ECG of the Month

A 9-year-old neutered male Cocker Spaniel was referred to the Texas A&M University Veterinary Medical Teaching Hospital for evaluation because of episodes of collapse that progressively increased in number over a 4-day period. The episodes were characterized by sudden collapse for 3 to 5 seconds with spontaneous recovery. A diagnosis of hypothyroidism had been made previously, and the dog was receiving thyroid hormone supplementation. On physical examination, the dog had an irregular cardiac rhythm and periods of tachycardia (typically lasting 2.5 seconds, during which heart rate was as high as 200 beats/min) alternated with pauses lasting up to 3 seconds, during which no heartbeat was detectable. Femoral pulses were strong and synchronous with the heartbeat during periods of tachycardia and absent during the intervening pauses. A grade 4/6 left apical systolic murmur consistent with mitral regurgitation was ausculted. Other physical examination findings were unremarkable. Diagnostic assessments included ECG, blood analyses, thoracic radiography, and echocardiography. A serum biochemical profile and CBC revealed no abnormalities. Radiographic findings included mild left atrial enlargement without left ventricular enlargement or evidence of congestive heart failure. Echocardiography revealed thickening of the mitral valve leaflets with moderate mitral regurgitation and mild left atrial enlargement. On the basis of fractional and area shortening measurements, left ventricular systolic function was apparently normal to increased, except during times of sinus arrest. The echocardiographic findings were consistent with chronic degenerative valvular disease. An ECG trace was recorded (**Figure 1**).

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ECG Interpretation

Electrocardiography revealed bursts of sinus tachycardia at a rate of 214 beats/min that alternated with periods of sinus arrest of 2.8 seconds' duration, indicative of sick sinus syndrome (Figure 1). The R waves were tall (3.0 mV), suggestive of left ventricular enlargement; a slight jag was detected in the downstroke of the R wave, which was suggestive of microabberant conduction.[1] An endocardial pacemaker programmed in ventricular demand (VVI) mode, which indicates that a single lead was used to sense and pace the ventricle, was successfully placed transvenously, and the dog recovered from the procedure without complication. The paced rate was set at 80 beats/minute. An ECG was recorded 24 hours postoperatively (**Figure 2**). Sinus beats alternated with complexes that lacked P waves, which were initiated by a pacing spike artifact. The instantaneous rate of the sinus beats was 136 beats/min. Sinus beats were narrow and upright in lead II, resembling the sinus

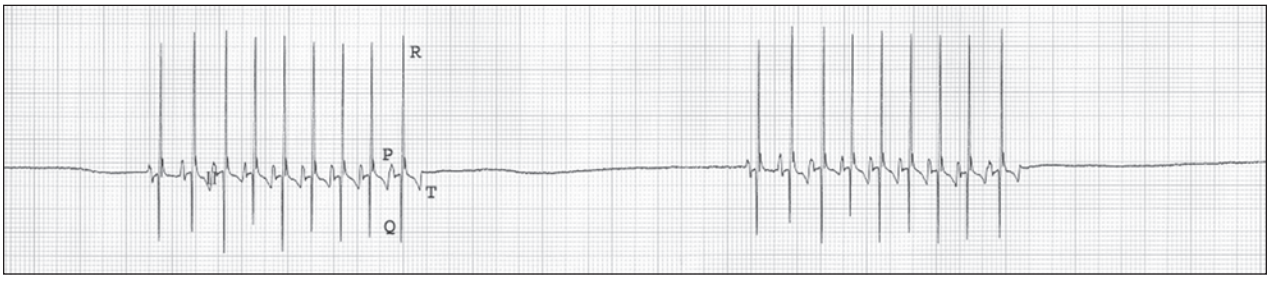

Figure 1—Lead II ECG rhythm strip obtained from a dog with sick sinus syndrome. Periods of sinus tachycardia alternate with periods of sinus arrest. Typical P, Q, R, and T waves are labeled. Paper speed = 25 mm/s; 1 cm = 1 mV.

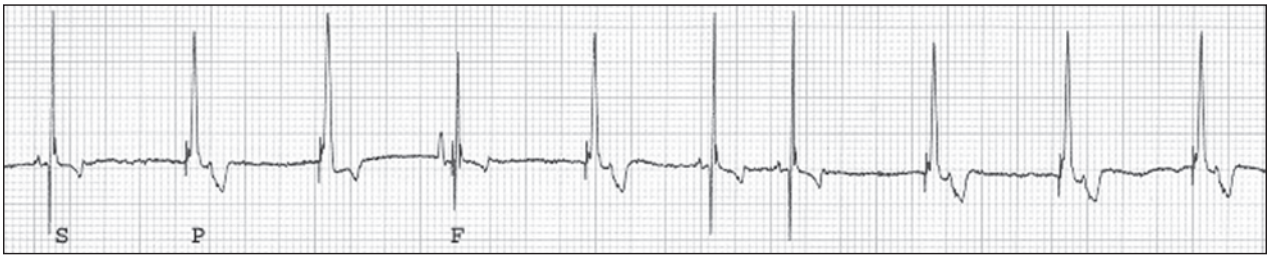

Figure 2—Portion of the lead II ECG recording from the dog in Figure 1 obtained after pacemaker placement. The sinus beats (S) occur at various intervals (eg, beats 1, 6, and 7 from the left) among paced beats (P) initiated by a pacing spike artifact. A single fusion beat (F) indicates that a sinus beat and paced beat occurred simultaneously. The pacemaker is effecting appropriate capture and sensing. Paper speed = 25 mm/s; 1 cm = 1 mV.

beats in the previously recorded ECG (Figure 1). All other beats were initiated by a relatively small pacing spike artifact indicative of a bipolar pacing lead.[2] The paced QRS complexes were wide (0.08 seconds) and had a positive deflection in lead II in accordance with the right ventricular location of the pacing lead. The paced rate was confirmed to be 80 beats/minute. P waves were present within the QT interval of the paced beats, representing atrioventricular dissociation. Examination of the ECG trace revealed normal capture and appropriate sensing by the pacemaker, which indicated that it was functioning properly. One beat was initiated by a P wave, and a pacing spike artifact was present at the onset of the QRS complex; the morphology of the QRS complex was similar to that of the sinus beats, but the complex was comparatively smaller. This complex represented a fusion of sinus and paced beats (Figure 2).

Discussion

Sick sinus syndrome reportedly[1,3] affects Cocker Spaniels as well as Miniature Schnauzers, West Highland White Terriers, Pugs, and Dachshunds. Sinoatrial nodal alterations associated with sick sinus syndrome are caused by structural abnormalities that result in a combination of arrhythmias; clinical signs include weakness and syncope.[3] Abnormalities including sinus bradycardia, sinoatrial block, sinus arrest, and paroxysms of supraventricular tachycardia may be detected electrocardiographically as isolated arrhythmias or in combination.[4] Sinus bradycardia and sinus arrest may result from structural sinoatrial nodal disease or from increased vagal tone. These can be differentiated by attempts to manipulate heart rate. If heart rate does not increase when sympathetic tone is augmented with exercise or by the administration of a parasympatholytic agent such as atropine, structural nodal disease is indicated. Often, the atrioventricular node and bundle branches are also dysfunctional, resulting in atrioventricular or bundle branch block.[4] Bradycardia-tachycardia syndrome refers to the presence of both slow and fast rhythms, as detected in the dog of this report.[1,4] In some instances, bradycardia develops as a result of the previous episode of tachycardia, referred to as overdrive suppression.[4] This occurs when sinoatrial activity is suppressed after a period of tachycardia and can be managed by preventing the tachycardia via administration of antiarrhythmic medications, including β-adrenergic receptor blockers and calcium channel blockers. Syncopal events can result from bradycardia or tachycardia. Pacemaker implantation is required to treat periods of sinus arrest that are associated with clinical signs, particularly prior to administration of antiarrhythmic agents that may result in slowed conduction and a decrease in heart rate. Twenty-four hour ambulatory ECG recordings may aid clinicians in identifying which component of the arrhythmia is the source of the syncope. If the syncopal events continue after pacemaker implantation as a result of persistent tachycardia, antiarrhythmic medications (including β-adrenergic receptor blockers and calcium channel blockers) can be administered without concern of prolonging the periods of sinus arrest or resultant clinical deterioration.

The pacemaker in the dog of this report was set in VVI mode, a 3-letter code indicating that the ventricle is the cardiac chamber being paced and sensed and that pacemaker activity is inhibited when inherent beats occur.[5] When evaluating pacemaker function electrocardiographically, it is important to assess capture and sensing. Capture refers to the ability of the pacemaker to electrically induce ventricular depolarization that results in myocardial contraction.[2] Inherent myocardial electrical activity is sensed by the pacemaker, and pacemaker activity is subsequently inhibited. If the patient's heart rate is lower than the set paced rate, the pacemaker will fire after waiting an appropriate interval, which was equivalent to the paced rate in the dog of this report.[6] In some instances, an inherent sinus beat and paced beat discharge at the same time, creating a fusion beat.[7,8] Pseudofusion describes the superimposition of a pacing spike artifact within the QRS complex of an inherent sinus beat that does not affect its conduction.[8] Both fusion and pseudofusion can occur in any patient with an implantable pacemaker. After pacemaker implantation, the dog of this report has had no further syncopal events to date.

References

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