Need a Fix for that Fit?
Feline Seizures

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Feline seizures offer unique challenges compared to dogs. Both dogs and cats can have generalized or partial seizures. However, cats commonly present with rather bizarre partial seizures. Complex partial seizures or psychomotor seizures can manifest with the cat acting aggressive for several minutes at a time, and may frighten their caretakers. Symptomatic epilepsy or secondary epilepsy is more common in cats and accounts for almost half of all seizure disorders in cats.

Meningiomas are the most common brain tumor in cats, followed by lymphosarcoma. Cryptococcosis, FIP, and toxoplasmosis are also potential causes of feline seizures. Hypoglycemia should be considered in cats receiving insulin therapy. Idiopathic epilepsy represents 21% to 59% of feline seizure disorders. Intercrinal neurological examination is normal in cats with idiopathic epilepsy, whereas cats with secondary epilepsy can have a normal or abnormal examination. Unilateral deficits identified on examination suggest a structural or vascular cause.

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The increased incidence of secondary epilepsy in cats warrants further investigation for the cause, even when the neurological examination is normal and signalment is consistent with idiopathic epilepsy. A thorough evaluation for the underlying cause for seizures should be pursued to optimize anticonvulsant therapy. CBC, chemistry, urinalysis, thoracic radiographs and abdominal ultrasound should be done to rule out extracranial causes for seizures, while MRI and CSF should be done to rule out intracranial causes. Results of diagnostic testing are normal in idiopathic epileptics. Cats with idiopathic epilepsy typically first seizure between the ages of one and five. Generalized tonic clonic seizures are typically seen with idiopathic epilepsy, although focal seizures are also possible. Autosomal recessive inheritance is most common in dogs, but has not been established in cats. Anticonvulsant therapy is typically started when seizure frequency increases, particularly when two or more seizures occur within six months. A baseline period is helpful to determine the natural seizure frequency and can help assess response to anticonvulsant therapy. However this baseline period should not be too long, as earlier antiepileptic treatment may lead to better seizure control. Patients with cluster seizures, status epilepticus or secondary epilepsy should have anticonvulsant therapy started without delay. Patients with an underlying etiology other than idiopathic epilepsy may require medication in addition to anticonvulsant therapy. Anticonvulsant therapy is targeted to increase quality of life, decrease the frequency and severity of seizures with as few adverse effects as possible.

Anticonvulsant therapy for cats is often frustrating when phenobarbital is unsuccessful. The use of potassium bromide is no longer recommended in cats due to the potential for asthma secondary to bromide therapy. Signs may resolve once bromide therapy is discontinued. The longer half-life of diazepam in cats makes them a possible candidate for long term anticonvulsant therapy. However, the use of oral diazepam is limited/not recommended due to the potential risk of severe and potentially fatal hepatopathy in cats. Extensive studies regarding safety and efficacy of newer anticonvulsants in cats are lacking. Gabapentin 5-10 mg/kg PO BID to TID may be used, however, it has not been highly efficacious for seizures in small animals. Newer anticonvulsants such as levetiracetam 20 mg/kg PO TID, zonisamide 5-10 mg/kg PO SID and pregabalin 1-2 mg/kg PO BID in cats appear promising. Due to the limited number of studies and anecdotable experience (lower incidence of seizures in cats compared to dogs) care should be used when prescribing these medications. Starting at the lower end of the dose range may be of benefit. Serial monitoring of the CBC and serum chemistry is recommended when cats are receiving anticonvulsant medication. Therapeutic levels for levetiracetam, zonisamide and pregabalin are relatively unknown for cats and dogs at this time.

**Phenobarbital**

Phenobarbital is typically dosed at about 2.5 mg/kg PO BID. Most cats are started at 8.1 mg PO BID, while small cats are started at four mg PO BID. Phenobarbital increases the seizure threshold and decreases the spread to surrounding neurons. It enhances the inhibitory postsynaptic effects of GABA, inhibits glutamate activity and decreases calcium flux across neuronal membranes. Peak levels are achieved...
4-6 hours post oral administration and its T ½ is 34-50 hours in cats. Phenobarbital metabolism increases with chronic therapy in dogs, but less commonly in cats. Adverse effects include sedation, ataxia, polyphagia, PU/ PD and liver disease.

**Levetiracetam**
Levetiracetam binds synaptic vesicular protein SV2A. Levetiracetam may prevent hypersynchronization of burst firing and seizure propagation. The T ½ is four hours in dogs and three hours in cats. Peak plasma concentration occurs two hours post administration in most cats with approximately 100% bioavailability. Levetiracetam is started at 20 mg/kg PO TID. Adverse effects appear minimal, with sedation, ataxia and decreased appetite occurring uncommonly. Food does not affect absorption of levetiracetam. Most of the drug is excreted unchanged through the kidneys. The cytochrome P 450 system does not appear to be involved. However, concurrent phenobarbital administration has been shown to decrease levetiracetam T ½ and decreases levetiracetam blood levels in dogs. Thus patients on both phenobarbital and levetiracetam concurrently may require higher dosages of levetiracetam.

**Zonisamide**
Zonisamide is a sulfonamide-based anticonvulsant drug. Zonisamide blocks voltage-dependent sodium channels and T-type calcium channels. Its T ½ is about 15 hours in the dog and 35 hours in cats. Zonisamide is metabolized by hepatic microsomal enzymes and is also renally excreted. Its T ½ is reduced with concurrent phenobarbital administration. Zonisamide is administered 5-10 mg/kg PO BID in dogs, with the higher dose range for dogs also receiving phenobarbital. Cats are given 5-10 mg/g PO q 24 hours, due to the longer T ½. Zonisamide is typically well tolerated, with anorexia, sedation and ataxia being the most common adverse effects. Adverse effects are much more common with increased dosing. About half of cats given 20 mg/kg had adverse effects such as anorexia, vomiting, diarrhea, sedation and ataxia.

**Gabapentin**
Gabapentin binds voltage-gated calcium channels and decreases intracellular calcium influx. It is excreted unchanged by the kidneys with about 30-40% hepatic metabolism in the dog. The T ½ is 3-4 hours in dogs and about three hours in cats. Cats are given 5-10 mg/kg PO BID to TID. Sedation and ataxia are the most common adverse effects.

**Pregabalin**
Pregabalin is a GABA analog structurally similar to gabapentin. Pregabalin has a higher affinity for the α 2 δ subunit of neuronal voltage-gated calcium channels than does gabapentin. The T ½ of pregabalin is 7 hours in dogs and 10.4 hours in cats. Pregabalin is administered 2 – 4 mg/kg PO BID to TID in dogs and 1-2 mg/kg PO BID in cats. Adverse effects include sedation and ataxia and are not uncommon in dogs. Thus, starting dose at a lower dose and titrating the dose upward is recommended.

**Conclusion**
A thorough evaluation is essential for the seizing cat. Therapy should be directed at the underlying cause of seizures, with anticonvulsant therapy to control seizures and optimize quality of life. Phenobarbital has been the mainstay of anticonvulsant therapy in cats. However, when phenobarbital therapy is unsuccessful or if adverse effects occur the use of newer anticonvulsants can be helpful. Serial monitoring of the CBC and serum chemistry is recommended when cats are receiving anticonvulsant medication.

**Suggested reading:**