

# Episodic collapse in the geriatric dog

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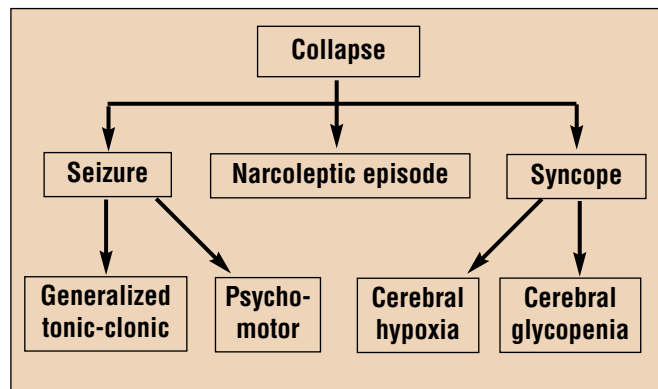


Figure 1 An illustration of a classification of collapse in the dog.

## KEY POINTS

- There are three types of collapsing episodes: seizure, syncope, and narcolepsy.
- The most common causes of seizures with an onset late in life are intracranial neoplasia and hypoglycemia.
- Hypoglycemia, to the point of causing seizures, can be caused by many different etiologies; however, insulinoma is the most common cause in the geriatric dog.
- A diagnosis of insulinoma is made by demonstrating an inappropriately high serum insulin concentration during episodes of hypoglycemia.
- Syncope can be caused by either cerebral glycopenia or cerebral hypoxia. Cerebral hypoxia in turn can be caused by a multitude of conditions, however, the most common cause of transient hypoxia is cardiac arrhythmia.
- Holter or event monitors may be necessary to diagnose and characterize the cardiac arrhythmia causing the syncopal episode.

## INTRODUCTION

The term collapse has been used inconsistently in veterinary medicine. In this article, collapse refers to a sudden onset of falling down that is associated with loss of consciousness. It is important to note that there are other conditions characterized by acute weakness in which the animal does not lose consciousness, such as myasthenia gravis, acute cervical disc herniation, or cataplexy. These conditions will not be discussed in this article.

In general, there are three types of collapsing episode, see Figure 1 (1):

- Seizure.
- Syncope.
- Narcolepsy.

Seizures are the most common type of collapse in dogs of any age and can either be generalized or partial. **Generalized seizures** are tonic-clonic in most cases, but **psychomotor seizures** (inaccurately called *petit mal* or absent-type seizures) can also occur. **Syncopal episodes** also occur commonly in older dogs. Narcolepsy is uncommon in the dog, and, since it occurs most commonly in young animals, the condition will not be discussed further.

The initial workup for a patient presenting for collapsing episodes should, at the very least, include: a careful neurological and ophthalmologic examination; an immediate blood glucose measurement; a complete blood count; a serum chemistry profile; and a urinalysis. Additionally, an electrocardiogram (ECG) should be obtained for patients presenting for episodes that seem either to be syncopal or psychomotor seizures.

## SEIZURE

A seizure is an episode that results from a paroxysmal disturbance of electrical activity in the brain. Seizures are initiated by neurones: 'a seizure focus'. These neurones may either show a reduced threshold of excitability or may lack sufficient control from inhibitory neurotransmitters. Neighboring neurones are depolarized, and, depending on the extent of the propagation of this spontaneous depolarisation, a partial or generalized seizure ensues (2).

Seizures can be caused by many different etiologies, as shown in Table 1 (1, 2). While some of the causes listed in Table 1 clearly do not occur in the geriatric dog (e.g., lissencephaly), others can be seen at any age (e.g., trauma, toxicosis). This article will focus here on those few causes which are particularly common in the geriatric

**Table 1**  
**The differential diagnoses of the causes of seizures**

Causes of seizures that occur more frequently in older than in younger dogs are in **bold**, even if they occur rarely overall.

*Intracranial causes*

- Degenerative
  - Lysosomal storage diseases
- Anomalous
  - Congenital hydrocephalus
  - Lissencephaly
- Neoplastic**
  - Primary brain tumor**
  - Metastatic brain tumor**
- Idiopathic
  - Idiopathic epilepsy
- Infectious
  - Viral (i.e., rabies, distemper)
  - Rickettsial (i.e., Rocky Mountain Spotted Fever)
  - Bacterial (rare)
  - Fungal (i.e., cryptococcosis, blastomycosis, and others less frequently)
  - Protozoal (i.e., toxoplasmosis)
  - Helminthal (i.e., aberrant larval migration)
- Inflammatory
  - Granulomatous meningoencephalitis
  - Steroid responsive meningoencephalitis
- Toxic
  - Ethylene glycol
  - Heavy metals (i.e., lead, others)
  - Organophosphates
  - Others
- Traumatic

*Extracranial causes*

- Hypoglycemia**
- Hepatoencephalopathy (i.e., portosystemic shunt, hepatic failure)**
- Uremia**
- Hypothyroidism**
- Hypocalcemia**
- Hyperlipoproteinemia**
- Severe hypernatremia and hyponatremia**
- Hypertension**
- Polycythemia**
- Hyperviscosity syndrome**
- Diabetic ketoacidosis**

dog. The most common cause of seizures in older dogs is **primary intracranial neoplasia**, followed by **hypoglycemia** and **hepatoencephalopathy**.

**Intracranial neoplasia**

Intracranial neoplasia can be primary or secondary. Metastatic neoplastic lesions in the brain are quite common in humans suffering from cancer and are more commonly observed in dogs as survival rates are increasing with improved therapeutic measures for the primary tumor (**Figures 2 and 3**) (3). Among primary intracranial neoplastic lesions, meningiomas (**Figure 4**) have recently been reported to be most common, followed by gliomas (4).

*Clinical presentation*

Many patients suffering from intracranial neoplasia develop seizures. In fact seizures are the first clinical sign observed in more

than 50% of patients with rostral cerebral tumors (5). However, many dogs go on to develop other neurological signs, such as loss of vision, head tilt, disorientation, conscious proprioceptive deficits, or behavioral changes, which may reflect the specific location of the neoplastic lesion.

*Diagnosis*

The initial diagnostic workup in a geriatric dog with seizures should include a thorough neurological and ophthalmic examination, complete blood count, serum chemistry profile, a serum thyroid hormone concentration, and a urinalysis. This is important in order to help rule out hypoglycemia, hepatic failure, and other causes of seizures but also in order to gain information about commonly found organ dysfunctions in older patients.

It is important to remember that a normal blood glucose concentration does not rule out hypoglycemia as a possible cause of the seizures.

Intermittent hypoglycemia must be ruled out by fasting, as explained later, before any invasive measures are taken. If there is any suspicion of hepatic failure (e.g., unexplained weight loss or intermittent vomiting), pre- and postprandial bile acids should be evaluated.

Three-view thoracic radiographs (left lateral, right lateral, and ventrodorsal views) should also be taken to check for possible metastatic disease. If, after these tests, intracranial neoplasia is still the most likely diagnosis, a rational next step is computed tomography (CT) or magnetic resonance imaging (MRI) (6, 7). While most practitioners will not have access to MRI equipment, it may be possible to use a CT scanner of a local human hospital, local rules permitting. Radiographs of the skull are rarely useful, except in the very few cases with extensive bony involvement, and are therefore not routinely recommended.

A cerebrospinal tap should follow the CT or MRI scan. This is important because fungal or other granulomatous inflammatory lesions can mimic the appearance of a neoplastic lesion in some cases. Typical findings on cerebrospinal fluid (CSF) analysis in patients with brain tumors are a mild to moderate elevation in protein and, in about 60% of the cases, a mildly elevated white blood cell count (8). Neoplastic cells are only rarely encountered. If intracranial hypertension is suspected, appropriate measures must be taken in order to prevent the possibility of brain herniation.

*Therapy*

Options for antineoplastic therapy are limited in cases of intracranial neoplasia. Surgery is possible in some cases but requires referral to a veterinary facility with personnel trained in neurosurgery.

Other therapeutic options are largely dependent on the tumor type, which will not be conclusively known unless a tissue biopsy is harvested. Radiation therapy can be successful in temporary tumor control of certain types of neoplasia (9). Less success has been reported with chemotherapeutic protocols, except in cases of lymphosarcoma. Controlled studies on the potential benefit of various therapeutic protocols for specific neoplastic conditions are needed.

Palliative therapy with anti-inflammatory doses of glucocorticoids is of benefit in many patients for at least a short period of time. Anticonvulsant therapy should be considered when seizures are frequent, long-lasting, or violent or occur in clusters.

**Hypoglycemia**

Hypoglycemia caused by insulinoma is the second most common cause of seizures in the geriatric dog. However, hypoglycemia to the point of seizures may have many other causes (**Table 2**). In order to

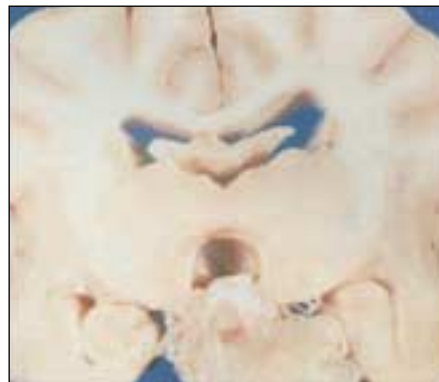




**Figure 2** Metastatic neoplasia: cerebral metastatic lesions (black areas) in a dog suffering from splenic haemangiosarcoma.  
Dr. David Waters, Purdue University.



**Figure 3** CT scan of intracranial metastatic lesions in the dog shown in Figure 2. Note that there are multiple mass lesions, a finding typical for metastatic neoplastic or granulomatous lesions.  
Dr. David Waters, Purdue University



**Figure 4** Meningioma in the area of the pituitary gland. Note that patients with masses in this particular location rarely suffer from seizures.  
Dr. Scott Schelling, Angell Memorial Animal Hospital, Boston.

prove a causative relationship between the hypoglycemia and the seizures, one must demonstrate Whipple's triad:

- Demonstration of a subnormal blood glucose concentration.
- Recognition of clinical signs of hypoglycemia.
- Cessation of clinical signs with resolution of hypoglycemia.

If Whipple's triad is present, the next effort is to establish the cause of the hypoglycemia. Insulinoma is by far the most common cause of hypoglycemia in the geriatric patient, but extrapancreatic neoplasia can also cause hypoglycemia, as can hepatic failure and sepsis.

#### Initial diagnosis

As for all patients presenting for collapsing episodes, the initial workup should include a careful neurological and ophthalmologic examination, complete blood count, serum chemistry profile, and a urinalysis. In addition, a serum insulin concentration should be obtained if the patient is hypoglycemic at the time of presentation.

However, because of secretion of counterregulatory hormones, many patients may not have a low serum glucose level at the time of presentation. These patients should have serial serum glucose concentrations evaluated during a fasting period of up to 48 hours. When the blood glucose concentration falls below 50 mg/dl (2.8 mmol/L), a serum sample for the evaluation of the serum insulin concentration should be collected and the animal should be fed. Because insulinoma cells secrete insulin autonomously, insulinoma patients have an inappropriately high serum insulin concentration in the face of hypoglycemia. In contrast, in patients suffering from extrapancreatic neoplasia and other causes of hypoglycemia, serum insulin concentrations will be appropriately low.

#### Extrapancreatic neoplasia as a cause of hypoglycemia

The most common extrapancreatic neoplasia causing hypoglycemia are hepatocellular carcinomas, but many other tumor types can also induce hypoglycemia (10, 11). When an extrapancreatic mass lesion has been identified in a patient with hypoglycemia, a tissue biopsy should be collected for definitive diagnosis by histopathology and the neoplastic disease should be staged. For appropriate therapy the reader is referred to discussions on the specific neoplastic condition diagnosed in each patient.

#### Sepsis as a cause of hypoglycemia

Sepsis is an uncommon cause of hypoglycemia but should be suspected in a patient with hypoglycemia in which the serum insulin

**Table 2**  
**The common causes of hypoglycemia**

Common causes of hypoglycemia in older dogs are in **bold**.

#### Spurious hypoglycemia

- Failure to separate serum from whole blood**
- Laboratory error**

#### Decreased glucose intake

- Puppy hypoglycemia
- Toy breed hypoglycemia
- Chronic starvation
- Chronic malabsorption

#### Decreased gluconeogenesis

- Hepatic insufficiency
- Portosystemic shunt
- Hepatic failure**
- Adrenocortical insufficiency

#### Decreased glucose storage

- Puppy hypoglycemia
- Toy breed hypoglycemia
- Hepatic insufficiency
- Portosystemic shunt
- Hepatic failure**

#### Increased glucose utilization

- Insulinoma**
- Extrapancreatic neoplasia**
- Sepsis**
- Hunting dog hypoglycemia

#### Iatrogenic

- Exogenous insulin overdose

concentration is appropriately low and that has other clinical signs or findings on blood work that are compatible with sepsis (12). Samples for blood cultures should be collected, and the patient should start on a rigorous antibiotic protocol.

#### Insulinoma as a cause of hypoglycemia

Dogs presenting with insulinoma can show neuroglycopenic signs, such as seizures, weakness, ataxia, or depression or signs related to an increased discharge of the sympathetic nervous system, such as behavioral changes, shaking, trembling, and muscle fasciculations. The incidence of clinical signs reported in 113 dogs with insulinoma is shown in **Table 3** (13).

Most patients with insulinoma show only intermittent clinical signs and will have a rather unremarkable physical examination. Only a few patients will present in status epilepticus or in a

**Table 3**  
**The clinical signs of insulinoma, in a series of 113 dogs**

Clinical Sign	No. of Patients	% of Patients
Seizures	77	68
Collapse	38	34
Generalized Weakness	37	33
Posterior Weakness	37	33
Depression/Lethargy	21	19
Ataxia	21	19
Muscle Fasciculation	20	18
Bizarre Behavior	17	15
Polyphagia	12	11
Exercise Intolerance	11	10
Shaking/Trembling	11	10
Polyuria/Polydipsia	8	7
Weight Gain	7	6

All other clinical signs were reported with a frequency of less than 5%

comatose state. Owing to the age of the affected population, a number of geriatric abnormalities may be evident that are unrelated to the presence of an insulinoma, and some may present with signs of peripheral polyneuropathy (14).

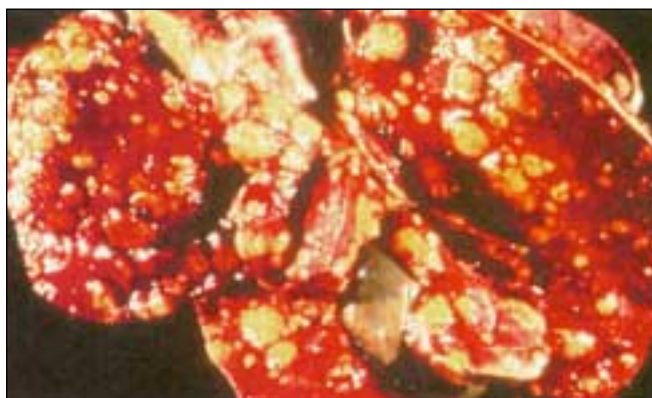
Radiographs of the thorax and abdomen are rarely diagnostic for an insulinoma and radiographically detectable pulmonary metastases of an insulinoma have not been reported in the veterinary literature, but these imaging studies can be helpful in excluding other causes of hypoglycemia. Abdominal ultrasound, however, is a valuable tool in the workup of a patient with suspected insulinoma. Even though only a small percentage of insulinoma can actually be visualized by abdominal ultrasound, it is very helpful in the assessment of possible organ metastasis or mesenteric lymphadenopathy.

Many diagnostic tests have been employed in human and veterinary patients with suspected insulinoma. The single most reliable test is the demonstration of an inappropriately high insulin concentration in the face of hypoglycemia (insulin–glucose pair). Serum to determine insulin concentration needs to be collected at the time of the hypoglycemia. This is accomplished by taking serial blood glucose measurements while the animal is fasted.

Several ratios have been evaluated to help document inappropriate insulin secretion: glucose–insulin ratio, insulin–glucose ratio, and the amended insulin–glucose ratio (15). Unfortunately none of these ratios improve diagnostic accuracy as they are associated with a high number of false-positive results (16). Their use is therefore not recommended. A variety of provocative tests have been suggested to aid in the diagnosis of insulinoma – all of which are time consuming, expensive, and potentially dangerous to the patient (they may promote hypoglycemic episodes) – they are not recommended.

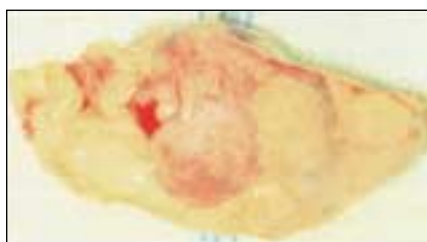
Emergency treatment is directed at correcting hypoglycemia and its complications. If hypoglycemia has been documented, a dextrose bolus (0.5 g/kg as a 25% solution intravenously over approximately 1 minute) should be administered and a dextrose infusion initiated. The goal is to eliminate clinical signs rather than to restore euglycemia. If neuroglycopenic signs persist, cerebral edema should be suspected and treatment using mannitol and glucocorticoids should be considered (15).

Surgical exploration is the treatment of choice for the initial management of canine insulinoma patients. Blood glucose must be monitored closely during the entire procedure. In order to stage the



**Figure 5** Hepatic metastases of a canine insulinoma. Note the severity of the metastatic disease in this dog.

Dr. T. VanWinkle, University of Pennsylvania, Philadelphia; reprinted with permission (13).



**Figure 6** Canine insulinoma after removal by partial pancreatectomy.

disease, a careful abdominal exploratory examination should be performed and biopsies of suspicious lymph nodes or areas within the liver should be obtained. In a compilation of 129 abdominal exploratory laparotomies of canine insulinoma patients, 66 (51%) had metastatic lesions detected (**Figure 5**) (13).

Identification of the insulinoma can be difficult. In 129 dogs surgically explored for an insulinoma, a pancreatic nodule could not be identified in 26 (20%) of the patients (13). Therefore, further evaluation of localization techniques seems warranted in canine insulinoma patients. The vast majority of the localization techniques reported in the human literature are either very expensive to perform or of limited availability in veterinary medicine. The most promising technique in veterinary medicine may be intraoperative ultrasound.

Extreme care should be exercised at all times when handling pancreatic tissue. The preferred surgical procedure is partial pancreatectomy (**Figure 6**) (17). If the tumor cannot be localized on abdominal exploration, a small pancreatic biopsy should be taken to evaluate the pancreas for diffuse neoplastic infiltration. Random removal of pancreatic tissue should not be performed. The most common postoperative complication is acute pancreatitis, but the risk can be minimized by gentle handling of pancreatic tissue during surgery and by appropriate postoperative care. Other common postoperative complications are persistent hypoglycemia and hyperglycemia.

Other antineoplastic therapies such as chemotherapy and radiation therapy have been evaluated in humans and in a limited number of cases of dogs. However, no recommendations can be made at this point.

Symptomatic treatment should be instituted in the following cases:

- If the owner declines surgical therapy.
- If the patient is an extremely poor anesthetic risk.
- If the insulinoma is considered to be surgically non-resectable.
- If the attempted surgery is unsuccessful.
- If clinical signs recur following initial surgical treatment.



The dog should be fed a high protein, high-fat, high-complex carbohydrate diet, divided into 4–6 feedings per day. Such a diet will probably require home preparation (under the guidance of a veterinary nutritionist), although some of the dry-type, high-energy diets formulated for growing dogs might be suitable. If the dog shows signs of weakness, it should be offered a small amount of food immediately. The owner should be instructed to apply a dextrose solution (a sweet syrup or honey) on the animal's mucous membranes and to contact their veterinarian in case of a seizure. Excitement should be avoided, and exercise should be limited to short leash walks (15).

Glucocorticoids should be administered if frequent feedings alone will not control clinical signs. Prednisone or prednisolone can be started at a dose of 0.25 mg/kg orally twice a day. If clinical hypoglycemia cannot be controlled, the dose can be increased up to a maximum dose of 2–3 mg/kg twice per day. The development of iatrogenic hyperadrenocorticism and gastrointestinal side-effects (gastritis, gastric ulceration, colitis), may warrant the discontinuation of steroid therapy.

Diazoxide is a nondiuretic benzothiadiazine that has anti-hypertensive as well as hyperglycemic properties. Diazoxide does not impair insulin synthesis, and it is not cytotoxic to beta cells. The recommended initial dose is 5 mg/kg orally twice per day. The dose can gradually be increased to 30 mg/kg twice a day if the initial dose does not result in an adequate response. In order to decrease the frequency of gastrointestinal side-effects, the drug should be administered with a meal (18).

Somatostatin decreases several polypeptides of the gastroenteropancreatic system, including insulin, glucagon, gastrin, secretin, motilin, and many others. Octreotide, a long-acting somatostatin compound, lowers the concentration of insulin in about 50% of human insulinoma patients and improves clinical signs in about 65% of these cases. The treatment of canine patients with octreotide has been investigated in five dogs with relapsing  $\beta$ -cell tumors. A dose of 10–20  $\mu$ g of octreotide was administered 2–3 times per day. Two of the five patients survived (for 9 and 12 months respectively) (19). However, one of these two dogs was treated with octreotide for only 1 week and was maintained on glucocorticoids after that time. Octreotide used at the dose mentioned is virtually without side-effects (19). Further work on optimizing the dose and frequency of administration is warranted.

The long-term prognosis for dogs with insulinoma is grave although the short-term prognosis is good. In a compilation of 114 dogs with insulinoma that had been treated with surgery and with symptomatic therapy after recurrence of clinical signs, the mean survival was 11.5 months with a median survival of 12 months in 25 dogs (13). In a study comparing the survival of canine insulinoma patients with stage M1 (metastatic disease present) versus stage M0 (no metastatic disease present) disease, dogs in the M0 group survived significantly longer than those in the M1 group (20).

### Hepatic failure

Hepatoencephalopathy can be the cause of seizures in dogs of any age. However, in older dogs, hepatoencephalopathy is most often caused by hepatic failure with or without secondary acquired portosystemic shunting. Hepatic failure can be caused by a multitude of aetiologic factors, which can be infectious, inflammatory, toxic, or neoplastic. However, the most common causes of hepatic failure in older dogs are idiopathic chronic hepatitis, anticonvulsive drug toxicity, and hepatic neoplasia, with metastatic neoplasia being more common than primary hepatic neoplasia (21).

In order for clinical signs of hepatoencephalopathy to develop, a major portion of hepatic functional reserve must be lost (Figure 7).



**Figure 7** Hepatic cirrhosis: This liver specimen was found at postmortem examination of a dog suffering from hepatic failure. Note the multiple regenerative nodules and the overall pale appearance of this liver.

Dr. Catharine Scott-Moncrieff,  
Purdue University.

Often the animal will have a history of weight loss, anorexia, or intermittent vomiting. Depression, diarrhea, and abdominal distension are other clinical signs commonly observed. Seizures and neurological changes often develop late in the course of the disease. It is rare for seizures to be the only clinical sign present.

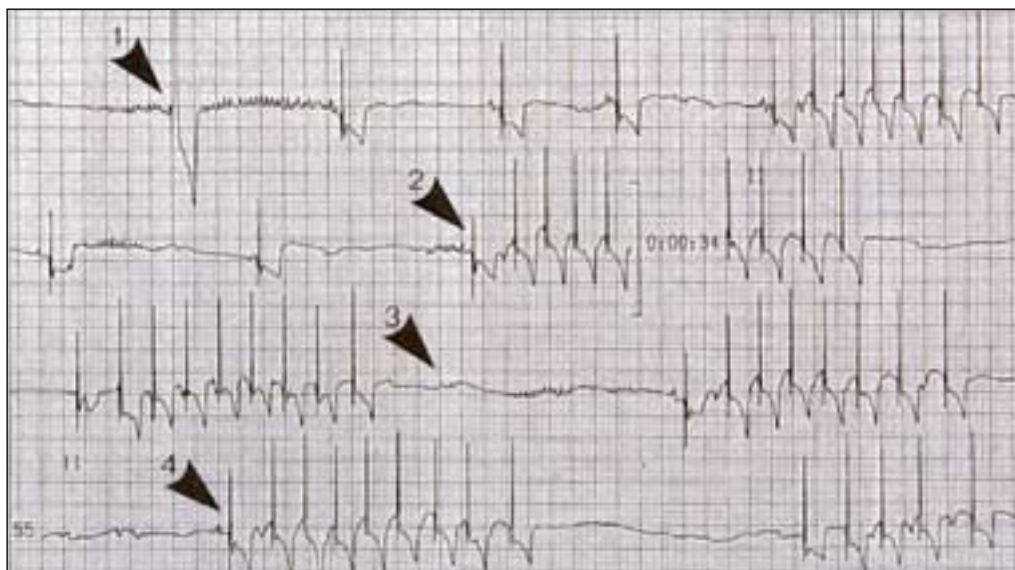
A geriatric dog presenting with seizures and other clinical signs compatible with hepatic failure should receive a careful neurological and ophthalmologic examination. The initial workup should also involve a complete blood count (including a platelet count), serum chemistry profile, urinalysis, and pre- and postprandial bile acids. Most patients suffering from end-stage liver disease will show different degrees of hypoalbuminemia, hypoglycemia, and hypocholesterolemia and a decreased serum urea nitrogen level. Hepatic enzymes are often only mildly elevated, while both pre- and postprandial bile acids are typically markedly elevated.

If the initial workup is suggestive of hepatic failure, the liver must be evaluated more carefully. Abdominal radiographs and abdominal ultrasound are important diagnostic tools. In most cases a conclusive diagnosis and specific therapy require a hepatic tissue biopsy. Cytological evaluation of fine needle aspirates can be useful, especially in cases of hepatic lymphosarcoma. However, an ultrasound-guided true cut biopsy, which can be examined by cytology and histopathology, is preferable. A coagulation profile must be evaluated, and, if it is abnormal, appropriate preventative measures should be taken to prevent hemorrhagic complications.

The therapy for hepatic failure may include specific measures if a cause has been identified. If hepatic failure is suspected to be due to anticonvulsive therapy, such as phenobarbital, medication should be changed to an alternative anticonvulsive support. Potassium bromide is the compound of choice, and a loading protocol should be applied. In many cases, symptomatic and supportive therapy are the only therapeutic options. The clinical signs of hepatoencephalopathy can often be managed successfully by a protein-restricted diet (e.g., WALTHAM® Veterinary Diet; Canine Low or Medium Protein), along with oral lactulose and oral antibiotic therapy with neomycin or metronidazole (22). Ursodeoxycholic acid may also be beneficial in the management of chronic hepatopathies (22). A variety of drugs, such as colchicine, have been suggested for the modulation of hepatic fibrosis (23).

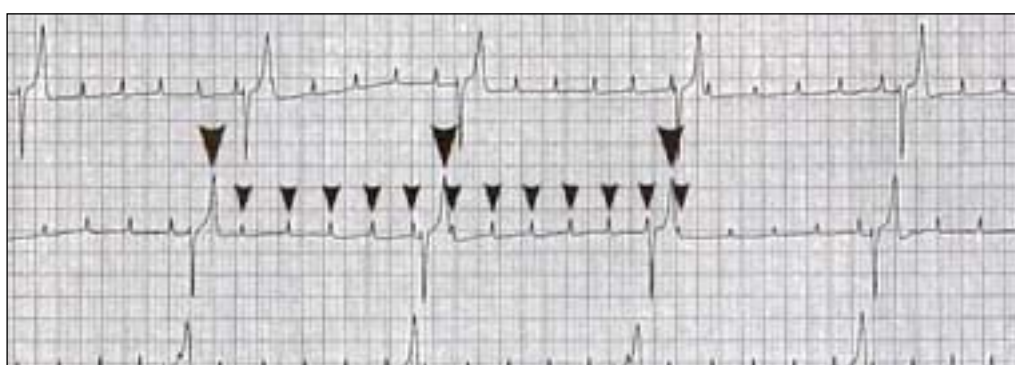
### Other causes of seizures

Hypothyroidism, end-stage renal failure, diabetic ketoacidosis, hypertension, polycythemia, hyperviscosity syndrome, and electrolyte disturbances also occur more commonly in older than in younger dogs. However, overall they are uncommon causes of seizures in the geriatric dog. The reader is referred to discussions elsewhere for information on these and other less commonly observed causes of seizures in the geriatric dog.



**Figure 8** Sick-sinus syndrome in an 8-year-old female spayed Miniature Schnauzer. This dog was presented for weakness and syncopal episodes. Note the heterogeneity of this ECG. Ventricular escape beats (1), fusion beats (2), atrial standstill (3), and episodes of supraventricular tachycardia (4) can all be observed.

Dr. Teresa DeFrancesco, North Carolina State University, Raleigh.



**Figure 9** Third-degree atrioventricular block in a 1-year-old male neutered English Springer Spaniel. This dog presented for syncopal episodes. Note the ventricular escape rhythm (large arrows), which does not break the atrial rhythm (small arrows).

Dr. Teresa DeFrancesco, North Carolina State University, Raleigh.

## SYNCOPE

Syncope is a sudden loss of consciousness that is caused by a temporary severe lack of oxygen or glucose in the brain resulting in cerebral hypoxia or glycopenia (1). A geriatric dog presenting for syncopal episodes must be carefully evaluated to rule out psychomotor seizures. The initial work up should include: a thorough neurological and ophthalmologic examination; an immediate blood glucose concentration; a complete blood count; a serum chemistry profile; an urinalysis; and an ECG. If the equipment is available and the episodes are frequent, the ECG should be continuously monitored for an extended period.

### Cerebral glycopenia

Glucose uptake into the brain is insulin-independent. Therefore cerebral glycopenia is a direct reflection of hypoglycemia. Hypoglycemia will more commonly lead to seizures than to syncope and has been discussed above.

### Cerebral hypoxia

Cerebral hypoxia can be due to many causes. However, only few of them are transient. Intracranial neoplasia, cerebral thromboembolic disease, cardiac tamponade, and anemia, for example, all can cause cerebral hypoxia, but it is less transient in nature in almost all cases.

The focus of this discussion will be causes of cerebral hypoxia that are typically transient, causing only intermittent episodes.

### Cardiac arrhythmia as a cause of cerebral hypoxia

The most common cause of transient cerebral hypoxia is cardiac arrhythmia, most commonly sick-sinus syndrome (Figure 8), high-grade second- and third-degree atrioventricular block (Figure 9), sinus arrest, and other forms of bradyarrhythmia (24, 25). Severe

tachyarrhythmia can also cause syncopal episodes. The diagnostic challenge is that many forms of arrhythmia are intermittent in nature and thus difficult to document on an ECG. Also, in some of these patients the sympathetic stimulation of hospitalization may be sufficient to abort the bradyarrhythmia.

In patients with a suspected arrhythmia, an ECG – preferably with at least four leads – should always be taken. If the ECG is normal and the initial workup, as described above, has not suggested a psychomotor seizure or cerebral glycopenic syncopal episode, a cardiac workup consisting of thoracic radiographs and an echocardiogram should be performed.

If no intrinsic cardiac disease is identified and the animal does not show any arrhythmias or syncopal episodes (or both) during hospitalization, an ECG study with either a Holter monitor or an event monitor should be performed. A Holter monitor is used to record a continuous ECG over a 24-hour period (26). The recorded tape is then analyzed for abnormalities using a computer. The event monitor also continuously records the ECG, but it overwrites the tape constantly. Whenever the owner observes an episode, he or she presses a button and the last few minutes and the following few minutes are permanently recorded. An event monitor is especially useful when episodes occur infrequently, as it may be worn for several days until an episode is observed.

While some forms of bradyarrhythmia are due to intrinsic heart disease and must be treated specifically, most are idiopathic. Some cases of sick-sinus syndrome and third-degree atrioventricular block can initially be treated medically. The compound used most successfully is probantheline bromide, a parasympatholytic agent with antimuscarinic activity. Another drug that may be of benefit is terbutaline sulphate, a  $\beta$ -sympathomimetic agent. However, many patients do not respond to either of these drugs, and some that respond initially only do so for a short period of time.

Where the patient cannot be treated medically, the placement of a cardiac pacemaker becomes necessary. Most leads are placed transvenously through the jugular vein and the pulse generator is anchored subcutaneously in the dorsolateral neck region (27, 28). Placement of a cardiac pacemaker has become routine in dogs, and, in the absence of progressive intrinsic heart disease, these patients often have a favorable prognosis.

#### *Other causes of cerebral hypoxia*

Another cause of transient cerebral hypoxia is severe episodic internal blood loss leading to a hypotensive crisis and a syncopal episode. This clinical picture is most common in dogs with splenic haemangiosarcoma, which can cause an acute hemorrhagic crisis (29). In many of these patients the hemorrhage actually stops without intervention and the patient seems to recover. These patients are often easily diagnosed on presentation because of their pale mucous membranes, their low haematocrit, and a palpable mass

in their cranial abdomen.

Haemangiosarcoma is best treated with supportive therapy followed by splenectomy and, potentially, chemotherapy. However, overall prognosis is poor (30).

Patients with bleeding gastric ulcers can develop a hypovolemic crisis and recover spontaneously after the hemorrhage has stopped or slowed. However, most of these patients do not stabilize spontaneously without aggressive intervention. There is not, as yet, a consensus on treatment for this condition. Supportive care with blood products is imperative, but in some patients a surgical approach is necessary.

#### **Acknowledgements**

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