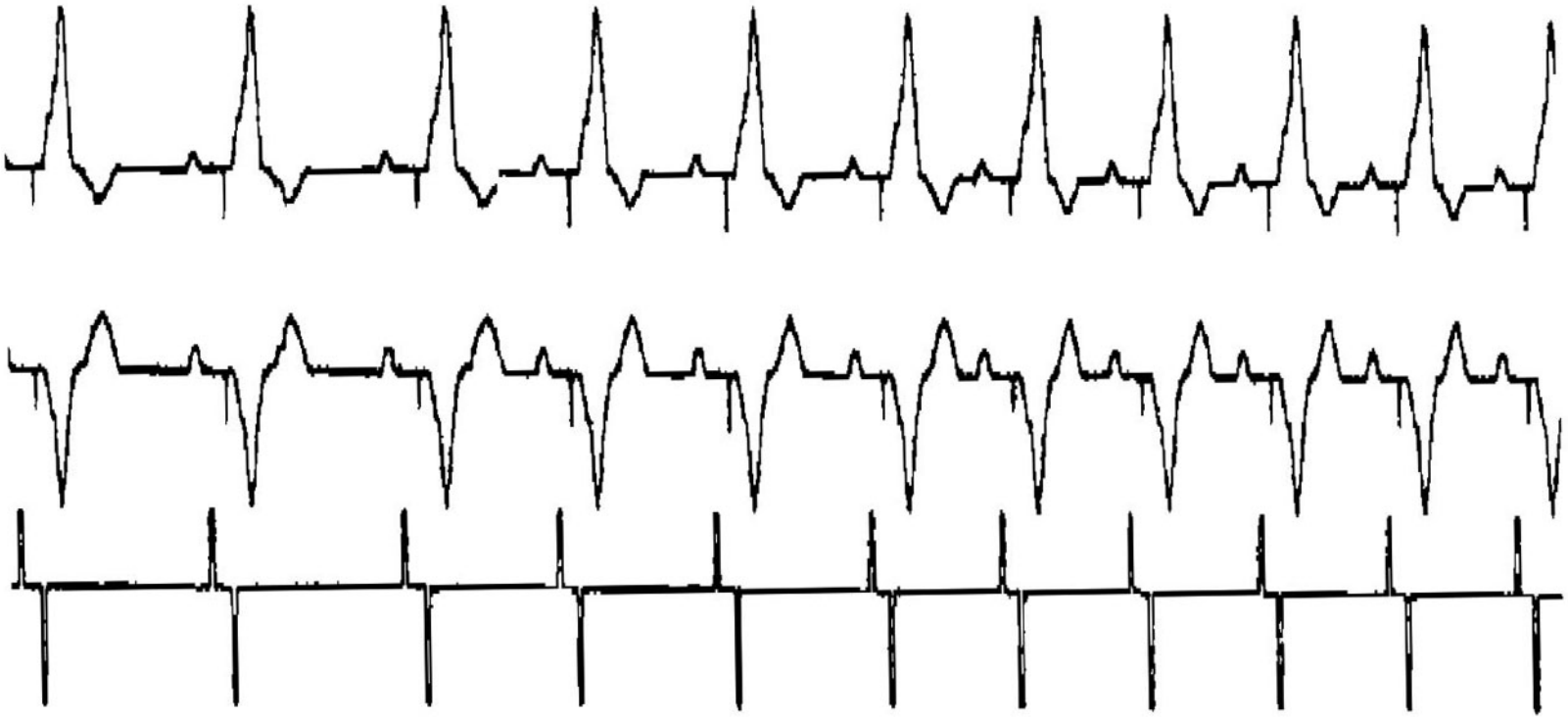

Figure 65.7 Shortening of the AV interval during pacing.

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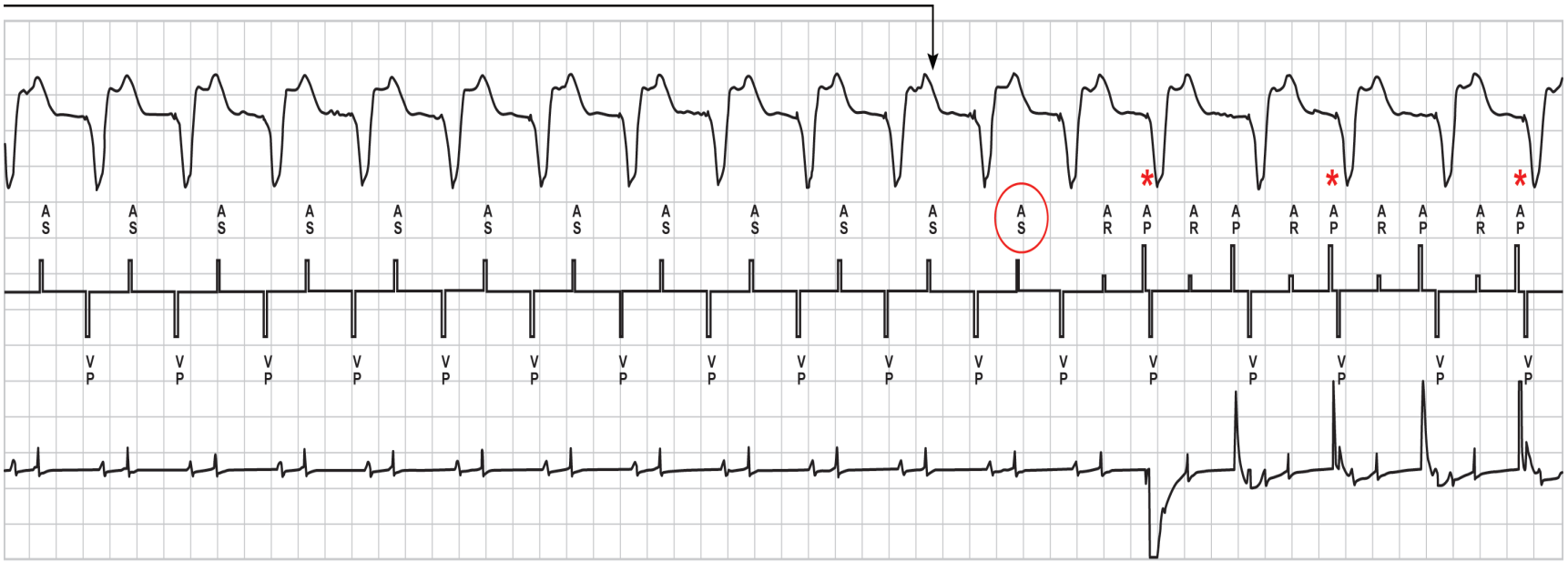
Figure 65.8 is obtained from a patient with a dual-chamber Medtronic pacemaker.



Courtesy of Medtronic, Inc.

Figure 65.8 Patient's tracing. Courtesy of Medtronic.

What is the likely cause of the very short AV intervals noted in Figure 65.8?

1. Negative AV search hysteresis
2. Ventricular safety pacing
3. Noncompetitive atrial pacing
4. Managed ventricular pacing

3. Noncompetitive atrial pacing

Noncompetitive atrial pacing (NCAP, Medtronic) is intended to prevent induction of atrial fibrillation from atrial pacing occurring during the relative refractory period of the atrium. At the beginning of the tracing (Figure 65.8), we note pacemaker-mediated tachycardia (bracket) that terminates with extension of the PVARP leading to a retrograde atrial activation falling in the refractory period. If an atrial pacing stimulus is delivered too soon after the atrial refractory event, atrial tachycardia/fibrillation may be triggered. The atrial paced event if falling in the atrial absolute refractory period will not capture, and AV dyssynchrony may result. Several features are available in present devices to minimize these phenomena (A pace on PVC option, St. Jude Medical). With the NCAP feature, an atrial refractory event in the PVARP triggers a 300-ms interval (NCAP interval) during which no atrial pacing can occur, and the VA *interval is extended*. The pacemaker then attempts to maintain the lower rate by *shortening* the paced AV. Thus, a very short AV interval may be observed, as noted in Figure 65.8, leading to AV dyssynchrony for that cycle. In this case, the AP fails to capture, leading to repeat retrograde conduction and recurrent triggering of NCAP.

In addition to manifesting as unexpectedly short AV intervals, NCAP may also affect atrial and ventricular timing to produce a change in the paced rate compared to programmed parameters. If an atrial pacing stimulus is scheduled to occur during the NCAP period, the VA interval is necessarily extended until this period expires. The pacemaker will attempt to maintain a stable ventricular rate by shortening the paced AV, as noted; however, it will not shorten the paced AV to less than 30 ms. However, when higher lower rates and longer PVARPs are programmed, NCAP operation may result in ventricular pacing slightly below the lower rate.

Another situation where differences in AV intervals may be transiently observed occurs in conjunction with one of the available algorithms to assess adequacy of ventricular capture (capture management, etc.).

In Figure 65.9, a fusion avoidance algorithm associated with auto capture (St. Jude Medical) is diagrammed. Capture is assessed based on whether an evoked response (local EGM) can be sensed after the paced stimulus. At times, fusion from intrinsic conduction may mistakenly create the impression of failure to capture. To allow for this, the AV interval (in the Figure, a bracket) is extended by 100 ms to see if intrinsic conduction (fusion) is occurring and thus varying AV intervals may be noted on telemetry or a Holter monitor.

Managed Ventricular Pacing (MVP, Medtronic) (answer 4) is another algorithm intended to maximize intrinsic conduction.

In a transtelephonic transmission (Figure 65.10), the patient is initially paced AAI with intrinsic AV conduction occurring with a long AV interval (beats 1, 2). There is loss of AV conduction over the third atrial paced beat. Following this transient loss of AV conduction, the device

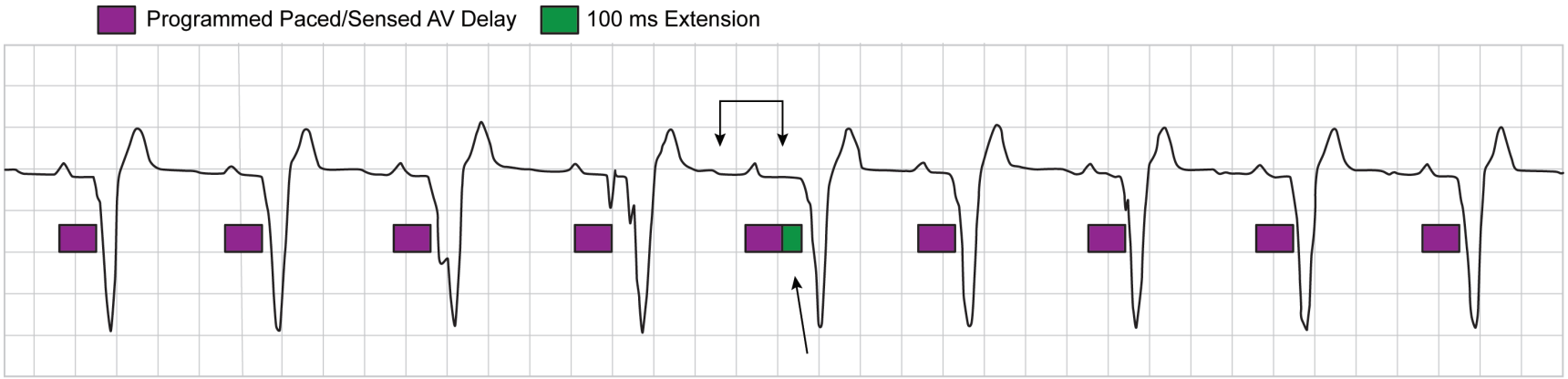


Figure 65.9 Fusion avoidance algorithm.

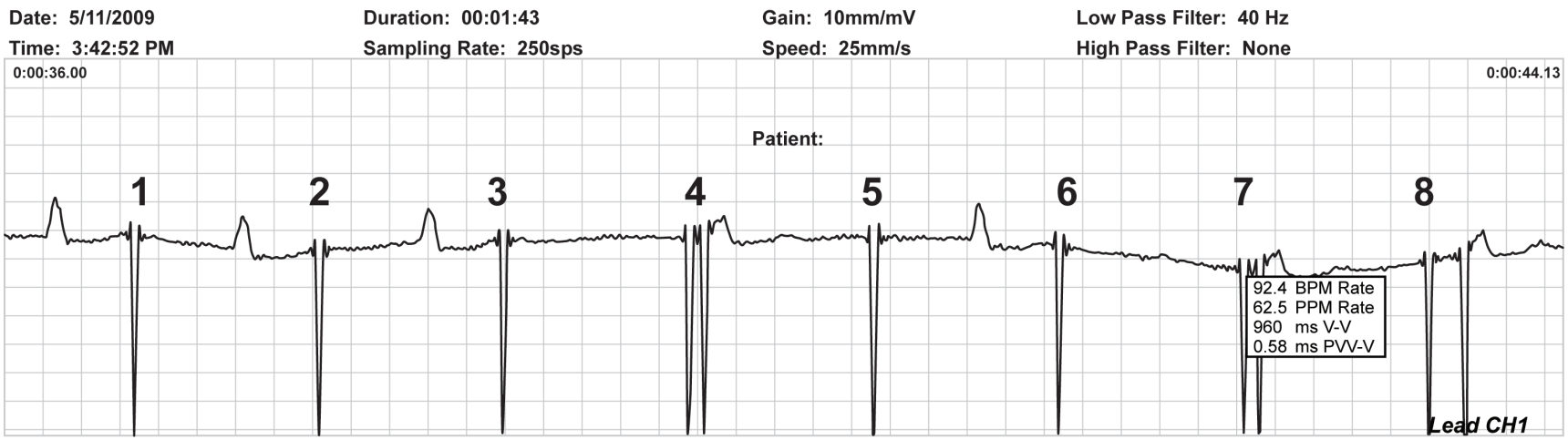


Figure 65.10 Patient's transtelephonic transmission.

delivers a backup ventricular pacing output with a *short* paced AV of 80 ms (beat 4). AAI pacing continues, and when beat 6 fails to conduct, the device delivers another backup ventricular pace (7). Persistent loss of AV conduction defined as the 2 most recent intervals missing a ventricular event is confirmed, and the device switches to DDD mode. The MVP algorithm may thus be associated with detected AV intervals different from the programmed parameters. Other features that may be noted on extended monitoring include:

- Periodic (1 min up to 16 h) single-cycle assessments of AV conduction, and resumption of AAI pacing
- Rarely in patients with 4:3 Wenckebach AV block, since every fourth beat is dropped and 2/4 are required for

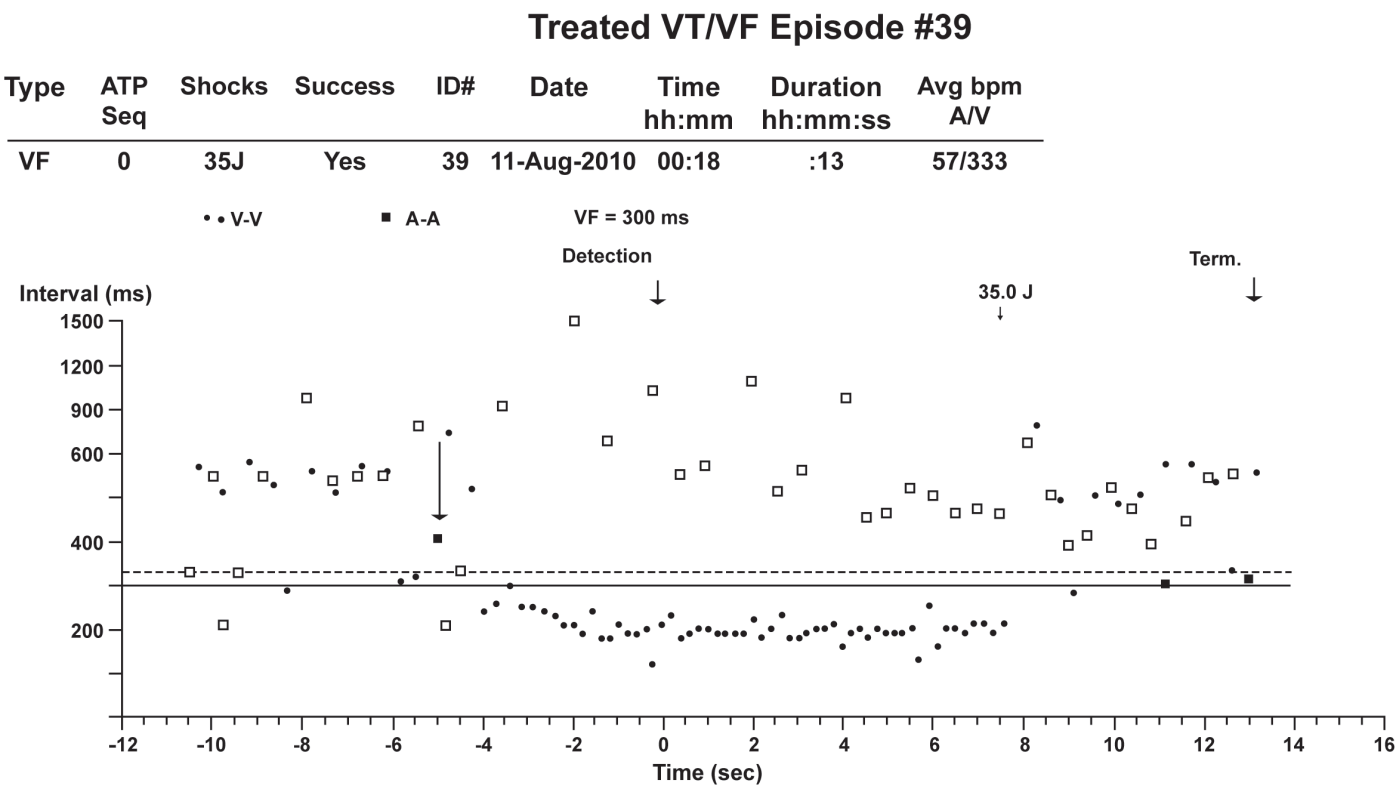


Figure 65.11 Treated VT/VF episode.

reversion to DDD pacing; continued AAI pacing with 4:3 block may continue

- Rarely, the variation in AV conduction and thus ventricular timing may be proarrhythmic (AVNRT induction from prolongation of atrial paced, ventricular sensed intervals) or in conjunction with frequent PVCs occasionally promoting ventricular arrhythmia (short-long-short sequences)

Figures 65.11 and 65.12 are obtained from a patient with abnormal repolarization (long QT syndrome). When switching from AAI to DDD pacing modes, variation in AV timing increased ectopy with further irregularity in ventricular activation promoting recurrent ventricular arrhythmia.

Figure 65.12 Increased ventricular ectopy.

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When the patient was switched to AAIR pacing only (MVP off), the rhythm was stable with less PVCs and no ventricular arrhythmia (Figure 65.13). If AV conduction had been very poor, an alternate method could have been DDD pacing with a stable AV interval. This case, however, is an exception, and in most instances, algorithms such as AV search hysteresis or MVP allow assurance of ventricular activation when needed without unnecessary ventricular pacing.

Another cause of AV interval variability and discordant with programmed parameters is *negative* AV search hysteresis.

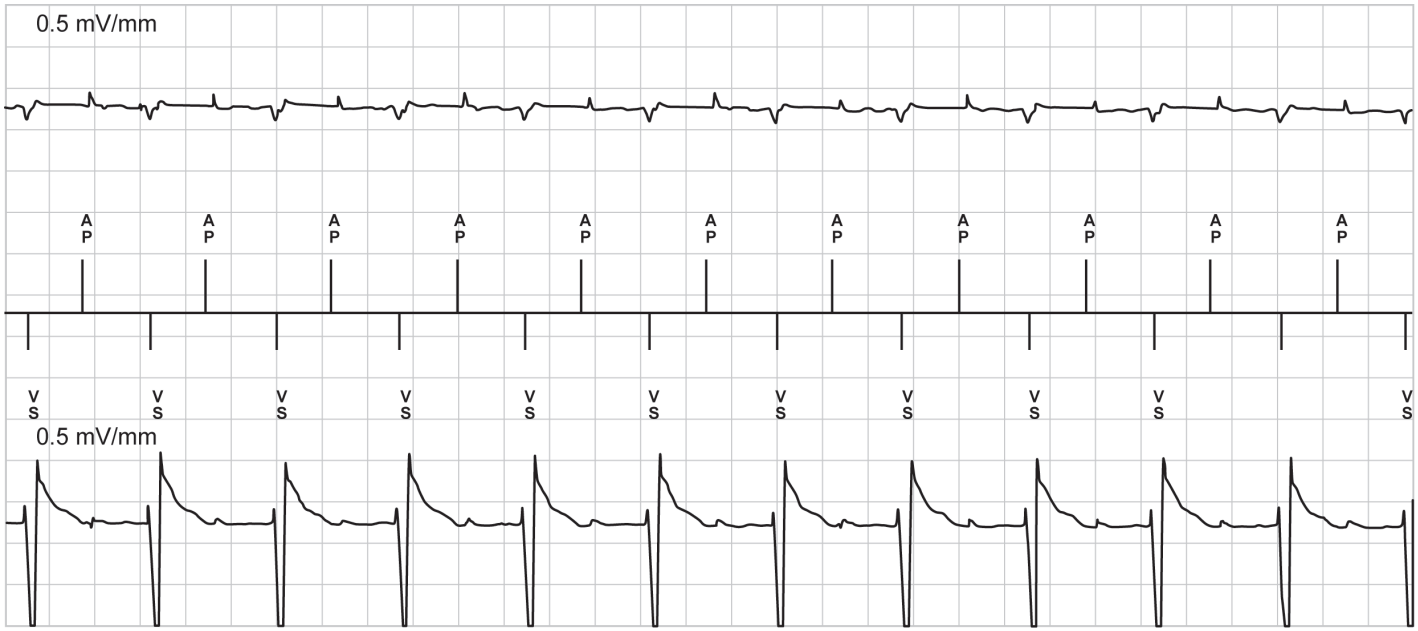

Figure 65.13 Reduced ectopy in AAI mode.

This algorithm is designed to promote ventricular pacing and prevent intrinsic conduct beats. In Figure 65.14, the programmed PVI and AVI are 150 ms and 175 ms, respectively. When intrinsic conduction occurs (AR), the AV interval is shortened with periodic lengthening of the AV interval (first arrow) to see if intrinsic conduction is no longer occurring and a relatively more physiological interval can continue. Negative AV search hysteresis may be useful with CRT devices and in patients with hypertrophic cardiomyopathy where the "pacing" is considered therapeutic and preferred to intrinsic conduction.

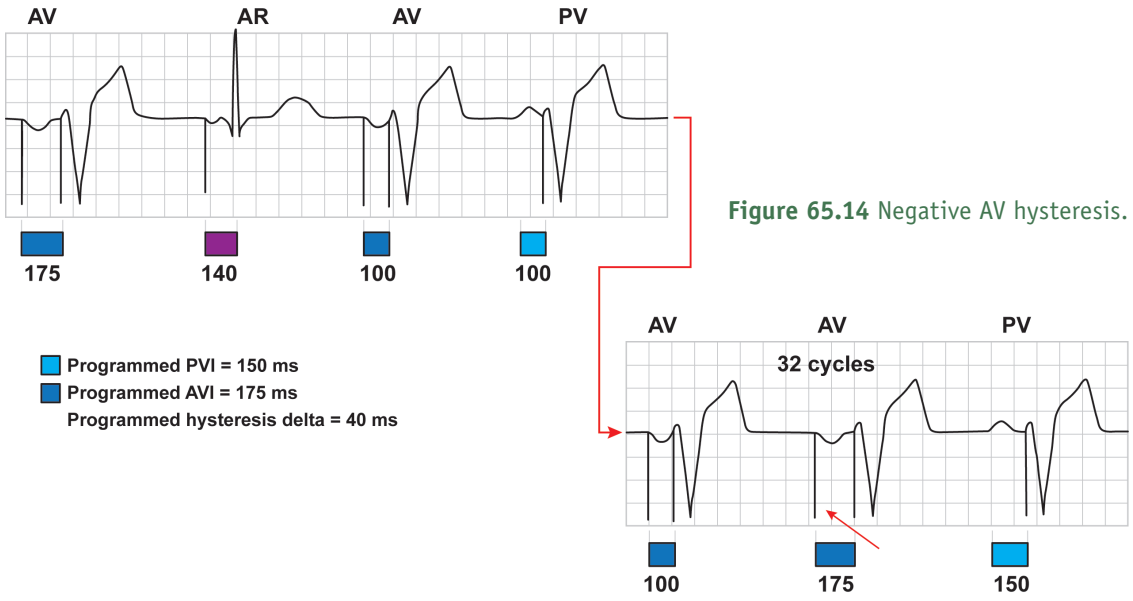

Figure 65.14 Negative AV hysteresis.

In Figure 65.15, initiation and termination of pacemaker-mediated tachycardia (PMT) is shown on a telemetry strip obtained from a patient with a dual-chamber pacemaker implanted 3 months ago for high-grade AV block. The patient presented with palpitation and was confirmed to have PMT.

Device settings:

- Mode: DDDR
- Pacing rate: 60 to 150 bpm
- AV delay: 100 ms; sensed AV delay: 100 ms
- PVARP: 250 ms
- Dynamic AV delay and AV search hysteresis: on

The device was reprogrammed to PVARP of 350 ms, and the upper tracking rate correspondingly decreased. However, as noted in this tracing, the patient continues to experience episodes of PMT.

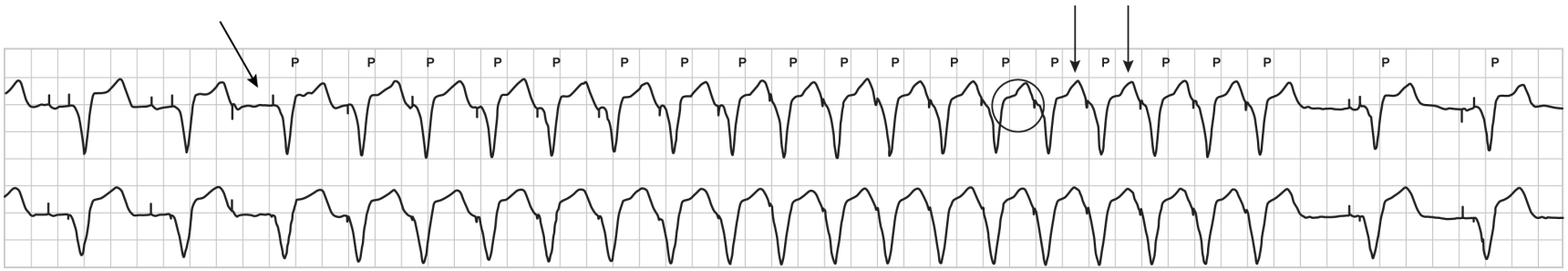

Figure 65.15 Patient's tracing.

Based on the observation in Figure 65.15, which of the following is the next best step in preventing future PMT episodes?

1. Increase PVARP further
2. Program off AV search hysteresis
3. Extend the PVAB
4. Program off dynamic AV delay