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An Overview on Wolff-Parkinson-White Syndrome

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Abstract:

Wolff-Parkinson-White (WPW) syndrome is a congenital cardiac condition characterized by the presence of an accessory conduction pathway (known as the Bundle of Kent) that connects the atria and ventricles, bypassing the atrioventricular (AV) node. This abnormal pathway can lead to atrioventricular reentrant tachycardia (AVRT) and, less commonly, atrial fibrillation with rapid ventricular response. WPW is typically identified on electrocardiogram (ECG) by three hallmark features: Short PR interval (<120 ms), Delta wave (slurred upstroke of the QRS complex) and Widened QRS complex (>110 ms). Although many patients remain asymptomatic, symptomatic cases may present with palpitations, dizziness, syncope, or sudden cardiac arrest (rare).Management may include vagal maneuvers, antiarrhythmic drugs (procainamide, flecainide, amiodarone in some contexts), catheter ablation of the accessory pathway, and avoidance of AV nodal blocking agents in atrial fibrillation (as these may worsen conduction through the accessory pathway).

Keywords: Wolff-Parkinson-White syndrome (WPW), Atrioventricular reentrant tachycardia (AVRT), Supraventricular tachycardia (SVT), Delta wave.

Introduction:

Wolff-Parkinson-White (WPW) syndrome is typified by the existence of an auxiliary atrioventricular conduction route that makes tachyarrhythmias more likely. The auxiliary pathway, also known as the Bundle of Kent, permits the ventricles to be pre-excited, which may show up as frequent episodes of atrial fibrillation or supraventricular tachycardia. (1) The presence of a short PR interval, a slurred upstroke of the QRS complex called a delta wave, and enlargement of the QRS complex are the electrocardiographic indicators that establish the diagnosis of WPW. Bypassing the typical AV nodal delay, conduction via the auxiliary channel results in early ventricular activation, as evidenced by these data. Clinically, WPW patients can exhibit a broad range of symptoms, from presyncope, dizziness, and palpitations to more severe presentations including syncope and, in rare instances, sudden cardiac death. Additionally, the syndrome could not exhibit any symptoms and only be discovered by chance during standard ECG screening. (3) Over the past few decades, WPW syndrome management techniques have undergone tremendous change. With its high success rate and low complication rate, catheter ablation of the accessory route has become the gold standard for treatment, particularly for patients who are symptomatic or at risk of potentially fatal arrhythmias. (4)

1.Definitions

WPW syndrome is a condition that can lead to atrial/ventricular tachyarrhythmias and potentially sudden cardiac death because it is defined by the existence of at least one accessory pathway (AP). Only patients with symptomatic tachyarrhythmias and preexcitation on the baseline ECG are eligible for this diagnosis. When symptoms are absent, the phrase is preferable **Wolff–Parkinson–White pattern**. **(5)**

2. Anatomy,Nomenclature of Acsesory Pathways

2.1. Anatomy

Accessory pathways (APs) are abnormal muscular bundles that, apart from the normal atrioventricular conduction pathway, link the atrium and the ventricle. They are remains of the embryo because

the atrioventricular (AV) annuli did not fully separate from the ventricles and atria during embryonic development. **(6)**

The tricuspid valve annulus is often discontinuous and less well-formed than the mitral annulus. As the right atrial and right ventricular myocardia intrude onto the tricuspid annulus, they frequently fold or overlap. Since there is typically a large space between the atrial and ventricular myocardium to accommodate the aortic outflow tract, APs are rarely found in the area of fibrous continuity between the aortic and mitral valves, although theoretically they can be located in any spot of the tricuspid valve annulus. (8) Free wall accessory AV connections are those that arise from the ventricle's attachments to the atrial appendage, the Marshall ligament, and the CS musculature. A continuous sleeve of atrial myocardium that stretches 25 to 51 mm from the CS ostium encircles the venous wall of the CS. Although adipose tissue typically separates this muscle from the left atrium, it is proximally contiguous with the right atrial myocardium. Electrical continuity between the left atrium and the CS musculature may be produced via strands of striated muscle that span this gap. (9) It is less typical for the CS musculature to electrically continue with the ventricle. Instead, CS musculature extensions over the middle and posterior cardiac veins may result in ventricular connections. (10) The Marshall vein is carried by the Marshall ligament, a pericardial fold, from its beginning as a branch of the distal CS to its end close to the left superior pulmonary vein. Bundles of muscle fibers that run parallel to the CS musculature may also be present in this arrangement. having close links between the ventricle and the CS muscles. (11) A direct epicardial muscle continuity between the ventricle and the atrial appendage is an additional uncommon type of AV connection. While left-sided connections are even less common, there are numerous accounts of connections between the right atrial appendage and ventricle. (picture) (1) (12)

After left free wall APs, posteroseptal APs are the second most prevalent type of AV connection seen in clinical settings. The posterior septal routes run along the posterior septal portion of the Mitral Annulus, to the roof of the CS, or inside the CS ostium or proximal extent (about 2 cm). A significant subgroup of posteroseptal Aps are epicardial accessory AV connections. (13)

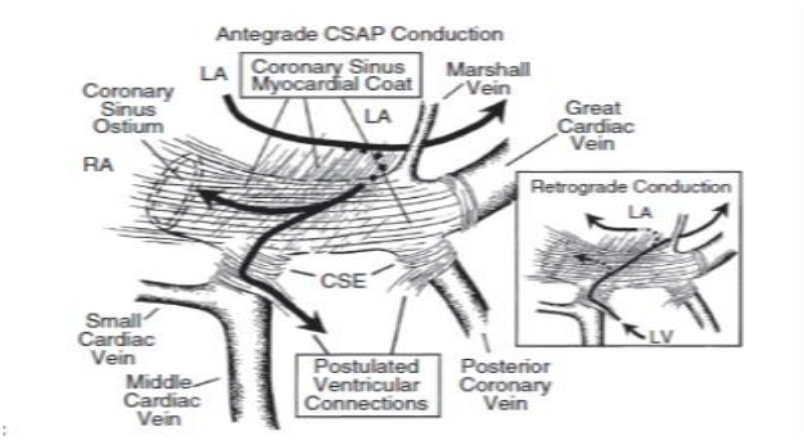

Figure (1) Schematic representation of Epicardial coronary sinus accessory pathway. **(10)**

2.2. Nomenclature

The conventional nomenclature for atrioventricular APs categorizes locations into right parietal, septal, and left parietal pathways based on the AV junctions as seen in the left anterior oblique radiography projection. Right parietal APs are classified as anterior, anterolateral, lateral, posterolateral, and posterior Aps based on where they are located. Septal APs are typically classified as anteroseptal, midseptal, and posteroseptal based on their position. (Fig. 2) However, the descriptive terms are not anatomically correct. (14) The apex of Koch's triangle was thought to contain anteroseptal pathways, which connect the ventricular and atrial septa in the region of the His bundle. These pathways are actually more appropriately referred to as superoparaseptal because the area anterior to the His recording location lacks an atrial septum, where the aortic root separates the atrial walls. (15) These channels, which were formerly referred to as posteroseptal, are not actually septal in position. Furthermore, this area can be regarded as inferior rather than posterior from a real anatomical standpoint. (16) Although pathways situated between these two septal boundaries have been referred to as

midseptal or intermediate septal, they can simply be referred to as septal since they are the only true septal linkages. (17) In 1999, an expert consensus conference proposed new nomenclature for auxiliary routes that included the precise anatomical locations of the currently used nomenclature. (16)

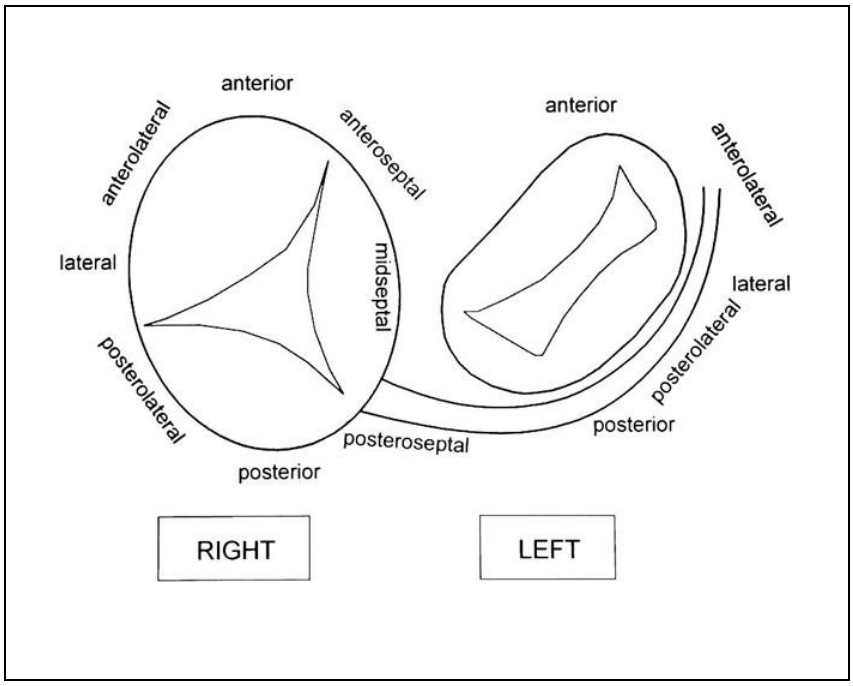

Figure (2): Schematic representation of AV junctions in left anterior oblique projection, showing Traditional nomenclature used to account for the locations of accessory pathways

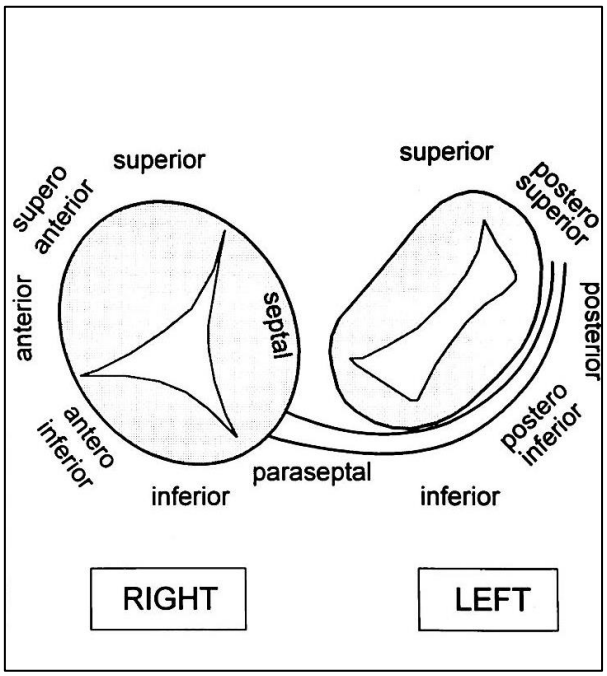

Figure (3): Schematic representation of AV junctions in left anterior oblique view, as shown. An anatomically correct nomenclature is shown for different segments of the junctions. The table shows the correct anatomical location of differ accessory pathways using the new nomenclature.

3. Epidemiology

Wolff–Parkinson–White (WPW) syndrome has a frequency of 0.68–1.7/1000. Transcatheter procedures may result in changes to the epidemiological profile. (18) 0.15% to 0.25% of people have the WPW pattern, and it is estimated that one-third of these individuals will experience arrhythmias throughout the course of a 10-year follow-up. (19)

In most regions of the world, WPW syndrome is the second most frequent cause of paroxysmal supraventricular tachycardia. According to extensive general population research involving both adults and children, the prevalence of WPW syndrome is between one and three out of every 1000 people. (20) The prevalence of life-threatening occurrences, such as sudden cardiac death or arrest, is quite high, ranging from 0.8 to 1.9 per 1000 person-years, particularly in youngsters. (21) While the majority of people with Wolff-Parkinson-White syndrome have normal anatomy, some also have multisystem disorders or congenital cardiac disease. The prevalence of Wolff-Parkinson-White syndrome is about 10% in people with Ebstein's abnormality. Atrial and ventricular septal defectsand corrected transposition of the great vessels are additional congenital cardiac defects that are linked to this condition. (2) WPW may be linked to hypertrophic cardiomyopathy, frequently when certain gene alterations are present. Surprisingly, patients with HCM phenocopies, such as Danon, Fabry, and Pompe disease, have also been found to have Accssory pathways. (22)

Less than 12% of pre-excitation patients experience several APs. They are generally more prevalent in people with structural heart disease, with about 50% of patients having Ebstein's abnormality. (23)

Multiple APs have been linked to increased risk of supraventricular tachycardia, antidromic re-entry, and the possibility of faster conduction during ventricular and atrial fibrillation. Although they could be discovered in any combination of pathway sites, right free wall and posteroseptal pathways were more common (24)

4. Pathophysiology

Only a few number of genes have been found to yet to be putative causes of WPW syndrome. Given the syndrome's inadequate penetration and unknown inheritance patterns in most people, little is known about its genetic basis. (25) Preexcitation is linked to thicker ventricular walls because of increased intracellular glycogen deposition in myocytes in the familial form of WPW, a rare early-onset autosomal dominant disease with complete penetrance and varying degrees of expression caused by mutations of the PRKAG2 gene. (26) Although AF/cardiomyopathy has been associated with specific genes like ANK2, NEBL, PITX2, and PRDM16 in the setting of WPW syndrome, PRKAG2 and MYH7 are known to be associated with both sporadic and familial instances. (27) PRKAG2 is the gene linked to a family variant of WPW syndrome that has been examined the most. (28) Unlike AV nodal conduction system, Accssory pathways usually have fast, nondecremental conduction that is reliant on a sodium current. Anterograde, retrograde, or both types of conduction are possible through the bypass tracts.The relative conduction to the ventricle over the AV node (AVN)—His bundle axis vs the auxiliary pathway—determines the degree of pre-excitation. (2)

Most of Accssory pathways have bidirectional Conduction. Retrograde conduction is more common (~50%) than exclusively anterograde conduction, which is found in just a small percentage (less than 10%). Decremental conduction qualities may be present in a small percentage of overt and hidden routes. (29)

5. Diagnosis

WPW syndrome is only diagnosed in patients who exhibit both pre-excitation and symptoms. Short PR interval (<0.12 s), prolonged QRS complex (>0.12 s), and slurred, slow-rising beginning of the QRS complex (known as a delta wave) are the primary electrocardiographic characteristics of pre-excitation. Depending on the AP's location and the AVN's conduction characteristics, different levels of pre-excitation are feasible. It is possible to estimate the location of the manifest auxiliary pathway based on the surface ECG. These are useful when thinking about an ablation procedure and may be significant for the risk of aberrant conduction-induced cardiomyopathy.

(30) Using various electrocardiographic criteria based on the analysis of the delta wave shape, QRS polarity, or the R/S amplitude ratio of QRS complexes, a number of algorithms (Chern-En Chiang's, Fitzpatrick's, Xie's, St George's, and Padrun's algorithms) have been used to predict the location of accessory pathways. However, those algorithms demonstrated reduced discriminative ability in the case of multiple accessory pathways and structural heart disease. (31) Even in modern practice, the most used algorithm is still the one created by Arruda et al. (32) using the surface ECG, which has an overall sensitivity of 90% and specificity of 99%.

Arruda et al. (32) used this algorithm to divide the AV ring into 15 locations for accessory pathways, 5 septal locations (anteroseptal tricuspid annulus and right anterior paraseptal, mid septal tricuspid annulus, posteroseptal tricuspid annulus, posteroseptal mitral annulus, and subepicardial posteroseptal accessory pathway), 5 right free wall locations (right anterior, right anterolateral, right lateral, right posterolateral, and right posterior), and 4 left free wall locations (left anterolateral, left lateral, left posterolateral, and left posterior) (figure (4)).

There are four steps in the algorithm.

Step 1: Examine the R/S ratio in V1 or the delta wave in lead I. A left free wall accessory pathway is present if the delta wave is negative or isoelectric, or if the V1R/S ratio is greater than 1. Lead AVF delta is checked in this situation; if it is positive, the accessory route is either left lateral or left anterolateral. The AP is either left posterolateral or left posterior if it is negative or isoelectric. The accessory pathway is either the right free wall or the septum if the first step requirements are not met. In this scenario, move on to step 2.

Step 2: Lead II's delta wave analysis. If the result is negative, the subpericardial posteroseptal auxiliary route is immediately identified. Go to step 3 if "positive" or "isoelectric."

Step 3: Lead V1's delta wave analysis. Examine the delta wave in lead AVF if the auxiliary channel is septal and the delta wave is negative or isoelectric. The AP is at the ostium of the coronary sinus or the posteroseptal tricuspid annulus if the delta wave in the AVF is negative. The AP is either the posteroseptal mitral annulus or the posteroseptal tricuspid annulus if it is isoelectric. To distinguish between anteroseptal/right anterior paraseptal and midseptal AP, the lead III R/S ratio is utilized if the lead AVF has a positive delta. In lead lead III, AP is antroseptal or right anterior paraseptal if $R \geq S$, while midseptal AP is identified if $R<S$.

After excluding the left free wall accessory pathway in step 1, if the delta wave in lead V1 is positive, the accessory pathway is the right free wall, therefore move on to step 4.

Step Four: If the delta wave in AVF is positive, it indicates right anterior/anterolateral AP in patients with right free wall. A positive delta in lead II indicates the right lateral accessory pathway, while an isoelectric delta in lead II indicates the right posterior/posterolateral accessory pathway, if the delta in AVF is negative.

According to Arruda et al., this algorithm offers a 99% specificity and 90% overall sensitivity. The method was particularly useful in accurately identifying auxiliary pathways that required ablation from within the coronary sinus (sensitivity 100%, specificity 100%) and anteroseptal (sensitivity 75%, specificity 99%) and mid-septal (sensitivity 100%, specificity 98%) pathways.

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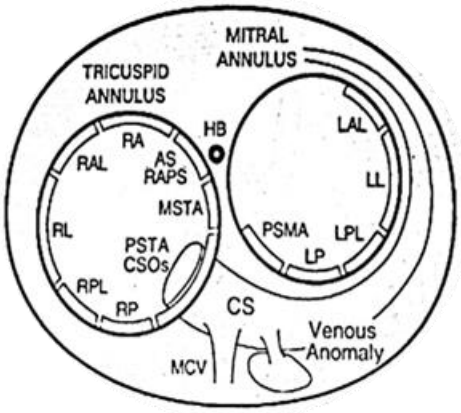

Figure (4): The possible sites of AP as seen in left anterior oblique projection.

AS=anteroseptal, CS =coronary sinus, CSOs =coronary sinus Os, LAL=left anterolateral, LL=left lateral, LPL=left posterolateral, LP= left posterior,, MCV=middle annulus cardiac vein, MSTA=midseptal tricuspid annulus PSMA=posteroseptal mitral annulus cardiac vein, PSTA=posteroseptal tricuspid annulus, RA=right anterior, RAL=right anterolateral, RAPS=right anteroparaseptal, RL= right lateral, RP= right posterior, RPL=right posterolateral

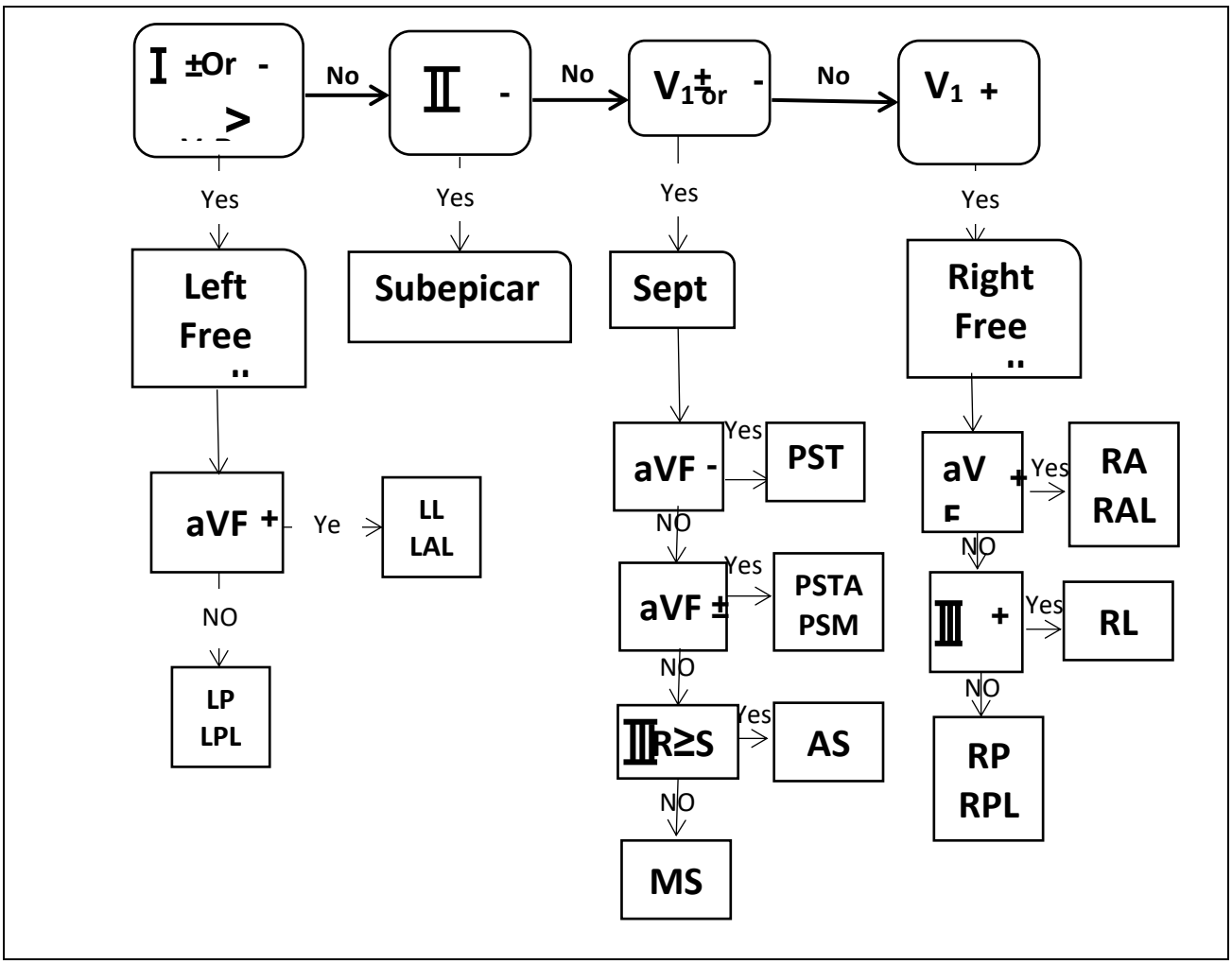

Figure (5) : Diagrammatic illustration of Arruda's algorithm. Same nomenclature as inPrevious figure

However, compared to more traditional algorithms (84 percent with Pambrun and 75 percent with Arruda), more recently created algorithms appear to have higher accuracy (93 percent), which was also verified in children. (33)

WPWsyndrome's clinical manifestation is typically nonspecific, highly variable, and—most importantly—usually occurs in conjunction with arrhythmias. 90% of kids, roughly 65% of teenagers, and 40% of adults over 30 with a WPW pattern on their resting ECG are thought to be fully asymptomatic (34)

However, as anterograde conduction over APs can only be concealed by augmented AVN conduction, this should be interpreted cautiously, particularly in children and adolescents. This could result in patients having a borderline or short PR interval with a relatively narrow QRS complex. (35)

The emergence of symptoms varies with age and may be influenced by the location and conduction characteristics of accessory pathways.Since its existence is linked to decreased exercise capacity in children as well (owing to APs-induced dysynchrony and LV dysfunction), the concept of asymptomatic overt pre-excitation is currently under scrutiny. This could help to explain why adults with overt pre-excitation have a higher risk of heart failure. (36)

Orthodromic atrioventricular reentrant tachycardia (O-AVRT) and atrial fibrillation are the two primary tachyarrhythmias that can occur in symptomatic WPW patients. O-AVRT is the most prevalent and, crucially, benign arrhythmia in people with WPW syndrome; it accounts for 90–95% of reentrant tachycardias inthose patients. (37) Only 3–8% of WPW patients have been reported to have antidromic AVRT (A-AVRT); nevertheless, in 30–60% of A-AVRT instances, several APs—whether visible or hidden—are seen, which may or may not function as the retrograde limb during an arrhythmia. Patients with atrioventricular nodal reciprocating tachycardia (AVNRT), atrial tachycardia, or atrial flutter may also have other pre-excited tachycardias in which the accessory route is functioning as an innocent bystander. (2) In patients with WPW syndrome, atrial fibrillation has a specific prognostic importance. If ventricular fibrillation develops from an abnormally fast ventricular response, it could be fatal. (38).

Among patients with WPW syndrome, the overall incidence of AF ranges from 12% to 39%. Usually, AF is paroxysmal. Rarely do these people have persistent AF. Given the low prevalence of concurrent structural heart disease or other AF contributing conditions, the occurrence of intermittent AF in patients with the WPW syndrome is remarkable. The fact that AF may not return after ablating the AV BT supports this observation, which implies that the AV BT itself may be connected to the development of AF. (39) It has been discovered that in individuals with WPW syndrome, the coexistence of a functional retrograde AP and prolonged episodes of AVRT significantly contributes to the onset of AF. There is convincing evidence that AF can be triggered by retrograde conduction to the atrium via multiple AP or multifiber AP during AVRT. (40) Fujimura et al. (41) noted that there were no appreciable variations in retrograde characteristics and that the anterograde AP effective refractory period (ERP) was shorter in the AF group than in the control group.

The induction of AF was likewise associated with the AP's placement. Inducible arrhythmia was shown to occur often in patients with an anteroseptal AP (62%). Inducible arrhythmia was rather uncommon in patients with a right free wall AP (21%). Induction rates were 36% in patients with posteroseptal AP and 44% in patients with left free wall AP. (42) Because of the effects of conduction delay and unidirectional block, structural heterogeneities are known to be significant in atrial reentry. Therefore, the increased structural heterogeneity brought about by the AP may contribute to the development and maintenance of atrial reentry in patients with WPW syndrome. (43) Although AP ablation is a curative treatment for the majority of WPW patients, trends of elevated AF risk in ablated WPW patients point to mechanisms of AF other than those directly associated with BT existence. When an underlying atrial illness is present in some patients, the AP is seen as an innocent bystander. (42) In addition, electrophysiological findings like increased intra-atrial conduction delay, increased induction of repetitive atrial firing, and altered atrial refractoriness point to an intrinsic atrial vulnerability as the mechanism of PAF in some WPW syndrome patients. (44)

However, it appears that if ablation is done before the age of 50, the long-term AF risk linked to WPW is greatly decreased (45).

6. Risk Stratification

WPW is a well-known cause of sudden cardiac death, and up to 33% of patients in previous research who were young survivors of sudden cardiac arrest and did not have structural heart disease had the syndrome. (46).

Though it accounts for as little as 3.6 per 10 million person-years ,more recent data revealed that WPW syndrome is indeed an uncommon cause of SCD, especially in children and the young. (47)

Patients with no symptoms typically have a reduced incidence of SCD. However, in patients with undiagnosed and/or asymptomatic pre-excitation syndrome, unexpected death could be the initial sign. (48).

6.1. Clinical Evaluation

Male sex, familial WPW syndrome (autosomal dominant, chromosome 7, PRKAG2 gene mutation), WPW pattern found within the first 20 years of life, history of atrial fibrillation and arrhythmic symptoms such as syncope, and congenital heart disease, particularly Ebstein's anomaly, are high-risk characteristics according to clinical evaluation. Additionally, priority should be given to high-risk professions like piloting, bus driving, and athletics. (20) Due to the possibility of unexpected mortality, all patients with WPW syndrome must go through risk stratification. Both invasive and noninvasive methods can be used for this. The objective is to evaluate the accessory route's anterograde refractory period, which serves as a proxy for the rate of conduction across the pathway during atrial fibrillation. (49)

6.2. Noninvasive Assessment

A lower-risk subgroup was identified based on the elimination of pre-excitation during exercise testing or following the administration of procainamide or ajmaline (50).

A low risk of SCD has been linked to intermittent pre-excitation; nevertheless, new evidence showed that at least 13% of these individuals still have high-risk APs. (51)

Furthermore, patients with intermittent vs. persistent pre-excitation do not appear to vary clinically, demographically, or electrophysiologically (including the prevalence of high-risk feature APs following isoproterenol testing). (52)

5.3. Invasive Assessment

As suggested by noninvasive tests, electrophysiological testing is advised for asymptomatic people with high-risk occupations and/or high-risk characteristics, as well as for symptomatic patients to clarify the pathophysiological basis of their arrhythmias. (53)

Previously thought to be extremely uncommon in asymptomatic individuals, more recent multicenter data revealed that the risk of SCD/SCA is considerably higher, even though it is still minor (54)

Despite its flaws, electrophysiologic testing is thought to have a very good negative predictive value in asymptomatic people, particularly when done without general anesthesia and with catecholamine infusion. (55)

The parameters that estimate the anterograde conduction over the APs (and thus the risk for VF) are the most crucial to ascertain during baseline and during catecholamine infusion electrophysiologic testing. These parameters include the accessory pathway effective refractory period (APERP), the shortest pre-excited paced cycle length during atrial pacing (SPPCL), and the shortest pre-excited RR interval in atrial fibrillation (SPERRI). Since AVRT and AF are the typical initiating causes of VF/SCA, inducibility (and ideally hemodynamic tolerance) for persistent arrhythmias is particularly crucial. Future arrhythmic events were also predicted by the presence of various auxiliary routes, including potential septal ones (56).

The best way to identify those who are at risk of VF is to measure their shortest pre-excited RR interval (SPERRI) during AF. Thus, when the shortest pre-excited RR interval is less than 250 ms in the control state in adults, less than 220 ms in children, or less than 200 ms following isoproterenol infusion, it is said that there is a significant risk of sudden cardiac death. (57).

In patients for whom sustained AF cannot be produced, SPPCL during atrial pacing has more recently demonstrated a strong connection with SPERRI both at baseline and during isoproterenol infusion. (58)

Teo (59) found that a SPERRI of less than 250 ms combined with the existence of several accessory routes produced a 92% specificity and a 22% positive predictive value for subsequent arrhythmic events.

There are a number of published guidelines outlining a strategy for treating asymptomatic WPW in children. Nevertheless, there was little to no connection between SPERRI and SPPCL during EPT and SPERRI as evaluated during a clinical episode of preexcited AF (Clinical-SPERRI). **(60)**

6-Sudden Cardiac Death

The initial indication of WPW syndrome may be sudden cardiac death, particularly in situations with familial WPW syndrome. (61)

Over a 3- to 10-year follow-up period, the rate of sudden cardiac mortality in patients with WPW syndrome has been estimated to range between 0.15% and 0.39%. (62)

Numerous markers that identify patients with WPW Syndeome who are at increased risk have been retrospectively identified through studies of WPW syndrome patients who have suffered a cardiac arrest. These markers include SPRRI less than 250 ms during spontaneous or induced AF, history of symptomatitachycardia, multiple accessory pathways (particularly septal APs), Ebstein's anomaly, and familial WPW. (49)

7 Management

7.1. Asymptomatic Patients

Because being asymptomatic does not prevent sudden cardiac death, managing asymptomatic patients with WPW patterns has long been contentious. There is currently no well-defined plan in place for these patients. (2) The majority of individuals will live their entire lives without experiencing any clinical symptoms associated with their ventricular pre-excitation. Risk stratification is still necessary for sudden cardiac death, even if they typically do not have indications for pharmacological treatment. The risk of SCD is evident, however slight, and is thought to be higher in males and younger individuals. (63).

Even in individuals who are asymptomatic, catheter ablation may be necessary for a number of reasons, depending on the noninvasive and/or invasive examination. Rapid conduction over the pathway during atrial pacing maneuvers or supraventricular arrhythmia is the basis for defining a high-risk pathway. (64).

It is generally accepted that preventive catheter ablation of the accessory route is advised if SPERRI during AF is < 250 ms, SPPCL≤ 250 ms, and APERP during planned atrial stimulation is ≤240 ms. (65).

Younger age, many routes, AVRT inducibility during EPS, and the accessory pathways' septal placement are further known risk factors for SCD. (2) If pre-excitation prevents a certain job (like pilots or sports), catheter ablation is also feasible in asymptomatic individuals. (66).

All patients with a WPW pattern who are willing to play competitive sports should have invasive EPS for risk assessment and ablation if the route has high-risk characteristics, according to the most recent guidelines. Since there appears to be little chance of a fatal incident among children under the age of twelve, a cautious approach is advised. (67).

As demonstrated by Etheridge et al. in a study that compared 96 case subjects with a history of major life-threatening events and 816 completely asymptomatic subjects, this does not imply that young patients with a WPW pattern do not experience life-threatening events in the absence of markers of high risk during the electrophysiological study. Of the case subjects, 60 out of 86 (69%) had at least two EPS risk stratification components completed; 22 out of 60 (37%) lacked EPS-identified high-risk characteristics, and 15 out of 60 (25%) lacked both inducible AVRT and worrying route characteristics. (54).

7.2. Symptomatic Patients

Currently, rather than relying on a particular clinical or electrophysiological strategy to prevent sudden death, the decision to deliver RFA or not has been rationally based on the existence or absence of symptoms. (68) The best way to treat tachycardia linked to WPW acutely is to use medications like adenosine to temporarily extend the AV node's refractory period. However, because adenosine lowers atrial ERP and can produce AF when combined with bradycardia post-PSVT conversion, it should be administered cautiously in patients who have known or suspected anterograde conduction over AP. This is because AF may be transmitted over the AP with a high ventricular rhythm. It is preferable to use ibutilide, procainamide, or flecainide since they can slow the conduction via the route (69). The fundamental idea behind treating preecxited AF is to reduce the pace of impulse transmission along the accessory pathway by extending the accessory pathway's anterograde refractory period in relation to the AV node. However, rapid cardioversion with external electrical shock is required in cases of hemodynamic collapse. (70) RFA has emerged as the preferred technique that may be used by all WPW patients. The outcomes of ablated and nonablated individuals differ significantly, according to long-term findings from registry studies. Complications were few, and success rates were good. Even more recently, a sizable German registry reported only issues that have a negligible effect on long-term quality of life (71, 72). An RF catheter ablation of WPW has a >94% success rate, a 6.2% recurrence rate, and a 1% complication risk, according to a recent meta-analysis(73).

The extensive use of cryo-ablation and a 3D electro-anatomical system has resulted in even greater success, a lower risk of problems and recurrences, and (almost) zero fluoroscopic exposure. (74)

Successful ablation is linked to a lower long-term mortality rate (because SCD is prevented) and a lower risk of heart failure (45).

However, it appears that if ablation is done before the age of 50, the long-term AF risk linked to WPW is greatly decreased (45).

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