

# CARDIAC ARRHYTHMIAS AND ECHOCARDIOGRAPHIC FEATURES IN WOLFF-PARKINSON-WHITE SYNDROME

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## SUMMARY

*Electrocardiographic features of the Wolff-Parkinson-White syndrome may be seen in normal individuals and in those with congenital or acquired heart disease. Predisposition to tachyarrhythmias and its misinterpretation are common. In this report a case of Wolff-Parkinson-White syndrome in a 25 year old Malay male who presented with cardiac arrhythmias is described. Echocardiographic findings and the role of echocardiography are discussed.*

## INTRODUCTION

THE Wolff-Parkinson-White (WPW) syndrome characterised by short P-R interval and prolonged QRS interval due to initial slurring of the QRS complex, the delta wave, was first described by Wolff *et al.* in 1930. The disorder produces a conduction anomaly of atrial impulse via two different pathways to the ventricles, one of which is the normal specialised conduction system and the other an anomalous pathway (pre-excitation). With pre-excitation the ventricular muscle is activated earlier subjecting such patients to the susceptibility of paroxysmal supraventricular tachyarrhythmias. The clinical significance of the WPW syndrome, in addition to the frequent predisposition to dysrhythmias, is the association of congenital and acquired cardiac disorders with

this syndrome (Newman *et al.*, 1966). Studies made to assess the influences of pre-excitation on ventricular contraction involved either invasive pressure recordings (Ferrer *et al.*, 1949) or tedious inconclusive procedures like roentgenkymography (Bandiera and Antongnetti, 1958) and electrokymography (Dack *et al.*, 1951). With the introduction of echocardiography a reliable and reproducible non-invasive method is available for the detection of abnormalities and studying the pattern of motion of the left ventricles with pre-excitation. It further serves in the detection or exclusion of other associated cardiac disorders.

This report describes the types of arrhythmias seen and the echocardiographic features in a 25 year old Malay clerk with the Wolff-Parkinson-White syndrome. The role of echocardiography and problems in diagnosis and management of such a case is discussed.

## CASE REPORT

N.H., a 25 year old Malay clerk, was admitted to the Coronary Care Unit for palpitations. He was well till the day of admission (3-5-80) when he was suddenly awoken by retrosternal discomfort. This was associated with palpitations, profuse sweating but no syncopal episodes or frequency of micturition. The palpitations persisted till he was admitted five hours later. In the past there was one similar episode 4 months ago which lasted 2 hours and subsided spontaneously. He smokes 10-15 cigarettes per day and had no relevant past medical or family history. Physical examination on admission revealed an apprehensive, well built young man. The pulse was 160 per minute and irregular in volume and rhythm. The blood pressure was 120/70mm.Hg. There was no cardiomegaly or significant murmurs heard. Examination of the other systems was normal.

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The haemoglobin was 13.8 Gms.% and the total white count was 4,800 with a normal differential. The ESR was 8 mm. in the first hour. Serum electrolytes, blood urea, fasting blood sugar, fasting serum lipids, cardiac enzymes on three consecutive days and serum  $T_4$  were normal. The electrocardiogram on admission (Fig. 1 A) showed rapid atrial fibrillation with a ventricular response of 150-160 per minute. Intermittent widening of the QRS complexes of a regular nature seen in  $V_1$  and  $V_3$  made differentiation of ventricular tachycardia or a supraventricular tachycardia with ventricular aberrancy difficult. Verapamil (Isoptin given intravenously slowed the heart rate to 75-80 per minute, but there was persistence of atrial fibrillation. Several hours after admission, he reverted to sinus rhythm spontaneously. Sinus rhythm was maintained only transiently before reverting back to atrial fibrillation. The electrocardiogram done during sinus rhythm showed a P-R interval of 0.16 seconds, normal QRS complexes and non-specific T wave inversion in the inferior leads (Fig. 1B). Over the next 2 days, continuous electrocardiographic monitoring

showed varying types of supraventricular tachyarrhythmias (Fig.2) which included rapid atrial fibrillation, atrial tachycardia, supraventricular tachycardia with aberrancy and slow atrial fibrillation. Digoxin was commenced on the day of admission and Quinidine sulphate (Kinidine) added 2 days later. The heart rate was satisfactorily controlled after this. He again reverted to sinus rhythm on 6-5-80 which was maintained. The electrocardiogram at this stage showed sinus rhythm, short P-R interval of 0.06 seconds, delta wave on the upstroke of the QRS complex, widened QRS complex of 0.16 seconds and secondary ST and T wave changes - features characteristic of the Wolff-Parkinson-White syndrome, type B (Fig.3). At echocardiography, an early abrupt systolic posterior movement of the interventricular septum 40 msec. after the onset of the QRS complex followed by a paradoxical mid-systolic anterior motion and a late systolic anterior movement was seen (Fig.4B). Posterior left ventricular wall motion was normal. The aortic and mitral valves and dimensions of the left ventricle, right ventricle and left atrium were also normal.

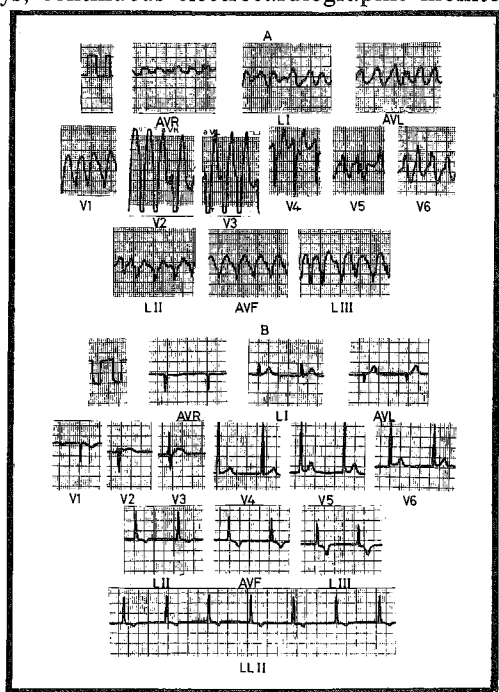


Figure 1. A is the electrocardiogram recorded on admission, and B the electrocardiogram obtained during sinus rhythm with normal AV conduction.

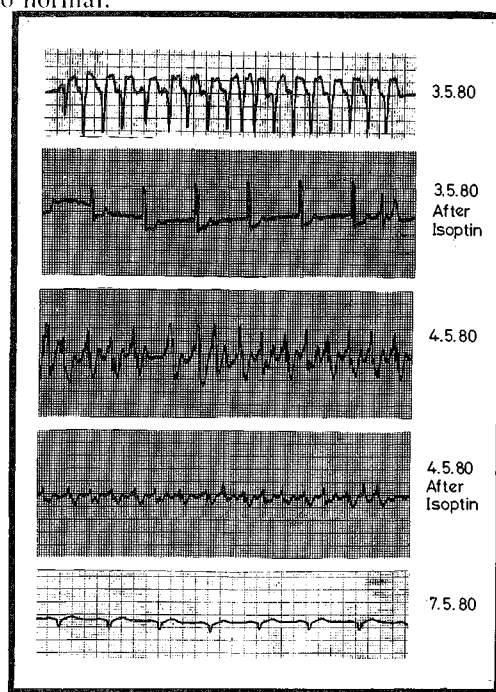


Figure 2. Rhythm strips obtained during continuous electrocardiographic monitoring [see text for description].

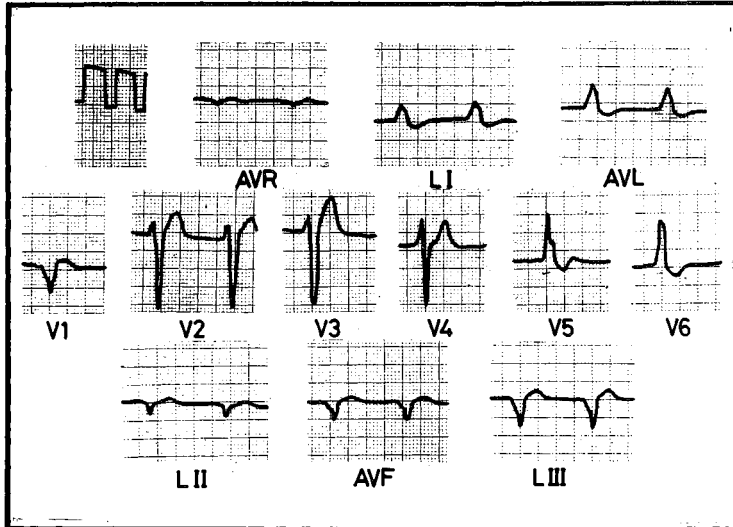


Figure 3. Sinus rhythm with anomalous AV conduction. Characteristic features of Wolff-Parkinson-White syndrome are seen more distinctly in Leads V<sub>3-5</sub>. Misinterpretations of left bundle branch block and inferior wall myocardial infarction are common with such patients.

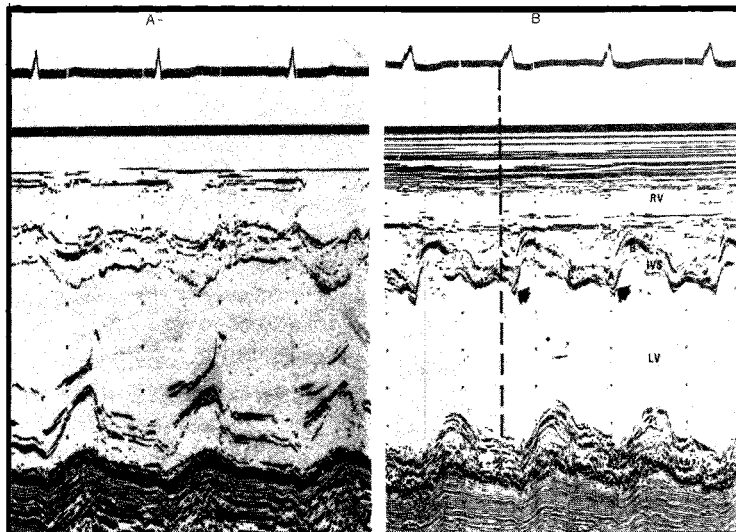


Figure 4. Panel A is an echocardiogram of the left ventricle showing normal interventricular septal and posterior wall motion. In panel B characteristic motion of the interventricular septum in Type B WPW syndrome is shown; abrupt early systolic posterior movement[A], mid-systolic paradoxical anterior septal motion[B] and delayed posterior movement in systole[C]. [RV-right ventricle, IVS-interventricular septum, LV-left ventricle, LVPW-left ventricular posterior wall, dashed line indicates the onset of electrical depolarization of the left ventricle].

## DISCUSSION

A true incidence of the WPW syndrome in adults is difficult to assess, and is estimated to range from 0.1-0.2 per cent of the general population with a male preponderance. 60 to 70% of those with this electrocardiographic anomaly are in healthy individuals with no organic heart disease. The incidence of tachyarrhythmias complicating ventricular pre-excitation is 40 to 80% (Chung *et al.*,1965). Tachyarrhythmias in the WPW syndrome vary from atrial tachycardia to atrial fibrillation or flutter. In the majority of cases regular supraventricular tachycardia with heart rates of 140-250 beats per minute is the commonest arrhythmia encountered. Atrial fibrillation and atrial flutter occur less commonly and account for 20-25% of all tachyarrhythmias (Chung *et al.*,1965). During these arrhythmias the ventricular complexes may be normal or widened due to aberrant ventricular conduction. The aberration of the QRS complexes may simulate ventricular tachycardia (Yahini *et al.*,1964) or ventricular fibrillation (Herrmann *et al.*,1957). The incidence of supraventricular tachycardia with aberration in the WPW syndrome ranges from 14% (Chung *et al.*,1965) to 60% [Herrmann, *et al.*,1957]. Aberration in ventricular response is more common in atrial fibrillation or flutter than in atrial tachycardia and can be distinguished by the rapidity of the ventricular response and the bizarre, broad irregularity of the QRS complexes. Although ventricular tachycardia is reported with this syndrome, an review of the electrocardiographic features described no unequivocal cases of ventricular tachycardia, were revealed. The described cases most probably represented misinterpretation of atrial fibrillation or flutter with anomalous atrio-ventricular conduction. On the other hand, ventricular fibrillation has been reported in the WPW syndrome (Dreifus *et al.*,1957., Klein *et al.*,1979). This dramatic complication generally occurs in patients with atrial fibrillation and rapid ventricular response, predisposing such patients to the risk of sudden death.

Recognition of the WPW syndrome is important not only because of its predisposition to

tachyarrhythmias. The abnormal QRS complex often resembles other electrocardiographic findings leading to erroneous diagnosis. Inferior and anteroseptal infarctions may be simulated by the inscription of Q or QS waves in leads II,III,aVf and V1+2 in Type B WPW(Kariv,1958). **Wide QRS complexes due to the delta wave** frequently resemble either right or left bundle branch blocks. When the electrocardiographic abnormality of this syndrome occurs intermittently it may resemble premature ventricular contractions or short runs of ventricular tachycardia (Chung *et al.*,1965). Should acute myocardial infarction occur in a patient with the WPW syndrome, diagnosis of infarction may be difficult or impossible electrocardiographically. The anomalous atrio-ventricular conduction produces changes in the QRS complex preventing the development of QRS abnormalities which are diagnosis of infarction. In such situations observation of normally conducted beats when it occurs spontaneously or induced to appear would obviate the masking effect of the WPW syndrome(Wolff and Richman,1953).

The role of echocardiography in the WPW syndrome is twofold. Firstly, it serves as a useful non-invasive method of studying the effects of pre-excitation on motions of the interventricular septum and posterior left ventricular wall. In addition, cardiac disorders associated with this syndrome which include Ebstein's anomaly, atrial and ventricular septal defects, tetralogy of Fallot, hypertrophic cardiomyopathy and ischaemic heart disease may be evaluated, detected or excluded by echocardiography (Newman *et al.*,1966., Schiebeler *et al.*,1959). In Type A WPW syndrome, DeMaria *et al.*(1976) described a localised area of premature contraction of the left ventricular posterior wall occurring during the initial 100 msec. Following the onset of the QRS complex accompanied by paradoxical interventricular wall was similarly demonstrated by Kotler *et al.*(1978) in 4 out of 6 patients with Type A WPW syndrome but could not detect any abnormality in the motion of the interventricular septum. In Type B WPW syndrome, abnormalities in interventricular septal motion are more characteristic. Francis *et al.*(1976) described 11 cases and

Hishida *et al.* (1976) reported 10 patients with Type B WPW syndrome showing similar inter-ventricular septal motions characterised by an abrupt early systolic posterior movement, a subsequent mid-systolic paradoxical anterior septal motion and a delay in the usual posterior movement in systole.

With this patient the diagnosis of WPW syndrome was only made several days after admission. This was after he spontaneously reverted to sinus rhythm the second time which showed anomalous atrio-ventricular conduction (Fig.3). He first reverted to sinus rhythm several hours after admission but a diagnosis could not be made as the electrocardiogram showed normal conduction (Fig.1B). Thus, a diagnosis of the WPW syndrome may be difficult if an electro-

cardiogram is not obtained during sinus rhythm with anomalous conduction. He, in addition, developed varying types of supraventricular tachyarrhythmias in the first two days after admission (Fig.2). During this period the diagnosis had not been established and treatment was aimed at slowing the heart rate. This case serves to illustrate the point that a diagnosis of WPW should be considered in the differential diagnosis when such arrhythmias are encountered in young adults in the absence of organic heart disease.

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