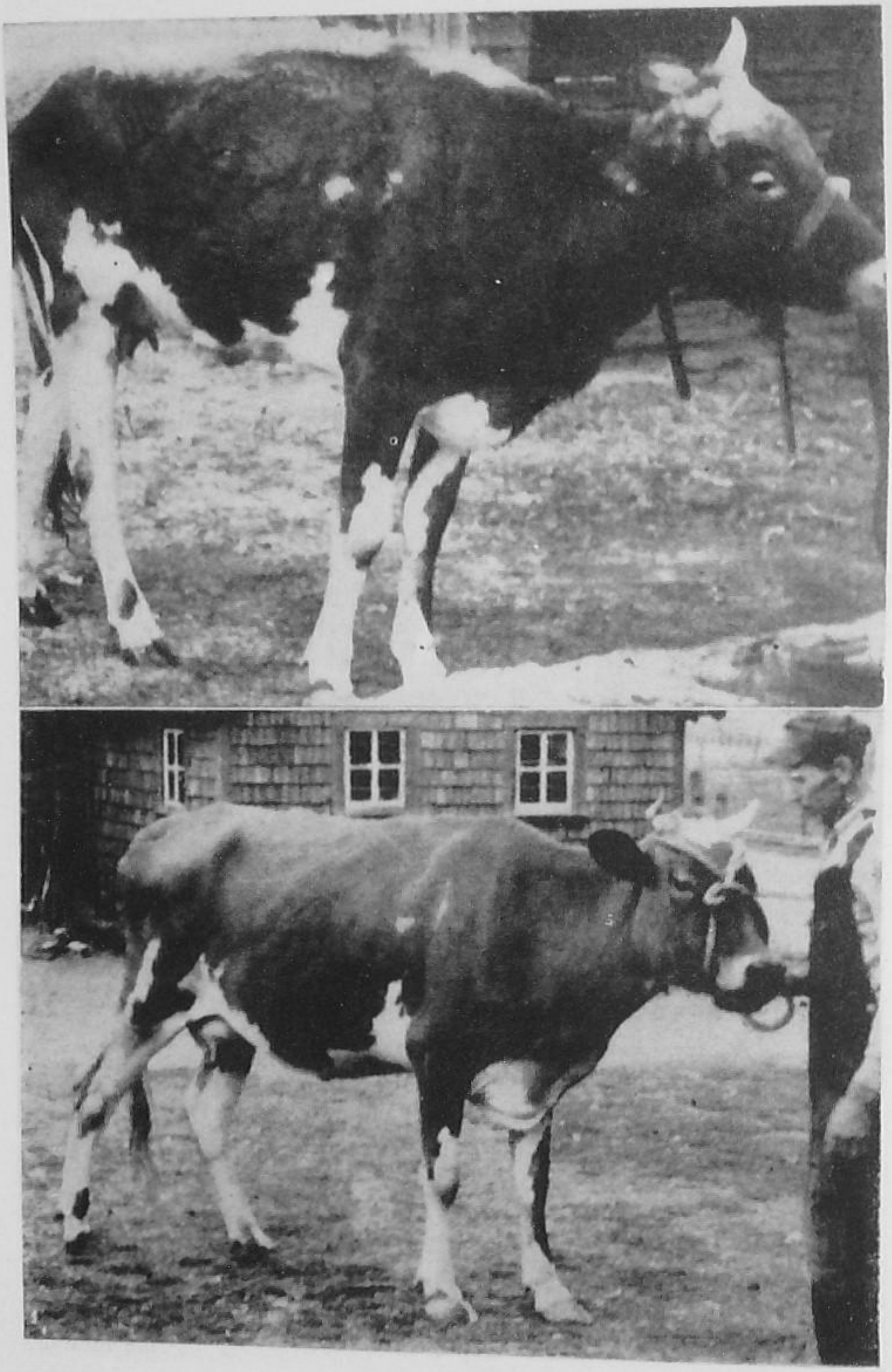


Figure 7 (above). A milking cow suffering from cobalt deficiency.
Figure 8 (below). The same cow after 16 days of cobalt feeding.

Calves are more subject to the disease than cows and often succumb at an early age. Diarrhea is frequently observed in cobalt-deficient calves. The feeding of cobalt results in improved appetite, usually within two to five days. Cobalt may be administered by feeding salt containing 33 gm. of cobalt sulphate or cobalt chloride per 100 kg. of salt. For calves, dissolve 6.6 gm. of either of the cobalt compounds in one liter of water and feed 4 to 5 cc. per calf per day.

Iodine

A deficiency of iodine in cows is usually manifested by the production of dead or nonviable goitrous calves as a result of a lack of iodine in the ration of the mother. There is a swelling of the thyroid gland of the calf, frequently referred to as "big neck." The trouble can be prevented by feeding iodized salt containing 0.015 percent iodine incorporated at a 1-percent level of the grain ration to the cow during the gestation period. When iodized salt is stabilized to retard the loss of iodine, a product containing 0.0076 percent iodine (0.01 percent potassium iodide) will probably provide the needed iodine supplement. The use of stabilized iodine is recommended.

Vitamin A

Under favorable farm conditions, all of the vitamins are generally furnished by natural feeds in adequate amounts to meet the needs of dairy animals. Certain conditions require special supplements of vitamins A and D. Only rarely is there a need for special sources of any of the other vitamins. Where dairy cattle are restricted and do not receive good quality green roughage or pasture over extended periods of time, vitamin A deficiencies may result, requiring special attention. When good-quality roughages are fed, the carotene intake is appreciably higher than the minimum values listed above. Carotene allowances are suggested to meet the vitamin A needs of dairy animals because carotene is the precursory substance of vitamin A obtained from plant sources. Vitamin A, as such, is not now generally fed, except to young calves. During the first few days after birth young calves should receive colostrum as a source of vitamin A and other essential factors. As an insurance measure it is wise to feed more vitamin A to very young calves than is necessary for the prevention of night blindness in order to prevent a high incidence of scours and death loss from pneumonia.

There is evidence of an increased demand for vitamin A during reproduction, especially during the last two or three months before calving, but the amounts needed for optimum performance are not known.

The earliest sign of a vitamin A deficiency is a lowered vitamin A level in the blood. The normal blood-plasma level for the young calf is 10 or more micrograms of vitamin A per 100 cc. of plasma. Cattle with a lower concentration than this soon develop clinical symptoms of a vitamin A deficiency. Levels of 7-8 micrograms of vitamin A per 100 cc. cause the calf to exhibit mild deficiency symptoms, while those with 5 micrograms of vitamin A or less exhibit all the symptoms associated with the advanced stages of the disease. The blood concentration of vitamin A in adult cattle during the long winter feeding period of the northern states of the U. S. A. invariably declines from a pasture level of 60 or more to about 15 micrograms per 100 cc. of blood plasma. Cows with blood plasma vitamin A concentration much below 15 micrograms may exhibit physiological dysfunction, such as "shy breeding." In the young calf, symptoms of the deficiency usually begin with "watery eyes," cold in the head with a nasal discharge, sometimes a cough, and scours or diarrhea—mild to begin with but severe if they continue. Calves exhibit these symptoms for several days to several weeks and usually succumb to pulmonary involvement, most often pneumonia.

The first easily detected gross symptom of vitamin A deficiency is night blindness, readily observed when animals are driven about in a dim light. Muscular incoordination. staggering gait, and convulsive seizures may develop as a result of an elevation of cerebro-spinal fluid pressure. Blindness in young growing cattle occurs without the classical signs of the vitamin A deficiency syndrome as the result of stenosis of the optic foramen and chronic optic neuritis. In these cases blindness develops without keratitis (corneal inflammation) and is accompanied by weakness, spasms, and paralysis. The eye changes are accompanied by the development of respiratory troubles. These symptoms arise as the result of epithelial metaplasis. A lack of vitamin A allows the transition from normal epithelial structures to stratified keratinized epithelium. The mucosa of the respiratory tract, buccal cavity, salivary glands, eyes, lachrymal glands, intestinal tract, urethra, kidney, and vagina are changed in the vitamin-A deficient bovine. Structures thus affected are very susceptible to infection and, as a result, colds and pneumonia frequently occur. Frequent convulsions are manifested in advanced stages of the deficiency. Diarrhea, loss of appetite, and emaciation are common features of the disease at this stage.

Subclinical vitamin A deficiency may be associated with the development of a roughened hair coat, general unthriftiness, emaciation, and dry pityriasis (flaky or bran-like scales of the skin) particularly about the neck, withers, and along the back extending to the tail-head. In the latter stages characteristic changes in the eye may take place: excessive lachrymation, keratitis, a softening of the cornea, xerophthalmia (dry form of conjunctivitis), opacity and cloudiness of the cornea, and total blindness from infection. In the pregnant animal, vitamin A deficiency results in abortion or birth at term of dead, weak, or blind calves (Figures 9 and 10).

Vitamin D

The deficiency of vitamin D conspicuously affects the growing bovine. One of the first symptoms of low vitamin D rickets is a decrease in the blood plasma concentration of calcium and/or inorganic phosphorus. These blood changes cause characteristic alterations in the bones, which indicate a markedly retarded calcification of the cartilaginous tissue.

Clinical symptoms begin with thickening and swelling in regions of the metacarpal (pastern or ankle) or metatarsal bone, or both. With the progress of the disease the forelegs bend forward, or sideways, or both. The joints, particularly the knee and back, become swollen and stiff, the pastern straight, and the back humped. In the more severe cases, synovial fluid accumulates in the joints. Posterior paralysis may occur as the result of fractured vertebrae. Advanced stages of the disease are marked by stiffness of gait, dragging of the hind feet, irritability, tetany, labored and fast breathing, anorexia except for milk, weakness, and retardation of growth (Figure 11). On autopsy, the gall bladder is frequently distended by accumulation of a viscous ropy orange-yellow bile. Enteritis not infrequently occurs.

Vitamin B Complex

Adult cattle obtain a sufficient supply of the vitamin B complex from natural feeds and bacterial synthesis in the rumen. In the young calf, however, there is danger of a deficiency of certain of the B vitamins.

Vitamin E

The need for vitamin E in the diet of cattle has not been demonstrated, nor is there evidence of rumen synthesis as for the B vitamins. Claims that vitamin E therapy gives beneficial results in reproduction or preventing abortion in cattle have not been substantiated.

Figure 9. This calf was born weak and blind and failed to survive because the cow was fed a ration too low in vitamin A activity.

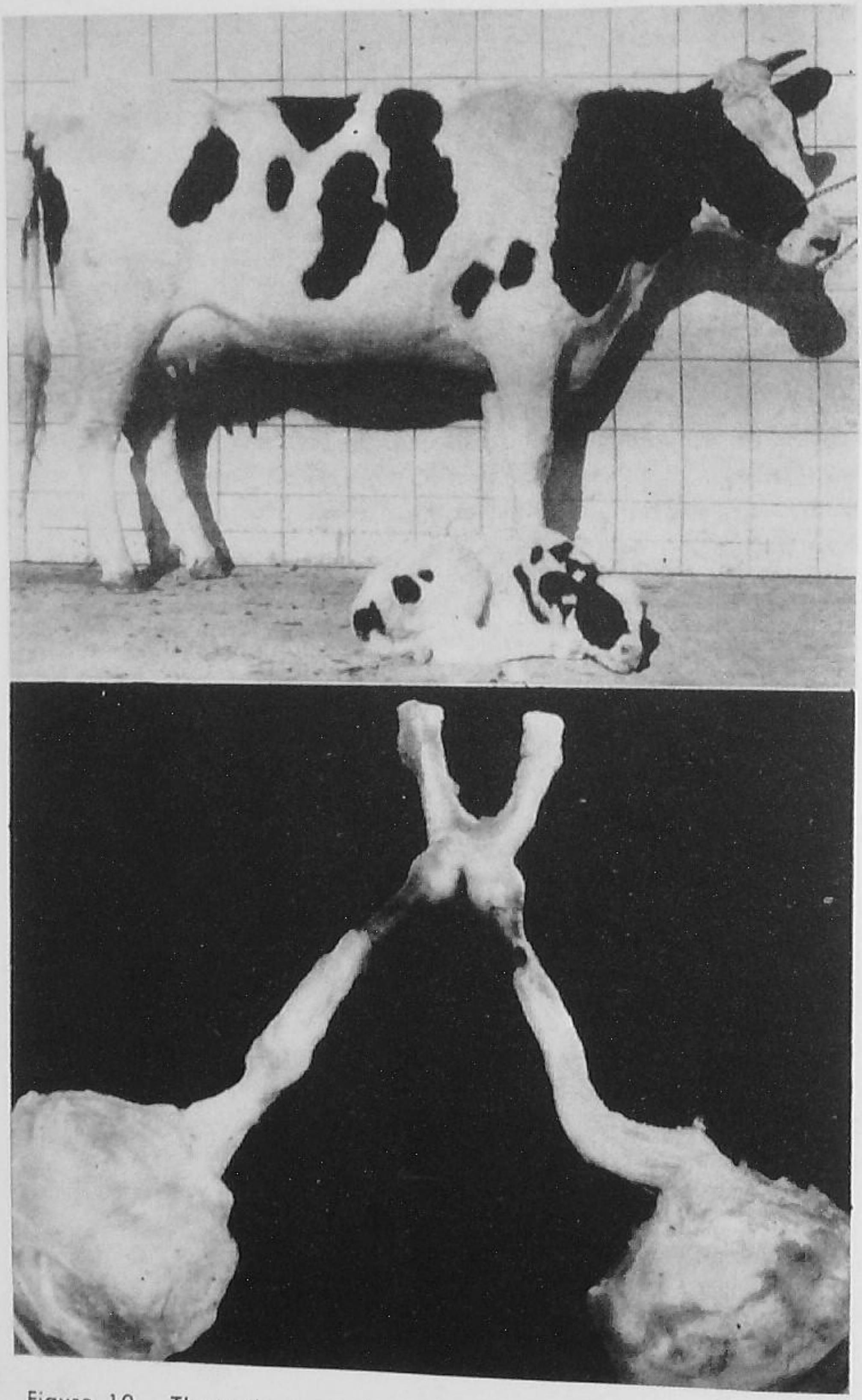


Figure 10. The optic nerves from a calf born weak and blind, showing constriction where they pass through the skull bones.

TABLE 3—Recommended Nutrient Allowances for Dairy Cattle

Weight of Animal	Total Digestible Nutrients	Net Energy	Digestible Protein	Calcium	Phosphorus	Carotene	Vitamin D
	DAILY ALLOWANCE PER ANIMAL [1, 6]						
	Kg.	**Therms**	**Gm.**	**Gm.**	**Gm.**	**Mg.**	**I. U.**
Growth							
23	0.45	1.0	136	4	3	([2])	150
45	0.91	2.0	204	8	6	6	300
68	1.36	2.9	272	12	8	10	450
91	1.81	3.8	317	13	9	12	600
181	2.95	5.9	363	14	11	25	1200
272	3.85	7.5	385	15	12	35	....
363	4.54	8.6	408	15	12	45	([3])
454	4.99	9.4	430	14	12	60	....
544	5.44	10.3	454	12	12	70	....
Maintenance [4]							
317	2.72	4.8	204	7	7	40	([3])
454	3.63	6.4	272	10	10	60	....
544	4.31	7.6	317	12	12	70	....
635	4.99	8.8	363	14	14	80	....
Pregnancy (per 454 Kg.)							
(Last 6 to 12 weeks)	6.35	11.8	544	22	17	90	([3])
Lactation (per kg. milk)[6]							
3.0% fat	0.28	0.57	40	2.2	1.5	([5])	([5])
4.0% fat	0.32	0.66	45	2.2	1.5	..	....
5.0% fat	0.37	0.75	50	2.2	1.5	..	....
6.0% fat	0.42	0.86	55	2.2	1.5	..	....

[1] Thiamin, riboflavin, niacin, pyridoxine, pantothenic acid, and vitamin K are synthesized by bacteria in the rumen and it appears that adequate amounts of these vitamins are furnished by a combination of rumen synthesis and natural feedstuffs. Manganese, iron, copper, and cobalt are clearly essential but the amounts needed are not known. For growth 1.3 gm. magnesium is needed per 100 kilograms body weight.

[2] Calves should receive colostrum the first few days after birth, as a source of Vitamin A and other essential factors.

[3] While Vitamin D is known to be required the data are inadequate to warrant specific figures for older growing animals and for maintenance, reproduction, and lactation. Adequate amounts are normally supplied by sun-cured roughage or provided by exposure to direct sunlight.

[4] When calculating the allowances for lactating heifers that are still growing, it is recommended that the figure for **growth** rather than **maintenance** be used.

[5] When adequate amounts of Vitamins A and D are fed for normal reproduction, extra amounts will probably not stimulate milk production but will increase the vitamin content of the milk.

[6] Salt (NaCl) should be supplied as 1 percent of grain mixture; in addition, dairy animals should have free access to salt.

Selected References

BOHSTEDT, G. 1942. Minerals in dairy cattle nutrition: A review. Jour. Dairy Sci. *25:*441-58.

BOYER, P. D., P. H. PHILLIPS, N. S. LUNDQUIST, C. W. JENSEN, and I. W. RUPEL. 1942. Vitamin A and carotene requirements for the maintenance of adequate blood plasma vitamin A in the dairy calf. Jour. Dairy Sci. *25:*433-440.

DAVIS, R. E., and L. L. MADSEN. 1941. Carotene and vitamin A in cattle blood plasma and observations on reproductive performances at restricted levels of carotene intake. Jour. Nutrition, *21:*135-46.

KEENER, H. A., S. I. BECHDEL, N. B. GUERRANT, and W. T. S. THORP. 1942. Carotene in calf nutrition. Jour. Dairy Sci. *25:*571-83.

LOOSLI, J. K., C. F. HUFFMAN, W. E. PETERSON, and P. H. PHILLIPS. 1945. Committee on Animal Nutrition, Report No. 3. *Recommended Nutrient Allowances for Dairy Cattle.*

MADSEN, LOUIS L. 1942. Nutritional diseases of cattle. U. S. D. A. Yearbook of Agriculture, *Keeping Livestock Healthy,* pp. 645-672.

MARSTON, H. R. 1939. Ruminant nutrition. Ann. Rev. Biochem. *8:*557-78.

MAYNARD, L. S. 1947. *Animal Nutrition.* 2d Ed. New York: McGraw-Hill Book Co. Pp. 494.

MEIGS, E. B. 1939. The feeding of dairy cows for intensive milk production in practice. U. S. D. A. Yearbook of Agriculture, *Food and Life,* pp. 566-591.

Figure 11. This calf developed severe rickets while deprived of sunlight and receiving a ration deficient in vitamin D.

MITCHELL, H. H., and F. J. MCCLURE. 1937. Mineral nutrition of farm animals. National Research Council Bul. 99, p. 135.

MORRISON, F. B. 1936. *Feeds and Feeding.* Ithaca, N. Y.: Morrison Publishing Co. Pp. 1050.

PALMER, L. S., T. W. GULLICKSON, W. L. BOYD, C. P. FITCH, and J. W. NELSON. 1941. The effect of rations deficient in phosphorus and protein on ovulation, estrus, and reproduction of dairy heifers. Jour. Dairy Sci. *24:*199-210.

PHILLIPS, P. H., N. S. LUNDQUIST, and P. D. BOYER. 1941. The effects of vitamin A and certain members of the B-complex upon calf scours. Jour. Dairy Sci. *24:*977-982.

SHEPHERD. J. B., and H. T. CONVERSE. 1939. Practical feeding and nutritional requirements of young dairy stock. U. S. D. A. Yearbook of Agriculture, *Food and Life,* pp. 597-638.

WALLIS, G. C. 1944. Vitamin D deficiency in dairy cows. Symptoms, causes and treatment. South Dakota Agr. Exp. Sta. Bul. 372, pp. 1-16.

TABLE 4.—CLINICAL SYMPTOMS OF DIETARY DEFICIENCIES IN SWINE

Deficient Nutrient	Slow or Interrupted Growth	Reduced Appetite	Poor Hair and Skin Condition	Lameness and Stiffness	Weakened Bone Structure	Hyperirritability	Convulsions	Diarrhea	Vomiting	Anemia	Impaired Reproduction	Dead or Weak Offspring at Birth	Other Effects
Energy	[1] +	[2] O	[3] . .	O	O	. . .	O	. . .	O	O	+	. . .	Reduced fatness in proportion to body weight.
Protein	+	+	. . .	O	O	. . .	O	O	O	. . .	. . .	+	Excessive fatness in proportion to body weight.
Calcium (Figs. 15 and 16)	+	+	+	+	+	+	. . . .	O	O	O	+	+	Severe cases may show reduced serum calcium.
Phosphorus	+	+	O	+	+	O	O	O	O	O	+	+	Reduced inorganic blood phosphorus.
Sodium	+	+	+	. . .	. . .	. . .	O	. . .	. . .	. . .	. . .	. . .	
Potassium	+	+	. .	+	. . .	[4] ±	O	. . .	. . .	. . .	. . .	. . .	Pain in joints, unsteadiness. Advanced cases, emaciation
Iodine	O	. . .	+	O	. . .	. . .	. . .	O	O	. . .	+	+	Birth of hairless pigs, goiter.
Iron and copper	+	+	±	O	O	. . .	O	. . .	. . .	+	. . .	. . .	Usually a disease of young pigs; high mortality, susceptibility to parasitic invasion, labored breathing on slight exertion (thumps).
Vitamin A (Fig. 13)	±	. . .	. . .	+	. . .	+	+	+	O	O	+	+	Lameness due to muscular incoordination, night and day blindness, weak malformed pigs, some diarrhea. Symptoms are slow in developing.
Vitamin D	+	±	O	+	±	+	±	O	O	O	. . .	. . .	Rickets and osteomalacia, enlarged joints, weak bones, lowered serum calcium, tetanic convulsions.
Thiamin (Fig. 12)	+	+	. . .	O	. . .	O	O	+	+	O	. . .	. . .	Flabby, degenerate heart muscle, slow pulse, low body temperature, rough hair in advanced cases.
Riboflavin	+	+	+	+	. . .	O	. . .	+	+	. . .	. . .	. . .	Stiffened limbs, sebaceous exudate over back and sides, secretions around eyes, cataract, nerve degeneration, yellow liver.
Niacin	±	±	+	O	O	O	O	+	O	. . .	. . .	. . .	Moderate slowing of growth, occasional vomiting, foul-smelling feces.
Pantothenic acid (Fig. 14)	+	+	+	+	O	O	O	±	±	. . .	. . .	. . .	Incoordinated, wabbly gait (goose-stepping), myelin sheath degeneration, scurfy, thin hair, brownish secretion around eyes, in severe cases bloody feces.
Pyridoxine	+	+	+	+	. . .	+	+	+	O	+	. . .	. . .	Epileptic-like fits, anemia, red cells small, hemoglobin deficient, slowing of growth after first convulsion.

[1] The symbol + indicates regular occurrence.
[2] The symbol O indicates absence of symptom.
[3] . . . indicates lack of information.
[4] The symbol ± indicates occasional occurrence.

7. Symptoms of Nutritional Deficiencies in Swine

The symptoms of various nutritional deficiencies are summarized in Table 4. In some cases, the symptoms are specific, but such conditions as reduced appetite, reduced growth, and unthriftiness are common to malnutrition in general.

Actual nutritional deficiency also may exist without the appearance of any definite symptoms. Conditions resulting from nutritional deficiency range all the way from slight tissue depletion or mild derangement of chemical processes to gross functional and/or anatomical lesions. The degree of severity that produces a recognizable clinical syndrome indicative of a deficiency in any particular dietary constituent varies with the animal and the constituent. Furthermore, because some acute conditions produced in the laboratory through the complete absence of some essential dietary constituent yield in a dramatic way to subsequent administration of the missing factor, it does not follow that chronic conditions of long standing will likewise recede when dietary adjustments are made. Indeed, many functional and anatomical lesions resulting from inadequate diets are irreversible.

Figure 12. Contrast in growth of litter mates owing to difference of thiamin intake. The pig on the right received no thiamin while the one on the left received the equivalent of 4.4 mg. of thiamin per 100 kg. live weight.

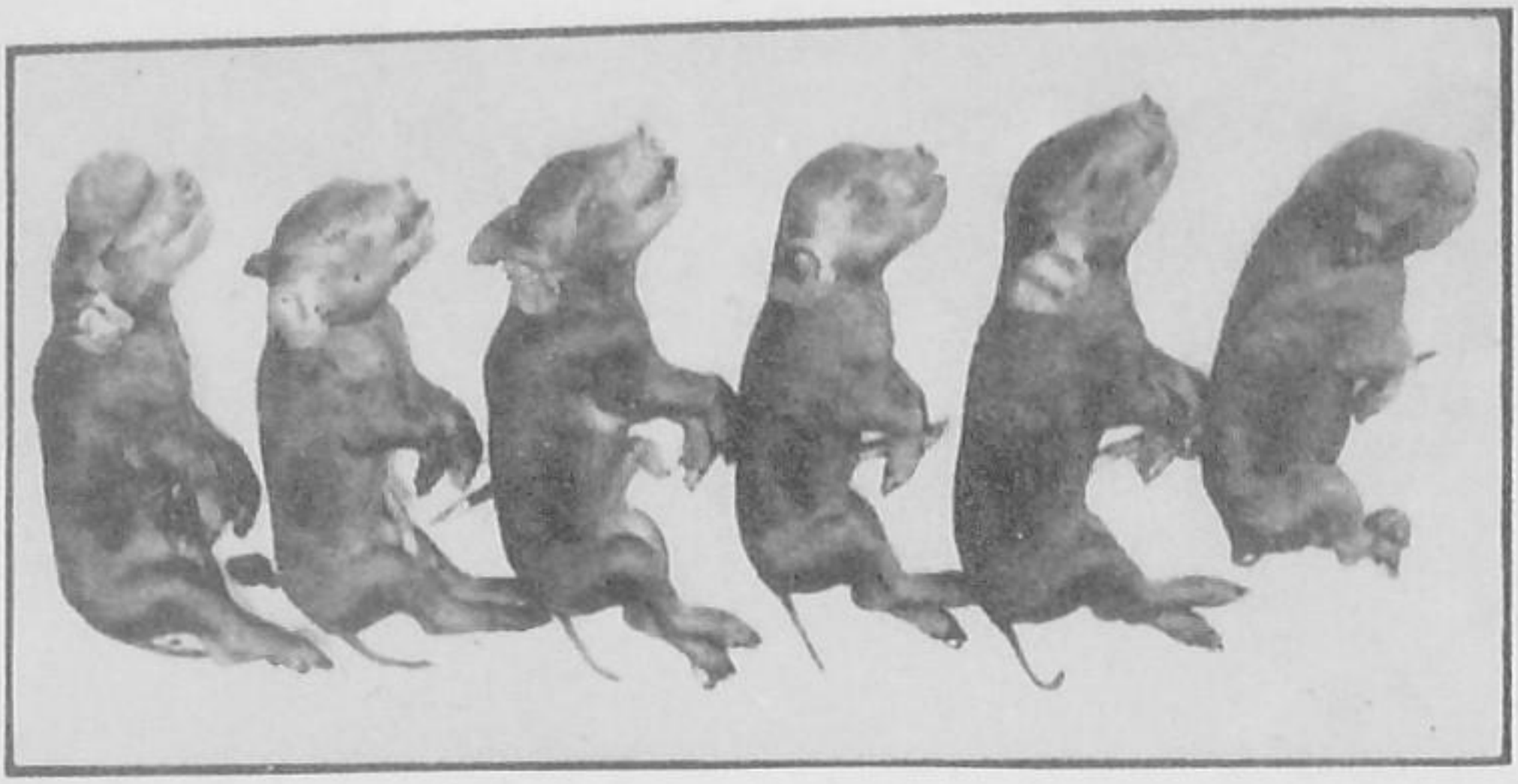
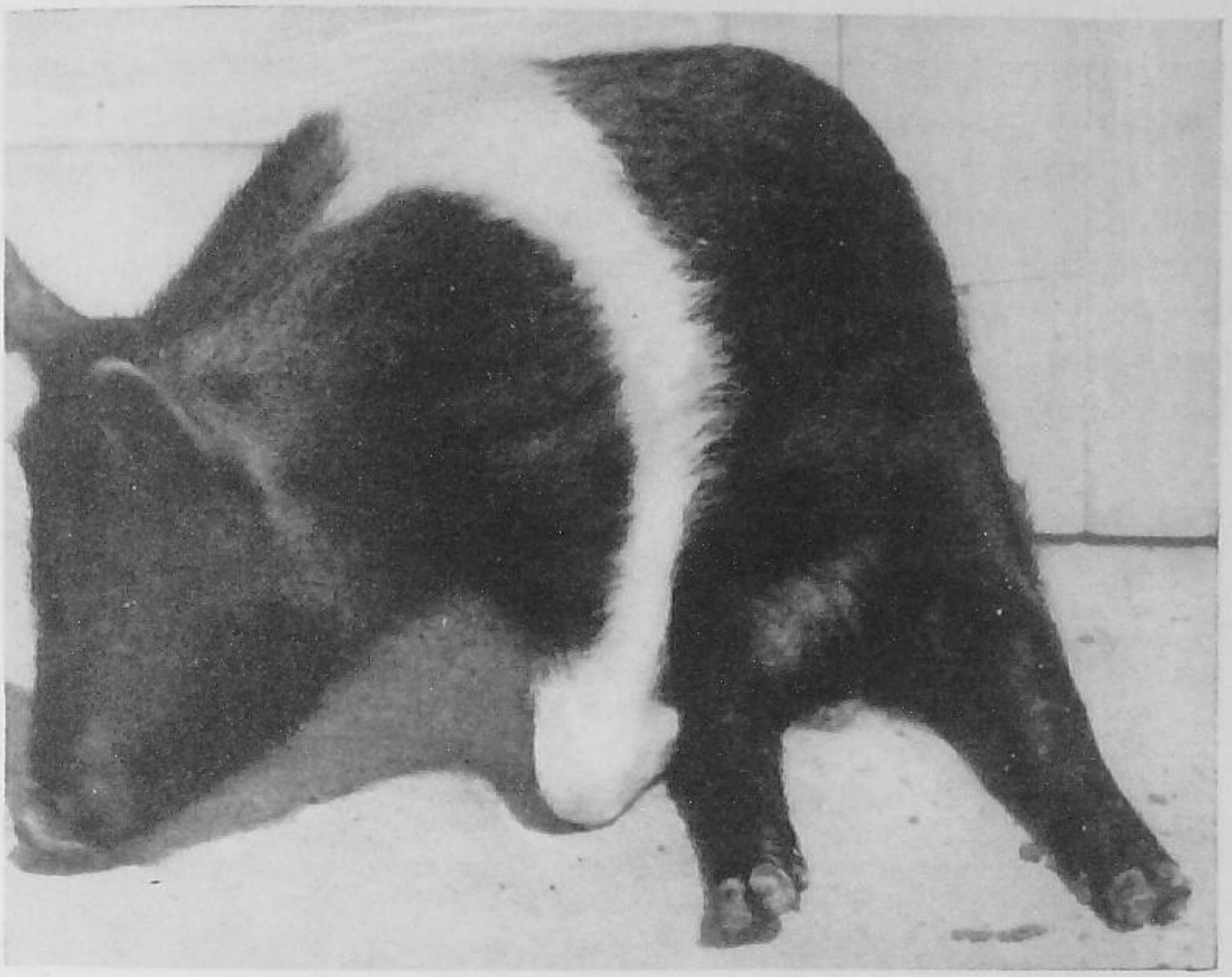
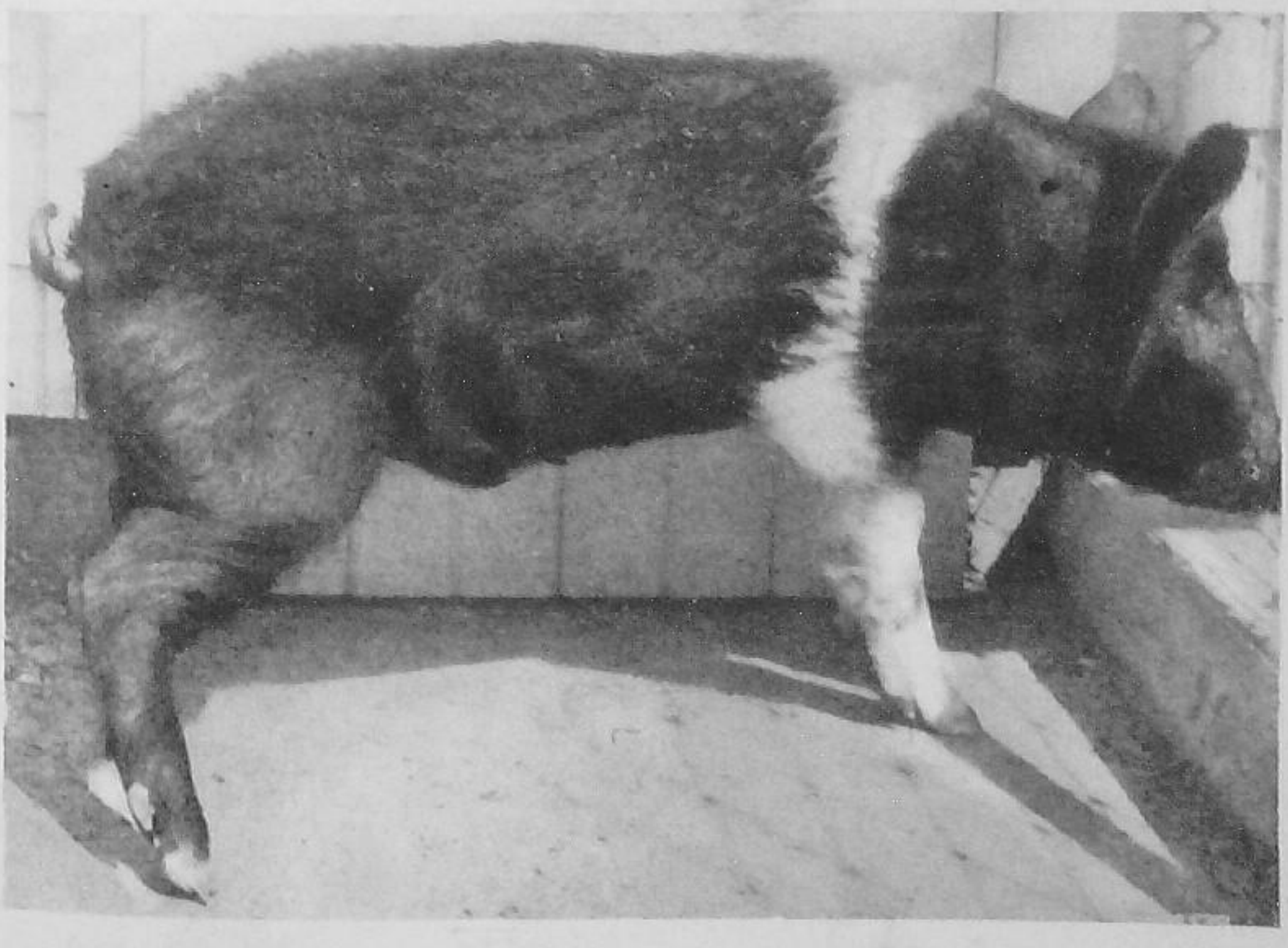


Figure 13. Litter of pigs born blind and with other abnormalities as the result of maternal vitamin A deficiency.

Figure 14. Moderate case of pantothenic acid deficiency showing "goose-stepping." This is the most striking symptom of an insufficient amount of this nutrient in the diet.



Figures 15 and 16. These pigs show symptoms of calcium deficiency. This condition developed after 12 weeks on a ration containing 0.25 percent of calcium. Note the lying position and the paralyzed hindquarters of the pig above and the weak and crooked hindlegs of the pig below.



The evidence gained from study of human deficiencies indicates that, in a large proportion of cases, the nutritional deficiency exists because some abnormal individual condition interfered with the proper utilization of the dietary factors supplied rather than because of a suboptimum initial intake. Thus, the uncertainty of diagnosis by the layman of a ration deficiency in animals, because of deficiency symptoms, is obvious. An actual nutritional deficiency may well be questioned unless the clinical symptoms are in evidence quite generally among pigs receiving the same feed and under the same management.

Selected References

ELLIS, N. R., and J. H. ZELLER. 1939. Nutritive requirements of swine. *Food and Life*, U. S. D. A. Yearbook of Agriculture, pp. 706-722.

——— and J. H. ZELLER. 1939. Practices in swine feeding. *Food and Life*, U. S. D. A. Yearbook of Agriculture, pp. 723-745.

HUGHES, H. E., E. W. CRAMPTON, N. R. ELLIS, and W. J. LOEFFEL. 1944. Recommended nutrient allowances for swine. National Research Council, U. S. A., Committee on Animal Nutrition. Report No. 2.

MADSEN, L. L. 1942. Nutritional diseases of swine. U. S. D. A. Yearbook of Agriculture, *Keeping Livestock Healthy*, pp. 810-827.

MAYNARD, L. A. 1947. *Animal Nutrition*. 2d ed. New. York: McGraw-Hill Book Co. Pp. 494.

MILLER, R. C., T. B. KEITH, W. T. S. THROP, and M. A. MCCARTY. 1943. Nutritive requirements of young pigs. Penn. Agri. Expt. Sta. Bul. 449.

MITCHELL, H. H., and F. J. MCCLURE. 1937. Mineral nutrition of farm animals. National Research Council Bul. 99.

——— and T. S. HAMILTON. 1936. The balancing of rations with respect to protein. Am. Soc. Anim. Prod. Proc. (1935). *28:*241-252.

MORRISON, F. B. 1945 (c. 1936). *Feeds and Feeding*. 20th Ed. Ithaca, N. Y.: Morrison Publishing Co. Pp. 1050.

WINTROBE, M. M. 1939. Nutritive requirements of young pigs. Amer. Jour. Physiol. *126:*375-387.

TABLE 5—Recommended Nutrient Allowances for Swine

CLASS	Live Weight	DAILY ALLOWANCE PER ANIMAL															
		Total Digestible Nutrients	Net Energy	Crude Protein	Calcium	Phosphorus	Sodium	Potassium	Carotene	Vitamin A	Vitamin D	Thiamin	Riboflavin	Niacin	Pantothenic Acid	Pyridoxine
Growing, fattening pigs.....	Kg.	Kg.	Therms	Kg.	Gm.	Gm.	Gm.	Gm.	Mg.	I. U.	I. U.	Mg.	Mg.	Mg.	Mg.	Mg.
	23	0.9	1.8	0.27	7.4	4.9	2.7	1.3	2.0	1,300	135	1.4	2.1	7.0	10.0	1.6
	45	1.7	3.5	0.36	13.7	9.1	5.0	2.5	4.0	2,600	250	2.5	3.8	12.5	18.5	3.0
	68	2.3	4.6	0.41	15.8	10.5	6.6	3.8	6.0	3,900	330	3.3	5.0	16.5	.....	.....
	91	2.5	5.1	0.45	17.9	11.9	7.5	5.0	8.0	5,200	375	3.8	5.7	19.0	.....	.....
	113	2.8	5.7	0.45	17.9	11.9	8.3	6.0	10.0	6,500	415	4.2	6.3	21.0	.....	.....
Pregnant gilts and sows; young boars.....	.....	2.0	4.1	0.41	16.4	10.9	6.0	6.0	20.0	13,000	300	3.0	.....	.....	.....	.....
Lactating sows; breeding boars.....	.....	3.4–5.1	7.0–10.5	0.68–1.04	27–41	18–27	12.5	12.5	40.0	26,000	625	6.3	.....	.....	.....	.....

NOTES: Vitamin D: This requirement may be fully met by ultra-violet radiation from the sun.

Thiamin: Amounts specified permit development of a normal pig but do not provide for thiamin storage.

Vitamin E: Required, but amounts unknown.

Iron: Requirements beyond weaning unknown. For suckling pigs 15 mg. iron daily for first three weeks maintains birth hemoglobin level.

Copper: Usually 5 percent of the iron administered.

Cobalt: For sheep, 124 gm. $CoSO_4$ per 1,000 kg. NaCl has been enough to relieve deficiency symptoms. Swine requirements unknown.

Iodine: For pregnant sows, 0.22 mg. iodine per 50 kgs. body weight has been proposed. Requirements for other swine are probably somewhat less.

Magnesium, Manganese, Zinc: Required, but amounts unknown.

8. Symptoms of Nutritional Deficiencies in Poultry

The more common gross pathological symptoms of poultry maintained on diets deficient in the various nutritional factors are discussed here. One shortcoming of such a description is that the symptoms are observed, for the most part, in poultry fed rations severely deficient in some specific factor. Under these conditions only the acute symptoms develop, which in most cases are quite characteristic for each nutritional factor, making a diagnosis relatively easy. On the other hand, the gross symptoms observed in cases of a chronic deficiency of any one of several factors may be similar (perhaps only retarded growth and ruffled plumage), making an accurate diagnosis difficult if not impossible. The chronic deficiency may be more serious in the long run than the acute, since in the latter case diagnosis and treatment may be readily obtained while the chronic deficiency continues to exist because of failure to diagnose it.

Vitamin A

On a severely deficient diet the symptoms of vitamin A deficiency begin to appear in approximately three weeks. Growth is markedly retarded, the chicks show general weakness, emaciation, staggering gait, and ruffled plumage. Resistance to infection is reduced and mortality is increased. Secretions fail in the intestinal mucous glands, the tear glands, and the salivary glands. An opaque appearance caused by keratinization of the third eyelid may be observed. Infection may occur, resulting in the production of a viscous fluid which may cause the eyelids to stick together (Figure 17).

Pathological lesions observed on autopsy are confined largely to the mucous membranes of the mouth, pharynx, esophagus, respiratory and urinary systems. Creamy white pustules are often found on the roof of the mouth and along the esophagus, sometimes extending into the crop (Figure 18). Ureates accumulate in the ureters and in the kidney tubules so that these organs are enlarged and creamy white in color. This ureate accumulation is detected easily on gross examination because of its whitish appearance (Figure 19).

In mature fowl the symptoms noted for chicks may develop much more slowly, but the eye disorder often becomes more acute. A cheesy exudate from the eyes often is observed, as well as a sticky discharge from the nostrils. Egg production and hatchability are markedly reduced.

The symptoms of a vitamin A deficiency in turkey poults are, in general, similar to those described for chicks, but are usually much more acute.

Vitamin D

A lack of vitamin D in the absence of direct sunlight results in the nutritional deficiency termed rickets. The chicks are retarded in growth, show a disinclination to walk, or walk with a lame, stiff-legged gait, and have an ungainly manner of balancing the body. The chicks appear generally unthrifty. In this disorder an upset occurs in the mechanism involving the absorption and retention of calcium and phosphorus, as a result of which these minerals are not deposited in normal amounts in the bony structure of the body. Abnormal bone development may be detected most readily in the legs, and at the junction of the ribs on the sides of the breast. The spinal column may be curved and the sternum usually shows acute lateral bending or depression. Enlargement of the hock joints and beading of the rib ends becomes apparent. The beak is soft and rubbery and may be easily bent (Figure 21).

Figure 17. Advanced stage of vitamin A deficiency. Note the exudate from the eye and the general ruffled appearance.

Figure 18. An advanced case of vitamin A deficiency, showing the pharynx and esophagus studded with pustules.

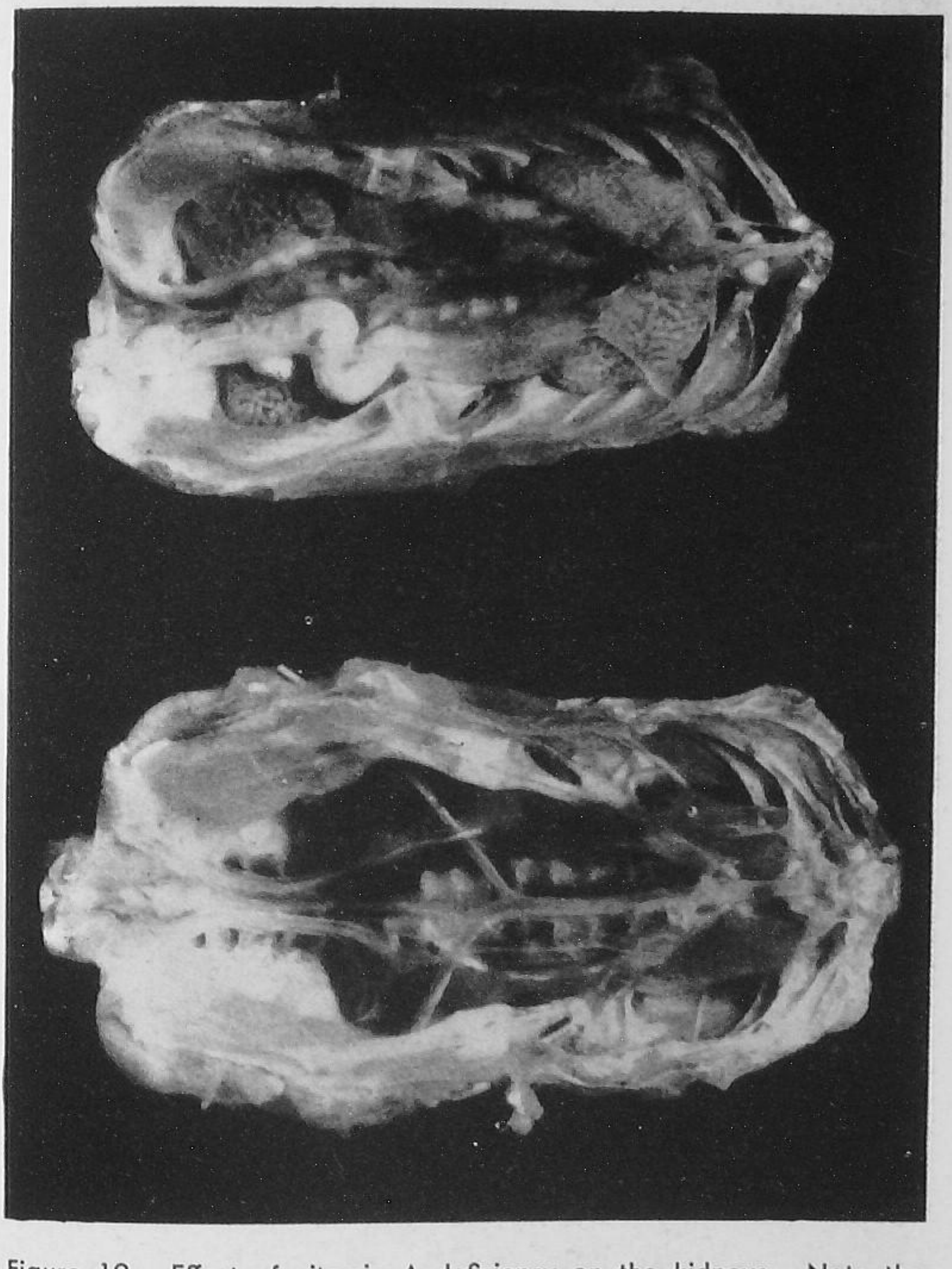


Figure 19. Effect of vitamin A deficiency on the kidneys. Note the whitish ureate deposits in the kidneys and the enlarged ureter of the top specimen in contrast to the normal at the bottom.

Inasmuch as vitamin D is concerned in calcium and phosphorus absorption and retention, a deficiency of either of these mineral elements may cause symptoms somewhat similar to those described for vitamin D. Grossly the symptom may not be distinguishable one from another, except that the legs appear normal in case of a phosphorus deficiency. Under practical conditions, however, vitamin D is the factor usually lacking, because a deficiency of calcium or phosphorus hardly ever becomes so acute as to bring about these symptoms.

In mature laying birds the first symptom of a vitamin D deficiency is the laying of thin-shelled eggs, followed very shortly by decreased egg production. The breast bone becomes soft and rubbery and the bones of the legs and wings become fragile and easily broken. Birds may temporarily lose the use of their legs and squat in a "penguinlike" manner, a symptom sometimes called "egg paralysis." Hatchability is markedly reduced.

The symptoms of a vitamin D deficiency in turkeys are very similar to those described for chickens.

Figure 20. Alpha tocopherol deficiency in a young chick.

Figure 21. A vitamin D-deficient chick showing ungainly manner of balancing body. Its beak is soft and rubbery.

Vitamin E—Alpha-Tocopherol

A lack of vitamin E in the ration of growing chicks results in the condition known as nutritionai encephalomalacia. Chicks afflicted with this deficiency disease suddenly become prostrated, lying with legs outstretched and spastic, with toes flexed. The head is retracted and often twisted laterally (Figure 20). Before the chicken becomes completely prostrated its gait and other movements are often incoordinated. Upon autopsy, lesions are found in the cerebellum and sometimes in the cerebrum. In many chicks necrotic reddish or brownish areas on the surface of the cerebellum can be seen by inspection. Under some conditions vitamin E deficiency results in subcutaneous edema and edema of the heart and pericardium.

In mature fowls a prolonged vitamin E deficiency results in sterility in the male and reproductive failure in the female. Degenerative changes in the testes of the male may occur, resulting in permanent sterility. In females, egg production apparently is not affected by a vitamin E deficiency, but hatchability is greatly reduced. During incubation the rate of growth and differentiation are slow, and many embryos die during the first

two days of development because of a circulatory failure. A definite critical period in the development of the embryo occurs about the fourth day.

In poults a deficiency of vitamin E results in the condition known as nutritional myopathy. This condition is characterized by lesions in the muscular wall of the gizzard. These lesions appear as circumscribed gray areas, which often are of firmer texture than normal muscle, and, in some cases, suggest scar tissue.

Vitamin K

A lack of vitamin K greatly delays the clotting time of the blood, and chicks fed a deficient ration may bleed to death from any injury or bruise that causes rupture of blood vessels. Hemorrhages may occur subcutaneously, intramuscularly, intraperitoneally, or in any part of the chick's body. The hemorrhages vary in size and appear to be the only symptom of the deficiency (Figure 22).

In mature birds vitamin K may be synthesized to some extent, as they do not seem to be subject to the acute deficiency. It has been shown, however, that laying birds fed a low vitamin K diet produce eggs low in vitamin K. When these eggs are incubated, chicks are hatched which have very low reserves of vitamin K with an accompanying prolonged blood-clotting time and they may bleed to death from an injury such as may result from wingbanding.

Vitamin B_1—Thiamin

Day-old chicks, when placed on a low thiamin ration, develop polyneuritis within nine to twelve days. In the acute stage of polyneuritis the head may be drawn over the back (Figure 24). Diets containing suboptimal amounts of thiamin, when fed to chicks, lead to loss of appetite, emaciation, impairment of digestion, general weakness, and frequently convulsions.

The symptoms of a thiamin deficiency in mature birds and turkeys are similar to those described for chicks.

Riboflavin

A lack of riboflavin in the diet of young chicks results in diarrhea, retarded growth, and paralysis of the legs, sometimes called nutritional leg paralysis. It occurs in a preliminary stage, which is curable, and in an acute stage, which is incurable. Nutritional paralysis is characterized by the

Figure 22. Generalized hemorrhage in a young chick caused by vitamin K deficiency.

sudden appearance of chicks walking on their hocks, with toes curling inward; otherwise, the chicks appear to be in excellent health (Figure 23). Chicks receiving rations only partly deficient in this factor often recover spontaneously. Severe cases of the paralysis show very marked hypertrophy and softening of the brachial and sciatic nerves, usually discernible by inspection. The symptoms are most pronounced and most often observed in the sciatic nerve. The nerves occasionally reach a diameter of four to five times the normal size.

In breeding birds a deficiency of riboflavin results in poor hatchability. The requirement for hatchability is considerably higher than that for egg production and maintenance of health. The embryos that fail to hatch as a result of a riboflavin deficiency are dwarfed, show a high incidence of edema, degeneration of the Wolffian bodies, and a characteristically defective down development, termed clubbed down. On a ration moderately deficient in riboflavin, many embryos die during the second week of incubation. The mortality reaches a peak at about the eleventh day of development.

Pantothenic Acid

A pantothenic acid deficiency in young chicks results in retarded growth, and feather development is extremely ragged. Within twelve to fourteen days a pellagra-like syndrome develops. The eyelids bcome granular and stick together as a result of a viscous exudate. Crusty scabs appear at the corners of the mouth and around the vent (Figure 26). Dermatitis of the feet sometimes is observed in pantothenic acid deficiency, though the lesions are seldom as severe as those brought about by a biotin deficiency (Figure 27). Liver damage and changes in the spinal cord may be seen on post-mortem examination.

Lesions in adult fowl similar to those of growing chicks have not been observed, although a deficiency of pantothenic acid results in lowered hatchability.

Nicotinic Acid (Niacin)

A deficiency of nicotinic acid in the diet of chicks results in "black tongue," a condition characterized by inflammation of the tongue and mouth cavity. Beginning at about two weeks of age the entire mouth cavity of the deficient chicks as well as the upper part of the esophagus becomes distinctly inflamed with a deep red color in contrast to the normal

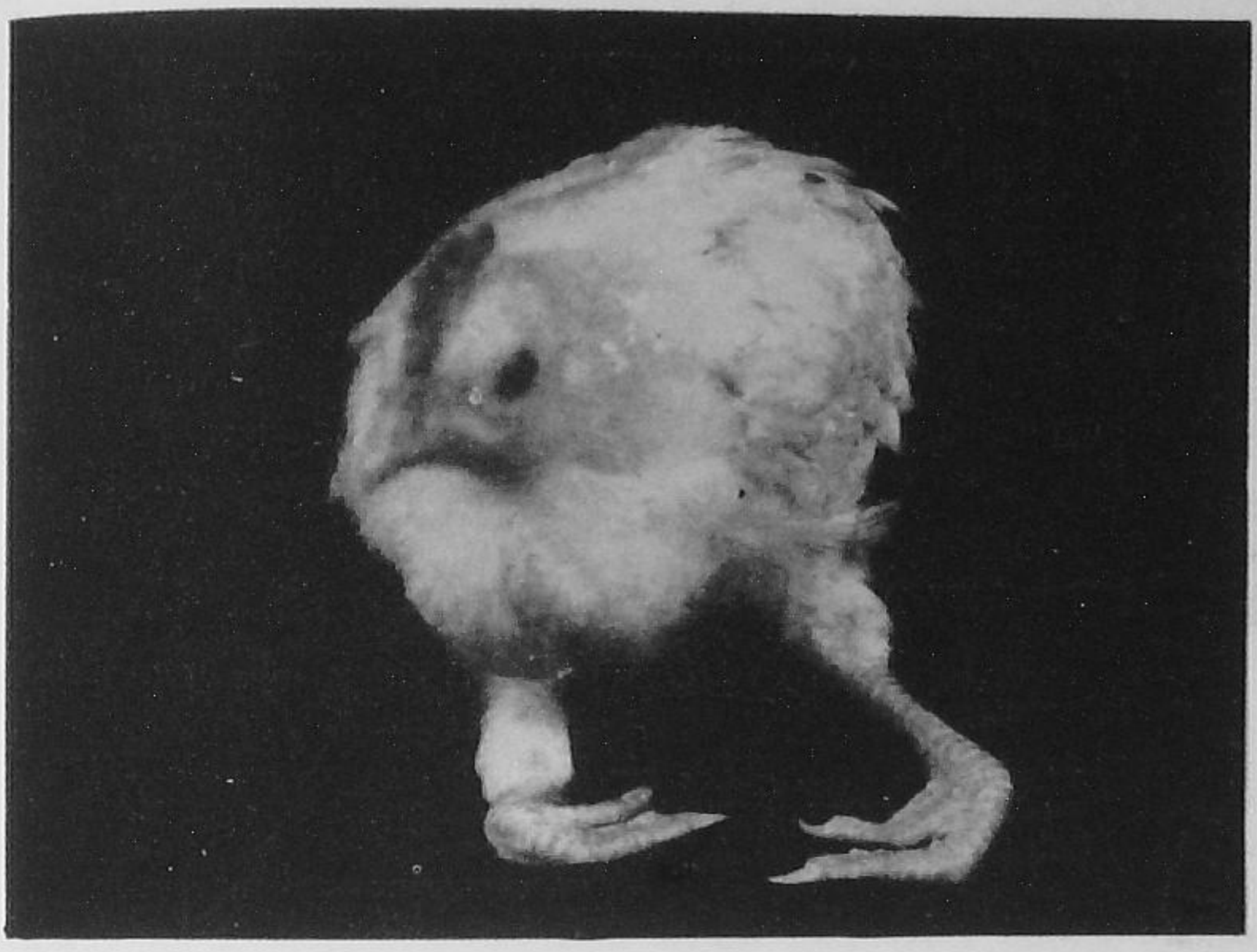


Figure 23. Riboflavin deficiency in a young chick, causing curled toes and a tendency to squat on the hocks.

Figure 24. Head retraction caused by a deficiency of thiamin.

Figure 25. Effect of nicotinic acid deficiency on chick growth. The chick on the left had ample nicotinic acid; that on the right had none.

pink of the healthy chick. Growth is retarded and feed consumption reduced (Figure 25). Poor feather development and, occasionally, scaly dermatitis of the feet and skin are also observed.

Figure 26. Advanced stage of pantothenic acid deficiency. Note the lesions at the corners of the mouth, on the eyelids and feet.

Nicotinic acid has not been shown to be essential for adult fowl.

Pyridoxine (Vitamin B_6)

Chicks fed a pyridoxine-deficient diet show a small initial gain, then cease to grow, or grow very slowly. Some chicks show abnormal excitability and, somewhat later, jerky convulsive movements. Chicks suddenly may run about aimlessly, often flopping their wings and keeping their heads down. Later, convulsions occur. During these convulsions the chick may rest on its breast, raise its feet off the ground and flop its wings. Chicks may fall on their sides or roll over on their backs and rapidly paddle their feet. The head often jerks up and down or retracts as in polyneuritis and sometimes moves convulsively in an up-and-down movement with the neck distended or twisted. Complete exhaustion follows one of these convulsions and is frequently fatal.

Pyridoxine deficiency in mature birds is characterized by loss of appetite, followed by rapid loss of weight and death. Egg production and hatchability are markedly reduced.

Choline

A lack of choline in the diet of young chickens and turkeys results in retarded growth and perosis. (*See Manganese* and Figure 28.)

Choline deficiency in mature birds has been reported to result in increased mortality and lowered egg production with an increased abortion of egg yolks from the ovaries.

Biotin

Biotin deficiency in chickens results in a dermatitis somewhat similar to that observed with pantothenic acid deficiency.

Lesions first appear in about three weeks, although considerable variation in time of appearance has been noted. The bottoms of the feet become rough and calloused and may be severely affected before mandibular lesions are apparent. As the syndrome progresses the entire bottom of the foot becomes encrusted and hemorrhagic cracks appear. The toes may become necrotic and slough off, but the top of the foot and leg usually show only a dry scaliness (Figure 27). The mandibular lesions which first appear in the corners of the mouth spread to include the area around the beak, and the eyelids eventually become swollen and stick together.

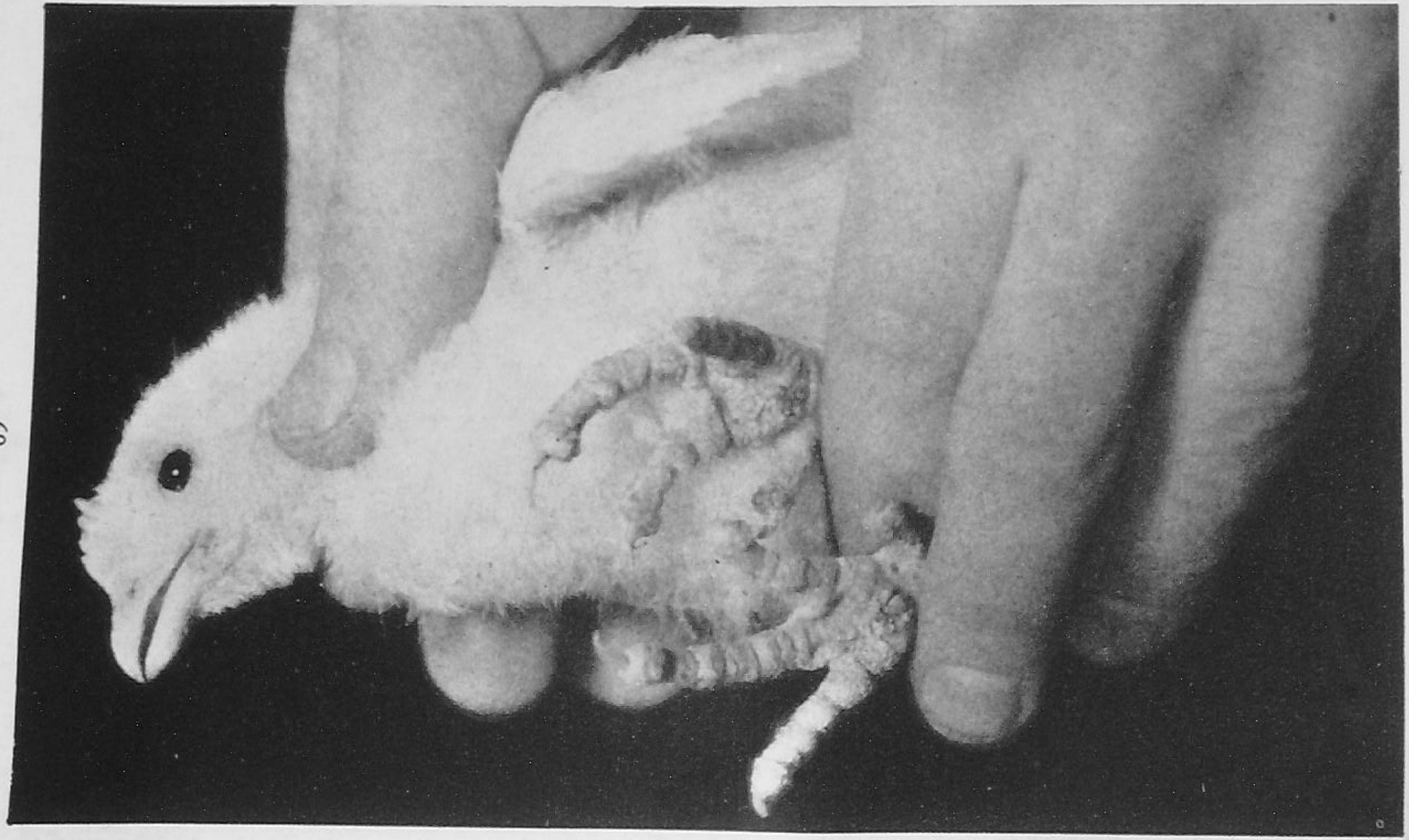


Figure 27. Biotin deficiency. Note the severe lesions on the bottom of the feet and the lesions at the corner of the mouth.

In contradistinction to these symptoms, the lesions in pantothenic acid deficiency are first evident in the corners of the mouth and eyes, and only in extreme cases do the lesions of the feet become so severe.

Biotin has been reported to be one factor necessary for the prevention of perosis in chicks and turkeys (Figure 28). Turkey poults exhibit symptoms very similar to those described for chicks when fed a biotin-deficient ration.

Feeding mature fowl a biotin-deficient ration causes reduced hatchability, but egg production is not adversely affected. This indicates that the requirement for the production of hatching eggs is much higher than that necessary for maintenance of good health and egg production. In hens, no dermatitis has been observed similar to that of chicks fed biotin-deficient rations.

Calcium and Phosphorus

Calcium, phosphorus, and vitamin D are closely interrelated in bone formation. A deficiency of any one of these results in rickets, although the blood picture may vary, depending on the factor that is lacking. Retarded growth and increased mortality are also symptoms of calcium and phosphorus deficiency.

Manganese

A manganese deficiency in the diet of growing chicks and poults results in perosis or slipped tendon (Figure 28). As has been mentioned, perosis may be caused also by a deficiency of choline or biotin.

Perosis is a malformation of the bones of chicks. The symptoms usually observed are swelling and flattening of the hock joint with subsequent slipping of the Achilles tendon from its condyles. The tibia and tarso-metatarsus may exhibit bending near the hock joint, and lateral rotation. One or both legs may be affected. A shortening and thickening of the long bones of the wings and legs is also observed. The disorder, insofar as manganese is concerned, is aggravated by excessive quantities of calcium and phosphorus in the ration.

In laying and breeding birds a manganese deficiency results in lowered egg production, eggshell strength, and hatchability. Numerous embryos that die as a result of manganese deficiency exhibit chondro-dystrophy, a condition characterized by a parrot-like beak, wiry down, and shortening of the long bones. This condition is not, however, specific for a manganese deficiency.

Magnesium

When fed a diet deficient in magnesium, chicks grow slowly for about one week, then cease growing and become lethargic. When disturbed, they exhibit symptoms of hyperirritability similar to those of other species fed diets deficient in magnesium. Chicks show a brief convulsion, then go into a comatose state which sometimes terminates fatally but usually ceases in a few minutes.

Iodine

A deficiency of iodine in the chick's diet results in goiter. The thyroid gland increases to many times the normal size. Histological examination

Figure 28. Perosis or slipped tendon resulting from a deficiency of manganese. A deficiency of choline or biotin may also result in perosis.

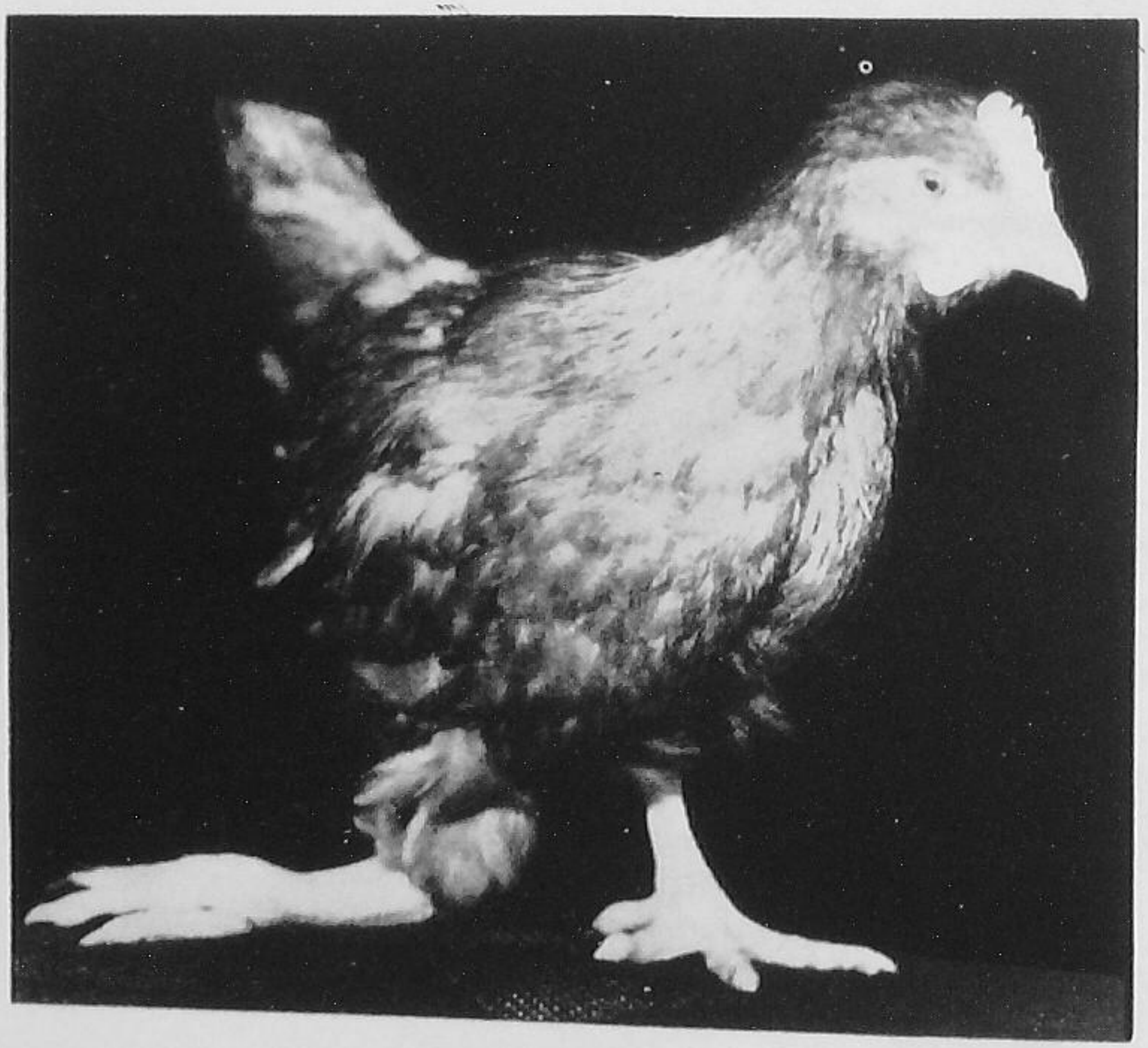


TABLE 6—Recommended Nutrient Allowances for Chickens

	Total Protein	VITAMINS									MINERALS				
		Vitamin A Activity [1]	Vitamin D	Thiamin	Ribo-flavin	Nico-tinic acid	Panto-thenic acid	Pyro-doxine	Bio-tin	Cho-line	Cal-cium	Phos-phorus [2]	Salt [3]	Mang-anese	Iodine
	Percent	I.U. per kg.	A.O.A.C. Units per kg.	Mg. per kg.							Percent			Mg. per kg.	
Starting Chicks, 0–8 weeks	20	4400	397	2.0	3.5	17.6	11.0	3.5	0.099	1,540	1.00	0.60	0.5	55	1.1
Growing chicks, 8–18 weeks	16	4400	397	?	2.0	?	?	?	?	?	1.00	0.60	0.5	?	1.1
Laying Hens	15	7300	992	?	2.0	?	5.5	3.5	?	?	[4] 2.25	0.75	0.5	?	1.1
Breeding Hens	15	7300	992	?	2.9	?	11.0	3.5	0.154	?	[4] 2.25	0.75	0.5	33	1.1

[1] May be fish-oil vitamin A or provitamin A from vegetable sources.
[2] Inorganic phosphorus should constitute 0.2 percent of the total feed.
[3] This figure represents added salt or sodium chloride.
[4] This amount of calcium need not be incorporated in the mixed feed inasmuch as calcium supplements fed free choice are considered as part of the ration.

of the enlarged thyroid glands show an absence of colloid and a hyperplasia of the living cells of the follicle.

Iron and Copper

A deficiency of iron or copper in poultry rations results in anemia. Recently pyracin and vitamin B_c have been reported as being essential for the prevention of anemia in growing chicks.

TABLE 7

TENTATIVE REQUIREMENTS OF CHICKS (0-8 WKS.) FOR CERTAIN AMINO ACIDS, VITAMINS, AND MINERALS [1]

Amino Acids:	
Glycine, percent	1.0
Arginine, percent	1.0
Methionine, percent or	0.9
(Methionine, percent	0.5
(Cystine, percent	0.4
Lysine. percent	0.9
Tryptophane, percent	0.25
Vitamins:	
Vitamin K, Mg./kg.	0.39
Minerals:	
Potassium, Gm./kg.	1.8
Magnesium, Mg./kg.	397.0
Iron, Mg./kg.	19.8
Copper, Mg./kg.	2.0

[1] Minimum quantities needed; do not include margin of safety.

NOTE: Numerous other factors aside from those given in Table 6, on recommended dietary allowances, have been shown to be essential for poultry. Because of the limited information on the actual requirements of poultry for these factors, or the limited knowledge of their distribution in feedstuffs, or both, these have not been included in the table of recommended allowances. Among these factors are certain of the essential amino acids, vitamin K, and several of the mineral elements. The available information on the requirements of growing chicks for these factors is given above.

Selected References

ALMQUIST, H. J., and E. MECCHI. 1939. Vitamin A requirements of laying hens. Poultry Sci. *18*:129-137.

CRAVENS, W. W., H. J. ALMQUIST, L. C. NORRIS, R. M. BETHKE, and H. W. TITUS. 1944. Report No. 1: Recommended nutrient allowances for poultry. National Research Council, U. S. A., Committee on Animal Nutrition.

BRIGGS, G. M. JR., T. D. LUCKEY, L. J. TEPLEY, C. A. ELVENJEM, and E. B. HART. 1943. Studies on nicotinic acid deficiency in the chick. Jour. Biol. Chem. *148*:517-522.

DAVIS, H. J., L. C. NORRIS, and G. F. HEUSER. 1938. Further evidence on the amount of vitamin G required for reproduction in poultry. Poultry Sci. *17*:87-93.

HEUSER, G. F. 1941. Protein in poultry nutrition. A review. Poultry Sci. *20*:362-368.

TABLE 8. RECOMMENDED NUTRIENT ALLOWANCES FOR TURKEYS

	Total Protein	VITAMINS				MINERALS			
		Vitamin A Activity [1]	Vitamin D [2]	Riboflavin	Choline	Calcium	Phosphorus [3]	Salt [4]	Manganese
	Percent	I. U. per kg.	A.O.A.C. Units per kg.	—Mg. per kg.—		——Percent——			Mg. per kg.
Starting Poults, 0–8 weeks	24	8800	1760	4.4	1980	2.00	1.00	0.5	55
Growing Turkeys 8–16 weeks	[5] 20	8800	1760	?	?	2.00	1.00	0.5	?
Turkey Breeders	15	8800	1760	3.5	?	[6] 2.25	0.75	0.5	33

[1] May be either fish oil vitamin A or provitamin A from vegetable source.

[2] This allowance should prove adequate for vitamin D from either fish oil or irradiated animal sterols when the ration contains the recommended allowances for calcium and phosphorus and when the minimum amount of inorganic phosphorus suggested in Footnote 3 is present in the ration. If the ration contains materially less calcium and phosphorus, it is necessary to increase the amount of vitamin D when it is obtained from fish oil.

[3] Inorganic phosphorus should constitute 0.4 percent of the total feed.

[4] This figure represents added salt or sodium chloride.

[5] The protein content of rations for growing turkeys from 16 weeks to market weight may be reduced to 16 percent.

[6] This amount of calcium need not be incorporated in the mixed feed inasmuch as calcium supplements fed free choice are considered as part of the ration.

JUKES, T. H., and H. J. ALMQUIST 1942. Avian biochemistry. Ann. Rev. of Biochem. *11:*511-530.

JULL, MORLEY ALLAN. 1938. *Poultry Husbandry.* 2nd ed. New York & London: McGraw-Hill Book Co. Pp. 548.

LIPPINCOTT, WILLIAM ADAM. 1946. *Poultry Production.* 7th ed. Philadelphia: Lea & Febiger. Pp. 440.

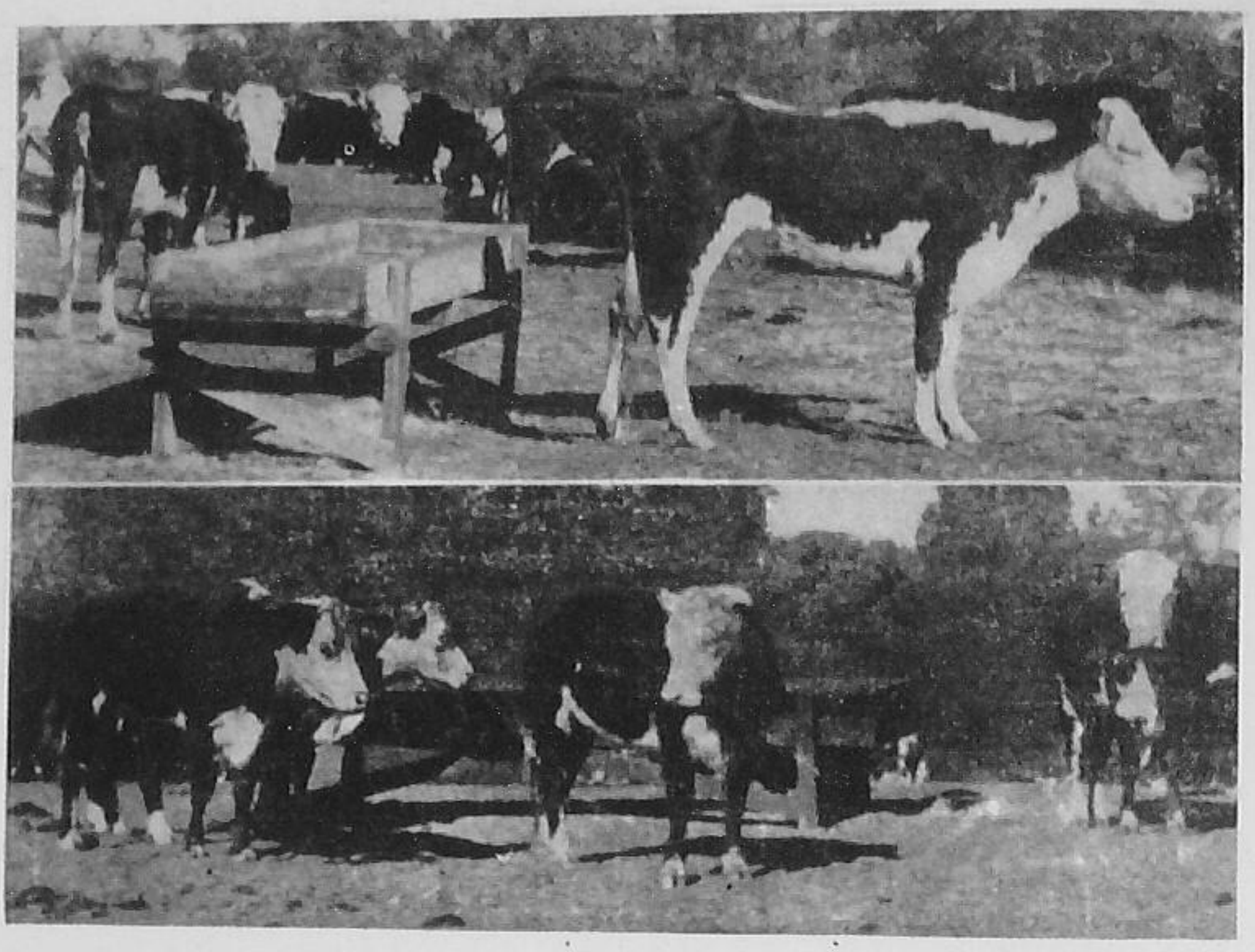
RUBIN, MAX, and H. R. BIRD. 1942. Relation of vitamin A to egg production and hatchability. Md. Agr. Expt. Tech. Bul. A 12.

———, H. R. BIRD, and H. M. DEVOLT. 1941. Avitaminosis A in commercial poultry flocks. Poultry Sci. *20:*155-160.

SCHAIBLE, P. J. 1941. The minerals in poultry nutrition. A review. Poultry Sci. *20:*278-287.

TITUS, HARRY W. 1939. Practical nutrition requirements of poultry. U. S. D. A. Yearbook of Agriculture, *Food and Life,* pp. 787-818. Practical feeding of poultry. U. S. D. A. Yearbook of Agri., *Food and Life,* pp. 819-843.

———, 1942. Nutritional diseases of poultry. U. S. D. A. Yearbook of Agri., *Keeping Livestock Healthy,* pp. 1075-1107.



Figures 29 and 30. Effect of protein deficiency and low energy intake on range cattle. The cows in the upper picture lost weight on dried range forage deficient in protein and low in phosphorus, were thin and weak after calving. Their calves weighed 175 kilograms at weaning time and they produced only a 61 percent cattle crop the following year. The cows in the lower picture were on the same range but were fed sufficient cottonseed cake to meet their protein requirements. Barley was added as a source of additional energy after calving until new forage was available. Their calves weighed 219 kilograms at weaning time and they produced a 91 percent calf crop the following year.

9. Symptoms of Nutritional Deficiencies in Beef Cattle

In some cases the symptoms are specific, but such conditions as reduced appetite or growth, rough hair coat, and general unthriftiness are common to most states of malnutrition. Since nutritional deficiencies may range from very mild to severe, they may exist without gross functional or anatomical alterations. Acute symptoms, though frequently modified from laboratory cases by the complication of unknown variables encountered in the field, are often dramatic and focus special attention on the problems. It is, however, the more insidious mild deficiencies resulting in suboptimal performance that are most difficult to diagnose and are commonly the source of greatest economic loss.

Energy Intake

Lack of sufficient total feed is probably the most common deficiency in beef cattle. In limited feeding on farms or overstocked ranges, low energy intake may be the sole deficiency, the results being slowing or cessation of growth (including skeletal growth), loss of weight, reproduction failure, and increased mortality. On ranges, low feed intake also commonly results in increased mortality from toxic plants and from lowered resistance to parasites and diseases. Very commonly, however, underfeeding is complicated by shortages of protein and other nutrients (Figures 29 and 31).

Protein

Shortage of protein is the second most common deficiency in beef cattle. It results in poor growth, depressed appetite, failure of milk secretion and of estrus, and rapid loss of weight.

Less than 8 percent of total crude protein in the dry matter of dry range forage, or in poor roughage low in digestibility, is deficient for all classes of cattle. Range forage often becomes lower than 5 percent in crude protein, and such feed is usually also deficient in phosphorus. To alleviate deficiency under these conditions and to promote efficient range

use, it is advisable to feed protein supplements in sufficient amount to approximate the allowances recommended in Table 9. Phosphorus requirements are usually met when sufficient protein supplement is supplied (Figures 30 and 32).

Salt (NaCl)

Salt deficiency is manifested by intense craving for salt, lack of appetite, and unthrifty appearance. In heavily producing milk cows the result may be collapse and sudden death. Cattle should have free access to salt.

Phosphorus

Areas of phosphorus deficiency in cattle feeds are widespread throughout the world, particularly in semiarid regions, and are commonly associated with soils deficient in the element. Phosphorus content generally decreases markedly when plants are fully mature; this decrease, along with protein deficiency, commonly occurs when cattle must subsist for long periods on mature, nonleguminous, dried grasses and herbage (Figure 35).

Cattle should be allowed free access to a phosphorus-rich mineral mixture if the phosphorus is apt to fall much below 0.15 percent of the dry matter, particularly when the protein content of such forage is not a serious limiting factor.

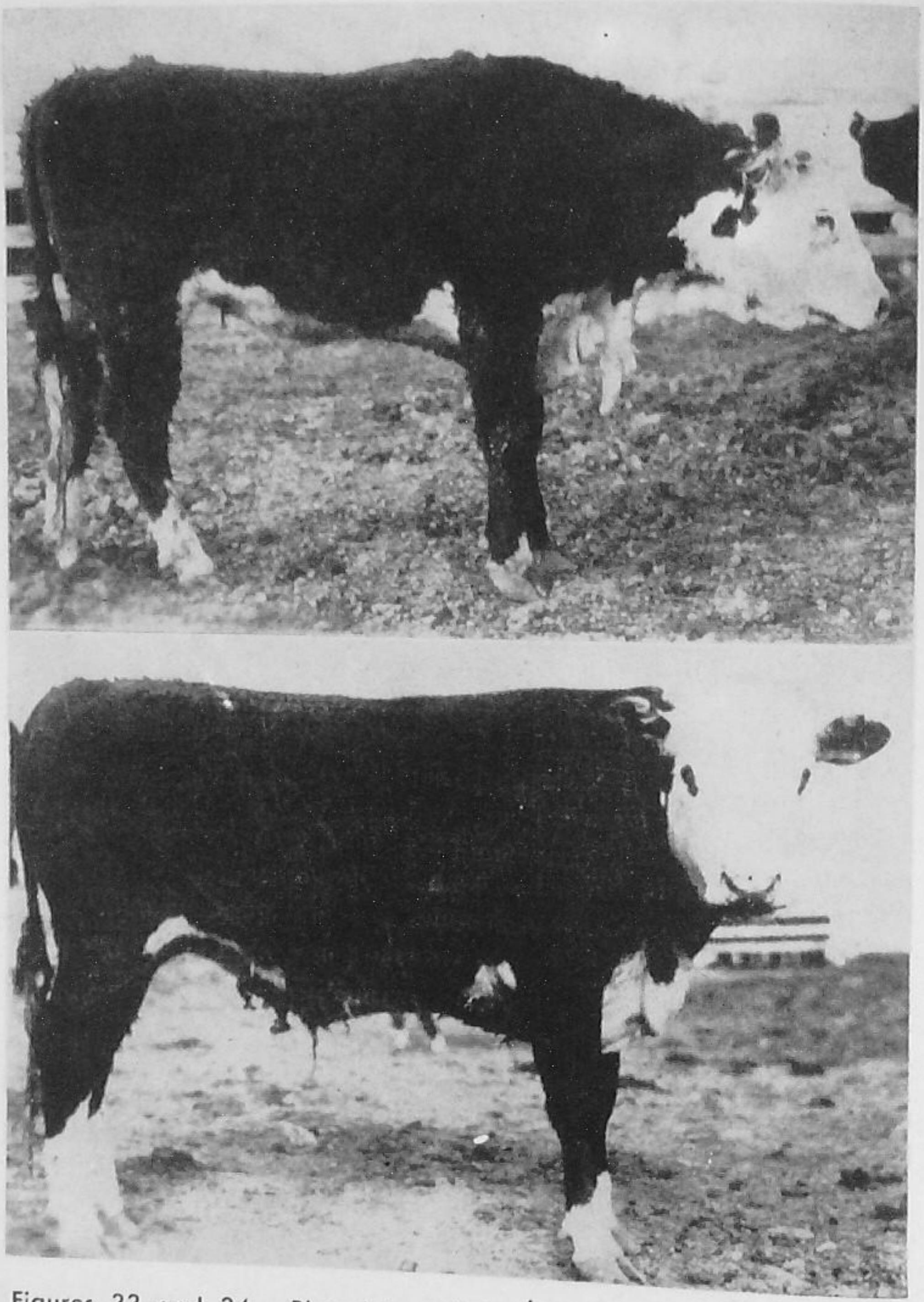
The earliest symptoms of phosphorus deficiency are decrease in blood phosphorus, in appetite, and in rate of gain. Milk production falls off. Efficiency of feed utilization, particularly of protein, is depressed. These effects are followed by pica, with specific craving for bones. Depraved appetite may lead to excessive salt ingestion, and, in the absence of bones, to the chewing of stones and wood and the eating of dirt. Carcass debris, if available, may be consumed. The result may be a secondary disease, characterized by paralytic symptoms. It is called "loin disease" in Texas, U. S. A., and *lamsiekte* in South Africa. The trouble is caused by a toxin of *C. botulinus* type organisms ingested with the putrid flesh. Long-continued phosphorus privation results in bone changes, lameness, and stiffness of joints. Bone fractures may occur. Low-phosphorus rickets in young animals, osteomalacia, osteoporosis, and ostitis fibrosa in adults are generally descriptive of the bone alterations.

Effects of a phosphorus-deficient feedlot ration are shown in Figure 33.

Figures 31 and 32. Effect of protein deficiency on the growth and development of weaning calves from the cows shown in Figures 29 and 30. They were weaned in July and turned onto mature dried forage. The group in the upper picture not only were lighter in weight at weaning but subsequently lost additional weight. The lower group, fed cottonseed cake to supply protein, phosphorus, and additional energy, gained weight on the nutritionally deficient forage and developed normally. Photos taken in December.

Calcium

Calcium deficiency in beef cattle is comparatively rare and mild, and the symptoms are inconspicuous. When fattening calves are fed heavily on concentrates, with limited quantities of nonlegume roughage, their calcium intake is insufficient for optimum gain and bone development. Dried mature range forage, if predominantly grasses, may contain less than the required minimum quantities; cereal straws are also usually defi-



Figures 33 and 34. Phosphorus deficiency in feedlot cattle. The steer in the upper picture was fed a ration consisting of wet beet pulp, alfalfa hay, and beet molasses containing 0.12 percent phosphorus. The steer shown in the lower picture received the same ration plus 45 grams daily of steamed bone-meal which brought the phosphorus content up to 0.18 percent and provided an average total intake of 17.0 grams of phosphorus daily.

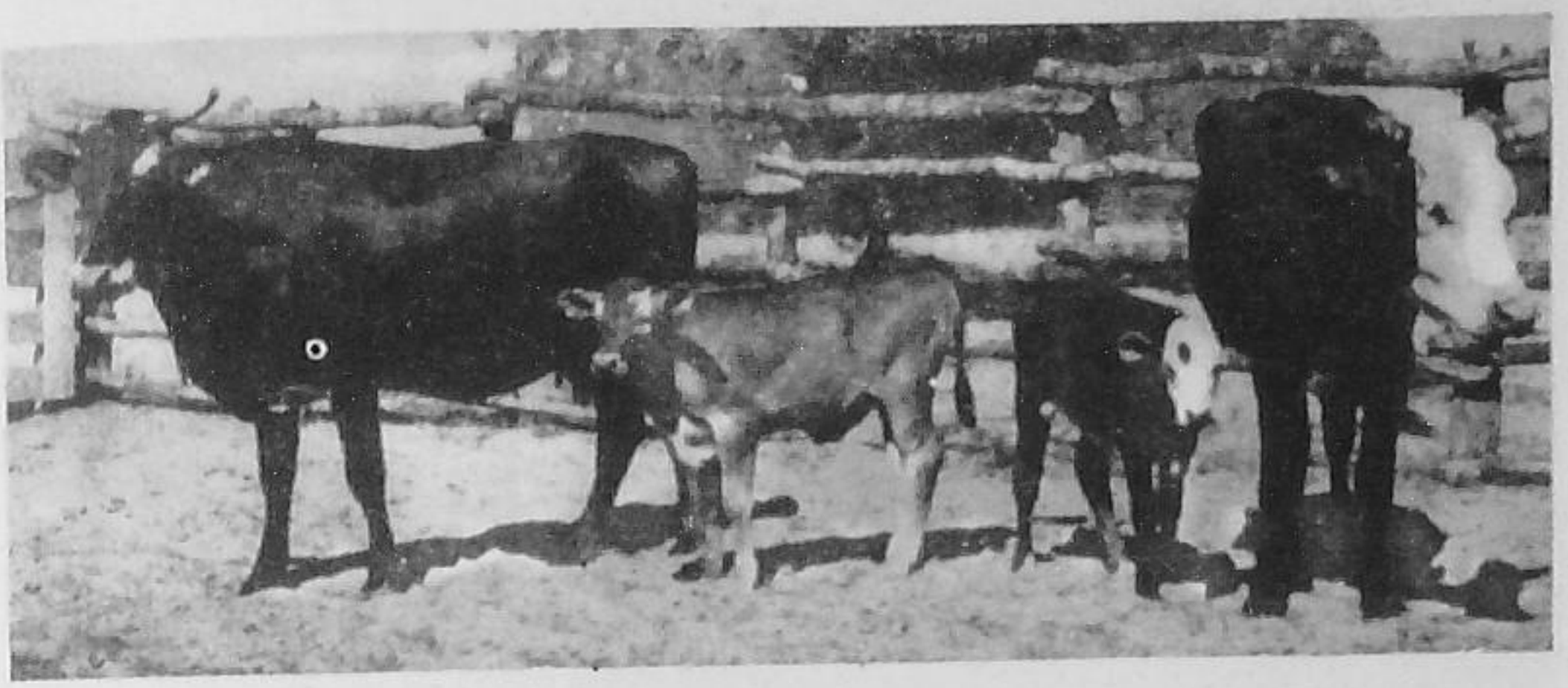


Figure 35. The cow on the left received a phosphorus supplement while grazing on a phosphorus-deficient range for 2½ years; the cow on the right received none. Note the more thrifty appearance of cow and calf that received the phosphorus supplement.

cient in calcium. Severe privation may so deplete the bones of calcium and phosphorus that fractures occur. The addition of calcium to a deficient ration for fattening calves increases the rate of gain, improves feed utilization, results in heavier bones with higher ash content and greater breaking strength, and enhances the market grade.

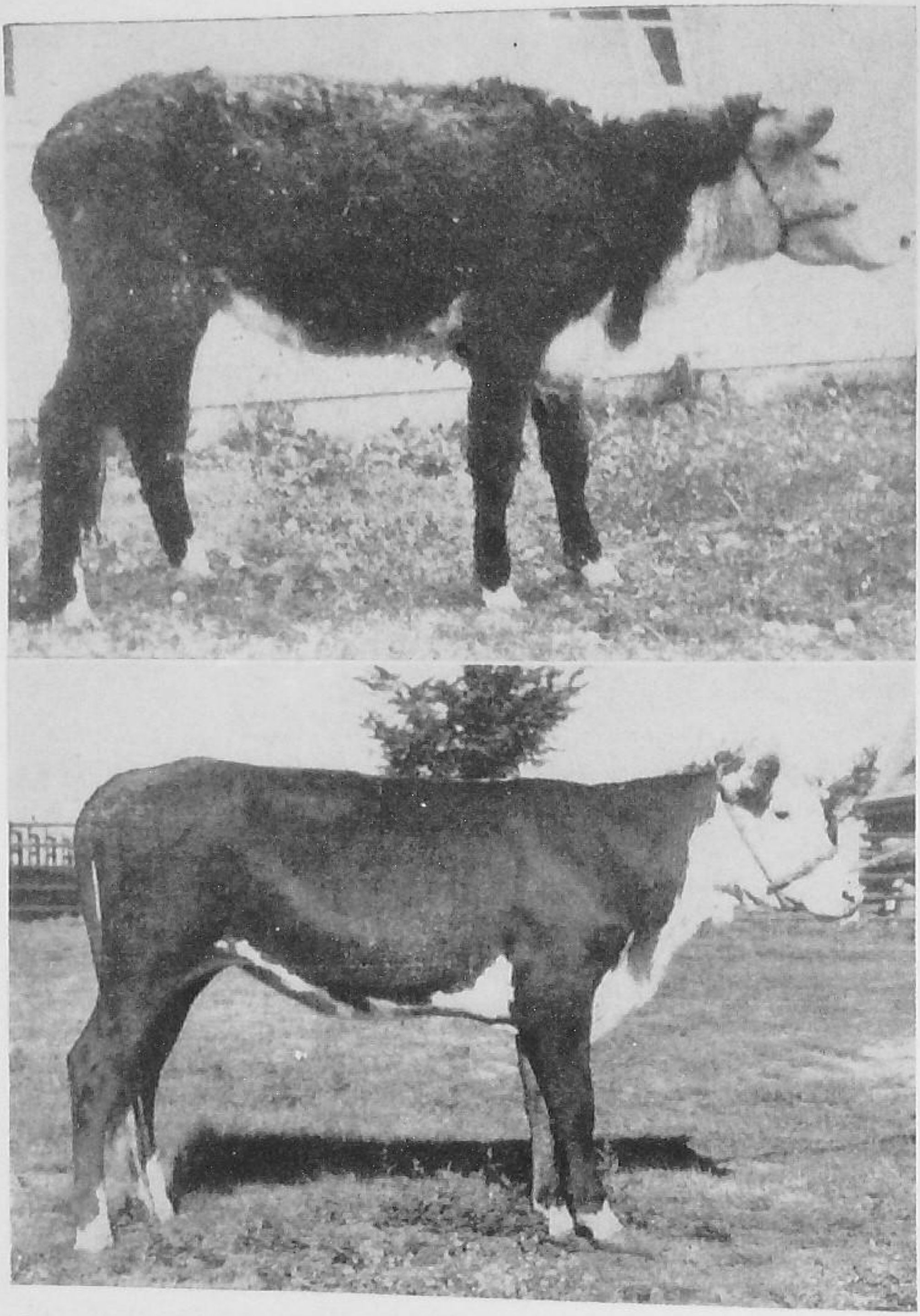
Iodine

This deficiency usually is manifested by the production of dead or nonviable goitrous calves. Occasional borderline cases may survive; in these, the moderate thyroid enlargement disappears in a few weeks.

Iodine requirements have not been definitely established. The use of salt containing 0.015 to 0.02 percent potassium iodide has effectively prevented goiter in iodine-deficient areas. Where iodine supplementation is indicated, iodized salt containing 0.01 percent potassium iodide (0.0076 percent iodine), stabilized to prevent loss of iodine, has proved satisfactory.

Iron

Uncomplicated iron deficiency has not been satisfactorily demonstrated under natural conditions; the usual complication is a shortage of copper, cobalt, or both. Iron deficiency results in anemia. Although the iron requirement of cattle is unknown, apparently the amount in ordinary feeds is ample.



Figures 36 and 37. Cobalt deficiency. The upper picture shows a heifer suffering from anemia, lack of appetite and exhibiting the characteristic roughness of coat. Administration of cobalt to the same heifer brought about a remarkable recovery of appetite and disappearance of symptoms, as shown in the lower picture.

Copper

A lack of sufficient copper in forage has been reported in different parts of the world. The copper-deficiency syndrome involves loss of condition, stunted growth, rough coat, and anemia. Depraved appetite and intermittent diarrhea are common. Estrus is suppressed. Young calves may have straight pasterns and may tend to stand on their toes. Without premonitory evidence, animals may suddenly fall dead after little or no struggle; hence the term "falling disease" in Australia. Post-mortem reveals congestion of abomasum and intestines, very friable liver, dark pulpy spleen, and glomerulo-nephritis. Bleaching of the hair coat has also been reported. For alleviation of copper deficiency a 1-percent mixture of copper sulfate in salt has been recommended. In experiments of cattle on pastures containing 2 to 3 p.p.m. and 1 to 4 p.p.m. of copper in the dry matter manifested anorexia, anemia, emaciation, and diarrhea responded to copper therapy. Pastures containing 7.5 p.p.m. were regarded as "healthy," and 20 to 30 p.p.m. in the dry matter of the forage proved to be curative.

Cobalt

A progressive emaciation and anemia caused by cobalt deficiency is known in different parts of the world as Denmark disease, coastal disease, enzootic marasmus, bush sickness, salt sickness, Nakuritis, and pining disease. The symptoms are loss of appetite, craving for hair and wood, scaliness of skin, listlessness, and general unthrifty appearance (Figure 36). Anemia due to reduction of red cells and hemoglobin may occur relatively late. Diarrhea is often observed.

Cobalt-deficient pastures contain 0.01 to 0.07 p.p.m. of cobalt, whereas "healthy" pastures generally afford 0.07 to 0.30 p.p.m. Samples of hay from farms where cobalt deficiency was found contained 0.03 to 0.06 p.p.m.; hay from unaffected areas, 0.12 p.p.m. of cobalt. Judging from these data, the minimum cobalt requirement is met by about 0.1 p.p.m. of cobalt in the dry matter of the feed, an intake of about 1. mg. daily by adult cattle. Daily doses of 0.3 to 1.0 mg. of cobalt sufficed for cattle on deficient pasture; 0.1 mg. for sheep. The requirement therefore appears to be in the order of 0.22 mg. daily per 100 kilograms of body weight; 31 gm. of cobalt chloride or sulfate in each 100 kg. of salt alleviates cobalt deficiency.

Vitamin A

The first easily detected clinical symptom of vitamin-A deficiency is night blindness, readily observed when animals are driven about in

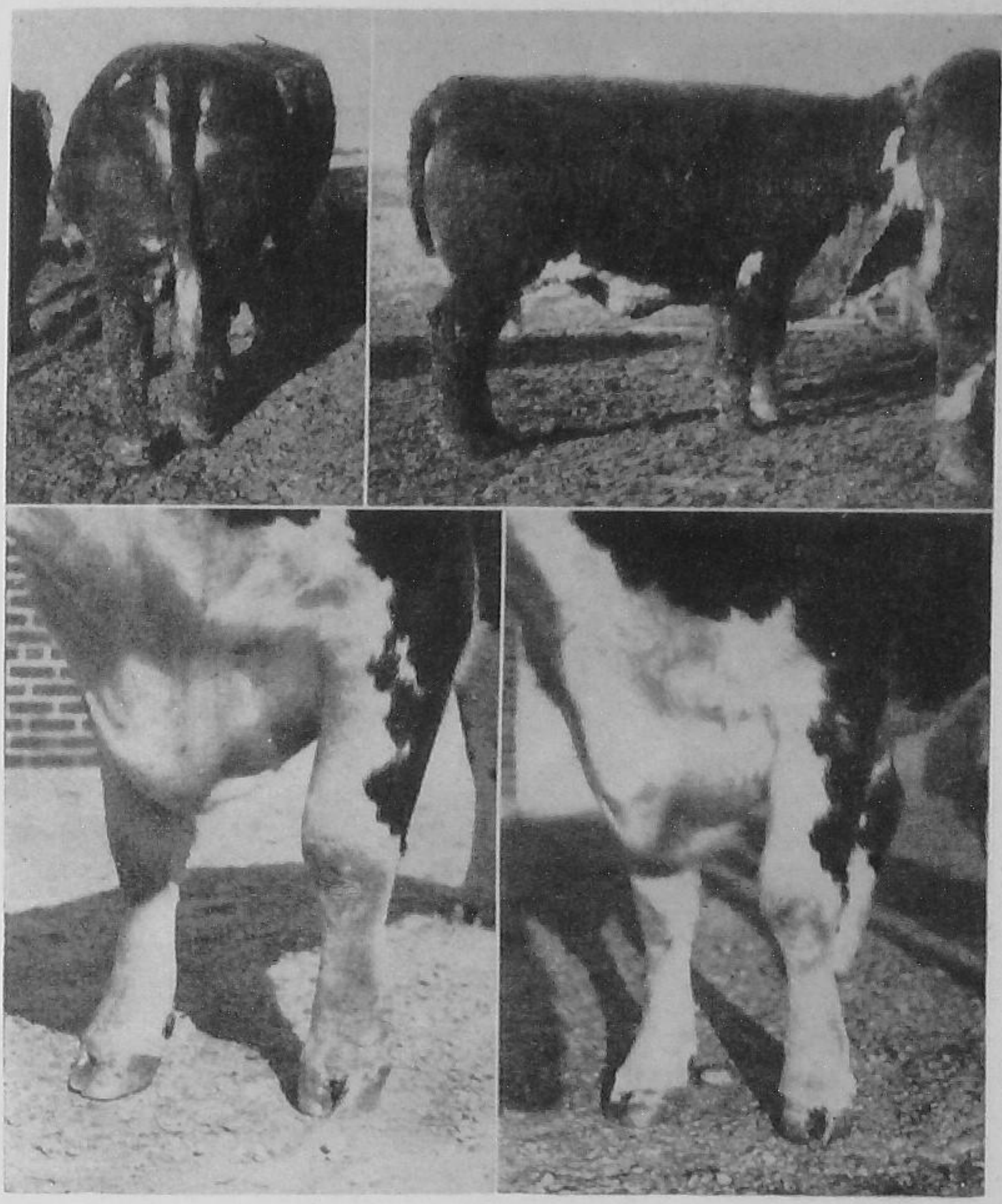
twilight, moonlight, or other dim illumination. Night blindness may be present even though the animals appear thrifty and are gaining at practically normal rates. When gross night blindness is evident, vitamin A in the blood is very low and liver reserves approach exhaustion. The next conspicuous symptoms usually developed are muscular incoordination, staggering gait, and convulsive seizures caused by elevation of the cerebrospinal fluid pressure, which also results in papillary edema. Total and permanent blindness in young animals results from stenosis of optic foramina and atrophy of optic nerves. Other localized paralyses may also occur. Excessive lachrymation rather than xerophthalmia is the rule in cattle. Unless death in convulsion or from intercurrent disease intervenes, the cornea of the eyes becomes keratinized and may, if subjected to infection, develop ulceration. Severe diarrhea in young calves and intermittent diarrhea at advanced stages of deficiency in adults are characteristic. In fattening cattle, generalized edema or anasarca may occur (Figures 38, 39, 40). In chronic severe deficiency, structural changes occur in the retina and in epithelial tissues. Degenerative changes in the kidneys and degeneration of testicular germinal epithelium have often been demonstrated.

Estrus may continue when the deficiency has advanced to the point where convulsions are common but ability to become pregnant is impaired. Deficiency in the pregnant animal results in abortion or birth at term of dead or weak calves (Figure 42).

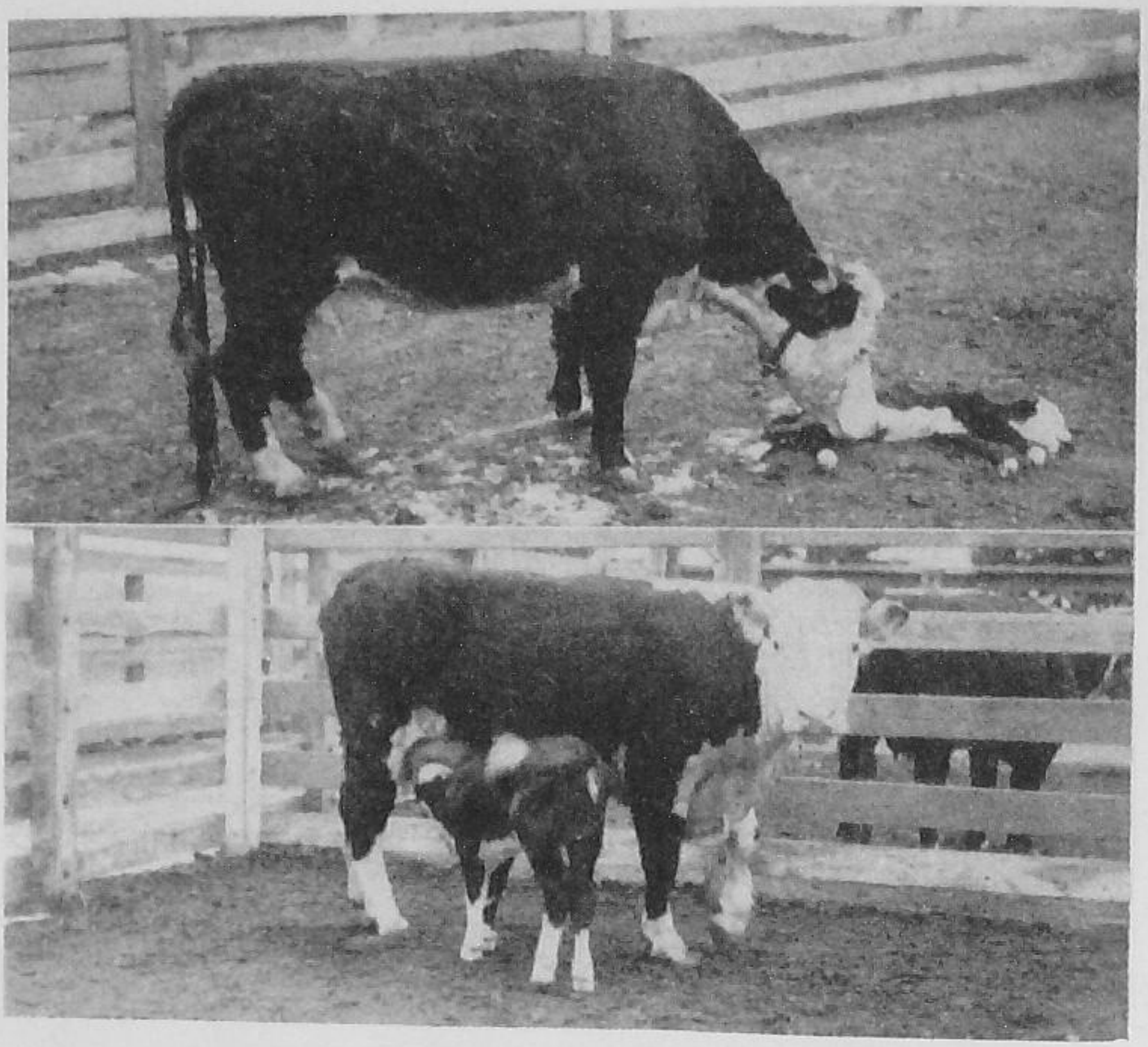
To assure production of vigorous calves, the feeding of vitamin-A supplements to breeding cows before calving should be considered if the cows have subsisted exclusively on dry bleached forage for as long as five months. Sometimes the breeding herd can be turned into green pastures. Even a few days on such forage may suffice to carry them over a critical period. The requirements set forth in Table 9 can usually be met by providing 2 to 3 kilograms daily of good green-colored leafy hay. Half a kilogram or less daily of highest-quality dehydrated alfalfa meal will suffice. Protein supplement fortified with liver oil offers another possibility. Sixty grams of liver oil containing 20,000 I. U. vitamin A per gram fed in 2 or 3 doses on grain or other feed should protect against deficiency of the vitamin for about a month.

Vitamin D

In calves, prolonged deficiency causes rickets (Figure 44) similar to that in other young animals. Clinical symptoms are usually preceded



Figures 38, 39, 40 and 41. Swelling (generalized edema, or anasarca) often develops in cattle suffering from vitamin-A deficiency. Deficiency under feedlot conditions has caused considerable loss to cattle feeders and slaughterers. In the top pictures of feedlot cattle, note swollen legs, dry coat, and edema in the abdominal region. The lower left picture shows anasarca in an experimental case; the lower right shows the disappearance of swelling following vitamin-A therapy.



Figures 42 and 43. Effect of vitamin-A deficiency on reproduction. The heifer in the upper picture received a ration deficient in vitamin A but otherwise complete; she became night blind and aborted during the last month of pregnancy. Note the retained placenta. The heifer in the lower picture received the same basal ration but during the latter part of the gestation period it was supplemented with half a kilogram daily of dehydrated alfalfa meal containing about 50 mg. of carotene. A normal vigorous calf was produced.

by a decrease in either or both blood calcium and inorganic phosphorus. This is usually followed by poor appetite, decrease in growth rate or loss in weight, digestive disturbances, stiffness of gait, and occasional convulsions. Later, enlargement of the joints, slight arching of the back, bowing of the legs, and erosion of the joint surfaces cause additional pain and difficulty in locomotion. Symptoms develop more slowly in older animals.

Figure 44. This cow developed rickets early in life when maintained on a vitamin-D deficient ration and was not allowed exposure to direct sunlight. Note the bowed front legs and enlarged joints.

Work with dairy cattle has shown that vitamin-D deficiency in the pregnant female may result in dead, weak, or deformed calves at birth.

Under usual conditions of management, beef cattle receive sufficient vitamin D from exposure to direct sunlight or from sun-cured roughages. The requirement for young calves is about 300 I. U. per 45 kilograms of live weight.

Vitamin B Complex

Thiamin, riboflavin, niacin, pyridoxine, pantothenic acid, biotin, and vitamin K are synthesized by microorganisms in the rumen. So far as is known, a dietary supply of these vitamins is not essential after cattle are two months old and rumen function has been established. Conceivably, however, acute protein or other dietary deficiencies may affect the amount of those vitamins synthesized in the rumen, by providing an unsatisfactory substrate for the organisms involved. Some preliminary evidence to this effect has been obtained

Vitamin E

The need for vitamin E in the diet of cattle has not been demonstrated, nor is there evidence of rumen synthesis of this vitamin. Claims made for beneficial results of vitamin-E therapy in reproduction in cattle have not been substantiated.

Selected References

BLACK, W. H., B. KNAPP, JR., and J. R. DOUGLAS. 1939. Nutritional requirements of beef and dual-purpose cattle. U. S. D. A. Yearbook of Agriculture, *Food and Life*, pp. 519-43.

———, 1939. Practices in the feeding of beef and dual-purpose cattle. U. S. D. A. Yearbook of Agr., *Food and Life*, pp. 544-565.

DAVIS, R. E., and L. L. MADSEN. 1941. Carotene and vitamin A in cattle blood plasma with observations on reproductive performance at restricted levels of carotene intake. Jour. Nutr. *21:*135-46.

DU TOIT, P. J., A. I. MALAN, and J. W. GROENWALD. 1934. Studies in mineral metabolism. XXXI. Minimum mineral requirements of cattle. Onderstepoort Jour. Vet. Sci. and Anim. Indus. *2:*565-606.

ELLIS, N. R., W. R. KAUFMAN, and C. O. MILLER. 1939. Composition of the principal feedstuffs used for livestock. U. S. D. A. Yearbook of Agr., pp. 1665-74.

GOSS, H. 1943. Some peculiarities of ruminant nutrition. Nutr. Abst. and Rev. *12:*531-38.

GUILBERT, H. R., and L. H. ROCHFORD. 1940. Beef production in California. Calif. Agr. Expt. Cir. *115:*1-125.

———, P. GERLAUGH, and L. L. MADSEN. 1945. Report No. 4 of the Committee on Animal Nutrition (National Research Council, U. S. A.). Recommended nutrient allowances for beef cattle.

HUFFMAN, C. F., and C. W. DUNCAN. 1944. Nutritional deficiencies of farm mammals on natural feeds. Ann. Rev. Biochem. *13:*467-86.

———, 1939. Roughage quality and quantity in the dairy ration, a review. Jour. Dairy Sci. *22:*889-980.

KLEIBER, M., H. GOSS, and H. R. GUILBERT. 1936. Phosphorus deficiency metabolism and feed utilization in beef heifers. Jour. Nutr. *12:*121-53.

MADSEN, L. L. 1942. Nutritional diseases of cattle. U. S. D. A. Yearbook of Agriculture, pp. 645-72.

———, 1942a. Nutritional diseases of farm animals. U. S. D. A. Yearbook of Agriculture, pp. 323-53.

MAYNARD, L. A. 1947. *Animal Nutrition.* 2d ed. New York: McGraw-Hill Book Co. Pp. 494.

MITCHELL, H. H., and F. J. MCCLURE. 1937. Mineral nutrition of farm animals. National Research Council Bul. 99, pp. 135.

MORRISON, F. B. 1945 (c. 1936). *Feeds and Feeding.* 20th ed. Ithaca, N. Y.: Morrison Publishing Co. Pp. 1050.

OTTO, J. S. 1938. The assimilation of calcium and phosphorus by the growing bovine. Onderstepoort Jour. Vet. Sci. and Anim. Indus. *10:*281-364.

SNAPP, R. R. 1939. *Beef Cattle.* 3rd ed. New York: John Wiley & Sons. Pp. 550.

WALLIS, G. C. 1944. Vitamin-D deficiency in dairy cows, symptoms, causes and treatment. So. Dak. Agr. Expt. Sta. Bul. 372. Pp. 1-16.

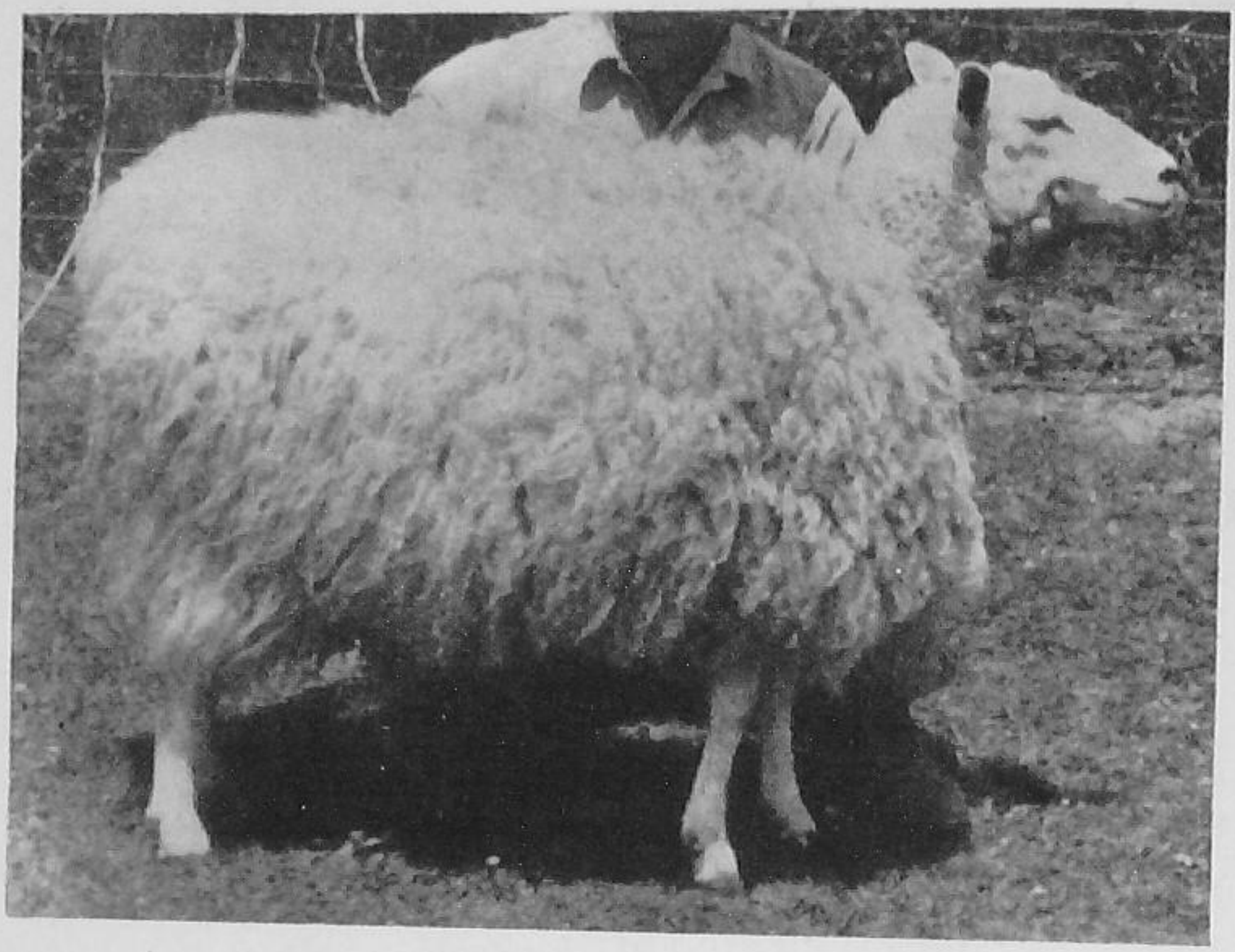
TABLE 9—RECOMMENDED NUTRIENT ALLOWANCES FOR BEEF CATTLE

Body Weight	Total Digestible Nutrients	Net Energy	Total Digestible Protein	Calcium	Phosphorus	Carotene [1]
	DAILY ALLOWANCE PER ANIMAL					
Kg.	**Kg.**	**Therms**	**Kg.**	**Gm.**	**Gm.**	**Mg.**
Normal Growth, Heifers and Steers						
181	3.2	6.3	0.41	20	15	25
272	3.9	7.5	0.41	18	15	35
362	4.3	8.3	0.41	16	15	45
453	4.8	9.0	0.41	15	15	55
Bulls, Growth and Maintenance (Moderate Activity)						
272	4.5	8.7	0.59	24	18	35
362	5.0	9.5	0.64	23	18	45
453	5.4	10.3	0.64	22	18	55
544	5.9	11.2	0.64	21	18	65
635	6.4	12.0	0.64	20	18	75
725	6.4	12.0	0.64	18	18	90
816	6.4	12.0	0.64	18	18	100
Wintering Weanling Calves						
181	2.7	5.0	0.32	16	12	25
226	3.2	5.8	0.36	16	12	30
272	3.6	6.5	0.36	16	12	35
Wintering Yearling Cattle						
272	3.6	6.4	0.36	16	12	35
317	3.9	6.8	0.36	16	12	40
362	4.1	7.2	0.36	16	12	45
408	4.1	7.2	0.36	16	12	50
Wintering Pregnant Heifers (Weights are for Beginning of Winter Period)						
317	4.5	8.0	0.41	18	16	55
362	4.5	8.0	0.41	18	16	55
408	4.1	7.2	0.36	16	15	55
453	4.1	7.2	0.36	16	15	55
Wintering Mature Pregnant Cows (Weights are for Beginning of Winter Period)						
362	5.0	7.1	0.45	22	18	55
408	4.5	6.5	0.41	18	16	55
453	4.1	5.9	0.41	16	15	55
498	4.1	5.9	0.36	16	15	60
544	4.1	5.9	0.36	16	15	65

TABLE 9—Recommended Nutrient Allowances for Beef Cattle—(Continued)

Body Weight	Total Digestible Nutrients	Net Energy	Total Digestible Protein	Calcium	Phosphorus	Carotene [1]
	DAILY ALLOWANCE PER ANIMAL					
Kg.	**Kg.**	**Therms**	**Kg.**	**Gm.**	**Gm.**	**Mg.**
Cows Nursing Calves 1st 3 to 4 Months after Parturition						
408–498.....	6.4	12.0	0.64	30	24	300
Fattening Calves Finished as Short Yearlings						
181.....	3.6	7.3	0.50	20	15	25
226.....	4.3	8.7	0.54	20	16	30
272.....	5.0	10.2	0.59	20	17	35
317.....	5.4	11.2	0.64	20	18	40
362.....	6.1	12.7	0.68	20	18	45
408.....	6.6	13.7	0.68	20	18	50
Fattening Yearling Cattle						
272.....	5.2	10.5	0.59	20	17	35
317.....	6.1	12.4	0.64	20	18	40
362.....	6.4	13.1	0.68	20	19	45
408.....	7.0	14.6	0.73	20	20	50
453.....	7.7	16.0	0.77	20	20	55
498.....	7.9	16.5	0.77	20	20	60
Fattening Two-Year-Old Cattle						
362.....	6.8	14.0	0.68	20	20	45
408.....	7.2	14.7	0.73	20	20	50
453.....	7.7	15.9	0.77	20	20	55
498.....	8.2	17.0	0.82	20	20	60
544.....	8.2	17.0	0.82	20	20	65

[1] The recommended carotene allowances for fattening animals is at the same rate as for cattle in other classifications. This is about the minimum rate that will result in significant storage and thus assure contribution of vitamin-A value for human use from the beef liver and fat. For optimum growth or feedlot gains and freedom from clinical symptoms, 3.3 mg. carotene for each 100 kilograms body weight suffices for cattle previously depleted of body stores, and this level may be used except for pregnant or lactating cows when economically necessary. The vitamin-A value of the liver and the body fat of animals so fed, however, would be practically nil. Actually, no dietary carotene or vitamin A is needed so long as the animals have sufficient storage reserve to meet physiological needs.



Figures 45 and 46. This illustrates the effect of the plane of nutrition. Figure 45 is a well-nourished ewe, while the ewe in Figure 46, which received only two-thirds the quantity of feed, showed partial loss of fleece, thin condition, irregular breeding, and poor milk production.

10. Symptoms of Nutritional Deficiencies in Sheep

The economical and efficient production of sheep and wool is contingent upon maximum production per unit of feed. The feeding of sheep for maximum and economic production is dependent upon adequate nutrition. This is an attempt to organize the pertinent data on the nutrition of sheep into a concise form for practical application.

Protein

Protein is of special importance for pregnant and lactating ewes and for young animals. It is suggested that the protein allowance be increased for pregnant ewes in poor condition. Insufficient protein results in reduced body and wool growth and muscular development. The feed intake is lowered because of a decline in appetite and the feed required per unit of gain is increased. Under extreme conditions there are severe digestive disturbances, nutritional anemia, and edema.

Energy

An inadequate allowance of energy in the form of carbohydrates and fat results in slow growth, emaciation, and possibly diminished wool growth (Figure 46). When the allowance of carbohydrates and fat is insufficient to meet the energy needs, protein may be used up for this purpose, decreasing the efficiency of protein utilization for growth and repair of body tissues.

Salt (NaCl)

Sheep that are deprived of adequate salt develop a craving and may resort to chewing wood, licking dirt, and similar manifestations of an unsatisfied appetite. Inadequate salt intake results in lowered feed consumption and in decreased efficiency in the use of feed nutrients.

Calcium and Phosphorus

Not all are agreed on the requirements of sheep for calcium and phosphorus nor on the optimum calcium-phosphorus ratio. The exact

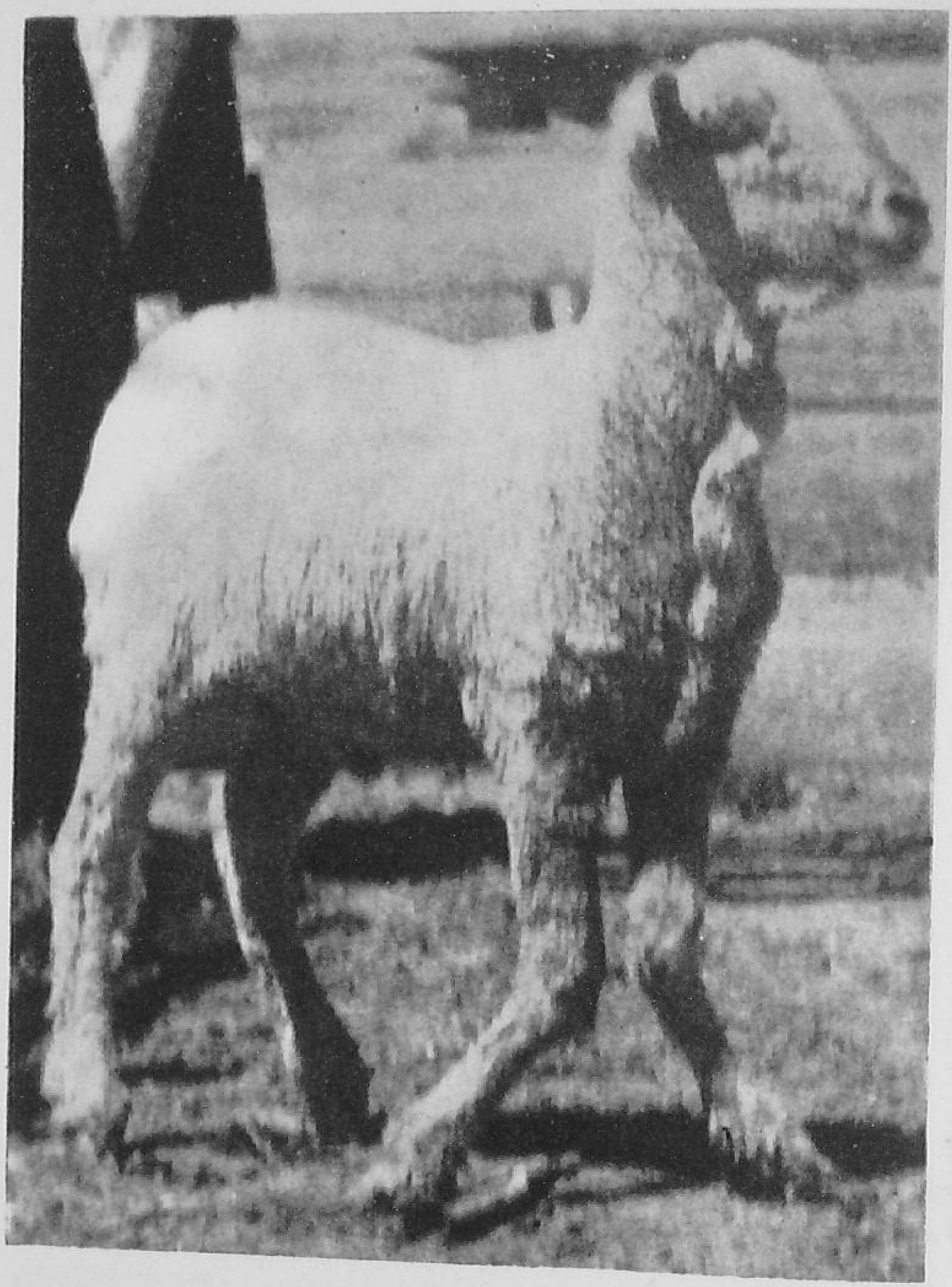


Figure 47. Lamb fed a ration deficient in phosphorus. Note the knock-kneed conformation.

ratio appears to be less important than having an adequate amount of calcium and phosphorus to satisfy the physiological needs of the animal. Rations that are decidedly lacking in calcium or phosphorus, or both, result in subnormal development of bone. Phosphorus deficiency may be indicated by slow growth, high feed requirement, depraved appetite, unthrifty appearance, listlessness, low blood phosphorus, and the development of knock-knees, with a carcass showing a general lack of covering (Figure 47). Aphosphorosis in ewes causes weak lambs and decreased milk production.

Iodine

In iodine-deficient areas, serious losses of lambs are prevented by feeding iodine, as iodized salt, to breeding ewes, especially during the gestation period. Iodized salt is now formulated in the U. S. A. with one part of potassium iodide in 10,000 parts of salt. This is 0.01 percent potassium iodide or 0.0076 per cent iodine. When such iodized salt is normally used it furnishes several times the iodine requirement. The continuous feeding of large intakes of iodine above the requirement may result in definite harm.

Although visible evidences of iodine deficiency are seldom observed in mature sheep, the condition must exist in iodine-deficient areas, since newborn lambs frequently show a characteristic enlargement of the thyroid, and practically every lamb with a large goiter is either dead at birth or dies soon afterwards (Figure 49). If the lambs survive, the enlargement may not be noticed after about a month, though enlargement may be found in post-mortem examinations of lambs in which no evidence of goiter had previously been observed.

Cobalt

The recommended allowances of cobalt for animals cover a wide range. It seems from the available data that 0.1 mg. of cobalt daily is sufficient to meet the needs of ewes. Adequate amounts of cobalt may be provided in deficient areas by feeding salt containing 62 grams of cobalt chloride or cobalt sulfate per 1,000 kg. of salt.

Cobalt deficiency is accompanied by a loss of appetite, lack of thrift, weakness, anemia, and a decrease in fertility and milk production.

Copper and Iron

Copper deficiency may exist as a primary deficiency or as an accompaniment of cobalt and iron deficiencies. The symptoms of inadequate

Figure 48. Ewe fed a ration low in vitamin A. One lamb was born dead and the other died six hours after birth.

copper are generally seen in young lambs. At birth the lambs are weak, often unable to nurse, and death may result from starvation. There is a lack of muscular co-ordination, and degeneration of the myelin of the nerves, and especially of the spinal cord. The disease caused by a lack of copper is referred to as enzootic ataxia.

A daily intake of 5 mg. of copper is adequate for pregnant ewes even when the pastures are extremely deficient in copper. The addition of from 0.25 to 0.5 percent of copper sulfate ($CuSO_4 \cdot 5H_2O$) to salt would furnish about 10 mg. of copper per day. Other investigators have reported that 1 percent of copper sulfate is better than 0.3 percent in preventing "sway-back" in lambs. Excessive amounts of copper are definitely toxic.

The iron requirements of sheep do not appear to have been determined.

Vitamin A

One of the first symptoms of vitamin A deficiency is night blindness, or inability to see in dim light. This is followed by nervous disorders resulting in various degrees of incoordination and spasms. Urinary calculi may occur in advanced stages of vitamin A deficiency and may cause im-

mediate death. A deficiency of vitamin A adversely affects reproduction, and may result in lambs being born weak or dead (Figure 48).

Carotene allowances are shown in Table 10, since it is the precursor of vitamin A and the form available in natural feeds consumed by sheep. Minimum carotene requirements of sheep, cattle, and swine are similar and vary from 25 to 35 micrograms per kilogram of live weight for the prevention of nyctalopia, or approximately 1.7 mg. per 50 kilograms of live weight. This amount does not allow for storage, reproduction, or other special demands in the body. The recommended allowance is approximately four times this minimum, to provide for moderate storage in the body and to meet the demands for reproduction and lactation.

In terms of vitamin A, the minimum for growth would be approximately 1,000 I. U. per 45 kilograms of live weight. For storage and reproduction 3,000 to 4,200 I. U. daily per 45 kilograms of live weight should be provided.

The carotene content of forage varies considerably. The stage of maturity, method of curing and preservation, length of storage period, and temperature affect the carotene content of forages. The best practical guide to the carotene value of forages, aside from actual chemical

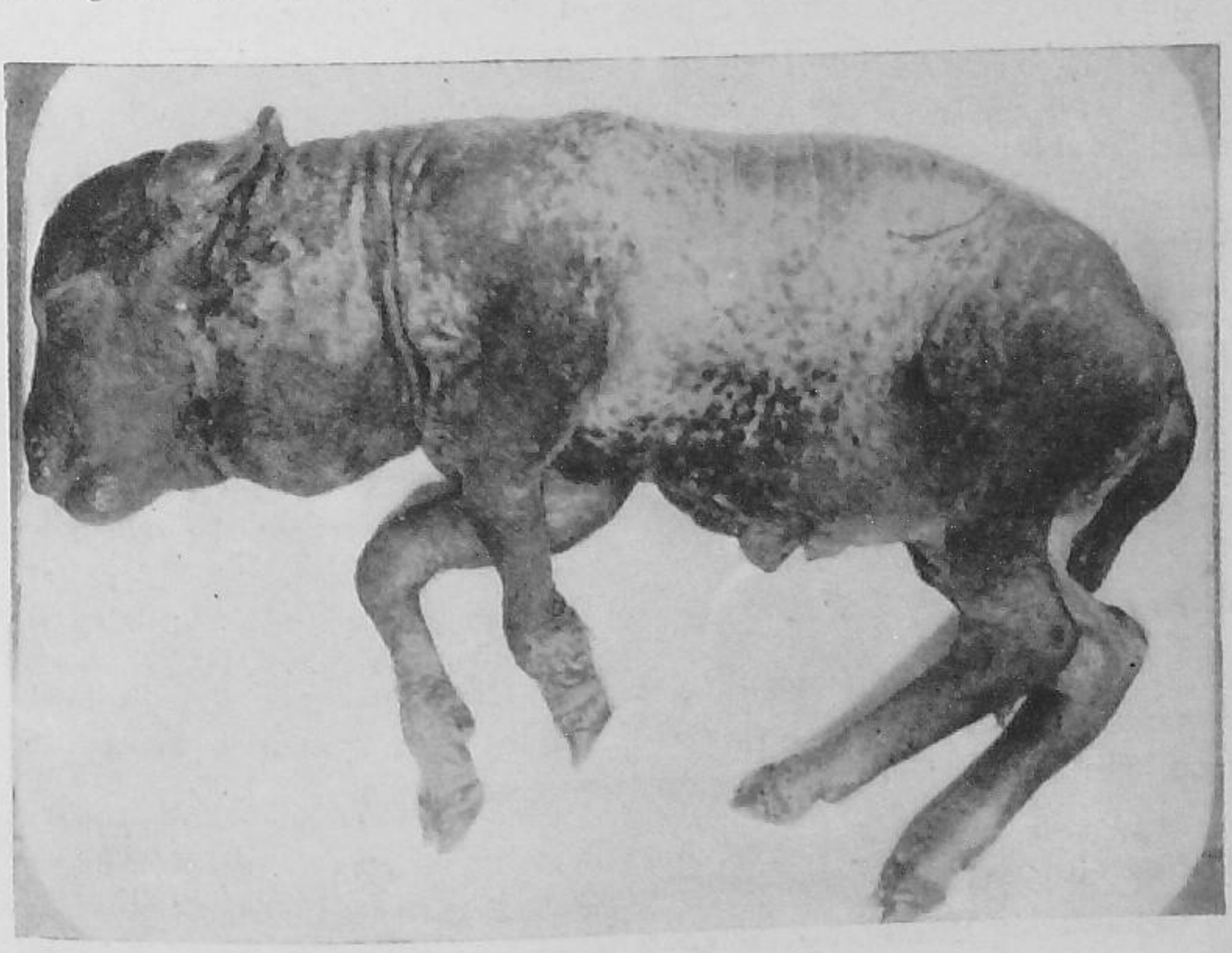


Figure 49. New-born lamb showing goiter and very thin wool.

analysis, is the degree of green color. As a general rule forages that have retained much of their original green color are far better sources of carotene than those that have been allowed to mature and weather.

Vitamin D

Lack of vitamin D may cause disturbance in the metabolism of calcium and phosphorus. An adequate supply of vitamin D is especially important for the normal development and calcification of bone during growth. A deficiency of vitamin D is manifest by one or more of the following symptoms: enlargement of the joints and bowing of the legs in the immature animal, stiffness in the anterior or posterior quarters, and an irregular gait. The joints appear to be very painful and the lamb may carry or drag one limb. Such stiffness may be accompanied by loss of appetite.

Under range conditions, sheep probably do not need added amounts of vitamin D. Where they are confined, or exposure to sunshine is for some reason restricted, lambs may develop rickets from a lack of vitamin D. It has been shown that lambs fed a ration low in vitamin D and not exposed to sunshine develop rickets. In these experiments rickets could be prevented by feeding a vitamin D concentrate. The quantitative requirements of the sheep for vitamin D have not yet been determined. In the absence of more exact information it is suggested that the proposed figure of 300 I. U. of vitamin D per 45 kilograms of live weight for calves might be used for sheep.

Vitamin B Complex

The vitamin B complex has been shown to have no effect on the performance of ewes during breeding and pregnancy. Lambs fed a ration low in nicotinic acid during an eight months period developed normally. Even though sheep are fed diets low in thiamin, riboflavin, pyridoxine, and pantothenic acid, these dietary factors are synthesized in the rumen.

Other Vitamins

If sheep require vitamin E in the diet for normal reproduction, their requirement is extremely low, and common feeds contain sufficient to meet the functional needs of the ewe for reproduction.

Other Nutritional Disturbances

Overeating disease, also referred to as apoplexy, gastroenteritis, food intoxication, or infectious enterotoxemia, is the cause of considerable

death loss in nursing and fattening lambs. The disease occurs among lambs that are fed a heavy allowance of grain. Recovery from the disease is rare and the lambs usually die within a short time. Lambs with overeating disease may throw back their heads, stagger, move in circles, or push against a fence, and then fall and die in convulsions. The disease may be prevented or kept to a minimum by preventing lambs from gorging on grain at any time, and by feeding a safe proportion of grain to roughage. Should losses occur, it is recommended that one feeding of grain be omitted or the amount of grain reduced for a few feedings.

Pregnancy disease, also referred to as ketonemia and acetonemia, is an ailment of ewes occurring in late pregnancy. It is much more common among ewes carrying twins or triplets than in ewes carrying single lambs. The first symptoms observed may be a sweetish odor like chloroform, accompanied by general sluggishness, loss of appetite, staggering gait, and nervousness. In the final stages of the disease there is impaired vision, and the ewe is unable to stand or to rise on account of weakness, stiffness, or partial paralysis. If parturition occurs during the earlier stages of the disease, recovery usually results. The disease has been produced experimentally by feeding pregnant ewes rations of poor quality or rations low in energy. It is associated with marked ketosis, both blood and urine containing excessive amounts of ketone bodies. It is essentially a disturbance of metabolism, especially with regard to the carbohydrates. The disease is avoided by feeding and management practices that ensure a uniform and adequate, but not excessive, intake of a balanced ration, especially during the last six weeks of pregnancy. Sudden interruptions in the feeding schedule, especially reducing the plane of nutrition, should be avoided.

Stiff-lamb disease, of nutritional origin, is a specific muscular stiffness characterized by whitish calcareous intermuscular deposits; it occurs in lambs a few days to several weeks old. The disease is reported as being rather widespread, with heavy losses in individual flocks. Lambs affected become stiff and have difficulty in walking, and frequently lag behind the band. The etiology of nutritional stiff-lamb disease is not definitely known. Some experiments have shown that the incidence of the disease can be reduced to a minimum by feeding wheat-germ meal. Experiments in which tocopherols have been used indicate that a lack of vitamin E in the ration may be the cause of stiff-lamb disease. Stiffness resulting from a nutritional deficiency should not be confused with a similar stiffness resulting from bacterial infection manifested particularly in older lambs. Stiffness may also occur in lambs from erysipelothritic arthritis.

Urinary calculi may cause considerable loss, particularly among lambs in the feedlot. The first symptoms usually observed are restlessness and an occasional straining to urinate. The etiology of urinary calculi is not fully known. A deficiency of vitamin A may cause it. Experiments have failed to find any evidence that feeding an excess of magnesium is the cause. Rations high in calcium, magnesium, and phosphorus have been fed to lambs, with no evidence of urolithiasis. Evidence that urinary calculi may be caused by an unbalance of minerals in the ration is afforded by the extensive studies on laboratory animals.

Selected References

DUCKWORTH, J., W. GODDEN, and W. THOMSON. 1943. The relation between rates of growth and rickets in sheep on diets deficient in vitamin D. Jour. Agr. Sci. *33*:190-96.

HART, G. H. 1940. Vitamin A deficiency and requirements of farm animals. Nutr. Abst. and Rev. *10*:261-72.

MADSEN, L. L. 1942. Nutritional diseases of farm animals. U. S. D. A. Yearbook of Agri., *Keeping Livestock Healthy*, pp. 323-353.

MAYNARD, L. S. 1947. *Animal Nutrition.* 2d ed. New York: McGraw-Hill Book Co. Pp. 494.

MILLER, R. F., G. H. HART, and H. H. COLE. 1942. Fertility in sheep as affected by nutrition during the breeding season and pregnancy. Calif. Agr. Expt. Sta. Bul. 672. Pp. 31.

MITCHELL, H. H., and F. J. MCCLURE. 1937. Mineral nutrition of farm animals. National Research Council Bul. 99. Pp. 135.

MORRISON, F. B. 1945 (c. 1936). *Feeds and Feeding.* 20th ed. Ithaca, N. Y.: Morrison Publishing Co.

PEARSON, P. B., H. M. BRIGGS, W. G. KAMMLADE, J. I. MILLER, and R. F. MILLER. 1945. Committee on Animal Nutrition, Report No. 5. Recommended nutrient allowances for sheep. National Research Council, U. S. A.

SCHMIDT, H. 1941. Vitamin A deficiencies in ruminants. Amer. Jour. Vet. Research. *2*:373-89.

THEILER, A., and H. H. GREEN. 1932. Aphosphorosis in ruminants. Nutr. Abst. and Rev. *1*:359-85.

TABLE 10—RECOMMENDED NUTRIENT ALLOWANCES FOR SHEEP[1]

Live Weight	Total Digestible Nutrients	Net Energy	Total Digestible Protein	Calcium	Phosphorus	Carotene	NaCl (Salt)
	DAILY ALLOWANCE PER ANIMAL						
Kg.	**Kg.**	**Therms**	**Gm.**	**Gm.**	**Gm.**	**Mg.**	**Gm.**
	Bred Ewes First 100 Days of Gestation						
45	0.77	1.4	77	3.2	2.5	5.5	14
50	0.82	1.5	82	3.2	2.6	6.0	14
54	0.86	1.6	86	3.3	2.7	6.5	14
59	0.91	1.7	91	3.4	2.7	7.0	14
	Bred Ewes Last 6 Weeks Before Lambing						
50	0.95	1.8	95	4.3	3.2	6.0	14
54	1.00	1.9	100	4.4	3.3	6.5	14
59	1.04	2.0	104	4.5	3.4	7.0	14
64	1.09	2.1	109	4.7	3.5	7.5	14
68	1.13	2.2	113	4.8	3.6	8.0	14
	Ewes in Lactation						
45	1.13	2.1	122	6.1	4.5	6.0	14
50	1.18	2.2	127	6.2	4.6	7.1	14
54	1.22	2.3	127	6.4	4.7	7.8	14
59	1.27	2.4	136	6.5	4.8	8.4	14
64	1.32	2.5	136	6.6	4.9	9.1	14
68	1.36	2.6	141	6.8	5.0	9.7	14
	Ewes—Lambs and Yearlings						
32	0.82	1.5	100	3.6	2.4	3.8	9
41	0.86	1.6	100	3.9	2.6	5.0	9
50	0.86	1.6	91	3.8	2.5	6.0	14
59	0.91	1.7	91	4.1	2.7	7.1	14
	Rams—Lambs and Yearlings						
34	0.95	1.8	109	4.3	2.8	4.1	9
45	1.04	2.0	109	4.9	3.2	5.5	14
57	1.09	2.1	109	4.9	3.2	6.9	14
68	1.18	2.4	104	4.7	3.1	8.2	14
79	1.18	2.4	104	4.9	3.2	9.6	14
	Fattening Lambs						
23	0.54	1.0	77	2.8	1.9	2.7	9
27	0.64	1.2	82	3.1	2.1	3.3	9
32	0.77	1.5	86	3.3	2.2	3.8	9
36	0.86	1.7	91	3.5	2.3	4.4	9
41	0.91	1.9	91	3.7	2.4	5.0	9

[1] These recommended nutrient allowances for sheep should satisfy the nutritional requirements of angora goats.

11. Symptoms of Nutritional Deficiencies in Horses and Mules

The principles of the nutrition of horses and mules[1] are not fundamentally unlike those of other domestic livestock, but naturally the quantities and importance of the various nutrients differ from those required by other farm animals.

Protein

In the young growing colt, the result of inadequate protein intake is usually evidenced by slow or stunted growth and improper development. With breeding stock, there may be impairment of the reproductive functions. Mature, idle stock ordinarily are not seriously affected if the lack of protein is not too great and does not extend over a long period. Work animals, however, may lack spirit and efficiency.

Energy

Lack of adequate amounts of total energy in the rations of horses or mules may result in a number of consequences. When the energy deficiency is great in the feed of young animals, the result is usually slow and stunted growth, with consequent underdevelopment. Work animals that do not receive adequate amounts of energy lose weight, get out of condition, and are unable to do their jobs without excessive fatigue. When the energy requirements of breeding stock and idle animals are not satisfied, the effect is usually a loss in body weight.

Improper balance of nutrients in the maintenance ration, caused by excessive amounts of feeds with wide nutritive ratios, may result in depression of digestibility, poor feed utilization, stunted or improper growth, and impairment of health, if continued for a very extended period.

---

[1] Although most of the research and investigation in the field of nutrition has not considered mules, the results obtained from some tests and from practical experience indicate that their requirements for feed are essentially the same as those of horses. The nutritive needs of horses and mules are accordingly treated as one in this paper.

Climatic conditions and the method of managing horses and mules affect their energy-maintenance needs. Very cold weather increases considerably the amount of energy required as does also any mismanagement that keeps the horse or mule in a restless, excitable condition for any considerable length of time.

Calcium and Phosphorus

A horse or mule must have a sound, fully developed body to be of economic importance. Such development is possible only when the skeletal framework is adequate, and this can be assured by the judicious use of rations containing rather liberal amounts of calcium and phosphorus.

Unfortunately there is little experimental data on the requirements for calcium and phosphorus by young horses and mules. On the basis of practical observations and other species requirements, calcium and phosphorus should each make up 0.2 percent of the dry ration of horses in a ratio of Ca:P of 1:1 or 2:1. Colts require more than this percentage, especially of calcium. Practical experience and general knowledge of nutrition indicate that mature work horses, with the possible exception of brood mares, do not require the addition of any minerals except common salt, to rations which contain an ordinary amount of good hay. Colts, pregnant mares, and mares nursing foals require much more calcium and phosphorus than mature horses. Well-cured legume hay or mixed hay high in legumes is, therefore, the best roughage for colts and brood mares during the winter season. In very young animals, calcium and phosphorus malnutrition is most evident in rickets.

Lack of calcium in the horse ration, when intensified by the presence of high phosphorus protein concentrates, is believed to produce the condition commonly known as osteomalacia or osteodystrophia fibrosa, which is prevalent in various parts of the world and affects not only horses but asses and their hybrids, with the young of the species most susceptible. This disease is usually characterized by an initial period of stiffness and lameness, followed by swelling of the jaws and the nasal and frontal bones of the head, bone fractures, detachment of ligaments, anemia, emaciation, and death. This is said to develop when the calcium-phosphorus ratio of the ration is 0.55 to 1 or less and to be arrested by the addition of calcium supplements until the ratio becomes 1.6 or more of calcium to 1 of phosphorus.

Whenever horses must be fed rations that are deficient in either calcium or phosphorus, care should be taken to supply a suitable mineral

supplement. Such a mineral mixture as shown in Table 11 should be available at all times.

TABLE 11

MINERAL MIXTURES FOR HORSES AND MULES

Ingredient	Calcium and phosphorus both deficient	Calcium deficient	Phosphorus deficient
	Parts by Weight		
Steamed bone meal (or other phosphorus supplement)	2	0	2
Limestone ($CaCO_3$) or other Calcium supplement	2	2	0
Salt (NaCl)	1	1	1

Additional salt should be fed. If a calcium deficiency alone exists, 30 gm. per head of ground limestone or other calcium supplement will correct the deficiency; if a phosphorous deficiency alone exists, 30 gm. per head daily of steamed bone meal or some other safe source of phosphorus should be used.

Salt (NaCl)

Horses and mules of all ages need sodium chloride—common salt—regularly in their diet. Since horses differ widely in the amount of salt they desire, it is a good plan to supply salt where they can take what they wish, either flake salt from a suitable box, or block salt. An allowance of 50-60 grams per head daily is ample. If salt is not provided, especially when the animals are doing heavy work in hot weather, they exhibit signs of excessive fatigue.

Iodine

In areas where symptoms of iodine deficiency occur, 1 gram of potassium iodide weekly for pregnant mares during the last five or six months of the gestation period, iodized salt containing 0 02 percent potassium iodide—stabilized to prevent loss of iodine—may be fed. Iodine-deficiency symptoms are weak foals with thick necks or goiter. Navel-ill and weakness of foals has sometimes been lessened by the feeding of potassium iodide to brood mares during pregnancy.

Vitamins

The role played by vitamins in the nutrition of the horse and mule has not been explored extensively. However, there is reason to suppose that

the vitamin requirements of horses and mules are similar to those of other animals, and it seems probable that ordinarily there are few serious deficiencies of any of the vitamins except A and D.

Recent evidence points to the importance of riboflavin in preventing degenerative changes in the eye. Periodic ophthalmia or moon blindness was prevented by feeding 40 milligrams of crystalline riboflavin daily.[2] This result shows that further research into the vitamin requirements of the horse should be carried out. Preliminary experiments have thrown some light on at least one of the qualities of pasture that makes it a valuable feed for breeding animals, this is its ascorbic acid content. The question of the importance of ascorbic acid to the horse and whether additional ascorbic is needed has not yet been demonstrated, but warrants investigation.[3]

The importance of vitamin A or its precursor, carotene, is indicated by its relation to two important attributes of a horse—eyesight and hoofs. Night blindness resulting from a deficiency of vitamin A has been demonstrated in horses, and the injurious effect of lack of vitamin A has been indicated by experiments with horses in the army of Finland. The uneven and poor development of the hoofs of these horses, which has been fed chiefly on old hay, was cured by supplementary feeding of pasture grasses, grass silage, and cod-liver oil. No especial attention need be given to the vitamin supply of mature work horses, except perhaps brood mares: the requirements of such horses for vitamins A and D are apparently low, and there seem to be no deficiencies in ordinary rations.

Foals, on the other hand, develop rickets, owing to lack of vitamin D or of calcium or phosphorus. An ounce of cod-liver oil a day, or preferably an equivalent amount of a vitamin-D concentrate, free access to bone meal or other suitable mineral mixture, with plenty of good legume hay or good pasture, cures the condition. The vitamin requirements of brood mares are met if they are fed good legume hay, mixed hay or pasture. Greenness is a good indicator of the vitamin-A content of hay or pasture. Vitamin D is only developed in the hay while it is being sun-cured. It should be borne in mind that the winter sun in the north and south temperate zones has very little vitamin D activity.

---

[2] N. R. Ellis. New ideas in feeding. U. S. D. A. *Yearbook of Agriculture*, 1943-47, p. 105

[3] R. S. Hudson and Byron Good. Factors affecting farm horse power. Michigan Agricultural Experimental Station (U. S. A.), Quarterly Bulletin, Vol. 28, February 1946.

TABLE 12—RECOMMENDED NUTRIENT ALLOWANCES FOR HORSES

Type and Weight of Animal	Dry Matter	Total Digestible Nutrients	Net Energy	Total Digestible Proteins
Kg.	**Kg.**	**Kg.**	**Therms**	**Kg.**
Idle Horses				
453	7.0	3.6	6.4	0.31
544	8.0	4.1	7.3	0.36
635	9.0	4.6	8.2	0.40
725	9.9	5.1	9.0	0.45
816	10.8	5.6	9.8	0.49
Horses at Light Work				
453	7.9	4.5	8.3	0.40
544	9.2	5.2	9.6	0.45
635	10.4	5.9	10.8	0.49
725	11.6	6.6	11.1	0.58
816	12.7	7.3	13.3	0.63
Horses at Medium Work				
453	8.4	5.4	10.2	0.49
544	9.8	6.3	12.0	0.58
635	11.2	7.3	13.7	0.63
725	12.6	8.2	15.4	0.72
816	14.0	9.1	17.1	0.81
Horses at Hard Work				
453	9.1	6.6	12.6	0.58
544	10.7	7.8	14.9	0.68
635	12.4	9.0	17.3	0.77
725	14.1	10.2	19.5	0.90
816	15.7	11.4	21.8	0.99
Brood Mares, Nursing Foals, not at Work				
453	8.4	4.8	8.8	0.58
544	9.7	5.5	10.2	0.68
635	11.0	6.2	11.5	0.77
725	12.2	6.9	12.8	0.86
816	13.4	7.6	14.1	0.95
Growing Draft Colts, after Weaning				
181	4.6	2.9	5.6	0.36
227	5.5	3.4	6.5	0.40
272	6.3	3.9	7.4	0.49
317	7.0	4.4	8.3	0.54
363	7.7	4.8	9.0	0.58
408	8.4	5.3	9.8	0.63
453	9.1	5.7	10.5	0.68
499	9.7	6.1	11.2	0.68
544	10.3	6.5	12.0	0.72

DAILY ALLOWANCE PER ANIMAL

Selected References

ELLIS, N. R., and J. O. WILLIAMS. 1945. Feeding horses. U. S. D. A. Farmers Bull. No. 1030.

GUILBERT, H. R., C. E. HOWELL, and G. H. HART. 1940. Minimum vitamin A and carotene requirements of mammalian species. Jour. Nutr. *19*:91-103.

KRANTZ, E. B., and S. R. SPEELMAN. 1939. Nutrition of horses and mules. U. S. D. A. Yearbook, *Food and Life*, pp. 763-786.

MADSEN, L. L. 1942. Nutritional diseases of farm animals. U. S. D. A. Yearbook, *Keeping Livestock Healthy*, pp. 323-353.

MITCHELL, H. H. 1947. The mineral requirements of farm animals. Jour. Anim. Sci. 6:365-377.

——— and F. J. MCCLURE. 1937. Mineral nutrition of farm animals. National Research Council Bull. 99.

MORRISON, F. B. 1945 (c. 1936). *Feeds and Feeding*. 20th ed. Ithaca, N. Y.: Morrison Publishing Co. Pp. 1050.

12. Nutritional Requirements of Goats[1]

In general, the same feeds, care, and management that are successful with dairy cows and sheep are suitable for milk goats. On the basis of body weight and amount of feed consumed, good milk goats are about as efficient milk producers as good cows. The maintenance requirements for goats per 50 kg. live weight are considerably higher than for cows, on account of their smaller size and greater body surface in proportion to live weight. Also, for the production of each pound of milk of a given fat content, goats apparently require slightly more digestible nutrients above maintenance requirements than cows.

However, they are able to consume much more feed per 100 kg. live weight than cows, even those of high productive capacity. Therefore, they have left, after their maintenance requirements are met, a sufficiently large proportion of the nutrients furnished by their feed to meet their needs. Goats browse more than sheep, but respond to good pasture and roughage.

Individual goats differ in their ability to turn feed into milk or growth. The variations may be due largely to individual differences in appetite and inherited ability to utilize feed. Milking does are fed at the rate of 1 kilogram of grain for each 2 to 4 kilograms of milk produced, depending on the quality of the roughage and condition of the goat. Thus, a doe that increases milk production with increased grain feed is permitted to demonstrate her maximum ability.

Because feeds vary considerably in composition, the nutritional requirements of animals can be more accurately expressed in terms of digestible nutrients than by kilograms of feed in the ration. W. L. Gaines, of the University of Illinois, U. S. A., reports the development of a standard equation for calculating the feed requirements of milking goats. The equation is given as $DN=0.016W\div0.3FCM$, in which DN equals the daily digestible nutrients intake in kilograms, W equals the live weight of the doe in kilograms, and FCM equals the daily milk energy yield in kilograms of 4-percent milk. The milk production of a doe may be corrected to a butterfat basis of 4 percent by use of the Gaines-Davidson

[1] See Table 10 for nutrient allowances.

formula: FCM (fat corrected milk) $=0.4M+15F$, in which M represents the weight of milk and F the weight of fat. By converting milk yields to an FCM basis, more accurate comparisons of the productive abilities of individual does is possible.

Goats are as fond of salt (NaCl) as are sheep, and, when they are accustomed to it, it should be kept where they can take as much as they want. Calcium supplements may be needed where the forage is non-leguminous and grown on soils low in calcium. About 110 grams of ground limestone or some other calcium supplement per head daily may be fed in the form of a mixture of 2 parts of calcium supplement to 1 part of common salt (NaCl). If phosphorus appears to be lacking in the forage it may be supplied in bone meal in a similar mixture with salt. Where there is trouble from goiter in new-born kids, the does should be fed iodized salt at least during the last half of pregnancy, so that they will get about 3.5 mg. of potassium iodide per head daily. Larger doses may be injurious.

There have been no developments to indicate that vitamins other than A and D are needed in goat feeding.

Angora Goats

Angora goats usually obtain most of their feed on the range, from browsing or pasturing. When supplemental feed is necessary they can use to advantage any of the common roughage or concentrate feeds. If the hay or other roughage is a nonlegume they should be fed a protein-rich concentrate. If the goats are confined, the daily allowance per head may need to be about 1.4-1.8 kilograms of good quality roughage and about 0.11 to 0.23 kilogram of concentrates. The bucks require a greater amount of feed than does in proportion to their weight. The kids in a herd of Angora goats are usually raised as suckling kids on their mothers' milk and on feeds available to their mothers.

Milk Goats

The feeding requirements of milking does are similar to those of dairy cows. About six to eight goats can be fed on the quantity of feed required by one cow. When does are in milk they need all the roughage they will consume. Succulent feed is beneficial with the dry roughage and concentrates. An example of a satisfactory ration for a confined doe in milk is 0.90 kg. legume hay, 0.68 kg. silage or roots, 0.45 to 0.90 kg. of grain mixture. When on good pasture the doe may need 0.45 to 0.68

kg. of a grain mixture. One-half kilogram of grain mixture is the average daily grain requirement per liter of milk produced during the entire period of lactation. The grain mixture should contain 25 percent of protein-rich feeds.

Young does should be kept in good growing condition. On good pasture it is not necessary to supply supplemental feed. In winter they need about half a kilogram of grain, 450 to 675 grams of silage or roots, and all the hay or other roughage they will consume.

When the milk is sold or used by the family, the feeding of kids to be raised for breeding and milk purposes requires special attention. Each kid should receive 675 to 900 grams of milk per day along with good pasture or other roughage, and a little grain. When the kids are about ten weeks old the milk in the ration may be replaced to a large extent by good alfalfa hay and mixed grain. Whole cow's milk can be fed successfully to kids. Skim milk can be used with a fair degree of success if the change from whole milk to skim milk is made gradually and the kids are allowed 0.90 or 1.36 kg. of milk a day in three feedings until they are six weeks old.

Choice alfalfa hay and grain should be fed in addition to skim milk. The kids can be weaned when they are three or four months old, although when they are raised as suckling kids it is not customary to wean them until they are about five months of age.

Goats for Meat Production

Although goats have not been especially improved for meat production, the meat of healthy, normal goats is wholesome food. Hundreds of thousands of goats are marketed for their meat annually, and the feeding of such animals has an important bearing on their market value.

In order to obtain the best finish, emphasis should be placed on such fattening feeds as corn, barley, or grain sorghum, along with good grazing forage or legume hay and succulent feeds. The daily allowance per head of stored feeds for fully grown goats should be approximately as follows: 900 grams of legume hay, 675 grams of silage or roots, 450 to 900 grams of grain mixture. The feed allowance varies for goats of different ages, weights, and conditions of flesh or fatness.

Possibilities of Improving Goats Through Nutritional Research

Progress in feeding practices has been made by some producers of improved goats, but there is need for more definite information on the efficiency of various feeds and combinations of feeds, including the forage

plants of pastures and range, from the standpoint of their influence on the quality and quantity of mohair and milk produced. As economy of production becomes increasingly important, the need for such information will be even more urgent than it is now. Fundamental research on the nutritional requirements of goats and their ability to utilize various feeds and combinations of feeds offers the primary means of increasing economy. Such research would include work on the precise role of minerals, protein, and vitamins in the nutrition of goats.

As among sheep, there is much evidence of wide variations in the efficiency of individual goats to utilize feed. It is also apparent that such variations exist among different families and strains. It should be possible to develop strains that are highly efficient in converting available feeds into mohair or milk of superior quality and quantity. Once such strains were developed, they could be used in suitable breeding practices to raise the average efficiency of these animals.

In such an effort, research workers in nutrition and genetics would have to co-operate with each other and enlist the co-operation of the producers of mohair and milk. The pioneer period of goat production is passing, just as it is with sheep. This inevitably means that the time has come for theoretical research and practical experiment to make these animals more efficient.

Selected References

GAINES, W. L. 1946. Live weight versus metabolism body size in dairy cows and goats. Jour. Dairy Sci. *29*:259-272.

——— 1943. Feeding standard equations for cows and goats in milk. Jour. Animal Sci. *2*:304-313.

MADSEN, L. L. 1942. Nutritional diseases of farm animals. U. S. D. A. Yearbook of Agriculture, *Keeping Livestock Healthy*, pp. 323-353.

MORRISON, F. B. 1945 (c. 1936). *Feeds and Feeding*. 20th ed. Ithaca, N. Y.: Morrison Publishing Co. Pp. 1050.

POTTS, C. G., and U. L. SIMMONS. 1942. Milk goats. U. S. D. A. Farmers Bull. (Revised) No. 920. Pp. 36.

TELFER, J. A. 1944. The milk goat in Canada. Dom. of Canada Dept. of Agriculture. Publication 763 (Farmers' Bulletin 125).

VAN DER NOOT, G. 1946. Dairy goat management. New Jersey Agricultural Expt. Sta. (U. S. A.) Circular 418.

TABLE 13—SUMMARY OF CLINICAL SYMPTOMS OF DIETARY DEFICIENCIES IN LIVESTOCK (*Not including Poultry*)

Deficiency	Decreased Rate or Cessation of Growth	Rough Coat, Unthriftiness	Reduced Appetite, Anorexia	Decreased Milk Production	Emaciation, Loss of Weight	Dead or Weak Offspring	Stiff Joints, Lameness	Pica	Impaired Reproduction	Collapse or Death	Fragile, Weak or Fractured Bones	Anemia	Diarrhea	Muscular Incoordination	Decreased Phosphorus in Blood Plasma	Staggering Gait	Convulsive Seizures	Straight Pasterns	Craving for Salt	Suppression of Estrus	Goiter	Day or Night Blindness	Decreased Vitamin A in Blood Plasma	Shy Breeding	Watery Eyes, Nasal Discharge	Secondary Pneumonia	Decreased Calcium in Blood Plasma	Arching of Back	Tetany	Edema	Enlarged Joints and Bowed Legs
Energy	x	x		x	x				x	x										x											
Protein	x		x	x	x	x														x											
NaCl (Salt)	x	x	x	x	x			x		x									x												
Calcium	x	x	x	x		x	x		x		x																x				
Phosphorus	x		x	x		x	x	x	x		x				x																
Iron	x	x	x			x						x		x																	
Copper	x	x	x			x		x				x	x	x				x		x											
Cobalt		x	x	x	x			x				x	x																		
Iodine		x				x															x										
Vitamin A	x	x			x	x	x		x	x			x	x		x	x					x	x	x	x	x				x	
Vitamin D	x		x		x	x	x				x				x	x	x	x									x	x	x		x
Thiamin [1]	x	x	x										x																		
Riboflavin [1]	x	x	x				x						x												x						
Niacin [1]	x	x	x										x																		
Pantothenic Acid [1]	x	x	x				x						x			x															
Pyridoxine [1]	x	x	x				x					x	x				x														

[1] Swine only.

NOTE: There is some difference between species in the manifestation of deficiency symptoms. All symptoms do not necessarily occur when a deficiency exists. Multiple deficiencies result in a combination of symptoms.

STABILIMENTO TIPOGRAFICO FAUSTO FAILLI — ROMA

SALES AGENTS FOR FAO PUBLICATIONS

ARGENTINA : Editorial Sudamericana, S. A., Alsina 500, Buenos Aires.
AUSTRALIA : H. A. Goddard Pty. Ltd., 225*a* George Street, Sydney.
AUSTRIA : Wilhelm Frick Verlag, Graben 27, Vienna 1.
BELGIUM : Agence et Messageries de la Presse, 14-22 rue du Persil, Brussels.
BRAZIL : Livraria Agir, rua Mexico, 98-B, Rio de Janeiro.
CANADA : The Ryerson Press, 299 Queen Street West, Toronto 2, Ontario.
COLOMBIA : " Agricultura Tropical ", Avenida Jiménez No. 8-74, Bogotá
COSTA RICA : Trejos Hermanos, Apartado 1313, San José.
CUBA : René de Smedt, La Casa Belga, O'Reilly 455, Havana.
CYPRUS : Marcos E. Constantinides, P. O. Box 473, Nicosia.
DENMARK : Ejnar Munksgaard, Norregade 6, Copenhagen K.
ECUADOR : " La Hacienda ", Malecón 710-711 y Roca, Guayaquil.
EGYPT : Librairie de la Renaissance d'Egypte, 9 Sh. Adly Pacha, Cairo.
EL SALVADOR : Manuel Navas y Cia., 1ª Avenida Sur 35, San Salvador.
FINLAND : Akateeminen Kirjakauppa, 2 Keskuskatu, Helsinki.
FRANCE : Les Editions A. Pedone, 13 rue Soufflot, Paris 5e.
GERMANY : Paul Parey, Lindenstr. 44-47, Berlin SW 68.
GREECE : " Eleftheroudakis ", Place de la Constitution, Athens..
GUATEMALA : Goubaud y Cia., Ltda., 5ª Avenida Sur No. 6ª y 9ª, Guatemala.
HAITI : Max Bouchereau, Librairie " A la Caravelle ", Boîte Postale IIIB, Port-au-Prince.
ICELAND : Halldor Jonsson, Mjostraeti 2, Reykjavik ; Jonsson & Juliusson, Garöstraeti 2, Reykjavik.
INDIA : The Oxford Book and Stationery Co., Scindia House, New Delhi ; The Mall, Simla ; 17 Park Street, Calcutta ; Messrs Higginbothams, Mount Road, P. O. Box 311, Madras ; Kitab Mahal Publishers 235, Hornby Road, Bombay.
IRELAND : The Controller, Stationery Office, Dublin.
ISRAEL : Blumstein's Bookstore Ltd., P. O. Box 4154, Tel Aviv.
ITALY : Libreria Internazionale, Ulrico Hoepli, Galleria, Piazza Colonna, Roma ; Libreria Internazionale, Dr. Romano Romani, Via Meravigli 16, Milano.
JAPAN : Maruzen Company Ltd., 6 Tori-Nichome, Nihonbashi, Tokyo.
LEBANON : Librairie Universelle, Avenue des Français, Beyrouth.
MEXICO : Manuel Gómez Pezuela e Hijo, Donceles 12, Mexico, D. F.
NETHERLANDS : N. V. Martinus Nijhoff, Lange Voorhout 9, The Hague.
NEW ZEALAND : Whitcombe & Tombs Ltd., Auckland, Wellington, Hamilton, Christchurch, Dunedin, Invercargill, Timaru.
NORWAY : Johan Grundt Tanum Forlag. Kr. Augustsgt. 7ª, Oslo.
PAKISTAN : *East :* Farcos' Publications, 2 Inglis Road, P. O. Box 13, Ramna, Dacca. *West :* Feroz-sons, 60 The Mall, Lahore ; Variawa Bldg., McLeod Road., Karachi ; 35 The Mall Peshawar :
PERU : Libreria Internacional del Perú, S. A., Casilla 1417, Lima.
PHILIPPINES : D. P. Pérez Company, 169 Riverside, San Jaun, Rizal.
PORTUGAL : Livraria Bertrand, S. A. R. L., Rua Garret 73-75, Lisbon.
SPAIN : Aguilar S. A. Ediciones, Juan Bravo 38, Madrid ; José Bosch Librero, Ronda Universidad II, Barcelona ; Salvat Editores, S. A., 41-49 Calle Mallorca, Barcelona.
SWEDEN : C. E. Fritze, Fredsgatan 2, Stockholm 16 ; Henrik Lindstahls Bokhandel, Odengatan 22, Stockholm ; Gumperts AB, Göteborg.
SWITZERLAND : Librairie Payot, S. A., Lausanne and Geneva ; Hans Raunhardt, Kirchgasse 17, Zurich 1.
SYRIA : Librairie Universelle, Avenue Fouad Ier, B. P. 336, Damascus.
TAIWAN : The World Book Company Ltd., 99 Chungking South Road, Section I, Taipeh.
THAILAND : Requests for FAO publications should be addressed to : FAO Regional Office for Asia and the Far East, Maliwan Mansion, Bangkok.
TURKEY : Librairie Hachette, 469 Istiklal Caddesi, Beyoglu, Istanbul.
UNION OF SOUTH AFRICA : Van Schaik's Book Store (Pty) Ltd., P. O. Box 724, Pretoria.
UNITED KINGDOM : H. M. Stationery Office, P. O. Box 569, London S. E. I.
UNITED STATES OF AMERICA : Columbia University Press, International Documents Service, 2960 Broadway, New York 27, N. Y.
URUGUAY : Héctor d'Elia, Oficina de Representación de Editoriales, 18 de Julio 1333, Montevideo.
VENEZUELA : Suma S. A., Sabana Grande 102, " El Recreo ", Caracas.
YUGOSLAVIA : Drzavno Preduzece, Jugoslovenska Knjiga, Belgrade.
OTHER COUNTRIES : Documents Sales Service, Food and Agriculture Organization of the United Nations, Viale delle Terme di Caracalla, Rome, Italy.

FAO publications are priced in U. S. dollars and pounds sterling. Payment to FAO sales agents may be made in local currencies.

Price $1.00