

## Imaging the Seizuring Patient

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Magnetic Resonance Imaging is the gold standard imaging technique to investigate the seizing patient. With technological and computing advances, and increased accessibility of MRI machines to the veterinary market, there has been a rapid expansion in our knowledge of the application and MRI findings in this area.

This test is useful to distinguish between 'idiopathic', structural and in some cases metabolic causes of seizures. Findings from the MRI can be used to guide prognostication and case management. It should be acknowledged that the MRI presents a significant cost burden to the client or pet insurance companies. It is therefore important that we are considered in selecting this test, are equipped with knowledge to appropriately interpret the study, and are aware of new MRI techniques as they emerge to help us investigate the seizing patient.

### 1. A brief refresher on contemporary definitions.

1.1. In 2015, the International Veterinary Epilepsy Taskforce published a series of consensus statements relating to various aspects of canine epilepsy. Importantly, they provided a series of guidelines on the definition of epilepsy, the classification of epilepsy syndromes and the terminology which should be applied to epilepsy in companion animals (Berendt 2015). Key definitions are provided below:

1.1.1. **Epileptic seizure** - a transient occurrence of signs which may be characterised by short episodes with convulsions or focal motor, autonomic or behavioural features due to abnormal excessive and/or synchronous epileptic neuronal activity in the brain

1.1.2. **Epilepsy** - a disease of the brain characterised by an enduring predisposition to generate epileptic seizures. This definition is usually practically applied as having at least two unprovoked epileptic seizures >24 h apart

1.1.3. **Structural epilepsy** - epileptic seizures which are provided by intracranial/ cerebral pathology, including vascular, inflammatory/infectious, traumatic, anomalous/developmental, neoplastic and degenerative disease

1.1.4. **Idiopathic epilepsy (IE)** can be sub-classified into three sub-groups:

1.1.4.1. **IE - genetic epilepsy** - a causative gene has been identified

1.1.4.2. **IE - suspected genetic epilepsy** - a genetic influence is supported by high breed prevalence (>2%), genealogical analysis and/or familial accumulation of epileptic individuals

1.1.4.3. **IE - unknown cause** - nature of cause is not known and there is no indication of structural epilepsy

- 1.2. Guidelines are also provided on the diagnostic approach to epilepsy in dogs (De Risio 2015). Idiopathic epilepsy is a diagnosis of exclusion, and is made based on the age at onset of epileptic seizures, unremarkable inter-ictal physical and neurologic examination, and exclusion of metabolic, toxic and structural cerebral disorders. There are three 'Tiers' of level of confidence for a diagnosis of IE, with increasing level of confidence with additional tests of MRI, CSF analysis and inter-ictal EEG. Idiopathic epilepsy has a typical age of onset between 6 months and 6 years, although has been reported outside that age range.
- 1.3. In cats, similar to dogs, IE is a diagnosis by exclusion. IE is most likely in cats with seizure onset between 1 and 5 years (Hazenfratz, 2018)

## **2. Judicious selection of MRI in the seizing patient - how signalment influences expectations of MRI findings in the seizing patient**

The cost of an MRI study is not inconsiderable to the owner, and also requires the patient to submit to general anaesthesia for typically an hour or more. Unless this diagnostic test is likely to change the outcome or management of the case, there may be little achieved by subjecting our patients to an MRI. There are many variables which influence the likelihood of identifying structural lesions, and *"It may be pertinent ... to consider risk on a patient-patient basis when recommending an MRI scan in these cases."* (Phillips 2025)

Clinical features which increase the likelihood of identifying structural lesions on MRI include an abnormal inter-ictal neurologic examination (Phillips 2025); in dogs and cats with a very young (<6 months to <1year) or very old (>6 or 7 years) age of seizure onset (Podell 1995, Smith 2008, Armasu 2014, Raimondi 2017); brachycephalic breed (Prodger 2025). Focal versus generalised seizure types were not useful to predict the likelihood of structural brain disease, as focal epileptic seizures have been reported in dogs with IE (Armasu 2014).

Putting it all together: The IVETF guidelines (DeRisio 2015) combine the interictal neurologic status and age at epileptic seizure onset to attempt to predict the probability of identifying structural disease. Their recommendations are to perform MRI of the brain and routine CSF analysis, after exclusion of reactive seizures, in dogs with:

- age at epileptic seizure onset <6 months or >6 years
- interictal neurological abnormalities consistent with intracranial neurolocalisation
- status epilepticus or cluster seizure
- a previous presumptive diagnosis of IE and drug-resistance with a single anti epileptic drug titrated to the highest tolerable dose

### 3. The epilepsy-specific brain MRI protocol

The rationale for having an epilepsy-specific brain MRI protocol centres around optimising image quality and planes of acquisition to maximise the ability to detect structural lesions, including subtle pathologies such as hippocampal sclerosis, cortical dysplasia or atrophy. A standardised protocol would also facilitate the use of case material in future clinical research investigating the pathophysiology of epilepsy.

The International Veterinary Epilepsy Taskforce issued a consensus statement on an epilepsy-specific brain protocol (Rusbridge 2015). This protocol focuses on planes which are aligned to the hippocampus, and has similarities with the Epilepsy MRI protocol used in humans.

The protocol for a 1.5T unit is summarised as follows, with details of technical parameters, and an alternative for low-field MRI available through links in the article:

*Slice thickness 3mm or less*

- *T2W - 3 sequence orientations*
- *Sagittal (enabling identification of the long axis of the hippocampus)*
- *Dorsal (perpendicular to the long axis of the hippocampus)*
- *Transverse (parallel to the long axis of the hippocampus)*
- *FLAIR - 1-2 sequence orientations for hippocampal angulation*
- *Dorsal (perpendicular to the long axis of the hippocampus)*
- *Optional Transverse (parallel to the long axis of the hippocampus)*
- *T1W*
  - *3D technique at 1mm isotropic voxel size, or routine T1W dorsal ((perpendicular to the long axis of the hippocampus)*
  - *T1W post-contrast, if indicated by other pathology or desired by the clinician*
- *Hemosiderin/calcification sensitive sequences eg. Gradient echo or SWI*
  - *Transverse (parallel to the long axis of the hippocampus)*

### 4. Imaging findings in Structural Epilepsy

Recent estimates of the prevalence of structural epilepsy in dogs range from 25% (Milne 2018) to 45% (Hall 2020). Brain tumours were the most common cause of structural epilepsy, representing 60% of dogs. In 11% of cases the diagnosis was meningoencephalitis of unknown aetiology. 6.7% of dogs had abnormal intracranial fluid accumulation, either hydrocephalus (n=4) or a large supracollicular fluid accumulation (n=2). 5.6% had a cerebrovascular accident, with diffusion restriction confirmed using diffusion-weighted imaging. Congenital malformations were only identified in 3 dogs, and infectious

encephalitis (neosporosis) was identified in a single dog. In 12% of cases, the diagnoses was not certain. (Milne 2018)

In a large study looking at the prevalence of structural epilepsy (Armasu 2014), 92% of dogs had neoplasia, inflammatory brain disease or vascular lesions, while 8% of dogs had hydrocephalus.

## **5. Imaging findings in “Idiopathic” Epilepsy**

By definition, patients with ‘idiopathic epilepsy of unknown cause’ should not have causative structural brain lesions. This is, of course, limited by the level of detail available on MRI, the knowledge and experience of the interpreter, and the physical limitations of interpretation for detecting some subtle structural change. See sections below.

There may be reversible MRI changes to the brain in dogs following seizures, which are generally attributed to a mixture of cytotoxic oedema and gliosis. In a study of 540 client-owned dogs with epilepsy that underwent brain MRI (Maeso 2021), 12.4% (67/540) showed post-ictal changes on MRI. Such post-ictal changes may be solitary or multiple but are usually bilaterally symmetric, are typically T2W and FLAIR hyperintense, T1W hypo- or isointense with no to mild contrast enhancement, are not confined to a vascular territory, and show no to mild mass effect (Nagendran 2021). The typical location for such lesions is in the piriform and temporal lobes, the cingulate gyrus and the hippocampus (Mellema 1999, Maeso 2021).

It is important to try and distinguish these post-ictal changes from a potentially epileptogenic lesion. As most post-ictal changes resolve within 16 weeks (Mellema 1999), a repeat MRI scan following a 16-week seizure-free interval would be useful to check for lesion resolution or associated brain atrophy.

## **6. In search of hippocampal sclerosis**

The hippocampi are paired structures and are part of the mesial temporal lobe. They are responsible for converting short-term to long-term memories, spatial memory and verbal memory. They have a high oxygen requirement and is thus susceptible to hypoxic injury and glutamate excitotoxicity.

Hippocampal sclerosis is neuronal loss or atrophy, and gliosis. On MRI, its features are a reduction in volume and an increase in T2W or FLAIR signal intensity. Hippocampal sclerosis may be the original cause of seizures, or a consequence of seizures leading to a new epileptogenic focus. In humans with temporal lobe epilepsy, hippocampal sclerosis is commonly identified and represents an important surgical target in patients with refractory epilepsy.

Hippocampal necrosis and sclerosis are well documented in cats (Wagner 2014, Claßen 2016, Klang 2018). A colony of cats with familial spontaneous epilepsy has been extensively

studied, and hippocampal asymmetry and atrophy is frequently identified in that cohort (Kuwabara 2010, Mizoguchi 2014). Hippocampal necrosis occurs in 6–30% of seizing cats (beyond the epileptic colony) and is most prevalent in cats with severe seizure disorders that have had at least one observed episode of cluster seizures or status epilepticus (Wagner 2014). Research by Klang and co-authors (Klang 2018) supports the notion that there is a gradual transition from hippocampal necrosis to hippocampal sclerosis.

Conclusive proof of the existence of hippocampal sclerosis in dogs has been a little more problematic. This may partly relate to the likely low incidence of this pathology in dogs, and methods of case selection for group-wise comparisons. Without prioritising the selection of dogs with temporal lobe epilepsy and refractory epilepsy, the statistical impact of any dogs with small hippocampal volumes will be diluted by dogs with other forms of epilepsy and normal hippocampal volumes. This case selection bias may have been the cause of the lack of a statistically significant difference in hippocampal volumes between epileptic and non-epileptic groups of dogs in previous research (Milne 2018, Lorincz 2021). An alternative approach uses a hippocampal asymmetry ratio with a threshold of 6% (borrowed from the human literature), which compares right and left hippocampal volume within the same individual, to detect unilateral hippocampal atrophy. Early work by Kuwabara and co-authors (Kuwabara 2010) found asymmetry in 12% of epileptic dogs. This work was, however, based on thick-slice transverse plane T2W sequences, was unable to detect bilateral hippocampal atrophy, was based on a human-derived threshold and was not supported by histology. Another approach is to compare the hippocampal volumes in individual epileptic dogs with an established lower reference limit from normal dogs. Interestingly, dogs with idiopathic epilepsy and a small hippocampus had a statistically significant younger age of onset of epilepsy (median 1.4 years, range 0.25 to 6 years) compared with epileptic dogs with a normal hippocampal size (median 4.9 years, range 0.25 to 14 years). They were also younger at the time of imaging (median 1.7 years compared to 5 years). (Milne 2018)

Other work in dogs employed the hippocampal asymmetry ratio and electroencephalography (Czerwik 2018), and found an association between the presence of temporal lobe epileptiform discharges and a decrease in the unilateral hippocampal volume. And a ‘new’ technique of T2 relaxometry, which quantifies the degree of T2 hyperintensity, was employed as a group-wise comparison between epileptic and non-epileptic dogs. Although their results did not reach statistical significance, they found higher hippocampal T2 values in the epileptic group than in the control (Lorincz 2017).

## **7. Newer developments on the use of MRI in epilepsy**

### **7.1. Atlas-based semi-automated volumetry**

More accurate and less labour-intensive volumetry can be achieved through the use of automated volumetry using canine brain atlases and template-based segmentation (Milne 2016, Milne 2018). Atlas-based volumetry has been coupled with EEG to correlate areas of atrophy and epileptiform discharges, not just in the hippocampus but also the temporal cortex (Drobot 2025) and cingulate gyrus (Banasik 2024).

## 7.2. Voxel-based analysis

Voxel-based morphometry (VBM) is a computational technique for estimating differential brain volume in subject groups, by analysing voxel by voxel differences in brain structure using automated software and statistical parametric mapping (SPM) techniques. This technique permits identification and characterisation of structural small-scale brain differences among populations, permitting detection of changes in grey matter before overt cortical atrophy is present. A pilot study in 2018 used this technique in a small number of epileptic and non-epileptic dogs, and found that epileptic dogs had a statistically significant reduction in grey matter volume in the olfactory bulb, parietal and temporal cortex, hippocampus and cingulate gyrus (Frank 2018). Similar work in familial spontaneous epileptic cats found 20% of affected cats showed significant decreases in the hippocampal and/or amygdaloid regions (Hamamoto 2018).

## 7.3. Diffusion-weighted imaging

Diffusion-weighted imaging measures the random movement of free water molecules, based on Brownian motion. In the brain, this also serves as a marker for perfusion. This technique is used in humans with temporal lobe epilepsy, where a reduction in diffusion in the temporal lobe is described during ictus and postictally, thought to represent cytotoxic or intramyelinic oedema. Increased diffusion is noted in cases of hippocampal sclerosis. A feasibility study in dogs compared diffusion-weighted imaging scans of dogs with idiopathic epilepsy and healthy controls. The authors found dogs with idiopathic epilepsy had significantly increased ADC values in the amygdala within the piriform lobe. They postulated that this may be due to loss of structural organisation and an expansion of the extracellular space. (Hartmann 2017) The technique has also been applied to cats with familial spontaneous epilepsy, where interictal hypoperfusion was noted in the hippocampus, postictal hypodiffusion and hyperperfusion in the hippocampus and amygdala. (Hamamoto 2017)

## 7.4. Diffusion tensor imaging

Diffusion tensor imaging takes diffusion-weighted imaging data in multiple directions, and from that estimates the preferred direction of motion of water molecules to provide a vector, the diffusion tensor. Within the brain, water has faster diffusivity parallel to the direction of axons; thus diffusion tensor imaging provides an estimation of the course of white matter tracts. In humans, diffusion tensor imaging is used to detect microstructure biomarkers of epilepsy, evaluating the white matter connections between areas of cortical and subcortical tissue, which act as an epileptogenic network. In a case-control study of 26 dogs with idiopathic epilepsy and 24 healthy controls, the authors observed subtle changes in white matter DTI limited to cingulate white matter (Beckmann 2023).

## 7.5. Magnetic resonance spectroscopy

Within a defined voxel or voxels, MRS measures the signal from protons other than those found in water. It provides a spectral 'signature' which indicates the concentration of various metabolites in the brain. In epileptic dogs, this technique has been employed to investigate

metabolites in the thalamus in dogs with and without antiepileptic drug treatment, compared to unaffected controls (Mauri 2022). The authors found a significant reduction in NAA/Cr, considered a sign of neuronal loss, in idiopathic epileptic dogs under anti epileptic drug treatment, compared to epileptic dogs without treatment and controls. They also found a significant reduction in Glx/Cr in the same group, contrary to expectations. Degreased Glx is also considered an early biomarker for neuronal degradation.

## 8. Summary

Magnetic resonance imaging is an important test in epileptic patients to distinguish between structural and idiopathic forms of epilepsy. Due to the not insignificant cost of MRI, it is important to select appropriate patients for this test. The best interpretation of MRI requires high-resolution scans and experience in interpretation. Proposed epilepsy MRI protocols suggested by the IVETF centre around optimising images for the hippocampus.

Structural epilepsy has a prevalence estimate of between 25 to 45%. Brain tumours are the most common cause, with meningioma and glioma most often recognised. Congenital malformations are more likely in younger dogs, but subtle malformations of cortical development may not be detectable with standard MRI techniques.

Many recent publications of newer techniques for imaging canine epilepsy are directed toward identifying the 'epileptogenic zone', or a functional area of cerebral cortex responsible for initiating and propagating seizures, that cannot be defined by standard anatomical imaging. Such an epileptogenic zone offers a surgical target for patients with refractory epilepsy. Hippocampal sclerosis represents one such potential epileptogenic zone, and areas of cortical atrophy are also being more frequently recognised as epileptogenic zones. Other advanced techniques are in the infancy of application in pets, but look at features such as white matter abnormalities in epileptogenic networks, and brain metabolites in dogs treated with anti epileptic drugs. Imaging in epilepsy is a rapidly expanding field of research, and is aiding our understanding of the pathophysiology of canine 'idiopathic' epilepsy.

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