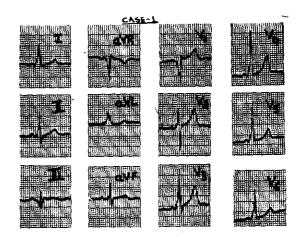
# **ELECTROCARDIOGRAPHIC DIAGNOSIS**

Surg Lt Cdr TK SAHNI\*, Surg Cdr A. BANERJEE SC\*\*, Surg Cdr PS LAMBA\*\*\*

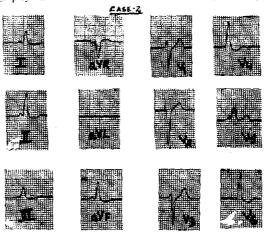
#### Case 1

25 year old, healthy young man with no significant past or family medical history underwent an ECG while being examined for fitness for diving. His clinical, haematological and biochemical parameters were all normal. ECG is shown alongside. Will he be made fit for diving?



### Case 2

43 year old male reported with prae cordial discomfort to his doctor. He had no previous history of any ailment, had no cardiovascular risk factors and no family history of coronary heart disease. Clinical examination revealed a normotensive individual with a normal cardiovascular system. ECG done is shown along side. Other metabolic and biochemical parameters were normal. He was treated as a case of coronary artery disease. Subsequently tread mill, stress MUGA and coronary arteriography showed normal coronaries. What is your diagnosis?



<sup>\*</sup>Graded Specialist Marine Medicine and Physician; \*\*Classified Specialist Medicine and Neurology; \*\*\* Classified specialist Medicine and Endocrinology, INHS Asvini Colaba, Bombay - 400 005.

# INTERPRETATION

The 12 lead ECG recording in both the cases show a normal sinus rhythm with a short PR interval (less than 0.12 second) and a slurring of the initial portion of the QRS complex (the delta wave), thus establishing a diagnosis of WPW syndrome. The second case also shows associated ST segment and T wave changes in a direction opposite to that of the QRS complex, which are secondary to WPW conduction defect. The presence of a dominant 'S' in leads VI and V2 classifies these ECG's as that of Type B WPW Syndrome.

### DISCUSSION

Preexcitation implies that part of the ventricular myocardium is activated before it would have been by an impulse descending via the normal A-V conduction system. The two main variants of preexcitation are Wolff Parkinson White (WPW) and the Lown Ganong Levine (LGL) Syndromes.[1]

The incidence of WPW syndrome is approximately 1-3 per 1000 in the population. It is a congenital anomaly that usually is not associated with other evidence of organic heart disease. It has been associated with certain congenital and acquired cardiac lesions such as Ebstien's anomaly, prolapse mitral valve syndrome, corrected transposition, idiopathic hypertrophic subaortic stenosis, septal defects, tetralogy of Fallot's, cardiomyopathies and thyrotoxicosis [2,3].

Amongst the preexcitation syndromes, WPW is the most common and is due to an accessory pathway (Kent bundle) connecting the atria and the ventricle. Two types of preexcitation patterns have been described in the past. In type A there are tall R waves in VI-2 and in Type B there are tall R waves in left precordial leads and negative QRS complexes in VI-2, However, there are many variations of the above [2].

The criteria for diagnosis of WPW are (a) a short PR interval, 0.12 second or less in 85% of cases in presence of normal P waves (b) a wide QRS interval which exceeds 0.10 seconds in 70% of cases (c) the presence of a delta wave which is the diagnostic feature. There is deformity and widening of the QRS

complex by a heavily slurred or notched initial component called the delta wave. When the QRS deflection is upright, notching or slurring occurs on the ascending limb of the deflection and when downwards, on the descending limb [3]. Secondary ST-T wave changes are present in a direction generally opposite to the major delta and QRS vectors. These patterns may mimic bundle branch block, myocardial infarction or ventricular hypertrophy [4]. In the second case, the diagnosis of ischaemic heart disease was inadvertently made due to the presence of these secondary or associated ST-T wave changes.

Approximately 50% of patients with WPW syndrome eventually experience tachycardias and 15-20% experience paroxysmal atrial fibrillaton [4]. The unpredictable episodes of atrial tachycardias make case 1, unfit for diving. An episode of tachycardia underwater could lead to unconsciousness and drowning [5,6].

Asymptomatic patients do not require empirical therapy. Supra ventricular tachycardía and atrial fibrillation should be managed with drugs and/or cardioversion. Prophylactic long term therapy is best governed by electrophysiology studies and surgical division of accessory pathways [4].

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