


The Murmur


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Is it a seizure or syncope? Tips for differentiating.

By: Kacie Schmitt, DVM, Diplomate, ACVIM (Cardiology)

Syncope is a transient loss of consciousness due to global cerebral hypoperfusion. The loss of consciousness results from a reduction of blood flow to the reticular activating system in the brainstem. Syncope occurs when cerebral blood flow is <30-50% of normal. It is characterized by rapid onset, short duration, and rapid recovery.

Differentiating a syncopal event from a seizure can be challenging as there can be some overlap in characteristics, such as vocalization, urination, and extensor rigidity. Below is a table of features to help determine which problem a patient is experiencing.

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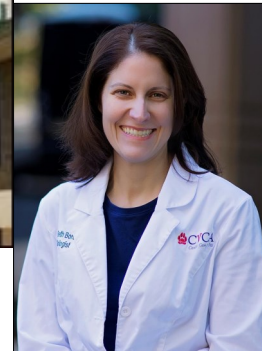
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con't from page page 1: Is it a seizure or syncope? Tips for differentiating.

CHARACTERISTIC	SEIZURE	SYNCOPE
Timing	Often at rest/sleep	Often when active
Precipitating event	Uncommon	Common Exercise Stress/startle Cough/gag Deglutition Micturition/defecation Pain
Presentation	Generalized/Convulsive (grand mal) Seizure Rapid loss of consciousness, twitching, rigidity progressing toward overt convulsive behavior/paddling Partial Seizure Altered behavior with complex motor activity, such as "fly biting"/jaw snapping or facial twitching	Collapse in lateral recumbency Flaccid collapse typical Opisthotonus, extensor rigidity possible No paddling, although can be mistaken for patient trying to "right" himself/herself
Urination	Common	Common
Defecation	Common	Uncommon
Hypersalivation	Common	Uncommon
Vocalization	Common	Common
Post-ictal/dementia period, Prolonged recovery to normal consciousness	Common	Uncommon When noted, typically associated with prolonged cerebral hypoxia and profound cardiac arrhythmia
Neurologic deficits	Possible	Not present

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Cardiogenic Syncope

Cardiogenic syncope is often classified as being caused by an arrhythmia (ventricular or supraventricular tachyarrhythmia or bradyarrhythmia), impaired cardiac output/poor myocardial systolic function (e.g. dilated cardiomyopathy, advanced degenerative valve disease, advanced hypertrophic cardiomyopathy, myocarditis, etc.), impaired or obstructed cardiac filling (e.g. restrictive cardiomyopathy, cardiac tamponade, etc.), or an outflow obstruction (e.g. valvular stenosis, pulmonary hypertension, etc.) .

When evaluating a patient for a possible syncopal event, a knowledge base of the most common cardiac diseases and the breeds they effect is helpful.

SIGNALMENT	COMMON ETIOLOGY OF CARDIAC SYNCOPE
Older, small breed dogs (e.g. those predisposed to degenerative valve disease, DVD)	New or recurrent congestive heart failure Pulmonary hypertension New atrial fibrillation Neurocardiogenic syncope
Large and giant breed dogs (e.g. those predisposed to dilated cardiomyopathy, DCM)	Ventricular arrhythmia Poor cardiac output
Boxer dogs and Bulldogs (e.g. those predisposed to arrhythmogenic right ventricular cardiomyopathy, ARVC)	Ventricular, and sometimes supraventricular, tachyarrhythmia Development of DCM phenotype and poor cardiac output
Young dogs with loud heart murmur (Grade 3/6 or louder)	Pulmonic stenosis Subaortic stenosis
Older Miniature Schnauzer	Sick sinus syndrome (including bradycardia-tachycardia variant)
Older Cocker Spaniel	Heart block
Cats	Generally during activity/exertion and related to systolic anterior motion (SAM) of the mitral valve causing a severe outflow obstruction High-grade second or third degree atrioventricular block

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Neurocardiogenic Syncope

Neurocardiogenic syncope is also referred to as neurally-mediated, reflex-mediated, vasodepressor, situational, and vasovagal syncope. It is used to describe a common abnormality of blood pressure regulation characterized by the abrupt onset of hypotension with or without bradycardia. Fortunately, it is rarely fatal.

Neurocardiogenic syncope can occur in normal dogs and those undergoing high-intensity activity. It is triggered by exertion and coupled with excitement of startle and can occur without evidence of cardiomyopathy.

Dogs with degenerative valve disease are also prone to neurocardiogenic syncope and there are two theories as to why it occurs. The first is referred to as “empty ventricle syndrome” in people. The already hyperdynamic left ventricular function under the influence of a sympathetic surge stimulates mechanoreceptors, or C fibers, in the atria, ventricles, and pulmonary artery. The afferent C fibers project to the vagal nucleus which leads to a paradoxical withdrawal of peripheral sympathetic tone and increase in vagal tone. Ultimately this results in vasodilation and bradycardia. The second theory is that that sympathetic stimulation, which causes venoconstriction and increased blood flow to the right heart causes increased right ventricular outflow and stretches the vagal afferents at the left atrial and pulmonary venous junctions. This may trigger the reflex leading to sympathetic withdrawal.

Situational syncope is often associated with coughing (“cough-drop syndrome”), sneezing, vomiting, micturition, defecation, excessive or difficult swallowing. There are two theories regarding “cough-drop syndrome”, both of which relate to changes in intra-thoracic pressure. The first theory is that increased intra-thoracic and intra-abdominal pressures cause increased venous pressures, which are transmitted to spinal and intracranial veins, leading to increased intracranial pressure, decreased cerebral perfusion, and syncope. The second theory is that increased intra-thoracic pressure causes decreased venous return leading to decreased cardiac output and syncope. Vomiting and swallowing syncope results from afferent neural impulses arising from the upper gastrointestinal tract which activate the vasovagal reaction. The pathophysiology of micturition syncope is not fully understood, but is likely related to activation of mechanoreceptors in the bladder, especially during rapid emptying of a full bladder. Defecation syncope results from neural inputs from gut wall tension receptors activating the vasovagal reaction.

Non-cardiogenic Syncope

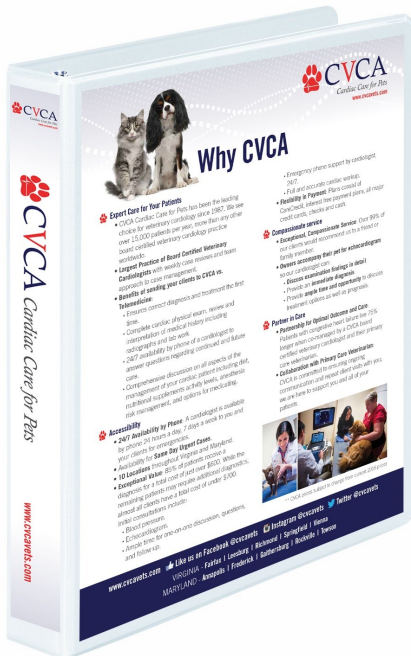
Non-cardiogenic syncope is also possible and should be considered for those patients with non-cardiac illnesses, no history of cardiac disease, and breeds at low-risk for heart disease. Neurologic syncope results from any disease process that compromises cerebral perfusion pressure, such as a cerebrovascular disease. Metabolic and hematologic causes are also possible and commonly include acute, severe hemorrhage, anemia, hypoxemia, and hypoglycemia.

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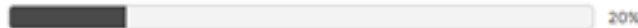
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1. Thank you for taking the time to complete our survey.



Thank you very much for taking the time to complete this survey. Your feedback is important. It is our goal at CVCA to serve as your partner in diagnosing and treating cardiac disease in your patients, as well as to offer the highest level of service to you and to your clients and patients.

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