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Case Report

# Synchronous diaphragmatic contraction associated with dual-chamber transvenous pacing in a dog<sup>☆</sup>



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## KEYWORDS

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**Abstract** A 3-year-old, 20-kg, spayed female, mix-breed dog received a dual-chamber pacemaker for management of symptomatic 3rd-degree atrioventricular block. Synchronous diaphragmatic contraction was documented to occur with atrial pacing. Various methods to reduce the occurrence of pacemaker-related phrenic nerve stimulation are discussed.

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<sup>☆</sup> A unique aspect of the Journal of Veterinary Cardiology is the emphasis of additional web-based images permitting the detailing of procedures and diagnostics. These images can be viewed (by those readers with subscription access) by going to <http://www.sciencedirect.com/science/journal/17602734>. The issue to be viewed is clicked and the available PDF and image downloading is available via the Summary Plus link. The supplementary material for a given article appears at the end of the page. Downloading the videos may take several minutes. Readers will require at least Quicktime 7 (available free at <http://www.apple.com/quicktime/download/>) to enjoy the content. Another means to view the material is to go to <http://www.doi.org> and enter the doi number unique to this paper which is indicated at the end of the manuscript.

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A 3-year-old, 20-kg, spayed female, mix-breed dog was referred after bradycardia was detected on physical examination by the primary care veterinarian during evaluation for a 3-day history of a dry cough. The dog was quiet but alert and was bradycardic (32 beats per minute; bpm) with a regular rhythm. A grade 3/6 holosystolic murmur was heard at the left heart base, and femoral pulses were strong. Electrocardiogram revealed 3rd-degree atrioventricular (AV) block with an atrial rate of 140 bpm and a regular ventricular escape rhythm at a rate of 34 bpm. Echocardiography<sup>c</sup>

<sup>c</sup> Vivid E9 GE Medical Systems, Milwaukee, WI, USA.

### Abbreviations

AP-VP	atrial pace-ventricular pace
AS-VP	atrial sense-ventricular pace
AV	atrioventricular
Bpm	beats per minute
DDD	dual chamber, dual lead, synchronous pacing
LV	left ventricular
RA	right atrial
VDD	dual chamber, single lead, synchronous pacing
VVIR	single chamber, single lead, ventricular inhibited, rate-responsive, asynchronous pacing

(Vivid E9 GE Medical Systems; Milwaukee, WI, USA) revealed mild-to-moderate four-chamber dilation (Video 1), with increased aortic and pulmonic outflow velocities (3.5 m/s and 2.1 m/s, respectively; reference intervals  $1.29 \pm 0.22$  and  $1.05 \pm 0.19$ , respectively) [1], and mild diastolic mitral regurgitation. Echocardiographic findings were consistent with volume overload secondary to bradycardia, and differentials for the increased aortic velocity included increased stroke volume related to bradycardia or mild aortic stenosis. Radiographs revealed generalized cardiomegaly (vertebral heart size of 13v) and a mild diffuse interstitial pattern. The results of the complete blood count, serum biochemistry profile, and urinalysis were normal. A SNAP test<sup>d</sup> (SNAP 4Dx® Plus Test; IDEXX Laboratories Inc., Westbrook, ME, USA) was negative for antigens against *Dirofilaria immitis*, *Borrelia burgdorferi*, *Anaplasma phagocytophilum* and *Anaplasma platys*, and *Ehrlichia canis* and *Ehrlichia ewingii*, and a quantitative C6 antibody test for Lyme disease<sup>e</sup> (Lyme Quant C6® Antibody by ELISA; IDEXX Laboratories Inc., Westbrook, ME, USA) was negative at  $<10$  U/ml ( $<30$  U/ml considered clinically insignificant). The total T4 level was normal at 3.5 ug/dl (reference range, 1.0–4.1 ug/dl).

The dog was admitted for permanent transvenous pacemaker placement. Given the dog's young age and apparently normal atrial function, a dual-chamber pacemaker<sup>f</sup> (Zephyr® XL DR 5826)

<sup>d</sup> SNAP 4Dx® Plus Test, IDEXX Laboratories Inc., Westbrook, ME, USA.

<sup>e</sup> Lyme Quant C6® Antibody by ELISA, IDEXX Laboratories Inc., Westbrook, ME, USA.

<sup>f</sup> Zephyr® XL DR 5826.

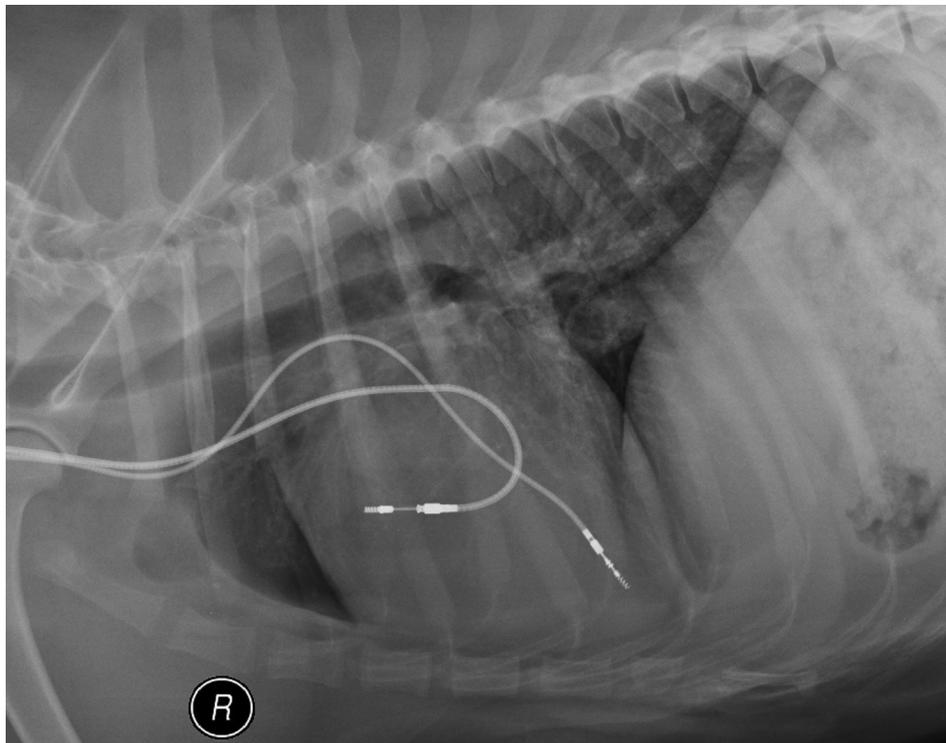
was placed. A ventricular lead<sup>g</sup> (Medtronic active fixation lead; 5076; 52 cm) was advanced into the right ventricular apex, and an atrial lead<sup>h</sup> (Medtronic atrial J-tip active fixation lead; 5568; 53 cm) was advanced into the right atrial (RA) appendage (Video 2). Both were secured with active fixation, and the pacemaker was programmed to DDD (dual chamber, dual lead, synchronous pacing) mode (base rate, 60 bpm; atrial and ventricular pulse amplitude, 2.5 V (V) and pulse width, 0.4 ms; atrial sensitivity, 0.75 mV and ventricular sensitivity, 2 mV). Intraoperative pacemaker testing revealed atrial and ventricular capture thresholds of  $<0.25$  V, atrial sensing of  $>5$  mV, and atrial and ventricular impedance of 681 and 709  $\Omega$ , respectively. Ventricular sensitivity testing was not performed because of total pacemaker dependence. The patient recovered well from surgery, and the following day, the echocardiogram revealed reduction of the previously measured left atrial and left ventricular (LV) dimensions (Video 1) and reduced aortic and pulmonic outflow velocities (2.1 m/s and 1.6 m/s, respectively). Pacemaker settings were altered to maximize physiologic pacing (base rate, 40 bpm; rest rate, 35 bpm), and the dog was discharged on lifelong clopidogrel (1.9 mg/kg PO q 24 h) due to concern for increased risk of future thrombus in a young dog who may have a pacemaker for many years [2,3].

On recheck 31 days after surgery, the owners reported that the dog had been doing clinically well at home, with resolution of the cough and increased activity level after pacemaker placement. However, they described rhythmic abdominal contractions that were occasionally present when the patient was at rest. The dog did not show signs of distress related to these events but was occasionally awoken from sleep. The contractions had not been reproducible at recheck suture removal with the primary care veterinarian. The owners had recorded an example that showed clear diaphragmatic contractions at a rate of 35 bpm, which was the programmed minimum rest rate of the pacemaker.

Upon interrogation of the pacemaker, diaphragmatic contractions could be reproduced by atrial pacing at atrial lead outputs of greater than 2 V (Video 3). Given the ability to create diaphragmatic contractions that coincided with the rate of atrial pacing, it was suspected that the phrenic nerve was being stimulated by the atrial lead. The recorded data showed that the time spent

<sup>g</sup> Medtronic active fixation lead; 5076; 52 cm.

<sup>h</sup> Medtronic atrial J-tip active fixation lead; 5568; 53 cm.



**Figure 1** Right lateral thoracic radiograph showing mild generalized cardiomegaly and pacing leads superimposed with the right atrium and right ventricle.

in atrial pace-ventricular pace (AP-VP) mode and atrial sense-ventricular pace (AS-VP) mode was 44% and 56%, respectively. Atrial and ventricular capture thresholds were less than 0.25 V and 0.75 V, respectively, and atrial sensing was 4.6–4.9 mV. The atrial lead output was decreased to 2 V, and the base rate was decreased to 30 bpm to allow the patient to spend more time in AS-VP mode, which did not result in diaphragmatic contraction.

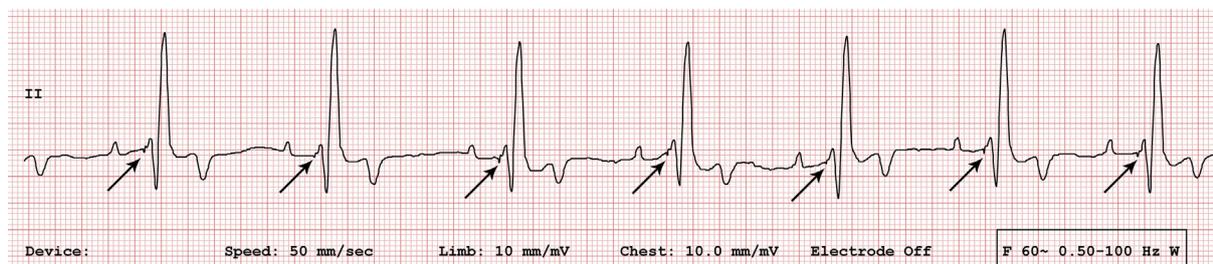
Radiographs revealed a reduction in cardiac size (vertebral heart size 11v) and resolution of the previously noted interstitial pattern. The pacing leads were visible, superimposed with the right atrium and right ventricle, as at the time of surgery (Fig. 1). Point of care blood work<sup>i</sup> (i-STAT; Abbott Laboratories, Abbott Park, IL, USA) revealed normal electrolytes, including ionized calcium. An electrocardiogram showed sinus arrhythmia with paced ventricular rhythm synchronized with intrinsic atrial beats (Fig. 2).

The dog presented for scheduled recheck 4 months after pacemaker placement. She had been doing well at home, with no abnormal clinical signs. The diaphragmatic contractions had not occurred since the pacemaker settings had been adjusted at

the previous visit. An echocardiogram revealed LV chamber dilation and decreased contractile function (fractional shortening 19%; Video 1). The LV walls were thinner than at prior examinations but still normal on weight-based normalized indices. The left atrium was normal in size, and pacing leads were visible within the right ventricle and RA. Differentials for the echocardiographic changes included primary cardiomyopathy, myocarditis, or bradycardia-induced changes resulting from sinus node dysfunction.

Pacemaker interrogation showed that time spent in AP-VP and AS-VP modes was 28% and 72%, respectively. The heart rate histogram showed that the heart rate was less than 50 bpm the majority of the time. Atrial pacing again resulted in diaphragmatic contractions at an output of 2 V, and visible contractions ceased at an atrial lead output of 1 V. Atrial capture was lost at 0.5 V, and an ECG was performed during the atrial capture test (Fig. 3). The settings were increased to a base rate of 55 bpm with a rest rate of 45 bpm, due to concern that a component of sinus node dysfunction could have contributed to the chamber dilation since the previous examination, based on pacemaker-recorded data indicating a persistently low atrial rate. However, sinus node function was not further characterized via Holter monitor. The

<sup>i</sup> i-STAT, Abbott Laboratories, Abbott Park, IL, USA.



**Figure 2** Lead II electrocardiogram showing sinus arrhythmia with paced ventricular rhythm (left bundle branch morphology) synchronized with intrinsic atrial beats (heart rate: 88–107 bpm). Small pacing artifacts are visible preceding the QRS complexes (arrows). Amplitude: 10 mm/mV. Paper speed: 50 mm/s.

atrial output was reduced to 1 V in an attempt to avoid diaphragmatic contraction with settings that would result in more frequent AP-VP mode. In addition, relatively low base rate settings were maintained to avoid constant atrial pacing.

Serial rechecks showed persistent LV dysfunction (Video 1) and elevated N-terminal pro-B-type natriuretic peptide concentration (4402 pmol/L; reference range, 0–900 pmol/L). The left atrium remained normal in size. The dog was started on pimobendan (0.24 mg/kg PO q 12 h), and LV cavity size (Video 1) and NT-proBNP levels (2335 pmol/L) were reduced at the following visit. Time spent in AP-VP and AS-VP modes has ranged from 31 to 41% and from 59 to 69%, respectively. The dog has occasional periods of diaphragmatic contraction during sleep but otherwise continues to do clinically well over 1 year after pacemaker implantation.

## Discussion

Permanent pacemaker placement is the treatment of choice for symptomatic bradyarrhythmias, with transvenous, VVIR (single

chamber, single lead, ventricular inhibited, rate-responsive, asynchronous pacing) being most commonly used in dogs [4–8]. The long-term superiority of physiologic VDD (dual chamber, single lead, synchronous pacing) or DDD pacing over single-chamber VVIR pacing has not been demonstrated in veterinary medicine [9–11]. However, synchronous AV pacing has been shown to have short-term hemodynamic and neurohumoral benefits in dogs [12] and has been associated with decreased occurrence of pacemaker syndrome and improved quality of life and functional status in people [13,14]. Therefore, VDD or DDD pacing is still a reasonable option for dogs, especially in young animals who may be reliant on a pacemaker for many years, or in dogs with structural heart disease or heart failure who may suffer from the loss of consistent atrial contribution to cardiac output.

Commonly reported pacemaker complications in dogs include peri-operative death, pacing lead dislodgement, infection, generator failure, mild incisional complications, and mild postoperative arrhythmias [4–6,9,15]. Rates of major complications appear to be similar for various pacing



**Figure 3** Lead II electrocardiogram performed during an atrial capture test showing transition from atrial sensing to atrial pacing and ultimately loss of atrial capture. There is a change in the P wave morphology with the appearance of atrial pacing artifacts (arrow). The final atrial pacing artifact does not result in a P wave (arrow head). The ventricular rhythm is paced, but ventricular pacing artifacts are not clearly visible. Amplitude: 10 mm/mV. Paper speed: 25 mm/s.

modalities and lead types [8–10]. Phrenic nerve stimulation has been noted as a minor complication in 2 dogs with dual-chamber pacemakers [9] but has not been described in detail. The phrenic nerves in the dog typically arise from the 5<sup>th</sup>–7<sup>th</sup> cervical nerves and are closely associated with the right and left atria as they traverse the thorax to innervate the diaphragm [16]. They can be inappropriately triggered by external electrical stimuli or by normal cardiac electrical activity in the setting of severe hypocalcemia [17–20].

In humans, pacemakers are commonly placed for bradyarrhythmias or cardiac resynchronization therapy [14]. Unintentional phrenic nerve stimulation is known to be a relatively common complication of cardiac resynchronization therapy where the LV lead is positioned within the coronary sinus in close proximity to the left phrenic nerve [14,17–19,21]. However, there are few reports of stimulation of the right phrenic nerve by an RA pacing lead. Atrial or ventricular lead displacement resulting in phrenic nerve stimulation has been described in people [22,23], and there is a single report of phrenic nerve stimulation by an appropriately positioned RA pacing lead [24]. The authors postulated that the abnormal stimulation resulted from the unusually large interelectrode distance of the atrial lead, and diaphragmatic contractions ceased to occur when the lead was reprogrammed to function as unipolar rather than bipolar.

In the present report, there was no evidence of atrial lead displacement, based on radiographic findings and continued normal atrial sensing and capture. However, local perforation of the RA wall cannot be ruled out as perforation can result in either no change or an increase in capture threshold, either no change or a decrease in sensing, and typically no change in impedance [17]. In addition, while the lead was thought to be within the RA appendage, one of the signs of appropriate lead placement is a ‘windshield wiper’ motion during atrial contraction [25,26]. In the present case, the RA lead moved with atrial contraction, but the motion was less dramatic than that sometimes observed, indicating that the lead may not have truly been within the RA appendage. An orthogonal radiographic view would have been beneficial in determining lead positioning. Electrolytes were normal, ruling out hypocalcemia-induced sensitivity of the phrenic nerve.

Phrenic nerve stimulation resulting in diaphragmatic contraction can cause discomfort, dyspnea, and altered hemodynamics in people [18,19,21]. Intraoperative testing is commonly

performed in people to assess for the presence of phrenic nerve stimulation and need for lead repositioning [21,26,27]. Maximal output pacing can reveal extracardiac stimulation that was not visible at standard outputs [26], and acoustic cardiography has been used to detect subclinical phrenic nerve stimulation [19]. In the present case, diaphragmatic contractions were not observed until recheck, and although reoperation to reposition the lead was an option, it was not pursued as the dog was doing clinically well. There are a variety of non-invasive pacemaker programming changes that may decrease the frequency of phrenic nerve stimulation in such a scenario, including adjustments to the mode, lead polarity, base rate, and pulse amplitude or duration.

One method that would stop phrenic nerve stimulation by the RA lead is to reprogram the pacemaker to VVIR mode, but this would eliminate the potential benefit of physiologic VDD or DDD pacing. In the present case, the dog was already showing signs of myocardial dysfunction, so it was desirable to maintain settings that allowed for sequential AV contraction. However, the actual benefit of AV synchrony in avoiding myocardial dysfunction remains unknown, and it is possible that VVIR pacing at a higher rate would have been more beneficial in this case. The pacemaker could also be reprogrammed to VDD mode, but this relies on having a normal sinus rate, and the dog in this report had chronic sinus bradycardia and may have had sinus node dysfunction. If the pacemaker is set to VDD mode and the intrinsic atrial rate drops below the base rate, this would, in effect, result in VVI mode pacing that would also result in loss of sequential AV contraction.

Another possible approach would be to reprogram the atrial lead to unipolar. This resolved phrenic nerve stimulation in a human case report of a lead with a wide interelectrode distance [24]. Experimental studies in dogs have shown that unipolar settings result in a higher threshold for phrenic nerve stimulation than bipolar settings [18]. The 17.8-mm interelectrode distance in this report was typical of atrial leads used in dogs without provoking phrenic nerve stimulation, but unipolar pacing at 2 V still may have mitigated phrenic nerve stimulation by failing to capture the extracardiac tissue. This tactic was not attempted in the present case because unipolar pacing can increase the risk of inappropriate sensing and skeletal muscle stimulation at the point of contact with the pacemaker generator [17], but it could be a consideration for the future.

Lowering the pacemaker rate to a point at which the patient's sinus rate is typically greater than the base rate would reduce the frequency of atrial pacing and therefore reduce phrenic nerve stimulation. Initially, the base rate in the present case was lowered to favor AS-VP mode, but this was ultimately not sustainable given the possibility that the patient may have had a component of sinus node dysfunction and bradycardia-induced cardiac remodeling or other underlying myocardial dysfunction.

Alternatively, the atrial lead output could be decreased to the point at which phrenic nerve stimulation did not occur, which was the approach taken in the present case. However, this may not always be possible as the myocardial and phrenic nerve thresholds may not be sufficiently different, and phrenic nerve stimulation can occur even at standard outputs [19].

Finally, in cases of phrenic nerve stimulation resulting from LV pacing, it has been shown that increasing the pulse duration can reduce the myocardial threshold to a greater extent than the phrenic nerve threshold [17,18,21]. This is a result of differences in the rheobase (minimum pulse amplitude that can cause depolarization at maximum pulse duration) and chronaxie (the minimum pulse duration that can cause depolarization at a pulse amplitude of twice the rheobase) of myocytes and nerve fibers [21]. Therefore, increasing pulse duration and decreasing the pulse amplitude is another approach that could be considered to reduce extracardiac stimulation. However, this could result in greater loss of battery life which could become clinically relevant in a young dog [17,18,21].

In conclusion, phrenic nerve stimulation is a rare but possible complication of atrial pacing lead placement in dogs. This does not appear to be dangerous for the dog but may be bothersome to both the dog and the owner. Intraoperative testing to assess for phrenic nerve stimulation and orthogonal radiographic views to confirm placement within the RA appendage can help avoid this complication. If phrenic nerve stimulation does occur and physical repositioning of the lead is not possible, there are several pacemaker programming changes that may resolve or reduce diaphragmatic contractions.

## Conflicts of Interest Statement

Emily Karlin, John Rush, and Edward Nobrega have no financial or personal interests to disclose that could influence the submitted work. In the last 5 years, Dr. John Rush has given lectures sponsored by, done research supported by, or participated as a consultant for Boehringer Ingelheim, Royal Canin, Nestle Purina, Aratana, and Idexx. Dr. Edward Nobrega is a former employee of St. Jude Medical and Guidant Corporations.

## Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jvc.2018.10.003>.

Video table		
Video	Video title	Description
1	Serial echocardiography	Serial echocardiography showing right parasternal short axis 2D images of the left ventricle.
2	Pacing lead placement – Fluoroscopy	Fluoroscopy showing an active fixation pacing lead positioned in the right ventricular apex while the J-tip active fixation atrial pacing lead is advanced into the right atrial appendage. Once in place, the atrial lead can be seen moving with atrial kick, just before ventricular contraction.
3	Synchronous diaphragmatic contraction	Video obtained on recheck examination; pacemaker settings were DDD with base rate of 120 bpm and atrial output of 2.5 V. The top of the video shows the pacemaker interrogation screen with pacing markers, atrial electrogram (EGM), and ventricular EGM, from top to bottom. Diaphragmatic contraction is seen with every atrial pacing spike.

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