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# CEREBROSPINAL PARELAPHOSTRONGYLOSIS IN FALLOW DEER[1]

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*Abstract:* Neurologic disease attributed to infection by meningeal worm (*Parelaphostrongylus tenuis*) was diagnosed in seven fallow deer (*Dama dama*) from the Land Between The Lakes region of Kentucky. Afflicted deer had paresis or paralysis of the hindquarters which quickly progressed to tetraplegia. Gross and microscopic cerebrospinal lesions were similar to those previously reported and consisted mainly of nonsuppurative meningitis and radiculitis, focal granulomas on the surface of the spinal cord, and nonselective malacia and glial scarring in brain and spinal cord. Living *P. tenuis* were recovered from brain or spinal cord in two deer, and degenerating nematodes were found in four. Possible epizootiologic relationships between the parasite, fallow deer and white-tailed deer (*Odocoileus virginianus*) are discussed.

## INTRODUCTION

White-tailed deer (*Odocoileus virginianus*) are considered usual hosts for meningeal worm, *Parelaphostrongylus tenuis*, and rarely suffer overt disease from this neurotropic nematode. In contradistinction, many other wild[2-4,6,9,11,14,15] and domestic[1,10,12,16] ungulates develop severe neurologic dysfunction when these parasites migrate through the cental nervous system. Spontaneous neurologic disease caused by *P. tenuis* was reported in a captive fallow deer (*Dama dama*) held in an enclosure with white-tailed deer in Georgia.[9] The clinical and pathologic findings associated with naturally-occurring cerebrospinal parelaphostrongylosis in fallow deer of the Land Between The Lakes area of Lyon and Trigg Counties, Kentucky, are reported herein.

## HISTORY

Land Between The Lakes (LBL) is a 68,800 ha strip of land under single ownership separating two large reservoirs (Kentucky Lake and Lake Barkley) in southwestern Kentucky and northwestern Tennessee (lat. 37°, long. 88°). The area is 9.7 to 12.9 km wide and 64 km long and has a shoreline boundary on three sides. Although the land has poor agricultual qualities, it is used extensively for outdoor recreation. A detailed description of the wildlife habitat, forest resources and hydrologic condition has been published.[5] Presently, the Tennessee Valley Authority manages the properties as a national demonstration area for conservation, recreation, and education.

Among the unique faunal features of the LBL is an established herd of fallow

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deer which intermingles with white-tailed deer. Approximately 20 fallow deer were introduced in 1918 by a private landowner. The fallow deer population increased during the ensuing years and presently consists of an estimated 600 animals. The population is centered around the original release site in an 8,100 ha region which includes the 2,000 ha Environmental Education Center (EEC).

White-tailed deer were relocated by the Kentucky Department of Fish and Wildlife Resources in the mid 1930's. Early white-tailed deer populations were depressed by poaching and an undiagnosed epizootic which occurred in 1949.[7] The population of white-tailed deer has increased in recent years, and managed sport hunting for white-tailed and fallow deer has been allowed since the mid 1950's. The present white-tailed deer density in the EEC is about 2.5 deer per 40 ha, while the fallow deer density probably is 3.5. Overall deer density recently was estimated by LBL biologists at 3.3 deer per 40 ha in regions around the EEC with white-tailed deer prevailing.

As a biologist for the Tennessee Valley Authority (TVA), one of the authors (Smith) often has received reports of fallow deer which showed locomotor difficulties, and several opportunities arose to capture afflicted animals on the EEC and surrounding areas for postmortem examination.

## MATERIALS AND METHODS

Living fallow deer were in moribund condition, and were difficult to assess neurologically. Tests for reflexes such as flexor (withdrawal), patellar, palpebral, pupillary and righting were attempted. The response of deer to loud noises, movements and general handling also were noted. Blood samples were collected in EDTA for hematologic studies. Cerebrospinal fluid was obtained by centesis with an 18 gauge spinal needle at atlanto-occipital junction. Pressure readings were measured with a manometer while the deer was in lateral recumbency.

Deer were killed humanely and detailed necropsies performed. Brains and spinal cords were removed and examined as previously described.[11] Tissue samples were taken from cerebrum, diencephalon, midbrain, cerebellum, medulla, spinal cord and visceral organs. Spinal cord was sampled at each spinal nerve root in five animals. Tissues were embedded in paraffin, sectioned at 7 $\mu$m and stained with hematoxylin and eosin.

Samples of minced lung and feces were examined by the Baermann technique for protostrongylid larvae. In addition, direct smears of tracheal mucus were examined under magnification (100 X).

## RESULTS

Between October, 1973 and August, 1976, seven fallow deer were examined because of neurologic impairment. Two deer were unable to rise from lateral recumbency when discovered. The remaining five had paresis or paralysis of the hindquarters. The latter animals were captured easily by hand, and each became tetraplegic and moribund during 3 to 5 days of captivity. Six of the afflicted deer were males, and ages ranged from 0.5 to 2.5 years. Deer were found in March, April, May, August, September and October.

Terminal illness was characterized by lateral recumbency and occasional thrashing movements of the legs and head. Flexor reflex usually was present in front and rear limbs, and most deer would raise their head in response to painful stimuli to the feet. One deer had rigid extension of the front legs. Although animals were not as alert as healthy deer, responses were made to visual and auditory stimuli.

Blood examinations were performed on three deer. Packed cell volumes ranged from 39 to 50% and leukocyte counts ranged from 4,800 to 10,280 cells/mm$^3$. Absolute values for various leukocytes were: neutrophils 2,745 to 5,242; lymphocytes 480 to 3,906; monocytes 192 to 925; and eosinophils 0 to 211 cells/mm$^3$. Cerebrospinal fluid analyses of three deer are presented in Table 1.

Five of the seven deer had moderate to heavy burdens of ticks (*Amblyomma americanum* and *Dermacentor variabilis*). Abrasions and contusions of the periorbital tissue were found on the recumbent side in four animals and two deer had subcutaneous hemorrhages overlying bony prominences. Pulmonary emphysema and inhalation of ruminal contents were found on three occasions. *Setaria yehi* were present in low numbers (2 to 3 worms) in the abdominal cavities of two deer. Organized plaques of fibrin attributed to these helminths were observed on the visceral pleura and liver capsule of three animals. Distension of the urinary bladder was found in three deer. General physical condition was poor in four deer, fair in two and good in one.

Gross cerebral lesions attributed to *P. tenuis* were subtle and consisted of slightly thickened and cloudy leptomeninges. Exceptions to this were massive subdural hemorrhages present in two deer as the result of self-inflicted trauma. Pathologic alterations were more common in the spinal cord and were best seen with a dissecting microscope. Slightly raised nodules, randomly distributed on the surface of the cord, were the most frequent finding. These nodules had a yellow to pink color, a caseous texture and varied in shape from small round lesions 2 to 3 mm in diameter to tracts up to 15 mm long. Such areas primarily were in the cervical and mid-lumbar region, but a few were found in the thoracic cord and one was seen in the cauda equina. Focal accumulations of green caseous exudate also were observed beneath the dura and around spinal nerve roots in several deer. Myelomalacia with cavitation was found throughout the lumbar region of one deer.

Brains of six deer had histopathologic lesions consisting of widely scattered foci of malacia, glial scarring, perivascular cuffing with mononuclear cells, microhemorrhages and, rarely, small granulomas. Spinal cord damage resembled that reported by Kistner *et al.*[9] in features such as mineralization and mononuclear cell infiltration beneath the dura and around spinal nerve roots, indiscriminate leuko- and myelomalacia, and perivascular cuffing. The small nodules observed on the surface of the spinal cord in the present cases were granulomatous accumulations of large and small mononuclear cells which often had germinal centers similar to lymph nodes. Four deer had cross-sections of dead nematodes contained within these nodules. Lesions were distributed throughout all regions of the spinal cord but were particularly severe in the cervical and lumbar areas.

Viable *P. tenuis* were recovered from only two deer. A male fawn had one worm in the cerebral meninges near the dorsal sagittal sinus, another in the internal capsule and two in the spinal

TABLE 1. Cerebrospinal fluid (CSF) values for three fallow deer, **Dama dama**, with cerebrospinal parelaphostrongylosis.

| Animal No. | CSF Pressure (mm) | Protein (mg%) | RBC/mm³ | WBC/mm³ | Lymph (%) | Neutr (%) | Mono (%) | Eosino (%) |
|---|---|---|---|---|---|---|---|---|
| 2 | Not measured | 112 | 250 | 10 | 0 | 100 | 0 | 0 |
| 4 | 170 | 94 | 54 | 10 | 100 | 0 | 0 | 0 |
| 5 | 200 | 100 | 25 | 1092 | 9 | 0 | 1 | 90 |

cord (T-3, L-4). An adult male deer had one immature *P. tenuis* in the ventral white matter at the thoraco-lumbar junction of the spinal cord. In addition, the aforementioned degenerating nematodes were discovered in four animals on histopathologic examination. Protostrongylid eggs or larvae were not found in fresh lung and feces or upon histologic examination of the cerebral meninges and lungs.

## DISCUSSION

The effect of *P. tenuis* on the LBL's fallow deer population has been considerably less devastating than that reported for other susceptible ruminants introduced elsewhere.[3,6,11,15] Perhaps there is a low prevalence and intensity of *P. tenuis* infection in resident white-tailed deer. In a previous study on the LBL, 2 of 5 white-tailed deer harbored single specimens of *P. tenuis*.[13] Differences in food habits or habitat preferences between white-tailed and fallow deer are considerations,[8] although such have not been noted by resident biologists. A third possibility is a sex-related difference in susceptibility. To date, 7 of 8 fallow deer (including previous report[9]) afflicted with neurologic disease have been males. Whether this unbalanced sex ratio occurred by chance is unknown (the EEC herd has 63 males/100 females). Should there be an increased exposure to or predilection for disease in male fallow deer, the reproductive potential probably would be reduced much less than vice versa. A fourth contingency is that fallow deer have some degree of innate resistance to invasion by the parasite.

We concur with the previous authors[9] in that fallow deer are not capable definitive hosts for meningeal worm since: (1) infected deer were moribund, (2) helminths found were immature or dead, and (3) protostrongylid larvae or eggs were not evident in meninges, lungs, or feces. Clinical, pathologic, and parasitologic features of *P. tenuis* infection in these fallow deer were similar to those reported previously for this species and other susceptible ruminants. In view of the game management problems meningeal worm has caused in many big game populations, *P. tenuis* should be considered a potential major mortality factor for fallow deer which share a common range with infected white-tailed deer.

## LITERATURE CITED

1. ALDEN, C., F. WOODSON, R. MOHAN and S. MILLER. 1975. Cerebrospinal nematodiasis in sheep. J. Am. vet. med. Ass. 166: 784-786.
2. ANDERSON, R. C. 1965. An examination of wild moose exhibiting neurologic signs, in Ontario. Can. J. Zool. 43: 635-639.
3. ————. 1971. Neurologic disease in reindeer (*Rangifer tarandus tarandus*) introduced into Ontario. Can. J. Zool. 49: 159-166.
4. ————, M. W. LANKESTER and U. R. STRELIVE. 1966. Futher experimental studies of *Pneumostrongylus tenuis* in cervids. Can. J. Zool. 44: 851-861.
5. BURBANK, J. H. and R. D. SMITH. 1966. Wildlife habitat, forest resource, and hydrologic condition inventory at Land Between the Lakes. Proc. Southeastern Assoc. Game and Fish Commissioners. 20: 6-14.
6. CARPENTER, J. W., H. E. JORDAN and B. C. WARD. 1973. Neurologic disease in wapiti naturally infected with meningeal worms. J. Wildl. Dis. 9: 148-153.
7. HAYES, F. A. and ANNIE K. PRESTWOOD. 1969. Some considerations for diseases and parasites of white-tailed deer in southeastern United States. Proc. Symp. White-tailed Deer in the Southern Forest Habitat. Nacogdoches, Texas. pp. 32-36.

8. KEARNEY, S. R. and F. F. GILBERT. 1976. Habitat use by white-tailed deer and moose on sympatric range. J. Wildl. Manage. 40: 645-657.

9. KISTNER, T. P., G. R. JOHNSON and G. A. RILLING. 1977. Naturally cccurring neurologic disease in a fallow deer infected with meningeal worms. J. Wildl. Dis. 13: 55-58.

10. MAYHEW, I. G., A. deLAHUNTA, J. R. GEORGI and D. G. ASPROS. 1976. Naturally occurring cerebrospinal parelaphostrongylosis. Cornell Vet. 66: 56-72.

11. NETTLES, V. F., ANNIE K. PRESTWOOD, R. G. NICHOLS and C. J. WHITEHEAD. 1977. Meningeal worm-induced neurologic disease in black-tailed deer. J. Wildl. Dis. 13: In Press.

12. NIELSEN, S. W. and J. AFTOSMIS. 1964. Spinal nematodiasis in two sheep. J. Am. vet. med. Ass. 144: 155-158.

13. PRESTWOOD, ANNIE K. and J. F. SMITH. 1969. Distribution of meningeal worm (*Pneumostrongylus tenuis*) in deer in the southeastern United States. J. Parasit. 55: 720-725.

14. SMITH, H. J., R. M. ARCHIBALD and A. H. CORNER. 1964. Elaphostrongylosis in maritime moose and deer. Can. Vet. J. 5: 287-296.

15. TRAINER, D. O. 1973. Caribou mortality due to the meningeal worm (*Parelaphostrongylus tenuis*). J. Wildl. Dis. 9: 376-378.

16. WHITLOCK, J. H. 1952. Neurofilariosis, a paralytic disease of sheep: II. *Neurofilaria cornellensis*, n. g., n. sp. (Nematoda, Filarioidea), a new nematode parasite from the spinal cord of sheep. Cornell Vet. 42: 125-132.

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