A 13-year-old 6.0-kg (13.2-lb) spayed female Shetland Sheepdog was referred to the University of Minnesota Veterinary Medical Center for evaluation of congestive heart failure. Four months earlier, a diagnosis of cardiac disease had been made following the auscultation of a heart murmur by the referring veterinarian. Thoracic radiography reportedly revealed cardiomegaly. There was no evidence of pulmonary edema, and the dog had no clinical signs of heart disease. During an examination performed by the referring veterinarian 2 months later, abdominal effusion was detected. At that time, thoracic radiography revealed evidence of mild pulmonary edema. An abdominocentesis was performed, and the dog was treated with furosemide (4 mg/kg [1.8 mg/lb], PO, q 12 h), benazepril (0.5 mg/kg [0.23 mg/lb], PO, q 24 h), and pimobendan (0.2 mg/kg [0.09 mg/lb], PO, q 12 h). Despite these interventions, the dog developed progressive abdominal distention over the following few weeks. Abdominocentesis was repeated, and the dog was referred to the Veterinary Medical Center for further evaluation.

On physical examination, the dog was bright, alert, and responsive. The heart rate was 180 beats/min, but the rhythm was regular. Thoracic auscultation revealed a grade 4/6 left-sided basilar continuous heart murmur. Abdominal distention was evident, and a fluid wave was palpable during abdominal ballottement. The initial diagnostic evaluation included a CBC, serum biochemical analyses, urinalysis, and thoracic radiography. Mild nonregenerative anemia (Hct, 35.8%; reference range, 37.5% to 60.3%) was detected. Serum biochemical abnormalities included moderate azotemia (BUN concentration, 98 mg/dL [reference range, 9 to 31 mg/dL] and creatinine concentration, 1.9 mg/dL [reference range, 0.6 to 1.6 mg/dL]) and mild hypoalbuminemia (2.4 mg/dL; reference range, 2.7 to 3.7 mg/dL).

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ECG Interpretation

The initial ECG tracing revealed a regular heart rhythm with a ventricular rate of 186 beats/min (Figure 1). The QRS complexes appeared normal in morphology and duration; these features were considered consistent with ventricular depolarization occurring via the His-Purkinje conduction pathway. The P waves were prolonged in duration (0.11 seconds; reference range, < 0.04 seconds), suggestive of a large left atrium. The R wave height was within reference limits (2.1 mV; reference range, < 2.5 mV) despite echocardiographic evidence of a large left ventricle. Each QRS complex was preceded by a P wave, and each P wave appeared to be succeeded by a QRS complex. An initial assessment of sinus tachycardia was made.

A vagal maneuver (ie, carotid sinus massage) was performed to confirm the presence of sinus tachycardia (Figure 2). The vagal maneuver caused an immediate reduction in the ventricular rate and revealed regular baseline undulations (F waves). The undulations had uniform morphology, polarity, and cycle length; these features were consistent with a diagnosis of atrial flutter. The atrial rate was 372 beats/min; this rate was exactly twice the initial ventricular rate, indicating 2:1 atrioventricular (AV) conduction prior to vagal stimulation. During the carotid sinus massage, AV conduction was variable, with the ratio of P waves to QRS complexes ranging from 3:1 to 6:1.

Figure 1—Lead II ECG tracing obtained from a dog with a patent ductus arteriosus and congestive heart failure. The heart rate is 186 beats/min; heart rhythm is regular. The QRS complexes appear normal in morphology and duration. Each QRS complex is preceded by a P wave, and each P wave appears to be succeeded by a QRS complex. There is prolongation of the P waves (duration, 0.11 seconds; reference range, < 0.4 seconds), which is suggestive of a large left atrium. An initial assessment of sinus tachycardia was made. Paper speed = 50 mm/s; 1 cm = 1 mV.
Following the recognition of atrial flutter, the initial ECG tracing was reexamined (Figure 1). It was determined that every second P wave was likely fused with the T wave of the preceding ventricular complex, creating the erroneous appearance of sinus tachycardia.

**Discussion**

Atrial flutter is a rare arrhythmia in dogs, and it remains poorly characterized in the veterinary medical literature. Historically, the mechanism underlying atrial flutter is controversial; conflicting reports suggested that either a rapidly firing atrial focus or a form of reentry is responsible. More recently, studies involving dogs with experimentally induced atrial flutter have confirmed that the arrhythmia is caused by macro-reentrant circuitry in the right atrium. Anatomic and functional conduction blocks that are critical to the development of this reentrant circuit have been identified, including the tricuspid valve annulus, caval vena cava, crista terminalis, and cavotricuspid isthmus. The well-defined anatomic localization has facilitated the development of curative catheter ablation techniques in human cardiovascular medicine.

In humans, atrial flutter is primarily paroxysmal and the duration is usually seconds to hours. In dogs, the rarity of this arrhythmia makes definitive clinical characterization difficult, although it appears to be an unstable rhythm that often converts to sinus rhythm or atrial fibrillation. Atrial flutter and atrial fibrillation are closely related, and many now consider that these arrhythmias are extremes of a flutter-fibrillation spectrum. This important clinical interrelationship is supported by the recent recognition that atrial flutter likely requires atrial fibrillation for its initiation and maintenance. It is proposed that atrial fibrillation creates the functional components necessary to complete the atrial flutter macro-reentrant circuit, which is principally a line of block between the two venae cavae.

In dogs, a diagnosis of atrial flutter can usually be made on the basis of surface ECG data. The continuing reentry of the electrical impulse in the atrial myocardium creates regular undulations that have a constant morphology and polarity. These undulations are termed flutter waves (F waves), and in sequence they create the characteristic saw-toothed appearance indicative of atrial flutter. The F waves typically occur at a rate of 300 to 600 beats/min. This rapid rate of atrial depolarization usually exceeds the conduction capabilities of the AV node, resulting in a functional AV block. Atio-ventricular conduction in atrial flutter is usually 2:1 or 4:1 (Figure 1) and results in a regular ventricular rhythm; however, conduction may be variable (Figure 2) and results in an irregular ventricular rhythm.

The most commonly reported ECG abnormality in dogs with a left-to-right-sided shunting patent ductus arteriosus is tall R waves (ie, >2.5 mV in lead II). This abnormality is reportedly present in approximately 50% of dogs with a large left ventricle. Despite radiographic and echocardiographic evidence of a large left ventricle in the dog of this report, the R wave height was considered normal, illustrating the insensitivity of this finding. However, this case was complicated by the dog’s pleural effusion, which can reduce the amplitude of R waves recorded at the body surface.

As evident in the dog of this report, vagal maneuvers can assist in the differentiation of supraventricular tachyarrhythmias. Vagal maneuvers stimulate the vagus nerve, thereby increasing parasympathetic tone to the heart. As a result, the rate of sinus nodal discharge is slowed, AV conduction is slowed, and the AV refractory period is prolonged. The impact of this intervention on a supraventricular tachyarrhythmia depends on the tachyarrhythmia’s underlying mechanism. In dogs with sinus tachycardia, vagal stimulation causes a gradual slowing of the heart rate. In contrast, arrhythmias that require the AV node for perpetuation may abruptly terminate and are classified as AV node dependent; such arrhythmias include AV node reentrant tachycardia, automatic junctional tachycardia, and bypass tract tachycardia. Vagal stimulation may also affect AV node-independent arrhythmias (eg, atrial ectopic tachycardia, atrial flutter, and atrial fibrillation) by decreasing the ventricular response rate to atrial depolarizations. In the dog of this report, the reduction in the ventricular response rate following vagal stimulation revealed flutter waves that were previously concealed by the QRS-T complexes, which enabled a diagnosis of atrial flutter to be made. Despite their clinical usefulness, vagal maneuvers may be unsuccessful, especially in the presence of high sympathetic tone. Therefore, the absence of a response to a vagal maneuver cannot be meaningfully interpreted, and pharmacologic interventions may be necessary (eg, IV administration of calcium-channel blockers or β-adrenoceptor blockers).

To the authors’ knowledge, inadequate information is available to formulate evidence-based treatment recommendations for atrial flutter in dogs. In humans, conversion to sinus rhythm by use of catheter ablation has emerged as the treatment of choice. In dogs, conversion via administration of antiarrhythmic drugs, including procainamide,
digoxin, quinidine, and sotalol, has been reported.\textsuperscript{2,3,11} Alternatively, when tachycardia is present, medications to slow the ventricular response rate may be administered.\textsuperscript{2,3} Digoxin, \(\beta\)-adrenoceptor blockers, and calcium-channel blockers are used most commonly for this purpose.\textsuperscript{2,3}

The dog of this report was treated with diltiazem (0.8 mg/kg [0.36 mg/lb], PO, q 8 h) in an attempt to decrease the ventricular response rate. At an examination 1 week later, the dog had an irregular tachyarrhythmia, and assessment of an ECG trace confirmed atrial fibrillation. The patent ductus arteriosus was occluded by use of a nitinol-based canine duct occluder\textsuperscript{a} under fluoroscopic guidance, and evaluation of a postprocedure color flow Doppler echocardiogram confirmed successful occlusion. At 3 months following the initial evaluation at the Veterinary Medical Center, the dog continued to receive medical treatment for heart failure with persistent atrial fibrillation.

\textsuperscript{a} Amplatz canine duct occluder, AGA Medical Corp, Plymouth, Minn.

**References**


