ECG of the Month

The Academy of Veterinary Cardiology sponsors this feature. Readers of the *JAVMA* are invited to submit contributions. Contributions should include a brief description of the case (150 words); good quality contrast glossy photographs (5 X 7 in) of tracings, with the components of a QRS complex labeled; figure legends with information on ECG lead, paper speed, and voltage calibration; an ECG interpretation; and a discussion of the abnormality. Two hard copies of the manuscript and each figure must be submitted, along with an electronic copy on a 3.5-in PC-formatted disk. Submissions that are complete will be sent to the feature coordinator, Dr. Robert Hamlin, at The Ohio State University for review.

An 8-month-old sexually intact female Shih Tzu was referred to the Mississippi State University College of Veterinary Medicine for evaluation of a heart murmur that was detected by the referring veterinarian during a routine examination. There was no history of exercise intolerance, collapse, or coughing reported by the owner. The dog was receiving heartworm preventative monthly. At the evaluation, the dog was bright, alert, and responsive. On physical examination, a grade 5/6 holosystolic ejection-type murmur with palpable thrill was detected. The point of maximal intensity of the murmur was located at the left heart base (at the level of the third or fourth intercostal space). Mucous membranes were pink and moist, and the capillary refill time was < 2 seconds. Pulses were strong and synchronous with the heartbeat, and no other abnormal physical examination findings were identified. Diagnostic evaluation included a CBC, serum biochemical analyses, urinalysis, thoracic radiography, ECG, and echocardiography. Results of the CBC, serum biochemical profile, and urinalysis were within reference limits. Thoracic radiography revealed right ventricular enlargement and a bulge in the main pulmonary artery consistent with poststenotic dilatation.

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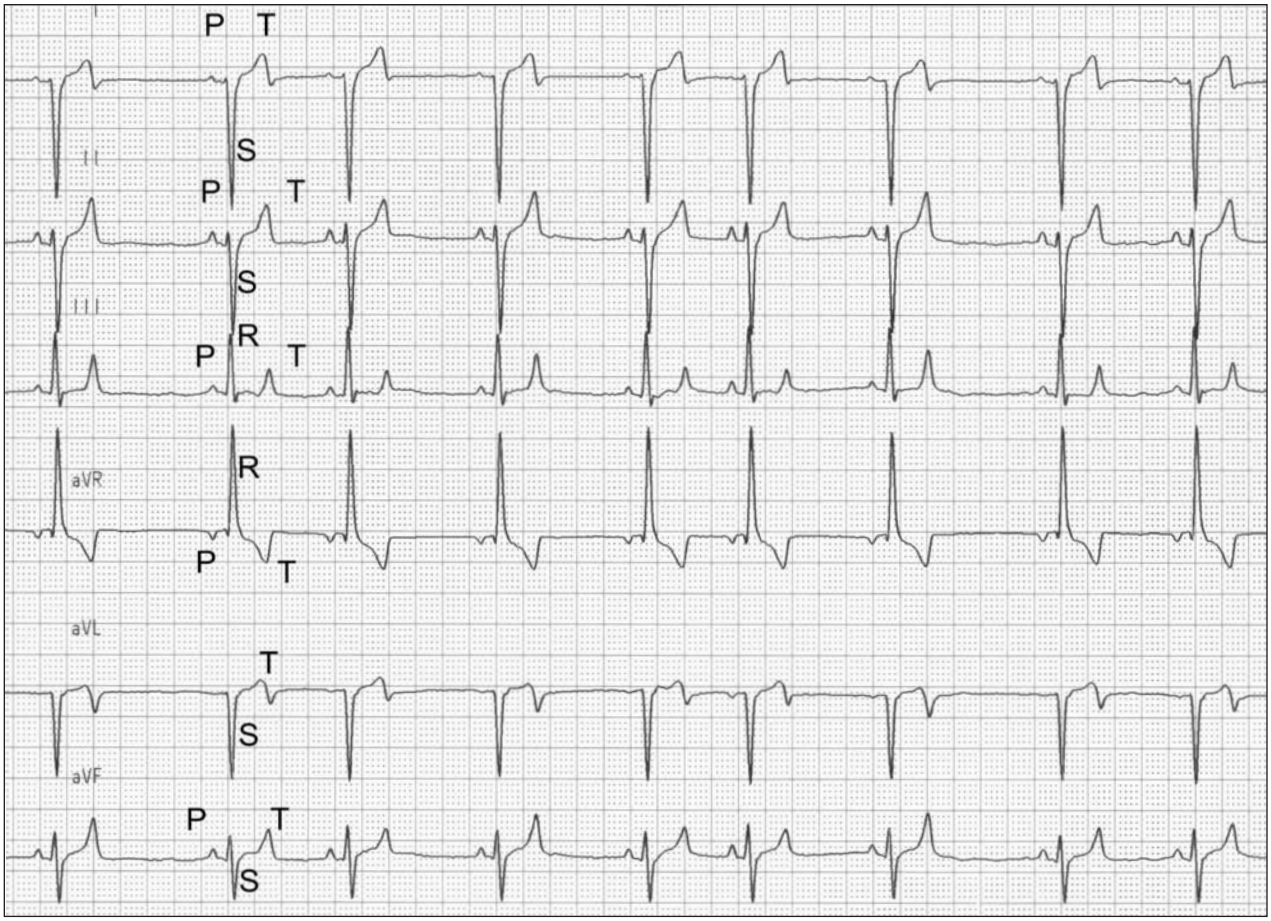

Figure 1—Six-lead ECG tracing obtained from an 8-month-old dog with congenital pulmonic stenosis at the time of initial evaluation. Notice the prominent S waves in leads I, II, and aVF that are suggestive of right ventricular enlargement. Paper speed 50 mm/s; 1 cm = 1 mV.

Echocardiography revealed right ventricular infundibular hyperplasia, tricuspid valve insufficiency, blood-flow turbulence in the main pulmonary artery detected via color-flow Doppler imaging, and increased velocity (15.31 m/s; reference limit, < 1 m/s) and pressure gradient (112.7 mm Hg; reference limit, < 4 mm Hg) across the pulmonic valve, as estimated by the modified Bernoulli equation (pressure gradient = 4 × velocity^2).[1] No structural abnormalities were identified in the pulmonic or tricuspid valve leaflets.

ECG Interpretation

Electrocardiography revealed a heart rate of 120 beats/min with a concurrent sinus arrhythmia (**Fig 1**). The mean electrical axis was estimated to be +180°, which suggested right axis deviation. Right ventricular hypertrophy was also evident with prominent S waves in leads I, II, and aVF and strongly positive deviations in leads III and aVR.[2]

An ECG plots the mean electrical potential generated by the heart muscle. The P waves represent the depolarization of the atria. The QRS complexes and T waves represent the depolarization and repolarization of the ventricles, respectively. The deep S waves in the ECG tracing (in leads I, II, and aVF) obtained from the dog of this report were indicative of delayed and unopposed depolarization of the right ventricular free wall, which is located in the cranial right border of the heart. In comparison, S waves associated with right bundle branch block are wide and bizarre in shape; the wide and bizarre conformation is a consequence of prolonged depolarization of the right ventricle that results from cell-to-cell depolarization rather than depolarization through the Purkinje network (eg, right bundle branch).[2]

Discussion

Pulmonic stenosis is a commonly reported congenital heart defect in dogs.[1] In a survey of medical records from North American veterinary schools from 1986 to 1990, pulmonic stenosis was the third most commonly diagnosed congenital heart defect in dogs (18%); the more commonly diagnosed congenital heart defects were subaortic stenosis (22%) and patent ductus arteriosis (32%).[3] Pulmonic stenosis can also be associated with tetralogy of Fallot (a combination of pulmonic stenosis, secondary right ventricular hypertrophy, subaortic ventricular septal defect, and an overriding aorta).[1] Pulmonic stenosis is overrepresented in Beagles, Samoyeds, Chihuahuas, English Bulldogs, Miniature Schnauzers, Labrador Retrievers, Bullmastiffs, Chow Chows, Newfoundlands, Bassett Hounds, West Highland White Terriers, and various spaniel breeds.[1] Both sexes may be affected, but males have been reported to predominate among dogs with pulmonic stenosis in the English Bulldog and Bullmastiff breeds.[4] The congenital defect in pulmonic stenosis is usually valvular in origin, resulting from pulmonic valve dysplasia. Subvalvular and supravalvular defects causing pulmonic stenosis have been reported infrequently. Subvalvular stenosis can be associated with a coronary artery anomaly, especially in English Bulldogs and Boxers.[1]

Pulmonic stenosis results in right ventricular outflow tract obstruction. The outflow obstruction leads to increased right ventricular systolic pressure and subsequent right ventricular concentric hypertrophy, which exaggerates the condition. The increased right ventricular systolic pressure results in tricuspid valve regurgitation and insufficiency. As the disease progresses, the hyperplasia and associated decreased compliance of the right ventricle can also result in right atrial dilatation. Guidelines for grading pulmonic stenosis are useful in determining prognosis. Pulmonic stenosis can be categorized as mild (pressure gradient, < 49 mm Hg), moderate (pressure gradient, 50 to 100 mm Hg), or severe (pressure gradient, > 100 mm Hg).[1] Some cardiologists consider pressure gradients > 80 mm Hg as severe. Animals in the severe categories are at increased risk for syncope, right-sided congestive heart failure, and sudden death.[1]

The decision to treat a dog with pulmonic stenosis is based on clinical signs, age, and other diagnostic findings. Medical treatment is not warranted unless right-sided heart failure develops. Intervention is then directed at reducing the right ventricular outflow tract obstruction and reducing the pressure gradient across the pulmonic valve. Treatment options include balloon dilation valvuloplasty and various surgical techniques. Treatment is not indicated in dogs with mild pulmonic stenosis (ie, pressure gradient < 49 mm Hg).[1,3,4]

After the diagnosis of pulmonic stenosis was made in the dog of this report, the prognosis and treatment options were discussed with the owner; a surgical patch-grafting procedure involving a pericardial patch was performed. Immediately after surgery (when the dog was still anesthetized), the pressure gradient across the pulmonic valve was estimated to be 10 mm Hg (by use of the modified Bernoulli equation) and pulmonic valve insufficiency was identified as a complication of the procedure. At an evaluation performed 1 month after surgery, the owner reported that the dog had no signs of right-sided heart failure and was more energetic than it was before surgery. A grade 4/6 holosystolic murmur with a palpable thrill was still evident. Echocardiography revealed right ventricular infundibular hyperplasia, pulmonic valve insufficiency, pulmonic stenosis (pressure gradient across the pulmonic valve was 30 mm Hg, compared with 113 mm Hg recorded at the initial examination), and no detectable tricuspid valve insufficiency.

References

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