



Review Article

WOLFF-PARKINSON-WHITE SYNDROME

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Article History:	Abstract
Received on: 19-06-2020 Revised on : 21-08-2020 Accepted on : 26-08-2020 Keywords: Atrioventricular Node, His-Purkinje System, Wolff-Parkinson-White Syndrome, supraventricular tachycardia.	WPW syndrome is a congenital heart disease that is characterized by the presence of an abnormal electrical connections between the atria and ventricles of the heart. In 1930, Louis Wolff, Sir John Parkinson, and Paul Dudley white published a seminal article describing the 11 young patients who suffered from attacks of tachycardia associated with an electrocardiographic pattern of 'bundle branch block' with a short PR interval. So from there onwards, it is called Wolff Parkinson white [WPW] syndrome. The normal conduction of the AV node occurs slowly than the accessory pathway conduction. Preexcitation is a process that the cardiac ventricles are activated earlier than the impulse of the AV node which leads to the shorter PR interval and formation of a delta wave. The supraventricular tachycardia associated with WPW syndrome is called AV reentrant or reciprocating tachycardia (AVRT). WPW syndrome is that there is an accessory pathway between the atrium and ventricles which cause rapid heartbeat or tachycardia.

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PREEXCITATION

It is the propagation of depolarization wave front that occurs more quickly than normal due to presence of an accessory conduction pathway that bypasses the normal conduction system. Normally the conducting system starts from the impulse of the SA node than it passes to AV node, HIS bundles, bundle of fibres, and Purkinje fibers. Impulse move to the conducting system normally with a brief delay occurring at the AV node so that the right and left atrium gets depolarized as soon as

AV node fires with a beating heart to an atrial contraction then after AV delay then this depolarization neutralizes the efficient and quickly HIS-PURKINJIE SYSTEM. The consequences of this in the EKG is that P-wave with PR interval lasts for 120-200-m/s followed by the sharp QRS complex of 120m/s. In the abnormal accessory pathway, when the impulse bypasses the AV node that has no intrinsic delay but due to connection in the atrium and the ventricles so that ventricular myocardium depolarized early which leads to short PR interval. In addition, the accessory pathway usually but not always inserts into non-specialised myocardium instead of directly into the his-purkinje system. The propagation of the aberrant conduction, once it gets to the ventricle conduction is relatively slow. This slow

in the intraventricular conduction leads to wide qrs comple.the qrs complex has specific characteristic addition in the initial slurring of the complex usually well defined as the primary cause of the qrs complex.The slurring is called delta wave. BUNDLE OF KENT -the extra or the accessory pathway between the atria and ventricle leads to preexcitation.

It has been estimated that most accessory pathways (60 to 75 percent) are capable of bidirectional conduction (antegrade and retrograde) between the atrium and ventricle. However, some accessory pathways (17 to 37 percent) are only capable of conduction in a retrograde fashion from ventricle to atrium [2]. When accessory pathways conduct exclusively in the retrograde direction (so-called "concealed" accessory pathways), they do not generate a delta wave (ie, slurred upstroke of the QRS complex) and the WPW pattern on the surface ECG but are still capable of supporting reentrant tachycardia. Retrograde conduction can occur following ventricular pacing or premature beats, and it can form the retrograde arm of an orthodromic AV reentrant tachycardia (AVRT) circuit. The vast majority of concealed accessory pathways are left-sided [3].

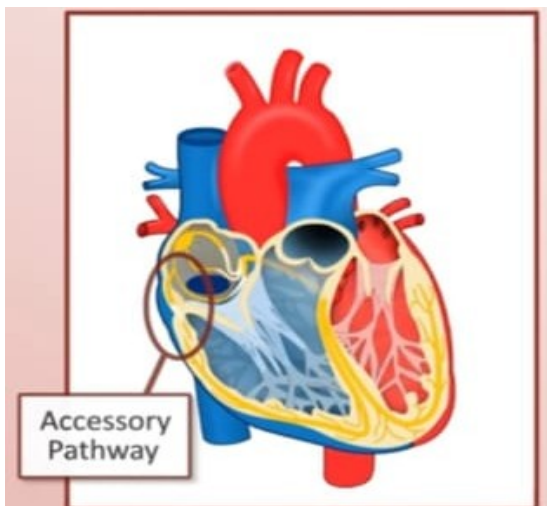


Fig 01: Accessory Pathway

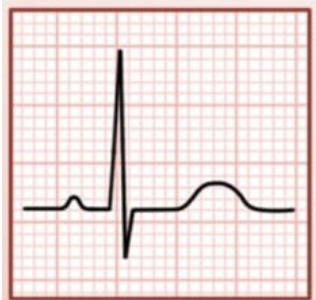


Figure 02: NORMAL EKG



Figure 03: Delta Wave

TYPES OF ACCESSORY PATHWAY

1. ATRIOVENTICULAR -MOST COMMONLY
 2. ATRIOFASCICULAR
 3. NODOFASCICULAR
 4. FASCICULOVENTICULAR
 5. ATRIONODAL-JAMES
 6. FIBRES/LGL PREEXCITATION
 7. ATRIO HISIAN-BRECHEN MACHU FIBRES.
- } MAHAIM FIBRES

SYMPTOMS

- ✓ Rapid palpitation
- ✓ Chest discomfort
- ✓ Shortness of breath
- ✓ Lightheadedness or syncope.
- ✓ Dizziness
- ✓ Sweating
- ✓ Onset and offset of the actions are typically sudden.
- ✓ Some patients may have the precipitating factors such as stress, coffee, menstrual period or pregnancy.
- ✓ Mostly symptoms are sporadic and unpredictable.
- ✓ Sudden death is rare but may sporadically be the initial presentation in previously asymptomatic individuals.
- ✓ A history of syncope raises concerns regarding a veryrapid arrhythmia with haemodynamic compromise.

HISTORY

- ❖ The earliest description of the accessory pathway was reported by Stanley kent in 1893 who suggested that impluses can travel from the atrium to the venticle over a node like structure other than the atrioventricular node
- ❖ Cohn and fraser reported the first case of the preexcitation syndrom in 1913.
- ❖ In 1930, Louis Wolff, Sir john parkinson and Paul dudley white published a seminal

article describing 11 young patient who suffered from attacks of tachycardia associated with an electrocardiographic pattern of 'bundle branch block' with a short PR interval.

- ❖ Ohnell was the first to use the term 'PREEXCITATION', whereas Seters described the slurred initial component of the QRS complex as a 'DELTA WAVE'.
- ❖ Curative therapy of WPW syndrome as demonstrated in 1976 when Cobb et al. successfully ablated an accessory pathway during open-heart surgery.
- ❖ The first successful catheter ablation of an accessory pathway by delivering direct current energy was reported by Moraday and Scheinman in 1984.
- ❖ In 1987, Borggrete et al. successfully ablated a right-sided pathway by delivering RF current. radiofrequency ablation of accessory pathways has become a first line therapy and is favoured over the medical treatment in most of the patients.

PATHOPHYSIOLOGY

In WPW syndrome there is abnormal electrical connection of heart by atria and ventricle. WPW symptom is the abnormal fast heart beat which is called tachycardia. In normal conduction the electric impulse start from the SA node and travel throughout the atria before they reach the AV node. AV node is the gate way to the ventricles. It relays the passage of the electric impulse to the ventricles and see that the all the amount of blood is ejected to the ventricles before the ventricles contract. This refractory property of the AV node is the centre in limiting the electrical activities to reach the ventricles. In this situation the atrial rate is excessively high during atrial fibrillation or atrial flutter. These fast ventricular rates can degenerate into ventricular fibrillation (VF) and cardiac arrest [4][5][6][7][8]. The AV node blocks most of the impulses from passing to the ventricles keeping the heart rate under control. In WPW there is an additional connection between the atria and ventricle which is the accessory pathway which is also called the bundle of Kent. This pathway is essentially a patch of the conducting tissue which pre-empt the shock up to the ventricles by passing the AV node. It allows the part of the electrical impulse to arrive to the ventricles soon which cause the

preexcitation, which leads to shorten the PR interval in ECG because the part of the ventricles depolarise occur and ventricular depolarisation develops more gradually and lasts longer, resulting the slow rise in the initial complex of QRS known as delta wave [QRS is prolonged].

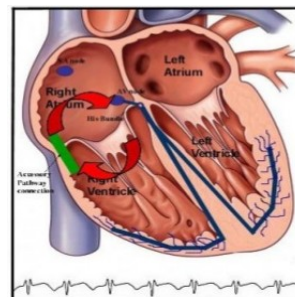
The presence of the accessory pathway along doesn't cause the tachycardia. Most the people in WPW doesn't develop any symptoms which is called WPW pattern and when it is symptomatic then it is called WPW syndrome.

MECHANISM WHICH MAY CAUSE WPW

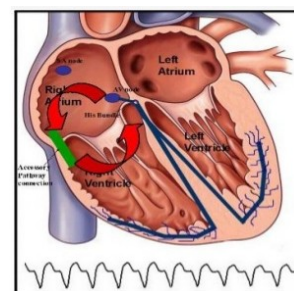
1. ATRIOVENTRICULAR REENTRY TACHYCARDIA [AVRT]

Most commonly tachycardia develops when electrical impulse develops and travel down in one pathway either the normal or accessory backup the other creating the self-propagating loop or reentrance circuit. The frequency of the loop determines the heart rate and can be very fast from 150-250 beats per minute. This is known as AVRT. AVRT can be orthodromic or antidromic depending on the direction of the loop:

- Orthodromic - down via AV node, 95%.
Descend - normal pathway
Ascend - accessory pathway



- Antidromic - up via AV node, 5%.
Less common pathway
Descend - accessory pathway
Ascend - normal pathway



2.WPW COMBINED WITH ATRIAL FIBRILLATION

- ❖ In this condition, atria contract at very high rate that most of the electric impulses don't make it through the AV node to the ventricles. This is where WPW pathway can have a detrimental effect. It provides a bypass to move impulses and reach the ventricles which causes the faster heart rate that could potentially be fatal. The severity of WPW tachycardia depends on how fast the accessory pathways are able to conduct.

MANAGEMENT

- ❖ Wolff-Parkinson-White (WPW) syndrome is a conduction disorder of the heart caused by pre-excitation accessory pathway resulting in tachyarrhythmias. WPW tachycardia varies from person to person and it can be evaluated by a procedure called programmed electrical stimulation. In which atria are stimulated with progressively higher rate with atrial to ventricular conduction ratio is monitored. Patients developing high risk with lethal tachycardia if the accessory pathway continues to the 1:1 ratio which is dangerous high atrial rates. High risk patients are treated with the catheter ablation to destroy the conductive tissue of the accessory pathway
- ❖ Patients are managed by class -1a drug called procainamide or IV infusion if the patient is hemodynamically stable.
- ❖ If the patients are unstable due to immediate synchronized cardioversion then catheter ablation of accessory pathway is done. This is the definite treatment of WPW because it is a destruction of abnormal electrical pathway
- ❖ Administration of amiodarone, adenosine, beta blockers, and calcium channel blockers should be avoided as these will isolate the accessory pathway and thus predispose to fatal arrhythmias such as ventricular fibrillation by increasing the ventricular rate [9,10,11,12,13].
- ❖ AV node blockers should be avoided in atrial fibrillation [AF] and atrial flutter with WPW which includes calcium channel blockers [CCB], digoxin, diltiazem, adenosine, verapamil and beta blockers.

- ❖ If the patient is hemodynamically stable
Normal QRS in ECG-AV nodal blocking drugs.
Narrow QRS without AF in ECG-adenosine, verapamil etc.
Wide QRS in ECG [orthodromic AVRT/antidromic AVRT/AF/atrial flutter]-Procainamide, sotalolol, amiodarone, flecainide.
- ❖ Ebstein anomaly and hypertrophic cardiomyopathy should be evaluated. [In case more than one accessory pathway].

CONCLUSION

WPW syndrome is a cardiac conduction disorder which is caused by the presence of an extra accessory AV pathway between atria and ventricle which is called bundle of Kent and its main symptom is the palpitations. We have to take close attention to the symptoms of the patients so that it helps in making the crucial diagnosis. Once the diagnosis is done it helps in the further evaluation and the management of this syndrome. For this syndrome the main and the first line treatment is the catheter ablation therapy.

REFERENCE

1. 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.
2. Miller JM. Therapy of Wolff-Parkinson-White syndrome and concealed bypass tracts part I. *J Cardiovascular Electrophysiol* 1996;7:85
3. Kuck KH, Friday KJ, Kunze KP, et al. Sites of conduction block in accessory atrioventricular pathways. Basis for concealed accessory pathway. *Circulation* 1990;82:407.
4. Mirzoyev S, McLeod CJ, Asirvatham SJ. Embryology of the conduction system for the electrophysiologist. *Indian Pacing Electrophysiol J*. 2010 Aug 15;10(8):329-38.
5. Pediatric and Congenital Electrophysiology Society (PACES). Heart Rhythm Society (HRS). American College of Cardiology Foundation (ACCF). American Heart Association (AHA). American Academy of Pediatrics (AAP).

- Canadian Heart Rhythm Society (CHRS). Cohen MI, Triedman JK, Cannon BC, Davis AM, Drago F, Janousek J, Klein GJ, Law IH, Morady FJ, Paul T, Perry JC, Sanatani S, Tanel RE. PACES/HRS expert consensus statement on the management of the asymptomatic young patient with a Wolff-Parkinson-White (WPW, ventricular preexcitation) electrocardiographic pattern: developed in partnership between the Pediatric and Congenital Electrophysiology Society (PACES) and the Heart Rhythm Society (HRS). Endorsed by the governing bodies of PACES, HRS, the American College of Cardiology Foundation (ACCF), the American Heart Association (AHA), the American Academy of Pediatrics (AAP), and the Canadian Heart Rhythm Society (CHRS). *Heart Rhythm*. 2012 Jun;9(6):1006-24.
6. Al-Khatib SM, et al. Risk Stratification for Arrhythmic Events in Patients With Asymptomatic Pre-Excitation: A Systematic Review for the 2015 ACC/AHA/HRS Guideline for the Management of Adult Patients With Supraventricular Tachycardia: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *J. Am. Coll. Cardiol*. 2016 Apr 05;67(13):1624-1638.
 7. Page RL, Joglar JA, Caldwell MA, Calkins H, Conti JB, Deal BJ, Estes NA, Field ME, Goldberger ZD, Hammill SC, Indik JH, Lindsay BD, Olshansky B, Russo AM, Shen WK, Tracy CM, Al-Khatib SM., Evidence Review Committee Chair. 2015 ACC/AHA/HRS Guideline for the Management of Adult Patients With Supraventricular Tachycardia: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *Circulation*. 2016 Apr 05;133(14):e506-74.
 8. Bhatia A, Sra J, Akhtar M. Preexcitation Syndromes. *Curr Probl Cardiol*. 2016 Mar;41(3):99-137.
 9. Yealy DM, Kosowsky JM. Chapter 79: Dysrhythmia. *Rosen's Emergency Medicine Concepts and Clinical Practice*. 2014.
 10. January CT, Wann LS, et al. 2014 AHA/ACC/HRS Guideline for the Management of Patients With Atrial Fibrillation: Executive Summary
 11. Blomstrom-Lundqvist C, Scheinman MM, Aliot EM, et al. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Supraventricular Arrhythmias) developed in collaboration with NASPE-Heart Rhythm Society. *J Am Coll Cardiol*. 2003;42:1493-531.
 12. McGovern B, Garan H, Ruskin JN. Precipitation of cardiac arrest by verapamil in patients with Wolff-Parkinson-White syndrome. *Ann Intern Med*. 1986;104:791-794.
 13. Simonian SM, Lotfipour S, Wall C, et al. Challenging the superiority of amiodarone for rate control in Wolff-Parkinson-White and atrial fibrillation. *Intern Emerg Med*. 2010;5(5):421-6.