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Masquerading Tachycardia

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Case Presentation

Mrs. BW is a 69 year old previously well woman with history of palpitations. Extensive workup showed no evidence of structural heart disease. Her baseline ECG was unremarkable. She was clinically documented to have narrow complex tachycardia. In tachycardia her ECG showed brief bursts of ectopic atrial activity with "saw-tooth" appearance in the inferior leads (Figure 1, Panel A) alternating with lesser amplitude p-waves positive in the inferior leads and in V1 (Figure 1, Panel B). During electrophysiology study a quadripolar catheter was placed at the right ventricular apex, a decapolar catheter in the coronary sinus, a duodecapolar catheter around the tricuspid annulus and a quadripolar catheter at the His bundle position. The study documented "atypical flutter/fibrillation" (Figure 2). No ablation was carried out at that time. She did not tolerate Sotalol secondary to fatigue. Subsequently she was treated with amiodarone but developed visual side effects. She then presented for a second electrophysiology study once 3D electroanatomical imaging became available at our center. The patient was instrumented with a decapolar coronary sinus catheter, quadripolar high right atrial catheter and a Navistar 4 mm tip ablation catheter. She had multiple spontaneous runs of tachycardia with varying activation sequence and at least 20 msec variation in the cycle length (Figure 3). It was felt that she may in fact have an ectopic atrial tachycardia occasionally entraining the right atrial flutter circuit. The cavotricuspid isthmus was ablated using 50W/60C delivered from a Stockert RF generator with bidirectional block confirmed by extension of conduction across the isthmus from 60 to 150msec. Following ablation, tachycardia organized and had the distinct left-to-right activation sequence judging by the available intracardiac electrograms (Figure 4, Panel A). At this point trans-septal access to the left atrium was accomplished, the patient was systemically anticoagulated and a 3D electroanatomical map of the left atrium was constructed. This clearly demonstrated a focal tachycardia originating at the ridge between the left upper pulmonary vein and the left atrial appendage (Figure 4, Panel B). Several radiofrequency lesions were delivered here with initial prolongation in the tachycardia cycle length and eventual termination and non-inducibility of the tachycardia. Pulmonary vein isolation was not targeted in this patient. She remains symptom free one and a half years following the ablation.

Discussion

There is accumulating evidence suggesting common etiology between atrial flutter and fibrillation, both initiated by ectopic atrial activity arising from the pulmonary veins.[1] This case illustrates interplay between a focal atrial tachycardia originating in the left upper pulmonary vein engaging the right atrial flutter circuit. This arrhythmia was difficult to map conventionally because of varying cycle length and activation sequence. Some may

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have offered pulmonary vein isolation to such a patient classifying their arrhythmia as atrial fibrillation. Elimination of the flutter circuit lead to successful unmasking, identification and ablation of the source of tachycardia without full pulmonary vein isolation with long term freedom from symptomatic arrhythmia. Retrospectively, elimination of the source of atrial ectopy may have sufficed, but this would have been difficult to map and in the presence of ongoing arrhythmia even if only in the form of the typical flutter, this patient would likely have ended up with a full pulmonary vein isolation procedure.

Figure 1: Clinical Arrhythmia Panel A. Morphology #1

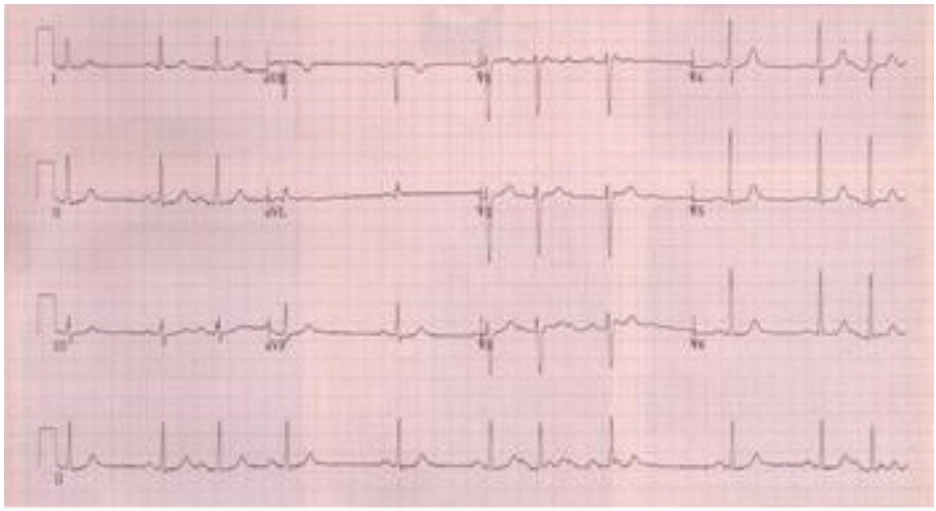
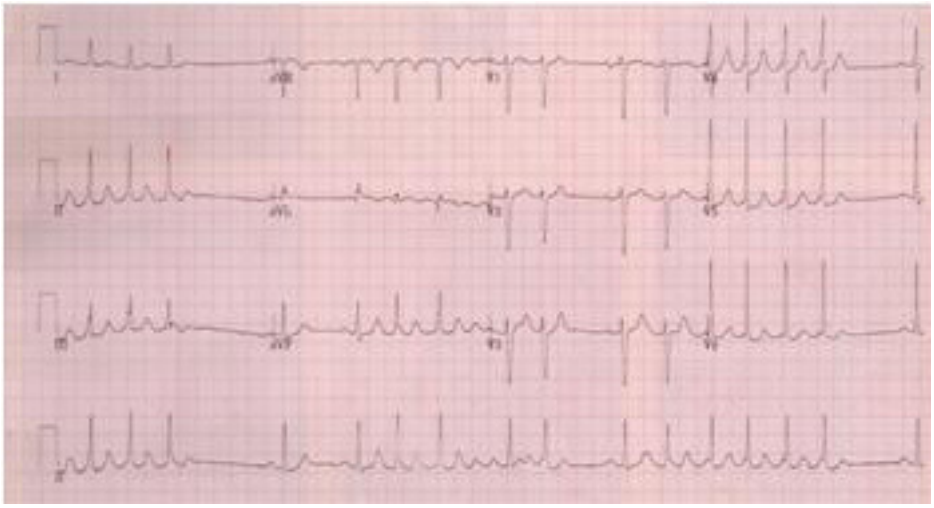


Figure 1: Panel B. Morphology #2



References

1. Wazni, O., et al., Randomized study comparing combined pulmonary vein-left atrial junction disconnection and cavotricuspid isthmus ablation versus pulmonary vein-left atrial junction disconnection alone in patients presenting with typical atrial flutter and atrial fibrillation. Circulation, 2003. 108(20): p. 2479-83.

Figure 2: 2. Electrophysiology Study #1 Panel A. Tachycardia Initiation

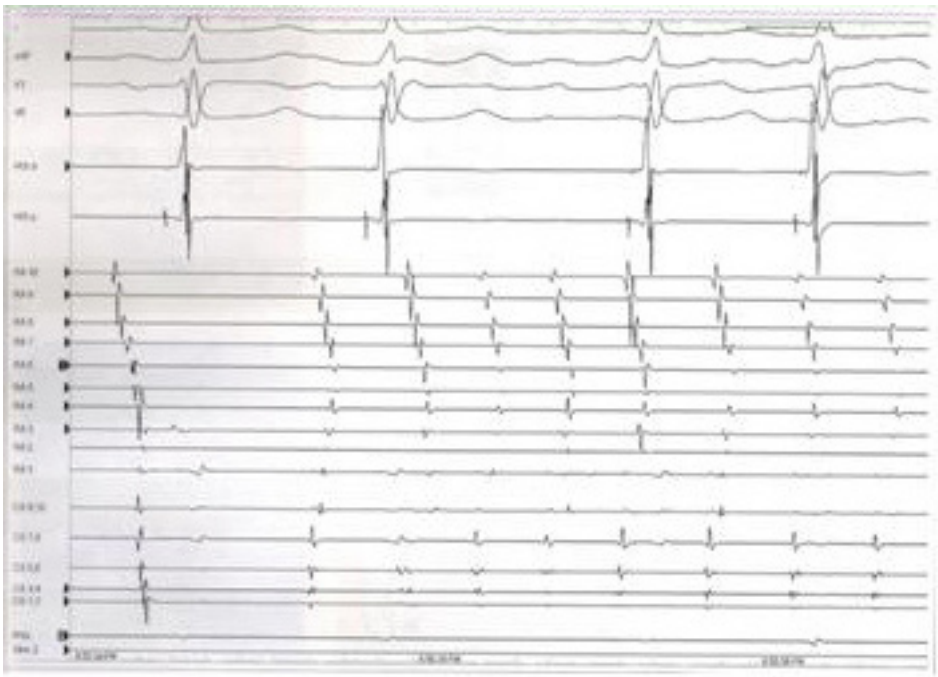


Figure 2: 2. Panel B. Tachycardia Propagation.

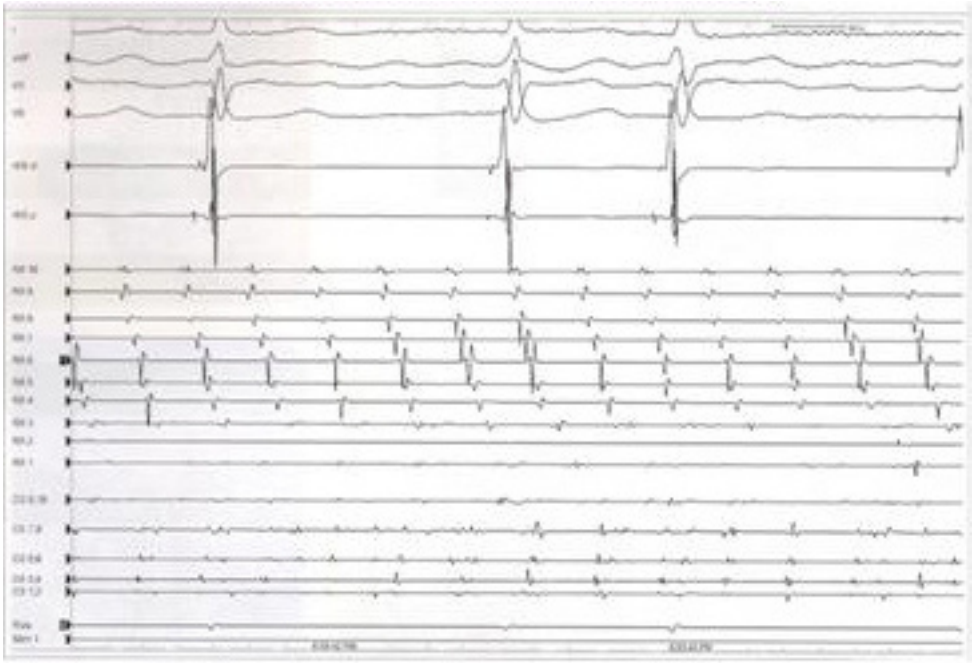


Figure 3: Second Electrophysiology Study Panel A. First activation sequence, CL 262 msec

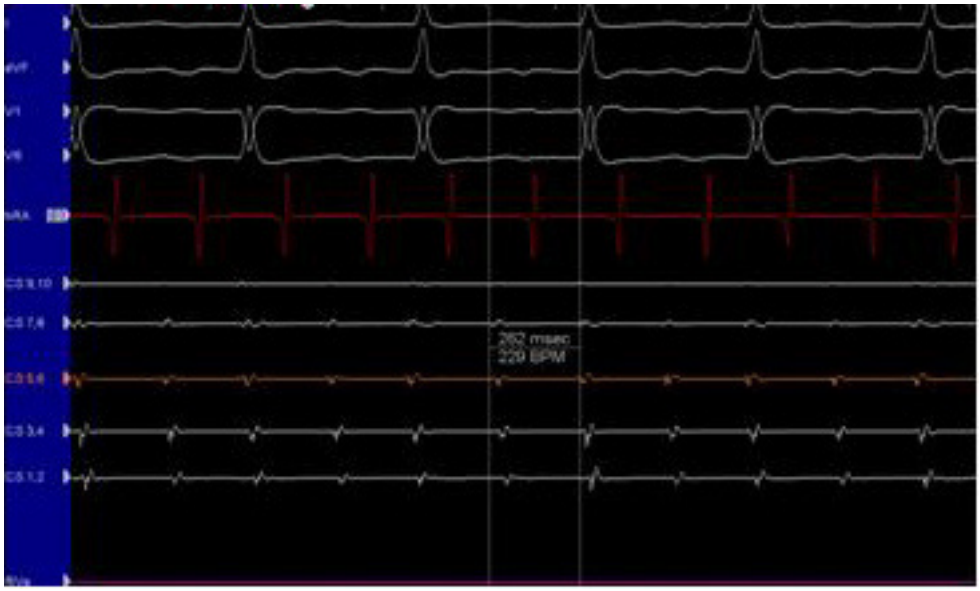


Figure 3: Transition to a second activation sequence, CL 243 msec

Figure 4: Tachycardia following cavo tricuspid isthmus ablation.