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Pacemaker-induced ventricular fibrillation during radiofrequency catheter ablation for ventricular tachycardia

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1 | CASE PRESENTATION

A man aged 80 years with previous inferior myocardial infarction, ischemic cardiomyopathy, and CRT-D underwent ventricular tachycardia ablation under general anesthesia. The patient had a baseline bradycardia, and so his cardiac implantable electronic device (CIED) was temporarily programmed to backup pacing mode (VVI 40bpm) with tachycardia therapies suspended. To reduce risk of inappropriate pacing, noise reversion mode was disabled.

During the procedure, programmed ventricular stimulation both from the right ventricular apex and around the scar areas on the inferior left ventricular myocardium failed to induce ventricular tachycardia. However, during radiofrequency energy application, the patient developed ventricular fibrillation requiring emergent defibrillation. Intracardiac electrogram review showed that the CIED switched to an asynchronous pacing mode during ablation (Figure 1). This resulted in a paced ventricular complex coinciding with the terminal portion of the intrinsic T wave, inducing ventricular fibrillation. All pacing therapies were disabled for the remainder of the case.

2 | DISCUSSION

An increasing number of electrophysiology procedures are performed on patients with CIEDs. The potential effects of radiofrequency catheter ablation on CIEDs include asynchronous pacing and noise reversion pacing modes, reversion to reset parameters, and oversensing causing pacing inhibition. In this patient, asynchronous pacing occurred during radiofrequency catheter ablation even with CIED noise reversion mode disabled. The reason for this occurring is unknown, but we speculate it may have been because of amplifier ringing, which occurs when the amplitude of a sensed signal "overloads" the front end of the sensing circuit, paradoxically resulting in undersensing. In this patient, device-sensed ventricular signal amplitude was low, thereby increasing the likelihood of this phenomenon occurring. As this was a ventricular tachycardia ablation case, external defibrillation was planned and rapidly administered during the event. However, this could potentially occur in any case of catheter ablation in patients fitted with CIED's. Operators should be prepared for such unexpected events to occur. Some CIED manufacturers allow disabling noise reversion mode switching, which inhibits the device safety algorithm switching to asynchronous pacing during radiofrequency application. This case has shown that asynchronous pacing can still occur despite this. Therefore, in nonpacing-dependent patients, disabling pacing therapies may be the safest approach, with pacing via electrophysiological catheters if required. Disabling pacing is also preferable from a technical perspective, as bipolar voltage mapping is best performed in sinus rhythm for the most validated identification of scar regions and border zones. A further potential approach is the administration of intravenous isopretenerol in order to maintain adequate heart rate for patients with underlying bradycardia. However, one must consider the potential risk associated with pharmacological chronotropic support within the context of ischemic heart disease. In pacing-dependent patients, programmed DOO or VOO pacing is recommended to ensure uninterrupted therapy throughout the procedure. In patients with biventricular pacing, it is preferable to switch to single site ventricular pacing if hemodynamically tolerated, because two ventricular activation wavefronts may have significant effects on myocardial scar characterization and reduce procedural

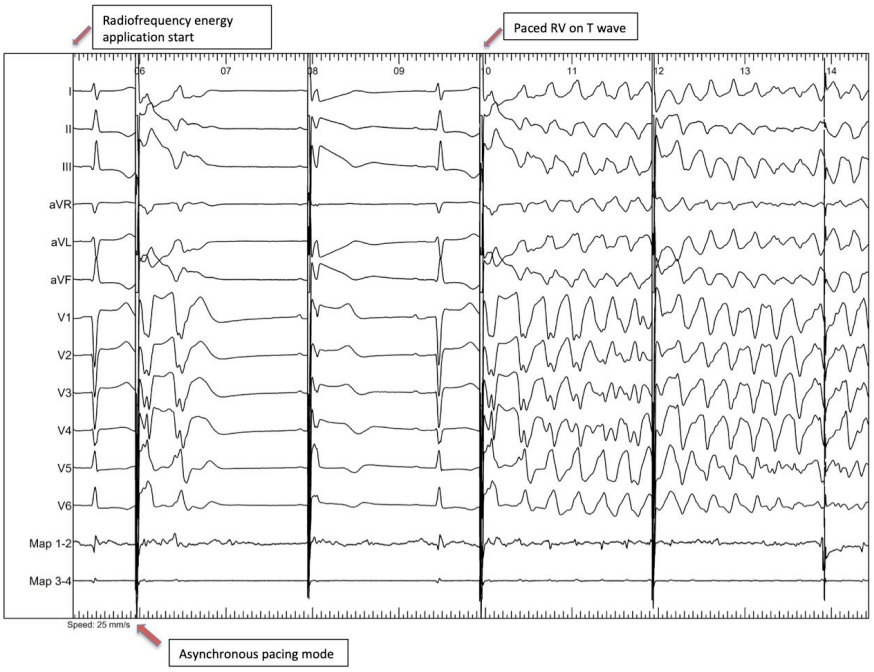

FIGURE 1 Twelve lead ECG recording during radiofrequency ablation energy delivery. Note asynchronous right ventricular pacing resulting in two paced complexes coinciding with the terminal portion of the T wave. The first resulted in two ventricular extrasystoles, while the second resulted in polymorphic ventricular tachycardia and subsequent ventricular fibrillation

efficacy. Lastly, pacing at higher rates may prevent QTc prolongation and reduce likelihood of ectopic-induced malignant arrhythmia.

CONFLICT OF INTEREST

None.

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