

Retrograde venography to navigate an occluded subclavian vein to achieve cardiac resynchronization therapy upgrade via His bundle pacing: a case report

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Background

Guidelines support upgrade to cardiac resynchronization therapy (CRT) through His-bundle pacing (HBP) in pacing-induced cardiomyopathy and moderate left ventricular systolic dysfunction (LVSD). Lead-related venous occlusion can represent an obstacle to upgrade procedures. We describe a technique to overcome venous occlusion through direct puncture of a collateral vein facilitating upgrade to HBP.

Case summary

An 84-year-old man with a right ventricular (RV) pacemaker was referred with New York Heart Association (NYHA) Class III breathlessness secondary to moderate LVSD (left ventricular ejection fraction [LVEF] 45%). Device interrogation revealed 100% RV pacing and AV-dyssynchrony. To optimize atrioventricular (AV) and interventricular (VV) synchrony a CRT upgrade with HBP was planned. Venography revealed an occluded left subclavian vein which was probed in a retrograde manner using a 6F MPA catheter from right femoral venous access. We were able to direct the catheter distal to the left brachio-cephalic vein and define the occlusion using contrast. A collateral branch was identified, a J-wire was left in this branch and venous access was secured at this medial subclavian site using the Seldinger technique. A right atrial lead was deployed and 69 cm ISI-1 His lead was deployed via a C315 sheath at the His-bundle. The resulted in non-selective HBP (Stim-QRS end 146 ms). There were no procedural complications. Two months later both symptoms and LV function (LVEF 55%) improved.

Discussion

Lead-related venous occlusion occurs frequently and can be probed in a retrograde manner from femoral venous access using contrast, facilitating direct percutaneous puncture of collateral venous branches to allow upgrade to CRT via HBP.

Keywords

His-bundle pacing • Venous occlusion • Cardiac resynchronization therapy • Case report

ESC Curriculum

5.11 Cardiac resynchronization therapy devices • 5.9 Pacemakers • 6.1 Symptoms and signs of heart failure • 6.2 Heart failure with reduced ejection fraction

Learning points

- Retrograde contrast injections can be used to define a pacing lead-related venous stenosis and identify potential collateral branches.
- These collateral branches can be punctured directly thus bypassing the stenosis and facilitates new transvenous lead placement.
- Cardiac resynchronization therapy may be achieved through His-bundle pacing leading to reversal of pacing-induced cardiomyopathy.

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Introduction

Pacing-induced cardiomyopathy (PIC) occurs in ~20% of cases with observational data demonstrating an increased risk in those with an RV pacing burden >20%.¹ In patients with a conventional pacemaker to treat bradyarrhythmias and who develop symptomatic heart failure with an LVEF of ≤35% despite guideline-directed medical therapy, and who have an RV pacing burden >20%, device upgrade to deliver cardiac resynchronization therapy (CRT) with an additional LV lead is recommended.²

Conduction system pacing has expanded in recent years and involves targeting either the bundle of His or the more distally located left-bundle area. Whilst randomized controlled trials with large patient numbers comparing His-bundle pacing (HBP) with RV and CRT pacing are awaited, observational studies have shown a promising reduction in heart failure hospitalizations and preservation of LVEF with HBP compared with RV pacing in patients with atrioventricular block (AVB).^{3,4} Recent ESC guidelines support HBP as an alternative to RV pacing in patients with (AVB) and an LVEF >40% with an expected ventricular pacing burden >20%.² The AHA guidelines similarly support HBP in patients with moderate LV impairment (LVEF 36–50%) with AVB with an expected ventricular pacing burden >40%.⁵

Prior to device upgrade, contrast venography is performed in order to assess vein patency as lead-related venous occlusion can occur in up to 50% of patients.⁶ Laser-lead extraction and balloon venoplasty are techniques used to overcome such occlusions but require specialist training and increase procedural risk. We describe a case of suspected PIC and lead-related subclavian vein (SCV) occlusion which was probed in retrograde manner from femoral venous access using contrast and facilitated direct percutaneous puncture of a collateral venous branch. Cardiac resynchronization therapy was then delivered through HBP.

Timeline

9 years prior to review	Third-degree atrioventricular block in sinus rhythm with single-chamber (RV) pacemaker implant at another institution. left ventricular ejection fraction >55%
DAY 0	Patient referred with NYHA III heart failure symptoms. Left ventricular systolic dysfunction on echocardiography—ejection fraction 40–45% RV pacing 100% on pacing check Loss of AV synchrony on 12-lead electrocardiogram
Day 30	Left-sided venography demonstrated lead-related subclavian vein occlusion Patient offered upgrade to cardiac resynchronization therapy with either conventional LV lead or His-bundle pacing
Day 65	Device upgrade—venous occlusion probed in a retrograde manner from femoral venous access. Collateral branch identified and punctured directly allowing placement of a right atrial and His-bundle lead
Day 120	Non-selective HIS capture confirmed. NYHA Class 1 symptoms and improvement in left ventricular ejection fraction to >55%

Case presentation

History of presentation

An 83-year-old man with a history of third-degree AV block and a single-chamber (RV lead) permanent pacing system (*in situ* 9 years) was referred to our institution with worsening breathlessness (NYHA III). There were no other heart failure symptoms, and clinical examination revealed signs of fluid overload (elevated jugular venous pressure and bilateral lower leg oedema) with no murmurs on auscultation.

Past medical history

This included an ablation for cavo-tricuspid isthmus dependent flutter in 2009, and mild chronic obstructive pulmonary disease. There was a remote history of alcohol excess and transthoracic echocardiography at the time of original device implantation showed preserved left ventricular systolic function (LVEF 55%).

Drug therapy included Ramipril 10 mg o.d., Spironolactone 25 mg o.d., Bendroflumethiazide 2.5 mg o.d., Simvastatin 40 mg o.d., Omeprazole 20 mg o.d., Tiotropium 18 µg o.d., Symbicort Turbohaler 200 µg/6 µg 1 puff BD, and Warfarin according to internal normalized ratio. Betablockers had not been commenced due to concerns about airways disease with a degree of reversibility on pulmonary functional testing.

Investigations

Twelve-lead electrocardiogram (ECG) showed sinus rhythm with AV dissociation because of RV-only pacing (*Figure 1*). Device interrogation confirmed 100% RV pacing with underlying complete AV block with a broad escape rhythm (left bundle branch block [LBBB], QRS duration 132 ms). Transthoracic echocardiography revealed a non-dilated LV with moderately impaired systolic function (LVEF 40–45%) with evidence of dyssynchrony (apical rocking and early septal activation) consistent with RV apical pacing (see [Supplementary material online, Video S1](#)). RV function and pulmonary pressures were normal, with no significant valve abnormalities.

Differential diagnosis

The most likely diagnosis explaining this presentation was PIC, differentials included alcohol-induced cardiomyopathy, or post-ablation atrial arrhythmia.

Management

Despite guideline-directed medical therapy his heart failure symptoms persisted. Considering the LV impairment and dyssynchronous ventricular activation, he was offered a device upgrade to a CRT-P with either a conventional LV lead or a His-bundle lead, in addition to a right atrial (RA) lead to improve VV and AV synchrony, respectively. Prior to implantation, left-sided venography was performed to evaluate vein patency which showed an occluded SCV (*Figure 2*). Options considered included the following: (i) a contralateral CRT-P implantation; (ii) implanting a right-sided RA and LV lead and tunnelling across to the left; (iii) venoplasty facilitated CRT upgrade; (iv) lead extraction of the functional RV lead for recanalization and upgrade; (v) surgical epicardial lead placement; and (vi) a conservative approach. After careful discussion, the patient elected to proceed with an upgrade strategy (including venoplasty and lead extraction). An epicardial approach was felt too high risk given his co-morbidities.

As a centre with experience in HBP, and in alignment with recent guidelines,^{2,5} we targeted CRT-P via this approach. The least aggressive strategy was preferable and therefore prior to opening the left-sided pocket, the SCV occlusion was probed in a retrograde manner using



Figure 1 Twelve-lead electrocardiogram showing sinus rhythm with AV dissociation because of RV-only pacing.

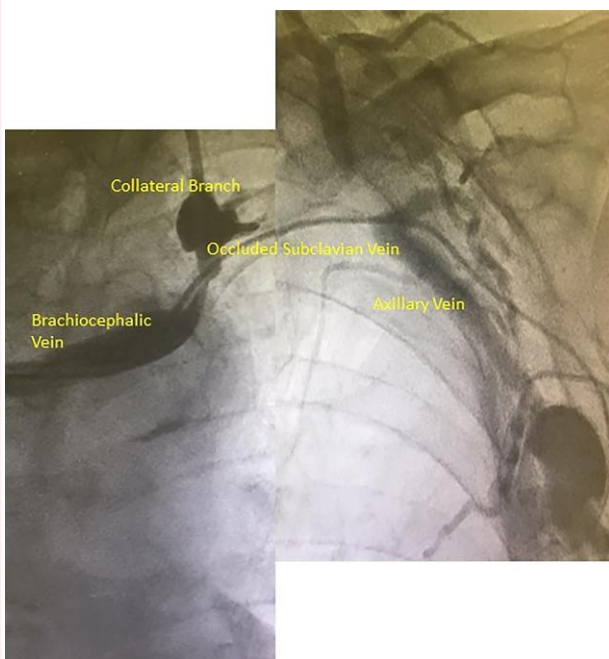


Figure 2 Composite image showing left-sided contrast venography demonstrating lead-related left subclavian venous occlusion. Retrograde venography via femoral venous access delineates site of vessel recanalization.

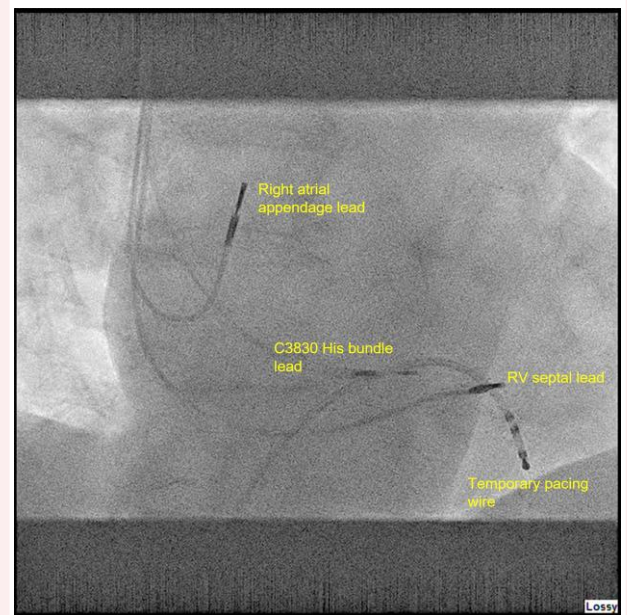


Figure 3 Fluoroscopy still demonstrating final lead position. Original RV lead in the septal position, C3830 His lead at the bundle of His, RA lead in the appendage, and a temporary pacing wire are highlighted.

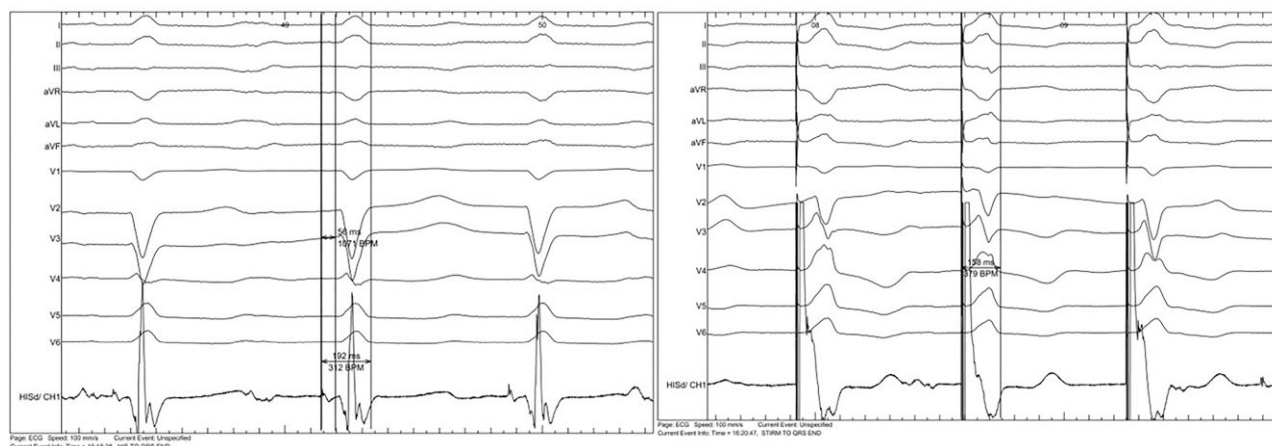


Figure 4 Intracardiac electrograms confirm supra-Hisian AV block (HV association maintained) and evidence of His injury current (left panel). Intrinsic His-QRS_{end} interval 192 ms (left panel). With His pacing stim-QRS_{end} reduced to 158 ms (right panel).

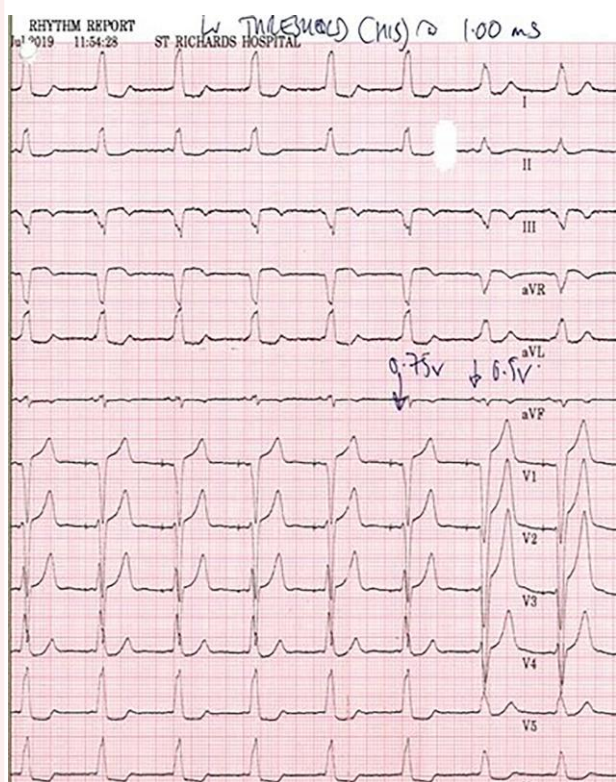


Figure 5 Twelve-lead electrocardiogram showing non-selective HIS capture with QRS narrowing at 0.75 V output and pulse width 1.0 ms and RV myocardial capture at 0.5 V output.

a multipurpose catheter (6F MPA1 Impulse, Boston Scientific, MA, USA) from right femoral venous access (secured for temporary pacing wire support). In doing so, we were able to direct the catheter to the brachio-cephalic vein and define the occlusion in detail. Furthermore, contrast highlighted a large collateral branch, which we felt could be punctured directly from a left sub-clavicular approach (see

Supplementary material online, Video S2). We left a 0.035 in J-wire in this branch, and successfully secured venous access through the Seldinger technique (see Supplementary material online, Video S3). A passive lead to the right atrial appendage was implanted and a 69 cm Select Secure 3830 lead (Medtronic Inc., MN, USA) was deployed via a C315 sheath (Medtronic) at the His-bundle. Final lead positions are shown in Figure 3.

Intracardiac electrograms confirmed underlying complete AV dissociation with an intrinsic His-QRS_{end} interval of 192 ms. With HBP, non-selective capture was achieved resulting in a shortened Stim-QRS_{end} of 158 ms with a threshold of 1 V at 0.5 ms (Figures 4 and 5). The device was programmed DDD-60 with an 'LV' to RV delay of 60 ms, to allow protective back up RV pacing in the event of loss of His capture. There were no complications and post procedural 12-lead ECG confirmed non-selective His capture.

Follow-up

After 2 months, the patient was NYHA I, euvoalaemic and LV systolic function had normalized (LVEF >55%, see Supplementary material online, Video S4). Device interrogation revealed atrial pacing of 56% and HBP 99% of the time.

Discussion

Venous occlusion is a well described complication after transvenous lead placement with an estimated prevalence of 20–26% depending on the time since implantation.^{6,7} In one observational study of 212 patients attending for replacement generator, device upgrade or lead revision, advancing age and the number of transvenous leads *in situ* were predictors of total venous occlusion.⁶ Balloon venoplasty and laser-lead extraction techniques have been developed to overcome these occlusions but carry with them inherent procedural risks, additional expense and need for specialist training.

Whilst retrograde contrast injections to define a venous stenosis has previously been described, its use has been to facilitate bypassing a stenosis with guidewires and ultimately performing venoplasty to allow percutaneous transvenous lead placement.⁸ We believe this is the first description of direct percutaneous venous puncture using this technique, avoiding the need for venoplasty or laser-lead extraction, thereby lowering procedural risks. Whilst in the present case no procedural complications occurred, direct puncture of a collateral branch would carry a risk of pneumothorax similar to SCV puncture.

Pacing-induced cardiomyopathy has been estimated to occur in 10–20% of individuals with normal baseline LV function receiving a high RV pacing burden.¹ RV pacing results in electrical and mechanical dyssynchrony⁹ and chronic RV pacing is associated with an increased risk of heart failure, atrial fibrillation and death.¹⁰ Compared with RV pacing, CRT with biventricular pacing (BVP) improves dyssynchrony, however, it does not restore normal ventricular activation.^{11,12} His-bundle pacing enables ventricular activation through the direct stimulation of the His-Purkinje system. This expanding technique results in more physiological ventricular contraction when compared with BVP¹¹ with reassuring safety and longer-term outcomes.¹³ Results from the HOPE-HF trial in which 160 patients with AV delay (PR prolongation >200 ms), LVEF <41% and either QRS duration (<141 ms) or right bundle branch block were randomized to receive either HBP or back up pacing are eagerly awaited.

To reduce the risk of PIC the latest ACC/AHA pacing guideline give HBP a Class IIa recommendation in patients undergoing pacemaker implantation for AV block, with mild-moderate LV impairment (LVEF 36–50%) with an expected RV pacing burden >40%.⁵ The ESC gives a IIb recommendation for those patients anticipated to have >20% ventricular pacing and moderate LV impairment.

Conclusions

The management of patients with PIC includes pacemaker upgrade to deliver CRT. Lead-related venous occlusion occurs frequently in clinical practice and can be probed in a retrograde manner from femoral venous access using contrast, facilitating direct percutaneous puncture of collateral venous branches. Cardiac resynchronization therapy can be successfully delivered through HBP.

Lead author biography



Dr Christopher Pavitt is a higher specialist cardiology trainee in the United Kingdom and is currently completing his medical doctorate at the Sussex Cardiac Centre, Brighton. His subspecialty interests are heart failure, complex pacing, and imaging.

Supplementary material

[Supplementary material](#) is available at *European Heart Journal – Case Reports*.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

Consent: In accordance with COPE guidelines the patient described in this article consented to the anonymized publication of their case for education and research purposes.

Conflict of interest: None declared.

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