Outcomes after atrioventricular node ablation and biventricular pacing in patients with refractory atrial fibrillation and heart failure: a comparison between non-ischaemic and ischaemic cardiomyopathy

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Received 28 July 2013; accepted after revision 18 November 2013; online publish-ahead-of-print 12 February 2014

Aims
Atrioventricular junction ablation (AVJA) combined with biventricular (BiV) pacing (AVJA/BiV) is an effective treatment for refractory atrial fibrillation (AF) and rapid ventricular response (RVR) associated with heart failure (HF). This study compared the outcomes between patients with non-ischaemic (DCM) and ischaemic cardiomyopathy (ICM) following AVJA/BiV for AF/RVR.

Methods and results
This was a retrospective study of 45 patients, comparing the response to AVJA/BiV in patients with ICM to those with DCM. The study compared (a) the change in echocardiographic parameters of HF (ejection fraction (EF) and left ventricular dimensions) prior to, and at least 6 months post AVJA/BiV; and (b) HF hospitalizations (HFH) and appropriate implantable cardioverter defibrillator (ICD) therapies occurring post-procedure. Ejection fraction improved significantly in the DCM group (ΔEF 11.2% ± 11.9; P < 0.01); however, EF remained unchanged (ΔEF 0.5% ± 9.9; P = NS) in the ICM group post-AVJA/BiV. Post-procedurely, HFH were significantly more common (15/18 vs. 4/25; P < 0.0001), and there were significantly more appropriate ICD therapies (9.4 ± 12.3 vs. 2.3 ± 6.1; P = 0.01) in the ICM compared with the DCM group.

Conclusion
After AVJA/BiV, there was significantly less post-procedural echocardiographic reverse remodelling, and more HFH in the ICM compared with the DCM group. In addition, significantly more appropriate ICD therapies occurred in ICM patients post-procedure. These differences may be due to the presence of more extensive discrete myocardial scar in patients with ICM. Furthermore, it is possible that tachycardia-induced cardiomyopathy plays more of a causative role in HF in patients with AF and DCM than those with ICM.

Keywords
Atrial fibrillation • Biventricular pacing • AV node ablation • Ischaemic cardiomyopathy • Non-ischaemic cardiomyopathy

Introduction
Both heart failure (HF) and atrial fibrillation (AF) are increasingly prevalent public health problems resulting in significant morbidity and mortality. The two conditions share several risk factors such as hypertension, obesity, coronary artery disease (CAD), and advanced age, all of which are increasing in incidence.

Atrial fibrillation is known to worsen the clinical course of HF through multiple mechanisms including rapid ventricular response (RVR), irregularity of ventricular rhythm, loss of organized atrial contribution to cardiac output, and in some cases, tachycardia-induced cardiomyopathy (TIC). Despite recent advances in radiofrequency ablation and anti-arrhythmic drug therapy, curative therapy for long-standing, persistent AF remains challenging in some patients.
What’s new?
- Patients with non-ischaemic cardiomyopathy have a significant improvement in EF, hospitalizations for heart failure, and appropriate ICD therapies after AVJ ablation and BiV pacing as compared with patients with ischaemic cardiomyopathy.
- Tachycardia-induced cardiomyopathy likely plays a significant role in patients with non-ischaemic cardiomyopathy and atrial fibrillation.
- The extent of pre-existing myocardial scar directly correlates with lack of response to BiV pacing in patients with CHF and refractory atrial fibrillation.

and AF is deemed ‘permanent’ which is treated with rate control and anticoagulation. In a significant number of patients, adequate ventricular rate control with atio-ventricular (AV) nodal blocking drugs is not possible, and atrioventricular junction ablation (AVJA) combined with institution of permanent pacing is indicated. In the presence of HF, biventricular (BiV) pacing is usually chosen to prevent right ventricular pacing-induced cardiac dysynchrony, and is often combined with an ICD.

A superior effect on outcomes has been noted in those with a de novo indication for cardiac resynchronization therapy (CRT) in patients with non-ischaemic (DCM) compared with ischaemic cardiomyopathy (ICM). This has not been previously studied following combined AVJA with BiV ICD implantation (AVJA/BiV) in patients who did not necessarily have a pre-existing de novo indication for CRT. Such patients are fundamentally different from prior studies since (1) they have permanent AF with poor rate control and have undergone AVJA; and (2) BiV pacing is often performed solely for the prevention, as opposed to the correction, of cardiac dysynchrony. The primary purpose of our study was to compare the echocardiographic and clinical response between patients with DCM and ICM, largely without a de novo indication for CRT, who had undergone AVJA/BiV for drug-refractory AF/RVR and systolic HF.

Methods

Ethics
This study complies with the ethical principles of the revised Declaration of Helsinki (Somerset West, Republic of South Africa, 1996). The Institutional Review Board at the Veterans Health Administration North Texas Healthcare System approved the study protocol.

Study population
This was a single-centre, retrospective analysis of 45 patients who had undergone concurrent AVJA/BiV (defined as AVJA within 1 month of BiV implant/upgrade) for (1) symptomatic HF requiring multiple hospital admissions, and an ejection fraction (EF) < 35%; and (2) permanent AF with documented poor pharmacologic ventricular rate control. The indication for BiV pacing in the majority of patients was to prevent RV pacing-induced cardiac dysynchrony after AVJA, although 4 of 45 patients had ≥ class IIa indication for CRT by QRS duration and morphology criteria. Patients undergoing BiV upgrade for the correction of cardiac dysynchrony due to RV pacing were excluded. All left ventricular (LV) leads were placed in a lateral or postero-lateral location. All patients received a CRT device with ICD capability (CRT-D).

Patients with prior ICDs undergoing AVJA/BiV upgrade were included. Thirteen patients (seven in the DCM group and six in the ICM group) had an ICD prior to AVJA/BiV upgrade. Ten of these 13 patients had their prior ICD implanted for primary prevention.

Patients were also excluded if (1) pre-and post-procedure transthoracic echocardiograms (TTE) were not available, and (2) accurate determination of aetiology of HF (ischaemic vs. non-ischaemic) could not be made.

Clinical classification
Patients were classified as having ICM if there was one or more of (a) a history of coronary artery bypass (CABG); (b) angiographic evidence of significant flow-limiting CAD in at least two major epicardial vessels; (c) definitive evidence of prior Q-wave myocardial infarction by both clinical and electrocardiographic (EKG) criteria.

Hospitalizations for worsening heart failure
Patient’s electronic medical records were analysed from the date of the procedure to the last follow-up or date of death. Hospitalization for heart failure (HHF) was defined as any hospital admission for management of decompensated HF as a primary admission diagnosis.

Echocardiography
Pre-procedure and post-procedure TTEs (minimum post-procedure interval of 6 months) were analysed. Patients underwent TTE in the left lateral decubitus position with a commercially available system.

Left ventricular dimensions and ejection fraction
Left ventricular dimensions were obtained from the M-mode parasternal long-axis view. Left ventricular end-systolic dimension (LVESD) was defined as peak downward motion of the interventricular septum, and LV end-diastolic dimension (LVEDD) as the point immediately prior to downward displacement of the septum. Left ventricular ejection fraction (LVEF) was calculated in pre-procedure echocardiograms by two different methods: Simpson’s method using the 4 chamber apical view in 29/45 patients, and the Teichholz method using M mode measurements of the LV in 16/45 patients. Post-procedure LVEF was calculated using Simpson’s method in all patients. Pre-procedure TTEs were performed during AF, with measurements averaged over 3 cardiac cycles. All images post-procedure were made during ventricular pacing at 80 beats-per-minute (bpm). Images were analysed offline by an investigator blind to the clinical classification of the patients.

Echocardiographic scar analysis
We looked for the presence, extent, and location of scar using TTE. Scarred segments were defined as those displaying both: (1) a reduction in end-diastolic wall thickness (EDWT) of ≤ 0.5 cm; and (2) an abnormal increase in acoustic reflectance noted by the loss of contractile, granular reflection of healthy myocardium, which is substituted by akinetic, highly echo reflective linear tissue, characteristic of scar. In addition, scar tissue demonstrates a lack of distinction between epicardial, myocardial, and endocardial layers. Each image set was analysed using a 17-segment analysis where segments were individually labelled as having scar or not. We compared the DCM and ICM groups with respect to: (i) number of patients with any scar; (ii) mean number of scarred segments; and (iii) presence or absence of scar in either lateral or posterolateral LV segments.
Appropriate implantable cardioverter defibrillator therapies

Post-procedure appropriate ICD therapies were analysed from ICD interrogations by an investigator blinded to the clinical classification of the patient. Appropriate ICD therapies were defined as either anti-tachycardia pacing, or shock for ventricular arrhythmia. Since patients had all undergone successful AVJA, discrimination between appropriate and inappropriate therapies for supraventricular arrhythmias was not necessary. Implantable cardioverter defibrillator therapies for lead noise or electromagnetic interference were excluded.

Statistical analysis

The primary endpoint for the study was a comparison of the change in LVEF (ΔEF) from pre-to post-procedure between patients with DCM and those with ICM. Secondary echocardiographic endpoints included the change (Δ) in LV dimensions, and comparison of scar burden between groups. Comparisons of baseline and follow-up echocardiographic data and the change (Δ) in parameters between DCM and ICM groups were analysed using a non-parametric test (Kruskal–Wallis rank sum). Scar burden comparisons between groups were performed using a t-test, a P value of <0.05 was considered significant. Continuous patient demographic data were presented as mean ± standard deviation and were analysed using the two-tailed t-test. Comparison of both post-procedural HFH, and appropriate ICD therapies between DCM and ICM groups was performed using a non-parametric (Mann–Whitney U) test. Comparisons of time-to-first HFH, as well as appropriate ICD therapy between groups. Comparisons of baseline and follow-up echocardiographic data and the change (Δ) in parameters between DCM and ICM groups were performed using a non-parametric (Mann–Whitney U) test.

Results

Baseline clinical characteristics of the study groups are displayed in Table 1. Mean number of AV nodal blocking agents prior to AVJA/BIV was 2.07 ± 0.55 in the DCM group, and 2.00 ± 0.49 in the ICM group (P = NS).

Patient classification

Of 45 patients, 18 were classified as ICM and 27 were classified as DCM. Of 18 ICM patients, 9 were classified by a history of prior CABG, 8 by angiographic, and 1 by EKG/clinical criteria. Of 27 DCM patients, 23 were classified by angiographic, and 4 by EKG/clinical criteria.

Baseline QRS and heart rates

The baseline heart rate (HR) was averaged for each patient from pre-procedural resting EKG, and pre-procedural physical examination. A correlation analysis was performed between HR obtained from the EKG and that from the physical examination, and this showed a strong correlation (R = 0.75; P < 0.01). The baseline HR for the DCM and ICM group was 88 ± 27 and 72 ± 18 bpm, respectively (P = 0.22). Baseline QRS duration for the DCM group was 111 ± 29 ms and for the ICM group was 128 ± 31 ms (P = 0.08).

Six of 27 patients in the DCM group had a QRS duration >120 ms; however, only 2 of 27 had a ≥ class IIa indication for CRT (by QRS duration and morphology criteria).10 Four of 18 patients in the ICM group had a QRS duration >120 ms; however, only 2 of 18 had a ≥ class IIa indication for CRT by QRS duration and morphology criteria.10

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<th>Table 1 Baseline characteristics of patients</th>
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<td>Patient characteristics</td>
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Values are given as mean ± standard deviation or no. (%).

COPD, chronic obstructive pulmonary disease; ACE, ACE inhibitors; ARB, angiotensin receptor blockers; LVEDD, left ventricular end diastolic diameter; LVESD, left ventricular end systolic diameter.

Left ventricular ejection fraction and left ventricular dimensions

Mean interval from pre-procedure to post-procedure echocardiogram was 20.2 ± 14.45 months. Baseline EF was 27.9% ± 10.0 in the ICM group compared with 32.5% ± 9.3 in the DCM group (P = 0.09). ΔEF was non-significant in the ICM group (0.5% ± 9.9; P = NS) compared with a significant improvement (11.2% ± 11.9; P < 0.01) in the DCM group, representing a significant difference in ΔEF between groups (P < 0.01) (Figure 1). Using a post-procedure LVEF of ≥50% to define advanced resolution of cardiomyopathy, 8 out of 27 DCM patients, and none of the ICM patients had advanced resolution of cardiomyopathy.

Baseline LVESD in the ICM group was 5.5 ± 1.3 cm compared with 5.0 ± 0.9 in the DCM group (P = 0.25). ΔLVESD was not significant in the ICM group (0.0 ± 0.7; P = NS), but was significantly decreased in the DCM group (−0.7 ± 0.08 cm; P < 0.05), representing a significant difference in ΔLVESD between groups (P = 0.02). Baseline LVEDD in the ICM group was 6.2 ± 1.1 cm compared with 5.6 ± 0.8 cm in the DCM group (P = 0.6). ΔLVEDD was not significant (0.3 ± 0.6 cm; P = NS) in the ICM group or in the DCM group (0.1 ± 0.7 cm, P = NS). Furthermore, there was no significant difference in ΔLVEDD between the groups (P = NS).
The principle findings of the study are as follows: (1) patients with DCM displayed significantly more favourable echocardiographic ventricular reverse remodelling than patients with ICM; (2) patients with DCM experienced significantly less HFH and a longer time-to-first HFH post-procedure compared with the ICM group; (3) patients with DCM received significantly fewer appropriate ICD therapies and had a longer time-to-first appropriate ICD therapy compared with the ICM group.

**Scar analysis**

Three of 27 patients (11%) in the DCM group had scar in any segment compared with 16 of 18 (89%) ischaemic patients. Mean number of scarred segments in the DCM group was 0.30 ± 0.89 compared with 5.22 ± 3.1 in the ICM group (P < 0.001). Only 1 of 27 patients (3.7%) in the DCM group had scar in one or more lateral or posterolateral segments compared with 12 of 18 (66%) of the ICM patients.

**Hospitalizations for heart failure**

Median survival post-procedure was 612 days. In the DCM group, 4 of 25 patients required HFH while 15 of 18 in the ICM group required HFH during the 2-year follow-up (P < 0.0001). Post-procedure HFH occurred at a rate of 0.26 ± 0.7 per patient in the DCM group compared with 2.39 ± 2.1 per patient in the ICM group (P < 0.001). The hazard ratio for HFH in ICM compared with DCM was 10.77 (P < 0.001). Time-to-first HFH was significantly longer in the DCM group compared with the ICM group (Figure 2).

**Appropriate implantable cardioverter defibrillator therapies**

Eight of 27 of the DCM patients received at least one appropriate ICD therapy, compared with 11 of 17 of the ICM patients (P = 0.01). There were an average of 2.3 ± 6.1 therapies in the DCM group compared with 9.4 ± 12.3 therapies in the ICM group (P = 0.01). The hazard ratio for receiving an appropriate ICD therapy in ICM compared with DCM was 3.67 (P = 0.01). Time-to-first appropriate ICD therapy was significantly longer in the DCM group compared with the ICM group (Figure 3). Our study was not powered to detect an effect of AVJA/BiV upgrade on the incidence of appropriate ICD therapies in patients who already had an ICD.

**Discussion**

This is the first study to compare the outcome after AVJA/BiV between patients with ICM and DCM who had permanent AF associated with poor ventricular rate control and HF. Our study had three principle findings: (1) patients with DCM displayed significantly more
between DCM and ICM groups in our study is likely a higher burden of discrete myocardial scar in patients with ICM compared with DCM. Such scar renders myocardial tissue non-contractile. In patients with ICM, the burden of scar has been found to correlate inversely with the response to CRT.15,16 Echocardiographic evidence of an apical wall motion abnormality and posterolateral scar identified by MRI have both been shown to predict a negative response of an apical wall motion abnormality and posterolateral scar identified by MRI have both been shown to predict a negative response.

We looked for myocardial scar burden and location using a well-validated definition of scar on TTE.7 This definition of scar has been shown to correlate with: (a) non-recovery after revascularization (nonviability),8 (b) myocardial viability as assessed by rest-redistribution thallium-201 uptake,9 and; (c) response to CRT for de novo cardiac dyssynchrony (outside of the setting of BiV pacing after AVJA for AF/RVR).7 Using this definition of scar, we found a significantly higher scar burden in the ICM group, indicating a higher burden of permanent non-viability.

A second major reason for the difference in response between DCM and ICM groups is likely a difference in the degree of TIC between groups. Significant TIC is diagnosed retrospectively if significant HF reversal occurs after cure or control of the putative tachycardia. ‘Pure’ TIC might be expected to be associated with complete normalization of the EF after effective tachycardia cure/rate-control. We used a post-procedure LVEF of ≥50% to define complete resolution of TIC and thus infer a diagnosis of ‘pure’ TIC. By this definition, 8 of 27 DCM patients (just less than 30%) had complete resolution of depressed EF and thus probably had ‘pure’ TIC. None of the ICM patients had complete resolution of depressed EF. Our results suggest that ICM patients presenting with HF and AF/RVR may have a degree of TIC, though in general, AF is more likely a contributory/exacerbating as opposed to a causative factor in the precipitation of HF in this patient group. Patients with DCM are more likely to have TIC as a significant causative factor of clinical HF, and in some instances this represents a pure TIC.

The pattern of echocardiographic reverse remodelling seen in the DCM group in the study (EF and LVESD improving out-of-proportion to LVEDD) is in keeping with resolution of TIC in which ventricular dilation has been reported to resolve disproportionately slower and to a lesser degree than EF in both animal models19 and patients.20 The third possible reason for the difference in response between groups is the finding that dense lateral or posterolateral scar in 66% of ICM patients may have resulted in effectively RV-only pacing in some of these patients. Several studies, including studies performed post-AVJA, have shown a potential detrimental effect of RV pacing on cardiac function.21–24

### Appropriate implantable cardioverter defibrillator therapies

In our study, significantly more post-procedure ICD therapies for ventricular tachyarrhythmias occurred in ICM compared with DCM patients. It may be that the patient group with DCM had less ventricular arrhythmia at baseline. Nevertheless, the magnitude of the difference between groups was substantial, likely pointing to a true differential effect of AVJA/BiV between these two patient groups. Due to the small number of patients who had an ICD prior to AVJA/BiV, or on the comparison between ICM and DCM patient groups in this setting. Previous studies examining the risk factors for tachyarrhythmia therapy in ICD recipients have not demonstrated a reduced risk of therapies in patients with DCM. In the 811 patients in the sudden cardiac death in heart failure trial who were randomized to ICD therapy, 269 received ICD therapies and the proportion of patients with DCM and ICM who received therapies was the same.25 In a study on 1382 patients implanted with an ICD for a broad range of indications, no significant difference in the rate of appropriate ICD therapies was detected between DCM and ICM patients.26 In this study, NYHA class predicted appropriate ICD therapy. Evonich et al.27 compared the incidence of appropriate ICD therapies in 153 consecutive patients with non-sustained ventricular tachycardia (VT) and a depressed EF undergoing ICD implantation. In this study, patients with DCM were more likely to receive therapies than those with ICM. In the retrospective study by McLeod et al.14 503 CRT recipients underwent CRT with no significant difference in ICD therapies (both appropriate and inappropriate) noted between DCM and ICM groups.

Appropriate ICD therapies have been shown to be reduced by manoeuvres that reduce HF and result in ventricular reverse remodelling. A reduction in VT inducibility in patients who respond to CRT has been reported.19,28 Higgins et al.29 reported a decreased incidence of appropriate ICD therapies when patients were in the active CRT phase of the VENTAK CHF trial. Markowitz et al.30 reported that responders to CRT had less ICD therapies for VT or ventricular fibrillation, and were more likely to have DCM than ICM. Upgrading an ICD-alone system to one combined with CRT has been reported to significantly reduce the frequency of appropriate ICD shocks.31,32 In summary, the aetiology of HF (whether ICM or DCM) does not appear to significantly influence the frequency of appropriate ICD therapies occurring post-procedure in DCM and ICM groups. DCM, dilated cardiomyopathy; ICM, ischaemic cardiomyopathy.

**Figure 3** Kaplan Meier curves depicting appropriate ICD therapies occurring post-procedure in DCM and ICM groups. DCM, dilated cardiomyopathy; ICM, ischaemic cardiomyopathy.
In contrast, a positive response to therapy does appear to have a significant impact on appropriate ICD therapies. Therefore, the more pronounced reverse remodelling of HF in the DCM group compared with the ICM group likely explains the reduced incidence of ICD therapies in the DCM group.

**Study limitations**

This was a retrospective study with a small sample size. Furthermore, the two groups differed in baseline EF although all had a history of multiple hospitalizations for systolic HF. These important limitations do somewhat reduce the strength of the data and the results of our study can only be regarded as hypothesis-generating. Nevertheless, the magnitude of differences in a variety of outcomes after AVJ/Biv suggests a true differential effect of the procedure in patients with non-ischaemic and ischaemic cardiomyopathy. As indicated, the calculation of pre-procedure EF was not standardized. Pre-procedure EF was calculated in 16 of 45 by the Teicholz method, and in 29 of 45 by Simpson’s method. All post-procedure EF calculations were made using Simpson’s method and in cases where images were not of sufficient quality, patients were bought back and the TTE was repeated with contrast material, and were therefore standardized. Although the difference in techniques between pre-procedure and post-procedure echocardiograms in some patients may have affected the delta EF that was calculated, we do not feel that this limitation substantially alters the findings particularly since the relative proportions of patients in each group studied by the various methods was similar. Furthermore, the difference in delta EF between groups was reflected in the differences in post-procedural HFH and appropriate ICD therapies between groups.

A final factor which could have influenced TTE data is that, though the investigator analysing the TTEs was blinded to the clinical classification of each patient, ICM classification could potentially have been inferred by regional wall motion abnormalities or regional wall thinning in post-infarction patients.

A further limitation is that baseline EF was higher, and both LVEDD and LVESD were smaller in DCM compared with ICM patients (although these differences were not statistically significant). Thus, HF was probably not as severe at baseline in the DCM group as in the ICM group. We do not believe that the relatively small baseline difference between groups would account for the highly significant difference in reverse remodelling between groups in terms of HF outcomes and ICD therapies.

While we were able to detect a difference in appropriate ICD therapies between the DCM and ICM groups, the small numbers of patients in our study with a pre-existing ICD prior to AVJ/Biv upgrade did not allow us to compare appropriate ICD therapies before and after the procedure between groups, which would best be assessed with a prospective study.

Curative ablation therapy for long-standing persistent AF has progressed in terms of safety and efficacy in the time period after many of our patients underwent AVJ/Biv implantation/upgrade (2004 was the earliest date). Therefore, it is possible that some of the patients presented in this study could currently be offered AF ablation, which would have the advantage of restoring SR and obviate the need for AVJ and permanent pacing. Nevertheless, even with progress in the ablation of long-standing persistent AF, patients sometimes require more than one procedure, and a significant proportion of patients are left with AF after such procedures. Patients such as those presented in our study often require a more certain and timely approach to avoid repeated hospital admissions for HF and to improve quality of life. Furthermore, there were patients in our study who had opted for AVJ/Biv implantation/upgrade when given the choice of this vs. AF ablation.

All 45 patients except for one were male, and therefore the results of this study cannot necessarily be generalized to female patients although we do not think this would be relevant.

Finally, the baseline HRs for our patients were calculated from EKG and physical examination data only.

**Conclusions**

This is the first study to compare outcomes between patients with DCM and ICM largely without a de novo indication for CRT, after AVJ/Biv for AF associated with poor ventricular rate control, and refractory HF. We found a significant difference between these two patient groups with dramatic HF remodelling in the DCM group and little change in the ICM group. The difference between outcomes in these two groups can be explained by the presence of more confluent scar in patients with ICM, and the possibility that TIC plays a more dominant role in the generation of HF in patients with DCM and AF than those with ICM. A greatly reduced ventricular arrhythmia burden was noted in patients with DCM compared with ICM post-procedure. This is likely an effect of the greater degree of ventricular remodelling in this patient group.

**Conflict of interest:** none declared.

**References**

Successful management of a late right ventricular and diaphragmatic perforation by a defibrillator lead

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A 54-year-old man was submitted to replacement of an automatic defibrillator because of battery depletion. During implantation, a laceration of the defibrillator lead cover of was observed. The lead could not be extracted and was abandoned. A new defibrillation lead was actively fixated at the right ventricular apex. Two months after the implant, the patient reported thoracic pain. The physical exam was normal. The electrocardiogram evidenced failure of sensing and capture. The fluoroscopic left anterior oblique view showed cardiac perforation and migration of the new lead to the abdominal cavity (see Figure). The patient did not show pericardial effusion or signs of abdominal organ damage.

After informed consent, with surgery back-up under fluoroscopic and echocardiographic surveillance, the active fixation mechanism was retracted. The lead was carefully pulled back to the right atrium. The patient did not present pericardial effusion and was haemodynamically stable. The same lead was actively fixated in the right ventricular outflow tract. Impedances were normal and a correct device function was corroborated.

The patient remained stable and was discharged 2 days later. After a 9-month follow-up, he remains asymptomatic, has a stable position of the lead, and a normal device function.

The full-length version of this report can be viewed at: http://www.escardio.org/communities/EHRA/publications/ep-case-reports/Documents/management-right-ventricular-diaphragmatic-perforation-defibrillator-lead.pdf.