**ECG of the Month**

A 14-year-old spayed female mixed-breed dog was referred to the Clinica Veterinaria Malpensa because of frequent episodes of syncope (frequency, 1 to 5 episodes/d). At the initial evaluation, the dog appeared mildly depressed. The femoral pulse was bradyarrhythmic with a mean heart rate of 40 beats/min. Results of auscultation of the heart and lungs were considered normal, and no other clinical abnormalities were detected. Thoracic radiographic and echocardiographic findings were also considered normal. Twelve-lead surface ECG (6 peripheral standard leads and 6 precordial leads as previously described; Figure 1) was performed with the dog placed in right lateral recumbency.

**ECG Interpretation**

The 12-lead surface ECG tracing (Figure 1) revealed a severe bradyarrhythmia with a mean ventricular rate of 40 beats/min. The P-wave duration was 40 milliseconds (reference range, < 40 milliseconds) and amplitude was 0.4 mV (reference range, < 0.4 mV), with a sinus rate of 140 beats/min and a normal P-wave axis on the frontal plane (+80°; reference range, −18° to +90°). An atrioventricular (AV) conduction disturbance with lack of concordance between atrial and ventricular depolarization rate was evident. Only a few P waves were followed by ventricular QRS complexes with an AV conduction ratio of 2:1 (ie, 2 P waves [1 unconducted and 1 conducted] for 1 QRS complex) that sometimes evolved into advanced (or complete) AV block (AV conduction ratios of 3:1 and 4:1). The PQ interval for the conducted beats was 120 milliseconds (reference range, 60 to 130 milliseconds).

These features were consistent with a second-degree (2:1 type) AV block with episodes of advanced AV block. Moreover, intraventricular conduction disturbance was evident because of a prolongation of the QRS complexes (duration, 80 milliseconds; reference range, < 70 milliseconds) and tall, slurred, and delayed R waves in the inferior leads (II, III, and aVF) and left precordial lead tracings. The rate-corrected QT interval (QTc) was slightly prolonged (duration, 249 milliseconds; reference range, 150 to 240 milliseconds). The mean electrical axis of the QRS complex was +82°, which was within reference limits (+40° to +100°);

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**Figure 1—**Twelve-lead surface ECG recordings obtained from a dog that was evaluated because of frequent episodes of syncope (frequency, 1 to 5 episodes/d). A severe atrioventricular (AV) conduction disturbance is present with second-degree AV block (2:1 conduction ratio characterized by 1 unconducted and 1 conducted P wave [black arrows] to 1 QRS complex) that changes into high-grade second-degree AV block with > 1 unconducted P wave (white arrows). This condition induces a severe bradycardia (mean ventricular rate, 40 beats/min) with a normal sinus rate (140 beats/min). Moreover, there is a prolongation of QRS complex duration (> 70 milliseconds) with positive complex polarity in the inferior lead tracings and the presence of Q waves in the lead I tracing. All of these findings are consistent with a postdivisional left bundle branch block (LBBB). The concomitant second-degree AV block and bifascicular block (LBBB) is defined as trifascicular block. Paper speed = 50 mm/s; 1 cm = 1 mV.
there was positive polarity of QRS complexes in the inferior lead tracings and negative polarity of QRS complexes in the V1 precordial lead tracing. Moreover, the V1 precordial lead tracing revealed an rS-type configuration of the QRS complexes. All of these findings were consistent with a complete left bundle branch block (LBBB). The QRS complexes of the conducted beats had normal Q waves in the inferior lead tracings. The presence of Q waves in the lead I tracing may have indicated normal early depolarization of the interventricular septum because the block of conduction occurred in the distal ramifications of the left bundle branch. This type of LBBB, called postdivisional block, is typically characterized by a marked prolongation of the QRS complex duration (> 80 milliseconds). On the other hand, the absence of Q waves in the lead I tracing suggests an abnormal direction of the first vector of depolarization of the ventricular mass caused by a proximal LBBB (truncular block or divisional block). In dogs, Q waves are sometimes evident in lead I even in the presence of a truncular LBBB, and this early depolarization force derives from the right ventricular lateral wall.2

Because of the concomitant presence of LBBB and an AV conduction disturbance, a trifascicular block was suspected. Given that the ventricular conduction system is composed anatomically of a right bundle branch and a left bundle branch (which is formed by 2 fascicles, the left anterior fascicle and the left posterior fascicle), a concomitant or intermittent interruption of all 3 branches is defined as trifascicular block.

Permanent endocavitary pacemaker implantation is indicated in dogs that are clinically affected by high-degree AV block because of the high risk of developing complete AV block. In the dog of this report, a VVI (single-chamber, ventricular-sensing, ventricular-pacing [in the inhibited mode]) pacemaker was implanted by introducing the single electrostimulator catheter through the right jugular vein and fixing its end on the right ventricular apex; the pacemaker was then positioned in a subcutaneous pocket on the dog’s neck. A ventricular stimulation rate of 60 beats/min was set with a hysteresis of 40 beats/min to promote spontaneous rhythm with normal AV conduction. VVI-type pacemakers operate both stimulating and sensing only at the ventricular level and at a planned activation rate. No atrial sensing is performed with this type of pacemaker used in this manner, and ventricular depolarization occurs slowly through myocardial cells, producing wide QRS complexes.

Electrocardiography was performed after pacemaker implantation (Figure 2). In the tracings, an alternation between paced and spontaneous beats occurred. The paced beats had a low amplitude spike followed by wide QRS complexes with negative voltage in the inferior lead tracings and positive voltage in left precordial lead tracings. The spontaneous beats had a bifascicular block configuration with an LBBB morphology of the QRS complexes. In the tracings, a pause of 1,500 milliseconds was recorded after a second spontaneous beat, which induced reactivation of the pacemaker. Some nonconducted P waves were evident in all of the ECG lead tracings.

After a few days, the dog was discharged from the hospital. During a 2-year follow-up period, no other syncopal events were reported by the owners and no pacemaker abnormalities were recorded during serial ECG examinations.

![Figure 2—Twelve-lead surface ECG recording obtained from the dog with trifascicular block in Figure 1 after implantation of a VVI (single-chamber, ventricular-sensing, ventricular-pacing [in the inhibited mode]) pacemaker. Pacing stimulation occurs for the first 3 beats (notice a low-voltage pacing stimulus before the wide QRS complex). Pacemaker stimulation is interrupted by 2 sinus beats (fourth and fifth beats) with an LBBB morphology of the QRS complex. After a 1,500-millisecond pause, reactivation of the pacemaker occurs (sixth beat). Some nonconducted P waves are present during the ECG examination (white arrows). Paper speed = 50 mm/s; 1 cm = 1 mV.](image-url)
Discussion

Anatomically, the AV and intraventricular conduction systems are complex and not completely understood. The distal AV bundle emerges from the central fibrous body with the nonpenetrating and, successively, penetrating portions of the bundle of His. The His bundle continues in the branching portion of the AV bundle from which right and left bundle branches originate. The left bundle branch successively divides into a long and thin left anterior fascicle and a wider and ramified left posterior fascicle. The trifascicular theory of the specified intraventricular conduction system was first proposed for humans by Rosenbaum in 1968, and it is still accepted.1,4

A lesion affecting 1 or more of these bundles may cause an aberration in conduction of the intraventricular stimuli with potential severe consequences. Primary diseases (eg, hereditable His bundle stenosis in Pugs) or complex heart lesions (eg, myocardial infarction, myocarditis, cardiomyopathies, or cardiac malformations) can result in partial or complete interruption of the conduction through 1 or more His bundle branches.5–8 On the basis of the number of branches affected, fascicular blocks are classified as mono-, bi-, or trifascicular. Mono- and multifascicular blocks are right bundle branch block (RBBB), left anterior hemiblock (LAH), and left posterior hemiblock. Bifascicular blocks are defined as the concomitant block of the right bundle branch and the left anterior or the left posterior fascicle or as the simultaneous block of the 2 left fascicles (LBBB). Finally, trifascicular block occurs when both left and right bundle branches are interrupted because of an infranodal disease of the conduction system (true trifascicular block) or when a bifascicular block accompanies a nodal block with evidence of an AV conduction disturbance.9–11

Electrocardiography is widely used to diagnose intraventricular conduction disturbances in both humans and dogs, and specific ECG patterns have been identified for each type of mono- or bifascicular block.8,11–13 The diagnosis of trifascicular block is more complex, and sometimes an electrophysiological evaluation is required to confirm it. In general, detection of alternating blocks of all 3 branches of the AV bundle in the same ECG examination is consistent with trifascicular block. Otherwise, trifascicular block should be suspected if a bifascicular block appears in association with prolonged conduction of the stimulus distally at the level of the branching portion of the His bundle, below the compact node of the AV junction. Although some authors consider the association of a first-degree AV block with bifascicular block sufficient to diagnose a trifascicular conduction disturbance, a simple P Q interval prolongation may reflect a delay at the AV node level rather than at the 3 intraventricular fascicles. Therefore, it would be more correct to consider true trifascicular disease as that in which bifascicular conduction disturbances are associated with second-degree AV block, thereby indicating real ramified fascicle damage instead of a predivisional His bundle or nodal block.10,16–26

In both humans and dogs, the prognosis for conduction disturbances may widely vary depending on the bundles involved and the underlying mechanism inducing the block. The presence of LBBB in dogs with advanced hypokinetic cardiomyopathies is associated with increased mortality risk. Moreover, the occurrence of a Mobitz type II or advanced AV block in the presence of a bifascicular block (LBBB or RBBB with hemiblock) is associated with a high mortality rate among humans with acute myocardial infarction. The cause of sudden death in those patients is most often cardiogenic shock secondary to complete heart block; thus, permanent pacemaker implantation is the best therapeutic option for improving prognosis in cases of advanced disease of the conduction system, such as trifascicular block.10–21

References