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CASE REPORT

Long-term management of atrial myopathy in two dogs with single chamber permanent transvenous pacemakers

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Abstract Two young Labrador retriever dogs with bradycardia-induced syncope resulting from atrial myopathy underwent permanent transvenous pacemaker implantation. Both dogs developed heart failure 3—5 years after pacemaker implantation. Both were managed medically for approximately 7 years after pacemaker implantation and, ultimately, were humanely euthanized due to refractory heart failure signs and quality of life concerns. Long-term management of dogs with atrial myopathy and secondary atrial standstill with pacemaker implantation and medical therapy for heart failure is feasible and prognosis may be better than previously reported or speculated.

Abbreviations

CBC	complete blood count
ECG	electrocardiogram
FS	fractional shortening
LA/Ao	left atrial to aorta ratio
LVd	left ventricular diastolic
VVIR	ventricular pacing, ventricular sensing with inhibition, rate modulated pacing

Case 1

A 14-month-old, male neutered Labrador retriever, weighing 29.4 kg, was referred for evaluation of recent onset syncopal events and a soft systolic murmur. The dog had been evaluated by the primary care veterinarian five days prior to referral for episodes of collapse. Referral blood work included a chemistry profile, complete blood count (CBC), total thyroxine level, and urinalysis. All values were within reference limits. Initially the dog was referred to a board-certified veterinary neurologist. The neurologic examination was unremarkable and a cardiology consultation was recommended. On physical

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examination, the dog had a grade III/VI systolic murmur over the mitral valve, an irregular rhythm, fair femoral pulse quality, and normal lung sounds. Electrocardiography revealed no consistently definable P waves and a narrow-complex, presumably junctional, escape rhythm with a variable rate of 34–85 bpm. Echocardiographic examination revealed high normal left atrial dimensions (left atrial to aorta ratio [LA/Ao] of 1.41 obtained from right parasternal short axis M-mode evaluation; reference range 1.27 ± 0.20) [1,2]. No structural changes of the right atrium, right ventricle or left ventricle (left ventricular diastolic [LVd] dimension of 4.32 cm by M-mode of right parasternal short axis view; LVd reference range 3.42–4.98 cm) [2] were noted on two-dimensional or M-mode transthoracic echocardiography. Indices of systolic function were mildly elevated (fractional shortening [FS] 49%, reference range 27 ± 7.3%; ejection fraction [EF, Teicholz method] 81%, reference range 56.85 ± 10.2) [1]. Color flow Doppler mapping revealed mild centrally-directed mitral valve regurgitation. Spectral Doppler evaluation revealed normal trans-aortic and trans-pulmonic forward flow velocities and patterns. Medical management of the bradycardia with terbutaline (0.17 mg/kg PO q12 h) was attempted but unsuccessful. Twelve days after initial presentation, a single-chamber, bipolar, permanent, transvenous pacemaker[a] with VVIR (ventricular pacing, ventricular sensing with inhibition, rate-modulated pacing) pacing mode was implanted as definitive therapy for the bradycardia. Medical therapy with terbutaline was discontinued. The initial pacemaker programming was as follows: basal rate 80 ppm, maximal pacing rate 140 ppm, pulse width 0.5 ms and pulse amplitude 4.0 V with an impedance of 740 Ω.

The dog was followed for 7.5 years after pacemaker implantation. Six months after pacemaker implantation, the dog was started on enalapril (0.5 mg/kg PO q24 h) due to concern for underlying myopathy. The enalapril frequency was increased to q12 h ten months later due to LA dilation (LA/Ao 1.61). Four years after pacemaker implantation, severe LA dilation (LA/Ao 2.45), moderate right atrial dilation based on subjective analysis, and an increasing left ventricular diastolic (LVd 4.95 cm) with decreasing indices of systolic (FS 32%, EF 61%) was noted on echocardiographic examination and therapy with spironolactone (1.49 mg PO q12 h) was initiated. Six months later, the dog was deemed to be in impending congestive heart failure and echocardiographic findings included progressive cardiac dilation and high velocity transmitral flow (E velocity 1.95 m/sec, reference range 0.52–0.91 m/sec) [3]. Low-dose diuretic therapy with furosemide (1.2 mg/kg PO q24 h) was implemented. Five years after pacemaker implantation, echocardiographic examination revealed stable cardiac dimensions, but new, low volume, anechoic pleural effusion was noted and presumed to be a manifestation of right-sided heart failure; furosemide was increased (1.7 mg/kg PO q12 h). During this recheck evaluation, pacemaker interrogation revealed the generator had reached end of life and the pacemaker was functioning at a fixed rate of 50 bpm with the rate adaptive mode off. Three days later, the generator was successfully replaced. Six months later, the dog was in mild left-sided congestive heart failure based on thoracic radiograph evaluation and therapy with pimobendan (0.2 mg/kg PO q12 h) was initiated. Recurrent right-sided heart failure (ascites) was noted eleven months later and hydrochlorothiazide was added to the treatment plan. During the 3 years that the heart failure was managed, the medication doses were adjusted based on recheck physical and echocardiographic examinations, recheck blood work, and clinical signs at home. Seven years and 5 months after pacemaker implantation, the dog was humanely euthanized due to refractory right-sided congestive heart failure manifesting as ascites, as well as variable appetite and energy. At the time of euthanasia the dog's medications included hydrochlorothiazide (0.72 mg/kg PO q12 h), pimobendan (0.2 mg/kg PO q8 h), spironolactone (2.17 mg/kg PO q12 h), enalapril (0.29 mg/kg PO q12 h), and furosemide (2.32 mg/kg PO q8 h).

A necropsy was performed with samples submitted for histopathologic evaluation. Gross examination revealed marked dilation of all cardiac chambers. Low volume serosanguinous-appearing pleural effusion was noted (Fig. 1). The

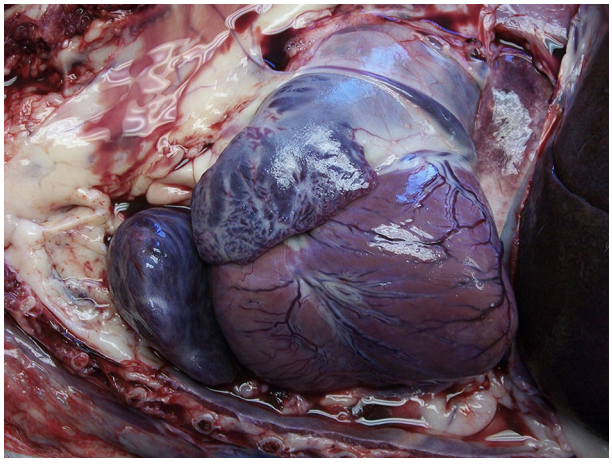

Fig. 1 Case 1: Heart in situ within thoracic cavity. There is marked cardiac dilation of all chambers. Small volume pleural effusion is present.

[a] Guidant Pulsar Max II, Model 1180, Serial 111054.