Obstacles preventing biventricular pacing mitigated with lead extraction and His bundle pacing to achieve effective cardiac resynchronization

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Introduction

Cardiac resynchronization therapy (CRT) achieved by implanting a lead via the coronary sinus (CS) system significantly improves well-being, symptoms, morbidity, and mortality in appropriately selected populations with advanced heart failure (HF) or pacing-induced cardiomyopathy.[1–4] Despite improvements in implant technique, lead design, and pacing algorithms, there still remains a significant nonresponder rate, high implant failure, and higher risk of complications.[5–7] The options for CRT are generally limited to surgical lead placement if lead delivery via the CS is not feasible.[8] His bundle pacing (HBP) engages and recruits the native His-Purkinje system distal to the level of block allowing for rapid and coordinated electromechanical ventricular activation avoiding dyssynchrony with hemodynamics and remodeling similar or superior to CRT via the CS.[9–14] Despite the potential for superior CRT and less complex procedures, HBP is underutilized in cases where CRT via the CS is not feasible. We present a case with several obstacles to CRT that was ultimately achieved with HBP.

Case report

A 75-year-old woman with diabetes mellitus, hypertension, dyslipidemia, and coronary artery disease who previously underwent 3-vessel coronary artery bypass grafting >10 years ago, and sinus node dysfunction requiring implant of a left-sided dual-chamber pacemaker in 2010, was referred in February 2017 for CRT in the setting of New York Heart Association (NYHA) functional class III HF due to ischemic cardiomyopathy (left ventricular ejection fraction [LVEF] 30%–35%) and 100% ventricular pacing.

KEYWORDS His bundle pacing; Cardiac resynchronization therapy; Heart failure; Heart block; Pacing-induced cardiomyopathy; Laser lead extraction; Pacemaker; Defibrillator

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At the time of initial device implantation in 2010, right bundle branch block was present with a PR interval of 158 ms and a QRS duration (QRSd) of 128 ms. By 2013, the patient was pacemaker dependent with underlying complete heart block and no stable escape rhythm >30 beats/min, presumably because of progressive conduction disease. In June 2016, she experienced a myocardial infarction requiring intervention of the saphenous vein graft to diagonal with placement of a drug-eluting stent. The LVEF was 25% after myocardial infarction and improved to 35% within 1 month of revascularization and medical optimization. Over the subsequent months, the patient developed progressive dyspnea (NYHA functional class IIIb) despite optimal medical therapy. Echocardiography revealed mild aortic stenosis and an LVEF of 30%. Coronary angiography in February 2017 revealed severe native 3-vessel coronary artery disease with patent bypass grafts. Medications included aspirin, clopidogrel, metoprolol succinate, lisinopril, torsemide, and atorvastatin. The pacemaker system was composed of a Adapta DR pulse generator (Medtronic, Minneapolis, MN), a 52-cm CapSureFix Novus (model no. 5076) active fixation lead (Medtronic, Minneapolis, MN) in the right atrial (RA) appendage, and a 58-cm CapSure SP Novus (model no. 4092) passive fixation lead (Medtronic, Minneapolis, MN) in the right ventricular (RV) apex.

The patient had a class IIa indication according to the 2012 ACCF/AHA/HRS guidelines for CRT with defibrillator therapy, given anticipated pacing in excess of 40%. The underlying QRS morphology and duration were unable to be assessed because of complete heart block. An echocardiographic assessment of ventricular dyssynchrony was not performed. Device upgrade would require insertion of a new RV defibrillator lead with or without extraction of the chronic RV pacing lead. The patient opted to avoid extraction and abandon the chronic RV pacing lead. A preimplant peripheral venogram (Figure 1) demonstrated a subtotally occluded subclavian vein with distal reconstitution primarily via bridging collaterals. At this point, the clinical considerations were to perform a deep subclavian puncture, venoplasty, or laser lead extraction. Laser lead extraction was considered the best option in light of the short-occluded segment, prior

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KEY TEACHING POINTS

- The current endovascular approach for cardiac resynchronization via the coronary sinus is limited by anatomical constraints precluding targeting the optimal pacing site, suboptimal resynchronization with a high nonresponder rate, and risk of failure or complications.
- Surgical left ventricular lead placement is used as rescue therapy but has similar limitations and outcomes with a more invasive procedure and higher risks.
- His bundle pacing offers an alternative approach to achieve cardiac resynchronization in technically challenging cases where the standard endovascular approach via the coronary sinus is not possible.
- His bundle pacing should be attempted in patients before considering epicardial lead placement.

coronary artery bypass grafting, pacing leads of <7 years, and ability to upgrade to a magnetic resonance imaging (MRI) conditional system. Venous intervention was thought to further risk vascular overload.

The patient was rescheduled for laser lead extraction 8 days later. Temporary backup pacing was established via the right femoral vein. The pocket was opened, and a partial capsulectomy was performed. The chronic RV pacing lead was firmly adherent at the level of the subclavian vein. The lead was cut, and a Spectranetics LLD EZ locking stylet was advanced to the tip of the RV lead. Laser lead extraction was performed successfully, and the lead removed fully intact with 5 applications of the laser at the level of the subclavian vein and 1 application at the superior vena cava. There was no hemodynamic instability or evidence of pericardial effusion. Venous access was retained through the laser sheath with 2 wires. A 55-cm Medtronic Sprint Quattro Secure (model no. 6935M) single-coil defibrillation lead was advanced to the RV apex where pacing impedance was 610 Ω, shock impedance 62 Ω, and threshold 0.5 V at 0.5 ms. The chronic RA lead was interrogated and demonstrated stable parameters with sensing of 1.9 mV, impedance 382 Ω, and threshold 0.5 V at 0.5 ms. The CS was cannulated with difficulty owing to a prominent Thebesian valve with a Medtronic CS-EH lead delivery sheath and steerable decapolar catheter. A balloon occlusive CS venogram demonstrated a small anterolateral branch and small and stenotic posterolateral branch (Figure 1). Multiple attempts at delivering low profile quadripolar and bipolar CS leads were unsuccessful in either branch. The only remaining branch was the anterior interventricular vein and was not considered a suitable target. In the absence of the ability to place a CS lead, epicardial left ventricular (LV) lead placement or HBP was then considered.

A Medtronic C315-His lead delivery sheath was advanced to the RA, and a 4-F Medtronic SelectSecure (model no. 3830) lead was advanced to the superior tricuspid annulus. A His bundle potential was unable to be visualized because of complete heart block. The pacing morphology was obtained from several sites along the superior tricuspid annulus to obtain the narrowest paced QRS complex with nonselective His bundle capture where the lead was torqued to affix the lead (Figures 2 and 3). An acute hemodynamic response was observed with systolic blood pressure increasing 10 mm Hg during HBP compared to RV apical pacing. The leads were connected to a Medtronic Claria MRI biventricular implantable cardioverter-defibrillator, with the His bundle lead connected to the LV port with a pacing configuration of LV_{tip} to RV_{coil} and LV offset programmed to −80 ms to maximally preexcite the His-Purkinje system and prevent fusion with RV apical pacing as the RV septum is likely to be refractory. The His capture threshold measured 2.8 V at 0.5 ms with an RV capture threshold of 0.5 V at 0.5 ms, pacing impedance 580 Ω, and paced and sensed atrioventricular delays 130 and 100 ms, respectively. The RV defibrillation lead output was set at 3.5 V at 0.5 ms to ensure capture while

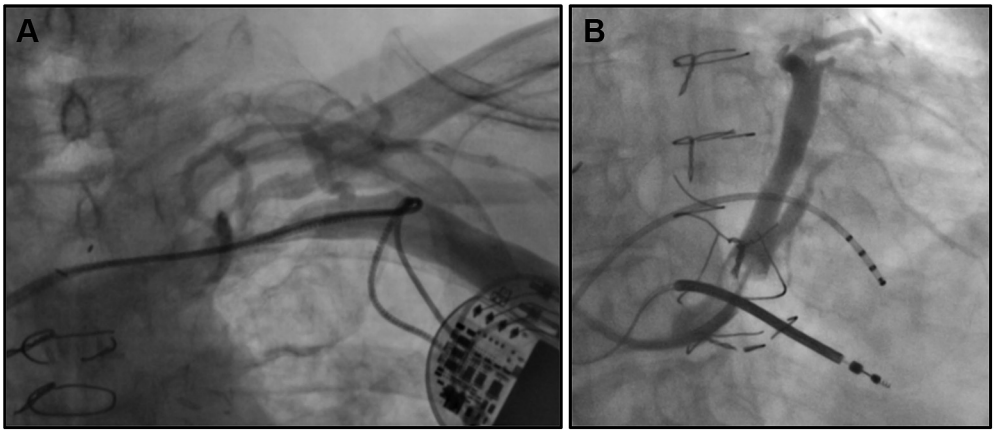

Figure 1 **A:** A peripheral venogram demonstrating a subtotally occluded left subclavian vein with distal reconstitution primarily via bridging collaterals (anteroposterior projection). **B:** A balloon occlusive coronary sinus venogram revealing a small and stenotic posterolateral branch and a smaller anterolateral branch into which leads were unable to delivered (right anterior oblique projection).

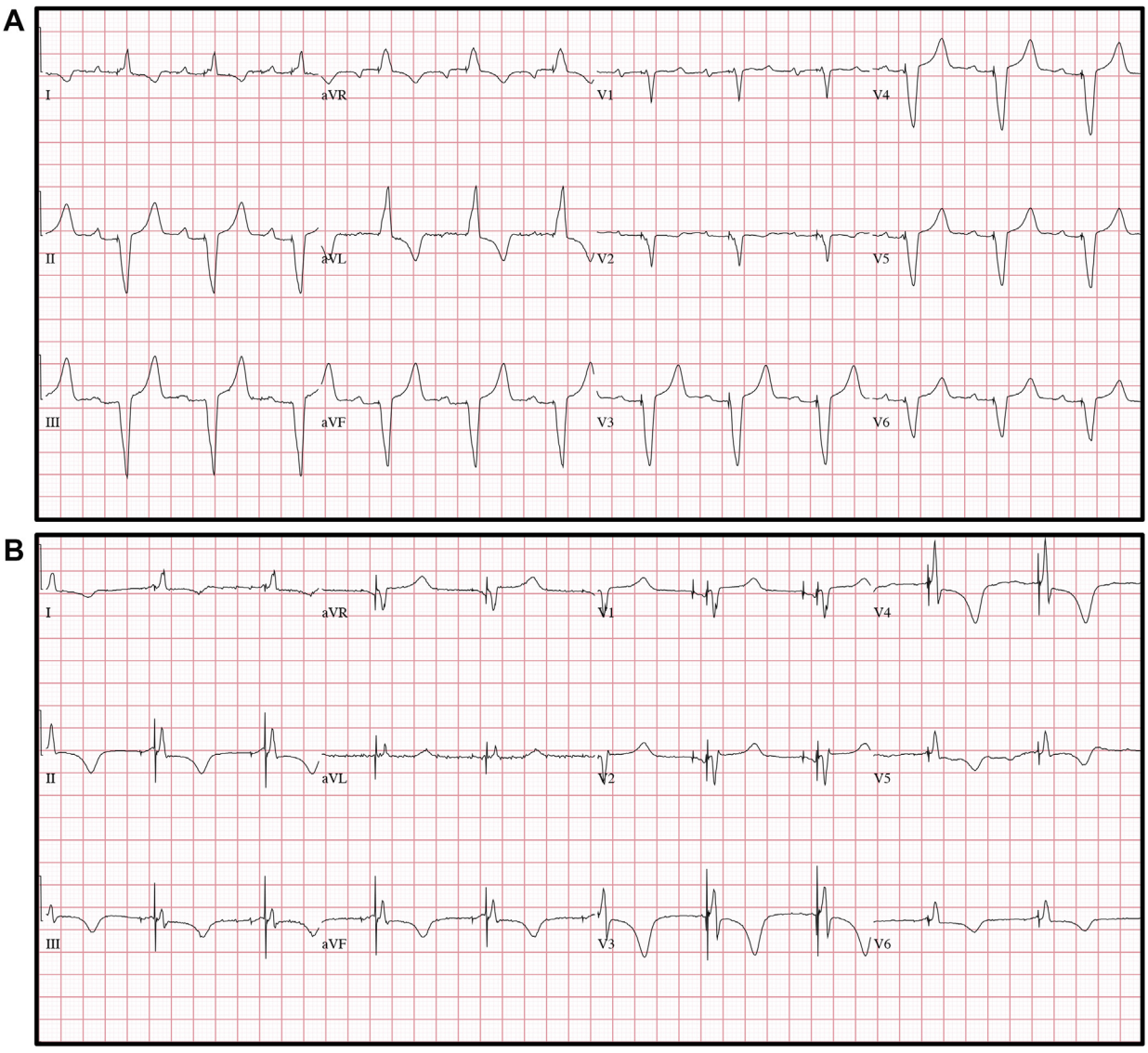

Figure 2 Twelve-lead electrocardiogram demonstrating the pre (**A**) and post (**B**) His bundle pacing QRS morphologies. The QRS duration in lead V_1 with His bundle pacing measures 84 ms.

monitoring His bundle capture stability. Postshock pacing was enabled and set at 8 V at 0.5 ms. Bradycardia settings were programmed to DDDR with a lower rate limit of 60 beats/min with adaptive CRT off. Tachyarrhythmia therapies were programmed according to Multicenter Automatic Defibrillator Implantation Trial–Reduce Inappropriate Therapy (MADIT-RIT), with a ventricular tachycardia monitor zone from 171 to 200 beats/min and ventricular fibrillation zone >200 beats/min treated with antitachycardia pacing during charging and full output defibrillation. The total fluoroscopy time was 47 minutes.

Upon 1 month follow-up, the patient has improved to NYHA functional class II, with reduced fatigue and dyspnea as well-marked improvement in exercise tolerance. LVEF improved to 45% after 2 months of HBP. HBP thresholds improved to 1.5 V at 0.5 ms and pacing output set at 3 V at 0.5 ms. The RV defibrillation lead output was programmed to threshold.

Discussion

The clinical benefits of CRT are well established by several randomized clinical trials in 2 populations of patients: those with advanced cardiomyopathy with left bundle branch block[1–3] and those with pacing-induced cardiomyopathy.[4] CRT improves quality of life, exercise tolerance, HF admissions, and mortality.[1–4] In both cases, dyssynchronous ventricular activation is the putative pathophysiologic mechanism that leads to detrimental hemodynamics and adverse remodeling. CRT via the CS exhibits significant heterogeneity in clinical response and up to one-third of the patients do not benefit.[5] Improvements in CRT have primarily focused on defining and targeting the optimal pacing site via the CS through improvements in lead design and delivery systems but significant challenges remain. Approximately 25% of CRT implants are device upgrades associated with the risks of lead abandonment or encountering venous occlusion.[15] Lead abandonment increases risk of infection, venous

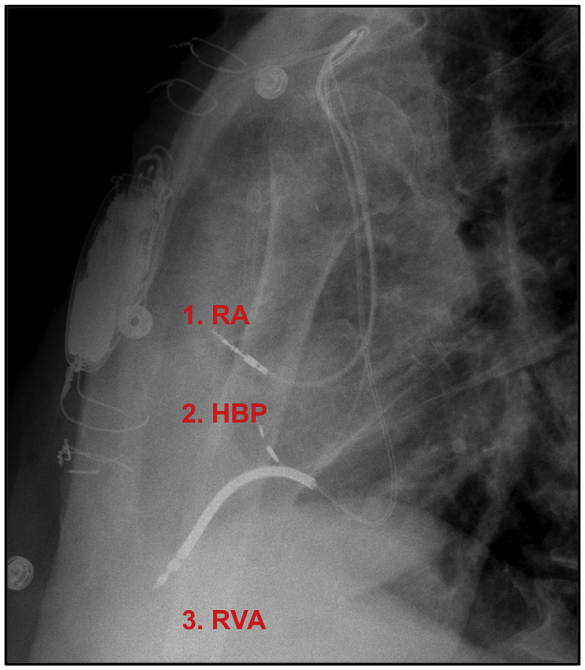

Figure 3 Lateral chest radiograph revealing the positions of the 3 leads: (1) right atrium (pacing lead), (2) His bundle region (pacing lead), and (3) right ventricular apex (implantable cardioverter-defibrillator lead).

obstruction, more complex future extractions, and MRI restriction. Total venous occlusion may be found in nearly 1 in 4 patients but is only clinically apparent in ~5% before device upgrade or lead revision.

Despite several different techniques and a multitude of specially designed tools for CS cannulation or lead delivery, up to 10% of attempts fail[3] because of anatomical constraints such as prominent Thebesian valve or valve of Vieussens, CS stenosis or tortuosity, or small or absent branches. If these anatomical barriers are overcome, there remains the issue of suboptimal pacing characteristics (high thresholds and latency) and phrenic nerve stimulation. These technical challenges lead to prolonged procedure time, greater radiation exposure, greater exposure to iodinated contrast, greater risk of CS dissection or perforation, and a relatively high rate of lead migration or dislodgment needing early intervention.[7] Surgical epicardial LV lead placement is the traditional approach if CS lead delivery is not possible but is associated with significant risks due to intubation, anesthesia, single lung ventilation, and thoracotomy; however, clinical response is not superior.[8]

Normalization of bundle branch block with temporary HBP was described in 1977, but Deshmukh et al[9] in 2000 first reported the feasibility of permanent HBP with narrow QRSd. Lustgarten et al[10] in 2010 demonstrated that temporary HBP, instituted at the time of biventricular pacemaker insertion, elicited a significantly shorter QRSd, and Barba-Pichardo et al[13] achieved HBP with high success when CRT via the CS was not successful. HBP preserves synchrony and myocardial systolic and diastolic performance when compared to RV apical pacing. Vijayaraman and coworkers demonstrated that HBP is highly successful in the setting of heart block[12] and superior to RV apical pacing in regard to HF hospitalization.[13] Lustgarten et al[14] subsequently demonstrated in a crossover trial that HBP improves quality of life, 6-minute walk, functional class, LVEF, and cardiac structure. The pacing characteristics of this lead are known to be stable over at least 2 years but are associated with a 5%–16% rate of lead complications including inability to deploy lead, inability to shorten QRSd (20%–35%), high thresholds, dislodgment, and lead revision.

Conclusion

This case demonstrates that there are several obstacles to achieving CRT and that despite significant improvement in implant technique and tools, CRT is associated with a significant risk of failure. Failure of CRT via the CS typically leads to surgical LV lead placement with attendant risks or abandoning CRT. HBP allows for normal electromechanical LV activation and may be achieved in the setting of advanced conduction disease. While HBP clearly has challenges, it offers the possibility of more physiologic pacing, thus avoiding or improving dyssynchrony, and should be attempted as rescue therapy if CRT via the CS fails before considering surgical lead placement.

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