Echocardiography-guided determination of reliable atrial pacing in a patient with congenital heart disease

Nishaki K. Mehta, MD, FHRS,* Sahitya Allam, BS,* Saurabh Rajpal, MD,[†]
Kenneth Bilchick, MD, MS, FHRS,* Michelle Gurvitz, MD[‡]

*From the *Division of Cardiovascular Medicine, University of Virginia, Charlottesville, Virginia, [†]Division
of Cardiovascular Medicine, The Ohio State University, Columbus, Ohio, and [‡]Division of
Cardiovascular Medicine, Boston Children's Hospital, Boston, Massachusetts.*

Introduction

Determination of the pacemaker atrial capture threshold is critical to ensuring appropriate pacemaker function and is routinely assessed during device interrogation. However, in dual-chamber pacing, it is sometimes difficult to identify atrial depolarization on surface electrocardiogram (ECG), which complicates ascertainment of reliable atrial capture.[1] Progressive cardiac disease can also result in elevated capture thresholds, which can manifest as a failure to capture. We present a case of echocardiography-guided determination of atrial capture for programming of atrial thresholds in a pacemaker-dependent patient with severely diseased atria resulting from congenital heart disease. Atrial activity was indiscernible on surface ECG. Ultrasound-guided transmitral inflows enabled confirmation of atrial capture and atrial pacing. This is yet another example of multimodality management with echocardiography.

Case report

A 42-year-old woman with repaired tetralogy of Fallot, an atrial septal defect, and a dual-chamber pacemaker for sinus node dysfunction and atrial arrhythmias presented for initial evaluation to our institution regarding indeterminate atrial lead function. She had an initial repair consisting of patch closure of both septal defects and placement of a transannular patch. She later required pulmonary valve replacement with a 25 mm porcine bioprosthesis. Subsequently, she developed severe tricuspid and pulmonary valve insufficiency, severe right ventricular and right atrial enlargement, and biventricular systolic dysfunction. Furthermore, she had intermittent loss of atrioventricular (AV) conduction at baseline and thus became pacemaker dependent. Her dual-chamber Medtronic pacemaker had been placed 4 years ago and was

Address reprint requests and correspondence: Ms Sahitya Allam, 1215 Lee Street, Charlottesville, VA 22903. E-mail address: sa3py@hscmail.mcc.virginia.edu.

programmed to DDDR 90–130 with an upper sensor rate of 125 beats per minute. The right atrial and right ventricular leads were Medtronic 5076 (active fixation, bipolar, 456 ohms) and Medtronic 5076 (active fixation, bipolar, 494 ohms), respectively. Her medications included lisinopril, furosemide, and sotalol.

At the time of her clinic visit, device interrogation revealed an atrial threshold of 0.5 mV at 1.2 ms. However, atrial capture could not be determined with atrial threshold testing conducted via the programmer analyzer system and continuous 12-lead ECG strips. Review of her chest radiograph showed no visible dislodgment of the leads (Figure 1), and review of her surface ECG demonstrated a corroborating absence of P waves following atrial pacing spikes (Figure 2A). Additionally, her intermittent AV conduction made it difficult to ascertain whether there was true atrial capture using the ECG tracing, given that there was also intermittent ventricular pacing (Figure 2A). There was no VA conduction at baseline. Furthermore, there was unreliable evaluation of the evoked electrical response on the local electrogram as well as an absence of an escape atrial rhythm at loss of capture. Putting all these findings together, the atrial capture threshold was suspected to be very high. Consequently, the atrial outputs were programmed to 6 V at 1.4 ms.

Based on these considerations, the electrophysiology service was consulted to interrogate the device and determine the underlying capture threshold. Lewis lead placement was also inconclusive for determination of atrial capture.[2] Therefore, echocardiography was employed to determine transmitral inflows. Evidence for sequential E and A waves on pulsed wave Doppler confirmed a contractile response to pacing, and thus, atrial capture. We were able to prove atrial capture down to 0.2 V at 0.4 ms, but at this threshold there was intermittent loss of atrial capture, as seen by loss of the A wave and an escape ventricular beat (circled QRS complex in Figure 2B). However, there were intrinsic QRS complexes at a threshold of 0.3 V at 0.4 ms suggestive of intrinsic AV nodal conduction with atrial pacing and definite atrial capture (circled QRS complex in Figure 2C). Based on these findings, the atrial capture threshold was nominally

KEY TEACHING POINTS

- Optimal device programming depends on the ability to determine the atrial capture threshold. Long-term cardiac dysfunction in patients with congenital heart disease may lead to an abnormal myocardial substrate, which can make it challenging to determine atrial activity.

- Transmitral inflow patterns assessed by Doppler echocardiography can be utilized to confirm atrial lead capture for optimization of the atrial capture threshold.

- Echocardiography can be employed when conventional atrial capture testing options have been exhausted, such as assessment of surface P waves on electrocardiogram and evoked intracardiac potentials on the local electrogram.

programmed to 2.5 V at 0.4 ms with significant expected improvement in battery longevity.

Discussion

There can be difficulty in ascertaining atrial depolarization on a surface ECG in an individual with severely diseased atria, but it is important to determine reliable atrial lead capture to establish AV synchrony. It is also critical to program the device with the appropriate capture threshold in order to preserve its battery life. Standard methods were utilized here to confirm atrial capture (the programmer analyzer system, use of an intracardiac electrogram to visualize the evoked electrical response and eradication of an escape atrial rhythm) but were inconclusive.[1] Additionally, Lewis lead (S5) placement was attempted. This is a modified lead I where the right-arm lead electrode is moved to the manubrium adjacent to the sternum and the left-arm electrode is moved to the right fifth intercostal space adjacent to the sternum. This allows for a different atrial vector, which is perpendicular to the ventricular depolarization vector. It was originally devised to appreciate flutter wave morphology. In our case, we were employing this to obtain a stronger P-wave root-mean-square signal strength, but this was also ultimately unsuccessful.[2] Lastly, lead dislodgment or maturation, battery depletion, and medications could also all be potential causes of failure to capture, but these were also systematically ruled out.

After conventional testing options had been exhausted, atrial capture and proper lead function were finally proven with a novel solution: echocardiography was used to provide electromechanical evidence of atrial contraction in response to pacing at certain atrial thresholds. The E and A wave represent the passive and active ventricular filling from the atrium, respectively. The latter is impacted by atrial lead capture and has the potential to augment ventricular filling. In the present case, the A wave clearly preceded intrinsic QRS complexes, illustrating atrial contraction alongside intrinsically conducted ventricular beats, proof of definite atrial capture and atrial pacing. This example demonstrates how pulsed-wave Doppler can be used for determining the A wave as a confirmation of reliable atrial lead capture, a reasonable alternative when conventional methods are not fruitful.

Additionally in this vignette, other strategies for determining atrial capture could have been considered. For instance, pacing with a short AV interval to mimic pacemaker syndrome could have yielded cannon A waves on jugular venous exam, evidence of atrial contraction against a closed AV valve.[3] Another strategy could have been to extend out the AV delays and recheck atrial thresholds on the local electrogram channel, which may have brought out atrial activity. This strategy is analogous to employing the AV search hysteresis algorithm, which is a proprietary algorithm that actively searches for intrinsic AV conduction and extends the AV delay by 10% to 100% to allow for intrinsic conduction.[4] It was demonstrated in the INTRINSIC RV trial to
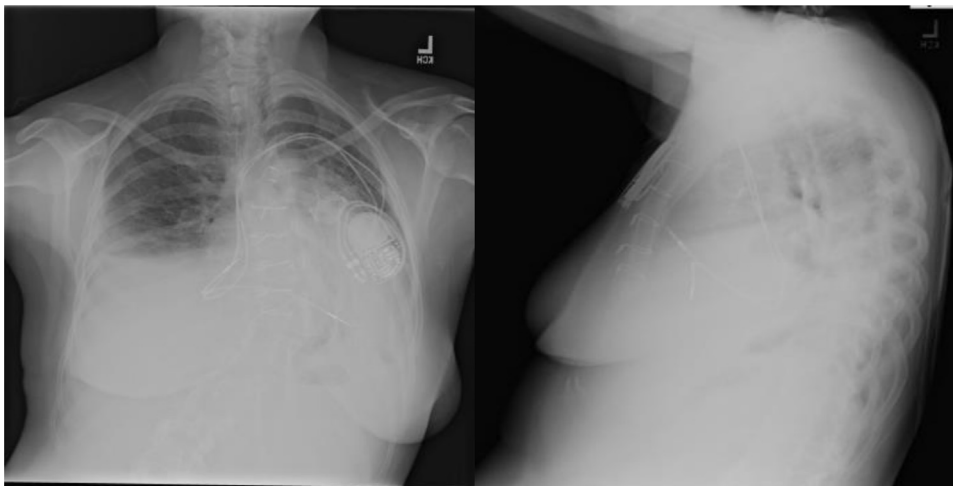

Figure 1   Chest radiograph, posteroanterior and lateral view. No evidence of macro-dislodgment of the pacemaker leads is seen.
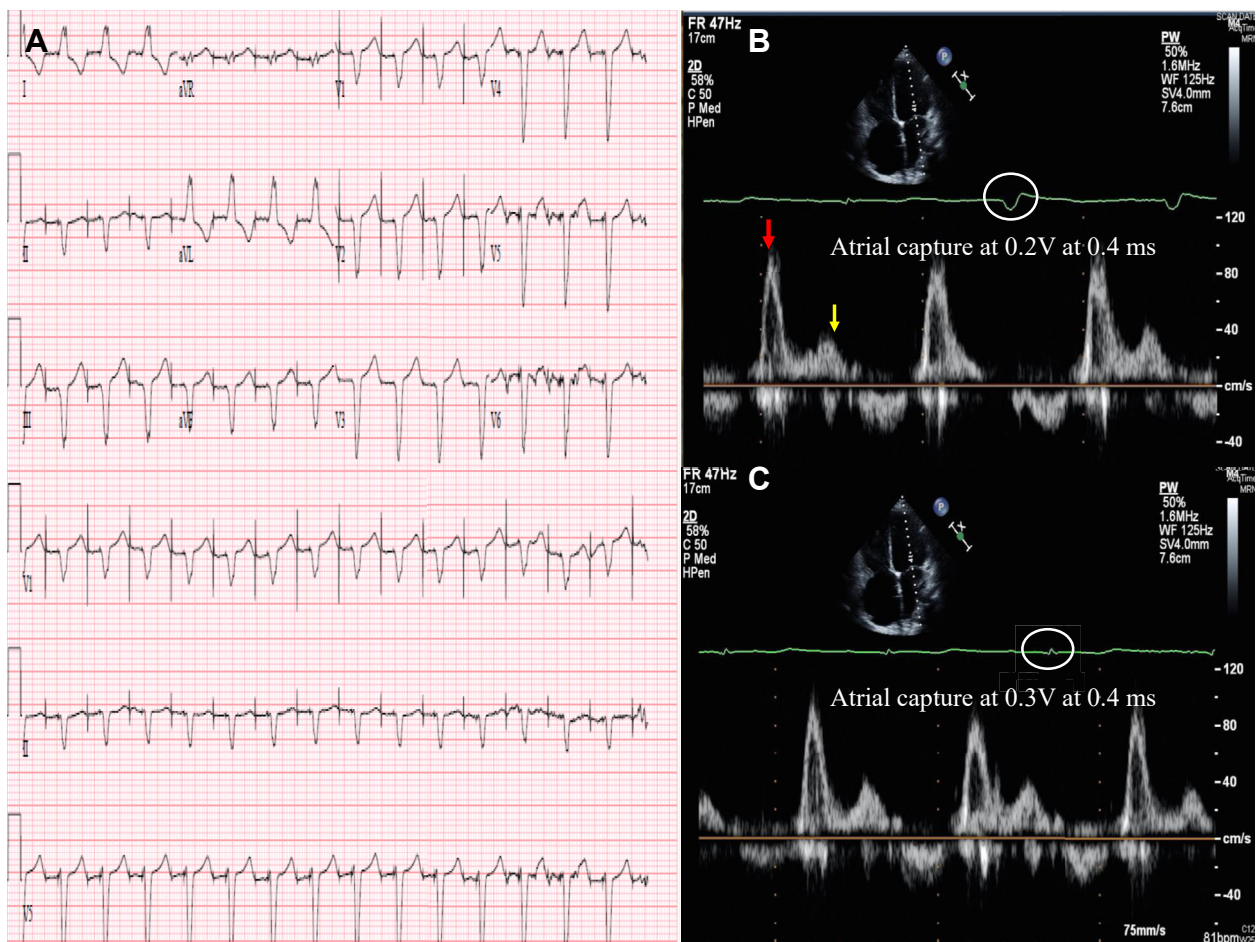
Figure 2   Demonstration of atrial capture. **A:** Atrial pacing spikes with intermittent ventricular pacing spikes and no clear atrial activity on surface electrocardiogram. **B:** Evidence of loss of atrial capture with no A wave noted and escape ventricular beat (*circled*). **C:** Evidence of atrial capture with E and A waves noted with intrinsic ventricular conducted beat (*circled*). Red arrow: E wave; yellow arrow: A wave.

lower the prevalence of ventricular pacing in dual-chamber DDDR 60–130 implantable cardioverter-defibrillators.[4] In addition, given that there was intermittent AV conduction, consideration of exercise, isoproterenol, or atropine to enhance AV nodal conduction could have been of value to eliciting atrial activity.[5] Lastly, invasive modalities like electroanatomical mapping of atrial activity or determining atrial electrograms with esophageal mapping were also possible options we could have explored.[6]

Conclusion

In conclusion, we have demonstrated in this example that evaluation of atrial lead capture with use of echocardiography is a promising new tool for device interrogation in patients with congenital heart disease and poor atrial capture, as well as in other patients with decreased intrinsic atrial activity from scarring and/or AV block. Echocardiography could be used in challenging situations, both in the device clinic and at the time of device implantation, in patients such as these in order to provide more accurate assessments of atrial capture, improve device longevity by helping reduce capture thresholds, and

prevent procedures for atrial lead revisions that could put patients at unnecessary risk. This novel application of echocardiography adds to its existing utility in pacemaker optimization and has the potential to be of significant clinical benefit if there is concern for lead threshold testing.

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