ECG of the Month

A 11-year-old (44.5-kg [97.9-lb]) sexually intact male Weimaraner was evaluated at the Veterinary Teaching Hospital of the University of Georgia because of a 2-day history of weakness and difficulty rising from a lying position associated with all 4 limbs. Several years earlier, the dog had bilateral cranial cruciate ligament disease and tibial plateau leveling osteotomies were performed bilaterally. Three months prior to evaluation at the hospital, the right tibial plateau leveling osteotomy plate was removed because of lameness and suspected infection. Electrocardiographic abnormalities were not detected during any of the dog’s previous anesthetic episodes. The dog had no other pertinent medical history.

Initial physical examination findings were unremarkable, and the dog’s heart rate was 112 beats/min. During auscultation of the thoracic cavity, no murmur or arrhythmia was detected and lung sounds were apparently normal bilaterally. Results of a neurologic examination were consistent with a lesion affecting C6 through T2 spinal cord segments. Abnormalities detected via a CBC and serum biochemical analyses included mild leukocytosis characterized by a mature neutrophilia (12.5 × 10³ cells/µL; reference range, 2.9 × 10³ cells/µL to 12 × 10³ cells/µL), monocytosis (2.0 × 10³ cells/µL; reference range, 0.1 × 10³ cells/µL to 1.4 × 10³ cells/µL), and mild hyperglycemia (124 mg/dL; reference range, 77 to 120 mg/dL). Results of a urinalysis were also within reference limits. Radiography of the thorax and cervical portion of the vertebral column revealed no abnormalities, with the exception of a narrow intervertebral disk space at C6-7.

To perform magnetic resonance imaging of the cervical portion of the vertebral column, the dog was anesthetized. After administration of glycopyrrolate (0.005 mg/kg [0.002 mg/lb], IV), butorphanol (0.2 mg/kg [0.09 mg/lb], IV), and midazolam (0.2 mg/kg, IV), anesthesia was induced with propofol (4 mg/kg [1.8 mg/lb], IV) and maintained via inhalation of isoflurane and oxygen. During anesthesia, pulse oximetry, assessment of end-tidal carbon dioxide tension, and noninvasive blood pressure monitoring were performed. Throughout the anesthetic episode, the dog’s heart rate was 120 to 130 beats/min with a regular rhythm (determined from the pulse wave provided by the pulse oxymeter). End-tidal carbon dioxide tension was 25 to 40 mm Hg. Systolic blood pressure ranged from 60 mm Hg at induction of anesthesia to 110 mm Hg throughout the remainder of the anesthetic episode. Magnetic resonance imaging revealed herniated intervertebral disks at the C5-6 and C6-7 disk spaces, which had resulted in compression of the spinal cord. The lesions were dynamic with traction. On extubation, the dog’s rectal temperature was 32.2°C (93.7°F). The dog was transferred to the intensive care unit of the hospital for monitoring and active warming by use of a forced-air patient warming unit. Cardiac auscultation performed during the dog’s recovery from anesthesia revealed tachycardia and an abnormal cardiac rhythm. Electrocardiography was performed to evaluate the tachyarrhythmia.

ECG Interpretation

During recovery from anesthesia, a 6-lead ECG was recorded (Figure 1). At the time of this initial recording, the dog’s rectal temperature was 32.4°C (90°F). The ECG traces revealed an irregularly irregular rhythm with a mean heart rate of 280 beats/min. Although P waves were absent, fibrillation waves (f waves) were detectable as fine undulations along the baseline. The QRS complexes were normal in appearance with variable amplitude and variable R-R intervals. The ECG diagnosis was atrial fibrillation.

The lack of discernable P waves was consistent with generation of an electrical impulse from an area other than the sinoatrial node. The QRS complexes were normal in appearance (not wide or bizarrely shaped), indicating that the arrhythmia was supraventricular in origin. The irregularly irregular rhythm associated with atrial fibrillation was attributable to the occasional conduction of fibrillation waves through the atrioventricular node, which then triggered ventricular depolarization.

Figure 1—Lead II ECG recording obtained from an 11-year-old Weimaraner that developed hypothermia following anesthesia, during which a magnetic resonance imaging evaluation was performed to investigate the cause of a 2-day history of weakness and difficulty rising from a lying position associated with all 4 limbs. There is an absence of P waves, but f waves (arrows) are visible. Paper speed = 50 mm/s; 1 cm = 1 mV.
No antiarrhythmic treatment was initially administered because there were no signs of hemodynamic compromise. Continuous ECG monitoring was initiated and active warming continued until the dog’s rectal temperature returned to within reference range. Echocardiography was then performed to evaluate the dog for underlying heart disease. At the time of the echocardiographic evaluation, sinus arrhythmia was evident and a small amount of mitral and tricuspid valve regurgitation was identified. In the right parasternal long-axis view, the left atrial diameter was 54.77 mm and the right atrial diameter was 36.53 mm. In M mode of the echocardiographic evaluation, the left ventricular dimension during diastole and systole was 44.04 mm and 28.86 mm, respectively. Fractional shortening was 34.48%, and the E point to septal separation was 5.85 mm (as determined in M mode). The echocardiographic findings were considered within reference ranges, and the only abnormalities detected were mild mitral and tricuspid valve regurgitation. No treatment was prescribed at that time.

Subsequently, ECG monitoring revealed that the heart rate varied between atrial fibrillation and sinus rhythm with atrial premature contractions and paroxysmal atrial tachycardia. There was a progression toward more frequent sinus rhythm and less frequent atrial fibrillation over an 8-hour interval. The atrial fibrillation spontaneously resolved after approximately 8 hours following return of the dog’s rectal temperature to within reference range (Figure 2).

Twenty-four hours following conversion to sinus rhythm, a Holter monitor was placed on the dog. The continuous ECG data revealed 11 paroxysms of ventricular tachycardia with a heart rate of 220 to 240 beats/min. There were instances of sustained and nonsustained supraventricular tachycardia (300 beats/min; Figure 3) with occasional ventricular premature contractions (isolated couplets and triplets). For treatment of supraventricular and ventricular tachyarrhythmia, the dog was administered sotalol (a class III potassium channel blocking and β-adrenergic receptor antagonist) at a dosage of 0.45 mg/kg (0.2 mg/lb), PO, every 12 hours.

**Discussion**

Atrial fibrillation is the most common cause of supraventricular tachycardia in dogs. Atrial fibrillation in dogs can develop secondary to underlying cardiac disease that leads to volume overload and atrial enlargement. Such causes include dilated cardiomyopathy, atrioventricular valve endocardiosis, or congenital heart defects (eg, patent ductus arteriosus) that result in volume overload. Transient atrial fibrillation has been associated with a shortened atrial refractory period caused by increased vagal tone, such as that which occurs during anesthesia with a narcotic drug in dogs that have not been premedicated with an anticholinergic agent.1 Atrial fibrillation can also develop in association with certain systemic conditions such as hypoadrenocorticism and gastric dilatation–volvulus. Some large- and giant-breed dogs may develop atrial fibrillation despite the lack of underlying heart disease. There have been a few reports1,3 of atrial fibrillation secondary to hypothermia in dogs. For dogs and cats, hypothermia is typically defined as body temperature (rectal or esophageal) < 35°C (95°F). At low body temperatures, circulation is altered as a result of vasoconstriction and decreased cardiac output, which can lead to myocardial injury and abnormal electrical conduction. Arrhythmias associated with hypothermia include atrial fibrillation, ventricular premature contractions, ventricular tachycardia, and ventricular fibrillation.2 Hypoxemic damage to the myocardium of the right atrium could progress to abnormal automaticity and the generation of impulses from areas other than the sinoatrial node.4

Potential sequelae of atrial fibrillation include decreased cardiac output, decreased tissue perfusion, and cardiac failure, which may be clinically evident as...
lethargy, ataxia, dyspnea, syncope, or death. The goal of treatment is to improve the patient’s hemodynamic condition either by terminating the arrhythmia or increasing cardiac output by slowing the heart rate and increasing diastolic filling time. Methods of treating supraventricular tachycardia include vagal maneuvers to slow the heart rate, administration of pharmacologic agents, or electrical defibrillation. Medications used to treat atrial fibrillation include digoxin, class II drugs (eg, β-adrenergic receptor antagonists), class III drugs (eg, sotalol and amiodarone), and class IV drugs (calcium channel blockers), all of which slow conduction of impulses through the atrioventricular node, thereby reducing the ventricular rate. In 1 report, hypothermia-induced atrial fibrillation in dogs was successfully converted to sinus rhythm via administration of quinidine, a class Ia drug that blocks sodium channels and prolongs the duration of the action potential. Conversion of atrial fibrillation to sinus rhythm in dogs is best accomplished with class III drugs or via electrical methods and is usually reserved for dogs with apparently normal-sized atria.

In the dog of this report, hypothermia likely played an important role in the development of atrial fibrillation. However, underlying cardiac disease, such as dilated cardiomyopathy or atrial enlargement secondary to mitral or tricuspid valve regurgitation, and subsequent volume overload cannot be completely ruled out as the source of the supraventricular tachyarrhythmia. Additionally, the dog’s neurologic disease may have been a contributing factor. Dogs undergoing decompressive surgery of the cervical portion of the spinal cord have an increased risk of developing dysrhythmia, ventricular premature contractions, and bradyarrhythmia. Alternatively, the dog’s neurologic disease may have had a direct impact on the myocardium. Myocardial necrosis may have been the inciting cause of the supraventricular tachyarrhythmia. Thus, the potential contribution of the underlying neurologic disease cannot be discounted as a possible cause for the dog’s arrhythmia. Follow-up echocardiography and Holter monitor assessments would be indicated to determine whether the supraventricular and ventricular tachycardia persist in the dog of this report.

References


Correction: Dietary fats and the skin and coat of dogs

In the report “Dietary fats and the skin and coat of dogs” (J Am Vet Med Assoc 2007;230: 1641–1644), the conversion for the dosage in the fourth line from the end of the section “Dietary Supplementation of n-3 Fatty Acids for Skin Disorders” on page 1642 was incorrect. The correct conversion for supplementation of a diet with marine fish oil should read as follows: 1 g of fish oil/4.54 kg (1 g of fish oil/10 lb) of body weight.