

Atrial fibrillation following slow pathway modification?

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Abstract: We are exposing a differential diagnostic problem in the case report of a young female with the diagnosis of atrial fibrillation who was referred to our Institute for pulmonary vein isolation. Based on the 12-lead ECG we found an unusual mechanism resulting in irregular, narrow QRS tachycardia, which was also confirmed by an electrophysiological study.

Keywords: atrial fibrillation, AV nodal physiology, double ventricular response

We are presenting the case report of a 25-year-old female who was admitted to our Institute for pulmonary vein isolation. Her history is remarkable for slow pathway modification due to typical slow-fast AV nodal re-entrant tachycardia in 2003. Following a short symptom-free period her palpitations recurred and the electrophysiological study was repeated in 2004 and again in 2008, both with negative result: neither arrhythmia was inducible nor dual AV nodal physiology was observed. One year later her symptoms worsened with a daily occurrence of her palpitations. 12-lead ECG showed an irregular narrow QRS tachycardia (*Fig. 1*) and the diagnosis of atrial fibrillation was established. *Per os* propafenone and sotalol proved to be ineffective preventing tachycardia episodes and the patient was referred to our Institute for pulmonary vein isolation. Echocardiography showed a structurally normal heart without clinically significant abnormalities and thyroid hormones were in normal range.

Although this irregular narrow QRS tachycardia appears to be atrial fibrillation at first glance, the presence of p waves can be appreciated with the careful examina-

tion of the tracing (*Fig. 2*) excluding the diagnosis of atrial fibrillation. Axis, morphology and regularity of p waves confirms that the rhythm is sinus with the atrial rate in the normal range. However, the atrioventricular conduction is not 1:1 as some of the p waves are followed by two QRS complexes resulting in a faster ventricular rate. A ladder diagram demonstrates the exact mechanism (*Fig. 3*): a sinus beat is conducting down the ventricles via the fast and the slow pathway as well, resulting in two QRS complexes. This phenomenon is called “double ventricular response” or “dual antegrade response” and the consequential arrhythmia is an incessant non-reentrant supraventricular tachycardia [1, 2]. Some of the p waves (1, 5, 6) are conducting through either only the fast or the slow pathway as a consequence of retrograde invasion of the previous activation from the another pathway. During an electrophysiological study this diagnosis was confirmed demonstrating one atrial EGM followed by two His and two ventricular electrograms (*Fig. 4*). There was no retrograde conduction which explains the lack and non-inducibility of re-entrant AV nodal tachycardia.

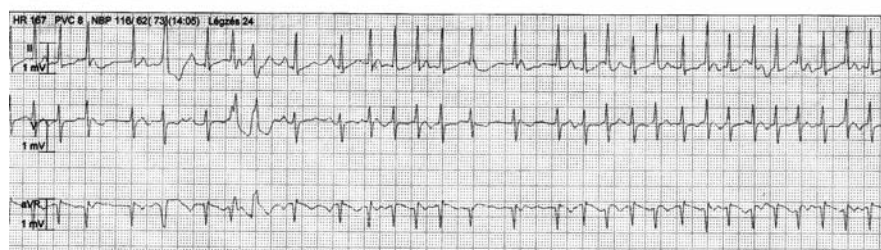


Fig. 1. ■ Surface ECG of the clinical arrhythmia

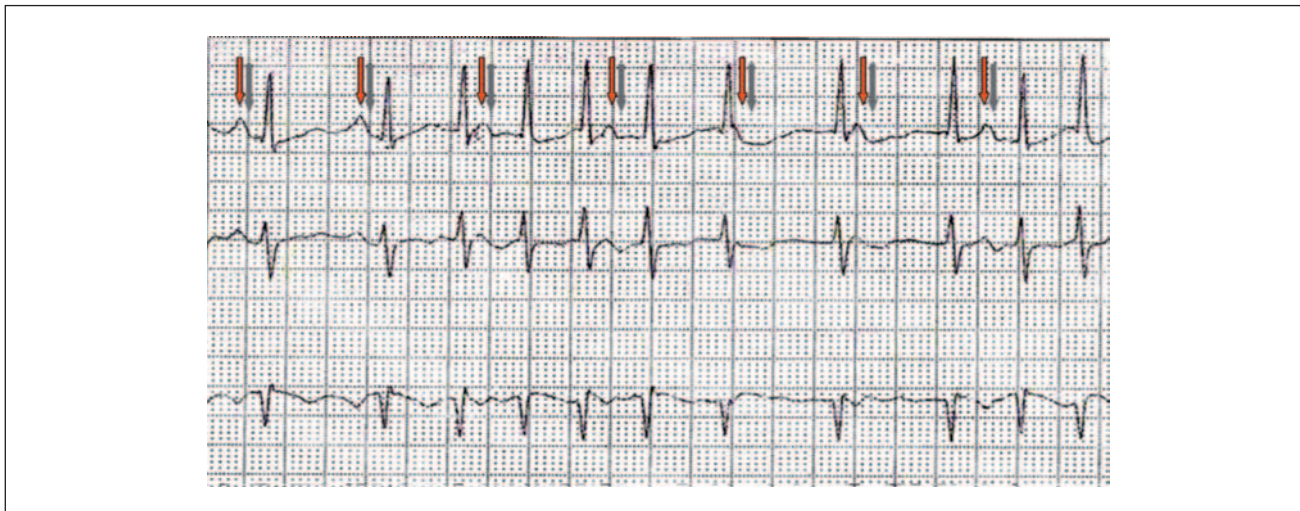


Fig. 2. ■ Surface ECG of the arrhythmia. P waves are marked with red arrows

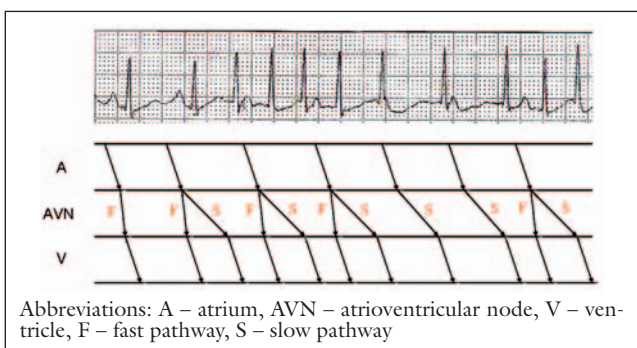


Fig. 3. ■ Ladder diagram of “double ventricular response”

Double ventricular response was promptly terminated by radiofrequency ablation of the slow pathway.

Double ventricular response is a rare phenomenon often presenting as a differential diagnostic challenge [2].

Features suggesting a misdiagnosis of atrial fibrillation include the clinical presentation and symptoms and the ECG with irregular narrow QRS tachycardia. Furthermore, it is usually refracter to antiarrhythmic medication. Electrophysiology study and catheter ablation offer the correct diagnosis and successful long-term therapy in these patients.

References

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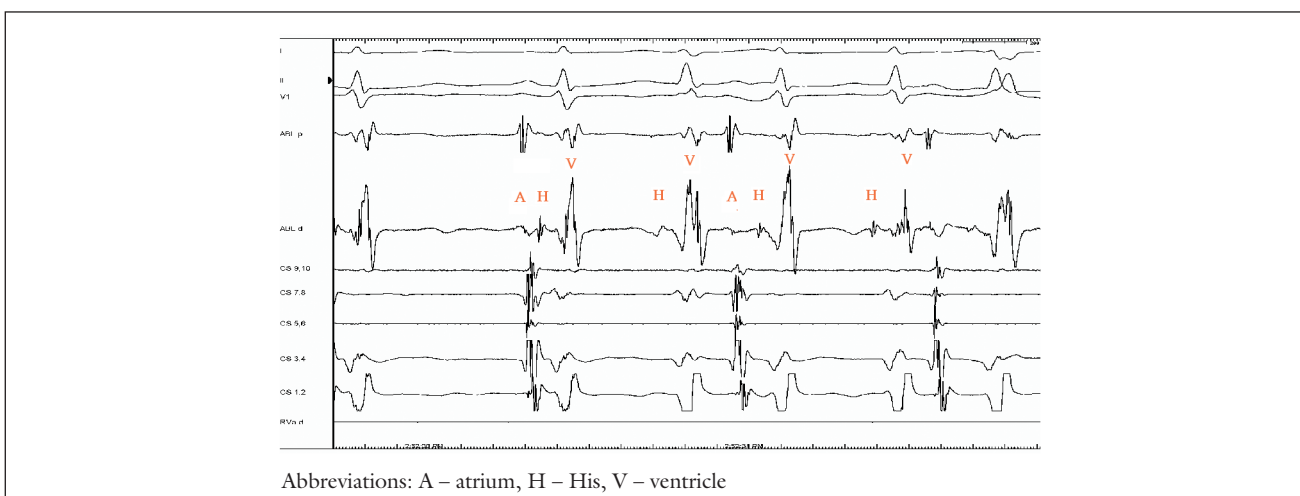


Fig. 4. ■ Intracardiac electrograms during “double ventricular response”