PROGRESSIVE PARALYSIS OF CHICKENS

Thesis for Degree of M.S.

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1926

THESIS

INTRODUCTION

Since 1921, numerous inquiries have been received at the Michigan State College as to the cause of an affection among chickens occurring at the age of ten weeks to maturity. The disease is commonly termed leg weakness with no attempt to differentiate it from polyneuritis, rachitis and other leg troubles known to occur in chickens. In reality, this malady is one of incoordination and paralysis, and not a weakness of the legs. It involves the legs and toes, and some times the wings and neck, causing a mortality of five to fifteen per cent of affected flocks.

In the review of literature, some material on diseases showing symptoms closely allied to those of progressive paralysis has been used in an effort to differentiate this paralytic disease from other forms of leg weakness.

REVIEW OF LITERATURE

De Marek (1) in 1907, reported a disease of chickens of Budapest, the chief symptom being a progressive paralysis of the legs. Nerves supplying affected parts show considerable changes, often an almost complete degeneration of nerve fibers, and an infiltration with round cells. Similar lesions

were found in the spinal cord, and the pia mater was also found to be infiltrated. De Marek stated that he was unable to determine the cause of this disease.

In 1924, (2) Van der Walle, Winkler and Lumius reported a disease among fowls, the chief symptom being that of paralysis. The appetite remains good, even to the last stages of the disease, and the birds look healthy, as indicated by the bright comb and the carriage of the head. The temperature is found to be normal. Affected birds generally die in a few weeks after the onset of the disease. Diseased birds fed on a ration containing yeast did not recover as did those suffering with polyneuritis as a result of an exclusive rice diet. These investigators confirmed the pathological findings of De Marek. They also found on cross section of affected nerves that the nerve sheath was swollen and degenerated, and cross section through the spinal cord revealed that the ganglion cells had lost their Nissl granules, and many were reduced or degenerated. Extravasation of blood around the blood vessels of affected nerves was common. They believed the degeneration was not a primary condition but the result of the infiltration.

Experiments were performed to ascertain the cause of the disease. Of two birds, one fed the same ration as the affected flock and given water used at the experimental station remained healthy, while the second bird fed the same ration and water from the well from which the affected flock received their supply, became paralyzed after two months. Blood from the affected bird was injected into a normal bird which later developed paralysis. Cultures were made from the blood and passed through a Chamberland filter. The filtrate was injected into a normal bird which showed symptoms after two months. Six birds were kept in the same pen with affected birds and remained healthy. They concluded that the cause of the disease is probably a filterable virus. Considering the pathological findings they decided that the most characteristic name for the disease would be Neuromyelitis gallinarum.

Doctor Jull, senior poultry husbandryman of the United States Department of Agriculture, said in a personal interview, that they were encountering more or less trouble among their experimental birds with a paralytic disease closely resembling the malady generally termed leg weakness or rachitis, and that

the disease could not be controlled by the use of cod liver oil, green feed and sunshine as in the deficiency diseases showing similar symptoms.

The idea of leg weakness in chickens held by Knox and Lamb (4) is that it may be the result of a number of causes. They say, "In some cases it is apparently a nervous condition which results in a lack of coordination in the muscles of the legs. In other cases it is due to a poor development of the bone, which may be referable to the ration and which may in some cases be corrected by feeding bone meal, and which may be a rachitic condition. In still other cases it may be merely a symptom of disease or general condition of malnutrition, in other words merely a sick chicken. In our opinion leg weakness is not a specific disease in chickens referable to definite deficiency in ration, but is a general symptom caused by any one of various disorders, such as a headache in the human family."

Knox and Lamb (5) quote Halpin as saying that it is his opinion that leg weakness is a rachitic condition and that it is prevented by the use of cod liver oil.

Emmett and Peacock (6) and later Halpin, Hart and

Steenback have proved the causual relation exsisting between dietary deficiency and leg weakness. These investigators found that chicks fed from the time they are hatched with a ration of corn and skimmed milk, with few exceptions, cease to grow in four to six weeks and soon die often showing symptoms of leg weakness. Upon adding five per cent cod liver oil to this ration uniform rates of growth were made up to seven hundred grams in weight.

Mitchell, Kendall and Card (7) state, "That whether the beneficial effects of cod liver oil are due to its content of vitamin A or to its content of anti-rachitic vitamin is not known, but cod liver oil is known to be a potent curative in infantile and experimental rickets."

Gwatkin, (8) a Canadian investigator, has reported a form of leg weakness occurring during the late summer and autumn months, when apparently everything necessary to a complete diet is available with sanitary conditions of the best.

Bushnell and Hinshaw (9) describe the symptoms produced in fowls, by a lack of vitamin B (anti-neuritic) and vitamin D (anti-rachitic) as follows: Vitamin B, "In birds the disease is characterized by

extreme nervous symptoms and inability to coordinate certain muscular movements. Sometimes there is a paralysis of the legs and a slight paralysis of the muscles of the neck. The internal organs appear shrunken and darker in color than normal. The muscles also appear to be darkened as if they had been exposed to the air. Symptoms other than those due to emaciation and nervous incoordination do not seem to be marked. The disease develops rapidly and the bird dies in about fifty-four days. Vitimin D (anti-rachitic) the most characteristic symptom of vitimin D deficiency in the diet is that of leg weakness in young chickens. This vitimin is present to some extent in green leaves, milk and in cod liver oil in large amounts. One to five per cent of cod liver oil added to the feed will prevent the appearance of this trouble in young chickens. "

Hart, Halpin, Steenbock and Johnson (10) state, "Leg weakness is rachitis. The chicken, and especially the baby chick, is very sensitive to leg weakness or rickets. This one malady is the occasion of tremendous losses in baby chick rearing. With the recognition that leg weakness is rachitis, a ready cure for the malady is forthcoming. There are certain food materials now recognized as having distinct anti-

rachitic properties, namely, the fish liver oils and hen's egg yolk. It finds its equivalent also in effect in sunshine or in ultra violet light."

Johnson((11) states, "It is well recognized that leg weakness, or going down on the legs, of chickens may be due to a number of causes. Differentiating the various types of leg weakness makes it better possible to apply more scientific measures for relief and also to distinguish between important and unimportant leg weakness troubles. Present knowledge shows that one type of leg weakness is accompanied by rickets, but information relative to the practical dianosis of this condition among chickens is worthy of further mention. While the common leg weakness of young fowls is a problem of lack of mineral deposition in bones, it is at the same time often accompanied by an arrested development of the soft tissues of the body. Examination of young fowls up to about six weeks of age, received at this station, reveals that the great majority of these chickens showing the condition which might be regarded as distinctly a leg weakness disturbance, evidence abnormal bone formation. Leg weakness in growing stock, of this section, older than this is not likely to be accompanied by this abnormal bone formation. One of the most striking

changes noted externally accompanying leg weakness symptoms is a buckling or inward curvature of the ribs at the junction of the vertebral and sternal portions associated with a distinct lack of rigidity of the distal portion of the bones."

Stafseth (12) makes the following statement, "Leg weakness is not, as the name would imply, a disease in itself but merely a symptom which may be seen in a number of diseases such as: tuberculosis, polyneuritis, enteritis, rachitis, articular gout, limber neck, asthenia, and diseases of the liver and perhaps others."

A paralytic disease of ostriches resembling that of chickens was observed by Marx (13), in 1900, in the Frankfort Zoological gardens.

In 1910, Robertson (14) describes a paralysis occurring in ostriches near Cape Town. The symptoms of the disease described are: sudden onset, inability to rise, paralysis of the toes, which are flexed on themselves. The paralysis often involves the wings and neck. The appetite remains good, and there is no apparent pain. Young birds are the most often affected. The only constant lesion observed by Robertson is a congestion of the duodenum. An organism was isolated from the mucosa by ordinary cultural methods. This

organism is aerobic and does not stain by Gram's method. A suspension of the organisms in physilogical salt solution when injected into a normal ostrich produced symptoms in seven days, and death in one month.

From the literature revived in this paper, it will be seen that the followig points appear to be established:

1. There are several separate and fairly distinct diseases often referred to as leg weakness.

2. Vitamins play a very important part in rickets and polyneuritis.

3. Cod liver oil and green feed are potent factors in preventing and curing leg weakness caused by a lack of vitamins.

4. There is a paralytic disease of chickens, commonly called leg weakness, which is not referable to the nutrition, and the cause of which is unknown.

SYMPTOMS OF THE DISEASE

The first symptom of the disease is often a flexion of the toes of one or both feet (Fig. 1) progressing as the disease develops until the entire leg is involved; sometimes the reverse is noted the entire leg showing incoordination. The foot and leg on the opposite side may or may not become involved. In the last stages of the disease one leg may be drawn

forward fully extended, the other retracted posteriorly. (Fig. 2) Thus it appears that the nerves controlling the extensor muscles of one leg and the flexor muscles of the other were the only nerves completely paralyzed. This no doubt accounts for the lack of coordination seen in the early stages of the disease. The wings may become involved, also the neck which may be drawn into an S shaped curve and at times in the last stages is found drawn around until the head is in a ventro-dorsal position. The bird appears alert and has a good appetite for weeks and even months if well fed, unless weakened by infestation with coccidia, worms or other parasites which are often found in tremendous numbers in the last stages of the disease; but this is by no means constant. The disease resembles, in some respects, the leg weakness caused by the lack of certain vitamins, which in some cases is a rachitic condition while in others it is a polyneuritic condition. This disease is not prevented nor cured by the use of cod liver oil.

PRESENT WORK

The observations recorded in this paper have been made from four flocks, one a commercial flock, the other three were of the small flock type seen on the

average farm. Post-mortem examinations were made on forty-six birds in various stages of the disease, and histo-pathological examinations were made of the nervous system. Bacteriological cultures were made from the intestinal tract of a number of birds. Particular attention was paid to the vitamin content of the ration fed to these flocks, to eliminate the chance of confusing this disease with rachitis and poly-neuritis.

HISTORY OF AFFECTED FLOCKS

Flock No. 1. A commercial flock of one thousand white leghorns had a mortality of one hundred fifty birds from this disease during the summer and autumn of 1924. It was found that during the early part of the season they were fed on a commercial mash and scratch feed, sprouted oats and semi-solid butter milk. After the trouble manifested itself the ration was changed to the following:

80 pounds corn meal
20 " meat scraps
1 pound sodium chloride
5 pounds bone meal
5 " lime stone

They were given sprouted oats and all the semi-solid butter milk they would eat.

The change in ration had no affect upon the disease, and the birds continued to become paralyzed during the autumn and winter. Many of the pullets came down with the paralysis after they had been laying for some time. Strong vigorous birds were as susceptible as those that were not as thrifty.

In the spring of 1925, a large number of eggs were hatched from this flock. Many of the baby chicks were sold to various parties in the state, and about two thousand chicks were kept by the owner of the flock. These were fed on a ration composed of mash, salt mixture and scratch feed. Mash was composed of equal parts of the following:

Bran,

Middlings,

Oats,

Glutin feed,

Corn meal,

Meat scraps.

Salt mixture was added to the mash as follows:

3% Calcium carbonate,

3% Acid phosphate,

1% Sodium chloride.

Scratch feed composed of the following:

Wheat,

Cracked corn.

The ration was supplemented with all the semi-solid butter milk the chicks would eat, to which cod liver oil was added in the following proportions:
One part of cod liver oil to sixteen parts of semi-solid butter milk. They were also given lettuce, rape, and sprouted oats. When the chicks were about six weeks of age the flock was divided, half of the chicks were placed on an alfalfa meadow where chickens never had been raised. In spite of the cod liver oil and plenty of green feed and free range, paralysis developed as soon in the experimental flock as in the home flock. The disease appeared when the birds were about ten to twelve weeks of age. The birds, sold as baby chicks and going to different parts of the state, were not troubled with this disease. Birds continued to become paralyzed until the last of December when it seemed to almost disappear.

HISTORY OF INDIVIDUAL BIRDS

Bird No. 1.- White leghorn pullet about twelve weeks of age with paralysis of both legs.
Autopsy-

Heart, normal.

Lungs, normal.

Liver, slightly enlarged, dark in color.

Gall bladder,distended.

Spleen, normal.

Kidneys appeared normal.

Gizzard, normal.

Intestines- numerous coccidia and congestion of the mucosa.

No tape worms present.

No round worms found.

Bird No. 2. White leghorn pullet, age twelve weeks, right leg and toes of left leg paralyzed.

Autopsy-

Liver, normal.

Heart, normal.

Gizzard, normal.

Proventriculus, normal.

Intestines- congested throughout the entire length.

No coccidia present.

No intestinal worms present.

Bird No. 3. White leghorn rooster, age fourteen weeks. When received the bird showed paralysis of the right wing, right leg to the hock joint and the toes of the left leg.

Autopsy-

Heart, normal.

Lungs, normal.

Liver, normal.

Spleen, atrophied.

Intestines- showed inflammation of the duodenum.

No tape worms present.

Three ascaride present.

No coccidia found.

Bird No. 4. Both legs were paralyzed, left leg drawn forward, right leg backward.

Autopsy-

Heart, normal

Lungs, normal

Gizzard, normal.

Liver showed a few necrotic areas.

Intestines- hemorrhages of the duodenum.

Numerous coccidia were found.

No tape worms found.

No ascarides present.

Ceca- numerous coccidia.

Bird No. 5. White leghorn pullet died in pen, left leg was drawn forward, right leg backward.

Autopsy-

Heart, normal.

Spleen somewhat smaller than normal.

Liver slightly enlarged.

Gall bladder very large.

Intestines- badly congested throughout the entire length.

Numerous tape worms about three-fourths of a centimeter in length were found.

Bird No. 6. White leghorn pullet.
The bird was slightly paralyzed in the toes of the right foot when received at the college, and was kept for observation. Ten days later the entire leg was paralyzed and drawn backward and the left leg showed signs of paralysis. At the end of three weeks both legs were completely paralyzed. The bird was unable to move even a few inches for food and water but retained a good appetite.. At the end of the fourth week it was necessary to hold the bird while it drank water. However, it was able to pick grain from the floor.

Autopsy-

Heart, normal.

Liver, normal.

Lungs, normal.

Intestines- hemorrhagic ares throughout the entire length. Two ascarides were found. No tape worms were found.

Ceca- a few coccidia.

Sciatic nerve enlarged and of a pink color.

Bird No. 7. White leghorn pullet. Both legs paralyzed from feet to hock joint. The bird could move about but showed considerable incoordination. Post-mortem examination was made during the early stages of the disease.

Autopsy-

Heart, normal.

Liver, normal.

Spleen, normal.

Gizzard, normal.

Kidneys, normal.

Lungs, normal.

Intestines- badly congested, fifteen tape worms, each about two inches long, were found. No coccidia.

Bird No. 8. White leghorn pullet. Right leg paralyzed and drawn backward, left leg apparently normal. Diarrhea was present.

Autopsy-

Heart, normal.

Liver slightly enlarged with a few necrotic foci.

Intestines- hemorrhages of the duodenum and of the ileum. No tape worms and no round worms were found. Coccidia were found in the duodenum, and also in the ceca..

Bird No. 9. White leghorn pullet. The right leg paralyzed from foot to hock joint, left leg paralyzed entire length and extended in a lateral position.

Autopsy-

Heart, normal.

Liver larger than normal.

Gall bladder very large.

Spleen atrophied.

Lungs normal.

Intestines- congested. No coccidia nor tape worms were found. Sciatic nerves and perineal nerve of the left leg were enlarged and pink in color.

Bird No. 10. White leghorn pullet about three months of age. When received, the toes of the left leg were flexed, otherwise the bird appeared normal.

One week later the paralysis had progressed to the hock joint, and the right toes became paralyzed. The paralysis steadily progressed until the end of the third week the left leg was extended forward and the right leg drawn backward. The appetite was good until the bird was killed.

Autopsy-

Heart, normal.

Liver, normal.

Gall bladder very large.

Spleen very small.

Intestines- badly congested the entire length. No tape worms, round worms or coccidia were found.

Ceca contained few worms.

Sciatic nerve of right leg was swollen and pink in color. Perineal nerve of left leg was swollen.

Bird No. 11. White leghorn pullet.

Both legs totally paralyzed. It was noted that in the right leg the sciatic nerve was enlarged, and pink in color while the perineal nerve appeared normal macroscopically. The leg was drawn backward fully retracted. The left perineal nerve was much larger and pink in color. The leg was drawn forward fully extended.

Autopsy-

Heart, normal.

Liver, normal.

Spleen enlarged.

Intestines- congested.

No tape worms, round worms or coccidia were found.

Bird No. 12. White leghorn pullet. Both leg and right wing were paralyzed.

Autopsy-

Heart apparently normal.

Liver slightly enlarged.

Spleen atrophied.

Intestines- congested in duodenum. No intestinal parasites. Nerves were swollen and inflamed as given in bird No. 11.

Bird No. 13. White leghorn pullet. Right leg paralyzed and drawn outward in a lateral position and left leg showed considerable incoordination.

Autopsy-

Sciatic and perineal nerves of the right leg were swollen and pink in color. The corresponding nerves of the left leg appeared normal.

Heart, normal.

Liver very dark in color.

Spleen under sized.

Gall bladder, normal.

Intestines- congested and no parasites found.

Bird No. 14. Black Minorca pullet.

Right leg normal, left leg paralyzed and drawn backward. Muscles appeared atrophied.

Autopsy-

Sciatic nerve slightly swollen and pink in color.

Heart appeared normal.

Spleen anemic and small.

Liver apparently normal.

Intestines- normal. No parasites were found.

Bird No. 15. White leghorn cockerel.

This bird showed incoordination in both legs.

Autopsy-

Sciatic and perineal nerves were apparently normal.

Liver slightly discolored.

Intestines- congested and numerous tape worms each about two inches in length were found. No

other parasites present.

Bird No. 16. Black Minorca pullet.
Right leg showed incoordination and left leg apparently normal.
Autopsy-

Sciatic and perineal nerves appeared normal.

Heart, normal.

Liver slightly enlarged.

Intestines congested and no parasites were found.

Bird No. 17. White leghorn pullet.
Incoordination of both legs.
Autopsy-

Heart, flabby.

Liver enlarged.

Gall bladder distended.

Spleen normal.

Intestines- very few round worms and no tape worms found.

Bird No. 18. White leghorn pullet.
Both legs were paralyzed and drawn backward.
Autopsy-

Heart, normal.

Lungs, normal.

Spleen, anemic.

Liver, normal.

Intestines- numerous coccidia but no other parasites found.

Ceca- numerous coccidia were found.

Rectum greatly distended.

Flock No, 2. The owner of this flock reported incoordination and paralysis of both legs in a few birds during the latter part of the summer and autumn of 1924. The first birds to become affected, during the summer of 1925, occurred the first of August. The birds were on free range with access to an alfalfa meadow and were fed on a ration composed of mash and scratch feed. Mash was composed of equal parts of the following:

Corn meal,

Middlings,

Ground oats,

Meat scraps.

Scratch feed was composed of the following:

Wheat,

Cracked corn.

Ten birds were examined, by the writer, and with but

one exception showed the same symptoms as those of flock No. 1, and the lesions found were not unlike those of the first flock. The exception will first be considered. A photograph was taken of this bird in the early stages of the disease Fig. 3, it will be noted that the neck is affected being drawn into an S shaped curve and the head is rotated. One week later the head was drawn around into a ventrodorsal position. This was the only bird, observed by the writer, to show these symptoms of the neck.

Flocks No. 3 and 4. It was reported that there was no paralysis in these flocks until the summer of 1925. Both were on free range and the rations were not materially different from that of flock number two. Symptoms and lesions were true to type as observed in flock No. 1. Six birds were examined from each flock.

The ration fed to flocks 2, 3, and 4 contained plenty of green feed but no cod liver oil. Birds of these flocks were given cod liver oil or fresh ox gall as a curative to differentiate the disease under study from rachitis and polyneuritis. The fresh ox gall treatment was first used by Kapsinaw and Jackson[3] as a preventative and cure for rickets in rats. The writer has found it to be a very effective treatment in rickets among chickens. A number of birds, which

had completely lost the use of their legs, were given 8 cubic centimeters of fresh ox gall daily, and recovered in a week to ten days. Cod liver oil is usually recognized as an agent for the prevention and cure of these cases. The use of fresh ox gall in the early stages of this paralytic disease resulted in an improvement for a few days after which the disease progressed as before. Results with cod liver oil were negative.

DISCUSSION OF POST MORTEM FINDINGS

In the post-mortem examinations of the first paralyzed birds, coming to the laboratory, no attempt was made to examine the nerves. Those examined later, however, showed varying degrees of enlargement of the sciatic or perineal nerves and sometimes of both. In the majority of cases the heart, liver, lungs, gizzard and kidneys appeared normal. A small anemic spleen was common. Of the abdominal organs, the intestines showed the most constant lesions; congestion or hemorrhage of the duodenum was present in nearly every bird examined. The presence of parasites was not constant, and the large numbers found in some birds can no doubt be accounted for by the lowered resistance of the bird, thus breaking down the protective forces of the body and permitting the para-

sites to develop.

BACTERIAL ISOLATION

This disease, resembles that described by Marx and Robertson not only in the paralysis of the legs, but also in the congestion or hemorrhages of the duodenum from which they isolated organisms capable of reproducing the disease. It was thought that an organism causing the paralysis of chickens might be isolated from the contents or mucosa of the duodenum. The following technique was used:

1.- Varying dilutions were made of the intestinal contents in physiological salt solution and inoculations were made from each dilution into the following:

Nutrient agar plates.

Liver agar plates.

Blood agar plates.

Endo agar plates.

Brilliant green agar plates.

Lactose agar tubes (shake cultures) to which sterile petrolatum was added to produce anaerobic conditions.

Liver agar tubes (shake cultures) were cultivated anaerobically under a two inch layer of sterile petrolatum.

2.- The contents of the duodenum were washed out

carefully with physiological salt solution. A small piece of the duodenum was washed in ten changes of physiological salt solution, using about 250 cc to each washing. The mucosa was then scraped with a sterile knife, the scrapings being suspended in physiological salt solution, which was re-diluted several times and inoculations were made as given under one.

3.- The intestinal contents were washed and a small section of the duodenum was washed again in saline solution, after which it was iodized with colloidal iodine just long enough to sterilize the surface. The mucosa was then scraped with a sterile knife and the scrapings were placed in a sterile physiological salt solution. Inoculations were made as given under one.

The plates and tubes were incubated at thirty-seven degrees centigrade and observed after twenty-four hours, at which time colonies were picked from the plates and transferred to liver agar slants. After twenty-four hours the agar slants were washed with physiological salt solution and the suspension of organisms was injected into normal white leghorn pullets about three months of age. At the end of

three months there were no symptoms of paralysis, among the birds injected. No attempt was made to classify the organisms isolated, as there was no indication of their connection with this disease.

TRANSMISSION OF THE DISEASE

1.- Blood of two affected birds was drawn into sterile test tubes; the clots were loosened with a sterile needle and the serum was separated by centrifuging. Three normal white leghorn pullets, about eight weeks of age, were each injected with 5 cc of the serum. One was injected intravenously, one subcutaneously and one intraperitoneally. Results were negative after three months.

2.- Blood of two affected birds was drawn into physiological salt solution containing citric acid to prevent blood from coagulating. Injections were as given above into three normal white leghorn pullets. Results were also negative after three months.

3.- The muscles, liver, spleen and heart of a paralyzed bird were ground up and fed to normal birds without results.

4.- The intestinal tract was cut into short pieces and fed to normal birds which remained healthy after

three months.

5.- Brains, spinal cord and nerve trunks were removed as quickly as possible and ground in a sterile mortar with fine sterile quartz , and suspension was made in warm physiological salt solution and injected into normal birds as given in No. 1. The results were also negative.

6.- It was observed that a great number of birds in flock No. 1, were roosting out of doors in apple trees, on fences, and on irrigation pipes. It appeared that the birds out of doors remained healthy while those resting in the coop continued to become affected. It was found that the coops were infested with red mites. This fact together with the fact that there are very few cases of the disease during the winter months when the mites are inactive. This suggests that the mites may play some part in the transmission of the disease, either acting as an intermediate host or as a carrier.

Ten normal birds were placed in a brooder coop with six diseased birds. The coops were infested with red mites. The result of this work was negative, but unfortunately this work was carried on during the latter part of September and October, 1925. The weather turned cool and it is doubtful if the mites were

sufficiently active to have transmitted the disease even if they were the infective agent.

HISTOLOGICAL TECHNIQUE AND EXAMINATION OF TISSUES

The following technique was employed in preparing the sections for histo-pathological study of the brain

The bird was quickly killed by cutting off the head with a sharp knife, for it was important that the bird should be killed with minimum shock, and without the use of drugs. The brain was immediately removed by carving away the skull with a knife. The brain tissues were cut into four parts and dropped into warm fixing solution, which consisted of four per cent formaldehyde in saturated aqueous solution of corrosive sublimate, and allowed to remain for twenty-four hours. The tissues were then washed for twenty-four hours in running water, after which they were carried through 60, 70, and 80 per cent alcohol to which a few drops of five per cent alcoholic iodine was added, until the excess corrosive sublimate was removed. The alcohol was changed as frequently as it becomes decolorized. The tissues were then dehydrated, cleared, imbedded and sectioned. The sections were cleared in the various grades of alcohol

and stained with methylene blue in saturated anilin oil water. They were hurriedly rinsed in a large volume of water after staining, and placed in 95 per cent alcohol until sufficiently destained. They were cleared in xylene, and mounted in neutral balsam.

The cerebellum being one of the centers of co-ordination, the Purkinje cells were studied very carefully, by comparing sections made from diseased birds with those of the normal which were passed through the same solutions and at the same time. There was apparently no change in these cells.

Brain tissues were also fixed by Zenker's method and were stained with eosin and methylene blue. Punctate cerebral hemorrhages were found.

The spinal cord was also fixed by Zenker's method and were stained with eosin and methylene blue. Sections show what appears to be a degeneration of the ganglion cells. Some appear to have lost the Nissl granules while others show no nucleus.

Examination of the sciatic and perineal nerves in sections fixed by Zenker's method and stained with eosin and methylene blue, show a degeneration of the nerve fibers, and an infiltration of round cells and in many places a complete disappearance of the fiber. These changes depend upon the stages of the disease.

In the early stages very little degeneration can be noticed by this method of staining. However, degeneration can be seen by osmic acid fixation, even in nerves that show but slight infiltration of mononeuclear leucocytes. In the advanced stages infiltration of leucocytes is extensive. Areas thickly infiltrated with leucocytes are seen in the affected trunks, in the early stages as well as in the latter stages of the disease. Chromophilic granules resembling microorganisms can be seen in these densely infiltrated areas.

PHYSIOLOGICAL EXAMINATION

A bird, completely paralyzed in the right leg which was drawn backward and the left leg badly affected, was placed under ether anesthesia. The muscles of the thigh and the sciatic and perineal nerves were exposed. Direct faradic stimulation of the muscles of the right leg on the posterior side of the femur produced contraction, lifting up the leg which was held straight backward. Direct faradic stimulation of the anterior muscles produced no contractions. The nerve was stimulated which produced a contraction of the posterior muscles but no contractions

of those on the anterior side of the femur. The muscles and nerves of the left leg were exposed in a similar manner. Direct stimulation of the muscles, with an induction coil, on the posterior and anterior side of the femur responded to the stimulant, but the contractions were not as strong as normal. The nerve trunks were stimulated producing a contraction of the muscles on the posterior part of the thigh in the right leg. The nerve trunks were severed and each end was stimulated. The lower end produced contractions the same as before it was cut. Stimulation of the proximal end produced no results.

DISCUSSION

Keeping in mind the pathological findings, the results of the nerve stimulation were about what one would expect. However, direct stimulation of the muscles without response, indicates that the ganglion cells and nerve trunks are not alone degenerated, but the nerve endings and possibly the muscles themselves may be degenerated.

This, however, is contrary to the findings reported by May, Tittsler and Goodner (15) in which they state, "Stimulation of the sciatic nerve in a recently killed bird produces a strong muscular reaction. This shows

that there is no incoordination between nerve and muscle and that both nerves and muscles appear to function normally."

EXAMINATION OF BLOOD

Blood was drawn from a paralyzed bird and smears were made on slides in the usual way. The smears were stained by Wright's method.

The number of leukocytes of one bird appeared normal in every respect. The other three birds showed a marked leucocytosis. The number of leucocytes of one bird were about three times the number found in a normal fowl. The other two showed an extremely high leucocytosis. The majority of these cells were of the rod-bearing polyneuclear type and small lymphocytes.

GENERAL DISCUSSION

It appears from the nature of the disease and the pathological and physiological findings that the malady is undoubtedly caused by some infectious micro-organism. However, the experiments recorded in this paper like those of other investigators, in this country, have failed to show the cause of the disease.

It also appears, from the experiments with chicks

in flock No. 1, in the spring and summer of 1925, that there may be a long incubation period of six to eight weeks or more.

The pathological findings of the brain and spinal cord confirm those of other investigators. The changes found in the sciatic and perineal nerves and the reactions to electric stimulation seem to disprove the opinion of other investigators, that the incoordination is purely of the central nervous system, would indicate that the primary lesions were in these parts and that the lesions of the central nervous system are of secondary origin.

The increase of leucocytes also points to the possibility of this being an infectious disease.

CONCLUSIONS

1. The disease is not due to a dietary deficiency.
2. The cause of the disease is unknown.
3. The seat of the disease is probably not in the central nervous system but in the peripheral nerves.

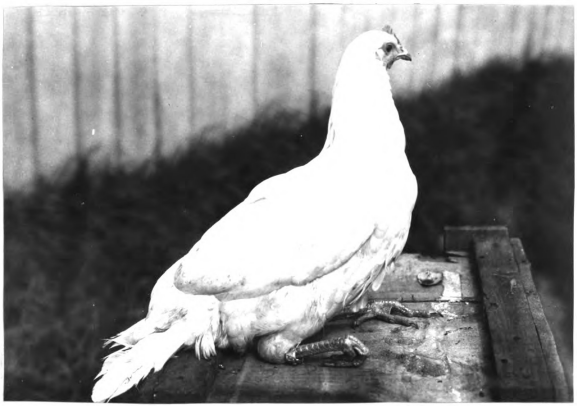

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

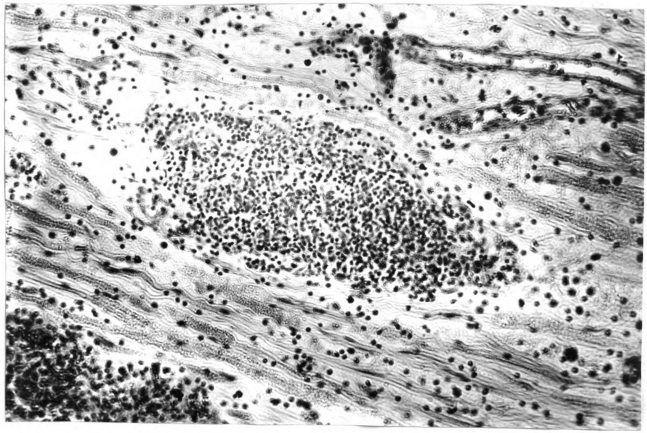

Fig. 5.

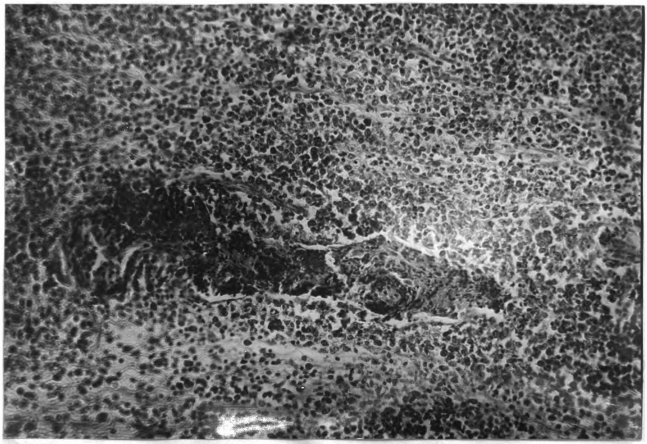

Fig. 6.

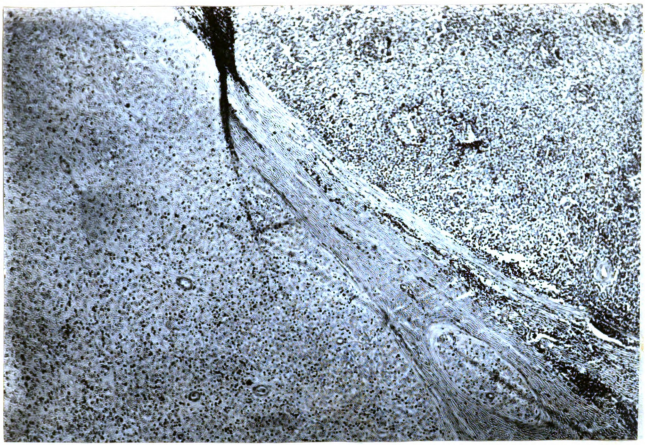

Fig. 7.

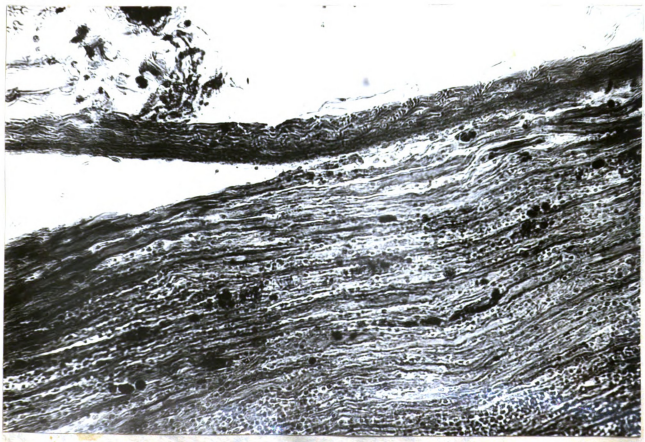

Fig. 8.

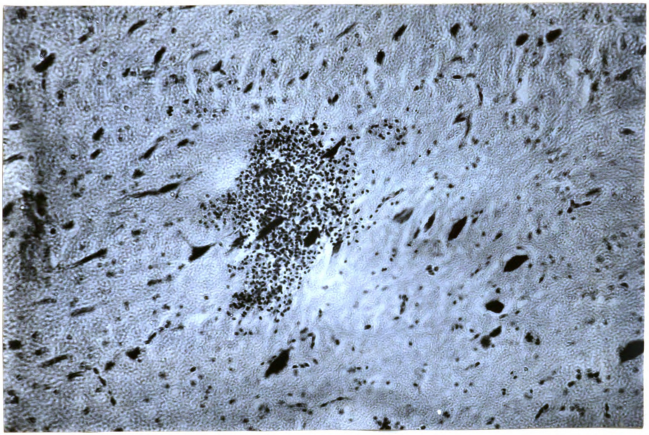

Fig. 9.

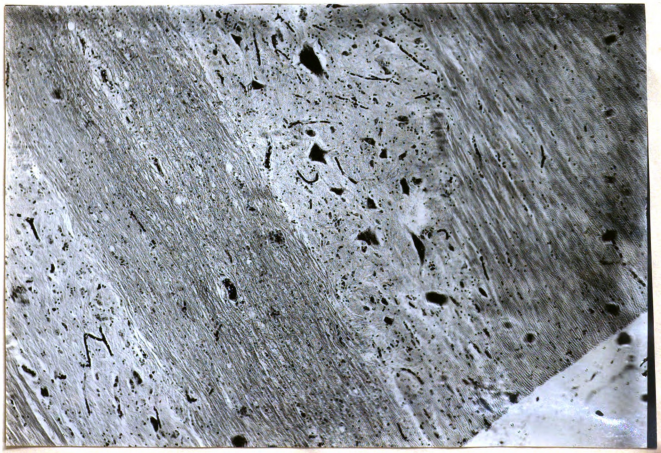

Fig. 10.

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ACKNOWLEDGMENTS

I gratefully acknowledge the assistance of Doctor Ward Giltmer, Doctor William L. Chandler, Professor William L. Mallman and others for suggestions and generous help in this work.

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