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Preliminary Studies on Neurolymphomatosis and Some More or Less Related Diseases

By Charles S. Gibbs

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A group of poultry **diseases, many probably of an infectious** nature, are taking a heavy toll from **Massachusetts flocks**. It is conceivable that most of these disorders may be controllable if properly understood. This investigation was undertaken to determine some of the fundamental characteristics and causes of so-called "fowl paralysis" which is rapidly becoming economically very significant.

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PRELIMINARY STUDIES ON NEUROLYMPHOMATOSIS AND SOME MORE OR LESS RELATED DISEASES

By Charles S. Gibbs,¹ Research Professor of Veterinary Science

Neurolymphomatosis, generally referred to as "fowl paralysis," is quite common in the United States and Europe, and has been recognized in Massachusetts since Kaupp (1921) and Pappenheimer (1926) described it. All breeds of chickens are susceptible, but turkeys, geese, ducks, other poultry, and wild fowl are not known to be affected. Ordinarily, it is a disease of pullets and cockerels of three to eight months of age, and the heaviest losses usually occur in the late summer and early fall, although there are exceptions to this rule.

The most noticeable symptoms are paralysis of either one or both legs or the wings, and blindness marked by a greyish appearance of the eye. The comb and wattles are usually of normal color, and the birds have a good appetite as long as they are able to eat. The symptoms may appear suddenly or gradually, and the birds may die in a few weeks, or the condition may be prolonged over a period of several months. Very few cases of neurolymphomatosis ever recover.

Diagnosis is usually made by observation of the disease in a flock and by necropsy of dead birds. A definite technique for the macroscopic diagnosis has been worked out by Jungherr (1933). The lesions are located in the nervous system and contiguous tissues, such as the blood, visceral organs, muscles, etc. Since the tissue changes are very difficult to recognize in some cases, the history of the outbreak, the symptoms of the sick birds, and the general appearance of the flock are of great aid in differentiating neurolymphomatosis from some other diseases and obtaining a satisfactory diagnosis.

The real cause of neurolymphomatosis is unknown. There have been considerable speculation and some laboratory study on this phase of the disease, but without any definite conclusions. In other words, to determine the etiology of neurolymphomatosis, the investigator should have at his disposal a wellequipped laboratory and a poultry plant with facilities for hatching and isolation of different groups of birds. The cost of such an undertaking has, up to the present, precluded any satisfactory demonstration of the etiological agent.

Etiology

Attempts to transmit neurolymphomatosis in this study have not been uniformly successful. In all, 400 chickens have been inoculated subcutaneously, intramuscularly, intraperitoneally, and intracranially with neoplastic suspensions of diseased nerves and tumor tissue in physiological saline solution without showing any symptoms of disease. Also 400 uninoculated controls from the same flocks and reared under the same conditions did not reveal any lesions of either neurolymphomatosis or lymphocytomatosis at necropsy. In addition, 60 chickens

¹The writer extends his sincere thanks to Oliver S. Flint, Department of Veterinary Science, Massachusetts Agricultural Experiment Station, for doing the photographic work for this bulletin; and to the Poultry Department of the College and Experiment Station, which made this study possible.

were inoculated intravenously with Berkefeld V filtrates of the neoplastic lesions of nerve tissue, which had been ground up in a sterile mortar with a sterile pestle and sand. Also, fresh lymphocytomas were transplanted into the visceral organs, muscles, and skins of 140 chickens, by means of a sterile trocar. These chickens were held for six months without showing any symptoms or lesions of lymphocytomatosis, a disease closely related to, if not the same as, neurolymphomatosis.

The stock for these etiological experiments was selected from 10 different flocks and an attempt was made to avoid chickens previously exposed to the disease, as such exposure might lead to erroneous results. Active symptoms of neurolymphomatosis do not appear for two or three months after infection has taken place. This precaution was deemed necessary because in some of the earlier experiments of the writer the same number of controls as inoculated birds came down with symptoms and lesions of neurolymphomatosis, indicating that the birds had previously contracted the disease.

Also it was found in the course of these experiments that chickens inoculated intraperitoneally and intramuscularly with suspensions of neurolymphomas, pyogenic bacteria, and tar developed tumors of the peritoneum, visceral organs, mesentery, muscles, celiac plexus, and small nerves of the visceral organs indistinguishable from lymphocytomas and neurolymphomas of spontaneous origin. Ruptured egg sacs and oviducts were believed to be responsible for similar conditions in spontaneous cases by first establishing peritonitis, which ultimately led to tumor formation.

The results of these laboratory transmission experiments are inconclusive, and in view of the work that has been done by Seagar (1933), Lerche and Fritzsche (1933), and McIntosh (1933) should not be considered final until further work has been done, because there are so many unknown factors associated with the etiology of neurolymphomatosis and lymphocytomatosis that it is doubtful whether any investigator has succeeded in keeping them all under control. However, persistent trial and error, cf which this study is a beginning, should not only reveal the etiology of these two diseases but show their relation, if any, to lympholeukosis.

The Development of Histological Methods

Plenty of material from spontaneous cases has been available for hematological and histopathological studies. It has been possible to select material from flocks of which the history was known, and the studies for the most part have been confined to one of these flocks for a period of four years. The first two years were devoted largely to developing staining methods, acquiring a familiarity with the histological elements in poultry pathology, and differentiating neurolymphomatosis from other diseases to which it appeared at first to be more or less related in spontaneous cases.

The blood staining methods suggested by Furth (1931), the bone marrow studies of Forkner (1929). and Mallory's (1921) eosin and methylene blue and phosphotungstic acid hematoxylin stains for tissues were especially valuable in the development of histological methods for this study. Wong's (1928) method for the determination of hemoglobin was used. The blood clotting time was estimated by the method of Wright (Morse and Looney, 1927).

It was found that the thrombocytes could not be counted with any degree of accuracy unless supravital staining was employed. Hence the counts for thrombocytes are omitted in this bulletin.

The Pathological Cell in Neurolymphomatosis and Lymphocytomatosis

First comparative studies were made of normal birds and those showing neurolymphomatosis with gross lesions, avian paralysis without gross lesions, myeloleukosis, erythroleukosis, and lymphocytomatosis. In distinction from Patterson et al. (1932), little difficulty was experienced in differentiating neurolymphomatosis from erythroleukosis and myeloleukosis, while lympholeukosis appeared to be closely related. The large lymphocyte observed in the infiltrated nerves of neurolymphomatosis, Fig. 1, appears to be similar to the pathological cell found in lymphocytomatosis, as stated by Matthews and Walkey (1929).

In this study there was evidence of perivascular infiltration in which the pathological cells passed in the blood and lymph until they filled the spaces between the nerve fibers, as shown in Fig. 2. The walls of the arteries and veins were

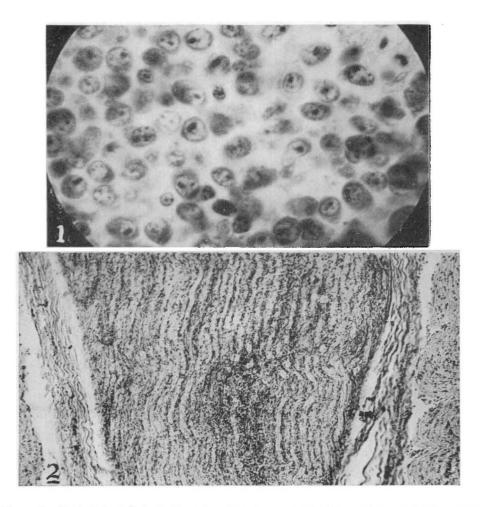


Figure 1. Pathological Cells in Neurolymphomatosis, showing the division of the nucleus and the characteristic concentric arrangement in some cells. (Magnified 1100 diameters.) Figure 2. Infiltration of Pathological Cells around Nerve Fibers. (Magnified 100 diameters.)

not destroyed, and the nourishment in the vascular system was diverted from the nerve fibers to the pathological cells, which appeared to increase very rapidly by further infiltration and mitosis until all of the neutral staining tissues were replaced by basic staining neoplastic cells. In other words, the nerve fibers appeared to die for the lack of nourishment and were finally dissolved and carried away by the blood stream, as the pathological cells at first surrounded them and later replaced them entirely. This is the physiological development of the neurolymphoma in the nerve of the fowl as observed histologically in these studies. Enzymes may have played a part in the destruction and absorption of the nervous tissue, but their presence could not be demonstrated in either living or dead tissues.

This tumor-like growth of the nervous system is what Pappenheimer et al. (1929) have called a neurolymphoma, and it leads to the pathological condition called neurolymphomatosis by Johnson (1932). These names are retained in this study and have already been used several times. Since the neurolymphoma tissue cannot be stimulated like nervous tissue, it naturally follows that muscles, organs, and other structures supplied by nerves suffer as a result of interference in normal functions, and symptoms of disease arise.

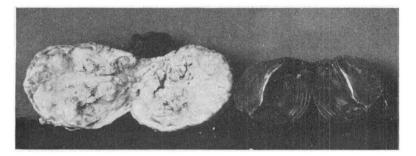


Figure 3. Lymphocytoma of the Gizzard. Left, diseased gizzard. Right, normal gizzard.

The fact that the neoplastic cell involved in neurolymphomatosis has been found in the blood and lymph shows that there is ample opportunity for it to find its way into the general circulation and be involved in metastatic conditions. Metastasis, as well as infiltration, has been found in this study and will be pointed out a little later.

Before proceeding further it is necessary to indicate that the neurolymphoma resembles the lymphocytoma in physical appearance and consistency, as well as in possessing the same type of pathological cell. Both develop from incipient perivascular infiltrations into the tissues. In advanced cases the neurolymphoma is usually smaller than the lymphocytoma. This is probably due to the fact that the blood and lymph spaces in the visceral organs and muscles are larger than in the nervous tissues, and growth proceeds more rapidly because more cells are able to crowd in and nourishment is more readily brought to them. Lymphocytoma of the gizzard is shown in Fig. 3. In advanced cases of lymphocytomatosis, in which the neoplasms had reached comparatively large sizes, anemia was present. Neurolymphomatosis was never accompanied by noticeable symptoms of anemia unless the birds were so paralyzed that they could not reach feed and water, or nerves supplying the digestive system were affected and malnutrition had set in.

In some cases lymphocytomas were found completely surrounding nerve trunks without showing any evidence of invasion, as stated by Matthews and Walkey (1929). On the other hand, many cases of avian paralysis were found in which visceral organs, muscles, the integument, and nervous tissue were simultaneously In some of these cases the diseased condition appeared to have spread involved. by infiltration and in others metastasis had apparently taken place through the The infiltrations were continuous and involved contiguous nerves blood stream. The most common simultaneous infiltration of nervous tissue and organs. and visceral organs occurred in the celiac plexus, ovary, and adjacent structures, with the vagus nerve and thymus glands next. The common invasion of the sciatic nerve and kidney was found in 10 cases from the same flock. It appeared from histological examination of diseased tissues that a common pathological cell was involved in both nerves and visceral organs, as appears in Fig. 4. This finding is in agreement with that of Biely, Palmer, and Lloyd (1933).

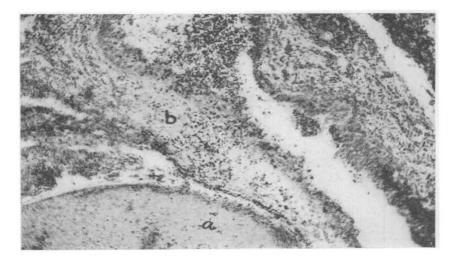


Figure 4. Neurolymphoma of Sciatic Nerve (a) and Lymphocytoma of the Connective Tissues and Surrounding Muscles (b). (Magnified 100 diameters.)

Neurolymphomatosis

In general, it cannot be stated that any particular nerve or plexus is more susceptible to invasion than another because neurolymphomas were found in all of the nerves of the fowl, and two birds were found in which every motor nerve in the body appeared to be affected. These two birds were in a most helpless condition. Paralysis is especially evident when the sciatic nerves, brachial plexus, celiac plexus, vagus, and cptic nerves are involved because important functions of the body are interfered with, and the lesions are more evident at necropsy in large nerves than in small ones. A careful checking up on the location of the gross lesions of neurolymphomatosis in the flocks studied did not reveal any outstanding flock specificity, such as might be attributed to family weakness or inherited predisposition, although such may have existed and our observations were not fine enough to detect them. It was physically impossible to examine all of the birds histologically. Table 1 shows the results of the necropsies of 304 birds from seven different flocks and the tabulation of the names of the nerves affected with neurolymphomas.

		Flocks												
Nerve	A	В	С	D	E	F	G							
Sciatic	41	69	31	8	23	20	13							
Vagus	39	35	30	6	14	27	12							
Brachial	29	28	12	3	20	19	5							
Celiac	12	3	9	4	7	15	10							
Optic	6	4	0	0	0	0	16							
Mesenteric	5	4	2	1	0	8	3							
Crural	1	1	3	1	3	7	1							
Peroneal	3	0	0	0	0	0	0							
Costal	0	0	0	1	0	0	0							
Cervical	0	1	0	0	0	0	0							
Plantar	1	0	0	0	0	0	0							
Laryngeal	2	0	0	1	1	4	0							
Meningeal	1	0	0	1	0	0	0							
Coccygeal	0	0	1	0	1	0	0							
Number of Birds	91	92	32	10	25	30	24							

TABLE 1. LOCATION OF NEUROLYMPHOMAS

This table shows that the sciatic, vagus, brachial, celiac, and optic nerves were most commonly affected. A comparison of Flock A with Flock F shows some important differences. In Flock A the sciatic nerves were most commonly involved, while in Flock F the involvements occurred largely in the vagus and brachial nerves. This may indicate flock specificity, but it cannot be definitely proved for all of these flocks represent different blood lines and the distribution of the neurolymphomas are rather uniform, except in Flock F referred to above, and Flock G, in which the optic nerve was frequently involved.

It was noticed that birds received from flocks on the range often improved after being isolated in individual cages. Histological and hematological examinations of the tissues and blood indicated that these birds were holding their own, temporarily at least. The pathological cell involved in neurolymphomatosis was not found in the blood and tissues outside of active lesions, which indicates that there was some antagonism to the disease. There was no laboratory evidence to indicate that this antagonism was due to immunity. The disease tends to lessen in severity after existing in a flock for two or three years, which demonstrates clinically that either some degree of immunity is developed or the susceptible birds die, leaving the more resistant to reproduce future generations. Three birds, apparently showing active symptoms of paralysis, recovered and at necropsy no lesions of disease were found.

Lymphocytomatosis

A study of uncomplicated lymphocytomatosis revealed that visceral organs, such as the ovary, mesentery, kidney, liver, and spleen were most commonly involved. These organs are closely associated with serous membranes. Next to be affected were organs and surfaces lined with mucous membranes, such as the peritoneum, oviduct, proventriculus, lungs, pleura, intestine, crop, ureter, and cloaca. Muscle tissue, skin, and ductless glands were involved in a few instances, showing that no tissue of the fowl is entirely resistant to lymphocytomatosis.

The lymphocytoma develops the same as the neurolymphoma, except that it is confined almost entirely to tissues other than the nervous type. The invading cell is indistinguishable from the pathological cell occurring in neurolymphomatosis. Parenchymatous organs, mucous membranes, and glandular tissues offer less resistance to invasion than muscle, or the integument.

Studies on the location of lymphocytomas are recorded in Table 2.

				Flocks			
Tissue	А	В	С	D	E	F	G
Ovary	21	19	3	2	6	8	4
Mesentery	12	15	5	1	3	3	2
Kidney	9	14	0	0	2	1	1
Liver	7	8	0	0	1	2	0
Peritoneum	8	8	0	2	4	0	0
Proventriculus	6	1	1	0	2	0	2
Pancreas	3	4	2	2	0	0	0
Muscle	4	4	0	0	1	0	0
Skin	6	4	0	1	3	1	0
Oviduct	4	5	0	0	1	4	2
Spleen	0	3	1	1	2	1	0
Pleura	1	2	0	0	1	0	1
Heart	1	1	0	1	0	0	0
Intestine	2	0	1	2	0	0	0
Thymus	1	0	0	1	0	0	0
Duodenum	0	1	0	0	1	0	0
Crop	1	0	0	0	0	0	0
Adrenal	1	0	0	0	1	0	0
Gizzard	1	0	2	1	3	0	0
Eye	0	1	0	0	1	0	0
Ureter	0	0	1	0	0	0	0
Cloaca	0	0	1	0	0	0	0
Number of Birds	33	55	8	5	15	16	8

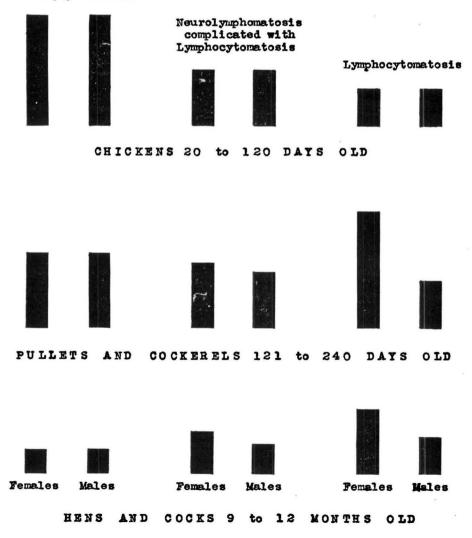
TABLE 2. THE LOCATION OF LYMPHOCYTOMAS

This table shows that the ovary, mesentery, kidney, liver, peritoneum, and glandular portion of the proventriculus are especially susceptible to lymphocytomatosis. The disease is most common at the age of laying the first egg, which indicates that if irritation is not the sole cause it is at least a contributing factor. Since lymphocytomatosis is more common in laying hens than it is in pullets, cockerels, and roosters, it may be that concentrated feeding and forced laying are important factors in the incidence of this disease.

Richardson (1933) has suggested "that catarrhal enteritis with extensive desquamation of the epithelium covering the villi is the primary lesion, with subsequent absorption of toxins from the digestive tract, causing a hyperplasia of the lymphoid tissue normally found in the various organs" may be responsible for the tumors in lympholeukosis. which are similar, if not the same, as in lymphocytomatosis. Lymphocytoma of the ovary is shown in Fig. 5.

Chart 1 represents the incidence of neurolymphomatosis, lymphocytomatosis, and neurolymphomatosis complicated by lymphocytomatosis at various age periods in the chicken, pullet, cockerel, hen, and cock.

Neurolymphomatosis



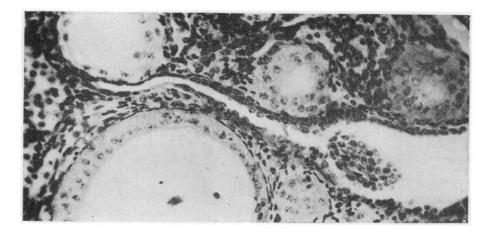


Figure 5. Lymphocytoma of the Ovary. The pathological cells show as black spots in the photograph. (Magnified 420 diameters.)

Neurolymphomatosis Complicated by Lymphocytomatosis

The hepatic, splenic, renal, ovarian, gastric, and small nerves were rarely affected unless neurolymphomatosis was complicated by lymphocytomatosis. Of the large nerves, the celiac plexus was most commonly involved with neurolymphoma as the ovary in the same bird was affected with lymphocytoma. The vagus, sciatic, and brachial nerves were affected by neurolymphomas which had apparently spread either by metastasis or direct infiltration to muscles of the legs, wings, thymus and thyroid glands, and visceral organs. Small lymphocytomas were found embedded in the skin or located in the subcutis immediately under it in 19 of the birds. The tumors were easily felt on palpation as irregular nodular masses. Some of these tumors were located on the breast and exposed to irritation when the birds were on the roosts, but ulceration was not found in any of them.

Representative tumors were studied histologically to see if the nerves supplying the skin were involved also, and they apparently were for all tissues within the infiltrated areas had been converted into tumor cells.

Neurolymphomatosis was complicated with lymphocytomatosis in Flocks A, B, C, and D and the results appear in Table 3. No complications of neurolymphomatosis and lymphocytomatosis occurred in Flocks E, F, and G and the results of the studies of these flocks appear in Tables 1 and 2.

Table 3 shows that in Flock A neurolymphomas were confined largely to the celiac plexus and ovarian nerve, and the lymphocytomas to the ovary. In Flock B the neurolymphomas were found mostly in the vagus nerves while the greatest number of lymphocytomas appeared in the thyroid glands, the thymus glands, liver, spleen, and skin. In Flock C the neurolymphomas occurred mostly in the celiac plexus, and the lymphocytomas were few and not confined to any particular group o organs or tissues. In Flock D the greatest numbers of neurolymphomas were equally divided between the celiac, sciatic, and brachial plexus, and the largest numbers of lymphocytomas were about equally divided between the ovary and muscles of the legs and wings.

NEUROLYMPHOMATOSIS IN CHICKENS

		Floc	ks	
Tissue	A	В	С	D
Ovary	19	9	2	9
Celiac plexus.	18	4	5	10
Vagus nerve	10	16	1	7
Sciatic nerve	7	8	1	10
Ovarian nerve	15	6	4	10
Brachial plexus.	6	5	0	10
Mesenteric nerve	5	2	0	5
Mesentery	1	0	0	0
Crural nerve.	4	0	0	0
Kidney	3	3	1	2
Renal nerve.	1	1	1	2
Muscle	1	3	1	16
Thyroid	2	15	0	0
Thymus	0	12	0	0
Skin	1	15	0	3
Liver.	1	14	1	1
Hepatic nerve	0	5	1	1
Spleen	0	15	1	0
Splenic nerve.	0	7	1	0
Optic nerve	1	0	0	1
Lung	0	1	0	0
Pulmonary nerve	0	1	0	0
Peritoneum	0	1	0	Ő
Peroneal nerve	1	0	Ō	Ő
Testis	1	õ	õ	Ő
Gizzard	Ô	Ő	Ő	1
Proventriculus	2	Ő	Ő	1
Gastric plexus.	1	0	Ő	1
Pancreas	1	0	0	0
Cervical nerve	0	ŏ	0	2
Intestine	0	1	0	0
Number of Birds	25	17	5	17

TABLE 3. THE LOCATIONS OF NEUROLYMPHOMAS AND LYMPHOCYTOMAS IN FOUR FLOCKS

In Flock A direct infiltration appeared to have occurred between the neurolymphomas of the celiac plexus and ovarian nerves, and the lymphocytomas of the ovary. The other neurolymphomas and lymphocytomas were apparently of metastatic inception. In Flock B direct infiltrations had apparently occurred between the neurolymphomas of the vagus nerves and the lymphocytomas of the thyroid and thymus glands. The neoplasms of the liver, spleen, skin, and other tissues were apparently of metastatic origin, as perivascular infiltration was demonstrated histologically in all of them. In Flock C the neurolymphomas

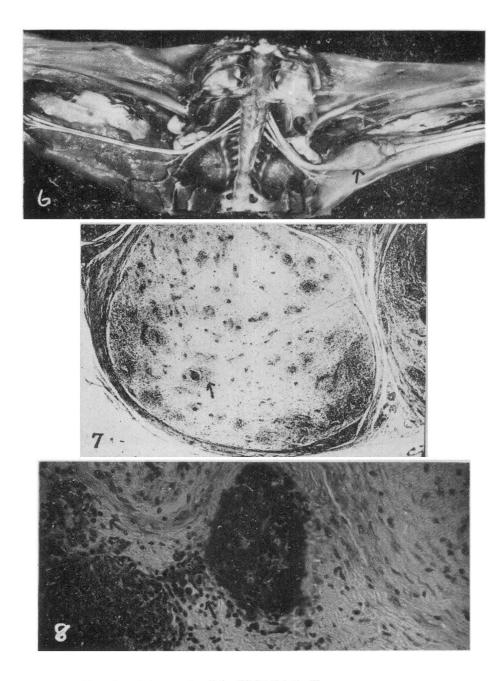


Figure 6. Neurolymphotomatosis of the Right Sciatic Nerve. Figure 7. Perivascular Infiltration in Sciatic Nerve. (Magnified 200 diameters.) Figure 8. Perivascular Infiltration in Sciatic Nerve. (Magnified 420 diameters.)

of the celiac plexus and ovarian nerve appeared to be primary, while the other neurolymphomas and lymphocytomas were undoubtedly established as a result of metastasis. All of the neurolymphomas in the celiac, sciatic, and brachial nerves of Flock D appeared to be of primary origin and led to the formation of lymphocytomas in the ovary and muscles of the legs and wings by direct infiltration. The malignant growths in other tissues appeared to be of perivascular inception. Figs. 7 and 8 show perivascular infiltration of the sciatic nerves.

In studying these cases it was not always possible to determine whether the nerves or the visceral organs were affected first, although it was demonstrated that metastasis as well as direct infiltration may take place through the nervous system, for the pathological cells appeared to be carried in the capillary spaces. This explains why the connecting link between neurolymphoma and lymphocytoma appeared to be the branches of the larger nerves leading to the visceral organs, and other tissues in which the neoplasms were found. While distant metastasis undoubtedly occurred in some cases, the evidence was not as convincing as in those instances in which direct infiltration or metastasis was traced. Apparently the pathological cell responsible for these malignant growths occasionally develops such invasiveness that any or all tissues are affected. If this deduction is correct, neurolymphomatosis and lymphocytomatosis are closely related and are differentiated on the basis of tissue selection only. Furthermore, this study indicates that neurolymphomatosis is a disease of young birds while lymphocytomatosis is more commonly found in adults ten months or more of age. However, in spite of the conclusiveness of these histological studies, the exact relation between neurolymphomatosis and lymphocytomatosis awaits etiological confirmation, if the theory of an inciting virus is accepted.

The Mortality and Morbidity of Flock C

Next an attempt was made to determine the mortality and morbidity occurring in Flock C from all causes. The results of this study are shown in Table 4.

Discases	Cases	Mortality	Number Recovering	Number not Improved	Average Age When Symptoms Appeared (Days)	Average Age When Death Occurred (Days)	Average Number of Days Sick
Neurolymphomatosis with Gross Lesions		30	1	2	79	132	53
Avian Paralysis without Gross Lesions		6	0	1	130	168	38
Lymphocytomatosis.		11	0	0	156	165	9
Neurolymphomatosis and Lymphocytomatosis	5	5	0	0	145	168	23
Myelcleukosis.		4	0	0	143	146	3
Erythroleukosis.		2	0	0	92	101	9
Myeleleukosis and Lymphocytomatosis	2	2	0	0	106	120	14
Chronic Coccidiosis.	18	2	2	14	145	164	Indefinite
Slipped Tendon		0	0	4	44	49	Indefinite
Ascariasis	2	0	0	2	113	163	Indefinite
Visceral Gout	1	1	0	0	167	236	Indefinite
Cannibalism	16	6	10	0	137	139	2

TABLE 4. A STUDY OF MORTALITY AND MORBIDITY IN FLOCK C

Table 4 shows that avian paralysis with gross lesions, or neurolymphomatosis, led all other diseases in mortality and morbidity. As has been stated before, three birds recovered from neurolymphomatosis, while birds having lymphocytomatosis and leukemia did not recover. In the last two diseases the function of blood-forming organs was seriously interfered with and the birds did not live as long as when affected with neurolymphomatosis. Birds suffering from chronic coccidiosis, slipped tendon, ascariasis, and visceral gout lived indefinitely in a run-down condition. According to the table, the victims of cannibalism lived two days. At the end of this period they were either dead or on the road to recovery. Birds showing complications of neurolymphcmatosis and lymphccytomatosis lived 24 days, which is 15 days longer than those showing lesions of uncomplicated lymphocytomatosis.

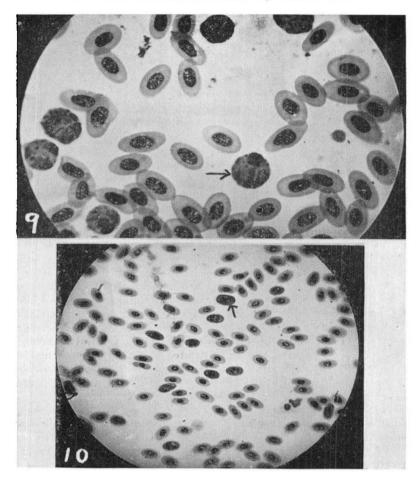
This study indicates that all of these conditions are serious and should be avoided as much as possible.

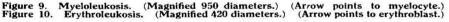
The Differentiation of Neurolymphomatosis from Lymphocytomatosis and Leukemia

Birds having lymphocytomatosis and leukemia are often unable to stand, and these diseases are mistaken for paralysis or neurolymphomatosis by the laity; but a trained diagnostician should observe that the comb, wattles, face, and mucous membranes in cases of the former are pale, while these parts in the latter are usually of good color. This observation is superficial and should not be depended upon entirely for diagnosis, yet it is of considerable help in the differentiation of neurolymphomatosis from lymphocytomatosis and leukemia in the field. The leukemias are further differentiated in that no immature red cells or immature eosinophilic white cells are found in the circulating blood in either neurolymphomatosis or lymphocytomatosis, and erythroblastic and myeloblastic hyperplasia are found only in the respective leukotic conditions.

The examination of the blood is of the greatest assistance in the differential diagnosis of poultry diseases. Neurolymphomatosis begins with a leukocytosis and ends with a lymphocytosis. Differential counts indicate that the first increase is in eosinophile leukocytes, while the last increase is in small mononuclear lymphocytes. Sometimes the leukocytosis and the lymphocytosis are found overlapping, but generally there is a period between in which the birds apparently hold their own for awhile and the blood count is the same as in normal fowls. In other words, red blood cells, polymorphonuclear leukocytes, neutrophiles, mast cells, and monocytes are the same as in normal birds. In uncomplicated lymphocytomatosis there is a decrease in hemoglobin, the clotting time of the blood is prolonged, and the red blood cells are diminished. Leukocytosis and lymphocytosis occur the same as in neurolymphomatosis. The remaining elements of the blood are seldom disturbed in this disease.

In myeloleukosis the blood is invaded by myelocytes in various stages of development, depending upon the severity of the disease. The pathological cell found in the blood and lesions of the tissues in this disease is the eosinophilic myelocyte, Fig. 9. Neutrophilic and basophilic varieties of this myelocyte may be present in small numbers. Fresh myelocytes usually gave positive oxydase reactions when tested according to the method of Graham (1918) in the early stages of the disease. The liver and spleen were enlarged in advanced cases, and histological sections stained by Mallory's eosin and methylene blue showed more or less myeloblastic hyperplasia. In some advanced cases the bone marrow showed-evidence of calcification and the absence of fat. Microscopical examina-





tion of the bone marrow showed a diminution, if not an entire absence, of mature leukocytes. The red blood cells were always reduced, the hemoglobin was low, and the clotting of the blood retarded.

Myeloleukosis was differentiated from lympholeukosis on the basis of the pathological cells appearing in the tissues of the birds affected with the respective diseases. In some cases the oxydase test was relied upon to identify the myeloblastic cells of the bone marrow from the cells of the lymphocytic series. A large number of cells with oxydase granules appeared to be characteristic of myeloleukosis just as the reverse seemed to be true of lympholeukosis. In advanced cases of acute myeloblastic leukosis many of the cells may be too immature to react to the oxydase test. This difficulty did not lead to diagnostic error in this study because the blood of the birds was examined at least once as soon as symptoms appeared and at other times later in the course of the disease. But some difficulty was experienced in differentiating generalized infiltration of lymphocytomas from lympholeukosis. Usually lymphocytomas appeared on gross and histological examination as irregular foci of neoplastic growths embedded in the visceral organs and other tissues, while lympholeukosis appeared to be limited to the liver, spleen, and sometimes the kidney, and always involved the whole of the affected organ.

Blood smears from birds suffering from erythroleukosis showed immature forms of erythrocytes, as shown in Fig. 10. Histological examination of tissues from the liver, spleen, and kidneys revealed intravascular accumulations of these immature cells in the capillary spaces, so that the organs were in a state of distention. Sometimes the distention was so great that hemorrhage occurred. The hemoglobin was reduced in erythroleukosis and the clotting of the blood delayed.

The blood of a representative number of birds for each disease included in this study was examined hematologically. It was impossible to examine all on the experiment because the methods were time consuming, and it was considered better to make a thorough study of a selected number rather than to attempt all of them hurriedly.

The Hematology of Birds from Flock C

The results of the examination of the blood of representative birds from Flock C are contained in Table 5.

The white blood counts recorded for Group 1, were higher than normal in birds Nos. 2, 11, 16, 18, and 19, and lower than normal in No. 1. Differential counts showed that the increase in birds Nos. 11, 16, and 18 was in eosinophilic granular leukocytes, while in bird No. 19 the eosinophilic leukocytes with rods were above normal. Also bird No. 18 showed a high lymphocyte count. The blood of Nos. 3 and 17 was normal, or the same as for healthy birds.

In Group 2, the white cell count was above normal in birds Nos. 13 and 20. Differential counts indicated that the increase in white cells in No. 13 was in polymorphonuclear leukocytes, while that of No. 20 was in small lymphocytes The blood of Nos. 10 and 14 was normal.

The average white blood cell count was higher in the birds severely affected with paralysis (Group 3) than in Groups 1 and 2. The differential count indicated that this increase was due in general to lymphocytosis, as all of the birds except No. 6 showed relatively high lymphocyte counts. It was found at necropsy that No. 6 had a heavy infestation of roundworms, which may have been responsible for leukocytosis and the probable masking of the lymphocyte count.

The birds in Group 4 were examined hematologically when first brought to the laboratory from the range and again just before death. The symptoms of paralysis had increased in every case. A study of this table shows in most cases a leukocytosis accompanied by a progressive lymphocytosis. The hemoglobin and blood clotting time were not determined in bird No. 33.

The averages of the first and last hematological examinations show some interesting comparisons as follows: The red blood cells are less in the last average count than in the first; the white blood cells are more in the last average count than in the first; the comparison of the first average leukocyte count and the first average lymphocyte count with the corresponding last counts indicates that leukocytosis is giving way to lymphocytosis.

Group 5 contains the results of the hematological studies of birds selected from Flock C that were down and unable to arise, but showed no lesions of paralysis at necropsy. Bird No. 52 gave a low white blood cell count when first examined, but three days later the count was nearly normal. Bird No. 53 showed a marked leukocytosis in which eosinophilic leukocytes with granules predominated. Bird No. 54 had a high white blood cell count when submitted to the laboratory, but it dropped to nearly normal in five days. Birds Nos. 55, 56, 57, and 58 gave normal blood counts. Gross and histological examination of the nervous systems did not reveal the cause of paralysis in these birds. Possibly they represent cases of leukosis in which hyperplasia is absent, as has been described by Furth (1933).

Group 6 shows the blood counts of birds from Flock C, which showed symptoms of paralysis and anemia during life and at necropsy were found to be affected with neurolymphomatosis and lymphocytomatosis. The eosinophilic granular leucocyte count was high in all of the fowls except No. 62. The significance of this finding, if of any importance, is not understood at present. The hemoglobin was low in all of the birds. The blood clotted rather slowly in fowls Nos. 62, 64, 65, 66, and 67, and the celiac plexus was involved in all of the birds in this group. The ovary was tumorous in three cases, the testicles in one, and the kidneys, liver, and mesenteries in the others. The connection between neurolymphomatosis and lymphocytomatosis of the visceral organs was close in every case. Evidences of metastatic infiltration were found. It may be that when the arteries are filled with lymphoblasts these cells lodge in the capillaries of tissues other than the nervous type, forming foci of infection which lead to the development of the so-called lymphocytoma, which is apparently of the same origin as the neurolymphoma.

The five birds listed in Group 7 had lymphocytomas of the visceral organs, and the nervous system appeared to be normal on gross and histological examination. All of the birds appeared anemic, and had high white blood counts and low hemoglobin determinations. Differential counts showed that all of them had leukocytosis. The blood of Nos. 88 and 89 was counted twice. Bird No. 88 showed no marked changes after 45 days, while the blood of Nos. 89 indicated a shift from a leukocytosis to a lymphocytosis in 10 days.

Four cases of myeloleukosis were included in the hematological studies and the results are given in Group 8. The red blood cells were low and the white blood cells were higher than normal in all four of these birds. The hemoglobin was very low, the clotting time prolonged, and the birds bled profusely as the blood was drawn from the wing vein for the tests. Differential staining brought out many eosinophilic myelocytes in the blood stream. Also the eosinophilic leukocytes with granules were comparatively increased in bird No. 98. It was observed in this study that the granules in the eosinophilic leukocytes resembled those in the granular myelocytes, but myelocytosis was not always accompanied by a high granular eosinophilic leukocyte count.

Three birds in Flock C were found to have erythroleukosis. These birds were anemic, as shown by the blood counts and hemoglobin determinations in Group 9. The clotting of the blood was much delayed in all three cases. The most characteristic feature of this disease was the presence of erythroblasts and polychrome erythrocytes in the circulating blood. If the blood had not been examined in these cases the diagnosis would have been missed. The liver and spleen were enlarged in all three cases, and histological sections showed that the capillaries of these organs were distended with immature erythrocytes in various stages of development. Erythrocytes were rarely found in the bone marrow of the femur, tibia, radius, and ulna of these birds. The most common cell appeared to be a nucleated, basophilic type, undifferentiated from the large monocyte by Romanosky stains. This cell may have been the precursor of the erythroblasts found in the blood stream.

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TABLE 5.	Hemat	TOLOGY C	of Birds	FROM FLOCK C	
		1		Eosinophilic Leukocytes	

								1			ophilic ocytes]		s S
Bird No.	Ake, Days	Days Under Observation	Red Blood Cells	White Blood Cells	Hemoglobin	Blood Clotting Time	F.rythroblasts	Polvchrome Erythrocytes	Eosinonhilic Myelocytes	With Rods	With Granules	Polymorphonuclear Leukocytes	Neutrophilic and Basonhilic Myelocytes	Neutrophilic Leukocytes and Mast Cells	Lymphoblasts	Small Lymphocytes	Large Lymphocytes and Mononuclears
				Group 1.	Birds S	Showing 1	Mild Syn	nptoms o	f Faralys	sis for L	ess Thar	n One W	leek				
1 2 3 11 16 17 18 19	$30 \\ 93 \\ 120 \\ 102 \\ 83 \\ 122 \\ 125 \\ 196$	$ \begin{array}{c} 1 \\ 5 \\ 5 \\ 7 \\ 2 \\ 2 \\ 1 \end{array} $	$\begin{array}{c} 2,830,000\\ 3,410,000\\ 2,846,000\\ 2,732,000\\ 2,872,600\\ 3,030,000\\ 2,798,000\\ 1,770,000\\ \end{array}$	$\begin{array}{c} 22,000\\ 33,800\\ 25,000\\ 48,000\\ 35,900\\ 26,500\\ 39,000\\ 41,600\end{array}$	$\begin{array}{c} 72.4 \\ 74.8 \\ 74.4 \\ 65.0 \\ 74.0 \\ 88.3 \\ 69.9 \\ 71.5 \end{array}$	1' 30'' 2' 00'' 2' 45'' 4' 30'' 2' 30'' 1' 30'' 2' 18'' 2' 40'' 2' 40'' 2' 40'' 2' 40'' 2' 40'' 2' 40'' 2' 40'' 2' 40'' 2' 40'' 2' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 3'' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 4' 30'' 2' 45'' 2' 45'' 1' 30'' 2' 45'' 3'' 4' 3'' 3'' 3'' 2' 45'' 3'' 3'' 3'' 3'' 2' 45'' 3'' 3'' 3'' 3'' 3'' 3'' 3'' 3'' 3'' 3''	0 0 0 0 0 0 0 0 0	0 0 0 0 0 0 0 0 0	0 0 0 0 0 0 0 0	$\begin{array}{c} 8.0 \\ 12.2 \\ 16.3 \\ 0 \\ 5.0 \\ 14.8 \\ 4.0 \\ 46.9 \end{array}$	$\begin{array}{c} 26.1 \\ 15.3 \\ 18.7 \\ 45.1 \\ 30.4 \\ 18.0 \\ 28.7 \\ 5.2 \end{array}$	$\begin{array}{r} 4.9\\ 3.0\\ 6.5\\ 10.0\\ 6.3\\ 5.2\\ 5.3\\ 10.4\end{array}$	0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	$\begin{array}{cccccccccccccccccccccccccccccccccccc$	0 0 0 0 0 0 0	$\begin{array}{c} 36.7\\ 29.7\\ 39.7\\ 30.2\\ 38.7\\ 40.0\\ 49.0\\ 1.8\\ \end{array}$	$\begin{array}{c} 20.4\\ 32.7\\ 13.3\\ 13.1\\ 17.2\\ 18.1\\ 5.3\\ 35.7 \end{array}$
Average	109	4	2,786,075	33,975	73.8	2' 28"	0	0	0	13.4	23.4	6.5	0 0	1.7 2.3	0	33.2	19.5
			(•			or Mode	erate Syn	nptoms o	of Paraly		lore Th	an One Week				
$ \begin{array}{c} 10 \\ 13 \\ 14 \\ 20 \end{array} $	$102 \\ 112 \\ 122 \\ 244 $	$ \begin{array}{r} 12\\10\\8\\15\\\hline \end{array} $	$\begin{array}{r} 4,150,000\\ 3,210,000\\ 3.450,000\\ 2,900,000\\ \end{array}$	$\begin{array}{r} 23,600\\ 35,400\\ 24,400\\ 49,000\end{array}$	$\begin{array}{r} 78.6 \\ 74.3 \\ 76.1 \\ 77.6 \end{array}$	2' 35" 2' 15" 3' 20" 2' 15"	0 0 0	0 0 0	0 0 0 0	$ \begin{array}{r} 14.4 \\ 2.7 \\ 10.0 \\ 4.8 \\ \end{array} $	$26.6 \\ 0 \\ 26.5 \\ 14.9 $	$\begin{array}{r} 4.4 \\ 51.3 \\ 12.6 \\ 1.0 \end{array}$	0 0 0 0 0 0 0 0	$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	0 0 0	$\begin{array}{r} 30.2\\ 35.2\\ 42.5\\ 65.7\end{array}$	$22.2 \\ 8.1 \\ 8.4 \\ 10.7$
Average	145	11	3,427,500	33,100	76.7	2' 36"	0	0	0	8.0	17.0	17.3	0 0	1.0 1.0	0	43.4	12.4
					Group		s Showin	ng Sever									
4 5 6 7 8 9 12 15 21	$141 \\ 118 \\ 124 \\ 115 \\ 84 \\ 123 \\ 123 \\ 127 \\ 257 $	$ \begin{array}{c c} 5 \\ 1 \\ 7 \\ 1 \\ 2 \\ 17 \\ 39 \\ 13 \\ 63 \\ \hline \end{array} $	$\begin{array}{c} 2,820,000\\ 2,320,000\\ 3,260,000\\ 2,600,000\\ 2,600,000\\ 2,876,000\\ 200,000\\ 3,475,000\\ 2,490,000\\ \end{array}$	$\begin{array}{c} 32,000\\ 40,200\\ 35,000\\ 46,000\\ 51,000\\ 54,840\\ 42,400\\ 40,000\\ 46,000\end{array}$	$\begin{array}{c} 72.6 \\ 72.0 \\ 81.3 \\ 80.0 \\ 72.0 \\ 73.8 \\ 74.3 \\ 78.0 \\ 72.0 \end{array}$	2' 30" 2' 40" 1' 33" 2' 3" 3' 40" 3' 10" 1' 14" 3' 00" 3' 10"			0 0 0 0 0 0 0 0	7.43.720.39.72.811.28.617.36.4	$\begin{array}{c} 31.3\\ 24.9\\ 9.4\\ 14.4\\ 19.2\\ 12.0\\ 1.5\\ 19.4\\ 21.8 \end{array}$	$ \begin{array}{c} 10.4 \\ 1.5 \\ 30.2 \\ 4.3 \\ 0 \\ 4.4 \\ 18.6 \\ 11.1 \\ 3.8 \\ \end{array} $	0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	0 0 P* P* 0 0 0	$\begin{array}{c} 44.1 \\ 54.3 \\ 30.3 \\ 57.6 \\ 52.6 \\ 51.5 \\ 54.0 \\ 48.4 \\ 54.3 \end{array}$	$\begin{array}{c} 3.1 \\ 12.8 \\ 6.0 \\ 10.4 \\ 25.2 \\ 17.4 \\ 12.8 \\ 2.6 \\ 12.4 \end{array}$
Average	136	16	2,604,555	43,049	75.1	2' 33"	0	0	0	9.7	17.1	9.4	0 0	1.3 1.6	P*	49.7	11.4

			-						-							
$22.\ldots.$	$\frac{1}{29}$	3,200,000 3,220,000	$25,000 \\ 28,000$	80.0 80.5	1' 30" 1' 30"	0	0	0 0	$\begin{array}{ccc} 8.0 & 18.0 \\ 11.0 & 28.9 \end{array}$	$\begin{bmatrix} 2.0\\ 10.3 \end{bmatrix}$	0 0	$\begin{bmatrix} 0\\0 \end{bmatrix}$	$\begin{array}{ccc} 0 & 2.0 \\ 1.0 & 1.6 \end{array}$	0 0	$\left \begin{array}{c} 42.0 \\ 44.1 \\ 3.1 \end{array}\right $	
$23,\ldots,\ldots, 5$ 88 (112)	$\frac{1}{24}$	$3,620.000 \\ 3,005,000$	$23,000 \\ 32,500$	$\frac{81.4}{79.5}$	2' 15" 1' 30"	0	0. 0.	0 0	${11.5 \ 43.0 \ 5.0 \ 23.0}$	$\begin{array}{c}11.5\\10.0\end{array}$	0 0	0 0	$egin{array}{cccc} 1.0 & 3.1 \ 0 & 2.0 \end{array}$	0 0	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	
$\begin{array}{c} 24\ldots\ldots\ldots \\ 91\\ 98\end{array}$	1 7	3,750,000 3,225,000	$91,\!800 \\ 84,\!600$	$75.0 \\ 71.5$	2' 15'' 2' 25''	0 0	0	0 0	$\begin{array}{ccc} 1.0 & 40.8 \\ 0 & 25.0 \end{array}$		0 0	0 0	$egin{array}{ccc} 0 & 1.3 \ 0 & 1.3 \ \end{array}$	0 0	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	
$25,\ldots,\ldots,$ $\begin{cases} 83\\ 132 \end{cases}$	$\frac{1}{49}$	3,400,000 3,215,000	$25,000 \\ 31,000$	$78.2 \\ 76.8$	2' 15" 2' 30"	0 0	0	0 0	$\begin{array}{rrr} 9.1 & 33.1 \\ 10.0 & 16.0 \end{array}$	$5.4 \\ 5.0$	0 0	0 0	${ \begin{array}{ccc} 3.1 & 1.3 \\ 0 & 2.5 \end{array} }$	0 0	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	
$26\ldots\ldots\ldots(115+132)$	1 16	3,200,000 3,000,000	28,000 30,000	$75.0 \\ 73.9$	1' 32'' 24''	0	0	0 0	$\begin{array}{cccc} 4 & 3 & 29 & 6 \\ 4 & 3 & 30 & 0 \end{array}$	$\frac{2.5}{3.2}$	0 0	0 0	$egin{array}{ccc} 0 & 1.9 \ 0 & 2.0 \end{array}$	0 0	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	
$\begin{array}{c} 27 \dots & \begin{pmatrix} 80 \\ 120 \end{pmatrix}$	1 -4	3,400,000 2,940,000	$22,000 \\ 38,000$	78.3 76.0	2' 10" 2' 30"	0.0	0	0 0	$\begin{array}{ccc} 21.1 & 38.4 \\ 9.9 & 19.5 \end{array}$	$5.7 \\ 5.4$	0 0	0 0	$\begin{array}{cccccccccccccccccccccccccccccccccccc$	\mathbf{h}_{0}^{*}	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	
$\begin{array}{c} 28 \ldots \ldots \ldots \\ 105 \\ 125 \end{array}$	$\frac{1}{20}$	3,100,000 2,900,000	$24,000 \\ 27,000$	$\begin{array}{c} 80.5 \\ 74.0 \end{array}$	$\frac{1'}{2'} \frac{36''}{51''}$	0 0	0	0 0	$\begin{array}{ccc} 0 & 32.0 \\ 2.4 & 25.0 \end{array}$	$\frac{1.8}{1.5}$	0 0	0 0	$\begin{smallmatrix}1.2&2.2\\.8&2.0\end{smallmatrix}$	0 P*	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	
$\begin{array}{c} 29 \ldots \ldots \ldots \\ 194 \end{array}$	1 49	2,280,000 2,150,000	$35,000 \\ 39,500$	$\frac{81.6}{77.9}$	2' 15'' 3' 18''	0 0	0 0	0 0	$\begin{array}{cccc} 10.0 & 7.0 \\ 2.4 & 11.8 \end{array}$	$\frac{10.5}{10.2}$	0 0	0 0	$\begin{array}{cccccccccccccccccccccccccccccccccccc$	0 P*	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	
$30,\ldots,\ldots, {igl(145 \ 165 \ 1$	1 20	2,620,000 2,340,000	$33,000 \\ 43,000$	80.0 71.7	2' 00" 2' 52"	0	0	0 0	$\begin{array}{ccc} 1.0 & 17.0 \\ 2.0 & 11.5 \end{array}$	$ \begin{array}{c} 31.5 \\ 21.5 \end{array} $	0 0	0 0	$\begin{array}{ccc} 0 & 2.5 \\ 0 & 2.5 \end{array}$	0 P*	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	
$\begin{array}{c} .31 \ldots \ldots . \\ 132 \\ 174 \end{array}$	1 12	2,560,000 2,400,000	$22,000 \\ 42,000$	$\frac{80.0}{73.0}$	1' 30" 2' 00"	0 0	0	0 0	$egin{array}{cccc} 6.5 & 15.4 \\ 4.2 & 10.7 \end{array}$	$\begin{array}{c} 10.0\\ -2.0 \end{array}$	0 0	0 0	$egin{array}{ccc} 0&2.9\\2.4&2.9\end{array}$	Р* Р*	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	
$32 \dots \dots \dots \begin{pmatrix} 82\\112 \end{pmatrix}$	$\frac{1}{30}$	3,100,000 2,900,000	$34,000 \\ 47,000$	$78.8 \\ 73.6$	3' 10" 2' 10"	0 0	0 0	0 0	${\begin{array}{*{20}c} 6.0 & 7.2 \\ 3.1 & 10.2 \end{array}}$	$\begin{array}{c} 28.4 \\ 5.8 \end{array}$	0 0	0 0	$\begin{array}{cccccccccccccccccccccccccccccccccccc$	0 P*	$\begin{array}{c ccc} 43.6 & 8.6 \\ 55.8 & 16.7 \end{array}$	
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	$\frac{1}{25}$	4,620,000 1,860,000	$26,000 \\ 21,000$			0 0	0 1.3	0 0	$\begin{array}{ccc} 4.7 & 20.6 \\ 1.0 & 14.7 \end{array}$	$\frac{1.1}{4.4}$	0 0	0 0	$ \begin{array}{ccc} 1.8 & 0 \\ 1.8 & 2.1 \end{array} $	0 0	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	
Averages 1st Count 113 2d Count 147	1 24	3,237,500 2,762,750	32,400 38,633	$79.0 \\ 75.3$	2' 3'' 2'' 22''	0	0	0	$\begin{array}{r} 6.9 & 25.2 \\ 4.6 & 18.8 \end{array}$	9.7 7.3	0	0	$\begin{array}{cccccccccccccccccccccccccccccccccccc$	р* Р*	$\begin{array}{ c c c c c c c c c c c c c c c c c c c$	

Group 4. Birds Examined When First Observed and Again Just Before Death

*P=Present, failed to show oxydase granules.

			s T	Cells				s		Eosinophilic Leukocytes	⁻ orphonuclear ikccytes				mphocytes	Lymphocytes Mononuclears
	s	ler ion	d Cells	Blood (nic	Blood Clotting Time	asts	Polvchrome Erythrocytes	Eosinorhilic Myelocytes	Rods Granules	honu ytes	ytes	Neutrophilic Leukocy tes and Mast Cells	lasts	oydu	npho
No.	Days	. Une	Blood	e Bl	oglol	H Cl	throblasts	thro	lor hi		rorp	roph ind chilic	roph Ikoc Ind Cell	hob	Ê	Ly. Mc
Bird	Agc,	Days Under Observation	Red	White	Hemoglobin	Tin	Erytl	Er	M ₃	With Rods With Gran	Pclym	Neutrophilic and Basophilic Myelucytes	Veuti Let Mast	Lymphoblasts	Small	l.arge and
					Group 5	Birds	Paralyze			o Lesions of Dis						
52		$\begin{vmatrix} 1\\ 3 \end{vmatrix}$	2,660,000 2,570,000	$15,000 \\ 19,642$	$\begin{array}{c} 70.4 \\ 68.4 \end{array}$	2' 00'' 2' 00''	0	00	0	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	$\begin{bmatrix} 48.0 \\ 30.0 \end{bmatrix}$	= 0 = 0	$\begin{vmatrix} 0 & 2.2 \\ 1.2 & 1.5 \end{vmatrix}$	0	$\begin{array}{c} 4.5\\ 25.1 \end{array}$	$\begin{array}{c}9.4\\12.7\end{array}$
53	. 157	1	2,890,000	93,000	64.6	1' 30"	0	0	0	24.0 50.0	0	- 0	1.0 3.7	0	18.5	3.7
54	$\{ \begin{array}{c} 132\\ 157 \end{array} \}$	1 5	3,060,000 2,680,000	$50,400 \\ 36,400$	$\begin{array}{c}40.5\\67.2\end{array}$	4' 30'' 2' 15''	0 0	0	0 0	$ \begin{array}{ccc} 0 & 40.0 \\ 4.2 & 29.8 \end{array} $	$\frac{8.0}{5.0}$	$\frac{-}{-}$ $\frac{1.0}{0}$	$\begin{array}{cccc} 2.0 & 2.0 \\ 0 & 2.6 \end{array}$	0 0	$\begin{smallmatrix} 22.0\\ 46.2 \end{smallmatrix}$	$25.0 \\ 21.2$
55	. 77	1	3,031,000	26,490	83.2	1' 30"	0	0	0	10.0 23.0	5.0	- 0	5.0 2.0	0	47.0	8.0
56	119	1	2,436,000	24,000	77.4	2' 33"	0	0	0	8.1 36.8	6.7	— 0	0 1.4	0	24.7	22.3
57	$-\begin{cases} 96\\ 120 \end{cases}$	$\begin{array}{c}1\\24\end{array}$	2,870,000 2,530,000	$20,400 \\ 22,500$	80.5 77.9	3' 20" 3' 20"	0 0	0	00	$\begin{array}{cccccccccccccccccccccccccccccccccccc$	$egin{array}{c} 1.1 \ 5.2 \end{array}$	$\begin{array}{c} - & 0 \\ - & 0 \end{array}$	$ \begin{array}{ccc} 0 & 0 \\ 0 & 2.8 \end{array} $	0	$\begin{array}{c} 39.7\\42.0\end{array}$	25.4 20.0
58	. 171	1	2,880,000	14,000			0	0	0	46.2 - 30.6	2.8	0	0 0	0	10.8	9.6
Average	. 140	5	2,760,700	32,183	70.6	2' 33"	0	0	0	11.0 31.3	11.2	— 0.1	0.9 1.8	0	28.0	15.7
61) <u>(</u>	2,360,000	Group 20,350	56.2	s Showin 1' 20"	g Lesion	s of Neu	rolymphe 0	matosis and Ly 10.0 20.5					1 44 6	
01			1,960,000	12,800	44.2	1' 20"	0 0	ŏ	0	$\begin{array}{cccc} 10.0 & 20.5 \\ 0.1 & 46.1 \end{array}$	$5.2 \\ -6.6$	- 0 - 0	$ \begin{array}{ccc} 0 & 1.8 \\ 2.0 & 6.0 \end{array} $	0	$\begin{smallmatrix} 44.0 \\ 23.8 \end{smallmatrix}$	$\begin{array}{c}18.5\\15.4\end{array}$
62	. 137		2,480,000	15,600	52.9	3' 44"	0	0	0	0 13.9	46.5	— 0	0 0.2	0	34.8	4.6
63			2,220,000	14,200	44.2	1' 20"	0	0	0	7.6 30.0	14.5	0	3.0 0.6	0	34.3	10.0
64	. 177))	1,740,000	16,000	40.6	4' 31"	0	0	0	$6.2 \ 34.7$	7.6	- 0	2.0 5.2	0	34.6	9.7
65	. 190		2,105,000	50,000	63.6	6' 00"	0	0	0	0.2 48.6	2.8	<u> </u>	0 0	0	34.3	14.1
66	. 96		3,600,000	36,000	64.9	5' 42"	0	0	0	6.9 40.0	21.1	— 0	1.1 3.3	0	25.0	2.6
67	. 114		1,392,000	48,700	62.0	4' 28"	0	0	0	9.0 32.0	14.8	0	0 2.1	0	33.7	8.4
Average	. 137	(2,232,125	26,706	53.6	3' 33″	0	0	0	5.0 33.2	14.9	- 0	1.0 2.4	0	33.1	10.4

TABLE 5. HEMATOLOGY OF BIRDS FROM FLOCK C-Continued

Group 7.	Birds Showing	Lesions of	Lymphoc	ytomatosis
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88 {108 153	$\frac{1}{45}$	$\begin{bmatrix} 2,032,000 \\ 1,460,000 \end{bmatrix}$	$27,000 \\ 28,000$	$\begin{array}{c} 71.0 \\ 63.0 \end{array}$	$\begin{bmatrix} 2' & 6'' \\ 2' & 10'' \end{bmatrix}$	0 0	0	0	0 0	$\frac{38.6}{38.6}$	$\begin{vmatrix} 18.6 \\ 18.5 \end{vmatrix}$		0 0	$\begin{array}{ccc} & 0\\ & 0.6 \end{array}$	-	$\begin{bmatrix} 24.3\\ 24.3 \end{bmatrix}$	18.5 18.0 2
$89.\ldots$ $\begin{cases} 157\\ 168 \end{cases}$	1 11	1,861,000 1,520,000	$38,160 \\ 44,000$	$\frac{38.7}{25.1}$	$\frac{2'}{3'} \frac{23''}{26''}$	0 0	0	0 0	$\begin{array}{c} 10.0 \\ 5.4 \end{array}$	$\begin{array}{c} 39.9\\ 18.2 \end{array}$	9.2 4.8		0 0	-2.6 -3.9		$egin{array}{c} 21.3 \\ 52.4 \end{array}$	17.0 (15.3)
90	11	1,550,000	32,000	67.9	1' 30"	0	0	0	0	78.7	1.3		0	- 0		20.0	0
91 214	9	1,600,000	44,000	63.0	2' 00"	0	0	0	0	50.0	5.0		0	- 4.5		25.0	15.5
92	9	2,450,000	28,400	33.4	2' 15"	0	0	0	1.0	53.3	2.5		0	- 3,5		9.7	30.0
Average 166	17	1,781,857	35,937	51.7	2' 16"	0	0	0	2.3	45.3	8.6		0	- 2.2	· _	25.3	16.3
			G	roup 8.	Birds Sho	owing Sy	mptoms	and Lesie	ons of M	lyeloleu	kosis						
97 112	4	1,462,000	36,800	23.6	10′ 35″	0	0	32.4	0	11.0	10.0	_	9	1.8	0	18.1	26.7
98	7	1,340,000	41,000	30.0	7′ 30″	0	0	10.0	0	57.2	2.2		0	0	0	0	30.6
99 227	1	1,230,000	45,200	25.4	7' 36"	0	0	43.0	10.0	20.5	2.9		0	1.9	0	21.7	0
100	5	1,300,000	42,000	21.0	7' 40"	0	0	45.0	2.0	30.5	3.0		0	2.0	2.5	9.0	6.0
Average 174	4	1,333,000	41,250	27.5	8' 20"	0	0	32.6	3.0	29.8	4.5		0	1.4	0.6	13.3	15.8
			Gre	oup 9. B	irds Shov	ving Syn	nptoms a	nd Lesior	ns of Erg	ythroleu	kosis						
101	1	1,238,000	41,000	26.6	8' 30"	2.5	11.4	0	8.4	15.8	1.4	0	0	1.0 - 2.3	0	42.8	14.4
$102.\ldots$ $\begin{cases} 108 \\ 198 \end{cases}$	$\begin{array}{c}1\\89\end{array}$	$1,470,000 \\ 1,345,000$	$54,000 \\ 41,000$	$rac{26.0}{23.2}$	$\frac{10'}{11'} \frac{30''}{20''}$	$\frac{5.5}{0}$	$\begin{array}{c}12.7\\24.2\end{array}$	0 0	$\begin{array}{c} 0 \\ 1.0 \end{array}$	$\begin{array}{c} 10.0 \\ 14.5 \end{array}$	10.0 0	$egin{array}{c} 1.2 \\ 2.0 \end{array}$	$\begin{array}{c} 3.3\\ 2.0\end{array}$	$\begin{array}{ccc} 1.0 & 1.0 \\ 1.4 & 1.4 \end{array}$	$\begin{array}{c} 1.0\\ 2.0 \end{array}$	$\begin{array}{c} 38.9\\ 45.9\end{array}$	$15.4 \\ 5.6$
Average 127	45	1,351,000	45,333	25.3	10' 7"	2.7	16.1	0	3.1	13-4	3.8	1.1	1.8	1.1 1.6	1.0	42.5	11.8

Coccidiosis

Fifteen uncomplicated cases of coccidiosis were found in Flock C and none in the other flocks. No evidence was found in these studies to indicate that coccidiosis is in any way related to neurolymphomatosis. The hematological studies of the birds affected with coccidiosis are omitted since they add nothing of importance to the investigation.

An Attempt to Differentiate Neurolymphomatosis in Sick Birds from the Range by the Blood Count

The birds in Table 6, Group 1 were sent to the Diagnostic Service from Flock D and it was impossible to hold them under observation for any great length of time. This explains why the leucocytes containing rods and granules were counted together. Also, the neutrophilic leukocytes were not distinguished

TABLE 6.	HEMATOLOGY OF BIRDS SHOWING GROSS LESIONS OF NEUROLYM-
	PHOMATOSIS

Bird No.	Age. Days	Red Blood Cells	White Blood Cells	Hemoglobin	Blood Clotting Time	Erythroblasts	Polychrome Erythrocytes	Eosinophilic Myclocytes	Eosinophilic Leukocytes (Rods and Granules)	Polymorphonuclear Leukocvtes	Neutrophilic and Basophilic Mvelocytes	Neutrophilic Leukocytes and Mast Cells	Small Lymphocytes	Large Lymphocytes and Mononuclears	Diagnosis
	Group 1. Birds from Flock D														
34 35 36 37 38 39 40 41 42 43 Ave.	$\begin{array}{c} 282\\ 208\\ 245\\ 138\\ 138\\ 232\\ 246\\ 368\\ 149\\ 169\\ 270\\ \hline 222\\ \end{array}$	3,024,000 2,250,000 3,030,000 1,960,000 3,224,000 3,224,000 3,124,000 3,390,000 2,250,000 2,2715,091	25,000 38,400 42,400 17,400 14,200 21,700 24,200 64,000 29,000 22,600 12,800 28,336	91.4 88.4 92.1 87.5 74.5 88.3 86.3 92.6 90.4 	1' 30" 1' 39" 3' 24" 2' 25" 1' 15" 2' 12" 2' 6" 2' 46" 2' 15" 1' 57" 		0 0 0 0 0 0 0 0 0 0 0 0 0	0 0 0 0 0 0 0 0 0 0	25.3 37.2 34.5 31.6 34.9 35.0 27.3 51.2 30.2 4.9 29.3 33.8	$12.2 \\ 15.0 \\ 20.0 \\ 8.4 \\ 53.6 \\ 15.9 $	0 0 0 0 0 0 0 0 0 0 0	2.605.912.30.51.502.407.1	$\begin{array}{c} 41.2\\ 37.3\\ 16.2\\ 46.3\\ 23.7\\ 42.3\\ 50.0\\ 22.4\\ 44.2\\ 41.5\\ 30.7\\ \hline \\ 36.0\\ \end{array}$	22.2 18.2 9.8 18.4 10.0 6.2 6.4 14.8 0 17.0	$\begin{vmatrix} 0 \\ + \\ + \\ 0 \\ 0 \\ + \\ + \\ 0 \\ 0 \end{vmatrix}$
				Group :	2. Birds	; fro	m F	locł	сE						
44 45 46 47 48 49 50 51		3,000,000 2,210,000 3,648,000 3,240,000 3,914,000 3,200,000 2,390,000 3,000,009	$\begin{array}{c} 22,580\\ 35,200\\ 24,000\\ 23,800\\ 23,000\\ 74,820\\ 20,400\\ 41,000\\ \end{array}$	72.0 72.2 	1' 30" 2' 15" 1' 38" 1' 24" 3' 26" 2' 36" 3' 29"	0 0 0 0 0 0 0 0	0 0 0 0 0 0 0	0 0 0 0 0 0 0	$21.3 \\ 21.9 \\ 25.3 \\ 25.0 \\$	$ \begin{array}{r} 11.7 \\ 7.4 \\ 5.6 \\ 11.7 \\ 9.5 \\ 12.7 \\ \end{array} $	0 0 0 0 0 0 0 0	$ \begin{array}{c} 0.6 \\ 2.5 \\ 4.6 \\ 7.7 \\ 0.5 \\ \end{array} $	57.3 37.0 44.3 55.6 41.8 15.0 36.7 47.9	$\begin{array}{c} 0 & 7 \\ 24 & 5 \\ 12 & 3 \\ 13 & 5 \\ 50 & 0 \\ 16 & 0 \end{array}$	+ 0 + 0 +
Avera	ıge	3,075,250	33,100	81.3	2' 20"	0	0	0	27.6	11.1	0	2.3	42 .0	17.0	62.5

from mast cells, and the presence or absence of lymphoblasts in the blood stream was not determined. Aside from these differences the blood was stained and counted the same as for the birds in Flock C. The object of this experiment was to determine whether it is possible to diagnose neurolymphomatosis by roughly counting the blood. The technician counting the blood did not see the birds until the blood had been counted and a report made. Birds Nos. 36 and 40 had high leukocyte counts and were diagnosed correctly by the technician. Birds Nos. 37 and 39 had high lymphocyte counts and were diagnosed as neurolymphomatotic before necropsy. The blood counts of the other six were normal and the diagnosis made by the technician was not confirmed at necropsy. In this experiment the technician relying on blood counts diagnosed only 36.6 per cent of the cases of neurolymphomatosis correctly.

The birds listed in Table 6, Group 2, were in Flock E, which was a mixed group of birds coming from several places in Massachusetts. Birds Nos. 44, 47, 49, and 51 were down and unable to arise. Necropsy revealed extensive nerve infiltration, and blood counts showed marked lymphocytosis. Positive diagnoses were made in these cases from the blood counts. Bird No. 45 showed mild symptoms of disease and an examination of the blood revealed a leukocytosis. This case was diagnosed correctly by the blood count. Birds Nos. 46, 48, and 50 gave normal counts which led to mistaken diagnoses. In this trial 62.5 per cent of the cases of neurolymphomatosis were diagnosed correctly from the blood counts.

The Hematology of Birds from Flocks D and E Affected With Diseases Other Than Uncomplicated Neurolymphomatosis

Two birds in Flock D appeared to be paralyzed, but no lesions of disease were found at necropsy. A study of Table 7, Group 1, shows that both birds gave high lymphocyte counts. The cause of disease in these two birds was not satisfactorily determined. Histological sections of the nervous system did not reveal any evidence of infiltration.

The average age of the birds in Flock D was six months greater than in Flock C and the number of cases of neurolymphomatosis complicated by lymphocytomatosis was doubled. Group 2, shows that all of the red blood counts, except in birds Nos. 71 and 77, were low and the hemoglobin was low in all of the birds. The blood clotting time was prolonged in Nos. 67, 70, 72, 73, 74, 76, 80, 81, and 82. Eosinophilia was noted in Nos. 70, 73, and 78. Lymphocytosis was evident in bird No. 69. This study indicates that there is a definite relation between lymphocytomatosis and anemia.

Four birds in Flock D (Group 3) were affected with lymphocytomas only. The white cell count in all four was high. The differential counts show that the eosinophilic leukocytes were high in Nos. 93, 95, and 96, and the small lymphocytes were above normal in bird No. 94.

Eight cases of myeloleukosis were found in Flock D (Group 4). All of these birds showed relatively low red cell counts and hemoglobin determinations. The blood clotting time was prolonged so that the birds bled easily, and the white cell count was high in all except No. 109. This bird gave a normal white cell count. Eosinophilic myelocytes were present in the blood of all of the birds in varying percentages and the eosinophilic granular leukocytes were above normal. Basophilic myelocytes were identified in the blood of No. 103.

			TABLE	1. 111	MATOLOG	1 01 1	JIKD3 F	KOM II	JOCKS D AND I						
			(0						Eosinophilic Leukocytes	ar			cs	es ars	
Bird No.	Age, Days	Red Blood Cells	White Blood Cells	Hemoglobin	Blood Clotting Time	Erythroblasts	Pelvchrome Erythrocytes	Eosinophilic Myelocytes	With Rods With Granules	Leukocytes	Neutrorhilic and Basonhilic Mvelocytes	Neutrophilic Leukocytes and Mast Cells	Small Lymphocytes	Large Lymphocytes and Mononuclears	
Group 1. Birds from Flock D, Paralyzed and Showing No Lesions of Disease															
20															
60 60	$\frac{207}{349}$	2,844,000	29,640	81.4 90.4	1' 26"	ŏ	ŏ	0	$\begin{array}{r} 20.6 \\ 28.2 \end{array}$	11.1	ŏ	$\begin{array}{c} 1.0\\2.2\end{array}$	46.6	11.9	
Average	278	2,776,000	32,320	85.9	2' 2"	0	0	0	24.4	7.2	0	1.6	52.2	14.6	
	Group 2. Birds from Flock D, Showing Lesions of Neurolymphomatosis and Lymphocytomatosis														
67 68	$ 246 \\ 303 $	2,160,000 2,865,000	19,320 29,800	$\begin{array}{c} 72.1 \\ 87.4 \end{array}$	3' 27'' 1' 46''	0	0	$\begin{vmatrix} 0\\0 \end{vmatrix}$	$\begin{array}{c} 37.9\\31.7\end{array}$	$15.4 \\ 9.8$		0	$\begin{array}{c} 43.3 \\ 40.3 \end{array}$	$\begin{array}{c} 3.4 \\ 17.1 \end{array}$	
69 70	$ 145 \\ 377 $	2,770,000 2,662,000	$15,800 \\ 36,000$	$69.4 \\ 56.6$	1' 36" 4' 10"	0		0	$\frac{31.5}{89.3}$	1.8	0	0.9	$\frac{55.7}{4.5}$	$11.1 \\ 6.2$	
71	350	3,568,000	30,200	87.3	1' 54"	ŏ	ŏ	ŏ	17.9	31,1	ŏ	3.6	44.1	3.2	
$\dot{72}$ 73	315	2,940,000	20,400	67.1	4' 48"	0	0	0	$\begin{array}{c} 34.9\\ 47.2 \end{array}$	12.7	0	1.3	33.2	17.9	
73	276	1,854,000 2,342,000	35,000 35,000	$54.2 \\ 60.2$	3' 20" 3' 14"	0		0	47.2 31.4	$ \begin{array}{c} 12.2 \\ 6.8 \end{array} $	0	$\begin{vmatrix} 0\\ 3.0 \end{vmatrix}$	$\begin{array}{c} 25.4 \\ 28.2 \end{array}$	$15.2 \\ 30.6$	
74 75	132	2,342,000	16,000	50.3	2' 15''	ŏ	0 0	0 U	$31.4 \\ 32.4$	18.5	l õ	1.5	$\frac{20.2}{35.3}$	12.3	
76 77	142	2,986,000	19,000	59.6	3' 22"	Ö	Ó	0	39.8	4.8	ŏ	1.0	40.4	14.0	
77	242	3,080,000	5,046	42.3	$\frac{2' 33''}{2' 33''}$	0	0	0	$\frac{39.4}{79.4}$	5.2	0	1.0	39.4	15.0	
78 79	246 271	1,950.000 2,124,000	40,000 14,800	60.3 50.3	1' 30"		1 ŏ	0	$\begin{array}{c} 73.4\\ 32.4\end{array}$	$\begin{bmatrix} 0\\ 18.2 \end{bmatrix}$	l ö	$\frac{2.0}{0.5}$	$\frac{21.2}{36.8}$	$3.4 \\ 12.1$	
80	143	2,100,000	47,000	63.2	3' 20"	ŏ	ŏ	ŏ	26.2	6.7	0	4.6	40.0	22.5	
81 82	375	1,980,000	44,000	53.0	4' 49"	0	0	0	$\begin{array}{c} \overline{23},\overline{2}\\ 32,\overline{7}\end{array}$	6.3	0	2.9	38.6	29.0	
82	385	1,360,000	41,000	33.6	3' 19"	0	0	0	32.7	8.1	0	1.2	37.0	21.0	
Average	254	2,437,188	28,023	60.4	3' 00"	0	0	0	38.8	9.9	0	1 1.5 1	35.2	14.6	
			Group 3	8. Birds (D, Show	ing Lesio	ns of Ly	nphocytomatosis						
93	393	2,330,000	83,200	41.9	3' 44"	0	0	0	40.2	10.2	0	3.5	42.6	3.5	
94 95	151 260	2,810,000 1,849,000	58,000 79,200	$61.3 \\ 56.0$	3' 27" 2' 30"	0			1.0 40.4	$19.4 \\ 5.7$	0	$ \begin{array}{c} 0 \\ 1.2 \end{array} $	$\begin{array}{c} 75.3\\ 46.2 \end{array}$	$\frac{4.3}{6.5}$	
95 96	260 176	1,849,000	79,200	47.5	3' 10"	Ö	ŏ	l õ	46.2	10.4	ŏ	1.2 1.1	$\frac{40.2}{28.3}$	0.0 14.0	
Average	245	2,209,000	72,650	51.7	3' 13"	0	0	0	32.0	11.4	0	$\frac{1.1}{1.5}$	48.1	7.1	

TABLE 7. HEMATOLOGY OF BIRDS FROM FLOCKS D AND E

103 104 105 106 107 108 109 110 Average		143 403 416 402 305 310 305 126 301		$1,162,000\\1,774,000\\1,500,000\\1,230,000\\1,230,000\\1,238,000\\2,273,000\\2,480,000\\1,622,125$		40,000 60,000 53,000 45,000 62,000 80,000 27,000 42,000 51,125 rp 5. Birds f		32.4 39.3 41.8 47.3 42.1 49.3 42.4 68.4 45.4	12' 36" 10' 26" 9' 30" 8' 42" 11' 30" 12' 31" 14' 37" 12' 37" 11' 34" Showing	0 0 0 0 0 0 0 0	$ \begin{array}{c c} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\$	$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$	1	0 0 0 0 0 0 0.1 1.3 is and I	25.5 20.6 25.3 30.2 24.2 20.6 35.0 37.0 27.3	$ \begin{array}{r} 10.0 \\ 3.4 \\ 6.5 \\ 4.6 \\ 6.4 \\ 8.2 \\ 1.7 \\ 5.2 \\ \hline 5.8 \\ \end{array} $	- -	0.7 0 0 0 0 0 0 0 0 0 0	$\begin{array}{c} 3.6\\ 0.1\\ 4.4\\ 2.7\\ 0.8\\ 1.1\\ 2.3\\ 0\\ \hline 1.9\\ \end{array}$	$\begin{array}{c} 42.3 \\ 47.5 \\ 41.8 \\ 37.0 \\ 47.3 \\ 52.0 \\ 40.8 \\ 37.3 \\ \hline 43.3 \end{array}$	$\begin{array}{c c} 12.7 \\ 6.0 \\ 16.2 \\ 15.3 \\ 5.5 \\ 14.4 \\ 15.2 \\ 8.0 \\ \hline 11.7 \end{array}$
				,	Fou	ip 5. Dirus i	rom	FIOCK E	, snowing	Lesions	or neu	σιγμιφπο	1114105	is and L	ymphocyt	UIIIatust	5				
83 84 85 86 87			, T	1,980,000 1,250,000 1,760,000 2,910,000 1,920,000		$\begin{array}{c} 25,900 \\ 42,000 \\ 40,000 \\ 90,200 \\ 41,000 \end{array}$		$53.2 \\ 51.0 \\ 50.4 \\ 60.9 \\ 53.6$	2' 16" 3' 20" 3' 00" 2' 30" 3' 32"	0 0 0 0	0 0 0 0 0	0 0 0 0			.8	$\begin{array}{c} 11.1 \\ 57.1 \\ 3.4 \\ 4.4 \\ 20.6 \end{array}$		0 0 0 0 0	$egin{array}{c} 1.3 \\ 2.3 \\ 0.9 \\ 1.4 \\ 2.1 \end{array}$	$\begin{array}{c c} 44.3 \\ -9.5 \\ 49.2 \\ -46.0 \\ -45.0 \end{array}$	$\begin{array}{c c}15.3\\28.5\\15.0\\11.4\\1.6\end{array}$
Average				1,964,000		47,820		53.8	2' 56"	0	0	0		25	.9	19.3		0	1.6	38.8	14.4
						Group 6. Bi	rds	from Flo	ck E, Shov	wing Syr	nptoms	and Les	ions of	f Myelo	leukosis						
111 112 113 114 Average		299 172 200 208 220	_	$1,496,000 \\996,000 \\1,120,000 \\1,426,000 \\1,259,500$		94,000 87,000 60,000 67,000 77,000		$ \begin{array}{r} 47.8 \\ 33.2 \\ 38.6 \\ 53.1 \\ 43.2 \end{array} $	10' 13" 4' 30" 10' 50" 7' 32" 8' 16"		0 0 0 0	$ \begin{array}{r} 8.0 \\ 9.2 \\ 16.5 \\ 3.8 \\ \overline{.4} \end{array} $		$ \begin{array}{r} 6.0 \\ 2.4 \\ 0 \\ 5.0 \\ \hline 3.4 \end{array} $	$ \begin{array}{r} 4.2 \\ 15.2 \\ 2.6 \\ 15.1 \\ \hline 9.3 \end{array} $		_ -	$ \begin{array}{c} 2.8 \\ 0 \\ 4.0 \\ 7.0 \\ \hline 3.5 \end{array} $		$ \begin{array}{r} $	$ \begin{array}{r} 4.0\\ 16.4\\ 45.3\\ 16.8\\ \hline 20.6 \end{array} $
				0	-	D: 1 / F															
				Grou	p7.	Bird from F	1001	CE, Sho	wing Lesio	ns of Ly	mpnocy	tomatosi	s com	plicated	by Myelo	neukosi	IS				
115	1	300		2,183,000	I	90,000	i	45.3	9' 34″	0	0 [15.2	2] (0	8.3 ¦	4.5	ł	3.0	3.0	54.6	11.4
					Gr	oup 8. Bird	fro	n Flock	E, Showing	g Lesion	5 of Ne	urolymph	omate	osis and	Coccidios	is					
116	1	102	ļ	1,769,000	I	27,000	Ι	66.5	3′ 47″	0	0	0	10	.0	17.2	5.1		0	3.2	51.6	12.9

Group 4. Birds from Flock D, Showing Symptoms and Lesions of Myeloleukosis

25

All of the birds in Flock E suffering from neurolymphomatosis complicated with lymphocytomatosis, as shown in Table 7, Group 5, had anemia, as indicated by the low red blood counts and hemoglobin determinations. The white blood cells of the birds were high, except in No. 83. The differential counts indicate that bird No. 84 had leukocytosis, while Nos. 85, 86, and 87 had both leukocytosis and lymphocytosis. At necropsy all of the birds showed lesions of neurolymphomatosis and lymphocytomatosis. The neurolymphomas were confined to the sciatic nerves and the lymphocytomas to the adjacent kidney tissues in birds Nos. 83, 85, 86, and 87. In No. 84 general metastasis had apparently taken place, for the left brachial plexus, liver, spleen, intestines, kidneys, proventricuius, lungs, and muscles adjacent to the left brachial plexus were involved in tumor formations. Histological sections indicated that these tumors were all lymphocytomas.

Four cases of myeloleukosis were found in Flock E (Group 6). The most characteristic lesions found in birds Nos. 111 and 112 were chloromas. The neoplasm in No. 111 had apparently originated in the liver and had metastasized to the muscles of the left breast. In bird No. 112 the myelocytoma was generalized and appeared in the liver, spleen, heart, and left thyroid gland. Myeloblasts and myelocytes were found in the blood of all four birds. Fresh myelocytes gave positive oxydase tests. Birds Nos. 113 and 114 did not show characteristic lesions of myeloleukosis on gross examination. But histological examination of tissues from the liver of No. 113 showed myelocytes in the blood of the arteries, sinuses, and capillary spaces. The kidneys were inflamed and swollen, and neoplastic cells were demonstrated in the interstitial tissues surrounding the glomeruli and tubules. The spleen was swollen and the tissues selected from it indicated that a generalized infiltration was taking place in this organ. The mucosa and muscular walls of the intestine were inflamed, but myelocytes were not found in the tissues selected from them. The thyroid glands and vagus nerves were normal to gross inspection, although myelocytes were found in the blood of arteries and capillaries leading into them. One of the thymus glands showed a diffuse infiltration of myelocytes on histological examination. All of the tissues selected from bird No. 114 for microscopical study were normal, except the bone marrow. This was inflamed, devoid of fat, low in eosinophilic leukocytes, and showed evidence of calcification.

One case of lymphocytomatosis complicated by myeloleukosis was found in Flock E, and this was in pullet No. 115. At necropsy lymphocytomas were found involving the liver, spleen, and mesenteries. Reference to Table 7, Group 7, shows that the eosinophilic myelocyte and the small lymphocyte predominated in the blood of this bird.

Only one complication of neurolymphomatosis and coccidiosis was found in this study and that was in Flock E. The hematology of this bird (Table 7, Group 8) shows anemia, eosinophilia, and lymphocytosis as prominent symptoms of this complication.

The Hematology of Healthy Birds

Fifteen healthy birds were examined hematologically for comparison with the results of those in diseased conditions. A study of Table 8 shows considerable fluctuation not only in different birds but in the same bird at different periods. While the examination of the blood is of considerable value in the diagnosis and differentiation of the various forms of leukemia, it is not of much help in the

Bird No. Age. Days	Days Under Observation	Red Blood Cells	White Blood Cells	Hemoglobin	Blood Clotting Time	Erythroblasts	Polychrome Erythrocytes	Eosinophilie Myelocytes		With Granules	Polymorphonuclear Leukocytes	Neutrophilic and Basophilic Myelocytes	Neutrophilic Leukocytes and Mast Cells	Small Lymphocytes	Large Lymphocytes and Mononuclears
$115,\ldots, 126\\191$	$\frac{1}{65}$	3,820,000 3,210,000	$24,600 \\ 24,800$	$97.2 \\ 93.7$	$\frac{1'}{1'} \frac{15''}{13''}$	0 0	0	0		$\frac{21.6}{25.4}$	$15.4 \\ 6.6$	0	5.7 4.2	$\begin{array}{c} 40.2\\ 40.2\end{array}$	$12.1 \\ 13.4$
$116,\ldots,\ldots, \begin{bmatrix} 102\\ (178$	1 76	3,980,000 3,845,000	$22,800 \\ 21,490$	$ \begin{array}{r} 92.3 \\ 89.8 \end{array} $	$\frac{1'}{1'}\frac{35''}{46''}$	0	0 0	0 0	8.1 11.4	$\frac{12.5}{24.2}$	$\frac{19.0}{16.4}$	0	7.1 1.5	$\frac{46.2}{41.0}$	$7.1 \\ -5.5$
117	1 -43	3,197,000 3,278,000	$20,682 \\ 22,794$	$\frac{85.4}{91.0}$	2' 10'' 1' 36''	0	0 0	0	8.7 2.2	37.5 34.7	$\frac{10.0}{5.0}$	0	$\frac{3.6}{4.2}$	$\frac{34.4}{41.8}$	$\frac{5.8}{12.1}$
118	$\frac{1}{52}$	3,700,000 3,442,000	$21,000 \\ 21,000$	$\begin{bmatrix} 73.8 \\ 71.5 \end{bmatrix}$	1' 15" 1' 40"	0	0	0	$\frac{5.7}{9.8}$	$\frac{19.5}{24.2}$	$7.6 \\ 8.0$	0	$rac{5.6}{2.6}$	44.8 39.0	$\frac{16.8}{16.4}$
119	1	3,100,000	30,000	91.0	2' 00"	0	0	0	8,0	10.0	12.7	0	4.0	59.6	5.7
$120\ldots\ldots120$	1	2,870,000 3,100,000	$27,000 \\ 23,400$	$76.3 \\ -80.4$	$\begin{bmatrix} 1' & 20'' \\ 2' & 24'' \end{bmatrix}$	0	0 0	0	-4.5 -11.5	$\frac{8}{7}$	$\begin{array}{c} 10.0 \\ -6.0 \end{array}$	0 0	$\frac{8.3}{3.3}$	$\frac{46.7}{51.6}$	$\frac{22.2}{17.2}$
121	1	2,945,000	24,746	85.4	1' 38″	0	0	0	0	25.3	2.8	0	2.8	50.7	18.4
122 100	1	2.240,000	24,000	88.4	2' 00"	0	0	0	5.0	33.4	15.0	0	3.3	40.0	3.3
123	1	2,970,000	17,500	87.3	4' 10"	0	0	0	0.2	20.7	4.6	0	7.2	45.6	11.7
124	1	3,050,000	20,000	75.6	1' 30"	0	0	0	10.0	20.0	15.0	0	5.0	40.0	10.0
125 131	1	3,040,000	19,400	79.5	1' 20″	0	0	0	0	39.7	15.1	0	5.6	16.9	22.7
126 133	1	2,450,000	31,600	88.2	1' 30"	0	0	0	3.1	22.5	30.7	0	3.0	27.9	12.8
127	T	3,290,000	33,800	77.1	1' 30″	0	0	0	0.3	46.9	3.3	0	3.3	31.9	14.3
128	1	2,930,000	28,800	91.0	1' 50"	0	Ð	0.	50.0	15.0	1.1	0	1.7	25.0	7.2
129	1	2,760,000	17,800	90.4	1' 40"	0	0	0	48.6	0.4	6.7	0	1.0	42.3	1.0
Average	19	3,160,850	24,026	85.3	1' 47"	0	0	0	10.3	23.0	10.6	0	4.2	40.3	11.8

TABLE 8. HEMATOLOGY OF BIRDS FROM FLOCKS C, D, AND E SHOWING NO SYMPTOMS OR LESIONS OF DISEASE

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diagnosis of either neurolymphomatosis or lymphocytomatosis. This investigation indicates that blood counting would not be effective in detecting birds in the early stages of neurolymphomatosis, as suggested by Johnson (1933) and Seagar (1933), because fluctuation in normal birds may temporarily surpass the leukocytosis and lymphocytosis occurring in paralyzed birds. Furthermore, there is usually a period between leukocytosis and lymphocytosis of birds affected with neurolymphomatosis in which there is a tendency for the progress of the disease to be arrested, and the blood picture is often normal during this period. All of these conditions are met in flocks coming down with neurolymphomatosis so that blood counts are unreliable and should not be accepted as a diagnosis, unless accompanied by gross or histological lesions.

During the course of this study the question arose, is there any way of checking the results? A search of the literature did not reveal any way of checking anything except the hematology of healthy birds, and that was not very satisfactory because so many different methods were used that the results are not comparable throughout. Table 9 contains the results of some of the more recent investigations on the hematology of healthy birds.

			x	ļ	Eosinop Leukoc		5		Lympho		
	Number of Birds	Red Blood Cells	White Blood Cells	Hemoglobin	With Rods	With Granules	Polymorphonuclear Leukocytes	Mast Cells	Small	Large	Monocytes
Blain, 1928.	6		18,630		49.4	8.7		3.6	32.8	3	5.7
Breusch, 1928		3,468,000	33,300		17.7	4.1		2.5	66.5	5	9.2
Burnett, 1917		3,300,000	17,921	76.0%	3.3		28.8	4.3	58.0)	5.5
Cook and Dearstyne,	li										
1934	78	2,841,000	16,359	10.8 mg	3.1		45.4	5.0	28.7	4.9	14.5
Fernstermacher, 1932.	12	2,594,188	24,425	76.0%							
Forkner, 1929	8	3,267,000	24,580	62.9%	34.7	1.8		4.2	41.8	3	17.1
Klieneberger and Carl,				[
1912		3,117,000	35,000	62.0%	29.5	4.5		2.2	63.8	3	0
Present Investigator				1	1		1	1	1		1
1934	20	3,160.850	24,025	85.3%	10.4	23.1	10.5	4.2	[40.3]	11.8	

 TABLE 9. THE HEMATOLOGY OF HEALTHY BIRDS AS DETERMINED BY

 Different Investigators

A study of Table 9 shows that the same method was used for counting the red and white blood cells by all investigators and the results are comparable. Since hematology has suffered from a lack of a single, simple, accurate method for determining hemoglobin, Pepper and Farley (1933), the results of the various methods in use cannot very well be expected to agree. The present writer used Wong's method and there is no comparative work with birds available. Burnett, Fernstermacher, Forkner, and Klieneberger and Carl used the Newcomer Hemoglobinometer adjusted to read in percentage, and their results are fairly uniform. Cook and Dearstyne determined the hemoglobin colorimetrically with the Newcomer Hemoglobinometer in grams per 100 cc. of blood and there is no other work available for comparison. Blain, Breusch, Forkner, and Klieneberger and Carl counted the eosinophilic leukocytes with rods and eosinophilic leukocytes with granules separately, but included the polymorphonuclear leukocytes without rods and granules in either one or the other of these groups. Burnett and Cook and Dearstyne recognized polymorphonuclear leukocytes but counted the eosinophilic leukocytes with rods and eosinophilic leukocytes with granules together. Fernstermacher did not include any differential counts. The present writer distinguished the three groups of leukocytes under discussion; but because of variations in counting, the results secured by the different investigators are not comparable.

The mast cell is rather easy to identify in the blood of the healthy fowl, and the variations in counts between the different investigators is probably no more than could be expected between different groups of birds. Therefore, these results are comparable.

Blain, Breusch, Burnett, Forkner, and Klieneberger and Carl did not distinguish small from large lymphocytes as did the more recent investigators, Cook and Dearstyne. The present writer included the monocytes with the large lymphocytes. The results of these investigations on the lymphocytes and monocytes are too variable for comparison.

Since the disagreements by hematologists are almost entirely due to differences in technique, their results do not invalidate the present study in which the same methods were used throughout and the results secured with healthy birds are comparable with those suffering from disease, but they do indicate the need for uniformity in reporting the results of hematological studies for comparison.

Summary and Conclusions

1. The clinical course of neurolymphomatosis varies in individual cases, usually extending over a period of several weeks or months. Complete recovery is rare. Temporary improvement may occur followed by relapse and death.

2. Blood counts indicate that the disease begins with leukocytosis and ends with lymphocytosis. During the period of temporary improvement the blood may be normal.

3. The disease spreads slowly in affected flocks, with only a few birds becoming affected at a time, but new cases continue to develop until it is partially arrested in the late fall.

4. The mortality from neurolymphomatosis is reduced after the first year or two, although it is likely to reappear with each batch of new chickens for an indefinite number of years after it is once established in a flock.

5. Neurolymphomatosis and lymphocytomatosis seem to be closely associated. The former is primarily a disease of young birds from 3 to 10 months of age, while the latter is more common in birds 10 months of age or older.

6. Anemia is a symptom of lymphocytomatosis and leukosis and is not characteristic of neurolymphomatosis.

7. Lympholeukosis is not readily differentiated from lymphocytomatosis.

8. No difficulty was experienced in differentiating neurolymphomatosis from either erythroleukosis or myeloleukosis as the pathological cell is different in all three diseases.

9. The pathological cell appeared to be the same in neurolymphomatosis and lymphocytomatosis, and was characterized by its basic staining properties and the arrangement of the nuclear and chromatin material. 10. This cell was found invading nerves and muscular and visceral tissues by metastatic and direct infiltration. In incipient cases it first appeared in the capillary spaces and destroyed the nerve fibers and interstitial tissues by utilizing the nourishment for the organization of a tumor.

11. Evidence is presented which indicates that incipient lymphocytomatosis and neurolymphomatosis are due to the irritating action of foreign bodies introduced into the peritoneal cavity and tissues. Since bacteria were isolated only from cases in which peritonitis was manifest, the inciting agent is not always bacterial.

12. Attempts to demonstrate the presence of a filtrable virus in neurolymphomatosis and lymphocytomatosis in these experiments failed.

13. Neurolymphomas were most commonly found in the sciatic, vagus, brachial, celiac, optic, and mesenteric nerves; and lymphocytomas were usually located in the ovary, mesentery, kidney, liver, spleen, muscle, and skin.

14. No evidence was found in these studies to indicate that coccidiosis was in any way related to either neurolymphomatosis or lymphocytomatosis.

15. Because of irregularities in the hematology of birds affected with neurolymphomatosis, blood counts were of little assistance in removing diseased birds from affected flocks.

16. On the basis of the experimental data secured in these studies neurolymphomatosis and lymphocytomatosis may be controlled by eradicating affected birds from the flock as soon as noticed, adopting the best sanitary procedures and increasing the vigor of the birds by judicious breeding.

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