A 6-month-old 19.8-kg (43.6-lb) sexually intact female German Shepherd Dog was referred to Michigan State University Veterinary Teaching Hospital for evaluation of slow pulses and unusual heart sounds that had been detected during a routine physical examination. On examination, the dog was bright, alert, and responsive. Respiratory rate was 24 breaths/min, and rectal temperature was 39°C (101.8°F). Mucous membranes were pink, and capillary refill time was < 2 seconds. Cardiac auscultation revealed no murmur; first and second heart sounds were considered normal and occurred at a rate of approximately 50 beats/min. Fluttering sounds were heard after the first and second heart sounds of most beats. Femoral pulses were strong and associated with the normal heart sounds, whereas no femoral pulses were palpable during the fluttering sounds. Electrocardiography was performed (Figure 1).

Transthoracic echocardiography revealed severe dilation of all 4 chambers of the heart as well as mild mitral valve and tricuspid valve regurgitation. Mildly decreased systolic function was evident as estimated by fractional shortening of 26% (M-mode reference range, 29% to 42%) and ejection fraction of 45% (Simpson’s 2-D reference range, > 52%), measured during periods of normal sinus beats. During periods of ventricular tachycardia (VT), there was no notable systolic activity and spectral Doppler evaluation of transaortic valve flow and transpulmonic valve flow revealed no substantial flow.

**ECG Interpretation**

The lead II ECG tracing (Figure 1) revealed sinus beats followed by nonsustained VT and ventricular premature contractions (VPCs). The ventricular arrhythmias (VAs) occurred 119 times during an approximately 2.5-minute period and followed every sinus beat (Figure 2). Although there were never 2 sinus beats in succession, the mean sinus beat-to-sinus beat coupling interval was approximately 56 beats/min. This corresponded with the heart rate and pulse rate determined during physical examination. Instantaneous heart rate during the paroxysms of VT ranged from 357 to 577 beats/min. The paroxysms of VT were not sustained and did not exceed 10 beats.

Several features of this case including signalment, lack of clinical signs, and ECG characteristics were consistent with a diagnosis of German Shepherd Dog–inherited VA (GSDIVA). However, because major echocardiographic changes in association with this disease are unusual, other causes of the dog’s VA were considered, including primary myocardial diseases such as myocarditis (eg, parvovirus or *Borrelia burgdorferi* infection) or dilated cardiomyopathy and secondary myocardial diseases as a result of systemic disease and electrolyte imbalances.3 For the dog of the present report, further diagnostic tests were recommended including a CBC, serum biochemical analysis, urinalysis, and 24-hour Holter monitoring; however, the owner declined further evaluation of the dog because it had no clinical signs. Despite the lack of clinical signs, the frequency, complexity, and high rate of the VT indicated that it was necessary to treat the dog in an attempt to reduce the risk of sudden cardiac death. Placement of an implantable cardioverter-defibrillator (ICD) was discussed with the owner but was declined. Consequently, treatment of the dog with antiarrhythmic drugs was undertaken.

Because the dog's indices of systolic function were diminished, mexiletine hydrochloride was the preferred treatment given its minimal effects on ventricular contractility.4 Unfortunately, mexiletine was commercially unavailable at the time that the dog required treatment; as an alternative, administration of sotalol hydrochloride (1 mg/kg [0.45 mg/lb], PO, q 12 h) was initiated. One month after beginning treatment with sotalol, there were no important changes in the ECG findings. At this time, mexiletine had become commercially available and administration of this drug (5 mg/kg [2.27 mg/lb], PO, q 8 h) was begun in addition to treatment with sotalol. A follow-up ECG examination performed 2 weeks later revealed no notable change in the arrhythmia. Again, placement of an ICD was recommended, but the procedure was declined by the owner. The dosage of mexiletine was increased to 6.25 mg/kg (2.84 mg/lb) every 8 hours; the dosage of sotalol was unchanged. At approximately 15 months of age and after treatment with antiarrhythmic agents for 9 months,
the dog died suddenly at home. Only 1 episode of collapse approximately 2 weeks prior to the dog's death was reported by the owner.

Discussion

The case described in this report illustrates the difficulty of identifying arrhythmias on the basis of results of auscultation alone. On examination of the dog, the referring veterinarian detected a low pulse rate and abnormal heart sounds and accurately concluded that these findings were not likely indicative of simple bradycardia. An ECG examination is essential for the identification of an arrhythmia and should be performed when an arrhythmia is suspected. In the dog of the present report, the slow pulses correlated with the sinus beats and the unusual heart sounds correlated with the VA. The marked pulse deficits were associated with the high-rate VT-simulated bradycardia.

Ventricular arrhythmias can be the result of abnormal electrical impulse generation (eg, abnormal automaticity or triggered activity), abnormal impulse conduction (eg, reentry), or a combination of both. In dogs with GSDIVA, triggered activity (spontaneous early afterdepolarization) in the Purkinje fibers of the left ventricle are thought to be the cause of the VA. In addition, abnormalities in the autonomic nervous system are thought to have important roles in the development of sudden death in dogs with GSDIVA. The risk of sudden cardiac death is thought to regress as dogs with GSDIVA age and their autonomic nervous system matures. Affected German Shepherd Dogs are vulnerable to VA and sudden death at approximately 3 to 12 months of age, with peak occurrence at 4 to 8 months of age. Prevalence of GSDIVA is currently unknown; however, it was initially identified in 4 families of German Shepherd Dogs from central and upstate New York, Illinois, and Michigan in 1987 through 1992.

In dogs with GSDIVA, laboratory data and echocardiographic findings are usually within reference ranges and prodromal signs for sudden death are usually absent. The disease can manifest in many arrhythmia phenotypes ranging from dogs that develop few VPCs to those that develop rapid polymorphic VT and sudden death associated with presence of frequent VT. Sudden death is thought to be a result of rapid (heart

Figure 2—Lead II ECG trace obtained during a 2.5-minute period from the dog in Figure 1. Notice the high frequency, complexity, and polymorphic nature of the ventricular arrhythmia. Paper speed = 25 mm/s; 0.5 cm = 1 mV.
rate > 300 beats/min) polymorphic VT that degenerates into ventricular fibrillation. 11 The rapid polymorphic form of VA develops more frequently during sinus bradycardia and sinus arrhythmia (e.g., during rapid eye movement sleep or rest after exercise and early in the morning). 11, 12 Less frequently, a slower monomorphic sustained VT (heart rate, 200 beats/min) that develops during sinus tachycardia can degenerate into ventricular fibrillation. 9

Until recently, knowledge regarding the treatment of dogs with GSDIVA was limited. No scientific studies evaluating treatment options were available, to our knowledge, at the time that the dog of the present report was evaluated. However, in 2010, Gelzer et al 13 reported that in dogs with GSDIVA, sotalol monotherapy had a proarrhythmic effect (most likely by exacerbating early afterdepolarization triggered activity), whereas a significant decrease in the total number of VPCs resulted from combination treatment with mexiletine (8 mg/kg [3.6 mg/lb], PO, q 8 h) and sotalol (2.5 mg/kg [1.1 mg/lb], PO, q 12 h). Although treatment with the mexiletine-sotalol combination was shown to reduce the number of VPCs, there was a lack of suppression of occurrences of VT. As such, this reduction in VPC frequency may not translate into a reduction in the risk of sudden cardiac death in dogs with GSDIVA because the frequency of VT appears to be a risk factor for sudden cardiac death.

Humans with VA who are at high risk for sudden cardiac death are typically managed with the placement of an ICD. Results of studies 14-17 have indicated the superiority of ICDs in reducing the risk of sudden cardiac death in people with VA, compared with results of antiarrhythmic agent administration. Implantable cardioverter-defibrillators can deliver defibrillation shocks or are able to deliver antitachycardic pacing that is capable of terminating VA. In veterinary medicine as a whole, ICD implantation is uncommon with only 2 reported cases: that of a Boxer with arrhythmogenic right ventricular cardiomyopathy 16 and a dog with GSDIVA. 17 In both of those dogs, it was reported that oversensing by the devices and high sinus heart rates resulted in inappropriate delivery of defibrillation shocks and with careful programming, these problems were minimized but not eliminated. 16, 17 Defibrillation thresholds were high in both dogs, and shocks in 1 dog were considered to be excessive by its owner who requested to have the defibrillation treatment turned off. 16 Antitachycardic pacing and low-energy cardioversion were continued in that 1 dog and appeared to be successful in terminating VT. In the dog with GSDIVA, 17 antitachycardic pacing was unsuccessful. In both dogs, the ICD was explanted as a result of infection.

Currently in veterinary medicine, administration of antiarrhythmic agents is still the most common treatment option for VA. Treatment strategies for VA involve decreasing the frequency of VA, improving clinical signs, and ultimately reducing the risk of sudden death. 18 Given that the number of VPCs and morphology of VA (shape of ventricular complexes) do not necessarily correlate to the risk of sudden death in veterinary species, multiple factors including severity of clinical signs, underlying cardiac disease, type of VA, and risk of adverse effects associated with surgical or medical management should be considered before selecting a treatment option.

References