Degenerative myelopathy in a family of Siberian Husky dogs—P. Bichsel, DVM, and M. Van de Velde, DVM, Institute of Comparative Neurology, University of Berne, PO Box 2735, CH-3001 Berne, Switzerland, J. Lang, DVM, Small Animal Clinic, University of Berne, PO Box 2735, CH-3001 Berne, Switzerland, S. Kull-Hächler, DVM, Bolligen, Switzerland

Twenty adult Siberian Husky dogs in a breeding colony were bred for sleigh racing and were kept in large kennels. Breeding stock had been obtained from several unrelated sources. Thirteen years earlier, a female was imported from Canada and she produced 3 litters (16 pups) from matings with 2 males from outside the colony. Of these 16 pups, 1 male and 2 females remained in the colony but have not been used for breeding. The other 13 pups were sold to various people. Two 12-year-old littermates from this group of 13 as well as their 14-year-old dam are the subject of this report. In 2 of the 3 affected dogs, a neurologic examination was done, and it included examination of CSF, plain radiography of the spine, and contrast myelography. All 3 dogs were euthanized with pentobarbital and subjected to postmortem examination. Representative specimens of extraneural tissues as well as brain and spinal cord were fixed in formalin and processed for routine histologic examination.

The 12-year-old male had been successfully treated for primary epilepsy since the age of 3 years. Difficulties in walking were first noticed at the age of 11 years. The attending veterinarian observed ataxia in the hindlimbs, which was unaffected by antiphlogistic treatment. The signs became progressively worse and urinary incontinence developed. About 1 year after the onset of signs, the dog was euthanized and submitted to us for pathologic examination.

The 12-year-old female littermate had a short episode of stiffness in the pelvic limbs and pain in the sacral area at the age of 5 years. These signs were attributed to trauma that developed during a race and were successfully treated with antiphlogistic drugs. Around the age of 11 years, the owner noticed intermittent hindpaw dragging. Months later, difficult defecation and marked atrophy of the pelvic and truncal musculature became evident. The dog had severe shuffling hindlimb ataxia, with extreme flexion of the tarsal joints. Pain sensation was intact.

Fig 1—Cross section at the level of the thoracic spinal cord. (Fissura ventralis depicted by arrow). Marked spongy appearance of the ventral columns. H&E stain; x 40.

Fig 2—Thoracic spinal cord: cluster of macrophages (arrow). H&E stain; x 250.

Fig 3—Thoracic spinal cord: swollen axon (arrow). H&E stain; x 250.
Spinal reflexes and postural reactions could not be examined because of the dog's aggressiveness. Plain radiographs of the spine were unremarkable. Myelographically, there was neither block nor deviation of the contrast medium in the subarachnoid space around the spinal cord. The spinal fluid was clear, contained 9 cells/mm³ (86% lymphocytes, 12% monocytes, 2% neutrophil granulocytes) and had a slight increase in protein content, as judged by the Pandy reaction. Hematologic and blood chemical findings were within normal limits. The 14-year-old dam had a history of progressive weakness in the hindlimbs for a period of at least 1 year. The gait in the hindlimbs was ataxic with paw dragging and strong flexion of the tarsal joints. Muscle tonus and flexor reflexes in the hindlimbs were weak. Patellar reflexes were exaggerated. Pain sensation was normal, but proprioceptive positioning of the hindlimbs was slow. Plain radiography revealed ventral spondylosis of the 1st to 3rd lumbar vertebrae. On myelography of the spine, neither blockage nor deviation of the contrast medium around the spinal cord was seen. The CSF was clear, contained 3 cells/mm³ and had an equivocal Pandy reaction. A few weeks after this examination the dog became recumbent, developed urinary incontinence, and was euthanized.

Two 10-year-old sisters of the two 12-year-old afflicted dogs have not had any neurologic abnormalities to date. A 10-year-old half sister originating from the same dam has mild paresis and ataxia in the hindlimbs.

No lesions were found outside the CNS. In all 3 dogs, marked changes of the spinal cord were found on histologic examination. The lesions were similar in distribution and appearance in the 3 dogs and are therefore described together. They consisted of disseminated vacuolation of the white matter at all levels of the cord, predominantly in the ventral and lateral columns (Fig 1). The thoracic segments were much more severely involved than were other segments. The lesions were more pronounced in the peripheral cord areas than in areas close to the gray matter, but they were neither symmetric nor associated with anatomically defined tracts. In several vacuoles, clusters of macrophages were found (Fig 2) and the vacuolation was sometimes associated with axonal swelling (Fig 3) and necrosis. There was little gliosis and there were no inflammatory changes.

Degenerative myelopathy of large dogs was first reported in the United States in 1973. Since then, additional cases from the United States and Europe have been reported. Degenerative myelopathy develops in aging dogs of large breeds, especially German Shepherd Dogs. Clinically there is slowly progressive weakness and ataxia, with loss of proprioceptive function and muscle atrophy in the hindlimbs. Spinal reflexes may be abnormal and there is occasional loss of urinary sphincter control. Pathologic findings have included degenerative changes in the white matter of the spinal cord in all reported cases, but there have been differences concerning the distribution of the lesions and whether or not the spinal roots have been involved in the disease process. Because of such differences and the fact that a variety of dog breeds may become affected, it is uncertain whether all the reported cases of degenerative myelopathy in older dogs represent a distinct disease entity. The cause of degenerative myelopathy is unknown. A genetic base of the disease in German Shepherd Dogs has been considered.

The Siberian Huskies of this report had clinical features similar to those in previous reports on degenerative myelopathy in large dog breeds. In addition, the histologic findings in the white matter of the thoracic spinal cord were similar to those in aging German Shepherd Dogs and other large dogs with progressive ataxia. The lesions consist of myelin loss as well as axonal degeneration. Since it is not clear at this stage whether demyelination prevails in these lesions, it is not justifiable to classify this disease as a demyelinating condition. There has been much speculation on the cause of degenerative myelopathy in other breeds of dogs. Spinal cord compression associated with osseous metaplasia of the dura mater or old age changes of the spine in our Siberian Huskies can be excluded on the basis of the negative myelographic findings, as has been reported in the literature for other breeds with a degenerative myelopathy. On the basis of pathologic findings, there is no evidence for an infectious cause of the disease. A disturbed immune response has been observed in German Shepherd Dogs with myelopathy but it was not clear as to how far such dysfunction could contribute to spinal cord degeneration. By all means there is no morphologic evidence for an immune mediated nature of the lesions in degenerative myelopathy, as in known allergic or infectious diseases involving the white matter. Apart from Averill's methylnalonic acid determinations excluding vitamin E deficiency as a cause of the degenerative myelopathy in German Shepherd Dogs, the possibility of an underlying metabolic disorder in dogs with such lesions has not been investigated. A morphometric study in German Shepherd Dogs with myelopathy ruled out a "dying back" type of disease, as has been seen in certain toxic neuropathies and myelopathies. Braund and Vandevelde thought that the uniformity of the lesions and the predictability of the clinical course in German Shepherd Dogs kept under varying conditions and in many areas of the world would suggest that hereditary factors may be decisive for the apparition of this disease. One would have to postulate some kind...
of inborn error of metabolism, with late onset of clinical signs. Known hereditary degenerative conditions of the spinal cord in dogs and cats are diseases of the immature animal often apparent soon after birth. Unfortunately, only a limited number of dogs could be examined in this study. Nevertheless the development of degenerative myelopathy in closely related dogs is suggestive of the hereditary nature of this condition in Siberian Huskies. We cannot exclude the possibility of a common exogenous factor causing the disease. Only breeding trials could confirm the genetic basis of myelopathy in Siberian Huskies. Unfortunately, the extremely late onset of the clinical signs is a serious obstacle for such studies.