ECG of the Month

A 14-year-old spayed female Schnauzer was evaluated for syncopal episodes and cough of 3 months’ duration. The owner reported that syncopal episodes occurred during periods of excitement or coughing. The dog had a prior history of degenerative mitral valve disease and congestive heart failure. Current treatments included furosemide (1.5 mg/kg [0.68 mg/lb], PO, q 12 h), spironolactone (1.5 mg/kg, PO, q 12 h), and enalapril (0.6 mg/kg [0.27 mg/lb], PO, q 12 h). A diagnosis of sick sinus syndrome was made by the referring veterinarian following analysis of Holter monitor data collected during a 24-hour period. The Holter monitor data indicated that the dog had periods of sinus arrest of <3 seconds’ duration. No episodes of syncope occurred while the dog was wearing the Holter monitor. The dog was referred to the Veterinary Medical Center of the University of Florida for pacemaker implantation.

Physical evaluation of the dog revealed a grade 5/6 systolic heart murmur but no other abnormal findings. Thoracic radiography revealed moderate left atrial enlargement but no evidence of congestive heart failure. Assessment of an echocardiogram confirmed the clinical diagnosis of degenerative mitral valve disease with severe mitral insufficiency and moderate left atrial enlargement. Systolic function was considered normal. The tricuspid valve was thickened and degenerative with mild tricuspid insufficiency. Subjectively, the right atrium and right ventricle were of normal size. A 6-lead ECG was performed.

**ECG Interpretation**

The initial ECG revealed a sinus arrhythmia (Figure 1). Irregularity was secondary to fluctuations in vagal tone that may or may not have been associated with the respiratory cycle. A wandering pacemaker, characterized by alteration of P wave morphology, was also detected in this ECG. A wandering pacemaker and sinus arrhythmia are frequently detected concurrently because both are vagally mediated. The sinoatrial node is large in dogs (approx 4 cm in diameter), and vagal and sympathetic stimulation...
induce shifts in the sites of impulse originiation. This results in taller P waves when the heart rate is fast (ie, sympathetic stimulation shifts the pacemaker site cranially) and smaller P waves when the heart rate is slow (ie, vagal stimulation shifts the pacemaker site caudally).

Pacemaker implantation was not performed because a definitive diagnosis of sick sinus syndrome could not be made. The dog was anesthetized, and an implantable loop recorder was placed in the subcutaneous tissue of the left ventral region of the dog's thorax during a 10-minute procedure. The dog was kept on the same prescribed medications, and the owners were instructed regarding the use of the implanted device.

The dog was returned 2 weeks later following another episode of syncope. The loop recorder was interrogated by use of a programmer to evaluate any arrhythmias that might have occurred during the syncope event (Figure 2). Immediately prior to the collapse episode, tachycardia was detected (during which time the owner reported that the dog was excited and running around). This was followed immediately by profound bradycardia and a 6-second period of sinus arrest resulting in collapse, as indicated by the activation marking on the recorded data (identified as triangular markings on the trace). The initial activation occurred at the end of the 6-second period of sinus arrest, and the owner then activated the device several more times during the dog's collapse. The dog recovered fairly quickly, and heart rate returned to within reference limits. These ECG findings were most compatible with vasovagal syncope. In addition to the previous medications, atenolol (0.75 mg/kg [0.34 mg/lb], PO, q 12 h) was administered in an attempt to prevent tachycardia that might result in reflex bradycardia and vasodilation associated with the vasovagal syncope.

Two days later, the dog was returned to the Veterinary Medical Center because the syncopal episodes had become more frequent since commencement of treatment with atenolol. Administration of atenolol was discontinued, and administration of theophylline (10 mg/kg [4.5 mg/lb], PO, q 12 h) was initiated. Ten months after the initial evaluation, the owners reported that no further syncopal events had occurred and the implantable recorder was removed.

Discussion

Syncopal episodes can be categorized as those that occur as a result of cardiac-related causes, those that occur as a result of noncardiac-related causes, and those for which the cause cannot be determined. There are numerous noncardiac-related causes of syncope that include vasovagal syncope. There is 1 anecdotal report of an excitement-induced syncopal event in a dog; the owner reported that when the dog became excited, it "passed out." It was presumed that the dog had vasodepressor syncope as a result of increased vagal output to the peripheral vasculature that induced vasodilatation and profound hypotension. Vagally mediated syncope has been reported in humans. Pathophysiologically, vasovagal syncope includes stimulation of the cardiac C fibers. Panic, excitement, fright, or exercise can cause an increase in sympathetic tone that results in increases in heart rate and contractility, which stimulate cardiac C fibers. Stimulation of these cardiac C fibers induces an abnormal autonomic response resulting in vasodilatation and increased vagal tone. The combination of vasodilation and bradycardia can result in a reduction in cardiac filling, which ultimately leads to the development of syncope.

In hypovolemia and other conditions of reduced preload, the autonomic response may be exaggerated because sympathetic tone is increased. Increased sympathetic tone leads to hypercontractility of the volume-depleted ventricles and subsequent stimulation of the cardiac C fibers. The dog of this report also
had severe mitral insufficiency, which likely exaggerated the mechanoreceptor response. During times of excitement and high sympathetic tone, the regurgitant fraction could increase, resulting in a smaller left ventricular volume and stimulation of the cardiac C fibers.

In humans, treatment of vagally mediated syncope consists of drug administration and implantation of a pacemaker as well as patient education. Affected persons are made aware of factors that would predispose them to a syncopal event (eg, dehydration, stress, or an extremely warm environment) and are instructed regarding anxiety management. Owners of affected dogs should be similarly advised of potential triggers of syncopal episodes.

Drug treatments for humans and other animals with vasovagal syncope may include β-adrenoceptor blockers, selective serotonin reuptake inhibitors, fludrocortisone, midodrine, and theophylline. Pacemaker implantation is rarely effective for this disorder, as many of the clinical signs occur as a result of vasodilation, which would not be prevented by pacemaker implantation. β-Adrenoceptor blockers are widely prescribed to humans with syncope on the basis of the hypothesis that the reflex syncope is initiated by excessive stimulation of ventricular contractile force. In addition, β-adrenoceptor blockers may also prevent epinephrine-induced arterial vasodilatation—a potential mediatior of hypotension in reflex syncope—through αβ-adrenoceptor blockade. β-Adrenoceptor blockade can potentially worsen syncope through negative chronotropic effects and atrioventricular node-blocking effects, which likely developed in the dog of this report. Recent studies in humans revealed that recurrent syncope was more frequent in patients receiving β-adrenoceptor blocker treatment than it was in patients receiving a control conservative treatment.

Midodrine hydrochloride, a direct α1-adrenoceptor agonist and vasoconstrictor that is approved in the United States for the treatment of symptomatic orthostatic hypotension in humans, is also administered for the treatment of recurrent vasovagal syncope. Because serotonin may have a role in regulating sympathetic nervous system activity, selective serotonin-reuptake inhibitors have been proposed as a potential treatment, and results of open-label studies have indicated that these agents may decrease recurrence of vasovagal syncope. Fludrocortisone (9α-fluorohydrocortisone) is a potent mineralocorticoid with minimal glucocorticoid effect. It is the most important agent for the treatment of chronic orthostatic hypotension because of its ready availability, low cost, and efficacy. This drug increases blood volume by stimulating renal sodium retention. It also sensitizes the vasculature to circulating catecholamines.

In a recent report, it was suggested that high circulating concentrations of adenosine might be present in a subset of humans in whom paroxysmal atrioventricular block is a prominent consequence of reflex syncope. The actions of adenosine, both direct and indirect, are antagonized at the receptor level by theophylline. Theophylline is a commonly used bronchodilator that also has sympathomimetic effects; its use has been evaluated in small clinical trials of humans with reflex syncope. As the role of adenosine in recurrent vasovagal events becomes known, treatment may be directed towards counteracting or blunting the effects of adenosine. Although this treatment is not currently considered first line, it may become more commonplace if research reveals that adenosine is a trigger of the vasovagal cascade.

References