ECG of the Month

A 6-year-old spayed female Basset Hound was referred to the Texas A&M University Veterinary Medical Teaching Hospital for evaluation of sudden onset of lethargy, polyuria-polydipsia, peripheral edema, abdominal distention, diarrhea, and hypoalbuminemia. The dog had a history of hypothyroidism, hyperadrenocorticism, and recurrent urinary tract infections. At the time of this initial evaluation, it was being treated with thyroid hormone supplementation, trilostane, nitrofurantoin, vitamin C (to control urine pH), and diphenhydramine (for seasonal allergies).

At the initial examination, the dog was bright, alert, and mildly tachypneic (respiratory rate, 42 breaths/min). Thoracic auscultation revealed normal bronchovesicular sounds, muffled heart sounds, and occasional premature beats with corresponding peripheral pulse deficits. The dog’s abdomen was pendulous with an evident fluid wave on ballottement. Mild peripheral edema was present. Multiple erythematous areas with crusts and scale were visible on the thorax and abdomen.

Diagnostic assessments included serum biochemical analyses, CBC, urinalysis, and assessment of the urine protein-to-creatinine ratio. Because of the abdominal distention, hypoalbuminemia, and history of hyperadrenocorticism, abdominal ultrasonography, abdominal fluid analysis, and serum bile acids assay were performed. Cardiothoracic evaluation included thoracic radiography, indirect blood pressure measurement, echocardiography, and ECG. Serum biochemical abnormalities included hypoproteinemia with hypoalbuminemia, hypomagnesemia, hypokalemia, and hypocholesterolemia. A CBC revealed a stress leukogram, but no other abnormalities. Via urinalysis, hypoalbuminemia without proteinuria was detected. Because the dog had hypoalbuminemia and hypoalbuminemia, the urine protein-to-creatinine ratio was assessed to further evaluate for proteinuria; results indicated the urine protein concentration was mildly high. Abdominal ultrasonographic findings included a large amount of echogenic effusion, evidence of chronic cholecystitis, and possible localized peritonitis in the portal region; rupture of the gall bladder was suspected. Abdominal fluid analysis revealed a modified transudate, and pre- and postprandial serum bile acids concentrations were within reference limits. Abnormal findings detected via thoracic radiography included car-

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**Figure 1**—Six-lead ECG trace obtained from a dog that was evaluated because of the sudden onset of lethargy, polyuria-polydipsia, peripheral edema, abdominal distention, diarrhea, and hypoalbuminemia. An irregular rhythm and negative P′ waves are present in lead II and are consistent with an underlying atrial arrhythmia. Notice that the P′ waves are negative and most are wide. The P′-R interval is prolonged, which is consistent with first-degree atrioventricular block; the P′-P′ interval is irregular, similar to findings associated with sinus arrhythmia. The T waves are narrow and upright, and their identification is aided by a constant Q-T interval. Two QRS complexes are preceded by a positive P wave in lead II (asterisks). The P waves are positive in leads I, III, and aVF; negative in lead aVR; and isoelectric in lead aVL. The morphology of the P waves that precede these 2 complexes differs from the P′ wave morphology in all leads except lead I. Paper speed = 25 mm/s; 1 cm = 1 mV.

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diomegaly with possible pulmonary edema and slight pleural effusion. Indirect systolic blood pressure was measured by use of an oscillometric flow detector; the dog had mild systolic hypertension (systolic arterial blood pressure, 162 mm Hg [reference range, 120 to 152 mm Hg]); diastolic arterial blood pressure, 64 mm Hg [reference range, 72 to 90 mm Hg]; mean arterial blood pressure, 111 mm Hg [reference range, 90 to 112 mm Hg]). Echocardiography revealed no abnormal cardiac morphology or function and no pericardial effusion; however, diastolic mitral valve regurgitation associated with periods of second-degree atrioventricular (AV) block was detected. An ECG was recorded (Figure 1).

**ECG Interpretation**

Electrocardiographic findings included an irregular rhythm and negative P' waves in the lead II trace, which were consistent with an underlying atrial arrhythmia. The P waves occurred without QRS complexes, resulting in a ventricular rate of approximately 90 beats/min and an atrial rate of 110 beats/min. The ECG revealed 2 QRS complexes that were each preceded by a P wave that was positive in leads I, II, and III; that suggested a P wave origin at or near the sinus node. However, the beats that were conducted with positive P waves occurred with a slightly shorter P'-P interval (380 milliseconds) than the beats conducted with negative P' waves, which had a variable P'-P' interval. The P'-P' interval ranged from 420 to 470 milliseconds, which was similar to findings associated with sinus arrhythmia. The P'R interval was prolonged and variable (range, 180 to 240 milliseconds), which was consistent with first-degree AV block. At 1 point during the ECG examination, the P'R interval became gradually prolonged prior to the nonconducted P' waves, which was consistent with second-degree AV block (Mobitz type I). The P' waves were negative in leads II, III, and aVF; positive in leads aVR and aVL; and mostly isoelectric in lead I. Thus, the atrial mean electrical axis (MEA) was approximately –90° (Figure 2). Most of the P' waves were also wide (80 milliseconds) and occasionally notched, suggestive of a large left atrium or aberrant intra-atrial conduction abnormality. The T waves were narrow and upright, and their identification was aided by a constant Q-T interval.

The presence of negative P' waves in the ECG recordings was consistent with an idioatrial rhythm that originated either in the caudal portion of left atrium, in the caudal portion of the right atrium near the tricuspid valve annulus, or in the caudal aspect of the AV node and that was conducted with first- and second-degree AV block. There appeared to be 1 period during which the sinus node overrode the left atrial rhythm, resulting in 2 sinus beats that were conducted with first-degree AV block.

**Discussion**

An idioatrial rhythm develops as a result of a combination of an accelerated ectopic atrial pacemaker coupled with either depression of sinus impulse formation or depression of sinus impulse conduction. Any factor that depresses the sinus rate, such as high vagal tone, to a rate less than that of an ectopic atrial pacemaker will allow the ectopic pacemaker to become the dominant pacemaker. Likewise, any factor that impedes conduction of the sinus impulse to the ectopic atrial focus, such as sinoatrial block, will allow the impulses originating in the ectopic focus to be detectable on the surface ECG. In each instance, the ectopic atrial pacemaker will continue to discharge at a regular rate until the sinus rate increases and exceeds that of the ectopic atrial rate or the sinoatrial block dissipates. Ectopic atrial pacemakers may be unifocal, but are often multifocal, resulting in multiform P'
waves that have the appearance of a wandering atrial pacemaker.\(^a\)

Mapping of ectopic atrial foci has been performed extensively in human medicine, primarily for the purpose of localizing atrial tachycardia for radiofrequency ablation. Specific surface ECG characteristics can often be associated with particular foci within the atria. Limb leads can provide some direction regarding localization; however, evaluation of precordial lead and, ultimately, intra-atrial recordings provides additional information for localization.\(^3\) Negative P waves in leads II, III, and aVF are detected in people with a left atrial, nodal, or coronary sinus focus.\(^2\) In 1 study\(^4\) of 31 humans, a positive P wave in lead aVL predicted a right atrial focus with a sensitivity and specificity of 88% and 79%, respectively. Specifically, positive P waves in leads I, aVR, and aVL and negative P waves in leads II, III, and aVF have been associated with a focus near the tricuspid valve annulus.\(^4\) Data from precordial leads can be integral for accurate determination of the location of ectopic atrial foci—a positive P wave in leads V1 and V6 confirms a left atrial focus.\(^5\) When the ECG recordings from the dog of this report were interpreted in light of the atrial MEA and morphology of the P' waves, an origin in the left atrium was suspected.\(^4\) In the absence of precordial lead data, the definitive origin of the idioatrial rhythm in the dog of this report could not be determined.

Ectopic atrial impulses can be conducted with normal intra-atrial, AV nodal, and intraventricular conduction.\(^2\) However, those impulses are also susceptible to interference at any of these levels of conduction. Within the atria, the ectopic atrial impulse may occur simultaneously with a sinus impulse, resulting in an atrial fusion complex with a P wave configuration that has a hybrid appearance of that of the sinus P wave and ectopic P' wave.\(^5\) The atrial escape impulse may also occur simultaneously with an AV nodal or ventricular escape beat, causing the 2 impulses to meet within the AV node and appear as a P' wave that is dissociated from the AV nodal or ventricular escape complex.\(^5\)

Idioatrial rhythm, first- and second-degree AV block, and irregular P'-P' interval support the presence of increased vagal tone,\(^2,5\) which may have been associated with concurrent diseases of the gastrointestinal and urinary tracts in the dog of this report. An exploratory laparotomy was performed, and biopsy specimens of several tissues were obtained. Histopathologic findings confirmed steroid hepatopathy and minimal to mild, nonspecific inflammation of the small intestine, abdominal wall, and mesenteric lymph nodes. The dog was treated with broad-spectrum antimicrobial agents, milk of magnesia, vitamin C, trilostane (for hyperadrenocorticism), and thyroid hormone supplementation (for hypothyroidism); the owner was instructed to feed it a more highly digestible diet. After 3 months, the referring veterinarian reported that the dog appeared to be well and was eating apparently normally. Ten months after the laparotomy, the dog continued to have recurrent urinary tract infections, but was otherwise doing well.

References