Chapter 15
Mahaim Accessory Pathway

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Abstract In any young patient with wide QRS tachycardia with LBBB pattern without structural heart disease, when SVT is considered be aware of Mahaim.

A 27-year-old man presented with frequent episodes of palpitation, without syncope. Some episodes of palpitations were during exercise and another episodes were occurred during rest without any known precipitating factor. Electrocardiography during palpitation revealed a wide QRS tachycardia with LBBB morphology and superior axis. Arrhythmia was adenosine sensitive and several episodes of arrhythmia were terminated with adenosine injection at the emergency ward. Patient was referred for ablation and electrophysiology study was revealed that arrhythmia is contributed to the atriofascicular accessory pathway (Mahaim) and successful ablation of the accessory pathway was done.

ECG demonstrated a wide QRS tachycardia with LBBB morphology with rate of about 170 bpm and left axis deviation. There is no evidence of AV dissociation. Morphology criteria were infavor of SVT. There was not any evidence of broad R wave in V1 or R/S nadir more than 70 ms in V1–V2 and QR or QS in V6.

EPS

Atrial overdrive pacing resulted in AH prolongation HV interval shortening and LBBB appearance of QRS (Fig. 15.1).

AES (Atrial extra stimulation) demonstrated LBBB morphology and AH prolongation with HV interval shortening all consistent with decremental conducting atriofascicular accessory pathway (Fig. 15.2).

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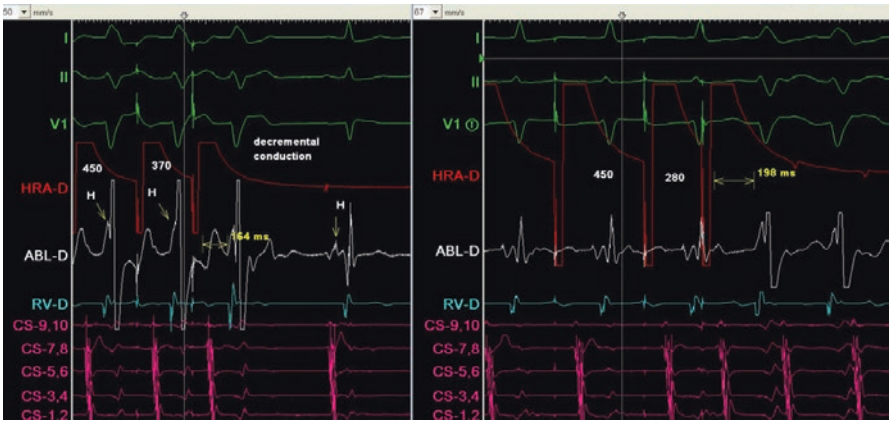

Fig. 15.1 With HRA pacing AH prolongation, HV shortening and LBBB pattern is seen

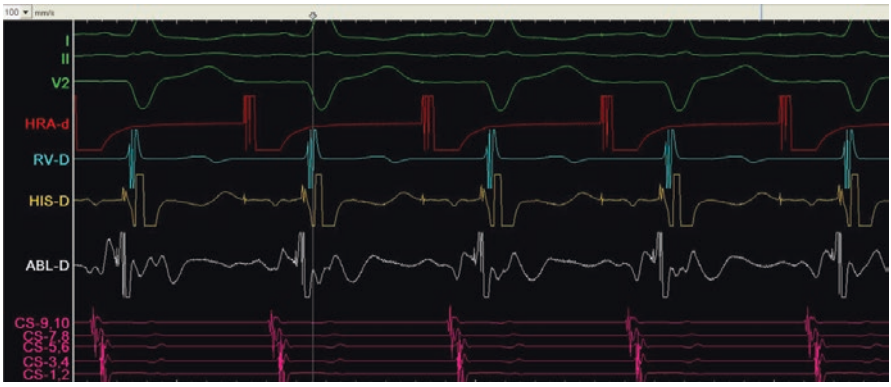

Fig. 15.2 LBBB pattern and HV shortening in Mahaim

With ventricular stimulation, a wide QRS tachycardia with LBBB morphology, similar to the patient's documented arrhythmia was initiated with negative HV interval (about 12 ms) and 1:1 AV association and earliest site of atrial activation in His consistent with antidromic AVRT due to right atriofascicular accessory pathway (Mahiam accessory pathway) (Figs. 15.3 and 15.4).

Patient's basic ECG evaluation demonstrated no evidence of preexcitation during sinus rhythm in favor of decremental conduction property of the accessory pathway.

Catheter ablation with radiofrequency was done during atrial stimulation with targeting the M potential in the lateral site of the tricuspid ring. During ablation ventricular automaticity was appeared, similar to the junctional rhythm during AVNRT ablation (Fig. 15.5).

After ablation no arrhythmia was inducible with AES or VES. Accessory pathway was not appeared as at the lateral side of the TV ring with atrial stimulation (Fig. 15.6).

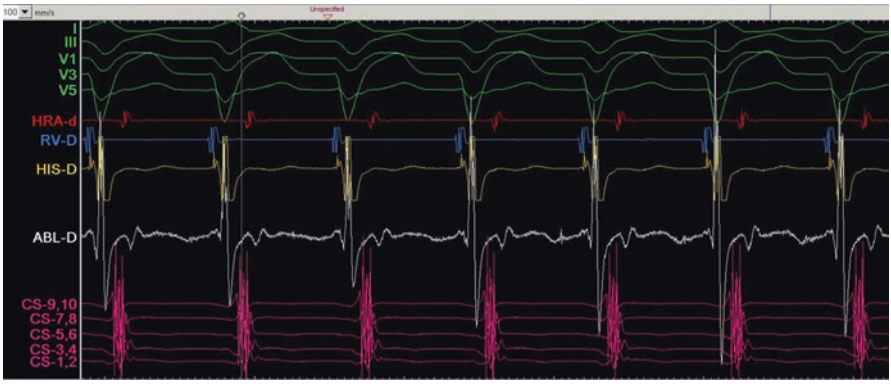

Fig. 15.3 Antidromic AVRT in Mahaim

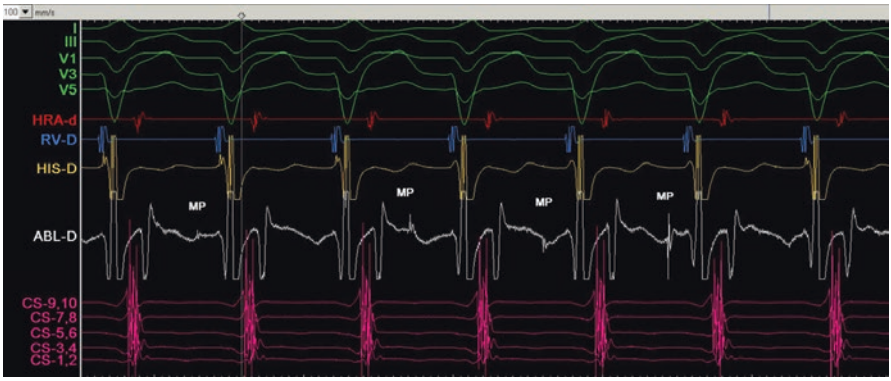

Fig. 15.4 Mahaim potential on the catheter ablation that is positioned in the lateral side if TV ring

Fig. 15.5 Mahaim automaticity similar to AV automaticity during RFA

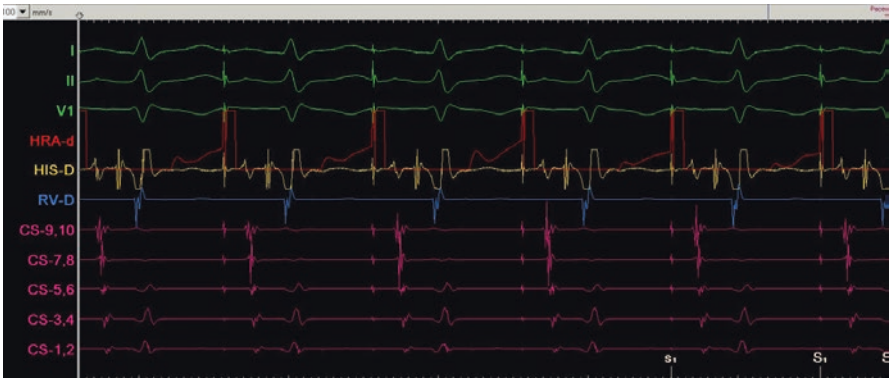

Fig. 15.6 No evidence of accessory pathway with atrial stimulation after ablation

Discussion

The first time, in 1938, nodoventricular fibers were described by Dr. Mahaim as fibers that connected from nodal region to the ventricular region by bypassing the His system. It was only a histologic description without any physiologic importance, but in early 1980, antidromic reciprocating tachycardias due to slowly conduction atrioventricular and atriofascicular accessory pathways were described and eponym of Mahaim was adopted for such fibers contributing in such arrhythmia [1–3].

These fibers are long, insulated fibers with decremental conduction properties that connect atrium directly to the distal part of His purkinje system. This structure is very similar to the nodal system with decremental conduction properties. Except for a single report of the left side Mahaim pathway, these pathways are almost always located at the right side, along the lateral part of the tricuspid annulus.

These pathways are associated with unique tachyarrhythmia using the accessory pathway as the antegrade limb and AV node as the retrograde limb, resulting in antidromic AV reciprocating tachycardia with LBBB morphology [4–6].

They can only conduct antegrade, and are mostly right-sided, giving rise to an antidromic AVRT with LBBB type morphology, which is sensitive to adenosine and verapamil. Similar to the AV node, there are some reports of ducal conduction properties in such accessory pathways [7].

Maximally preexcited QRS, during rapid atrial pacing or during antidromic tachycardia, is very similar to typical LBBB, without broad R wave in V1, with mid precordial R were transition (beyond V4), and an R wave in lead I. QRS duration during tachycardia is less than 150 ms, because atriofascicular pathway allows for more rapid activation to the His-Purkinje system. The distal insertion of atriofascicular pathways is at the distal part of the right bundle in the anterior free wall of

the right ventricle. Similar to the AV node, decremental conduction in the atriofascicular pathways occur in the proximal portion, between the atrial insertion and the M potential. Initiation of the tachycardia could be done by atrial or ventricular stimulation.

In the atrial stimulation initiation of the tachycardia may be more easily done by atrial stimulation near the AV node, such as proximal of coronary sinus stimulation, because it may allow more time for AV node for recover and facilitate retrograde conduction. Because initiation of tachycardia by atrial stimulation requires AV nodal block for conduction through the accessory pathway, and because these accessory pathways cannot conduct retrogradely, ventricular stimulation can almost always initiate the tachycardia [8].

Ventricular stimulation when could result in sufficiently short retrograde V-H interval, or sufficient delay on retrograde HPS activation because of retrograde RBBB could result in the initiation of the tachycardia. The AV interval during antidromic tachycardia is often more than 150 ms. Direct insertion of the pathways to the distal RBB or fascicle results in the rapid simultaneous activation of His and ventricular with a very short V-H interval the V-H interval during tachycardia is shorter than V-H interval during sinus rhythm, because of the simultaneous activation of the His and the ventricular via right bundle [9, 10].

In the setting of the retrograde RBBB, V-H, and V-A interval may be prolonged and in this situation, the tachycardia cycle length increase, because the conduction occurs across interventricular septum via myocardium and then up to system via left bundle to reach to the His bundle and then to the atrium. In the absence of retrograde RBBB, rapid retrograde conduction over the right bundle to the His may allow for antegrade left ventricular activation via left anterior fascicule, resulting in a narrower QRS with the inferior axis.

A late PAC, could preexcite the tachycardia and advance or delay the following ventricular activation without affecting the septal atrial activation, and reset the tachycardia. Cycle length oscillation following extrastimulus is common and is characteristic for such pathways. Mapping of the atriofascicular accessory pathway is done by locating to the M (Mahaim) potential along the tricuspid ring during atrial stimulation or during antidromic tachycardia.

The site of successful ablation is defined by simultaneous of the A, M, and RV potential in the lateral part of the tricuspid annulus [11].

In some situations ablation of the atrial insertion of the accessory pathway is done that can be achieved by recording the shortest stimulus to the fully preexcited ventricular electrogram, or by mapping the tricuspid annulus during antidromic AVRT. The second one could be done by mapping of the site where the latest PAC could reset the tachycardia cycle length without activating the atrial tissue near the AV node.

Radiofrequency ablation of atriofascicular pathways has a success rate of about 90–100%.

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