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Focus on ECGs: Case #33

Pacemaker-Mediated Tachycardia Interpolated Into Ventricular Tachycardia

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In a 77-year-old patient with a history of inferior myocardial infarction and dual-chamber pacemaker (Effecta DR, Biotronik) implanted for carotid sinus syndrome, a routine Holter electrocardiogram recorded 3 consecutive tachycardias: irregular monomorphic ventricular tachycardia (VT), pacemaker-mediated tachycardia (PMT), and reoccurred VT (re-VT) of the same morphology as the initial one, but regular and faster than the PMT (Fig. 1).

The pacemaker settings were as follows: DDD mode; pacing rate, 50 to 130 bpm; programmed atrioventricular delay, paced 145 ms and sensed 105 ms; and postventricular atrial refractory period (PVARP), 250 ms and 400 ms following premature ventricular contraction.

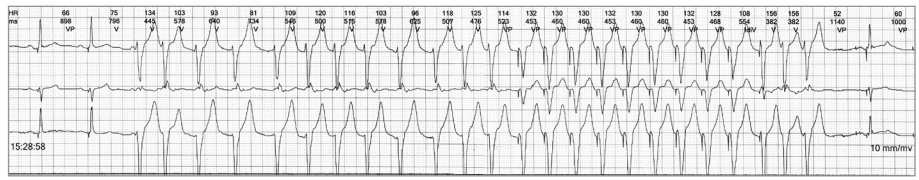

Fig. 1 *Pacemaker-mediated tachycardia interpolated into ventricular tachycardia.*

What was the most likely mechanism of this arrhythmia?

A) PMT was triggered by the VT and terminated by retrograde conduction block

B) PMT was triggered by atrial premature beat and terminated by the re-VT

C) PMT was triggered by external interference and terminated by PMT termination algorithm

D) PMT was triggered by asystolic pause and terminated by shock

See the next page for the answer.

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B) PMT was triggered by atrial premature beat and terminated by the re-VT (Fig. 2)

Concerning the mode of the PMT initiation, the VT could not trigger PMT because its retrograde P wave (in Fig. 2, stars denote retrograde P waves; those of the VT and re-VT are marked below and those of the PMT above the middle electrocardiographic channel) occurred during the PVARP (gray horizontal marks in the ladder diagram). Given that the interrogation showed no pacemaker malfunctions and that the electrocardiogram showed no artifacts, an atrial ectopic beat (with isoelectric P wave) is the only likely trigger for PMT.

Concerning the mode of PMT termination, the episode was too short to activate the device's PMT termination algorithm. Furthermore, the retrograde P wave from the last PMT beat, which appeared after 344 ms, indicates that retrograde conduction has been prolonged, but without occurrence of a block that would have stopped the PMT. Thus, the re-VT is what terminated the PMT by the first impulse following fusion (f2 in Fig. 2), which depolarized ventricles before the possible onset of the next paced beat. Retrograde conduction prolongation (by 24 ms) and programmed atrioventricular delay prolongation (by 50 ms, for Biotronik's PMT status confirmation) during the last PMT cycle provided a time for the onset of the re-VT and thus indirectly participated in the PMT termination.

Based on the constant cycle length (382 ms) of the re-VT during the prolongation of ventriculoatrial conduction (from 170 to 240 ms), the re-VT was most probably microreentrant. Different retrograde conduction times of the VT (160 ms) and PMT (315 ms) might suggest the existence of a dual retrograde conduction with separate pathways for each of the 2 tachycardias, but any evidence for such speculation is lacking. In any case, this case illustrates the complexity of possible interactions between spontaneous and device-mediated arrhythmias.

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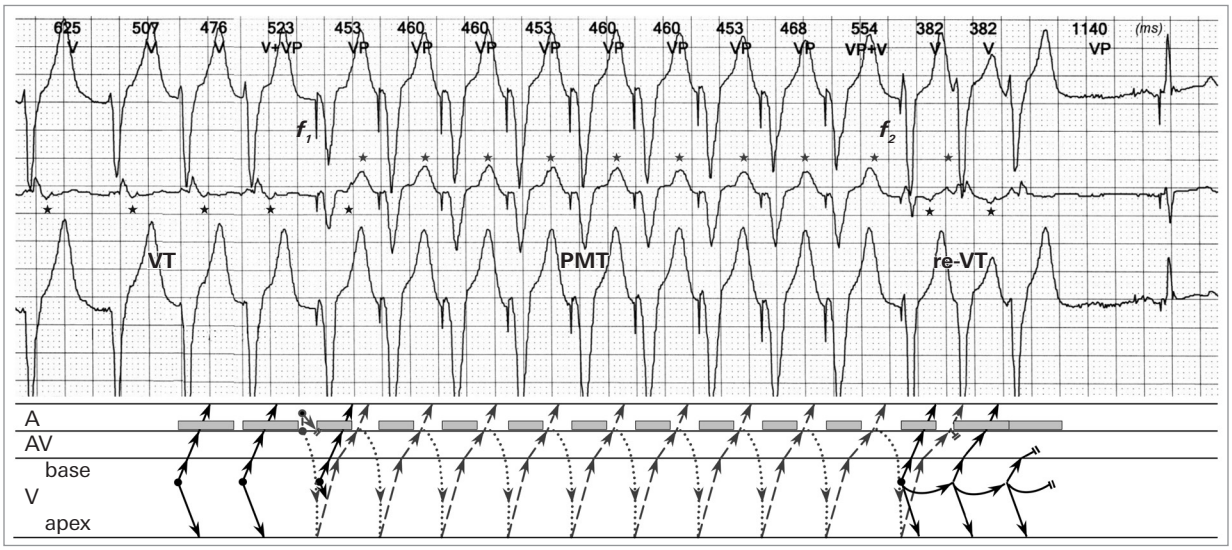

Fig. 2 *Explanation of the arrhythmia mechanism.*