

# A case report of a patient with wide complex tachycardia due to Wolff–Parkinson–White syndrome mimicking ventricular tachycardia

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## Background

Wide complex tachycardia (WCT) associated with syncope as manifestation of an underlying, life-threatening arrhythmia might potentially be the harbinger of sudden cardiac death. Identifying the aetiology of a WCT is imperative to provide appropriate treatment and prevent recurrence.

## Case summary

We report the case of a 22-year-old male who had been experiencing haemodynamically significant WCT leading to syncope at the age of 13 years. As the patient and the family rejected an electrophysiological (EP) study, he had received an implantable cardioverter-defibrillator (ICD) for secondary prevention. After 7 years of experiencing multiple shocks, the patient finally gave consent to an EP study, which identified a left-sided accessory atrioventricular pathway that was successfully ablated during the same procedure.

## Discussion

The differential diagnosis of WCT might be challenging and includes both ventricular and supraventricular tachycardias. In young patients without structural heart disease experiencing WCT, an EP study should be offered before ICD implantation to make a final diagnosis with the potential to provide definitive treatment.

## Keywords

Case report • Accessory atrioventricular pathway • Wolff–Parkinson–White syndrome • Wide complex tachycardia • Electrophysiological study • Radiofrequency ablation

## Learning points

- An electrophysiological (EP) study is a safe and effective method to evaluate the aetiology of wide complex tachycardia (WCT), with the possibility of curative treatment using catheter ablation.
- An EP study should be offered to every patient with WCT of unclear aetiology before implantation of an implantable cardioverter-defibrillator.
- The presence of an accessory atrioventricular pathway in young patients without a structural heart disease suffering WCT should always be considered as signs of pre-excitation on the surface electrocardiogram might be very subtle.

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## Introduction

Wide complex tachycardia (WCT) is characterized by a heart rate of more than 100 beats per minute along with a wide QRS complex.<sup>1</sup> In a young patient, WCT causing syncope raises suspicion of an underlying life-threatening arrhythmia. Identifying the aetiology is essential to provide appropriate treatment.<sup>2</sup> We report the case of a young patient with WCT of unknown aetiology who finally underwent an electrophysiological (EP) study leading to the diagnosis of Wolff–Parkinson–White (WPW) syndrome.

## Timeline

July 2009	Syncope during physical education, with no cause identified
September 2013	Syncope while exercising, with an underlying wide complex tachycardia (WCT); termination under intravenous administration of amiodarone
October 2013	Implantation of an implantable cardioverter-defibrillator (ICD) for secondary prevention; magnetic resonance imaging performed with no evidence of arrhythmogenic right ventricular cardiomyopathy or other, structural heart diseases
2013–2021	In total, seven episodes of WCT were recorded by the ICD, requiring up to five shocks per episode for termination
March 2021	Electrophysiological study performed with identification and successful ablation of a left-sided accessory atrioventricular pathway; no ventricular tachycardia (VT) inducible

## Case presentation

A 22-year-old male was admitted to our electrophysiology department for an EP study. For more than 10 years, he had been experiencing WCT (Figure 1) of unclear aetiology leading to syncope. An EP study had been offered at different times but was rejected. The patient's parents who were taking medical decisions at that time were very reluctant towards an EP study as they were afraid of procedure-related complications. However, in view of the potentially life-threatening event (after a first syncope while exercising 4 years earlier), the parents accepted implantation of a subcutaneous implantable cardioverter-defibrillator (ICD; SX-RX 1010, Cameron Health Inc.) in 2013. Since ICD implantation, multiple WCT episodes were recorded and shocked by the device (Supplementary material online, Figure S1). Most episodes developed when exercising. During the episodes, he experienced dizziness, rapid heartbeats, heavy sweating, and intense fear. Otherwise, he reported hardly any physical complaints, but over time felt highly restricted in his daily living because of the fear of receiving shocks at any moment. The patient had no other pre-existing medical conditions and denied use of alcohol

and illicit drugs. The only medication he was taking on a regular basis was the beta-adrenergic blocking agent bisoprolol (2.5 mg bid). His family history was unremarkable with respect to sudden cardiac death (SCD), cardiac arrhythmias, or channelopathies. Accordingly, electrocardiograms (ECGs) obtained from both parents were unremarkable. Exercise stress tests and Holter ECGs were performed without significant findings. A genetic testing had been scheduled but was not attended. He had undergone magnetic resonance imaging in 2013 with no evidence of a structural heart disease, in particular no signs of arrhythmogenic right ventricular cardiomyopathy.

After several years of receiving numerous ICD shocks, the patient felt increasingly frustrated with his situation. As a 22-year-old adult, he came back to the suggestion of an EP study and gave his consent.

Physical examination on the day of admission revealed no significant findings and lab values were normal apart from mild hypokalaemia. An ECG was recorded showing sinus bradycardia without signs of pre-excitation, ST-segment alterations, or a prolongation of the QT interval (Supplementary material online, Figure S2). Echocardiography displayed a normal left ventricular systolic function.

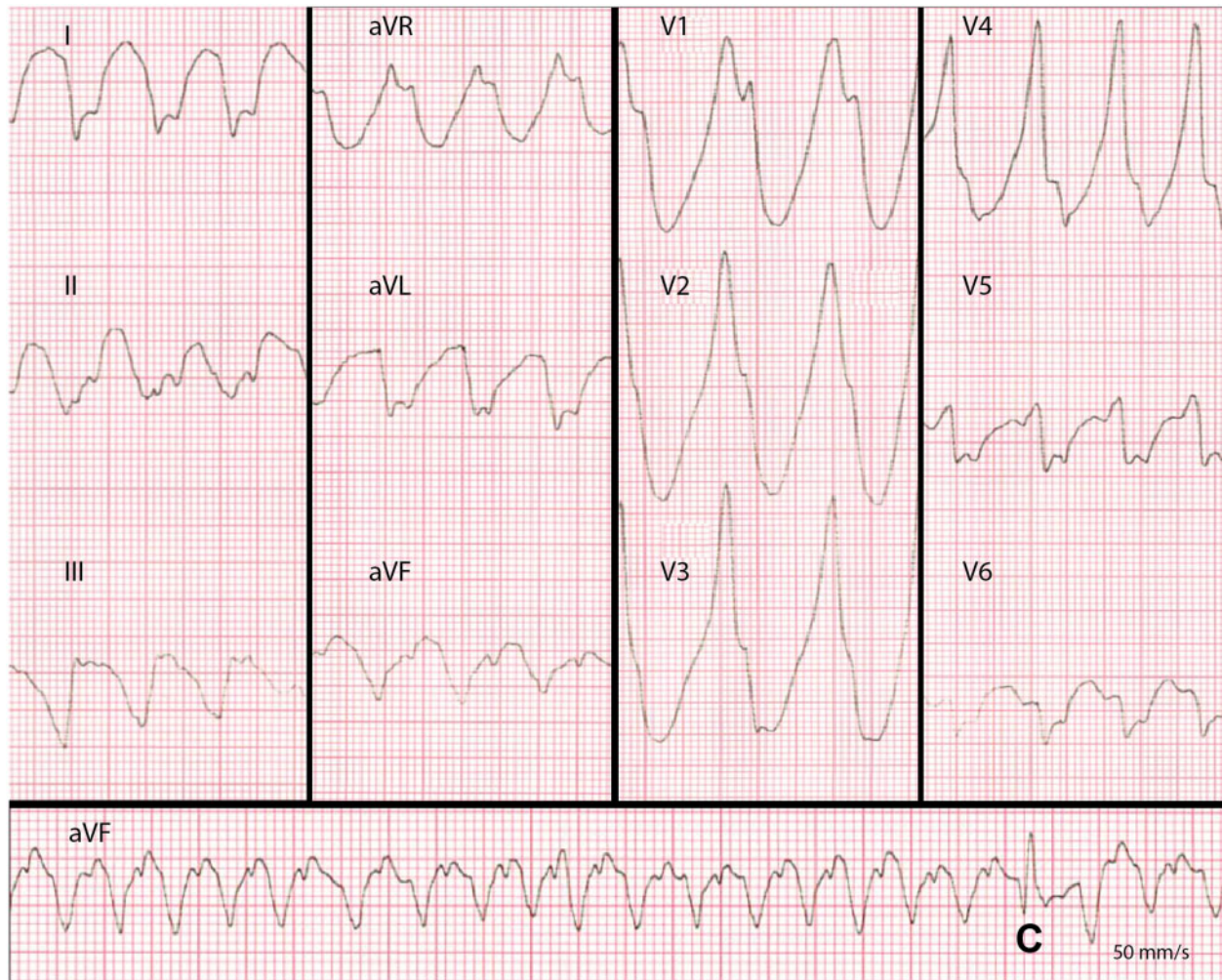
The patient underwent an EP study on the following day. Intracardiac electrogram tracings [coronary sinus (CS) 1/2] during sinus rhythm revealed premature ventricular activation of the lateral mitral annulus (Figure 2). This initial finding indicated the presence of a left-sided accessory atrioventricular pathway (AP). Accordingly, direct pacing at CS 1/2 produced an increasing degree of pre-excitation (Supplementary material online, Figure S3). Rate incremental ventricular pacing resulted in a distal to proximal atrial activation sequence with the earliest site of atrial activation at the distal CS due to a non-decremental conduction of the AP (Figure 3). The anterograde effective refractory period of the AP was defined as the longest basic cycle length which failed to conduct with pre-excitation and yielded 240 ms in the patient indicating an AP with an increased risk of SCD. Ultimately, orthodromic atrioventricular reentrant tachycardia (AVRT) without aberrancy and a cycle length of 316 ms (Figure 4) was induced. Neither atrial fibrillation (AF) nor a WCT were inducible during the EP study.

Using a retrograde transaortic approach, the AP was localized using a mapping catheter by displaying the earliest ventricular activation in pre-excited sinus rhythm. Electroanatomic mapping denoted activation of the left ventricle by conduction via the AP as well as impulse propagation (Video 1). Radiofrequency energy was delivered at the site of earliest ventricular activation at the lateral mitral annulus (CS 1/2), resulting in disappearance of pre-excitation (Figure 5). Programmed ventricular stimulation did not induce ventricular tachycardia (VT).

Follow-up on the day after the procedure revealed no complications and the patient was discharged without medications. An appointment for the ICD explantation was scheduled to take place 3 months after the EP procedure.

## Discussion

In WPW syndrome, pre-excitation may not always be evident on ECG, potentially obscuring the presence of an AP.<sup>3</sup> We present the case of a young patient experiencing haemodynamically significant



**Figure 1** Twelve-lead electrocardiogram recorded in 2013 showed a wide complex tachycardia with right bundle branch block-shaped wide QRS complexes with intermittent narrow QRS complexes (C, capture beats).

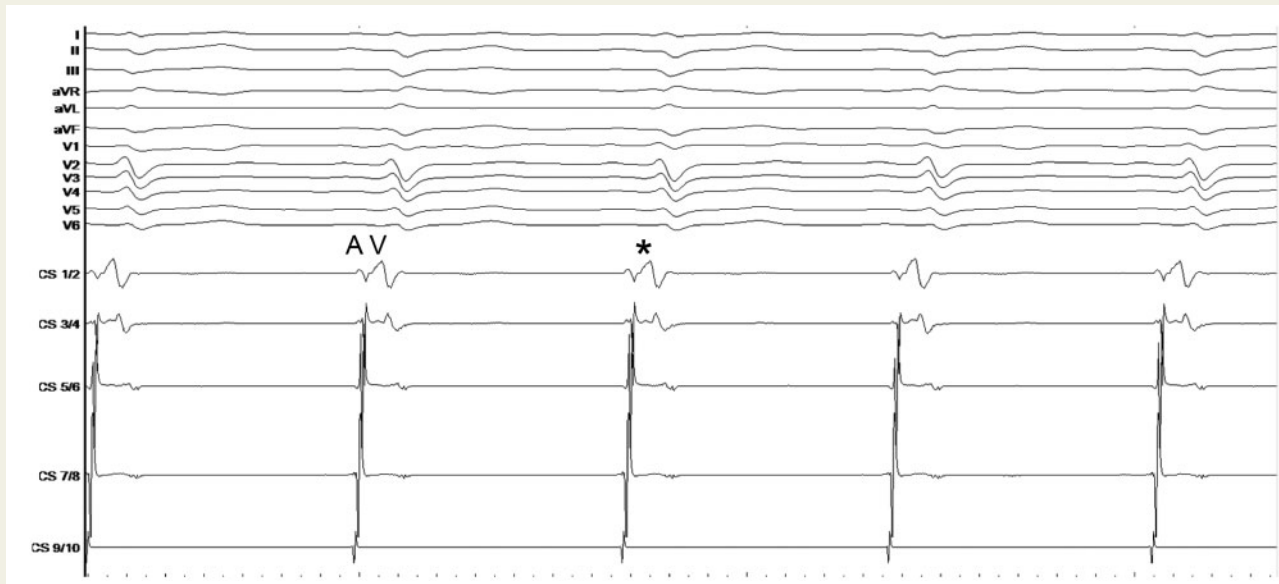
WCT in whom the presence of an AP became evident only after undergoing an EP study which was initially refused.

While the prevalence of pre-excitation on ECG is reported to range from 0.1% to 0.3%,<sup>4</sup> it is not exactly clear how frequently an AP is existent without being manifest on ECG, given that the majority of patients remains asymptomatic.<sup>5</sup> Pre-excitation may be very subtle or inapparent if atrioventricular (AV) nodal conduction is faster than conduction via the AP. This is especially true for left (lateral) APs as seen in our case (*Video 1*) favouring AV nodal conduction.<sup>6</sup>

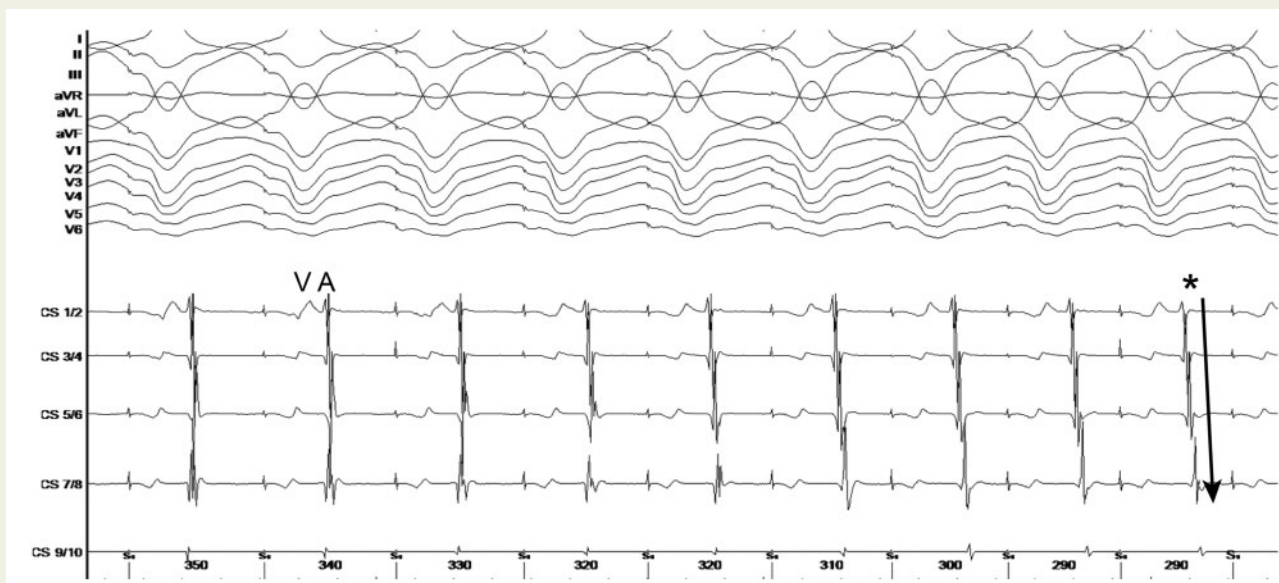
Wide complex tachycardia in the presence of an AP can be due to the following: AF/atrial flutter with conduction via the AP; antidromic AVRT; orthodromic AVRT with a bundle branch block; VT; and other forms.<sup>7</sup> Interestingly, AF is present in nearly one-third of patients with a WPW syndrome. If an AP with a short anterograde refractory period is present (as seen in our patient), AF can result in fast ventricular rates potentially degenerating into ventricular fibrillation and leading to SCD. Atrioventricular reentrant tachycardia might also degenerate into AF.<sup>8</sup> Atrioventricular reentrant tachycardia by

itself, in contrast, is usually well tolerated<sup>9</sup> and coexisting VT is very uncommon, especially if no structural heart disease is present.<sup>10</sup> Accordingly, VT was not inducible during the EP study.

In retrospect, it is hard to determine the underlying WCT mechanism (*Figure 1*) in the patient. In synopsis of the above considerations, AF either occurring spontaneously or as a result of degeneration from preceding AVRT with rapid conduction to the ventricles over the AP appears to be the most plausible mechanism. In this context, the 'capture beat' seen in the WCT (*Figure 1*) is highly interesting. We assume that it represents an atrial activation that was conducted by the AV node while the AP was refractory. Alternatively, it might represent a premature ventricular contraction (PVC) fused with an activation conducted by the AP. Given the comparatively narrow morphology of the capture beat, the PVC would have to be parahisian or arise from the right ventricle. In the latter case, the PVC from the right ventricle would coincidentally occur at the same time as activation via the AP, creating something like a spontaneous resynchronization. In summary, in our case the 'capture beat' that is a finding



**Figure 2** Surface electrocardiogram and electrogram tracings of the coronary sinus indicated pre-excitation with the earliest site of atrioventricular conduction in the lateral mitral annulus (coronary sinus 1/2, asterisk). A, atrial potential; V, ventricular potential.

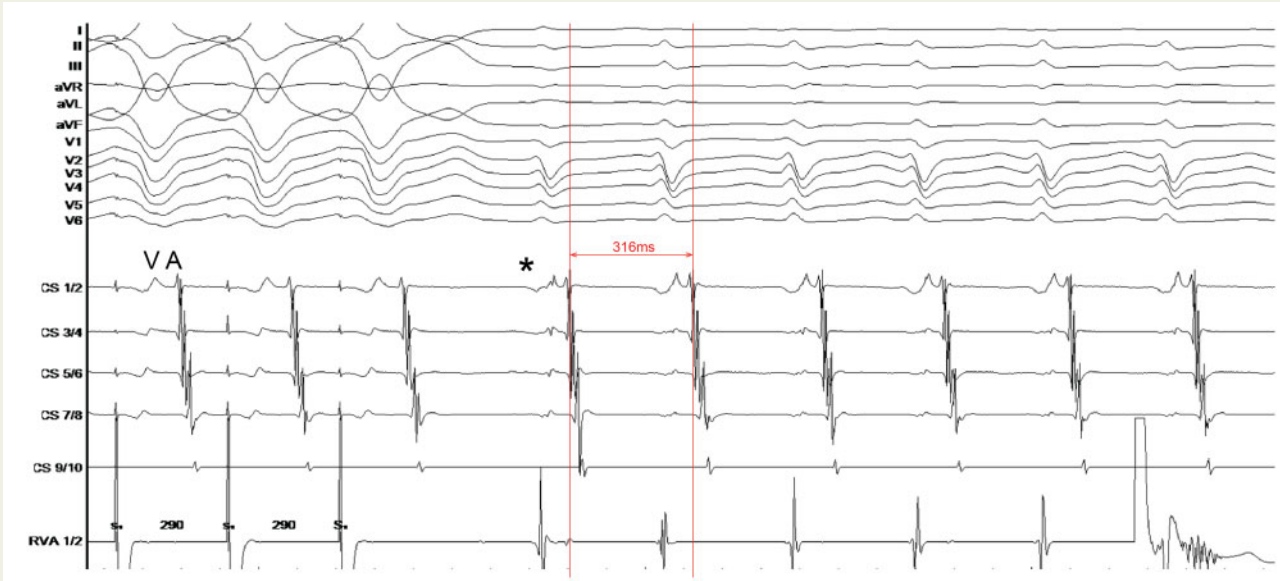


**Figure 3** Rate-incremental ventricular pacing lead to a distal to proximal atrial activation sequence with the earliest atrial signal at the lateral mitral annulus (coronary sinus 1/2, asterisk) due to decremental conduction in the atrioventricular node (arrow). A, atrial potential; CS, coronary sinus; V, ventricular potential.

normally strongly indicative of VT was one of the factors triggering the wrong diagnosis of VT.

We would also like to highlight the importance of the EP study itself in our patient. Only after undergoing the EP study, the presence of an AP became evident and the diagnosis WPW syndrome was made. The AP was successfully targeted within the same procedure

(Figure 5). Success rates of AP ablation have been reported to be as high as 95%, while recurrence is seen in <5% of cases.<sup>11</sup> Procedure-related complications are rare.<sup>12</sup> In contrast, medical therapy of WPW syndrome, which includes numerous antiarrhythmics targeting the AV node, AP, or both, is moderately successful. Medical therapy is often used only after the onset of tachycardia to terminate it and is



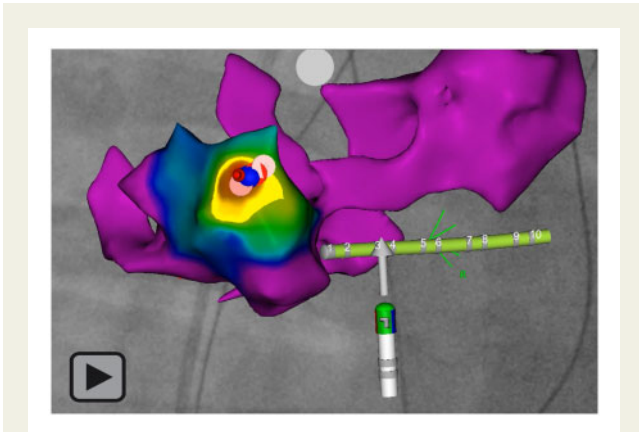
**Figure 4** Ventricular pacing with a cycle length of 290 ms resulted in an orthodromic atrioventricular reentrant tachycardia with a cycle length of 316 ms. The onset is indicated by an asterisk. A, atrial potential; CS, coronary sinus; RVA, right ventricular apex; V, ventricular potential.



**Figure 5** Mapping of the atrioventricular pathway was achieved by displaying the earliest ventricular activation. Applying radiofrequency energy, the atrioventricular pathway was successfully eliminated (arrow). Note that following ablation, the ventricular signal on both the mapping catheter (Map 1/2) and the distal coronary sinus (1/2) occurs after the onset of the QRS. A, atrial potential; CS, coronary sinus; RVA, right ventricular apex; V, ventricular potential.

further limited by side effects.<sup>13</sup> Of note, AF can resolve following successful elimination of the AP.<sup>8</sup> VT being the most common aetiology of WCT overall is also amenable to ablation.<sup>14</sup> Performing the EP study at an earlier point, at the latest perioperatively just before ICD implantation, would have prevented the ICD implantation and subsequently the occurrence of many unpleasant shocks.

In conclusion, in young patients without structural heart disease experiencing WCT, performing an EP study seems mandatory to make a final diagnosis before implanting an ICD. Various aetiologies including an AP or VT might effectively be treated using catheter ablation, thus rendering ICD implantation unnecessary.



**Video 1** Activation map of the left ventricle, posterior view. Impulse propagation highlights the presence of a left lateral atrio-ventricular pathway facing the distal coronary sinus (1/2). Green-Blue, onset of activation; Purple, non-depolarized tissues; Red, depolarized tissues. Horizontal catheter: coronary sinus. Vertical catheter: mapping catheter.

## Lead author biography



Stefan Preisendörfer is a resident of cardiology at the German Heart Center in Munich, Germany. He received his medical training in Germany, Denmark, and Australia.

## Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

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**Slide sets:** A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

**Consent:** The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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