Narrow complex tachycardia with alternating atrial activation: What is the Mechanism?

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Abstract

Case Presentation: A 50-year male was referred for consideration of a redo ablation procedure. 2 years before this referral, he was diagnosed with an episode of pre-excited atrial fibrillation, and underwent an attempt at a left lateral pathway ablation. The index attempt with radiofrequency ablation did not eliminate the pathway and he was discharged on oral flecainide. The procedure was also complicated by a right-sided deep vein thrombosis and further attempts at a redo procedure were delayed. However, he developed recurrent episodes of palpitations 2 years after his index procedure. Hence, he was referred for a second attempt at ablation. His baseline 12-lead ECG did not show any evidence of manifest pre-excitation. During his EP study, pre-excitation was seen with atrial extrastimuli and with shorter intervals, a narrow complex tachycardia was induced (Figure [1](#fig-cap-0001)). However, the atrial activation appeared to alternate at times, despite the tachycardia being sustained. What is the mechanism of the alternating atrial activation?

EP Rounds:

Narrow complex tachycardia with alternating atrial activation: What is the Mechanism?

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Case Presentation:

A 50-year male was referred for consideration of a redo ablation procedure. 2 years before this referral, he was diagnosed with an episode of pre-excited atrial fibrillation, and underwent an attempt at a left lateral pathway ablation. The index attempt with radiofrequency ablation did not eliminate the pathway and he was discharged on oral flecainide. The procedure was also complicated by a right-sided deep vein thrombosis and further attempts at a redo procedure were delayed. However, he developed recurrent episodes of palpitations

2 years after his index procedure. Hence, he was referred for a second attempt at ablation. His baseline 12-lead ECG did not show any evidence of manifest pre-excitation. During his EP study, pre-excitation was seen with atrial extrastimuli and with shorter intervals, a narrow complex tachycardia was induced (Figure 1). However, the atrial activation appeared to alternate at times, despite the tachycardia being sustained.

What is the mechanism of the alternating atrial activation?

Discussion:

The tracing in Figure 1 demonstrates a 1:1 narrow complex tachycardia with alternating atrial activation. The atrial activation appears to alternate between "concentric" activation and eccentric activation. However, a closer look demonstrates that the distal CS activates the earliest in both cases. A differential diagnosis of this tracing includes AVNRT with intermittent conduction via a bystander pathway, AVNRT with intermittent premature atrial contractions, junctional tachycardia with intermittent conduction via a bystander accessory pathway, and finally, AVRT with intermittent mitral isthmus block.

The tachycardia had a VAHV response with RVA overdrive pacing, ruling out atrial tachycardia as the mechanism. The VA time was 118msec, and a His-refractory PVC during tachycardia advanced the atrium and reset the tachycardia. The post pacing interval minus tachycardia cycle length was 23 msec. Hence, the most likely diagnosis is AVRT.

A closer look at the tachycardia itself demonstrated some interesting observations (Figure 2). The TCL, VA time (measured to CS 1,2) and H-H intervals were all fixed, irrespective of the pattern of retrograde atrial activation. In addition, there appears to be a double atrial potential on CS 3,4 on the alternate beats. Given the history of a previous attempt at ablation of the left lateral pathway, a conclusion can be made that lesions where also delivered to the mitral isthmus, creating a functional line of block. The latter allowed for intermittent conduction in a retrograde fashion into the AV node across the mitral isthmus with every other beat. The beats with the "Pattern A" retrograde atrial activation (Figure 3a) conducted retrogradely via the left lateral AP, across the mitral isthmus in a lateral to septal fashion and subsequently to the AV node. In contrast, the beats with "Pattern B" conduct to the AV node via the roof of the left atrium (Figure 3b). The likely explanation for this is that with Pattern B, block occurs across the mitral isthmus and retrograde activation must occur via the roof of the left atrium for the tachycardia to be sustained. Interestingly, the tachycardia circuit of AVRT is sustained, despite the difference in retrograde activation. The likely explanation for this is that the electrical activation fortuitously reaches the AV node at the same time with each pattern, despite the two different circuits. There is likely some delay in conduction across the mitral isthmus with Pattern A, allowing a similar VA time to Pattern B despite the longer anatomical distance via the roof.

Mitral isthmus conduction block can occur in patients redo procedures for left lateral pathway ablations and most likely occurs due to poor mapping or unstable catheter positioning. The operator can be fooled by visualizing "concentric" atrial activation given the mitral isthmus line of block [1]. Voltage mapping can mitigate this issue by demonstrating scarring, low voltage and fractionation over the mitral isthmus.

The left lateral pathway was successfully ablated with mapping on the mitral annulus during ventricular pacing, with demonstratable VA block during ventricular pacing and failure to reinduce tachycardia.

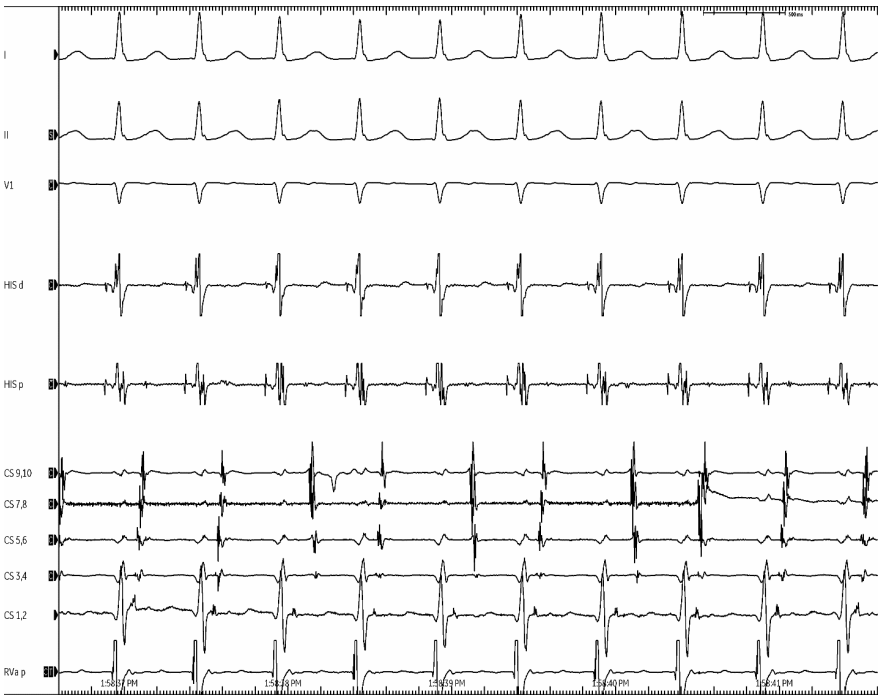

Figure 1: Narrow complex tachycardia with alternating atrial activation.

Figure 2: Narrow complex tachycardia with alternating retrograde atrial activation. The TCL, VA time and H-H intervals are all fixed. TCL = Tachycardia cycle length, H-H= His-His

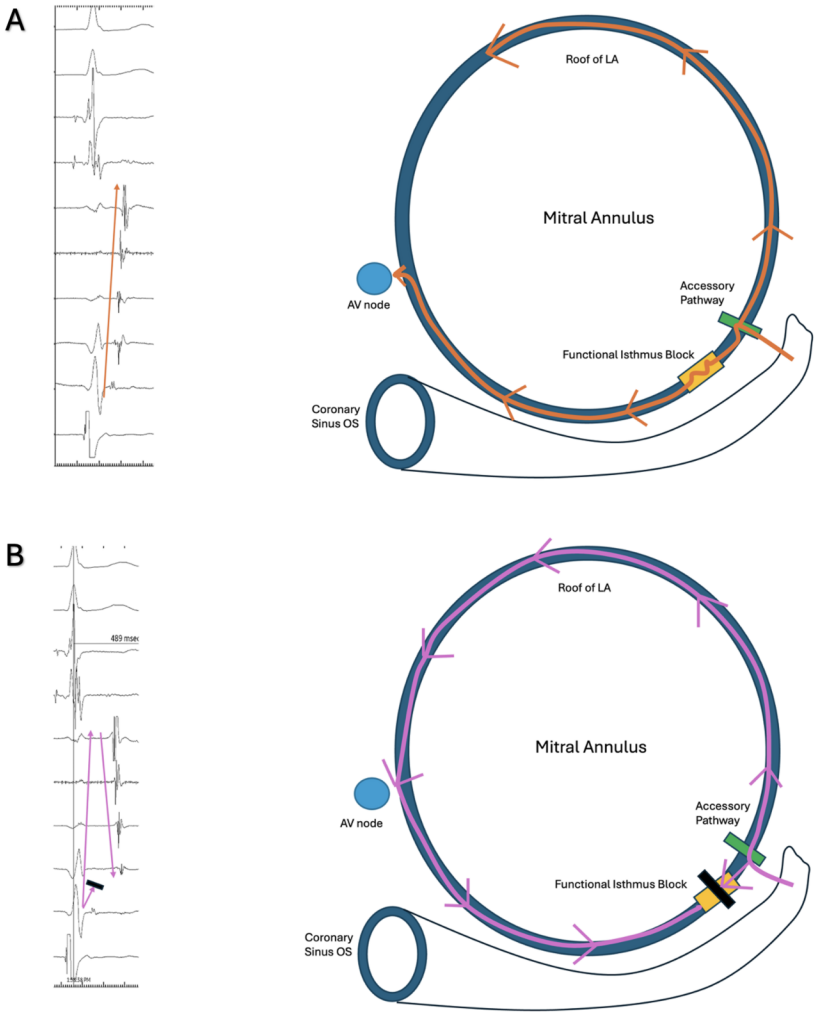

Figure 3: A schematic illustration of the findings seen in figures 1 and 2. 3A: Retrograde atrial activation with Pattern A via the accessory pathway and subsequently lateral to septal via the mitral isthmus and then the AV node. 3B. Pattern B occurs due to functional block across the mitral isthmus, and subsequent activation of the LA in a counterclockwise fashion via the roof, atrial septum and into the node. The "concentric" pattern is seen following activation of the CS from this same impulse.

Reference:

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