Pseudorabies in the Dog and Cat

John D. Boucher  
_Iowa State University_

George Beran  
_Iowa State University_

Follow this and additional works at: [http://lib.dr.iastate.edu/iowastate_veterinarian](http://lib.dr.iastate.edu/iowastate_veterinarian)  
Part of the [Small or Companion Animal Medicine Commons](https://lib.dr.iastate.edu/smallcompanionanimalmedicines), and the [Veterinary Pathology and Pathobiology Commons](https://lib.dr.iastate.edu/veterinarypathology)

Recommended Citation

Boucher, John D. and Beran, George (1977) "Pseudorabies in the Dog and Cat," _Iowa State University Veterinarian_ : Vol. 39 : Iss. 1 ,  
Article 6.  
Available at: [http://lib.dr.iastate.edu/iowastate_veterinarian/vol39/iss1/6](http://lib.dr.iastate.edu/iowastate_veterinarian/vol39/iss1/6)

This Article is brought to you for free and open access by the Student Publications at Iowa State University Digital Repository. It has been accepted for inclusion in Iowa State University Veterinarian by an authorized editor of Iowa State University Digital Repository. For more information, please contact digirep@iastate.edu.
**Pseudorabies in the Dog and Cat**

by

John D. Boucher*  
and  
Dr. George Berant†

**Introduction**

Pseudorabies, also called Aujesky’s Disease, Mad Itch, and Infectious Bulbar Paralysis, is a viral disease which primarily affects pigs. It occurs in a wide variety of domestic and wild animals and birds, but not in apes, reptiles, or insects. The natural viral reservoir is swine, in which it produces high mortality in suckling pigs, relatively low mortality in older pigs, and may produce carriers showing few or no symptoms.

Pseudorabies has been reported throughout the world. References in the literature indicate that the disease was present in the United States as early as 1813. In 1962, severe outbreaks occurred on several farms in Indiana; since that time, it has been recognized in other areas of the nation where swine are raised. Due to increased awareness on the part of veterinarians and swine producers or to an actual increased incidence, the disease is being reported with greater frequency throughout the United States.

It is essential that large and small animal practitioners be cognizant of pseudorabies in small animals. In a recent pseudorabies outbreak in central Iowa, small animals contracted the disease and died on 12 of the 16 farms involved. On 4 of these farms the small animal cases occurred before the disease was recognized in the swine. In many instances, such as the clinical case reported here, the death of a dog or cat on a farm has been the only indication that the virus was present. Therefore, familiarity with the disease in small animals may be an important aid in diagnosing pseudorabies in swine.

**Clinical Case**

On September 26, 1976, a veterinarian in Gainesville, Missouri, referred a dog to the Iowa State University Clinic. It was the fourth dog he had examined for a farmer client during the past 48 hours. The first three had exhibited sudden profuse salivation followed rapidly by death. The fourth was now showing similar clinical signs.

The dog, an 18 month old female of mixed breeding, arrived at the ISU Veterinary Clinic in clonic spasms and unable to rise. Her pupils were constricted and she was salivating profusely. She moved her jaws in a chomping motion, snapped at hands held near her head, and exhibited hysteria. Her temperature was 107°; the heart and respiratory rates were elevated. While being examined, the dog vomited blood and mucus. These clinical signs had become progressively worse.

A preliminary diagnosis of organophosphate toxicity was made and the dog was given fluids, bicarbonate, Solu-Delta-Cortef®, Valium®, and atropine, and placed in a cold water bath. The bath succeeded in bringing the temperature down; the atropine injection resulted in dilated pupils and alleviated the excessive salivation. The other symptoms, the spasms, chomping, and hysteria, continued and, four hours after initial treatment, 2 cc of Myothesia® were given. The dog died 10 minutes later and was sent to post mortem for necropsy.

The following day, another dog and cat from the same farm were sent for necropsy.

*I. Boucher is a senior student in veterinary medicine, Iowa State University.
†Dr. Beran is Professor of Microbiology, College of Veterinary Medicine, Iowa State University.
No significant gross lesions were found in any of the three animals. Liver, lung, spleen, and mesenteric lymph nodes from all three animals were saved for bacterial culture. Stomach contents and brains were saved for organophosphate, chlorinated hydrocarbon, rabies, and pseudorabies tests. No significant pathogenic microorganisms were cultured from the bacteriological specimens, but pseudorabies virus was isolated from the brain tissues.

In a telephone conversation with the owner, it was learned that all five dogs were kept outdoors, but two were kept tied. He also remarked that he had sprayed his house two weeks previously with chlordane. Upon further questioning, he noted no problems in his swine herd. However, he added that he had fed the dogs some dead pigs, indicating some health problems. The owner's veterinarian was asked to collect blood samples from the pigs for testing at the Iowa State University Diagnostic Laboratory. The swine tested were found to have high pseudorabies titers, indicating an active infection was present in the swine herd.

**Etiology**

The pseudorabies virus is a DNA virus of the Herpes group. It may survive in nature from 14 to 48 days or longer depending upon temperature and humidity. Putrefaction will destroy the virus in approximately 11 days and it is killed instantly by 1% sodium hydroxide (NaOH) or by heating to 100°C. The virus will replicate and produce intranuclear inclusions in a wide variety of mammalian cell cultures, providing a microscopic method for its detection. Cells of swine, rabbit, or dog kidneys are most sensitive and the virus produces an easily recognizable cytopathic effect.

**Transmission**

The probable sources of pseudorabies to dogs and cats are ingestion of dead and infected pigs, cows, or rats, or being bitten or slashed by infected wild or domestic swine. The amount of infected material which produces infection by the oral route seems to vary. In an experiment performed at Iowa State University, dogs were fed 2-3 grams each of liver from which pseudorabies had been isolated, but failed to develop the disease. Another Iowa State research project involved feeding a dog increasingly larger amounts of infected material but also failed to produce the disease. When a suspension of the same tissues was inoculated subcutaneously, the dog developed pseudorabies and died. In a case reported to the ISU Clinic, however, a dog belonging to a veterinarian was infected and died after licking at several blood spots following a post mortem examination of an infected pig.

Possible causes of such variable susceptibility include age, varying virulence of the different strains of virus, acquired or maternal resistance, or other unknown factors. These variables are presently being researched.

The possibility that infected dogs and cats may shed the virus and be involved in the transmission of the virus is also under investigation. There is epidemiological evidence to support this hypothesis, but it has not yet been confirmed by laboratory studies.

**Pathogenesis**

After gaining entry through a skin abrasion, the pseudorabies virus invades the local peripheral nerves and passes along them toward the central nervous system. It initially invades the spinal ganglia, then passes into the spinal cord via the dorsal horns. The virus continues to move centrally to the medulla oblongata or, in some cases, more rostrally in the brain stem. In some animals, experimental inoculation has been followed by transitory viremia and secondary infection of other tissues. From each new location it also moves centrally along the afferent nerves.

An acute serous inflammation may develop at the point of inoculation but direct tissue reaction to the virus is negligible. Self inflicted trauma does result in severe inflammation and hemorrhagic necrosis. Lymphocytes collect along the infected nerves and within the infected ganglion; the neurons degenerate and necrose. A few of these neurons and some mesenchymal cells develop intranuclear inclusion bodies. Foci of neuronal
degeneration and inflammation within the brain and spinal cord, particularly in the ganglia, elicit a severe itching sensation. Death is probably due to neuronal injury in the medulla resulting in respiratory failure.

The pathogenesis of pseudorabies following exposure by ingestion has not been clearly elucidated. In swine and perhaps in other animals as well, the virus travels to the medulla and pons via the 1st (olfactory), 5th (trigeminal) and 9th (glossopharyngeal) cranial nerves. Many dogs have been reported to display pruritis of the area of the head after oral ingestion.

Clinical Signs
The incubation period for pseudorabies in both the dog and cat is 72-96 hours. The most characteristic clinical manifestations are intense localized pruritis with scratching or chewing to the point of self mutilation and sudden death. Of the many cases reviewed in the literature, nearly all infected dogs and cats displayed pruritis and self mutilation. However, in a few cases, such as the clinical case reported here, pruritis was not observed; perhaps it did not occur or it occurred early in the course of the disease, before the animal was professionally observed. This sign will not always be seen and its absence should not preclude consideration of pseudorabies in the differential diagnosis.

Other clinical signs include high temperature, unresponsiveness to owners, refusal to eat or drink, profuse salivation, vomiting blood and mucus, clonic spasms, incoordination, convulsions, collapse, coma and death. All or only some of these symptoms may occur in any given case.

The disease is considered to be nearly 100% fatal in small animals. One case of pseudorabies antibodies in a clinically normal cat has been reported. In any case of sudden death in a farm dog or cat, pseudorabies should be included in the differential diagnosis.

Diagnosis
A presumptive diagnosis of pseudorabies can be made whenever the classical clinical signs of pruritis, salivation, and sudden death occur, especially in a farm dog or one exposed to farm animals. The diagnosis can be confirmed by the subcutaneous inoculation of mice or a rabbit with a suspension of tissues from the suspect animal. Neural tissue from the site of intense itching or sections of spinal cord nearest the affected site are the tissues of choice. Positive material causes intense local pruritis 4-6 days post inoculation, followed by death. In the laboratory several methods are commonly used for confirmation of pseudorabies. One of the most important is histological examination of the brain and spinal cord for typical lesions and the presence of intranuclear inclusion bodies. Other means of identification are virus isolation in cell cultures and typing by fluorescent antibody or virus neutralization tests.

It is important to differentiate pseudorabies from the furious form of rabies. The shorter course, lack of vicious attacks on other animals and man and the intolerable pruritis are important differential characteristics which indicate pseudorabies infection.

Prevention and Treatment
Although vaccines for pseudorabies have been developed and are used for disease prevention in several foreign countries, none are yet available in the United States. Prevention must, therefore, be directed toward avoiding contact with infected animals. Dogs and cats should not be allowed to eat any dead pigs, nor should they have access to the live pigs.

Symptomatic treatment of animals with severe pruritis and pain, accompanied by convulsions is indicated, although death is almost certain. General anesthesia is the treatment of choice.

REFERENCES
Dow, C., (1963), Aujeszky's Disease In The Dog And The Cat, Veterinary Record, Vol. 75, No. 4, pp. 1099-1101.
Hutchcroft, T., Beran, G., Pseudorabies In Cattle, Iowa State University Veterinarian, Vol. 37, No. 3, p. 85.