Syncope vs. Seizure

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INTRODUCTION

Collapse events in small animals are a common occurrence. They can arise secondary to many conditions, a few of them being either cardiac in nature or neurologic in nature. The veterinary technician's role in helping the veterinarian differentiate the cause of a collapse event is imperative and begins with a thorough history. Understanding which questions to ask a client regarding a collapse event can help the veterinarian understand possible causes of the collapse. The goal of this presentation is for the technician to understand and recognize what could be a collapse event, and understand how the answers to those questions help to narrow the veterinarian's diagnosis list.

HISTORY QUESTIONS

The following table displays a comprehensive list of questions that should be asked of each client who suspects their pet collapsed. The table also indicates if the clinical sign is common for seizures, cardiac syncope, or both.

Table 1. Typical questions asked of a client and answers for syncope vs. seizure

Questions for the client	Common for seizure	Common for cardiac syncope
Was the patient active prior to the event?	No	Yes
Was the patient asleep when the event initiated?	Yes	No
Was the event relatively short or was it prolonged?	Short or prolonged	Relatively short
How long until the patient was fully recovered?	Generally prolonged recovery period, can be short	Relatively short recovery period unless severely hypoxic

Was the patient mentally aware after the event?	Not common (patient will have a post-ictal phase)	Typical
Did the patient vocalize during the event?	Not common	Typical
Was the patient unconscious during the event?	Usually	Yes
Did the patient urinate or defecate during the event?	Both are common	Urination is common, defecation is not
Did the patient display opisthotonus?	Can occur depending on event	Yes
Did the patient salivate or have excessive jaw tone during the event?	Yes	No
Did the patient display tonic clonic movement of the limbs?	Yes	No
How often does the event occur?	Can be days to weeks to months apart	Can be frequent depending on the cause
Has there been recent trauma or toxin exposure?	Common	Uncommon

SEIZURE-LIKE EVENTS: INTRODUCTION

Most patients that are seen for seizures on appointment are not actively seizing or don't have a seizure while they are in the appointment, so it is important to inquire in depth about the event. The definition of seizures is abnormal electrical activity in the brain due to an abnormal balance of excitatory and inhibitory neurotransmitters, and they come from the forebrain. Generalized seizures are also referred to as grand mal seizures. These patients are unconscious, lateral, tonic clonic paddling (convulsing) of all four limbs, mouth chattering/chomping, and often excessive salivation. They may also urinate and defecate. Simple focal seizures can also be called partial seizures. With these seizures, patients usually do not fall to their side. They may experience twitching of the face, pulling back of the

lips and face, and can also sometimes stare off into space or salivate. There can also be an involuntary flexion or extension of a certain limb. Complex focal (partial) seizures are also known as psychomotor seizures. Fly-biting behaviors, running around like they are scared, and looking behind them are typical signs of complex focal seizures. These patients can have combinations of altered mentation and abnormal limb and body movements. Sometimes it is difficult to tell whether a patient is having a true seizure, especially since most patients are not having an episode when they come in for an appointment. Electroencephalogram (EEG) is a tool that can be used to differentiate between true seizures and other paroxysmal events, although it is not readily available. It is performed by placing electrodes on the patient, monitoring them for 1-hour increments, and looking for a seizure focus.

Seizure-like Events: Causes

There are three main types of seizures: Idiopathic epilepsy, symptomatic or secondary epilepsy, and reactive epilepsy. Patients that have idiopathic epilepsy have a genetic predisposition to seizures and usually have their first seizure within 1–5 years of age. Australian shepherds and German shepherds are a few breeds that commonly get idiopathic epilepsy. Idiopathic epilepsy is a diagnosis of exclusion, so blood work, MRI, and cerebral spinal fluid sampling will need to be performed prior to the diagnosis, with the results showing no significant findings.

Symptomatic, or secondary epilepsy is seizures caused secondarily to something intracranially. Anomalous or congenital problems, such as hydrocephalus or lissencephaly (smooth brain), are disease processes that animals can be born with that can cause seizures. Infectious and inflammatory diseases have the potential to cause seizures as well. Infectious diseases are not very common, but include fungal infections such as *Cryptococcus*, blastomycosis, and coccidioidomycosis (Valley fever). Most of the viral infections we vaccinate dogs for (distemper virus), so it is much less likely but can occur as well. Inflammatory disease (meningitis) is usually an autoimmune disease in dogs where the immune system attacks the nervous system, and it is not very common in cats. It is also known as granulomatous meningoencephalitis (GME) or necrotizing meningoencephalitis (NME). Middle-aged small/toy breeds such as poodles, French bulldogs, pugs, and Yorkies have a predisposition to this disease. Unfortunately, neoplasia is a common reason older dogs start having seizures.

The most common types of tumors dogs and cats get are a meningioma or a glioma. Meningiomas grow from the meninges (outer covering of the brain) and press down on the brain tissue, while gliomas grow from the nerve cells themselves within the brain tissue and

push out. Lastly, a vascular event or infarct is another reason a patient may start to have seizures. These are referred to as “stroke-like” events and can be either hemorrhagic (a bleed in the brain) or ischemic (blockage of blood flow to an area in the brain).

Reactive epilepsy is seizures caused by a metabolic cause (extracranial) and usually subside when the underlying cause is treated. The main two causes are metabolic or a toxin. Baseline blood work is an important diagnostic tool, as there may be obvious, treatable abnormalities that can be found, such as hypoglycemia. Once the hypoglycemia has been resolved, the seizures will likely stop. Liver failure can cause a hepatic encephalopathy that can also lead to seizures. Toxins such as metaldehyde (snail bait), ethylene glycol (antifreeze), and recreational substance toxicities can all cause seizures. Treatment with supportive care usually stops the seizures if implemented in a timely manner.

Cardiac Syncope: Introduction

There are numerous causes of cardiac syncope. The general mechanism of loss of consciousness in cardiac syncope is lack of cerebral blood flow, caused by any number of the following conditions. A key equation to remember is CO = SV x HR, where CO is cardiac output, SV is stroke volume, and HR is heart rate. Syncopal events typically present after the patient has been active. The patient will often vocalize and fall laterally recumbent. He/she may display opisthotonos for a brief period of time and may urinate during the event. Typically, the patient recovers from the event fairly quickly, with normal mentation very shortly after the episode concludes. Patients are often described as regaining their gait and drinking water normally after the event. Most events do not occur while the patient is in the hospital, and most clients are unaware of what they have witnessed. The emphasis, then, is placed on a comprehensive and thorough history in order to ascertain true aspects of the event.

Cardiac Syncope: Causes

Arrhythmias are a very common reason for collapse events in small animal patients. Bradyarrhythmias like sick sinus syndrome, atrial standstill, or 3rd-degree atrioventricular block with a low ventricular escape rate will decrease cardiac output and diminish blood flow/oxygenation to the brain. These patients either experience sinus arrest (very long pauses between heart beats) or very slow heart rates in general. Tachyarrhythmias can also cause collapse events due to low cardiac output. Ventricular tachycardia, supraventricular tachycardia, atrial fibrillation, and atrial flutter have the capacity to decrease ventricular filling by shortening the diastolic period, which will ultimately decrease cardiac output. General

mechanisms of arrhythmias are changes in automaticity and/or abnormal impulse conduction.

Low-output, or forward heart failure, affects many patients with valvular degeneration or primary myocardial diseases, as well as patients with diminished cardiac filling. Patients with myxomatous mitral valvular degeneration (MMVD) experience a combination of increased left atrial pressure and a severe insufficiency jet across the mitral valve. This reduces output to systemic circulation, alters the pressures within the left heart, and has the capacity to alter systemic blood pressure as well. Left atrial rupture is a possible consequence to severe left atrial enlargement, and also a possible cause of a collapse event in a patient with MMVD. These patients experience acute blood loss combined with fast acquisition of pericardial effusion, possibly causing impaired cardiac filling of the right heart. Other causes of impaired cardiac filling include cardiac tamponade from pericardial effusion or constrictive pericarditis from coccidioidomycosis. Patients that have depressed systolic function (decreased pumping function), as in dilated cardiomyopathy or some instances of arrhythmogenic right ventricular cardiomyopathy, have diminished cardiac output because of a reduced shortening fraction (low stroke volume/contractility).

Pulmonary hypertension is defined as elevated blood pressure within the pulmonary vasculature, and it can cause collapse in small animal patients. There are numerous causes of pulmonary hypertension, both pre-capillary and post-capillary in nature. Cardiac patients most often suffer from post-capillary pulmonary hypertension, in which prolonged, increased left atrial pressure increases pulmonary vascular resistance and increases workload on the right ventricle (the increased pressure initiates in the left heart and cascades backward through the vasculature in order to maintain forward blood flow). One cardiac cause of pre-capillary pulmonary hypertension is Eisenmenger’s syndrome, in which pulmonary hypertension is suspected to develop due to overcirculation of the pulmonary vasculature from a left-to-right shunt. Pulmonary obstructive diseases like COPD, heartworm infection, pulmonary thromboembolism, and pulmonary fibrosis are noncardiac causes of pulmonary hypertension that can cause cor pulmonale (changes to the right side of the heart that are secondary to the primary disease process) and possible collapse events.

Obstructive diseases like subaortic stenosis, pulmonic stenosis, neoplasia, or thrombi have the potential to diminish cardiac output due to either a fixed or dynamic obstruction to the outflow tract(s) of the heart.

Neurocardiogenic (vasovagal) syncope occurs when there is increased parasympathetic tone alongside an acute decrease in sympathetic tone, causing hypotension and bradycardia.

Tussive syncope is a cough-induced collapse event, where changes in intrathoracic pressure and increased vagal tone cause the patient to faint. Any cause of significant coughing has the potential to cause this.

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