A 2-year-old 41.0-kg (90.2-lb) neutered male Bernese Mountain Dog (dog 1) was evaluated at the Veterinary Medical Center, University of Florida because of 2 episodes of syncope within a 2-day period. The dog had no previous history of weakness or lethargy, but was inactive because it had undergone a tibial plateau leveling osteotomy 10 days earlier. It also had a history of hypothyroidism, recurrent pyoderma, and chronic weight loss. The dog was receiving treatment with tramadol (1.2 mg/kg [0.55 mg/lb], PO, q 12 h) and thyroxine (0.007 mg/kg [0.003 mg/lb], PO, q 12 h).

At the initial evaluation, the dog was bright, alert, responsive, and ambulatory. Mucous membranes were pale, and the capillary refill time was 3 seconds. The dog was panting, and pulse quality was poor and variable. Cardiac auscultation revealed tachycardia (250 to 300 beats/min) with a predominantly regular rhythm and no heart murmur. The incision site on the hind limb was clean, dry, and closed. The remainder of the physical examination findings were unremarkable. A CBC, thoracic radiography, and echocardiography revealed no abnormalities. Serum biochemical abnormalities included mildly high aspartate aminotransferase activity (75 U/L; reference range, 9 to 42 U/L), moderately high alkaline phosphatase activity (611 U/L; reference range, 16 to 111 U/L), and severely high alanine aminotransferase activity (1,525 U/L; reference range, 17 to 86 U/L); all other variables were within reference limits. Electrocardiography was performed (Figure 1).

ECG Interpretation

The ECG recording revealed a regular supraventricular tachycardia with a rate of 250 beats/min (Figure 1). The height and duration of the QRS complexes appeared normal (< 2.5 mV) with a predominantly regular rhythm and no heart murmur. The incision site on the hind limb was clean, dry, and closed. The remainder of the physical examination findings were unremarkable. A CBC, thoracic radiography, and echocardiography revealed no abnormalities. Serum biochemical abnormalities included mildly high aspartate aminotransferase activity (75 U/L; reference range, 9 to 42 U/L), moderately high alkaline phosphatase activity (611 U/L; reference range, 16 to 111 U/L), and severely high alanine aminotransferase activity (1,525 U/L; reference range, 17 to 86 U/L); all other variables were within reference limits. Electrocardiography was performed (Figure 1).

A vagal maneuver consisting of bilateral application of manual ocular pressure and carotid massage was performed to decrease the ventricular response rate and reveal the underlying arrhythmia. This maneuver transiently decreased the heart rate (mean ventricular rate, 120 beats/min) and revealed a rhythm of atrial flutter, with prominent sawtooth waveforms of consistent morphology and length (Figure 2). At this time, F waves occurred at a rate of 500 beats/min. The atrioventricular conduction ratio was 4:1 to 2:1. The QRS complexes are normal in appearance. The ECG assessment was atrial flutter (atrial rate, 500 beats/min). Paper speed = 50 mm/s; 1 cm = 1 mV.
ular (AV) conduction ratio of F waves to ventricular beats varied from 2:1 to 4:1, which was reduced from the initial ECG tracing that indicated consistent 2:1 conduction. Two doses of diltiazem (0.24 mg/kg [0.11 mg/lb], IV) were administered slowly at an interval of 5 minutes. The heart rate decreased to 80 to 115 beats/min with persistent atrial flutter. For continued treatment, the dog received diltiazem (1.09 mg/kg [0.5 mg/lb], PO, q 8 h) and was monitored via ECG telemetry. The dosage of diltiazem was increased within the first 12 hours of hospitalization (1.46 mg/kg [0.66 mg/lb], PO, q 8 h) to better control the ventricular response rate; however, the heart rate remained inadequately controlled.

Because of the unsuccessful control of the dog's heart rate, sotalol treatment (1.95 mg/kg [0.89 mg/lb], PO, q 12 h) was initiated the following morning. An ECG recording obtained 2 hours after initiation of sotalol administration treatment revealed a sinus rhythm and an atrial couplet (mean heart rate, 100 beats/min; Figure 3). All other ECG measurements were within reference limits.

During the subsequent 24 hours, the dog was monitored via ECG telemetry in the intensive care unit. The recording revealed a predominant sinus rhythm with sporadic paroxysms of nonsustained atrial flutter. The following day, the dosage of sotalol was increased (2.44 mg/kg [1.11 mg/lb], PO, q 12 h), and the dog was fitted with a 24-hour Holter monitor and discharged from the hospital. Holter monitor evaluation revealed a normal sinus rhythm with frequent atrial premature contractions, nonsustained occurrences of atrial tachycardia, and a 4-hour period of continuous atrial flutter. The dosage of sotalol was increased (2.93 mg/kg [1.33 mg/lb], PO, q 12 h), and a follow-up 24-hour Holter monitor evaluation performed 1 month later revealed a normal sinus rhythm with infrequent single atrial premature contractions. Additional follow-up consisted of a recheck examination and 24-hour Holter monitor evaluation 3 months later; at that time, findings were similar. Administration of sotalol was continued at the same dosage, and the owner reported at the 3-month recheck that the dog was doing well at home with no additional syncopal episodes.

Another dog (a 3-year-old 72-kg [158.4-lb] spayed female Newfoundland [dog 2]) was examined because of lethargy of 1 month's duration and an acute episode of tachypnea and mild epistaxis. The diagnosis of atrial flutter with no underlying structural cardiac disease was also made for this dog. Results of a CBC, serum biochemical analyses (including assessment of circulating thyroid hormone concentrations), thoracic radiography, and echocardiography were all unremarkable. The dog was medically managed for atrial flutter with sotalol (1.11 mg/kg [0.50 mg/lb], PO, q 12 h) and was successfully converted to sinus rhythm within 12 hours. A 24-hour Holter monitor recording obtained approximately 1 month after discharge from the hospital revealed maintenance of sinus rhythm with infrequent atrial premature contractions. At the 1-month recheck, the owner of this dog reported that the dog continued to receive sotalol at the initial dosage and was doing well.

Discussion

Atrial flutter is defined as a rapid and regular form of atrial tachycardia, which is suspected to involve a conduction block that results in a macroreentrant circuit that typically travels around the caudal vena cava, crista terminalis, and cranial vena cava. Electrocardiographically, sawtooth-shaped, rapid undulations (F waves) replace normal distinct P waves. The flutter waves typically have constant morphology, polarity, and rates of 300 to 500 beats/min. On ECG tracings, the ventricular rate is commonly high and the number of impulses that reach the ventricle is dependent on the refractory period of the AV node, its conduction characteristics, and generalized autonomic tone. The depolarization rate of atrial flutter typically exceeds the refractory period of the AV node, which results in functional second-degree AV block.

It can be difficult to diagnose rapid atrial flutter with 2:1 functional second-degree AV block because the F waves are superimposed within the T waves and within the QRS complexes. A pathological supraventricular tachycardia should be suspected when a very fast heart rate is combined with upright and narrow QRS complexes. A functional 2:1 AV block should be suspected with additional P waves hidden within the QRS complexes. These principles were evident in the initial ECG tracing obtained from dog 1; as the conduction pattern was altered by increasing parasympathetic tone via a vagal maneuver, the F waves were exposed and the true atrial rate became apparent. Decreased cardiac output and hypoperfusion can develop secondary to very rapid ventricular conduction of supraventricular
arrhythmias. In addition to allowing diagnosis of the arrhythmia, a vagal maneuver or IV administration of medications to decrease AV nodal conduction leads to improvement in cardiac output and may prevent life-threatening consequences.

Although the initial goal of treatment for atrial flutter is to maximize cardiac output by controlling the rate of conduction through the AV node, another therapeutic option is to restore a normal sinus rhythm. If the ventricular rate is adequate for sufficient cardiac output, it is controversial whether conversion is necessary or helpful. Conversion of atrial flutter is possible with either direct current cardioversion, radiofrequency catheter ablation, or treatment with antiarrhythmic agents.1,3 Numerous medications have been used in attempts to convert atrial flutter in dogs and humans. To the authors’ knowledge, there are no specifically recommended antiarrhythmic drugs for the treatment of atrial flutter in dogs, but several antiarrhythmic drug classes (particularly Ia, Ic, and III) have been used with unknown or variable efficacies.1,6 Sotalol is a class III antiarrhythmic drug that also has nonselective β-adrenergic receptor–blocking effects.7 It is an approved medication for the maintenance of sinus rhythm in humans with symptomatic atrial fibrillation or atrial flutter who are currently in a sinus rhythm.8 It is effective in the treatment of atrial flutter because of its β-adrenergic receptor–blocking action, which decreases the ventricular response rate, and its potassium channel–blocking action, which prolongs repolarization in myocardial tissue for potential conversion to a sinus rhythm.1,9 Prolongation of the atrial and ventricular refractory periods inhibits conduction along any bypass tract in both directions.8,10 The prolongation of the atrial refractory period may extend the wavelength (the product of the propagation velocity and the action potential duration) longer than the path of the atrial flutter reentrant circuit, thereby causing conversion to a normal sinus rhythm.1,9,11 Although a rare occurrence, any antiarrhythmic drug that causes prolongation of the QT interval has the potential to induce torsades de pointes—a rapid polymorphic ventricular tachycardia that can degenerate to fatal ventricular fibrillation. In both of the dogs of this report, sotalol decreased the heart rate by causing conversion to and maintenance of a sinus rhythm.

In humans, medical management for atrial flutter is often unsuccessful and direct current cardioversion or radiofrequency catheter ablation is necessary.12 The dogs of this report may have converted more readily with medical treatment because of their lack of underlying cardiac disease. In 1 study,11 14 of 15 dogs with no underlying cardiac disease and experimentally induced atrial flutter underwent successful conversion to a normal sinus rhythm following IV administration of d-sotalol. Another possible reason for conversion in the 2 dogs may be related to the duration of atrial flutter. Although this was unknown in each case, both dogs had a short duration of clinical signs. It is known that the duration of atrial fibrillation is inversely related to the duration of sinus rhythm maintenance after cardioversion in dogs.13 It is possible that the same clinical correlation can be made for dogs with atrial flutter and that the dogs of this report had a short duration of atrial flutter prior to intervention.

The underlying cause of atrial flutter was unknown in the 2 dogs of this report. Atrial fibrillation and atrial flutter are often caused by primary cardiac disease.3 Large-breed dogs can develop atrial fibrillation simply because of their large atrial mass.1,13 It may be hypothesized that atrial flutter in each of the 2 large-breed dogs of this report could have developed because the atrial mass was sufficiently large to allow development of a macroreentrant circuit. In addition, dogs with structurally normal hearts can develop atrial fibrillation as a result of other causes such as anesthesia, gastrointestinal tract disease, and hypothyroidism.3 Other noncardiac diseases, triggers, and modulating factors can predispose or cause atrial flutter in dogs. The dogs of this report may have also had underlying systemic disease that was not definitively diagnosed at the time of evaluation.

Oral treatment with sotalol caused conversion of atrial flutter to a sinus rhythm and the conversion was maintained over several months in both dogs. Further investigation of the use of sotalol administration to treat atrial flutter in dogs with no cardiac disease may be warranted.

References