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Journal
Journal of Veterinary Internal Medicine, 38(4)

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Publication Date
2024

DOI
10.1111/jvim.17125

Peer reviewed

Received: 14 December 2023 | Accepted: 14 May 2024

DOI: 10.1111/jvim.17125

Journal of Veterinary Internal Medicine Open Access

ACVIM American College of Veterinary Internal Medicine

STANDARD ARTICLE

Primary phenotypic features associated with caudal neck pathology in warmblood horses

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Funding information
Funding provided by gifts from anonymous donors towards the Equine and Comparative Neurology Research Group, UC Davis (Aleman), Grant/Award Number: V435104

Abstract

Background: Detailed descriptions of clinical signs associated with radiological findings of the caudal cervical vertebral column are not available.

Objectives/Hypotheses: Describe the clinical features associated with neck pain or stiffness, neck-related thoracic limb lameness, proprioceptive ataxia consistent with a cervicothoracic spinal cord or nerve lesion, and their frequency of occurrence compared with control horses.

Animals: A total of 223 Warmblood horses.

Methods: Case-control study. Controls and cases were recruited prospectively. All horses underwent predetermined lameness and neurologic examinations. The frequency of occurrence of each clinical feature was compared between cases and controls and relative risk (RR) were calculated.

Results: Ninety-six cases and 127 controls were included. Forty-seven (49%) of the cases were classified as neurologic, 31 (32.3%) had thoracic limb lameness, and 18 (18.7%) had neck stiffness or pain or both. Focal caudal cervical muscle atrophy (46, 47.9%), hypoesthesia (38, 39.6%), patchy sweating (16, 16.7%), hyperesthesia (11, 11.5%), and pain upon firm pressure applied over the caudal cervical articular process joints and transverse processes (58, 60.4%) were only observed in cases ($P < .001$). Sideways flexion of the neck was restricted in a higher proportion of cases (47/96, 49%) compared with controls (40/127, 31.8%; $P = .009$, RR 1.5). Hopping-type thoracic limb lameness was only observed in cases, (30, 31.6%). Deterioration in lameness after diagnostic anesthesia occurred in 13/31 (41.9%) cases.

Conclusions and Clinical Importance: Systematic clinical evaluation using the methods described should enable clinical differentiation between horses with caudal cervical lesions and horses with other causes of gait abnormalities.

KEYWORDS
ataxia, lameness, neck pain, neck stiffness, spinal cord

Abbreviations: CRD, complex repetitive discharges; EMG, electromyography; FP, fibrillation potentials; MD, myotonic discharges; PSW, positive sharp waves; RR, relative risk; RHPE, Ridden Horse Pain Ethogram; WB, Warmblood.

1 | INTRODUCTION

Cervical pain, neck stiffness (decreased range of motion), thoracic limb lameness[1-4] and ataxia[5,6] have been associated with radiological abnormalities of the caudal cervical vertebrae. Some clinical characteristics of horses with primary neck lesions have been documented.[1,3-8] A recent review described clinical signs that might be associated with cervical pain.[8] Other authors investigated if an association between morphological variations of the caudal cervical vertebrae and clinical signs occur in Warmblood (WB) horses with conflicting results, but clinical evaluations were not performed in a systematic fashion.[9,10] Prospective studies using strict, consistent, and systematic orthopedic and neurologic examinations performed by experienced specialists in a large sample of horses, to investigate the frequency of occurrence of clinical features potentially associated with primary caudal neck pathology compared with those seen in control horses are needed.

With the advent of computed tomography and digital radiography, it has become increasingly easy to acquire radiographic images of the caudal cervical and cranial thoracic vertebrae. It is well-documented that a large spectrum of radiological abnormalities of variable severity can be seen in the caudal cervical vertebrae of clinically normal horses and horses with unrelated problems, as well as in horses with neck-related pain.[9,11-15] The clinical relevance of some radiological abnormalities might be overinterpreted unless viewed with consideration of the horses' clinical signs. It is therefore important to better understand which clinical features might indicate the possible presence of a primary caudal neck problem.

Our aims were to describe the clinical features that might be associated with neck pain or stiffness, thoracic limb lameness attributable to neck pathology, or general proprioceptive (spinal) ataxia, and their frequency of occurrence compared with healthy control horses. It was hypothesized that the frequency of occurrence of clinical features reflecting neck stiffness, neck pain, neck-related thoracic limb lameness, and neurological dysfunction consistent with a caudal cervicothoracic spinal cord lesion would be higher in horses with primary neck pathology compared with control horses.

2 | MATERIALS AND METHODS

2.1 | Animals

This case-control study included 2 participating institutions, the William R. Pritchard Veterinary Medical Teaching Hospital at the University of California Davis in the USA, and the Centre for Equine Studies, Animal Health Trust in the UK. Horses were recruited prospectively and consecutively from mid-2017 to 2019. Horses enrolled for the study had to be of WB breeds (ie, Belgian WB, Dutch WB, Hanoverian, Holsteiner, Oldenburg, Rhinelander, Trakehner, Westphalian, and others). Based on the observed clinical signs, horses were classified into 1 of 2 groups: (1) control or (2) case. The control group consisted of horses undergoing prepurchase examinations with no relevant clinical abnormalities indicating a caudal cervical problem or those with lameness or poor performance with identified causes based on diagnostic local or intra-articular anesthesia not related to the neck. The case group consisted of horses with neck-related thoracic limb lameness, neck pain or stiffness, spinal cord disease localized to C1-T2 spinal cord segments with both sensory (eg, spinal ataxia, postural reactions, proprioceptive deficits) and motor (eg, dysmetria, paresis, weakness) deficits, or any combination of these. Horses with suspected or confirmed clinical diagnosis of equine protozoal myeloencephalitis and neuroaxonal dystrophy/equine degenerative myelopathy were excluded. Presence of congenital variants and abnormalities of the cervical and first thoracic vertebrae was determined by imaging in both groups of horses but not reported here because it was not the purpose of our study. The study was approved by the Clinical Ethical Review Committee of the Animal Health Trust (AHT 33-2016) and the UC Davis Institutional Animal Care and Use Committee (#19891). Informed owner consent was obtained for inclusion in the study.

2.2 | Physical variables

Information obtained for each horse included age, sex, body condition score,[16] body weight, height, and physical activity (ie, eventing, show jumping, dressage, and general purpose including driving and trail riding). All horses had normal physical examination results.

2.3 | Lameness and neurologic examinations

The examination included a standardized clinical assessment according to predefined criteria agreed by 2 of the authors (MA, SD; Data S1). This examination included systematic visual evaluation (posture, muscle development, and sweating) and palpation (muscle tension, hypoesthesia, hyperesthesia, allodynia, pain, and cervical range of motion) at rest, dynamic examination without a rider, turning in small circles, lungeing when safe on soft and firm surfaces, and ridden exercise if safe. To define horses with thoracic limb lameness associated with cervical pathology, local perineural, and intra-articular anesthesia at multiple anatomical levels and imaging were performed to rule out other sources of pain causing lameness. Horses with neck-related thoracic limb lameness showed no improvement in lameness after median and ulnar nerve blocks, or intra-articular anesthesia of the humeroradial and scapulohumeral joints, and no radiological or ultrasonographic abnormality of the sternum, cranial ribs, shoulder, and elbow regions. Other concurrent lameness (eg, pelvic limbs) was recorded when observed. A published lameness scale from 0 to 8 was used (Data S1).[17]

The neurologic examination included evaluation of behavior and state of consciousness, function of the cranial nerves, posture, and postural reactions (head, neck, trunk, thoracic and pelvic limbs, and tail), segmental reflexes (spinal reflexes limited in the standing horse to cervicofacial, cutaneous trunci, anal and perianal, withdrawal), palpation (determination of muscle tone, symmetry, and pain),

TABLE 1 Comparison of clinical features in warmblood horses: 96 cases with primary caudal neck pathology (neck-related thoracic limb lameness, $n = 31$; neck pain or stiffness $n = 18$; neurologic, $n = 47$) versus 127 control horses.

	Total Number	Case ($n = 96$)	Control ($n = 127$)	Relative risk	95% CI	P value
Work discipline						
Eventing	48	21 (21.9%)	27 (21.3%)	1.02	0.77–1.34	.91
Showjumping	57	41 (42.7%)	16 (12.6%)	2.38	1.55–3.66	<.001
Dressage	99	28 (29.2%)	71 (55.9%)	0.63	0.5–0.79	<.001
General purpose	19	6 (6.3%)	13 (10.2%)	0.82	0.59–1.13	.29
Sex						
Gelding	148	68 (70.8%)	80 (63.0%)	1.16	0.92–1.46	.22
Stallion	6	1 (1.0%)	5 (3.9%)	0.67	0.46–0.98	.19
Mare	69	27 (28.1%)	42 (33.1%)	0.91	0.72–1.15	.43
Neck posture						
Normal	199 (89.2%)	72 (75.0%)	127 (100%)	0.36	0.3–0.44	<.001
Low head and neck	8 (3.6%)	8 (8.3%)	0 (0%)	2.44	2.08–2.87	<.001
Extended	21 (9.4%)	21 (21.9%)	0 (0%)	2.69	2.25–3.22	<.001
Lateral neck flexion ($n = 222$)						
Normal	96 (43.2%)	30 (31.3%)	66 (52.4%)	0.6	0.42–0.84	.01
By tilting	118 (53.2%)	61 (63.5%)	57 (45.2%)	1.54	1.11–2.12	.01
Limited	87 (39.2%)	47 (49.0%)	40 (31.8%)	1.49	1.11–2.00	.01
Absent	4 (1.8%)	2 (2.1%)	2 (1.6%)	1.16	0.43–3.14	.36
Different to left and right	72 (32.4%)	41 (42.7%)	31 (24.6%)	1.55	1.16–2.08	.004
Neck extension						
Normal	187 (83.9%)	60 (62.5%)	127 (100%)	0.32	0.26–0.40	<.001
Limited	22 (9.9%)	22 (22.9%)	0 (0%)	2.72	2.27–3.26	<.001
Reluctance	27 (12.1%)	27 (28.1%)	0 (0%)	2.84	2.35–3.43	<.001
Response to palpation						
Normal	185 (83.0%)	58 (60.4%)	127 (100%)	0.31	0.25–0.39	<.001
Moves away sideways	26 (11.7%)	26 (27.1%)	0 (0%)	2.81	2.33–3.4	<.001
Moves away backwards	16 (7.2%)	16 (16.7%)	0 (0%)	2.59	2.18–3.07	<.001
Walk and trot						
Normal head and neck position	179 (0.8%)	54 (56.25%)	125 (98.43%)	0.32	0.25–0.4	<.001
Low head and neck carriage	28 (0.13%)	26 (27.08%)	2 (1.57%)	2.59	2.09–3.2	<.001
Turning in small circles						
Normal neck movement	144 (64.6%)	32 (33.3%)	112 (88.2%)	0.27	0.20–0.38	<.001
Neck stiffness	65 (29.2%)	52 (54.2%)	13 (10.2%)	2.87	2.17–3.8	<.001
Lunging ($n = 115$; not lunged $n = 8$)						
Normal neck posture	164 (0.8%)	42 (48.3%)	122 (96.1%)	0.28	0.22–0.38	<.001
Neck stiffness	42 (0.2%)	39 (44.8%)	3 (2.4%)	3.33	2.58–4.29	<.001
Tilting head and neck	51 (0.2%)	46 (52.9%)	5 (3.9%)	3.59	2.71–4.74	<.001
Ridden ($n = 166$; not ridden $n = 57$)						
Normal	139 (0.8%)	26 (56.5%)	113 (94.2%)	0.25	0.17–0.38	<.001
Neck stiffness	22 (0.1%)	20 (43.5%)	2 (1.7%)	5.04	3.47–7.31	<.001
Different to turn to left and right	24 (0.1%)	17 (37.0%)	7 (5.8%)	3.47	2.29–5.25	<.001
Tilting head and neck	43 (0.4%)	27 (58.7%)	16 (13.3%)	4.06	2.53–6.52	<.001
Head and neck tilt during lunging ($n = 173$)						
Head with nose to left	27 (0.2%)	17 (34.0%)	10 (8.1%)	3.69	2.29–5.95	<.001

TABLE 1 (Continued)

	Total Number	Case ($n = 96$)	Control ($n = 127$)	Relative risk	95% CI	P value
Head with nose to right	17 (0.1%)	11 (22.0%)	6 (4.9%)	3.79	2.26–6.37	<.001
No head and neck tilt	129 (0.75%)	22 (44.0%)	107 (87.0%)	Ref	Ref	Ref
Change in severity of thoracic limb lameness during ridden exercise						
Worse with rein tension	16 (0.1%)	16 (34.8%)	0 (0%)	5	3.63–6.89	<.001
Worse with no rein tension	7 (<0.1%)	6 (13.0%)	1 (0.8%)	3.41	2.27–5.10	.002
Concurrent pelvic limb lameness	181 (0.8%)	62 (64.6%)	119 (93.7%)	0.42	0.33–0.54	<.001
Neck-related thoracic limb lameness only when ridden	10	10 (100%)	0 (0%)	–	–	–
Thoracic limb lameness	8 (80%)	8 (100%)	0 (0%)	–	–	–
Neck pain or stiffness	1 (10%)	1 (100%)	0 (0%)	–	–	–
Neurological	1 (10%)	1 (100%)	0 (0%)	–	–	–
Thoracic limb stumbling	51 (0.2%)	40 (41.7%)	11 (8.7%)	2.41	1.86–3.12	<.001
Neck pain/stiffness	17 (33.3%)	17 (100%)	0 (0%)	–	–	–
Neurological	23 (45.1%)	23 (100%)	0 (0%)	–	–	–
Control	11 (21.6%)	0 (0%)	11 (100%)	–	–	–
Other static and dynamic observations						
Pain induced by extension of a thoracic limb	29 (0.1%)	29 (30.2%)	0 (0%)	2.9	2.39–3.51	<.001
Thoracic limb lameness exacerbated by proximal limb nerve blocks	13 (0.1%)	13 (13.5%)	0 (0%)	2.53	2.14–2.99	<.001
Root signature	12 (0.1%)	12 (12.5%)	0 (0%)	2.51	2.13–2.97	<.001
Neck locking	4 (1.8%)	4 (4.2%)	0 (0%)	2.38	2.04–2.78	0.03
Generalized poor neck muscle development	43 (0.2%)	38 (39.6%)	5 (3.9%)	2.74	2.16–3.48	<.001
Focal muscle atrophy	46 (0.2%)	46 (47.9%)	0 (0%)	3.54	2.80–4.48	<.001
Patchy sweating	16 (0.1%)	16 (16.7%)	0 (0%)	2.59	2.18–3.07	<.001
Hypoesthesia	38 (0.2%)	38 (39.6%)	0 (0%)	3.19	2.58–3.95	<.001
Hyperesthesia	11 (0.1%)	11 (11.5%)	0 (0%)	2.49	2.12–2.94	<.001
Abnormal stance to graze						
Thoracic limbs spread sideways	28 (0.1%)	28 (29.2%)	0 (0%)	4.63	3.45–6.21	<.001
Thoracic limbs spread forwards and backwards	31 (0.1%)	31 (32.3%)	0 (0%)	4.63	3.45–6.21	<.001
Normal stance to graze	162 (0.7%)	35 (36.5%)	127 (100%)	ref	ref	ref
Thoracic limbs crossed	2 (<0.1%)	2 (2.1%)	0 (0%)	0.22	0.16–0.29	.05
Normal reaction to light palpation of thoracic or pelvic limb						
Left thoracic	207 (99.1%)	82 (86.3%)	125 (100%)	1.02	0.77–1.34	<.001
Right thoracic	210 (99.0%)	85 (89.5%)	125 (100%)	2.02	0.77–1.35	<.001
Left pelvic	220 (100%)	95 (100%)	125 (100%)	–	–	–
Right pelvic	218 (100%)	94 (100%)	124 (100%)	–	–	–

Note: Where appropriate relative risk values with 95% confidence intervals (CI) are presented. Percentages refer to the number of horses assessed for each clinical feature, when it was less than the total number of cases and control horses. *P*-values refer to the results of Chi-squared or Fishers exact tests, as appropriate. One missing: Could not assess because of dangerous behavior with food.

and gait evaluation. Evaluation of passive neck flexion (ventral and lateral) and extension was done by tempting the horse using apple cookies or carrots. Gait evaluation consisted of observation at the walk in a straight line, head elevation, zigzag, changing surfaces, tight circles, backing up, and walking up and down a curb and hill. When safe, tail pull was used to evaluate for strength and postural reactions. Neck and shoulder extension was performed and walk in circles repeated to evaluate possible exacerbation of signs. Horses classified in the neurologic group were graded according to a modified published score for ataxia and proprioceptive deficits (grades 1–5).[18] When safe, horses with neurologic dysfunction were lunged and neurologic examination repeated to evaluate for possible exacerbation of signs. Electromyography (EMG) was performed to further evaluate a neck-related problem and a published scoring system was used for grading abnormalities (refer to Data S1 for detailed information about EMG technique, muscles examined, and scoring system).[19]

2.4 | Statistical analysis

Results were recorded in a purpose-designed Microsoft Excel spreadsheet (Version 2010). All data analyses were performed using SAS 9.4 (SAS Institute Inc), with significance set at $P < .05$. The distribution of continuous variables (horse age, body weight, and height) was tested for normality using the Shapiro-Wilk test, in combination with visual assessment of histograms, with overlaid kernel density plots. Results were expressed as mean ± SD and range. Differences between groups of continuous variables were assessed using an unpaired Student's *t*-test. Categorical variables included horses' signalment, case classification, and static and dynamic examination results, and are expressed as percentages. Denominators for controls and cases were 127 and 96, respectively, except when stated otherwise. Relationships between categorical variables were assessed using the Chi-squared (χ^2), or Fisher's exact test when observed counts in any comparison group were <5. Where appropriate, relative risks (RR) with 95% confidence intervals (CI) were calculated, comparing cases with controls, and presented in a table and figures.

3 | RESULTS

3.1 | Animals

Two-hundred and twenty-three WB horses were included in this case-control study, 127 control horses and 96 cases, comprising 148 geldings, 6 stallions, and 69 mares. No significant difference in sex distribution was found between cases and controls. Work discipline included dressage ($n = 99$, 44.4%), show jumping ($n = 57$, 25.6%), eventing ($n = 48$, 21.5%) and general purpose (including unaffiliated competition; $n = 19$, 8.5%). Show jumpers were more likely to be a case than a control ($P < .001$; RR, 2.38) whereas dressage horses were less likely to be a case than a control ($P < .001$; RR, 0.63; Table 1). Control horses were younger (mean ± SD, 8.5 ± 3.0 years; range, 3–19) than case horses (mean ± SD, 9.6 ± 4.0 years; range, 1–22; $P = .03$). Mean height ($n = 159$) was 167 ± 5 cm (range, 145–180) and mean weight ($n = 187$) was 584 ± 59 kg (range, 372–700); there was no difference between control horses and cases. Body condition score was lower for cases (5.5 ± 0.7; range, 3–7) than control (5.9 ± 0.8; range, 4–8; $P < .001$) horses.

3.2 | Case classification

Forty-seven of 96 (49%) cases were classified as neurologic, 31 (32.3%) had thoracic limb lameness, and 18 (18.7%) had neck stiffness, pain, or both. Primary neurological cases ranged in severity from grade 1 to 4: grade 1 (15/47, 31.9%), grade 2 (17/47, 36.2%), grade 3 (14/47, 29.8%), and grade 4 (1/47. 2.1%). Eight case horses with the main complaint of thoracic limb lameness, also showed mild neurological signs (grade 1, $n = 4$; grade 2, $n = 4$). Of the 96 cases, 47% (AHT, $n = 24$; UCD, $n = 21$) also showed concurrent unrelated lameness. Horses from the AHT had thoracic limb or pelvic limb lameness or both with or without a component of lumbosacroiliac pain. Clinical signs related to these concurrent problems were abolished by diagnostic anesthesia. Horses from UCD had concurrent lameness associated with weakness of the stifle and delayed release of the patellae. Electromyogrphy was performed and found to be abnormal in 27/27 horses (neurologic, 17/47; thoracic limb lameness, 8/31; neck pain/stiffness, 2/18).

3.3 | Static examination

Cases (38/96, 39.6%) were more likely to have poor muscle development of the neck compared with controls (5/127, 3.9%; $P < .001$; RR, 10.1). Focal muscle atrophy (46/96, 47.9%), hypoesthesia (38, 39.6%), patchy sweating (16, 16.7%), and hyperesthesia (11, 11.5%) were only observed in cases ($P < .001$). Standing at rest, only cases had an abnormally low (8, 8.3%) or extended (21, 21.9%) head and neck posture ($P < .001$). Root signature posture (12, 12.5%) was only observed in cases ($P < .001$). Six horses were neurologic cases and 6 had primary neck-related thoracic limb lameness. Neck locking (the neck being fixed in a low position, with the horse reluctant to move[2]) was only observed in cases (4, 4.2%; $P < .001$), 2 of which were neurologic, and 2 had primary neck pain or stiffness.

Lateral flexion of the neck was restricted in a higher proportion of cases (47/96, 49%) compared with controls (40/126, 31.7%; $P = .01$; RR, 1.5). Lateral flexion was performed by tilting the head in a higher proportion of cases (61/96, 63.5%) compared with controls (57/126, 45.2%; $P = .01$; RR 1.5). In addition, lateral neck flexion was asymmetrical to the left and to the right in a higher proportion of cases (41/96, 42.7%) compared with controls (31/126, 24.6%; $P = .004$; RR, 1.6). Limited extension of the neck was only observed in cases (22, 22.9%; $P < .001$).

Pain upon palpation over the caudal cervical articular process joints and transverse processes was only observed in cases (58, 60.4%; $P < .001$). Twenty-six (27.1%) cases stepped backwards and 16 (16.7%) stepped sideways when pressure was applied, compared with no controls (both $P < .001$). Extension of 1 or both thoracic limbs induced a pain response only in cases (29, 30.2%; P < .001). An abnormal reaction to light pressure applied to a thoracic limb was only observed in cases as follows: 13/95 (13.7%) in left and 10/95 (10.5%) in right thoracic limbs (both $P < .001$).

An abnormal stance during grazing was only observed in cases (61/96, 63.5%; $P < .001$). Twenty-eight cases straddled the thoracic limbs sideways, 31 spread the thoracic limbs widely with 1 thoracic limb protracted and the other retracted, and 2 crossed the thoracic limbs. When led at a walk or trot in straight lines, the neck was held low in 26 cases (27.1%) compared with only 2 (1.6%) control horses ($P < .001$; RR, 1.5). When turned in small circles the neck was held stiffly in 52 (54.2%) cases and 13 (10.3%) controls ($P < .001$; RR, 2.9).

3.4 | Dynamic examination

All 47 horses classified as neurologic exhibited ataxia, tetraparesis, postural reaction deficits (eg, limb interference, stepping on its own limbs), pivoting, circumduction, and had postural reaction and proprioceptive deficits. Neurologic alterations of gait, postural reactions and proprioceptive deficits were asymmetrical in 21 of 47 horses (1 side mildly worse: right 13/21, left 8/21). Horses classified as exclusively neurologic with no thoracic limb lameness (24/47) had a hypermetric gait of all limbs with upper motor neuron tetraparesis, whereas those with concurrent intermittent lameness or stumbling (23/47) had mild lower motor neuron paresis of the thoracic limbs with upper motor neuron paraparesis. Clinical signs, including grade of ataxia, of horses with concurrent thoracic limb lameness were exacerbated (grade increased by approximately 1 grade) by neck extension. Similarly, these horses worsened after light exercise comprising 5 minutes of luneging. Scoring of ataxia and proprioceptive deficits consisted of grades 1 (7/47), 2 (26/47), 3 (13/47), and 4 (1/47). Nine neurologic horses were not observed moving on the lunge because of the severity of their gait abnormalities. There was a decreased range of motion of the neck when moving on the lunge in 39/87 (44.8%) cases compared with 3 (2.4%) controls ($P < .001$; RR, 3.3). The head and neck were tilted during lungeing in 46/87 (52.9%) cases and 5 controls (3.9%; $P < .001$; RR, 3.6). Twenty-six horses with proprioceptive (spinal) ataxia had apparently weak stifles (bilateral, 25/26; symmetrical, 19/26, 1 side more affected, 6; and unilateral, 1/26), decreased quadriceps muscle mass, and intermittent mild delayed release of the patellae at the walk and trot. Horses with 1 side more affected had worse ipsilateral neurological deficits pertaining to spinal cord disease. EMG performed in 17 of 47 horses with spinal ataxia identified moderate to severe abnormalities worse in the ipsilateral side.

Fifty-seven horses were not evaluated ridden, 7 controls and 50 cases. Decreased range of motion of the neck during ridden exercise was observed in 2/120 (1.7%) controls compared with 20/46 (43.5%) cases ($P < .001$; RR, 5.0). Resistance to turning to the left or the right was observed in more cases 17/46 (37.0%) than controls 7/120 (5.8%; $P < .001$; RR, 3.5). Tilting of the head and neck was seen more frequently in cases (27/46, 58.7%) compared with control horses (16/120, 13.3%; $P < .001$; RR, 4.1).

Of the 31 horses with primary neck-related thoracic limb lameness only 18 (58.1%) showed lameness in hand (grade 1/8, $n = 3$; grade 2, $n = 6$; grade 3, $n = 3$; grade 4, $n = 4$; grade 5, $n = 1$; grade 6, and $n = 1$). Twenty-one (67.7%) and 20 (64.5%) horses exhibited lameness on the lunge on soft and firm surfaces, respectively, ranging from grades 1 to 6 (0–8 scale). Twenty-one of 31 horses with neck-related thoracic limb lameness were assessed ridden. In 16 of these horses (76.2%), lameness was more severe (increased at least 1 grade) when the horse was ridden to a contact (the presence of rein tension) compared with long reins (no rein tension), and in 6 horses (28.6%) lameness was more severe when ridden with long reins compared with to a contact. Eight horses with primary neck-related thoracic limb lameness only showed lameness when ridden, in addition to 1 horse with neck pain. One horse with primary neck-related thoracic limb lameness was not detectably lame when ridden but was extremely difficult to turn to the left. Lameness grades when ridden for primary neck-related thoracic limb lameness ranged from 0 to 6 (grade 0, $n = 1$; grade 2, $n = 1$; grade 3, $n = 6$; grade 4, $n = 5$; grade 5, $n = 4$; grade 6, $n = 4$).

Hopping-type thoracic limb lameness was only observed in cases and was seen in 30/96 horses (31.6%), in 22 of which the hopping-type thoracic limb lameness was seen either on the lunge or ridden. One additional thoracic limb lameness case showed elevation of the

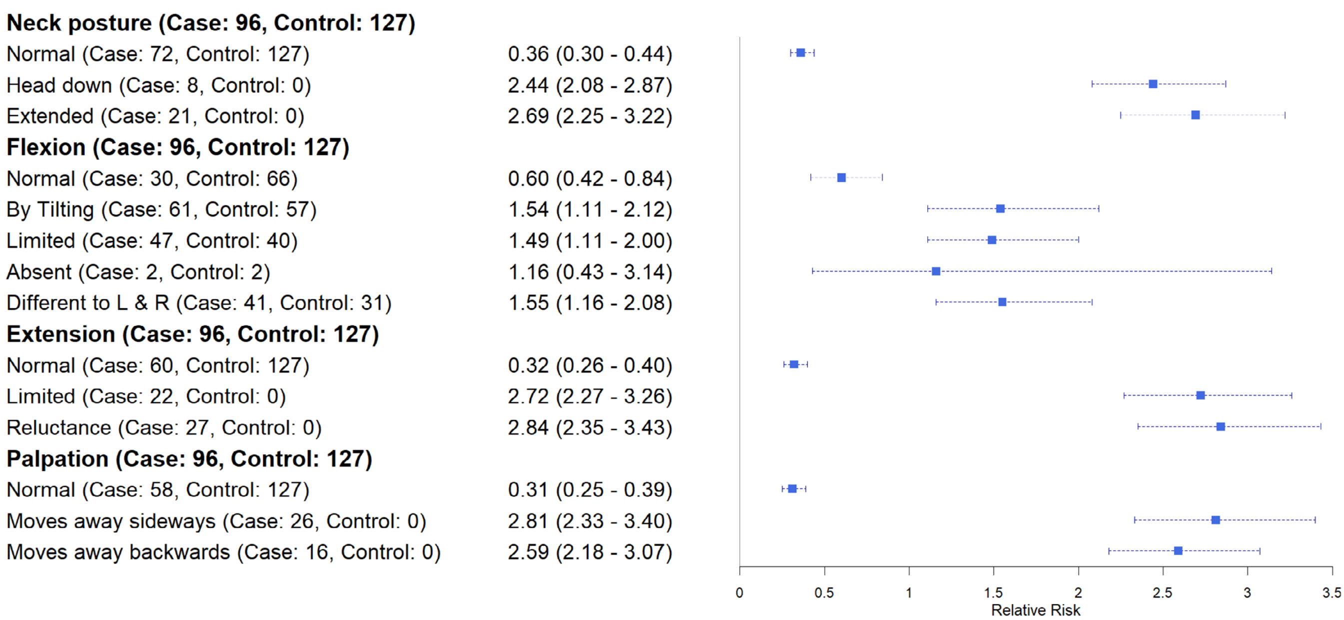

FIGURE 1 Comparison of cases (neck-related thoracic limb lameness, neck stiffness or pain or both, and neurologic; $n = 96$) and control horses ($n = 127$) illustrated using forest plots and providing relative risk values (blue boxes) and 95% confidence intervals (blue lines). Neck posture refers to the position of the head and neck at rest. Flexion refers to the ability of a horse to flex the neck to the left (L) and to the right (R). Extension refers to elevation of the head and neck in the sagittal plane. Palpation refers to the reaction to firm pressure applied to the caudal cervical articular processes and transverse processes.

head during protraction of the lame limb when ridden. Hopping type thoracic limb lameness was observed in 8 horses in hand and on the lunge; 4 of these horses also showed weakness and a propensity to stumble and were classified as primary thoracic limb lameness with mild neurologic deficits, whereas 4 were ataxic with proprioceptive deficits and classified as primary neurologic. In these horses, the

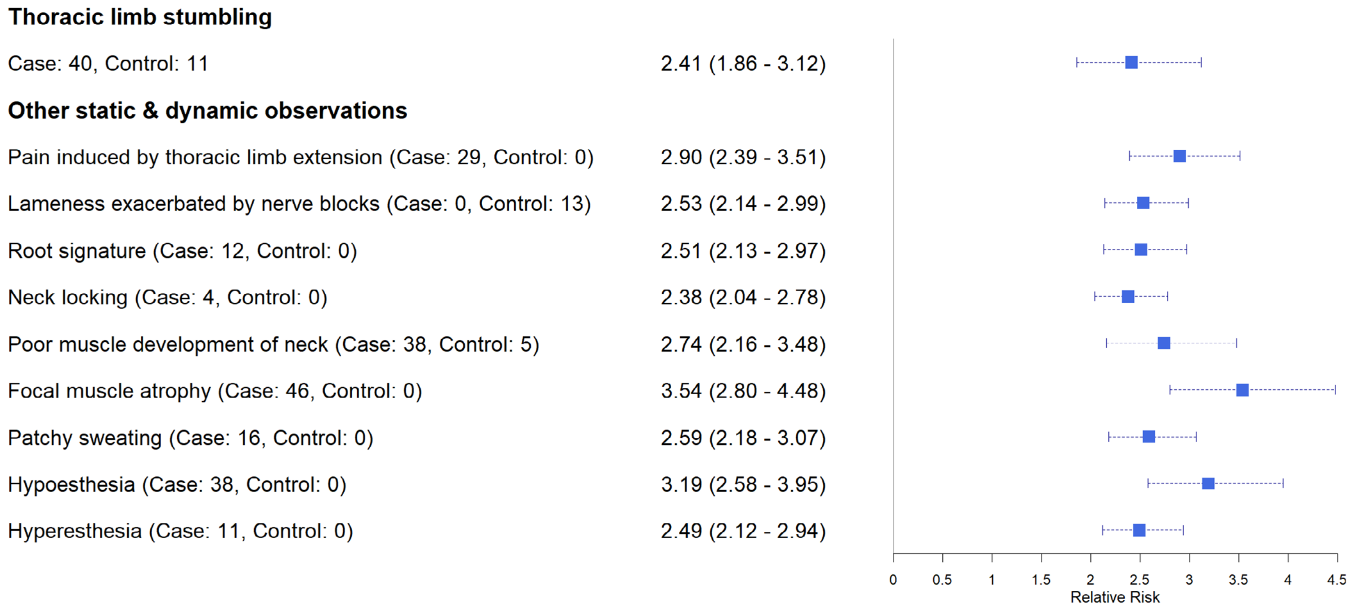

FIGURE 2 Comparison of cases (neck-related thoracic limb lameness, neck stiffness or pain or both, and neurologic; $n = 96$) and control horses ($n = 127$) illustrated using forest plots and providing relative risk values (blue boxes) and 95% confidence intervals (blue lines). Thoracic limb stumbling refers to stumbling either in hand, on the lunge or when ridden. Lameness exacerbated by nerve blocks refers to thoracic limb lameness, which was increased in severity after diagnostic anesthesia (perineural or intra-articular) performed proximal to the metacarpophalangeal joint.

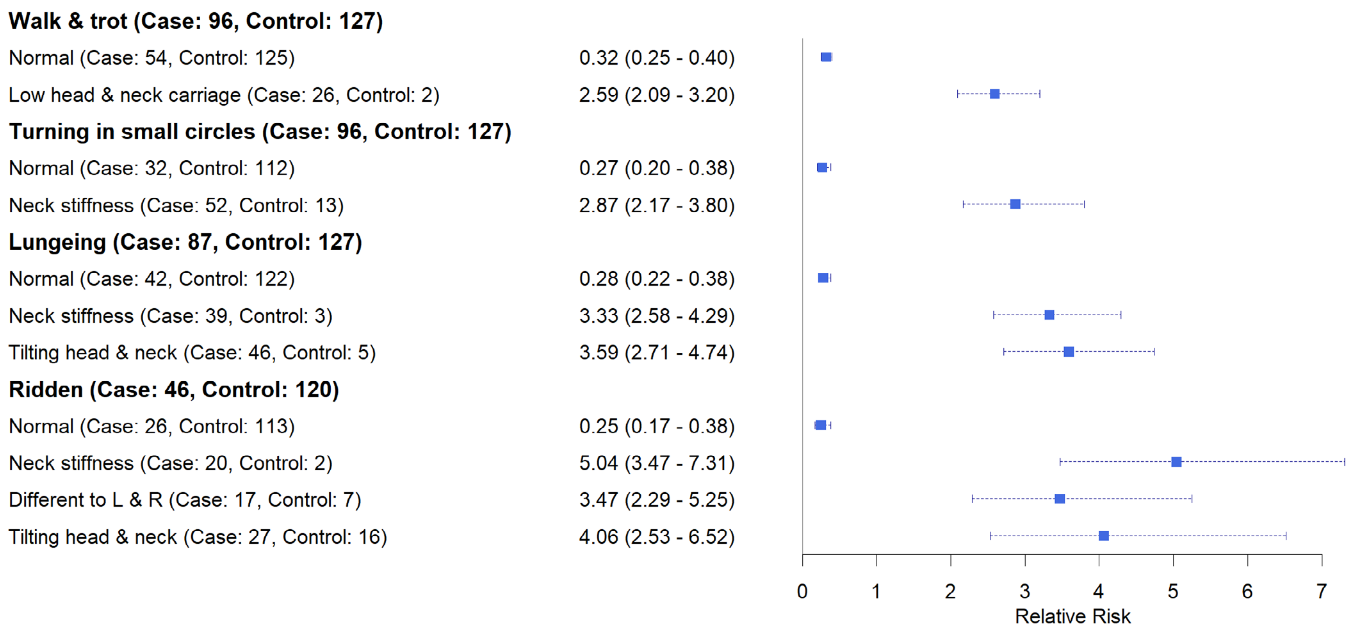

FIGURE 3 Comparison of cases (neck-related thoracic limb lameness, neck stiffness or pain or both, and neurologic; $n = 96$) and control horses ($n = 127$) illustrated using forest plots and providing relative risk values (blue boxes) and 95% confidence intervals (blue lines). Walk and trot refers to the posture of the head and neck when a horse was evaluated moving in hand in straight lines at walk and trot. Turning in small circles and neck stiffness refers to the posture and range of motion of the horse's neck when the horse was turned at walk about its own length to the left and right. Lunging refers to the posture and range of motion of the neck on the left and right reins in trot and canter (when safe to do so). For ridden exercise the range of motion of the neck, the ease of turning to left and right and the presence of a tilt of the head and neck were assessed.

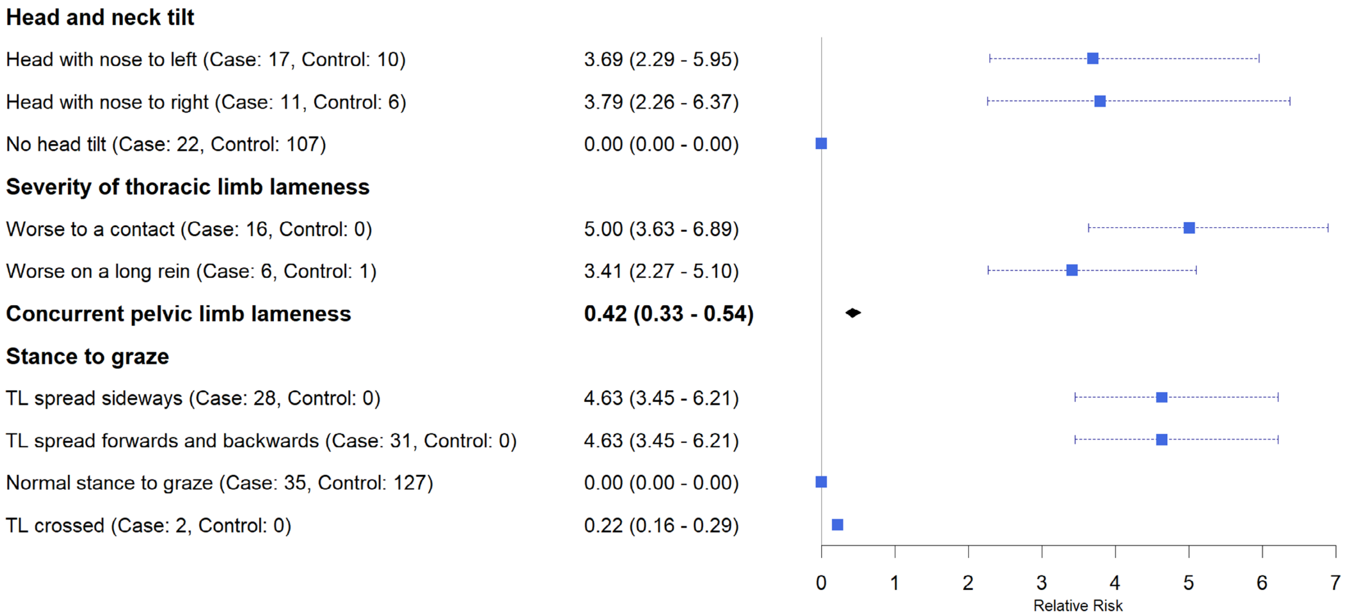

FIGURE 4 Comparison of cases (neck-related thoracic limb lameness, neck stiffness or pain or both and neurologic) and control horses illustrated using forest plots and providing relative risk values (blue boxes) and 95% confidence intervals (blue lines) during ridden exercise and when grazing. Head and neck tilt refers to the direction in which the nose pointed during ridden exercise. Severity of thoracic limb lameness refers to the accentuation of thoracic limb lameness by at least 1 grade (on a scale of 0–8), when ridden with or without rein tension. Stance to graze describes the position of the thoracic limbs (TL). Pelvic limb lameness (black diamond) was more likely to be seen in control horses compared with cases; 22/96 (22.1%) cases had concurrent pelvic limb lameness, which was abolished by diagnostic anesthesia (these do not include horses with delayed patellar release).

hopping type gait was most obvious when horses first started to trot and in transitions from walk to trot and from canter to trot. Lameness attributable to caudal cervical radiculopathy or neuropathy was confirmed by moderate to severe EMG abnormalities in these 8 horses.

Thoracic limb stumbling and tripping were seen with higher frequency in cases (40/96, 41.7%) compared with controls (11, 8.7%; $P < .001$; RR, 2.4). Intermittent stumbling in cases was observed in horses with neck pain or stiffness (17/18, 94.4%), neurologic dysfunction (23/47, 48.9%), and thoracic limb lameness (8/31, 25.8%). Although accentuation of lameness by palmar digital nerve blocks performed at the base of the proximal sesamoid bones was seen in some control horses, none showed exacerbation of lameness after more proximal nerve blocks. However, 13/31 (41.9%) cases with neck-related thoracic limb lameness showed deterioration in lameness by at least 1 grade after more proximal nerve blocks. Results including CI are presented in Table 1 and Figures 1–4. Moderate to severe EMG abnormalities were found in 10 of 23 horses with spinal ataxia and concurrent intermittent stumbling, and in 2 of 18 horses with neck pain or stiffness.

4 | DISCUSSION

In our study, the frequency of occurrence of clinical features considered likely to reflect primary neck stiffness or pain, neurological dysfunction and thoracic limb lameness was higher in horses with primary neck pathology compared with control horses. Some features, such as focal cervical muscle atrophy, patchy sweating on the neck, focal hypoesthesia and hyperesthesia of the neck and an abnormal posture to graze, were only seen in cases. Although all of the assessments were subjective, repeatable patterns were observed among horses with different primary problems, demonstrating the value of thorough standardized systematic clinical examinations performed by experienced specialists in a prospective manner.

Observation of localized or generalized neck muscle atrophy might be an important indicator of a neck-related problem. A simple method for semiobjective assessment of muscle mass has been described recently.[20] In our study, cases horses had lower body condition scores than control horses, perhaps reflecting both neurogenic and disuse muscle atrophy of the cervicothoracic region, and overall disuse muscle atrophy caudal to this region because of decreased muscle tone from altered neurologic input in horses with spinal ataxia, overall movement restriction, lack of regular physical activity, and in some cases decreased food intake secondary to neck pain. Pain upon palpation, hypoesthesia, and hyperesthesia also were important findings, as previously documented.[1,3,4,21,22] In our study, the frequency of occurrence of focal hyperesthesia was considerably less than reported in a retrospective study of horses with cervical vertebral compressive myelopathy (48% of 91).[21] Focal unilateral hypoesthesia previously was documented in a horse with neck-related thoracic limb lameness.[1] Alterations in cervical muscle tone and hypersensitivity to stimulation of myofascial trigger points might occur in association with cervical pain,[8] but were not assessed in all horses in our study.

It was suggested recently that the *brachiocephalicus* muscles are commonly painful on manual compression in horses with caudal cervical pain or stiffness.[8] The response to palpation of the *brachiocephalicus* muscles was not systematically compared between cases and controls in our study, although a routine part of the clinical assessment. In our experience, hypertonicity and pain upon palpation of the caudal aspect of the *brachiocephalicus* muscles either unilaterally or bilaterally are common observations in horses with a variety of primary sources of musculoskeletal pain and are not specific to caudal cervical pain, as previously reported.[2] However, subtle lameness attributable to primary *brachiocephalicus* pain has been documented.[2] Tactile hyperesthesia of 1 or both thoracic limbs was observed in our study in cases but not in control horses. Apparent paraesthesia was not detected in any horse, but it could be challenging to identify and interpret. Paraesthesia has been reported involving the antebrachium of 1 or both thoracic limbs and characterized by scratching the limb with the teeth, in association with other likely neck-related clinical signs.[23]

An abnormally low neck posture at rest and when moving was observed in horses with primary neck problems, as has been described previously.[8] However, a similar stance at rest has been observed in horses described as withdrawn or lethargic, probably secondary to chronic musculoskeletal pain, and not necessarily related to the neck region.[24,25] The neck becoming "stuck" in a fixed low position, with or without some neck rotation, for variable periods of time (so-called "neck locking") is a poorly documented condition.[2,4,17,22] Its transient nature means that the clinical signs cannot be reliably replicated in a horse with a history of this complaint. However, in our experience, during an episode severe pain often is present, with the horse being reluctant to move, exhibiting focal hyperesthesia, with or without localized sweating.

Horses with normal cervical mobility can readily flex the neck to the left and right and position the muzzle near the girth region, tuber coxae, or tarsus.[26] However, similar to the thoracolumbar region of humans (eg, the ability to touch the toes with the palm of the hand), the range of cervical flexion is probably variable among horses performing normally. In our study, the range of motion, the symmetry of flexion to the left and right and the way in which horses flexed the neck differed between cases and controls, indicating that together with other clinical signs restricted neck flexion might indicate cervical pathology in some horses. Some horses with restricted cervical flexion placed the ipsilateral thoracic limb cranially, however the frequency of occurrence of this observation was not recorded. Tilting of the head when on the lunge or ridden is a non-specific indicator of musculoskeletal pain and is 1 of the 24 behaviors of the Ridden Horse Pain Ethogram (RHpE),[27] which decreases in frequency of occurrence after removal of pain using diagnostic anesthesia.[28,29] Horses with neck pain might tilt the head, but careful observation often reveals that there is a tilt of both the neck and head,[4] as observed in our study.

Root signature posture was only observed in neurological cases and horses with primary neck-related thoracic limb lameness, as previously documented.[1,4] This posture might be observed at rest, or after a period of exercise, and might resolve rapidly. Thoracic limb lameness associated with a caudal cervical lesion might only be present during ridden exercise, as observed previously,[30] emphasizing the importance of ridden exercise as part of an evaluation of poor performance. It is important to recognize that the presence of lameness and its severity might be influenced by the position of the neck, and assessment of the horse ridden with and without rein tension is important, as indicated in our study and previous observations.[4,29] Lameness associated with cervical radiculopathy often is associated with high RHpE scores.[29]

Repetitive thoracic limb or pelvic limb stumbling is another non-specific sign of musculoskeletal pain and is another of the 24 behaviors of the RHpE. In our study, thoracic limb stumbling was seen with higher frequency in cases compared with controls, although control horses included lame horses, and among cases was observed in horses with ataxia or neck pain or stiffness or both, as previously documented,[2,15,17] and in a small proportion of horses with neck-related thoracic limb lameness as previously observed.[3,4] Neck pain, compression of the caudal cervical and cranial thoracic spinal cord segments, compression of nerve roots or nerves in the cervicothoracic region or some combination of these can cause or contribute to a propensity to stumble in horses with neck pathology. Other gait alterations associated with caudal neck pain in our horses included a short stepping thoracic limb gait.

Hopping type thoracic limb lameness was first described in ridden horses[3] but subsequently was observed on the lunge.[2] In our study hopping type thoracic limb lameness was most frequently seen in ridden horses with primary neck-related thoracic limb lameness but also was seen in hand and more clearly on the lunge in 8 horses (4 with weakness and stumbling, reflecting concurrent neurological dysfunction, and 4 with spinal ataxia and proprioceptive deficits). To our knowledge, this finding has not been described before.

Observation of transitions between gaits (eg, walk, trot, and canter) is important when assessing gait abnormalities. A common observation in horses with mild ataxia is an irregularly irregular pelvic limb rhythm and variability in height of pelvic limb steps when decelerating from trot to walk. The difference in pelvic limb kinetics between ataxic and lame horses has been quantified previously.[31] Primary pelvic limb lameness and neck-related problems might co-exist. However, clinical signs mimicking a primary neck problem, such as an abnormally high head and neck posture when ridden, uneven rein tension, and difficulties in turning might be induced by primary pelvic limb lameness.[2,29,30] Twenty-two of 96 (22.9%) AHT case horses in our study exhibited concurrent pelvic limb lameness, which was abolished by diagnostic anesthesia of the affected limb, indicating a lameness not associated with a neck problem. In contrast, bilateral delayed release of the patella was observed commonly in horses with neurologic disease in our study and attributed to decreased muscle tone, strength, and disuse muscle atrophy because of deficient upper motor neuron output to the pelvic limbs as the result of a C1-T2 myelopathy. The quadriceps muscle, the main supporting extensor muscle of the stifle, is of particular importance. Furthermore, diagnostic anesthesia did not abolish delayed release of patella, and the side that was most affected was ipsilateral to the worst neurologic deficits in horses with spinal ataxia.

It is well-recognized that desensitization of the foot by perineural anesthesia might result in an increase in lameness severity if the foot is not the primary source of pain causing lameness.[32] Deterioration in lameness after more proximal limb nerve blocks previously has been reported in horses with suspected neck-related thoracic limb lameness,[3,4] as was observed in 42% of the horses in our study. This figure might be an underestimate. All horses examined in the UK underwent diagnostic anesthesia performed by SD, and 11/13 (85%) showed deterioration of lameness, whereas in most horses examined in the USA, diagnostic anesthesia was performed by the referring veterinarian, who might not have observed, recorded, or communicated any deterioration in lameness.

Electromyography proved to be useful to further map the specific location of affected spinal cord segments in horses with spinal cord disease or nerve roots or nerves in horses with thoracic limb lameness, stumbling, and weakness. The EMG abnormalities observed in the latter horses supported the suspicion of a lameness of neurologic origin because of spinal nerve root or nerve compression by finding alterations in specific muscle groups innervated by corresponding nerve roots and nerves. Similar observations were made by other authors.[33] Although EMG was useful to support neuroanatomical localization and thoracic limb lameness of neurologic origin in our horses, lack of abnormal EMG findings of specific muscle groups does not rule out spinal cord or nerve root disease (MA). Lack of EMG abnormalities could result from site tested, patchy distribution of alterations, stage of disease (eg, acute, subacute, chronic), and muscle atrophy or fibrosis.[19] Histologic studies in a small number of horses with neck-related thoracic limb lameness have verified the presence of cervical radiculopathy.[1,3,4] A study using quantitative EMG demonstrated accurate prediction of nerve root lesions in the caudal cervical region.[33] With the introduction of foraminotomy for treatment of cervical spinal nerve compression,[34] EMG might become increasingly important for case selection for surgery in addition to advanced imaging.

In our study, show jumping horses were overrepresented and dressage horses were underrepresented among cases compared with control horses. This observation was interesting given the controversy surrounding hyperflexion in dressage horses and whether it imposes an injury risk, and the observation that upper level jumping horses might compete successfully despite radiologic evidence of caudal cervical articular process joint osteoarthritis.[14] Both sudden onset severe lameness associated with ipsilateral caudal cervical hyperesthesia or severe stumbling sometimes resulting in a fall have been observed in show jumping horses with primary cervicothoracic problems (SD, MA). The caudal neck region is in extension when landing from a fence and the ground reaction forces in the thoracic limbs at landing, propagated proximally, with engagement of the muscles of the thoracic sling, combined with the descending mass of the horse's trunk, and the need to shift the center of gravity caudally require optimal neuromuscular function and musculoskeletal strength and coordination for ideal performance.[35,36] The intervertebral foramina are smaller in extension than flexion,[37] with a higher likelihood of nerve root compression in extension compared with flexion. An additional clinical presentation in show jumping horses with primary caudal neck problems is repeated decreased flexion of 1 thoracic limb in the airborne phase of the jump, which might be neurologically or pain mediated (SD, MA).

Our study had some limitations. It previously has been documented that horses with caudal neck pain might show pain if 1 thoracic limb is picked up and the horse is pushed sideways, having to hop on the contralateral limb,[1] however the response to hopping was not assessed in all of our horses. The response to neck flexion with the ipsilateral thoracic limb flexed[38] was not routinely evaluated. Gait variations in canter were not documented for all horses, although a shortened thoracic limb step length and decreased height of arc of foot flight in association with caudal neck pathology have been described previously.[1,4,30] Only a small proportion of the control horses had no clinical abnormalities (horses undergoing pre-purchase examinations); the remainder had musculoskeletal pain that was abolished by diagnostic anesthesia. The observations from only 2 clinicians provided a consistent approach, but also a potential for bias. These results relate to WB horses and might not apply to other breeds.

In conclusion, many clinical features described in our study were seen either only in horses with neck-related problems or with significantly higher frequency in cases compared with control horses. Systematic observation of the presence or absence of these signs should aid differentiation between horses with caudal cervical lesions and horses with other causes of gait abnormalities. The use of EMG can support neurological dysfunction and further localize the anatomical site or sites.

ACKNOWLEDGMENT

Funding provided by gifts from anonymous donors toward the Equine and Comparative Neurology Research Group, UC Davis (Monica Aleman), Gift#V435104. The authors thank the referring clinicians.

CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Approved by the Clinical Ethical Review Committee of the Animal Health Trust (AHT 33-2016) and the University of California Davis IACUC #19891.

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Dyson S, Zheng S, Aleman M. Primary phenotypic features associated with caudal neck pathology in warmblood horses. *J Vet Intern Med*. 2024;38(4):2380-2390. doi:10.1111/jvim.17125