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Encephalitis in the Silver Fox

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The history stated that the gelding was slightly lame in all ankles, and that “shin bucks” (a horseman's term meaning periostitis of the metacarpal bones) were present. Upon examination it was found that the principal trouble was chronic tendinitis of the flexor tendons of both front legs. No treatment was attempted for several days because of the poor condition of the animal. During this time one ounce of Fowler's solution was administered daily. Later, a fecal examination indicated severe strongylidosis and a few ascarids.

It was decided to fire the flexor tendons. The animal was given three ounces of chloral hydrate intravenously and was cast with very little difficulty. The hair was clipped over the fetlock joints and volar aspect of the metacarpal bones of both forelegs. The area was anesthetized by infiltration with a two percent procaine solution. Three rows of point firings were made on each leg over the flexor tendons, using the electric cautery. A biniodide blister (1-10) was applied, and cotton was bandaged into place over the firing wounds.

The next day the horse showed mild colic. Equal parts of aromatic spirits of ammonia and turpentine (one and one-half ounces) were administered per orum. Later, the horse lapsed into a coma and was given strychnine sulfate (one-quarter grain) sub-cutaneously. The animal died the next afternoon without regaining consciousness.

Necropsy

On necropsy examination soft yellow thrombi which were caused by strongyloides larvae were found in the branches of the anterior mesenteric artery. An acute hemorrhagic cecitis was present. The right dorsal colon was impacted and the entire colon was filled with hay. Lesions of a septicemia were present. These lesions included epicardial and endocardial hemorrhages, pulmonary hyperemia and pulmonary hemorrhage. No bacterial cultures were made. Gasterophilus intestinalis larvae were found in the stomach and Gasterophilus nasalis larvae in the duodenum, which accounted, at least in part, for the poor condition of the animal.

Since this animal was infected with strongyloides it was thought that the colic might be thrombo-embolic and brought about by casting of the animal. This could not be established at necropsy as no emboli were found. However, very minute emboli might have showered the blood stream producing the acute hemorrhagic cecitis and, by lodging in the cerebrum, the coma.

Since the patient was anesthetized with chloral hydrate no water was given for a short period of time. It is possible that this lack of water might have caused a general dehydration of the animal which by drawing water from the colon might have produced the impaction. It is also possible that an atonic colon was responsible for the impaction. The exact cause of the atony would be hard to establish unless one would incriminate the anesthesia. This would almost be unlikely as chloral hydrate is not a smooth muscle depressant. In any case, since the animal had an abundance of feed in the digestive tract, perhaps purging the patient prior to anesthesia might have been desirable. Since no cultures were made one should not exclude a septicemia as a possible cause of death.

This case is an excellent example of the dangers attending surgical procedures upon animals that are devitalized from any cause. Treatment to remove the gastro-intestinal parasites, followed by several weeks of good feeding and tonics might have prevented this fatality. This was suggested to the owner when the horse was brought to the clinic, but he was anxious to have the horse fired as soon as possible so training could be started.

—V. D. Ludwig, fall '43

Encephalitis in the Silver Fox.

To the silver fox industry, encephalitis is a disease of great importance. It seems to occur only in foxes raised in captivity and is epizootic in character. In the fall and winter months the foxes would be...
which will be pelted in the near future are put in one pen, and it is this grouping together which seems to bring on this dreaded disease.

**Filtrable Virus**

The etiological agent is a filtrable virus which is small enough to pass through a Berkefeld N filter. The virus can be found in the brain, blood, spleen, upper respiratory tract and spinal cord. The reason the virus becomes pathogenic when large groups of foxes are put together is not positively known. Direct contact from quarreling and the cannibalistic tendency of a fox, are thought to be causes as well as eating and drinking from the same containers. The portals of entry are the respiratory tract, digestive tract, and skin wounds.

Adult foxes (over one year old) are twice as resistant as are the younger foxes. When the disease occurs in a large group of mixed-aged foxes, the mortality rate is about 15-20 percent. Experimental inoculation shows about 80 percent mortality in foxes below the age of 6 months and approximately 15-20 percent mortality in adult foxes.

**Immunity**

A high degree of active immunity is produced by recovery from this disease. Hyperimmune serum seems to have some inhibitory action upon the virus; however, it must be noted that this action is only temporary. The mortality rate has been reduced to one-half when virus attenuated with sodium ricinoleate has been administered.

Intranuclear inclusions in the endothelial cells of the central nervous system, the ependymal cells and the epithelial cells of the upper respiratory tract aid greatly in diagnosis. Also the meningeal infiltration of polymophonuclear leucocytes and slight hemorrhages in the medulla and spinal cord are significant. The feces usually contain large amounts of mucin which may be streaked with blood. At one time epizootic fox encephalitis was classed with the central nervous system type of distemper, but it has been proved that the two are entirely different.

The prolonged symptoms are seldom seen, because of the acuteness of the disease. In most cases an apparently normal fox will suddenly start staggering, and go into violent convulsions with death occurring in ten to fifteen minutes. When the symptoms occur a little slower, the fox will show partial anorexia, hyperexcitability, and will gradually go into a lethargic state resulting in paralysis, hallucination, coma, and death.

Dogs are quite susceptible to this disease while coyotes have been found to be as susceptible as foxes. The gray fox, mink, rabbit, white rat, squirrel, guinea pig, cat, ferret, and sheep are resistant.

(The author is greatly indebted to Dr. R. G. Green of the Bacteriology and Immunology Department of the University of Minnesota, and his co-workers who have done extensive research on this subject.)

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**Foot-Rot in Cattle.** A six-month old Hereford bull was brought to the Stange Memorial Clinic on February 2, 1943. The history revealed that the animal had started to limp on its left front foot about two weeks previous and that this symptom had progressively increased until the animal refused to place any weight on the leg.

The affected foot showed extensive swelling between and above the digits. At the proximal end of the interdigital space was a small fistula from which discharged a purulent exudate containing synovial fluid. Excoriation of the epithelium around this area had taken place. A diagnosis of foot-rot with a possible joint involvement was made.

**Operation Performed**

The bull was restrained on the operating table and the claws and lower part of the left front leg were thoroughly scrubbed. The area was shaved and tincture of iodine applied. Two percent procaine was used to infiltrate the tissue surrounding the