Poultry Pathology

Nutritional diseases of poultry

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THIS group of disorders comprises dis-Lease conditions in which infections or transmissible agents cannot be demonstrated and which are often benefited by minor changes in the ration. According to statistical data the percentage of cases falling under this heading is comparatively high, a fact which is surprising as feeding of poultry is fairly well standardized. It is not definitely known to what extent factors of management, such as brooder temperature, space, and ventilation influence the appearance of nutritional diseases. Our ignorance of the importance of congenital factors in the development of nutritional maladies is often cloaked under such expressions as "weak stock" and "lack of vitality" which cannot be determined by field or laboratory diagnosis.

Nutrition in Heredity

Not a great deal is known regarding the influence of nutrition of the parent stock on the livability of chicks because most experimental study along this line has ended with ascertaining the hatchability of eggs. It has been quite definitely demonstrated that nutrition of the parent stock has a direct influence on the occurrence of early rickets in chicks. How far this may hold in other early nutritional disorders constitutes a fruitful field for further inquiry.

Certain types of nutritional disorders in poultry are known which are the effect of experimental rations, but it is not always certain that the conditions observed in the field and in experimental birds are exactly comparable. Two rations may contain the same per cent of crude protein, carbohydrates, fats, etc., yet one may cause serious nutritional disorders while the other does not. The only feasible way of comparing two rations is by controlled feeding trials under similar conditions of housing and management.

Diagnosis

The practicing veterinarian is concerned chiefly with symptoms, lesions, differential diagnosis, methods of prevention and control, and practical sources of the various nutritive elements responsible for the deficiency disorders. The diagnosis of nutritional disorders in poultry in the field or in the laboratory must be arrived at by a process of excluding infectious diseases and other factors as possible causes for the losses. The evidence may be entirely negative in nature or may consist of the imprint which faulty nutrition has left upon the body. Diagnosis is mostly indirect in character and is subject to error. It is to be remembered that varying degrees of nutritional deficiencies may occur; and furthermore, individuals rarely behave in a prescribed manner. It is only the exceptional cases which develop all phases of the typical symptom syndrome.

Vitamin A Deficiency or Nutritional Roup

This is a disease that usually appears in chicks at approximately the same age as

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rickets. It is characterized by an unsteady gait and mild colds generally followed by appearance of swellings about the head. The eyes are apt to become involved and show a deposit of a yellow cheese-like material in the conjunctival sac with some cloudiness of the eye. The eyelids become glued together and are generally free from inflammation. Many birds show follicles in the esophagus and degenerative changes in the kidneys. Thus, it is advisable to inquire into the ration fed when young chicks or poults appear to be suffering from roup.

In the Chick

The initial symptoms, failure to grow and development of an unsteady gait, usually appear in chicks at about four or five weeks of age and sometimes later. This is chiefly due to a deficiency of reserve vitamin A which the young chick carries over in the egg yolk, from which it was hatched. The amount of vitamin A fed to the hen will determine the amount of vitamin A reserve in chicks to a large degree.

In the Hen

In laying hens, deficiencies of vitamin A cause a cessation in egg production, nutritional roup, and death. The common lesions produced are emaciation and the characteristic lesions are small white ulcerative and raised areas in the mouth and extending down the esophagus. These lesions somewhat resemble canker, but are confined to the digestive tract rather than the respiratory tract. Under practical conditions variations of vitamin A deficiency from mild to severe may exist. In marginal cases there may be no external symptoms, but the birds will be more susceptible to common colds and respiratory disorders. Turkeys are more susceptible to vitamin A deficiencies than are chickens.

In cases of vitamin A deficiency, the only requirement is to supply the element lacking in the diet. The principle sources^{*} of viamin A are fresh green feed, dehydrated alfalfa, cod liver oil or other fish oils, yellow corn and egg yolk.

Polyneuritis

This is a deficiency disease due to the absence of vitamin B_1 . The symptoms are staggering, loss of appetite, cessation of growth, retraction of the head with the birds turning cartwheels and doing all sorts of contortions, cyanosis, dypsnea, and finally death. Atrophy of the lymphoid tissue and enlargement of adrenals, thymus and testes may be observed.

Recent work has established vitamin B_1 , chemically known as thiamine hydrochloride, to be necessary for the prevention of polyneuritis in poultry. There is very little storage of vitamin B_1 in the animal body and the amount in the egg yolk is greatly dependent upon the diet of the hen. Therefore, this deficiency may be expected to appear earlier in chicks when the diet of the hen was lacking in vitamin B_1 . Symptoms usually appear in the second and third week after hatching.

Vitamin B_1 is present in the germ and bran or outer coats of grains, green feed, and milk. Brewers yeast is the greatest source and wheat germ ranks second. Fortunately the wide distribution of vitamin B_1 in nature makes this deficiency improbable in most poultry rations.

The disorder also occurs in turkeys.

Nutritional Paralysis

This is a condition that makes its appearance betwen the tenth and twenty-first day and gives rise to typical symptoms in that the long toes curl forward. Later birds squat and walk on their hocks and become completely paralyzed in a sprawling position with the toes curled inward. The internal organs remain normal and no pathologic changes are noted in the nervous tissue. The exact nutritional factor is not definitely known, but it is thought to be part of the vitamin B complex.

The disease may be treated and prevented by liberal amounts of milk products, dried brewers yeast and alfalfa meal. Fresh skim milk has given good results. This condition should not be confused with

^{*} See "Feeding for Hatchability" by Dr. Wilcke for vitamin A requirements. Page 73.

so called range paralysis.

Dietary Dermatitis (Pellegra like disease)

This is a disorder that has been observed in chickens and more frequently in turkey poults. The symptoms of dermatitis in poults consist of a sore mouth, encrustations at the corners of the mouth, diarrhea resulting in an inflamed encrusted vent, and encrustations which may appear on the legs and skin of any portion of the body. The eyelids become thickened and stick together. The feathers become ruffled and the birds in general have a listless, unthrifty appearance. This condition has been found to be due to a deficiency of vitamin G which contains at least two factors essential to the life of poults. The first is called flavin or lactoflavin and poults deficient in this develop symptoms of dermatitis as described above. Feed stuffs that are good sources of flavin are fresh or dried greens, alfalfa meal, dried milk and dried whey. The plant leaves are the richest source of flavin in plants.

The second of the vitamin G group is a vitamin which prevents dermatitis in chicks. It is known as the filtrate factor or pantothenic acid. It will not prevent dermatitis in turkeys, but if poults are put on a diet deficient in this factor they fail to grow and die in a short time. Cane molasses is a good source of this vitamin but should not be fed in excess of 5 per cent because of its laxative action. Wheat germ, fresh kale, and corn are sources of this vitamin.

Rickets

This disorder is due to insufficient mineralization of the bones. Under normal conditions fixation or deposit of the necessary minerals, especially calcium and phosphorous goes on under the influence of vitamin D. Absence of this vitamin results in leg weakness, crooked breastbones, enlarged joints, beaded enlargements on the ribs, failure of bones to calcify so they remain thick, soft and rubbery, low calcium and phosphorous levels in the blood, enlargement of parathyroids, and cessation of growth. The chicks lie about in a sprawled attitude and finally succumb in a state of emaciation. If the parent stock received plenty of vitamin D the chicks are resistant to the disease for about three weeks after hatching. Otherwise rickets may appear as early as the second week. Statistical data shows that most cases occur at about six to eight weeks. In mature birds egg shell texture is poor, production decreased and hatchability is very low. The first evidence in laying hens is thin egg shells, soft shelled eggs and cessation of production.

Vitamin D, and Ca and P

Vitamin D is concerned directly with mineral metabolism, controlling the calcium and phosphorous equilibrium in the blood stream and in the bone. While vitamin D is essential, the individual needs depend upon the amount and the ratio of calcium and phosphorous. No amount of vitamin D will compensate for deficiencies in these minerals. Thus, vitamin D deficiences may develop in birds with adequate vitamin D if the calcium and phosphorous ratio or amounts are low. The chick requires a minimum of .75 per cent calcium and .50 per cent phosphorous for normal calcification.

The common source of vitamin D for poultry is direct sunlight. In fall and winter there is not enough direct sunlight to supply their needs and other sources must be used. To correct or prevent vitamin D deficiency or rickets fish oils such as cod liver or sardine oil are used. They are generally used in the mash in 1 or 2 per cent concentration.

Nutritional Encephalomalacia

(Crazy chick disease)

This is a disease occurring rather frequently in the eastern states and in a few isolated incidences in the Middlewest. It generally occurs in chicks from two to eight weeks old. It has been observed in

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has been overcome, the head of the femur will drop into the acetabulum. This will be readily discernible with the left hand which has been kept over the acetabular area.

Classification of the injury is then made to determine the type of fixation, if any, to be employed. Care must be taken at this point to classify the dislocation correctly if the desired results are to be obtained.

Classification of Coxo-femoral Dislocations

Group I. If the head of the femur **snaps** back into the acetabulum and **remains there when tension is released** from the extremity, we know that the case falls in group I or II.

To differentiate between groups I and II we proceed as follows: Placing the right hand lightly on the foot of the affected member, we move the leg in all directions without making any willful attempt to keep the dislocation reduced. We increase the motion until we are satisfied that normal motion will not again cause a dislocation. No splints or other means of fixation are required for the first group.

Group II. The reappearance of the dislocation upon the release of tension and after carrying out the movements described above characterizes the second group.

Fixation used in group II: Flex the leg completely. Place cotton padding from the toes upward over the hock, and then bandage as shown in Figure 1. Keep in mind that all efforts should strive to rotate the leg inward. Therefore, bandage clockwise on the right leg and counter-clockwise on the left leg. Start the bandage at the toes, bandage the lower extremity and then bandage the groin. When good fixation has been accomplished, tape may be applied over the bandage.

It has been our experience that 7 to 10 days of this type of fixation is sufficient to prevent any further dislocation. We favor this kind of fixation because it maintains a good degree of inward rotation, places muscular tension on the trochanter exerting lateral pressure, and permits the patient to move about.

Group III. In this group, we place those cases which will redislocate immediately upon release of the tension after reduction. It is often difficult to determine when reduction has been accomplished in these cases. However, reduction can be accomplished with patience and care.

Fixation, in these cases, must be of the type that will insure pressure of the femoral head into the acetabulum with a marked degree of inward rotation. The spreader cast described by Ehmer* has proven to be al lthat he claimed it to be when used on this group. For brevity here we suggest that the reader carefully pursue Ehmer's description. This spreader cast should be kept on the animal for about 10 days. We do not use it on all cases of coxo-femoral dislocations because it is somewhat uncomfortable.

Years of experience indicate that if the described classification and treatment are followed coxo-femoral dislocations will no longer disturb the small animal practitioner.

* Ehmers, E. E. 1934. Special casts for the treatment of pelvic and femoral fractures and coxofemoral luxations. No. Amer. Vet. 15 (Dec.):34.

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field cases; late hatches may be more apt to come down than early hatches.

In general, symptoms vary from droopiness to muscular incoordination, retraction of the head, somersaults, rotation of the head or rotation in a laterally prone position. The condition is afebrile and of rapid onset.

In most cases gross lesions are confined to the brain. These gross alterations are characterized by edematous enlargement of the cerebellum associated with slight greyish discoloration and minute hemorrhages either on the cut surfaces or the convexities. This may be observed by longitudinal splitting of the head and gross examination of the brain.

The protective factor against encephalomalacia is present in certain edible oils such as corn oil, cottonseed oil, peanut oil,

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and soy bean oil. The factor in these oils is thermostable. The protective factor appears to be vitamin E.

Myopathy of the Gizzard in Turkeys

This condition occurs frequently in turkeys and is characterized by formation of grey areas in the gizzard muscle which are firmer in texture than normal muscle. The lesions are not always conspicuous but may blend into normal muscle. This condition may be controlled or prevented by use of wheat germ oil and other vegetable oils.

Nutritional Myopathy of Ducks

This is a condition manifested in young ducks characterized by their inability to stand, leg weakness, and listlessness. Complete loss of use of the limbs eventually occurs. The mortality is usually high, 35 per cent. The gross lesions are found in the muscle of the gizzard and appear as irregular circumscribed white or grayish areas often elevated above the healthy or brown muscle. Microscopically these appear as liquefaction of the fibers, and necrosis. Skeletal muscles may also be affected principally the leg and back muscles, and the muscles of mastication.

The protective factor for this condition is also present in vegetable oils as for encephalomalacia. Five per cent soy bean oil meal or other vegetable oils will act as protective agents.

Gout

This has been known for a long time as a disease principally of older chickens and turkeys. It is characterized by enlarged pale kidneys covered with a chalklike material; this same chalklike material is found coating the heart muscle, abdominal wall, liver and intestine and occasionally in the joints of the legs or wings. The materials deposited are urates of uric acid.

The cause may not always be determined, but it is generally believed that the condition is due to excessive feeding of protein.

In articular gout the joints of the legs and wings may be involved. The joints are at first swollen and painful. The lesions may form tumor-like nodular growths which may rupture the joint membrane erupting a yellow turbid fluid containing urates. Emaciation, diarrhea and death follow. Visceral gout is discernable only upon autopsy examination. The internal organs are covered with a chalklike deposit of urates. The kidneys are swollen and grey in color.

Heavy or repeated doses of baking soda as used for a flush may produce lesions undistinguishable from gout, especially if previous injury has occurred to the kidneys.

The quantity of meat scrap or other protein should be reduced and replaced by green feed. Epsom salts may be given as a laxative, 1 pound to 100 adult birds in the amount of water they will consume in one day.

Protein Poisoning

This condition occurs in young chicks and poults between the second and twentieth week of life. It occurs most frequently in fast growing chicks or poults.

The common symptoms are lack of muscular control, unbalance, and a peculiar twitching of the head. The kidneys become enlarged, engorged and congested. The exact cause is unknown, and it is recommended that a change to another ration following a milk flush will remedy the condition.

Many cases of protein poisoning are diagnosed in adult turkeys. The tendency for most turkey producers is to feed a ration as high as 26 per cent protein. This amount is near the critical stage of protein assimilation, and in many instances birds fed for proloned periods on such high protein diets develop a severe enteritis and nephritis with a more or less water logging of the kidneys. Reduction of the protein in the mash or addition of 10 to 15 per cent alfalfa leaf meal usually corrects the condition.

Perosis

This condition, slipped tendon, hock disease, or spraddle legs, appears in chicks and poults raised under confinement usually from the fourth week on, but has been

observed in chicks and poults one week after hatching. The disease constitutes a deformity of the leg bones of the tarsal joints in which the bone tissue itself is normal. The shanks are slightly curved and flattened, and the hock joints appear puffy, discolored, and bowlegged. If dissected, one finds the achilles tendons have slipped to the medial or lateral side of the condyles causing the bowlegged appearance of the bird. The disease may be produced by feeding an excess of minerals. especially bone meal, and by deviating from the normal calcium-phosphorous ration of 1.3 to 1 in the mash. Certain evidence suggests the possibility of a specific vitamin factor contained in rice bran.

The control of this condition consists in prevention rather than treatment as the condition cannot be cured after the deformity has taken place. The addition of 4 oz. manganese sulfate to the ton of mash prevents this condition in chickens. Eight ounces manganese sulfate per ton of feed will help to prevent this condition in turkeys.

Fatty Liver

This is a condition frequently seen in in baby chicks, especially when they have died between the fourth and seventh day of brooding. The post mortem examination is essentially negative except for the liver, which is intensely yellow and under the microscope appears flooded with minute particles of fat globules. The unabsorbed egg yolk is small. The exact cause of this condition is unknown, but the pathologic picture suggests an overloading of the liver with fat possibly due to a too rapid absorption of the egg yolk. The small initial losses come to a stop without treatment.

Ulcerated Gizzard

This is a condition frequently occurring in young chicks and is more a lesion than a disease. Many cases are associated with infectious diseases, but it is frequently found independent of infection. There are no symptoms, but birds upon post mortem

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show significant changes in the gizzard. The hard lining cracks, shows furrows and upon peeling it off the underside shows dry bloody ulcers. It has been suggested that this condition is associated with low cholic acid of the bile.

Various feeds, such as dried or fresh green feed, wheat bran, oathulls, and rice bran aid in preventing this condition.

Feather Pulling

This is a vice which may originate from many causes such as: overcrowding, lack of exercise and lack of sufficient mineral or animal matter in the feed. The condition may be prevented by the addition of meat scraps ,or by the addition of 10 to 15 per cent oat hulls to the ration.

In addition to the above mentioned nutritional disorders there are many occurring each year which are as yet unidentified. It is good general practice, when the possibility of infectious diseases can be ruled out and one encounters one or more of these so-called unidentified disorders, to make minor changes in feeding practices. Many times this is all that is needed to correct the disorder.

WINTERMEZZO

The annual formal dance of the Veterinary Division was held on Jan. 24 in Memorial Union. The dance, which was held jointly with the Science Division, was attended by 350 couples.

George Gitz, '42, was in charge of ticket sales, while Joe Sexton, '42, was cochairman of the affair.

Music for the dance was by Charlie Fisk and his orchestra who had previously been featured at the Palladium Ballroom in St. Louis, Mo. Clifford Orton, '42, was in charge of securing the orchestra.

The decorations carried out the "Wintermezzo" theme. Jack Denton, '42, served as chairman of the decoration committee. They were constructed in such a manner that they could be used for the other dances of the quarter.