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A narrow QRS tachycardia with changed atrial activation after previous ablations: what is the mechanism?

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Short title: coronary sinus activation signal disconnection

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Case presentation

A 25 year-old female presented following an episode of pre-excited atrial fibrillation (AF) following three previous failed ablations for Wolf-Parkinson-White syndrome (WPW) at another institution over the preceding 2 years, with a diagnosis of a more than one left sided accessory pathway.

The electrophysiology study (EPS) demonstrated a short HV interval of 28ms. The retrograde curve showed a non-decremental pattern with an concentric atrial activation. Mapping with the ablation catheter inside the CS os revealed an accessory pathway (AP) potential followed by sudden but temporary increase in the local ventriculo-atrial (VA) time with catheter pressure during ventricular pacing. A CS-venogram showed normal CS anatomy. Following spontaneous recovery of retrograde AP conduction, 20W irrigated Radiofrequency (RF) ablation was delivered (45 seconds) which caused a persistent change in the retrograde pattern during ventricular pacing within 3 seconds of RF onset. Antegrade pre-excitation remained.

During subsequent antegrade EPS, narrow complex orthodromic atrio-ventricular reentrant tachycardia (AVRT) was induced (cycle length 330ms; mechanism confirmed with standard

testing), with earliest atrial activation in the His "A" although at this location the VA time was long (Figure 1). Mapping of the tricuspid annulus with the ablation catheter showed later atrial activation timing again (Figure 1). What are the possible explanations and what are the possible locations of the AP? What should be the next step for mapping this tachycardia?

Discussion

Mapping of this AP in the retrograde direction was initially confusing. No early atrial activation could be observed in the coronary sinus, His position or on the right atrial free wall. This could indicate that this is a slowly conducting AP or that there are multiple pathways.

The HA interval increased to the extent of increase of the HV interval (HV predicted HA) as seen in figure 1 during tachycardia with bundle branch block confirming the diagnosis of AVRT. However, the earliest A during orthodromic AVRT was recorded in the His. Furthermore, in this case another clue was that prior ablations had targeted a left AP, double potential seen at CS1,2 is actually indicating CS1,2 straddling in the line of block or conduction delay . When the CS catheter was advanced to a distal location (Figure 2A) a region of conduction block with discrete double potentials was found indicating a region of prior atrial ablation (Figure 2B, asterix in CS bipoles 7,8 and 9,10). Distal to this location atrial activation was early and "bracketed" at CS 5,6 at approximately 3:00 on the mitral clockface (Figure 2b). Ablation via a transeptal approach to this location eliminated both the pre-excitation and the retrograde eccentric sequence. Tachycardia was no longer inducible. A careful scrutiny of the atrial activation during AVRT (Figure 1) suggests that instead of complete block which would cause proximal to distal CS activation (when the entire CS catheter is medial to the block), incomplete mitral isthmus block (or delay) causes slow clockwise CS activation to collide with faster counterclockwise CS activation at CS 5-6.

The phenomenon of intra-atrial conduction block during left accessory pathway ablation is reported in ~7% of cases, and has been hypothesized as due to creation of conduction block in the mitral isthmus between the mitral annulus and the left inferior pulmonary vein.[1] It is improbable that block is complete along the length of this line but local block can markedly change the activation pattern on the CS catheter. With the CS catheter in a traditional posterior location the activation pattern may no longer suggest a left lateral pathway. Awareness of this possibility is important when prior unsuccessful ablation of a left sided pathway has been performed. Multiple accessory pathways too can result in confusing findings during the EP study and may result in a lesser success rate of an initial ablation (2).

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Figure Legend

Figure 1. Intracardiac recording during the electrophysiology study is shown at 200mm/sec recording speed. The first two beats are antegrade curve pacing from CS-5,6 bipole followed by beats during tachycardia. During tachycardia earliest A was seen in His channel, and ablation

catheter that was positioned in tricuspid annulus also showed late A signal. HV and HA intervals differences during tachycardia with bundle branch block narrow complex tachycardia also noted; a double potential at CS1,2 bipole was also noted.

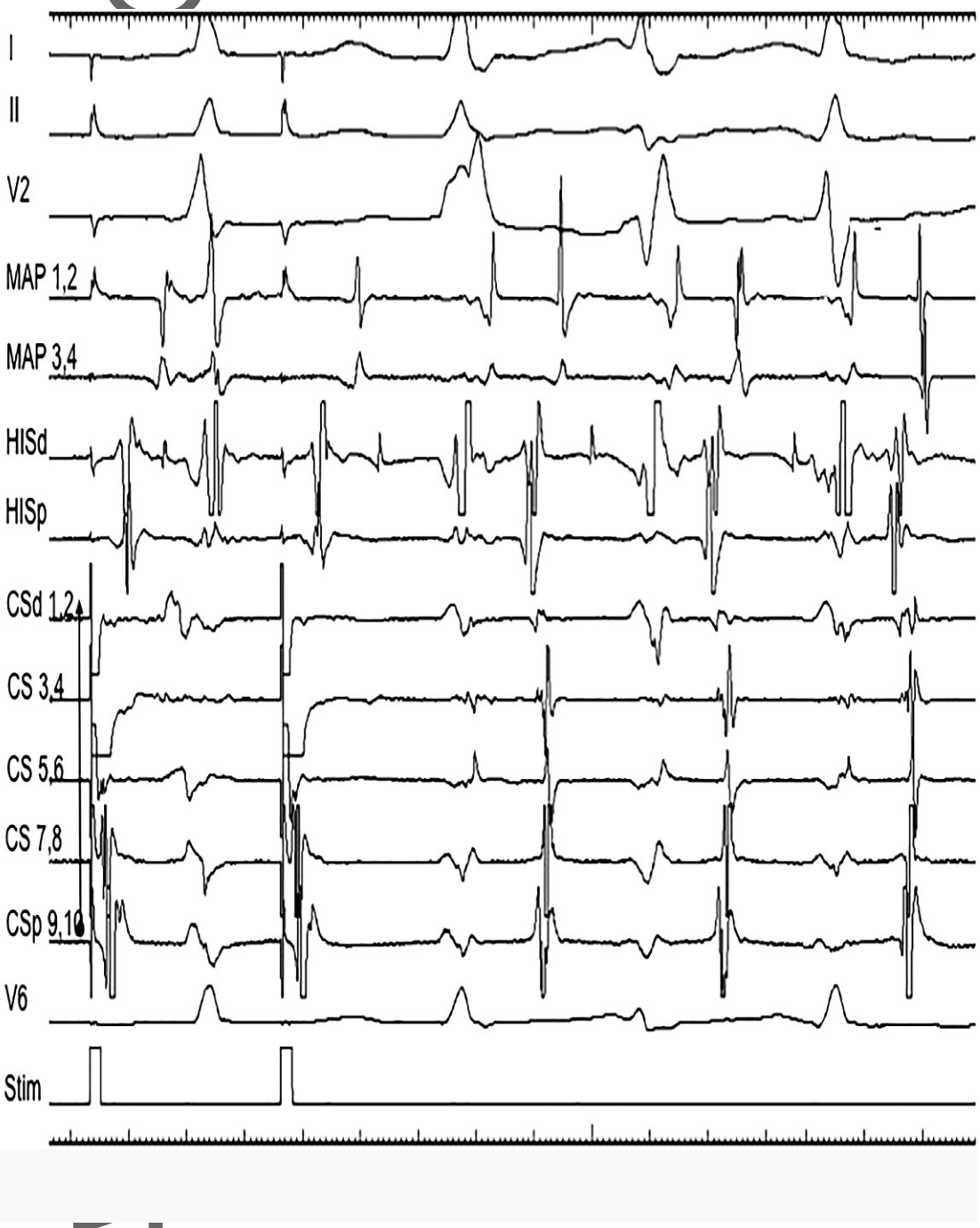

Figure 2. A. Fluoroscopy of catheter position in LAO 20 degree view after coronary sinus catheter advancement. B. Intracardiac recording during the electrophysiology study after CS catheter advancement is shown at 200mm/sec recording speed. The beats during tachycardia

showed early and “bracketed” atrial activation in CS-5,6 bipole (short arrow), moreover discrete double potentials (asterix) marked regions of conduction block as seen in CS7,8 and CS9,10.