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Case Report

A Case of Loss of Ventricular Pacing Due to Inappropriate Hysteresis Function Programming After AV Node Ablation

Vladimir Poletaev, MD, Vidal Essebag, MD, PhD, and Martin Louis Bernier, MD

McGill University Health Centre, Montreal, Quebec, Canada

Modern implantable devices capable of pacing are armed with a multitude of programmable and automated features. Although some features represent important advances in device safety and performance, many also can mimic device malfunction and cause clinical manifestations and unnecessary hospital admission. We report a case of "pseudo-malfunction" of a pacemaker due to hysteresis programming that caused symptomatic bradycardia after atrioventricular (AV) node ablation.[1]

Novel Teaching Points

- A slow heart rate in a patient with a pacemaker is not always a sign of pacemaker dysfunction.
- Checking the hysteresis function status in every pacemaker-dependent patient is important, particularly for device models where hysteresis function is automatically activated when enabling device features such as sensor or autocapture.

Case Presentation

An 81-year-old woman underwent atrioventricular (AV) node ablation. Her past medical history included permanent atrial fibrillation with poor rate control, dyslipidemia, hypertension, type II diabetes mellitus, and cerebral ischemic attack. Six months earlier, the patient underwent implantation of a permanent pacemaker St. Jude/Abbott Medical 1272 Assurity SR MRI. All physical parameters of the pulse generator and right ventricular lead were under normal limits during routine follow-up after implantation. The device was programmed in VVI mode with a lower rate limit (LRL) of 60 beats per minute (bpm) initially. Programming was changed to VVI 80 bpm after AV node ablation. Several hours after the procedure, pacing at a rate of 80 bpm stopped and was followed by bradycardia with a ventricular escape rate of 37 bpm (Fig. 1), and the patient complained of dizziness. Pacemaker malfunction was suspected. Magnet application was performed and led to resumption of pacing. The patient returned to the electrophysiology department for additional investigations. Initial interrogation showed unchanged parameters of the electrode and good longevity of the battery. R waves were 11 mV, right ventricular lead impedance was 531 Ohms, and the capture threshold was $<$ 1 V/0.5 msec. Provocative testing with deep respiration and active movement of the arms and the generator pocket did not evoke pacing inhibition or show artefact on ventricular electrogram. A 12-lead electrocardiogram (ECG) showed ventricular pacing at the programmed rate, and no loss of capture. Chest radiograph showed no obvious lead dislodgement or fracture.[2] The magnet rate was normal. Re-interrogation revealed active hysteresis function at a rate of 30 bpm, which explained the documented bradycardia episode with an escape rate just slightly higher than the hysteresis rate. Hysteresis was triggered after a premature ventricular contraction (PVC) and remained active in the presence of an escape at around 35 bpm.

Discussion

In our case, we had clear documentation of symptomatic bradycardia in a patient with a pacemaker. On an ECG tracing, pacemaker spikes were missing (Fig. 1A). In such a situation, the term "failure to pace" usually applies, and differential diagnosis includes the following:

- failure of pacemaker output;
- oversensing; and
- "pseudo malfunction."

Failure of pacemaker output

Causes of "failure to pace" include lead failure due to fracture, lead displacement, Twiddler's syndrome, generator failure due to battery depletion, pacemaker turned "off" or to

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Corresponding author: Dr Vladimir Poletaev, Department of Electrophysiology, McGill University Health Centre, Montreal, Quebec, Canada. Tel.: +1-438-985-8056.

E-mail: vladimir.poletaev@mail.mcgill.ca

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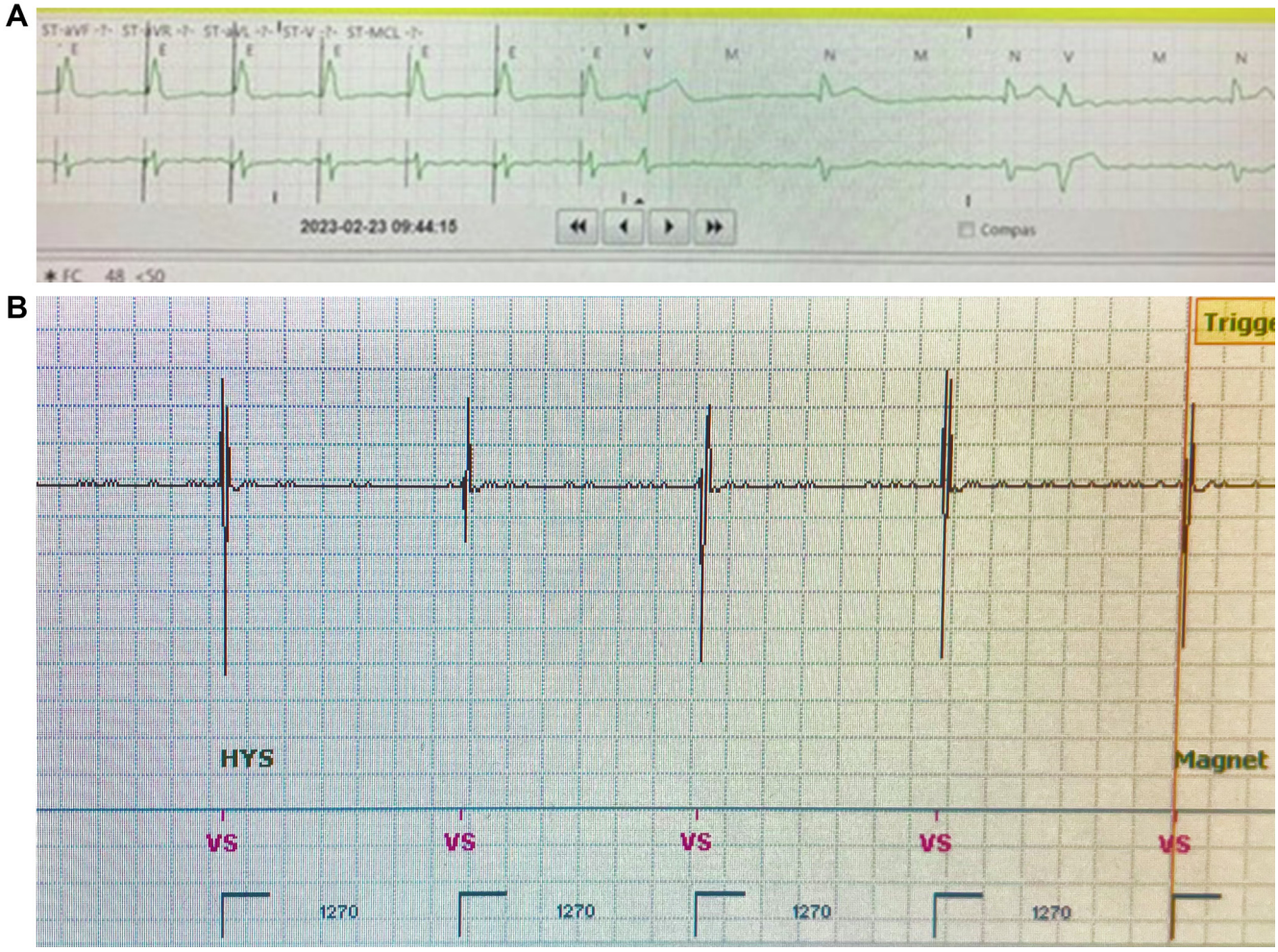

Figure 1. (**A**) Telemetry recording of bradycardia episode due to suddenly interrupted pacing. (**B**) Intracardiac recording during bradycardia episode that demonstrates intrinsic rhythm at 47-50 beats per minute with hysteresis marker.

nonpacing mode, poor connection at connector block, and loose set screw.[3]

Oversensing

Pacemaker oversensing occurs due to multiple factors that can be categorized as either extrinsic or intrinsic. Extrinsic factors include lead noise or artefact caused by electromagnetic interference from machinery and sources of high voltage. Intrinsic factors can be from physiological or nonphysiological sources. Physiological sources include cross-talk due to oversensing of signal in the opposite chamber. Nonphysiological sources include lead fracture or a misaligned connection of the lead connector pin into the device connector block. If all of these causes are ruled out, then a "pseudo malfunction" due to device-specific features should be considered.[1]

"Pseudo malfunction"

On occasion, clinicians reviewing device diagnostics such as electrograms or rate histograms may observe paced or sensed atrial or ventricular rates below the programmed LRL. Frequently, these temporary "slow" rates indicate normal device function resulting from programmed features or standard sensing algorithms. "Pseudo malfunction" can be attributed to one of the following causes[4]: rate hysteresis; AV search algorithms that promote intrinsic conduction; and PVCs.

Rate hysteresis

Rate hysteresis is a rate enhancement feature designed to promote longer periods of intrinsic rhythm by temporarily allowing both atrial and ventricular intrinsic rates to fall below the programmed LRL. If the rate hysteresis feature is programmed to the "on" setting, the programmed LRL is lowered by the programmed hysteresis offset or to the hysteresis rate. This lowering allows a patient's atrial and ventricular rate to fall below the LRL for one or more cardiac cycles.

Classic hysteresis programming is less relevant in new-generation devices, because every major vendor developed unique algorithms for minimizing right ventricular pacing, but it still can be useful in certain situations. Hysteresis usually is available in single-chamber modes (AAI, ADI, VVI, VDI, AAT, VVT) and when "rate response" is set to "off." The hysteresis feature is nominally "off." When hysteresis is programmed, a sensed event temporarily suspends the lower

rate, and the pacemaker determines its escape rate from the hysteresis rate instead. As long as the intrinsic rate remains above the hysteresis rate, pacing is inhibited. The first occurrence of an escape pace at the hysteresis rate suspends hysteresis operations and reestablishes the lower rate as the minimal rate. In this case, the hysteresis rate was programmed unintentionally to 30 bpm.

In our case, after atrioventricular node ablation, the patient became partially pacemaker dependent, with an underlying slow ventricular escape rhythm. Premature ventricular contraction activated the inappropriately programmed undesirable hysteresis function and allowed the pulse to drop down to 37 bpm. "Pseudo malfunctions" are unusual and unexpected ECG findings that appear to be caused by pacemaker malfunctions but actually indicate normal pacemaker behavior under specific programmed parameters. We suspect that hysteresis was inadvertently programmed to the "on" setting during programming in preparation for the AV node ablation (with the LRL decreased to 40 bpm during ablation). A point worth noting is that certain St. Jude/Abbott pacemakers will automatically turn on hysteresis at 10 beats below the programmed LRL when either the sensor (rate response) or autocapture feature is enabled. Important to note is that when this occurs, the hysteresis remains programmed at the same rate, even if these features are disabled or the programmed lower rate is increased. In our case, if hysteresis was automatically programmed to 30 bpm when the lower rate was programmed to 40 bpm, the hysteresis rate remained at 30 bpm when reprogrammed to VVI 80 bpm. To rectify the problem, we turned the hysteresis function off.

Conclusion

Inappropriate pacing inhibition may be an undesired outcome from inappropriate activation of hysteresis rate programming, and it is an example of a "pseudo malfunction" of a pacemaker. In this case report, our goal is to draw attention to this possibility and emphasize the importance of verifying whether hysteresis rate programing is active and whether it is necessary in all pacemaker-dependent patients.

Ethics Statement

The paper reported is adherent to the ethical guidelines.

Patient Consent

The authors confirm that patient consent is not applicable to this article. This case report uses de-identified data; therefore, the IRB did not require consent from the patient.

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Disclosures

The authors have no conflicts of interest to disclose.

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