Focal Atrial Tachycardia With a V-A-V Response Seen on Ventricular Overdrive Pacing

Khalil Kanjwal, MD, FACC, FHRS[1]; Asim Kichloo, MD, DM[2,3]; Khalid Mohiuddin Mir, MD, DM[4]; Abdul Qadir Haji, MD[5]

Supraventricular tachycardias (SVT) are common arrhythmias that can be a source of significant palpitations. Radiofrequency ablation is commonly performed in these patients, with a very high success rate of 95%. For a safe and successful ablation, a thorough electrophysiological evaluation of the arrhythmia is performed before attempted ablation. Various observations noted during sinus rhythm as well as during tachycardia can provide clues to the mechanism of the tachycardia. Ventricular overdrive pacing (VOP) is commonly performed during narrow complex tachycardia, and the post-pacing response and postpacing interval (PPI) can provide a diagnosis of the type of SVT. A response of V-A-V is noted in AV node reentry (AVNRT) and AV reentry (AVRT), and a V-A-A-V response is consistent with the diagnosis of atrial tachycardia (AT).[1] Herein, we present an interesting case of an atrial tachycardia with a short VA interval and V-A-V response noted on ventricular overdrive pacing. We discuss possible mechanisms of V-A-V response in this patient and why we believe it was a pseudo V-A-V response.

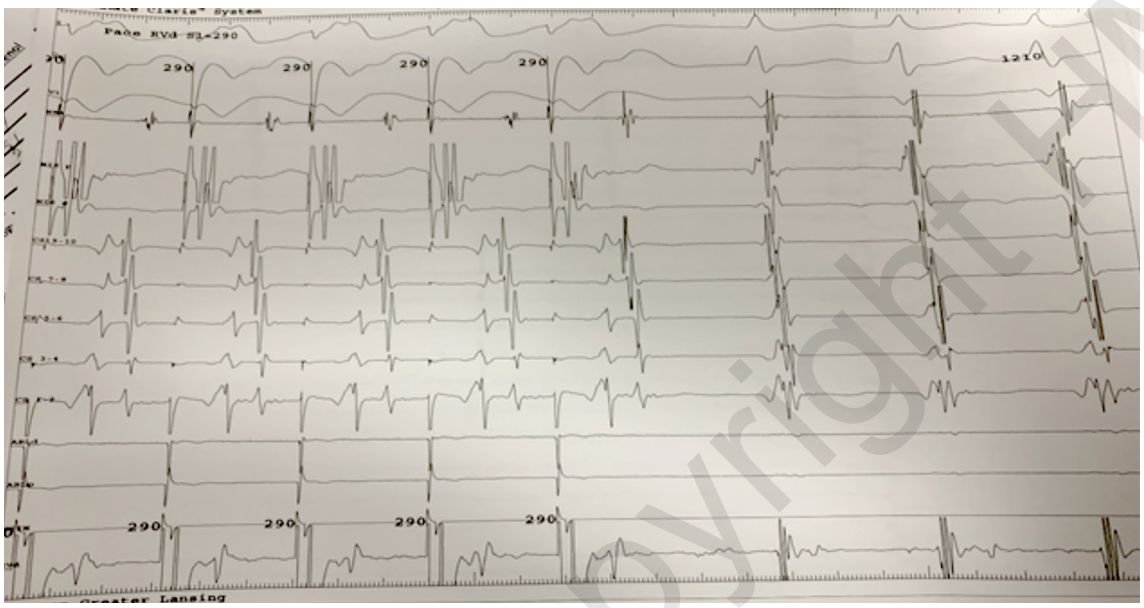

Figure 1. Entrainment of a narrow complex A on V tachycardia. At first glance, it looks like the tachycardia is entrained with a V-A-V response and long PPI-TCL (190 ms), consistent with AVNRT.

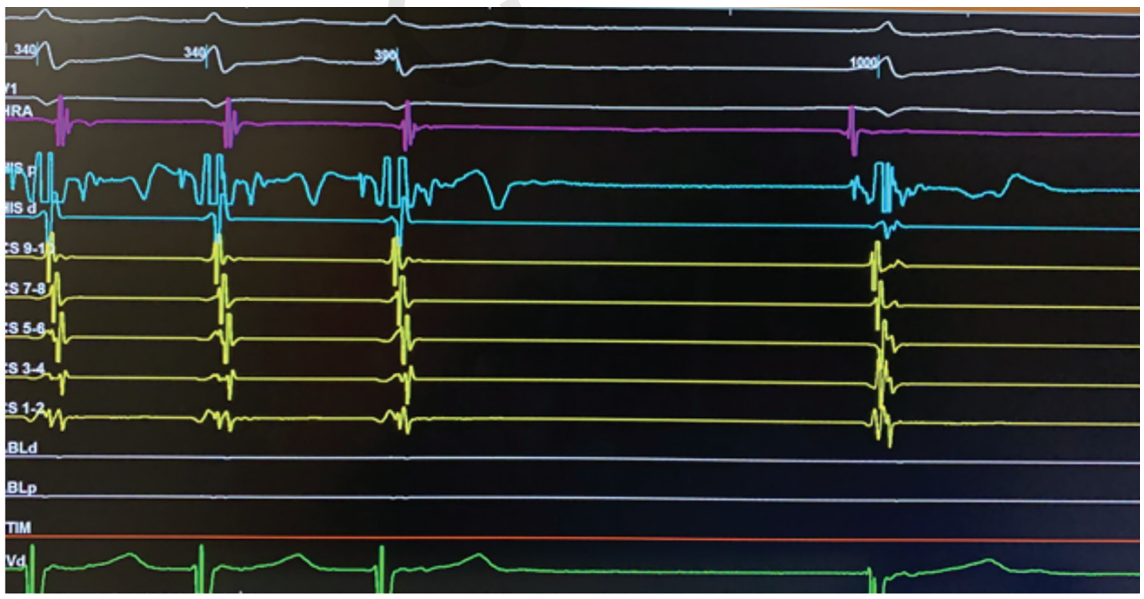

Figure 2. Termination of the tachycardia with AV block. Note the increased tachycardia cycle length before termination.

Case Presentation

A 57-year-old female with a history of recurrent palpitations was evaluated at our arrhythmia clinic. She reported recurrent palpitations that would come on suddenly and last a few seconds or up to 20 minutes. She would also feel chest discomfort during these episodes. She was evaluated with a 30-day event monitor, but she did not have any episodes during that period. Her echocardiogram revealed a structurally normal heart and normal ejection function. She also had a stress test that was normal. An electrocardiogram showed a normal sinus rhythm and had no pre-excitation. Given her recurrent episodes of palpitations, she was offered an EP study. She presented to the EP lab in normal sinus rhythm. Her HV interval was 45 ms. During catheter manipulation, she went into a regular narrow complex tachycardia with a tachycardia cycle length (TCL) of 310 ms. During tachycardia, her septal VA interval was 45 ms. Ventricular entrainment of the tachycardia was performed. The postpacing response was noted to be V-A-V with a long postpacing interval (PPI-TCL 190 ms) (Figure 1). These entrainment features were consistent with a diagnosis of AVNRT. A very short VA interval during tachycardia was against the diagnosis of AVRT. Termination of the tachycardia with AV block was consistent with AVNRT (Figure 2). Ablation in the slow pathway area was performed. An intermittent junctional rhythm was noted during ablation. The EP study was repeated, and the patient remained easily inducible with burst pacing.

After multiple attempts at failed ablation in the area of the slow pathway, the ablation catheter was moved superiorly. The tachycardia remained easily inducible despite ablation in this area. The EP study was again repeated. There was a very subtle change noted in the activation in the coronary sinus (CS) (Figure 3) and during termination it was noted the TCL prolonged before termination of the tachycardia. On closer look, it was noted during the initial part of entrainment that the tachycardia was clearly entrained. However, towards the end of the entrainment, the tachycardia accelerated to a TCL of 267 ms (Figure 4). There was a clear wobble in the TCL, resulting in delay in the next A after the acceleration. Due to this wobble or variation in TCL, the response appeared to be a V-A-V response, but this clearly was the pseudo V-A-V response, mainly because of the wobble in the TCL. The VA interval in the first return tachycardia beat was greater than the VA interval

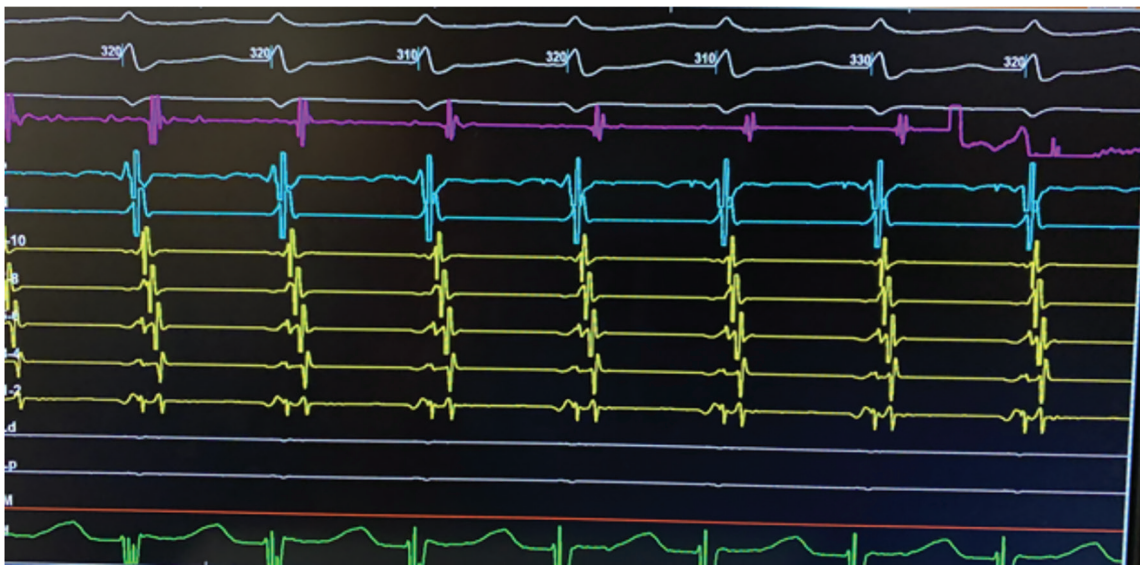

Figure 3. **The tachycardia showing subtle tachycardia cycle length wobble and change in atrial activation.**

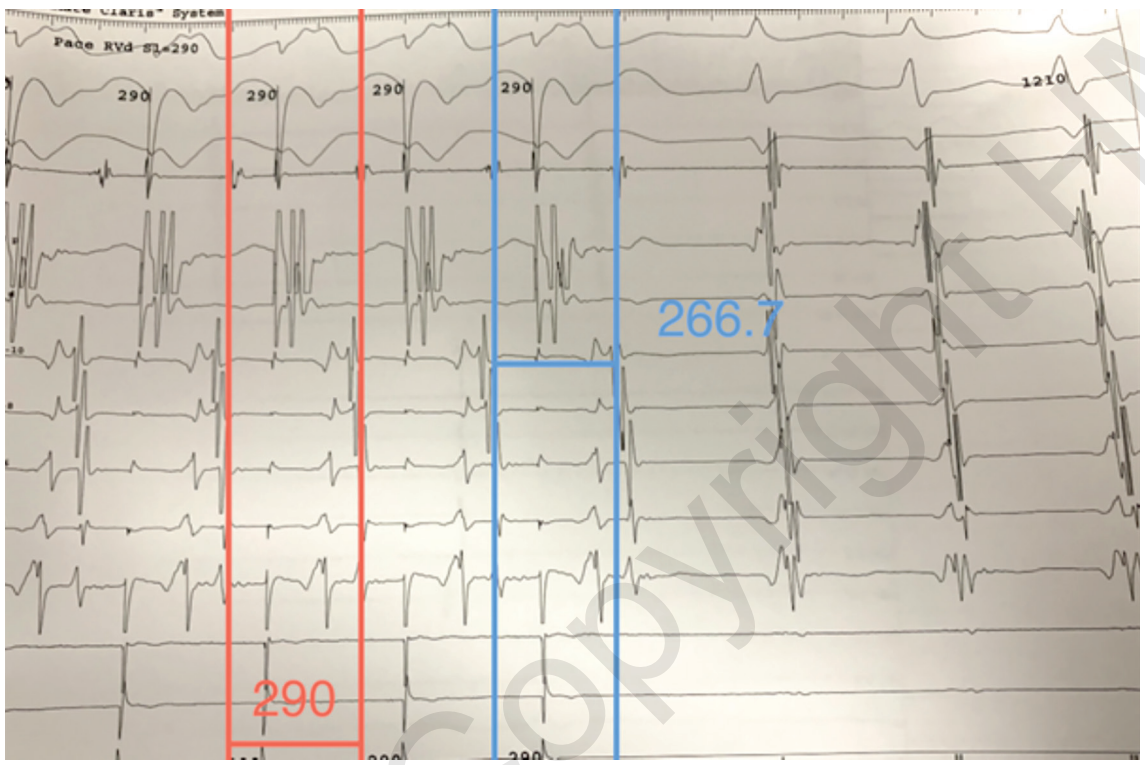

Figure 4. **During the initial part of entrainment, the tachycardia is clearly entrained. However, towards the end of the entrainment, the tachycardia accelerates to a TCL of 267 ms. There is a clear wobble in the TCL, resulting in delay in the next A after the acceleration. Due to this wobble or variation in the TCL, the response appears to be a V-A-V response, but this is clearly the pseudo V-A-V response, mainly because of the wobble in the TCL. The VA interval in the first return tachycardia beat is greater than the VA interval in the second beat, suggestive of variability in the TCL.**

This case illustrates that a pseudo V-A-V response can be seen in atrial tachycardia due to variability in the tachycardia cycle length and overdrive suppression of the atrial focus. An activation map may allow for correct diagnosis in such patients. Electrophysiologists should pay close attention while interpreting the maneuvers during evaluation of various tachycardias.

in the second beat, suggestive of variability in the TCL. It was decided to perform activation mapping of the tachycardia using CARTO (Biosense Webster, Inc., a Johnson & Johnson company). The area of earliest activation was noted to be just superior to the roof of the CS ostium (Figure 5). This area was tagged on the 3D map. Ablation was performed in sinus rhythm. The tachycardia, which was easily inducible earlier, remained noninducible despite atrial burst and atrial extrastimuli pacing both on and off high-dose isoproterenol.

Discussion

The most common reason for A on V tachycardia remains AVNRT. However, atrial tachycardias originating from the septum may have a short VA interval as well.

We believe this case is a very unusual presentation of focal atrial tachycardia originating from the area just superior to the CS ostium. There were certain interesting observations noted in the tachycardia. The tachycardia revealed a very short VA interval, giving an appearance of AVNRT. Furthermore, VOP yielded a V-A-V response and long PPI-TCL. Even the termination of the tachycardia was consistent with AVNRT. The ablation performed in the slow pathway region and during ablation of accelerated junctional rhythm was noted, but the tachycardia remained inducible after multiple ablation attempts within the slow pathway region. It was interesting to note that the tachycardia had some change in activation sequence, especially in the CS catheter, although the TCL remained the same.

This observation, along with the tachycardia remaining inducible despite multiple attempts at ablation in the slow pathway, led to the use of electroanatomic mapping and an activation map. On the activation map, the area of earliest focal activation was noted just superior to the roof of the CS ostium. In a typical AVNRT, the earliest atrial signal during tachycardia is noted on the His catheter. However, in this tachycardia, the earliest (-30 ms) atrial signal was away from His. This area was tagged on the map and ablation was performed in sinus rhythm. There were no junctional beats noted during ablation in this area. We preferred to perform ablation in sinus rhythm to detect any AV conduction problem at the earliest opportunity and come off the ablation. The tachycardia was no longer inducible post ablation, suggesting this was an atrial tachycardia. We believe that subtle activation changes noted in the CS and successful focal ablation in atria superior to the CS ostium suggests AT as the mechanism of the tachycardia. However, tachycardia features such as V-A-V response during ventricular entrainment and a long PPI were strongly suggestive of AVNRT. We were not able to demonstrate dual AV nodal physiology because the tachycardia was very easily inducible with atrial pacing. We believe the V-A-V response noted on VOP was likely

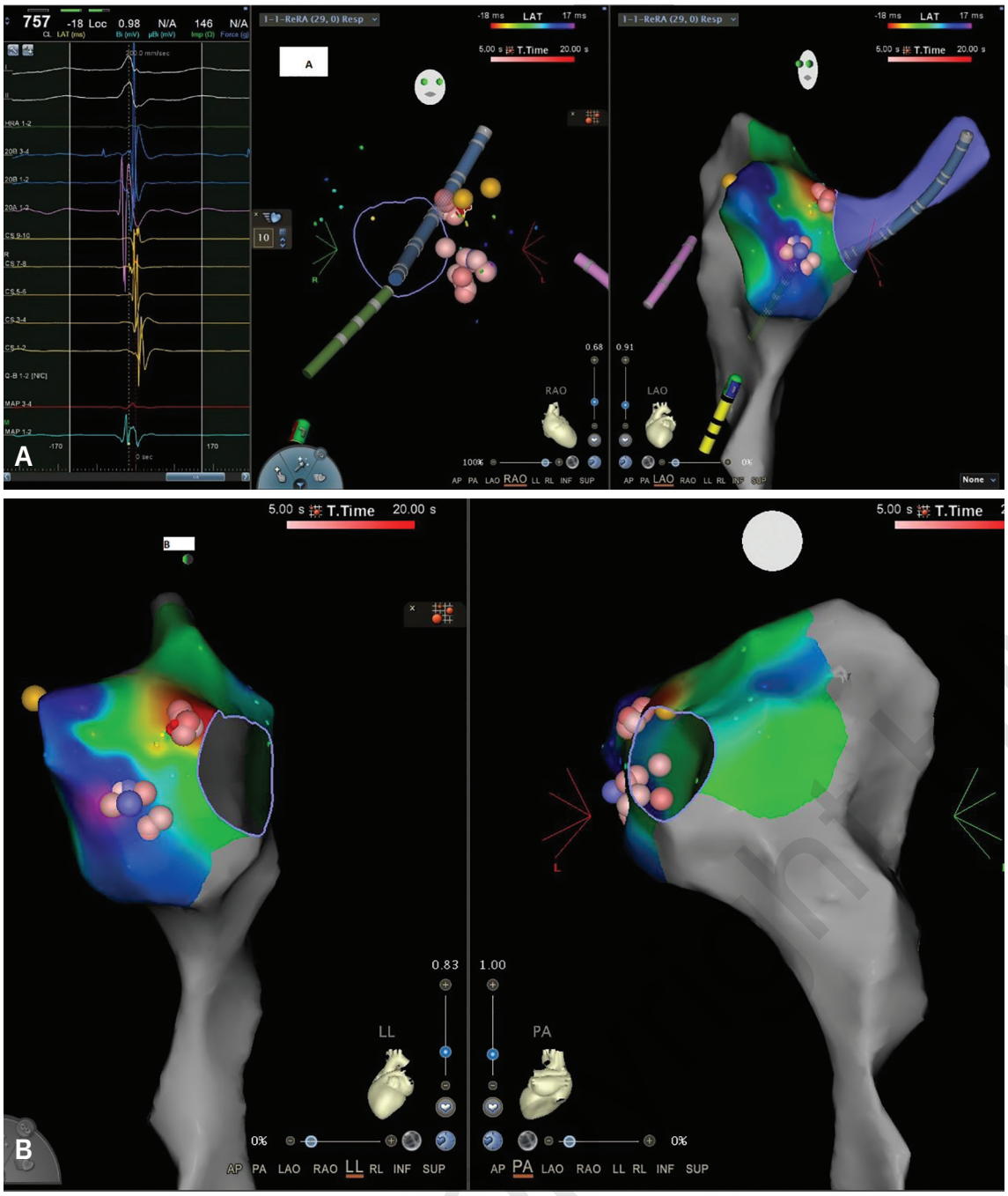

Figure 5. (A) Three-dimensional and activation maps showing slow pathway ablation anterior to the CS, which was unsuccessful despite junctional beats noted during the ablation. (B) Earliest focal activation noted in the area superior to the roof of the coronary sinus ostium. Ablation in this area resulted in non-inducibility of the tachycardia.

due to a combination of wobble in the atrial cycle length (acceleration) and subsequent suppression of the atria focus that resulted in the next AT signal occurring slightly after the ventricular signal. However, looking closely at the first tachycardia beat, the VA interval on the high right atrium is longer that the subsequent beat. There are reports of AT having a pseudo V-A-V response noted during transient AV dissociation following VOP.[2,3]

The termination of the tachycardia was interesting, as there was a clear slowing of the AT before termination with AV block. These features should also make one suspicious of focal AT as being the mechanism of the tachycardia. During typical AVNRT, the retrograde atrial signal is also earliest in the His region, which was not noted in our patient. Although we felt the diagnosis here was atrial tachycardia, we recognize that typical AVNRT utilizing a slow pathway with left posterior extension into the CS, which did not respond to conventional slow pathway ablation despite the appearance of junctionals, cannot be definitively excluded.

We believe this case is a very unusual presentation of focal atrial tachycardia originating from the area just superior to the coronary sinus ostium.

Conclusion

This case illustrates that a pseudo V-A-V response can be seen in AT due to variability in the TCL and overdrive suppression of the atrial focus. An activation map may allow for correct diagnosis in such patients. Electrophysiologists should pay close attention while interpreting the maneuvers during evaluation of various tachycardias. ■

Acknowledgements. The authors would like to acknowledge Jackie Buckley, senior representative at Biosense Webster, for her help with images.

Khalil Kanjwal, MD, FACC, FHRS[1]; Asim Kichloo, MD, DM[2,3]; Khalid Mohiuddin Mir, MD, DM[4]; Abdul Qadir Haji, MD[5]

[1]Director of Electrophysiology, Clinical Associate Professor of Medicine, McLaren Greater Lansing Hospital, Department of Electrophysiology, Lansing, Michigan;
[2]Samaritan Medical Center, Department of Internal Medicine, Watertown, New York;
[3]Central Michigan University, Department of Internal Medicine, Saginaw, Michigan;
[4]Super Speciality Hospital Srinagar, Kashmir, India;
[5]Martinsburg VA Medical Center, Department of Cardiology, Martinsburg, West Virginia

Disclosures: The authors have no conflicts of interest to report regarding the content herein.

References

1. Knight BP, Zivin A, Souza J, et al. A technique for the rapid diagnosis of atrial tachycardia in the electrophysiology laboratory. *J Am Coll Cardiol.* 1999;33:775-781.
2. Kwon DS, Marcus GM. A supraventricular tachycardia: what is the mechanism? *Heart Rhythm.* 2009;6:1378-1379.
3. Jastrzebski M, Kukla P. The V-A-V response to ventricular entrainment during atrial tachycardia: what is the mechanism? *J Cardiovasc Electrophysiol.* 2012;23(11):1266-1268.