ECG of the Month

A 13-year-old 20-kg (44-lb) castrated male English Springer Spaniel was evaluated at the University of Florida Veterinary Medical Center for transvenous pacemaker implantation because of third-degree atrioventricular (AV) block. The AV block was initially diagnosed several months prior to this evaluation, and no treatment had been initiated. One month prior to evaluation, the dog was treated for congestive heart failure, which prompted referral for further treatment and pacemaker implantation. At the time of the evaluation, the dog was receiving furosemide (2 mg/kg [0.91 mg/lb], PO, q 12 h) and enalapril (0.5 mg/kg [0.23 mg/lb], PO, q 12 h). On initial physical examination, heart rate was 50 to 60 beats/min and irregular. Electrocardiography was performed (**Figure 1**). Thoracic radiography revealed left-sided cardiomegaly with evidence of pulmonary venous congestion and mild pulmonary edema. Echocardiographic evaluation revealed bilaterally large atria, volume overload of the left ventricle, and normal systolic function.

ECG Interpretation

Evaluation of the initial ECG recordings revealed a sawtooth baseline appearance with an atrial rate of approximately 600 depolarizations/min (Figure 1). The ventricular response rate was approximately 60 beats/min and was somewhat irregular. An ECG diagnosis of atrial flutter with complete or third-degree AV block was made.

Typically, medical treatment of atrial flutter is undertaken initially with the aim of either controlling the ventricular response rate or converting the atrial flutter back to normal sinus rhythm. However, because of the dog's concurrent complete heart block and congestive heart failure, anesthesia and pacemaker implantation along with attempted electrical cardioversion of the atrial flutter were planned. A temporary pacing lead was placed percutaneously with fluoroscopic guidance through the left jugular vein and into the right ventricular apex prior to induction of anesthesia. The dog's heart was paced at 90 beats/min. Following routine induction of anesthesia, the dog was placed in dorsal recumbency and direct-current–synchronized cardioversion was performed by use of a biphasic defibrillator.[a] Initial energy delivery was 10 J, which did not result in conversion. Following the second delivered shock of 20 J, the ECG recordings revealed conversion of the atrial flutter to normal sinus rhythm (**Figure 2**). Additionally, each P wave was now conducted in a 1:1 pattern (ie, a QRS complex was present for every P wave), with a prolonged PR interval of 0.17 seconds (first-degree AV block).

Although the dog no longer had third-degree AV block, concern for recurrence of atrial flutter and heart block resulted in the decision to implant a single-chamber, ventricular-

Contributed by Maggie C. Machen, BS; Amara H. Estrada, DVM, DACVIM; and Robert Prošek, DVM, DACVIM; from the Department of Small Animal Clinical Sciences, College of Veterinary Medicine, University of Florida, Gainesville, FL 32610-0126.
Address correspondence to Dr. Estrada.

Figure 1—Electrocardiographic traces (12 leads) obtained from a dog that was evaluated because of third-degree AV block and planned pacemaker implantation. A sawtooth baseline (heart rate of approx 600 beats/min) characteristic of atrial flutter is visible most prominently in V2 through V6. Paper speed = 50 mm/s; 5 mm = 1 mV.

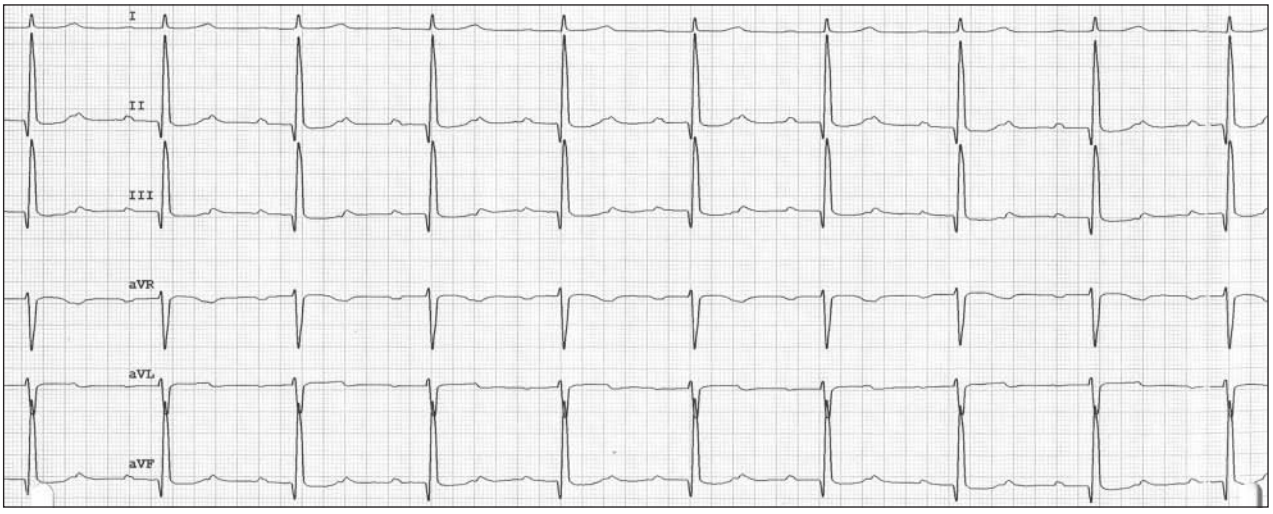

Figure 2—Electrocardiographic traces (6 leads) obtained from the dog in Figure 1 following electrical cardioversion of atrial flutter to normal sinus rhythm. Following restoration of sinus rhythm, each P wave is conducted through the AV node to cause a ventricular depolarization. Heart rate is approximately 100 beats/min. The PR interval is prolonged at 0.17 seconds (reference range, 0.06 to 0.13 seconds). There is also a left ventricular enlargement pattern indicated by increased R-wave amplitude of 3.4 mV (reference range, < 2.5 mV). Additionally, P waves are notched indicative of a large left atrium. Paper speed = 25 mm/s; 1 cm = 1 mV.

based permanent pacemaker as a backup safety mechanism. Pacemaker implantation was performed routinely, as was described previously in the veterinary medical literature,[1] and the dog recovered in an intensive care unit. The following day, ECG evaluation revealed continued sinus rhythm; each P wave was conducted in a 1:1 pattern with a first-degree AV block.

Discussion

Atrial flutter is a relatively uncommon cause of supraventricular tachycardia in dogs.[2] The rhythm is a result of a macro-reentrant loop (a single large circuit) that repetitively depolarizes the atria, traveling around either anatomic or functional boundaries.[2,3] Reentrant loops are promoted by dispersion of refractoriness between myocytes, a condition that can occur with stretch, fibrosis, scarring from previous surgeries, or other anatomic or functional barriers to conduction.[4] Characteristically, atrial rates in flutter are in the range of 230 to 450 beats/min, and the ventricular rate and ratio vary in accordance with the refractory properties of the AV conducting system.[5–7] On a surface ECG recording, P waves that originate from the sinus node are replaced with regular sawtooth waves called flutter (F) waves, and a clear delineation of the baseline is not possible.[2]

The ventricular rhythm and rate in atrial flutter will depend on both the atrial rate and state of AV nodal conduction. Because of the properties of the AV node, AV block almost always exists in some form during atrial flutter.[8] The ventricular response to the high atrial rate is determined by 3 factors: the refractory period of the AV node, the level of autonomic tone, and the degree of so-called concealed conduction within the node.[9] The effective refractory period of the AV node limits the maximal rate at which depolarizations can occur because the AV node is known to have a longer effective refractory period than atrial or ventricular tissue.[10] The refractory period of the AV node is influenced heavily by many factors including drugs, circulating electrolyte concentrations, and autonomic tone. Sympathetic activity increases the conduction velocity of the AV node and shortens the refractory period, whereas parasympathetic activation has the opposite effect.[11]

The ventricular response in atrial flutter may be regular or irregular depending on the ratio of AV conduction. For example, with 1:1 AV nodal conduction, every P wave results in a ventricular response leading to a regular fast rhythm. Similarly, if AV nodal conduction is always in a 2:1 pattern, the rhythm will be slower but regular. If AV nodal conduction varies so that P waves are conducted sometimes in a 2:1 and sometimes in a 3:1 ratio, then the rhythm will be irregular. One-to-one conduction during atrial flutter produces perhaps one of the most serious arrhythmias and may occur if AV refractoriness is short, as is the case with some humans who have either enhanced or anomalous AV conduction.[5] Treatment with antiarrhythmic agents can also provoke 1:1 conduction by decreasing the atrial rate while conduction through the AV node remains increased.[5] Numerically, even-numbered conduction ratios (ie, 2:1 and 4:1) are more common in humans than odd-numbered conduction ratios (ie, 3:1 and 5:1).[12] The most common AV conduction ratio in humans with untreated atrial flutter is 2:1, with an atrial rate of 300 beats/min and a ventricular rate of 150 beats/min.[13] If AV nodal conduction remains constant, the ventricular rhythm will be regular; however, if the ratio of conducted beats varies, the ventricular rhythm will be irregular. This irregular ventricular response is frequently attributable to Wenckebach periodicity. Alternation between 2:1 and 4:1 AV nodal conduction often occurs and can be the result of 2 levels of block—2:1 at a location high in the AV node and 3:2 at a location lower in the node.[13]

Recurrent alternation of short and long ventricular intervals can also be a result of concealed conduction. Flutter impulses can penetrate into the AV node to varying degrees, which can also influence conduction. Atrial impulses that are blocked in the AV node and not conducted to the ventricle may slow conduction of subsequent atrial impulses through the AV node, a

phenomenon called concealed conduction. Repetitive concealed conduction is the mechanism responsible for a slow ventricular rate with variable degrees of penetration of the AV node during atrial flutter.[14] In the presence of AV conduction abnormalities or antiarrhythmic drugs, a 4:1 conduction ratio or higher can develop and cause a slow ventricular response rate, as was likely present in the dog of this report.

Typically, treatment of atrial flutter involves either control of the ventricular response rate by slowing conduction through the AV node or conversion to sinus rhythm. When rapid control of tachycardia is required, calcium channel blockers, β-adrenoceptor blockers, or both are effective in slowing AV nodal conduction.[2] However, because of the presence of concurrent suspected high-grade AV nodal block in the dog of this report, slowing the AV conduction rate was thought to be contraindicated. Conversion to a normal sinus rhythm can be attempted by use of medical treatments or electrical methods. Pharmacologic interventions that are reported to convert atrial flutter to sinus rhythm include class III antiarrhythmics (eg, amiodarone, sotalol, and ibutilide), class Ia or Ic antiarrhythmics (eg, propafenone and flecainide), and calcium channel blockers.[2,3] Because many of these drugs would also likely slow AV nodal conduction in the dog of this report, these options were also thought to be contraindicated. Direct current cardioversion is another commonly used treatment for atrial flutter, in which electricity is used to depolarize all vulnerable cardiac myocytes simultaneously and disrupt the reentrant loop.[3] A 95% success rate for conversion is reported.[7] Animals that require this treatment must be completely anesthetized and intubated prior to receiving a transthoracic electrical shock.[3] The electrical energy setting used for treatment of atrial flutter is much lower (typically 50 J) than settings required for treatments of other arrhythmias.[b] Potential complications are generally dose dependent, and more complications develop with repeated or high-dose electrical shock.[3]

In the dog of this report, the initial cause of the atrial flutter remained undetermined. Following electrical cardioversion and conversion to normal sinus rhythm, the dog was able to conduct all sinus node depolarizations (P waves) through the AV node with a first-degree AV block. The dog initially had presumed complete heart block, but the bradycardia was likely a result of structural AV nodal disease compounded to a large extent by the concealed conduction and high-grade AV nodal block produced by the atrial rhythm. One-to-one conduction of sinus node depolarizations through the AV node was possible following conversion to normal sinus rhythm. Because of the suspected AV nodal disease and concurrent congestive heart failure, a pacemaker was implanted as a precaution and initially was not activated. At a recheck examination 3 months later, the dog's activity and attitude had greatly improved but despite continued administration of furosemide and enalapril, cardiomegaly had not resolved. Pacemaker interrogation revealed that 37% of the dog's heartbeats were paced and the pacemaker was sensing and pacing normally.

a. Medtronik Lifepak 12 biphasic defibrillator, Medtronic Emergency Response Systems Inc, Redmond, Wash.

b. Resnekov L. *Synchronized capacitor discharge in the management of cardiac arrhythmias with particular reference to the hemodynamic significance of atrial systole*. MD thesis, Department of Medicine, University of Cape Town, Cape Town, South Africa, 1965.

References

1. Kobayashi M, Hoshi K, Hirao H, et al. Implantation of permanent transvenous endocardial pacemaker in a dog with atrioventricular block. *J Vet Med Sci* 2003;65:1131–1134.
2. Moïse NS. Diagnosis and management of canine arrhythmias. In: Fox PR, Sisson D, Moïse NS, eds. *Textbook of canine and feline cardiology*. 2nd ed. Philadelphia: WB Saunders Co, 1999;331–385.
3. Olgin JE, Zipes DP. Specific arrhythmias: diagnosis and treatment. In: Zipes DP, Libby P, Bonow RO, et al, eds. *Braunwald's heart disease: a textbook of cardiovascular medicine*. 7th ed. Philadelphia: Saunders, 2005;803–863.
4. Leier CV, Meacham JA, Schaal SF. Prolonged atrial conduction; a major predisposing factor for the development of atrial flutter. *Circulation* 1978;57:213–216.
5. Kennelly BM, Lane GK. Electrophysiological studies in four patients with 1:1 atrioventricular conduction. *Am Heart J* 1978;96:723–730.
6. Cosio FG. Atrial flutter update. *Card Electrophysiol Rev* 2002;6:356–364.
7. Bromberg BI, Schuessler RB, Gandhi SK, et al. A canine model of atrial flutter following the intra-atrial lateral tunnel Fontan operation. *J Electrocardiol* 1998;(suppl 30):85–93.
8. Braunwald E. Mechanisms and diagnosis of arrhythmias. In: *Heart disease: a textbook of cardiovascular medicine*. 3rd ed. Philadelphia: Saunders, 1988;655.
9. Kawabata M, Hirao K, Higuchi K. et al. Clinical and electrophysiological characteristics of patients having atrial flutter with 1:1 atrioventricular conduction. *Europace* 2008;10:282–286.
10. Linhart JW, Braunwald E, Ross J Jr. Determinants of the duration of the refractory period of the atrioventricular nodal system in man. *J Clin Invest* 1965;44:883–890.
11. Cunningham J, Klein B. Cardiovascular physiology. In: *Textbook of veterinary physiology.* 4th ed. St Louis: Saunders Co, 2007;203.
12. Kay GN, Plimb VJ. Atrial fibrillation, atrial flutter, and atrial tachycardia. In: Fuster V, Alexander RW, O'Rourke RA, eds. *Hurst's the heart*. 11th ed. New York: McGraw-Hill Book Co, 2004;825–853.
13. Castellanos A, Moleiro F, Pastor JA, et al. Reverse alternating Wenckebach periods and other modes of regression of ≥ 8:1 to 2:1 atrioventricular block. *Am J Cardiol* 1998;82:528–531.
14. Kilborn MJ, McGuire MA. Electrocardiographic manifestations of supernormal conduction, concealed conduction, and exit block. In: Zipes DP, Jalife J, eds. *Cardiac electrophysiology from cell to bedside*. 4th ed. Philadelphia: Saunders, 2004;733–738.