ECG of the Month

This feature is being sponsored by the Academy of Veterinary Cardiology. Readers of the *JAVMA* are invited to submit contributions. Contributions should include: a brief description of the case (150 words); good contrast glossy photographs (5 in × 7 in) of tracings, with ECG lead, voltage calibration scale, and paper speed indicated; and a discussion of the abnormality.

Send comments and tracings to Dr. Phillip Ogburn, Department of Small Animal Clinical Sciences, College of Veterinary Medicine, University of Minnesota, 1352 Boyd Ave, St Paul, MN 55108, or Dr. John-Karl Goodwin, Department of Veterinary Clinical Science, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA 70803-8410.

A 9-year-old 15-kg male Tibetan Terrier was evaluated because of ascites and subcutaneous edema of approximately 2 months' duration. Previous evaluation by the referring veterinarian had included a CBC and serum biochemical analysis, results of which were within reference limits. Proteinuria (3+) was found on urinalysis of a free-catch sample. Ascites and subcutaneous edema had initially responded to treatment with furosemide (2.5 mg/kg of body weight, PO, q 48 h), but had become increasingly refractory to treatment.

Abnormal physical examination findings included generalized muscle wasting; abdominal distention, with a palpable fluid wave; and jugular venous distention. Heart rate was 78 beats/min. On admission, thoracic radiography revealed generalized cardiomegaly, with biatrial enlargement and a distended caudal vena cava. Results of analysis of fluid obtained by paracentesis were consistent with a modified transudate (protein, 3.1 g/dl). An ECG was obtained.

Echocardiographic findings included severe right atrial and left atrial enlargement (left atrial diameter, 3.16 cm), as well as right ventricular enlargement. Left ventricular dimensions and systolic function variables were within reference limits. A mitral A wave was not detected on M-mode and Doppler echocardiography. Color flow mapping did not document mitral or tricuspid valve regurgitation.

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ECG Interpretation

Heart rate was 70 beats/min. P waves were not evident on any of the 6 frontal leads (Fig 1). The QRS duration was within reference range. Examination of ECG revealed atrial standstill, with a nodal or high-ventricular escape rhythm (Fig 2). The R-wave amplitude in lead II (3.0 mV) exceeded reference limits for a small-breed dog, and was suggestive of left ventricular enlargement. The mean electrical axis (49°) was within reference range. These findings were compatible with temporary or persistent atrial standstill, with possible left ventricular enlargement.

Discussion

Electrocardiographic features of atrial standstill include lack of detectable P waves in any lead and a slow junctional or high-ventricular escape rhythm, with nearly normal QRS complex shape.[1] Atrial standstill can be a temporary condition associated with hyperkalemia, hypothermia, or drug toxicosis, or may represent a terminal cardiac event.[2,3] Persistent atrial standstill is defined as the presence of atrial standstill despite normal electrolyte concentrations and body temperature, and without a history of exposure to drugs.[3]

Clinical features of persistent atrial standstill may include weakness, syncope, and dyspnea, with or without ascites, pulmonary edema, or pleural effusion.[4] Although the nodal or ventricular pacemaker usually prevents a profoundly slow heart rate, clinical signs may develop because of loss of the atrial contribution to ventricular filling. Animals with normal ventricular function should be able to maintain normal cardiac output at rest, without the atrial contribution to left ventricular filling. However, at a higher heart rate (as with exercise), ventricular filling time is decreased and compensation may not be possible, resulting in clinical signs. Additionally, in an animal with decreased ventricular systolic function or impaired ventricular filling, the loss of the atrial contribution to ventricular filling may result in profound clinical signs.

Persistent atrial standstill is typically divided into 2 subsets. The first is persistent atrial standstill secondary to chronic cardiac disease with diffuse atrial involvement (eg, valvular dysfunction, congenital heart disease, cardiomyopathy, and myocarditis). With such diseases atrial standstill may be temporary at first, with conversion to normal sinus

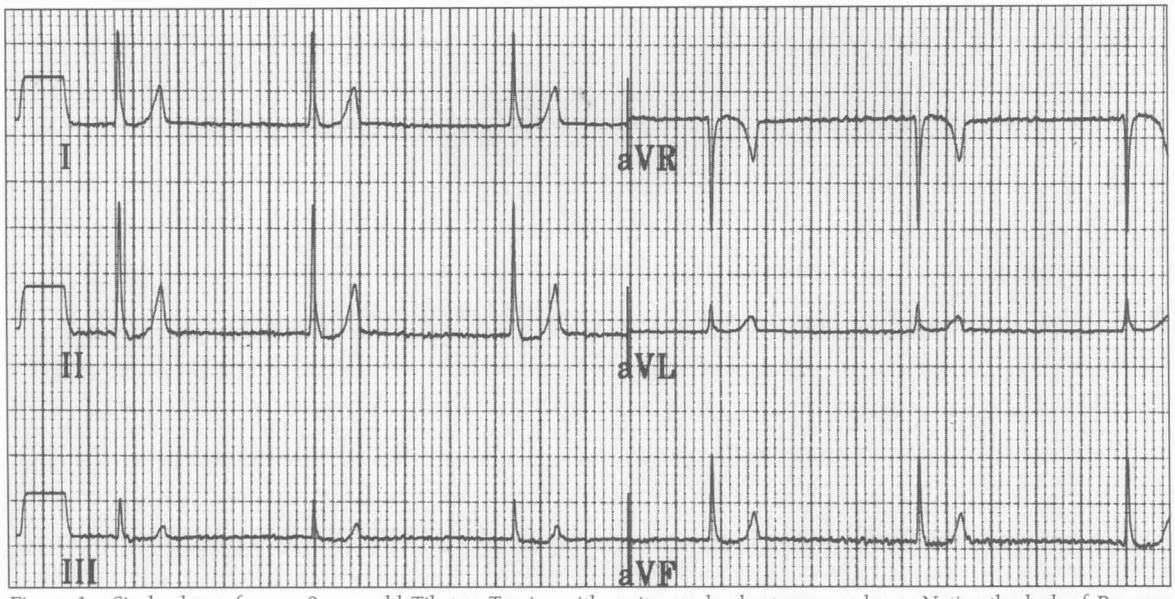

Figure 1—Six-lead ECG from a 9-year-old Tibetan Terrier with ascites and subcutaneous edema. Notice the lack of P waves in all leads. Paper speed, 25 mm/s; 0.5 cm = 1 mV.

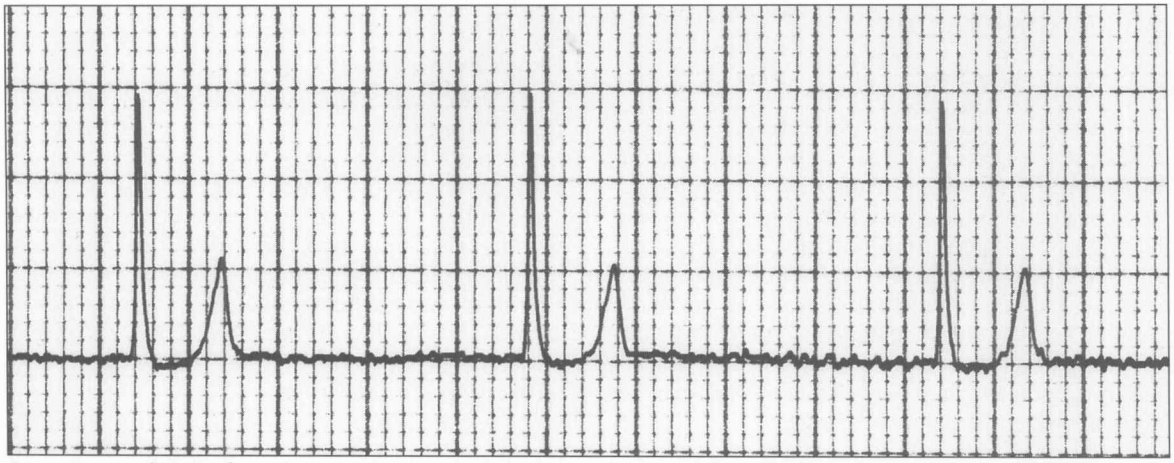

Figure 2—Lead-II ECG from the same dog. Notice the lack of P waves and the tall R waves. Paper speed, 25 mm/s.

rhythm without treatment, but eventually will become permanent. This course is most likely attributable to progression of atrial enlargement, with diffuse disruption and fibrosis.[2,5]

The second type is persistent atrial standstill caused by neuromuscular disease with atrial involvement.[5] The most common neuromuscular disease responsible for persistent atrial standstill in human beings is Emery-Dreifuss muscular dystrophy, an X-linked disorder characterized by slowly progressive muscle wasting and weakness, with a humeral-peroneal distribution.[2] Although this condition has not been documented in dogs, several cases of persistent atrial standstill in dogs have been associated with severe muscle wasting.[3]

Persistent atrial standstill in dogs has been reported most commonly in English Springer Spaniels, but other affected breeds include Shih Tzu, Old English Sheepdog, German Shorthaired Pointer, and mixed-breed dogs.[6,7] Persistent atrial standstill in human beings has been found sometimes to be familial.[2]

A progressive idiopathic myocardial disease, characterized by myocardial destruction, fibrosis, and atrial standstill, also has been reported in dogs.[8] The atria are most severely affected, but the ventricles, particularly the right ventricle, can be involved. This syndrome has been referred to as atrioventricular myopathy (AVM).

The etiopathogenesis of the disease in this dog is unknown. Clinical history or echocardiographic evidence of long-standing cardiac disease was not found. The possibility of a neuromuscular syndrome in this dog was supported by the observed muscle wasting. Although muscle wasting also could have been a consequence of the cachexia commonly associated with heart failure, a primary cause of heart disease was not determined.[9] In such cases, a diagnosis of AVM or an isolated case of persistent atrial standstill should be considered.

Why this dog had clinical signs consistent with congestive heart failure, with a normal heart rate (70 beats/min) and apparently normal cardiac function, is not clear. Although echocardiographic variables of ventricular function (fractional shortening, end-systolic ventricular index) were within reference ranges, these are indicative of primarily left ventricular systolic function. In this dog, clinical signs of right-sided heart failure predominated (ascites, jugular venous distention). Unfortunately, the accuracy of noninvasive assessment of right ventricular function is questionable. Because AVM is characterized by myocardial destruction and fibrosis, this syndrome could have affected ventricular diastolic function considerably.[8] Right ventricular diastolic function, however, was not evaluated.

Current therapeutic recommendations for permanent atrial standstill suggest that the most reliable method of treatment is permanent ventricular pacing.[3] However, adrenergic drugs may sometimes be helpful on a temporary basis, by increasing the rate of the escape pacemaker. Terbutaline has been suggested to be the most useful orally administered medication, although vagolytic drugs are usually not effective.[8]

The prognosis for animals with persistent atrial standstill is guarded at best.[8] The owners of this dog declined pacemaker implantation, and the dog was managed conservatively with medical treatment, including furosemide (2 mg/kg, PO, q 12 h) and enalapril (0.5 mg/kg, PO, q 24 h). Eight months after initial evaluation, the dog has only mild clinical signs (persistent ascites).

References

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