INTERMITTENT APPEARANCE OF PRE-EXCITATION IN THE WOLFE PARKINSON WHITE SYNDROME

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Dr Guha highlights an interesting phenomenon that is occasionally seen and is worthy of possible explanation.[1] Ventricular pre-excitation is due to an accessory pathway (AP) or electrical connection, between the atria and ventricles, which is additional to the normal atrioventricular nodal connection. Such APs comprise myocardial fibres 'draped' across the electrically insulating, left and right atrio-ventricular (AV) rings. The AP generally conducts electrical impulses from atria to ventricle more rapidly than occur over the AV-node, thus 'pre-exciting' the ventricles. Electrocardiographically this is manifest by a short PR interval and slurring of the initial phase of a relatively broad QRS complex (giving rise to the so-called delta wave), along with concomitant ST-T wave repolarisation abnormalities. This observation is seen in the population with an incidence of approximately 0·15% (the majority of individuals being asymptomatic), but where it does form the substrate for supra-ventricular tachycardia, it is often referred to as the Wolfe Parkinson White syndrome. It is usually a benign condition, though occasionally rapidly conducted, pre-excited atrial fibrillation can degenerate into ventricular fibrillation.

How could ventricular pre-excitation be intermittent, as with the case of Dr Guha's (see Figure 1)? Ventricular pre-excitation is not an 'all or none' phenomenon, and may be minimal or absent or vary from beat-to-beat, even in the presence of an AP proven at electrophysiological study. This intermittent appearance may have a number of explanations:[2]

1. It could be due to relatively rapid conduction through a 'slick' AV-node, leaving little time for pre-excitation over the AP. AV-nodal conduction is influenced by the autonomic nervous system and will be more rapid under enhanced sympathetic and reduced parasympathetic tone, such as exercise.
2. Conduction over the AP is occasionally slow; again, under such unusual circumstances the ventricles will be depolarised preferentially via the AV-node and His-Purkinje system.
3. The AP may be anatomically remote from the sinus node, e.g. positioned on the free wall of the mitral annulus; by the time the atrial depolarisation wave front reaches the AP, it may have penetrated the AV node and His-Purkinje system.
4. The refractory period of the AP (the time it takes for it to recover from depolarisation and be amenable to excitation once more) may be long enough for conduction within it to block intermittently, even from beat-to-beat or following supraventricular ectopics. Accessory pathway refractoriness is also influenced by autonomic tone, and may be prolonged at times of reduced sympathetic tone. Supraventricular and ventricular ectopic beats may partially penetrate the AP, exhibiting so-called 'concealed conduction' and reset the refractory period of the AP; conduction through the AP may then fail following the next sinus beat. Finally certain antiarrhythmic drugs may prolong AP refractoriness.

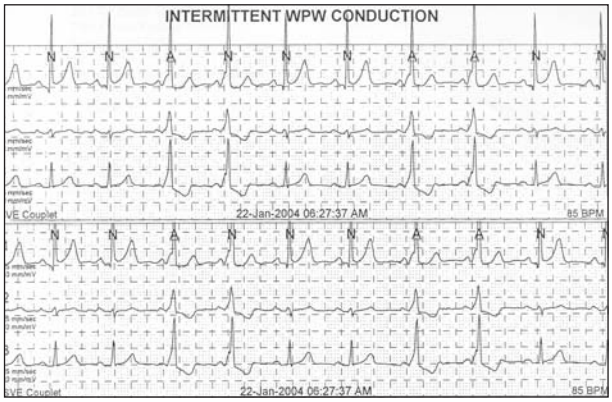

FIGURE 1
Intermittent Wolfe Parkinson White syndrome.
This ECG accompanied Dr Guha's original Letter to the Editor.[1] Note: A = aberrantly conducted beat (WPW syndrome), N = normally conducted beat.

The likely explanation in Dr Guha's patient is:

4. A long refractory time with intermittent failure in conduction through the AP, though an electrophysiological study would help to prove this. If a long refractory period in the AP is suspected or proven, such patients are said to be at low risk of a life-threatening, rapid ventricular response if atrial fibrillation supervenes.

REFERENCES

1 Guha N. Intermittent Wolfe Parkinson White syndrome (Letter to the Editor). *J R Coll Physicians Edinb* 2004; **34(2):**161.

2 Pre-excitation syndromes. In: Josephson ME. *Clinical Cardiac Electrophysiology* (3rd ed). Philadelphia: Lippincott, Williams & Wilkins; 2002; 380.