ECG of the Month

A 2-year-old 30-kg (66-lb) sexually intact male Golden Retriever was referred to the Clinica Veterinaria Malpensa because of exercise intolerance and episodic weakness of 1 month's duration. During 1 episode of weakness, an ECG examination performed by the primary veterinarian revealed a narrow–QRS complex tachycardia with a cycle length of 200 milliseconds. The patient was treated with sotalol[a] (2 mg/kg [0.9 mg/lb], PO, q 12 h) to control episodic supraventricular tachycardia and to reduce clinical signs.

At the initial evaluation, a physical examination revealed no abnormalities. Findings of survey thoracic radiography and transthoracic echocardiography were considered normal. An initial 12-lead ECG revealed short PQ intervals, slurring of the onset of the QRS complexes (delta wave), and alteration of the QRS-ST segment morphology. These findings were consistent with ventricular pre-excitation.

The presence of an accessory pathway (AP)–mediated tachycardia was strongly suspected, and an electrophysiological assessment was performed. Sotalol administration was discontinued for 24 hours (ie, a period equivalent to 5 half-lives of the drug) before the procedure. The patient was anesthetized and placed in dorsal recumbency during the entire interventional procedure. Multiple access sites on jugular and femoral veins were used to allow introduction of endocardial catheters by use of the Seldinger technique. The electrophysiological assessment was conducted in accordance with standard techniques.[1,2] A decapolar catheter[b] was introduced at the level of the coronary sinus and positioned in contact with the epicardal surface, and a quadripolar catheter[c] was inserted near the bundle of His. An ablation catheter[d] was used to perform programmed atrial and ven-

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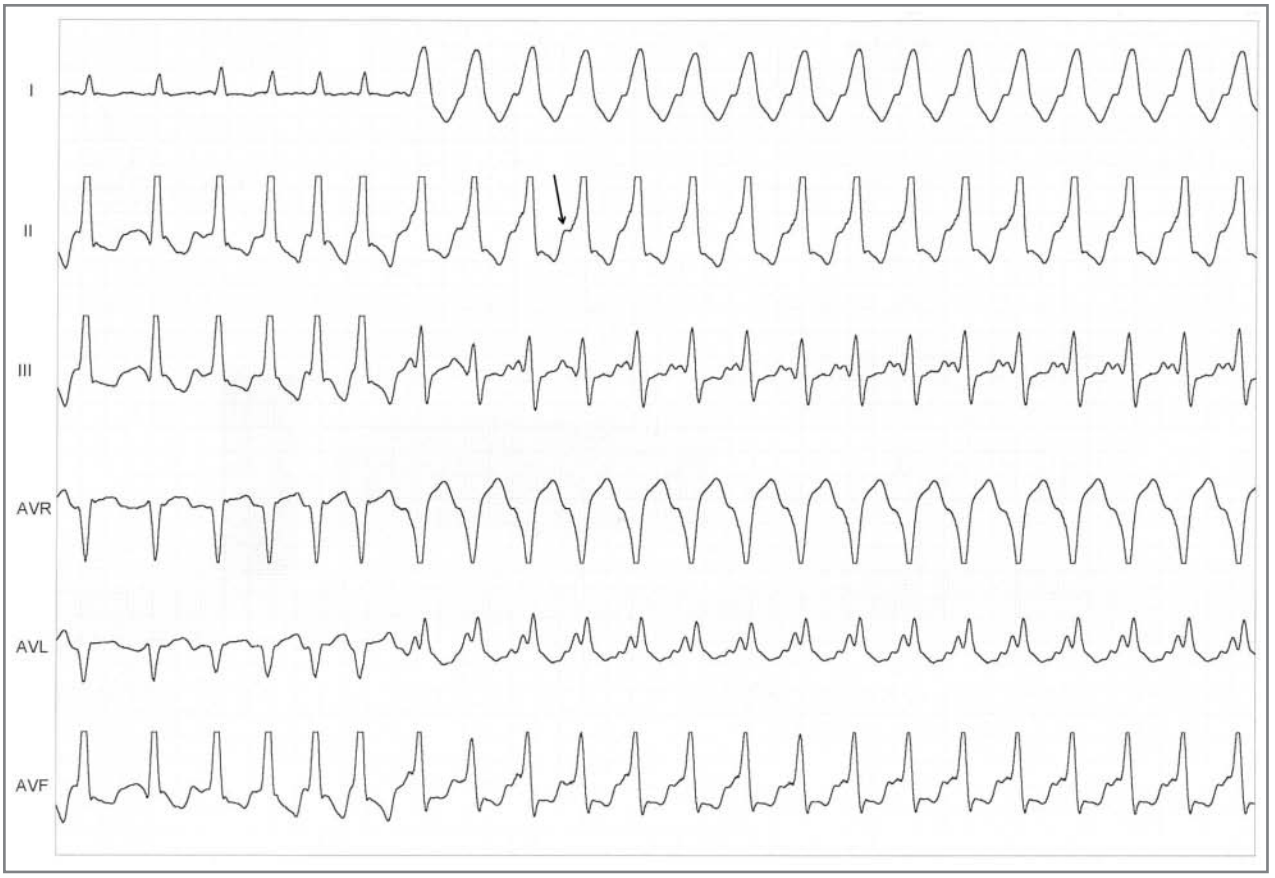

Figure 1—Surface ECG tracings (6 leads of a 12-lead ECG) obtained during electrophysiological assessment of a dog that was evaluated because of exercise intolerance and episodic weakness of 1 month's duration. On the basis of initial ECG findings, an accessory pathway (AP) was suspected. The electrophysiological examination revealed the presence of an AP that was subsequently ablated by use of a radiofrequency catheter. During the procedure and before the ablation, the dog developed a paroxysm of sustained atrial fibrillation (AF). The first 6 beats of the ECG tracings have features typical of AF with orthodromic conduction through the His-Purkinje system, including absence of the P wave, narrow QRS complex (duration, 60 milliseconds; reference range, < 70 milliseconds), and irregular R-R intervals. From the seventh beat onward, antegrade activation of the AP with signs of ventricular pre-excitation is evident. Wide QRS complexes (duration, 100 milliseconds), the presence of a delta wave preceding each R wave, and persistence of irregular R-R intervals are indicative of the occurrence of pre-excited AF. Paper speed = 50 mm/s; 1 cm = 1 mV.

tricular electrical stimulation for unipolar and bipolar endocardial mapping. All catheter tips were positioned with guidance via fluoroscopy and intracardiac ECG. In addition, a 12-lead surface ECG was displayed and analyzed on an electrophysiological recorder.[e]

Conduction and refractoriness of the atrioventricular node and AP were assessed, and the anatomic position of the anomalous bundle was localized. A single atrioventricular AP, which was characterized by bidirectional (not decremental) conduction, was located in the right atrial free wall. The AP had an antegrade effective refractory period < 120 milliseconds and an effective retrograde refractory period of 150 milliseconds. During the procedure, the dog had an episode of sustained paroxysmal atrial fibrillation (AF), which was evident via surface ECG (**Figure 1**).

ECG Interpretation

During the electrophysiological procedure, surface ECG revealed a 5-minute phase of paroxysmal AF. The first part of the 12-lead ECG tracings (first 6 beats) revealed an absence of P waves, narrow QRS complexes (duration, 60 milliseconds; reference range, < 70 milliseconds), and irregular R-R intervals (Figure 1). These findings were consistent with AF that had high ventricular response (ventricular rate, 270 beats/min) and apparently normal atrioventricular conduction. The second part of the ECG tracings (from the seventh beat onward) revealed wide QRS complexes (duration, 100 milliseconds); a low-voltage deflection preceded each QRS complex, which was consistent with a delta wave. In this portion of the tracings, the R-R intervals appeared less irregular with a ventricular rate of 270 beats/min. These latter findings were consistent with a wide–QRS complex tachycardia attributable to AF with concomitant ventricular pre-excitation (ie, pre-excited AF).[3] A ladder diagram was drawn to better elucidate the conduction along the accessory pathway during the paroxysm of pre-excited AF (**Figure 2**). External electrical cardioversion was applied to resolve the life-threatening tachyarrhythmia, delivering a single shock of 100 J. Sinus rhythm was restored, and no other episodes of AF were observed.

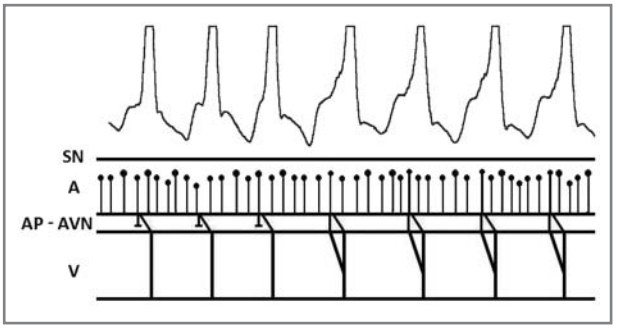

Figure 2—Ladder diagram of the lead II ECG tracing in Figure 1 from the 4th to 11th QRS complex. In the constructed diagram, vertical lines mark the atrial fibrillating waves (atrial [A] level) and QRS complexes (ventricular [V] level); connecting lines between the vertical lines indicate the antegrade conduction along the accessory pathway, the atrioventricular node, or both. Notice that the rapid and nonsynchronized atrial f waves reach both the AP and atrioventricular node (AVN), but only a few of them cross those structures to activate the ventricle. The first 3 QRS complexes are formed by the antegrade activation of the ventricle along the AVN because the AP provides an antegrade unidirectional block. The remaining QRS complexes displayed represent a fusion of antegrade conduction along the AP and the AVN. On the surface ECG of Figure 1, the pre-excitation of the ventricle along the AP can be recognized by the occurrence of the delta wave. SN = Sinus node. Paper speed = 50 mm/s; 1 cm = 1 mV.

The dog underwent radiofrequency ablation of the AP by use of a thermocouple-tipped electrode[f] with an ablation temperature of 65°C and a maximum current of 75 W for 60 seconds. At the end of the procedure, pre-excitation was no longer evident and the dog was allowed to recover from anesthesia. During the 24 hours of hospitalization after the intervention, no ECG abnormalities were detected with telemetry and the dog was subsequently discharged. After 2 years, a follow-up examination was performed; no clinical signs were evident, and reccurrence of an arrhythmia was not detected via Holter monitoring.

Discussion

Accessory atrioventricular pathways in humans and dogs are described as anomalous muscular bundles that directly connect the atrial myocardium to the ventricular myocardium, thereby bypassing the His-Purkinje system.[4–9] Accessory pathways can be classified according to their location along the atrioventricular groove (ie, left free wall, right free wall, posteroseptal, midseptal, or anteroseptal APs).[6,9–11] Accessory pathways may be present as single or multiple anomalies and may have different electrophysiological properties (ie, involving only antegrade, only retrograde [concealed], or both types of conduction). Moreover, the conduction can be decremental or nondecremental with variable durations of refractory periods.[10,12,13]

In people, the classification of APs is as follows: left free wall APs, 44% to 70%; right free wall APs, 5% to 20%; posteroseptal APs, 10% to 35%; midseptal APs, 2% to 11%; and anteroseptal APs, 4% to 10%.[9–12] In contrast to humans, the abnormal atrioventricular connection in dogs is most frequently located in the right free wall (40%) or in the posteroseptal area (27%).[13] Humans affected by an Ebstein anomaly may have multiple APs that are typically located in the right free wall or in the posterior part of the septum, locations that are more frequently described in dogs.[14,15] Most dogs that have APs are Labrador Retrievers and Boxers, suggesting a possible predisposition of these breeds to develop anomalous atrioventricular bundle connections.[2,13,16]

In humans, APs allow antegrade and retrograde conduction with an all-or-none pattern.[9,17] Antegrade conduction via the accessory connection may result in overt signs of ventricular pre-excitation in surface ECG tracings because of premature activation of the ventricles through the AP. Evidence of pre-excitation includes short PQ intervals, slurring of the onset of the QRS complex (delta wave), and alteration of the QRS-ST segment morphology.[4,6,16] The dog of this report had both antegrade and retrograde conduction, and signs of ventricular pre-excitation were detected during the electrophysiological procedure. However, because most APs in dogs involve only retrograde conduction, the absence of evidence of ventricular pre-excitation on surface ECG tracings cannot rule out the presence of a latent AP.[13]

Atrial fibrillation and orthodromic atrioventricular reciprocating tachycardia (OAVRT) are the most common arrhythmias associated with APs in humans and dogs.[2,6–8,10,13,18] In patients with reciprocating tachycardia, the refractory period of the AP during sinus rhythm ex-

ceeds the refractory period of the normal conduction system. When a premature beat occurs, it may be blocked in the antegrade direction in the AP and be propagated to the ventricle via the normal conduction system, resulting in a normal QRS complex; however, the depolarization wave then propagates through the ventricles, reaches the AP, and depolarizes the atria through retrograde conduction, allowing macro-reentry to occur. Rarely, this mechanism may operate in the reverse direction with antegrade conduction via the AP (resulting in an anomalous QRS complex) and subsequent retrograde conduction via the atrioventricular node. This latter type of tachycardia is called antidromic reciprocating tachycardia. The occurrence of antidromic reciprocating tachycardia in humans has rarely been described; its occurrence in dogs has never been previously reported, to the authors' knowledge.[6,16]

Atrial fibrillation is detected in 19% to 38% of humans treated for Wolff-Parkinson-White syndrome.[18] Moreover, AF is inducible during electrophysiological assessments, especially if the AP has an anteroseptal location. Although the pathogenesis of AF in humans and dogs with APs is not completely understood, several possible explanations have been proposed. Because the occurrence of AF correlates with the presence of OAVRT, tachycardia-induced atrial stretch and vulnerability to AF are believed to contribute to AF initiation and perpetuation. Furthermore, branching of the AP may provide the substrate for micro-reentry, which would initiate or perpetuate AF by contributing reentrant wavelets and preventing their spontaneous extinction.[13,18] In addition, in the presence of an AP, single or multiple ventricular premature beats may conduct in a retrograde manner to the affected atrium. The resulting wave front may collide with that initiated from the sinus node, resulting in nonuniform depolarization of the atria and initiating AF.[6]

In the dog of this report, AF involved orthodromic conduction initially, but subsequently changed to antidromic conduction through the atrioventricular node, thereby pre-exciting the ventricles through the AP. To the authors' knowledge, this is the first report of pre-excited AF in a dog. The fact that orthodromic conduction of AF occurs more frequently in dogs may be related to the location of the APs in that species, the presence of a concealed conduction, and the long antegrade refractory period of the AP. In the dog of this report, bidirectional conduction of the AP and a variation in the refractory period might be the basis for the pre-excitatory behavior of the AF.[13,18]

Atrial fibrillation with antegrade conduction through an AP may represent a life-threatening arrhythmia because of the high ventricular rate. During orthodromic conduction of AF, the atrioventricular node reduces the ventricular response to atrial activations because of the influence of the parasympathetic nervous system on the normal cardiac conduction apparatus. This negative chronotropic effect is absent in muscular APs, which allows most of the atrial stimuli to reach the ventricles, thereby promoting the development of fatal arrhythmias such as malignant ventricular tachycardia and ventricular fibrillation.[3,18]

In the dog of this report, ECG abnormalities were not detected in the follow-up period after the ablation of the AP. Endocardial catheter ablation of the AP was a definitively therapeutic solution for OAVRT in this dog. Possibly, resolution of the tachyarrhythmia might have prevented the occurrence of those electrophysiological abnormalities that were the basis of AF development.

a. Sotalolo, TEVA Italia S.r.l., Milan, Italy.
b. Polaris X, 7F, 2/5/2, Boston Scientific Corp, Genova, Italy.
c. Explorer 360, 5 F, 5/5/5, Boston Scientific Corp, Genova, Italy.
d. Polaris C, 4 mm, 7F, Boston Scientific Corp, Genova, Italy.
e. EMS, 16 channels, Mennen Medical Ltd, Manta, Genova, Italy.
f. Polaris C, 4 mm, 7F, Boston Scientific Corp, Genova, Italy.

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