

# Left ventricular lead misplacement discovered a decade after cardiac resynchronization therapy-defibrillator implantation: a case report

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Introduction	Satisfactory left ventricular (LV) lead placement into the coronary sinus (CS) can be achieved in the majority of patients but there are still instances of acute failure most often due to anatomical differences, for example due to tortuous CS anatomy. Chronic LV lead misplacement and its delayed discovery is not a common scenario. It is unclear if chronic dual right ventricular pacing can hasten the progression of heart failure.
Case presentation	A 73-year-old lady presented to our cardiac centre with severe heart failure. She had non-ischaemic dilated cardiomyop- athy with underlying left bundle branch block and a cardiac resynchronization therapy-defibrillator device <i>in situ</i> for the past decade. She also had a chronic pericardial effusion of unknown aetiology. Whilst the patient was being treated for acute heart failure, it was noted on patient telemetry that the QRS morphology for supposed bi-ventricular pacing was unusual. This led to a lateral chest radiograph and a CS venogram to be performed, both of which confirmed that the LV lead was in fact not in the CS. Plans were made to place a new LV lead but unfortunately the patient continued to clinically deteriorate despite maximal treatment and died before this could be performed.
Discussion	It is only with thorough review of the electrocardiographic data and chest radiography that led to the discovery of chronic LV lead misplacement. This case illustrates the importance of expert review of radiographic imaging and electrocardiographic data in patients with implanted cardiac devices.
Keywords	LV lead • CRT-D • Heart failure • Chronic lead misplacement • Radiographic and electrocardiographic review • Case report

#### Learning points

- Expert radiographic review of patients with implanted cardiac devices is important. The lateral chest radiograph is especially informative on the course of the leads. In this case, if the lateral film was done earlier, it would have led to the earlier discovery of lead misplacement.
- Expert electrocardiographic review of patients with supposed bi-ventricular pacing is important to identify any cardiac device problems. In this case, the initial telemetry review identified an unusual bi-ventricular paced QRS morphology, which eventually led to the discovery of the left ventricular lead misplacement.

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#### Introduction

Cardiac resynchronization therapy (CRT) is an integral part of heart failure management in the appropriate setting.<sup>1</sup> Left ventricular (LV) lead placement into the coronary sinus (CS) is generally considered to be safe, with a high success rate at first procedural attempt. Chronic lead misplacement is rare.<sup>2</sup>

# Timeline

Time	Events
2008	Elective implantation of cardiac resynchronization therapy-defibrillator at another cardiac centre for non-ischaemic dilated cardiomyopathy and left bundle branch block.
Current admis	
Day 1	Acute admission to the heart failure unit with de-
Day	compensated heart failure.
Day 2	Pacing check 97% 'bi-ventricular' pacing.
Day 4	Consultant electrophysiologist review of patient
Duy	case, following observation of the unusual BiV-
	paced QRS morphology on patient telemetry.
	Chest radiograph review in AP and lateral views:
	the lateral film showed the left ventricular (LV)
	lead not going into the coronary sinus (CS). CT
	thorax from a few years earlier was reviewed
	which seemed to also show abnormal course of
	the CS lead, into the RV myocardium.
Day 5	A CS venogram confirming misplacement of the LV
	lead. (episode of pyrexia delayed plans for new
	LV lead placement).
Day 20	Further deterioration continued despite treatment.
	The deterioration was gradual prior to rapid de-
	velopment of cardiogenic shock and multi-organ
	failure. The patient was also being treated for
	Gram negative cocci bacteraemia.
Day 21	Admission to the intensive care unit for inotropic
	support and haemofiltration for related acute
	renal failure with hyperkalaemia.
Day 28	RIP.

#### **Case summary**

A 73-year-old lady was admitted from home directly to the heart failure unit with symptoms of gradually worsening breathlessness, over a duration of 4 weeks. The patient had a background of nonischaemic dilated cardiomyopathy, chronic pericardial effusion of unknown aetiology, severe LV systolic impairment (ejection fraction <35%), and left bundle branch block. She had CRT-defibrillator implantation at another cardiac centre in 2008. Regular medications included Bumetanide, Carvedilol, and weekly Metolazone. Prior to admission, there had been unsuccessful attempts at offloading by the Community Heart Failure Team. Clinical examination revealed tachypnoea at rest and coarse crepitations. On auscultation of the heart sounds there were pansystolic murmurs audible at both the lower left sternal edge and at the apex in keeping with tricuspid and mitral regurgitation. The jugular venous pulse was raised at >5 cmH<sub>2</sub>0 and there was bilateral pitting oedema above the knee levels. Vital signs were measured: Blood pressure 101/67 mmHg, heart rate 80 b.p.m. and oxygen saturations at 97% on room air. Acute management of heart failure was initially with administration of intravenous furosemide (240 mg/24 h infusion). Over time, there was clear resistance to treatment despite escalation in management.

A transthoracic echocardiogram showed that the LV systolic function had further deteriorated, with an ejection fraction of 10–15%, with no change in the size of the pericardial effusion (0.8–1.3 cm, anteriorly and posteriorly) and no associated haemodynamic impact. There was severe tricuspid and mitral regurgitation; this was a longstanding finding.

A device check showed apparent 97% bi-ventricular pacing with satisfactory parameters. The device was Boston Scientific, set at VVIR at base rate of 80. The right atrial lead sensing was at 0.3 mV and impedance was at 513 Ohms. There was underlying atrial fibrillation. The right ventricle (RV) lead sensing was at 14.1 mV, impedance at 884 Ohms, and the threshold was 1.3 V at 0.4 ms. The LV lead sensing was at 16.4 mV, impedance was 884 Ohms, and the threshold was 2.2 V at 1 ms.

During the electrophysiology team ward round, a telemetry review led to focus on this particular patient. The paced QRS morphology was atypical for bi-ventricular pacing. Electrocardiogram (ECG) during supposed bi-ventricular pacing showed a QRS duration of 174 ms in a right bundle branch block pattern. The intrinsic QRS duration was 196 ms (*Figure 1*). A postero-anterior chest radiograph was compatible with a satisfactory LV lead position but the lateral film showed clear malposition (*Figure 2*).

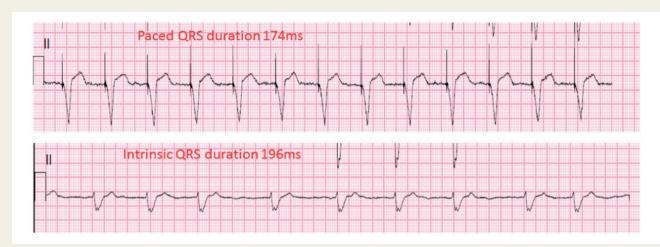
The following day, a CS venogram revealed a CS that was intact and confirmed that the LV lead did not pass through any part of the coronary venous system (*Figure 3*).

A review of a computerized tomography performed 3 years earlier (for a non-cardiac indication) showed that the lead was embedded in the right ventricular myocardium, breaching into the pericardium (*Figure 4*), an abnormality that had not been recognized by the reporting radiologist. It is possible then, that the misplaced LV lead is the cause of the chronic pericardial effusion. On serial echocardiographic imaging, the pericardial effusion had never been significant enough to require pericardiocentesis; it measured 0.9 cm anteriorly around the RV and 1.3 cm posteriorly.

The original implantation report revealed that the procedure was undertaken in an elective surrounding and no particular difficulty was documented. The LV lead was a Guidant 4555 Acuity Spiral<sup>®</sup> IS1 BI (Boston Scientific).

The patient was scheduled for placement of a new LV lead but the procedure was delayed due to an episode of pyrexia. The plan was to leave the chronically misplaced LV lead *in situ* as the risk of cardiac perforation and death, with attempted extraction would be extremely high.

The patient gradually further deteriorated before a rapid descent into cardiogenic shock. Inotropic support and haemofiltration was



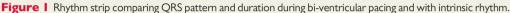




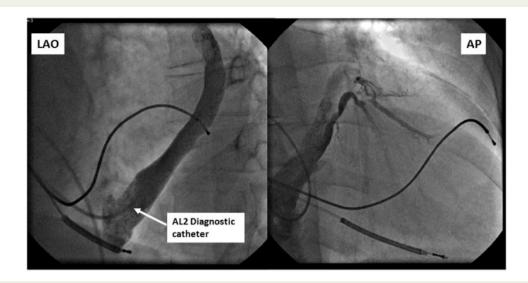
Figure 2 AP and lateral chest radiographs. The 'left ventricular lead' is situated, coiled into RV myocardium, close to the epicardial surface.

given in an intensive care setting. She was also treated for Gram negative cocci bacteraemia. The patient was eventually palliated before her unfortunate but expected death.

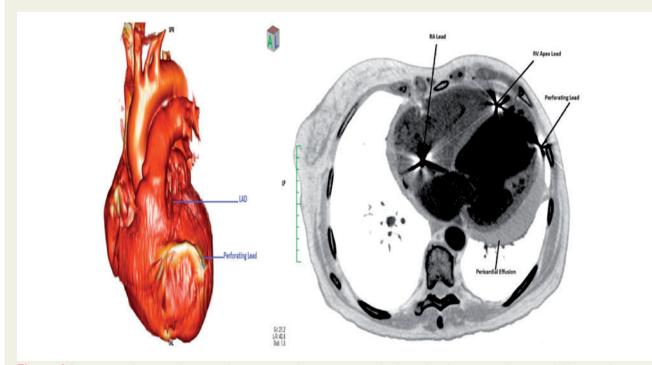
# Discussion

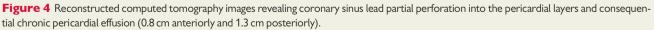
Satisfactory LV lead positions can be obtained via the coronary venous system in >99.8% of patients.<sup>3</sup> It is not clear why the malpositioning occurred or why it was not recognized and corrected. The original device implantation report did not reveal any unusually challenging circumstances. A CS venogram had been undertaken at the time of implant, and it was believed that the LV lead was deployed into a branch of the CS. It is most likely that the lead was misplaced and was somehow not recognized. The AP fluoroscopy projection, alongside a dilated and rotated cardiomyopathic heart would certainly give the false impression that there was satisfactory LV lead placement. In addition, the LV lead parameters have always been satisfactory, giving no cause for a review into the LV lead integrity or position. The other possibility is that the lead had completely displaced later on after implant but this reason is less plausible, given the position of the lead and no recorded interval change in lead appearance from historical imaging.

The LV lead used was a Guidant 4555 Acuity Spiral<sup>®</sup>. This lead is not known to be problematic at implant or during follow-up, when compared with similar generation modern LV leads. Steffel et al.,<sup>4</sup> conducted a retrospective study of long-term



**Figure 3** A coronary sinus venogram using AL2 diagnostic catheter via right femoral vein. The coronary sinus is intact with good lateral branches and confirms that the 'left ventricular lead' is not in the coronary sinus but embedded in the RV myocardium.





performance of modern CS leads (a range of leads, with implant dates from 2003–10) and found there was macro-displacement in 3.6% of 193 patients, across a median of 111 days. There were no cases of misplacement. Overall, modern leads tend to have a good stability profile at follow-up with low rates of macro or micro displacements.<sup>5,6</sup>

It cannot be objectively proven if dual right ventricular pacing actively accelerated the progression of this patient's heart failure. However, given our knowledge of the potential harm of chronic right ventricular pacing<sup>7,8</sup> and the fact that this patient had underlying severe non-ischaemic dilated cardiomyopathy, it is likely that dual right ventricular pacing had contributed to her chronically sub-optimal functional status in addition to worsening cardiac function. Providencia *et al.*<sup>9</sup> recently published a study looking at the possibility of dual-site right ventricular pacing for patients with heart failure, after failure to deliver a CS lead. Earlier reports suggested this alternative possibility.<sup>10</sup> The dual RV group had worse clinical outcomes compared to the matched control group with higher rates of all-cause mortality and heart transplantation.

In the 10 years that passed, it is only with retrospective review of patient ECG and imaging that we have found that the evidence of LV lead misplacement was there all along but not previously recognized. This patient only had a lateral chest radiograph after we requested this, to confirm the suspicion. Her previous films in the AP or PA projection may have given the false impression of a satisfactory lead position. Cardiac resynchronization therapy optimization clinic assessments did not identify the problem with the LV lead.

# Conclusion

A review of the patient telemetry eventually led to the unexpected discovery of chronic LV lead misplacement. This case illustrated the importance of expert review of radiographic and electrocardiographic data in patients with implanted cardiac devices.<sup>11,12</sup>

**Consent:** The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the next of kin of the deceased patient has given consent for this anonymised case report to be written and published.

Conflict of interest: none declared.

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