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## Atrial fibrillation and preexcitation - A licence to kill

### A B S T R A C T

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Atrial fibrillation becomes a potentially lethal arrhythmia in the presence of preexcitation because the rapid ventricular activation can result in ventricular fibrillation. Fortunately, radiofrequency ablation is an effective treatment for these patients. Specific points of interest regarding this association are the mechanism of increased incidence of atrial fibrillation and the current management of patients presenting in atrial fibrillation. These are discussed in this editorial.

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During atrial fibrillation (AF), the chaotic atrial activation results in multiple impulses bombarding the atrioventricular conduction system. Normally, the atrioventricular node forms part of this conduction system and its electrophysiological properties ensure that the ventricular rate remains controlled. In patients with an accessory pathway that conducts in the antegrade direction, this safeguard is lost and, especially when the pathway has a short refractory period, rapid conduction to the ventricle can result, giving AF a licence to kill. In this issue, Debasis Acharya et al. describe the acute outcomes and follow up in patients with preexcitation and AF and it is heartening that radiofrequency ablation is an effective therapy with high acute success rate and very low rate of recurrence [1]. However several aspects of this curious association remain of great interest, two specifically being the underlying mechanism of AF in this condition and the management of the patient presenting with AF and preexcitation with a rapid ventricular rate.

While preexcitation is benign for the most part, occurrence of sudden death in some patients was recognised early [2]. It was also recognised that this was likely due to ventricular fibrillation [3]. Atrial fibrillation with rapid conduction to the ventricle was understood as the precipitating cause and this transformation was also documented in some cases. Thus, the mechanism of sudden death in these patients was understood, but the reason for the predisposition to develop AF remained a mystery. It was shown that with programmed stimulation, AF was more commonly induced in those with manifest preexcitation than in those with concealed pathways or those without preexcitation, and even in these, it was more often induced when there was a prior history of AF [4]. AF may develop from degeneration of atrioventricular reentrant tachycardia (AVRT), as also documented in this series, and this suggests that the rapid atrial activation during tachycardia and atrial stretch from the short ventriculo-atrial interval may contribute. This, however, does not explain the higher incidence in comparison

to other forms of supraventricular tachycardia. What appears most plausible is alteration of electrophysiological properties in the atrium due to the presence of an accessory pathway. Even before the advent of radiofrequency ablation, it was noted that AF was cured by surgical treatment of accessory pathways [5]. Similar findings have been noted with catheter ablation. However, long term registry data shows that adult patients with preexcitation may have a higher propensity to develop atrial fibrillation life long, and this may not decrease with ablation, suggesting the presence of some form of atrial myopathy in these patients.

In patients presenting with atrial fibrillation and rapid conduction to the ventricle over an accessory pathway, the preferred approach to management is direct current cardioversion, especially when there is associated hemodynamic instability. However, when the patient is stable, an attempt is made sometimes to avoid cardioversion and treat the patient with drugs. It is well understood that drugs that slow atrioventricular nodal conduction like beta-blockers, calcium-blockers or adenosine, which are useful in many other supraventricular arrhythmias are to be avoided in this situation. In fact, the first documented conversion of atrial fibrillation to ventricular fibrillation occurred after the patient was treated with propranolol and digoxin [6]. Intravenous procainamide or ibutilide are the recommended agents to use in this situation, but amiodarone is often used because of more widespread availability and familiarity of physicians with the drug. Intravenous amiodarone can convert atrial fibrillation to sinus rhythm and this was the reason for recommending this in the past. However, current guidelines recommend against using amiodarone because of reports of ventricular fibrillation in a few patients with administration of the drug. Amiodarone, especially when given intravenously, may have potent beta-blocking and calcium-channel blocking effects which can prove dangerous when atrial fibrillation conversion is not achieved. Therefore it is prudent to avoid the use of intravenous amiodarone in these patients.

There still remains much to be understood about this fascinating combination of conditions, but what is clear at present is that cardioversion remains the best approach to manage these patients

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acutely while radiofrequency ablation is an excellent long term solution, with a high success rate in curing this potentially lethal condition.

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