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.

An 8-year-old spayed female Bernese Mountain Dog was evaluated because of tachycardia detected at rest. For 3 days, the owner had seen a rapid and vigorous apex beat (a "racing heartbeat") when the dog was lying down. The dog was otherwise without clinical signs. Previous medical history included lethargy and anorexia associated with a circumscribed mass in the right caudal lung lobe that was treated successfully with a lobectomy 9 months earlier. Histologic analysis of that mass revealed malignant histiocytosis; there was no evidence of metastasis at that time or in the subsequent months. No medications were being administered to the dog. Vaccination status was up to date, including vaccination against borreliosis given annually for at least the previous 3 years. Physical examination findings were unremarkable, except for a heart rate of 320 beats/min. The pulse was weak but synchronous with the heartbeat. Results of CBC and serum biochemical analysis were within reference ranges, with the exception of mildly high liver enzyme activities (aspartate aminotransferase, 82 U/L [reference range, 21 to 41 U/L]; alanine aminotransferase, 219 U/L [reference range, 19 to 136 U/L]; and alkaline phosphatase, 213 U/L [reference range, 11 to 174 U/L]). The dog was weakly seropositive against *Borrelia burgdorferi* (1:32); this was considered to have been a vaccine-related finding, but a western immunoblot test was not performed. An ECG was performed at admission (**Fig 1**) and 2 minutes later during a vagal maneuver (**Fig 2**).

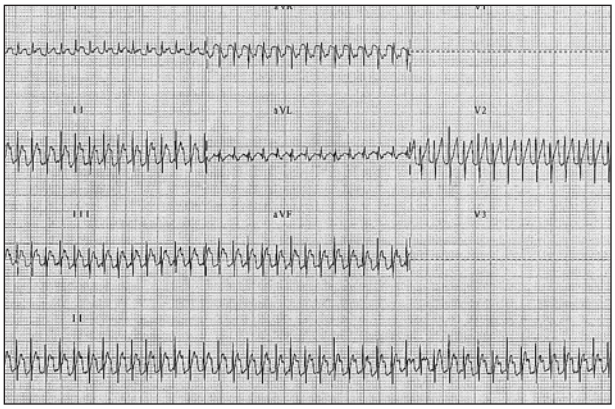

Figure 1—Seven-lead ECG tracing (6 limb leads and 1 precordial lead [V2]; lead II rhythm strip at bottom) from an 8-year-old dog with tachycardia. Paper speed = 25 mm/sec; 1 cm = 1 mV.

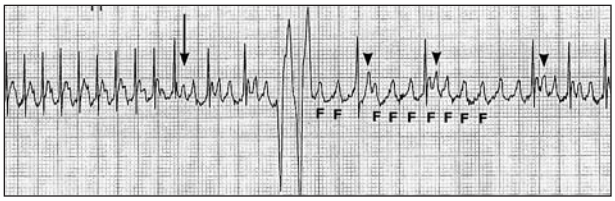

Figure 2—Lead II ECG rhythm strip from the same dog as in Fig 1. A vagal maneuver (carotid sinus massage) was initiated 7 seconds before the onset of atrioventricular (AV) block (arrow). The AV block allows the rapid, incessant atrial flutter (AFL) waves to be seen clearly (F); the AFL waves proceed through the rhythm and are not confused with the T waves (arrowheads). Paper speed = 25 mm/sec; 1 cm = 1 mV.

ECG Interpretation

The initial ECG tracing revealed narrow-complex, monomorphic tachycardia with a ventricular rate of 320 beats/min (Fig 1). In the second ECG tracing, a vagal maneuver slowed conduction through the **atrioventricular (AV)** node (Fig 2); the ventricular rate slowed to 120 beats/min, and a sawtooth baseline between the QRS complexes was observed. This sawtooth appearance was attributable to **atrial flutter (AFL)** waves that replaced the P waves entirely. The QRS complexes were irregularly irregular in their occurrence. Absence of distinct P waves, a sawtooth baseline, and an irregularly irregular ventricular rhythm are characteristics of AFL.[1,2] An additional sign that may suggest AFL is the Bix rule, which states that AFL is suspected if the T wave is spaced evenly between QRS complexes (not a feature of the ECG in this case).[1] An AFL wave can be seen interposed between some QRS complexes and T waves, but not others; the AFL waves proceed through the ECG uninterrupted and with regularity.

The ECG tracing also revealed a pair of wide, bizarre QRS complexes that occurred immediately after application of the vagal maneuver. These were **premature ventricular complexes (PVCs)**; they were unlikely to represent escape beats, as subsidiary pacemaker depolarization is inhibited when a pacemaker cell is driven by a rate exceeding its intrinsic rate. These

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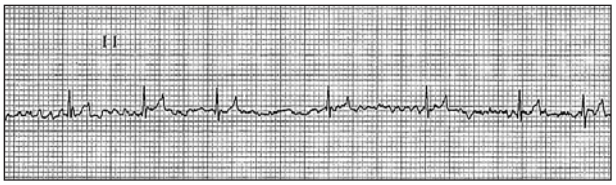

Figure 3—Lead II ECG rhythm strip from the same dog as in Fig 1 obtained 18 months after initiation of treatment. The ventricular rhythm remains irregularly irregular, flutter waves are no longer visible, and the baseline is undulating and patternless, findings that are consistent with atrial fibrillation. Paper speed = 25 mm/sec; 1 cm = 1 mV.

PVCs were considered an incidental finding, and because they did not recur, they did not influence treatment decisions. Digoxin elixir (0.0075 mg/kg [0.0034 mg/lb], PO, once, then 0.0038 mg/kg [0.0017 mg/lb], PO, q 12 h) and atenolol (0.15 mg/kg [0.068 mg/lb], PO, q 12 h) were administered. This treatment was associated with successful AV block and slowing of the ventricular rate to acceptable levels. The dog was discharged with continuation of this treatment regimen 36 hours after initial evaluation. An ECG performed after 1 week of digoxin and atenolol treatment revealed a ventricular rate of 100 to 120 beats/min, with the AFL rate unchanged at 340 beats/min.

Eighteen months after treatment was initiated, an ECG tracing (**Fig 3**) revealed coarse atrial fibrillation. The dog had remained free of clinical signs during this period. This dog was re-evaluated 2 years after the initial examination. Primary clinical signs at that time were weight loss, lethargy, and anorexia of several days' duration. Radiography revealed diffuse splenomegaly and multiple pulmonary masses > 3 cm in diameter. Cytologic evaluations of fine-needle aspirate specimens of 1 of these pulmonary masses and of the spleen were consistent with malignant histiocytosis. Because of the compromised condition and poor long-term prognosis, the dog was euthanatized at the owner's request; a necropsy was not performed.

Discussion

Tachycardia (tachyarrhythmia) can be supraventricular or ventricular in origin. Distinguishing between these 2 major classes of arrhythmia is essential because the underlying causes, treatment options, and prognoses are usually different. However, at high heart rates, the characteristic ECG features of each of these types of arrhythmia may be difficult to detect; thus, rapid tachycardia can pose a diagnostic challenge. Electrocardiographic features that help to classify a tachyarrhythmia as supraventricular or ventricular in origin include shape of the QRS complexes, association of P waves and QRS complexes, presence of fusion beats, and effect of vagal maneuvers.[1-4]

In general, most tachycardias in which the QRS complexes are narrow are supraventricular in origin, whereas most wide-complex tachycardias are ventricular in origin. Supraventricular tachycardias typically have a pattern of ventricular depolarization that produces a QRS complex of normal, sinus appearance. By contrast, the aberrant propagation of impulses through the ventricles that occurs when an impulse originates in the ventricles (eg, PVCs or ventricular tachycardia) generates a QRS complex that is usually wide and bizarre in appearance. An exception to this rule is observed occasionally during the supraventricular tachycardia that occurs simultaneously with right or left bundle branch block, severe ventricular enlargement, pre-excitation, or aberrancy; these conditions can widen the QRS complexes, producing a shape on the ECG tracing that is similar to that of PVCs.

An additional criterion to help distinguish between supraventricular and ventricular tachycardias is the location of P waves. Generally, in supraventricular tachycardias, either a P wave is present for every QRS complex or P waves are entirely absent (eg, atrial fibrillation). In contrast, with PVCs and ventricular tachycardia, P waves usually are seen but are not associated consistently with each QRS complex. Rather, they are interposed throughout the tracing, often within the wide and bizarre QRS complexes or T waves.

The presence of fusion beats also helps to distinguish supraventricular from ventricular tachycardias. These hybrid complexes are generated by the electrical collision between a normal depolarization and a PVC. The result is a QRS complex that occurs appropriately after a P wave, but is intermediate in shape; that is, the QRS complex is neither normal in appearance, nor as wide and bizarre as a PVC. Fusion beats are associated commonly with ventricular tachycardia, but not with supraventricular tachycardia.

As seen in the dog of this report, vagal maneuvers can assist in the differentiation of supraventricular tachycardia from ventricular tachycardia. In the first ECG tracing (Fig 1), 1-to-1 conduction of AFL waves to the ventricles makes it difficult to see the AFL waves; therefore, an accurate ECG diagnosis on this tracing alone is challenging. Increased vagal tone, induced by carotid sinus massage, can slow AV nodal conduction by means of hyperpolarization and decreased action potential amplitude.[1] The effect of vagal maneuvers on supraventricular tachycardia is not universal, because rapid supraventricular tachycardia may be associated with high sympathetic tone; however, the vagal maneuver was successful in this dog, slowing conduction at the AV node and revealing the AFL waves. Vagal maneuvers rarely have any effect on ventricular tachycardia.

Atrial flutter is a macro–re-entrant supraventricular tachycardia. The source of the arrhythmia is an abnormal, large (eg, pan-atrial), discrete, self-perpetuating circuit, usually within the right atrium in dogs as in humans.[5,6] The right atrium is still the most common source of AFL, even when enlargement of the left atrium predominates, as observed in dogs with mitral regurgitation.[5] Atrial flutter is an arrhythmia that is initiated and sustained by a depolarization wave crossing an area of nonuniform conduction and excitability. With AFL, there is a single, intra-atrial re-entrant circuit within the atria; anatomic barriers are thought to establish an area of slowed conduction, thereby facilitating re-entry.[7-9] Unlike atrial fibrillation, in which persistence of the arrhythmia is ascribed to scores of micro–re-entrant circuits, the single macro–re-entrant circuit of AFL is susceptible to surgical (catheter-

based) interruption and, thus, straightforward and permanent cure of the arrhythmia. In humans, catheter-based ablation cures 90 to 95% of patients with the most common forms of AFL.[6] Even with successful ablation, however, the condition recurs in 10% of patients. Factors associated with development of AFL in dogs include atrial enlargement, quinidine treatment for atrial fibrillation, and cardiac catheterization.[2] Paroxysmal AFL has been documented in patients without structural heart disease.[9-11]

Atrial flutter may be categorized as type I or type II,[6,10,12] a distinction made in human medicine, and by extrapolation in veterinary medicine, on the basis of atrial rate. In type-I AFL, the atrial rate is typically 240 to 340 beats/min; in type II, the atrial rate is typically 340 to 433 beats/min. The clinical importance of this distinction is that type-I AFL is almost always abolished by rapid (overdrive) atrial pacing whereas type-II AFL is not. Also, type-I AFL is much more amenable to current catheter ablation techniques. Type-II AFLs mimic atrial fibrillation on the surface ECG and are poorly characterized at this time.[6,12]

Type-I AFLs have been further categorized and described in detail on the basis of the orientation of the AFL waves in the different ECG leads; the positive flutter waves in leads II, III, and aVF in the ECG tracings shown here indicate that this is reverse typical (also called atypical, uncommon, clockwise, or rare) AFL.[12,13]

Electrocardiograms that have the characteristics of AFL may in fact represent distinct, though similar-looking, supraventricular arrhythmias. In humans with AFL that have been evaluated with electrophysiologic studies (intracardiac ECG), AFL proper, other macro–re-entrant atrial rhythms, and focal atrial tachycardias generate identical surface ECGs. Electrophysiologic testing is important to establish a precise diagnosis if the goal is to permanently eliminate the AFL by use of invasive techniques. In veterinary practice, however, making this distinction is much less important because the goal is to prevent an excessively rapid ventricular rate in response to repetitive supraventricular depolarizations. Therefore, noninvasive, palliative treatment involves medications (eg, digoxin, β-blockers, calcium-channel blockers) that decrease or prevent a rapid ventricular rate, regardless of whether the rhythm is truly AFL or some other supraventricular tachycardia.

In the dog of this report, the diagnosis of type-I reverse typical AFL on the surface ECG (Fig 1 and 2) was not corroborated by intracardiac ECG studies, because electrophysiologic evaluation was declined by the owner. Confirmation that the rhythm was re-entrant by use of cardioversion would have provided the opportunity for either overdrive pacing or ablation and cure, but this was not performed.

At the time of evaluation, this dog did not have overt clinical signs associated with tachycardia. An echocardiogram revealed no evidence of structural heart disease, and thoracic radiographic findings were within normal limits for a dog with a history of thoracotomy. Because antiarrhythmic medications are associated with adverse effects and have contraindications, the potential risks and benefits of such treatment were evaluated prior to initiating treatment in a dog that was free of clinical signs.

Whether to treat the arrhythmia remains a challenging question in many patients.[3] It has been proposed that an arrhythmia should be treated if it compromises hemodynamic status, is associated with clinical signs (eg, syncope), contributes to increased risk for sudden death, or causes myocardial damage.[1,14] Specifically, rapid ventricular rates result in decreased cardiac output, decreased coronary perfusion, and low systemic blood pressure.[1] In addition, tachycardia-mediated cardiomyopathy may develop, the result of myocardial exhaustion caused by an incessantly rapid heart rate.[15,16] For example, a ventricular rate of ≥ 250 beats/min maintained for 3 to 4 weeks will reliably cause ventricular enlargement and eventually myocardial failure in dogs.[1] Sensations of tachycardia that commonly lead human patients to seek emergency medical attention (eg, dizziness or chest palpitations) may not cause overt clinical signs in dogs; thus, a logical argument can be made for treating arrhythmias in dogs without clinical signs if an equivalent arrhythmia in a human patient is invariably associated with symptoms.

Because of the dog's high ventricular rate and weak pulse, treatment was initiated. The catheter-based ablation procedures were recommended but were declined by the owner. If conversion or cure is not possible, slowing AV nodal conduction to obtain a ventricular rate < 130 beats/min[17] without signs of bradycardia (ie, excessive decrease in ventricular rate) is attempted.

The relationship of the dog's previous medical problem (malignant histiocytosis) to this arrhythmia was not determined. No arrhythmia had been evident at the time of lung lobectomy 9 months earlier, but extension of the neoplastic process to disrupt the atrial myocardium may have developed.

Although AFL and atrial fibrillation are distinct, there appears to be a relationship between the 2 conditions that is incompletely understood. In humans, AFL is often an unstable rhythm that can spontaneously revert to a normal sinus rhythm or degenerate into atrial fibrillation.[10,18] In dogs, rapid AFL may progress to atrial fibrillation, as in the dog of this report; if the rate is slow, AFL may convert to a sinus rhythm.[1]

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