A 14-year-old 5.5-kg (12.1-lb) Miniature Schnauzer was referred for evaluation of syncopal episodes during the preceding 3 days. Auscultation revealed an irregular rhythm, with tachycardia alternating with bradycardia and pauses. The dog was receiving treatment with enalapril maleate (0.25 mg/kg [0.11 mg/lb], PO, q 24 h) because of cardiac enlargement that had been detected radiographically 6 months before. No abnormalities were detected via a CBC; results of serum biochemical analyses indicated that renal variables and potassium concentration were within reference limits. Systolic arterial blood pressure (determined via a Doppler ultrasonographic method) was 140 mm Hg. Electrocardiography was performed as part of the diagnostic testing (Figure 1).

**ECG Interpretation**

Electrocardiography was performed with a 1-channel electrocardiograph (Figure 1). The ECG findings...
were interpreted as atrial standstill or sinus arrest with junctional escape beats (lead I) and ventricular escape rhythm (leads II through aVF). Bradycardia (heart rate, 60 beats/min) was evident in the lead I trace; no P waves were identified, and the QRS complexes were narrow throughout all lead traces. However, heart rate varied among the lead tracings. On lead II, the heart rate was 120 beats/min; there were no identifiable P waves, and the QRS complexes were prolonged (0.07 seconds) and notched, suggestive of an idioventricular or ventricular escape rhythm. In the aVR trace, there was evidence of a normally conducted supraventricular junctional beat, yet without an identifiable P wave.

The dog underwent Holter monitoring for 24 hours. During that period of monitoring, abnormal sinus automatism (sinus arrest or sinoatrial block; Figure 2) with pauses up to 7 seconds (not shown) and periods of atrioventricular block were detected. Frequent supraventricular tachycardia periods, periods of various degrees of atrioventricular block, and isolated or paired ventricular premature complexes with R-on-T phenomenon were also observed (Figure 3). On some occasions, a short PR interval was also evident. Overall, the ECG findings were suggestive of an atypical sick sinus syndrome (SSS). Pacemaker implantation was suggested to the referring veterinarian and owner of the dog.

Discussion

Sick sinus syndrome comprises a variety of conditions involving sinoatrial node dysfunction, resulting in abnormal conduction. It has also been described as tachycardia-bradycardia syndrome or sinoatrial syncope. The etiology of the syndrome is not well understood, and most cases are considered idiopathic. Although the dog of this report was a male Miniature Schnauzer, SSS more commonly develops in females of this breed. However, SSS in other dog breeds such as Dachshund, Pug, West Highland White Terrier, Cocker Spaniel, and Boxer has also been reported, and there is a case report involving a Bull Terrier.

Associated conditions include replacement of the sinoatrial node by fibrous tissue, possibly related to cardiomyopathy (eg, muscular dystrophy, chronic atrial myocarditis, or dilated cardiomyopathy) or genetic alterations (as in the Miniature Schnauzer breed). Extracardiac causes (eg, immune-mediated, endocrine, metabolic and neurologic disturbances, and

![Figure 2](image-url) — Tracings obtained during a 24-hour period of Holter monitoring of the dog in Figure 1. A combination of tachycardia and bradycardia is evident. In the upper tracing, the first 3 complexes (only half of the first QRS complex is shown) are at the end of an episode of supraventricular tachycardia, which is followed by atrioventricular block with different P-P intervals. The fourth P wave is conducted with a longer PR interval, compared with the following one, and there is a pause of almost 3 seconds followed by junctional escape beats with varying R-R intervals. In the lower tracing, sinus bradycardia with pauses of approximately 2 seconds’ duration followed by a junctional escape beat is apparent. Numbers at the top represent the R-R intervals in milliseconds. Paper speed = 25 mm/s; 1 cm = 1 mV.

![Figure 3](image-url) — Tracings obtained during a 24-hour period of Holter monitoring (3 channels; 1 tracing/channel) of the dog in Figure 1. In panel A, supraventricular tachycardia paroxysm (first 7 beats) followed by 3 sinus complexes and a run of faster rate (premature nonsinus) P waves (white arrows) with various degrees of atrioventricular block (gray arrows) are evident in these tracings; these are followed by a short sinus pause and then a sinus beat with a shorter PR interval, probably because of recovery of atrioventricular node conduction as a result of the longer P-P interval. In panel B, the first QRS complex is of sinus origin, followed by paired ventricular premature complexes with R-on-T phenomenon and then an episode of supraventricular tachycardia (7 complexes); this is followed by a short pause and 3 sinus arrhythmia complexes. Numbers on the top represent the R-R intervals in milliseconds. In both panels, paper speed = 25 mm/s; 1 cm = 1 mV.
digitalis toxicosis) should also be considered as differential diagnoses, especially when sinus bradycardia is detected. Most patients with extracardiac SSS do not require pacemaker placement as part of their treatment.

In most dogs with SSS, abnormalities of the atrioventricular node and subsidiary intraventricular pacemakers coexist, resulting in failure of appropriate escape rhythm generation. Abnormalities include a defect in sinoatrial node activity (eg, sinus bradycardia or sinoatrial block), atrioventricular node conduction disturbances (eg, first- or second-degree atioventricular block), and simultaneous supraventricular and ventricular excitability changes, all of which were observed in the dog of this report. Electrocardiographically, alternating recurrent paroxysms of supraventricular tachycardia and sinus bradycardia or sinus arrest characterize the tachycardia-bradycardia syndrome. In many portions of the Holter monitor tracings obtained from the dog of this report, supraventricular tachycardia and various degrees of atrioventricular block were more frequently identified than was the typical tachycardia-bradycardia combination associated with SSS. Another atypical finding was the appearance of short PR intervals with no PR segment, which could represent nonconducted P waves; it was also intriguing that the QRS complexes followed the same rhythm as the P waves. These QRS complexes may have been junctional beats in which the impulse was generated from the junction and traveled retrogradely and rapidly via internodal pathways to depolarize the atria via a nearly normal pathway, but it should also be considered that they were sinus beats with accelerated atioventricular conduction.

The dog of this report was initially taken to the attending veterinarian because of frequent syncopal episodes. Patients with SSS may have no clinical signs or may develop lethargy, syncope, collapse, or even seizures. Heart rate may be sufficiently slow or sufficiently fast to result in decreased cardiac output. Clinical signs are often related to hypoperfusion of vital organs and typically include syncope and lethargy.

Dogs with SSS that are clinically unaffected or that have minimal clinical signs do not require treatment. In general, pharmacological treatment (eg, atropine or digoxin) is not successful. The treatment for 1 type of arrhythmia may promote other arrhythmias. Presently, no long-acting pharmacological agents for treatment of bradyarrhythmias are available, and the drugs used (eg, atropine or isoproterenol) may cause undesirable adverse effects.

Dogs with clinical signs of SSS can be grouped into 2 subsets: those with predominating bradycardia, sinoatrial block, or sinus arrest and those with supraventricular tachycardia followed by sinoatrial block or sinus arrest. For dogs in the former subset, anticholinergic agents (eg, propantheline) may be used; if that treatment is ineffective, theophylline or terbutaline can be administered instead. For dogs in the latter subset, digoxin or atenolol may be used in an attempt to decrease tachycardia periods, both in the frequency with which they occur and the duration of each period. However, without prior pacemaker implantation, this treatment is associated with considerable risk because it can worsen the severity of bradyarrhythmias.

The treatment of choice for supraventricular tachycardia control is pacemaker implantation, followed by administration of antiarrhythmic agents. The placement of a pacemaker may reduce or eliminate up to 90% of clinical signs. The recommended antiarrhythmics are the same as those recommended for tachyarrhythmias that are not associated with SSS. β-Adrenergic receptor blockers may be an option, depending on whether signs of heart failure are present. Other options are administration of digoxin, diltiazem, or amiodarone. However, treatment without implantation of a pacemaker has a poor prognosis because response is most commonly inadequate.

For the dog of this report, the owner was a physician and was unwilling to pursue pacemaker implantation. Thus, treatment with digoxin was initiated at his request, and the dog has been free of clinical signs since administration of the drug was commenced. Although Holter monitoring could not be repeated, it appeared that digoxin reduced the dog’s tachycardia periods, thereby preventing sinoatrial node suppression by the faster heart rhythm and leading to resolution of the clinical signs.

References