Journal of Feline Medicine and Surgery (2010) **12**, 910–916
doi:10.1016/j.jfms.2010.07.001

ELSEVIER

jfms

Clinical comparison of primary versus secondary epilepsy in 125 cats

Ákos Pákozdy DVM, DECVN[1]*, **Michael Leschnik** DVM[1], **Ali Asghar Sarchahi** DVM, PhD[2], **Alexander G Tichy** DSC[3], **Johann G Thalhammer** DVM[1]

[1]*Clinic for Internal Medicine and Infectious Diseases, University of Veterinary Medicine, Neurology Service, Veterinärplatz 1, A-1210 Vienna, Austria*
[2]*Department of Veterinary Clinical Studies, School of Veterinary Medicine, Shiraz University, Iran*
[3]*Institute of Physics and Biostatistics, University of Veterinary Medicine, Vienna, Austria*

In the present study 125 cats with recurrent seizures were analysed. The main goal was to investigate the aetiology and compare primary epilepsy (PE) with secondary epilepsy (SE) regarding signalment, history, ictal pattern, clinical and neurological findings. Seizure aetiology was classified as PE in 47 (38%) and SE in 78 (62%) cats. SE was caused mainly by intracranial neoplasia (16), hippocampal necrosis (14), toxicosis (eight), and encephalitis (seven). A significant difference between PE and SE was found in: age, body weight, duration of seizure, occurrence of status epilepticus and neurological deficits. Status epilepticus, altered interictal neurological status and seizure onset over the age of 7 years indicated SE more frequently than PE. If the seizures occurred during resting conditions and rapid running occurred the aetiology was more likely to be PE than SE.

Date accepted: 4 July 2010

Epilepsy is a condition characterised by recurrent seizures.[1] It can be categorised as symptomatic (or secondary), probable symptomatic (cryptogenic) or idiopathic based on aetiology.[2] Epilepsy is classified as idiopathic or primary epilepsy (PE) when no underlying cause can be identified and genetic mechanisms are presumed.[3] A recent paper used the nomenclature PE and secondary epilepsy (SE) for feline seizure disease.[4] Although PE has been described in many dog breeds and a genetic background has been suggested,[5–17] no genetic origin has been established in cats.

Despite a few references dealing with PE and SE in dogs,[18–20] there is only one recent study on the aetiology and classification of feline epilepsy based on large number of cases.[21] Furthermore controversial data were published about PE. Some studies suggested that PE is rare in cats,[23,24] although other studies reported a considerably higher rate of PE in cats.[22,25,26] In the present study 125 cases of secondary and PE in cats were analysed. The main aim was to find ictal signs that could help differentiate between PE and SE. The second goal was to compare PE with SE with respect to age, body weight, gender, breed, and clinical and neurological findings in big case material. And third, as there is much more known regarding the aetiology and diagnosis of canine epilepsy, we looked for differences between dogs and cats regarding PE and SE.

Materials and methods

A total of 235 cats with a history of recurrent seizures were reviewed at the author's clinic, between January 2003 and November 2008. The lack of accurate examination or incomplete follow-up information resulted in exclusion of 110 cases. Every patient (125) included in this study underwent a thorough physical and neurological examination. The following main ancillary diagnostic tests were used in the work-up: routine serum biochemistry and haematology (123), cerebrospinal fluid (CSF) analysis (65), brain computed tomography (CT; CT Pace High Speed, Fa General Electric, Vienna, Austria) (15), magnetic resonance imaging (MRI; MR unit 0.23 Tesla, Outlook, Gold Performance, Philips Medizinische Systeme, Vienna, Austria) (19) and pathohistological examination (49). PE was considered when the results of work-up were normal, or the work-up was not complete but more than 1 year had passed since the onset of seizures without any interictal neurological deficits. Cats with PE did not have any evidence of neurological disease other than seizures at any time during their lives. All cats with PE were re-evaluated at least

*Corresponding author. Tel: +43-1-25077-5101; Fax: +43-1-25077-5101. E-mail: akos.pakozdy@vetmeduni.ac.at

1098-612X/10/120910+07 $36.00/0

12 months after diagnosis by one of two authors (AP, ML) and no abnormalities were identified on physical and neurological examination. Cluster seizures were considered if there was more than one seizure within 24 h, and status epilepticus if the seizure lasted longer than 5 min, or if a series of seizures occurred with interictal impairment of neurological status. A focal seizure was characterised by motor activity in some muscles or muscle groups with or without generalisation. The seizure was considered to be generalised when motor activity involved the whole body. The diagnosis of SE was based on the history of seizures and confirmed pathological findings in haematology, serum biochemistry, CSF analysis or morphological changes of the brain as identified by CT/MRI or histopathological examination. Special emphasis was placed on signalment, history, the characteristics of ictal and postictal phases, and the results of clinical and neurological examination. Data were analysed by using SPSS 17.0 for Windows. For statistical analysis between PE and SE groups, χ^2 tests and Student's *t*-tests were used. To compare the mean values of the two groups a *t*-test for independent samples was performed. To compare the frequencies, χ^2 tests were carried out. A value of $P < 0.05$ was considered significant. The following variables were analysed: age at first seizure, body weight, gender, breed, activity during seizure (focal seizure, generalised seizure, trembling, urination, defecation, salivation, vocalisation, rapid running), duration of seizure, presence of status epilepticus, postictal presence of polyphagia, polyuria, polydipsia, blindness, deafness, aggression and the results of clinical and neurological examinations.

Results

Prevalence, age, breed, body weight

During the observation period 235 cats with a seizure in their medical history were examined, amounting to 3.5% of the hospital population at that time.

The breeds of cats represented were European domestic shorthair (117), Persian (two), Siamese (two), Chartreux (one), European domestic longhair cat (one), Somali (one), and Maine Coon (one). In the present study 93.6% (117/125) of cats were European shorthair, a proportion that is considered to be high because only 82.1% (5946/7243) of cats referred to our clinic are domestic shorthairs ($P = 0.001$). On the other hand only 1.6% (2/125) of seizuring cats were Persian while 7.4% (533/7243) of cats referred to our clinic are Persian, and thus the proportion of this breed was low ($P = 0.013$). Age at the first seizure ranged from 4 months to 20 years (mean ± SD: 6.99 ± 5.31). The mean age of patients at the onset of seizures was 4.6 years in PE (ranging from 0.5 to 14 years) and 8.4 years in SE (ranging from 0.3 to 20 years). There were significant differences in the onset of seizures between PE and SE (Table 1).

Between 1 to 7 years of age the number of cats with PE was significantly higher than in cats with SE (68.1% versus 37.2%, $P < 0.0001$). Outside this range the proportion was inverse (31.9% versus 62.8%, $P = 0.004$). If the seizure onset was between 1 and 7 years of age, the diagnosis was 1.84 times more likely to be PE than SE. If the seizure onset was after 7 years of age the diagnosis was 3.5 times more likely to be SE than PE similarly to dogs (Fig 1).

The mean body weight of the PE group was 5.14 kg (ranging from 2 to 10). This was significantly higher than the mean body weight of the SE group, which was 4.03 kg (ranging from 1 to 9) (Table 1). There were 27 male and 20 female cats with PE and 45 male and 33 female with SE and there was no significant difference in gender between groups.

Aetiology

SE was diagnosed in 78 cats and PE was suspected (PE) in 47 cats. The most common aetiology of SE was intracranial neoplasia ($n = 16$), hippocampal necrosis ($n = 14$), toxicosis ($n = 8$), and encephalitis ($n = 7$). The classification and cause of seizures are summarised in Table 2.

Characteristics of seizure

Different clinical parameters were analysed and compared between PE and SE groups regarding to type of seizure, ictal and postictal signs, ictal characteristic and interictal neurological abnormalities. The occurrence of different parameters and *P* value of *t*-tests for independent variables are listed in Table 3. The significantly different independent variables were: ictal rapid running, ictal mydriasis, postictal blindness/deafness, status epilepticus, ictus during sleep or resting and interictal neurological abnormalities. After correction the *P* values using Bonferroni-correction for multiple variables (ictal and postictal signs) ictal mydriasis and postictal blindness/deafness were not significantly different (Table 3).

Discussion

In our study, 47/125 cats (38%) were suffering from PE. Quesnel et al found that all cats with seizures have structural brain disease, which led to the assumption for period of time that PE occurs only very rarely in cats.[23] However, PE was diagnosed in a high percentage of cats suffering from seizures by several other authors: Schwarz-Porsche 59%, Cizinauskas et al 21.4%, Rusbridge 54%, and Schriefl et al 25%.[21,22,25,26] The prevalence of PE in cats is thought to be lower than in dogs; however, only one author[26] has previously compared the prevalence in the two species at the same clinic. We found that PE in cats is proportionally less frequent (38%) than in dogs (48%),[27] which is similar

Table 1. Classification and cause of seizures in cats ($n = 125$).

Cause of seizure	Number of cats	%
PE	47	37.6
Brain neoplasia Meningioma (9), oligodendroglioma (3), spindle cell tumour (1), lymphoma (1), mass in MRI (1), astroglioma (1)	16	12.8
Hippocampal necrosis	14	11.2
Toxicosis Permethrin (4), inhibitors of acethycholinesterase (4)	8	6.4
Encephalitis Unknown origin (5), feline infectious peritonitis (2)	7	5.6
Hypoxia Severe anaemia (2), severe lungs oedema (2), Narcosis induced (1)	5	4
Brain degeneration of unknown origin	4	3.2
Erytrocytosis	4	3.2
Renal insufficiency	3	2.4
Hyperthyroidism	3	2.4
Hypertension	2	1.6
Brain trauma	1	0.8
Ischaemic encephalopathy	1	0.8
Other extracranial disorders Hyperosmolality (2), hepatic encephalopathy (2), diabetic ketoacidosis (2), ileus (1), hypoglycaemia (1)	8	6.4
Total	125	100%

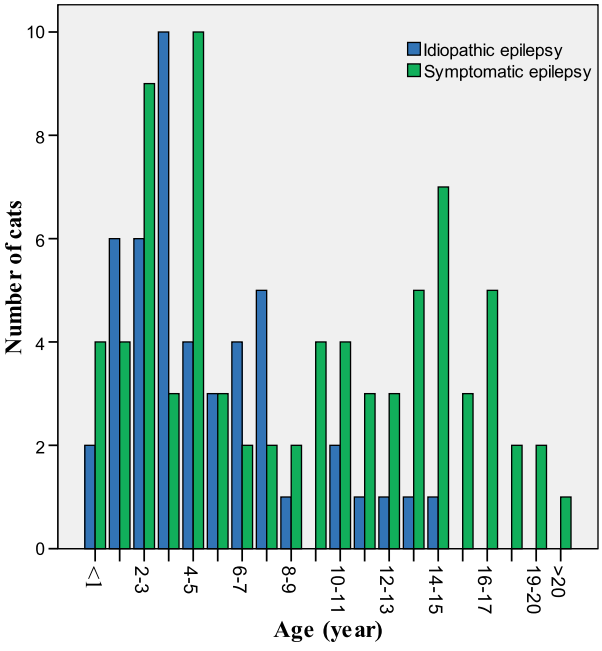

Fig 1. Age at onset of first seizure and number of cats with idiopathic and symptomatic epilepsy.

to the findings of Rusbridge[26] of 54 and 68%, respectively. Our data support the opinion that PE should be considered as an important differential diagnosis in cats with recurrent seizures.

The diagnosis of PE is typically tentative as at this time it is not possible to confirm the diagnosis PE other than by elimination of possible aetiological factors.

In the previous report on feline epilepsy only 11/91 cats (12%) were examined with CT or MRI and compared to 34/125 cats (27%) in the present study.[21] More extended use of advanced diagnostic imaging, particularly MRI might influence the diagnostic results, as it might detect more lesions and lead to the diagnoses of a higher proportion of SE; however, even in high-field MRI, small cerebrovascular or inflammatory lesions may not be visible which means if the aetiology cannot be identified by the diagnostic work-up it does not necessarily mean that the epilepsy is primary. This inherent limitation in the diagnostic of PE will not be changed until other, eg, genetic tests, will be accessible. That is why one of selection criteria was that cats without full work-up need to be re-examined at least 12 months after the first seizure in order to minimise the chance of false diagnoses. We used these criteria mainly for cats

Table 2. Statistical results of age, body weight and duration of seizure for PE and SE groups.

Variable	PE group		SE group		*t*	*P*
	M	SD	*M*	SD		
Age	4.62	3.43	8.42	5.73	4.129	<0.0001
Body weight	5.14	1.63	4.03	1.41	2.713	0.009
Duration of seizure	1.52	1.09	2.42	2.46	2.364	0.02

M = mean, SD = standard deviation.

where the diagnostic work-up was not complete with diagnostic imaging. This may have added some bias, as older cats not surviving for 12 months may be excluded. We also cannot rule out the possibility that some cats had mild self-limiting meningoencephalitis, vascular or degenerative disease despite a negative work-up and unremarkable results in repeated neurological examinations. By excluding such cats from the PE group due to the lack of complete diagnostic work-up, we would lose a very important group of patients that is often seen in the practice. Such cats have occasional seizures but otherwise are in good clinical condition. On the other hand, the owners of such cats may tend to use less intensive veterinary service and may not undergo complete diagnostic work-up. Contrary to that, the owner whose cat has not only seizures but obvious neurological deficits too may tend to use more complete diagnostic work-up. Thus, we think that the exclusion of these patients would falsify our results.

Another limitation of the diagnostic imaging is that borderline changes (for example, mildly enlarged ventricle) may have or may not have epileptogenic function. Furthermore mild hyperintensity of the

Table 3. Comparison of sex, type of seizure, ictal and postictal signs, ictal characteristic and interictal neurological abnormalities between PE and SE groups*.

Main variable	Parameter	PE		SE		*P*
		N	%	*N*	%	
Sex	Male	27	57.4	45	57.7	0.979
	Female	20	42.6	33	42.3	0.979
Type of seizure	Focal seizure	5	10.6	3	3.8	0.133
	Generalised seizure	20	42.6	43	55.1	0.173
	Focal and generalised seizure	22	46.8	32	41.0	0.527
Ictal signs	Tremor	26	55.3	47	60.3	0.587
	Rapid running	19	40.4	10	12.8	0.0004
	Facial twitching	20	42.6	27	34.6	0.375
	Vocalisation	14	29.8	18	23.1	0.529
	Urination	22	46.8	23	29.5	0.182
	Defecation	6	12.8	13	16.7	0.354
	Mydriasis	33	70.2	35	44.9	0.032†
	Salivation	28	59.6	35	44.9	0.260
Postictal signs	Aggression	14	29.8	20	25.6	0.887
	Polyphagia	6	12.8	3	3.8	0.115
	Polydipsia	4	8.5	2	2.6	0.213
	Blindness/deafness	11	23.4	6	7.7	0.045†
	Ataxia	20	42.6	22	28.2	0.404
Cluster seizures		25	53.2	46	59.0	0.449
Status epilepticus		9	19.1	31	40.0	0.013
Ictus during sleep or resting		14	29.8	3	3.5	0.003
Interictal neurological abnormalities		2	4.3	57	73.1	<0.0001

*The alpha-level was adjusted for multiple testing within the main variables using Bonferroni-correction. For ictal signs the corrected alpha level = 0.007; for postictal signs 0.0125.
†Not significant after alpha adjustment.

temporal lobe cannot be determined as cause or consequence of seizures.

The most common cause of SE in our study was intracranial neoplasia (13%). This is not surprising as intracranial neoplasia was reported to be a frequent aetiology for feline seizures.[21] Brain neoplasia was also the most frequent aetiology in dogs with seizures in our hospital.[27] The most frequent brain neoplasia in the present study was meningioma, as in the study by Schriefl et al.[21] Interestingly, lymphoma was only diagnosed in one cat, although another study found lymphoma a frequent neoplasia as seizure aetiology.[28]

Feline hippocampal necrosis (FHN) was originally described as the seizure aetiology in detail in Switzerland.[29] Since the original report, other authors also observed FHN in connection with seizures.[30–32] In our study FHN was a frequent aetiology (11%). The frequency of FHN to be the aetiology varies considerably among different reports. Cizinauskas et al[25] reported FHN to be the most frequent aetiology of seizures at 25%. In contrast, Schriefl et al[21] found FHN in only 2/91 cases and other authors did not even mention FHN among causes of seizure.[23]

The big difference regarding the incidence of PE in cats may also be associated partly with uncertainness in the assessment of hippocampal changes. This may be a cause and consequence of seizure as well. Even the International League Against Epilepsy could not determine whether 'hippocampal sclerosis (HS) is a non-specific result of primary epileptogenic lesion and not in itself epileptogenic or secondary to the primary epileptogenic lesion but also epileptogenic or a primary HS that coexists with other epileptogenic lesions'.[33] The authors included FHN as a possible aetiology of SE similar to a recent review on feline seizure diseases.[4]

The age of onset of canine epilepsy is an important piece of information and is helpful for the diagnosis of PE, which typically occurs between 1 and 5 years of age,[1,27,34] but only Schriefl et al evaluated age from this point of view in cats.[21] In our study the mean age of onset was different between PE and SE. The mean age in the PE and SE groups (4.6/8.4) was very similar to the findings of this latter study (3.5/8.2) in that cats with PE were younger. Parent and Quesnel (1996) and Bailey and Dewey (2009) also reported similar findings.[4,35] Our observations suggest that if the first seizure occurred after 7 years of age, SE is more likely and complete diagnostic work-up with advanced diagnostic imaging is strongly recommended.

In dogs PE is assumed to be usually genetic in origin but no information supports such an assumption in cats. Our study is the first indicating that some breeds may have predisposition for PE. Although the European shorthair was slightly overrepresented, the low significance is no proof for a genetic origin.

Historically, focal seizures have been associated with structural brain disease,[23] but in agreement with a more recent opinion of authors we could not confirm these findings.[4,21]

Mydriasis during ictus and blindness or deafness during the postictal phase were slightly more frequent in the PE group. This may be connected with the more accurate observations made by the owners of cats with PE due to the longer observation period and higher number of observed seizures, in some cases over a period of years. The occurrence of other ictal signs such as tremor, facial twitching, vocalisation, urination, defecation, salivation, and postictal signs (aggression, polyphagia, polyuria/polydipsia (PU/PD), ataxia) did not differ between the two groups. Therefore, these signs did not allow any clinical differentiation between PE and SE.

Wild running behaviour is often reported earlier in feline seizure diseases.[4,22,35,36] Interestingly, we observed this more frequently in PE than in SE. Such running behaviour in connection with seizure has been observed in epileptic rats. It is presumed that specific neuronal circuits of the deep layers of rostral colliculus play a major role in the production of running behaviour.[37] These neuronal circuits may be functionally intact more often in PE than in SE. Ambulatory automatism such as circling, walking or running can be observed in human temporal lobe epilepsy.[38] The rapid running may indicate temporal lobe involvement in cats. We could not exclude the possibility that this is a fearful behavioural response to the seizure in cats and not an ictal phenomenon itself.

Seizures were more frequently reported during resting conditions in the PE. Also in dogs, seizures commonly occur during sleep or under resting conditions.[1,12,20,27,39] An increase in cortical neuronal synchronisation takes place during sleep possibly decreasing the seizure threshold.[40] This effect is probably less important if an underlying disease (SE) exists, because impact of this disease on seizure threshold may not depend on the time of day.

Our study has similar limitations to previous clinical studies. There is an inherent limitation in the classification of epilepsy. Not only that PE cannot be confirmed but the confirmation of SE frequently seems to be problematic. This is particularly true for cases with erytrocytosis, renal insufficiency, hyperthyroidism and hypertension. These conditions commonly occur in cats and could be coincidence as well. However, the included cases were severe enough to cause seizures.

Erytrocytosis was diagnosed in four cases. The initial haematocrit was considered very high 61, 67, 68, 74%. The suspected diagnosis was renal neoplasia in one case and polycitaemia vera (PCV) in three cases. The follow-up period was over 3 years in two cases of PCV with hydroxiurea treatment and occasionally phlebotomia. Seizures were never observed in all these cases as the haematocrit was under 55%. Based on these findings, erytrocytosis can be considered as the cause of epilepsy.

Renal insufficiency was as severe as stage III in three cases according to IRIS system. These three cases were presented with chronic history of weight loss,

PU/PD and acute seizures. The histopathology supported renal encephalopathy in two examined cases and no other brain lesion could be detected. One further cat had evidence of diabetic ketoacidosis and stage II chronic renal insufficiency.

The aetiological connection between recurrent seizures and hyperthyroidism or hypertension is questionable in this study. However, all five cats in these groups showed evidence of more than one medical problem which can result in recurrent seizures. We categorised three cats as having hyperthyroidism based on increased serum T_4. All cats showed further evidence of thyrotoxic cardiomyopathy, in one case histopathology confirmed thyroid gland adenoma without structural brain damage. Two cats categorised with hypertension (systolic blood pressure over than 210 mmHg) showed recurrent seizures for several days, and stage II chronic renal insufficiency. One cat showed blindness and retinal detachment. We think that these cases may have had inherently lower seizure threshold and some tendency to epilepsy as these disorders could elicit seizures; however, the classification as PE in such cases is contradictory and not logical.

Based on our study, clinical examinations are one of the most important indicators allowing us to distinguish between PE and SE.[41] The clinical and neurological examinations were suggestive of SE in 73% of cases in the SE group. As postictal behavioural changes could be observed occasionally, the examination should be repeated when the patient is believed to have fully recovered.[4] As analysis of the ictus was based on the owners' reports, it should be considered with caution. More precise semiology of seizures based on video observation would increase the objectivity of clinical observations.

Conclusion

In conclusion, PE is an important aetiology for seizure in cats (47/125, 38%), however, in comparison with dogs it is less frequent. SE was mostly caused by intracranial tumours (16/125, 12.8%) and hippocampal necrosis (14/125, 11.2%). Infectious disease was rarely diagnosed (2/125, 1.6%). As for dogs, it was not possible to differentiate between PE and SE based on the ictal signs alone. Status epilepticus, altered interictal neurological status and seizure onset over the age of 7 years were highly indicative of SE. If the seizures occurred during resting conditions and rapid running occurred the diagnosis was more likely to be PE than SE.

Acknowledgements

We would like to thank the staff of the Clinic of Diagnostic Imaging and Institute of Pathology for their diagnostic assistance. Furthermore we are grateful to P Karlhuber for her help in collecting the data.

References

1. Thomas CW. Seizures and narcolepsy. In: Dewey CW, ed. A practical guide to canine and feline neurology. Ames: Iowa State University Press, 2003: 193–212.
2. Podell M. Seizures. In: Platt SR, Olby NJ, eds. BSAVA manual of canine and feline neurology. 3rd edn. Gloucester: BSAVA British Small Animal Veterinary Association, 2004: 97–112.
3. Cunningham JG, Farnbach GC. Inheritance and idiopathic canine epilepsy. *J Am Anim Hosp Assoc* 1987; **23**: 421–4.
4. Bailey KS, Dewey CW. The seizuring cat. *J Feline Med Surg* 2009; **11**: 385–94.
5. Koestner A, Rehfeld CE. Idiopathic epilepsy in a beagle colony. *ANL Rep* 1968; 178–9.
6. Van Der Velden NA. Fits in tervueren shepherd dogs: a presumed hereditary trait. *J Small Anim Pract* 1968; **9**: 63–9.
7. Falco MJ, Barker J, Wallace ME. The genetics of epilepsy in the British Alsatian. *J Small Anim Pract* 1974; **15**: 685–92.
8. Srenk P, Jaggy A, Gaillard C, Busato A, Horin P. Genetische grundlagen der idiopatischen epilepsie beim Golden Retriever. *Tierarztl Prax* 1994; **22**: 574–8.
9. Hall SJG, Wallace ME. Canine epilepsy: a genetic counselling programme for Keeshonds. *Vet Rec* 1996; **138**: 358–60.
10. Famula TR, Oberbauer AM, Brown KN. Heritability of epileptic seizures in the Belgian Tervueren. *J Small Anim Pract* 1997; **38**: 349–52.
11. Heynold Y, Faissler D, Steffen F, Jaggy A. Clinical, epidemiological and treatment results of idiopathic epilepsy in 54 Labrador Retrievers: a long-term study. *J Small Anim Pract* 1997; **38**: 7–14.
12. Jaggy A, Bernardini M. Idiopathic epilepsy in 125 dogs: a long-term study. Clinical and electroencephalographic findings. *J Small Anim Pract* 1998; **39**: 23–9.
13. Kathmann I, Jaggy A, Busato A, Bärtschi M, Gaillard C. Clinical and genetic investigations of idiopathic epilepsy in the Bernese Mountain Dog. *J Small Anim Pract* 1999; **40**: 319–25.
14. Nielen ALJ, Janss LLG, Knol BW. Heritability estimation for diseases, coat color, body weight, and height in a birth cohort of Boxer. *Am J Vet Res* 2001; **62**: 1198–206.
15. Patterson EE, Mickelson JR, Da Y, et al. Clinical characteristics and inheritance of idiopathic epilepsy in Vizslas. *J Vet Intern Med* 2003; **17**: 319–25.
16. Patterson EE, Armstrong PJ, OBrien DP, Roberts MC, Johnson GS, Mickelson JR. Clinical description and mode of inheritance of idiopathic epilepsy in English Springer Spaniels. *J Am Vet Med Assoc* 2005; **226**: 54–8.
17. Casal ML, Munuve RM, Janis MA, Werner P, Henthorn S. Epilepsy in Irish Wolfhounds. *J Vet Intern Med* 2006; **20**: 131–5.
18. Croft P. Fits in dogs: a survey of 260 cases. *Vet Rec* 1965; **77**: 438–45.
19. Palmer AC. Pathological changes in the brain associated with fits in dogs. *Vet Rec* 1972; **90**: 167–73.
20. Podell M, Fenner WR, Powers JD. Seizure classification in dogs from a non-referral-based population. *J Am Vet Med Assoc* 1995; **206**: 1721–8.
21. Schriefl S, Steinberg TA, Matiasek K, Ossig A, Fenske N, Fischer A. Etiologic classification of seizures, signalment, clinical signs and outcome in cats with seizure disorders: 91 cases (2000–2004). *J Am Vet Med Assoc* 2008; **233**: 1591–7.
22. Schwartz-Porsche D, Kaiser E. Feline epilepsy. *Probl Vet Med* 1989; **1**: 628–49.

23. Quesnel AD, Parent JM, McDonell W, Percy D, Lumsden JH. Diagnostic evaluation of cats with seizure disorders: 30 cases (1991–1993). *J Am Vet Med Assoc* 1997; **210**: 65–71.
24. Barnes HL, Chrisman CL, Mariani CL, Sims M, Alleman AR. Clinical signs, underlying cause, and outcome in cats with seizures: 17 cases (1997–2002). *J Am Vet Med Assoc* 2004; **225**: 1723–6.
25. Cizinauskas S, Fatzer R, Schenkel M, Gandini G, Jaggy A. Can idiopathic epilepsy be confirmed in cats? *J Vet Intern Med* 2003; **17**: 246 [abstract].
26. Rusbridge C. Diagnosis and control of epilepsy in the cat. *In Pract* 2005; **27**: 208–14.
27. Pakozdy A, Leschnik M, Tichy A, Thalhammer JG. Retrospective clinical comparison between idiopathic versus symptomatic epilepsy in 240 dogs with seizures. *Acta Vet Hung* 2008; **56**: 471–83.
28. Tomek A, Cizinauskas S, Doherr M, Gandini G, Jaggy A. Intracranial neoplasia in 61 cats: localisation, tumor types and seizure pattern. *J Feline Med Surg* 2006; **8**: 243–53.
29. Fatzer R, Gandini G, Jaggy A, Doherr M, Vandevelde M. Necrosis of hippocampus and piriform lobe in 38 domestic cats with seizures. A retrospective study on clinical and pathologic findings. *J Vet Intern Med* 2000; **14**: 100–4.
30. Brini E, Gandini G, Crescio I, Fatzer R, Casalone C. Necrosis of hippocampus and piriform lobe: clinical and neuropathological findings in two Italian cats. *J Feline Med Surg* 2004; **6**: 377–81.
31. Schmied O, Scharf G, Hilbe M, Michal U, Tomsa K, Steffen F. Magnetic resonance imaging of feline hippocampal necrosis. *Vet Radiol Ultrasound* 2008; **49**: 343–9.
32. Pakozdy A, Kneissl S, Gruber A, et al. Therapy and follow-up in 7 cats with suspected hippocampal necrosis. In: 22th ECVN/ESVN Congress; 2009 Sept 25–26; Bologne, Italy. 2009: 65.
33. Wieser HG. ILAE Commission on Neurosurgery of Epilepsy. ILAE Commission Report. Mesial temporal lobe epilepsy with hippocampal sclerosis. *Epilepsia* 2004; **45**: 695–714.
34. Podell M. Seizures in dogs. *Vet Clin North Am Small Anim Pract* 1996; **26**: 779–825.
35. Parent JML, Quesnel AD. Seizures in cats. *Vet Clin North Am Small Anim Pract* 1996; **26**: 811–25.
36. Kline KL. Feline epilepsy. *Clin Tech Small Anim Pract* 1998; **13**: 152–8.
37. Faingold CL, Randall ME. Neurons in the deep layer of superior colliculus play a critical role in the neuronal network for audiogenic seizures: mechanisms for production of wild running behaviour. *Brain Res* 1999; **815**: 250–8.
38. Shorvon SD. Handbook of epilepsy treatment. 1st edn Oxford: Blackwell Science, 2000; 5.
39. Lengweiler C, Jaggy A. Klinische, epidemiologische und therapeutische Aspecte der idiopatischen Epilepsie bei 25 Golden Retrievern: Resultate einer Langzeitstudie. *Schweiz Arch Tierheilkd* 1999; **5**: 231–8.
40. Tanaka T, Naquet R. Kindling effect and sleep organization in cats. *Electroencephalogr Clin Neurophysiol* 1975; **39**: 449–54.
41. Jaggy A. Krampfanfälle. In: Jaggy A, ed. Neurologische Notfälle beim Kleintier. Stuttgart: Enke, 1997: 20–31.