Bit-mapped quantitative EEG analysis in a tiger (*Tiger felis*) with partial seizures: a case report

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Summary

We report electroencephalographic findings in an anesthetized 4-month-old female drug-naive tiger (*Tiger felis*) affected by partial seizures with secondary generalization. Both clinical signs and electroencephalographic abnormalities were consistent with a forebrain lesion. Recurring epileptiform activity was noted in the left frontal, central and temporal derivations upon visual inspection of the electroencephalogram (EEG). A quantified EEG, displayed on brain maps, showed the predominance of slow rhythms. As regards the absolute power, a prevalence of left frontal-temporal activity was noted. An infectious or inflammatory condition was thought to be the most probable cause of the symptomatic epilepsy in our patient. Unfortunately, other differential diagnoses could not be ruled out.

KEY WORDS: Epilepsy, q-EEG, tiger.

Introduction

Ideally, brain electrical activity would be evaluated with no other variables, such as anesthesia, to consider. However, anesthesia is sometimes necessary in order reliably to perform diagnostic tests in animals (1), especially when pain, anxiety or aggressive behavior is present. Although not used routinely in veterinary clinical practice, the electroencephalogram (EEG) is a useful and non-invasive investigative tool in patients with neurological disease and especially epilepsy. In addition, the quantitative EEG (q-EEG) can be of clinical assistance in diagnosing various conditions affecting brain function (2). Unfortunately, this kind of experience in veterinary medicine is lacking. The aim of this study was to assess a q-EEG in an anesthetized drug-naive tiger (*Tiger felis*) affected by partial seizures with secondary generalization.

Case description

A 4-month-old female tiger (*Tiger felis*) was referred to our practice with a two-month history of episodic circumscribed to the right and behavioral disorders followed by generalized tonic-clonic seizures. The length of observed seizures was between thirty seconds and one minute. Initially, depression, pacing, circling and blindness was observed interictally, as a result of a partial epileptic status. Later, aggressive, self-mutilating attacks involving the left front limb, resulting in self-inflicted wounds, were noticed. Interestingly, these behavioral changes were exacerbated during feeding, while no aggressiveness was displayed towards other animals or humans. Neurological abnormalities were noticed shortly after vaccination with a combined, attenuated panleucopenia-rhinotracheitis-calicivirus vaccine. The patient was born of a consanguineous mating and generalized seizures were reported in one of her male littermates. At the time of clinical examination, the patient was cachectic, unable to feed herself, indifferent to her environment, drug-naive and showed no generalized seizures. The tiger was severely depressed, somewhat disoriented and appeared to be bilaterally blind. When she moved, compulsive walking, pacing, slight hypermetria and circling to the right were noticed. Postural reactions, especially in the rear, were bilaterally decreased. All spinal reflexes and perception of noxious stimuli were normal. Menace reaction and physiological nystagmus were absent on both sides and convergent strabismus was present bilaterally. On the basis of the clinical evaluation, she was presumed to have a forebrain lesion involving the limbic system. Possible differential diagnoses were meningoencephalitis, metabolic encephalopathy, brain anomaly, cerebral infarction, trauma and neoplasia. Blood hematology and serum biochemistry profile were performed. Serology for *Toxoplasma gondii*, *Neospora caninum* and Distemper virus was assessed.

Methods

To perform the EEG recording, the animal was sedated using medetomidine (Domitor, Orion; 20 µg/kg i.m.). After five minutes, a propofol bolus (Diprivan, Zeneca; 5 mg/kg i.v.) was given to induce anesthesia. Then the patient was intubated for oxygen administration (10 ml/kg/min) and placed in sternal recumbancy for place-
ment of the electrodes. Lactated Ringer’s solution was administered i.v. at a rate of 10 ml/kg/h. Anesthesia was maintained for 15 minutes by continuous propofol infusion (0.2-0.5 mg/kg/min i.v.). A 17-channel monopolar montage (F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, O2; reference: on the bridge of the nose; ground: caudal to the external occipital protuberance; sensitivity = 10 µV/mm, time constant = 0.1 s, HF = 30 Hz, impedance < 3 Kohm) was used for EEG recording (3). Electrocardiogram and respiration rate were recorded via polygraphic EEG electrodes (X1 and X2 respectively) connected to alligator clips and a volumetric transducer applied to the chest. Recording parameters were the same as those of the EEG, except for X1 sensitivity (50µV/mm). EEG recording was started within five minutes of intubation, lasted for 10 minutes and was stored in an acquisition station (Galileo Planet, Esaote). Before performing the quantitative analysis of the EEG, visual inspection of the EEG traces was performed. The quantitative analysis of the EEG (4) was performed using a server (Galileo Star, Esaote). The spectral bands of δ (2.0-4.0 Hz), θ (4.5-8.0 Hz), α (8.5-12.0 Hz) and β (12.5-30.0 Hz), to be used in the analysis, were selected. Two-second artifact-free EEG epochs were selected for examination and non-suppressed epochs with epileptiform activity (A: total 150), epochs with generalized paroxysmal activity (B: total 50), and epochs with burst suppression activity (C: total 20) were analyzed. No significant samples of non-suppressed epochs without epileptiform activity were available in the EEG. The data derived expressed the absolute power of the bioelectrical activity (µV²). Topographic maps were produced by the server.

The suboccipital cerebrospinal fluid (CSF) tap was performed after the EEG session. After recovery from the general anesthesia, phenobarbital (2.5 mg/kg p.o. bid) therapy was started. Prednisolone (2 mg/kg p.o. sid) was given for one week and later continued in decreasing dosages for two weeks to reduce suspected central nervous system inflammation. Additionally, an intensive feeding programme was started.

Results

Blood hematology and serum biochemistry profile (including ammonia and bile acids) were normal. Serology for Toxoplasma gondii, Neospora caninum and Distemper virus was negative. Visual inspection of the EEG revealed a frequent and diffuse burst suppression pattern, which can be considered pharmacologically induced (Fig. 1a). Yet, corresponding with non-suppressed periods, recurring epileptiform activity (mean frequency: 5 Hz; mean amplitude: 19.8 µV) was present in all the derivations. This paroxysmal activity, consisting of sharp waves, spike- and polyspike-wave complexes, appeared more prominent in the left hemisphere. On the contrary, a lower amplitude of the diffuse “burst-suppression” pattern could be noted in the right hemisphere.

![Figure 1](image_url)
The q-EEG showed the prevalence of δ and θ slow rhythms. Faster α and β rhythms, which can be ascribed either to spindle activity (8-12 Hz) or to paroxysms (sharp waves and spike-wave complexes), were also displayed on quantitative brain maps (Fig. 1b). These mostly offer information about the topographic distribution of the frequency content, showing, as regards the absolute power, a prevalence of the left frontal-temporal activity.

Mean absolute power values recorded from left and right homologous electrode sites in the different sets of analyzed epochs are summarized in Table I, and suggest an asymmetrical distribution of the frequency content between the two hemispheres. In addition, δ-bands and, to a slightly lesser degree, θ-bands could include both the slow background activity ascribable to the pharmacological actions of the anesthetic used and the slow components of the paroxysmal activities. Conversely, faster frequency bands could be mostly affected by the fast components of the interictal paroxysmal activity (sharp waves, spike-waves, polyspike-wave complexes).

CSF analysis revealed increased protein concentration (Pandy test = +) and normal cell count was found. Bacteriological CSF examination was negative. After one month of the above described therapy, the animal presented a reduced number of seizures, achieved a body weight comparable to that of her litter-mates, and regained her vision and normal social life. Phenobarbital blood concentration measured one month after the start of treatment was within the therapeutic range (24 mg/dl). At the time of submission of this manuscript, the mean time between two epileptic seizures was two months and phenobarbital therapy was still being continued at the same dosage.

**Discussion**

Diagnostic EEG recordings under propofol anesthesia have been performed in dogs with idiopathic epilepsy (5), and the paroxysmal discharges detected on the EEG were believed to be consistent with the interictal epileptic activity. Yet, EEG burst suppression has been reported under propofol anesthesia both in the dog (6) and in the cat (7), suggesting that the EEG findings are propofol dose-related. Additionally, in veterinary medicine, propofol has been used successfully to control seizures occurring after surgical treatment of portosystemic shunts in dogs and cats (8) and refractory seizures of intracranial origin (9). Unfortunately, neither data on propofol anesthesia in the tiger, nor the EEG database of the background activity of healthy subjects of the same species, are as yet available.

Visual inspection of the EEG showed a frequent pattern of pharmacological induction (7); however, recurring epileptiform activity was detected throughout the EEG recording, frequently appearing in the non-suppressed period of the raw EEG, particularly in the left hemisphere. Conversely, a lower amplitude of the diffuse “burst suppression” pattern could be noted in the right hemisphere, mostly affecting frontal, central and anterior-temporal areas.

It has been reported in veterinary literature that, in adult cats (10), and also in one old African lion (11), a naturally occurring disease syndrome causes acute destruction of the cerebral tissue, by an extensive ischemic necrosis. The signs of cerebral disturbance reported in these cases were often unilateral, the animals showed seizures and these lesions were usually in the frontal lobe or rostral thalamus with involvement of the limbic system. The hematological profiles of these subjects were normal, and CSF tap often showed a mildly raised protein level, with little to no cell accumulation. Yet, scintigraphy and EEG only sometimes indicated the localization of the lesion. Unfortunately, no data on similar syndromes in tigers are reported and neuroimaging and histopathological examination of our patient was not possible because the owner refused to allow further diagnostic work-up.

However, the age of our patient would suggest other neuropathologies, such as an inflammatory process, rather than an ischemic encephalopathy (10). With regard to pets affected by complex partial seizures, a recent study on the EEG of epileptic dogs (12) reported “focal low frequency patterns without

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**Table I - Absolute power (µV²) mean values recorded from left and right homologous electrode sites in the different sets of analyzed epochs**

<table>
<thead>
<tr>
<th>Frequency bands</th>
<th>A (µV²)</th>
<th>B (µV²)</th>
<th>C (µV²)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Left</td>
<td>Right</td>
<td>Left</td>
</tr>
<tr>
<td>δ 2.0 - 4.0 Hz</td>
<td>80.22</td>
<td>46</td>
<td>94.6</td>
</tr>
<tr>
<td>θ 4.5 - 8.0 Hz</td>
<td>46</td>
<td>24.4</td>
<td>64.3</td>
</tr>
<tr>
<td>α 8.5 - 12.0 Hz</td>
<td>20.1</td>
<td>9.5</td>
<td>28.5</td>
</tr>
<tr>
<td>β 12.5 - 30 Hz</td>
<td>14.9</td>
<td>8.3</td>
<td>21.5</td>
</tr>
</tbody>
</table>

A: non-suppressed epochs with epileptiform activity; B: epochs with generalized paroxysmal activity; C: epochs with burst suppression activity.
spikes" in one dog with complex partial seizures, but no data on felines are yet available.

The EEG spectral analysis performed on our patient was not intended to substitute the traditional visual interpretation of the raw data, because it should be never diagnostic. q-EEG can only provide information that contributes to a diagnosis, along with other available clinical information. This is an extension of the old rule that one never diagnoses epilepsy from the EEG alone. The EEG spectral analysis performed on this patient was just an attempt to extend this specific analysis and data representation technique to a wild animal, in full awareness that further long-term studies need to be performed to achieve greater knowledge of and more precise information on the EEG of "non-conventional animal species".

The diffuse EEG pattern and CSF results suggest that an infectious or inflammatory condition was the most probable cause of the symptomatic epilepsy in this tiger. Unfortunately, due to the owner’s refusal to allow further investigations, we were not able to exclude other differential diagnoses, such as brain anomaly, cerebral infarction, post-traumatic and space-occupying lesions including neoplasia. It is our hope that this case report (unusual, given the species involved) could provide a stimulus for further investigations.

References