

# Unchanged QRS morphology with atrial pacing in a case of WPW syndrome. What is the mechanism?

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A 55 year-old woman presented to our clinic for short episodes of palpitation without documented arrhythmia. Her family history was unremarkable except for an uncle implanted with a pacemaker for atrioventricular (AV) block. Her surface electrocardiogram showed sinus rhythm with wide QRS morphology and very short PR interval (Fig 1A) consistent with WPW syndrome. Echocardiography was normal. After informed consent she underwent an electrophysiological study. At baseline the His to ventricular (HV) interval was 41 ms (Fig 1B). There was no retrograde conduction. During incremental atrial pacing (Fig 1C) and extrastimulation the atrium to His (AH) interval progressively lengthened, **the stimulus to QRS stayed constant**, while the HV shortened, without any change in the morphology of the QRS, until the atrial extrastimulus failed to conduct to the ventricles (Fig 1D). What can be the explanation for the constant QRS morphology, despite shortening HV during atrial pacing?

#### Discussion

The HV interval was unusually long for ventricular pre-excitation, but progressively shortened with atrial pacing, suggesting an AV accessory pathway (AP). In case of an AV AP antegrade ventricular activation usually results from a fusion of conduction through the AV node and the AP. During atrial pacing progressive slowing of conduction in the AV node results in increasing pre-excitation and changing of QRS morphology as progressively larger part of the ventricles are activated through the AP. The lack of fusion during atrial pacing in case of an AV AP may be related to ventricular activation present only through the AP and lacking through the AV node (1).

Due to assumed high risk features of the AP (effective refractory period **(ERP) < 240 ms on isoproterenol infusion**) a decision was made to ablate it. The earliest ventricular activation was found on the right posteroseptal region close to the coronary sinus ostium, where no His potential was recorded. During the first radiofrequency application, after 3 seconds complete AV block occurred without any junctional rhythm (**Fig 2A**). Energy delivery was stopped immediately and an infra Hisian AV block was revealed but after 5 minutes AP conduction recovered. At this point preexisting infra-His AV block in addition to the presence of the AV AP was realized. After discussion with the patient about the unpredictable conduc-

tion properties of the AP in the long term, a DDD pacemaker was implanted. The procedure was repeated after 3 months. After ablation of the AP, 1:1 AV conduction with left bundle branch block and a long HV interval of 60 msec was observed (Fig 2B). We hypothesize that during the first procedure complete AV block was related to preexisting left bundle branch block plus mechanical right bundle block due to catheter trauma in the His position.

In hindsight, several findings pointed to the absence or significant prolongation of antero-grade conduction over the normal AV conduction system even before the first ablation attempt. The QRS was maximally preexcited even in sinus rhythm and did not change with atrial pacing. The HV interval (a pseudointerval in this case) had no relation to AV conduction: it was long at baseline, then progressively shortened with atrial pacing, but the stimulus to QRS remained constant, with constant QRS morphology verifying absence of the contribution of the normal AV conduction system to the QRS. Finally, there was no normal QRS after the His potential when the AP blocked (Fig. 1. D).

Whether the ablation of this AP and the implantation of the pacemaker was necessary can be debated. Guidelines state that ablation is reasonable of the AP capable of rapid conduction (AP ERP < 240-250 ms), but observation is also a reasonable alternative in the asymptomatic adult (2). Whether this patient's symptoms were related to the AP remains uncertain in view of the lack of retrograde conduction and inducible arrhythmia. The conduction capacity of an AP is thought to decrease with ageing (3) posing potential risk of AV block to a patient with impaired normal conduction system. These dilemmas were discussed thoroughly with the patient before proceeding with ablation and implantation.

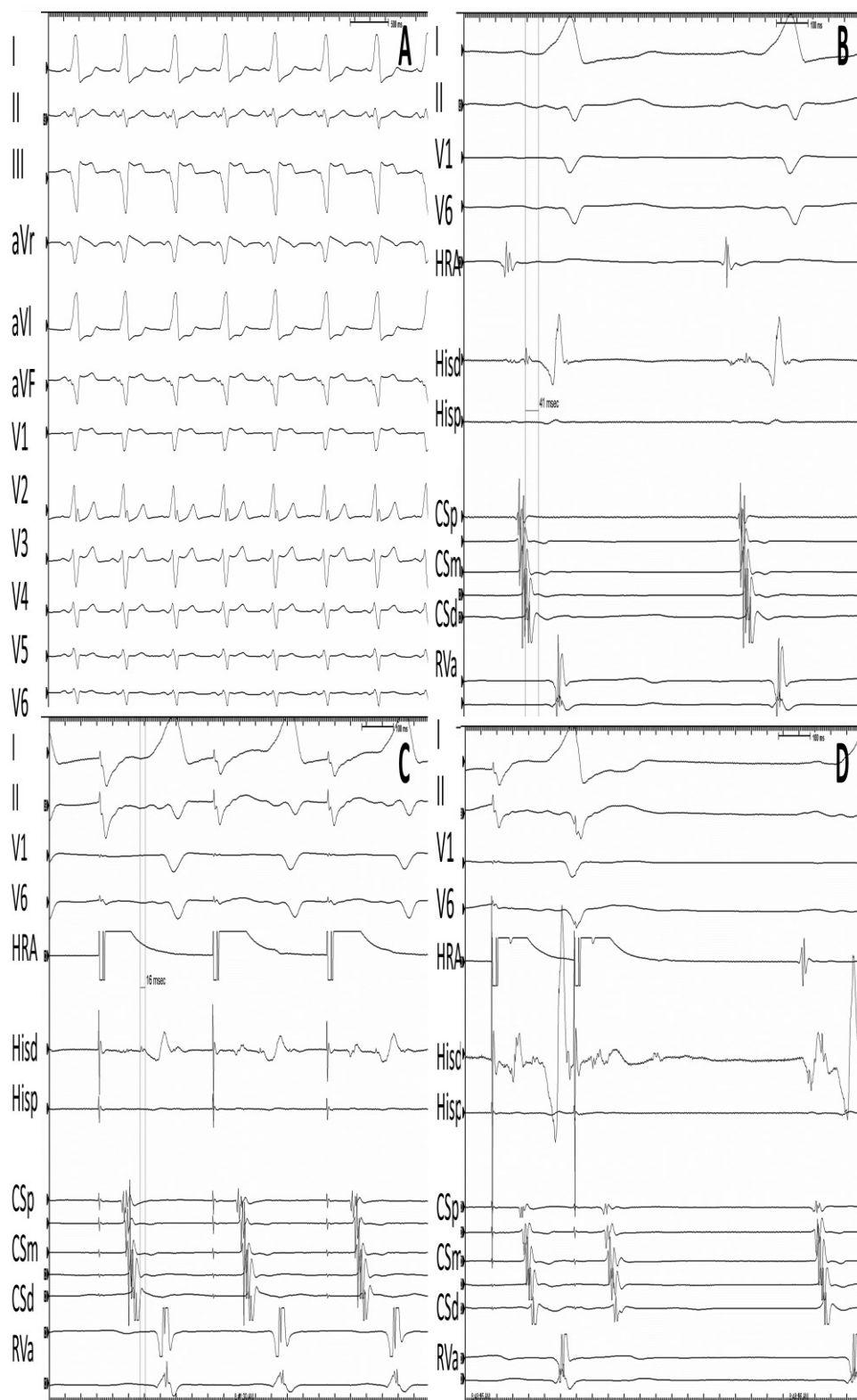
This case reminds us that when no change is observed in the amount of ventricular fusion during atrial pacing with an AV AP, occult AV block should be suspected.

## References

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## Figures Legend

**Figure 1.** Twelve-lead ECG during sinus rhythm (Panel A) Paper speed 50 mm/s. Surface electrocardiogram lead I, II, V<sub>1</sub>, V<sub>6</sub> along with intracardiac recordings from high right atrium (HRA), coronary sinus (CS) and mapping catheter placed on the His region (His) during sinus rhythm (Panel B), during atrial incremental pacing (Panel C) and during atrial extrastimulation (Panel D). Paper speed 200 mm/s.



**Figure 2.** Surface electrocardiogram lead I, II, V<sub>1</sub>, V<sub>6</sub> along with intracardiac recordings from high right atrium (HRA), coronary sinus (CS), right ventricular apex (RV) and ablation catheter (Abl) during

ablation in the first procedure (Panel A). Paper speed 50 mm/s, and during the successful ablation in the second procedure (Panel B). Paper speed 200 mm/s.

