

Approach to the diagnosis of seizures

Part 2: Diagnostic procedures

Part 1: Epileptic and nonepileptic seizures was published in Focus™ Volume 8 No 4

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KEY POINTS

- Paroxysmal events can have many different causes, most of them resulting from epilepsy.
- Epileptic seizures are the result of paroxysmal, bioelectric functional disturbances of the brain.
- These functional disturbances can have both cerebral and extracerebral causes.
- Extracerebral causes can be metabolic or toxic. They affect the brain's metabolism, and therefore the whole brain, leading to generalized seizures.
- Cerebral seizures also have various causes and can lead to focal and generalized seizures.
- An optimal therapy and reliable prognosis can be achieved only from a definitive diagnosis.

DIAGNOSTIC PROCEDURES

The goal of diagnosis is an etiological distinction between the different seizure disorders – only then is prognosis reliable and successful treatment possible. One has to distinguish between epileptic and nonepileptic seizures, and between extracerebral and primary cerebral seizure disorders, and to recognize early progressive and still active brain diseases. The diagnosis of epilepsy can be made only by exclusion. In addition, one should distinguish between symptomatic and idiopathic epilepsy. Using the diagnostic measures listed in **Table 1** and the flow chart in **Figure 1**, it should be possible to make such a diagnosis.

CASE HISTORY

A carefully documented case history, which delves into the past, is of cardinal importance (**Table 2**) (1–5). The **breed** and **age** alone can be of etiological significance, since seizures can occur as a result of congenital brain diseases – for example, the congenital hydrocephalus of certain miniature breeds such as Maltese, Chihuahuas, and Yorkshire Terriers (2, 4). This is also true of brachycephalic dogs that, with increasing age, show a disposition for brain tumors and, therefore, for epileptic seizures (4).

Age-dependence can signify both extracerebral and cerebral causes of seizures (**Table 3**). In general, idiopathic epilepsy manifests itself between the ages of 6 months and 5 years. Reports in the literature differ somewhat (1–9). In some breeds the manifestation is apparent early in life, with others it is later (7, 9), suggesting a genetic disposition. Early manifestation occurs when there has been a high rate of inbreeding (7, 10). Age-dependence is not a factor in symptomatic epilepsy, which occurs frequently before the age of 1 year and after the 7th year (2).

Immunization status, previous diseases, and trauma can provide an indication of postencephalitic or posttraumatic epilepsy, while a previous tumor operation can suggest cerebral metastasis. Illness of the mother during pregnancy, difficulties during the birth, and very early diseases can be the origin of pre-, peri-, and postnatal brain lesions. Certain **medications** (for example, phenothiazine) lower the seizure threshold, which may contribute to seizure manifestation. **Related dogs with epilepsy**, an increasing number of epileptics in the breeding line or in the breed itself, confirm or suggest a hereditary origin.

The **seizure history** is particularly important. Idiopathic epilepsy usually begins with a single generalized seizure; the initial interictal periods can last for weeks or months (2, 3, 5, 9). In most cases the frequency of seizures gradually increases, although this can vary widely between individuals (5, 9). Some breeds (Irish Setters and German Shepherd Dogs, among others) increasingly suffer from clusters of seizures – several seizures over a period of a few hours to 2 days. A focal seizure start, a short first interictal interval (2), a quick increase in frequency (11), the presence of several seizure types, and a change in the duration of seizures indicate active morphological changes in the brain. In cases of progressive brain disease, the seizures frequently manifest themselves as a cluster of seizures or as *status epilepticus*.

In seizure disorders of extracerebral etiology the seizure event can vary greatly: with nonepileptic seizures there is no postictal stage and there are seldom vegetative symptoms (urination or defecation) (2, 3).

The **association of the seizure** with activity, time of day, and



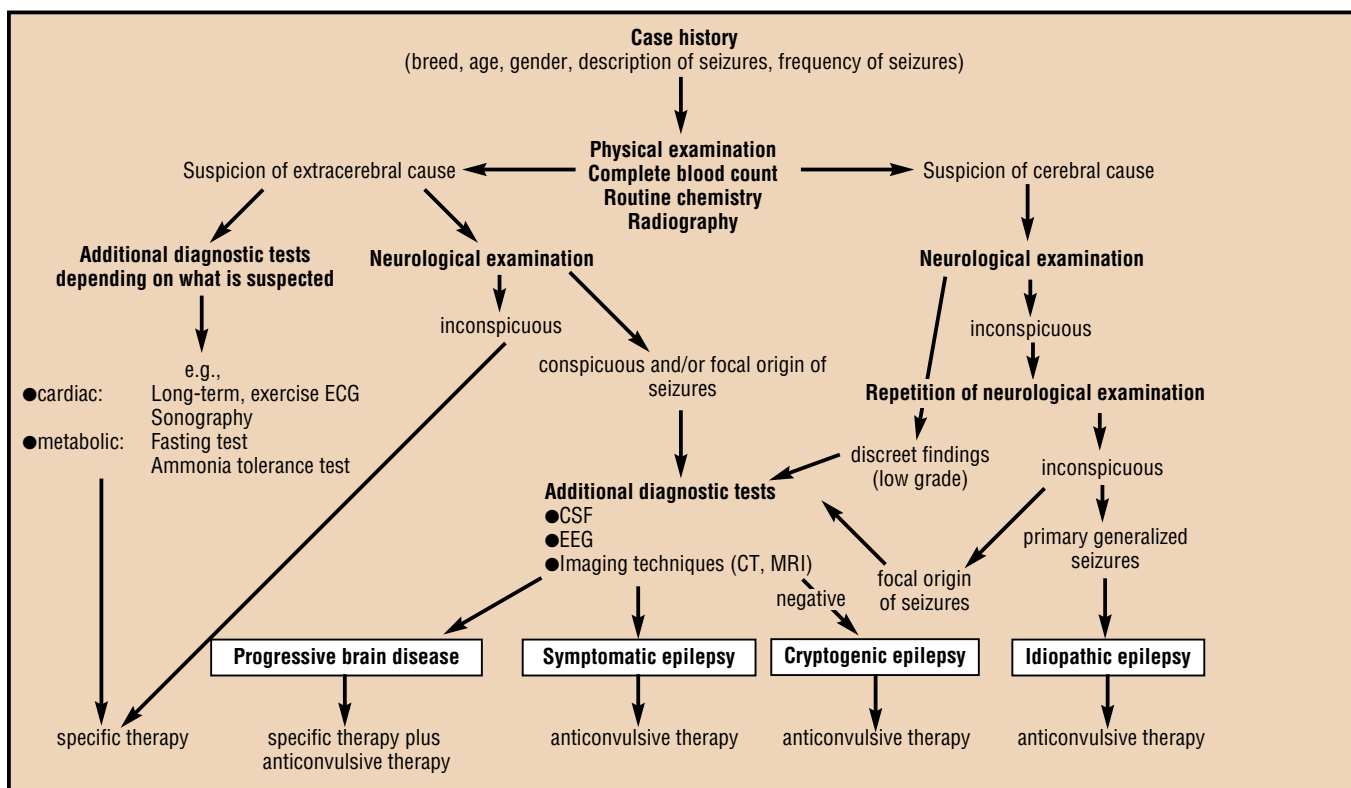


Figure 1 Algorithm illustrating a diagnostic approach to seizures.

eating is of diagnostic importance. In idiopathic epilepsy many seizures occur while the animal is sleeping or resting, and usually while it is at home. This association is less frequent in symptomatic epilepsy, and it is usually absent with seizures resulting from progressive brain disease. Pre- and postprandial seizures may be an indication of metabolic disorder, especially hypoglycemia resulting from insulinoma. Seizures that occur on excitement or physical exertion can also be hypoglycemic in origin. They can also point to cardiac causes. Even with idiopathic epilepsy, taxing experiences can lead to a seizure, although usually only when the animal has quieted down.

No **interictal symptoms** are seen in idiopathic epilepsy. They may be present, although only in a mild form, in symptomatic epilepsy. In progressive and still-active brain disorders there are usually interictal CNS symptoms. In extracerebral seizure disorders, depending on the organ affected, more or less distinct interictal symptoms can be observed.

CLINICAL, HEMATOLOGICAL, AND BIOCHEMICAL INVESTIGATIONS

Physical and cardiological examination, as well as the usual hematological and biochemical findings, permits correct diagnosis of the causes of most extracerebral seizure disorders. Only occasionally is further investigation necessary.

A thorough physical examination can provide important information. A dome-shaped head and an open fontanelle may indicate congenital hydrocephalus. A severe rhinitis due to bacterial infection or mycosis may extend to the cranial cavity (3, 11).

Cats suffering from a chronic rhinitis may have seizures, especially when they are also suffering from a cardiomyopathy and/or when they are under stress. Finally, tumors of the nasal cavity can also spread to the brain (3, 11, 12). Respiratory and gastrointestinal disturbances can be an indication of canine distemper; a pronounced pain on bending the head down can be a symptom of bacterial meningitis or meningoencephalitis. Salivation,

miosis, tremor, and diarrhea are symptoms of ingestion of an organophosphate. The more carefully an animal is examined, the more quickly the cause of the seizures will be recognized. Hematological, biochemical, and serological findings can confirm or disprove a presumptive diagnosis. The following short review lists the causes of the most important extracerebral seizures (Table 4).

Extracerebral seizure disorders

Cerebral hypoxia is among the more frequent causes of extracerebrally triggered seizures. Generally it has a cardiac etiology and is found in both dogs and cats. Asystole, paroxysmal bradycardia (Figure 2), and tachycardia (Figure 3) can lead to a short-term reduction in the blood flow to the brain, resulting in a short loss of consciousness (syncope). If the reduction in blood flow is of longer duration, generalized tonic-clonic seizures may ensue (13). Disturbances of cardiac origin in the cerebral blood supply (Adams-Stokes syndrome) occur in middle-aged and older animals. In contrast, heart and vessel malformations (for example, aortal or pulmonary stenosis), which may also result in reduced cerebral blood flow, might be expected to be present in juvenile and young adult animals, depending on the severity of the condition. Auscultation, taking of the pulse, and ECG are part of the routine diagnostic process. These investigations may be complemented by echocardiography, if deemed necessary. Postexercise and long-term ECG may be necessary for definitive diagnosis of some cases of paroxysmal arrhythmia. Coincidental cardiac- and cerebrally derived seizures are possible, since both forms occur frequently (13). In these cases great care must be taken to evaluate any interaction between these etiologies. Intense, prolonged epileptic seizures can result in considerable strain on the heart and in cases with pre-existing cardiac damage there may be further deterioration in cardiac function. Equally, a prolonged episode of reduced cardiac output may result in such profound cerebral hypoxia that seizures result and cerebral damage ensues.

Paroxysmal hypoxia may also have a respiratory origin. In addition to the above-mentioned diseases of the pharyngonasal

Table 1
Diagnostic measures for distinguishing among the various seizure disorders

Thorough case history

Physical examination

- Palpation: head, muscles, lymph nodes, skeleton, joints
- Auscultation, palpation of pulse
- ECG and echocardiography
- Exercise and/or long-term ECG
- X-ray: thorax, abdomen

Complete blood count

Routine chemistry

- Fasting blood glucose – if low, then assay serum insulin concentration
- Liver enzymes, bile acids
- Ammonia – ammonia tolerance test
- Serum electrolytes: sodium, potassium, calcium
- Blood urea nitrogen, creatinine, phosphate

Urinalysis

Serological examinations

- Distemper
- Toxoplasmosis, borreliosis
- FeLV, FIV, FIP, etc.

Neurological examination

Ophthalmological examination

Video monitoring

CSF analysis

Electroencephalogram

Imaging techniques

- (Radiography)
- Computed tomography (CT)
- Magnetic resonance imaging (MRI)

Table 2
History in seizure disorders

Case history

- Breed, age at onset of seizures, gender
- Immunizations
- Previous illnesses or trauma, surgery
- Birth complications
- Medications
- Management, feeding
- Possible intoxication
- Previous treatment

History of family

History of seizures

- First seizure event*
 - Single seizure
 - Cluster of seizures
- Status epilepticus*
 - Interval between first and second seizures

Description of seizures

- Focal or generalized occurrence of seizure
- Vegetative symptoms
- One or more types of seizures
- Duration of seizure
- Frequency of seizures
- Duration of intervals

Seizure association with ...

- Degree of activity
- Time of day
- Food intake

Seizure trigger

- Excitement
- Exhaustion

Interictal symptoms

Table 3
Prevalence of common seizure disorders in relation to age at onset

Age at onset before 8 months

Frequent

- Congenital brain disorders (e.g., malformations, hydrocephalus)
- Encephalitis or meningitis
- Trauma
- Storage diseases

Extracranial causes

- Hepatic encephalopathy (e.g., portocaval shunt)
- Hypoglycemia
- Electrolyte disturbances
- Storage diseases
- Intoxications
- Hypoxia
- Intestinal parasitism

Rare

- Idiopathic epilepsy

Age at onset 8 months to 4 years

Frequent

- Idiopathic epilepsy

Rare

- Encephalitis or meningitis
- Acquired hydrocephalus
- Trauma
- Congenital brain disorders (e.g., malformations, hydrocephalus)
- Neoplasia

Extracranial causes

- Hepatic encephalopathy (e.g., portocaval shunt, severe liver disease)
- Hypocalcemia
- Electrolyte disturbances
- Hypothyroidism
- Intoxications

Age at onset above 4 years

Frequency increasing with age

- Neoplasia
- Degenerative disorders
- Vascular disorders

Extracranial causes

- Hypoxia
- Hypoglycemia (insulinoma)

Frequency decreasing with age

- Idiopathic epilepsy

Rare

- Trauma
- Encephalitis or meningitis
- Acquired hydrocephalus

Extracranial causes

- Hepatic encephalopathy (e.g., serious liver disease)
- Hypocalcemia
- Electrolyte disturbances
- Hypothyroidism
- Intoxications



Figure 2
ECG from a 12-year-old male Poodle with complete AV block. Under sudden stress (e.g., ringing of doorbell) the dog collapsed with convulsions. The ventricular frequency is 30–40/minute. Calibration 1 cm = 1 mV, paper speed 25 mm/s. Lead I–III from top to bottom.

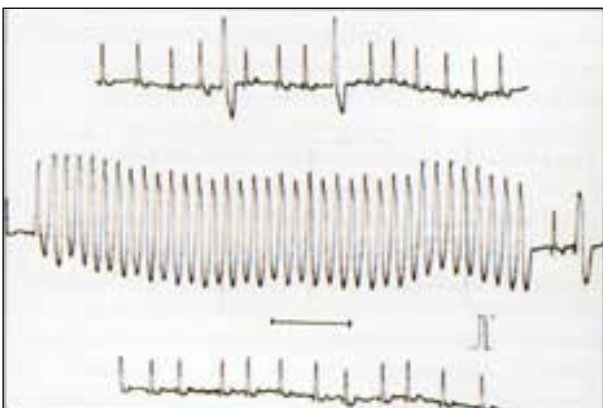


Figure 3
ECG leads (Lead II) on different days from a 5-year-old male Boxer with aortic stenosis and Adams-Stokes syndrome due to a tachycardic dysrhythmia. Ventricular tachycardia is present in the second trace. The first and third traces illustrate atrial fibrillation, with different frequencies and two ventricular premature contractions.



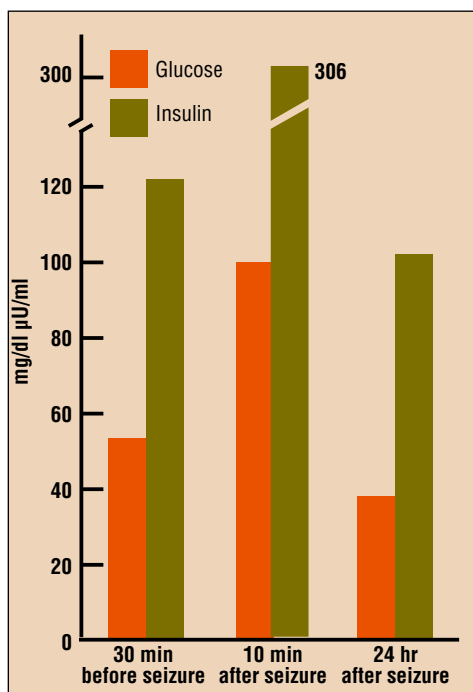


Figure 4
Concentration of blood glucose and insulin in a 6-year-old female Cairn Terrier with insulinoma before and after epileptic seizure.

cavity, a collapsed trachea and other types of respiratory disturbances are also important factors.

Hypoglycemia can cause many different symptoms, since glucose is the main substrate of cerebral energy metabolism. Consequences can range from episodic attacks of weakness to epileptic seizures. The clinical symptoms depend not only on the degree of hypoglycemia but also on how quickly the blood glucose level falls. When the decrease is slow, there may be severe convulsions without sympathetic symptoms (ravenous appetite, tachycardia, restlessness, hyperexcitability, trembling).

In puppies and juvenile animals, especially in miniature and toy breeds, hypoglycemia is caused by anorexia and gastrointestinal and metabolic disturbances, but rarely by storage diseases (3, 5, 11). In dogs older than 5 years the cause is often an insulinoma, which is frequently diagnosed late in the stage of the disease. Since the hypoglycemic conditions are episodic, about one third of all cases of insulinoma are treated incorrectly over weeks and months with anti convulsive drugs, delaying definitive diagnosis (14). Confirmation of hypoglycemia can require a period of fasting of more than 24 hours (11, 14). If the blood sugar value is low, the insulin concentration should be determined. Insulin concentrations that are raised or in the upper normal range are proof of an insulinoma, while very low insulin values indicate an insufficient supply of glucose or an increase in consumption. Glucose values determined after a seizure can be diagnostically misleading, since an increased counter-regulation, caused by the seizure, can raise the blood sugar far into the normal range despite a high insulin value (**Figure 4**).

Hepatoencephalopathy can be caused by a congenital or acquired portosystemic shunt, as well as by severe liver disease (15). The capacity of the liver to perform detoxification and synthesis is reduced. Ammonia and other enteric protein metabolites enter directly into general circulation and the brain, where they impair brain metabolism, leading not only to the formation of false neurotransmitters but also to possible neuronal damage. The result is a great variety of CNS symptoms, including epileptic seizures, the severity of the symptoms depending on the amount and type of protein intake. Neuronal damage can also result in epileptic foci, leading to seizures of cerebral etiology. Both dogs and cats can suffer from hepatoencephalopathy. Radiography usually demonstrates a small liver. Hematological and biochemical findings (anemia, low blood urea nitrogen, low protein) can be a sign, but

they are nonspecific. Pre- and postprandial bile acid and ammonia values should be determined. In the case of questionable values (the ammonia concentration can vary widely, so causing episodic symptoms) an ammonia tolerance test is recommended.

Hypocalcemia can lead to excitatory disturbances in the peripheral and central nervous systems, which then cause tetany or tonic-clonic seizures. Hypocalcemia can have various causes. The most frequent cause of hypocalcemia is kidney insufficiency, but because of acidosis the likelihood of tetany and convulsions is reduced. The most frequent cause of hypocalcemic tetany and seizures is hypoparathyroidism. Determining the serum phosphate and, if possible, the parathormone concentration is important. Seizures can also occur in cases of **aberrations in serum electrolyte concentration**, particularly if the serum sodium is considerably raised or lowered.

In cases of **hypothyroidism**, there may be neuromuscular disturbances as well as generalized seizures. These symptoms disappear on adequate substitution therapy (16). Miniature Schnauzers can suffer from a massive, genetically caused **hyperlipidemia**, which can cause seizures (4).

Intoxication, which has a varied symptomatology, can lead to more or less severe convulsions. Treatment depends on the clinical picture and the suspected toxin. It requires a direct or indirect proof of the toxin.

Cerebral seizure disorders

Since cerebrally caused seizures can be the result of cerebral inflammation (**Table 5**), particularly in cats (17), hematological findings (number of leukocytes, differential blood count, erythrocyte sedimentation rate) are particularly important. In cats, but rarely with dogs, polycythemia can be the cause of seizures. The biochemical findings, except for coincidental changes, are often within normal limits.

NEUROLOGICAL EXAMINATION

After excluding extracerebral causes for a seizure, in most cases it should be possible, with a carefully conducted neurological examination (**Table 6**), to distinguish progressive or still-active brain diseases from an epilepsy by means of differential diagnosis.

Close observation and evaluation of consciousness, behavior, posture of head, trunk and limbs, as well as gait, are necessary. Lesions of the cerebral cortex, which can cause seizures, can lead to a more or less severe impairment of consciousness and behavior, with compulsive movements that are performed often in large circles in the direction of the lesion. The head may be lowered and the animal may stand with its legs slightly straddled. The gait may be hardly influenced at all, except when the animal is walking on uneven ground or climbing stairs.

All levels of the central and peripheral nervous systems, including sensory and motor activity, are involved in the postural reactions, as well as the conscious proprioception. The results of these investigations may point to a disease of the cerebral cortex, contralateral to the lesion. The spinal reflexes are normal or somewhat increased. Cranial nerve deficits can be seen when the lesion extends into diencephalon (optical nucleus) and the brain stem. Lesions of the occipital cortex can lead to contralateral blindness, despite normal pupillary reflexes.

In idiopathic epilepsy the neurological findings are normal. In symptomatic epilepsy, depending on the etiology, mild neurological findings are possible. Active and, above all, progressive brain disorders at an advanced stage usually show distinct neurological findings.

Examinations that are conducted postictally have to be repeated,

Table 4
Etiology of extracerebral epileptic seizures

Metabolic disturbances

Hypoxia

- Congenital and acquired cardiac diseases
- Respiratory disturbances

Hypoglycemia

- Excess of insulin
- Increased glucose consumption
- Insufficient glucose supply

Hepatoencephalopathy

- Portosystemic shunt
- Severe liver diseases

Hypocalcemia

- Hypoparathyroidism
- Chronic renal diseases

Electrolyte disturbances

Hypothyroidism

Hyperlipemias

Intoxication

- Organophosphates, carbamates
- Chlorinated hydrocarbons
- Rodenticides, herbicides
- Heavy metals (especially lead)
- Various plants, etc.

Table 5
Etiology of cerebral seizures caused by primary brain diseases (structural changes in the brain)

Space-occupying lesions

- Neoplasia
- Cysts

Cranial trauma

- Hemorrhages
- Scars

Meningitis and/or encephalitis

- Viruses*
distemper, FIP, FeLV, etc.

- Bacteria*
various types

- Fungi*
cryptococcosis, etc.

- Protozoa*
toxoplasmosis

- Unknown causes*

Hydrocephalus

- Acquired
- Congenital

Malformations

Vascular disorders

Degenerative changes

Storage diseases

Pre-, peri-, and postnatal brain lesions

Table 6
Neurological examination

Consciousness, behavior

Posture of the head, trunk and legs, gait

Postural reactions

Spinal reflexes

Cranial nerves

Sensation

Table 7
Parameters of cerebrospinal fluid

Constituent	Normal range
Color	Colorless
Turbidity	Crystal clear
Protein concentration	<30 mg/dl
Pandy	Negative
Cell count (nucleated)	<5/dl
Erythrocytes	None
White cell type	Mononuclear

since the seizure itself can influence the findings hours and even days later, depending on its severity (2, 3, 6, 11). At the start of therapy, anticonvulsives and sedatives can also produce false-positive findings (11). When neurological findings are negative, examinations should be repeated at regular intervals. Tumors of the rostral cerebral cortex can cause seizures as the sole clinical symptom over an extended period (18). In brain disorders that are healing (encephalitides, cranial trauma, etc.), the neurological deficits gradually recede but the epileptic seizures can continue to exist or even make their first appearance after the neurological symptoms and positive neurological findings have gone.

OPHTHALMOLOGICAL EXAMINATION

Sometimes (although seldom) an ophthalmological examination can be a diagnostic adjunct to the neurological one (1, 3, 11, 17). Chorioretinitis can be caused by a virus (FIP, FeLV, FIV), fungus (cryptococcosis, histoplasmosis, blastomycosis), or a protozoal infection (toxoplasmosis). An optic neuritis can be a sign of a granulomatous meningoencephalitis. A papilloedema can result from increased intracranial pressure caused by brain edema, inflammation, or a brain tumor.

VIDEO RECORDING AND MONITORING

The clinical features of a seizure are of critical importance for differential diagnosis of the various seizure disorders and the epilepsies. Since seizures arise paroxysmally and at varying intervals, it can be quite difficult, or even impossible, except in cases with clusters of seizures, to record a seizure by video from its very onset. Nonetheless, the owner of the animal should be encouraged to make videos, since even an incomplete record can yield important information. The most useful information can be obtained with long-

term video monitoring. For inpatient animals, cage doors made of safety glass facilitate such monitoring.

ADDITIONAL DIAGNOSTIC TESTS

Depending upon the clinical features of the seizures; other symptoms, clinical and neurological findings; and the history, breed, age, and gender of the animal, a list of possible cerebral alterations can be made and further diagnostic procedures planned. Additional diagnostic tests include cerebrospinal fluid analysis, EEG recordings, and imaging examinations.

Cerebrospinal fluid (CSF) analysis

Obtaining CSF is an invasive procedure requiring general anesthesia and a certain degree of practice. Determination of the most important CSF parameters (Table 7), on the other hand, is quite easy and their diagnostic value is high.

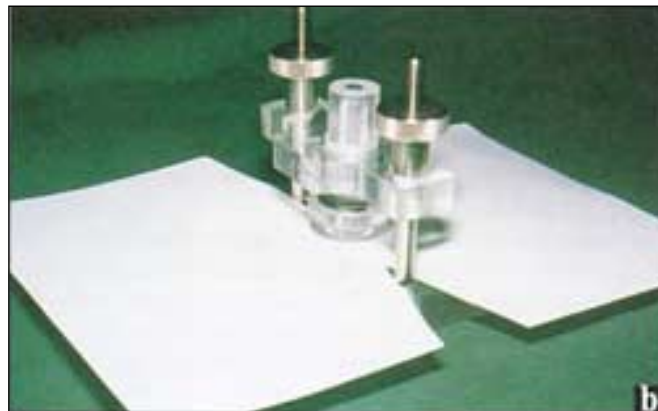
Normally, CSF is colorless and clear. Cloudiness points to an increase in protein and/or cell content.

The protein concentration has no specific diagnostic significance since the most varied changes can lead to its increase. The protein concentration can be determined qualitatively by the simple Pandy reaction. An albuminocytological dissociation (an increase in protein while the cell count remains unchanged) is a sign of an impaired blood-brain barrier. This is mainly seen with brain tumors.

The cell count and the cell distribution have to be performed within 30 minutes after drawing the fluid since cell deterioration is very rapid. Except as a result of fresh bleeding (trauma), the CSF is free of erythrocytes, their presence usually being the result of the tap itself. An increased cell count, a pleocytosis, results from inflammatory or necrotic changes. The highest cell counts are seen with bacterial meningitides and steroid-responsive meningo-



Figures 5a and 5b Sayk sedimentation chamber (modified by Kölmel). Filter paper is put onto an object slide lying on a floor plate. In the middle of the paper there is a hole 5–12 mm in diameter. A hollow cylinder with exactly the same inner diameter as the hole is placed on top. A spring and plate exert downward



pressure on the cylinder onto the object slide so that after the cylinder has been filled with CSF (0.5–1.0 ml) no CSF diffuses onto the paper. Once the spring pressure is slightly reduced, CSF can diffuse slowly into the filter paper, leaving cells on the object slide. The process should take no longer than 30 minutes.

encephalitis. The cytology is determined by the type of inflammation; there are rarely tumor cells. A cytological examination requires estimation of cell concentration. This is determined by cytocentrifugation or, very easily and inexpensively, by the Sayk sedimentation procedure (19) (**Figures 5a** and **5b**). Positive CSF findings are much more frequent in cats than in dogs, since in cats inflammation is more frequently the cause of seizures (17).

In addition, bacteriological investigations (evidence of bacteria and resistance tests), as well as virological, mycological, serological, and immunological investigations, can be performed. The presence of antibodies in the CSF is of diagnostic significance, as opposed to their presence in serum, since antibodies appear only when there is a viral infection of the CNS. They are not present in the CSF after

vaccination.

CSF investigations should not be performed postictally since seizures have an effect on the blood–brain barrier, leading to an increase in protein and pleocytosis (5, 6, 17, 20). Glucocorticoid administration can have the opposite effect.

A change in CSF findings may point to morphological brain alteration, although a negative finding is no proof of the absence of a CNS lesion. Expectation of high CSF pressure is a contraindication for a CSF tap since there is a danger of caudal herniation with fatal consequences.

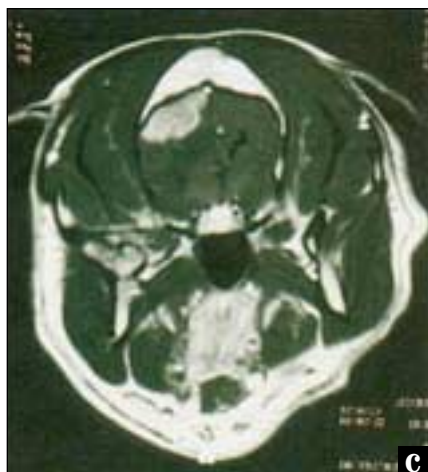
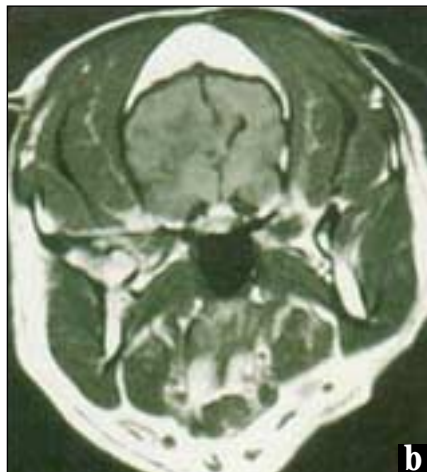


Figure 6

MRI scans from a 7-year-old female Rottweiler with a seizure disorder of 21 months' duration, caused by a meningioma.

(a) T_2 -weighted transverse image through the forebrain. A shift of midline and the compression of the left ventricle indicate a space-occupying lesion. In the white matter there is an increase in signal intensity, which points to a perifocal edema. In T_2 -weighted images, edema and fluids such as CSF appear hyperintense.

(b) T_1 -weighted image through the forebrain. The same changes can be seen but instead of an increase there is a decrease in signal intensity in the left cerebral medulla. Fluids cause a decreased signal intensity in T_1 -weighted images.

(c) T_1 -weighted image through the forebrain after gadolinium-DTPA application. The contrast agent led to an intensive enhancement of the rim of the left dorsal cortex, where in the native image there was neither a clear change in structure nor in signal intensity as compared with normal brain tissue. The cortex-medulla interface shifts concavely (i.e., toward the center of the brain), a typical finding with meningiomas. A further sign of a meningioma is a gadolinium enhancement of the meninges adjacent to the tumor. This illustration shows, rather atypically for meningiomas, an uneven uptake of the contrast medium, due to tumor necrosis.

(d) T_1 -weighted dorsal image after gadolinium-DTPA application. The considerable extent of the tumor is more visible in this section. All the features of the mass can be identified.

Electroencephalographic investigations (EEG)

In humans, EEG recordings are among the most important techniques for diagnosis of epilepsy. With animals, however, the lack of cooperation makes EEG examination considerably more difficult. In addition, head shapes and skull thicknesses vary widely, and the heavy head muscles of the dog cause the superimposition of muscle action potentials and other artifacts onto the brain waves (21). Electroencephalographic recordings are, therefore, relatively rarely performed. If deemed necessary, they are usually (but not always) performed on sedated animals. The sedatives and narcotics used mostly influence normal brain activity. Nonetheless, interictal EEGs are valuable in the diagnosis of seizure disorders, above all for confirming structural changes in the brain (6).

Examination by imaging techniques

Several such techniques are considerably important in diagnosing intracranial diseases. While X-rays can be taken in almost every veterinary practice, it is rare that a veterinarian has direct access to computed tomography or magnetic resonance imaging. It has become increasingly possible, however, to work in cooperation with institutes of human medicine.

Radiography

Because of the cranial bones, the brain is not accessible to X-rays. In dogs, X-rays of the head are to be recommended only when cranial fractures, congenital hydrocephalus (altered head shape), or alterations in the skull are suspected. In cats, radiography may be useful when a brain tumor is suspected, since feline meningiomas may calcify and be visible.

Computed tomography (CT)

In CT, which is also based on X-rays, the brain is divided into a series of imaging slices. Structural alterations in the brain can be seen well in these cross-sectional images. The administration of iodine-containing contrast agents improves the diagnostic capability. At sites where the blood-brain barrier is impaired, there is an enhancement due to the contrast agent. Computed tomography examinations of the brain are possible only under general anesthesia. In small dogs and cats, bone artifacts can cause difficulties when tissues of different densities are adjacent to each other. This happens particularly in the middle and posterior cranial fossae, where a relatively small amount of soft tissue is surrounded by bone.

Magnetic resonance imaging (MRI)

Magnetic resonance imaging is based on the magnetic properties of the hydrogen atom and its nuclear magnetic resonance. Tissue is imaged in varying gray-tone intensities. By means of multiplanar imaging, the location and extent of a lesion can be recorded. Image weighting (T_1 -, T_2 -weighted images) emphasizes tissue alteration (edema, bleeding, necrosis) (Figure 6 a, b). MRI views of anatomical soft tissue structures are superior to those obtained by CT. By the administration of paramagnetic contrast agents (gadolinium-DPTA) the sensitivity and specificity of the diagnosis can be improved (Figure 6 c, d).

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