ARTICLES

A retrospective study of nineteen ataxic horses

Germain Nappert, André Vrins, Luc Breton, Michel Beauregard

Abstract

A retrospective study of 19 ataxic horses admitted to the College of Veterinary Medicine of the University of Montreal during the period of January 1985 to December 1988 is presented. There were 11 cases of cervical vertebral malformation, four of equine degenerative myeloencephalopathy, two of equine protozoal myeloencephalitis, one each of vertebral osteomyelitis and intervertebral disc protrusion. The clinical diagnosis of ataxia in horses requires neurological, radiographic, myelographic, and laboratory examinations.

Résumé

Étude rétrospective de dix-neuf cas d'ataxie équine

Les auteurs présentent une étude rétrospective de 19 chevaux référés pour ataxie à l'hôpital des grands animaux de la Faculté de médecine vétérinaire de l'Université de Montréal de janvier 1985 à décembre 1988. Onze d'entre eux souffraient de malformation vertébrale cervicale; quatre, de myéloencéphalopathie dégénérative équine; deux, d'encéphalomyélite à protozoaire; un, d'ostéomyélite de l'atlas et un autre, de hernie discale. Le diagnostic clinique d'ataxie équine se base sur des examens neurologique, radiographique, myélographique et des analyses de laboratoire.

Can Vet J 1989; 30: 802-806

Introduction

Many diseases of the spinal cord produce ataxia and paresis in horses (1,2). Traditionally, the term "wobbler" has been broadly applied to describe signs of ataxia, weakness, spasticity, and paresis in horses (3). However, with improved diagnostic techniques, several specific diseases have been recognized. Cervical vertebral malformation is a common cause of spinal cord compression in many breeds of horses. It is subdivided into cervical static stenosis when the stenosis of the vertebral canal exists independently of the angulation between involved vertebrae, and cervical vertebral instability when the stenosis occurs only with moderate to severe flexion of the neck (3-5).

Département de Médecine (Nappert, Vrins, Breton) and Département de Pathologie (Beauregard), Faculté de Médecine vétérinaire, Université de Montréal, C.P. 5000, Saint-Hyacinthe, Québec J2S 7C6.

We present herein a retrospective study of 19 ataxic horses admitted to the College of Veterinary Medicine of the University of Montreal during the period of January 1985 to December 1988. Neurological, radiographic and laboratory examinations were performed to identify the anatomical site and extent of the problem. The definitive diagnosis was determined by necropsy.

Materials and methods

On admission of all ataxic horses, physical and neurological examinations were performed as described elsewhere (1,6). Blood samples were collected for hematology and biochemical analysis. Standing lateral radiographs of the neck were performed with the neck in a neutral position, using an 8:1, 41 lines/cm, 100 cm focal distance grid with high speed film and screens. Cerebrospinal fluid (CSF) was withdrawn from the cerebellomedullary cistern at the atlanto-occipital site to determine the refractive index, the red blood cell and leukocyte counts, the differential leukocyte count, and the total protein content in 12 cases (7,8). Myelographic examinations under general anesthesia were performed using the water-soluble contrast medium, metrizamide (Amipaque, Winthrop, Dorval, Quebec) in seven cases (9). Myelographic spinal cord compression was considered to be present when both dorsal and ventral contrast columns were narrowed by more than 50% (10). Gross necropsy examinations were done immediately following euthanasia with barbiturates; the brain and the spinal cord were then fixed in 10% formalin.

Results

Eight horses with cervical static stenosis (CSS) had ataxia of one to 12 month duration (Table 1). Neurological signs of ataxia and paresis were symmetric and more prominent in the hindlimbs at a slow walk. Circumduction of the outside limb when walking in small circles and difficulty in backing were also noted (Figure 1). Samples of CSF were obtained in three horses and were normal on clinicopathological examination (8). Radiographs of the cervical vertebral column revealed signs of degenerative joint disease related to osteochondrosis of the articular facets in six horses, and no skeletal change in two horses.

TABLE 1
Breed, age, sex, site of lesion and diagnostic procedures in 19 ataxic horses

Breed	Age	Sex	Lesion	Diagnostic techniques
Cervical vertebral malformation				
a) Cervical static stenosis				
Standardbred	6 mo	F	C3-4	Radiography, myelography and necropsy
Standardbred	8 mo	M	C5-6	Radiography and necropsy
Standardbred	1 yr	F	C5-6	Radiography and necropsy
Standardbred	1 yr	M	C4-7	Radiography and necropsy
Arabian X Percheron	2 yr	M	C6-7	Radiography, myelography, CSF analysis and necropsy
Thoroughbred	6 yr	M	C3-6	Radiography and necropsy
Hannoverian X Thoroughbred	2 yr	M	C6-7	Radiography, myelography, CSF analysis and necropsy
Canadian Hunter	8 yr	M	C5-6	Radiography, myelography, CSF analysis and necropsy
b) Cervical vertebral instability				
Thoroughbred	7 mo	F	C3-4	Radiography, myelography, CSF analysis and necropsy
Thoroughbred	8 mo	M	C4-5	Radiography and necropsy
Appaloosa	1 yr	F	C3-4	Radiography, CSF analysis and necropsy
Equine degenerative myeloencephalopathy				
Quarter Horse	6 mo	F		Radiography, CSF analysis and necropsy
Anglo-Arabian	10 mo	F		Radiography, myelography, CSF analysis, necropsy and serology
Thoroughbred	14 mo	F		Radiography and necropsy
Standardbred	18 mo	M		Radiography, myelography, CSF analysis and necropsy
Equine protozoal myeloencephalitis				
Thoroughbred	14 yr	M		Radiography, CSF analysis and necropsy
Standardbred	12 yr	F		CSF analysis and necropsy
Disc protrusion				
Quarter Horse	13 yr	M	C6-7	Radiography, CSF analysis, serology, and necropsy
Vertebral osteomyelitis				
Standardbred	3 mo	M	atlas	Radiography, CSF analysis and necropsy

Myelographic examination in four horses revealed dorsal compression of the spinal cord (Figure 2). At necropsy, one horse had compression at C3-4, three at C5-6, one at both C3-4 and C5-6, and two at C6-7. One horse had evidence of spinal cord compression at three sites (C4-5, C5-6 and C6-7). Necropsy findings revealed compression of the spinal cord caused by mild osteosclerosis of the dorsal lamina and enlargement of the ligamentum flavum. Histologically, there was degeneration of axons at the site of compression.

Cervical vertebral instability (CVI) was diagnosed in three horses, seven to twelve months of age, at the midcervical vertebrae (C3-4,C4-5). Plain radiographs of the cervical vertebrae did not reveal any important changes. In one horse, myelographic examination showed evidence of spinal cord compression (Figure 3). The CSF findings were normal in two horses. Results of the complete blood count and serum chemistry profile were within normal limits in all cases of cervical vertebral malformation.

Four horses affected with equine degenerative myeloencephalopathy (EDM) showed progressive signs of ataxia of one to eight months duration. Pelvic limb ataxia alone was present in one foal. Pelvic and thoracic limb ataxia were noted in two others, although this was less severe in the thoracic limbs. Symmetric tetraparesis and ataxia were noticed in another case. Radiographic and myelographic examinations of the cervical vertebral column were normal. Laboratory studies on blood and CSF were normal except in one foal which had chronic anemia, neutrophilia and elevated plasma fibrinogen related to guttural pouch empyema caused by *Streptococcus zooepidemicus*; the serum neutralization titer to equine herpesvirus-1 was negative in that foal. At necropsy, there were no gross lesions in the four cases of EDM. Microscopically, the characteristic degeneration was demonstrated mainly in the dorsolateral and ventromedial funiculi in the white matter of the cervical and thoracic segments. Abnormal axonal swellings (spheroids) of various sizes and proliferation of glial cells, specially astrocytes, were present in the grey matter of the cord and brainstem. Accumulation of lipofuscin-like pigments in the cytoplasm of some neurons was also noted in one case.

Two cases clinically and pathologically resembling equine protozoal myeloencephalitis (EPM) were presented: a 14-year-old Thoroughbred gelding, born

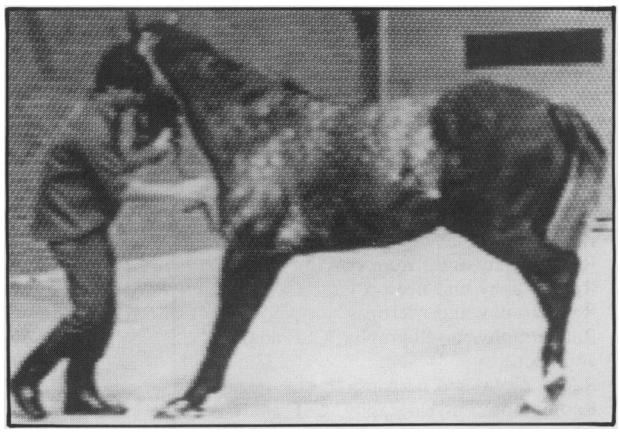
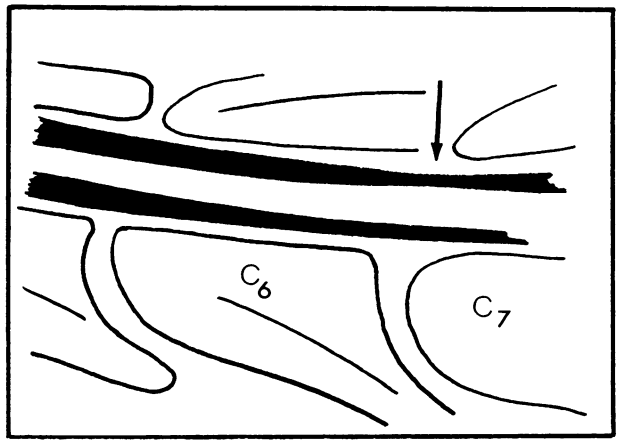


Figure 1. A five-year-old Thoroughbred gelding demonstrating difficulty in backing.

and raised in Ontario, and a 12-year-old Standardbred mare, born in Pennsylvania and brought to Quebec six years later. Both horses were referred for evaluation of asymmetrical ataxia, hemiparesis, muscular atrophy of the rump, and lateral recumbency of acute onset. However, the Thoroughbred had a history of static ataxia of the left forelimb for two years. Cerebrospinal fluid analyses were within normal limits except for elevated protein levels (0.92 g/L) without xanthochromia in the Thoroughbred. Therapy consisted of a trimethoprim-sulfamethoxazole and pyrimethamine combination for a month. The Thoroughbred did not respond to the treatment and later developed laminitis following administration of high dosages of corticosteroids. Euthanasia was then recommended. The Standardbred mare improved for two months and was readmitted for euthanasia after a sudden increase of neurological signs. On gross examination, the Thoroughbred had a yellow focus, 2 cm in diameter, in the right side of the pons with hemorrhagic foci throughout the cerebral cortex. Histologically, a multifocal necrotizing nonsuppurative encephalitis, with extensive perivascular cuffing involving lymphocytes, eosinophils and astrocytes, was seen. Protozoal organisms were observed in an occasional rosette formation of merozoites within neurons. No gross lesions were found in the Standardbred mare and histological changes were similar but localized mostly to the thoracic portion of the spinal cord. However, protozoal organisms were not observed.

A 13-year-old quarter Horse gelding was presented with a history of pelvic limb ataxia of sudden onset. On admission, a dysmetric ataxic gait was noted in the pelvic limbs with a spastic gait in the thoracic limbs. The horse exhibited difficulty in backing and a tendency of crossing the thoracic limbs when circling. Abnormalities were not noted on plain radiographs of the cranial cervical vertebrae. Results of complete blood count were within normal limits. Analysis of CSF demonstrated xanthochromia with an elevated protein content (1.0 g/L). Neutralization titers to equine herpesvirus-1 were low in serum and CSF. There was no clinical improvement with dexamethasone and dimethyl sulfoxide, and the owner opted for euthanasia three days later. At necropsy, an intervertebral disc protrusion between the sixth and seventh cervical vertebrae was found. Histologically, there was



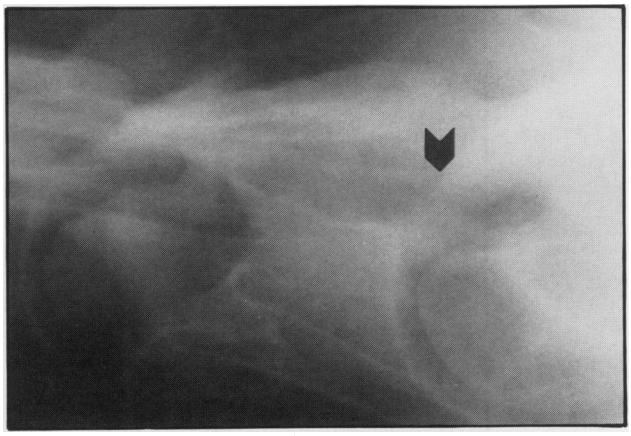


Figure 2. Myelogram (and line drawing) illustrating a compressive spinal cord lesion with narrowing of the dorsal and ventral contrast columns at the intervertebral space C6-C7 (arrows).

degeneration of the spinal cord in the area of the protrusion.

A three-month-old Standardbred colt was presented because of incoordination that had progressed over a month, until he became recumbent. On admission, clinical signs included a stiff neck with swelling in the area of Viborg's triangle, fever, tachypnea, neutrophilic leukocytosis, and an elevated plasma fibrinogen concentration. Cerebrospinal fluid analysis, with an increased neutrophil and protein content, suggested a bacterial meningitis. Lateral radiographs revealed osteolytic lesions at the occipital condyles. *Rhodococcus equi* was cultured from CSF and from the exudate of the right guttural pouch. At necropsy, a vertebral fracture of the atlas associated with severe osteolytic lesions was found.

Discussion

In the population studied, there were no predispositions for males or Thoroughbred horses to cervical vertebral malformation. Our data were biased by several factors, including the retirement of affected females for breeding and the higher frequency of Standardbred admissions to the College. Our CVI cases occurred frequently in young horses as reported elsewhere (4,5). They were mostly characterized by narrowing of the spinal canal at the midcervical vertebrae during neck flexion. Horses affected with CSS were older and the lesions

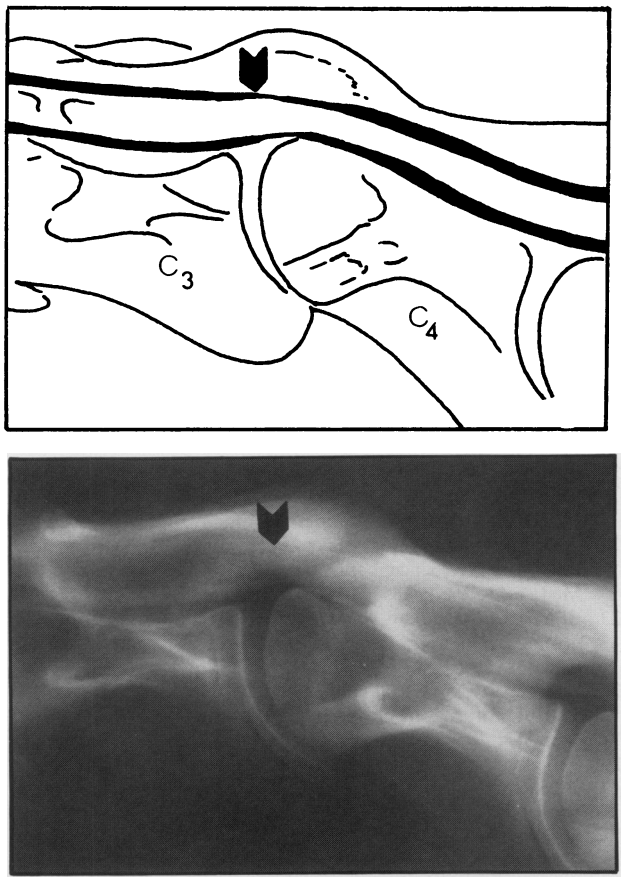


Figure 3. Myelogram (and line drawing) illustrating cervical vertebral instability at the intervertebral space C3-C4 (arrow).

usually occurred in the caudal cervical spine, which resulted in dorsal or dorsolateral compression of the spinal cord regardless of the neck position.

At necropsy, the pathogenesis of CVM seemed to be associated with the development of osteochondrosis of the articular processes which led to degenerative joint disease (DJD), and hypertrophy and/or degeneration of the ligamentum flavum with the formation of scars (5,11). The heritability of the condition, the genetic predisposition for rapid growth combined with overnutrition or abnormal biomechanical forces and trauma were not studied, but they have been reported elsewhere (1-5,12,13).

Our antemortem diagnosis was based on signs of ataxia, which was more pronounced in the pelvic limbs, and on radiographic and myelographic examinations when available (13,14). Hemograms, serum chemistries, CSF analysis and serological tests were usually within normal limits. In all cases of CVM, signs became progressively worse in spite of conservative medical therapy with anti-inflammatory and analgesic agents (4,13). No surgical procedures were performed because the prognosis reported elsewhere was guarded; less than 56% of patients returned to athletic activities (15-18).

Equine degenerative myeloencephalopathy (EDM) has not been reported in Canada previously. In our study, horses affected with EDM were of light breeds, had an early age at onset of progressive symmetrical tetraparesis, ataxia and spasticity of the pelvic or thoracic limbs or both. Clinically, one horse affected with EDM was distinguished from CVM because of the forelimb deficits were as pronounced as those in the pelvic limbs. In the other three cases, diagnosis was based on ruling out other neurological conditions with normal laboratory, radiographic and myelographic evaluations (1,3,14,19). All horses affected with EDM were euthanized and definitive diagnosis was made at necropsy with typical histopathological findings as described above (1,20). The causes of diffuse degeneration of the brain stem and spinal cord were unknown but they have been reported elsewhere to be toxic, nutritional, metabolic or heritable in origin (7,20).

Equine protozoal myeloencephalitis (EPM) has been recognized as a disease of young adult horses of all sexes and breeds, particularly in Thoroughbreds and Standardbreds (1,3,7,21). Equine protozoal myeloencephalitis has been reported more frequently in the eastern United States, but it is also known to occur in California (22), western Canada (23) and southern Brazil (24). A *Sarcocystis* protozoal organism has been suspected as the etiological agent of EPM (25). The disease is probably not contagious, because the horse is a dead-end host (3,21). The parasite is responsible for diffuse or multifocal, nonsuppurative, inflammatory lesions of the brain and spinal cord (1,3,22,23,26).

Clinically, our two cases of EPM exhibited signs of multifocal gray matter involvement such as asymmetrical ataxia, muscular atrophy, sensory loss or hypoalgesia, and cranial nerve deficits. The disease appeared to be progressive with an unfavorable prognosis. The Thoroughbred had an elevation of the total protein in the CSF, which could suggest a herpes viral myelitis (3,26). However, protozoal organisms were observed microscopically. The Standardbred mare had been treated with antiprotozoal drugs, which may explain why typical lesions of nonsuppurative necrotizing encephalomyelitis were present without protozal organisms (21).

Intervertebral disk protrusion is a rare cause of ataxia in horses (27-29). Antemortem diagnosis is based on history, CSF analysis, apparent neck pain, and radiographic and myelographic examinations (28). Successful decompression has not yet been reported (29). Vertebral body osteomyelitis (VBO) is frequently secondary to another infection in foals that have not received proper immunological protection (30). Vertebral body osteomyelitis is usually suspected with clinical signs of neck or back pain, paresis, fever, hyperfibrinogenemia and neutrophilic leukocytosis (3). Nuclear scintigraphy with plain radiographs has been found to be the most accurate diagnostic method (30). Early detection and the use of appropriate antimicrobial agents will ensure successful treatment.

Cervical vertebral malformation is the most common cause of spinal cord compression (1,2). Other conditions that should be included in the differential diagnosis of equine ataxia are: vertebral fractures (1,7,14), occipitoatlantoaxial malformation (3,7,14,31), neuritis of the cauda equina (1,14), neoplasia (7,26,32), thromboembolic ischemic myelopathy (33), rhinopneumonitis myeloencephalitis (3,7,26,34), cerebellar

abiotrophy (1), parasitic myelitis (3,35), equine infectious anemia (31), and congenital abnormalities of venous drainage (36). An accurate clinical diagnosis requires neurological, radiographic, myelographic and laboratory examinations.

Acknowledgments

We extend our thanks to Dr. Jean-Pierre Lavoie and Mrs. Martine Brault for their assistance in the preparation of this paper. Special thanks are given to Drs. Joanne Hardy, Louise Duplain and Yves Rossier, for their collaboration. We are grateful to Mr. Richard Bourrassa and Mr. Denis Brouillette for providing the illustrations.

References

1. Reed SM, Bayly WM, Traub JL, Gallina A, Miller LM. Ataxia and paresis in horses. Part I. Differential diagnosis. Compend Contin Educ Pract Vet 1981; 3: S88-S99.
2. Mayhew IG, De Lahunta A, Whitlock RH, Krook L, Tasker JB. Spinal cord disease in the horse. Cornell Vet (Suppl) 1978; 68: 1-207.
3. Wagner PC. Disease of the spine. Equine Medicine and Surgery. 3rd ed. Santa Barbara: American Veterinary Publishing Inc., 1982: 1214-1223.
4. Wagner PC. Cervical vertebral malformation. In: Robinson NE, ed. Current Therapy in Equine Medicine. 2nd ed. Philadelphia: WB Saunders Company, 1987: 355-359.
5. Nixon AJ, Stashak TS, Ingram JT. Diagnosis of cervical vertebral malformation in the horse. Proc 28th Am Assoc Equine Pract 1983: 253-265.
6. Smith JM, Cox JH, DeBowes RH. Central nervous system disease in adult horses. Part I. A data base. Compend Contin Educ Pract Vet 1987; 9: 561-569.
7. De Lahunta A. Cerebrospinal fluid and hydrocephalus. In: De Lahunta A, ed. Veterinary Neuroanatomy and Clinical Neurology. 2nd ed. Philadelphia: WB Saunders Company, 1983: 30-52.
8. Mayhew IG, Whitlock RH, Tasker JB. Equine cerebrospinal fluid: Reference values of normal horses. Am J Vet Res 1977; 38: 1271-1274.
9. Rantanen NW, Gavin PR, Barbee DD, Sande RD. Ataxia and paresis in horses. Part II. Radiographic and myelographic examination of the cervical vertebral column. Compend Contin Educ Pract Vet 1981; 3: S161-S171.
10. Papageorges M, Gavin PR, Sande RD, Barbee DD, Grant BD. Radiographic and myelographic examination of the cervical vertebral column in 306 ataxic horses. Vet Radiol 1987; 28: 53-59.
11. Powers BE, Stashak TS, Nixon AJ, Yovich JV, Nordin RW. Pathology of the vertebral column of horses with cervical static stenosis. Vet Pathol 1986; 23: 392-399.
12. Falco M, Whitwell K, Palmer AC. An investigation into the genetics of "wobbler disease" in Thoroughbred horses in Britain. Equine Vet J 1976; 8: 165-169.
13. De Lahunta A. Large animal spinal cord disease. In: De Lahunta A, ed. Veterinary Neuroanatomy and Clinical Neurology. 2nd ed. Philadelphia: WB Saunders Company, 1983: 215-237.
14. Smith JM, DeBowes RM, Cox JH. Central nervous system disease in adult horses. Part II. Differential diagnosis. Compend Contin Educ Pract Vet 1987; 9: 772-778.
15. Wagner PC, Grant BD, Gallina AM, Bagby GW. Ataxia and paresis in horses. Part III. Surgical treatment of cervical spinal cord compression. Compend Contin Educ Pract Vet 1981; 3: S192-S202.
16. Nixon AJ, Stashak TS. Dorsal laminectomy in the horse. I. Review of literature and description of a new procedure. Vet Surg 1983; 12: 172-176.
17. Nixon AJ, Stashak TS, Ingram JT, Norrdin RW, Colter SB, Lebel JL. Dorsal laminectomy in the horse. II. Evaluation in the normal horse. Vet Surg 1983; 12: 177-183.
18. Nixon AJ, Stashak TS, Ingram JT. Dorsal laminectomy in the horse. III. Results in horses with cervical vertebral malformation. Vet Surg 1983; 12: 184-188.
19. Beech J. Equine degenerative myeloencephalopathy. In: Robinson NE, ed. Current Therapy in Equine Medicine. 2nd ed. Philadelphia: WB Saunders Company, 1987: 353-355.
20. Mayhew IG, De Lahunta A, Whitlock RH, Geary JC. Equine degenerative myeloencephalopathy. J Am Vet Med Assoc 1977; 170: 195-201.
21. Brewer BD. Equine protozoal myeloencephalitis. In: Robinson NE, ed. Current Therapy in Equine Medicine. 2nd ed. Philadelphia: WB Saunders Company, 1987: 359-363.
22. Dorr TE, Higgins RJ, Dangler CA, Madigan JE, Witham CL. Protozoal myeloencephalitis in horses in California. J Am Vet Med Assoc 1984; 185: 801-802.
23. Clark EG, Townsend HGG, McKenzie NT. Equine protozoal myeloencephalitis: A report of two cases from western Canada. Can Vet J 1981; 22: 140-144.
24. Lombardo De Barros CS, De Barros SS, Dos Santos MN. Equine protozoal myeloencephalitis in southern Brazil. Vet Rec 1986; 13: 283-284.
25. Simpson CF, Mayhew IG. Evidence for *Sarcocystis* as the etiologic agent of equine protozoal myeloencephalitis. J Protozool 1980; 27: 288-292.
26. Smith JM, DeBowes RH, Cox JH. Central nervous system disease in adult horses. Part III. Differential diagnosis and comparison of common disorders. Compend Contin Educ Pract Vet 1987; 9: 1042-1051.
27. McKelvey WAC, Owen RR. Acquired torticollis in eleven horses. J Am Vet Med Assoc 1979; 175: 295-297.
28. Foss RR, Genetzky RM, Riedesel EA, Graham C. Cervical intervertebral disc protrusion in two horses. Can Vet J 1983; 24: 188-191.
29. Nixon AJ, Stashak TS, Ingram JT, Norrdin RW, Park RD. Cervical intervertebral disk protrusion in a horse. Vet Surg 1984; 13: 154-158.
30. Markel MD, Madigan JE, Lichtensteiger CA, Large SM, Hornof WJ. Vertebral body osteomyelitis in the horse. J Am Vet Med Assoc 1986; 188: 632-634.
31. McClure JJ, Lindsay WA, Taylor W, Ochoa R, Issel CJ, Coulter S. Ataxia in four horses with equine infectious anemia. J Am Vet Med Assoc 1982; 180: 279-283.
32. Traver DS, Moore JN, Thornburg LP, Johnson JH, Coffman JR. Epidural melanoma causing posterior paresis in a horse. J Am Vet Med Assoc 1977; 170: 1400-1403.
33. Taylor HW, Vandevelde M, Firth EC. Ischemic myelopathy caused by fibrocartilaginous emboli in a horse. Vet Pathol 1977; 14: 479-481.
34. Pursell AR, Sangster LT, Byars TD, Divers TJ, Cole JR. Neurologic disease induced by equine herpesvirus 1. J Am Vet Med Assoc 1979; 175: 473-474.
35. Frauenfelder HC, Kazacos KR, Lichtenfels JR. Cerebrospinal nematodiasis caused by a filariid in a horse. J Am Vet Med Assoc 1980; 177: 359-362.
36. Gilmour JS, Fraser JA. Ataxia in a Welsh cob filly due to a venous malformation in the thoracic spinal cord. Equine Vet J 1977; 9: 40-42.