Although the prevalence of seizures in pediatric dogs and cats is unknown, the overall incidence in the pet population is reportedly 2% to 3%.\(^1,2\) Seizures in young dogs and cats are a diagnostic dilemma for practitioners. In young animals, seizures usually signal the onset or coexistence of significant central nervous system (CNS) disease. A seizure in puppies and kittens often requires prompt medical attention with special considerations for medical management. For the purpose of this discussion, immature or young animals are defined as those younger than 6 months of age. Categorically, the neonatal period is 0 to 2 weeks of age, the socialization period is 3 to 12 weeks of age, and the juvenile period is 12 weeks of age to adult (i.e., 3 to 6 months of age).\(^3\)

**PATHOPHYSIOLOGY**

An immature brain is more prone to seizures than is a mature brain because of multiple changes that occur during development. Epileptogenesis (i.e., generation of seizures) in an immature brain is influenced by inhibitory and excitatory systems, ionic microenvironment, and degree of myelination. Developing neurons appear to be less vulnerable to damage and loss after seizure activity. Dogs and cats younger than 1 year of age are more likely to have symptomatic epilepsy. Early recognition of potential causes of seizures in young dogs and cats is important for appropriate diagnostic considerations and timely therapeutic interventions.
ductance (GABA$_A$) or potassium conductance (GABA$_B$). Ultrastructural studies comparing immature with adult rat brains show that GABA terminals in immature brains are smaller and contain fewer synaptic vesicles. Likewise, there are fewer synapses and lower concentrations of GABA receptors. The rate of GABA formation and catabolism changes during maturation. Differences lie not in receptor composition but rather in maturational changes to the chloride ion gradient that govern the equilibrium potential for GABA$_A$ channels. Consequently, in the immature brain, GABA$_A$ responses result in depolarization with subsequent activation of sodium ion (Na$^+$) and calcium ion (Ca$^{2+}$) channels.

In contrast to the inhibitory system, the excitatory system is overdeveloped. Glutamate is the major excitatory neurotransmitter in the brain, and several subtypes for the glutamate receptor exist, including N-methyl-D-aspartate (NMDA), kainate, and $\alpha$-amino-3-hydroxy-5-methyl-4-isoxazolepropionate (AMPA). The hippocampus of the prenatal brain has an excess number of recurrent excitatory synapses and an overabundance of NMDA receptors. As the brain continues to develop, these excitatory synapses are modulated or “pruned” to adult levels. Expression of glutamate transporters that play a role in glutamate uptake is also developmentally regulated. Decreased expression of glutamate transporters and variation of subtypes can lead to increased seizure susceptibility and to a lower seizure threshold.

Differences in the ionic microenvironment that surrounds neurons and glial cells also contribute to epileptogenesis in the immature brain. The potassium concentration is increased in the extracellular fluid of immature brains. Glial function immaturity may allow the extracellular potassium concentration to increase, causing excitability. Thus the action potentials in immature neurons last longer because of altered potassium channel conductance, thereby causing a lower resting membrane potential and increased neuronal excitability.

Catecholamines, especially norepinephrine, play a role in the generalization of epileptic activity during kindling. Lower levels of norepinephrine have been associated with loss of kindling antagonisms. Kindling is defined as local, repeated, and initially subconvulsive stimulation of neurons that progresses to alter the excitability of other nearby neurons and to develop into a seizure focus. The threshold of kindling varies with age, and spontaneous seizures occur more readily in developing animals than in adults. The lower epinephrine level in the immature brain may be a factor responsible for facilitating kindling.

Incomplete myelination contributes to seizure expres-

Developmental changes in the brain may increase or decrease the window of susceptibility for seizures in young dogs and cats.

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Incomplete myelination contributes to seizure expres-
ever, there is increasing evidence that recurrent seizures affect neuronal development by mechanisms that alter synaptogenesis and neurogenesis.\(^6,28\)

The long-term effects of continued seizure activity are unknown. Neonatal seizures can increase the susceptibility of the developing brain to subsequent seizure-induced injury.\(^{29,30}\) Experimental studies of immature rats showed that various pathologic changes developed after 50 short seizures.\(^{31}\) Histopathologic studies of epileptic beagles have shown evidence of astrocytic swellings and ischemic changes in cerebral cortical neurons.\(^{32}\) Magnetic resonance imaging (MRI) of the brain has shown reduced myelination in children who have had neonatal convulsions.\(^{21,33}\) However, a recent analysis of humans with recurrent seizures concluded that the risk to developing individuals was low.\(^34\)

**CLASSIFICATION**

Classifying seizures is useful in identifying the type and determining an underlying cause. In humans, controversy exists over defining a solitary classification scheme for clinically describing seizures in neonates. The scheme used in adults established by the International League Against Epilepsy\(^35\) has been unreliable and difficult to apply to infants.\(^36\) Important differences exist in the clinical expression of seizures in adults and neonates.\(^37\) Adults more commonly present with partial seizures, whereas neonates have postural abnormalities. Neonatal seizures are often described as subtle and fragmentary, and some neonatal behaviors can mimic the phenomenology of true seizures.\(^38\) These seizure-like behaviors have been referred to as reflex or release phenomena and must be distinguished from true seizures. Neonatal seizures are currently classified using electroencephalography and simultaneous video monitoring. Neonatal seizures have been described as seizures with a close correlation to electroencephalogram (EEG) seizure discharges, seizures with an inconsistent or no relationship to EEG ictal discharges, infantile spasms, and EEG seizures without clinical seizures.\(^39\) This has led to classification of epileptic and nonepileptic neonatal seizures with further categorization according to clinical features (i.e., focal clonic, focal tonic, or myoclonic seizures; spasms).\(^40,41\)

A classification scheme for seizures according to their clinical appearance in domestic animals has not been thoroughly defined, and currently used schemes are extrapolated from the human literature.\(^42,43\) In veterinary medicine, seizure types are classified into two major categories: generalized and focal.\(^44\) A generalized seizure often reflects a widespread seizure focus, producing loss of consciousness, autonomic activity, and whole body movements with alternating tonic and clonic phases of movements. A focal seizure reflects the activity of a local seizure focus in an area producing motor activity. It has been believed that generalized motor seizures are the most common seizure type in dogs.\(^42,45,46\) This has recently been brought into question by Berendt and Gram\(^47\) who applied a human classification scheme for epilepsies to dogs. Results showed that focal seizures with and without generalization were most common and further emphasized reevaluation of currently used terminology for epilepsy in veterinary medicine. In puppies, generalized tonic seizures have been observed as early as 4 weeks of age.\(^48\) Immaturity of the brain and neurotransmission processes may prevent more accurate recognition of seizures in younger animals.

**CAUSE**

Recurrent seizures are more broadly defined as epilepsies. Podell et al\(^42\) adopted a nomenclature scheme from human epilepsies based on identifiable cause. Primary epileptic seizure (i.e., idiopathic) is the term used if an underlying cause cannot be identified. If seizures result from a structural lesion, they are defined as secondary epileptic seizures. The term reactive epileptic seizure is used when there is a reaction of the normal brain to transient systemic insult or physiologic stresses; these seizures are not considered recurrent. Epilepsies are also described as asymptomatic (i.e., primary, idiopathic) and symptomatic (i.e., secondary).\(^49\) This article uses the terminology of asymptomatic and symptomatic epilepsies.

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**Seizures in young dogs and cats often reflect a symptomatic or an acquired cause; however, idiopathic epilepsy is being recognized more frequently as a diagnostic differential in younger animals.**

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June 2005
### Table 1. Causes of Seizures in Young Dogs and Cats

<table>
<thead>
<tr>
<th>Disorder/Category</th>
<th>Affected Breeds/Specific Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Developmental Anomalies</strong></td>
<td></td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>Boston terrier, Chihuahua, English bulldog, Maltese, Lhasa apso, Pomeranian, toy poodle, Cairn terrier, pug, Pekingese, Siamese cat</td>
</tr>
<tr>
<td>Hydranencephaly, porencephaly</td>
<td>Secondary to infection</td>
</tr>
<tr>
<td>Lissencephaly</td>
<td>Lhasa apso, Irish setter, wire fox terrier, domestic shorthaired cat, Korat cat</td>
</tr>
<tr>
<td>Polymicrogyria</td>
<td>Poodle</td>
</tr>
<tr>
<td>Agenesis of corpus callosum</td>
<td>Domestic shorthaired cat, Labrador retriever</td>
</tr>
<tr>
<td>Dandy-Walker syndrome</td>
<td>Not breed specific in dogs and cats</td>
</tr>
<tr>
<td>Chiari malformation</td>
<td>Cavalier King Charles spaniel, other small-breed dogs</td>
</tr>
<tr>
<td>Intracranial arachnoid cyst</td>
<td>Not breed specific</td>
</tr>
<tr>
<td><strong>Degenerative</strong></td>
<td></td>
</tr>
<tr>
<td>Leukodystrophy</td>
<td>Dalmatian, Labrador retriever, Shetland sheepdog, Samoyed, silky terrier</td>
</tr>
<tr>
<td>Metachromatic leukodystrophy</td>
<td>Domestic shorthaired cat</td>
</tr>
<tr>
<td>Subacute necrotizing encephalomyelopathy (mitochondrial encephalopathy)</td>
<td>Australian cattle dog, Alaskan husky, Maltese</td>
</tr>
<tr>
<td>Spongiform encephalopathy</td>
<td>Saluki, Labrador retriever, silky terrier, Egyptian Mau cat</td>
</tr>
<tr>
<td>Glycogen storage disease</td>
<td>Type 1 (silky terrier dog, domestic shorthaired cat, toy breeds), type 2 (domestic shorthaired cat), type 4 (Norwegian forest cat)</td>
</tr>
<tr>
<td>Lysosomal storage disease</td>
<td>GME1 gangliosidosis (German shorthaired pointer, Portuguese water dog, beagle, Alaskan husky, Siamese cat [type 2], domestic shorthaired cat [types 1 and 2], Korat cat [type 2])</td>
</tr>
<tr>
<td></td>
<td>GME2 gangliosidosis (Siamese cat, domestic short-haired cat, Korat cat)</td>
</tr>
<tr>
<td></td>
<td>Fucosidosis (English springer spaniel)</td>
</tr>
<tr>
<td></td>
<td>Glycoproteinosis (Lafora’s disease; Basset hound, miniature poodle, beagle)</td>
</tr>
<tr>
<td>Ceroid lipofuscinosi (infantile, juvenile)</td>
<td>Chihuahua, Dalmatian, English setter, dachshund, saluki, Australian blue heeler, Australian cattle dog, Border collie, Siamese cat</td>
</tr>
<tr>
<td>Multisystemic neuronal degeneration</td>
<td>Cocker spaniel, Rhodesian ridgeback</td>
</tr>
<tr>
<td>Hereditary quadriplegia and amblyopia</td>
<td>Irish setter</td>
</tr>
<tr>
<td><strong>Metabolic</strong></td>
<td></td>
</tr>
<tr>
<td>Portosystemic shunting</td>
<td>Yorkshire terrier, Maltese, schnauzer, Irish wolfhound, Old English sheepdog</td>
</tr>
<tr>
<td>Hepatic microvascular dysplasia</td>
<td>Poodle, schnauzer, dachshund, Yorkshire terrier, shih tzu, cocker spaniel</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>Toy and miniature breeds</td>
</tr>
<tr>
<td>Hypoxemia</td>
<td>Neonatal asphyxia</td>
</tr>
<tr>
<td>Hypocalcemia</td>
<td>Primary hypoparathyroidism</td>
</tr>
</tbody>
</table>
Specific seizure types have been associated with specific disease processes in humans; however, this still needs further evaluation in veterinary medicine. Podell et al.\textsuperscript{42} found a higher probability of symptomatic epilepsy in dogs with a short interictal interval and focal seizures, supporting the belief that focal seizures are an indication of a structural cerebral lesion. Similar results were reported in later studies by Berendt and Gram\textsuperscript{47} as well as Patterson et al.\textsuperscript{50} In contrast, these results additionally documented that dogs considered idiopathic epileptics also exhibited focal seizures. A more recent study of vizslas with inherited idiopathic epilepsy showed that focal seizures of variable frequency were the predominant seizure type.\textsuperscript{50} Age is also a significant predictor of symptomatic epileptic seizures in young dogs (i.e., <1 year of age).\textsuperscript{42} In Table 1, the DAMNIT-V classification scheme is used to summarize disorders associated with or that cause seizures in immature animals.\textsuperscript{51–53}

### Table 1. Causes of Seizures in Young Dogs and Cats\textsuperscript{a} (continued)

<table>
<thead>
<tr>
<th>Disorder/Category</th>
<th>Affected Breeds/Specific Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nutritional</strong></td>
<td></td>
</tr>
<tr>
<td>Thiamine deficiency</td>
<td>Seizures in dogs fed diets mainly of meat and fish</td>
</tr>
<tr>
<td><strong>Inflammatory</strong></td>
<td></td>
</tr>
<tr>
<td>Viral</td>
<td>CDV, FIP, feline panleukopenia, FIV, nonsuppurative meningoencephalomyelitis in cats</td>
</tr>
<tr>
<td>Fungal</td>
<td>Cryptococcus spp infection</td>
</tr>
<tr>
<td>Bacterial</td>
<td>Infection with aerobes or anaerobes, abscessation</td>
</tr>
<tr>
<td>Protozoal</td>
<td>Toxoplasmosis, encephalitozoonosis</td>
</tr>
<tr>
<td>Parasitic</td>
<td>Cuterebra spp larval myiasis</td>
</tr>
<tr>
<td>Rickettsial</td>
<td>Ehrlichiosis, Rocky Mountain spotted fever</td>
</tr>
<tr>
<td>Noninfectious inflammatory</td>
<td>Eosinophilic meningoencephalitis, encephalitis (pug, Maltese, Yorkshire terrier), GME, periventricular encephalitis</td>
</tr>
<tr>
<td><strong>Idiopathic</strong></td>
<td></td>
</tr>
<tr>
<td>Epilepsy</td>
<td>Beagle, Belgian Tervuren, keeshond, British Alsatian, Labrador retriever, golden retriever, collie, dachshund, vizsla</td>
</tr>
<tr>
<td><strong>Traumatic</strong></td>
<td></td>
</tr>
<tr>
<td>Acute and chronic</td>
<td>Posttraumatic epilepsy</td>
</tr>
<tr>
<td><strong>Toxicosis</strong></td>
<td></td>
</tr>
<tr>
<td>Pesticides (organophosphates, carbamates, pyrethrins, metaldehyde), rodenticides (bromethalin, strychnine, rotenone, zinc phosphate, vacor), herbicides, heavy metals (lead, triethyltin, thallium), drugs, poisonous plants, mycotoxins (amanita mushrooms, penitrems A), antifreeze (ethylene glycol), disinfectants (hexachlorophene), methylxanthines, street drugs</td>
<td></td>
</tr>
</tbody>
</table>

\textsuperscript{a}Lists only breeds and diseases in which seizures have been clinically documented in young dogs and cats. CDV = canine distemper virus; GME = granulomatous meningoencephalomyelitis.

**Symptomatic Epilepsies**

**Degenerative/Anomalous**

Disorders related to neuronal migration and some forms of cranial malformations are apt to induce seizures. Brain malformations associated with seizure activity include hydrocephalus, Dandy-Walker syndrome, hydranencephaly, lissencephaly (Figure 1), Chiari-like malformation, intracranial intraarachnoid cyst, polymicrogyria, and agenesis of the corpus callosum.\textsuperscript{44,51}

Congenital or acquired hydrocephalus is a common diagnostic differential for seizures in young dogs and cats.\textsuperscript{44} Hydrocephalus was found to be the most common cause of secondary epileptic seizures in dogs younger
than 1 year of age. Internal (Figure 2) and external hydrocephalus refer to increased fluid accumulation within the ventricular and subarachnoid spaces, respectively. Noncommunicating (i.e., obstructive) hydrocephalus refers to increased cerebrospinal fluid (CSF) only within the ventricular system, and communicating refers to increased CSF within the ventricular system and subarachnoid space. Congenital hydrocephalus often represents a secondary manifestation of a developmental (e.g., Chiari type 1-like malformation) or an acquired (e.g., perinatal exposure to toxins or infectious disease) disorder. Congenital hydrocephalus is associated with fusion of the rostral colliculi, causing secondary mesencephalic aqueductal stenosis. Head conformation often involves a dome shape and an open bregmatic fontanelle. Clinical signs of hydrocephalus vary in severity and typically manifest with seizure activity and forebrain dysfunction. Forebrain signs include mentation changes such as disorientation, obtundation, and stupor as well as behavioral abnormalities that can consist of an inability to learn skills such as housebreaking. Hydrocephalus can be diagnosed using MRI or computed tomography (CT), ultrasonography, and electroencephalography.

Medical therapies can reduce the severity of clinical signs, presumably by altering CSF production. The goal of surgical management is shunting CSF from the ventricles to another space (e.g., atrium, abdominal cavity). Shunting procedures are the mainstay of therapy for hydrocephalus in human medicine and are now advocated in veterinary medicine.

Only a few inborn errors of metabolism (e.g., organic/mitochondrial encephalopathies) involving the cerebral cortical tissue can cause clinical signs of seizure in dogs. Neuronal metabolism is directly affected when the enzyme defect is located in a major metabolic pathway. Clinical descriptions for some of the organic/mitochondrial encephalopathies include episodic extensor rigidity, myoclonus, and epileptiform-like seizures. Lysosomal storage diseases cause seizures by interference of neuronal metabolism or accumulation of intracellular by-products. Seizure events that occur with some storage disorders usually manifest at the end stage of the disease process. Storage disorders for which seizure activity is a predominant clinical fea-
ture include ceroid lipofuscinosis, glycoproteinoses, and leukodystrophies.67

**Metabolic**

Hypoxemia in young animals is often suspected after severe respiratory and cardiovascular compromise. In addition, hypoxemia may increase the anesthetic risk in patients undergoing early spay and neuter procedures. Brain injury in newborn fetuses may be associated with hypotensive effects instead of hypoxia or acidosis.68 During the period of hypoxia–ischemia and reperfusion injury, various cytotoxic processes include cellular energy failure, excitotoxicity, free radical damage, and intracellular calcium accumulation.69 Hypercapnia may also be an important component of neonatal asphyxia. Fourteen-day-old neonatal dogs had increased seizure susceptibility during the recovery phase of experimentally induced hypercapnia (i.e., partial pressure of carbon dioxide values: 50 to 100 mm Hg).70

Hypoglycemia at glucose concentrations less than 40 mg/dl can precipitate neuroglycopenia. Neuroglycopenia is manifested by depression, hypothermia, weakness, seizures, and coma. Factors responsible for clinical signs of neuroglycopenia include rate of decrease, level of hypoglycemia, and duration of hypoglycemia.71 Glucose is the predominant energy substrate for the adult and neonatal brain. Although the receptor numbers for glucose transport protein are low in the immature brain, the transport protein for ketone bodies as well as lactate and pyruvate is high. Studies of newborn dogs have shown that during hypoglycemia, lactic acid is not only incorporated into the perinatal brain but also consumed to the extent that the metabolite can support up to 60% or more of total cerebral energy metabolism.72 Although the neonatal brain can readily metabolize ketone bodies, lack of body fat and prolonged time necessary to produce ketones prevent this mechanism from protecting neonates from acute hypoglycemia.73 Juvenile-onset hypoglycemia occurs because of immature hepatic enzyme systems, deficiency of glucagon, and deficiency of gluconeogenic substrates. Fatty liver syndrome causes hypoglycemia in toy breed puppies at 4 to 16 weeks of age.74 Persistent juvenile hypoglycemia is often related to a glycogen storage disorder.75 Extrinsic factors that cause hypoglycemia include stress, hypothermia, parasitism, and low birth weight. In addition, hypoglycemia combined with hypoxia–ischemia is more deleterious to the immature brain than either condition alone.76

Portosystemic shunts (PSSs) are common congenital defects causing hepatocerebral disease in dogs and cats.77,78 Common clinical signs of hepatocerebral disease include ataxia, circling, depression, behavior changes, and seizures. A variety of compounds have been implicated in the pathogenesis of hepatic encephalopathy, although this process is poorly understood. Postulated causes include elevated ammonia, altered ratios of neurotransmitters, and increased brain concentrations of benzodiazepine-like neurotransmitters.79 Recent studies found higher concentrations of glutamine, tryptophan, and quinolinol, a metabolite of tryptophan, in the CSF of dogs with PSS.80 Quinolinol is an agonist of the NMDA receptor, and the developing brain is more sensitive to NMDA activation.81 This may play a role in development of seizure activity in young dogs with PSSs. Pathologic lesions are characterized by protoplasmic astrocytic proliferation (as in Alzheimer type 2 reactions) and spongiform changes in the brains of dogs with PSSs.82 Treatment involves managing the hepatic dysfunction and encephalopathy. Seizures and neurologic sequelae following PSS attenuation have been well documented.83–85 Neurologic complications have been associated with all of the occlusion methods.86 Potential risk factors for neurologic complications include older dogs and dogs with single extrahepatic and portoazygous shunts.86 The pathophysiologic mechanisms of postligational seizures are poorly understood.87

**Inflammatory**

Seizures occur in about 13% of dogs with CNS inflammatory diseases.88 Cats with seizures were frequently

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**Inflammatory**

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diagnosed with inflammatory disease (47% [14 of 30] of cats) of suspected viral or immune-mediated origin. Canine distemper virus (CDV) encephalitis is the most common infectious inflammatory cause of seizures in dogs younger than 1 year of age. Specifically, CDV causes acute polioencephalitis in young dogs. Seizures often are focal, which is characterized by “chewing gum” seizures, or consist of generalized motor activity. Similarly, seizures have been reported with postvaccinal CDV encephalitis in puppies. These seizures are often progressive and refractory to antiepileptic drug therapy. Identifying the underlying inflammatory disease process is important because the disease continues to progress without appropriate treatment.

Dogs with noninfectious inflammatory disorders with cerebral cortical involvement clinically manifest seizure activity. Granulomatous meningoencephalomyelitis (GME) rarely affects young dogs and is an inflammatory disease of the white matter of the brain. The disseminated form is usually acute and rapidly progressive, whereas the focal form progresses more slowly. About 20% of affected dogs have seizures along with other neurologic deficits. Breed-specific meningoencephalitis occurs in pugs, Yorkshire terriers, and Maltese. Pathologic features consist of nonsuppurative necrotizing meningoencephalitis, with a predilection for the cerebrum in pugs and Maltese. Seizures have also been reported in Yorkshire terriers, but brain-stem signs are more commonly manifested. Young dogs are predisposed and are usually 6 months of age or older. Dogs with the chronic form more often have clinical signs of generalized or focal seizures. Definitive diagnosis of the noninfectious inflammatory meningoencephalitides is based on a histopathologic diagnosis. A diagnosis can be suspected based on patient signalment as well as findings from advanced imaging and CSF analysis. Serology can assist in ruling out infectious causes.

Eosinophilic meningoencephalitis is characterized by eosinophilic pleocytosis of the CSF in dogs and a cat. Neurotoxic substances are released from the granules of eosinophils, causing secondary neurologic signs. This disease has been reported in young dogs. Neurologic signs are variable, but seizures can be a presenting sign. The diagnosis is based on CSF analysis. Recovery or remission of clinical signs can occur with glucocorticoid therapy.

**Trauma**

Seizures that occur after traumatic head injury can have an early or delayed onset. Early onset of seizures occurs within days of the injury and may pose an increased risk of seizure activity later. Controversy exists in human and veterinary medicine regarding the role of prophylactic antiepileptic drug therapy for patients with head injuries. Current management involves waiting to begin antiepileptic drug therapy until seizures actually occur.

Electric shock resulting from curious behaviors in young animals may induce seizures and potentially life-threatening noncardiogenic pulmonary edema.

**Toxicosis**

The CNS is primarily or secondarily involved with a variety of toxic substances. Dorman reported that seizures occurred in 8.2% of all cases of suspected toxicosis. Inquisitive behaviors, lack of discretionary eating habits, and physiologic alterations in drug disposition render pediatric patients more susceptible to toxicant exposure. Neonates have a more permeable blood–brain barrier than do adults, thus increasing the potential for CNS exposure to toxins. Skin hydration is highest in neonates, and topical exposure to lipid-soluble compounds (e.g., hexachlorophene, organophosphates) places pediatric patients at higher risk of significant absorption. Toxins induce seizures through a number of different mechanisms: increased excitation, decreased inhibition, and interference with energy metabolism.

**Asymptomatic Epilepsy**

**Idiopathic Epilepsy**

Epilepsy is characterized by recurrent seizures. The term idiopathic epilepsy, also known as primary or asymptomatic epilepsy, is used when there is no identifiable cause of seizures. The term inherited epilepsy is used when there is a genetic cause of seizures. Epilepsy suspected of having an inherited basis frequently occurs in dogs.
younger than 1 year of age. Although most dogs with idiopathic epilepsy have their first seizure at 1 to 5 years of age, seizures have been reported in Labrador retrievers as young as 2 months of age. An inherited basis, familial transmission, or a higher incidence has been recognized in many breeds. Based on pedigree analysis, a genetic basis is strongly suspected in keeshonds, Belgian Tervurens, Alsatian shepherds, Labrador and golden retrievers, vizslas, and a colony of laboratory-raised beagles. The mode of inheritance has been suggested in some breeds. Hall and Wallace found evidence of a single recessive gene contributing to a predisposition of epilepsy in keeshonds. Statistics suggest that seizures in Belgian Tervurens result from a complex pattern of inheritance. A polygenic multifactorial mode of inheritance is suggested in golden and Labrador retrievers. An autosomal recessive pattern of inheritance is suspected in vizslas with idiopathic epilepsy. In humans, genes have been identified for ion channel defects in some epilepsies.

DIAGNOSTIC CONSIDERATIONS

The appropriate diagnostic procedures for seizures in young animals are variable and depend on the most likely differentials. Tests may be subdivided into procedures that do and do not require anesthesia (Figure 3). The minimum database should include patient signalment, history, physical and neurologic examinations, and clinical pathology. The patient’s history plays an important role, especially if toxin exposure is a consideration. The history can also help identify vaccination status, pedigree information, environmental factors, and seizure patterns. Abnormal findings on physical and neurologic examinations lend support for symptomatic causes of seizures and more extensive diagnostic testing. Neuroanatomic localization for seizure activity is in the forebrain. Neurologic examination findings may reveal forebrain dysfunction or evidence of a multifocal or diffuse disease process. A funduscopic examination may show active or previous signs of chorioretinitis. Clinical pathology testing should include a complete blood count (CBC), serum chemistry profile, and urinalysis. Abnormal findings may further support a metabolic or toxic cause of seizures. Screening tests in a serum biochemical profile should include blood urea nitrogen, alkaline phosphatase, alanine transaminase, calcium, and blood glucose levels. Interpretation of results should also take into account an animal’s age because adult and immature animals have different serum values. Based on clinicopathologic abnormalities, additional diagnostic testing is indicated when specific organ pathology is suspected. Liver function tests, such as pre- and postprandial bile acid concentrations and blood ammonia levels, can provide evidence of hepatic dysfunction. Scintigraphy and ultrasonographic studies can further aid in identifying a PSS. Profiles for metabolic screening of blood, urine, and CSF are useful when storage disorders or inborn errors of metabolism are suspected.

Serology and immunologic testing may indirectly lend further support to infectious causes. Overall, viral causes are difficult to definitively diagnose with less invasive diagnostic testing. Results of serologic testing are also difficult to interpret because of the presence of circulating antibodies from maternal immunity, vaccination, or environmental exposure. Immunofluorescent antibody staining of epithelial cells from the conjunctiva has reportedly identified about 54% of dogs with CDV infection. Additional neurodiagnostic testing can further determine the type and extent of intracranial pathology. Ultrasonography is useful in animals with an open bregmatic fontanelle or intracranial arachnoid cysts. Findings of only mild hydrocephalus should be carefully interpreted because this may not be the inciting cause.

It is important to identify the underlying inflammatory or metabolic disease because clinical signs continue to progress without appropriate treatment.

Advanced imaging such as CT or MRI can aid in diagnosing structural abnormalities related to cranial and intracranial malformations, inflammatory disorders, and neoplasms. MRI is more ideal for identifying soft tissue abnormalities. In human medicine, the efficacy of using CT in children after the first nonfebrile seizure has been questioned based on lack of abnormal findings.

Electroencephalography in animals may show characteristic patterns in disease, such as hydrocephalus and
encephalitis. However, an animal’s age should be carefully considered because EEG patterns in young animals can mimic patterns associated with disease. An electroencephalograph can develop the pattern of an adult dog by approximately 5 months of age. In our experience, continual electroencephalography is also useful in monitoring persistent seizure activity and adequate response to therapy.

CSF analysis is particularly useful in identifying the presence of inflammatory disease. Because inflammatory disease is a more likely differential than neoplasia in younger animals, CSF analysis may more often provide a greater diagnostic yield than imaging procedures alone. CSF can be collected by puncturing the cerebellomedullary cistern. Analysis should include a nucleated cell count, protein concentration, and cytologic evaluation within 30 minutes of sample collection. Unfortunately, results of CSF analysis tend to be nonspecific for some disease processes. Intrathecal antibody production can be determined to further assist in diagnosing some infectious diseases.
CONCLUSION

Early recognition of the cause of seizures in young dogs and cats is important for appropriate therapeutic intervention. Inappropriate therapy can delay cessation of seizures and increase patient morbidity and mortality.

Watch for an upcoming article on managing seizures in young dogs and cats.

REFERENCES


2. Which of the following occurs within the ionic microenvironment of the immature brain?
   a. The resting membrane potential is increased.
   b. The potassium concentration is increased in the extracellular fluid of immature brains.
   c. The action potentials for immature neurons are of short duration because of prolonged potassium channel conductance.
   d. Glial function immaturity results in a decreased extracellular potassium concentration.
   e. An increased extracellular potassium concentration results in neuronal hyperpolarization.

3. An area within the brain in which myelination is completed last is the
   a. cerebrum.
   b. brain stem.
   c. olfactory region.
   d. hippocampus.
   e. pyriform lobe.

4. _________________ is the most common cause of secondary epilepsy in young dogs.
   a. Lissencephaly
   b. Hepatic encephalopathy
   c. Hydranencephaly
   d. Hypoglycemia
   e. Hydrocephalus

5. Which statement regarding glucose use in the immature brain is correct?
   a. Receptor numbers for the glucose transport protein are high.
   b. Lactic acid can support 60% of cerebral energy metabolism.
   c. Receptor numbers for the ketone body transport protein are low.
   d. Receptor numbers for the lactic acid transport protein are low.
   e. The neonatal brain has a readily available source of ketones to prevent acute hypoglycemia.

6. Which statement regarding CDV encephalomyelitis is correct?
   a. CDV encephalomyelitis is the most common inflammatory cause of seizures in dogs younger than 1 year of age.
   b. CDV causes acute leukoencephalomyelitis in young dogs.
   c. Seizure activity is characterized only by “chewing gum” seizures.
   d. Seizures are often controlled well with antiepileptic drug therapy.
   e. Seizure activity has not been associated with post-vaccinal CDV encephalitis.

7. Which noninfectious inflammatory disorder is least likely to occur in young dogs with seizure activity?
   a. pug encephalitis
   b. Yorkshire terrier encephalitis
   c. Maltese encephalitis
   d. steroid-responsive meningoencephalomyelitis
   e. GME

8. Which factor is likely to predispose young dogs and cats to toxin exposure?
   a. increased blood–brain barrier permeability
   b. increased skin hydration
   c. inquisitive behavior
   d. alterations in drug disposition
   e. all of the above

9. Which statement regarding primary epilepsy is correct?
   a. Inherited epilepsy can manifest with seizures in dogs younger than 1 year of age.
   b. Inherited epilepsy is a rare form of idiopathic epilepsy.
   c. Epilepsy does not occur in Labrador retrievers.
   d. All inherited epilepsies have an autosomal recessive mode of inheritance.
   e. Inherited epilepsy is a form of symptomatic epilepsy.

10. Which diagnostic method can provide a greater diagnostic yield if an inflammatory disorder is suspected of causing seizures in young animals?
    a. serology
    b. CBC
    c. CSF analysis
    d. funduscopic examination
    e. CT or MRI