Atrioventricular Reentry Tachycardia

14

Eun-jeong Kim and Gregory F. Michaud

Introduction

Atrioventricular reentry tachycardia (AVRT) is a supraventricular tachycardia (SVT) that involves anomalous pathways between atrium and ventricle that bypass normal conduction via the atrioventricular (AV) node. The pathways create a direct AV communication and may prompt an earlier ventricular activation than normal conduction, manifesting as ventricular preexcitation. In 1930, Wolff, Parkinson, and White described a syndrome of short PR and bundle branch block in young patients prone to SVT [1]. Subsequently, the syndrome correlated with an anatomic substrate of cardiac myocytes bridging the fibrous AV junction. Several other unusual variants of APs have been discovered, and these are best described by their anatomic relationships and not the historical names which have been inconsistently ascribed to particular APs. Atriofascicular and nodofascicular pathways are decremental and insert into or near the right bundle branch, producing SVT with left bundle branch block. Nodoventricular pathways are decremental and may insert into the right or left ventricle. Unusual variants that do not participate in tachycardia are atrio-Hisian and fasciculoventricular pathways.

E.-j. Kim (✉)
Vanderbilt Heart and Vascular Institute, Vanderbilt University Medical Center, Nashville, TN, USA
e-mail: eunjeong.kim@vumc.org

G. F. Michaud
Division of Arrhythmia, Vanderbilt Heart and Vascular Institute, Vanderbilt University Medical Center, Nashville, TN, USA

G.-X. Yan et al. (eds.), *Management of Cardiac Arrhythmias*, Contemporary Cardiology, https://doi.org/10.1007/978-3-030-41967-7_14

Clinical Features

An ordinary accessory pathway (AP) is a group of muscle fibers bridging the AV groove, providing electrical connection between atrium and ventricle and bypassing the AVN and His-Purkinje system [2]. It can be found anywhere along the mitral valve or tricuspid annulus except for the left anteroseptal region in the aortomitral continuity. The most frequent location is left lateral (46–60%), followed by posteroseptal (25–30%), right lateral (13–21%), and anteroseptal (2–5%) [3]. Most APs can conduct in both antegrade and retrograde directions (60–75%). However, some APs are only able to conduct unidirectionally. When an AP is only able to conduct in retrograde fashion (ventricle to atrium), it is called a "concealed accessory pathway" and does not demonstrate preexcitation on ECG. The majority of concealed pathways are left-sided [4]. The least common form of AP is capable of antegrade conduction only. If tachycardia is poorly tolerated symptomatically and/or hemodynamically, it requires a definitive approach with catheter ablation.

Most APs have non-decremental conduction properties except for uncommon exceptions. AP tissue is similar to atrial, His-Purkinje, or ventricular tissue where depolarization relies on rapid inward sodium current; thus, rapid conduction occurs until it is blocked at the refractory period (i.e., non-decremental). In contrast, the AV node and some APs are rich in inward calcium current and exhibit decremental conduction with incremental pacing. Due to the non-decremental, rapid conduction of most APs, very rapid ventricular rates during atrial fibrillation can cause degeneration in ventricular fibrillation [5]. Widely different properties between APs and the AV node establish the foundation for differentiating AVRT from any other supraventricular tachyarrhythmias, except for a minority of APs with AV node-like properties. AVRT can be classified based on different properties and anatomic substrates (Table 14.1). It may be associated with a clinical syndrome such as hypertrophic cardiomyopathy and Ebstein's anomaly. Management of AVRT depends on the

Table 14.1 Classification of an accessory pathway

Directionality	Antegrade Retrograde (concealed) Both antegrade and retrograde
Location	Left free wall Septal Right free wall
Anatomic origin and insertion sites	Atrioventricular Atrio-Hisian Atriofascicular Nodofascicular Nodoventricular Fasciculoventricular
Conduction property	Fast conducting, non-decremental Slow conducting and decremental

Antegrade only: atrio-Hisian, atriofascicular, fasciculoventricular
Decremental: atriofascicular, nodofascicular, nodoventricular
No participation in SVT: atrio-Hisian, fasciculoventricular

clinical picture, symptom severity, and hemodynamic effect of tachycardia. With high success and low complication rates, catheter ablation remains the treatment of choice for AVRT. Understanding of anatomic substrate and properties is key to successful ablation.

ECG Manifestation and Localization

Classic ECG features of WPW are short PR interval, wide QRS complex, and a delta wave, which represent the initial relatively slow preexcitation of ventricular tissue at the AP insertion point prior to engagement of the His-Purkinje system over the AV node. These findings are only seen with antegrade conduction over AP. The QRS morphology is thus a fusion complex of ventricular preexcitation through the pathway as well as normal His-Purkinje conduction. Enhanced AV nodal conduction that occurs with exercise usually reduces the degree of preexcitation or eliminates it altogether. APs remote from the sinus node, such as a left lateral AP, may result in minimal to no preexcitation. In this case, subtle findings such as absent septal Q in V6 can suggest minimal preexcitation (Fig. 14.1) [6]. Atrial pacing to prolong AV nodal conduction time or CS pacing to decrease AP conduction time can exaggerate the delta wave in subtle cases.

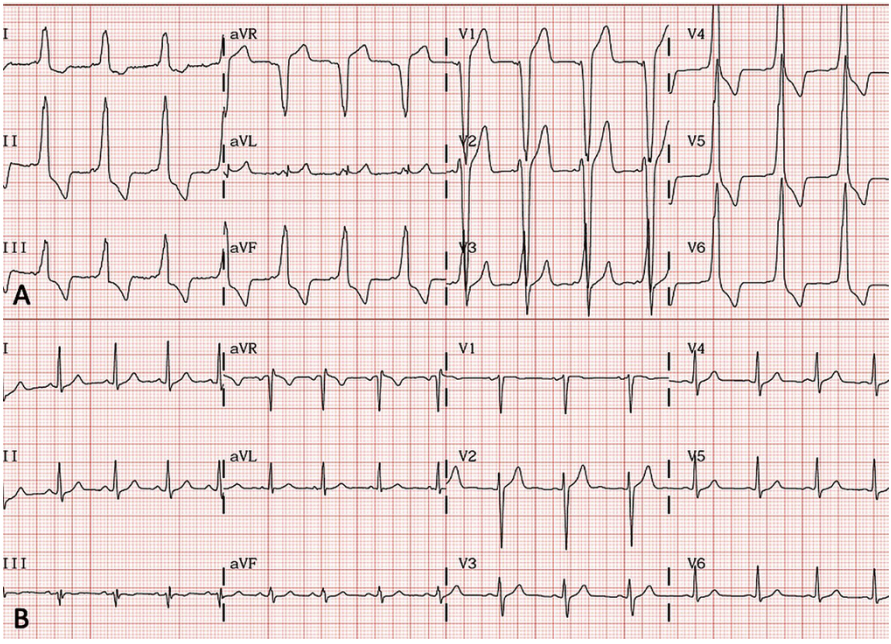

Fig. 14.1 Electrocardiogram of accessory pathway. (**a**) Right anterior pathway (**b**) Left lateral pathway. Right-sided pathway tends to manifest classic findings of short PR and wide QRS with delta wave, while left lateral pathway demonstrates minimal preexcitation. Absent septal Q wave in V5 and V6 can be a subtle finding of preexcitation

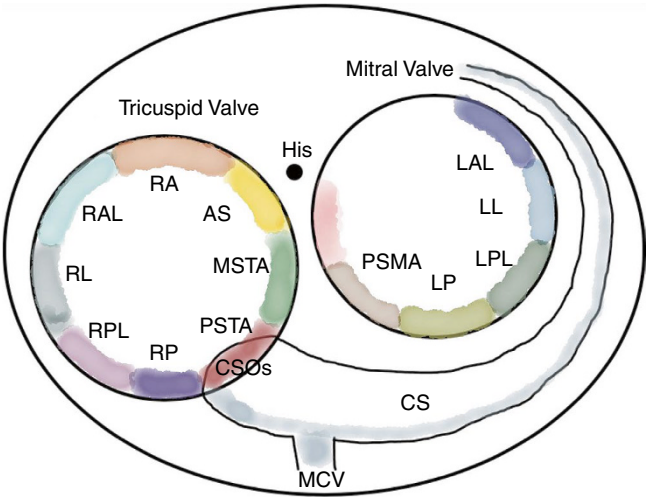

Fig. 14.2 AV accessory pathway localization. RA right anterior, AS anteroseptal, MSTA midseptal tricuspid annulus, PSTA posteroseptal tricuspid annulus, RP right posterior, RPL right posterolateral, RL right lateral, RAL right anterolateral RA right anterior, LAL left anterolateral, LL left lateral, LPL left posterolateral, LP left posterior, PSMA posteroseptal mitral annulus, LS left septal [7]

Baseline ECG

ECG pattern of preexcitation can be helpful to localize the AP by evaluating (1) delta wave in sinus ECG if present and (2) P wave morphology during orthodromic reciprocating tachycardia (ORT). Multiple algorithms have been developed that help localize the AP (Fig. 14.2) [7–10]. Although it is useful to understand the algorithm, there are limitations in their application when there is a lack of clear delta wave or presence of multiple accessory pathways.

The main principle of pathway localization involves inspecting the horizontal QRS axis, QRS transition (V1–V4), and delta wave polarity. A large R wave in V1 or negative delta wave in the lateral limb leads indicates a left-sided pathway. If the transition occurs at V2, pathways are likely overlying the right posterior or midseptum. If the transition is late, it is more likely to be a right-sided pathway. Delta wave polarity helps delineate an anterior vs. inferior location of pathways. If delta polarity is (+) in aVF, it is more likely to be anterior and if it is (−) or isoelectric in aVF, it is likely located inferiorly (posteriorly).

- Posteroseptal (PS): Leftward and superior delta wave, Rs/RS in V1–V3, sum of delta wave polarity in inferior leads (II, III, aVF) δ-2.
  - If delta wave is steeply negative in II and steeply positive in aVR with deep S wave in V6, it suggests an epicardial posteroseptal AP in the middle cardiac vein, coronary sinus, or diverticulum [11].
- Mid-septal (MS): Sum of delta wave polarities in inferior leads (II, III, aVF) –1 to +1.

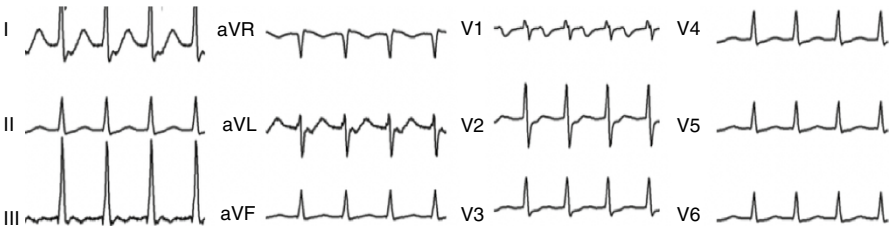

Fig. 14.3 P wave axis during tachycardia. During an ORT with left lateral pathway, P wave has a rightward axis (positive aVR and negative aVL)

- Right anteroseptal (RAS): Inferior delta wave with sum of delta wave polarity in inferior leads ε +2, away from leftward leads (Q or isoelectric in I, V6, aVL) and LBBB, +30° δ QRS axis < 120°
- Right lateral (RL): Posterior and leftward delta wave with late QRS transition ε V4 and LBBB type (no Rs, RS in V1–V3) with −120° < QRS axis ≤ +30°.
- Left lateral (LL): RBBB type and negative lateral leads.
- Para-Hisian AP: Inferior delta wave (positive in I, II, aVF) and negative delta wave in V1–V3 [12].

P Wave During Tachycardia

P wave analysis during orthodromic reciprocating tachycardia can be helpful for localization (Fig. 14.3).

- Rightward P wave (positive aVR and negative aVL) for left-sided AP
- Midline, superior P waves for mid- or posteroseptal AP
- Midline, inferior P waves for anteroseptal AP
- Leftward axis (negative aVR, positive aVL) for right-sided AP

Rhythm Tracing Analysis

Analyzing the rhythm ECG tracing can provide clues to narrow the differential diagnosis of supraventricular tachycardia by observing the behavior of the rhythm, especially during transitions.

Initiation

If the initiation has a gradual increase in the heart rate ("warm-up period"), it is more likely an automatic atrial tachycardia (AT). Abrupt onset following an atrial premature depolarization (APD) favors a reentrant SVT, but the differential diagnosis remains broad: atrioventricular nodal reentry tachycardia (AVNRT), AVRT,

automatic, or reentry AT. If initiation is preceded by late-coupled ventricular premature depolarization (VPD), it favors ORT or atypical AVNRT.

During Tachycardia

If spontaneous AV block or AV dissociation is observed, AVRT is essentially excluded. Occasionally 2:1 AV block occurs during AVNRT, particularly in the EP lab as a transient phenomenon facilitated by long-short induction sequences. However, if variable AV block is observed, the most likely diagnosis is AT. Rare exceptions to this rule would include pathways arising at the level of or distal to the AV node including nodoventricular, nodofascicular, and fasciculoventricular pathways or unusual AVNRT. QRS alternans or beat-to-beat variation in R wave amplitude (ε 1 mm) during tachycardia can be observed. Presumably, this phenomenon is driven by oscillations in the relative refractory period of the His-Purkinje system. There is some evidence that QRS alternans suggests ORT, but it is more likely a rate-related phenomenon and not specific for the type of tachycardia [13].

Termination

Tachycardia terminating with a gradual deceleration ("cool-down period") suggests an automatic mechanism, while a sudden termination more likely indicates reentrant or triggered tachycardia. If tachycardia reproducibly terminates with AV block with P wave being the last complex, it is highly likely to be an AV node-dependent tachycardia (e.g., AVNRT, ORT), and AT becomes unlikely since the chance of simultaneous block in atrium and AV node is very low. If tachycardia terminates with VA block (last complex is QRS) following a late-coupled PVC that fails to reach atrium, it is more likely to be ORT.

Unusual Accessory Pathways

Unusual AP variants that can participate in SVT are atriofascicular, nodofascicular, and nodoventricular fibers with distinct functional characteristics (Fig. 14.4). Atriofascicular fibers participate in tachycardia with a LBBB pattern, with decremental and generally antegrade-only conduction although there are case reports of decremental AP that conducts retrogradely and blocks at the proximal insertion site (AP potential not followed by atrial activity) [14, 15].

Atriofascicular fibers share AV node-like properties such as decremental conduction, conduction block from adenosine, heat-induced automaticity during RF catheter ablation, as well as connection to His-Purkinje tissue that generates a distinct "His"-like accessory pathway potential at the AV groove, usually at the lateral TV annulus. Generally, preexcitation in sinus rhythm is subtle or absent. Minimal preexcitation with rS pattern in lead III is seen in approximately 50% of patients, or

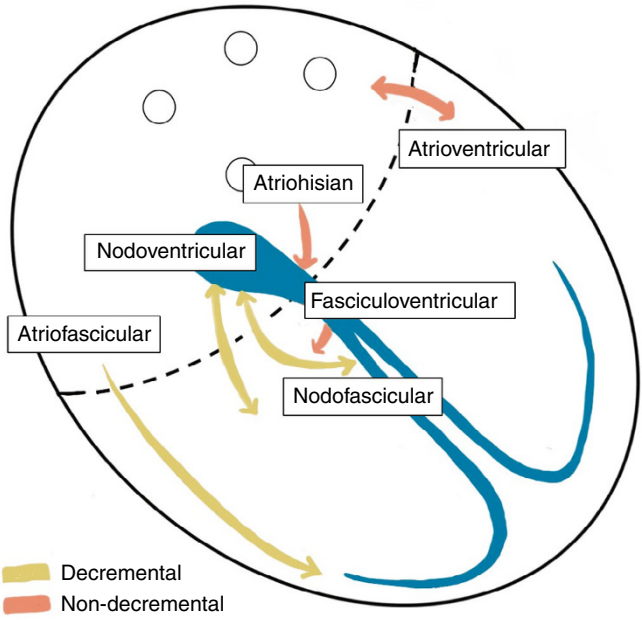

Fig. 14.4 Types of accessory pathways

loss of septal Q in lateral leads may be the only finding [16, 17]. Further details of electrophysiologic properties and implications are discussed later in the chapter.

Electrophysiologic Study

An invasive electrophysiology study is indicated to understand the mechanisms of arrhythmia and localize the successful site for catheter ablation. There are multiple mechanisms of SVT including reentry, automaticity, and triggered activity. AVRT is a reentrant arrhythmia, and it can be initiated with a variety of pacing maneuvers or pharmacologically with a beta agonist such as isoproterenol. The preexcitation in ECG manifests as short (<35 ms) or negative HV and long VA (ε70ms), which is the ECG equivalent of a long RP interval [3]. Typically, accessory pathways are non-decremental or minimally decremental, and the circuit involves atrium, AVN and His-Purkinje system, ventricle, and AP. Rare exceptions to these rules are discussed later in the chapter.

Retrograde Atrial Activation Sequence

Understanding the atrial activation sequence is important in the differential diagnosis for narrow complex tachycardia. Typically, atrial activation through the AV node

demonstrates earliest atrial activation in the anterior septum just posterior to the His bundle. This pattern of activation is also seen in typical forms of AVNRT, ORT using anteroseptal AP, automatic junctional tachycardia (JT) with retrograde conduction via the fast AV nodal pathway, or septal AT. If the earliest site of atrial activation is seen in the inferior septum in the CS os recording, differential diagnosis includes atypical AVNRT, ORT utilizing posteroseptal AP, AT arising near CS os, or JT with retrograde slow pathway. Most forms of ORT have eccentric retrograde conduction, and the earliest atrial activation site depends on the pathway location. Accessory pathways located on the right free wall of the tricuspid annulus have the earliest atrial activity in the lateral RA catheter, while left lateral AP have earliest atrial activation in the distal CS catheter.

However, there are exceptions to these rules. AVNRT may display eccentric atrial activation pattern via left inferior nodal extension, slow CS conduction, or bystander Marshall bundles [18–22]. Some septal pathways demonstrate decremental properties, making a diagnosis challenging.

Atrial Extrastimulus (AES)

Atrial extrastimuli (AES) are APDs delivered at shorter coupling intervals with 10 ms decrements after a drive chain (typically eight beats) of fixed cycle length or during spontaneous rhythm. AES can assess the refractory period of the AV node, atrium, and AP as well as induce tachycardia. AH intervals gradually prolong with each earlier AES due to the decremental property of AV node. Dual AV node physiology is seen when there is an abrupt increase in AH interval (usually >50 ms) in response to a 10 ms decrement in the coupling interval of AES (Fig. 14.5). AES are used to measure the refractory period of AP. The longest A1A2 interval that fails to conduct is the effective refractory period (ERP). Given the difference in ERP between AV node and AP, A1A2 interval that is longer than AVN ERP but shorter than AP ERP will only conduct via AV node. On the other hand, if AP ERP is greater than AV node ERP, decremental AES approaching AV node ERP will result in full preexcitation.

Atrial Overdrive Pacing (AOP)

Atrial overdrive pacing (AOP) is a drive chain of multiple stimuli at a fixed interval. AOP is used to evaluate how fast the AP can conduct antegrade 1:1 or to induce tachycardia or atrial fibrillation with rapid pacing. The mechanism of sudden death in patients with WPW is thought to be due to rapid conduction of atrial fibrillation through the AP which degenerates into ventricular fibrillation [5, 23]. The refractory period of an AP correlates with the shortest RR interval and mean ventricular rate when conducting antegrade [24]. For this reason, the AP ERP and shortest RR interval in the setting of atrial fibrillation and antegrade ERP of AP have prognostic

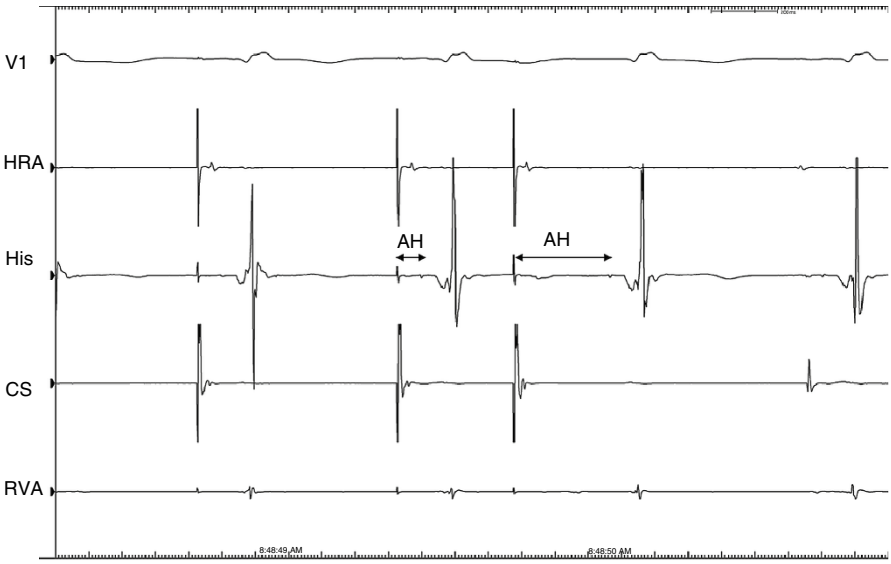

Fig. 14.5 AH jump. Atrial extrastimulus with 10 ms decrement in S2 results in AH increase of more than 50 ms

implication in patients with preexcitation. One notable low-risk feature is the shortest RR during atrial fibrillation >250 ms. These cutoff values may not apply to patients under general anesthesia [25, 26]. Also, sudden AP block with exercise or intermittent preexcitation indicates a poorly conducting AP with a long antegrade refractory period and is associated with low risk for sudden death [27, 28].

Ventricular Extrastimulus (VES)

Ventricular extrastimuli (VES) are ventricular premature depolarizations (VPD) delivered in 10 ms decrements after a drive chain of a fixed cycle length or during a spontaneous rhythm. VES are very useful diagnostic tools for delineating the mechanisms of supraventricular tachycardia. VA intervals and retrograde refractory periods of the AV node or accessory pathways can be measured with decremental VES. An abrupt increase in the VA conduction time via the AV node (>50 ms) in response to a 10 ms decrement of the coupling interval suggests retrograde dual AV node physiology. However, it is important to rule out retrograde RBBB when analyzing this phenomenon as retrograde RBBB will result in VA interval increases of up to 50 ms due to the paced wavefront entering the His-Purkinje system transseptally via the left bundle branch.

Initiation

The reentrant SVT can be induced with a burst pacing or extrastimulation. The prerequisite for reentrant tachycardia induction includes (1) a unidirectional conduction block in one pathway and (2) slow conduction in the second pathway creating a sufficient delay to allow recovery of the first pathway for retrograde conduction. Generally, antegrade AP ERP is longer than AV node ERP, and AVRT is induced with AES that causes AP block and sufficient prolongation of normal AV node conduction. In this case, PR prolongation and sudden loss of preexcitation precede the initiation of tachycardia [29]. In concealed accessory pathways, retrograde AP ERP is usually shorter than that of the AV node. Sequential VES with shorter cycle lengths can increase AV nodal refractoriness and induce tachycardia. The AVRT circuit must be sufficiently slow enough or large enough to prevent the leading wavefront from reaching refractory tissue. Delay in the AV node manifests as AH prolongation, either from physiological AH prolongation or switching from fast pathway to slow pathway conduction, thus allowing perpetuation of tachycardia (Fig. 14.6). Block in the His-Purkinje system, i.e., bundle branch block (BBB) on the ipsilateral side of AP either following AES or VES, may lengthen the circuit enough to maintain SVT. The induction of retrograde RBBB with ventricular stimulation is defined as an increase in VH > 50 ms during pacing [30]. The ipsilateral bundle branch block forces conduction over to the contralateral bundle via transseptal conduction, subsequently expanding the circuit and slower conduction

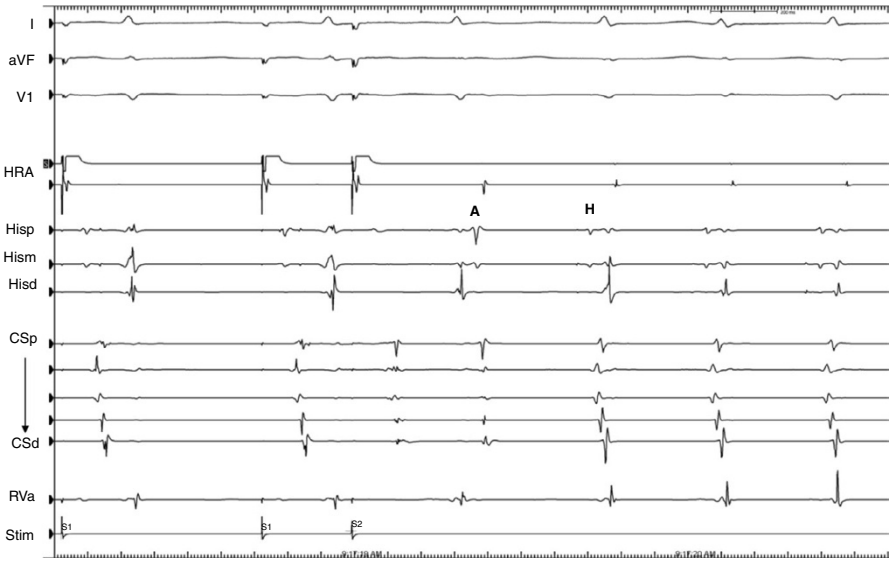

Fig. 14.6 Tachycardia initiation. AH interval prolongation from atrial extrastimulus which fortuitously produces a premature atrial depolarization that prolongs AH sufficiently to initiate tachycardia. Double atrial extrastimuli can be delivered to mimic this phenomenon and aid in SVT initiation

facilitating the induction of tachycardia. With AES, functional RBBB is more frequently seen, while functional LBBB is more common with VES due to differential bundle branch refractory period. Once functional BBB occurs with extrastimuli, the same type of aberrant conduction can persist through the next beat via transseptal linking from the retrograde invasion from the contralateral bundle [31, 32].

VA Relationship

VA relationship can be evaluated during tachycardia or with ventricular pacing. AVRT is the only common SVT that incorporates ventricular myocardium as part of the circuit and obligates a 1:1 AV relationship. AV block is possible in AT, JT, and very uncommonly in AVNRT, usually a transient phenomenon with block occurring in the His-Purkinje system (more As than Vs) or upper common final pathway (more Vs than As) (Fig. 14.7). Typically, AT shows more reliable and sustained AV block. Therefore, the presence of AV block or AV dissociation can rule out AVRT, with rare AP variants such as nodofascicular/nodoventricular pathway being the exceptions.

BBB During Tachycardia

Functional bundle branch block can be observed following ventricular pacing during sinus rhythm or spontaneously during tachycardia. Ipsilateral BBB forces

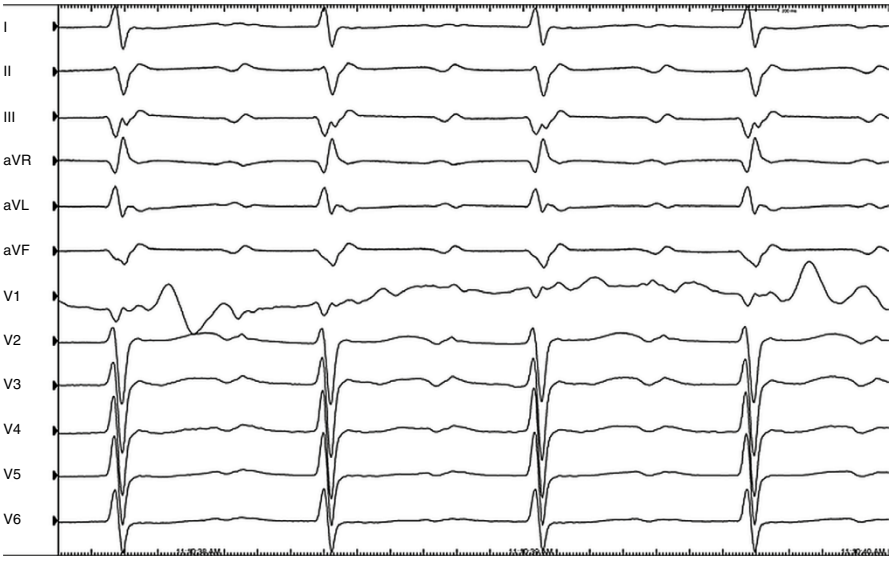

Fig. 14.7 AV block in the setting of AVNRT. AVNRT does not obligate atrium or ventricular as part of the circuit and AV dissociation or AV block can be occasionally seen

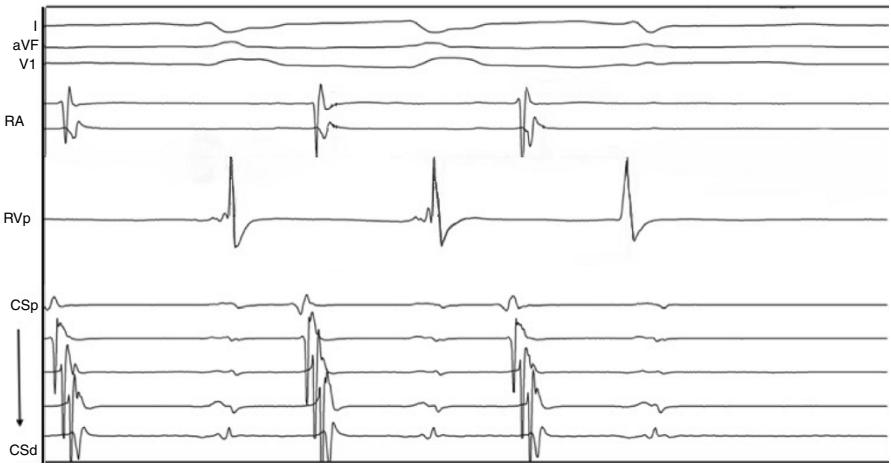

Fig. 14.8 Loss of RBBB and termination of ORT. During tachycardia with RBBB, the antegrade wavefront propagates down the left bundle and through the septum, which allows sufficient time for the pathway to recover and allows retrograde conduction to the atrium. With the loss of RBBB, the earlier wavefront is blocked because the accessory pathway is refractory

conduction over the contralateral bundle through transseptal link and enlarges the tachycardia circuit. Ipsilateral bundle branch block causes an increase in VA time and sometimes TCL due to a larger circuit during ORT (Coumel's sign) [33]. This finding is diagnostic of pathway participation in the tachycardia, whereas failure to observe the interval change does not rule out ORT (Fig. 14.8). Likewise, TCL may not change when an increase in VA is canceled by a shortening of AV interval, particularly with septal pathways. Changes related to BBB in ORT can help localize the pathway as well (Fig. 14.9). The anteroseptal pathways demonstrate an increase in VA interval with RBBB, while posteroseptal pathways are associated with a change in VA interval in the setting of LBBB. Also, VA interval increase ε 35 ms is observed for the free wall and ≤ 25 ms for the septal pathways [34].

Diagnostic Maneuvers

In an electrophysiology study for narrow complex tachycardia, the common differential diagnosis is AT, AVNRT, and AVRT with a concealed pathway (ORT). Junctional tachycardia is possible but rare in the adult population. Unusual AP variants are extremely rare. There are a variety of pacing maneuvers that help make the diagnosis. The key is to understand the difference in the tachycardia mechanism and limitation of each maneuver (Table 14.2).

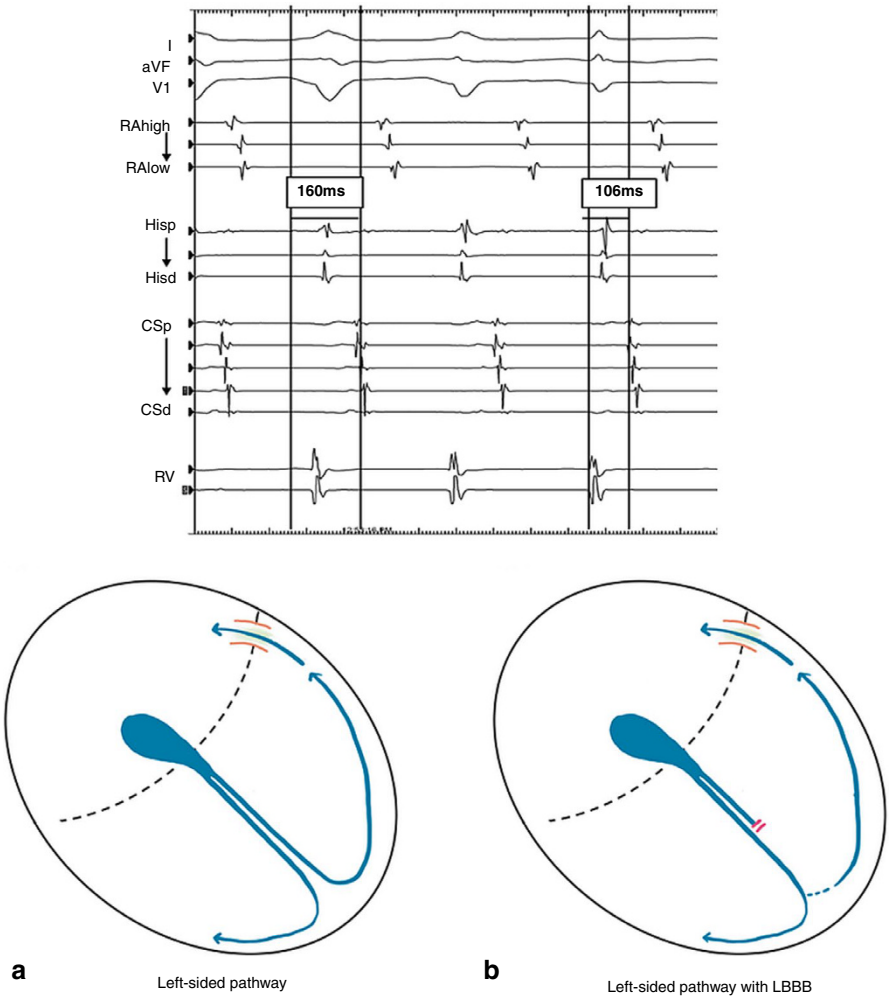

Fig. 14.9 Ipsilateral bundle branch block. A bundle branch block (BBB) ipsilateral to the side of the pathway forces the conduction over to the contralateral bundle via transseptal conduction, subsequently prolonging VA interval and enlarging the tachycardia circuit. (**a**) Left-sided pathway. (**b**) Left-sided pathway with LBBB

VA Interval (V Pacing SR)

In the most common form of AVNRT, atrium and ventricle are activated almost simultaneously resulting in short VA interval (interval from the onset of QRS to the earliest site of atrial activation), although atypical forms of AVNRT can have a longer VA interval. VA interval < 70 ms excludes ORT, as the conduction from the ventricle to the atrium via bypass tract takes longer than 70 ms, although in children and young adults the cutoff value is 50 ms (Fig. 14.10) [35].

Table 14.2 Major observation and diagnostic maneuvers for SVT

Finding	Interpretation
Observation	
Reproducible AH jump to initiate tachycardia	Rules out AT/JT
1:1 VA relationship	ORT
VA interval > 70 ms (for young adults and pediatric 50 ms)	ORT
VA increase ≥30 ms with ipsilateral BBB	ORT
Maneuver	
His-refractory VPD that (1) advances A and resets tachycardia, or (2) delays A, or (3) terminates tachycardia	ORT
VAV response	Rules out AT

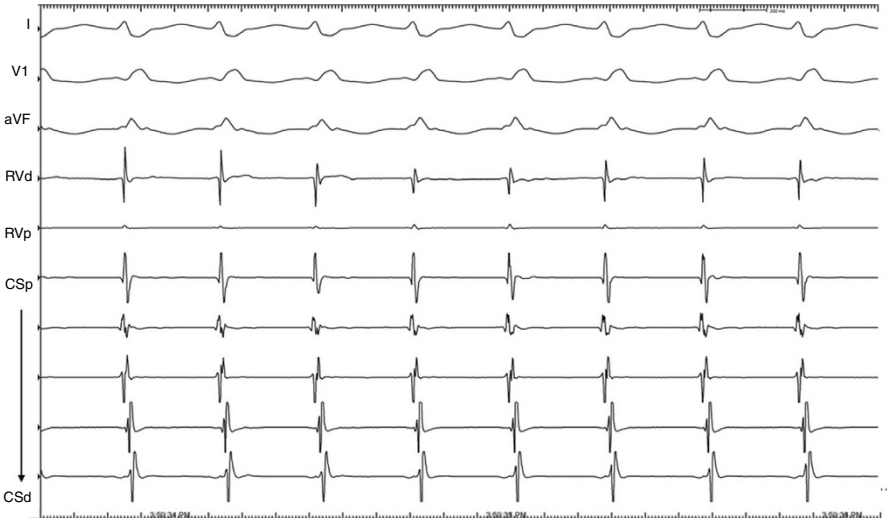

Fig. 14.10 VA interval. AVNRT with VA interval < 70 ms excludes ORT

Differential RV Pacing

RV apical pacing results in a shorter VA interval through the AV node than RV basal pacing due to an earlier invasion of the distal His-Purkinje system and is called a nodal response. In the presence of a septal AP, RV basal pacing results in shorter VA interval compared to apical pacing due to the basal location of most APs (Figs. 14.11 and 14.12). This may not be true with left lateral, slow, or rare APs.

Para-Hisian Pacing

Para-Hisian pacing can be used to distinguish septal APs from retrograde AV nodal conduction [36]. A pacing wavefront from the RV base travels more apically to enter the distal His-Purkinje system where it travels to the AV node and then to the atrium, arriving just posterior to the His bundle recording site. The usual retrograde

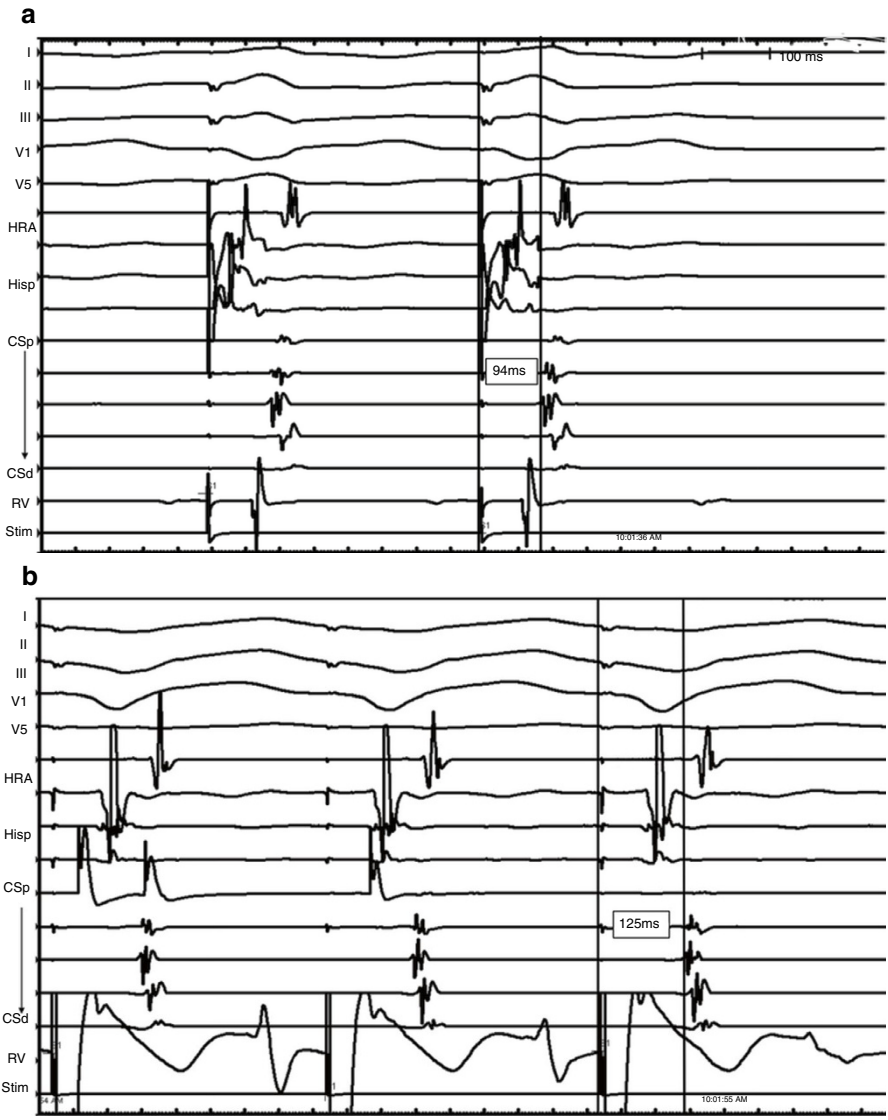

Fig. 14.11 Differential RV pacing in the presence of septal accessory pathway. (**a**) RV basal pacing (**b**) RV apical pacing

atrial activation would start at the His bundle recording catheter followed by CS proximal activation and then the HRA catheter. However, more unusual retrograde atrial activation sequences exist that occur more inferiorly or leftward through the AV node. By increasing the pacing output or with slight respiratory movements, His bundle capture will occur with or without local RV capture. Since this provides a shortcut to the AV node (the usual apical route is bypassed), a significant decrease in Stim-A interval with no change in atrial activation is observed (i.e., nodal

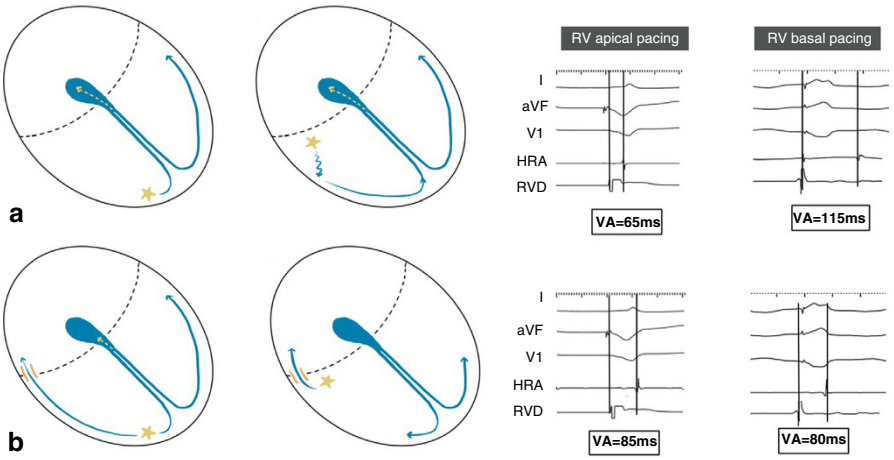

Fig. 14.12 Differential pacing. (**a**) In normal conduction via AV node, RV basal pacing produces a longer VA interval than that of RV apical pacing which engages retrograde conduction system more directly. (**b**) In the presence of a septal AP, RV basal pacing results in a similar or shorter VA interval compared with RV apical pacing, as the impulse conducts retrograde through the pathway to reach the atrium without engaging the normal conduction system

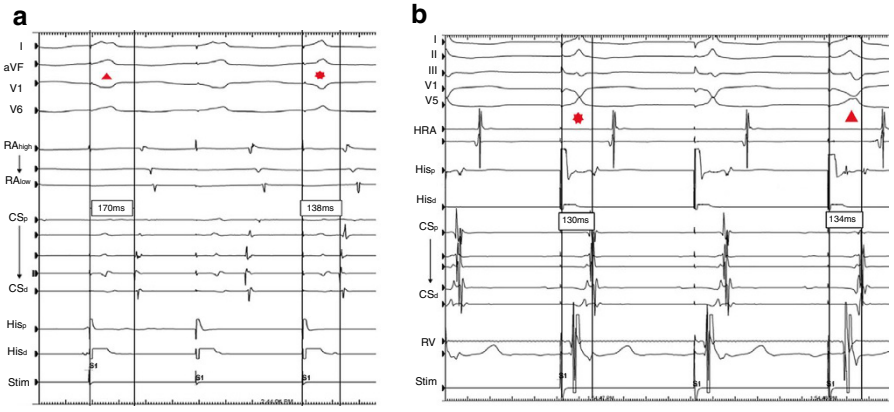

Fig. 14.13 Para-Hisian pacing example. (**a**) Nodal response: VA interval with ventricular capture only (triangle) is 32 ms longer than that with His capture (asterisk). (**b**) Extranodal response (pathway present): VA interval with ventricular capture only (triangle) is similar to that with His capture (asterisk). Note the His deflection in the His catheter with loss of direct His capture

response). In the presence of a retrograde conducting septal AP, the ventricular wavefront propagates over a small area of ventricular tissue and back to the atrium through AP. Capture of the His bundle does not provide a shortcut, so Stim-A remains the same as that without capture of the His bundle and there is no change in the atrial activation pattern. If ΔStim-A < 35 ms, it raises suspicion for the presence

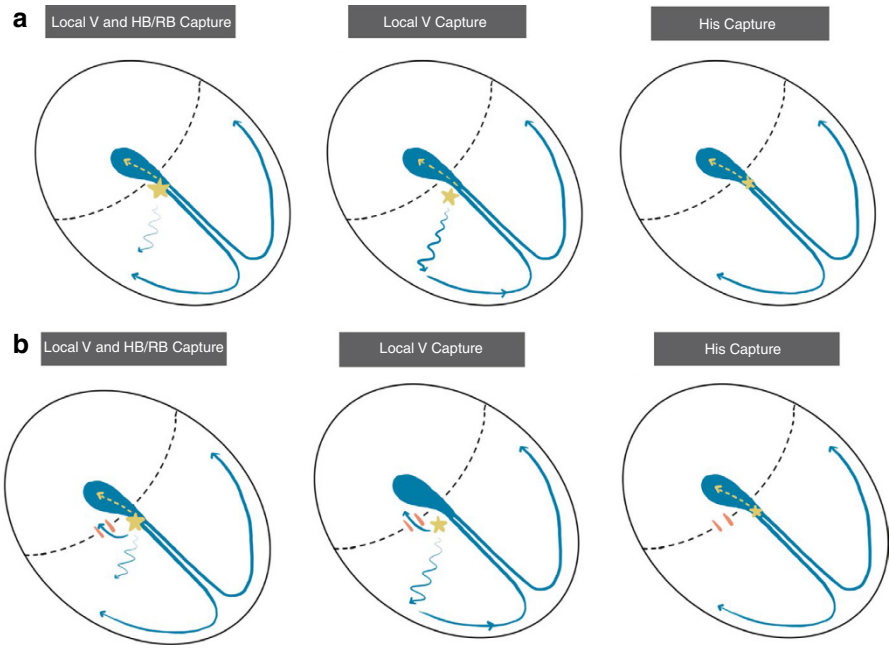

Fig. 14.14 Para-Hisian pacing. (**a**) In a normal AV nodal conduction ("nodal response"), a high output para-Hisian pacing with His bundle (HB) and proximal right bundle (RB) capture in addition to local ventricular capture produces a short Stim to atrium (SA interval). A low output pacing with ventricular capture only results in a longer SA interval, as the impulse propagates through local myocardial tissue to engage the retrograde RB up to the atrium. With HB capture alone, SA interval and atrial activation patterns are similar to HB/RB capture and QRS is normal. (**b**) In the presence of septal pathway ("extranodal response"), a high output pacing with local ventricular and HB/RB capture generates a short SA interval, and this remains similar when there is loss of HB capture, as the impulse bypasses AV nodal conduction via the pathway. With HB capture alone, atrial activation pattern remains the same; but SA interval is longer, as the impulse must now travel fully through the His-Purkinje system and retrograde through the ventricle (equivalent to an echo with atrial extrastimulus)

of AP (Figs. 14.13 and 14.14) [36]. It is important to avoid inadvertent atrial capture to avoid error in interpretation. This maneuver may not be accurate in the setting of a left lateral, or decremental pathway and mixed responses between AP and AV node or multiple APs may be confusing. Para-Hisian pacing may need to be performed at different CLs to allow separate conduction through AV node or AP.

An important caveat in both differential RV and para-Hisian pacing is that these maneuvers demonstrate the structures responsible for the retrograde conduction but do not prove that this circuit is operative during tachycardia. This limitation can be overcome by differential RV and para-Hisian entrainment during tachycardia. Also, failure to identify the pathway with para-Hisian pacing does not rule out the possibility of an AP.

HA Interval (V Pacing During SR or Entrainment)

During AVRT, HA interval measured during tachycardia requires sequential conduction through the His-Purkinje system, ventricular myocardium, and finally AP. In contrast, ventricular pacing during sinus rhythm in the presence of an AP will result in conduction from ventricular tissue to the AP, while simultaneously conducting through the His-Purkinje system in a parallel fashion. Therefore, the HA interval measured during ventricular pacing will be shorter than that during tachycardia. The opposite is seen during AVNRT. During tachycardia, there is a parallel activation of the atrium and the His-Purkinje system, while ventricular pacing results in a sequential activation. Studies demonstrated that the cutoff value of ΔHA (HAEntrainment/Pacing-HASVT) = 0 reliably differentiated AVNRT (positive value) from ORT (negative value), incorporating a septal pathway [37, 38]. Accurate recording of the retrograde His bundle potential is essential to utilizing this criterion but is not always easy to obtain.

His-Refractory Extrastimulation (V Pacing During Tachycardia)

His-refractory ventricular extrastimulation refers to a VPD delivered either simultaneously or within 55 ms of His bundle activation, such that retrograde conduction through the AV node is not possible. Therefore, any effect on the subsequent atrial activation or tachycardia cycle length would require a separate retrograde pathway (Fig. 14.15) [39]. If atrial activity is not affected by His-refractory VPDs, it favors AVNRT or, at least, suggests that the pacing site is far from the pathway. If a His-refractory VPD advances the atrium during tachycardia, it indicates the presence of pathway but does not confirm pathway participation in the tachycardia. If His-refractory VPD not only advances atrium but resets the tachycardia (e.g., all subsequent beats occur ahead of where they would be predicted during stable tachycardia before the VPD), it confirms accessory pathway-mediated tachycardia [40, 41]. Similarly, tachycardia terminated by His-refractory VPD without reaching the atrium excludes AT and AVNRT. If His-refractory VPD during tachycardia delays atrial activation, it proves ORT utilizing a decremental pathway (Fig. 14.16) [42]. Exceptions to these rules would be the presence of bystander AP or unusual AP variants that could affect AVNRT or AT with a His-refractory VPD.

Transition Zone (VOP During Tachycardia Regardless of the Success of the Entrainment)

RV pacing during narrow complex tachycardia results in a progressive fusion of QRS complexes before there is a constant fully paced or fused beat. A transition zone lies between the first pacing stimulus with QRS fusion and the first paced beat with a stable QRS morphology. Evaluating the atrial timing and the Stim-A interval during and after a transition zone can be helpful to distinguish AVRT from other

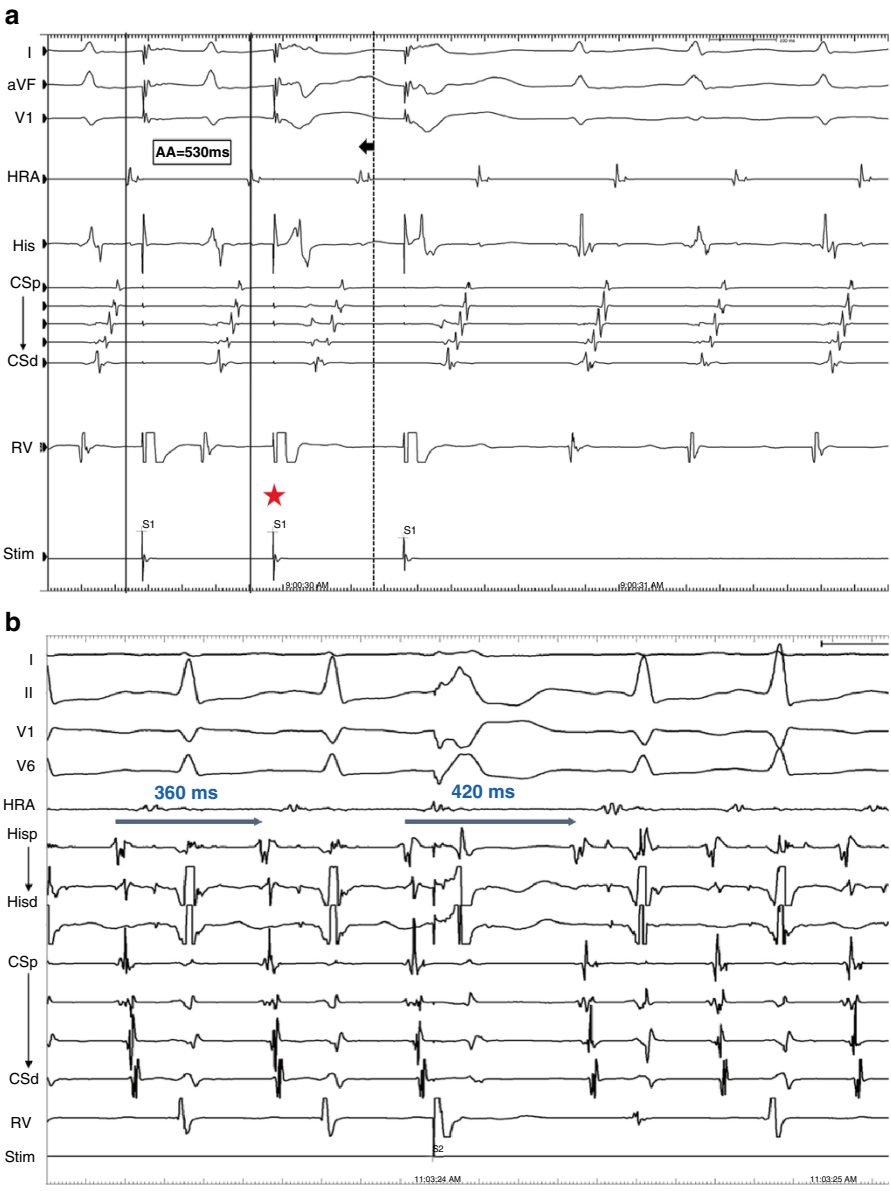

Fig. 14.15 His-refractory VPD examples. (**a**) Advance A: When VPD was delivered after His is committed, atrium may be advanced in the presence of pathway. (**b**) Delay A: His-refractory VPD delays atrial activation and this proves the pathway-mediated tachycardia. (**c**) Terminate tachycardia: If His-synchronous VPD terminates the tachycardia, it proves pathway-mediated tachycardia

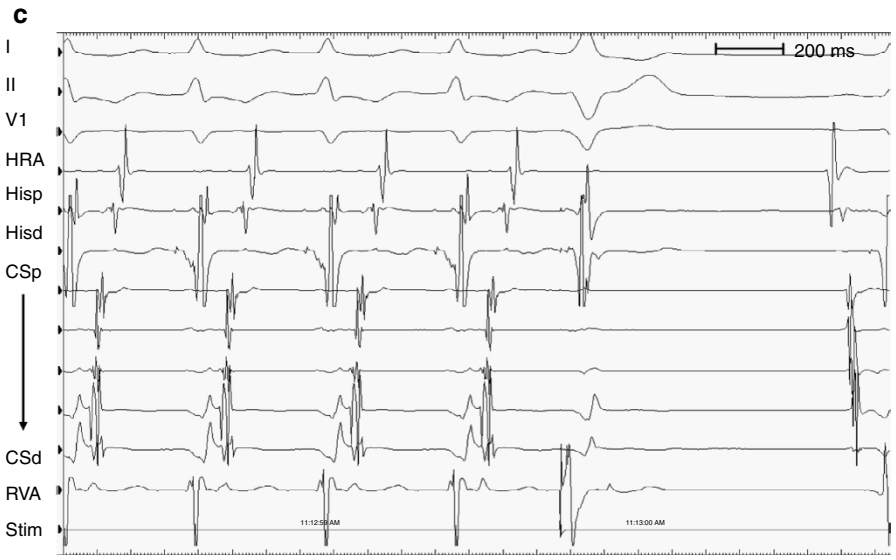

Fig. 14.15 (continued)

SVTs. The Stim-A interval becomes fixed before or at the time of the first fully paced QRS in AVRT. However, in AVNRT and AT, the perturbation of atrial timing is not seen until a few beats after the transition zone and Stim-A interval becomes fixed after this zone. This finding is based on the fact that the ventricle is the part of the reentrant circuit in ORT unlike other SVT mechanisms (Fig. 14.17) [43–45]. Identifying the first fully paced stable beat is the key for accurate interpretation, although studies showed that the slight interobserver variation in judging the timing of the first fully paced beat did not alter the diagnosis [45]. The advantage of this maneuver is that it does not require successful entrainment and it can be used when VOP train terminates tachycardia. It also seems less dependent on the pathway location relative to the RV pacing site, but this is still a problem. This maneuver would not work for tachycardia with significant oscillation of the cycle lengths or with a bystander accessory. It will work better if you move the V pacing site to the earliest atrial activation site, which allows early penetration of an AP when present.

Entrainment

Entrainment is an essential technique that identifies whether or not a specific pacing site is an integral part of the reentrant circuit. The requirement of reentry includes (1) functional or anatomic circuit, (2) unidirectional block, and (3) zone of slow conduction.

During pacing at a constant rate slightly faster than the tachycardia, any of the following could indicate entrainment.

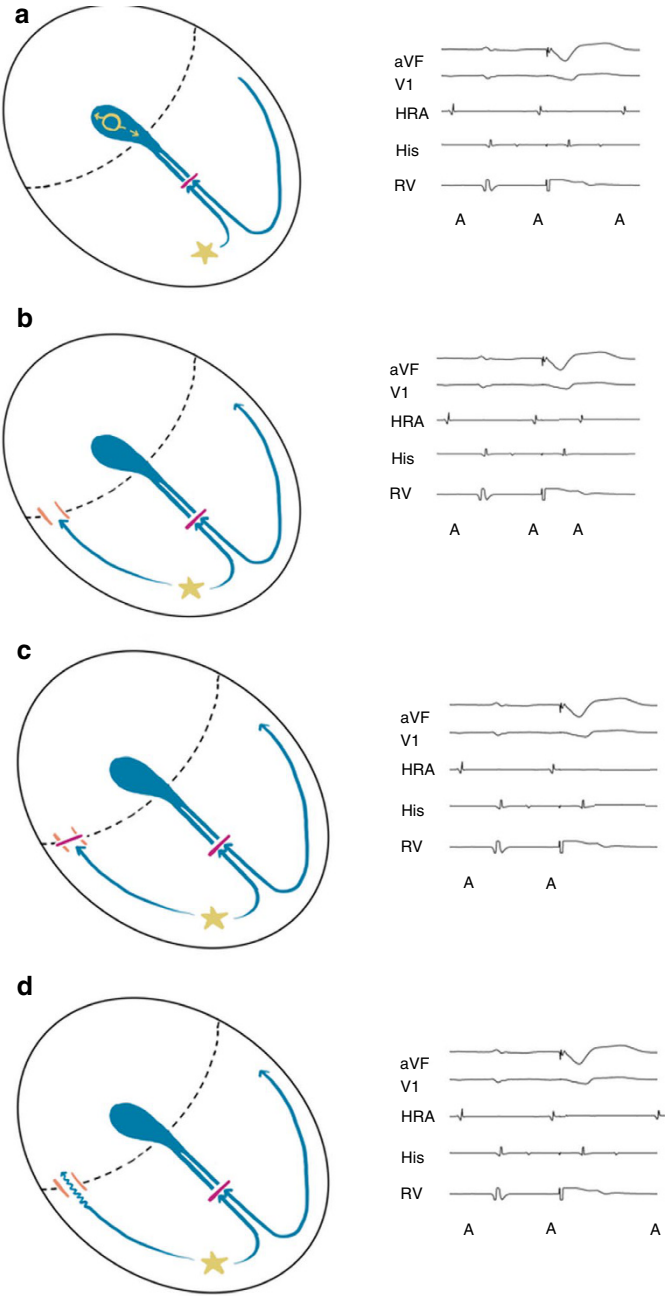

Fig. 14.16 His-refractory VPD responses. (**a**) In AVNRT, or if pacing far from the pathway, His-refractory VPD does not perturb tachycardia. (**b**) During ORT, His-refractory VPD may advance atrial electrogram. If tachycardia is resent, it proves pathway participation in the tachycardia. (**c**) His-refractory VPD may terminate the pathway-mediated tachycardia if pathway is refractory. (**d**) His-refractory may delay the atrial electrogram if pathway is slowly conducted or decremental

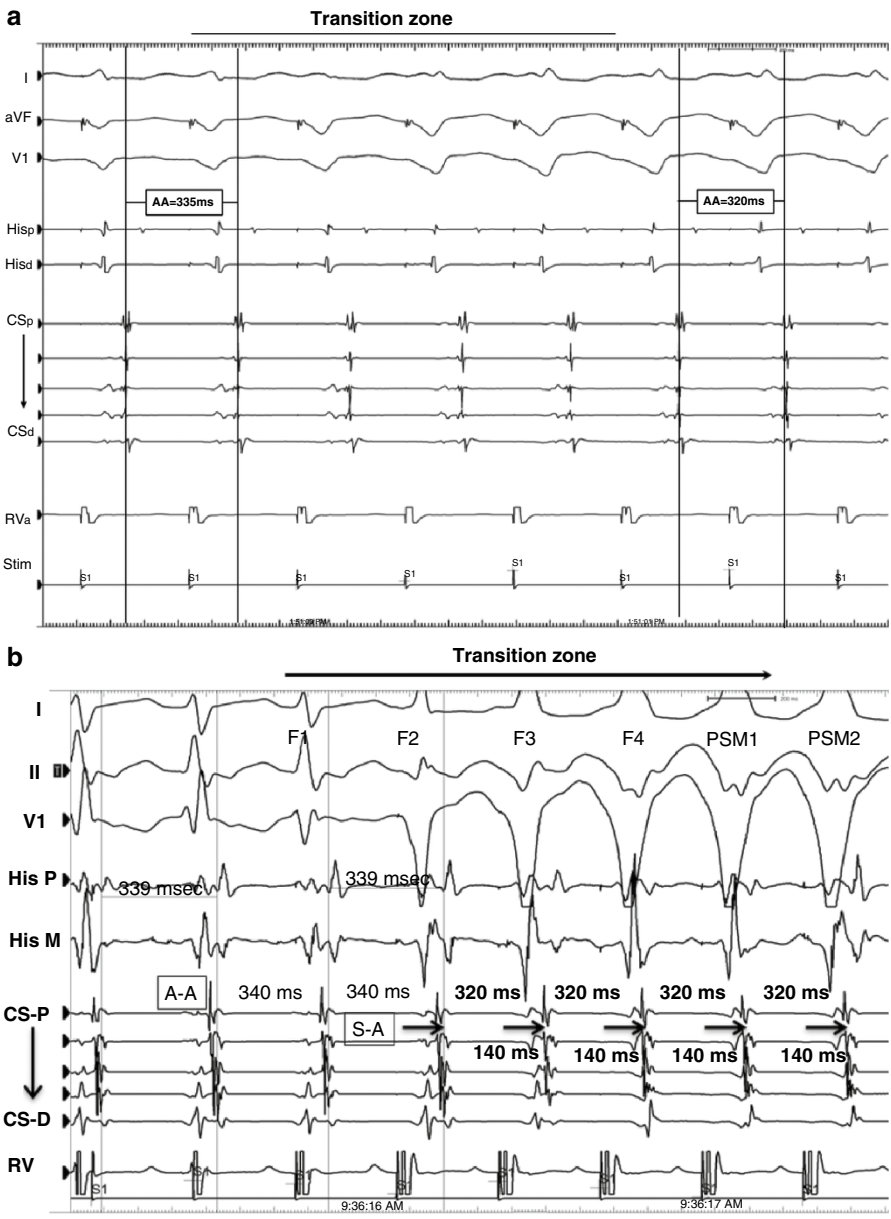

Fig. 14.17 Transition zone. (**a**) Tachycardia is advanced following the transition zone consistent with AVNRT. (**b**) Tachycardia is advanced during the transition zone consistent with ORT

- Constant ECG fusion except for the last entrained beat which is fully paced and not fused
  - Fused QRS complexes during SVT entrainment indicates ORT.
- Progressive ECG fusion
  - The degree of QRS fusion in the ECG is different for different pacing rates while pacing during SVT.

Observation and maneuvers during and post-entrainment further assist in making a diagnosis [46–48].

A(H)V Versus AA(H)V Response (VOP or Entrainment)

The post-ventricular overdrive pacing (VOP) or entrainment response can be useful in differentiating AT from other AV node-dependent tachycardia such as AVRT or AVNRT (Figs. 14.18, 14.19, and 14.20). In patients with AV node-dependent tachycardia, the last ventricular paced beat is followed by a retrograde atrial electrogram and then ventricular activation. This finding is called an AV response where the last atrial electrogram accelerated to the pacing CL (i.e., the first response after pacing stopped) is the first electrogram counted in the interpretation. In AT, however, the

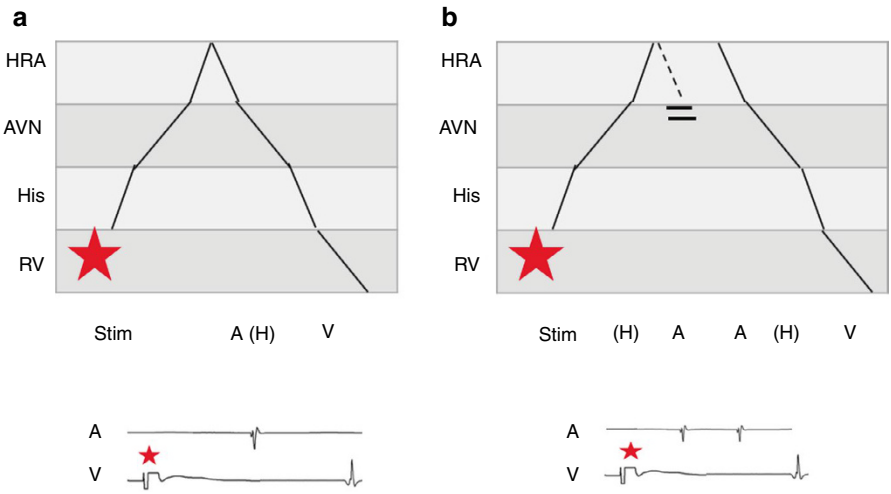

Fig. 14.18 Post-VOP response. (**a**) In ORT, the previous wavefront collides with the antidromic wavefront from the paced beat, and the last paced beat conducts retrograde up the pathway and antegrade down the AV node, thus generating VA(H)V response. (**b**) During atrial tachycardia, the last paced beat conducts retrograde to the atrium, but the tachycardia resumes and results in a "VAA(H)V" response

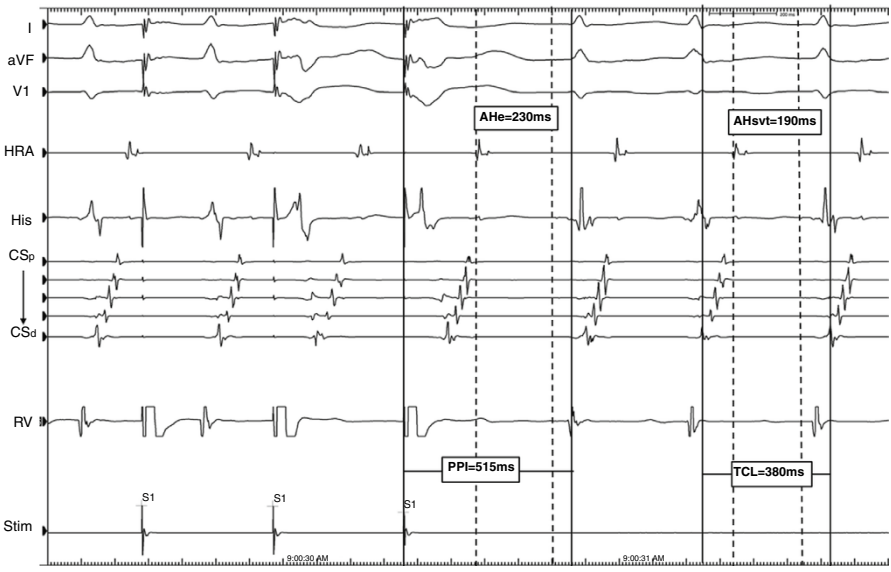

Fig. 14.19 Post-ventricular overdrive pacing response. Following two extrastimuli, there is VAV response. Post-pacing interval (PPI) is 551 ms; tachycardia cycle length (TCL) 380 ms is 135 ms. This value needs to be adjusted with the delta AH interval (AHEntrained 230 ms – AHSVT 190 ms = 40 ms) and corrected PPI-TCL (135–40 ms) is 95 ms, consistent with ORT

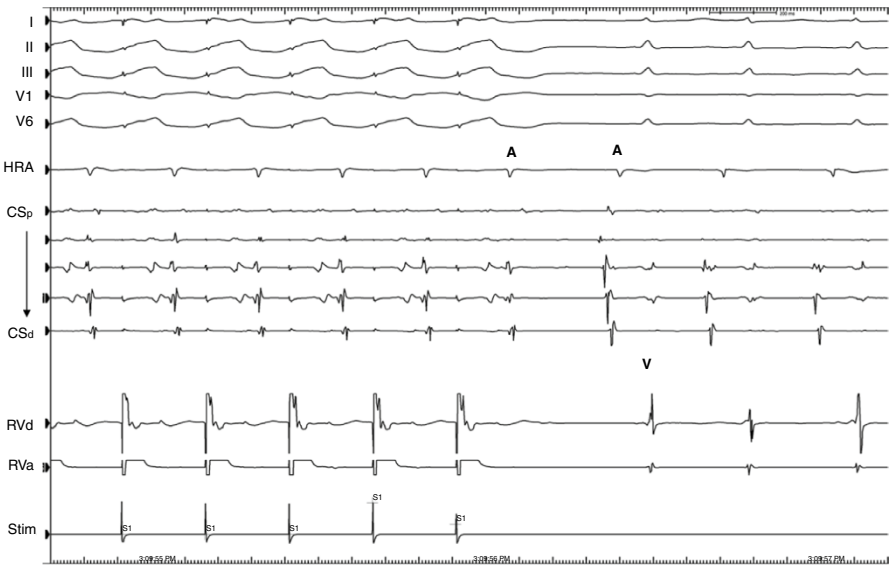

Fig. 14.20 AAV response during septal atrial tachycardia

discontinuation of pacing results in conduction through the AV node producing a concentric atrial activation sequence followed by resumption of AT (usually with a different activation sequence) producing an AAV response [49]. In practice, VA dissociation is more commonly present than the AAV response during AT. It is important to understand the pitfalls of post-VOP interpretation such as "pseudo-AAV response." Sometimes if the HV interval exceeds the HA interval or the HA interval is short during AVNRT, an atrial electrogram will precede the ventricular electrogram during ongoing AVNRT. In this case, using the His bundle potential instead of ventricular potential in the analysis (e.g., AH vs. AAH instead of AV vs. AAV response) can help avoid the misinterpretation [50]. When the retrograde limb is slow in the setting of "slow-slow" or "fast-slow" atypical AVNRT or AVRT with a slowly conducting AP, the VA interval is longer than the VV interval, and post-pacing response can be challenging to interpret but can be sorted out by measuring the last A accelerated to the pacing cycle length. It is important to ensure that AAV response is reproducible at different pacing rates [51].

PPI-TCL

Following termination of V pacing during tachycardia, the return cycle length to the pacing catheter is the sum of tachycardia cycle length (TCL) and conduction time to and from the tachycardia circuit. Post-pacing interval (PPI) is measured from the last pacing impulse to the first recorded local impulse on the pacing channel. When the tachycardia is stable and entrained, the post-pacing interval (PPI) can be useful in differentiating AVNRT from AVRT using a septal AP. If PPI-TCL is less than 115 ms, it suggests that the ablation catheter is relatively near the tachycardia circuit and the ventricle may be part of the circuit. If PPI-TCL > 115 ms, it supports the diagnosis of AVNRT [52]. This equation was further modified to incorporate a pacing-induced AV nodal conduction delay. AH interval following the last entrained atrial electrogram is longer than AH during tachycardia due to decremental conduction over AV node and/or AP. You can correct the delay by calculating the difference between AHSVT and AHEntrainment and subtract the differential from PPI-TCL. When cPPI-TCL (i.e., PPI-TCL – {AHEntrainemnt- AHSVT}) < 110 ms, it favors ORT (Fig. 14.19) [53]. Alternatively, entrainment via basal RV pacing generates an even longer PPI-TCL in AVNRT, and this maneuver is found to be superior to RV apical pacing [54]. PPI-TCL maneuver may not be valid in the setting of ORT with decremental AP or left lateral pathway [55]. The discriminatory ability of this maneuver can be enhanced by pacing near the earliest atrial activation point.

SA-VA Difference (VOP or Entrainment)

As we discussed earlier, atrium and ventricle have a simultaneous parallel activation in AVNRT. During entrainment with ventricular pacing, their activation is in series. In ORT, atrium and ventricle have a serial activation during both tachycardia and

ventricular pacing. The difference between SA (ventricular stimulation to the earliest site of atrial activation) and VA is greater in AVNRT than ORT. ΔSA–VA < 85 ms is consistent with ORT, whereas ΔSA–VA > 85 mg suggests AVNRT. [52]. Again, this maneuver may not be accurate when AP has decremental property or if pacing is too far from AP such as RV apex pacing for the left-sided AP.

Ventricular Fusion

If entrainment with ventricular pacing results in constant or progressive QRS fusion, it rules out AVNRT [56]. However, the absence of QRS fusion does not rule out ORT with a distant AP insertion. For instance, QRS fusion with RV pacing is not evident in the left-sided Aps, whereas RV pacing and entrainment cause a clear QRS fusion on AVRTs with the septal or right-sided APs [54].

Differential Entrainment

Differential entrainment from the basal and apical RV pacing can be useful. In AVNRT, cPPI-TCL and VA intervals are significantly longer from base than apex, while there is no significant difference in entrainment intervals among patients with ORT. Differential cPPI-TCL > 30 ms or VA interval > 20 ms reliably identifies the patient with AVNRT [57, 58]. This maneuver is not validated in patients with atypical AVNRT or ORT with decremental pathways.

Antegrade His Capture

During ORT entrainment, His bundle can be captured either orthodromically or antidromically. If an orthodromic capture of the His bundle is seen, this is intracardiac evidence of fusion occurring in the circuit distal to the His bundle (Fig. 14.21). This finding is diagnostic for AVRT [59, 60]. There are several ways to confirm orthodromic His capture: (1) the His bundle morphology is identical during tachycardia and entrainment; (2) AH interval during entrainment is the same as the first AH interval following pacing; and (3) the last entrained His follows the atrial electrogram after pacing [37]. In contrast, the antidromically captured His bundle will have a different morphology from His electrogram during tachycardia, and AHEntrainment and Stim-HEntraiment are shorter than first AHSVT and Stim-HVPacing, respectively.

Para-Hisian Entrainment

As discussed earlier, para-Hisian pacing is a useful diagnostic maneuver if AV node or AP is the retrograde pathway, although it does not prove the participation of the

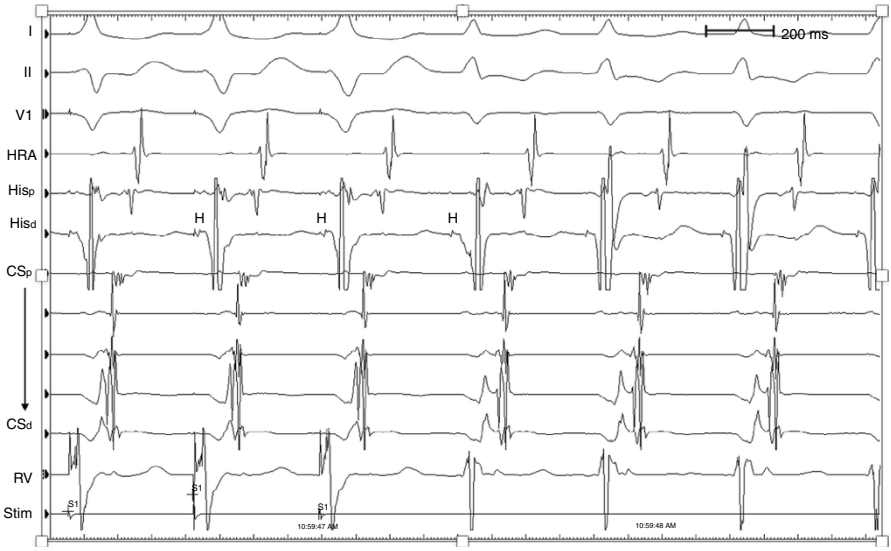

Fig. 14.21 Antegrade His capture during manifest QRS fusion. Ventricular pacing demonstrates antegrade His activation during manifest QRS fusion. These findings are consistent with ORT as a tachycardia mechanism

pathway in the tachycardia. Para-Hisian entrainment overcomes this limitation and proves the pathway-mediated tachycardia by measuring differences in SA-VA intervals and PPI-TCL with and without a His bundle capture [61, 62]. In patients with AVNRT, entrainment without a His-bundle capture as opposed to the one with a His bundle capture results in significant change in both SA (> 40 ms) and VA intervals, whereas these values are not significantly different in AVRT. Although useful, this technique is rather cumbersome to practice. It is at times difficult to ensure the His bundle capture during entrainment, and a high-output pacing may inadvertently capture the nearby AV node or atrium. Also, the local ventricular signal can be difficult to ascertain due to a pacing spike. One study also demonstrated that the change in QRS width increase ≥40 ms during entrainment compared with that during tachycardia is a good indicator for an absent His-bundle capture during entrainment [62].

AH interval (Atrial Pacing During SR)

Although not as useful as ventricular pacing overall, atrial pacing may also be used for diagnostic maneuvers in long RP tachycardias. During AVNRT, AH interval represents a parallel activation of the atrium and the His bundle, while an atrial pacing during AVNRT results in a sequential activation. In patients with AVRT, AH intervals in both pacing and tachycardia represent a sequential activation of the atrium and the His bundle, as both structures are part of the circuit. The change in AH interval during atrial pacing and tachycardia is >40 ms in atypical AVNRT, < 20 ms in AVRT and < 10 ms for AT [63].

ΔVA Interval (VA Linking)

Relatively constant VA intervals while pacing from different sites in the atrium (e.g., high right atrium or coronary sinus) make AT less likely. Post-pacing VA intervals are measured from the last captured ventricular signal to the earliest return atrial electrogram. When calculating the maximal difference in post-pacing VA intervals from different pacing sites, ΔVAmax was >14 ms in AT and < 14 ms in AVRT/AVNRT with 100% sensitivity, specificity, PPV, and NPV. This phenomenon speaks to the "VA linking" in AVRT and AVNRT [64].

Ablation of Accessory Pathway

Catheter ablation is an effective treatment for patients with symptomatic atrioventricular reciprocating tachycardia with a high success rate. For a successful ablation, an accurate localization of the AP is the key to guide the therapy.

Access

Having a general idea of pathway location before the EP study can be helpful in procedure planning. For the left-sided pathway, either transseptal or retrograde transaortic approach can be used. Either IVC or SVC is utilized for mapping and ablation of the right-sided pathway.

Mapping and Ablation

For a left-sided AP, a multipolar CS catheter is useful to bracket the pathway to guide precise localization, while a multipolar circular Halo catheter can be placed along the tricuspid annulus for right-sided APs. The mapping approach is determined by the direction of the pathway conduction. For an AP with antegrade conduction, the pathway is mapped based on (1) the earliest ventricular electrogram (pre-delta), (2) AV fusion location (general idea), and most importantly (3) AP potential. The earliest antegrade ventricular activation during sinus rhythm indicates the ventricular insertion site of the pathway and should precede the onset of QRS (delta) by up to 20 ms [65]. The unipolar recording at the tip of the ablation catheter can be particularly helpful as it confirms all ventricular activation propagating from that point [66]. Also, AV fusion (shortest local AV < 40 ms) is predictive of successful ablation but is not reliable since most APs have an oblique course between atrium and ventricle [67]. Thus, depending on wavefront direction, fusion of VA signals may not represent an ideal ablation location. Reversing the direction of wavefront in both atrium and ventricle and looking for variations of local VA (earliest atrial activation) and AV (earliest ventricular activation) can facilitate the localization and ablation of an oblique pathway [68]. In some very oblique

pathways, atrial or ventricular activation can occur late despite being in an excellent ablation location. For this reason, identification of an AP potential is more reliable as an ablation target.

During either ORT or ventricular pacing with retrograde conduction over AP, the earliest atrial activation can be investigated. If mapping AP retrograde with ventricular pacing is challenging due to retrograde conduction via AVN, slowing down AV nodal conduction pharmacologically or changing the pacing rate can be helpful (Fig. 14.22). Typically, pathways sit along the AV groove with some rare exceptions (e.g., atriofascicular pathways), and other diagnosis can be entertained if the earliest signal is found elsewhere. For retrograde AP ablation, the shortest VA time during ORT or RV pacing usually results in successful ablation; however, locating the local AP potential is associated with a high success rate if ablated [69].

Once mapping is completed, ablation may be pursued. Most AP ablation is performed with RF energy, but focal cryoablation is used for high-risk ablation such as a mid-septal AP with the highest risk of AV nodal injury. The predictors of successful ablation include ablation of the location with (1) shortest VA interval, (2) pathway potential [70], and (3) QS pattern in unipolar signal [12]. The goal of ablation is the loss of AP conduction. However, caution needs to be taken during anteroseptal pathway ablation. Junctional complexes can be mistaken as the loss of preexcitation; but rather it indicates possible injury to the AV node, and one must come off RF energy when junctional rhythms are encountered. It is important to differentiate the sinus beat with loss of preexcitation from the junctional beats (Fig. 14.23).

Adenosine inhibits both antegrade and retrograde conduction over the AV node, while it has no or little effect on non-decremental APs. It also facilitates conduction over "stunned" APs. Given its short half-life, it can be easily used in the lab following ablation to test the completeness of the pathway ablation. Presence of AV and

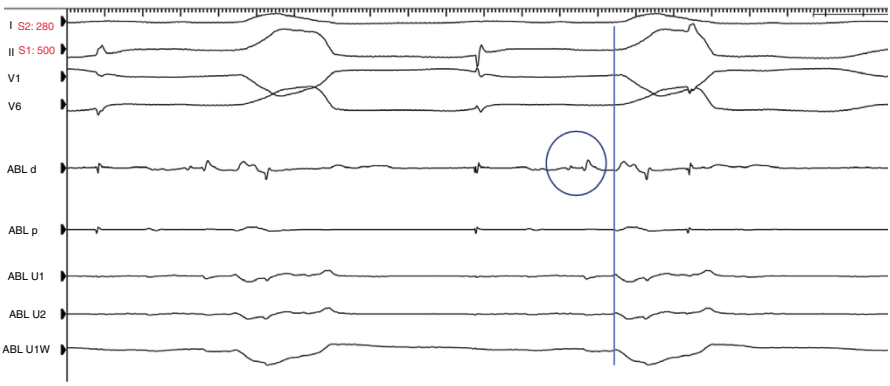

Fig. 14.22 Signal at the successful ablation site. Atrial extrastimulus shows only far-field atrial electrogram and reveals multiple sharp potentials preceding the delta wave which represents anteroseptal AP connection. The distance of AP potential from the ventricular electrogram implies a very slanted pathway. There is a QS configuration at the unfiltered unipolar electrode (U1W), and the AP potential is best seen on the filtered unipolar signal representing the ablation tip (U1). Successful ablation was performed with resolution of pathway potential in 2 s resulting in RBBB

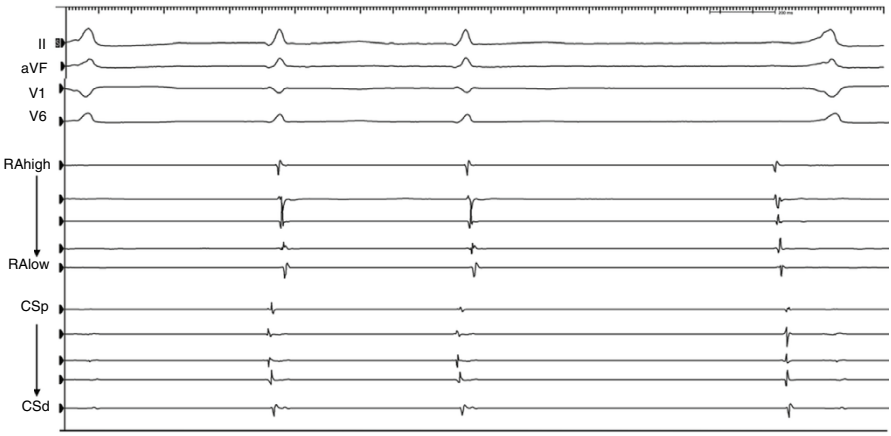

Fig. 14.23 Junctional beat during anteroseptal pathway ablation

VA block with adenosine infusion with atrial and ventricular pacing provides a reliable and immediate assessment of successful pathway ablation.

It is reported that the main factors for ablation failure include a difficulty with catheter manipulation, inadequate mapping and localization, multiple pathways or insertions, or a congenital heart disease [71]. Approximately 10% of patients with AVRT have more than one pathway [72, 73]. When there is more than one preexcited QRS, or P wave morphologies during tachycardia, or spontaneous transition from ORT to ART, it raises suspicion for multiple pathways [74–76]. AVRT may coexist with other SVT mechanisms, particularly AVNRT, which should be sought after successful AP ablation [77].

Rare Types of SVT

Permanent Junctional Reciprocating Tachycardia (PJRT)

PJRT is often described as an incessant type of ORT with a concealed, slowly conducting, decremental pathway [78–80] but can also be due to atypical forms of AVNRT. It manifests as a narrow complex tachycardia that is midline with long RP and superior P wave axis. AP is located near the CS os in the posteroseptal region, so the earliest atrial activation is seen in the proximal CS catheter. Typically, it initiates spontaneously, and the slow and decremental retrograde conduction results in long RP (long VA) and ultimately self-termination. His-refractory VPD may advance, terminate the tachycardia, or delay the atrial electrogram due to its decremental property [42]. Usually, AV fusion is not seen in electrogram due to its decremental properties. Medications are not successful for suppression in general and ablation may be necessary for a cure. Locating the pathway potential or earliest atrial electrogram is helpful to guide ablation [79, 81].

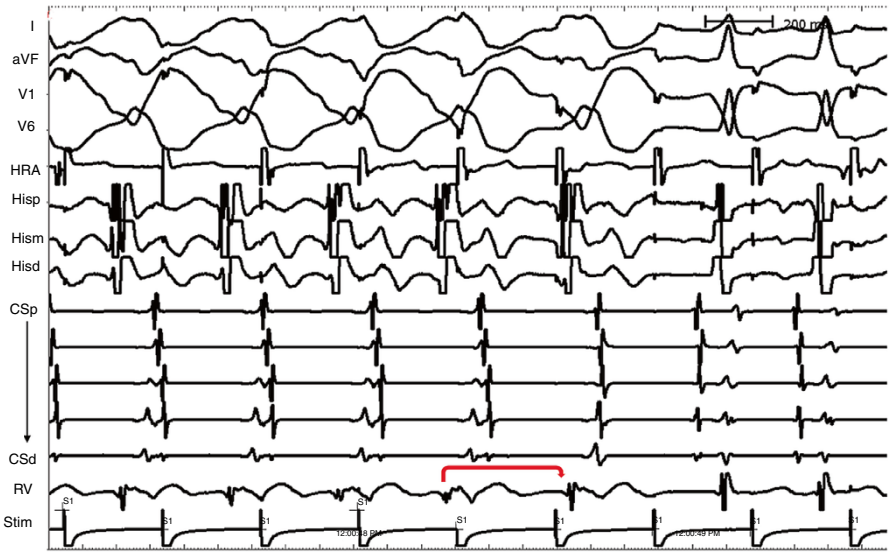

Fig. 14.24 Unusual variant. Antidromic tachycardia with decremental conduction prior to termination. This unusual AP variant inserted at the tricuspid valve annulus

Atriofascicular (AF) Pathway

Atriofascicular pathway inserts proximally in the lateral, anterolateral, and posterolateral side of the tricuspid annulus and extends to the distal right bundle branch near the RV apex. Clinically, it causes antidromic LBBB tachycardia (Fig. 14.24). Since the atrium is part of the circuit, it has an obligatory 1:1 AV relationship. Its classic characteristics are best seen with a selective His bundle and a right bundle branch recording. The reversal of His and RBB activation sequence with progressive preexcitation occurs in the setting of decremental atrial pacing. Just prior to the initiation of tachycardia, RBBB electrogram occurs before His bundle potential, demonstrating antegrade conduction down the AP and retrograde conduction up through the His bundle [14, 15]. Since the proximal insertion is in the atrium, His bundle pacing or His extrasystole can normalize preexcitation. This pathway generally only conducts antegrade. However, its long course and distal arborization with a broad insertion make mapping of the ventricular insertion site challenging [17]. For this reason, ablation is usually targeted at the proximal atrial insertion of the pathway by recording the AP potential. It generates a large, high frequency AP potential recorded at the tricuspid annulus during tachycardia or preexcited atrial pacing [82, 83].

Nodofascicular/Nodoventricular (NF/NV) Pathway

Nodofascicular or nodoventricular pathways originate in the AV node and insert into the right bundle in the anteroapical region of RV [84, 85]. NF/NV pathways

have decremental properties, commonly with concomitant dual AV node physiology or multiple pathways in the setting of congenital heart disease. Generally, tachycardia manifests with LBBB and long AV with either short VH or long VH. Short VH is seen in the setting of retrograde conduction via RBB, while long VH occurs with retrograde RBBB which forces conduction across the septum to LBB. The variation of TCL with LBBB and RBBB confirms His-Purkinje fiber is an integral part of the tachycardia circuit. Decremental atrial pacing generally causes both AV and AH prolongation but fixed VH in the setting of greater preexcitation with LBBB morphology. Once AH prolongation exceeds AV prolongation, HV shortens with increasing preexcitation. Maximum preexcitation is seen with block in the AV node distal to the AP origin, resulting in exclusive conduction over AP and retrograde conduction of His bundle [84]. NF/NV pathways share similar characteristics with other pathways. His-refractory PVC can reset or terminate tachycardia [15, 86]. Ipsilateral bundle branch block causes prolongation of TCL. Differentiating NV/NF from anteroseptal or mid-septal AV AP can be challenging. Since NF/NV pathways do not insert into atrial myocardium, AV dissociation seen spontaneously or with atrial pacing is a distinguishing feature for NF/NV pathways [87]. They can be innocent bystanders in the setting of AVNRT as well. If losing preexcitation does not affect tachycardia or presence of variable preexcitation in a stable tachycardia, AVNRT with bystander NF/NV pathway is more likely. Ablation is performed typically on the ventricular insertion site near the tricuspid valve, given the proximity to the AV node. However, this is not possible for even more rare cases where AP only conducts retrograde. Functionally, it is linked to a slow pathway and slow pathway ablation in the posterior septum or Koch triangle region is performed with variable success [85, 88]. Successful ablation of NF/NV pathways can result in a complete RBBB.

Fasciculoventricular AP (FV) [89, 90]

Fasciculoventricular pathways originate in the right bundle and insert into the right ventricle. Unlike other pathways, FV pathway manifests with fixed preexcitation and HV or H-delta with variable AH and AV from atrial pacing. Therefore, FV pathway should be suspected when there is fixed preexcitation with decrementing AV node and persistent preexcitation during His extrasystole, as the pathway inserts distal to His bundle.

In contrast to other slow, decrementing pathways such as AF, NF/NV pathways, using adenosine results in fixed preexcitation despite PR prolongation or junctional escape or loss of preexcitation with AV block [89]. Fasciculoventricular pathways are extremely rare, although the actual prevalence is likely higher due to underrecognition. They have been reported as bystanders only and do not participate in clinical tachycardia [84].

Subepicardial AP

Posteroseptal or posterior APs can involve myocardial fibers surrounding CS and create a subepicardial communication between the left atrium and the ventricle [91, 92]. Some studies demonstrated that the anatomic CS anomalies such as aneurysms or diverticula are associated with these pathways, thus emphasizing the importance of CS venogram during ablation. However, other studies showed that subepicardial pathways can occur even in the absence of CS anomalies and are closely associated with the middle cardiac vein [93]. In the surface ECG, negative delta wave in lead II suggests epicardial localization of the pathway. However, ablation of these pathways may not be necessarily effective with epicardial ablation and may rather require CS mapping and ablation at the atrial insertion site [94, 95]. Coronary angiography should be considered when ablating in the MCV, since a branch of the right coronary artery or left circumflex artery is often nearby.

Summary

Radiofrequency ablation has become the cornerstone of therapeutics for symptomatic patients with pathway-mediated tachycardia with a high success rate and rare complications. In order to achieve a cure with catheter ablation, understanding different behaviors and the mechanisms of arrhythmia during electrophysiology study is essential.

References

1. Wolff L, Parkinson JP, White PD. Bundle-branch block with short P-R Interval in healthy young people prone to paroxysmal tachycardia. Am Heart J. 1930;5(6):685–704.
2. Wood FC, Wolferth CC, Geckeler G. Histologic demonstration of accessory muscular connections between auricle and ventricle in a case of short P-R interval and prolonged QRS complex. Am Heart J. 1943;25(4):454–62.
3. Cain ME, Luke RA, Lindsay BD. Diagnosis and localization of accessory pathways. PACE. 1992;15(5):801–24.
4. Kuck KH, Friday KJ, Kunze KP, Schluter M, Lazzara R, Jackman WM. Sites of conduction block in accessory atrioventricular pathways. Basis for concealed accessory pathways. Circulation. 1990;82(2):407–17.
5. Klein GJ, Bashore TM, Sellers TD, Pritchett EL, Smith WM, Gallagher JJ. Ventricular fibrillation in the Wolff-Parkinson-White syndrome. N Engl J Med. 1979;301(20):1080–5.
6. Bogun F, Kalusche D, Li YG, Auth-Eisernitz S, Gronefeld G, Hohnloser SH. Septal Q waves in surface electrocardiographic lead V6 exclude minimal ventricular preexcitation. Am J Cardiol. 1999;84(1):101–4, A9.
7. Arruda MS, McClelland JH, Wang X, Beckman KJ, Widman LE, Gonzalez MD, et al. Development and validation of an ECG algorithm for identifying accessory pathway ablation site in Wolff-Parkinson-White syndrome. J Cardiovasc Electrophysiol. 1998;9:2–12.

8. Milstein S, Sharma AD, Guiraudon GM, Klein GJ. An algorithm for the electrocardiographic localization of accessory pathways in the Wolff-Parkinson-White syndrome. PACE. 1987;10(3 Pt 1):555–63.
9. Szabo TS, Klein GJ, Guiraudon GM, Yee R, Sharma AD. Localization of accessory pathways in the Wolff-Parkinson-White syndrome. PACE. 1989;12(10):1691–705.
10. Fitzpatrick AP, Gonzales RP, Lesh MD, Modin GW, Lee RJ, Scheinman MM. New algorithm for the localization of accessory atrioventricular connections using a baseline electrocardiogram. JACC. 1994;23(1):107–16.
11. Takahashi A, Shah DC, Jais P, Hocini M, Clementy J, Haissaguerre M. Specific electrocardiographic features of manifest coronary vein posteroseptal accessory pathways. J Cardiovasc Electrophysiol. 1998;9(10):1015–25.
12. Haissaguerre M, Marcus F, Poquet F, Gencel L, Le Metayer P, Clementy J. Electrocardiographic characteristics and catheter ablation of parahissian accessory pathways. Circulation. 1994;90(3):1124–8.
13. Morady F, DiCarlo LAJ, Baerman JM, de Buitleir M, Kou WH. Determinants of QRS alternans during narrow QRS tachycardia. JACC. 1987;9(3):489–99.
14. Klein GJ, Guiraudon GM, Kerr CR, Sharma AD, Yee R, Szabo T, et al. "Nodoventricular" accessory pathway: evidence for a distinct accessory atrioventricular pathway with atrioventricular node-like properties. JACC. 1988;11(5):1035–40.
15. Tchou P, Lehmann MH, Jazayeri M, Akhtar M. Atriofascicular connection or a nodoventricular Mahaim fiber? Electrophysiologic elucidation of the pathway and associated reentrant circuit. Circulation. 1988;77(4):837–48.
16. Sternick EB, Timmermans C, Sosa E, Cruz FES, Rodriguez L-M, Fagundes M, et al. The electrocardiogram during sinus rhythm and tachycardia in patients with Mahaim fibers. JACC. 2004;44(8):1626–35.
17. Haıssaguerre M, Cauchemez B, Marcus F, Le Metayer P, Lauribe P, Poquet F, et al. Characteristics of the ventricular insertion sites of accessory pathways with anterograde decremental conduction properties. Circulation. 1995;91:1077–85.
18. Hwang C, Martin DJ, Goodman JS, Gang ES, Mandel WJ, Swerdlow CD, et al. Atypical atrioventricular node reciprocating tachycardia masquerading as tachycardia using a left-sided accessory pathway. JACC. 1997;30(1):218–25.
19. Vijayaraman P, Kok LC, Rhee B, Ellenbogen KA. Unusual variant of atrioventricular nodal reentrant tachycardia. Heart Rhythm. 2005;2(1):100–2.
20. Katritsis DG, Becker AE, Ellenbogen KA, Karabinos I, Giazitzoglou E, Korovesis S, et al. Right and left inferior extensions of the atrioventricular node may represent the anatomic substrate of the slow pathway in humans. Heart Rhythm. 2004;1(5):582–6.
21. Katritsis DG, Giazitzoglou E, Korovesis S, Karvouni E, Anagnostopoulos CE, Camm AJ. Conduction patterns in the cardiac veins: electrophysiologic characteristics of the connections between left atrial and coronary sinus musculature. J Interv Card Electrophysiol. 2004;10(1):51–8.
22. Gupta N, Kangavari S, Peter CT, Chen P-S. Mechanism of eccentric retrograde atrial activation sequence during atypical atrioventricular nodal reciprocating tachycardia. Heart Rhythm. 2005;2(7):754–7.
23. Dreifus LS, Haiat R, Watanabe Y, Arriaga J, Reitman N. Ventricular fibrillation. A possible mechanism of sudden death in patients and Wolff-Parkinson-White syndrome. Circulation. 1971;43(4):520–7.
24. Wellens HJ, Durrer D. Wolff-Parkinson-White syndrome and atrial fibrillation. Relation between refractory period of accessory pathway and ventricular rate during atrial fibrillation. Am J Cardiol. 1974;34(7):777–82.
25. Chang RK, Stevenson WG, Wetzel GT, Shannon K, Baum VC, Klitzner TS. Effects of isoflurane on electrophysiological measurements in children with the Wolff-Parkinson-White syndrome. PACE. 1996;19(7):1082–8.

26. Hino H, Oda Y, Yoshida Y, Suzuki T, Shimada M, Nishikawa K. Electrophysiological effects of desflurane in children with Wolff-Parkinson-White syndrome: a randomized crossover study. Acta Anaesthesiol Scand. 2018;62(2):159–66.
27. Klein GJ, Gulamhusein SS. Intermittent preexcitation in the Wolff-Parkinson-White syndrome. Am J Cardiol. 1983;52(3):292–6.
28. Strasberg B, Ashley WW, Wyndham CR, Bauernfeind RA, Swiryn SP, Dhingra RC, et al. Treadmill exercise testing in the Wolff-Parkinson-White syndrome. Am J Cardiol. 1980;45(4):742–8.
29. Goldreyer BN, Damato AN. The essential role of atrioventricular conduction delay in the initiation of paroxysmal supraventricular tachycardia. Circulation. 1971;43(5):679–87.
30. Kapa S, Henz BD, Dib C, Cha Y-M, Friedman PA, Munger TM, et al. Utilization of retrograde right bundle branch block to differentiate atrioventricular nodal from accessory pathway conduction. J Cardiovasc Electrophysiol. 2009;20(7):751–8.
31. Lehmann MH, Denker S, Mahmud R, Tchou P, Dongas J, Akhtar M. Electrophysiologic mechanisms of functional bundle branch block at onset of induced orthodromic tachycardia in the Wolff-Parkinson-White syndrome. J Clin Invest. 1985;76:1566–74.
32. Spurrell RA, Krikler DM, Sowton E. Retrograde invasion of the bundle branches producing aberration of the QRS complex during supraventricular tachycardia studied by programmed electrical stimulation. Circulation. 1974;50(3):487–95.
33. Coumel P, Attuel P. Reciprocating tachycardia in overt and latent preexcitation. Influence of functional bundle branch block on the rate of the tachycardia. Eur J Cardiol. 1974;1(4):423–36.
34. Kerr CR, Gallagher JJ, German LD. Changes in ventriculoatrial intervals with bundle branch block aberration during reciprocating tachycardia in patients with accessory atrioventricular pathways. Circulation. 1982;66(1):196–201.
35. Ceresnak SR, Doan LN, Motonaga KS, Avasarala K, Trela AV, Reddy CD, et al. 50 is the new 70: Short ventriculoatrial times are common in children with atrioventricular reciprocating tachycardia. Heart Rhythm. 2015;12(7):1541–7.
36. Hirao K, Otomo K, Wang X, Beckman KJ, McClelland JH, Widman L, et al. Para-Hisian pacing. Circulation. 1996;94(5):1027–35.
37. Ho RT, Mark GE, Rhim ES, Pavri BB, Greenspon AJ. Differentiating atrioventricular nodal reentrant tachycardia from atrioventricular reentrant tachycardia by DeltaHA values during entrainment from the ventricle. Heart Rhythm. 2008;5(1):83–8.
38. Miller JM, Rosenthal ME, Gottlieb CD, Vassallo JA, Josephson ME. Usefulness of the delta HA interval to accurately distinguish atrioventricular nodal reentry from orthodromic septal bypass tract tachycardias. Am J Cardiol. 1991;68(10):1037–44.
39. Sellers TDJ, Gallagher JJ, Cope GD, Tonkin AM, Wallace AG. Retrograde atrial preexcitation following premature ventricular beats during reciprocating tachycardia in the Wolff-Parkinson-White syndrome. Eur J Cardiol. 1976;4(3):283–94.
40. Zipes DP, DeJoseph RL, Rothbaum DA. Unusual properties of accessory pathways. Circulation. 1974;49(6):1200–11.
41. Ho RT, Rhim ES. Metamorphosis of a tachycardia: what is the mechanism? Heart Rhythm. 2008;5(1):155–7.
42. Bardy GH, Packer DL, German LD, Coltorti F, Gallagher JJ. Paradoxical delay in accessory pathway conduction during long R-P' tachycardia after interpolated ventricular premature complexes. Am J Cardiol. 1985;55(9):1223–5.
43. Dandamudi G, Mokabberi R, Assal C, Das MK, Oren J, Storm R, et al. A novel approach to differentiating orthodromic reciprocating tachycardia from atrioventricular nodal reentrant tachycardia. Heart Rhythm. 2010;7(9):1326–9.
44. AlMahameed ST, Buxton AE, Michaud GF. New criteria during right ventricular pacing to determine the mechanism of supraventricular tachycardia. Circ Arrhythm Electrophysiol. 2010;3(6):578–84.

45. Rosman JZ, John RM, Stevenson WG, Epstein LM, Tedrow UB, Koplan BA, et al. Resetting criteria during ventricular overdrive pacing successfully differentiate orthodromic reentrant tachycardia from atrioventricular nodal reentrant tachycardia despite interobserver disagreement concerning QRS fusion. Heart Rhythm. 2011;8(1):2–7.
46. Waldo AL, MacLean WA, Karp RB, Kouchoukos NT, James TN. Entrainment and interruption of atrial flutter with atrial pacing: studies in man following open heart surgery. Circulation. 1977;56(5):737–45.
47. Waldo AL. From bedside to bench: entrainment and other stories. Heart Rhythm. 2004;1(1):94–106.
48. Stevenson WG, Sager PT, Friedman PL. Entrainment techniques for mapping atrial and ventricular tachycardias. J Cardiovasc Electrophisiol. 1995;6:201–16.
49. Knight BP, Zivin A, Souza J, Flemming M, Pelosi F, Goyal R, et al. A technique for the rapid diagnosis of atrial tachycardia in the electrophysiology laboratory. JACC. 1999;33(3):775–81.
50. Vijayaraman P, Lee BP, Kalahasty G, Wood MA, Ellenbogen KA. Reanalysis of the "pseudo A-A-V" response to ventricular entrainment of supraventricular tachycardia. J Cardiovasc Electrophysiol. 2006;17(1):25–8.
51. Veenhuyzen GD, Quinn FR, Wilton SB, Clegg R, Mitchell LB. Diagnostic pacing maneuvers for supraventricular tachycardia: part 1. PACE. 2011;34(6):767–82.
52. Michaud GF, Tada H, Chough S, Baker R, Wasmer K, Sticherling C, et al. Differentiation of atypical atrioventricular node re-entrant tachycardia from orthodromic reciprocating tachycardia using a septal accessory pathway by the response to ventricular pacing. JACC. 2001;38(4):1163–7.
53. González-Torrecilla E, Arenal A, Atienza F, Osca J, García-Fernández J, Puchol A, et al. First postpacing interval after tachycardia entrainment with correction for atrioventricular node delay: a simple maneuver for differential diagnosis of atrioventricular nodal reentrant tachycardias versus orthodromic reciprocating tachycardias. Heart Rhythm. 2006;3(6):674–9.
54. Veenhuyzen GD, Coverett K, Quinn FR, Sapp JL, Gillis AM, Sheldon R, et al. Single diagnostic pacing maneuver for supraventricular tachycardia. Heart Rhythm. 2008;5(8):1152–8.
55. Bennett MT, Leong-Sit P, Gula LJ, Skanes AC, Yee R, Krahn AD, et al. Entrainment for distinguishing atypical atrioventricular node reentrant tachycardia from atrioventricular reentrant tachycardia over septal accessory pathways with long-RP [corrected] tachycardia. Circ Arrhythm Electrophysiol. 2011;4(4):506–9.
56. Ormaetxe JM, Almendral J, Arenal A, Martinez-Alday JD, Pastor A, Villacastin JP, et al. Ventricular fusion during resetting and entrainment of orthodromic supraventricular tachycardia involving septal accessory pathways. Implications for the differential diagnosis with atrioventricular nodal reentry. Circulation. 1993;88(6):2623–31.
57. Segal OR, Gula LJ, Skanes AC, Krahn AD, Yee R, Klein GJ. Differential ventricular entrainment: a maneuver to differentiate AV node reentrant tachycardia from orthodromic reciprocating tachycardia. Heart Rhythm. 2009;6(4):493–500.
58. Platonov M, Schroeder K, Veenhuyzen GD. Differential entrainment: beware from where you pace. Heart Rhythm. 2007;4(8):1097–9.
59. Nagashima K, Kumar S, Stevenson WG, Epstein LM, John RM, Tedrow UB, et al. Anterograde conduction to the His bundle during right ventricular overdrive pacing distinguishes septal pathway atrioventricular reentry from atypical atrioventricular nodal reentrant tachycardia. Heart Rhythm. 2015;12(4):735–43.
60. Veehuyzen GD, Quinn FR. Principles of entrainment: diagnostic utility for supraventricular tachycardia. Indian Pacing Electrophysiol J. 2008;8(1):51–65.
61. Reddy VY, Jongnarangsin K, Albert CM, Sabbour H, Keane D, Mela T, et al. Para-Hisian entrainment: a novel pacing maneuver to differentiate orthodromic atrioventricular reentrant tachycardia from atrioventricular nodal reentrant tachycardia. J Cardiovasc Electrophysiol. 2003;14(12):1321–8.
62. Perez-Rodon J, Bazan V, Bruguera-Cortada J, Mojal-Garcia S, Manresa-Dominguez JM, Marti-Almor J. Entrainment from the para-Hisian region for differentiating atrioventricular

node reentrant tachycardia from orthodromic atrioventricular reentrant tachycardia. EP Europace. 2008;10(10):1205–11.
63. Man KC, Niebauer M, Daoud E, Strickberger SA, Kou W, Williamson BD, et al. Comparison of atrial-His intervals during tachycardia and atrial pacing in patients with long RP tachycardia. J Cardiovasc Electrophysiol. 1995;6(9):700–10.
64. Sarkozy A, Richter S, Chierchia G-B, De Asmundis C, Seferlis C, Brugada P, et al. A novel pacing manoeuvre to diagnose atrial tachycardia. Europace. 2008;10(4):459–66.
65. Lin JL, Schie JT, Tseng CD, Chen WJ, Cheng TF, Tsou SS, et al. Value of local electrogram characteristics predicting successful catheter ablation of left-versus right-sided accessory atrioventricular pathways by radiofrequency current. Cardiology. 1995;86(2):135–42.
66. Grimm W, Miller J, Josephson ME. Successful and unsuccessful sites of radiofrequency catheter ablation of accessory atrioventricular connections. Am Heart J. 1994;128(1):77–87.
67. Silka MJ, Kron J, Halperin BD, Griffith K, Crandall B, Oliver RP, et al. Analysis of local electrogram characteristics correlated with successful radiofrequency catheter ablation of accessory atrioventricular pathways. PACE. 1992;15(7):1000–7.
68. Otomo K, Gonzalez MD, Beckman KJ, Nakagawa H, Becker AE, Shah N, et al. Reversing the direction of paced ventricular and atrial wavefronts reveals an oblique course in accessory AV pathways and improves localization for catheter ablation. Circulation. 2001;104(5):550–6.
69. Calkins H, Kim YN, Schmaltz S, Sousa J, el-Atassi R, Leon A, et al. Electrogram criteria for identification of appropriate target sites for radiofrequency catheter ablation of accessory atrioventricular connections. Circulation. 1992;85(2):565–73.
70. Chen SA, Tai CT. Ablation of atrioventricular accessory pathways: current technique-state of the art. PACE. 2001;24(12):1795–809.
71. Sacher F, Wright M, Tedrow UB, O'Neill MD, Jais P, Hocini M, et al. Wolff-Parkinson-White ablation after a prior failure: a 7-year multicentre experience. Europace. 2010;12(6):835–41.
72. Calkins H, Langberg J, Sousa J, el-Atassi R, Leon A, Kou W, et al. Radiofrequency catheter ablation of accessory atrioventricular connections in 250 patients. Abbreviated therapeutic approach to Wolff-Parkinson-White syndrome. Circulation. 1992;85(4):1337–46.
73. Lesh MD, Van Hare GF, Schamp DJ, Chien W, Lee MA, Griffin JC, et al. Curative percutaneous catheter ablation using radiofrequency energy for accessory pathways in all locations: results in 100 consecutive patients. JACC. 1992;19(6):1303–9.
74. Wellens HJ, Atie J, Smeets JL, Cruz FE, Gorgels AP, Brugada P. The electrocardiogram in patients with multiple accessory atrioventricular pathways. JACC. 1990;16(3):745–51.
75. Akiyama T. Electrocardiographic clues for multiple accessory pathways in patients with preexcitation syndromes. JACC. 1990;16(4):1029–31.
76. Fananapazir L, German LD, Gallagher JJ, Lowe JE, Prystowsky EN. Importance of preexcited QRS morphology during induced atrial fibrillation to the diagnosis and localization of multiple accessory pathways. Circulation. 1990;81(2):578–85.
77. Kuo JY, Tai CT, Chiang CE, Yu WC, Chen YJ, Tsai CF, et al. Mechanisms of transition between double paroxysmal supraventricular tachycardias. J Cardiovasc Electrophysiol. 2001;12(12):1339–45.
78. Coumel P. Junctional reciprocating tachycardias. The permanent and paroxysmal forms of A-V nodal reciprocating tachycardias. J Electrocardiol. 1975;8(1):79–90.
79. Ticho BS, Saul JP, Hulse JE, De W, Lulu J, Walsh EP. Variable location of accessory pathways associated with the permanent form of junctional reciprocating tachycardia and confirmation with radiofrequency ablation. Am J Cardiol. 1992;70(20):1559–64.
80. Critelli G, Gallagher JJ, Monda V, Coltorti F, Scherillo M, Rossi L. Anatomic and electrophysiologic substrate of the permanent form of junctional reciprocating tachycardia. JACC. 1984;4(3):601–10.
81. Gaita F, Haissaguerre M, Giustetto C, Fischer B, Riccardi R, Richiardi E, et al. Catheter ablation of permanent junctional reciprocating tachycardia with radiofrequency current. JACC. 1995;25(3):648–54.

82. McClelland JH, Wang X, Beckman KJ, Hazlitt HA, Prior MI, Nakagawa H, et al. Radiofrequency catheter ablation of right atriofascicular (Mahaim) accessory pathways guided by accessory pathway activation potentials. Circulation. 1994;89(6):2655–66.
83. Brugada J, Martinez-Sanchez J, Kuzmicic B, Figueiredo MO, Matas M, Pava LF, et al. Radiofrequency catheter ablation of atriofascicular accessory pathways guided by discrete electrical potentials recorded at the tricuspid annulus. PACE. 1995;18(7):1388–94.
84. Gallagher JJ, Smith WM, Kasell JH, Benson DWJ, Sterba R, Grant AO. Role of Mahaim fibers in cardiac arrhythmias in man. Circulation. 1981;64(1):176–89.
85. Ellenbogen KA, Ramirez NM, Packer DL, O'Callaghan WG, Greer GS, Sintetos AL, et al. Accessory nodoventricular (Mahaim) fibers: a clinical review. PACE. 1986;9(6):868–84.
86. Mantovan R, Verlato R, Corrado D, Buia G, Haissaguerre M, Shah DC. Orthodromic tachycardia with atrioventricular dissociation: evidence for a nodoventricular (Mahaim) fiber. PACE. 2000;23(2):276–9.
87. Mark AL, Basta LL. Paroxysmal tachycardia with atrioventricular dissociation in a patient with a variant of pre-excitation syndrome. J Electrocardiol. 1974;7(4):355–64.
88. Haïssaguerre M, Campos J, Marcus F, Papouin G, Clémenty J. Involvement of a nodofascicular connection in supraventricular tachycardia with VA dissociation. J Cardiovasc Electrophysiol. 1994;5:854–62.
89. Sternick EB, Gerken LM, Vrandecic MO, Wellens HJJ. Fasciculoventricular pathways: clinical and electrophysiologic characteristics of a variant of preexcitation. J Cardiovasc Electrophysiol. 2003;14(10):1057–63.
90. Lau EW. Infraatrial supraventricular tachycardias: mechanisms, diagnosis, and management. PACE. 2019;31:490–8.
91. Chauvin M, Shah DC, Haissaguerre M, Marcellin L, Brechenmacher C. The anatomic basis of connections between the coronary sinus musculature and the left atrium in humans. Circulation. 2000;101(6):647–52.
92. Jackman WM, Friday KJ, Fitzgerald DM, Bowman AJ, Yeung-Lai-Wai JA, Lazzara R. Localization of left free-wall and posteroseptal accessory atrioventricular pathways by direct recording of accessory pathway activation. PACE. 1989;12(2):204–14.
93. Sun Y, Arruda M, Otomo K, Beckman K, Nakagawa H, Calame J, et al. Coronary sinus-ventricular accessory connections producing posteroseptal and left posterior accessory pathways. Circulation. 2002;106(11):1362–7.
94. Cipoletta L, Acosta J, Mont L, Berruezo A. Case report posterior coronary vein as the substrate for an epicardial accessory pathway. Indian Pacing Electrophysiol J. 2013;13(4):142–7.
95. Ho I, D'Avila A, Ruskin J, Mansour M. Percutaneous epicardial mapping and ablation of a posteroseptal accessory pathway. Circulation. 2007;115(16):1–4.