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## **From ECG to Ablation: A Modern Review of Wolff-Parkinson-White Syndrome**

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## **Abstract**

### **Introduction and purpose**

Wolff-Parkinson-White (WPW) syndrome is a clinical manifestation of pre-excitation syndrome, characterized by the congenital presence of an additional conduction pathway in the form of a muscle fibre - Kent's bundle between the atria and ventricles, resulting in paroxysmal supraventricular tachycardia and increasing the risk of sudden cardiac death when atrial fibrillation occurs.

The aim of this study is to show a structured knowledge of the pathophysiology and epidemiology, clinical manifestations, diagnosis, risk assessment and management of Wolff-Parkinson-White syndrome based on a review of the literature, Pubmed and Google Scholar databases.

### **Description of state of knowledge**

Based on current data, the prevalence of electrographic features of pre-excitation is estimated at 0.15-0.25% in the general population. The presence of an additional conduction pathway between the ventricles, predominantly conducting impulses like healthy His and Purkinje fibres, results in a characteristic ECG pattern, allowing the diagnosis of pre-excitation syndrome. Each excitation from sinoatrial node is conducted not only by the physiological pathway, but also by an accessory pathway, the clinical consequences of which will be the occurrence of supraventricular tachycardias such as an orthodromic and antidromic atrioventricular reentrant tachycardia (AVRT), atrial fibrillation or even the ventricular fibrillation, which can be the first symptom of the syndrome. The diagnosis is aided by electrophysiology studies (EPS) to confirm the presence of an accessory pathway, with usually simultaneous invasive treatment - percutaneous ablation, which is also a causal therapy with an efficacy of approximately 92%. Pharmacological treatment of patients with WPW is based on group IA and IC antiarrhythmic drugs, but these act only for symptoms, they are ineffective in eliminating the underlying cause of disease.

### **Conclusions**

The development of cardiology and electrophysiology, as well as the possibility of applying ablation in patients with Wolff-Parkinson-White syndrome, has made this condition curable. Despite the generally low risk of sudden cardiac death (SCD), it is important to properly assess the factors that may contribute to ventricular fibrillation.

**Key words:** Wolff-Parkinson-White syndrome, sudden cardiac arrest, sudden cardiac death, accessory pathway, AVRT, radiofrequency ablation, pre-excitation

## **1. Introduction**

Wolff-Parkinson-White (WPW) syndrome is rare, but important condition that affects the electrical system of the human heart, leading to episodes of arrhythmias. It occurs when an extra electrical pathway, known as an accessory pathway (AP), forms in early stage of embryological differentiation between the heart's upper and lower chambers. This abnormal pathway can cause arrhythmias, leading to symptoms like palpitations, dizziness, and even fainting. While WPW syndrome can be diagnosed in individuals of all ages, as a congenital abnormality, it is often identified in young, otherwise healthy individuals. Though it is generally manageable with treatment, if left untreated, it can increase the risk of more severe complications, including sudden cardiac arrest. In this article, we will explore the causes, symptoms, diagnosis, and treatment options for Wolff-Parkinson-White syndrome, shedding light on this intriguing cardiac disorder and how it can be effectively managed.

## **2. Definitions and epidemiology**

WPW syndrome is a main type of pre-excitation syndrome, a congenital abnormality characterized by the presence of at least one muscular bundle that allows electrical activation of part of the ventricle outside the normal conduction system, bypassing it [1,5]. This muscular bundle is a so-called Kent's bundle. The terms "WPW pattern" and "WPW syndrome" are commonly used to describe patients with ventricular pre-excitation, but they do not denote equivalent concepts. The "WPW pattern" refers to the ECG findings that show ventricular pre-excitation (shortening of PR interval and the presence of delta wave), while "WPW syndrome" refers to the combination of these ECG findings along with symptoms that suggest arrhythmia related to the pre-excitation, such as palpitations, occasional dizziness, near-fainting, syncope, or even cardiac arrest [7]. The prevalence of WPW pattern is 0,15% - 0,25%, moreover, it is estimated that one-third of these individuals will experience arrhythmias over a 10-year period of follow-up [8,9,10,11]. It is indispensable to acknowledge the potential for an increased incidence of APs, attributable to the existence of latent accessory pathways. This term refers to an AP that is undetectable or only minimally visible, primarily due to its location or because of fast conduction through the atrioventricular node (AVN). Concealed AP refers to an AP that conducts only retrograde [6].

Wolff-Parkinson-White syndrome is diagnosed 1,5 - 2 times more frequently in men than in women, though this gender disparity is not seen in children [1,3]. Brugada et al. [6] highlight

that multiple pathways are found in fewer than 12% of adult patients with pre-excitation, whereas in less than 9% of the pediatric population [12]. Additionally, multiple APs occur in patients with structural heart disease, around 50% in patients with Ebstein's anomaly [13].

In the majority of cases, WPW syndrome is observed in structurally normal hearts, but there is an association with some congenital heart disease such as Ebstein's anomaly, mitral valve prolapse syndrome, hypertrophic cardiomyopathy (HCM), aortic stenosis or transposition of the great arteries [14]. Uncommon inherited forms of pre-excitation, which are linked to left ventricular hypertrophy and multisystem disorders, have also been reported. These include mutations in the PRKAG2 gene (encoding the gamma 2 subunit of AMP-activated protein kinase), as well as conditions like Danon disease and Fabry disease, among others [6].

### **3. Pathophysiology**

The accessory atrioventricular (AV) conduction pathway arises during fibrous ring development as a congenital anomaly. From a histological perspective, the cells of the APs are typical working myocardium, with connexin43 gap-junction distribution common for such cells [1,15], which, unlike AVN, usually transmit fast, with no-delay [7], likewise Purkinje fibers. Approximately 8% of the accessory pathways present decremental conduction. Those bypass tracts frequently occur in the posteroseptal region and conduct only retrograde [1].

While most APs conduct impulses in both the anterograde and retrograde directions, some transmit signals in only one direction. Pathways that conduct exclusively in the anterograde direction are rare ( $\leq 10\%$ ), while those that only conduct retrogradely (concealed APs) are more common ( $\leq 50\%$ ) [6]. The last-mentioned manifests only with AVRT and on resting ECG pre-excitation pattern cannot be detected, hence, their true occurrence is unidentified [29].

The majority of APs are located on the left side, regardless of demographic factors. About 60% are situated along the mitral valve and are called left free wall APs. Around 25% are located at the septal side of the mitral or tricuspid annulus, while approximately 15% are found along the right free wall [6]. However, Jamal SZ. et al. described a significant association between the gender of the patient and the localization of accessory pathway. Left anterior (LA) and left lateral (LL) were found in 48% of the males, followed by right posteroseptal (RPS) at 20.4%. Whereas, RPS was found in 33.9% of the females, followed by LL and LA pathways in 32.2%. The least common pathway amongst both genders is the

mid-septum pathway, while males are generally predominant in patients with this particular pathway [16].

#### **4. Clinical symptoms**

Patients with a WPW pattern who have not experienced any arrhythmias will typically be asymptomatic, meaning their medical history and physical examination will generally appear normal. The pattern may have been identified on a previous ECG, and the patient might be aware of their condition. However, some accessory pathway conduction can be transient or hidden, which could result in previous ECGs being normal or intermittently normal [17].

The clinical presentation of WPW syndrome is typically non-specific, highly variable, depending on AP location and conduction properties and, crucially, is often associated with arrhythmic episodes [3]. Patients with a WPW syndrome, who develop a tachyarrhythmia, commonly experience symptoms related to the arrhythmia, such as palpitations, chest pain, shortness of breath, dizziness, lightheadedness, presyncope, and most worrisome: syncope, collapse, and in even sudden cardiac death [17], which may be the first symptom of WPW syndrome.

The main tachyarrhythmias found in populations with Wolff-Parkinson-White syndrome are atrioventricular re-entrant tachycardia (AVRT), categorized into orthodromic and antidromic conduction, and atrial fibrillation (AF). Others, such as atrial tachycardia, atrial flutter or atrioventricular nodal reciprocating tachycardia (AVNRT) are infrequent, where AP do not play an important role in the mechanism of re-entry [6].

Orthodromic AVRT stands for 90-95% of re-entrant tachycardia among patients with AP, which involves a circuit that first conducts in the anterograde direction through the AV node and His-Purkinje system, followed by conduction through the ventricle. This is followed by retrograde conduction through the accessory pathway (AP), completing the circuit by returning through the atrium to the AV node [5]. Orthodromic AVRT is typically a rapid tachycardia, with heart rates usually ranging from 150 to, in rare cases, over 220 beats per minute. Throughout the circuit, ECG displays narrow QRS (specifically, when patient presents pre-existed or rate-related bundle branch block), RP interval constant and, usually but not invariably, up to one-half of the tachycardia cycle length ST segment depression [5,6]. On the other hand, antidromic AVRT appears in 3-8% of patients [18,19,20]. In contrary to orthodromic AVRT, this tachyarrhythmia transmits anterograde from the atrium through the AP, to the ventricle, which results in wide QRS. The retrograde limb are Purkinje cells and His bundle or another AP, conducting back to the AVN. In 30-60% of patients with

spontaneous antidromic AVRT, multiple accessory pathways (either manifest or concealed), which may or may not function as the retrograde limb during the AVRT, can be identified [6]. Paroxysmal AF occurs in up to 38-50% of patients with WPW. While atrial fibrillation can occasionally originate on its own, it is more commonly caused by elevated atrial pressure resulting from decreased ventricular filling time, which is triggered by oAVRT. Manifest APs with rapid anterograde conduction and a short refractory period may lead to life-threatening ventricular fibrillation (VF) and SCD [3,6,21].

## **5. Diagnostics**

The resting ECG in terms of WPW pattern have main typical features: shortening of PR interval ( $\leq 120\text{ms}$ ), prolonged QRS duration ( $>120\text{ms}$ ) and the slurred upstroke (or downstroke) of QRS complex (a delta wave). The lack of this pattern does not exclude the possibility of an accessory pathway, as some pathways can conduct impulses only under specific conditions or in a retrograde direction [17]. Pre-excitation on the surface ECG can be temporary and may even resolve permanently in up to 35% of cases over time. Additionally, the extent of pre-excitation can vary depending on the location of the AP and the conduction properties of the AVN. [1,6]. The ECG in WPW syndrome can be deceptive and may result in the incorrect diagnosis of various other ECG-clinical conditions [22-24]. When the interpretation of ECG causes difficulties, the examination can be repeated after administration intravenous 6 mg of adenosine, which will block the AVN node and the AP will be revealed [1].

The direction of the delta wave and the shape of QRS have become the basis of assessment of localization of AP [1]. Various algorithms, including those by Pambrum, Arruda, Chiang, St George or Milstein have been employed to predict the location of the accessory pathway, utilizing different electrocardiographic criteria derived from delta wave analysis. Some can be applied for either children and adults, whereas others exclusively for a specific age group. One of the most recent studies, from El Hamriti M. et al., developed an algorithm, EASY-WPW, which applied to the overall study population outperformed Pambrun or Arruda systems in accuracy [3, 25].

## **6. Risk assessment**

The non-decremental nature of conduction is a key factor in the potential for sudden death in individuals with WPW syndrome. Due to the possibility of an absent delay in conduction between the atria and ventricles, atrial fibrillation—where atrial rates can reach up to  $>350$

beats per minute—may result in rapid ventricular depolarization. This can lead to dangerously high ventricular rates, which have the potential to progress into ventricular fibrillation (VF) [7]. Moreover, despite being quite rare and unusual, sudden death may be the first manifestation of pre-excitation syndrome, particularly in the children and young adults population [26].

The risk of cardiac arrest in asymptomatic patients with WPW is very low, and the risk of death is even lower, with the rate of cardiac arrest ranging from 0.85 to 1.5 per 1000 patient years [7], though it can not be unacknowledged.

High-risk features that should be estimated on clinical evaluation are: young age (approximately first two decades of life) of detecting WPW pattern, male sex, familial WPW syndrome (PRKAG2 gene mutation), history of symptomatic tachycardia, history of atrial fibrillation, concomitant congenital heart disease, especially Ebstein's anomaly. It needs to be emphasized that the functional properties of the accessory pathways might change in the setting of adrenergic stress as encountered in sporting or other activities during daily life, so risk factors such as a profession of high public responsibility should be taken into consideration [5,27].

All patients with WPW syndrome should undergo risk stratification due to the potential risk of sudden death. This can be achieved through both invasive and non-invasive methods. The aim is to evaluate the anterograde refractory period of the accessory pathway, which serves as a surrogate marker for the conduction rate over the pathway during atrial fibrillation [3,28].

Non-invasive tests involve 12-lead ECG, 24h ECG Holter monitoring and exercise testing or interpretation of ECG after the administration of procainamide, propafenone, or disopyramide [1,3]. The abrupt loss of delta wave and normalization of PR interval during above mentioned methods has been identified as markers of low risk. However, the varying sensitivity of accessory pathways changes in adrenergic stimulation and makes the clinical utility of non-invasive tests questionable [6].

Electrophysiological study (EPS) is an invasive diagnostic procedure used to assess accessory pathways. It allows to verify the presence of accessory pathways, their number, location, conduction properties, descending and retrograde refractoriness, to induce tachyarrhythmias and is commonly an introduction to the catheter ablation [1].

The key measures evaluated during EPS at baseline and amid catecholamine infusion are the shortest pre-excited RR interval in atrial fibrillation (SPERRI), the accessory pathway effective refractory period (APERP), and the shortest pre-excited paced cycle length during atrial pacing (SPPCL). Above mentioned parameters aim to determine the anterograde



conduction over the APs and therefore, the risk of VF. It is also crucial to assess inducibility (and, ideally, hemodynamic tolerance) of sustained arrhythmias such as orthodromic atrioventricular reentrant tachycardia (oAVRT), atrioventricular reentrant tachycardia (aVRT), and atrial fibrillation (AF), as these are common triggers for VF or sudden cardiac arrest (SCA) [3].

SPERRI  $\leq 250$ ms at baseline and AP ERP  $< 240$  ms are considered to be high-risk electrophysiological factors of SCD [30, 31, 32]. The rationale of performing an EP study in pre-excitation are symptoms to risk-stratify for life-threatening arrhythmias events and asymptomatic population of competitive athletes or with high risk occupations and findings suggesting an increased chance of sudden arrhythmic events on non-invasive testing [27].

## 7. Treatment

The management of patients with WPW syndrome differs depending on the presence of symptoms. Acute therapy of tachyarrhythmias should be concentrated on ceasing the recurrent conduction over the AV node and APs. In AVRT, vagal maneuvers, including Valsalva and carotid sinus massage, should be a first-choice procedure to intermit the supraventricular tachycardia since they can be done quickly. However, in hemodynamically unstable patients or when drug therapy is ineffective, synchronized electric cardioversion should be performed. Adenosine should be administered carefully when treating AVRT, due to the risk of triggering fast atrial fibrillation, which could be conducted over the anterograde AP to the rapid ventricular rate and further to the VF. Pharmacological therapy includes drugs acting on the AVN, such as beta-blockers, diltiazem or verapamil or on the AP - antiarrhythmic drugs - ibutilide, procainamide, propafenone, or flecainide, which are preferred in antidromic AVRT. Additionally, in the case of treatment-resistant antidromic AVRT, amiodarone may be considered [6].

In pre-excited atrial fibrillation (AF), electric cardioversion is a primary approach, especially in hemodynamically unstable patients. Administration of ibutilide, propafenone, procainamide or flecainide could also be effective in restoring the sinus rhythm. However, it is important to emphasize that IC (flecainide, propafenone) drug agents are not without risk, as they may impact the AV node. Intravenous digoxin, intravenous amiodarone, intravenous or oral beta blockers, diltiazem, and verapamil are potentially harmful for treatment in patients with pre-excited AF [3, 6, 27, 34].

Catheter ablation in WPW syndrome turned out as a breakthrough in its management, providing a high success rate of  $>94\%$ , with a recurrence rate of  $6.2\%$  and a complication rate

of 1% [35], emerging as the preferred method that is potentially accessible to all WPW patients. The method is stated to be first-line therapy for patients with symptomatic and recurrent AVRT or pre-excited AF. Borregaard et al. evaluated that the patients with radiofrequency ablation(RFA)-treated WPW syndrome have a post-procedure mortality comparable with the background population, also emphasized that RFA do not seem to decrease the incidence of AF after the procedure [2]. Ablation of APs in septal or midseptal areas or near coronary sinus are related with lower success rate, respectively 50%, 73%, 50% [35, 36]. Serious complications, such as third-degree atrioventricular block (0,17 - 2,7%) or cardiac tamponade (0,13 - 1,1%) were associated with ablation of AP localized in the intraventricular septum [38]. Marazzato J et al. achieved a lower frequency of AV block while using cryoenergy during ablation [39], however, the recurrence of previously blocked pathways has been found to be considerably greater when cryoenergy is applied [40].

The most challenging for cardiologists is the management of individuals with only the WPW pattern. For patients with asymptomatic and infrequent episodes, treatment decisions should weigh the overall risks and benefits of the invasive nature of ablation against the long-term commitment to pharmacological therapy. Recent guidelines suggest that invasive screening using an EPS is now advised for individuals in high-risk occupations or competitive athletes, and it should also be considered for other patients. The catheter ablation in asymptomatic patients is recommended when during EPS high-risk factor for SCD such as SPERRI  $\leq 250$ ms, AP ERP  $\leq 250$ ms, multiples APs and inducibility of AVRT are identified. In this population, it is important to discuss the risks and benefits of the procedure, especially of heart block associated with ablation of septal APs [6]. Telishevska M. et al. reached a 91% success rate during ablation in 182 asymptomatic patients with WPW pattern, both children and adults, without major complications [41]. In children under 12 years old, the likelihood of a fatal event appears to be low, so a more cautious approach is typically recommended [42], however the findings of Pappone C. et al. registry from 2004 should reassure physicians and parents alike that in asymptomatic children with the Wolff–Parkinson–White syndrome who are at high risk for arrhythmias, ablation is an appropriate option [43].

Symptomatic patients with WPW syndrome, for whom ablation is not indicated or who have not consented to invasive treatment, may use antiarrhythmic drugs from group Ic to inhibit orthodromic AVRT, unless ischemic or structural heart disease has been excluded. If no pre-excitation is observed on resting ECG, the addition of a beta-blocker, verapamil, or digoxin should be considered [6]. It is important to note that persistent AVRT involving an accessory pathway can lead to tachyarrhythmic cardiomyopathy and heart failure [1].

## 8. Conclusion

Wolff-Parkinson-White syndrome, while a relatively rare condition, presents significant challenges in both diagnosis and management due to its potential for life-threatening arrhythmias, such as atrioventricular reentrant tachycardia (AVRT) and atrial fibrillation (AF), which can lead to sudden cardiac death. Advances in electrophysiology and the development of catheter ablation have provided a curative treatment option, significantly improving patient outcomes. Early identification through electrocardiographic (ECG) findings and electrophysiological studies is crucial for risk stratification and timely intervention. Although the risk of sudden cardiac death in asymptomatic individuals is low, those with high-risk features such as young age, symptomatic arrhythmias, or concomitant structural heart disease, should be closely monitored and appropriately treated. Radiofrequency ablation has emerged as the first-line therapy for symptomatic patients, offering a high success rate and low complication rate. As the understanding of WPW syndrome continues to evolve, personalized treatment plans, including careful consideration of ablation for asymptomatic patients with high-risk features, are key to optimizing patient care. Ultimately, proper risk assessment, timely intervention, and personalized management strategies can significantly reduce the risk of serious complications associated with this syndrome, improving both quality of life and long-term outcomes for affected individuals.

## Authors contributions

Conceptualization - Agata Krupa and Michał Pałuchowski; methodology - Jan Mateńko and Wiktor Możarowski, software - Julia Kielbratowska; check - Maciej Mozer, Anna Krawczyk and Przemysław Klasicki; formal analysis - Wiktoria Pietruszka and Maria Potrykus; investigation - Agata Krupa, resources - Wiktoria Pietruszka; data curation - Maciej Mozer; writing (rough preparation) - Michał Pałuchowski and Julia Kielbratowska; writing (review and editing) - Przemysław Klasicki and Wiktor Możarowski; visualization - Jan Mateńko, Wiktoria Pietruszka, Maria Potrykus; supervision - Anna Krawczyk; project administration - Agata Krupa.

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### **Conflict of Interest Statement**

All authors declare that they have no conflicts of interest.

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