

Atrioventricular node ablation: patient monitoring and pacing rate adjustment might be needed

Sok-Sithikun Bun*, Decebal Gabriel Latcu, and Nadir Saoudi

Department of Cardiology, Princess Grace Hospital, Pasteur Avenue, Monaco (Principality)

*Corresponding author. Tel: + 37797989771; fax: +377 97989732, E-mail address: sithi.bun@gmail.com

A 60-year-old patient with tachycardia-induced cardiomyopathy (fast permanent atrial fibrillation with a mean heart rate of 130 b.p.m.) experienced ventricular fibrillation 2 h after atrioventricular node radiofrequency ablation and biventricular pacemaker implantation, despite a pacing rate at 90 b.p.m. No arrhythmia recurrence was observed after programming the device at 110 b.p.m.

A 60-year-old male patient, treated for mild arterial hypertension, was referred to our institution for highly symptomatic long-standing persistent atrial fibrillation ($\text{CHA}_2\text{DS}_2\text{VAS}_c = 1$) after a previous failed radiofrequency biatrial catheter ablation procedure. A fast ventricular response was present despite β -blocker therapy (the mean heart rate of 130 b.p.m. on 24-h Holter despite Atenolol 100 mg/day), and the patient developed tachycardia-induced cardiomyopathy [left ventricular (LV) ejection fraction 45%] and heart failure (NYHA Class III). He also received an angiotensin-converting enzyme-inhibitor (Ramipril 5 mg/day). A biventricular pacemaker was successfully implanted (Evia HF-T, Biotronik). The day after the implantation, atrioventricular node (AVN) ablation was performed. The pacing rate was programmed at 90 b.p.m. Paced QT interval was measured at 460 ms.

Two hours after ablation, the patient developed frequent premature ventricular contractions (PVCs) with bigeminy (coupling interval of 400 ms), and finally ventricular fibrillation (VF) (Figure 1A) requiring urgent external cardioversion. Coronary angiogram performed after the episode was normal. Serum potassium level was normal before ablation, and after defibrillation. The pacing rate was then increased to 110 b.p.m. (Figure 1B) during 1 week with high-dose β -blocker continuation with rapid disappearance of the PVCs after 24 h. The pacing rate at discharge (after 9 days of hospital stay) was finally set at 100 b.p.m. for the first month. Device remote monitoring was used to assess for any recurrent ventricular arrhythmia. β -Blocker treatment was progressively stopped with resting pacing rate diminution. No ventricular arrhythmia (zero PVC recorded on the pacemaker automatic counter) was observed after 1-year follow-up, the LV function completely recovered and the patient became asymptomatic.

Discussion

Haemodynamic collapse occurring after AVN ablation can be explained by several mechanisms: cardiac perforation/tamponade after a cardiac catheterization procedure, ventricular lead dislocation in a newly pacemaker-dependent patient, inappropriate pacing on T wave with the device, direct pro-arrhythmic effects of LV pacing itself (see below),¹ and finally torsades de pointes due to a bradycardia-dependent prolongation in the QT interval. A phase of electrical instability due to an initial and then slow adaptation of repolarization has been attributed to the reduction in the outward I_{to} potassium channel and an enhanced I_{CaL} influx.²

The direct pro-arrhythmic effect of LV epicardial pacing itself has been reported and attributed to different mechanisms: reversal of typical activation sequence delaying endocardial depolarization/repolarization and increasing transmural dispersion of repolarization (with facilitation of re-entrant arrhythmias), increased QT interval and R-on-T PVCs, and finally modulation of autonomic tone.¹ It has been observed that arrhythmias occurring soon after cardiac resynchronization therapy implantation were resolved by turning LV pacing off. In contrast with previous cases described in the literature, our patient underwent AVN ablation. A pro-arrhythmic effect of LV pacing may have been involved in our patient, and it would have been interesting to observe arrhythmias termination with right ventricular pacing only. Nevertheless, the fact that the arrhythmias resolved by faster ventricular pacing may exclude this mechanism.

There is no clear cut-off value concerning the minimal pacing rate after AVN ablation. It is of common use to programme the pacing rate at 90 b.p.m. after previous large studies that compared two strategies (75 vs. 90 b.p.m. in the first one, and 60 vs. 90 b.p.m. in the second one) in which a higher proportion of events was observed in the lower paced rate group in both studies.^{3,4} The ideal duration of high pacing rate after AVN ablation is also not known. The only available argument for a short duration of the fast pacing period is the rapid normalization of the QT/RR relation after AVN ablation, e.g. after the second day. Other authors reported a strategy of higher rate pacing during 1 week associated with in-hospital monitoring for 3 days, which has shown efficient in significantly decreasing the occurrence of post-procedural malignant ventricular arrhythmias.² Thus, 1 week should be the minimal time period for higher pacing rate after AVN ablation, although many electrophysiologists prefer to extend this period for a month.³ A sufficient in-hospital post-procedural monitoring after AVN ablation is mandatory, as most ventricular arrhythmias have been reported from day 1 to day 7 after the intervention. This monitoring phase after AVN ablation should be prolonged according to the presence of risk factors known for increasing the risk of malignant arrhythmias. One of the highest risk factors is the presence of congestive heart failure (as presented by our patient) or impaired LV function.²

Rate control was hardly achieved in our patient in whom AF alternated with atrial flutter. Even though the patient started to develop a tachycardia-induced cardiomyopathy, the choice was made to favour a strong β -blocker agent on AVN (Atenolol), in comparison with those usually used in heart failure.

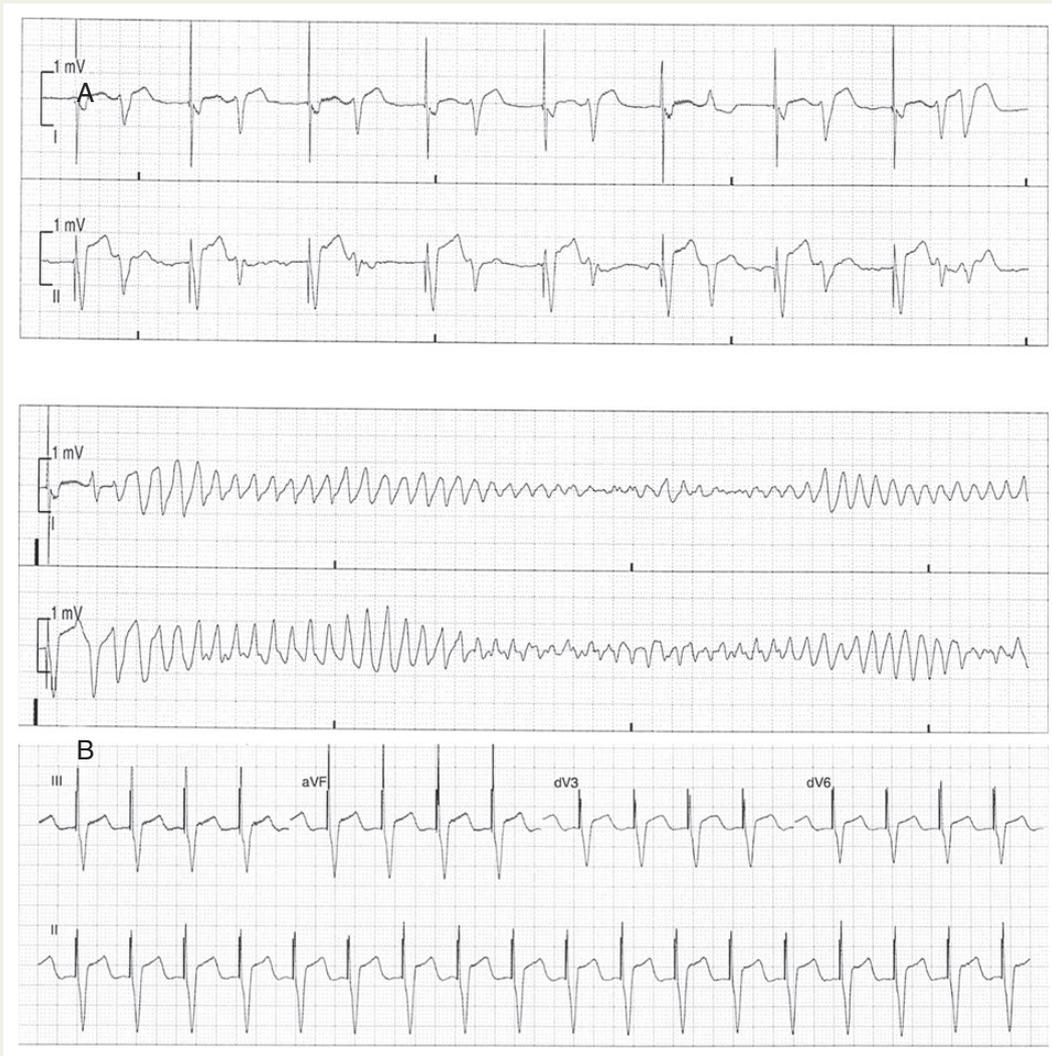


Figure 1 Ventricular fibrillation occurring two hours after atrioventricular node ablation in a patient with a biventricular pacemaker programmed at 90 b.p.m. (A). Disappearance of any premature ventricular contraction after programming the device at 110 b.p.m. (B).

No study specifically assessed the paced QT/RR relations in correlation with the mean RR intervals before the AVN ablation. We think that a particularly high mean rate pre-ablation may favour VF occurrence. The later may be prevented by further increase in the post-ablation pacing rate. The lack of data supporting the identification of an appropriate post-ablation pacing rate as a function of the pre-ablation ventricular rate leads us to suggest a pacing rate of 20 b.p.m. below the average heart rate pre-ablation. This proved appropriate in this case but needs further evaluation.

Conclusion

Ventricular fibrillation may occur after AVN ablation even in spite of 'standard' programming at 90 b.p.m. This suggests that pacing rate adjustment might be needed, particularly in case of pre-ablation persistent fast ventricular rate despite drug therapy.

Conflicts of interest: none declared.

References

1. Basu Ray I, Fendelander L, Singh JP. Cardiac resynchronization therapy and its potential proarrhythmic effect. *Clin Cardiol* 2007;**30**:498–502. Review.
2. Nowinski K, Gadler F, Jensen-Urstad M, Bergfeldt L. Transient proarrhythmic state following atrioventricular junction radiofrequency ablation: pathophysiologic mechanisms and recommendations for management. *Am J Med* 2002;**113**:596–602. Review.
3. Geelen P, Brugada J, Andries E, Brugada P. Ventricular fibrillation and sudden death after radiofrequency catheter ablation of the atrioventricular junction. *Pacing Clin Electrophysiol* 1997;**20**:343–8.
4. Wang RX, Lee HC, Hodge DO, Cha YM, Friedman PA, Rea RF et al. Effect of pacing method on risk of sudden death after atrioventricular node ablation and pacemaker implantation in patients with atrial fibrillation. *Heart Rhythm* 2013;**10**:696–701.