

Urgent left bundle branch pacing for heart block and cardiogenic shock, facilitating percutaneous mechanical circulatory support removal



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Introduction

Acute myocardial infarction with cardiogenic shock and heart block may require mechanical support and pacemaker placement. Atrioventricular and interventricular dyssynchrony with right ventricular (RV) pacing may worsen cardiac output. We describe a case of urgent placement of dual-chamber left bundle branch (LBB) pacemaker in such a patient, thereby allowing Impella removal.

Case report

A 77-year-old man with hypertension presented with 2 days of dyspnea at rest and exertion without chest pain. In the emergency department, his heart rate was 86 beats per minute, blood pressure was 138/89 mm Hg, respiratory rate was 22 breaths per minute, and oxygen saturation was 96% on 3 L/min of oxygen. Cardiopulmonary examination was notable for intermittent cannon a waves and bilateral crackles on lung auscultation. Laboratory values were notable for white blood cell count of 32.79 K/ μ L, platelet count of 632 K/ μ L, and creatinine 2.63 mg/dL. High-sensitivity troponin was >10,000 ng/L, and proBNP was 56,368 pg/mL. The initial electrocardiogram (ECG) revealed complete heart block (CHB), junctional escape with right bundle branch block (RBBB), and ST elevations in inferior leads and V₁–V₃ (Figure 1A); however, shortly afterwards, the QRS changed to a left bundle branch block (LBBB) with QRS duration of 174 ms (Figure 1B). Transthoracic echocardiogram revealed an ejection fraction of 25% with severe hypokinesis of the anterolateral, anteroseptal, and inferoseptal walls. Intravenous heparin and dobutamine were started, and the patient was taken for emergent coronary

KEY TEACHING POINTS

- Left bundle branch pacing (LBBP) may be a useful tool in restoring atrioventricular and interventricular synchrony even when conduction block or delay is distal to the His bundle.
- Heart block and dyssynchrony have a significant contribution to refractory cardiogenic shock.
- LBBP may be used in critically ill patients with renal disease to avoid the use of intravenous contrast often required for cardiac resynchronization therapy pacing.

angiography, which showed a 100% stenosis of the proximal left anterior descending artery. A 3.5 mm \times 8mm synergy drug-eluting stent (Boston Scientific, Marlborough, MA) was successfully deployed, with <1% residual stenosis (Figure 2).

Owing to worsening bradycardia with CHB, a temporary transvenous pacemaker was placed via the right femoral vein. A pulmonary artery catheter was also placed at the same time to closely monitor hemodynamics; initial cardiac output (CO) was 3.2 L/min and cardiac index was 1.6 L/min/m² with a left ventricular end-diastolic pressure of 31 mm Hg, prompting placement of Impella device for post-myocardial infarction cardiogenic shock support (Table 1, column A). Despite Impella support at P7 and dobutamine infusion of 10 mcg/kg/min, he remained in cardiogenic shock with a CO of 3.1 L/min and cardiac index of 1.5 L/min/m² (Table 1, column B).

During repositioning of the Impella, the previously properly positioned transvenous pacemaker had pulled back, revealing an underlying CHB with a junctional escape rhythm at 60 beats per minute with LBBB and QRS duration of 174 ms. The electrophysiology team was consulted, as it was deemed that the refractory cardiogenic shock would improve with either cardiac resynchronization therapy

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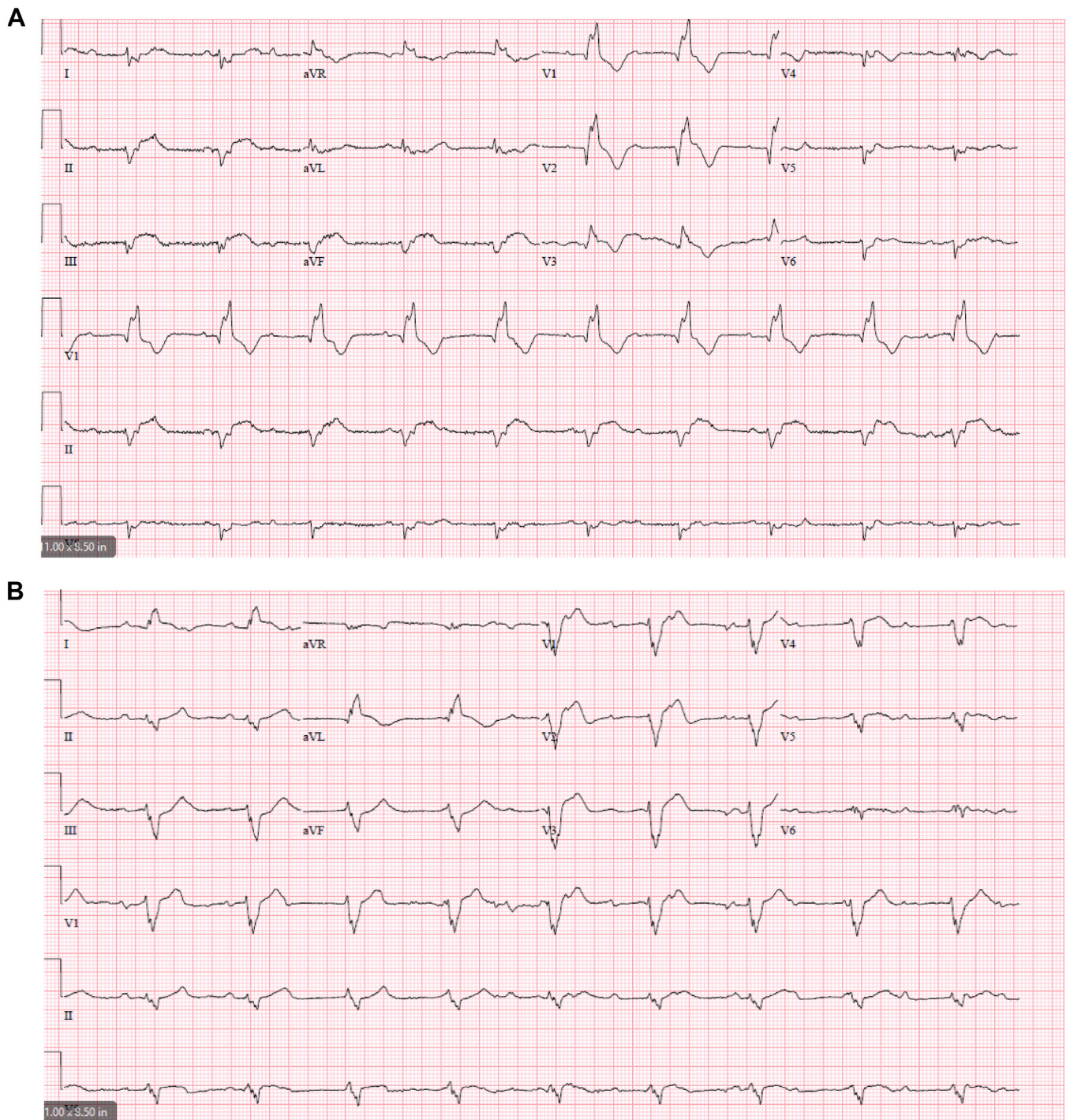


Figure 1 Presenting electrocardiograms. **A:** Initial electrocardiogram (ECG) with right bundle branch block and complete heart block. **B:** ECG with left bundle branch block and complete heart block.

(CRT) or dual-chamber left bundle branch pacing (LBBP). Serum creatinine was 3.7 mg/dL, swaying the electrophysiology team to attempt LBBP, given the risks of contrast nephropathy associated with CRT placement.

He was taken urgently to the electrophysiology lab, with successful implantation of a left subclavicular dual-chamber pacemaker (Medtronic, Minneapolis, MN). The RV lead (Medtronic 3830) was advanced to RV mid to upper

septum, achieving QRS pacing morphology compatible with capture of the deep septum and left-sided Purkinje system. The lead was turned 4–5 times with retesting before further advancement, given the presence of anteroseptal infarction. Testing with unipolar pacing was done to assure that the lead tip did not go through the infarcted septum. The procedure duration was 80 minutes, and total fluoroscopy time was 8.1 minutes. His initial postpacing ECG showed a QRS

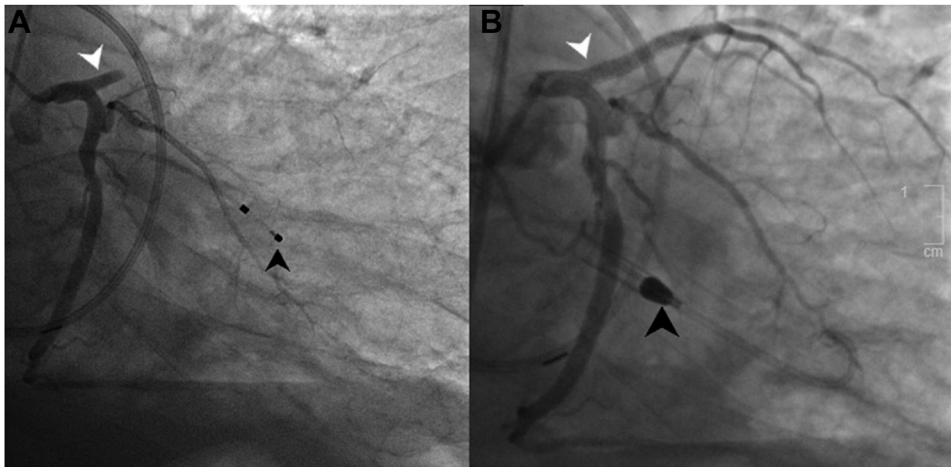


Figure 2 Left heart catheterization. **A:** Cardiac catheterization showing transvenous pacing wire (black arrow) in the right ventricle and 100% stenosis in the proximal left anterior descending artery (LAD) (white arrow). **B:** Post-stent placement in the LAD (white arrow) with Impella device placement (black arrow).

duration of 134 ms with an RBBB pattern and normal axis unmasking the infarction, with clear Q waves and ST elevation in the anterior precordial leads (Figure 3A). Interestingly, after a few minutes of pacing, his native conduction intermittently resumed showing RBBB and left posterior hemiblock, with QRS duration of 160 ms (Figure 3B). Within 1 hour of the implant, while on the same dose of dobutamine at 10 mcg/kg/min and Impella at P9, the CO improved from 3.1 to 7.0 L/min (Table 1, column C). The Impella device was removed within 48 hours and dobutamine was discontinued 1 day later. His QRS interval was further shortened to 90 ms after resolution of RBBB (Figure 3C). Position of the LBBP lead in the RV septum is shown in Supplemental Figure 1.

Discussion

In patients with CHB, dual-chamber cardiac pacing remains the only effective treatment. ACCF/AHA/HRS and the European Society of Cardiology recommend RV pacing in this population.^{1,2} However, RV pacing is not without its own adverse effects, such as left ventricle dysfunction, increased atrial arrhythmias, and heart failure hospitalizations.³ The mechanism of such events is likely linked to ventricular dyssynchrony.⁴ Hence, other sites of lead placement, such as RV outflow and RV septum, have been considered, but they have not been proven to have clinical benefit according to several large randomized control trials.^{5,6} To combat the issue of ventricular dyssynchrony, the lead should ideally be placed in the His bundle (HB) to allow for simultaneous activation of the left and right bundle. Unfortunately, HB pacing may be technically challenging and there remain concerns about lead stability and increased pacing thresholds.⁷ Additionally, if conduction block is distal to the HB, QRS narrowing may not occur as would have been in this case, as evidenced by the shorter QRS with pacing in comparison to that seen when conduction resumed (Figure 3A and 3B). The paced QRS showed a normal frontal plane axis and incomplete RBBB compared to RBBB and left posterior hemiblock with

conduction. This suggests probable anisotropic conduction of the left posterior fascicle or septal fascicle with more rapid conduction retrograde during pacing in comparison to during antegrade conduction. The return of conduction may have been coincidental; however, it can also represent relief of phase III block within the HB owing to retrograde conduction during pacing. Left posterior hemiblock is absent during pacing owing to left posterior fascicle conduction distal to the site of block during pacing and retrograde capture of the left anterior fascicle. The lessened RBBB with pacing may be due to the anodal RV capture, distal to the site of RBBB.

While the possibility that percutaneous revascularization eventually led to recovery of conduction cannot be excluded, CHB persisted for more than 48 hours, even after revascularization and despite Impella support. Figure 3C shows a paced narrow QRS with evidence of the anteroseptal infarction without RBBB. There is a premature atrial depolarization that conducts with a slightly wider QRS with an incomplete LBBB (red arrow) and notching of the upstroke akin to

Table 1 Hemodynamics data

	A	B	C
Medications/support	None	Impella P7 Dobutamine 10 mcg/kg/min	Impella P9 Dobutamine 10 mcg/kg/min
HR (bpm)	57	57	AS-VP at 90
RA (mm Hg)	14	10	7
PA (S/D/M) (mm Hg)	48/ 18/ 26	46/8/21	40/12/25
PaO ₂ (%)	56	49	72
CO (L/min)	3.2	3.1	7.0
CI (L/min/m ²)	1.6	1.5	3.5
SVR (dsc)	1250	1496	753

AS-VP = A sensed, V paced; CI = cardiac index; CO = cardiac output; D = diastolic; HR = heart rate; LBBP = left bundle branch pacemaker; M = mean; PA = pulmonary artery; PaO₂ = pulmonary artery oxygen saturation; RA = right atrium; S = systolic; SVR = systemic vascular resistance.

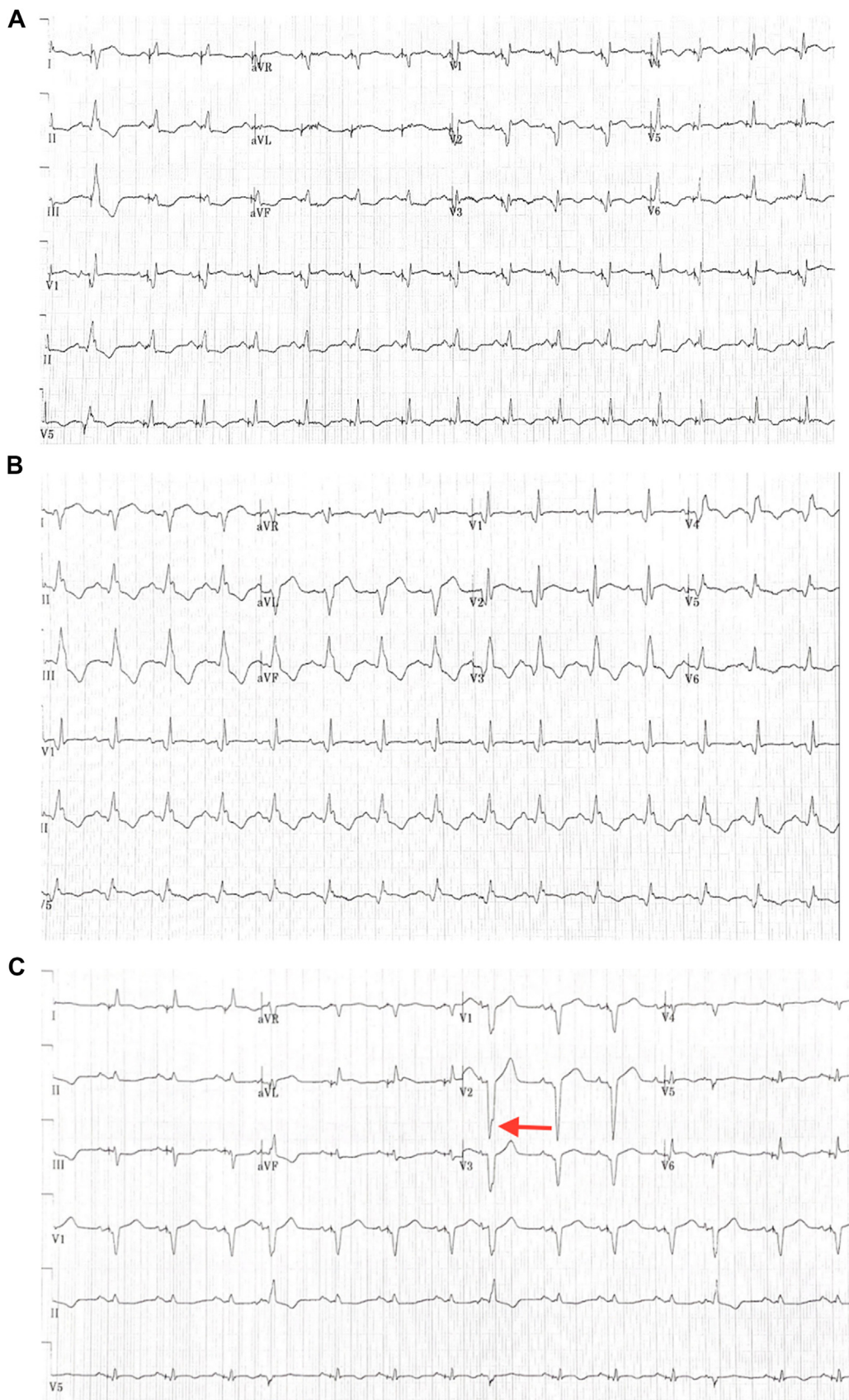


Figure 3 Post-left bundle branch pacemaker electrocardiograms. **A:** A-sensed, V-paced, QRS 134. Q waves in anterior precordial leads indicating anterior infarct with a pseudofusion beat (first complex) suggesting recovery of conduction similar to the QRS shown in panel B. **B:** A-sensed-V-sensed. Resolution of complete heart block and Q waves in anterior precordial leads indicating anterior infarct and left posterior hemiblock after a few minutes of pacing. **C:** Paced electrocardiogram 2 days post dual-chamber device placement. QRS duration 90 ms. Red arrow shows a natively conducted premature atrial depolarization with a wider QRS than the pace complex and with notching of the upstroke.

Cabrera's sign, compared to the paced beats. This suggests recovery of right bundle conduction and the paced beats represent a fusion of LBBP with intrinsic right bundle conduction, though improved anodal capture of the right side of the septum cannot be excluded. We opted to maintain a sensed and paced AV delay of 120 ms to minimize left ventricular conduction delay even after conduction had returned.

LBBP has emerged as an alternative to achieve physiologic activation and optimize hemodynamics.⁸ Although large-scale trials are pending on this technique, small studies have shown a high success rate and favorable lead thresholds.^{9,10} In this patient with severe left ventricle dysfunction and CHB, dramatic hemodynamic improvement was observed. Furthermore, the diagnosis of acute myocardial infarction was made with the help of elevated cardiac enzymes and transthoracic echocardiogram findings, not with his ECG. His LBBB did not meet Sgarbossa's criteria, but it is clear that after his LBB was engaged, the anterior infarct pattern was unmasked, as evidenced by the Q waves and ST elevation in the anterior precordial leads, which would not have been observed with RV or CRT pacing.

Finally, we did consider the benefit/risk ratio of attempting a CRT vs LBB area pacing, particularly given the antero-septal infarction. Similar outcomes would likely have occurred with CRT pacing; however, owing to the patient's tenuous hemodynamics and stage IV chronic kidney disease, we felt an attempt to place the LBB lead in the high septum, if successful, would result in a shorter procedure and avoidance of intravenous contrast use. As seen on the echo in [Supplemental Figure 1](#), the lead is in the noninfarcted, thicker part of the basal septum.

Conclusion

Dual-chamber LBBP may be an option to achieve atrioventricular and biventricular resynchronization with a single

ventricular pacing lead and improve cardiac output in patients with CHB in the setting of refractory cardiogenic shock.

Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hrcr.2021.11.016>.

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