Journal of Veterinary Cardiology (2019) **22**, 113—120

Journal of Veterinary Cardiology
ESVC

www.elsevier.com/locate/jvc

Case Report

Identification and management of a subacute right ventricular perforation by an active-fixation permanent pacemaker lead in a dog☆,☆☆

L. García-Guasch, DVM, PhD*, C. Sá Borges, DVM, MS , P. Sousa, DVM , J. Manubens Grau, DVM

Cardiology Service, Hospital Veterinari Molins, Pol. Ind. Molí Dels Frares, B-27, 08620 Sant Vicens Dels Horts, Barcelona, Spain

Received 23 April 2018; received in revised form 13 November 2018; accepted 14 November 2018

KEYWORDS
Third-degree atrioventricular block;
Pacing;
Complication

Abstract A 5-year-old short-haired dachshund was referred with a history of repeated syncope associated with a third-degree atrioventricular block. A permanent transvenous pacemaker with an active-fixation lead was implanted. In the following 3 weeks, the syncopal episodes reappeared owing to a loss of ventricular capture. The pacemaker was reprogrammed to higher output, and effective pacing was reestablished. Thoracic radiographs and echocardiography failed to identify any evidence of lead displacement. One month later, the patient presented a new episode of loss of capture. After fluoroscopy, cardiac perforation was suspected and subsequently confirmed by thoracotomy. An epicardial pacemaker lead was implanted

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* Corresponding author.
E-mail address: laingarcia@gmail.com (L. García-Guasch).

https://doi.org/10.1016/j.jvc.2018.11.006

without removing the perforating lead as there were no bleeding complications or damage to adjacent organs, and the length of time elapsed since implantation was assumed to have allowed for significant fibrotic adhesions to develop. Nineteen months after epicardial pacemaker implantation, endocardial lead dislodgement occurred. Simultaneously, the dog presented with gastrointestinal and respiratory abnormalities and severe thrombocytopenia. Once the dog was stabilized, the endocardial lead was percutaneously removed. One month later, loss of ventricular capture recurred. The owners declined any further treatment, and euthanasia was elected. Cardiac perforation after pacemaker implantation is an infrequent complication. In this case, the dog lived 22 months after subacute right ventricular perforation. Despite the poor prognosis associated with cardiac perforation by pacemaker leads, different approaches are possible to successfully manage this major complication. Extraction of the displaced lead remains controversial as, if the lead is not removed, late lead migration can occur.

Abbreviations

AV	atrioventricular
RV	right ventricle
VVI	ventricular sensing and ventricular pacing with inhibition

A 5-year-old neutered, male short-haired dachshund weighing 10 kg was referred to the cardiology service of the Hospital Veterinari Molins for further evaluation of bradycardia. A decreased activity level and repeated syncope had been observed during the preceding weeks. On presentation, the dog was bright and responsive. Physical examination revealed no abnormalities apart from bradycardia at 60 beats per minute. The ECG diagnosis was third-degree atrioventricular (AV) block with a ventricular escape rhythm of 60 beats per minute. Thoracic radiographs, echocardiography, and basic hematology were unremarkable. Serology tests for *Dirofilaria immitis, Anaplasma phagocytophilum, Anaplasma platys, Borrelia burgdorferi, Ehrlichia canis,* and *Ehrlichia ewingii* were negative.

Transvenous implantation of a permanent pacemaker was recommended, and owner consent was obtained. Anesthesia was induced, and the dog was positioned in the right lateral recumbency. The ventricular escape rhythm remained stable after induction, and temporary pacing was not required. A unipolar, steroid-eluting endocardial lead with active fixation[a] (60-cm long) was inserted through a small incision into the left jugular vein and advanced under fluoroscopy guidance to the apex of the right ventricle (RV). Although access via the right jugular vein usually is the preferred approach, the left jugular vein was used following the institutional protocol. The pacing lead was sutured to the left jugular vein using an anchoring sleeve. Subsequently, the proximal tip of the lead was tunneled subcutaneously to the left lateral aspect of the abdomen and connected to the pulse generator[b], which was then secured in an abdominal subcutaneous pocket. Subcutaneous tissues over the pulse generator and skin incisions were closed routinely. The pacing mode was set for ventricular sensing and ventricular pacing with inhibition (VVI). Pacemaker programming data at the time of pacemaker implantation are detailed in Table 1. Postsurgical thoracic radiographs confirmed an appropriate lead position, and an ECG confirmed appropriate ventricular sensing and capture. The neck was bandaged for 10 days, and the patient was discharged 3 days after surgery, with ampicillin at 22 mg/kg, orally, twice daily for 10 days.

Two weeks later, the dog experienced another syncope. An ECG revealed loss of ventricular capture and third-degree AV block with a ventricular escape rhythm of 40 beats per minute (Fig. 1). Pacemaker interrogation indicated that lead impedance was within normal limits (Table 1, Recheck-1). Thoracic radiographs and echocardiography failed to identify any signs of lead displacement. The pacemaker was reprogrammed to produce higher output, and effective pacing was re-established. One week later, owing to a recurring failure in pacing, pulse amplitude and

[a] Sorin Tilda R60, Biotronik SE & Co., Berlin, Germany.

[b] Esprit SR, Sorin Group, Saluggia, Italy.

Table 1 Pacemaker programming data obtained at the time of pacemaker implantation and revisions.

Telemetry data	Implantation	Recheck-1	Recheck-2	Recheck-3	Recheck-4	Recheck-5
Pacemaker **Lead**	Esprit SR Sorin Group Tilda R60	Esprit SR Sorin Group Tilda R60	Esprit SR Sorin Group Tilda R60	Reply VDR Medtronic Inc. 5071-53 cm	Reply VDR Medtronic Inc. 5071-53 cm	Reply VDR Medtronic Inc. 5071-53 cm
Battery						
Magnet rate (min^{-1})	96	96	96	96	96	96
Impedance (kΩ)	0.39	0.15	0.20	0.15	0.19	0.29
Longevity	N/A	3 years 5 months	2 years 9 months	N/A	6 years 8 months	6 years 1 months
Basic parameters						
Mode	VVI	VVI	VVI	VVI	VVIR	VVIR
Basic rate (min^{-1})	80	80	80	80	80	80
Maximum tracking rate (min^{-1})	130	130	130	130	130	130
Pacing/sensing						
Pulse amplitude (V)	3.5	5.0	7.5	3.5	3.5	3.5
Pulse duration (ms)	0.35	1.00	0.75	0.50	0.50	0.50
Sensitivity (mV)	2.5	2.5	2.5	2.5	2.5	2.5
Sensing polarity	Unipolar	Bipolar	Bipolar	Unipolar	Unipolar	Unipolar
Pacing polarity	Unipolar	Unipolar	Unipolar	Unipolar	Unipolar	Unipolar
Special features						
Smoothing	Off	Off	Off	Off	Off	Off
Ventricular autothreshold	Off	Off	Off	Off	Off	Off
Rate responsive	Off	Off	Off	Off	Auto	Auto
Tests						
Impedance (Ω)	N/A	456	448	531	654	718
Voltage threshold (V)	N/A	N/A	N/A	0.50	1.25	1.00

kΩ: kiloohm; min: minute; ms: millisecond; mV: millivolt; N/A: not available; V: volt; VVI: ventricular sensing and ventricular pacing with inhibition; Ω: ohm.

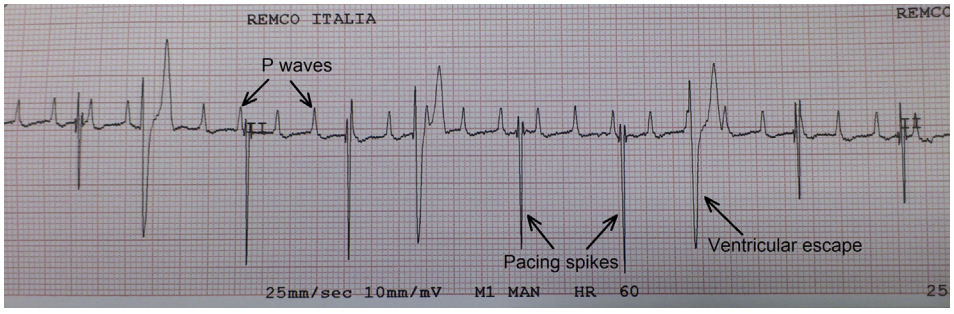

Fig. 1 Electrocardiogram showing a loss of capture of the transvenous pacing system. The pacing spikes are not followed by electrical capture, and ECG reveals a third-degree atrioventricular block with a ventricular escape rhythm.

duration was increased (Table 1, Recheck-2). These programming changes re-established ventricular capture. Correct positioning of the lead was again confirmed via thoracic radiographs and echocardiography.

Two weeks later, the dog presented with recurrence of syncopal episodes, and an ECG recording was suggestive of loss of capture. Both thoracic radiographs and fluoroscopy indicated a possible migration of the pacemaker lead tip through the apex of the RV (Supplemental Video 1; available online). No pericardial or pleural effusion was present. Exploratory thoracotomy was elected to assess the transvenous lead positioning and to implant an epicardial lead. Anesthesia was induced, and a left lateral thoracotomy was performed. The tip of the perforating lead was abnormally located 1 cm outside the RV's

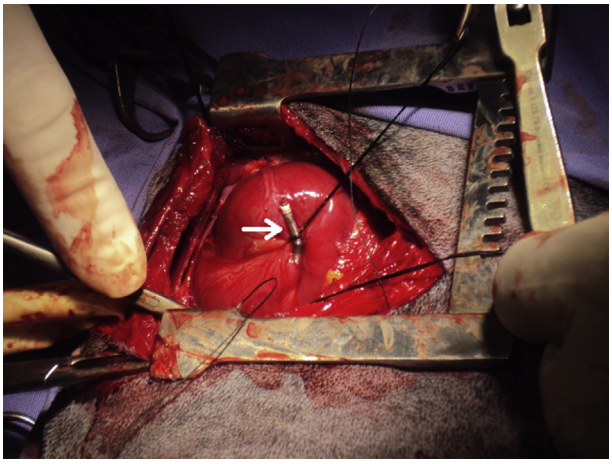

Fig. 2 Intraoperative photograph of the heart. The complete penetration of the right ventricle and pericardium by the active-fixation lead (arrow). Top of the image is cranial, and left of the image is ventral.

epicardial surface (Fig. 2). Therefore, a purse-string suture was placed on the surface of the RV around the original lead, and a piece of the pericardium was sutured over the lead tip to the epi/myocardium, and the remaining pericardium was excised. The pulse generator[b] was removed, and the endovascular lead[a] was cut near the point of entry into the jugular vein, sutured in the region of the neck and sealed with a blind cap[c] and medical adhesive[d]. Subsequently, a sutureless, unipolar myocardial screw-in epicardial pacing lead[e] was attached to the ventral aspect of the left ventricular apex. The pericardium was closed over the lead, and the proximal tip of the lead was connected to a new pulse generator[f] that was placed in an abdominal pocket (Fig. 3A and B). The pacemaker programming data (Recheck-3) are listed in Table 1.

Two months later, the generator was programmed to a rate responsive mode with a maximal rate of 130 beats per minute (Table 1, Recheck-4). Normal pacemaker function was confirmed at rechecks 3 and 6 months after implantation.

Twenty-one months after the initial visit, the dog was presented with a 3-day history of vomiting, melena, tachypnea, and weakness. On physical examination, the mucous membranes were pale. Cardiac auscultation, ECG, and telemetry (Table 1, Recheck-5) revealed normal pacemaker function, with a constant pacing rate of 80 beats per minute. Hematology revealed severe thrombocytopenia (9000/μL; normal values 186,000—545,000/μL) and anemia (hematocrit: 15%, normal values 38%—53%). Thoracic radiographs showed a dislodgement of the endocardial lead into the RV chamber (Fig. 4A and B). No pericardial or pleural effusion was present. The agglutination test and Coombs test were positive, and an immune-mediated thrombocytopenia and anemia were suspected. The patient was hospitalized to receive fluid therapy, oxygen supplementation, and methylprednisolone at 1 mg/kg, intravenously, twice daily. A fresh whole-blood transfusion was administered. The initial rate started with 2 mL over the first 5 min to monitor for a potential transfusion reaction; the transfusion rate was subsequently increased to 3 mL/kg/hr and was administered within 4 h. Three days later, complete blood analysis revealed no abnormalities; both the platelet and the hematocrit levels were within the normal range (platelets: 304,000/μL and hematocrit: 32%). At that point, the endocardial RV lead was extracted by simple traction under general anesthesia. Removal of the lead was performed without complications, and the dog was discharged 2 days later.

One month later, the dog presented with a 12-h history of repeated syncope. An ECG revealed recurrence of the third-degree AV block with a ventricular escape rhythm of 40 beats per minute associated with pacemaker loss of ventricular capture. The owner declined any further treatment, and euthanasia was elected. A postmortem examination revealed a large area of myocardial necrosis and chronic inflammation at the site of epicardial lead fixation (Supplemental Figs. I and II; available online). The RV endocardial lead insertion/perforation site was unfortunately not evaluated histologically because macroscopic findings of this area only included an old healed lesion.

Discussion

Pacemaker implantation in a dog with a third-degree AV block was first described in 1967 [1]. The most common major complications of this procedure include lead dislodgement or malpositioning, causing loss of capture, generator failure, lead damage, twiddler's syndrome, generator migration, redundant lead loops leading to dislodgement, cardiac arrest during implantation, lead infection, lead thrombus, and myocardial perforation [2]. Minor complications

[c] BK-IS, Biotronik SE & Co., Berlin, Germany.
[d] Medical adhesive SI-8, Biotronik SE & Co., Berlin, Germany.
[e] 5071-53 cm, Medtronic Inc., Minneapolis, USA.
[f] Reply VDR, Sorin Group, Saluggia, Italy.

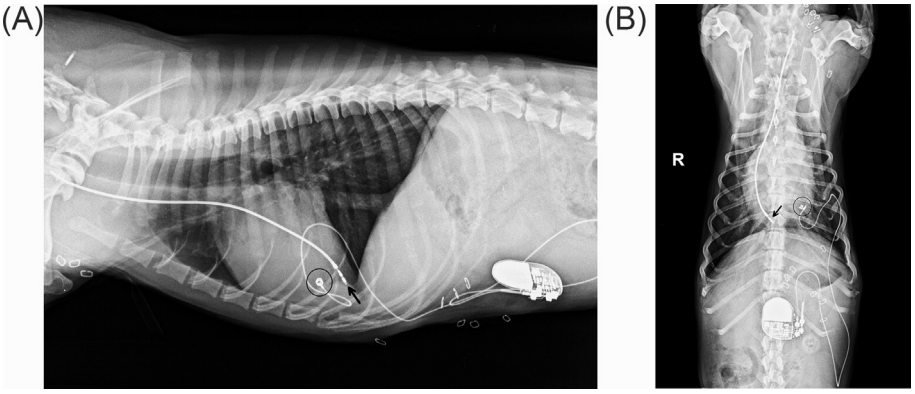

Fig. 3 Right lateral (A) and dorsoventral (B) thoracic radiographs obtained after the epicardial pacemaker implantation. The epicardial pacing lead is screwed into the left ventricle apex (black circle). The endocardial lead has not been removed (arrow).

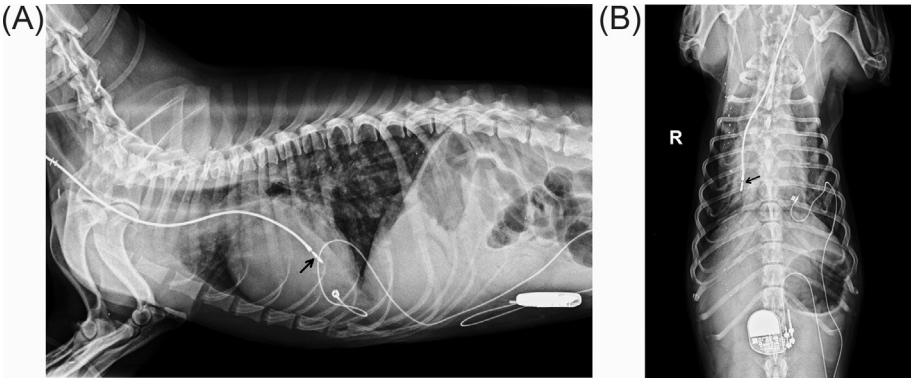

Fig. 4 Right lateral (A) and dorsoventral (B) thoracic radiographs showing the endocardial pacemaker lead dislodgement. The tip of the endocardial lead has moved from the right ventricle apex (arrow).

include seroma formation, muscle twitch, and arrhythmias [2—4].

Cardiac perforation after pacemaker implantation is an infrequent complication [5]. The incidence of cardiac perforation in people is approximately 1.2% and is less likely with passive-fixation leads (0.1%—0.8%) [6—8]. The estimated risk of lead perforation in dogs is 1%—2% [2,9]. Nevertheless, the incidence rate may be underestimated because there is a high occurrence of asymptomatic lead perforations with an otherwise normal function [10]. Perforation can occur acutely (developing during the first 24 h after implantation), subacutely (up to 1 month), or chronically. Another classification system divides perforations into early (occurring during the first month after placement) and late [11—13].

Cardiac perforation can be associated with any factor that induces myocardial injury or myocardial weakening. Long-term use of corticosteroids may induce skeletal muscle atrophy and myocardial atrophy [5]. Some pacemaker leads have a steroid-eluting tip to prevent excess scar tissue formation and subsequent loss of capture from exit block. The presence of a steroid at the tip of the lead could result in a focal weakening of the right ventricular free wall [8].

The lead type may also increase the risk of cardiac perforation. Active-fixation screw-in leads tend to be more prone to perforation than are passive-fixation tined leads [8]. Excessive lead length has also been implicated in generating significant pressure on the heart wall, causing perforation [10]. Small diameter active-fixation pacing leads may be associated with increased risk for delayed right ventricular perforation [11]. Modifications in the lead design that have resulted in increased stiffness at the tip of the lead may increase the rate of perforation [14]. To decrease the force on the myocardial wall, newer softer leads have been developed with more flexible tips [10].

A study performed on 4280 permanent pacemaker-implanted human patients found that one of the strongest predictors of cardiac perforation was the concomitant use of a temporary

transvenous pacemaker. Usually, temporary pacing leads are stiffer than are permanent leads, which could result in more severe myocardial damage [15]. Other factors that may influence the rate of lead perforation include physicians' experience, training level [16,17], and the location of the lead tip in the ventricle. In people, most perforations occur through the right ventricular apex because the myocardial wall is thinner in that location, compared with other common pacing sites such as the interventricular septum and right ventricular outflow tract [13,17]. Experimental animal models have shown that overtorquing during lead placement is associated with increased tissue distortion that might result in cardiac perforation due to the abrasion of the visceral pericardial layer by the protruding helix of the active-fixation leads. This observation suggests that the patient size could be a risk factor for perforation [18].

The case presented here was not treated with corticosteroids previously; the length of the lead was suitable for the size of the patient; temporary pacing was not used during pacemaker implantation; and no echocardiographic changes that could have indicated right ventricular myocardial dystrophy, dysfunction, or right ventricular dysplasia were noted. The only independent risk factor of perforation was the use of an active-fixation screw-in lead.

In the case reported here, the perforation seemed to have occurred 2 weeks after implant, based on the clinical signs of the dog. The absence of pericardial tamponade secondary to lead perforation has previously been reported both in human and in veterinary medicine [6,8,9,11], probably due to a combination of the self-sealing properties of the ventricular wall by muscle contraction and by the lead itself [10]. In the presence of small perforations, lead dislodgement might be clinically insignificant. In these cases, if the cathode remains close to the epicardium and the anode maintains its positioning within the endocardium, pacemaker function is properly maintained [11,16]. Therefore, normal pacemaker function cannot exclude cardiac perforation.

Thoracic radiographs are useful to compare the lead tip position with the previous radiographs, but misdiagnosis may occur if the lead tip is contained within the cardiac silhouette [19]. On an echocardiogram, the lead is difficult to visualize in a single plane and may appear in a correct position. Owing to these limitations, computed tomography is considered the gold standard for the diagnosis of lead perforation in people, but the metal edges of the electrode tip cause the 'star artifact' which makes it difficult to precisely identify the lead tip position [10]. Magnetic resonance imaging would, arguably, be a better cross-sectional imaging technique to delineate soft tissue structures with metal implants. There is evidence that with adequate preparation and monitoring, magnetic resonance imaging can be performed in people equipped with pacemakers [20]. However, in older devices, this technique may result in life-threatening pulse generator dysfunction as ferromagnetic components within the pulse generator and/or lead can be displaced by the strength of the static magnetic field. Furthermore, the pacing electrode may act as an antenna, absorbing the radiofrequency pulses and localizing this energy in the electrode tip as heat, which may burn and potentially rupture the myocardium [20].

The management question of lead perforation is whether to remove it or not. If the lead tip is inside the mediastinum and there are no bleeding complications or potential risk of pulmonary damage, lead extraction remains controversial [10,21]. In this dog, lead extraction was not performed as there were no bleeding complications or damage to adjacent organs, and the length of time elapsed since implantation was assumed to have allowed for significant fibrotic adhesions.

The endocardial lead dislodgement occurred 19 months after epicardial pacemaker implantation. Simultaneously, the dog presented with gastrointestinal and respiratory abnormalities and severe thrombocytopenia. Endothelial damage produced by the lead moving freely within the RV chamber could have triggered an immune response, thus increasing plasma concentrations of both proinflammatory and anti-inflammatory cytokines [22]. Circulating cytokines could have activated the endothelium, platelets, and coagulation cascade, leading to microvascular thrombosis and cellular injury [22]. It is not possible to demonstrate if the lead dislodgement caused an immune response or if immune-mediated hemolytic anemia and thrombocytopenia occurred independently from the presence of the pacemaker implant. The most recent checkup had been performed 5 months before the bleeding complications. At that visit, the owner did not report any clinical signs, physical examination was unremarkable, and the endocardial lead remained in its original position. The presumptive etiological diagnosis of immune response was made from agglutination and Coombs tests results and the clinical response to standard treatment.

The histological examination of the lesion located at the contact site of the epicardial lead consisted of a large area of myocardial tissue necrosis and chronic inflammation. Similar lesions have been

reported to be associated with an exit block in veterinary medicine [2], and in human medicine, they have been attributed to a foreign body reaction to a pacemaker system [23]. Foreign body granuloma is a type of hyperimmune reaction with a characteristic histological appearance: granuloma with a necrotic center surrounded by foreign body multinucleated giant cells. In the present case, a chronic reaction with necrosis and fibrosis was seen but not the typical multinucleated giant cells. Therefore, it is not possible to confirm if this chronic reaction is the result of a previous foreign body granuloma after a healing process.

There are only a few cases of cardiac perforation associated with a pacemaker lead that have been reported in dogs. In a retrospective study, this complication was reported in three animals. One dog died suddenly during the procedure, another one died 2 weeks after the lead was removed and replaced with an epicardial lead, and the last dog lived 14 months after cardiac perforation, with unknown cause of death [2]. In another case, the dog was euthanized just after confirming the diagnosis of cardiac perforation [9], and in a third report, the animal was also euthanized approximately 2 months after implantation of an epicardial lead because of sudden respiratory distress [8]. The case reported here lived 22 months after a cardiac perforation. Therefore, despite the poor prognosis associated with cardiac perforation by pacemaker leads, there are different approaches to successfully manage this major complication. Repositioning the perforating lead may be sufficient in most cases [19]. Another option consists of replacing the endocardial pacemaker lead with an epicardial lead with or without removing the endocardial perforating lead. In any case, extraction of the displaced lead remains controversial as, if the lead is extracted, there is risk of severe bleeding, but if it is not removed, late lead migration can occur.

Conflict of Interest Statement

The authors do not have any conflicts of interest to disclose.

Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jvc.2018.11.006.

Video table

Video 1	Dorsoventral thoracic fluoroscopy view.	The lead tip is apparently out of the cardiac silhouette, suggestive of a cardiac perforation.

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