ECG of the Month

The Academy of Veterinary Cardiology sponsors this feature. Readers of the *JAVMA* are invited to submit contributions. Contributions should include a brief description of the case (150 words); good quality contrast glossy photographs (5 X 7 in) of tracings, with the components of a QRS complex labeled; figure legends with information on ECG lead, paper speed, and voltage calibration; an ECG interpretation; and a discussion of the abnormality. Two hard copies of the manuscript and each figure must be submitted, along with an electronic copy on a 3.5-in PC-formatted disk. Submissions that are complete will be sent to the feature coordinator, Dr. Robert Hamlin, at The Ohio State University for review.

A 6-year-old castrated male Greyhound was evaluated during a routine annual examination at the Veterinary Medical Teaching Hospital at the University of Florida. The dog was a retired racing dog and was being used as a blood donor. Abnormal findings detected on physical examination included an irregular cardiac rhythm and a right-sided systolic murmur (grade 3/6); mean heart rate was 100 beats/min. Results of physical examination of the jugular vein and palpation of the abdomen were normal. Arterial pulse quality was variable. Assessment of an ECG tracing indicated atrial fibrillation (**Fig 1**). Two-dimensional echocardiography revealed moderate enlargement of the right atrium and right ventricle; in addition, there was dysplasia of the tricuspid valve with direct papillary muscle insertion and leaflet tip curling. Color Doppler echocardiography revealed moderate tricuspid regurgitation. The velocity of the tricuspid regurgitation recorded by continuous wave Doppler imaging was 2.5 m/s, which corresponded to a Doppler-derived pressure gradient of 25 mm Hg. Although right atrial pressure was not directly measured, this pressure gradient was suggestive of normal right ventricular and pulmonary artery pressures. Thoracic radiographic findings included moderate right-sided cardiomegaly and mild enlargement of the caudal vena cava. Radiographically, there was no evidence of congestive heart failure. A Holter monitor was attached to the dog to evaluate its heart rate during a 24-hour period.

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ECG Interpretation

The initial ECG tracing revealed an irregular, supraventricular rhythm with a mean heart rate of 120 beats/min (Fig 1). Evaluation of the tracing indicated an absence of P waves and presence of baseline oscillations (f waves) that were consistent with atrial fibrillation. The QRS complexes were splintered and of low voltage (0.7 mV), with a normal mean electrical axis of +50°. Furthermore, the QRS complexes were prolonged at 0.07 seconds, which reflected ventricular enlargement or an intraventricular conduction defect. The T waves were positive, each with an amplitude of 1 mV (143% of the R wave amplitude). The ST segment was normal, and the QT interval was within normal limits. The electrocardiographic diagnosis was atrial fibrillation with a slow ventricular response rate and splintered QRS complexes.

Analysis of the 24-hour Holter recording revealed persistent atrial fibrillation. The heart rate ranged from 70 to 120 beats/min for most of the examination period, and the mean hourly heart rate was 94 beats/min. During times of excitement, the heart rate increased to 150 to 160 beats/min for 1 to 2 minutes. The maximum heart rate was 204 beats/min during the final 3 minutes of the recording (while the monitor was being removed).

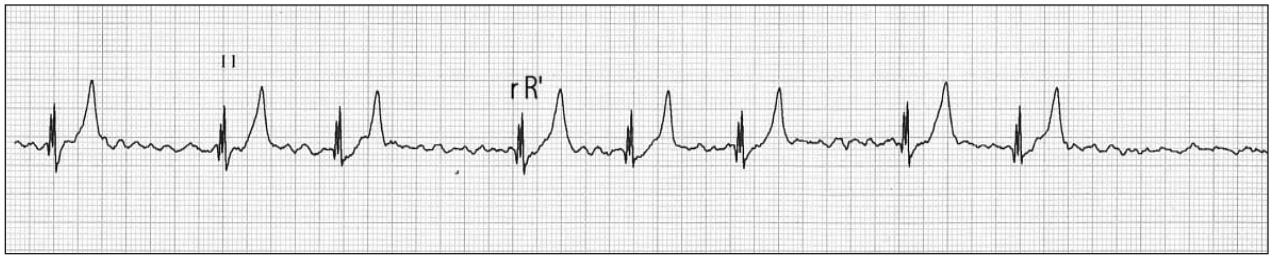

Figure 1—Lead II ECG tracing obtained from a 6-year-old castrated male Greyhound with tricuspid valve dysplasia. Notice the absence of P waves and the irregular, supraventricular rhythm indicative of atrial fibrillation. The ventricular response rate is slow (mean rate, 120 beats/min). The R waves are splintered with an rR′ configuration, which is a common finding in dogs and cats with tricuspid valve dysplasia. Paper speed = 50 mm/s; 1 cm = 1 mV.

Discussion

Until recently, the accepted mechanistic theory for atrial fibrillation has been based on the multiple wavelet hypothesis, which suggests that the atria are activated randomly by many unorganized wavelets.[1,2] However, results of recent studies[1,3] in animals and humans suggest that in some instances, atrial fibrillation may be caused by 1 or a small number of reentrant sites in 1 atrium that fragment to cause a complex pattern of atrial activation. These sites have been termed mother rotors.[3] In dogs, horses, and humans, vagal stimulation and atrial enlargement both predispose to the development of atrial fibrillation.[3,4] Vagal stimulation and cholinergic drugs cause inhomogeneous refractory periods in atrial myocardial cells, predisposing animals to atrial fibrillation. Large-breed dogs, horses, and humans with the aforementioned arrhythmia often have atria that are dilated and increased in size.[4] Although atrial fibrillation may develop in the absence of identifiable cardiac disease (ie, lone atrial fibrillation), it more commonly develops with conditions associated with atrial enlargement, such as dilated cardiomyopathy and severe **atrioventricular (AV)** valve regurgitation.[1,5] In a study by Guglielmini et al,[6] absolute left atrial enlargement represented the best predictor of development of atrial fibrillation in dogs. In addition, atrial fibrillation and other supraventricular arrhythmias have been described in dogs with tricuspid valve dysplasia.[7-9] In the dog of this report, right atrial enlargement secondary to congenital tricuspid valve dysplasia appeared to be the underlying cause of atrial fibrillation.

Atrial fibrillation is usually a rapid rhythm and is identified electrocardiographically as an irregularly irregular supraventricular rhythm without P waves.[10] The depolarization rate of the atria in atrial fibrillation can exceed 500 depolarizations/min, and these depolarizations continually and randomly invade the AV nodal tissue.[1] If the AV node allowed all of the depolarizations to be conducted to the ventricles, ventricular fibrillation could occur; however, the slow-conducting properties of the AV node fibers allow the AV node to act as a filter, conducting only a limited number of fibrillatory waves to the ventricles.[1,4] Despite the filtering effect of the AV node, the ventricular response rate in atrial fibrillation is usually fast, and concealed conduction of impulses into the nodal tissue results in an irregular ventricular response rate.[1,4] The total number of impulses that reach the ventricles primarily depends on the refractory period of the AV node and, to a lesser extent, its conduction characteristics.[11] These variables are markedly influenced by autonomic tone. If vagal tone is low and sympathetic tone is high (as they are in heart failure), a high number of impulses will reach the ventricles.[1,4,5]

Despite the detection of atrial fibrillation in the dog of this report, it was not tachycardic during most of the 24-hour period that it was examined electrocardiographically. Possible causes of a slow ventricular response rate to atrial fibrillation include structural AV nodal disease, hypothyroidism, and high vagal tone. Structural AV node disease, such as fibrosis or inflammation, creates a physical barrier to cardiac impulses. This is an unlikely explanation for the slow ventricular response rate to atrial fibrillation in the dog of this report because it did become briefly tachycardic during removal of the Holter monitor. Hypothyroidism decreases the density of β-adrenergic receptors in myocardial cells, which results in reduced responsiveness to sympathetic stimulation and a slower than normal heart rate.[12] Serum thyroxine concentration in the dog of this report (12.4 ng/mL) was within the reference range (12 to 30 ng/mL), and no other clinical signs suggestive of hypothyroidism were present. High vagal tone prolongs AV node conduction, resulting in fewer impulses traversing the AV node and a slower ventricular response rate. The most likely explanation for the slow ventricular response rate in the dog of this report is that high vagal tone lengthened the refractory time of the AV node, which slowed conduction to the ventricles. The dog may have had more vagal than sympathetic influence to the AV node because it was acclimated to the hospital environment and procedures. Additionally, unlike many dogs with atrial fibrillation, the dog of this report was not in congestive heart failure (a situation in which sympathetic tone predominates and contributes to the tachycardia typically seen with this arrhythmia).

Splintered QRS complexes have been reported as a distinctive and common ECG finding in dogs and cats with tricuspid valve dysplasia.[13,14] Electrocardiographically, approximately 40% of dogs with tricuspid valve dysplasia have splintered QRS complexes that occur with an RR', Rr', or rR' configuration in leads II, III, and aVF.[13,14] In the dog of this report, QRS complexes were splintered with an rR' morphologic appearance in these leads. Additionally, Kornreich and Moïse[13] found that 8 of 39 dogs with right AV valve malformation had high T-wave amplitudes, similar to that detected in the dog of this report. Other ECG abnormalities identified in animals with tricuspid valve dysplasia (including right axis deviation; increased P-wave amplitudes; Q waves deeper than 0.5 mV in leads II, III, and aVF; and deep S waves in precordial leads) were not detected in the dog of this report.[7,14]

Historically, restoration of sinus rhythm has been the main treatment goal in humans with atrial fibrillation; however, conversion to a sinus rhythm may be difficult in patients with severe underlying cardiac disease. Heart rate control is also used in humans with atrial fibrillation and is the most common treatment goal in veterinary medicine.[1,5] Rate control is achieved with drugs that prolong the AV nodal refractory period and slow conduction velocity, such as β-adrenoceptor blockers, calcium channel blockers, and digoxin. Recent medical experience[15] in humans has indicated that rate control may not be inferior to rhythm control for the prevention of death and illness resulting from cardiovascular causes. Conversion to and maintenance of sinus rhythm for the dog of this report would likely have been unsuccessful because of the large size of the right atrium. Because the dog's heart rate was within normal limits and within the suggested target range throughout most of the 24-hour period examined, administration of medication to achieve negative

chronotropism and negative dromotropism was deemed unnecessary.[4]

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