

the urogenital system.¹ The natural reservoir of streptococcal strains pathogenic for cats is unknown, as is the prevalence of this organism in the cat population. Although group G streptococci are considered part of the normal flora of skin, pharynx, upper respiratory tract, vagina, and prepuce in cats,² the source of the outbreak described here could not be determined.

A common route of infection for pyothorax is via trauma to the thoracic cavity; however, no visible evidence of thoracic injury was found in either of the animals. Bacterial organisms causing pyothorax include *Pasteurella multocida* and *Nocardia asteroides*. These infections are often secondary to primary viral infections, e.g., with feline herpesvirus and calicivirus. Group G streptococcal tonsillitis and cervical lymphadenitis with resultant pulmonary thromboembolism and infarct has been described in juvenile cats,² but tonsillitis, cervical lymphadenitis, and pulmonary thromboembolism and infarct were not seen in the 2 cats examined. Nevertheless, infection of cervical lymph nodes with group G streptococci and subsequent lymphatic spread, resulting in pyothorax, pleuritis, and pneumonia, could not be completely ruled out.

Because infections with Lancefield group G streptococci have been reported in humans,⁶ all personnel working in the cat colony were asked to submit throat swabs for bacterial culture. No beta-hemolytic group G streptococci were recovered from those swab cultures.

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Sources and manufacturers

- a. Sigma Chemical Co., St. Louis, MO.
- b. Boehringer Mannheim, Indianapolis, IN.
- c. Promega, Madison, WI.
- d. Microns Separations, Westboro, MA.
- e. Fuji Film, Tokyo, Japan.

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Lower motor neuronopathy in a Dutch Belted-Sembra bull

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Motor neuronopathies have been described in a variety of species, including dogs, cats, horses, swine, cattle, and humans, and have been subcategorized based on lesion distribution (upper or lower motor neuron) and pathologic features (neurofilament accumulation).^{1,8} Lower motor neuron

disease with spinal muscular atrophy and neurofilament accumulation has been recognized in these species, with particular breed predilections.⁸ Here, we report a lower motor neuronopathy in a cross-breed bull that has not been previously described, including features unique to this case, and compare this with other lower motor neuron diseases.

A recently purchased 15-month-old Dutch Belted-Sembra cross bull was referred to the University of Tennessee College of Veterinary Medicine after approximately 5 days of progressive posterior ataxia with no known exposure to tox-

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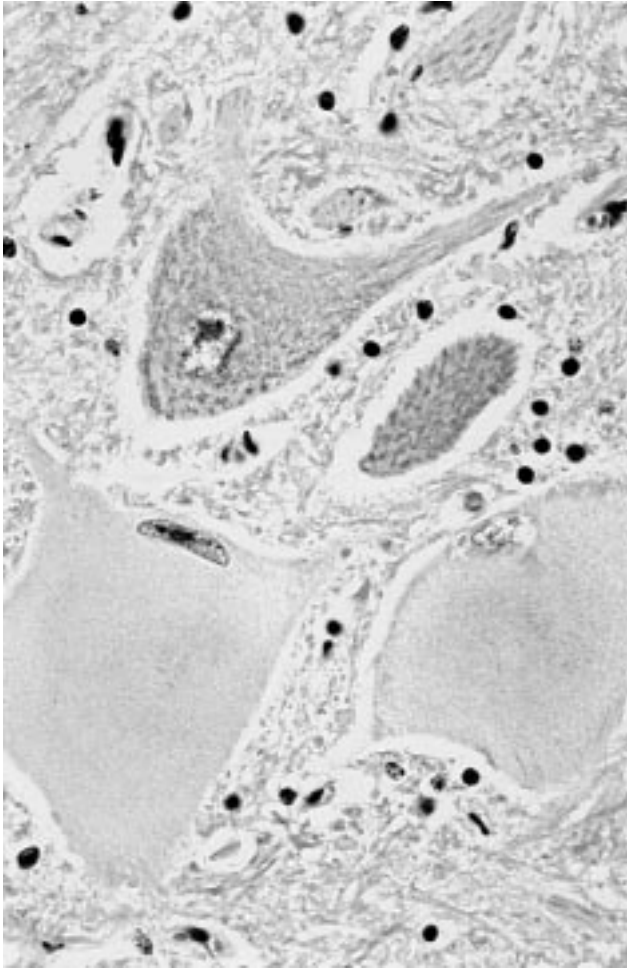


Fig. 1. Lumbo-sacral spinal cord of a young bull. Enlarged, swollen neurons with complete chromatolysis (ghost cells) are visible throughout the ventral horn gray matter. Hematoxylin and eosin.

ins or trauma. On physical examination, the bull was responsive with alert mental status, intact spinal reflexes, and deep pain perception in all limbs. There were no cranial nerve deficits, and no abnormalities were noted in the front limbs. Complete blood count, serum chemical analyses, and cerebrospinal fluid analysis were within normal limits. Additionally, aerobic and anaerobic cultures of cerebrospinal fluid were negative. Two days after referral, the bull became recumbent, with refusal to stand or bear weight when supported by a full body sling. At this time, the owner elected that the bull be euthanized.

The necropsy exam revealed multiple coalescing, reddened, discrete linear erosions throughout the esophagus, with more extensive involvement of the distal esophageal mucosa. The rumen contained a mixture of short plant fiber ingesta and lesser amounts of oats, with a focally extensive (9×1.5 cm) erosion of the cranial ruminal pillar. The abomasum contained a 3.2-cm-diameter firm, depressed ulcer just anterior to the pylorus. There were no gross abnormalities within the central nervous system and no evidence of muscle atrophy or trauma.

The most significant histologic findings were restricted to

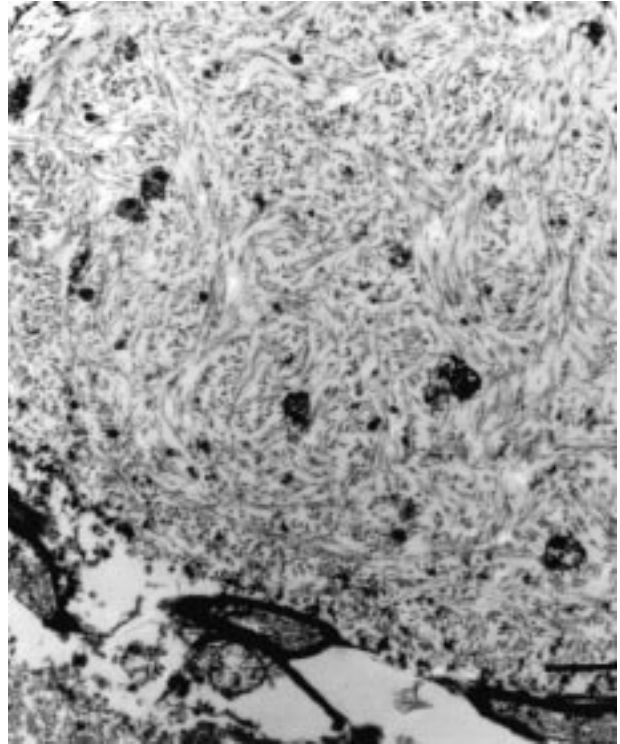


Fig. 2. Electron micrograph of a lower motor neuron in the ventral gray horn of a young bull. The neuron is distended by numerous neurofilaments that encircle cytoplasmic organelles. Bar = 125 nm.

the spinal cord in the areas of the lumbar intumescence and, to a much lesser extent, the cervical intumescence. In these regions, the ventral gray matter horns contained numerous swollen, round nerve cell bodies with either complete or central chromatolysis and prominent peripherally displaced, occasionally karyolytic nuclei (Fig. 1). The nearly clear cytoplasm of these "ghost cells" contained faint eosinophilic fibrillar material that frequently extended into and expanded dendrites and proximal axons. The cytoplasm of the swollen neurons was distended by large aggregates of argyrophilic fibrils, as demonstrated by Sevier-Munger/Luxol fast blue and Guillery-stained sections. Rare necrotic neurons within the ventral horn were hypereosinophilic and shrunken with hyperchromatic nuclei. Occasional swollen hyaline axons were scattered throughout the ventral horn gray matter but were not present within the surrounding white matter. Demyelination and prominent spheroids were not present.

Transmission electron microscopic examination revealed a large number of 10-nm-diameter, haphazardly arranged, occasionally clustered neurofilaments that wrapped around and encircled mitochondria, endoplasmic reticulum, and free ribosomes in the cytoplasm of the swollen neurons (Fig. 2).

The ulcerated and eroded areas of the rumen, abomasum, and esophagus were associated with prominent infiltrates of macrophages, neutrophils, and plasma cells, and the submucosa was replaced by moderate amounts of granulation tissue. Bovine viral diarrhea virus (BVDV) was not isolated from esophageal lesions. Additionally, an immunoperoxidase test for BVDV was negative using paraffin-embedded samples of esophagus, abomasum, spinal cord, and cerebrum.

The diagnosis of lower motor neuronopathy was based on the physical exam and histopathologic and electron microscopic findings, but the cause of these changes was not determined. Possible causes include heavy-metal intoxication, poisonous plant ingestion, viral infection, and nutritional imbalances. The localized distribution of the noninflammatory central nervous system lesions in the absence of other significant neuropathologic changes, such as Wallerian degeneration and the lack of significant axonal and myelin involvement, make these etiologies unlikely. However, not unlike other reported cases of neuronopathies, many of these causes could not be definitively confirmed or ruled out. Additionally, the cause of the esophageal, rumenal, and abomasal ulcers is not known. A direct relationship between these lesions and the spinal cord lesions could not be found, but these ulcers may be the result of an undetected infectious agent or mechanical trauma.

A hereditary basis for the neuronopathy in this bull must also be considered because these lesions are very similar to hereditary neuronopathies previously reported in other bovine breeds and species. Hereditary canine spinal muscular atrophy of the Brittany Spaniel is characterized by similar localized neuronal swelling and "ghost cells" within the ventral gray matter horns of the spinal cord due to neurofilament accumulation in the perikarya.^{2,6,7} The bundles of neurofilaments are the result of impaired transport, synthesis, or catabolism of cytoskeletal proteins and may be the underlying pathogenesis of neurofilament accumulation in this bull.^{2,3,8} However, in Brittany Spaniels, unlike in the bull in this report, there are lesions in neurons within the brain stem (trigeminal motor and hypoglossal nuclei) and prominent muscular atrophy of the fore- and hind limbs.² Additionally, the intermediate phenotypic variant of canine spinal muscular atrophy is slowly progressive, whereas in this bull there was sudden onset with rapid progression, which may explain the lack of prominent spinal muscular atrophy.

Additional hereditary lower motor neuronopathies have been described in Brown Swiss, horned Hereford, and Red Danish calves that had similar lesions within both the spinal cord and brain stem associated with skeletal muscle atro-

phy.^{1,4,5} However, in addition to the differences in lesion distribution and absence of muscular atrophy, the age of onset in this affected bull was much later (15 months) than that described in the Brown Swiss, horned Hereford, and Red Danish calves (birth to 21 weeks).^{1,8} In the present case, it is not clear whether these differences represent a unique disease or whether they are simply variations of the pathogenesis associated with these other lower motor neuronopathies. Additionally, a hereditary basis for the neuronopathy in the bull of this report cannot be confirmed or ruled out because pedigree information was not available. However, this case does represent a gross and histologically unique neuronopathy with features similar to other hereditary neuronopathies.

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Bilateral testicular leiomyosarcoma in a stallion

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Testicular neoplasia is uncommon in the horse. Actual incidence of equine testicular neoplasia is difficult to determine because most male horses are castrated at an early age.

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Primary testicular tumors that have been reported are interstitial (Leydig) cell tumor,^{7,21} seminoma,^{2,8,11,13,14,20,24–26} Sertoli (sustentacular) cell tumor,¹⁶ teratoma,^{3,17,22,23,28} teratocarcinoma,¹⁸ embryonal carcinoma,²⁷ a mixed germ cell–sex cord–stromal neoplasm (gonadoblastoma),^{4,14} and a mast cell tumor.¹⁴ A leiomyoma, believed to be a portion of a teratoma,¹⁵ and paratesticular leiomyoma involving the vascular tunic have also been reported.^{12,14}

An 11-month-old cryptorchid Thoroughbred stallion was presented for necropsy because of progressive ataxia, poor growth rate, and mandibular swellings. The owner had pur-