A STUDY OF TWO YEARS AUTOPSIES PERFORMED AT THE POULTRY PATHOLOGICAL LABORATORY, OREGON AGRICULTURAL EXPERIMENT STATION

by

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INTRODUCTION

This thesis presents a study of the diseases encountered while performing two thousand three hundred thirty-four autopsies at the Poultry Pathological Laboratory, Department of Veterinary Medicine, Oregon Agricultural Experiment Station. The work was accomplished during the biennium July 1, 1931, to June 30, 1933. It offers a limited survey of the diseases that are commonly encountered in the poultry flocks of Oregon. The diseases studied during this period were in general the same as those usually met within most parts of the United States where specialized poultry farms are commonly found. It presents the seasonal and geographical distribution of the specimens examined, but deals more specifically with the methods of diagnosis. The toxic effect of daffodil bulbs, force-fed to chickens, is demonstrated, and three cases of poisoning in the turkey from eating daffodil bulbs are reported.

The object of this thesis is to present a study of the poultry diseases encountered and their distribution in Oregon. It is felt a two year period of study supplies most of the information desired. Methods and observations

employed in making the diagnosis of these diseases are offered by text and illustrations.

MATERIALS

This study covers two thousand three hundred thirty-four specimens that were sent or brought to the Poultry Pathological Laboratory for examination. The material received is classified in Tables II and IV according to the species of bird or animal examined. In order that the pullorum disease material might be shown in a satisfactory manner the chicken diseases were presented under two separate headings according to the age of the birds.

Specimens were received from all sections of the state. Tables I and III show the distribution by counties of the specimens examined. Most of the specimens were received from the counties in which specialized poultry farming is practised extensively.

The material that was sent to the laboratory often had little, if any history that was helpful in making the diagnosis. When the owner brought the material to the laboratory, a history of the case often could be obtained which was of considerable aid in making the diagnosis. Both live and dead specimens were received for examination. It was not thought necessary to record here the condition in which each specimen was received. Specimens that were

"unsatisfactory" for examination are so recorded in Tables II and IV. Several specimens that were normal and not diseased were received for examination. Tables II and IV have such cases listed under the heading "no diagnosis". Also under this heading are recorded a few diseased cases the cause of which was not determined.

SEASONAL DISTRIBUTION

Tables II and IV present the diseases encountered each month. From this limited amount of data it is not possible to draw definite conclusions on the seasonal distribution of the diseases diagnosed. Three of the diseases most frequently encountered (coccidiosis, paralysis and blindness, and fowl-pox) appear to have a seasonal fluctuation. In making this observation one must not lose sight of the possibility that factors other than the season may be responsible for this variation. The age of the birds during the season and their susceptibility at this age may account for more of the increase of some of these diseases at certain seasons than the season itself.

GEOGRAPHICAL DISTRIBUTION

Tables I and III present by counties the monthly distribution of the specimens examined. These tables show very clearly that most of the specimens were received from

the counties with large poultry populations (4). From July 1, 1931, to June 30, 1932, Linn County poultrymen contributed the most specimens. From July 1, 1932, to June 30, 1933, Benton County poultrymen contributed the most specimens. No doubt the proximity of the people in these two counties to the laboratory influenced these numbers somewhat. From July 1, 1931, to June 30, 1932, there were four counties from which no specimens were received. They were Wheeler, Wallowa, Malheur, and Curry. From July 1, 1932, to June 30, 1933, there were three counties from which no specimens were received. They were Wheeler, Wallowa, and Harney. Specialized poultry farms are extremely scarce, if found at all, in the counties just mentioned.

DIAGNOSIS

Diagnosis is one of the essentials in the successful control of disease. An accurate diagnosis may be arrived at by macroscopic examination in some cases, while in others complex laboratory technique and procedure are necessary. The methods used in making the diagnosis of the common diseases that were encountered in this study are presented. Diseases requiring special technique as well as macroscopic observations for diagnosis are treated in text and illustrations. Since many diseases are

diagnosed by macroscopic consideration of lesions, symptoms, and a satisfactory history, the most important of this group are dealt with in more or less table form supplemented with illustrations. It is hoped that in describing these methods of diagnosis the reader will more easily understand the material offered in Tables II and IV.

In many cases more than one disease condition was encountered in the same specimen. In each instance an effort was made to determine the disease that was causing the most trouble and which seemed to be the predominating influence in connection with the bird's diseased condition. Tables II and IV present only the diagnosis that was considered to be the primary cause for the diseased condition of the bird.

DISEASES DIAGNOSED BY SPECIAL TECHNIQUE

Coccidiosis

Diagnostic procedure. The diagnosis of coccidiosis was accomplished with the aid of the microscope. Johnson (7) states, "Diagnosis of coccidiosis cannot be definitely determined without the microscope. Further, satisfactory diagnosis frequently depends upon the recognition of other forms than the oöcyst." In all cases, finding in numbers some of the coccidial forms (oöcysts, merozoites, or

schizonts) was considered basic in establishing the diagnosis. Observation of the gross lesions produced by the parasites aided in confirming the diagnosis and in estimating the severity of the disease. In the preparation of material for examination the smear method was used. Moderately thin smears were made with the content and scrapings of mucous membrane taken from four or five different places in the small intestine and one place in each caecum examined. These locations were as follows: about five or six inches from the gizzard, about the middle of the small intestine, about three or four inches anterior to the attachment of the caeca to the small intestine, and about the middle of each caecum examined.

Lesions. Lesions produced by some of the species of coccidia are very characteristic. The production of characteristic lesions is dependent upon the bird's ingesting a massive dosage (9) of sporulated oöcysts.

Eimeria tenella invades the caeca primarily and the rectum to a lesser degree. Tyzzer (16) states that "in severe infections, it [E. tenella] .... often occurs in patches scattered along the lower small intestine not infrequently even as high as the attachment of the yolk stalk". A severe infection of this parasite produces sloughing of the caecal mucosa and profuse hemorrhage into the lumen. If the bird survives after a few days the

hemorrhagic content becomes a caseous core the center of which is usually pinkish in color and crumbly in consistency.

Eimeria necatrix invades the small intestine along its entire length although the first half of the small intestine usually shows the most severe lesions and effects from the invasion. Dilation of the small intestine is pronounced, and from the serous surface extensive ecchymotic and petechial hemorrhages and small grayish-white spots may be seen. (Fig. 2). There is profuse hemorrhage and sloughing of the mucous membrane into the lumen of the small intestine. This content of blood, sloughed mucous membrane, and mucous is usually dark red or brownish in color and has a very fetid odor.

Eimeria acervulina may attack any portion of the small intestine although the anterior half usually develops the most severe lesions. The distinct lesions produced by this species and its prevalence have probably been responsible for what has often been called "duodenal coccidiosis". A massive infection of this parasite is characterized by a swollen spongy-appearing mucosa and an excessive amount of mucous in the lumen of the small intestine. Grayish-white patches in the mucosa, due to masses of oocysts, may be found the entire length of the small intestine, although they are more numerous in the

anterior half. The patches have a characteristic outline, their long axis being transverse to the length of the intestine. This makes the mucous membrane appear with grayish-white areas of cross striations. (Fig. 3).

Eimeria maxima attacks the small intestine, producing the most severe lesions in the anterior two-thirds. A severe infection of this parasite produces intense inflammation, and the mucous membrane appears swollen and congested. An excessive amount of mucous, flecked with blood, is often found in the lumen of the small intestine. At no time does the quantity of hemorrhage produced by this species compare with the profuse hemorrhage produced by a severe infection of Eimeria necatrix.

Eimeria mitis and Eimeria praecox usually do not produce lesions that are especially typical of the species. A severe infection of either species usually causes an excessive production of mucous. A massive dosage of E. mitis has been known to produce some hemorrhage. It is generally felt that of the six species of coccidia known to infect chickens E. praecox has the least detrimental effect on the host.

Occurrence. Tables II and IV present the monthly distribution of the occurrence of coccidiosis. For a more accurate consideration of the disease the cases were

divided into two groups, immature and mature chickens, according to the development of the birds. Birds that had not come into production were considered as immature. Birds that had come into production were considered as mature.

These tables show an apparent seasonal variation. The fact that young chickens are often highly susceptible is very likely one of the important factors contributing to this variation. During the first two or three months of the fowl's life the general practise is to use special sanitary precautions. In many cases after this preliminary period the growth of the birds makes necessary the moving to larger and more suitable quarters. This often necessitates placing the young birds in contact with older birds or in houses and on yards recently occupied by older birds. Under such circumstances these highly susceptible young birds are subjected to an increased amount (9) of infectious material before they have attained sufficient immunity (8) to withstand this increased dosage. It seems that this seasonal variation is more likely to be the result of common poultry practises rather than seasonal influence.

Most of the outbreaks were caused by mixed infections. Under the circumstances it was not practical to identify each species involved in the mixed infections. However,

it was generally observed that the species affecting the small intestine (E. acervulina, E. maxima, E. mitis, E. praecox, and E. necatrix)were found more frequently in mature chickens. The species E. tenella, which attacks the caeca, was found more frequently in the immature chickens.

An account of this disease as it is encountered in the state of Oregon may be found in a recent publication (10) from the Oregon Agricultural Experiment Station.

Pullorum Disease

The routine procedure followed in the diagnosis of pullorum disease at autopsy has been about the same in mature chickens as in chicks. Although the lesions are not the same in mature birds as they are in chicks, characteristic lesions often may be found in both the mature and young chickens. Characteristic lesions when found, are an aid in confirming the diagnosis. Whether lesions are present or not bacteriological methods must be relied upon to identify the infection and confirm the diagnosis.

Bacteriological procedure. A modified North's gelatin agar medium (3) was used to isolate the infection from the lesions or viscera of the bird. In the identification of the organism recovered as Salmonella pullorum,

blood serum from a bird known to be infected with S. pullorum was used to agglutinate the suspected organism. For further identification of the organism it was necessary to obtain the following fermentation reactions (5, 15, 12) to the following sugars in nutrient broth medium.

Dextrose		Mannite		Maltose		Lactose		Dextrin	
Acid	Gas	Acid	Gas	Acid	Gas	Acid	Gas	Acid	Gas
+	±	+	±	-	-	-	-	-	-

+ = positive
- = negative
± = positive or negative

Lesions. In most cases of pullorum disease in the mature female chicken, characteristic lesions may be found in the ovary. Typical lesions are malformed, angular ova of various sizes that have an attachment to the ovary longer than normal. (Fig. 6). In some cases the lesions are adhered to each other, to the surrounding viscera, and to the abdominal wall. The content of typical lesions is pasty or cheesy in consistency and creamy-yellow in color. This content is usually surrounded with an oily substance not unlike linseed oil.

In a few cases inflammation of the heart with pericarditis and myocarditis is found. Usually there are adhesions between the pericardial sac and the epicardium. A cheesy fibro-purulent exudate may also be found in the pericardial sac. Heart lesions can not be depended upon

for the gross diagnosis as they may be the result of some other infection. Bacteriological procedure is necessary to determine the specific infection responsible for the heart lesions.

Mature male chickens do not form typical lesions that might suggest the presence of S. pullorum infection. Lesions that are caused by S. pullorum in the male must be examined bacteriologically for an accurate diagnosis.

Lesions in chicks (Fig. 8, 9) are often suggestive of pullorum disease but as there are other diseases that may cause similar lesions, one can not depend entirely upon the lesions for a positive diagnosis. The lesions that are significant are found in the caeca and lungs.

The lesions in the caeca are cheesy-like cores of a yellowish or grayish-white color. When these cores are cut across they are found to be solid and without fecal material. The yellowish or grayish-white color extends into the center of the core. The center may be a little lighter in color than the outer surface. When lesions of this nature are found in chicks under three weeks old they may be considered as practically positive for pullorum disease. Common brooding practises are such that it would be most unlikely that coccidia (E. tenella) would infect chicks at this early age in sufficient numbers to produce lesions that might be confused with those of pullorum disease.

Lung lesions appear as nodular abscesses of a yellowish or grayish color. As the lung lesions may be confused with aspergillosis infection, bacteriological procedure is necessary to establish the specific cause of the lesions. Since aspergillosis in chickens is seldom encountered in Oregon one may be reasonably sure that most of the lung lesions are the result of infection with S. pullorum.

Occurrence. From Tables II, b, and IV, b, it is evident that pullorum disease in chicks was diagnosed more times than any other disease. Most of the cases were encountered during a comparatively short season of the year. This increase in number of cases during the spring can hardly be considered as due to any seasonal factor, but rather to the common practise among poultrymen of hatching most of the chicks at this time of year.

An account of this disease as it occurs in the state of Oregon may be found in recent publication (11) from the Oregon Agricultural Experiment Station.

Negative for pullorum disease. Chicks recorded under "negative for pullorum disease" in Tables II, b, and IV, b, were the occasional chicks from which no infection was obtained when infection had been isolated from other chicks from the same flock.

Fowl Cholera

The diagnosis of fowl cholera depended entirely upon isolating the causative organism Pasteurella avicida. Satisfactory history and lesions usually suggested a possible septicemic condition. Forms of the infection (6) that were not generally septicemic in nature were not considered.

Diagnostic procedure. The procedure followed in searching for the organism P. avicida consisted of making bacteriological cultures on a modified North's gelatin agar medium from the heart blood, liver, and spleen. Several microscopic slide preparations of the heart blood usually were made. Some of the preparations were stained with Loeffler's methylene-blue and others with Gram's method of staining. Dilute carbol-fuchsin was used to counter-stain after Gram's method as P. avicida is "Gram-negative" and bipolar (2) in staining characteristics. When the organism was recovered on the culture medium a suspension of it was injected into guinea-pigs. When the guinea-pigs died, usually within forty-eight hours, an examination was conducted in the same manner as that previously described for the chicken, in an attempt to recover the bipolar organism. If the organism killed the guinea-pig and was recovered again and identified, the diagnosis of fowl cholera was considered justified.

Lesions. There are no lesions in connection with fowl cholera that may be considered typical of that disease. There are lesions that suggest a general septicemia and with this guide a bacteriological and microscopic study can be made in an effort to isolate the infection. Usually the history reveals a sudden onset with varying degrees of mortality. At the beginning of the outbreak the birds often are found dead without having shown symptoms of illness. One usually finds the dead birds to be fat and in good flesh with cyanotic comb and wattles. Internally there is considerable congestion in all of the organs, particularly the liver, spleen, and kidneys. In the internal fat petechial and ecchymotic hemorrhages are often found. Usually there are petechial hemorrhages in the coronary fat of the heart. In some cases petechial hemorrhages may be found under the serous membrane that lines the thoracic cavity. The intestines may show areas of diffuse hyperemia under the serous as well as in the mucous membrane. Most of the visible blood vessels (veins) appear highly injected with blood. Under such conditions one should not fail to make a thorough bacteriological and microscopic search for an invading organism.

Occurrence. Fowl cholera is seldom encountered in Oregon. Two outbreaks, one in a flock of chickens, the other in a flock of young turkeys, with two specimens

from each outbreak were so diagnosed in the two years of this study. A possible explanation for the scarcity of cases of fowl cholera in commercial flocks in Oregon may be that Oregon poultrymen are primarily exporters of breeding stock and commercial poultry and therefore do very little importing. It would seem that the greatest danger lies in the return of contest and exhibition birds from districts where the disease is prevalent.

Tuberculosis

The diagnosis of tuberculosis depended largely upon the isolation, by staining, of "acid-fast" organisms, although lesions typical of the disease were essential in suggesting its presence.

Diagnostic procedure. The first step was to determine the extent of the infection in the various organs. Microscopic slide preparations were then made from lesions in the liver and spleen. It has been observed on many examinations that the preparations from the spleen have usually revealed the organism more frequently and more abundantly than the preparations from the liver. For this reason the preparation from the spleen usually was stained first in order to expedite the examination. The Ziehl-Neelsen method (13) of staining acid-resisting

organisms was used. When "acid-fast" bacteria were recovered from typical tubercular lesions the diagnosis of tuberculosis was considered complete.

Lesions. As a general rule birds are a year old or older before they develop lesions of sufficient severity to cause symptoms of illness. The organs most often affected are the liver, spleen, and intestines. Less frequently other organs may become involved. Typical lesions are ivory white in color and calcareous in consistency. They may vary in size from tiny microscopic abscesses to a large mass of abscesses involving an entire organ. Ordinarily the lesions are about the size of small peas and usually are easily separated from the spleen and liver tissue.

Occurrence. None of the cases of tuberculosis reported came from a specialized commercial poultry flock. All cases examined came from general farm flocks where insanitary conditions prevailed because of improper flock management. At present the indications are that avian tuberculosis is not a serious problem on Oregon poultry farms. However, this disease should be eradicated whenever encountered or the poultrymen of this state may duplicate the unfortunate experience now confronting many of the poultrymen of the middle west.

Paralysis and blindness (neuro-lymphomatosis gallinarum)

Diagnosis. The diagnosis of fowl paralysis and blindness was based almost entirely upon a satisfactory case history and the appearance of typical symptoms and lesions. There is controversy at present as to whether fowl paralysis and blindness are separate entities or whether they are manifestations of one or more of the types of leucosis. The wide-spread distribution of paralysis and blindness and the lack of information concerning many important phases, places them among the most serious menaces to the poultry industry today. A great deal of the investigational work published on fowl paralysis and blindness to the present time has confused rather than clarified our conception of many important factors in relation to the disease. This study lists the cases of fowl paralysis and blindness under one heading, notwithstanding there is no general agreement about the relationship between these two conditions.

Anamnesis. The case history usually reveals the following course of the disease in the flock. Ordinarily the flock is between the ages of two and twelve months when the first case appears. Rarely were cases encountered under two months of age. Birds twelve months old and older develop the disease, but seldom does the disease

make its first appearance in a commercial flock after the birds have reached the age of twelve months or more. The vast majority of cases were encountered between the ages of three and ten months. Unless some other disease was complicating the condition the bird usually would be fat and in good flesh when symptoms first appeared. After the disease was definitely established in the bird, recovery was exceedingly rare.

Symptoms. At first there would be a slight lameness or limp which might last for a short time or for several days before the bird would go down and be unable to use its legs normally. Usually the birds do not develop a complete paralysis but merely an incoordination of the legs. When the birds are unable to stand they often assume a rather characteristic posture (Fig. 10).

In a few cases the disease may manifest istelf by a drooping wing. The bird is unable to retain the wing in its proper position, and as the disease progresses the wing may droop till the flight feathers touch the floor. The manifestation by the wing has not been observed as often as the manifestation by the leg.

What was thought, in this study, to be another manifestation of this disease was lesions in the iris of one or both eyes. The iris of one or both eyes becomes

grayish in color and malformed in shape, and because of this abnormality the pupil becomes irregular in size and outline. When the iris becomes extensively involved the eyeball may enlarge and bulge slightly from the eye socket. The symptoms of lameness and drooping wing are readily detected. The eye lesions are more likely to escape detection until the bird begins to show symptoms of blindness. In this connection iritis (blindness) has not been observed in birds as young as those in which paralysis of the legs or drooping wings have been the first manifestation.

Lesions. Gross lesions (Fig. 12, 13) when found in the nerve tissue, usually appear as enlargements of the brachial or sciatic nerve plexuses or enlargments of the nerve trunk as a result of leucocytic infiltration. The infiltered areas become somewhat translucent and grayish in color in contrast to the milk white color of the normal nerve. In many cases excessive leucocytic enlargements in other organs may be found. The liver, spleen, and kidneys may contain areas of leucocytic infiltration. The ovary often develops leucocytic tumors of various sizes. These lesions, that often are associated with paralysis and blindness, may also be found in connection with leucosis. At the present time available information has not established the relationship between the various

leucocytic manifestations in connection with this disease.

The eye lesions (Fig. 11) are the result of the leucocytic infiltration, in front of the pigment, in the iris of the eye. This cellular infiltration causes the area affected to appear light grayish in color. Since the cellular infiltration progresses unevenly it produces an irregular margin to the iris that forms the edge of the pupil. As the infiltration advances it may involve the entire iris. It may also be noted that as the infiltration progresses the iris loses its ability to contract and expand. When this function becomes too greatly impaired the sight becomes affected. With intense infiltration the pupil may become practically obliterated, the eyeball enlarged and bulging slightly from the eye socket.

Microscopic sections (Fig. 14) of characteristic lesions reveal intense leucocytic infiltration. Some cases do not exhibit characteristic gross lesions. In these cases a microscopic study of suspected nerve tissue may aid in establishing the correct diagnosis.

Occurrence. Fowl paralysis and blindness have been observed in chickens from practically all sections of the state. Pappenheimer, Dunn, and Cone (14) report the presence of the disease from practically all of the United States and Canada. The disease has been reported

also from several foreign countries. It is generally considered to be very widely disseminated. It may be noted from Tables II and IV that most of the cases were encountered in the fall and winter months although the disease was found to occur during all seasons of the year. This seasonal increase may be accounted for by the general practise of hatching most of the chicks in the spring months. Because of this practise most of the birds reach the age of the most frequent occurrence of the disease during the fall and winter months.

Narcissus Bulb Poisoning

Although plants of the genus Narcissus are generally considered toxic (1) the available literature made no mention of the effect of this bulb when eaten by fowls. Poisoning from eating daffodil bulbs was encountered in a flock of about four hundred twenty-five turkeys in July 1931. Over a period of several weeks over seventy-five birds had sickened in the same manner and died. It was therefore thought advisable to conduct some feeding tests in order to obtain some specific data on the toxic effect of this bulb to poultry.

Experimental procedure. A supply of daffodil bulbs was obtained from the turkey range upon which the trouble had been diagnosed. These bulbs were force-fed to Single

Comb White Leghorn cockerels to test their toxicity. The following table presents the results of these tests.

Fowl No.	Body Wt. Lbs.	Bulbs fed: Number	Bulbs fed: Total Wt. Gms.	Bulbs fed: How prepared	Hours to death	Remarks
11	1¼	1	3½	pulp	39	None
12	1½	4	23	pulp	24	None
13	1½	3	12	quartered & halved	74	All pieces except one still in crop.
14	1½	1	9½	quartered	126	All pieces in crop except few layers.
15	1¼	4	22	crystals*	-	No ill effects.
15	1½	5	28½	crystals*	-	No ill effects.
16	1½	4	30	juice*	36	Crystals removed by centrifuge.

* Juice squeezed from the bulbs was centrifuged to remove the crystals. The juice was then decanted.

Symptoms and diagnosis. There was a sudden onset of symptoms which manifested themselves within a short time after a portion of the bulb left the crop. It seemed that there was very little, if any, of the toxic principle absorbed through the mucous membrane of the crop. The symptoms that were consistently shown by the birds were a progressive narcosis and cyanosis of the face, comb, and wattles. The birds stood with necks flexed and heads pulled in. As the poisoning progressed they squatted

down, became comatose and died without changing position. There were no characteristic lesions observed as a result of narcissus bulb poisoning. One must depend upon finding and identifying the bulbs in the crop or gizzard in order to establish the diagnosis.

Conclusions. From these feeding tests the following conclusions are drawn: (a) Daffodil bulbs are toxic to chickens. (b) Needle-like crystals centrifuged from the juice of the daffodil bulbs proved to be neither toxic nor injurious to chickens. (c) Juice from which the crystals had been removed by centifugalization was toxic when fed to a chicken.

DISEASES DIAGNOSED BY MACROSCOPIC OBSERVATION

Diagnosis	Anamnesis and symptoms	Macroscopic lesions
Fowl-pox	Lesions on comb, face, and wattles. Anorexia, decrease in flock activity and egg production. Usually low mortality.	Wart-like scabs on comb, face, and wattles. Cankers in the mouth, pharynx, and larynx (Figs. 24,25).
Laryngo-tracheitis	Sudden onset. Usually considerable mortality. Gasping, rales, and death from suffocation. Seldom encountered in Oregon.	Live birds have varying amounts of muco-hemorrhagic exudate in trachea. Fibrous muco-hemorrhagic plug in trachea of dead birds, causing asphyxiation.
Colds and roup	Onset of disease often follows sudden severe change of environment. Sneezing, rales, and sero-mucous discharge from eyes and nostrils.	Sero-mucous discharge from eyes and nostrils. Sinuses may swell with content becoming caseous. Foul odor from discharge.
Avitaminosis A (nutritional roup)	Flock appears dull and listless. Ration lacking in vitamin A. Sinuses swollen with pus (Fig.20) No particular odor from pus.	Swollen sinuses filled with caseous pus. Often eyeball is partly destroyed. Oesophageal glands become swollen and filled with caseous pus (Fig. 21).

DISEASES DIAGNOSED BY MACROSCOPIC OBSERVATION (cont)

Diagnosis	Anamnesis and symptoms	Macroscopic lesions
Visceral gout	Birds fed ration high in portein. Occasional bird affected, dies suddenly. Bird usually fat and in good flesh. No characteristic history or symptoms.	Silvery deposit of urates covering heart, liver, and other viscera. Deposits in pericardial sac and on heart are usually heaviest. Kidneys are distended with urates. In advanced cases joints of legs and feet may contain deposits of urates.
Leucemia	Bird may die suddenly or linger and become emaciated. Face, comb, and wattles become pale when bird lingers. Droppings may contain considerable yellowish or greenish mucous. No typical symptoms or history.	Mottled grayish color and various degrees of enlargement of the liver, kidneys, and spleen due to infiltration with leucocytes. Other organs may become involved in the same manner. Leucocytes may clump in tumorous masses (Fig. 23).
Botulism (limber-neck)	Spoiled food (such as canned vegetables, meat, etc.) eaten by bird several hours prior to appearance of symptoms. Bird gradually becomes prostrate, there being gradual relaxation of the muscles of the body. Muscles of neck are first to be noticeably affected (Fig. 22).	No characteristic lesions. History of eating spoiled food and typical symptoms establish diagnosis. Cyanosis and passive congestion is usually marked.

DISEASES DIAGNOSED BY MACROSCOPIC OBSERVATION (cont)

Diagnosis	Anamnesis and symptoms	Macroscopic lesions
Rickets	Growing chicks fed ration deficient in vitamin D and no direct sunlight available. Chicks squat down, do not like to walk.	Costo-vertebral joints are enlarged (Fig. 18). Vertebral-sternal joints of the ribs are enlarged. Ribs are wavy and show lack of proper calcification.
Demineralization	Mature birds in heavy production fed ration deficient in vitamin D and no direct sunlight available. Leg weakness in best birds. Production gradually decreases.	Vertebral-sternal joints of the ribs are buckled and wavy. Bones of the body show decalcification and are easily broken.
Perosis (slipped tendon)	Growing chicks or poults on wire floors or in battery brooders affected. Often birds being forced for rapid growth. Vitamin content of ration satisfactory. Birds go down with twisted legs and slipped tendons.	Tibia and metatarsal bones seem to have grown in twisted malformed manner. Calcification of bones is normal. Flexor tendons may be to one side of the groove over hock. Posteriorly the hock joint appears broad and flat.
Osteoporosis	Only occasional bird develops the condition. Some of the bones become malformed and enlarged. Often abnormal bones are not observed until bird is cooked.	Malformed irregular exostosis on one or several bones of the body (Fig. 17). Periosteum seems to lose control over the bone development.

DISEASES DIAGNOSED BY MACROSCOPIC OBSERVATION (cont)

Diagnosis	Anamnesis and symptoms	Macroscopic lesions
Entero-hepatitis (blackhead)	Chicken seldom affected. Turkey highly susceptible host. Some birds die suddenly, others become droopy, emaciated, and linger for many days before dying; still others may be carriers without showing symptoms. Sulfur-yellow caecal droppings often indicate the disease.	Liver shows circular areas of necrosis the surface of which is often slightly concave (Fig. 16). Mucosa of caeca show areas of necrosis that penetrates deeper tissue manifesting lesions on the serous surface (Fig. 15).
Aspergillosis	Symptoms appear slowly. Anorexia and at times rales and gasping. Gradual loss of weight. Birds kept under conditions favorable for the development of the fungus.	Yellowish abscesses in lungs. Fibro-purulent exudate attached to the wall of one or more air sacs. Conidia of Aspergillus fumigatus may be seen on surface of exudate. Bacteriological methods may be necessary to isolate the fungus.
Bumble foot	Birds flying down from high elevations to hard surface, such as roosts higher than normal and no litter on floor. Birds become lame do not like to walk.	Hot, painful swelling on pad of one or both feet. Swelling usually contains caseous pus.

SUMMARY

1. This thesis presents a survey chiefly of poultry diseases in Oregon made from July 1, 1931, to June 30, 1933, involving two thousand three hundred thirty-four diagnoses.
2. Methods used to diagnose the most common diseases encountered are offered in the text and illustrations.
3. The seasonal and geographical distribution of the specimens examined are shown.
4. Practically all of the specimens examined were either chickens (1964) or turkeys (342).
5. Some of the common diseases in chickens and their per cent of occurrence are: pullorum disease (31.2), coccidiosis (13.1), paralysis and blindness (6.6), tapeworms (4.5), fowl-pox (3.6), leucemia (2.1), tuberculosis (2.1), colds and roup (1.8)
6. Some of the common diseases in turkeys and their per cent of occurrence are: entero-hepatitis (14.9), gizzard impaction (11.9), necrotic enteritis (9.6). An uncommon case of poisoning in turkeys from eating daffodil bulbs was investigated. Feeding tests proved the bulbs to be highly toxic for chickens.
7. The economic importance of the diseases was found to be not necessarily in direct ratio to their percentage of occurrence.

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BIBLIOGRAPHY

1. Bernhard-Smith, A. Poisonous plants of all countries, Bailliere, Tindall and Cox, London, 1923.

2. Buchanan, Robert Earle and Murry, Charles. Veterinary bacteriology, W. B. Saunders Co. Philadelphia, 1916.

3. Difco Laboratories. Manual of dehydrated culture media and reagents. Fourth edition. Difco Laboratories, Detroit, Mich. 1923.

4. Fifteenth census of the United States, Agriculture, Oregon statistics by counties, First series. U. S. Department of Commerce, 1930.

5. Hendrickson, J. M. The differentiation of Bact. pullorum (Rettger) and Bact. sanguinarium (Moore). Report N. Y. State Veterinary College, 1926.

6. Hughes, Thomas P. and Pritchett, Ida W. The epidemiology of fowl cholera. III. Portal of entry of P. avicida; reaction of the host. The Jour. of Exp. Med., 51:239-248, 1930.

7. Johnson, W. T. Avain coccidiosis. Poultry Science, 2:146-163, 1923.

8. Johnson, W. T. Immunity or resistance of the chicken to coccidial infection. Oregon Agr. Expt. Sta. Bul. 230, 1927.

9. Johnson, W. T. Two basic factors in coccidial infection of the chicken. Jour. of Amer. Vet. Med. Assoc., N. S. 23, 70:560-583, 1927.

10. Johnson, W. T. Coccidiosis of the chicken. Oregon Agr. Expt. Sta. Bul. 314, 1933

BIBLIOGRAPHY (cont)

11. Johnson, W. T. and Dickinson, E. M. Pullorum disease (contagious white diarrhea) of poultry. Oregon Agr. Expt. Sta. Bul. 313, 1933.

12. May, Henry G. and Goodner, Kenneth. Comparative studies of Salmonella gallinarium and Salmonella pullorum. R. I. Agr. Expt. Sta. Bul. 204, 1926.

13. Morrey, Charles Bradfield. Laboratory exercises in general bacteriology. Eighth edition, Ohio State University press, Columbus, Ohio, 1922.

14. Pappenheimer, Alwin M., Dunn, Leslie C. and Cone, Vernon. A study of fowl paralysis (Neuro-lymphomatosis gallinarum). Storrs Agr. Expt. Sta. Bul. 143, 1926.

15. Rettger, Leo F. and Plastridge, Wayne N. Pullorum disease of domestic fowl, a monograph. Storrs Agr. Expt. Sta. Bul. 178, 1932.

16. Tyzzer, Ernest Edward. Coccidiosis of gallinaceous birds. The Amer. Jour. of Hygiene, [Reprint], Vol. X, No. 2, 1929.

TABLE I. REPORT BY COUNTIES OF DIAGNOSES FOR 1931-1932

COUNTIES	July	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	June	Total
Baker	-	-	-	-	1	-	-	-	6	-	-	-	7
Benton	8	4	15	13	6	4	5	3	18	11	28	23	138
Clackamas	6	1	1	6	9	6	-	-	21	8	4	-	62
Clatsop	-	-	-	-	-	-	-	4	2	14	-	-	20
Columbia	2	5	4	-	2	1	-	-	6	-	1	3	24
Coos	-	5	-	-	3	-	-	-	4	8	-	-	20
Crook	-	-	-	-	-	-	-	-	-	-	6	-	6
Curry	-	-	-	-	-	-	-	-	-	-	-	-	0
Deschutes	-	-	-	-	1	6	1	-	2	-	-	1	11
Douglas	23	8	9	3	3	4	-	5	-	20	5	13	93
Gilliam	-	1	-	-	-	-	-	-	-	-	-	-	1
Grant	1	-	-	1	-	-	-	-	1	-	-	-	3
Harney	-	-	-	-	-	-	-	-	-	2	-	-	2
Hood River	3	-	-	-	2	-	2	-	7	8	-	-	22
Jackson	-	1	2	-	-	-	3	4	6	4	2	1	23
Jefferson	-	-	2	-	-	-	-	-	-	-	-	-	2
Josephine	1	-	8	-	-	-	-	-	1	2	2	3	17
Klamath	2	-	-	1	-	-	1	1	-	12	-	6	23
Lake	-	-	-	-	-	-	-	-	-	3	-	-	3
Lane	15	7	8	12	6	5	6	11	3	10	20	19	122
Lincoln	2	-	-	-	1	-	-	1	1	2	-	-	7
Linn	3	6	5	14	8	15	13	7	20	17	32	10	150
Malheur	-	-	-	-	-	-	-	-	-	-	-	-	0
Marion	6	30	5	1	9	14	8	9	8	33	11	9	143
Morrow	-	-	-	-	-	3	-	-	-	1	-	-	4
Multnomah	3	5	-	2	2	14	4	5	7	11	-	1	54
Polk	4	5	-	7	-	1	2	1	-	7	14	1	42
Sherman	-	-	2	1	-	-	-	-	-	-	5	-	8
Tillamook	1	-	-	2	-	-	5	-	-	-	7	-	15
Umatilla	-	-	4	5	1	-	-	-	9	8	2	4	33
Union	-	-	-	-	-	-	1	-	-	-	-	-	1
Wallowa	-	-	-	-	-	-	-	-	-	-	-	-	0
Wasco	-	-	-	10	-	-	-	-	6	-	1	-	17
Washington	6	-	7	2	8	5	6	-	19	42	21	-	116
Wheeler	-	-	-	-	-	-	-	-	-	-	-	-	0
Yamhill	14	1	-	2	2	1	2	7	11	5	11	9	65
	100	79	72	82	64	79	59	58	158	228	173	103	1254

TABLE II. MONTHLY REPORT OF DISEASES FOR 1931-1932

a. CHICKEN (Four wks. old or older)	July	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	June	Total
Coccidiosis													
mature birds	5	1	10	12	11	13	4	1	3	2	1	1	64
immature birds	15	4	8	6	1	-	-	-	1	10	15	17	77
Tapeworms	10	5	4	11	4	3	5	1	8	3	2	-	56
Capillaria	3	-	1	1	2	-	-	2	-	1	5	-	15
Ascarids	1	4	1	-	-	1	-	-	-	1	1	-	9
Gizzard worms	2	-	-	1	-	-	-	1	1	-	-	-	5
Lice	1	-	-	1	2	1	1	1	1	1	3	-	12
Paralysis and blindness	4	4	9	17	4	2	1	-	3	-	6	12	62
Fowl-pox	-	-	-	2	3	10	10	-	3	-	3	-	31
Tuberculosis	1	2	1	-	1	-	6	4	2	6	1	1	25
Colds and roup	2	4	2	7	6	2	-	-	2	-	1	-	26
Pullorum disease	-	1	-	2	6	4	2	1	1	4	1	2	24
Laryngotracheitis	1	4	-	2	-	-	-	-	-	-	-	3	10
Fowl cholera	-	2	-	-	-	-	-	-	-	-	-	-	2
Aspergillosis	-	-	-	-	-	-	-	-	-	-	1	-	1
Unsatisfactory	1	1	1	2	1	2	-	-	1	-	1	-	10
No diagnosis	-	-	5	-	1	4	2	3	1	4	1	-	21
Leucemia	-	-	3	-	2	3	3	3	7	1	3	-	25
Tumors	1	-	2	2	4	6	-	-	7	-	1	1	24
Egg concretions	-	1	3	-	2	2	2	4	2	-	-	1	17
Peritonitis	-	2	2	2	1	-	1	3	1	-	-	1	13
Bumblefoot	-	-	1	-	-	-	-	-	-	-	-	-	1
Vent infection	-	-	-	-	-	-	3	1	-	-	1	-	5
Necrotic enteritis	5	-	-	-	-	-	-	-	-	-	1	-	6
Proventriculitis	4	2	-	4	-	2	-	-	2	-	-	-	14
Canker	-	4	-	1	-	-	1	-	-	-	1	-	7
Edema of wattles	-	-	-	-	1	-	-	-	-	-	-	-	1
Entero-hepatitis	-	-	1	-	-	-	-	-	-	-	-	-	1
Botulism	-	-	2	-	-	-	-	-	-	-	-	1	3
Rickets	-	-	-	-	-	-	-	-	-	-	3	-	3
Demineralization	-	-	-	-	-	-	-	2	-	1	-	-	3
Perosis	2	-	-	-	-	7	-	-	-	-	-	-	9
Visceral gout	-	-	1	-	-	-	1	1	1	-	-	-	4
Osteoporosis	1	-	-	-	1	-	-	-	-	-	-	-	2
Pericarditis	-	-	-	-	-	-	-	1	-	-	-	-	1
Wounds, lacerations	-	-	-	1	-	-	-	-	-	1	-	-	2
Fractures	-	-	-	-	2	-	-	-	-	-	-	-	2
Impactions	-	1	-	-	-	1	-	-	-	-	4	1	7
Jaundice	-	-	-	-	-	-	-	1	-	-	-	-	1
Poisoning	-	-	1	-	-	-	-	-	-	-	2	1	4
Hemorrhage	1	-	-	-	-	1	-	-	-	-	-	-	2
Prolapse	-	-	-	-	-	-	2	-	-	-	1	-	3
Diverticulum	-	-	-	1	-	-	-	-	-	1	-	-	2
Intussusception	-	-	-	-	-	-	-	1	-	-	-	-	1
Abscess	-	-	-	-	-	-	3	1	1	-	-	-	5
	60	42	58	75	55	64	47	32	48	36	59	42	618

TABLE II (cont). MONTHLY REPORT OF DISEASES FOR 1931-1932

b. CHICK (Under four weeks of age)	July	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	June	Total
Pullorum disease	-	14	11	-	-	8	-	20	87	114	33	12	299
Negative for pullorum disease	-	8	-	-	-	5	-	2	12	34	16	2	79
No diagnosis	-	-	-	-	-	-	6	-	3	19	11	-	39
Unsatisfactory	-	-	-	-	-	-	-	-	3	9	3	2	17
Rhinitis	-	-	-	-	-	-	-	-	-	6	-	-	6
Aspergillosis	-	-	-	-	-	-	-	-	-	3	-	-	3
	0	22	11	0	0	13	6	22	105	185	63	16	443
c. TURKEY (All ages)													
Entero-hepatitis	6	5	-	-	2	-	1	2	1	5	1	3	26
Necrotic enteritis	7	-	-	-	-	-	1	-	-	-	4	8	20
Tuberculosis	-	-	-	1	-	-	-	-	-	-	-	-	1
No diagnosis	-	-	-	-	-	1	-	-	-	-	7	6	14
Unsatisfactory	6	1	-	-	-	-	-	1	-	-	1	3	12
Fowl-pox	-	-	-	2	-	-	-	-	2	-	-	-	4
Aspergillosis	-	-	-	-	1	-	1	-	-	-	-	-	2
Colds and roup	-	-	2	-	1	-	-	-	-	-	-	-	3
Pullorum disease	8	-	-	-	-	-	-	-	-	-	2	-	10
Tapeworms	2	-	-	-	1	-	-	-	-	1	-	-	4
Botulism	-	2	-	-	-	-	-	-	-	-	-	-	2
Crop mycosis	-	-	-	-	-	-	-	-	1	-	-	-	1
Omphalitis	-	-	-	-	-	-	-	-	-	-	7	1	8
Catarrhal enteritis	-	-	-	2	-	1	1	-	-	-	-	6	10
Hemorrhagic enteritis	-	-	-	-	-	-	-	-	-	-	1	1	2
Slipped tendons	-	-	-	-	-	-	-	-	-	-	4	-	4
Rickets	3	-	-	-	-	-	-	-	-	-	-	-	3
Bumblefoot	-	1	-	-	-	-	-	1	-	-	-	-	2
Pneumonia	-	-	-	-	1	-	-	-	-	-	-	-	1
Legweakness	-	-	-	2	-	-	-	-	-	-	-	-	2
Foreign bodies	-	-	-	-	1	-	-	-	-	-	-	-	1
Poisoning by narcissus bulbs	3	-	-	-	-	-	-	-	-	-	-	-	3
Impaction (gizzard)	3	-	1	-	-	-	-	-	-	-	12	14	30
Ascites	-	-	-	-	-	-	-	-	-	-	11	-	11
Peritonitis	-	-	-	-	-	-	-	-	-	-	-	1	1
Coccidiosis	-	-	-	-	-	-	-	-	-	-	-	2	2
	38	9	3	7	7	2	4	4	4	6	50	45	179
d. CHINA PHEASANT													
Tuberculosis	-	-	-	-	-	-	1	-	-	-	-	-	1
No diagnosis	-	5	-	-	-	-	-	-	-	-	-	-	5
e. GROUSE													
Entero-hepatitis	1	-	-	-	-	-	-	-	-	-	-	-	1
f. GEESE													
No diagnosis	-	1	-	-	-	-	-	-	-	1	-	-	2
g. RABBITS													
Coccidiosis	1	-	-	-	2	-	1	-	1	-	-	-	5
	2	6	0	0	2	0	2	0	1	1	0	0	14

TABLE III. REPORT BY COUNTIES OF DIAGNOSES FOR 1932-1933

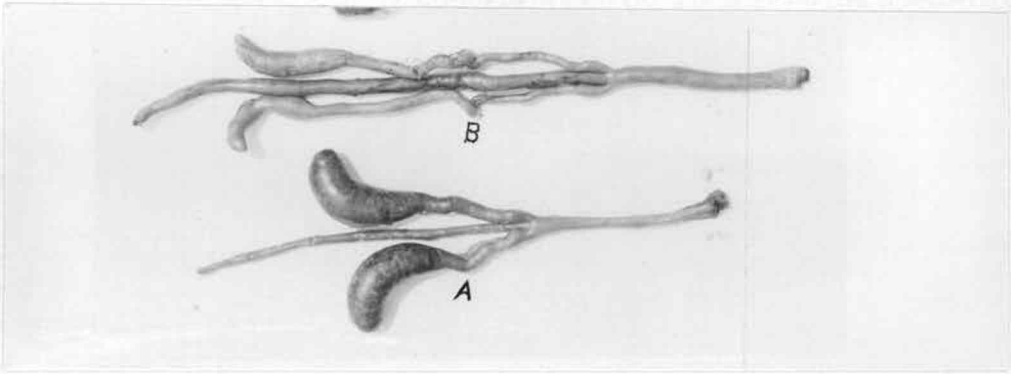
COUNTIES	July	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	June	Total
Baker	-	-	-	-	1	-	1	-	-	-	-	-	2
Benton	25	7	14	5	12	4	20	9	5	20	34	8	163
Clackamas	-	-	6	2	6	6	1	9	21	23	9	6	89
Clatsop	-	-	-	2	-	-	-	2	2	-	3	-	9
Columbia	-	-	-	5	2	-	4	5	-	-	19	-	38
Coos	1	-	-	-	-	1	-	-	-	3	1	-	6
Crook	5	2	-	-	1	-	-	-	-	-	-	-	8
Curry	-	-	-	-	-	-	-	1	-	-	-	-	1
Deschutes	1	2	-	-	1	-	1	-	-	1	7	-	13
Douglas	3	2	5	1	4	1	10	3	3	4	14	15	65
Gilliam	-	-	-	3	-	-	-	-	3	-	-	2	8
Grant	-	-	-	-	-	-	1	-	-	-	-	-	1
Harney	-	-	-	-	-	-	-	-	-	-	-	-	0
Hood River	1	-	-	-	-	-	-	1	-	4	-	-	6
Jackson	-	-	1	1	4	-	2	-	2	5	6	3	24
Jefferson	1	-	-	-	-	-	-	-	-	-	-	-	1
Josephine	-	-	-	2	1	-	6	1	1	12	2	8	33
Klamath	-	2	2	-	-	-	-	-	3	3	-	-	10
Lake	-	1	1	2	-	-	-	-	-	-	-	-	4
Lane	19	11	4	2	13	7	2	-	13	13	18	8	110
Lincoln	1	2	1	2	-	1	1	-	-	-	-	-	8
Linn	8	7	12	8	4	10	9	9	12	15	24	13	131
Malheur	-	-	-	-	-	-	-	-	-	7	1	-	8
Marion	7	6	6	6	6	1	11	-	4	17	21	-	85
Morrow	5	-	-	-	-	-	-	-	-	-	-	-	5
Multnomah	3	4	1	1	5	-	3	2	9	11	21	4	64
Polk	2	2	4	-	3	1	-	1	2	7	3	5	30
Sherman	2	-	-	-	-	-	-	2	-	1	1	-	6
Tillamook	-	3	2	-	-	-	-	-	1	-	-	-	6
Umatilla	3	-	-	-	5	2	1	-	1	-	-	2	14
Union	-	-	1	-	1	2	-	-	-	-	3	2	7
Wallowa	-	-	-	-	-	-	-	-	-	-	-	-	0
Wasco	-	-	2	2	-	-	-	-	-	-	-	-	4
Washington	3	5	9	2	-	-	6	-	-	6	29	10	70
Wheeler	-	-	-	-	-	-	-	-	-	-	-	-	0
Yamhill	1	-	4	3	2	6	1	5	2	11	6	10	51
	90	56	75	49	71	42	80	50	84	163	222	97	1080

TABLE IV. MONTHLY REPORT OF DISEASES FOR 1932-1933

a. CHICKEN (Four wks. old or older)	July	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	June	Total
Coccidiosis													
mature birds	3	2	5	2	11	6	9	8	5	-	11	6	68
immature birds	18	7	11	3	3	1	-	-	-	1	5	5	54
Tapeworms	1	7	4	4	8	1	2	2	2	2	1	-	34
Capillaria	-	-	-	-	-	-	-	-	2	4	1	-	7
Ascarids	-	2	-	1	1	-	-	-	-	-	-	-	4
Lice	2	4	8	5	2	1	1	-	3	6	5	-	37
Scaly leg	-	-	-	-	-	-	-	-	1	-	-	-	1
Paralysis and blindness	5	3	12	5	5	4	7	6	6	2	7	6	68
Fowl-pox	-	1	7	2	17	3	2	3	-	6	-	-	41
Tuberculosis	2	-	-	1	-	-	2	-	3	5	2	2	17
Colds and roup	-	2	-	-	-	1	5	-	-	1	-	2	11
Pullorum disease	2	-	3	1	1	1	3	7	-	1	2	2	23
Aspergillosis	-	-	-	1	-	-	-	-	-	-	-	-	1
Unsatisfactory	-	-	1	-	-	1	-	-	-	-	1	-	3
No diagnosis	3	-	1	2	3	-	4	4	-	1	3	7	27
Leucemia	-	-	2	1	3	-	1	3	5	2	-	-	17
Tumors	-	5	1	2	2	2	9	3	3	-	1	-	28
Egg concretions	2	3	3	1	1	1	1	-	1	1	5	-	19
Peritonitis	-	-	-	-	1	2	-	2	2	-	1	2	10
Bumblefoot	1	2	-	-	-	1	-	-	-	1	-	-	5
Vent infection	-	-	1	-	-	-	2	1	-	-	-	1	5
Necrotic enteritis	-	-	-	-	-	-	-	-	-	-	-	2	2
Canker	1	-	-	-	-	-	-	-	-	-	-	-	1
Botulism	-	-	-	2	-	-	-	-	2	2	-	-	6
Rickets	-	-	-	-	-	-	-	2	2	-	2	-	6
Demineralization	1	2	-	-	-	-	-	-	-	-	2	-	5
Perosis	-	-	1	2	-	-	-	-	-	4	-	-	7
Avitaminosis A	-	-	-	-	-	-	-	-	1	-	2	-	3
Visceral gout	-	-	-	-	-	1	-	-	2	-	-	-	3
Osteoporosis	-	-	-	-	-	-	1	-	-	-	-	-	1
Wounds, lacerations	1	-	-	1	1	-	-	-	-	-	-	-	3
Fractures	-	-	-	-	-	-	1	-	1	-	-	-	2
Impactions	-	-	1	-	-	-	-	-	-	-	1	-	2
Jaundice	1	-	-	-	-	-	1	-	-	-	-	1	3
Poisoning	1	-	-	-	-	-	-	-	-	-	-	-	1
Hemorrhage	-	1	-	-	1	-	-	-	-	-	-	-	2
Prolapse	-	-	1	2	-	-	-	-	-	-	-	-	3
Diverticulum	-	-	-	-	-	1	-	-	-	-	-	-	1
	44	41	62	38	59	27	51	41	41	39	52	36	531

TABLE IV (cont). MONTHLY REPORT OF DISEASES FOR 1932-1933

b. CHICK (Under four weeks of age)	July	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	June	Total
Pullorum disease	3	4	1	-	-	2	21	6	39	84	87	17	268
Negative for pullorum disease	2	-	-	-	-	-	4	-	2	23	20	-	51
No diagnosis	-	-	5	-	-	-	-	-	-	8	21	-	34
Unsatisfactory	-	-	-	-	-	-	-	-	2	3	5	3	13
Omphalitis	-	-	-	-	-	-	-	-	-	6	-	-	6
	5	4	6	0	0	6	25	6	43	124	133	20	372
c. TURKEY (All ages)													
Entero-hepatitis	6	1	-	2	4	4	1	-	-	-	2	5	25
Necrotic enteritis	2	3	-	-	-	-	-	-	-	-	-	8	13
Tuberculosis	1	-	-	-	-	1	-	-	-	-	-	-	2
No diagnosis	2	2	5	-	-	-	-	-	-	-	5	5	20
Unsatisfactory	4	-	-	-	-	-	-	-	-	-	4	3	11
Fowl-pox	-	-	-	-	-	1	1	-	-	-	-	-	2
Aspergillosis	2	-	-	1	-	-	-	-	-	-	2	1	6
Colds and roup	2	-	-	2	1	-	-	-	-	-	-	-	5
Tapeworms	-	1	-	-	-	-	-	-	-	-	-	1	2
Botulism	2	-	-	-	-	-	-	-	-	-	-	-	2
Omphalitis	-	-	-	-	-	-	-	-	-	-	4	2	6
Catarrhal enteritis	-	2	-	-	-	-	-	-	-	-	-	-	2
Hemorrhagic enteritis	4	2	-	-	-	-	-	-	-	-	-	-	6
Perosis	3	-	-	-	-	-	1	-	-	-	-	2	6
Rickets	-	-	-	-	-	-	-	-	-	-	5	3	8
Foreign bodies	1	-	-	-	-	-	-	-	-	-	-	-	1
Impaction (gizzard)	7	-	-	1	-	-	-	-	-	-	3	-	11
Ascites	-	-	-	-	-	-	-	-	-	-	5	-	5
Coccidiosis	-	-	-	-	-	-	-	-	-	-	4	10	14
Lice	2	-	1	-	-	-	-	-	-	-	-	-	3
Fluke	1	-	-	-	-	-	-	-	-	-	-	-	1
Canker	-	-	-	-	-	-	-	-	-	-	2	1	3
Abscess	-	-	1	1	-	-	-	-	-	-	-	-	2
Visceral gout	-	-	-	4	1	-	-	-	-	-	-	-	5
Fowl cholera	2	-	-	-	-	-	-	-	-	-	-	-	2
	41	11	7	11	6	6	3	1	0	0	36	41	163
d. PEAFOWL													
No diagnosis	-	-	-	-	-	-	-	1	-	-	-	-	1
e. CANARY													
No diagnosis	1	-	-	-	-	-	-	-	-	-	-	-	1
f. SPARROWS													
Unsatisfactory	-	-	-	-	3	-	-	-	-	-	-	-	3
g. PARAKEET													
Tumor (ovarian)	-	-	-	-	-	-	-	1	-	-	-	-	1
h. MOUNTAIN QUAIL													
Necrotic enteritis	-	-	-	-	3	-	-	-	-	-	-	-	3
Lice	-	-	-	-	-	-	1	-	-	-	-	-	1
No diagnosis	-	-	-	-	-	2	-	-	-	-	-	-	2
Unsatisfactory	-	-	-	-	-	-	-	-	-	-	1	-	1
i. RABBITS													
Coccidiosis	-	-	-	-	-	1	-	-	-	-	-	-	1
	1	0	0	0	6	3	1	2	0	0	1	0	14



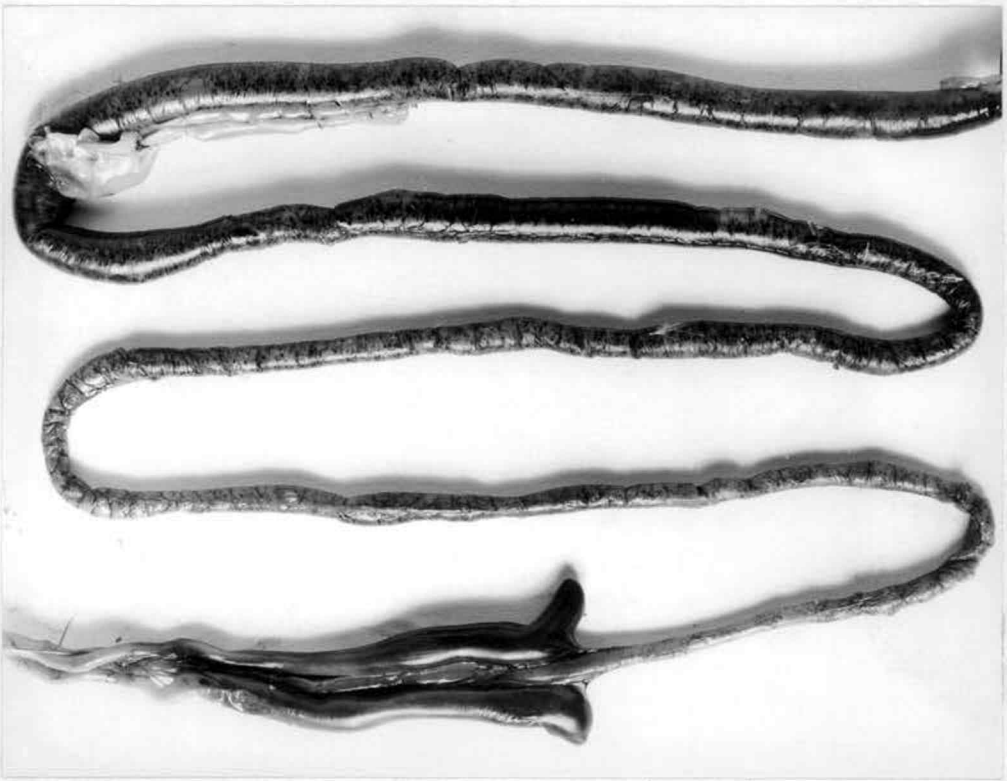
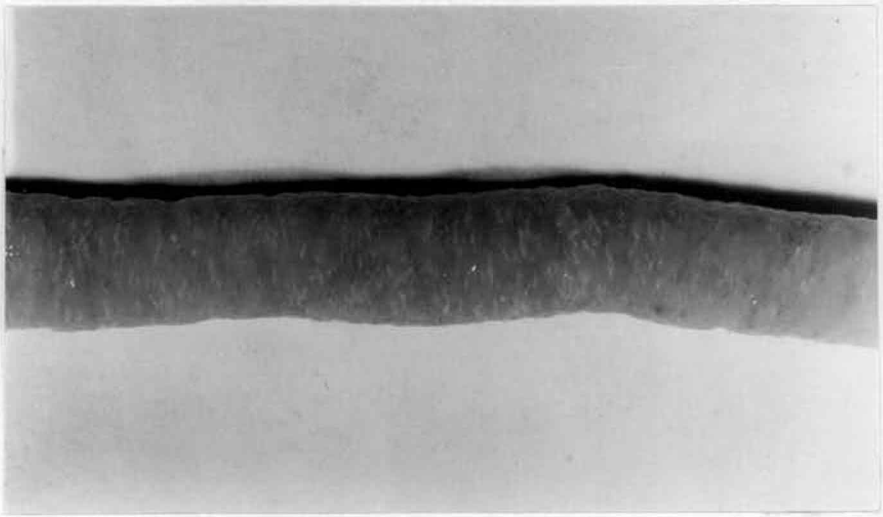
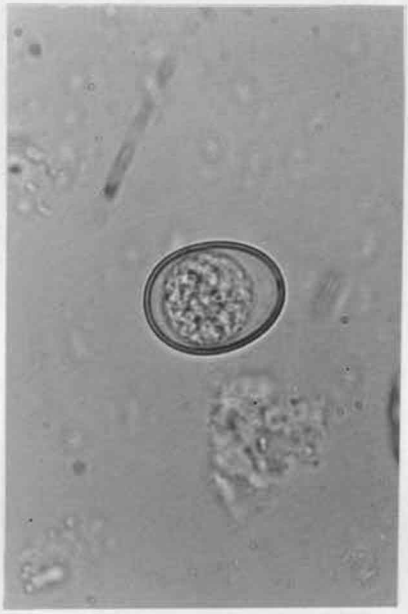


Figure 1. (Above) Eimeria tenella infection in the caeca (A) of a chick, showing hemorrhages to the serous surface and distention. Compare with the caeca (B) of a normal chick.

Figure 2. (Below) Eimeria necatrix infection in the small intestine of a chicken, showing hemorrhages to the serous surface and distention of the upper part.





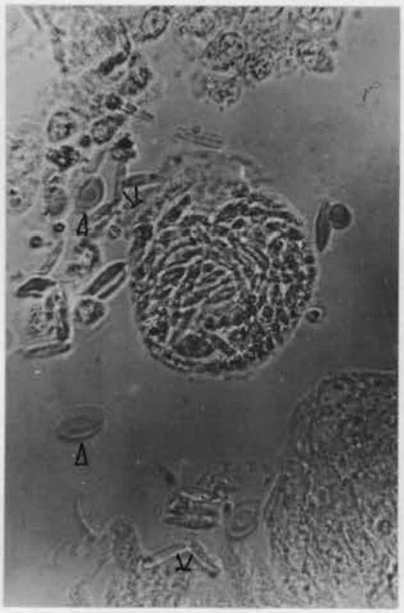


Figure 3. (Above) Mucous membrane of the duodenum of a chicken showing typical whitish patches due to Eimeria acervulina.

Figure 4. (Below left) Typical unsporulated oocyst (Eimeria acervulina). About 900 diameters.

Figure 5. (Below right) Typical large schizont (Eimeria tenella). Note the red blood cells (Δ) and free merozoites (⊻) also. About 450 diameters.

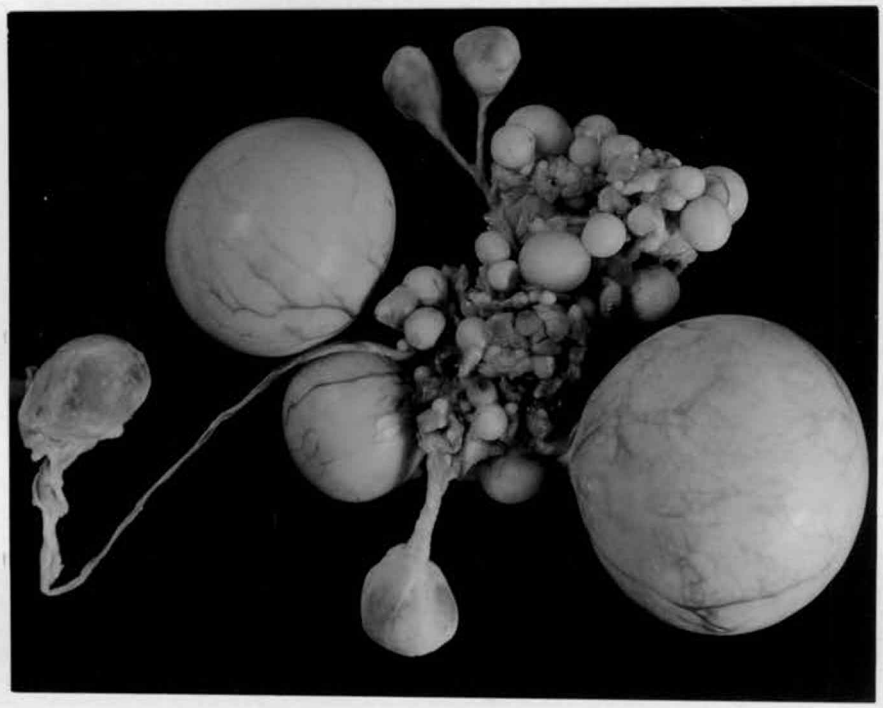
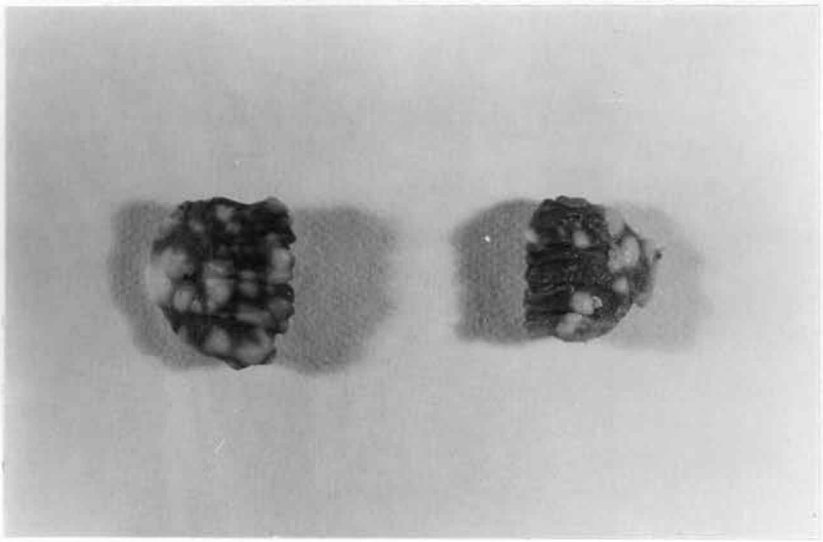


Figure 6. (Above) Pullorum disease in the ovary of a laying fowl.

Figure 7. (Below) The same ovary as Fig. 6, except all "yolks" typical of pullorum disease have been removed, leaving only normal appearing ones.



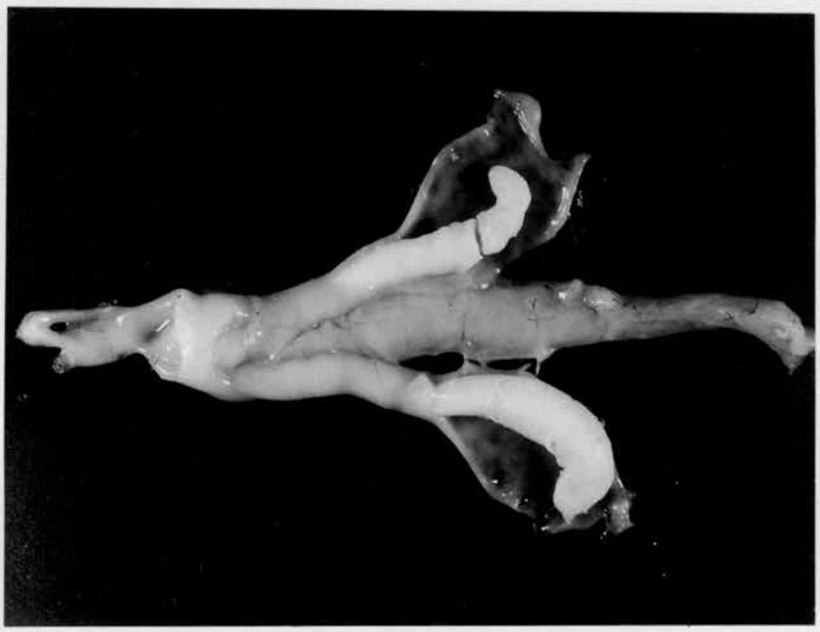


Figure 8. (Above) Lungs of a chick affected with pullorum disease, showing caseous nodules caused by the infection.

Figure 9. (Below) Caeca of a chick affected with pullorum disease, showing caeca cut open at the blind end exposing cheese-like cores.

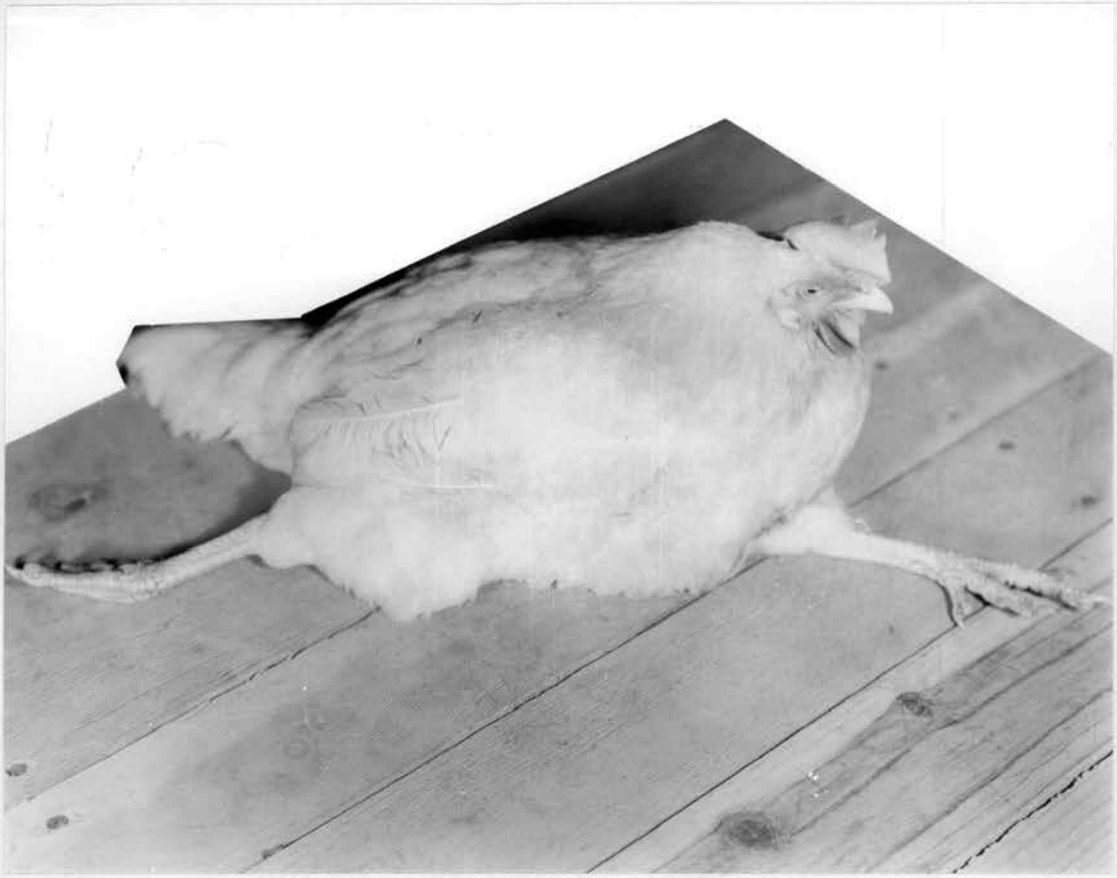


Figure 10. A nine-month-old pullet affected with paralysis. The posture is quite typical of this disease. The bird was in good flesh and had been laying. This bird also had a typical case of iritis.

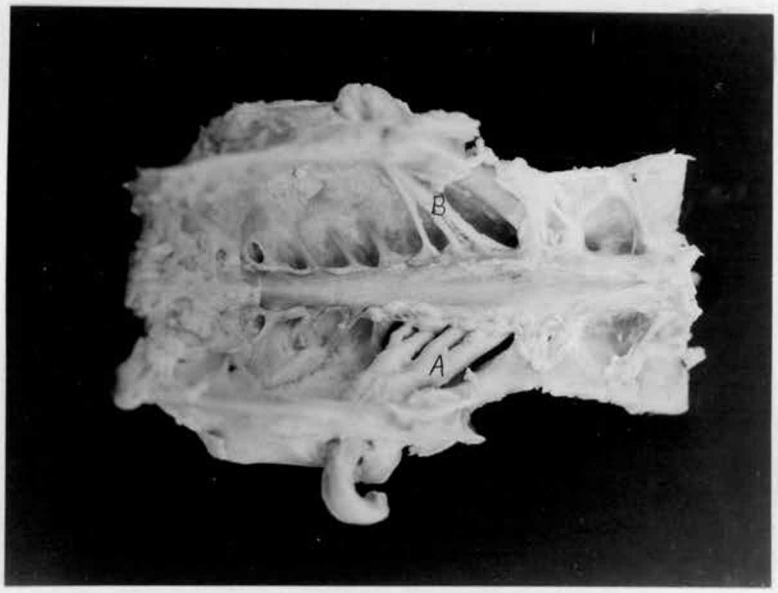
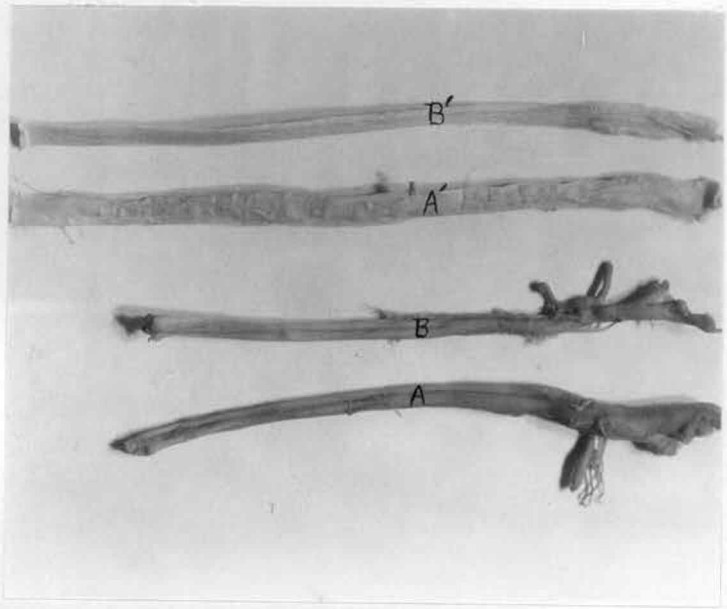


Figure 11. (Above) Typical case of iritis (blindness). Note the malformed pupil and the grayish iris. This is the head of the bird in figure 10.

Figure 12. (Below) Typical lesions of paralysis in the left lumbo-sacral plexus and the sciatic and femoral nerves. (A) abnormal nerve. (B) normal nerve.



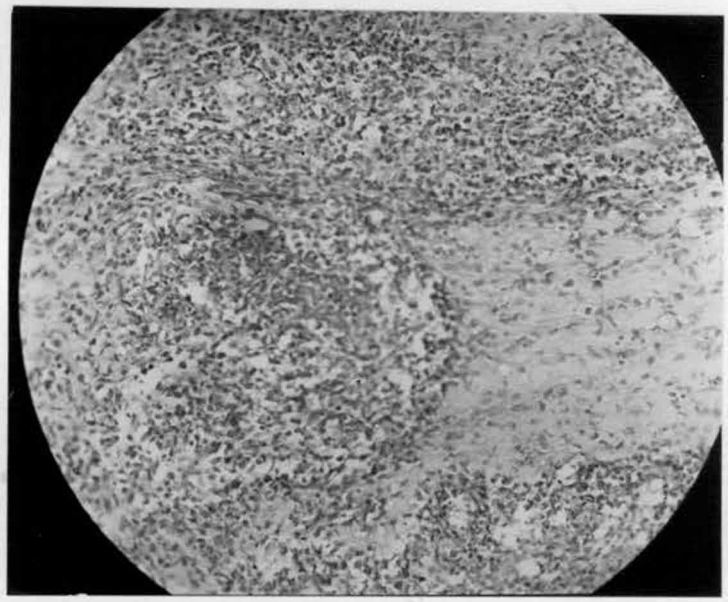
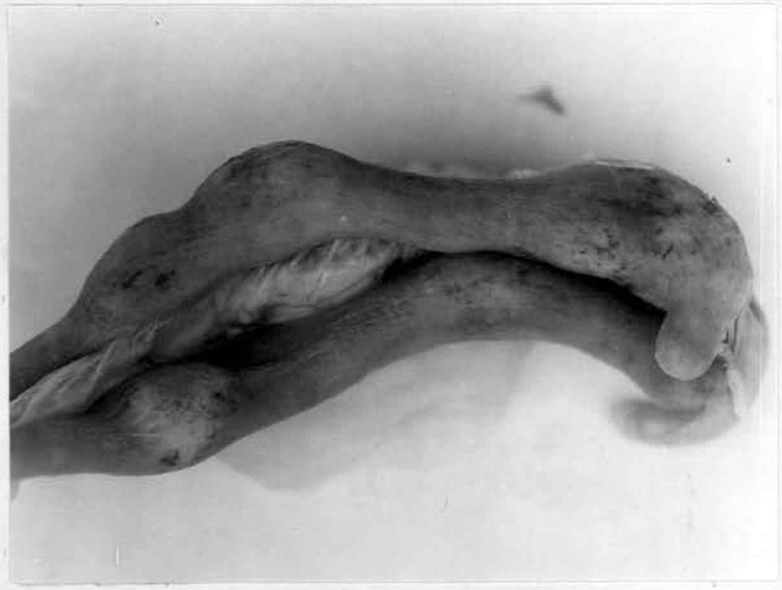


Figure 13. (Above) Lesions of fowl paralysis.
(A) Involving the nerve plexus.
(A') Involving the nerve trunk.
(B, B') Normal nerves from opposite legs.

Figure 14. (Below) Microphotograph showing cellular infiltration of nerve. 240 diameters.



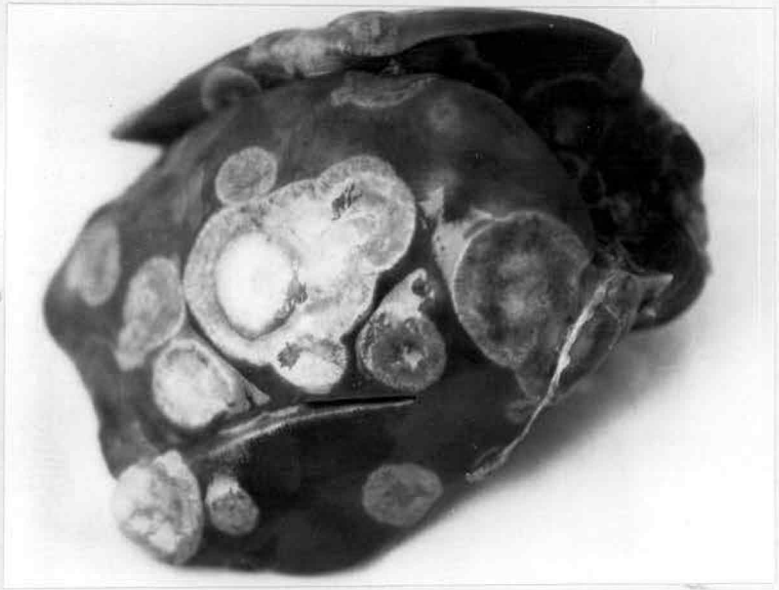
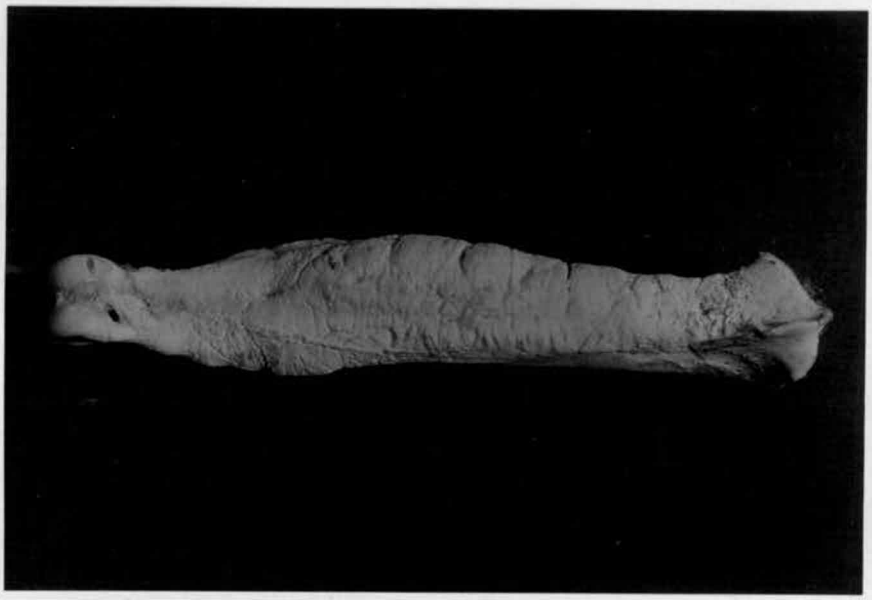


Figure 15. (Above) Typical lesions of infectious entero-hepatitis in the caeca of a turkey.

Figure 16. (Below) Typical lesions of infectious entero-hepatitis in the liver of a turkey.



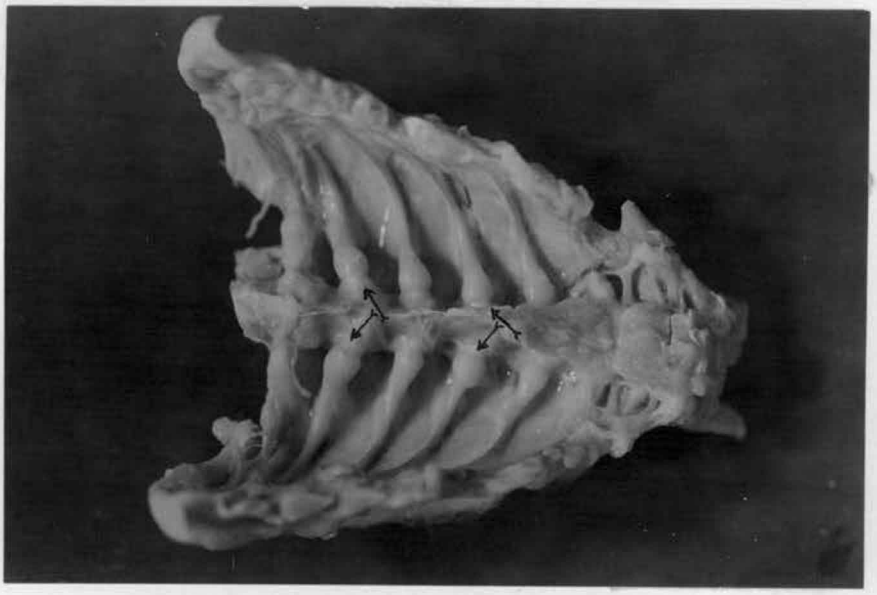


Figure 17. (Above) Tibia from a case of osteoporosis showing exostosis of the bone.

Figure 18. (Below) Thorax of six week old poult with sternum cut away and viscera removed. Arrows pointing to rachitic lesions (cartilaginous enlargements) in the costo-vertebral joints.

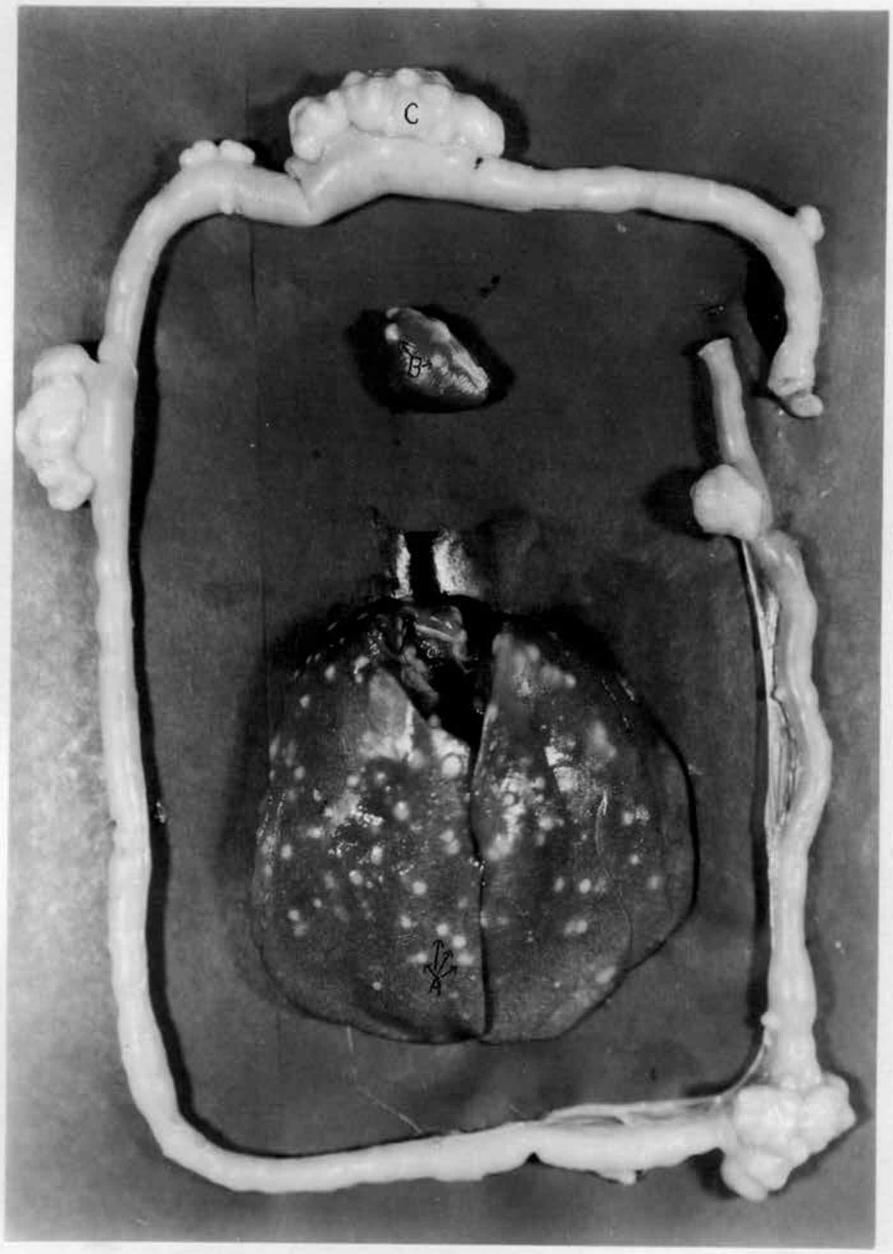


Figure 19. Typical lesions of tuberculosis in the liver (A), spleen (B), and small intestine (C).

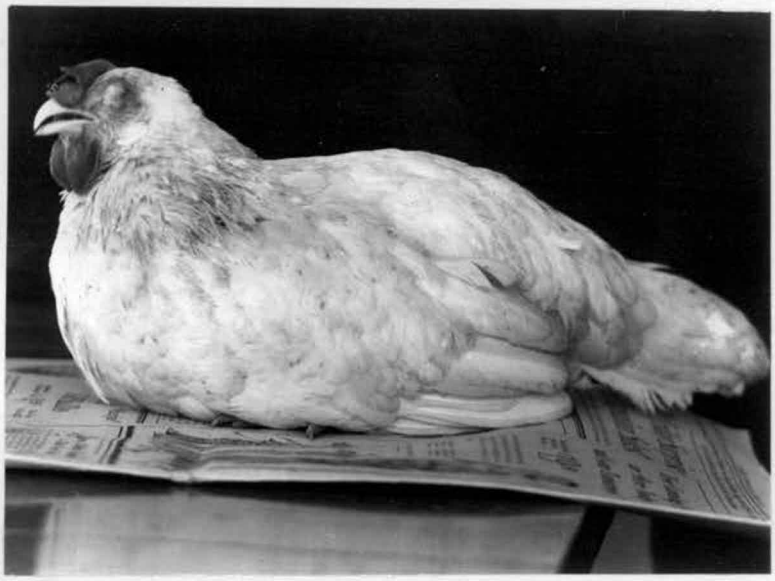


Figure 20. (Above) A case of avitaminosis A. Note the swollen eye containing cheesy-like exudate.

Figure 21. (Below) Oesophagus from the above case of avitaminosis A. Arrows point to some of the pustules in the oesophageal glands.

Figure 22. Chicken affected with botulism.
Note the "limberneck" condition although
the bird is still able to sit on the perch.

Figure 23. Liver from a bird with leucemia. The liver weighed 307 grams. Note the increased size by comparison with the scale. The areas of leucocytic infiltration are larger and more densely infiltered than one usually will find in the average case.

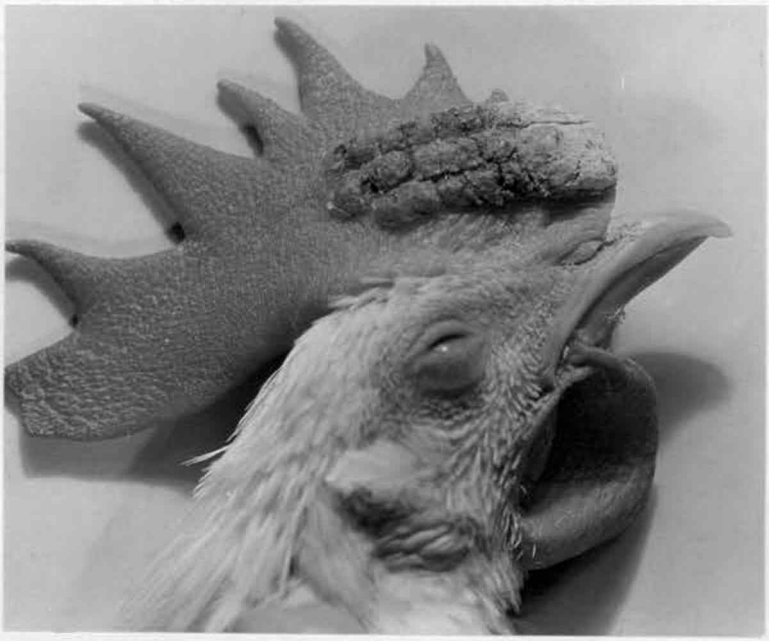


Figure 24. (Above) Experimental case of fowl-pox produced by inoculating virus into three needle scratches on the comb.

Figure 25. (Below) Natural case of fowl-pox.
(A) Canker occluding the larynx.
(B) Lesions on the comb.

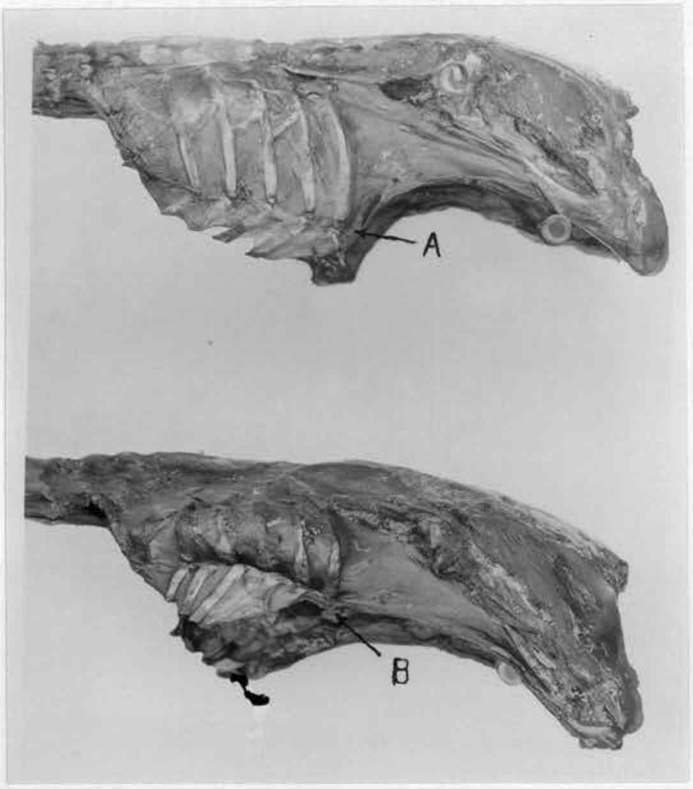


Figure 26. (Above) (A) showing normal vertebral-sternal joints. (B) showing buckling of ribs at the vertebral-sternal joints.

Figure 27. (Below) Chicken with demineralization showing legweakness.