Is it 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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**Is it a seizure or syncope? Tips for differentiating.**

<table>
<thead>
<tr>
<th>CHARACTERISTIC</th>
<th>SEIZURE</th>
<th>SYNCOPE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Timing</td>
<td>Often at rest/sleep</td>
<td>Often when active</td>
</tr>
<tr>
<td>Precipitating event</td>
<td>Uncommon</td>
<td>Common</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Exercise</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stress/startle</td>
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<tr>
<td></td>
<td></td>
<td>Cough/gag</td>
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<tr>
<td></td>
<td></td>
<td>Deglutition</td>
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<tr>
<td></td>
<td></td>
<td>Micturition/defecation</td>
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<tr>
<td></td>
<td></td>
<td>Pain</td>
</tr>
<tr>
<td>Presentation</td>
<td>Generalized/Convulsive (grand mal) Seizure</td>
<td>Collapse in lateral recumbency</td>
</tr>
<tr>
<td></td>
<td>Rapid loss of consciousness, twitching, rigidity progressing toward overt</td>
<td>Flaccid collapse typical</td>
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<tr>
<td></td>
<td>convulsive behavior/paddling</td>
<td></td>
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<tr>
<td></td>
<td>Partial Seizure</td>
<td>Opisthotonus, extensor rigidity possible</td>
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<tr>
<td></td>
<td>Altered behavior with complex motor activity, such as “fly biting”/jaw</td>
<td></td>
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<tr>
<td></td>
<td>snapping or facial twitching</td>
<td></td>
</tr>
<tr>
<td>Urination</td>
<td>Common</td>
<td>Common</td>
</tr>
<tr>
<td>Defecation</td>
<td>Common</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Hypersalivation</td>
<td>Common</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Vocalization</td>
<td>Common</td>
<td>Common</td>
</tr>
<tr>
<td>Post-ictal/dementia period,</td>
<td>Common</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Prolonged recovery to normal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>consciousness</td>
<td></td>
<td>When noted, typically associated with prolonged cerebral hypoxia and</td>
</tr>
<tr>
<td></td>
<td></td>
<td>profound cardiac arrhythmia</td>
</tr>
<tr>
<td>Neurologic deficits</td>
<td>Possible</td>
<td>Not present</td>
</tr>
</tbody>
</table>

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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.

<table>
<thead>
<tr>
<th>SIGNALMENT</th>
<th>COMMON ETIOLOGY OF CARDIAC SYNCOPE</th>
</tr>
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</table>
| 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 atroventricular block |
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 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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