Nutritional Diseases of Swine

BY LOUIS L. MADSEN

THE SYMPTOMS, causes, and prevention of the mineral and vitamin deficiencies that affect swine—often with serious consequences for the producer—are discussed in this article, which also includes the results of recent experiments with some of the little-known B vitamins.

The control of nutritional diseases among swine is one of the first essentials in successful pork production. The importance of adequate nutrition is further emphasized by the fact that the cost of feed alone, including pasture, is estimated as making up 65 to 85 percent of the total cost of producing hogs for market. Swine are able to convert a larger proportion of their feed into edible meat and fat than any other farm animal, but errors in diet which slow their growth or lead to poor utilization of feed or the development of nutritional diseases can result in heavy death losses and otherwise seriously interfere with the economical production of pork.

Many nutritional diseases of swine can be readily controlled if feed requirements are understood and the required nutrients are provided. Early recognition of deficiency symptoms and prompt application of curative or preventive measures will frequently prevent heavy losses. A knowledge of the nutritive value of feeds is very helpful in this respect.

Swine are raised under a wide variety of conditions and are fed many kinds of feed. Concentrated feeds make up the bulk of most ordinary rations, but some bulky feeds, such as good pasture plants or a limited amount of well-cured roughage (particularly alfalfa), have proved to be of value as sources of essential nutrients. Pigs

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fed in the dry lot and especially in floored pens depend entirely on their daily allowance of feed to meet their requirements. Such animals are particularly likely to suffer from nutritional diseases unless their ration is carefully selected.

Since this article deals primarily with the nutritional diseases of swine, for additional information on the nutritive requirements of swine and practices in swine feeding the reader is referred to articles in the 1939 Yearbook of Agriculture (7, 31).

**THE MINERAL-DEFICIENCY DISEASES OF SWINE**

**Calcium and Phosphorus Deficiency and the Interrelationship of Vitamin D**

Calcium deficiency is more likely to occur in swine than a deficiency of phosphorus because swine are often fed rations made up largely of grains or grain byproducts, together with some protein-rich concentrates, and these feeds are usually good sources of phosphorus but low in calcium. Vitamin D is closely related to the metabolism of calcium and phosphorus and is therefore considered with the deficiency diseases involving these two minerals.

**Rickets**

Rickets, a disease of young animals, occurring before the bones cease growing in length, is most often seen in winter and early spring when pigs are kept housed because of inclement weather, but it may occur at any time of the year if the animals are improperly fed and not allowed free access to direct sunlight.

The symptoms of rickets in swine (22) are similar to those in other young animals. (See Nutritional Diseases of Farm Animals, p. 323.) Affected animals frequently have a poor appetite and a generally unthrifty appearance, soft, fragile bones, and usually lameness, especially in the advanced stages of the disease. The degree of bone and joint involvement varies; there may be merely a slight stiffness and stilted movements, or the animals may be able only to crawl on their knees; or they may lie on one side, unwilling or unable to get up. The front and hind legs may become bowed, and the joints are frequently enlarged. The pasterns in some pigs become abnormally straight and thickened, while in others they sag until the pigs almost walk on their dewclaws. In standing, the front and hind feet are often brought very close together. The back is usually abnormally arched.

In advanced cases of vitamin D deficiency, blood-serum calcium is usually lowered from a normal value of 10 to 12 milligrams per 100 milliliters to 6 milligrams or less. Blood phosphorus also may be low. At the lower levels of blood calcium, the animals may go into tetanic convulsions, frequently referred to as fits, and sometimes die in convulsions.

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2 Italic numbers in parentheses refer to Literature Cited, p. 826.
Symptoms appear earlier and their onset is somewhat more gradual in young weanling pigs than in older animals. Fattening pigs may "go off their feet" rather suddenly. Such animals usually lose appetite quickly, appear sick, and may have marked incoordination of movement or suffer from partial or complete paralysis. Broken or dislocated leg and pelvic bones or fractured vertebrae resulting in pressure on the nerves and spinal cord may be the cause of these symptoms in some cases. The death rate from rickets is not usually very high, but animals suffering from the disease may succumb to pneumonia or other communicable diseases to which they are predisposed by their weakened condition.

**CAUSE, PREVENTION, AND TREATMENT**

Rickets is usually due to an inadequate intake of calcium or phosphorus or both, or to a faulty proportion of these minerals in the diet, together with a deficiency of vitamin D. A deficiency of vitamin D or of this vitamin and calcium is the chief cause of rickets in swine. Pigs fed indoors on a ration made up largely of cereal grains, without access to palatable sun-cured roughage, direct sunlight, or other source of vitamin D, are therefore very likely to develop this deficiency disease.

An adequate intake of vitamin D cannot compensate for faulty mineral nutrition. The quantitative requirement for vitamin D is lowest, however, when the ratio of calcium to phosphorus lies within the range of 1:1 or 2:1 (2). Too much calcium in the ration interferes with the utilization of phosphorus. An excess in the diet of other minerals, such as salts of iron, magnesium, beryllium, manganese, strontium, thallium, and aluminum, may also interfere with phosphorus assimilation.

Rickets can be readily prevented or cured in the early stages by correcting the diet. Young pigs that have access to direct sunshine store up considerable amounts of vitamin D. It is not safe, however, to depend entirely on stored vitamin D during long feeding periods in confinement unless the pigs are fed in outdoor lots so that direct sunshine is available. When the body stores of this vitamin are largely depleted, rickets will develop. Johnson and Palmer (20) found that colored pigs, when allowed access to fall sunshine before being confined for the winter, stored sufficient vitamin D to protect them from rickets for about 4 to 8 weeks, while white pigs under comparable conditions were protected about twice as long.

The most economical source of vitamin D is direct sunshine, but this may be inadequate in northern latitudes during the winter. High-grade sun-cured legume hay, particularly alfalfa, is a very good source of vitamin D as well as of calcium. Alfalfa of good quality may be fed either free choice in racks or chopped or ground and added to the grain ration at the rate of 5 to 10 percent for fattening pigs and 10 to 15 percent for sows and boars. Artificially dehydrated alfalfa meal is lower in vitamin D than meal made from sun-cured hay. Other hays, such as soybean, lespedeza, sweetclover, red clover, etc., may be used in place of alfalfa, although they are
not usually as palatable. When a good grade of hay is not available for winter feeding and available sunshine is limited, vitamin D may be furnished by such supplements as fish oils, fortified oils, and such irradiated products as yeast and activated sterols of animal origin.

Insuring an adequate calcium and phosphorus intake is as important as supplying vitamin D. Many feeds, including buttermilk, skim milk, tankage, fish meal, and meat scraps, are rich in both calcium and phosphorus. Legume pasture and hay furnish an abundance of calcium, while wheat bran and the protein-rich meals such as linseed, cottonseed, and soybean are rich in phosphorus but low in calcium.

The choice of a mineral supplement, if any is needed, depends on the mineral content of the grain ration. Sources of calcium and phosphorus are often fed with common salt. Other essential minerals such as iodine, iron, and copper may be included in such a mixture when necessary. Some suggested mineral mixtures that can be very economically mixed on the farm according to the individual needs of pigs are given in Table 1. These mixtures may be fed free choice or added to the ration at the rate of 1 to 1½ pounds to each 100 pounds of grain mixture. Additional salt may be given free choice.

<table>
<thead>
<tr>
<th>Mineral supplement needed</th>
<th>Suggested mixture</th>
<th>Ground limestone</th>
<th>Steamed bonemeal</th>
<th>Common salt</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Very high calcium.</td>
<td></td>
<td>75</td>
<td></td>
<td>25</td>
</tr>
<tr>
<td>2 Very high calcium; medium phosphorus</td>
<td>50</td>
<td>25</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>3 High calcium; high phosphorus</td>
<td>33½</td>
<td>33½</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>4 High calcium; very high phosphorus</td>
<td>75</td>
<td></td>
<td>25</td>
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</tr>
</tbody>
</table>

Mineral supplement No. 1 should be used when the ration is low in calcium only. Phosphorus in this case must be supplied by the rest of the ration of whole grains, grain byproducts, and protein-rich concentrates. As previously explained, excessive calcium feeding should be avoided. Mineral supplements No. 2 and No. 3 furnish large amounts of calcium and supply phosphorus in addition. Mixture No. 3 is probably the most widely recommended mineral supplement and has been fed with success to swine of all ages as well as to other farm animals. Mineral supplements Nos. 2 and 3 are appropriate for rations that supply less phosphorus and may also be used successfully in the case of young breeding animals with which the total grain allowance is somewhat restricted to avoid excessive fattening. No. 4 furnishes calcium and more phosphorus than No. 2 or No. 3, but this is not often essential when ordinary well-balanced swine rations are fed. Bonemeal is usually more expensive as a source of calcium than a good grade of limestone or oystershell. Defluorinated rock phosphate prepared for animal feeding may also be used as a source of calcium and phosphorus.
Breeding swine, particularly pregnant or lactating sows, develop serious symptoms if their ration is deficient in calcium. This deficiency is most common in swine fed in floored pens, without access to pasture, on a ration consisting largely of cereal grains without an appropriate mineral mixture. In a study of the effects of calcium deficiency on the general appearance and reproductive performance of sows and gilts, Evans (8) describes this condition as follows:

* * * the main manifestations of calcium deficiency—as typified by all the pigs in the group—were: (1) Rough scaly skins, dirty in appearance. (2) The pigs were periodically “off their feet” with their legs bending inwards, and developed what appeared to be characteristic signs of rickets. (3) The sows often refused their feed towards parturition, this indicating nutritive disturbances. (4) They had great difficulty in farrowing and could not stand on their feet for two or three days afterwards. (5) No signs of milk could be seen before or after farrowing, and their udder was very flabby especially in comparison with the normal sows. (6) Very few pigs survived at weaning time and even the few left made very poor live weight gains. It should be observed, however, that the sows themselves when they farrowed appeared to be in good condition.

Hogan (18) in studying the calcium requirement of brood sows found that their rations should contain not less than 0.4 percent of calcium. Sufficient calcium will usually be furnished if 1 to 1½ pounds of mineral mixture No. 1 (table 1) is added to the ordinary ration made up largely of cereal grains.

Good pasture and legume forage should be included in the ration of the brood sow whenever possible. Well-cured legume hay, particularly alfalfa, may be fed in a rack or ground and added to the grain mixture at the rate of 10 to 15 percent. When the ration of the sow contains such feeds as fish meal, tankage, buttermilk, and skim milk in liberal quantities, the intake of calcium and phosphorus is usually adequate, and it is unnecessary to supply these minerals in a special supplement.

As already noted, phosphorus is less likely to be deficient in ordinary swine rations than calcium (the reverse of the situation with cattle). When swine are grazed on pasture or on cultivated root crops with little or no grain and no protein-rich or phosphorus-rich mineral supplement, however, phosphorus deficiency may develop. Aubel, Hughes, and Leinhardt (7) fed swine on low phosphorus rations and reported the following symptoms:

* * * (1) A loss of appetite, (2) a poor utilization of feed and storage of energy, (3) a failure to make normal growth and to develop bone and muscle normally, (4) a lowering of inorganic phosphorus in the blood, and (5) a marked increase in thirst and a corresponding excretion of urine.

The increased water intake and excessive urine output were associated with enlarged kidneys, which were pale in color.2

2 On histological examination these “large white kidneys” showed “chronic diffuse nephritis of the parenchymatous type, and presented widened glomerular spaces around the glomerular tufts and also widely distended uriniferous tubules with flattened epithelial cells. A granular debris was present in some of the tubules.
Under the conditions of the experiment, the phosphorus intake was adequate when the ration contained at least 0.27 to 0.3 percent of phosphorus.

Ordinary feeds that are good sources of phosphorus for swine are wheat bran, wheat middlings, and especially the protein-rich concentrates such as tankage, fish meal, and soybean, linseed, cottonseed, and peanut meals. Dairy byproducts, such as skim milk and buttermilk, are good sources of phosphorus as well as of calcium. When phosphorus-rich feeds are used in the ration, it may not be necessary to supply additional quantities of phosphorus. In cases where additional phosphorus is needed, mineral mixtures Nos. 2, 3, or 4 (table 1) may be offered the pigs in a self-feeder. Too much calcium in the ration will adversely affect the utilization of phosphorus.

**Nutritional Anemia of Suckling Pigs**

Nutritional anemia is one of the principal causes of unthriftiness and death among young swine. It is particularly prevalent in pigs farrowed in late fall, winter, and early spring which, because of inclement weather or for other reasons, are kept confined to floored pens without access to soil or vegetation. The condition usually appears in pigs 1 to 6 weeks of age, with a somewhat higher incidence about the third week of life. Affected pigs show paleness of the skin and the mucous membranes, lack of vigor, sluggishness, failure to gain normally in weight, and unusual fatigue and labored breathing (thumps) on the slightest exertion. Young pigs may be severely anemic and yet appear fairly well developed and fat. Some die suddenly; others linger for several weeks, lose condition, and become emaciated. Post mortem examination reveals watery blood, excessive fluid in body cavities, grayish-yellow, mottled liver, and marked paleness of the muscles, lungs, and kidneys. Pneumonia and digestive disturbances may also occur. Swine that have recovered from anemia frequently show scattered areas of fibrosis—formation of fibrous tissue—in the liver, due presumably to a healing and replacement process following damage during the anemia.

The milk of all animals is extremely poor in iron and copper, and since young swine are born with a limited store of these minerals, a regular supply is needed shortly after birth to meet the needs of increased blood formation and rapid growth. Soil and forage normally supply sufficient quantities of iron and copper, but newborn pigs kept in pens with surfaced floors or in paved lots have no opportunity to get these minerals. Anemia may also be caused by intestinal parasitism or may accompany severe disorders such as necrotic enteritis, but in these cases factors other than primary iron and copper deficiency are involved.

Several methods have proved successful in the control of anemia. Putting the sow and pigs out to pasture as soon as the weather and other conditions are satisfactory is a good way to prevent anemia. A few shovelfuls of sod or soil taken from areas not traversed by swine (to limit parasitic infestation) placed in the pen or lot may
also provide ample protection, but it may be better to enrich the soil with iron and copper salts. Willman, McCay, and Morrison (29) were successful in preventing anemia in confined suckling pigs by treating them with a saturated solution of dried ferrous sulfate (copperas, 1 pound per quart of water, dissolved as completely as possible with hot water in the following ways: (1) Drenching the pigs once a week until they are 4 or preferably 6 weeks old. (2) Drenching them twice a week until they are 4 weeks old. (3) Swabbing the udder of the sow once daily until the pigs are 6 weeks old.

One-third of a teaspoonful of the solution should be the maximum dose for pigs under 1 week of age. This dosage may be increased until the pigs are receiving 1 teaspoonful when they are about 3 or 4 weeks old. Drenching or swabbing, preferably the latter which is usually the most convenient method of controlling anemia, should be started when the pigs are 3 or 4 days old. It has been observed that too large doses of the iron solution given as a drench to young pigs will cause scours and occasionally vomiting. Treatment may be discontinued whenever the pigs are turned out to pasture. Pigs older than 6 weeks usually are eating enough supplemental feeds to prevent anemia. Extra iron need not be provided in salt mixtures for adult swine except in areas where the soil is deficient in this mineral.

**IODINE DEFICIENCY AND THE BIRTH OF WEAK PIGS**

A deficiency of iodine in the ration of pregnant sows leads to the birth of weak offspring. The young pigs may be stillborn (fig. 1), or they may be born alive and die shortly afterward (28). The symptoms and prevention of this disease in swine and other farm animals are discussed in the article on Nutritional Diseases of Farm Animals, page 332.

**MANGANESSE DEFICIENCY AND LAMENESS IN PIGS**

Millet and coworkers (24) describe a lameness in pigs fed rations consisting of yellow corn, tankage, soybean meal, ground alfalfa, and
salt. The condition is manifested by stiffness, enlarged hock joints, and crooked legs, as in rickets, but adding calcium and phosphorus or various sources of vitamin D is ineffective in treatment. In initial experiments, manganese sulfate added to the ration at the rate of 50 to 60 parts of manganese per million was effective in preventing the lameness but not in curing it once it was established. Wheat and oats are usually higher in manganese than corn.

**TOXIC MINERALS**

**Fluorine and Selenium Poisoning**

Fluorine and selenium poisoning of swine and other livestock is discussed in the article on Nutritional Diseases of Farm Animals. Kick, Bethke, and Edgington (31) found that pig rations containing 0.03 percent or more of fluorine derived either from rock phosphate or sodium fluoride were harmful.

Symptoms of fluorine poisoning in swine include slow growth, low feed consumption, stiffness, marked changes in the bones and teeth (fig. 2), increased thirst, and kidney injury. The bones become thicker than normal, break more easily, and are rough, white, and

![Figure 2](image-url)

**Figure 2.**—*A*, Thickened jawbone, the result of the consumption of too much fluorine in raw rock phosphate. *B*, A normal jaw, shown for comparison. (Courtesy R. M. Bethke, Ohio Agricultural Experiment Station.)
lusterless. The mandibles (jawbones) are also enlarged and rough.

Swine suffering from the toxic effects of fluorine may improve considerably if the source of fluorine is eliminated from the ration, but they may be permanently injured to some extent.

Moxon (25) states that hogs seem to be even more susceptible than cattle and horses to selenium poisoning.

THE VITAMIN-DEFICIENCY DISEASES OF SWINE

Vitamin A Deficiency

Vitamin A deficiency occurs most commonly in swine fed in dry lots on rations of grain other than yellow corn, without access to green feed, well-cured forage, or other sources of carotene or vitamin A.

One of the earliest indications of the deficiency is night blindness, although Guilbert, Miller, and Hughes (10) found that in experimental vitamin A deficiency of swine some of the animals developed partial posterior paralysis before defective night vision had been demonstrated. Hostetler, Foster, and Halverson (13) found that pigs suffering from advanced vitamin A deficiency would easily become excited and fall over on one side in a spasm or convulsion, roll their eyes, struggling a little or lying with legs extended, squeal as if in pain, and give evidence of labored breathing. The animals also had a watery discharge from the eyes and were partly or completely blind. Other symptoms are loss of appetite, diarrhea, unthrifty appearance, staggering gait, and in the later stages complete incoordination of the hind limbs due to degeneration of the nerve and spinal cord. Gilts or sows usually show considerable irregularity in breeding behavior. Some fail to breed entirely, while in others, according to Hughes, Aubel, and Lienhardt (19), the heat periods are more frequent than normal and of longer duration. They state:

* * * The vulva of some of the gilts in the later stages of the disorder remained swollen as though in continuous estrum. * * * The ovaries of these sows on examination showed the unusual condition of containing at the same time, ripe Graafian follicles, freshly ruptured follicles, and both new and old corpora lutea.

If a sow moderately deficient in vitamin A is bred, it may abort, or a resorption of the fetus may take place. In some cases the young may be born at the regular term but may be either dead or so weak that they die shortly after birth. In such cases the sow frequently fails to lactate normally. Hale (11) has made a series of interesting observations on embryonic injury to swine due to maternal vitamin A deficiency previous to and during the early period of gestation. He found various eye defects and abnormalities, including absence of eyes. There were various developmental defects, such as accessory ears, subcutaneous cysts, harelip, cleft palate, faulty kidney development, and malformed hind legs. (Fig 3.)

The value of green pasture or soilin crops (rape, soybeans, field peas, alone or in combination with cereal grains, etc.) for sows and young growing swine cannot be overemphasized. On these feeds the young feeder pigs or breeding animals store up a considerable amount of vitamin A in their bodies, and these stores are utilized to good
Pigs born to sows that have been fed rations deficient in vitamin A may have various developmental abnormalities, are usually weak at birth, and often die. 

A. A portion of a litter of 10 pigs all born without eyeballs. The pig on the left has a cystic enlargement on the head. 

B. A pig with a double cleft lip. 

C. A pig with a cleft palate. 

D and E show an eyeless pig with extra earlike growths. (Courtesy of Fred Hale, Texas Agricultural Experiment Station.)
advantage later during periods of dry-lot fattening or in winter, when the supply of carotene-rich feeds is limited.

Well-cured, green, leafy legume hay, particularly alfalfa, is an excellent source of carotene and prevents acute vitamin A deficiency among swine in dry lots. If the hay is of good quality, a sufficient amount may be eaten if fed in racks; otherwise, 5 to 10 percent of alfalfa meal should be added to the grain ration of young fattening swine or 10 to 15 percent to the ration of breeding animals. When yellow corn is the principal grain fed, it can usually be depended on to furnish enough vitamin A for fattening animals, but there is still some question whether the amount needed for reproduction will be sufficient when furnished by yellow corn only. If a good grade of hay or alfalfa meal is not available, vitamin A may be furnished by cod-liver oil or some other fish oils. Other feeds rich in carotene are yellow sweetpotatoes and yellow carrots. Swine suffering from vitamin A deficiency will usually respond very quickly when the vitamin is supplied in adequate amounts, but animals with advanced symptoms such as marked muscular incoordination or blindness seldom recover completely.

**Thiamin (Vitamin B₁) Deficiency**

Symptoms of thiamin deficiency have recently been produced in swine by feeding experimental rations, but no direct evidence is available to indicate that pigs given ordinary feeds suffer from a deficiency. It is recognized, however, that the requirement for thiamin is very high for reproduction and lactation, though little attention has been given to this problem in the case of swine.

Hughes (14, 15) lists decrease in appetite with a tendency towards leg weakness, a lowering of body temperature, decreased respiratory rate, and, on autopsy, flabby heart with some changes in the digestive tract as characteristic symptoms in pigs on diets deficient in one or more vitamins of the B complex. Van Etten, Ellis, and Madsen (27) produced thiamin-deficiency symptoms by feeding nurslng pigs diets treated with sodium sulfite and sulfur dioxide to destroy the vitamin. They found that after several weeks on the diet the young pigs started to refuse food, vomited occasionally, became emaciated, and had a marked lowering of the body temperature (fig. 4). Death usually occurred within 5 weeks unless thiamin was added to the diet. On autopsy, a flabby heart and liver damage were often found. Some individuals have increased amounts of fluid in the body cavities and also pathological changes in the intestine. Histological (cell) changes were reported in the heart and liver. Electrocardiographic studies recently made in the Animal Nutrition Division, Bureau of Animal Industry, revealed a lowered heart rate and abnormalities of conduction in the heartbeat in thiamin deficiency.

Thiamin deficiency probably never occurs if the diet contains a liberal amount of whole cereal grains or if the animals have access to pasture or well-cured legume forage. Other feeds rich in thiamin are mill byproducts (such as wheat bran), yeast, and legume seeds (such as beans, peas, and peanuts). Increasing the fat in a thiamin-deficient ration lowers the requirement.
Riboflavin deficiency has been produced experimentally by Hughes (15) by feeding a diet low in this factor. He describes the symptoms as slow growth, frequent scours, and walking with difficulty owing to a crippled condition of the legs. Feeding a source of flavin slightly improved the growth and well-being of the pigs, but no improvement was noted in their walking ability. Work in the Bureau of Animal Industry confirmed the fact that riboflavin is essential to swine. Nursling pigs fed a riboflavin-deficient diet failed to grow normally, vomited frequently, had severe diarrhea, developed a rough skin and hair coat, had considerable secretion about the eyes, developed a peculiar gait, became very sick, and died (fig. 5).

Hughes (16) reports that the minimum daily requirement of riboflavin for young growing swine is between 1 and 3 milligrams per 100 pounds of live weight. The value of dairy byproducts such as skim milk, buttermilk, and whey in swine feeding is probably partly due to their high riboflavin content. Yellow corn, an important swine food, is a fair source of riboflavin. Wheat and barley are lower in this vitamin.
Nicotinic Acid Deficiency and Necrotic Enteritis in Swine

When nicotinic acid therapy for blacktongue in dogs was announced in the United States, Chick and associates (3) in England had two young pigs that had developed a characteristic dietary disease from having been maintained on a Goldberger (pellagra-producing) corn diet. The principal symptoms were severe diarrhea, refusing food, dirty-yellow skin, severe scabbery dermatitis (skin inflammation), loss of hair, and a muscular difficulty in the use of the hind legs. Two intramuscular injections of 100 milligrams of nicotinic acid in one pig and three injections in the other at 3-day intervals, followed by a daily allowance of 60 milligrams in the food, produced a striking effect: The appetite returned within 24 hrs. of the first injection, the diarrhea abated and they began to increase in weight * * * The scabs on the skin began to be detached after 1 week, leaving clean, healthy skin. At the same time, the colour of the animals improved gradually and after 6 weeks of treatment was that of healthy pigs.

At the time of this report, about 2 years’ work at the Michigan station, started by Madsen and continued by Davis, was reported briefly by Gardner (9). This preliminary note pointed out that in certain feeding experiments swine developed intestinal disorders resembling necrotic enteritis and that supplementary feeds, such as raw liver, brought about remarkable recovery from the disease. Barley also had a preventive and curative influence. In some cases nicotinic acid was just as beneficial as liver. The Michigan work, later published in detail (5), suggests that necrotic enteritis is a secondary complication caused by intestinal invasion by organisms following nicotinic acid deficiency in the pig. It was pointed out, however, that necrotic enteritis probably involved additional dietary factors.

Common swine feeds, such as corn, milk, and milk byproducts and soybean and linseed meals, are relatively low in nicotinic acid. Peanut meal, however, is reported to be high in this factor. Barley is usually higher in nicotinic acid than corn, while wheat and oats have intermediate amounts.

In the Michigan experiments (5) more cases of necrotic enteritis developed on the basal ration of corn than when barley was fed.
Supplementing the corn ration with fresh liver (200 grams daily), yeast or liver meal instead of tankage, or changing the grain ration to barley often resulted in curing the condition. It was also found that it was necessary to give as much as 100 to 300 milligrams of nicotinic acid a day in order to prevent or cure the symptoms of necrotic enteritis which were attributed to nicotinic acid deficiency. Successful use of nicotinic acid to treat this condition in swine under farm conditions was reported by the Michigan workers and also by Madison and coworkers (23) at the Pennsylvania station.

Because of the widespread losses of pigs due to necrotic enteritis and similar conditions, more work needs to be done to define the relationships of diet and infection as causes of this disease more accurately, as well as to obtain additional information on the content of protective factors in available swine feeds.

**Pyridoxine (Vitamin B₆) Deficiency**

Chick and associates (4) fed young swine on a purified diet and noted that the animals developed typical epileptic fits and a microcytic anemia (characterized by small red blood corpuscles) if they were deprived of the fraction of yeast containing vitamin B₆. Van Etten, Ellis, and Madsen (27), in studies on the thiamin requirement of swine, noted convulsions in swine fed autoclaved (heated) diets, and in later studies of the Bureau of Animal Industry, convulsions and anemia were found to occur in young pigs on both autoclaved and synthetic diets which did not contain crystalline pyridoxine. This deficiency is not apt to occur under farm conditions unless the swine are fed a restricted diet low in this factor or unless the vitamin has been destroyed by unusual treatment of the ration.

Pyridoxine is of little interest at this time in livestock feeding because of its wide distribution in natural foods, including the cereal grains and legumes. Yeast and glandular tissue such as kidney and liver are also good sources. This factor is synthesized by bacteria in the digestive tract of ruminants but must be present in the diets of chickens, dogs, and swine.

**Pantothenic Acid Deficiency**

Pantothenic acid was first recognized as a growth-promoting factor in yeast. It has now been established as an essential vitamin for several species of animals. Dry-heat treatment of feed readily destroys the factor. Bacteria synthesize this vitamin in the digestive tracts of ruminants, but chickens, dogs, swine, and possibly some other animals require the factor preformed in the diet. Experiments in feeding purified and heat-treated rations at the Beltsville Research Center indicate that young swine on rations deficient in this factor lose their appetite, grow slowly or stop gaining in weight for a time, then lose weight and die (fig. 6). Other prominent symptoms of this deficiency are diarrhea (frequently bloody), loss of hair, weakness and incoordination in the use of the legs, and the formation of a brownish-red incrusted material on the eyelids. Microscopic exam-
ination of the nerves and spinal cords from pantothenic-acid-deficient pigs has revealed degeneration of the myelin (sheath) of the nerve fibers similar in all details to that found in pigs that developed incoordination on natural feeds and heat-treated rations (6), as described in the following section of this article.

Hughes (17) and Hughes and Ittner (18) have also studied pantothenic acid deficiency in swine, and they suggest that the minimum requirement for this factor fed as calcium pantothenate lies between 7.8 and 11.8 milligrams daily for each 100 pounds of live weight.

**Locomotor Incoordination in Swine Due to Nerve Degeneration**

A varying number of pigs fed in pens on rations that have heretofore been considered nutritionally complete—such as yellow corn and trio protein mixture (tankage, linseed meal, and alfalfa), with a supplementary mineral mixture—develop incoordination in the use of their legs. This disease was probably first described by Wehrbein in 1916 at the Iowa State College as posterior paralysis and incoordination in movement, associated with degeneration of the sheath of various nerves. Communications to the Bureau of Animal Industry indicate that the disorder is of serious economic importance and is more prevalent during some years than others. It has occurred in the Bureau's herd at the Beltsville Research Center, Beltsville, Md.

Recent work on the problem by Ellis and Madsen (6) has shown that feeding normal diets that have been subjected for 30 to 40 hours to dry heat at a temperature of 115°-120° F. greatly increases the incidence of the disease and the severity of myelin degeneration in the nerves and spinal cord (fig. 7). Supplemental or replacement feeds fed with the heated diet have given some encouraging results. Barley,

**Figure 6.**—Both these pigs were fed the same purified diet except that the pig on the right did not get a supplement of pantothenic acid. This animal has a poor appetite, is weak and emaciated, and suffers from severe diarrhea.
oats, liver, and concentrated milk products have given the most protection. Tests with many other feeds plainly indicate that the dietary factor or factors involved are present in variable or insufficient amounts in many of the common swine feeds. Further work with purified and natural-food diets at the Beltsville Research Center, together with the work of Wintrobe and associates (30) and the recent report by Hughes (17), indicates that pantothenic acid deficiency is probably one of the causes if not the main cause of this disease.

**ACUTE HYPOGLYCEMIA IN NEWBORN PIGS, OR SO-CALLED BABY PIG DISEASE**

For a number of years the Illinois Agricultural Experiment Station has been investigating a highly fatal disease of newborn pigs. Sampson, Hester, and Graham (36) describe the symptoms of this condition as follows:

In the typical syndrome apparently normal pigs varying from approximately 24 hours to 72 hours old or slightly older show symptoms of shivering, dullness, and anorexia [loss of appetite]. The animals have a tendency to isolate themselves and burrow under the bedding. **Co**

Coincident with the loss of appetite and onset of weakness, the hair coat becomes rough, the skin cold and clammy, and the heart action slow and feeble. Finally the pig lapses into coma. Death of several or all pigs in the litter often occurs within 24 to 36 hours after the first symptoms are manifested.

These investigators report that the affected animals have a marked lowering of the blood sugar, or hypoglycemia, and indicate that the symptoms are probably due to this feature of the disease. In experimental work, baby pigs were found to be highly susceptible to a lowered blood sugar caused by fasting up to about 24 hours after birth. Affected pigs have been successfully treated in the early stages of the disease by repeated intraperitoneal injections of glucose supplemented by hand feeding of sugar solution or milk. Suggested preventive measures include feeding sows and gilts a liberal ration throughout pregnancy and not reducing the amount of grain fed during the last few weeks of the gestation period as commonly practiced. Newborn pigs should be closely watched for the first few days to be sure that they are nursing properly, that the sow has sufficient milk, and that the young pigs do not become chilled. If symptoms develop, hand feeding should be begun immediately.
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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.