HI EVERYONE!

This edition of FourLeg News is one that was inspired by a human physiotherapist, Dr. Bahram Jam (based in Thornhill, ON) and a wonderful poster he has created entitled Don't Be a VOMIT. VOMIT stands for Victim of Medical Imaging Technology.

Bahram has done a wonderful job on the human-side to expose human medicine’s tendency to dramatize the often incidental findings on medical imaging which can lead to inaccurate treatment and catastrophizing of minor ailments in human patients.

I e-mailed Bahram to see if I could utilize the VOMIT term he had coined and to utilize some of the information he has presented in this vein of study, and he was delighted that someone else was taking up the charge to enlighten more people about the dangers of medical imaging over-reliance.

So I bring to you the Canine Rehab VOMIT edition of FourLeg News!

By the way, if you are looking for brilliant, up to date, easily digestible, readily implementable, mind expanding, and inspiring physical therapy relevant information, then I highly recommend www.aptei.com. This is Bahram’s website. He has a quarterly newsletter, some e-blasts, and a great clinical library of papers.

Okay… I hope you enjoy this edition, and I welcome your feedback! Remember… DON'T BE A VOMIT!

Cheers,
Laurie
Fibrocartilaginous Metaplasia Can be found in Clinically Normal Tendons!

A study was designed to describe histopathological lesions of the biceps brachii tendon of origin in dogs with confirmed biceps tenosynovitis and to compare those findings with those from the biceps tendon of normal control dogs.

So the group of researchers evaluated the medical records of 17 dogs diagnosed with biceps tenosynovitis at a university veterinary teaching hospital. Diagnosis of biceps tenosynovitis based on radiographic and arthrographic findings, surgical exploration of the joint, and histopathological changes of the biceps tendon and bicipital bursa, tendon sheath, or joint capsule. All of these dogs underwent surgical treatment for biceps tenosynovitis.

They also looked at thirteen biceps tendons from 10 clinically normal dogs euthanized for unrelated causes and put them through histopathological examination. None of these dogs had a history of clinical orthopedic disease.

What did they find?
Of the tenosynovitis dogs, the degree of lameness was variable and was chronic and progressive, lasting on average 10.6 months. On physical examination, palpation of the biceps tendon within the intertubercular groove or manipulation of the shoulder through flexion and extension was painful. Muscle atrophy of the infraspinatus and supraspinatus muscles was also common.

The decision to perform surgery was based on a history of chronic progressive lameness and the radiographic and arthrographic findings. In all dogs, joint capsule thickening, osteophyte formation, a roughened and thickened bicipital groove, or roughening, thickening, fibrosis, or adhesions of the biceps tendon was grossly evident at surgery.

What could be discussed?
Radiography and arthrography were instrumental in diagnosing biceps tenosynovitis. Survey radiographs and arthrograms were abnormal in all dogs. Radiographic findings included periarticular swelling, osteophyte production along the intertubercular groove, dystrophic mineralization of the biceps tendon, and mineralized densities (i.e., joint mice) within the bicipital bursa. Arthrography was helpful in evaluating the biceps tendon and bursa, successfully detecting roughening or narrowing of the tendon and filling defects of the bursa secondary to synovial proliferation.

In these lame dogs, radiographic and arthrographic findings were subjective and variable and did not predict degree of histopathological changes found. The most common histopathological changes in the affected dogs were fibrosis and collagen fragmentation, synovial villous and vascular hyperplasia, and inflammatory cell infiltration. These lesions were associated with degree, but not duration, of lameness.

Contd overleaf …
... Fibrocartilaginous Metaplasia contd

In fact, there appeared to be a very slight trend for dogs with longer duration of lameness to have less severe histopathological lesions with ischemic necrosis, edema, arterial medial hyperplasia, osseous metaplasia, and vascular dilatation found in fewer tendons.

All normal dogs had mild to severe cartilage metaplasia, the degree of metaplasia generally more severe than that seen in affected dogs. The authors speculated this might have been an incidental finding in the biceps tendons of dogs. There were no other histopathological changes. If tendons of supposedly clinically normal dogs commonly have cartilaginous metaplasia, it seems that this change would not be clinically observable.

And what could be concluded?

This study suggests that metaplasia can be found in clinically normal biceps tendons and does not necessarily imply pathological change. Biceps tendon fibrocartilaginous metaplasia can’t be ruled in or out as a prerequisite to clinically evident biceps tenosynovitis.

A few words if I may as they pertain to VOMIT:

- So this study is telling us that normal dogs may have abnormal cells (for a tendon) in the biceps tendon of origin. Normal dogs. (However the normal dogs were not radiographed or inspected by arthrography to know how they would compare to the clinically lame dogs.)

- We also learn that radiography & arthrography do not predict degree of histopathologic changes, but degree of lameness was a better indicator.

- This study also tells us that dogs that have been lame for the longest amount of time actually had the least amount of histopathologic issues. (PS Bet I could have rehabbed these dogs!!!)

- So why base our decisions on radiography & arthrography if these are not good histopathologic indicators?

Source:
Clinically Normal Dogs May Have Borderline Hip Dysplasia

The purpose of this study was to detect any changes in joint kinematics of clinically sound dogs with or without radiographically detectable borderline hip dysplasia (CHD).

How did they do it?
The study utilized twenty Belgian Malinois dogs, mean age 2.75 years, with no clinical signs of hip dysplasia (orthopedic and neurologic examinations did not detect lameness or pain on palpation). All dogs were subject to radiographs to determine if the animal had no hip dysplasia or borderline hip dysplasia. Ground reaction forces were measured by force plate and kinematic gait analysis was performed while walking on a treadmill. (12 dogs were identified to have borderline CHD & 8 were determined to be normal on x-ray.)

AND so?
Ground reaction forces found no differences between groups.

Dogs with borderline CHD showed an increase in stifle flexion and decreased hip flexion in the swing phase of walking and had faster flexion of the tarsal joints.

Conclusions
This study revealed that dogs with borderline HD had altered joint kinematics. The study determined that even subclinical and subtle radiographic signs of HD can influence joint kinematics in Belgian Malinois dogs.

Findings suggest that a lack of visible lameness and evidence of pain during manipulation does not exclude altered joint biomechanics as a result of HD. These subtle changes suggest complex compensatory mechanisms and interactions between joints of the involved limb.

A few words from the VOMIT perspective:
So what?!! Pain and function matter most.

These were normal dog with incidental findings. Let’s rethink some of these findings. Someone with shorter legs jumps, runs, and grabs things from the top shelf differently than someone with longer legs. Horses with a short back jump differently than horses with longer backs (if you don't believe me, try this out sometime… while jumping over a small creek and trying to stay on!!!)

I’m going to rephrase this conclusion to read:
Normal dogs may have incidental findings of borderline CHD!

Source:
Surprising Joint Findings

In a couple of studies looking at radiologic changes in disease-prone dogs under a year of age... joint abnormalities were indeed found. However they also found something unexpected.

So firstly, of disease-prone dogs, radiologic changes, indicative of hip dysplasia were seen, by 1 year of age. Among dogs between 3 and 11 months of age that had joint abnormalities, 71% had hip joint involvement; 38%, shoulder joint involvement; 22%, stifle joint involvement; and 40% had multiple joint involvement.

Polyarthritis was asymptomatic and unexpected. Radiographic examination of older dogs also revealed evidence of degenerative joint disease in many joints.

Necropsy found more abnormal joints than were identified on radiographic examination and multiple joint involvement was found in young, adult and elderly dogs. Joint disease was usually an incidental finding, unrelated to the clinical disease or to the cause of death.

Changes in coxofemoral joints included mild nonsuppurative synovitis, increased volume of both synovial fluid and the ligamentum teres, and focal degenerative articular cartilage lesions. In the most severely affected joints subluxation of the femoral head was seen.

Synovial inflammation with increased synovial fluid and ligament volumes were indicators of early degenerative joint disease in dogs.

These changes may precede, microscopic evidence for articular cartilage degeneration and visible radiologic changes.

The VOMIT talk:
So these studies are particularly of interest. X-rays reveal unexpected arthritis in joints other than the ones being tested for (i.e. testing for hips). In other words, the dogs did not show clinical signs in these other joints. But dissection / necropsy found more ‘abnormal joints’ than x-rays revealed.

Now, two things come to mind when reading these studies. 1. We know in human literature that a physical therapy manual evaluation is able to detect signs of OA before it is radiographically evident. (And we can also therefore treat it at an earlier stage as well!) 2. Radiographic or even visual joint inspection (i.e. necropsy... but that seems a little drastic) can detect INCIDENTAL, ASYMPTOMATIC, CLINICALLY IRRELEVANT joint abnormalities.

So let’s press harder for better manual physical assessments skills (such as the ones that physical therapists bring to the field of canine rehab and are teaching to veterinarians) to be the gold standard in evaluation of dogs (lame or not!)

Sources

There is No Significant Relationship between Limb Function and the Severity of Stifle Osteoarthritis on X-ray!

**Objective**
To evaluate the relationship between limb function and radiographic evidence of stifle osteoarthrosis (OA) in dogs as measured by force platform gait analysis.

**How did they do it?**
Forty-one dogs with visible lameness and radiographic evidence of stifle OA, and no therapeutic interventions for a period of time, were utilized. Force platform data and radiographic OA score was collected and evaluated on 2 separate days.

**Results**
No significant relationship was found between force platform data and OA score. Data strongly suggests that there is no relationship between limb function and the severity of radiographic signs of stifle OA and that that limb function actually improves as OA increased.

**Conclusions**
The presence of OA in the stifle joint does not correlate with clinical function. Radiographic evidence of stifle OA provides evidence of pathology, it does not represent animal limb function well. Radiographic outcome should thus be used cautiously as a predictor of clinical outcome.

**VOMIT-ilicious!!!!**
Oh yeah! This is what I’m talking about! So if an x-ray reveals OA… does that OA match the clinical signs of the dog? If not… who cares!!! Keep an eye on that joint… by all means, and treat it if it becomes clinical. But most importantly, as rehab professionals, we should be the ones standing on the rooftops shouting out about the importance of FUNCTION, FUNCTION, FUNCTION!!!

Source:
MRI of Disc… Let’s Not Jump to Conclusions!

Case Description
A 3-year-old French Bulldog was evaluated because of acute signs of back pain and spastic paraparesis. Neuroanatomic localization indicated a lesion in the T3-L3 spinal cord segment. Magnetic resonance imaging revealed extradural spinal cord compression at the ventral right aspect of the intervertebral disk space L3-4. A diagnosis of sequestrated Hansen type 1 disk extrusion without extradural hemorrhage was made.

The dog was treated conservatively with cage rest, restricted exercise on a leash, and NSAIDs. Results of follow-up examination 5 weeks later indicated neurologically normal gait and posture, and ambulation without obvious neurologic deficits or signs of pain. The displacement and compression of the spinal cord had resolved. Almost complete regression of sequestrated extruded intervertebral disk material was found.

Clinical Relevance?
Findings in this case indicated resolution of spinal cord compression attributable to extruded intervertebral disk material. Functional improvements in dogs with such problems may be partly attributable to spontaneous regression of intervertebral disk extrusions.

[Psst… We see this in humans too!]
Human reports are similar to this dog; results of other studies of ambulatory and non-ambulatory dogs with thoracolumbar disk herniations also indicating successful functional outcomes following conservative treatments, although follow-up imaging was not performed in earlier studies.

Recovery of function was attributed to healing of a ruptured fibrous annulus, a decrease in inflammation over time, and restoration of spinal cord function by reversal of spinal cord damage and mechanisms of neuroplasticity.

Findings for this dog suggest that resorption of extruded thoracolumbar intervertebral disk material in chondrodystrophic dogs is possible, and full resolution of neurologic signs may be detected after 5 weeks. Dogs with such problems treated without surgery may have good outcomes because of functional compensation of neurologic deficits caused by spinal cord compression, a decrease in inflammation, and a substantial reduction of the size of herniated disk material.

Let us VOMIT-analyze this study and rephrase the conclusions:
MRI evidence of Type 1 disc extrusion with clinical signs of cord compression does not predict outcome OR the need for surgery.

Oh, just let us get our hands on more of these dogs! Sooner! Before surgery! I am positive that we can expedite the healing even further!!

Source:
Steffen F, Kircher PR, Dennier M. Spontaneous regression of lumbar Hansen type 1 disk extrusion detected with magnetic resonance imaging in a dog. J Am Vet Med Assoc 2014;244:715–718
Chiari-Malformation and Syringomyelia, Clinically Relevant or Not?

Objectives
The objectives of the study were to report the incidence of Chiari-like malformation and syringomyelia in normal (asymptomatic) French Cavalier King Charles Spaniels.

Method
Sixteen clinically normal adult cavalier King Charles spaniels underwent ultrasonographic examination of the spinal cord and caudal fossa. Computed tomography (CT) was used to measure the caudal fossa and magnetic resonance imaging allowed for identification of syringomyelia and cerebellar herniation.

Neurological examination was performed prior to testing. Dogs were excluded from the study if signs of CM/SM had been noticed by the breeders or were detected during neurological examination.

Results
All study dogs had cerebellar herniation, suggesting Chiari-like malformation and also a tendency to occipital dysplasia. Seven of the 16 dogs in the study also had syringomyelia (43.7%).

Computed tomography measurements of the caudal fossa in this clinically normal population, found no statistical difference in the size of the caudal fossa between dogs with or without syringomyelia.

A syrinx was identified by ultrasonography in one study dog. The only difference between dogs with or without syringomyelia was that dogs with Chiari like malformation/syringomyelia were statistically older.

Clinical Significance
The incidence of Chiari-like malformation and syringomyelia may be high in an asymptomatic population of Cavalier King Charles Spaniels.

As well, perhaps a population of asymptomatic CKCS that are free of syringomyelia, Chiari like malformation, and occipital dysplasia does not exist!

VOMIT-Take Home
You can't rely on imaging to diagnose whether a Chiari-malformation of syringomyelia is or will become symptomatic.

Source:
Old Dog Spines – Don’t Get Freaked Out When You Look at That CT Scan!

Studies of lower back disease in humans, have demonstrated that the severity of CT abnormalities does not always correlate with the severity of clinical signs, especially in older patients. Surgical intervention is often not recommended unless the presenting complaint and neurologic exam findings are consistent with the location of imaging abnormalities. The objective of this study was to determine if some CT abnormalities in older dogs may also be clinically insignificant.

What did they do?
A prospective study of six large-breed dogs undergoing CT imaging for problems unrelated to the lumbosacral spine. All dogs were asymptomatic for lumbosacral stenosis on neurologic examination. A regional CT examination of the lumbosacral spine was performed immediately after the area of clinical concern was scanned.

What did they find?
Five out of six dogs had lumbosacral CT abnormalities, with many occurring at more than one disc level. The most common CT abnormality was idiopathic stenosis. Other abnormalities included loss of foraminal fat, disc margin bulging into the vertebral canal, bone proliferation into the vertebral canal or foramen, and nerve tissue displacement. Vertebral subluxation was absent in all dogs.

What did they discuss?
Findings support the theory that some lumbosacral CT abnormalities may be clinically insignificant, especially in older dogs. This is similar to findings in humans where in one study CT scans of the lumbar spine in 52 asymptomatic human volunteers aged 21-80 years, an average of 50% abnormal CT scans was found in the group aged over 40 years.

And they nailed the conclusion!
The authors of this study propose that some lumbosacral CT abnormalities may be clinically insignificant in older dogs, and that the best surgical candidates will be those where there is a good correlation between the clinical findings and CT anatomic locations of compressive tissues.

Poignant VOMIT thoughts
I’ve heard it said: “Dogs don’t walk on their x-rays or MRI scans” or “Treat the dog, not the x-ray.” And I’d like to add: “You should have some idea of what you might find before you send a dog for further diagnostics” and “The only imaging findings you should pay great attention to, are the ones that fit with your clinical exam findings and clinical reasoning”.

Source:
No Way Toe OA

Osteoarthritis of Metacarpophalangeal (MCP) and Metatarsophalangeal (MTP) joints has unique radiographic features that can complicate accurate diagnosis, particularly differentiation from primary bone neoplasia.

**What did they look at?**
Medical record review of 49 dogs with a radiographic diagnosis of MCP or MTP OA presented over a 7-year period to the Colorado State University Veterinary Teaching Hospital.

Cases with radiographs available for review were divided into two groups, one composed of dogs with lameness attributed to MCP/MTP OA as a primary complaint, the other of dogs with MCP/MTP OA as an incidental finding secondary to some other pathology. All radiographs made of the MCP or MTP joints at any time were reviewed by a board-certified radiologist.

**What they found…**
Of the 49 dog in the study OA was an “incidental finding” for the majority of animals (n=35), with only 14 dogs identified as clinically lame as a result of MCP or MTP OA.

No statistically significant relationship was found between lameness as a result of MCP/MTP OA and the presence of radiographic signs of OA, but that those in the primary complaint group were significantly more likely to have firm swelling over their affected digits. Swelling over the digits should raise suspicion that MCP/MTP OA may be causing lameness, and should be an indication for taking radiographs of these joints in a lame dog.

**VOMIT-away**
70% of the dogs in this study had incidental findings of OA in their toes! So quit relying on x-rays to determine what’s wrong with the animal. Go back to basics and learn to manually assess!

Source:
Supraspinatus Mineralization – Don’t Jump to Surgery!

The role mineralization of the supraspinatus tendon in causing lameness is unclear. The appearance of mineralization does not necessarily mean that it is the source of lameness. The objective of this study was to evaluate the long-term clinical outcome and radiographic recurrence of the mineralization of the supraspinatus tendon in large breed dogs.

**Method**
Medical records of dogs presented to a University Veterinary Teaching Hospital between 1986 and 1995 with the diagnosis of mineralization of the supraspinatus tendon, believed to be the cause for lameness, were reviewed. A questionnaire was sent to owners, and they were asked to bring their dog for reevaluation (gait evaluation, palpation of the shoulder and elbow joints, and sedated radiographs of the shoulders and elbows) joint were taken.

**Results and Discussion**
All 24 dogs in the study had histories of intermittent or progressive weight-bearing lameness that worsened during or after exercise. They were grouped into ‘operated’ versus ‘conservative management’ and all but one dog in each group were lame. After surgical removal of the supraspinatus tendon mineralization, lameness decreased significantly. In the non-surgical group, one dog had persistent but mild lameness and two dogs had of occasional lameness after rising and exercise. (NOTE: Conservative management would have only included rest and meds.)

Mineralization had recurred radiographically in all cases, some being larger and others smaller than at the time of initial diagnosis/treatment. The speed of recurrence is slow, and may be associated with lameness, or it may not cause any clinical signs. It is often a bilateral radiographic finding, similar to the human rotator cuff, where 51% of the mineralizations occur in the supraspinatus tendon and are also often incidental radiographic findings in asymptomatic patients.

**Conclusions**
Mineralization of the supraspinatus tendon as a cause for lameness can be a challenging diagnosis. Conservative treatment should be tried first and other causes for lameness should be excluded. Surgery should be performed when rest and NSAIDs have failed to alleviate the lameness. Supraspinatus tendon mineralizations can recur after surgical treatment without causing any return of clinical signs.

**VOMIT? No thank you!**
Learn to palpate the supraspinatus and stretch it to determine if the supraspinatus is painful and a potential cause of lameness. Save the radiograph and try rehab!

Source:
Osseous-Associated Wobblers and Clinical Signs in Dogs

The pathophysiology of cervical spondylomelopathy (CSM) is not well understood, but accepted as being complex and multifactorial. Two forms have been described, disc-associated cervical spondylomyelopathy (DA-CSM) and osseous-associated cervical spondylomyelopathy (OA-CSM). DA-CSM's whose pathophysiology includes a main finding of intervertebral disc protrusion and it typically seen in middle aged large breed dogs. In contrast osseous-associated cervical spondylomyelopathy (OA-CSM), occurs secondary to bony proliferation surrounding the spinal cord and is typically seen in young giant breed dogs.

While studies have investigated the pathophysiology of CSM, a potential link between changes seen on magnetic resonance imaging (MRI) in OA-CSM and clinical signs has not been explored. The aim of this study was to retrospectively evaluate MRI findings, investigating potential correlations between these changes, signalment, and clinical signs.

Method
Twenty-six dogs (20 giant and 6 large breeds), diagnosed with OA-CSM were included in the study, and clinical signs and MRI findings were assessed and graded.

Results / Discussion
Spinal cord compression was identified in 36.81% of the intervertebral spaces assessed and all 26 dogs had at least 1 compressed space. As with other studies, giant breeds tended to have multiple, mainly bilateral compressions more often than large breed dogs.

All dogs had degenerative changes at the articular processes and/or the intervertebral discs in at least 6 of 7 intervertebral spaces analyzed. The 2 most commonly seen degenerative changes were synovial fluid signal loss and articular process surface sclerosis which were seen in all dogs. Foraminal stenosis occurred in 12 dogs. Intervertebral disc degeneration occurred in 19 dogs. Disc herniations were identified in 15 dogs.

Giant breeds presented at a younger age than large breeds and had multiple sites of compression. The age difference between breeds could be the result of different underlying causes for the same OA-CSM condition.

BUT... the whole point of including this in the VOMIT-edition
No significant associations were found between MRI findings and clinical signs.

Source:
Great Danes with MRI Abnormalities

Magnetic resonance imaging (MRI) is the imaging modality of choice for dogs with suspected CSM. It is recognized in the human neurology literature that MRI has the potential for over-interpretation. Studies in humans have found abnormal MRI findings in the cervical vertebral column of asymptomatic people, highlighting that MRI can display detailed pathological changes but cannot determine their clinical importance.

So, the objective of this study was to compare the MRI morphological features of the cervical vertebral column of Great Danes with and without clinical signs of cervical spondylomyelopathy (CSM).

*How did they do the study?*
They took 30 Great Danes, one year of age or older; Fifteen were clinically normal (on physical examination and neurologic examination) and 15 were CSM-affected, and they put them all through an MRI of the cervical vertebral column.

*And????*
They looked at everything. Eleven compressive sites were identified in the clinically normal dogs and 61 compressive sites in CSM affected dogs. All CSM-affected dogs had one or more sites of spinal cord compression; only one normal dog had spinal cord compression. Foraminal stenosis was seen in 11 of the 15 clinically normal and all CSM-affected dogs.

There was more (in terms of numbers) severe stenosis sites in the CSM-affected group. Significant differences were identified between clinically normal and CSM-affected dogs with regard to amount of synovial fluid evident (greater in CSM-affected), regularity of articular surfaces (CSM-affected dogs had a greater number of articular process joints with sclerosis and irregular articular surfaces), degree of articular process joint proliferation, and visibility of the internal vertebral venous plexus.

*And their comments?*
Results of the present study confirmed that clinically normal Great Danes can have cervical vertebral column abnormalities detectable with MRI. There are considerable differences between findings in normal and CSM affected Great Danes, including severe spinal cord compression, number of stenotic foraminal sites, and signal changes within the spinal cord.

These results reiterate the importance of interpreting MRI findings in conjunction with a neurologic examination before making treatment decisions.

*Don’t be a VOMIT with your Great Dane patients!*
Is it a Great Dane? Then there is a high chance that it has foraminal stenosis. No imaging based-diagnosis can be trusted without a hands-on assessment!

Source:
MRI’s of Doberman Necks

Cervical spondylomyelopathy, (Wobbler syndrome), is the most common neurologic disorder of the cervical vertebral column of large-breed dogs. It is characterized by abnormalities of the cervical vertebral column that result in neurologic deficits, and/or cervical hyperesthesia.

There is little information available on the cause, pathogenesis, and progression of CSM. The objectives of this study were to compare features of the cervical vertebral column and spinal cord of Doberman Pinschers with and without clinical signs of cervical spondylomyelopathy (CSM) through the use of MRI.

What did they do in the study? What did they find?
Sixteen clinically normal (on basis of history and results of physical and neurologic examinations, CBC, and serum biochemical analyses.) and 16 CSM-affected Doberman Pinschers were enrolled in the study.

On MRI, 4 of 16 clinically normal and 15 of 16 CSM affected dogs had spinal cord compression. 12 clinically normal and all CSM-affected dogs had disk degeneration. Multiple disks were affected in 11 of the clinically normal dogs.

All clinically normal dogs had some degree of disk protrusion, with twelve having multiple disks affected. Findings in the CSM-affected dogs were similar to those in clinically normal dogs, in that all dogs had disk protrusion. The overall extent of this disk protrusion was more severe in CSM-affected dogs.

Foraminal stenosis was detected in 11 clinically normal and 14 CSM-affected dogs. Vertebral canal and spinal cord areas were consistently smaller in CSM-affected dogs, compared with clinically normal dogs.

In neutral and traction positions, the intervertebral disks of CSM-affected dogs were wider than those of clinically normal dogs but the amount of disk distraction was similar between groups.

What did they conclude?
The authors were surprised at the percentage of clinically normal Doberman Pinschers that had severe abnormalities of the cervical vertebral column. The MRI abnormalities of some clinically normal dogs were more severe than those of CSM-affected dogs.

Continued overleaf …
.... MRI’s of Doberman Necks continued

The study found a high incidence of intervertebral disk degeneration and foraminal stenosis in clinically normal Doberman Pinschers. Cervical spinal cord compression was found to be present without clinical signs, and a combination of static factors (relatively stenotic vertebral canal and wider intervertebral disks) distinguished clinically normal from CSM affected dogs. These abnormalities likely are a key feature in the pathogenesis of CSM.

VOMIT synopsis
If it’s a Doberman, chances are its neck MRI will show signs of caudal cervical spondylomyelopathy (Wobblers). So make sure you look at EVERYTHING on these dogs (and others too, really) before jumping to conclusions based on MRI alone!

Source: