

Wolff-Parkinson-White (WPW) Syndrome — Causes, ECG and Treatment

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Louis Wolff published an article in 1930 that described 11 patients who had episodes of tachycardia with characteristic ECG findings (ECG pattern showed bundle branch block and a shortened PR interval). In 1943, the anatomical accessory pathway of conducting tissue was described and showed that this pathway bypassed the atrioventricular conduction system. This article discusses the definition, pathophysiology, clinical manifestations, diagnosis, and treatment of Wolff-Parkinson-White Syndrome.



Definition

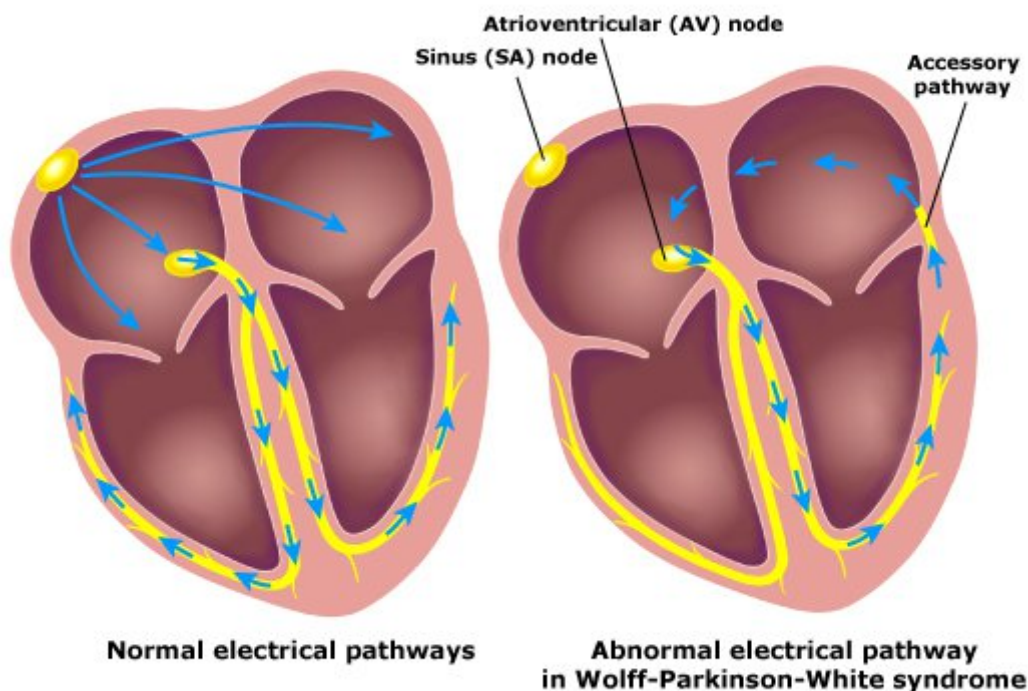


Image: Wolff-Parkinson-White Syndrome. By Tom Lück, License: [CC BY 3.0](#)

Wolff-Parkinson-White (WPW) syndrome is defined as **tachyarrhythmia in which attacks of tachycardia occur in a patient due to the presence of an abnormal accessory pathway in the heart, which bypasses the conduction, fully or partially, via the atrioventricular (AV) node to the ventricles**. The prognosis and long-term outcome of WPW syndrome are very good, especially if treatment aims to

eliminate the abnormal accessory pathway.

Epidemiology

The prevalence of WPW syndrome and pattern is **less than 1% of the population** with the WPW pattern being 100 times more common than WPW syndrome. The difference between both presentations will be discussed later in this article.

Pathophysiology

First, it is important to understand the normal conducting pathway of the heart, and how the impulses spread uniformly throughout the heart. Normally, **the atria and ventricles are electrically isolated, where the discharged electrical impulses move from the SA node in the right atrium to the ventricles via the AV node and His-Purkinje system.**



[Image](#): A characteristic delta wave seen in a patient with WPW. Note short PR interval. By James Heilman, MD, License: [CC BY-SA 3.0](#)

Patients with the pre-excitation syndrome have an anatomical accessory pathway that directly conducts impulses from the atria to the ventricles, bypassing the AV node of the heart and conducting impulses faster than the AV node without its characteristic delaying property. This results in a short PR interval seen on [ECG](#). This accessory pathway is congenital in origin and results from the failure of resorption of the myocardial syncytium at the annulus fibrosus during fetal development.

- About 75% of the accessory pathway can conduct impulses in both directions (anterograde and retrograde) between the atrium and ventricle.
- About 35% of the accessory pathway is only able to conduct the impulses in a retrograde fashion from the ventricle to the atrium – the so-called ‘concealed’ accessory pathways (most of them are left-sided, which does not create a delta wave and the WPW pattern on ECG, but is still able to support reentrant tachycardia).
- About 25% of the accessory pathway only conducts impulses in an anterograde manner from the atria to the ventricles.

The mechanism of the unidirectional conduction of impulses along the accessory pathway

in either an **anterograde** or **retrograde** direction remains undetermined.

Anatomy of accessory atrioventricular pathways

Based on electrophysiological studies, accessory atrioventricular pathways are located in any place along the AV ring or in the interventricular septum. The most frequently documented locations are:

- 50% → Left lateral
- 30% → Posteroseptal
- 10% → Right anteroseptal
- 10% → Right lateral

Wolff-Parkinson-White pattern and syndrome

Patients with an accessory pathway can be described as having either the WPW pattern or WPW syndrome based on the presence or absence of [arrhythmias](#).

Wolff-Parkinson-White pattern

This term applies to a patient with ECG findings of pre-excitation in the **absence of symptomatic arrhythmias**.

Wolff-Parkinson-White syndrome

This term applies to a patient with ECG findings of pre-excitation and **symptomatic arrhythmias**. In either situation, anterograde conduction of impulses, from the atria to the ventricles through an accessory pathway, will result in earlier activation or pre-excitation of part of the ventricles.

Clinical Manifestations

The vast majority of patients with WPW pattern are **asymptomatic**, while a **small percentage have arrhythmias** (for example, atrial fibrillation with rapid ventricular response) as a part of the WPW syndrome. Patients with WPW syndrome and developed arrhythmia will present with any of the following manifestations:

- Palpitations
- Lightheadedness and/or dizziness
- Syncope or presyncope
- Chest pain
- Sudden cardiac death

Arrhythmias associated with Wolff-Parkinson-White

WPW syndrome can be associated with either:

1. **Tachycardias requiring an accessory pathway for initiation and maintenance**

Atrioventricular reentrant tachycardia (AVRT): This occurs when the heart has a circuit that consists of 2 pathways – a normal AV conduction system and the AV

accessory pathway, where both are linked by tissues. If there are adequate differences in conduction time and refractoriness between the normal conducting system and the bypass tract, premature impulse from the atrium of the ventricle can initiate reentry.

The 2 main types of this arrhythmia in WPW syndrome are **orthodromic and antidromic AVRT**.

2. Tachycardias not requiring an accessory pathway for initiation and maintenance

The heart consists of an accessory pathway, but is not involved in the initiation of arrhythmia, and includes **atrioventricular nodal reentrant tachycardia (AVNRT)**, **ventricular tachycardia**, and **ventricular fibrillation**.

Patients with accessory pathways are at risk of [atrial fibrillation](#). If the accessory pathway rapidly conducts impulses in a retrograde direction from the atrium to the ventricle in a patient with atrial fibrillation, a rapid ventricular response would occur which may result in ventricular fibrillation.

Diagnosis

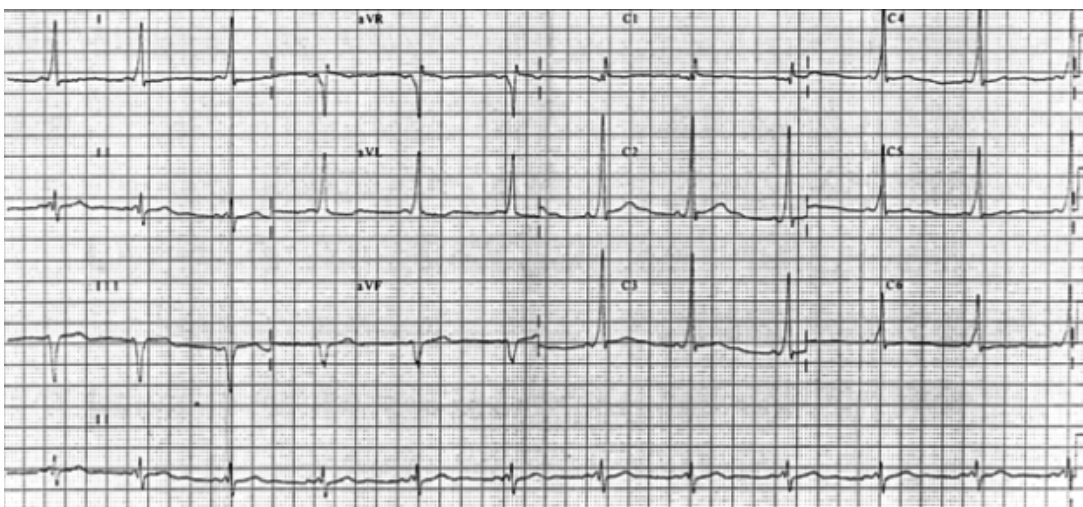
Wolff-Parkinson-White pattern

Diagnosis of the WPW pattern requires only **ECG** which shows characteristic findings.

Wolff-Parkinson-White syndrome

Diagnosis of WPW syndrome involves the **identification of the WPW pattern on surface ECG** of a patient who develops arrhythmia, especially in young adults presenting with paroxysmal arrhythmia.

ECG findings



[Image](#): A 12 lead ECG demonstrating Wolff-Parkinson-White syndrome with characteristic delta waves. By Ksheka, License: [CC BY-SA 3.0](#)

The main feature of the AV accessory pathway is **pre-excitation**, where the ventricles are activated by the direct activation of the myocardium throughout the accessory pathway. Thus, the ventricles are activated earlier than expected after atrial

depolarization, resulting in:

- **Shortening of the PR interval (< 0.12 seconds)** occurs as a result of rapid AV conduction via the accessory pathway, bypassing the AV node
- **Delta wave**, which arises because the beginning of ventricular depolarization is slowed and the QRS complex upstroke is slurred because of the slowing of the conduction from muscle fiber to another muscle fiber. Slow muscle fiber-to-muscle fiber conduction is noted.
- **Widening of the QRS complex**

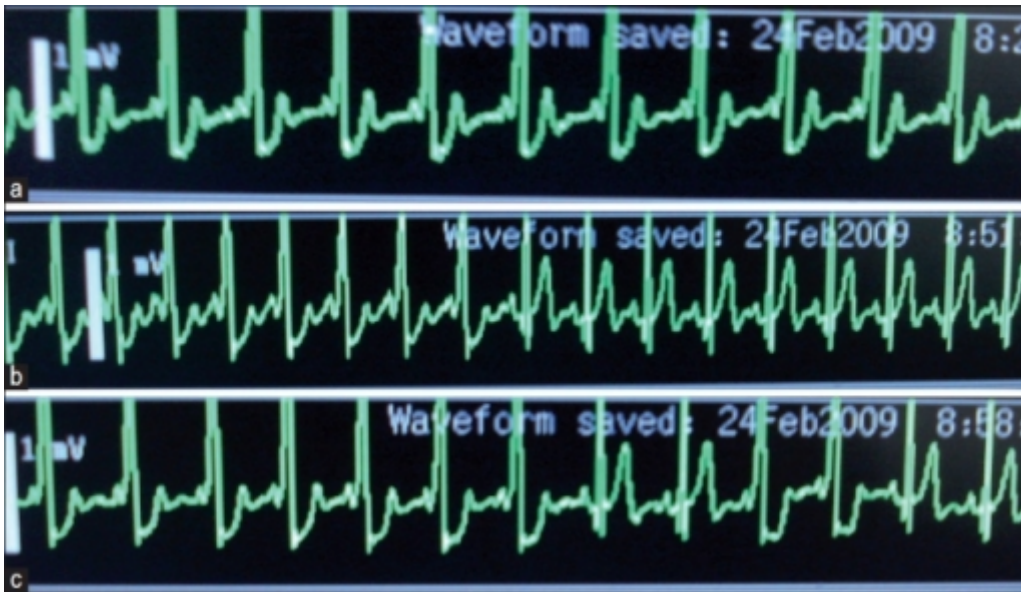


Image: (a) Sinus rhythm followed by Wolff-Parkinson-White, (b) Sinus rhythm, (c) Wolff-Parkinson-White rhythm.
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Concealed accessory pathways

As mentioned earlier, in about 35–50% of patients, accessory pathways conduct the impulses in the retrograde direction only from the ventricle to the atrium. In this condition, the accessory pathway manifests only during sustained tachycardia. Concealed accessory pathways can be identified by the time, and by how the atrium is activated during tachycardia:

- **P wave follows ventricular depolarization and a short RP wave interval** can be seen.
- Sometimes accessory pathways conduct the retrograde impulses very slowly, which may result in longer retrograde conduction, developing a **longer RP interval during tachycardia** (long RP tachycardia).

Therapy

Patients with WPW syndrome are treated because they either have:

1. Symptomatic arrhythmia
2. Risk of arrhythmia, for example, pre-excited atrial fibrillation, or atrial flutter with a rapid ventricular response

Asymptomatic WPW pattern does not need treatment.

Orthodromic AVRT (tachycardia with a narrow QRS complex)

The antegrade limb (the pathway that conducts impulses to the ventricle) is the AV node and His-Purkinje system, thus, ventricles are not pre-excited.

Acute termination

The immediate treatment of macroreentrant orthodromic tachycardias is similar to that for AVNRT, which aims to alter impulse conduction in the AV node.

- **Maneuvers that increase vagal tone:** Valsalva maneuver and carotid sinus pressure can depress AV nodal function, resulting in AV node block and tachycardia termination
- **IV adenosine and calcium channel blockers:** IV adenosine is the first-line pharmacologic therapy for the termination of tachycardia, followed by IV calcium channel blockers such as verapamil
- **Intravenous procainamide, B-blockers, and digoxin:** They are the second-line treatment for terminating the tachycardia.

Antidromic AVRT (tachycardia with a wide QRS complex)

The antegrade limb is the accessory pathway, thus, ventricles are pre-excited. Treatment should be aimed at **preventing rapid ventricular response** if the patient manifests with pre-excitation and atrial fibrillation in order to avoid atrial fibrillation.

- **In unstable patients:** Electrical DC cardioversion should be used to rapidly correct the atrial fibrillation.
- **In stable patients:** Procainamide administered at a dose of 15 mg/kg will slow the rapid ventricular response and may correct the atrial fibrillation.

Note: Care is necessary when attempting to slow the AV nodal conduction with digoxin or calcium channel blockers because they may result in accelerating conduction over the accessory pathway (accessory pathway) which may result in atrial fibrillation.

Catheter ablation therapy

WPW: Catheter Ablation of Accessory Pathway

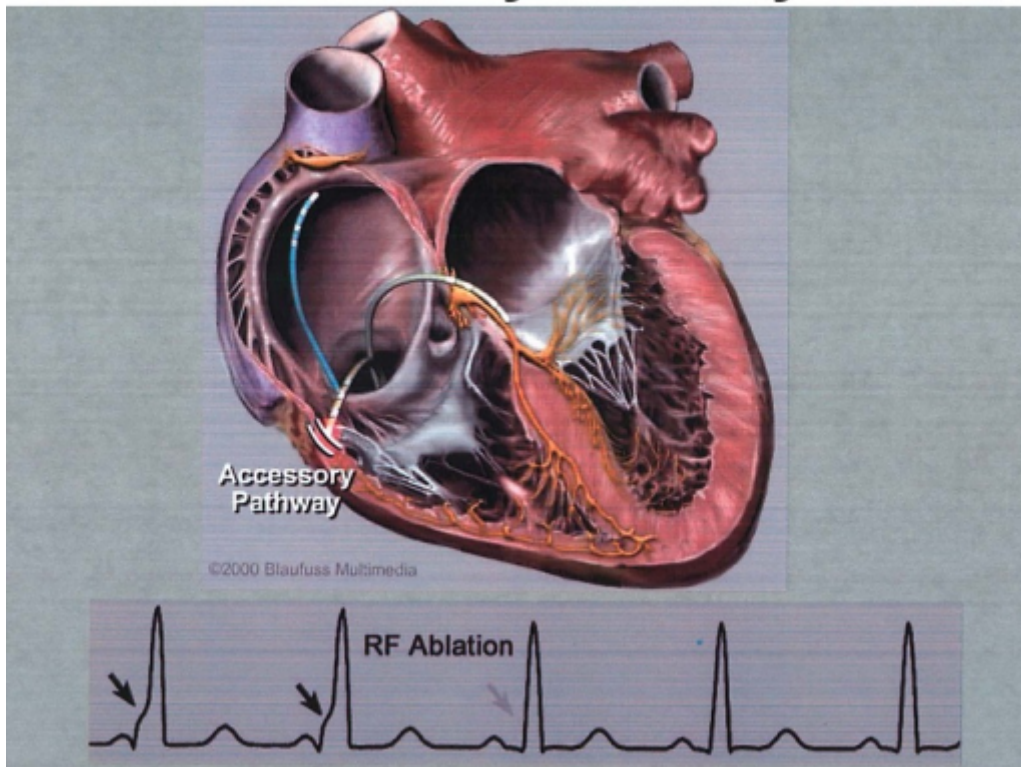


Image: Schema showing the use of catheter technique for ablation of a right free wall accessory pathway. The rhythm strip shows the disappearance of the delta wave with the application of radiofrequency energy. By Openi, License: [CC BY 3.0](https://creativecommons.org/licenses/by/3.0/)

Catheter ablation therapy appears to be **effective in more than 90% of patients** with WPW syndrome regardless of age. It is indicated in a patient with a history of:

1. Recurrent symptomatic supraventricular tachycardia
2. Incessant supraventricular tachycardia
3. Heart rates > 200/min with supraventricular tachycardia

Prevention

Prevention of recurrent supraventricular reentrant tachycardias that are associated with accessory pathways (accessory pathways) is possible with the chronic oral administration of **B-blockers and/or calcium channel blockers (verapamil or diltiazem)**.

Patients with atrial fibrillation and rapid ventricular response should receive class IA or IC antiarrhythmic drugs, such as quinidine, flecainide or propafenone, to slow conduction through the accessory pathway and increase its refractory period.

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