

Conclusions: To obtain an immediate pleural reaction by means of tubercle bacilli, previous sensitization appears necessary.

Tuberculous pleurisy in guinea pigs would appear to be a re-infection as is believed to be the case in man.

The reaction of the sensitized pleura on reinfection with tubercle bacilli is another illustration of the Koch phenomenon, and offers advantages for the study of the protective significance of this reaction.

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Philippine Fowl Disease.

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What is believed to be a new disease of fowls recently appeared in the Philippines. The epizootic began in September, 1927, and proved to be highly contagious. The first case of the disease appeared in Manila and rapidly spread over an area having a radius of about 50 miles with Manila as the center of the infection. By February 1, 1928, it has been estimated that at least 50,000 fowls have succumbed as a result of this disease. Both males and females are affected and fowls of all ages are susceptible. The disease has been prevalent in chickens, and a few cases have been noted in ducks and geese.

Symptoms: The onset of the disease is sudden. Chickens at first show an indisposition to move about, preferring to sit or stand quietly in a secluded spot. Very early in the course of the disease there is a diarrhea which gradually improves if the fowl is to recover. Gasping for air associated with jerking movements of the head downward and backward is a characteristic symptom. This is caused by large quantities of tenacious mucus which obstructs the posterior nares and pharynx. Many fowls die of suffocation early in the course of the disease. Excessive thirst is noted in many cases and the crop is often filled with large quantities of water and mucus as well as foul smelling gas in some instances.

Occasionally there is a bloody diarrhea but this is not a common symptom. The fowls usually die within 1 to 7 days following the

* This preliminary report has been prepared by Dr. McKinley and will appear in this form in his forthcoming review of the filterable virus diseases.

onset of symptoms. If the fowl is to recover from the disease the mucus in the nose and throat disappears but the fowl gradually develops a paralysis of the legs which may be either unilateral or bilateral. Of those fowls which recover from the acute symptoms and develop paralysis only a small percentage (about 5%) fully recover from the paralysis. Paralyzed fowls may live for indefinite periods provided they are fed by hand and given water at regular intervals by medicine dropper. Fully 99% of fowls develop paralysis if they survive the acute stages of the disease.

The Philippine fowl disease is similar in some respects to other diseases known to occur in fowls. It is similar to infectious bronchitis of fowls in that there is the characteristic posture of the sick fowl, head extended, beak open, gasping for breath in both conditions. The paralysis somewhat resembles the chicken paralysis disease of fowls which has been described by Pappenheimer, Dunn, and Cone, but is different from this disease in many of its other clinical manifestations. In the early stages the Philippine fowl disease resembles both infectious and nutritional roup. It is similar to fowl pest especially in the per-acute cases which die within 24 hours. Fowl pest is transmitted by minute quantities of blood to healthy fowls, but this disease is not so transmitted.

Pathology: Up to the present only the gross changes have been studied. The disease is essentially an infection of the alimentary tract. There is a peri-esophageal inflammation in all cases which may involve the crop and extend throughout the entire length of the esophagus. This inflammation is accompanied by hemorrhages which vary in size from small petechial hemorrhages to large hemorrhagic streaks or extravasations of blood. The changes in the intestinal tract are hemorrhagic in nature and vary greatly in degree. Petechial hemorrhages are also found on all the serous membranes. The eyes, nose and throat are all filled with a tenacious glary mucus during the first days of the disease and the crop frequently contains large quantities of this material. Histologic studies have only been made of the brain and cord taken from fowls during the stages of paralysis, but no definite changes have been found so far in the small amount of material which has been examined.

Etiology of the Disease: No bacterial incitant of the disease has been recovered from infected animals. The disease can be transmitted to healthy fowls by feeding the washed tissue of the intestinal wall. The disease can also be transmitted by subcutaneous inoculation of an emulsion of the intestinal mucous membrane which has been passed through a Berkefeld N filter. Rodier infected 10 out of 11 fowls which he inoculated with a Berkefeld filtrate of the

intestinal mucous membrane. All developed typical symptoms of the disease with the exception of one. In another experiment 13 fowls were inoculated with a Berkefeld filtrate of an emulsion prepared with the contents of the crop. Of these, 10 fowls developed typical symptoms of the disease while 3 appeared to be resistant. The virus is apparently present in the blood stream of infected fowls only in the early stages of the disease. Bacteria isolated from the feces of infected animals appear to be innocuous. Injection of liver, kidney and lung tissues from infected fowls into healthy fowls produces the disease in some instances. Bile in 1 instance has proved infective. Brain emulsions prepared from brain tissue of birds which have died of the disease have in all instances proved negative.

No attempts to cultivate the virus of this disease have as yet been made. From these preliminary experiments there appears to be strong evidence that this disease is caused by a filterable virus and that the disease represents a disease of fowls heretofore unknown. Since January the spread of the disease has been effectively checked by rigid quarantine measures. The causative filterable virus has been passed serially through a large number of fowls and a more complete study of the virus, the disease, and its pathology is contemplated.

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Recovery of Excitability in the Turtle Ventricle.

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Ashman and Hafkesbring¹ report a progressive increase in the excitability of turtle ventricle with repeated stimulation, *i. e.*, a *treppe* of excitability. One of us in a recent study² on the recovery of contractility in turtle ventricle, has indicated that the *treppe* in contractility depends upon the alteration of the rate of recovery after contraction. And further, that this rate of recovery varies with 2 factors: (1) the amplitude of the preceding contraction, and (2) the interval between the 2 preceding contractions. In the course

¹ PROC. SOC. EXP. BIOL. AND MED., 1928, xxv, 525.

² Blum, H. F., *Am. J. Physiol.*, 1927, xxxii, 157.