

# ECG of the Month

An 8-year-old 40-kg (88-lb) neutered female Dogue de Bordeaux was referred for evaluation of weakness and dyspnea of 24 hours' duration. On physical examination, the dog had tachypnea, dyspnea, abdominal distension, and pale mucous membranes. Femoral pulses were irregular in rate and intensity. Auscultation revealed muffled heart sounds, and after a lateral thoracic radiographic view was obtained and assessed, pericardial effusion was strongly suspected. Cardiac tamponade was diagnosed on the basis of subsequent echocardiographic findings. Abdominal ultrasonography revealed ascites. Electrocardiographic monitoring was performed before and during pericardiocentesis and revealed supraventricular irregular tachyarrhythmia (rate, 200 beats/min) interrupted by cyclic periods of asystole. The dog recovered well from pericardiocentesis, and 2 days later, echocardiography revealed a heart-base tumor measuring 5 × 4 × 3 cm (1.95 × 1.56 × 1.17 inches), which appeared as a solid mass with central hyperechogenic spots. The mass was located medial to the ascending aorta and dorsal to the pulmonary artery bifurcation and was in contact with the right atrium and cranial vena cava. The rest of the echocardiographic examination findings at that time were unremarkable.

Because sympathetic outflow by the tumor was included in the differential diagnoses, treatment with digoxin<sup>a</sup> (0.005 mg/kg [0.002 mg/lb], PO, q 12 h) was started to decrease the ventricular rate, but the arrhythmia remained unchanged in successive ECG assessments. Attention was paid to the search for a synchro-

nism between the heart rhythm pauses and respiratory cycle, but no relation was found.

## ECG Interpretation

Before and during pericardiocentesis, at a follow-up examination 1 week later (Figure 1), and at subsequent assessments, ECG monitoring revealed evidence of the same supraventricular irregular tachyarrhythmia with cyclic periods of asystole. In 3-lead ECG tracings, the supraventricular tachycardia was associated with a QRS complex duration of 60 milliseconds (reference range, < 70 milliseconds) and irregularly irregular R-R intervals. The baseline was irregular because of the presence of f waves, indicative of chaotic atrial discharge. Ventricular rate was variable and characterized by a cyclic trend of 6 to 8 consecutive beats at a high rate (150 to 200 beats/min), followed by 1 to 4 consecutive beats at a slower rate (80 to 45 beats/min). The asystolic pause of longest duration recorded at the follow-up examination 1 week after the dog underwent pericardiocentesis was 1.4 seconds, but longer pauses were observed in the ECG tracings obtained during pericardiocentesis. The ECG diagnosis was atrial fibrillation (AF). The atrioventricular (AV) conduction was cyclically slowed but unaffected by digoxin administration.

Two months after the pericardiocentesis, the dog's physical condition began to worsen. At that time, cor pulmonale and the absence of pericardial effusion were noted via echocardiography, but asystolic pauses were not detected with the simultaneous ECG signal on the echographic screen. Blood samples were collected for analysis in an attempt to identify other possible illnesses or complications. Results indicated that the dog had leishmaniasis, and subsequently, because of abrupt clinical deterioration, the dog was euthanatized and underwent necropsy. The heart-base tumor was identified as a chemodectoma with metastatic foci in the spleen and pancreas. Immunohistochemical analysis of samples of the tumor revealed that the tissue was

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Figure 1—Lead I, II, and III ECG tracings obtained from a Dogue de Bordeaux with an echocardiographic diagnosis of heart-base chemodectoma. The dog initially had cardiac tamponade and recovered well from pericardiocentesis; these tracings were obtained 1 week after that procedure. Notice the irregularly irregular R-R intervals together with the absence of P waves, which are replaced by f waves. Cyclic slowing of ventricular rate is also noticeable, highlighted by the variable duration of R-R intervals and changes in heart rate (HR). Before and during pericardiocentesis and during 2 months of subsequent observation, this arrhythmia was detected repeatedly during serial ECG examinations. Paper speed = 25 mm/s; 1 cm = 1 mV.

negative for chromogranin A and positive for neuron-specific enolase. An interesting gross finding was the presence of multifocal (0.2- to 0.3-cm-diameter) solid whitish areas in the AV junction wall at the midpoint of Koch's triangle. In particular, in longitudinal sections of the AV junction, these lesions were located cranial to the coronary sinus and caudal to the septal leaflet of the tricuspid valve. Histologic examination of multiple sections of the AV junction at the level of Koch's triangle revealed large areas composed of fibrous and chondroid tissue with severe compression of the AV node and bundle of His. A large and transmural area of replacement fibrosis and chondroid metaplasia was observed in the right atrium.

## Discussion

Atrial fibrillation is common in giant breeds of dog even in the absence of structural abnormality such as atrial dilation (lone AF).<sup>1</sup> Atrial fibrosis in the absence of dilation can also function as an arrhythmogenic substrate, representing an inhomogeneous conducting tissue or tissue that is able to facilitate the development of ectopic pacemakers.<sup>2</sup> However, atrial fibrosis can be either the cause or the effect of AF. In the dog of this report, a large and transmural area of replacement fibrosis and chondroid metaplasia was observed in the right atrium, which was considered a result of regional ischemia caused by mass compression or by lone AF. High plasma concentrations of catecholamines, such as those generated by functioning chemoreceptor tumors, can be responsible for initiation of various types of arrhythmias (including AF) in dogs.<sup>3</sup> Functioning aortic body tumors may develop in dogs but have not been identified, to our knowledge.<sup>4-6</sup> For the dog of this report, urine was not collected over a 24-hour period for analysis and the functioning properties of the tumor could not be determined. However, results of immunohistochemical analyses were not consistent with a secretory function, given that the tumor tissue was negative for chromogranin A, a protein common in neurosecretory cells.<sup>6</sup>

On the basis of these considerations, atrial fibrosis was most likely the cause or at least the substrate of AF in the dog of this report. The preexistence of the arrhythmia before the pericardial puncture definitively ruled out the sudden onset of AF as a result of atrial stretch or other complications following pericardiocentesis.<sup>7</sup> With regard to the cyclic delayed AV conduction evident in the dog, various factors would be able to influence AV conduction during AF: autonomic tone imbalance, concealed conduction, drugs, and intrinsic AV nodal disease.<sup>8,9</sup>

Typically, lone AF in giant-breed dogs is characterized by slow heart rate; however, increasing sympathetic tone (as in cases of heart failure) would understandably increase AV conduction and cause greater ventricular response.<sup>10</sup> In the dog of this report, the same heart rate variability was observed when either sympathetic or vagal dominance was expected (ie, with and without cardiac tamponade). The dog had the same variable ventricular response in different conditions and settings. No relationship between heart rate and respiratory rate

or cycle was identified. Long R-R intervals were observed during expiration as well as during inspiration, thereby excluding a possible ventilatory vagal stimulation. Slow ventricular response to AF is considered a consequence of persistent slow AV conduction associated with high vagal activity and can disappear with exercise or excitement.<sup>8</sup> Moreover, for the dog of the present report, administration of digoxin (a drug known to increase the refractoriness of the AV node) resulted in no variations in AV conduction. Although  $\beta$ -adrenergic receptor blockers are perhaps more useful for unmasking a functioning chemoreceptor tumor and for neutralizing its concomitant catecholamine-related adverse effects, such drugs were intentionally avoided in the case of the present report because of the potential for secondary hypotensive effects, especially in a dog with AF and at risk for cardiac tamponade. Neoplasia of the AV node, endocarditis, myocarditis, borreliosis, and hypothyroidism were ruled out specifically on the basis of the results of echocardiography, laboratory testing, and necropsy. For the dog of this report, the only concomitant disease was leishmaniasis, which was judged to be irrelevant and not related to the arrhythmia because typical signs of that disease were not observed.

Concealed AV conduction during AF is the mechanism responsible for the typical irregularity of R-R intervals.<sup>8</sup> The concealment is provided by atrial impulses that are unable to emerge from the AV node because of the refractoriness of the tissue in that instant, but that are responsible for influencing the refractory period of AV node and inhibiting the conduction of successive impulses. However, for the dog of this report, large areas of chondroid metaplasia and fibrous tissue in the AV junction were detected during necropsy. The degenerative tissue was located in the posterior aspect of AV node. This anatomic region has been associated in various species with the location of the so-called slow pathway, a component of a dual nodal AV pathway. The latter, although not yet fully elucidated, is based on the existence of 2 pathways, one slow and the other fast, with different refractory periods.<sup>11,12</sup> The 2 pathways can generate high heart rate during AV node reentrant tachycardia and AF by providing 2 means of propagating the atrial wave fronts toward the ventricles.

Ablation of the slow pathway is a clinical electrophysiologic procedure used in humans to decrease the ventricular rate during AF because that pathway has the shorter refractory period and allows conduction of many atrial impulses.<sup>11,12</sup> The slow pathway ablation technique is also called modification of the AV node and is preferred to complete ablation because the latter results in complete AV block and warrants the implantation of a pacemaker. In contrast, the AV node modification procedure results in decreases in ventricular rate similar to that observed in the dog of this report.<sup>11</sup> Specifically, injection of fibroblasts in that site slows ventricular response during AF in dogs.<sup>13</sup>

At the initial evaluation and at all subsequent follow-up examinations in the dog of this report, the asystolic pauses were not observed concomitantly with cor pulmonale, but were present and had a cyclic alternation with tachycardia. These observations suggested a possible reciprocation between heart rate and cham-

ber filling. At a slow heart rate, there was an increase in chamber volume via distension of myocardial fibers and the slow pathway was less compressed by the surrounding degenerative tissue, thereby enabling it to conduct more impulses; the resultant increase in heart rate provided less chamber filling, shortening of myocardial fibers, and compression of the slow pathway with a renewed decrease in heart rate. Because other factors able to influence AV conduction were not identified in the dog of this report, a degenerative process in the tissue of the posterior region of the AV node was considered the most probable cause of cyclic delay in the AV conduction. The implication of a dual nodal AV pathway was rationally suspected.

a. Lanoxin, GlaxoSmithKline, Verona, Italy.

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