Circulation

## CASES AND TRACES

# Broad... Narrow... Broad QRS Tachycardia

**Gabriel Ballesteros, MD**
**Diego Menéndez, MD**
**Pablo Ramos, MD**
**Ignacio García-Bolao, MD, PhD**

## ECG CHALLENGE

This 40-year-old man was diagnosed with arrhythmogenic right ventricular cardiomyopathy (ARVC) 6 years prior when he presented to a different institution with ventricular tachycardia (VT), which was confirmed by an electrophysiology study. The electrophysiology study report stated that several VT origins were observed. He was advised against vigorous exercise.

He visited our clinic for evaluation of recurrent episodes of palpitations despite β-blocker treatment. The baseline ECG was normal, which cast doubt on the pre-

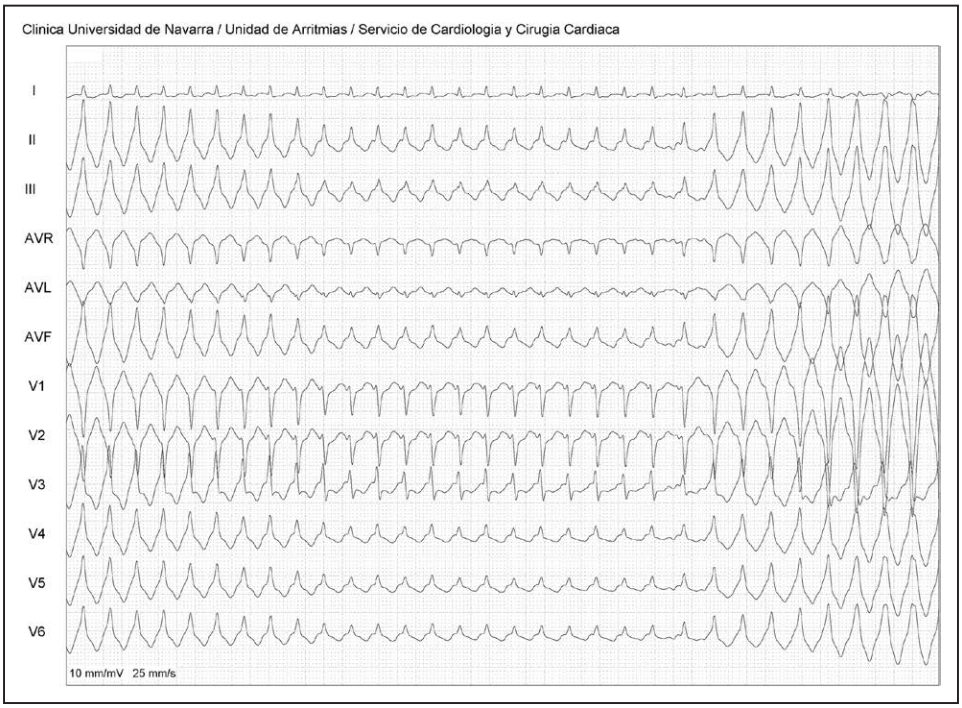


**Figure 1. During stable wide QRS complex tachycardia, the QRS progressively narrows until it becomes a regular narrow QRS tachycardia and subsequently broadens again, showing the same morphology at the end of the tracing as at the onset.**

The wide QRS complex tachycardia shows the following features that suggest ventricular tachycardia: (1) QRS >200 ms; (2) slow initial velocity of ventricular activation (Q wave >40 ms in AVR, R wave peak time in lead II ≥50 ms, and S wave nadir ≥80 ms in $V_1/V_2$); and (3) lack of RS complex in the precordium.

vious diagnosis, because in a majority of patients with symptomatic ARVC ECG abnormalities are observed, the most frequent of which is the presence of negative T waves in the anterior precordial leads ($V_1$ to $V_4$). Cardiovascular magnetic resonance imaging was performed, which showed no abnormalities, and a new electrophysiology study was then performed.

During the electrophysiology study, the ECG (Figure 1) was recorded.

Please turn the page to read the diagnosis.

## RESPONSE TO ECG CHALLENGE

In the ECG (Figure 1), the QRS complex progressively narrows until it becomes a regular narrow QRS complex tachycardia and subsequently broadens again, showing the same morphology at the end of the tracing as at the onset, simulating a polymorphic tachycardia.

During stable wide QRS complex tachycardia, the intracardiac readings show an apparent 1:1 atrioventricular conduction ratio, which was, in fact, isorhythmic dissociation, ie, 2 dissociated rhythms, one atrial and the other ventricular, with very similar rates. The continuation of the wide QRS complex tachycardia during spontaneous transient interruption of the atrial rhythm (Figure 2) excludes supraventricular tachycardia with aberrant conduction or with antegrade conduction via an accessory pathway; the isorhythmic dissociation was diagnostic of VT. On several later occasions it was observed that, during stable VT, the atrial tachycardia suddenly disappeared and atrial sinus rhythm resumed for 1 or 2 beats, to be followed by a further spontaneous induction of the atrial tachycardia, which excluded the possibility that atrial acceleration was ventriculoatrial conduction or sinus tachycardia.

During the stable wide QRS complex tachycardia, a ventricular cycle length of 302 ms (198 bpm) and an atrial cycle length of 316 ms (189 bpm) are observed. The atrial rate spontaneously and progressively accelerates until reaching a cycle length of 290 ms (206 bpm), whereas the ventricular rate remains stable, with increasing amounts of ventricular myocardium depolarizing via the normal atrioventricular conduction system; a progressive fusion is observed until the VT is suppressed because of overstimulation (Figure 3). Finally, the atrial rate progressively lowers again, and a stable wide QRS complex tachycardia is observed anew.

In short, this is a supraventricular tachycardia, with behavior suggestive of focal atrial tachycardia, and a simultaneous right ventricular outflow tract tachycar-

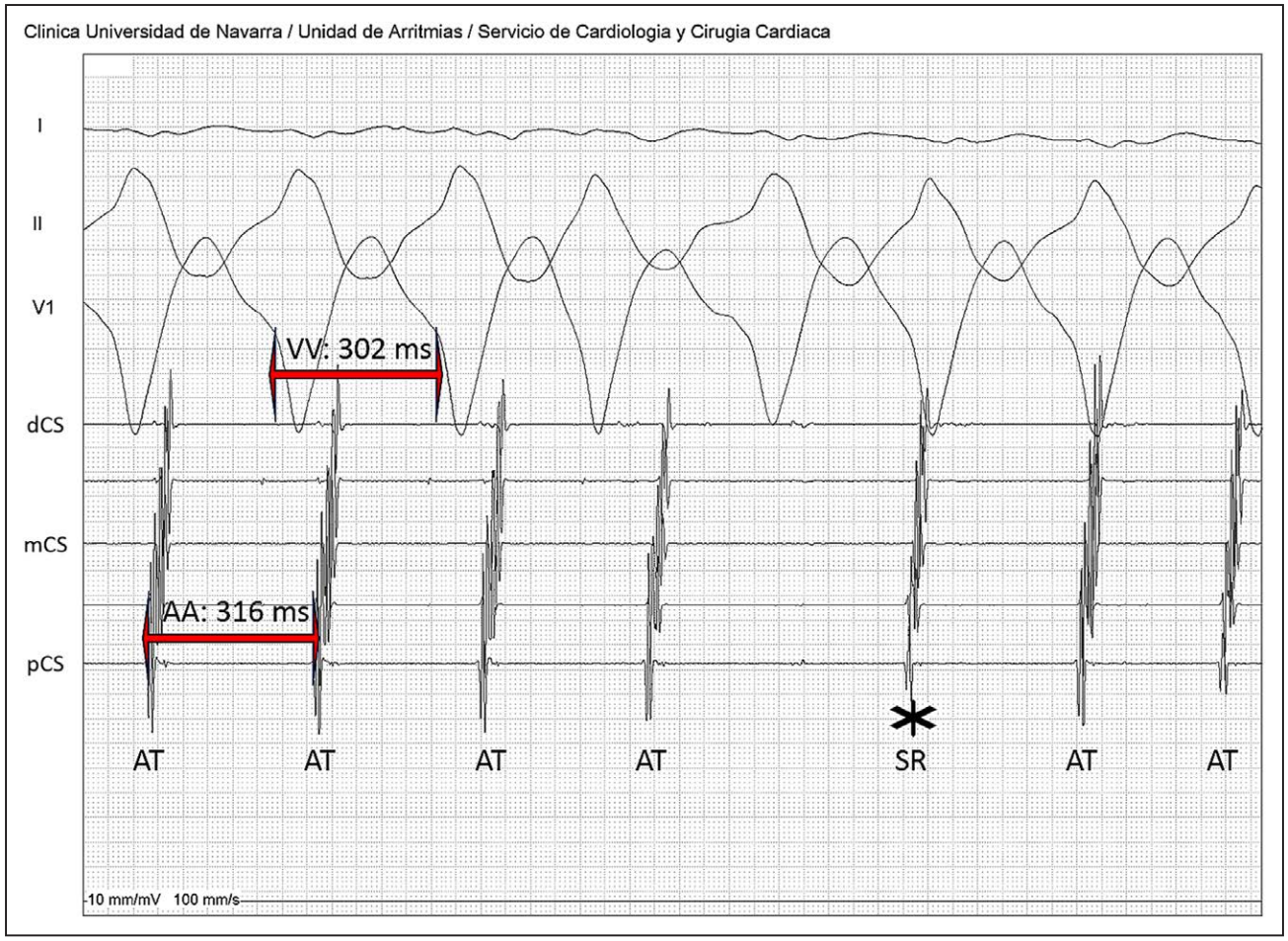


**Figure 2. Continuation of the wide QRS complex tachycardia during spontaneous interruption of the atrial rhythm.** **Upper**, ECG leads I, II, and V1 show a stable wide QRS complex tachycardia. **Lower**, Atrial electrograms from distal, middle, and proximal coronary sinus (dCS, mCS, and pCS, respectively) catheter show transient interruption (*) of the high atrial rate without modification of the ventricular rhythm, excluding ventricular acceleration as a consequence of a supraventricular tachycardia. AA and VV indicate the interval between 2 atrial and 2 ventricular electrograms, respectively; AT, atrial electrograms during atrial tachycardia; and SR, atrial electrograms during sinus rhythm beat.

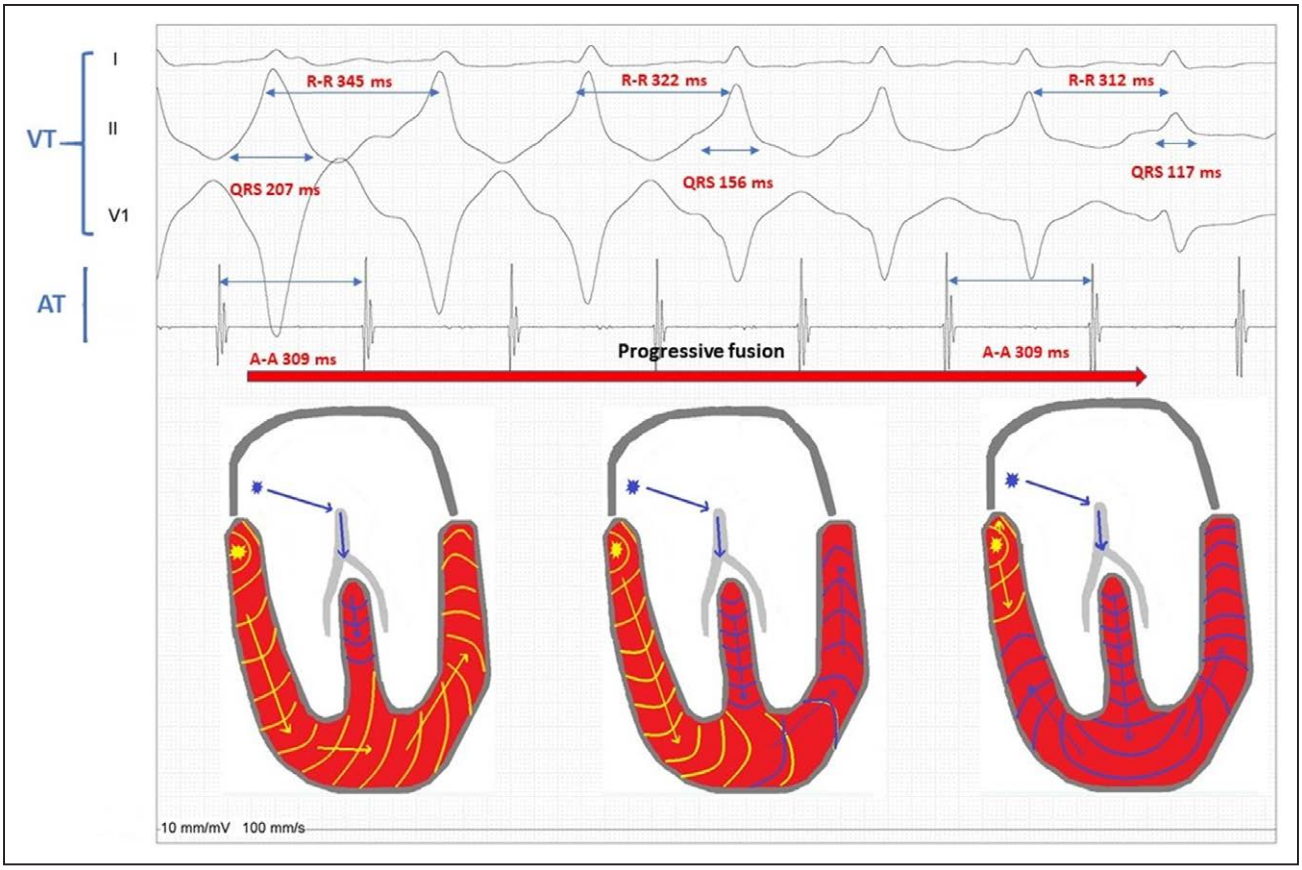


**Figure 3. There are 2 simultaneous, independent activation fronts: an atrial tachycardia (AT) that passes through the normal atrioventricular conduction system, and a ventricular tachycardia (VT) that originates in the right ventricle outflow tract.**

Because the frequency of the AT accelerates spontaneously, a greater amount of ventricular myocardium is captured through the normal conduction system, causing the QRS to narrow progressively. This is only possible if the atrial rate is higher than, and independent of, the ventricular rate, which excludes ventricle-to-atrial conduction of a VT as the cause of the atrial acceleration, leaving double tachycardia as the only possibility. A-A and R-R indicate the intervals between 2 atrial and 2 ventricular electrograms, respectively.

dia. Both have similar rates without ventriculoatrial conduction, during which various degrees of fusion are produced because of slight variations in the atrial tachycardia rate, simulating a polymorphic VT.

After the diagnosis of double tachycardia was reached, the patient underwent a focal ablation of the atrial tachycardia, which had its origin in the lower posterior part of the right atrium. After the atrial tachycardia ablation, the wide QRS complex tachycardia was monomorphic and could be mapped without difficulty. Electroanatomic mapping of VT activation demonstrated that this was a focal right ventricular outflow tract tachycardia, which terminated after the application of radiofrequency. The voltage map confirmed the magnetic resonance imaging findings of no signs of structural heart disease, definitively excluding a diagnosis of ARVC.

The patient subsequently remained asymptomatic, underwent a stress test during which no impairment was observed, and took up physical activity without recurrence of the arrhythmia.

In summary, this is an example of dual tachycardias, a focal atrial and a VT. In addition, a prior diagnosis of ARVC was excluded. The key to diagnosis for this patient was close attention to the ECG, which changed the diagnosis from ARVC, a progressive, potentially fatal genetic cardiomyopathy, to idiopathic right ventricular outflow tract tachycardia, a curable disease.

## ARTICLE INFORMATION

### Correspondence

Ignacio García-Bolao, MD, PhD, Avenida Pío XII, 36 (31008) Pamplona, Spain. E-mail igarciab@unav.es

### Affiliation

Department of Cardiology and Cardiovascular Surgery, Clínica Universidad de Navarra, Pamplona, Spain.

### Disclosures

None.

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