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Abstracts

ABSTRACTS

LEPTOSPIROSIS IN CATTLE is an infectious disease that may have existed in this country for some time, but was not recognized until 1944. Since that time researchers have obtained sufficient basic information to assure accurate diagnosis and to apply preventive measures.

In discussing the clinical symptoms of bovine leptospirosis it is convenient to refer to the disease as the severe and mild forms, although both are caused by the same organism.

Severe Form. This form is usually fatal within two to ten days. The onset is sudden or preceded by a day of inappetence and a drop in milk yield. During the acute phase there is fever, depression, anorexia, dyspnea and a marked reduction in milk yield. The temperature may vary from 103 to 107°F., and fever persists throughout the illness. In a day or so the mucous membranes become icteric. The milk is bloody with a pink, red, or brownish tinge. The udder is soft and pliable resembling the limp gland of a dry cow. Hemoglobinuria is usually seen, the color of the urine being bright red or dark brown. Pregnant animals are quite likely to abort early or during convalescence, and usually the placenta is expelled with the fetus. Before death the symptoms are aggravated. If severely infected cattle recover, convalescence is prolonged because of continued fever, anemia and nephritis.

Mild Form.—This form is a similar but less severe infection, rarely fatal, and lasts for two to four days. There may be depression, anorexia, dyspnea and drop in milk yield, or the animal may appear normal except for a lowered production and a change in the character of the milk and urine. Serologic tests show that inapparent infections are not uncommon. A fever of 102 to 105°F. persits for two to three days. The milk may be bloody but it is more commonly thick, yellowish and viscid. The mammary gland is always soft and limp. Abortions do not occur as frequently as in the severe disease, and hemoglobinuria is uncommon.

Diagnosis can be made by (1) the inoculation of cattle or laboratory animals with the blood, milk, or urine from cases of the disease; (2) isolation of the organisms in special media containing horse or rabbit serum; (3) microscopic examination of tissue sections stained with silver, or the demonstration of the organism in the blood or tissues under dark-field illumination; and (4) serologic examination of the blood from recovered cases. The spirochetes can most often be isolated from the milk sample. Usually the sera of cows bled at the onset of the infection fail to agglutinate or lyse suspensions of the spirochete, but two to four weeks later the reaction of the blood is positive in a dilution of 1:200 or higher.

The natural mode of transmission of the infection is not known. The organism is probably carried through the winter in the bovine with a chronic infection (nephritis) since it it readily destroyed by freezing temperatures.

[Little, Ralph B., and Baker, James A. *Leptospirosis in Cattle* J.A.V.M.A. 116:105-111 (Feb.) 1950.]

THE PATHOLOGY AND BACTERIOLOGY OF INFECTIOUS ATROPHIC RHINITIS IN SWINE. Investigation has shown conclusively that atrophic rhinitis is infectious, although the causative agent is still undetermined. Transmission occurs by contact or by instilling an emulsion of the damaged turbinate bones from early clinical cases of rhintis, if pigs are exposed shortly after birth. Pigs infected in this manner readily transmit the disease to litter mates. Feces and urine from infected swine, when instilled into the nasal passage of piglets, have failed to reproduce the disease. The critical age of susceptibility has not been determined, but increasing age is associated with an increase in resistance.

Atrophy of the turbinate bones is the most outstanding characteristic of the disease. The direct cause of the resorption of the bony tissue of the turbinates is still undetermined. Inflammation of the turbinates in swine (rhinitis) is commonly seen; yet, apart from this disease, severe atrophy is unknown. This points to the influence of a specific infectious agent. Observable vascular changes do not precede the disappearance of the bone, although they may follow the resorption. Some nutritional change is the most likely factor. The marked reduction in the depth of the bone is apparent. The loose connective tissue in the medullary area disappears, and with the removal of the earthy salts and the proliferation of the osteoblasts and fibrous tissue, a characteristic atrophy followed by an almost complete resorption of bone takes place.

Since an important function of the mucous membrane lining the turbinate and ethmoid bones is the arrest of bacteria and foreign matter, a rich and varied normal flora can be expected. Results of bacteriological examinations did not suggest that the primary etiological factor was bacterial in nature. No particular species of bacteria was isolated consistently.

Filtrated emulsions prepared from the mucous membrane and turbinate bones of acutely and chronically affected pigs were instilled into the nostrils of normal pigs. Negative results indicated that apparently a filterable virus is not a causative agent.

[Schofield, F. W. and Jones, T. L. *The Pathology and Bacteriology of Infectious Atrophic Rhinitis in Swine.* Jl. A.V.M.A., 875:120–123. (Feb.) 1950.]

FOX ENCEPHALITIS (INFECTIOUS CANINE HEPATITIS) IN THE DOG. Fox encephalitis is apparently quite widespread and appears to be a popular diagnosis at the present time. Confusion arises from the many descriptive names of the disease, which include encephalitis, fox encephalitis, canine fox encephalitis, infectious canine hepatitis, contagious canine hepatitis, enzootic fox encephalitis, epizootic fox encephalitis and canine endotheliitis.

Fox encephalitis in the dog is caused by a virus that is a distinct entity in itself, separate from the virus of Carré, but comparable to the Rubarth virus. It is a known fact that we have variations in the pathogenicity and potency of the fox encephalitis virus. All carriers of this virus are not known; there may be some of which we have no knowledge.

Clinical symptoms in the dog vary from those that occur in the fox. When this virus is injected experimentally into dogs, they promptly show an elevation of temperature varying from 103 to 106. This temperature is maintained from five to ten days. A purulent discharge appears in the eyes and nose; the dog is definitely sick and his coat becomes rough. About 75 percent of affected animals recover promptly. The other 20 to 25 percent succumb to the infection. These enumerated symptoms of fox encephalitis resemble the symptoms of distemper, and due to this fact there are many complicating factors which make a diagnosis extremely difficult. One of the striking differences between this disease and distemper is that some of the infected dogs remain on feed to within

24 to 48 hours prior to death. The progress of fox encephalitis is far more rapid than that of distemper. Dogs infected with the fox encephalitis virus are usually dead within two weeks or have recovered completely by the end of the third week. Young dogs appear to be more susceptible than adults and have a higher mortality rate.

On autopsy it is found that there are very few pathological lesions from which an accurate diagnosis can be made. Subendocardial hemorrhages of the heart appear to be the only primary diagnostic lesion.

The inclusion bodies of fox encephalitis have been observed or demonstrated in the endothelium of the liver, brain, adrenal glands, kidney and tonsil. A rapid diagnosis of fox encephalitis has been made by the injection of macerated brain or liver material from the suspect intraocularly in 0.5cc. quantities into the anterior chamber of the eye of a young dog or a young fox. A positive diagnosis can be made within a period of three days and confirmed by the finding of intranuclear inclusion bodies in the epithelium of the anterior chamber. The true incidence of this disease will be unknown until there can be some rapid method developed whereby the practicing veterinarian will be able to make a diagnosis of fox encephalitis without the use of detailed laboratory procedures.

The injection of fox encephalitis hyperimmune antiserum, used therapeutically, has proved to be effective when given in 1 cc. quantities for each five pounds of body weight of the suspected animal. Response to the therapeutic use of this material will occur within 12 to 24 hours. Attempts to develop an efficient vaccine from killed virus have not given satisfactory results. Extensive attempts to develop a serum-virus combination for vaccinating against fox encephalitis have shown this procedure to be highly dangerous because of the development of delayed infections.

[Chaddock, T. T., and Carlson, W. E. *Fox Encephalitis (Infectious Canine Hepatitis) in the Dog.* No. Am. Vet., 31:35-40. (Jan.) 1950.]

BRUCELLOSIS IN SWINE. Brucellosis of swine is a disease which has been recognized as a serious problem for many years but Brucella infections in swine are not always easily recognized by swine producers. Frequently the abortion rate in an infected herd is low and the disease in hogs is frequently self-limiting within the individual animal. Cross infections between swine, cattle and goats do occur although there is a specific species of Brucella for each of these animals. Man is susceptible to all three types of Brucella but the disease is not transmissible from man to man. Thus the control of the disease in man is largely dependent on the control of the disease in animals.

Brucellosis in swine is first manifested as a bacteremia in which the organisms may persist in the blood stream up to sixty or ninety days. During and following the bacteremic stage, the organisms may localize in the body. The points of localization are unpredictable since *Brucella suis* has been isolated from most, if not all, of the organs and tissues of infected swine. The organism is most commonly recovered from the various regional lymph nodes, spleen, uterus, testicle, accessory genital glands, bone marrow and the liver. The symptoms are quite varied as a result of the many different organs and tissues where the organisms may localize. The most common symptoms are abortion, sterility, orchitis, lameness, posterior paralysis and occasionally metritis and abscess formation of the extremities or other areas of the body. The incidence of abortion may vary from none in some herds to 50 or 80 per cent in other herds. Unobserved abortions may occur early in pregnancy.

Swine of all ages are susceptible, but most suckling pigs reach weaning age (8 to 12 weeks) as noninfected.

Brucellosis in swine is spread by direct contact or less often by contact with infectious material. The boar is commonly incriminated as an important means of spread.

The principal means of diagnosis is by the serum aggutination test. However,

Brucella may be isolated from swine which are negative to the blood test. Thus is is necessary to make the diagnosis on a herd rather than on an individual basis. As a rule of thumb, serum agglutination reactions below the 1:100 dilution are not considered indicative of brucellosis unless there are definite reactors at the 1:100 dilution, or higher, in the herd.

Attempts to produce immunity with strain 19 (*Brucella abortus*), heat killed strains of *Brucella suis,* and modified live strains of *Brucella suis* have failed to give satisfactory results. Chemotherapeutic agents have also proved of little value.

The author suggests two plans for control and eradication of swine brucellosis. Both plans are based on test and slaughter. The first plan is based on sale of the entire herd for slaughter, cleaning and disinfecting the premises, and replacement of the infected herd with clean animals. Periodic blood tests should be conducted on the newly purchased herd as a means of determining infection that may be resident on the premises. Early detection of these animals is important as brucellosis is primarily an animal to animal contact disease.

The second plan is based on test, segregation and delayed slaughter of the herd. The pigs from the infected animals are weaned and tested at eight weeks of age and the negative pigs are then isolated on clean premises. Thus good blood lines may be maintained and a clean herd may be salvaged from an infected one. The old herd is then sold to be slaughtered.

[Hutchings, L. M., *Brucellosis In Swine* J.A.V.M.A. 116:200–203. (March) 1950.]

ANTHELIN—A NEW COMPOUND FOR REMOVING TAPEWORMS AND ROUNDWORMS FROM DOGS. A limited study has been made of the taeniacidal and ascaricidal properties in dogs of a new synthetic anthelmintic, N-methyl-tetrahydro - methyl - nicotinate-p-carboxyphenyl stibonic acid (anthelin).

Previous work indicated that certain antimony compounds possessed some anthelmintic activity. On the basis of screening tests, anthelin was selected for evaluation.

Test dogs were confined in individual pens and given one dose of anthelin (4.69 mg./lb. of body weight) approximately 18 hours after a light meal (about half the regular meal). Food was withheld until four hours after dosing, although free access to water was allowed. In cases where purging did not occur in an animal within 60 minutes after dosing, 1 to 1.5 cc. of lentin was administered subcutaneously. The worms eliminated in the tests were identified as *Dipylidium caninum, Taenia* of unidentified species, *Toxocara canis,* and *Toxascaris leonina.* Elimination of the worms generally occurred within three hours after dosing. All animals were normal in apperance and action 24 hours after dosing.

On postmortem examination, the intestinal tracts of all the animals were normal in appearance with the exception of two, the tracts of which showed moderate irritation, and one which showed severe irritation. The stomachs of all animals were normal.

At a dose of 4.69 mg. per lb. of body weight, anthelin removed 97 percent of the tapeworms (*Dipylidium caninum* and *Taenia sp.*) and 91 percent of the ascarids (*Toxocara canis and Toxascaris leonina*) in ten dogs.

Dogs show good tolerance to twice the recommended dose of anthelin administered on three consecutive days.

[Kartsonis, P. L., and Austin, James A. Anthelin—*A New Compound for Removing Tapeworms and Roundworms from Dogs.* Jl. A.V.M.A., 877:301-303. (April) 1950.]

ISOLATION OF THE VIRUS OF NEWCASTLE DISEASE FROM HUMAN BEINGS. Two naturally occurring cases of conjunctivitis thought to be due to the virus of Newcastle disease are re-

ported. One case occurred in a broiler plant operator on whose premises Newcastle disease was in progress; and the second case was a veterinary student who, in the course of his clinical work, autopsied three chickens affected with acute cases of Newcastle disease. In the case of the broiler plant operator, the virus identification was confirmed by hemagglutination hemagglutination inhibition, serum neutralization techniques, and antibody response in chickens.

In the case of the student the virus identification was confirmed by hemagglutination, hemagglutination inhibition, and serum neutralization.

In addition to the above, virus sent to the Pathological Division, U.S. Bureau of Animal Industry was identified as Newcastle Virus.

This is believed to be the first reported case in the United States in which the virus of Newcastle disease was isolated from man.

[Ingalls, W. L. and Mahoney, A., Isolation of the Virus of Newcastle Disease from Human Beings. Am. Jl. Publ. Health, 39: 737-740 1949.]

Q FEVER. EXPERIMENTAL Q FEVER IN CATTLE. Q fever infection in lactating cows has been produced consistently by inoculation of *Rickettsia Coxiella burneti* via the lactiferous duct. Rickettsiae were found in the milk of these cows for long periods of time, in some cases for over 200 days after inoculation. Exposure of the mammary gland to massive doses of a yolk-sac culture of the organism excited an acute mastitis accompanied by a marked systemic reaction. These symptoms appeared to be of brief duration and recovery was spontaneous, yet rickettsiae continued to be shed in the milk. In animals showing these symptoms, rickettsiae were recovered from the blood during the acute period and from numerous other tissues obtained from cows sacrificed at various intervals up to 63 days after inoculation.

Q fever rickettsiae were recovered from the feces of calves feeding on infected milk. However, infection by this method was not definitely proved.

Experiments involving attempts to infect cattle by other routes of inoculation and by infestation with infected ticks (*Otobius megnini*) are described.

[Bell, E. J., Parker, R. R. and Stoenner, H. G., Q Fever. Experimental Q Fever in Cattle. Am. Jl. Publ. Health, 39: 478-484 1949.]

Aureomycin

A drug that may prove to be the means for dairymen to prevent over $100,000,000 from slipping through their fingers each year as a result of a widespread disease of dairy cows is now available. Aureomycin, the "miracle drug" of 1949 in human medicine, now can be used for treating the most costly disease of the dairy industry, mastitis, which is said to affect about one-third of the nation's 30 million dairy cows each year. Mastitis lowers the milk yield of an infected cow; it may render the milk unsalable. Frequently, the cow goes to an untimely end at the slaughter house, or dies of septicemia started by mastitis.

Veterinarians have revealed that aureomycin has passed certain field tests in treating cows with mastitis and that it will be available immediately as an ointment in specially designed tubes for direct infusion into the udders of the infected cows. Aureomycin has been proved superior to penicillin, heretofore the most effective mastitis treatment, because it cures cases of mastitis caused by a wider variety of bacteria. Aureomycin, in one dose, will clear up staphylococcal mastitis about 70 percent of the time and will cure more than 90 percent of cases caused by streptococci. The exceptional effectiveness of one dose is achieved by blending 200 mg. of a finely powdered (micronized) aureomycin into a special base that is less dense than milk. Thus, the lighter-than-milk ointment floats up through the milk and spreads to all parts of the udder. In stubborn cases, a second application of aureomycin ointment after 48 hours has usually been sufficient to stamp out the infection.