Use of general anesthesia to suppress incessant atrial fibrillation in a patient undergoing ablation for supraventricular tachycardia

Moussa Saleh, MD, Gourg Atteya, MD, Jonathan Willner, MD, Roy M. John, MD, PhD, FHRS

From the Department of Cardiology, Arrhythmia Services, North Shore University Hospital, Manhasset, New York.

Introduction

Atrial fibrillation (AF) can occur in up to 30% of patients with supraventricular tachycardias (SVT) mediated by atrioventricular accessory pathways (AP).[1–4] When it occurs in the course of an electrophysiological (EP) study, mapping and ablation of the SVT is difficult, especially when ventricular pre-excitation is not present. In this case, we report a patient with atrioventricular reciprocating tachycardia (AVRT) mediated by a concealed posterior septal AP in whom repetitive and sustained AF during EP study required conversion from conscious sedation to general anesthesia (GA) for suppression of AF.

Case report

The patient is a 62-year-old man with a past medical history of hypertension who presented with recurrent palpitation that he had experienced since the age of 20. He had been on amiodarone 200 mg daily for the previous 3 years. He had no prior documentation or history of AF. On this occasion, he presented with acute cholecystitis and after placement of cholecystectomy tube and was noted to have recurrent SVT at a heart rate of 160 beats per minute. The SVT had a long R-P interval and was reproducibly terminated with vagal maneuvers. Review of telemetry showed tachycardia initiating with premature atrial contractions (PACs) and consistent termination with a P wave, suggesting an AV nodal–dependent mechanism. His baseline electrocardiogram showed no evidence of ventricular pre-excitation.

Because of recurrent arrhythmia unresponsive to escalating beta blockers in addition to chronic amiodarone, the patient was brought to the EP lab and underwent a comprehensive EP study under conscious sedation. A narrow complex, long VA (190 ms) tachycardia with a cycle length of 460 ms was easily inducible with catheter manipulation, burst atrial pacing, and right ventricular burst pacing. Earliest atrial activation during tachycardia was at the coronary sinus bipole situated at the proximal coronary sinus. Pacing from the right ventricular outflow tract resulted in entrainment of the tachycardia, and an A-H-V response on cessation of pacing with a postpacing interval that was 86 ms in excess of the tachycardia cycle length (Figure 1). His-synchronous premature ventricular contractions advanced the subsequent atrial activation. A diagnosis of orthodromic AVRT was made. His course in the lab was complicated by recurrent AF. AF was seen to initiate with right-sided PACs, left-sided PACs, and ventricular burst pacing, and spontaneously while in AVRT (Figure 2), making it difficult to map the AP. He was given 0.5 mg of intravenous ibutilide in an attempt to maintain sinus rhythm. However, he continued to have recurrent sustained AF that terminated and reinitiated. External DC cardioversion for a sustained episode restored sinus rhythm briefly, but AF reinitiated. He was subsequently

KEY TEACHING POINTS

- In patients with longstanding paroxysmal supraventricular tachycardia (SVT), atrial electrical remodeling can result in difficult-to-control paroxysmal atrial fibrillation (AF).
- When sedation and anxiolytic agents fail to control incessant AF, general anesthesia attenuates adrenergic drive sufficiently to suppress incessant AF during SVT ablation.
- Ablation of the accessory pathway (AP) in patients with atrioventricular reciprocating tachycardia can result in prevention of further AF episodes in about 90% of patients. However, the incidence of AF in patients with APs is higher than that of the general population even after AP ablation.

KEYWORDS Atrial fibrillation; Accessory pathway; Ablation; Supraventricular tachycardia; Sedation

(Heart Rhythm Case Reports 2021;7:87–90)

Funding: None. Conflict of Interest: None. **Address reprint requests and correspondence:** Dr Moussa Saleh, Department of Cardiology, Arrhythmia Services, North Shore University Hospital, 300 Community D, Manhasset, New York, 11030. E-mail address: Moussa.a.saleh@gmail.com.

2214-0271/© 2020 Heart Rhythm Society. Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

https://doi.org/10.1016/j.hrcr.2020.11.007

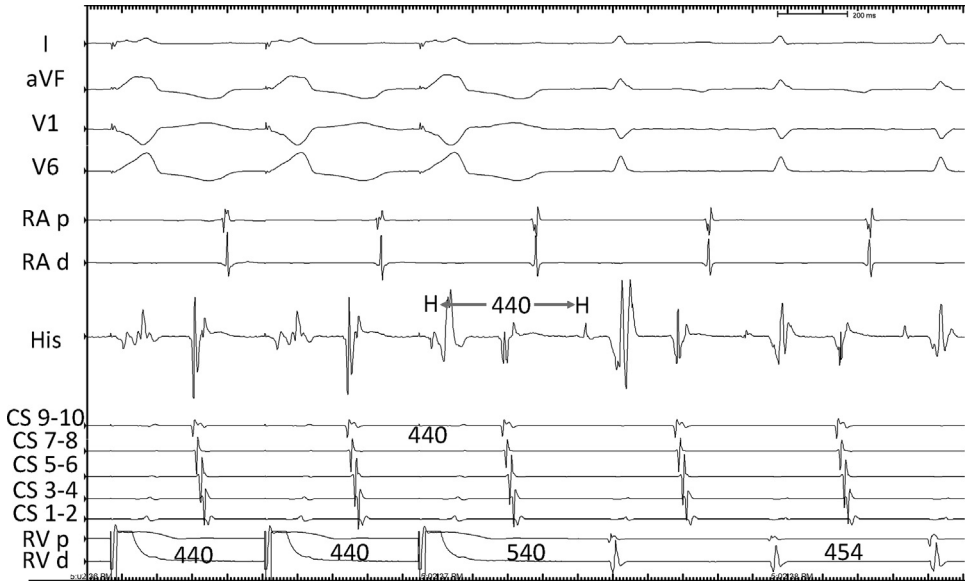

Figure 1 Right ventricular (RV) pacing during supraventricular tachycardia shows entrainment with anterograde activation of the His, postpacing interval of 86 ms in excess of tachycardia cycle length. RV pacing cycle length 440 ms; His bundle activation is anterograde as shown by H-H interval of 440 ms after cessation of pacing; tachycardia cycle length = 454 ms; postpacing interval = 540 ms.

intubated and placed under GA, which resulted in complete suppression of AF. Earliest atrial activation was mapped to the posterior septum at the tricuspid annulus in AVRT and with ventricular pacing. Ablation at the site resulted in loss of conduction across the pathway. The patient was no longer inducible for AVRT. A waiting period of 30 minutes was observed without reoccurrence of conduction across the pathway. No further AF was observed after extubation or during the subsequent period of observation in the hospital. Amiodarone was discontinued and replaced with metoprolol tartrate 25 mg twice a day.

Discussion

This case represents an unusual but vexing issue of AF interrupting an SVT study, rendering mapping and ablation difficult. If overt ventricular pre-excitation is present, earliest ventricular activation can be targeted to ablate the ventricular insertion of the pathway.[5] However, when SVT is due to a concealed AP, mapping is not possible when AF occurs. Usually, the arrhythmia is short-lived, and cardioversion is successful in maintaining sinus rhythm, allowing for ventricular pacing to identify pathway location. Use of antiarrhythmic drugs such as ibutilide may have a suppressive effect on AF but also runs the risk of masking AP function. In the present case, AF was repetitive with periods of sustained AF despite adequate sedation and ibutilide 0.5 mg. Induction of GA resulted in complete suppression of AF, allowing us to continue mapping and ablation without further interruption

Recurrent SVTs are well recognized to predispose to the development of AF, most likely owing to recurrent rapid atrial stimulation resulting in electrical remodeling. There may be additional mechanisms in patients with APs.[6,7] Ablation of the AP can result in prevention of further AF episodes in about 90% of patients. However, the incidence of AF in patients with APs is higher than that of the general population even after AP ablation.[8] It is possible that the patient in the present report may manifest AF over time and will therefore need continued surveillance for the arrhythmia. Given the low CHADs-VASc score of 1, we did not feel he merited anticoagulation at this stage.

GA is well known to attenuate sympathetic drive and suppress arrhythmias. The use of GA is known to be an effective intervention for patients who present with ventricular tachycardia storm.[9,10] In patients with automatic arrhythmia mechanisms of focal origin, use of GA can result in suppression of inducibility and hamper mapping and ablation.[11] Arrhythmias dependent on re-entrant mechanisms are less likely to be influenced by GA. In SVT mediated by APs, it has the benefit of maintaining intact AP function, unlike with use of sodium or potassium channel blockers. Hence, when recurrent incessant AF interrupts SVT mapping and ablation, the use of GA should be a consideration. In patients with a prior history of AF, the arrhythmia may well be triggered by a source such as the pulmonary veins. In such situations, electrical isolation of the pulmonary vein or other focal sources may have to be performed to allow ablation of the SVT substrate.

Conclusion

When recurrent AF occurs during an EP study for SVT, the use of GA can result in suppression of AF, allowing for continued arrhythmia induction and ablation.

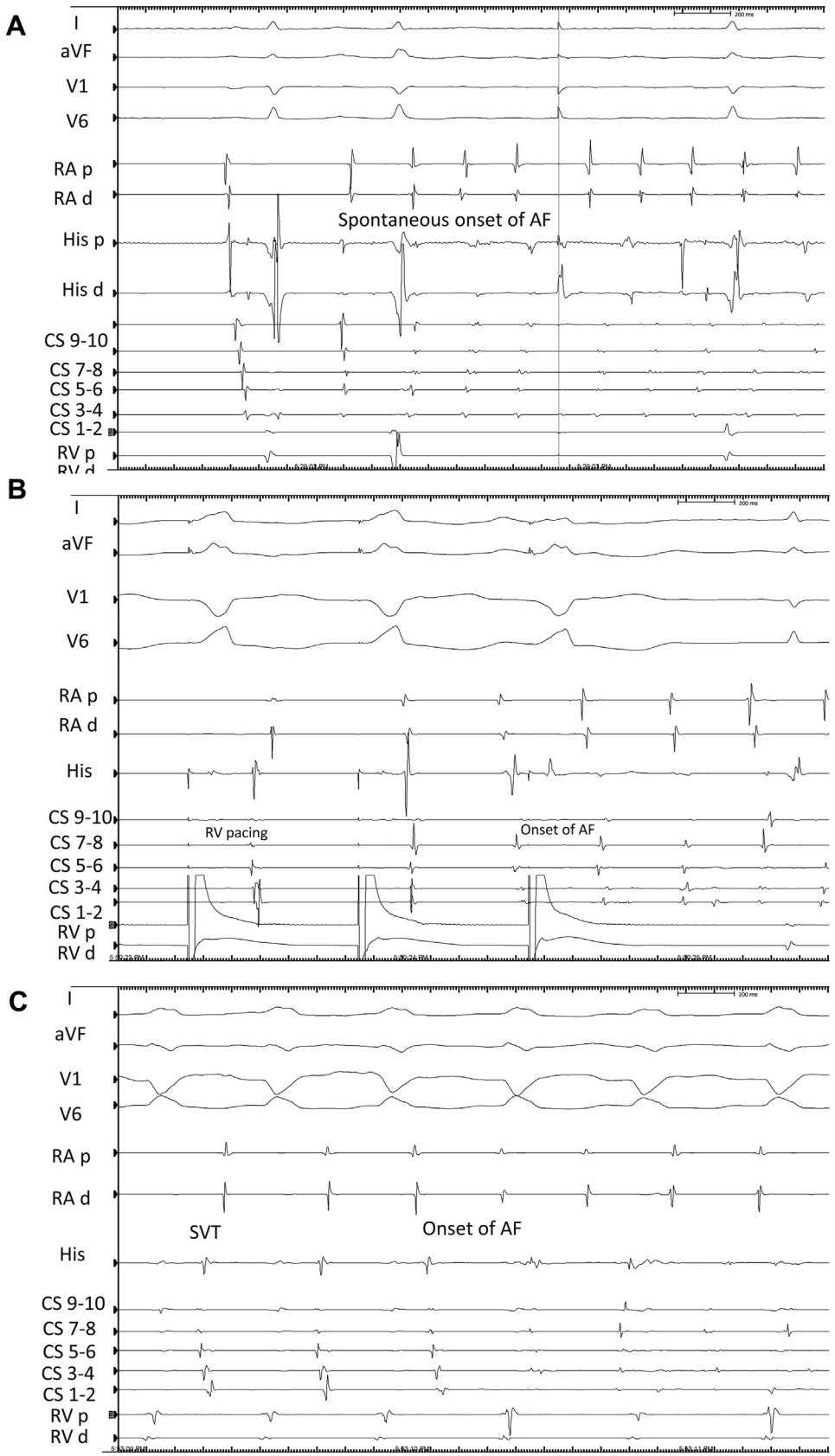

Figure 2 Intracardiac electrograms showing onset of atrial fibrillation (AF) spontaneously (**A**), onset provoked by right ventricular (RV) pacing (**B**), and onset provoked by supraventricular tachycardia (SVT) (**C**).

References

1. Berkman NL, Lamb LE. The Wolff, Parkinson, White electrocardiogram. N Engl J Med 1968;278:492–494.
2. Sharma AD, Klein GJ, Guiraudon GM, Milstein S. Atrial fibrillation in patients with Wolff-Parkinson-White syndrome: incidence after surgical ablation of the accessory pathway. Circulation 1985;72:161–169.
3. Haissaguerre M, Fischer B, Labbé T, et al. Frequency of recurrent atrial fibrillation after catheter ablation of overt accessory pathways. Am J Cardiol 1992;69:493–497.
4. Borregaard R, Lukac P, Gerdes C, et al. Radiofrequency ablation of accessory pathways in patients with the Wolff-Parkinson-White syndrome: the long-term mortality and risk of atrial fibrillation. Europace 2015;17:117–122.
5. Jackman WM, Wang XZ, Friday KJ, et al. Catheter ablation of accessory atrioventricular pathways (Wolff-Parkinson-White syndrome) by radiofrequency current. N Engl J Med 1991;324:1605–1611.
6. Centurion OA. Atrial fibrillation in the Wolff-Parkinson-White Syndrome. J Atr Fibrillation 2011;4:287.
7. Nattel S, Burstein B, Dobrev D. Atrial remodeling and atrial fibrillation: mechanisms and implications. Circ Arrhythm Electrophysiol 2008;1:62–73.
8. Bunch TJ, May H, Blair TM, et al. Long-term natural history of adult Wolff-Parkinson-White syndrome patients treated with and without catheter ablation. Circ Arrhythm Electrophysiol 2015;8:1465–1471.
9. Mulpuru SK, Patel DV, Wilbur SL, Vasavada BC, Furqan T. Electrical storm and termination with propofol therapy: a case report. Int J Cardiol 2008; 128:e6–e8.
10. Cronin EM, Bogun FM, Maury P, et al. 2019 HRS/EHRA/APHRS/LAHRS expert consensus statement on catheter ablation of ventricular arrhythmias. J Interv Card Electrophysiol 2020;59:145–298.
11. Nof E, Reichlin T, Enriquez AD, et al. Impact of general anesthesia on initiation and stability of VT during catheter ablation. Heart Rhythm 2015; 12:2213–2220.