Entrainment mapping is one of the most important tools in the localization of reentrant circuits and identifying critical sites for ablation. However, there are limitations to the use of this technique. This case report illustrates a pitfall of entrainment mapping.

**Background**

A 70-year-old gentleman with known ischemic heart disease was admitted with a 1-month history of exertional dyspnea (New York Heart Association class III) and presyncope. His 12-lead electrocardiograph showed atrial flutter, with 2:1 atrioventricular conduction resulting in a ventricular rate of 100 beats per minute (Figure 1). An echocardiogram showed a dilated left ventricle, with severe impairment of systolic function (estimated ejection fraction of 35%) and significant biatrial enlargement. He was referred for flutter ablation.

**Procedure**

With the patient under conscious sedation, right femoral venous access was obtained. A deflectable decapolar catheter (Dynamic XT, Bard, Lowell, CA) was introduced into the coronary sinus (CS), and a 3.5-mm irrigated-tip catheter (Thermocool, Biosense Webster, Diamond Bar, CA) was introduced into the right atrium (RA). The tachycardia cycle length (TCL) was 274 ms, and CS activation was proximal to distal. Entrainment was initially performed at a cycle length of 250 ms from the proximal CS (CSp) and distal CS bipolar pairs. The postpacing intervals (PPIs) were 341 and 405 ms, respectively. Further entrainment was performed in the RA at the lateral RA wall and cavotricuspid isthmus (CTI), showing PPIs of 343 and 345 ms (online-only Data Supplement Figure). Activation mapping showed that the majority of TCL was measured around the tricuspid valve annulus, activating in a counterclockwise fashion. Although PPIs from entrainment at sites around the tricuspid annulus were significantly >30 ms longer than TCL, the opinion was that this represented a CTI-dependent flutter. CTI ablation was performed at the typical position (6 o’clock in the left anterior oblique view). There was progressive lengthening of the TCL before termination of flutter (online-only Data Supplement Figure). Pacing from CSp at 600 ms showed a conduction time to the lateral CTI of 198 ms. Bidirectional block was confirmed by differential pacing.

Subsequently, a cardiac resynchronization therapy-implantable cardiac defibrillator was implanted. Sensed P-wave measurements at multiple sites in the RA were small (<1 mV), indicating a degree of atrial scarring. Eventually, a position on the lateral right atrium with a sensed P wave of 1.4 V was accepted.

**Discussion**

Conventionally, entrainment mapping allows the identification of sites within a reentrant circuit if the PPI is within 30 ms of the TCL. In this case, the PPI from pacing at the CTI was ≈70 ms longer than the TCL, suggesting that it was not within the flutter circuit. However, it was apparent from activation mapping and termination of the flutter during ablation that this was a CTI-dependent flutter. What was the explanation for the long PPIs when performing entrainment from within the flutter circuit?

The PPIs from pacing at sites around the tricuspid annulus were similar (CSp, 343 ms; CTI, 345 ms; lateral RA, 343 ms) and short compared with the PPI after pacing at distal CS (405 ms). If one assumed that this was a counterclockwise flutter that was entrained during pacing at the CTI and CSp (as shown in Figure 2A–2C), the 345 ms PPI after entraining with pacing at the CTI is a combination of the time taken for the paced impulse to travel around the flutter circuit (y ms) and the journey to and from the CTI pacing site into the flutter circuit (x ms). The following equation can be derived:

\[ y = 2x + 345 \]  

(1)

Because the PPIs from entrainment with pacing from CSp and CTI were almost identical, one can assume that the time taken for the paced wave front to enter the flutter circuit from CSp is also x ms (Figure 2A and 2B). Figure 2B illustrates that the time taken to reach CSp when entraining from the CTI was 143 ms and is made up of the time from the CTI pacing site to reach the circuit (x ms), the time to travel counterclockwise around a portion of the circuit toward the CS, and the time taken to leave the circuit and reach CSp (x ms) and thus incorporates 2x and part of y.
The time taken to reach the CTI when entraining from CSp was measured to be 210 ms (Figure 2C) and represents the time to reach the circuit from the CSp pacing site (x ms), travel in a counterclockwise direction around the remaining portion of the flutter circuit, and then travel from the circuit to the CTI pacing site (y ms); it again incorporates 2x and the remaining part of y.

The sum of these 2 times (143 ms+210 ms) is, therefore, equal to the time taken to travel round the flutter circuit (y ms) plus 4x ms (Figure 2B and 2C).

\[ y + 4x = 353 \]  
(2)

By solving Equations 1 and 2, y is 337 ms and x is 4 ms. This is compatible with the original assumption that both the CTI and CSp are close to the flutter circuit.

During entrainment at 250 ms, the time taken to travel around the flutter circuit was 337 ms compared with the TCL of 274 ms. This would suggest that the longer time during entrainment was because of decremental conduction within the circuit or latency at the pacing sites when pacing at 250 ms (just 20 ms less than TCL). This phenomenon appeared at multiple sites around the flutter circuit. The time from CTI to CSp during flutter was 116 ms (Figure 2D), and it increased to 143 ms during entrainment from CTI (Figure 2B). The time from CSp to CTI during flutter also increased from 154 ms (Figure 2D) to 210 ms during entrainment from CSp (Figure 2C).

Further evidence of decremental conduction in the RA comes from measurements of counterclockwise conduction times around the RA after achieving CTI block. The conduction times to the lateral CTI during CSp pacing at cycle lengths of 600, 900, and 1500 ms were 198, 169, and 166 ms, respectively (Figure 3). This suggests that the decremental conduction manifested to a greater degree at shorter cycle lengths.

There were also clues from the beginning. The flutter cycle length of 274 ms in the absence of any antiarrhythmic drugs is relatively slow compared with typical flutter cycle lengths closer to 200 ms. This may indicate either an abnormally long distance to travel (ie, a large RA) or abnormally slow conduction velocities. The atrial electrograms recorded by the CS catheter also showed significant fractionation, suggesting that the tachycardia cycle length (274 ms) is close to the refractory period of the atrial tissue. The finding of a much enlarged RA on echocardiography suggests there may be an atriopathy. This is further supported by the lack of suitable sites for atrial pacing during insertion of the cardiac resynchronization therapy-implantable cardiac defibrillator.

**Conclusion**

This case is a reminder that decremental conduction in atrial myocardium is a potential pitfall in entrainment mapping, which can be overcome with repeated measurements and a little thought.

**Disclosures**

None.

**References**


EDITOR’S PERSPECTIVE

The postpacing interval (PPI) after entrainment is a useful indicator of the conduction time between the pacing site and the reentry circuit and usually a good reflection of the distance between the pacing site and the reentry circuit. In this Teaching Point, Wong et al show a case of common atrial flutter with a misleadingly long PPI during entrainment from the cavotricuspid isthmus. Prolonged PPIs because of conduction slowing during pacing are not unusual and were observed in 18% of common right atrial flutters in 1 series. It is important to be aware of this limitation of the PPI.

The PPI, measured from the last stimulus to the next depolarization at the pacing site, indicates the conduction time from the pacing site to the circuit, through the circuit, and then back to the pacing site. During pacing at a site that is in the circuit, the conduction time between the pacing site and the circuit is 0, and the PPI equals the tachycardia cycle length, provided that 3 fundamental assumptions are true.2,3 First, pacing must capture and entrain the tachycardia. Second, the electrograms used for measurement of the PPI are because of depolarization at the pacing site. Third, conduction through the reentry circuit must not be slowed or altered by pacing.

What analyses should be considered when the PPI is long in the suspected isthmus site of a reentry circuit? First, consider the assumptions above. Does pacing capture and accelerate all electrograms to the pacing rate? This determination is not always easy. Pacing is usually performed only slightly faster than the tachycardia to reduce the possibility that pacing slows conduction in the reentry circuit (as in this case), alters the reentry circuit, or terminates the tachycardia. It may be necessary to repeat pacing at a faster rate to be certain. Second, make sure that far-field potentials are excluded from measurement. If the electrogram is visible preceding the pacing stimulus, it is clear that the stimulus is not directly depolarizing that tissue, and it is a far-field signal that should be excluded from analysis.6,9 Far-field signals are a common source of a PPI that is falsely shorter than the tachycardia cycle length.

Once these 2 common confounders are absent, there are 3 possibilities remaining. First, the site may be outside the reentry circuit. Second, the arrhythmia may not be because of reentry. Third, that the site may be in the circuit, but conduction in the circuit slows or the reentry path prolongs during pacing. With automatic tachycardias, the PPI will be shortest closest to the reentry circuit but may never be as short as the tachycardia cycle length.7 Activation mapping, starting in the region with the shortest PPI, can be useful. If the entire circuit cycle length is defined, as in this case, macroreentry is supported.6,8 Focal spread away from a point supports a focal tachycardia.

A falsely prolonged PPI because of slowing of conduction in the reentry circuit is beautifully illustrated in this report. Wong et al provide further support for this assumption by pacing from the coronary sinus at different rates after block is present in the cavotricuspid isthmus and measuring the change in conduction time between the CS and the lateral cavotricuspid isthmus on the opposite side of the line of block. With increasing pacing rate, the conduction time prolongs. As the authors indicate, this patient had features of atrial disease, raising the possibility of abnormal atrial conduction. Conduction that behaves in a decremental manner, slowing as the pacing rate increases, will also often result in oscillations of the tachycardia cycle length after pacing. Pacing at progressively faster rates may further prolong the PPI, supporting this mechanism. There are problems with repeated pacing and pacing at faster rates, however. Tachycardia may be terminated or accelerated to another arrhythmia. Finally, ablation at the presumptive critical isthmus terminating tachycardia helps confirm the diagnosis.

In summary, be aware of the limitations of entrainment mapping, including the potential for slowing of conduction during pacing, as shown in this report. When the results do not fit with the clinical picture, obtain additional data, map the activation sequence, and consider ablation targets carefully.

References

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