STUDIES ON FOWL PARALYSIS (NEUROLYMPHOMATOSIS GALLINARUM).*

I. Clinical Features and Pathology.

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Plates 6 to 12.

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The disease known to poultrymen as fowl paralysis or range paralysis is one which, in spite of its economic importance and great biological interest, has been little studied.

The first account of the disease to which we have found reference is that of Marek (1) in 1907. In four roosters, three of them Orpingtons, and all from the same farm, there developed during the winter months, symptoms of paresis of the legs and wings, unequal on the two sides, and accompanied by more or less muscular atrophy. The diet was adequate, comprising potatoes, barley, bran, clover and mixed grains, and the chickens were allowed free range. Two of the birds made a partial recovery, one died after 25 days, one was killed 5 weeks after the onset of symptoms, and the description of the pathology is based upon the careful study of this bird.

Grossly, there was noted marked thickening of the right lumbar plexus (the side on which the paralysis was more marked). Most of the spinal nerve roots, especially on the right side, were also thicker than normal.

Microscopic examination was made of four segments of the cervicodorsal and lumbosacral regions of the cord with their nerve roots, of the lumbar plexus, sciatic and femoral nerves and the extensor quadriceps femoris muscle. The tissue was

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fixed in Mueller's fluid, stained with Weigert-Wolters and counterstained with alum-cochineal and Van Gieson.

In the plexus and sciatic nerves, there was almost a complete loss of nerve fibers. There was a dense and uniform infiltration of mononuclears, in places aggregated into dense clumps. The perineurium was but slightly thickened and contained only scattered cells. Nests of mononuclears were present in the surrounding connective tissue, chiefly in the vicinity of small blood vessels.

The spinal cord and the nerve roots, especially upon the right side, were also infiltrated. In the cord, the cellular infiltrations were almost limited to the white matter, but in one segment, there was a perivascular accumulation in the central grey.

The degeneration of the nerve fibers was regarded by Marek as secondary to the cellular invasion. The cause of the polyneuritis could not be determined, but the disease was obviously not identical with the neuritis described by Eijkman (2) in chickens fed on polished rice. In the latter condition, death ensued within 10 days, and the changes in the nerves were purely degenerative.

Kaupp (3) in 1921 reported rather fully upon observations first made in 1914. In this paper, reference is made to outbreaks occurring in North Carolina, Maryland, Virginia, New Hampshire, New Jersey, Connecticut and Massachusetts. No information could be obtained by him as to the occurrence of a similar disease in foreign countries.

The clinical features as described by Kaupp are lameness in one or both legs, drooping of wings, partial blindness, finally diarrhea, complete paralysis and death days or weeks after the onset. Recovery, in Kaupp's experience, never occurred. All breeds were susceptible.

As regards the lesions, Kaupp found no changes in the cerebellum, medulla, crura or brachial plexus. In the cord, however, corresponding segmentally to the paralysed part, there was congestion of peripheral veins, round cell infiltration in both ganglionic and non-ganglionic portions of the cord, chiefly perivascular. There was degeneration of the ganglion cells, atrophy of the fibers and at times a liquefaction necrosis. The process was interpreted as a transverse myelitis. Kaupp studied the disease experimentally, but without arriving at any definite results. Repeated efforts to isolate a causative organism were without success. Inoculation of heart blood, liver, kidneys, spleen, various parts of brain and cord, especially of the affected segments, into rabbits, guinea pigs, young and year old fowls all failed to reproduce the disease. In spite of these negative experiments, Kaupp regarded the disease as infectious.

An interesting paper treating of an apparently identical disease occurring in the Netherlands, is that of Van der Walle and Winkler-Junius (4). The authors studied an epizootic which occurred in the months of October and November, 1921. A large number of fowl were affected with paralysis, which ended acutely after a few days or weeks. Most of the affected birds died, but the appetite and general appearance of the chickens was unchanged even in the last stages of the

disease. Avitaminosis could be excluded, and the food contained neither arsenic, copper, lead nor zinc.

Two of the cases were examined pathologically. Inflammatory infiltrations of polynuclear and mononuclear leucocytes were found in the swollen dorsal ganglia; the meninges and spinal cord were likewise infiltrated, and in one of the birds, the sacral and lumbar plexus showed mononuclear infiltration.

The alterations of the nerve elements were regarded as secondary to the infiltrations, and the apparently inflammatory nature of the lesions suggested the term "neuromyelitis gallinarum" for the disease. No mention is made of lesions of the brain.

A number of experiments to demonstrate the infectious nature of the disease led the authors to conclude that this epizootic was due to a filtrable virus. We shall discuss these in connection with our own experiments on transmission of the disease.

May, Tittsler and Goodner (5) in December, 1925, issued a preliminary report of field observations and laboratory findings in paralysis of the domestic fowl. The lesions are very briefly and incompletely described. The authors state that microscopic examination of brain and spinal cord has given little information in regard to the seat of the disease. Sections of brain and cord confirmed Kaupp's findings, but the lesions are not described in detail, and no mention is made of alterations in the peripheral nerves. Both aerobic and anaerobic cultures from the tissues of infected birds consistently failed to show any infective organisms. Attempts to transmit the disease by association and contact, by feeding material from paralysed birds or by inoculation of blood, emulsified brain or spinal cord, intravenously, intraperitoneally or intraspinally were all negative. None of the inoculated animals developed paralysis during an observation period of 2 months.

The authors discuss the possible rôle of intestinal parasites and reach the conclusion that there is no relation, or at least that the presence of intestinal worms or coccidia does not necessarily produce paralysis.

The most recent paper on the subject is that of Doyle (6) from the Indiana Agricultural Experiment Station. Both the symptoms and the pathology of the disease are very completely described. The disease occurs in birds from 4 to 8 months old, and is more prevalent in summer and fall. The symptoms depend upon the portion of nervous system involved. The occurrence of encephalitis is responsible for the lethargic symptoms frequently noted. Iritis, when present, causes a contracted, non-responsive pupil. There is a good description of the histopathology of the brain, cord and peripheral nerves.

Efforts to demonstrate the infectious nature of the disease were fruitless; the author's experiments will be considered in detail in the second paper, but it is interesting to note here that in spite of his failure to establish the transmissibility of the disease, he is still convinced that it is due to an infectious agent.

Clinical Features of the Disease.

The first appearance of the disease is usually signalized by drooping of a wing on one side, a lack of coordination in walking, followed by a lame or limping gait, and ending in complete prostration. The limbs are not symmetrically affected, even in advanced cases. The chickens usually lie upon the side showing the greater paralysis, and the impairment of mobility is at first unilateral, or at least more marked upon one side than the other.

In the earlier stages of the disease, there is little muscular atrophy, but after the chicken has been helpless for a longer period, the wasting may become extreme. Whether the atrophy is due to the affection of the nerves, to the inactivity or to the inability of the chicken to procure food has not been determined.

The paralysis is more often spastic than flaccid, and sometimes accompanied by increased reflexes or even by clonic spasms. The paralysis is only exceptionally complete, and even in birds which lie immobile and helpless, the struggles during anesthetization are attended by vigorous movements of flexion or extension. Reflex movements and muscular reaction to mechanical nerve stimulation have been noted in the paralysed limbs after death. The curious postures assumed by the paralysed birds are illustrated by Figs. 1 and 2.

The duration of the disease, as judged from the onset of the paralytic symptoms to the termination in death or rarely in recovery, is very variable. With proper care, life may be prolonged indefinitely, and the nutrition and general condition is often surprisingly good, even after months of complete helplessness when the birds are kept in confinement with access to food and water. Thus one of ten early cases in the station flock at Storrs became prostrated early in October, 1922. With careful feeding in an individual coop, it lived until January 1, 1923. Recovery has been noted in several instances, and we have gained the impression that there frequently occur periods of transient improvement. On the other hand, death may suddenly occur with few premonitory symptoms, the diagnosis being revealed only by the pathological study of the cases.

The onset of the paralysis may be very sudden. We have observed one case in which complete loss of power in the legs developed in less than 3 hours.

Partial or complete blindness in one or both eyes, accompanied by a change in the color of the iris from yellow-brown to slate-grey, was observed in several paralysed chickens, and in two non-paralysed birds from the same flock. The alterations which underlie this condition will be described below.

As regards the age incidence, it would appear that young birds are most frequently affected. In our experience, the earliest symptoms in a spontaneous case were observed at 12 weeks, and the oldest case in our records developed paralysis at 15 months, 18 days. In the experimental material, the earliest clinical case was observed at 10 weeks of age. As will become apparent later, the characteristic lesions do not always reveal themselves in typical paralytic symptoms. We shall show also that mild cases exist in which the diagnosis cannot be made from symptoms alone.

Epidemiology and Distribution.

The records of the Storrs Experiment Station flock of White Leghorns in which each year about 1500 growing chicks and 800 adult chickens come under observation, have yielded data of some value in regard to the natural history of the disease, and instructive information has been obtained from visits to affected farm flocks. It is impossible to present an analysis of these data within the confines of this present article, but we may refer briefly to some of the more interesting deductions.

(1) The disease appears to be epidemic and to reappear year after year on the same farm. In cases where its origin has been observed, it has appeared suddenly and has thereafter persisted and become endemic in certain foci. Efforts to prevent its recurrence by rearing new flocks on new ground on the same farm, have, in several cases, shown no significant effect on the persistence of the disease. (2) Paralysis occurs in both sexes and in all of the chief breeds of fowls. There is some indication of breed differences in susceptibility to the disease, although the observations on which these are based are in no sense experimentally controlled and the difference may have been due to other conditions. (3) The disease first appears usually in growing chickens from 3 to 6 months of age, and new cases continue to de-

velop throughout the first 18 months of life. It is probably rare in older fowls although a few clinical cases have been observed. (4) In flocks in which an outbreak has occurred, paralysis usually causes an appreciable mortality each year thereafter. Mortality may range from a few per cent, up to 60 per cent and over.

Pathology.

The following description is based upon a study of 60 spontaneous cases in all stages of the disease. Many more have been autopsied in the course of the experimental work. The chickens were obtained from a number of different sources, and characteristic lesions have been found in material from Maine, New Hampshire, Massachusetts, Pennsylvania, New Mexico, California and Ontario. The following breeds are represented: White Leghorns, Silkies, Silver-Spangled Hamburgs, Plymouth Rocks, Buff and White Wyandottes and Rhode Island Reds. In this series, the lesions were identical, irrespective of breed.

Numerous chickens presenting no clinical evidence of paralysis, have been studied.

The brain after fixation in Zenker's was cut in serial blocks so as to include sections of cerebrum, optic lobes, cerebellum, pons and medulla. The entire spinal cord was removed, and longitudinal sections examined at all levels. The dorsal root ganglia, and the brachial plexus and sciatic nerves were also taken for study, and routine sections from all the principal viscera. Zenker's fluid, and 10 per cent formol or Bouin's fluid in special cases, were used as fixatives. In addition to routine histological stains, sections from individual cases were studied by the Marchi and Pal-Weigert methods, and a variety of staining methods were used in the attempt to demonstrate microorganisms in the tissue.

Gross Lesions.

The general nutrition of the birds varied, depending upon the duration of the disease and the care which the chickens had received. Many typical examples showed excellent nutrition; others extreme emaciation. Often the feathers about the cloaca were matted with excrement, owing to a terminal diarrhea or incontinence.

No recognizable alterations were found in the brain, or its covering. The posterior root ganglia of the cord were found enlarged, and in the majority of cases, to such a degree that, after a little experience, the histological changes could be

predicted with fair certainty. Not only were the ganglia enlarged, but they appeared abnormally translucent, and usually they had a slightly yellowish tinge in comparison with the cream-white of the cord itself. On close inspection, one could frequently discern a translucent strip extending into the contiguous tissue of the cord. The enlargement of the ganglia, in most cases, was not symmetrical (Fig. 3).

In one case there was found a translucent, tumor-like enlargement of the acoustic nerve. In another, one of the trigeminal nerves was thickened. We have made no systematic study of the cranial nerves.

No striking gross changes were found in the spinal cord itself. The *peripheral nerves*, in particular those of the brachial plexus and the sciatic nerves, were frequently thickened and the same smoothness, translucency and yellowish discoloration were noted as in the posterior root ganglia (Fig. 3). Small hemorrhages were occasionally seen.

Lymphomatous tumor-like masses were discovered in six birds of this series, or about 10 per cent. These appeared to originate in the ovary, but growths were seen in the liver, kidneys, lungs, adrenals and muscle, and in one instance, the spinal cord was infiltrated with the same type of tissue. The finer structure of these lymphomatous masses and their possible relation to the lesions of the nervous system, will be discussed below.

None of the lesions occasionally met with, such as edema and atelectasis of the lung, suppurative bronchiectasis, ulceration of the intestine associated with severe coccidiosis, etc., seemed to be related to the disease under consideration. *Heterakis papillosa* was present in the ceca in variable numbers in practically every case, *Heterakis perspicillum* in several and cestodes occasionally.

Microscopic Lesions.

Striking alterations were found in the brain, spinal cord, dorsal root ganglia, spinal nerve roots and peripheral nerves. The distribution and intensity of the lesions varied from case to case, but in general the lesions resembled one another sufficiently to establish a definite pathological entity.

Peripheral Nerves.

The most severe lesions were seen in the peripheral nerves and nerve roots. They consisted essentially of an infiltration of smaller and larger mononuclear cells between the nerve fibers, separating the individual fibers and accompanied by more or less edema. This infiltration was at times so massive as to replace almost completely the nerve tissue. More commonly, the cells were loosely distributed and in some areas, very sparsely. It was rare for all the bundles of the nerve to be affected, and never did the infiltration extend uniformly throughout the entire length of the section (Figs. 4 to 6).

Degenerative changes in the nerve fibers varied greatly in the different cases examined, and were roughly correlated with the extent and intensity of the cellular infiltration. In many instances, where the lymphoid invasion was sparse and scattering, the degeneration of the nerve fibers was not detectable with the usual stains. At the other extreme, many of the fibers had disappeared, their places being taken by a vacuolar or foamy tissue, often filled with coagulated edema fluid, and containing free fat globules and numerous fat-laden phagocytes, derived in part from the swollen sheath cells. The myelin sheaths appeared collapsed, and Pal-Weigert preparations gave evidence of advanced degeneration (Figs. 7, 8). In some sections, there could be noted a proliferation of the spindle-shaped sheath cells, amongst which mitotic figures were abundant.

Even when these degenerative changes were well advanced, the persistence of the neuraxones in the infiltrated nerves could be demonstrated in silver carbonate preparations (method of Rio del Hortega), and it is probably due to this fact that paralysis, even in advanced cases, is rarely complete.

The smaller forms of infiltrating cells appear to be morphologically identical with lymphocytes. In the nodular accumulations which crowd aside the adjacent nerve fibers, the center of the mass is composed of somewhat larger elements, with more vesicular nuclei, and here mitoses are frequent. In addition to cells of the lymphocytic type, which predominate, there are numerous cells with more abundant basophilic cytoplasm, peripherally disposed chromatin and a juxtanuclear clear space, which closely resemble mammalian plasma cells. These are vividly brought out in sections stained with Pappenheim's methyl green pyronine. There are also large mononuclear cells with vacuolar cytoplasm corresponding to the fat-laden phagocytes derived from the cells of Schwann's sheath. In paralysed chickens stained intravitally with trypan blue, many of the large mononuclears in the infiltrated nerves, particularly in the vicinity of the blood vessels, were found to accept the dye though less intensely than the Kupffer cells, or the clasmotocytes of the spleen and other tissues.

In a case recently studied, most intense lymphoid infiltrations were present in the macroscopically thickened vagus and mesenteric nerves.

Spinal Cord.

In the spinal cord, in addition to the regional infiltrations accompanying the entrance of the nerve roots, there are found focal accumulations of wandering cells most commonly in the white matter, but in some instances also in the central grey. Lesions of this type, which may be termed submiliary nodules, seem to be limited to the vicinity of small blood vessels. The cells composing them are in part definitely lymphoid in type, in part, cells with paler, less spherical and somewhat larger nuclei, the origin of which could not be accurately determined. They are most probably mobile glial elements. A very constant feature of these nodules is the presence of spherical, deeply stained chromatin particles of varying size and shape interspersed amongst the intact nuclei. These appear to be derived

by a massing of the chromatin against the nuclear membrane and its subsequent extrusion; often they are surrounded by a narrow clear zone.

In no case was there definite necrosis in the center of the nodules, nor have we observed the occurrence of giant cells.

Degenerative changes in the ganglion cells of the cord were seen only where the lymphomatous infiltration was so massive as to bring about extensive destruction of the cord tissue. The most obvious type of degenerative change was a hyalinization of the cytoplasm, with chromatolysis, and karyolysis.

Posterior Root Ganglia.

The posterior root ganglia, as one might expect from the alterations in the gross appearance, frequently show an intense infiltration with mononuclear cells (Figs. 10, 11). This was sometimes diffuse, sometimes rather circumscribed. The extent and intensity of the infiltrations varied greatly in different ganglia from the same case. The ganglion cells were usually intact, even when the lymphoid infiltrations were extreme.

Brain.

The majority of the birds showed lesions in the cerebrum, cerebellum, optic thalamus and pons. These were always exquisitely focal in distribution. The massive and diffuse infiltrations seen in the nerves and spinal cord were never found in the brain. The lesions found were of the two types; either compact perivascular rings of small densely staining lymphoid cells, or submiliary nodules composed of lymphoid cells and paler elements, like those described as occurring in the white matter of the cord. In two cases, the lesions seen in the molecular layer of the cerebellum had a slightly different character. They were very sharply circumscribed, and composed almost exclusively of pale epithelioid elements showing a more or less definite radial arrangement. There were very few lymphoid cells in the nodules, which gave the impression of healing lesions.

The foci of cellular infiltration in the cerebrum were often very scarce, and in a few of the birds, even careful study of a number of sections at different levels failed to disclose them.

Although perivascular infiltration of the meningeal vessels was repeatedly seen, in only one case were there observed a more diffuse infiltration and thickening, accompanied by the presence of foamy, fat-containing cells.

Sympathetic Nervous System.

A systematic study of the sympathetic nervous system was not undertaken, but sympathetic ganglia and nerves were frequently included in the visceral sections, particularly in the section of adrenal (Fig. 12). In a number of cases, mononuclear infiltrations more or less intense were present in the ganglia and nerve trunks, so that it may be said with certainty that the disease involves the

sympathetic system. To what extent this affects the clinical picture of the disease, can only be surmised.

Thickening and cellular infiltrations have also been repeatedly found in the vagus nerves and in the nerve trunks accompanying the mesenteric vessels.

Eye Lesions.

In three birds showing partial or complete blindness, there was observed in the living animals a change in the color of the eye, from a yellow-brown to a bluish grey. This was readily explained by the microscopic changes. The iris was found to be thickened, edematous and infiltrated with wandering cells. These were of two distinct types—small, deeply staining lymphoid cells with little cytoplasm, and larger mononuclears resembling mammalian plasma cells, the nucleus being eccentric, and the cytoplasm strongly basophilic. These larger elements were aggregated into compact clusters, situated especially near the anterior surface. Occasional eosinophilic polymorphonulcears were also seen. There was slight edema, and a granular coagulum, and in some sections mononuclear cells of various types were present in the anterior chamber (Fig. 13).

The cornea was normal in structure. The retina was unaltered and the optic nerve heads showed only a minimal infiltration. Groups of lymphoid cells, however, were present in the conjunctiva and in the extraocular muscles.

Visceral Lesions.

Lungs.—No lesions were found which could be correlated with the cellular infiltrations present in the central nervous system. The only exception to this statement is in the case of chickens in which there were present lymphomatous masses in the abdominal viscera. In several of these, the lungs were also the seat of lymphomatous growths in the form of diffuse infiltrations in the walls of the bronchi and perivascular areolar tissue. In general, however, the lymphoid tissue which is normally found in the mucosa of the larger bronchi, was not hyperplastic.

Myocardium.—In the routine study of sections both from paralysed and normal chickens, one finds almost without exception, focal aggregations of wandering cells amongst the muscle fibers or in the loose connective tissue about the larger blood vessels. Sometimes these are composed entirely of lymphoid or larger mononuclear cells, with basophilic cytoplasm; at times there is a predominance of eosinophilic polymorphonuclears or of larger mononuclears with coarse eosinophilic granules—presumably, myelocytes. So frequently are these small cell aggregations to be found that it seems difficult to regard them as the expression of an infectious lesion, and still more difficult to believe that they are in any way related to the disease under consideration. It seems more probable that they are merely extramedullary leuco- and lymphopoietic foci.

On the other hand, we have encountered instances in which the myocardial infiltrations are so intense and widespread that they cannot be interpreted merely

as areas of heterotopic blood formation. Two types of changes have been seen. The first is a simple accumulation of small round cells crowding apart the intact muscle fibers, mantling the small arterioles and at times forming large oval aggregations which completely replace the muscle tissue over a considerable area.

The other type of lesion is more definitely inflammatory, and has been seen but once. It occurred in a Barred Plymouth Rock, which presented somewhat atypical lesions. There was iritis with blindness. The brain and cord were the seat of intense lesions of the usual character. The nerve trunks were not involved, but there was extensive perineuritis. A cross-section of the ventricle showed that a large part of the myocardial tissue was replaced by granulomatous areas containing a variety of cell forms—fibroblasts, giant cells, often surrounding remains of necrotic muscle fibers, lymphocytes, many of them fragmented and pycnotic, and rare eosinophilic polymorphonuclears. Although the individual muscle fibers in the affected areas were degenerated or necrotic, there were no larger areas of coagulative necrosis, nor tubercle-like lesions. The pericardium was invaded by the granulomatous tissue, leaving the overlying serous cells intact. It is of course possible that in this case the myocardial lesions, which are frankly inflammatory in type, may have been due to some other agent than that causing the lesions in the iris and central nervous system. It seems possible, however, that we are dealing with an unusual and atypical reaction to the same agent.

Liver.—In this organ, also, it is difficult to draw a sharp line between the normal and the pathological. Infiltrations of the periportal connective tissue with lymphoid cells, and not infrequently with eosinophilic, mono- and polymorphonuclear leucocytes, are present in practically every section examined, whether of normal or paralysed fowl. One finds also small, compact spherical masses of small mononuclears in any portion of the liver lobule, and these appear to be a normal feature of the chicken liver.

In certain of the paralysis cases, these infiltrations have become so massive as to leave no doubt as to their pathological nature (Fig. 14). They may even reveal themselves macroscopically as greyish areas obliterating the normal architecture of the liver lobule, and in several of the birds with large lymphomatous masses in the region of the ovary, the liver lesions were of a distinctly nodular character, grossly and microscopically suggesting a neoplasm. It would seem, therefore, that the same agent which is responsible for the infiltrations of the nervous system may at times incite the lymphoid tissue of the liver to abnormal and excessive growth.

Pancreas.—Interstitial lymphoid masses were present in a few cases. There were no lesions of the parenchyma.

Spleen.—We have found neither gross nor microscopic lesions.

Kidney.—The situation here is analogous to that in the liver. In every section of kidney, lymphoid infiltrations of variable extent may be found between the tubules. In certain cases, these may be so extreme as to separate widely or actually replace the parenchymal elements. In several of the birds with large

tumors arising about the ovary, there was direct extension into the kidney substance.

Adrenal.—Here again in routine sections, one finds very frequently compact aggregations of lymphoid cells in any portion of the gland, and such lymphoid foci, as in the adult human adrenal, may be looked upon as a normal histological feature of the gland. In many of the paralysed fowls, these infiltrations are very extreme. They appear to take their origin in the sympathetic ganglia and nerve trunks, which, in the chicken adrenal, are intimately incorporated with the glandular elements. We have already referred to the periadrenal nerves as very frequently and characteristically involved.

Ovary.—In the majority of the typical cases the ovary showed no pathological changes. In a certain proportion of the cases, this organ, as has been stated, gave origin to large lymphomatous masses of an invasive character.

Testes.—No infiltrations have been found in the testes of normal or paralytic fowl, and the male gonads seem to be, of all the viscera studied, the least favorable site for a localization of the lesions.

Our material does not provide a certain answer to the question as to whether the presence of the disease arrests or suppresses spermatogenesis. In the series of 60 spontaneous cases, there were but four males; three of these showed immature testes, but the exact ages were not known. In the fourth case, the testes were not examined. Amongst the positive cases uncomplicated by coccidiosis, which occurred in experimentally inoculated chickens, there were but two males, one killed on the 122nd day, the other killed on the 137th day, or 132 days after inoculation. The testes in these cockerels were also immature, the seminal epithelium being almost wholly undifferentiated. By the 122nd and 137th days, spermatogenesis should be well established. Selecting from our control material healthy cockerels killed on the 109th, 111th, 122nd, 125th (2), 128th (2), 131st, 135th and 137th days, we find active spermatogenesis, with ripe spermatozoa in the tubules, in every instance. It would seem that the disease in these two cases, which was of a severe type, definitely delayed or suppressed the onset of spermatogenesis. To what extent this is a characteristic effect cannot be decided without further data.

Alimentary Tract.—No lesions have been found in proventriculus or gizzard. The great majority of the birds, both in the spontaneous and experimental cases of the disease, showed no lesions of the intestinal tract. In a few instances in which there were lymphomatous tumors of other abdominal viscera, the intestinal mucosa, especially that of the duodenum, was thickened by diffuse infiltration of mononuclear cells. Lymphomatous plaques also occurred in the subserous tissue.

The relation of worm infestation and coccidiosis to the disease will be discussed below.

Thymus, Thyroid and Parathyroid.—Examined in a few of the cases, showed no alteration.

Visceral Lymphomata.

Visceral tumors, as has been stated, were found in six of the 60 spontaneously paralysed birds. Four additional cases occurred in a group of 120 chickens used in experiments.

Histologically, these were all identical in structure. They were composed of closely packed, small round cells with deeply stained nucleus and relatively scant cytoplasm. In the more actively proliferating areas where mitotic figures were extremely numerous, the cells were larger in size, the nuclei more vesicular, with distinct chromatin structure, and the cytoplasm more abundant (Fig. 15). Such cells are perhaps comparable to the larger lymphoid elements present in the centers of the germinal follicles.

With special stains (Foot's modification of the Bielschowsky method, *J. Lab. and Clin. Med.*, 1924, ix, 3), a delicate reticulum surrounding almost every individual cell was demonstrated.

The manner of growth of these tumors into adjacent healthy tissues was characteristic of that of the malignant lymphoblastomata. The cells invaded the stroma of the organs, pushing apart the parenchymal elements, and eventually replacing them completely. Thus, in the kidney, one found glomeruli and tubules widely separated by dense masses of the tumor cells, but showing little or no degenerative change. So, too, in the ovary, liver and lung, the normal elements remained intact until entirely replaced by the tumor tissue.

Necroses occurred in some of the larger tumor masses, in which case there usually remained a broad collar of living cells about the blood vessels.

The ovary has been the apparent site of origin of the main tumor mass in all instances, no males being included in our tumor cases. The growths have shown regional invasiveness into oviduct, kidney, adrenal, adjacent fat and muscle; in one case, the tumor grew directly through the vertebral column into the spinal cord. In several of the cases, there have been nodules in the liver, lungs, peritoneum and other situations. Whether these were true metastases, or whether the agent which incited the local proliferation of lymphoid cells in the ovary produced the same effect upon the lymphoid tissue in other situations, it is impossible to say.

What does seem plain, however, is the fact that these visceral lymphomata—in spite of their apparently neoplastic appearance—are but a manifestation of the same unknown agent which brings about the infiltrations in the nervous tissues. This is very clearly shown in the case alluded to, in which the ovarian tumor had penetrated the

spinal canal. In the region where the growth had invaded the cord, the closely packed cells with numerous mitoses destroyed and replaced the nervous tissue after the manner of a malignant neoplasm. But as one passed away from this site, the cellular infiltrations became distinctly perivascular, and in the remainder of the cord and brain, the lesions were identical with those found in the paralysed birds in which no tumorous growth was associated with the nervous lesions. The scattering infiltrations of the peripheral nerves were also like those usually found in the paralysis chickens, and, as is shown by our tabulated protocols, they were present in every case of visceral lymphomatosis included in our material.

We shall reserve for our final discussion the interpretation of this interesting association.

Because of the obvious resemblance of the lesions to leucemic infiltrations, careful attention was given to the blood within the vascular channels. In no instance did there appear to be an excessive proportion of lymphoid cells or other leucocytic elements, even in situations where the infiltration of the tissues was most intense. Furthermore, a study of stained smears, from paralysed birds and healthy controls, showed no significant differences in the percentage of the various types of leucocytes. Time did not permit a systematic study of the blood, but even with the data at hand, it seemed that the lesions were not associated with a leucemic blood picture.[1] That the cells forming the infiltration are not derived wholly from the circulating blood is shown by the fact that the capillaries in the infiltrated areas contain no excessive number of leucocytes, and the occurrence of numerous mitoses is proof that the cells proliferate *in situ*.

Lesions Encountered in Non-Paralysed Chickens.

One of the puzzling facts which has made the experimental study of the disease very confusing and difficult, is the frequent occurrence of lymphoid infiltrations in the nervous tissue of chickens which are not paralysed, and which betray no clinical evidence of disease. While it is true that in most of these apparently healthy birds, the

[1] Through the kindness of Dr. H. F. Pierce, we have had the opportunity to compare our blood smears with two cases of fowl leucemia, from the material of Professor Ellermann of Copenhagen.

lesions are trifling, and indeed may fall within the range of normal variation, in others they are so intense and widespread that they cannot but be regarded as pathological. It has thus been of interest to study carefully birds from various sources, so as to obtain a clear idea of what one may consider "normal."

In 32 non-paralysed birds of various ages, and from various localities, lymphoid infiltrations of the nervous system were found in ten—approximately one-third of the cases. The brain was most frequently affected, the spinal cord next and in only one case were infiltrations present in the peripheral nerves.

Fifteen of the 32 examined came from farms or experiment stations where paralysis had occurred during the present year.[2] Lesions were

TABLE I.

Occurrence of Lesions in Non-Paralysed Chickens.

Source	Number examined	Lesions			
		Brain	Cord	Dorsal ganglia	Nerves
From localities where paralysis occurs.....	15	6	5	1	1
From localities where paralysis is absent....	17	6	5	2	—
Total.....	32	12	10	3	1

found in six of these fifteen; or if we exclude two which were less than 2 months old, in five out of seven.

Seventeen birds were obtained from sources where paralysis is definitely stated not to occur. In this group, infiltrations occurred in five birds.

From these small numbers, it is impossible to draw conclusions as to the comparative incidence of lesions in non-paralysed chickens from affected and unaffected localities. One may, however, make the definite assertion that perivascular lymphoid infiltrations of the brain and cord are often found in seemingly healthy chickens from sources where paralysis is known not to occur, and in certain instances they may be of considerable intensity and wide distribution.

There is no qualitative distinction between the infiltrations found

[2] In one instance, no data as to the source of the chicken were obtainable.

in the healthy birds and in those with frank paralysis; and it would seem that only when the infiltrations implicate the peripheral nerves to such a degree as to block motor impulses, or to bring about actual destruction of nerve fibers, do paralytic symptoms become manifest. One is driven to the conclusion that the condition exists in masked form in a very considerable number of seemingly healthy chickens. We cannot, from our experience, state whether or not these birds with unrevealed lesions are on their way to develop obvious paralysis. The fact that such cases have been found on farms where paralysis does not occur suggests that the birds with mild lesions are immune to the more severe forms of the disease.

The question may be raised at this point as to whether the presence of lymphoid tissue about the meningeal and cerebral vessels may not, after all, be a normal histological feature in the chicken. It is not easy to reach a definite decision as to this, and we have found no reference in the literature to guide us. In a certain number of birds, a thorough and systematic study of numerous sections from all parts of the brain and cord has failed to disclose any perivascular lymphoid tissue. On the other hand, as has been indicated, we have frequently found small lymphoid nodules in the walls of the cerebral and spinal vessels, and in the dorsal root ganglia, in well nourished, healthy stock from unaffected sources. Such small compact focal masses of lymphoid cells have been arbitrarily regarded within the range of "normality." The line between these and the milder grades of pathological infiltration, it must be admitted, is not a sharp one, and the difficulty becomes especially troublesome in attempting to interpret the effects of experimental inoculation.

Bacteriology. Attempts to Demonstrate an Infective Microorganism.

Efforts to identify an infecting organism in smears and sections of the brain, spinal cord, peripheral nerves and heart blood have thus far proved unavailing. Sections have been stained by the Gram-Weigert method, with toluidine blue, Giemsa's method, Wright's stain, Levaditi's method and Jahnel's modification of Levaditi's method for spirochetes. None of these procedures has disclosed any structures which can with reasonable certainty be recognized as bacterial or protozoan. We have had in mind the possibility of

rickettsial infection, and we have carefully searched our preparations for intranuclear or cytoplasmic inclusions.

We have made numerous attempts to obtain cultures of organisms from the nerve tissue of paralysed chickens. Among the media used were peptone and meat extract broth, with and without addition of glucose; plain and glucose agar, with and without addition of inactivated chicken serum or whole blood, and adjusted to various pH ranging from 7.4 to 6.0; beer-wort agar; chopped chicken meat broth, and potato. Cultures were incubated at 37° and at room temperature. Following a suggestion of Dr. Rettger, we tested the possible stimulating effect of an atmosphere of approximately 10 per cent CO_2. Although diphtheroids, sarcinæ, yeasts and torulæ were occasionally obtained, none of the strains isolated appeared to be of etiological significance and they were regarded as accidental contaminants.

TABLE II.

	Scrapings + Sections +	Scrapings − Sections −	Scrapings + Sections −	Scrapings − Sections +
Duodenum	10	48	2	1
Cecum	12	7	3	0

Fowl Paralysis and Coccidiosis.

The view that coccidial infection may play a rôle in the etiology of paralysis has been expressed by a number of poultrymen and experiment station workers. Thus Beach and Davis (7) describe and picture (Fig. 3 of their article) paralysis as a form of chronic coccidiosis occurring in older birds between the ages of 4 and 8 months. The authors do not refer to "fowl paralysis" as an independent disease, but appear to regard it merely as a late symptom of the coccidial infection. The opposite point of view is represented by May, Tittsler and Goodner (5), who state that coccidia are frequently present in paralysed birds but not in all cases; and that birds may die from coccidiosis without exhibiting symptoms of paralysis.

Our material, we believe, affords a decisive answer. As a routine, sections were taken from duodenum and ceca in practically every case examined; during the past summer, we have also checked the

histological findings by the examination of fresh scrapings of the duodenal and cecal mucosæ. There is, in general, close agreement between the findings of coccidia in fresh preparations and in the stained tissue sections. This is brought out in Table II.

Thus, in only six out of 83 examinations was there any discrepancy as regards the presence or absence of coccidia between fresh preparations and the stained sections. From this one may conclude that the absence of coccidia in histological sections is strong presumptive evidence against the existence of coccidial infection.

In 60 spontaneous cases of paralysis, coccidia were found in the sections in ten, or roughly, 16 per cent; in 33 non-paralysed birds from various sources, coccidia occurred in four, or approximately 12 per cent. Obviously, there is no significant difference in the two groups.

Our experimental material is also instructive. In Experiment VI, only one of thirteen cases of paralysis was infected with coccidia. In 83 chickens which did not develop paralysis, coccidial infection occurred in eleven, or roughly, 13 per cent, about the same ratio as in the non-experimental material. In Experiment VII, coccidiosis appeared in both inoculated and control animals during the 3rd and 4th months. Of the four positive cases, two showed coccidia, and two were free. Fifteen of the non-paralysed chickens were affected, and 33 were free. The higher incidence of coccidiosis in this experiment may be attributed to the crowding and close confinement.

From the foregoing data, it is very evident that there is no correlation whatever between infection with *Eimeria avium* and the incidence of fowl paralysis.

DISCUSSION.

The disease as thus described is one with striking and characteristic pathology, and one which has no close counterpart, so far as we are aware, in mammalian pathology. The essential changes are cellular infiltration of the nervous tissues, peripheral nerve trunks of the extremities and viscera, spinal ganglia and cord—less strikingly of the brain and rarely of the cranial nerves. In the nerves, the cells, which are predominantly of the lymphoid type, but mixed with plasma cells and clasmatocytes, push apart the nerve fibers and

eventually cause considerable destruction of the myelin. The axis cylinders tend to be preserved so that it is not surprising that paralysis is rarely complete. That sensation as well as motion is affected, is evident from the involvement of the spinal ganglia and posterior spinal roots in most of the cases.

In the cord and brain the infiltrations are largely confined to the vicinity of the vessels and the lesions often bear a close resemblance to those found in various types of mammalian encephalomyelitis. Although these lesions of the nervous system are the most distinctive features of the disease, and probably the ones responsible for the more obvious clinical symptoms, it seems probable that the massive lymphomatous infiltrations and growths occurring in the ovary and other viscera are integral features of the disease. They have been present—and we refer here only to the grossly evident, tumor-like growths—in 10 per cent of our spontaneous cases; and they have developed, as will be pointed out in a following paper, in fowl experimentally inoculated with suspensions of brain and cord from paralysed fowl. Although these masses often take on the character of a malignant neoplastic growth, pushing aside and invading normal structures, their cytological composition, and the frequent continuity of the neoplastic tissue with the characteristic accumulation in the nervous tissues make it highly probable that the same agent or stimulus is concerned.

This brings up the difficult questions of the relation of this disease to the malignant lymphoblastomata on the one hand, and to the leucemias and pseudoleucemias on the other.

Numerous tumors in fowls variously termed lymphoma, lymphosarcoma and small round cell sarcoma have been described.

In certain cases these have been associated with a leucemic blood picture, and considerable evidence has been presented by Ellermann and Bangs (8), Ellermann (9), Hirschfeld and Jacoby (10), Schmeisser (11) and others that the leucemia of fowls is a transmissible disease. Of the non-leucemic lymphomatous cases, we may cite those reported by Hathaway (12), Butterfield (13), Tyzzer and Ordway (14), Wernicke (15), Joest and Ernesti (16), Pentimalli (17), Beatti (18), Hobmeyer (19), Elsner (20), Schuchmann (21) and Michalka (22). It is unnecessary to review this literature in detail. That the disease which we have studied is not a form of leucemia has been shown by the examination of blood, smears and by the absence of enlargement of the spleen and liver, which has been a characteristic feature of the reported cases.

It has seemed to us that the diagnosis of "pseudoleucemia" and "leucanemia" used by Ellermann to describe cases in which lymphoid or myeloid infiltrations were present in the viscera without a leucemic blood picture, must be accepted with considerable reserve. Ellermann regards such cases as being produced by the injection of his leucemic virus, and includes them amongst the positives. In our experience, such visceral infiltrations occur in practically every chicken, although there is considerable individual variation in the amount of lymphoid or myeloid tissue present in the different organs. Neither Ellermann nor subsequent workers with fowl leucemia appear to have made a systematic study of control "normal" material. It is regrettable also, that none of the investigators of fowl leucemia has included a study of the nervous system. We cannot state, therefore, whether brain, cord and nerves in this disease may show lymphoid infiltrations such as we have described in fowl paralysis. That such infiltrations may be found in human leucemias, is well known; 30 such cases have recently been collected from the literature by Fried (23). That they cannot involve the peripheral nerves to the degree found in the paralysis of chickens, is obvious from the absence of neuritic symptoms in leucemic birds.

Whether some of the cases which have been described as "pseudoleucemia" may have been instances of paralysis with visceral infiltrations, it is impossible to say. The literature contains no references to a careful study of the nervous system in these cases. Only in the case reported by Beatti from Buenos Aires, is it recorded that the chicken during life could not stand erect, but rested on the right foot, using the left wing as support. The cause for this was a lymphoblastomatous mass in the lumbosacral region, infiltrating the kidney, and invading the lumbosacral plexus. Tumor cells were distributed among the nerve fibers. There was no leucemic blood picture. It is probable that this was a true case of fowl paralysis, of the type associated with regional lymphomatous growth, and that other cases in the literature, if the nervous system had been carefully studied, would have revealed themselves as examples of this disease.

A striking association between paralysis and a high tumor incidence (12.9 per cent) was in evidence also in the "farm flock" cited by Schneider (1926) (24). From a study of material supplied to us, we have learned that paralysis was prevalent in this particular flock.

In the 60 cases of spontaneous paralysis here reported, macroscopic ovarian tumors, usually with lymphomata also in the other viscera, were found in six instances or 10 per cent and we have found a similar high incidence in our experimental chickens. The abnormally high

rate in our paralysis material is in itself suggestive of a correlation, and we have given pathological evidence for the view that the lymphomatous masses in the ovary and other viscera are no different in their cytological composition from the infiltrations found in the nervous tissues. Our material does not permit us to state whether all cases of visceral lymphomatosis in fowls are accompanied by lesions of the nervous system, and this question demands further investigation.

Our studies at least indicate that the agent which is responsible for the pathological changes in the nervous system is not exclusively neurotropic; it may in certain cases stimulate the proliferation of lymphoid cells in the viscera, and to a degree which makes them take on the morphological character of a neoplasm. One of the most interesting and as yet, wholly obscure phases of our problem, is the undoubted transition between the lesions which histologically appear to be inflammatory and those which assume the character of a true neoplasm.

Nomenclature.

No satisfactory designation for this disease has yet been suggested. The term "polyneuritis" used by Marek in 1907, is open to several objections—it is not sufficiently inclusive, leaving out of account the lesions of the cord, ganglia and brain; and it does not distinguish this disease from the nutritional polyneuritis gallinarum, which is etiologically and pathologically unrelated. Kaupp, and May, Tittsler and Goodner speak simply of "paralysis of the domestic fowl"—a term which is unsatisfactory, since paralysis is but a symptom, and one which is inconstantly present in this disease, and which may be due to a variety of other disorders. The term "neuritis" is used by Doyle, and is open to the same objections as the term "polyneuritis." Van der Walle and Winkler-Junius suggest the name "neuromyelitis gallinarum" to differentiate this condition from the nutritional polyneuritis.

It is perhaps not possible to find an altogether satisfactory name for this disease until the cause of it shall have been definitely established. The outstanding pathological features, as we have seen, are the lymphoid infiltrations of the peripheral nerves, and the frequently associated lymphomatous growths in other situations. We wish, therefore, to propose tentatively the term neurolymphomatosis gallinarum, as indicating more specifically than any of the

terms previously suggested, the most striking pathological features of the disease.

CONCLUSIONS.

1. Fowl paralysis (neurolymphomatosis gallinarum) is a disease entity, with characteristic clinical and pathological features.

2. The disease occurs in all parts of the United States, Holland, Austria and probably South America.

3. The disease appears to be endemic in certain foci. Having once appeared, the disease tends to persist through successive years.

4. It occurs with about equal frequency in both sexes; all common breeds may be affected.

5. Symptoms appear between the 3rd and 18th months. Typical clinical cases have not been observed outside of these limits.

6. The conspicuous symptoms are (*a*) asymmetrical, partial and progressive paralysis of the wings and both legs, and rarely of neck muscles; (*b*) occasional grey discoloration of iris, with blindness. Nutrition is usually preserved.

7. The duration is variable; the outcome is usually fatal, but spontaneous recovery may rarely occur.

8. The principal pathological changes are found in the nervous system. In the peripheral nerves, the essential feature is an intense infiltration of lymphoid, plasma cells, and large mononuclears. This is accompanied by a myelin degeneration in the more advanced lesions, but the cellular infiltrations appear to precede the degenerative changes. In brain, cord and meninges, there are similar infiltrations predominantly perivascular.

Infiltrations of the iris with lymphoid and plasma cells are found in the cases showing gross discoloration of the iris.

Visceral lymphomata, originating usually in the ovary, are associated in a certain percentage of the cases. Evidence is presented in favor of the view that this association is not accidental, and that the lymphomata are a manifestation of the disease.

9. Infiltrations of the spinal cord and brain, rarely of the peripheral nerves, are frequently present in birds showing no clinical symptoms. These are interpreted as mild cases of the same disease.

10. No microorganisms of etiological significance have been demonstrated in the tissues or by cultural methods.

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EXPLANATION OF PLATES.

PLATE 6.

FIG. 1. Fowl paralysis. Showing weakness of legs and drooping of left wing.

FIG. 2. Fowl paralysis. Terminal stage, complete prostration.

FIG. 3. Fowl paralysis. Dissection of spinal cord and brachial plexus. On the right side there is massive nodular thickening of the nerve trunks, extending into the corresponding segment of the cord.

PLATE 7.

FIG. 4. Section through sciatic nerve. Intense mononuclear infiltration of upper bundle, moderate infiltration of lower.

FIG. 5. Fowl paralysis. Infiltration of peripheral nerve. High power.

PLATE 8.

FIG. 6. Fowl paralysis. Section of sciatic nerve, showing intense cellular infiltration. Note nodular foci.

FIG. 7. (*A*) Brachial plexus. Normal. (*B*) Advanced paralysis. Myelin degeneration, fibrosis, mononuclear infiltration.

PLATE 9.

FIG. 8. Typical paralysis. Sciatic nerve. Pal-Weigert stain, showing advanced degeneration of myelin sheaths.

FIG. 9. Typical paralysis. Sciatic nerve stained by the Rio del Hortega silver carbonate method. Persistence of neuraxones in infiltrated nerve.

PLATE 10.

FIG. 10. Low power photograph of spinal cord with attached ganglia and nerve roots, showing asymmetrical enlargement and extension of infiltration into adjacent tissue of cord.

FIG. 11. Spinal ganglion, showing lymphoid infiltration between ganglion cells.

PLATE 11.

FIG. 12. Fowl paralysis. Infiltration of sympathetic ganglia and nerves about adrenal.

FIG. 13. Section through cornea, anterior chamber of eye, iris and lens. Note cellular infiltration of iris.

PLATE 12.

FIG. 14. Fowl paralysis. Massive lymphoid infiltrations in liver.

FIG. 15. Fowl paralysis. Section of ovarian tumor, associated with typical lesions of the nervous system. Note numerous mitoses. High power.

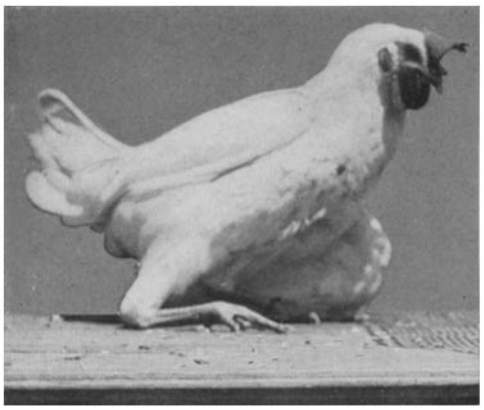

FIG. 1.

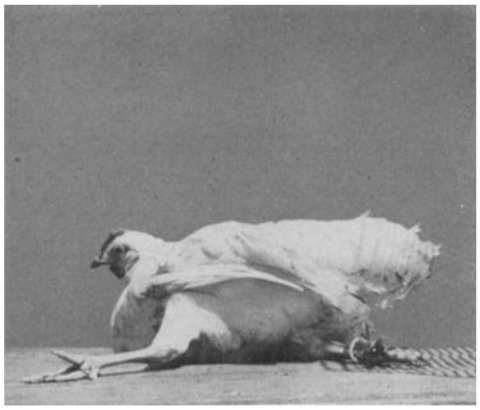

FIG. 2.

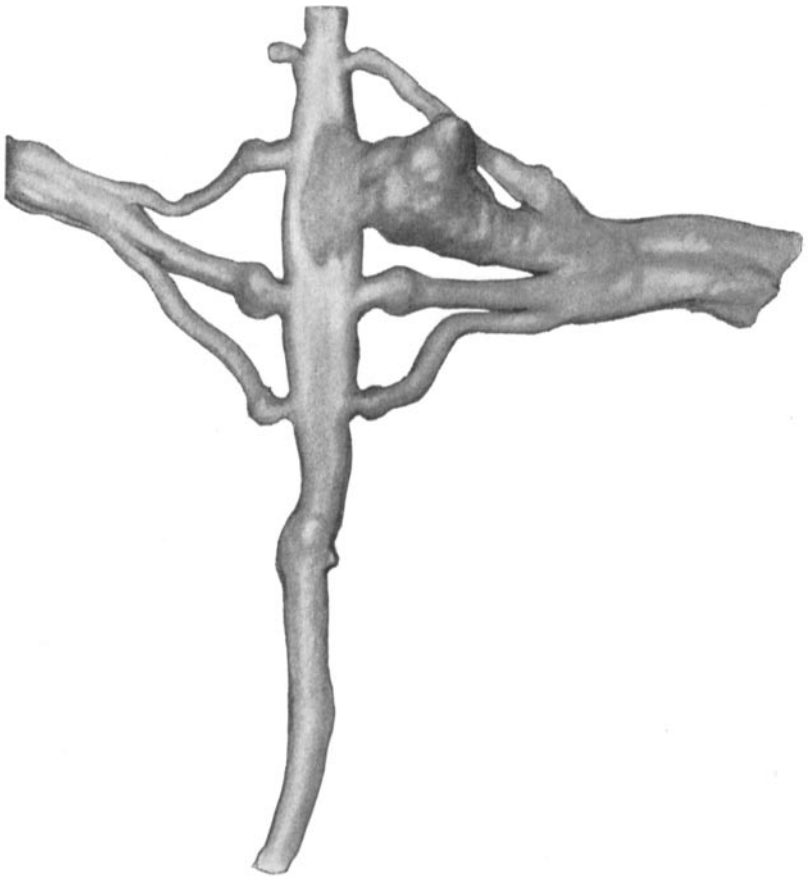

FIG. 3.

(Pappenheimer *et al.*: Fowl paralysis. I.)

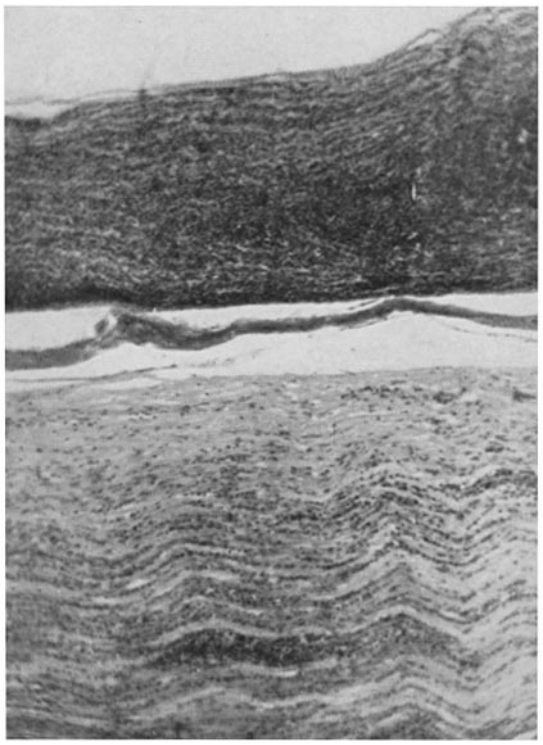

FIG. 4.

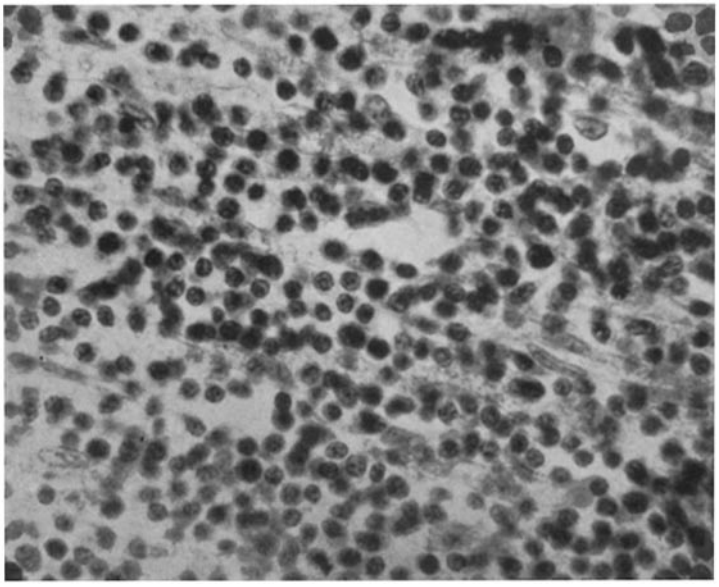

FIG. 5.

(Pappenheimer *et al.*: Fowl paralysis. I.)

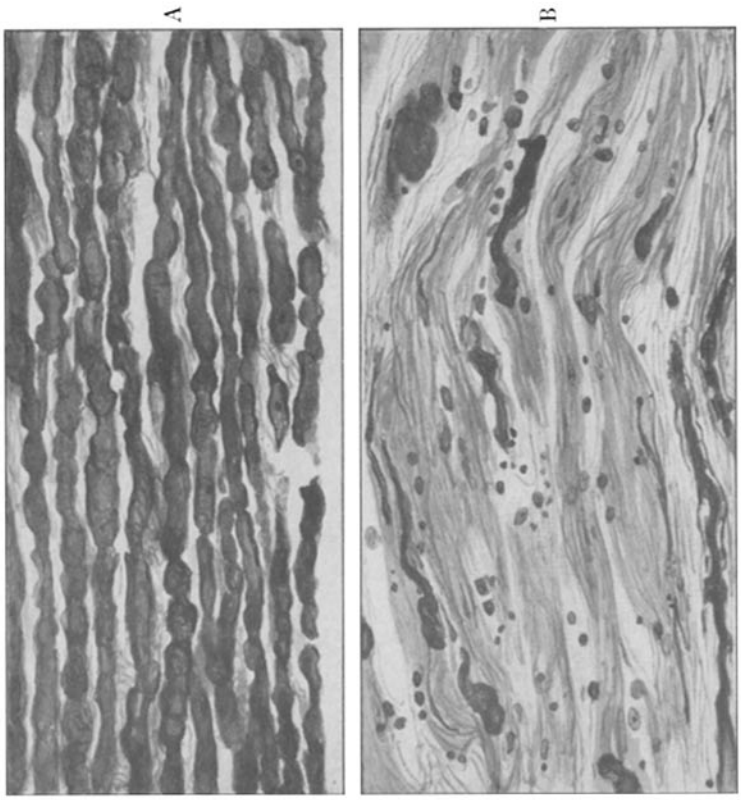

FIG. 7.

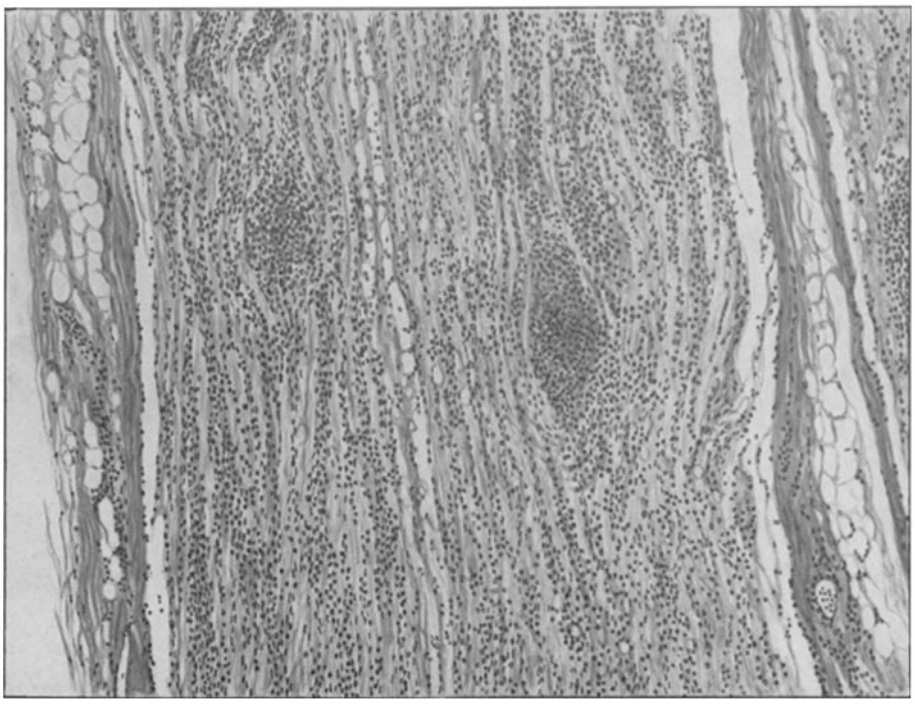

FIG. 6.

(Pappenheimer *et al.*: Fowl paralysis. I.)

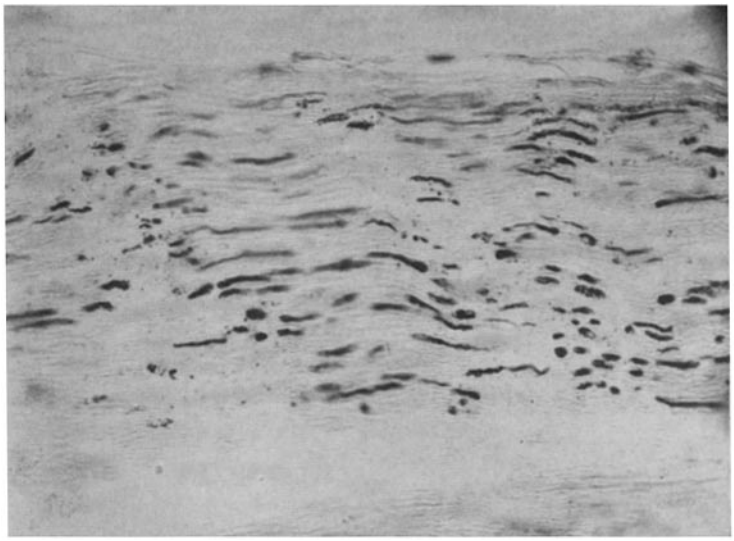

FIG. 8.

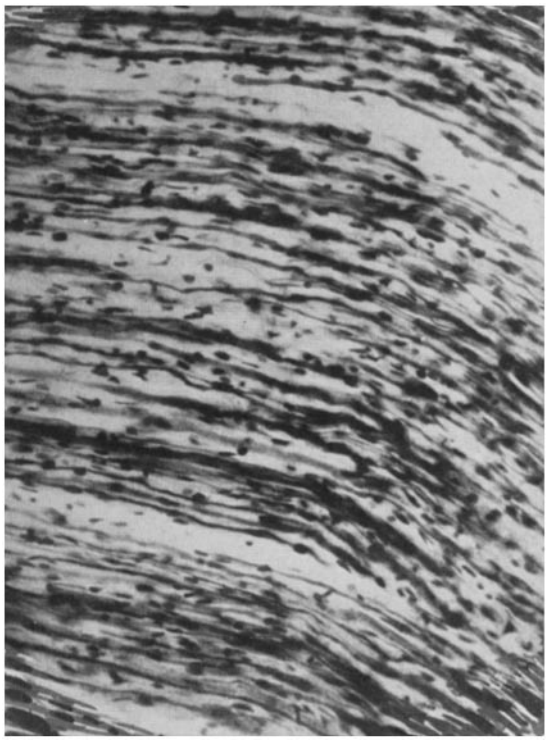

FIG. 9.

(Pappenheimer *et al.*: Fowl paralysis. I.)

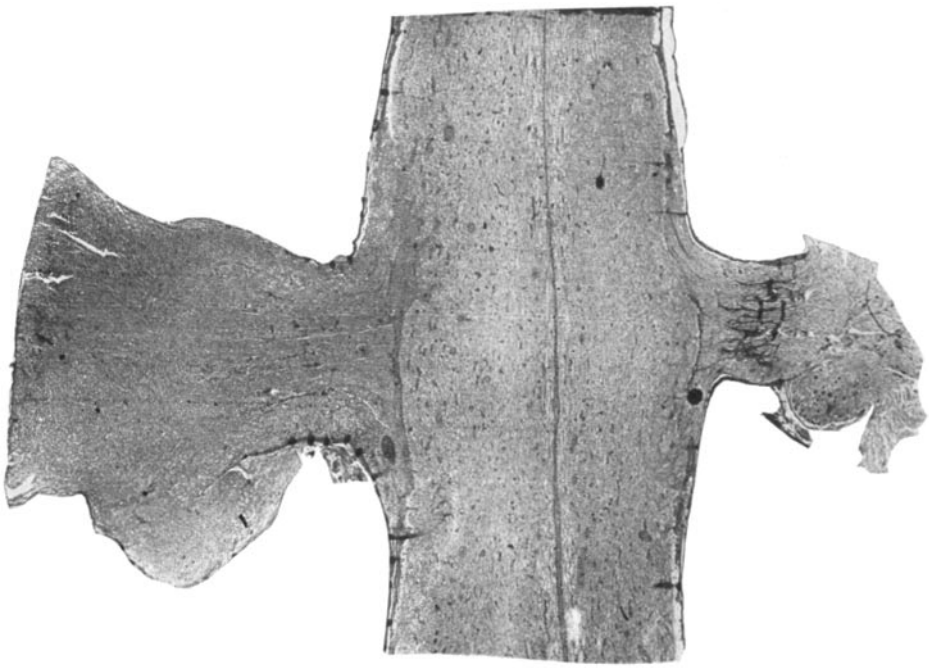

FIG. 10.

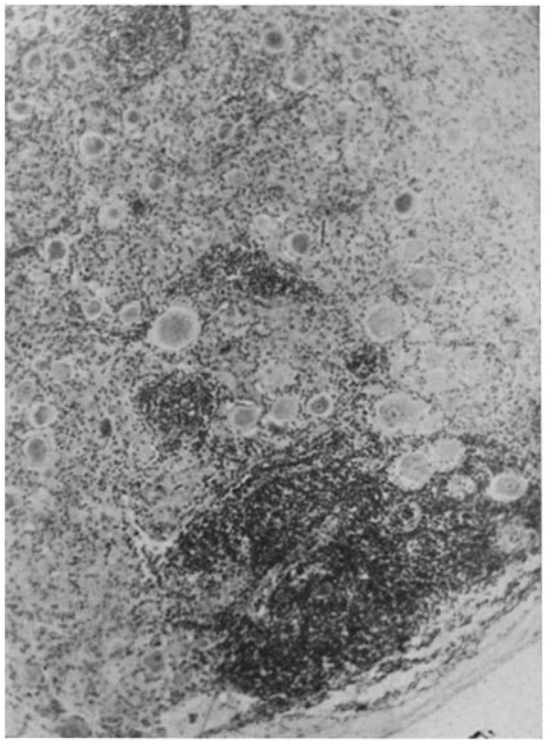

FIG. 11.

(Pappenheimer *et al.*: Fowl paralysis. I.)

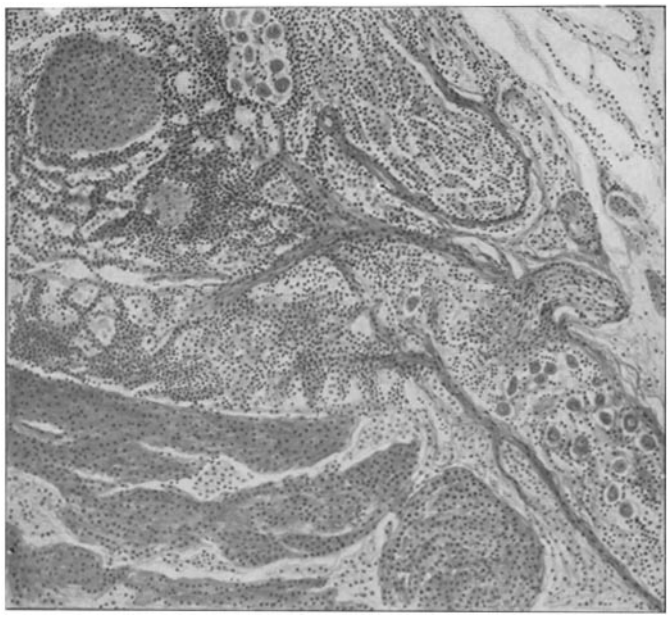

FIG. 12.

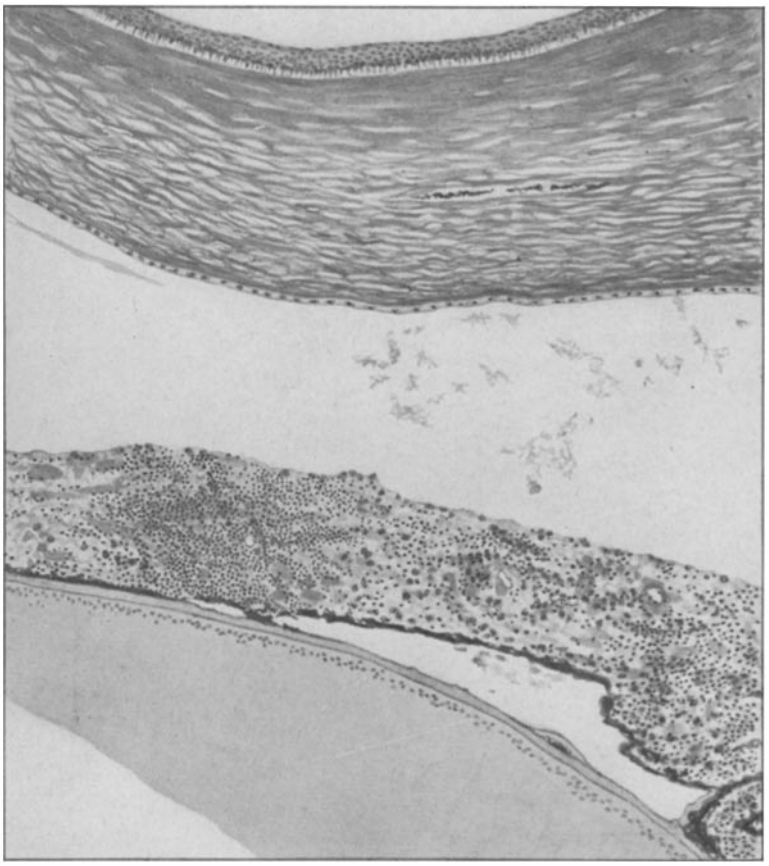

FIG. 13.

(Pappenheimer *et al.*: Fowl paralysis. I.)

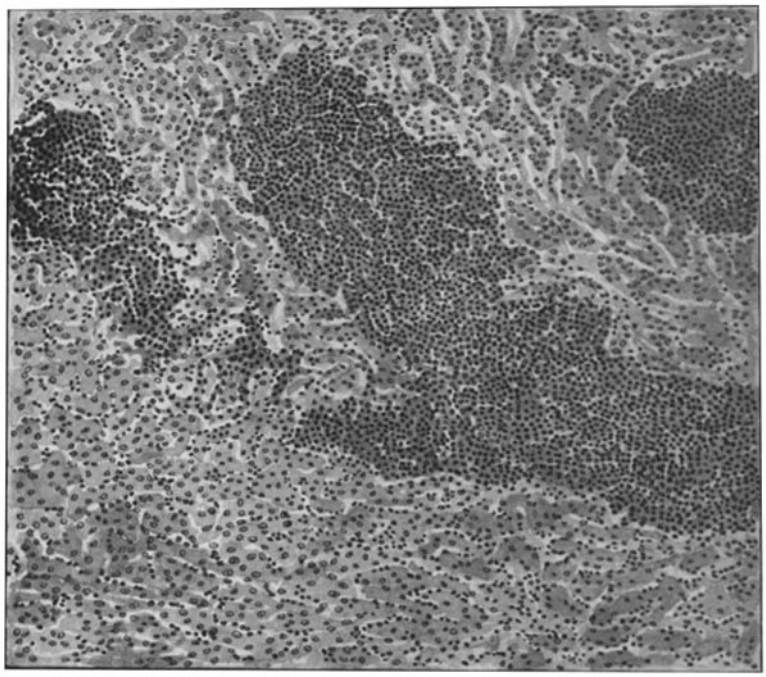

FIG. 14.

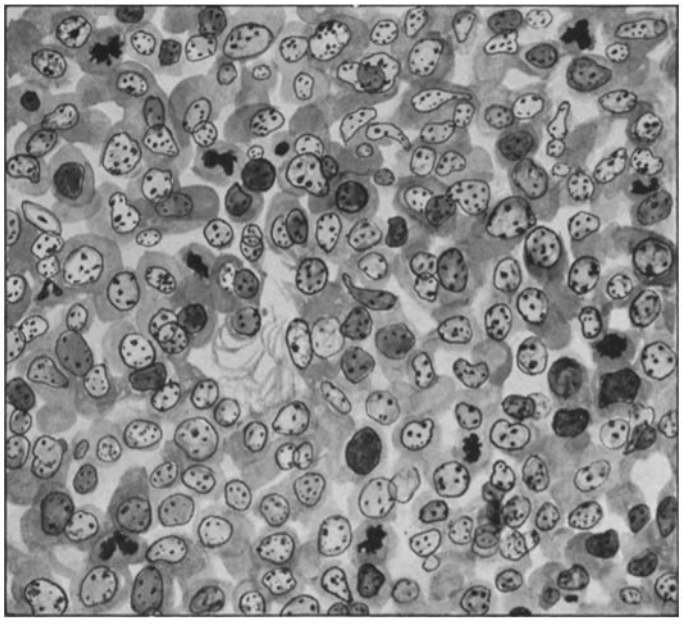

FIG. 15.

(Pappenheimer *et al.*: Fowl paralysis. I.)