Indian Pacing and Electrophysiology Journal 17 (2017) 58–62

Contents lists available at ScienceDirect

Indian Pacing and Electrophysiology Journal

journal homepage: www.elsevier.com/locate/IPEJ

Wide QRS tachycardia in a patient with pre excitation; what are the pathways involved? Pacing manoeuvres to characterize a unique pathway

Krishna Kumar Mohanan Nair, Anees Thajudeen*, Narayanan Namboodiri, Ajitkumar Valaparambil

Department of Cardiology, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum -11, Kerala, India

ARTICLE INFO

Article history:
Received 2 December 2016
Received in revised form
14 January 2017
Accepted 16 January 2017
Available online 20 January 2017

Keywords:
Antidromic reciprocating tachycardia
Posteroseptal accessory pathway
Epicardial pathway

ABSTRACT

A 30year old patient presented to us with recurrent episodes of palpitation and documented tachycardia. In all his presentations a wide QRS tachycardia was recorded. The baseline ECG showed pre excitation. The 12 lead ECG of the tachycardia and the baseline ECG is shown in Fig. 1A. During EP study the patient had baseline pre excitation and the HV interval was 16 ms. A duo-decapolar halo (HL) catheter was used to map right atrium and a decapolar coronary sinus (CS) catheter was used to map coronary sinus. In addition a His bundle and right ventricular (RV) quadripolar catheters were used. The delta wave morphology was suggestive of a posteroseptal pathway. Ventricular pacing from RV apex showing central decremental conduction with ventriculo-atrial Wenkebach at 290 ms. Ventricular extra-stimulation also showed decremental conduction and VA block at S1 S2 of 400,240. The intra cardiac recording of tachycardia and its initiation is shown in Fig. 1B. Pacing from lateral RA (HL 5, 6 electrodes) showed progressive pre excitation with extrastimulation and induction of tachycardia. The QRS morphology was same as the patient's clinical tachycardia and the tachycardia cycle length (TCL) was 304 ms. An atrial entrainment protocol showed entrainment with the same QRS morphology while pacing from right atrium. The VA interval of the first return cycle was the same as the subsequent VA intervals. A ventricular entrainment protocol showed V-A-V response and post pacing interval of 414 ms. An atrial extra systole was given from the mid CS electrodes (CS 5, 6) – the effect is shown in Fig. 3. In sinus rhythm a parahisian pacing manoeuvre was done as shown in Fig. 4A. What is the mechanism of the tachycardia and what are the pathways involved?

1. Commentary

The differential diagnoses for a wide QRS tachycardia in the setting of baseline pre excitation includes 1) orthodromic re-entrant tachycardia (ORT) with aberrancy 2) Atrial tachycardia or flutter with pre excitation 3) Classical antidromic re-entrant tachycardia (ART) 4)Antidromic (duodromic) tachycardia due to pathway to pathway conduction 5) AVNRT with bystander pathway conduction 6) ORT with bystander pathway activation 7) Ventricular tachycardia 8) Junctional tachycardia with aberrancy or fasciculo-ventricular connection and 9) Nodofascicular or nodoventricular tachycardia.

In this case, the baseline ECG is suggestive of a posteroseptal atrio ventricular accessory pathway and the tachycardia QRS morphology closely approximates the expected QRS morphology during anterograde conduction through such a pathway (Fig. 1A). Thus antidromic tachycardia utilizing the posteroseptal accessory pathway is likely. Classical antidromic tachycardia (ART) which involves the Purkinje system in the retrograde direction is a very rare arrhythmia with a septal pathway because of the closeness of the pathways involved. Generally, an anatomical separation of 4 cm between the pathways along the annulus is required for maintaining an antidromic tachycardia [1]. In this case the induction of tachycardia in Fig. 1B shows that it follows maximum pre excitation following the extra stimulus beat and the QRS morphology of tachycardia is similar to the QRS resulting from the paced beat. The HV interval (Fig. 2) was negative (−60) showing that it was a pre

* Corresponding author.
E-mail address: dranees2001@gmail.com (A. Thajudeen).
Peer review under responsibility of Indian Heart Rhythm Society.

http://dx.doi.org/10.1016/j.ipej.2017.01.004

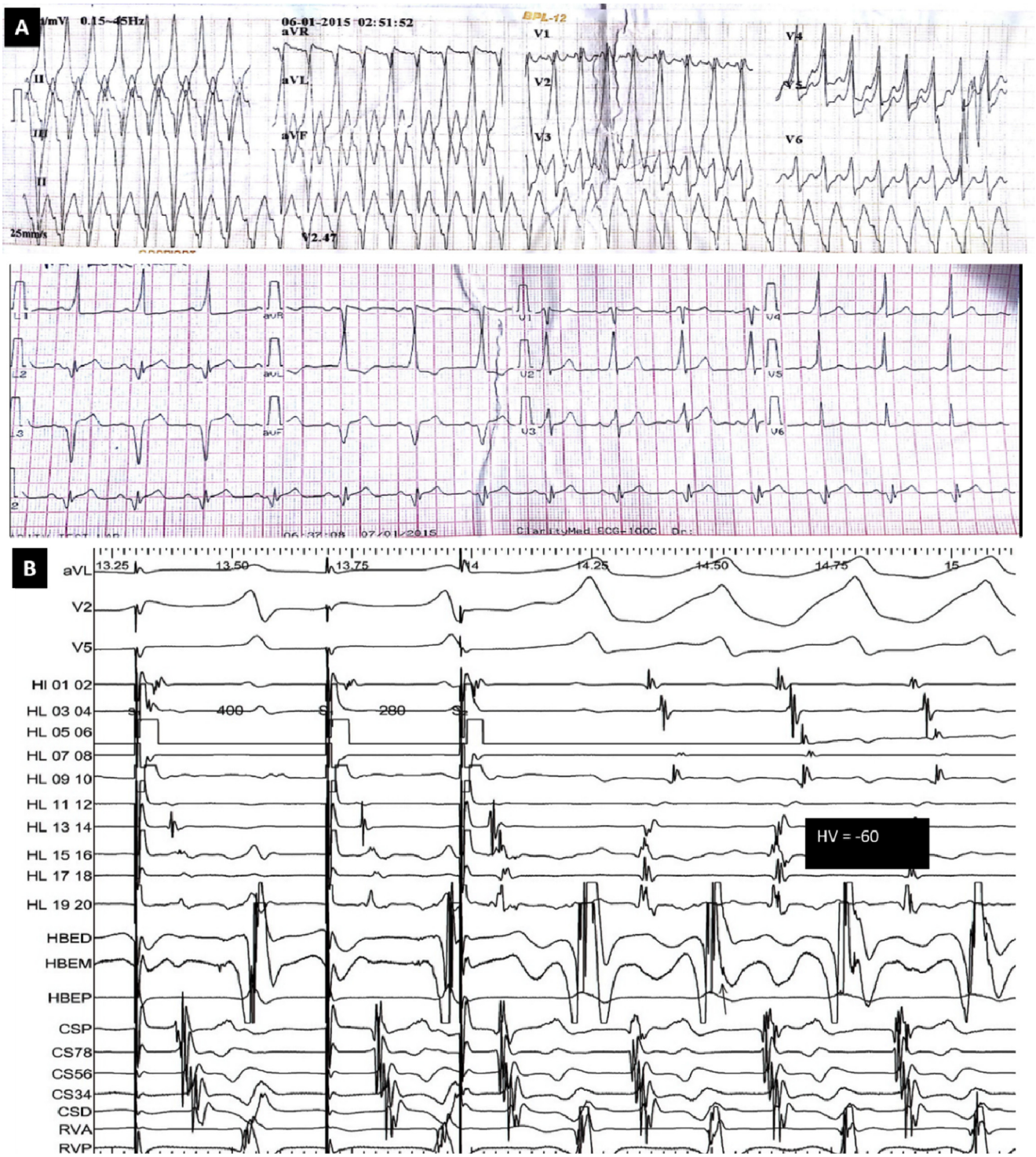

Fig. 1. A)Baseline 12 lead ECG showing manifest preexcitation via posteroseptal atrio ventricular accessory pathway and the tachycardia 12 lead ECG showing QRS morphology closely approximating the expected QRS morphology during anterograde conduction through such a pathway.B) Selected surface ECG leads and intracardiac recording from right atria (Halo cathter – HL 0102 to HL 19 20), His Bundle (HBE distal, mid and proximal) and coronary sinus (CS Proximal – to CS Distal) showing induction of broad QRS tachycardia with atrial extra. Tachycardia initiation follows maximum pre excitation in the last paced beat and the QRS morphology of tachycardia is similar to the QRS resulting from the paced beat. The arrow heads represent retrograde His deflections during tachycardia and HV interval during tachycardia was -6oms.

excited tachycardia including atrial tachycardia (AT) or atrial flutter with preexcitation and antdromic tachycardia. An atrial tachycardia with pre excitation was ruled out by a ventricular pacing maneuver (Fig. 2) which showed entrainment with V-A-V response and because after atrial entrainment the VA interval of the first returning beat was same as during the subsequent beats. In the case of wide QRS tachycardia, if it was AT, a narrow QRS after the last paced beat is possible. This indicates retrograde block in the His

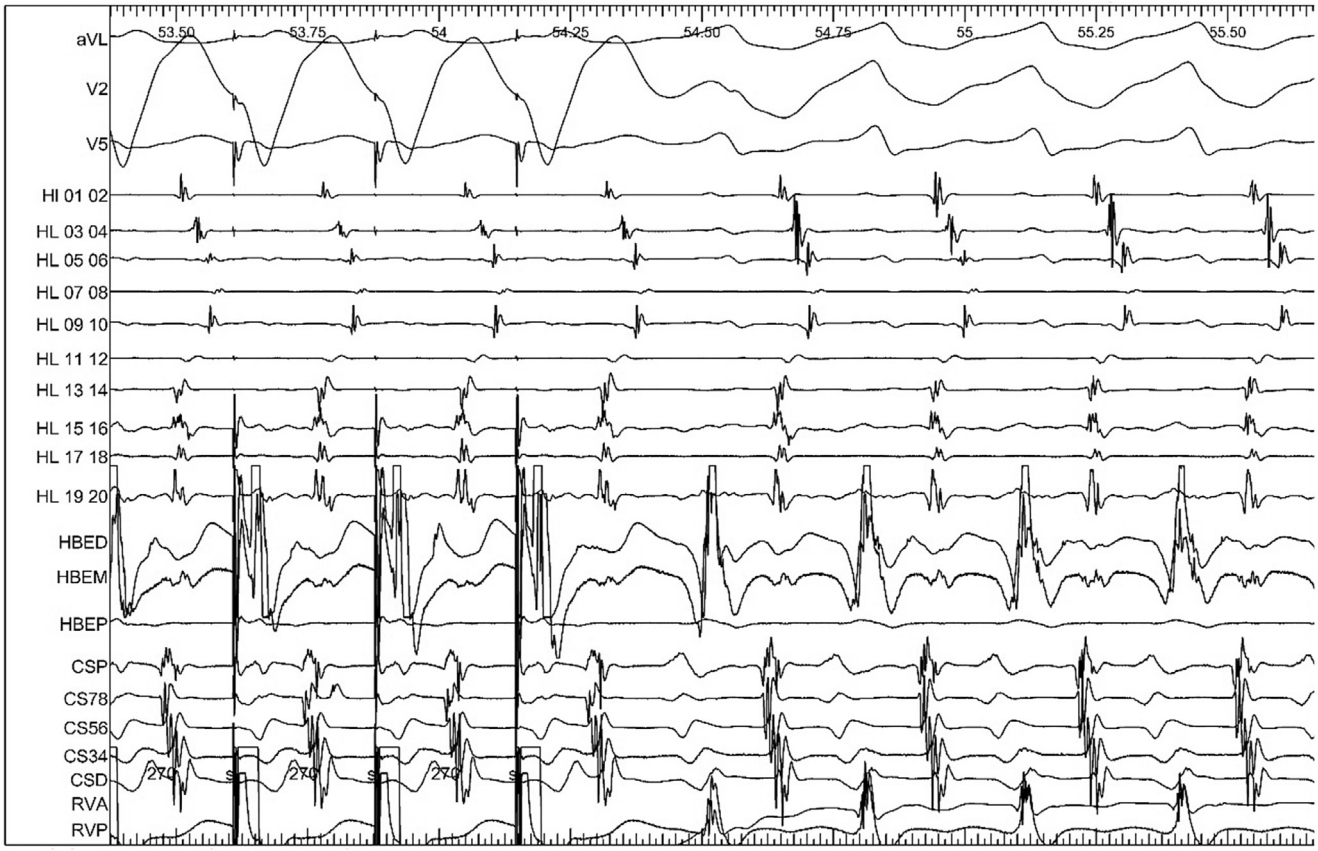

Fig. 2. Selected surface ECG leads and intracardiac recording from right atria (Halo cathter – HL 0102 to HL 19 20), His Bundle (HBE distal, mid and proximal) and coronary sinus (CS Proximal – to CS Distal) showing entrainment by V pacing – showing VAV response, note that the atrial activation is same during tachycardia and pacing and the first V after pacing is wide, and the tachycardia continues without termination by pacing.

purkinje system during pacing and conduction via the accessory pathway. This happens only with termination of tachycardia and the actual return beat of the tachycardia starts with the atrial electrogram and pre excited ventricular electrogram (broad QRS) occurs follows after the narrow QRS. Atrial entrainment and re setting, with the same QRS morphology during atrial pacing ruled out a ventricular tachycardia. Ventricular pacing at baseline showed central decremental VA conduction. A parahisian pacing maneuver was done to examine the nature of VA conduction. As shown in Fig. 4A, the parahisian pacing showed an AV nodal response, implying that the AV accessory pathway conducts only anterogradely. In this case the anterograde conduction was good but the retrograde could not be demonstrated by ventricular pacing or parahisian pacing maneuvers. Nor was an orthodromic tachycardia ever inducible. All this proves that retrograde conduction was absent for all practical purposes. Beyond this it is not possible to demonstrate unless we can suppress spontaneous sinus nodal and AV nodal activity with drugs which may also affect pathway conduction. This is an important finding in the case, which would explain the easy induction of a wide QRS rhythm even though the pathway was septal in location. This also rules out a duodromic tachycardia.

Another important maneuver which is required to be done is an atrial extra stimulus given during late in the cycle during tachycardia (Fig. 3) - the extra stimulation is given near the atrial end of the pathway and is timed such that the retrograde atrial activation over the septal region is not disturbed. The stimuli captured part of the atria close to the pathway and advanced the next V and the resultant retrograde A, thus re setting the tachycardia. This shows that the pathway is integral to the tachycardia and rules out AVNRT with bystander accessory pathway conduction or a nodoventricular pathway. It also excludes VT as the underlying mechanism. Thus we made a certain diagnosis of classical antidromic tachycardia mediated by a posteroseptal antegrade only conducting pathway.

The accessory pathway was epicardial and was localized around the neck of a diverticulum (Fig. 4B) in the coronary sinus. The signal at the successful ablation site at the neck of the diverticulum showed no apparent fusion in sinus rhythm, but ablation was immediately successful. There was loss of pre excitation and no further induction of any tachycardia.

Classical ARTs by posteroseptal pathways are very rare and can happen if pathways have very short refractory periods [1,2]. It can also manifest if one of the limbs have a slow or decremental conduction property as when the retrograde conduction involves a slow AV nodal pathway or if the anterograde pathway has decremental property [3,4]. In our case two factors predisposed to easily induced and sustainable ART by the posteroseptal pathway – 1) the pathway had no retrograde conduction and 2) the pathway was epicardial in location and related to a coronary sinus diverticulum – allowing for more functional delay in conduction from atria to ventricle. An early ventricular extra stimulus advanced the retrograde A significantly enough that the subsequent AV got prolonged showing that the pathway also had decremental property. Progressive pre excitation and longer AV interval during shorter cycle

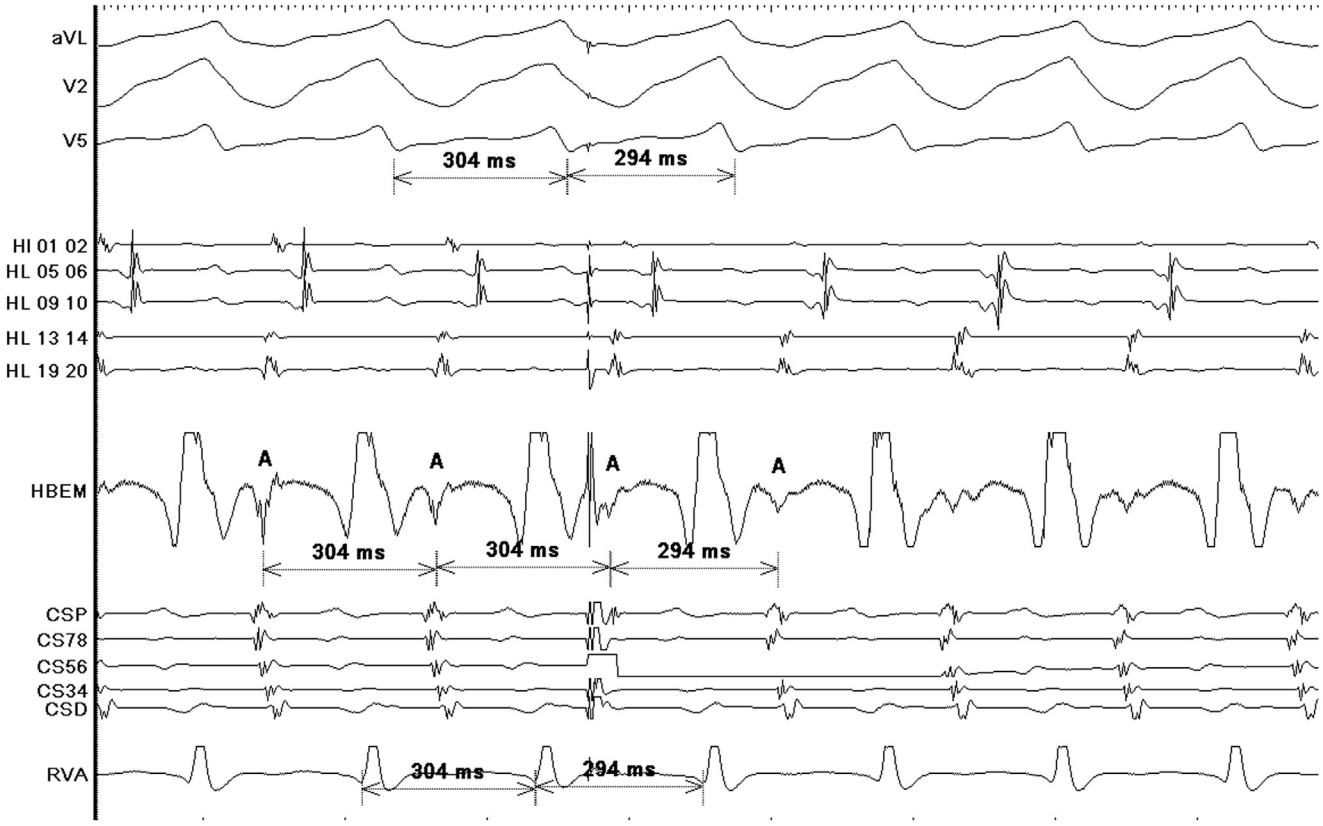

Fig. 3. Selected surface ECG leads and intracardiac recording from right atria (Halo cathter – HL 0102 to HL 19 20), His Bundle (HBE distal, mid and proximal), coronary sinus (CS Proximal – to CS Distal) and the Right ventricle (RVA and RVP). A late atrial extra stimuls given from the CS catheter (CS 5,6) did not affect the septal atrial activation, shown by electrogram recorded in HBEM and HL 19 20. However it advanced the next ventricular activation and reset the tachycardia without changing the QRS morphology.

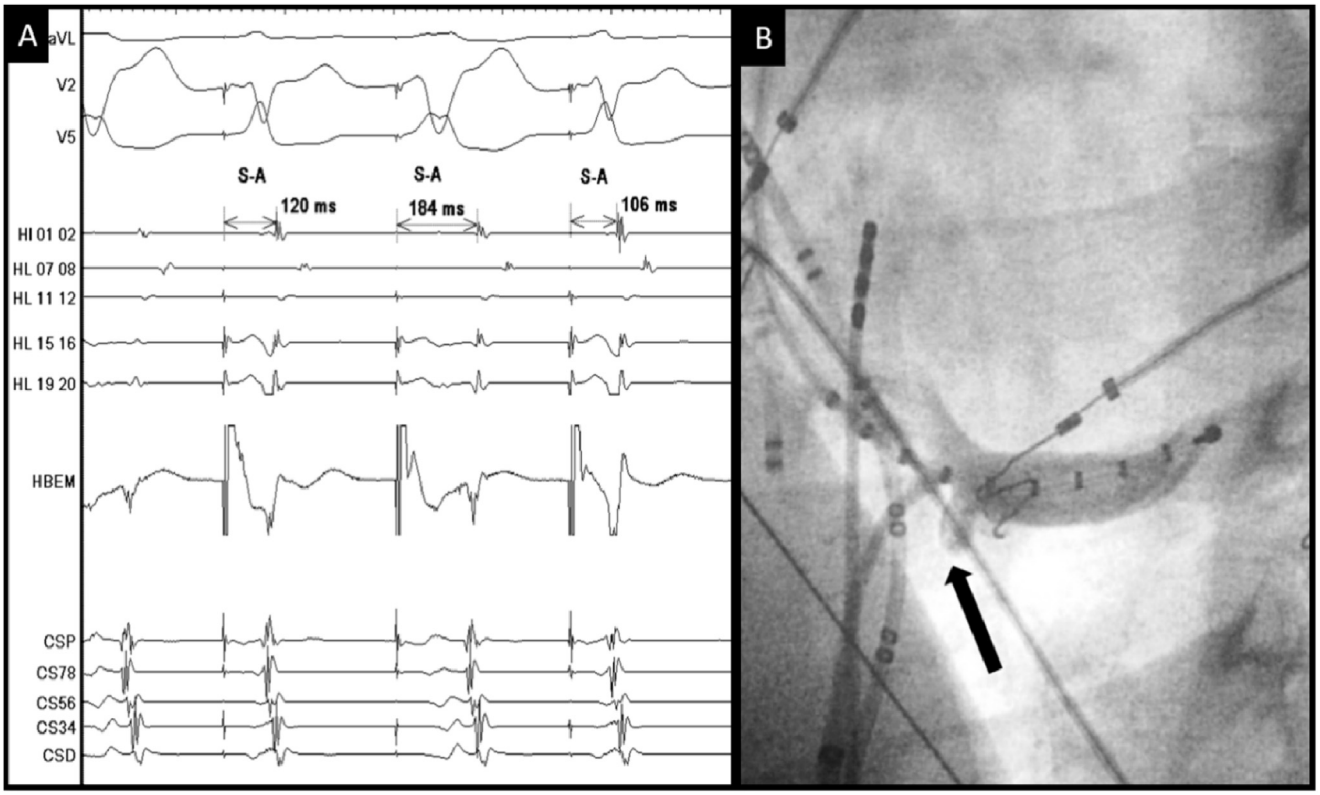

Fig. 4. A) Selected surface ECG leads and intracardiac recording from right atria (Halo cathter – HL 0102 to HL 19 20), His Bundle (HBE, mid) and coronary sinus (CS Proximal – to CS Distal) Parahisian pacing, showing different VA intervals with and without his capture(narrow and wide surface QRS) showing the absence of retrograde VA conduction through a pathway. B) Coronary sinus venogram using a diagnostic catheter showing a diverticulum near the CS ostium (Black arrow). The pathway was succesfully ablated at the mouth of the diverticulum.

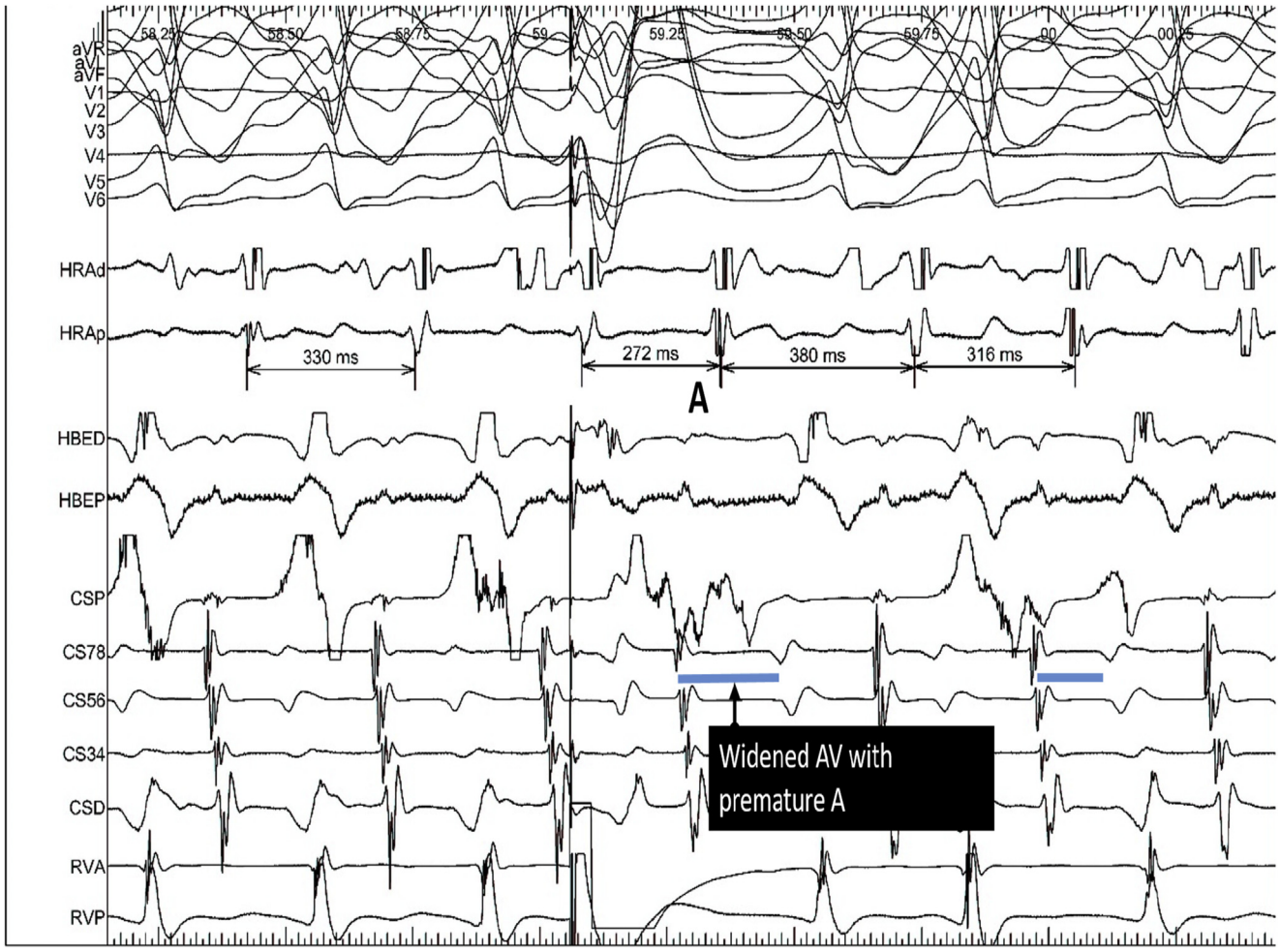

Fig. 5. Surface ECG leads and intracardiac recording from right atria (Halo cathter – HL 0102 to HL 19 20), His Bundle (HBE, mid) and coronary sinus (CS Proximal – to CS Distal) showing the ventricular extra beat advanced the A – this reset the tachycardia with a long AV delay indicating that the pathway was showing decremental property.

length pacing could not be demonstrated as patient was easily going into tachycardia. Instead, decremental conduction was inferred from ventricular extra during tachycardia (Fig. 5). The ventricular extra beat advanced the atrial electrogram – this reset the tachycardia with a long AV delay indicating that the pathway was showing decremental property as shown in the figure. There was no delay in VA during the extra or the reset beat. There was no change in VA interval, only in the AV interval to account for the increase in CL from 330 ms to 380 ms, as shown in the figure. Absence of retrograde conduction, a property similar to atrio fascicular (Mahaim) pathways, predisposes to easy induction of antidromic tachycardia by atrial or ventricular extra stimuli. Thus a classical antidromic tachycardia through an anterograde only conducting epicardial pathway, within a coronary sinus diverticulum, with decremental property was diagnosed. Ablation resulted in loss of pre excitation and there was no subsequent inducible tachycardia.

Such epicardial pathways with antegrade only conduction and Mahaim like properties are rarely described in relation to Coronary sinus diverticulum.

Conflict of interest

none.

References

[1] M E Josephson. Pre excitation syndromes, in Clinical cardiac electrophysiology techniques and interpretations; fourth ed.. Pages 366–367.
[2] Man DC, Sarter BH, Coyne RF, Callans DJ, Schwartzman D, Gottlieb CD, et al. Antidromic reciprocating tachycardia in patients with paraseptal accessory pathways: importance of critical delay in the reentry circuit. Pacing Clin Electrophysiol 1999 Feb;22(2):386–9.
[3] Bardy GH, Packer DL, German LD, Gallagher JJ. Preexcited reciprocating tachycardia in patients with Wolff-Parkinson-White syndrome: incidence and mechanisms. Circulation 1984 Sep 1;70(3):377–91.
[4] Packer DL, Callagher IJ, Prystowsky EN. Physiologic substrate for antidromic reciprocating tachycardia. Circulation 1992;85:574–88.