PSITTACOSIS

I. EXPERIMENTALLY INDUCED INFECTIONS IN PARROTS

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Plates 5 to 7

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The widespread outbreak of psittacosis in man in 1929–30 led to renewed interest in the disease which quickly resulted in the discovery of the facts that the causative agent is not Nocard's bacillus but a filterable virus (1) and that the incidence of laboratory infections is high. Because of these facts it became imperative that further work on the nature of the etiological agent and the mode of spread of the infection be undertaken.

Krumwiede and his coworkers (2) were among the first to show that the inciting agent of psittacosis is capable of passing filters and that mice are susceptible to the disease. Due to his ill health, however, and because of the development of psittacosis in several of his assistants, investigations so admirably begun by him had to be discontinued and two strains of the virus with which he was working were given to us for further study. The purpose of this paper and the three that follow is to present in detail investigations, already described in preliminary papers (3–5), concerning psittacosis experimentally produced in parrots, mice, rabbits, guinea pigs, and monkeys. Special attention has been paid to the mode of spread of the disease and to the pathological changes induced, depending upon the host and upon the portal of entry of the virus.

Methods and Materials

Virus—Krumwiede provided us with two strains of virus, one from a patient, the other from a parrot. Parrot Wenz C after feeding upon lung and spleen from a fatal case of psittacosis was transferred to our laboratory. Parrots N, O, and R were inoculated intramuscularly with spleen emulsion from Parrot M, and Parrot K was infected intramuscularly and intraorally with organ filtrate from Parrot
F. Following inoculation these birds were sent to The Rockefeller Institute. From examination of Text-fig. 1, it will be seen that the virus with which Parrots K, N, O, and R were inoculated came originally from a natural infection in Parrot 17. In Text-fig. 1, the doubly starred birds were handled only by Krumwiede and his coworkers, the singly starred parrots were inoculated by them and given to us, the birds without stars were seen and handled only by us.

Parrots.—Most birds used were Amazon parrots, while the remainder came either from Cuba or Mexico.

Inoculations.—Inoculations were effected by instillation of the virus in the nose and mouth, by pollution of the food and drinking water with the inciting agent, or by injection of the virus into the pectoral muscles.

Examination of Tissues.—The majority of the autopsies was performed immediately after the animals had died or after they had been sacrificed by means of chloroform. In all instances, aerobic and anaerobic cultures from various organs were made for the detection of ordinary bacteria. Sections from tissues fixed in Zenker’s fluid and in 10 per cent formalin were stained with eosin and methylene blue or according to Wolbach’s modification of Giemsa’s method.

Measures Employed to Prevent Laboratory Infections.—At the time we began our work it had already become apparent that laboratory infections are a menace. Consequently measures were instituted to prevent as far as possible the spread of the disease to those actually working with the malady and to protect completely other individuals in the Institute.

Two rooms on the same floor that contained our laboratory and the central media rooms were placed at our disposal for the housing of animals. The openings into the animal rooms were so fixed that no cracks between the doors and windows and their frames, large enough to permit the passage of insects, remained. Furthermore, the doors and windows were doubly screened with coarsely and finely meshed wire. Thus the mechanical spread of the virus by mice and insects was absolutely prevented. One room housed the animals known to be infected, while the other was used for animals inoculated with material undergoing tests for the presence of virus. In the “clean room” the animals were caged in individual units and workers before going from one section to another washed their gloved hands with 5 per cent lysol.

All cages were cleaned and then sterilized with 5 per cent lysol 3 times a week as well as between experiments. Droppings and bedding were collected in large covered cans containing 5 per cent lysol. After the exterior of the cans had been washed with lysol and while the containers were still wet, they were conveyed to the incinerator into which the refuse was dumped and immediately burned. Animals to be autopsied were dipped, after death, in 5 per cent lysol and wrapped in towels moistened with the same solution before being taken to the laboratory. Finally, the floors were cleansed each day with lysol and allowed to remain wet.

The cleaning of the cages and rooms and the examination of the sick animals constituted the most dangerous duties connected with the work. This is true, because the virus probably enters man through the upper respiratory tract, i.e.,
the conjunctival sac, nose, and mouth. When the work was begun, however, this fact was not definitely known. Consequently a costume to prevent all modes of infection was devised. A photograph of this costume is shown in Fig. 1. The uniform consists of air-tight goggles, the lenses of which were treated with a patented preparation of soap and glycerol to prevent steaming, a mask made of several thicknesses of gauze to cover the nose and mouth over which was placed a respirator frame, a hood covering the head and shoulders (the goggles were held in the hood by firmly tied draw-strings), a surgeon's gown over which was placed a heavy rubber apron, heavy rubber obstetrical gloves reaching to the elbows, and heavy rubber boots long enough to cover the legs as far up as the knees. When handling the animals, the workers wore heavy leather gloves wet with lysol in order to prevent injury to the hands and destruction of the rubber gloves. As is well known it is almost impossible to prevent the inhalation of dried infectious material unless a gas mask is used. The costume employed by us was fairly efficacious chiefly because of the dead space created by the respirator frame placed between the gauze mask and hood. When working with the virus in the laboratory, we wore goggles, a gauze mask, a surgeon's gown, and rubber gloves. The parts of the costume made of cloth, upon being removed, were dipped in lysol solution before being sent to the laundry. Rubber portions of the costume were sterilized either by boiling or by immersion in lysol solution.

All containers with liquid media were inclosed in tin cans while being incubated. Petri dishes with cultures were sealed by means of strips of rubber cut from Ford inner tubes. These measures were used to prevent the entry of insects into the plates and the consequent spread of infection, and to protect other workers using the incubator.

Three people were actively engaged in the work, while 5 other individuals were employed in different capacities in the laboratory. Furthermore, our laboratory and animal house are the most centrally located ones in the Institute. The measures we used prevented a general outbreak of psittacosis, but, for some reason not known to us, they were insufficient to protect one of the doctors studying the disease. He came down with it. We believe that the infection took place by way of the upper respiratory tract, the portal of entry most difficult to protect.

EXPERIMENTAL

In the experimental work concerning psittacosis in parrots our chief interests were centered around the portal of entry of the virus, the distribution of the incitant in the body, and the portal of exit of the active agent. Information regarding these matters seemed essential for an understanding of the spread of the disease from bird to bird, and from birds to man. Incidentally, we were anxious to learn something of the clinical and pathological manifestations of the disease in parrots.
From examination of Text-fig. 1, one learns that Krumwiede demonstrated that emulsions of mixed organs or filtrates of these emulsions administered intraorally or intramuscularly were capable of infecting birds. Furthermore, he showed that an emulsion of spleen alone injected intramuscularly was infectious. It remained for us to determine whether the virus is in the blood, in the liver, in the nasal secretions, and in the feces.* For this purpose, the following experiments were performed.

*We knew that the virus had been demonstrated in the droppings from infected parrots (Armstrong, C., McCoy, G. W., and Branham, S. E., Pub. Health Rep., U. S. P. H., 1930, 45, 725). The fact, however, that the inciting agent is found in droppings collected from the floor of cages is not definite evidence that the virus is excreted in the feces, because the droppings might become contaminated with virus after passage from the body.
Parrot 83, Feb. 21, inoculated intramuscularly with 1 cc. of blood from Parrot K. Feb. 26, seems slightly ill, feathers somewhat ruffled. Mar. 10, bird died rather unexpectedly, inasmuch as it had never seemed very sick. Autopsy showed normal lungs, liver, and brain, purulent (sterile) pericarditis, and friable spleen. Cultures of heart muscle, liver, and spleen remained sterile.

Parrot 86, Mar. 12, received 2.5 cc. and 0.5 cc. of an emulsion of liver and spleen intramuscularly and intraorally respectively. Remained well until Mar. 20, when for the first time it looked sick, feathers roughened, stools loose. Mar. 23, better, stools formed. Mar. 29, very sick again, weak, feathers rough, stools loose. Mar. 30, chloroformed and autopsied immediately. Emaciated, lungs normal, no pericarditis, liver large and friable, spleen twice normal size and soft, intestines injected, brain normal. Cultures of lungs, liver, and spleen remained sterile.

Parrot 91, Mar. 30, inoculated intramuscularly with 4 cc. of a 10 per cent liver and spleen emulsion from Parrot 86. Also fed 1 cc. of the emulsion. Apr. 7, sick for the first time. Apr. 11, sick, stools loose. Apr. 18, very sick, stools watery, stools collected for filtration experiment. Apr. 19, died and was autopsied immediately. Not emaciated. Lungs fairly normal, small hemorrhages in visceral pericardium. Liver large and friable; throughout the organ numerous yellowish white areas of varying size (Fig. 4). Spleen 3 or 4 times normal size and spotted (Fig. 3). Intestines injected. Cultures of lungs showed several kinds of bacteria. Cultures of liver and spleen remained sterile.

Parrot 95, Apr. 18, stool collected from Parrot 91 was diluted with Locke’s solution and filtered (half-hour) through a Berkefeld V. It was again filtered (10 minutes) through another Berkefeld V. The filtrate was free from ordinary bacteria. Of this filtrate Parrot 95 received 2 cc. intramuscularly, 1 cc. introrally, and 5 cc. in its drinking water. Apr. 20, stools loose for first time. May 8, bird has gradually become worse; weak, eats poorly, feathers roughened. May 14, died. Immediately after death, material was collected from nose. Autopsy: Lungs normal; heart covered with a flaky exudate; liver large, friable, and mottled with yellowish areas of varying size; spleen 3 times normal size; flaky exudate over spleen and liver. Cultures of liver, spleen, and exudate remained sterile.

Parrot 96, Apr. 19, received intraorally and intranasally 1 cc. of a 10 per cent liver and spleen emulsion from Parrot 91. 3 cc. of the emulsion was also put on the food and in the drinking water. Apr. 24, stools loose. Apr. 29, bird very sick and weak, stools watery. Apr. 30, bird worse, chloroformed. After death a whitish material came from nose (probably from procrop also). The bird was held over a Petri dish into which the material was allowed to drop. Autopsy: Lungs normal; no pericarditis; liver fatty and friable, studded with numerous white spots; spleen 5 times normal size, pale and friable; intestines normal; brain injected. Cultures of liver and spleen remained sterile. Spleen was contaminated while being removed. Cultures of nasal secretions showed no non-lactose-fermenting bacilli.

Parrot 98, Apr. 30, 0.25 cc. of the nasal secretions collected from Parrot 96 were...
dropped into the nose of Parrot 98. May 2, stools loose, but bird seems to be in fairly good condition. May 5, bird died rather unexpectedly. Autopsy: Hemorrhages in pericardium; lungs normal; liver large, fatty, friable, and studded with whitish spots; spleen 5 times the normal size and friable. Increase in the amount of peritoneal fluid. Cultures of this fluid remained sterile. Anaerobic and aerobic cultures of the spleen and the aerobic cultures of the liver showed no bacteria. From the anaerobic cultures of the liver a small Gram-negative influenza-like bacillus, that grew on blood agar and not on plain agar, was obtained.

Parrot 105, May 14, Parrot 95, immediately after death, was suspended, head down, over a Petri dish. 0.25 cc. of a thick whitish material were collected in this manner, and, having been diluted with a small amount of Locke's solution, were instilled in the nose and mouth of Parrot 105. May 16, stools slightly loose. May 22, stools watery, bird quiet, feathers roughened. July 7, the bird has been having recurring attacks of diarrhea. In spite of these attacks the parrot seems in good condition. Chloroformed and immediately autopsied. Not emaciated. Lungs and heart normal. Liver is extraordinary: the organ is adherent to surrounding structures, left lobe reduced to an unrecognizable small mass of scar tissue; right lobe about one-half normal size, mottled, rubbery, sectioned with difficulty. An a-p section through the whole lobe shows scar tissue in the center with normal liver tissue posteriorly. The appearance of the tissues suggests a healing or a chronic psittacosis infection. Spleen small with a whitish thickened capsule. Intestines adherent to each other in a manner suggesting a healing peritonitis. Duodenum red and inflamed. Cultures of liver and spleen remained free from ordinary bacteria.

Parrot 106, May 14, after Parrot 95 was dead and when its feathers had been plucked, the bird was dipped in 5 per cent lysol. Then the lysol was washed off with alcohol. By means of a sterile catheter attached to a syringe, 5 cc. of Locke's solution were injected into the cloaca and then withdrawn again. In this manner, fecal material was collected free from outside contaminants. 1 cc. of this material was instilled in the nose of Parrot 106. May 19, stools loose. May 22, bird worse, feathers roughened. May 25, bird very weak, right eye closed, stools watery. May 26, found dead. Autopsy: Bird emaciated. Pericardium, heart, and lungs normal. Liver large, fatty, friable; along interlobar fissure are yellowish areas of necrosis surrounded by bright red hemorrhagic zones, numerous similar but smaller areas ranging from pin points to peas in size were scattered through the organ. Spleen 4 times normal size and friable. Cultures of liver revealed no ordinary bacteria.

Parrot 107, May 28, stool collected from Parrot 106 in a manner similar to that employed with Parrot 95 (see Parrot 106 for details). 1 cc. of this fecal material was instilled in the nose and mouth of Parrot 107. May 30, stools loose. June 2, bird worse. June 11, the bird has been gradually getting worse; weak, feathers roughened, severe diarrhea. Chloroformed and autopsied immediately. Lungs normal. Pericardium contains 0.5 cc. of purulent-looking, sticky exudate. Liver
smaller than normal and shows numerous necrotic zones. Spleen 3 times normal size and friable. Cultures of liver remained sterile. Smears from liver and spleen showed no "minute bodies." Preparations from the pericardial exudate stained according to a modification of Castaneda's methylene blue safranin method* revealed numerous "minute bodies," of the type first described by Levinthal (6).

The experiments detailed above are summarized in Text-fig. 1. From an examination of the results, certain facts become obvious. In the first place, the virus of psittacosis is found in the stools, in a mixture of nasal secretions and material from the procrop, in the blood, in the liver, and in the spleen of infected birds. Moreover, parrots are capable of being infected by intramuscular, intranasal, or intraoral inoculations of the virus. These facts indicate the manner in which the disease spreads from bird to bird, and also suggest the source of infection for man.

The clinical picture of psittacosis in birds varies. The disease may be acute, running its course in a few days, or it may be chronic, enduring for several months. The birds may die suddenly without showing appreciable signs of illness. As a rule, however, loss of weight, roughening of the feathers, weakness, watery stools, and discharge from the nose are observed during the course of the disease. In our experience, the majority of the infected birds died. The gross pathology is characterized by an occasional sterile pericarditis, enlarged, fatty livers, many of which show areas of necrosis (Fig. 4) or infarction, enlarged friable spleens (Fig. 3), and injected intestines. Regardless of the mode of inoculation, none of the parrots revealed changes in the lungs that might be attributed to psittacosis.

Microscopic Pathology

Spleen.—Changes in the lymphoid follicles vary from slight alterations to an almost complete obliteration of their normal architecture (Figs. 5 and 6), while

* Phosphate buffer pH 7.0 ....................................... 95
Formalin .................................................... 5
Loeffler's methylene blue ...................................... 10

Stain 2 minutes, rinse with tap water, and quickly counterstain with aqueous safranin.
TEXT-Fig. 1. Diagrammatic representation of the experimental work concerning psittacosis conducted in parrots. The doubly starred birds were handled only by Krumwiede, the singly starred parrots were handled both by Krumwiede and by us, the birds without stars were handled only by us. † indicates that the bird died.
the reticular and sinus structures are well preserved. The organ is infiltrated with wandering phagocytic cells with vacuolated cytoplasm containing amorphous debris, pigment, and globules of fat. The increase in the size of the spleen seems to be due to the content of blood and the enormous number of mononuclear cells.

Liver.*—The characteristic lesion of psittacosis in parrots consists of multiple discrete areas of necrotic liver cells irregularly distributed throughout the organ, but with a tendency to be more numerous near its periphery. The condition appears to have its onset in the death of isolated liver cells or groups of cells; the cytoplasm becomes acidophilic and granular, and shrinks from contact with other cells. The nuclei become hyperchromatic and pyknotic, and eventually disappear completely. At this stage, mononuclear phagocytes and a few polymorphonuclear cells surround and infiltrate the lesion. As the process progresses, the necrotic liver cells disintegrate, leaving strands of acidophilic, hyaline material which may or may not show collections of leucocytes and depositions of fibrin (Fig. 7). When necrotic areas penetrate to the surface of the liver, accumulations of inflammatory cells are seen under Glisson's capsule, and an extension of the process leads to the perihepatitis and peritonitis that are encountered. Around the zones of necrosis, proliferation of liver cells, indicated by mitotic figures, occurs. Within and around the lesions, cells, for the most part “endothelial leucocytes,” filled with the “minute psittacosis bodies,” are also found (Fig. 2).

Throughout the liver there is a proliferation of Kupffer cells and focal accumulations of wandering mononuclear cells of the same general type. Some of these have a highly vacuolated cytoplasm containing masses of fat. Many plasma cells are also present.

The bile ducts do not escape injury. Within the necrotic areas, they are dilated, and at times contain numerous mononuclear phagocytes. The cells that form the walls of the ducts may undergo necrosis, becoming granular with pyknotic or missing nuclei. In chronic and in healing lesions, irregularly shaped collections of hepatic cells separated from each other by various sized bile ducts undergoing proliferation and surrounded by lymphocytes and fibrous tissue (Fig. 8) are observed. The dilatation of the bile ducts and the character of the lesions in the later stages of the disease which resemble those produced by ligation of the common duct, lead one to believe that occlusions of the biliary system in the parrot may occur in some manner as the result of an infection with the virus of psittacosis.

Striking alterations in the vascular channels are not usually seen, but in certain

* The anatomy of the parrot's liver differs from that of mammalian livers in that the bile duct draining the left lobe enters directly into the duodenum. There are also differences in the microscopic appearances of the two kinds of livers. For example, lobulation in the parrot's liver is not well developed and arrangement of the parenchymal cells in chains occurs only to a slight extent. Moreover, the bile ducts, which in mammals are invariably associated with blood vessels, may at times be found unassociated with such structures in the parrot.
<table>
<thead>
<tr>
<th>Parrot No.</th>
<th>1st inoculation Material, Route, date</th>
<th>Virus in inoculum*</th>
<th>Course after inoculation</th>
<th>Time between inoculations days</th>
<th>Re inoculation Material, Route, date</th>
<th>Course after inoculation</th>
<th>Autopsy</th>
<th>Active immunity + or −</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Parrot spleen i.m. 2-22-30</td>
<td>+</td>
<td>Sick 1 mo.</td>
<td>95</td>
<td>Negative</td>
<td>Died on 9th day</td>
<td>Psittacosis</td>
<td>−</td>
</tr>
<tr>
<td>McG</td>
<td>Human blood i.m. 3-7-30</td>
<td>−</td>
<td>Negative</td>
<td>82</td>
<td>Died on 9th day</td>
<td>Psittacosis</td>
<td>−</td>
<td></td>
</tr>
<tr>
<td>N1</td>
<td>Parrot liver Oral and i.m. 3-8-30</td>
<td>+</td>
<td>Sick 3 wks.</td>
<td>81</td>
<td>Occasional mild diarrhea</td>
<td>Lived</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>89</td>
<td>Mouse organs Oral and i.m. 3-28-30</td>
<td>+</td>
<td>Sick 1 mo.</td>
<td>61</td>
<td>Negative</td>
<td>Lived</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>92</td>
<td>Human blood Oral, i.p., i.m. 4-3-30</td>
<td>−</td>
<td>Negative</td>
<td>55</td>
<td>Typical illness, killed after 9 days</td>
<td>Psittacosis</td>
<td>−</td>
<td></td>
</tr>
<tr>
<td>93</td>
<td>Human nasal washings Oral 4-3-30</td>
<td>?</td>
<td>Very slight diarrhea</td>
<td>35</td>
<td>Typical illness, killed after 6 days</td>
<td>Psittacosis</td>
<td>−</td>
<td></td>
</tr>
<tr>
<td>107</td>
<td>Control—Inoculated by oral and nasal instillation of 1 cc. of stool from Parrot 106, sick with psittacosis. Was sick 48 hrs. after inoculation; ran fulminating course; was killed when moribund on 14th day. Autopsy showed typical psittacosis</td>
<td>−</td>
<td>Negative</td>
<td>31</td>
<td>Mouse organs Oral and i.m. 6-9-30</td>
<td>Typical illness, killed on 8th day</td>
<td>Psittacosis</td>
<td>−</td>
</tr>
<tr>
<td>102</td>
<td>Human lung Oral and i.m. 5-9-30</td>
<td>−</td>
<td>Negative</td>
<td>31</td>
<td>Mouse organs Oral and i.m. 6-9-30</td>
<td>Typical illness, killed on 8th day</td>
<td>Psittacosis</td>
<td>−</td>
</tr>
</tbody>
</table>

Control—Material used for reinoculation of Parrot 102 produced psittacosis in Mice WC4 and Monkeys H, I, and J.

* Presence or absence of virus in inoculum determined in addition by other animal inoculations.
cases fibrin thrombi, involving portal vessels, are found. In most instances, only small vessels are occluded, but occasionally a large branch is involved. The question as to whether the areas of necrosis result from vascular thromboses or whether the thromboses are caused by necrosis cannot be definitely answered. But the evidence is such that it seems unlikely that much of the necrosis is the result of vascular occlusions.

**Immunity**

Having investigated the portal of entry and exit of the virus in parrots and upon concluding the study of the clinical and pathological pictures of the disease in its natural host, we then became interested in determining whether the birds that had recovered from psittacosis were resistant to reinfection.

For the work on active immunity, 7 parrots were available; 3 had recovered from psittacosis experimentally induced 61–91 days prior to the reinoculation, 3 had been inoculated 31–82 days previously with material subsequently shown to be free of virus, 1 had received intraorally either a very small amount of virus or none at all. In any event, the last bird mentioned evidenced few if any signs of illness following the first inoculation. 6 of the birds (O, McG, N, 89, 92, 93) and a control received intranasally and intraorally 1 cc. each of an unfiltered stool from Parrot 106 sick with psittacosis. The seventh parrot in the group (102) was tested for immunity by means of a virus-containing emulsion from mice. The 3 parrots (O, N, 89) that had recovered from a previous infection lived, while the others developed psittacosis and reacted in a manner similar to that of the control. The results of these experiments are summarized in Table I.

From the experiments described above and summarized in Table I, it is obvious that parrots are actively immune following an attack of psittacosis.

**DISCUSSION**

The results of our investigations concerning psittacosis experimentally induced in parrots need few comments. It seems advisable, however, to emphasize again the danger of studying the disease in parrots. Due to the presence of virus in the nasal secretions and feces of infected birds and because of the parrot's filthy habits, to protect oneself against the entry of dried virus into the upper respiratory tract is extremely difficult. In view of this fact, and since mice are suitable for diagnostic and experimental work, as demonstrated in the second paper of this series, investigations with parrots should be limited as much as possible.
The "minute bodies" found in exudates and in certain infected organs were first described by Levinthal (6) who thinks that they are of etiological significance and probably represent small bacteria of a nature similar to that of *B. tularense*. Lillie (7) believes these structures are *Rickettsiae* and proposes for them the name *Rickettsia psittaci*. Coles (8) speaks of them as "x-bodies" and is also of the opinion that they probably constitute the causal agent. We have experienced no difficulty in finding these bodies in some animals, while in others extensive search has failed to reveal them. When present they take stains with ease, are Gram-negative, and closely resemble minute microorganisms with a diameter of about 0.2μ. As yet, however, no one has succeeded in cultivating them on ordinary laboratory media, and their exact nature and relation to psittacosis is still an open question.

CONCLUSIONS

1. The virus of psittacosis is present in the nasal secretions, feces, blood, spleen, and liver of an infected parrot.

2. Parrots are susceptible to intraoral, intranasal, or intramuscular inoculations of the virus.

3. The most constant pathological changes produced by psittacosis in parrots occur in the spleen and liver. The lesions exhibited in the latter organ consist of areas of necrotic liver cells and damage to bile ducts. In no instance, in our experience, were lesions observed in a parrot's lungs comparable to those found in the lungs of men.

4. "Minute bodies" similar to those described by Levinthal and others were found in many, but not in all of the infected birds.

5. Parrots that have recovered from one attack of psittacosis exhibit an active immunity against reinfection.

REFERENCES


EXPLANATION OF PLATES

PLATE 5

**Fig. 1.** Photograph of costume worn in animal rooms. A parrot with psittacosis is perched on the left wrist of the worker. The other bird is normal.

**Fig. 2.** "Minute bodies" in mononuclear cells of the liver. ×1700.

**Fig. 3.** An enlarged and mottled spleen from a parrot with psittacosis. ×1.

**Fig. 4.** Liver from a parrot infected with psittacosis. The necrotic areas are white. ×1.

PLATE 6

**Fig. 5.** Section from a parrot's spleen injured by the virus of psittacosis. Normal architecture destroyed. ×170. Eosin and methylene blue.

**Fig. 6.** Normal parrot's spleen. Compare with Fig. 5. ×170. Eosin and methylene blue.

PLATE 7

**Fig. 7.** Section from a liver of a parrot with psittacosis. Early lesions showing degeneration of liver cells and depositions of fibrin. ×115. Eosin and methylene blue.

**Fig. 8.** Section from a liver late in the disease, showing proliferation of bile ducts, infiltration of mononuclear cells, and deposition of connective tissue. ×450. Eosin and methylene blue.