

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

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Wolff-Parkinson-White Syndrome (WPW) is a term referring to Louis Wolff who published an article in 1930 that describes 11 patients suffering from episodes of tachycardia with characteristic ECG findings (ECG pattern shows bundle branch block and shortened P-R interval). In 1943, anatomical accessory pathway of conducting tissue has been described that bypasses the atrioventricular (AV) conduction system. This article will describe the definition, pathophysiology, clinical manifestations, diagnosis, and treatment of Wolff-Parkinson-White Syndrome.



Definition of WPW

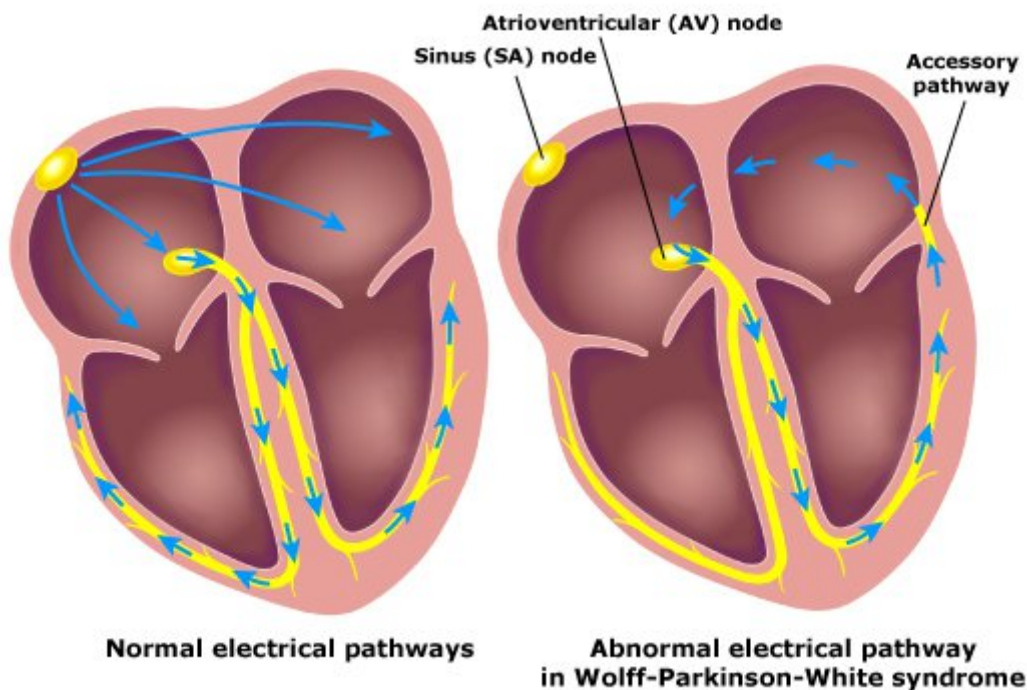


Image: "Wolff-Parkinson-White Syndrome" by Tom Lück. License: [CC BY 3.0](#)

Wolff-Parkinson-White Syndrome (WPW) 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 atrioventricular (AV) node to the ventricles. The prognosis and long-

term outcome of WPW is very good, especially if the syndrome treatment was targeted to eliminate the abnormal accessory pathway.

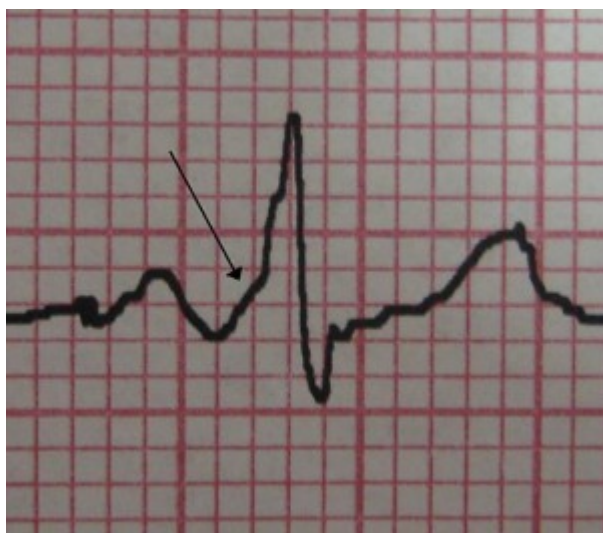
Epidemiology of WPW

Spread of Wolff-Parkinson-White Syndrome

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

Pathophysiology of WPW

Firstly, it is important to know about the normal conducting pathway of the heart, and how the impulses spread uniformly in the whole heart. Normally, **both atria and ventricles are electrically isolated, where the discharged electrical impulses move from the SA node in the right atrium, to the ventricles via atrioventricular node (AV) and the 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 anatomically accessory pathway (AP) which conducts the impulses from atria to ventricles directly, bypassing the AV node of the heart and conducts the impulses faster than the AV node without its characteristics delaying property, resulting in a short P-R interval seen on an [ECG](#). This accessory pathway is congenital in origin, and results from the failure of resorption of the myocardial syncytium at the annulus fibrosis in the intrauterine life during fetal development.

- About 75% of the AP can conduct the impulses in both directions (anterograde and retrograde) between the atrium and ventricle.
- About 35% of the AP are only able to conduct the impulses retrograde from the ventricle to the atrium and so called "concealed" accessory pathways (most of them are left-sided, which don't create a delta wave and the WPW pattern on the ECG, but are still able to support reentrant tachycardia).

- About 25% of the AP only conducts the impulses anterograde from the atria to the ventricles.

The mechanism of the unidirectional conduction of impulses along the accessory pathway in either **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

WPW pattern and WPW syndrome

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

Wolff-Parkinson-White pattern

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

Wolff-Parkinson-White syndrome

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

Clinical Manifestations of WPW

The vast majority of patients with WPW pattern are **asymptomatic**, while a **small percentage have arrhythmias** (for example, AF with rapid ventricular response) as a part of the WPW syndrome. Patients with a 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 WPW

WPW syndrome can be associated with either:

1. Tachycardias requiring an accessory pathway for initiation and maintenance

Atrioventricular reentrant tachycardia (AVRT): Occurs when the heart has a circuit which consists of 2 pathways: Normal AV conducting 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 main two 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 the 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 AP conducted the impulses retrograde rapidly from the atrium to the ventricle in a patient with AF, a rapid ventricular response would occur which may result in ventricular fibrillation.

Diagnosis of WPW

WPW pattern

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

WPW syndrome

Diagnosis of WPW syndrome involves the **identification of 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 EKG demonstrating Wolff-Parkinson-White syndrome with characteristic delta waves." by Ksheka.
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The main feature of AV accessory pathway is **pre-excitation**, where the ventricles are activated by 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 (less than 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 the ventricular depolarization is slowed and the QRS complex upstroke is slurred because of slowing the conduction from muscle fiber to another muscle fiber; slow muscle fiber-to-muscle fiber conduction
- **Widening of the QRS complex**

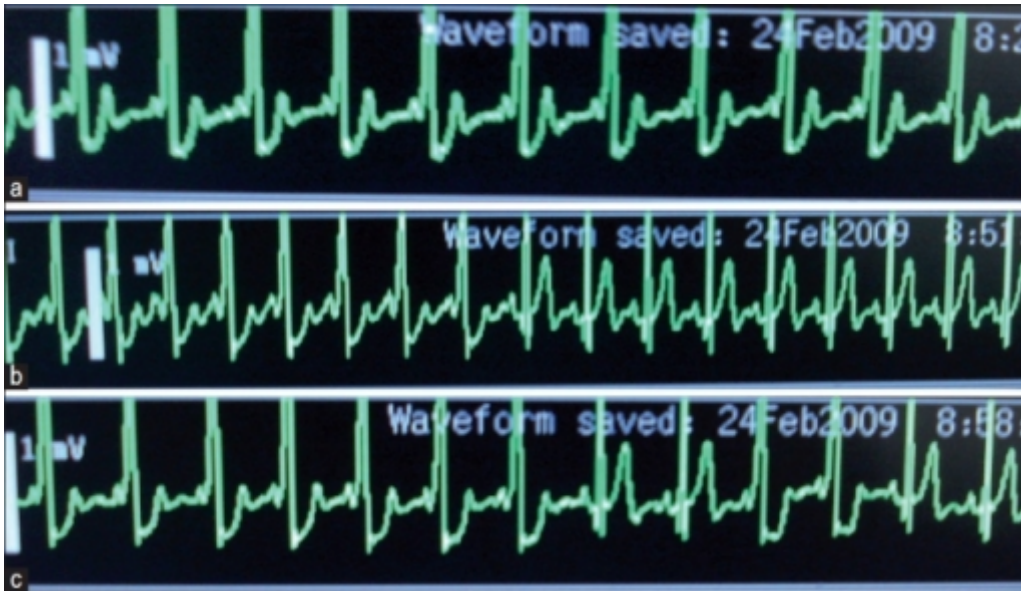


Image: "(a) Sinus rhythm followed by Wolff-Parkinson-White, (b) Sinus rhythm, (c) Wolff-Parkinson-White rhythm" by Openi. License: [CC BY 2.0](https://creativecommons.org/licenses/by/2.0/)

Concealed APS

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

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

Therapy of WPW

Treatment of Wolff-Parkinson-White Syndrome

Patients with Wolff-Parkinson-White 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 doesn't 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 AVNR tachycardia, which aims to alter the 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.
- **Intravenous adenosine then CCBs:** IV Adenosine is the first-line pharmacologic therapy for the termination of tachycardia, followed by IV CCBs such as verapamil.
- **Intravenous procainamide, B-Blockers and digoxin:** They are the second choice treatment to terminate 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 the rapid ventricular response** if the patient manifests with pre-excitation and AF to avoid atrial fibrillation.

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

Note: You should be careful in attempting to slow the AV nodal conduction with digoxin or CCBs; because they may result in accelerating conduction over the accessory pathway (AP) and which may result in AF.

Catheter ablation therapy

WPW: Catheter Ablation of Accessory Pathway

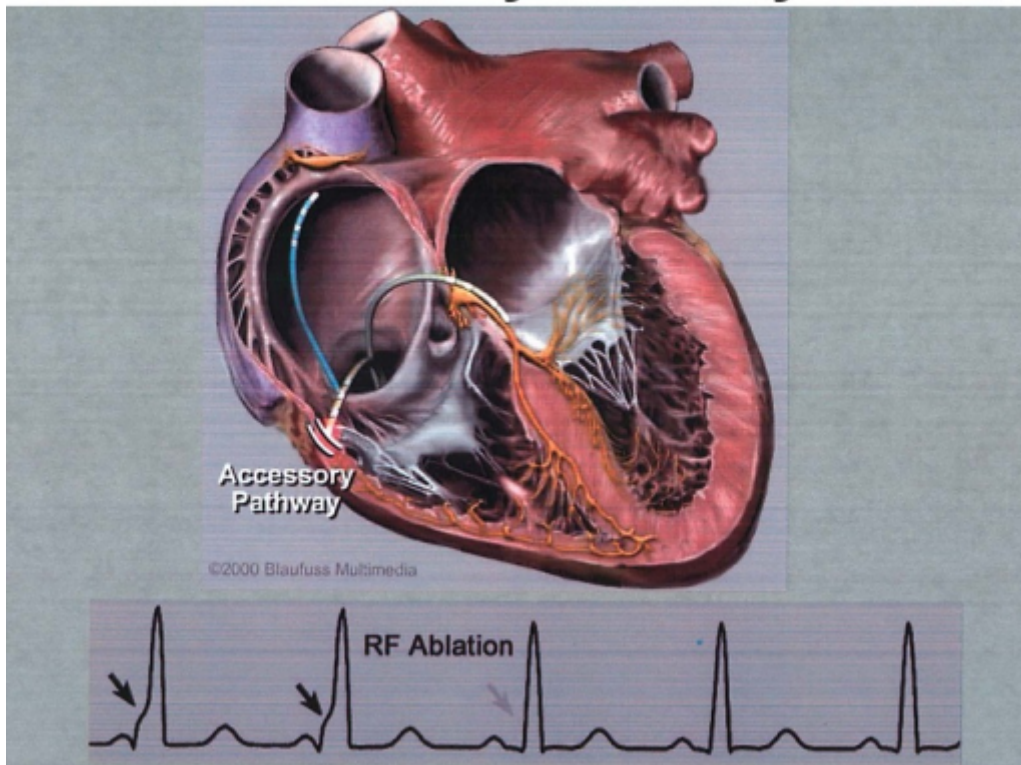


Image: "Schema showing the use of catheter technique for ablation of a right free wall accessory pathway. The rhythm strip shows the disappearance of the delta wave with the application of radiofrequency energy." by Openi. License: [CC BY 3.0](https://creativecommons.org/licenses/by/3.0/)

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

1. Recurrent symptomatic SVT episodes
2. Incessant SVT
3. Heart rates > 200 beats/min with SVT

Prevention of WPW

Prevention of recurrent supraventricular reentrant tachycardias that are associated with accessory pathways (APs) can be done by chronic oral administration of **B-blockers and/or CCBs (verapamil or diltiazem)**.

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

Review Questions

The correct answers can be found below the references.

1. A 75-year-old male patient presented with palpitations and pre-syncope several times. On examination, the patient looks well, lying flat, chest is clear and there is no lower limb edema. EXG shows AF with rapid ventricular

response and WPW syndrome with pre-excitation. The most appropriate drug to control his heart rate is which one?

- A. Procainamide
- B. B-Blockers
- C. Calcium channel blockers (CCBs)
- D. Digoxin

2. The characteristic ECG findings of WPW syndrome are...

- A. ...rapid AF with irregular rhythm.
- B. ...early hyperpolarization.
- C. ...long PR interval with narrow QRS complex.
- D. ...short PR interval with wide QRS complexes with a slurred upstroke - 'delta wave.'

3. A 50-year-old male patient presented to the ER with AF with pre-excitation and the patient is hypotensive and hemodynamically unstable. What is the most appropriate next step to control his symptoms?

- A. DC cardioversion
- B. Procainamide
- C. Sotalol
- D. Adenosine

References

Wolff L, Parkinson J, White PD. Bundle-branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia. 1930. *Ann Noninvasive Electrocardiol* 2006; 11:340.

Miller JM. Therapy of Wolff-Parkinson-White syndrome and concealed bypass tracts: Part I. *J Cardiovasc Electrophysiol* 1996; 7:85.

Kuck KH, Friday KJ, Kunze KP, et al. Sites of conduction block in accessory atrioventricular pathways. Basis for concealed accessory pathways. *Circulation* 1990; 82:407.

Colavita PG, Packer DL, Pressley JC, et al. Frequency, diagnosis and clinical characteristics of patients with multiple accessory atrioventricular pathways. *Am J Cardiol* 1987; 59:601.

Zachariah JP, Walsh EP, Triedman JK, et al. Multiple accessory pathways in the young: the impact of structural heart disease. *Am Heart J* 2013; 165:87.

Vidaillet HJ Jr, Pressley JC, Henke E, et al. Familial occurrence of accessory atrioventricular pathways (preexcitation syndrome). *N Engl J Med* 1987; 317:65.

Milstein S, Sharma AD, Guiraudon GM, Klein GJ. An algorithm for the electrocardiographic localization of accessory pathways in the Wolff-Parkinson-White syndrome. *Pacing Clin Electrophysiol* 1987; 10:555.

Fitzpatrick AP, Gonzales RP, Lesh MD, et al. New algorithm for the localization of accessory atrioventricular connections using a baseline electrocardiogram. *J Am Coll Cardiol* 1994; 23:107.

Correct answers: 1A, 2D, 3A

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Notes