Changing atrial activation patterns during narrow complex tachycardia

Ramalingam Vadivelu[1], Yash Lokhandawala[1], and Raghav Bansal[1]

[1]Bandra Holy Family Hospital and Research Centre

July 7, 2020

A 53- year old woman presented with recurrent episodes of paroxysmal tachycardia with a structurally normal heart. She had undergone an electrophysiology study in an outside hospital which revealed orthodromic atrioventricular reentrant tachycardia (ORT) using a concealed left posterolateral accessory pathway (AP), for which radiofrequency ablation was performed. But tachycardia recurred three months later. In view of significant symptoms, she came to us for a repeat procedure. The AH and HV intervals were 77 ms and 45 ms respectively. Narrow complex tachycardia with a cycle length (CL) of 300 ms was easily and repeatedly induced and terminated (Figure 1, left panel) by a premature ventricular complex (PVC). Shortly another tachycardia with CL of 255 ms was induced and terminated (Figure 1, right panel) by a premature ventricular complex. Later, during another tachycardia, a spontaneous change was seen (Figure 2). Are we dealing with a single tachycardia mechanism?

Discussion

The tachycardia in Figure 1, left panel, shows an eccentric atrial activation pattern with the earliest 'A' in channels CS34 and CS12, located in the left posterolateral region. The ventriculoatrial (VA) time even at this site is quite long and there is no bracketing. A His-refractory PVC delivered from the left ventricle terminates the tachycardia without activation of the atrium, confirming ORT as the mechanism, using a left free wall accessory pathway. The second tachycardia (Figure 1, right panel) has a clearly different atrial activation pattern with the earliest atrial activation in CS78 and CS910, near the mouth of the coronary sinus, though with a relatively long VA interval. This is also an ORT, being terminated by a His-refractory PVC delivered from the left ventricle, without activation of the atrium. Hence the differential diagnoses include: i) Only 1 left free wall accessory pathway, with intermittent local periannular (mitral isthmus) conduction block resulting in coronary sinus disconnection, due to the previous ablation; ii) Two APs, left lateral and inferoparaseptal (posteroseptal) and iii) Two APs, left lateral and right sided. A single left ventricular His-refractory PVC terminating tachycardia made a right sided AP unlikely and this was ruled out by mapping the tricuspid annulus during the tachycardia.

Figure 2 shows tachycardia with two different atrial activation patterns. A later His-refractory PVC from the left ventricle results in following changes: i) Fusion QRS complex; ii) Prolongation of tachycardia CL from 255 ms to 300 ms and iii) Change in mitral annular atrial activation pattern from concentric to eccentric with a longer initial local VA time. So here differential diagnoses could be either 1) Two left sided APs, the ORT utilising the left posterior AP being terminated by the PVC and the ORT utilising the left lateral AP being immediately initiated or ii) ORT using the same left lateral AP, with concentric mitral annular atrial activation in the initial two beats due to local mitral isthmus block; the PVC 'peels away' this conduction block.

The left Panel of Figure 3 explains the concentric activation pattern. Local conduction block due to previous ablation does not allow the coronary sinus to get activated from the atrial end of the AP during tachycardia. The activation then could have proceeded up the left atrium to the Bachman's bundle and thence, to the

right atrium. However, in that case the atrium in the peri-AV nodal area would have been activated before the coronary sinus ostium region. In our case, the coronary sinus ostium is activated earlier, with the 'A' in the His bundle region later. So the previous ablation energies had also produced a conduction block higher up, preventing the Bachman's bundle from getting engaged. This is further confirmed by Figure 2, where we see that the 'A' maintains the same rsr'S'R" morphology in the HISD channel and the same delay after the 'A' in CS910 during both types of tachycardia.

Hence it was decided to map in the lateral mitral annular region during left ventricular pacing. At the left posterolateral region 1 cm above the mitral annulus, the earliest fragmented atrial activation was found. Upon starting radiofrequency energy the AP conduction was immediately abolished and retrograde AV nodal conduction was seen. After this successful ablation, complete VA block was seen with adenosine during left ventricular pacing. Despite isoprenaline, no AP conduction was seen and no tachycardia was inducible.

Left atrial- coronary sinus disconnection, also termed mitral isthmus conduction delay or block, is a peculiar phenomenon that can occur during ablation of a left free wall AP, when several energies are delivered inferiorly while the actual AP inserts higher up.[1,2] This is one of the important reasons for failure of ablation, as the operator may erroneously think that the AP pathway conduction was abolished when retrograde conduction changes from an eccentric to a concentric pattern.

References

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