Late cardiac perforation by a passive-fixation permanent pacemaker lead in a dog

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**Case Description**—A 12-year-old miniature Dachshund with a history of permanent endocardial pacemaker implantation performed 7 weeks previously was admitted for routine dental prophylaxis.

**Clinical Findings**—Preanesthetic ECG revealed normal ventricular capture. Thoracic radiographic findings included caudomedial displacement of the endocardial pacemaker lead. Echocardiography revealed moderate chronic degenerative valve disease with moderate left atrial and ventricular dilation. After induction of anesthesia, loss of ventricular capture was detected. The dog recovered from anesthesia and had improved ventricular capture. The following day, surgical exposure of the cardiac apex revealed perforation of the right ventricular apex by the passive-fixation pacemaker lead.

**Treatment and Outcome**—A permanent epicardial pacemaker was implanted through a transxiphoid approach. Appropriate ventricular capture and sensing were achieved. The dog recovered without complications. Approximately 2 months later, the dog developed sudden respiratory distress at home and was euthanized.

**Clinical Relevance**—In dogs with permanent pacemakers and loss of ventricular capture, differential diagnoses should include cardiac perforation. If evidence of perforation of the pacemaker lead is found, replacement of the endocardial pacemaker lead with an epicardial pacemaker lead is warranted. (J Am Vet Med Assoc 2008;233:1291–1296)

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A 12-year-old spayed female 4.8-kg (10.6-lb) long-haired miniature Dachshund was brought to the referring veterinarian for routine preanesthetic evaluation prior to a dental prophylaxis. Bradycardia was detected on physical examination. Decreased activity level had been seen during the previous several months, but no other clinical signs had been noticed at that time. The dog was referred to a local cardiologist for further evaluation and subsequently referred to the Texas A&M University Veterinary Medical Teaching Hospital for evaluation as a candidate for pacemaker implantation. The dog was receiving no medications other than heartworm prevention. Physical examination revealed a bright, alert, and responsive dog. Thoracic auscultation revealed a heart rate of 66 beats/min but no auscultable cardiac murmurs. The respiratory rate was 26 breaths/min with normal bronchovesicular sounds bilaterally. An initial ECG revealed third-degree AV block with a monomorphic ventricular escape rhythm conducted with right bundle branch block morphology. Additional ECG traces revealed normal ventricular conduction. Thoracic radiographic findings included left-sided cardiomegaly (vertebral heart score = 11.6), pulmonary venous congestion, and radiographically normal lung parenchyma. Results of a CBC were within reference ranges, and a serum biochemical profile revealed no important abnormalities. Echocardiographic findings included moderate left ventricular and left atrial enlargement, thickened mitral valve leaflets, and predominantly diastolic mitral regurgitation.

Implantation of a permanent pacemaker was recommended. The following morning, the dog was given furosemide (1 mg/kg [0.45 mg/lb], SC) prior to surgery for an increased respiratory rate and suspected congestive heart failure. A temporary transvenous pacemaker lead was placed in the right ventricle through the left jugular vein and connected to a temporary pacemaker set to 100 beats/min. Anesthesia was then induced, and a permanent bipolar 58-cm tined lead was placed through the right jugular vein into the right ventricular apex. Following placement of the lead, it was secured to the jugular vein with 3 nonabsorbable sutures. The lead was then tunneled subcutaneously to the right lateral aspect of the abdomen. A second skin incision was made in the right lateral aspect of the abdomen, and a subcutaneous pocket was formed. The pacing mode was set for ventricular sensing and ventricular pacing with inhibition and was accomplished by use of a permanent pacemaker set to 80 beats/min, which was placed subcutaneously in the right lateral aspect of the abdomen. Confirmation of ventricular capture with the permanent pacemaker was achieved by decreasing the base rate of the temporary pulse generator to 60 beats/min. After surgery, the temporary pacing lead was removed. The dog recovered from anesthesia without complications. Thoracic radiography performed 1 day after the procedure revealed appropriate lead position.
in the right ventricular apex (Figure 1). A postoperative ECG revealed normal ventricular sensing and capture. Pain was managed with buprenorphine, the dog was discharged from the hospital, and clindamycin was prescribed in an attempt to prevent or minimize complications from severe periodontal disease.

Approximately 7 weeks later, the dog was returned for reevaluation of the pacemaker and a dental prophylaxis. An ECG revealed appropriate ventricular capture, but ventricular sensing was not assessed because of lack of inherent QRS complexes (Figure 2). Thoracic radiography results were suggestive of caudomedial displacement of the pacemaker lead, with apparent migration beyond the radiographic extent of the pericardial silhouette (Figure 1). Because of the verified normal pacemaker function, this was initially suspected to be an artifact of positioning during radiography. Echocardiographic findings included a substantially thickened mitral valve with moderate mitral valve prolapse, systolic and diastolic mitral regurgitation, and moderate left ventricular and left atrial dilatation. There was no tricuspid regurgitation; however, high-velocity pulmonic insufficiency was present and indicated pulmonary hypertension (maximum velocity, 2.9 m/s). The presence of pulmonary hypertension may have been secondary to increased left atrial pressure from chronic valve disease or subclinical pulmonary thromboembolism.

Later that day, the dog received general anesthesia for a dental prophylaxis. Immediately after induction, the

![Figure 1](image-url)
heart rate began declining and reached 35 to 40 beats/min. An ECG that was performed but not recorded revealed loss of ventricular capture, and anesthesia was terminated. The pulse amplitude was increased from 3.5 to 7.5 V, the pulse width was increased from 0.4 to 0.6 milliseconds, the base rate was increased to 100 beats/min, and the lead was changed from a bipolar setting to a unipolar setting. These programming changes regained ventricular capture on most impulses, but occasional loss of capture still occurred (Figure 3). Thoracic radiography was repeated, and with the exception of lead tip position, critical radiographic evaluation of the lead and generator revealed no abnormalities. There was no radiographic evidence of pleural effusion, and evaluation of a brief echocardiogram confirmed the absence of pericardial effusion. At that time, the decision was made to allow full recovery from anesthesia and monitor a continuous ECG overnight.

Overnight, continuous ECG revealed infrequent loss of ventricular capture. The following morning, pacemaker programmed variables were evaluated. Capture threshold was evaluated, and with decreasing pulse amplitude, loss of ventricular capture was noted at 3.5 V. The pulse amplitude was reset to 5.0 V, the pulse width was reset to 0.3 milliseconds, and the base rate was decreased to 80 beats/min. The decision was made to perform surgery for removal of the transvenous permanent pacemaker lead and placement of an epicardial lead. To reduce the risk of contamination during explant and transfer, the decision was made to also exchange the original permanent pacemaker for a new pacemaker. Prior to anesthesia, the pulse amplitude was increased to 7.0 V, the pulse width was increased to 0.6 milliseconds, and the base rate was increased to 100 beats/min.

Anesthesia was induced, and shortly thereafter, progressive loss of ventricular capture was detected. A transvenous temporary pacemaker lead was placed in the right ventricle through the left jugular vein, and the temporary pacemaker was set to 80 beats/min. A transxiphoid approach was made to view the apex of the heart. Electrosurgical dissection exposed the xiphoid, which was transected proximally and distally with bone-cutting forceps. Entry into the caudoventral portion of the thorax was accomplished without entry into the abdominal cavity. The mediastinum was removed sufficiently to expose the apex of the heart. The transvenous lead was clearly visible penetrating the heart and pericardium at the apical junction of the right ventricle to the ventricular septum (Figure 4). The

![Figure 4](image-url)
pericardium was opened, and the lead was freed from its hole in the pericardium. Three pericardial stay sutures were placed to control the apical portion of the heart. A purse-string suture was placed in the right ventricle around the lead. Two other sutures that included a piece of pericardium were placed dorsolateral and ventral to the perforating lead. The tined end of the lead was gently pulled and snipped with wire-cutting forceps. As the severed lead retracted into the ventricle, the purse-string was closed, and the 2 sutures with pericardium were tied. There was no observable hemorrhage from the site.

A screw-in epicardial pacemaker lead was attached just ventral to the left ventricular apex. The pericardium was loosely closed over the lead. A new permanent pacemaker was set to pace ventricular sensing and ventricular pacing with inhibition at 100 beats/min. A blind pocket was developed with digital manipulation between the peritoneum on the midline and the rectus abdominus muscle. The pacemaker was placed in the retroperitoneal pocket. Redundant pacing lead was loosely coiled in the pocket. The thoracic cavity was closed with interrupted sutures. Subcuticular sutures were placed to close the skin. Intraoperative ECG monitoring confirmed ventricular capture by the epicardial lead.

An incision was made over the original permanent pacemaker, located subcutaneously over the right lateral portion of the abdomen. The pacemaker and attached endocardial lead were severed with wire cutters from the remaining lead under the skin and removed. Then an incision was made over the right jugular vein to remove the remainder of the transvenous pacemaker lead still located under the skin and within the right jugular vein. Removal of the original pacemaker and endocardial lead was performed without complication. After surgery, the temporary pacing lead was removed.

The dog recovered from anesthesia without complications. The following morning, an ECG revealed an intermittent idioventricular rhythm conducted with a similar morphology to the paced beats, suggesting an origin near the pacemaker lead insertion into the left ventricle. When the base rate was increased, normal ventricular capture was evident. Thoracic radiography revealed the lead position on the apex of the left ventricle (Figure 5). After surgery, pain was managed in-hospital with buprenorphine. The dog was discharged; clindamycin was prescribed to attempt to prevent or minimize bacteremia from severe periodontal disease, and tramadol was prescribed for pain management.

Persistent intermittent coughing was evident after the dog was discharged. No other clinical signs were reported. Approximately 2 months later, the dog developed sudden respiratory distress, possibly caused by congestive heart failure secondary to moderate chronic degenerative valve disease. Pulmonary thromboembolism or primary lung disease was also possible. Thoracic radiography performed by the referring veterinarian revealed a severe bronchial pattern without evidence of pulmonary venous congestion. Respiration did not improve with a single dose of furosemide (2 mg/kg [0.91 mg/lb], IV), which, in light of the radiographic findings, made congestive heart failure less likely. The owner decided to take the dog home, and within several hours, the dog was cyanotic, dyspneic, and minimally responsive. The owner chose to have the dog euthanized at that time.

Discussion

When intermittent loss of capture is detected, differential diagnoses should include poor lead position or a dislodged lead (and, less commonly, lead perforation), lead fracture, poor connection between the lead and the generator, insulation breakage, and battery failure.3 Many of these differential diagnoses can be ruled
out by evaluation of lead impedance, but this variable was not available for the dog in this report. However, all other pacemaker variables were within reference ranges. The displacement of the pacemaker lead detected via thoracic radiography made cardiac perforation the likely diagnosis.

In the human literature, cardiac perforation by a permanent pacemaker lead was first described as a rare complication of pacemaker implantation in the 1960s.\footnote{8-10} The frequency of acute perforation was reported to be as high as 5% to 7%.\footnote{4} However, contemporary pacing leads are smaller and more flexible, and the frequency of acute perforation has decreased to < 1%.\footnote{8-10} Late cardiac perforation, especially by a tined, passive fixation lead, is even rarer.\footnote{7,11-13} Human patients with cardiac perforation may remain asymptomatic; however, complications associated with lead perforation include chest pain, diaphragmatic stimulation, pericardial effusion and cardiac tamponade, pericarditis, pneumothorax, hememothorax, and sudden death.\footnote{5,14-16} Another important complication with cardiac perforation is not bleeding into the pericardium or pleural cavity, but pacing failure.\footnote{7,17,18} Cardiac perforation by a permanent pacemaker lead, both acute and late onset, has not been reported in dogs.\footnote{19,20}

There are several theories on the cause of cardiac perforation. The right ventricular free wall is thin, reportedly as little as 4 to 5 mm in humans,\footnote{4} and was approximately 3 mm in this dog. However, right ventricular wall thickness is likely not the only reason. Active fixation, screw-in leads tend to be more prone to perforation than passive fixation, tined leads.\footnote{11} Acute perforation is suspected to occur secondary to maneuvering of pacing leads during the implantation procedure, use of small electrode tips, or by positioning the lead with the guidewire still in place.\footnote{7,9,16,21} Excessive lead length resulting in too much slack has been implicated in creating tremendous tension on the lead and causing perforation.\footnote{8} Newer low-profile active fixation leads have also been implicated, potentially because of decreased diameter and increased force per unit area.\footnote{14} Despite many theories, only 1 study identifying risk factors for perforation has been reported. Independent predictors of perforation identified were use of a temporary pacemaker; corticosteroid use within 7 days prior to pacemaker implantation; and use of an active fixation, screw-in lead.\footnote{19} Mahapatra et al\footnote{22} theorized that the use of a temporary pacemaker increased the risk of perforation because of the increased number of leads in the right ventricle. Corticosteroids are associated with delayed wound healing and skeletal muscle weakness and atrophy, which suggests possible explanations for the use of corticosteroids as a predictor for cardiac perforation.\footnote{22} The dog described in this report had not received corticosteroids orally and did not have hyperadrenocorticism, but did have a temporary pacemaker placed prior to both procedures. In addition, pacemaker leads often have a steroid-eluting tip, which is designed for human use with the goal of preventing excess scar tissue at the site of lead insertion and subsequent loss of capture. In this case, the lead contained 1 mg of dexamethasone sodium phosphate, corresponding to a dose of 0.2 mg/kg (0.091 mg/lb). This could have created delayed incorporation of the lead tip into the myocardium, allowing the lead to migrate freely in the right ventricle, as well as potentially weakening the right ventricular free wall in that location.

Diagnosis of cardiac perforation can present a challenge. An increased capture threshold can be suggestive of perforation or dislodgement of the pacing lead.\footnote{21} Radiographically, the lead may be dramatically displaced and even visible outside of the cardiac silhouette. Other specialized radiographic methods, such as computed tomography scan and right ventriculograms, have been used to diagnose perforation.\footnote{10,16} Alternatively, the lead may appear to be unchanged in position. Echocardiographically, the lead is difficult to visualize in a single plane and may appear to still be within the right ventricle. On surface ECGs, a number of changes may occur; however, none are diagnostic for cardiac perforation. Ventricular pacing can be unchanged, intermittent, or absent depending on the degree of contact between the myocardium and tip of the pacing lead.\footnote{23} Results of a human study\footnote{22} indicate that a right bundle branch block in the paced beats is suggestive of perforation; however, many other situations can result in this pattern. Intracardiac ECG has been used in humans to diagnose perforation.\footnote{21}

A thoracotomy is not often necessary in human cases of cardiac perforation. If there is cardiac tamponade, pericardiocentesis should be performed.\footnote{10,11,13,21} If the lead tip is in the mediastinum and there is no bleeding, a new lead can be placed without extracting the original perforating lead. Alternatively, in some cases, the lead can be pulled back, ideally with guidance by transesophageal echocardiography, and extracted or repositioned.\footnote{8,14} However, if there is late perforation, the lead may be adhered to extracardiac structures, and removal could be dangerous.\footnote{8} In particular, tined leads have bulky tips and would likely damage tissues during removal. Active fixation tips are less bulky and cause less trauma during withdrawal.\footnote{8} In this case, a transxiphoid window was created to enable viewing of the cardiac apex and facilitate placement of an epicardial pacemaker. As such, the perforating lead tip was directly visible outside of the pericardium, and surgical removal was possible. When the lead tip was cut off, the risk of bleeding or tissue damage during withdrawal was minimized, and the remaining lead was safely removed.

References


