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# Wolf-parkinson-white Syndrome-current Concepts in Anesthetic Practice

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## Authors' contributions

*This work was carried out in collaboration between all authors. Author VM designed and wrote the first draft of the manuscript and did the corrections whenever needed. Authors SK and BB managed the literature searches. All authors read and approved the final manuscript.*

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## ABSTRACT

Wolf-Parkinson-White (WPW) syndrome is a disorder of conduction system of heart caused by presence of an abnormal accessory conduction pathway between the atria and ventricles. It is associated with tachyarrhythmias diagnosed by electrocardiography (ECG). The anesthetic management of these patients is challenging as they are known to develop life threatening arrhythmias. Magnesium sulfate ( $MgSO_4$ ) is a good agent for control of peri-operative dysrhythmias.

**Keywords:** *Wolf-Parkinson-White (WPW) syndrome; tachyarrhythmias; regional anesthesia;  $MgSO_4$ .*

## 1. INTRODUCTION

Wolf-Parkinson-White (WPW) syndrome is a disorder of the conduction system of the heart, commonly referred to as "Pre-excitation syndrome". WPW is caused by presence of an abnormal accessory conduction pathway between the atria and the ventricles [1,2]. Accessory pathways are congenital and represent remnants of fetal atrio-ventricular muscular connection left by incomplete development of *annulus fibrosus* (bundle of Kent and

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mahaim), so they are associated with congenital heart disease like EBSTEIN anomaly or Transposition of great arteries [3]. This pathway allows atrial activity to circumvent the His-Purkinje system, conducting in an anterograde, retrograde or bidirectional manner [4]. This allows a part of ventricle to become excited before it should and manifest on electrocardiography (ECG) as earlier than normal deflection of QRS complex called DELTA wave (slurred upstroke of QRS). Electrical signals travelling down this abnormal pathway may stimulate ventricles to contract prematurely, resulting in unique type of supraventricular tachycardia (SVT) referred to as atrioventricular reciprocating tachycardia. Supra ventricular tachycardia (SVT) and atrial fibrillation (AF) can occur in WPW syndrome triggered by atrial, ventricular or junctional premature beats. Paroxysmal supraventricular tachycardia (PSVT) is the most common arrhythmia associated with WPW syndrome [5]. The arrhythmias can be narrow or broad complex tachycardia, with or without pre excitation.

Extensive investigations, both in laboratory and in clinical studies have been undertaken to define the role of magnesium in the genesis and the treatment of cardiac arrhythmias [6]. Hypomagnesaemia along with Hypokalemia have been implicated in the genesis of both atrial and ventricular arrhythmias [7]. The mechanism by which magnesium acts is probably by slow inward calcium current block, which decreases sinus node rate, prolongs atrio-ventricular (AV) conduction time and increases AV node refractoriness without major changes in ventricular physiology [8].

## **2. PATHOPHYSIOLOGY**

In the normal human heart, electrical activity arises from the sino-atrial (SA) node, which is located in the right atrium. From there it is transmitted to the AV node. After a brief delay at the AV node the stimulus is conducted through the bundle of His to the left and right bundle branches and then to the Purkinje fibres and the endocardium at the apex of the heart, then finally to the ventricular myocardium.

The AV node limits the electrical activity reaching the ventricles. In case of atrial fibrillation or atrial flutter the AV node limits the number of signals conducted to the ventricles. In atrial fibrillation, when atrial activity is 300 beats per minute, half of the impulses are blocked by the AV node, so ventricles are stimulated at 150 beats per minute. But in individuals with WPW syndrome there is an accessory pathway which does not share the rate slowing properties of AV node, causing ventricles to contract at the same rate as atria. Extremely rapid heart rate such as this may result in hemodynamic instability or cardiogenic shock. In some cases, the combination of an Accessory pathway and Atrial Fibrillation can trigger Ventricular fibrillation (VF).

The short PR interval and slurred upstroke of the QRS complex known as the delta wave is the result of conduction through accessory pathway.

There can be two types of Pre excitation "Type A pre excitation"- in which the accessory pathway communicate between the left atrium and the left ventricle.

'Type B pre excitation' – in which the accessory pathway exist between the right atrium and the right ventricle [9].

Magnesium inhibits basal, myogenic and hormone-induced smooth muscle contraction and also has a direct vasodilator effect [10]. Magnesium blocks calcium entry into vascular

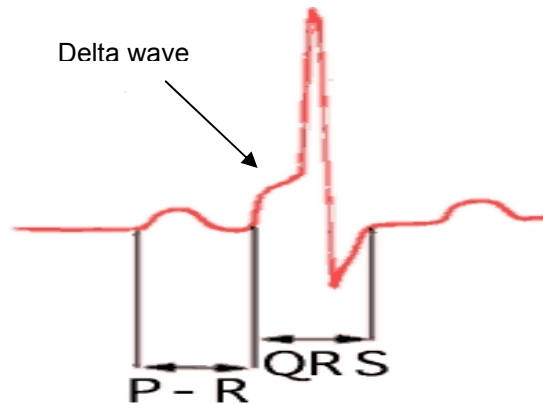
smooth muscle cells via voltage and receptor operated channels and it diminishes the reactivity of these cells to a variety of pressor agents.

### 3. SIGN AND SYMPTOMS

The patient may experience palpitation, dizziness, shortness of breath or syncope during Supraventricular tachycardia. The “delta waves” may be seen on electrocardiogram.

### 4. DIAGNOSIS

WPW is commonly diagnosed by ECG in an asymptomatic patient. The diagnostic criteria in ECG include (1) PR interval <0.12 sec in presence of sinus rhythm (2) Abnormally wide QRS complex >0.10 sec and (3) Presence of initial slurring in first 0.03 sec to 0.05 sec of the QRS complex as shown in Fig. 1.



**Fig. 1. Shows the slurring of QRS complex forming “Delta Waves”**

If a patient with WPW syndrome experiences episodes of AF, the ECG will show a rapid polymorphic wide-complex tachycardia which is considered dangerous and most anti arrhythmic drug are contraindicated.

In case of Type A pre excitation (left atrioventricular connection) a positive R wave will be seen in  $V_1$  (positive delta) on the precordial leads of ECG, while in Type B pre excitation (right atrioventricular connection), a predominately negative delta wave will be seen in lead  $V_1$  (negative delta).

All patients with WPW syndrome should undergo 24 hour holter monitoring. Patient with confirm Supraventricular tachycardia caused by accessory pathway should undergo Electrophysiological testing for risk stratification and radiofrequency ablation of the accessory pathway in Electrophysiology (EP) laboratory [11].

People with WPW syndrome may have more than one accessory pathway—in some cases—as many as eight abnormal pathways have been found. This has been seen in individuals with Ebstein anomaly.

WPW syndrome is associated with Leber's hereditary optic neuropathy, a form of mitochondrial disease [12].

## **5. PREDICTION OF MORTALITY AND MORBIDITY**

The incidence of WPW is 0.1-0.3% [13,14,15] in the general population. Sudden cardiac death in people with WPW is rare i.e. less than 0.6% [16] and is usually caused by the propagation of an atrial tachydysrhythmia to the ventricles by abnormal pathway. Risk stratification is performed to determine the treatment and risk of sudden cardiac death in an individual. A good history should be taken to determine whether an individual has factors suggestive of a previous episode of unexplained syncope or palpitation as they may be associated with tachycardia along accessory pathway. Individuals with WPW syndrome in whom the delta wave disappears with increase in heart rate are associated with lower risk of sudden cardiac death. This is because the loss of delta wave shows that the accessory pathway cannot conduct electrical impulses at high rate.

Risk stratification is best performed via programmed electrical stimulation in the cardiac electrophysiology laboratory. This is an invasive procedure, in which the rate of impulse propagation via accessory pathway is determined by stimulating the atria and by inducing transient atrial fibrillation.

High risk features includes effective refractory of accessory pathway less than 270 millisecond, multiple pathway, septal location of pathway and inducibility of supraventricular tachycardia. The individuals are at increased risk of sudden cardiac death however, it is unclear whether invasive risk stratification is necessary in asymptomatic patient [17].

## **6. MEDICAL MANAGEMENT**

If WPW syndrome is associated with SVT and the patient is hemodynamically stable- vagal maneuver should be tried and amiodarone is given. If they are ineffective- procainamide [18] is given. If the patient is hemodynamically unstable and is in AF, then synchronized DC cardio version is done. Drugs that slow AV conduction like verapamil, beta-blocker and digoxin are contraindicated as these slow normal conduction and increase conduction through accessory pathway. I.V procainamide up to the dose of 10mg/kg slows conduction through accessory pathway.

## **7. ANESTHETIC CONSIDERATIONS**

Cardiac antidysrhythmic drugs should be continued throughout the perioperative period. Our goal is to avoid sympathetic activity triggered by pain, anxiety or hypovolemia or drug causing sympathetic activity. Regional anesthesia is preferred because multiple drugs are avoided and noxious stimulus of laryngoscopy is omitted but hemodynamic stability is worrisome [19,20].

Segmental Epidural anesthesia is preferred to spinal anesthesia thus ensuring greater hemodynamic stability [21].

If general anesthesia is given, a deeper plane of anesthesia should be achieved before direct laryngoscopy and the duration of laryngoscopy should be as short as possible.

Atropine, ketamine and glycopyrrolate can precipitate tachycardia and can lead to paroxysmal supraventricular tachycardia (PSVT) or AF.

As an induction agent Propofol has got no effect on refractory period of accessory pathway. However, in one reported case propofol causes delta waves to disappear [22]. Thiopentone and benzodiazepenes could be given as they have no effect on accessory pathway. Etomidate being more cardiac stable is preferred.

Many volatile agents can precipitate conduction via pre existant anomalous pathway. Isoflurane prolongs the antegrade effective refractory period of accessory pathway as well as refractory period of atrial and ventricular muscle and is the agent of choice [23,29].

For neuromuscular blockade rocuronium is preferred over pancuronium which causes tachycardia. Atracurium causes histamine release with less autonomic safety. Newer drugs cis atracurium and mivacurium can be used safely [19,20] because reversal is not required with neostigmine and glycopyrrolate as neostigmine may enhance accessory pathway during AF associated with WPW syndrome [24]. Sugammadex, a newer reversal agent is not associated with hemodynamic instability and prolonged QT interval, so can be used in a patient of WPW syndrome [25].

The conditions precipitating arrhythmias like hypoxia, hypercarbia, hypotension, hypoglycemia, acidosis, electrolyte imbalance and light plane of anesthesia should be avoided.

Patient with WPW syndrome can be managed successfully under general and combined spinal and epidural anesthesia [26] provided adequate perioperative preparations are done and this case report suggests that use of propofol, fentanyl, isoflurane and vecuronium can be successfully used in anesthetic management of WPW syndrome.

## **8. ROLE OF MAGNESIUM SULFATE (MgSO<sub>4</sub>)**

MgSO<sub>4</sub> is an antiarrhythmic agent which causes prolongation of PR interval without any significant effect on refractory period of accessory pathway and atrioventricular (AV) conduction time. Also magnesium inhibits the release of catecholamine and might blunt the hemodynamic response to inadequate analgesia. Magnesium has been used for the treatment of paroxysmal atrioventricular tachycardia in WPW syndrome [27]. Magnesium is effective in treating a variety of serious arrhythmias, including ventricular arrhythmias resistant to proven antiarrhythmic agent with low serum magnesium level [28].

We have used MgSO<sub>4</sub> in two known case of WPW syndrome posted for surgery. First, a case of fibroid uterus for hysterectomy under epidural analgesia with Ropivacaine 0.5% and second, a case of traumatic hematoma in flanks under general anesthesia using Propofol, Fentanyl, Vecuronium and Isoflurane. In both the cases MgSO<sub>4</sub> in dose of 40 mg/kg body weight was given along with pre medication in 100ml of isotonic saline over 10 minutes and then 15mg/kg by continuous i.v infusion till end of operation. MgSO<sub>4</sub> prevented development of tacharrhythmias.

Siders et.al conducted a study to see the effect of Mg<sup>2+</sup> and potassium on WPW syndrome. He found that both magnesium and potassium transiently abolish pre-excitation in some patients especially during tachyarrhythmias [30].

At present limited literature is available on use of MgSO<sub>4</sub> in WPW syndrome hence further studies are required to establish its use for management of tachyarrhythmias in WPW syndrome.

## **9. DISCUSSION**

Wesley and colleagues describe the effect of single bolus dose of Magnesium 2gm in SVT and demonstrated slowing or termination of tachycardia when AV node was a part of re-entrant circuit in 7 out of 10 patient magnesium is also being used successfully in the treatment of ventricular arrhythmias after acute myocardial infarction, long QT syndrome and digoxin toxicity [31].

Magnesium has been incorporated into algorithms for management of broad complex tachycardia in the Resuscitation Council Advance Life Support manual [32].

S.Yamaguchi et.al reported the anesthetic management using propofol and fentanyl in a patient with concealed WPW syndrome which was diagnosed electro physiologically 7 years before. They concluded that anesthesia using above agents with WPW syndrome is useful in preventing paroxysmal tachycardia [33].

Sharp et al studied 21 patient WPW syndrome undergoing surgical ablation, they were anaesthetized with sufentanyl 20µg/kg, lorazepam 0.06mg/kg, and vecuronium 20mg. This study showed Sufentanyl-lorazepam had no clinical significant effect on the electrophysiological expression of accessory pathway. Of the volatile agents and enflurane most, isoflurane next and halothane least increased the refractoriness within the accessory end atrioventricular pathway [34].

Sharpe MD, in a study stated that propofol has no significant effect on the electrophysiological expression of accessory pathway and the refractoriness of the normal AV Conduction system, in addition there is no direct effect on SA node activity or intra-arterial conduction, and therefore, it does not directly induce bradyarrhythmia. It is thus a suitable agent for use in patient undergoing ablative procedure that requires either neuroleptic or general anesthesia [35].

## **10. CONCLUSION**

To summarize WPW syndrome is a disorder due to abnormal conduction pathway in heart. Anesthetic management of these patients is challenging as they may develop perioperative tachyarrhythmias and hypotension leading to cardiac arrest. Meticulous monitoring along with use of MgSO<sub>4</sub> and other antiarrhythmic drugs is the key for peri-operative management of these cases.

## **COMPETING INTERESTS**

Authors have declared that no competing interests exist.

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