Atrioventricular hysteresis pacemaker mode promoting a short-long-short sequence at the onset of ventricular tachycardia

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Introduction

There is a longstanding love–hate relationship between pacing and tachycardias.[1] Pacemakers keep being the main treatment for most bradycardia issues, but carry with them the ability to induce or mediate many forms of tachycardias, such as ventricular tachycardia (VT) due to the "R on T" phenomenon, endless-loop tachycardia, and pacemaker-mediated tachycardia.[1]

New technology features in modern pacemakers helped to avoid many of these problems, but others were created. In this article we describe a potentially fatal condition caused by a pacemaker where a hysteresis mode induced a short-long-short (SLS) sequence and VT.

Case report

An 84-year-old man with a history of previous coronary artery bypass graft, heart failure, and an intermittent atrioventricular (AV) block treated with an Entovis-Biotronik pacemaker implanted 5 years ago was admitted to perform a carotid bypass after an ischemic stroke due to severe carotid obstruction. Two days after the procedure, he had a cardiac arrest recorded by the cardiac monitor (Figure 1). During this episode, potassium and magnesium levels were within normal limits.

The pacemaker was programmed DDD with heart rate of 85 beats/min, AV interval of 200 ms, and IRS-plus (Intrinsic Rhythm Support) hysteresis mode on. IRS-plus may facilitate VT onset, acting as an induced SLS cycle.

Right after the cardiac resuscitation, the patient had his pacemaker adjusted and all electrograms were lost because the Entovis-Biotronik erased records after any reset.

The patient remained unstable after the cardiac arrest and developed refractory cardiac shock, dying 2 days later despite the treatment.

KEYWORDS Electrocardiography; Hysteresis; Pacemaker; Short-long-short sequence; Ventricular tachycardia
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KEY TEACHING POINTS

- Pacemakers carry with them the ability to induce or mediate many forms of tachycardias, such as ventricular tachycardia (VT).
- Short-long-short (SLS) sequences may precede initiation of VT because of abrupt changes in cycle length duration and activation sequence. SLS sequences increase the dispersion of repolarization and the likelihood of unidirectional block and slow conduction, and may simplify reentry.
- Some conditions could facilitate the occurrence of SLS in patients with hysteresis, such as electrolyte imbalances, chronic heart failure, coronary artery disease, drugs, and bradycardia. In patients with permanent or temporary low atrioventricular intrinsic conduction, it would be reasonable to switch off hysteresis mode to avoid SLS and malignant ventricular arrhythmia.

Discussion

SLS sequences may precede initiation of VT because of abrupt changes in cycle length duration and activation sequence. SLS sequences increase the dispersion of repolarization and the likelihood of unidirectional block and slow conduction, and may simplify reentry. The SLS is a characteristic onset pattern for torsades de pointes. The reported frequency of SLS sequence preceding VTs, other than torsades de pointes, varied between 2% and 44%.[2]

Long-term right ventricular pacing is associated with adverse remodeling of the left ventricle,[3] which can contribute to or cause left ventricular systolic dysfunction or overt heart failure.[4] AV hysteresis permits prolongation of the first paced escape interval after a sensed event. The advantage is to maintain AV synchrony and avoid the pacemaker-induced dysfunction. There are algorithms designed to increase the intrinsic AV conduction. Entovis-Biotronik has IRS-plus,

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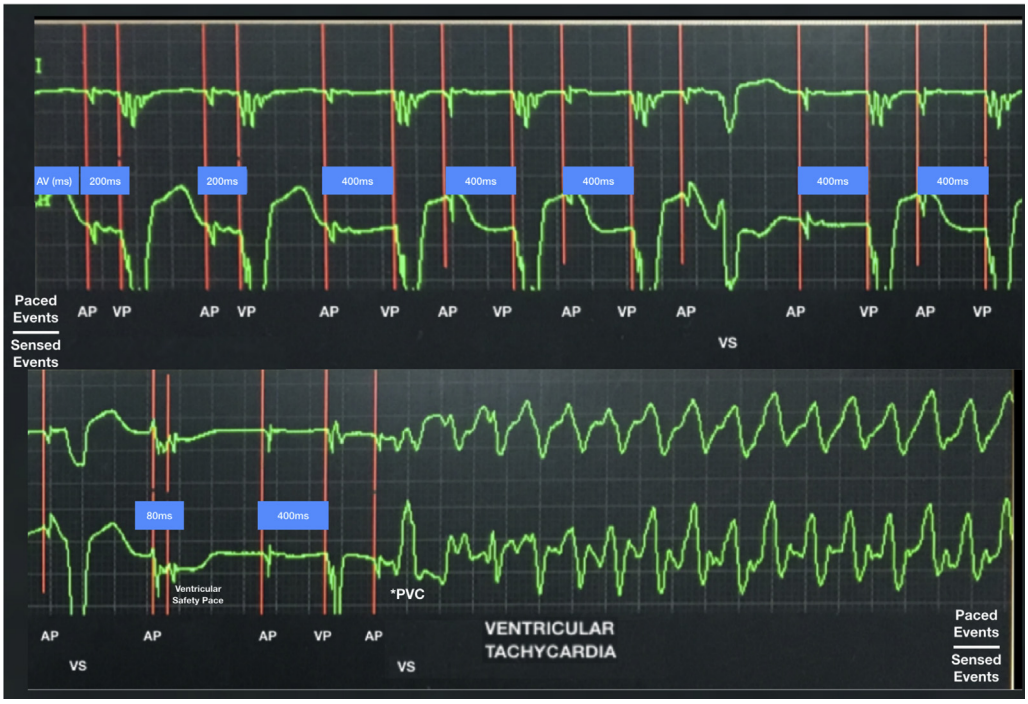

Figure 1 Pacemaker when it started the IRS-plus algorithm. Atrioventricular interval was increased from 200 ms to 400 ms, and then after 3 ventricular paces (VP) there was a premature ventricular contraction (PVC) sensed as a normal ventricular sense (VS), followed by 2 VP and another PVC sensed as a normal VS; then the patient had a ventricular safety pace, followed by another VP and a PVC. Last cycles performed a long-short-long sequence, which may have induced ventricular tachycardia. AP = atrial paced.

which, after 180 consecutive pacing cycles, searches for intrinsic conduction by prolonging the AV delay to 400 ms.[5]

VT in pacemaker patients could be initiated by SLS sequences that might be facilitated by normal operation and might constitute a mechanism of ventricular proarrhythmia.[6]

In the telemetry we could see an SLS sequence trigged by IRS-plus mode on and a sequence of premature ventricular contractions (PVCs). All ventricular sensing was due to PVCs; the patient did not have AV intrinsic conduction at that moment. We might consider the dependency of ventricular cardiac pacing before turning IRS-plus mode on to avoid symptoms and proarrhythmia phenomena. Furthermore, we should switch it off in patients with low AV intrinsic conduction.

There are 2 more things to reduce arrhythmias in these patients: to abolish SLS caused by pacing and to avoid the functional reentry caused by PVCs and right ventricular pacing. We had different possibilities: (1) to increase the lower rate to avoid post-PVC pauses and SLS, and (2) to program the rate-smoothing algorithm with the aim of forcing atrial pacing after long pauses; again, this avoids SLS.[7]

We also believe the patient's previous history of coronary disease and post–carotid bypass surgery condition predisposed the development of VT. Patients with prior myocardial infarction have nonhomogeneous recovery of excitability in the left ventricle. Thus, if an SLS sequence occurs, dispersion of recovery and the likelihood of reentry may further increase.[8,9]

Conclusion

Modern cardiac pacemakers have functions that can prevent many conditions, such as avoiding the pacemaker-induced dyssynchrony through Biotronik IRS-plus hysteresis. We should be cautious before turning IRS-plus on in some patients, because acute stress conditions allied to ischemic heart disease and pacing-dependent ventricle might precipitate fatal VT.

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