Atrial Fibrillation in the Wolff-Parkinson-White Syndrome

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A 38 year old paramedic was admitted with symptoms of palpitations. These had come on suddenly following recent cocaine use. He had previously been told he had an abnormal ECG following a US army medical assessment in his twenties. He had subsequently been told the ECG changes had normalised and was not under follow up.

He was haemodynamically stable on admission. Initial ECG is shown in figure A.

![Figure A](image1)

**Figure A:** Initial ECG (25mm/sec) demonstrating irregular, broad complex tachycardia.

A diagnosis of pre-excited atrial fibrillation (AF) was made and he underwent urgent DC cardioversion in A&E. His post cardioversion ECG is shown in figure B.

![Figure B](image2)

**Figure B:** ECG (25mm/sec) following DC cardioversion demonstrating restoration of sinus rhythm.

He underwent three wire electrophysiology study the following day. The traces are presented in order in figures C - M.
**Figure C:** Surface ECG leads II, III, V1 and V3.

His catheter, coronary sinus decapolar catheter, RV apical catheter. Basic intervals in sinus rhythm: R-R interval 988ms, A-H interval 86ms, H-V interval 34ms, QRS 92ms, QT interval 384ms.

**Beat 4:** Atrial extra-systole, distal to proximal coronary sinus activation. Broad QRS complex. Signal on distal His catheter is a right bundle branch potential (as virtually no atrial electrogram) which is later than earliest ventricular activation (negative H-V time) which is seen on surface ECG.

**Figure D:** During retrograde curve.

RV apical pacing with 600ms drive train and extra stimulus coupling interval of 440ms. Eccentric atrial activation throughout. No decrement with extra-stimulus.
Figure E: Spontaneous termination of arrow complex tachycardia.

- Tachycardia cycle length 410ms
- Narrow QRS complex on surface ECG leads
- Eccentric atrial activation pattern
- Short RP interval
- H-V interval 34ms
- Terminates with atrial beat and no His signal (AV node)
- Orthodromic atrio-ventricular re-entrant tachycardia
- Differential diagnosis: Main differential diagnosis is atrial tachycardia from left atrial origin (but termination in the AV node makes this less likely – would have to postulate AV block at the same time as the atrial tachycardia terminates which would be possible but unlikely if a repeated observation), AVNRT with leftward AVN extension, atypical AVNRT with bystander left lateral accessory pathway. These are all much less likely as the ECG suggests antegrade conduction down a left sided pathway (in pre-excited AF and with atrial ectopic), and retrograde conduction during V pacing shows conduction up a left sided pathway, with CS activation in SVT similar to V pacing.

Figure F: 12 lead ECG of orthodromic AVRT, termination of tachycardia and sinus rhythm following termination.
QRS morphology different during AVRT and in sinus beat following termination of tachycardia (Lead V2 has dominant S wave in tachycardia but not SR, leads V3 – V4 have small S waves during tachycardia which are absent with sinus beat) because during tachycardia there is no fusion and ventricle is depolarised purely through the AV node.

Beat 10: Likely atrial extrasystole with manifest pre-excitation – clear delta wave (positive in all chest leads), broad QRS complex.

Figure G: Antegrade curve from proximal C-S bipole.

Drive train 600ms, atrial extra stimulus coupling interval 340ms:
- Negative H-V time during drive train (not measured)
- Broad QRS morphology on extra-stimulus
- Equal Stim – RVA signal on drive train and extra – stimulus
- Broad QRS morphology on atrial extra – stimulus
- Possible late His signal (arrow) on His-distal bipole following atrial extra-stimulus with significantly longer A-H time than during drive train

Conclusions:
- Left sided accessory pathway present.
- ( Likely) orthodromic AVRT induced.
- During pre excited AF, Shortest Pre-Excited R-R Interval (SPERRI) of 200ms documented.
- On this basis a decision was made to ablate the accessory pathway for symptomatic and prognostic indications- this patient's presentation was with haemodynamically compromising pre-excited atrial fibrillation which was the basis for the 'prognostic' ablation in this case.

Following transeptal puncture, a 4mm Mullins catheter was used to map mitral annulus during distal C-S pacing.
During distal C-S pacing the earliest ventricular signal on the ablation catheter was 12ms prior to the onset of the delta wave. A QRS-V time of ≤ 0ms is reported as a predictor of successful ablation with a single lesion when treating manifest left free wall pathways.

Ablation at this site during C-S pacing resulted in loss of pre-excitation within 3.1s RF energy applied for 60s. Following this the retrograde curve was repeated.
Figure J: RV apical pacing following ablation of left lateral accessory pathway.

Intermittent V-A conduction during 600ms pacing (VA block on last beat, no atrial signal followed the last V paced beat). On conducted beats there is concentric atrial activation demonstrating loss of retrograde conduction of accessory pathway.

No tachycardia was inducible with ventricular extra-stimuli following 600ms and 400ms drive train.

Figure K: Following application of RF energy 12 lead ECG demonstrates different QRS morphology with dominant S wave in V2 and clear S wave in lead V3.

There is only a subtle difference between the 12 lead ECG prior to ablation of the left sided accessory pathway and post ablation (figure N).
Figure L: Comparison of 12 lead ECG leads pre and post ablation of left sided accessory pathway.

**Discussion Points**

**Latent pre-excitation**

As demonstrated in figure L, the difference between the pre-excited and non-pre-excited QRS morphology on the 12 lead ECG is marginal. A ‘latent’ accessory pathway is one where there is no evidence of pre-excitation on the ECG. When a latent pathway is present ventricular depolarisation is exclusively or almost exclusively via the atrio-ventricular node (AVN). This is typically seen with far left free wall pathways when there is rapid AVN conduction. Ventricular depolarisation via the AVN is almost complete by the time the wave of depolarisation reaches and traverses the accessory pathway.

Rapidly conducting accessory pathways may be latent, with no ECG indication to their presence. As well as being relevant to the differential diagnosis in patients with documented tachycardia and a normal 12 lead ECG, it enters into the differential diagnosis of substrate for cardiac arrest in apparently healthy patients without overt cardiac disease. Intravenous adenosine or diagnostic electrophysiology study readily confirms or excludes the presence of latent accessory pathway conduction.

**Prognostic ablation of rapidly conducting accessory pathways**

The Wolff-Parkinson-White (WPW) syndrome is that of electrocardiographic evidence of pre-excitation and symptoms of tachycardia. The widespread use of screening ECGs has resulted in the identification of a group of patients with a WPW-ECG pattern without symptoms of tachycardia. The overall prevalence of WPW-ECG changes is estimated at 1 – 3 per 1000 individuals and it is estimated that 65% of adolescents and 40% of adults over 30 with ECG evidence of pre-excitation on a 12-lead ECG are asymptomatic. Antegrade conduction down an accessory pathway is associated with an increased risk of sudden death, primarily due to rapidly conducted atrial arrhythmias degenerating into ventricular fibrillation. Identifying those individuals at greatest risk of sudden death and the most suitable method for risk stratification is an ongoing challenge.
The absence of symptoms associated with a WPW-ECG is associated with a good prognosis, on meta analysis the overall risk of sudden cardiac death of 0.05 – 0.2% per year\(^9\). Despite the favourable prognosis, sudden cardiac death is reported\(^5\) and there is great incentive to identify those at greatest risk on order to offer appropriate counselling and, where appropriate, effective treatment. When asymptomatic patients with WPW-ECG are identified, various non invasive and invasive options are available intended to assist risk stratification.

### Non-invasive risk stratification

12 lead ECG or ambulatory ECG demonstrating intermittent pre-excitation is associated with a lower incidence of re-entrant SVT (8.3%) when compared with those constant pre-excitation (23%)\(^10\) and is a predictor of poor antegrade accessory pathway conduction\(^11,15\) however there are reports of ventricular fibrillation in the context of intermittent antegrade conduction\(^13\). 12 lead ECG during atrial fibrillation allows an assessment of the fastest rate that the accessory pathway is able to conduct in the antegrade direction, the Shortest Pre-Excited R-R Interval (SPERRI). A SPERRI of below 250ms is more commonly seen in patients with WPW who have experienced cardiac arrest with a stronger association seen with a SPERRI below 220ms\(^14,15\). 

Exercise testing has been employed to non-invasively assess the antegrade characteristics of an accessory pathway. During exercise testing, only abrupt loss of pre-excitation on 12 lead ECG was associated with long antegrade Accessory Pathway Effective Refractory Period (APERP)\(^16\) but the persistence of pre-excitation is associated with a low positive predictive value in the identification of those patients with SPERRI < 250ms or APERP < 250ms\(^17\). In addition the exercise test is compromised by inter-observer variability and its usefulness reduced by the small proportion of patients in whom abrupt loss of the delta wave is identified\(^5\).

### Invasive risk stratification

Invasive electrophysiological (EP) assessment of accessory pathway conduction properties may be offered for prognostic or symptomatic reasons. In the symptomatic patient, the benefits of an EP study would be the potential treatment of the cause of their symptoms as well as to allow an invasive assessment of the antegrade conduction characteristics of the accessory pathway. In the context of previous documentation of AF with a SPERRI below 250ms\(^5\), the primary aim of the EP study is to locate the accessory pathway prior to ablation, as the SPERRI itself is the adverse prognostic feature indicating ablation. An EP study in the asymptomatic patient would be offered purely for prognostic reasons. In the asymptomatic patient whose ECG abnormality is identified incidentally, current guidelines recommend initial risk stratification with ambulatory monitoring and exercise testing. In those in whom lower risk features (intermittent pre-excitation or abrupt loss of antegrade accessory pathway conduction) are not identified, invasive risk stratification with an EP study is recommended. At EP study, a SPERRI during AF below 220 – 250ms is the best discriminator of those at risk of ventricular fibrillation\(^6,7,18\). APERP is a less powerful predictor of life threatening events\(^6,7\).

### Indication for catheter ablation

The decision regarding whether to proceed to catheter ablation in the asymptomatic patient is a challenging one. While in general considered to carry a low level of risk, complications are reported to occur following catheter ablation of accessory pathways in around 2% of cases and may include venous thrombosis, thromboembolism, infection and atrioventricular block\(^19,20\).

Current guidelines suggest that the decision to proceed to ablation should be made in the context of a frank discussion with the patient regarding the potential risks and benefits of catheter ablation versus conservative management\(^5\). Some authors have called for a more aggressive approach to the use of catheter ablation in asymptomatic individuals and provided evidence of a higher adverse event rate in the ‘asymptomatic’ group than previously reported\(^21\). In Pappone et al’s study, 13 out of 550 (2.4%) asymptomatic patients experienced cardiac arrest during a median follow up of 22 months\(^21\). Other experts, however, highlight the development of symptoms in the majority of those patients in this study prior to their development of a potentially catastrophic arrhythmia\(^22\) and the overall excellent prognosis
of even those experiencing ventricular fibrillation (VF) during the course of the study, in which there were no reported deaths or adverse neurological consequences in those experiencing VF\textsuperscript{21}. While this most recent contribution to the literature provides important new information contributing to ongoing debate, it does not resolve the debate regarding the optimal strategy to identify and treat those at risk of sudden cardiac death.

At present, the absence of symptoms continues to represent a favourable feature in the overall risk stratification of asymptomatic patients with a WPW-ECG\textsuperscript{22}. For asymptomatic patients with a SPERRI below 250ms, consideration of catheter ablation for prognostic reasons is justified, with a class IIa level of evidence\textsuperscript{5}, should the patient decide that they wished to pursue this, when they have been fully informed of the relative risks and benefits of doing so.

References


