ECG of the Month

A 14-year-old 38.9-kg (85.6-lb) castrated male Golden Retriever was evaluated because of tachypnea of 2 to 3 weeks' duration and detection of a globoid cardiac silhouette on thoracic radiographs obtained by the dog's regular veterinarian. Echocardiography revealed pericardial effusion with cardiac tamponade and a mixed echogenic mass (1.64 × 1.71 cm) originating from the right auricular appendage, which was assessed as most consistent with cardiac hemangiosarcoma. Initial ECG identified electrical alternans and low-voltage QRS complexes (data not shown). Pericardiocentesis was performed, during which 300 mL of hemorrhagic fluid was evacuated. During pericardiocentesis, uniform ventricular premature contractions of left bundle branch morphology were identified; a short paroxysm of ventricular tachycardia was evident, which was treated with a dose of lidocaine (80 mg, IV). Following pericardiocentesis, ECG evaluation was repeated (**Figure 1**).

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The authors declare no conflicts of interest.

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ECG Interpretation

In the ECG recording obtained following pericardiocentesis, the underlying rhythm was irregularly irregular. Saw-tooth waves or F waves had replaced P waves with variable degrees of atrioventricular (AV) conduction evident. Some of the F waves were buried in the T wave of the preceding QRS complex, as evidenced by the variable T-wave morphology. The atrial rate was approximately 500 beats/min, and the ventricular rate was approximately 160 to 170 beats/min. Findings were consistent with a diagnosis of atrial flutter with 2:1 to 4:1 AV block. Administration of a lidocaine bolus (80 mg, IV) was repeated and resulted in conversion

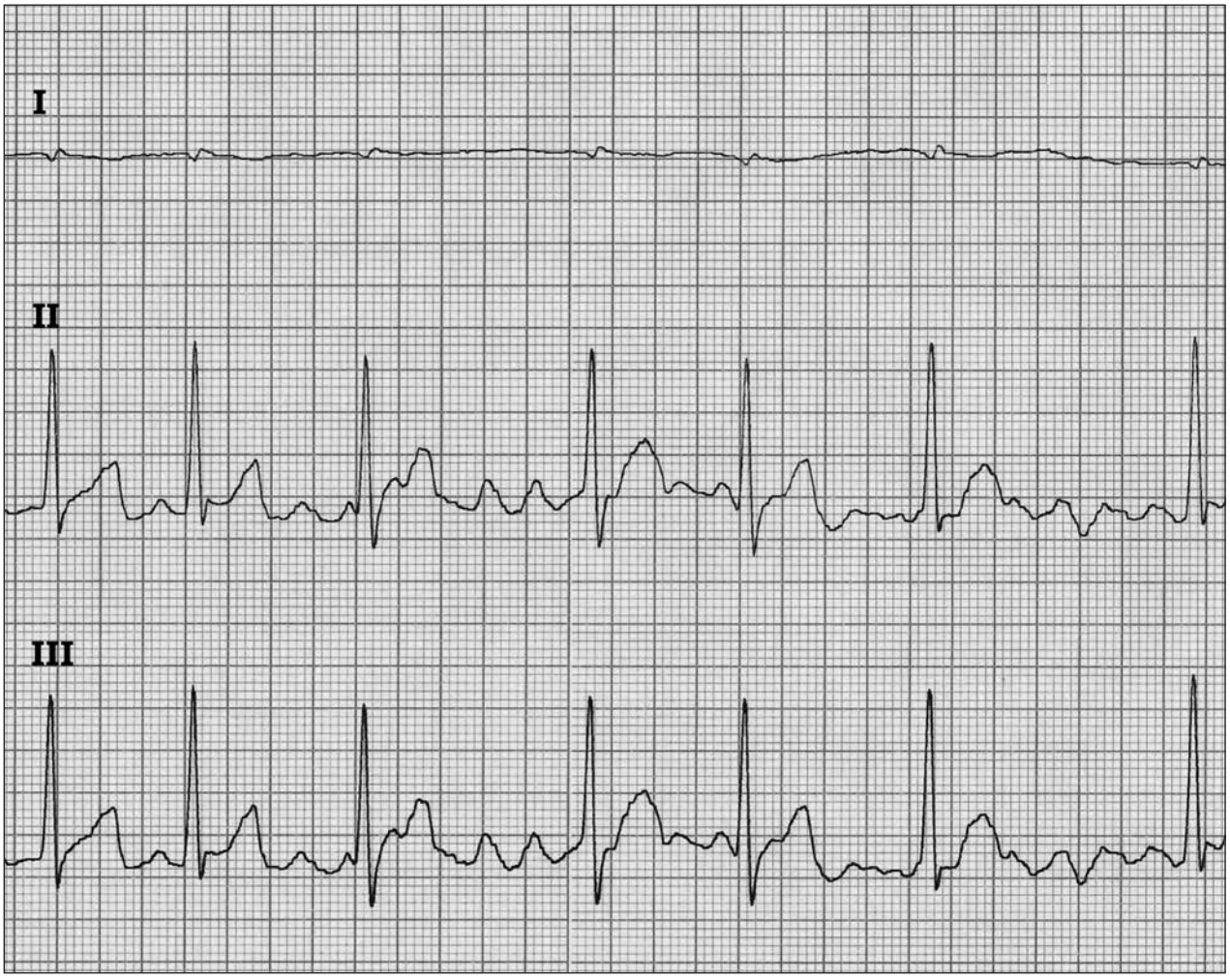

Figure 1—Postpericardiocentesis lead I, II, and III ECG tracings obtained from a dog with a mass originating from the right auricular appendage (most likely cardiac hemangiosarcoma), pericardial effusion, and cardiac tamponade. Paper speed = 50 mm/s; 1 cm = 1 mV.

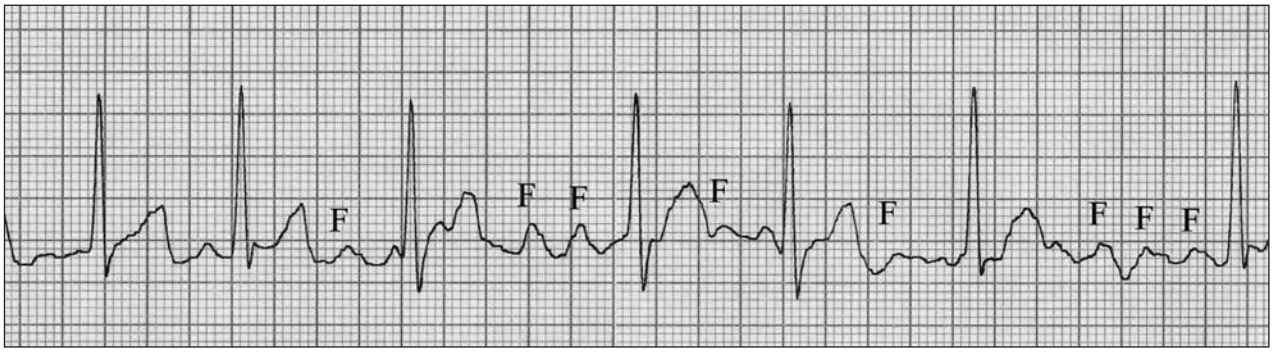

Figure 2—Selected portion of the postpericardiocentesis lead II ECG tracing obtained from the dog in Figure 1. The underlying rhythm is irregularly irregular. Sawtooth waves or F waves have replaced P waves with variable degrees of atrioventricular (AV) conduction present. The variable T-wave morphology indicates that some of the F waves are buried in the T wave of the preceding QRS complexes. The atrial rate is approximately 500 beats/min, and the ventricular rate is approximately 160 to 170 beats/min. The ECG findings are consistent with a diagnosis of atrial flutter with varying degrees of 2:1 to 4:1 AV block. Paper speed = 50 mm/s; 1 cm = 1 mV.

of the atrial flutter to normal sinus rhythm. At recheck examinations performed 1 week and 1 month later, no recurrence of atrial flutter was detected.

Discussion

Atrial flutter is a rapid and regular form of atrial tachycardia with an atrial rate that typically varies from 300 to 500 beats/min in dogs, cats, and humans. In ECG recordings obtained from companion animals and people during periods of atrial flutter, sawtooth-shaped rapid undulations called F waves replace the normally distinct P waves. These flutter waves typically have a constant morphology and polarity.[1–3] The ventricular rate typically exceeds the reference limit and is dependent on the refractory period of the AV node as well as the conduction characteristics and autonomic tone of the heart.[1,2] The depolarization rate of atrial flutter typically exceeds the refractory period of the AV node, resulting in a functional second-degree AV block.[1–3] The primary mechanism of atrial flutter is with a macroreentry circuit that develops in the right or left atrium.[2,3] In humans, there are 2 types of atrial flutter: type I and type II.[2,3] Type I atrial flutter is also called typical atrial flutter; it is associated with an atrial rate of 240 to 350 beats/min, and the reentrant loop encircles the tricuspid valve apparatus. Type I atrial flutter can circle in either a counterclockwise or clockwise direction. If it circles in a counterclockwise direction, which is the more common form in people, the flutter waves are inverted in ECG recordings from leads II, III, and aVF. When the atrial flutter circles in a clockwise direction, the flutter waves are upright in ECG recordings from leads II, III, and aVF.[2,3] Type II atrial flutter in people has not been studied as extensively, to our knowledge, and is associated with incisional scars in the right atrium or originates from the left atrium, pulmonary veins, or mitral valve annulus. Type II atrial flutter is typically characterized by atrial rates that are faster than those associated with type I atrial flutter.[2,3] This classification scheme has not been adopted in veterinary medicine; however, experimentally induced atrial flutter in dogs has been shown to circle in either the clockwise or counterclockwise direction.[4]

The development of postpericardiocentesis atrial flutter in a dog has not been reported previously, to our knowledge.[5,6] In the dog of this report, the presence of a right atrial mass may have contributed to the development of a reentry circuit in the diseased myocardium and precipitated the atrial flutter. However, if this was the case, the dog should have had atrial flutter at the time of the initial referral evaluation rather than developing atrial flutter only after pericardiocentesis was performed. Furthermore, because the mass was not removed, atrial flutter would have been expected to recur and been identified during subsequent recheck ECG evaluations.

For the dog of this report, the authors hypothesize that the atrial flutter may have been precipitated by the relief of the pericardial tamponade. Following pericardiocentesis, the previously collapsed right atrium could have stretched as preload to the heart was improved. Results of an experimental study[7] in dogs indicated that the effective refractory period of the thin portion of the atrium was substantially increased following stretching by infusion of high volumes of saline (0.9% NaCl) solution. In that study,[7] prolongation of the effective refractory period in the atrium was sufficient to precipitate atrial fibrillation. The development of postpericardiocentesis atrial fibrillation in dogs has been rarely reported in the veterinary medical literature.[8,9] Thus, the authors postulate that a differential prolongation of effective refractory period in the right atrium would create different refractory periods among the atrial cells, thereby enabling the development of the reentry circuit necessary for atrial flutter formation.

A second possibility for the development of the atrial fibrillation in the dog of this report is that as a result of the relief of cardiac tamponade, the preload to the heart was increased and subsequently cardiac output was increased. An increase in cardiac output would be sensed by the baroreceptors in the aortic arch and carotid sinus through the vagus and glossopharyngeal nerves, respectively, and the nucleus tractus solitarii of the brainstem would be innervated, which would increase vagal input and decrease sympathetic stimulation to the heart. In experiments in dogs, increases in vagal tone to atrial cells result in differential effects on the effective refractory period, thereby favoring a macroreentry phenomenon,[10] which is a necessary condition for the development of atrial flutter.

For the dog of this report, the exact role of these pathophysiologic processes in the development of the atrial flutter is unclear because after pericardiocentesis, all dogs undergo an increase in cardiac output as well as increases in atrial stretch and vagal tone to the heart. Therefore, it seems likely that the atrial flutter developed secondary to a combination of these processes with some possible influence of the right atrial mass as well as treatment with the lidocaine.

Treatment of atrial flutter in humans typically involves catheter ablation of the reentrant circuit because medical management of atrial flutter is often unsuccessful.[2,3,11] A study[12] in dogs with experimentally induced atrial flutter revealed that administration of D-sotalol had the highest conversion rate of atrial flutter to normal sinus rhythm (14/15 dogs), followed by administration of quinidine (9/15) and lidocaine (2/10). Treatment with procainamide, digoxin, quinidine, and sotalol have been reported to successfully convert naturally occurring atrial flutter in dogs.[13–15] To our knowledge, this is the first report of administration of lidocaine to convert atrial flutter to normal sinus rhythm in a dog. The optimal antiarrhythmic strategy for dogs with atrial flutter requires further investigation.

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Correction: ECG of the Month

In the ECG of the Month article published in the May 1, 2013, issue (*J Am Vet Med Assoc* 2013;242:1222–1224), the authors' names were listed incorrectly. The authors' names should have appeared as follows:

This report was submitted by Ilaria Spalla, DVM; Paola Giuseppina Brambilla, DVM, PhD; Paola Scarpa, DVM, PhD; Stefano Romussi, DVM, PhD; Giulia Riscazzi, DVM; and Chiara Locatelli, DVM, PhD; from the Department of Veterinary Science and Public Health (DIVET), University of Milan, 20133 Milan, Italy.