ORIGINAL ARTICLE

Head tilt as a clinical sign of cervical spinal or paraspinal disease in dogs: 15 cases (2000-2021)

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[Correction added on 08 January 2024 after first online publication: The article title has been updated in this version.]

Objectives: To characterise head tilt as a rare clinical sign of cervical spinal or paraspinal disease in dogs.

Materials and Methods: Retrospective single-centre case-series study of dogs with head tilt and cervical spinal or paraspinal disease in the absence of intracranial abnormalities. Descriptive statistics were used.

Results: Fifteen dogs met the inclusion criteria of this study. Median age at onset was 6 years (range 2.5 to 12 years). Onset of neurological signs was mainly chronic (9/15, 60%). Most common presenting complaints included head tilt (9/15, 60%) and cervical hyperaesthesia (8/15, 53%). Most common neurological findings included head tilt (15/15, 100%), generalised proprioceptive ataxia and tetraparesis (6/15, 40%) and cervical hyperaesthesia (8/15, 53%). Diagnoses included post-operative complication of C2 spinal nerve root mass removal (2/15, 13%), C3-C4 intervertebral disc extrusion (2/15, 13%), cervical paraspinal myositis (2/15, 13%) and one of each: C2 vertebral malformation, C2 spinal nerve root mass, C1-C2 meningioma, C2 vertebral fracture, C4-C5 intervertebral disc extrusion, C4 vertebral body mass, C5-C7 osseous-associated cervical spondylomyelopathy, and concurrent C5-C6 and C6-C7 intervertebral disc protrusions. Two dogs were euthanased shortly after diagnosis and two of 15 were dogs lost to follow-up. No *post-mortem* examination was performed for these cases. For the 11 of 15 remaining dogs, head tilt resolved in eight of 15 (53%) dogs after treatment of the underlying condition and in three of 15 (20%) dogs, it remained static.

Clinical Significance: Head tilt can be a rare clinical sign of cervical spinal or paraspinal disease in dogs.

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INTRODUCTION

Head tilt (*later torticollis* or *laterocollis*) is described as a rotation of the head around the first cervical vertebra or atlas, such that one of the ears is held lower than the other. This is known to be a clinical sign indicative of unilateral vestibular syndrome (Ferreira *et al.* 2007, Muñana 2014).

Although head tilt in the veterinary literature remains strongly associated with vestibular dysfunction, this clinical sign has been associated with a wider aetiological variety in human medicine. Underlying causes of head tilt in human medicine include congenital muscular (*e.g.* sternomastoid "tumour" of infancy), osseous (*e.g.* atlanto-axial subluxation), central/peripheral nervous system (brain – posterior fossa or basal nuclei, cervical spinal cord or spinal nerve root), ocular (*e.g.* strabismus) and soft tissue (*e.g.* retropharyngeal abscess; cervical muscle abscess) disease; whilst there is also a paroxysmal phenotype (*i.e.* Sandifer's syndrome, benign paroxysmal torticollis, spasmodic cervical dystonia etc.) (Tomczak & Rosman 2012, Beasley 2021).

There is currently little known about head tilt in cervical spinal and paraspinal disease in dogs (Harris *et al.* 2011, Fernandes *et al.* 2019, De Lahunta *et al.* 2021a, De Lahunta *et al.* 2021b). The aim of this study is to characterise head tilt as a clinical sign of dogs with cervical spinal or paraspinal disease.

MATERIALS AND METHODS

The digital medical database of the Royal Veterinary College was retrospectively searched to retrieve the records of all dogs presenting with head tilt between January 1, 2000 and November 1, 2021. Initial search terms were "head tilt," and then, sub search terms included "head tilt," and "spinal cord" or "cervical" or "neck." Inclusion criteria were (1) complete medical records, (2) head tilt clearly described and documented in the neurological examination that occurred no longer than 6 months before presentation, (3) advanced diagnostic imaging [CT or/and magnetic resonance imaging (MRI)] of the head and cervical region and (4) presence of a lesion at the cervical region in the absence of intracranial abnormalities on advanced imaging studies. The reasoning behind including head tilts of no longer than 6 months onset was an effort to avoid recruitment of cases with historical head tilt that could be attributable to previously diagnosed "idiopathic geriatric peripheral vestibular disease," "otitis media/interna" or other previous but now resolved pathology with a residual head tilt. Cases with (1) historical head tilt, (2) presence of middle ear effusion or other intracranial abnormalities on diagnostic imaging, (3) a diagnosis of Chiari-like malformation and associated syringomyelia and (4) a clinical suspicion or laboratory diagnosis of metabolic disease such as hypothyroidism were excluded.

Complete medical records included signalment, clinical history (onset and duration of neurological signs), clinical and neurological examination findings and advanced diagnostic imaging findings. Advanced diagnostic imaging included CT with a 16-slice helical scanner (PQ 500, Universal Systems, Solon; GE Healthcare) under sedation or general anaesthesia or MRI with a high-field unit (1.5 T, Intera; Phillips Medical Systems) under general anaesthesia. All diagnostic imaging studies were reviewed by a board-certified neurologist. Some dogs underwent additional diagnostic tests, including clinical pathology (complete blood count, serum biochemistry, thyroid profile and infectious disease tests), cerebrospinal fluid (CSF) analysis, conventional radiography or abdominal ultrasound, which were reviewed where available.

Onset of neurological signs was categorised into hyperacute (<24 hours), acute (1 to 7 days), subacute (7 to 15 days) and chronic (>15 days) (Harrison *et al.* 2021). All clinical and neurological examinations were performed by a board-certified neurologist or a neurology resident under the direct supervision of a board-certified neurologist.

Statistical analysis was descriptive, where median [interquartile range (IQR)] was used.

RESULTS

Initial search revealed 2881 cases with head tilt over the period 2001 to 2021. Of these, 677 cases were identified to correspond to search terms regarding spinal cord pathology. Of these, only 15 cases met the inclusion criteria (Table 1). Duplicates, cases with concurrent hypothyroidism, middle ear effusion or Chiari-like malformation and syringomyelia, or cases identified with random use of the search terms but not associated with underlying pathology were excluded.

Clinical presentation

Breeds represented were: French Bulldog (n=4) and one of each: Staffordshire Bull Terrier, West Highland White Terrier, Labrador retriever, boxer, Dogue de Bordeaux, Yorkshire terrier, Chow-Chow, German shepherd dog, Lurcher, Bull Mastiff-Cross and crossbreed. Twelve dogs were males (seven neutered) and three were females (one neutered). Median age at presentation was 6.1 years (IQR: 4.8 years, range: 2.5 to 12 years). Onset of neurological signs was chronic (9/15, 60%), acute (4/15, 26.7%) and hyperacute (post-operatively) (2/15, 13%). Mean duration of clinical signs was 30 days (IQR: 72.5 days, range: 1 to 120 days). Presenting complaints as reported by the owners included cervical hyperaesthesia (8/15, 53%), head tilt (9/15, 60%) of which two of nine reported to be episodic, incoordination (2/15, 13%), lethargy (2/15, 13%), collapse episode (2/15, 13%), neck spasms (2/15, 13%) and one of each (1/15, 7%): ambulatory tetraparesis, falling to one side, scuffing pelvic limbs, episodic pain, body turn, reluctance to exercise, episodic weakness, lameness, stiff gait, kyphosis and knuckling. One dog was referred for a witnessed road-traffic accident. Two dogs developed head tilt immediately after undergoing a C2 modified hemilaminectomy, durotomy, rhizotomy (n=2) with myelotomy (n=1) for resection of a C2 spinal nerve root mass.

On neurological examination, mentation was normal in all dogs. Posture revealed head tilt in all dogs, which was right-sided in nine dogs and left-sided in the remaining six dogs (Fig 1). Head tilt was episodic in three of 15 (20%) dogs. Two (13%) dogs had a low-head carriage and two (13%) dogs had a head turn with pleurothotonus ipsilateral to the head tilt. Gait and postural reaction analysis revealed ambulatory tetraparesis with generalised proprioceptive ataxia in all limbs (6/15, 40%), or non-ambulatory tetraparesis (1/15, 6%). Proprioceptive deficits were detected in eight of 15 (53%) dogs, which was symmetrical in three dogs and worse ipsilateral to the side of the head tilt in five dogs. Leaning towards the side of head tilt was noticed in two dogs (13%), whilst falling towards the side of the head tilt was noticed in another dog (1/15, 7%%). Spinal reflexes were intact in all dogs. Ipsilateral ventral positional strabismus was noticed in one (7%) dog, whilst ipsilateral miosis was noticed in one (7%) dog. On palpation, cervical hyperaesthesia was observed in eight (53%) dogs, whilst symmetrical cervical muscle atrophy ventral to the nuchal ligament was noticed in one (7%) dog. The neuroanatomical localisation was consistent with a C1-C5 myelopathy (9/15; 60%), C1-C5 or C6-T2 myelopathy (1/15, 7%), multifocal with involvement of the C6-T2 spinal cord segments and central vestibular system (1/15, 7%) or central vestibular system (1/15, 7%). In three dogs (20%), the neurological examination did not reveal any deficits and the observed head tilt was considered secondary to cervical hyperaesthesia.

Table 1. Signalment, diagnostics, diagnosis and outcome of dog with head tilt in cervical spinal or paraspinal disease

C	Signalment	Onset of signs and presenting complaints	Neurological findings	Diagnostic test results	Clinical diagnosis	Treatment	Outcome of head tilt
1	7yo ME French bulldog	Onset: hyperacute Presenting complaints: L head tilt after surgery for L C2 nerve root mass	NE: L head tilt; ambulatory tetraparesis; symmetric generalised proprioceptive ataxia NAL: L C1-5 SCS	Infectious disease tests: IFA – *Toxoplasma gondii* IgG/IgM: negative IFA – *Neospora caninum* IgG: negative MRI (pre-op): intradural extramedullary mixed T2W intense and uniformly contrast enhancing mass lesion at C1-C2 affecting the L nerve root and compressing the spinal cord. Muscle atrophy of the muscles innervated by C2 spinal nerve CSF analysis (cisternal): mildly inflammatory Thoracic radiography: WNL Abdominal radiography: WNL Histopathology: malignant peripheral nerve sheath tumour	Post-operative complication after C2 spinal nerve root tumour (MPNST) surgery	Prednisolone Hydroxyurea L C1-2 modified hemilaminectomy, durotomy, rhizotomy	Residual head tilt Development of C3 root mass in 2 m post-op
2	5.7 yo MN Staffordshire bull terrier	Onset: hyperacute Presenting complaints: L head tilt after surgery for L C2 nerve root mass	NE: L head tilt; ambulatory tetraparesis; symmetric generalised proprioceptive ataxia NAL: L C1-C5 SCS	Coagulation profile: WNL Infectious disease tests: ELISA – *Angiostrongylus vasorum*: negative MRI (pre-op): intradural extramedullary lesion on the left side causing dorsolateral compression of the spinal cord at C1-C2 Thoracic radiography: WNL Abdominal ultrasound: WNL Histopathology: benign peripheral nerve sheath tumour	Post-operative complication after C2 spinal nerve root tumour (BPNST) surgery	Lomustine L C1-2 modified hemilaminectomy, durotomy, myelotomy, rhizotomy	Resolved
3	11.4 yo FE West Highland white terrier	Onset: chronic Presenting complaints: scuffing thoracic limbs	NE: L head tilt; ambulatory tetraparesis (left worse than right); generalised proprioceptive ataxia; postural reaction deficits NAL: L C1-5 SCS	CBC: WNL SB: ALB: 40.9 g/L (RI: 26.3 to 38.2) MRI: left cranial cervical intradural-extramedullary masses with extension along the left C2 and C3 nerve roots into the paravertebral musculature. Secondary marked left cervical neurogenic muscle atrophy	L C2 and C3 spinal nerve root mass	Palliative treatment (gabapentin)	Lost to follow-up
4	10.9 yo MN Labrador retriever	Onset: chronic Presenting complaints: head tilt, hemiparesis, neck spasms and pain	NE: R head tilt; R head turn; R pleurothotonus; ambulatory tetraparesis (right worse than left); generalised proprioceptive ataxia; postural reaction deficits; cervical hyperaesthesia NAL: R C1-5 SCS	CBC: NEUT:11.89 ×10e9/L (RI: 3 to 11.5); LYMPHO: 0.51 ×10e9/L (RI: 1 to 4.8) SB: CK: 1159 U/L (RI: 67 to 446) MRI: large tubular mass-like structure which extends along the expected course of the right C2 nerve and invades intramedullary causing marked compression and infiltration of the spinal cord with contrast-enhancement. A tubular portion of the mass extends laterally into the adjacent paravertebral musculature and extends caudoventrally along the surface of C2 in multilobulated shape	R C2 spinal nerve root mass	Palliative treatment (prednisolone; gabapentin)	Euthanasia

Table 1. (Continued)

C	Signalment	Onset of signs and presenting complaints	Neurological findings	Diagnostic test results	Clinical diagnosis	Treatment	Outcome of head tilt
5	10 yo FN Crossbreed	Onset: chronic Presenting complaints: head tilt, neck pain	NE: L head tilt; ambulatory tetraparesis (left worse than right); generalised proprioceptive ataxia; postural reaction deficits; cervical hyperaesthesia NAL: L C1-5 SCS	CBC: LYMPHO: 0.76109/l (RI: 1.5 to 7) SB: UREA: 2.30 mmol/O (RI: 3 to 9.1); CREA: 86 umol/L (RI: 98 to 163) MRI: extradural mass at the level of C4 vertebral body adjacent to the vertebral canal on the left severely compressing the spinal cord CSF analysis (cisternal): WNL Thoracic radiography: WNL Abdominal ultrasound: multiple cystic lesions in the spleen, (DDx: myelolipomas or regenerative nodules)	L C4 vertebral body mass	Palliative treatment (fentanyl patch)	Euthanasia
6	7 yo FE Dogue De Bordeaux	Onset: chronic Presenting complaints: head tilt, neck pain, lethargy, lameness and knuckling of one limb	NE: R head tilt; postural reaction deficits; patellar hyperreflexia NAL: C1-5 SCS	CBC: mild anaemia MRI: right-sided extramedullary intradural mass strongly contrast enhancing causing compression to the spinal cord at the level of C1-2 Thoracic radiography: WNL Abdominal ultrasound: WNL	R C1-2 spinal cord mass (papillary meningioma)	Surgical treatment (C1-2 hemilaminectomy and removal of mass) Medical treatment (prednisolone; hydroxyurea)	Resolved
7	3.4 yo MN French bulldog	Onset: acute Presenting complaints: head tilt, neck pain, thoracolumbar scoliosis	NE: R head tilt; thoracolumbar scoliosis to the right; cervical hyperaesthesia; four limb spinal hyperreflexia; cervical hyperaesthesia NAL: C1-5 SCS	MRI: intervertebral disc extrusion at C3-C4 and a mild bulging of the C2-C3 intervertebral disc with secondary spinal cord compression CSF analysis (cisternal): WNL	C3-4 IVDE	Surgical treatment (C3-4 ventral slot) Medical treatment (carprofen; diazepam)	Resolved
8	4.5 yo MN French bulldog	Onset: acute Presenting complaints: neck pain, reluctance to exercise	NE: L head tilt; non ambulatory tetraparesis; postural reaction deficits NAL: C1-5 SCS ±central vestibular system	Venous blood gas analysis: WNL MRI: extradural material causing moderate ventral and left sided spinal cord compression at the level of C3-4 intervertebral disc space consistent with extrusion CSF analysis (cisternal): WNL	L C3-4 IVDE	Surgical treatment (L C3-4 dorsal hemilaminectomy) Medical treatment (gabapentin; paracetamol)	Resolved
9	4.6 yo ME French bulldog	Onset: chronic Presenting complaints: neck pain	NE: low-head carriage; R head tilt; cervical hyperaesthesia NAL: normal with cervical hyperaesthesia	MRI: right-sided extradural material compressing the spinal cord consistent with C4-5 intervertebral disc extrusion, and syringomyelia at C2-C4 likely associated with altered CSF flow secondary to compression at C4-5 Thoracic radiography: aspiration pneumonia	R C4-5 IVDE & C2-4 syringomyelia	Surgical treatment (C3-4 ventral slot) Medical treatment (meloxicam)	Resolved

Table 1. (Continued)

C	Signalment	Onset of signs and presenting complaints	Neurological findings	Diagnostic test results	Clinical diagnosis	Treatment	Outcome of head tilt
10	12 yo ME Yorkshire terrier	Onset: chronic Presenting complaints: episodic pain; episodic weakness	NE: R head tilt; postural reaction deficits; thoracic limb hyporeflexia NAL: C6-T2 SCS	CBC: WNL SB: WNL MRI: intervertebral disc protrusion and some compression of the spinal cord at C5-C6 and also at C6-C7 with central canal dilation (syringomyelia) at C2-C4 and C5-C6. Normal brain Thoracic radiography: WNL Abdominal ultrasound: bladder calculi; prostatic cyst ACTH Stim Test: WNL	C5-6 and C6-7 IVDPs & C2-6 syringomyelia	Surgical treatment (C5-C6 ventral slot)	Resolved
11	3.1 yo MN boxer	Onset: chronic Presenting complaints: head tilt; episode of collapse; weakness; incoordination	NE: R head tilt; ambulatory tetraparesis; generalised proprioceptive ataxia; postural reaction deficits NAL: C1-5 or C6-T2 SCS	MRI: asymmetrical vertebral articular processes at C5/6 and C6/7 with evidence of enlargement of left one, causing impingement on dorsal and lateral aspect of dura. Multiple small fluid-containing structures associated with the synovial cysts at C4/5-C6/7 are identified. Consistent with osseous-associated cervical spondylomyelopathy	C5-7 OA-CSM	Medical treatment (meloxicam)	Residual
12	8.7 yo ME Chow-Chow	Onset: chronic Presenting complaints: episode of collapse; weakness; incoordination	NE: R head tilt; ambulatory tetraparesis; generalised proprioceptive ataxia; postural reaction deficits; ipsilateral positional ventral strabismus; ipsilateral miosis NAL: central vestibular system	CBC: WNL SB: WNL TT4/TSH: WNL MRI: focal spinal cord atrophy at C1 secondary to impingement by the dens CSF analysis (cisternal): albuminocytological dissociation Cervical radiography: uneven dorsal aspect of the vertebral body of C2, possible exostosis; no malalignment identified	C2 vertebral malformation with spinal cord compression	None	Lost to follow-up
13	6 yo ME cocker spaniel	Onset: chronic Presenting complaints: head tilt; previous road-traffic accident 2 months ago	NE: R head tilt; cervical hyperaesthesia NAL: C1-5 SCS	CT: displacement of the C2 fragments (fracture). The displacement is not causing compression/narrowing of the spinal canal and there is evidence of fibrotic fracture healing	C2 vertebral fracture and displacement with associated R impingement of the spinal cord	Medical treatment (gabapentin; carprofen)	Residual
14	6.1 yo MN Lurcher	Onset: acute Presenting complaints: head tilt; neck spasms; neck pain; lethargy; stiff gait	NE: L head tilt; L head turn; cervical hyperaesthesia NAL: normal with cervical hyperaesthesia	CBC: WNL SB: WNL Infectious disease tests: IFA – Toxoplasma gondii IgG/IgM: negative IFA – Neospora caninum IgG: negative MRI: T2W and STIR hyperintensity of the paravertebral musculature, ventral to C2/3, lateral to C4-T1, within the right lateral musculature at C6, longus colli muscles with diffuse paravertebral muscle contrast enhancement. R>L paravertebral myositis considered most likely CSF analysis (cisternal): WNL Cytology (FNA of epaxial muscles): WNL	Cervical paravertebral myositis	Medical treatment (prednisolone; metronidazole; amoxyclav)	Resolved

Table 1. (Continued)

C	Signalment	Onset of signs and presenting complaints	Neurological findings	Diagnostic test results	Clinical diagnosis	Treatment	Outcome of head tilt
15	2.5 yo MN GSD	Onset: acute Presenting complaints: neck pain; kyphosis	NE: R head tilt; low-head carriage; cervical hyperaesthesia NAL: normal with cervical hyperaesthesia	CBC: WNL SB: ALB: 25.5 g/L (RI: 28 to 39) MRI: there is an ill-defined T2W increased signal intensity with T1W post contrast enhancement within the right epaxial musculature adjacent to C4 and C5 vertebrae. The abnormal signal intensity extends along the muscular fascia to the right prescapular region where there is also T2W increased signal intensity, best seen on dorsal STIR sequence. Abnormalities of the epaxial musculature and related fascia of suspected inflammatory origin (e.g. cellulitis, immune mediated or infectious). Mild abnormalities of the right articular facet joint at C3/4, C4/5 and C5/6, causing minimal impingement of the spinal cord at C5/6. Regional lymphadenopathy (most likely reactive) CSF analysis (cisternal & lumbar): albuminocytological dissociation Cytology (FNA of epaxial muscles): WNL	Cervical paravertebral myositis	Medical treatment (gabapentin; firocoxib)	Resolved

ALB Albumins, BPNST Benign peripheral nerve sheath tumour, C Case number, CBC Complete blood count, CK Creatine kinase, CSF Cerebrospinal fluid, DDx Differential diagnoses, FE Female entire, FN Female neutered, FNA Fine needle aspiration, h Hours, IFA Indirect immunofluorescence, IV Intravenous, IVDE Intervertebral disc extrusion, IVDP Intervertebral disc protrusion, L Left, m Months, ME Male entire, MN Male neutered, MPNST Malignant peripheral nerve sheath tumour, NAL Neuroanatomical localisation, NE Neurological examination, OA-CSM Osseous-associated cervical spondylomyelopathy, PL Pelvic limb, PO Per os, R Right, RI Reference intervals, SB Serum biochemistry, SC Subcutaneous, SCS Spinal cord segments, TL Thoracic limb, TNCC Total nucleated cell count, WNL Within normal limits, Y Yes

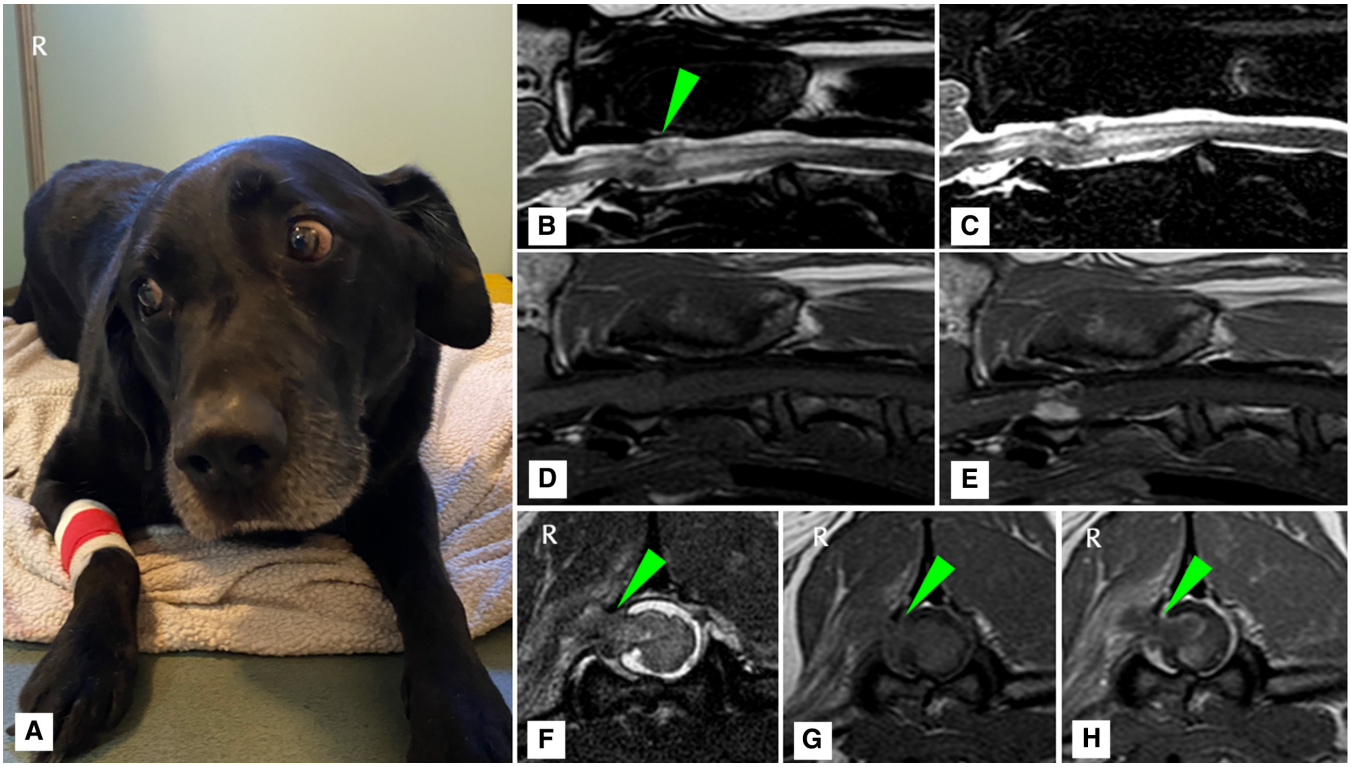

FIG 1. (A) Photograph of a dog with head tilt to the right in a dog diagnosed with a C2 spinal nerve root mass. (B to H) MRI of this dog revealing a large tubular mass which extends along the expected course of the right C2 spinal nerve and invades intramedullary causing marked compression and infiltration of the spinal cord with contrast enhancement, A tubular portion of the mass extends laterally into the adjacent paravertebral musculature and extends caudoventrally along the surface of C2 in multi-lobulated shape. This portion of the mass was heterogeneous and poorly contrast enhancing. These lesions were consistent with a suspected right C2 spinal nerve root mass. Right epaxial muscle atrophy was noticed at the level of C2. There was also diffuse T2W and FLAIR intramedullary hyperintensity likely to represent syringomyelia cranial and caudal of the mass lesion. (B) T2W sagittal of the neck showing the mass (arrowhead). (C) FLAIR sagittal of the neck. (D) T1W sagittal of the neck. (E) T1W sagittal post-contrast. (F to H) T2W transverse (F), T1W transverse pre-contrast (G) and T1W transverse post-contrast (H) of the neck at the level of C2 showing the invasion of the mass to the epaxial musculature (arrowhead). (G) T1W transverse of the neck at the level of C2. (H) T1W post-contrast at the level of C2

Diagnostic findings

Complete blood count was performed in nine of 15 (56%) dogs, of which three of nine (19%) had a stress leukogram and two of nine (13%) mild anaemia. Serum biochemistry was performed in eight of fifteen (50%) dogs, of which one (6%) dog had hypercholesterolemia. The remaining dogs (6/15) had complete blood count and serum biochemistry performed at the referring veterinary practice which revealed stress leukogram (2/6, 33%) and the remaining values were within normal limits.

All dogs had MRI performed, apart from one which had a CT. MRI was performed in 14 dogs revealing a C2 spinal nerve root mass (4/15, 27%) (Fig 1), of which one had an affected C3 spinal nerve root as well, a C1-C2 intradural-extramedullary mass (1/15, 7%), C4 vertebral body mass (1/15, 7%), C2 vertebral malformation (1/15, 7%) with impingement of the spinal cord by the dens and secondary spinal cord atrophy at the level of C1, C3-C4 (2/15, 13%) and C4-C5 (1/15, 7%) intervertebral disc extrusion (IVDE), concurrent C5-C6 and C6-C7 intervertebral disc protrusions (IVDP) (1/15, 7%), C5-C7 osseous-associated cervical spondylomyelopathy (1/15, 7%) and cervical paraspinal myositis (2/15, 13%). CT was performed in 1 dog revealing a C2 vertebral fracture (1/15, 7%) and displacement with associated lateral impingement of the spinal cord. No abnormalities were detected on MRI (14/15) or CT (1/15) of the head.

Cisternal CSF analysis was performed in 6 dogs. Total nucleated cell count was within normal limits in all samples [reference intervals (RI): <6 cells/mm^3], whilst increased total protein concentration was detected in two dogs (32 and 31 mg/dL, respectively, RI: <25 mg/dL).

Thyroid profile (total thyroxine and thyroid-stimulating hormone) was performed in one of 15 (7%) dog, whose serum biochemistry revealed hypercholesterolemia, and was normal. Serology (indirect immunofluorescence) for *Toxoplasma gondii* (IgG/IgM) and *Neospora caninum* (IgG) were performed for two of 15 (13%) dogs and was negative.

Treatment and outcome

Two dogs, one of them diagnosed with a C2 spinal nerve root mass and the other with a C4 vertebral body mass, were euthanased immediately after a diagnosis was made. For the remaining 13 dogs, treatment was dependent on aetiologic diagnosis. Treatment of two dogs diagnosed with head tilt as a post-operative complication of surgery [modified hemilaminectomy, durotomy, myelotomy (n=1), rhizotomy] for resection of C2 spinal nerve root mass included prednisolone and hydroxyurea (n=1) and

lomustine (n=1). Histopathology of the C2 spinal nerve root masses was consistent with peripheral sheath tumour (PNST) (n=2; one malignant, one benign). Treatment of two other dogs, one diagnosed with a right-sided C2 and one with left-sided C2 and C3 spinal nerve root mass, was palliative including gabapentin 100 mg/kg PO q8h (n=2) and prednisolone 0.5 to 1 mg/kg PO q24h (n=1). Treatment of a dog diagnosed with intradural-extramedullary mass included a C1-C2 hemilaminectomy with resection of the mass, prednisolone 0.5 to 1 mg/kg PO q24h and hydroxyurea 50 mg/kg PO q48h. Histopathology of that mass was consistent with papillary meningioma (n=1). Three dogs with IVDE and one with IVDP were treated with a ventral slot surgical procedure (n=3), modified hemilaminectomy (n=1) and gabapentin 10 mg/kg PO q8h (n=1), meloxicam 0.1 mg/kg PO q24h (n=1), paracetamol 10 mg/kg PO q12h (n=1), diazepam 0.5 mg/kg PO q8h (n=1), carprofen 4.4 mg/kg PO q24h (n=1). One dog with a C2 vertebral fracture was treated medically with gabapentin, carprofen and strict rest. In one dog with a C2 vertebral malformation no treatment was initiated. Two dogs with cervical paravertebral myositis were treated with prednisolone 0.5 to 1 mg/kg PO q24h (n=1), gabapentin 10 mg/kg PO q8h (n=1), firocoxib 5 mg/kg PO q24h (n=1), metronidazole 10 mg/kg PO q12h (n=1) and amoxicillin-clavulanic acid 20 mg/kg PO q12h (n=1). One dog with C5-C7 osseous-associated CSM was treated medically with meloxicam and restricted exercise.

Follow-up information was available for 11 of the 13 dogs in which treatment was initiated. Median follow-up time was 60 days (IQR: 225.5 days, range: 5 to 720 days). In eight of 15 (53%) dogs head tilt resolved after initiation of treatment, whilst in three of 15 (20%) dogs it remained static. In dogs with post-operative head tilt after C2 spinal nerve root mass removal, head tilt resolved in 1 dog and remained unchanged in the other dog. One of them had a follow-up MRI immediately after surgery which revealed left-sided T2W hyperintensity of the spinal cord suggestive of oedema at the level of C1-C2. In that dog, head tilt resolved 2 months post-operatively. The other dog had a follow-up MRI 2 months later, where a C3 nerve root mass was then revealed. In that dog, head tilt remained unchanged. In all cases diagnosed with IVDE or IVDP, head tilt had resolved after surgery. In all cases with cervical paraspinal myositis, head tilt had resolved after treatment. In the dog with a C2 vertebral fracture, head tilt remained unchanged after medical management. The dog with C5-C7 osseous-associated CSM remained with head tilt and was euthanased 1 month after diagnosis due to owners' inability to cope with its demanding management as per neurological deterioration.

DISCUSSION

Head tilt is one of the most recognisable clinical signs in dogs with vestibular syndrome. Observation of the clinical sign is therefore typically associated with a lesion in the peripheral or central components of the vestibular system. Nevertheless, abnormal posture of the head and trunk has been previously reported with cervical myelopathies, such as head turn (*rotational torticollis*), body turn (*pleurothotonus*), and head and neck turn (*cervical scoliosis*) (Rusbridge *et al.* 2019, Nagendran *et al.* 2021, Poad *et al.* 2021). This study highlights that in rare cases head tilt might not imply a neuroanatomical localisation to the vestibular system, but can also be considered as a sign of cervical myelopathy (neurological) or cervical myopathy (non-neurological antalgic posture).

Head tilt, as a clinical sign, might be a result of different pathophysiologies in animals. In vestibular disease affecting the brainstem or the peripheral vestibular system, head tilt occurs as a consequence of loss of ipsilateral excitatory input to the extensor muscles of the neck resulting in a head tilt towards the side of the lesion when the lesion is in the brainstem, the vestibulocochlear nerve or the inner ear (Lorenz *et al.* 2011). When the lesion affects the cerebellum (flocculonodular lobes; caudal cerebellar peduncles), head tilt can be a result of loss of the cerebellar inhibitory influence of ipsilateral vestibular nuclei of the brainstem, resulting in relative excessive discharge from the vestibular nuclei forcing a head tilt towards the opposite side off the lesion. This is also referred to as paradoxical vestibular syndrome (Lorenz *et al.* 2011). Ipsilateral or contralateral head tilt has also been described in forebrain disease affecting the ventrolateral and paramedian thalamus, respectively, and it has been attributed to damage to adjacent midbrain regions involved in vestibular function (Concalves *et al.* 2011).

Nevertheless, head tilt – usually in the absence of other vestibular signs (nystagmus, positional strabismus and vestibular ataxia) and in the presence of signs of cervical myelopathy (tetra/hemiparesis, generalised proprioceptive ataxia, cervical hyperaesthesia) has sporadically been reported as a consequence of cervical myelopathy (Harris *et al.* 2011, De Lahunta *et al.* 2021a) including Chiari-like malformation and syringomyelia (De Lahunta *et al.* 2021a). Specifically, signs of vestibular dysfunction can occur with lesions affecting the spinal nerve, dorsal roots or dorsal grey matter of the C1-C3 spinal cord segments. Such lesions can result in loss of general proprioceptive afferents from neuromuscular spindles resulting in abnormal function of the epaxial muscles which maintain the normal orientation of the head and neck (De Lahunta *et al.* 2021b). This study identified 15 cases of head tilt secondary to cervical spinal or paraspinal disease over a 21-year period in a busy referral hospital. Although the prevalence of head tilt in dogs with cervical spinal or paraspinal disease remains unknown, this finding suggests that head tilt should be considered a rare finding in dogs with a neck problem. When an intracranial or extracranial cause for head tilt is not identified, advanced imaging of the neck can be considered.

The exact pathophysiological mechanism to explain the development of head tilt in dogs with cervical spinal or paraspinal disease is currently unknown and should possibly be considered multifactorial. This study describes three different suggested anatomical origins to cause head tilt in cervical spinal or paraspinal disease: (1) cranial cervical spinal cord, (2) caudal cervical spinal cord and (3) paraspinal muscles. Specific anatomical structures associated with the cranial cervical spinal cord, such as C1-C3 spinal cord segments, roots and ganglia including the spinocerebellar and vestibulospinal pathways, have been suggested to play a role in regulating vestibular input in the spinal cord. This study

describes two dogs that only developed a head tilt immediately after a C2 modified hemilaminectomy with rhizotomy for surgical resection of a PNST. This manifestation has been previously observed in experimental animals after resection of these nerve roots or blocking them with local anaesthetics (De Lahunta *et al.* 2021b). In rats, the C2 and C3 dorsal roots and ganglia provide afferent projections to vestibular nuclei, and therefore these connections reflect the significant role of proprioceptive neck afferents for the control of head position (Neuhuber & Zenker 1989). Temporary vestibular signs have been previously observed in three dogs after resection of extraparenchymal spinal cord tumours at the level of C1-C2 vertebrae presumably from surgical trauma to the spinal cord segments or dorsal roots. These clinical signs resolved in all three dogs within 3 to 5 days (De Lahunta *et al.* 2021b). In our study, head tilt resolved in one case shortly after surgery, whilst it remained static in the other case in which a C3 spinal nerve root mass was revealed 2 months after surgery. Why these dogs did not manifest head tilt due to the original pathology before surgery whilst other dogs would do, is not completely understood; however, it could be attributed to a possible anatomical variance. For example, some people and possibly animals may have either a smaller number of rootlets or a complete lack of the dorsal root associated with the first spinal cord segment (De Lahunta *et al.* 2021b). Therefore, in some dogs a pathological process at that level can cause head tilt, whilst in others not. One of the cases had post-operative MRI, which revealed a poorly demarcated T2-weighted intramedullary hyperintensity ipsilateral to the head tilt. A possible pathophysiologic mechanism to explain post-operative head tilt could include postoperative extensive oedema affecting the spinal cord or even expanding up to the brainstem. Alternatively, iatrogenic surgical trauma can potentially be associated with more complete and focal damage compared to the initial pathology which eventually is able to cause a head tilt.

Two dogs in this study, one with C2 and C3 spinal nerve masses and one with a C1-C2 papillary meningioma, manifested head tilt. A C1 spinal cord meningioma has been previously reported to cause equivocal head tilt (De Lahunta *et al.* 2021a). A possible disruption of the spinocerebellar pathways from the first two cervical spinal cord segments could potentially explain this clinical sign (De Lahunta *et al.* 2021a).

One dog with a C2 vertebral malformation and another one with a C2 vertebral fracture were included in this study. A possible disruption of spinocerebellar pathways is suspected in these cases, whilst similar diseases causing head tilt have been previously reported in both dogs and humans. A C2-C3 articular process hypertrophy/malformation with associated vertebral canal stenosis has been associated with head tilt in a dog (Harris *et al.* 2011). Another dog with C2-C5 multiple vertebral malformations (fusion) manifested cervical torticollis (Fernandes *et al.* 2019). In humans, cervical disc degeneration or facet osteoarthritis, the erroneous proprioceptive input distorts the direct linear interaction between neck proprioception and vestibular information, resulting in subjective body orientation and other vestibular signs (Yang *et al.* 2017). Additionally, in dogs dorsal angulation of dens in the vertebral canal decreases the vertebral canal diameter and compresses the spinal cord and possibly the spinocerebellar tract causing associated signs (Loughin & Marino 2016).

Whilst head tilt in cranial cervical lesions can be caused by involvement of specific anatomical structures, belonging to or carrying information for the vestibular system, head tilt with more caudal cervical lesions is likely to have a different pathophysiological mechanism, not directly related to the vestibular system. Four dogs in this study had lesions caudal to C3. Two dogs had IVDE (C3-C4 or C4-C5) with associated syringomyelia starting from C2. It is therefore possible that the observed head tilt in these dogs was associated with the more cranially located syrinx (Mulroy *et al.* 2019). Head tilt has been reported in Cavalier King Charles Spaniels with Chiari-like malformation and syringomyelia. It has been hypothesised that this results from the extension of the syrinx into the superficial dorsal horn contralateral to the head tilt (Rusbridge *et al.* 2018). One dog had a C3-C4 IVDE and another had a C4 vertebral mass both without associated syringomyelia. In these dogs, an antalgic – developed in a way to avoid pain –voluntary head tilt cannot be excluded. In one dog with C5-C7 CSM, the head tilt could be either antalgic or neurological. Interestingly, reticulospinal pathways in the ventrolateral funiculus with terminations in the C5-C6 and C7-C8 spinal cord segments have been observed and given a role in maintaining posture in rats (Reed *et al.* 2008). Therefore, if not antalgic, head tilt in our case with osseous-associated CSM could be a result of interruption of the reticulospinal tract.

Unlike the cranial or caudal spinal cord pathology, head tilt appeared to be a non-neurological clinical sign of cervical paraspinal muscle disease. Two dogs manifested head tilt with cervical paraspinal myositis which resolved after treatment. In humans, head tilt with or without cervical scoliosis is a clinical sign of cervical paraspinal abscess and it is a result of pain or muscle contraction. Therefore, its pathophysiological mechanism in those cases is non-neurological in origin (Tomczak & Rosman 2012, Beasley 2021) and it can possibly represent different muscle tone-contraction or/and antalgic posture.

Head tilt resolved in seven cases of this study after the causative agent was eliminated (*e.g.* tumour removal, treatment of myositis, decompression of IVDE). In two cases, one with C2 vertebral fracture and one with osseous-associated CSM, head tilt remained residual. The reason why the head tilt remained might be either due to permanent damage of the vestibular pathways of the spinal cord in conjunction with lack of reparative surgery, similarly to the commonly seen residual head tilt in brainstem or cerebellar vestibular disease in dogs (Bongartz *et al.* 2020). Nevertheless, a residual antalgic head tilt due to persistent neck pain and subsequent muscle contracture cannot be ruled out in those cases.

Limitations of this study consist of (1) limited number of cases, (2) lack of photographic evidence of head tilt in most cases, (3) lack of systematic thyroid profile testing in all dogs regardless of clinical suspicion, (4) lack of follow-up MRI of cases and (5) inconsistent follow-up. In animals, it might be difficult to recognise subtle differences in head position. Due to the retrospective nature of this study, it was not possible to evaluate if "vestibular head tilt" and "antalgic head tilt in response to cervi-

cal hyperaesthesia" can be differentiated. Further studies are necessary to compare the clinical features of these two different types of head tilts and investigate further whether a difference between those exists. Additionally, terminology regarding description of the different head and neck positions is not well established in veterinary literature. Therefore, it can be debated whether the term head tilt should apply in animals without a lesion in the intracranial components of the vestibular system.

In conclusion, although vestibular syndrome is the most common cause of head tilt, this study illustrates that head tilt can rarely be a neurological sign of cervical spinal or paraspinal disease. Head tilt in cranial cervical myelopathy usually affecting the C1-C3 dorsal spinal cord segments, spinal nerve roots or ganglia, might be a result of disruption of the vestibular pathways within the spinal cord. Head tilt can also occur in dogs with more caudal cervical lesions, in which it is unclear if this is a result of vestibular pathway disruption or antalgic due to cervical hyperaesthesia. At last, head tilt can be a clinical sign of cervical paraspinal myopathy most likely as an antalgic sign. In the absence of intracranial or extracranial causes of vestibular dysfunction in a dog with head tilt, considerations should be given to a lesion localised in cervical spinal cord or associated structures.

Conflict of interest

None of the authors of this article has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

Author contributions

Theofanis Liatis: Conceptualization (equal); data curation (equal); formal analysis (equal); investigation (equal); methodology (equal); project administration (equal); resources (equal); writing – original draft (equal); writing – review and editing (equal). **Steven De Decker:** Methodology (equal); supervision (equal); validation (equal); writing – review and editing (equal).

References

Beasley, S. W. (2021) Torticollis. In: Pediatric Surgery. Ed P. Puri. Springer, Berlin, Germany. pp 31-43

Bongartz, U., Nessler, J., Maiolini, A., *et al*. (2020) Vestibular disease in dogs: association between neurological examination, MRI lesion localisation and outcome. *The Journal of Small Animal Practice* **61**, 57-63

Concalves, R., Carrera, I., Garosi, L., *et al*. (2011) Clinical and topographic magnetic resonance imaging characteristics of suspected thalamic infarcts in 16 dogs. *Veterinary Journal* **188**, 39-43

De Lahunta, A., Glass, E. & Kent, M. (2021a) Case descriptions. In: De Lahunta's Veterinary Neuroanatomy and Clinical Neurology. 5th edn. Elsevier, Philadelphia, PA, USA. pp 547-578

De Lahunta, A., Glass, E. & Kent, M. (2021b) Vestibular system: special proprioception. In: De Lahunta's Veterinary Neuroanatomy and Clinical Neurology. 5th edn. Elsevier, Philadelphia, PA, USA. pp 345-373

Fernandes, R., Fitzpatrick, N., Rusbridge, C., *et al*. (2019) Cervical vertebral malformation in 9 dogs: radiological findings, treatment options and outcomes. *Irish Veterinary Journal* **72**, 2

Ferreira, J. J., Costa, J., Coelho, M., *et al*. (2007) The management of cervical dystonia. *Expert Opinion on Pharmacotherapy* **8**, 129-140

Harris, K. P., Saveraid, T. C. & Rodenas, S. (2011) Dorsolateral spinal cord compression at the C2-C3 junction in two Cavalier King Charles spaniels. *The Veterinary Record* **169**, 416a

Harrison, E., Grapes, N. J., Volk, H. A., *et al*. (2021) Clinical reasoning in canine vestibular syndrome: which presenting factors are important? *The Veterinary Record* **188**, e61

Lorenz, M. D., Coates, J. R. & Kent, M. (2011) Ataxia of the head and limbs. In: Handbook of Veterinary Neurology. 4th edn. Elsevier Saunders, St Louis, MO, USA. pp 250-281

Loughin, C. A. & Marino, D. J. (2016) Atlantooccipital overlap and other craniocervical junction abnormalities in dogs. *The Veterinary Clinics of North America. Small Animal Practice* **46**, 243-251

Mulroy, E., Balint, B., Latorre, A., *et al*. (2019) Syringomyelia-associated dystonia: case series, literature review, and novel insights. *Movement Disorders Clinical Practice* **6**, 387-392

Muñana, K. (2014) Head tilt and nystagmus. In: BSAVA Manual of Canine and Feline Neurology. 4th edn. Eds S. R. Platt and N. J. Olby. British Small Animal Veterinary Society, Gloucester, UK. pp 195-212

Nagendran, A., Jose-Lopez, R., Sunol, A., *et al*. (2021) Head turn: a study of neurolocalisation. Proceedings of 33rd ESVN-ECVN Symposium Online. September 16 to 18, p 86

Neuhuber, W. L. & Zenker, W. (1989) Central distribution of cervical primary afferents in the rat, with emphasis on proprioceptive projections to vestibular, perihypoglossal, and upper thoracic spinal nuclei. *The Journal of Comparative Neurology* **280**, 231-253

Poad, L., De Decker, S. & Fenn, J. (2021) Acquired cervical scoliosis in two dogs with inflammatory central nervous system disease. *Journal of Veterinary Internal Medicine* **35**, 2421-2426

Reed, W. R., Shum-Siu, A. & Magnuson, D. S. K. (2008) Reticulospinal pathways in the ventrolateral funiculus with terminations in the cervical and lumbar enlargement of the adult rat spinal cord. *Neuroscience* **151**, 505-517

Rusbridge, C., Stringer, F. & Knowler, S. P. (2018) Clinical application of diagnostic imaging of Chiari-like malformation and syringomyelia. *Frontiers in Veterinary Science* **5**, 280

Rusbridge, C., McFadyen, A. K. & Knower, S. P. (2019) Behavioral and clinical signs of Chiari-like malformation-associated pain and syringomyelia in Cavalier King Charles spaniels. *Journal of Veterinary Internal Medicine* **33**, 2138-2150

Tomczak, K. K. & Rosman, P. (2012) Torticollis. *Journal of Child Neurology* **28**, 365-378

Yang, L., Yang, C., Pang, X., *et al*. (2017) Mechanoreceptors in diseased cervical intervertebral disc and vertigo. *Spine (Phila Pa 1976)* **42**, 540-546