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 13-year-old spayed female Dalmatian was referred because of tachypnea and weakness. The owner reported that the dog had been hospitalized 3 weeks previously, and at that time a diagnosis of congestive heart failure secondary to dilated cardiomyopathy was made, and the dog was treated. A written summary of the previous hospitalization revealed that the dog had an episode of paroxysmal ventricular tachycardia that was responsive to lidocaine administered IV and that a resting ECG obtained at the time of hospital discharge revealed no evidence of ventricular ectopy. The dog had received standard dosages of digoxin, enalapril, and furosemide for the preceding 3 weeks. On physical examination, the dog had signs of depression and was unable to stand for more than a few moments. Respiration was labored at a rate of 60 breaths/min, and arterial pulses were weak but regular at a rate of 180 beats/min. The mucous membranes were pale pink with a 2- to 3-second capillary refill time. Auscultation of the thorax revealed bilateral pulmonary crackles, and a II/VI holosystolic murmur was heard best at the point of the left cardiac apex. The remainder of the physical examination was unremarkable. An ECG was recorded (**Fig 1**).

ECG Interpretation

Examination of the ECG revealed sinus tachycardia with **left bundle branch block (LBBB)**, characterized by P mitrale and P pulmonale. The wide bizarre QRS complexes in this case could be mistaken for ventricular tachycardia, especially in light of the history and physical examination findings (Fig 1). The regular P waves consistently coupled with each QRS complex effectively rule out this possibility. The frequency, morphologic characteristics, and normal orientation of the P waves, combined with the consistent and reasonable PR interval, confirm the diagnosis of sinus tachycardia.

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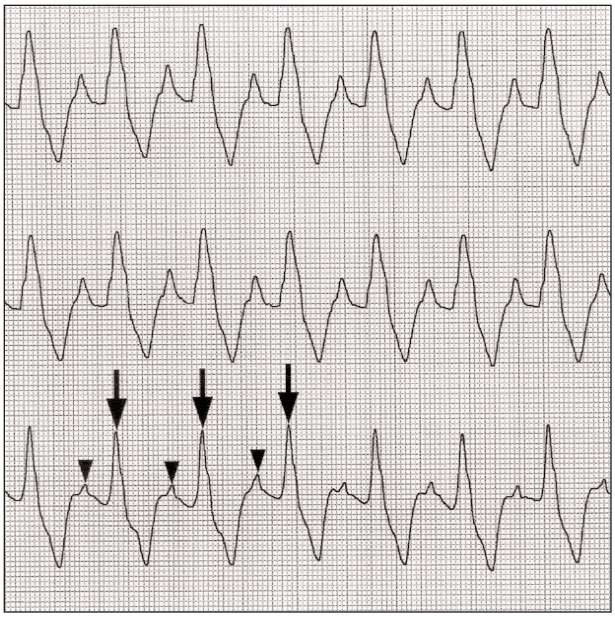

Figure 1—Leads II (top), aVF (center), and V3 (bottom) of an ECG from a dog with left bundle branch block. Notice the P waves (arrowheads) regularly coupled with the wide QRS complexes (arrows). Paper speed = 50 mm/s. 1 cm = 1 mV.

The distinction between a ventricular conduction disturbance (such as LBBB) and ventricular ectopy is clinically important, because ventricular tachycardia in a hemodynamically compromised patient prompts the administration of antiarrhythmic drugs with potentially adverse hemodynamic or proarrhythmic effects. Because the underlying rhythm in this case was sinus, treatment was aimed at improving cardiac output and relieving congestive heart failure, which was subsequently confirmed radiographically.

Discussion

The wide QRS complexes (100 milliseconds) in the ECG from the dog of this report are caused by a delay in the specialized conduction system that prolongs ventricular depolarization. In dogs, the specialized ventricular conduction system consists of Purkinje cells that conduct the electrical impulse extremely rapidly once the **atrioventricular (AV)** node has been depolarized, initiating a stereotypical pattern of ventricular depolarization. The anatomic distribution of these rapidly conducting bundles of Purkinje fibers (called bundle branches), coupled with the subsequent depolarization of the ventricular myocytes, gives rise to the normal QRS complex and orientation recorded on the ECG.[1] In dogs, the entire process of ventricular depolarization generally lasts < 50 milliseconds. Although it may take slightly longer for the ventricles to depolarize in large dogs or those with ventricular enlargement and myocardial hypertrophy, the

duration of the QRS complex associated with such hypertrophy seldom exceeds 60 milliseconds.

Left bundle branch block describes a considerable conduction delay or block in the main left bundle branch such that the impulse fails to be distributed rapidly to the normal arborizations of the left main bundle branch (the left posterior and anterior fascicles). A delay or block in conduction to these specialized pathways results in slower than normal depolarization of the left ventricular myocardium.[2] In LBBB, the ECG is characterized by wide QRS complexes (> 80 milliseconds in most breeds and > 70 milliseconds in toy breeds) with normal complex orientation (normal electrical axis). The QRS complex in LBBB is, therefore, positive in leads I, II, III, aVF, CV6LL, and CV6LU and inverted in aVR, aVL, and CV5RL.[1]

Similarly, a markedly widened but normally oriented QRS complex would also be present if a conduction block were located in the left posterior and anterior fascicles, rather than the main left bundle branch that gives rise to these pathways. However, there are indications in the ECG that the conduction disturbance is affecting the main left bundle branch. Normally, electrical depolarization of the main left bundle branch contributes to the initial activation of the interventricular septum (by the so-called septal perforators), represented electrocardiographically as a Q wave in leads I, II, or aVF. The absence of Q waves in the ECG from the dog of this report indicates that the electric activity is blocked above the level of the septal perforators, at the level of the main left bundle, which alters the normal pattern of early septal activation. A conduction disturbance at the level of both the left posterior and anterior fascicles would presumably still allow conduction through the initial part of the main left bundle branch, preserving the normal Q wave.

The left bundle branch is a robust structure; thus, damage severe enough to substantially affect conduction is generally associated with severe heart disease. Associated underlying disorders in dogs include dilated cardiomyopathy, subaortic stenosis, doxorubicin toxicosis, and ischemic injuries.[3] Studies[4-6] in humans have documented serious ventricular functional abnormalities in patients with isolated LBBB, compared with clinically normal controls, as well as in patients with LBBB and dilated cardiomyopathy, compared with patients with dilated cardiomyopathy alone. These patients with LBBB have shortened diastolic filling times, increased preejection and relaxation times, asynchronous interventricular septal motion, reduced left ventricular ejection fractions, reduced maximal **rate of change of left intraventricular pressure (dP/dt)**, and prolonged time to peak dP/dt, compared with their respective controls. The defects are potentially severe enough that pacemakers are sometimes implanted in an effort to restore synchrony and subsequently increase ventricular function.

The tall (P pulmonale) and wide (P mitrale) P waves seen in the ECG from the dog of this report are often manifestations of right and left atrial enlargement, respectively. In this case, the echocardiographic study confirmed the presence of biatrial enlargement. It should be stated, however, that neither P pulmonale nor P mitrale is a specific marker for atrial enlargement. The morphologic characteristics of P waves are subject to variation because of differences in breed, age, chest conformation, lead placement, conduction disturbances, heart rate, and presence of intrathoracic effusions.[1,3] The slightly prolonged PR interval (130 milliseconds) in this case could be normal, although it could also be related to the underlying conduction system disease or an effect of digoxin.

References

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