

# ECG of the Month

An 8-year-old castrated male German Shorthaired Pointer (weight, 33 kg [72.6 lb]) was evaluated at the Foster Hospital for Small Animals, Cummings School of Veterinary Medicine, Tufts University, because of a persistent tachyarrhythmia that was refractory to antiarrhythmic treatment. Five days prior to referral, the dog was examined by the referring veterinarian because of exercise intolerance of 1 week's duration. A tachyarrhythmia was auscultated on examination, and an ECG recording was interpreted as ventricular tachycardia. The arrhythmia was unchanged following a constant rate infusion of lidocaine (75  $\mu\text{g}/\text{kg}/\text{min}$  [34.1  $\mu\text{g}/\text{lb}/\text{min}$ ]); administration of procainamide IM (15 mg/kg [6.8 mg/lb], q 6 h) was also ineffective.

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At the time of referral, the dog was ambulatory but weak. The heart rate was 280 beats/min, and the rhythm was markedly irregular. A grade 1/6 systolic murmur was auscultated at the region over the mitral valve. The jugular vein was distended with an estimated central venous pressure of 8 to 10 cm of water. The quality of the femoral arterial pulses was variable, and pulse deficits were present. Thoracic radiography revealed mild cardiomegaly and a fine interstitial pulmonary pattern consistent with mild pulmonary edema. Echocardiography revealed moderate left atrial and ventricular dilatation with decreased fractional shortening (23%; reference range, 28% to 40%).

A presumptive diagnosis of ventricular tachycardia was made on the basis of the initial ECG tracing (Figure 1). The arrhythmia was not controlled following IV administration of several boluses of lidocaine (2 mg/kg [0.9 mg/lb]), and sotalol was administered PO once (2.4 mg/kg [1.1 mg/lb]). The dog was monitored by use of a continuous ECG, and 90 minutes after administration of sotalol, the ventricular rate

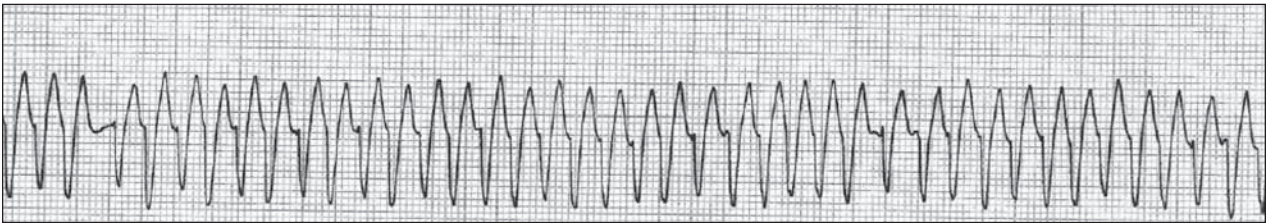


Figure 1—Initial lead II ECG obtained from a dog with tachycardia that did not respond to treatment with lidocaine and procainamide. Paper speed = 25 mm/s; 1 cm = 1 mV.

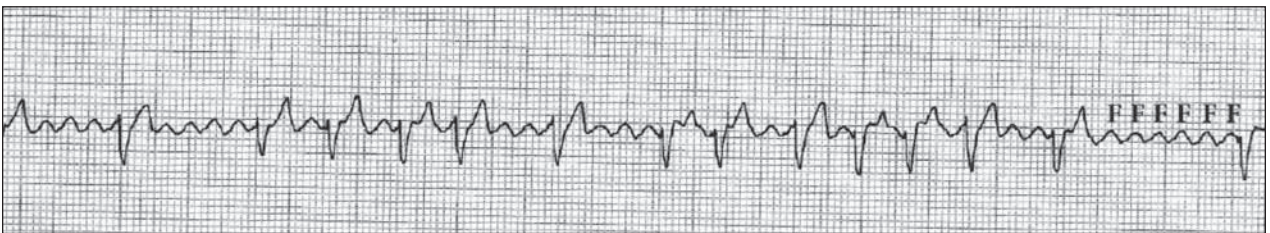


Figure 2—Lead II ECG recorded from the dog in Figure 1 after administration of sotalol PO and diltiazem IV. Notice the sawtooth baseline with flutter waves (F waves) resulting from atrial flutter. Paper speed = 25 mm/s; 1 cm = 1 mV.

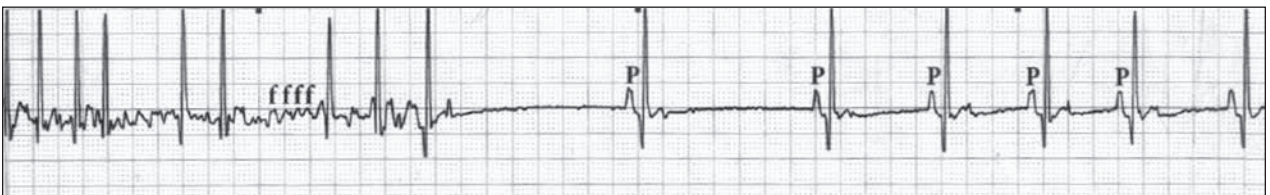


Figure 3—An ECG tracing obtained from the dog in Figure 1 by use of a transthoracic lead system illustrating the conversion from atrial fibrillation to normal sinus rhythm. The fine, unorganized undulations in the baseline (fibrillation or f waves) result from atrial fibrillation. The f waves disappear and the rhythm becomes more regular as sinus rhythm is restored. During sinus rhythm in the latter portion of the tracing, P waves are noted in front of the QRS complexes. The QRS complexes appear relatively normal as a result of the use of transthoracic leads. Paper speed = 25 mm/s; 1 cm = 1 mV.

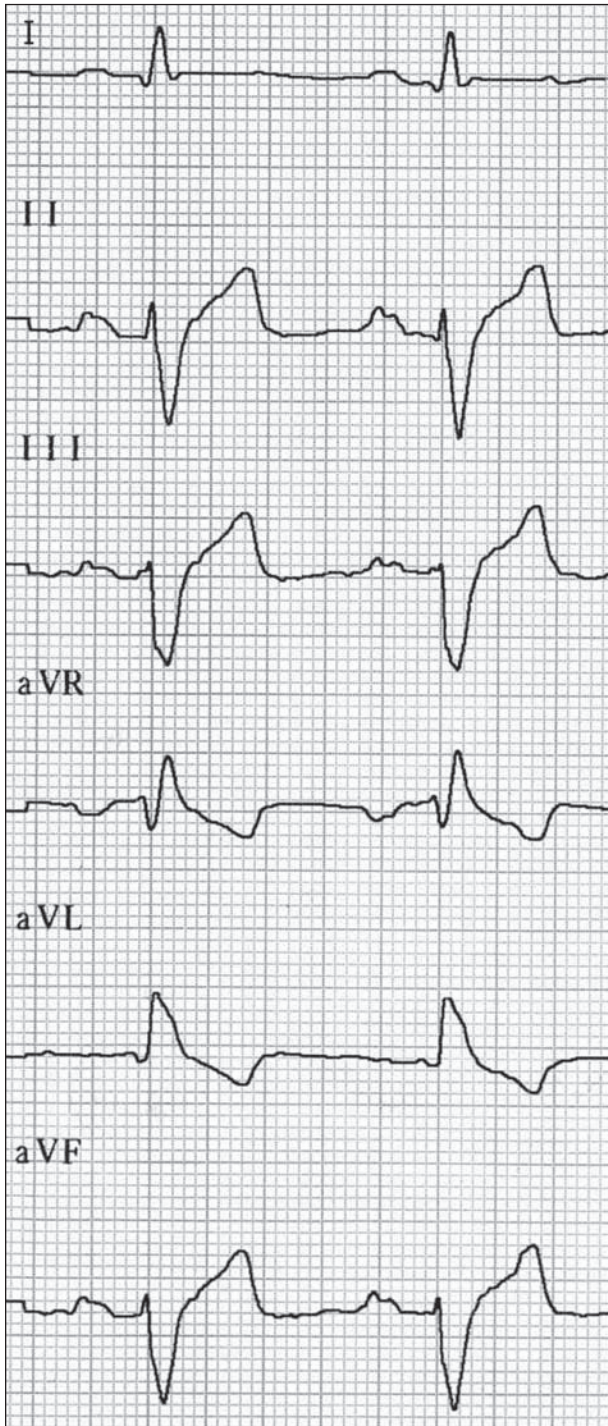


Figure 4—Portions of a multilead ECG recording obtained from the dog in Figure 1 (shortly after the tracing in Figure 3 was recorded) illustrating sinus rhythm, an axis shift, and prolonged QRS duration resulting from an intraventricular conduction disturbance. Paper speed = 50 mm/s; 1 cm = 1 mV.

slowed to approximately 200 beats/min. At this rate, baseline undulations that reflected organized atrial activity became apparent in the ECG tracing. This led to a diagnosis of supraventricular tachycardia with aberrant ventricular conduction. Diltiazem (0.3 mg/kg [0.14 mg/lb]) was administered IV during a period of 10 minutes to further decrease the ventricular response

rate. After diltiazem administration, the ventricular rate slowed and sawtooth baseline undulations were apparent, consistent with a diagnosis of atrial flutter. Treatment of the dog with a sustained-release formulation of diltiazem<sup>a</sup> (2.7 mg/kg [1.23 mg/lb], PO, q 12 h), sotalolol (1.8 mg/kg [0.82 mg/lb], PO, q 12 h), furosemide (1.5 mg/kg [0.68 mg/lb], PO, q 12 h as required), and digoxin (0.004 mg/kg [0.0018 mg/lb], PO, q 12 h) was started. The day after admission to the hospital, the dog developed atrial fibrillation. The following day, atrial fibrillation converted spontaneously to normal sinus rhythm.

### ECG Interpretation

Evaluation of the initial ECG revealed a rapid, irregular rhythm with a ventricular rate of 290 to 320 beats/min (Figure 1). The QRS complexes had a wide and bizarre appearance (QRS duration, 0.09 seconds), and P waves were not evident. An initial diagnosis of ventricular tachycardia was made on the basis of this initial ECG tracing. In retrospect, the initial rhythm was likely supraventricular tachycardia with aberrant intraventricular conduction.

In the ECG tracing recorded after administration of sotalolol PO and diltiazem IV, regular sawtooth undulations in the baseline (F waves) were evident, which were consistent with a diagnosis of atrial flutter (Figure 2). In this tracing, the atrial rate was 420 beats/min and the ventricular response rate had slowed to 120 to 140 beats/min. The conduction ratio of F waves to ventricular beats was variable, ranging from 2:1 to 8:1. The QRS complexes appeared abnormal with deep and wide S waves; the complexes were markedly prolonged (duration, 0.09 seconds), indicative of an intraventricular conduction disturbance. The QRS duration and appearance were similar to those features of the initial ECG, which supported a diagnosis of supraventricular tachycardia with aberrant conduction, rather than ventricular tachycardia.

On day 2 of hospitalization, the dog's cardiac rhythm deteriorated from atrial flutter to atrial fibrillation; the atrial fibrillation persisted for 18 hours until the third day of hospitalization, at which time an ECG tracing revealed that the dog had converted from atrial fibrillation to normal sinus rhythm (Figure 3). The appearance of the QRS complexes in this tracing was different from their appearance in the earlier ECG tracings because the strip was recorded by use of a transthoracic lead system. In a multilead ECG tracing recorded shortly after conversion to sinus rhythm, persistence of the nonspecific intraventricular conduction disturbance with prolonged QRS complex (0.09 seconds) was confirmed (Figure 4).

### Discussion

This report illustrates the difficulty in differentiating between ventricular tachycardia and wide complex tachycardias of supraventricular origin in dogs. Ventricular arrhythmias are characterized in ECG tracings by wide, bizarre QRS complexes with no associated P waves. Conversely, in ECG tracings, supraventricular tachycardias typically have narrow QRS complexes that appear similar to normally conducted sinus

beats and are frequently associated with a preceding P wave. The ability to discriminate between ventricular and supraventricular arrhythmias is more challenging in animals with supraventricular tachycardia and abnormally wide QRS complexes. In the dog of this report, the presence of wide QRS complexes resulted in an incorrect initial diagnosis of ventricular tachycardia. Wide supraventricular complexes may occur secondary to severe left or right ventricular enlargement, preexistent left or right bundle branch block, **atrioventricular (AV)** conduction over an accessory pathway, or aberrant ventricular conduction.<sup>1</sup>

The initial treatment and long-term management of ventricular and supraventricular arrhythmias are very different, and every effort should be made to diagnose the arrhythmia accurately prior to instituting treatments. If the distinction cannot be made via ECG, application of a vagal maneuver (ie, carotid sinus massage) during the ECG recording may further aid in diagnosis. Interventions that increase vagal tone to the heart may profoundly affect supraventricular, but not ventricular, arrhythmias. Increased parasympathetic tone decreases the rate of firing of the sinoatrial node and increases conduction time and refractoriness in the AV node. However, a vagal maneuver is often unsuccessful as a means of terminating supraventricular tachyarrhythmias.

Atrial flutter is an uncommon arrhythmia in dogs and cats. The rhythm is characterized by the replacement of discrete P waves with undulating flutter waves (F waves). The F waves represent sequential depolarization and repolarization of the atria. They are regular and rapid with a constant appearance and a rate that usually ranges from 300 to 500 beats/min.<sup>2</sup> This extremely rapid atrial rate exceeds the conduction capacities of the AV node, and impulses that reach the conducting tissue during refractory periods are blocked. Atrioventricular conduction of the F waves to the ventricles may occur at a fixed or variable rate. The dog of this report had an AV conductance ratio that ranged from 2:1 to 8:1. The ventricular rate and rhythm are dependent on the atrial rate and the state of conductance of the AV node.<sup>2</sup>

The F waves that appear to be completely blocked may actually penetrate to different levels of the AV node. This phenomenon is known as concealed conduction.<sup>3,4</sup> It is proposed that the mammalian AV node is composed of 2 or more functional tiers with complex and variable refractory periods.<sup>3,5</sup> During atrial flutter, conduction of atrial impulses occurs at the proximal level of the node at a conductance ratio of 2:1, whereas 3:2 Wenckebach periodicity is evident at the distal level of the node.<sup>5</sup> For an impulse to reach the ventri-

cles, it must reach the AV node when all levels are non-refractory. This occurs at highly variable intervals and may explain irregular ventricular response rates, such as that described in the dog of this report. Proposed mechanisms of atrial flutter include circus movement of impulses traveling in a ring of tissue spanning the right atrium, multiple reentry loops, unifocal ectopic atrial impulse formation, and multi-focal ectopic impulse formation.<sup>2,6</sup>

Any condition that results in atrial enlargement can predispose a patient to the development of atrial flutter or fibrillation. Typically, the QRS configuration in animals with atrial flutter is normal in appearance, unless a ventricular conduction abnormality is also present. Conversion of atrial flutter may be accomplished via antiarrhythmic drug treatment, direct current cardioversion, rapid atrial pacing (entrainment), or catheter ablation. Prior to low-voltage cardioversion, calcium channel blockers or  $\beta$ -adrenoreceptor blockers (administered alone or in combination with digoxin) may be used to slow the ventricular response rate. Medical cardioversion by use of digoxin and quinidine has been proposed to be an effective treatment in dogs,<sup>7</sup> and d-sotalol can terminate experimentally induced atrial flutter in isolated, perfused dog hearts.<sup>8</sup> In the dog of this report, conversion occurred following administration of a combination of sotalol, digoxin, and diltiazem.

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a. Diltiazem hydrochloride extended release, Mylan Pharmaceuticals Inc, Morgantown, WV.

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## References

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