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CASE REPORT

Anodal stimulation in two dogs with transvenous permanent bipolar pacemakers

M.A. Oyama, DVM, MSCE*, M.J. Hezzell, VetMB, PhD , D.S. Laughlin, DVM , C.L. Thorn, DVM , M.S. Kraus, DVM , A.R. Gelzer, Dr. med. vet., PhD

Department of Clinical Studies-Philadelphia, School of Veterinary Medicine, University of Pennsylvania, 3900 Delancey St., Philadelphia, PA 19104, USA

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Abstract Management of symptomatic bradyarrhythmias such as complete atrioventricular block often involves permanent implantation of a transvenous pacemaker. Both during and after implantation, the operator can telemetrically assess and adjust a variety of electrical parameters associated with the pacemaker function in order to optimize the sensitivity, reliability, and power consumption of the device. Herein, we report an unexpected change in the paced electrocardiographic QRS complex morphology in two dogs undergoing bipolar pacing associated with changes in the pacemaker output amplitude settings first detected during threshold testing. The exclusivity of the electrocardiographic changes solely on pacemaker output settings, consistency between the surface electrocardiogram and ventricular endocardial electrogram, and resolution of this phenomenon when dogs were re-programmed to unipolar pacing is consistent with depolarization of the ventricular myocardium by the anodal electrode of the pacing lead at high pacemaker amplitudes. Anodal stimulation is a potential cause of varying QRS complex morphology witnessed during pacemaker evaluation and interrogation.

Two dogs that underwent permanent transvenous pacemaker implantation at the Ryan Veterinary Hospital of the University of Pennsylvania

* Corresponding author.
E-mail address: maoyama@upenn.edu (M.A. Oyama).

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were suspected of experiencing anodal stimulation during pacemaker programming. Dog 1 was a 7 year old, 26 kg, male castrated English Bulldog with a history of exercise intolerance, intermittent poor appetite, and ascites. Physical examination of dog 1 revealed a heart rate of 40 bpm with a regular rhythm and a distended abdomen. Dog 2 was a 7 year old, 45 kg, male castrated Shiloh Shepherd with a history of five syncopal episodes during the previous 48 h. Physical examination of the Dog 2 revealed a heart rate of 40 bpm with a regular rhythm, I/VI left-sided systolic murmur, intermittent isolated low intensity diastolic heart sounds, and occasional jugular vein pulses. Six-lead electrocardiograms (ECGs) were performed in both dogs and revealed complete atrioventricular block. In dog 1, there was a ventricular escape rhythm at 40 bpm and an atrial P wave rate of 136 bpm. In dog 2, there was a ventricular escape rhythm at 35 bpm and an atrial P wave rate of 150 bpm. Echocardiography was performed in both dogs and revealed mild dilation of the left ventricle (normalized left ventricular end-diastolic dimension: dog 1, 1.8; dog 2, 1.7; 5—95% values of the reference range, 1.4—1.7), but no evidence of clinically significant myocardial or valvular cardiac disease. During the echocardiogram small volumes of pericardial and pleural fluid were detected in dog 1. Along with the abdominal effusion that was previously detected on physical exam, the pericardial and pleural effusions in dog 1 were thought to represent congestive heart failure as a result of the bradycardia. Abdominocentesis was performed and yielded 1,380 mL of serosanguinous fluid. In dog 2, the intermittent diastolic heart sounds and jugular pulses noted on physical examination were assumed to be a S4 (atrial contraction) heart sound and a cannon 'a' wave resulting from atrial contraction against a closed atrioventricular valve, respectively. Placement of a permanent single-chamber transvenous ventricular paced, ventricular sensed, and inhibited (VVI) pacemaker was performed in both dogs during the subsequent 12 h. Pacemaker implantation was performed as previously described [1]. In both dogs, a 58-cm permanent endocardial pacing lead with active fixation[a] was introduced into the jugular vein and the helical lead tip was screwed into the endocardial surface of the right ventricular apex under fluoroscopic guidance. The opposite end of the lead was secured into a pacemaker generator[b] and successful capture of heart rhythm was confirmed by inspection of the surface ECG during the procedure. The pacemaker generators were secured in the subcutaneous tissues of the neck. Dogs were recovered and their ECGs were continuously monitored in the intensive care unit until discharge the following day. Pacing parameters at the time of implantation in dog 1 included the following: bipolar pacing and sensing; pulse amplitude, 2.75 V; pulse width, 0.40 ms; sensitivity, 2.0 V; lead impedance, 644 Ω; lower pacing rate, 90 bpm. Pacing parameters in dog 2 included the following: bipolar pacing and sensing; pulse amplitude, 3.30 V; pulse width, 0.34 ms; sensitivity, 2.0 V; lead impedance, 633 Ω, lower pacing rate, 75 bpm. Within 14 days after initial implant, both dogs underwent routine recheck and a ventricular strength duration threshold test was performed using a telemetric pacemaker interrogator.[c] The principles and practice of the strength duration threshold test have been previously described [2]. In brief, the test identifies safe and economic output settings by determining the lowest threshold at which the pacemaker discharge is able to generate cardiac depolarization. The test involves sequential reductions of the amplitude and pulse width of the pacemaker output until capture of the heart is no longer achieved. From these values, the pacemaker is programmed to an output setting with at least a 2× safety margin to ensure consistent capture of the heart, while avoiding unnecessarily high outputs that would deplete the battery life of the generator. In both dogs, the morphology of the paced QRS complexes on surface ECG was noted to suddenly change as the pacemaker amplitude was reduced by 0.25 V increments during threshold testing. This phenomenon was further explored by recording standard 6-lead ECGs from each dog at two different amplitude settings. Dogs were placed in right lateral recumbency and neither the position of the dogs nor placement of the limb electrodes on the dog's extremities was altered between the different recordings. In dog 1 (Fig. 1), the QRS complex morphology during a pacing rate of 90 bpm, pulse width of 0.4 ms, and pulse amplitude of 2.75 V resembled a left bundle branch block—like morphology. The QRS complex in lead II was described as Rs with a notch in the R wave. The net polarity of the QRS complex was positive in leads I, II, III, and aVF, and negative in aVL. The mean electrical axis in the frontal plane using the method of vectors based on leads I and aVF was 66°. When pacing amplitude was

[a] 5072, Medtronic, Minneapolis, MN, USA.

[b] Dog 1, Sensia SESR01; Dog 2, Adapta ADVDD01, Medtronic, Minneapolis, MN, USA.

[c] 2090, Medtronic, Minneapolis, MN, USA.

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