An 8-year-old 36.1-kg (79.4-lb) spayed female Rottweiler was evaluated at the North Carolina State University Veterinary Teaching Hospital because of increasing frequency of episodes of collapse. On the basis of the owner's description, the collapse episodes were most consistent with syncope. Initially, the collapse occurred only with moderate exercise; over a period of 4 days, episodes occurred even during normal walking activity. The frequency of the collapse during the 4-day period increased from 1 episode/d to 3 to 5 episodes/d. The duration of each episode was <30 seconds; after a variable period of walking or running, the dog would appear disoriented, fall to the ground, lie still for <5 to 10 seconds, and then stand up panting. No urination or defecation was evident during these episodes. On the day prior to the initial evaluation at the hospital, the dog was taken to a local emergency clinic; results of a CBC and serum biochemical analyses were unremarkable. An ECG obtained at the emergency clinic revealed intermittent arrhythmia. The dog was referred to the cardiology department of the hospital for further evaluation.

On physical examination at the hospital, the dog was bright, alert, and responsive but panted continuously. The dog was obese and had dry skin and a dull coat. No murmur was identified via auscultation; however, intermittent arrhythmia was detected. Findings of thoracic radiography, abdominal ultrasonography, and echocardiography were unremarkable. A series of ECGs was obtained when the dog was resting and after a walk-induced episode of collapse (Figure 1). After walking a distance of approximately 100 feet, the dog became weak and collapsed and an ECG was immediately acquired. The combination of collapse and subsequent ECG evaluation was repeated 3 times with the same results.

Figure 1—Lead II ECG traces obtained at rest (A) and immediately after an episode of exercise-induced collapse (B; left-sided precordial lead) from a dog that was evaluated because of increasing frequency of collapse during exertion. At rest, normal sinus rhythm is present (heart rate, 100 beats/min) with apparently normal P-QRS-T amplitudes and intervals. After exercise-induced collapse, the initial rhythm appears to be third-degree atrioventricular (AV) block because of the series of nonconducted P waves and 2 idioventricular complexes (thin arrows). During the subsequent third-degree AV block, the atrial rate is approximately 250 beats/min and the ventricular rate is 50 beats/min. The rhythm spontaneously converts to 1 sinus beat; then to high-grade (4:1) second-degree AV block; and, subsequently, to lower grade (2:1) second-degree AV block (arrowhead). After a pause, sinus rhythm resumes (thick arrow). Paper speed = 25 mm/s; 1 cm = 1 mV.
EKG Interpretation

Evaluation of the initial EKG obtained while the dog was resting revealed sinus rhythm at a rate of 100 beats/min; the PR interval was 120 milliseconds (reference range, 60 to 130 milliseconds; Figure 1). The EKG trace obtained immediately after the collapse episode revealed a transient exercise-induced third-degree atrioventricular (AV) block that spontaneously reverted to second-degree AV block and then to sinus rhythm while the dog rested quietly. In all QRS complexes, J point (or ST segment) depression was noted. When in complete AV block, the atrial rate was approximately 250 beats/min and the idioventricular rhythm was 50 beats/min. During the transient third-degree AV block, prominent T waves were evident in the EKG trace; these T waves appeared as slight downward deflections of the trace immediately after the P waves and represented atrial repolarization. The third-degree AV block was terminated by a normally conducted sinus beat that was followed by a transient period of second-degree AV block. During the second-degree AV block, atrial-to-ventricular conduction progressively increased from 4:1 to 2:1; the AV block resolved with 1:1 atrial-to-ventricular conduction, leading to a short paroxysm of supraventricular tachycardia. The atrial rate slowed (abruptly) to approximately 80 beats/min, and a normal sinus rhythm was resumed. In an atropine response test, the dog was administered atropine (0.04 mg/kg [0.018 mg/lb]) SC once, and an EKG evaluation was repeated after 20 and 30 minutes. Atropine administration did not result in improvement of the AV block and extended the interval until reversion to normal sinus rhythm occurred. On the same day of admission, the dog was anesthetized and a permanent transvenous pacemaker was placed. A VVIR pulse generator (providing ventricular pacing and sensing with a variable rate response associated with activity) was used with the initial low rate set at 55 beats/min and initial high rate set at 135 beats/min. After surgery, the dog recovered uneventfully; continuous EKG monitoring did not detect runs of supraventricular tachycardia during the postoperative period. An EKG trace obtained 1 day after surgery revealed a mixture of appropriately paced and normal sinus beats (Figure 2). The dog was discharged the day after pacemaker implantation. Thyroid gland testing was performed approximately 1 month after pacemaker implantation because of the dog's physical appearance; however, findings were considered normal. At a recheck evaluation approximately 5 months later, the owner reported complete resolution of the collapse episodes. Results of pacemaker interrogation indicated that approximately 50% paced beats and 50% intrinsic sinus beats had occurred since implantation. During this examination, an EKG performed while the dog was resting revealed complete AV block with an atrial rate of 110 beats/min and an entirely paced rhythm at 60 beats/min.

Discussion

To our knowledge, paroxysmal exercise-induced third-degree AV block has not been previously reported in the veterinary medical literature. In humans, exercise-induced AV block that occurred either spontaneously or as a rare complication of an exercise-stress test has been reported. One proposed mechanism for this tachycardia-dependent AV block is acute ischemia of the conduction system as a result of the exercise-induced tachycardia. The myocardial ischemia during exercise is secondary to increased myocardial oxygen consumption and decreased duration of diastole, both of which result in decreased oxygen delivery. In humans, exercise-induced AV block typically indicates severe coronary artery disease, usually disease of the right coronary artery. The other proposed mechanism is severe infra-nodal conduction block from an abnormal conduction system, which has been identified via electrophysiologic testing in affected humans. In humans, when coronary artery disease is present, optimal management involves correction of the myocardial ischemia via angioplasty and medical treatments, but implantation of a permanent pacemaker is usually an inevitable necessity.

The underlying etiology of the exercise-induced AV block in the dog of this report was unknown. Coronary artery disease and myocardial ischemia are uncommon causes of heart disease in dogs. The J-point depression on the dog's EKG tracing was noteworthy. The J point is the point where the QRS complex joins the ST segment. Negative deflection of this point is indicative of subendocardial ischemia of the left ventricle. Hypothyroidism has been associated with coronary atherosclerosis in dogs. Because of the dog's dry skin, dull coat, and overweight body condition, thyroid gland testing was performed approximately 1 month after pacemaker implantation, but results were within reference limits. Regardless of the underlying mechanism, the finding of tachycardia-dependent, exercise-induced paroxysmal AV block may reflect severe His-Purkinje conduction disturbance, and pacemaker implantation should be considered for dogs with this arrhythmia. It is possible that exercise-induced AV block is an early manifestation of progressive conduction system disease. For the dog
of this report, the finding of complete AV block, even at rest, during the 5-month recheck examination suggested possible progressive disease. Nevertheless, following permanent cardiac pacing, the collapse episodes resolved completely in this patient.

References