

Epilepsy in the dog and cat: Clinical presentation, diagnosis, and therapy

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SUMMARY

Seizures in the dog and cat are frequently seen in clinical practice. Typically, the veterinarian is contacted by the owner subsequent to a seizure, which, to the owner, can appear quite dramatic. These animals, therefore, often present as acute patients. Even though the seizure has, in most cases (with the exception of status epilepticus), ceased, when owner and pet have reached the clinic, the owner is often worried and in the need of advice.

The veterinarian's immediate task is to treat acute seizure activity. Thereafter, the origin of the seizure must be ascertained, so that one can determine if the cause of the seizure is epilepsy or another disease with symptoms imitating epilepsy.

Animals with chronic epilepsy should undergo antiepileptic treatment. Haematological/biochemical parameters, seizure status, medical therapy and any side effects should all be monitored. The owners of these patients need information about the nature of the disease, as well as support from the veterinarian, especially in the initial phase and in cases of refractory epilepsy.

The goal of this paper is to give a concise introduction to epilepsy and to present a practice-relevant overview of diagnostic and therapeutic possibilities.

Keywords: Epilepsy, seizures, seizure classification, antiepileptic drugs, dog, cat

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Introduction

Epilepsy is a disease characterised by recurrent seizures (two or more) originating from the brain. Seizure activity arises as a sequel to abnormal hypersynchronous electrical activity which stems from a group of local cerebral neurons or from the whole cortex in collaboration with subcortical structures. Epilepsy is, above all, a consequence of an imbalance between excitatory and inhibitory mechanisms in the brain. Factors involved in epileptogenesis involve, amongst other things: neuroplasticity/synaptic plasticity, decreased GABA (gamma amino butyric acid) activity, increased glutamate activity, new formation of excitatory

circuits, receptor alterations, ion channelopathies, dysfunction of glia cells, cortical dysgenesis, and intracranial pathology.

Epilepsy is characterised by a sudden loss of control, paroxysmic (episodic) manifestation (seizures which suddenly occur and suddenly cease), transient seizures (seldom longer than two minutes), and the clinical seizure symptoms in the individual patient follow a repetitive pattern where symptoms and sequence are identical from episode to episode. Clinical signs include an altered level of consciousness, and motor, sensory, autonomic and behavioural disturbances [1]. Epileptic seizures occur typically when the animal is quiet, sleeping, or upon awakening. This is because the brain's excitatory threshold is lower in these states than when the animal is awake and active. In individuals with epilepsy, seizures can be provoked by external factors such as excitement/agitation, flashes of light, and sleep deprivation.

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Epilepsy is the most frequently occurring neurological disease in the dog and cat. In the literature, prevalence is estimated to vary between 1.5 – 5.7% in the dog and to be 0.5% in the cat [2-6]. An epidemiological population study of Labrador retrievers demonstrated a prevalence of epilepsy of 3.1% in this breed in Denmark [7]. A familial predilection for epilepsy is reported in many breeds, including, among others, the Beagle, Keeshound, Belgian Tervueren, Golden retriever, Labrador retriever, Vizla, Bernese mountain dog, Shetland sheepdog, and English springer spaniel [4.7-15].

A more detailed review of the disease can be found in neurology textbooks [1].

When a veterinarian is presented with a patient suffering from seizures, he/she must determine the origin of the seizures, and thus clarify whether one is dealing with epilepsy or with another disease with epilepsy-like signs.

Classification of Epilepsy and Epileptic Seizures

From the times of antiquity, classification has been employed in an attempt to identify and characterise a specific disease and/or syndrome. The first attempt at classifying epilepsy in people is documented on stone tablets found in Babylon [16]. The veterinary epilepsy terminology is traditionally based on the human ILAE (International League Against Epilepsy) classification system [17-18]. Human, as well as veterinary, classification and terminology systems are constantly under development [19-21]. A standard veterinary classification model for epilepsy, universally accepted by all does not, as yet, exist.

Classification of epilepsy

The classification of epilepsy is based upon the underlying aetiology. This classification includes three categories: idiopathic epilepsy, symptomatic epilepsy, and probable symptomatic epilepsy (previously known as cryptogenic, which means ‘of indeterminate origin’).

The term idiopathic epilepsy refers to epilepsy of unknown cause (there is no structural cerebral pathology). Idiopathic epilepsy in humans is associated with a possible or known genetic component. Animals suffering from idiopathic epilepsy will interictally appear clinically and neurologically normal.

Symptomatic epilepsy is defined as epilepsy caused by a known/identified disorder of the CNS, i.e. focal cerebral pathology can be identified (Table 1). A very common cause of symptomatic epilepsy in people is micro-structural changes of the cerebral cortex (for example neuronal migration disorders) [26]. In these patients, seizures are often the only clinical indication of the subtle CNS pathology. Hippocampal changes are described in cats with epilepsy, and changes in the motor cortex are described in the dog [27-28]. In a group of dogs with medicine-refractory epilepsy, changes in the temporal region – a region in people that is often the site of an epileptic focus - have also been identified [29]. Symptomatic epilepsy is characterised by partial seizures with or without secondary generalisation (see the section on classification of seizures). A patient with symptomatic epilepsy can have concurrent neurological signs, but the patient can also be free from clinical signs in the interictal period.

Congenital

Lissencephaly
Hydrocephalus
Lysosomal storage disease
Neuronal migration disorder (neuronal heterotopia/dysplasia)

Traumatic

Haemorrhage (diffuse/haematoma)
Cerebral oedema
Post-traumatic scar tissue

Cerebro-vascular accidents (stroke)

- Ischemic infarction (vascular occlusion due to thrombus or embolus)
- Haemorrhage (vessel lesion or rupture)

Inflammatory

Aseptic meningitis/meningoarteritis
Granulomatous meningoencephalitis (GME)
Pug dog encephalitis

Infectious

Viral (meningitis/meningoencephalitis, for example canine distemper, FIV, FIP)
Bacterial (meningitis/meningoencephalitis/abscess)

Tick-born

Tick born encephalitis (TBE)

Neoplasia

Primary neoplasia
Metastasis

Table 1: Examples of possible aetiologies of symptomatic epilepsy

Probable symptomatic epilepsy is used as a designation for epilepsy with a suspected symptomatic cause, which, however, remains obscure. This type of epilepsy is, just like symptomatic epilepsy, characterized by partial seizures with or without secondary generalisation.

Classification of Epileptic Seizures

The clinical manifestation of epileptic seizures is directly related to the amount and distribution of abnormal electrical activity in the brain.

Epileptic seizures can be classified as primary generalised seizures, focal seizures (formerly known as partial seizures), or focal seizures with secondary generalisation.

With primary generalised seizures, there is a sudden outburst of abnormal electrical activity throughout both cerebral hemispheres. Without warning, the patient collapses with tonic, clonic, or tonic-clonic seizures.

With focal seizures, an abnormal electrical activity arises in a group of neurons localised to a specific area of the brain (an epileptic focus), and the clinical signs reflect the functions of

the area involved. Focal motor, sensory, autonomic, or psychic behavioural signs can be seen [30].

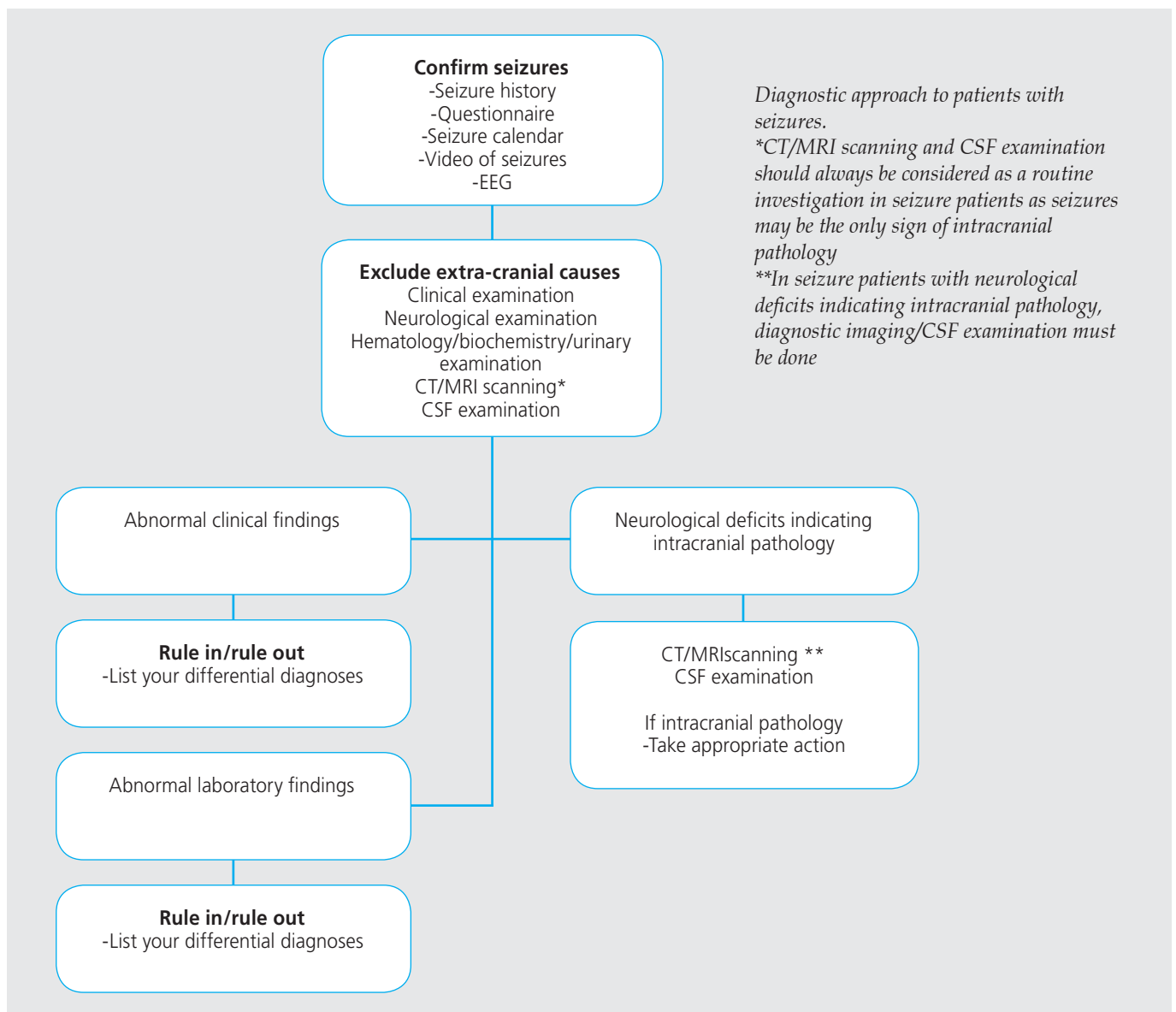
Focal seizure signs seen in animals with localised motor phenomena, could be, for example, rhythmic twitching of an extremity, abnormal rhythmic blinking and, in cats, twitching of the whiskers. Focal seizure activity representing psychic and/or sensory seizure phenomena in the brain will, in dogs, result in behavioural changes such as e.g. uneasiness, anxiousness, restless wandering, pacing or abnormal clinging to its owner. Finally, focal seizures with parasympathetic/epigastric components may present as for example, hypersalivation, vomiting, or dilated pupils [30].

Focal seizures in the human are traditionally divided into two categories: simple focal seizures (where the state of consciousness is maintained), and complex focal seizures (where the state of consciousness is impaired). It is difficult to appraise the state of consciousness in seizing animals, as this is limited to subjective interpretations by the one observing the seizures, whereas in people a personal account can in many cases be given when the seizure is finished.

If the electrical activity in the epileptic focus becomes sufficiently massive, focal seizures can spread through subcortical structures to involve the entire brain (called a focal seizure with secondary generalisation). The initial seizure symptoms will be localised then followed by tonic, clonic, or tonic/clonic convulsions. This is the most common seizure type observed in the dog [1]. The onset of the focal seizure is often very brief (from a few seconds to minutes) and the secondary generalisation (convulsions) follows rapidly. The focal seizure onset may therefore be difficult to detect.

The term "aura" has, in human terminology, historically been synonymous with the simple focal seizure signs that precede a complex focal seizure alone or one followed by secondary generalisation (convulsions). In humans the simple focal seizure signs are recognised as a warning sign of a forthcoming seizure development. In some epileptic animals, owners do indeed also report that they can predict a convulsion when the familiar initial focal seizure signs appear. In humans, aura has been used only to denote symptomatology that encompasses subjective sensory phenomena as well as vegetative signs (for example, the

Figure 1



epigastric sensations accompanying mesial temporal epilepsy). Thus, in humans, motor phenomena are not included in the symptomatology of aura.

An epileptic seizure can be divided into two phases: ictus (seizure activity) and postictal phase (reestablishment of normal brain function). Ictus can consist of a primary generalised seizure, a focal seizure, or a focal seizure with secondary generalisation. In some cases, ictus can be preceded by a prodrome – a long-lasting (hours or days) change of disposition, in humans e.g. in the form of anxiety, irritability, withdrawal and other emotional aberrations. In dogs the most common prodromal sign described is restlessness, a state lasting from hours to days [23]. Prodromes are traditionally considered to be a pre-ictal phenomena because abnormal electroencephalographic (EEG) brain activity cannot be registered. It is probable, however, that prodromes represent long-lasting, weak (subtle) focal seizure activity [1].

In the postictal phase, the brain returns to its normal function. Typically, the animal is tired, disoriented, ataxic, hungry, thirsty and/or, in some cases, aggressive. The postictal phase can last for few minutes or up to several hours.

Diagnostic Evaluation

The clinical diagnosis of epilepsy is primarily based upon the owners' observations of seizure activity, video recordings of seizures, neurological examination, and the ruling out of differential diagnoses. The primary aim of the initial examination is therefore to differentiate between epileptic seizures and non-epileptic episodes mimicking epilepsy and the diagnostic work-up includes both clinical and paraclinical examinations (figure 1/ Table 2).

Electroencephalography (EEG) is the measurement of brain activity and is used to identify (record) abnormal electrical activity in epilepsy patients. It is not a routinely available veterinary diagnostic option, but is available in some specialist clinics. EEG readings are not abnormal in all patients suffering from epilepsy, especially in those with focal seizures. A normal EEG does not, therefore, rule out epilepsy. The diagnostic work-up should be expanded to include computerized tomography (CT) or magnetic resonance imaging (MRI) as well as examination of the

Table 2: Possible causes of seizure like episodes of non-epileptic origin (differential diagnosis)

- Cardiovascular dysfunction
- Respiratory dysfunction (especially upper airway)
- Syncope
- Anaemia
- Hypoglycaemia
- Electrolyte unbalances
- Hepatic dysfunction
- Renal dysfunction
- Neuromuscular disease (episodic e.g. myasthenic syndromes)
- Hyper-thyroidism
- Pyrexia
- Intoxication
- Behaviour

cerebrospinal fluid (CSF) in patients suspected of suffering from symptomatic epilepsy (neurological signs occurring interictally and/or signs of active brain disease). It should be stressed that in some patients with intracranial pathology epileptic seizures are the only indication of intracerebral abnormalities. The use of diagnostic imaging should therefore always be considered in such patients.

A thorough anamnesis with the focus on seizure description is essential in the diagnosis of epilepsy in clinical practice. The veterinarian is limited to relying on owner description of seizures, as it is a rare occurrence that the animal has a seizure while in the clinic. Many owners focus, of course, on the most dramatic aspects of the seizure event (the convulsion) and leave out essential details (for example, events and clinical signs preceding the convulsion) which he/she do not consider important. To avoid loss of this type of information, the veterinarian must ask probing questions. A questionnaire focussing on seizure phenomenology and classification of seizures is an important diagnostic tool when working with suspected epilepsy patients (Table 3). Home video recordings of the seizure are a good supplement to the owner's description of the event.

A veterinarian should be suspicious of a non-epileptic cause in the following circumstances: whenever the clinical examination and haematology/biochemistry indicate organic/systemic disease; in those cases where the anamnesis indicates prolonged (>5-10 minutes) episodes, in cases where the seizures (or seizure-like episodes) only occur in connection with physical activities; and where the owner reports that the animal faints or collapses, becomes cyanotic or episodically exhausted and reluctant to walk (Figure 2). It is very important that the veterinarian distinguishes between ictus and the postictal phase when questioning the owner about the event; otherwise there is the possibility that the clinician will erroneously conclude that the duration of the seizure is too great to be epilepsy. The owner, who cannot distinguish between the seizure and the restitution phase, will consider everything that occurred as the seizure. Therefore, the owner who is not questioned carefully will impulsively describe the episode as lasting longer than the few minutes the seizure activity actually took.

A suspicion of an underlying pathological process in the brain (symptomatic epilepsy) should be considered in animals with an unusually early onset of epilepsy (e.g. a congenital defect) or a very late-in-life onset of epilepsy (e.g. neoplasia). In animals suddenly afflicted with many and/or extreme seizures, and in animals without epilepsy who suddenly go into status epilepticus (a state in which an animal experiences repeated seizures while not regaining consciousness between seizures) the list of differential diagnoses will include neoplastic, inflammatory and infectious disease.

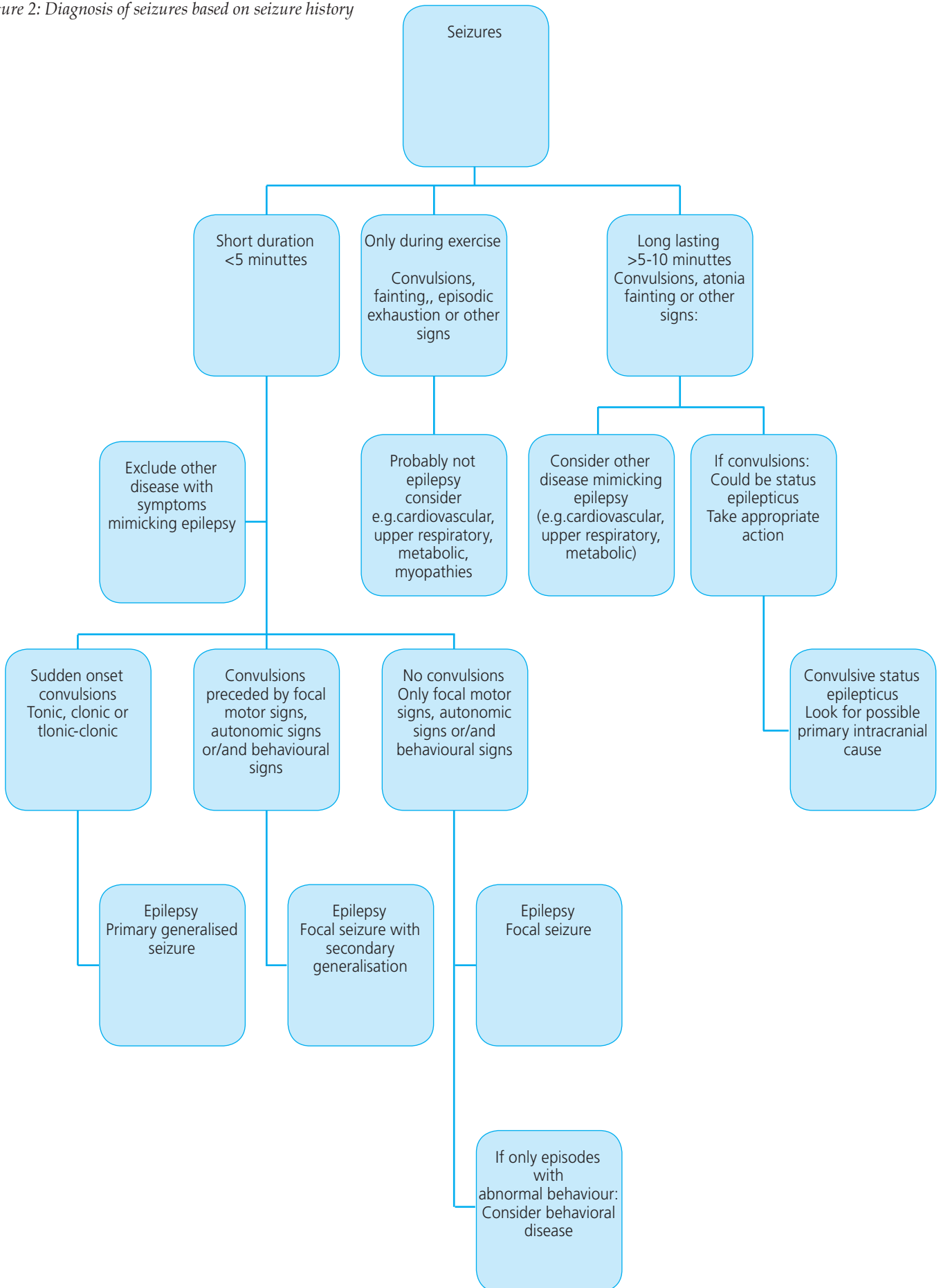
Medical Therapy of Epilepsy

When to start therapy in patients with epilepsy depends on the frequency of seizure activity and on the owner's attitude to medical therapy. Some owners do not like the idea of treating their pets at all, while others do not tolerate more than a few seizures per year. The decision to begin therapy must, therefore, be made on the merits of each case. Generally, treatment should be started if there are four to six seizures within a six

Table 3

Questionnaire for seizure patients						
Name/ID	Date of birth	Breed	Gender	Neutered yes/no	Weight	Date of interview
Medical history						
Epilepsy among close relatives						
Known birth complications						
Previous head trauma						
Previous CNS infection						
Chronic disease apart from seizures						
Medication						
Seizure history						
Day, month, year at first seizure						
How many seizures have occurred since the first seizure episode						
Describe a typical seizure and seizure development (allow the owner to report his observations spontaneously)						
Are there any symptoms that are repeatedly seen in association with seizures? If yes give a description of these signs						
For how many minutes does a seizure last						
Do the seizures occur						
<ul style="list-style-type: none"> • During rest (day or night or both) • At awakening • During normal activity (walking, playing) • During excessive activity (hunting, training, excessive playing) 						
Do seizures occur solely or in clusters (more than one seizure in 24 hours)						
If clusters of seizures occur, how many seizures occur during 24 hours						
If clusters of seizures occur, how many minutes/hours elapse between each seizure						
Does the animal recover fully between each seizure						
Mark seizure episodes in a calendar						
Can e.g. light blinks, excitement, high sounds or stressful surroundings provoke seizures						
If the animal is a female, have seizures then been clustered solely around the time of heat						
Ictal signs – primary generalized seizures						
Does the animal experience sudden convulsions (kicking, cycling movements of the extremities or increased body tonus/body stiffness and/or neck extension)						
Ictal signs – focal seizures with secondary generalization						
If the animal does experience convulsions, are the convulsions then consistently preceded by other symptoms of short duration e.g.						
<ul style="list-style-type: none"> • Head shaking • Head turning to the left or to the right • Contractions of one extremity • Quick continuous blinking of the eyes • Twitching of the whiskers • Abnormal eye movements (horizontally or vertically) • Excessive salivation • Vomiting • Wide open pupils • Barks for no reason • Attention seeking • Follow you around/stick close to you? • Seem to be worried? • Express signs of fear • Seem disoriented • Appear to not be able to recognize you • Appear hallucinated • Bump into things • Appear blind • Aggressiveness • Anything else 						
Focal seizures alone						
If the animal does not experience convulsions does one or more of the above mentioned symptoms occur episodically where they follow a repetitive pattern and where symptoms and sequence are identical from episode to episode						
If yes, what symptoms						
Duration of signs (minutes)						
Other information						
Is consciousness preserved or lost during seizures						
Any urine or faecal incontinence during seizures						
Postictal signs						
How many minutes/hours will it take for the animal to recover fully after an episode with convulsions or an episode with other signs than convulsions has come to an end						
These signs could be e.g.						
<ul style="list-style-type: none"> • Appear to have transient impaired vision • Appear to be disoriented/confused • Hyperactive • Wants to go in the garden • Hunger/thirst • Aggressiveness • Exhausted/Sleepy • Anything else 						

Figure 2: Diagnosis of seizures based on seizure history



month period, if seizures begin to accumulate, if seizures are prolonged, and/or in cases of status epilepticus, where there is an increased risk of neuronal damage and continued seizure activity. The treatment of epilepsy is not necessarily life-long. Spontaneous remission is possible, and some patients "grow out of" the disease [7]. It is therefore a possibility to attempt to wean the animal off therapy in cases where there has been no seizure activity within the preceding year. This must not be done abruptly, but should occur with a gradual dose reduction over a long period of time (six to eight months). A rapid dose reduction should be avoided, because there is an increased risk of seizure relapse, and because, in the case of phenobarbital, the drug is addictive.

Many of the newer antiepileptic drugs are not, unfortunately, effective in the dog and cat, because they are efficiently metabolised in the liver in these animals. The drugs of choice in the long-term therapy of epilepsy are limited to two drugs with long half-lives: phenobarbital and potassium bromide (Table 4). Phenobarbital should be given to dogs and cats at a dosage of 3 – 5 mg/kg/day. The total daily dosage is divided and administered twice a day to reduce both fluctuations in the serum concentration and side-effects. The serum concentration of phenobarbital varies greatly between individuals and it is therefore necessary to monitor the serum concentration of phenobarbital in the individual patient. Phenobarbital is metabolised in the liver. A steady state is achieved after about two weeks, and only after that period can full therapeutic effect be expected. At this point the patient's serum concentration of phenobarbital can be measured. The therapeutic serum concentration (the level within which the drug is expected to be therapeutically effective) is 65-150 µmol/l (15-45 µg/ml). The dosage of phenobarbital is not adjusted if the animal is seizure free in the presence of a low serum concentration of phenobarbital. The phenobarbital dosage is increased if the animal is not seizure free. The serum concentration of phenobarbital should be measured with every dosage change

(but only after two weeks have passed) and if significant alterations in the clinical seizure pattern arise. If the patient is still not seizure free or the seizures are not satisfactorily reduced despite high normal serum phenobarbital concentrations, add-on therapy with bromide is indicated. Up to 50% of dogs whose seizures are not adequately controlled with phenobarbital therapy become satisfactorily controlled or seizure free when phenobarbital therapy is combined with potassium bromide. The phenobarbital dosage can often be gradually reduced by 25 – 50% in these patients. Due to the extremely long half-life of bromide the complete therapeutic effect is first attained after 100 to 200 days. Ideally, reduction in phenobarbital dosage should be begun after this time. In practice, however, it may be necessary to begin a phenobarbital dosage reduction at an earlier time due to the side effects associated with combination therapy (at the earliest two months after the start of add-on therapy, when bromide is expected to begin to have some effect). Phenobarbital dosage reduction should be carried out slowly (over several months). In countries where bromide is not available as a commercial formulation, it can be dispensed from a pharmacy (bromide 100 mg/ml aqueous solution or bromide 200 mg/ml aqueous solution). When potassium bromide is given as an add-on medication in dogs a dosage of 30 – 40 mg/kg/day should be used (preferably with a late evening meal). Potassium bromide may also be used as a first choice and then the dosage should be 40 – 60 mg/kg/day. The therapeutic range of bromide in dogs is 100-200 mg/dl when potassium bromide is used as an ad-on drug and 250-300 mg/dl when used as monotherapy. Excretion occurs via the kidneys and is dependent on concomitant chloride intake. Attention should therefore be given to the dietary influence of the excretion of bromides. A high dietary chloride content shortens the elimination half-life causing decreased therapeutic serum bromide concentrations and thereby loss of therapeutic efficacy [31]. Bromide is a first choice for seizure control in canine patients with hepatic dysfunction.

Table 4: Antiepileptic drugs

	Mechanism of acting	Dosage mg/kg/24 hours	Therapeutic serum value	Most common side-effects	Indication
Phenobarbital	Increases GABA causing a decrease in pre- and postsynaptic excitability	3-5 divided into 2 daily dosages	65-150 µmol/l (15-45 µg/ml)	Sedation Ataxia Polyphagia Polydipsia Incontinence	Mono-therapy dog and cat
Potassium bromide	Intracellular neuronal accumulation of bromide causes hyperpolarization of postsynaptic membranes	Ad-on: 20-40 Monotherapy: 60 Given as one daily dosage	100-200 mg/dl	Sedation Ataxia Depression Dermatological signs Gastrointestinal signs	Ad-on therapy with phenobarbital Mono-therapy
Diazepam	Increases GABA and hereby suppress neuronal firing	Dog: 1-2 Cat 0.5-1		Sedation	Acute anti-convulsive treatment Status epilepticus

Bromide should not be used for the treatment of epilepsy in the cat, as there is little documentation of its effect and side-effects when used in this species. In dogs both phenobarbital and bromide may cause side-effects. The most common adverse effects seen with phenobarbital are sedation, ataxia, and polyphagia. Signs of bromide toxicosis include ataxia, depression, stupor, anisocoria, muscle pain, dermatological signs such as rash and nodular pustular skin lesions, and gastrointestinal signs such as anorexia, vomiting and constipation. Necrolytic dermatitis has been described in dogs treated with phenobarbital [32]. An increased risk of development of acute pancreatitis has been reported in connection with long-term treatment with phenobarbital and bromide [33 – 34]. Bile acid levels should be monitored as anticonvulsants can cause hepatotoxicity [35 – 37]. As changes in blood parameters are sometimes seen in patients receiving long-term treatment with phenobarbital, haematology and clinical chemistry profiles should be obtained every six months. Immune-mediated neutropenia and thrombocytopenia have been described due to phenobarbital [38]. In dogs one may then have to wean the patient off phenobarbital and replace it with bromide. Other changes of minimal clinical significance include a rise in alanine aminotransferase and alkaline phosphatase (due to enzyme induction), as well as a reduction of serum total and free thyroxine with normal thyroid-stimulating hormone values and no concurrent clinical signs of hypothyroidism [39 – 41].

It is important to give the owner a realistic description of expected side-effects, emphasising that they appear with greater intensity at the start of the treatment, only to subside after two to four weeks. Otherwise, the owner may gain the false impression that the animal's quality of life will be permanently impaired by the antiepileptic treatment and may, therefore, choose to have the animal euthanased. Combination therapy results in more side-effects than monotherapy.

A big problem in the treatment of patients with epilepsy is dealing with the not so small group of epilepsy patients that are refractory to treatment with phenobarbital and/or bromide. In recent years, it has been shown that in some dogs not responding well to therapy with phenobarbital and potassium bromide, a reduction in seizure activity may be achieved with a combination of these two drugs with the human antiepileptic drugs gabapentin, levetiracetam, or zonisamide [42 – 44].

Primidone (Mysoline®) has been used in dogs with epilepsy and is an effective drug. The anticonvulsant effect can be attributed to the 85% of the drug that is metabolised to phenobarbital in the liver. Primidone has a greater hepatotoxic potential than phenobarbital and it is often more expensive. Phenobarbital is therefore the preferred product.

Diazepam is an especially effective anticonvulsant in the acute treatment of epilepsy (the drug of choice in the initial treatment of status epilepticus), but is not useful in the long-term treatment of epilepsy. Dogs develop tolerance to the drug within a few days and long-term treatment in cats can lead to liver damage [45]. For a more thorough review of the various antiepileptic drugs, the reader is referred to the neurology literature [1,46,47].

In patients suffering from symptomatic epilepsy due to an active pathological condition in the brain, anticonvulsant treatment should be combined with treatment targeted directly towards the primary CNS disease. Euthanasia may be a better option than anticonvulsant treatment in patients suffering from progressive,

incurable brain disease causing unnecessary suffering.

It has been stated that neutering of animals suffering from epilepsy could contribute to the reduction of seizure activity. This applies only in animals that experience clusters of seizures in connection with oestrus (catamenial epilepsy) and in male animals whose sexual drive is judged to be so profound that it is stressful and therefore seizure provoking (1).

Monitoring epilepsy patients

An epilepsy patient should be re-assessed at least twice a year to assure clinical control of the patient's epilepsy status. The owner should bring in an overview of seizures in that period (keep a seizure calendar), so that treatment efficacy can be evaluated. The serum concentration of phenobarbital/potassium bromide should be measured when there is an increase in seizure frequency, when there is a resumption of seizure activity after a seizure-free period, and after adjustments are made in drug dosages.

If there is treatment failure, the following possible reasons should be considered:

- Incorrect diagnosis
- Incorrect choice of antiepileptic drug
- Incorrect dosage of the antiepileptic drug
- Failure to monitor drug levels
- Owner non-compliance
- Presence of a systemic disease causing increased metabolism or excretion of the antiepileptic drug
- Patient has gained weight (drug dosage needs to be adjusted)
- The animal has developed a tolerance to the drug
- Monotherapy is insufficient

It is crucial for the success of the treatment that the following points are discussed with the owner:

- Not all patients achieve a seizure-free existence (but often a satisfactory reduction in seizure activity)
- For some animals it is not possible to achieve a satisfactory therapeutic effect consistent with good quality of life
- The animal's disease can be a psychological strain on the family
- Prepare the owner for the side-effects of the drug
- Regular case monitoring is necessary
- Stress can provoke seizures
- Clustering of seizures is potentially dangerous – a veterinarian must be contacted immediately
- Spontaneous remission is possible

Conclusion

Attention must be paid to both clinical evaluation and owner instruction when dealing with dogs and cats suffering from epilepsy. The owner typically asks questions concerning the nature of the disease, the influence of the disease on animal's quality of life, the effect of the disease on work performance, the expected economic and emotional demands, and the expected efficacy of the treatment. Many dogs and cats can live a life without, or with tolerable, complications associated with antiepileptic therapy. However, it is important to emphasise that

in some animals the seizures cannot be reduced to a satisfactory level.

The veterinarian can, with careful explanation, promote an understanding of a disease which otherwise appears frightening and unmanageable. In this way, the owner can be motivated to continue living with an animal that needs lifelong treatment without necessarily attaining a totally seizure-free existence.

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