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Manuscript Title:

Postpacing interval is shorter than tachycardia cycle length - What's the mechanism?

Running Title:

PPI shorter than TCL

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Case presentation:

33year old gentleman who underwent surgery for Tetralogy of Fallot at 5 years of age presented with recurrent episodes of palpitations. ECG taken during the tachycardia showed atypical atrial flutter with right bundle branch aberrancy. His baseline ECG showed sinus rhythm with the same aberrancy. Therapy with a combination of beta-blockers and amiodarone was ineffective.

After stopping amiodarone for 4 weeks, he was taken up for a cardiac electrophysiological study (EPS) with the intent to proceed with 3-D mapping guided Radio-frequency ablation using an Abbott Ensite NAVX Navigation system. Three diagnostic EP catheters were placed for EPS – i) a quadripolar catheter (2-5-2 spacing) at the right ventricular (RV) apex, ii) a

decapolar catheter (2-5-2 spacing) in the coronary sinus (CS), and iii) a duo-decapolar catheter (2-10-2 spacing) - around the tricuspid annulus (TA) with its tip near the os of CS and the proximal poles at the atrial septum. On atrial burst pacing, atrial flutter identical to the clinical tachycardia (positive flutter waves in leads V1, II, III & aVF with an isoelectric interval between the flutter waves) was easily inducible. The tachycardia cycle length was 280ms. The activation pattern in the duo-decapolar showed earliest signal in the mid poles 9-10 [which was along the mid lateral right atrium (LRA)].

Entrainment mapping revealed manifest fusion on pacing from the proximal and distal CS while pacing from the mid Duodecapolar electrodes 9-10 revealed concealed fusion with PPI-TCL of -35 ms as depicted in Figure 1.

What is the mechanism for PPI shorter than the TCL at this site?

Discussion:

Based on the entrainment mapping, we could surmise that the distal and the proximal CS electrodes were distant from the tachycardia re-entrant circuit, while the duodecapolar 9-10 electrodes was either close to the circuit or near the exit zone of the critical isthmus region.

At this point, a high-density activation map of the right atrium was created using Advisor™ HD Grid Mapping Catheter (Abbott Laboratories, Chicago, IL, USA). At the site of interest (SOI) – the mid lateral right atrium, there were highly fragmented signals which spanned 70% of the tachycardia cycle length (Figure 2). Tachycardia was ‘bump’- terminated while mapping this probable isthmus and this site during sinus rhythm showed double potentials with an intervening isoelectric line suggesting prior atriotomy scar or crista terminalis.

The causes of pseudoshort PPI are intermittent capture, spontaneous TCL variability, transient acceleration of the tachycardia, and incorrect annotation of the return cycle^{1,3}. In our case, all these pseudoshort PPI causes were excluded.

We were left with the possibility of down-stream capture, due to high-output pacing resulting in a large virtual electrode, being the mechanism behind the negative PPI-TCL at that site^{1,3}. Additional contributing factors could have been the chance orientation of the pacing electrodes that confronted a zone of anisotropy (involving the narrow critical isthmus) which was overcome by high-output pacing. The capture of adjoining transitioning tissue with better conduction velocity further down-stream in the circuit by the leading edge of the depolarisation wave front, aids in fore-shortening of the return cycle duration. With a smaller pacing virtual electrode, or lower pacing output during the tachycardia, these factors would have prolonged the overall conduction velocity within the circuit and the PPI duration. Further, the size of the virtual electrode showing the truncation of the PPI-TCL duration may not have been sufficient to result in manifest fusion with the given catheter-position and its resolution characteristics.

The sites showing shorter PPI than TCL were commonly located in the region of narrow critical isthmus with slow conduction velocity¹. Since the SOI (mid lateral right atrium) was in such an area, pacing from here showed PPI shorter than TCL. It can be deduced that the tachycardia was due to localised reentry in the region of mid lateral right atrium as we demonstrated reentry with atrial overdrive pacing, simultaneous recording of fragmented signals spanning 70% of the TCL using the HD grid catheter and the entire circuit was confined to the lateral RA wall² (Figure 3). Radiofrequency energy application here was immediately successful in terminating the tachycardia. Ablation was carried out around this isthmus to eliminate all fragmented potentials. Post ablation there was no tachycardia inducible despite aggressive atrial burst and programmed pacing in the presence of isoprenaline.

To our knowledge this is the first case report to demonstrate shorter PPI than TCL in a congenital heart disease substrate. The demonstration of shorter PPI than TCL in the mid-lateral right atrium region aroused our suspicion and prompted extensive mapping in that region.

Conclusion:

The sites demonstrating shorter PPI than TCL are usually markers of slow conducting narrow critical isthmus and hence constitute good ablation targets. This is applicable in a stable tachycardia with constant capture during entrainment where errors in measurement are ruled out.

References:

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Figure legends:

Figure 1: Intracardiac electrogram showing entrainment from midpoles (9-10) of duo-decapolar catheter

From top to bottom, II and V1 represent leads II and lead V1 of 12 lead ECG. CS 9-10 to 1-2 (not in picture) represent proximal to distal poles of decapolar catheter in coronary sinus. DD1-2 represent distal poles and DD19-20 (which are not shown here) represent proximal poles of duo-decapolar catheter.

Figure 2: High density mapping with multipolar catheter showing highly fragmented signals in the mid lateral right atrium covering around 70% of TCL.

Figure 3: Activation map of the atrial flutter showing nearly the entire cycle length of the flutter circuit confined to the lateral right atrium with early (red colour coded) meeting the late (purple colour coded). Arrows indicate the probable reentry circuit of the atypical atrial flutter and the squiggly arrows represent zone of slow conduction in the critical isthmus. The right half of the figure demonstrates high out pacing leading to a large virtual electrode and downstream capture of tissue. PPI – Postpacing interval, TCL – Tachycardia cycle length