Nutritional Diseases of Dogs and Cats

BY I. P. EARLE

AS THEY OCCUR under practical conditions, nutritional diseases are more likely to be the result of several dietary deficiencies than of any single one. It is well to know the characteristics of single vitamin and mineral deficiencies, however, as a guide to the proper feeding of dogs and cats and an aid to the diagnosis of nutritional diseases.

The ancestors of the dog and cat hunted food for themselves and their young from the plains and the woods. Since they ate the vitamin-rich organs and viscera of their prey and the mineral-rich bones as well as the muscles, they probably suffered from no nutritional deficiencies as long as game was plentiful and they were skillful and lucky as hunters. Their meat diet was probably supplemented occasionally with a few green leaves, and they had free access to sunshine. The descendants of these hardy ancestors for the most part no longer hunt their own food but remain in the kennel, around the house, or in the house and accept the food provided for them by man. The food may be the scraps from the table, or it may be a mixture of various products of the slaughterhouse and the mill. In either case the adequacy of the diet largely depends on the care and judgment of the human master.

Some metabolic disturbances in dogs and cats are produced by hereditary defects, but many other conditions of this nature are brought about by a deficiency or unbalance of the nutritional factors required by the animal for maintenance, growth, reproduction, and physical activity. Sometimes nutritional deficiencies are secondary to other conditions which interfere with the proper assimilation and

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utilization of nutritive factors, but more often they are the direct result of an inadequate supply of the required factors in the diet.

Although many studies have been made of the nutritive requirements of dogs and of the effects of specific deficiencies, little is known concerning the nutritional requirements or nutritional diseases of cats. However, since the dietary habits of the wild members of the cat and dog families are somewhat similar, it is assumed that the two species are probably subject to similar deficiency diseases. This assumption is supported by what scant knowledge is available concerning the nutritional diseases of cats.

The most commonly occurring errors in the diet of domestic pet animals are deficiencies in vitamins, minerals, and proteins of good quality. Except in the case of rickets, a positive diagnosis of which can be made by means of the X-ray, some information concerning the previous diet of the animal is often essential in diagnosing a disease of nutritional origin. The careful investigations of the laboratory have been concerned chiefly with the effects of deficiencies of single factors, whereas the disease conditions resulting in actual practice from dietary deficiencies are more often the result of multiple deficiencies. Also the characteristic effects of specific deficiencies usually occur in combination with nonspecific symptoms which may be induced by one or more of several causes. The most common of these nonspecific symptoms is the retardation of growth, which may result from simple starvation, inadequate proteins of good quality, deficient or unbalanced calcium or phosphorus, or a deficiency of one or more of several vitamins. Likewise a degeneration of the central and peripheral (outer) nervous system, with resulting incoordination of movements, may occur as a result of vitamin A deficiency or of some type of vitamin B deficiency.

Some of the more common diseases which are fairly easily recognized as of nutritional origin when they occur in dogs are discussed here. Although the discussion shows that the use of pure vitamins, either by injection or by addition to the feed, may be of value in the treatment of a disease condition for the immediate relief of symptoms, the use of a well-balanced diet of natural feedstuffs which contain adequate amounts of the essential factors constitutes the most important treatment for deficiency diseases, as well as the best preventive measure.

**RICKETS**

Any disturbance in the mineral metabolism that results in defective or abnormal calcification, or hardening, of growing bones is usually termed rickets. The clinical indications of rickets include lethargy and listlessness, arched neck, crouched stance, knobby and deformed joints, bowed legs, and flabby muscles. The changes characteristic of defective calcification in the young animal are most marked in the zones of growth of the long bones of the legs—at the junction of the end (epiphysis) and shaft—and at the cartilaginous junction of the ribs. At such points there is a cessation of calcification with an excessive production of cartilage and a deposition of fat. In the more advanced stages the entire bone becomes soft and easily deformed or broken. The development of the teeth is also retarded.
The X-ray is a reliable and practical means of diagnosing early rickets and of determining the severity of the disease and the progress of healing. In the dog the characteristic changes are easily recognized, especially in the lower end of the ulna—the large bone of the foreleg—at the joint which corresponds to the wrist joint in man. In the young animal the calcified shaft of the long bone is separated from its more lightly calcified head by a band of uncalcified cartilage. Normally this cartilage grows and is rapidly calcified at the end of the shaft; as the animal matures the cartilage decreases in width and eventually becomes calcified. In the rachitic animal, however, the cartilage continues to grow but is not calcified, thus increasing the width of the uncalcified band. Changes in the normal calcification of bone may be detected by means of the X-ray at a much earlier stage than that at which clinical evidence of rickets appear.

It is now known that rickets may result from a deficiency of calcium, phosphorus, or vitamin D and that it may be prevented and cured, if it is not too advanced, by the inclusion in the diet of adequate amounts of calcium and phosphorus and enough vitamin D to regulate the absorption and assimilation of the minerals.

The dog's requirements for the three factors calcium, phosphorus, and vitamin D have been studied extensively by Morgan and associates (17, 18, 19, 21, 22). She has demonstrated that in puppies fed on diets deficient in either calcium or phosphorus severe bone deformities result. The conditions produced by the mineral deficiency can be ameliorated but neither prevented nor cured by vitamin D. She has noted, however, that rickets in dogs is usually of the low-phosphorus type, and has ascribed the condition to a faulty utilization of phosphorus rather than to a deficiency of phosphorus in the diet, since low-phosphorus diets are less likely to be encountered than low-calcium diets. The phosphorus in the cereals which frequently form a large part of the dog's ration is not well utilized by the dog.

There is a wide divergence in the estimates made in different laboratories of the requirements of the dog for vitamin D. The requirements vary with the age of the animal, and apparently with the breed, the amount of sunlight the animal receives, and the ratio of calcium to phosphorus, as well as with the content of calcium and phosphorus, and apparently also a number of other factors in the diet.

It is evident that puppies of the small breeds require far less vitamin D for normal bone development than the large, rapidly growing types with heavy bones. It is also recognized that the requirements of puppies raised indoors are far greater than those of puppies with even limited outdoor life and moderate exposure to sunlight. How-

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2 Italics numbers in parentheses refer to Literature Cited, p. 1214.
3 Arnold and Elvehjem (1) found that 5.5 International units of vitamin D per pound of body weight was sufficient to prevent rickets in large-boned puppies on rations containing calcium and phosphorus in a ratio of 1.2:1, whereas when the calcium-phosphorus ratio was increased to 2:1 this amount of vitamin D was insufficient. It has been reported from Morgan's laboratory (19) that with the usual purified ration used there, which has a calcium-phosphorus ratio of 1.7:1, 33 units of vitamin D per pound per day is sufficient for Cocker Spaniels, but severe rickets can develop in German Shepherds on this same level. The Fieschmann laboratories (27) have reported that although 13 units of vitamin D per pound of body weight per day is sufficient for terrier puppies, growing dogs of large breeds may require more than 123 units of vitamin D per pound of body weight per day. The diet that was used in these studies supplied calcium and phosphorus in a ratio of 2.2:1.
ever, the minimum requirements obviously vary widely with other factors in the ration.

Experiments (19) with very large doses of irradiated ergosterol equivalent to 4,545 or more International units of vitamin D per pound of body weight per day for several weeks have shown that a condition of hypervitaminosis D—too much vitamin D—can be induced in the dog. This condition is characterized by vomiting, greasy hair, bloody diarrhea, and deposition of calcium in the arteries and organs. The range between the body requirements and the toxic dose is wide, but the possibility of overdosage should not be overlooked.

The specific treatment of rickets consists in the administration of vitamin D in addition to an adequate supply of calcium and phosphorus. The best sources of vitamin D are fish-liver oils, irradiated ergosterol, irradiated foods, and the action of sunlight on the skin, which enables the animal to manufacture its own vitamin D.

Bone meal at a level of 1 or 2 percent of the ration will usually provide sufficient supplementary calcium and phosphorus for puppies of small breeds. Puppies of the larger breeds should have bones and milk in addition. About 0.5 percent each of calcium and phosphorus (in the mineral form, not as bone meal) in the diet is probably sufficient, however. Arnold and Elvehjem (7) have given as an optimum figure 0.27 percent for calcium and 0.22 percent for phosphorus.

Although the requirements of older dogs for calcium and phosphorus and vitamin D are much less than those of young dogs, a condition called osteomalacia, or late rickets, is sometimes observed in grown dogs as a result of the same kinds of deficiencies that cause infantile rickets. In such cases a softening of the bones leads to lameness and deformity.

**TETANY OF PARTURITION, OR ECLAMPSIA**

Eclampsia occurs sometimes in female dogs and cats shortly before, during, or after the delivery of young. It is apparently the result of a calcium deficiency, possibly associated with a vitamin D deficiency. It is most common in females nursing large litters, in which case the demands of the mother for calcium for milk production are heavy. The symptoms vary in severity from nervousness and mild convulsions to severe attacks which may terminate in coma and death. It has been found that the seizures can be controlled by the administration of calcium, and recurrence is prevented by additions of readily utilized calcium and vitamin D to the diet.

**SCURVY**

Efforts to produce experimental scurvy in the dog with vitamin C-deficient rations have been unsuccessful, and the evidence is that dogs normally synthesize their own vitamin C. Nevertheless, there are occasional reports of a spontaneously occurring condition resembling scurvy which is relieved by the administration of ascorbic acid (vitamin C) or some vitamin C-rich substance such as lemon juice. Collet (4) has described such a condition in young dogs that
had been maintained on a diet low in vitamin C. The symptoms observed were a slight anemia, a swelling of the jaw, and signs of pseudoparalysis (a condition resembling paralysis) of the legs. The condition was completely relieved by the administration of lemon or orange juice. Flohil (9) also described symptoms resembling those of scurvy which occurred in dogs fed entirely on cooked feeds and which cleared upon the administration of lemon juice. A similar report has been made by Jordan (15). Gregoire (11) has ascribed a condition in young dogs which he calls Barlow's disease to a vitamin C deficiency in the ration. The animals became anemic, signs of rickets were present, and there was considerable pain on pressure of certain bones near the joints. Although in good condition, the affected animals remained lying down much of the time. He found a lower than normal excretion of vitamin C in the urine of these animals. Some individuals responded to massive doses of ascorbic acid, and others did not. The condition seems to occur only in young animals and appears to result from a failure in vitamin C metabolism, suggesting a failure in the normal functioning of the animal organism in the synthesis of ascorbic acid, the causes of which are not known.

NUTRITIONAL ANEMIAS

Anemia is a deficiency in hemoglobin, the oxygen-carrying pigment in the blood. The physiological effects of anemia result from the diminished power of the blood to absorb oxygen and remove carbon dioxide and include an increase in pulse and respiration rates, rapid onset of fatigue with exercise, and general weakness. Even a mild degree of anemia is recognizable by the pale color of the visible mucous membranes and by blood examination. Anemias may result from such causes as hemorrhage, internal parasites, chronic infectious diseases, and poisoning, or from a faulty diet. Nutritional anemias occur as a result of diets inadequate in the quantity of the constituents that function in the production of hemoglobin. They are most commonly associated with a deficiency of iron or copper or of the B vitamins, or with long-continued underfeeding of proteins essential for the formation of red blood cells.

Copper itself is not a constituent of the hemoglobin molecule, but it is required by the canine as well as by some other species for the utilization of iron, which is essential in hemoglobin formation. Only minute amounts appear to be needed, however. Potter, Elvehjem, and Hart (24) found that growing dogs made anemic by bleeding responded unfavorably when kept on a diet poor in iron and copper supplemented with 30 milligrams of iron a day, but a rapid regeneration of hemoglobin occurred when the ration was supplemented with 30 milligrams of iron and 4 milligrams of copper. Since copper is required in such small amounts it seems unlikely that the ordinary ration of the dog will ever be deficient in the mineral unless the animal is kept on a diet of milk, which is low in both iron and copper. However, the addition of minute amounts of copper seems advisable when an inorganic iron supplement is used in the treatment of anemia.
An iron deficiency in the dog’s ration is not rare, but the iron requirements of the normal dog are amply met by a ration containing as much as 10 percent of meat scraps or a reasonable amount of fresh meat. The requirements of the bitch are increased during pregnancy, when she may need a more liberal allowance of iron-containing feeds. The normal pup, in common with other sucklings, has at birth a store of iron which is considerably depleted during the suckling period when the principal food is milk. This loss of iron is repaired as soon as iron-containing feeds are added to the diet to supplement the milk. If the pup’s diet fails to include feeds that are good sources of iron, an anemia may result which can be cured by correcting the iron deficiency. Feeds of particular value as sources of iron are liver, kidney, red meat, egg yolk, apricots, peaches, and prunes. Small amounts of inorganic iron (iron oxide or iron citrate) may also be used effectively as sources of available iron.

Another type of anemia results from a deficiency of one of the vitamins of the B complex. The production of anemia in dogs maintained on a blacktongue-producing diet (one lacking in part of the B vitamins) was observed by Spies and Dowling (26). They effected an immediate correction of the anemic condition by the administration of yeast. They suggested as a possible explanation of the multiplicity of symptoms seen in dogs on this diet that several essential dietary factors are present in that part of the B complex which was lacking in the diet. The more recent work of Fouts, Helmer, and Lepkovsky (10), in which an anemia characterized by small, pale red blood cells was produced in dogs maintained on a synthetic diet amply supplemented with other known B factors, has identified the antianemic factor as apparently identical with vitamin B₆ (pyridoxine), the rat antidermatitis factor. Fresh liver, liver concentrates, liver extract, and yeast are most effective in providing the factor B₆, which is required by both pups and older dogs for the maintenance of a normal hemoglobin level.

The treatment of the primary cause of an anemia depends on the factor responsible for the condition, but the treatment of the anemia itself is much the same in all cases. It consists, aside from emergency measures, in the administration in readily available form of ample supplies of the blood-building materials, including proteins of good quality, iron, copper, and certain of the B vitamins. Liver, either fresh or dried, is a good source of all these factors. Medicinal supplements of inorganic iron and copper or of liver extract or liver concentrates can sometimes be used to good advantage, together with feeds rich in good proteins and in the B vitamins. Meat diets have been found to be strikingly more efficient in regenerating the blood of dogs after repeated blood depletion than diets rich in carbohydrates or in which the proteins were largely supplied by milk and cereals (29).

**VITAMIN A DEFICIENCY**

A diagnosis of vitamin A deficiency in dogs is usually based on symptoms that appear in the advanced stages of the disease, when some animals may fail to recover even when the deficiency is cor-
rected. Probably the most easily recognized symptom is a characteristic disease of the eye called xerophthalmia. Many other conditions less specific and often not so easily recognized as of dietary origin are associated with a deficiency of vitamin A. These include loss of weight, rough coat and scaling skin, incoordination of movements, probably lowered resistance to infections, and, in growing puppies, a stunting of growth.

Frequently the diet supplies almost but not quite enough vitamin A to meet the dog's requirements, and a subacute deficiency extremely difficult to diagnose results. Crimm and Short (5) found that the disease in its subacute stages, before the appearance of obvious clinical symptoms, was characterized by a shift in the percentage of immature cells among the white blood cells of a certain type. This they interpreted as indicating a disturbance in the normal formation of white blood cells. Dogs kept for a year on rations in which the vitamin A deficiency was not severe enough to produce loss of weight or xerophthalmia showed changes in the epithelial (surface) cells lining the small bronchial tubes of the lungs as well as changes in the white blood cells.

Experimental work with some other animal species has indicated that one of the earliest symptoms of a vitamin A deficiency is night blindness, or a failure in the adaptability of the eyes to dim light. Although apparently little attention has been given to the effects of vitamin A on the night vision of dogs, it seems likely that the vitamin plays the same role in this species as in the others studied.

While xerophthalmia may develop relatively early in young dogs deprived of sufficient vitamin A, it is indicative of an acute deficiency that develops gradually. First the eyes appear watery and glassy, and the conjunctiva becomes congested. Later the tear ducts become blocked, infection appears, and there is inflammation and edema (watery swelling) of the cornea and finally ulceration. Permanent blindness usually results from xerophthalmia after it has reached the advanced stages of severe degeneration of the cornea. Eye symptoms are usually accompanied by other symptoms of disturbances in the body, such as loss of appetite, unhealthy skin and coat, and a muscular incoordination and weakness that eventually end in paralysis. This condition is the result of the nerve degeneration induced by lack of the vitamin.

Deafness appears to be another manifestation of a deficiency of vitamin A which occurs in young dogs. Mellanby (16) reported in 1938 that puppies fed for some months on diets of natural feedstuffs deficient in vitamin A evidenced incoordination in movements, inattentiveness, and deafness. He found in such animals an overgrowth of bone near the brain in such a position as to stretch the auditory nerve, thereby causing it to degenerate. Poor growth and faulty tooth development are also associated with a deficiency of vitamin A in the pup.

Higgins (14) has reported the experimental production of urinary stones in dogs deprived of vitamin A. There are occasional reports of the finding of kidney or bladder stones in vitamin-deficient dogs on post mortem examination. It has been shown in some other
species that a relationship exists between the formation of urinary stones and vitamin A deficiency, but apparently other factors are also involved. The use of vitamin A therapy in the treatment of urinary stones is a subject for further experimentation.

Fish-liver oil is commonly used as a concentrated source of vitamin A for supplementing the diet as well as for medicinal treatment. Bradfield and Smith (3), however, have reported that puppies utilize vitamin A in cod-liver oil, pure carotene in oil, and carotene as it occurs naturally in carrots and other foods equally well as a source of vitamin A in repairing a deficiency. Carotene, however, is probably utilized less economically than true vitamin A for storage in the body. Feeds that are particularly valuable as sources of vitamin A in the dog's diet are liver, green leaves of vegetables, and alfalfa leaf meal of good quality.

Various estimates of the amount of vitamin A needed by the dog range from 10 International units to more than 360 per pound of body weight. According to suggestions made by Guilbert, Howell, and Hart (12), 80 units (16 micrograms) of vitamin A per pound of body weight per day is a good allowance for dogs. Morgan (19), however, has recommended an amount in excess of 360 International units per pound of body weight per day. In treating a deficiency very large doses can apparently be given with safety.

DEFICIENCIES OF THE B VITAMINS

**Thiamin, or Vitamin B₁, Deficiency**

Spontaneous outbreaks of a disease in dogs which closely parallels beriberi in humans have been reported. Symptoms are a loss of appetite, vomiting, either a diarrhea or constipation, cutaneous edema (watery swelling of the skin), muscular tenderness, and a periodic inability to stand. The symptoms yield rather promptly to injections of thiamin and to the addition to the diet of dried brewers' yeast or some other good source of vitamin B₁.

The antiberiberi factor (vitamin B₁, or thiamin) is required preformed in the ration for the normal growth of young dogs and the maintenance of health in both young and adults. The earliest and most conspicuous effect of a deficiency is a marked loss of appetite, which occurs in dogs more promptly as a result of thiamin deficiency than of any other vitamin deficiency. In general, the symptoms associated in the dog with a deficiency of vitamin B₁ may be grouped as arising either from the failure of normal functioning of the gastrointestinal tract, with a marked loss of tone, or from disorders of the nervous system. The appearance of symptoms varies with the severity of the deficiency and undoubtedly with the extent of complications arising from other deficiencies that may result from decreased food consumption or impaired adsorption and assimilation. The early stages of the deficiency are marked by loss of appetite, fatigue, nervousness, restlessness, and irritability, whereas the advanced stages are characterized by manifestations of polyneuritis (inflammation of
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many nerves at once) such as muscular tenderness, incoordination of movements, and paralysis of the hindquarters.

Although degenerative changes in the nerves have often been demonstrated in association with thiamin deficiency, more recent evidence (8) shows that the nervous symptoms of uncomplicated thiamin deficiency are not due to any actual break-down of tissues in the nervous system. When degeneration of nerve tissues occurs, it may probably be attributed to deficiencies of vitamin A or of other factors of the B complex. The profound nervous disturbances which occur in an uncomplicated deficiency of thiamin and which respond within a few hours to the administration of thiamin are probably produced by disturbances of nerve metabolism rather than by degenerative changes in the nerve tissues themselves.

Thiamin is believed to have a specific role in the carbohydrate metabolism of nerve tissue. The thiamin requirements of the animal are related directly to the nonfat calories of the diet rather than to body weight; hence diets low in fat and high in carbohydrates require a higher level of thiamin than diets high in fat. Arnold and Elvehjem (1) have shown that both growing and mature dogs can be protected from the loss of appetite associated with thiamin deficiency by 360 micrograms, or 120 International units, of thiamin to each pound of a ration low in fat. This supplies 3 to 4.5 units of thiamin per pound of body weight per day for an average-sized mature dog. Therapeutic (medicinal) doses of 10 times this amount or more have been used to give prompt relief from nervous symptoms of thiamin deficiency.

Of the group of B vitamins, thiamin is the most easily destroyed by heat. It has been shown that as much as 80 percent of the thiamin content may be lost during the canning of dog food. Likewise the processing of meat scrap at high temperatures reduces the thiamin content. Grain byproducts from which the germ and outer covering have been removed, as well as all processed foods, are likely to be low in this vitamin.

Thiamin occurs, along with other members of the B complex, in organ and muscle meats (pork muscle is 7 to 8 times as rich in thiamin as beef muscle), yeast, the germ and outer portions of grains, milk, egg yolk, and most fruits.

Fright Disease

Fright disease is adequately discussed in the article by C. D. Stein, page 1193. However, since there have been many reports of a response of the condition to a change of ration and other reports of the immediate relief of symptoms with thiamin treatment in cases characterized by the nervous-running-howling-convulsions complex, the possible relation of the condition to a thiamin deficiency should be mentioned here. Patton (33) has recently observed the production of fright disease in dogs fed a certain commercial feed low in vitamin B1. The nervous condition was relieved within a few hours by thiamin injection. Many attempts have been made to produce the condition on experimental diets low in thiamin, diets low in protein, and
diets containing protein denatured by heat. Arnold and Elvehjem (2) succeeded in producing the symptoms with a diet of meat scrap and wheat which had been heated to 392° F.; the animals failed to respond to thiamin administration.

**Riboflavin Deficiency**

Since a deficiency of riboflavin, or vitamin B_2_, probably never occurs spontaneously in the diet of the dog except in the presence of deficiencies of other B vitamins, the specific effects of a riboflavin deficiency alone may never be observed uncomplicated by other factors except in the laboratory. These effects consist, in the early stages, of a variable dermatitis, or skin inflammation, partial loss of appetite, and loss of weight and, in the acute stages, of sudden collapse followed by death unless treatment with riboflavin intervenes. The dermatitis appears first as a reddening of the skin, followed by a dry scaliness. It occurs usually on the chest and abdomen and the inner surfaces of the legs near the body. In the male it involves the scrotum. In the acute stage of the deficiency the animal becomes apathetic, walks with a staggering gait if it walks at all, and evidences a marked weakness. If no treatment is given the animal soon passes into a coma and dies within several hours, apparently from respiratory failure. Sebrell and Onstott (25) have described a characteristic yellow mottling of the liver and degenerative changes in the central nervous system in such animals.

The injection of 0.34 milligram of riboflavin per pound of body weight into animals in this collapsed state results in prompt recovery if given in time. Street and Cowgill (28) have found that 11.4 micrograms of riboflavin per pound of body weight per day in the ration is sufficient to satisfy the dog's requirements for an extended period.

Riboflavin is widely distributed in plant and animal tissues. It is present in milk, meat, yeast, eggs, and many fruits and vegetables. It occurs in higher concentration in the liver and kidney than in muscle meat. Yeast is an especially rich source. Although it is less sensitive to heat than thiamin, riboflavin is destroyed by high temperatures somewhat above boiling. The content of riboflavin in meat is decreased by frying or roasting.

**Blacktongue and Nicotinic Acid**

Elvehjem, Madden, Strong, and Woolley (6) were the first to report that nicotinic acid will prevent and cure the condition in dogs known as blacktongue. It has recently been shown by Heath, Mac-Queen, and Spies (13) that cats are subject to a similar condition which responds to the same treatment. However, as this disease occurs spontaneously, it is probably the result of a multiple deficiency, since ordinary rations deficient in nicotinic acid are likely to be deficient in other vitamins of the B complex also. The occurrence of blacktongue has been observed most often in those areas of the South where the dog's ration is somewhat similar in character to the
diets producing pellagra in man, that is, where the ration consists largely of such products as corn meal, salt pork, cowpeas, and sweetpotatoes.

The onset of typical blacktongue is characterized by lassitude, loss of appetite, and sometimes vomiting. The mouth gives off a characteristic foul odor. The mucous membranes of the cheeks, gums, and tongue are reddened and inflamed, sometimes showing more or less extensive purplish-red areas where the congestion is most marked. The front end of the tongue is most likely to be affected in this way. The inner surfaces of cheeks and lips may even be covered with pustules and ulcers. Constipation is often noted at the onset of the disease, and a diarrhea develops in the later stages.

If the disease has not progressed too far, recovery may be effected by proper treatment; otherwise, death results. Nicotinic acid or liver extract may be given by injection as an emergency measure. Attention should be given to cleansing the mouth, if this is needed. Aside from these measures, treatment consists in adjustment of the diet to include foods such as fresh liver, dried yeast, beef muscle, wheat germ, eggs, and milk, which are good sources of nicotinic acid and other vitamins of the B complex.

It has been shown that 0.23 milligram of nicotinic acid per pound of body weight per day will cure the symptoms of blacktongue in the dog (7). Half this amount is probably sufficient to give protection against blacktongue, although it will not promote normal growth in young dogs.

Morgan (20) has called attention to the possible dangers of administering large amounts of nicotinic acid in the presence of a deficiency of other factors of the B complex. She has observed that dogs receiving purified rations deficient in both nicotinic acid and the so-called filtrate fraction of the B complex suffer a more rapid decline if nicotinic acid alone is administered than if neither factor is given. In the treatment of blacktongue, care should be exercised to avoid bringing about a condition of unbalance among the various B factors by using nicotinic acid alone without including other factors of the B complex.

**Deficiencies of Other Factors of the B Complex**

The anemia produced by a deficiency of pyridoxin, or vitamin B₆, has already been mentioned.

In addition to thiamin, riboflavin, nicotinic acid, and pyridoxin, dogs require two or more of the remaining factors of the B complex. It has been shown that pantothenic acid is necessary for normal growth and health of the dog, and that filtrate factor W, referred to as the anti-gray-hair factor, is necessary for the maintenance of a healthy condition of the fur and for neuromuscular control. Morgan (20) has demonstrated an interrelation between nicotinic acid, pantothenic acid, and the filtrate factor W and has indicated that there may be other factors of the B complex which have a function in canine nutrition. The requirements for pantothenic acid and the factor W are not yet determined.
LITERATURE CITED

(1) Arnold, Aaron, and Elvehjem, C. A.

(2) ——— and Elvehjem, C. A.

(3) Bradfield, Dorothy, and Smith, Margaret Cammack.

(4) Collet, P.

(5) Crimm, Paul D., and Short, Darwin M.

(6) Elvehjem, C. A., Madden, R. J., Strong, F. M., and Woolley, D. W.

(7) ——— Madden, Robert J., Strong, F. M., and Woolley, D. W.

(8) Engel, R. W., and Phillips, P. H.

(9) Flohil, J.


(11) Gregoire, C.

(12) Guilbert, H. R., Howell, C. E., and Hart, G. H.


(14) Higgins, C. C.

(15) Jordan, Marjorie G.

(16) Mellenby, Edward.

(17) Morgan, Agnes Fay.

(18) ———

(19) ———

(20) ———
(21) Morgan, Agnes Fay, and Garrison, E. Alta.

(22) Garrison, E. Alta, and Hills, Marguerite J.

(23) Patton, John W.


(25) Sebrell, W. H., and Onstott, R. H.

(26) Spies, Tom D., and Dowling, Alexander S.

(27) Standard Brands Incorporated.
1939. A syllabus on vitamin D and methods of supplying it to farm animals. Vitamin D Digest 1: 37-40.

(28) Street, Harold R., and Cowgill, George R.

(29) Whipple, G. H., Roscheit, F. S., and Hooper, C. W.
At the time this book went to press, the drugs and other materials mentioned in various articles—chiefly as disinfectants, insecticides, and anthelmintics—were still available for veterinary and medical use. Under war conditions, however, it is possible that some of these materials may become scarce or unavailable. In that case, the reader should obtain professional advice from the Department of Agriculture, the State experiment station, a local veterinarian, or the county agent as to available substitutes.