

Atrial Fibrillation in Wolff Parkinson White (WPW) Syndrome: Case Report and Review of the Literature

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Abstract

The occurrence of atrial fibrillation (AF) transmitted to the ventricles by an accessory pathway is a rare, potentially serious arrhythmia. The risk of ventricular fibrillation is a fatal complication that can be increased by certain medications such as digitalis. AF can be the initial presentation of WPW syndrome, in which case it is often poorly tolerated. We report the case of a 42-year-old patient with no particular history, who consulted for palpitations that appeared at rest, a few hours previously; systolic blood pressure was 80 mm Hg without signs of shock. The ECG showed rapid AF with wide QRS. After reduction by external electric shock, the post-ictal ECG was in favor of WPW syndrome. The initial assessment was quickly carried out, and the patient was treated with a combination of two antiarrhythmic drugs. The occurrence of rhythm disturbances requires an etiological assessment on which the specific treatment will depend. In WPW syndrome, stratification of the individual risk of sudden death is essential in the therapeutic management of patients.

Keywords: FA, Super Wolff, Individual risk

Introduction

In 1893, Stanley Kent describes connection pathways connecting the ventricles to the atria by linking them to a physiological situation [1]. These tracts later received the name Kent, since in 1930 the syndrome was described by doctors Wolff, Parkinson and White [2,3] but isolated cases were previously published as early as 1915 [4]. In 1943 the relationship between the syndrome and the accessory pathway was mentioned [5]. There sudden death, by ventricular fibrillation, was suspected as early as 1971 [5,6]. The first electrophysiological studies date from the end of the 1960s, establishing the mode of beginning and end of paroxysmal tachycardias by a reentry circuit [7,8].

WPW syndrome is defined by the presence of a patent accessory pathway (Figure 1) leading to ventricular pre-excitation on the ECG, associated with recurrent tachyarrhythmias. It is most often expressed in young patients with healthy hearts. The search for

a structural cardiac anomaly must be systematic by performing a cardiac Doppler echo and/or cardiac MRI. The ECG associates a short PR interval (less than 120 ms), an initial thickening of the QRS foot (Delta wave), and a widening of the QRS complex (Figure 1). Secondary abnormalities of repolarization are common.

The treatment initially consisted of surgical ablation of the accessory pathway with initial success described in 1968 [9]. Since the 1980s, there have been several attempts to ablate the Kent bundle intracavitarily, first by high-energy electric shock [10], then by radiofrequency at the end of these same years [11].

The occurrence of atrial fibrillation transmitted to the ventricles by the accessory bundle remains among the arrhythmias, fortunately rare, but more serious due to the very high risk of sudden death secondary to ventricular fibrillation (VF) faced with such a picture, support must be early and immediate.

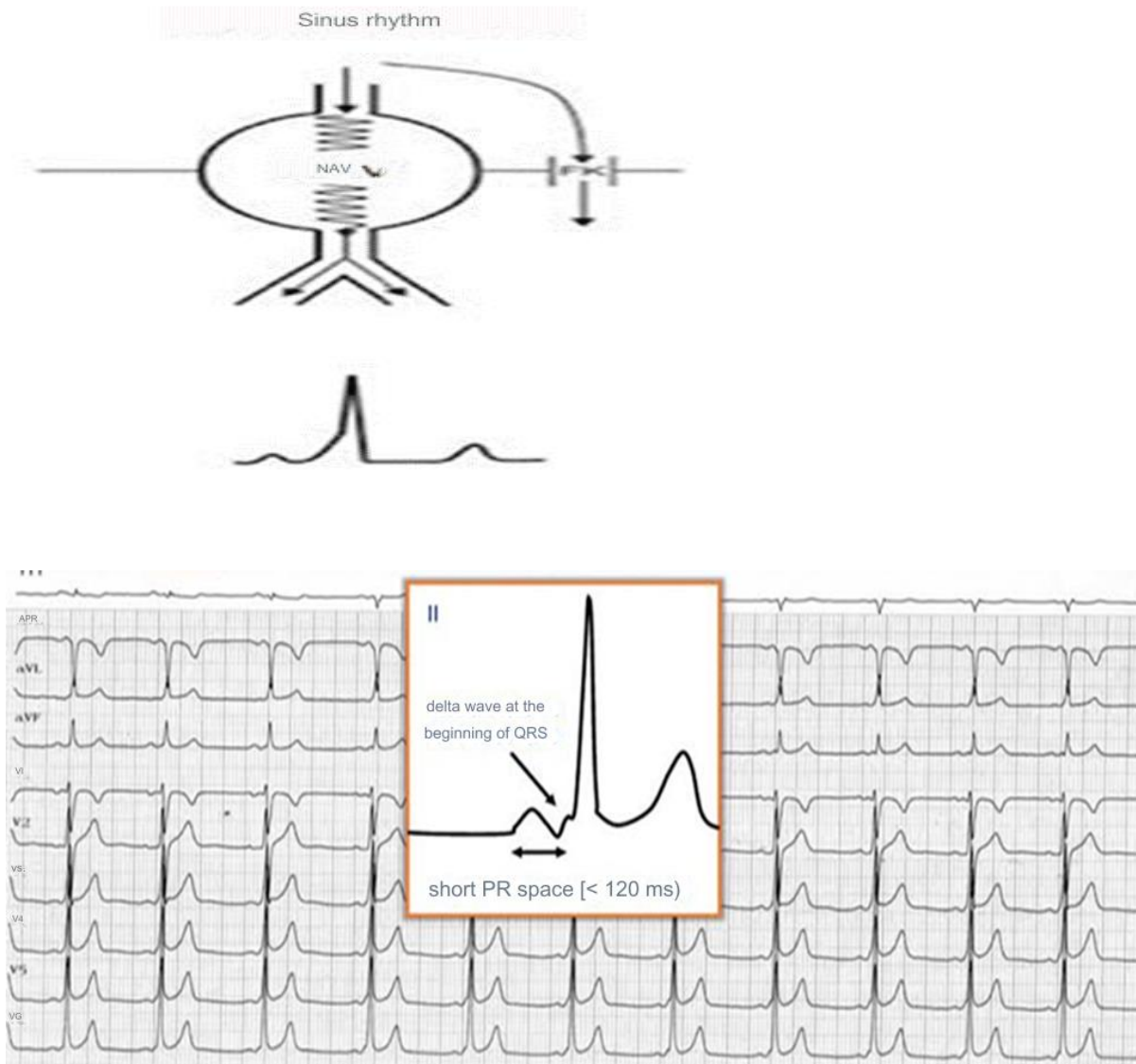


Figure 1: (A) Accessory pathway, Kent bundle, connecting the atria to the ventricles, bypassing the atrioventricular node (AVN). (B) ECG of an accessory pathway: short PR, delta wave leading to impingement of the ascending branch of the R wave [23].

We present the case of Mr. NR aged 42, without any particular personal or family history, who consults for a first episode of palpitations occurring at rest. On admission, the patient was hemodynamically stable; PA=130/80, irregular pulse at 170bpm.

The remainder of the examination is unremarkable. The ECG showed an irregular accordion-shaped wide QRS tachycardia, the average ventricular rate was 170 cycles per minute (Figure 2).

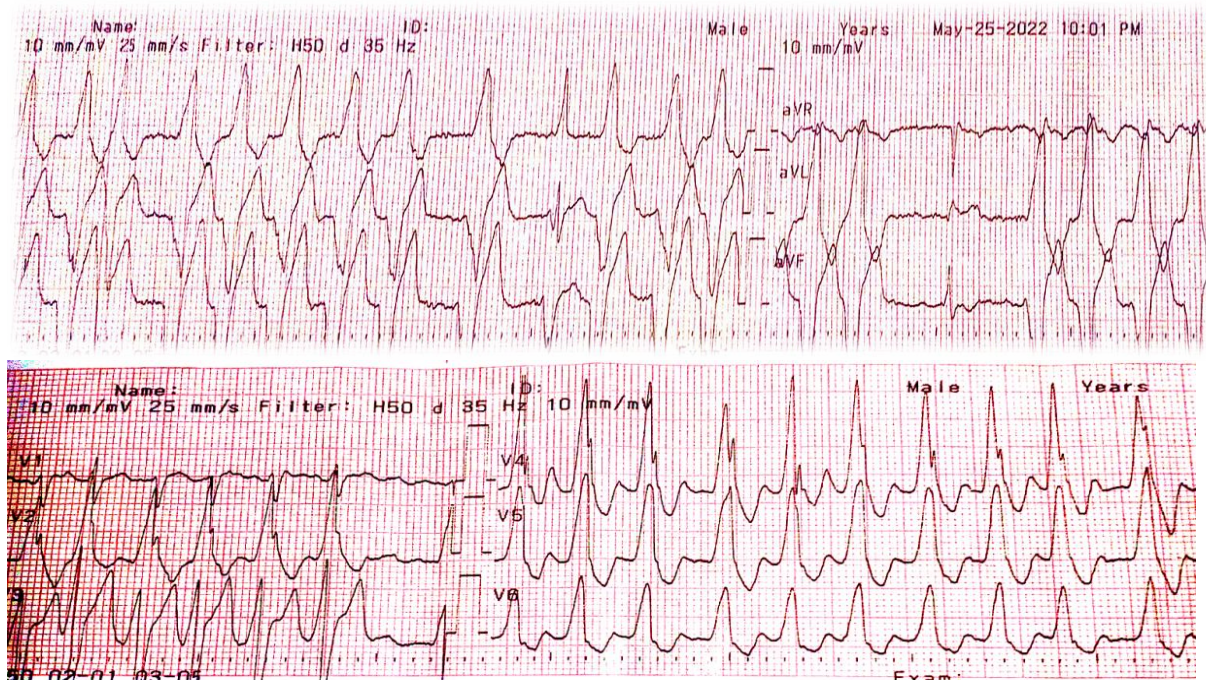


Figure 2: ECG recorded intracritically.

On transthoracic echocardiography, the systolic function of the left ventricle is preserved: ejection fraction at 61%, absence of cavitory dilatation, absence of valvulopathy, absence of LV hypertrophy. Absence of pulmonary hypertension, absence of signs pointing to Ebstein's disease.

Biology: The blood count, renal assessment and blood ionogram did not show any abnormalities.

Initial Support Measures were quickly initiated, combining electrocardiographic and blood pressure monitoring, an approach and external electrical cardioversion at 150 joules after brief sedation.

The Post Cardioversion ECG (Figure 3) highlights a regular sinus rhythm at 66 cycles per minute, with the presence of pre-excitation, suggesting a left posterior septal localisation of the accessory bundle.





Figure 3: ECG recorded post-critically: Left posterior septal accessory bundle.

The maintenance treatment was introduced combining flecainide with bisoprolol, without anticoagulation given that the CHA2DS2-VASc score was zero. The patient subsequently benefited from ablation of the accessory pathway by radio frequency.

Discussion

Paroxysmal tachycardia with fine QRS, by orthodromic reciprocal rhythm, is the most common arrhythmia, 95% of cases (Figure 4). Wide QRS tachycardias can correspond either to an orthodromic tachycardia with bundle branch block, or to an antidromic tachycardia, or to atrial fibrillation or atrial flutter with conduction through the accessory pathway (Figures 5,6).

The occurrence of atrial fibrillation with rapid and wide ventriculograms (Figure 6), (major pre-excitation) alternating with thin QRS or fusion, exposes the risk of ventricular fibrillation in the event of an accessory bundle with a short refractory period [14] or multiple beams, or in case of septal location [15] or right from the accessory pathway; this risk is increased by pre-existing heart disease. Electrophysiological exploration makes it possible to assess this risk.

Several elements allow the diagnosis of AF associated with an accessory bundle: the absence of a typical appearance of right or left bundle branch block, on the 12-lead ECG during the arrhythmia (Figure 5), the presence in the precordial leads of a positive concordance which excludes a bundle branch block and the extremely rapid ventricular response argue in favor of conduction by the accessory pathway having a very short refractory period, unlike that of the atrioventricular node whose properties rarely allow a ventricular response greater than 200 cpm. The

almost exclusive passage through the accessory pathway during AF explains the widening of the QRS and its atypical morphology with a very rapid ventricular rate. Hemodynamic tolerance is often poor and the risk of VF degeneration is significant.

The management of Wolff-Parkinson-White syndrome was the subject of the publication of European recommendations, in 2019 [12] and American, in 2015 [13]. Emergency treatment uses, depending on the case, vagal maneuvers, antiarrhythmic drugs, external electric shock in poorly tolerated forms, or transesophageal stimulation (Figure 7).

Subsequent management depends on the type of arrhythmia, its tolerance, the patient, and the electrophysiological characteristics of the atrioventricular accessory pathways. Antiarrhythmics represent an alternative to ablation in cases of asymptomatic or symptomatic WPW at low risk. Radiofrequency ablation is offered to patients with symptomatic Wolff-Parkinson-White syndrome despite drug treatment and those who are considered “at risk” of sudden death [14]. If the radiofrequency ablation of the accessory route is successfully carried out, the subject is sometimes considered cured [15,16].

It should be noted that drugs that slow conduction through the AVN are ineffective in interrupting tachycardia, and are even dangerous, because they risk triggering VF by promoting atrioventricular conduction through the AV.

These medications should be avoided. Only drugs that can restore the RS and block conduction in the accessory pathway are useful, such as Flecainide. The use of Ibutilide or Procainamide is also possible [17-22].

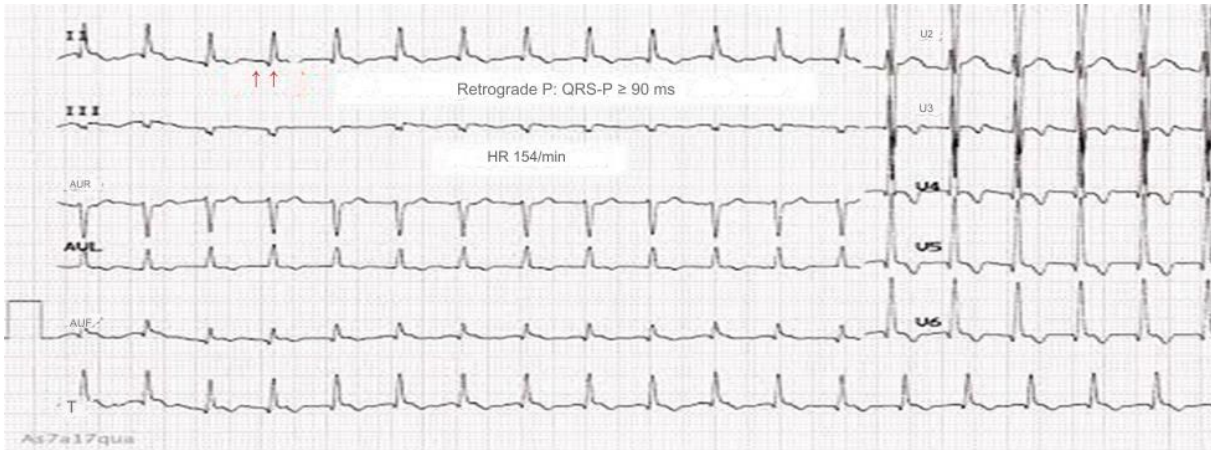
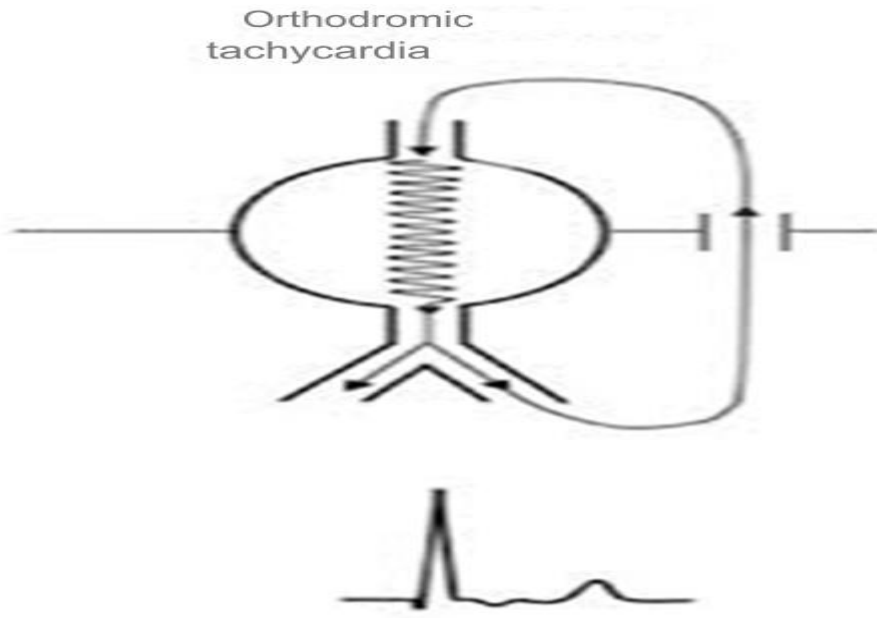


Figure 4: Orthodromic tachycardia follows the AVN in this case the QRS are thin with retrograde activation of the atria (P' retrograde) [23].

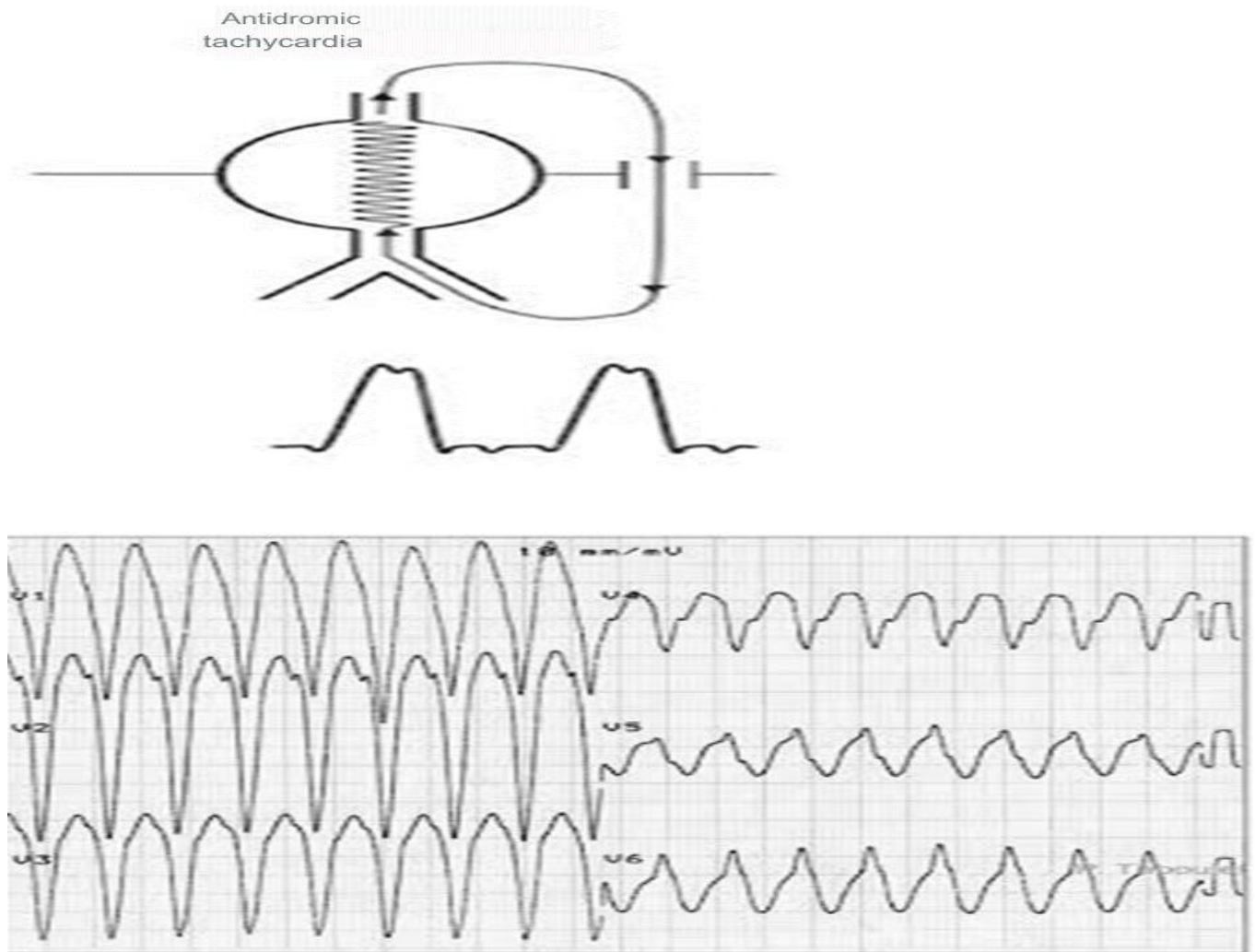


Figure 5: Antidromic tachycardia using the accessory pathway, in this case the QRS are wide [23].

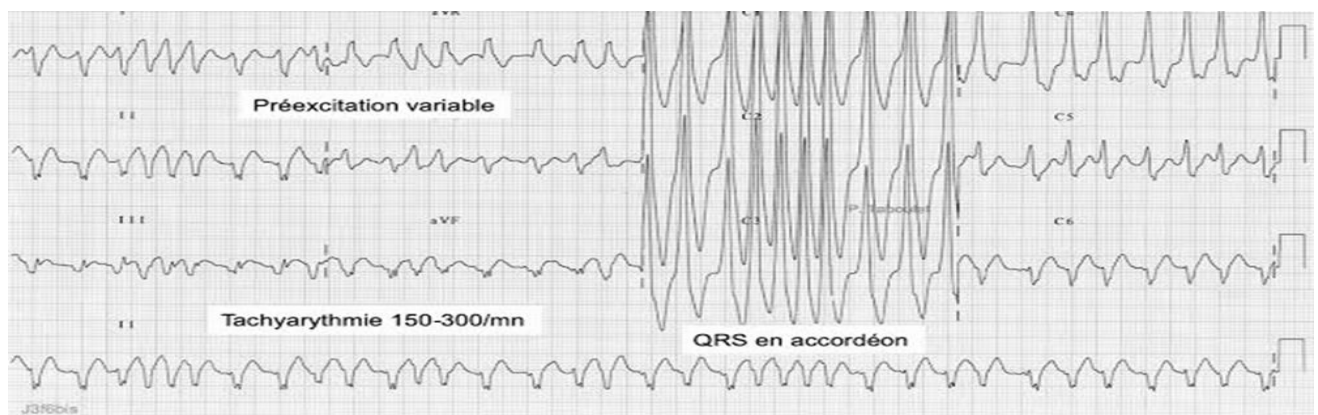


Figure 6: Super Wolff: Atrial fibrillation with accordion QRS appearance [23].

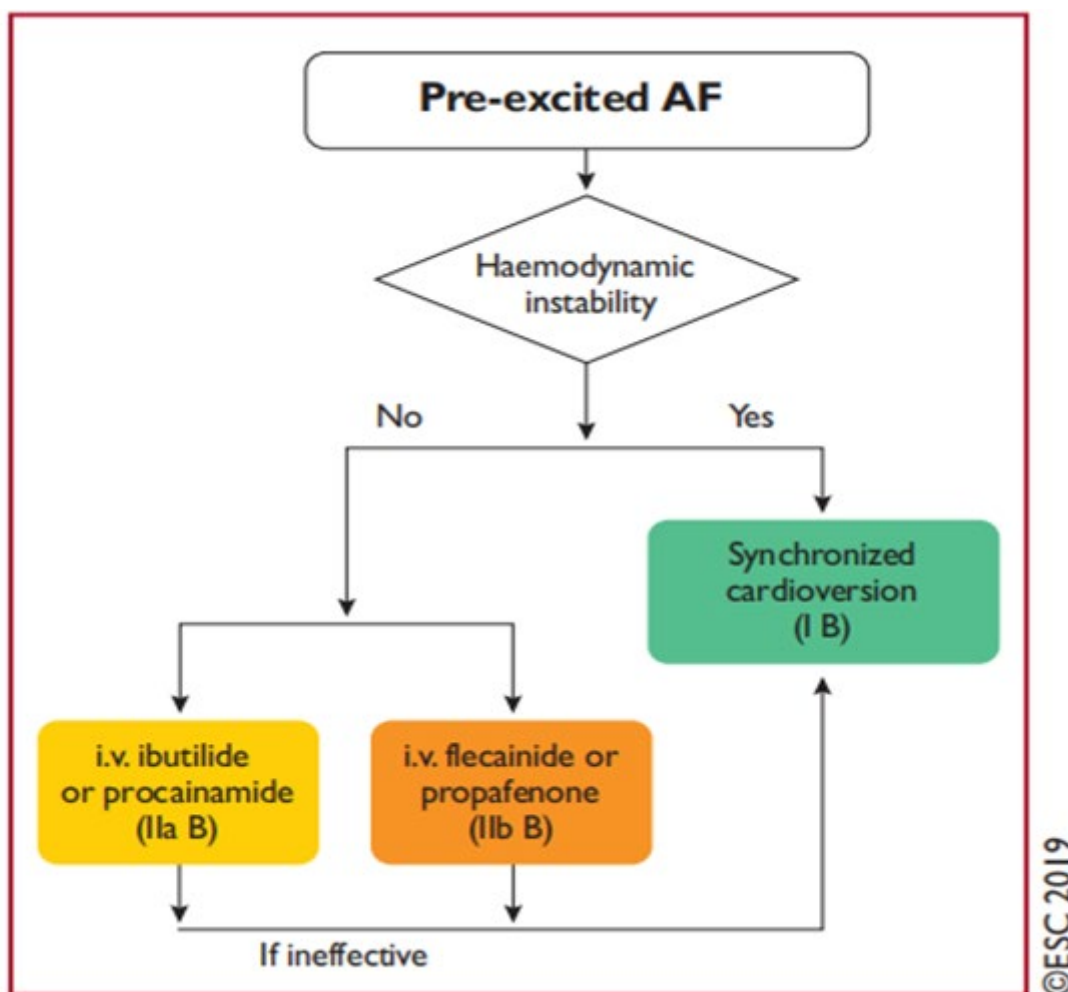


Figure 7: Acute therapy of pre-excited atrial fibrillation. AF=atrial fibrillation; Iv=intravenous.

Conclusion

The occurrence of AF during WPW syndrome can be life-threatening in the short term. It is a prognostic factor for sudden death. The electrocardiographic presentation can be misleading and wrongly direct towards the prescription of beta-blockers or digitalis which are potentially dangerous in this context. This should be considered in the face of any wide QRS tachycardia in a young subject. Removal of the accessory pathway in question must be carried out as quickly as possible to prevent sudden death and recurrence.

References

1. AFS Kent (1893) Researches on the structure and function of the mammalian heart. *J Physiol.* 14:233.
2. Scheinman MM (2005) History of Wolff-Parkinson-White syndrome. *Pacing Clin Electrophysiol* 28:152-156.
3. Wolff L, Parkinson J, White PD (1930) Bundle-branch block with short PR interval in healthy young people prone to paroxysmal tachycardia. *Am Heart J* 5:685-704.
4. FN Wilson (1915) A case in which the vagus influenced the form of the ventricular complex of the electrocardiogram. *Arch Intern Med* 16:1008-1027.
5. Wood FC, Wolferth CC, Geckeler GD (1943) Histologic demonstration of accessory muscular connections between auricle and ventricle in a case of short PR interval and prolonged QRS complex. *Am Heart J* 25:454-462
6. Dreifus LS, Haiat R, Watanabe Y, Arriaga J, Reitman N (1971) Ventricular fibrillation: a possible mechanism of sudden death in patients and Wolff-Parkinson-White syndrome. *Circulation* 43:520-527.
7. Durrer D, Schoo L, Schuilenburg RM, et al. (1967) The role of premature beats in the initiation and the termination of supraventricular tachycardia in the Wolff-Parkinson-White syndrome. *Circulation* 36:644-662.
8. Wellens HJ, Schuilenburg RM, Durrer D (1971) Electrical stimulation of the heart in patients with Wolff-Parkinson-White syndrome, type A. *Circulation* 43:99-114.
9. Cobb FR, Blumenschein SD, Sealy WC, et al. (1968) Successful surgical interruption of the bundle of Kent in a patient with Wolff-Parkinson-White syndrome"[archive] *Circulation* 38:1018-1029.
10. Fisher JD, Brodman R, Kim SG et al. (1984) Attempted nonsurgical electrical ablation of accessory pathways via the coronary sinus in the Wolff-Parkinson-White syndrome. *J Am*

Coll Cardiol 4:685-694.

11. Borggrefe M, Budde T, Podczeck A, et al. (1987) High frequency alternating current ablation of an accessory pathway in humans. *J Am Coll Cardiol* 10:576-582.
12. Brugada J, Katritsis DG, Arbelo, et al. (2019) ESC Guidelines for the management of patients with supraventricular tachycardia The Task Force for the management of patients with supraventricular tachycardia of the European Society of Cardiology (ESC): Developed in collaboration with the Association for European Pediatric and Congenital Cardiology (AEPC). *Eur Heart J* 41:655-720.
13. RL, Joglar JA, Caldwell MA, et al. (2015) ACC/AHA/HRS Guideline for the Management of Adult Patients With Supraventricular Tachycardia: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *Circulation* 133:e506-e574.
14. Bromberg BI, Lindsay BD, Cain ME, Cox JL (1996) Impact of clinical history and electrophysiologic characterization of accessory pathways on management strategies to reduce sudden death among children with Wolff-Parkinson-White syndrome. *J Am Coll Cardiol* 27:690-669.
15. Gaita F, Giustetto C, Riccardi R, Mangiardi L, Brusca A (1989) Stress and pharmacologic tests as methods to identify patients with Wolff-Parkinson-White syndrome at risk of sudden death. *Am J Cardiol* 64:487-490.
16. Pappone C, Vicedomini G, Manguso F, et al. (2012) Risk of malignant arrhythmias in initially symptomatic patients with Wolff-Parkinson-White syndrome: results of a prospective long-term electrophysiological follow-up study. *Circulation* 125:661-668.
17. Timmermans C, Smeets JL, Rodriguez LM, Vrouchos G, Van den Dool A, et al. (1995) Aborted sudden death in the Wolff-Parkinson-White syndrome. *Am J Cardiol* 76:492-494.
18. Harahsheh A, Du W, Singh H, Karpawich PP (2008) Risk factors for atrioventricular tachycardia degenerating to atrial flutter/fibrillation in the young with Wolff-Parkinson-White. *Pacing Clin Electrophysiol* 31:1307-1312.
19. Triedman J, Perry J, Van Hare G (2005) Risk stratification for prophylactic ablation in asymptomatic Wolff-Parkinson-White syndrome. *N Engl J Med* 352:92-99
20. Pappone C, Santinelli V, Manguso F, et al. (2003) A randomized study of prophylactic catheter ablation in asymptomatic patients with the Wolff-Parkinson-White syndrome. *N Engl J Med* 349:1803-1811.
21. Antz M, Weiss C, Volkmer M, et al. (2002) Risk of sudden death after successful accessory atrioventricular pathway ablation in resuscitated patients with Wolff-Parkinson-White syndrome. *J Cardiovasc Electrophysiol* 13:231-236.
22. Katritsis DG, Boriani G, Cosio FG, Hindricks G, Jaïs P, et al. (2017) European Heart Rhythm Association (EHRA) consensus document on the management of supraventricular arrhythmias, endorsed by Heart Rhythm Society (HRS), AsiaPacific Heart Rhythm Society (APHRs), and Sociedad Latinoamericana de Estimulación Cardíaca y Electrofisiología (SOLAECE). *Europace* 19(3):465-511.
23. ECG from A to Z, Pierre Taboulet

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