ECG of the Month

A 7-year-old 47.3-kg (104.1-lb) spayed female Mastiff with an arrhythmia was referred for cardiac evaluation prior to an elective orthopedic procedure. Historical medical problems included complete surgical excision of a granulosa cell tumor and a colonic stromal neoplasm via 2 separate laparotomies. Clinical signs of cardiac disease had not been observed by the owners. Thoracic auscultation revealed an abnormal heart rhythm, which included long pauses between beats with periods of paroxysmal tachycardia. The heart sounds were considered normal, and no murmur was ausculted. The strength of the femoral arterial pulse was variable. An echocardiogram revealed normal cardiac dimensions and function. Electrocardiography was performed.

ECG Interpretation

A lead II ECG revealed atrial fibrillation with third-degree atrioventricular block (AVB) and a slow ventricular escape rhythm that was coupled with ventricular premature contractions. Narrow supraventricular QRS complexes were occasionally observed; these complexes appeared to be of atrioventricular (AV) junction or His bundle origin (Figure 1). Because of the complexity of the rhythm, lack of clinical signs, and unremarkable echocardiographic findings, treatment was not initiated at that time. Reevaluation in 6 weeks was recommended to the owners.

The dog was lost to follow-up until the owners returned the dog for evaluation because of restlessness and panting 5 months later. At that time, the dog was weak and lethargic and had moderate abdominal distension. Oral mucous membranes were pink and moist, and capillary refill time was 2 seconds. Thoracic auscultation revealed a grade 2/6 systolic murmur with a left apical point of maximum intensity, heart rate of 40 beats/min with low femoral pulse pressure, and resting tachypnea (60 breaths/min) with diffusely harsh-sounding lung fields. Electrocardiography was repeated, and an ECG diagnosis of atrial fibrillation with third-degree AVB and an escape complex rate of 42 beats/min was made (Figure 2). In lead II traces, a bigeminal pattern was evident in which each escape complex was consistently followed by a wide QRS complex with a right bundle-branch block configuration. Administration of atropine (0.04 mg/kg [0.018 mg/lb], IV) did not result in any change detectable via ECG. Thoracic radiography revealed generalized cardiomegaly, mild pleural effusion, and a moderate interstitial pattern. An echocardiographic examination revealed a moderately large right atrium and markedly large left atrium, mild mitral valve regurgitation, and moderate biventricular eccentric hypertrophy; fractional shortening was within reference limits. A lim-

Figure 1—Lead II ECG trace obtained from 7-year-old spayed female Mastiff with an arrhythmia that was referred for cardiac evaluation prior to an elective orthopedic procedure. Notice that the heart rate is highly variable with long (1.3-second) pauses between ventricular depolarizations and periods of paroxysmal polymorphic ventricular tachycardia. Two of the initiating complexes (black arrows) are narrow complex depolarizations with positive deflections, consistent with third-degree atrioventricular block (AVB) with junctional escape complexes to which are coupled ventricular premature depolarizations. Paper speed = 50 mm/s; 1 cm = 1 mV.

Figure 2—Lead II ECG trace obtained from the dog in Figure 1 five months later. Notice the coarse atrial fibrillation with complete AVB. There are 3 narrow (positive deflection) escape complexes that are of AV junctional or His bundle origin, followed by a wide bizarre complex with a consistent 0.30-second R-R interval. The R-R interval between initiating escape complexes is 1.44 seconds (heart rate, 84 beats/min). Paper speed = 50 mm/s; 1 cm = 1 mV.
itied ultrasonographic examination of the abdomen revealed hepatic congestion and moderate abdominal effusion that was consistent with right-sided heart failure. Results of serum biochemical analyses, CBC, and total thyroxine concentration assessment were all within reference limits. The dog was seronegative for *Borrelia burgdorferi*.

On the basis of clinical and diagnostic findings, pacemaker implantation was deemed necessary. Furosemide was administered (2 mg/kg [0.91 mg/lb], IV, q 6 h) during the 24-hour period before induction of anesthesia, and the clinical signs of respiratory distress largely resolved. Under fluoroscopic guidance, a ventricle-paced, ventricle-sensed, ventricle-disabled, rate-responsive (VVIR) bipolar pacemaker was implanted transvenously via the right external jugular vein. The dog initially developed ventricular tachycardia when pacer stimulation was commenced, but this resolved in the immediate postoperative period following administration of sotalol (1.7 mg/kg [0.77 mg/lb], PO, q 12 h). The dog was subsequently discharged for outpatient care; the owners were instructed to administer furosemide (1.7 mg/kg, PO, q 12 h), sotalol (1.1 mg/kg [0.50 mg/lb], PO, q 12 h), and enalapril (0.2 mg/kg [0.09 mg/lb], PO, q 12 h). Subsequent reevaluations at 1 and 3 weeks after the surgical procedure revealed appropriate pacing (Figure 3) and resolution of biventricular heart failure. Treatment with all cardiac medications was eventually discontinued, and the dog was alive at the last reevaluation, 15 months after pacemaker placement.

**Discussion**

In the dog of this report, the initial ECG examination revealed atrial fibrillation with third-degree AVB and an AV junctional or His bundle escape rhythm to which premature ventricular depolarizations were coupled. Because the dog had no clinical signs of cardiac disease, therapeutic intervention was not instituted after the initial evaluation to avoid the risk of abolishing the escape rhythm or exacerbating the polymorphic ventricular tachycardia. Subsequent ECG analysis performed 5 months later revealed atrial fibrillation with third-degree AVB and a narrow QRS complex escape rhythm. A bigeminal pattern was evident in which each escape complex was consistently followed by a wide QRS complex that had a right bundle-branch block configuration. Electrocardiographically, third-degree AVB is defined by P waves that are not associated with QRS complexes. The QRS complexes result from an escape focus that is located distal to the AVB. In cases of atrial fibrillation, there is an absence of P waves, which have been replaced by the F waves associated with atrial fibrillation. When AV conduction is normal, atrial fibrillation results in an irregular tachycardia that is supraventricular in origin. The apparent lack of F-wave conduction is consistent with a complete or third-degree AVB.

An escape rhythm initiated by an AV junctional or ventricular subsidiary pacemaker normally develops when more proximal sites fail to initiate or effectively conduct supraventricular depolarization. In the dog of this report, the ventricular escape rhythm was characterized by narrow QRS complexes; the complexes had predominantly positive deflection in lead II, consistent with an escape focus of AV junctional or high His bundle origin that followed the normal lower ventricular conduction pathways. Each narrow complex was consistently followed by a wide QRS complex that had predominantly negative deflection in lead II; the wide negative deflection QRS morphology suggested an origin within the left ventricle (R-R interval, 0.30 seconds). The regular fixed coupling of the 2 types of complexes was indicative of a triggered activity or reentry mechanism within the ventricular myocardium. Although the diagnostic possibility of second-degree AVB with ventricular bigeminy cannot be excluded, the regular R-R interval made this less likely.

It is interesting that the normal clinical consequences of atrial fibrillation were not initially evident in the dog because of the presence of concurrent AVB. Typically, the loss of atrial systolic contraction decreases late diastolic ventricular filling, resulting in preload reduction and decreased cardiac output. Furthermore, high ventricular rates that are commonly associated with atrial fibrillation further compromise diastolic filling and may result in tachycardia-induced cardiomyopathy. The presence of third-degree AVB in the dog of this report prevented the development of tachycardia. The long diastolic filling period associated with the slow escape rhythm resulted in a relatively increased preload, which may explain the dog's relatively long period of clinical compensation (5 months' duration).

In dogs, atrial fibrillation is most commonly associated with underlying heart disease and left atrial enlargement; however, atrial fibrillation alone can develop and is most frequently detected in giant-breed dogs. Presently, there is no consensus on the etiopathogenesis of atrial fibrillation, but a large atrial surface area or mass is thought to be required to sustain the arrhythmia.
Third-degree AVB is attributable to altered conduction of atrial depolarization at the level of the AV node. 

Treatment of dogs with complete AVB includes a thorough attempt to elucidate and address the underlying cause. Possible etiologies of complete AVB in dogs include congenital defects (eg, aortic stenosis, ventricular septal defect, or isolated AVB), infiltrative disease of the AV node (eg, neoplasia or amyloidosis), excessive vagal tone, myocardial infarction, bacterial endocarditis, idiopathic fibrosis, hypothyroidism, infection (eg, *Trypanosoma cruzi* infection [Chagas disease] and *B burgdorferi*-associated myocardiitis), electrolyte disturbances (eg, hyperkalemia), or pharmacotherapy (eg, administration of digitalis, β-adrenergic receptor blockers, or calcium channel blockers). However, the etiology of third-degree AVB is uncommonly elucidated antemortem. Treatment with anticholinergic agents can be attempted, but third-degree AVB is usually refractory to such medications. Pacemaker implantation is generally required to resolve clinical signs and provides a more favorable prognosis for duration of survival. In the dog of this report, successful transvenous implantation of a VVIR pacemaker resulted in improved cardiac performance and led to eventual resolution of biventricular congestive heart failure. Intuitively, the increased cardiac output associated with an increased heart rate after pacing should resolve signs of bradycardia-induced congestive heart failure in the absence of substantial myocardial or anatomic dysfunction. Chronic bradycardia-induced heart failure and its resolution via ventricular pacing are described anecdotally but infrequently reported in the veterinary medicine literature.

References