ECG of the Month

The Academy of Veterinary Cardiology sponsors this feature. Readers of the *JAVMA* are invited to submit contributions. Contributions should include a brief description of the case (150 words); good quality contrast glossy photographs (5 X 7 in) of tracings, with the components of a QRS complex labeled; figure legends with information on ECG lead, paper speed, and voltage calibration; an ECG interpretation; and a discussion of the abnormality. Two hard copies of the manuscript and each figure must be submitted, along with an electronic copy on a 3.5-in PC-formatted disk. Submissions that are complete will be sent to the feature coordinator, Dr. Robert Hamlin, at The Ohio State University for review.

A 7-year-old 51.5-kg (113.4-lb) sexually intact male Golden Retriever was referred to the Mississippi State University Animal Health Center for evaluation of a suspected pericardial effusion. The dog was brought to the referring veterinarian 2 days earlier following an episode of acute collapse. On physical examination, the dog was lethargic, tachypneic, and dyspneic. Auscultation of the thorax revealed loss of clinically normal lung sounds in the ventral lung fields as well as muffled heart sounds. Jugular pulses were evident, whereas femoral pulses were not palpable. The abdomen was large and pendulous. There was a moderate amount of edema found in the pelvic limbs as well as the scrotum. Rectal temperature was slightly high at 39.6 C (103.3 F). Results of serum biochemical analyses, CBC, and urinalysis were within reference ranges. Electrocardiography revealed normal sinus rhythm with a slightly prolonged QRS complex (**Fig 1**). Thoracic and abdominal radiography indicated severe abdominal and bilateral pleural effusion. Echocardiography indicated a moderate pericardial effusion with a thick pericardium (0.5 cm; **Fig 2**). Attempted pericardiocentesis was unsuccessful. Thoracocentesis was performed, and a total of 2.7 L of hemorrhagic fluid was removed from the pleural space. A thoracotomy tube was placed to facilitate drainage of pleural fluid. The presumptive diagnosis was constrictive pericarditis, and an exploratory thoracotomy with possible subtotal pericardectomy was scheduled following stabilization.

Four days after admission, the dog was stable enough for surgery. Medication before anesthesia consisted of oxymorphone (0.07 mg/kg [0.03 mg/lb] of body weight, IV) and diazepam (1.0 mg/kg [0.5 mg/lb], IV). Anesthesia was induced with etomidate (1.0 mg/kg [0.5 mg/lb], IV) and maintained with isoflurane in oxygen. A median sternotomy was performed. On opening the thoracic cavity, the presence of pleural fluid was confirmed. The pericardium was thick and cartilage-like in appearance. A subtotal pericardectomy was performed. Medications for signs of pain after surgery consisted of a morphine drip (0.03 mg/kg/h [0.014 mg/lb/h], IV) for 16 hours followed by oxymorphone (0.04 mg/kg [0.018 mg/lb], IV) given as needed, and bupivicaine given through the chest tube (1.2 mg/kg [0.55 mg/lb]) every 6 hours. Cefazolin (20 mg/kg [9.0 mg/lb], IV) was also given every 6 hours. Cardiac arrhythmias were not observed during the perioperative period. Subsequent microbiologic culture of pleural and pericardial fluids and of pericardial tissue resulted in moderate growths of *Aspergillus nigar*. A histopathologic diagnosis of active chronic pericarditis was made. Final diagnosis was cardiac tamponade secondary to *A nigar*-induced constrictive pericarditis.

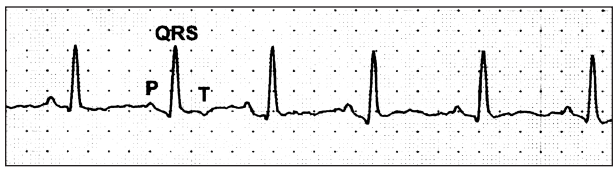

Figure 1—Lead-II ECG from a 7-year-old Golden Retriever that was referred for evaluation of a suspected pericardial effusion. At the time of admission, the dog had a heart rate of 120 beats/min with a normal sinus rhythm. Notice the slightly prolonged QRS interval (0.07 seconds; reference range ≤ 0.06 seconds). Paper speed = 50 mm/s; 1 cm = 1 mV.

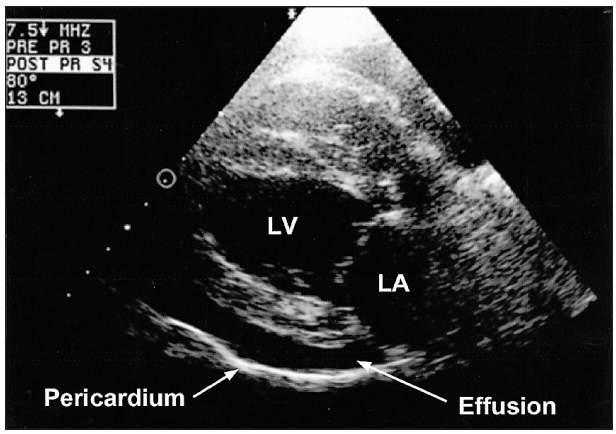

Figure 2—Echocardiograph of the right parasternal long-axis 4-chamber view of the heart of the same 7-year-old Golden Retriever as in Figure 1 at the time of admission. Notice the moderate amount of pericardial effusion and the thick pericardium (0.5 cm width). Pericardial effusion resulted in cardiac tamponade. LA = Left atrium; LV = Left ventricle.

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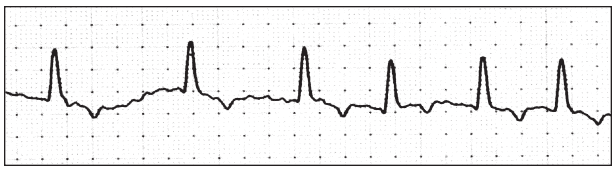

Figure 3—Lead-II ECG from the same 7-year-old Golden Retriever as in Figure 1 sixteen hours after pericardectomy. The dog had a heart rate of 120 to 140 beats/min. Notice that the rhythm is irregularly irregular, the QRS complexes are slightly large and have subtle variations in shape and size, and there are f waves. Paper speed = 50 mm/s; 1 cm = 1 mV.

Approximately 16 hours following subtotal pericardectomy, an arrhythmia developed (**Fig 3**). The arrhythmia resolved in approximately 14 hours without treatment and did not recur. Four days following surgery, the dog was discharged to the owner, and a long course of itraconazole was prescribed.

ECG Interpretation

The ventricular rate from the ECG readout (Fig 3) is 120 to 140 beats/min, the rhythm is irregularly irregular, the QRS complexes are slightly large and have subtle variations in shape and size, and there are f waves present. These are classic electrocardiographic signs of atrial fibrillation.

Discussion

Atrial fibrillation is most commonly seen in middle-aged male dogs with severe congestive heart failure related to **atrioventricular (AV)** valvular insufficiencies or cardiomyopathies but has also been reported with patent ductus arteriosus, ventricular septal defects, pulmonic or aortic stenosis, cardiac catheterization, doxorubicin toxicosis, constrictive pericarditis, and trauma.[1-7] Atrial fibrillation is more commonly seen in large breeds of dogs (Doberman Pinschers, Great Danes, and Irish Wolfhounds) rather than cats and toy dog breeds.[7,8] This likely results from the need for a critical mass of atrial tissue to be present before atrial fibrillation can be sustained.[8] Atrial fibrillation is characterized by a large number of disorganized atrial impulses that bombard the AV node.[9,10] Atrial fibrillation is usually the result of multiple ectopic foci in the atria that have a firing rate greater than that of the **sinoatrial (SA)** node. Because there is no organized atrial contraction in patients with atrial fibrillation, there is a resultant loss of atrial kick. Atrial kick is the coordinated emptying of the atrial contents associated with atrial contraction at the end of ventricular diastolic filling and is responsible for approximately 20% of the ventricular end diastolic volume.[9,10] Ventricular response rate is determined by the atrial rate, autonomic nervous tone, and the refractoriness of the AV node.[8,11] Typically, with atrial fibrillation, the atrial rate is 300 to 500 beats/min, and the ventricular rate often exceeds 180 beats/min.[10,12] The loss of atrial kick and the rapid ventricular rate can combine to reduce diastolic ventricular filling and thus decrease cardiac output by as much as 20 to 30%, leading to weakness, collapse, or syncopal episodes.[9,10]

On an ECG, atrial fibrillation is associated with loss of typical P waves and rhythm.[7] Instead of P waves, there are small to large oscillations called fibrillatory or f waves that create an irregular undulating baseline.[1,7-9] The QRS complexes typically are normal in appearance, because ventricular conduction still develops via fairly usual pathways, but they may vary in amplitude and duration as the result of aberrant ventricular conduction and associated bundle branch block.[7-9] Aberrant conduction results from the variable timing of the atrial interaction with the AV node and the refractory state of the ventricle.[8] If the atrial impulse interacts when the ventricular conduction pathways are completely refractory, then there is no ventricular contraction. Conversely, if the conduction pathways are completely recovered, then there is conduction with a QRS complex that is normal in appearance. If the ventricular conduction pathways are between absolute refractoriness and complete recovery, then aberrant ventricular conduction can develop, giving the QRS complex an altered size and appearance.[8,9] Common differentials for atrial fibrillation include atrial premature complexes, AV junctional tachycardia with AV block, supraventricular tachycardia with AV block, or atrial flutter with AV block.[9]

Treatment of atrial fibrillation revolves around controlling cardiac failure and reducing ventricular rates.[1,9] A major goal of treatment is to reduce the ventricular rate to < 140 beats/min.[9] Conversion of atrial fibrillation to a normal sinus rhythm in dogs with a recent development of fibrillation can be attempted by use of quinidine, verapamil, or electrical cardiac defibrillation (cardioversion).[7,9,13] Conversion of atrial fibrillation is, however, only likely to be effective in dogs without severe cardiac disease. Because most dogs with atrial fibrillation have advanced cardiac disease, conversion to a normal sinus rhythm is rarely attempted; instead, the main goal of treatment is usually to slow the ventricular response to the bombardment of atrial impulses with digoxin, beta blockers such as propranolol, or calcium channel blockers such as diltiazem.[9] In the dog of our report, initial treatment was not warranted, because ventricular rates never exceeded 140 beats/min despite the presence of atrial fibrillation. If the dog's atrial fibrillation had persisted, attempted conversion to a normal sinus rhythm with either drugs or electrical cardioversion would have been indicated.

Common electrocardiographic findings associated with constrictive pericarditis are similar to those reported for pericardial effusion and include decreased QRS complex amplitude (< 1.0 mV), prolonged P wave duration, and electrical alternans.[2,14-16] Although most dogs with pericardial disease present with either a normal sinus rhythm or a sinus tachycardia, a small number can present with atrial fibrillation.[9]

In our report, the dog presented with *A nigar*-induced constrictive pericarditis and had a normal sinus rhythm with a mildly elongated QRS complex. This cardiac rhythm converted to atrial fibrillation approximately 16 hours after subtotal pericardectomy. To our knowledge, atrial fibrillation has not previously been reported as a possible complication following subtotal pericardectomy but anecdotally does occasionally develop following pericardiocentesis. One recent paper reported atrial fibrillation following centesis of a hemorrhagic pericardial effusion secondary to

brodifacoum toxicosis, although the atrial fibrillation was only one of a number of different arrhythmias.[14]

In the dog of our report, atrial fibrillation may have been precipitated by stretching of the right atrium, as cardiac filling improved during and following alleviation of cardiac tamponade. Results of recent studies indicate that the effective refractory period of the thin portion of the atrium is substantially increased following stretching by infusion of high volumes of saline (0.9% NaCl) solution during a short period.[15] Atrial stretch and an increased effective refractory period are felt to be conducive to the development of atrial fibrillation.[15] Atrial stretching may also lead to localized myocardial hypoxia, which could in turn create ectopic foci that could then act on an atrium primed for the development of atrial fibrillation. Another possible cause for the development of atrial fibrillation could have been an alteration in myocardial membrane potentials associated with inflammation. Although only a small number (approximately 3%) of dogs with pericardial disease are admitted in atrial fibrillation, atrial fibrillation is much more common in humans with pericardial disease, especially people with constrictive pericarditis (up to 35% of affected humans have atrial fibrillation).[4,5,9] Because the atrial fibrillation in the dog of our report developed transiently several hours following pericardectomy, the arrhythmia is more likely to be associated with the relief of the cardiac tamponade rather than the preexisting pericarditis. The delay in the appearance of the arrhythmia may have been related to the use of isoflurane for anesthesia, which increases the atrial fibrillation threshold.[16] Regardless of the cause of the arrhythmia, we believe that all dogs should be closely monitored for the development of atrial fibrillation following pericardiocentesis or subtotal pericardectomy. Should the arrhythmia persist or substantially impair cardiac output, treatment with quinidine, verapamil, or cardioversion may be indicated in an attempt to convert the atrial fibrillation back to normal sinus rhythm.

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