

Two unusual cases of coincident atrioventricular nodal reentrant tachycardia and ventricular tachycardia

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The simultaneous occurrence of narrow and wide QRS complex tachycardias was observed in 2 patients evaluated at our electrophysiological centers. Electrophysiological testing revealed the coexistence of two types of arrhythmia (atrioventricular nodal reentrant tachycardia and verapamil-sensitive left ventricular tachycardia) in one patient and of three types of arrhythmia (atrioventricular nodal reentrant tachycardia, ventricular tachycardia originating from the right ventricular outflow tract, and left ventricular tachycardia) in the other. Both patients underwent successful radiofrequency ablation of all the types of tachycardia.

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Introduction

Simultaneous tachycardias are rare arrhythmias which usually consist of atrial with ventricular tachycardia (VT) and, less commonly, atrial with junctional tachycardias. We report 2 cases of patients who presented with an unusual combination of narrow and wide QRS complex tachycardias and who underwent successful radiofrequency ablation in a single session.

Description of cases

Case 1. Clinical history. The patient was a 73-year-old male affected by arterial hypertension and coronary artery disease. In 2002, he had undergone coronary artery bypass grafting of the left anterior descending artery and of the first obtuse marginal branch for angina. No history of myocardial infarction. He was treated with diuretics, atenolol 50 mg/day, aspirin 100 mg/day, and pravastatin 40 mg/day. For about 10 years he had been suffering from undocumented, rare and short episodes of palpitations. The patient was referred to the emergency department in March 2003, where he presented with palpitations and dyspnea, which had started 1 week earlier. ECG showed a wide QRS complex tachycardia with a right bundle branch block morphology and left-axis deviation at a rate

of 130 b/min transforming into a narrow QRS complex tachycardia at a rate of 140 b/min (Fig. 1, upper panel). The narrow QRS tachycardia appeared to temporarily overdrive the wide QRS tachycardia, which was the predominant arrhythmia. Adenosine 20 mg failed to terminate the wide QRS tachycardia, but intravenous injection of lidocaine 40 mg restored sinus rhythm. Results of chest roentgenography and blood examination were normal. Transthoracic echocardiography was also normal. A treadmill exercise test was performed, but neither inducible arrhythmia nor ischemic changes were observed.

Electrophysiological study and radiofrequency ablation. Electrophysiological study was performed at the baseline, antiarrhythmic drug-free state. Baseline activation sequence and time were normal. Atrial pacing revealed no evidence of preexcitation or rate-related aberrant conduction, and dual atrioventricular nodal pathways were demonstrated. Ventriculo-atrial (VA) conduction was present; this showed decremental properties and retrograde activation sequence suggesting the absence of an accessory VA connection. Programmed and incremental atrial stimulation repeatedly induced a narrow QRS complex tachycardia at a cycle length of 330 ms. The mechanism of tachycardia was confirmed to be a slow-fast atrioventricular nodal reentrant

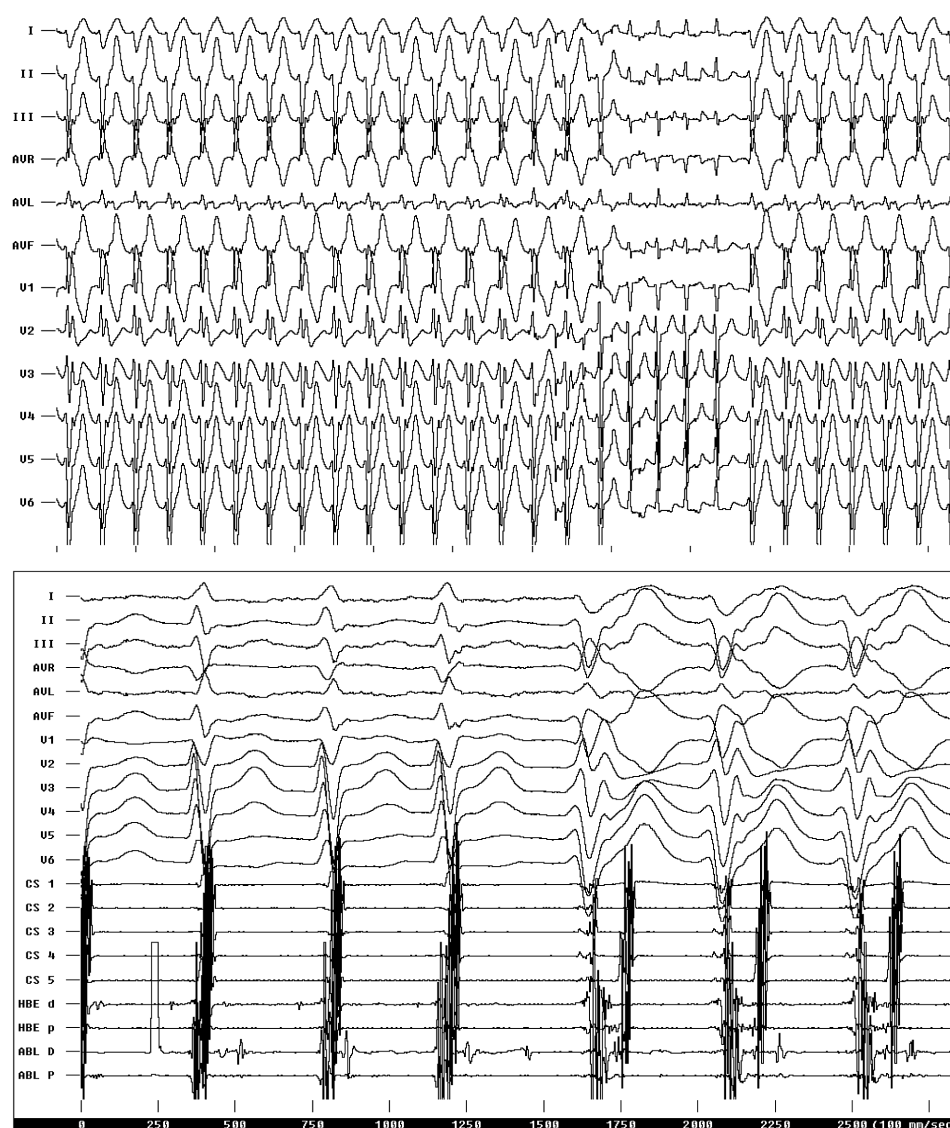


Figure 1. Case 1. Upper panel: 12-lead ECG obtained during an emergency room visit showing the wide QRS complex tachycardia with a right bundle branch block morphology and left-axis deviation at a rate of 130 b/min transforming into a narrow QRS complex tachycardia at a rate of 140 b/min. Lower panel: 12-lead ECG and intracardiac tracings (at a recording speed of 100 mm/s) during transition from a narrow to wide QRS complex tachycardia. Intracardiac ECG from coronary sinus (CS) distal to proximal regions (1 = distal; 5 = proximal); from His bundle distal and proximal (HBE d and HBE p) regions; from the ablation catheter (ABL) temporarily located at the right ventricular apex for stimulation.

tachycardia (AVNRT) on the basis of the classic criteria described by Josephson¹. On several occasions, the tachycardia spontaneously converted to a wide QRS complex tachycardia with a right bundle branch and left-axis deviation morphology, identical to the wide QRS tachycardia observed clinically (Fig. 1, lower panel). During this tachycardia a 1:1 VA relationship was present, with a VA interval of 110 ms. Ventricular pacing was repeatedly able to terminate and induce the wide QRS tachycardia, which was induced on one occasion also by atrial stimulation. Intravenous administration of 6 and 12 mg of adenosine did not affect the cycle length of the wide QRS tachycardia, but produced a temporary VA dissociation. On the other hand, intravenous verapamil 10 mg significantly slowed the wide complex tachycardia cycle length (from 400 to

480 ms) and blocked retrograde VA conduction. On the basis of the QRS morphology and the electrophysiological characteristics, the wide QRS tachycardia was defined as a verapamil-sensitive left VT². Radiofrequency ablation of the slow nodal pathway was performed along the postero-medial portion of the tricuspid annulus, and resulted in an accelerated junctional rhythm. Shortly after ablation, AVNRT was no longer inducible, whereas long runs of VT – unchanged in morphology, cycle length and response to verapamil – remained inducible. Pace-mapping from the inferior apical left interventricular septum reproduced the QRS morphology of the tachycardia. In addition, this same area exhibited the earliest endocardial activation times prior to the onset of the surface QRS during tachycardia, and a sharp high-frequency potential preceding the

onset of the local endocardial signal was recorded (Fig. 2). Application of radiofrequency energy at this site resulted in the prompt termination of the tachycardia and in the occurrence of a transient "irritative" ventricular rhythm with the same morphology. After ablation, neither VT nor premature ventricular beats with the same morphology could be induced either at baseline or during isoproterenol infusion. The patient has suffered no clinical recurrence during follow-up for 10 months.

Case 2. Clinical history. The patient was a 76-year-old male affected by arterial hypertension and on treatment with amlodipine. No history of structural heart disease. He was referred for electrophysiological evaluation because in the last year he had had three pre-syncope episodes, preceded by palpitations. Twenty-four-hour Holter monitoring documented runs of non-sustained wide QRS complex tachycardia at a heart rate of 170 b/min, which caused mild dizziness. Twelve-lead ECG morphology of the tachycardia was not available. The patient was treated for a short time with mexiletine, because of the suspicion of pre-syncope VT. No documentation of narrow QRS tachycardia existed. Two-dimensional echocardiography was normal and stress testing was negative for coronary artery disease and induction of arrhythmias. Head-up tilt testing and carotid sinus massage were negative.

Electrophysiological study and radiofrequency ablation. Electrophysiological study was performed at the baseline, antiarrhythmic drug-free state; baseline activation sequence and time were normal and there was no evidence of manifest or concealed ventricular pre-excitation during atrial and ventricular stimulation. Dual atrioventricular nodal pathways were demon-

strated and a slow-fast AVNRT at a cycle length of 350 ms was inducible by means of programmed and incremental atrial stimulation. Programmed ventricular stimulation also induced a sustained wide QRS tachycardia with left bundle branch block and inferior-axis morphology (Fig. 3). Endocardial mapping and pace-mapping enabled the site of origin of the tachycardia to be located at the antero-medial part of the right ventricular outflow tract (RVOT). We considered this tachycardia to be an idiopathic VT originating from the RVOT³. Slow pathway ablation was therefore performed; this was unable to reinduce the tachycardia and disappearance of atrioventricular nodal duality. Subsequently, the RVOT was still inducible by means of ventricular stimulation. This tachycardia was ablated at the site of origin at the RVOT, where the endocardial potential preceded the QRS surface signal by 40 ms, the unipolar signal was completely negative and pace-mapping reproduced tachycardia in 12/12 leads. The ablation procedure was successful and the RVOT tachycardia was no longer inducible. However, spontaneous runs of non-sustained wide QRS tachycardia with right bundle branch block and left-axis deviation morphology were subsequently observed (Fig. 4, upper panel) and were inducible by programmed ventricular stimulation. Owing to the non-sustained character of the tachycardia, sensitivity to verapamil could not be tested. Endocardial mapping (Fig. 4, lower panel) and pace-mapping of the left ventricle revealed the origin of this tachycardia from the apical portion of the interventricular septum. At that site, the endocardial activation preceded the QRS surface ECG by 50 ms and an initial sharp high-frequency potential was recorded. Applications of radiofrequency energy to the site determined the stable disappearance of the arrhythmia. The patient was unable to recognize which

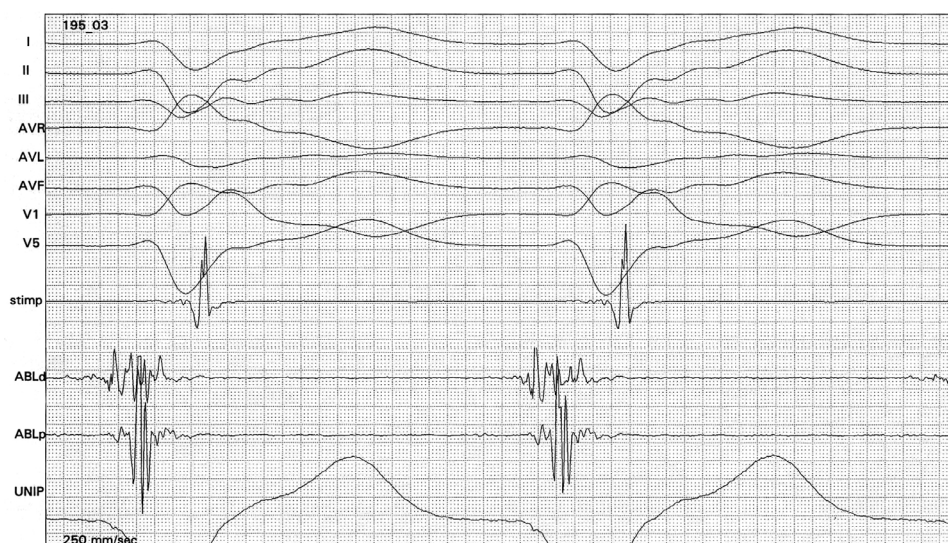


Figure 2. Case 1. Site of successful ablation of the ventricular tachycardia, located at the inferior apical left interventricular septum. Surface leads I, II, III, AVR, AVL, AVF, V₁ and V₅ are shown with intracardiac electrograms from the right ventricle (stimp) and from the ablation catheter located in the left ventricle (ABL distal, ABL proximal). UNIP = unipolar signal derived from the ablation catheter.

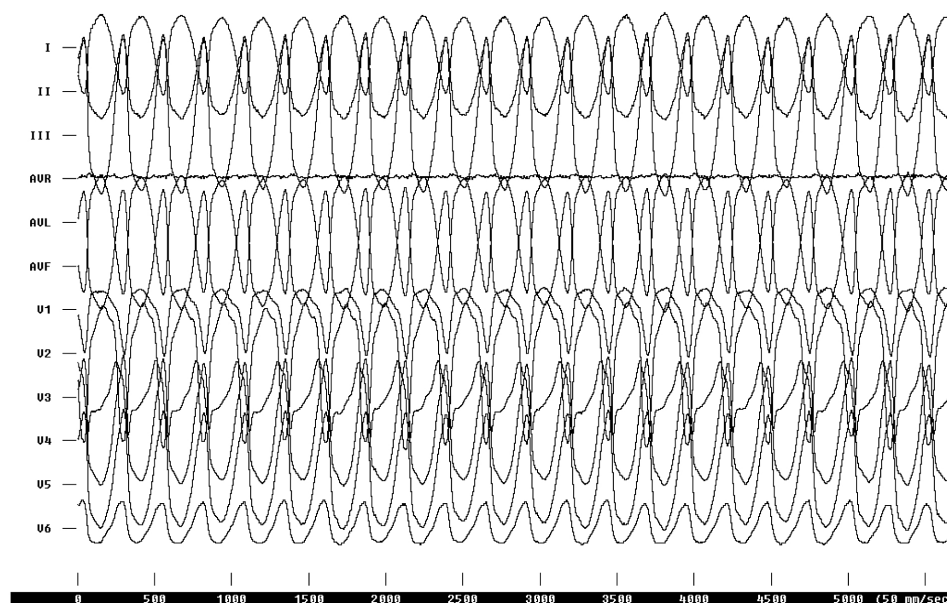


Figure 3. Case 2. Twelve-lead ECG showing the wide QRS complex tachycardia with a left bundle branch block and inferior-axis morphology at a rate of 200 b/min induced during the electrophysiological study.

of the three types of tachycardia induced during the procedure corresponded to his clinical symptoms. The patient has remained asymptomatic during a follow-up of 6 months.

Discussion

The coexistence of left or right idiopathic VT and supraventricular tachycardias, including AVNRT and atrioventricular reentrant tachycardia, has been previously described⁴⁻¹⁹. This coexistence represents a challenging diagnostic situation, especially if a transition between the two arrhythmias is observed. The substrate may be correctly identified on the basis of surface ECG data. However, complete characterization of the arrhythmias is achieved only during electrophysiological study and is a prerequisite for successful catheter ablation. We report 2 unusual cases of patients with multiple tachycardias. Some characteristics seem to differentiate our cases from those previously reported in the literature. Regarding the first case, some papers describe the association of AVNRT and verapamil-sensitive left ventricular VT^{4,7,8,10,14,18}. The peculiarity of our case, however, lies in the coexistence of these two types of tachycardia in a patient affected by coronary artery disease. AVNRT is often observed in elderly patients with structural heart disease, while verapamil-sensitive VT is generally reported in healthy subjects. On the other hand, cases of this latter arrhythmia are rarely reported in the presence of heart disease²⁰. The following elements were considered to contrast with a diagnosis of ischemic VT and suggested a diagnosis of verapamil-sensitive left VT: the possibility of inducing the arrhythmia by atrial pacing, the sensitivity to vera-

pamil, the characteristic QRS morphology, and the absence of manifest scar regions during left ventricular mapping. Because slow nodal pathway ablation did not modify the inducibility of VT, we decided to perform ablation of the ventricular arrhythmia during the same session. Moreover, other authors have observed that, although one tachycardia may trigger the other, catheter ablation of one arrhythmia substrate has not been found to influence either inducibility or clinical occurrences of the concurrent tachycardia¹⁷. It is possible that the intravenous administration of verapamil during the diagnostic electrophysiological study could have altered the acute ablation results. On the other hand, the absence of arrhythmic recurrence during follow-up does not support this hypothesis.

The second patient presented with the coexistence of three types of arrhythmia during electrophysiological study: AVNRT, sustained VT originating from the RVOT, and non-sustained VT originating from the left part of the interventricular septum. The left VT was not demonstrated to be verapamil-sensitive, but other characteristics suggested this diagnosis: the particular QRS morphology and the site of successful catheter ablation, which was located near to the distal part of the interventricular septum and was characterized by the typical spike potential preceding the local ventricular signal. Because the patient's pre-syncope episodes, which were always preceded by palpitations, were not documented by ECG (non-sustained VT observed at the ambulatory ECG caused only dizziness), it was hard to extrapolate the clinical relevance of the various types of arrhythmia. However, because all these arrhythmias were reproducible during the electrophysiological study, we decided to ablate all three tachycardias.

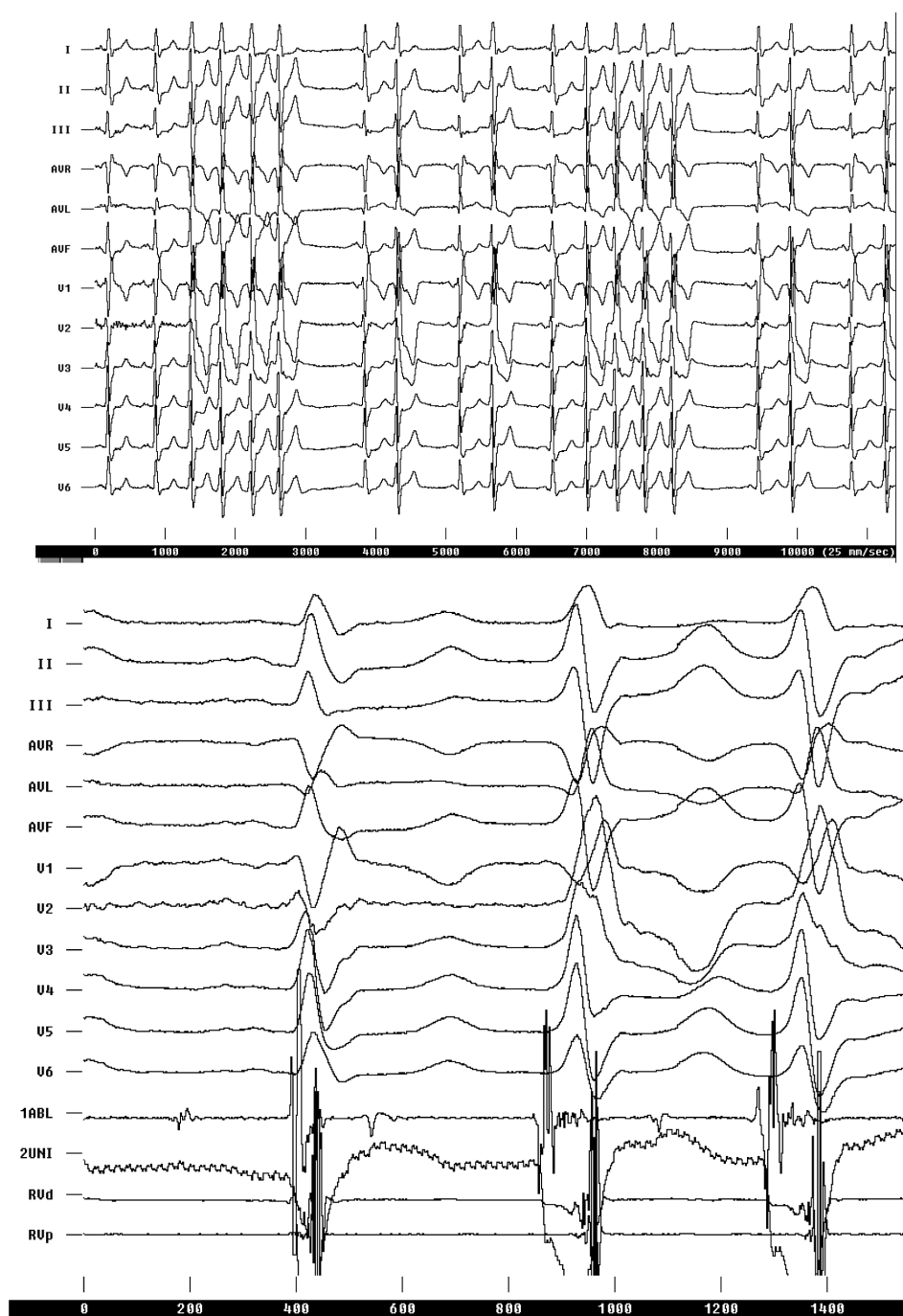


Figure 4. Case 2. Upper panel: 12-lead ECG showing spontaneous runs of non-sustained wide QRS tachycardia with right bundle branch block and left-axis deviation morphology recorded during the electrophysiological study. Lower panel: site of successful ablation of the left ventricular tachycardia. Surface leads are shown with intracardiac electrograms from the ablation catheter (ABL) located at the apical portion of the interventricular septum and from the apex of the right ventricle (RV). UNI = unipolar signal derived from the ablation catheter.

Some authors have recently speculated on the association of AVNRT and idiopathic outflow tract VT¹⁷. It is believed that both paroxysmal and repetitive forms of outflow tract VT result from cyclic adenosine monophosphate-mediated triggered activity that is typically induced by the Purkinje fibers located in the deep layer of the ventricular wall and interventricular septum. It is therefore possible that these patients may have more abundant specialized myocytes both in the

perinodal area and in the outflow tract. However, whether the coexistence of idiopathic VT and supraventricular tachycardias is a mere coincidence or derives from a common pathophysiological mechanism remains unknown. Finally, this report underlines the fact that the proper understanding of the arrhythmogenic mechanism is a fundamental prerequisite to effective treatment, and that even cases with coexisting multiple tachycardias of different origin can often be

treated by means of a single ablation procedure. Moreover, correct evaluation of the unipolar signals, especially with regard to a rapid initial negative deflection, is of great help during mapping of focal VT.

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