

A rare mechanism of tachycardia and aberrancy



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Introduction

A dual atrioventricular (AV) nodal response is a rare mechanism underlying arrhythmias and can be difficult to diagnose. In the present case report we describe a stepwise diagnostic approach that leads to the diagnosis of a dual AV nodal response.

Case report

A 40-year-old man without any past medical history was referred to our hospital because of episodes of rapid palpitations associated with dizziness existing since 6 months. Treatment with atenolol and verapamil, respectively, was not successful in preventing the patient's symptoms. Physical examination did not reveal any abnormalities except for an irregular heart rate of 105 beats per minute (bpm). A 24-hour Holter recording showed many episodes of narrow complex tachycardia with a maximum heart rate of 240 bpm. Twelve-lead electrocardiography was performed at rest (Figure 1A) and during exercise (Figure 1B). Transthoracic echocardiography and cardiac magnetic resonance imaging showed a dilated left ventricle with global hypokinesia resulting in an ejection fraction of 42%. No delayed enhancement was observed.

The electrocardiogram (ECG) at rest shows a sinus rhythm of 60 bpm with normal PR and QRS width. Furthermore, QRS complexes with a different morphology (left anterior fascicular block sometimes in combination with complete or incomplete right bundle branch block [RBBB] morphology) persistently occur in bigeminy with sinus rhythm, resulting in a heart rate of 120 bpm at rest. The continuous presence of this group beating and relatively high heart rate at rest may have been responsible for the reduced left ventricular ejection fraction measured by echocardiography and cardiac magnetic resonance imaging.

KEYWORDS Aberrancy; Dual AV nodal response; Equalization of conduction delay; Slow pathway ablation; Supraventricular tachycardia; Tachycardiomyopathy

(Heart Rhythm Case Reports 2018;4:447–450)

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KEY TEACHING POINTS

- A dual atrioventricular (AV) nodal response is a rare cause of arrhythmias but can be diagnosed by a detailed evaluation of the electrocardiogram at rest and during exercise.
- A dual AV nodal response can cause cardiomyopathy, which can be totally reversible after treatment of the arrhythmia.
- Ablation of the slow pathway is an excellent way to treat the arrhythmia.

Discussion

Resting electrocardiogram

When assessing the ECG at rest only, the differential diagnosis of the arrhythmia that results in this group beating is diverse. At first sight the second and broader QRS complex of every group may theoretically represent a premature ventricular complex, a premature atrial complex, ectopy from the AV node or His-Purkinje system, or a ventricular echo beat (owing to reentry in the AV node or via an accessory pathway) or may be the result of a dual AV nodal response.

Electrocardiogram during exercise

The ECG during exercise shows a sinus tachycardia of 110 bpm, signified by positive P waves in leads II, III, and aVF with negative P waves in aVR (superimposed on the T wave), which can be recognized throughout the ECG with similar P-P intervals (560 ± 10 ms). However, on ventricular level the heart rate starts at 110 bpm, but suddenly doubles to a frequency of 220 bpm. When one evaluates this tachycardia in more detail it can be appreciated that there is also a group beating pattern (as was observed at rest) and, although less pronounced, alternating QRS morphology (most clearly seen in V1). The QRS complexes are narrow, which makes a ventricular origin unlikely. Before the onset of the tachycardia the R-R intervals and P-P intervals are similar (560 ± 10 ms), with a PR interval of 520 ± 10 ms. During the tachycardia every P wave

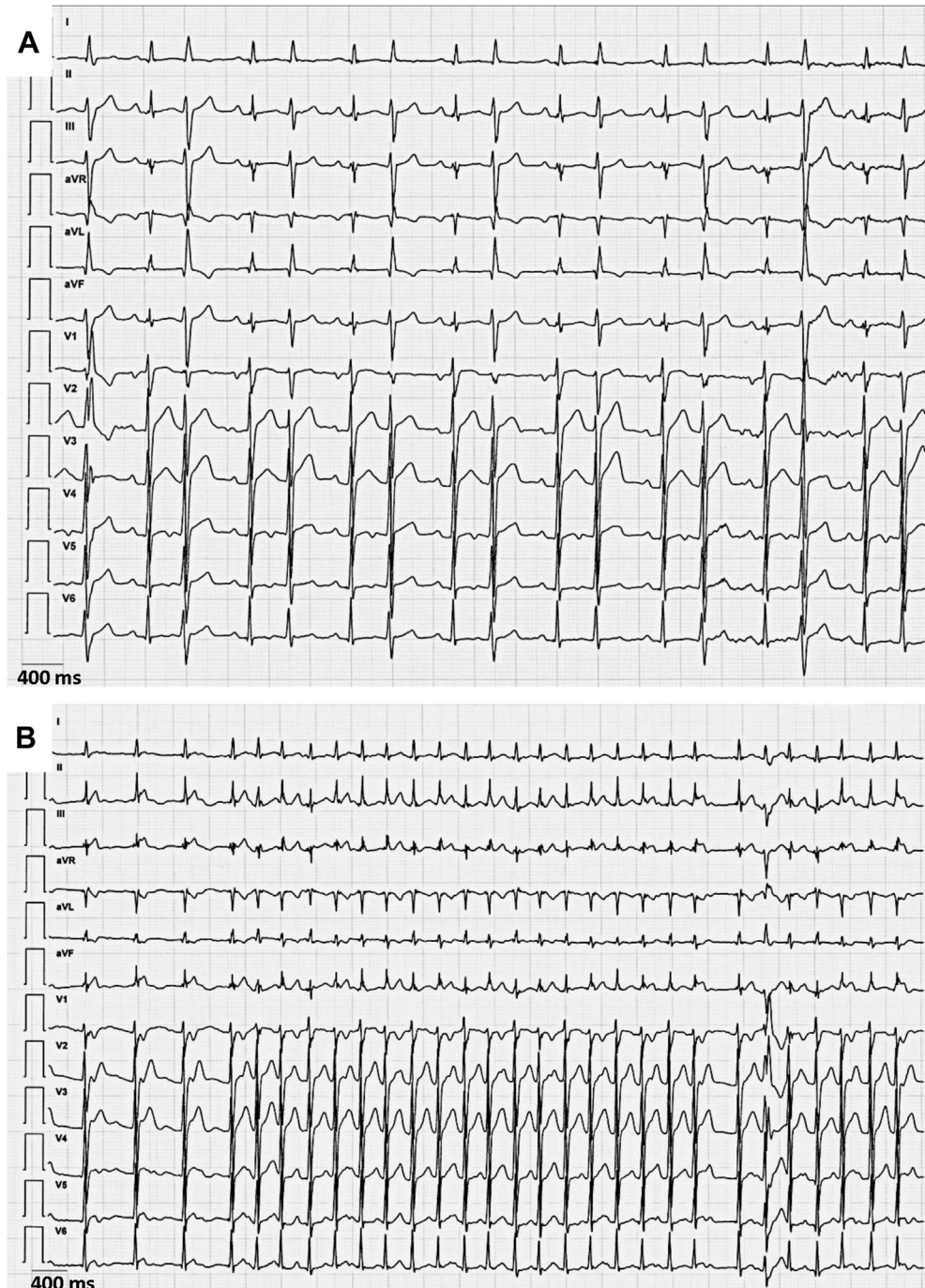


Figure 1 Twelve-lead electrocardiogram. **A:** at rest; **B:** during exercise.

is followed by 2 QRS complexes with a P to the second R interval (PR2) of 520 ± 10 ms. The fact that the PR interval before the onset of the tachycardia is the same as the PR2 interval during the tachycardia makes ectopy from the atria, AV node, or His-Purkinje system very unlikely. This phenomenon was observed at several stages during the exercise test, every time with a different sinus node frequency per stage, which indicates the presence of AV association. Because all P waves have an inferior heart axis and the P-P and R-R intervals are the

same before the onset of the tachycardia, a junctional rhythm (with retrograde atrial activation) is absent. This combination of findings also rules out reentry using an accessory pathway and make reentry in the AV node (with block to the atrium) very unlikely.

The best explanation for the combination of findings is a dual AV nodal response. During group beating every P wave is followed by 2 QRS complexes as a result of simultaneous conduction through the fast and slow AV nodal

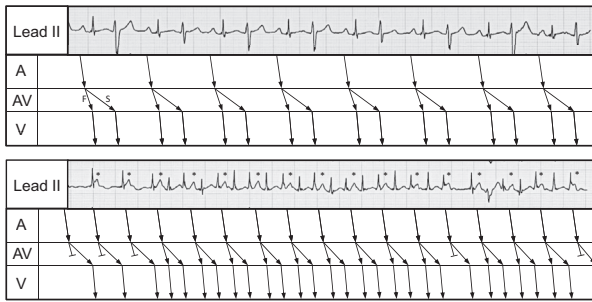


Figure 2 Ladder diagram at rest (upper panel) and during exercise (lower panel). A = atrium; AV = atrioventricular node; F = fast pathway; S = slow pathway; V = ventricle. * indicates P wave during exercise.

pathway, which both proceed through the bundle of His owing to sufficient dissociation of conduction velocity in the fast and slow pathway overcoming the refractory period of the bundle of His and ventricles.^{1,2} Figure 2 shows the ladder diagram of the proposed mechanism of dual AV nodal conduction at rest and during exercise.

Invasive electrophysiological study

An invasive electrophysiological study was performed to confirm the diagnosis and to treat the arrhythmia. Figure 3 illustrates the position of catheters (by radiograph and NavX; Abbott, Hoofddorp, The Netherlands) and electrograms at baseline. A hexapolar catheter was placed in the right ventricular apex (RVA), and an octopolar catheter was placed in the coronary sinus (CS 1-8). A 20-polar catheter was placed along the AV node, bundle of His, and right bundle branch (RBd-20). A quadripolar mapping catheter was positioned at the proximal bundle of His (HBE, HBE uni = unipolar electrogram from distal mapping electrode). The blue dots in the NavX indicate the location of the bundle of His. The His bundle electrogram shows an atrial deflection (A) subsequently followed by a His potential (H1), ventricular deflection (V1), His potential (H2), and ventricular deflection (V2). So, no atrial deflection is recorded before the second His potential (H2), ruling out atrial ectopy or reentry via an accessory pathway. The sharp, initially negative deflection in the unipolar recording at both the first and second His potential

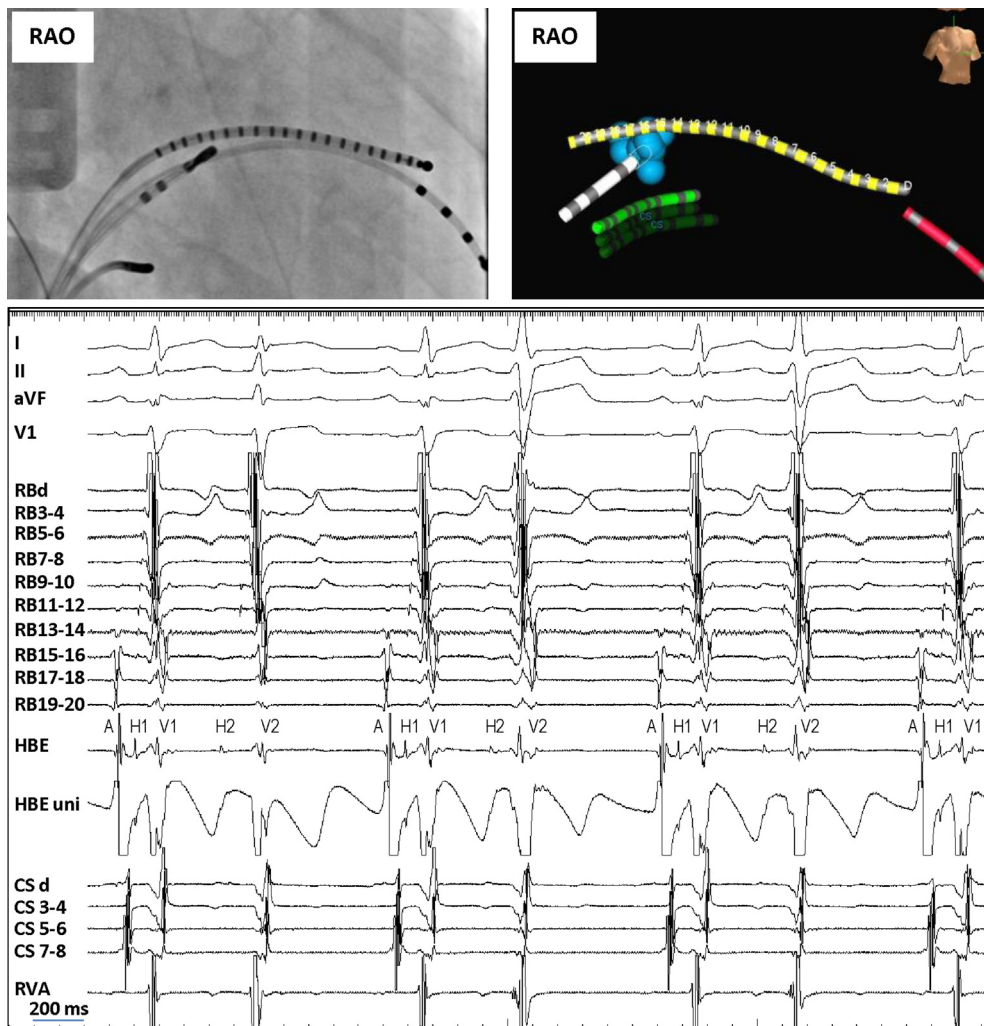


Figure 3 Position of catheters (radiograph and NavX) in relation to the bundle of His (blue dots). Surface electrocardiogram leads and endocardial electrograms of right bundle (RBd-RB11-12), His bundle (HBE), His bundle unipolar (HBE uni), coronary sinus (CS), and right ventricular apex (RVA). See text for explanation. RAO = right anterior oblique.

indicate that the distal electrode of the catheter was positioned at the transition of the AV node and proximal His bundle. Every V is preceded by an H and the H2-V2 interval is longer than the H1-V1 interval, which rules out ventricular ectopy. The fact that the H2-V2 interval is longer than the H1-V1 interval furthermore underlines that ectopy from the His-Purkinje system is very unlikely, in which case one would expect H2-V2 to be shorter than H1-V1. Although a ventricular echo beat, as a result of reentry in the AV node with block to the atrium, cannot be ruled out completely, this is very unlikely. In conclusion, the surface ECGs at rest and during exercise, in combination with the intracardiac recordings, best fit the diagnosis of a dual AV nodal response after a single atrial activation.

Mechanism of dual AV nodal response

During a dual AV nodal response atrial depolarization is followed by antegrade conduction through the fast and slow pathway simultaneously. However, the difference between the conduction velocity of the fast and slow pathway must be large enough to allow conduction through the slow pathway to proceed through the bundle of His (overcoming the refractory period of the bundle of His). In other words, the slow pathway conduction time must be longer than the conduction time of the fast pathway plus the refractory period of the bundle of His. Furthermore, retrograde invasion of the slow pathway after antegrade conduction of the fast pathway must be absent. In addition, when atrial depolarization occurs too early (H2-A too short), fast pathway conduction arrives at the bundle of His when it is still refractory, resulting in only proceeding of slow pathway conduction through the bundle of His, which was also observed in our patient (Figures 1B and 2).^{1,2}

Proposed mechanism of aberrancy

As mentioned above, during group beating every second QRS complex has a different morphology as compared to the first QRS complex. Since a dual AV nodal response is the likely diagnosis, this must be the result of various degrees of aberrancy. In Figure 3, 3 aberrant QRS complexes are shown, each with a different pattern of aberrancy. The first shows almost no aberrancy, the second shows a left anterior fascicular block with incomplete RBBB morphology. The third only shows a left anterior fascicular block. Between these forms of aberrancy, the interval between the

His potential and the right bundle branch potentials (in RBd-RB11-12) remains unchanged, but is longer than H-RB intervals during the first QRS complexes of every group (Supplemental Figure). Furthermore, when the QRS duration during aberrancy is longer, the H-V interval becomes shorter. Taking these findings together, the QRS complexes that have almost no aberrancy and are relatively narrow must be the result of the phenomenon of equalization of conduction delay between the left fascicles (posterior and anterior) and the right bundle branch. However, the fact that the QRS complex shows complete RBBB when it is preceded by a long R-R interval, as can be observed in Figure 1B (sixth QRS complex from the right), is an Ashman phenomenon due to prolongation of the refractory period of the right bundle branch.

Ablation of slow pathway

Since the diagnosis of a dual AV nodal response was very likely, we performed radiofrequency catheter ablation of the slow pathway, immediately resulting in the total absence of the arrhythmia. The patient became free of any complaints and 3 months after ablation there was a complete recovery of the left ventricular ejection fraction (67%) as measured by echocardiography.

Conclusion

A dual AV nodal response is a rare mechanism of tachycardias, but can be suspected from the electrocardiogram at rest and during exercise. This tachycardia may result in a tachycardiomyopathy, and slow pathway ablation is an excellent way to treat the tachycardia and can lead to a complete recovery of cardiac function.

Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hrcr.2018.06.011>.

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