Title: Supraventricular tachycardia with shifting atrial activation patterns caused by extrastimuli: What is the mechanism?

Short title: SVT with changing atrial activation patterns

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Case summary:

A 45-year old female presented with recurrent symptomatic palpitations responsive to adenosine. An electrophysiology study was performed with decapolar catheter in the coronary sinus (CS) and quadripolar catheters in the His and right ventricular (RV) apex. A concealed retrograde decremental left posterolateral accessory pathway (AP) with ERP of 340ms was confirmed to be mediating an orthodromic atrioventricular re-entry (ORT; tachycardia-1; TCL-370ms; VA interval- 230ms). After an attempted first radiofrequency (RF) burn at the posterolateral mitral annulus (MA) which was delivered for 20 sec, there was reproducible initiation of tachycardia-2, which had a prolonged VA time (270ms), slower cycle length (410ms) and change in atrial activation pattern. (Figure 1) There were different instances of conversion of tachycardia-1 to tachycardia-2 and vice versa with varying CS activation patterns, which were observed with premature ventricular complexes (PVCs) (Figure 1) and premature atrial complexes (PACs) (Figure 2A, B). What is the likely mechanism behind the shifting activation patterns?

Commentary:

Delivery of His-synchronous PVCs from the left ventricle (LV) reproducibly reset the tachycardia-2 (Figure 2C). Thus, participation of a right sided AP or AVNRT with left sided atrial extensions or an atypical AVNRT were unlikely. Mapping the mitral annulus confirmed earlier activation of the left posterolateral mitral annulus both during tachycardia-1 and 2. (Figure 2D) Further RF burns were delivered at the lateral mitral annulus which was 8 mm lateral to the earlier lesion and at the earliest atrial insertion. This terminated the tachycardia-2. Persisting AP conduction or additional APs were ruled out by documenting absent VA conduction during VA block induced by adenosine.

A fixed concentric (proximal to distal CS) atrial activation pattern observed during ablation of left lateral accessory pathways has been described as a sequel to mitral isthmus block.[1] However, shifting activation patterns in response to PVCs and PACs and the mechanisms behind are the intriguing features of this case. The proposed mechanism should account for both change in the atrial activation pattern as well as the change in the conduction properties of the accessory pathway. (Figure 3) The changing activation between two set patterns suggest persisting conduction between the surviving bundles of the same AP or two different closely spaced APs. Manifestation of a second left lateral AP in form of tachycardia-2 is unlikely as it was seen only after the first RF burn and more so because the final RF burn terminating the tachycardia was at the same region albeit at a more proximal atrial insertion site. The different atrial activation patterns can be explained by block in the medial bundle of the AP leading to counterclockwise activation of the mitral annulus. (Figure 3) This was also confirmed by mapping the CS distal to the site of the first RF lesion.

Longitudinal dissociation of accessory pathways has been described, wherein the cycle length of the tachycardia changes however with the same atrial activation pattern.[2] In this particular case, in spite of the changing atrial activation patterns observed, we propose an acquired form of longitudinal dissociation of the accessory pathway due to an incomplete RF burn to explain the interchanging activation patterns. (demonstrated in figure 3) The effect of extra stimuli give evidence for shift in conduction from a certain bundle of fibres within the AP to the other. In figure 1, the PVC has rendered one of the limbs of the AP refractory leading to shift in conduction to the other limb. The effect of PACs is seen in figure 2A, B wherein the early and late coupled PACs have advanced the subsequent His and the ventricular electrogram. As a result, the retrograde conduction had shifted from one of the refractory limbs of the AP to the other. It has to be noted that the shift in the VA timing observed during tachycardia-2 was not dependent on any change in AH intervals. The prolongation of the VA timing and the cycle

length of the tachycardia could be explained by the lengthening of the circuit as it would take more time for the impulse to reach the AV node due to the mitral isthmus delay. Rate-related refractoriness in the AP is also unlikely as the tachycardia-2 was manifest with a longer cycle length.

Thus, this case demonstrates that longitudinal dissociation of a concealed AP is possible by an incomplete RF lesion and in such a setting the extrastimuli can expose the differential refractoriness within the surviving bundles of the AP leading to shifting conduction between the dissociated limbs of the AP. This can further lead to remarkable change in atrial activation patterns due to the mitral isthmus delay in one of the limbs of the AP.

References:

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Figure legends:

Figure 1 shows a programmed premature ventricular complexes (PVC) causing the conversion of tachycardia-1 (TCL-370ms) to tachycardia-2 (TCL-410ms); Note the prolongation of the VA interval and the cycle length of the tachycardia along with the change in the activation pattern of the coronary sinus (CS-1,2 represents distal electrodes); All intervals are in milliseconds

Figure 2 shows multiple instances of conversion between tachycardia-1 (TCL-370ms) and tachycardia-2 (TCL-410ms) with spontaneous early premature atrial complexes (panel A) or with programmed late coupled atrial extra-stimuli (panel B)- note the advancement of the subsequent His and Ventricular electrograms followed by change in activation pattern and shift between the tachycardias; The figure also shows resetting of the tachycardia-2 with premature ventricular complexes (PVCs) from the LV (panel C) and earliest atrial activation (red dotted line) mapped using the ablation catheter (RF-distal) alongside the lateral mitral annulus (panel D); The premature complexes are annotated as *; All intervals are in milliseconds

Figure 3 is an illustration with a ladder diagram to show the longitudinal dissociation of the accessory pathway (AP) after the radiofrequency ablation (RFA) lesion and block in one of the limbs of the AP caused by a premature ventricular complex (PVC) leading to shift of conduction to the other limb of the AP (panel A); The figure also demonstrates change in the atrial activation pattern due to block in one of the limbs of the AP (panel B, C)

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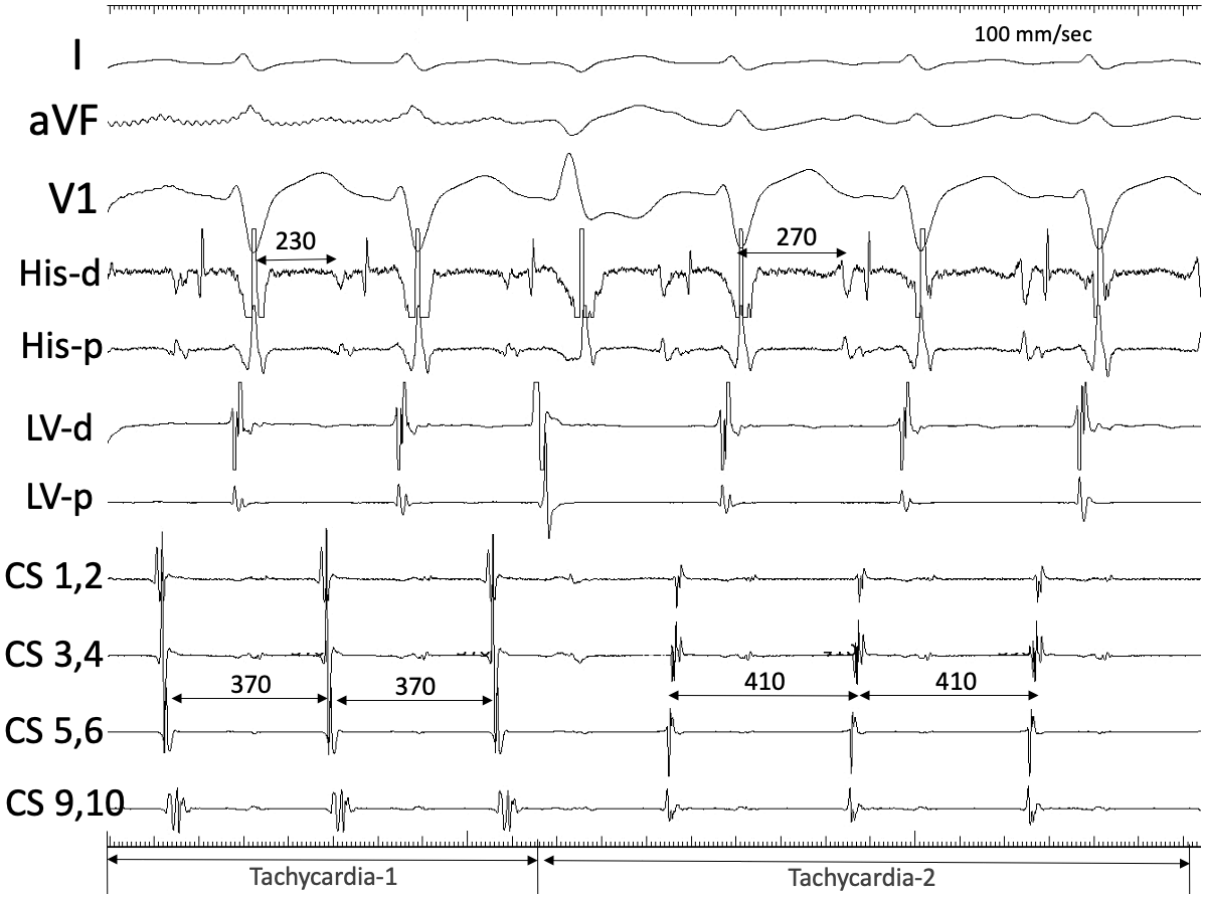

Figure 2

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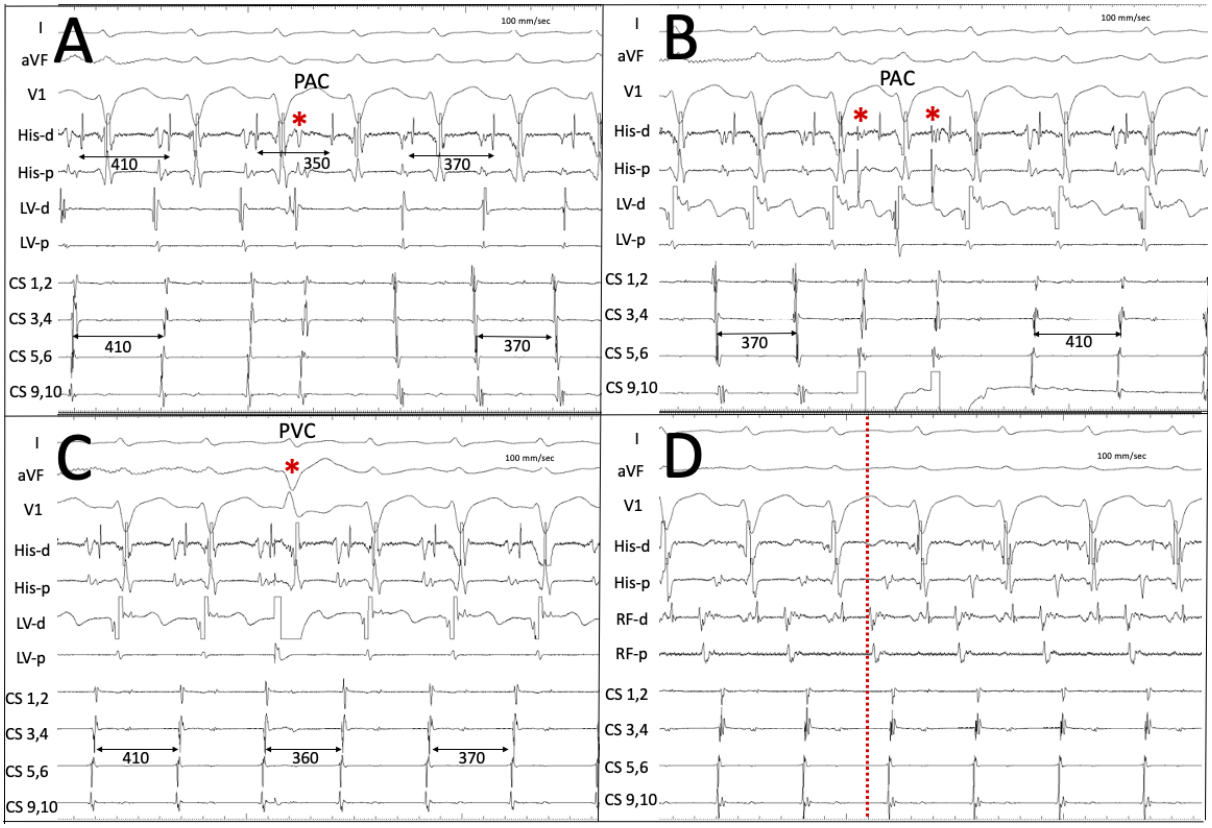

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