Dual Chamber Pacing – Case 1 Discussions

Is the Pacing System Functioning Properly?

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These tracings were recorded during a routine follow-up evaluation for a patient with a Medtronic Elite model 7074 pulse generator that had been implanted in 1993. The original indication for pacing was sinus node dysfunction. The AV delay had been programmed to 300 ms to achieve functional single chamber atrial pacing and programmed base rate is 75 ppm. At the time of this follow-up evaluation, the patient was asymptomatic. The following rhythm strip (Figure 1) was recorded.

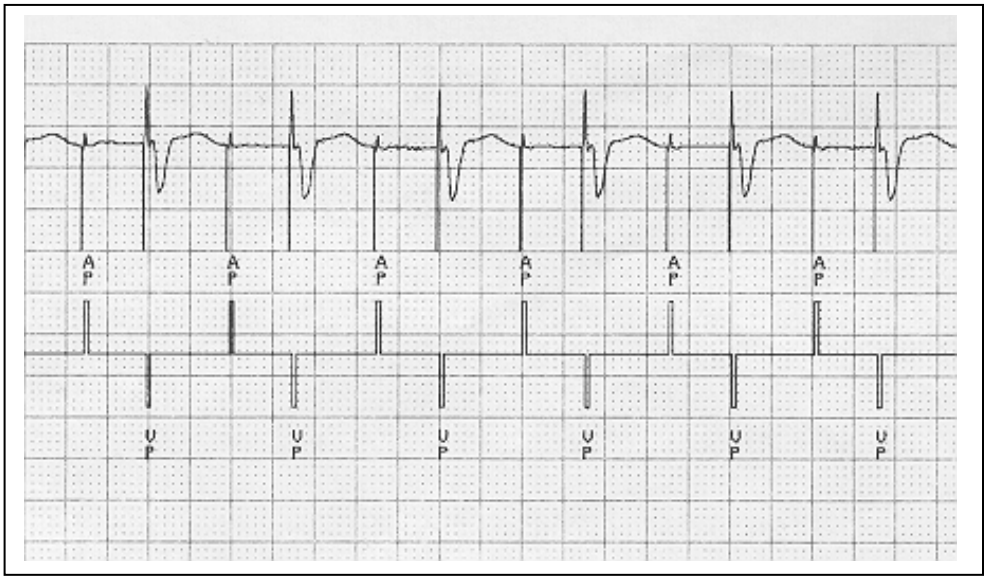

Figure 1: Baseline rhythm strip during a standard pacing system evaluation

Is atrial capture present?
Is ventricular capture present?
How might the pacemaker be programmed to evaluate atrial and ventricular capture?

Diagnosis: Atrial capture is present
Ventricular capture cannot be determined from this rhythm strip

Discussion:

Below the rhythm strip are a series of event markers labeled AP for Atrial Pacing and VP for Ventricular Pacing. These do not mean that capture was present. These simply mean that the pacemaker delivered an atrial output followed by a ventricular output. If one were to place the programming wand over the pulse generator while it was still in the sterile package, it would also demonstrate consistent output pulses but clearly it would not be effective since the pulse generator was not connected to one or more leads that were then connected to the patient.

The rate shown by the rhythm strip is effectively 85 ppm. This is the magnet rate and is consistent with normal battery status in the Medtronic family of pacemakers but the reader was not told that this was a Medtronic pacemaker nor is it pertinent to this discussion. The AV delay during magnet application is the programmed AV interval. In the Medtronic pacemakers, magnet application results in an initial 3 cycles with a foreshortened AV delay to allow assessment of ventricular capture. In addition, the pulse width on the last complex is reduced by 25% as a screening threshold margin test. The ECG was not running when the magnet/telemetry module was initially placed over the pacemaker so this data was not available.

The fact that there are two stimuli with the ventricular stimulus preceding the QRS does not mean that there will not be intact AV nodal conduction at a slower rate. Atrial pacing at an increased rate may induce first degree AV block even in individuals with normal AV nodal conduction. Note that the “paced” QRS is relatively narrow. This suggests that, at a minimum, there is either intact AV nodal conduction with or without fusion. Baseline recordings in this particular ECG lead would be needed to make a more precise diagnosis. Could the entire rhythm strip reflect loss of atrial capture and loss of ventricular capture? Could, in this very short rhythm strip, could the intrinsic rhythm simply be coincidental with the paced output pulses? However, with the relatively narrow QRS, one can be confident that there is atrial capture although a similar level of confidence is not possible with respect to ventricular capture.

To confirm atrial capture and knowing that the original indication for pacing was sinus node dysfunction, the pacemaker was temporarily programmed to the AAI mode (Figure 2).

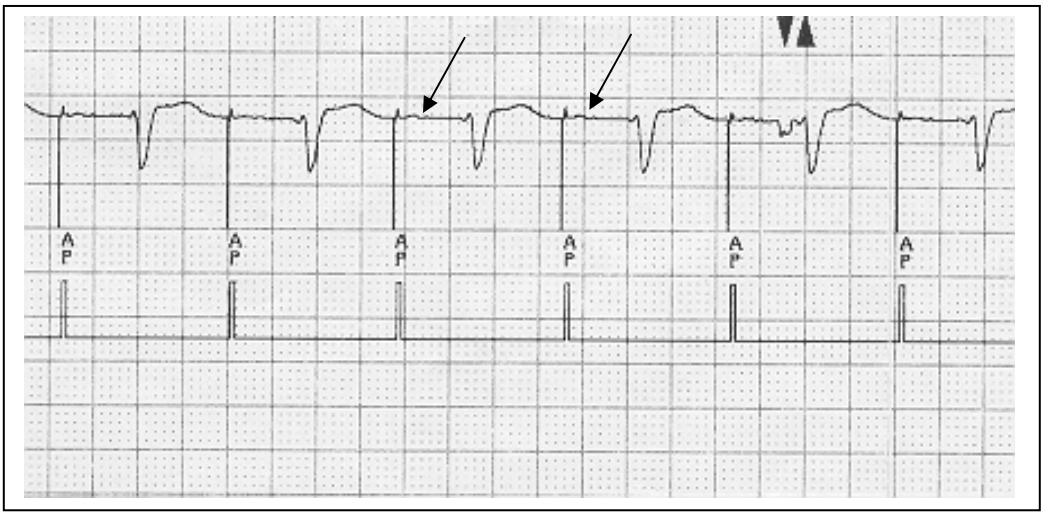

Figure 2: Programmed to the AAI mode at 75 bpm, the heart rate slows from that shown in Figure 1. There is also a visible atrial depolarization (arrow) and a significant first degree AV block. Also, note that the morphology of the QRS complex is identical to that shown in Figure 1. The double solid arrows indicate a programming command delivered to the pulse generator by the programmer.

Based on the AAI mode, now at a rate of 75 ppm which is the slower rate than the baseline recording, there is a stable relationship between the atrial stimulus and the subsequent QRS. If the prior rhythm had been coincidence with loss of atrial capture, the faster rate should persist despite the AAI mode. In addition, if one close closely, a P wave can be seen to follow the atrial stimulus (AP). It is apparent that there is also a significant first degree AV block in addition to atrial capture. The conducted QRS is virtually identical to the QRS associated with "AV" pacing. As such, it is not known if the ventricular output is simply subthreshold or if the prior complex was really fusion with the predominant component being due to intact AV nodal conduction.

To evaluate the ability of the pacing system to effectively capture the ventricle at the programmed output, the AV delay was shortened to 200 ms (Figure 3).

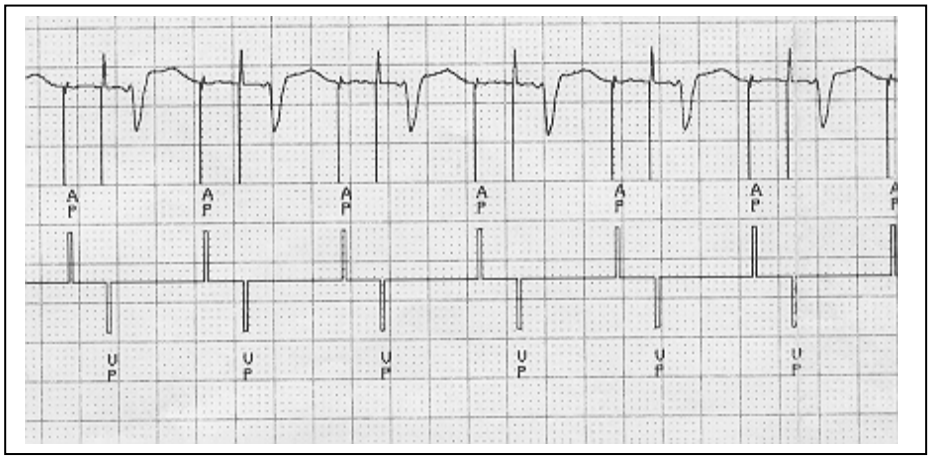

Figure 3: With the AV delay shortened to 200 ms and no change in the ventricular output, the ventricular stimulus is shown to be ineffective.

At the programmed output of 2.5 volts and 0.5 ms pulse duration, there was loss of ventricular capture. The patient was asymptomatic. The etiology for the loss of capture is either a marked increase in the capture threshold, a mechanical problem with the lead such that the delivered energy is not reaching the heart or a component problem with the pacemaker. While a component problem is always included in any differential diagnosis, it is extremely uncommon and should be placed at the bottom of any differential diagnosis. Interrogation of a pacemaker with measured data as to stimulation (lead) impedance will usually demonstrate a mechanical problem with the lead, particularly if the abnormal function is manifest during the evaluation. A high impedance (> 2000 ohms) will identify an open circuit or most commonly a lead fracture while a low impedance (< 200 ohms) will identify an internal short circuit or insulation failure in a bipolar lead. Lead impedance measurements were normal. The third explanation is an increased fibrosis at the electrode-tissue interface. In that setting, simply increasing the output should restore capture.

Capture was restored at an output of 3.3 Volts at 0.12 milliseconds with the pacemaker subsequently programmed to 4.2 Volts at 0.5 ms (Figure 4).

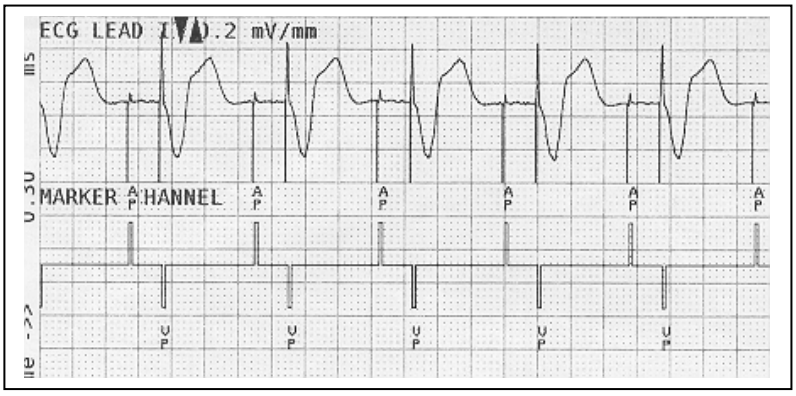

Figure 4: The rhythm strip recorded after increasing the ventricular output and shortening the AV delay.

Lesson: Without baseline tracings for comparison, the coincidence of a pacing stimulus with a QRS complex combined with markers on the programmer printout does not mean that there is capture. To confirm capture, one will need to manipulate the system – in this case the rate was changed combined with a temporary change in the programmed mode from DDD to AAI confirming atrial capture and the AV delay was shortened unmasking loss of ventricular capture. Had the ECG been recorded when the magnet was initially placed over the pacemaker, we would also have seen loss of ventricular capture since the initial magnet application in the Medtronic pacemakers result in a shortening of the AV delay for 3 cycles. This would not have been sufficient to identify the reason for the loss of capture nor the necessary changes to restore normal pacing system function.

Programming: There have been multiple recent studies demonstrating that a disordered ventricular activation sequence associated with ventricular pacing may have deleterious hemodynamic consequences. By the same token, even with "normal" ventricular activation, marked first degree AV block has been shown to be hemodynamically compromising in independent studies by Mabo[1], Jutzy[2] and Vardas[3]. The ACC, AHA and NASPE Guidelines[4] have also elevated symptomatic first degree AV block to a Class II indication for a permanent pacemaker in the most recent Guidelines. If this patient was receiving his initial implant for sinus node dysfunction and first degree AV block in 2004, it would be appropriate to consider alternative sites for the ventricular lead, perhaps the RVOT. The adverse consequence associated with lead placement in the RV apex was not appreciated when this system was implanted more than a decade ago. Rather than subject the patient to an operative intervention to

[1] Mabo P, et al, Permanent DDD pacing for very long PR interval alone, PACE 1992; 15: 509
[2] Jutzy R, et al, Comparison of intrinsic versus paced ventricular function, PACE 1992; 15: 1919-1922
[3] Vardas P, et al, AAIR vs DDDR pacing in patients with impaired sinus node chronotropy, an echocardiographic and cardiopulmonary study, PACE 1997; 20: 1762-1767
[4] Gregoratos G, et al, ACC/AHA/NASPE 2002 Guideline update for implantation of cardiac pacemakers and antiarrhythmia devices, Circulation 2002; 106: 2145-2161

place a new ventricular lead, the patient had normal ventricular function and would very likely be able to compensate for the abnormal ventricular activation sequence associated with pacing from the RV apex. Still, it would be appropriate to try to optimize the AV delay using Echo-Doppler techniques. On balance, AV pacing in this patient was superior to functional AAI pacing with the marked first-degree AV block but to achieve successful ventricular capture, the output had to be increased. The pacemaker was already showing signs of significant battery depletion and increasing the output will accelerate depletion but one needs to be cognizant that the reason for the pacemaker is to treat the patient. The optimal settings of the pacemaker for a given patient should always take precedence over the "longevity" of the pacemaker.

Discussion:

This experience provided another valuable lesson for the clinical team caring for the patient. Transtelephonic monitoring can effectively identify signs of battery depletion but may not be fully able to evaluate capture and sensing. The TTM tracings over the preceding 6 months showed "AV pacing" in both the demand and magnet modes and were interpreted as demonstrating normal atrial and ventricular capture because two stimuli were demonstrated and there was a QRS associated with each ventricular stimulus. Upon retrospective review of all of those tracings, the "paced" QRS complex was relatively narrow suggesting, in retrospect, that there might have been a loss of capture. This further demonstrates the need to periodically perform a detailed assessment of the pacing system including capture and sensing thresholds. In this case, the ventricular capture threshold had markedly increased and first degree AV block had developed. As such, the previous programmed parameters were no longer appropriate for this patient and needed to be changed in a manner analogous to the periodic assessment and adjustment of a medication for any chronic medical condition.