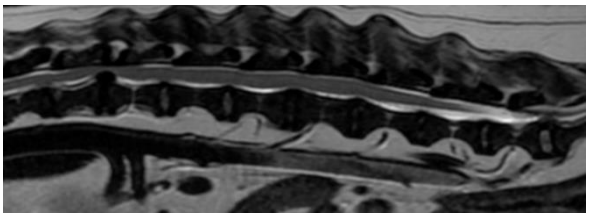
cpdsolutions
transferring knowledge through excellence in training

# CT and MRI for Advanced Practitioners Mini Series

## Session Two: CT and MRI of the brain and spine

Luis Mesquita DVM DipECVDI MRCVS



## Introduction

Nowadays computed tomography (CT) and magnetic resonance imaging (MRI) are routinely used in the investigation of spinal and brain diseases in dogs and cats. They are both superior to radiography in the diagnosis of spinal and brain conditions. MRI has a better overall diagnostic sensitivity than CT for neurological disorders and can be used to diagnose virtually all conditions of the spine and brain. However, there are patients in which CT is still the preferred method, for example, trauma caused by a gunshot wherein metallic foreign material is present that would cause considerable artefact in MRI, or for the diagnosis of subtle bone lesions, such as vertebral subluxation or small vertebral or skull fractures. Although a number of spinal conditions can be diagnosed with radiographs (e.g., discospondylitis), CT and MRI may still be warranted to better assess the extent of the condition, which may be important for diagnostic or therapeutic purposes. Earlier diagnosis of some conditions may also be permitted by CT or MRI.

## Basic MRI examination and interpretation principles

A standard MR examination of the canine or feline brain and spine involves the acquisition of multiple pulse sequences in different anatomic planes. A pulse sequence is a set of MRI parameters that culminate in images having contrast controlled by particular tissue properties. Pulse sequences are selected to accentuate different tissue properties to maximize lesion conspicuity. Images are usually acquired in transverse, sagittal, and dorsal planes (Fig.1).

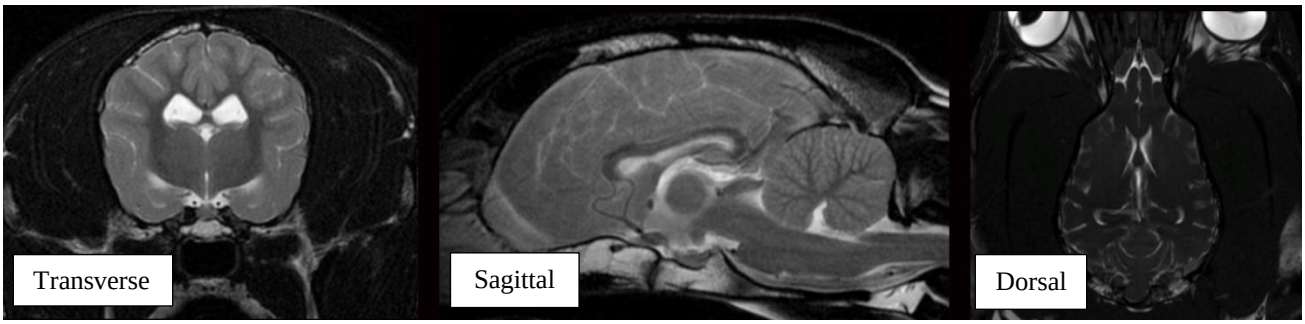



Fig.1 – T2w images in the transverse, sagittal and dorsal plane

Tissues emitting a high signal in a pulse sequence appear white in the image. This is often referred to as being “bright”, hyperintense relative to either normal tissue or other tissue within the same image, or as having sequence-specific hyperintensity (e.g., a focal region of T2 hyperintensity within the spinal cord). Tissues not emitting signal in a given pulse sequence appear dark or black, and these tissues are described as being hypointense relative to either normal tissue or other tissue within the same image, or as having sequence-specific hypointensity (e.g., a focal region of T1 hypointensity within the medulla oblongata).

A rationale selection of MRI sequences is important fundamental.

T2-weighted sequences are useful for detecting regions of increased fluid within tissues. In T2-weighted images, free fluid (e.g., CSF) is extremely bright. Most common pathologic processes, whether neoplastic or inflammatory, result in increased fluid within tissues, and this results in an increase in brightness of the abnormal tissue relative to the surrounding tissue; therefore T2-

weighted images are good to detect pathology. Sometimes it is difficult to identify a T2-hyperintense lesion when it is adjacent to a normal region of high signal, such as adjacent to CSF within the ventricles. Free fluid can be nulled using a fluid attenuating inversion recovery (FLAIR) sequence. In FLAIR images, free fluid has very low signal while other hydrated lesions remain bright. This makes the detection of subtle lesions adjacent to regions of fluid accumulation easier such as areas of oedema (Fig.2 arrow).

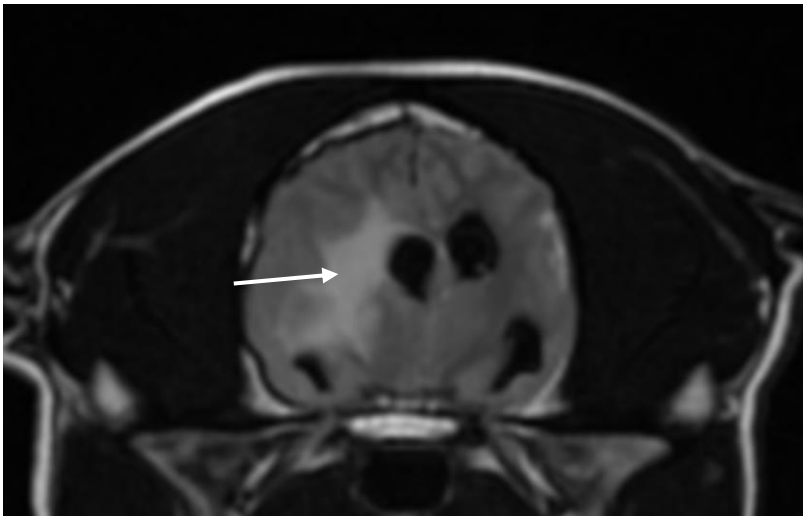


Fig.2

A FLAIR sequence may also provide additional information about T2-hyperintense lesions that must be distinguished from CSF, such as cystic meningioma, dermoid and epidermoid cysts, and arachnoid cysts. A cavity containing pure fluid will have markedly reduced signal in a FLAIR image, whereas a cavity with proteinaceous fluid or blood will have less or no signal reduction.

In T1-weighted sequences, fluid and hydrated lesions have reduced signal intensity appearing dark. A T1-weighted image is excellent for evaluation of extracranial anatomy, but on its own has poor sensitivity for detection of most intracranial lesions because of their hydrated nature, which leads to low T1 signal. However, regions of disruption of the blood-brain barrier and regions of altered perfusion can be detected on T1-weighted images acquired after the administration of an MR contrast medium. In regions of higher contrast concentration, signal will be increased in T1-weighted images. It is important to mention that the contrast medium should be administered after all other imaging sequences have been acquired, because contrast medium can sometimes affect the appearance of tissues/organs in other sequences. The following table resumes the signal intensity in T1w and T2w of some common materials (Fig.3).

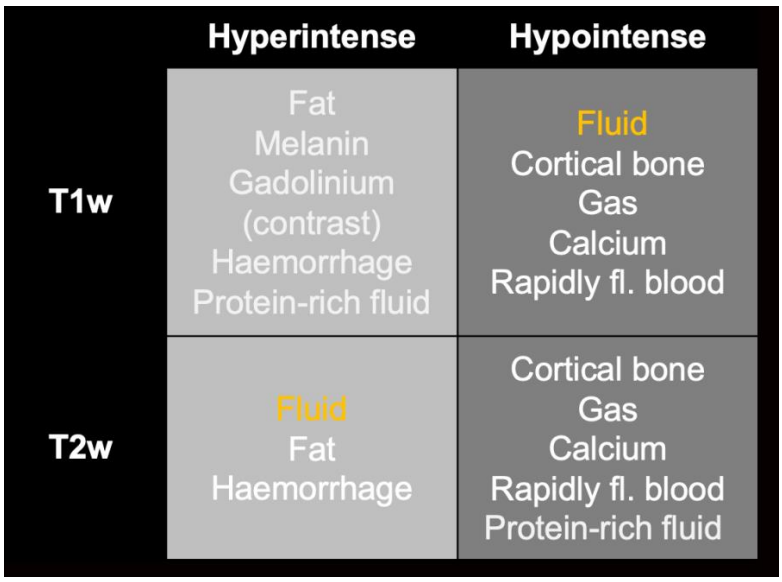



Fig.3

Images acquired using gradient recall echo (GRE) pulse sequence are more susceptible to magnetic field inhomogeneity, and this is exploited when assessing the brain or the spine for evidence of chronic haemorrhage (longer than 2 to 3 days). As the haemorrhage is broken down, the magnetic properties of the ferric/ ferrous ions in haemoglobin metabolites cause a local field distortion that destroys the MR signal. This appears as a signal void (a black, so-called susceptibility artefact), which is an extremely sensitive indicator of chronic haemorrhage as might occur in patients with intracranial trauma, haemorrhagic tumours, haemorrhagic infarcts, coagulopathies, or haemorrhagic metastasis.

A short inversion time recovery (STIR) sequence is commonly used in musculoskeletal imaging. This sequence suppresses the signal from the fat.

Putting all together, a minimum standard MRI study of the brain should comprise T2w, FLAIR, and T1w sequences in the transverse plane, a T2w sequence in the sagittal and in the dorsal plane and one T1w sequence in the transverse plane acquired after the administration of intravenous-contrast medium. The post contrast images should be acquired using the same parameters as the pre-contrast sequence to allow a direct comparison. A GRE sequence to assess for haemorrhage can also be performed if clinically indicated. A minimum standard MRI study of the spine should include T2w sequences in the dorsal and sagittal planes and T2w transverse images through the lesions noticed on the previous planes. T1w sequences pre and post contrast should also be performed when there is suspicious of inflammatory/infectious disease or a neoplastic process. A GRE sequence to assess for haemorrhage can also be performed if clinically indicated.

By evaluating the imaging characteristics of a lesion on various sequences, a more accurate assessment of the underlying disease process can be established.

## Introduction to the intracranial disorders

Many intracranial disorders may result in similar MRI or CT findings, and familiarity with signalment and pertinent history are crucial when evaluating the images. Intracranial lesions may be

extra-axial (i.e. originating outside actual brain parenchyma) or intra-axial (i.e. originating from brain parenchyma). Differential diagnoses for extra-axial lesions include neoplastic (e.g. meningioma), inflammatory (e.g. meningitis) and traumatic lesions (e.g. epidural hematoma). Differential diagnoses for solitary intra-axial lesions include hematoma, cyst, abscess/granuloma, infarct and neoplasia. Although inflammatory brain diseases usually appear as multifocal lesions, solitary masses may be encountered on occasion. Differential diagnoses for multifocal brain lesions include inflammation, infarcts, metabolic/toxic/nutritional encephalopathies and some intracranial neoplasms. Contrast enhancement of a lesion indicates vascularization and disruption of the blood–brain barrier.

Concurrent findings in intracranial disease

Pathologic sequelae associated with intracranial disease include mass effect, vasogenic edema, brain herniation, hydrocephalus, haemorrhage and various degrees of contrast uptake.

Space-occupying lesions within the cranial vault (e.g. tumour or edema) are commonly associated with a mass effect, even if the underlying lesion itself cannot be delineated. On MRI and CT this is usually seen as midline shift, transtentorial or transoccipital herniation and compression of the ventricular system.

Vasogenic edema is visible when there is damage to brain capillaries that results in leakage of fluid into the extracellular space, which migrates along the white matter fibber tracts. On CT vasogenic oedema appears as extensive hypodense areas, often exhibiting mass effect. On MRI vasogenic oedema usually appears as extensive areas of T2w and FLAIR hyperintensity and T1w hypointensity. Oedema does not uptake contrast in any of the modalities.

Brain herniation is usually caused by increase in intracranial pressure (e.g. due to an intracranial mass) that lead to compression and displacement of brain parenchyma. Evaluation of brain herniation is easier on sagittal images and may occur as:

- Foramen magnum herniation: herniation of the caudal portion of the cerebellum into and through the foramen magnum (Fig.4).
- Caudal transtentorial herniation: displacement of portions of the cerebral cortex (or cortices) ventral to the tentorium cerebelli.

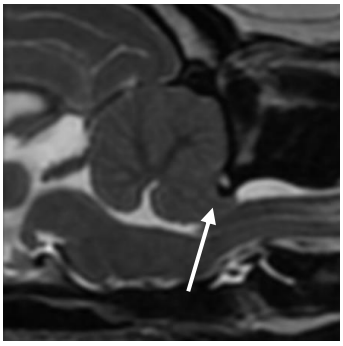


Fig.4

Hydrocephalus is defined as abnormal accumulation of cerebrospinal fluid within the cranium and is seen as dilatation of one or more ventricles and/or dilatation of the subarachnoid space. Potential

additional findings dependent on ethology may also be present (e.g. congenital anomalies, intracranial mass, traumatic lesions).
Intracranial haemorrhage can be classified based on location as epidural, subdural, subarachnoid, intraparenchymal or intraventricular. Epidural, subdural and subarachnoid haemorrhages are typically associated with trauma, while intraparenchymal haemorrhage may occur secondary to a variety of conditions including vascular malformation, coagulopathy, neoplasia and parasite migration. Intraventricular haemorrhage may be associated with trauma or extension of intraparenchymal haemorrhage into the ventricular system. The CT attenuation and MRI signal of intracranial blood are determined by the aggregation of globin molecules in the hematoma therefore changes with time. Gradient recall echo (GRE) MRI is an extremely sensitive indicator of chronic haemorrhage (see above).
MRI and CT studies are typically obtained before and after IV contrast administration and in normal conditions the blood–brain barrier prevents contrast medium from reaching the neural tissues. However, many intracranial conditions (e.g. inflammatory or neoplastic lesions) damage the blood–brain barrier resulting in contrast uptake. Extra axial lesions (e.g. meningiomas, pituitary tumours) typically have a strong contrast uptake due to their location outside of the blood brain barrier.

## Congenital and developmental brain anomalies

Congenital hydrocephalus

Congenital hydrocephalus occurs predominantly in brachycephalic and toy breeds and can be seen in CT and MRI as a generalised dilatation of the ventricular system with variable severity. In some instances, mechanical obstruction from such entities as mesencephalic duct stenosis explain the presence of hydrocephalus; in other cases, no underlying cause is recognized. Normal CSF has a density near that of pure water and will therefore have a HU value of close to 0 on unenhanced CT images and will be hypoattenuating to surrounding brain parenchyma. On unenhanced MR images, normal CSF will appear T1 hypointense and T2 hyperintense to brain parenchyma and will have no or low signal on FLAIR.

Chiari-like malformation

Chiari-like malformation is a disorder similar to Chiari type I malformation in humans and has been reported in dogs. Cavalier King Charles spaniels are most commonly affected, but the disease is seen in a variety of small and toy breeds and can be found in symptomatic and asymptomatic animals. This pathology is due to reduced volume of the caudal cranial fossa, resulting in cerebellar to caudal cranial fossa volume mismatch. The reduced caudal fossa volume results in crowding and repositioning of the cerebellum, which may sometimes encroach on or herniate through the foramen magnum. Cerebellar crowding also causes extramural compression of the fourth ventricle and central canal, which leads to obstructive hydrocephalus and syringohydromyelia. Clinical signs include pain, positional pain, hyperesthesia, and neurologic deficits, but severity of clinical signs correlate poorly to imaging findings.
On CT images, the caudal fossa will appear subjectively smaller than normal, which may be best appreciated on sagittal reformatted images. Obstructive hydrocephalus and syringohydromyelia may also be seen. Similar features will be seen on MR images, and a sagittal T2 sequence is often best for

detecting ventricular and central canal distension (Fig.5 white arrow) and for recognizing cerebellar displacement and foraminal herniation (Fig.5 black arrow).

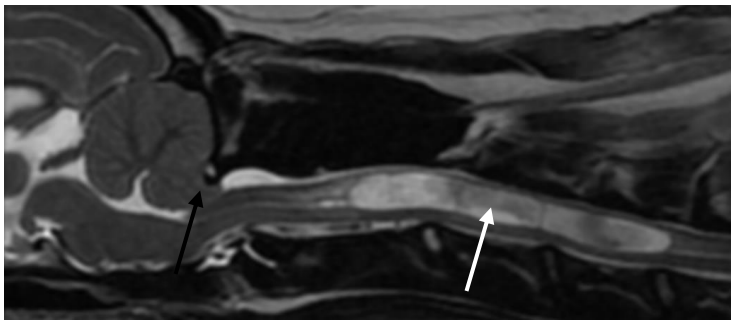


Fig.5

## Head trauma

CT is an excellent modality to assess patients with head trauma because it is fast and very accurate in the evaluation of bony structures and intracranial haemorrhage. 3D reformations of CT images of the skull may be helpful in depicting the spatial location of fragments. However, small fractures are often best seen in the two-dimensional images. CT findings in dogs and cats with head trauma include intracranial haemorrhage, skull fractures and brain oedema with mass effect associated. In chronic cases meningeal and brain parenchymal changes compatible with meningoencephalomyelitis may also be present.

## Inflammatory brain diseases

Inflammatory brain diseases can affect brain parenchyma (encephalitis), meninges (meningitis) or both (meningoencephalitis), and can be subdivided into infectious inflammatory and non-infectious inflammatory disorders. Although MRI is considered the diagnostic imaging modality of choice for inflammatory brain diseases, they may not cause changes detectable on MRI or CT. Most commonly, an analysis of CSF, including cytologic and immunologic testing, is required to help establish a definitive diagnosis. The CT and MRI studies of patients with inflammatory brain diseases can be normal or multifocal, intra-axial lesions may be seen. These lesions may have a small volume of perilesional oedema associated and are usually T2w hyperintense on MRI and hyperattenuating or hypoattenuating on CT. On post-contrast, these intra-axial lesions and the meninges may uptake contrast. A normal MR or CT study does not rule out the possibility of inflammatory disease, and a CSF tap is required. In one study of 25 patients with CSF alterations consistent with inflammatory brain disease, the specificity of MRI to detect brain changes was 76%.

## Intracranial tumours

Intracranial tumours can be characterized by anatomic location, distribution (intra-axial, intraventricular, extra-axial), CT density or MR signal characteristics, intensity and pattern of contrast enhancement, tumour margin definition, secondary mass effects, and the extent of associated brain edema. Although biopsy is necessary for definitive diagnosis, this constellation of imaging features can often lead to a specific or differential clinical diagnosis.

Extra-axial tumours

Meningiomas are extra-axial tumours that arise from dural elements. They are the most common intracranial tumour in cats and one of the most common in dogs. Meningiomas are usually benign and grow slowly. They are variable in size and shape and may be irregular, nodular, ovoid, lobulated, or plaque-like, ranging from a few millimetres to several centimetres in diameter. Meningiomas are often firm and encapsulated, are usually discrete, and may contain mineralization or pockets of fluid. Basal and plaque like meningiomas are common in the floor of the cranial cavity, especially in the optic chiasm and suprasellar regions. They also occur commonly over the cerebral hemispheres, less commonly in the cerebello-pontomedullary region, and rarely in the retrobulbar space, arising from the optic nerve. An important differential for a meningioma is round cell neoplasia (histiocytic sarcoma and lymphoma). Multiple meningiomas can occur; this is more common in cats than in dogs. Thickening of bone adjacent to meningiomas, termed hyperostosis, may occur, especially in cats.

On both MR and CT images, approximately 60–70% of meningiomas show marked, uniform contrast enhancement (Fig.6), with the remainder being heterogeneous and often associated with cystic, haemorrhagic, or mineralized components. Contrast enhancement usually reveals well-defined tumour margins; a globoid, plaque like, or irregular shape; and a broad-based superficial margin conforming to the meningeal plane. Meningiomas are often associated with a dural tail, which is a linear enhancement of thickened dura mater adjacent to an extra-axial mass seen on postcontrast images. It is uncertain whether the dural tail represents neoplastic infiltration beyond the margins of the meningioma or a manifestation of associated inflammation. The amount of disruption to adjacent parenchyma is variable, depending on tumour size and location. Some meningiomas have minimal peripheral edema, although others may have extensive peripheral edema that can result in ventricular compression and brain herniation.

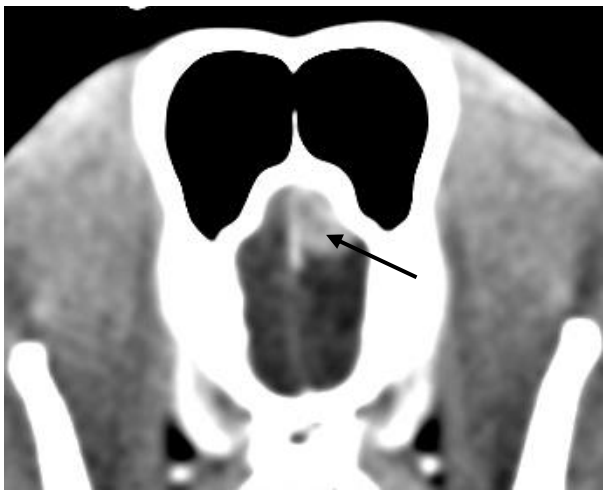


Fig.6

Pituitary tumours are common in dogs but uncommon in cats. They may be non-functional or functional. Functional pituitary tumours are typically characterized by pituitary-dependent hyperadrenocorticism (PDH). Up to 60% of dogs with PDH but without neurologic signs have a

pituitary tumour 4 to 12 mm in diameter at greatest vertical height. Most pituitary tumours tend to grow dorsocaudally, leading to compression and obliteration of the infundibulum, ventral aspect of the third ventricle, hypothalamus, and thalamus. They eventually impinge on the internal capsule and optic tracts. Pituitary tumours may be visible in CT and MRI but MRI is useful for visualizing the presence of both microtumours (3 to 10 mm in diameter) and macrotumors (>10 mm) in dogs with PDH, with or without neurologic signs, especially when endocrine test results are equivocal. Pituitary tumours are always better visualized after contrast medium administration. Usually these tumours have minimal peritumoral edema, uniform contrast enhancement, and well-defined margins. Cystic regions, or evidence of chronic or recent haemorrhage, sometimes extensive, may be present. Pituitary tumours less than 3 mm in diameter may not be visible with MRI.

Choroid plexus tumours are common in dogs, occurring most commonly in the third ventricle and in the lateral recess of the fourth ventricle. Choroid plexus tumours tend to bleed, and exfoliation of choroid plexus cells, from both benign and malignant variants, may occur with subsequent tumour seeding to other areas of the brain or spinal cord through the CSF. Because of the intraventricular location of choroid plexus tumours, obstructive hydrocephalus is common and may be life-threatening.

Additionally, some choroid plexus tumours will cause an overproduction of CSF, which exacerbates any obstructive process. Like meningiomas, choroid plexus tumours usually have marked contrast enhancement and sometimes have evidence of haemorrhage and/or dystrophic mineralization.

The MRI and CT characteristics of intraventricular ependymomas are similar to choroid plexus tumours, but ependymomas are much less common. Other ventricular tumours include meningiomas.

Intra-axial tumours

The term glioma is used to describe tumours that arise from the neuropil. Gliomas include astrocytomas, oligodendrogliomas, and glioblastoma multiforme and are particularly common in brachycephalic breeds, such as the boxer, Boston terrier, and bulldog. Gliomas range in malignancy from low grade and slow growing, to high grade, poorly differentiated highly malignant tumours.

Gliomas vary widely in their MR and CT features. They are often difficult to detect using contras enhanced CT imaging of the brain because, unlike meningiomas, many gliomas do not enhance, or enhance only minimally, after contrast-medium administration. These tumours are more easily detected with MRI, and this is one of the many reasons that MRI is so clinically superior to CT imaging when evaluating the brain. Gliomas are often ill-defined, have variable degrees of perilesional edema, and variable contrast enhancement. Occasionally no contrast enhancement is present. A glioma can be difficult to differentiate from a brain abscess and other focal inflammatory conditions of the brain parenchyma or even from massive infarction. The MR findings are nonspecific, mimicking many much more common conditions, including encephalitis, leukodystrophies, and metabolic encephalopathies. Typically, one might expect to see a diffuse increase in T2 and FLAIR signal throughout regions of the cerebrum with some mass effect but overall relative preservation of the neural morphology and minimal contrast medium enhancement. The CT findings are variable, ranging from an ovoid or amorphous mass to a diffuse infiltrate with distinct to poorly defined margins. On pre-contrast gliomas are usually hypodense, isodense to hyperdense and their contrast enhancement

on post-contrast ranges from none to strong.

## Metabolic, nutritional and toxic brain diseases

There are a number of nutritional disorders that result in morphological signal change within the brain that are detectable with MRI. This includes lysosomal storage diseases, L-2-hydroxyglutaric aciduria thiamine deficiency, hepatic encephalopathy, etc. Most commonly nutritional, toxic, and metabolic disturbances are bilaterally symmetrical. There is considerable overlap in the MR features of many nutritional, metabolic, and toxic disorders; and this confounds the MR assessment. Generally, the presence of lesion symmetry should alert the reader to the possibility of such disorders.

## Intervertebral disc disease

The normal intervertebral disc

Intervertebral discs are made of a peripheral annulus fibrosus and a central nucleus pulposus. The disc lies in close contact with the cartilaginous vertebral end plates with fibers from the nucleus pulposus and annulus fibrosus being interwoven with the collagen fibers of the cartilaginous end plates and bony trabeculae. Both the annulus and the nucleus are made of fibrocartilage but differ in the amount of collagen and ground substance. There is more collagen and less ground substance in the annulus than in the nucleus. The ground substance is composed of hyaluronic acid and glycosaminoglycans that hold water because of their strong negative charge. A normal nucleus pulposus has a gelatinous consistency. Water, bound to large proteoglycan molecules, is the principal component of the nucleus pulposus (80% to 88%). On MRI, normal nucleus pulposus has very bright signal of on T2-weighted images.

Intravertebral disc degeneration, intervertebral disk extrusion and protrusion

As a disk degenerates, the nucleus pulposus and, to a lesser extent, the remainder of the disk dehydrate, causing narrowing of the disk. Nonphysiologic loading of the disk can lead to annular tears and cartilaginous endplate fissures. Structural changes to the disk lead to herniation or extrusion.

Disk degenerative changes differ between chondrodystrophic and nonchondrodystrophic breeds. In chondrodystrophic breeds starts earlier (3–7 years), affects the entire vertebral column and commonly results in dystrophic mineralization as a sequela. In nonchondrodystrophic breeds the process is usually more focal and occurs later (6–8 years) in life.

Intervertebral disk lesions are classified as type I or type II using a system first introduced by Hansen. Hansen's type I disk extrusion occurs when degenerated nucleus pulposus herniates through all layers of a ruptured annulus fibrosis. Type I disease occurs predominately in chondrodystrophic breeds but is also seen in larger nonchondrodystrophic breeds. Type I disk extrusion tends to be acute and explosive. Because of the eccentric position of the nucleus within the disk, herniation occurs dorsally into the vertebral canal or dorsolateral into the intervertebral foramina. Hansen's type II disk protrusion occurs when fibroid degenerated disk material migrates dorsally or dorsolateral because of partial tearing or rupture of the annulus. Because the nucleus pulposus is still contained

within the remaining annulus fibrosus, disk material is not extruded. Hansen's type II disk protrusion results from fibrous degeneration and is most common in nonchondrodystrophic breeds.
There are several studies that have compared the accuracy of unenhanced CT, contrast-enhanced CT, MRI, and conventional myelography for detection of Hansen's type I disk herniation. Unenhanced CT has been reported to be 89–100% accurate for lesion localization, and CT myelography is slightly bet- ter. MRI is thought to be the most accurate imaging method, but the degree of improvement compared to CT myelography is minor. CT features of type I disk extrusion include the presence of disk material in the epidural space, with the density depending on the degree of mineralization (Fig.7). Disk material can migrate horizontally along the floor of the vertebral canal and circumferentially around the spinal cord. Material can also be dorsolateral extruded into the intervertebral foramina. Depending on the volume and distribution of extruded disk material, the spinal cord is displaced and compressed. Subarachnoid contrast columns are attenuated at the site of compression on CT myelography. Diffuse alterations with mixed attenuation in the epidural space can be seen in acute disease associated with haemorrhage, and edema can cause an increase in cord diameter. The affected intervertebral disk space is often narrowed, and residual mineralized in situ disk material is sometimes present. Similar features are seen on MR images, with disk material appearing T1 and T2 hypointense. Attenuation of the T2 hyperintense cerebrospinal fluid layer occurs at the site of cord compression, and T2 hyperintensity of cord parenchyma may also be seen as a result of edema. When present, haemorrhage appears as variable, mixed T1 and T2 intensity.

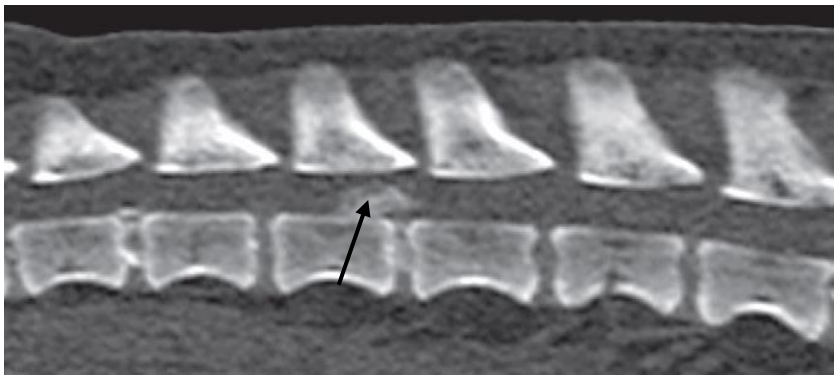


Fig.7

For detecting type II disk protrusions CT may be less accurate. CT features include a variable decrease in intervertebral disk space width and a mildly hyperattenuating mass arising from the dorsal aspect of the affected disk and extending into the ventral or ventrolateral vertebral canal. The bulging annulus cannot be distinguished from the overlying dorsal longitudinal ligament. The spinal cord is displaced, and its shape is often distorted by impingement of the disk even when overt compression is absent. Contrast columns are attenuated at the site of impingement or compression on CT myelographic images. MR features of type II disk protrusions are similar to those seen on CT images. Protruding disk material is T1 and T2 hypointense and appears contiguous with in situ disk material and the overlying longitudinal ligament. The spinal cord can be displaced, distorted, and compressed, and the T2 hyperintense cerebrospinal fluid columns are attenuated at the site of protrusion. It is common to see multiple sites of involvement with varying degrees of disk

protrusion. In patients with chronic disease, the spinal cord can be focally atrophic, with syringohydromyelia and T2 parenchymal intensity suggesting gliosis. Uninvolved disks are often T2 hypointense because of dehydration.

Hydrated nucleus pulposus extrusion

Extrusion of apparently normal disk material can also occur as a result of physical activity or overt trauma. These are sometimes referred to as high-velocity extrusions because of the force of extrusion and the predominately liquid composition of normal nucleus pulposus. Acute spontaneous extrusion of hydrated disk material seemingly unrelated to activity or trauma can also occasionally occur. Clinical signs include acute onset tetraparesis or tetraplegia, and the mid to caudal cervical intervertebral disks are most commonly affected.

Hydrated nucleus pulposus extrusions may be non-compressive or compressive. In the non-compressive form, the normally hydrated nucleus material, diffuses in the epidural fat, leaving only the secondary changes attributable to acute spinal cord contusion with little or no spinal cord compression. In the compressive form, T2w hyperintense of extruded disk material is visible but is difficult to distinguish from epidural fat. Sometimes a characteristic "seagull sign" may be present on T2w transverse images representing the dorsal margin of the extruded material. In both forms narrowing of the intervertebral disk may be present. In CT, non-compressive hydrated nucleus pulposus extrusion are normal and the compressive form can only be visible with myelography.

## Fibrocartilagenous embolism

Fibrocartilaginous embolism (FCE) occurs primarily in middle-aged to older, large- and giant-breed dogs; however, a more recent review in which diagnosis was based on clinical signs and MR imaging findings suggests that small and medium-sized dogs are also commonly affected. The disorder has also been reported in cats. The clinical presentation is often a peracute onset of symmetrical or asymmetrical motor dysfunction immediately following exercise or minor trauma, with lower motor neuron signs also present in some patients. Initial clinical signs can include transient pain and can be progressive for the first 2 hours but are often nonprogressive thereafter. The cervicothoracic (C5–T2) and lumbosacral (L3–S3) regions appear to be predisposed. Pathology in histologically confirmed patients includes spinal cord infarction and haemorrhage with cartilaginous emboli in meningeal or spinal vessels. A poorer prognosis is seen in patients with involvement of the intumescences, symmetrical neurological signs, and decreased deep pain sensation. A recent review of dogs with ischemic myelopathy found that a combination of lesion length greater than twice a vertebral length and cross-sectional involvement of greater than 67% had a positive correlation with an unsuccessful outcome. Recovery rates for FCE are unclear since patients who do respond are not definitively diagnosed. However, a majority of patients with stable disease seem to partially or fully recover neurologic function.

CT imaging features are very limited to a non-compressive focal increase in spinal cord diameter, suggestive of an intrinsic lesion. MRI features are similar to the non-compressive hydrated nucleus pulposus extrusion and include focal T1w iso- to hypointensity and T2w hyperintensity within the affected spinal cord segments. Lesions preferentially affect gray matter and can be either

symmetrical or asymmetrical. Spinal cord diameter can also appear locally enlarged but without compression.

## Spinal tumours

Spinal tumours can be classified on the basis of their location relative to the dura mater and can be described as intramedullary, intradural-extramedullary, or extradural. Most extradural tumours are malignant tumours of constituents of the vertebral column, such as the vertebrae or the soft tissues within or surrounding the vertebral column. Intradural-extramedullary tumours are mostly nerve sheath tumours and meningiomas. Intramedullary neoplasms are usually of glial cell origin, such as astrocytomas or ependymomas, but multicentric non-neural neoplasia (such as, lymphoma) or metastatic lesions (such as, hemangiosarcoma) also occur.

Extradural tumours

The most common extradural spinal cord tumours in dogs include osteosarcoma and chondrosarcoma. In cats, extradural lymphosarcoma is also frequent. Solitary or disseminated histiocytic sarcoma can also affect the vertebrae and cause spinal cord compression. Other disseminated canine tumours with possible vertebral involvement include lymphosarcoma and multiple myeloma. Metastatic vertebral tumours are also common, in particular prostatic carcinoma or transitional cell carcinoma of the bladder and urethra in dogs. Tumours of the paravertebral soft tissues or extradural soft tissue components of the vertebral canal are also possible, and local invasion can cause spinal cord compression. Examples include soft tissue sarcoma, extradural lymphosarcoma, and infiltrative lipoma. Both MRI and CT are more sensitive than radiography in detecting vertebral lesions and in particular bone lysis that is not apparent radiographically can be demonstrated readily using CT. Vertebral canal tumours can be difficult to localize to the intradural, extradural, or intramedullary compartments on non-contrast CT images. Injection of intravenous contrast medium and, more important, CT myelography are more precise for determining the location of masses in the vertebral canal with respect to the dura. MRI is also good for localizing these lesions. There are no specific CT or MRI features that are associated with a specific tumour type. Vertebral tumours produce variable degrees of bone lysis or proliferation, which is detected easily on CT images. On MR images, alteration of the shape of the affected vertebra can be seen, as well as disruption of the normally hypointense vertebral cortex. In MR images, periosteal reaction appears as hypointense material surrounding the lesion in the affected vertebra. The signal intensity of the lesion itself varies between patients and depends on tumour type. Fat-suppression images are useful for differentiating bone marrow fat accumulation from true lesions. Both appear hyperintense on T1- and T2-weighted images, but with fat suppression, fat signal will decrease but tumour will remain bright. Paravertebral soft tissue tumours can also be imaged well with CT and MRI and can be recognized before they become visible radiographically. MRI and CT also allow assessment of surgical respectability and can be used for radiation-therapy planning. Infiltrative lipomas have specific imaging characteristics. When in a paravertebral location, an infiltrative lipoma can invade the vertebral canal and cause neurologic signs. On CT imaging, infiltrative lipomas are characteristically hypoattenuating, which is similar to normal fat. On MRI, infiltrating lipomas are

T1w and T2w hyperintense, and fat suppression techniques are useful to confirm their fatty composition.

Intradural-extramedullary tumours

Meningioma is the most common central nervous system neoplasm of the spinal cord in dogs. Median age at onset of clinical signs is 9 years, and Golden Retrievers and Boxers appear to be overrepresented. Most canine spinal meningiomas are World Health Organization (WHO) grade I or II, with a small minority being more biologically aggressive grade III. Nearly 70% are located in the cervical region, about 25% are lumbar, and the remainder are thoracic or multifocal. Although less common, spinal meningioma has also been reported in the cat.

On CT images, spinal meningiomas are soft-tissue attenuating space-occupying masses within the vertebral canal that variably displace and compress the spinal cord, depending on tumour size in relation to the vertebral canal diameter. Meningiomas uniformly enhance following intravenous contrast administration and appear as a contrast-filling defect within the subarachnoid space on CT myelography. On MR images, meningiomas are mildly to moderately T1w hyperintense, mildly to markedly T2 hyperintense, and uniformly and intensely contrast enhancing. A dural tail sign may be present in some instances but is not consistent. Using either imaging modality, localizing a meningioma to the intradural–extramedullary compartment may not be possible when the tumour mass is large.

Peripheral nerve sheath tumours (PNST) includes neoplasms that originate from Schwann cells, fibroblasts, or perineural cells. Age of onset in dogs is reported to be bimodal, peaking at 2–3 years and 7–9 years, with no apparent breed predilection. Small PNSTs arising from nerve roots contained within the meninges and limited to an intradural–extramedullary distribution within the vertebral canal have CT and MR imaging features similar to spinal meningiomas and cannot be reliably differentiated from other intradural– extramedullary neoplasms. However, PNSTs are more likely to invade spinal cord parenchyma and can also extend along peripheral nerves external to the vertebral canal, taking on a more tubular or lobular shape.

Intramedullary tumours

In a report of 53 dogs with intramedullary spinal cord neoplasia, approximately two thirds of the tumours were of neuroepithelial origin. The remainder were metastatic neoplasms, the most common of which were hemangiosarcoma and transitional cell carcinoma. In this study, ependymoma was the most common neuroepithelial tumour, followed by astrocytoma. The imaging appearance of spinal cord neoplasms are similar intracranial neuroepithelial neoplasms. A common feature of all intramedullary neoplasms is the presence of an intraparenchymal mass that causes an increase in spinal cord diameter and annular narrowing of the surrounding subarachnoid space. This appears as circumferential attenuation of the subarachnoid space on CT myelographic or T2 and STIR MR images.

## Discospondylitis

Discospondylitis occurs commonly in dogs and is rare in cats. Infection is caused by a wide variety of bacterial and mycotic species. Although the vertebral imaging features of bacterial and fungal discospondylitis can appear similar, the underlying clinical manifestations are quite different.

Bacterial (suppurative) discospondylitis

In a large retrospective study involving over 500 canine patients diagnosed with discospondylitis, two thirds were male, older dogs were more likely to be affected, and Great Danes were overrepresented. Staphylococcus, Brucella, Streptococcus, and Escherichia species are most frequently isolated, although many others have been reported. Dogs with bacterial discospondylitis most often have an underlying infection of the urinary tract, skin, or other organ system, which leads to bacteraemia and embolic seeding of vulnerable disks.

Conventional radiographic examination is a good test for diagnosis and monitoring of discospondylitis but the radiographic changes of discospondylitis lag behind changes that are apparent in MR images, thus negative radiographs do not rule out a diagnosis of discospondylitis. Imaging features vary widely and depend on the stage of the disease. CT features of early active disease can include vertebral endplate osteolysis and intervertebral joint space widening. In later phases of active disease, more pronounced endplate destruction is seen, which is associated with underlying bone sclerosis, reactive new bone formation, and collapse of the disk space. If there is significant proliferative soft tissue inflammatory response or intervertebral joint subluxation, spinal cord compression can occur with resultant neurologic signs. In the convalescent or reparative phase, complete collapse of the joint may occur with bridging reactive new bone. Soft tissues within the disk space, medullary bone, and surrounding soft tissues moderately to markedly contrast enhance during the active phases of disease, reflecting the presence of discitis, osteomyelitis, and cellulitis.

MR features of bacterial discospondylitis are similar and include mixed T2w intensity within the disk space and T1w hypointensity and T2w and STIR hyperintensity within affected vertebral bodies and adjacent soft tissues during the early active phase of disease. The disk, medullary bone, and adjacent soft tissues intensely contrast enhance during the active phases of disease. MR may be less sensitive than CT for monitoring bone destruction and production in the active and reparative phases, respectively.

Mycotic (granulomatous) discospondylitis

Mycotic discospondylitis is almost always a component of systemic infection with Aspergillus or Paecilomyces species, although other fungi have also been reported. Age of onset is 2–8 years, and German Shepherd Dogs and females are highly overrepresented. Affected dogs are thought to be immunocompromised, resulting in multiorgan involvement.

Imaging features of mycotic discospondylitis are similar to those of bacterial discospondylitis, and often multiple intervertebral disks are affected.

## Meningomyelitis

Meningitis and myelitis can have an infectious or non-infectious origin. Infectious agents in dogs include viruses (e.g., canine distemper), bacteria (e.g., Staphylococcus, Pasteurella, Actinomyces, and Nocardia spp.), fungi (e.g., Cryptococcus, Blastomyces, Histoplasma, and Coccidioides spp.), Rickettsiae (e.g., Ehrlichia spp., Rickettsia rickettsii), protozoa (e.g., Toxoplasma gondii, Neospora caninum), parasites (e.g., Dirofilaria immitis, Angiostrongylus spp.), and rarely algae (e.g., Prototheca wickerhamii, Prototheca zopfii). In cats, feline infectious peritonitis and toxoplasmosis have been reported.

Non-infectious causes of canine meningomyelitis include granulomatous meningoencephalomyelitis, pyogranulomatous meningoencephalomyelitis, and steroid-responsive meningitis arteritis.

CT changes in dogs with meningomyelitis include spinal cord swelling, spinal cord hypoattenuation, and occasionally intramedullary or meningeal contrast enhancement.

On MRI, abnormalities include irregular areas of intramedullary T2-hyperintensity. On pre-contrast T1w images, these areas are either isointense or hypointense to the spinal cord and have various amounts of contrast enhancement. The pattern is not specific for meningomyelitis, and other conditions (such as, myelomalacia or ischemic myelopathy) can produce similar changes. Therefore, correlation with clinical signs and laboratory test results is necessary. In cats, lymphoma can produce similar MRI changes.

## Bibliography


- Thrall DE. Textbook of veterinary diagnostic radiology 7th edition.
- Schwarz T, Saunders J, editors. Veterinary computed tomography.
- Wisner E, Zwingenberger A. Atlas of small animal CT and MRI.