Suppression of Atrial Fibrillation Following Successful Ablation of Atrioventricular Nodal Reentrant Tachycardia: A Case Report

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Inducible atrioventricular nodal reentrant tachycardia was demonstrated by electrophysiological studies in a 55-year-old female who suffered from intermittent palpitation, in which paroxysmal atrial fibrillation (AF) was consistently documented by electrocardiogram recordings. After ablation of the slow pathway, the atrioventricular nodal reentrant tachycardia and AF were not inducible. During 2 years of follow-up, there were no recurrences of AF in terms of symptoms or findings from Holter electrocardiograms. We suggest that the AF was triggered by the atrioventricular nodal reentrant tachycardia and the successful ablation of atrioventricular nodal reentrant tachycardia was associated with freedom arising from ablation of AF.

Key Words: atrial fibrillation, atrioventricular nodal reentrant tachycardia
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Atrioventricular nodal reentrant tachycardia (AVNRT) has been reported to be a trigger for the induction of atrial fibrillation (AF) via the mechanism of "reentrant–mother rotor" [1]. We present a patient with paroxysmal AF. Typical AVNRT and AF were inducible during electrophysiology studies. The paroxysmal AF was no longer inducible after ablation of the slow pathway and it has not recurred during 2 years of follow-up.

Case Presentation

A 55-year-old female patient suffered from several episodes of sudden onset of palpitation and near syncope sensation. The electrocardiograms performed at the emergency department during each attack of palpitation consistently demonstrated AF (Figure 1). The AF could be converted to normal sinus rhythm by intravenous amiodarone. She also suffered from hypertension and had a history of cerebral vascular disease. No significant organic heart disease could be demonstrated by physical examination or echocardiography. She was referred for catheter ablation because of her symptomatic paroxysmal AF, which was difficult to control by drugs. Three quadripolar electrode catheters were introduced into the right femoral vein and advanced to the high right atrium (HRA), right atrioventricular junction and the right ventricular apex, respectively, for recording and stimulation. A decapolar catheter was positioned from the internal jugular vein into the coronary sinus. The effective refractory periods of the antegrade fast and slow pathway of the atrioventricular node, and the HRA at a pacing cycle length of 600 ms, were 300 ms, <260 ms and 240 ms, respectively. The common slow-fast AVNRT with a cycle length of 260 ms could be induced from the HRA by incremental atrial pacing or by double premature

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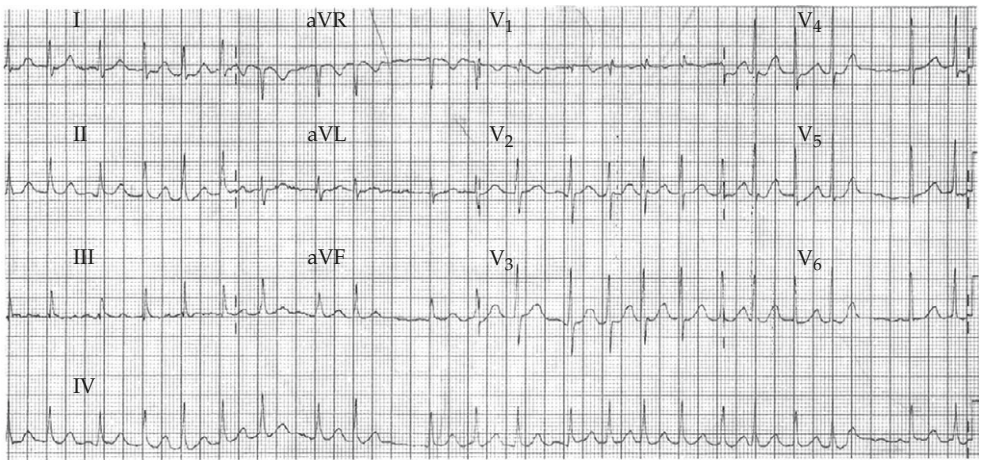

Figure 1. *Electrocardiogram showing atrial fibrillation during an attack of palpitation occurring in the emergency department.*

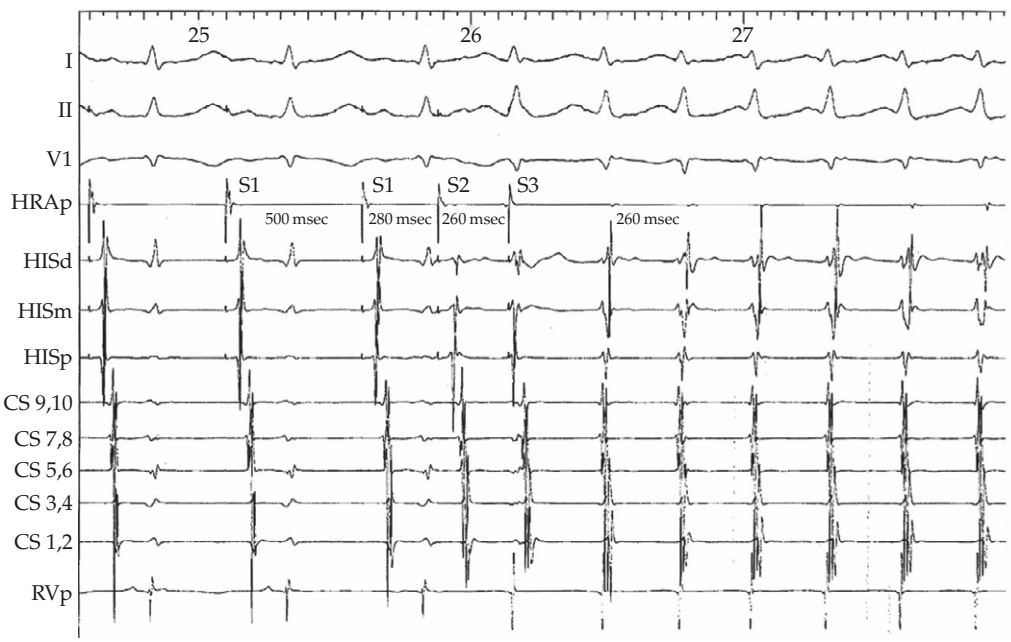

Figure 2. *At the basic pacing cycle length of 500 ms (S1S1) from the high right atrium, double atrial premature extrastimuli (S2, S3) with coupling interval of 280 ms and 260 ms induce sustained common slow-fast atrioventricular nodal reentrant tachycardia with a cycle length of 260 ms. RVp = right ventricle apex; CS = coronary sinus; HIS = His bundle; HRA = high right atrium.*

extrastimuli (Figure 2). During sustained AVNRT, AF was induced by overdrive pacing from the HRA at the pacing cycle length of 240 ms (Figure 3A). After 6 minutes, the AF spontaneously reverted to the common slow-fast AVNRT (Figure 3B). Successful catheter ablation of the slow pathway was performed via a posterior anatomical approach [2]. The slow pathway conduction was totally eliminated. After ablation, the AVNRT could not be induced. In addition, the AF could not be induced by three extrastimuli and rapid atrial pacing at a pacing cycle length of 200 ms from the HRA and proximal coronary sinus, respectively. The trans-septal approach and pulmonary vein isolation were not performed for AF ablation. During 2 years of clinical follow-up, the patient has been free of any palpitations. The Holter electrocardiogram also showed normal sinus rhythm without paroxysmal AF attack.

Discussion

AF is a common arrhythmia which needs the trigger and the atrial substrate to be maintained. The triggers

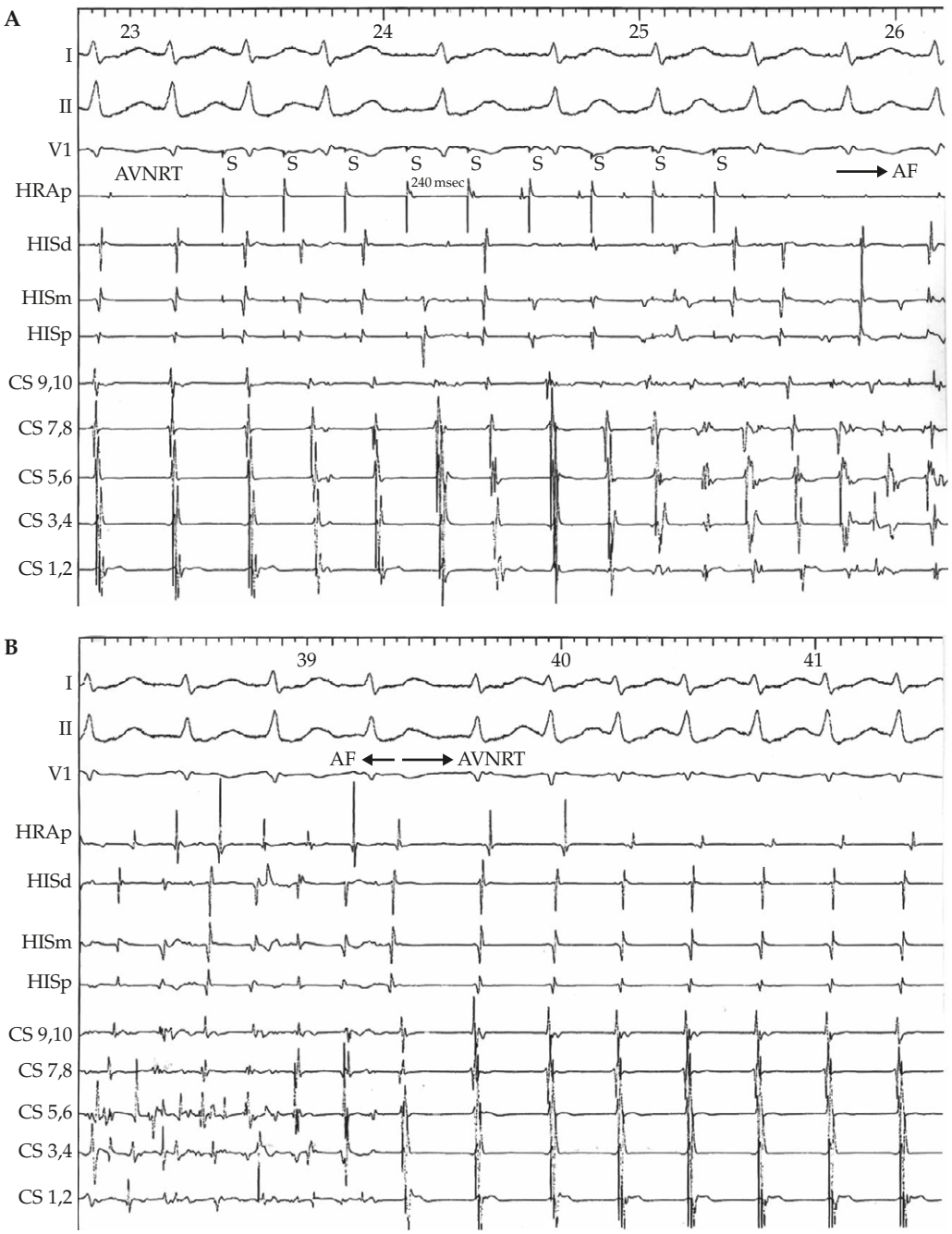

Figure 3. *During sustained atrioventricular nodal reentrant tachycardia, atrial fibrillation is induced by overdrive pacing from the high right atrium at a pacing cycle length of 240 ms (A) and the atrial fibrillation spontaneously reverts to common slow-fast atrioventricular nodal reentrant tachycardia (B). CS = coronary sinus; HIS = His bundle; HRA = high right atrium.*

of AF may include multiple wavelets, focal sources from cardiac or extracardiac foci and reentrant mechanisms of the mother rotor [1,3]. In patients referred for catheter ablation of AF, inducible supraventricular tachycardias during EP study may include AVNRT, atrioventricular reentry tachycardia, atrial flutter and atrial tachycardias [3,4]. Several reports have described the association between AVNRT and AF, particularly in relation to relatively young patients who were referred for AF ablation and who are more likely to have inducible AVNRT at the time of ablation. The possible mechanisms that have been proposed include: (1) an increase in atrial vulnerability resulting from atrial stretch caused by simultaneous contraction of the

atrium and ventricle during AVNRT; (2) increased heart rate and vagal activation during AVNRT; and (3) during AVNRT, ectopy from pulmonary vein or non-pulmonary vein foci possibly triggering onset of AF. The reported incidence of AVNRT-triggered AF ranges from 1.7% to 4.3% [4,5]. Chang et al reported that patients with non-pulmonary vein foci of AF had a higher incidence of AVNRT than those with pulmonary vein foci, and they suggested a close relationship between superior vena cava AF and AVNRT [3]. In our patient, because of her frequent presentation with paroxysmal AF at the emergency department, she was referred for ablation of AF. During her baseline electrophysiology study, the inducible common form of AVNRT and its transition to AF suggested a diagnosis of AVNRT-triggered AF. After modification of the slow pathway conduction by catheter ablation, both AVNRT and AF were totally eliminated, even under aggressive electrophysiological stimulation. This result favored the diagnosis of AVNRT as the trigger for AF in our patient and trans-septal puncture and pulmonary vein isolation were avoided. In addition, after 2 years of clinical follow-up, no palpitation has occurred, and no clinical AVNRT and AF can be demonstrated. These results indicate that the diagnosis of AVNRT-triggered AF is more likely in our patient.

Previous studies have reported that the local conduction delay and local non-uniform anisotropic characteristics of the posterior triangle of Koch may be critical for AF induction, and these findings can explain part of the mechanism by which coronary sinus pacing or biatrial pacing can partially prevent the induction of AF [6–8]. The proposed mechanisms involved in the treatment of AF by ablation of the slow pathway in AVNRT-triggered AF include the following: (1) the atrial vulnerability caused by the dual atrioventricular nodal pathway and the non-uniform anisotropic characteristics of the posterior triangle of Koch may simultaneously be ablated during the procedure of slow pathway ablation [6–8]; (2) the vagal input from the inferior vena cava–inferior atrial vagal ganglionated plexus to the AV node may be interrupted during the slow pathway ablation and may eliminate some vagal-related AF [9–11]; and (3) elimination of AVNRT can block the AVNRT-related increase in atrial vulnerability, as described above.

In conclusion, for patients referred for AF ablation therapy, inducible supraventricular tachycardia should be identified before the AF ablation procedure. In selected patients, only ablation of the AVNRT may cure the AF and avoid regular AF ablation procedures [12].

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對房室結迴旋頻脈成功的電燒治療後亦抑制心房顫動 — 病例報告

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一個心悸的 55 歲女性其心電圖顯示心房顫動，在接受心臟電氣生理學檢查時發現可誘發的房室結迴旋頻脈。電燒掉慢速傳導路徑之後，房室結迴旋頻脈和心房顫動便無法再被誘發。在兩年的追蹤當中，以病人症狀及霍特氏心電圖來評估，並無心房顫動再發的證據。因此我們推論此病人之心房顫動是由房室結迴旋頻脈所引起。在成功的將房室結迴旋頻脈電燒術後，即不需再對於心房顫動作電燒術。

關鍵詞：心房顫動，房室結迴旋頻脈

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