A 2-year-old 24-kg (53-lb) spayed female American Staffordshire Terrier was referred to the Texas A&M Veterinary Medical Teaching Hospital for potential pacemaker placement. Six days prior, the dog began to vomit persistently and became lethargic, anorectic, and hypoplastic. Results of a CBC (performed at the referring veterinary clinic) were within reference ranges. An abbreviated serum biochemical profile performed at the referring clinic revealed high BUN (66.4 mg/dL; reference range, 9.0 to 29.0 mg/dL) and creatinine (1.8 mg/dL; reference range, 0.4 to 1.4 mg/dL) concentrations; serum electrolyte concentrations were not measured. The referring veterinarian had administered a dose of barium contrast agent orally prior to abdominal radiography, the results of which were unremarkable, and had treated the dog with IV fluid therapy, an antiemetic, and antimicrobials. The dog's condition did not improve, and it was persistently weak. When examined by the referring veterinarian on the day of referral, the dog was profoundly bradycardic with a heart rate of 30 beats/min.

On initial evaluation at the teaching hospital, the dog was obtunded and unable to stand. Body temperature was 35.9°C (96.6°F), heart rate was 30 beats/min, and respiratory rate was 20 breaths/min. Mucous membranes were pale and tacky, with a capillary refill time of 3 seconds. Patient-side serum electrolyte analysis indicated that the dog had hyperkalemia (6.17 mmol/L; reference range, 3.9 to 4.4 mmol/L); hypernatremia (122 mmol/L; reference range, 146 to 153 mmol/L); hypochloremia (101.6 mmol/L; reference range, 110 to 116 mmol/L); and hypokalemia (107 to 116 mmol/L; reference range, 9.0 to 29.0 mg/dL), and hypophosphatemia (76 mg/dL; reference range, 5 to 29 mg/dL), and persistent electrolyte abnormalities. Serum sodium and chloride concentrations were low at 116 mmol/L (reference range, 139 to 147 mmol/L) and 90 mmol/L (reference range, 110 to 116 mmol/L), respectively; potassium concentration was high (7.9 mmol/L; reference range, 3.3 to 4.6 mmol/L). On the basis of these findings, a blood sample was collected for determination of serum cortisol concentration; cosyntropin (10.4 µg/kg [4.7 µg/lb], IV) was administered, and a second blood sample was collected 60 minutes later for similar analysis.

Echocardiographic examination revealed that the left atrial and ventricular dimensions were within the reference ranges and there was no evidence of right-sided cardiac enlargement. Systolic myocardial function was within reference limits with a fractional shortening of 52.33%. Because of the dog's apparently normal cardiac structure and function, long-standing bradycardia was considered unlikely. Blood samples were collected to test for serum antibodies against Trypanosoma cruzi (to rule out Chagas disease) and determine circulating cardiac troponin I concentration. Two-view thoracic radiography revealed microcardia, but other findings were unremarkable. Results of biochemical analysis of a blood sample collected at the time of admission to the teaching hospital confirmed high BUN concentration (49 mg/dL; reference range, 5 to 29 mg/dL), hypocholesterolemia (76 mg/dL; reference range, 120 to 247 mg/dL), and persistent electrolyte abnormalities. Serum sodium and chloride concentrations were low at 116 mmol/L (reference range, 139 to 147 mmol/L) and 90 mmol/L (reference range, 107 to 116 mmol/L), respectively; potassium concentration was high (7.9 mmol/L; reference range, 3.3 to 4.6 mmol/L). On the basis of these findings, a blood sample was collected for determination of serum cortisol concentration; cosyntropin (10.4 µg/kg [4.7 µg/lb], IV) was administered, and a second blood sample was collected 60 minutes later for similar analysis.

This report was submitted by Audrey K. Cook, BVM&S; Sonya G. Gordon, DVM, DVSc; Randolph L. Winter, DVM; and Carly E. Waugh, DVM; from the Department of Small Animal Clinical Sciences, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, College Station, TX 77843.

Address correspondence to Dr. Cook (akcook@cvm.tamu.edu).

Figure 1—Lead II ECG trace from a 2-year-old spayed female American Staffordshire Terrier that was evaluated because of bradycardia and weakness. Notice that the P waves are regular but unrelated to the QRS complexes. Paper speed = 25 mm/s; 1 mV = 2 cm.
Pending results of that testing, the dog was treated for presumptive hypoadrenocorticism, with appropriate IV fluid support, dexamethasone (0.08 mg/kg [0.036 mg/lb], IV), and fluocortisone acetate (0.0125 mg/kg [0.006 mg/lb], PO). The following day, the dog was alert and more active. Serum concentrations of potassium and sodium were 5.2 and 127 mmol/L, respectively. Results of the ACTH stimulation test confirmed hypoadrenocorticism; serum cortisol concentration following ACTH stimulation was 1.4 µg/dL (reference range, > 7 µg/dL). Treatment with prednisone was started at a dosage of 0.4 mg/kg (0.18 mg/lb), PO, every 24 hours, and desoxycorticosterone pivalate was administered (2 mg/kg [0.9 mg/lb], IM, once).

An ECG examination was repeated 2 days following hospital admission and revealed a marked sinus arrhythmia with a wandering atrial pacemaker (Figure 3). The mean heart rate was 80 beats/min. The P-R interval duration was 120 milliseconds. Atropine was administered at a dose of 0.04 mg/kg, which increased the heart rate to 150 beats/min with a sinus rhythm and 1:1 conduction. The P-R interval duration was 120 milliseconds.

Results of the serologic testing indicated that the dog was negative for Chagas disease. Serum cardiac troponin I concentration was within reference range (0.032 ng/mL; reference range, 0.006 to 0.06 ng/mL). The dog was discharged from the hospital after 3 days, with a recommendation to the owner to return the dog in 3 to 4 weeks for 24-hour continuous ECG recording. The owner declined this suggestion, and the dog was not reexamined at the teaching hospital. The referring veterinarian continued management of the dog’s hypoadrenocorticism with administration of prednisone (0.1 mg/kg [0.045 mg/lb], PO, q 24 h) and desoxycorticosterone pivalate (1.7 mg/kg [0.77 mg/lb], IM, q 30 d). The dog was reported to be doing well 5 months following discharge from the hospital, with no detectable bradycardia and weakness.
Discussion

Third-degree atrioventricular heart block is a common cause of profound bradycardia in dogs. In many affected dogs, an underlying cause is not identified and the disorder is classified as idiopathic. Other causes of third-degree atrioventricular block include congenital defects, drug administration (e.g., digoxin), and organic cardiac disease. High vagal tone, which may be related to gastrointestinal, neurologic, or endocrine disorders, is also reported to cause third-degree atrioventricular block.

Hyperkalemia may develop as a consequence of bradycardia. The mechanism for this bradycardia-induced electrolyte abnormality is not well-defined. It has been suggested that compromised renal perfusion secondary to decreased cardiac output may have a role. In addition, dogs with long-standing bradycardia often undergo cardiac remodeling as a consequence of volume overload. Chamber distention or dilatation may trigger the release of natriuretic peptides with subsequent suppression of angiotensin II–induced secretion of aldosterone.

Hyperkalemia per se is a well-recognized cause of bradycardia, which is characterized by a progressive decrease in P-wave amplitude and prolongation of P-wave duration and ultimately culminates in atrial standstill and a sinoventricular rhythm. The QRS complexes may become progressively prolonged and attenuated in amplitude prior to asystole. T-wave changes include tenting and prolongation. In general, the P-wave changes represent the first detectable ECG abnormalities associated with hyperkalemia, but the presence and extent of ECG abnormalities are variable among patients and cannot be predicted on the basis of the severity of hyperkalemia. Despite substantial hyperkalemia (7.9 mmol/L as determined by the serum biochemical profile) at the referral evaluation, the dog of this report had P waves, but they were increased in duration and both P-wave and QRS complex amplitudes were relatively low. In addition, despite conducting with second-degree atrioventricular block following atropine administration, this dog did not have the ECG abnormalities expected in a dog with hyperkalemia. However, tremendous variability in the cardiac response to spontaneous hyperkalemia in dogs has been reported, and many patients maintain normal sinus rhythm despite marked increases in serum potassium concentrations. This is potentially a reflection of the complex electrolyte disorders in dogs with hyperkalemia, in which concurrent sodium, chloride, calcium, and acid-base disturbances are likely.

Hypoadrenocorticism has been previously reported in 2 dogs with third-degree atrioventricular heart block. In both cases, the heart block was persistent and did not resolve with management of the adrenal gland insufficiency. Because hypoadrenocorticism is likely due to an immune-mediated process, this may suggest a common cause for these conditions. Although the third-degree atrioventricular block in the dog of this report was reversible, the suboptimal response to anticholinergic administration suggested the presence of an intrinsic conduction defect.

The case described in this report illustrated both the importance of performing an atropine response test when evaluating a dog with third-degree atrioventricular heart block and the potential influence of noncardiac disease on electrical events within the heart. For this dog, the conversion to second-degree block suggested that high vagal tone contributed to the bradycardia and negated the immediate need for pacemaker placement.

References