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An unusual cause of variable paced QRS complexes in a child with heart failure

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1. Case

A 7-year-old male with a complex cardiac history was admitted to our cardiac care unit for treatment of progressive heart failure. In infancy, he had undergone surgical repair of double outlet right ventricle, ventricular septal defect and pulmonary stenosis. At 5 years old, he underwent surgical pulmonary valve replacement at another institution for severe pulmonary insufficiency and right ventricular enlargement; at that time an epicardial dual chamber biventricular pacemaker was implanted for high grade 2nd degree atrioventricular (AV) block and intermittent complete AV block with the expectation of near-100% ventricular pacing (Fig. 1a). Over the last 2 years, he developed progressive left ventricular (LV) systolic dysfunction, possibly related to LV non-compaction. Despite changes in outpatient therapy, echo-guided reprogramming of the pacemaker and intermittent inpatient "tune-ups", he was ultimately admitted for continuous intravenous inotropic therapy and listed for cardiac transplantation.

Initial interrogation of his Medtronic™ Serena generator showed DDD mode (70–180 bpm), with bipolar atrial output 2 V @ 0.40 ms (capture threshold 0.75 V @ 0.40 ms), bipolar right ventricular (RV) output 4 V @ 0.40 ms (capture threshold 2 V @ 0.40 ms) and bipolar LV output 5 V @ 0.60 ms (capture threshold 2.75 V @ 0.60 ms). Ventricular capture management had been disabled due to intermittently elevated automated thresholds triggering high outputs that substantially reduced predicted battery longevity. Soon after admission, continuous inpatient telemetry recorded intermittently abrupt changes in paced QRS morphology on consecutive days, occurring at ~2:45 a.m. and lasting 3–4 min (Fig. 1b).

2. Discussion

Review of telemetry recordings show the changed QRS complexes occurring during stable background AV pacing at 70 bpm. The QRS complexes were immediately preceded by what appeared to be A-pacing spikes, and intermittently followed by biventricular pacing spikes that occurred late in the QRS complex at unchanged paced AV times (Fig. 1b). The abrupt change in the QRS complexes could be due to several potential causes, including variable intrinsic cardiac rhythms competing with background stable pacing, pacemaker malfunction, or auto-diagnostic device algorithms.

As this patient's AV conduction had been intermittently intact, perhaps these recordings reflected "sinus" or ectopic atrial rhythm and intact AV conduction, with very low amplitude P waves not readily apparent on surface recordings and intrinsic QRS complexes different from the paced QRS complexes. We suspected this was not the case because: (1) repeat device interrogation confirmed complete AV block with ventricular escape rate of ~50 bpm, 80% A-pacing and 100% V-pacing; (2) telemetry showed predominantly AV pacing in the early AM hours; and (3) at other times, P waves of intrinsic sinus rhythm and intermittent premature atrial complexes were clearly visible. An alternative explanation for changing QRS complexes could be episodic idioventricular rhythm occurring at rates and timing quite close to stable AV pacing rate, with isorhythmic dissociation accompanied by atrial pseudo-pseudofusion

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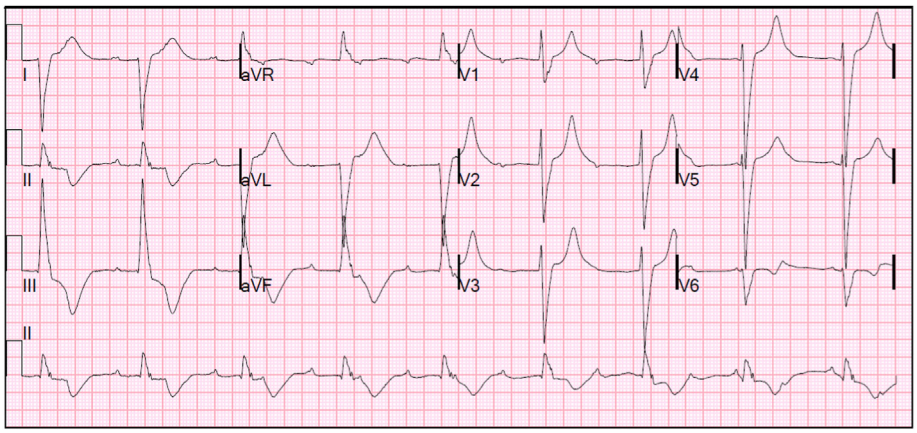

Fig. 1a. 12-lead-ECG demonstrating complete atrioventricular block with wide QRS escape rhythm.

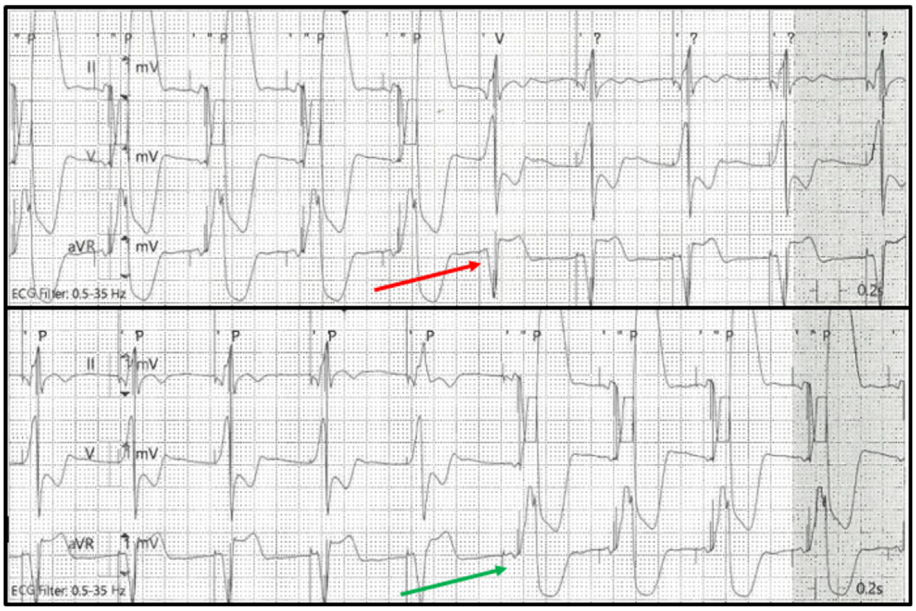

Fig. 1b. Top panel - Telemetry strip demonstrating transition from appropriate atrial-biventricular pacing to a different QRS complex that immediately follows atrial pacing spikes **(red arrow).** Note ventricular pacing spikes are sometimes recorded late in the QRS complex. **Bottom panel -** Telemetry strip demonstrating transition back to the prior QRS complexes associated with earlier atrial-biventricular pacing **(green arrow**).

[1]. The perfectly steady temporal relationship between the atrial pacing spikes and the different QRS complexes, persisting throughout each minutes-long occurrences on consecutive days, suggested this explanation was unlikely.

To evaluate for lead-related problems, repeat interrogation was performed simultaneous with continuous 12-lead ECG recordings (Fig. 2). Both ventricular leads showed stable impedances and capture thresholds, and outputs were still twice the amplitude capture thresholds. In addition, the occurrence of biventricular pacing artifacts late in the changed QRS complexes (Fig. 1b) made intermittent noncapture by one of the ventricular leads an unlikely explanation.

The recurrence of this phenomenon at the same time in the early AM hours on consecutive days suggested device auto-testing feature might be the culprit [2]. Ventricular capture management for both leads had been disabled, eliminating this as a cause. Perhaps intermittent ventricular capture was occurring during atrial pacing, a phenomenon we have occasionally encountered with temporary epicardial pacing leads in children immediately following CHD surgery. Pacemaker interrogation revealed tall far-field R waves on the atrial channel. During high output atrial pacing (8 V @ 0.40 ms and at 6 V @ 1 ms) (Fig. 2), there was clear capture of the ventricles with QRS complexes unlike those during biventricular, RV-only and LV-only pacing. Atrial pacing-ventricular capture (AP-VC) beats were followed by intermittent sensing on the ventricular channel: biventricular pacing spikes occurred late in the QRS complexes during ventricular undersensing, and were not recorded during appropriate sensing. These findings, similarity to inpatient telemetry recordings, and previous chest computed tomography scan demonstrating the bipolar epicardial atrial lead had been implanted quite close to the tricuspid valve annulus (Fig. 3), strongly suggested that atrial pacing could be intermittently capturing the ventricles. Why did this occur in this patient? Either the AP-VC threshold was intermittently much lower, or the device was sometimes delivering unusually high output impulses. What would cause intermittent high output atrial pacing?

The auto-diagnostic “atrial lead position check” (ALPC) feature incorporated in some Medtronic™ pacemakers was the likely

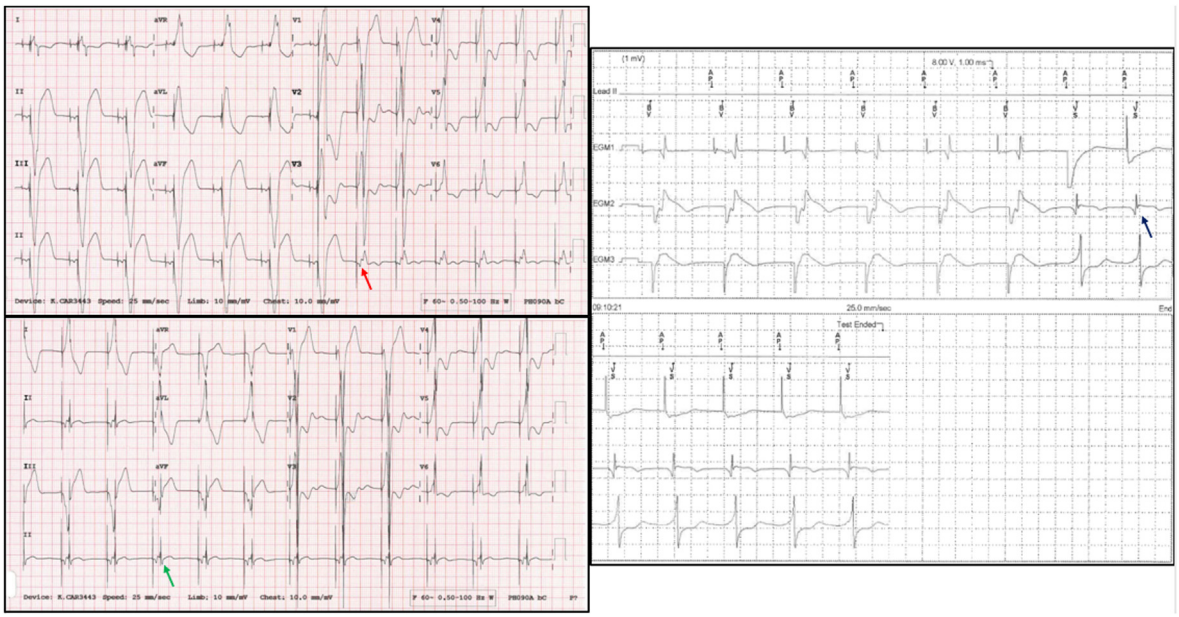

Fig. 2. Top left panel - 12-lead-ECG demonstrating abrupt change in QRS morphology (starting one beat after recording on V1–V3) due to engaging high output atrial pacing, resulting in ventricular capture **(red arrow).** Furthermore, ventricular pacing was inhibited at this time. **Bottom left panel -** 12-lead-ECG demonstrating atrial pacing with ventricular capture and ventricular undersensing leading to inappropriate ventricular pacing spikes **(green arrow). Right panel** – Electrograms and marker channels recorded during pacemaker interrogation demonstrating ventricular capture with high output atrial pacing and inhibition of ventricular pacing **(blue arrow).** The AP-VS intervals recorded by the device were 110 ms.

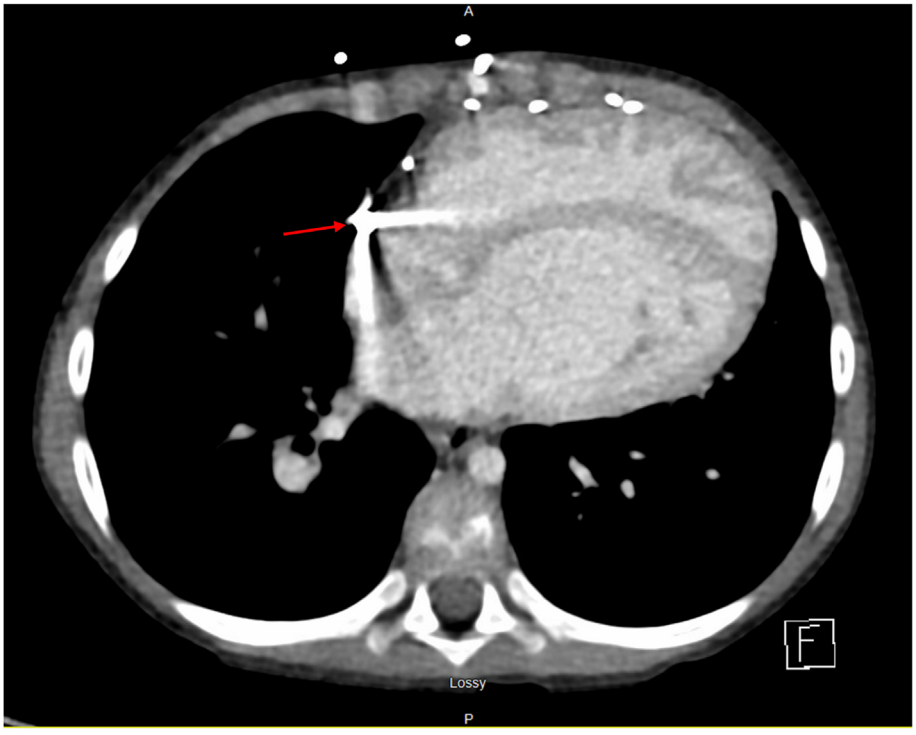

Fig. 3. Chest computed tomography scan demonstrating close proximity of the bipolar atrial leads to the tricuspid valve annulus **(red arrows)**.

explanation [3]. ALPC in devices with atrial anti-tachycardia pacing (A-ATP) therapies is designed to ensure the position of the atrial lead has not dislodged, since rapid high output impulses to treat atrial tachycardia could inadvertently induce ventricular arrhythmias. The ALPC algorithm runs every 24 h at 2:45 a.m., only if the anti-bradycardia pacing mode includes atrial pacing (AAI or DDD) as was the case in our patient. During the automated check, the atrium is paced 255 times with high-output pulses (defaulting to settings for A-ATP therapies) and searches for AP-VS intervals <80 ms that may indicate ventricular capture; a high proportion of short AP-VS intervals confirms probable lead dislodgement and automatically disables all atrial ATP therapies. We noted that this

check would take ~3½ minutes to complete when programmed DDD @ 70 bpm, consistent with the duration of the phenomenon noted on our patient's telemetry.

While the ALPC auto-testing feature is important for confirming atrial lead position in transvenous pacemakers with some chance for lead dislodgement, its use in epicardial pacemakers, often implanted in children with or without concomitant congenital heart disease (CHD), introduces some interesting challenges. As expected for leads firmly sutured onto the heart, epicardial atrial lead dislodgement with subsequent capture of the ventricles is extremely rare [4]; indeed, we have not personally encountered this problem. Unfortunately, epicardial atrial leads are not infrequently implanted by CHD surgeons near the AV annulus, resulting in tall far field R waves (FFRW) recorded on the atrial channel during device follow-up. Pediatric electrophysiologists often face problematic FFRW that overestimates "atrial high-rate events" (AHRE) and can interfere with appropriate DDD function. Fortunately, this can often be addressed by closely reviewing AHRE recordings for FFRW artifact, and reprogramming atrial lead sensitivity, or refractory or blanking intervals.

However, this case highlights another significant problem we suspect is not well-known by most clinicians caring for these children, but could have serious clinical ramifications for patients with A-ATP-therapies engaged.

ATP outputs are generally programmed much higher than antibradycardia settings to ensure effectiveness of delivered ATP sequences; the default setting in Medtronic devices is 6 V at 1.5 ms. Ventricular capture at these outputs at rapid rates, triggered by true atrial arrhythmias or artifactual double-counting, could potentially induce serious ventricular arrhythmias. Importantly, the safety measure whereby the ALPC automatically disables A-ATP therapies when high output AP-VS intervals are short (<80 ms) may be protective. However, short AP-VS intervals were not always detected in our patient (AP-VS intervals of 110 ms), possibly related to progressive myocardial disease causing markedly delayed intraventricular conduction, also evidenced by progressively prolonged QRS complexes on ECG. Fortunately for our patient, A-ATP therapies were not indicated and had not been engaged. It is noteworthy that in those patients who might benefit from A-ATP, the problem of AP-VC could either prevent its use due to auto-disabling with frequent short AP-VS sequences, or potentially be proarrhythmic if ventricular depolarization was undersensed. ALPC cannot be turned off in these devices, and it continues to run even if ATP is not engaged. After lowering the atrial output for ATP therapies and thus the output at which ALPC is performed, AP-VC was no longer recorded on telemetry.

This experience highlights the need for careful intraoperative and postoperative testing of atrial epicardial leads in children beyond simply ensuring acceptable atrial sensing and capture thresholds and minimizing FFRW. High output pacing of the atrial lead to evaluate for inappropriate ventricular capture can be particularly important for patients with devices having A-ATP therapies, and for those benefiting from atrial pacing in general as atrial capture thresholds and attendant need for increasing programmed outputs can rise over time, potentially increasing the chance of inadvertent ventricular capture.

Declaration of competing interest

The authors have nothing relevant to disclose with respect to submission of this manuscript.

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