A 10-year-old 17.2-kg (37.8-lb) spayed female Golden Retriever was evaluated at the College of Veterinary Medicine, North Carolina State University 16 months after undergoing splenectomy and chemotherapy for hemangiosarcoma; the evaluation was part of continued monitoring of the dog's condition. The chemotherapy protocol used to treat the dog included doxorubicin administered IV (cumulative dose, 160 mg/m\(^2\)) and cyclophosphamide administered orally over a period of 4 months. At the time of this ECG evaluation, the dog received enalapril because of mild renal insufficiency that had been detected several months earlier. Prior to treatment with the final dose of doxorubicin (approx 55 weeks earlier), the dog was administered dexrazoxane IV because echocardiographic findings at that time indicated diminished left ventricular systolic function, compared with findings of previous serial echocardiographic assessments (fractional shortening had decreased from 35.8% to 24.4%); mild mitral valve insufficiency attributable to mitral valve endocardiosis was also detected. Previous ECG findings included apparently normal sinus arrhythmia, and the mean electrical axis was within reference limits.

At the time of the 16-month posttreatment evaluation, the dog was bright, alert, and mentally appropriate. Physical examination revealed that the dog was in good body condition and was afebrile. Upon cardiac auscultation, a left apical systolic murmur (grade 2/6) was detected; heart rhythm was regular rhythm, and femoral pulse quality was considered normal. As part of the evaluation, Doppler systolic blood pressure measurement, a CBC, serum biochemical analyses, assessment of serum cardiac troponin I concentration, abdominal ultrasonography, and thoracic radiography were performed; echocardiography was repeated to assess left ventricular systolic function. Results of these tests indicated that the dog had azotemia (BUN concentration, 28 mg/dL [reference range, 6 to 26 mg/dL]; serum creatinine concentration, 2.1 mg/dL [reference range, 0.7 to 1.5 mg/dL]), high serum amylase concentration (1,690 U/L; reference range, 347 to 1,104 U/L), high serum cardiac troponin I concentration (0.83 ng/mL; reference range, < 0.2 ng/mL), and apparently normal systolic arterial blood pressure (145 mm Hg). Thoracic radiography and abdominal ultrasonography revealed no abnormalities. Echocardiography revealed a slightly thick mitral valve with mild mitral valve insufficiency (fractional shortening, 37%; reference range, 28% to 40%). Electrocardiography was also performed.

**ECG Interpretation**

Electrocardiography at the time of the 16-month posttreatment evaluation revealed a sinus rhythm (Figure 1). The heart rate varied from 125 to 167 beats/min (Figure 2). The rhythm was interpreted as sinus arrhythmia with intermittent sinus tachycardia. Sinus tachycardia could be consistently initiated with patient stimulation. The P-wave morphology and P-R interval (120 milliseconds) remained constant throughout. The QRS complexes in lead II during the salvos of sinus tachycardia were wide (80-millisecond duration) and had deep S waves in leads I, II, aVF, and V\(_3\), which were consistent with right bundle branch block (RBBB).

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**Figure 1**—Lead I (top), II (center), and V\(_3\) (bottom) ECG tracings obtained from a 10-year-old dog 16 months after it had undergone splenectomy and chemotherapy for the treatment of hemangiosarcoma; the evaluation was part of continued monitoring of the dog’s condition. Notice the baseline artifact (arrow) that occurred following patient stimulation and preceded the onset of sinus tachycardia, which was conducted with aberrancy. Paper speed = 25 mm/s; 1 cm = 1 mV.
The ECG tracings included normally conducted QRS complexes whenever the cycle length was > 400 milliseconds; this implied that the RBBB was tachycardia dependent and that the critical rate was 167 beats/min (360-millisecond cycle length). The diagnosis was tachycardia-dependent RBBB.

**Discussion**

Right bundle branch block occurs when there is conduction delay or block through the right bundle branch of the His-Purkinje system. Depolarization of the right ventricle relies on conduction from the left ventricle, which is transmitted through myocytes rather than through the specialized conduction system when there is a delay or block associated with RBBB. The delay in depolarization of the right ventricle results in a cranial and rightward axis deviation and prolongation of the QRS complex. The cranial and rightward axis deviation is attributable to unopposed, delayed right ventricular activation that occurs because there is no longer synchronous activation of the left and right ventricles. The prolongation of the QRS duration is a result of cell-to-cell conduction from the left to right ventricle, which is much slower than the His-Purkinje conduction rate.

The term aberrant ventricular conduction is usually reserved for transient bundle branch block. There are 3 potential mechanisms that may be responsible for aberrant ventricular conduction. The first is phase 3 aberration in which a stimulus occurs during phase 3 of the action potential. When a stimulus occurs during phase 3 of the action potential, the membrane potential at the time of stimulation is reduced (less negative) and conduction is compromised. Conduction velocity is dependent upon the resting membrane potential. Optimal conduction velocity relies on the rate of increase of phase 0 (ie, change in voltage with respect to time [expressed as $dV/dt$]) and the attained maximum voltage of phase 0 (expressed as $V_{\text{max}}$). There are a greater number of fast sodium channels available at more negative resting membrane potentials; this results in a greater influx of sodium into the cell during phase 0 of the action potential, which optimizes the $dV/dt$ value and $V_{\text{max}}$.

Functional or physiologic phase 3 aberration occurs in apparently normal fibers when the impulse is adequately premature such that it reaches the fiber during electrical systole of the preceding beat. Phase 3 aberration can occur if a premature stimulus reaches the right bundle branch during phase 3 of the action potential, when the membrane potential is $-65$ mV. At this time, only approximately half of the fast sodium channels are available for activation. Phase 3 aberration can be pathologic if electrical systole or the refractory period of the action potential is abnormally prolonged and if the fascicle involved is stimulated at a relatively rapid rate. Terms used to describe this pathologic type of phase 3 aberration include systolic block or tachycardia-dependent bundle branch block. \(^1,2\)

The second mechanism also involves phase 3 ventricular aberrancy in the form of retrograde concealed conduction. Retrograde concealed conduction occurs when an impulse only travels a limited distance within the specialized conduction system, resulting in an unexpected conduction delay or increase in cycle length in the following impulse. In humans, retrograde concealed conduction is a common mechanism for aberrancy. \(^1,2\) Right bundle branch block may occur when a ventricular premature beat retrogradely activates the right bundle branch late, thereby allowing the left bundle branch to recover for the next sinus beat while the right bundle branch remains refractory. The aberrancy is perpetuated because the timing of recovery of the distal portion of the right bundle branch allows retrograde conduction, which again leaves it refractory for the next sinus beat. Another ventricular premature beat is required to interrupt the aberrant ventricular conduction because the right bundle branch is refractory to retrograde conduction. The ventricular premature beat is typically followed by a compensatory pause, which allows return of normal conduction over both bundle branches with the following sinus beat. \(^1\)

Phase 4 aberrancy is the third mechanism that may be responsible for aberrant ventricular conduc-
Doxorubicin-associated cardiac toxic effects may develop during or several hours after IV administration of doxorubicin and includes cardiac ar-
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myocytolysis, and development of interstitial fibrosis and edema may all result from doxorubicin adminis-
tration with myofibrillar loss, cytoplasmic vacuolization, and lipid membrane peroxidation.

The mechanism is thought to involve free radical generation with a mean doxorubicin dose of 120 mg/m², but the effects may develop at doses as low as 90 mg/m². Persistent arrhythmias and conduction disturbances are associated with high (> 150 mg/m²) cumulative doses, and their occurrence does not necessarily correlate with advanced myocardial failure. Isolated RBBB is not hemodynamically important and does not require specific treatment.

References

5. Moise S. Right bundle branch block in a dog with sinus tachy-