

Atrial paced beat rocks the boat of a broad QRS tachycardia causing AVVA response. What is the mechanism?

Anand Manickavasagam¹, Sirish Chandra Srinath Patloori¹, Hariharan Narasaiyan¹, Javiad Ahmad¹, David Chase¹, and John Jacob¹

¹Christian Medical College and Hospital Vellore Dodd Memorial Library

October 27, 2022

Title Page

Manuscript Title:

Atrial paced beat rocks the boat of a broad QRS tachycardia causing AVVA response. What is the mechanism?

Key words: Atriofascicular pathway, BBRVT, dual AV nodal physiology

Authors details:

1. Dr Anand Manickavasagam, MBBS, MD, DM, Department of Cardiology, Christian Medical College, Vellore, Tamilnadu, India
2. Dr Sirish Chandra Srinath Patloori, MBBS, MD, DM, CEPS, Department of Cardiology, Christian Medical College, Vellore, Tamilnadu, India
3. Dr Hariharan Narasaiyan, MBBS, MD, DM, Department of Cardiology, Christian Medical College, Vellore, Tamilnadu, India
4. Dr Javiad Ahmad, MBBS, MD, DM, Department of Cardiology, Christian Medical College, Vellore, Tamilnadu, India
5. Dr David Chase, MBBS, MD, DM, Department of Cardiology, Christian Medical College, Vellore, Tamilnadu, India
6. Dr John Roshan, MBBS, MD, DM, CEPS, CCDS, Professor, Department of Cardiology, Christian Medical College, Vellore, Tamilnadu, India

Corresponding Author:

Dr John Roshan,
Cardiology Unit IV, Department of Cardiology,
Christian Medical College,
Vellore, Tamilnadu, India
PINCODE – 632004

Phone Number: 91-04162283572, 91-9443312680

E-mail id: johnroshanjacob@gmail.com

Acknowledgements : None

Financial disclosure: None for all the authors

No conflict of interest

Ethical Statement: As this was a case report ethical clearance was not obtained. Patient consent was obtained prior to the procedure for publication of the patient clinical materials if found suitable in a medical journal. Hence, we request you to waive off the ethical committee clearance.

Data availability statement : The data that support the findings of this article are available on request from the corresponding author.

Abstract : None (As it is EP rounds)

Case Presentation

46-year-old women who had a history of recurrent palpitations was cardioverted elsewhere for a broad QRS tachycardia of left bundle branch block (LBBB) morphology (Figure 1a). On evaluation, her baseline ECG showed SR with narrow QRS which changed to LBBB morphology (Figure 1b) at heart rates greater than 90bpm which was suggestive of acceleration dependent block.¹ Echocardiogram showed severe left ventricular (LV) dysfunction. Cardiac MRI was done which showed no scar or myocardial edema. She underwent a coronary angiogram which showed normal coronary arteries. Levophase of the coronary angiogram showed atresia of the coronary sinus [CS] os and it drained partially through the small cardiac vein into the right atrium (RA) and partially through a persistent left SVC.

She was taken up for an electrophysiology study with the intent of radiofrequency ablation. Three quadripolar catheters were placed in the His, right ventricular apex, and right atrial appendage [RAA]. As the CS os was atretic, it could not be cannulated. Baseline AH (Atrial-His 70ms) and HV (His-Ventricular 45ms) intervals were normal. On atrial pacing, there were two subtly different LBBB morphology QRS seen (Figure 1c and Figure 1d). QRS in lead II in Figure 1d was biphasic similar to the tachycardia ECG in Figure 1a while it was monophasic in Figure 1b and 1c. On pacing from the RAA, predominant QRS morphology was LBBB with a duration of 130ms with a positive HV interval of 45ms – suggestive of aberrant conduction (Figure 1c). On pacing from lateral RA, the LBBB widened to 150ms and the His was retrogradely activated with HV being negative and VH interval fixed at 10ms (figure 1d). On incremental atrial pacing, the AV interval was decremental and VH interval remained the same, suggestive of atriofascicular pathway. With programmed ventricular extra stimulus, a broad QRS tachycardia of LBBB morphology (Figure 2) with a QRS as in Figure 1d was induced. During the tachycardia, His was retrogradely activated, VH was 10ms and atrial activation was earliest in the His region. Septal refractory APC (in Figure 2) from lateral RA was delivered. There seems to be an AVVA response followed by an antegrade His (marked as H in figure 2) with positive HV of 45ms. This was followed by a resumption of the original tachycardia. What is the likely mechanism for the response seen?

Discussion:

The broad QRS tachycardia had LBBB morphology with retrograde His activation and VH interval of 10ms and morphology similar to Figure 1a – suggesting atriofascicular pathway as antegrade limb. The cycle length of the tachycardia was 310ms (Figure 3). Septal refractory APC advanced the next ventricular electrogram (V EGM) by 20ms, and this advanced the next His activation by 20ms (arrow in Figure 3). The atrial activation after the septal refractory APC is most likely a spontaneous APC. It could not have resulted from activation via a retrograde fast AV nodal pathway since it occurs before the His activation. This APC conducted decrementally down the atriofascicular pathway leading to delay in the next V and His EGM. Until this event, changes in the VV interval predicted changes in the HH interval. The next HA interval was 140ms which was shorter than the tachycardia HA interval of 160ms. This was followed by antegrade His activation with a HH interval of 250ms and a positive HV interval of 45ms. The ventricular activation that resulted after the antegrade His activation had a QRS with morphology as in Figure 1c suggestive of LBBB aberrancy. This was followed by a prolonged HA interval of 190ms and then the antidromic AVRT involving the atriofascicular pathway continued. The possible mechanisms to explain these findings are:

1. Antidromic tachycardia with antegrade limb being atriofascicular pathway. Due to decremental conduction in the atriofascicular pathway, there was a prolongation of HH interval to 330ms. This HH prolongation facilitated rapid conduction in the retrograde direction through the AV node resulting in a short HA of 140msec. The impulse conducted back to the ventricle through another AV nodal slow pathway. Since the atriofascicular pathway was refractory this AV nodal slow pathway activated the His antegradely. The atrium was subsequently activated retrogradely through another slow AV nodal pathway with a prolonged HA interval of 190ms. The antidromic AVRT involving the atriofascicular pathway subsequently got reinitiated.
2. Dual tachycardia – antidromic tachycardia involving atriofascicular pathway switching transiently to bundle branch reentrant ventricular tachycardia (BBRVT). However, this will still require the presence of dual AV node physiology to explain the antegrade His activation that occurred prior to resumption of the antidromic tachycardia. BBRVT with His dissociation has been described and presence of atriofascicular pathway makes deciphering the mechanism more complex.²
3. Antidromic tachycardia involving atriofascicular pathway and bystander septal accessory pathway with decremental property which took the impulse retrogradely up for the beats where HA interval was 140ms and 190ms. However, there was no evidence of retrograde accessory pathway conduction by parahisian pacing and VA block was demonstrated with intravenous adenosine.

During the EP study, the tachycardia had two differing cycle lengths with AV, VH interval being constant and change in HA interval causing change in tachycardia cycle length. HA interval changed from 160ms to 210ms suggestive of dual AV nodal physiology. The atriofascicular pathway potential was mapped to lateral tricuspid annulus at 8 o clock position and was ablated with radiofrequency energy. Postablation of atriofascicular pathway, there was dual AV nodal physiology with AH jump. However, there was no tachycardia inducible even on isoprenaline and with aggressive atrial and ventricular pacing protocols. Hence, slow pathway modification with radiofrequency energy was not done.

In conclusion, a patient with an atriofascicular pathway and multiple AV nodal pathways can have a pseudo AVVA response due to intermittent shift from antidromic AVRT involving the atriofascicular pathway to pathways in the node.

Acknowledgements : None

References

Wit AL, Josephson ME. Basic foundations of clinical electrophysiology. Electrophysiological mechanism of phase 4 conduction block in the His Purkinje system: a comparison with phase 3 block. *Arrhythmia Grand Rounds* 2015;1(2):79-86. DOI: [http:// dx.doi.org/10.12945/j.agr.2015.033-15](http://dx.doi.org/10.12945/j.agr.2015.033-15)

Jain A, Gangwar P, Selvaraj RJ. Wide Complex Tachycardia: What is the mechanism? *J Cardiovasc Electrophysiol.* 2021;32:2746-2748. <https://doi.org/10.1111/jce.15216>

Figure legend:

Figure 1 . Figure 1a – 12 lead ECG showing wide QRS tachycardia of LBBB morphology. Figure 1b – 12 lead ECG showing sinus rhythm with LBBB aberrancy. Figure 1c – On pacing from RA appendage, antegrade His and HV interval of 45ms with LBBB aberrancy is seen. Figure 1d – On pacing from lateral RA, retrograde His with VH interval of 10ms and QRS of LBBB morphology is seen. Figure 1c and Figure 1d, His P – proximal bipole and His D – distal bipole of quadripolar His catheter and RVA – distal bipole of catheter in RV apex.

Figure 2. Intracardiac electrogram when septal refractory APC was given. From top to bottom – 12 leads of surface ECG, LRA – distal bipole of catheter in lateral right atrium, His P, His D and RVA as in Figure 1.

Figure 3 . Intracardiac electrogram when septal refractory APC was given. From top to bottom – I, aVF and V1 represent leads of surface ECG, LRA p – proximal bipole and LRA d - distal bipole of catheter in

