Feline Epilepsy

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KEYWORDS

• Feline seizures • Epilepsy • Phenobarbital • Levetiracetam

KEY POINTS

- Seizure disorders can be classified as idiopathic epilepsy, structural epilepsy, or reactive seizures.
- Phenobarbital is commonly the drug of choice.
- Levetiracetam may be more effective for cats with audiogenic seizures.
- Response to treatment is typically favorable, regardless of underlying diagnosis.

INTRODUCTION

Seizures are a common reason cats are presented to a veterinary neurologist. Despite the high prevalence of seizures in cats, debate exists on the correct terminology for seizure classification. Historically, veterinary seizure classification has been based on the human terminology published by the International League Against Epilepsy[1–3]; however, this has been problematic, especially for cats, because etiology and diagnostic investigation differ between human and cat species. The International Veterinary Epilepsy Task Force (IVETF) was formed in 2014 to advance a veterinary seizure classification system, based on the International League Against Epilepsy classification system, with modifications specifically designed for veterinary species. Differences between the International League Against Epilepsy and IVETF classification systems can be found in **Table 1**.

DESCRIPTION OF SEIZURES

The neuroanatomic lesion localization for any animal with seizures is the prosencephalon (the forebrain). Seizures are caused by hypersynchronous neuronal activity that results from an imbalance of excitation and inhibition within the neural network.[4] Animals may have 1 seizure or multiple seizures. After multiple seizures are confirmed, the disorder may be termed epilepsy if a metabolic or toxic cause is not identified. Epilepsy is a neurologic disorder that is defined by recurrent seizures.[5] There are 3

Disclosure Statement: The author has nothing to disclose.
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Vet Clin Small Anim 48 (2018) 31–43
http://dx.doi.org/10.1016/j.cvsm.2017.08.011 **vetsmall.theclinics.com**

Table 1
Comparison of the International League against Epilepsy (ILAE) and International Veterinary Epilepsy Task Force (IVETF) seizure classification systems

ILAE (2011) Classification	IVETF Classification	Definition
Genetic epilepsy	Idiopathic epilepsy	An intracranial disorder that has a confirmed genetic predisposition in the breed or animal.
Unknown epilepsy	Idiopathic epilepsy	No known genetic predisposition, but no identifiable cause on diagnostic testing (MRI, cerebrospinal fluid, laboratory).
Structural epilepsy	Structural epilepsy	An identifiable cause on diagnostic testing. for example, brain tumor, or encephalitis.
Metabolic seizures	Reactive seizures	Not epilepsy, but is a seizure disorder, for example, hypoglycemia.

phases to a seizure: (1) preictal phase, (2) ictal phase, and (3) postictal phase. The preictal phase is the time before the ictus (ictal phase) in which cats may display behavior or attitudes that owners can recognize as a preamble to a seizure. This activity may include hiding or seeking behavior, nausea, vomiting, or aggression. The preictal phase may last for seconds or hours. The ictus, or "seizure," typically involves both somatic and autonomic systems. Description of the different seizure semiology is discussed elsewhere in this article. Resolution of the ictus leads to the postictal phase, in which neuronal "resetting" occurs. Common clinical signs during the postictal phase may include hiding, blindness, or ataxia.

Seizures may be described as focal, complex focal, or generalized. These descriptions apply to the ictal phase only. Generalized seizures involve bilateral body movements with impaired consciousness. Agreement about the presence or absence of awareness may be difficult, even between veterinarians; therefore, caution should be taken not to overemphasize the state of awareness by practitioners when interrogating owners.[6] Focal seizures may manifest as abnormal movement of 1 part of the body, with or without altered mentation. Complex focal seizures include altered consciousness and are often described as abnormal running behavior, acute changes in mentation with abnormal facial movements, and ptyalism.[7] Complex focal seizures are common in cats. Focal seizures may rapidly generalize therefore a focused, detailed history should be taken to obtain as complete of a description as possible of the seizure event.

Reflex seizures are defined as seizures that occur immediately after a specific, identifiable stimulus.[8] Such stimuli may include visual (eg, photic), auditory (eg, music), or tactile (eg, bathing) stimuli. A reflex seizure may manifest as a generalized or focal seizure; however, they are most often associated with focal seizures.[9,10] Audiogenic reflex seizures have been described in a group of cats in the United Kingdom in which the trigger was a specific, often high-pitched sound.[11] Reflex seizures assume the same lesion localization and differential list as any epileptic seizure phenotype.

SEIZURE ETIOLOGY

The IVETF broadly classifies canine and feline epileptic syndromes by etiology as (1) idiopathic epilepsy (IE) and (2) structural epilepsy (SE).[10] Seizure secondary to metabolic disease or toxin exposure are classified as reactive seizures, but are not considered epileptic events by the IVETF.

Idiopathic Epilepsy

IE (also known as epilepsy of unknown origin), occurs in approximately 30% to 60% of cats.[2,12–16] The diagnosis of IE is achieved through complete metabolic screening, acquisition of a normal brain MRI and cerebrospinal fluid (CSF) analysis. A clinical diagnosis of IE is made in absence of diagnosable etiology in a cat with a history of 2 or more seizures and a normal interictal examination. One study found significantly more cats with IE displayed seizure activity during rest or sleep compared with cats with SE.[13] Spontaneous genetic epilepsy has rarely been documented in cats[17]; however, documentation of inheritance is seldom pursued in clinical practice; therefore, the prevalence may be higher than published data suggest. Age at diagnosis of IE varies widely in cats; therefore, cats with seizure onset greater than 6 years of age should not automatically be given a poor prognosis.[2] Cats diagnosed with IE are typically younger than cats diagnosed with SE.[2,12–14] IE may also be diagnosed in very young cats. IE accounted for approximately 26% of the cats diagnosed with seizures at less than 1 year of age.[18]

Structural Epilepsy

SE is defined by the IVETF as "epileptic seizures which are provoked by intracranial/cerebral pathology."[10] SE is the most common etiologic classification in cats of all ages, and may be caused by neoplastic, inflammatory, infectious, traumatic, vascular, or degenerative causes.[2,13,14,18,19] The diagnosis of SE requires the identification of an intracranial disease in an area of the brain likely to promote seizures. To accomplish this, MRI and CSF analysis may be used. MRI is superior to computed tomography for optimal visualization of intracranial structures.

Neoplasia

Neoplasia and meningoencephalitis (infectious or noninfectious) are the most common causes of SE in adult cats.[2,13,14] Cats with intracranial neoplasia are most frequently diagnosed with meningioma or lymphoma; however, not all cats with intracranial neoplasia develop seizures.[20,21] Tomek and colleagues[21] reported the incidence of seizures was approximately 33% for cats with intracranial neoplasia and the most common anatomic location of the neoplasm was the parietal lobe in these cats. Other types of neoplasia that have been associated with the development of seizures in cats include glial cell neoplasia (astrocytoma, oligodendroglioma), olfactory neuroblastoma, pituitary macroadenoma, and ependymoma.[2,21–23] Treatment options may include surgical treatment, medical treatment, or radiotherapy and are outlined in other resources.

Infectious/noninfectious

The most commonly reported infectious etiologies of meningoencephalitis include feline infectious peritonitis, *Toxoplasma gondii*, and *Cryptococcus* spp. Infection; however, other fungal and bacterial causes may occur.[2,14,24–26] The incidence of seizures is low for cats with feline infectious peritonitis; therefore, the presence of seizures may indicate extensive infection of the prosencephalon and a poorer prognosis.[27] Confirmation of feline infectious peritonitis should be obtained through histopathologic evaluation at necropsy or through brain biopsy. Noninfectious meningoencephalitis, also termed meningoencephalitis of unknown etiology, may be immune-mediated or secondary to a yet unidentified infectious etiology.[2,14,15,24] A diagnosis of meningoencephalitis of unknown etiology is obtained through identification of intracranial inflammation without concurrent evidence of infection. CSF analysis may be more sensitive than MRI for the detection of meningoencephalitis in cats; however, it is a

nonspecific test and, therefore, it is not recommended to perform CSF analysis without MRI.[25] Treatment for infectious meningoencephalitis is directed at the infectious etiology, often with concurrent antiinflammatory medication to decrease secondary central nervous system inflammation. Treatment for meningoencephalitis of unknown etiology involves immunosuppression and clinical response is variable. The long-term prognosis depends the clinical response of the cat to treatment.

Traumatic brain injury

Posttraumatic seizures are classified as early if they occur within 7 days of traumatic brain injury or late if they occur after 7 days. Cats sustain crush injuries most commonly; therefore, seizures may result from primary or secondary traumatic brain injury. The incidence of posttraumatic seizures in cats is unknown. Grohmann et al[28] reported no cases of posttraumatic seizures in 52 cats evaluated a minimum of 2 years after mild to moderate traumatic brain injury. Qahwash and colleagues[18] reported 2 of 7 cats (29%) less than 12 months of age at time of first seizure with posttraumatic seizures. Both cats had an abnormal neurologic examination at presentation and were alive at follow-up 13 and 15 months after injury. Hyperglycemia may occur secondary to moderate head trauma in cats; however, no association with prognosis has been identified to date.[29] A diagnosis is made based on historical or physical evidence of trauma paired with advanced brain imaging (MRI or computed tomography scan).

Cerebrovascular disease

Cerebrovascular disease is an uncommon cause of seizures in cats and accounts for 10% to 20% of cats with epilepsy.[14,19] Cerebrovascular disease may be secondary to infarct or hemorrhage. In 1 study, cerebrovascular infarcts were diagnosed in 75% of cats with vascular induced cerebral signs and seizures were reported in 42% of these cats.[30]

Clinical signs reflect the area of the brain affected by poor perfusion. Perfusion to the feline brain differs from the canine brain, which may lead to a different distribution of clinical signs in cats when compared with dogs.[30] Alterations in cerebrovascular perfusion commonly result in acute onset clinical signs. Typically, a cerebrovascular injury results in asymmetric findings on neurologic assessment, however if global ischemia has occurred (eg, anesthetic accident) neurologic examination findings may be symmetric. An underlying etiology has been reported for the majority of cats with cerebrovascular disease; therefore, an investigation into the systemic health of the cat should be pursued after a diagnosis of feline cerebrovascular disease.[30]

Hippocampal sclerosis

Mesial temporal lobe epilepsy with hippocampal sclerosis is a distinct syndrome in human epileptics with common orofacial seizure presentation.[31,32] Hippocampal sclerosis has also been described in cats with a similar seizure semiology.[12,13,33–35] Debate is ongoing in the veterinary literature if this diagnosis is a cause of, or secondary to, seizures in cats. In human epileptics, there are 2 forms of hippocampal sclerosis described. Primary hippocampal sclerosis seems to coincide with seizure onset, thus supporting the hypothesis that it is the underlying seizure etiology. Secondary hippocampal sclerosis occurs after ongoing, long-term seizures, supporting the hypothesis that hippocampal sclerosis occurs as a result of chronic seizures. Secondary hippocampal sclerosis was suggested by the authors of a case report in which 2 cats had a normal brain MRI at seizure onset, and were later diagnosed with hippocampal sclerosis on repeat brain MRI (2 cats) and necropsy (1 cat).[34] Secondary hippocampal sclerosis was considered more likely by others because it is more

commonly diagnosed on necropsy in cats with status epilepticus or chronic seizure history.[35] Hippocampal sclerosis may be idiopathic or associated with concurrent intracranial pathology. The majority of cats in 1 report diagnosed with hippocampal sclerosis had concurrent intracranial pathology, including infectious meningoencephalitis, inflammatory meningoencephalitis, vascular disease, or neoplasia.[35] Abnormalities in the temporal lobe on MRI have been described primarily on the fluid-attenuated inversion recovery, T2-weighted and T1-weighted postcontrast sequences for humans diagnosed with mesial temporal lobe epilepsy with hippocampal sclerosis and in cats diagnosed with hippocampal sclerosis. Classen and colleagues[33] reported that hippocampal abnormalities on MRI did not differ significantly among cats with and without seizures. However, when cats with only orofacial seizures, cluster seizures, or a history of status epilepticus were segregated for analysis a significant difference was found in MRI appearance of the hippocampus. Cats with orofacial seizures often have cluster seizures or status epilepticus; therefore, the semiology may be less important than the severity of the seizures to generate MRI-detectable hippocampal pathology; however, further research is needed to evaluate this hypothesis.

Identification of intracranial pathology through evaluation of a brain MRI or CSF analysis may lead to a diagnosis of SE; however, one must exercise caution in this approach. Ictal or interictal electroencephalography is rarely performed in cats and, therefore, the origin of the epileptic focus cannot be documented in most cases. Without identification of the epileptic focus, one is making an assumption that the intracranial pathology identified is associated with the seizure disorder.

Degenerative/Congenital

Reported congenital malformations suspected to cause seizures in cats include hydrocephalus,[18,36,37] porencephaly,[18] occipital arachnoid diverticula,[18] neuronal heterotopia,[38] and a malformation complex in Toyger cats, including commissural malformations, ventriculomegaly, and interhemispheric cysts.[39] Treatment may include surgical or medical management. Surgical correction with a ventriculoperitoneal shunt may improve, or in rare situations eliminate, neurologic abnormalities in young cats with hydrocephalus.[37] Storage disorders have also been reported to cause seizures in cats.[40] A storage disorder should be considered in a young cat with multifocal neurologic abnormalities in which metabolic testing, MRI, and CSF analysis are unremarkable.

Reactive Seizures

Reactive seizures are less common than IE or SE in adult and young cats.[2,13,18,24] Reported causes of reactive seizures in cats include hepatic encephalopathy, hepatic lipidosis, hyperthyroidism, polycythemia, renal disease, hyperosmolality, hypoglycemia, and toxins.[2,13,18,24] Serum bile acids have a high sensitivity and specificity for supporting a diagnosis of portosystemic shunts in cats and should be considered in any young cat presenting with seizures.[41] Additional metabolic testing, including complete blood count, serum biochemistry analysis, and urinalysis, aids in the diagnosis of reactive seizures. A historical exposure to toxins should increase the suspicion of reactive seizures.

DIAGNOSTIC APPROACH

Pursuit of diagnostic testing is recommended for cats presenting with a history of 2 or more seizures within a short time interval. If following the recent IVETF recommendations for dogs, pursuit of testing should be undertaken if 2 or more seizures occur within a 6-month period.[42] Initial blood sampling for a complete blood count, serum

biochemistry, and urinalysis may yield a metabolic abnormality, rendering a diagnosis of reactive seizures. If no significant biochemical or hematological abnormalities are detected, advanced imaging of the brain using MRI may be recommended. MRI is the recommended diagnostic imaging tool for the diagnosis of seizures.[43] If no structural abnormalities are identified, removal of CSF via cisternal puncture may be recommended. General anesthesia is recommended for MRI and CSF centesis to minimize risk and allow for immobility during the procedures. Electroencephalography may be used to detect epileptiform patterns in sedated or anesthetized animals, aiding in the identification of interictal activity suggestive of seizures.[44] Routine monitoring with electroencephalography is rarely reported in cats owing to technical difficulties obtaining diagnostic recordings.

The results of the diagnostic testing will lead the clinician to an etiologic classification and confirmed or presumptive diagnosis for the cause of the seizure disorder.

ANTIEPILEPTIC DRUGS

The IVETF published guidelines for initiation of therapy for dogs, which could be extrapolated to cats. Among other parameters, the IVETF recommends starting antiepileptic drugs (AED) when any dog has 2 or more seizures in 6 months and/or any 1 seizure lasting longer than 5 minutes.[42] A secondary reason to begin therapy is the presence of severe (eg, aggression) or long-lasting (eg, longer than 24 hours) clinical signs during the postictal phase. The author also encourages initiation of AED if the cat is at reasonable risk of additional seizures, even if the frequency recommendations are not met, for example, a cat with a brain tumor in the parietal lobe, or a young cat with severe hydrocephalus. Other authors have advocated for more aggressive initiation of treatment and suggested AED treatment should be initiated if 2 or more seizures occur in 6 weeks.[12] A delay in AED initiation may result in a longer time to seizure control, or seizure freedom.[45] Choosing the best AED should be based on the seizure semiology, the cat's health status, and owner considerations (frequency of administration, cost, and liquid vs tablet formulation). A summary of the mechanism of action, dosage, and therapeutic interval for the following AED is in **Table 2**.

Phenobarbital

Phenobarbital is the most commonly used AED in cats.[1,45–47] Rare side effects and no adverse biochemical or hematological events have been reported after use in cats.[48] Side effects may include sedation, ataxia, and weakness, especially during the first 2 weeks of treatment. In a single case report, pseudolymphoma was reported after oral administration of phenobarbital in a cat.[49] Phenobarbital is rapidly absorbed after oral administration, reaching maximal concentration within 1.5 hours. The elimination half-life is similar to what has been reported for dogs and, therefore, a steady state is expected in most cats 10 to 14 days after initiation of treatment.[48] Phenobarbital is lipid soluble, rapidly crosses the blood–brain barrier, and has a high volume of distribution in experimental models. Standard dosing for cats is 2 to 5 mg/kg by mouth every 12 hours (reported ranges, 1.8–10.0 mg/kg/d)[1,12,45,48,50] The therapeutic interval has not been established in cats; however, the dog reference interval of 15 to 45 μg/mL may be used or a more narrow therapeutic window between 23 and 30 μg/mL has been recommended.[1,12,47,50] In 1 study, seizure control was achieved in 93% of cats with a serum phenobarbital concentration between 15 and 45 μg/mL, regardless of underlying etiology.[1] As an alternative to oral administration, topical administration with transdermal phenobarbital has been evaluated in healthy cats using 2 different carrier molecules: pluronic lecithin organogel and Lipoderm Activemax.[51,52] Serum

Table 2
Antiepileptic drugs prescribed for use in cats with seizures

Antiepileptic Drug	Mechanism of Action	Dosage Range	Therapeutic Interval
Phenobarbital	Inhibition of acetylcholine release, norepinephrine and glutamate. Ca^{2+} channel inhibitor, GABA mimetic.	2–5 mg/kg PO q12h[1,12,50] 9 mg/kg transdermal q12h[51]	15–45 μg/mL[c]
Levetiracetam – standard release	Selectively binds SV2A protein thus modulating the release of neurotransmitters.	20 mg/kg PO q8h[53,54]	5–45 μg/mL[a]
Levetiracetam – extended release	Same as above.	500 mg/cat PO q12–24h (in press)	5–45 μg/mL[a]
Zonisamide	Blocks Na and Ca^{2+} channels and may reduce the spread of seizures.	5–10 mg/kg PO q12h[64]	10–40 mg/L[a]
Imepitoin	GABA mimetic.	0–80 mg/kg/d PO[b]	Unknown
Pregablin	Binds voltage-gated Ca channels.	1–2 mg/kg q12h[71]	2.8–8.2 μg/mL[a]
Gabapentin	Same as pregabalin.	10 mg/kg q12h[72]	Unknown

Dosage is based on available literature and clinical studies when applicable.
Abbreviation: PO, by mouth.
[a] Human reference interval. Feline reference interval not established.
[b] Use caution if prescribing this dose because it has not been verified in clinical trials in cats.
[c] Canine reference interval. Feline reference interval not established.

concentrations within the reference interval were not obtained in any cats at standard doses of 3 mg/kg every 12 hours. Dose escalation to 9 mg/kg every 12 hours resulted in serum concentrations of greater than 15 μg/mL for most cats, using both carrier molecules. However, application was easier for owners using Lipoderm Activemax ; therefore, the phenobarbital in Lipoderm Activemax was recommended.[51,52] A clinical trial comparing serum phenobarbital concentrations in epileptic cats on oral therapy and transdermal therapy is currently underway. Midpoint analysis suggests serum concentrations achieved with transdermal phenobarbital are within 20% of serum concentrations achieved with oral phenobarbital, indicating transdermal phenobarbital may be a viable alternative to oral administration for cats.

Levetiracetam

Levetiracetam is an AED that has a novel mechanism of action compared with other common AED.[53,54] The therapeutic interval for levetiracetam is unknown and has been extrapolated from humans for use in dogs and cats (5–45 μg/mL).[53,55,56] Reported side effects in cats receiving levetiracetam include mild transient hypersalivation, inappetance, and mild lethargy.[53,54] There are currently 2 formulations available: (1) standard release levetiracetam and (2) extended-release levetiracetam (XRL). After pharmacokinetic analysis, a dosage of 20 mg/kg by mouth every 8 hours was recommended for cats.[53] A greater than 50% reduction in seizures was noted in 7 of 10 cats after levetiracetam was added to phenobarbital for seizure management.[54] XRL is currently available in 500 mg and 750 mg size tablets. Crushing, splitting, or chewing the tablets is not recommended; therefore, its usefulness in cats has been limited. Single dose pharmacokinetics after 500 mg XRL by mouth were evaluated in healthy cats and a dosing interval of once to twice daily was suggested.[57] A 10-day multidose trial of 500 mg once daily XRL was completed in 9 healthy cats. Seven of 9 cats maintained serum levetiracetam concentrations above the minimum human therapeutic interval at trough samples and minimal side effects were noted (Barnes Heller HL, in progress). Use of XRL in cats allows a decreased frequency of oral administration, thereby improving the quality of life for the cat and client.

Levetiracetam resulted in marked improvement in seizure control, compared with phenobarbital, in cats with suspected audiogenic reflex seizures.[58] This finding is in agreement with a survey of human epileptologists, in which levetiracetam was identified as the first drug of choice for adults and adolescents with seizures involving myoclonus.[59] Further studies are needed to determine if levetiracetam retains greater effectiveness for specific seizure semiology in epileptic cats long term.

Tolerance of levetiracetam has been suggested in dogs; however, this difficulty has yet to be documented in cats.[60,61] The lack of documentation should not exclude the possibility of long-term tolerance (termed the “honeymoon effect” by some authors); rigorous seizure frequency monitoring should be maintained if a cat receives long-term levetiracetam. Administration of intermittent, or pulse, levetiracetam for several days after a seizure has been recommended in dogs to avoid development of long-term tolerance.[62] This approach has not been validated in cats and, therefore, should be used with caution.

Zonisamide

Zonisamide is a sulfa-derived AED developed first in cats and rats in the late 1970s in Japan.[63] The reported half-life of zonisamide in cats (68 hours) is longer than that reported for dogs, which is suspected to be due to the decreased hepatic glucuronide conjugation in cats.[64] Gastrointestinal upset (vomiting, diarrhea, nausea) was reported in one-half of the cats receiving long-term treatment with zonisamide at 20 mg/kg;

therefore, a lower dose may be required to limit these side effects in some cats.[64] Other side effects, including ataxia and sedation, were also noted. Three of 5 cats treated with a mean dose of 11.54 mg/kg/d were reported to have a greater than 50% reduction in seizures in 1 study.[65] Rare, mild side effects were reported and no hematological or biochemical changes were detected after 90 days of administration. Few reports are available discussing the usefulness of zonisamide in cats with naturally occurring epilepsy.[15,65] In 2015, the IVETF did not find sufficient data to recommend using zonisamide either as monotherapy or adjunctive AED therapy in dogs.[42] Given the lesser amount of information available in cats, clients should be counseled on the lack of clinical evidence supporting the use of zonisamide in cats for the treatment of epilepsy before initiation of this medication.

Imepitoin

Imepitoin is licensed in Europe and Australia for use in the management of IE in dogs, and is not approved for use in cats. It is not currently available in the United States. Imepitoin has been used at dosages up to 80 mg/kg/d in healthy cats.[66] Adverse gastrointestinal effects (reduced appetite, vomiting) were noted at the higher dosages; however, it is unclear in the published literature how many of the cats were affected with these side effects. No clinical trials have been published evaluating efficacy of this drug in epileptic cats. Caution should be exercised if prescribing this medication to cats owing to the lack of pharmacokinetic profiling, safety reporting, and clinical efficacy for seizure control.

Bromide

Potassium bromide is not recommended for use in cats owing to the high occurrence of adverse reactions.[67] Most commonly bromide results in coughing and/or dyspnea with resolution in most, but not all, cats after discontinuation of the drug. The respiratory complications are suspected to be secondary to airway hypersensitivity, which is best managed with discontinuation of bromide administration.[68]

Other

Gabapentin, pregablin, and topiramate have been evaluated in limited clinical trials in dogs. To date, no clinical trials have been published evaluating these drugs in cats. There are published dosages for each of these drugs; however, one must be aware of the limited clinical and pharmacokinetic data available when using these drugs in epileptic cats.

Drug Resistance and Recurrence

Drug resistant epilepsy is defined as "a failure of adequate trials of two appropriately chosen and used antiepileptic drug schedules (whether as monotherapies or in combination) to achieve sustained seizure freedom."[69] Limited data are available for drug resistance in cats; however, because a greater number of cats are diagnosed with IE, this issue may become more prevalent in this population. Current recommendations in humans encourage switching from one monotherapy to another monotherapy, rather than administration of adjunct AED.[59] For cats, poor response to the primary AED warrants trial of a second AED either as addon therapy or a second monotherapy. The pharmacokinetic profile of individual drugs, metabolic state of the cat, and economic situation of the client should be considered before initiation of AED for epileptic cats.

It is well-known that abrupt discontinuation of AED may result in withdrawal seizures and, therefore, this action should be avoided whenever possible. Caution should be exercised when considering reducing or tapering the AED after a period of seizure

freedom as well. Six of 8 cats had a recurrence of seizures after reduction of AEDs in 1 study.[45] Given the scarcity of literature addressing recurrent seizures after reduction of AED in cats, owners should be counseled about the risk and benefits of reducing or discontinuing AED before starting a drug tapering protocol.

PROGNOSIS

The prognosis for long-term survival or seizure control depends the underlying etiology, treatment pursued, and response to treatment. The overall long-term survival is good for many cats, regardless of the diagnosis.[70] Similar to dogs, cats with adult-onset seizures secondary to IE have a significantly longer survival compared with cats with adult-onset seizures secondary to SE.[2] In another study evaluating cats less than 12 months of age at seizure onset, cats diagnosed with SE had a slightly longer survival than those diagnosed with IE or reactive seizures.[18] Treatment delay could result in an increased seizure frequency; therefore, AED should be considered if a cat meets the recommendations for initiation of treatment. Given that cat seizures are typically well-controlled with AED, prompt treatment seems to be integral to improved long-term outcome.[12]

ACKNOWLEDGMENTS

The author thanks Dr Helena Rylander for her meticulous review of this article.

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