STUDIES IN THE PATHOLOGY OF AVIAN COCCIDIOSIS

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Henrik Joakim Stafseth

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Studies in the Pathology of Avian Coccidiosis.

THESIS

Contents.

Studies in the Pathology of Avian Coccidiosis.

Introduction.

Coccidiosis in chickens is a disease which is steadily gaining in prevalence and importance. It is no longer considered to be a menace to young stock only, as we now know that fully grown birds are susceptible and may suffer rather severely from this malady. Hundreds of chickens, young and old, are sent to this laboratory annually to be examined for positive evidence as to the presence of coccidia in diseased birds. The history of the disease outbreaks, submitted with the various consignments, together with the gross and microscopic findings seemed to indicate that lesions, pathognomonic of coccidiosis, usually occur during the first two weeks or so of the disease but may later change to such an extent that it is difficult to make a diagnosis merely on the basis of pathological changes, i.e., when one is dealing with the disease as it occurs in the small intestines. Moreover, it was noted that chickens, sent in during the early stages of the disease, invariably showed numerous coccidia in the intestinal contents or scrapings from the mucous membrane, while birds, that had been sick long enough to show such symptoms as paralysis or extreme weakness often appeared to be entirely free from these parasites.

This study was undertaken in the hope that enough might be learned concerning the pathology of coccidiosis to enable us to diagnose this disease more exactly in its chronic forms.

Historical.

There is not a great deal of literature available covering the pathology of avian coccidiosis, excepting, of course, the usual descriptions of the gross lesions of the common form of the disease as it affects chicks. As most books and bulletins on poultry diseases treat this phase of coccidiosis quite well no further mention of it will be made here. The chronic form or the type that affects mostly the small intestines is less fully described and very few publications indeed contain anything concerning microscopic lesions.

Eckardt (1) states that in subacute or chronic cases the intestinal mucous membrane is reddened in spots, coated with tenacious mucus and as though dusted with flour. Such dustlike points occasionally occur also in the liver; they are then also due to coccidia. Hadley (2) describes quite fully what he calls subepithelial infection. He found "merozoite cysts" adjacent to the base of the crypts and in the core of the villi and some times they were packed in the cores to the exclusion of nearly all other cell structures. In such cases they crowded closely on the basement membrane and were often packed solidly along the

muscular wall or in the muscularis mucosae. Ward and Gallagher (3) have merely this to say about the anatomical changes in the small intestines: "The lining membrane is deeply congested or hemorrhagic". Hutyra and Marek (4) mention intestinal catarrh or enteritis with patchy desquamation of epithelium. They suggest that the sudden death may be due to secondary infection. Reinhardt (5), besides describing the anatomical changes occuring in the acute form, mentions the occurrence of diphtheritic membranes and small pinhead sized grayish foci in the mucous membrane of chickens affected with subacute or chronic coccidiosis. He says that those foci are nothing else but immense masses of macro- and microgametocytes in the epithelium of the gland tubules. Joest (6) found coccidia in the epithelium of the villi and the intestinal glands with desquamation of epithelium. In places the epithelium was completely lacking. The connective tissue of the propria was infiltrated with leucocytes. Otte's (7) description of the intestinal lesions of coccidiosis is very similar to those by Leckarut and Reinhardt. He states that in the liver of older birds one can notice light spots. Krijgsman (v. Heelsbergen) (8) describes the pathological anatomy of peracute, acute, subacute and chronic forms, mentioning very briefly some microscopic lesions. The changes noted do not differ from those already referred to. He mentions grayish white foci in the liver as being lesions of

coccidiosis but adds that it is not sure that those lesions have not been confused with those of blackhead. Tyzzer(9) has recently published his results of some very extensive studies on the nature of coccidiosis in gallinaceous birds. He describes four species occurring in chickens: 1. Eimeria tenella the cause of cecal, acute or hemorrhagic coccidiosis. Epithelial cells infected with this parasite become phagocytic and actively infiltrate underlying tissue hence the apparent sub-epithelial infection. This parasite may also infect the lower part of the small intestines. There is sloughing of mucous membrane five days after infection. There is more or less eosinophilia in the region of glands infected with first generation merozoites. He also finds lymphoid and plasma cell infiltration with increase of connective tissue in these areas. 2. Eimeria mitis (n.s.) occurs mostly in the upper portion of the small intestines, does not tend to occur in swarms but tends to penetrate beneath nucleus of infected cell. Little damage is done by this species. 3. Eimeria necatrix associated with chronic coccidiosis and perhaps the most common of the four species, found mostly in the upper part of the small intestine, tends to mass in limited areas producing gray spots in mucous membrane. This species merely penetrates the epithelial cell causing patchy desquamation of epithelium. Eimeria maxima may be distributed throughout the entire length of the small intestines, the greatest

numbers being found in the middle portion. The mucous membrane becomes swollen, covered with dirty grayish exudate and may show some hemorrhage. The pathological changes are associated with the sexual portion of the life cycle. The gametes are large and develop deep in tissues. The asexual forms are small and are situated superficially. He states that deficiency diseases may develop as a result of chronic coccidiosis. No evidence was found to show that coccidia produce lesions in the liver. Haupt's (10) description of the post mortem findings agrees with those of the other workers whose work has been reviewed.

Experimental

Source of material. One part of the material consisted of chicks, growing stock or adult birds sent to this laboratory for examination. The other part consisted of experimentally infected birds. Such birds were kept under observation for some time to see if they showed any evidence of being infected with coccidia. Their droppings were examined for the presence of oocysts and the general appearance of each bird was carefully noted. If symptoms of disease were observed or coccidia found in the droppings the bird concerned was not used.

Methods: The coccidia were sporulated in wide glass dishes on the bottom of which were placed two layers of filter paper. A two per cent solution of potassium dichromate was used to keep the "culture" moist. Prior

to feeding the "culture" to chickens it was either washed with 0.6 per cent lacl solution or it was diluted with water and grain mash. This was done either to remove the potassium dichromate or to dilute it to such an extent that it would not be likely to injure the sporozoites. This precaution may be entirely unnecessary as some birds that were fed cultures containing considerable amounts of the dichromate solution promptly developed coccidiosis. The "cultures" were fed either in grain mash or with 10 c.c. volumetric pipettes inserted into the esophagus. The "cultures" used were sufficiently rich in organisms so that every microscopic field (high power, dry) showed from one to five or six coccidia. If it was desired that reinfection should take place the birds were kept in cages with solid bottoms and no precaution was taken to keep the cages, feed and water clean. When reinfection was not desired wire bottoms were used in the cages and the feed and water dishes were placed so that contamination was not likely to take place.

Since I was mainly interested in studying chronic coccidiosis most of the birds were given several moderate doses or they were kept in contaminated pens or cages for several weeks if necessary.

The tissues taken for sectioning were fixed in Zenker's solution and stained with eosin and methylene

blue.

Symptoms.

In the summer and fall of 1924, 1925 and 1926 rather extensive outbreaks of leg weakness and paralysis occurred in a flock over which we had close supervision. The birds affected were kept on a piece of ground that had been used as a poultry range for many years. These birds showed rather heavy tape worm infestation (Davainea proglottina and cesticillus) together with coccidiosis. A portion of this flock, kept on a range lying on the side of a hill the soil of which, being of a sandy or even gravelly consistency, remained free from paralysis and coccidiosis. Until this fact was noticed the caretakers had thought the lameness and paralysis to be due to vitamin and mineral deficiency. That the feed could have nothing to do with this ailment was very evident since all birds in this flock received the same ration.

In order to study the possible relation between leg weakness (paralysis) and coccidiosis twelve normal birds (White Leghorns) were placed in a pen August 31, 1926 and fed sporulated coccidia August 31, September 1, 6, 17, October 10 and November 17th. On September 8th one cockerel was found to be very lame after having shown incoordination for two or three days. On October 4th this bird was completely paralyzed and was killed. The following is the autopsy record of this

bird:

Bird Number 108. Gross Lesions.

Liver: Several clusters of grayish spots and a few such foci scattered over the entire surface.

Intestines: Hemorrhagic enteritis with numerous grayish spots resembling minute necrotic foci also visible through serous membrane. Much slimy material throughout intestinal canal anterior to the ceca.

Ceca: Several petechial hemorrhages visible through serous coat. Contents are caseous, grayish to grayish brown and red in areas. The cheesy mass adhering to the walls of the ceca leaves a raw surface when removed.

Microscopic.

Serosa of mucous membrane: Innumerable small coccidia in various stages, oocysts and intracellular elements.

Sciatic nerves: Normal.

Intestines, sections: Several large foci of small coccidia in epithelium and subepithelial tissue. The coccidia have invaded the tissues down to the fundus of the crypts in some places. There are hemorrhages and sloughing of the mucous membrane.

Liver, sections: Numerous areas of perivascular infiltration with undifferentiated mesenchymal cells.

Nerves: Normal.

On October 28th, 1930 another cockerel was found

very emaciated and completely denuded. The autopsy record is as follows.

Bird No. 666, Gross Lesions.

Pericardium: Much serous fluid.

Spleen: Very small.

Gall Bladder: Distended.

Intestines: Numerous grayish spots in duodenal mucosa. There is some evidence of sloughing of the mucous membrane in some areas.

Ceca: Normal in appearance.

Nerves: Normal.

Microscopic.

Scrapings of mucous membrane: Innumerable very small coccidia.

Intestines, sections: Several extensive areas of epithelial and subepithelial infection with coccidia. There is marked destruction of epithelium and displacement of subepithelial tissues in these areas. Some villi seem completely broken down with only a little muscular tissue remaining. There seems to be no cell reaction in the infected areas.

Nerves: No cellular infiltration.

On October 23, 1926 a third cockerel was found in this pen showing the following symptoms; excitability, anemia, emaciation and some diarrhea (not bloody). This bird was killed and the autopsy record follows:

Bird No. 380. Gross Lesions.

Heart: Very flabby.

Liver: A few small gray spots.

Intestines: Pale with small grayish spots in duodenal mucosa. Several round worms.

Ceca: Normal.

Microscopic.

Scrapings from ceca and duodenum: A number of coccidia.

Liver: There is a moderate perivascular infiltration with undifferentiated mesenchymal cells. Some of the capillaries are injected.

Intestines: Marked areas of infiltration with undifferentiated mesenchymal cells in the stroma and muscularis mucosae. The epithelium is well preserved except over the tips of the villi where there appears to have been some mechanical destruction. There is congestion of the capillaries in the deeper portions of the mucous membrane. In some sections the greater portion of the villi is missing. This may be due to mechanical influences due to the handling of the tissues, but might also be due to a recent coccidiosis infection. No coccidia were recognized in the sections studied.

Nerves: Normal.

Later three more birds developed chronic coccidiosis showing anemia and emaciation. About the middle of November, 1926, the remaining six birds were moved to

Cecal and Duodenal Coccidiosis	
35	
With leg weakness or paralysis	Without leg weakness or paralysis
19	16
54.29	45.71

Table I.

Incidence of Leg Weakness or Paralysis in 326

Consignments of Coccidiosis Infected Chickens.

	Duodenal Coccidiosis			Cecal Coccidiosis		Cecal and Duodenal Coccidiosis	
Total No. of Consignments	113			174		35	
	With blindness	With leg weakness or paralysis	Without leg weakness or paralysis	With leg weakness or paralysis	Without leg weakness or paralysis	With leg weakness or paralysis	Without leg weakness or paralysis
No. of Consignments	4	80	33	5	169	19	16
Per Cent	3.54	70.80	29.20	2.87	97.13	54.29	45.71

a clean pen with concrete floor and a few days later another bird was found showing leg weakness. A post mortem examination revealed duodenal coccidiosis. None of the other birds developed any symptoms of disease and were disposed of late in November. A number of birds (more than fifty) were kept in three pens adjacent to the one used for this experiment for more than one year and not a single case of lameness or paralysis had developed among them. All these birds received the same kind of feed.

Following the publication of a preliminary report (11) on this experiment it was suggested to me that the lameness and paralysis observed in coccidiosis might be due to nothing but general weakness. However, further studies have shown that leg weakness seems to be associated more often with duodenal coccidiosis than with cecal coccidiosis. Therefore, since cecal coccidiosis is usually the more severe type one can hardly feel justified in attributing leg weakness or paralysis merely to general weakness. Table I shows the incidence of lameness or paralysis in 480 consignments of coccidiosis infected chickens (young and adults) sent in for examination over a period of three years.

While it is true that these data do not prove that duodenal coccidiosis is the cause of leg weakness and paralysis, they at least suggest a significant trend.

Paralysis has been observed in chicks as young as

three weeks. An outbreak of paralysis occured in a large number of seven week old chicks on May 21, 1937. The owner brought in nine paralyzed chicks, some White Leghorns, some Barred Rocks, for examination and the history as given by him was as follows: "Had done well until this week. Only a few pens affected. There is no bowel trouble. The first symptoms are dizziness and twisting of the neck. Some throw head right back. Toes are cramped in some of them. In others the first thing noted is that they are down completely paralyzed. Chicks ultimately die and twenty-five have died so far". The owner had examined several chicks and found no signs of cecal coccidiosis. All nine chicks showed severe catarrhal duodenitis and scrapings from the duodenal mucosa of eight of them revealed innumerable small coccidia. The ceca seemed normal. Coccidiosis control measures were suggested and to date no more serious trouble has been encountered.

On June 10, 1937 two pigeons and two chicks were brought to the laboratory from a nearby town. The history, as given by the owner, was as follows: "Leg weakness and diarrhea occur in some chicks and pigeons. The latter also loose control of wings. Several of the pigeons had become paralyzed without showing diarrhea. A few pigeons "went light", lost appetite and became emaciated. On post mortem examination one pigeon showed numerous small white spots in the liver. A mixed coccidiosis infection (small and large coccidia) was found in the chicks. In the pigeons there was duodenal catarrh and scrapings from the duodenal mucosa showed numerous

small coccidia similar to those found in the chicks.

Deficiency Disease Secondary to Coccidiosis.

Rickets has been observed in growing chickens affected with coccidiosis. Not infrequently such birds have received cod liver oil and lime in addition to a well balanced grain and mash ration. Some of them have also been out-doors and have had access to fresh food and sunlight. In a case report (12) on this subject it was suggested that the coccidiosis infection might so interfere with the function of the mucous membrane as to make it impossible for these chicks to utilize their food properly.

Coccidiosis in Sparrows.

On June 23, 1927 seven chicks, about six weeks old, were brought to the laboratory by a member of the college staff who was carrying on some nutrition experiments. These chicks were extremely pale and had acute, hemorrhagic cecal coccidiosis. The contents of the ceca in every instance consisted of liquid and partly hemolyzed blood. As these chicks were kept in clean quarters on the second floor of a college building the question was asked: "How do coccidia get up there, can it be that sparrows carry them from the poultry yards?" Cole and Hadley (13) and Mayer (14) have suggested that coccidia may be carried by sparrows. Smith and Smillie (15) found this not to be the case. In order to see what local conditions might reveal regarding this problem forty-nine sparrows were caught in the poultry yards of the college and examined for the presence of coccidiosis.

enty-t o or 44.0 per cent of them had coccidia of the genus Isospora. These coccidia were found in the posterior part of the intestines, the rectum and cloaca. Numerous white spots were found in the mucous membrane of the cloaca. These spots proved to be colonies of coccidia. None of the sparrows revealed any coccidia of the genus Eimeria. This would seem to indicate that if sparrows carry coccidia from one place to another they probably do it mechanically.

Relationship of Coccidiosis to Leg Weakness, Paralysis, Torticollis and Blindness.

In a further attempt to prove or disprove the relation between chronic coccidiosis and such manifestations as leg weakness, paralysis, torticollis and blindness a more or less systematic study was carried out on twenty-two birds showing one or more of these symptoms. The birds were nearly fully grown or adults obtained from different sources. Table II gives a summary of the results of these studies. Some of the most interesting ones of the case reports from this work follow:

Case 1003

Bartlett Red Bullet

Symptoms

Weak on both legs, especially on the right. Toes turned back under feet.

Gross Lesions

Enteritis, sciatic and peroneal nerves enlarged, heart flabby,

Table II.

Lesions observed in Twenty-two Chickens Showing Leg Weakness, Paralysis, Torticollis and Blindness

	Found in No. of Birds.	Per Cent
Brain		
Undifferentiated mesenchymal cell infiltration.	1	4.5
Ceca		
Cecum worms numerous	3	13.6
Inflammation	3	13.6
Large and small coccidia in contents	2	9.0
Medium sized coccidia in contents	3	13.6
Condition of bird		
Emaciation	5	22.7
Weak and listless	3	13.6
Droppings		
Chalky	1	4.5
Slightly bloody	1	4.5
Duodenum (lesions etc. limited to duodenum		
Catarrhal inflammation	17	77.27
Petechial hemorrhages	1	4.5
Small coccidia in scrapings	8	36.3
Small and large coccidia in scrapings	1	4.5
White spots in muc. membrane	5	22.7
Eyes		
Blind	1	4.5
Heart		
Flabby	7	31.8
Pericarditis (dry)	1	4.5
Petechial hemorrhages	2	9.0
Serous fluid in pericardium	2	9.0
Intestines (lesions in duodenum and other parts of int.)		
Total No. of birds in which coccidia were found	19	86.36
Coccidia, large	1	4.5
Coccidia, medium sized	14	63.6
Coccidia, small	9	40.8
Coccidia, large and small	1	4.5
Coccidia, medium sized and small	4	18.1
Coccidia in epithelium	7	31.8
Coccidia in subepithelial tissue	14	63.6
Coccidia in epithelium and subepithelial	4	18.1
Coccidia in exfoliated mucous membrane debris	1	4.5

Table II. Continued.

	Found in No. of Birds.	Per Cent
Epithelium intact with considerable subepithelial coccidial infection	1	4.5
Coccidiosis lesions, no coccidia	2	9.0
No coccidia, no lesions	1	4.5
Enteritis, extending throughout intestines	4	18.1
Enteritis, hemorrhagic throughout, diffuse	1	4.5
Enteritis, hemorrhagic throughout, petechial	1	4.5
Lesions of lymphoma cell infiltration	4	18.1
Undifferentiated mesenchymal cell infiltration	5	22.7
Round worms	1	4.5
Sloughing of epithelium	11	50.0
Sloughing of mucous membrane	11	50.0
Tapeworms	7	31.8
White spots, foci of coccidial infection	5	22.7
White spots and streaks	1	4.5
Legs		
Paralysis of both legs	7	31.8
Paralysis of right leg	2	9.0
Paralysis of left leg	1	4.5
Rickets	1	4.5
Weak in both legs	6	27.2
Leukemic lesions in:		
Kidneys and ovary	1	4.5
Muscles and ovary	1	4.5
Generalized	1	4.5
Liver		
Blackhead lesions	2	9.0
Fatty degeneration	2	9.0
Hemorrhage (fairly large subserous)	1	4.5
Hydropic degeneration	1	4.5
Undifferentiated mesenchymal cell infiltration around bile ducts	2	9.0
Perivascular undifferentiated mesenchymal cell inf.	5	22.7
Petechial hemorrhages	2	9.0
White spots	6	27.2

Table II. Continued.

	Found in No. of Birds.	Per cent
Neck		
Torticollis	4	18.1
Nerves		
Peroneal, enlarged	1	4.5
Sciatic, both enlarged	2	9.0
Sciatic, one enlarged	4	18.1
Undifferentiated mesenchymal cell infiltration	8	36.3
Proventriculus		
Small ulcers	1	4.5
Spinal cord		
Perivascular cell infiltration	8	36.3
Spleen		
Atrophied	2	9.0
Enlarged	2	9.0
Pericapillary undifferentiated mesenchymal cell infiltration	2	9.0
Reticulo endothelial cell infiltration	1	4.5

Microscopic Lesions

Nerves: Moderate undifferentiated mesenchymal cell infiltration of sciatic nerve, with almost complete degeneration of nerve fibres and replacement by connective tissues. One piece of nerve examined was normal.

Intestines: Marked infiltration with undifferentiated mesenchymal cells of mucous membrane and to a lesser degree of the muscular coats. In some sections there is decided eosinophile and plasma cell infiltration. Some sections reveal a number of coccidia imbedded in the mucous membrane which shows extensive sloughing.

Liver: Pericapillary infiltration with undifferentiated mesenchymal cells, (focal infection). Parenchyma in fairly good condition.

Spleen: Numerous foci of reticulo-endothelial cells surrounding capillaries.

Case 203 (Lo)

Light Brahma cockerel.

Symptoms

Completely paralysed on both legs, toes bent back under feet. Beak "B" shaped. Droppings slightly bloody. Marked emaciation.

Gross Lesions

Heart: Serous exudate in pericardium. Muscle very flabby.

Spleen: Very small.

Liver: Lesions suggest blackhead.

Intestines: Thickened with numerous specks and streaks of grayish color.

Ceca: Inflammation and many cecum worms.

Nerves: Normal

Microscopic

Sections show a few coccidia in endoneum.

Spinal chord: Perivascular undifferentiated mesenchymal cell infiltration.

Intestines: A number of medium sized coccidia in subepithelial tissues. Numerous small coccidia in epithelium. Extensive sloughing of mucous membrane.

Case 20% ('Bob')

Light brown Pullet.

Symptoms

Paralysis of right let, extreme emaciation and lassitude.

Gross Lesions

Heart: Flabby.

Liver: Numerous necrotic foci, resembling bluehead lesions.

Intestines: Duodenum slightly thickened.

Nerves: No changes.

Microscopic

Sections from intestines show numerous coccidia.

Sciatic Nerve: Marked undifferentiated mesenchymal cell infiltration mostly on one side of the nerve, degeneration of nerve fibres.

Intestines: Numerous medium sized coccidia in subepithelial tissues and innumerable small coccidia in epithelium, which shows extensive sloughing. The small coccidia are found in foci, the larger ones are more

evenly distributed in this case. Many coccidia are situated very deeply in the mucous membrane.

Brain: Some perivascular undifferentiated mesenchymal cell infiltration.

Case 501

Rhode Island Red Cockerel.

Symptoms

No symptoms recorded, brought in with a number of birds showing lameness or paralysis. The bird was in fair condition and very active.

Macroscopic Lesions

Intestines: Considerable inflammation.

Microscopic

Scrapings from intestines showed innumerable coccidia in duodenum.

Duodenum (sections): Numerous foci of coccidial invasion of epithelium, lamina propria and subepithelial tissues. Some of these foci are large. In some places the coccidia have caused a wide separation of the epithelium from the subepithelial portion of the mucous membrane. In other places the epithelium is completely destroyed leaving ragged portions of the villi. All these coccidia are of the very small type.

Case 502

White Leghorn hen, Hartford.

Symptoms

Emaciation, cyanotic comb.

Gross Lesions

Liver: Numerous small grayish spots of the size of a pinhead.

Heart: A few petechial hemorrhages.

Intestines: Numerous tapeworms (D. caesticillus), round and cecum worms. No record of coccidiosis lesions or coccidia in intestines.

Microscopic

No record of coccidia in scrapings.

Intestines, sections: A considerable number of small and a few medium sized coccidia in subepithelial tissue. Epithelium is broken up as if by mechanical means. In one section there is a very large focus of coccidia covering an area too large to be seen at one time with the high power objective (Zeiss 6 Leitz), Figure I. This focus is located deeply in the subepithelial tissue, in the middle portion of a villus. The tissues surrounding this focus seem not to have been injured. In the area of the focus, however, the coccidia have completely replaced the normal tissue. A few areas, nearly as large as the focus just mentioned, are found in other villi, in which there are no coccidia but there are open spaces partly filled with tissue debris suggesting that this may be the result of focal coccidiosis infection, the coccidia having disappeared. The location of these areas correspond exactly to that of the colonies of coccidia. Near some of these areas

coccidia, which seemed to be in the gametocyte stage, were found.

Liver: Numerous areas of perivascular undifferentiated mesenchymal cell infiltration, Figure XII. These are fairly well distributed throughout the liver. Near the posterior border of one lobe, immediately under the surface are conglomerations of numerous small areas of this sort. These areas are often observed in chickens. Bacterial cultures from livers showing such lesions are usually negative. Escherichia coli and occasionally Salmonella pullorum have been isolated.

Case 271

White Leghorn.

Symptoms

Paralysis.

Gross Lesions

Sciatic nerve of left leg enlarged, slight enlargement of nerve in right leg.

Microscopic

No record of coccidia or parasites in intestinal contents.

Nerves, sections: Moderate diffuse and focal undifferentiated mesenchymal cell infiltration. A few of these cells are also found throughout the sections. There is marked degeneration of the nerve fibres, medulary substance and the axis cylinder. The picture corresponds well with the description of Papenheimer (16) (17). In areas there is wide separation of nerve fibres, partial

and complete collapse of myelin sheaths and vacuolization.

Intestines: Considerable breaking down of mucous membrane. In one such area was found one schizont. The epithelium in this area is intact but the subepithelial tissue is broken up as if by mechanical means.

Case 260

White Leghorn.

Symptoms

Leg weakness.

Biological Tests

Tuberculin test negative.

Agglutination test for pullorum disease negative.

Gross Lesions

Sciatic nerve of both legs enlarged.

Proventriculus: Small ulcers.

Duodenum: Small hemorrhages and what seem to be small necrotic foci in mucous membrane. Lavaliea costicillus found in large numbers.

Microscopic

Considerable number of coccidia in intestinal contents.

Pancreas: Congested capillaries.

Intestines, duodenum: Mucous membrane very ragged. Most villi have no epithelial covering over the free end and in many places the greater portion of the villus is denuded. In some places the epithelium has a "coagulated" appearance. There are large foci of

coccidia of the intermediate or small size. They are in and beneath the epithelium. In one villus the tissues surrounding the largest focus of coccidia are only partly destroyed. The epithelium seems merely to have been pushed out from the stroma. On the other side of the villus the tissues are very ragged, the epithelium is almost completely gone and here only a few coccidia are found. This may represent the oldest focus of the two.

Sciatic Nerve: Extreme, diffuse undifferentiated mesenchymal cell infiltration. There are so many of these cells that it is impossible to detect normal nerve fibres. There are congestion of the capillaries and, in a few places, hemorrhages.

Case 274

White Leghorn.

Symptoms

Leg weakness.

Biological Tests

Tuberculin test negative.

Agglutination test for bacillary white diarrhea negative.

Gross Lesions

Liver: Very pale with a few grayish specks near the border.

Intestines: A few small grayish spots in mucous membrane also a few small hemorrhages.

Microscopic

Scrapings showed no coccidia in this bird but from

another one from the same source coccidia were obtained. There were some small tapeworms.

Liver, sections: There is moderate perivascular undifferentiated mesenchymal cell infiltration, extreme hydropic degeneration and retention of embryonic characteristics (gland like tubules).

Intestines: Coccidia in considerable numbers have penetrated down to the muscularis mucosae. The mucous membrane is broken up in the area surrounding the coccidial focus. This has given rise to a hemorrhage. One large focus of coccidia was found in the inter-glandular tissue. There is the usual destruction of superficial parts of the mucous membrane. In one section showing a focus of coccidia near the muscularis mucosae the latter is broken up and there is marked undifferentiated mesenchymal cell infiltration in and around this focus, Figures III and IV.

Nerves: Very slight if any undifferentiated mesenchymal cell infiltration of the nerves. There seems to be some collapse of some of the myelin sheaths and there is also some marked vacuolization. The fibres are spread apart in some areas and in others they have disappeared.

The data obtained from the study of these lives show that nineteen of the twenty-two or 86.36 per cent

of them harbored coccidia. Enteritis was found in all but one bird. Duodenitis with no appreciable inflammation of the other parts of the intestines was found in seventeen of the birds or 77.27 per cent. These data are in themselves not convincing one way or another, because of the wide prevalence of coccidia in poultry plants.

Whether the coccidium per se may cause leg weakness, paralysis, incoordination, torticollis and blindness, or the destruction of the intestinal epithelium merely opens up an avenue of entrance for other pathogens or whether toxic substances, taken into the system through denuded mucous membranes may exert the pathogenic influence observed remains to be proved.

Immune birds or coccidia of low virulence.

On August 1, 1933 twenty-five normal fully grown young White Leghorn chickens were fed sporulated coccidia. The "culture" used consisted mostly of what was termed medium sized coccidia. This experiment was undertaken for the purpose of showing, if possible, progressive lesions and repair processes. Two birds were killed and autopsied at 2 to 4 day intervals as shown in Table III. This table also shows the gross and microscopic lesions found as well as the occurrence of coccidia.

The irregular occurrence of coccidia in these chickens makes it very difficult to say whether the lesions observed in the various organs were due to

Table III.

Lesions Observed in 25 Birds Given a Single Dose of Coccidia

Coccidia Fed to All Birds August 1, 1928.

No. of Bird	Killed and Autopsied	Symptoms	Gross Pathology: Heart	Intestines	Liver	Sciatic Nerves	Micropathology: Heart	Intestines	Liver
1	Aug. 4, 1928	Pale	Fl.	1	Y.	Normal	F.D.	N/C I	F.
2	" 4 "	Pale	N.	0	P.H.	"	N.	I	P. I.
3	" 6 "	Normal	Fl.	2	Sl.Y.	"	N.	C.f.s.	0
4	" 6 "	Pale, weak	N.	3	Sl.Y.	"	N.	0	0
5	" 6 "	Died	N.	P.M.	Y.	"	Not. Sec.	0	Not. Sec.
6	" 8 "	Pale, emaciated	Fl.	3	W.S.	"	D.	I.M.	P.I.
7	" 8 "	Normal	Fl.	3	Y.	"	D.	0	0
8	" 10 "	Normal	Fl.	4	2 W.S.	"	F.D.	I.M.	P.I.
9	" 10 "	Normal	Fl.	2	0.	"	D.	I.Sch.	P.I.
10	" 13 "	Normal	Fl.	2	0.	"	D.	I.	P.I.
11	" 13 "	Normal	Fl.	2	0.	"	D.	I.M.C.i.E.	P.I.
12	" 15 "	Normal	Fl.	5	2 S.W.S.	"	N.	0	P.I.
13	" 15 "	Pale	N.	6	0.	"	N.	H.f.	0
14	" 18 "	Emaciated Lame, right leg	E.Fl.	3	Con.M.	"	D.	I.M. N/C	P.I.
15	" 18 "	Normal	N.	3	0.	"	D.H.	I.	P.I.M.
16	" 21 "	Emaciated, lame	Fl.	0	4 W.S. 2 P.H.	"	D.	0	N.A.
17	" 21 "	Normal	N.	0	W.S.P.H.	"	N.	Cap.	P.I.
18	" 21 "	Died	N.	5	Y.	"	N.	C.f.	P.I.
19	" 24 "	Slightly emaciated but active.	Fl.	0	0	"	N.	C.i.l.	0
20	" 24 "	Slightly emaciated but active	E.	H	S. H. A.	"	D.	I.H.	P.I.
21	" 28 "	Normal	N.	0	0	"	N.	0	P.I. Cong.
22	" 28 "	Normal	S. Fl.	0	D	"	N.	0	P.I. Cong.
23	" 31 "	Emaciated	N.	0	A. D.	"	N.	0	0
24	" 31 "	Normal	N.	0	0	"	N.	Act.	P.I.
25	Sept.4, "	Normal	Fl.	0	0	"	D.	C.i.E.N.	P.I. Cong.

Code to Gross Anatomical Changes in Heart:

Fl. = flabby

N. = normal

E. = enlarged

S. = small

Code to Gross Lesions in Intestines:

0. = no lesions

1 = catarrhal inflammation

2 = catarrhal inflammation with pinhead grayish foci in mucous membrane.

3 = same as 2 with petechial hemorrhages

4 = same as 3 with tape worms

5 = same as 1 with more or less diffuse scattered hemorrhagic areas

P. M. = post mortem changes

6 = same as 5 with several round worms

H. = hemorrhagic area, otherwise normal

Code to Gross Lesions in Liver:

Y. = yellow

P. H. = petechial hemorrhages

Sl. Y. = slightly yellowish

W. S. = white spots or foci

0. = no changes

S. W. S. = small white spots, the numbers indicate number of foci or spots found

Con. H. = congested and mottled

S. H. ... = small somewhat diffuse hemorrhagic

areas

D. = dark

A. D. = atrophied and dark

Code to Microscopic Changes in Heart:

F. D. = distinct fatty degeneration with loose musculature

N. = normal

Not. Sec. = not sectioned

D. = changes suggest fatty degeneration, musculature seems somewhat loose

H. = hemorrhage

Code to Microscopic Findings in Intestines:

I/C = coccidia below nuclei of epithelial cells

I. = infiltration with undifferentiated mesenchymal cells

C. f. s. = coccidia few and scattered

O. = no coccidia, no lesions

I. L. = marked infiltration with undifferentiated mesenchymal cells

C. sch. = coccidia in schizont stage

C. i. E. = coccidia in epithelium

H. = hemorrhage

H. f. = few hemorrhages

Cap. = capillaria

C. f. = few coccidia

C. i. l. = coccidia in lumen

Act. = actinomycosis lesion

C. i. e. n. = numerous coccidia in epithelium

Code to Microscopic Lesions in Liver:

F. = fatty degeneration

P. I. = perivascular infiltration with undifferentiated mesenchymal cells

O. = no lesions

Not. sec. = not sectioned

P. I. e. = extensive perivascular infiltration with undifferentiated mesenchymal cells

N. = necrotic areas

Congr. = congestion

coccidiosis. The intestinal changes resembled very closely those found in birds suffering from typical attacks of this disease. It will be noticed from the table that coccidia were found on the 4th, 6th, 9th, 11th, 14th, 18th, 19th and 20th day after infection and, very unexpectedly, the heaviest infection was found in bird No. 23 which was autopsied 23 days after infection. All of these birds were kept in wire bottom cages (1/4 inch mesh) and the feed and drinking vessels were placed as high as possible without depriving the birds of a chance to eat and drink freely, in order to guard against contamination of water and feed with droppings. If the coccidiosis infection is truly selflimiting as claimed by Johnson (16) and by Fyzzer in the publication already referred to and as has been taken for granted by myself these birds must have picked up coccidia from the hardware cloth, thus becoming reinfected. Bird No.24 showed a simple lesion resembling unmistakably an actinomycosis lesion in the stroma mucosae of the small intestines.

The symptoms and lesions were not as pronounced as had been hoped for. Whether this was due to immunity on the part of the birds or to a lack of virulence on the part of the culture used is difficult to say. Johnson (16) and Fyzzer (9) have showed that chickens will develop immunity to coccidiosis. Fyzzer (9) has also showed that the different species of coccidia

vary in pathogenicity, the Eimeria mitis being practically nonpathogenic. At the time this work was done I had no knowledge of Lynch's work and while I felt certain of having observed at least three different kinds of coccidia, undoubtedly E. tenella, E. acervulina and E. maxima and perhaps also E. mitis, a coccidium which has been referred to in my notes as a strikingly round form, I had no information as to their immunological relationship. It may be of interest to note here that the birds used came from a flock where coccidiosis had been prevalent for some time. In 1924 and 1925 very severe losses were experienced in this flock due to so-called range paralysis. Since that time powdered milk feeding as recommended by Beach and Davis (19) together with other sanitary measures have been employed for the control of coccidiosis and for five years losses due to paralysis have been conspicuously absent.

This seems like an argument against Doyle's (20) suggestion to the effect that range paralysis is transmitted through the egg. It is also interesting to note that paralysis and leg weakness occurring in coccidiosis infected flocks are reported to have disappeared following the use of colloidal iodine. Some of the most interesting case reports from this experience follow:

Bird No. 1.

Fed coccidia August 1, 1923. Killed and autopsied August 4, 1923.

Symptoms

Pale and tailfeathered.

Gross Lesions.

Liver: Yellow, fatty.

Heart: Flabby.

Intestines: Moderate catarrhal enteritis.

Microscopic.

Liver: Fatty degeneration.

Heart: Some fatty degeneration, loose musculature.

Intestines: There are a few evenly scattered coccidia (one to three coccidia per villus). These are located just below the epithelium. The coccidia have either caused a separation of epithelium and underlying tissue or they have pressed on the epithelium so that the cells have become flattened and the subepithelial tissue has been pushed aside. A few schizonts seem to be located within greatly enlarged cells. While there is extensive infiltration with undifferentiated mesenchymal cells in the stroma mucosae and in a few noninfected villi there is no cellular infiltration around the infected areas. The coccidia seem more bunched near the tips of the villi.

Bird No. 11.

Fed coccidia August 1, 1928. Killed and autopsied August 18, 1928.

Symptoms.

Emaciated, lame on right leg.

Gross Lesions.

Liver: Congested and mottled.

Intestines: Small pin-point red and purplish foci.

Sciatic nerves: No visible changes.

Heart: Slightly enlarged and somewhat flabby.

Microscopic.

Liver: A few necrotic foci and some perivascular infiltration with undifferentiated mesenchymal cells.

Intestines: There are areas of infiltration with undifferentiated mesenchymal cells in the stroma mucosae. Some of these areas show no coccidia but others show a number of what appears to be sections in the middle of the infiltration areas and in others the middle portion consists of tissue debris numerous red blood cells (hemorrhage) and around the borders of the area are numerous sections, Figure VII. One section shows a number of coccidia in the upper part of the villi.

Bird No. 25.

Fed coccidia August 1, 1928. Killed and autopsied September 4, 1928.

Symptoms:

Appears normal.

Gross Lesions.

Heart: Somewhat flabby.

Liver: Small, otherwise normal in appearance.

Intestines: No visible pathological changes.

Sciatic nerves: Normal.

Microscopic.

Liver: There is congestion and some hemorrhagic areas.

There are also areas of infiltration with undifferentiated mesenchymal cells.

Intestines: There is desquamation of epithelium over the tips of the villi. In the epithelium of the tips of some villi and near the tips of others there are swarms of coccidia. They are located below the nuclei for the most part, Figure VI. A few are located superficially and others are seemingly in the subepithelial tissue. In this section one can recognize trophozoites, a few schizonts and many gametes. One schizont is very large.

On September 3, 1935 nine fully grown males coming from the same flock as the twenty-five birds used in the previous experiment, were fed sporulated coccidia. These males were kept in wire bottom cages. They were kept until September 27th in order to see if they would develop conspicuous symptoms. No such symptoms developed. A few of the birds discharged slightly bloody droppings on September 14th, 16th and slimy droppings September 18th. Two birds were killed September 17th, two October 2nd and on October 4th the remaining five birds were given another dose of sporulated coccidia. These were of the very large type. A slight amount of blood appeared in the droppings a few days afterwards and coccidia were found in the droppings but no other manifestations of disease appeared. Two of these birds were killed and autopsied October 11th and the remaining three on October 17th.

The only post mortem changes noted in these birds were: slight flabbiness of the heart in five of them and very mild catarrhal enteritis in all. Since the pathological changes were so insignificant no sections were made from these birds.

It may seem unwise to have used birds coming from coccidiosis infected stock for such experiments, but it must be remembered that coccidia are considered nearly ubiquitous and that until very recently such a thing as immunity to coccidiosis was not seriously thought of.

Coccidiosis in Chicks.

In the summer and fall of 1929 a study was made of gross and microscopic lesions found in twenty-five chicks showing various stages of coccidiosis. A description of the lesions found in chicks Nos. 10, 17, and 19 follows. The other chicks showed nothing of special interest that was not found in these three chicks.

Chick No. 10.

Autopsied July 6, 1929.

Symptoms.

Anemic and droopy.

Macroscopic lesions.

Scattered petechiae in small intestines.

Ceca: Distended with gas but no lesions of coccidiosis.

Microscopic Lesions.

Small intestines show considerable patchy desquamation of epithelium. There are considerable numbers of coccidia in and just below the epithelium of the villi. These coccidia are rather evenly scattered. The gland epithelium is entirely free from coccidia. Most of the coccidia are found near the tips of the villi only a few having reached down to within one-third to one-fifth of the distance from the bottom of the crypt to the free end of the villus. A considerable number of trophozoites were found varying considerably in size (11 to 14 microns in diameter). The smaller ones stain rather uniformly blue, while the larger ones show a rather distinct pinkish nucleus. Some of them are situated above and some below the nuclei of the epithelial cells. The schizonts varying in size from 26 x 28 microns to 31.4 x 32.9 microns were located below the epithelium. There is no cell reaction in the infected areas. In the stroma mucosae there is infiltration with undifferentiated mesenchymal cells and some indication of proliferation of glandular epithelium.

Chick No. 17.

Autopsied July 15, 1930.

Symptoms.

Too weak to stand up.

Macroscopic changes.

Typical coccidiosis lesions in ceca.

Microscopic changes in small intestines.

Many macrogametocytes staining red and several colonies of schizonts staining bluish were found in subepithelial tissues toward the base of the villi. The schizonts vary considerably in size and shape. Some look almost round others are ovoid, one is 21.4 x 27.1, one is 27.1 x 28.4 microns and another 21.4 x 21.4. In another portion of the same section there is a large colony of schizonts (stained bluish). They are in the stroma mucosae, figure VII. This lesion is too large to be seen at one time using a No. 6 objective (Leitz). There is marked infiltration with undifferentiated mesenchymal cells, considerable hemorrhage and gland tubules are either destroyed or displaced. The schizonts, twenty-six in all, are situated around the periphery of the lesion. In an almost adjacent area of similar size and outline, also in the stroma mucosae, there is one mature schizont in the neighborhood of which are three open vacuoles of the size of this schizont and several smaller ones indicating that they represent spaces out of which schizonts have disappeared. Several merozoites about 17 x 28 microns long are found in this lesion together with much tissue debris and large numbers of lysis cells. The cell infiltration extends through the muscularis mucosae and at one point, at which the fibres of the muscularis mucosae are completely destroyed, for a considerable distance into the muscular coat. Only a few plasma and

undifferentiated mesenchymal cells and small connective tissue fibres occupy this otherwise open space. Oocysts are found here and there throughout the villi in the subepithelial tissues and in other places open spaces as large or smaller than oocysts are found. These probably represent spaces out of which the cysts have passed. In many places the epithelium shows similar vacuoles and in these places the epithelium seems to be closing in from the sides.

Ceca: There is extensive sloughing of mucous membrane, the villous portion being almost entirely destroyed. Many places show oocyst in the lumen, Figure X (a). In the gland epithelium there are a few trophozoites and numerous vacuoles which very likely represent spaces evacuated by oocysts. Numerous oocysts are found in the interglandular tissues where there is also marked infiltration with undifferentiated mesenchymal cells in many quite extensive areas. In some of these areas there are also hemorrhages. The contents consist of tissue debris, necrotic material and innumerable oocysts.

Bird No. 19.

Autopsied July 19, 1929.

Symptoms.

Droopy and slightly cyanotic. Legs seemed stiffened.

Macroscopic Lesions.

Typical coccidiosis lesions in ceca.

Microscopic changes.

Liver: Areas of perivascular infiltration with

undifferentiated mesenchymal cells. Congestion.

Ceca: The epithelium of the villi shows large swarms of developing forms of coccidia, Figure V. They are so numerous that they have caused extensive desquamation of epithelium. Almost every gland tubule shows immense numbers of coccidia in the epithelium, Figure VI, mostly trophozoites and schizonts. In the interglandular tissues are large and smaller schizonts and numerous oocysts, Figure I. There are some fairly large hemorrhagic areas. In one microscopic field (No. 6 objective, Leitz) three gland tubules were found in which there were so many coccidia that not a single intact epithelial cell could be detected. Throughout this section several tubules were found in a similar stage of destruction. The liberation of the coccidia from the gland epithelium takes place into the surrounding tissue and not into the lumen of the gland unless to a far lesser degree. Most of the schizonts present are nearly round and about 13.5 microns in diameter. Thus they are most likely of the third generation. The cecal contents consist of tissue debris immumerable oocysts, blood and necrotic material.

Discussion.

In young chicks coccidia most frequently infect the ceca and some times the lower part of the small intestines. The result of such an infection may be fatal hemorrhage, due to the development of immense numbers of coccidia in the cecal mucous membrane causing such extensive

destruction of tissues as to bring about hemorrhage through the denuded and disintegrated mucosa. As a consequence chicks may die without premonitory symptoms. In such cases a post mortem examination will show numerous petechial hemorrhages, visible through the serous membrane in the ceca, and quite often also in the lower third or half of the small intestines. The contents of the ceca will be found to consist of liquid or semiliquid, partly hemolyzed blood.

Less acute attacks generally manifest themselves with paleness, chills (crowding) ruffled feathers, drooping wings, general droopiness and weakness. Bloody, bloodstained or brownish droppings are almost sure signs of coccidiosis. Chicks so affected will die a few hours or days after showing the first signs of disease. A post mortem examination will reveal cheesy, more or less blood stained masses in the ceca. These cheesy masses adhere more or less to the cecal wall and leave a raw surface when removed. The lower part of the small intestines usually shows hemorrhagic enteritis and catarrhal inflammation may be present in the upper part of the intestinal tract.

Young chicks affected with pullorum disease often have white or yellowish white smooth cheesy "plugs" in the ceca. These "plugs" are never mixed with appreciable quantities of blood, do not adhere to the cecal walls nor leave a raw surface when removed.

Chicks six weeks of age or older not infrequently have duodenal coccidiosis; i.e., they may have a rather severe duodenal infection with less involvement of the lower part of the small intestines while the ceca are only very slightly or not at all affected. Such chicks show general unthriftiness, the appetite may be increased or decreased and, as is the case with most chicks affected with subacute or chronic coccidiosis, they may show increased thirst. Leg weakness and paralysis are rather common symptoms and not infrequently the birds look alert and quite well with the exception of the inability to use their legs. The appetite may be good for some time after paralysis has set in. Recovery does not take place but the chicks may live for many days with such an affliction.

In less severe cases all one may notice is a retardation of growth and general unthriftiness with more or less anemia. Such manifestations are undoubtedly due to the destruction of epithelium and mucous membrane which has taken place in the small intestines resulting in increased permeability to secondary infection and toxins as well as decreased power of digestion and assimilation, or it may be due to absorption of toxic material from necrotic material in the ceca. Since most birds that are affected in this way seldom show involvement of the ceca it seems logical to conclude that disease processes in the small intestines are more often back of this trouble. A study of the microscopic changes

of coccidiosis of the small intestines showed that destruction of glands, desquamation of epithelium and sloughing of the mucous membrane may be too extensive to allow complete repair to take place. This may account for the fact that apparently healthy chickens fail to put on flesh even when fed in the best way possible.

Duodenal coccidiosis affects fully grown birds as well as growing stock. It has been observed in fowls eighteen months old. The symptoms manifested are general unthriftiness, loss of flesh, more or less anemia or a peculiar pale, pinkish color of the comb. Leg weakness, paralysis, incoordination of movement and increased excitability may also be observed. More or less diarrhea may be present in young and adult birds affected with duodenal coccidiosis, but this is not always a conspicuous symptom. Very seldom do we see blood in the droppings of chicks or adults so affected. Slimy droppings are noticed more often. A post mortem examination of birds killed in the early stages of the disease will usually show more or less thickening of the intestinal wall, mostly in the duodenum but also once in a while throughout the small intestines. Now and then the middle portion will show the most marked swelling and, in rather exceptional cases, the lower part. Quite distinct, grayish spots and hemorrhages are often observed through the serous coat. The grayish spots are deep seated colonies of coccidia. The mucous membrane is swollen, some times with gray spots or transverse small grayish streaks,

representing colonies of coccidia in the epithelium of the villi and petechial or more or less diffuse hemorrhagic areas. Slimy, dirty exudate is common. Ulcers may occur and diphtheritic membranes have also been observed. If the bird has been ill for about two weeks before the examination the intestines will appear thin or shrunken, empty, reddish and inflamed. There will also be evidence of sloughing of mucous membrane. Coccidia are usually easily found except in the cases in which birds have been sick for about two weeks and show contraction of the intestines. An examination of the intestinal or cecal contents may fail to reveal coccidia while scrapings taken more or less deeply in the mucous membrane may show large numbers of them and vice versa.

The liver of affected birds may show whitish or grayish spots evenly scattered or clustered in such a way as to suggest the presence of blackhead. Now and then typical blackhead lesions have been found in chicks four to eight weeks or so of age. In no instance have coccidia been found in livers showing either the grayish spots or the more or less typical blackhead lesions. Areas of perivascular infiltration with undifferentiated mesenchymal cells were of common occurrence in the birds studied. These areas might be thought to be due to secondary infection but very few positive bacterial cultures were obtained from livers showing such lesions. The livers showing blackhead lesions revealed a protozoan parasite, Figure XIII, now being studied by Doctor A. A.

Chandler, parasitologist and protozoologist at this station.

A glistening rather uniformly brownish color of the heart was usually observed and the heart muscle was generally flabby. An increase in pericardial fluid was not uncommon. On microscopic examination there seemed to be more or less fatty degeneration in the muscle fibres and the musculature seemed loose in many cases.

Enlargement of nerves was observed quite often. Undifferentiated mesenchymal cell infiltration was found in the brain, spinal chord and peripheral nerves some times even if no thickening of these nerves was noticable.

Whether there is any connection between the changes noted in the heart and nervous tissues and coccidiosis infection is difficult to say.

Summary.

Coccidiosis in young chicks usually has its seat in the ceca and lower portion of small intestines. The gross pathological changes are: various degrees of hemorrhage and formation of more or less bloodstained cheesy material in the ceca. Microscopically one sees: destruction of epithelium of the villi and gland tubules and sloughing of the mucous membrane. The cecal contents consist of tissue debris, necrotic material, blood cells and coccidia.

In birds over eight weeks of age one usually finds that the ceca are very seldom visibly affected. Duodenal infection is most common and the other parts of the small intestines may be involved at times. The gross pathological

changes are: thickening of the intestinal canal in the early stages and shrinking after sloughing of mucous membrane has taken place. Colonies of coccidia may appear as grayish spots or streaks in the mucous membrane, some times visible through the serous coat. There may be hemorrhages, dirty mucous exudate, ulcers and diphtheritic membranes. Microscopically we find: some coccidia seem to do little damage infecting only single cells here and there. Others infect extensive areas of epithelium causing desquamation, some infect glandular epithelium as well as villous epithelium causing desquamation of both and again we have those which cause "subepithelial" * infection and produce sloughing of mucous membrane. It appears from this study that there are several species of coccidia, the Eimeria tenella and the E. necatrix having been quite definitely recognized.

Areas of infiltration with undifferentiated mesenchymal cells occured frequently in the liver of infected chickens and always around the foci of infection found in the stroma mucosae. Many such areas were found showing no coccidia but it can not be stated with any degree of assurance that they always represent lesions of a more or less recent coccidiosis infection.

* As observed by Tyzzer coccidia in the subepithelial tissues are evidently contained within enlarged epithelial cells.

Leg weakness and paresis occur more frequently in connection with duodenal than cecal coccidiosis.

Coccidia morphologically indistinguishable from some of those found in chickens were found in pigeons showing leg weakness. Coccidia were not found in the livers of infected chickens.

No evidence was found to show that sparrows carry the Eimeria.

Description of Plate I.

Fig. I. Very extensive "subepithelial" infection with Limeria tenella of villi in the small intestines of an adult bird. Trophozoites, schizonts, gametocytes and oocysts were found in these areas.

Fig. II. Epithelial infection with Limeria acervulina in duodenum of adult bird. Every epithelial cell from (a) to (b) seems to be infected. Note that villi on the left are denuded of epithelium and that patchy desquamation has begun to take place in the infected villus.

Fig. III and IV. Deep seated foci of coccidial infection. The bird from which these sections were obtained showed no coccidia in intestinal contents. The intestines were shrunken, empty, and showed macroscopic evidence of sloughing of mucous membrane. Note injury to glands and muscularis mucosae and the extensive cell infiltration of the interglandular tissue and muscularis mucosae as well as the adjacent muscular coat.

Plate I

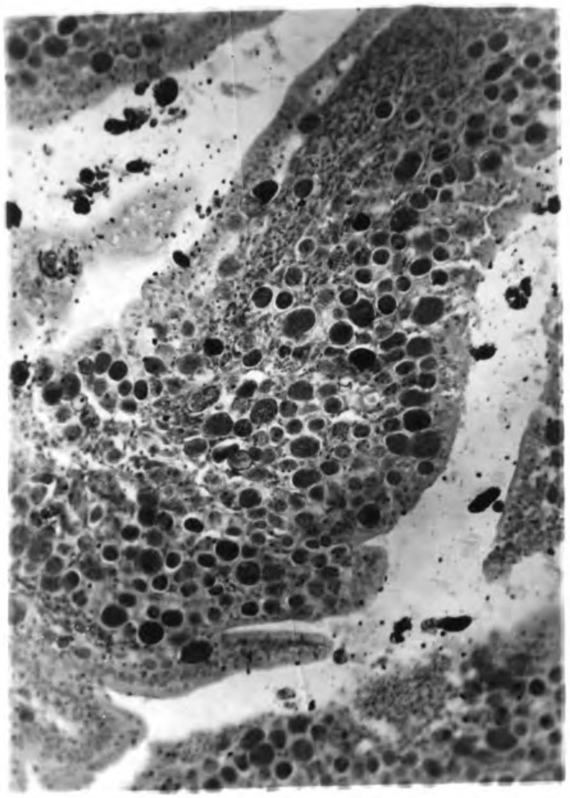

Fig. I

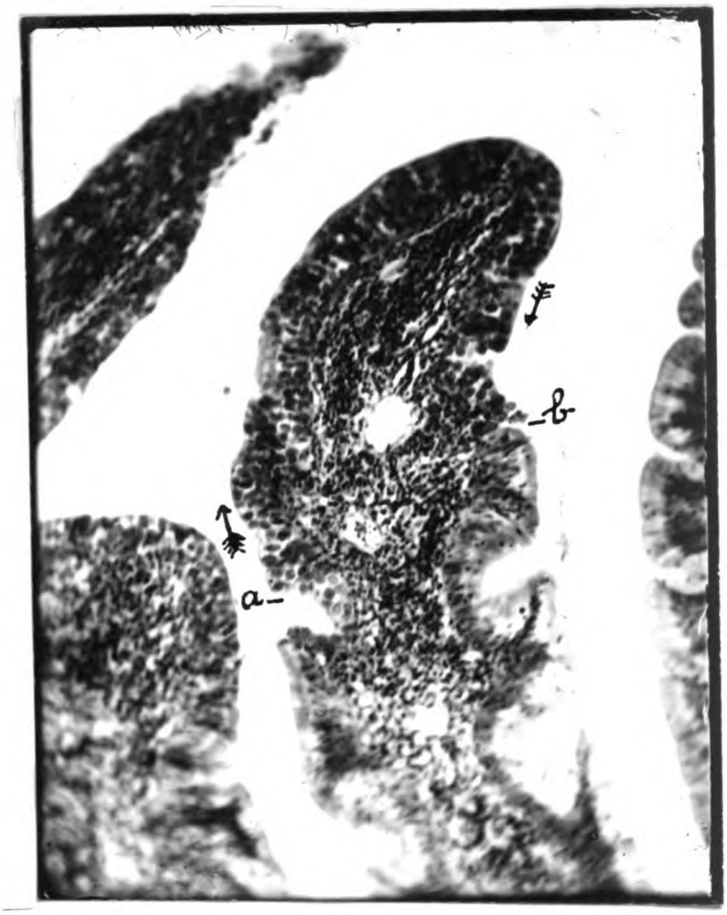

Fig. II

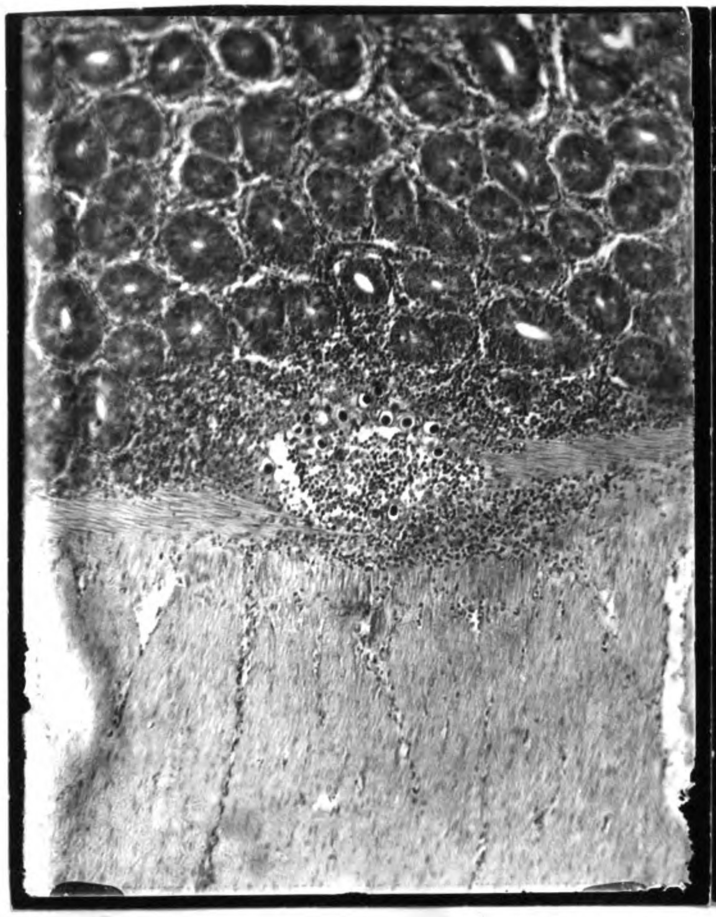

Fig. III

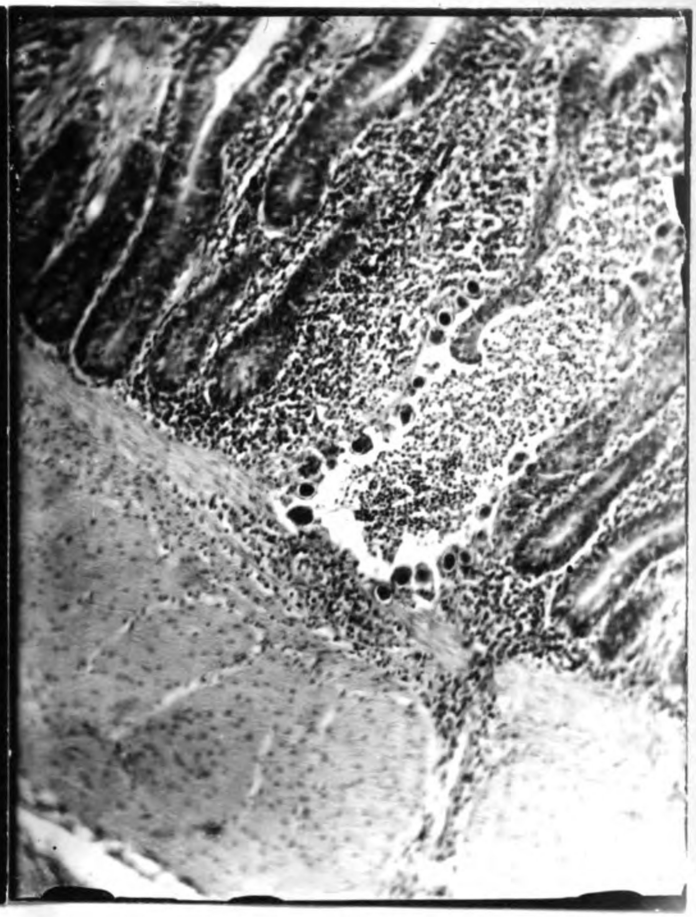

Fig. IV

Description of Plate II.

Fig. V. Marked epithelial infection with desquamation of epithelium over the tips of the villi. Note the large oocyst located just below the epithelium. (a)

Fig. VI. Moderate infection with Eimeria tenella showing coccidia below nuclei of epithelial cells (a) and just below epithelium (b). Note ragged appearance of epithelium over tip of villus and separation of epithelium from subepithelial tissues and cellular infiltration in core of villus.

Figs. VII and VIII. Schizonts in inter-glandular tissue. Note extensive cellular infiltration and disappearance of gland tubules, also large number of schizonts in Fig. VIII. Eimeria tenella

Plate II

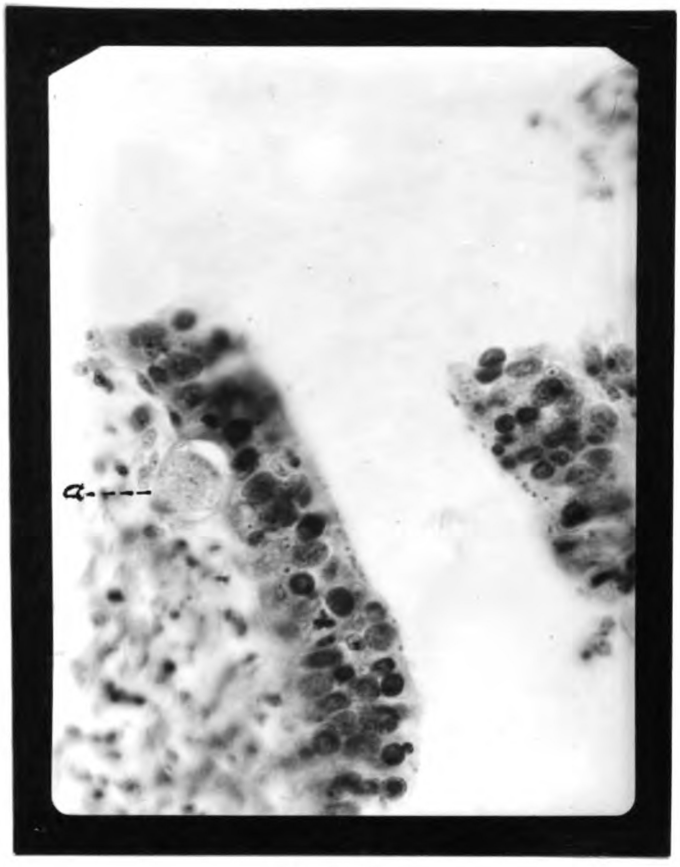

Fig. V

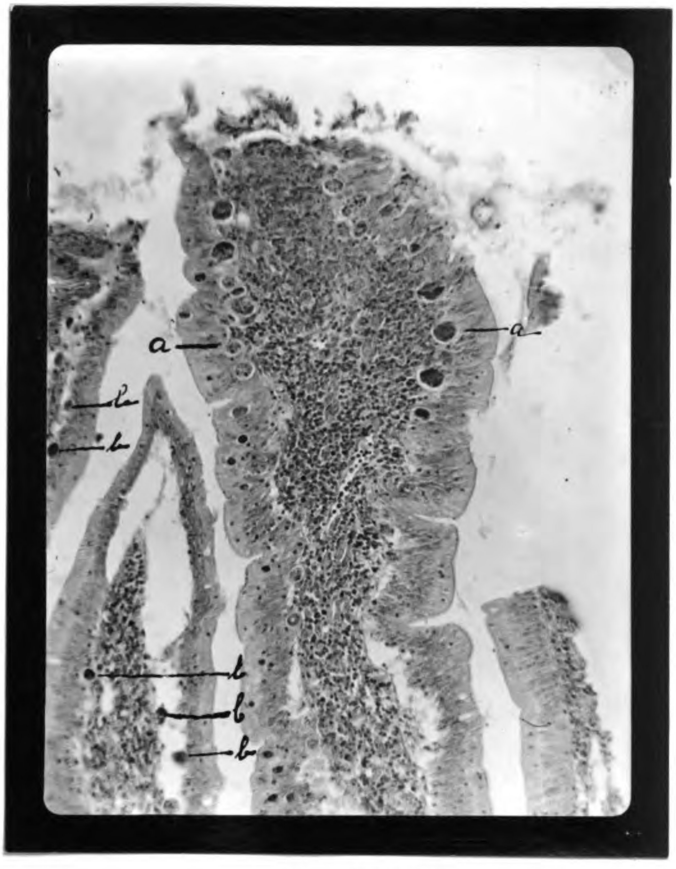

Fig. VI

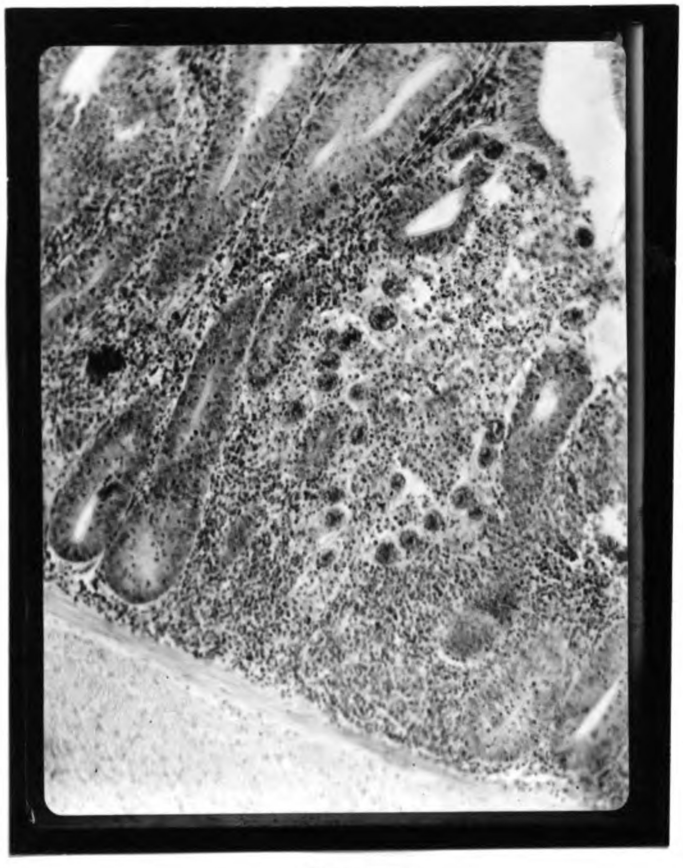

Fig. VII

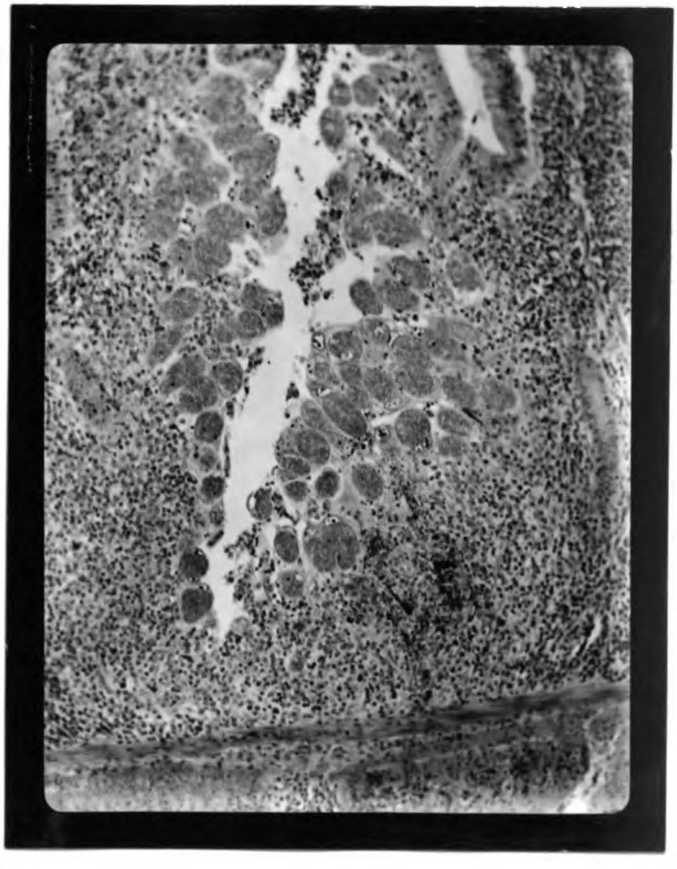

Fig. VIII

Description of Plate LI.

Fig. LI. (a) Schizonts in interglandular tissue of cecum of chick.
(b) Infection of gland epithelium. Eimeria tenella

Fig. LII. (a) Tissue debris and oocysts in lumen of gland. Note sloughing of villous portion of mucous membrane and cecal contents (b) consisting of tissue debris, necrotic material and oocysts. Eimeria tenella.

Fig. LI. (a) Massive infection of gland epithelium and (b) cellular infiltration in cecum of a chick.

Plate III

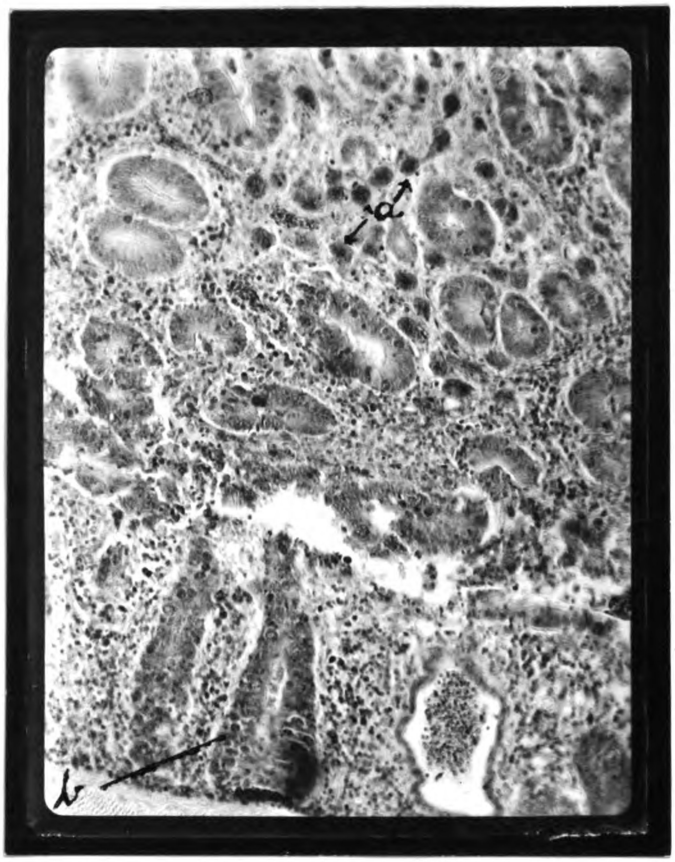

Fig. IX

Fig. X

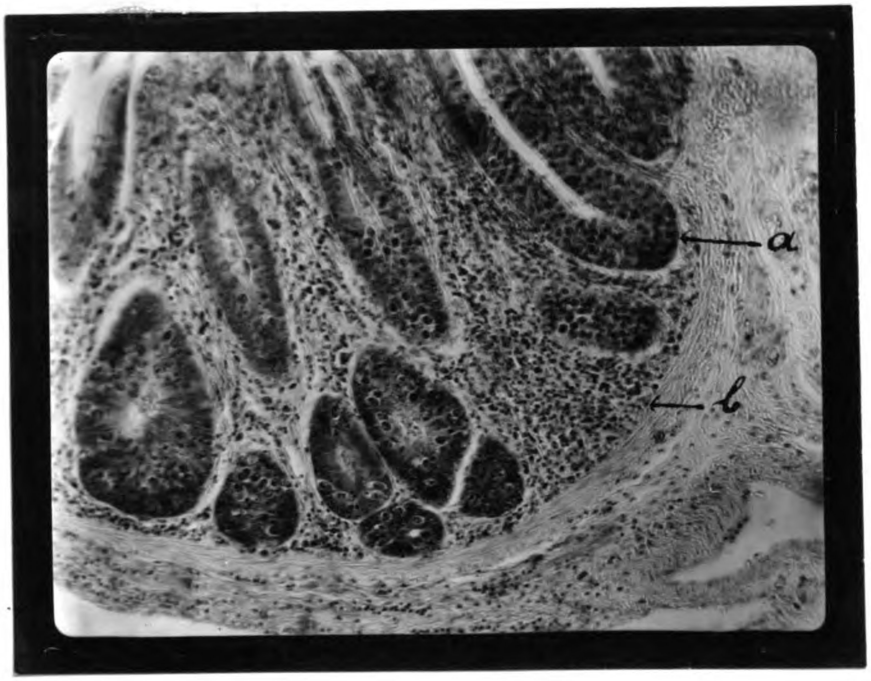

Fig. XI

Description of Plate IV.

Fig. XII. Area of undifferentiated mesenchymal cell infiltration as commonly seen in livers of chickens.

Fig. XIII. Section from the liver of a chick showing blackhead lesions, (a) causative organism.

Plate IV

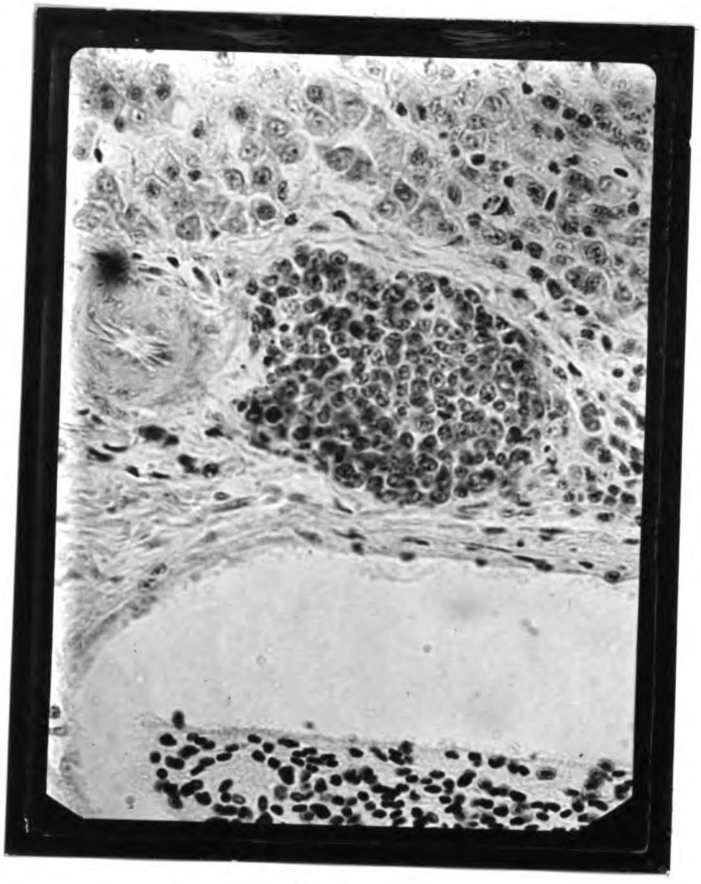

Fig. XII

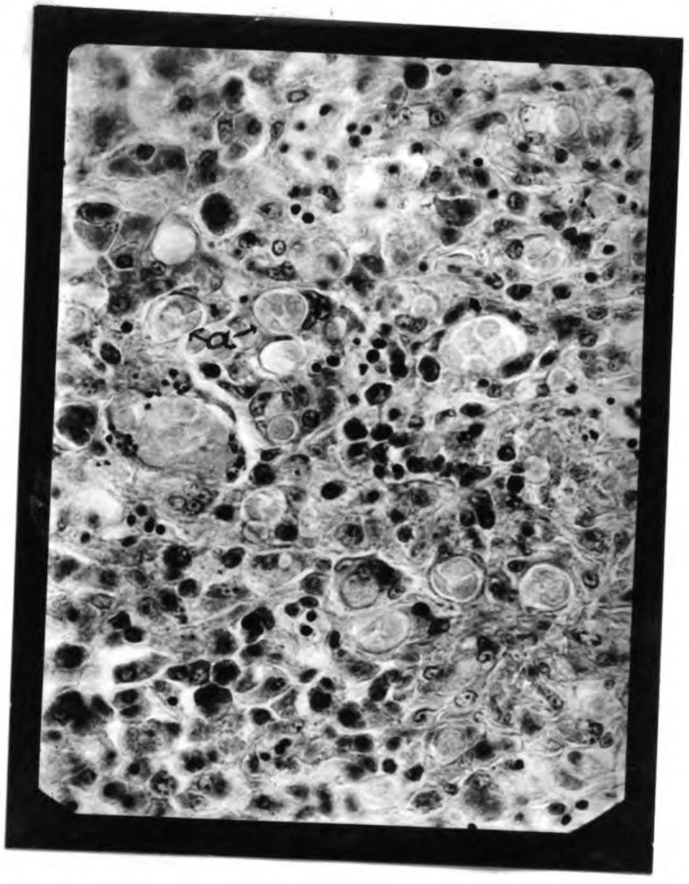

Fig. XIII

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Bibliography.

1. Eckardt, Uber Coccidiosis intestinalis beim Geflügel. Berl. Tierarztl Wochenschr. Jahrg 177, 1903.
2. Hadley, P. B., Coccidia in Subepithelial Infections of the Intestines of Birds. Jour. of Bact., 2, No. 1, 1917.
3. Hard, A. R., and Gallagher, B. A., Diseases of Domesticated Birds. The Macmillan Co., N. Y. 1920.
4. Hutyra, F. and Marek, J., Special Pathology and Therapeutics of Diseases of Domesticated Animals (American Edition). Alexander Eger, Chicago, 1926.
5. Reinhardt, R., Lehrbuch der Geflügelkrankheiten, 2 Aufl. Verl. L. u. H. schaper, Hannover, 1925.
6. Joest, E., Spezielle pathologische anatomie der Haustiere, 1, 2 Aufl. s. 766, Verl. Richard Schoetz, Berlin 1926.
7. Otte, W., Krankheiten des Geflügels, Richard Schoetz, Berlin 1928.
8. Krijgsman, B. J., in Handbuch der Geflügelkrankheiten und der Geflügelzucht, herausgegeben von T. V. Heelsbergen, Verl. Ferdinand Enke, Stuttgart, 1929.
9. Tyzzer, E. E., Coccidiosis in Gallinaceous Birds. Am. Journal of Hyg. Vol. X. 2, Sept. 1929.
10. Kaupp, B. F., Poultry Diseases, Fifth Ed., Alex Eger, Chicago 1929.
11. Stafseth, H. J. and Johnson, E. P., Range Paralysis in Poultry, Quarterly Bulletin, Mich. Agr. Exp. Sta. Vol. 9, No. 3, Tech. 1927, pp. 105 - 108.

12. Stafseth, H. J., Rickets as a Secondary Manifestation in Coccidiosis. Journal Am. Vet. Med. Assn., Vol. 27, (n.s.) No. 7, 1929, pp. 1069.

13. Cole, L. S. and Hadley, P. B., Blackhead in Turkeys: A Study in Avian Coccidiosis. Rhode Island Agr. Exp. Sta. Bul. 141, 1910.

14. Haupp, B. F., Poultry Diseases, Fifth Ed., Alex Eger, Chicago 1929.

15. Smith, T. and Smillie, L. W., A Note on Coccidia in Sparrows and Their Assumed Relation to Blackhead in Turkeys. Jour. Exper. Med., 25, No. 3, 1917, 415.

16. Pappenheimer, A. M., et al., Fowl Paralysis, Storrs Agr. Exp. Sta. Bul. 143, 1926, pps. 209 - 212.

17. Idem. Fowl Paralysis (Neurolymphomatosis gallinarum) Jour. Exper. Med. XLIX, 1929, p. 65.

18. Johnson, W. T., Coccidiosis of the Chicken, Oreg. Agr. Exp. Sta. Bul. 238, Dec. 1928.

19. Beach, J. R. and Davis, D. E., The Influence of Feeding Lactose or Dry Skim Milk on Artificial Infection of Chicks with Eimeria avium, Milgardia, 1, No. 8, 1925, p. 167.

20. Doyle, L. P., Paralysis in Chickens. Purdue University Extension Leaflet, No. 146, Dec. 1927, p.8.