Warren Jackman's Art of War:

A Sniper's Approach to Catheter Ablation

Sunny S. Po

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Foreword

Sunny Po has hit a home-run with his recently released book " Warren Jackman's Art of War: A Snipers Approach to Catheter Ablation". This book details Warren Jackman's approach to catheter ablation, as viewed from Sunny Po's perspective. The book reviews in detail all of the "secrets" that Warren Jackman has learned over the years to improve the outcomes of catheter ablation. This book makes it clear that the "devil is in the details". And no detail is left out. A review of the index, tells us that a wide range of topics are included such as positioning and types of electrode catheters, noise reduction in the EP laboratory, the biophysics or RF ablation, differential diagnosis of narrow and wide complex tachycardias, as well as chapters on ablation of all types of ventricular and supraventricular arrhythmias. the array of accessory pathways, and AVNRT. Each chapter provides remarkable detail about how each of these arrhythmias is best mapped and ablated. The drawings and images are plentiful and greatly augment the educational value of this book. At the end of the day, this book is a timely masterpiece. In my mind this is a "must read" for all electrophysiologists now and in the future. What is also remarkable about this book is that it is being made available free of charge. This is of course a reflection of Warren Jackman's and Sunny Po's remarkably generous and collaborative approach to improving the outcomes of catheter ablation. We owe both of these wonderful electrophysiologists a huge debt of gratitude. Congratulations!

Hugh Calkins, M.D., FHRS, FACC, FAHA, FESC
Catherine Ellen Poindexter Professor of Cardiology
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Foreword

It is with great honor and with great delight that I write this foreword to the book that Dr. Sunny Po has written in recognition of Dr. Jackman. Like all fields, electrophysiology has its pioneers and innovators, and Dr. Jackman certainly is one of the brightest minds of our field. He shaped electrophysiology like few others did. He also shaped my personal career after I was lucky enough to be admitted for a fellowship in his lab in the early 90's. I witnessed Dr. Jackman's daily relentless search for new pacing and recording maneuvers, and for new ablation approaches, in order to solve the electrophysiological puzzles that we were confronted with. I still remember attendants struggling to analyse arrhythmias of patients who were referred from all over the US for a second, third or fourth redo ablation attempt. Then Dr. Jackman entered the lab, sat down in front of the computer, started directing the nurse or fellow holding the catheters to different places in the heart, while stimulating and recording. Like a pathologist, he dissected the arrhythmias to their very essence. We started to understand, complexity turned into simplicity. Many hours later, jubilation was often celebrated with a late evening pizza in the lab! When reading through Dr. Po's book, I relive those pioneering moments, and I am happy that so many more can learn about Dr. Jackman's approach. I call it the 'dissection by an EP pathologist', while Dr. Po refers to the sniper's approach. Both are deadly for the arrhythmia…

Apart from his ability to meticulously dissect arrhythmias, Dr. Jackman also has the great gift of teaching. A whole generation of electrophysiologists passed through Oklahoma City in the 90's for courses on invasive electrophysiology and ablation. To some of those, I lend my car so that they could relax their brains and explore some of the Oklahoma scenery… Many more colleagues have attended Dr. Jackman's innumerable lectures around the world. They were and are the TED talks of EP! Thanks to Dr. Po, we all now have a definitive capture of that legacy. In a world where fast ablation, 'learn-while-you-burn', and mainly anatomically directed approaches are the talk of the day, Dr. Po reminds us of the intricacies of noise reduction, methodological electrophysiological diagnosis, and clever ablation with knowledge of the biophysics of energy delivery in the back of our mind. This book is a reminder that sitting in front of a recording system and stimulator, with an analytical mind, not only is needed to help our patients but also is great fun. The book immortalises a way of doing things in EP that never officially got a name, but can rightfully be called the 'Oklahoma

School of Jackman'. All former trainees will immediately recognise each other in the number, amplification, ordering and clipping of the traces as you will find in this book. I only noticed that the early 90's Oklahoma jargon of "TA catheter" (from tricuspid annulus) has now been converted to MAP catheter!

While we can hope and wish that Dr. Jackman has still many years of unraveling and traveling, of lecturing and teaching before him, Dr. Po has made sure that his legacy will stay forever. Dear reader, enjoy discovering, since it is worth it!

Hein Heidbuchel
Chair, Department of Cardiology, Antwerp University, Belgium
President, European Heart Rhythm Association, 2018-2020

Preface

When I finished my EP fellowship at the Johns Hopkins Medical Institute, I had the opportunity to work with Dr. Jackman. My mentor Dr. Hugh Calkins, in his typical upbeat and egoless voice, told me "Sunny, go and learn every trick from Sonny Jackman and come back to teach us two years from now". After two years, it was evident that I was not smart enough to learn all of Dr. Jackman's tricks. After 19 years, I am an old dog now but still learn new tricks from Dr. Jackman and still take notes when I sit in his lectures.

Years ago when I was watching the movie "The Hurt Locker", there was a scene of two opposing snipers. All day long, each sniper only fired one or two shots. It dawned on me that Dr. Jackman's approach to catheter ablation is like what a sniper would do. A sniper patiently searches for the best target and waits for the right moment to fire a lethal shot with minimal collateral damage.

Many electrophysiologists are aware that Dr. Jackman is a perfectionist. Most electrophysiologists do not have the work force and resources to practice like a perfectionist. This sniper's approach may not stand out in a bread-and-butter case but is highly effective when ablation becomes challenging and the operator does not know how to find the ablation target. In this book, I did not intend to provide a comprehensive literature review. Instead, I present to the readers a digested and synthesized version of a perfectionist's approach to ablation. If 1000 electrophysiologists learn the important details in this book and help 1000 patients in his/her EP career, I will help one million patients indirectly. This 1: 1,000,000 conversion is what drives me to write this book.

In this book, I also include my own interpretations and modifications of Dr. Jackman's approach. These modifications are clearly stated as "The author prefers …." or "The author's own practice…". Dr. Jackman may or may not agree with my modifications.

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Author's Note

I hope this book is the first step toward a new paradigm where all new EP books are e-books that are published by major EP societies and sold at a deep discount, or are donated to major EP societies for members to download free of charge. In this way, dissemination of knowledge will be in the hands of EP societies and good EP books will never be out of print.

If anyone wants to use the content in this book for publication or scientific communication, s/he does not need to seek my permission. Please cite the origin of the content as "Sunny S. Po. Warren Jackman's Art of War: A Sniper's Approach to Catheter Ablation. Self-published digital book". Please do not reproduce the figures that I acquired publishers' permission for reuse. For slide presentations, please indicate "courtesy of Sunny Po" somewhere on the slide. If figures are to be used for commercial purposes, please seek my permission. I will ask you to donate $1000 to an EP society of your choice.

Sunny S. Po, MD, PhD

December 12, 2019

Acknowledgements

I am indebted to my former and present fellows (Drs. Steven Hamilton, Edward Koomson, Sunil Mathew, Daniel Sohinki, Andrea Uradu and Ali Yousif) as well as Mr. Mark Burnett and Jody Olsen (Biosense Inc.) for the many constructive comments on this book.

Sunny S. Po, MD, PhD

Chapter 1:
Start an Electrophysiological Study

Dr. Jackman's philosophy of mapping and ablation

1. An EP laboratory free of electromagnetic noise is every electrophysiologist's dream; however, trouble-shooting noise in the midst of an ablation procedure can be very time consuming and is often fruitless. Minimizing the noise level in the EP laboratory has to start at the beginning of the case. Noise reduction in the EP laboratory will be discussed in detail in **Chapter 2**.

2. The fewer catheters the operator uses, the smarter the operator has to be as s/he will have to make many educated guesses. Dr. Jackman often humbly indicates that the reason he uses multiple closely-spaced electrode catheters is because he is not smart enough to figure out the arrhythmia without these catheters.

3. If one wants to learn a new technique or technology, it is best to try it on something that s/he already understands very well. The best example of this occurred when CARTO mapping was still in its infancy. Dr. Jackman used this technology on typical right atrial flutter. At that time, the reentrant circuit of typical flutter was well understood and a high success rate of ablation could be achieved without CARTO mapping. Applying this new technology to something already well understood, Dr. Jackman identified the shortcomings of the CARTO system and worked painstakingly with the Biosense engineers to improve their product until it was eventually suitable for mapping complex arrhythmias.

For example, the pacing maneuver described in **Chapter 7** to reset slow/fast AVNRT to identify the slow pathway participating in AVNRT requires regular practice to ensure that when slow/fast AVNRT is difficult to ablate, operators can effectively use this pacing maneuver to find the antegrade slow pathway participating in AVNRT. Similarly, the author always practices the pacing maneuver to differentiate junctional tachycardia from slow/fast AVNRT when sustained junctional rhythm occurs during isoproterenol infusion.

4. Choose the target carefully and avoid "test burns". This philosophy is indeed the basis of the sniper's approach. Essentially, it says, "patiently search for the best target and then fire one lethal shot." It is well-known that tissue edema occurs within seconds after the onset of RF application. If the target is intramural or epicardial, 15 seconds of RF application may not have affected the target. Premature termination of the RF application here only causes edema and further increases the distance between the ablation electrode and the intramural or epicardial target. In a typical example, accessory pathway conduction returned a few minutes after the initial ablation; however, when the operator repositioned the catheter at the same location where the AP conduction was suppressed, repeated ablations at this location failed to affect the AP conduction anymore.

5. The term "early", "earlier" and "earliest" are relative terms. The site of earliest activation identified by 5 minutes vs. 30 minutes of mapping may be completely different. Since there is no guarantee that the earliest site identified after 30 minutes of mapping is indeed the true origin of the targeted arrhythmia, Dr. Jackman's approach relies heavily (or entirely) on the unipolar electrogram (EGM) recorded on the distal electrode of the ablation catheter. This technique will be discussed in later chapters. That is, Dr. Jackman uses the timing on the bipolar EGM to search for the target and uses the timing and morphology of the distal unipolar EGM (UNI-1) to fine-tune the search. The author worked with Dr. Jackman for nearly 20 years and cannot remember a single case Dr. Jackman did not use unipolar EGMs for mapping and ablation.

6. Triggered sweeps are incredibly helpful when operators are mapping the earliest atrial or ventricular activation. Sadly, Bard Electrophysiology (now Boston Scientific) is the only recording system that reliably provides this function but most electrophysiologists using the Bard system do not take advantage of this incredibly useful function. The timing reference of electro-anatomical mapping systems shares the same concept. In the Bard recording system, the triggered live screen and review screen can be synchronized. When the mapping catheter records timing earlier than the previously recorded earliest timing, the vertical line indicating the earliest timing can be moved synchronously to the current earliest timing on both the triggered live screen and review screen (**Figure 1.1**). During the process of mapping, Dr. Jackman carefully examines the earliest timing on the live screen and continuously updates the earliest timing on both the triggered live screen and review screen by moving the vertical lines progressively earlier. Essentially, the timing of earliest activation was moved progressively earlier until nothing earlier is found. Dr. Jackman uses this triggered sweep function to map any arrhythmia of a focal source (PVC, focal AT, accessory pathway). For example, to map RVOT PVCs, Dr. Jackman may set the trigger at the tall R wave in lead II, III or aVF; the only QRS complex exhibiting on the live and review screens will be the targeted PVC beats (**Figure 1.1D**). This minimizes the distraction from the QRS complexes of the sinus rhythm.

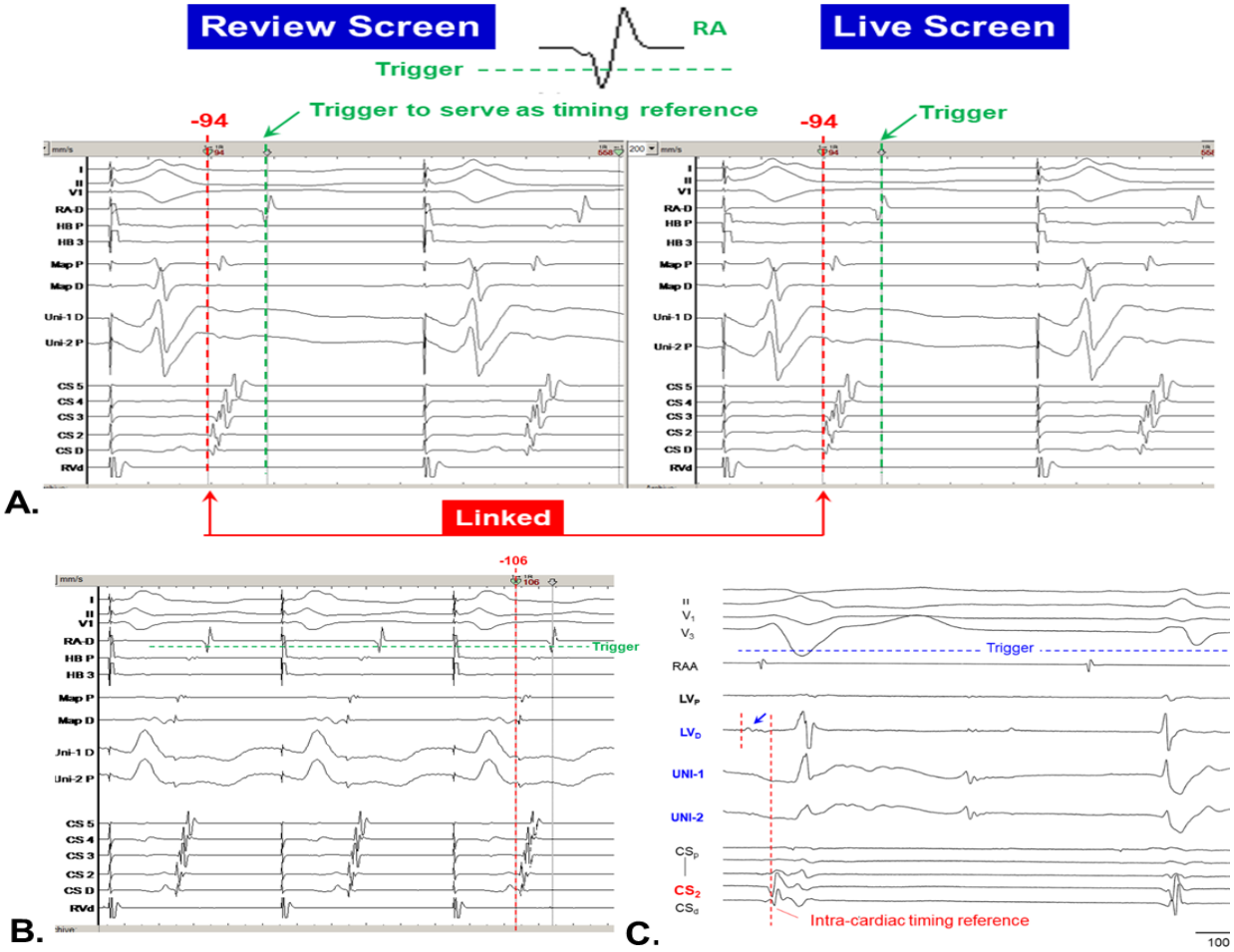


Figure 1.1. Using the triggered sweep function in the Bard recording system to map a focal atrial tachycardia (AT). A. The negative deflection of the RA electrogram (EGM) was set to be the trigger for mapping (horizontal red line in inset). The earliest activation timing (vertical green line) was 74 ms before the trigger. The triggered live screen (right) and review screen (left) are linked. If operators identify earlier timing, the timing lines (vertical green lines) can be moved synchronously to the new earliest timing in both screens. At this moment, the earliest timing was -74 ms. Any EGM crossing this line means it is even earlier. **C.** The timing at another site was even earlier (-106 ms). **C. Using triggered sweeps to map PVCs.** The trigger is set at the negative deflection of lead V3 so that all the QRS complexes from sinus rhythm will be ignored. Both the live and review screens display only the beats that have a deep S wave in lead V3. Because the QRS morphology

tends to vary beat by beat, it is more accurate to set a stable intracardiac ventricular EGM as the timing reference. In this example, only the QRS complex with a deep S wave in lead V3 will be brought to the operator's attention; the timing of the pre-potential (blue arrow) can be assessed with better accuracy using the ventricular EGM in CS2 as the timing reference.

7. Catheter position should be dynamic, depending on the arrhythmia of interest. For instance, the coronary sinus catheter provides limited information about the arrhythmia originating from the right atrial free wall. If ablation reimbursement does not allow the use of another catheter, the coronary sinus catheter can be positioned along the right atrial free wall to provide more information about the tachycardia. Another example is the position of the right ventricular catheter in SVT, which should be dynamic according to the site of the earliest atrial activation (see **Chapter 4** for more details).

8. The magnification or gain of the EGMs should be appropriately scaled to allow EGMs to be easily appreciated. Certainly, it is the operator's personal preference to select the favorite gain. It is advisable to individualize the gain based on the type of arrhythmia. For example, the gain setting of EGMs to map an AP in a healthy heart may be too low to map a left atrial tachycardia in a patient with 3 prior AF ablations. Dr. Jackman also prefers to clip the EGMs to avoid EGM overlapping and crowding in order to recognize small but important changes in the EGM morphology. All the tracings in this book were recorded based on the aforementioned principles.

9. An arrhythmia model is developed according to available data. When a model cannot explain newly acquired data, the model needs to be modified or even abandoned. For example, the "donut model" for the AVNRT reentrant circuit was overhauled because it could not explain why ablation of the slow pathway several centimeters away from the compact AV node can eliminate AVNRT.

10. Mapping of an arrhythmia should start from sites *outside* the area of interest to avoid "seeing the trees but not the forest". Dr. Jackman likes to use the analogy of reading a chest x-ray by examining the bony structures first, before paying full attention to the pulmonary parenchyma and vasculature.

How to position diagnostic catheters

1. His bundle (HB) catheter is the most important catheter for an EP study. The timing of the HB potential often serves as the GPS for the differential diagnosis. The HV interval of a wide-complex tachycardia often provides the clue to the mechanism of tachycardia (**Figure 1.2A-B**).

2. The distal electrode pair of the HB catheter needs to record a distal HB or proximal RBB potential. The proximal electrode pair should record an easily identifiable atrial EGM (**Figure 1.3)**. The electrode size and spacing of the HB catheter plays an important role in the morphology of the atrial, HB and ventricular potentials recorded by the HB catheter. Dr. Jackman prefers a closely-spaced octapolar HB catheter for its accuracy with minimal far-field potentials. The use of closely-spaced electrode catheters led to a lot of Dr. Jackman's unique observations on the reentrant circuits of AVNRT (see **Chapter 7**).

   Operators also need to know if the recorded HB potential is far-field or near-field. A near-field HB/RBB potential is always sharp. For the purpose of diagnosis, a far-field HB potential, which is often triangular or rounded, is perfectly fine. However, for ablation near the AVN, interpreting a distal RBB potential or far-field HB potential as the true HB potential can lead to erroneous localization of the AVN and increase the risk of AVN injury **(Figure 1.3B, right panel).**

3. In Dr. Jackman's practice, the default position for the RV catheter is the parahisian area for all SVTs. The RV catheter should be dynamically positioned according to the site of earliest atrial activation of the SVT (**Figure 1.4A)**.

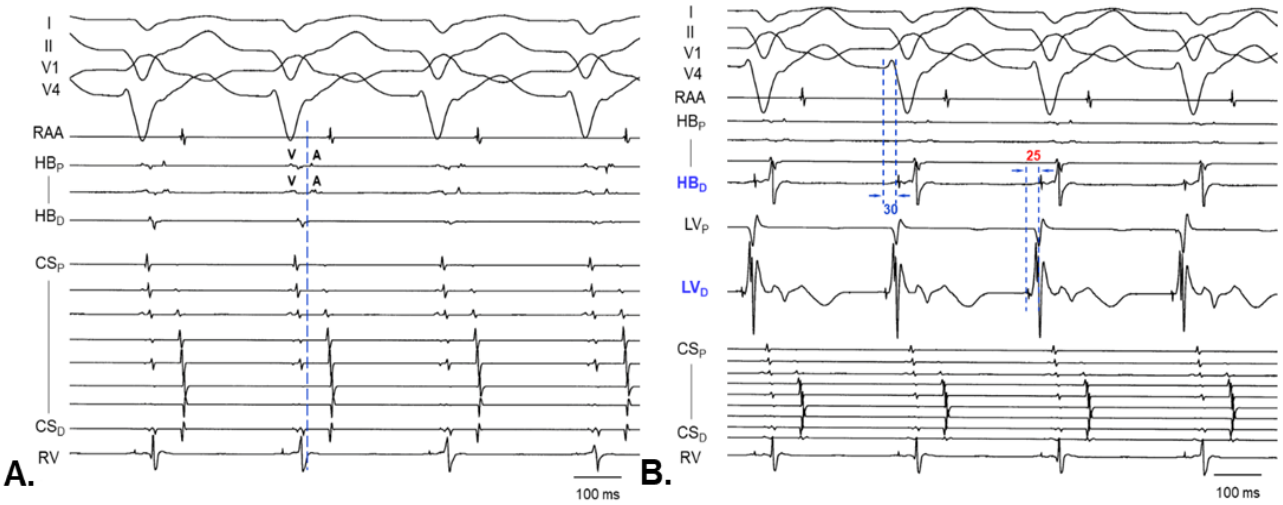


Figure 1.2. HV interval provides important clue to a wide-complex tachycardia. A. A wide-complex tachycardia with RBBB morphology and superior-rightward axis. The site of earliest atrial activation (vertical blue line) was recorded from the His bundle area. Differential diagnosis includes VT and SVT with aberrancy. **B.** The HB/RBB potential was 30 ms after the onset of QRS, excluding the diagnosis of SVT with aberrancy. The LV catheter recorded a Purkinje potential that conducted retrogradely to the HB/RBB (Purkinje-HB=25 ms). VT originating from the left posterior fascicle was successfully ablated.

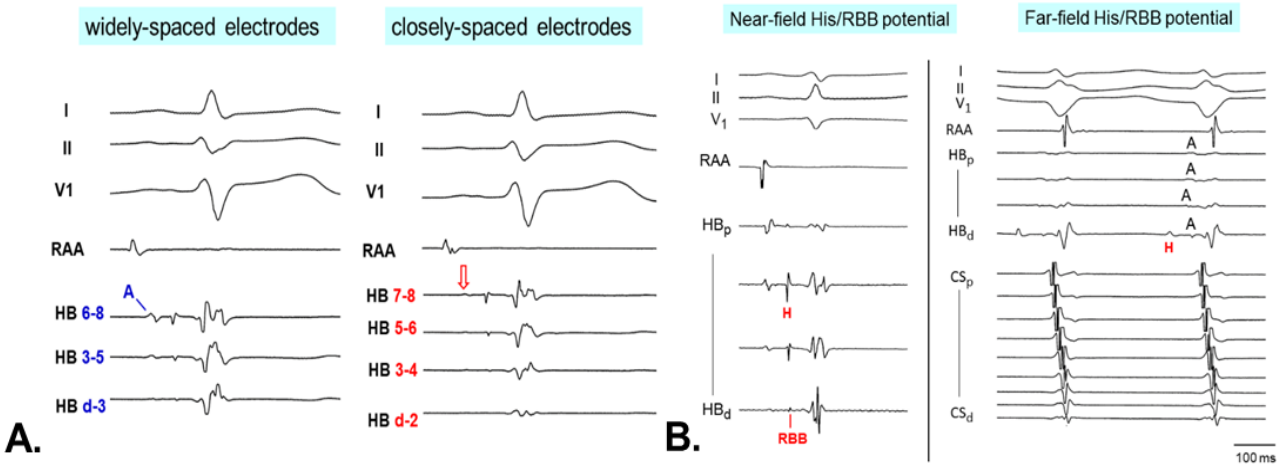


Figure 1.3. A. Differences between the His bundle (HB) potential recorded by widely-spaced vs. closely-spaced electrode catheters. Both panels were recorded by the same 8-pole closely-spaced HB catheter 10 seconds apart. Bipolar EGMs were made of electrodes 1-3, 3-5 and 6-8 (**left panel**) vs. 1-2, 3-4. 5-6 and 7-8 (**right panel**). Note that the left panel indicates that the AVN may be in close proximity to HBp (electrode 6-8) but the right panel shows that the AVN was not near any of the electrode pairs. There was barely any atrial EGM (red empty arrow) recorded on the most proximal electrode pair, indicating that AVN is located more proximal to HBp. The atrial electrogram recorded on bipole 6-8 came from far-field atrial potential more proximal to electrode 8. **B.** HB potentials recorded by closely-spaced octapolar electrode catheters. **Left panel.** HB and RBB potentials were sharp, indicating good contact between the HB catheter and septum. Note that HB_d barely recorded an atrial potential, indicating that the sharp potential recorded by HB_d was not a HB potential but a RBB potential. **Right panel**. Rounded (far-field) HB potential indicates that the HB catheter was not touching the septum. A HB potential like this one should not be used to localize the AVN. However, it is perfectly fine to use it to make differential diagnosis.

4. For slow/slow or fast/slow AVNRT or orthodromic AVRT using a posteroseptal accessory pathway (AP) for retrograde conduction, Dr. Jackman prefers the internal jugular or subclavian venous access for the CS catheter in order to touch the floor of the CS to record the connections between the CS muscle and left atrium that are part of the epicardial posteroseptal AP as well as the reentrant circuit of slow/slow or fast/slow AVNRT (**Figure 1.4A)**. CS catheter should pass the Vieussens valve for AF

or AF-related ATs. During an atrial tachycardia, it is not uncommon to see dissociated atrial and CS muscle potentials masquerading as AF (**Figure 1.4B**).

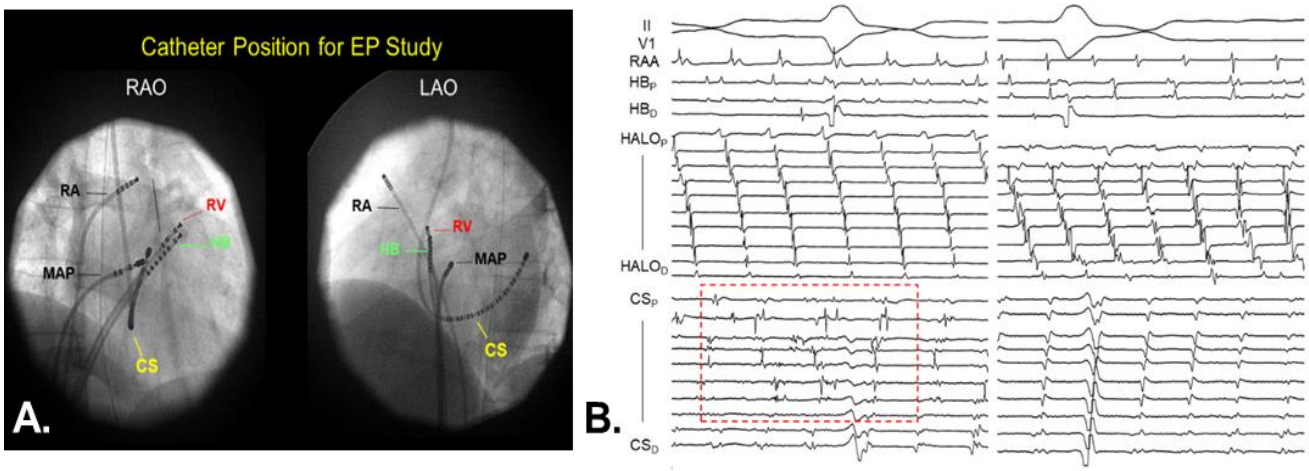


Figure 1.4. Position of catheters in the OU-EP laboratory. The tip of the RA catheter is positioned in the RA appendage. For slow/slow or fast/slow AVNRT or posteroseptal accessory pathways (AP), Dr. Jackman prefers to use either the internal jugular or subclavian venous access in order for the CS catheter to have good contact with the floor of the CS. To perform parahisian pacing, the RV catheter is positioned superior to the HB catheter and advanced to the basal anteroseptal RV. In this particular example, the mapping catheter was positioned near the fast pathway to determine if this tachycardia was a slow/fast AVNRT. For SVTs, Dr. Jackman prefers to position the mapping catheter in the inferior triangle of Koch in order to record slow pathway conduction or posteroseptal AP conduction. **B. Left Panel.** Disorganized EGMs were recorded from the proximal CS due to dissociation of the atrial and CS muscle potentials (in red dotted box). This tachycardia appeared to be AF. **Right panel.** After the CS catheter was advanced beyond the Vieussens valve, it was evident that this was an atrial tachycardia, not AF.

5. The tip of a HALO catheter should be positioned inside the CS ostium for atrial flutter or right free-wall AP; far-field ventricular potentials should be visible on some of the electrodes. This is to ensure that the HALO catheter is positioned along the tricuspid annulus. If the tip of the HALO catheter is positioned behind the tendon of Todaro, it can generate confusing patterns of activation (**Figure 1.5)**.

6. The RA catheter is best positioned in the RA appendage for SVTs for the following reasons (**Fig. 1.4A)**. First, RA appendage is covered with pectinate muscle and in close proximity to the Bachmann's bundle. Pacing from the RA appendage conducts to both atria quickly. Second, the RA appendage is a very stable location and can serve as a favorable timing reference. In the middle of mapping a complex atrial tachycardia, the relative atrial timing among the RA appendage, His-bundle region and coronary sinus can be used to monitor if the atria tachycardia has changed. If the right atrial catheter had been positioned along the RA free wall, it could easily drift off and defeat this purpose. Third, the RA appendage is located at the 10 to 11 o'clock position along the tricuspid annulus, providing useful information about a right free wall accessory pathway and atrial tachycardias originating in the RA appendage.

7. Operators may find out that it takes more effort to record good EGMs using a closely-spaced electrode catheter. This paradox comes from the fact that a closely-spaced electrode catheter records substantially less far-field EGMs. Basically, unless there is good catheter-tissue contact, the operator may not see any EGMs on the electrodes that do not touch the cardiac tissue.

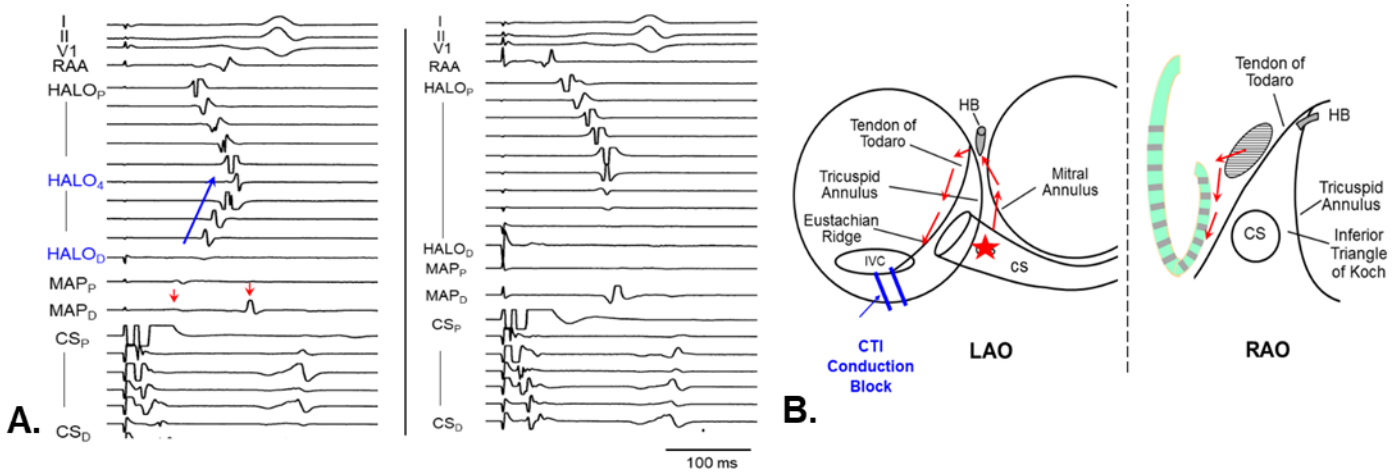


Figure 1.5. Position of the HALO catheter can affect judgment of conduction block across the cavo-tricuspid isthmus (CTI) line. A. Left Panel. During ablation of the CTI, despite a double potential on the ablation catheter (red arrows), the pattern of atrial activation along the HALO catheter (blue arrow) was not affected at all after 20 RF applications (30 watts, 30 seconds for each). This finding suggests that conduction block across CTI was not achieved yet. Note that no ventricular EGM was recorded on the HALO catheter, indicating that the HALO catheter was not positioned close to tricuspid annulus. The tip of the HALO catheter ($HALO_d$) was behind the tendon of Todaro. **Right Pane**l. The HALO catheter was rotated toward the tricuspid annulus. Although the HALO catheter still was not at the tricuspid annulus (no ventricular activation recorded on the HALO catheter), the activation pattern changed significantly. One more RF application produced conduction block across the CTI. **B.** The distal electrodes of a HALO catheter were positioned behind the tendon of Todaro. In the presence of CTI conduction block, paced wave front from proximal CS may preferentially conduct to the left interatrial septum and then enter the right atrium from the true interatrial septum, leading to the activation from $HALO_d$ to $HALO_4$ as illustrated in **Figure 1.5A**.

Parahisian Pacing

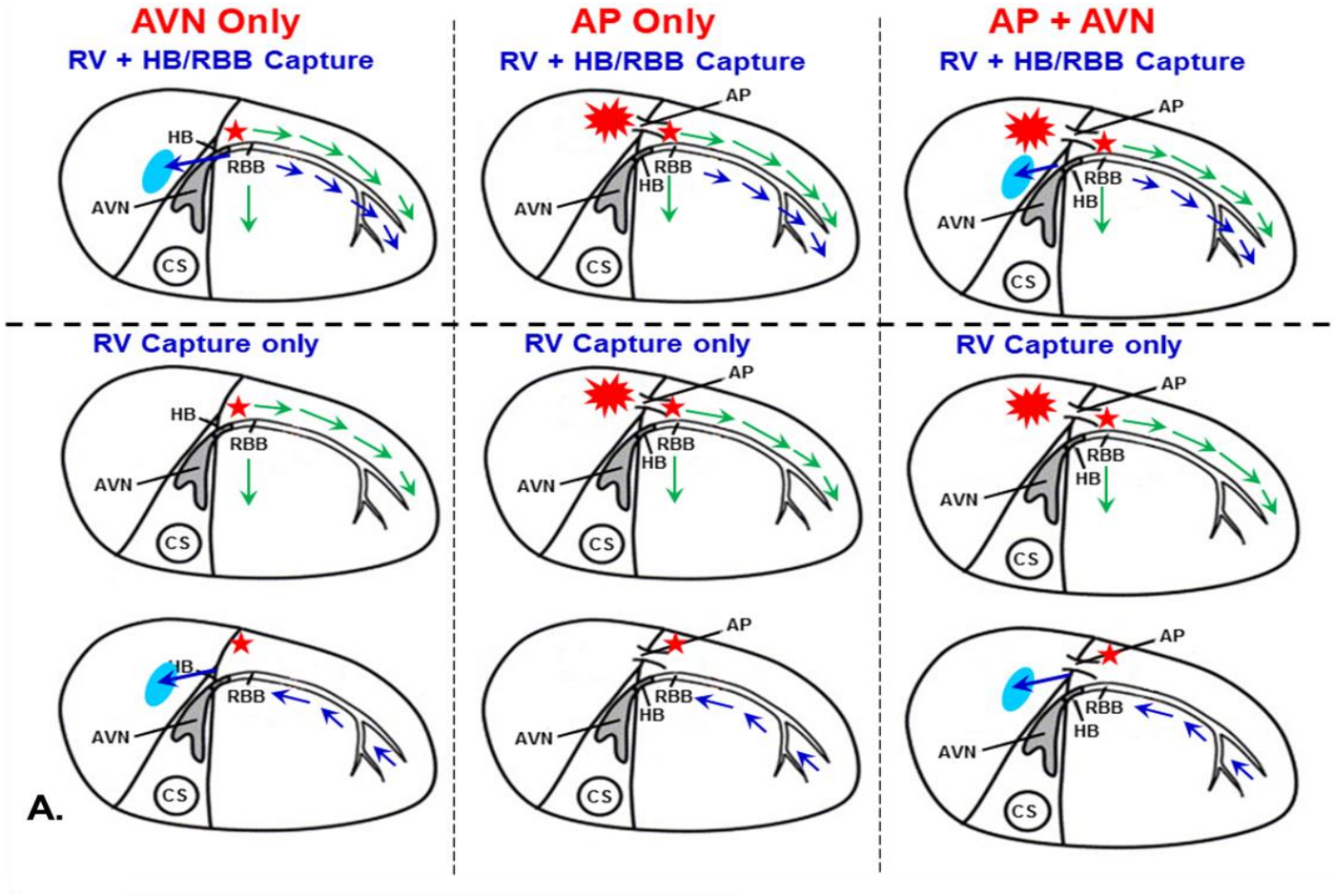
Dr. Jackman pioneered the technique of parahisian pacing to differentiate retrograde conduction over a septal AP from AVN. The HB catheter needs to be positioned at a site where the distal pair of electrodes records a clear HB potential and the proximal electrode pair records an easily visible atrial EGM. To position the HB catheter, operators may follow the superior aspect of the HB catheter to advance the RV catheter into the RV and then position the tip of the RV catheter slightly more ventricular to the tip of the HB catheter (**Fig. 1.4A**). Dr. Jackman prefers to set the pacing output at the level capturing the HB/RBB and simply wait for the RV catheter to slide back and forth during respiration to lose HB/RBB capture. An alternative is to increase and decrease the pacing output to capture HB/RBB and lose HB/RBB capture, respectively.

Figure 1.6 illustrates the electrophysiological basis for parahisian pacing. Indeed, parahisian pacing does not really capture the HB but the proximal RBB. The term parahisian pacing was adopted because “para-right bundle branch pacing” sounded awkward. Parahisian pacing can be summarized into 3 steps: (1) determine RV capture only vs. RV and HB/RBB capture, (2) compare the atrial activation sequence between the HB/RBB captured beat and non-captured beat and (3) compare the stimulus-atrial interval or HB/RBB-atrial interval between the captured beat and non-captured beat.

1. Determine RV capture vs. RV and HB/RBB capture

When the HB/RBB is captured, rapid retrograde AVN conduction activates the fast pathway area posterior and inferior to the HB area (light blue area, **Figure 1.6**), producing a shorter stimulus-atrial interval. Both ventricles are activated by rapidly conducting wave fronts through the His-Purkinje system: (1) rapid antegrade conduction over the RBB and (2) rapid retrograde conduction to the bifurcation of the distal HB, followed by rapid antegrade conduction over the LBB. Therefore, nearly simultaneous activation of the LBB and RBB produces a narrow QRS complex. If only RV is captured, ventricular activation is not mediated by the His-Purkinje system and it produces a wider QRS complex, similar to PVCs originating from the inferior aspect of

the RV outflow tract. Ventricular activation eventually engages peripheral RBB and conducts retrogradely to the atrium, producing a longer stimulus-atrial interval.



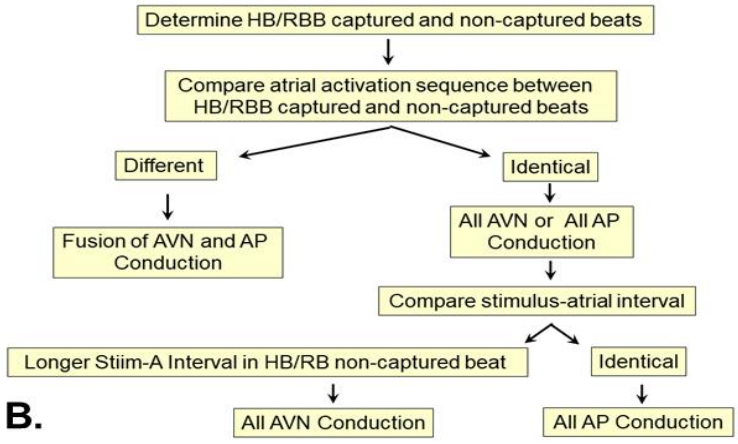


Figure 1.6: A. Three major patterns of parahisian pacing: AVN only, AP only and fusion. Red asterisk: pacing site; red area: activated by retrograde AP. Light blue area: activated by retrograde AVN fast pathway. Green arrows: ventricular myocardial activation. Blue arrows: antegrade or retrograde RBB activation. **B.** Algorithm of interpreting parahisian pacing results.

One can determine if the pacing site is good for parahisian pacing by looking at the interval between the stimulation artifact and the local ventricular activation. The stimulation artifact should be followed immediately by the local ventricular activation in the HB region (**Figure 1.7A-C**). If there is a clear iso-electrical interval between the two, the RV catheter needs to be pulled back toward the annulus (**Figure 1.7D**).

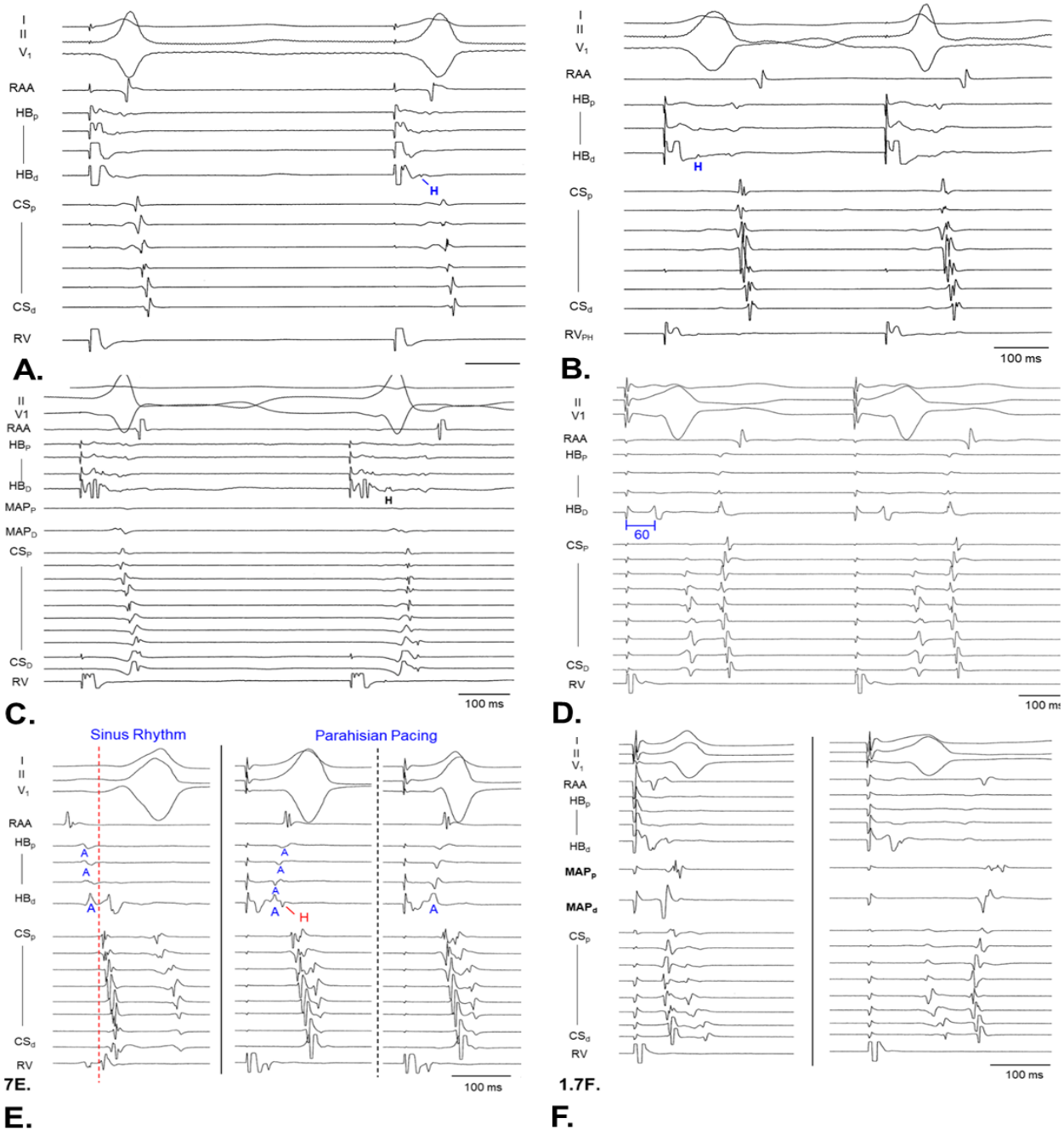


Figure 1.7. Examples of parahisian pacing. A. Parahisian pacing before ablation: all AP. Note that the stimulus-atrial interval remained unchanged regardless of HB/RBB capture. left beat: HB/RBB capture; right beat: HB/RBB non-capture. **B.** Parahisian pacing after ablation: all AVN. Note that the stimulus-atrial interval was longer if HB/RBB was not captured (left beat). **C.** In a different patient with a posteroseptal AP, HB/RBB capture and non-capture produced different atrial activation sequences (fusion). When HB/RB was captured (left beat), retrograde AVN conduction leads to a short stimulus-atrial interval. The atrial activation sequence reflects pure retrograde fast pathway conduction. **D.** In another patient with an anteroseptal AP, the RV catheter was deep in the RV, as evidenced by a long stimulus-ventricular interval (60 ms). **E.** In another patient with an anteroseptal AP, the ventricular activation in the anteroseptal area was early (red dotted line) in sinus rhythm (**left panel**). **Middle and right panels**: parahisian pacing. The retrograde HB potential was inscribed *after* local atrial activation because AVN was not involved in VA conduction. **F. Left panel:** The stimulus-atrial interval was very short (25 ms, measured at RA). When the pacing catheter was advanced further into the RV, the VA interval was markedly lengthened (**right panel**), indicating that the short VA interval was a result of direct RA capture.

Since the parahisian pacing manuscript was published in 1996, many patterns of responses have been discovered, some of which were caused by partial ventricular capture or partial HB/RBB capture as well as the presence of RBBB or LBBB. The QRS morphology produced by parahisian pacing depends on the mass of ventricular muscle that is captured as well as the timing of the paced ventricular wave front that engages the His-Purkinje system. Although a narrower QRS complex is produced by HB/RBB capture in the vast majority of cases, determining HB/RBB capture solely by narrowing of the QRS complex can be misleading. The OU-EP group has many examples in which HB/RBB was not captured in a narrower QRS complex or HB/RBB was captured in a wide QRS complex. The most reliable means to determine HB/RBB capture is disappearance of the HB/RBB potential. The author appreciates the fact that many EP laboratories do not have the resources of using closely-spaced HB catheter to record the retrograde HB/RBB potential. RV pacing by widely-spaced catheter also produces a longer period of amplifier saturation, rendering the results of parahisian pacing difficult to interpret. For confusing patterns of parahisian pacing, the ablation catheter can be positioned in the HB region to record the retrograde HB/RBB or to pace the RV.

2. Compare the atrial activation sequence between HB/RBB captured beat and non-captured beat

Similar to preexcitation caused by fusion of the antegrade AP and AVN conduction, retrograde conduction can be all AVN, all AP or fusion of AVN and AP.

1. In the absence of retrograde AP conduction at a given pacing CL, the activation sequence between the HB/RBB captured beat and non-captured beat will be identical. The site of earliest activation is usually the fast pathway area. If retrograde fast pathway conduction is absent, retrograde conduction may be mediated by the retrograde slow pathway. The site of earliest atrial activation will be located in the inferior triangle of Koch (the usual slow pathway area) or inside the CS. These patients have poor retrograde fast pathway conduction and often have slow-slow or fast-slow AVNRT.

2. In the presence of retrograde AP conduction, if retrograde AVN conduction is absent at that pacing CL, both atria are activated by retrograde AP conduction. Retrograde atrial activation sequence reflects retrograde AP conduction.

3. In the presence of both retrograde AP and AVN conduction, the retrograde atrial activation sequence reflects both retrograde AP and AVN conduction. At shorter pacing CL, either the retrograde AP or AVN conduction may block, leading to shift of the retrograde atrial activation sequence.

Typically, the HB/RBB captured beat engages the His-Purkinje system immediately, leading to retrograde conduction that favors AVN conduction. Retrograde AP conduction is therefore more visible in the HB/RBB non-captured beat (**Figure 1.7C**). If the stimulus-atrial interval of an atrial area is not affected by HB/RBB capture or non-capture, this area is most likely activated by retrograde AP conduction.

4. A rule of thumb is that if the atrial activation sequence is identical, retrograde conduction is mediated entirely by AVN or entirely by AP, which can be differentiated by Step-3. If the retrograde atrial activation sequence is different between the HB/RBB captured and non-captured beats, it indicates the presence of retrograde AP conduction.

Occasionally, parahisian pacing may reveal retrograde fast pathway conduction in one beat and slow pathway conduction in another beat. Comparing to the retrograde AVN slow pathway, the path length of the retrograde fast pathway is significantly shorter and the conduction velocity is substantially faster. When retrograde fast pathway conduction is normal, retrograde slow pathway conduction is usually not observed. Therefore, it is theoretically not possible to observe fusion of retrograde fast pathway and slow pathway in a single conducted beat. Dr. Jackman has never seen this, either. However, it is not uncommon to see retrograde conduction switch between retrograde fast pathway and slow pathway when HB/RBB capture or non-capture occurs (**Figure 1.8F**). Of note, fusion of retrograde slow pathway and AP conduction is not uncommon if retrograde fast pathway conduction is weak.

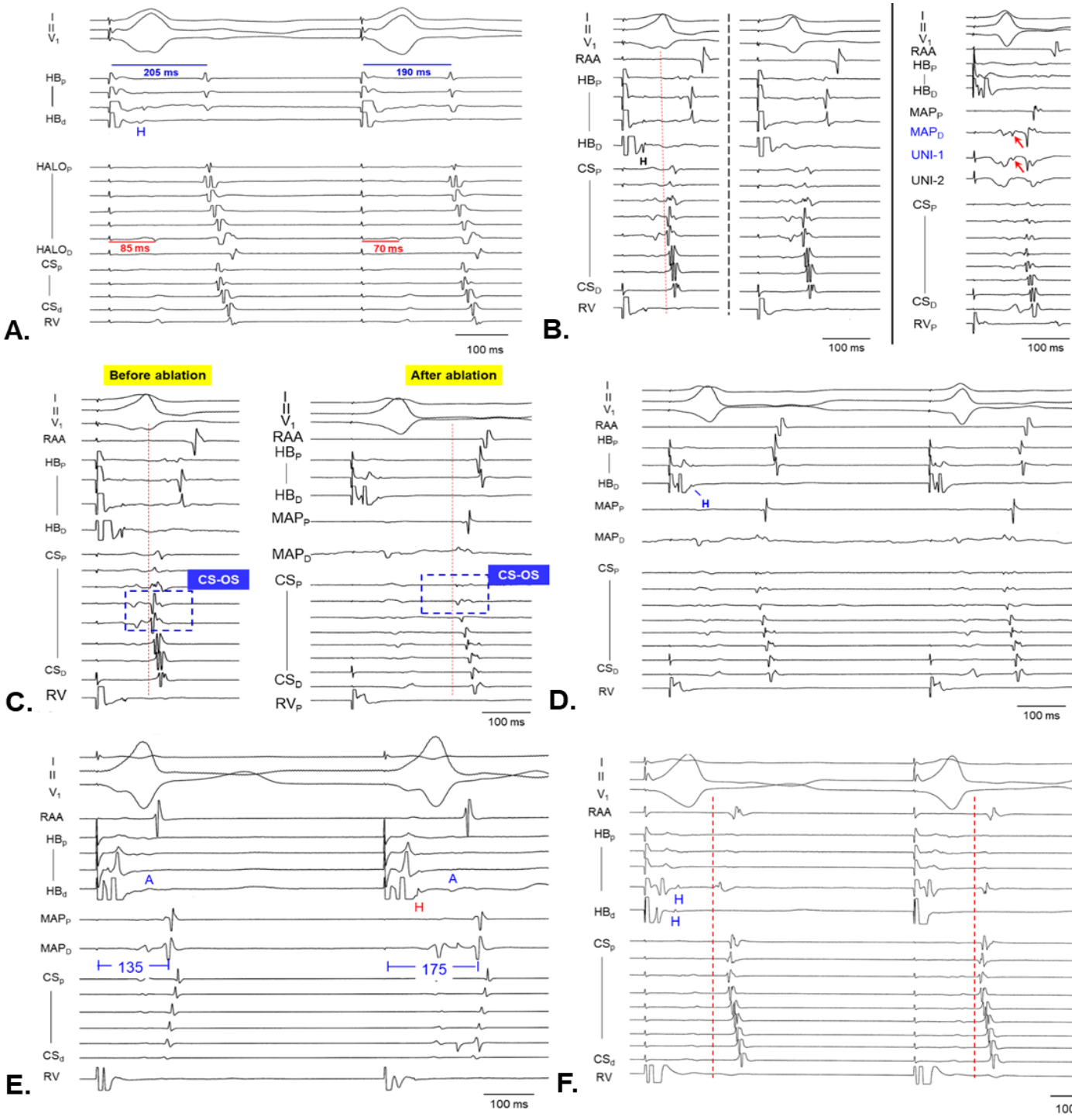


Figure 1.8. Examples of parahisian pacing. A. it appears that retrograde conduction was mediated by AVN because the atrial activation sequence remained unchanged and the stimulus-atrial interval increased from 190 ms to 205 ms. Note that the stimulus-ventricular interval was increased from 70 ms to 85 ms, accounting for the 15 ms increase in the stimulus-atrial interval. Retrograde conduction was over a right free wall AP in this case. **B.** In a patient with a posteroseptal AP, parahisian pacing showed retrograde AP conduction only (left and middle beats). The site of earliest atrial activation was recorded from proximal CS (red vertical line). Note that the stimulus-atrial interval was not affected by HB/RBB capture (middle beat). An AP potential (red arrow) was recorded from the CS ostium (right beat). Ablation here led to prolongation of the VA interval. **C.** The site of earliest atrial activation remained at the ostium of CS after ablation but the VA interval was substantially longer after ablation. **D.** After ablation, parahisian pacing showed that the retrograde conduction was mediated by the slow pathway of the AVN. Ablation was successful. **E.** In another patient, HB/RBB capture (left beat) did not narrow the QRS complex. The stim-

atrial interval was lengthened from 135 to 175 ms when HB/RBB was not captured (right beat). This common observation indicates that judging HB/RBB capture solely by QRS narrowing can be misleading. **F**. In a different patient with both fast/slow and slow/fast AVNRT, parahisian pacing produced two different atrial activation sequences. When HB/RBB was not captured (**left panel**), retrograde conduction was mediated by the fast pathway. When HB/RBB was captured (**right panel**), it conducted to the atrium over the slow pathway. Vertical red line: timing of earliest atrial activation.

5. The stimulus-atrial interval of AP conduction over an anteroseptal AP is usually short; the retrograde HB potential may be inscribed after the local atrial activation (**Figure 1.7E**). However, if stimulus-atrial interval is very short (e.g. 30 ms), one needs to consider the following possibilities. First, the pacing catheter was on the atrial side of the anterior septum, thereby capturing both the atrium and ventricle (**Figure 1.7F**). In this situation, the stimulus artifact is usually immediately followed by atrial EGMs. If the pacing catheter is advanced slightly into the RV and the VA interval becomes substantially longer, the very short VA interval is most likely caused by direct atrial capture. Second, RV pacing directly captured the anteroseptal AP or the pacing site is very close to the ventricular end of the AP, leading to a very short VA interval.

3. Compare the stimulus-atrial interval or HB/RBB-atrial interval between the HB/RBB captured beat and non-captured beat.

In the absence of retrograde AP conduction, the HB/RBB non-captured beat has to propagate to the RV apex or trans-septally to the LV to engage the peripheral Purkinje system, followed by retrograde conduction back to the AVN and atrium. This conduction pathway leads to a longer stimulus-atrial interval in comparison to that of a HB/RBB captured beat.

In the absence of retrograde AVN conduction, the ventricular wave front of either the HB/RBB captured beat or non-captured beat engages the ventricular end of the AP, regardless of how or when the ventricular activation engages the peripheral Purkinje system. The stimulus-atrial interval will therefore be identical between the two beats. In other words, retrograde AP conduction does not care if HB/RBB capture occurs or not.

One important caveat to consider while measuring the stimulus-atrial interval is that an increase in the stimulus-atrial interval of a HB/RBB non-captured beat may be caused by delay in the stimulus-ventricular interval. In the example shown in **Figure 1.8A**, it appears that retrograde conduction was mediated by AVN because the atrial activation sequence remained unchanged and the stimulus-atrial interval increased from 190 ms to 205 ms. However, all the observed delay was caused by a 15 ms increase in the stimulus-ventricular interval. Retrograde conduction was over a right free wall AP in this case. Essentially, stimulus-atrial interval is a surrogate for VA interval. If the stimulus-ventricular interval changes, the stimulus-atrial interval must be corrected to be used as a surrogate for VA interval.

Parahisian pacing is most helpful:

i. To differentiate retrograde fast pathway conduction (e.g. slow/fast AVNRT) from retrograde conduction over an anteroseptal or mid-septal AP (**Figure 1.7**).

ii. To differentiate retrograde slow pathway conduction (e.g. slow/slow or fast/slow AVNRT) from retrograde conduction over a posteroseptal AP (**Figure 1.8**).

iii. To differentiate retrograde fast pathway conduction from slow pathway conduction (**Figure 1.9A-B**).

iv. To evaluate the success of ablation if atrial activation sequence appears to be confusing (e.g. slow pathway vs. posteroseptal AP or fast pathway vs. anteroseptal AP; **Figure 1.7, 1.8**).

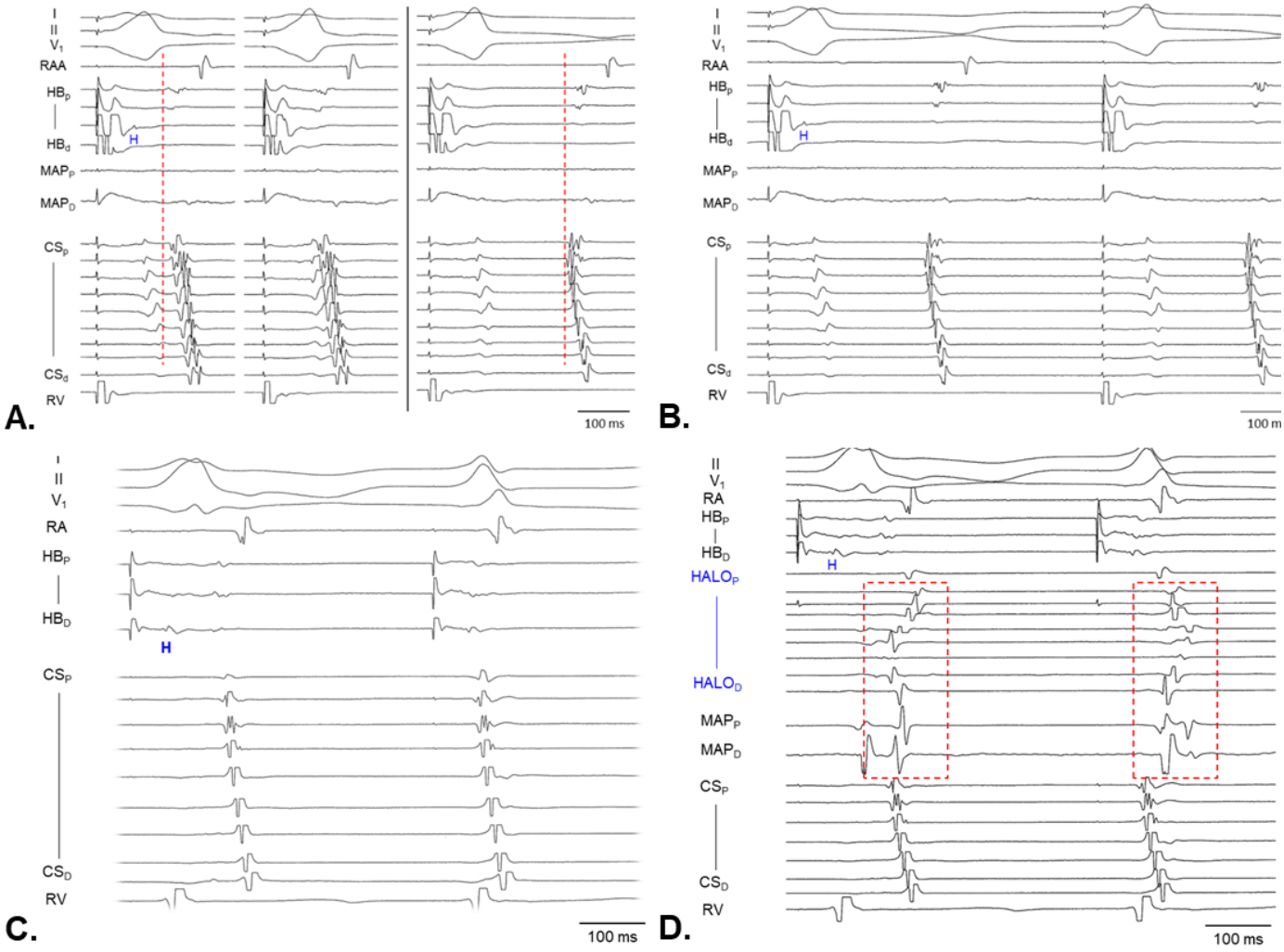


Figure 1.9. Examples of parahisian pacing. A. Left panel. Parahisian pacing showed retrograde conduction over the fast pathway. **Right panel.** At shorter pacing CL, atrial activation changed. Vertical red line: timing of earliest atrial activation. **B.** Parahisian pacing was performed immediately when the atrial activation changed, demonstrating retrograde conduction over the retrograde slow pathway. This patient had both slow/fast and slow/slow AVNRT. **C-D. Parahisian pacing missed a right lateral AP. C.** Parahisian pacing showed AVN conduction. Note that the RA catheter was mistakenly positioned at the posterior RA free wall distant from the tricuspid annuls, leading to failure of detecting AP conduction. **D.** With the help of a HALO catheter along the tricuspid annulus, different atrial activation sequence between the left and right beat (fusion) was evident, indicating the presence of retrograde AP conduction.

Parahisian pacing was never developed to be a shortcut to determine the presence or absence of retrograde AP conduction by using the fewest number of catheters. Indeed, parahisian pacing may miss the presence of an AP if the site adjacent to the AP is not covered by a catheter (**Figure 1.9C-D**). In addition, sympathetic tone, which enhances retrograde AVN conduction, may produce confusing patterns of atrial activation. During mapping of an anteroseptal or midseptal AP, it is advisable to check parahisian pacing periodically to make sure that retrograde conduction is not "contaminated" by the fast pathway conduction to avoid unknowingly mapping retrograde AVN conduction (**Figure 1.10**). If it still looks confusing, a few beats of AVRT will serve as the template for comparison. In the presence of fusion, operators may try the following maneuvers to map retrograde AP conduction.

1. Pace from a ventricular site in close proximity to the AP to eliminate the "contamination" caused by retrograde AVN conduction. For example, to map a posteroseptal AP, pacing from the basal posteroseptal RV may produce "pure" retrograde AP conduction.

2. Pace the ventricle at the CL that only retrograde AP conduction is present.

3. Map retrograde AP conduction during orthodromic AVRT, which eliminates the contamination of retrograde AVN conduction. However, catheter stability may be challenging.

4. If sites demonstrating fusion are distant from the AP, operators may ignore fusion after carefully comparing the atrial activation sequence between pacing and orthodromic AVRT to verify that the area of interest is activated by retrograde AP conduction (**Figure 1.10D**).

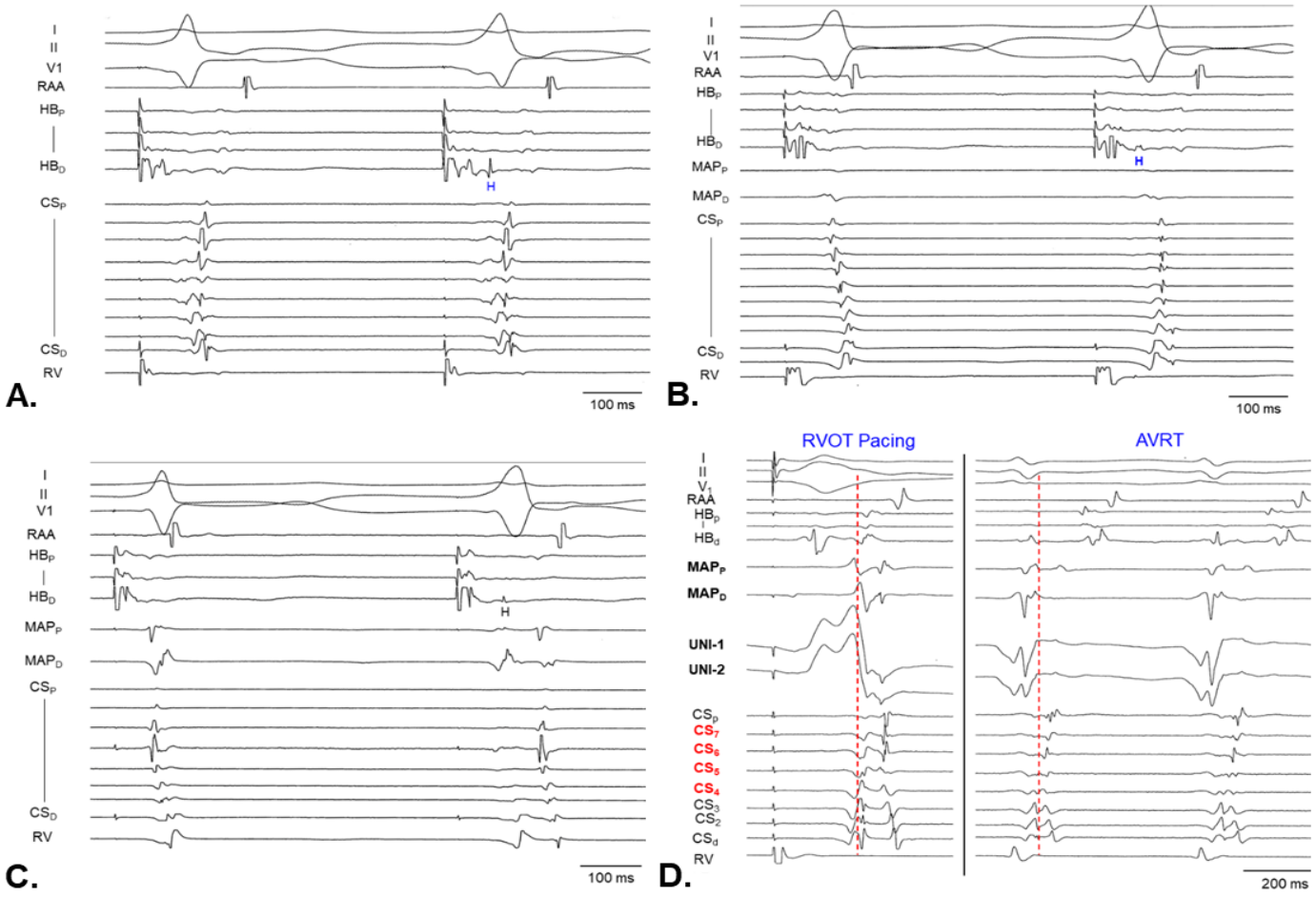


Figure 1.10. Results of parahisian pacing can be affected by higher sympathetic tone or infusion of isoproterenol that enhances retrograde fast pathway conduction. A. Baseline state. Parahisian pacing indicated that retrograde conduction was over a posteroseptal AP. **B.** After isoproterenol infusion, fusion of AP and AVN was evident (different atrial activation sequence between the left and right beat). **C.** After successful ablation: all AVN conduction. **D.** In another patient with a left lateral AP, pacing from the RVOT showed that retrograde atrial activation was fusion of AVN and AP conduction (**left panel**). AVRT was induced to determine if CS-4 to CS-7 were activated by retrograde AP conduction (**right panel**). It was evident that during RVOT pacing, the RA and HB region were activated by AVN conduction. CS-4 to CS-7 were activated by AP conduction. Mapping was therefore performed during RVOT pacing for better catheter stability. Vertical dotted line: timing of earliest atrial activation.

When parahisian misses AP conduction

1. The result of parahisian pacing often depends on the pacing CL. That is, one cannot declare there is no AP conduction based on one pacing CL. The author prefers to start at CL 20-30 ms shorter than the sinus CL and deliver decremental RV pacing to expose all different atrial activation sequences (**Fig. 1.11, 1.12**). Importantly, the stimulus-to-atrial interval cannot be used to differentiate fast pathway from slow pathway or AP conduction. **Figure 1.12D** illustrates an example of a long stimulus-to-atrial

interval (245 ms). Mapping of retrograde conduction proved that it was mediated by the fast pathway, not slow pathway,

2. The following APs can be missed by parahisian pacing:

   I. Left anterior or left anterolateral AP: The ventricular end of the AP is too far from the pacing site and AVN wins the retrograde conduction race.

   II. Slowly or decrementally conducting AP (i.e. PJRT): AVN wins the race because AP conduction is too slow.

   III. On rare occasions, a right free wall AP can be missed: If the RA catheter is not positioned in the RAA, which is adjacent to the anterolateral tricuspid annulus, some of the right free wall AP can be missed by parahisian pacing. This problem can occur if prior ablation causes regional conduction delay or block that allows the AVN to win the race (**Fig. 1.11**). It also underscores the importance of not relying on one single pacing CL of parahisian pacing to evaluate retrograde conduction.

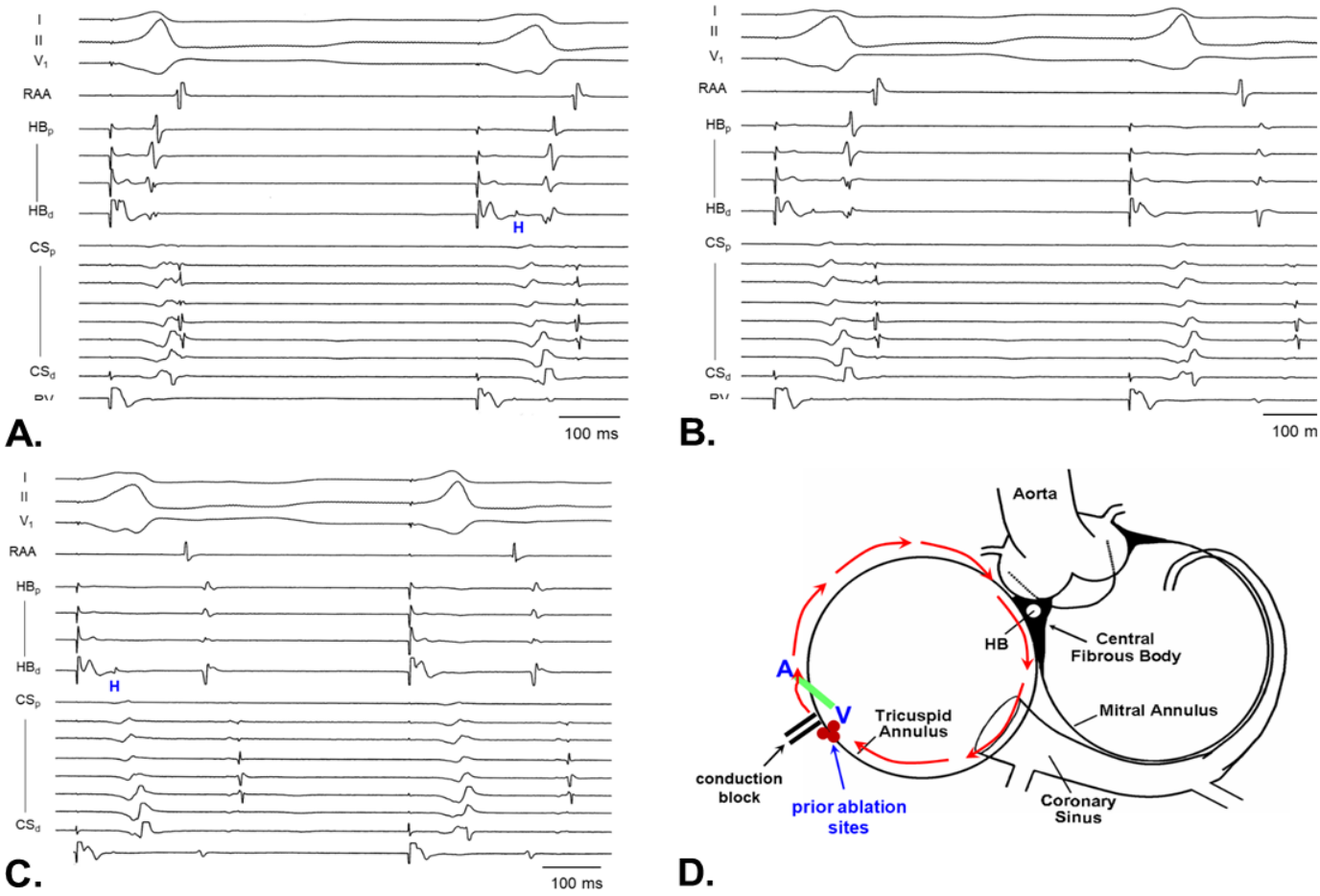


Figure 1.11. Parahisian pacing at a fixed pacing CL may miss AP conduction. A. In a young woman with two prior ablations, parahisian pacing at 640 ms showed retrograde AVN conduction. **B.** At pacing CL of 580 ms, atrial activation sequence changed. **C.** Parahisian pacing at 580 ms revealed retrograde AP conduction. **D.** Prior ablation, which was too septal to the AP location, created a line of block. The atrial activation wave front had to propagate in a clockwise direction around the annulus. A and V: atrial and ventricular end of the AP.

Start an EP Study

Based on Dr. Jackman's meticulous approach, the following description is a simplified stepwise approach that the author uses in the EP laboratory. The rationale of each step will be discussed in various chapters of this book.

1. Position all catheters at relevant and stable positions. If the patient is known to have slow/slow AVNRT, fast/slow AVNRT or a posteroseptal AP, the subclavian or internal jugular vein will be used for CS catheter placement in order to have good contact with the floor of the CS to record the CS-atrial connections that are part of the connections between the slow pathway and atrium or between the atrium and an epicardial AP.

2. Start parahisian pacing at a CL slightly shorter than the sinus CL, followed by decremental RV pacing. Repeat parahisian pacing at each CL when the atrial activation sequence changes to determine the mechanism of each retrograde conduction pattern until VA block is reached (**Figure 1.11, 1.12**). It is not uncommon to see retrograde AP, slow pathway and fast pathway conduction in the same patient who has both AVRT and AVNRT. The mechanism of tachycardia often can easily be appreciated if the mechanism of each activation sequence has been verified at the beginning of the EP study. Of note, decremental RV pacing should be performed at the pacing output not capturing the HB/RBB because sudden shortening of the VA interval by HB/RBB capture impairs one's judgment on subtle changes in the atrial activation sequence.

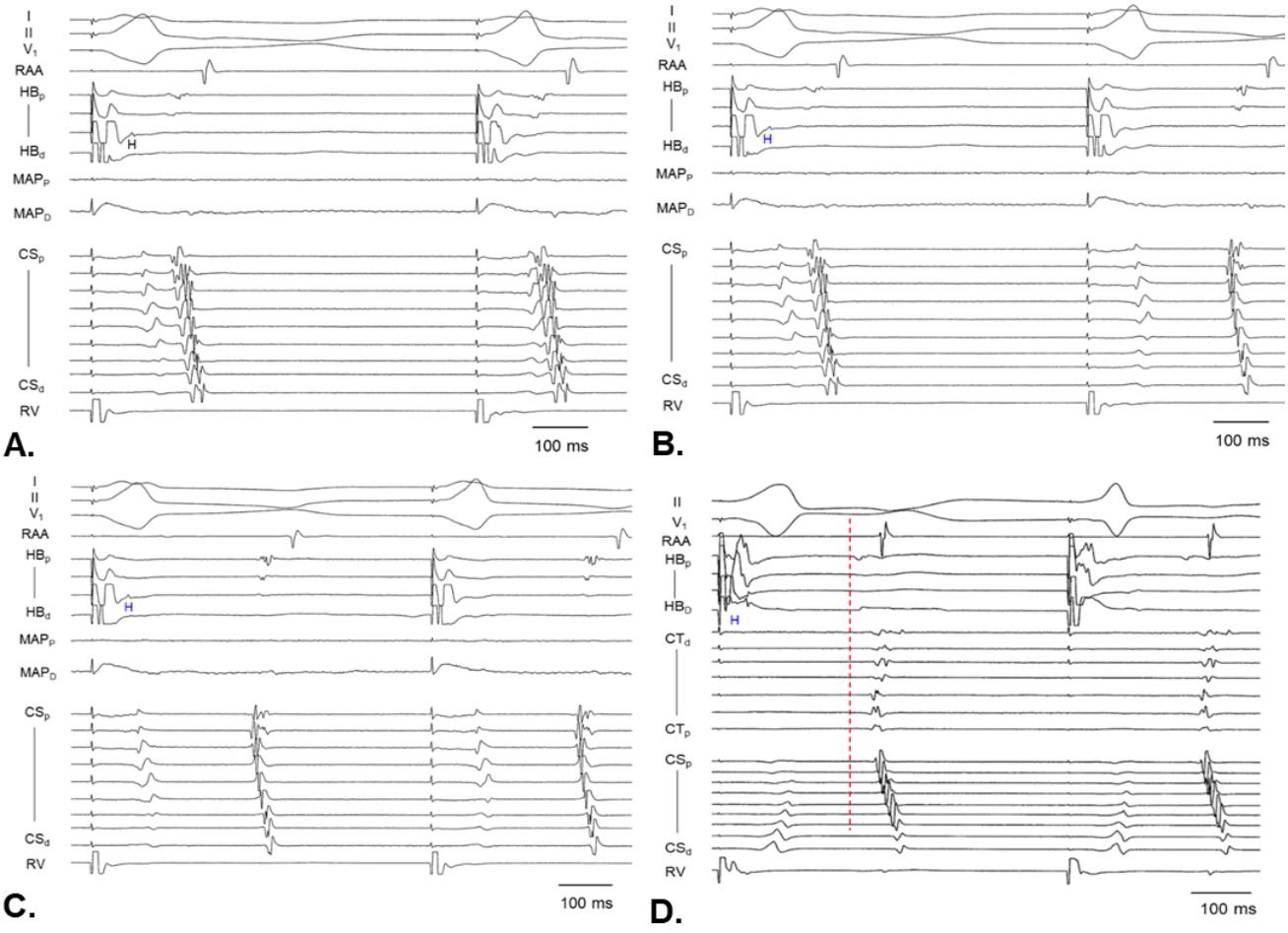


Figure 1.12. Decremental RV pacing exposed different pathways of retrograde conduction. A. RV pacing at 660 ms revealed retrograde AVN conduction. **B.** At pacing CL of 650 ms, retrograde conduction changed suddenly. **C.** Parahisian pacing at 650 ms showed retrograde slow pathway conduction. **D.** In another patient, retrograde conduction was mediated by the fast pathway but stimulus-atrium interval was >200 ms. Therefore, one cannot use the stimulus-atrial interval to judge if retrograde conduction is mediated by AP, fast pathway or slow pathway. Vertical red line: timing of earliest atrial activation.

3. RA-CS differential pacing to identify any AP that conducts in the antegrade direction by comparing the ventricular activation sequence and QRS morphology of the 12-lead ECG (**Figure 1.13A**). The ideal CS pacing site is approximately the 4 o'clock position along the mitral annulus. Pacing from here ensures the wave front engages the atrial end of a left free wall AP quickly. For a left free wall AP, differential pacing from proximal and distal CS can help determine the direction of the oblique course of the AP and help choose a good pacing site for mapping and ablation (see **Chapter 5** for detail).

4. Decremental RAA pacing to assess AVN and AP function. Atrio-ventricular AP, atrio-fascicular AP and fascicular-ventricular AP can easily be differentiated by the responses of AH and HV intervals to decremental atrial pacing (see **Chapter 5** for detail).

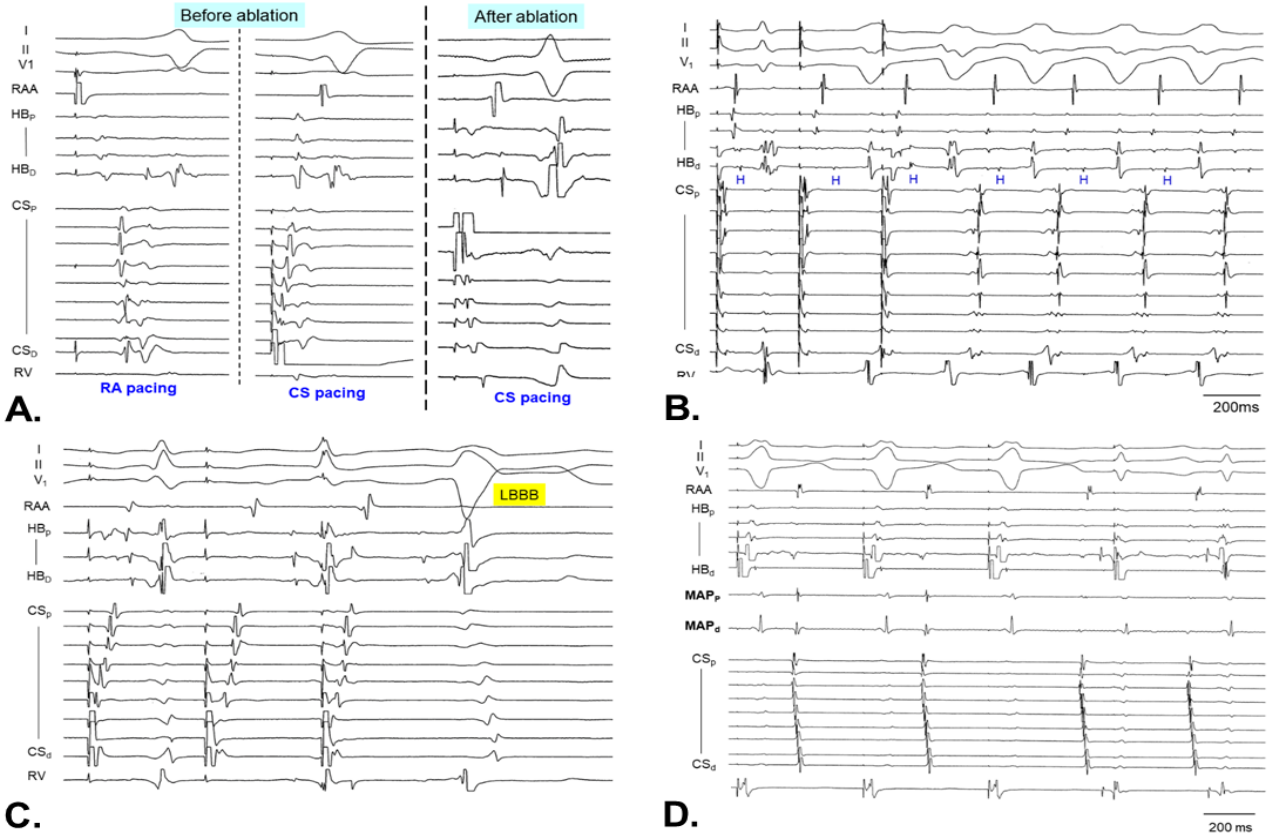


Figure 1.13. Posterolateral CS pacing to identify antegrade AP conduction of a left free wall AP. A. Before ablation, RA (left panel) and CS (middle panel) pacing showed identical ventricular activation sequence and preexcitation pattern (12-lead ECG not shown here). This observation indicates that there is only one AP exhibiting antegrade conduction. After ablation, CS pacing (right panel) produced a long local AV interval at the site of the AP, indicating the absence of antegrade AP conduction. **B.** Induction of AVRT by pacing near the AP and inducing LBBB. **C.** After a successful ablation, rapid CS pacing induced a long AV interval and LBBB but failed to induce an AP echo beat or AVRT. **D.** Induction of fast/slow AVNRT by RV pacing. Note that the third paced beat caused retrograde conduction to change from the fast pathway to slow pathway and initiated fast/slow AVNRT.

5. Induction of tachycardia:

   (a) Before tachycardia induction, Dr. Jackman ensures a stable position of the HB catheter that records a clear HB potential and atrial potential. Disappearance of the HB potential during tachycardia excludes the diagnosis of SVT with aberrancy and favors diagnoses such as VT, antidromic AVRT or atrio-fascicular AVRT (Mahaim).

(b) In Dr. Jackman's practice, the most effective pacing maneuver to induce SVT is to deliver short runs (2-4 beats) of rapid atrial pacing slightly shorter than the Wenckebach CL. Pacing is immediately stopped when AV or AH interval is substantially prolonged which often brings out AVN or AP echo beats or tachycardia.

- For AVRT induction, rapid atrial pacing is most effective to be delivered closer to the location of the AP (e.g. pacing distal CS for a left anterior AP). The atrial tissue near the atrial end of the AP is activated first by pacing and therefore will recover first and be ready for retrograde AP conduction to initiate AVRT**.**

- This pacing maneuver is most effective if LBBB is induced for a left free wall AP or RBBB for a right free wall AP. In these cases, the presence of LBBB or RBBB further delays the ventricular activation to engage the ventricular end of the AP and allows more time for the atrial pacing site near the AP to recover from refractoriness (**Fig. 1.13B**).

(c) AVNRT or AVRT that requires a very long AH or AV interval to initiate or maintain may require atrial extra-stimulation with S1S2 coupling interval slightly longer than the slow pathway ERP or AP ERP. The S1S2 method is particularly helpful if AVNRT requires a very long AH interval to induce.

(d) Induction of SVT by ventricular pacing is usually not Dr. Jackman's first choice, except for (1) fast/slow or slow/slow AVNRT (**Figure 1.13D**) and (2) atrio-fascicular AP (Mahaim AP). Two prerequisites for reentrant tachycardia are unidirectional block and slow conduction, which are inherently present in atrio-fascicular APs (**Figure 1.14**).

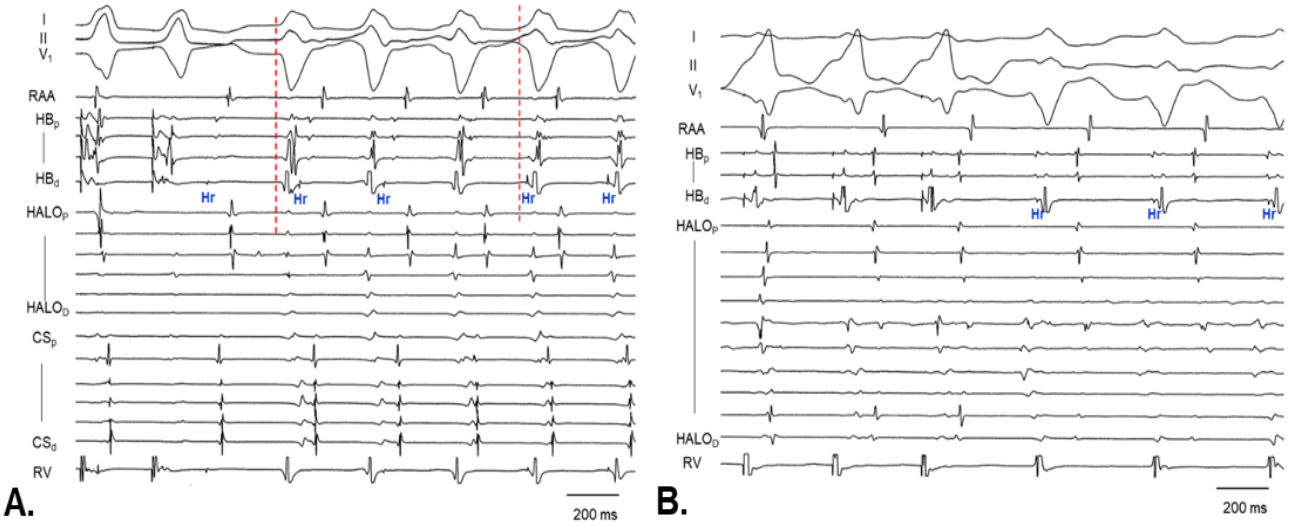


Figure 1.14. Induction of atrio-fascicular (Mahaim) AVRT by RV pacing. A. Note that the retrograde HB/RBB potential (Hr) of the first two beats of tachycardia was significantly delayed due to RBBB. After RBBB resolved, the HB/RBB potential was 20 ms after the onset of the QRS complex and the VA interval shortened accordingly. **B.** Another example of Mahaim AVRT easily induced by straight RV pacing. Vertical red lines: beginning of the QRS complex.

Using single ventricular extra-stimulation to induce SVT:

i. Whether the ventricular wave front enters the AP or AVN is often dependent on the refractory period of the Purkinje system.

ii. In the presence of a longer S1-S1 interval, S2 often induces retrograde His-Purkinje block, allowing the S2 ventricular wave front to conduct into the AP. Given the AVN

was not invaded by S2, it is ready for antegrade conduction and thus, the initiation of orthodromic AVRT.

iii. Shorter S1-S1 CL may shorten the refractoriness of the retrograde Purkinje conduction, facilitating the S2 ventricular wave front to conduct to the AVN and initiate AVNRT. Some of the slow/slow or fast/slow AVNRT has to be induced by ventricular pacing.

(e) To induce typical right atrial flutter, the easiest maneuver is to deliver rapid pacing from the proximal CS and stop pacing immediately when unilateral conduction block (septal to later) is observed (**Figure 1.15).**

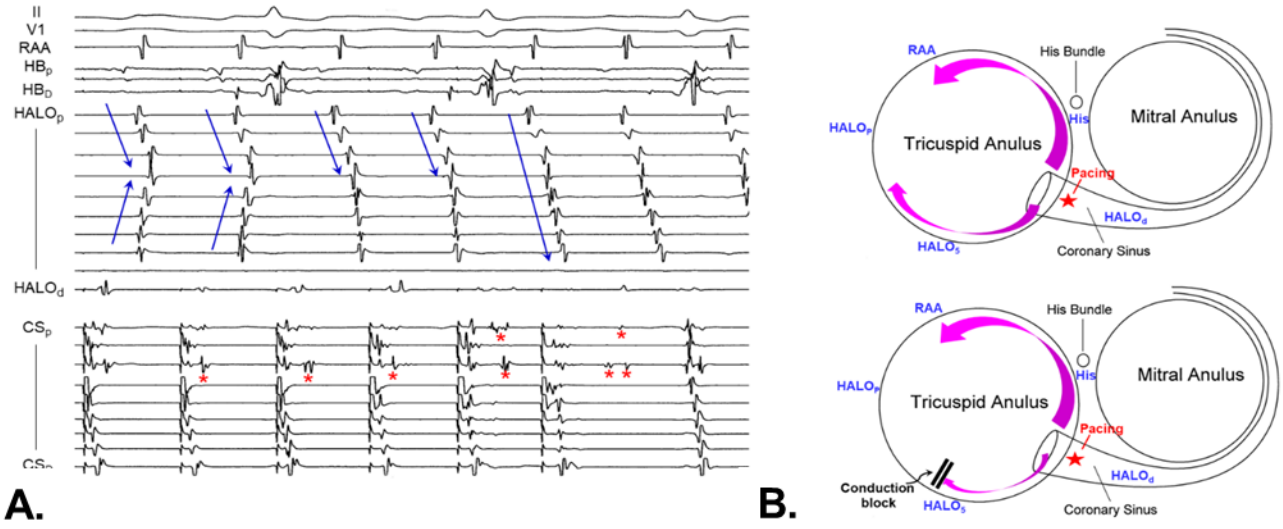


Figure 1.15. Induction of typical right atrial flutter by burst pacing from the CS. A. The HALO catheter was positioned along the tricuspid annulus. Conduction around the annulus (blue arrows) in both clockwise and counterclockwise directions was noted in the first two beats. The fifth beat showed unidirectional conduction block, initiating counterclockwise atrial flutter. Asterisks indicate CS muscle potentials that were dissociated from the atrial potentials during CS pacing. This observation explains why an atrial tachycardia shown in **Figure 1.4B** can be misdiagnosed as AF. **B.** Schematic illustration of conduction (**top panel**) and conduction block (**bottom panel**) across the cavo-tricuspid isthmus.

6. To determine the mechanism of a narrow complex tachycardias, single, double or triple ventricular extra-stimuli are delivered to the ventricular site adjacent to the site of earliest atrial activation (see **Chapter 4** for detail).

7. To determine the mechanism of a wide complex tachycardia with 1:1 VA relationship, single atrial extra-stimuli are delivered to the atrial site adjacent to the site of earliest ventricular activation (see **Chapter 4** for detail).

8. To map and ablate an AP over antegrade conduction, the foremost thing is to choose a good pacing site that produces the longest AV interval and to find a stable *ventricular* EGM to serve as a timing reference (see **Chapter 5 and 6** for detail).

9. To map and ablate an AP over retrograde conduction, the foremost thing is to choose a good pacing site that produces the longest VA interval and to find a stable *atrial* EGM to serve as a timing reference (see **Chapter 5 and 6** for detail).

10. For AVNRT, focused mapping of the fast and slow pathway area will determine if the tachycardia is slow/fast, slow/slow or fast/slow AVNRT. For slow/slow and fast/slow AVNRT, mapping of the retrograde slow pathway is performed first. Then, the operator can decide to ablate the retrograde slow pathway first or anatomically ablate the antegrade slow pathway first.

11. For focal atrial tachycardia or PVCs, activation mapping heavily relies on the morphology (QS pattern) of the minimally filtered unipolar EGM recorded on the distal electrode of the ablation catheter.

12. For reentrant tachycardias, electro-anatomical mapping is used to delineate the reentrant circuit before attempting to entrain the tachycardia to avoid premature termination of the tachycardia.

Figure 1.16 illustrates the closely-spaced diagnostic catheters routinely used in the OU-EP laboratory. Note that the RA catheter has 6 electrodes. The 5th and 6th electrodes serve as the unipolar reference electrodes in the inferior vena cava. A good unipolar reference is an electrode that is distant from the heart (no far-field potential) but is inside the body so that the impedance changes on the skin will not affect the quality of the reference electrode. This reference electrode in the inferior vena cava works much better than the Wilson's central terminal.

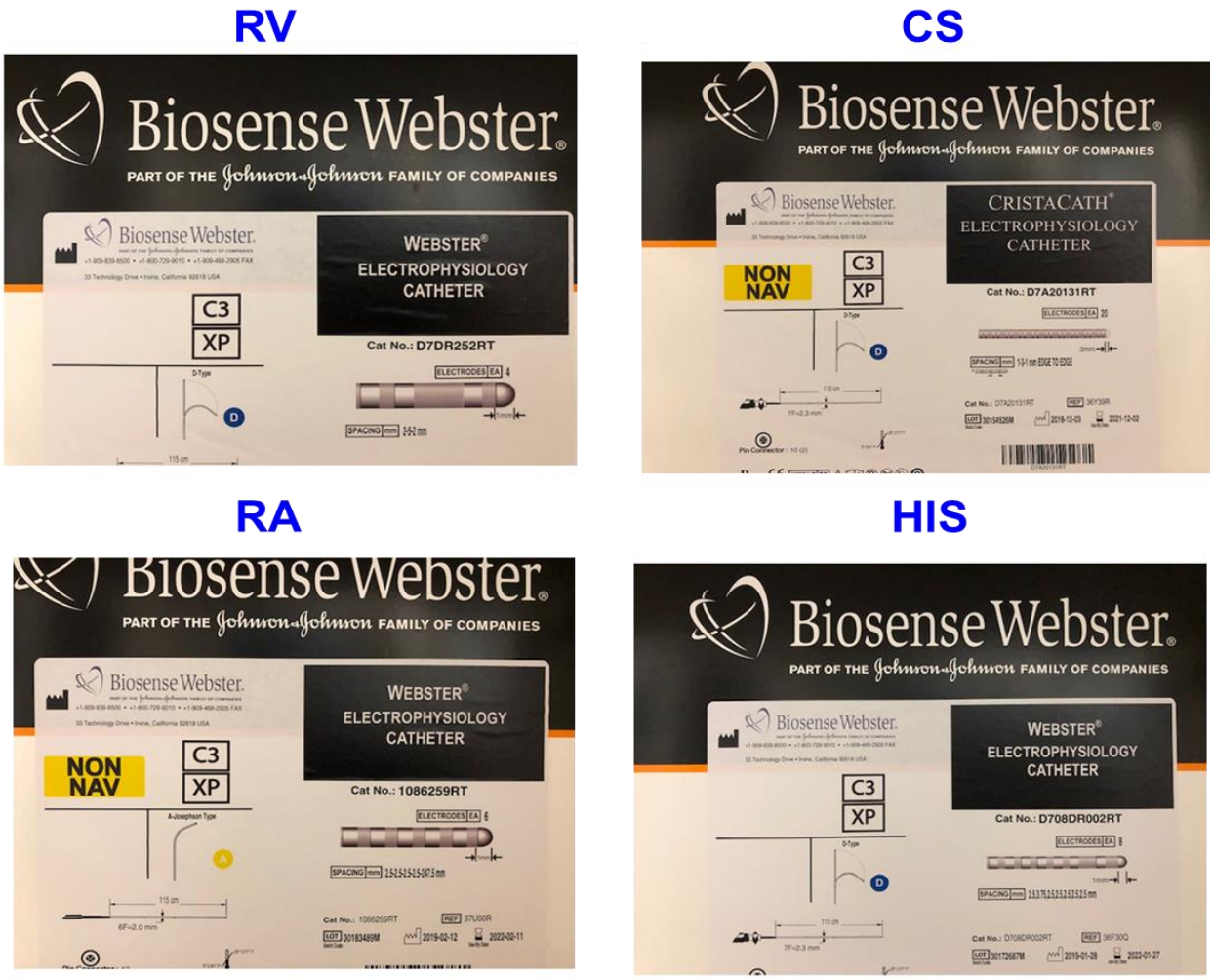


Figure 1.16. Diagnostic catheters used in the OU-EP laboratory. Note that the RA catheter has 6 electrodes. The 5th and 6th electrodes serve as the unipolar reference electrodes in the inferior vena cava. They are 25 cm distant from the 4th electrode.

Chapter 2:
Signal Processing and Noise Reduction in the EP Laboratory

The author wants to extend his gratitude to Professor John Dyer (University of Oklahoma, Department of Electrical Engineering) and Mr. Thomas Hayes (clinical engineer of the OU-EP laboratory) for their support. Some of the figures in this chapter are provided by Dr. Dyer and Mr. Hayes.

Despite the field being called electrophysiology, many of us received little or no education on how cardiac electrical signals are acquired and processed. Troubleshooting the source of noise on surface ECG or intracardiac electrograms (EGM) is therefore a daunting and time consuming task. This chapter focuses on the practical knowledge about signal acquisition and signal processing in hope that it helps electrophysiologists troubleshoot the noise in EP laboratories.

Bipolar and Unipolar Recording

All EP recording systems record the voltage generated by myocardium. Voltage and current are tightly coupled; we cannot get one without the other. Electrical excitation of myocardium and neural tissue is modeled as electric charges moving through an electric field; thus, we get current (charge moving) and voltage (the electric potential of moving a charge through an electric field). The tissue is the resistive medium (impedance). Depolarization of tissue as it sweeps across the myocardium can be modeled as a *moving dipole*. A *dipole* consists of two equal and opposite charges, $+q$ and $-q$, spaced some distance apart (**Figure 2.1A**). As the myocardium depolarizes, a wave front that has a positive potential on its leading edge (depolarization wave front), and negative on its trailing edge (repolarization wave front), sweeps across the tissue.

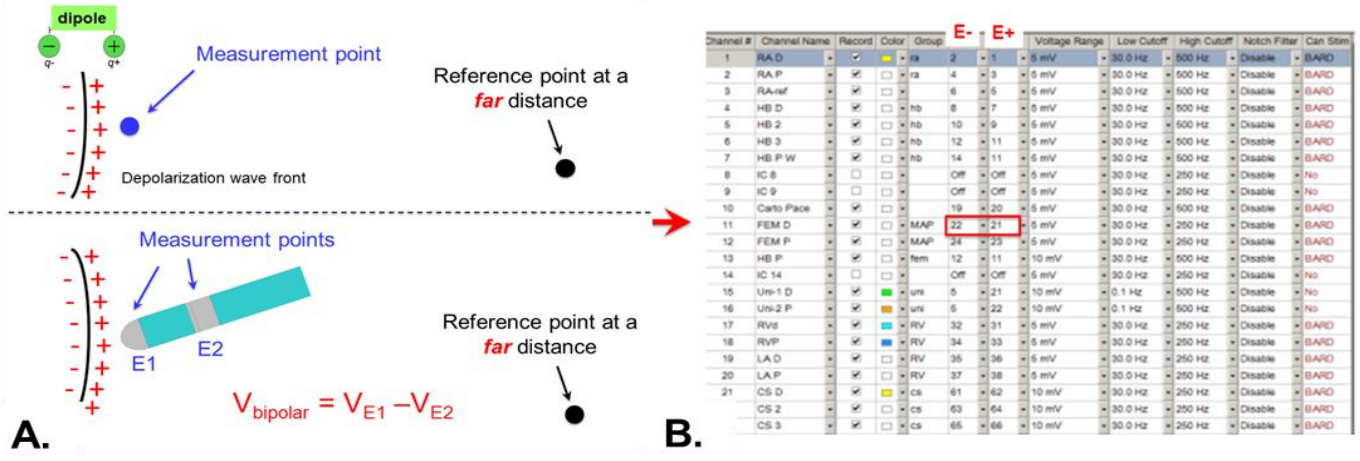


Figure 2-1. A. Top: unipolar measurement. The unipolar measurement is measured with respect to a point at a far distance. **Bottom.** A bipolar measurement measures the difference between the two electrodes. E1 and E2: distal and proximal electrode, respectively. **B.** Configuration of the electrodes in the Bard recording system. The mapping catheter electrodes were assigned to 21, 22 (distal pair) and 23, 34 (proximal pair). By convention, the positive electrode is assigned to the distal electrode (electrode 21 and 23 in each pair).

The electrical potential of a dipole is measured with respect to something. Generally, a reference point is chosen at a far a distance. This type of measurement is called a *unipolar* measurement (**Figure 2.1A**). A *bipolar* measurement measures the difference between the two electrodes. Conventionally, it is measured by subtracting the voltage of one electrode (negative) from the other electrode (positive). By convention, the positive electrode is assigned to a smaller number of the electrode pair (**Figure 2.1B**). For example, if the

distal electrode pair of an ablation catheter is assigned to pin 21 and 22 in the EP recording system, the tip electrode should be assigned to pin 21 (positive); the ring electrode (negative), 22. The EGM recorded by electrode 22 will then be subtracted from that of electrode 21 to get the bipolar EGM. If the tip electrode is assigned to pin 22, the polarity of the EGM will be reversed but the relative timing of each component of the EGM will not change (**Figure 2.2**). Reversing the polarity in this way has little influence on interpreting EGMs recorded by diagnostic catheters. It has a profound impact on localization of a focal source of arrhythmia if operators mistakenly connect the unipolar reference electrode to the positive input and the tip electrode of the ablation catheter to the negative input. In this way, the distal unipolar EGM is derived from (reference - tip). If the operator uses the QS morphology of the unipolar EGM to select an ablation target, at the site of the focal arrhythmia, it will exhibit an R, not a QS morphology, misleading the operator that this site is not the origin of the focal arrhythmia **(Figure 2.2B**). The OU-EP laboratory is not immune from this type of mistake. Throughout this book, readers may find some examples that the distal electrode pair of the ablation catheter was connected backwards (e.g. the distal electrode assigned to negative input and proximal electrode to positive input, respectively) (**Figure 9.1A**).

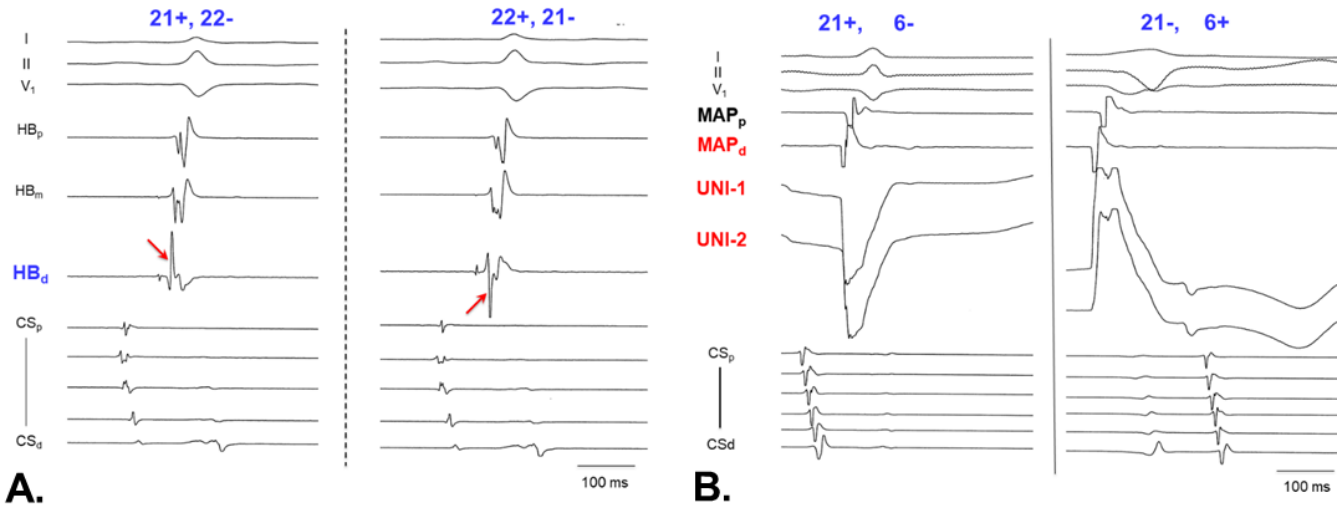


Figure 2.2. Reversal of polarity of bipolar recording. A. Left panel: HBd was connected the correct way: electrode-21 assigned as positive, electrode-22 as negative. **Right panel**: HBd was connected as follows: electrode-22 assigned as positive, electrode-21 as negative. Although the morphology of the EGM was reversed, local ventricular activation measured by the first steep deflection (red arrow) was not affected. **B. Left panel.** The distal electrode (electrode-21) of the mapping catheter was assigned as positive and the reference electrode (electrode-6) was assigned as negative. The distal unipolar EGM (UNI-1) exhibited a "QS" pattern when the tip electrode was positioned at the origin of the PVC. **Right panel**. The distal electrode (electrode-21) was assigned as negative and the reference electrode (electrode-6) was assigned as positive. The distal unipolar EGM (UNI-1) exhibited an "RsR" pattern, misleading the operator that this site was not the origin of the PVC.

Figure 2.3 and **Figure 2.4** illustrate simulated wave front propagation and EGMs recorded on a pair of electrodes. If the wave front is generated by the tissue directly beneath the recording electrode, both unipolar and bipolar EGM should begin simultaneously. The local unipolar EGM should begin with a steeply negative deflection (qs or QS pattern**; Figure 2.3F**). Of note, simulation was based on the assumption that the electrode pair is laid in parallel with the direction of the wave front and the size of the two electrodes is similar. These two assumptions have a great impact on the morphology of the EGM (see discussion later). **Figure 2.4A** shows a tip electrode significantly larger than the ring electrode. Since it takes 2-3x more time to pass the larger tip electrode, the EGM recorded on the tip electrode now is wider and rounded. Because the ring electrode is smaller, the sharp component of the *bipolar* EGM is more likely to be recorded on the smaller ring electrode. This feature has important clinical implications. The larger the size of the electrode, the larger the recording range (**Figure 2.4B**). If an operator uses an 8-mm tip ablation catheter to map an arrhythmia, the bipolar EGM is almost always rounded. If a sharp EGM appears on the bipolar EGM, it is most likely recorded on the ring electrode, not the tip electrode. If the sharp *bipolar* EGM is the ablation target (e.g. an accessory pathway potential), ablation at that site may not be successful. For this reason, the OU-EP

group abandoned the 8mm-tip electrode catheter as soon as saline-irrigated ablation catheters (3.5mm tip) became available.

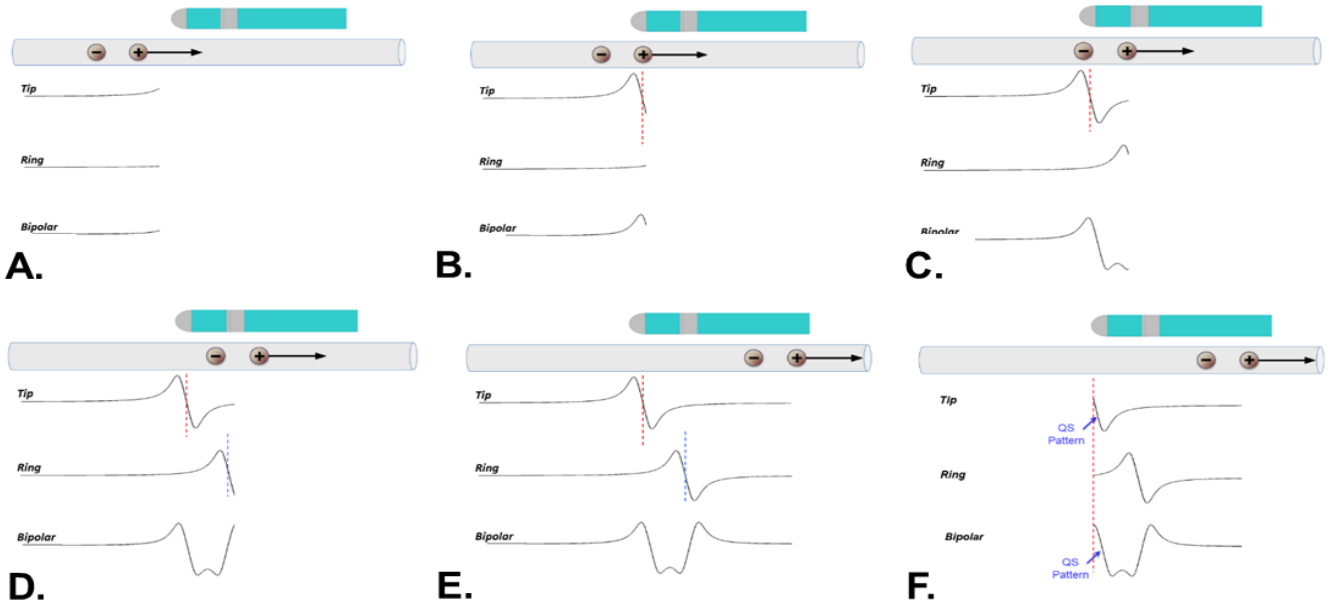


Figure 2.3. Simulated wave front propagation as well as unipolar and bipolar EGM. A-E. If wave front propagates toward a recording electrode, the electrode records an EGM beginning with a positive deflection; if wave front propagates away from the electrode, the electrode records an EGM beginning with a negative deflection. When the wave front propagates to the tissue directly beneath the recording electrode, EGM changes quickly from a positive to a negative deflection, creating a steeply negative deflection (vertical red and blue lines) which represent activation of the tissue directly beneath the recording electrode. **F.** If the wave front is generated by the tissue directly beneath the recording electrode, both unipolar and bipolar EGM should begin simultaneously (vertical red line). The local unipolar EGM should begin with a steeply negative deflection (blue arrow) and generates a QS or qs pattern.

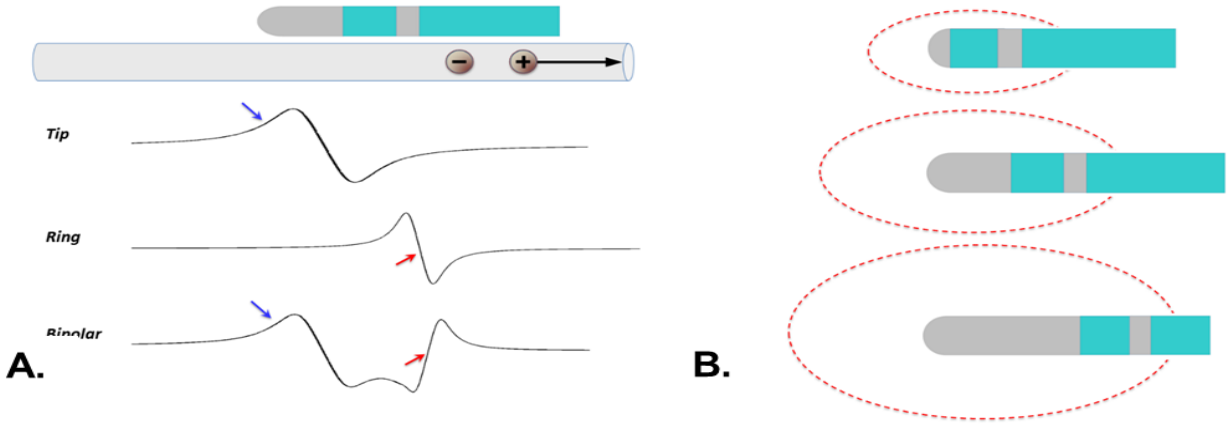


Figure 2.4. Effects of electrode size on EGM. A. If the tip electrode is significantly larger than the ring electrode, the unipolar EGM recorded by the tip electrode will be more rounded and wider. The onset of EGM will be slow (blue arrow). If there is a sharp component (red arrow) in the bipolar EGM, it is more likely recorded by the ring electrode, not the tip electrode because the ring electrode is smaller. **B.** Correlation between the electrode size and recording range.

Electrode and Wave Front Orientation

The orientation between the recording electrode and direction of the depolarization wave front has a significant impact on the morphology of the EGM. When the recording electrode pair is not aligned with the direction of the wave front (e.g. a 45° angle), the EGM has a smaller peak and smoother appearance (**Figure 2.5A**). **Figure 2.5B** depicts different EGM morphologies if the operator positions the catheter in an angle that

is 180 degrees different but the tip electrode still touches the same site. The morphology of the distal *unipolar* EGMs remain unchanged but the timing of each unipolar EGM is different. The morphology of the *bipolar* EGM is therefore different when the electrode orientation changes 180 degrees. This explains a common scenario that the operator noted an interesting EGM and annotated it on the electro-anatomic mapping system. Later, when the tip electrode of the ablation catheter is returned to the same location, the EGM looks different due to a difference in catheter orientation.

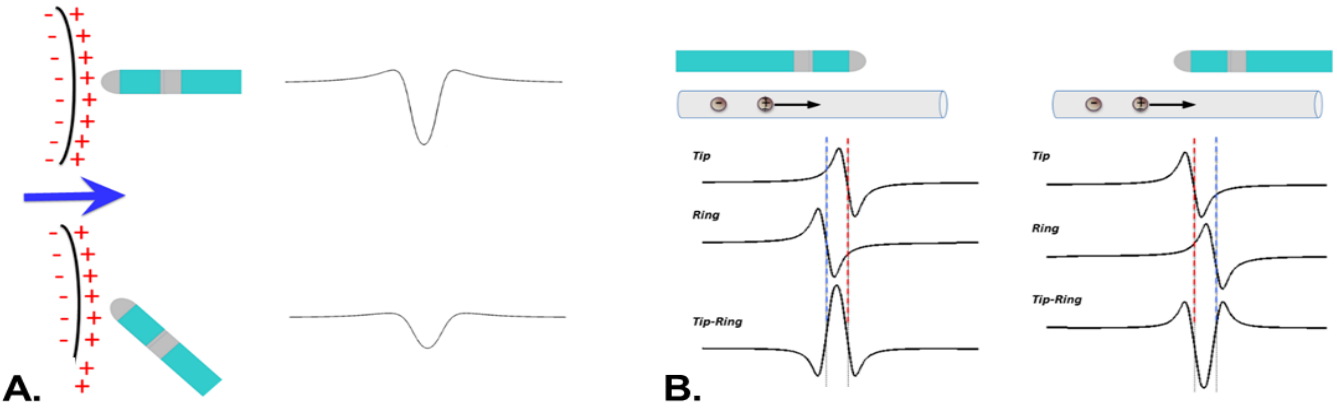


Figure 2.5. Effects of electrode-wavefront orientation on EGM. A. When the electrode-wave front orientation is 45 degrees, the bipolar EGM is expected to have a smoother appearance and smaller peak. **B.** If the electrode pair is positioned in the opposite direction (180-degree off) but the tip electrode remains at the same site, the morphology of distal unipolar EGM will remain the same. However, the morphology of the bipolar EGM will be different. Red and blue dotted line indicate local activation recorded by the tip and ring electrode, respectively.

Figure 2.6 illustrates wave front propagation and EGMs recorded on a pair of electrodes oriented perpendicular to the wave front. Note that the proximal electrode (ring electrode) is distant from the wave front, thereby recording an EGM with a lower voltage amplitude as well as lower frequency (rounded EGM). If the distance between the tip and ring electrode increases progressively, the contribution from the ring electrode to the bipolar EGM becomes progressively smaller. Essentially, nearly the entire bipolar EGM is derived from the tip electrode if the inter-electrode distance is large. The ring electrode of the distal pair of a mapping catheter is smaller than the tip electrode and is expected to record a sharp EGM if the ring electrode is touching the tissue. If the EGM of the ring electrode shows smooth appearance with a small amplitude, one may presume that the distal electrode pair is not oriented in parallel with the target tissue unless both electrodes are touching a scar where all sites show low amplitude EGMs.

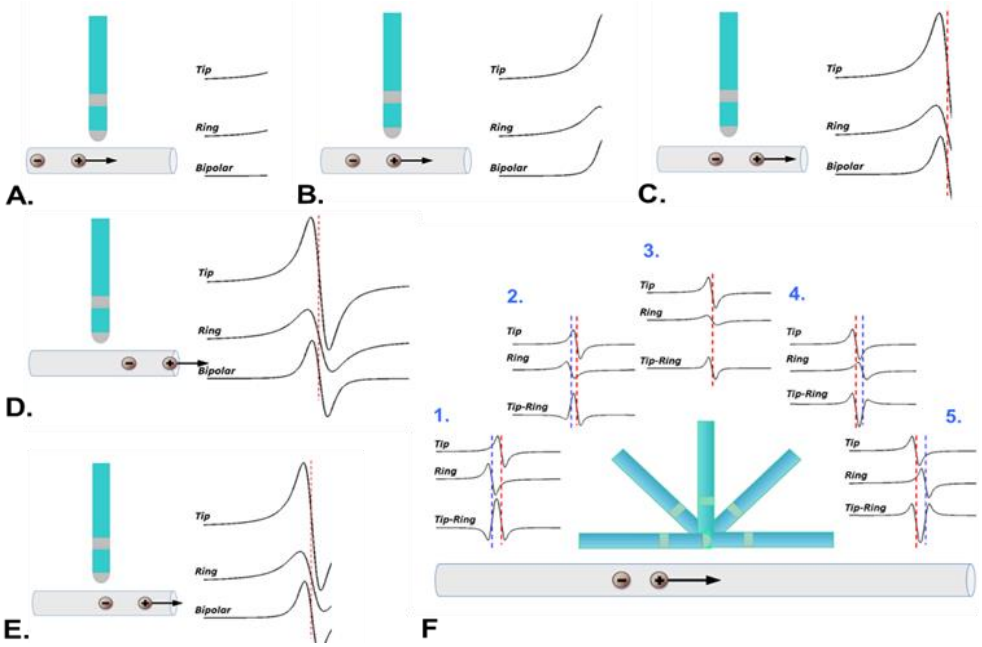


Figure 2.6. A-E. Relatively small contribution from the ring electrode to the bipolar EGM when the electrode pair is oriented perpendicular to the wave front. If the inter-electrode distance is large, the contribution from the ring electrode to the bipolar EGM is even smaller. **F.** Five different orientations. Blue and red vertical line: local activation of the distal and proximal electrode, respectively

Figure 2.6F shows the unipolar and bipolar EGM in different orientations between the bipolar electrodes and depolarization wave front. In orientation-3, the ring electrode records slow, rounded and low amplitude EGM; most of the bipolar EGM is contributed by the tip electrode. In practice, orientation-2, orientation-3 and orientation-4 are the most common orientations during mapping or ablation. Because the tip electrode is longer than the ring electrode (e.g. 3.5 or 4 mm vs. 1-2 mm), operators need to be aware that a large tip electrode makes the EGM more rounded and wider but the ring electrode in the blood pool also makes the EGM more rounded and wider. Therefore, in orientation-2 and orientation-4, the signal of interest displayed on the bipolar EGM can come from either the ring or the tip electrode. This underscores the importance of displaying *both* proximal and distal unipolar EGM during mapping to accurately localize the origin of the EGM of interest. The author has seen many EP laboratories that only display the distal unipolar EGM of the ablation catheter. This practice should be avoided because displaying the proximal unipolar EGM costs nothing but provides important information complementary to the EGM recorded on the tip electrode.

An extreme example of the impact of electrode-wave front orientation on EGM is when the wave front moves in a path perpendicular to the orientation of the recording electrodes and the band of tissue is narrow enough that it passes between the electrodes. In this extreme (i.e. when maximal symmetry exists), the measured amplitude of both the tip and ring electrodes will be the same. This implies that the bipolar recording will be 0! (**Figure 2.7A**).

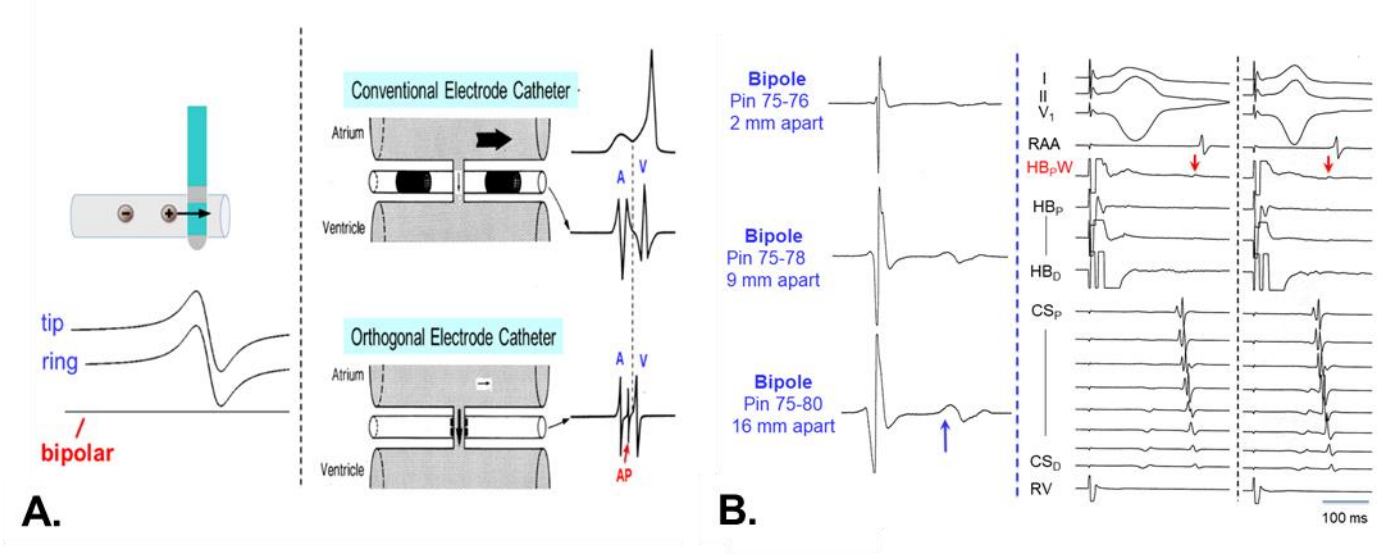


Figure 2.7. A. **Left panel.** An extreme example that the wave front passes through the middle of two electrodes. In theory, the bipolar EGM will not record any signal. **Right panel.** Conventional vs. orthogonal electrode catheters. If an accessory pathway traverses the annulus perpendicular to the plane of the annulus, an orthogonal catheter has a better chance of recording an isolated AP potential. **B. Effect of electrode spacing on EGM. Left panel.** Note that with progressively larger inter-electrode spacing, the EGM becomes progressively larger. Far-field potentials (blue arrow) become more prominent. **Right panel.** During parahisian pacing, HB-W was created by connecting electrode 14 and 11 as a widely-spaced electrode pair to record far-field atrial EGM (red arrows) in the anteroseptal area. However, this type of widely-spaced electrodes is not suitable for mapping or localizing the location of the AVN.

Historic Vignette:

*Before Dr. Jackman figured out that most accessory pathways (AP) do not traverse the AV annulus perpendicular to the plane of the AV annulus, he thought that the reason why AP potentials are difficult to record is because the wave front of AP conduction is perpendicular to the recording electrodes on the CS catheter. He designed orthogonal CS catheters for this specific purpose but still had difficulty in recording AP potentials (**Figure 2.7A**). Eventually, Dr. Jackman figured out that AP potentials are often*

*obscured by local atrial and ventricular potential. AP potentials can be exposed by changing the pacing site (see **Chapter 5** for detail).*

Electrode size and spacing

The spacing of the bipolar electrodes is very important. For a unipolar measurement, the unipolar EGM is derived from subtracting the signal recorded on the reference electrode distant from the signal on the recording electrode. Basically, unipolar recording has a very large recording range and picks up all kinds of far-field signals. A bipolar measurement, in sharp contrast, measures the difference between the two electrodes, thereby eliminating *most but not all* of the far-field signals. The closer the spacing between the electrode pair, the higher the likelihood that both electrodes "see" the same far-field signals, thereby cancelling them out. The bipolar EGM therefore reflects the electrical activity of the tissue beneath this electrode pair. When the spacing is increased, common-mode rejection does not work as well. The bipolar EGM is contaminated with more far-field signals, leading to EGMs with a larger amplitude, more rounded morphology and longer duration (**Figure 2.7B**). For this specific reason, all the catheters used in the OU-EP laboratory are closely-spaced electrode catheters. Using the Biosense PentaRay catheter as an example, the OU-EP group prefers the 2-6-2 mm spacing to 4-4-4 mm spacing. Another problem associated with far-field potential contamination is that the tip electrode of an ablation catheter is larger than the ring electrode. The two electrodes therefore "see" different far-field potentials, which cannot be eliminated by common mode rejection, underscoring the importance of displaying *both* unipolar EGMs.

The beneficial effects of small electrode size and short inter-electrode spacing are demonstrated by using the PentaRay catheter (electrode size: 1 mm, 2 mm spacing) to map the arrhythmogenic channels in infarct scars. Bipolar EGM recorded by the PentaRay catheter reflects a small area of cardiac tissue beneath the bipole. Because the electrode size of the PentaRay catheter is only 1 mm, it takes the wave front less time to pass through the smaller recording range, creating a sharp EGM. On the contrary, the tip of the mapping catheter is 3.5-4 mm; EGM recorded by a mapping catheter is wider and more rounded because it takes the wave front more time to pass the larger recording range of the electrode. In addition, the orientation of the PentaRay bipolar electrodes is almost always in parallel with the plane of the cardiac tissue (orientation-1 or orientation-5 in **Figure 2.6F**), different from the most common orientation (orientation-2, 3, and 4) of an ablation catheter. The differences between a PentaRay catheter and a mapping catheter may account for sharper local abnormal ventricular activity (LAVA) in infarct scars recorded on the PentaRay catheter.

While closely-spaced electrode catheters have a great advantage of eliminating far-field signals, it requires good contact with the tissue to record a good EGM. In the OU-EP laboratory, the 8-pole HB catheter has the following design: 2 mm electrode size; 2 mm spacing. Sometimes, the distal two or three pairs of electrode have stable contact with the top of the triangle of Koch, recording a stable HB potential, but the proximal one or two pairs do not touch the atrial tissue well, thereby recording a very small atrial EGM. The small atrial EGM can impose a challenge on differential diagnosis of tachycardia. To solve this problem, the author may use two widely-spaced proximal electrodes to create a new bipole to record a larger, far-field *atrial* EGM for timing purpose. For example, the 8 electrodes are usually assigned to pin 7-8, 9-10, 11-12 and 13-14. The author makes the fifth electrode pair by connecting electrode 11 and 14 to increase the inter-electrode spacing to 10 mm (edge to edge) (**Figure 2.7B**). However, the author would not use this wide-spaced electrode to estimate the location of the AV node.

Signal Formation and Signal Processing

All electrophysiologists know that a signal is made up of many different frequencies. Specifically, frequencies in a signal may be presented as a series of sinusoids, both sine and cosine. The electrical signals that electrophysiologists are interested in are measured in time domain. Understanding signals in frequency domain is important because filtering of signals is processed in frequency domain. Joseph Fourier, while working for Napoleon Bonaparte on heat transfer in cannons, showed mathematically that any signal could

be written as an *infinite* summation of individually weighted sines and/or cosines. In real world, we choose enough *discrete* frequencies to approximate the real signal. Basically, Fourier transform decomposes signals in time domain into infinite numbers of frequencies that make up the signal.

A time-varying signal, $x(t)$, can be written as:

$$x(t) = a_0 + 2a_1\cos(f_1 t + q_1) + 2a_2\cos(f_2 t + q_2) + \cdots + 2a_n\cos(f_n t + q_n) \qquad n \hat{I}\ \mathrm{N}$$

or more concisely

$$= a_0 + 2\mathring{a}_{n=1}^{¥} a_n\cos(f_n t + q_n)$$

- a_0 is the DC term, which describes how the signal is offset from a mean value of 0 (zero)
- Each value, a_1, a_2, etc. is the *weight*, or amount of contribution of each frequency and each term; θ_n is the "phase" of the cosine waveform
- The weights and/or phases may be zero for many frequencies

Figure 2.8 depicts a few examples of how various frequencies make up a signal. Note that the frequency component with a higher amplitude tends to contribute more to the morphology of the signal. This property created a potential problem when scientists hypothesized that the site with the dominant frequency in AF may be the site harboring the AF drivers. The amplitude of the EGM *in AF* from completely normal atrial myocardium is usually significantly lower than the EGM recorded from the same site *in sinus rhythm*. The difference may be much larger where atrial scar is located. If the site driving AF has a high frequency but a very low amplitude, the signal of this site may not stand out as the dominant frequency. Another site with a lower frequency but a higher amplitude may stand out to be the dominant frequency. Ablation targeting the latter is not likely to work.

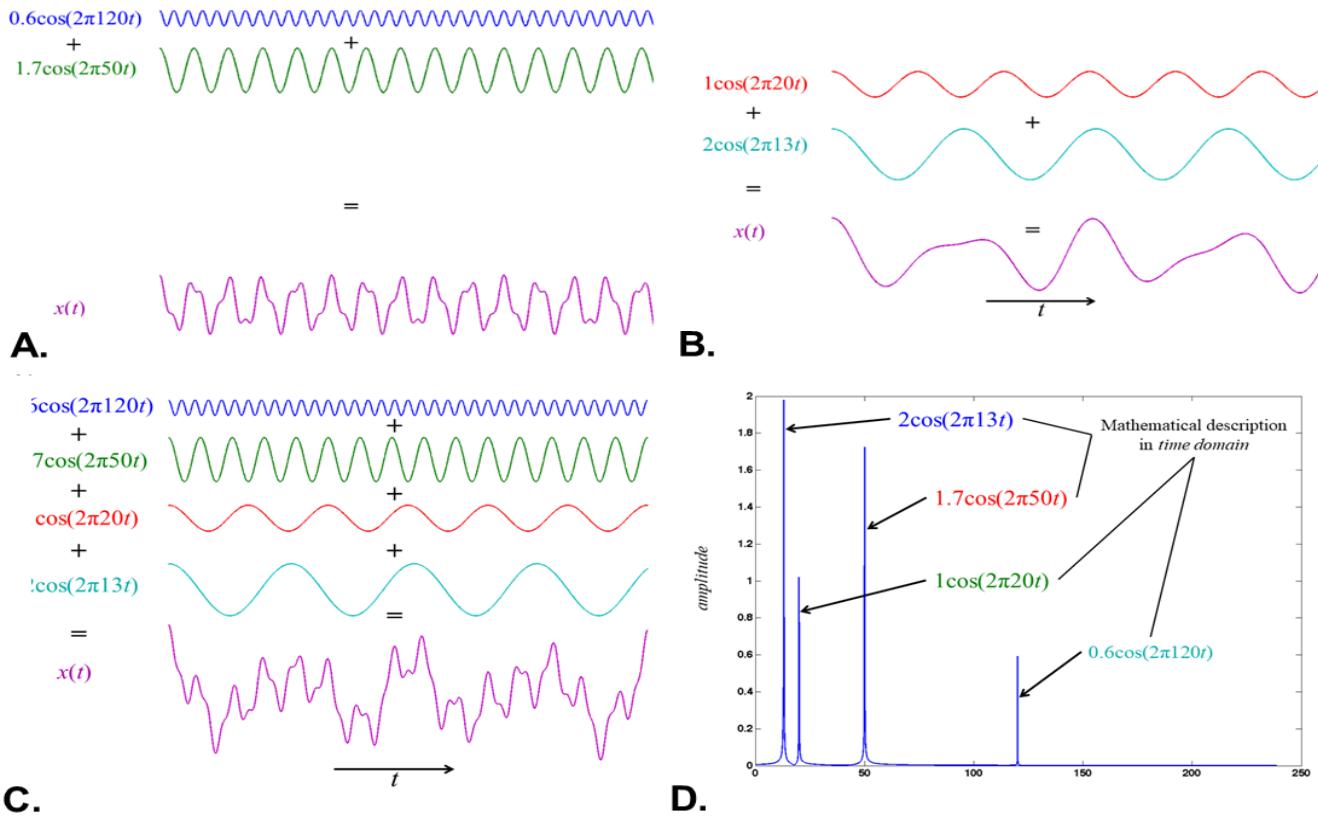


Figure 2.8. A-C. Examples of how signals are reconstructed by components of various frequencies. Note that the component with the largest amplitude appears to determine the shape of the signal. **D.** Signals expressed in amplitude and frequency.

Signal Processing

In signal processing, one can think of a signal as the *measured* or *quantified* realization of a physiological phenomenon, such as heart rate. For many physiological phenomena, the parameter has to be transduced into something easily measured, generally a *voltage.* In electrophysiology, we do not need the transduction step; the signal-of-interest is already a voltage. We electrophysiologists deal with two types of signals: surface ECG signals and intracardiac signals, both of which are minute in amplitude, require substantial amplification and are prone to electric or magnetic interference (**Figure 2.9A**). Acquisition and processing of ECG and intracardiac signals are in general independent but are not completely separated. For example, skin is the organ shared by the circuitry of radiofrequency (RF) ablation and ECG. A loose ECG patch can cause horrendous noise during RF application, obscuring the EGM displayed on the ablation catheter (**Figure 2.9B)**. In addition, a loose patch of a limb lead can introduce noise to the Wilson's central terminal that also serves as the reference point for precordial leads and may be the reference point for intracardiac EGM and unipolar EGM for the ablation catheter. That is, a bad limb lead can introduce noise to surface ECG and intracardiac EGM.

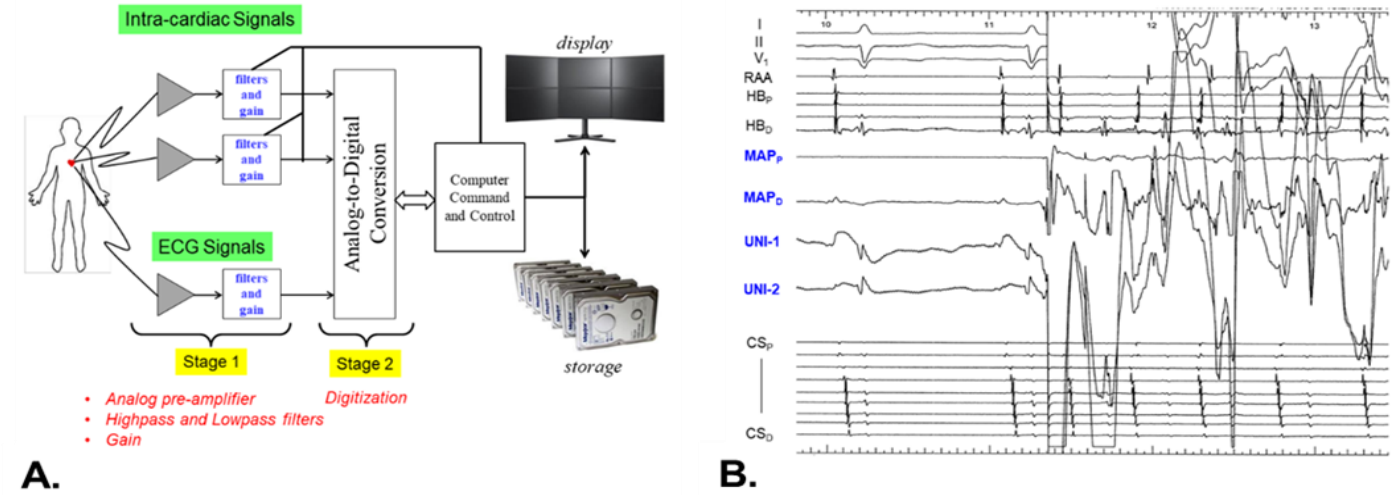


Figure 2.9. A. A simplified diagram of how cardiac signals are processed by going through a series of amplification, filtering, and analog-to-digital conversion. **B.** A bad RF ground pad introduced horrendous noise to both ECG and EGM recorded on the ablation catheter, demonstrating that the surface and intracardiac signals are not independent from each other.

Signal amplification

EP signals are analog signals in the range of 0.02 to 20 mV, requiring substantial amplification. This is usually done by differential amplifiers which do not amplify the signals common to the two inputs, known as common mode rejection. For example, an EP catheter typically has paired, twisted wires. The more turns of twist of the two wires, the more likely that the noise will be rejected by common mode rejection. When the difference between the two wires are amplified, the noise is less likely to be amplified at the same time.

Analog to Digital Conversion

All biological signals e.g. blood pressure and heart rate are analog signals in reference to time. An analog-to-digital converter (ADC) converts a continuous-time and continuous-amplitude analog signal to a discrete-time and discrete-amplitude digital signal. To exactly reproduce the original analog signal, the sampling rate of the ADC needs to be fast enough. This cannot be accomplished unless the sampling rate is higher than twice the highest frequency of the signal based on the Shannon-Nyquist sampling theorem. Because the highest frequency component of EGM rarely exceeds 500 Hz, most, if not all, of the EP recording

systems sample the analog signals at a frequency of 1 kHz or higher. Some of the electro-anatomical mapping systems even sample at 8 kHz.

Filtering

Noise is defined as any part of the signal that is *not* the signal of interest. Sources can be AC power supplies or the circuitry of equipment. Noise may share the same frequency as the signal of interest. The human body is like a large antenna that can pick up many electromagnetic signals in the EP laboratory and introduces noise to the EP recording system. The higher the signal-to-noise ratio, the easier it is to study the signal of interest (**Figure 2.10A**). Unfortunately, many EP signals such as the EGMs in diseased myocardium are very small (<0.1 mV). If the noise level in the EP laboratory is 0.06 mV, the signal-to-noise ratio is <2, leading to difficulty in identifying the signal of interest. Filters, operating in the frequency domain, may get rid of the noise as well as signals of that frequency.

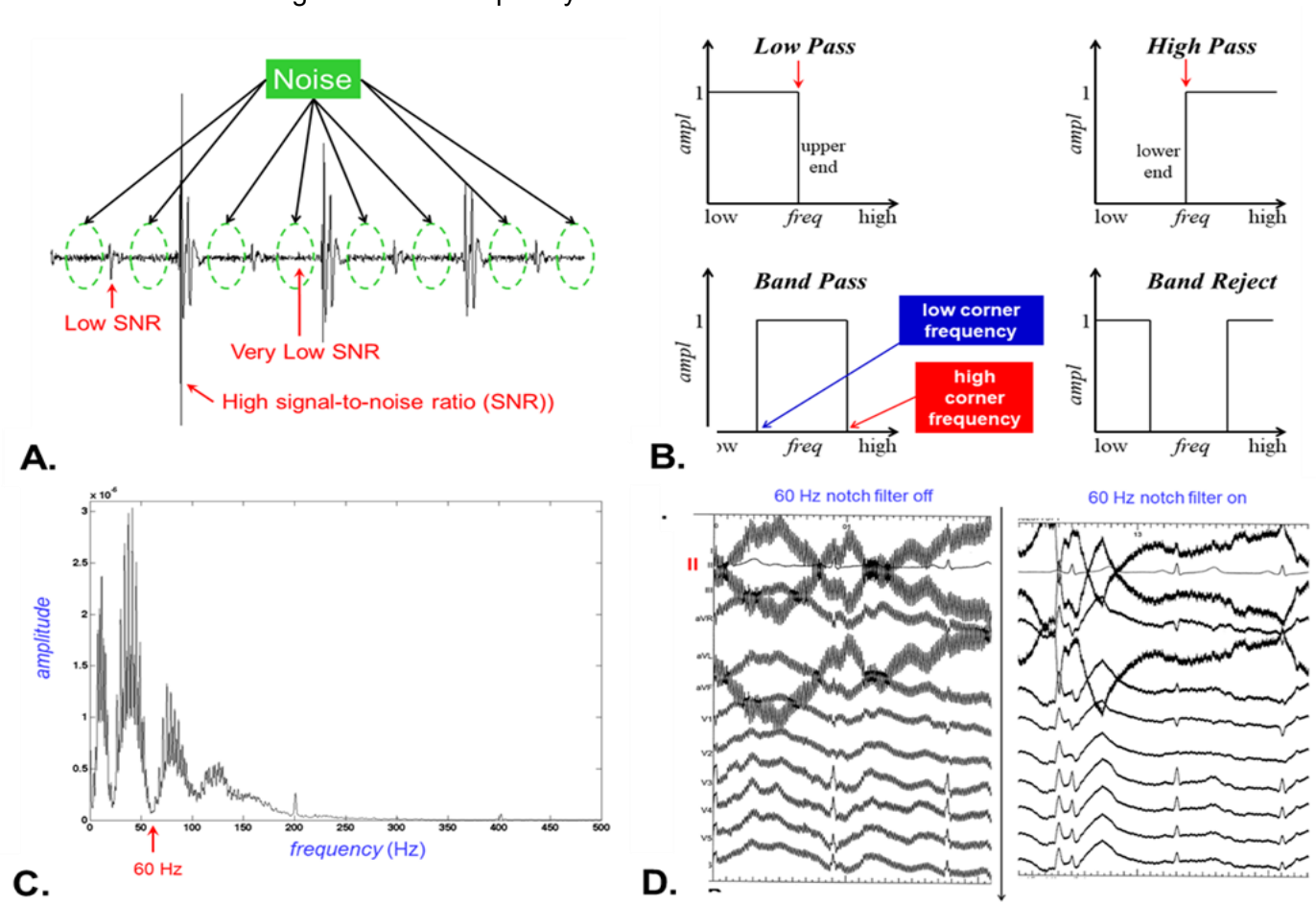


Figure 2.10. Noise and filter. A. Signals of various signal-to-noise ratio (SNR). Signals with very low SNR are most vulnerable to distortion by filtering. **B. Top panel:** High and low corner frequency (red arrows) of low-pass and high-pass filter, respectively. In theory, all the frequencies above the high corner frequency in a low-pass filter are removed. **Lower panel.** Combine a high-pass and low-pass filter, one can create a band-pass filter. **C.** A 60-Hz band-rejection filter (notch filter) rejects all the 60 Hz signals and noise. **D.** When the ECG left arm patch was loose, it introduced horrendous noise to all the 12 leads except lead II (right arm to left leg vector). When the 60-Hz notch filter was turned on, noise diminished but was still very visible.

To deal with electromagnetic noise in the EP laboratory, filtering is the most common solution to eliminating the noise. However, filtering itself will distort the original signals and may introduce new noise (often higher frequency) into the signals. In general, the least filtering is the best filtering. In the past, all the electrical signals from the EP catheters were routed to the recording system first to allow for signal processing. With the rapid advancement of electro-anatomical mapping (EAM) systems, cardiac electrical signals are

often first routed to the patient interface unit (PIU) of the EAM system and then routed to the recording system. That is, the original electrical signals may be subjected to different filter settings in the recording system and EAM system. For example, if the 60-Hz notch filter is turned on in the GE recording system but not in the CARTO system, the same EGM may look different between the two systems. In the OU-EP laboratory, the filter settings are identical between the Bard and CARTO system.

1. Band-pass filter:

In most of the EP recording systems, analog filtering was applied to the signals to allow passage of only the frequencies that are deemed to be present in the signal of interest. In general, all EP recording systems allow electrophysiologists to choose what frequencies to be filtered out. A low-pass filter (e.g. 80 Hz) only allows signals with a frequency <80 Hz to pass through (**Figure 2.10B, top left panel**). A high-pass filter (e.g. 1 Hz) only allows signals with a frequency >1 Hz to pass through (**Figure 2.10B, top right panel**). Such selected frequencies are called corner frequencies. If one puts a high-pass filter and a low-pass filter in series, it creates a band-pass filter which only allows the frequency between 1 and 80 Hz to pass through (**Figure 2.10B, lower left panel**). This is very similar to the frequency spectrum of a radio station. Another type of filter is called band-rejection filter or notch filter which reject certain frequencies (e.g. 50 or 60 Hz). Notch filters are widely used in EP recording to remove the 50 Hz or 60Hz noise from the power supply. Filtering out noise of certain frequency will have very significant impact on the characteristics and relative timing of the signal of interest (see discussion below).

All EP recording and mapping systems allow electrophysiologists to select different band-pass filters and notch filters to maximize the quality of signals of interest. In the environment of an EP laboratory, there are many potentials sources of electromagnetic interferences in a wide range of frequencies. For example, in the USA, cell phones operate at 0.7 to 5.2 GHz while Bluetooth wireless communication operates at 2.4 to 2.5 GHz, the frequencies of which are much higher than that of cardiac electrical signals (mostly <200 Hz). Such high frequency electromagnetic interference can be filtered out safely by a low-pass filter without distorting the cardiac electrical signals. The CARTO-3 system indeed filters out the high frequency noise (kHz and higher) as soon as the signals enter the CARTO-3 patient-interface unit (PIU). This is to remove the electromagnetic noise introduced by the electromagnetic sensor of CARTO-3 itself as well the high-frequency environmental noises.

While band-pass filters can remove certain frequencies in the electrical signal that are potentially important, the signal quality is usually not affected too much if the corner frequencies are chosen appropriately. For bipolar intracardiac signals, setting the high-pass filter at 25-30 Hz and low-pass filter at 250-300 Hz usually does not significantly affect the quality of the signals. For unipolar intracardiac signals, they should be minimally filtered. In the OU-EP laboratory, we set the high-pass filter for *unipolar* EGM at 0.1-1 Hz and low-pass filter at 500-600 Hz. To be inclusive, the highest frequency available in the recording and mapping system may be chosen as the corner frequency of the low-pass filter. Selection of the corner frequency of the *unipolar* high-pass filter is a bit tricky. In theory, one should select the lowest frequency available (e.g. 0.05 Hz). However, it would allow the frequency of respiration (0.2 Hz, 12 respiratory cycles per minute) to pass through, causing baseline drift shown in the unipolar EGM (**Figure 2.11**). Choosing 1 Hz as the high-pass corner frequency will eliminate such respiratory drift but potentially filter out the low-frequency component on the distal unipolar EGM, creating a pseudo “QS” pattern and leading the operator to believe that the tip electrode of the ablation catheter is touching the source of a focal tachycardia. Mapping a focal arrhythmia (e.g. focal AT or PVC), the author prefers to set the band-pass filter of the distal unipolar EGM at 0.1-500 Hz to avoid filtering out low-frequency signals and creating a “pseudo QS” pattern. Baseline drift with respiration is usually not visible if the sweep speed is set at 200 mm/sec.

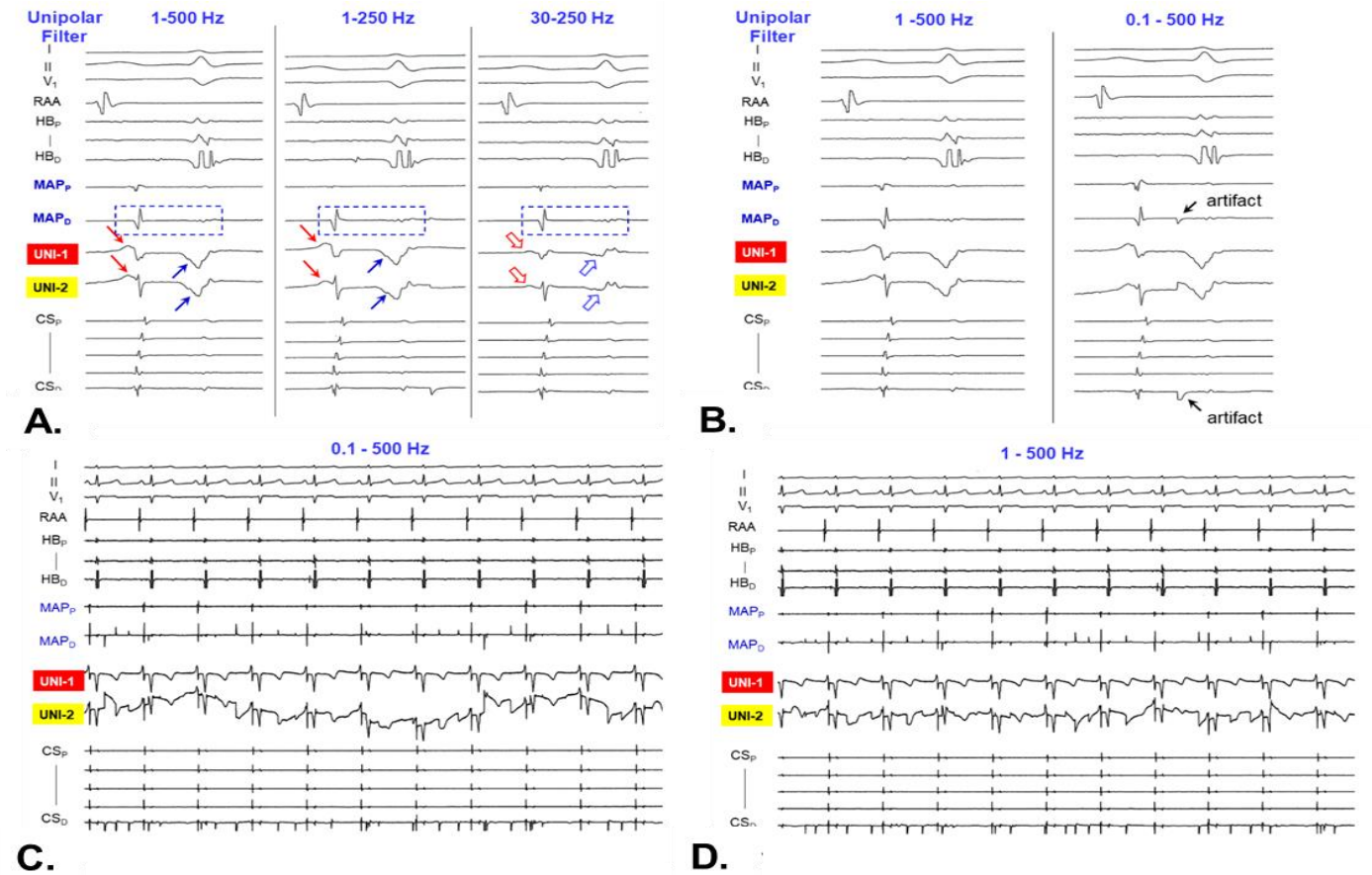


Figure 2.11. Effects of high-pass corner frequency on unipolar and bipolar EGM. A. In this example, different filter settings of the *unipolar* EGMs had no effect on the bipolar EGM (blue dotted boxes) because the filter setting of the unipolar EGMs was more inclusive (1-250 or 1-500 Hz) than the *bipolar* EGM filter (30-250 Hz). When the band-pass filter of the unipolar EGM was set to be 1-250 Hz (**middle panel**) or 1-500 Hz (**left panel**), there was no difference between the *unipolar* atrial potential (red arrow) and far-field *unipolar* ventricular potential (blue arrow) because all the frequency components of the unipolar EGMs are in the range of 1-250 Hz. No signal was lost to filtering. However, when it was set to be 30-250 Hz (**right panel**), the slow component of the unipolar atrial potential disappeared (red empty arrow). The morphology of the far-field unipolar ventricular potential changed substantially (blue empty arrow). **B.** There is no difference in the morphology on distal unipolar EGM (UNI-1) between filter setting of 1-500 Hz and 0.1-500 Hz because all components of the unipolar signals were in the range of 1-500 Hz. Sweep speed was 200 mm/second. **C.** Respiration (12 times a minute, 0.2 Hz) led to fluctuation of the UNI-2 baseline (filter setting: 0.1-500 Hz) but not the UNI-1 baseline (filter setting: 1-500 Hz). This fluctuation was only evident at a lower sweep speed, not at 200 mm/second. **D.** No respiratory fluctuation of baseline when the filter setting of UNI-2 was adjusted to 1-500 Hz.

2. Notch filter

In an EP recording or mapping system, a band-rejection or notch filter is set to reject 50 Hz or 60 Hz noise introduced by the AC power supply. However, there are 50 or 60 Hz cardiac signals as well. While rejecting the 50 or 60 Hz noise, a notch filter is notorious for introducing new noise (usually higher frequency) into the original signal. It also distorts the original signal. For EP signals, notch filter is the worst type of filter in terms of distorting the original signals, followed by the high-pass filter. For this reason, the 60-Hz notch filter for intracardiac recording is *never* turned on in the OU-EP laboratory. If 60-Hz noise appears on intracardiac EGMs, efforts are made to identify and remove the sources of noise rather than trying to cover it up with notch filter. Because 60-Hz notch filter does not significantly affect the ECG signals, it is often turned on in the OU-EP laboratory. Dr. Jackman prefers *not* to turn it on.

An idealized filter response is to multiply the frequencies in the range of "pass" by a factor of 1 but multiply the frequencies in the range of "not pass" by a factor of 0 (**Figure 2.12A**). However, the response is rarely idealized. Most of the analog electronic band-pass filter is an RLC circuit (a resistor–inductor–capacitor circuit). It takes time to charge and discharge the capacitor, thereby introducing phase delays into various frequency components of the signal of interest. The output signal after filtering may look different from the input signal because some frequencies in the input signal have been delayed. If a 10 ms delay is introduced to a frequency component carrying the largest positive amplitude, this delay may allow the positive amplitude to be "neutralized" by another frequency component carrying a negative amplitude. The largest amplitude of the output signal may become smaller than that of the original signal. Ideally, a band-pass filter should have a very high roll-off (the steepness of the transition between the passband and stopband) but it is usually not the case (**Figure 2.12B**), which potentially can distort the signal. Another problem of filtering is that it can introduce ringing. Typically, a 60-Hz notch filter will have to filter out frequencies surrounding 60 Hz as well (e.g. 59.6 – 60.4 Hz) because in theory, to filter out only the 60 Hz signal, it can ring forever (**Figure 2.12C**).

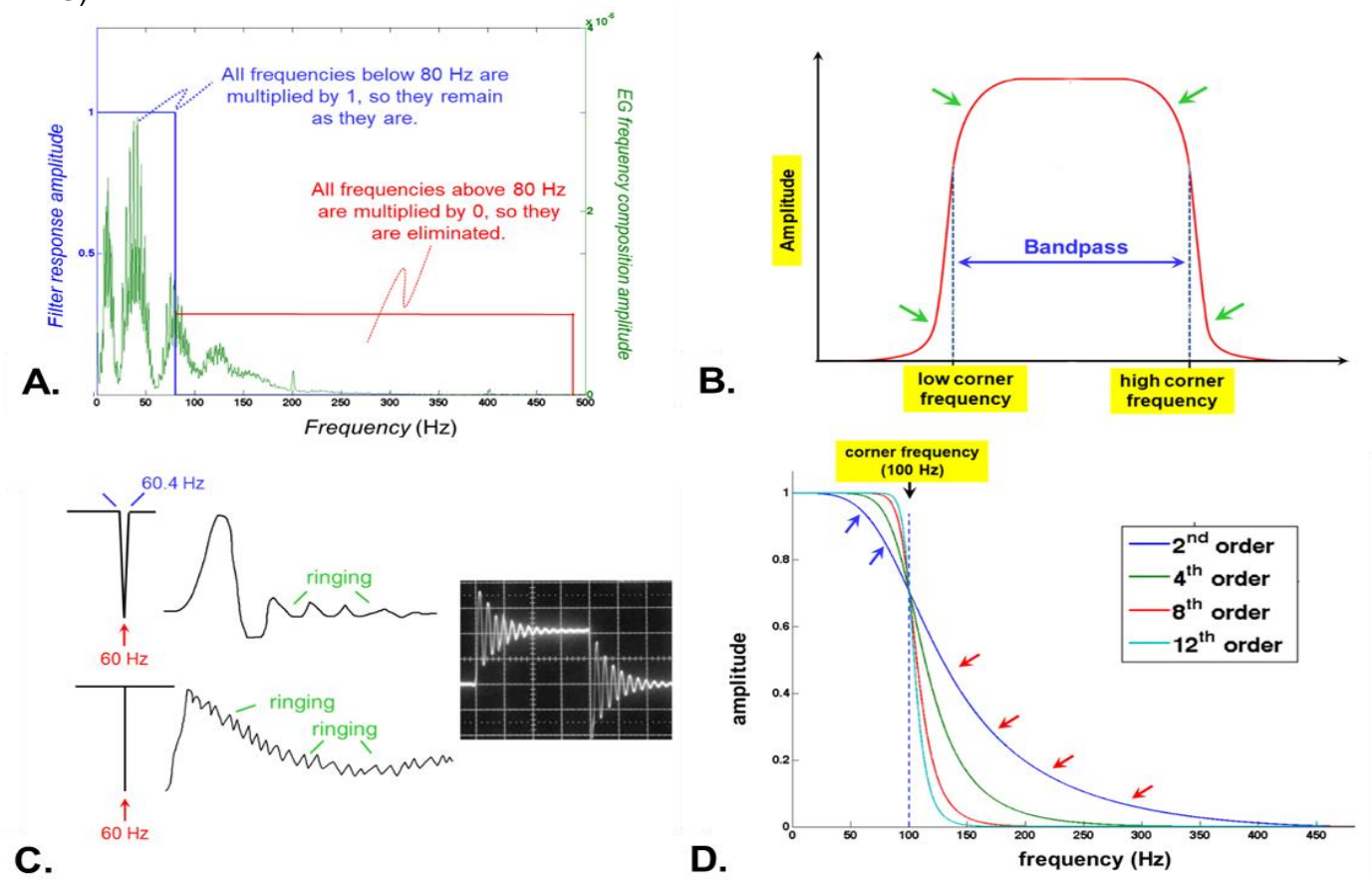


Figure 2.12. Effects of filtering on signals. A. In theory, all the signals above the corner frequency (80 Hz) will be eliminated by a low-pass filter and signals below 80 Hz will be preserved. **B.** Most filters are NOT ideal, with a slower roll-off (green arrows). **C.** Filters can introduce "ringing" (inset). Most filters will filter out frequencies surrounding the targeted frequency (**left upper panel**); otherwise, it may introduce ringing that lasts forever (**left lower panel**). **D. Compare roll-off of different orders of filters**. A 2nd order filter has slow roll-off and starts attenuating signals (blue arrows) well before the corner frequency (100 Hz) as well as allows frequency>100 Hz to pass (red arrows). A higher order filter (e.g. 8th and 12th order) has sharp roll-off; the passband resembles the assigned corner frequency; however, they introduce more phase delays.

3. Filter order

Filter order can determine how *sharp* the roll-off is between the passband and the stopband. Basically, the number of the order is determined by the number of active electronic components that affect the filter's frequency response. For example, a first order filter has one capacitor or one inductor that affects the filters

frequency response. A second order filter may have two capacitors or two inductors, or one capacitor and one inductor affecting the filter's frequency response. The higher the number of the order, the sharper the roll-off (**Figure 2.12D**) but higher order filters also introduce more delays in various frequency components of the signal because of more active electronic components involved. It is important to know that phase delay is related to the frequency of the signal. Different frequency component of the signal of interest will have different phase delays when the signals are filtered. The morphology of the signal of interest may look different from the original signal. As already discussed, in the OU-EP laboratory, intracardiac signals are routed to the CARTO-3 patient interface unit (PIU) first; signals are then routed to the Bard recording system. If the band-pass filter settings of the Bard and CARTO-3 system are very different, signals displayed on the two systems may look different.

Figure 2.13 illustrates the effects of notch and band-pass filtering on the original EGM. While a notch filter is a poor choice to eliminate the 50- or 60-Hz noise, an adaptive filter is a slightly better choice, which is a form of digital filter (**Figure 2.13D**). Adaptive filters have pre-programmed optimization algorithms to track the actual frequency of the noise as it fluctuates and subtract the noise from the recording. It functions similar to a noise-cancellation headset. Signals processed by adaptive filters in general have less distortion than signals processed by notch filters but may still introduce noise into signals. One should avoid using them, too.

In general, digital signals are much easier to process than analog signals. Digital filters are a lot more powerful and sophisticated than the aforementioned analog filters. An ideal catheter is one that has an analog-to-digital converter connected to the end of the catheter. In this way, signals are digitized before they are exposed to electromagnetic interferences, which should eliminate most of the noise seen in the EP laboratory.

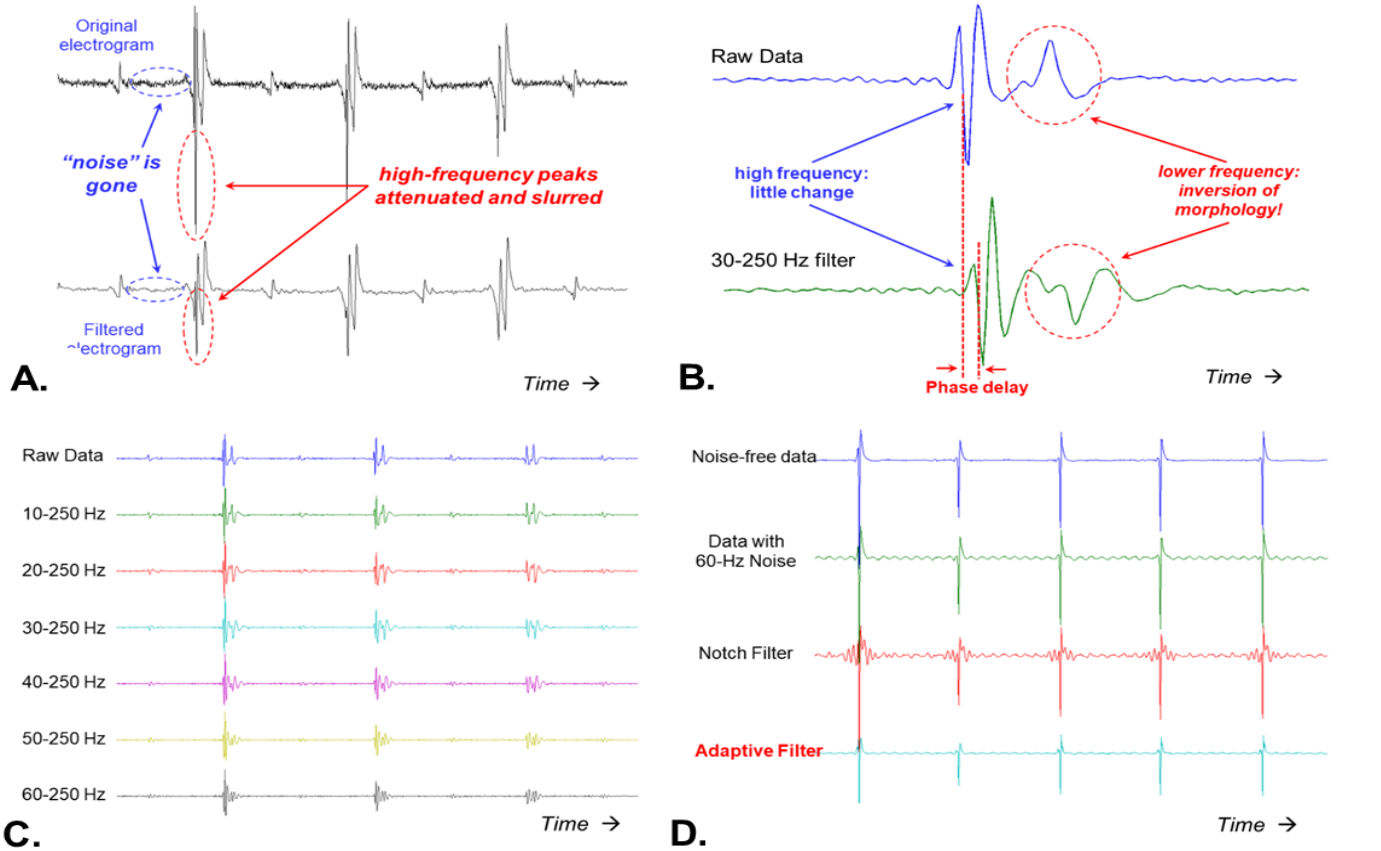


Figure 2.13. Effects of filtering on EGM. The original EGMs in these illustrations came from the raw data in the Bard recording system**. A.** 60 Hz noise was removed by notch filter but signals were changed as well. **B**. Another example of EGM affected by filtering. Note that there is a phase delay between the raw and filtered signals. **C.** Effects of different filter setting on EGM morphology. Note that high frequency noise was introduced by filtering. **D.** Compared to notch filter, adaptive filter (similar to a noise cancellation headset) introduces less noise to the raw signals.

4. Artifacts caused by filtering

ECG

Figure 2.14 and Figure 2.15 illustrate typical examples of how the original QRS complex was distorted by inappropriate setting of the band-pass filter. Similar to intracardiac EGM, phase shift (time delay) introduced by filtering plays a major role in the changes in the QRS complex as well. Many cardiology fellows have received unnecessary consults from the ICU team for ST elevation on the ICU monitor screen; 12-lead ECG at the same time showed no ST elevation at all. This discrepancy is usually caused by different high-pass filter setting of the monitor screen (e.g. 0.5-40 Hz) and 12-lead ECG (0.05 to 100 or 150 Hz). In general, inappropriate setting of the high-pass filter has more impact on the ST segment (**Figure 2.14**) while low-pass filter settings are more likely to affect the morphology of the QRS complex. The main purpose of telemetry monitoring is to monitor the patient's heart rate and detect arrhythmias. It is more important to filter out noise such as motion, respiratory movement (low frequency) and electromagnetic interference (high frequency). While many telemetry monitoring systems set their band-pass filter at 0.5-40 Hz, some of them have a band-pass of 0.67-21 Hz! Erroneous ECG band-pass filter setting in the EP laboratory can create problems when operators try to match induced PVCs with clinical PVCs or try to analyze the morphology of the QRS complex to localize the site of origin of PVCs or VT (**Figure 2.15**).

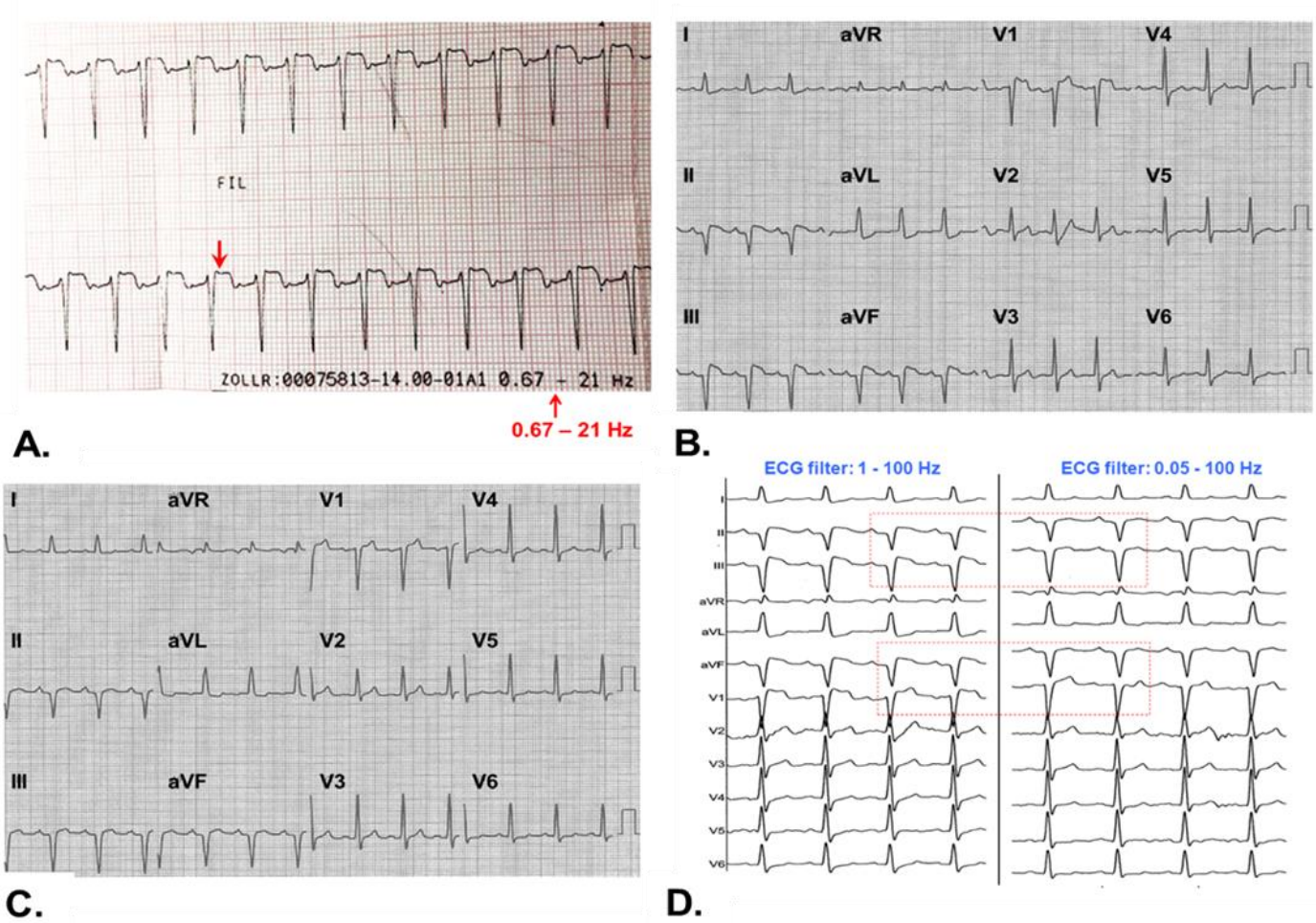


Figure 2.14. Effects of high-pass filter on ECG. A. ST elevation (red arrow) was noticed on a hospital monitor with a band-pass filter setting of 0.67-21 Hz. **B**. In a 49 y/o male with ischemic cardiomyopathy, ST elevation in lead II, III, aVF and V1 suggests inferior wall aneurysm from prior myocardial infarction. **C.** ECG recorded 10 minutes later showed that ST elevation resolved. Coronary artery spasm was suspected. **D.** It turned out that the band-pass filter was set to be 1-100 Hz in (**B**) and 0.05-100 Hz in (**C**). Note that QRS complexes with a QS or rS pattern had ST elevation (red dotted boxes) when the high-pass filter was set to be 1 Hz.

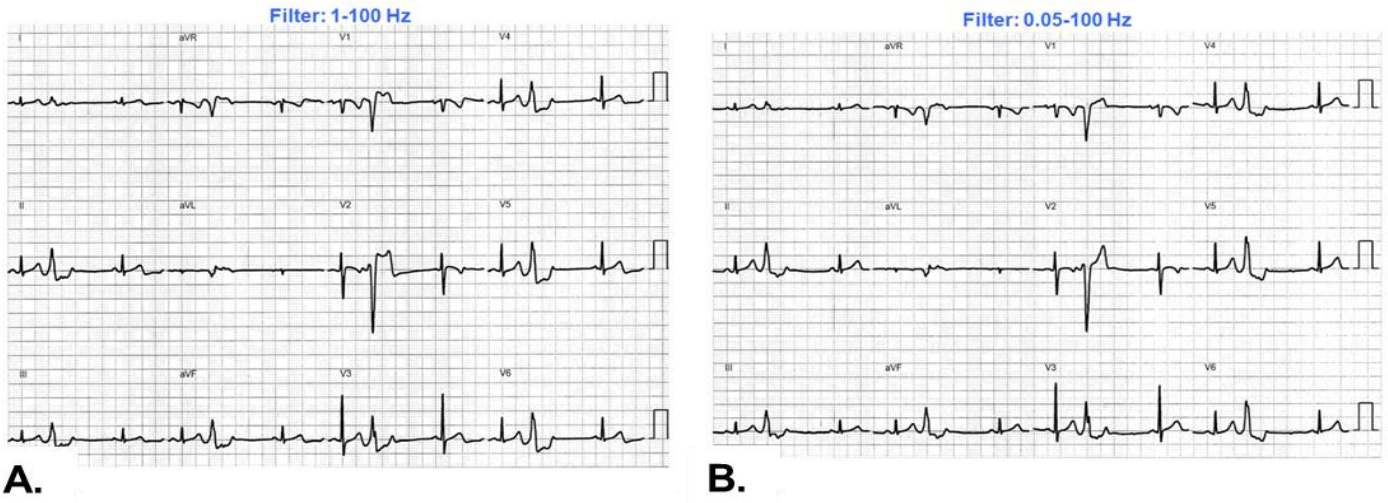


Figure 2.15. Effects of high-pass filter on ECG localization of PVCs. Note the striking differences in the QRS morphology of PVCs when the high-pass filter was set differently.

Intracardiac EGM

Figure 2.16 illustrates the combined effects notch filter and widely-spaced electrode spacing on the quality of EGM. If the band-pass filter of the distal electrode pair of the ablation catheter is set to be 30-400 Hz, the site of apparent "earliest" activation on the bipolar EGM would have excluded any signal with a frequency <30 Hz. On the other hand, minimally filtered unipolar EGM (e.g. 0.1-500 Hz) may show a low-frequency far-field signal, informing the operator that this site is not the source of the focal arrhythmia (**Figure 9.1**). In theory, the morphology of the bipolar EGM should reflect the difference between the two unipolar EGM. However, because of the different settings of the band-pass filter (e.g. 30-400 Hz for bipolar EGM and 0.1-500 Hz for unipolar EGM), the bipolar EGM morphology may significantly deviate from the difference between the two unipolar EGM. As discussed early, notch filter (50 or 60Hz) is notorious for introducing high frequency noise into the signal and distorting the signal of interest. **Figure 2.17 and 2.18** illustrate several examples in which 60 Hz notch filter distorted EGM and introduced high frequency noise into the EGM.

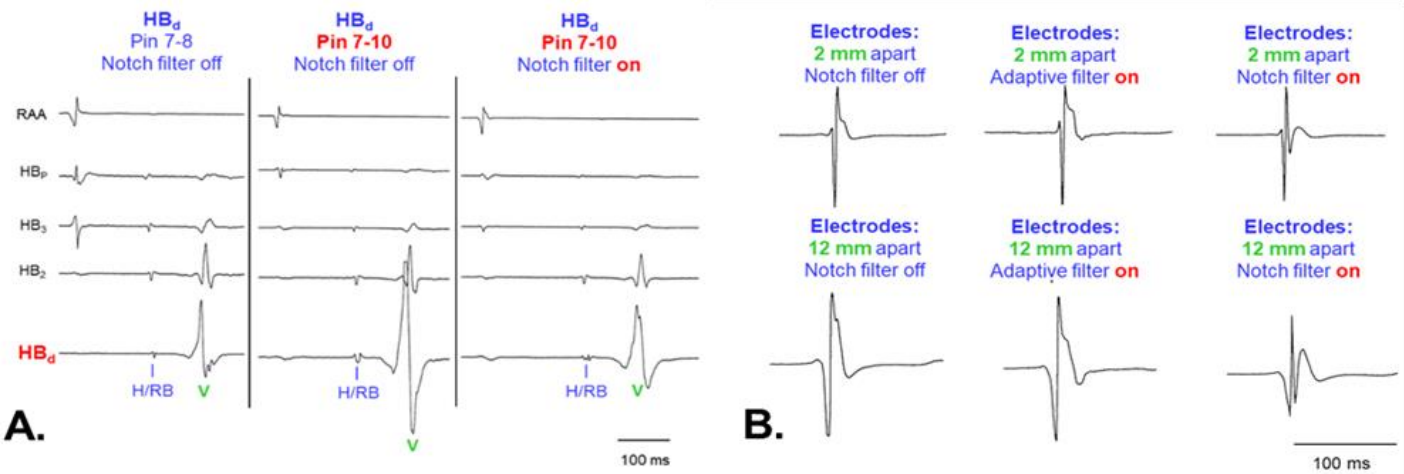


Figure 2.16. Effects of electrode spacing and 60-Hz notch filter on EGM. A. Left panel. The bipolar electrode pair (HBd) was a closely-spaced electrode pair (electrode size: 2mm, spacing 2mm). **Middle panel.** Electrode spacing was changed to 10 mm. Note that the local ventricular potential (V) became much larger when the electrode spacing increased. The HB or RBB potential (HB/RBB) became wider. **Right panel**. When the 60-Hz notch filter was turned on, the HB/RBB potential recorded on the widely-spaced electrode pair became smaller and fractionated; the ventricular potential was smaller. **B.** Examples of coronary sinus EGM affected by electrode spacing and filtering.

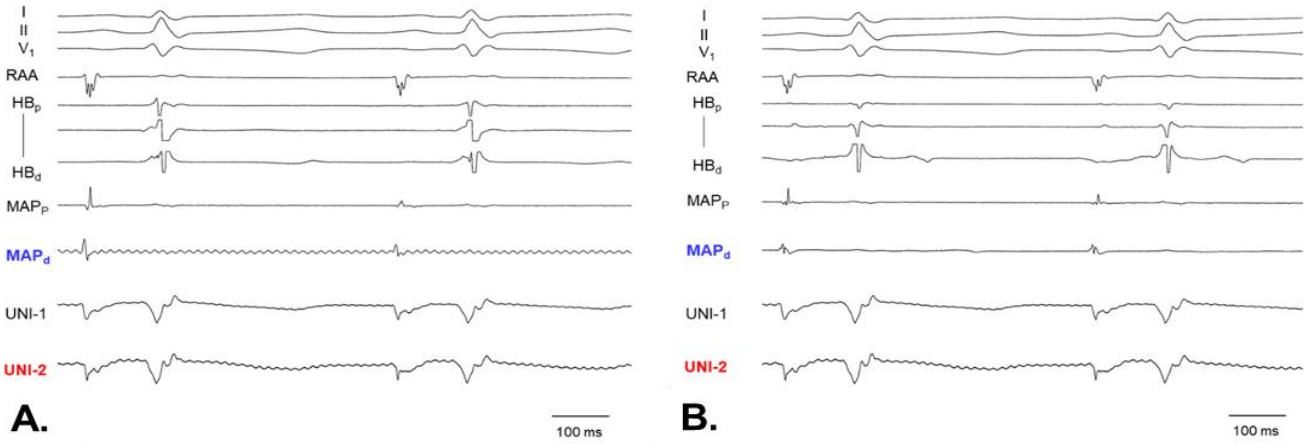


Figure 2.17. Effects of 60-Hz notch filter on EGM. A. Noise was visible on the bipolar EGM (MAPd) and proximal unipolar (UNI-2) EGM, suggesting that the source of noise came from UNI-2. **B.** Instead of eliminating the noise source, the 60-Hz notch filter of the bipolar electrodes was turned on. The noise on the bipolar EGM was eliminated but the morphology of the bipolar EGM was distorted. Note that the source of noise from the UNI-2 electrode remained unchanged.

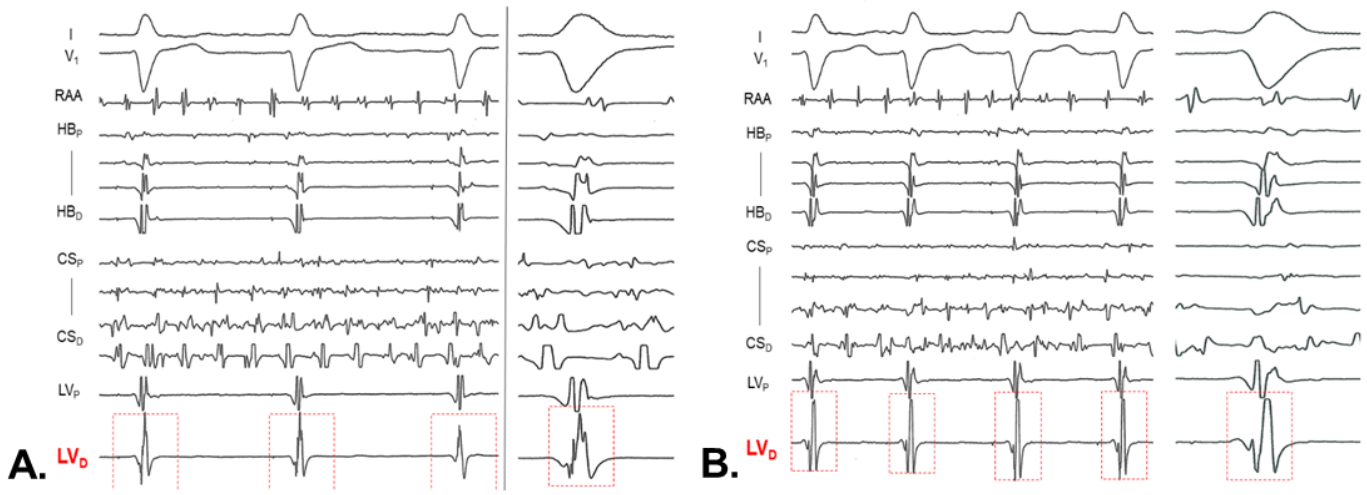


Figure 2.18. Effects of 60-Hz notch filter on EGM. A. In a 48 y/o male with non-ischemic cardiomyopathy and AF, LVd EGM showed multiple high-frequency component, suggestive of local abnormal ventricular activity (LAVA), when the 60-Hz notch filter was turned on (**left panel**: 100 mm/sec sweep speed; **right panel**: 200 mm/sec sweep speed). **B**. High-frequency components disappeared when the notch filter was turned off. Note that the EGM recorded on the proximal electrode pair (LVp) was identical between **A** and **B**, indicating that the catheter remained at the same site.

Noise Reduction in EP Laboratory

The patient's body functions like an antenna, capable of picking up all kinds of electromagnetic interferences in the EP laboratory. A noise-free EP laboratory is every electrophysiologist's dream. In the OU-EP laboratory, the average noise level is about 15-20 μV when irrigated catheters are in use. The author would like to share our experience in noise reduction with the readers. Most noise is introduced before the ECG or intracardiac signals reaching the EP recording system or EAM system. The noise can be alleviated by following a daily routine of patient preparation, cable management and equipment placement. In fact, careful cable and equipment placement can eradicate most of the noise in the EP laboratory. For example, the signal of interest is often in the range of tens of μV to 10 mV. To deliver a 25-watt RF application in the presence of 100 Ω impedance, the RF generator puts out 70 volts of voltage, ≥7,000x higher than the intracardiac signal of interest. The voltage of the power line is even higher (110-220 volts). If a signal cable is placed in close proximity to an RF cable or a power cable, signals of interest are doomed to interference from

adjacent electromagnetic fields. With the advances in recording, mapping and ablation technologies, it often requires multiple independent systems operating at the same time. The clinical support staff of each manufacturer sometimes may not even have a good grasp of the technology s/he supports, not to speak of the interactions with technologies of other manufacturers. Noise troubleshooting therefore becomes a daunting task. In the OU-EP laboratory, we use Bard (Boston Scientific) for EP recording and mainly the Biosense system for mapping. The discussions below are based on these two systems but the principles should be applicable to all recording and mapping systems. Readers may want to consult with the clinical support staff of each manufacturer to help alleviate the noise in the EP laboratory.

1. First thing first

The surface ECG system is often viewed as independent of the intracardiac recording system; however, the patient's body is the shared domain of the two systems. For example, the dispersive patch of the RF generator, the location pads of the mapping system and ECG patches are all placed on the skin. A loose skin patch of any of the three systems can introduce noise into other systems. A loose patch or bad lead wire of a limb lead often causes horrendous noise as Wilson's central terminal is commonly used as the reference point for the intracardiac unipolar EGM and precordial ECG leads. Wilson's central terminal is formed by connecting 3 high impedance resistors from three limb leads (LA, LL and RA; **Figure 2.19A**). The clinical engineer of the OU-EP laboratory runs an ECG simulator (**Figure 2.19B-C**) once a week to screen for bad ECG lead wires or connectors. This simulator generates electrical signals like what a live patient should generates. If any ECG lead shows noise, it indicates defects in the lead wire that requires replacement.

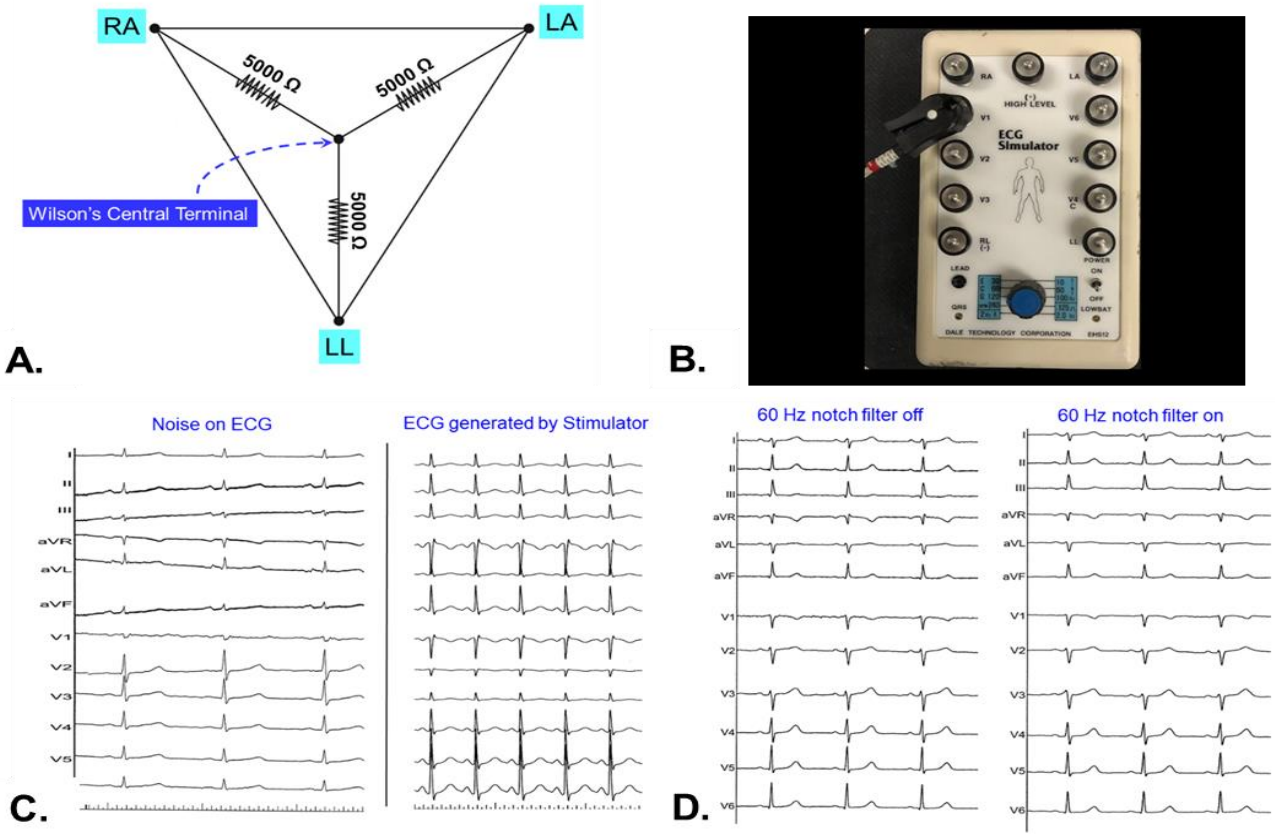


Figure 2.19. The first step to a noise-free EP laboratory is a noise-free ECG. A. Wilson's central terminal serves as the reference point for unipolar (precordial) ECG leads and some intracardiac EGMs. **B. ECG simulator used in the OU-EP laboratory.** If any ECG lead looks noisy, it indicates that lead wire or clip is defective. **C. Left panel.** High frequency noise was noted on 12-lead ECG, both in the Bard recording system and CARTO, indicating noise came from sites before ECG cables were plugged into the CARTO patient-interface unit. **Right panel.** When all 12 lead wires were connected to the ECG simulator, there was no ECG noise, indicating that the ECG lead wires and clips were good. In this example, the noise was introduced by the electromagnetic field of the lateral x-ray tube. **D.** Representative ECGs in the OU-EP laboratory. **Left panel.** 60-Hz notch filter was turned off. **Right panel.** notch filter turned on. There was minimal difference in the noise level between the two.

The very first thing that the staff of the OU-EP laboratory does in the beginning of an ablation procedure is to record a 12-lead ECG *without* turning on the 60-Hz notch filter to expose electromagnetic interferences. If the noise level without the 60-Hz notch filter is high, patches and lead wires are inspected carefully. Importantly, the sequence of action is to connect the ECG leads of the EP recording system first, then connect anesthesia ECG leads, followed by the defibrillator leads. This helps to identify noise induced by individual system. The noise level with and without notch filter should not vary too much (**Figure 2.19D**). To leave the 60-Hz notch filter on or off for the remainder of the procedure is up to each operator. Dr. Jackman's preference is to *turn off* the notch filter so that he can detect new noise occurring in the middle of the procedure.

2. Equipment placement

All equipment is placed adjacent to the x-ray table to minimize the distance that the signal cables have to travel to reach the amplifiers of the recording and mapping system because cables can function like antenna and pick up noise. The longer the signal cables are, the more likely they will pick up some noise. However, equipment with an AC power source should not be anchored to the metal rail of the x-ray table because equipment carrying an AC power supply (120-220 volts) will be too close to the cables carrying low-voltage signals. In the OU-EP laboratory, only the equipment that does not carry an AC power supply is anchored to the rail of the x-ray table (**Figure 2.20A**). RF generators, Bard amplifier, pump of the irrigated catheter and all other equipment with an AC power supply are placed on a boom that is about 2 feet distant from the foot end of the x-ray table (**Figure 2.20B**). This boom is grounded to the earth ground. The 2 feet distance allows the equipment to be placed in close proximity to the patient but not too close to be in contact with the signal cables. The chassis of each piece of equipment on the boom is grounded to the ground pin at the bottom of the x-ray table (**Figure 2.20C**). This practice is to prevent noise introduced by ground loop in which two points intended to be at the same ground reference potential indeed have a potential between them. When a signal cable is connected between them to transfer data, current can flow from one to the other and introduce noise into the signal of interest. The floor of the EP laboratory may have static electricity or have power cords, both of which can introduce noise to the signal of interest. Signal cables and RF cables should not be left on the floor (**Figure 2.20D**).

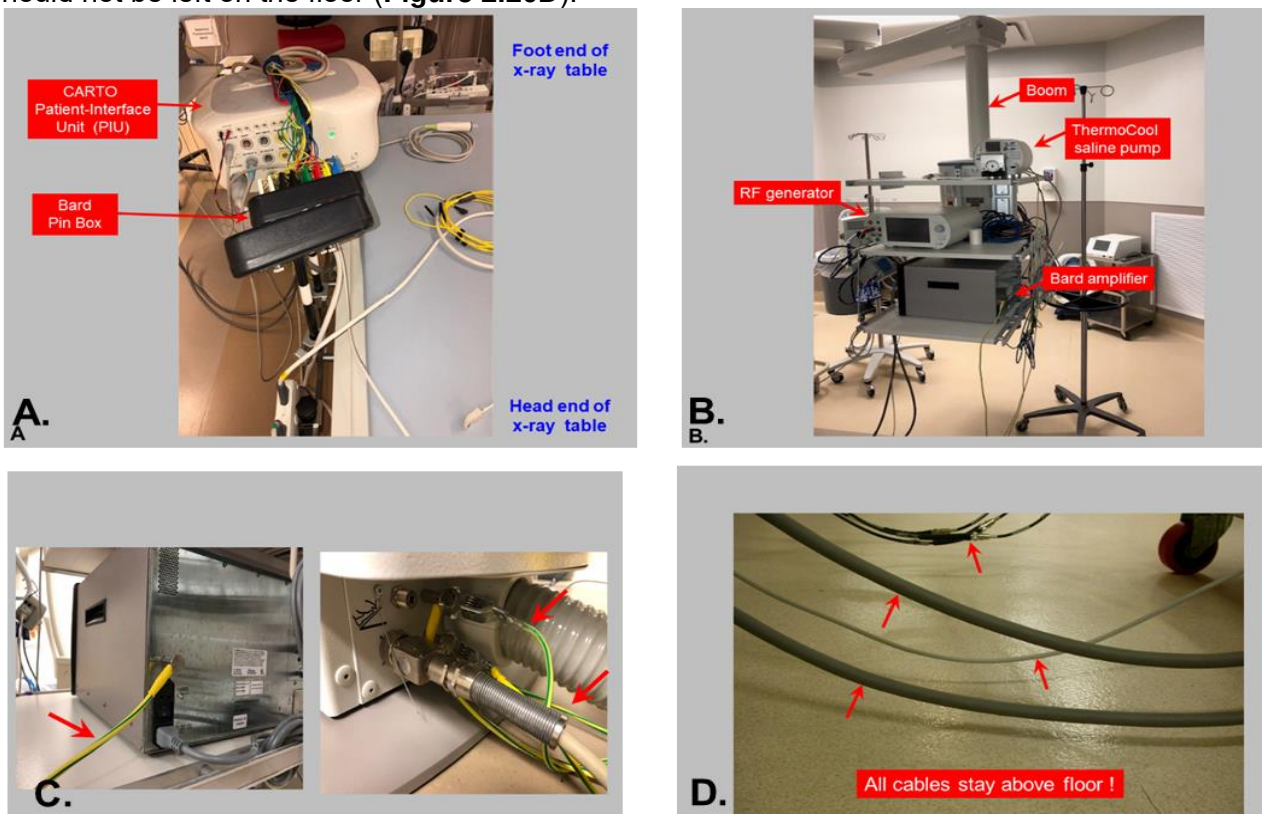


Figure 2.20. Equipment placement. A. The CARTO-3 PIU and Bard pin box are mounted to the rail at the foot end of the x-ray table because they do not carry AC power supplies. **B.** The Bard amplifier, RF generator, ThermoCool saline pump are placed on a boom. Each of the equipment on the boom is connected to the common group at the bottom of the x-ray table. The boom itself is connected to the earth ground. **C.** A ground cable (red arrow) connecting the chassis of the Bard amplifier to the ground pin at the bottom of the x-ray table **(right panel)**. **D**. All cables are arranged to be above the floor to avoid picking up static electricity.

In the OU-EP laboratory, the Biosense PIU and the pin boxes of the Bard recording system are placed adjacent to the foot end of the x-ray table (**Figure 2.20A**). This practice is to shorten the distance that catheter cables are exposed to potential sources of noise. Of note, the Biosense PIU is encased in a plastic box and is operated on DC current, which rarely introduces electrical noise to the signals. The AC current is converted to DC current by a transformer, which is placed on the floor under the foot end of the patient table at a distance to the PIU (**Figure 2.21A**). Because the PIU is run by DC current and in a plastic box, unused jumper wires are placed on top of the PIU to avoid picking up noise (**Figure 2.21B**). On the other hand, the Bard amplifier has an AC power source; the amplifier is placed on the boom at a distance to the x-ray table (**Figure 2.20B**).

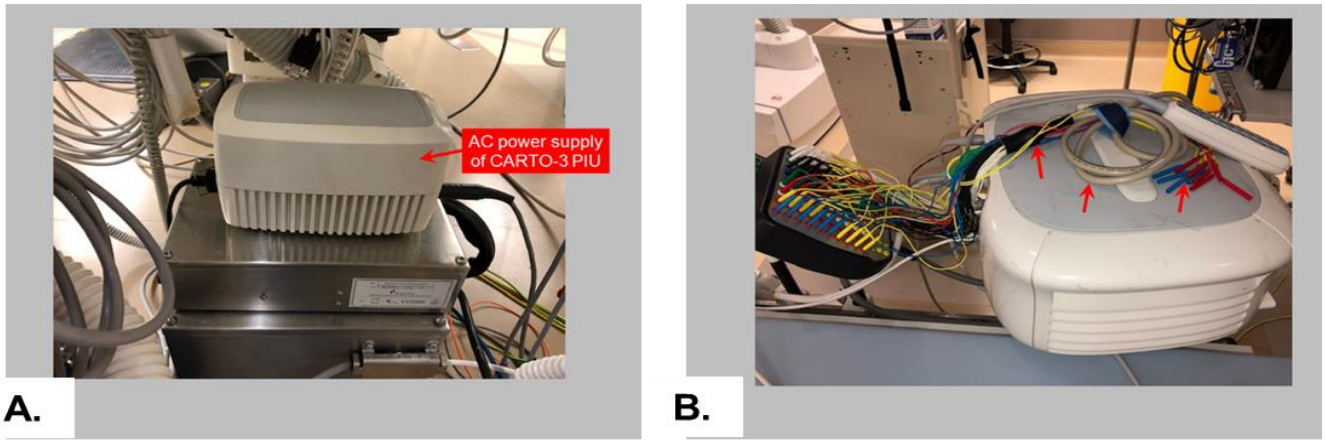


Figure 2.21. Equipment placement. A. CARTO power supply converts AC to DC; PIU is run by DC power sources. This power supply is placed at a distance from the CARTO-3 PIU which is mounted on the rail of the x-ray table. **B.** Unused cables are placed on top of the CARTO-3 PIU to avoid picking up electromagnetic interferences because this plastic box is powered by DC current.

3. Cable management

All signal cables are placed on top of the x-ray table and routed toward the patient's feet to exit the table. Cables with excessive length are coiled and taped to sheets of the x-ray table to avoid falling off from the side of the x-ray table or falling onto the floor (**Figure 2.22A**). Most of the cables of the EP recording system and mapping system are shielded, in which the core cable carrying electrical signals is covered by insulated, metal shielding connecting to ground and plastic jackets. A cable shield functions like a Faraday cage to block electromagnetic interferences (**Figure 2.22B,** left panel). Shielded cables prevent crosstalk between cables. In contrast to shielded cables, unshielded cables (e.g. the jumper cables between the CARTO PIU and Bard pin box **Figure 2.22B,** right panel) are more prone to stress and fracture. Unshielded cables should be handled with more caution. When part of the jumper cables is not used, they should still be plugged into the pin box because unplugged pins can function like an antenna and pick up noise (**Figure 2.22C**). If the entire bunch of jumper cables are not used, they can be laid on top of the plastic case of the CARTO PIU which does not have an AC power supply (**Figure 2.21B**). Fiber optic cables rarely introduce noise to the recording or mapping system. Placement of this type of cable is less of a concern.

If a pin is loosely plugged into the pin box, it may function as an antenna, picking up and introducing noise into the pin box. To avoid noise introduced by loose pins, all the pins are securely plugged into the pin box all the time (**Figure 2.22C**). If a catheter is withdrawn out of the patient's body, the pins of the catheter cable should still be plugged into the pin box. The catheter and its cable should remain on the x-ray table to

stay away from potential noise sources. If the catheter end of the cable is thrown off the table, it can pick up noise from the floor or the nearby AC power supply. Another common source in the EP laboratory is when multiple mapping systems are used in the laboratory, the cables of one system have to be disconnected and reconnected. At a glimpse, cables appear to be securely plugged in. Loose connection to the amplifier can introduce noise to multiple catheters if this cable carries the signals from multiple catheters. The clinical engineer of the OU-EP laboratory, Mr. Thomas Hayes, conducts a "wiggle" test periodically. He gently wiggles the connectors to the pin boxes or amplifier (**Figure 2.22D**) to make sure they are securely plugged in.

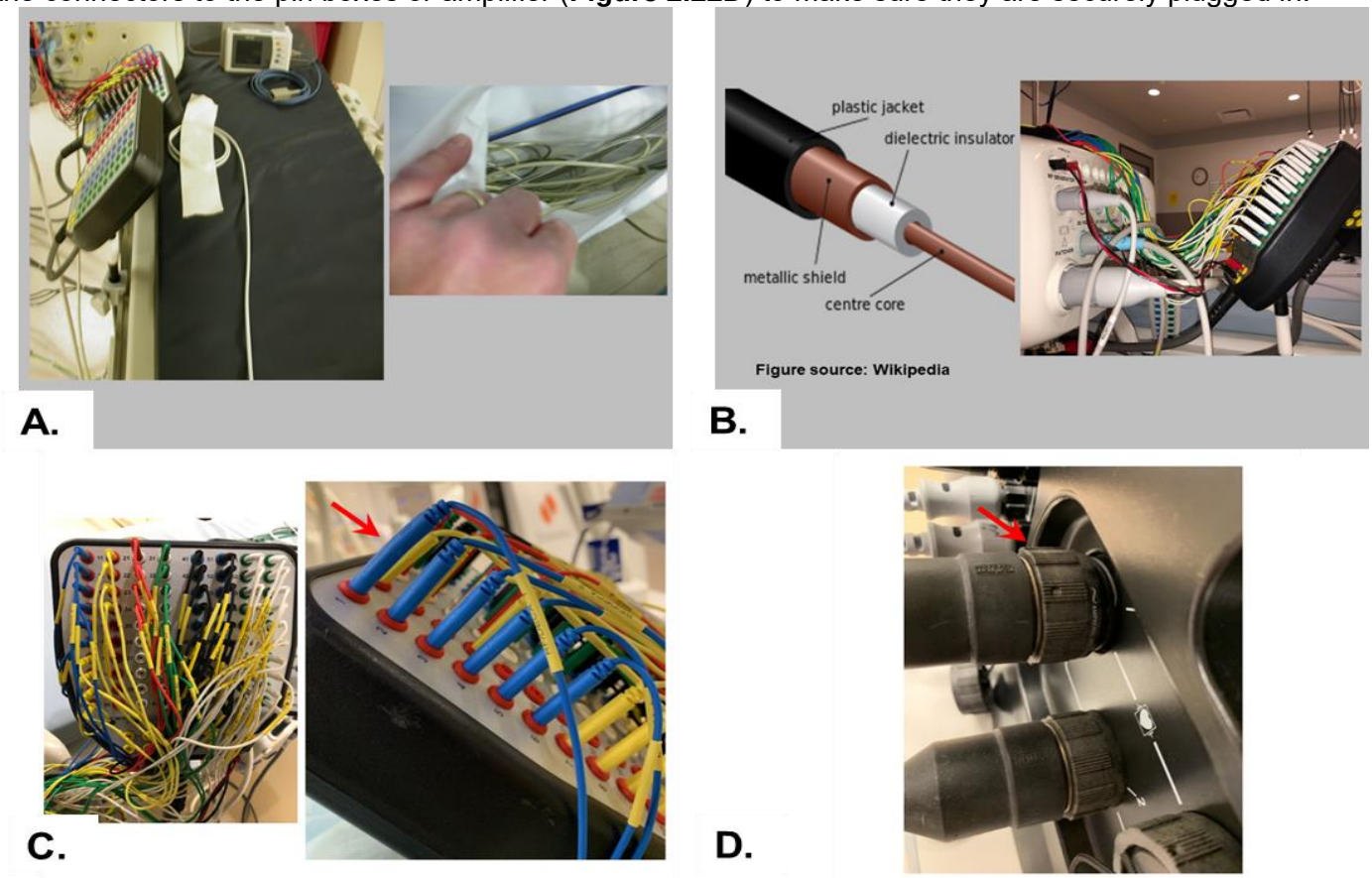


Figure 2.22. Cable management. A. Excess length of cable is looped and held in place by tape or table sheet. **B. Left panel:** Major components of a shielded cable. **Right panel:** The color-coded jumper cables from the CART-3 PIU to Bard pin box are not shielded, prone to mechanical damage and noise. **C. Left panel:** The connecting cables between the CARTO PIU and Bard pin box are plugged in all the time, regardless if corresponding catheters are used or not. Unplugged pins may function like antenna and introduce noise. **Right panel:** A pin was not plugged all the way into the pin box (red arrow), which can introduce noise. **D.** The interface cable connecting the Bard pin box to Bard amplifier appeared to be ok but was loosely connected (red arrow), which failed the "wiggle test". This problem can introduce noise to multiple catheters.

Operators should be aware that as soon as operators start using the fluoroscope, the power of the fluoroscopic table is operating all the time to allow the operator to move the x-ray table and use fluoroscopy at any moment. In some x-ray systems, the x-ray tube is spinning at 10,000 rpm all the time, generating a horrendous magnetic field (**Figure 2.23**). The magnetic field near the power supply of the fluoroscopic machine is usually high. Allowing the signal cable to exit from the middle of the patient table and fall below the table can expose the signal cables to strong magnetic field. The floor of the EP laboratory may have static electricity or have power cords, both of which can introduce noise into the signal of interest. Signal and RF cables should not be left on the floor. Another potential source of noise is the monitor screen. **Figure 2.23** showed that the large monitor screen can generate 8 mGauss of magnetic field. Because any equipment carrying an AC power supply can introduce noise to the signal by its electromagnetic field, one can use a Tri-field meter to probe around the EP laboratory to know the sites with high magnetic field and avoid routing signal cables through these sites (**Figure 2.24A**).

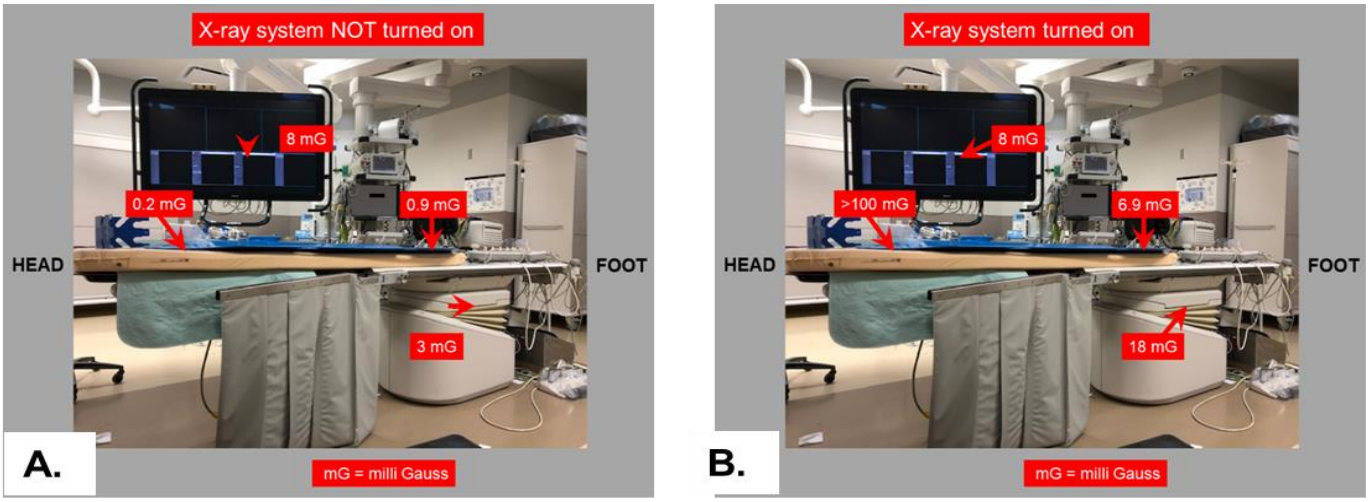


Figure 2.23. Magnetic field strength near the x-ray table. A. When the x-ray system is not turned on, the base of the table, where the AC power supply and electronic components driving the table are located, has a higher magnetic field. It is worth noting that the large screen displaying fluoroscopic images, EGM and electro-anatomical mapping also has a high magnetic field. Signal cables should not be in close proximity to a monitor screen. **B.** When the x-ray system is turned on, the magnetic field, at the site where the x-ray tube is located, can skyrocket to >100 mGauss.

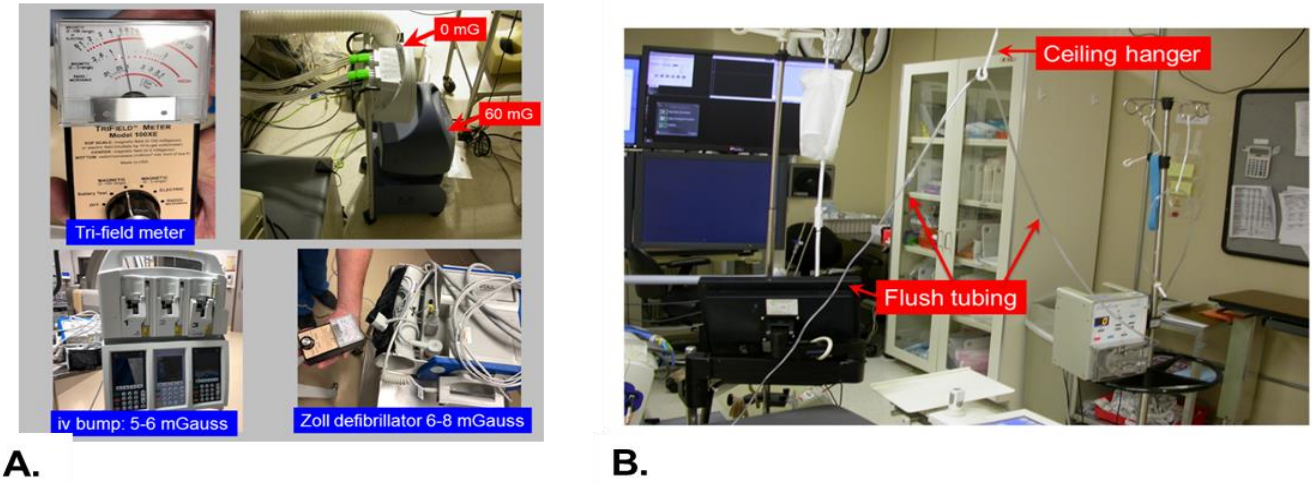


Figure 2.24. Magnetic field strength of various equipment. A. Left upper panel: A Trifield meter is routinely used by the clinical engineer of the OU-EP laboratory to monitor areas with high magnetic field. **Right upper panel:** The tube of the patient warmer did not generate a magnetic field but the case housing the power supply generates 60 mGauss of magnetic field. **Bottom panels.** Magnetic fields generated by iv pump (left panel) and Zoll defibrillator (right panel). **B.** If the saline flush tubing generates noise, it can pass through a plastic hanger on the ceiling and directly connect to the end of the irrigated catheter without touching anything en route.

When current flows through a straight wire, it induces a circular magnetic field. When current flows through a wire wrapped in the shape of a coil, it induces magnetic field lines that point in the direction of the coil's long axis. The magnetic field induced by current raises a question: how should the signal cables be arranged? Should signal cables be arranged in parallel or be coiled? Should signal cables crisscross? Each EP laboratory does it differently. Bundling cables together is convenient but if one cable is bad, it can introduce noise to other cables in the same bundle. Since cardiac signals, in the range of 20 μV to 10-15 mV, are carried in cables with relatively high resistance, the current flow through the cable is very small. The OU-EP's anecdotal experience is that how the signal cables are physically arranged probably make very little difference in terms of noise induction. The most important practice is not to allow the signal cables to be in close proximity to RF or power cables or any equipment with an AC power supply. In the OU-EP laboratory, excessive length

of cables is arranged in loops to prevent them from falling off the table to be in contact with noise sources (**Figure 2.22A**).

Hoses carrying warm air to the patient to maintain patient's body temperature can be a source of noise. The OU-EP laboratory uses a unit named "Bair Hugger" in which the plastic tube is supported by metal coil (**Figure 2.24A**). Although the metal coil is not supposed to carry any current, the hose sometimes introduces noise by serving as an antenna. Blowing warm air through a plastic hose can create static electricity as well. If noise caused by patient warmer is suspected, the hose of a patient warming device should not be in close proximity to signal cables.

Saline itself is a conductor. Therefore, saline flush tubing (e.g. for irrigated ablation catheter) can serve as an antenna and pick up noise between the catheter and pump. In the OU-EP laboratory, if noise introduced by the saline tubing is suspected, there is a plastic ceiling hanger that allow the saline tubing of the irrigated catheter to go straight to the end of the catheter without being in close proximity of any potential source of electromagnetic interference (**Figure 2.24B**). Saline can get into the connecting cable and cause noise. Therefore, operators should always connect the catheter and cable first before connecting the catheter to saline flush tubing to prevent this problem. Sometimes, there are defects in the circuitry or power supply of iv pump. If so, it can be unplugged and operated on battery (DC current). There was an occasion in the OU-EP laboratory that the source of 60 Hz noise on the ablation catheter was nowhere to be found. When the noise suddenly disappeared for a few seconds, it was found that the anesthesiologist just turned off the stopcock carrying warm saline to the patient. There was a defect in the circuitry of the blood warmer that introduced 60 Hz noise to the patient through the iv line.

The cables from the defibrillator pads exit the x-ray table from the level of the patient's shoulder. The defibrillator is placed approximately 1-1.5 meter from the x-ray table. Although defibrillator cables do not carry signals of interest, they should not touch the floor. A bad cable or bad power supply in a defibrillator can introduce noise to surface ECG or *multiple* intracardiac signals. There was an occasion in the OU-EP laboratory that the source of 60 Hz noise on the ablation catheter was nowhere to be found. Eventually, when the power cord of the defibrillator was unplugged, the noise disappeared. After replacing this defibrillator that had a defect in its power supply, the noise never recurred.

Noise Troubleshooting

Noise troubleshooting is a daunting task because most EP laboratories use multiple mapping and recording systems and these systems are interconnected. It often requires holistic understanding of each system to track down the source of noise. Even with a good grasp of all the technologies in the EP laboratories, occurrence of some noise still makes no sense and is difficult to remove. Therefore, the OU-EP laboratory's philosophy of noise reduction is to have careful patient preparation, equipment placement and cable management in the beginning of the procedure so that noise troubleshooting is not needed. Troubleshooting Intracardiac noise is easier when we start with clean surface ECGs. If intracardiac noise appears, recheck surface leads first! Notably, noise is cumulative. If the EP laboratory has a higher noise level every day, to reduce noise to an acceptable level may require many steps. It typically requires better equipment placement and cable management as already discussed. If noise suddenly occurs during the procedure, it often is caused by a single source. Cyclic noise tends to come from sources external to patient or the recording system.

1. First and foremost

All electrophysiologists need to understand how the signal cables are routed to the EP recording system and mapping system in their EP laboratories. For example, in the OU-EP laboratory, the pins of all the diagnostic and mapping catheters are plugged into the CARTO-3 PIU. From there, signals are split and

routed to the Bard pin boxes, followed by the Bard amplifier. If the same noise appears on both the Bard and CARTO system, it indicates that the source must be in the catheter itself or the cable connecting the catheter to the CARTO-3 pin box or PIU or somewhere in the PIU before signals are split to Bard. One may start with disconnecting and reconnecting the catheter cable from the catheter, followed by replacing the cable if noise remains. Connecting cables are often re-sterilized multiple times and are prone to residue deposition, fracture and insulation break. Simply by disconnecting and reconnecting the cable 2-3 times may remove the residue on the pins. If a new cable does not eliminate the noise, it indicates that the catheter may be bad or less likely, the pin box or PIU has a problem.

Another important step is to know where in the EP laboratory has high electric or magnetic field (**Figure 2.23, 2.24**). These sites are often the sources of noise. One may start noise troubleshooting from inspecting if signal cables are routed through these areas. Before troubleshooting, ask yourself the following questions:

1. Was the same noise present in another patient? If so, noise is more likely introduced by equipment, not catheter.
2. Is the noise present on both ECG and intracardiac signals? If so, check ECG patches and lead wires first.
3. Is the noise present all the time? If not, what was done just before the noise occurred?
4. Is the noise cyclic? If so, it is often from external sources (outside catheter or PIU).
5. Is the noise only present in one catheter or multiple catheters? If it is on multiple catheters, pin box, PIU, amplifier or external sources are the usual suspects.
6. Is the noise present on other equipment (e.g. defibrillator, anesthesia equipment)? If so, noise often comes from these devices. **Figure 2.25** illustrates noise in the EGM of multiple catheters. Because the local ventricular abnormal activity was all obscured by the noise, exhaustive search eventually identified that noise came from a blood warmer operated by the anesthesiology service.

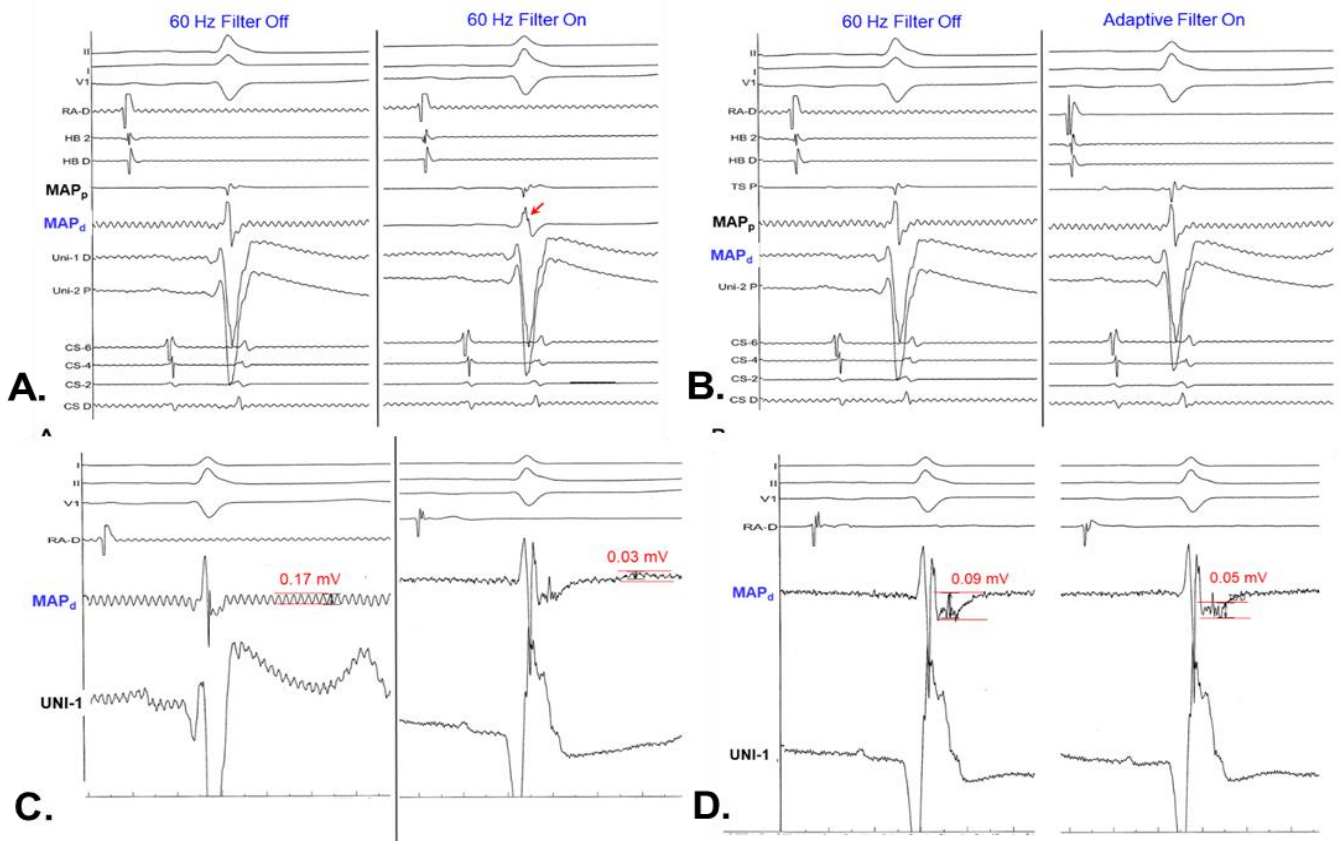


Figure 2.25. Noise introduced by anesthesia blood warmer. A. Left panel: Noise is seen on RA, mapping and CS catheters, indicating that this is not a catheter or connector problem. **Right panel**. 60-Hz notch of the mapping catheter (MAPd) was

turned on. Note that noise on MAPd was gone but new high-frequency components appeared in the EGM (red arrow). **B.** Adaptive filter of MAPd was turned on. Note that the noise persisted. **C. Left panel.** The noise was 0.17 mV. **Right panel**. After the blood warmer was turned off, the baseline noise was 0.03 mV. **D.** Two examples of the voltage amplitude of LAVA in this patient, 0.09 mV (**left panel**), 0.05 mV (**right panel**), much smaller than the amplitude of the noise

2. Surface ECG noise

Baseline drift of ECG is most commonly caused by respiration, motion, bad ECG lead wire or poor patch-skin contact. The latter is the most common cause of ECG noise. In the EP laboratory when the patient is under deep sedation or anesthesia, the last two possibilities are more likely to be the cause of baseline drift. Unless replacing bad cables or patches does not solve the problem, the high-pass filter (usually 0.05 Hz) should not be extended to 0.5 Hz or higher frequencies to remove artifact or noise because inappropriate high-pass filter setting distort the QRS complex, particularly the ST segments that contains low frequency signals (**Figure 2.14, 2.15**).

Precordial leads are unipolar leads, referenced to a virtual point in the chest (Wilson's central terminal). If noise is on a single precordial lead, it is most likely to be caused by a bad patch, bad lead wire or bad connector. Limb leads are bipolar leads, created by the difference between the right arm, left arm and left leg leads. If limb leads are noisy, one should identify the lead that does *not* have noise. Based on how that clean bipolar lead is created, one can pick out the bad lead easily (**Figure 2.26**). If a limb lead is bad, it may introduce more noise than a bad precordial lead because Wilson's central terminal is created by limb leads (**Figure 2.10D**). For the same reason, noise can be horrendous during RF application if a limb lead is bad. The right leg lead in modern ECG recording systems often serves as a noise reference. The computer subtracts the noise of the right leg lead from all other leads. If all ECG leads have the same noise, the right left lead needs to be checked.

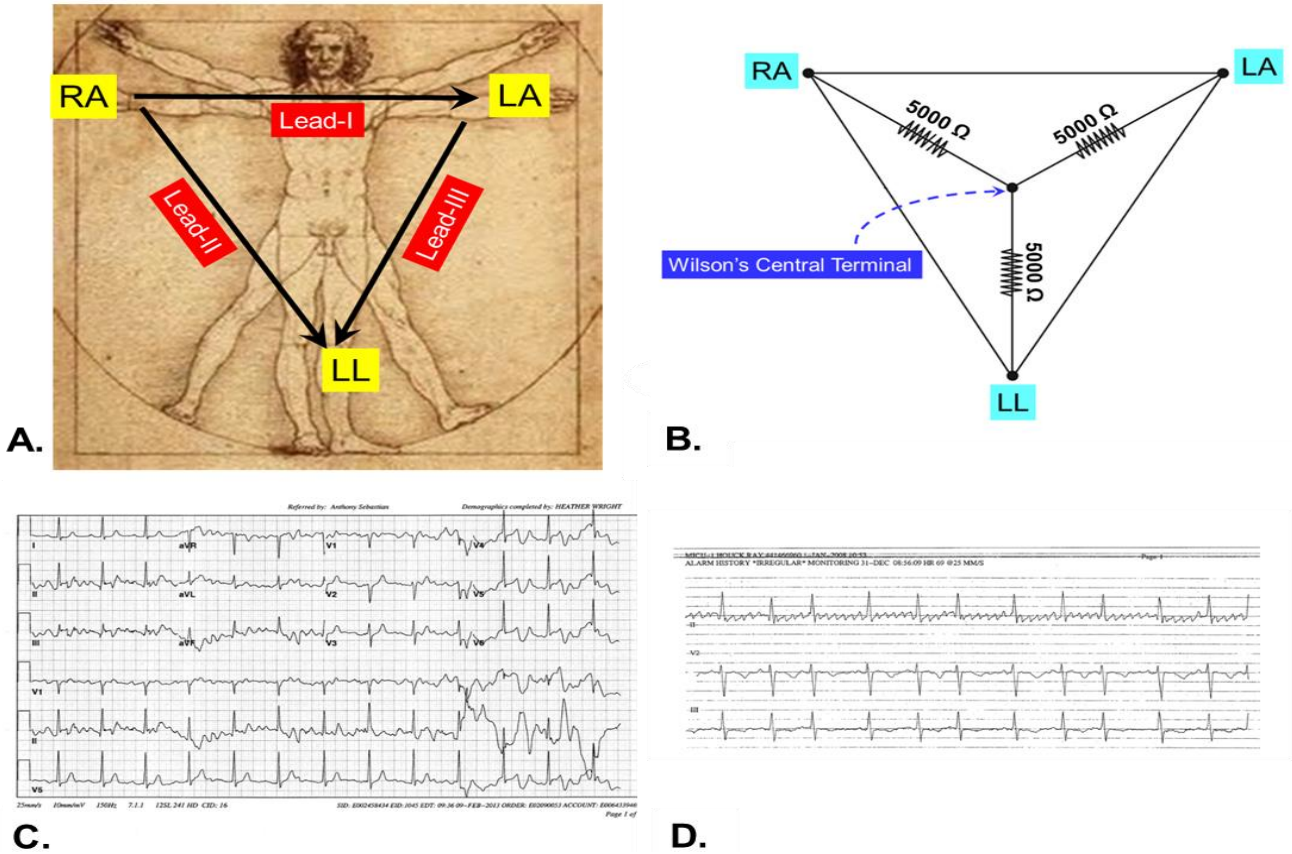


Figure 2.26. Troubleshooting ECG noise. A. Einthoven's triangle, formed by lead I, II and III. **B.** Wilson's central terminal is created by connecting three high impedance resistors to create a virtual site in the chest that has zero potential to serve as the

reference point for unipolar leads such as V1-V6. **C.** Lead I (right arm to left arm) was free of noise; other limb leads were noisy. Precordial leads had noise, too. In this case, the left leg patch or lead wire was loose. **D.** A patient with a hemodialysis shunt on the right arm. Note that lead III (left arm to left leg) was free of noise.

3. Noise during Ablation

Bad ECG leads are often revealed during ablation because RF current returns to the dispersive patch placed on patient's skin. Skin is therefore the shared domain between RF current and ECG signals. Connectors are supposed to have negligible resistance. A bad connector becomes a resistor. A bad ECG patch usually has very high resistance. When a small RF current encounters a very larger resistance, it produces a high-frequency, large voltage artifact seen on ECG. Noise is often seen on the EGM of the tip electrode of the ablation catheter as well (**Figure 2.9B**). This problem underscores the importance of making sure ECG lead wires/patches are in good condition in the beginning of the case. The author has heard plenty of stories about horrendous noise during RF ablation when EP laboratories used low quality ECG patches. Another common source of ECG noise is bad metal clips of the ECG leads that are too loose or rusted.

A bad limb lead connection may introduce more noise during ablation than a bad connection of a precordial lead (**Figure 2.27**). The voltage of a virtual point (Wilson central terminal) is created by connecting 3 large resistors (≥5000Ω) from the right arm, left arm and left leg to create a reference point, which often is used as the reference point for unipolar EGM by various recording or mapping systems. A bad left arm patch can mess up the Wilson's central terminal; any EGM that uses Wilson's central terminal as the reference point can show horrendous noise. During ablation, when RF current flows through the bad, high resistance limb lead, noise is amplified. Similarly, a defect in the dispersive patch (ground pad) of the ablation circuitry can introduce horrendous noise to the ECG during ablation (**Figure 2.28**).

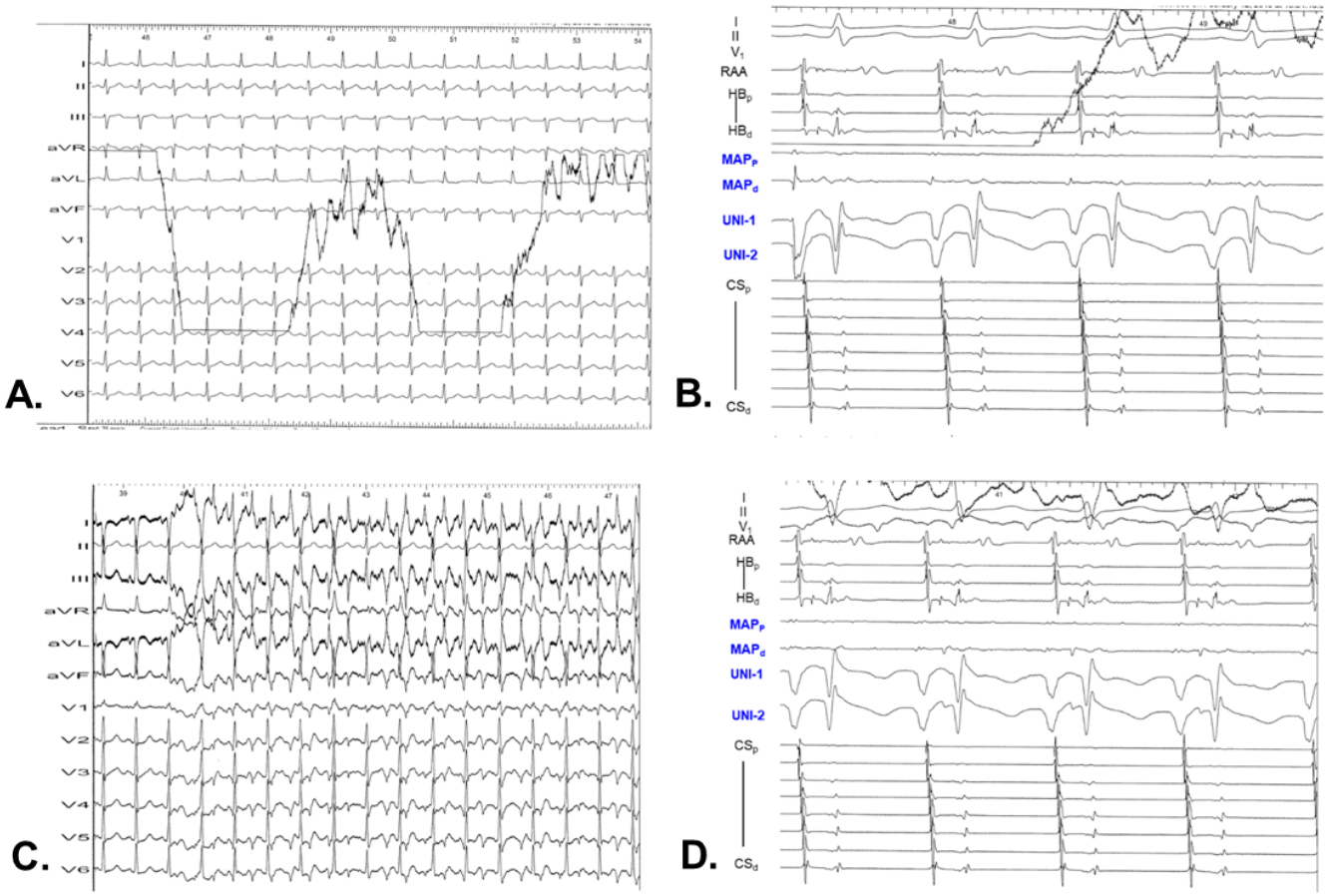


Figure 2.27. A test RF application (10 watts, 5 seconds) in blood pool. A-B. Ablation with the lead V1 patch disconnected. Note that the surface ECG was noisy only in the V1 lead. **C-D.** Ablation with the left arm patch disconnected. Note that the

surface ECG was free of noise only in lead II (right arm to left leg). The precordial leads had noise as they were referenced to the Wilson's central terminal. The CARTO unipolar EGM was very noisy (not shown here) but the unipolar EGMs displayed on the Bard recording system were clean because the unipolar reference was the 5th electrode on the RA catheter in the IVC. Noise in the Wilson's terminal caused by disconnecting the left arm patch did not affect any bipolar intracardiac EGMs because they were not referenced to the Wilson's terminal.

In the CARTO-3 PIU, a circuit board named "ablation relay card" controls both the ablation and pacing function. A defect in this circuit board can lead to noise during ablation if any intracardiac electrode pair is set to pace (**Figure 2.29**). If the electrode pair is set for pacing, some stimulators may inject a very small amount of current into the electrode pair in preparation for pacing even though pacing is not delivered. If defects in the circuit board are present, noise during ablation may be observed.

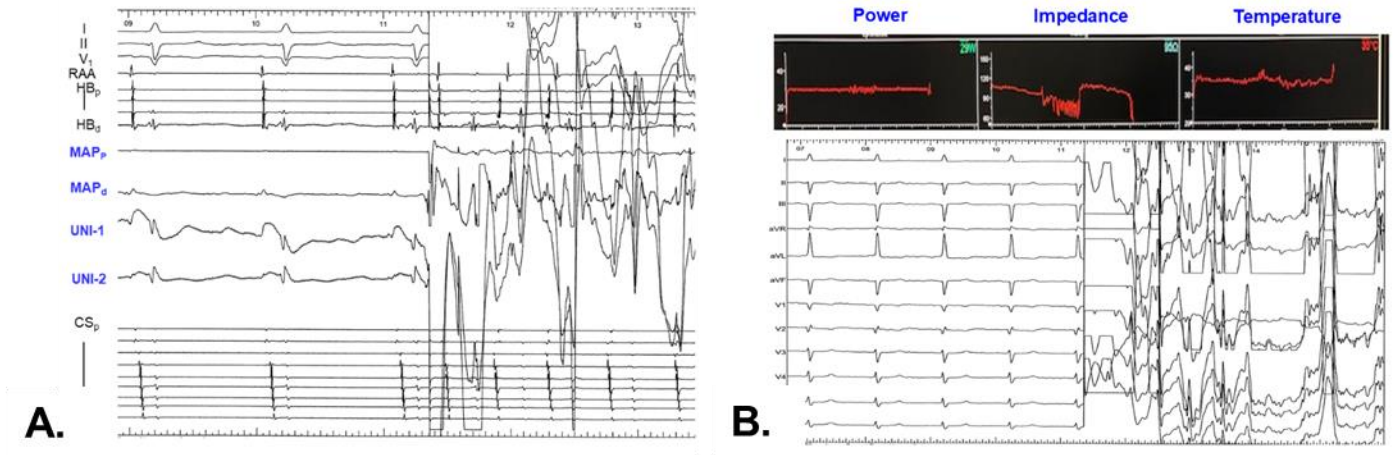


Figure 2.28. Noise caused by a bad ablation ground pad. A. Noise on ECG appeared immediately after RF application began**.** Note that noise appeared on surface ECG and ablation catheter, not on any other diagnostic catheters. **B. Top: Display of Bard ablation graph.** Note erratic changes in impedance. These observations should prompt the operator to look for problems in the ground pad. After the bad ground pad was replaced, the noise was gone.

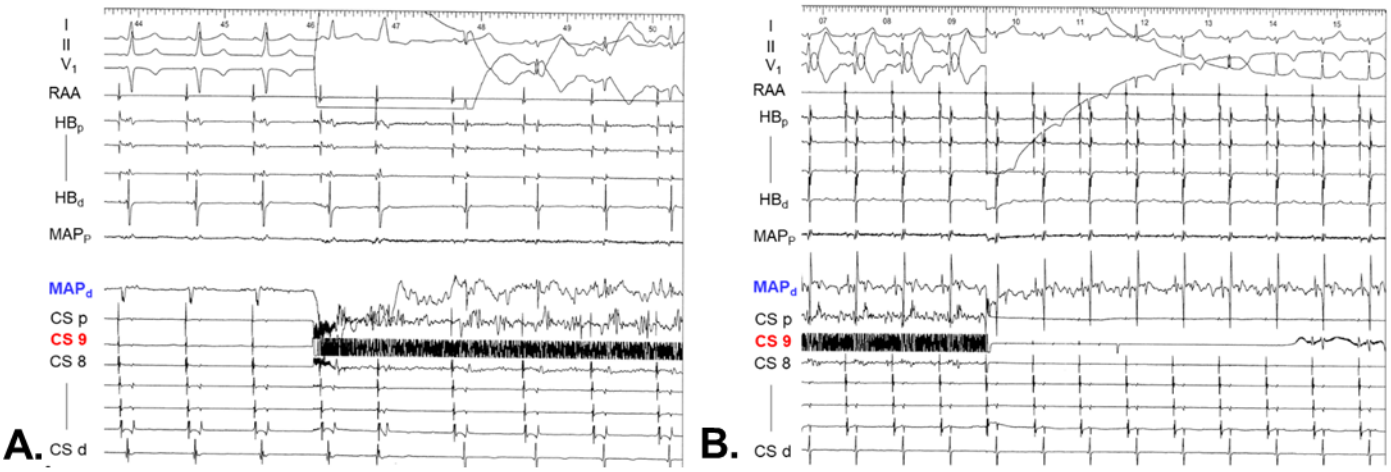


Figure 2.29. Noise introduced by the bipolar lead (CS-9) that was set to pace. A. Note that as soon as RF application began, high frequency noise appeared on CS-9, CS-10 and mapping catheter. Surface ECG started drifting as well. **B.** Noise disappeared soon after the pacing assignment to CS-9 was deleted. In the CARTO-3 PIU, a circuit board named "ablation relay card" controls both the ablation and pacing function. There was a defect in the PIU, leading to this problem.

4. Intracardiac EGM noise

The clinical engineer of the OU-EP laboratory, Mr. Thomas Hayes, uses a "*cut in half*" strategy. When noise appears, one evaluates all the possible causes of noise and divides them in half. After determining which half the noise comes from, that half is then divided by half. This process repeats itself until the noise source is found. **Figure 2.30** illustrates this "*cut in half*" strategy. Because all the signal cables in the OU-EP laboratories are routed to the CARTO-3 PIU first, if noise appears on both CARTO-3 and Bard, noise must be introduced before the signals in the CARTO-3 PIU are split to Bard. Although a bad circuit board in the CARTO-3 PIU is possible, the most likely culprit is the connecting cable because it is usually re-sterilized multiple times. A bad catheter or loose connection between the catheter cable and CARTO-3 pin box or PIU (**Figure 2.30A**) need to be checked as well. If the CARTO-3 signals are clean, the first site that noise can be introduced is the jumper cables between the CARTO-3 and Bard pin box. If the pins of a catheter connector are directed plugged into the Bard pin box without being routed to the CARTO-3 PIU first, the "*cut in half*" strategy can be implemented to determine if the pins are bad or the pin box is bad. In **Figure 2.30**, HB_d to HB_3 were free of noise; therefore, pin 7 to pin 12 and the corresponding slots in the pin box must be good. One can plug pin 11 and 12 (good pins) into the pin box slots for pin 13 and 14. If there is no noise on HBp bipole, the slots (13 and 14) in the pin box are good. Then, one can make a new bipole with pins 12-13 and pins 12-14. If the bipole 12-14 is noisy, pin 14 must be a bad one. In **Figure 2.30B**, both pin 13 and 14 were bad, indicating either a bad connector or a bad catheter. A new connector solved the problem.

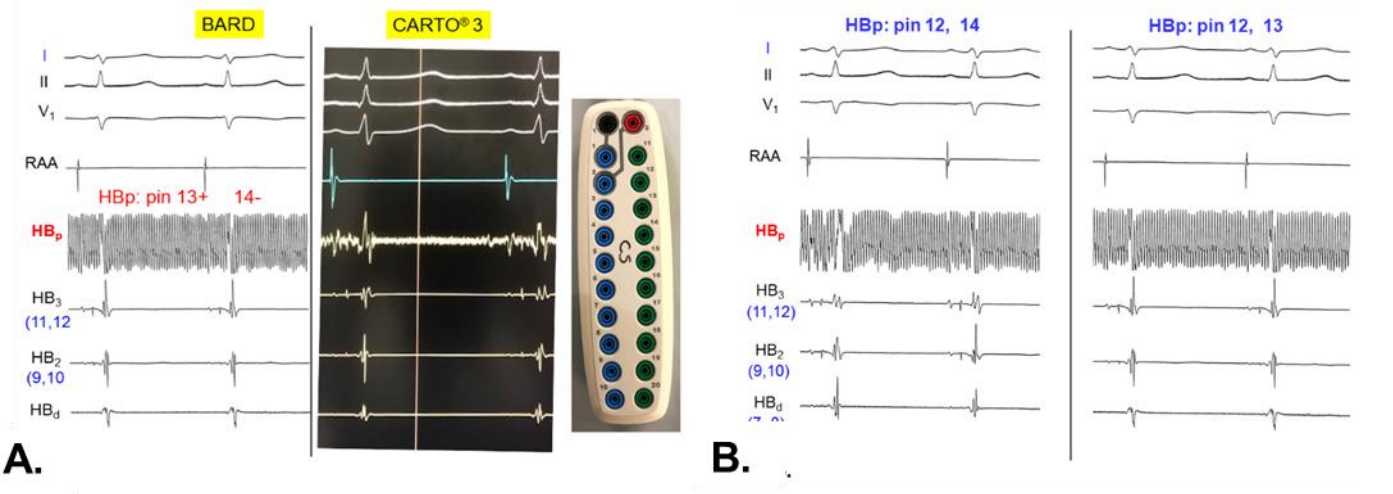


Figure 2.30. The "cut in half" strategy. A. HB-P is a bipolar lead made of pin 14 (negative), 13 (positive). The same high frequency noise was seen on both the Bard recordings (**left panel**) and CARTO-3 recordings (**middle panel**), indicating that noise may come from (1) a bad catheter, (2) a bad connector, (3) CARTO pin box (**right panel**) or (4) a bad circuit board in the CARTO-3 PIU. In this case, it was a bad catheter cable. **B.** To demonstrate the "cut in half" approach, the HB catheter was connected directly to the Bard pin box. HB_d to HB_3 were free of noise; therefore, pin 7 to pin 12 and the corresponding slots in the pin box must be good. There was no noise after plugging pin 11 and 12 (good pins) into the pin box slots for pin 13 and 14 (not shown here); therefore, the slots in the pin box for pin 13 and 14 must be good. Then, new bipolar electrodes with pins 12-13 and pins 12-14 were made. Note that noise continued when the HB-p bipole was assigned to either 12-14 or 12-13, indicating that both pins 13 and 14 were bad as result of a bad catheter or a bad connecting cable.

Figure 2.31 illustrates another example of this "*cut in half*" strategy. The noise on the unipolar EGM of the mapping catheter only appeared on the Bard recording system, not the CARTO-3, indicating that the source of noise is downstream from the CARTO-3 PIU. In the Bard recording system, the unipolar EGM was referenced to the 6^{th} electrode of the RA catheter, which was in the inferior vena cava. After referencing the unipolar EGM to the 5^{th} electrode of the RA catheter, noise disappeared, indicating a bad electrode of the RA catheter. **Figure 2.32 and Figure 2.33** illustrate other example of this "cut in half" strategy.

Occasionally, the ablation catheter appears to move out of position as soon as RF application starts but the operator is sure that catheter has not moved based on fluoroscopic images. If this problem becomes recurrent, it most likely results from defective ground pad (dispersive patch). Operators can replace the ground

pad and deliver a 5-second RF application in the blood pool to see if the catheter still moves out of position before replacing an expensive ablation catheter.

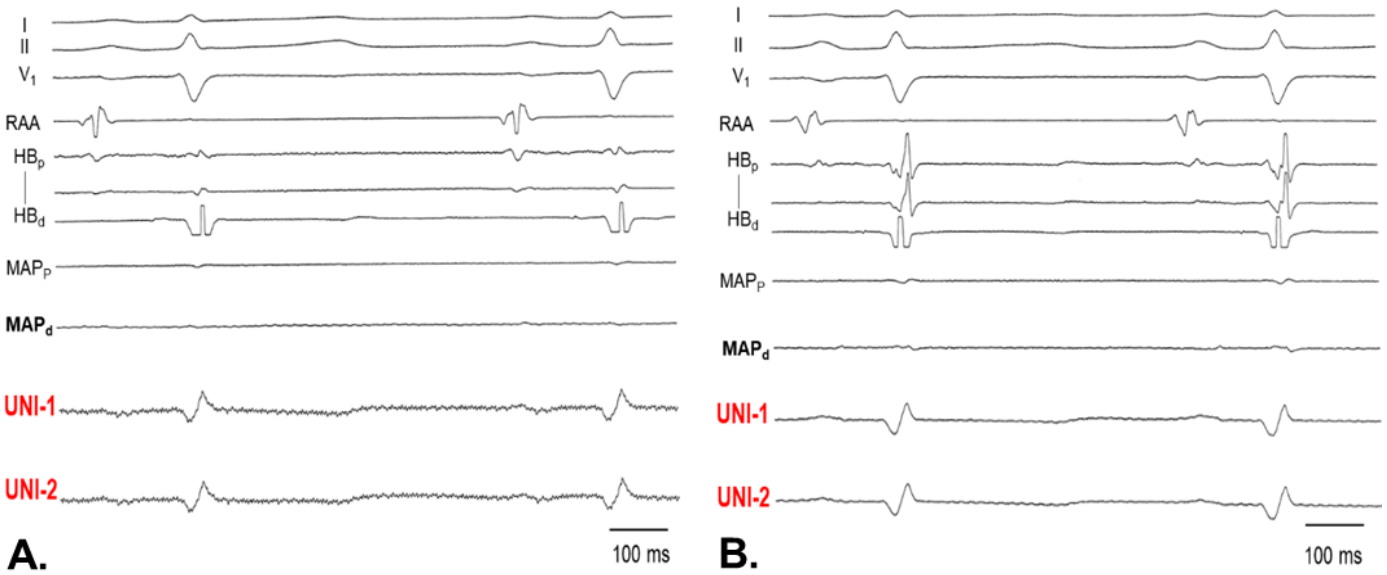


Figure 2.31. Noise only seen on unipolar EGM of the ablation catheter. A. CARTO did not show similar unipolar noise, indicating that noise did not come from the mapping catheter, connecting cable or the CARTO PIU. In the Bard recording system, the unipolar EGM was referenced to the 6th electrode of the RA catheter, which was in the inferior vena cava. **B.** After referencing the unipolar EGM to the 5th electrode of the RA catheter, noise disappeared, indicating that the 6th electrode on the RA catheter was bad.

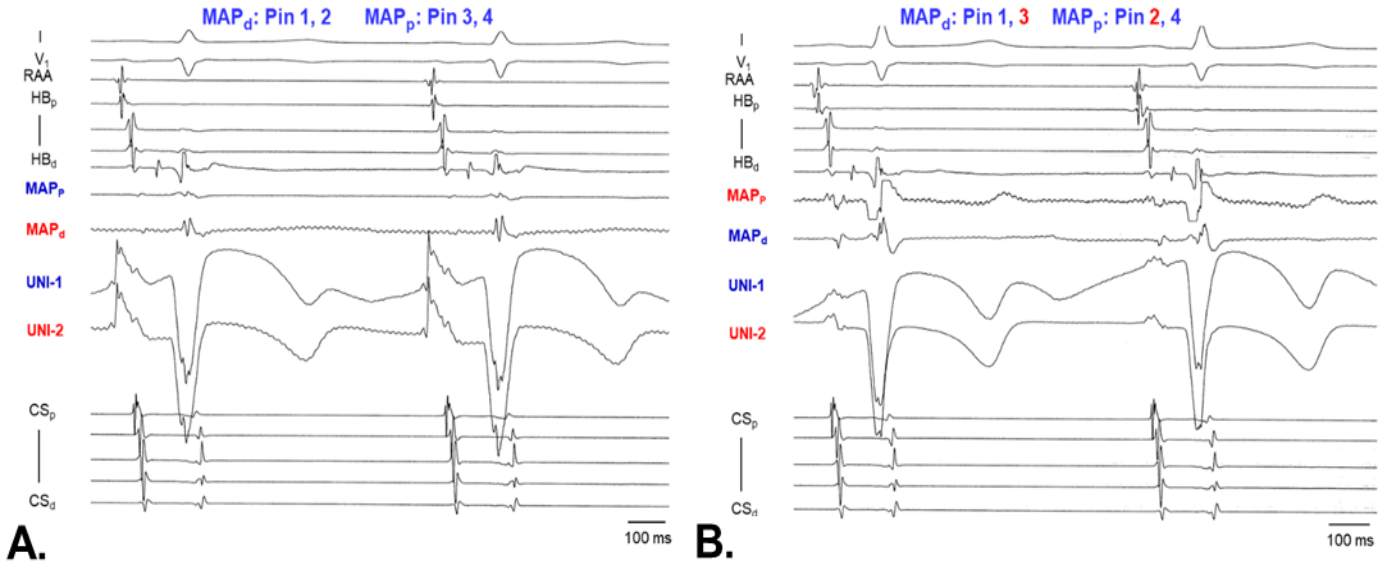


Figure 2.32. A. Noise on the distal electrode pair (MAPd) as well as the proximal unipolar electrode (UNI-2). CARTO did not have the same noise, indicating that it was not a catheter, connector or CARTO pin box problem. Noise was introduced *after* signals were split to Bard**.** It also indicates that the slots in the pin box for pin 3 and pin 4 must be good. **B.** The 2nd pin of the jumper cable from CARTO to the BARD pin box was plugged into the slot for the 3rd pin. The 3rd pin was plugged into the slot for the 2nd pin. Note that the noise is now on the proximal pair of ablation catheter. Both unipolar EGMs were free of noise. This observation indicates that the pin box was good but the 2nd pin of the jumper cable connecting CARTO and Bard was bad. If the pin box was bad, no matter which pin was plugged into the slot for the 2nd pin, the EGM of UNI-2 would be noisy.

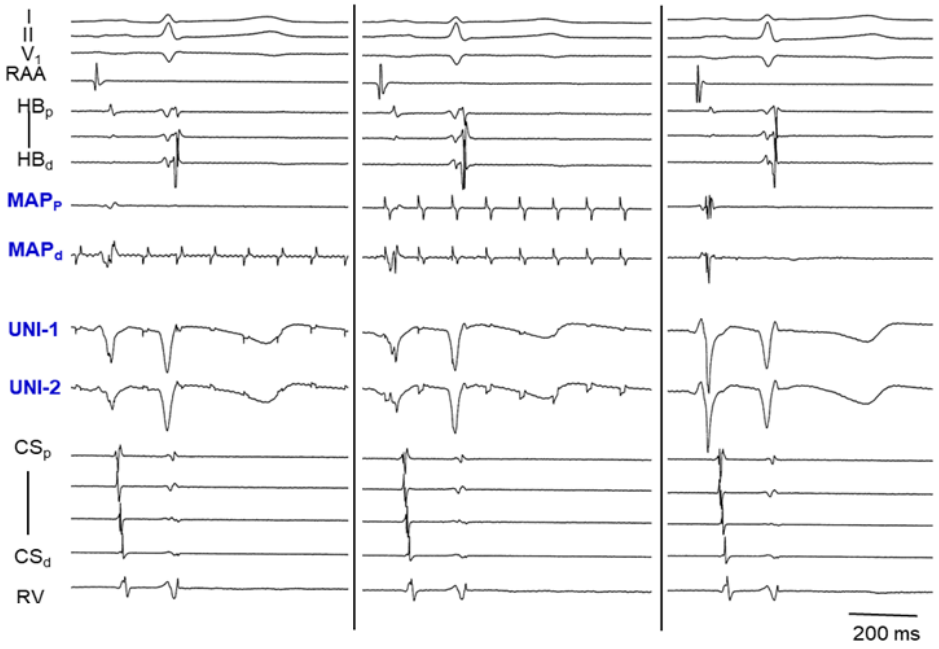


Figure 2.33. Troubleshooting. Left. 10 Hz noise was seen on the mapping catheter. CARTO showed the same noise, indicating that noise was introduced before the intracardiac signals were split between Bard and CARTO. Thus, the most likely culprit was catheter and the connecting cable. **Middle.** Noise remained (or appeared to be worse) after the connecting cable was replaced, indicating that a bad mapping catheter was the most likely culprit. **Right**. Noise disappeared after the mapping catheter was replaced.

The protocol listed below was written by Mr. Thomas Hayes, the clinical engineer of the OU-EP laboratory, as a step-by-step instruction to the nurses and technologists of the OU-EP laboratory.

1. Patient Prep:

A. Prep patient skin for surface lead connection. Excessive use of alcohol may dry the skin too much and lead to higher skin impedance.
B. After connections to patient are accomplished, the excess cable should be rolled up and taped to table top. Do not allow to hang below table top.
C. Check surface ECGS with the notch filter off, and troubleshoot noise as necessary. Most noise at this point is caused by fractured surface leads, or stress on cables. Keep patient warmers and power cables from these devices away from surface and Intracardiac cables. When signals are as clean as possible, then you may turn notch filters on.
D. Connect recording system first! Noise can be induced by bad surface lead cables from anesthesia carts and defibrillation units. If you start with a clean recording system signals, it is easier to tell where noise comes from when it appears.

2. Set up and Prepare cables:

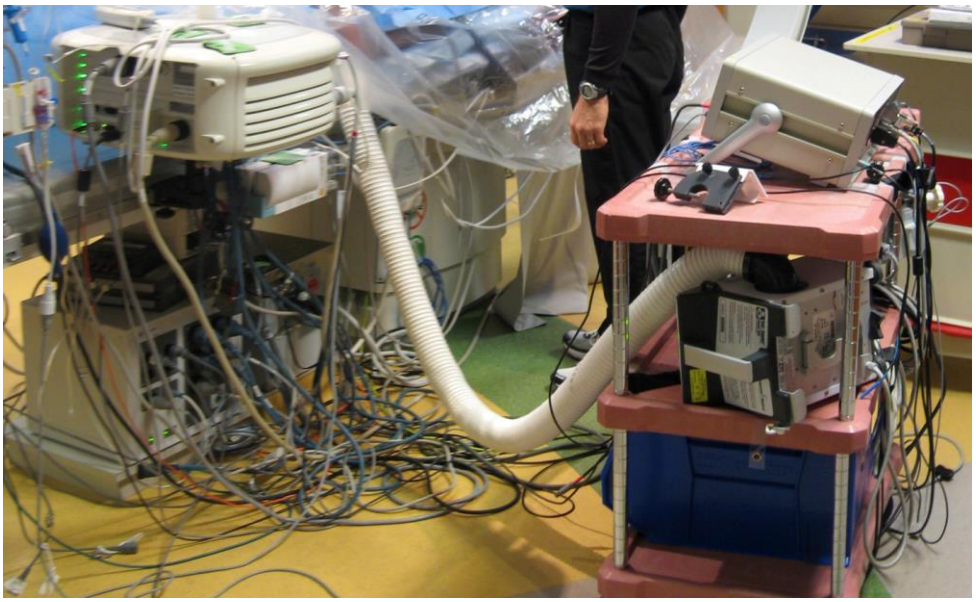
A. Prepare intracardiac interface cables. These should be routed down the table top to the foot end of the table. These should connect to the amplifier without touching the floor, and separated from any power or RF cables.
B. Prepare the surface lead ECG cable for connection to the patient by coiling up excess trunk cable and taping to table top. This prevents cable from falling to floor and being induced with noise.
C. When using CARTO, ensure the ECG cable from CARTO to the recording system follows same route as intracardiac cables, staying on top of table and exiting at the foot end. Also, keep this cable off the floor.
D. Ensure RF interface cable from ablation generator and ground patch cable are routed off the floor and away from all other cables. Keep away from Intracardiac and surface lead cables as well.
E. Check that power cable routing from recording system amplifier, and mapping systems, are routed away from intracardiac and surface ECG cables.

F. Remove any “jumper” connections that are not being used. NEVER leave one end of a jumper connected and one end hanging. Remove completely when not in use.
G. Use care when routing cables near monitor screens.

3. Intracardiac Noise:

A. Troubleshooting the intracardiac signals is much easier when you start with clean surface signals. If you have excessive noise, recheck surface leads first.
B. Check interface cables. Often re-sterilized cables can become stressed.
C. Check peripheral equipment again, ensure that nothing was moved closer to Intracardiac or surface lead cables.
D. If no other problems are found and noise is on a specific catheter, then there is a good chance that the connector or catheter is bad. Even new catheters can be bad!
E. If noise is on multiple catheters, there is probably a bad connection at the pin box or amplifier. Disconnect and reconnect.
F. If noise is on mapping catheter bipolar or unipolar signals, check peripheral equipment for proximity to RF cables, and ensure RF cable is not on the floor.
G. Check unipolar reference interface. If using CARTO, ensure that unipolar reference is also going thru Carto. For example, in the OU-EP laboratory, the unipolar reference is the 5th or 6th electrode on the RA catheter in the IVC. All six pins of the RA catheter go thru CARTO then back out to the Bard recording system.
H. Moisture in catheter connectors cause noise. Usually, replacement of interface connector will alleviate the problem.
I. 60 Hz noise is usually caused by bad connections, or power cables too close to signal cables. Higher frequency can be caused by interference from peripheral equipment, such as patient warmers or x-ray tubes. Often, use of a Tri-field meter can help identify such noise sources.
J. Finally, patients that are awake often have more noise due to movement, muscle twitch, and sweating, all of which cause patches and cables to become loose

The following picture was sent to me by a friend asking for advices to reduce the noise in his EP laboratory. Please take a close look at the picture and see what you would do differently.



Chapter 3:

Biophysics of Radiofrequency Ablation

It is a truth universally acknowledged that ablation lesion formation must follow the law of physics. For radiofrequency (RF) lesions, the tissue temperature has to reach approximately 50°C to cause necrosis. The size of an RF lesion is determined by the following factors:

1. Power delivered to the tissue (not the power displayed on the RF generator)
2. Tissue temperature (not the temperature displayed on the RF generator)
3. Impedance at the electrode-tissue interface (not the impedance displayed on the RF generator)
4. Cooling of the ablation electrode by either blood flow or irrigation
5. Size of the ablation electrode (bigger electrodes do *not* make bigger lesions!)
6. Duration of RF application
7. Contact force

At first, we should know the differences between power-controlled mode and temperature-controlled mode. Basically, the former assumes that higher power creates larger lesions, while the latter assumes that higher electrode temperature creates larger lesions. Both assumptions are correct and are based on preclinical and clinical biophysical studies. EP fellows often ask the following question: are ThermoCool catheters operating in a temperature-controlled mode or power-controlled mode? The correct answer is that ThermoCool catheters operate in a temperature-limited (e.g. 43°C), power-controlled mode. The reason to set the maximal temperature at 43°C is to reduce the incidence of thrombus formation at the electrode-tissue interface.

Temperature-controlled mode was pioneered by David Haines and others when catheter ablation was still in its infancy. They found that electrode temperature correlates with RF lesion size. Later studies by others showed that ablation lesion size correlates better with RF power than electrode temperature, establishing the biophysical basis of the power-controlled mode. When RF current encounters tissue impedance, it generates heat. In theory, RF current flowing into the targeted tissue should provide the best correlation with RF lesion size. However, current-controlled mode is not available at this stage.

Basics of RF Energy

In RF ablation, the RF energy is produced by alternating current in the radiofrequency range, typically around 500 kHz. Theoretically, frequencies significantly lower than 500 kHz may stimulate myocardium and nerves, causing arrhythmia or discomfort, while energy transfer to cardiac tissue is less predictable if frequencies are significantly higher than 500k Hz.

Electric power (in watts) equals voltage (in volts) times current (in amperes). Based on Ohm's law (voltage = current x impedance; V = I x R), the electric power equation can be presented as "$P = I \times V$", "$P = I^2 \times R$" or "$P = V^2/R$". RF lesion size is proportional to the power (or current) delivered to the target tissue. For most RF generators, increasing power delivery is achieved by increasing voltage. RF current does not penetrate deep into the tissue; the site with the highest current density is always at the electrode-tissue interface. Based on Ohm's law, tissue impedance changes during ablation have a large impact on the power ($P = V^2/R$) delivered to the tissue. If impedance does not change, current and voltage will be constant as well ($V = I \times R$); however, this is not observed clinically. At the beginning of an RF application, tissue impedance decreases quickly by 10-15% due to tissue injury. In power-controlled mode, if tissue impedance decreases, voltage will decrease ($P = V^2/R$) to maintain the same power level. In temperature-controlled mode, if impedance changes, power will change accordingly to maintain the target electrode temperature.

Resistive heating vs. conductive heating

The ablation electrode passes RF current to the target tissue. When RF current encounters tissue resistance, it generates heat, known as *resistive heating*. (**Figure 3.1**). Heat is transferred back to the ablation electrode as a temperature rise displayed on the RF generator. Heat can be transferred by 3 methods: (1) radiation, which takes place in the form of electromagnetic waves mainly in the infrared region (negligible in RF ablation), (2) convection, which is a transfer of heat through moving fluids (in ablation, this is called *convective cooling* in which heat is lost to the blood pool or saline irrigation) and (3) conduction, which allows heat to dissipate throughout a material that is in contact with the hot material. In ablation, this is called *conductive heating*. Both conductive and resistive heating, as well as the effects of convective cooling, are important determinants of RF lesion growth.

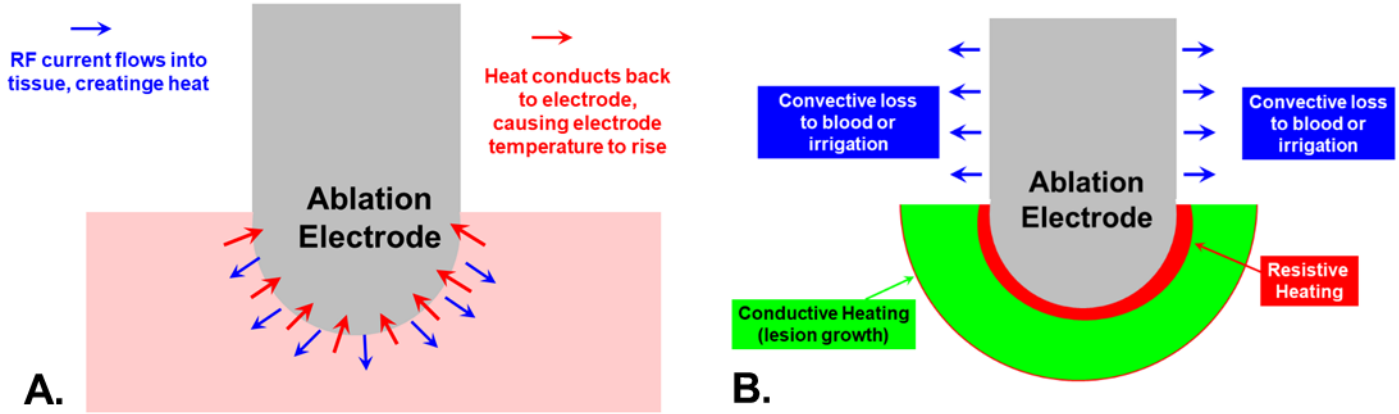


Figure 3.1. Tissue heating. A. When RF current flows into tissue, it encounters a resistance medium (tissue) and generates heat (resistive heating) at the electrode-tissue interface. Heat is transferred to adjacent tissue (conductive heating). Heat is also transferred back to the ablation electrode, causing the electrode temperature to rise. **B.** In most RF lesions, only a small rim of tissue is injured by resistive heating. Lesion growth mainly depends on conductive heating.

Resistive heating, also known as Joule heating or Ohmic heating, occurs when electric current passes through a resistor, converting electrical energy into heat energy. In RF ablation, the resistor is the targeted cardiac tissue. Conductive heating subsequently occurs because of the temperature gradient between heated tissue in direct contact with the ablation electrode and the relatively cooler surrounding tissue. RF lesion formation is therefore determined by direct resistive heating of tissue in contact with the ablation electrode and by conductive heating, in which heat is slowly transferred to adjacent tissue. The former occurs immediately after RF current is delivered; the latter can take up to >1 minute to complete. The magnitude of tissue heating is a function of power density; power density is a function of the square of current density (**Figure 3.2A**). As RF current passes from the tip ablation electrode to the indifferent electrode (dispersive patch or ground pad), the great difference in surface area between the tip electrode (0.23 mm in diameter for a 7 Fr. catheter) and dispersive patch (>50 cm^2) renders the tip electrode the site with the highest current density in the entire circuitry. RF current density decreases in proportion to the square of the distance from the center of the tip ablation electrode. Since RF power density is a function of the square of current density, RF power density decreases in proportion to the fourth power of the distance from the center of the tip ablation electrode (**Figure 3.2A**). That is to say, tissue heating decreases as a function of $1/r^4$ through conductive heating.

In the presence of good electrode-tissue contact, the first 10-20 seconds of RF application play a critical role in RF lesion formation (**Figure 3.2B**). After 20 seconds, the RF lesion continues to grow but at a slower pace; after 30-40 seconds, the increment is even smaller. While the curve shown in **Figure 3.2B** would be different if a different ablation catheter (e.g. irrigated vs. non-irrigated) is used, the principle that the first 20 seconds of RF application plays a critical role in RF lesion formation holds true. Importantly, the rate of heat transfer (both resisting and conductive heating) is not a temperature-dependent process. This essentially means that with higher heat source temperature (e.g. a 50-watt RF application), heat transfer to surrounding tissue is not faster than that with a lower heat source temperature (e.g. a 20-watt RF application). Lesion

growth of RF ablation is not faster if higher RF power is delivered. Higher RF power provides a higher temperature (a better heat source) to allow heat to be transferred to a greater distance into the tissue.

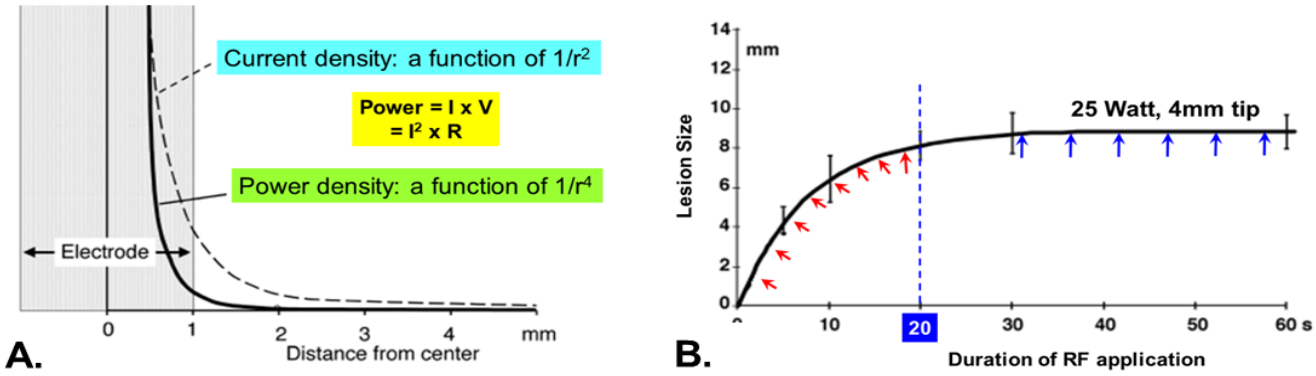


Figure 3.2. A. Current and power density. Current density is inversely proportional to r^2 (r: distance to the center of the ablation electrode). Because power is proportional to the square of current, power density is inversely proportional to r^4. **B.** In the presence of good electrode-tissue contact, RF lesions grow at the fastest pace in the first 10-20 seconds (red arrows). After 30-40 seconds, lesion size continues to grow but with a smaller increment (blue arrows). How fast lesion growth reaches the plateau depends on many factors including power, contact force, irrigation rate and electrode size. *Modified with permission from: Wittkampf FH, Nakagawa H. Pacing Clin Electrophysiol. 2006 Nov;29(11):1285-97.*

Thermal latency

This important phenomenon is caused by the different time course of resistive heating and conductive heating. After termination of RF application, resistive heating stops immediately, but heat continues to be dissipated to surrounding tissue through conductive heating until steady-state isotherm is reached (**Figure 3.3**). An important clinical implication of thermal latency is that an RF lesion grows at its fastest pace in the first 10-20 seconds (**Figure 3.2B**). Specifically, tissue temperature grows exponentially in the first 10-20 seconds. If the electrode-tissue interface temperature is high enough (a high source temperature), a 5-second RF application can lead to significant lesion growth *after* ablation is terminated.

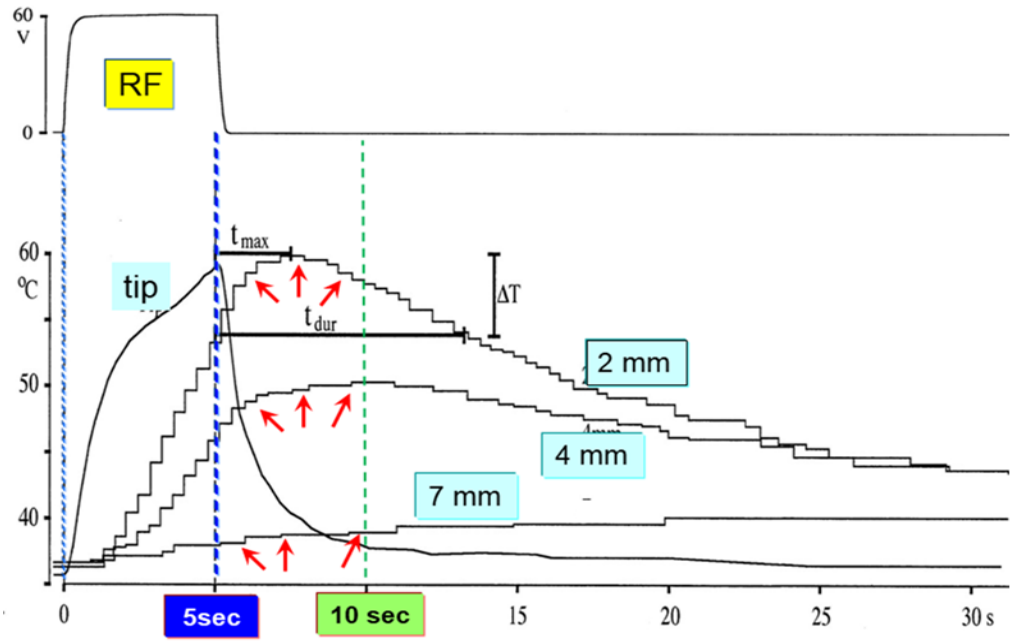


Figure 3.3. Thermal latency. A 5-second RF application was delivered. Temperature was measured at the electrode tip as well as 2, 4 and 7 mm beneath the electrode-tissue interface. Note that tissue temperature at 2, 4 and 7 mm depth continued to grow in the first 5 seconds (red arrows) *after* the RF application had been terminated (vertical blue line). *Modified with permission from: Wittkampf FH, Nakagawa H. Circulation. 1996 Mar 15;93(6):1083-6.*

As an example, if one is ablating a para-hisian PVC, s/he may want to start with a lower power so that the interface temperature is not too high (a moderate heat source) to limit the extent of continuous RF lesion

growth if ablation has to be prematurely terminated due to evidence of AVN injury. The negative impact of thermal latency underscores the importance of selecting the ablation target wisely and titrating the RF power when the ablation target is in the vicinity of the AVN. Moreover, operators should be mindful that during the first 5-10 seconds of RF application, when the RF lesion grows rapidly, there is only a limited number of heart beats for the operator to recognize AH interval prolongation. Thermal latency may lead to permanent AVN injury despite immediate termination of the RF application when the AH interval begins to prolong. That is to say, to avoid the negative impact of thermal latency resulting from the first few seconds of ablation, selecting the ablation target wisely is a much better strategy than applying “test burns”.

Circuitry of RF ablation

RF current flows between the distal ablation electrode at the tissue-electrode interface and the indifferent ground electrode (dispersive patch) on the skin. The circuitry is illustrated in **Figure 3.4A**. The impedance value displayed on the RF generator represents the sum of impedance of the entire circuitry, including the internal impedance of the RF generator itself. For a 4mm-tip ablation electrode, the total impedance of the entire circuitry is approximately 100 Ω, one third of which (30-35Ω) is contributed by tissue outside the heart and circuit elements outside the body. That is to say, approximately 1/3 of the RF power is lost (not used for creating RF lesions). The rest of the impedance (65-70Ω) is contributed mainly by the ablation electrode (1) where the electrode touches myocardium (electrode-tissue interface) and (2) where it is exposed to the blood pool. The impedance of the former is approximately twice the magnitude of the latter, which has a profound impact on the overall impedance displayed on the RF generator as well as RF lesion formation. One can view the ablation electrode as two resistors in parallel that function as a current divider. If the impedance of the tip electrode exposed to the blood pool is further reduced by a longer tip electrode (e.g. 8mm), more RF current will be shunted to the blood pool, thereby having a negative impact on RF lesion formation.

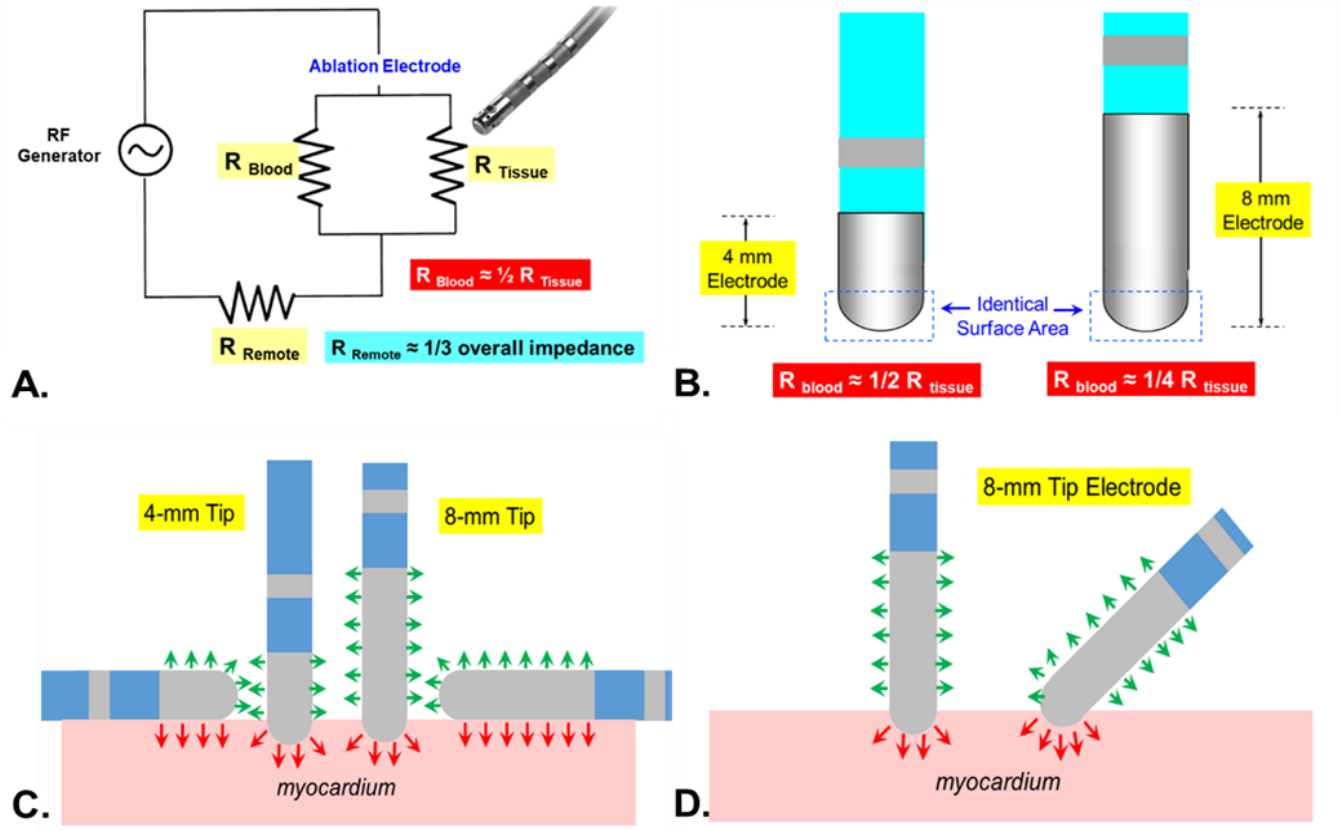


Figure 3.4. Impedance of the RF ablation circuitry. A. The total impedance of an RF ablation circuitry can be viewed as ablation electrode impedance and non-ablation electrode impedance (R_{Remote}) connected *in series*. The impedance of ablation electrode consists of electrode-tissue interface impedance (R_{Tissue}) and electrode-blood interface impedance (R_{Blood}) connected *in parallel*. R_{remote} consists of the impedance of the skin patches, cables and tissue outside the heart. **B**. If the catheter is positioned perpendicular to the tissue, the surface area of tissue-electrode interface is identical between a 7 Fr. 4mm-tip and a 7Fr. 8mm-tip electrode. However, the surface area exposed to the blood pool is about twice larger for the 8mm-tip catheter, leading to a substantially smaller contribution of the electrode-tissue interface impedance to the total impedance. **C.** The longer the tip electrode, the larger the impact on RF lesion formation that electrode-tissue orientation makes. If one can lay an 8mm-tip electrode in parallel with the tissue, it may make a bigger RF lesion than that of a 4mmi-tip electrode. However, positioning a stiff 8mm-tip electrode catheter in parallel with the tissue can be difficult. The electrode-tissue orientations shown in (**D**) are the most common orientation when an 8mm-tip electrode catheter is used.

Impedance

As noted above, while impedance from the electrode-tissue interface is most relevant to RF lesion formation, the interface impedance only represents a portion of the total impedance displayed on the RF generator. For a 4-mm electrode, the rounded distal end of the electrode accounts for approximately 25% of the total surface area of the tip electrode. This has important clinical implications because a large impedance change at the electrode-tissue interface may or may not be reflected by the total impedance displayed on the RF generator. Moreover, the longer the ablation electrode (e.g. 8-mm tip), the smaller the percent of the overall impedance that is contributed by the electrode-tissue interface because a greater percentage of the ablation electrode's surface area is now exposed to the blood pool (**Figure 3.4B**). The length of the ablation electrode has the most impact on total impedance and RF current shunting to the blood pool when the ablation electrode is perpendicular to the myocardium (**Figure 3.4C).** A larger electrode results in greater current shunting to the blood pool. The ineffective current has to pass through ineffective impedance (R_{remote}). Therefore, a larger proportion of the total RF voltage is lost to R_{remote}, decreasing the voltage available for tissue heating.

If the electrode-tissue contact is good, impedance usually drops by approximately 10-15% in the first 5-10 seconds of RF applications as a result of tissue injury, which lowers the tissue impedance. A rapid, large magnitude impedance drop may suggest tissue injury occurring too rapidly, and may be a prelude to perforation. As described above, the electrode-tissue interface impedance only accounts for a small fraction of the total impedance displayed on the RF generator. Thus, in the presence of good contact force, absence of a significant impedance drop should *not* be interpreted as not making effective lesions. In the presence of good contact force, it is not advisable that operators increase the power or RF duration simply because an initial impedance drop was not observed.

A *sudden* increase in impedance during RF application is often interpreted as thrombus or char formation at the electrode-tissue interface because the impedance of desiccated tissue (e.g. char) is much higher than that of myocardium. Intracardiac echo often shows that the sudden appearance of micro- or macro-bubbles as a result of rapid evaporation of the water content in tissue (tissue boiling) is the cause of a *sudden* rise in impedance, because air bubbles are good electrical insulators. For a 4-mm electrode catheter to show a 10Ω *sudden* impedance rise, there must be a large enough amount of bubbles to insulate the ablation electrode. A moderate amount of bubbles may not increase the impedance at all. Micro-bubbles, macro-bubbles and steam pops basically represent tissue boiling in ascending orders.

Frequently, a thrombus or char is formed at the electrode-tissue interface but is accompanied by a minimal or no change in impedance. For example, when the electrode-tissue interface temperature reaches 75^{o}C, thrombus may begin to form as a result of denatured blood protein before tissue boiling. Therefore, thrombus formation at the electrode-tissue interface occurs much earlier than impedance rise shown on the RF generator. Thus, monitoring impedance rise *cannot* predict thrombus formation. Additionally, absence of an impedance rise does not ensure that tissue overheating has not occurred. If impedance falls initially but *gradually* rises during RF application, it is advisable to terminate the RF application as slow impedance rises like this often herald the beginning of tissue desiccation. If this problem becomes recurrent, the tip electrode of the ablation catheter should be inspected for char formation. Importantly, when an 8-mm electrode ablation

catheter is used, there is rarely an impedance increase when a thrombus is formed at the electrode-tissue interface due to its large surface area exposed to the blood pool; the electrode-tissue interface impedance makes a much smaller contribution to total impedance.

1. Impedance fluctuation

During inspiration, the lungs are filled with air (an electrical insulator), leading to a small increase in the total impedance. For the same reason, the impedance is usually higher if the ablation electrode falls into a pulmonary vein. With deep inspiration, the impedance reading of an ablation catheter may fluctuate significantly, making it difficult to detect small impedance changes during RF application. If the tip electrode of the ablation catheter falls into a trabeculated area, the surface area of electrode-tissue interface is markedly increased and the surface area exposed to the blood pool is markedly reduced, leading to a higher total impedance. By contrast, if the tip electrode is “standing” on top of the trabeculated area, the total impedance may be lower due to poor electrode-tissue contact and more surface area exposed to the blood pool.

Occasionally, operators may notice that, regardless of the magnitude of contact force and where the ablation catheter touches, the impedance stays elevated (e.g. 140Ω). This problem is most likely caused by problems in the ablation circuitry outside the heart (e.g. poor dispersive patch/skin contact, rusted connectors). If defects in the circuitry cannot be identified, ablation can be performed in power-control mode only if the baseline impedance increase is moderate. To maintain the preset power level, the RF generator will increase the voltage to compensate for elevated impedance ($P = V^2/R$). As discussed earlier, RF power delivered to the target tissue is a better indicator for lesion formation than electrode temperature. In this situation, operators can set the desired power level and let the RF generator adjust itself to an increased impedance. If the baseline impedance is substantially higher than usual (e.g. >180 Ω) despite replacing patches and cables, it is advisable to replace the ablation catheter, followed by replacing the RF generator.

2. Total impedance does not reflect the impedance at the electrode-tissue interface

As already discussed, the impedance reading on the RF generator represents the total impedance of the entire circuitry. Significant changes in total impedance should prompt the operators to look for an underlying reason and adjust the ablation parameters (e.g. power and time) accordingly. Notably, it is very common for the ablation electrode to be devoid of char or thrombus when removed from the body despite repeated impedance rises during RF application that may signal thrombus formation. In this situation, the thrombus may be left behind on the endocardium unless very high interface temperature leads to char formation on the tip electrode. As already discussed when thrombus/char is formed at the electrode-tissue interface, the overall impedance may not show any significant change due to its poor correlation with the interface temperature. The larger the size of the tip electrode (8-mm tip), the less sensitive impedance rise is in predicting interface temperature because the steep rise in impedance at the electrode-tissue interface is averaged out by the impedance from the rest of the large ablation electrode. Preclinical studies showed that even if the 8-mm electrode was completely surrounded by thrombus, there was barely any impedance rise because thrombus (denatured protein) still conducts RF current.

Several catheter manufacturers have devoted substantial resources to developing novel ablation catheters in which local impedance at the electrode-tissue interface can be measured in hopes that the local impedance drop can help operators estimate RF lesion formation and local impedance rise can help predict interface thrombus/char formation. Local impedance measurement can remove confounding factors such as the size of the tip electrode and the impedance from the circuit outside of the heart. While technical challenges are yet to be overcome, one catheter manufacturer has developed an intermediate solution called “bipolar impedance” by measuring impedance between the tip electrode and the third electrode on the ablation catheter. For bipolar impedance to work, the 3rd electrode (reference electrode) must be free-floating in the blood pool without touching myocardium. This means the orientation between the catheter and target myocardium plays a critical role in the accuracy of bipolar impedance, with more accurate measurements resulting from perpendicular catheter orientation with respect to the myocardium.

The take-home message is that the total impedance change is not sensitive enough to inform the operator about what is happening at the electrode-tissue interface. However, if impedance behaves abnormally, the operator should take the ablation catheter out of the body and inspect the tip electrode for char formation. If impedance continues to be abnormal, the ground pad and its connector as well as the ablation catheter may need to be replaced.

Electrode Temperature

1. How is electrode temperature measured

Temperature feedback during RF ablation is one of the most important parameters used to monitor electrode-tissue contact and lesion formation. However, the electrode temperature reading displayed on the RF generator is the sum of the temperature of the entire ablation electrode. Electrode temperature during ablation is greatly influenced by the surface area of the electrode-tissue interface and the surface area of the ablation electrode exposed to the blood pool. Thrombus is most likely formed at the electrode-tissue interface where the highest temperature is reached. Early studies indicated that thrombus or char begins to form when the electrode-tissue interface temperature reaches 100^{o}C, when tissue boiling occurs. Later studies found that soft thrombus begins to form when the temperature of blood in contact with the ablation electrode reaches 60^{o}C, leading to blood protein aggregation. Thrombus or char can form at the interface when the interface temperature reaches 80^{o}C.

When the target tissue is heated by resistive and conductive heating, heat is transferred back to the ablation electrode as a temperature rise. The electrode temperature is measured by one or several thermocouples embedded in the ablation electrode. A main limitation of temperature monitoring of the tip electrode is that it grossly underestimates both the temperature at the electrode-tissue interface as well as the tissue temperature underneath the interface. All major catheter manufactures are making great efforts to design ablation catheters with multiple thermocouples near the surface of the ablation electrode to better reflect the temperature at the electrode-tissue interface. The location of the thermocouple in the tip electrode is very important. Ideally, the thermocouple should be located *on the surface* of the ablation electrode to accurately measure the electrode-tissue interface temperature to guide ablation. This would eliminate “contamination” from the temperature contributed by the ablation electrode exposed to the blood pool (37^{o}C), particularly when the ablation catheter is positioned perpendicular to the target tissue (**Figure 3.4C**). For various technical and manufacturing design reasons, thermocouples of most ablation catheters have not been positioned on the surface of the tip electrode.

2. Preclinical biophysical studies by the OU-EP group

Most of the ablation catheters had been tested by the OU-EP group using a canine thigh muscle preparation pioneered by Drs. Nakagawa and Jackman (**Figure 3.5**). In brief, the fascia of canine thigh muscle is gently dissected to expose the underlying muscle. The edges of the skin are raised to form a cradle, filled with heparinized canine blood at body temperature. Ablation catheters are positioned on top of the blood-immersed thigh muscle. Temperature probes are positioned at the electrode-tissue interface, 1, 3 and 7 mm below the interface. Parameters such as blood flow and contact force can be manipulated to acquire biophysical data under various conditions. Conclusions from this thigh muscle preparation are used as the foundation for beating heart biophysical studies and eventually clinical trials. It is important to point out that exposing the electrode to circulating blood, instead of saline, is a critical step because saline is a much better conductor than blood. Electrode temperature, impedance and lesion formation differ significantly when measured with exposure to saline bath vs. blood pool. Therefore, data acquired from exposing the ablation electrode to a saline bath poorly reflect RF lesion formation *in vivo*.

Figure 3.6 illustrate a series of preclinical studies conducted by Drs. Nakagawa and Jackman to investigate the effects of electrode size and blood flow on the incidence of thrombus/char formation. Note that without blood flow (similar to ablating an accessory pathway in the middle cardiac vein), a 4mm-tip electrode could deliver only 4 watts of power at the steady state if the electrode temperature was set to be 75^{o}C (**Figure**

3.6A). With blood flow, 12 watts was delivered (**Figure 3.6B**). However, there was no impedance rise when large thrombus or char was formed on the electrode itself or at the electrode-tissue interface. Importantly, thrombus only formed on the side that was opposite to the blood flow, underscoring the importance of electrode cooling on preventing thrombus formation (**Figure 3.6C**). The discrepancy between electrode temperature and interface temperature was greater with the 8-mm electrode, leading to more frequent and larger thrombus/char formation (**Figure 3.6D**). This series of preclinical studies was the basis for Biosense Inc. to recommend the maximal temperature to be set at 55°C and 45°C for 4mm-tip and 8mm-tip electrode catheters in a temperature-controlled mode, respectively.

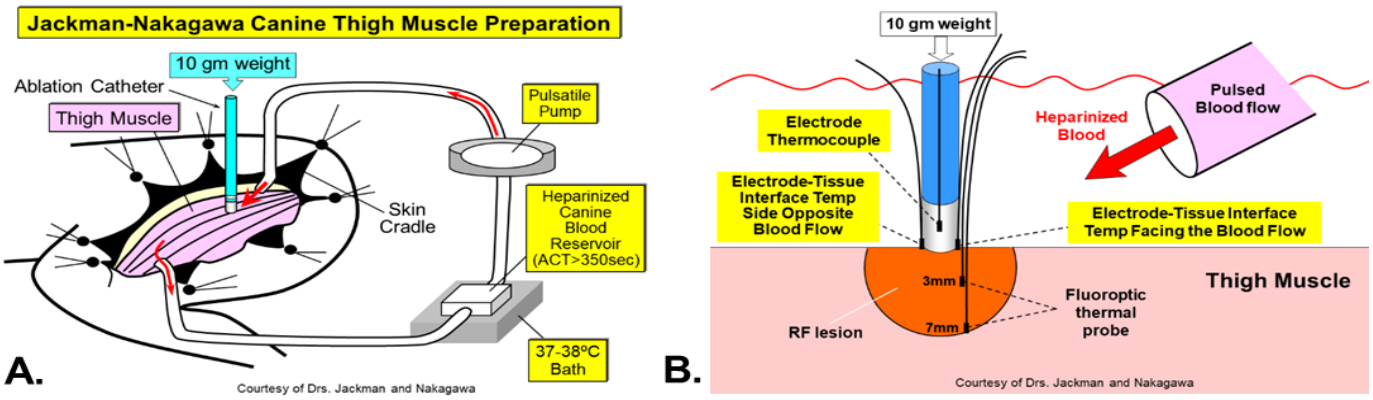
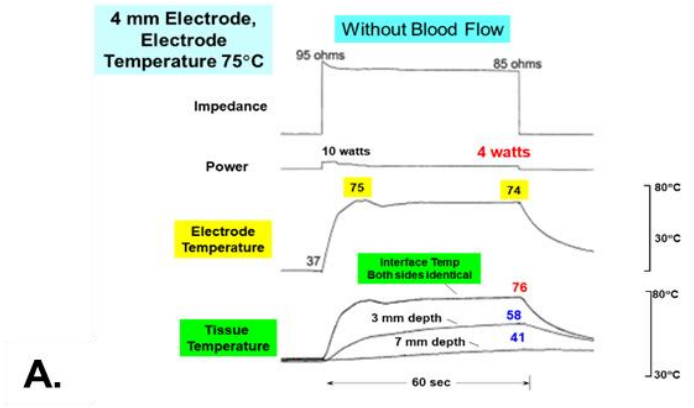
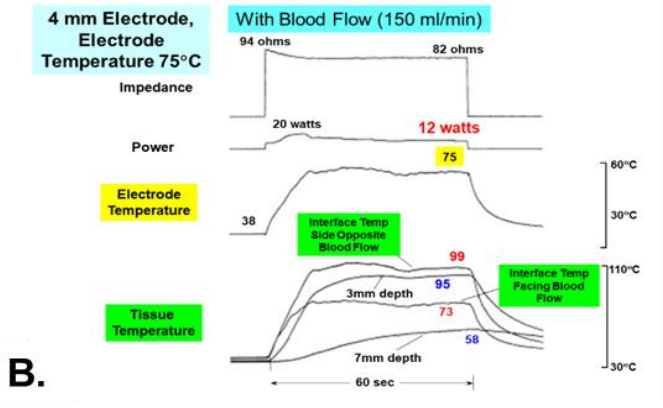
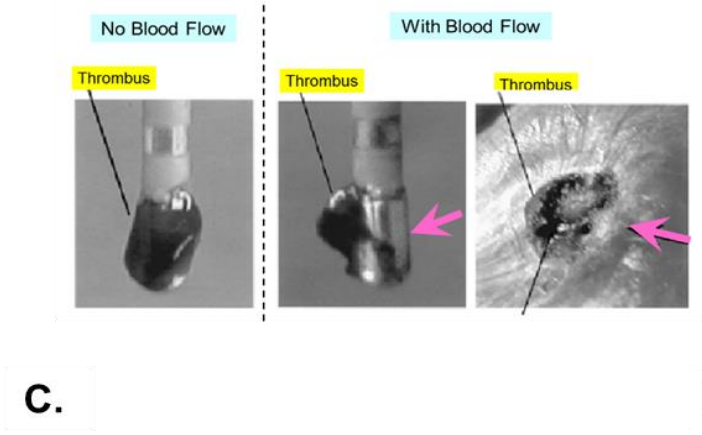


Figure 3.5. Nagakawa-Jackman canine thigh muscle preparation. A. Thigh muscle is exposed to circulating heparinized blood pool in a cradle formed by raising the edges of skin. Contact force and electrode-tissue orientation can be adjusted to different experimental protocols. **B.** Electrode-tissue interface temperature is measured from the side facing vs. opposite to pulsed blood flow to evaluate the effects of blood flow on interface temperature and lesion formation. Modified with permission from *Circulation 1998 Aug 4;98(5):458-65. Courtesy of Dr. Jackman.*







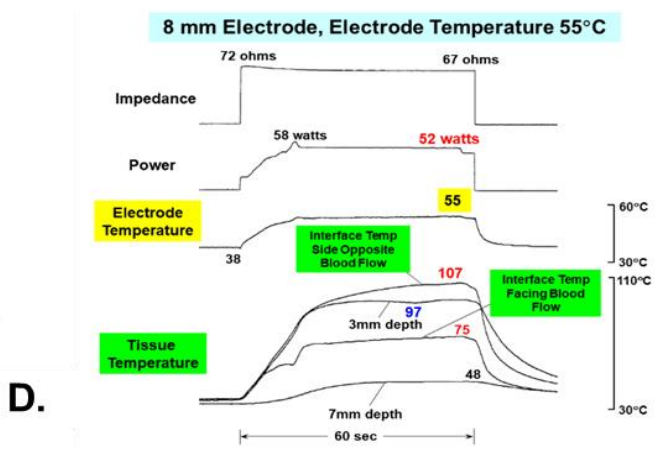


Figure 3.6. Effects of blood flow on impedance, tissue temperature and thrombus formation. **A-C**. A 60-second RF application was delivered to canine thigh muscle using a 4-mm electrode; electrode temperature was set to be 75°C in a temperature-controlled mode. **A.** Only 4 watts was delivered to the target due to lack of blood flow. The electrode-tissue interface temperature (76°) was identical between different sides of the electrode due to absence of blood flow. Tissue temperature at 3 mm depth reached 58°C, capable of inuring tissue. Despite the absence of impedance rise and an interface temperature of 76°C, thrombus formation was noted on the ablation electrode. **B.** With blood flow, higher power (12 W) was required to maintain the same electrode temperature (75°). The peak interface temperature, on the side of the electrode opposite the blood flow, was 99°C (24°C greater than the electrode temperature of 75°C). Conversely, the electrode-tissue interface temperature on the side facing the blood flow was 73°C, similar to the electrode temperature. At depths of 3 and 7 mm, the tissue temperature reached 95°C and 58°C, respectively. Despite the absence of an impedance rise, both thrombus and char formed on the electrode and electrode-tissue interface were present. **C.** Arrows indicate the direction of blood flow. Note the thrombus and char were located on the side opposite the blood flow, where electrode-tissue interface temperature was greatest. **D.** A 60-second RF application was delivered to canine thigh muscle using an 8-mm electrode; electrode temperature was set to be 55°C in a temperature-controlled mode, with blood flow at 150 mL/min. Maintaining the target electrode temperature required a steady-state power of 52 W. Impedance decreased from 72 to 67 Ω. The peak electrode-tissue interface temperature, on the side opposite the blood flow, was 107°C (52°C higher than the electrode temperature of 55°C). The peak electrode-tissue interface temperature on the side facing the blood flow was 75°C, 20°C higher than the electrode temperature. Tissue temperature at depths of 3 and 7 mm reached 97°C and 48°C, respectively. Despite the absence of an impedance rise, large thrombus formed on the electrode and electrode-tissue interface. *Modified with permission from: Matsudaira K et al.Pacing Clin Electrophysiol. 2003 May;26(5):1227-37.*

3. Effects of blood flow on electrode temperature

RF power delivered to tissue is countered by convective heat loss to the blood pool and saline irrigation. At the targeted electrode temperature (e.g. 55°C for a 4-mm electrode), the lesion size varies because blood flow has a profound impact on the electrode temperature and electrode-tissue interface temperature. In low blood flow areas (e.g. coronary sinus or its tributaries, pouch in the cavo-tricuspid isthmus), cooling of the electrode-tissue interface is insufficient, leading to rapid rises of impedance and temperature. The author always uses the analogy of a high-speed dental drill to explain this phenomenon to fellows. To prevent overheating at the drill-tooth interface, the tip of the drill spreads out cold water to cool the interface. An irrigated ablation catheter serves the same purpose to cool the electrode-tissue interface. Therefore, the most important contribution of irrigated catheters to ablation lesion formation is to cool the electrode-tissue interface to allow sufficient RF current to flow into the target tissue to create appropriate lesions, as well as to reduce the incidence of thrombus formation at the electrode-tissue interface.

Higher blood flow improves electrode cooling, leading to an increase in the power required to maintain the target electrode temperature in a temperature-controlled mode. In an area with high blood flow (e.g. RVOT, AV annulus), the electrode-tissue interface is appropriately cooled by blood flow through convective cooling. An irrigated catheter therefore does not have a significant impact on the lesion size but helps reduce the incidence of thrombus formation. For this very reason, ablation of accessory pathways or RVOT PVCs usually does not require an irrigated catheter.

For a given electrode temperature, ablation lesion size varies with local blood flow. In a temperature-controlled mode, the electrode temperature may not reach 55°C due to high blood flow and efficient convective cooling. To maintain the target temperature (e.g. 55°C), the RF generator increases power output. If the operator's mind is fixed on the target temperature, s/he may deliver a longer RF application with higher power to an RVOT target based on the wrong assumption that lesion formation is poor as a result of lower electrode temperature; this misconception may lead to myocardial perforation. In general, in a high blood flow area, if power is sufficient, the ablation lesion should be adequate despite not reaching the target temperature. A counterexample is that a pouch in the cavo-tricuspid isthmus often has very poor blood flow. Ablation using a non-irrigated ablation catheter may deliver only 3-5 watts when the electrode temperature reaches the target temperature (e.g. 55°) in a temperature-controlled mode. If the operator believes "good electrode temperature makes good ablation lesions" and continues to ablate, conduction block across the CTI line may never be accomplished. Without saline irrigation, RF lesion size is often larger with a lower electrode temperature and high blood flow as compared to a higher electrode temperature with low blood flow. Another important point

about blood flow is that at the same site, blood flow can be very different between sinus rhythm and tachycardia. The operator may need to adjust the target temperature or power accordingly.

4. Discrepancy between electrode temperature and electrode-tissue interface temperature

Electrode-tissue interface temperature is one of the most important parameters determining ablation efficacy and safety. Higher interface temperature serves as a better heat source to allow resistive and conductive heating to make an effective RF lesion. Poorly regulated high interface temperature can lead to thrombus/char formation, tissue boiling (e.g. steam pop) and cardiac perforation. Unfortunately, owing to the lack of thermocouples reliably measuring the interface temperature, the temperature information displayed on the RF generator can greatly underestimate the electrode-tissue interface temperature, particularly when the blood flow is high, allowing for cooling of the ablation electrode. That is, high electrode-tissue interface temperature is averaged out by a lower temperature (37°C) of the rest of the tip electrode exposed to the blood pool. For a 4-mm ablation electrode, the electrode-tissue interface temperature can be 20°C higher than the electrode temperature displayed on the RF generator (**Figure 3.7**). The discrepancy can be as high as 40-50°C for an 8-mm electrode. Blood flow and the length of the ablation electrode have a profound impact on the discrepancy between the overall electrode temperature and interface temperature. Discrepancies are greater with non-irrigated catheters than with irrigated catheters. The discrepancy further increases with higher power and lower blood flow.

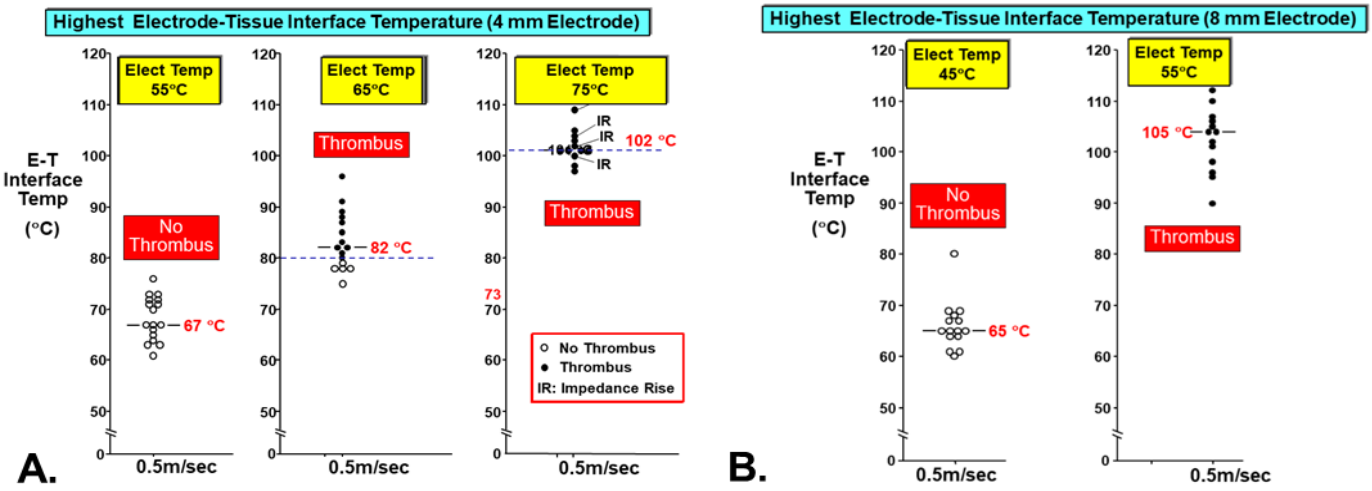


Figure 3.7. Discrepancy between electrode temperature and highest electrode-tissue interface temperature as well as the incidence of thrombus formation. **A. Left panel.** With a 4-mm electrode and moderate blood flow (0.5m/sec), the average peak electrode-tissue interface temperature was 67°C if the electrode temperature was set to be 55°C. No thrombus formation was detected. In contrast, setting the electrode temperature at 65°C (**middle panel**) or 75°C (**right panel**) led to much higher electrode-tissue interface temperature (82°C and 102°C, respectively). Large thrombus formation (black dots) was noted without impedance rise. **B.** With an 8-mm electrode and moderate blood flow (0.5m/sec), the average peak electrode-tissue interface temperature was 65°C without impedance rise if the electrode temperature was set to be 45°C. No thrombus formation was detected. In contrast, setting the electrode temperature at 55°C led to much higher electrode-tissue interface temperature (105°C). Large thrombus formation was noted. Note that the discrepancy between the electrode temperature and interface temperature is 50 °C. *Modified with permission from: Matsudaira K et al.Pacing Clin Electrophysiol. 2003 May;26(5):1227-37.*

Historical Vignette

In early days of catheter ablation, operators often set the target temperature at 70-75°C in hopes that higher temperature would create larger ablation lesions. It was not uncommon to encounter situations when operators had difficulty withdrawing the ablation catheter out of the heart because the ablation electrode was stuck to desiccated tissue. Sometimes, when the catheter was removed, there was large thrombus/char on the tip electrode. The large discrepancy between the electrode-tissue

interface and electrode temperature displayed on the RF generator was recognized later. By lowering the target temperature to 55-60°C, this problem was largely solved.

In the 1990s, one of the world's most prestigious EP laboratories began to do extensive linear left atrial ablation using a 4-mm non-irrigated ablation catheter. The targeted temperature was set at 65°C. Operators often saw extensive coagulum on the tip electrode of the catheter. After the target temperature was reduced to 55°C, coagulum problems improved substantially.

5. Effects of electrode-tissue contact

A common scenario during ablation in the RV in a power-controlled mode is that the temperature suddenly rises during RF application. This scenario often occurs when the tip electrode of the ablation catheter falls into trabeculated RV myocardium or a crevice. The surface area of electrode-tissue interface suddenly increases and convective cooling by the blood pool suddenly decreases, leading to a temperature rise. By the same token, if the ablation electrode is wedged into myocardium (e.g. high contact force), the surface area exposed to blood pool is smaller, which can lead to a higher temperature during ablation when operated in a power-controlled mode.

The orientation between the ablation electrode and targeted tissue (e.g. parallel vs. perpendicular) also affects the electrode temperature displayed on the RF generator. In the parallel orientation, there is more electrode-tissue contact surface area and less surface area facing the blood pool; the electrode temperature may be higher than that in the perpendicular orientation (**Figure 3.4C**).

Irrigated vs. Non-irrigated Electrode Catheters

As already discussed, blood flow at the target tissue has a profound impact on lesion size and thrombus formation. If a dentist uses a high-speed dental drill to treat a cavity without irrigating the drill-tooth surface with cold water, the patient probably will immediately scream in pain and drilling has to be stopped immediately. The original idea of the irrigated ablation electrode, proposed by Dr. Fred Wittkampf, was to overcome this problem that limits the delivery of prolonged high-power RF applications. For a given electrode size and contact force, RF lesion size is determined by the magnitude of power and the duration of ablation. At higher power, the duration of ablation is often limited by a high electrode-tissue interface temperature. If tissue desiccation occurs at the interface, substantially higher interface impedance will limit the RF current flowing into the tissue. Therefore, the main purpose of irrigation is to avoid overheating the electrode-tissue interface so that operators can increase the power to create an effective ablation lesion. At the same time, avoiding overheating at the electrode-tissue interface can prevent thrombus formation. One important benefit of an irrigated-tip catheter is that it lessens the effect of low blood flow/convective cooling; it allows appropriate power to be delivered to areas with low blood flow (**Figure 3.8**). Notably, irrigated ablation at a given wattage will result in a similar lesion size regardless of the blood flow.

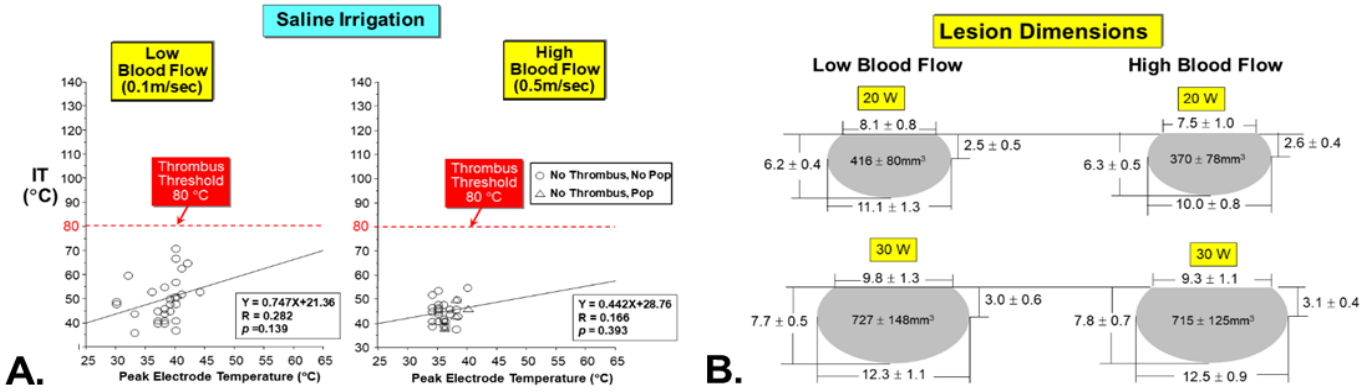


Figure 3.8. Correlation between peak electrode temperature (abscissas) and electrode-tissue interface temperature (ordinate) in the presence of saline irrigation. A. With saline irrigation, the electrode temperature does not reflect the interface temperature because irrigated saline is usually at room temperature; the interface temperature was significantly below the thrombus threshold (80°C) regardless of the blood flow. **B.** With saline irrigation, the RF lesion size was similar regardless of blood flow in a given wattage. Modified with permission from *Circulation. 1998 Aug 4;98(5):458-65.*

Historic Vignette:

In the late 80s, Dr. Wittkampf presented the idea of the irrigated electrode at a conference. After his presentation, Dr. Jackman rushed out, borrowed some coins from his friends, found a pay phone and called Mr. Will Webster (the founder of the Webster catheter company). The first irrigated electrode catheter was made a few months later.

Has one ever wondered why the upper temperature limit of a non-irrigated-tip catheter made by Biosense Inc. is set at 55°C and an irrigated-tip catheter (e.g. ThermoCool) is set at 43°C? Settings like these were derived from preclinical biophysical studies investigating lesion size, thrombus formation and risk of perforation. Since their advent in the early 2000s, irrigated-tip catheters have been widely used, particularly for left atrial and left ventricular ablation. The design of the irrigation system varies among manufactures. Clinically available catheters have different numbers of irrigation holes, ranging from 6 holes to 56 holes. Different designs of the irrigation system have a significant impact on the reading of the electrode temperature, lesion formation and the risk of thrombus formation. Regardless of the catheter design, the electrode-tissue interface temperature is significantly lower using irrigated catheters than that of non-irrigated catheters because the temperature of irrigated fluid is usually room temperature. Despite lower electrode-tissue interface temperature, tissue temperature (e.g. 3 and 7 mm beneath the interface) is similar to or moderately higher than that of non-irrigated electrode catheters if the non-irrigated electrode is positioned at a site *with good blood flow*. The lesion size and geometry are therefore similar.

Occasionally, the irrigation hole can be obstructed if the tip electrode is deeply wedged into myocardium, leading to higher electrode-tissue interface temperature and even thrombus formation. If some of the irrigation holes are obstructed by firm electrode-tissue contact, irrigation flow naturally will be diverted to the non-obstructed holes, defeating the purpose of irrigation. If the design of the irrigation system cannot increase the flow to the obstructed holes, significant thrombus/char can occur despite irrigation. This scenario typically occurs during VT ablation when higher contact force, higher power and longer RF duration are pursued. If impedance rises repeatedly during RF applications, the tip electrode should be inspected. Power, force and time should be adjusted accordingly.

1. Electrode temperature during saline irrigation

With saline irrigation, electrode temperature information is nearly lost except extreme temperatures (low 30s°C vs. low 40s°C using a 6-hole irrigated catheter). It may be difficult to reach 43°C using a 6-hole irrigated catheter unless the tip electrode is laid in parallel with the tissue or wedged deep into tissue. If the electrode temperature during ablation is 43°C with 6-hole irrigation, the ablation electrode may have very good contact with the tissue (e.g. wedged deep into the tissue). In this case, the smaller surface area exposed to the blood pool and the larger surface area at the electrode-tissue interface provides more effective tissue heating; the electrode temperature therefore reaches 43°C. If the electrode temperature is low (e.g. in the low 30s°C), the ablation electrode may have very poor contact with the tissue (e.g. floating in the blood pool without electrode-tissue contact at all). In most cases, the electrode temperature displayed on the RF generator provides little information about the electrode-tissue interface temperature or lesion formation. The maximal temperature of 43°C for 6-hole ThermoCool catheters is for safety (to avoid thrombus formation), not for efficacy. With a 56-hole irrigated electrode catheter, the electrode temperature is even lower due to a more effective irrigation system that allows the formation of a shield of saline around the tip electrode. It is therefore even more difficult to correlate the electrode-tissue interface temperature with the electrode temperature reading displayed on the RF generator. For this reason, one should not judge RF lesion formation based on electrode temperature with irrigated RF ablation.

Electrode-tissue orientation (e.g. parallel vs. perpendicular) also impacts the effect of irrigation. For a ThermoCool catheter, saline irrigation comes out from the 6 holes near the tip of the electrode; the proximal edge of the electrode is not irrigated. If the tip electrode is laid in parallel with the targeted tissue, thrombus tends to form at the myocardium in contact with the proximal edge of the tip electrode due to the edge effect and lack of active cooling. The edge effect refers to the enhanced current density at the edge of the electrode. This problem usually does not exist if the tip electrode is laid perpendicular to the targeted tissue because the proximal edge is not in contact with tissue and is cooled by the blood flow. Certainly, the 56-hole irrigated-tip catheter is preferred in terms of providing evenly distributed irrigation flow to cover the entire surface area of the tip electrode and minimize the risk of thrombus formation. More effective irrigation by 56 irrigation holes leads to a lower electrode temperature. Operators may mistakenly think the tip electrode is too cold and attempt to increase the contact force or power, which can lead to perforation. Pertinent to this issue, delivering higher power to compensate for lower electrode temperature initially led to an increased incidence of tamponade when 56-hole irrigated catheters were introduced to the market without being equipped with contact force sensors. If operators use a 56-hole irrigated catheter without contact force sensors, lower electrode temperature is not an indication of poor lesion formation and does not justify an increase in power.

Due to the poor correlation between total impedance and impedance at the electrode-tissue interface, there may be minimal or no impedance rise during RF application if irrigation rate is reduced. For a 6-hole ThermoCool catheter, thrombus can occur without any impedance rise at all, particularly when irrigation flow rate is <15 cc/min. Based on the thigh muscle and beating heart studies, Biosense Inc. recommended an increase in the irrigation flow rate to 30 cc/min if power is ≥30 watts to prevent thrombus formation. In the OU-EP laboratory, the standard practice is to irrigate the 6-hole ThermoCool catheter at 30 cc/min regardless of power because operators frequently forget to adjust the flow rate according to power. A few doses of diuretics usually take care of the problems related to additional saline infusion.

2. Saline, half saline and glucose water (D5W)

Two decades ago when the irrigated-tip catheter was still in its infancy, investigators considered glucose water, saline and half saline as the irrigation fluid. Ideally, the fluid of choice should be something that has poor electrical conductivity thereby serving as an insulator around the tip electrode. That is, there will be less RF current shunting to irrigation fluid and more RF current delivered to tissue. Among the three candidates, glucose water has negligible electrical conductivity, allowing it to act as an effective insulator during RF applications. However, it has been shown to create large fluctuations of the electrode impedance as a result of the unpredictable mix of blood and D5W, leading to large fluctuation of the RF current (Ohm's law). Another issue with D5W is that, if a large volume of irrigation is required for a long procedure, hyperglycemia and electrolyte imbalance may lead to serious consequences.

For electrical conductivity, half saline is halfway between D5W and saline. However, the lesion size made by half saline irrigation was only 5-7% larger than that made by 6-hole saline irrigation in pre-clinical studies (canine thigh muscle and beating heart preparations). Considering the possibility that several liters of half saline may be infused into the body for long procedures, the benefit of the relatively small increase in lesion size is outweighed by the potential risks of electrolyte and osmolarity imbalance. Recently, there has been a resurgence of interest in using half saline irrigation for VT ablation in hopes of making deeper RF lesions. In theory, there are two prerequisites for half saline to substantially increase the lesion size. First, it would require a 56-hole irrigated catheter in order to create a "low conductivity shell" around the tip electrode of the ablation catheter. Second, the blood flow should be slow enough to allow half saline to form this low-conductivity shell. Consider the following analogy: one walks into a cold creek and desires to keep his/her feet warm by pouring hot water around the feet to insulate them from the cold creek water. If the flow of the creek is rapid, there is little chance that his/her feet can stay warm. Similarly, for half saline irrigation to make a larger lesion, the ideal scenario would be that the site of ablation is in a low blood flow area and a 56-hole irrigated electrode catheter is used. It may be more effective for ablation in the epicardial space where no blood flow exists. In addition, if a low-conductivity shell is formed, the baseline impedance should be significantly higher due to its insulation effect. However, the magnitude of impedance drop during RF application that is safe and effective in this situation remains unknown. Based on the pre-clinical studies done

by Drs. Jackman and Nakagawa over the past 3 decades, the OU-EP group has not utilized half saline irrigation because the lesion size increase is very small (5-7%) in the presence of good electrode-tissue contact. We instead increase the force, power or duration of RF application to create a larger lesion.

3. Special concerns about epicardial ablation

In contrast to endocardial ablation, the ablation electrode is not exposed to the blood pool in epicardial ablation. This leads to a higher overall impedance. When a saline-irrigated ablation catheter is used, the pericardial space progressively accumulates more and more saline (a better conductor than epicardium), leading to reduced overall impedance. RF application in the saline-filled pericardial space can lead to shunting more RF current to saline and reduce RF lesion formation. Therefore, pericardial saline needs to be drained periodically to maintain ablation efficacy and prevent tamponade. Another issue related to epicardial ablation is that the ablation catheter does not always point toward the epicardial surface but to the lung. In pre-clinical studies, many of the RF lesions were noted to be made on the pleural surface. To make an effective RF lesion, the vector of the ablation electrode must be directed toward the epicardium.

Contact Force

Higher contact force, pushing the tip electrode into the target tissue, leads to increased surface area of the electrode-tissue interface, which translates into a larger source of heat, thereby increasing both resistive heating and conductive heating (**Figure 3.9A**). Another factor contributing to greater lesion size with higher contact force is tissue compression in which the target tissue is stretched thinner by increasing force, facilitating the creation of a transmural lesion. Higher force potentially can increase thrombus formation because pushing the tip electrode deep into the tissue may obstruct irrigation holes.

Earlier pre-clinical studies on RF lesion size created in beating animal hearts all had the same problem: approximately 10-15% of RF lesions were nowhere to be found in histologic studies because the operator was not aware that the ablation catheter did not make contact with the cardiac tissue. This type of problem has largely been solved with the advent of contact force sensing catheters.

In the early stages of the contact force catheter trials, operators were blinded to contact force information but had to assess the quality of contact based on electrograms, impedance, fluoroscopic images and tactile sense. Theoretically, higher contact force should correlate with higher impedance due to a larger electrode-tissue interface (e.g. the tip electrode is wedged into tissue). Lower contact force should correlate with lower impedance due to more electrode surface being exposed to the blood pool. To many operators' surprise, the correlation between registered contact force and impedance as well as self-awareness was quite poor. Using a stiff deflectable sheath often diminishes the feedback from operators' tactile sense. After completion of these studies, it is clear that EGM amplitude (both unipolar and bipolar) as well as impedance do not correlate with contact force, both in AF and in sinus rhythm (**Figure 3.9B**). Injury current recorded on the unipolar EGM suggests the electrode-tissue contact is good (**Figure 3.9C**). However, after an initial change in the morphology of the unipolar EGM (similar to ST elevation), more contact force may not lead to more changes in the unipolar EGM morphology.

Power, Force and time

As described above, lesion size is determined by multiple factors such as electrode-tissue orientation, force, power and duration of RF applications. Because the orientation between the tip electrode and tissue (e.g. perpendicular vs. parallel) is very difficult to control, operators usually depend on adjusting power, force and duration of ablation to make effective lesions and prevent perforation. Biosense Inc. has put forward a novel function named the Ablation Index as a summative measure of lesion effectiveness. In brief, the Ablation

Index calculation is based on the lesion size and corresponding power, time and force measured in preclinical studies. With numerous samples of lesion size and geometry (diameter, depth and shape), engineers derived a complex equation to correlate power, time and force with lesion size.

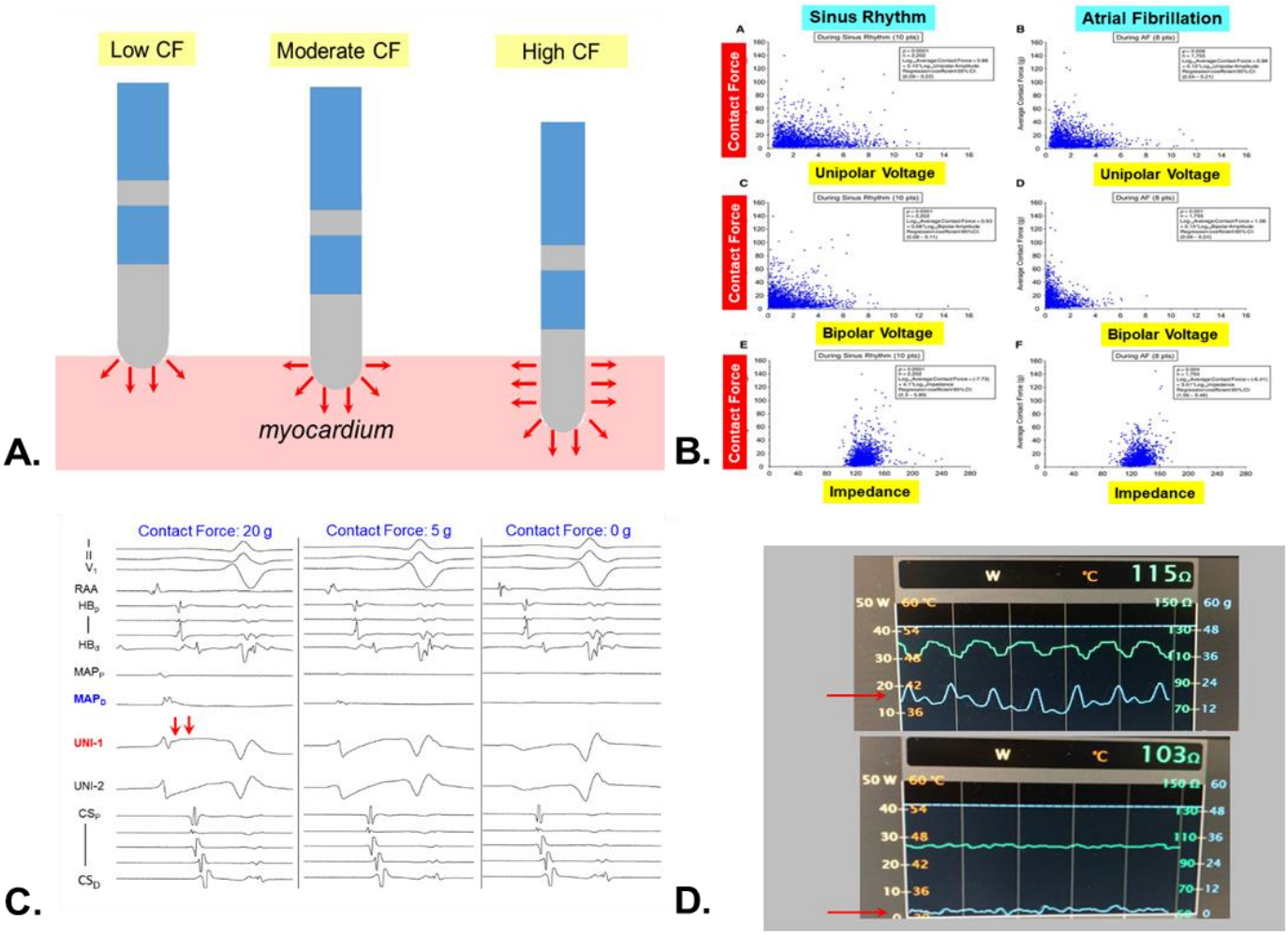


Figure 3.9. Contact force sensing catheters. **A.** High contact force may lead to a larger surface area of electrode-tissue interface and creates a larger heat source (red arrows) to facilitate RF lesion growth. **B.** The correlation between contact force and impedance as well as voltage (unipolar and bipolar) is very poor. **C.** Higher contact force may generate EGMs with "injury current" (red arrow) recorded on the tip electrode but one cannot use the degree of injury current to gauge the contact force. **D.** The contact force curve (red arrow) also provides information about the contact force particularly when it is low. The top panel shows that the force fluctuates with each cardiac cycle (red arrow). The bottom panel shows that the force stays flat regardless of cardiac cycles. The latter indicates poor electrode-tissue contact and is not likely to be compensated by increasing the power and time. *Modified with permission from: Nakagawa et al. Circulation Arrhythm Electrophysiol 2013;6(4):746*

Although lesion size is increased by increasing any one of the three parameters (power, time and force), it is imperative to know that the three parameters carry different weight in the Ablation Index calculation. For example, 20 seconds of ablation with 30 watts and 15 gram of force does not make the same lesion size as 20 seconds of ablation with 15 watts and 30 grams of force (power x force x time=90,000 for both cases). In the Ablation Index calculation, time carries more weight than power and force. With stable tissue contact, even 3-4 grams of force can make a good lesion. Another important factor to consider is the lack of information about the thickness of the target tissue. One may assume that the wall on the LA roof is thin, particularly at the RSPV-roof junction. However, the wall in the center of the roof might be significantly thicker than that of the RSPV-roof junction. The main drawback of the Ablation Index is the lack of information about target tissue thickness; operators can only rely on anecdotal experience (e.g. the LA inferior wall is generally thicker than the roof, or the LA anterior wall is generally thicker than the posterior wall). To account for this, operators may

target a different ablation index depending on ablation site (e.g. 380 on the posterior wall and floor of the LA, and 500 on the roof and anterior wall of the LA).

The percentage of time in a cardiac cycle that the catheter stays in contact with the targeted tissue is also very important. If the catheter is on top of or wedged into an RV trabeculation, the former may stay in contact with the tissue 30% of the cardiac cycle but the latter 100%. In theory, the impedance in these two scenarios should be different, but as noted above, overall impedance is not sensitive enough to tell the difference. The same power, time and force will produce very different lesions in these situations. In addition to monitoring the digital display of the force and direction of the contact force vector, Dr. Jackman watches the curve of the force (**Figure 3.9D**). If the contact force is low but the force curve correlates with the cardiac cycle, it suggests that the electrode-tissue contact is reasonable. Higher power and longer duration of RF application can compensate for low force. This scenario often occurs during ablation of the LAA-LPV ridge when the catheter is laid in parallel with the ridge. If the contact force is low and the force curve is flat, it indicates that the tip electrode is in the blood pool; there is no use in increasing the power or duration of RF application, and the catheter must be repositioned.

RF lesion formation is different between normal myocardium and myocardial scar (e.g. post-infarct). With the same power and contact force, the lesion size in scar tissue is significantly smaller than that of normal myocardium. It is well known that many of the arrhythmogenic channels (survival myocardial bundles) are buried deep in the infarct scar. Therefore, higher power, longer RF applications and good contact force are required to create good lesions to penetrate the scar tissue to eliminate these myocardial bundles. This is very important when operators intend to “homogenize” a large infarct scar. Without enough power, time and force, covering a large scar with ablation tags may not correlate with effective ablation of the arrhythmogenic channels. (**Figure 11.15**).

Unipolar vs. Bipolar Ablation

In unipolar ablation, RF current passes from the ablation electrode to the ground electrode (dispersive patch). Bipolar ablation requires two ablation catheters, the tip electrode of one catheter serves as the active ablation electrode and the tip electrode of the other catheter serves as the ground electrode. This approach bypasses the intervening tissue between the tip electrode and dispersive patch (muscle, fat, skin, etc.) in the unipolar ablation mode. Bipolar ablation had been attempted by a catheter manufacturer to isolate pulmonary veins using a Lasso-like catheter design in which RF current was delivered between neighboring electrodes of the Lasso-like catheters. This approach was abandoned due to thrombus formation that occurred without saline irrigation. Another problem is that shallow lesions are made when RF current flows between two neighboring electrodes.

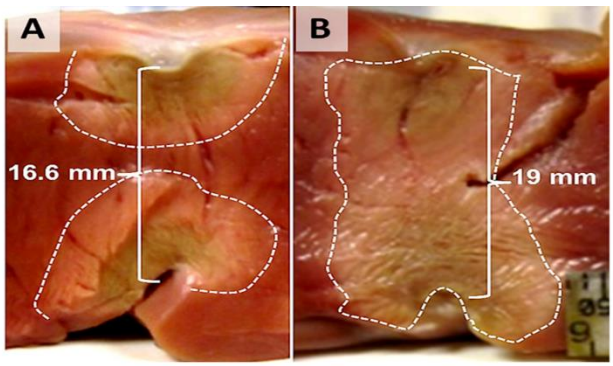


Figure 3.10. Different lesion geometry by (A) sequential unipolar ablation and (B) bipolar ablation. Bipolar ablation may create a transmural lesion in thick myocardium such as the inter-ventricular septum. Modified with permission from; Koruth JS et al. Heart Rhythm. 2012 Dec; 9(12):1932-41.

Since the RF generator for bipolar ablation is not an FDA-approved device, manufacturers do not support the use of bipolar ablation. It requires each EP laboratory to make its own off-label modifications to conduct bipolar ablation. The OU-EP laboratory has not attempted bipolar ablation; the discussion below is only based on biophysical principles. Currently, bipolar ablation is performed to treat VT of intramural sources. Bipolar ablation has been shown to be superior to two sequential unipolar RF applications in which two ablation catheters connecting to two RF generators deliver RF current sequentially in an attempt to “sandwich” the intramural source of VT. The shape of RF lesions created by bipolar ablation appears to be different from those created by sequential unipolar ablation (**Figure 3.10**). In theory, bipolar ablation works best for VT sources located in the inter-ventricular septum because the two ablation catheters are directly facing each other and exposed to the blood pool in a similar way. In other words, the impedance mismatch between the two catheters is minimal,

and the current density at the electrode-tissue interface of catheter is similar. If one attempts endocardial-epicardial bipolar ablation, an impedance mismatch occurs because the impedance of the epicardial ablation catheter is much higher than that of the endocardial catheter. If the catheter is positioned on top of a thick layer of epicardial fat, the impedance mismatch may be even worse. Ablation lesions in this scenario are likely to be much smaller on the epicardial side.

Some electrophysiologists have attempted *simultaneous* unipolar ablation in which two ablation catheters and two RF generators are delivering RF current at the same time. The lesion size appears to be larger than those made by *sequential* unipolar ablation. This approach may be an alternative to bipolar ablation to treat intramural VT or PVCs.

Frequently Asked Questions

Question: Does an irrigated electrode make a bigger lesion than a non-irrigated electrode?

Not really! Indeed, in the presence of identical contact force, high blood flow, electrode-tissue orientation and power, the lesion size may be slightly smaller at the electrode-tissue interface because the interface is irrigated by saline (**Figure 3.11**). As discussed already, lesion size would be similar comparing lesions made by an irrigated electrode vs. a non-irrigated electrode if the blood flow (electrode cooling) is good. In the presence of poor blood flow (e.g. middle cardiac vein), saline irrigation allows higher power to be delivered to the target tissue and an increase in lesion size. However, the geometry of the lesion made by an irrigated electrode does look different from that made by a non-irrigated electrode. The largest diameter of lesions made by an irrigated electrode is 3-4 mm below the electrode-tissue interface. The reason for this shape is that the surface near the electrode-tissue interface is cooled by irrigation fluid, making the surface of the lesion smaller. A non-irrigated electrode makes a lesion with its largest diameter at the electrode-tissue interface because there is no irrigation fluid to cool the electrode-tissue interface.

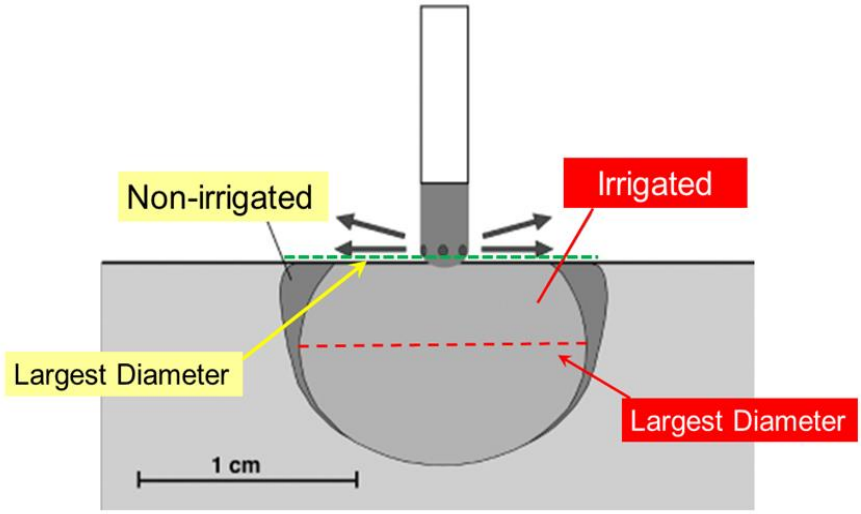


Figure 3.11. Different geometry of RF lesions made by irrigated vs. non-irrigated electrode. The surface area of a lesion made by irrigated electrode is slightly smaller; the site with the largest diameter is usually 3-4 mm below the electrode-tissue interface (red dotted line). Contrarily, the site with the largest diameter of an RF lesion made by a non-irrigated electrode is at the electrode-tissue interface (green dotted line). The total volume of RF lesions formed by an irrigated catheter is slightly *smaller* than that made by a non-irrigated catheter. *Modified with permission from: Wittkampf FH, Nakagawa H. Pacing Clin Electrophysiol. 2006 Nov;29(11):1285-97.*

Question: Is it appropriate to raise the target temperature (e.g. from 55°C to 65°C) of a non-irrigated ablation catheter if only low wattage (e.g. 4-5 watts) could be delivered to tissue with low flood flow (e.g. middle cardiac vein)?

The answer is yes, but this approach may not increase the wattage very much because the main problem limiting RF energy delivery is lack of cooling. Increasing the target temperature may allow the generator to deliver a few more watts. If slightly higher wattage appears to affect the arrhythmogenic tissue, the operator may want to maintain the same effective power. Further increases in power may lead to tissue

overheating and an impedance surge that shuts down the RF generator. If higher power still does not affect the arrhythmogenic tissue, a better solution is to use an irrigated catheter. One needs to be aware that if the tip electrode is in the middle cardiac vein or other CS tributary, a low power (e.g. 10 watts), high impedance RF application via an irrigated electrode may still make a good RF lesion because less RF current is shunted to the blood pool and electrode-tissue contact may be very good.

Question: Does the size and location of the dispersive patch (ground electrode) affect ablation lesion size?

In theory, the percent of total impedance contributed by the dispersive patch will be smaller if the dispersive patch is large and close to the heart. When catheter ablation was still in its infancy, many electrophysiologists experimented on various ideas such as placing the dispersive patch on the back behind the heart, using a larger patch or two patches. Only a very modest increase in the ablation lesion size was found. It appears that as long as the dispersive patch is placed on the torso, it makes little difference. However, if the patch is loosely attached to skin or the connecting cable of the patch is rusted, the total impedance of the circuit can be increased substantially. In the OU-EP laboratory, we use two dispersive patches in hopes of lowering the total impedance and enhancing ablation efficacy. Patches are positioned on the flank below the rib cage. The average impedance of the entire circuit is between 100 and 110 ohms.

Is RF lesion size proportional to the duration of RF application?

Generally, the longer the RF application, the larger the ablation lesion size. However, the correlation is not linear. In the presence of good contact force, the first 20-30 seconds of RF application play a critical role in lesion formation; a 60-second RF application usually makes a sizable lesion. As RF application continues, the lesion size continues to grow but with a much smaller increment (**Figure 3.2B**). For most ablation targets, unless suboptimal contact force cannot be improved, it does not make sense to deliver a 4-5 minute RF application. Instead, getting better electrode-tissue contact for the first 30 seconds of RF application may create a better lesion. One of the exceptions is when ablating VT/PVCs originating from an intramural source. High power, high contact force and long RF applications may be needed to eliminate a deep intramural arrhythmogenic focus. The author has successfully ablated several LV summit PVCs from the LV endocardium in cases where either the ablation catheter could not reach the anterior interventricular vein, the epicardial target was in close proximity to a major coronary artery, or RF applications in the coronary sinus tributary were limited by high impedance and low wattage. In these cases, a Stereotaxis ThermoCool catheter was positioned in stable contact with the site showing the earliest far-field potential on the LV endocardium. It took >3 minutes (50 watts) to suppress the VT/PVC and another 3-5 minutes to eliminate the source of the arrhythmia. In this scenario, the slow and small increment in lesion growth during a very long RF application is needed to eliminate the intramural source of the arrhythmia. An alternative approach is to use bipolar ablation or a needle catheter if available. The author doubts if half saline would help in this situation because the blood flow in the LVOT area is very high, making it difficult to form a half-saline “low conductivity shell”.

Does a large electrode make a bigger lesion?

It depends on how one defines the term “large”. Ablation lesion size is a function of the surface area that is heated by the ablation electrode. Dr. David Haines’ pioneering work showed that lesion size is proportional to the *radius* (not length) of the tip electrode (**Figure 3.12**). If the *radius* of the electrode is larger (e.g. a 10 Fr. electrode vs. a 7 Fr. electrode), a larger electrode will make a larger lesion because of a much larger electrode-tissue interface (in other words, a much larger heat source). If the *length* of an electrode is longer (e.g. 7 Fr. electrode, 4 mm vs. 8 mm), the lesion size made by the 8-mm electrode is usually smaller in the presence of identical contact force and power. The lesion is even smaller if the electrode-tissue orientation is perpendicular (**Figure 3.13**). In this orientation, it has the smallest surface area at the electrode-tissue interface but largest surface area exposed to the blood pool. In contrast, the largest lesion is formed by laying the distal electrode in parallel with the targeted tissue, which is exceedingly difficult to do with a very

stiff 8-mm electrode catheter. The large recording range and far-field potential also severely limit the mapping accuracy of an 8-mm electrode catheter.

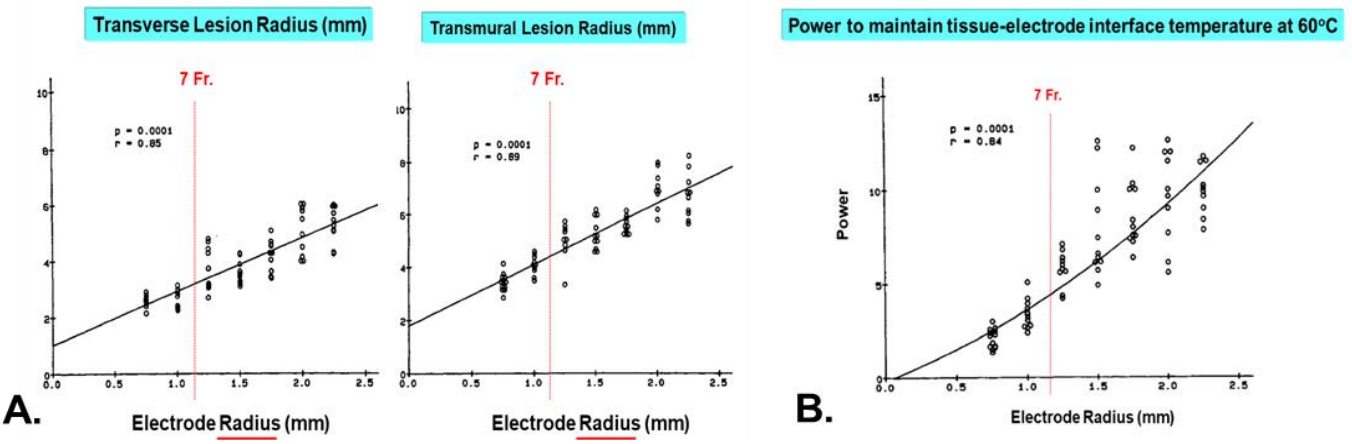


Figure 3.12. A. Transverse lesion radius (left panel) and transmural lesion radius (right panel) are proportional to electrode radius (abscissa), not length. **B.** Power required to maintain electrode-tissue interface temperature at 60°C is proportional the electrode radius. The diameter of a 7 Fr. catheter is 2.3 mm (0.33mm x7). The radius of it is therefore 1.165 mm. *Modified with permission from: Haines DE et al. Circ Res. 1990 Jul;67(1):124-9.*

Dr. Jackman's dream ablation catheter (but no manufacturer would make it!)

*Has one ever wondered why the size of the distal electrode of the ThermoCool catheter is 3.5 mm, not 4 mm? It originated from a series of studies conducted by Drs. Jackman and Nakagawa. With a progressively longer length of the distal electrode, the lesion size is more and more dependent on the electrode-tissue orientation. For a 2-mm irrigated electrode, the surface area of the tip electrode in contact with the targeted tissue is minimally dependent on the angle between the electrode and tissue, because the diameter of the tip electrode of a 7 Fr. catheter (0.033 mm x7=0.231 mm) is not very different from the length of the tip electrode (**Figure 3.13**). Different orientation between the tip electrode and tissue has an insignificant impact on the surface area of the electrode-tissue interface. Moreover, the surface area exposed to the blood pool is minimal, directing most of the RF current to the electrode-tissue interface. For the two aforementioned reasons, a 2-mm irrigated electrode catheter produces much larger lesions than a 4-mm catheter in the presence of identical contact force and power. Another great advantage of a 2-mm electrode is its higher mapping accuracy (less far-field potential).*

When the ThermoCool catheter was in the developmental stage, Biosense Inc. had three major concerns about Dr. Jackman's dream catheter. First, Biosense Inc. had to reeducate electrophysiologists to believe that a smaller-tip electrode catheter could make larger lesions. Second, the smaller electrode-tip catheter is very powerful; widespread use of it may potentially lead to more frequent cases of cardiac tamponade. Third, the technology at that time was not capable of making a 2-mm irrigated catheter. Eventually, a 3.5-mm electrode was selected.

Facing more and more VTs originating from intramural or epicardial sources, a 2mm-tip irrigated electrode catheter could provide better mapping accuracy and may accomplish what bipolar ablation or needle catheters can accomplish. Hopefully, one of the catheter manufacturers will reconsider the possibility of making a 2-mm irrigated catheter.

Question: Is high power, short RF application safe and effective?

Most of the left atrial posterior wall is quite thin. Ideally, AF ablation lesions should be wide but shallow so as to quickly complete the circumferential ablation lines with minimal risk of tamponade or collateral injury. One approach is to modulate lesion formation contributed by resistive heating and conductive heating. As

already discussed, resistive heating occurs immediately after RF current flows into the myocardium in contact with the tip electrode; conductive heating occurs relatively slowly and plays a major role in lesion growth. High power, short RF applications were intended to increase the contribution of resistive heating but decrease the contribution from conductive heating in hopes that the RF lesions would be wider and shallower. Different groups have reported safety and efficacy data. In general, they reported that lesions are wider and shallower than the standard RF lesions made by lower power (up to 35 watts) and longer duration. Importantly, the maximal diameter of the lesion is located near the endocardial surface, in sharp contrast to the lesion made by irrigated-tip catheters in which the maximal diameter of the lesion is located 3-4 mm below the endocardial surface (**Figure 3.11**). One of the protocols (90 watts, 4 seconds) was conducted using a new Biosense catheter (QDOT) in which there are 6 thermocouples in a 56-hole irrigated electrode. Three of the 6 thermocouples are located very close to the tip of the ablation electrode, providing almost real-time interface temperature feedback. As soon as the interface temperature exceeds 60°C, the RF generator is shut down immediately. Unless an EP laboratory is equipped with this special catheter, it is not advisable to use an irrigated catheter to attempt high power (>50 watts), short duration AF ablation due to the lack of real-time temperature feedback and safety data. Other groups have reported that shorter RF applications with 50 watts appear to be safe and as effective as the conventional approach.

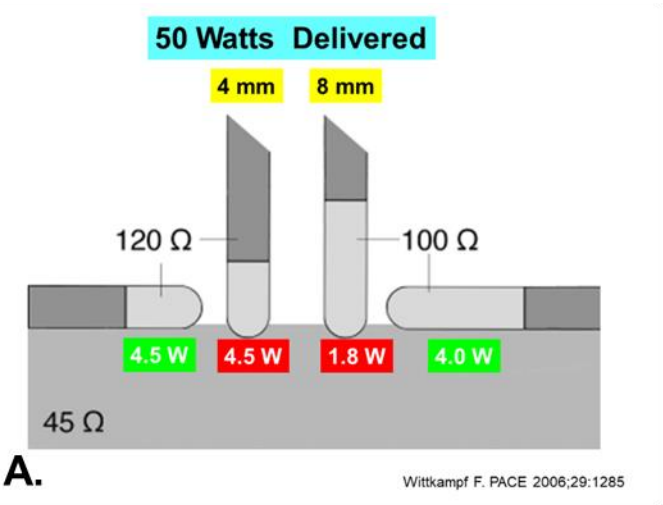
How high power, short RF applications make wider but shallower lesions is poorly understood because heat is dissipated equally in all directions. It is important to point out that resistive heating generates the heat source for conductive heating. To make a larger but shallower lesion, heat transfer has to occur preferentially along (or below) the irrigated endocardial surface but somehow be limited toward the epicardium. Based on 3 decades of experience in biophysical studies, the OU-EP group has not adopted the high power, short duration strategy because the safety margin is very narrow. Dr. Nakagawa's pre-clinical studies showed that the geometry of RF lesions formed by higher power, short duration RF applications were smaller both in the diameter at the electrode-tissue interface as well as the depth of the lesion. In addition, changes in the lesion geometry are greatly influenced by the mode and rate of irrigation. In the QDOT catheter, a modified mode of irrigation in the 56-hole catheter is used to make a larger but shallower lesion. In addition, high power of 90 watts, 70 watts and 50 watts mean very different heat sources. The duration of RF application for each power setting which is effective and safe remains poorly understood. Safety issues have to be addressed by carefully designed clinical trials, not by pooled, anecdotal experience. Meanwhile, using Ablation Index, monitoring changes in the unipolar EGM or visualizing RF lesion formation with intracardiac echo may help operators customize the duration of each high-power RF application.

Question: Is low irrigation flow ablation (e.g. 2 cc/min) safe?

A lasso-type ablation catheter was developed by Ablation Frontiers Inc. to provide rapid PV isolation. This decapolar catheter can deliver RF energy in both unipolar configuration (between an individual electrode and the reference patch) and bipolar configuration (between adjacent electrodes). Early clinical data were promising but MRI studies revealed a much higher incidence of silent stroke as compared to irrigated catheters. This device was never approved by the US Food and Drug Administration. With this lesson in mind and experience in irrigated catheter studies (**Figure 3.6C**), the OU-EP group has not adopted the low irrigation flow strategy. The safety concerns of this approach must be addressed by MRI studies showing no increase in silent stroke.

Should impedance drop be used as an indicator for RF lesion formation?

The answer is yes and no. It is the author's opinion that impedance drop by 5-10% at the beginning of the RF application might serve as a *moderately good* positive predictive value for lesion formation. The absence of impedance drop should alarm the operator to look into contact force and power. As discussed already, impedance is also determined by the electrode-tissue orientation, irrigation, blood flow and electrode size. It is not prudent to increase the power/force/time simply because the drop of impedance is less than what the operator expected.



A.

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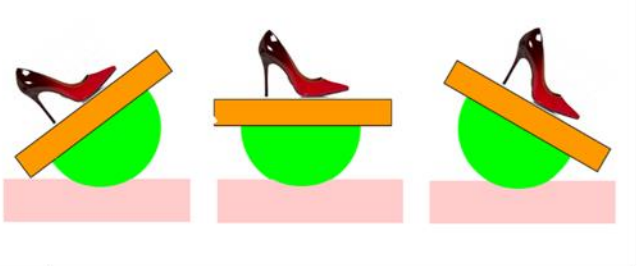

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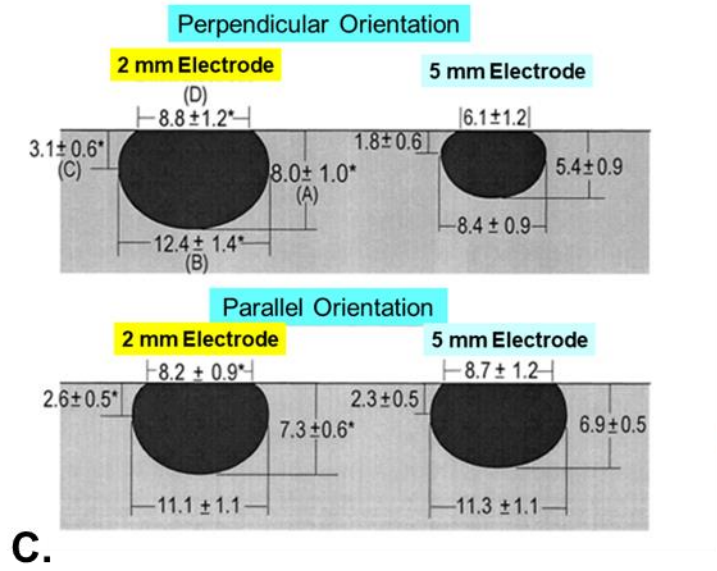


C.

Figure 3.13. Effects of ablation electrode length and electrode-tissue orientation on RF lesion formation. A. Comparing to a 4-mm electrode catheter, lesion formation created by an 8-mm electrode catheter depends more on the electrode-tissue orientation. **B**. The tip of a 2-mm electrode behaves like a half hemisphere. Regardless how it is rotated, the surface area of electrode-tissue interface remains stable. **C.** Diagram of lesion dimensions for a 2-mm and 5-mm irrigated electrode catheter (*P<0.05 between 2-mm and 5-mm ablation electrodes within same electrode-tissue orientation). Note that the RF lesion size made by a 2-mm electrode catheter was not affected by electrode-tissue orientation. *Modified with permission from: Wittkampf FH, Nakagawa H. Pacing Clin Electrophysiol. 2006 Nov;29(11):1285-97.*

Chapter 4:

Differential Diagnosis of Narrow-Complex and Wide-Complex Tachycardia

There have been many publications addressing differential diagnosis of wide-complex tachycardias (WCT) and narrow-complex tachycardias (NCT). Dr. Jackman's approach to differential diagnosis of tachycardias in the EP laboratory can be summarized as follows:

1. For NCT, Dr. Jackman delivers single or multiple ventricular extra-stimuli (VES) to evaluate how VES conduct to the atrium to reset the tachycardia. Dr. Jackman rarely uses the overdrive pacing or entrainment response for differential diagnosis of NCT.

2. For WCT, Dr. Jackman delivers single atrial extra-stimulus (AES) to evaluate how AES conduct to the ventricle to reset the tachycardia.

Recording a stable His bundle (HB) potential is paramount before attempting to induce tachycardia. The disappearance of the HB potential during tachycardia suggests that the HB potential may be dissociated from the tachycardia. Though this favors the diagnosis of VT, the diagnosis is not fully established because HB potentials may be buried within the local ventricular potential as occurs in antidromic AVRT. If during tachycardia, the HV interval becomes a negative value and the HB potential maintains a stable 1:1 relationship with the QRS complex, this tachycardia is most likely a His-Purkinje related tachycardia (e.g. fascicular VT, preexcited atrio-fascicular AVRT; **Figure 4.1)**.

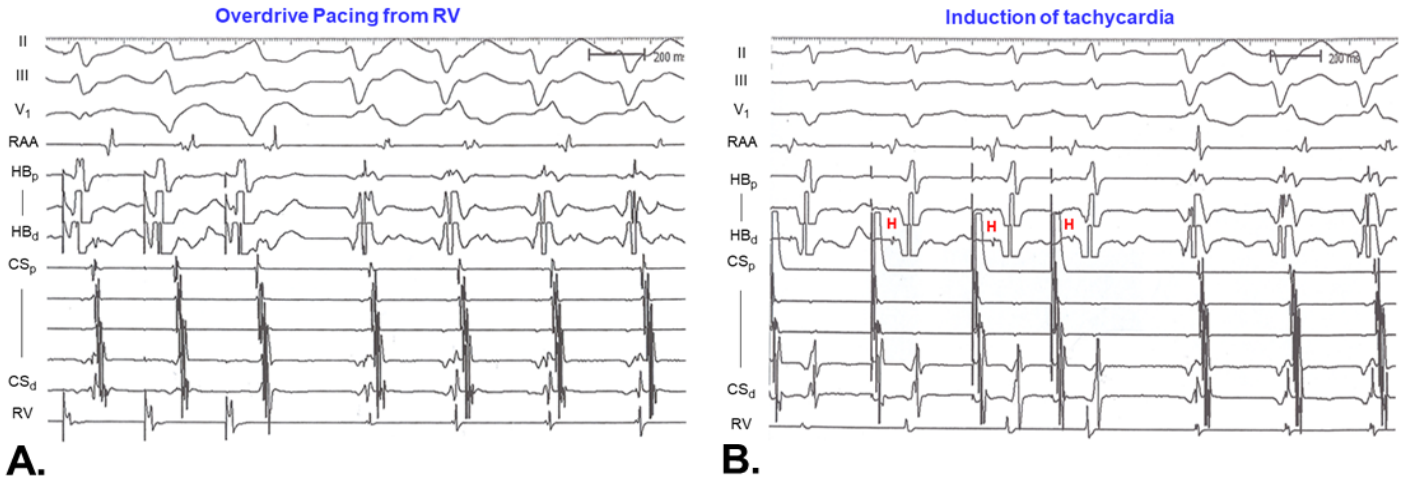


Figure 4.1. A 26 y/o female referred for failed AVNRT ablation. A. In a tachycardia with RBBB and superior axis, the AV relationship in tachycardia was 1:1. The overdrive pacing response was VAV with a long PPI, leading to the diagnosis of AVNRT with aberrant conduction. **B.** The tachycardia was initiated by an atrial extra-stimulus. However, the HB potential disappeared during tachycardia, indicating that this was not an SVT with aberrant conduction. VT originating in the left posterior fascicle was successfully ablated.

Sometimes, wobbling of the tachycardia cycle length (CL) causes difficulty in assessing if tachycardia has been perturbed by the pacing maneuver. Dr. Jackman always takes two CLs to average out the wobbling in CL while assessing the response to pacing maneuvers. When a reentrant tachycardia exhibits a constant "short-long-short-long" wobbling pattern, it often suggests that the reentrant circuit is conducting at its maximal capacity. An analogy is that a jogger is running on a circuit track at his/her maximal capacity. S/he slows down a bit when tired and then accelerates to the original speed. This deceleration and acceleration process constantly repeats itself.

Narrow-Complex Tachycardia

Before attempting any pacing maneuver, Dr. Jackman would compare the atrial activation sequence between the NCT and what parahisian pacing revealed earlier. This step underscores the importance of performing parahisian pacing at the beginning of the EP study so that the operator has the knowledge of the mechanism of each retrograde activation sequence. **Figure 4.2** summarizes Dr. Jackman's approach to differential diagnosis of NCT in the EP laboratory.

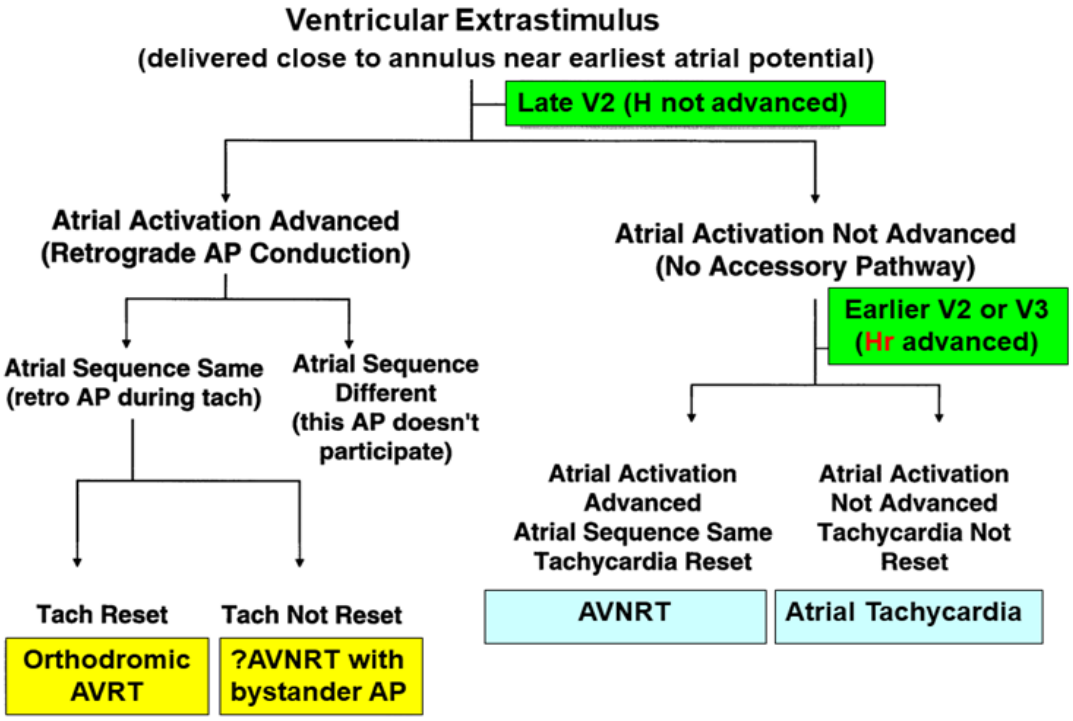


Figure 4.2. Dr. Jackman's algorithm of differential diagnosis for NCT in the EP laboratory. Hr: retrograde HB potential.

Step-1. Deliver VES during tachycardia at a site that fulfills the following two criteria:

(a). At the base of the ventricle: Because both the AVNRT and AVRT reentrant circuits are located near the AV annulus, delivering VES at the base of the ventricle ensures that the VES wave front can easily engage the reentrant circuit.

(b). As close to the site of earliest atrial activation as possible: This practice is to ensure that VES can engage the retrograde limb of the reentrant circuit quickly. For example, if the site of earliest atrial activation is adjacent to the HB region, VES are delivered to the anteroseptal RV (parahisian position). If the site of earliest atrial activation is adjacent to the posteroseptal area, VES are delivered to the basal posteroseptal RV.

There have been many patients referred to the OU-EP group after multiple failed SVT ablations in which the site of earliest atrial activation was in the posteroseptal area. A common scenario is that the arrhythmia was ablated in one procedure as "atypical" AVNRT but as AVRT in the other. Such diagnostic mistakes were made because the pacing site (RV apex) was too far from the posterior septum; the paced wave front failed to engage the retrograde limb of the accessory pathway (AP), erroneously excluding the diagnosis of orthodromic AVRT. For the aforementioned reasons, Dr. Jackman does not deliver VES at the RV apex for differential diagnosis of NCT.

Step-2. Evaluate how VES affect VA conduction and reset the tachycardia

There are only two possible routes for a ventricular wave front to propagate to the atrium: AP and AVN. If the H-H interval is not perturbed by the VES, the HB potential is still activated in the antegrade direction

(**Figure 4.3A**). HB cannot be used immediately for retrograde conduction. Therefore, for a "His-synchronized" VES or "VES on His" to reset a tachycardia, it must conduct to the atrium through an AP that serves as the retrograde limb of the AVRT circuit if the atrial activation sequence remains identical. However, Dr. Jackman does not fully agree with the concept of "VES on His" or "His synchronized VES". As long as the H-H interval is not perturbed by the VES, it indicates that the HB is used for antegrade conduction. If a VES is delivered to a site distant from the AP, it may have to be 50 ms earlier than the HB potential to reset the tachycardia but this VES is still legitimate as long as the HH interval remains unchanged. This practice is particularly important if the ventricular pacing site is distant from the ventricular end of the AP (e.g. RV apex pacing for a left anterior AP; **Figure 4.3B**).

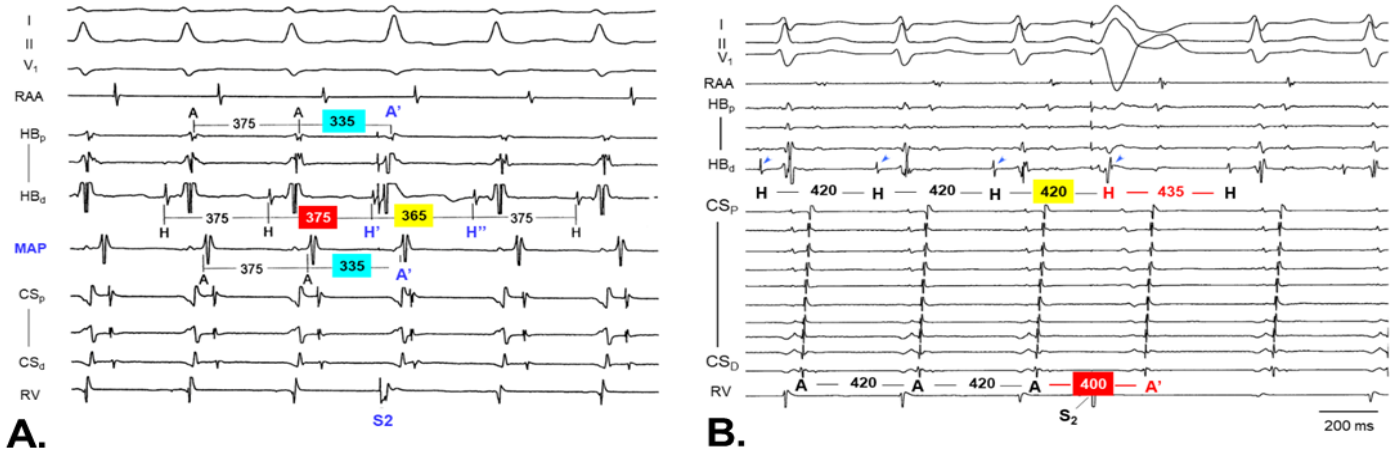


Figure 4.3. VES (S2) reset AVRT. A. A VES was delivered to the basal anteroseptal RV during the HB refractory period, adjacent to the site of earliest atrial activation. The H-H' interval remained unchanged (375 ms, highlighted in red color), indicating that the HB potential (H') was activated in the antegrade direction. This VES advanced the next atrial activation timing (AA') by 40 ms and reset the tachycardia (H'-H"=365 ms). In addition, the atrial activation sequence was not affected by the VES**. B. A VES earlier than the HB potential reset the NCT**. VES delivered to the anteroseptal RV, 40 ms before the HB potential, advanced the next atrial timing by 20 ms (A-A'=400 ms) and reset the NCT, proving the diagnosis of orthodromic AVRT. Note that the timing of the VES can be substantially earlier than the inscription of the HB potential. If the HB potential is on time, it indicates that the HB is activated in the antegrade direction; the AVN cannot be used for retrograde conduction by the VES.

If single VES (V_2) fail to perturb the tachycardia, Dr. Jackman delivers double VES (V_2V_3) or triple VES ($V_2V_3V_4$) to a site adjacent to the site of earliest atrial activation to reset the tachycardia (**Figure 4.4**). The first VES (V_2 of V_2V_3) or first two VES (V_2V_3 of $V_2V_3V_4$) serve the purpose of shortening the ventricular refractory period to allow the last VES to engage the retrograde limb of the reentrant circuit. It is uncommon that a double VES (V_2V_3) fails to verify or exclude the diagnosis of AVRT because the ventricle is part of the reentrant circuit. AVNRT, however, may require 3 or more VES to allow the last extra-stimulus to engage the retrograde limb of the reentrant circuit.

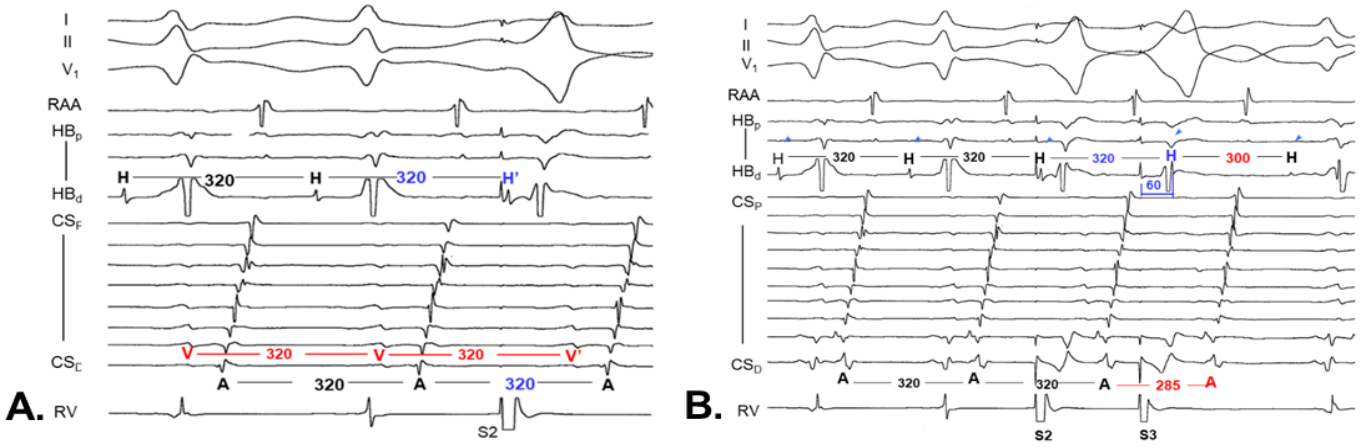


Figure 4.4. Double VES to reset a NCT (CL=320 ms). A. "His synchronized VES" or "VES on His" delivered to the anteroseptal RV failed to perturb the NCT. Note that the ventricular potential near the site of earliest atrial activation (CS-D) was not advanced by this VES (V-V'=320 ms). This VES therefore failed to engage the ventricular end of the AP. **B.** Two VES were delivered. S3 was 60 ms earlier than the HB potential but advanced the next atrial activation timing and reset the NCT, proving the diagnosis of orthodromic AVRT.

If a VES can advance the next atrial activation timing during the HB refractory period but fails to reset the tachycardia, the operator must consider the possibility that this AP does not participate in the tachycardia. This pathway is therefore a bystander. In general, a bystander AP during AVNRT or AT is relatively rare; it requires repeated attempts of perturbation before one can conclude that this AP is a bystander. If a NCT is difficult to reset, operators may consider the following pacing maneuvers: (1) shortening the coupling interval of the VES. If earlier VES (V2) fall into the ventricular refractory period but still fail to reset the tachycardia, Dr. Jackman would deliver double VES (V2V3) or triple VES (V2V3V4) to reset the tachycardia (**Figure 4.5**), (2) moving the RV pacing catheter to a site in close proximity to the site of earliest atrial activation (presumably adjacent to the ventricular end of the AP; **Figure 4.6**).

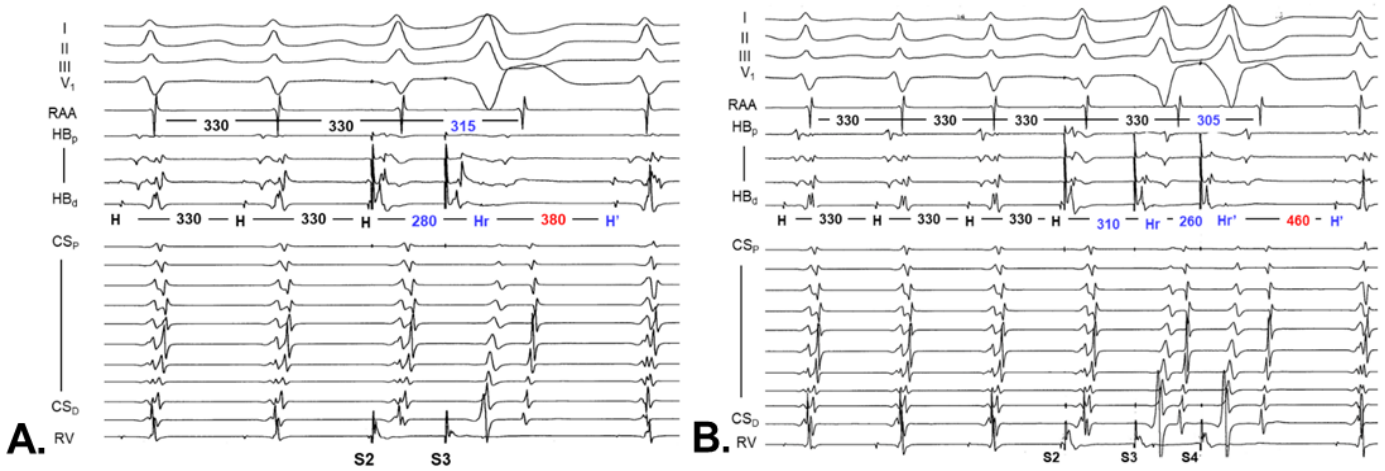


Figure 4.5. Multiple VES to prove the mechanism of a NCT. A. S3 advanced the retrograde His (Hr) by 50 ms and the next atrial activation timing by 15 ms but failed to reset tachycardia (H-Hr + Hr-H'= 660=330 ms x2). However, one cannot claim this tachycardia cannot be reset by VES until more attempts are made such as shorter coupling intervals or delivering S4. **B.** Earlier S3 fell into the ventricular refractory period (not shown). S4 was delivered which advanced the retrograde HB potential and reset the tachycardia, proving that it was AVNRT (HHr + HrHr'+ Hr'H'=310+260+460 ms=1030 ms >330 ms x3).

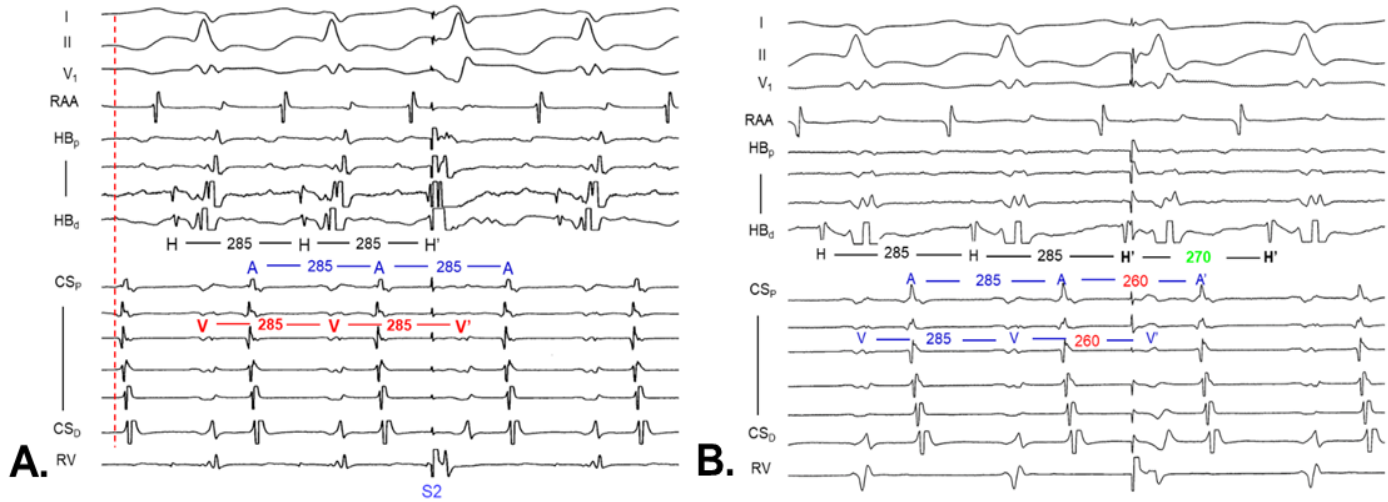


Figure 4.6. The location where VES are delivered matters. A. NCT (CL 285 ms) with the site of earliest atrial activation in proximal CS (vertical red line). A VES (S2) delivered to the anteroseptal RV during the HB refractor period (HH' interval=285 ms) failed to perturb the NCT. Note that the VV' interval adjacent to the site of earliest atrial activation was not advanced by the VES (V-V' =285 ms), indicating that this His-synchronized VES was not early enough to engage the ventricular end of the AP to reset the NCT. **B.** The RV catheter was moved from the anteroseptal RV to midseptal RV. Another His-synchronized VES delivered to that site advanced the V-V' interval adjacent to the site of earliest atrial activation by 25 ms (V-V'=260 ms), thereby advancing the next atrial activation by 25 ms (A-A'=260 ms) and resetting the NCT. The diagnosis of orthodromic AVRT was made.

Step-3. Deliver early or multiple VES to engage the AVNRT reentrant circuit

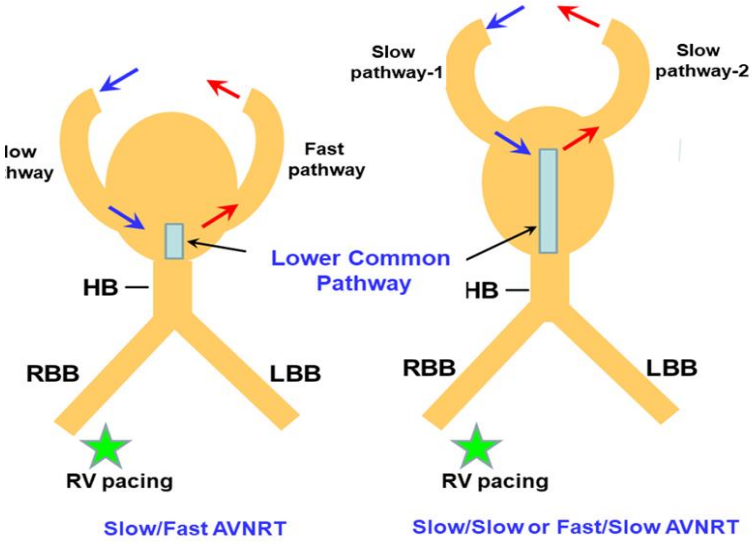


Figure 4.8. Lower common pathway of slow/fast AVNRT (left panel) and slow/slow AVNRT (right panel). Fast/slow and slow/slow AVNRT usually have a long lower common pathway. Therefore, paced ventricular wave front has to advance the retrograde HB potential by a large amount to engage the AVNRT reentrant circuit.

To verify the diagnosis of AVNRT, Dr. Jackman delivers progressively earlier VES from a site adjacent to the site of earliest atrial activation. For example, if the site of earliest atrial activation is in the posteroseptal area, VES will be delivered to the basal posteroseptal RV. If the local ventricular timing *near the site of earliest atrial activation* has been advanced by 70-80 ms, it should be able to engage the ventricular end of the AP and perturb an orthodromic AVRT. If such early VES still fail to affect the atrial activation timing, it is extremely unlikely that this tachycardia is AVRT except the rare occasion when the AP exhibits significant decremental conduction property (see discussion below). If the atrial activation timing cannot be advanced and tachycardia cannot be reset until the timing of the retrograde HB potential is advanced enough, it proves that this tachycardia is related to AVN and cannot be an atrial tachycardia (**Figure 4.7**). The diagnosis of atrial tachycardia is not established until both AVRT and AVNRT are ruled out.

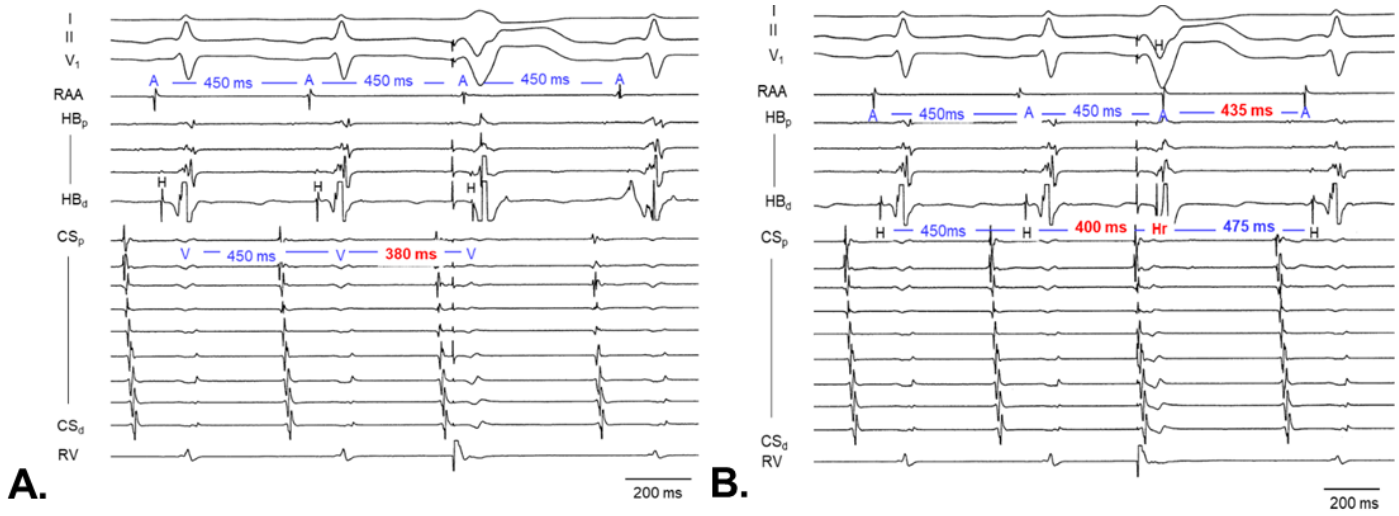


Figure 4.7. VES response in fast/slow AVNRT. A. VES was delivered to the basal posteroseptal RV, adjacent to the proximal CS where the site of earliest atrial activation was located. VES advanced the local ventricular activation at CS-p by 70 ms without affecting the next atrial activation, making the diagnosis of orthodromic AVRT using a posteroseptal AP for retrograde conduction extremely unlikely. **B.** An earlier VES advanced the retrograde HB potential (Hr) by 50 ms and the atrial activation timing. Tachycardia was reset. This observation indicated that the tachycardia was AVN-dependent and was AVNRT.

In an AVNRT reentrant circuit, the lower common pathway represents the tissue between the HB and the junction of the end of the antegrade limb and the beginning of the retrograde limb (**Figure 4.8; Figure 7.5).** For slow/fast AVNRT, the lower common pathway is usually very short; the tachycardia usually can be reset if the retrograde HB potential is advanced by 15-20 ms. That is, “HA linking” is easy to demonstrate. For slow/slow or fast/slow AVNRT, the lower common pathway is usually long, indicating a long distance between the HB and the reentrant circuit. In this scenario, the retrograde HB potential may need to be advanced by >70 ms to advance the next atrial activation timing and reset the tachycardia. This is a very important point because many slow/slow or fast/slow AVNRT were misdiagnosed as AT after a run of RV overdrive pacing

that was not long enough to allow the paced ventricular wave front to engage the retrograde limb of the AVNRT reentrant circuit.

For a NCT whose atrial activation sequence and short VA interval suggests slow/fast AVNRT, the author prefers to start with delivering runs of double VES from the basal anteroseptal RV because single VES often encounters the ventricular refractory period without affecting the tachycardia. The first VES of each run is delivered 10 ms after the HB potential to shorten the ventricular refractory period to allow the 2nd VES (V3) to advance the retrograde HB potential and subsequently engage the retrograde limb of the AVNRT reentrant circuit. The V2-V3 coupling interval is first set 20-30 ms shorter than the tachycardia CL and is progressively shortened by 10 ms each time. If V3 reaches the ventricular refractory period without perturbing the NCT, runs of triple VES (V2V3V4) are delivered, which almost always confirms the diagnosis of AVNRT.

Operators must carefully compare the atrial activation sequence following the VES to that of the tachycardia. If differences exist, one must interpret the resetting response carefully. If the tachycardia can be reset when the HB is in the refractory period, it indicates that the 2nd atrial activation sequence exposed by the VES is mediated by retrograde AP conduction. In this scenario, the results of parahisian can offer great help. If parahisian pacing indicates that the atrial activation sequence of the tachycardia is caused by AP conduction, the 2nd atrial activation sequence indicates the presence of a 2nd AP which does not participate in this tachycardia but may participate in another tachycardia. If parahisian pacing indicates that the atrial activation sequence of the tachycardia is caused by AVN conduction, the 2nd atrial activation sequence indicates the presence of a bystander AP, not participating in this AVNRT.

Dr. Jackman's resetting protocol works very well and provides an accurate diagnosis with a low incidence of terminating the tachycardia. If tachycardia happens to be terminated by this resetting protocol, termination is almost always diagnostic (**Figure 4.9**). Therefore, Dr. Jackman's first choice for differential diagnosis of NCT is not overdrive pacing or entrainment responses, which can easily terminate the NCT without revealing the tachycardia mechanism.

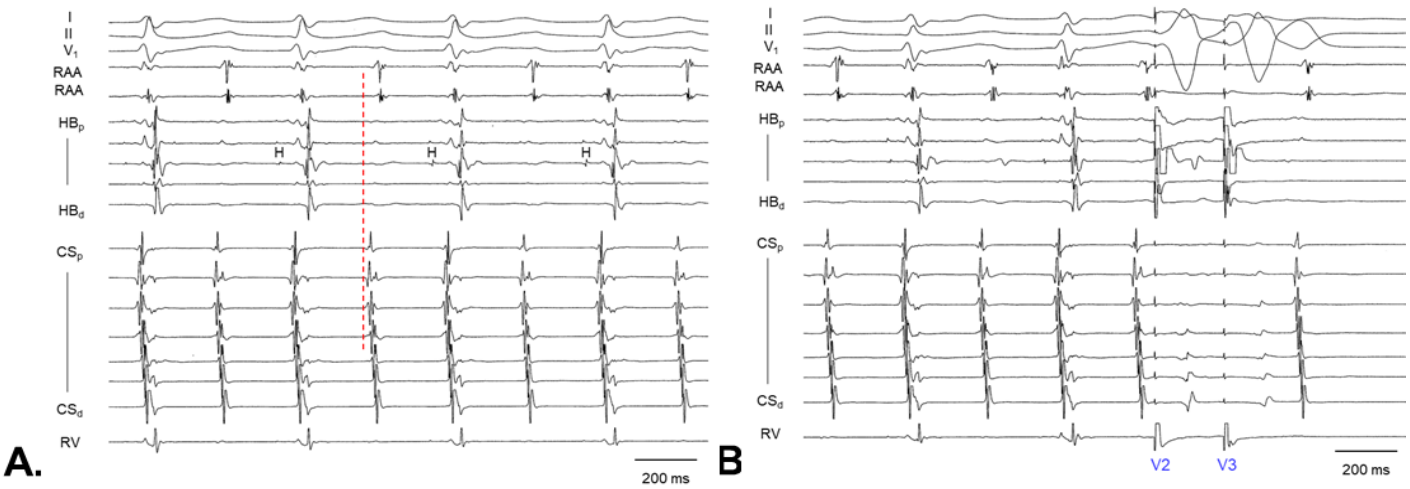


Figure 4.9. Double VES verified the diagnosis of AVNRT. A. NCT with 2:1 AV conduction was induced; AVRT was excluded by 2:1 AV conduction. The activation timing in the HB area and proximal CS was equally early (red line). **B.** Double VES was delivered to midseptal RV, adjacent to the site of earliest atrial activation. The first VES (V2) terminated the NCT with VA block (without reaching the atrium), excluding AT. By exclusion, the diagnosis is AVNRT. The 2nd VES conducted to the atrium with the same activation sequence as the NCT. Parahisian pacing performed immediately after tachycardia termination also verified that retrograde conduction was mediated by the AVN (not shown), which also supports the diagnosis of AVNRT.

A significant number of patients were referred to Dr. Jackman's practice after failed ablation under the wrong diagnosis. Dr. Jackman's resetting protocol takes more time to make the diagnosis, but it is highly effective when the mechanism of tachycardia is difficult to discern. Simply by moving the RV catheter to the basal posteroseptal RV and delivering VES there, it becomes very straightforward to discern slow/slow or fast/slow AVNRT from orthodromic AVRT using a posteroseptal AP for retrograde conduction (**Figure 4.10A**).

On rare occasions, retrograde conduction may exhibit significant decremental property (e.g. permanent junctional reciprocating tachycardia, PJRT). In this case, earlier engagement of the ventricular end of the AP leads to decremental AP conduction, neutralizing the expected earlier atrial activation timing. The operator may interpret the result as “VES cannot advance the next atrial activation timing” and erroneously excludes the diagnosis of orthodromic AVRT. However, if progressively earlier VES are delivered, NCT can be reset or terminated while the HB is still activated in the antegrade direction. The diagnosis of orthodromic AVRT can therefore be made (**Figure 4.10B-D**). Of note, overdrive pacing and entrainment often produce confusing responses (pseudo VAAV response and a very long PPI) as well. This unique problem underscores the importance of performing parahisian pacing and decremental RV pacing at the beginning of the procedure; an AP with decremental conduction property can easily be identified in this way.

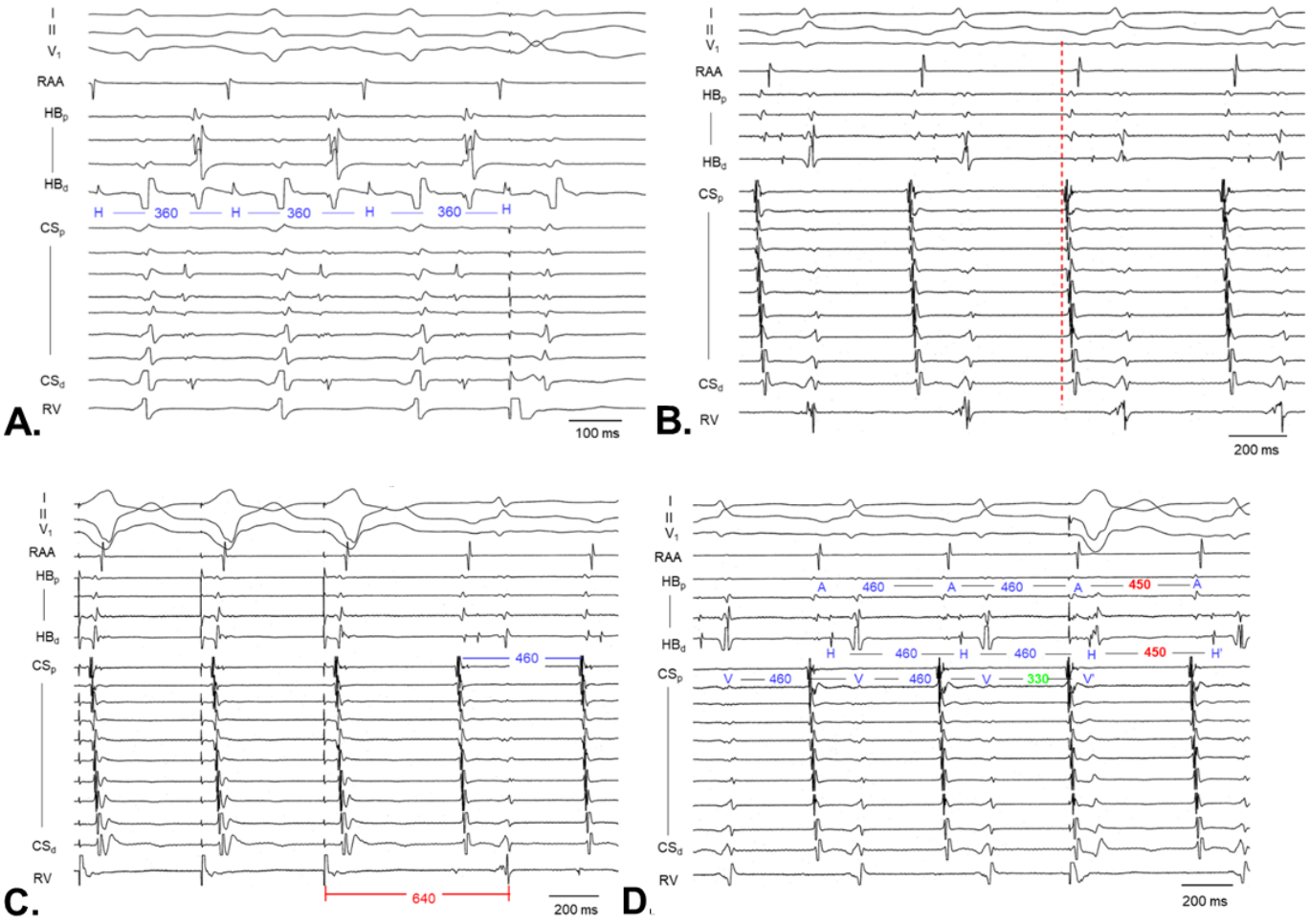


Figure 4.10. A. This female patient had two prior ablation procedures. Ablation was performed under the diagnosis of atypical AVNRT in one procedure and posteroseptal AP in the other. VES delivered from basal posteroseptal RV, adjacent to the site of earliest atrial activation, terminated the tachycardia when the HB potential was activated in the antegrade direction (HH interval stable at 360 ms), verifying the diagnosis of orthodromic AVRT. **B.** In another patient with incessant PJRT (permanent junctional reciprocating tachycardia) caused by a right posteroseptal AP with decremental conduction properties. **C.** Ventricular overdrive pacing resulted in a pseudo VAAV response and the PPI was 180 ms longer than the tachycardia CL, suggesting that this tachycardia was not orthodromic AVRT. **D.** VES delivered to the basal posteroseptal RV, adjacent to the AP, advanced the ventricular activation near the AP by 130 ms (V-V’=330 ms) but only advanced the next atrial activation by 10 ms due to decremental conduction property of this AP. Tachycardia was reset as well (not shown here).

Resetting with fusion

Sometimes, recording a stable HB potential can be a challenge. If a VES resets the NCT and the morphology of the VES represents fusion of the QRS complex of the NCT and ventricular pacing, it is obvious

that the HB potential must be activated in the antegrade direction to produce a QRS complex that fuses with the paced QRS complex (**Figure 4.11**). The VES wave front cannot advance the next atrial activation through retrograde AVN conduction. The diagnosis of AVRT is therefore confirmed. For this reason, when evaluating the response to overdrive pacing, the author first focuses on how the NCT is perturbed by overdrive pacing before examining the VAV/VAAV response. It is very common to observe that NCT was perturbed by a paced beat that happened to be delivered during the HB refractory period or it was a fusion beat.

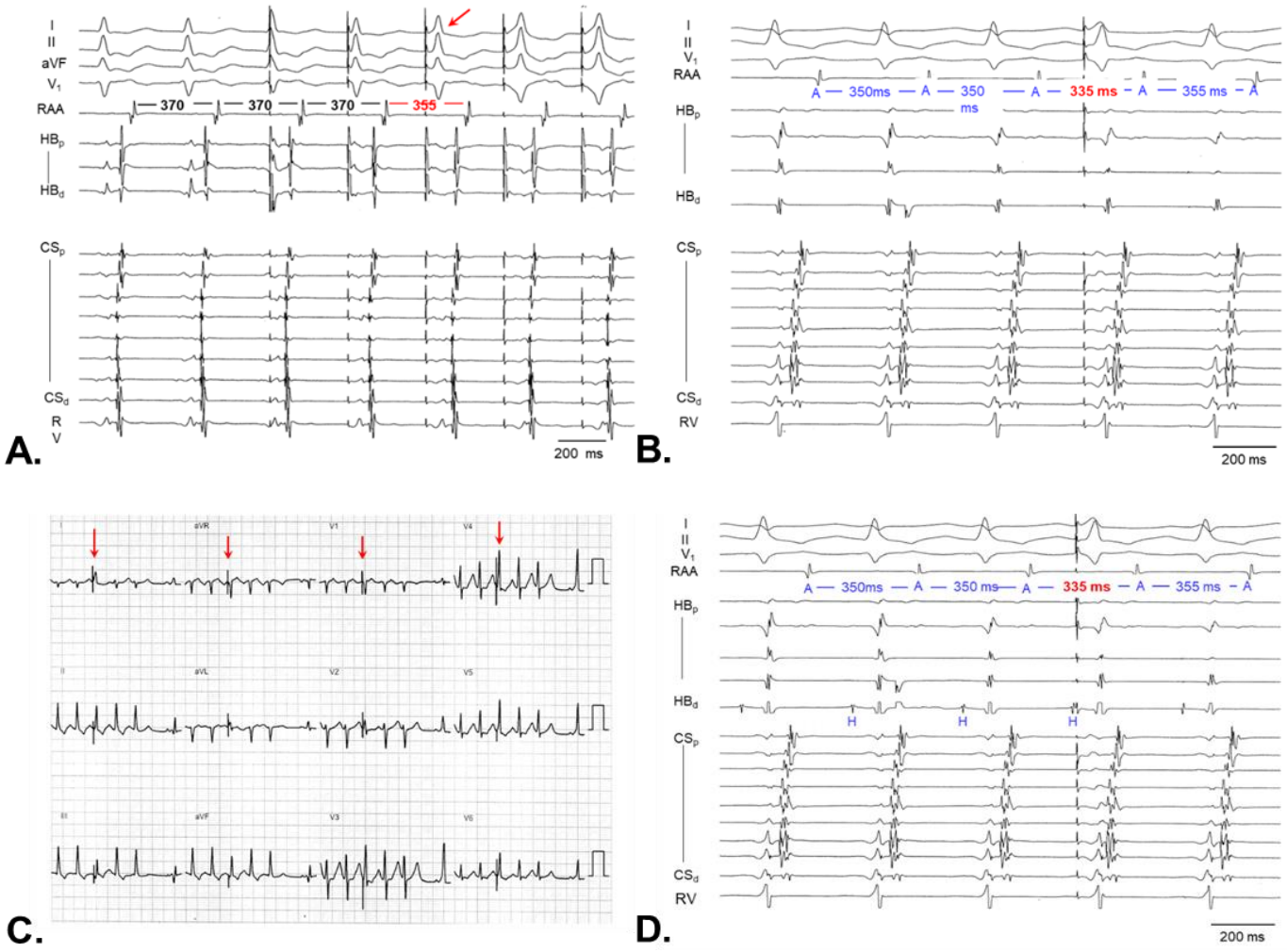


Figure 4.11. Proof of AVRT by ventricular fusion. A. The 3rd paced beat (red arrow) advanced the atrial timing by 15 ms. Note that the QRS morphology represented fusion of the tachycardia QRS complex and fully captured QRS complex. **B-C.** In another patient with AVRT, a HB potential was not recorded. A VES advanced the next atrial activation by 15 ms and reset tachycardia. 12-ECG showed that the morphology of this VES (red arrow) was similar to but different from the QRS complex during tachycardia, indicating that this was a fusion beat. **D.** After repositioning of the HB catheter, a HB potential was recorded. This VES was indeed delivered during the HB refractory period.

Dr. Jackman rarely uses an interval to confirm or exclude a diagnosis. He prefers to use physiological explanations to confirm or exclude a diagnosis because measuring an interval is prone to error. The same interval measured at 100 mm/sec sweep speed may be different from that measured at 200 mm/sec speed. For this very reason, the sweep speed of the recording and mapping systems in the OU-EP laboratory is always 200 mm/sec. The following observation, “a VA interval shorter than 70 ms excludes AVRT,” has been taught like a doctrine for decades. If an operator finds a VA interval of 66 ms or 74 ms, how certain is s/he that this VA interval excludes or does not exclude AVRT? A recent study indicates that in pediatric population, the VA interval of orthodromic AVRT can be as short as 50 ms. Another problem with using an interval to make differential diagnosis is that such an interval is usually made arbitrarily according to the result of

statistical analysis in that study. It is advisable to read the original study carefully to understand the strength and weakness of making a differential diagnosis using the proposed interval.

Issues related to overdrive pacing or entrainment response

Although overdrive pacing and the resultant entrainment response are not Dr. Jackman's choice maneuver for differential diagnosis, they can be very helpful when the tachycardia CL wobbles significantly, making the resetting response difficult to interpret. It cannot be overemphasized that the CL of pacing must be chosen wisely. It is well known that the return CL after entrainment greatly depends on the pacing CL because shorter pacing CLs tend to encroach upon the refractory period of the reentrant circuit, leading to a longer return CL and the false impression that the pacing site is not adjacent to the reentrant circuit. The author prefers to set the pacing CL 7% shorter than the tachycardia CL, based on a prior publication by Prof. Mark Josephson's group.

There are three common pitfalls of overdrive pacing that the operator should be aware. First, in some NCTs with a long VA interval (e.g. PJRT) or a long HA interval (e.g. fast/slow AVNRT), the entrained atrial EGM can be one beat behind the paced ventricular beats, leading to the false impression of a VAAV response (**Figure 4.10, 4.12A**). Second, in some cases, overdrive pacing in fact never entrained the NCT but created a false VAV or VAAV response (**Figure 4.12B**). Although NCT that cannot be entrained by RV pacing is almost always an atrial tachycardia, failure to entrain does not exclude the diagnosis of AVNRT since the paced ventricular wave front must reach the HB before it can engage the retrograde limb of the AVNRT reentrant circuit (see discussion below). Third, if overdrive pacing terminated the tachycardia, one cannot see the VAV or VAAV response. However, how the tachycardia is terminated may provide a diagnostic clue. The author first examines if the paced beat that terminated the tachycardia was delivered during the HB refractory period or if the paced beats resulted in a fusion beat. If it is, the diagnosis is AVRT (**Figure 4.13**).

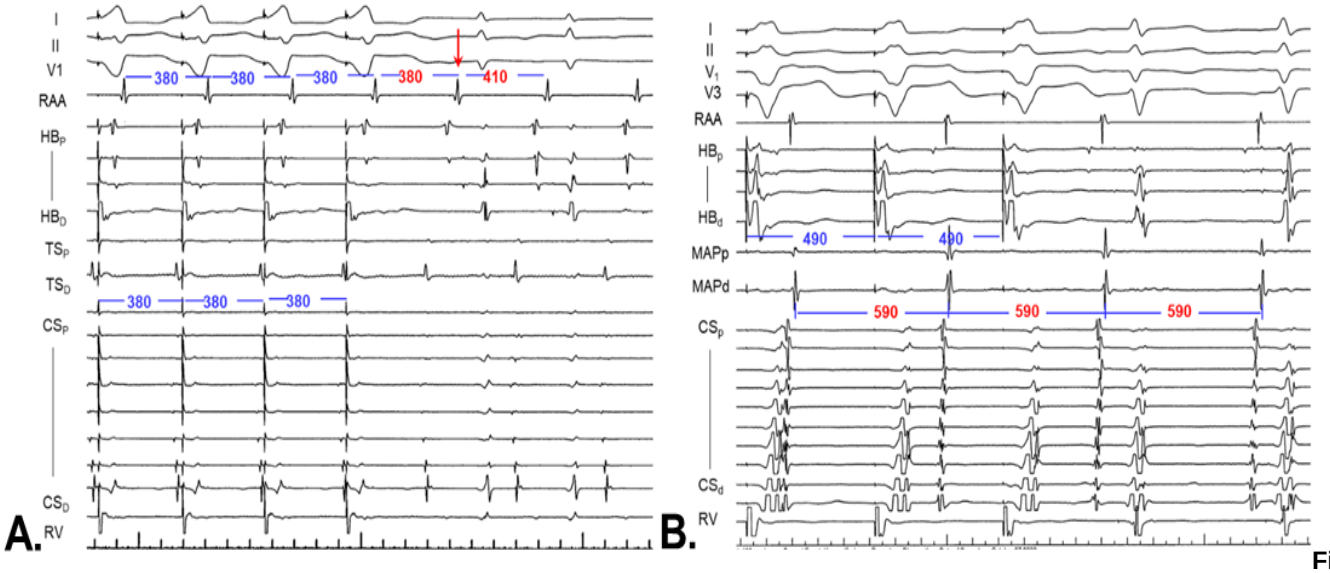


Figure 4.12. Common pitfalls of ventricular overdrive pacing. A. Ventricular overdrive pacing appeared to result in a VAAV response. Because of this long RP tachycardia, the last entrained beat (red arrow) was one beat behind the last paced ventricular beat. Indeed, this was a VAV response. This SVT was fast/slow AVNRT. **B.** Ventricular overdrive pacing resulted in a VAV response, suggestive of AVNRT or AVRT. However, the SVT (CL 590 ms) was never entrainment by RV pacing (490 ms). This SVT was a focal atrial tachycardia.

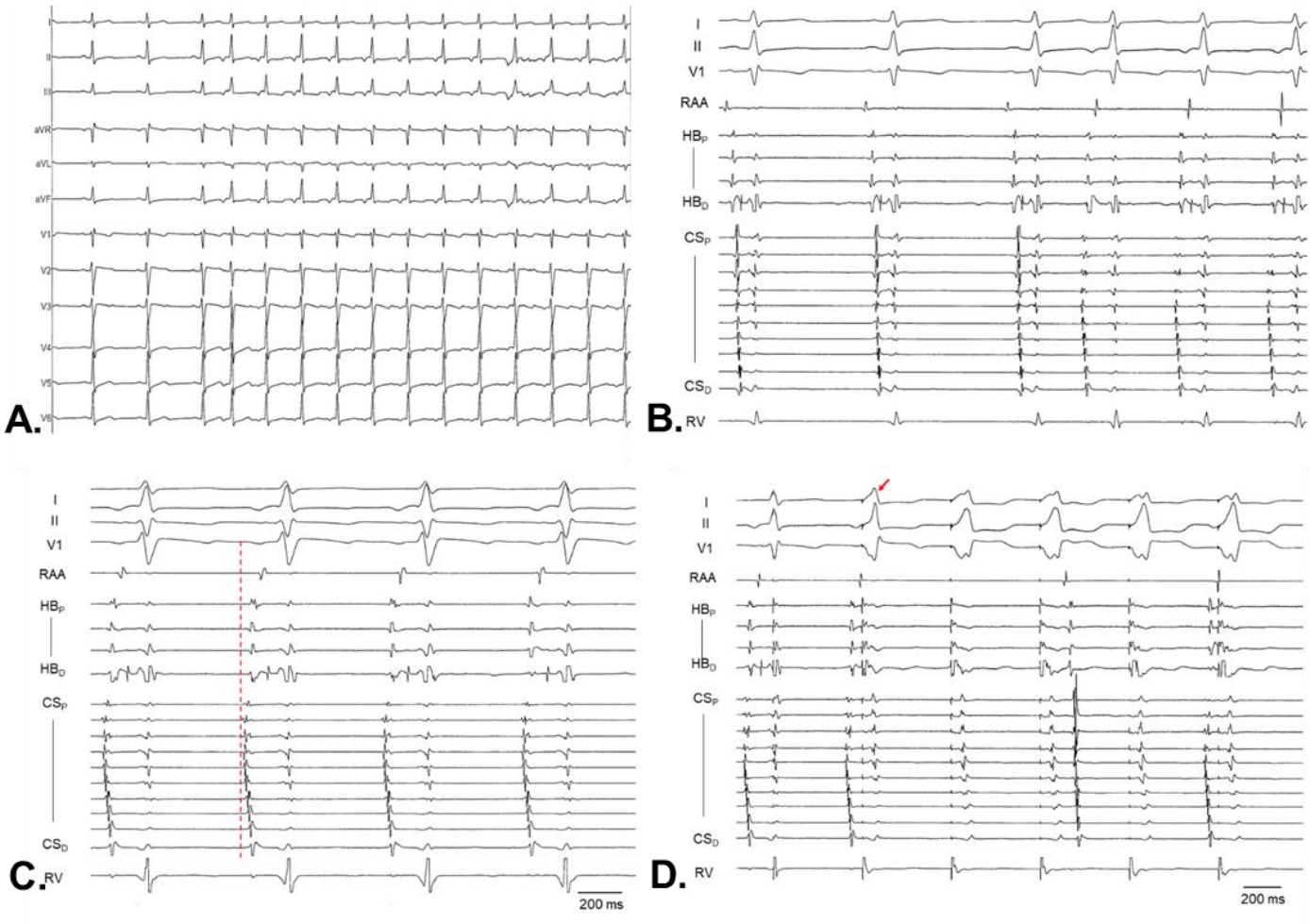


Figure 4.13. A long RP tachycardia was always terminated by RV overdrive pacing. A and B. Spontaneous initiation of a long RP tachycardia without the requirement of a premature atrial beat or AH/AV prolongation. Two prior ablations were performed under the diagnosis of focal AT. **C.** The site of earliest atrial activation was in proximal CS (vertical red line). **D.** Attempts to entrain this tachycardia repeatedly terminated the tachycardia. The paced beat that terminated the tachycardia was a fusion beat (red arrow), proving the diagnosis of AVRT.

Common mistakes we electrophysiologists make:

<u>The author compiled these observations by reading the reports of prior failed ablations.</u>

Mistake 1: Logic mistakes

Read the following inference:

"All pigs are fat". Therefore, if Sunny Po is fat, Sunny Po is a pig.

This inference is clearly wrong but we electrophysiologists may make similar mistakes such as misusing a finding with a high positive predictive values as a finding with a high negative predictive values. The author had seen the following logic mistakes multiple times:

"The VA interval of orthodromic AVRT is always > 70 ms"

Therefore, if the VA interval of an SVT is >70 ms, it is not a slow/fast AVNRT.

Mistake 2: If "VES on His" or "His synchronized VES" fails to perturb the SVT, it excludes the diagnosis of AVRT

For a VES to engage the ventricular end of an AP, it has to overcome the refractoriness of the intervening tissue between the pacing site and the AP. "VES on His" or "His synchronized VES" only means that the HB potential is activated in the antegrade direction and VES cannot immediately use the HB for retrograde conduction. Therefore, if "VES on His" can advance the next atrial activation timing and reset the NCT, it proves that an AP is used for retrograde conduction. However, if "VES on His" fails to advance the local ventricular activation adjacent to the site of earliest activation, it is not possible for this VES to prematurely engage the ventricular end of the AP (**Figure 4.4 and 4.5**). Many of us fail to deliver VES earlier than the HB potential and draw a wrong conclusion to exclude orthodromic AVRT. In this scenario, the correct maneuver is to deliver earlier VES or move the pacing catheter site closer to the AP in order to advance the local ventricular activation near the AP to engage the ventricular end of the AP**.** As already discussed, a VES can be as early as it needs to be as long as the operator is sure that HB potential is activated antegradely. Another trick that can be used when the HB potential is obscured by local ventricular activation is to examine if the VES that reset the NCT is a fusion beat. If fusion is observed, it proves that the HB potential is activated antegradely and the diagnosis must be orthodromic AVRT (**Figure 4.11).**

Mistake 3: Operators are confused by diagnostic criteria that "confirm or exclude" a diagnosis vs. "favor or disfavor" a diagnosis.

The author vividly remembers several occasions in fellows' training courses conducted by master educators such as Dr. Mark Josephson, Dr. Eric Prystowsky, Dr. George Klein and Dr. Mel Scheinman. We were pressed to answer the following question: do you think this observation confirms, excludes or suggests your diagnosis?

'If NCT is induced with an AH jump, this NCT is more likely to be an AVNRT or AVRT."

This observation "suggests" the diagnosis of AVNRT or AVRT; however, there are many cases of AT that are induced concurrently with an AH jump. The diagnosis of AT should not be excluded just because a concurrent AH jump at the moment of induction. On the other hand, there are many cases of AVNRT or AVRT that are induced without a concurrent AH jump. AVNRT or AVRT cannot be excluded by the absence of a concurrent AH jump.

If no confirmatory finding is observed or elicited, the next step is to try pacing maneuvers to elicit multiple lines of supportive evidence. Making the diagnosis by one supportive finding can be misleading. The supportive evidence should be reproducible. For example, if *sustained* NCT *repeatedly* terminated spontaneously with a P wave, it strongly suggests that this tachycardia is not an AT because it would require an AT to terminate at the same time of AV block. If termination of AT and AVB occurs once, it can be a fluke. If they occur concurrently multiple times, it is very unlikely to be an AT. However, it should be noted that non-sustained AT with unstable CL can terminate with AVB repeatedly. If the CL is stable, *sustained* AT should not repeatedly terminate with a P wave.

Mistake 4: AVNRT is excluded if VA dissociation is observed or RV pacing cannot entrain the NCT.

In AVNRT, the ventricle is not part of the reentrant circuit. For a paced wave front to engage the retrograde limb of the AVNRT circuit, the wave front must overcome the refractoriness of the intervening tissue between the pacing site and the peripheral Purkinje system, engage the peripheral Purkinje system, conduct to the His bundle (evidenced by advancing the timing of the retrograde HB potential) and eventually engage the retrograde limb of the AVNRT circuit. Significant conduction delay in any of the aforementioned steps or cumulative conduction delay from these steps may cause the paced wave front not to engage the AVNRT reentrant circuit, leading to the wrong impression that the NCT cannot be entrained or perturbed by RV pacing. The diagnosis of AVNRT is mistakenly excluded (**Figure 4.14**).

The most common mistake is that the duration of RV pacing is too short, not allowing the ventricular wave front to overcome the obstacles mentioned above to engage the retrograde limb of the AVNRT circuit. Dr. Jackman does not exclude the diagnosis of AVNRT unless the retrograde HB potential has been entrained for 3-4 beats but NCT still cannot be entrained. This is based on the assumption that if retrograde HB potential is entrained by RV pacing, the wave front should be able to entrain the AVNRT reentrant circuit very quickly (HA linking). Failure to do so excludes the diagnosis of AVNRT. Notably, slow/fast AVNRT usually has a short lower common pathway. As soon as the retrograde HB potential is entrained, AVNRT is entrained. Fast/slow and slow/slow AVNRT which have a long lower common pathway may require the retrograde HB potential to be entrained for 3-4 beats before entraining AVNRT.

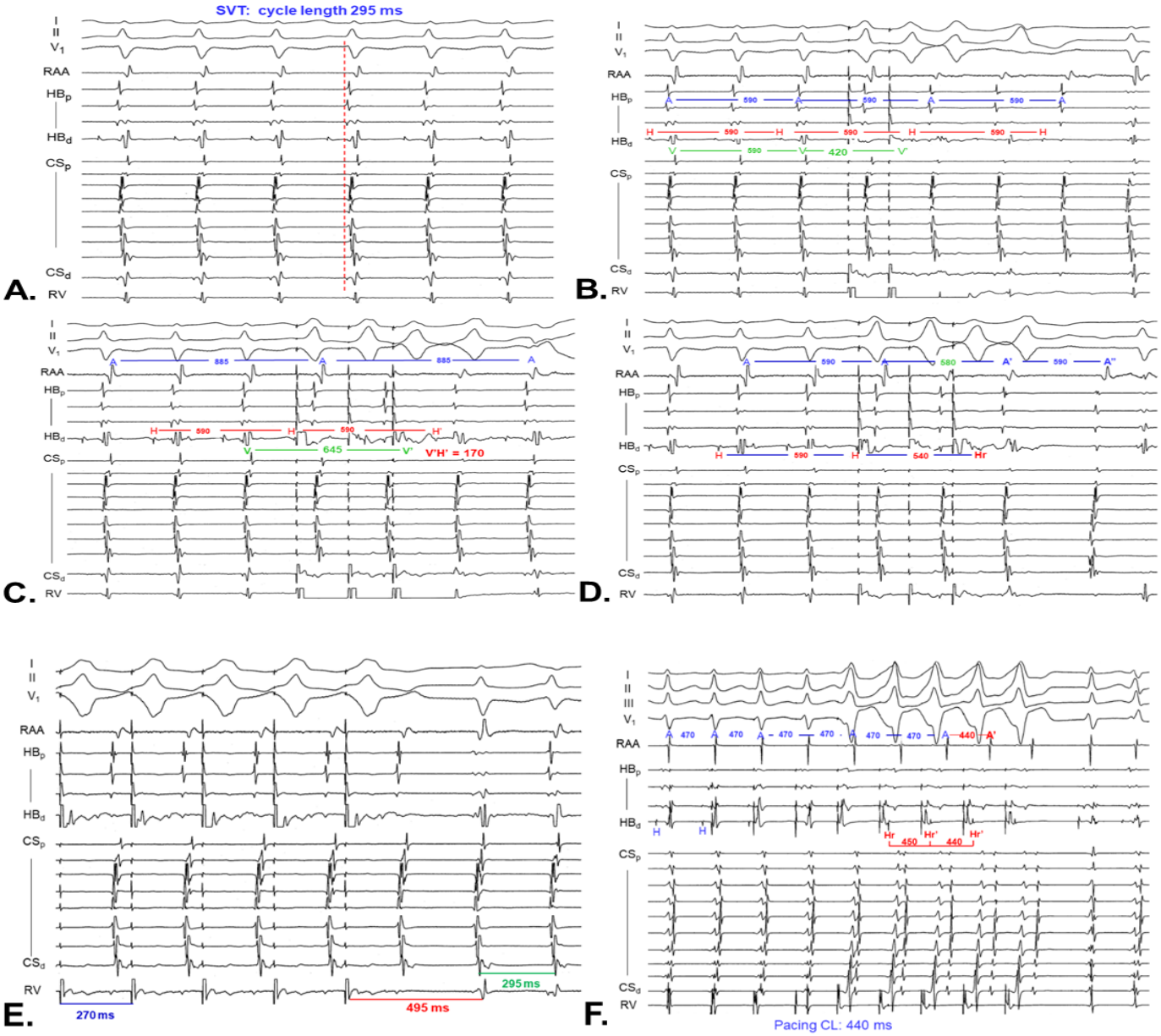
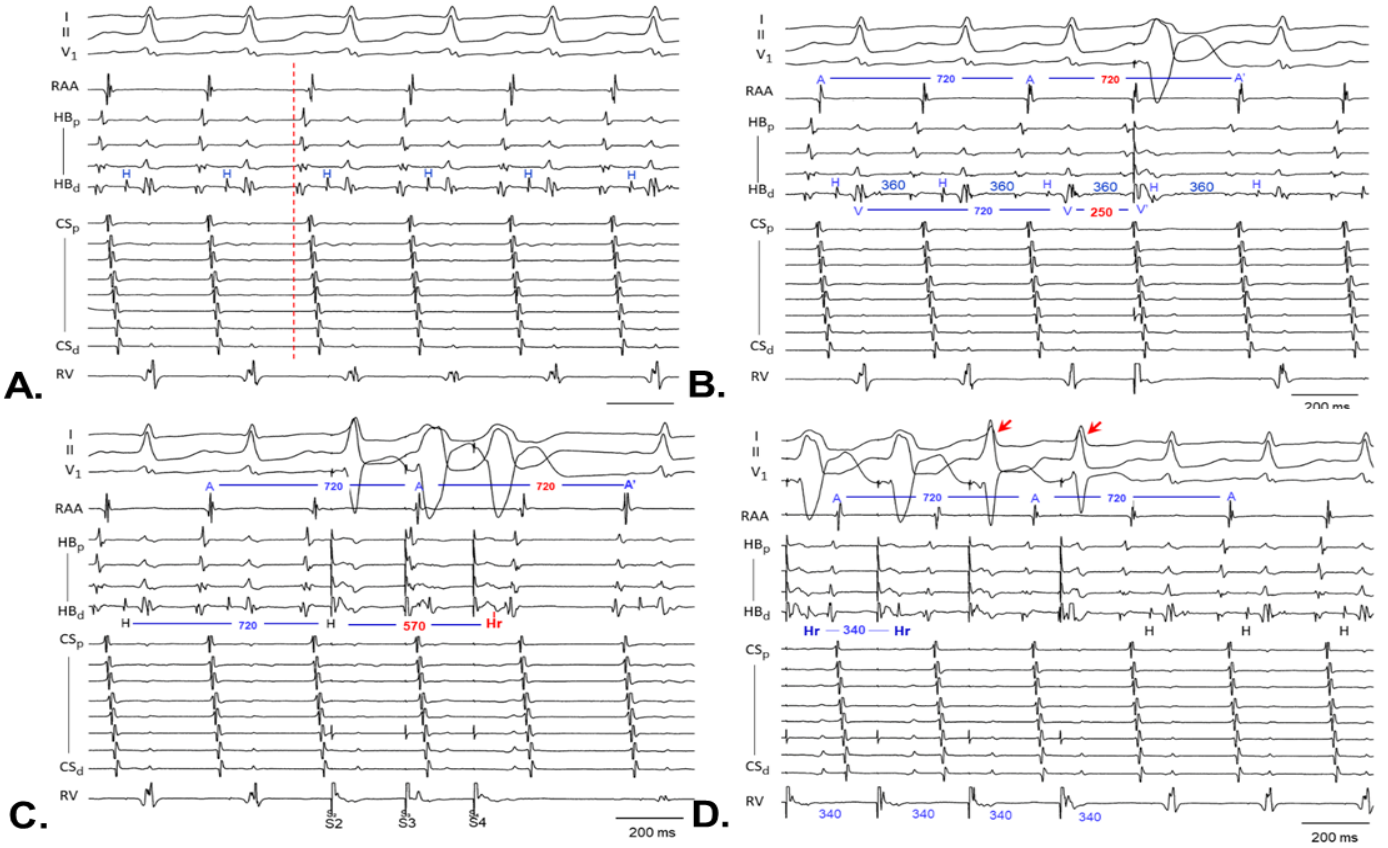


Figure 4.14. Long RV-to-His conduction time in a slow/fast AVNRT. A. NCT (CL of 295 ms) was induce. The site of earliest activation was in the HB region (vertical red line). **B.** Two VES induced transient VA dissociation. The local ventricular activation at the HBd site was advanced by 170 ms (V-V'=420 ms) without affecting the HB potential or NCT. **C.** Three VES induced transient VA dissociation again. The local ventricular activation at the HBd site was advanced by 240 ms (V-V'= 645 ms; 3xCL=885 ms) without affecting the HB potential or NCT. Despite delivering VES at a site very close to the HB, VES still failed to advance the retrograde HB potential. VES could not overcome the refractoriness of the intervening tissue between the pacing site and HB (V'-H'=170 ms). **D.** Eventually, VES advanced the retrograde HB potential by 50 ms (H-Hr = 540 ms, measured at 2 CL) and advanced the next atrial activation by 10 ms and reset the tachycardia, proving that this tachycardia was AVNRT. **E.** It required prolonged RV pacing (not shown here) to overcome the refractoriness of the intervening tissue to eventually entrain the tachycardia and produced a VAV

response with a long PPI (200 ms longer than tachycardia CL)**. F.** In another patient with AVNRT, the atrial activation timing was advanced by 30 ms (A-A'=440 ms = pacing CL) and tachycardia was reset as soon as the retrograde HB potential was entrained (pacing CL=440 ms).

The author is fully aware that many electrophysiologists do not have HB catheters with closely-spaced electrodes to the record retrograde HB potential. A simple alternative is to position the RV catheter in the parahisian area. High output pacing should produce narrow QRS complex indicating HB/RBB capture. If the operator is certain that RV overdrive pacing continuously captured the retrograde HB/RBB potential without perturbing the NCT, AVNRT can be excluded (**Figure 4.15**). That is, if the HB potential has been entrained, it should produce HA linking and reset AVNRT. If HA linking is still missing, it excludes AVNRT. **Figure 4.15** illustrates a tachycardia that could not be entrained by RV pacing after the retrograde HB potential has been entrained for several beats. In this example, the tachycardia was a septal atrial tachycardia, not an AVNRT. In addition, continuous ventricular capture without affecting the atrial timing also excludes AVRT by VA dissociation.

Some anteroseptal ATs are very difficult to differentiate from slow/fast AVNRT. There have been quite a few patients referred to the OU-EP group for anteroseptal AT ablation after the referring physicians had eliminated the slow pathway, only to find out that the NCT might be a septal AT. Ablation of the septal AT becomes an endeavor with a very high risk of AVN injury. To prevent this mistake, operators can position the RV catheter in the parahisian area. Operators should make great efforts to record a stable HB potential or entrain the NCT from the parahisian position. If basal anteroseptal RV pacing can entrain the retrograde HB potential or continuously captured the HB/RBB for 4-5 beats without affecting the NCT, this tachycardia must be an AT (**Figure 4.15**).



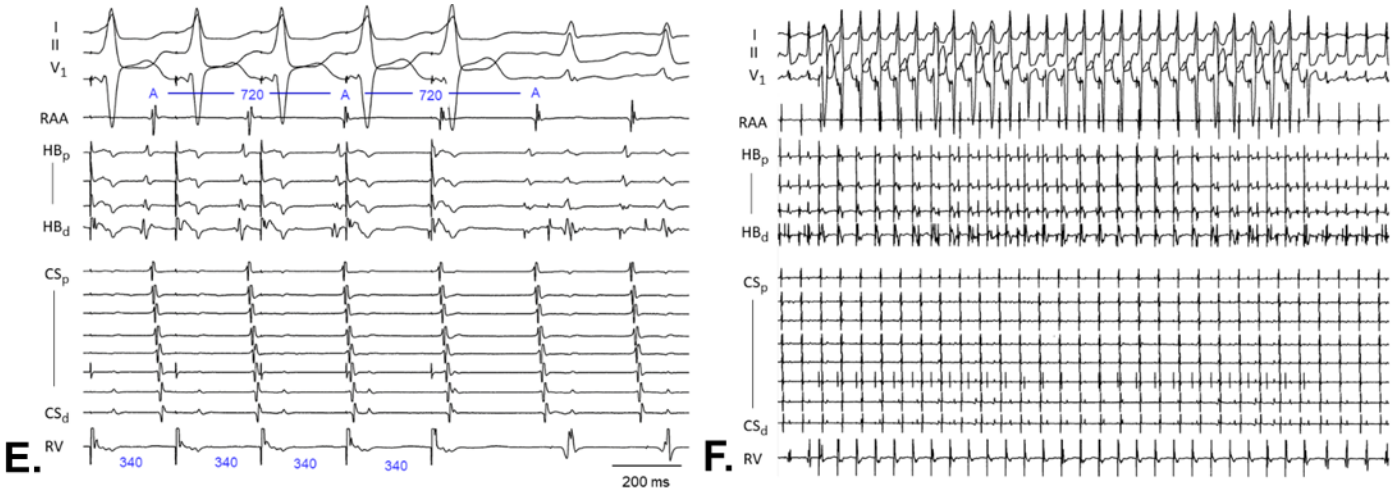


Figure 4.15. A patient with an anteroseptal AT. A. SVT was easily induced (CL=360 ms). The site of earliest atrial activation was anterior septum (vertical red line); the HA interval was fixed at 250 ms. **B.** A VES advanced the ventricular activation in the anterior septum (adjacent to the site of earliest atrial activation) by 110 ms without affecting the SVT (A-A'=720, 2xCL, unchanged), excluding AVRT using an anteroseptal or midseptal AP for retrograde activation. **C.** When triple VES were delivered to the basal anteroseptal RV, S4 advanced the retrograde HB potential (Hr) by 150 ms without perturbing the SVT (A-A'=720 ms, unchanged), excluding the diagnosis of AVNRT. **D.** Overdrive pacing (CL 340 ms) was delivered to the basal anteroseptal RV. Note that the retrograde HB potentials were entrained in the first two paced beats (Hr-Hr=340 ms) and the 3rd and 4th paced beats captured the HB potential (red arrows). These observations indicated that entraining the retrograde HB potential could not perturb the SVT. **E.** Again, basal anteroseptal RV pacing captured the HB/RBB 5 beats in a row without affecting the SVT. **F.** A long run of RV pacing (9.2 seconds) failed to perturb the SVT. This focal atrial tachycardia was successfully ablated at the anterior-superior edge of the fossa ovalis.

Mistake 5: If the site of earliest atrial activation of an SVT is in the posteroseptal area, I just ablate the site of earliest atrial activation regardless it is AVNRT, AT or AVRT.

The author always teaches the fellows in this way:

"If you misplaced your key but you know it is in your house, you need to search the entire house."

"If you know your key is in your bed room, you only have to search the bed room."

"If you are sure your key is in the dresser of your bed room, you only need to search the dresser."

If the diagnosis is orthodromic AVRT using a posteroseptal AP for retrograde conduction, the target can be anywhere from the posteroseptal mitral annulus, CS, a CS tributary (e.g. middle cardiac vein), CS ostium to posteroseptal tricuspid annulus. However, if the diagnosis is fast/slow AVNRT, the target is usually in the inferior triangle of Koch or CS ostium. Knowing the correct diagnosis can narrow the scope of search and avoid complications such as coronary artery or AVN injury. **Figure 4.16** illustrates three different long RP tachycardias, all of which showed earliest activation in the proximal CS. SVTs were caused by different mechanisms and ablated from different sites.

Specific Issues

Junctional tachycardia vs. slow/fast AVNRT

True junctional tachycardia (JT) is rare but junctional automaticity frequently occurs in young patients during isoproterenol infusion or after slow pathway ablation. Because of JT's rarity, there has been a paucity of studies addressing differential diagnosis between slow/fast AVNRT and JT. The most cited paper authored by Drs. Padanilam and Prystowsky actually used junctional rhythm as a surrogate for JT (JACC 2008;52[21)1711-17). In the author's opinion, the most effective pacing maneuver to differentiate between AVNRT and JT is to deliver *late* atrial extra-stimuli (AES) at the CS ostium in close proximity to the slow

pathway (**Figure 4.17**). The atrial activation timing recorded in the HB area should not be advanced by the AES to ensure that the retrograde fast pathway is not affected by the AES. If this *late* AES does not affect the timing of the HB and the timing of the atrial potential in the HB region but advances or delays the next HB potential and reset the tachycardia, it not only excludes JT but also indicates that the AES must have engaged the antegrade slow pathway to perturb AVNRT.

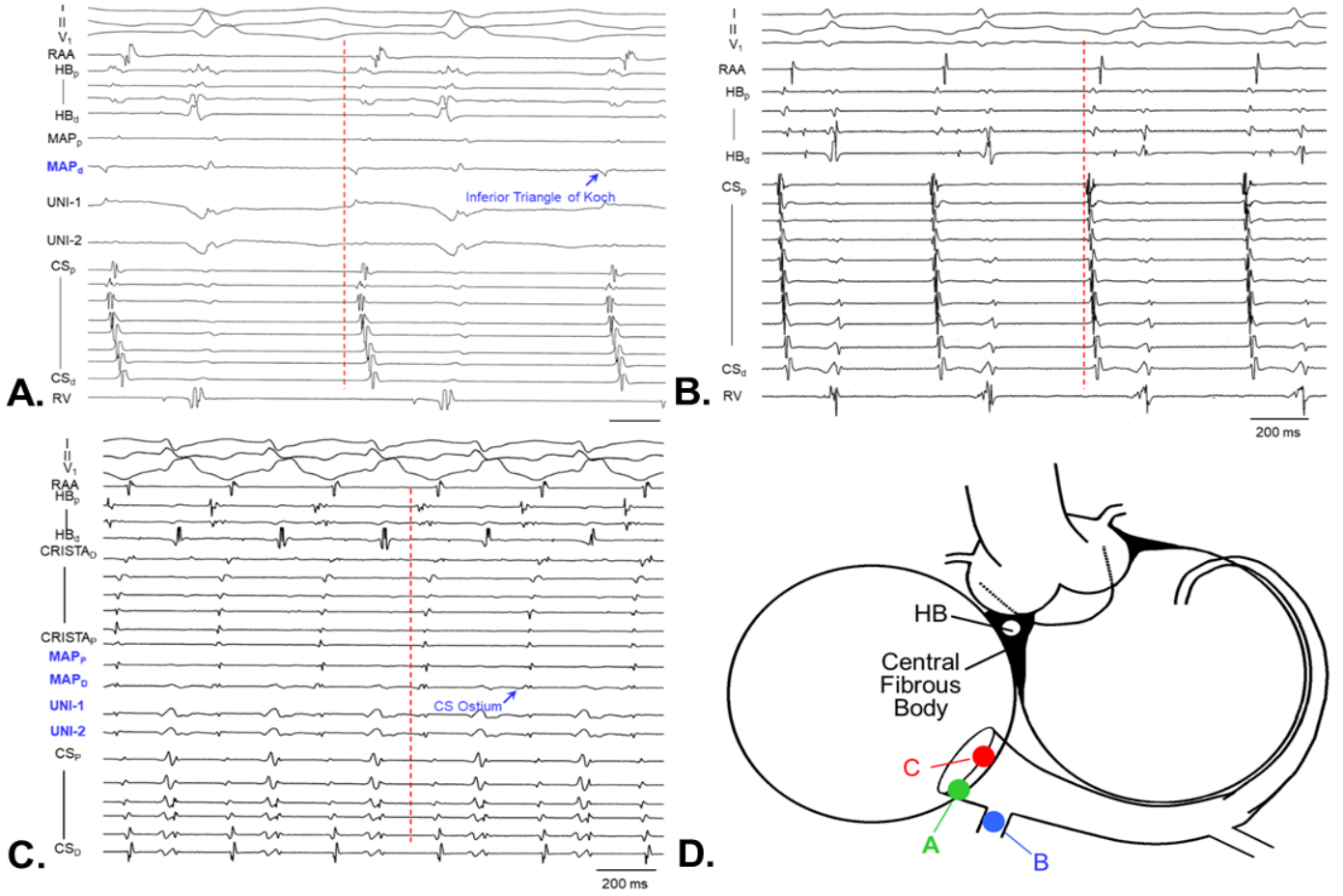


Figure 4.16. Three different long RP tachycardia with earliest atrial activation in the posteroseptal area. A. fast/slow AVNRT. **B.** AVRT using an epicardial posteroseptal AP for retrograde conduction. **C.** Focal AT. **D.** Site of successful ablation of each SVT.

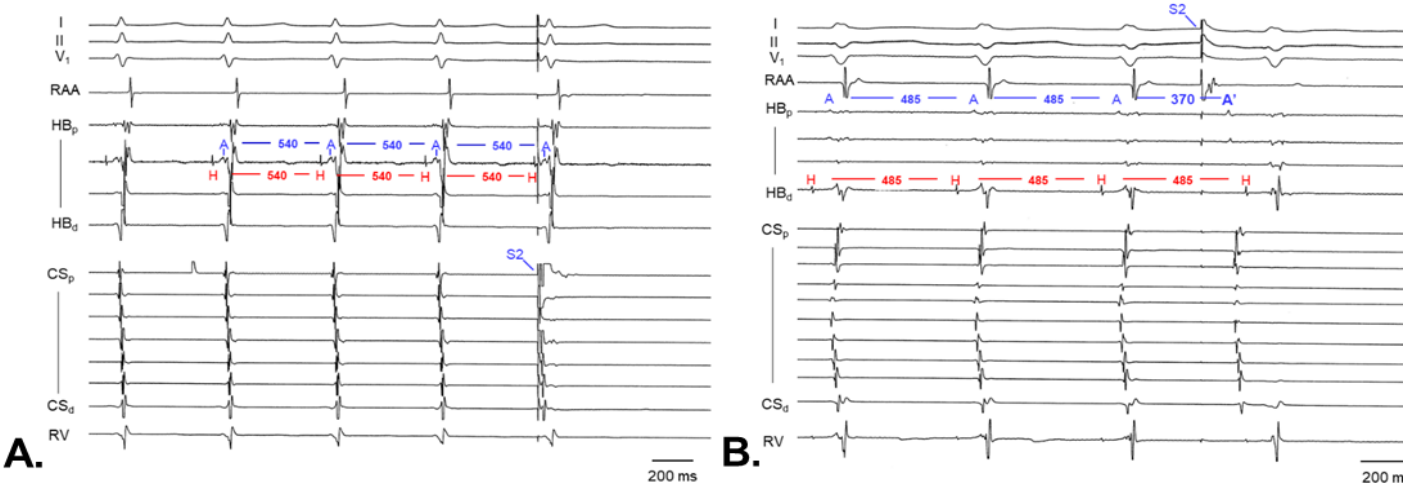


Figure 4.17. Deliver AES to exclude junctional tachycardia. A. In a patient with slow/fast AVNRT, an AES (S2) delivered to the CS ostium, in close proximity to the slow pathway, terminated the tachycardia. Note that the atrial timing recorded in the HB area was not affected by the AES, indicating that the retrograde fast pathway was not perturbed by the AES. The H-H interval remained unperturbed but tachycardia was terminated, excluding JT. It also indicates that the AES terminated the tachycardia by affecting the

antegrade slow pathway. **B**. In another slow/fast AVNRT patient, a very early AES was delivered to the RA appendage, which advanced the atrial timing of the HB region by 115 ms (A-A'=370 ms) and terminated the tachycardia. Note that the H-H interval remained unperturbed. This AES might have affected either the retrograde fast pathway or the intervening tissue between the slow pathway and fast pathway to terminate this tachycardia.

Only nonsustained NCT can be induced

When only non-sustained NCT is induced, proving the tachycardia mechanism can be very challenging. In this situation, the author delivers parahisian pacing *immediately* after termination of the non-sustained NCT when all the catheters are in the same places, followed by comparing the activation sequence between NCT and parahisian pacing. If parahisian pacing reveals retrograde conduction over an AP and the NCT activation sequence is identical to that, the mechanism of the tachycardia is most likely AVRT (**Figure 4.18**). If the activation sequence of the NCT is identical to that of the retrograde AVN conduction revealed by parahisian pacing, this nonsustained NCT is most likely AVNRT**.**

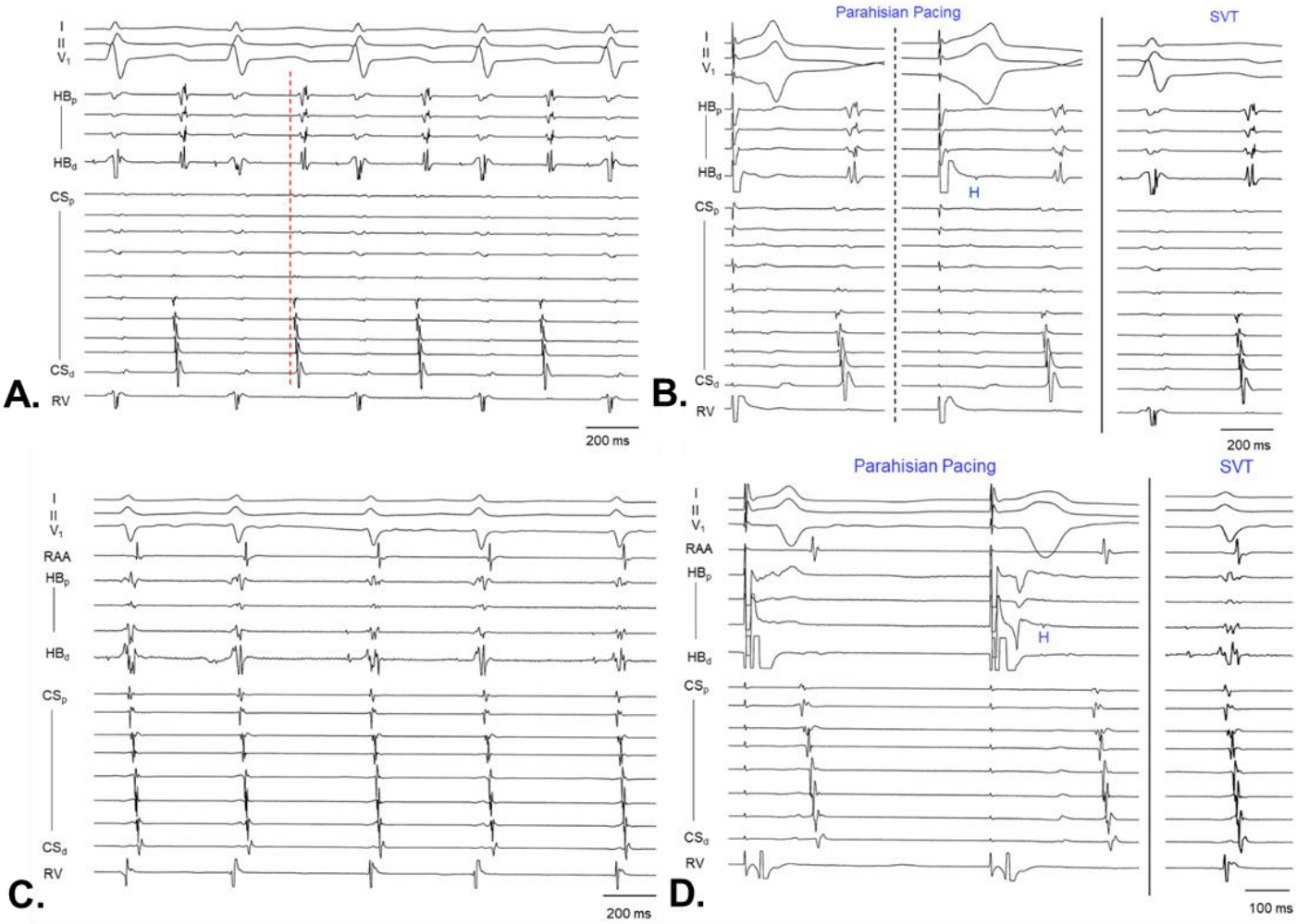


Figure 4.18. Parahisian pacing to diagnose non-sustained SVT. A. A young man with two prior ablations for low septal AT. Non-sustained long RP tachycardia was repeatedly induced but did not last long enough for pacing maneuvers to verify the mechanism of SVT. The site of earliest activation was at the CS ostium. Note diffuse low voltage EGM in the proximal CS due to extensive prior ablations. **B.** Parahisian pacing performed immediately after termination of non-sustained SVT showed that the atrial activation sequence in SVT was identical to retrograde AP conduction revealed by parahisian pacing. This patient had a posteroseptal AP was successfully ablated at the orifice of the middle cardiac vein**. C.** A 64 y/o female with a non-sustained SVT that had variable AV interval, making it difficult to prove the mechanism underlying this SVT. **D**. Immediately after SVT terminated, parahisian pacing was performed (**left panel**), which demonstrated retrograde AVN conduction. The atrial activation was identical to that of the SVT (**right panel**), strongly suggesting that this SVT was AVNRT. SVT was eliminated by slow pathway ablation.

NCT with VA conduction block

In the author's opinion, the most difficult differential diagnosis to make is NCT with VA conduction block. The differential diagnosis includes a number of rare arrhythmias: AVNRT with a long upper common pathway, junctional tachycardia, orthodromic AVRT using a nodo-ventricular/nodo-fascicular AP for retrograde conduction and high septal VT near the HB area. In high septal VT near the HB, the HB potential is either dissociated from the tachycardia or the HV interval during tachycardia is shorter than that in sinus rhythm because the HB potential in high septal VT is a retrograde HB potential. The QRS morphology in VT is very similar but not identical to that in sinus rhythm. It is important to record a HB potential, not a RBB potential. The former is accompanied by an easily identifiable atrial EGM; the latter is not. If the "HV" interval in sinus rhythm is misrepresented by a RBB potential, the "HV" interval in sinus rhythm will be artificially shorter than the true HV interval. Therefore, a shorter HV interval during high septal VT can be missed because the baseline "HV" interval (correctly, RBB-V interval) measured in sinus rhythm is erroneously short.

Differential diagnosis between slow/fast AVNRT and JT has already been discussed. Orthodromic nodo-ventricular or nodo-fascicular AVRT is very rare. The leading expert in this arrhythmia, Dr. Melvin Scheinman, believes that a lot of nodo-ventricular or nodo-fascicular AVRT with 1:1 VA conduction were misdiagnosed as AVNRT. Because the "nodal" end of the AP is often in the AVN slow pathway, ablation of the slow pathway eliminated most nodo-ventricular/nodo-fascicular AVRT as well (**Figure 4.19**). In a nodo-ventricular/nodo-fascicular AVRT with variable VA conduction, VES introduced during HB refractory should be able to perturb the tachycardia by advancing or delaying the next HB potential and reset the AVRT.

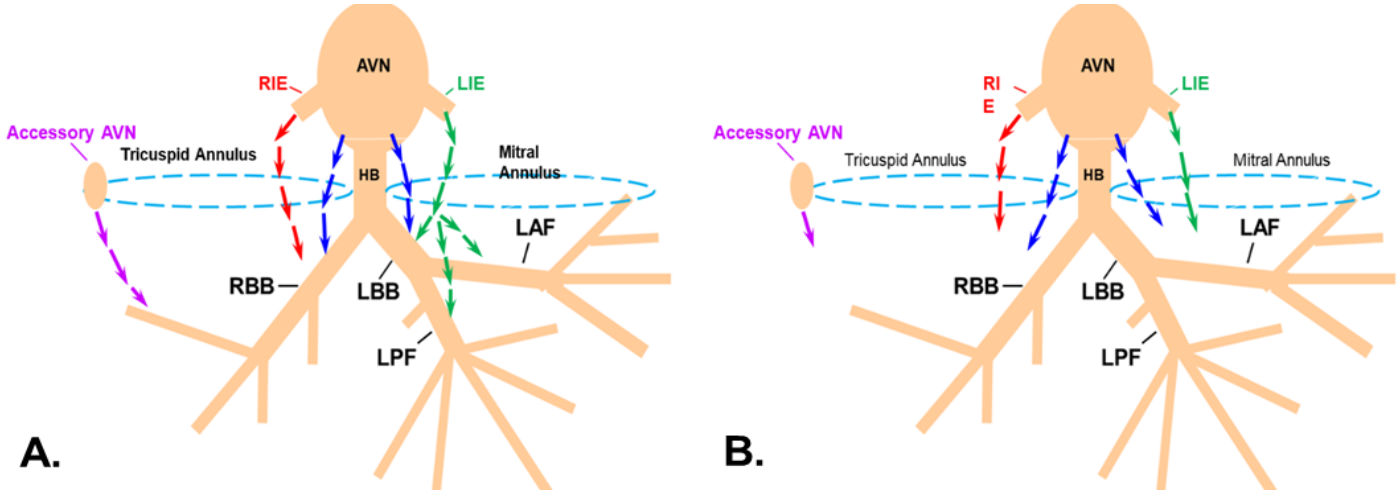


Figure 4.19. Atrio-fascicular AP, nodo-fascicular AP and nodo-ventricular AP. A. An atrio-fascicular AP (Mahaim) connects the accessory AVN to peripheral RBB (purple arrows). One can view a nodo-fascicular AP as a septal atrio-fascicular AP. The proximal end of a nodo-fascicular AP can be located in the right inferior extension (RIE, red arrows) or left inferior extension (LIE, green arrow) of the AVN or in the compact AVN itself (blue arrows). The most common type of nodo-fascicular APs connects the RIE to the proximal RBB. There have been reports showing that nodo-fascicular APs can connect the LIE to LBB, left anterior fascicle (LAF) or left posterior fascicle (LPF). **B.** Similar to a nodo-fascicular AP, the proximal end of a nodo-ventricular AP can be located in the compact AVN, RIE or LIE. The distal end does not connect to the His-Purkinje system but to ventricular myocardium. Therefore, the VH interval is longer.

Wide-Complex Tachycardia

The most important tool for differential diagnosis of wide-complex tachycardia (WCT) is a 12-lead ECG. Differential diagnoses of WCT include pre-excited AVRT, SVT (AT or AVNRT) with a bystander AP, SVT with aberrant conduction and VT. Although all the aforementioned tachycardias exhibit either LBBB or RBBB morphology, it is critical to differentiate typical BBB from atypical BBB. A WCT with typical LBBB or RBBB indicates that ventricles are activated by the His-Purkinje system. The HV interval during WCT often provides the most important clue to the diagnosis and underscores the importance of recording a stable HB potential in sinus rhythm before attempting to induce tachycardia (see discussion below).

WCT with typical LBBB morphology

1. SVT (AT, AVNRT or orthodromic AVRT) with aberrant conduction. The HV interval in tachycardia is ≥ the HV interval in sinus rhythm.

2. Preexcited AVRT using an atrio-fascicular AP (Mahaim) for antegrade conduction. The HV interval is typically -15 to -25 ms because the HB potential is activated in the retrograde direction. However, the HV interval value can be more negative or the HB potential can be obscured by the local ventricular potential if RBBB is present (**Figure 4.20A**).

3. Bundle branch reentrant VT. The baseline HV interval is typically long (e.g. >70 ms) due to extensive His-Purkinje disease. The HV interval in tachycardia is typically ≥ the HV interval in sinus rhythm (**Figure 4.20B**) but on rare occasions, the HV in VT can be shorter than the HV interval in sinus rhythm. Because the HB potential is retrograde in bundle branch reentrant VT, the HV interval is determined by the relative timing between ventricular activation and HB activation.

4. Preexcited AVRT using a nodo-fascicular AP for antegrade conduction. Similar to atrio-fascicular AVRT, the HV interval is typically a negative value because the HB potential is activated in the retrograde direction. To give rise to a typical LBBB morphology, the AP has to connect to the RBB. That is, the RV is activated first by the RBB, giving rise to the morphology of typical LBBB.

5. Focal VT originating from the RBB. The HV interval is usually fixed in tachycardia and is shorter than the HV interval in sinus rhythm.

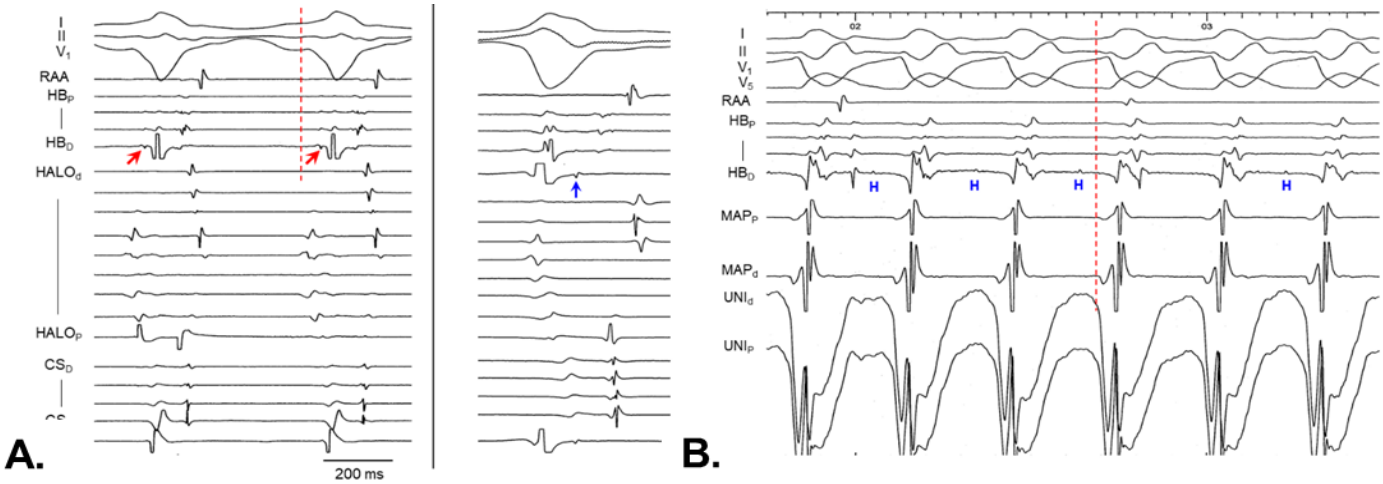


Figure 4.20. HV interval in WCT with typical LBBB morphology. A. Preexcited AVRT caused by an atrio-fascicular AP (Mahaim). **Left panel:** HV interval = -25 ms. Red arrows: retrograde HB potential. Vertical dotted line: onset of the QRS complex. **Right panel:** A late retrograde HB potential (blue arrow) was a result of RBBB that prolonged the conduction to the HB. **B.** Bundle branch reentrant VT. The HV interval was 75 ms, slightly longer than that during sinus rhythm (not shown).

WCT with typical RBBB morphology:

1. SVT (AT, AVNRT or orthodromic AVRT) with aberrant conduction. The HV interval in tachycardia is ≥ the HV interval in sinus rhythm.

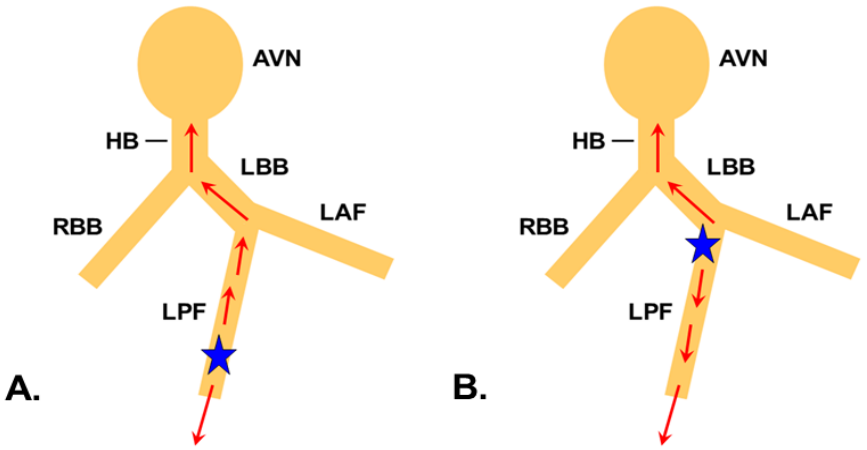
2. Left anterior or posterior fascicular VT. The HV interval in tachycardia is shorter than the HV interval in sinus rhythm because the HB potential is activated in the retrograde direction.

3. Preexcited AVRT using a nodo-fascicular AP for antegrade conduction. To give rise to a typical RBBB morphology, the AP has to connect to the LBB.

4. Focal VT originating from the LBB. The HV interval is usually fixed in tachycardia and is < the HV interval in sinus rhythm.

Using HV interval to predict the origin of a His-Purkinje related tachycardia

For a His-Purkinje related tachycardia, the HV interval provides an important clue to the location of the site of origin of the WCT. The HV interval in a WCT is determined by two factors: (1) how fast the source of arrhythmia conducts to the ventricle to create a QRS complex and (2) how fast the source of arrhythmia conducts retrogradely to the HB region. The wave fronts propagation of (1) and (2) are often in opposite directions. If the source of arrhythmia is in the distal Purkinje system, it quickly produces a QRS complex. However, it takes a longer period of time to conduct retrogradely to the HB region, leading to a relatively long VH interval (a more negative HV interval, e.g. -30 ms; **Figure 4.21**). If the arrhythmia originates in the proximal Purkinje system, it takes a longer period of time to produces a QRS complex. However, it quickly conducts retrogradely to the HB region, leading to a relatively short VH interval (a less negative HV interval, e.g. -5 ms). When the source of arrhythmia is in the very proximal His-Purkinje system (e.g. distal HB region), the HV interval is typically a positive value but shorter than the HV interval in sinus rhythm. Notably, the QRS complex is usually very similar to that in sinus rhythm in this situation and is often erroneously diagnosed and ablated as SVT. Simply by measuring the HV interval during tachycardia, one can gain deep insight into the site of origin of a His-Purkinje related tachycardia.



4.21. Using HV interval to predict the origin of a His-Purkinje related tachycardia. A. If the origin is located in distal left posterior fascicle (LPF), it exits quickly to ventricular myocardium to produce a QRS complex. In contrast, it has to travel a relatively long distance to the HB area to generate a retrograde HB potential. The HV interval is therefore a negative value (typically -15 to -30 ms). **B.** If the origin of VT is located in proximal LPF, it has to travel a longer distance to exit but a shorter distance to the HB area, producing a less negative HB interval. If the origin of VT is located in the LBB or HB area, the HV interval will be a positive value and the QRS complex will be narrow, resembling the QRS complex in sinus rhythm. LAF: left anterior fascicle; LPF: left posterior fascicle

Typical vs. Atypical Bundle Branch Block

To differentiate typical BBB from atypical BBB, one must understand how the ventricles are activated by the LBB and RBB. Interventricular septum is the very first part of the ventricle to be activated. While most cardiologists were taught that LBB bifurcates into the left anterior and posterior fascicle, the Purkinje system indeed is a complex network. There are many branches on the endocardial surface of the left ventricular septum (e.g. left septal fascicle), activation of which leads to ventricular septal activation (**Figure 4.22A**). Branches from the proximal RBB also contribute to ventricular septal activation. A smaller mass of the ventricular septum is activated by proximal branches of the RBB. The net vector of ventricular septal activation therefore points straight at the V_1 or V_2 lead, resulting in a small, sharp r wave in V_1 and/or V_2. For ECG leads facing lateral LV (e.g. lead I, V_6), the vector of ventricular septal activation may produce a small q wave, creating the “Q” component of the QRS complex. Because the conduction velocity of normal His-Purkinje system is 5-10 times faster than that of myocardium, ventricular septal activation is finished in 20-30 ms. The duration of this small r wave on V_1 or V_2 is almost always shorter than 40 ms (a small grid on ECG) in people without significant His-Purkinje diseases. In patients with prior septal infarct, this small, sharp r wave in V_1 and/or V_2 may disappear or becomes a q wave.

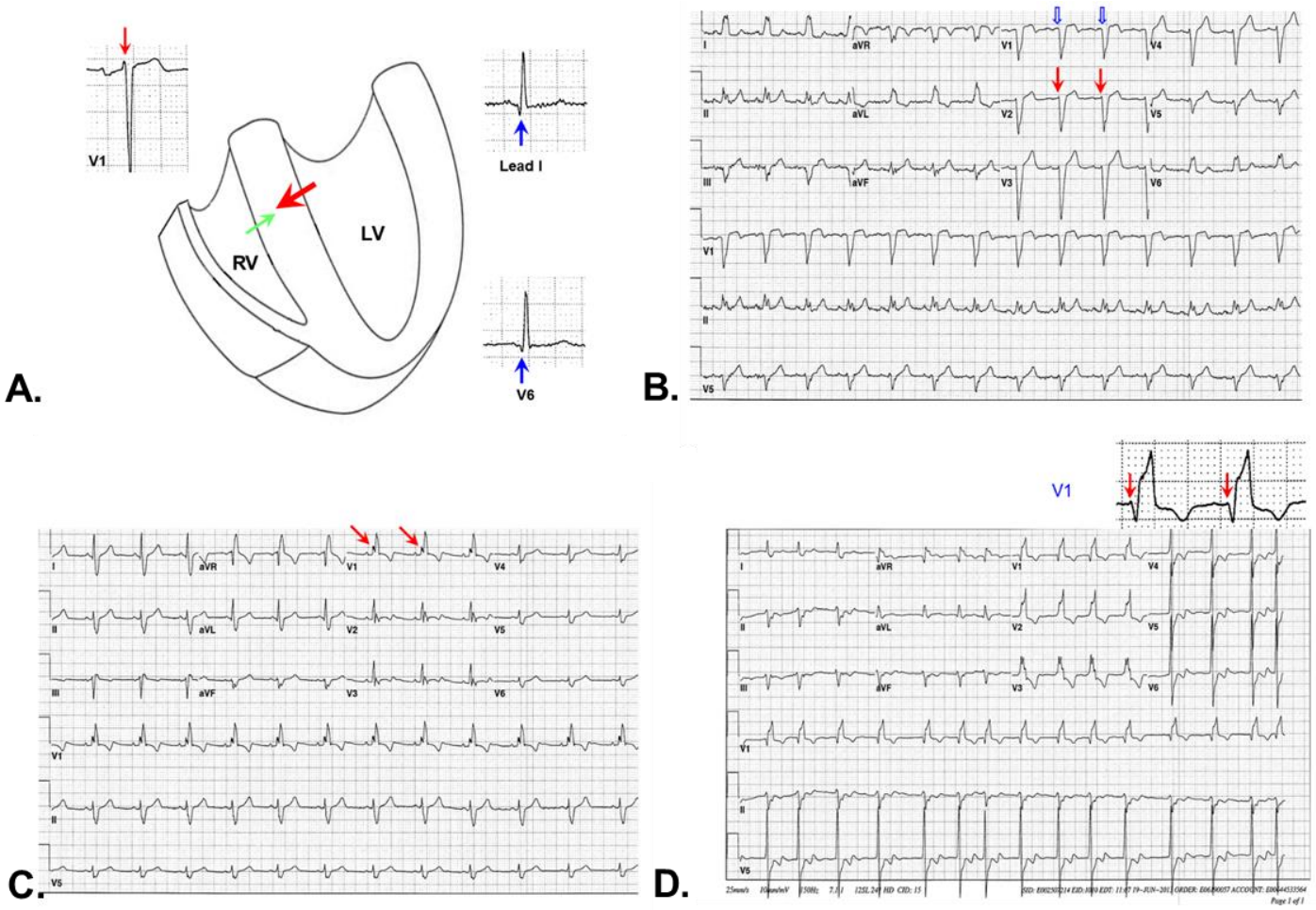


Figure 4.22. Ventricular septal activation in sinus rhythm, LBBB and RBBB. A. Ventricular septum is the first part of the ventricle that is activated by the Purkinje network of the left ventricle (red arrow). Approximately 5 ms later, the septal branch of the RBB activates the septum as well (green arrow). Ventricular septum is activated within 20-30 ms. The net vector is therefore left to right, posterior to anterior, creating a small, sharp r wave in V1 and/or V2 (thin red arrow) as well as a q wave (blue arrows) in lateral leads such as lead I and V6. **B. Typical LBBB.** Although V1 shows a QS pattern (without the small r wave, blue empty arrow), a small, sharp r wave (red arrow) was visible in lead V2. **C. Typical RBBB**. A small, sharp r wave (red arrow) is visible in lead V1. **D. Typical RBBB.** The QRS complex in lead V1 appears to be a qrR' morphology. More careful examination identifies a small, sharp r wave (red arrow, inset).

In the presence of RBBB, ventricular septum is activated entirely by the septal branches of the LBB, the r wave on lead V_1 and/or V2 remains sharp (<40 ms) but the amplitude of the r wave may be moderately larger because the opposing factor provided by the proximal branches of the RBB is now missing. If the beginning of the QRS complex in lead V_1 is a q wave or a large R wave, it strongly suggests that the beginning of the ventricular activation does not result from His-Purkinje conduction (**Figure 4.23**), favoring the diagnosis of VT or preexcited tachycardia. There have been many publications on differential diagnosis of WCT. For example, if lead V_1 shows an Rsr morphology (left rabbit ear is taller than the right rabbit ear), it indicates VT. Essentially, this diagnostic criterion says "if it does not look like a typical RBBB, it is most likely a VT".

The QRS morphology of V_1 and/or V_2 in LBBB is more complex. If the blocked (or delayed) site is proximal to the septal branches of the LBB that activates the ventricular septum, V_1 and/or V_2 will show a QS pattern. On the other hand, if the blocked (or delayed) site is more distal, V_1 and/or V_2 will show an rS pattern because the ventricular septum is activated normally. The r wave remains sharp as well (**Figure 4.22**).

When the author was a fellow, one of the master educators, Prof. George Klein, made the following comment:

"Guys, I know you try to remember the Brugada criteria, Wellen's criteria and other criteria to distinguish VT from SVT with aberrancy. Look at the morphology of bundle branch block first. If it not typical, it is not SVT with aberrancy".

The author has followed Prof. Klein's advice for longer than 20 years and always advises cardiology or EP fellows to print out every ECG showing sinus rhythm with LBBB, RBBB or hemi-block to build a database of how a typical BBB or hemi-block should look like. In the author's opinion, V_1 and V_2 leads should be viewed as a single lead. If the heart is rotated slightly to the right, V_1 lead will record what a V_2 lead should have recorded. On the contrary, the sharp, small r wave sometime is only seen in lead V_2 if the heart is rotated slightly to the left. As long as lead V_1 or V_2 shows a sharp, small r wave, the author interprets it as ventricular depolarization initiated by His-Purkinje activation.

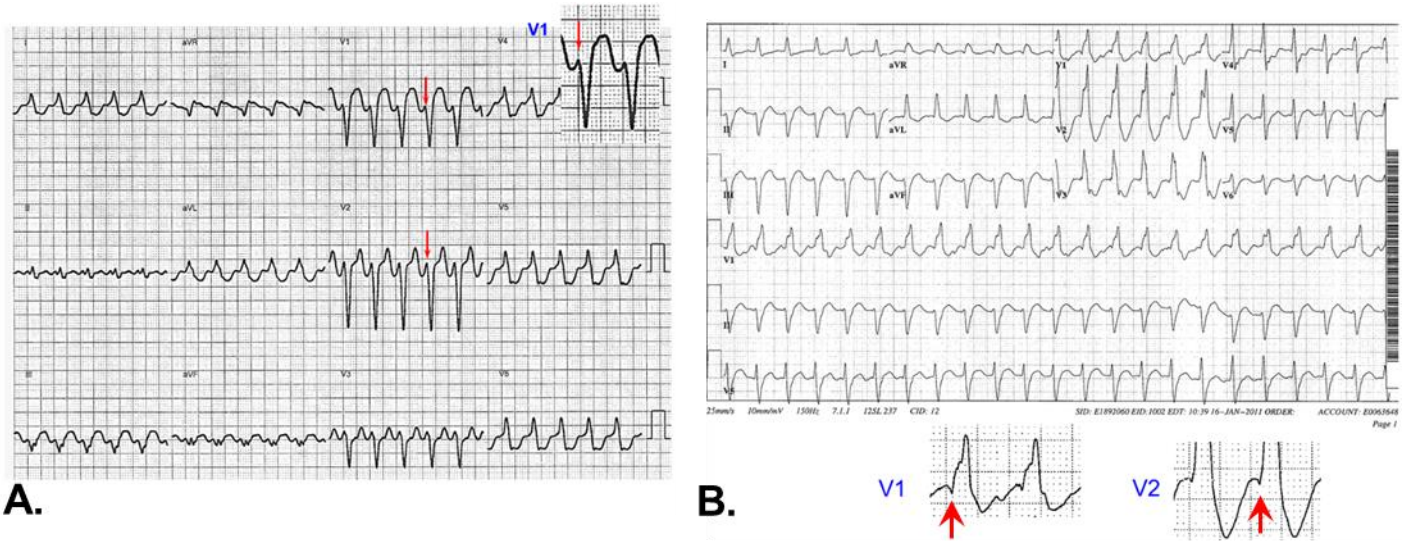


Figure 4.23. Atypical BBB. A. A small but wide r wave in lead V1 and V2 is visible (red arrow). This WCT was an antidromic AVRT**. B.** A small q wave in lea V1 and V2 is visible (red arrow, inset). This WCT was a papillary muscle VT.

The very first ECG lead the author examines to diagnose any arrhythmia is lead V_1 for the following reasons. First, P waves are usually visible on lead V_1. The morphology of the P waves, the RP interval and the presence of AV dissociation provide important clues to the mechanism and origin of arrhythmia. Second, the presence of absence of a small, sharp r wave reveals if the first 40 ms of ventricular activation is activated by the His-Purkinje system. For example, in a WCT with RBBB morphology and superior-leftward axis, the differential diagnosis includes SVT with aberrant conduction, left posterior fascicular VT as well as VT originates from the posterior-medial papillary muscle. Left posterior fascicular VT causes early activation of the septal LBB branches, often giving rise to a sharp r wave in lead V_1 and/or V_2. In contrast, VT originating from the posterior-medial papillary muscle does not quickly activate the septal branches of the LBB. The vector of the first 40 ms of ventricular activation often points away from lead V_1 and/or V_2, giving rise to a qR wave ion lead V_1 and/or V_2 (**Figure 4.23B**). Therefore, if one sees a small, sharp r wave in V1 in this scenario, it is almost always a fascicular VT. A qR pattern in V1 favors papillary muscle VT.

It is the author's opinion that the presence of a small, sharp r wave in V1 or V2 has a good positive predictive value for ventricular activation by the His-Purkinje system. The absence of it does not exclude the possibility of ventricular activation by the His-Purkinje system because when the heart rate is fast, conduction block can occur anywhere in the His-Purkinje system. A fascicular VT or an SVT with aberrant conduction may lose the small, sharp r wave in lead V1 and exhibits an atypical RBBB pattern (e.g. qR in V_1 lead), similar to the ECG morphology of a papillary muscle VT. In this scenario, a fixed HV interval (typically 0 to -30 ms) during WCT still indicates fascicular VT, not papillary muscle VT because papillary muscle VT often causes dissociated HB potential or a very late HB potential (e.g. -60 ms) due to late engagement into the peripheral Purkinje system. That is to say, a small, sharp r wave in V1 or V2 lead has a very good positive predictive value that ventricular activation is mediated by the His-Purkinje system. When the small, sharp r wave is

absent, the possibility of His-Purkinje activation is lower. A fixed and short VH interval strongly suggests that it is a His-Purkinje related tachycardia.

Dissociation vs. 1:1 association between the HB potential and WCT

At first, operators need to differentiate “HB potential obscured by local ventricular activation” from “HB potential dissociated from tachycardia”. In the former, the HB potential is always not visible. If a HB potential is visible intermittently and the HV interval is not fixed, this observation indicates that the HB potential is dissociated from the tachycardia. If the HB potential and WCT show 1:1 relationship with a fixed HV interval, differential diagnoses include antidromic AVRT, SVT with aberrant conduction and VT related to the His-Purkinje system. In antidromic AVRT, the retrograde limb of the reentrant circuit is AVN. However, if the HB potential overlaps with the ventricular activation of the anteroseptal area, the HB potential may not be visible. For VT related to the His-Purkinje system, the HB potential is activated in the retrograde direction with a fixed HV (or HV) interval. On rare occasions, the proximal His-Purkinje system is significantly diseased, leading to retrograde conduction block to the HB area and dissociation between the HB potential and QRS complex.

If the HB potential and WCT are dissociated, this observation is “almost” diagnostic of VT. However, in an AP-to-AP tachycardia (the antegrade limb and retrograde limb of the tachycardia are formed by two separate APs), the HB can be dissociated from the WCT because HB activation is not required to maintain the tachycardia.

Differential diagnosis of WCT in EP laboratory

SVT with aberrant conduction can be identified in the EP laboratory by (1) a fixed HV interval that is *not* shorter than the HV interval in sinus rhythm *and* (2) typical BBB pattern unless the rate of tachycardia is very fast that produces an atypical BBB pattern. Dr. Jackman’s approach to diagnose a WCT in the EP laboratory can be summarized as follows (**Figure 4.24**).

1. Deliver single AES at a site as close to the site of earliest *ventricular* activation as possible. If ECGs suggest a left-sided AP or a left ventricular tachycardia, AES are delivered to the CS electrodes adjacent to the site of earliest ventricular activation to ensure that AES can quickly engage the *atrial* end of the AP if present (**Figure 4.24, 4.25**).

2. Evaluate how the AES(s) conducts to the ventricle to reset the tachycardia. There are only two routes that an AES can conduct to the ventricle: AVN and AP. For an AES to advance the next ventricular activation timing through the AVN, the anteroseptal atrial activation timing has to be advanced first. If the AES can advance the next ventricular activation timing without affecting the timing of the anteroseptal atrial activation, it proves the presence of an AP that is capable of conducting in the antegrade direction. However, to prove that this AP participates in the WCT, not a bystander AP during AVNRT or AT, the WCT must be reset by such AES.

3. If WCT is proven to be caused by pre-excited AVRT, Dr. Jackman will then deliver VES at a site adjacent to the site of earliest *atrial* activation to evaluate if VES conduct to the atrium to reset the tachycardia through AVN or another AP (**Figure 4/24F; 4-25D**).

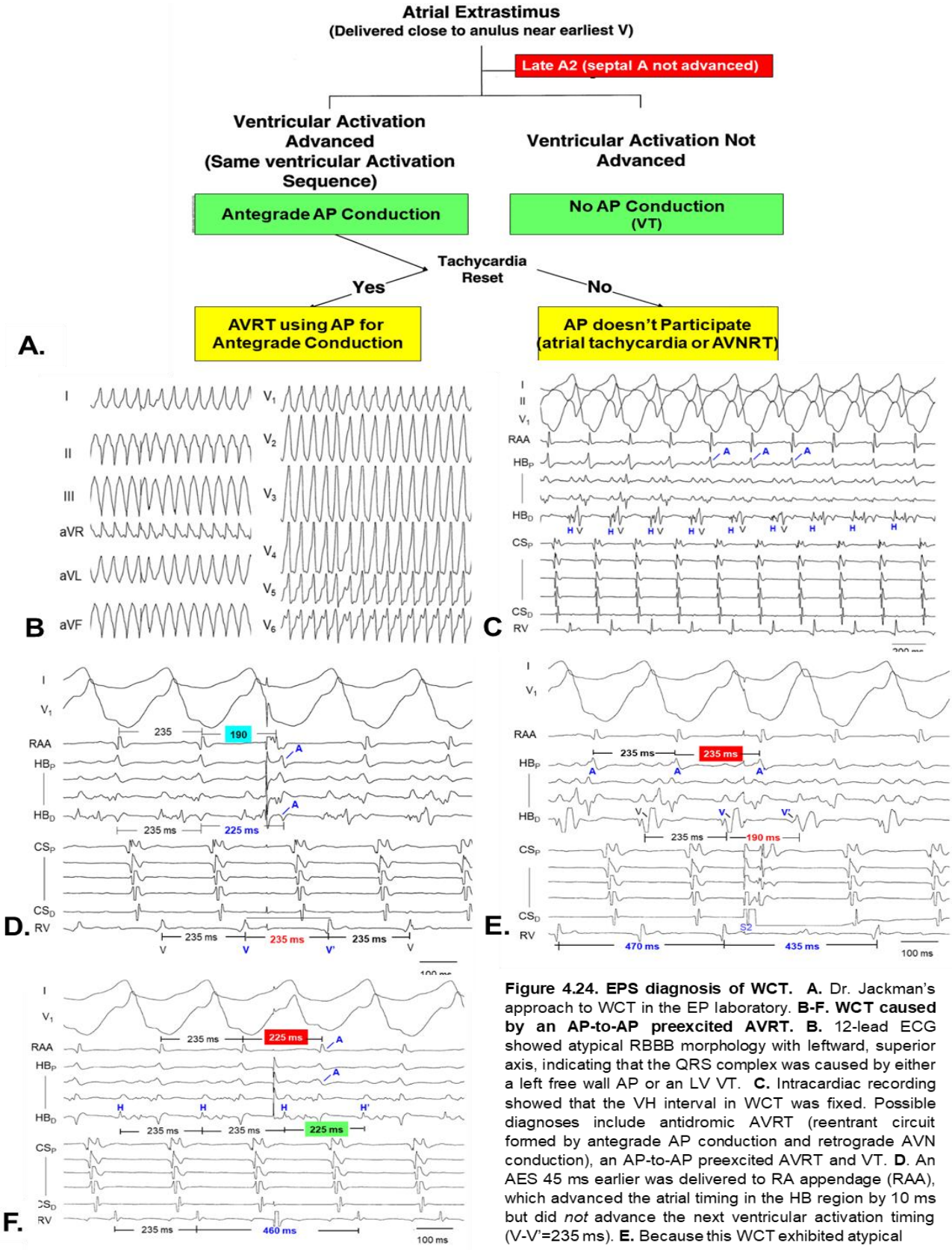


Figure 4.24. EPS diagnosis of WCT. A. Dr. Jackman's approach to WCT in the EP laboratory. **B-F. WCT caused by an AP-to-AP preexcited AVRT. B.** 12-lead ECG showed atypical RBBB morphology with leftward, superior axis, indicating that the QRS complex was caused by either a left free wall AP or an LV VT. **C.** Intracardiac recording showed that the VH interval in WCT was fixed. Possible diagnoses include antidromic AVRT (reentrant circuit formed by antegrade AP conduction and retrograde AVN conduction), an AP-to-AP preexcited AVRT and VT. **D.** An AES 45 ms earlier was delivered to RA appendage (RAA), which advanced the atrial timing in the HB region by 10 ms but did *not* advance the next ventricular activation timing (V-V'=235 ms). **E.** Because this WCT exhibited atypical RBBB morphology, it is most likely a left free wall AP or LV VT. An AES was therefore delivered to lateral CS, presumably adjacent to the location of the AP or the VT exit point. This AES advanced the next ventricular activation by 45 ms (V-V'=190 ms) and reset the WCT, without affecting the atrial timing of the HBp (A-A'=235 ms). This AES could not conduct to the ventricle through the AVN. A left free wall AP is therefore the antegrade limb of this WCT. **F.** A VES was delivered to the parahisian area, adjacent to the site of

earliest atrial activation, during the HB refractory period. The next atrial activation timing was advanced by 10 ms and WCT was reset, verifying that the retrograde limb of this preexcited AVRT was formed by another AP.

To exclude AVRT, *very early* AES must be delivered in close proximity to the site of earliest ventricular activation (the presumed AP location) to ensure that AES can engage the atrial end of the AP if present. If the WCT cannot be reset by *very early* AES, it essentially excludes the diagnosis of pre-excited AVRT (**Figure 4.26**). Choosing a pacing site to deliver AES plays a determinant role in the success of this maneuver. It is not uncommon that after multiple AES failed to reset the tachycardia, a very early AES eventually conducts to the ventricle through the AVN, producing a captured beat or fusion beat. This observation indicates no alternative route for AV conduction except for the AVN, further strengthening the diagnosis of VT.

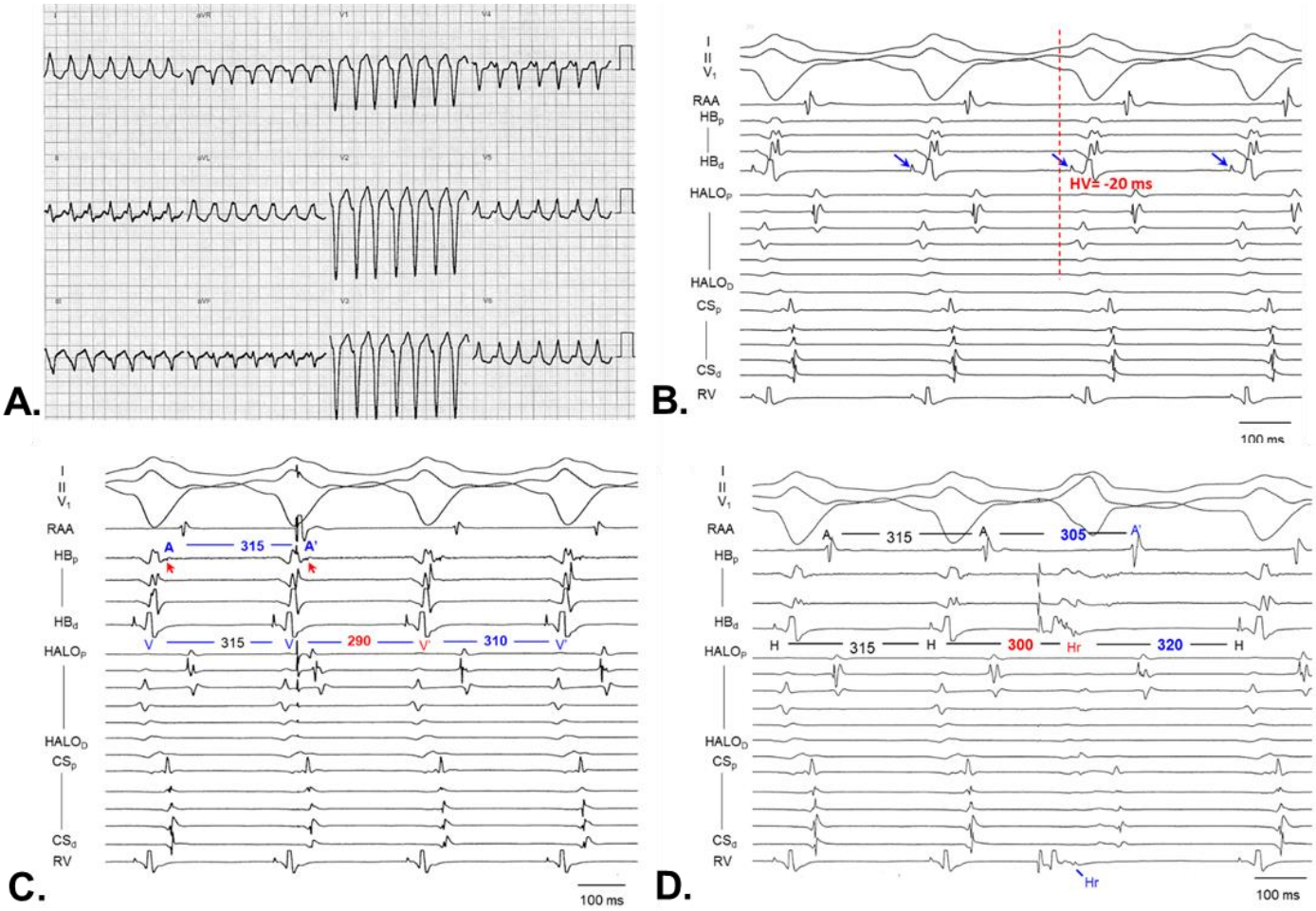


Figure 4.25. A 45 y/o female with WCT. A WCT showed typical LBBB morphology. **B.** Tachycardia CL was 315 ms; HV interval was -20 ms (blue arrow: HB potential), excluding SVT with aberrant conduction. **C.** An AES delivered to the RAA advanced the next ventricular activation timing by 25 ms (V-V'=290 ms) and reset the tachycardia. Note that the atrial timing of the HBp (red arrow) was not affected by the AES, proving that this WCT was a pre-excited AVRT. An atrio-fascicular AP forms the antegrade limb of the reentrant circuit of the tachycardia. **D.** A VES delivered from the parahisian area had to advance the retrograde HB potential by 15 ms before it could advance the next atrial activation timing by 10 ms and reset the tachycardia, proving that the retrograde limb of the reentrant circuit was the AVN fast pathway.

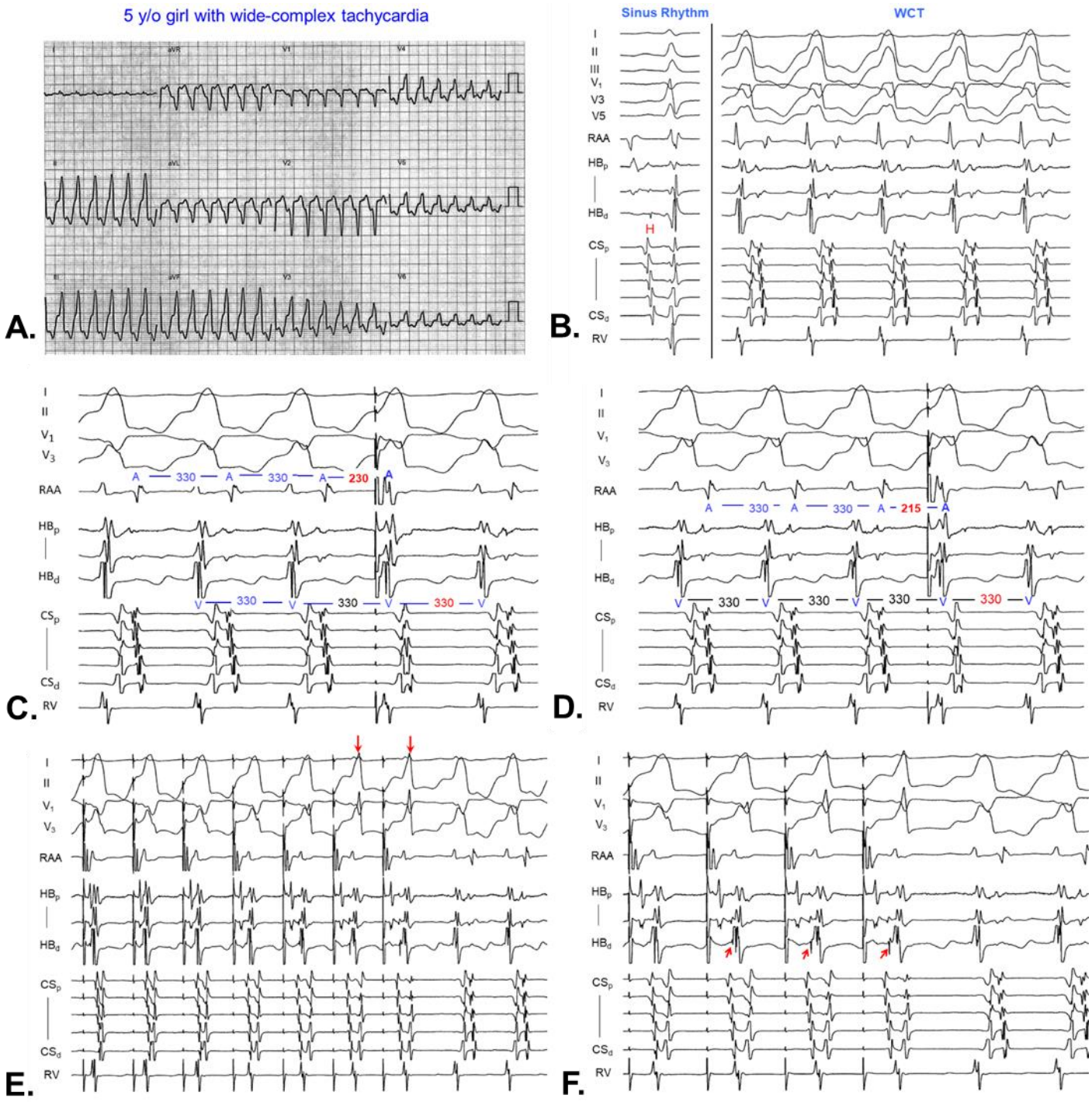


Figure 4.26. A 5 y/o child with wide-complex tachycardia. A. ECG suggests that this WCT was either an outflow tract VT or an antidromic AVRT using a right anteroseptal AP for antegrade conduction. **B. Left panel.** A HB potential was recorded before induction of tachycardia. **Right panel.** HB potential was not visible during WCT (CL=330 ms). **C**. Because this WCT showed LBBB with inferior axis, an AES 100 ms earlier was delivered to the RAA, adjacent to the site of earliest ventricular activation. This very early AES failed to affect the next ventricular activation timing. **D.** An earlier AES (115 ms earlier) still failed to affect the ventricular activation, making the diagnosis of antidromic AVRT using a right anterior/anteroseptal AP extremely unlikely. **E.** With prolonged atrial overdrive pacing, tachycardia was eventually perturbed (red arrow: narrower QRS complex) by RAA pacing. **F.** The paced atrial wave front had to conduct to the AVN (narrower QRS complex, fusion beats) before perturbing the tachycardia, making the diagnosis of antidromic AVRT using a right anterior or anteroseptal AP for antegrade conduction extremely unlikely. This WCT was successfully ablated in the RVOT area. Red arrow: antegrade HB potential.

Chapter 5:

Ablation of Accessory Pathways

Over half of the patients referred to Dr. Jackman's practice for accessory pathway (AP) ablation had failed prior ablation. Many others without prior ablation had APs in areas that are known to be challenging to ablate or the ablation carries a high risk of AVN injury. Dr. Jackman summarized the causes of AP ablation failure as follows.

(1) Localization error (60-70% cases). This is mainly caused by failure to appreciate that most APs traverse the AV annulus with an oblique angle. In other words, the angle between the course of an AP and the plane of the AV valve is not 90 degrees (**Figure 5.1A**).
(2) Wrong diagnosis (5-10% cases). For example, atrio-fascicular AVRT (Mahaim) may be misdiagnosed as AVNRT with aberrant conduction.
(3) Difficult to ablate or high risk of AVN injury (10-15% cases). Anteroseptal or mid-septal AP ablation carries a high risk of AV block; stable electrode-tissue contact can be difficult to maintain for a right free wall AP.
(4) Unusual location (5% cases). APs located in the coronary cusp or left anterior septum below the coronary cusp can be difficult to map and ablate.

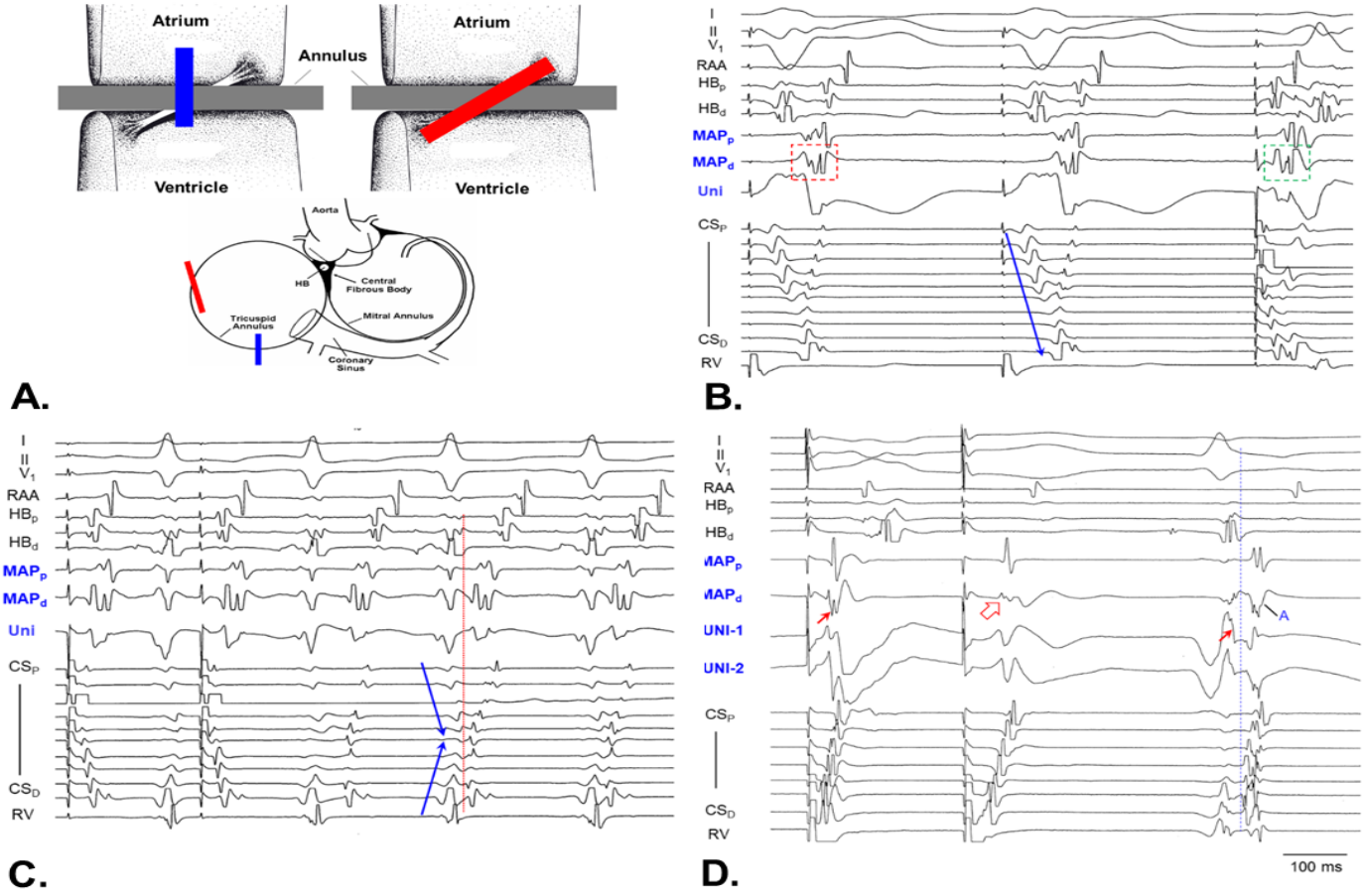


Figure 5.1. Oblique course of accessory pathway (AP) traversing the AV annulus. A. An AP takes a perpendicular (blue line) vs. an oblique (red line) course to traverse the AV annulus. **B.** The mapping catheter (MAP) recorded a continuous electrogram (VA fusion, in red box), viewed as a good ablation target by many electrophysiologists. Ventricular activation along

the CS was from CSp to CSd (blue arrow). Note that during antegrade AP conduction (3rd beat), the mapping catheter recorded AV fusion as well (in green box). **C.** When AVRT was induced a few seconds later, atrial activation at the same site was late. Note that the wave front of ventricular activation (blue arrows) changed during AVRT, leading to a longer VA interval recorded on the mapping catheter. The successful ablation site was 2 cm away from here. Vertical red line: earliest atrial activation. **D.** In another patient, the mapping catheter recorded a sharp AP-P (red arrow) which disappeared (empty red arrow) when antegrade AP conduction blocked. A sharp AP-P was recorded during retrograde AP conduction as well (3rd beat). Note that at the site where an AP-P was recorded, the atrial timing was significantly later than the timing of earliest atrial activation (vertical blue line), indicating that this AP traversed the annulus in an oblique course.

Causes of Accessory Pathway Ablation Failure

1. Localization Error:

One of the most important discoveries that Dr. Jackman made in AP ablation is that most APs traverse the AV annulus with an oblique angle. Conventional wisdom states that APs traverse the AV annulus in a direction perpendicular to the plane of the AV valve. A number of inferences come from this "wisdom". First, it would be exceedingly difficult to record an AP potential (AP-P) as it will be buried within the local atrial and ventricular electrograms (EGMs). Second, the best ablation target should be the site where the atrial and ventricular EGM fuse. Third, the AV or VA interval adjacent to the AP should be fixed regardless of the pacing site. Unfortunately, none of the three inferences hold true. Dr. Jackman also found that approximately two thirds of the failed AP ablation resulted from failure to appreciate the oblique course of APs. As soon as the oblique course is revealed by differential pacing, ablation becomes much easier. **Figure 5.1B** shows a possible ablation target with a continuous EGM (VA fusion). This site would be a good target if the AP were to traverse the mitral annuls perpendicular to the plane of the AV valve. When AVRT was induced a few seconds later, it was evident that atrial activation there was not early as shown in **Fig. 5.1C** and no AP-P was recorded at that site, either. Ablation was successful at a site 2cm away where a sharp AP-P was recorded.

The conclusion that most APs traverse the annulus with an oblique angle is supported by the following observations. The local AV interval during atrial pacing or the local VA interval during ventricular pacing at a site adjacent to the AP indeed changes significantly when pacing is delivered to the septal vs. lateral side of the AP (**Fig.5.2**). This viewpoint is also supported by a very common observation that the VA interval during RV pacing is substantially different from that during AVRT (**Fig. 5.1B-C**). Regardless of how the ventricle is activated, the ventricular activation wave front has to engage the ventricular end of the AP first before propagating to the atrium. Changes in the VA interval adjacent to the AP can easily be demonstrated by differential ventricular pacing. **Figure 5.2A-B** illustrates how a short VA interval is produced if the ventricular wave front propagates orthodromically to the ventricular end of the AP. In this scenario, the ventricular wave front propagation is aligned with the orientation of the AP. The ventricular wave front and AP conduction propagate in the same direction, masking the AP-P as well as the site of earliest atrial activation. When the wave front is reversed (**Figure 5.2C-F**), the ventricular wave front has to bypass the entire length of the AP before it engages the ventricular end of the AP. When the AP conduction propagates toward the atrium, it is no longer masked by the local ventricular activation thereby unmasking the AP-P and the site of earliest atrial activation.

Dr. Jackman defines an AP as taking an oblique course if the AV interval over antegrade AP conduction or the VA interval over retrograde AP conduction changes more than 15 ms by changing the wavefront entering the AP. Importantly, measuring the local AV or VA interval has to be performed at a site in close proximity to the AP. Obviously, the more oblique that an AP traverses the annulus (more deviation from the angle perpendicular to the AV annulus), the more change in the AV or VA interval one would observe. **Figure 5.3A** provides a detailed explanation of this important observation. Dr. Jackman's first choice of ablation target is always the site recording a sharp AP-P. In the best-case scenario, an isolated AP-P is spanned by brief iso-electrical intervals, which suggest that the target is the middle segment of the AP. In this way, mild catheter movement septally or laterally during ablation will not miss the ablation target. The site of earliest atrial or ventricular activation is Dr. Jackman's 2nd choice when an AP-P is nowhere to be found.

Figure 5.3B illustrates the problem of targeting the site of earliest atrial activation. To accurately identify the site of earliest atrial activation, the operator needs to choose the pacing site wisely (**Figure 5.1** and **5.2**). Even though the ablation catheter is positioned in close proximity to the site of earliest activation, there is still a good likelihood that the catheter may deviate away from the true site of earliest activation, leading to transient AP conduction block due to edema and recurrence of AP conduction later (blue hatched area in **Figure 5.3B**). For the vast majority of the left free wall APs, the ventricular end is more septal (of inferior) than the atrial end (**Figure 5.3C**). The right free wall APs follow a similar pattern but with more exceptions. Note that for the vast majority of the anteroseptal AP, the ventricular end is more lateral than the atrial end. Targeting the ventricular end is a safer approach (see **Chapter 6** for more details). Dr. Jackman usually does not target the site with AV or VA fusion because sites showing AV or VA fusion are often distant from the AP location. In the left panel of **Figure 5.3A**, sites beyond the atrial end of the AP will show VA fusion because the local ventricular activation is so late that it fuses with the local atrial activation; thus, creating a false impression that VA fusion is caused by very early atrial activation. If operators select the pacing site carefully to maximize the VA interval, a site with VA fusion is unlikely to be caused by a late local ventricular activation, but by an early local atrial activation. Ablation at this site is more likely to be successful.

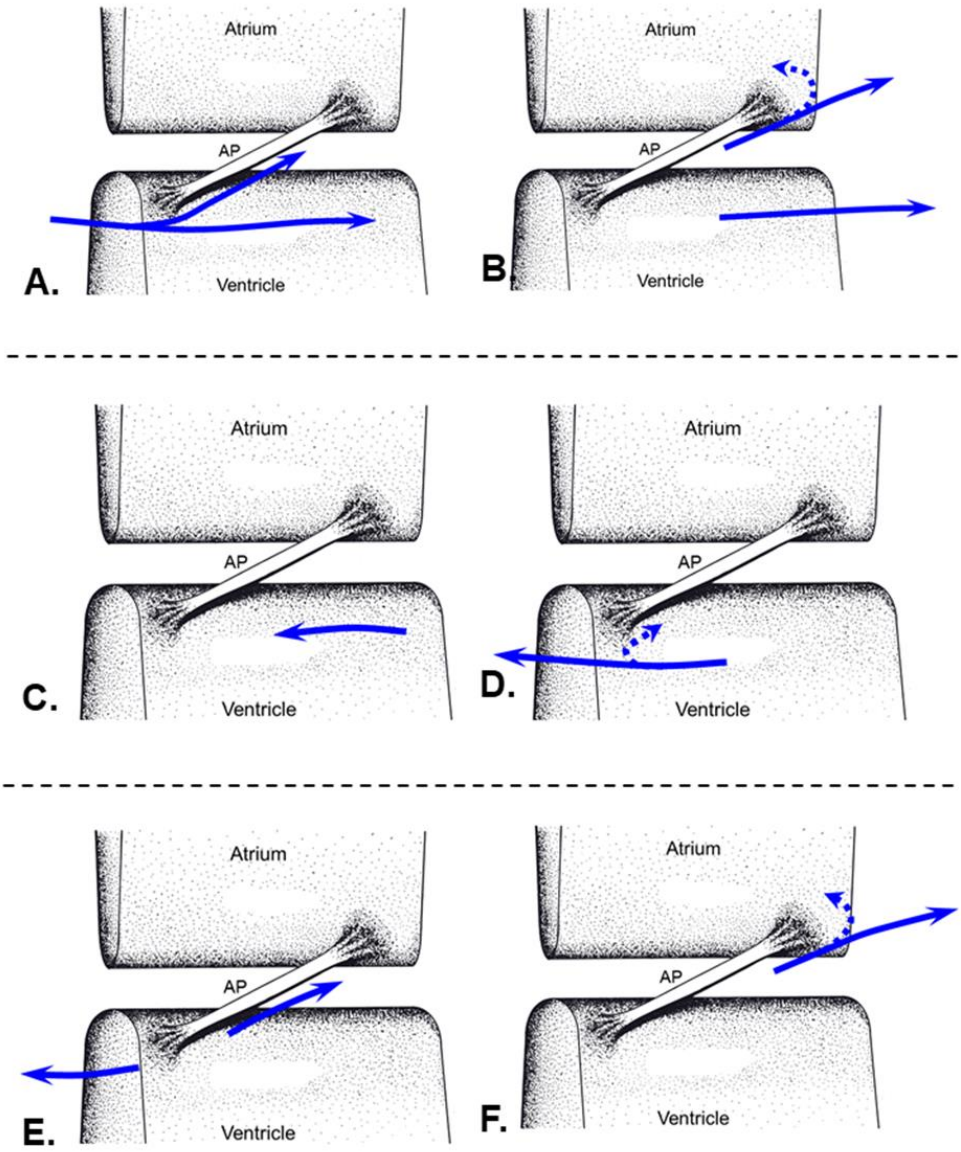


Figure 5.2. A-B. The ventricular wave front propagates in the same direction as retrograde AP conduction. The AP potential is masked by the local ventricular activation along the AP. **C-F**. When the wave front is reversed, the ventricular wave front has to bypass nearly the entire length of the AP before it engages the ventricular end of the AP. When the AP conducts toward the atrium, it is no longer masked by the local ventricular activation thereby exposing the AP potential. Courtesy of Dr. Jackman.

Before delivering differential pacing to determine the direction of the oblique course, Dr. Jackman prefers to quickly map the area of interest to figure out the vicinity of the site of earliest ventricular activating during atrial pacing or site of earliest atrial activation during ventricular pacing. This step is of great importance because meaningless results may be acquired if the most dramatic AV or VA interval change during differential pacing occurs at a site distant from the AP (**Figure 5.3D**). This is a major problem for a right free wall AP if no catheter is positioned along the tricuspid annulus. If the operator uses the change of the AV or VA interval in the CS to determine the oblique course of a right free wall AP, the results are essentially useless. For this very reason, the oblique course of a right free wall AP is often overlooked for the

absence of a catheter positioned along the tricuspid annulus, contributing to ablation failure of right free wall AP ablation. For a right free wall AP, Dr. Jackman either position a HALO catheter along the tricuspid annuls or position a mapping catheter in the vicinity of the AP before delivering differential pacing.

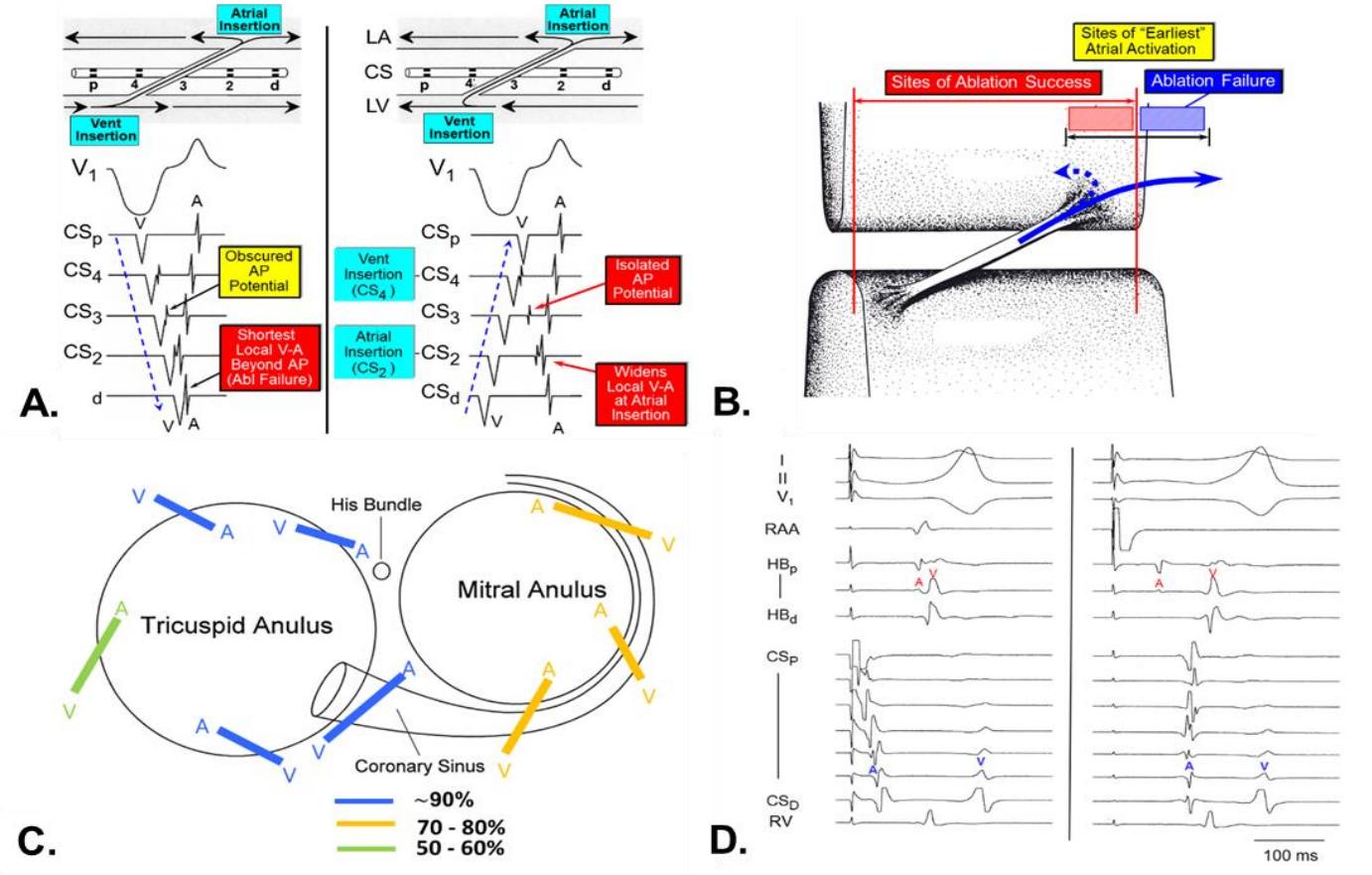


Figure 5.3. A. Illustration of how changes in ventricular wave front propagation can change the VA intervals and reveal the AP potential (courtesy of Dr. Jackman). The ventricular and atrial insertion of this AP is located at CS4 and CS2, respectively. **Left panel.** Ventricular wave front propagates from CSp to CSd, entering the AP at CS4. Because the AP and ventricular wave front propagate in the same direction, the AP potential in the center portion of the AP (CS3) is obscured by ventricular activation. Then, the ventricular and atrial wave front continue to propagate in the same direction, leading to a short VA interval at CS2. CSd, which already passes the atrial insertion of the AP at CS2 and shows VA fusion as well. Ablation there would not eliminate the AP. **Right panel.** When the pacing site is changed, ventricular wave front propagates from CSd to CSp, bypassing CS3 (the center portion of the AP) before entering the ventricular end of the AP at CS4. After the ventricular activation enters the AP at CS4, it propagates to CS3 at the time when the ventricular activation of CS3 is long gone, creating a long VA interval and exposing the AP potential. **B**. Even though the ablation catheter is positioned in close proximity to the site of earliest atrial activation, there is still a high likelihood that the catheter may deviate away from the true site of earliest activation, leading to ablation failure (blue hatched area). Therefore, Dr. Jackman prefers to target the center portion of the AP where an AP potential is recorded. **C**. The direction of the oblique course of APs around the mitral and tricuspid annulus is illustrated here. **D.** In a patient with an anteroseptal AP, the AV interval recorded by the HB catheter was significantly longer during RAA pacing than that during CS pacing, indicating that the atrial end of this anteroseptal AP was more septal than the ventricular end. The AV interval recorded by the CS catheter also showed significant changes during differential pacing but these changes are meaningless because the AP is not a left free wall AP. *Modified with permission from: Otomo K et al. Circulation. 2001 Jul 31;104(5):550-6.*

Dr. Jackman prefers to map both antegrade and retrograde AP conduction to select the best ablation site. Clearly, this approach gathers more information in deciding how to ablate an AP effectively and safely. To map a manifest AP, Dr. Jackman prefers to start with differential atrial pacing and ventricular pacing to determine if the AP takes an oblique course across the annulus as well as the ventricular and atrial end of the AP (**Figure 5.4**). To map antegrade AP conduction, differential pacing from the RA and CS is performed first. If it appears to be a left free wall AP, differential CS pacing from sites septal and lateral to the presumed AP location can help determine how the AP is slanted (**Figure 5.5A**). If the atrial end is more lateral than the

ventricular end, pacing from proximal CS will produce longer AV separation to facilitate mapping and ablation.

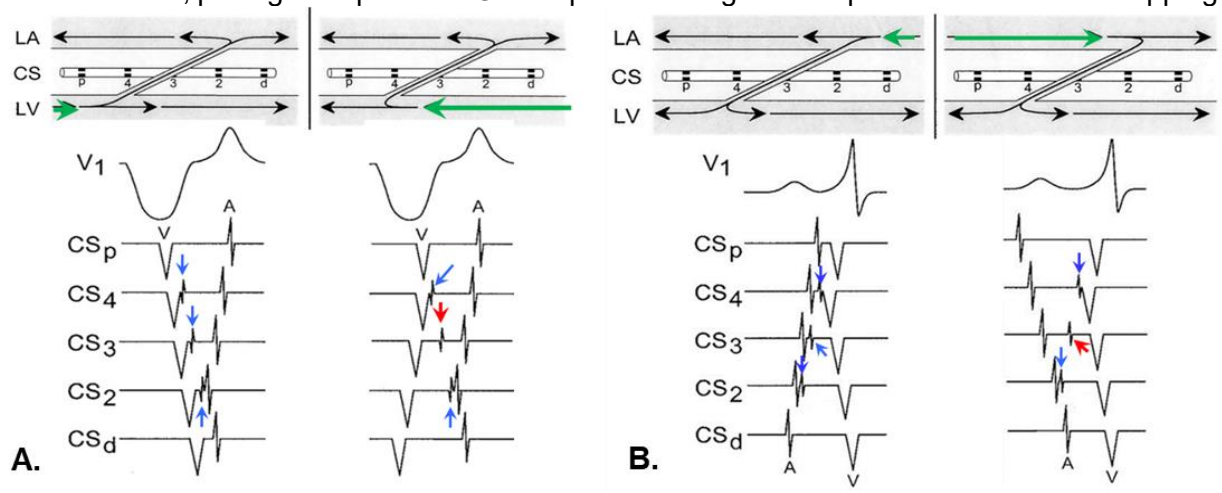


Figure 5.4. **Schematic representation of a left free wall AP with an oblique course revealed by differential ventricular (A) and atrial (B) pacing.** Green arrows: paced wave front. Note that reversal the paced wave front can expose the AP-P as well as the site of earliest ventricular and atrial activation. Blue arrows: obscured AP-P; red arrow: isolated AP-P exposed by reversal of wave front. *Modified with permission from: Otomo K et al. Circulation. 2001 Jul 31;104(5):550-6.*

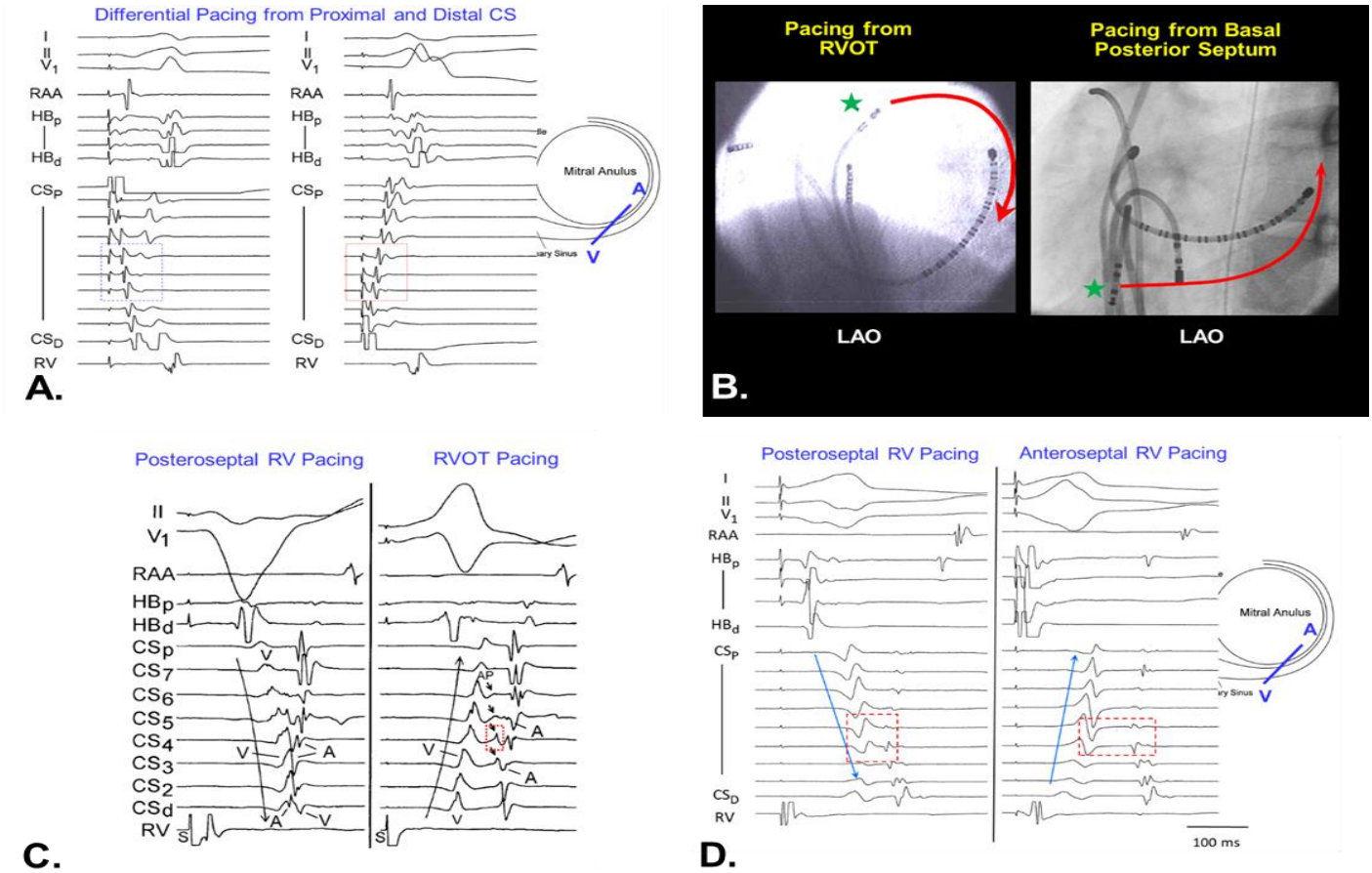


Figure 5.5. Differential pacing of a left free wall AP. A. In a patient with a left free wall AP, differential pacing was delivered to the proximal (CSp) and distal (CSd) CS. Pacing from CSd led to AV fusion (red box). Reversal of the atrial wave front by proximal CS pacing increased the AV interval (blue box), indicating that the atrial end of this AP is more lateral than the ventricular end. **B.** The ventricular pacing catheter was positioned at the RV outflow tract (**left panel**) and basal posteroseptal RV (**right panel**). Curved red arrows indicate the direction of the ventricular wave front. Green stars: pacing site. **C.** Differential pacing from posteroseptal RV (left panel) and RVOT (right panel). Note that posteroseptal RV pacing led to ventricular activation from CSp to CSd. RVOT pacing reversed the direction of ventricular activation thereby lengthening the VA interval and unmasking the AP potential (arrows). **D.** In a patient with a left free wall AP, reversal of the ventricular wave front by anteroseptal RV pacing lengthened the VA interval, indicating

that the ventricular end of the AP was more septal (or inferior) than the atrial end. *Modified with permission from: Otomo K et al. Circulation. 2001 Jul 31;104(5):550-6.*

In most of the left free wall APs, the ventricular end of the AP is more septal (or inferior) than the atrial end; pacing the ventricle lateral to the atrial end provides the best VA separation. For a left free wall APs located between the 1 and 3 o'clock position along the mitral annulus, RVOT pacing usually provides the longest VA separation to unmask the AP-P or the site of earliest atrial activation (**Figure 5.5B-C**). Operators can position the pacing catheter in the left pulmonary artery and slowly pull back the catheter during pacing until it stably captures the RVOT. Basal anteroseptal (parahisian site) pacing is the 2nd choice (**Figure 5.5D**) for mapping left anterior and left anterolateral APs. Dr. Jackman's practice is to start with differential pacing first. If it is a concealed left free wall AP, he would pace from the RVOT (or basal anteroseptal RV) and basal posteroseptal RV to select the pacing site that provides the best VA separation for mapping/ablation. If it is a manifest AP, he would pace the proximal and distal CS as well (**Figure 5.5A**). For a right free wall AP, differential pacing from the basal anterolateral (or anteroseptal) RV and basal posteroseptal RV is generally sufficient for the operator to determine the direction of the oblique course (**Figure 5.6**). For a manifest right free wall AP, differential pacing from the RAA and proximal CS helps the operator gain insight into the direction of the oblique course.

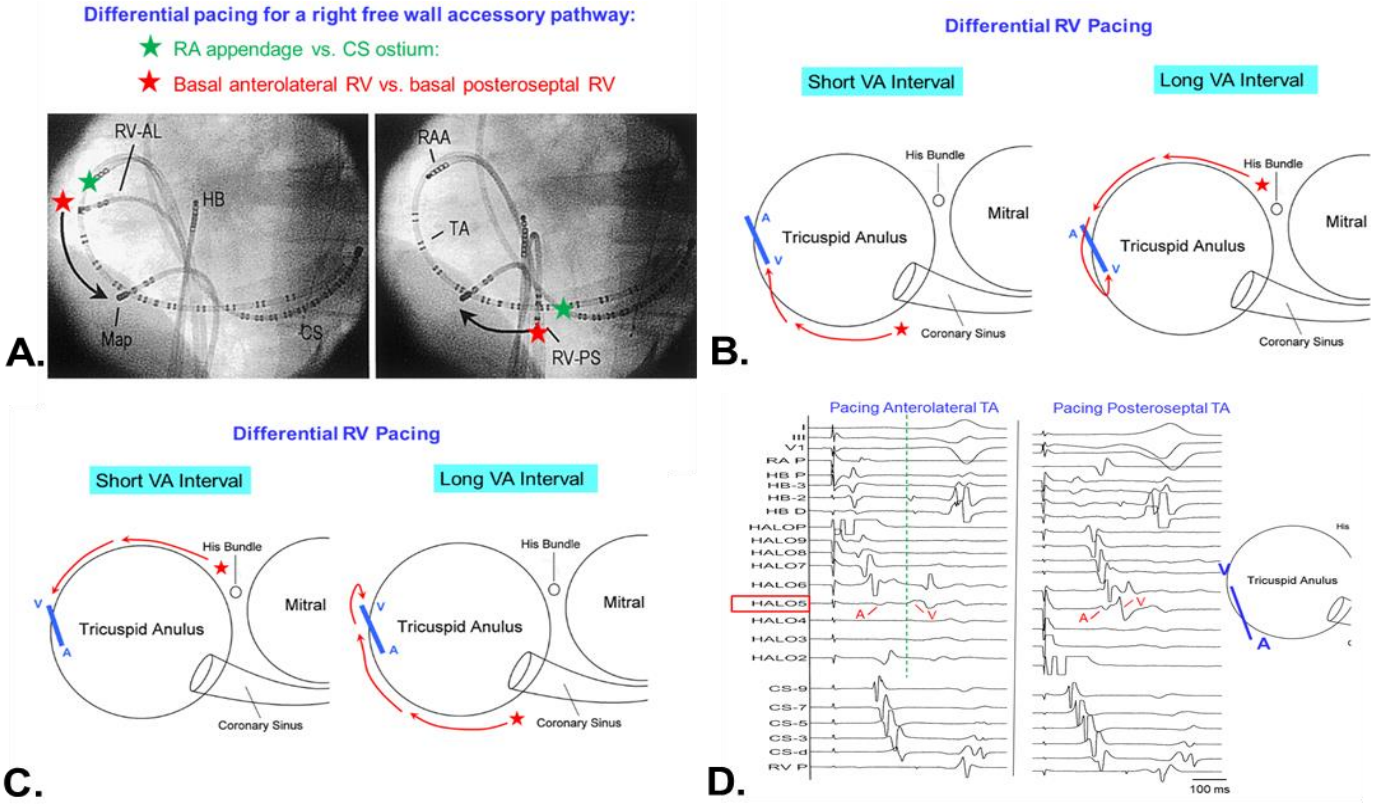


Figure 5.6. Differential pacing for a right free wall AP. A. Typical pacing sites for a right free wall AP. Atrial pacing: RA appendage vs. CS ostium. Ventricular pacing: basal anterolateral RV vs. basal posteroseptal RV**. B**. Schematic representation of VA interval change by reversal of the ventricular wave front in an AP whose ventricular end is more inferior and septal to the atrial end. **C.** Schematic representation of VA interval change by reversal of paced wave front in an AP whose ventricular end is more superior and lateral to the atrial end. **D.** A representative example of AV interval change caused by reversing the atrial wave front by pacing anterolateral (**left panel**) vs. posteroseptal RA (**right panel**). Note that the AV interval at the site of earliest ventricular activation (HALO-5) was significantly increased by pacing from the anterolateral tricuspid annulus. This observation indicates that the atrial end is more inferior and septal to the ventricular end.

Differential pacing is particularly helpful in mapping septal APs. For the vast majority of the anteroseptal (or parahisian) APs, the ventricular end is more lateral than the atrial end (**Figure 5.7A**). This characteristic has important clinical implications (see **Chapter 6** for details) as the AV node is on the atrial

side of the tricuspid annulus. Targeting the ventricular end of the AP is a preferred approach, which is a few millimeters lateral to the AV node and is also on the ventricular side of the annulus. Ablation of the ventricular end of the AP may cause RBBB but not AV block. Ablation failure of posteroseptal APs is often caused by diagnostic errors (see **Chapter 4**) or VA fusion along the course of the AP during RV apex pacing. In this scenario, VA fusion prevents the operator to identify the AP-P or the site of earliest atrial activation (see **Chapter 6** for more details). Dr. Jackman's favorite pacing site to map a concealed posteroseptal or left posterior AP is the lateral coronary vein, the same vein favored by operators implanting CRT leads (**Figure 5.7B**). Pacing from the lateral coronary vein lengthens the VA interval, unmasking the AP-P and the site of earliest atrial activation. If CS is too small to accommodate a pacing catheter and a mapping catheter, the pacing catheter can be positioned at the basal lateral LV using the retrograde trans-aortic approach.

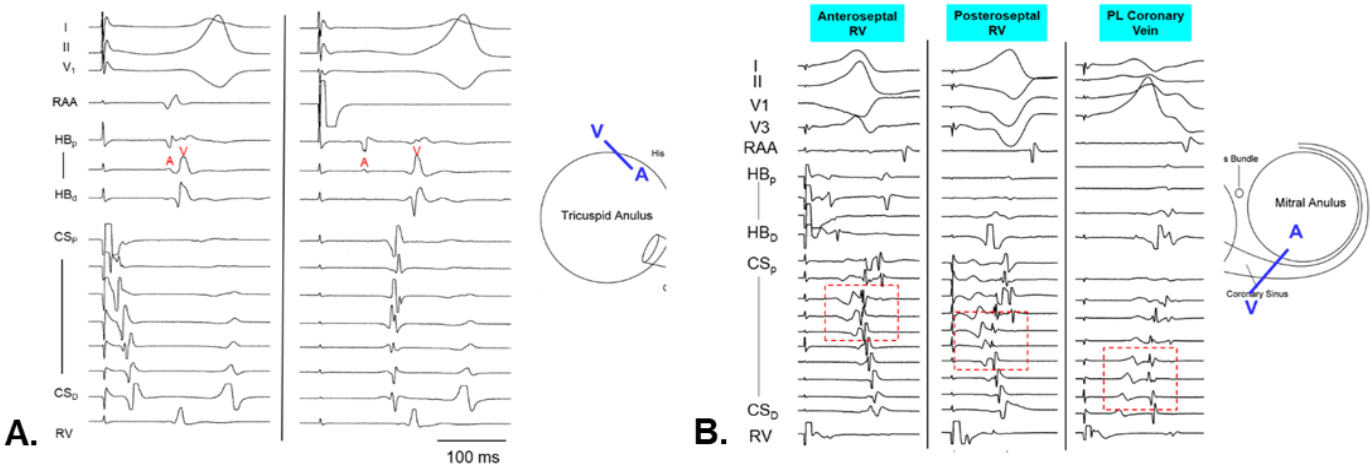


Figure 5.7. Effects of wave front change on AV or VA intervals of septal APs. A. In a patient with an anteroseptal AP, RAA pacing produced a longer AV interval at the site of earliest ventricular activation, indicating that the atrial end is inferior and septal to the ventricular end**. B.** In a patient with a posteroseptal AP, pacing from posterolateral coronary vein produced the longest VA interval at the site with earliest atrial activation (in red box), indicating that the ventricular end is more posterior and septal to the atrial end.

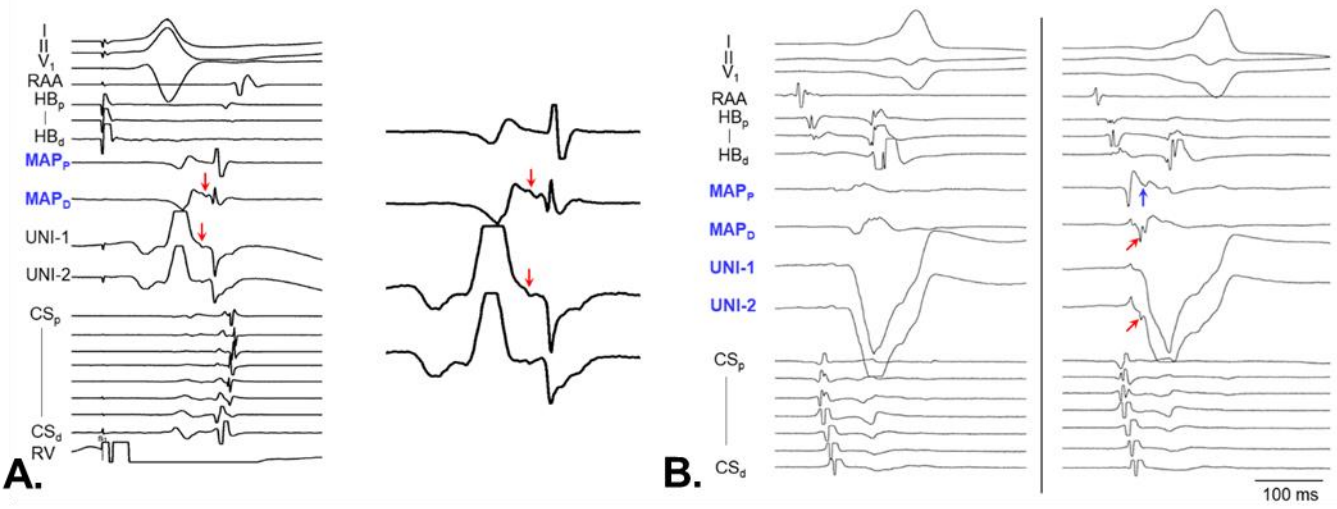


Figure 5.8. Far-field and near-field AP-P. **A.** A small far-field AP-P (red arrow) was recorded on the mapping catheter. Ablation here transiently blocked the retrograde AP conduction. **B. Left panel:** Mapping catheter positioned at the mitral annuls. The proximal electrode pair (MAP_p) recorded far-field, continuous potentials. **Right panel:** After adjusting the position of the mapping catheter, the proximal electrode pair (MAP_P) recorded a far-field AP-P (blue arrow). The distal electrode pair (MAP_D) recorded a sharp AP-P (red arrow). However, the AP-P was on the proximal ring electrode (UNI-2), not the distal electrode (UNI-1). This position is not ideal for ablation. AP conduction was eliminated immediately when the mapping catheter was repositioned and the AP-P was recorded on the distal electrode (UNI-1).

An important point that cannot be overemphasized is that differential pacing should be delivered near the annulus in order to facilitate the paced wavefront to engage the AP as all APs are located along the annulus. Dr. Jackman never paces from the RA free wall or RV apex to map an AP. One may think differential pacing to select the ideal ablation target is too time-consuming; however, in Dr. Jackman's experience, failure to effectively prolong the AV or VA interval accounts for 60-70% AP ablation failure!! This approach is definitely worthwhile if operators cannot find a good ablation target due to short AV or VA interval. In patients with prior ablation failure, local atrial or ventricular potential may have multiple components resulting from prior ablation. Poor choice of pacing sites may produce AV or VA fusion, misleading the operator to target the component of the local EGM that does not connect to the AP.

Validation of AP-P

The site recording a sharp AP-P is always Dr. Jackman's first choice of ablation target. An AP-P is almost always a sharp potential (similar to or just a bit wider than a HB potential) but the amplitude of the AP-P varies substantially. In Dr. Jackman's experience, the largest AP-P is often recorded in patients with Ebstein's anomaly. If a rounded AP-P is recorded, it probably is a far-field AP-P (**Figure 5.8A**); a sharp AP-P may be recorded a few millimeters away. If an AP-P is nowhere to be found (e.g. an epicardial AP) or the AP does not traverse the annulus with an oblique angle, the site of earliest atrial or ventricular activation is his second choice of ablation target. Dr. Jackman does not prefer to target the site of AV or VA fusion for the reasons already discussed. The only exception is *when the AV or VA interval has been maximized by differential pacing*. A site showing VA fusion is not because the local ventricular activation is late but the AP-P or atrial activation is early. A VA fusion site like this represents the V-AP-A junction but one cannot differentiate an AP-P from the local atrial and ventricular potential. Ablation there often works as well.

Differentiating an AP-P from a local ventricular or atrial potential can be a daunting task. **Figure 5.9** illustrates the technique which Dr. Jackman uses to prove the potential of interest is indeed an AP-P. In brief, this technique is to deliver single atrial extra-stimuli (AES) or single ventricular extra-stimuli (VES) to dissociate the potential of interest from the local atrial and ventricular potential. If the potential of interest is neither the local atrial or ventricular potential, it has to be an AP-P. Certainly, operators need to ensure the potential of interest is not a HB potential before starting ablation. In a manifest AP, particularly an anteroseptal AP, the HB potential may look like a sharp AP-P. The easiest way to differentiate a HB potential from an AP-P is to deliver decremental RA pacing. If the sharp potential of interest is progressively delayed along with more preexcitation, this potential is a HB potential. For a concealed AP, it is obvious that during sinus rhythm, this sharp should not be simultaneous with the HB potential.

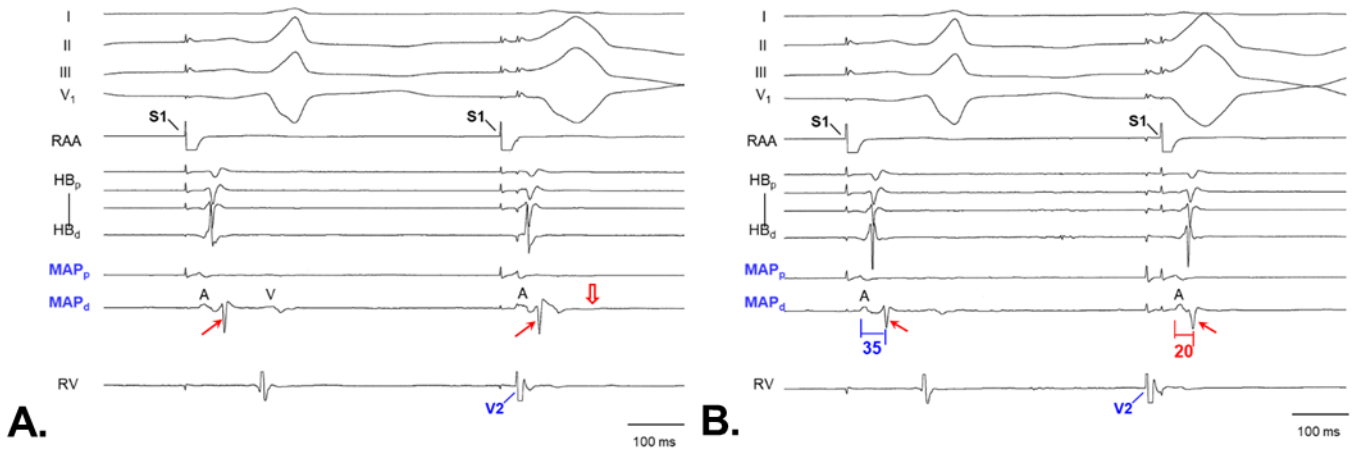


Figure 5.9. Pacing maneuvers to verify an AP-P. A. A sharp potential was noted (red arrow) during atrial pacing (S1). A ventricular extra-stimulus (VES, S2) advanced the local ventricular potential (empty red arrow) without affecting the sharp potential of interest, proving that this sharp potential was not part of the local ventricular potential. **B.** During atrial pacing, a much earlier VES (S2) engaged the ventricular end of the AP, allowing retrograde AP conduction from the ventricle to AP. The interval between the local atrial activation and sharp potential was shortened from 35 ms to 20 ms, proving that this sharp potential of interest was not part of the local atrial potential.

Another maneuver that is easy to implement but may not prove or disprove the potential of interest is to induce nonsustained AVRT or deliver decremental pacing until AP conduction blocks (**Figure 5.10**). Then, the operator can compare the EGM of AP conduction and AP block to identify the critical element of AP conduction. The author prefers to start decremental pacing at a CL 30-40 ms longer than the AP block CL to ensure good catheter stability and stable local atrial or ventricular EGMs at the time of AP block for accurate comparison between the AP conducted beat and AP blocked beat. If a sharp potential at the end of the local ventricular potential disappeared when retrograde AP conduction blocks, that sharp potential cannot be part of the ventricular potential, suggestive of an AP-P or a very early atrial potential (**Figure 5.10A**); ablation there has a high likelihood to be successful. However, the limitation of this technique is that if the potential of interest persists at the time of retrograde AP conduction block, this potential can still be an AP potential (**Figure 5.10B**) because the site of AP conduction block is between the AP and atrium. The author avoids using the S1S2 protocol because when S2 is delivered, the catheter tends to move. The local activation potential of the S2 may look different from that of S1, making comparison between the conducted beat and blocked beat difficult (**Figure 5.10C**).

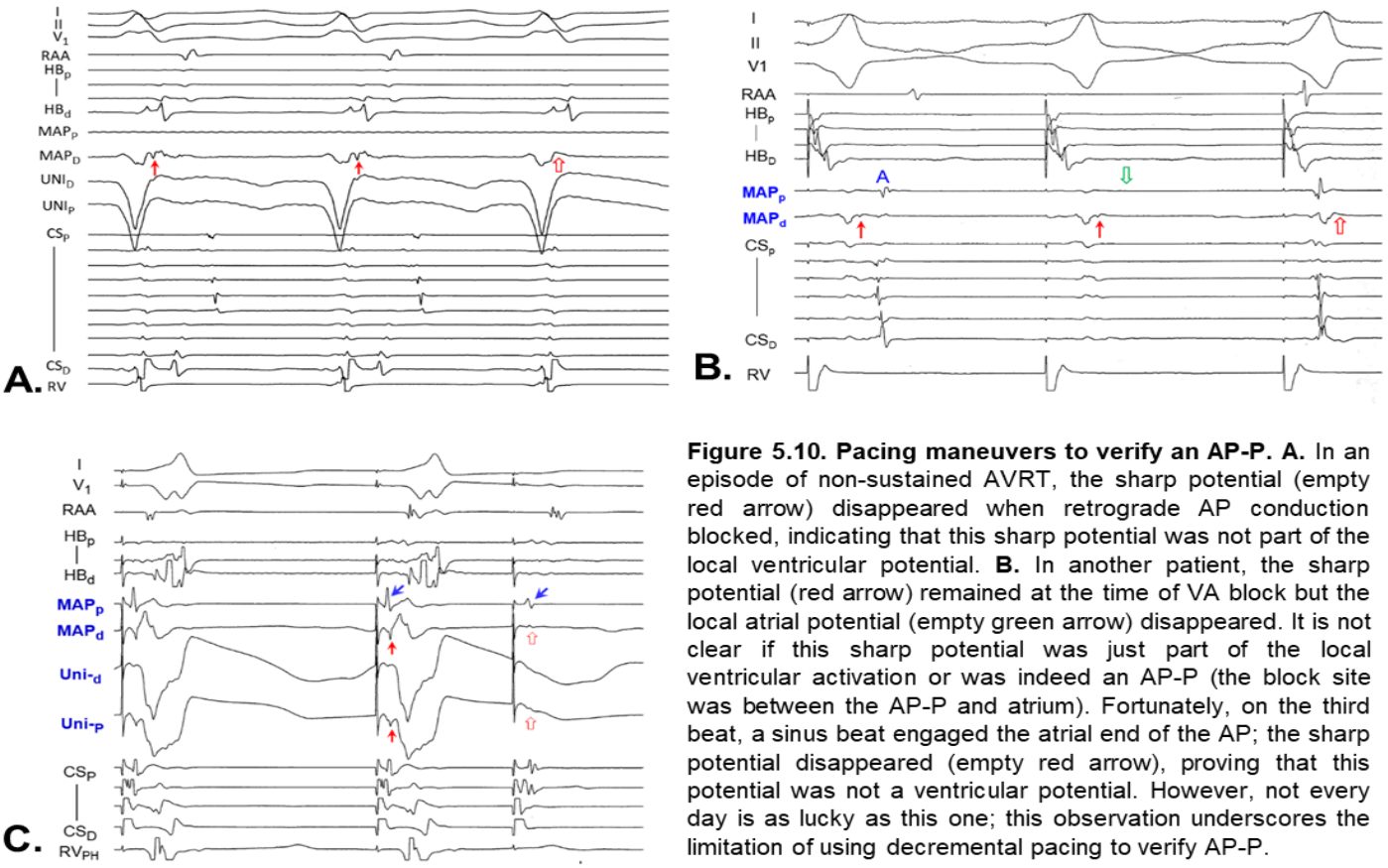


Figure 5.10. Pacing maneuvers to verify an AP-P. A. In an episode of non-sustained AVRT, the sharp potential (empty red arrow) disappeared when retrograde AP conduction blocked, indicating that this sharp potential was not part of the local ventricular potential. **B.** In another patient, the sharp potential (red arrow) remained at the time of VA block but the local atrial potential (empty green arrow) disappeared. It is not clear if this sharp potential was just part of the local ventricular activation or was indeed an AP-P (the block site was between the AP-P and atrium). Fortunately, on the third beat, a sinus beat engaged the atrial end of the AP; the sharp potential disappeared (empty red arrow), proving that this potential was not a ventricular potential. However, not every day is as lucky as this one; this observation underscores the limitation of using decremental pacing to verify AP-P.

C. In another patient with a mid-septal AP, an atrial extra-stimulus led to antegrade conduction block and disappearance of the sharp potential (empty red arrow). Note that the local atrial potential (blue arrow) looked very different, suggesting that the catheter might have moved by the extra-stimulus. Sometimes, catheter movement can lead to difficulty in identifying if the potential of interest is affected by extra-stimuli.

Based on Dr. Anton Becker's description, APs appear to be an extension of the atrial myocardium. The atrial end may have multiple branches that Dr. Becker called it "mangrove tree roots". This finding leads to a common observation that over retrograde AP conduction, a relatively large area may record earliest atrial activation, particularly when widely-spaced electrode catheters are used. Antegrade conduction from atrium to AP is usually robust. For a left free wall AP, antegrade AP conduction block typically occurs at the ventricular end (A→AP→block); conduction block like this occurs in only half of the patients with a right free wall AP. Such differences may result from a larger source-sink mismatch of left free wall APs caused by a larger LV mass. If antegrade AP conduction block occurs at the ventricular end (the AP-V junction), an *antegrade* AP potential may still be recorded in a concealed AP.

Localization of AP by preexcitation pattern

There is a wealth of literature about predicting the location of an AP based on the preexcitation pattern. Decades ago, one of Dr. Jackman's favorite fellows, Dr. Xunzhang Wang, discovered that if lead V1 begins with a small, broad r wave, followed by two downward deflections, it is almost always a right free wall AP (**Figure 5.11A-C**). This observation holds true in the vast majority of cases. It is not uncommon for a patient who was referred to the OU-EP group for an anteroseptal AP ablation with an ECG showing that lead V1 began with a small but broad r wave, followed by two downward deflections. Ablation site was always not adjacent to the septum. Dr. Jackman also uses the delta wave in aVF to help determine if the ventricular end is superior or inferior to the CS. If the delta wave in aVF is positive, the AP is usually above the level of the CS ostium; if it is negative, the AP is usually at or below the level of the CS ostium.

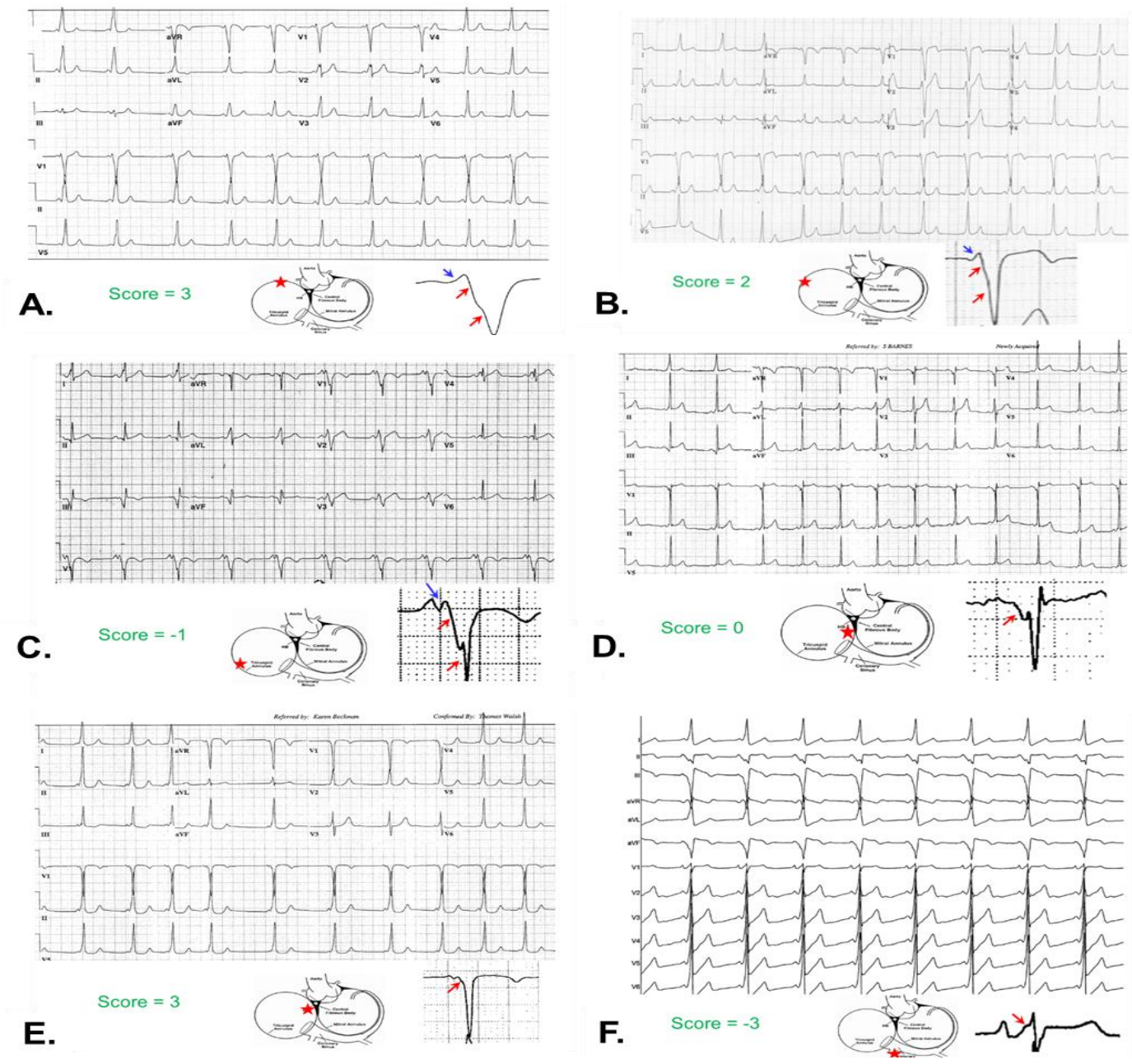


Fig 5.11. ECG localization of right-sided APs. A. A right anterior AP. Lead V1 began with a small, broad r wave (blue arrow) followed by two downward deflections (red arrows). This is a typical pattern for a right free wall AP. The score of the delta wave polarity in lead II, III and aVF is 3, indicating that this AP is superiorly located. The red star indicates the location of the AP

where it was successfully ablated. **B.** A right lateral AP. **C.** A right posterolateral AP. **D.** A right midseptal AP. Note that the delta wave in lead V1 begins with a q wave, different from a free wall AP shown in A-C. **E.** An anteroseptal AP. The delta wave also begins with a q wave. **F.** A posteroseptal AP using the middle cardiac vein as part of the AP-LV connection. Note that the delta wave in lead V1 begins with an r wave and the QRS complex exhibits an rs morphology, suggestive of a left-sided AP. Inset: location of successful ablation and ECG of lead V1.

By combining Dr. Jackman's algorithm with several published algorithms, the author uses the following criteria to predict the ventricular end of an AP.

1. Analyze only the first 30-40 ms of the delta wave, not the polarity of the entire QRS complex because the rest of the QRS complex may be formed by AVN conduction. Sometime, it can be difficult to determine where delta wave begins. Dr. Jackman uses the onset of the delta wave in lead I to examine the beginning of the delta wave in other leads.

2. Look at lead V1 first. If V1 starts with a QS pattern and V2 shows a positive delta wave (early precordial transition), it is almost always a septal AP (**Figure 5.11D-E**). If V1 shows an rS pattern, the larger or wider the r wave, the more likely it is an AP at a safe distance from the AV node.

3. Score the delta wave in lead II, III an aVF to determine how superior or inferior this AP is located. If the delta wave is positive, give it a score of 1. If it is negative, give it a score of -1. Iso-electrical delta wave gets a score of 0. Adding the scores up, an anteroseptal AP or anterior AP usually gets a score of 3; a posteroseptal AP or posterior AP usually gets a score of -3. Mid-septal or lateral free-wall AP usually gets a score between +1 to -1 (**Figure 5.11**).

4. If the delta wave is positive in both lead I and aVL, it is usually a right-sided AP (**Figure 5.11**). However, a left posteroseptal or left posterior AP can produce a positive delta wave in both lead I and aVL because the preexcitation wave front propagates toward these two leads. Only detailed mapping can differentiate a left posteroseptal AP from a right posteroseptal AP.

5. For a left free wall AP, the delta wave in lead V1 usually is isoelectric or positive. The entire QRS complex of V1 usually exhibits an atypical RBBB pattern or an rs/RS pattern (**Figure 5.12**). The QRS complex of the V1 lead is more likely to be rS or QS in the presence of a right-sided AP.

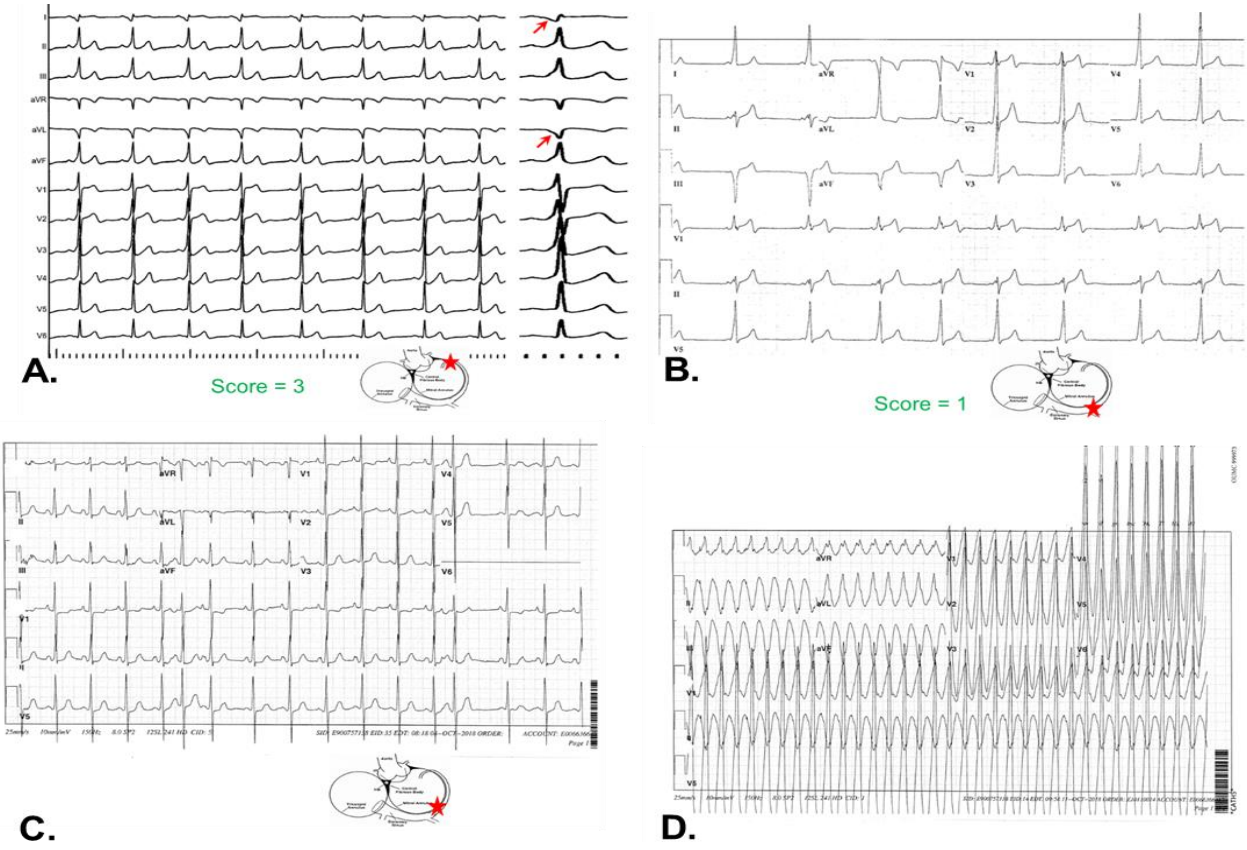


Figure 5.12. ECG localization of left-sided APs. A. A left anterior AP located at the 1:30 o'clock position along the mitral annulus. Note that the delta waves in both lead I and aVL was negative. **B.** A left posterior AP located at the 4:30 o'clock position along the mitral annulus. Note that the delta wave was positive in both lead I and aVL. The morphology of V1 (positive delta wave, an R complex) helps predict that this is a left free wall AP. **C and D. In a patient with a left posterolateral AP located at the 4:00 o'clock position along the mitral annulus.** Note that in the baseline state, there was only minimal preexcitation. During preexcited tachycardia, the preexcitation looked different from that in the baseline state but the polarity of the first 40 ms was identical.

6. For a left free wall P, if the delta wave is negative in both lead I and aVL, it is most likely an AP located along the 1:00 to 2:00 position along the mitral annulus (**Figure 5.12A**). If the delta wave is positive in both lead I and aVL, it is most likely an AP located along the posteroseptal or posterior mitral annulus because the preexcitation wave front propagates toward these two leads (**Figure 5.12B**).

7. The baseline preexcitation pattern can be very different from that in preexcited AF or antidromic AVRT because of lack of the contribution from antegrade AVN conduction. In the presence of full preexcitation, the entire QRS complex is formed by antegrade AP conduction; the polarity of the QRS complex may look completely different from that in sinus rhythm when only the first 30-40 ms of the QRS complex is formed by antegrade AP conduction. However, the polarity of the 40 ms of the QRS should be identical. Most, if not all, of the published ECG algorithms were based on sinus rhythm, not full preexcitation.

2. Diagnosis Error

Chapter 4 provides a detailed discussion on differential diagnosis of SVT. In brief, orthodromic AVRT caused by a concealed posteroseptal AP can easily be confused with fast/slow or slow/slow AVNRT (**Figure 5.13A-B**). Most of such diagnostic errors can be prevented by parahisian pacing as well as delivering ventricular extra-stimuli (VES) during AVRT from the basal posteroseptal RV adjacent to the ventricular end of the AP. Slow/fast AVNRT may present with a longer HA interval and a VA interval >70 ms, masquerading as orthodromic AVRT using an anteroseptal or mid-septal AP for retrograde conduction (**Figure 5.13C-D)**.

Tachycardias using the His-Purkinje system as part of the reentrant circuit (e.g. fascicular VT or atrio-fascicular AP) can be misdiagnosed as SVT with aberrant conduction when 1:1 VA conduction is present. This mistake can easily be prevented by recording a stable HB potential during the tachycardia. In fascicular VT or atrio-fascicular AVRT, the HV interval during tachycardia is typically a negative value or substantially shorter than the HV interval in sinus rhythm (**Figure 11.18**). The rule of thumb is that if the HV interval during tachycardia is shorter than that during sinus rhythm, the HB potential during tachycardia is retrogradely activated, thereby excluding the diagnosis of SVT with aberrant conduction. Dr. Jackman's practice is to secure a stable HB catheter position before any attempts to induce tachycardia to avoid terminating the tachycardia by manipulating the HB catheter to record a HB potential during tachycardia.

3. Difficult to ablate or too close to the AVN

APs using the musculature of the CS or its tributary vein(s) are notorious for being difficult to map and ablate. Ablation in this location faces 3 main challenges. First, EGMs recorded from this location contains ventricular, atrial and CS myocardial potentials. Pacing from the RV apex often produces a short VA interval and fusion of the 3 potentials, leading to localization errors and ablation failure. Second, a large branch of the right coronary artery (the posterior-lateral branch) is often in close proximity to the floor of proximal CS or the orifice of the middle cardiac vein. Ablation within 5 mm distance from the coronary artery carries a high risk of coronary artery injury. Third, poor electrode cooling and high impedance lead to low wattage of RF current delivered to the target. Fortunately, this problem is mostly solved by irrigated-tip catheter that provides appropriate cooling of the ablation electrode to allow delivery of >15 watts of RF current. Ablation of anteroseptal (parahisian) and mid-septal APs is often considered a high-risk procedure due to their close proximity to the AV node. In Dr. Jackman's practice, the incidence of significant AVN injury is approximately 1-2% including many patients who had multiple prior unsuccessful ablations. Dr. Jackman's approach to septal AP ablation is discussed in great detail in **Chapter 6.**

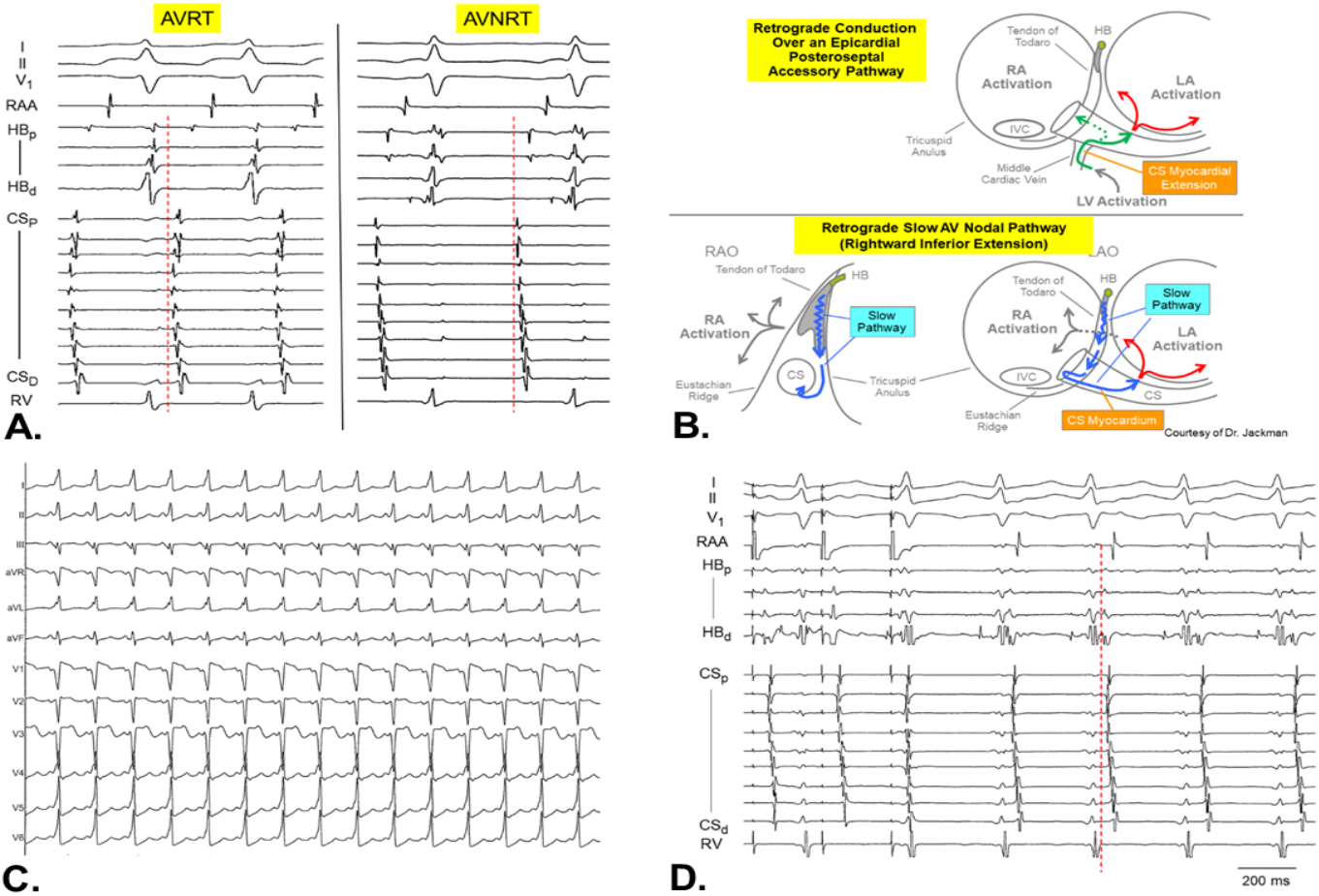


Figure 5.13. AVNRT may masquerade as orthodromic AVRT. A. Both AVNRT and AVRT were induced in the same patient. The site of earliest atrial activation (vertical red lines) was recorded from the proximal CS in both tachycardias. Note that both pathways activated the CS muscle extension and LA in a similar way. **B.** Schemas depict retrograde conduction over an epicardial posteroseptal AP (top) and retrograde slow pathway conduction (bottom). Courtesy of Dr. Jackman. **C**. In another WPW patient, preexcitation pattern predicts a midseptal AP. **D.** SVT with a VA interval of 75 ms was induced; the site of earliest atrial activation (vertical red line) was in the HB region. This SVT was proven to be slow/fast AVNRT, not AVRT (now shown here). Ablation of the slow pathway eliminated this tachycardia.

Poor electrode-tissue contact

The main cause of ablation failure of right free wall APs is poor electrode-tissue contact. For left free wall APs, poor electrode-tissue contact is also a major cause of ablation failure, particularly using the trans-septal approach. Three decades ago, Dr. Jackman did a canine study showing that ablation above or below the mitral valve created similar lesions. The retrograde trans-aortic approach is still widely used outside the USA. This approach is more difficult to master and carries a small risk of stroke and myocardial infraction. However, in experienced hands, the ablation electrode is tugged under the mitral valve with great stability. If appropriate cooling of the ablation electrode can be achieved, the AP is almost always eliminated quickly. In contrast, the trans-septal approach ablates the AP above the mitral valve; catheter stability is always a challenge. Dr. Jackman prefers a long sheath to bring the ablation catheter to the AV annulus so that the catheter can be laid in parallel to, not perpendicular to the annulus. In addition, the trans-septal puncture should aim at the anterior and superior aspect of the fossa ovalis, not the posterior aspect like an AF ablation. This is particularly important for a dilated LA in which a posterior puncture will require a lot of counterclockwise rotation of the sheath to provide acceptable electrode-tissue contact along the mitral annulus.

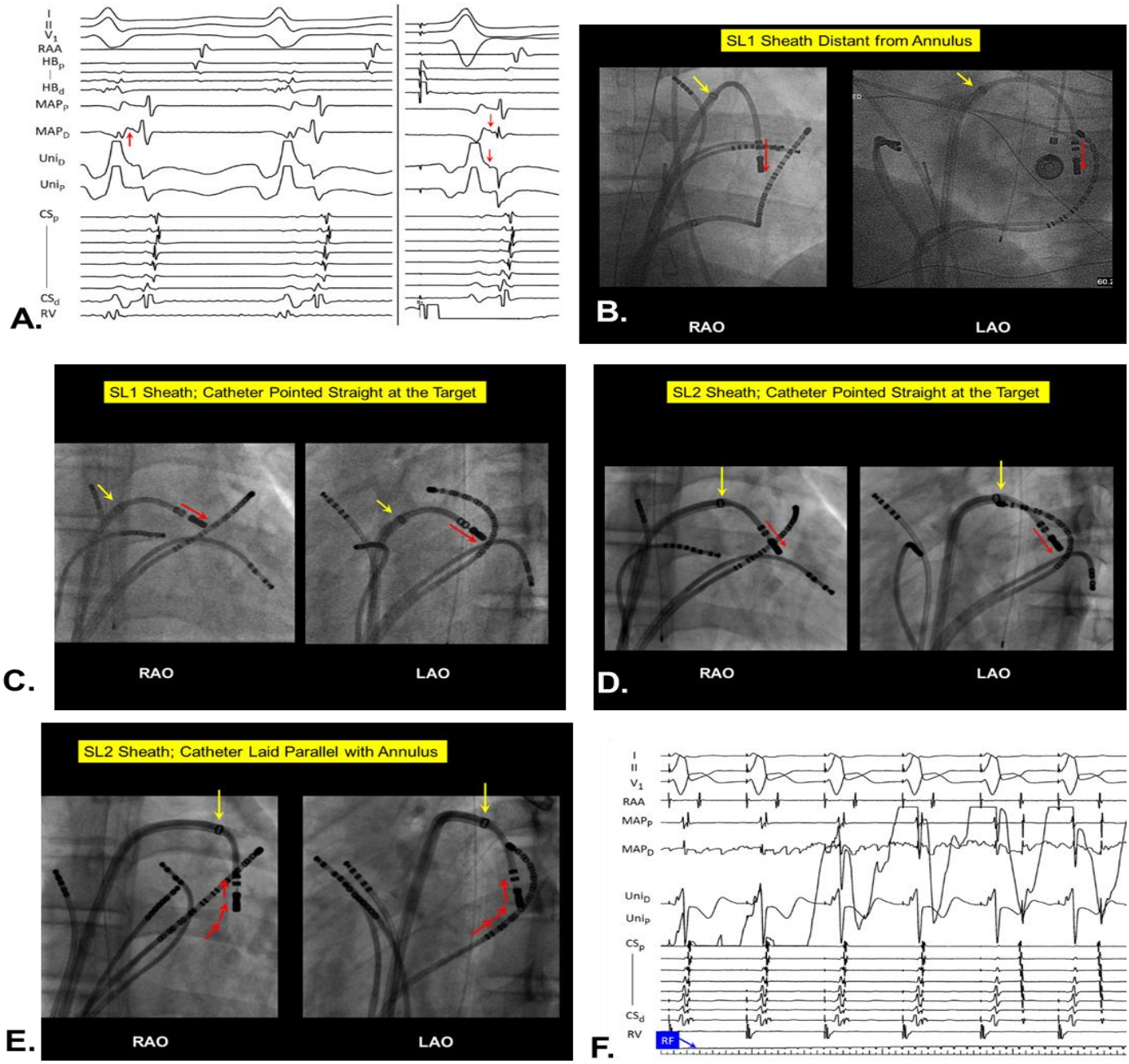


Figure 5.14. Catheter stability for ablation of a left free wall AP. A. Left panel: AVRT; Right panel: RV pacing. A small far-field AP potential (red arrow) was recorded on the mapping catheter. Ablation here transiently blocked the retrograde AP conduction. **B.** The trans-septal puncture site was too posterior; the SL1 sheath was raised upward in attempt to allow the mapping catheter to lay in parallel to the mitral annulus (red arrows) but stability remained poor as the tip of the sheath (yellow arrows) was too far away from the mitral annulus, not serving as a good anchor. **C.** The SL1 sheath was pulled down. The ablation catheter was pointing straight at the target (red arrow), in perpendicular to the plane of the mitral valve. It took 10" to cause AP conduction block. EGM appeared stable but x-ray showed otherwise. Note that stable EGM does not equal stable electrode-tissue contact. The tip of the SL1 sheath (yellow arrow) was positioned posteriorly in the LA and distant from the mitral annulus, not providing enough support for the ablation catheter. **D**. An SL2 sheath brought the catheter toward the mitral annuls. However, the ablation catheter still pointed straight at the target. **E.** The SL2 sheath took the ablation catheter to the mitral annulus. The ablation catheter was laid in parallel with the plane of the annulus to improve electrode-tissue contact. The ablation catheter was initially positioned inferior (septal) to the presumed target. The catheter was then slowly withdrawn toward the target (red arrows). **F.** AP was successfully eliminated in 2 seconds.

Figure 5.14 illustrates different orientations of the catheter in relation to the mitral annulus and ablation target. Dr. Jackman uses the technique of rotating the trans-septal sheath counterclockwisely toward the mitral annulus and rotate the ablation catheter clockwisely to contact the annulus. This approach is a bit

counter-intuitive but provides more stable electrode-tissue contact. The ablation electrode is first positioned inferior (or septal) to the presumed target. The most important step is to lay the ablation catheter in parallel with the annulus; then, the ablation catheter is slowly withdrawn until it reaches the target. During ablation, the ablation electrode moves in parallel with the movement of the annulus, providing better stability. Dr. Jackman avoids pointing the ablation catheter straight at the target. The author calls this approach "woodpecker's approach" because the ablation electrode moves perpendicular to the movement of the annulus. Catheter stability is at the mercy of ventricular contraction! Dr. Jackman only uses SL1 when the location of the AP is very anterior (e.g. one to two o'clock position along the mitral annulus).

Dr. Jackman use the same approach in ablating right free wall APs, including the atrio-fascicular AP (Mahaim fiber) which is notorious for being mechanically traumatized. In difficult right free wall AP ablations, Dr. Jackman may use a HALO catheter at first to help localize the AP-P. For ablation, the HALO catheter is positioned slightly atrial to the tricuspid annulus in order to "lock" the ablation catheter between the annulus and the HALO catheter (**Figure 5.15**).

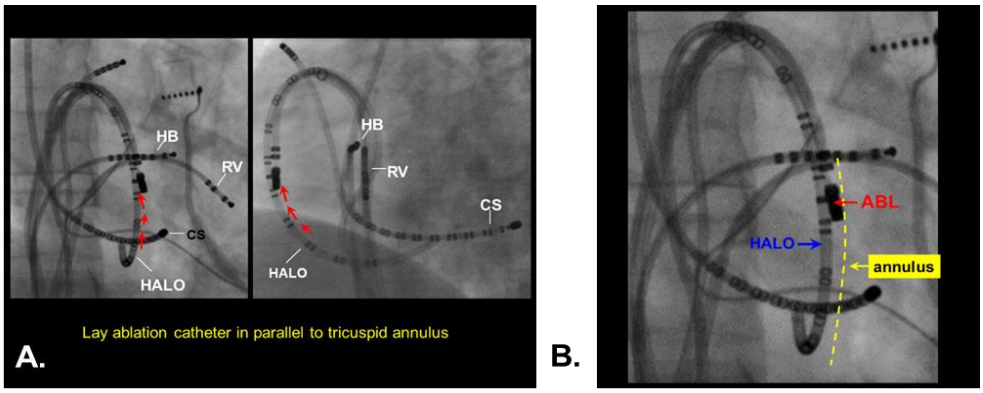


Figure 5.15. Catheter stability for ablation of a right free wall AP. A. A HALO catheter was deployed to assist mapping. The ablation catheter was positioned in parallel to the plane of the tricuspid valve and inferior (septal) to the presumed target. The ablation catheter was then slowly withdrawn toward the target (red arrows). **B.** Enlarged view. Note that the HALO catheter was used to "lock" the ablation catheter between the tricuspid annulus and HALO catheter to improve electrode-tissue contact.

Dr. Jackman may use the following sheaths to assist ablation:

1. SR1 or SR1: right anterior or anterolateral AP
2. SR2: right lateral or posterolateral AP
3. SR3: right posterolateral or posterior AP

If the RA is small, an SR3 sheath will bring the catheter deep into the RV. An SR2 sheath may be more suitable for a right posterior AP. For both right and left free wall APs, the most important maneuver is to keep the sheath superior and annular enough to allow the ablation catheter to be positioned in parallel with the AV valve and allow the tip of the ablation catheter to be positioned posterior and/or septal to the AP. The ablation catheter is then slowly withdrawn toward the target in parallel with the annulus.

AV balance at the target site

The AV balance of the EGM on the AV annulus is an important factor in terms of choosing the ablation target. In the OU-EP laboratory, the A/V or V/A ratio of the EGM was not used as part of the target selection criteria because ventricular EGMs are often clipped to avoid overlapping with other EGMs. Dr. Jackman's first choice is a site recording a sharp AP-P, with a large ventricular EGM and small atrial EGM; this is particularly important for left free wall APs (**Figure 5.16**). The basal LV myocardium adjacent to the mitral valve is substantially thicker than that of the atrial myocardium. A site showing an EGM with an A:V ratio of 1 is likely in the left atrium, not on the mitral annulus. For right free wall APs, the basal RV myocardium adjacent to the tricuspid annulus is not as thick as that of the LV. The A:V ratio at a successful ablation site for a right free wall AP is therefore not as small as that of the left free wall APs (**Figure 5.16C-D**). However, the ideal ablation target typically still should exhibit a larger ventricular EGM to ensure that the ablation site is not too atrial. Dr. Jackman would not settle for an A:V<1 site unless a sharp AP-P is recorded at that site as well (atrial end of the AP). If one wants to target the earliest atrial or ventricular activation when the ablation catheter is

positioned under the AV valve (e.g. retrograde trans-aortic approach for a left free wall AP), s/he needs to find a site with the largest atrial potential. When the ablation catheter is positioned above the AV valve (e.g. trans-septal approach for a left free wall AP), s/he needs to find a site with the largest ventricular. In this way, operators can be certain that the tip electrode of the ablation catheter is positioned on the AV annulus.

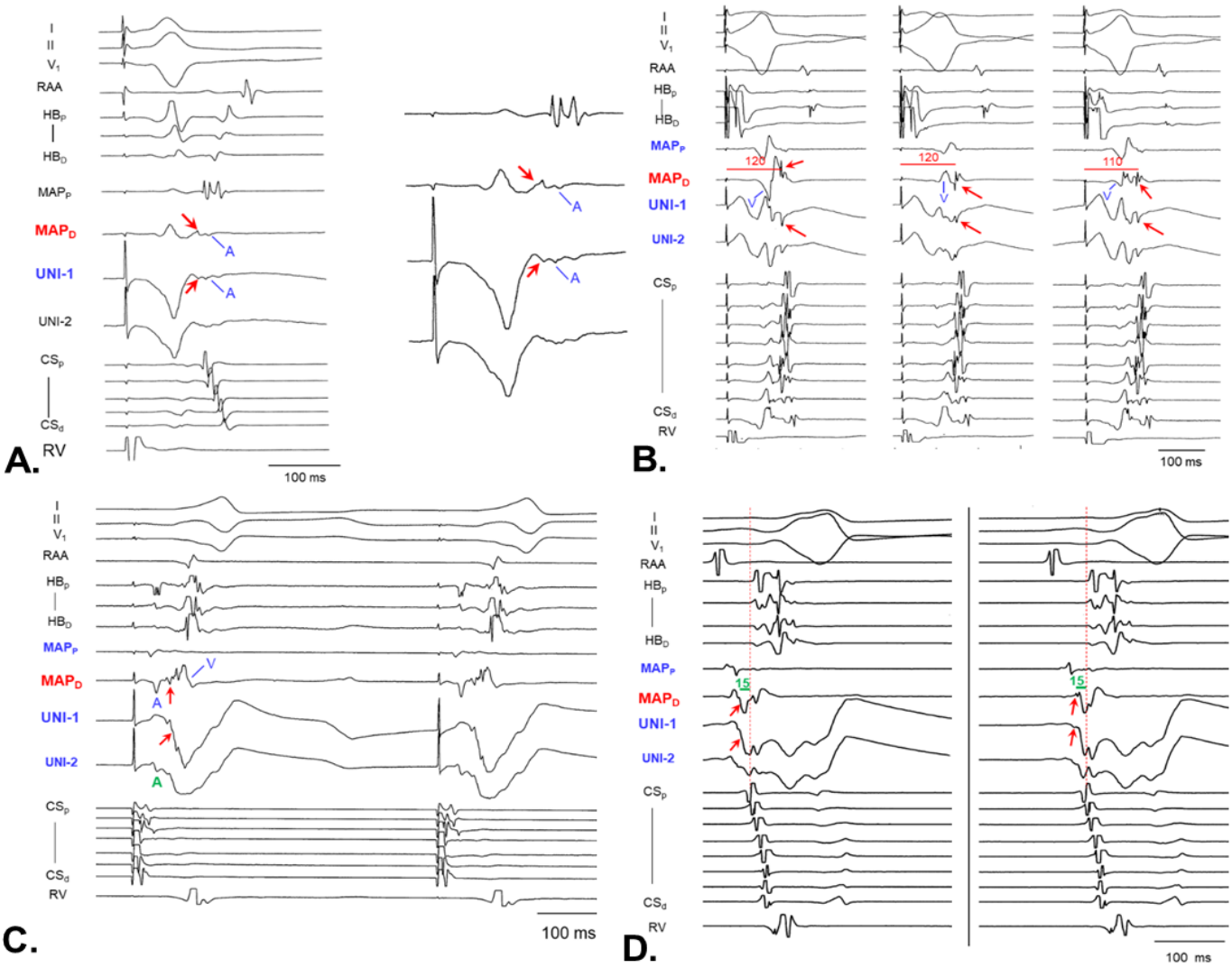


Figure 5.16. AV balance at successful ablation sites. A. For a left free wall AP, the target site should have a very small atrial EGM (blue arrow) and a much larger ventricular EGM to ensure that the ablation site is on the mitral annulus. In this example, the ventricular potential is relative small and rounded, suggesting suboptimal electrode-tissue contact. The ideal target is a site recording an AP-P. However, the AP-P recorded on the mapping catheter appeared to be far-field (red arrow). **B.** In another patient, the ablation catheter was positioned on the mitral annulus, targeting a sharp AP-P (red arrow). From the left to the right panel, the ablation catheter was sliding from the ventricular side of the annulus to the atrial side of the annulus due to respiration. This observation is very common when trans-septal approach is used because the catheter is laid on top of the annulus. Note that the AP-P was recorded in all the three panels (stimulus to AP-P interval=110-120 ms). **C.** In a patient with a right free wall AP, Dr. Jackman prefers a target recording a sharp AP-P along with the ventricular EGM moderately larger than the atrial EGM. In this example, the A:V ratio is approximately 1. **D.** In a patient with a right anterior AP, ablation catheter recorded an AP-P (red arrow), 15 ms before the earliest far-field ventricular potential (vertical red line) recorded on the HB catheter (**left panel**). When the tip of the ablation catheter slipped into the RV with respiration (**right panel**), the AP-P was still visible.

A common problem occurs when an operator has made multiple atrial lesions a few millimeters away from the true annulus, which eventually form a line of block along the atrial side of the annulus. Retrograde AP conduction continues to propagate bi-directionally out of the “isthmus” created by linear ablation, leading to progressive changes in atrial activation sequence with ablation (**Figure 5.17)**. Mistakes like this one can easily be prevented by choosing a site with a small atrial EGM and a large ventricular EGM. Decades ago,

when surgical WPW ablation was still performed, a common cause of ablation failure is that the surgical lesion set was too atrial to avoid injury of the AV valve or coronary artery, leaving an isthmus between the lesion set and annulus (**Figure 5.17A**). Ablation should target both ends of the isthmus to block AP conduction to the atrium.

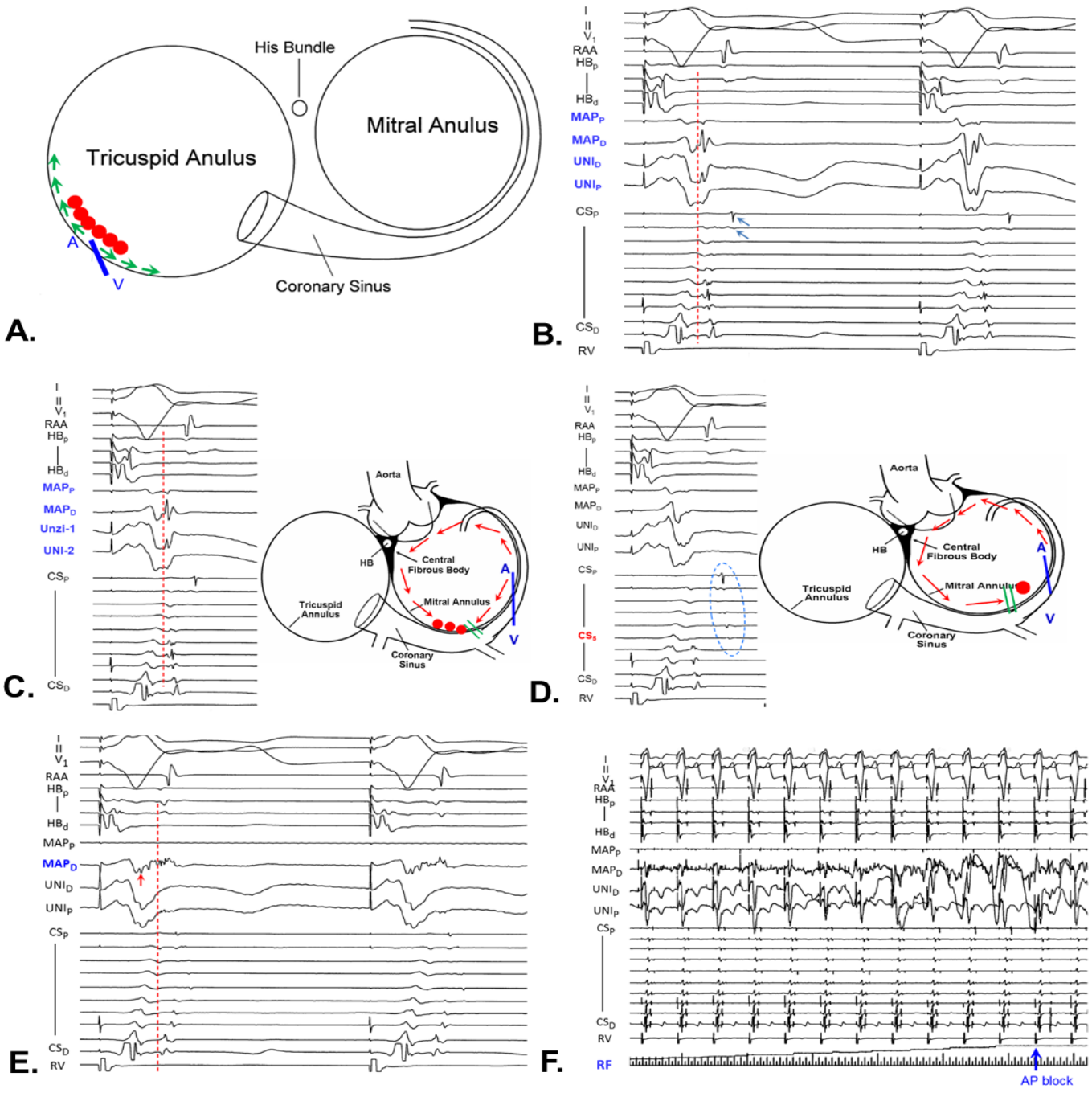


Figure 5.17. Annular isthmus created by prior ablation. A. A series of RF applications were delivered to target the earliest atrial activation. Ablation sites were not on the annulus thereby missing the atrial end of AP. Ablation only altered the atrial activation sequence without affecting the AP. The operator unknowingly made a linear lesion and created an isthmus between the annulus and ablation lesions. This type of ablation failure is often reported as “multiple accessory pathways” due to progressive changes of the activation sequence without AP conduction block. **B.** A patient with prior failed ablation due to “multiple left free wall APs”. Note the late potential recorded on the proximal CS electrodes (blue arrows). Mapping catheter was positioned at a site of early atrial activation. Note that the mapping catheter was sliding across the mitral annulus with cardiac cycle. The mapping catheter on the right panel recorded a small atrial but a large ventricular potential, indicating that

the tip electrode was on the mitral annulus. On the left panel, the mapping catheter recorded a large atrial potential, indicating that the tip electrode was in the left atrium, not on the annulus. **C.** Schematic explanation of wide double potentials in proximal CS due to prior ablation (red dots) that targeted the VA fusion sites. The ablation site was septal to the AP and created conduction block (double green line). The late potential recorded on CSp came from counterclockwise conduction of the atrial wave front (red arrows). **D.** The author was stupid enough to ablate the site of "earliest" atrial activation in the presence of VA fusion (the same site as the left panel in **B.** Note that double potentials now extended to CS-5. **Right panel.** Schematic explanation of the change in atrial activation sequence after an unsuccessful RF application. **E and F.** Ablation targeting an AP-P (red arrow) eliminated AP conduction. Note that the AP-P was significantly earlier than the site of "earliest" atrial activation (vertical dotted red line) recorded on CS-d.

If an AP-P cannot be found (e.g. an epicardial AP), ablation may target the earliest atrial or ventricular activation. For a right free wall AP, the earliest ventricular activation is usually 30 ms or more before the onset of the delta wave. For a left free wall AP, earliest ventricular activation15-20 ms before the onset of the delta wave is common. In theory, all APs are epicardial because they traverse the AV annulus on the epicardial surface but they are close enough to the annulus that ablation on the endocardial surface of the annulus can eliminate them. The term "epicardial AP" is therefore reserved for APs that connect with the atrium and ventricle at sites distant from the annulus; ablation along the AV annulus cannot eliminate them. Typical examples are atrial appendage-ventricular APs and APs involving CS-ventricular connections. Dr. Jackman defines an epicardial AP as an AP where the site of earliest atrial activation and ventricular activation is >1cm away from the AV annulus. In the presence of a right-sided epicardial AP, if one rotates the HALO catheter from the tricuspid annulus toward the atrium, where a large atrial EGM and a very small or no ventricular EGM is recorded, atrial activation there is earlier than that along the annular site. To ablate an epicardial AP, Dr. Jackman targets the site with the earliest local atrial or ventricular activation, judged by the maximal dV/dt (the steepest slope of the unfiltered unipolar EGM that has a QS shape) (**Figure 5.18).** Operators should be aware of the possibility that a very slow r-wave in the unipolar EGM (<1 Hz) may be filtered out by the high-pass filter; the QS pattern on the unipolar EGM is not a true QS pattern. Operators may find multiple adjacent sites showing the same early bipolar and unipolar EGM and a fake "QS" unipolar EGM. Ideally, the high pass filter should be set at 0.1 or 0.2 Hz to avoid this problem. The true site of earliest activation is the site where the local EGM fulfill the following 3 criteria: (1) activation timing is earlier than any near-field or far-field potential of any bipolar or unipolar recordings (2) the bipolar EGM and distal unipolar EGM of the ablation catheter begin simultaneously and (3) the distal unipolar EGM of the ablation catheter begins with a steeply negative component (QS or qs pattern).

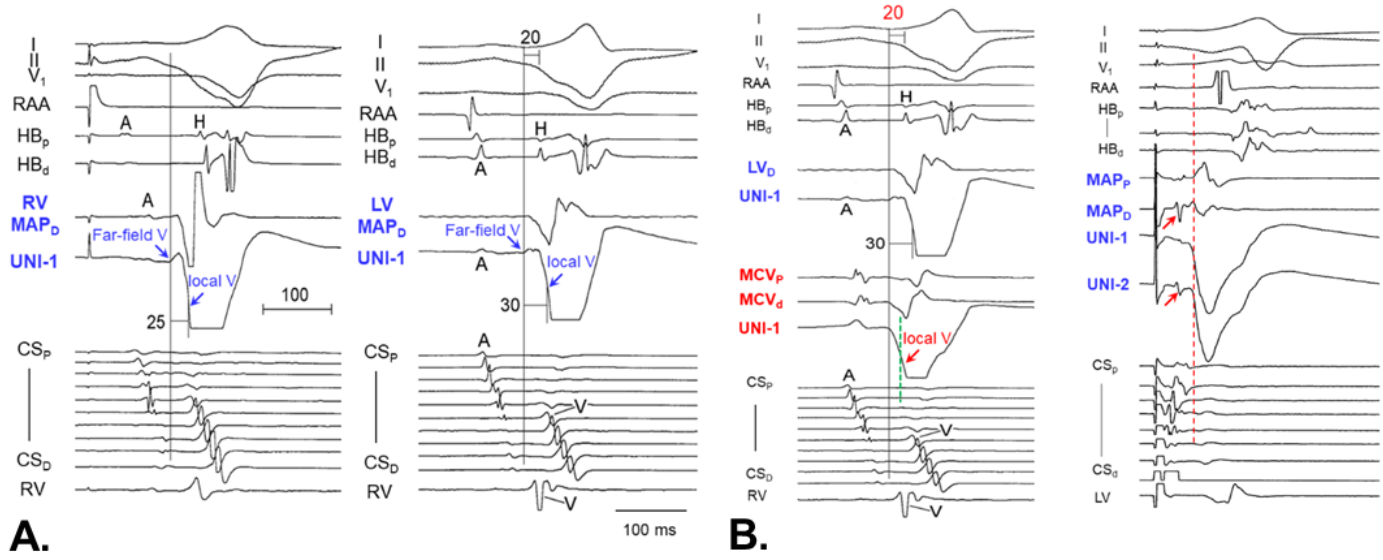


Fig 5.18. An epicardial AP with middle cardiac vein-LV connections. A. Mapping from the endocardium of posteroseptal RV (left panel) and posteroseptal LV (right panel) where earliest endocardial ventricular activation was found. Note that the local ventricular activation, measured by the largest dV/dt, was 25 ms (RV) and 30 ms (LV) later than the site recording earliest far-field ventricular potential (vertical line). These observations indicate that the site of earliest ventricular

activation was not located along the AV annulus, therefore fitting the definition of an epicardial AP. **B. Left panel:** The mapping catheter was positioned in the middle cardiac vein (MCV). The local ventricular potential was only 20 ms later than the earliest far-field potential. **Right panel:** In another patient with a similar AP, the ablation catheter was positioned in MCV and recorded an AP-P (red arrows). Note that the local ventricular activation (vertical red line) exhibited a QS pattern but was significantly later than the AP-P. *Modified with permission from: Sun Y et al. Circulation. 2002 Sep 10;106(11):1362-7*

Ablation during AVRT

If retrograde conduction during RV pacing shows fusion, operators must discern which EGM represent AP conduction. This is less of a problem for a left free wall AP. As long as the CS catheter records AP conduction, operators can map and ablate the AP in the presence of "contamination" from AVN conduction. Fusion can lead to great confusion in septal AP ablation. Operators may try pacing sites closer to the ventricular end of the AP to minimize AVN conduction. Pacing faster than the retrograde AVN block CL may eliminate retrograde AVN conduction but may lead to poor electrode-tissue contact due to short pacing CL. It is advisable to periodically induce AVRT or perform parahisian pacing to ensure that the operator is not unknowingly mapping retrograde AVN conduction.

Sometimes, ablation has to be performed during tachycardia due to incessant AVRT. When AVRT is terminated by ablation, it often induces a pause followed by vigorous contraction of the ventricle. Catheter is often thrown out of the target. In this scenario, Dr. Jackman would attempt to entrain the AVRT at CL 10-15 ms faster than the AVRT. As soon as the AVRT is entrained, RF application is started. If retrograde AP conduction blocks, pacing CL is quickly lengthened to ensure catheter stability. If VA block occurs and catheter stability is in question, pacing CL is quickly lengthened toward the sinus CL in order to allow a smoother transition between RV pacing and sinus rhythm to maintain stable electrode-tissue contact (**Figure 5.19**).

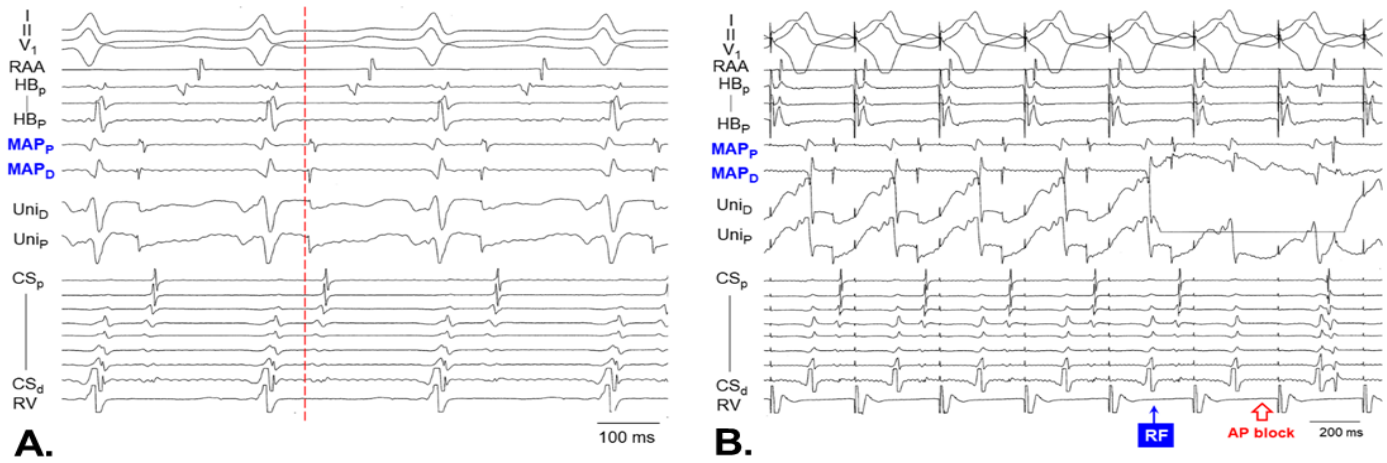


Figure 5.19. Ablating incessant AVRT. A. Incessant AVRT used a left lateral AP for retrograde conduction. RV pacing for mapping and ablating this AP could not be accomplished due to incessant AVRT. **B.** Entrain AVRT from RV pacing at a cycle length only 10 ms faster than AVRT. As soon as AVRT was entrained, ablation was started, which immediately eliminated AP conduction.

4. Unusual Location

In the 1980s, it was thought that APs cannot exit in the aorto-mitral continuity as it is an area with only fibrous valvular tissue without myocardium. There have been many case series of successful ablation of APs located in the aorto-mitral continuity, coronary cusps and left anteroseptal area below the coronary cusp. The author's practice is not to ablate an anteroseptal or midseptal AP until the coronary cusps, left anteroseptal and left midseptal areas have been mapped (**Figure 5.20**). This is of particular importance for the anteroseptal

AP whose ventricular end is in close proximity to the HB potential and a sharp AP-P cannot be recorded along the tricuspid annulus. As discussed before, the ventricular end of most of the anteroseptal AP is located more lateral than the atrial end; ablating the ventricular end is therefore preferred. If the ventricular end appears to be located on the septum or an AP-P cannot not be found along the tricuspid annulus, it should alarm the operator to search the atrial end of the AP in the coronary cusp because ablation in the noncoronary or right coronary cusp carries a significantly lower risk of AV block than ablation in the right anteroseptal area. If the site of earliest atria activation of an "anteroseptal" AP is behind the tendon of Todaro, in other words, not along the tricuspid annulus, it should prompt the operator to map the noncoronary cusp, left mid-septum and left anterior septum before attempting high-risk ablation of an anteroseptal AP (**Figure 5.20B**). Left anteroseptal and midseptal pathways located at the aorto-mitral continuity will be discussed in **Chapter 6**. Some APs ablated in unusual locations are illustrated in **Figure 5.21.**

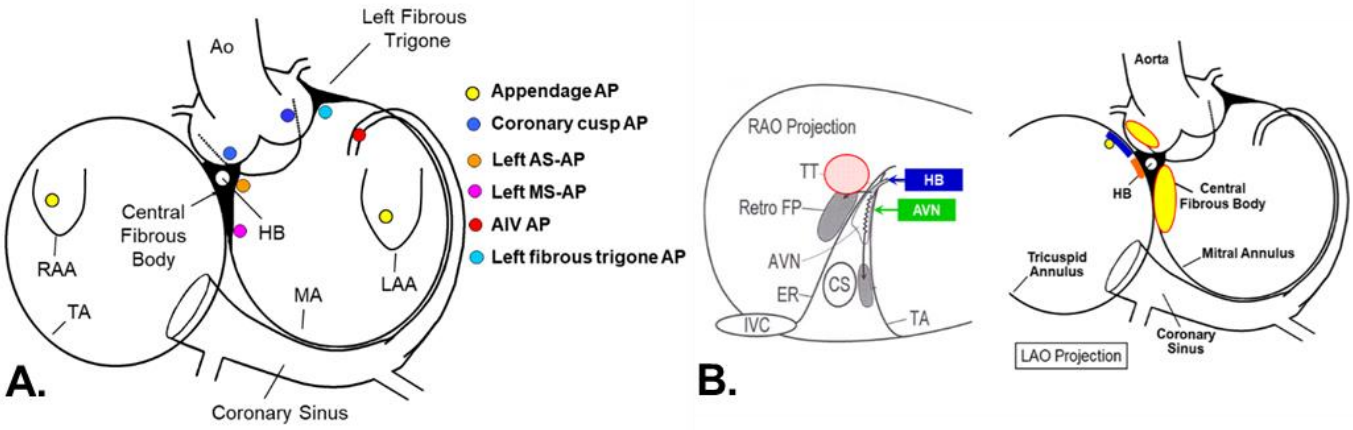
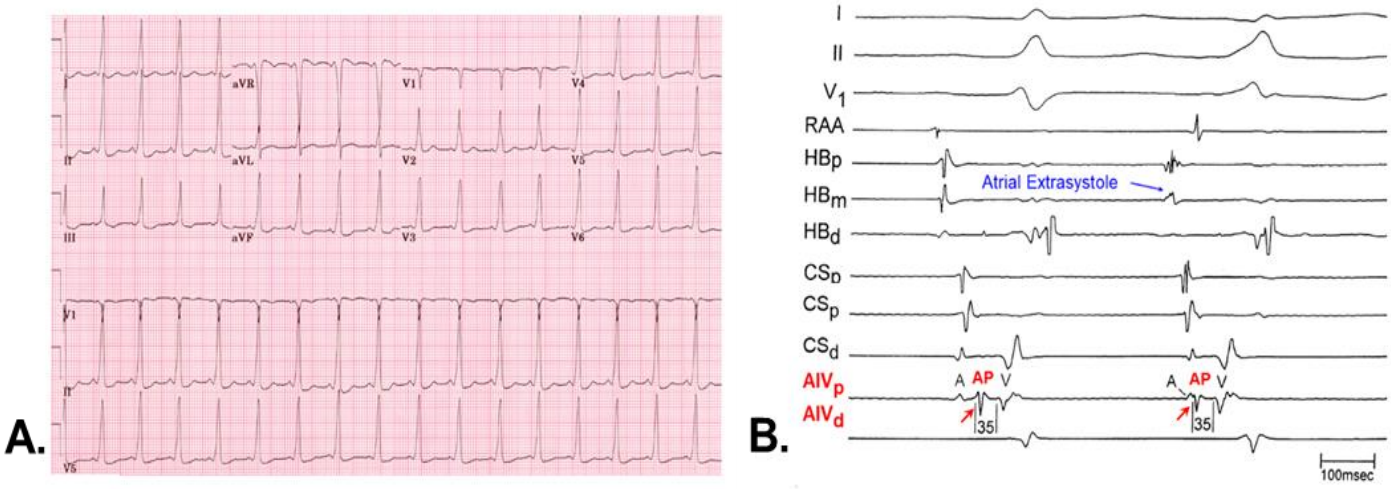


Figure 5.20. A. APs in unusual locations. **B.** If the site of earliest atrial activation is behind the tendon of Todaro (pink area in the left panel), it is advisable to search the AP in the coronary cusp, left mid-septum and left anterior septum (yellow areas in the right panel).



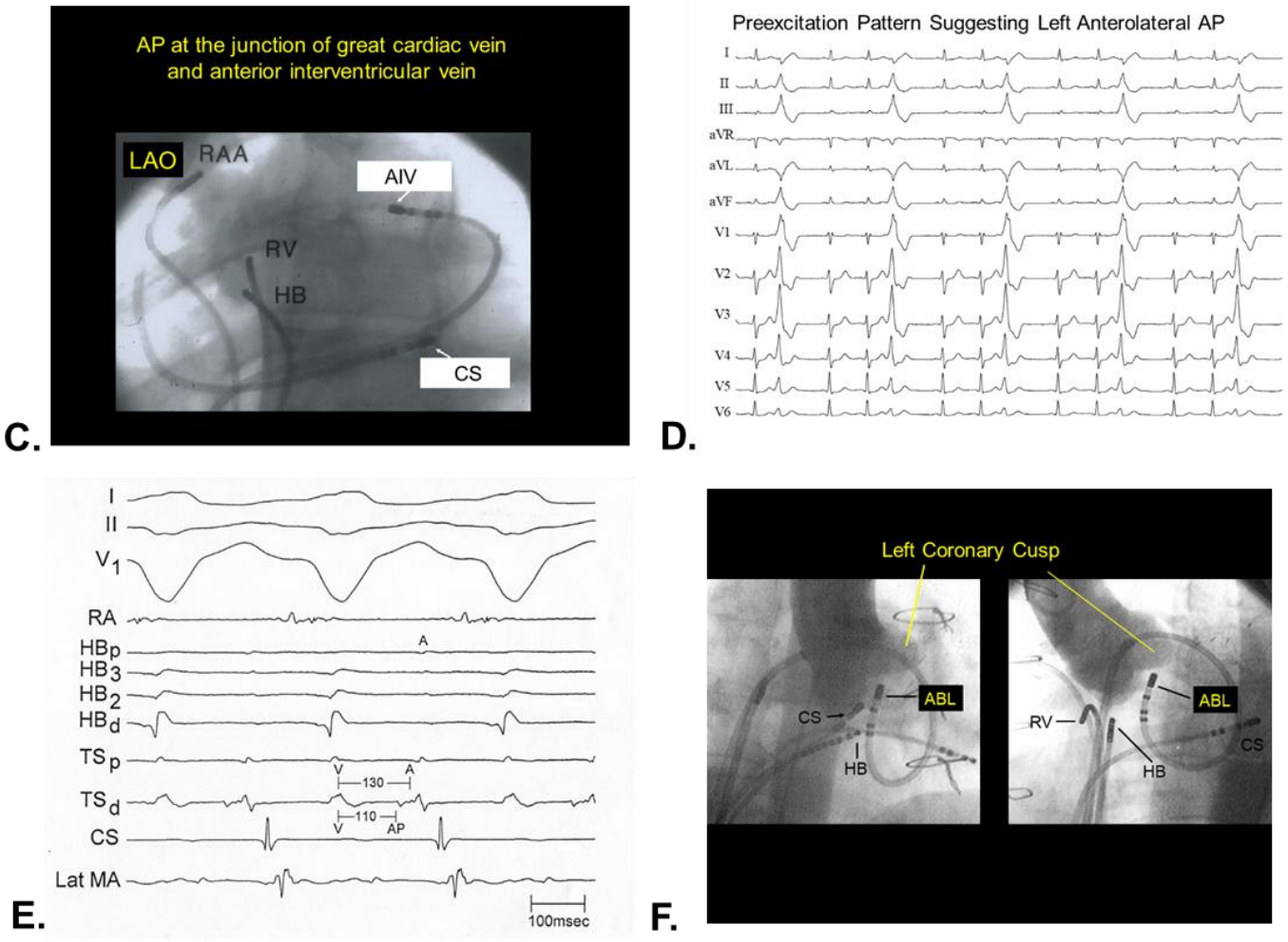


Figure 5.21. APs ablated in unusual locations. A. The preexcitation pattern predicts a right anteroseptal location. **B.** When the ablation catheter was positioned at the junction of the great cardiac vein and anterior inter-ventricular vein (AIV), a sharp potential (red arrow) was recorded. An atrial premature beat led to dissociation of this potential from the local atrial potential but the interval between the potential and local ventricular potential remained unchanged, indicating that this potential was not part of the local atrial potential but was connected to the ventricle. **C.** Radiographs of the site where ablation eliminated AP conduction. **D.** In another patient, the preexcitation pattern predicts a left anterolateral location. **E and F.** An AP-P was recorded from the left fibrous trigone below the left coronary cusp. AP conduction was eliminated by ablation at that site.

Appendage-ventricle APs

As already discussed, Dr. Jackman defines an epicardial AP as an AP in which the site of earliest atrial activation and ventricular activation, based on the steepest slope of the unipolar EGM, are located at sites >1 cm away from the annulus. Based on the limited surgical experience of the OU-EP group, the appendage-ventricle APs tend to be formed by a large muscle bundle, making catheter ablation difficult. These APs usually exhibit both antegrade and retrograde conduction. A typical history of patients with an RAA-RV AP is that ECG shows profound pre-excitation of a right anterior or anterolateral AP. Despite extensive ablation along the tricuspid annulus, no transient AP block was ever observed. For an ordinary left free wall AP, ECGs usually exhibit minimal preexcitation in the baseline state. Profound pre-excitation of a left anterior or anterolateral AP should alarm the operator that this AP may be an LAA-LV AP or the antegrade AVN conduction is poor.

While making the diagnosis of an appendage-ventricle AP is not difficult (**Figure 5.22**), ablation of appendage-ventricle APs from the endocardial surface usually requires a large number of RF applications to cover a large area showing diffusely early activation. If the A-V connection is at the base of the appendage, it requires significantly more RF applications. Epicardial ablation using the Sosa technique may be successful if the appendage-ventricle connection can be reached by the ablation catheter. If the atrial end of the AP is located near the tip of the atrial appendage, epicardial or epicardial+endocardial ablation may have a better chance of success. The operator needs to be aware that proximal circumflex artery courses below the base of the left atrial appendage. Epicardial ablation there can lead to total occlusion of the proximal circumflex artery.

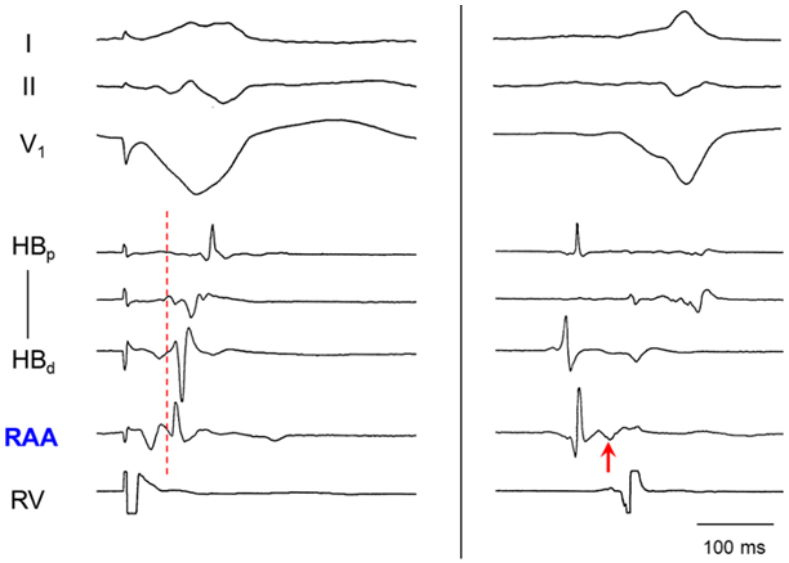


Figure 5.22. RA appendage-ventricle AP. Left panel. During retrograde AP conduction, the site of earliest atrial activation was in the RAA. **Right panel.** During antegrade AP conduction, a very early far-field ventricular potential (red arrow) was recorded in the RAA.

For an RAA-RV APs, the timing of the far-field RV potential in the RAA during sinus rhythm as well as the atrial potential within the RAA during RV pacing is very early (**Figure 5.22**). In the OU-EP laboratory, there has not been a single case of RAA-RV AP that was missed because the default position of the RA catheter is RAA, not RA free wall. For an LAA-LV AP, if retrograde atrial activation is early adjacent to the one o'clock position along the mitral annulus, the LAA should be mapped.

If the AV connection is at the tip of the appendage, thorascopic surgical excision of the appendage may be more successful than catheter ablation. If the AV connection is at the base of the appendage, the success of surgical ablation depends on the completeness of appendage excision. If the patient undergoes surgical ablation, it is important for an electrophysiologist to map the location of the site of AV connection before the surgeon attempts to dissect any tissue near the appendage. There was a case from the OU-EP laboratory years ago that the surgeon "cleared" the field and lost preexcitation before the arrival of the electrophysiologist. Since there was no AV conduction to map, the appendage was excised but pre-excitation recurred in a few days.

Accessory Pathways formed by the His-Purkinje System

Three types of APs are formed by the His-Purkinje system: fascicular-ventricular AP, atrio-fascicular AP (Mahaim) and nodo-ventricular/nodo-fascicular AP. The term "Mahaim AVRT" may indicate atrio-fascicular AVRT or nodo-fascicular/nodo-ventricular AVRT, creating great confusion among electrophysiologists. In this book, Mahaim AVRT indicates antidromic AVRT using an atrio-fascicular AP for antegrade conduction.

1. Fascicular-ventricular AP (FV-AP)

FV-APs can be considered as a form of "insulation breach" of the distal HB or proximal bundle branch, leading to preexcitation of a small mass of the ventricular myocardium near the base of the RV or LV. Because fascicular-ventricular APs do not participate in AVRT, they are considered as "cosmetic preexcitation" and ablation is not indicated. The OU-EP group had received referrals for anteroseptal AP ablation, which turned out to be FV-APs. Since preexcitation predicts an anteroseptal AP, mapping and ablation was done only during antegrade AP conduction under the erroneous assumption that the earliest

ventricular activation in the anteroseptal area is caused by an anteroseptal AP. Some electrophysiologists did not attempt to induce AVRT but attempt to ablate this harmless AP under the wrong assumption that this is an anteroseptal AP participating in AVRT. The clinical arrhythmia is indeed AT, AVNRT for AVRT using a concealed AP for retrograde conduction. FV-AP APs have the following unique features (**Figure 5.23A**): (1) the site of earliest ventricular activation is in the anterior septum or mid-septum, leading to the wrong diagnosis of anteroseptal or mid-septal APs, (2) in sharp contrast to an ordinary atrio-ventricular AP, the AH interval becomes progressively longer but the HV interval of a FV-AP does not shorten during decremental atrial pacing, and (3) the magnitude of preexcitation remains unchanged at different heart rates. These electrophysiological features can easily be explained by the observation that FV-AP is essentially a form of "insulation breach" of the distal HB or proximal bundle branch. Atrial electrical impulse has to pass the entire AV node before reaching the AP. From this point, normal His-Purkinje conduction and AP conduction propagate in parallel. While a small area of the RV or LV in the basal anterior septum or mid-septum is preexcited by the fascicular-ventricular AP, the rest of the ventricle is activated by normal His-Purkinje conduction, leading to fixed and minimal preexcitation at different heart rates. If the HB potential falls behind the local ventricular potential, this AP cannot be an F-V AP. **Figure 5.23B** illustrates the electrophysiological properties of an ordinary atrio-ventricular AP. With shorter pacing CL, the AV interval shortens, HV interval gradually becomes a negative value and the magnitude of preexcitation becomes greater.

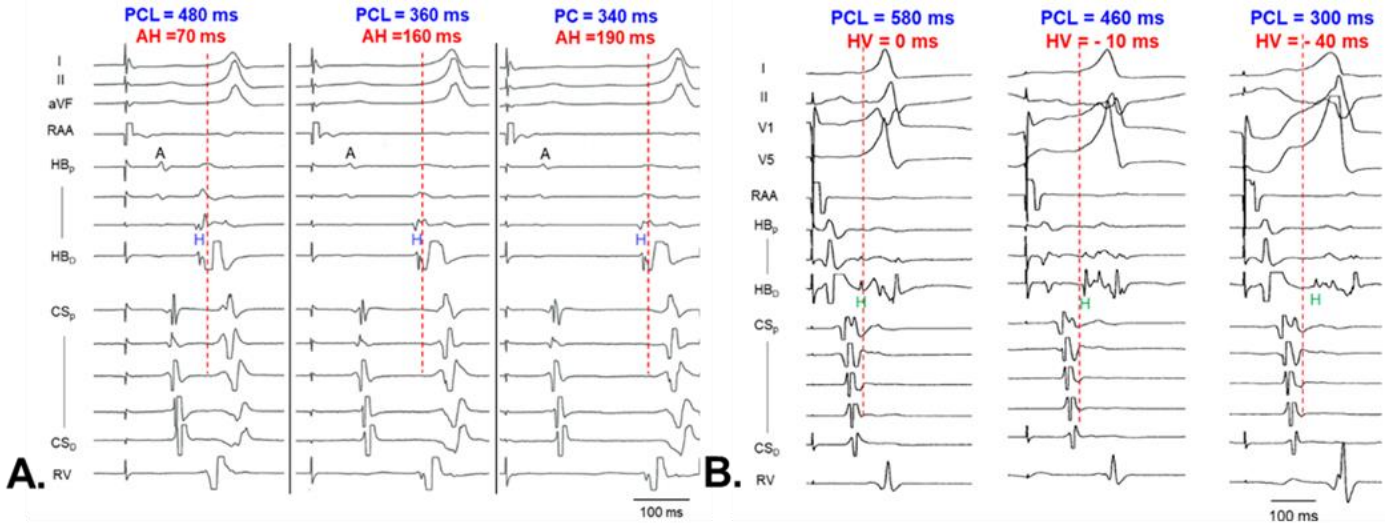


Figure 5.23. A. F-V AP. With progressively shorter pacing cycle length (PCL), the AH interval lengthened gradually but the HV interval and the magnitude of preexcitation remained unchanged. Vertical red line: onset of preexcitation on ECG. **B. An ordinary atrio-ventricular AP.** With progressively shorter PCL, the AV interval shortened, the HV interval shortened and the magnitude of preexcitation increased.

2. Atrio-fascicular (Mahaim) Pathway

Atrio-fascicular (Mahaim) pathway is a unique form of APs. Histologically, Mahaim AP is formed by a remnant accessory AVN located along the tricuspid annulus, usually between the 7:00 and 10:00 o'clock position (**Figure 5.24A**). The accessory HB, originating from the accessory AV node, extends toward the apical RV free wall and eventually connect to peripheral RBB. The His-Purkinje origin of Mahaim AP leads to the following unique EP properties.

a) Similar to the AVN, Mahaim APs exhibit decremental antegrade conduction properties. For an ordinary AP, the extent of preexcitation during decremental atrial pacing is determined by the competition between antegrade AP and antegrade AVN conduction. As the atrial pacing CL shortens, AVN progressively loses the competition due to its decremental conduction property, leading to progressively more profound preexcitation and shorter stimulation-QRS interval (**Figure**

5.23B). Contrarily, the extent of preexcitation of a Mahaim AP is determined by two pathways, both of which exhibit antegrade decremental conduction properties. During decremental atrial pacing, progressive preexcitation is observed; however, the stimulation-QRS interval prolongs progressively as well; similar to a typical AVN response (**Figure 5.24B-C**). If adenosine is administered, both antegrade AVN and AP conduction will be blocked (**Figure 5.24D**), in sharp contrast to an ordinary AP which is usually not sensitive to adenosine.

b) In the vast majority of patients with Mahaim AVRT, the reentrant circuit is formed by the atrio-fascicular AP for antegrade conduction; the retrograde limb of the reentrant circuit is formed by the RBB and AVN (mostly the retrograde fast pathway) (**Figure 5.25A**). The HV interval is typically -20 to -30 ms (or VH interval of 20-30 ms) due to fast His-Purkinje conduction time. If RBBB occurs during preexcited AVRT, retrograde conduction requires trans-septal conduction to engage the LBB. The HV interval will be more negative (or longer VH interval) and AVRT CL will be significantly longer (**Figure 5.25B**). In some patients, another AP is used for retrograde conduction, essentially an AP-to-AP AVRT.

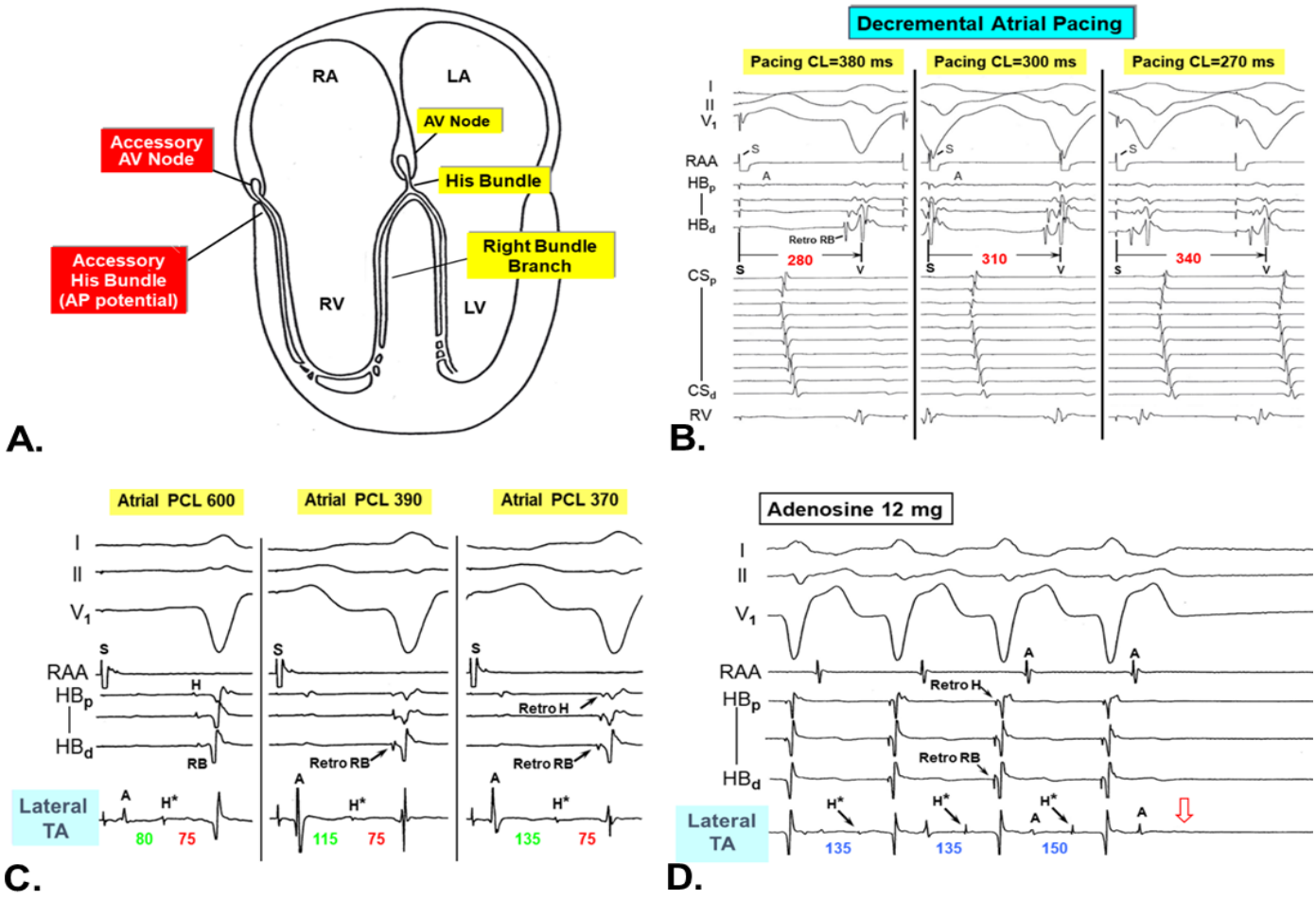


Figure 5.24. Atrio-fascicular AV pathway (Mahaim). A. A Mahaim AP pathway is formed by a remnant accessory AVN that connects with the peripheral RBB system through an accessory His bundle. The HB-like potential is the AP-P of an atrio-fascicular AP. **B.** During decremental atrial pacing, the HV interval became progressively shorter but the AV interval became progressively longer. **C**. An accessory HB potential (H*) was recorded from the tricuspid annulus. Similar to decremental conduction of the AVN, the AH*' interval (or the A-to-APP interval) became progressively longer with decremental atrial pacing. Note that the interval between this accessory HB potential and local ventricular activation remained unchanged at 75 ms (without decremental conduction property between the accessory HB and ventricle). **D.** Adenosine led to both AVN and AP conduction block and disappearance of the H* potential (empty red arrow). Courtesy of Dr. Jackman.

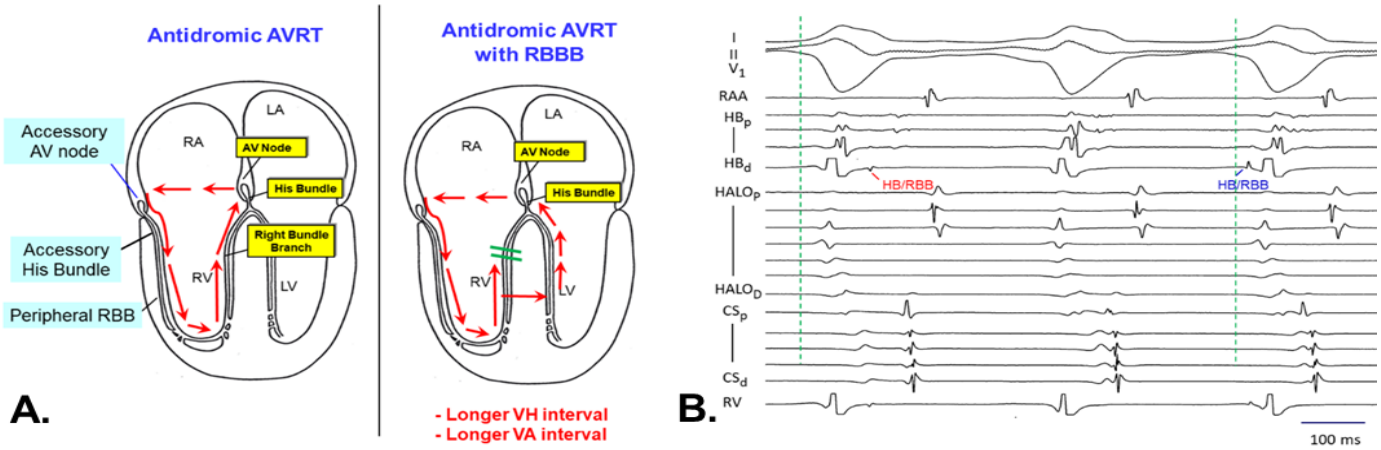


Figure 5.25. Reentrant circuit of antidromic AVRT mediated by an atrio-fascicular AP. A. Left panel: The reentrant circuit is formed by the atrio-fascicular AP for antegrade conduction; the retrograde limb of the reentrant circuit is formed by the RBB and AVN (mostly the retrograde fast pathway). **Right panel:** In the presence of RBBB during preexcited AVRT, retrograde conduction requires trans-septal conduction and then engages the LBB. The HV interval is more negative (or longer VH interval) and the tachycardia CL is significantly longer. **B.** In antidromic AVRT, transient RBBB occurred (left beat). With RBBB, the HB/RBB potential was significantly later than the onset of the QRS complex (vertical green line). After RBBB disappeared (right beat), the VH interval was again 25 ms.

Diagnosis of Mahaim AVRT is not difficult. The OU-EP group had received multiple referrals for failed ablation due to misdiagnosis of Mahaim AVRT as AVNRT with LBBB aberrancy. The first step to make the correct diagnosis is to examine the 12-lead ECG. It has a long AV interval and typical LBBB pattern because of early engagement of the peripheral RBB, leading to a typical LBBB morphology (**Figure 5.26**). These two features distinguish Mahaim AVRT from other forms of preexcited AVRT. The HV interval (-20 to -30 ms) is also a great discriminator. Wide-complex tachycardia with a typically LBBB morphology has 4 major differential diagnoses: SVT with LBBB aberrancy, bundle-branch reentrant VT, preexcited nodo-fascicular tachycardia and Mahaim AVRT. In both SVT with LBBB aberrancy and bundle-branch reentrant VT, the HV interval during tachycardia is equal to or longer than that in sinus rhythm. Preexcited nodo-fascicular AVRT and Mahaim AVRT both have a negative value of the HV interval. These two forms of preexcited AVRTs are indeed very similar (see discussion below).

Like any wide-complex tachycardia, the most important pacing maneuver is to deliver single atrial extra-stimuli (AES) during tachycardia and examine how AES conduct to the ventricle to reset the tachycardia (**Figure 5.26**). After the mechanism of antegrade AV conduction is verified, single ventricular extra-stimuli (VES) were delivered to a site adjacent to the site of earliest atrial activation to determine if the retrograde conduction is mediated by AVN or another AP.

The target of the Mahaim AP ablation is the accessory HB potential (AP-P) along the tricuspid annulus, not the site of earliest ventricular activation. Because the “ventricular” end of the Mahaim AP connects to the peripheral RBB, the ventricular activation along the tricuspid annulus is usually very late (**Figure 5.26F**). An atrio-fascicular AP is very superficial and notorious for mechanical trauma. Dr. Jackman often jokes; “I am an obsessive-compulsive person. However, there are two arrhythmias that perfection is the enemy of good-enough: Mahaim AP and fascicular VT.” With that being said, if the ablation catheter lands on a small, rounded AP, which fits Dr. Jackman’s definition of a far-field AP-P, Dr. Jackman will start ablation immediately. To minimize the incidence of “bumping” the Mahaim AP, Dr. Jackman prefers to lay the ablation catheter in parallel with the plane of the tricuspid annulus and inferior-septal to the site of the presumed accessory HB (AP-P) location. Similar to the maneuver described in **Figure 5.15**, as the operator slowly pulls back the ablation catheter toward the target, ablation is started immediately if the accessory HB potential shows up on the distal unipolar EGM. An SR1, SR2 or SR3 may help stabilize the electrode-tissue contact, depending on the location of the target along the tricuspid annulus. If AP conduction was bumped before ablation, Dr. Jackman will wait for a few minutes at the same site and start ablation if AP conduction remains blocked.

Fortunately, there has been no recurrence in any patients that Dr. Jackman was forced to adopt this “bump then ablate” strategy.

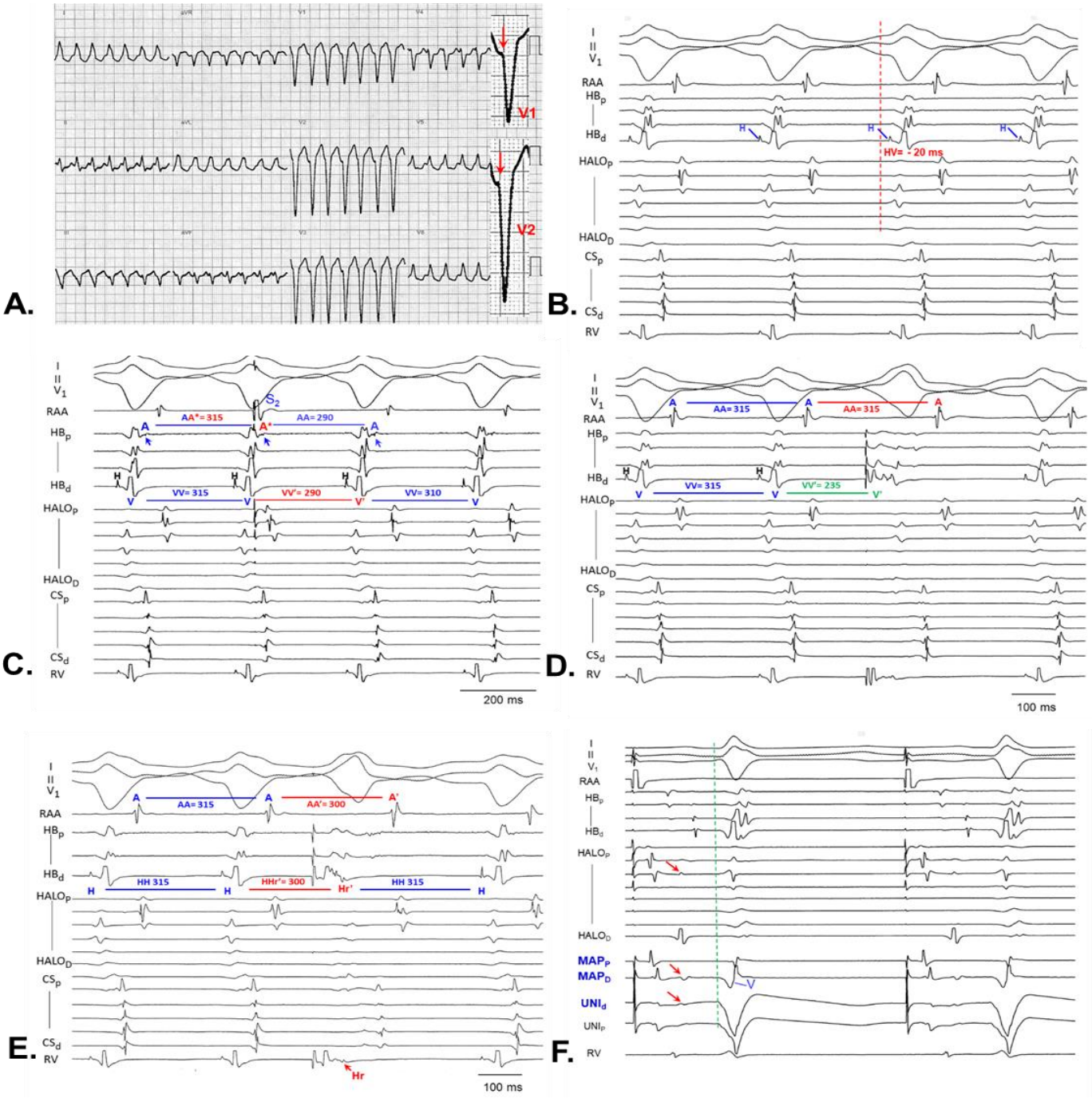


Figure 5.26. Antidromic AVRT mediated by an atrio-fascicular AP. ECG showed typical LBBB with a small, sharp r wave in the beginning of the QRS complex in lead V1 and V2. **B.** A wide-complex tachycardia with 1:1 AV relationship and an HV interval of -20 ms, strongly supports the diagnosis of Mahaim AVRT. **C.** In AVRT (CL=315 ms), an atrial extra-stimulus advanced the next ventricular activation by 25 ms (V-V’ interval = 290 ms) and reset the tachycardia. Note that the atrial timing in the HB region (blue arrows) was not affected by the atrial extra-stimulus (A-A* = 315 ms). This extra-stimulus therefore could not conduct to the ventricular through the AV node, verifying the diagnosis that this AP participated in the preexcited tachycardia. **D.** To evaluate the retrograde limb of the reentrant circuit, a series of single ventricular extra-stimuli were delivered to basal anteroseptal RV in close proximity to the site of earliest atrial activation. Single ventricular extra-stimuli advanced the local ventricular activation in the HB region by 80 ms without affecting the tachycardia, excluding the possibility that the retrograde limb of the reentrant circuit was an anteroseptal or mid-septal AP. **E.** An earlier ventricular extra-stimulus advanced the retrograde HB potential by 15 ms (HHr’ = 300 ms), which then advanced the next atrial activation and reset the tachycardia, proving that the retrograde limb of the reentrant circuit

was AVN fast pathway**. F**. An AP-P (accessory HB potential; red arrow) was recorded at the tricuspid annulus. Note that this AP-P was not sharp and local ventricular activation was very late (after the onset of the QRS complex, vertical green line).

Historical Vignette:

In Dr. Jackman's very first atrio-fascicular Mahaim AP ablation, before the reentrant circuit was elucidated, he targeted the site of earliest ventricular activation at the apical RV free wall. Ablation there only prolonged the VA interval and made AVRT incessant. However, necessity is the mother of invention. Dr. Jackman eventually figured out at that night that the primary target should be the accessory HB potential along the annulus, not the site of earliest ventricular conduction.

If the ventricular activation of a Mahaim AP is earlier along the tricuspid annulus than the onset of the preexcited QRS complex, this Mahaim AP may be an atrio-ventricular type of Mahaim in which the accessory HB connects directly to basal RV myocardium like an ordinary free wall AP. There are some cases that atrio-fascicular Mahaim APs also connect to ventricular myocardium adjacent to the tricuspid annulus. In these cases, an accessory HB potential (AP-P) can be recorded along the annulus but the ventricular activation along the annulus is also very early (**Figure 5.27**). In these cases, the primary ablation target is still the accessory HB potential along the annulus. Of note, the OU-EP group had received plenty of referrals for atrio-fascicular AP ablation in which the referring physician could not find the accessory HB potential. In nearly all these cases, an accessory HB potential was found and successfully ablated after careful mapping. That is to say, the accessory HB potential can be very small and look far-field. If the diagnosis is verified, careful mapping along the tricuspid annulus, particularly between the 7:00 and 10:00 o'clock position is advised. Atrio-ventricular Mahaim APs are rare. The operator may view them as APs that share the same EP properties with an atrio-fascicular Mahaim AP (decremental antegrade conduction, no retrograde conduction, preexcited AVRT). The ablation target of atrio-ventricular Mahaim APs is also an AP-P along the tricuspid annulus.

3. Nodo-fascicular AP (NF-AP) and nodo-ventricular AP (NV-AP)

NF-AP is probably the rarest type of AP. The world's leading expert on this, Dr. Melvin Scheinman, believes that many AVRTs using a concealed NF-AP for retrograde conduction were misdiagnosed as AVNRT but were successfully treated with standard slow pathway ablation because most of the NF-APs connect with the AVN through the slow pathway (mainly the right inferior extension). One can view a manifest NF-AP as a "septal" atrio-fascicular AP; its "atrial end" connects to the compact AVN or one of the extensions of the AVN, mainly the right inferior extension (**Figure 4.19**). With this in mind, the electrophysiological properties of NF-APs atrio-fascicular APs were very similar. During decremental atrial pacing, progressive preexcitation along with progressive prolongation of the stimulation-to-QRS interval can be observed in both manifest NF-AP and atrio-fascicular APs. During preexcited AVRT, the HV interval is a negative value for both types of AP as well. For nodo-ventricular APs, the VH interval may be longer (e.g. >50 ms) because the tachycardia does not use the peripheral Purkinje system for conduction.

AVRT using a concealed NF/NV AP for retrograde conduction is very difficult to diagnose. Because atrium is not part of the reentrant circuit, this type of AVRT may exhibit 1:1 VA conduction or VA dissociation. As mentioned in **Chapter 4**, AVRT using a concealed NF/NV AP for retrograde conduction needs to be differentiated from AVNRT with an upper common pathway, junctional tachycardia and high septal VT originating from a site adjacent to the HB area. As discussed earlier, NF/NV AP often connects to one of the slow pathways of the AVN, particularly the right inferior extension. The operator may unknowingly eliminate the NF/NF AP by slow pathway ablation. The most important pacing maneuver to diagnose orthodromic AVRT caused by a concealed NF/NV AP is to deliver VES during the HB refractory period which can reset or terminate this narrow-complex SVT similar to what VES would do to an ordinary AP.

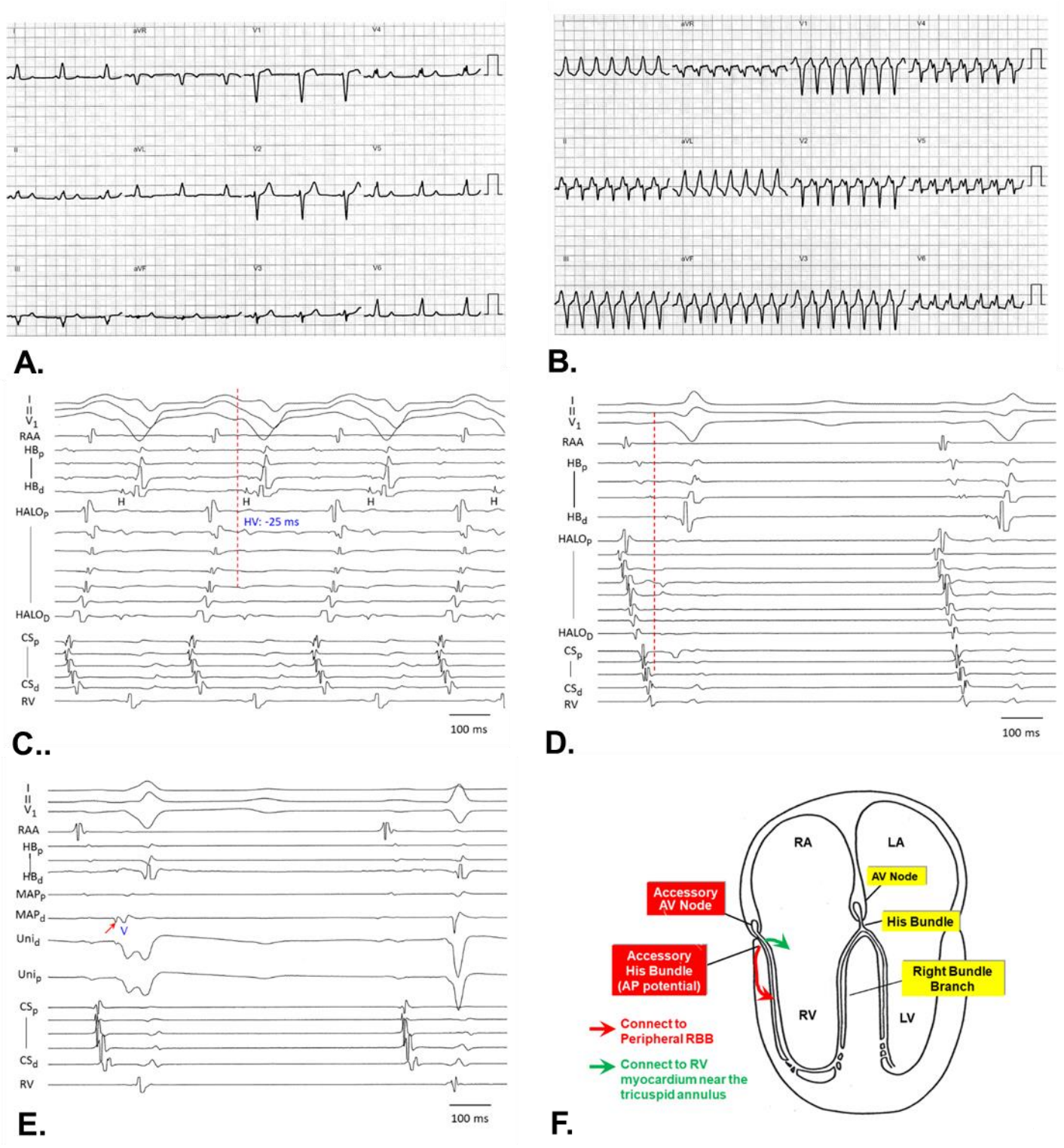


Figure 5.27. A variant of atrio-fascicular AP in a 10 y/o female. A. Baseline ECG showed minimal preexcitation. **B.** Preexcited AVRT with typical LBBB morphology. **C.** In AVRT, the VH interval was 25 ms. **D.** In sinus rhythm, the site of earliest ventricular activation (vertical red line) was recorded from tricuspid annulus, indicating that this atrio-fascicular AP also had an atrio-ventricular component. **E.** The successful ablation site recorded an AP-P (red arrow) and very early ventricular activation as well. **F.** Schematic diagram of atrio-fascicular AP that also have an atrio-ventricular component.

Chapter 6:

Ablation of Septal Accessory Pathways

Ablation of a septal accessory pathway (AP) faces four major challenges. First, an orthodromic AVRT using a septal AP for retrograde conduction can be confused with a focal septal atrial tachycardia (AT) or AVNRT (**Figure 6.1**). If the VA interval of a slow/fast AVNRT is longer than 70 ms, differentiating AVNRT from a septal AT or orthodromic AVRT can be a challenging task (**Figure 6.2**). Second, the risk of AV block is always a major concern. As the AV node is situated inferior to the HB, the risk of AVN injury of midseptal AP (MS-AP) ablation may be higher than that of anteroseptal AP (AS-AP) ablation. Cryoablation has been shown to reduce the risk of AV block; however, a larger ablation electrode has a substantially negative impact on the accuracy of mapping due to its large recording range and far-field signals. Also, extra stiffness of the cryoablation catheter easily causes mechanical trauma ("bump") of an AS-AP or MS-AP. Third, some of the APs may be located in unusual locations such as the aorto-mitral continuity (left anteroseptal area below the aortic valve), the left mid-septal area or the bottom of the coronary cusp. Fourth, posteroseptal APs (PS-APs) using CS or its tributary as part of the AP connections can be very difficult to map since the posteroseptal pyramidal space is the junction of five chambers (RA, RV, CS, LA and LV). Ablation in the CS venous system also carries a significant risk of coronary artery injury.

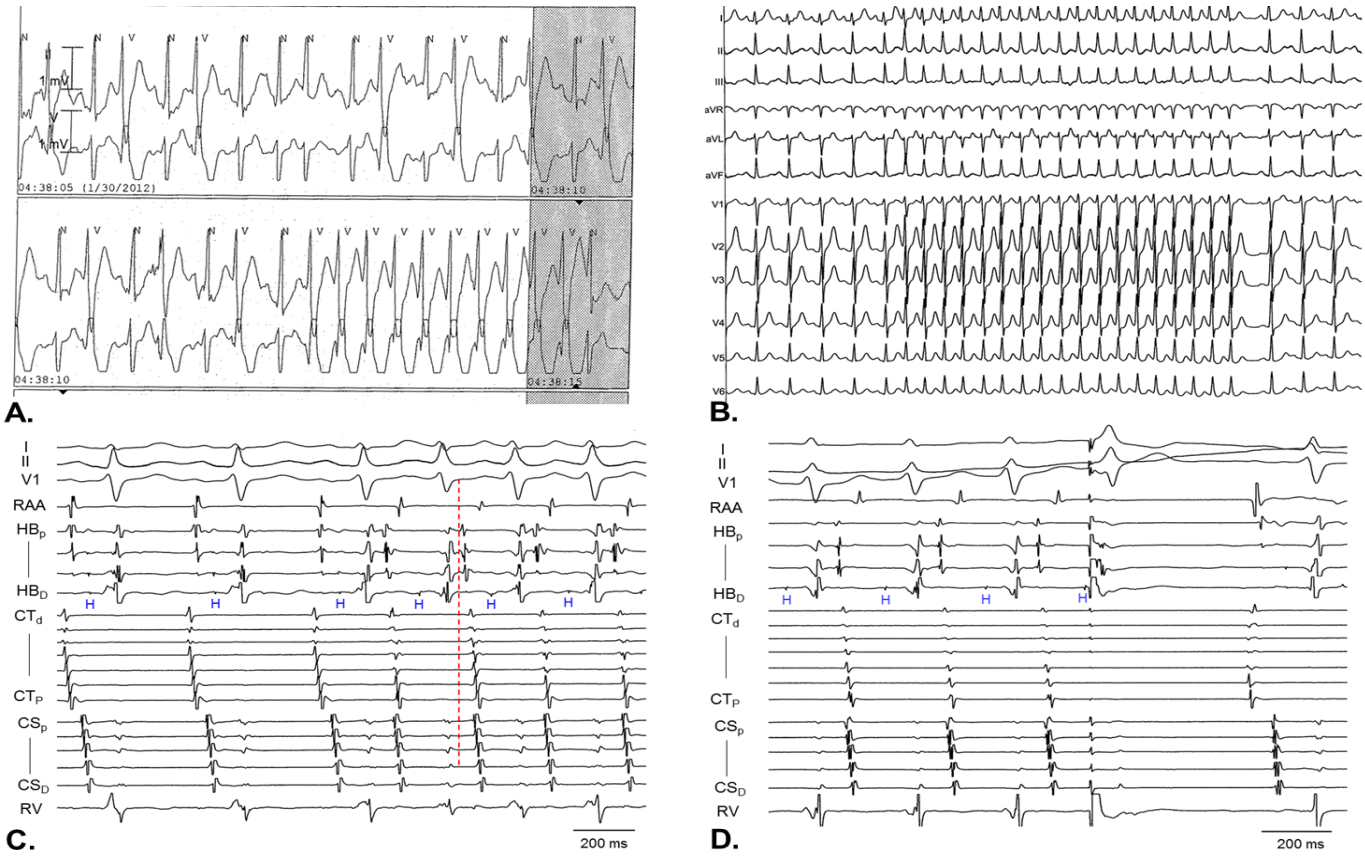


Figure 6.1. A young man with two failed ablations to treat septal atrial tachycardia. A. Recurrent episodes of nonsustained SVT were always initiated by prolonged "atrial bigeminy". **B.** PAC without PR interval prolongation repeatedly initiated nonsustained SVT. **C.** Intracardiac recording showed that the site recording earliest atrial activation (vertical red line) was in the HB region. **D.** PVC delivered during HB refractory period terminated SVT, proving the diagnosis of orthodromic AVRT using an AS-AP for retrograde conduction.

Ablation of a septal AP might be slightly easier in the presence of preexcitation. However, elimination of antegrade AP conduction may not have any impact on the clinical SVT. It is not uncommon that a manifest

AP does not have retrograde conduction at all. The clinical SVT is AVNRT or AT; this manifest AP does not participate in SVT at all. If parahisian pacing at progressively shorter cycle length (CL) fails to demonstrate retrograde AP conduction, it should alarm the operator that this manifest AP may not conduct in the retrograde direction and this AP may not be the cause of the clinical SVT (**Figure 6.2**). More aggressive induction of the clinical arrhythmia should be attempted to prove its mechanism. Therefore, it is not advisable to ablate a manifest AP without first verifying its role in SVT, particularly an AP adjacent to the AVN.

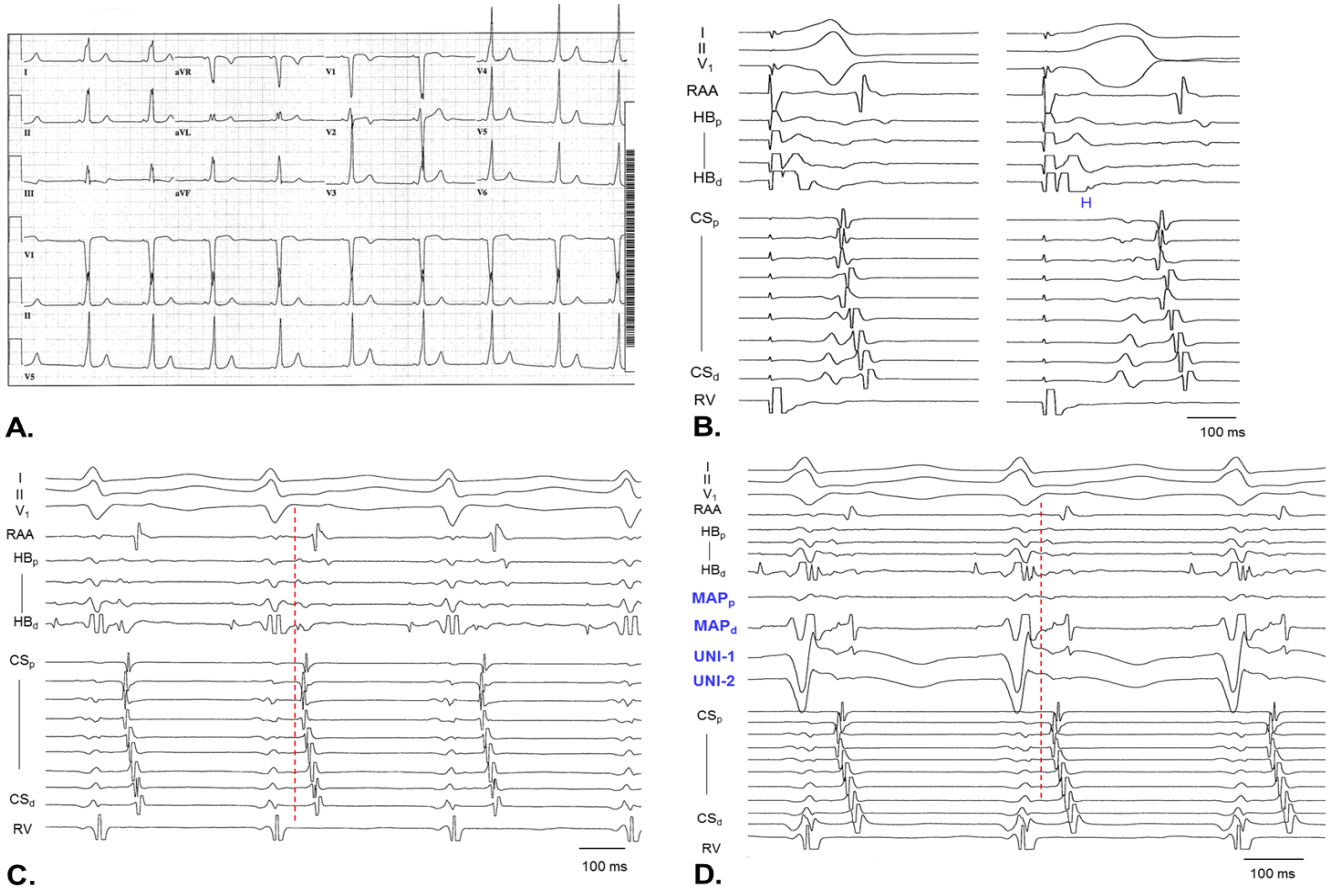


Figure 6-2. AVNRT in a young woman with WPW. A. Preexcitation pattern predicts a mid-septal AP. **B.** Parahisian pacing at multiple CL showed that retrograde conduction was mediated by AVN. **C**. Slow/fast AVNRT with a VA interval of 85 ms was induced. Because of a longer than usual VA interval, this slow/fast AVNRT can easily be misdiagnosed as orthodromic AVRT. Slow pathway was ablated and tachycardia was no longer inducible. Patient did well for several years. **D**. In another EP study 8 years later, orthodromic AVRT was eventually induced after many attempts. Note that the activation sequence was very similar between AVNRT and AVRT. This AS-AP was successfully ablated. Vertical red line: earliest atrial activation

In some cases, after successfully ablating the antegrade AS-AP conduction (loss of preexcitation), retrograde AP conduction persists and continues to make orthodromic AVRT (**Figure 6.3**). The site of earliest atrial activation remains in the anteroseptal area and it is difficult to differentiate retrograde fast pathway conduction from retrograde AS-AP conduction. In this scenario, parahisian pacing can provide the correct answer in most cases. If parahisian pacing still demonstrates retrograde AP conduction after elimination of the antegrade AP conduction, it is obvious that more mapping and ablation are needed.

Dr. Jackman's approach to septal AP ablation can be summarized as follows.

1. Use parahisian pacing to determine if retrograde conduction is mediated by AP, AVN or AP/AVN fusion. This is particularly important for mapping an AS-AP or MS-AP. If the operator attempts to ablate a manifest septal AP during RV pacing but is not aware that retrograde conduction at that pacing CL is mediated by both AVN and AP, ablation during ventricular pacing may end up targeting the AVN, not AP. This problem often occurs if isoproterenol has been administered because isoproterenol enhances retrograde AVN conduction.

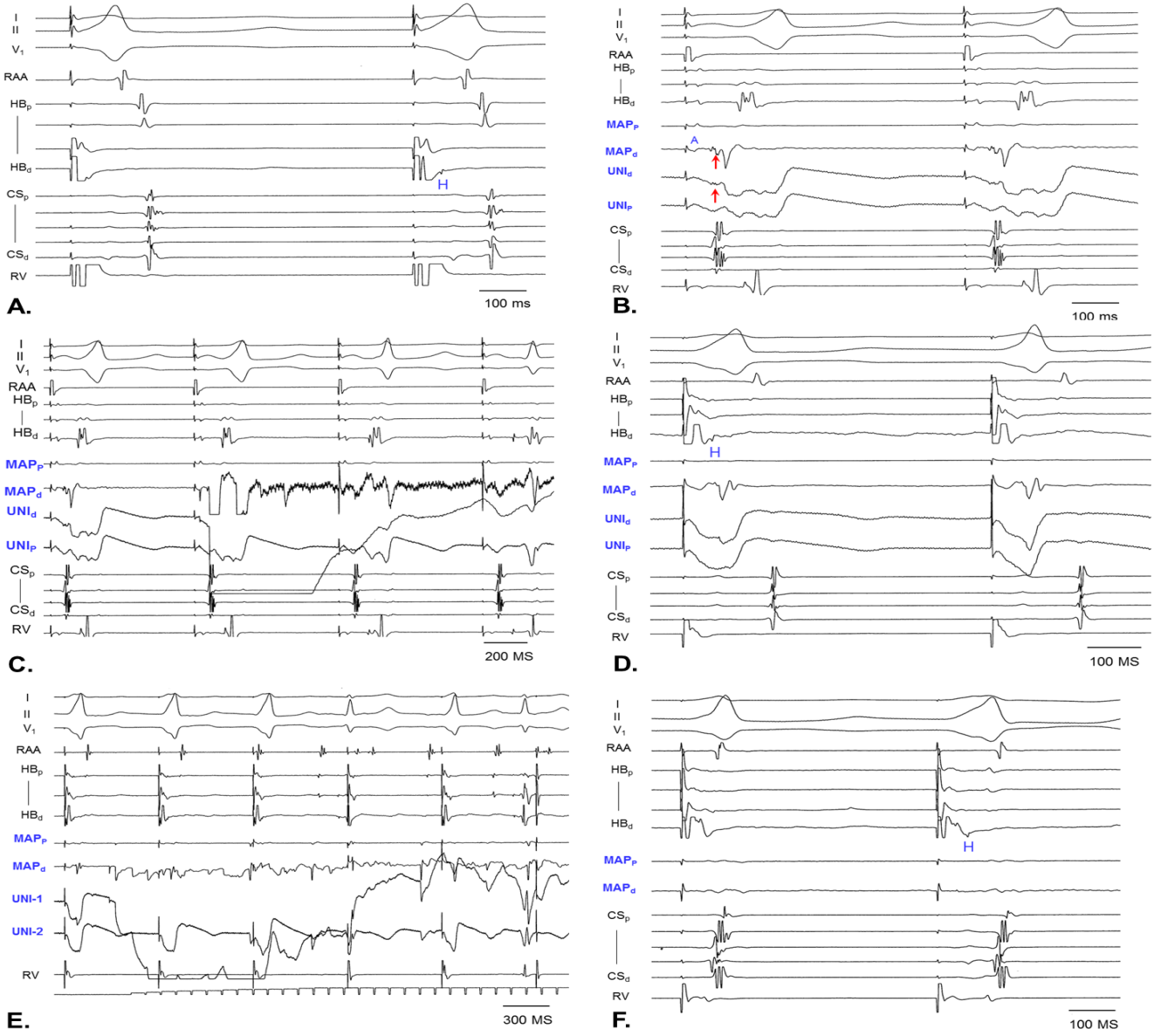


Figure 6.3. Parahisian pacing to evaluate ablation success. A. Parahisian pacing before ablation showed that retrograde conduction was mediated by AP only. **B.** An AP-P (red arrow) was recorded during antegrade AP conduction. **C.** Ablation there led to immediate loss of preexcitation. **D**. Parahisian pacing after RF application showed that retrograde AP conduction was only delayed slightly. Note that the atrial timing of the RAA was significantly delayed, which may mislead the operator to believe that retrograde conduction was mediated by the AVN. **E.** Ablation caused VA block. **F.** Parahisian pacing after ablation showed retrograde AVN conduction.

2. Induce the clinical SVT and verify the mechanism of SVT by resetting or entrainment responses (see **Chapter 4** for detail).
3. Pace multiple atrial or ventricular sites to find the best pacing site that produces the longest AV or VA interval (see **Chapter 5** for detail). The direction of the oblique course can be identified as well. The end of the AP more distant from the septum is Dr. Jackman's preferred target (see discussion below).
4. Prove the targeted accessory pathway potential (AP-P) is truly an AP-P. The first RF application is the most important one, which should only target the AP-P. If an AP-P cannot be found after search the 4 chambers and coronary cusps, the site of earliest ventricular activation during antegrade AP conduction or the site of earliest atrial activation during retrograde AP conduction can serve as the target of ablation. However, this has to be done by pacing at sites that produce the longest AV or VA interval. In this way, the site of earliest activation will not be obscured by fused AV or VA electrograms (EGMs). Dr. Jackman had received many referrals for failed AP ablation; most of the failures could have been prevented by choosing the pacing site wisely to avoid ablating the AV or VA fusion site.

5. Stop ablation immediately if junctional automaticity occurs during RF application to an AS-AP or MS-AP because it is a prelude to AVN injury.

Anteroseptal AP (AS-AP) and Midseptal AP (MS-AP)

It is well known that AS-APs and MS-APs can easily be bumped by catheters. Dr. Jackman's approach to positioning the HB catheter to record a stable HB potential without bumping the AP is to cross the tricuspid annulus low and laterally. If a deflectable catheter is used, Dr. Jackman prefers to cross the annulus with a tight curve so that the tip of the catheter will not traumatize the AP. After the HB catheter or RV catheter entered the RV, the curve of the catheter is released while withdrawing the catheter toward the AV junction. This maneuver is also applicable to a stiff ablation catheter that tends to mechanically traumatize the AP.

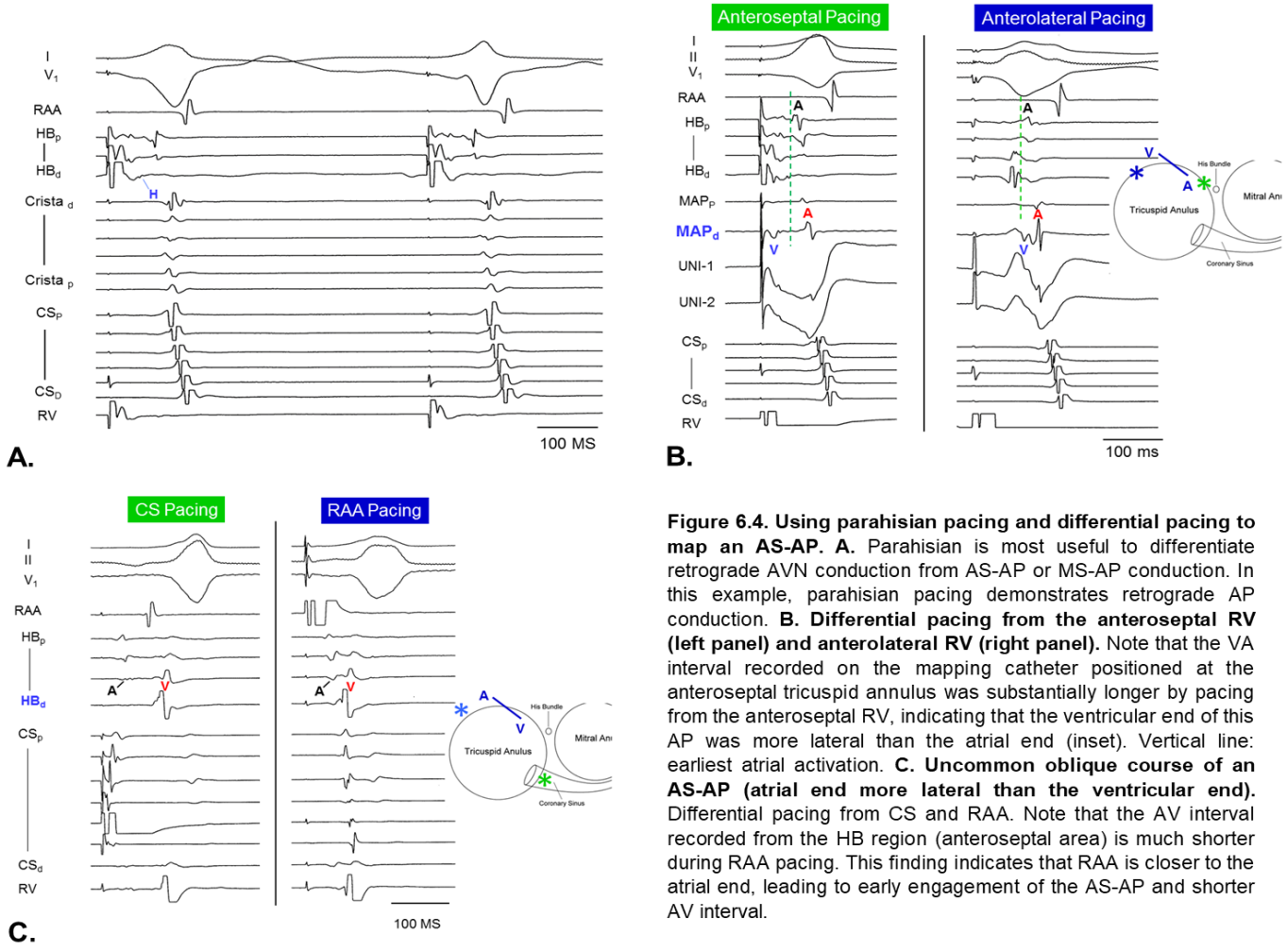


Figure 6.4. Using parahisian pacing and differential pacing to map an AS-AP. A. Parahisian is most useful to differentiate retrograde AVN conduction from AS-AP or MS-AP conduction. In this example, parahisian pacing demonstrates retrograde AP conduction. **B. Differential pacing from the anteroseptal RV (left panel) and anterolateral RV (right panel).** Note that the VA interval recorded on the mapping catheter positioned at the anteroseptal tricuspid annulus was substantially longer by pacing from the anteroseptal RV, indicating that the ventricular end of this AP was more lateral than the atrial end (inset). Vertical line: earliest atrial activation. **C. Uncommon oblique course of an AS-AP (atrial end more lateral than the ventricular end).** Differential pacing from CS and RAA. Note that the AV interval recorded from the HB region (anteroseptal area) is much shorter during RAA pacing. This finding indicates that RAA is closer to the atrial end, leading to early engagement of the AS-AP and shorter AV interval.

Dr. Jackman absolutely would not pace the RV apex to map an AS-AP or MS-AP. Indeed, he developed the parahisian pacing technique specifically to avoid the confusion introduced by RV apical pacing that can easily capture a distal branch of the RBB, leading to retrograde AVN conduction that fuses with retrograde AP conduction. In Dr. Jackman's practice of mapping an AS-AP or MS-AP, the most important step is to use differential pacing to find out how this AP traverses the AV annulus and identify the atrial or ventricular end that is more distant from the AV node. The end of the AP more distant from the AVN will be the ablation target to minimize the risk of AVN injury (**Figure 6.4A-B**). For most AS-APs, the ventricular end is more lateral than the atrial end; the ventricular end is the ablation target. However, in a minority of patients,

the oblique course is reversed, in which ablating the atrial end more lateral to the anterior septum will be safer (**Figure 6.4C**). For MS-APs, the ventricular end of the AP is usually inferior to the atrial end. Pacing CS ostium may lengthen the AV interval to facilitate mapping and ablation.

Of note, differential pacing is best to be conducted when the ablation catheter is positioned adjacent to the expected location of the AP to visualize the change of local AV or VA interval adjacent to the AP. Certainly, the ablation catheter should not be positioned directly on the AP to try this pacing maneuver to avoid bumping the AP. For example, in the presence of a right free wall AP, lateral CS, which is distant from the AP, may show the largest AV interval change caused by differential pacing. However, measuring the local AV or VA interval change at a site distant from the AP is meaningless.

Dr. Jackman's practice to map and ablate an AS-AP is summarized as follows.

1. Start with parahisian pacing to examine the mechanism of retrograde conduction (all AP or all AVN or fusion). Pacing CL producing "pure" retrograde AP conduction is selected for mapping and ablation.
2. Deliver differential pacing from the anteroseptal and anterolateral RV to verify if the ventricular end of the AP is more lateral than the atrial end. If pre-excitation is present, differential atrial pacing (RAA and proximal CS) can help determine the oblique course of the AP as well.
3. It is advisable to periodically check retrograde AP conduction with parahisian pacing because the patient's sympathetic tone may increase with time, leading to improvement of retrograde AVN conduction. The operator may not be aware that at the same pacing CL, retrograde conduction is no longer mediated by AP alone but fusion of AP and AVN. Unknowingly mapping and ablating the latter may cause AV block.
4. If fusion of retrograde conduction is identified, pacing at shorter CL may reestablish pure retrograde AP conduction. Sometimes, retrograde AVN conduction becomes very robust; mapping has to be conducted during AVRT. Operators can use the method described in **Figure 5.19** to entrain the AVRT by RV pacing immediately before ablation in order to prevent catheter jump when AVRT is terminated by ablation.
5. Mapping during both antegrade and retrograde AP conduction provides more information than just mapping in one direction of AP conduction.
6. The primary ablation target is always the AP-P, not earliest atrial or ventricular activation. If an AP-P is nowhere to be found (e.g. an epicardial AP), Dr. Jackman may target the site of earliest atrial or ventricular activation while pacing from a site producing the longest AV or VA interval.

In the event that the best apparent target is dead-on at the anterior septum or mid-septum, Dr. Jackman would map the aortic cusp and septal aorto-mitral continuity first before taking a riskier shot at the anterior septum or mid-septum (**Figure 6.5A**). This is particularly helpful when the pre-excitation pattern suggests an anteroseptal location but the local ventricular activation in the HB region appears to be late or far-field. It is not uncommon to find a sharp AP-P in the non-coronary cusp representing the atrial end of the AP; ablation there carries a lower risk of AV block.

The author prefers to use a right subclavian vein or internal jugular vein approach can be used to target the ventricular end of an AS-AP (**Figure 6.5B-C**). As the AVN is in the triangle of Koch and is on the atrial side of the annulus, ablating the ventricular end of the AP may cause RBBB but much less likely to

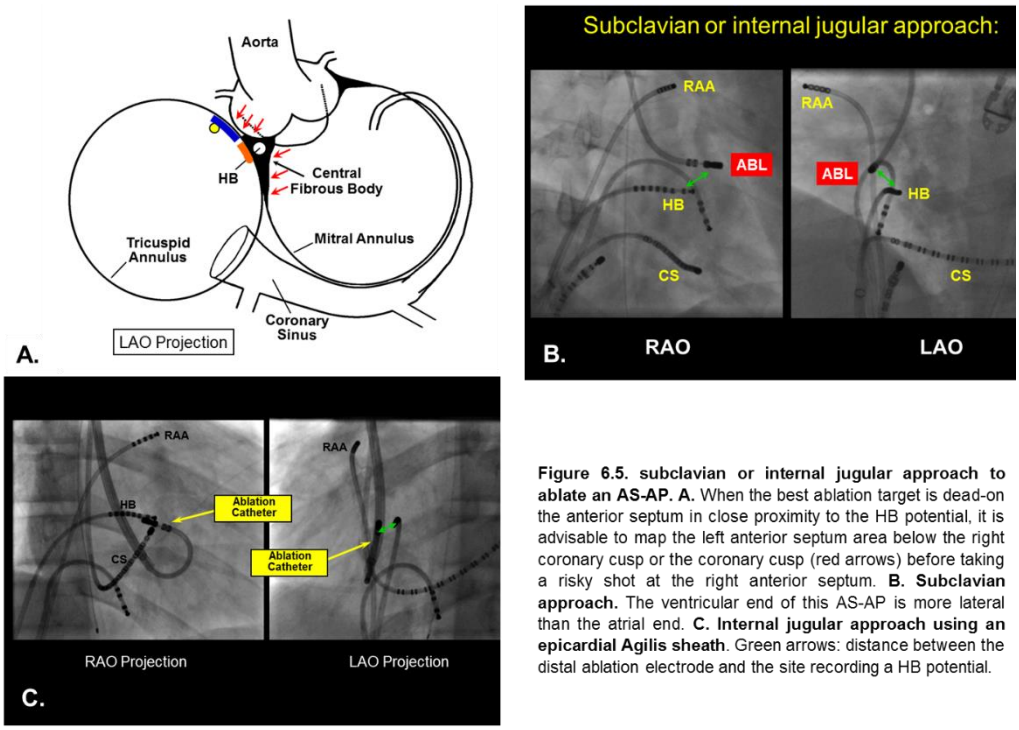


Figure 6.5. subclavian or internal jugular approach to ablate an AS-AP. A. When the best ablation target is dead-on the anterior septum in close proximity to the HB potential, it is advisable to map the left anterior septum area below the right coronary cusp or the coronary cusp (red arrows) before taking a risky shot at the right anterior septum. **B. Subclavian approach.** The ventricular end of this AS-AP is more lateral than the atrial end. **C. Internal jugular approach using an epicardial Agilis sheath**. Green arrows: distance between the distal ablation electrode and the site recording a HB potential.

cause AV block. This approach requires the tip of the ablation catheter to be hooked under the ventricular side of the tricuspid annulus. A small-radius catheter may not be able to cross the annulus while a larger-radius catheter may not be curled under the valve. In this scenario, a short deflectable sheath from the right internal jugular vein allows a small-radius catheter to cross the valve and be hooked under the valve.

In the presence of a widely separated AV or VA interval, ablating the site of earliest atrial or ventricular activation of a left or right free wall AP is probably as successful as ablating the AP-P. However, in AS-AP or MS-AP ablation, every millimeter counts. Certainly, not every sharp potential is an AP-P. A HB potential is sharp as well. Dr. Jackman would prove that this sharp potential is not a HB potential (see discussion below), followed by meticulously proving that the sharp potential is indeed an AP-P before targeting it. If the sharp potential is neither an atrial potential nor a ventricular potential, this sharp potential has to be an AP-P (**Figure 6.6**). The pacing maneuver to prove an AP-P is summarized as follows.

1. A quick and dirty way is to employ decremental pacing until AP conduction blocks to determine if the sharp potential of interest disappears with AP conduction block. In **Figure 6.6A**, it is clear that the sharp potential of interest is not part of the local atrial potential, indicating that it is either an AP-P or part of the local ventricular potential. Similarly, decremental RV pacing can be employed to see if the sharp potential disappears when retrograde AP conduction blocks.
2. Another quick and dirty way is to deliver single atrial or ventricular extra-stimuli until AP conduction block to examine if the sharp potential of interest is missing at the moment of AP conduction block. The author usually avoids this maneuver because extra-stimuli often make catheter move and the EGM of the conducted beat and blocked beat can look very different, making it difficult to determine if the sharp potential is missing at the moment of AP conduction block (**Figure 6.6B**).
3. On a very lucky day, non-sustained AVRT is easily inducible. Operators may look for any potential that is missing when AVRT terminates spontaneously (**Figure 6.6C**).
4. Dr. Jackman prefers to deliver atrial extra-stimuli (AES) during RV pacing to see if AES can move the local atrial potential and dissociate it from the sharp potential of interest (**Figure 6.6D**). In the presence of antegrade AP conduction, progressively earlier AES eventually engage antegrade AP conduction. In this case, the sharp potential is advanced by antegrade AP conduction but the local ventricular potential is still activated by RV pacing, dissociating the local ventricular potential from the sharp potential (**Figure 6.6E**). Certainly, the same principle can be applied to deliver ventricular extra-stimuli during atrial pacing.

Operators must differentiate a HB potential from an AP-P. For a concealed AS-AP, the HB potential is obvious during sinus rhythm or orthodromic AVRT. For a manifest AP, decremental RA pacing can be

helpful. A HB potential will progressively move into the local ventricular potential while an AP-P will persistently stay ahead of the onset of the delta wave. Another way is to induce orthodromic AVRT. If a patient presents with an anteroseptal or midseptal AP with profound pre-excitation but the HB potential cannot be found, operators should be suspicious of poor antegrade AVN conduction, possibly AVN injury from prior ablation. Dr. Jackman may look for a HB potential by delivering parahisian pacing. When the HB/RBB is not captured, a retrograde HB potential may be visible. One might have to search for a HB from the left anterior septum.

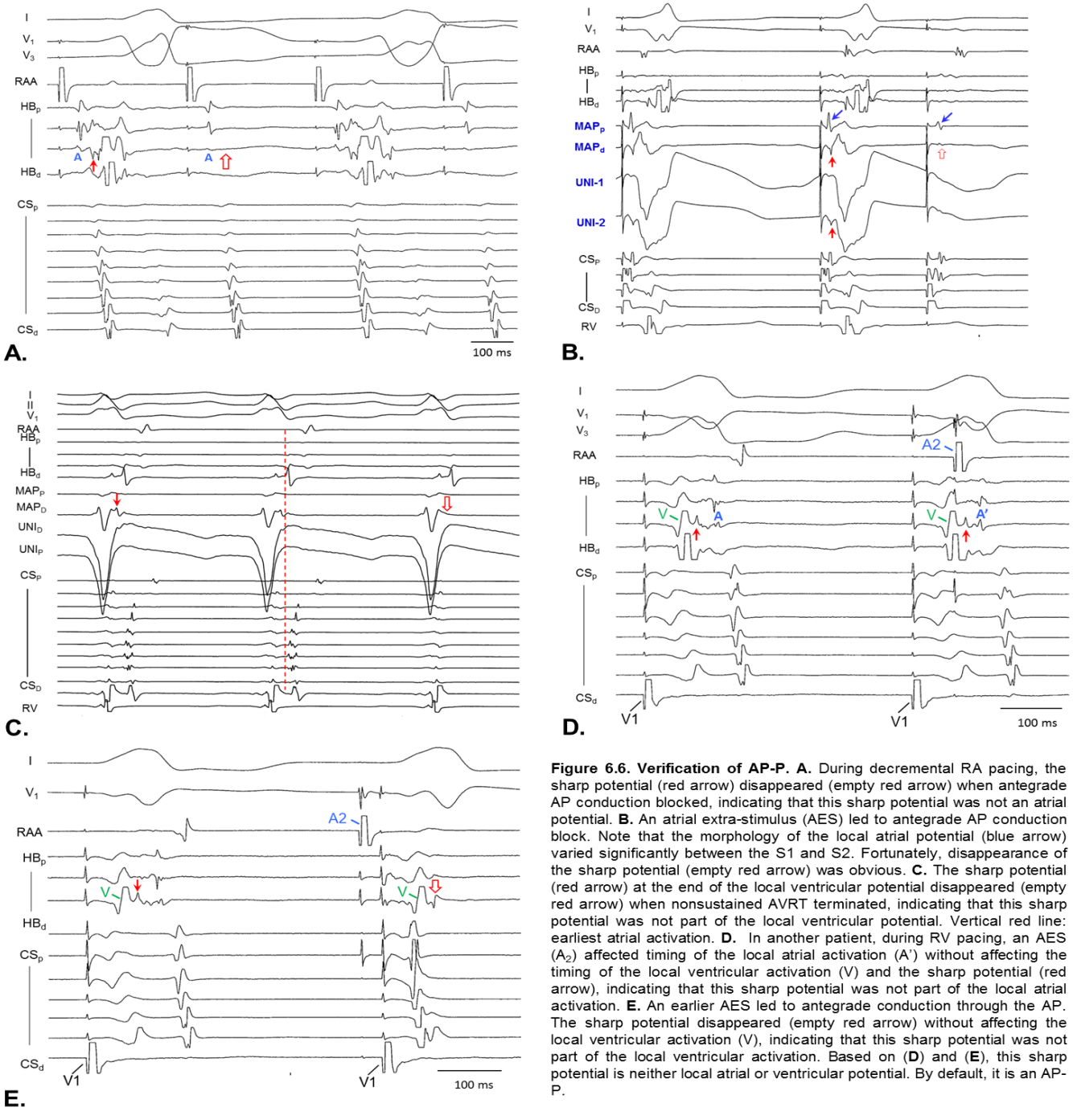


Figure 6.6. Verification of AP-P. A. During decremental RA pacing, the sharp potential (red arrow) disappeared (empty red arrow) when antegrade AP conduction blocked, indicating that this sharp potential was not an atrial potential. **B.** An atrial extra-stimulus (AES) led to antegrade AP conduction block. Note that the morphology of the local atrial potential (blue arrow) varied significantly between the S1 and S2. Fortunately, disappearance of the sharp potential (empty red arrow) was obvious. **C.** The sharp potential (red arrow) at the end of the local ventricular potential disappeared (empty red arrow) when nonsustained AVRT terminated, indicating that this sharp potential was not part of the local ventricular potential. Vertical red line: earliest atrial activation. **D.** In another patient, during RV pacing, an AES (A_2) affected timing of the local atrial activation (A') without affecting the timing of the local ventricular activation (V) and the sharp potential (red arrow), indicating that this sharp potential was not part of the local atrial activation. **E.** An earlier AES led to antegrade conduction through the AP. The sharp potential disappeared (empty red arrow) without affecting the local ventricular activation (V), indicating that this sharp potential was not part of the local ventricular activation. Based on (**D**) and (**E**), this sharp potential is neither local atrial or ventricular potential. By default, it is an AP-P.

An AP-P is almost always sharp but its amplitude can be quite small. A rounded “AP-P” should alarm the operator that this “AP-P” is far-field; the ideal ablation site is elsewhere. Dr. Jackman’s favorite target is a site where the AP-P is recorded only on the distal unipolar electrode (UNI-1). The distal unipolar electrode does not record any atrial EGM while the proximal unipolar electrode (UNI-2) records a small atrial EGM

(**Figure 6.7)**, providing convincing evidence that the tip electrode of the ablation catheter is on the ventricular side of the annulus to minimize the risk of AV block. If the proximal unipolar electrode does not record a small atrial EGM, it suggests that the distal electrode pair of the ablation catheter may be deep in the ventricle; the catheter needs to be withdrawn back to the annulus.

It is obvious that it is safer to ablate a manifest AS-AP during antegrade AP conduction that allows the operator to monitor the AVN conduction. In the event that the HB potential is obscured by the local ventricular activation, the author's preference is to pace proximal CS in which the atrial wave front directly enters the triangle of Koch and AVN, thereby shortening the AH interval to allow the operator to see the HB potential (**Figure 6.8**). This approach does not always work but is worth trying. For example, RAA pacing may provide the widest AV separation for mapping. When the ideal ablation target is identified, the operator can switch to proximal CS pacing to see if the HB potential becomes visible. If so, the operator can switch back and forth between RAA and CS pacing to ensure that the ablation catheter has not moved due to proximal CS pacing. If the ablation catheter is still on the target, ablation can be started during proximal CS pacing to monitor the AH interval.

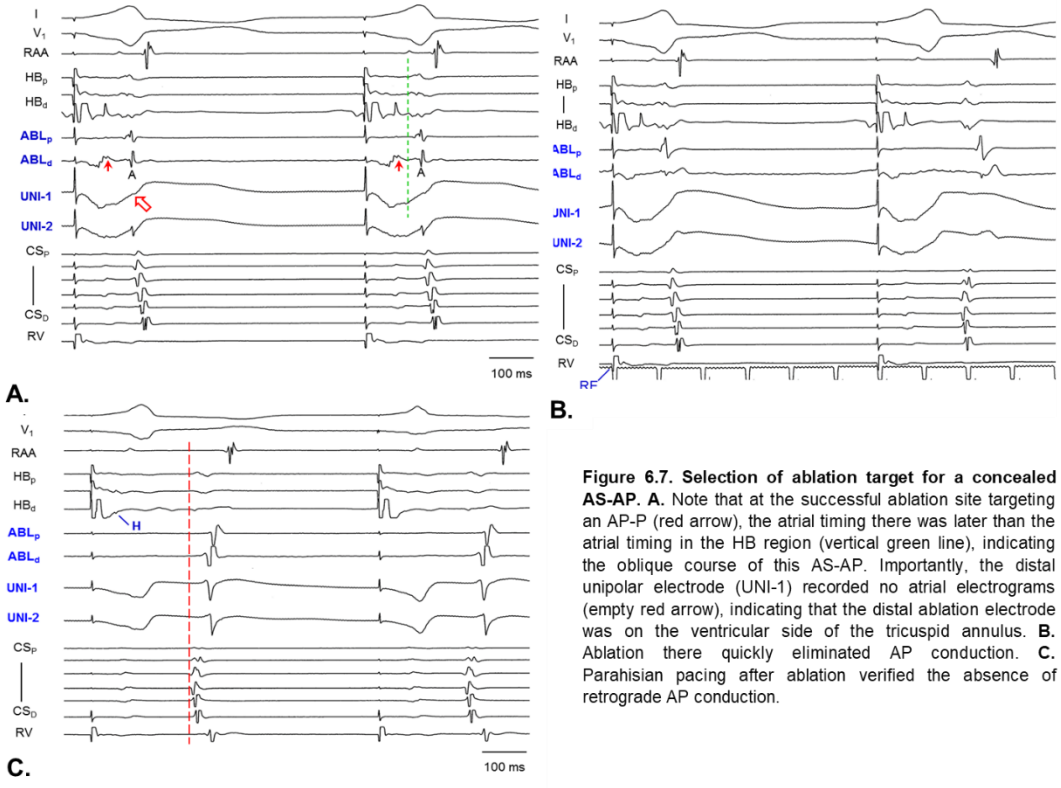


Figure 6.7. Selection of ablation target for a concealed AS-AP. A. Note that at the successful ablation site targeting an AP-P (red arrow), the atrial timing there was later than the atrial timing in the HB region (vertical green line), indicating the oblique course of this AS-AP. Importantly, the distal unipolar electrode (UNI-1) recorded no atrial electrograms (empty red arrow), indicating that the distal ablation electrode was on the ventricular side of the tricuspid annulus. **B.** Ablation there quickly eliminated AP conduction. **C.** Parahisian pacing after ablation verified the absence of retrograde AP conduction.

To ablate a concealed AS-AP is substantially more difficult as it is very challenging to monitor the AVN conduction during RV pacing, particularly when VA block occurs after eliminating retrograde AP conduction. In Dr. Jackman's practice, as long as the tip electrode (UNI-1) of the ablation catheter does not record an atrial potential, indicating the tip electrode is on the ventricular side of the annulus, he continues RF application despite VA block. If parahisian pacing before ablation demonstrates retrograde fast pathway conduction, Dr. Jackman would pace the RV at a CL longer than the retrograde fast pathway block CL. When retrograde AP conduction is eliminated, he continues ablation during RV pacing as long as retrograde AVN fast pathway conduction is not affected. The author prefers to set the ventricular pacing CL 20-30 ms shorter than the sinus CL. When retrograde AP conduction block occurs, pacing cycle length is lengthened to allow sinus rhythm and antegrade AVN conduction to resume quickly and smoothly after AP block. This maneuver can avoid AVN injury due to brisk catheter movement when RV pacing is abruptly discontinued in an attempt to monitor the AVN function. If the AP was "bumped" before RF application, Dr. Jackman waits for a minute or two. If AP conduction does not return, Dr. Jackman delivers RF application at that site in sinus rhythm. If accurate mapping was performed, the recurrence rate of ablating the bumped site is very low in Dr. Jackman's practice. Of note, Dr. Jackman delivers empirical RF applications in this way only if an AP-P was clearly recorded and proven before losing AP conduction.

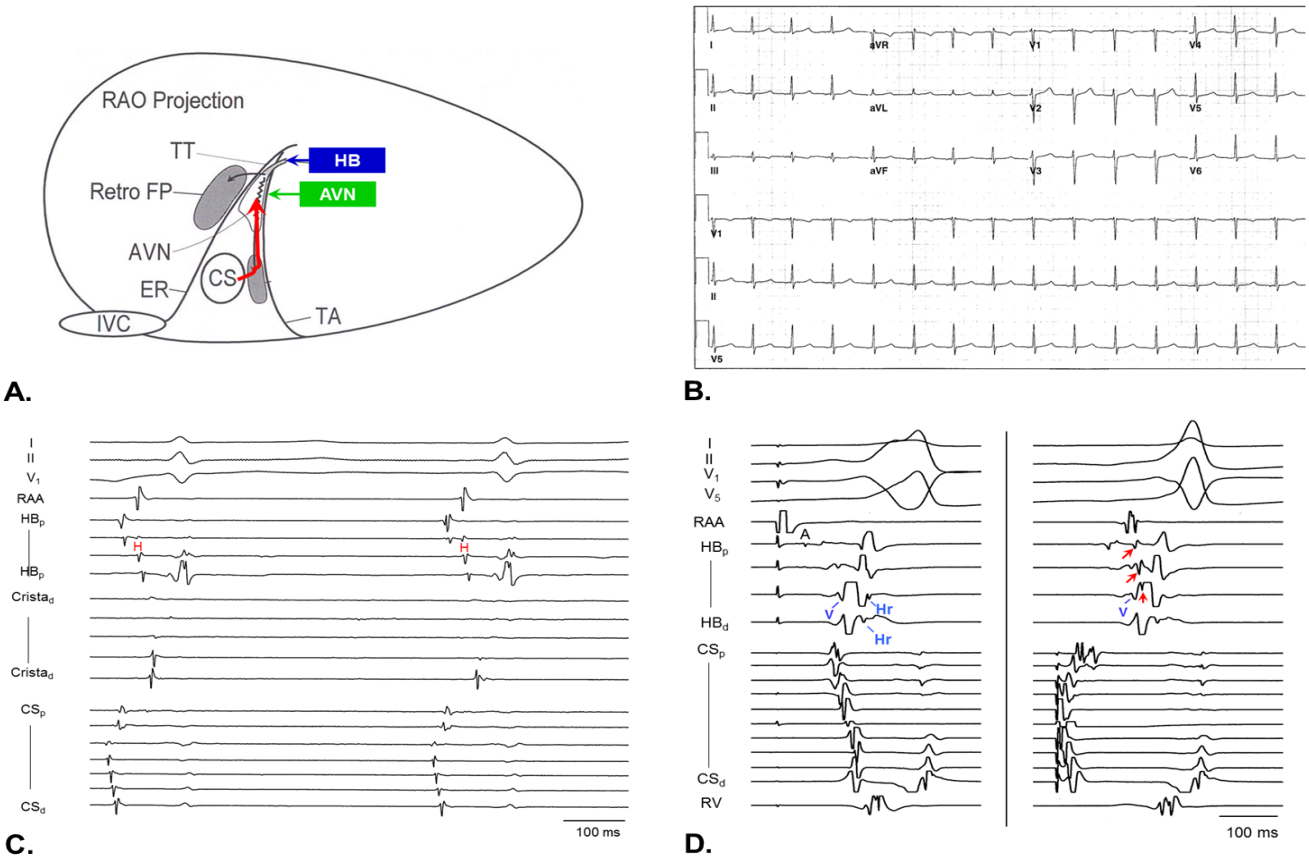


Figure 6.8. Pacing from CS ostium to shorten the AH interval. A. Pacing from proximal CS often creates an activation wave front that enters the AVN sooner than that from the sinus node, leading to a shorter AH interval. **B and C.** ECG and EGM of a patient with ectopic atrial rhythm originated in proximal CS. Note that the P wave was nearly invisible due to a very short PR interval; the AH interval was very short. **D.** A patient with a manifest anteroseptal AP. **Left panel**: Retrograde HB potential (Hr) was buried in local ventricular activation. **Right panel**: During proximal CS pacing, the wave front entered the AVN quickly, producing a short AH interval and making the HB potential visible now (red arrow: HB potential). Notably, the HB potential conducted in the antegrade direction from HB_p to HB_d

The author worked with Dr. Jackman for 19 years and has never seen Dr. Jackman use a cryocatheter to ablate an AS-AP or MS-AP. The main reason is cryocatheter's larger recording range, leading to poor localization of the AP. In addition, a cryocatheter is a lot stiffer than an RF ablation catheter, leading to a higher incidence of bumping the AS-AP during mapping. There have been plenty of reported cases that cryoablation led to permanent AVN injury, possibly resulting from operators' false sense of safety. Despite treating many patients with prior failed AS-AP or MS-AP ablation, AVN injury occurred in less than 2% patients in Dr. Jackman's practice using the RF energy source. For young patients with an AS-AP or MS-AP, the author maps the pathway using a 4mm-tip or 3.5mm-tip RF catheter first. If the risk of AVN injury is high, the RF catheter is exchanged for a cryocatheter only after the ideal ablation site has been identified.

Dr. Jackman's approach to MS-AP ablation is essentially the same as AS-AP ablation. The oblique course of an MS-AP often means that the atrial end of the AP is superior or inferior to the ventricular end. RV differential pacing is delivered from basal anteroseptal and posteroseptal RV in order to "sandwich" the MS-AP. RAA and proximal CS differential pacing is employed as well if antegrade AP conduction is present. After the direction of the oblique course of the MS-AP is determined, mapping is performed during pacing from the site that provides the longest AV or VA interval. Again, the ideal ablation target is on the ventricular side of the annulus without recording an atrial EGM on the distal unipolar electrode (**Figure 6.9)**. If junctional automaticity occurs, RF application should be stopped immediately because it indicates that the distal ablation electrode is in the triangle of Koch (too atrial). The ablation catheter needs to be gently advanced into the RV until the distal unipolar EGM (UNI-1) no longer records an atrial potential.

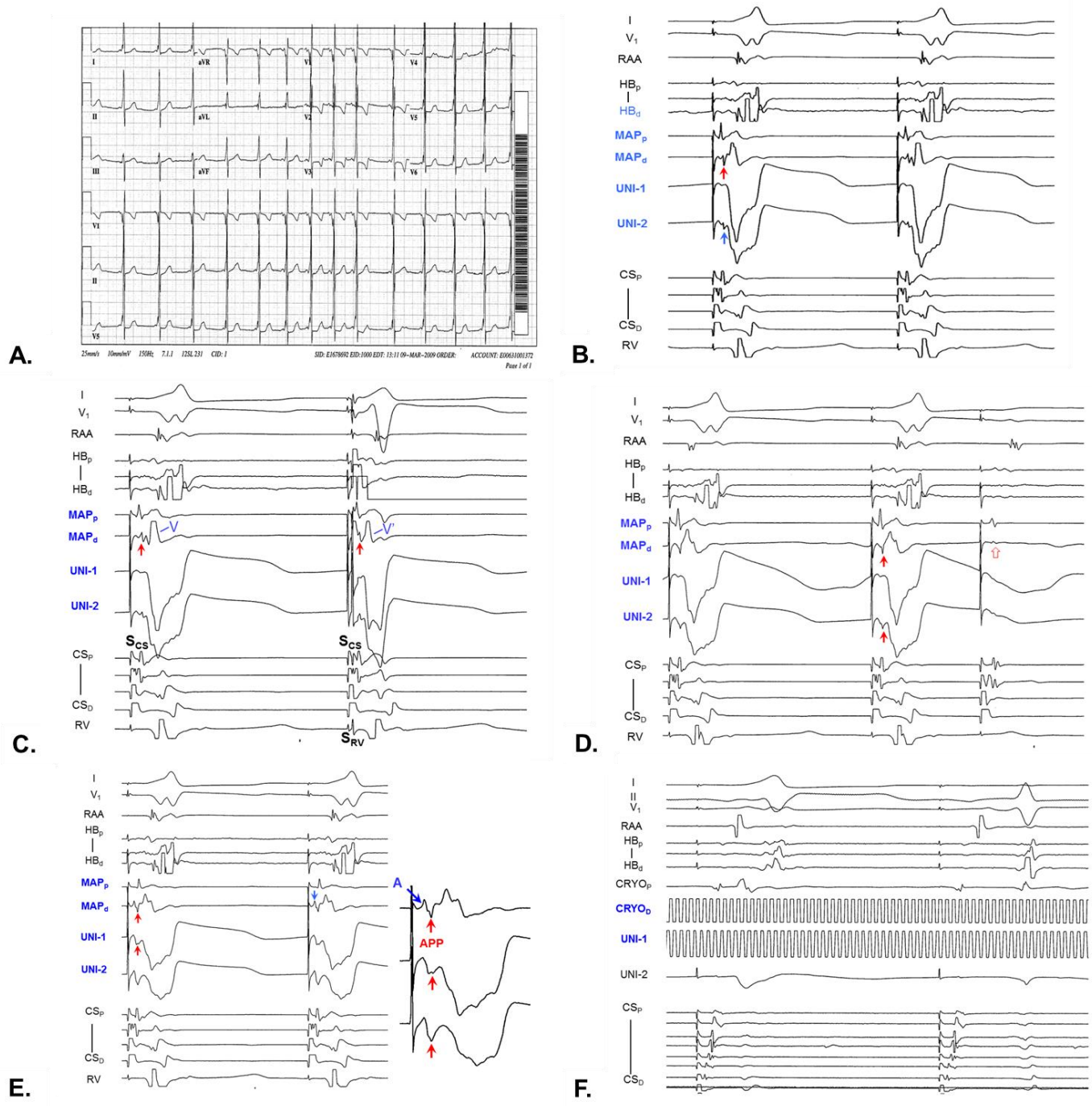


Figure 6.9. **Ablation of an MS-AP in a child. A.** The preexcitation pattern was consistent with a high MS-AP or AS-AP. **B.** A sharp potential recorded on the distal electrode pair (red arrow) as well as on the proximal unipolar electrode (UNI-2; blue arrow). **C.** At pacing CL of 500 ms (S_{CS}), a ventricular extra-stimulus (S_{RV}) advanced the local ventricular potential (V') by 15 ms without affecting the sharp potential, indicating that this sharp potential was not part of the local ventricular potential. **D.** A premature atrial stimulation induced antegrade AP conduction block. The sharp potential disappeared (red blank arrow), indicating that this sharp potential was not part of the local atrial potential. This sharp potential was deemed as an AP-P since it was neither part of the local atrial potential or ventricular potential. Notably, this AP-P was recorded on the proximal unipolar electrode (UNI-2), not the distal unipolar electrode (UNI-1). The ablation catheter needs to be withdrawn slightly until the AP-P is recorded by the distal unipolar electrode. **E.** EGM near the successful ablation site. Note that the AP-P (red arrow) was recorded on the bipolar electrodes as well as both of the proximal and distal unipolar electrode. The atrial potential (blue arrow) appeared to be recorded on both the distal and proximal electrode. The ideal ablation site should be slightly more ventricular. **F.** After carefully localizing the ablation target, a 6-mm cryocatheter was used to ablate this high MS-AP. Note that Dr. Jackman would have not switched to a cryocatheter.

If operators notice the degree of preexcitation is increasing while ablating a septal AP, RF application should be terminated immediately. Increasing preexcitation during ablation can be caused by either AVN injury (bad) or heating up the AP (good). Occasionally, patients with a septal AP and failed prior ablation may

present with profound preexcitation and a HB potential is nowhere to be found. Dr. Jackman would implement the following steps to evaluate antegrade AVN conduction.

a. If retrograde fast pathway conduction is OK, antegrade fast pathway conduction is usually OK as well. If retrograde conduction is completely mediated by AP at multiple pacing CL, there is a high likelihood that the AVN function is very poor.

b. Try different pacing sites (e.g. CS ostium).

c. Try parahisian pacing. When HB/RBB capture is lost, a retrograde HB potential may be visible.

d. Try to record a HB potential from the left ventricle.

e. Induce orthodromic AVRT to look for a HB potential.

In an awkward situation that AVN conduction is very poor or is absent, Dr. Jackman's inclination is to eliminate the AP and refer the patient to pacemaker implantation. His practice is based on the following anecdotal experience.

1. These patients often develop preexcited AF with rapid ventricular rate because antegrade AP conduction has no competitor anymore.

2. Antegrade AP conduction is not always stable; it also appears to decline with age. If the history indicates that AVN was injured in prior ablation, Dr. Jackman would schedule a repeat ablation procedure sooner than usual because he had patients with AVN injury died suddenly while waiting for ablation. Dr. Jackman suspected that unstable antegrade AP conduction might have led to asystole.

Left MS-AP

This rare type of AP is the one that has to be ablated from the left mid-septum on the aorto-mitral continuity where no AP should exist due to absence of myocardium there. Certainly, exceptions exist. The OU-EP group have collected at least 15 patients with this type of AP. The preexcitation pattern does not look different from that of a typical right midseptal AP (**Figure 6.10A**). The VA interval recorded in the HB region and CS ostium is often very similar because left MS-APs are located halfway between the HB and CS ostium (**Figure 6.10B**). On some occasions, a far-field AP-P may be recorded when the mapping catheter is directed upward from the roof of the CS ostium, pointing straight at the left MS-AP (**Figure 6.10C**). This observation should clue the operator to map the left mid-septum. Dr. Jackman would not attempt to ablate the roof of CS near the ostium because of high risk of AVN injury.

The ablation target again is the AP-P. Because recording of the ventricular end of a left MS-AP is often hindered by the aorto-mitral continuity, ablation of an MS-AP usually targets the AP-P at a site with a balanced atrial and ventricular EGM or a site with a slightly larger atrial EGM (**Figure 6.10D-E**). This is in sharp contrast to the right MS-AP or left free wall AP. The preferred ablation targets of the former records no atrial potential at all on the distal unipolar electrode (UNI-1); the preferred ablation site of the latter typically exhibit a small atrial EGM and a large ventricular EGM. If junctional rhythm occurred during RF application to a left MS-AP, ablation should be stopped immediately to check antegrade AVN conduction. This is particularly important because mitral valve is less apical than the tricuspid valve; therefore, septal mitral annulus may be in close proximity to the triangle of Koch. Ablation of left MS-AP and left AS-AP (below the right coronary cusp) carries a significant risk of AVN injury. Dr. Jackman prefers to ablate a left MS-AP with the trans-septal approach using an SL2 or sometimes SL3 sheath to provide stable electrode-tissue contact.

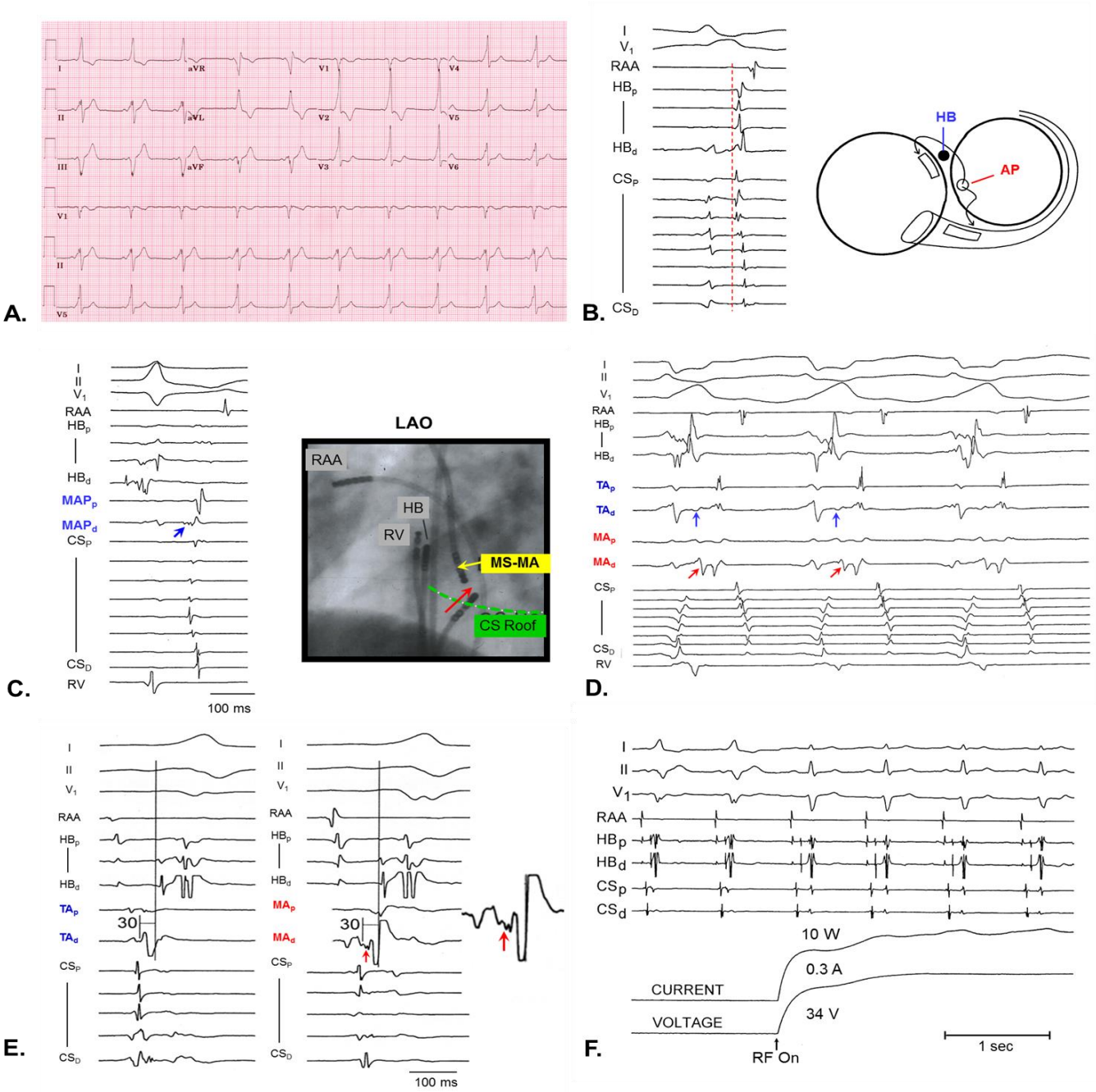


Figure. 6.10. Left MS-AP. A. The preexcitation pattern predicts a right MS-AP. **B.** The atrial timing recorded from the His bundle area and CS ostium was very similar (**left panel**) due to simultaneous activation of the posteroseptal and anteroseptal area (**right panel**). **C.** A far field AP-P (blue arrow) was recorded from the roof of the CS ostium when the mapping catheter was gently pushed upward (red arrow) toward the left midseptal area. MS-MA: midseptal mitral annulus. **D.** In AVRT, a far-field AP-P was recorded from the right midseptal area (blue arrows). A sharp AP-P was recorded from the left midseptal area (red arrow). **E.** In another patient, over antegrade AP conduction, no AP-P was recorded from the right midseptal area (**left panel**). Far-field ventricular activation there began 30 ms before the onset of the delta wave. **Right panel.** An AP-P, 30 ms before the onset of the delta wave, was recorded from the left midseptal area. **F.** Ablation at that site quickly eliminated this AP. Courtesy of Dr. Jackman.

AS-AP located in or below the coronary cusp

Most of the AS-AP traverse the annulus with an oblique angle. If the ventricular end of the AP is on the septum, the atrial end can be either rightward or leftward to it. In this case, the operator should carefully search for either an AP-P or earliest atrial activation along the tricuspid annulus rightward to the HB area. If no atrial potential is earlier there or no AP-P can be found, there is a high likelihood that the atrial end of the AP may be more leftward to the HB area. Careful mapping in the coronary cusps and below the cusp may

identify the atrial end of the AP. Similar to the left MS-AP, left-sided AS-APs are located along the aorto-mitral continuity where no AP should exit due to absence of myocardium there. The OU-EP group have collected at

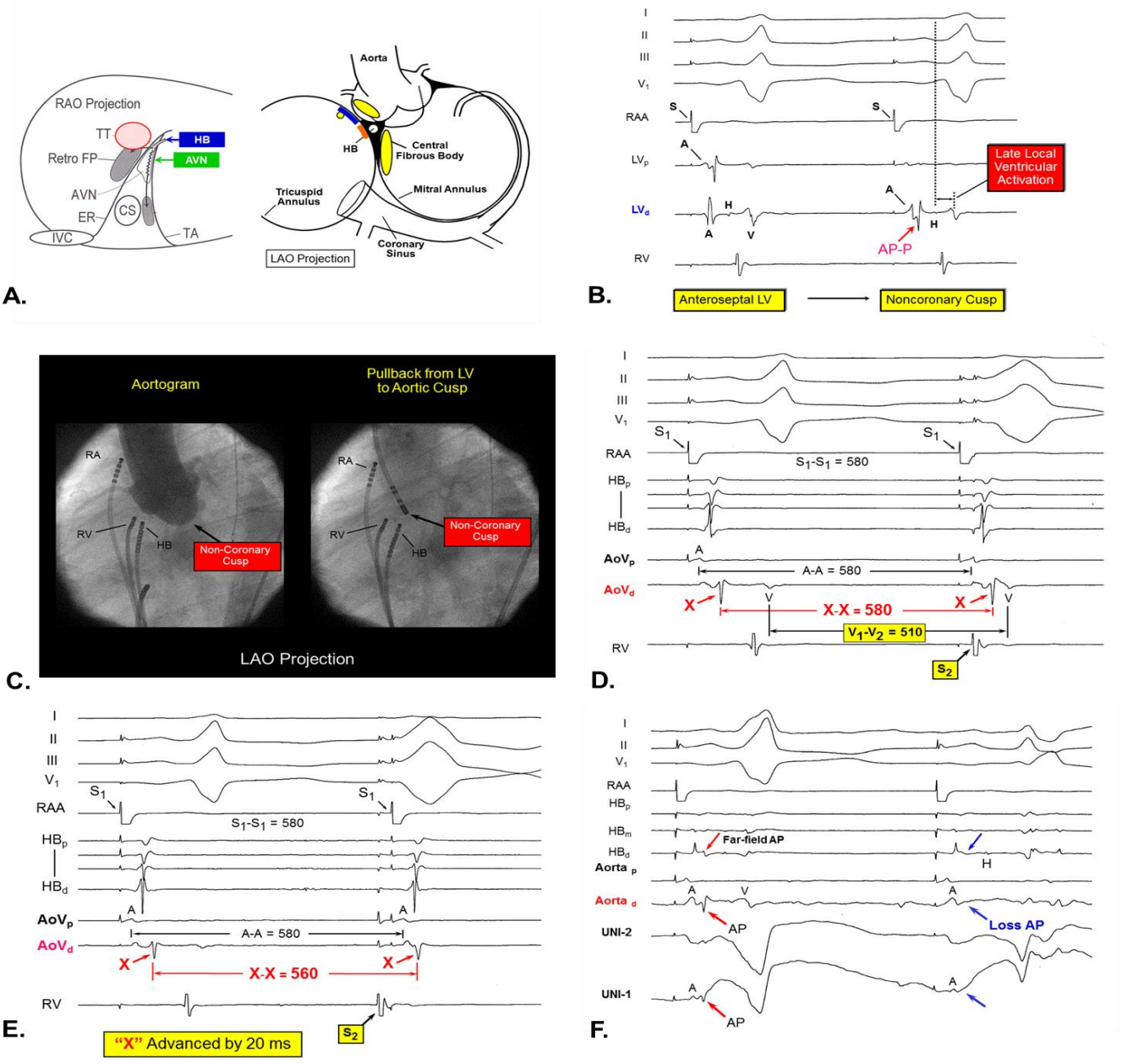


Figure 6.11. An AP located at the bottom of the noncoronary cusp. A. If the atrial activation timing is early behind the tendon of Todaro (hatched red area), it should alert the operator that the AP may be located in the left anteroseptal or left midseptal area (hatched yellow area) because mitral valve is less apical than the tricuspid valve. **B.** When the mapping catheter was pulled back from the anteroseptal LV (left beat) to the noncoronary cusp (right beat), a sharp potential (potential-X, red arrow) was recorded. Note that the local ventricular potential was a very late far-field potential, indicating that the ventricular insertion of this AP was distant from the noncoronary cusp. **C.** Radiographs show the location of the ablation catheter. **D.** During atrial pacing at a cycle length of 580 ms, a ventricular extra-stimulus (S2) was delivered, which advanced the ventricular potential by 70 ms without affecting potential-X. This observation indicated that potential-X was not a local ventricular potential. **E.** An earlier ventricular extra-stimulus advanced potential-X by 20 ms without affecting the local atrial potential, indicating that potential-X was not a local atrial potential. Potential-X was therefore an AP-P. **F.** Ablation there led to loss of the AP-P and loss of preexcitation. Modified with permission from *Circulation 2007;115:2465-2478.*

least 15 patients with this type of AP. Most of the APs were located at the bottom of the non-coronary cusp. A clue to this type of AP is that if the preexcitation pattern suggests an anteroseptal location but the local ventricular activation in the anteroseptal area is either not early or is far-field, mapping the coronary cusp is recommended. In the OU-EP laboratory, mapping the high RA septum behind the tendon of Todaro is part of the mapping procedure for an anteroseptal AP. If atrial activation is earlier behind the tendon of Todaro than that of the anteroseptal tricuspid annulus, it provides an important clue that the AP is located in the aorto-

mitral continuity (e.g. coronary cusp, below the right coronary cusp or left MS-AP) (**Figure 6.11A**). It is known that the His bundle is located just below the junction of the non-coronary and right coronary cusp. High RA septum behind the tendon of Todaro (therefore behind the HB potential) is directly opposite to the bottom of the non-coronary cusp. In the OU-EP laboratory, if an earlier atrial potential is recorded there, mapping of the non-coronary and right coronary cusp is commenced immediately. Since the non-coronary cusp is situated atop the epicardium of inter-atrial septum, ablation targets usually have a large atrial EGM, a sharp (sometimes large as well) AP-P and a small, far-field ventricular EGM (**Figure 6.11B**). Ablation targeting the AP-P usually runs a low risk of AV block. The ablation site at the junction of the right and non-coronary cusp may show a balanced amplitude of the atrial and ventricular EGMs.

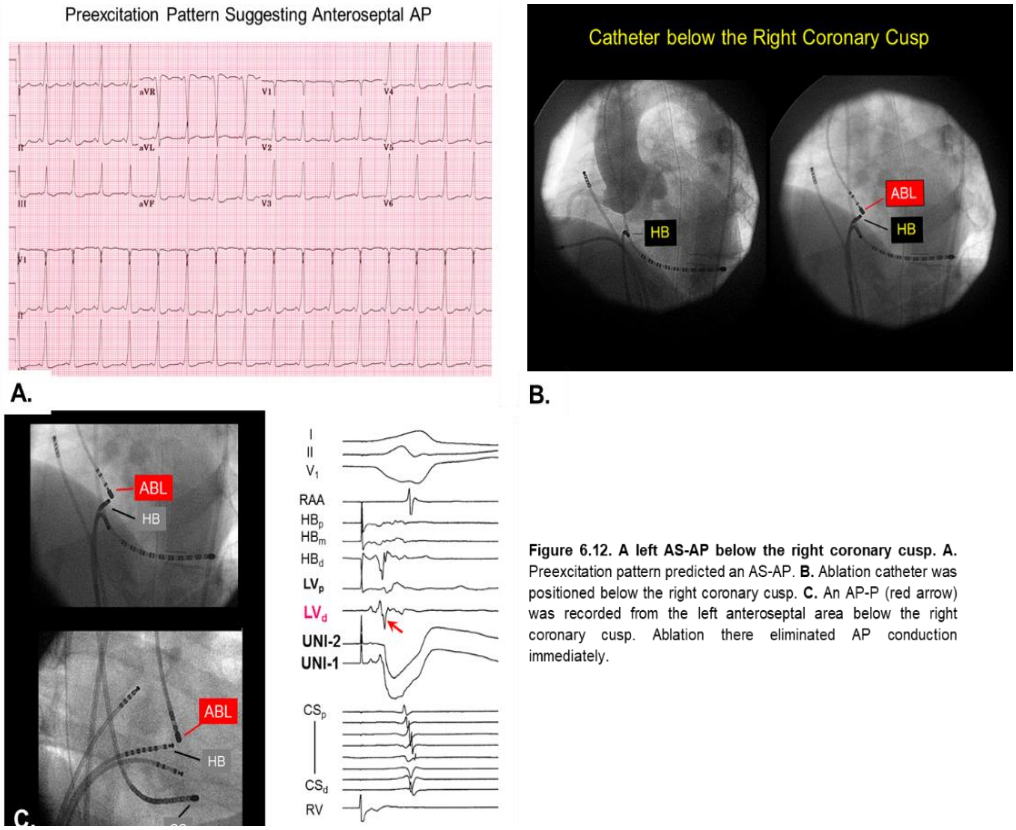


Figure 6.12. A left AS-AP below the right coronary cusp. A. Preexcitation pattern predicted an AS-AP. **B.** Ablation catheter was positioned below the right coronary cusp. **C.** An AP-P (red arrow) was recorded from the left anteroseptal area below the right coronary cusp. Ablation there eliminated AP conduction immediately.

Ablation of a left AS-AP below the right coronary cusp carries a very high risk of AV block because the His bundle here is no longer protected by the central fibrous body (**Figure 6.12**). The ablation target should be an AP-P; operators should make every attempt to prove that the targeted potential is indeed an AP-P. The AVN injury risk is similar to that resulting from ablating PVCs below the right coronary cusp where the HB emerges from the membraneous septum, no longer encased in the central fibrous body. Of note, ablation there often does not elicit junctional automaticity to warn the operator of imminent AVN injury. AV block can occur suddenly.

Posteroseptal AP (PS-AP)

The posteroseptal space does not have a septum at all; it is indeed a pyramidal space between the crux of the RA and LA. This pyramidal space is filled with fat and importantly coronary artery branches. As it is the junction of the 5 chambers (RA, LA, RV, LV and CS), mapping and ablation of PS-APs may require mapping of all the 5 chambers. Importantly, the tricuspid annulus is more apical than the mitral annulus. A posteroseptal AP may be formed by many possible combinations of connections between the 5 chambers (e.g. RA-RV, RA-LV, LA-LV, LA-RV, CS-LV, CS-RV). While most PS-AP can be successfully ablated from the tricuspid or mitral annulus, others require ablation inside the CS or its tributary (e.g. middle cardiac vein). This type PS-AP is referred to as epicardial PS-AP because the myocardium of the CS or its tributary is part of the AP connecting the atrium and epicardial surface of the LV.

Operators should be aware that atrial activation sequence over retrograde slow pathway conduction and epicardial PS-AP conduction may be very similar but the ablation target may be several cm apart (**Figure 6.13**). Parahisian pacing works very well to differentiate retrograde slow pathway conduction from retrograde AP conduction. Differential diagnosis between AVRT and slow/slow or fast/slow AVNRT can be a great challenge. There have been plenty of patients referred to the OU-EP group for ablation of SVT in which the

site of earliest atrial activation was the posteroseptal area. It is very common to find that the same arrhythmia had been ablated previously under the diagnosis of atypical AVNRT in one procedure and PS-AP in another procedure. If the AP happens to be located at the CS ostium or the inferior triangle of Koch where the right inferior extension of the AVN is located, ablating the slow pathway under the wrong diagnosis of fast/slow or slow/slow AVNRT may eliminate the PS-AP in some lucky cases.

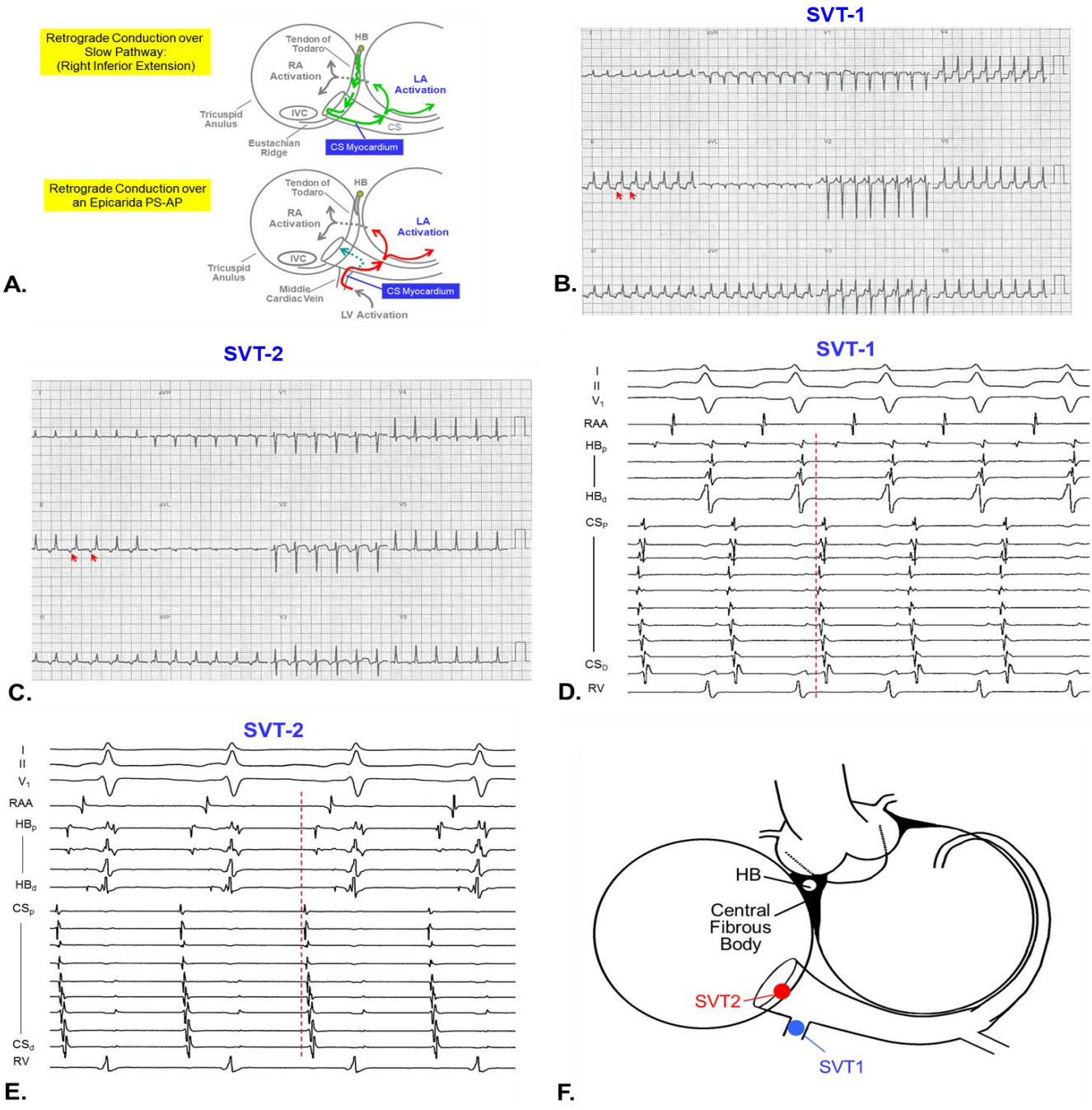


Figure 6.13. Similar atrial activation sequence between retrograde slow pathway and epicardial PS-AP. A. Both retrograde slow pathway conduction and epicardial PS-AP conduction involve CS myocardium and left atrium. They cannot be differentiated by activation sequence alone. **B and C.** ECG of two tachycardias in the same patient referred for paroxysmal AF ablation. Both SVTs were long RP tachycardia (red arrows denoting P waves). **D and E.** The site of earliest atrial activation (vertical red line) of both SVTs was located in proximal CS. SVT1 was an AVRT using an epicardial PS-AP for retrograde conduction. SVT2 was a fast/slow AVNRT. **F.** Site of successful ablation.

The author prefers to start the EP study with parahisian pacing, followed by decremental RV pacing (**Figure 6.14A**). Typically, pacing starts at a CL 20-30 ms shorter than the sinus CL. After the mechanism of retrograde conduction at the longest pacing CL is determined (e.g. AVN, AP or fusion of AVN/AP), pacing CL

is gradually shortened at a pacing output that does not capture the HB/RBB. The operator should avoid using pacing output that captures HB/RBB during decremental pacing because when intermittent HB/RBB capture suddenly shortens the VA interval, it is very easy to miss subtle changes in atrial activation sequence (e.g. from retrograde slow pathway conduction to retrograde AP conduction). If atrial activation sequence suddenly changes, parahisian pacing at that pacing CL is performed immediately to determine if the new atrial activation sequence is mediated by AVN, AP or fusion. Then, the process repeats itself until VA block. In most of the cases, the operator should already have a good idea about the mechanism of each atrial activation sequence before induction of SVT.

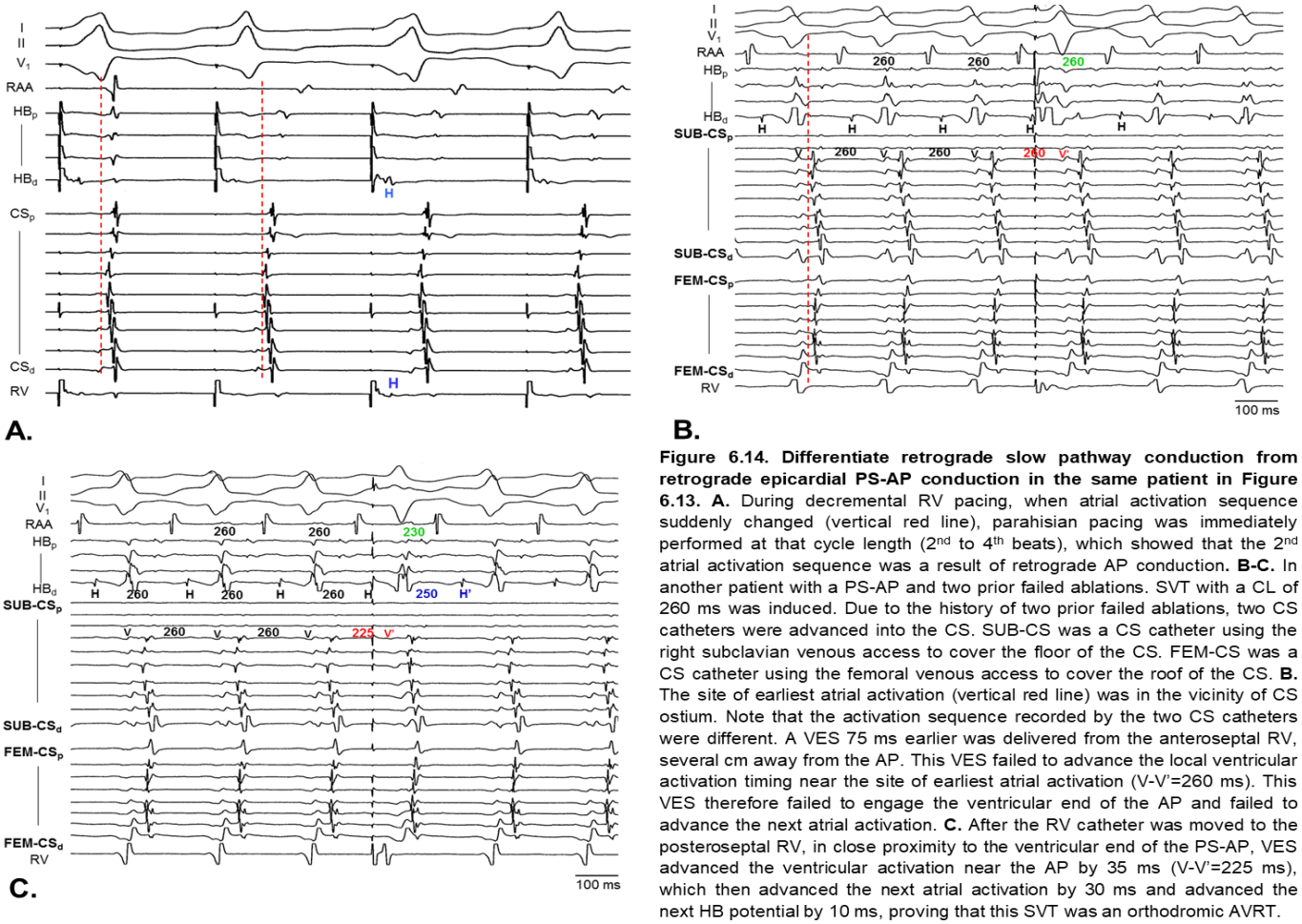


Figure 6.14. Differentiate retrograde slow pathway conduction from retrograde epicardial PS-AP conduction in the same patient in Figure 6.13. A. During decremental RV pacing, when atrial activation sequence suddenly changed (vertical red line), parahisian pacing was immediately performed at that cycle length (2nd to 4th beats), which showed that the 2nd atrial activation sequence was a result of retrograde AP conduction. **B-C.** In another patient with a PS-AP and two prior failed ablations. SVT with a CL of 260 ms was induced. Due to the history of two prior failed ablations, two CS catheters were advanced into the CS. SUB-CS was a CS catheter using the right subclavian venous access to cover the floor of the CS. FEM-CS was a CS catheter using the femoral venous access to cover the roof of the CS. **B.** The site of earliest atrial activation (vertical red line) was in the vicinity of CS ostium. Note that the activation sequence recorded by the two CS catheters were different. A VES 75 ms earlier was delivered from the anteroseptal RV, several cm away from the AP. This VES failed to advance the local ventricular activation timing near the site of earliest atrial activation (V-V'=260 ms). This VES therefore failed to engage the ventricular end of the AP and failed to advance the next atrial activation. **C.** After the RV catheter was moved to the posteroseptal RV, in close proximity to the ventricular end of the PS-AP, VES advanced the ventricular activation near the AP by 35 ms (V-V'=225 ms), which then advanced the next atrial activation by 30 ms and advanced the next HB potential by 10 ms, proving that this SVT was an orthodromic AVRT.

Regardless of the results of parahisian pacing, the most important step to verify the diagnosis is still the resetting or entrainment response to ventricular extra-stimuli (VES) or RV overdrive pacing during SVT. If only non-sustained SVT can be induced, the author prefers to perform parahisian pacing immediately after termination of SVT when all catheters are still in place. By comparison of the atrial activation between the SVT and parahisian pacing at various CL, it provides important clue to the mechanism of the non-sustained SVT. As discussed in **Chapter 4**, the most effective site to deliver VES or RV overdrive pacing is the basal posteroseptal RV in close proximity to the PS-AP (**Figure 6.14B-C**). By pacing the basal posteroseptal RV, AVRT can quickly be ruled in or ruled out in the vast majority of cases. In most of the incorrect diagnoses made by our referring electrophysiologists, resetting or overdriving pacing was uniformly delivered to the RV apex, not the basal posteroseptal RV. Most of the mistakes in differential diagnosis between AVNRT and PS-AP are rooted from misunderstanding of the term “PVC on His” or “His synchronized PVC”. The report often mistakenly states “PVC on His did not preexcite the next atrial activation, therefore the diagnosis of AVRT was excluded”. As discussed in **Chapter 4**, unless VES significantly advanced the local ventricular activation

near the AP but they still fail to advance the next atrial activation, the diagnosis of orthodromic AVRT cannot be excluded (**Figure 6.14B**). Dr. Jackman's approach to differential diagnosis of an SVT with the site of earliest atrial activation in the proximal CS is to deliver VES from the basal posteroseptal RV (near the presumed AP) to rule in or rule out AVRT. If AVRT is ruled out, VES are then delivered from the basal anteroseptal RV to rule in or rule out AVNRT.

An important caveat of all the diagnostic maneuver is that if the AP exhibits decremental conduction property, it may require a very early VES to reset the AVRT (**Figure 4.10**). Usually, a VES engaged the ventricular end of the PS-AP 20-30 ms prematurely is expected to advance the next atrial activation timing by approximately 10-20 ms. In the event that the PS-AP exhibits decremental conduction properties, it may cause a 20-30 ms delay over AP conduction, leading to no change in the next atrial activation timing. The resetting response appears to be negative and orthodromic AVRT may be mistakenly excluded. Similarly, the entrainment response may produce falsely long PPI to exclude AVRT. In this situation, it is very important to deliver VES to the basal posteroseptal RV (adjacent to the ventricular end of the AP) during the HB refractory period. *Very early* VES usually can reset or terminate the tachycardia and the diagnosis of orthodromic AVRT is then made (**Figure 4.10**). If the operator delivered decremental anteroseptal RV pacing at the beginning of the EP study, s/he should be detected that this AP exhibits decremental conduction properties.

Epicardial PS-AP (CS-epicardial LV connections)

There is a special type of PS-AP in which the PS-AP connects with the epicardial LV through the CS myocardial coat. This type of PS-AP is referred to as epicardial PS-AP in this chapter. Dr. Jackman named the CS myocardial coat covering the proximal segment of the middle cardiac vein (MCV) or posterior cardiac vein "CS myocardial extension, CSE" (*Circulation* 2002; 106:1362-1367; (**Figure 6.15**). Dr. Jackman proposed that one can view the entire CSE between the atrium and ventricle in the posteroseptal area as an epicardial PS-AP, which may have multiple connections with the atrium and ventricle. It can be very difficult to eliminate all the atrial-CSE or ventricular-CSE connections. The best ablation target is the CSE potential (equivalent to an AP-P) in the MCV or in the posterior coronary vein. The author usually teaches the EP fellows that this type of AP is like two Chinese/Japanese hand fans connected by a handle (**Figure 6.15**). Cutting the handle makes more sense

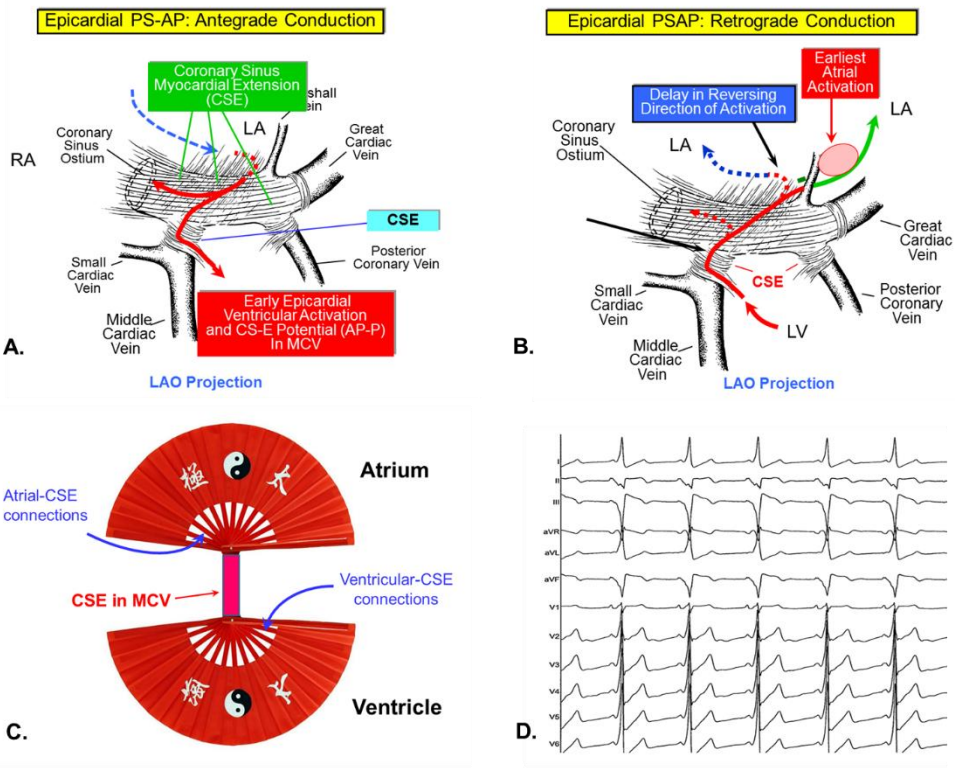


Figure 6.15. Epicardial PS-AP whose ventricular end is formed by connections between the CS muscle extension (CSE) and epicardial surface of the LV. A. Antegrade conduction. Activation wave front in the LA (curved blue line) enters the atrial end of the AP and uses CSE to conduct to both RV and LV. The site of AP-P (CSE potential) is usually located in the middle cardiac vein (MCV) or occasionally in the posterior coronary vein. **B. Retrograde conduction.** Retrograde AP conduction follows the sequence of epicardial LV, CSE and LA. At the CSE-LA junction, the wave front propagating septally has to take a sharp turn; the conduction velocity slows down (dotted blue line). On the contrary, the wave front propagates laterally does not have to slow down (green line). The site of earliest atrial activation over retrograde conduction of an epicardial PS-AP is therefore always lateral to the CSE-LA junction. If the site of earliest atrial activation is selected as the ablation target, the site of earliest atrial activation will move progressively toward the septum. **C.** An epicardial PS-AP has multiple connections to the atrium and ventricle. It is exceedingly difficult to eliminate all the atrial-CSE or ventricular-CSE connections. The best ablation target is the CSE potential (equivalent to an AP-P) which is often in the MCV. **D.** Typical pre-excitation pattern of an MCV-related epicardial PS-AP. Note that the delta wave was negative in all the inferior leads. Modified with permission from Circulation. 2002 Sep 10;106(11):1362-7.

than cutting each spline of the fan. The ECG of most manifest epicardial PS-APs shows a negative delta wave in lead II, III and aVF. In about 70% cases, the delta waves in lead II is steeply negative (**Figure 6.15D**) but this finding is only 70% sensitive and 70% specific. However, a PS-AP with a steeply negative delta wave in lead II should prompt the operator to consider an MCV-related PS-AP. In general, MCV contracts with the ventricle, regardless if it is connected with an AP or not. One cannot use MCV contraction to judge if this MCV is part of an epicardial PS-AP.

It is known that CS diverticulum is associated with epicardial PS-APs; however, most of the epicardial PS-APs occur in the absence of a CS diverticulum. In the presence of a CS diverticulum, the primary ablation target is the CSE potential (equivalent to an AP-P) in the neck of the diverticulum connecting to the floor of the CS. Sometimes, the neck is broad but it is still a much better target than the diffusely early ventricular potential inside the diverticulum. Another important pearl is that diverticulum almost always pushes the posterolateral branch of the right coronary artery away from the neck of the diverticulum. Based on our anecdotal experience, the OU-EP group no longer performs coronary angiogram before ablating an epicardial PS-AP if the ablation target (a sharp CSE potential or AP-P) is located in the neck of a diverticulum (see discussion below).

Mapping of Posteroseptal AP (PS-AP)

If the patient has a history of prior PS-AP ablation or the preexcitation pattern is consistent with a PS-AP, Dr. Jackman will start the procedure with a CS angiogram to delineate the major CS tributaries. After the HB catheter is positioned at the top of the triangle of Koch and records a stable HB potential, the LAO angle is adjusted to make the HB catheter en-face. In other words, the inter-atrial septum is facing directly at the operator for future anatomical reference. The balloon-occlusion technique is then used to obtain the CS angiogram, focusing on the area from the CS ostium to Veussen valve. Dr. Jackman prefers to keep the balloon in the 1:30 to 2:00 position along the mitral annulus in the LAO projection to allow the best visualization of the MCV and proximal CS. For a concealed epicardial PS-AP, if preliminary mapping suggests that the atrial activation is early along the floor of the CS, Dr. Jackman will obtain a CS angiogram before more detailed mapping.

After the diagnosis of AVRT is verified, Dr. Jackman starts mapping from lateral tricuspid annulus toward posteroseptal annulus, followed by mapping from the anteroseptal area toward the posteroseptal area, He likes to use the analogy of reading a chest x-ray film by examining the bony structures first, before paying full attention to the pulmonary parenchyma and vasculature. Mapping of any arrhythmia should start from site distant from the “presumed” site of interest to avoid the mistake of “not seeing the forest for the trees”.

As mentioned above, Dr. Jackman never mapped AP conduction by pacing the RV apex. Dr. Jackman had received many similar phone calls from desperate electrophysiologists struggling with epicardial PS-AP ablations. Uniformly, the site of shortest VA interval or VA fusion during RV apex pacing was selected as the ablation target. VA fusion misled the operator to targets sites lateral to the atrial end of the PS-AP. During ablation, the site of earliest atrial activation kept moving toward the septum (**Figure 6.15B**); the desperate electrophysiologists usually ask Dr. Jackman’s advice on “how to ablate an AP with multiple atrial connections “or “how to ablate multiple APs”. This problem is universally caused by a poor choice of pacing site that obscured the true ablation target.

Mapping a posteroseptal AP, Dr. Jackman first chooses a pacing site that provides the best AV or VA separation. In the vast majority of PS-APs, the ventricular end of the AP is situated more septal than the atrial end. While Dr. Jackman prefers to deliver ventricular stimuli to reset the SVT from the basal posteroseptal RV (near the ventricular end of the AP) to quickly engage in retrograde AP conduction, pacing basal posteroseptal RV is often a poor choice for mapping due to very short VA interval or VA fusion it produces

(**Figure 6.16**). In a small minority of patients in whom the ventricular end of the AP is more lateral than the atrial end, pacing posteroseptal RV produces longer VA intervals.

Mapping a PS-AP, Dr. Jackman starts with mapping the tricuspid annulus and CS ostium, followed by mapping the roof of CS from the posterolateral CS toward the roof of CS ostium and then the floor of the CS in a similar manner. The CS tributaries, including the MCV and the posterior or posterolateral coronary vein are mapped as well. Of note, Dr. Jackman prefers to use the subclavian or internal jugular venous access for the CS catheter in patients with a PS-AP as well as slow/slow or fast/slow AVNRT because CS myocardium is part of the reentrant circuits of these arrhythmias. Notably, activation sequence of the roof and floor of CS are different because CS is essential the 5th chamber of the heart (**Figure 6.14C**). For a PS-AP, subclavian or jugular venous access allows the CS catheter to touch the floor of CS, providing important clues to the operator if the timing of the floor of the CS is earlier than that of the roof. When the CS catheter comes from the femoral vein, it tends to touch the roof of the CS and misses important small potentials on the floor of the CS or at the orifice of the CS tributaries. For cases such as VT or AF, the venous access where the CS catheter comes from is not important.

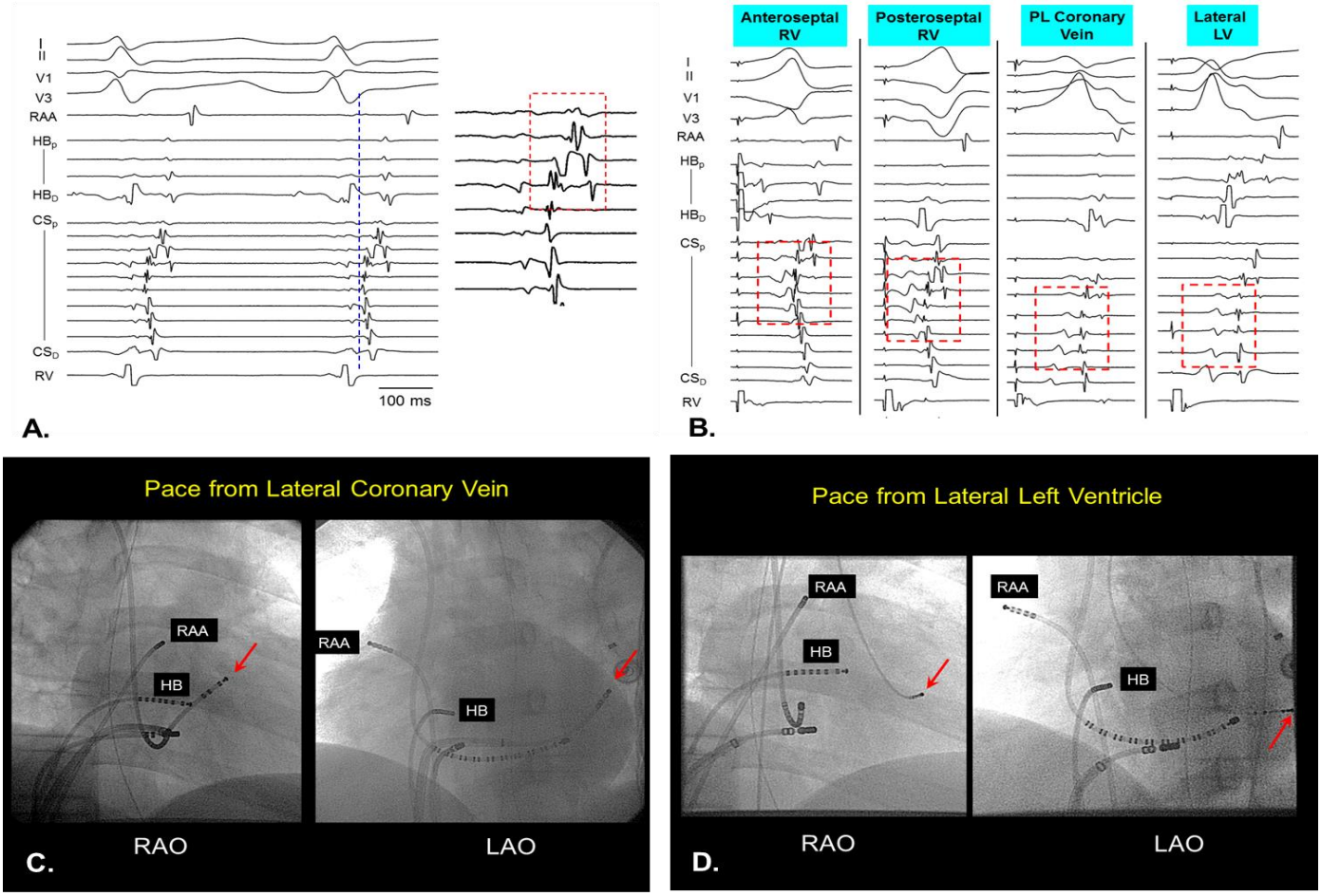


Figure 6.16. Selection of pacing site for a PS-AP. A. In AVRT, site of earliest atrial activation was in the proximal CS. Note that prior ablation was septal to the site of earliest atrial activation, creating double potentials highlighted in the dotted red box (inset). That ablation target was selected during RV apical pacing, leading to a wide area along the posteroseptal area showing VA fusion. **B.** Pacing from posterolateral coronary vein and LV lateral wall produced the best VA separation. Position of pacing catheter in the posterolateral coronary vein and basal lateral LV are shown in **C** and **D** respectively.

If the patient has a history of multiple prior ablation failure to treat a PS-AP or atypical AVNRT, the OU-EP group sometimes position two CS catheters, one through the subclavian or internal jugular vein, the other through the femoral vein. This approach is certainly an expensive one but for patients who have had multiple failed ablation procedures before, one CS catheter covering the activation timing of the roof of the CS and the other catheter covering the floor of the CS provide important insight into the mechanism as well

as the ideal ablation target. **Figure 6.17** illustrates the catheter position of a patient with two failed ablation attempts (one under the diagnosis of PS-AP, the other atypical AVNRT). By pacing from the posterolateral coronary vein and positioning two CS catheters, the site of earliest atrial activation was very easy to localize; the first RF application quickly eliminated the PS-AP conduction and AVRT. This figure also illustrates that the activation sequence of the floor and roof of the CS was different.

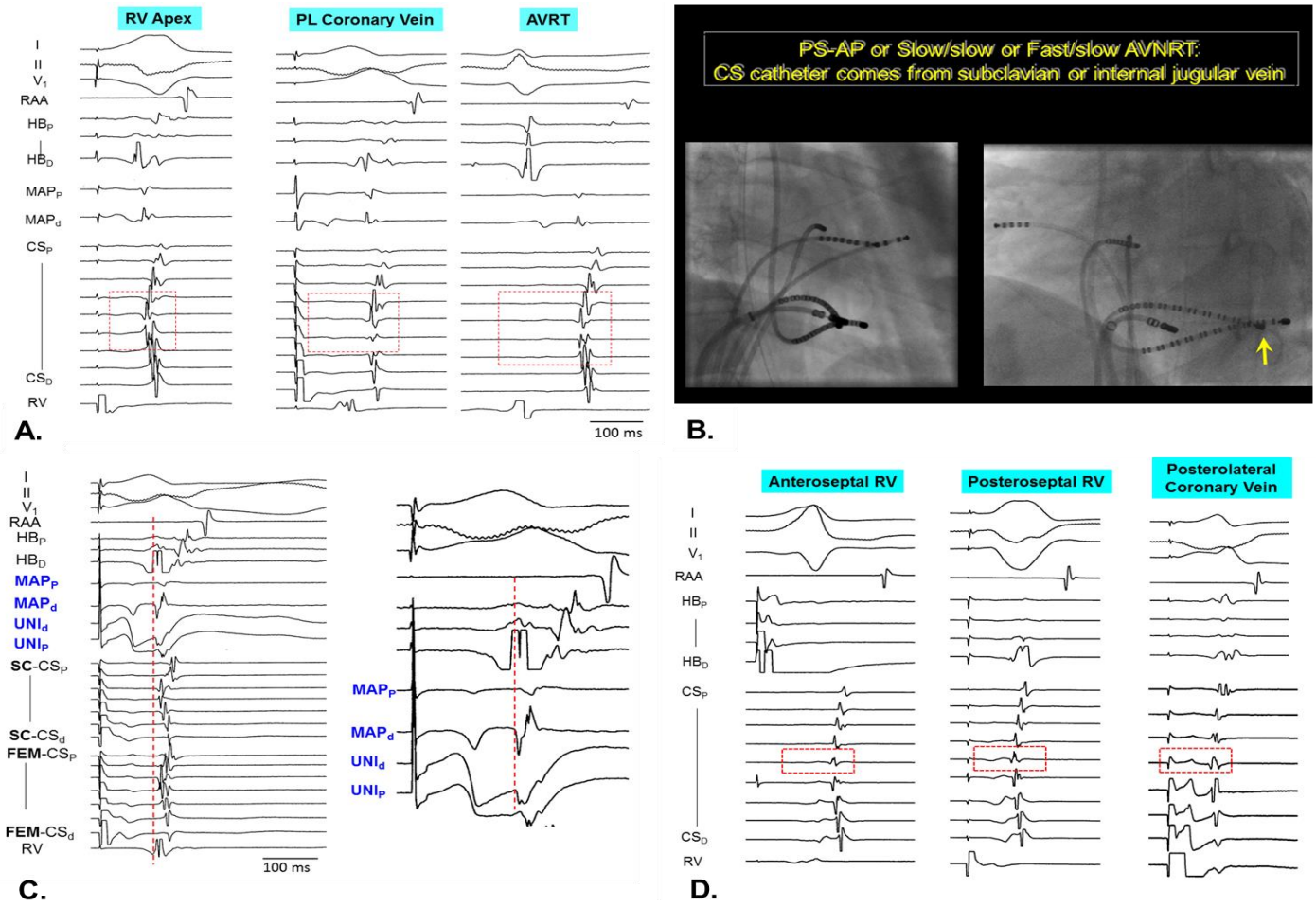


Figure 6.17. A patient with a concealed posteroseptal AP after two prior failed ablations. **A.** Differential pacing clearly showed much better VA separation by pacing from the posterolateral coronary vein (middle panel) than RV apex pacing (**left panel**). AVRT produced a long VA interval as well **(right panel**). **B.** Two CS catheters, one from femoral vein and the other from right subclavian vein, were positioned in the CS to record the EGM of the roof and floor of the CS, respectively. Left ventricular pacing was delivered from the posterolateral coronary vein (yellow arrow). **C.** With the help of widely separated VA intervals by coronary vein pacing, the site of earliest atrial activation (dotted red line) was easily identified. RF application at this site eliminated AP conduction immediately. Note that the distal unipolar EGM showed a QS pattern at the site recording earliest atrial activation. In addition, the CS activation sequence recorded by the two CS catheter were different. **D.** In another patient with a concealed epicardial PS-AP, pacing from the posterolateral coronary vein provided the best VA separation.

To ablate a posteroseptal AP, operators often have to make a tough decision to map the mitral annulus first or map the coronary sinus first. Dr. Jackman's rule of thumb is as follows. If the atrial timing during retrograde AP conduction along the roof is earlier than that along the floor, it suggests that the AP is located along the mitral annulus. Dr. Jackman will then proceed with trans-septal puncture to map the mitral annulus. If the atrial timing on the floor of the CS is earlier than that on the roof, it suggests that this AP uses the CS muscle extension (CSE) as part of the AP conduction (an epicardial PS-AP). High-density mapping of the floor of CS and its tributaries will be performed. To be specific, the earlier "atrial" timing along the floor of the CS is indeed CSE potential, not atrial activation. Over retrograde AP conduction, the atrium is not activated until the CSE has been activated.

On a lucky day, the epicardial PS-AP uses the MCV to connect with the ventricle. A discrete AP-P (CSE) can be found in the MCV. In this case, the target (an isolated AP-P), can be identified by mapping over

antegrade or retrograde AP because the ventricular end of the AP can be accessed from the MCV (**Figure 6.18, 6.19**). Differentiating an LA potential from a CSE can be a daunting task but it often plays a critical role in selecting ablation targets for epicardial PS-APs. In essence, Dr. Jackman uses the same technique as he employs to verify an AP-P to differentiate a CSE potential (equivalent of an AP-P) from an LA or LV potential. Epicardial PS-APs using MCV as part of the connection between LA and LV are indeed the “easiest” epicardial PS-AP to map and ablate because the target is usually very discrete (a sharp AP-P) in the MCV).

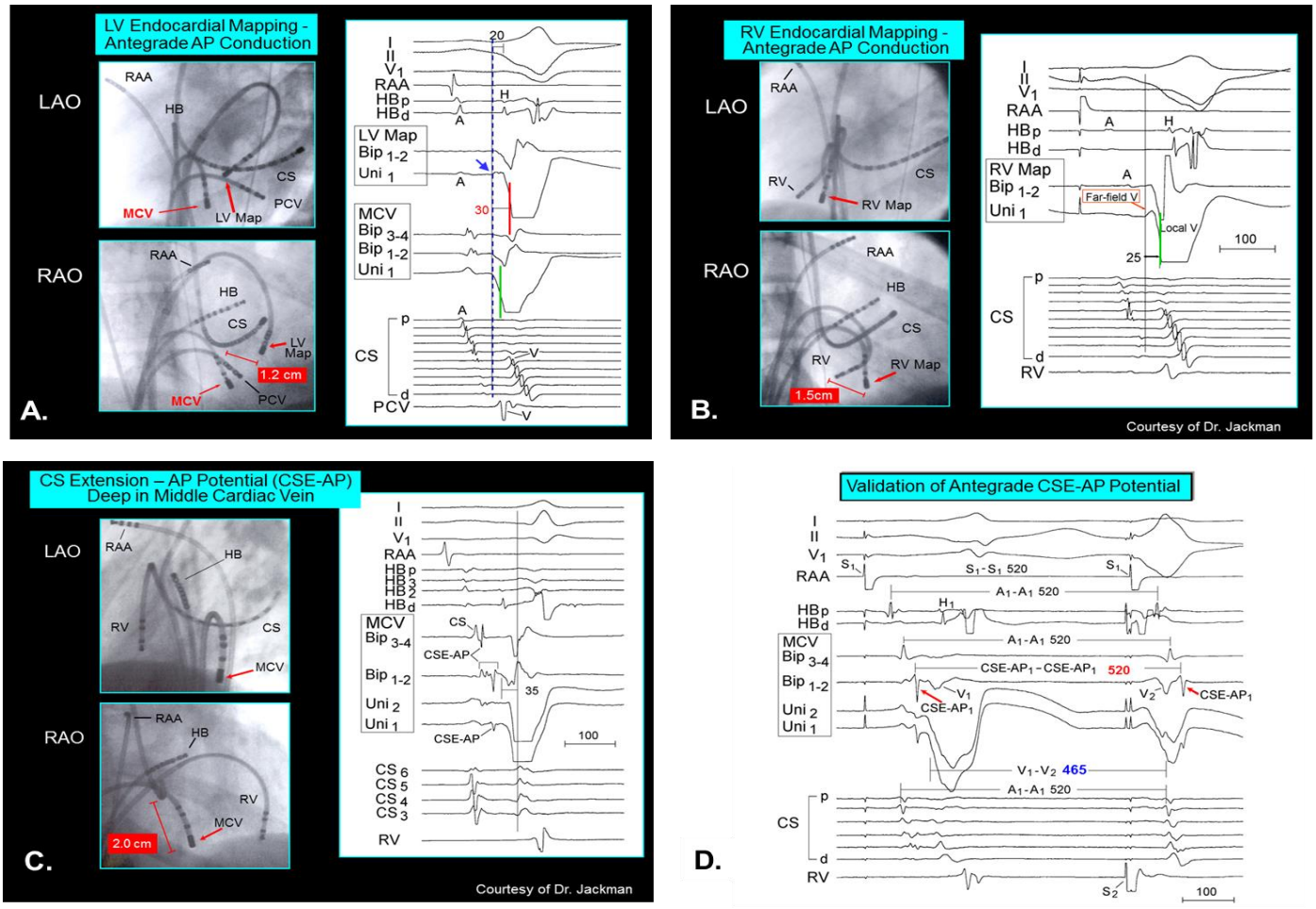


Figure 6.18. The ventricular end of an epicardial PS-AP formed by CSE-ventricular connections. A. Late timing of endocardial ventricular activation during antegrade AP conduction. An early far-field ventricular activation (blue arrow and blue vertical line) was recorded 20 ms before the onset of the delta wave. Earliest endocardial LV activation (vertical red line, rapid down stroke in the unipolar EGM) was recorded 30 ms after the onset of the far-field potential, 1.2 cm from the posteroseptal mitral annulus (LV Map catheter in radiographs). Earlier ventricular activation was recorded epicardially from deep within the MCV (MCV-Uni1 EGMs, vertical green line). **B,** Earliest endocardial RV activation (Local V, vertical green line) was recorded 25 ms after the onset of the far-field potential, 1.5 cm from the posteroseptal tricuspid annulus (RV Map catheter in radiographs). **C.** Recording of antegrade CS myocardial extension (CSE) activation potentials along a 2-cm length of the MCV. In this location, CSE is equivalent to an AP potential. **D. Validation of the antegrade CSE potential recorded from the MCV.** During RA pacing at CL of 520 ms (S1), a late ventricular extra-stimulus (S2) advanced the local ventricular potential in the MCV electrograms by 55 ms (V1V2 = 465 ms) without altering the timing or morphology of the CSE1 potential, dissociating CSE1 from local ventricular activation. **E.** An earlier S2 advanced the CSE potential by 25 ms (CSE1-CSE2: 495 ms) without altering the timing or morphology of the surrounding atrial potentials (A1A1 = 520 ms in HB, MCVBip3–4). Modified with permission from Circulation. 2002 Sep 10;106(11):1362-7.

If the CSE/atrial timing is early on the floor of the CS but *late* in the MCV or posterolateral coronary vein, ablation of this type of AP is usually very difficult because the CSE-ventricular connection may be very broad. In this situation, the author prefers to map the AP conduction during ventricular pacing. Because this type of AP uses CSE to connect to the LV epicardium, the ventricular end of the AP can be distant from the mitral annulus or CS. Mapping of this type of AP during antegrade AP conduction often identifies far-field ventricular activation that may be diffusely early in a large area and difficult to reach. It may be easier to identify and ablate segments of the AP connecting to the CS or LA, which is less likely to be far field and may be easier to reach (**Figure 6.15**). However, the atrial connection may be multi-focal and may require ablation of multiple adjacent sites. Fortunately, it is rare to see an epicardial PS-AP without retrograde AP conduction; thus mapping over retrograde AP conduction can always be performed.

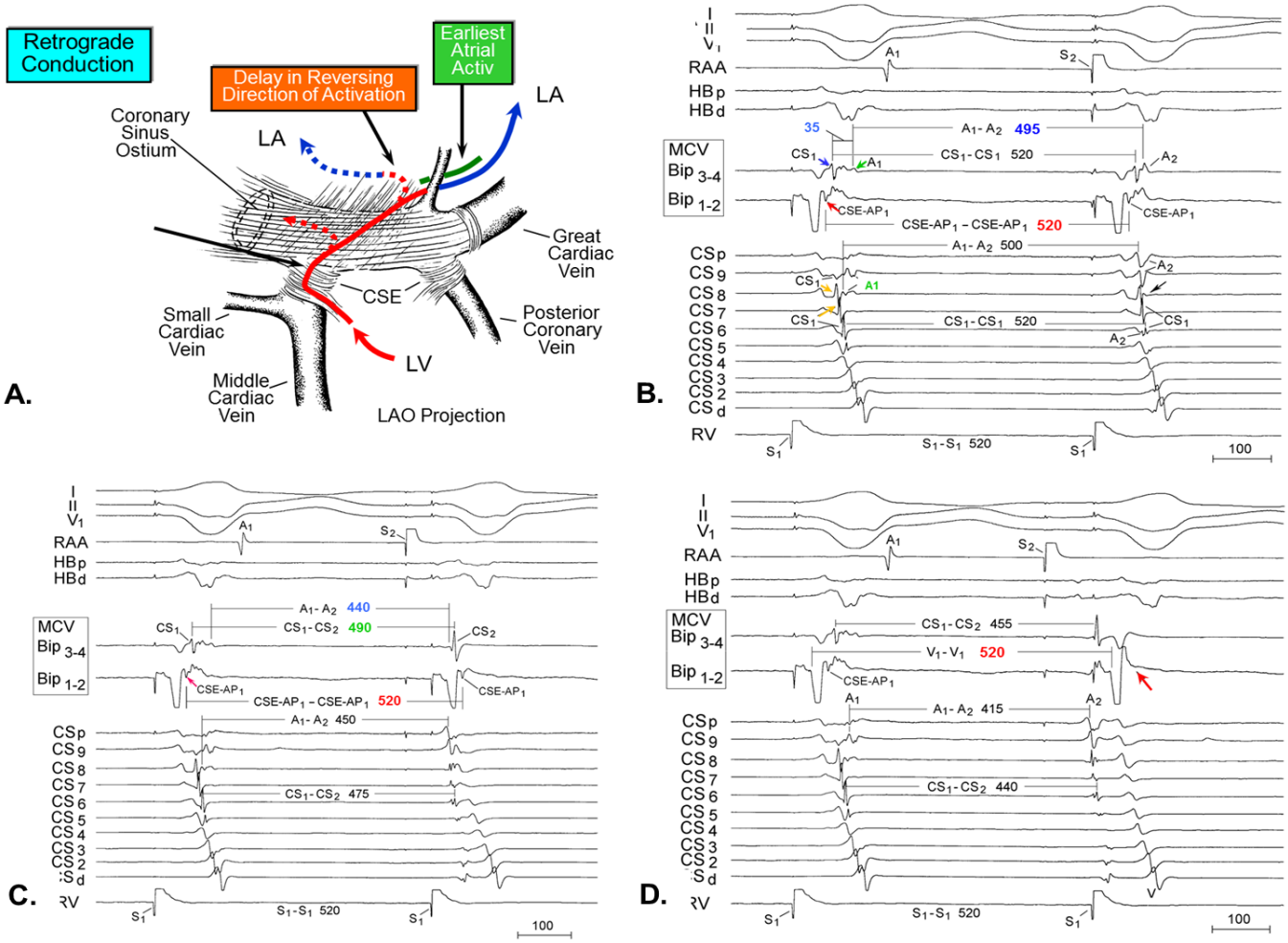


Figure 6.19. Validation of retrograde CSE potential (AP-P). A, Schematic representation of an epicardial PS-AP using CS-ventricular connections as part of the retrograde AP conduction. **B.** Left complex. During RV pacing at CL of 520 ms (S1), the earliest retrograde potential (red arrow) was recorded from the MCV (CSE1 in MCV-Bip1–2). This was followed by a CS myocardial potential (blue arrow) at the orifice of the MCV (CS1 in MCV-Bip3–4). Leftward propagation of CS myocardial potentials (CS1 in electrograms CS9 to CS7, brown arrow) preceded the left atrial potential (A1, green arrow). Because of the distance between the MCV and the left atrial activation site and the delay associated with reversing direction, A1 recorded from the proximal MCV is relatively late (CS1-A1=35 ms in MCV-Bip3–4). A late atrial extra-stimulus (S2) advances atrial activation by 25 ms in the MCV-Bip3–4 EGM (A1-A2=495 ms) without affecting the timing of CS1 potentials, dissociating CS1 from local atrial activation. **C.** An earlier S2 advances both atrial activation (A1-A2=440 ms) and CS myocardial coat activation (CS1-CS2=490 ms) in MCV-Bip3–4 without altering the timing or morphology of the CSE1 potential, dissociating CSE1 from local atrial activation and activation of the CS myocardial coat connecting to the left atrium. **D.** An earlier S2 advances CSE1, resulting in loss of the CSE1 potential (red arrow), without altering the timing or morphology of the local ventricular potential, dissociating CSE1 from local ventricular activation. Modified with permission from Circulation. 2002 Sep 10;106(11):1362-7.

Ablation inside an MCV can be technically challenging. An irrigated tip catheter should be used. The tip electrode should be advance as deep as possible but the distal unipolar electrode (UNI-1) still records a

CSE potential (AP-P equivalent). Dr. Jackman prefers to deliver the first RF application at a site where UNI-1 records a small CSE potential but UNI-2 records a larger CSE potential. The reason the first RF application is slightly deeper than the ideal target is because it is may be difficult to re-advance the catheter into the MCV after ablation. Dr. Jackman therefore prefers to start deeper and gradually pull the tip electrode back to where the ideal target should be and ablate there. RF applications starts at about 15 watts and very slowly titrates the power up. As soon as AP conduction is blocked, power is maintained at the same level as long as possible (60-90 seconds). Any increase in impedance (as little as 2 ohms) should prompted the operator to lower the power or stop ablation to avoid tissue desiccation that causes the tip of the catheter to be stuck in the MCV.

Risk of coronary artery injury

Ablating the floor of CS always carries a significant risk of injuring the posterolateral branch of the right coronary artery (RCA) or the equivalent branch from the circumflex artery if the coronary artery circulation is left dominant. The posterolateral branch of the RCA typically makes an upward loop toward the floor of CS. However, the location of the coronary artery in the posteroseptal pyramidal space varies greatly among patients. Sites with a dangerously short distance (<5 mm) between the coronary artery and the floor of CS can be located anywhere from the floor of the CS ostium to the orifice of the posterior coronary vein (*Circulation Arrhythmia and EP*, 2014;7(1):113-119) (**Figure 6.20**). Therefore, ablation anywhere along the floor of proximal CS carries a significant risk of coronary artery injury.

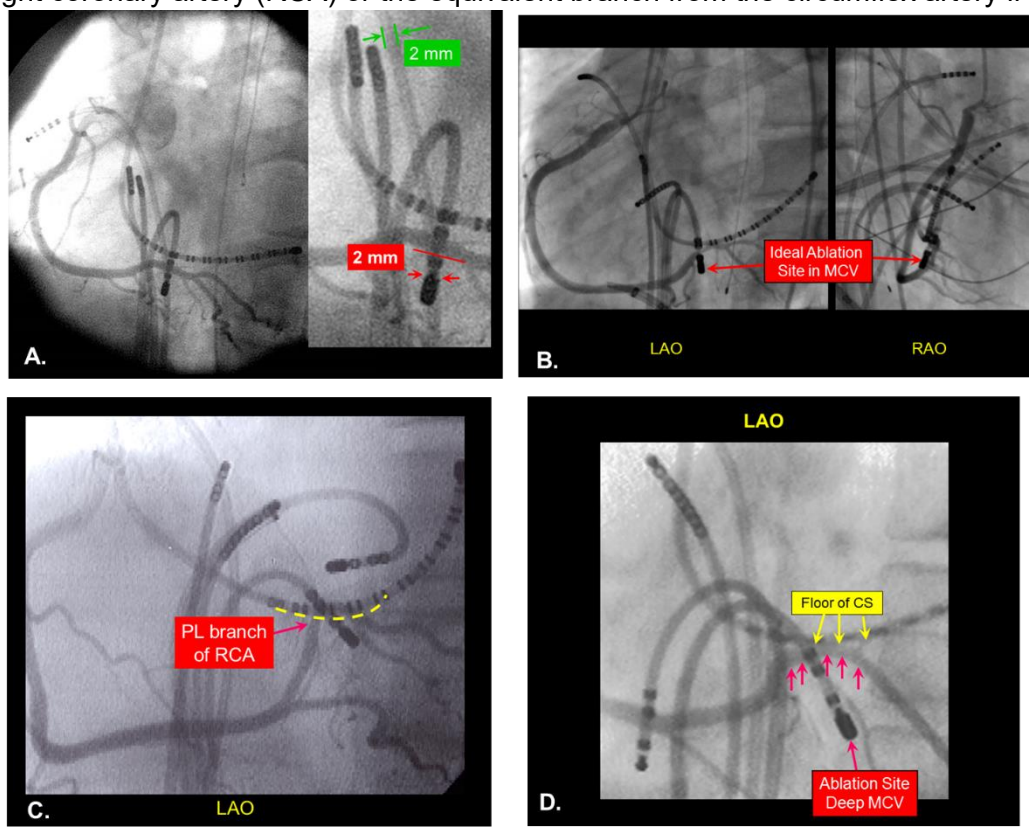


Figure 6.20. Short distance between coronary artery and the floor of proximal CS. A. The ablation catheter was positioned at the ideal ablation site, recording a sharp CSE potential but the proximal edge of the distal ablation electrode (red arrows) was 2 mm away from a large posterolateral branch of the right coronary artery (red line). The RA catheter (6 Fr., 2 mm wide) was used as the reference to measure the distance between the target and artery. Cryoablation successfully eliminated AP conduction. **B.** Another example of a short distance between the tip ablation electrode and a large coronary branch. **C.** Although the tip electrode was not very close to the coronary artery branch, this artery courses along the floor of the proximal CS (dotted yellow line). **D.** In another patient, the tip ablation electrode was deep in the MCV, distant from a large coronary artery branch (red arrows); RF applications delivered to this site quickly eliminated AP conduction without coronary injury. However, the floor of proximal CS was in close proximity to the coronary artery. The radiographs in **C and D** illustrate the danger of ablating the floor of the proximal CS without knowing the proximity to major coronary artery branches.

Operators must be aware that most of the coronary artery injuries caused by ablation in the posteroseptal pyramidal space do *not* lead to ST-T changes in ECG. The most striking example that Dr. Jackman always talks about is a young child referred to him for ablation of a posteroseptal AP. Before ablation, a coronary angiogram showed 90% stenosis of a large posterolateral branch. The pediatric electrophysiologist who did the first ablation procedure was on the scene and swore that no ablation on the floor of the CS was ever done. This is indeed a quite common scenario in which the operator is not aware that the ablation catheter is inside the CS ostium and in contact with the floor of the CS. In the OU-EP laboratory, the rule is to obtain a coronary angiogram if ablation target is on the floor of the CS or CS ostium. Leaving the tip ablation electrode at the ideal site of ablation, a right coronary angiogram is performed to measure the distance between the coronary artery branch and the ablation catheter from multiple planes and angles. If it is found

that coronary circulation is left-dominant, a left coronary angiogram will then be performed. We use a 6 Fr. RA catheter as the reference of distance (the width of a 6 Fr. catheter is 0.033mm x 6=2mm). If any part of the tip electrode of the ablation catheter is within 5 mm from a major coronary artery branch, RF current is deemed not safe. Cryoablation will be performed (**Figure 6.21**). Operators must pay close attention to the proximal edge of the tip electrode and ensure that it stays at least 5 mm away from an artery because electrode edge has the highest current density (edge effect). An artery is more likely to be injured if it is close to the edge than the tip of the ablation electrode. Another important assessment is to determine if the tip electrode is pushing against the artery; the incidence of severe arterial injury is much higher in this situation because the coronary arterial branch is usually sandwiched between the MCV and LV epicardium. Ablation catheter in the MCV tends to push against the artery and subject the artery to ablation injury.

One may think that MCV is just slightly larger than the width of the cryocatheter; cryoablation of an MCV-related PS-AP must be easy and quick. In fact, it often requires multiple 4-minute freezes to achieve permanent conduction block. It was very common to see AP conduction returns after 60 minutes. The recurrence rate is about 20% as well. For this reason, the OU-EP group jumped on the bandwagon of cryoballoon ablation for AF very late because of our experience in cryoablation of epicardial PS-APs.

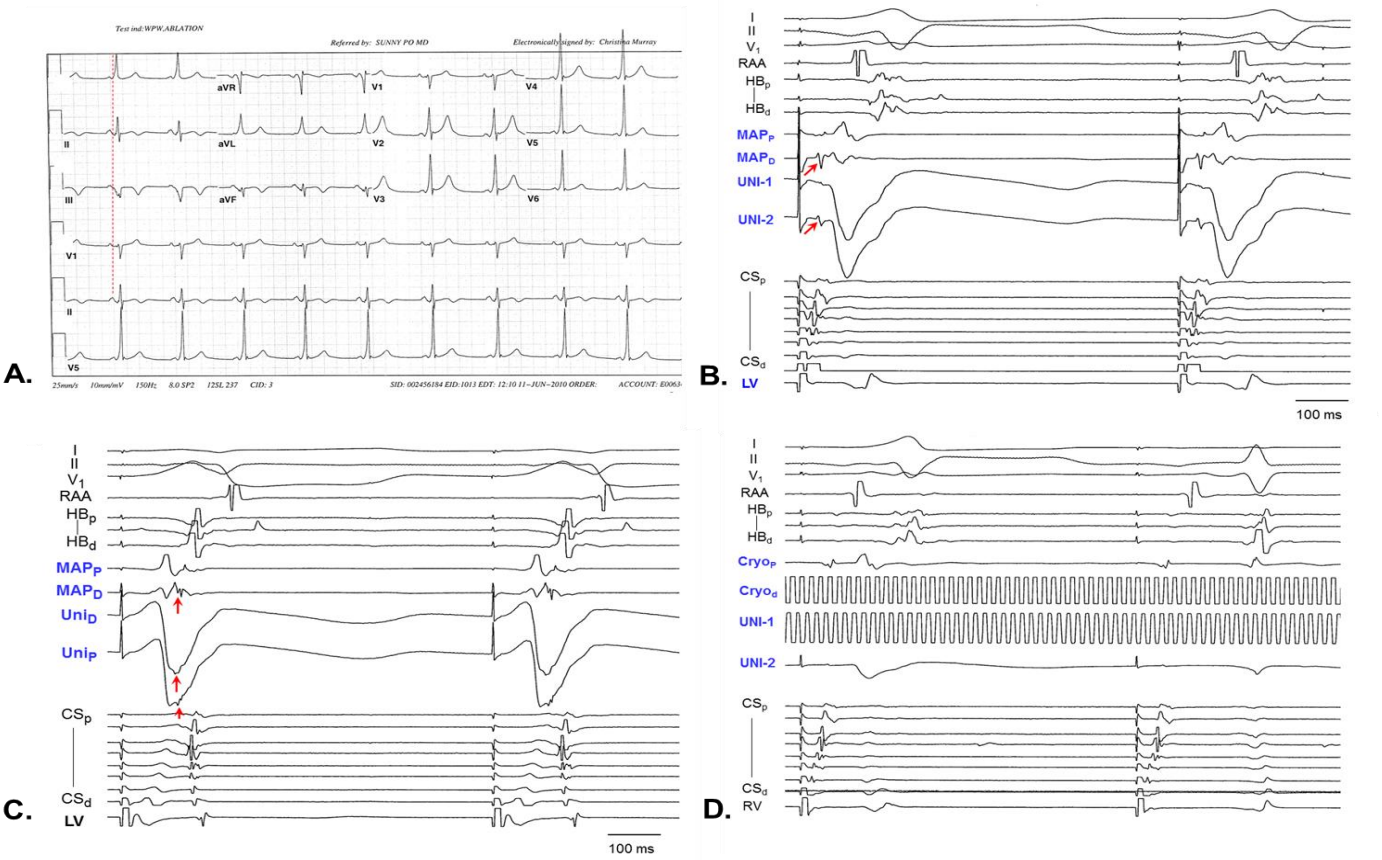


Figure 6.21. **A patient with a manifest MCV-related epicardial PS-AP. A.** ECG showed preexcitation (vertical red line: onset of delta waves). **B.** A sharp AP potential (red arrow) was recorded on the mapping catheter positioned in the MCV during antegrade AP conduction. **C.** During retrograde AP conduction, the same AP potential was recorded as well. This sharp potential was proven to be an AP potential using the same method described earlier in this chapter (not shown here). **D.** A large posterolateral branch of the right coronary artery was in close proximity (<2 mm) to the ideal ablation site. Cryoablation was applied to the MCV and eliminated both antegrade and retrograde AP conduction.

MCV may connect to the floor of CS at a site very close to CS ostium (**Figure 6.22A**). It is not uncommon that during ablation of AVN slow pathway or cavo-tricuspid isthmus, the operator is not aware that the ablation catheter has fallen into the MCV. Ablation inside the MCV can cause severe coronary artery injury. In the OU-EP laboratory, if the ablation catheter is pointing septally and downward, the operator will apply gentle counterclockwise torque to the ablation catheter in the LAO projection. If the catheter falls into the RA without resistance, the tip of the catheter is not inside the MCV. However, if it takes substantially more

force to rotate the catheter to the RA, the tip of the catheter is stuck in the MCV and RF current should not be delivered there without a coronary angiogram.

One day, the author received a call from a colleague to deal with “repeated immediate impedance rise during slow pathway ablation”. The position of the ablation catheter from both the RAO and LAO view appeared to be at the inferior triangle of Koch. When the operator was asked to rotate the ablation catheter in a counterclockwise direction, the tip of the catheter was stuck there, indicating that the tip of the ablation catheter accidentally fell into the MCV. A CS angiogram showed that MCV originated from the floor of the CS ostium (similar to **Figure 6.22A**). Fortunately, there was no coronary artery in that area. This story emphasizes the importance of using this “counterclockwise rotation” technique before RF ablation in the posteroseptal area for any type of arrhythmia. Fast anatomical mapping of the CS ostium and floor of proximal CS can help avoid this mistake.

On rare occasion, after extensive ablation, the last piece of AP-atrial connection is in or lateral to the cavo-tricuspid isthmus, probably through the small cardiac vein which originates from the MCV or proximal CS and courses toward lateral tricuspid annulus (**Figure 6.22B**). A typical example is that the MCV could not be accessed due to stenosis caused by prior ablation. An epicardial PS-AP had to be ablated by eliminating all connections to the LA, RA and CS. After exhaustive ablation in the CS and at CS ostium, the VA interval was substantially prolonged but AP conduction could not be eliminated. The site of earliest atrial activation was nowhere to be found in the posteroseptal area or in the CS. In this scenario, mapping along the tricuspid annulus may identifies the last connection to the atrium, possibly through the small cardiac vein.

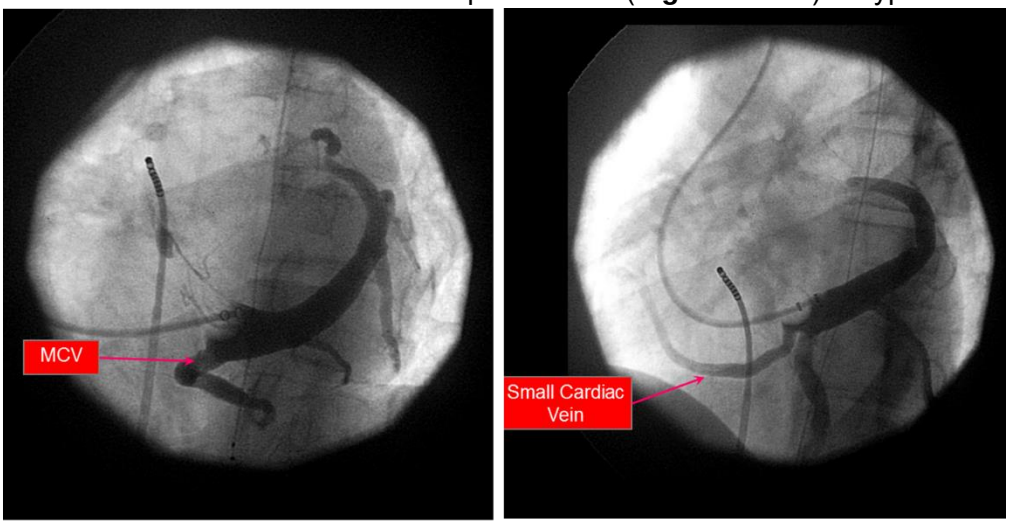


Figure 6.22. Variations of CS tributaries. **A.** MCV orifice is located at the floor of the CS ostium. When slow pathway ablation is performed, the ablation catheter can easily fall into MCV and injure a coronary artery branch. **B.** Occasionally, an epicardial PS-AP connects with the RA free wall through small cardiac vein.

Chapter 7:

Ablation of AVNRT

Although Dr. Jackman's landmark paper describing slow pathway ablation in 1992 (NJEM, 1992 Jul 30;327(5):313-8) led to high ablation success rates with a low incidence of AV block, it had the unfortunate effect of nearly completely halting research into the AVNRT reentrant circuit. The location of the antegrade and retrograde limb of the AVNRT reentrant circuit as well as where the two limbs connect remain poorly understood. In the past two decades, Dr. Jackman continued to painstakingly investigate the AVNRT reentrant circuit, leading to his working hypothesis that the antegrade and retrograde limb of the reentrant circuit connect in the CS and left atrium. In other words, the left atrium and CS are integral components of the reentrant circuit. For this reason, an upper common pathway, manifesting as VA block during AVNRT, is rarely observed. The AVNRT reentrant circuits that Dr. Jackman has proposed are based on the results of anatomical evidence, as well as activation mapping and resetting responses. The latter involves delivery of single atrial extra-stimuli from various sites during AVNRT. The latest extra-stimulus that can reset AVNRT indicates that this site is in close proximity to the reentrant circuit (see discussion later in this chapter).

To date, Dr. Jackman's working hypothesis has yet to be verified by high-density mapping. This chapter is not intended to provide a detailed description of the hypothesized circuits of various forms of AVNRT. Detailed illustration and explanation of Dr. Jackman's working hypothesis can be found in the book "Cardiac Electrophysiology: from Cell to Bedside", edited by Drs. Jalife and Zipes. Whether one adopts this working hypothesis or not, the concepts and pacing maneuvers described herein are highly effective when AVNRT ablation is challenging, which usually stems from unusual locations of the antegrade and/or retrograde limb of the reentrant circuit located in the CS or LA. For example, every electrophysiologist has a similar nightmare that slow/fast AVNRT remains inducible after a "scorch the earth" type of ablation in the triangle of Koch and CS ostium. However, the last strand of the antegrade slow pathway participating in slow/fast AVNRT is nowhere to be found. The resetting technique described in this chapter is highly effective in identifying the residual strand of the antegrade slow pathway and avoiding ablating progressively superior in the triangle of Koch.

> *Some of the observations that Dr. Jackman made over the past 3 decades appear to contradict the conventional wisdom. Electrophysiologists should be aware that using His bundle (HB) catheters with closely-spaced electrodes (**Figure 1.16B**), pacing from the parahisian area (not RV apex) and anesthesia/deep sedation may all contribute to many of Dr. Jackman's unique observations.*

Fast pathway and slow pathways of the AV node

A salient paper of Dr. Anton Becker (Circulation. 1998;97(2):188-93) reported that there are two posterior (or inferior) extensions of the human compact AV node (**Figure 7.1A**). In 21 human hearts, 13 hearts showed inferior extensions on both the right and left sides; seven hearts had a single right inferior extension (RIE); and one heart showed a single left inferior extension (LIE). An RIE was present in 20 of 21 hearts. In 16 of these 20 hearts, the RIE continued to the level of the coronary sinus ostium.

Based on Dr. Becker's report and Dr. Jackman's own observations, Dr. Jackman proposed that there are two major slow pathways participating in AVNRT: RIE and LIE, both of which connect to the RA and LA through the proximal CS (**Figure 7.1B**). Both RIE and LIE can participate in AVNRT and serve as either the antegrade or retrograde limb of the reentrant circuit. During retrograde slow pathway conduction over the LIE, the site of earliest atrial activation is located along the roof of the proximal CS (2-4 cm distal to the ostium; **Figure 7.1B**). During retrograde slow pathway conduction over the RIE, the site of earliest atrial activation is located in the inferior triangle of Koch and the lower edge of the CS ostium. Although

retrograde LIE and RIE conduction activates the CS ostium differently, they activate the LA in a very similar way (septal to lateral).

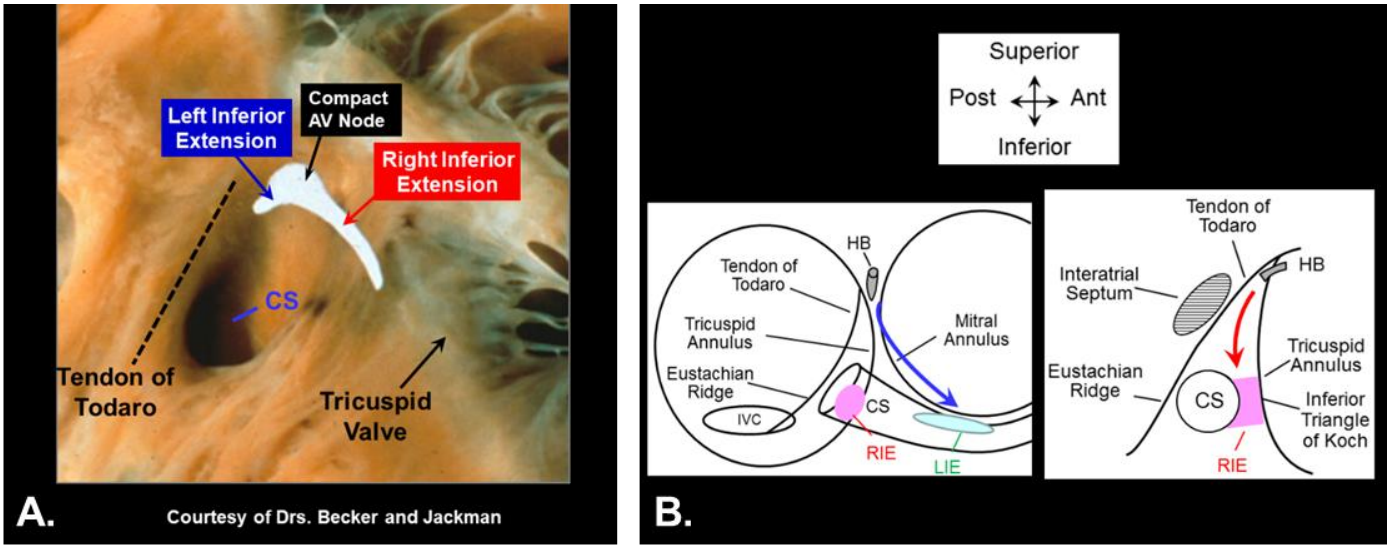


Figure 7.1. Left inferior extension (LIE) and right inferior extension (RIE) of the AV node. A. Because the LIE "dives" into the CS, the LIE appears short from this view. The RIE courses inferiorly along the triangle of Koch. **B. Retrograde conduction over LIE and RIE.** The atrial component of the LIE is located along the roof of proximal CS, 2-4 cm distal to the CS ostium. The atrial component of the RIE is located in the CS ostium as well as the area between the tricuspid annulus and CS ostium. *Modified with permission from: Inoue S, Becker AE. Circulation. 1998 Jan 20;97(2):188-93.*

Retrograde conduction over the AV nodal fast pathway

Earlier studies using catheters with widely-spaced electrodes recorded far-field potentials from a large area along the inter-atrial septum. The earliest atrial activation over retrograde fast pathway conduction was recorded in the same widely-spaced bipolar electrogram (EGM) that recorded the HB potential, leading to the conclusion that earliest atrial activation was recorded within the triangle of Koch. When Dr. Jackman used closely-spaced electrode catheters to investigate retrograde fast pathway conduction, the site of earliest retrograde atrial activation was actually not in the triangle of Koch but rather located posterior to the tendon of Todaro, 5-10 mm inferior and posterior to the site recording the most proximal HB potential (**Figure 7.2**). Atrial activation timing at this site usually precedes the atrial activation at the site recording the proximal HB potential by approximately 10 ms (**Figure 7.2B).** One can envision that this site is between the HB and fossa ovalis. Dr. Jackman defines retrograde conduction as occurring over the fast pathway when earliest retrograde atrial activation is recorded behind the triangle of Koch, 5-10 mm inferior and posterior to the site recording the most proximal HB potential, *regardless of the HA interval*. If operators map retrograde fast pathway conduction during AVNRT, a short VA interval may preclude accurate assessment of the site of earliest atrial activation. In this scenario, Dr. Jackman would deliver single ventricular extra-stimuli to separate the local ventricular potential from the atrial potential (**Figure 7.2B**).

To determine whether the mapping catheter is located posterior to the tendon of Todaro (outside of the triangle of Koch), the HB catheter needs to record a stable HB potential to ensure that the HB catheter is laid against the septum. Then, operators can set the angle of the fluoroscope in the LAO projection so that the catheter recording a HB potential is perpendicular to the plane (en face) of the fluoroscopic image (**Figure 7.2A**). When the mapping catheter is oriented leftward of the His bundle catheter in the LAO projection, the mapping catheter is posterior to the tendon of Todaro (**Figure 7.2A**) because the tendon of Todaro no longer pushes the mapping catheter rightward. At the fast pathway site, the local ventricular potential is very small (**Figure 7.2B**) because it is distant from the tricuspid annulus. To avoid traumatizing the fast pathway, the author prefers to put the mapping catheter in the high septal area and pull the catheter

back in a motion similar to trans-septal puncture. This “high-to-low” approach is less likely to traumatize the fast pathway than the “low-to-high” approach in which the mapping catheter is pushed upward from the low septal area.

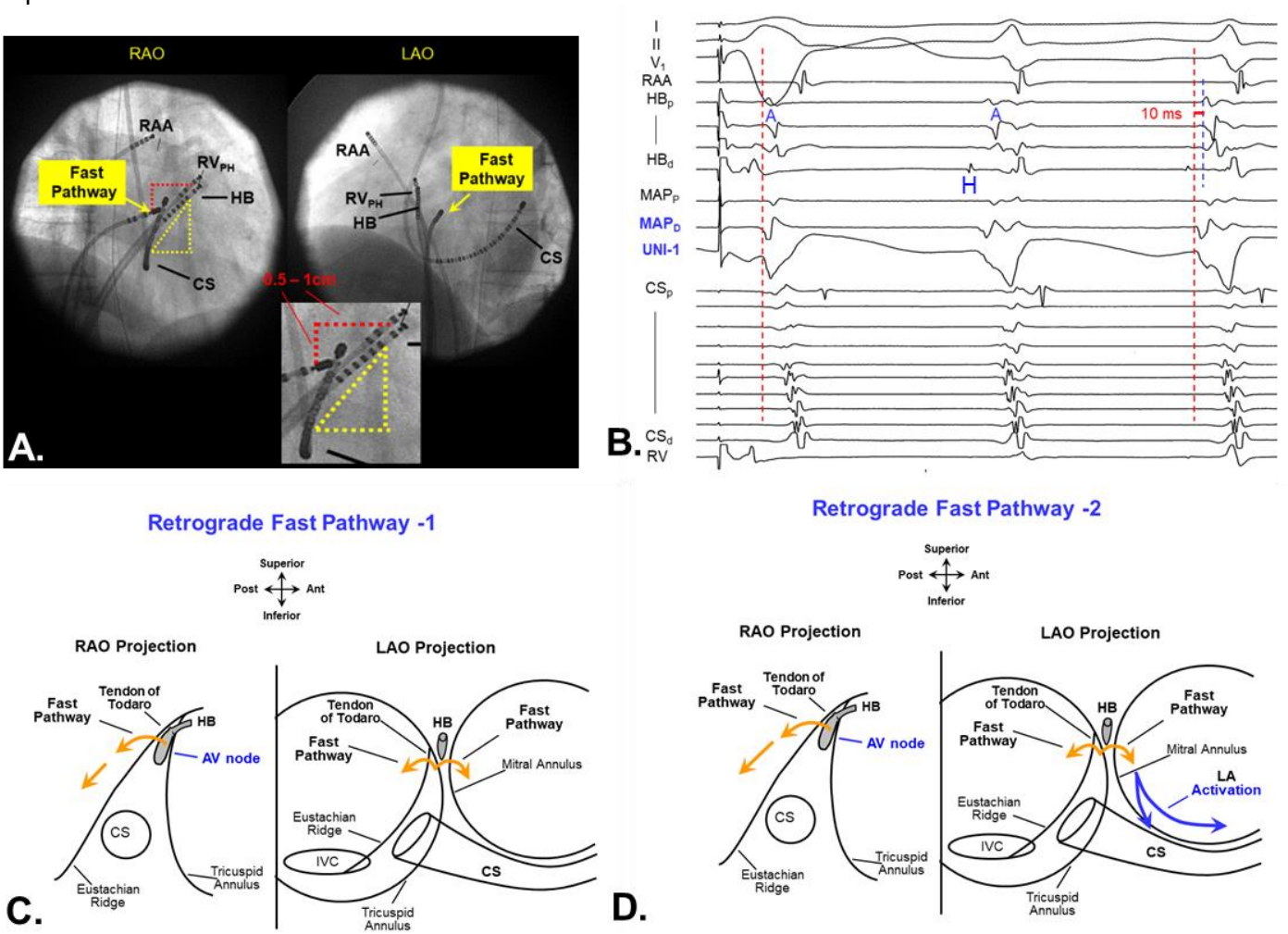


Figure 7.2. Hypothesized wave front propagation during retrograde conduction over the fast pathway. A. The site of earliest activation is not in the triangle of Koch (yellow triangle) but 0.5-1 cm posterior and inferior to the site recording the most proximal HB potential. Note that the catheter at the site of earliest activation is pointing leftward in the LAO view because it is behind the tendon of Todaro (TOT), no longer being pushed rightward by the TOT. **B.** A ventricular extra-stimulus (left beat) was delivered to separate the local ventricular activation from local atrial activation recorded on the mapping catheter to measure the atrial activation timing. At this site, the earliest atrial activation timing (vertical red lines) was approximately 10 ms earlier than the atrial activation timing (dotted blue line) at the site where a HB potential was recorded (far right beat). Note that the distal unipolar electrode (UNI-1) recorded an EGM with a QS pattern, suggesting that the atrial activation began there. **C.** Wave front propagates from the compact AV node to the site of earliest atrial activation through transitional fibers. This wave front activates the RA and LA septum nearly simultaneously. **D.** Wave front continues along the septal inferior wall of the LA, followed by activating the CS myocardium 1-3 cm from the CS ostium. *Courtesy of Dr. Jackman.*

Retrograde conduction over the AV nodal slow pathway

Dr. Jackman defines retrograde conduction as occurring over a slow pathway when earliest retrograde atrial activation is recorded within the inferior triangle of Koch (RIE) or within the coronary sinus (LIE), *regardless of the HA interval* (**Figure 7.3, 7.4)**. It is the author’s opinion that defining retrograde slow pathway conduction based on the site of earliest atrial activation is far better than defining it based on a long HA interval for the following two reasons. First, the anecdotal experience of the OU-EP group is that in approximately 20% of AVNRT with a short HA interval, the site of earliest atrial activation was in the inferior triangle of Koch or near the CS ostium. In other words, these 20% cases were indeed slow/slow AVNRT,

not slow/fast AVNRT. The incidence of AVNRT recurrence was substantially higher if the retrograde slow pathway was not ablated in these patients. Second, operators can map the site of earliest atrial activation of the retrograde slow pathway participating in AVNRT and select it as the ablation target. If a slow/slow AVNRT were to be classified as slow/fast AVNRT based on a short HA interval, operators are unlikely to map the retrograde "fast" pathway and consider ablating it to treat "slow/fast" AVNRT. It is the author's opinion that slow/slow or fast/slow AVNRT is usually easier to ablate than difficult slow/fast AVNRT because the retrograde limb of the slow/slow or fast/slow AVNRT reentrant circuit can be mapped and targeted accordingly.

In theory, one should be able to record continuous activation of retrograde RIE or LIE conduction along the right mid-septal area in the triangle of Koch. However, it is uncommon to record retrograde RIE conduction until it reaches the level of CS ostium or to record retrograde LIE conduction until it reaches the roof of proximal CS. Dr. Jackman hypothesized that the RIE and LIE may take an intramural or epicardial course until conduction emerges to the endocardial surface at the level of CS ostium (RIE) or the roof of proximal CS (LIE).

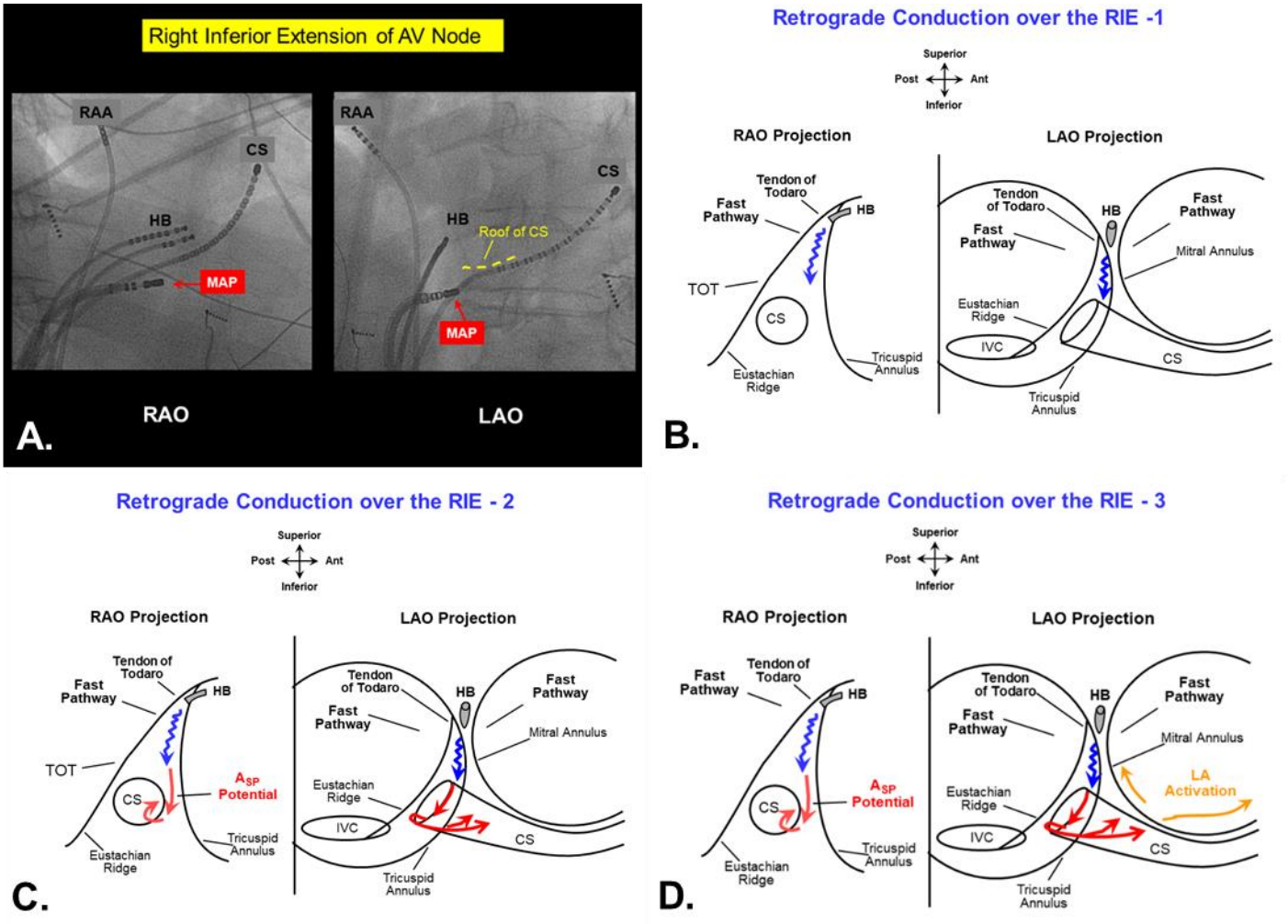


Figure 7.3. Hypothesized wave front propagation during retrograde conduction over the RIE. **A.** Radiographs demonstrate the location of the RIE. **B.** Wave front propagates from the compact AVN toward the inferior triangle of Koch. The wave front may take an intramural course in the anteroseptal and midseptal area; therefore, it is uncommon to record retrograde RIE conduction there. **C.** Wave front activates the myocardium in the inferior triangle of Koch and CS ostium and then propagates into the floor of the proximal CS. A slow pathway potential (Asp) may be recorded in the inferior triangle of Koch. **D.** The roof of the proximal CS is activated which subsequently activates the LA myocardium. *Courtesy of Dr. Jackman.*

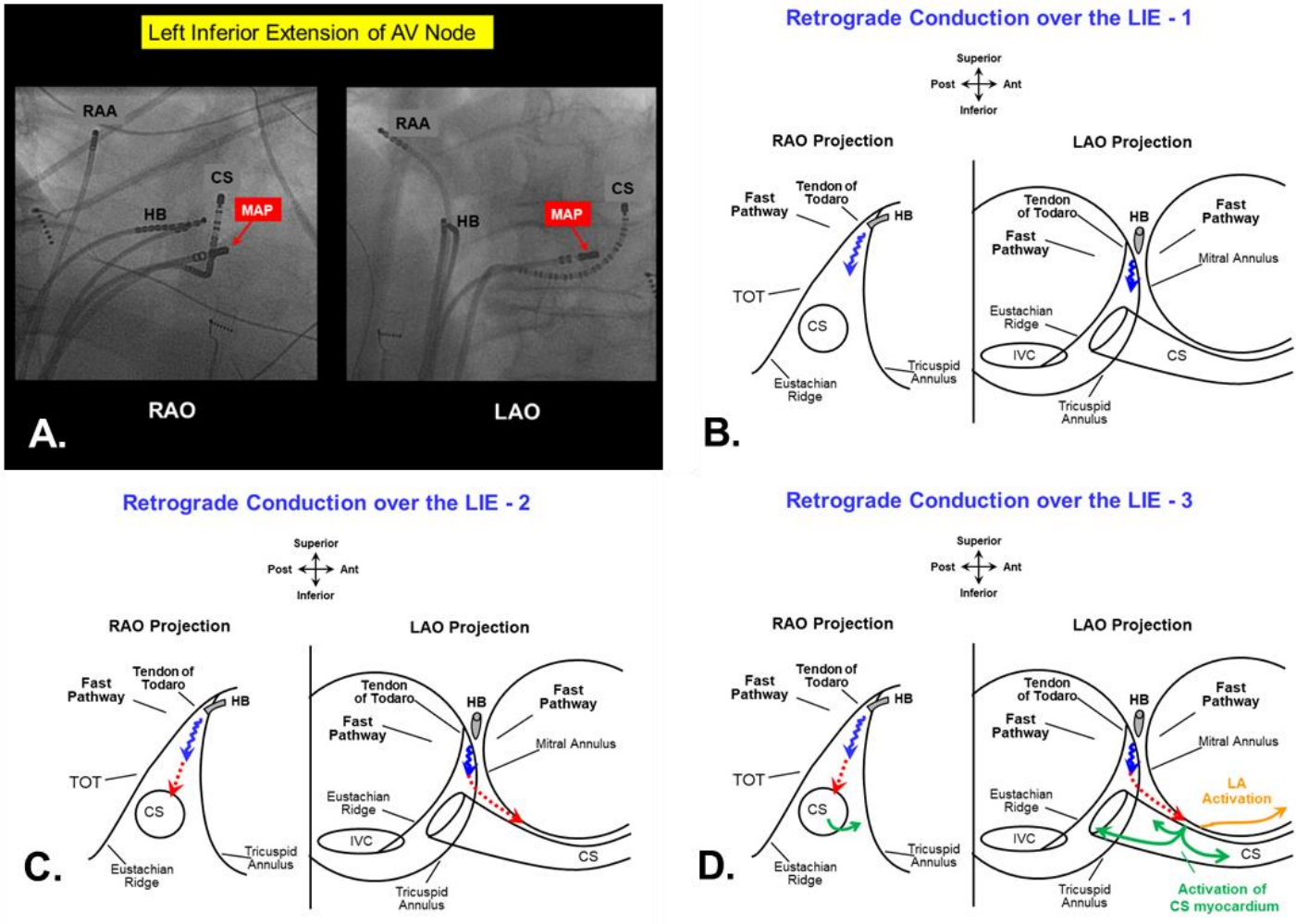


Figure 7.4. Hypothesized wave front propagation during retrograde conduction over the LIE. A. Radiographs demonstrate the location of the LIE. **B-C.** Wave front propagates from the compact AVN toward the inferior triangle of Koch. Dr. Jackman hypothesized that the wave front turns leftward to activate the LA septum based on Dr. Anton Becker's observations (**Figure 7.1A**). Activation usually cannot be recorded until it reaches the roof of proximal CS. **D.** The roof of proximal CS is activated which subsequently activates the LA myocardium. Wave front also propagates toward the CS ostium and then activates the RA. *Courtesy of Dr. Jackman.*

Lower common pathway

The lower common pathway (LCP) in AVNRT represents the tissue between the HB and the junction of the *end of* the antegrade limb and the *beginning* of the retrograde limb (**Figure 7.5)**. The presence of a LCP indicates that the HB is not part of the AVNRT reentrant circuit; therefore, AV conduction block should be a common observation during AVNRT. It is indeed very common to observe AVNRT with 2:1 AV block, particularly in the first few seconds of AVNRT. In the presence of a long LCP, ventricular extra-stimuli may need to advance the retrograde HB potential by >60-70 ms before engaging the reentrant circuit to reset AVNRT. In slow/fast AVNRT, as soon as a ventricular extra-stimulus advances the timing of the retrograde HB potential by 10-20 ms, the next atrial potential is advanced and the tachycardia is reset (HA linking), suggestive of a very short LCP. The short LCP in slow/fast AVNRT may explain the relatively constant H-A interval during different episodes of AVNRT. If one attempts to deliver ventricular overdrive pacing to entrain any form of AVNRT, tachycardia cannot be entrained until the retrograde HB potential has been entrained first. The diagnosis of AVNRT can be verified or excluded by how VES or RV overdrive pacing affects the retrograde HB timing before it perturbs the tachycardia (see **Chapter 4** for detail)

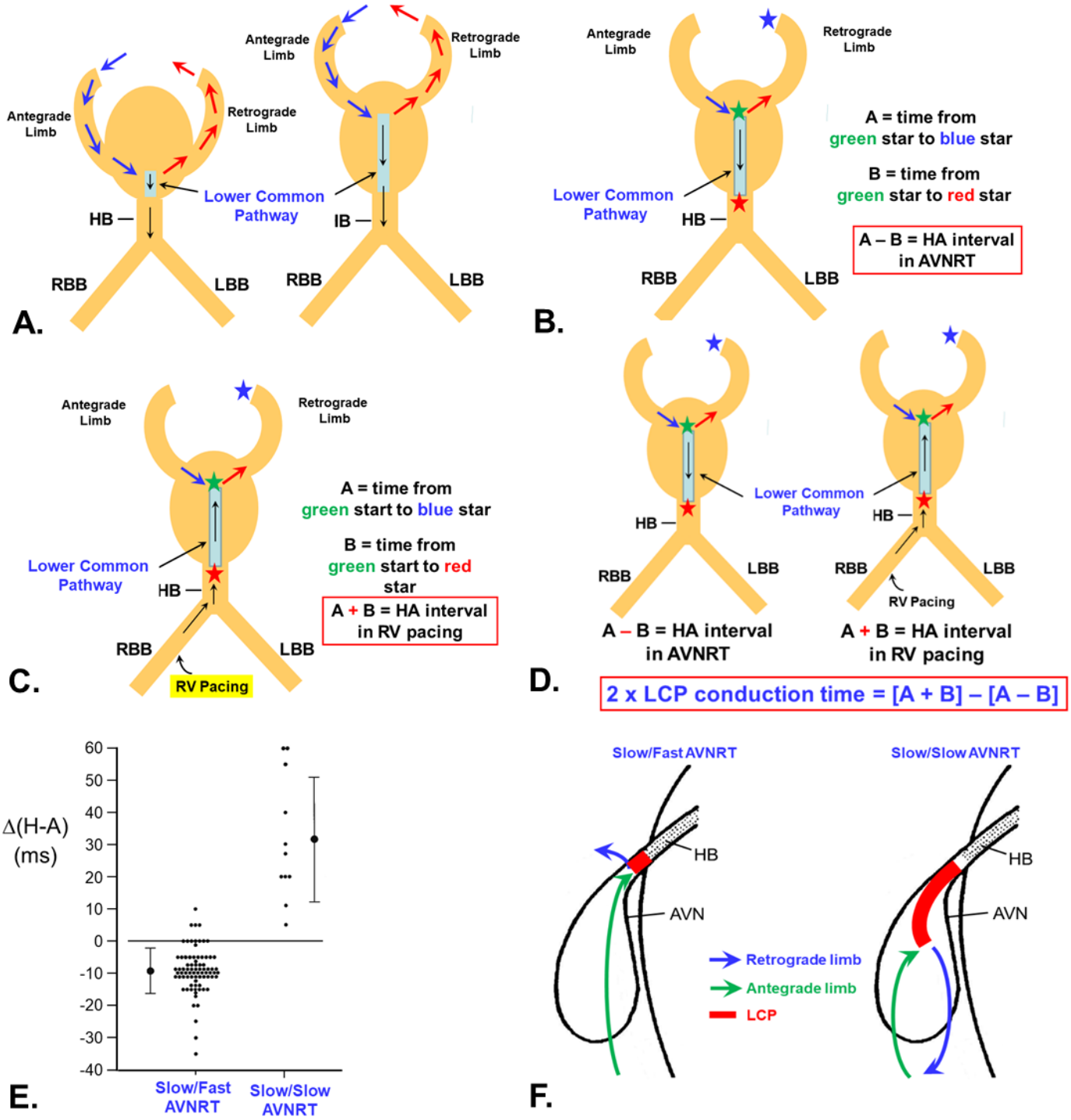


Figure 7.5. Lower common pathway (LCP) in AVNRT reentrant circuit. A. left panel: short LCP. **Right panel:** long LCP. **B**. Green, red and blue stars represent the beginning of LCP, the end of LCP and the atrial end of the retrograde limb of the reentrant circuit, respectively. The HA interval equals A - B. Therefore, a long LCP (a larger B value) can lead to a short HA interval. When the conduction time over the LCP is very long (a very large B value), the HA interval can be a negative value. Importantly, one needs to use the timing of the most proximal HB potential for these measurements. If a proximal RBB potential is used instead, it will artificially increase the conduction time of the LCP and artificially shortens the HA interval. **C and D**. In RV pacing, the HA interval equals A + B. The conduction time over the LCP can be estimated by comparing the HA interval during RV pacing and AVNRT ([A + B] – [A - B] = 2B). **E.** The difference of the HA interval between RV pacing and AVNRT is significantly larger in slow/slow AVNRT than that of slow/fast AVNRT due to a longer LCP in slow/slow AVNRT. **F. Left panel.** For slow/fast AVNRT, the site where the antegrade and retrograde limb connect may be in the distal compact AVN, leading to a short LCP. **Right panel.** A longer LCP in slow/slow or fast/slow AVNRT may be caused by a more inferior location in the triangle of Koch where the antegrade and retrograde limb of the reentrant circuit connect.

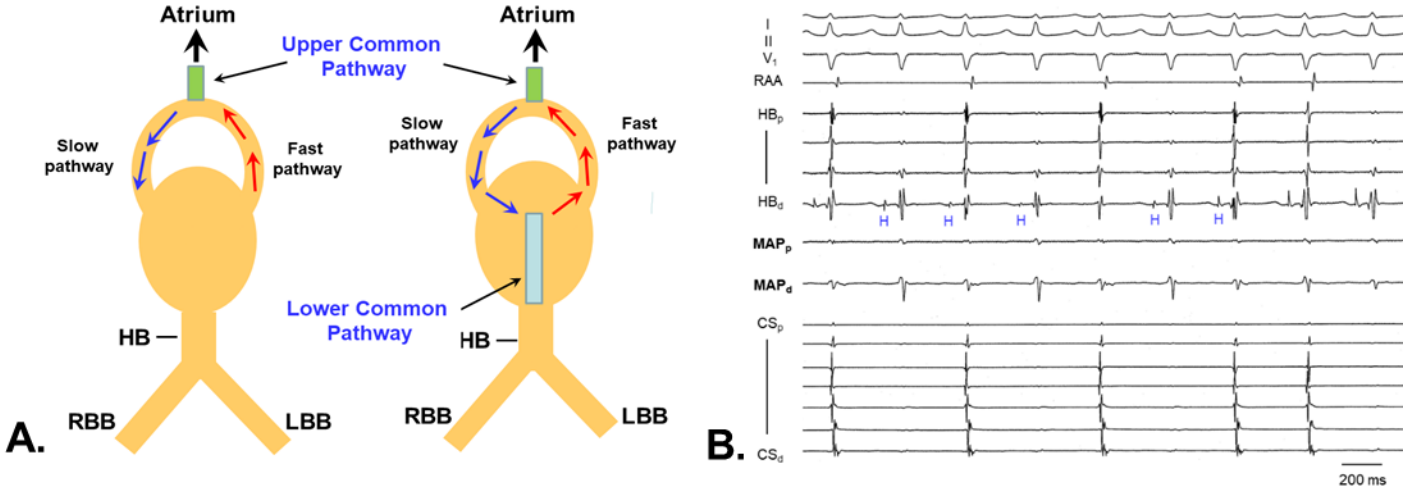
The length of the LCP, assessed by its conduction time, differs among various forms of AVNRT. Dr. Jackman uses two techniques to estimate the conduction time over the LCP.

1. Compare the H-A interval during tachycardia to the H-A interval during ventricular pacing at the tachycardia CL. Right ventricular pacing is delivered from the parahisian area without capturing the HB/RBB. To maintain the same autonomic tone, ventricular pacing is initiated immediately after termination of tachycardia. Qualitatively, the greater the difference between the H-A interval during pacing and the H-A interval during tachycardia, the longer the conduction time over the LCP. Quantitatively, one can calculate the conduction time over the LCP by subtracting the HA interval during AVNRT from that during RV pacing (**Figure 7.5C-D**); the difference is usually larger in slow/slow or fast/slow AVNRT than that of slow/fast AVNRT due to a long LCP (**Figure 7.5E**). Dr. Jackman hypothesized that a longer LCP in slow/slow or fast/slow AVNRT may be caused by a more inferior location in the triangle of Koch where the antegrade and retrograde limb of the reentrant circuit connect (**Figure 7.5F**).

2. Deliver ventricular extra-stimuli during AVNRT. The paced RV wave front has to engage the distal HB and traverse the entire LCP to enter the AVNRT reentrant circuit. The longer the LCP conduction time, the greater the advance in HB activation is required to reset AVNRT. For this reason, slow/slow and fast/slow AVNRT may require the ventricular extra-stimulus to advance the retrograde HB potential by ≥60-70 ms to reset the tachycardia (**Figure 7.5; Figure 4.7B**).

Upper common pathway

The upper common pathway in AVNRT represents the tissue between the atrium and the junction of the *beginning* of the antegrade limb and the *end* of the retrograde limb (**Figure 7.6**). In other words, the presence of an upper common pathway in AVNRT indicates that the entire atrium does not participate in the AVNRT reentrant circuit and VA conduction block during AVNRT should be a common observation. However, AVNRT with VA block or VA dissociation is a relatively rare phenomenon. Dr. Jackman's working hypothesis states that both the proximal CS and LA form critical elements in the AVNRT reentrant circuit. In the OU-EP laboratory, we have confirmed only a handful of cases of AVNRT with VA block or VA dissociation, suggesting that upper common pathway may exist only in a very small percentage of AVNRT patients. This underscores the important role of the atrium and proximal CS in the AVNRT reentrant circuit.



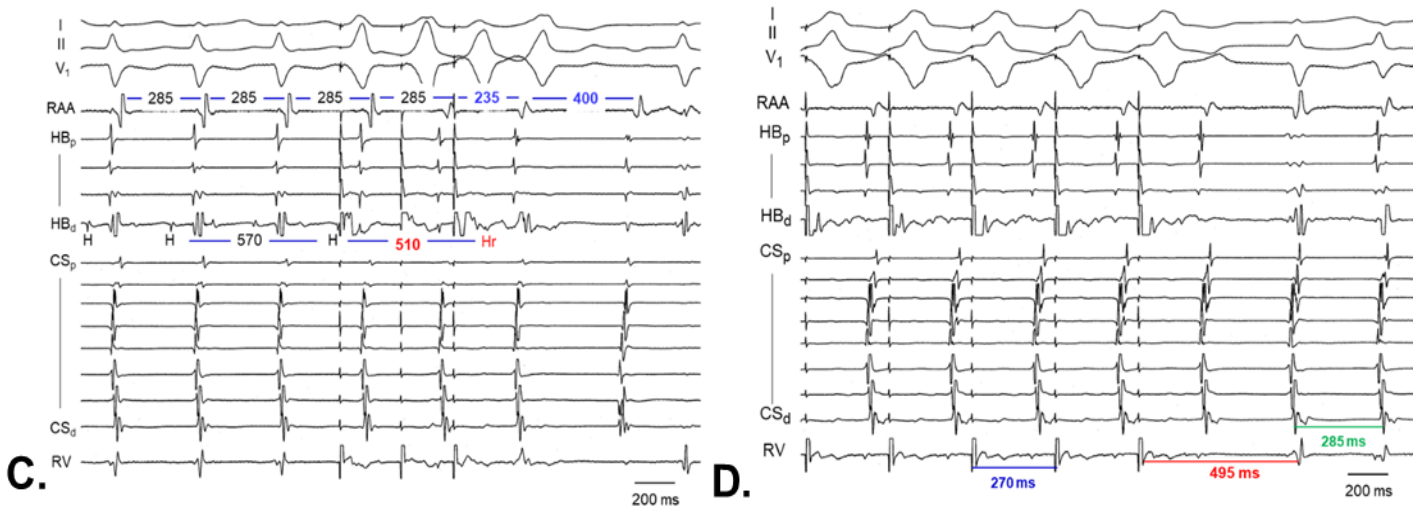


Figure 7.6. Upper common pathway (UCP). Left panel. A. AVNRT reentrant circuit with an UCP. UCP is the tissue between the atrium and the junction of the *beginning* of the antegrade limb and the *end* of the retrograde limb. The activation wave front has to traverse the UCP to exit to the atrium. VA conduction block should be a common phenomenon if an UCP is present. **Right panel.** An AVNRT reentrant circuit with a LCP and an UCP. In theory, both AV block and VA block can occur in AVNRT. If both AV and VA block occur simultaneously, AVNRT would look like asystole. **B.** An episode of AVNRT with VA block was induced. **C.** Later, stable 1:1 VA conduction resumed. A series of triple ventricular extra-stimuli failed to affect the tachycardia (not shown) until the retrograde HB potential (Hr) was advanced by 60 ms (H-Hr=510 ms), which advanced the next atrial activation by 50 ms and reset the tachycardia, proving that it was AVNRT with a relatively long lower common pathway. **D.** Tachycardia was entrained by RV pacing with a VAV response and a long PPI, also proving that it was AVNRT.

Classification of AVNRT

In Dr. Jackman's working hypothesis, variants of AVNRT (slow/fast, slow/slow or fast/slow) are *not* defined by the HA intervals but rather by the site of earliest atrial activation, namely the atrial component of the retrograde limb of the reentrant circuit. For example, the site of earliest retrograde atrial activation located in the proximal CS or in the inferior triangle of Koch indicates that a retrograde slow pathway is used for retrograde conduction and this AVNRT is either slow/slow or fast/slow AVNRT, regardless of the HA interval.

Dr. Jackman's hypothesis that the LA and CS account for the majority of the length of the AVNRT reentrant circuit has not been accepted by all electrophysiologists because most of the AVNRT can be successfully ablated in the inferior triangle of Koch, implying that the LA and CS are not part of the reentrant circuit. Dr. Jackman's counterpoint is that only a small segment of the AVNRT reentrant circuit is in the inferior triangle of Koch. To interrupt a reentrant circuit, ablation at any point along the circuit can eliminate the reentry. The aforementioned "sites of earliest activation" of retrograde conduction over the RIE or LIE are indeed the earliest site where activation can be recorded in the RA or CS. When the AVNRT reentrant circuit is not close to the inferior triangle of Koch (e.g. LIE, **Figure 7.4**), conventional slow pathway ablation there cannot interrupt the reentrant circuit. In this scenario, Dr. Jackman's rule of thumb is to search for other AVN extensions (e.g. LIE) in the LA and CS when ablation of the RIE does not eliminate AVNRT.

Dr. Jackman's observations classify AVNRT into the following forms. The proposed reentrant circuit and successful ablation site of each form will be discussed later in this chapter. In essence, AVNRT reentrant circuits are formed by different permutations of the fast pathway and at least 4 different slow pathways.

1. Slow/fast AVNRT using the RIE for antegrade conduction. This is the most common form of AVNRT. The successful ablation site is located in the inferior triangle of Koch, between the tricuspid annulus and CS ostium.

2. Slow/fast AVNRT using the LIE for antegrade conduction accounts for approximately 5% of slow/fast AVNRT. The successful ablation site is located along the roof of the proximal CS, 2-4 cm distal to the ostium. The RIE is a bystander in this type of AVNRT; therefore, ablation of the RIE can elicit junctional automaticity without affecting AVNRT.

3. Slow/fast AVNRT using a left atrial slow pathway for antegrade conduction. Left atrial ablation is required to eliminate this rare form of AVNRT. Both RIE and LIE are bystanders in this type of AVNRT.

4. Slow/fast AVNRT using a superior form of slow pathway. The reentrant circuit of this type of AVNRT was recently described and remains poorly understood. To eliminate this extremely rare form of AVNRT, ablation of the superior inter-atrial septum (superior to the HB) or in the coronary cusp may be required.

5. Slow/slow AVNRT using the RIE for antegrade conduction and LIE for retrograde conduction.

6. Fast/slow AVNRT using the LIE for antegrade conduction and RIE for retrograde conduction. Dr. Jackman hypothesized that both fast/slow and slow/slow AVNRT use the RIE and LIE as the major components of the reentrant circuit but in opposite directions (see discussion later in this chapter).

Proposed reentrant circuit of various forms of AVNRT

Reentrant circuit in slow/fast AVNRT

Dr. Jackman proposed the most common form of slow/fast AVNRT as using the RIE for antegrade conduction and the fast pathway for retrograde conduction (**Figure 7.7**). As already discussed, the site of earliest atrial activation of retrograde fast pathway conduction is recorded posterior to the tendon of Todaro, 5-10 mm posterior and inferior to the site recording a proximal HB potential. Atrial activation is then followed sequentially by activation of the left atrial septum, posteroseptal LA, proximal CS, CS ostium, inferior triangle of Koch generating the slow pathway potential (A_{SP}) and finally propagating superiorly in the triangle of Koch to the compact AVN to complete the reentry. That is to say, much of the reentrant circuit for the most common type of AVNRT is located in the LA and proximal CS. Most of the right atrium is a bystander and can be dissociated from AVNRT by atrial extra-stimuli, leading to the long-held notion that the atrium is not part of the AVNRT reentrant circuit. Another important point related to the critical role of the proximal CS in the slow/fast AVNRT reentrant circuit is that atrial extra-stimuli are best delivered from the proximal CS or CS ostium, not RA, if one tries to differentiate slow/fast AVNRT from junctional tachycardia (**Figure 4.17**).

In approximately 5% of patients with slow/fast AVNRT, the antegrade limb of the reentrant circuit is formed by the LIE (**Figure 7.8**). The site of earliest atrial activation (retrograde fast pathway) is recorded posterior to the tendon of Todaro. Atrial activation is then followed sequentially by activation of the left atrial septum, posteroseptal LA and then the atrial end of the LIE. Activation then propagates to the mid-septal are and then compact AVN to complete the reentry. In this form of slow/fast AVNRT, the RIE is a bystander but ablation in the RIE area often elicits copious junctional automaticity without affecting AVNRT.

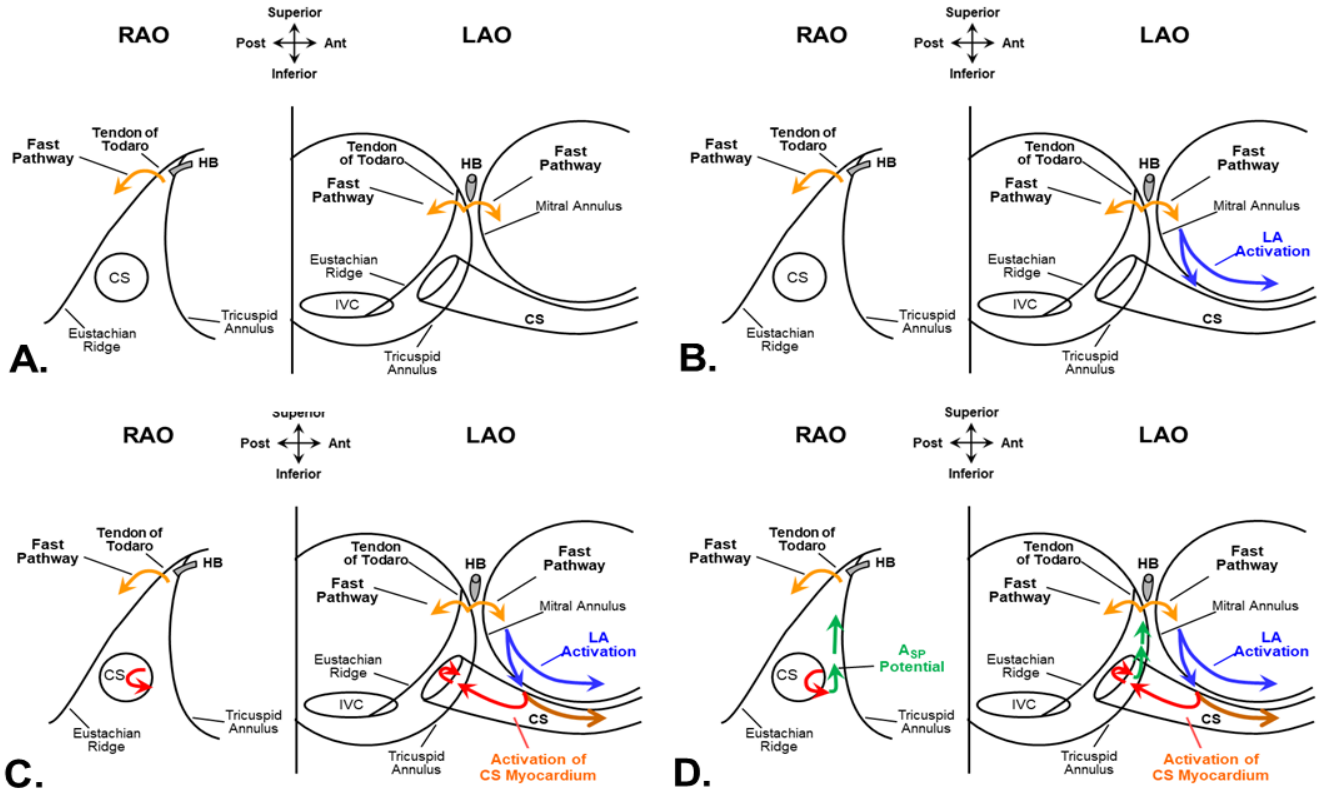


Figure 7.7. Hypothesized reentrant circuit for slow/fast AVNRT using the RIE for antegrade conduction. A. Retrograde fast pathway conduction activates both the right and left atrial septum at a site slightly inferior and posterior to the His bundle. **B.** Wave front propagates to the posteroseptal LA and activates the proximal CS myocardium. **C.** Wave front propagates toward the CS ostium. **D.** Inferior triangle of Koch is activated, a slow pathway potential (A_{SP}) is generated and wave front propagates to the compact AVN to complete the reentry. *Courtesy of Dr. Jackman.*

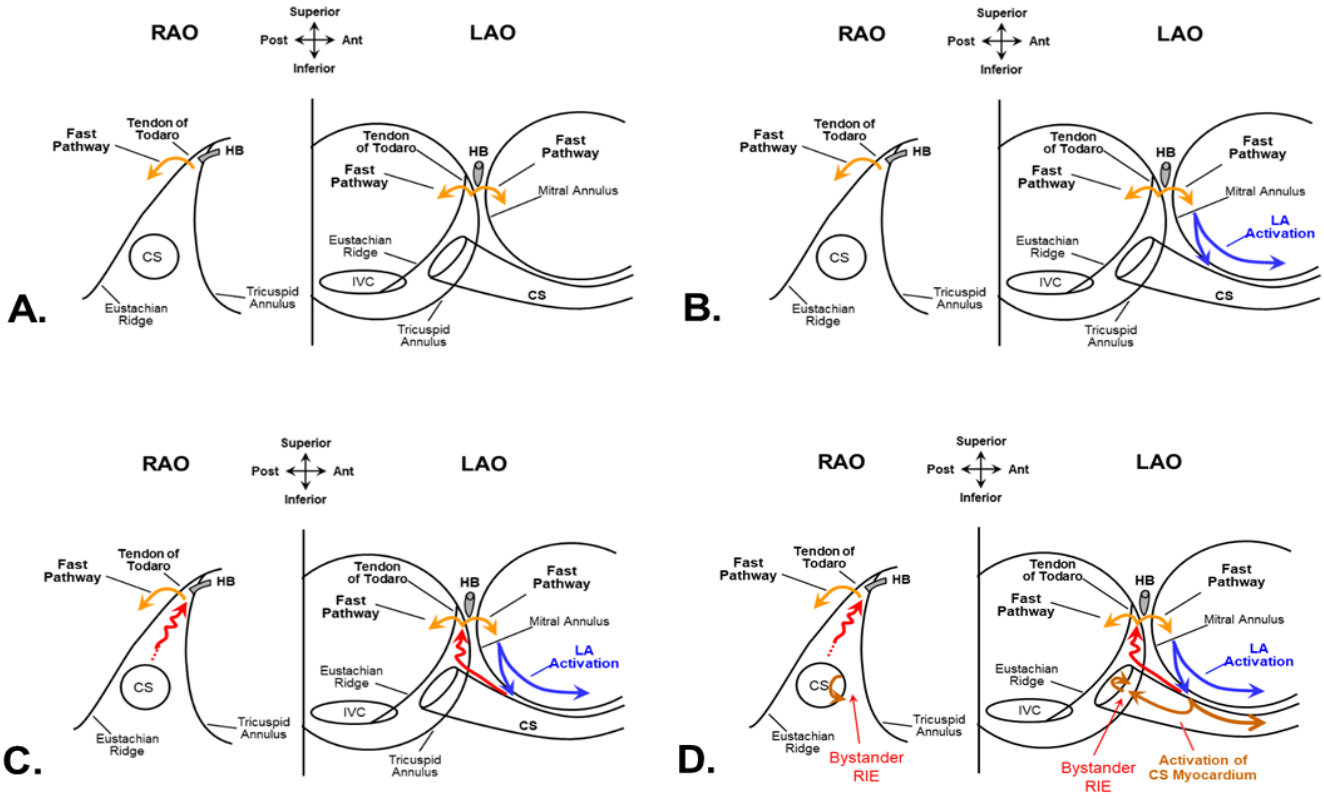


Figure 7.8. Hypothesized reentrant circuit for slow/fast AVNRT using the LIE for antegrade conduction. A. Retrograde fast pathway conduction activates both the right and left atrial septum at a site slightly inferior and posterior to the His bundle. **B-C.** Wave front propagates to the posteroseptal LA and activates the LIE, which conducts back to the mid-septal area and then compact AVN to complete the reentry. **D.** Wave front also conducts toward the CS ostium and inferior triangle of Koch. However, RIE and CS ostium are bystanders in this type of AVNRT. Ablation of the RIE usually elicits copious junctional automaticity but without effect on AVNRT. *Courtesy of Dr. Jackman.*

In less than 1% of patients with slow/fast AVNRT, the antegrade limb of the reentrant circuit is formed by a left atrial slow pathway. The site of earliest atrial activation (retrograde fast pathway) is recorded posterior to the tendon of Todaro. Atrial activation is then followed sequentially by activation of the left atrial septum, posteroseptal LA and the atrial component of the left atrial slow pathway located in the posterolateral LA adjacent to the mitral annulus but not on the annulus like a left free wall accessory pathway. Activation then propagates to the LA posterior septum, connecting to the compact AVN to complete the reentry (**Figure 7.9**). In this rare form of slow/fast AVNRT, the entire reentrant circuit is located in the LA. Both RIE and LIE are bystanders but ablation in the RIE area often elicits copious junctional automaticity without affecting AVNRT.

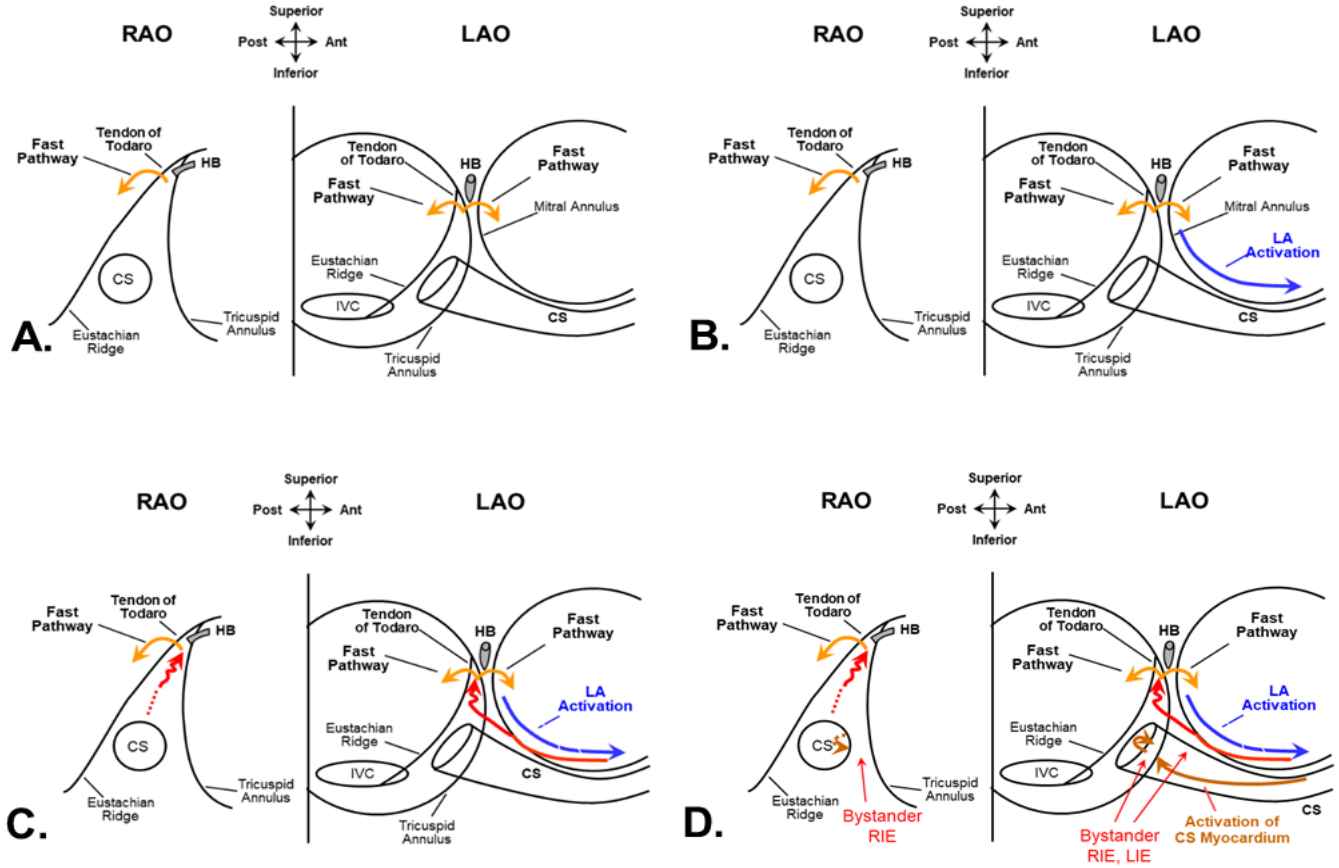


Figure 7.9. Hypothesized reentrant circuit for slow/fast AVNRT using the left atrial slow pathway for antegrade conduction. A. Retrograde fast pathway conduction activates both the right and left atrial septum at a site slightly inferior and posterior to the His bundle. **B.** Wave front activates the atrial component of the slow pathway located in the posterolateral LA. **C.** Wave front propagates back to the compact AVN to complete the reentry. **D.** Wave front also conducts toward the CS ostium and inferior triangle of Koch. However, RIE, LIE and CS ostium are bystanders in this type of AVNRT. Ablation of the RIE usually elicits copious junctional automaticity but without effect on AVNRT. *Courtesy of Dr. Jackman.*

Reentrant circuit in slow/slow and fast/slow AVNRT

Before Dr. Jackman pioneered slow pathway ablation, the standard approach was to ablate the fast pathway to treat AVNRT. In Dr. Jackman's experience, a significant number of patients recurred with

fast/slow or slow/slow AVNRT after successful fast pathway ablation, which prompted Dr. Jackman to investigate the reentrant circuit of slow/slow and fast/slow AVNRT and concluded that the fast pathway does not participate in slow/slow or fast/slow AVNRT. From Dr. Jackman's detailed mapping studies and the observed sites of successful ablation, he hypothesized that slow/slow and fast/slow AVNRT used the same reentrant circuit but in opposite directions (**Figure 7.10**). That is, the two limbs of the reentrant circuit are the same but activated in opposite directions. Dr. Jackman differentiates slow/slow AVNRT from fast/slow AVNRT on the basis of the site of earliest atrial activation (**Figure 7.3 and 7.4**) as well as the A-H and H-A intervals. AVNRT with earliest atrial activation recorded in the triangle of Koch or CS ostium and with a significantly shorter A-H interval than H-A interval is classified as fast/slow AVNRT. AVNRT with earliest atrial activation recorded along the roof of the proximal CS but with a longer AH interval is classified as slow/slow AVNRT. Slow/slow AVNRT most often uses the RIE as the antegrade limb and the LIE as the retrograde limb of the reentrant circuit. Fast/slow AVNRT most often uses the LIE as the antegrade limb and the RIE as the retrograde limb of the reentrant circuit.

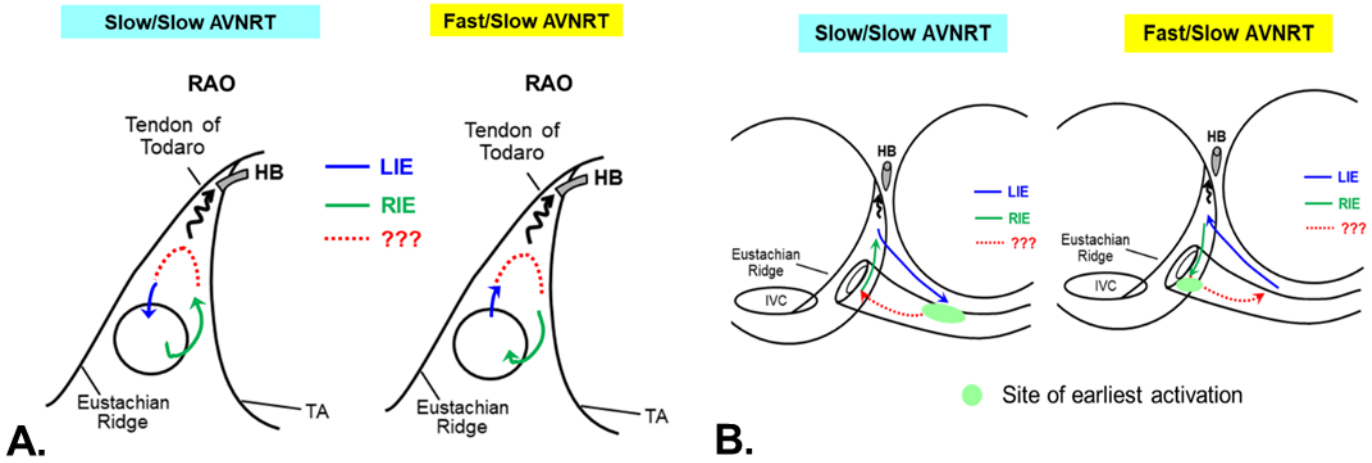


Figure 7.10. Hypothesized reentrant circuit for slow/slow and fast/slow AVNRT. Most of the slow/slow AVNRT uses the RIE for antegrade conduction and LIE for retrograde conduction. The site of earliest atrial activation is usually located along the roof of the proximal CS. Most of the fast/slow AVNRT uses the LIE for antegrade conduction and RIE for retrograde conduction. The site of earliest atrial activation is located in the inferior triangle of Koch or CS ostium. In both forms of AVNRT, where the two limbs of the reentrant circuit connect is still poorly understood.

The hypothesized clockwise and counterclockwise reentry between the RIE and LIE (**Figure 7.10**) is probably the most confusing and controversial part of Dr. Jackman's working hypothesis. After working with Dr. Jackman for 19 years, the author is still frequently puzzled by the complexity of the reentrant circuit. For a reentrant tachycardia, if one picks a different timing reference, early timing can become late timing. How on earth can "the site of earliest atrial activation" of a reentrant tachycardia be consistently recorded at the same site in different patients regardless how the operator set the timing reference? The answer is that these sites of "earliest atrial activation" are not true sites of earliest activation. They are the locations where the retrograde conduction wave front emerges from its intramural or epicardial course and can be recorded by catheters positioned on the endocardial surface. The author's opinion is that AVNRT patients often have multiple slow pathways; some of them only conduct in one direction but others in both antegrade and retrograde directions. Some of them are "lone wolf" pathways, not connecting with other slow or fast pathway to make AVNRT. When the distance in space and the differences in the refractory period are set just right, slow/slow or fast/slow AVNRT can occur. Regardless of whether or not one adopts the hypothesis of clockwise and counterclockwise reentry, the RIE and LIE sites shown in **Figure 7.1 to 7.3** can be mapped and effectively ablated to treat slow/slow or fast/slow AVNRT.

In general, the A-H interval during slow/slow AVNRT is >200 ms but the H-A interval has a very wide range of 20-320 ms (mean: approximately 100 ms). The H-A interval can become negative if the lower common pathway is very long (**Figure 7.5),** in which the conduction time from the most proximal part of the LCP to the atrium is substantially shorter than the conduction time over the LCP to the HB. Slow/slow AVNRT with a short H-A interval can be differentiated from slow/fast AVNRT by the site of earliest

retrograde atrial activation as already discussed. In Dr. Jackman's experience, the shortest HA interval (e.g.<0 ms) in AVNRT was observed in patients with slow/slow, not slow/fast AVNRT due to the presence of a very long LCP.

In the presence of a long LCP, RV pacing from the parahisian area may preferentially conduct through the fast pathway and mask retrograde LIE or RIE conduction. For this reason, 1:1 retrograde conduction over the slow pathway during RV pacing may be absent in many patients with fast/slow or slow/slow AVNRT. Mapping and ablation have to be performed during slow/slow or fast/slow AVNRT. During RV pacing, approximately half of the patients with slow/slow AVNRT have retrograde conduction only over the slow pathway, without retrograde fast pathway conduction at all. Operators need to expect VA block when the retrograde slow pathway conduction is eliminated by ablation during RV pacing. In this scenario, VA block during ablation does not imply fast pathway injury. In this situation, the author prefers to quickly increase the ventricular pacing CL to allow sinus rhythm to resume smoothly to maintain catheter stability and monitor the AH interval when RF application is continued.

Figure 7.11 and **Figure 7.12** illustrate typical examples of different forms of AVNRT. It cannot be overemphasized that different forms of slow/fast AVNRT cannot be differentiated by the site of earliest atrial activation because the real discriminator among them is the antegrade slow pathway participating in the tachycardia. A short HA interval cannot exclude slow/slow AVNRT. As already discussed, the shortest HA interval (<0 ms) often occurs in slow/slow AVNRT due to a long lower common pathway.

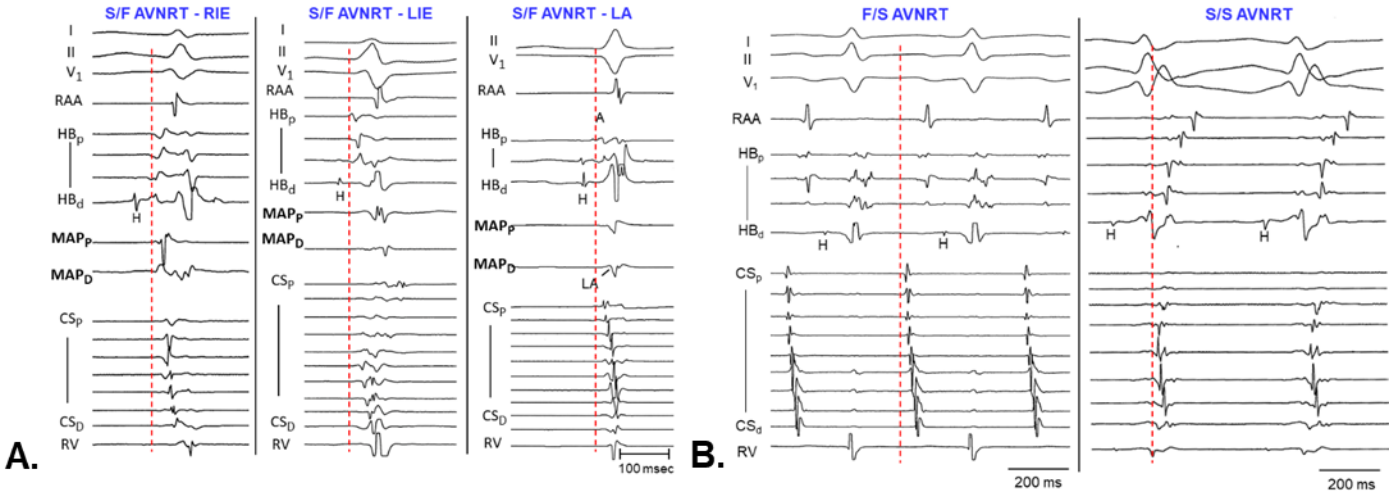
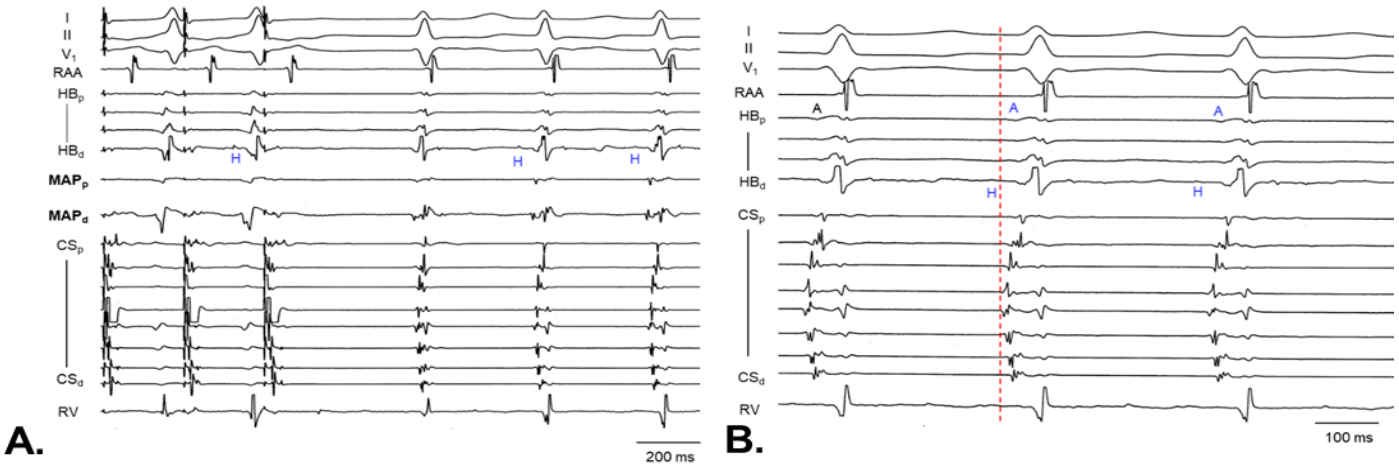


Figure 7.11. Intracardiac recordings of different forms of AVNRT. A. Three different forms of slow/fast AVNRT using different slow pathways for antegrade conduction. Left: RIE; middle: LIE, right: posterolateral LA. Note that all three forms have a short HA interval and atrial activation timing is earliest near the site recording a HB potential. **B.** Slow/slow (left panel) and fast/slow (right panel) AVNRT. Vertical red lines: timing of earliest atrial activation recorded by diagnostic catheters. Note that the HA interval of this slow/slow AVNRT was short.



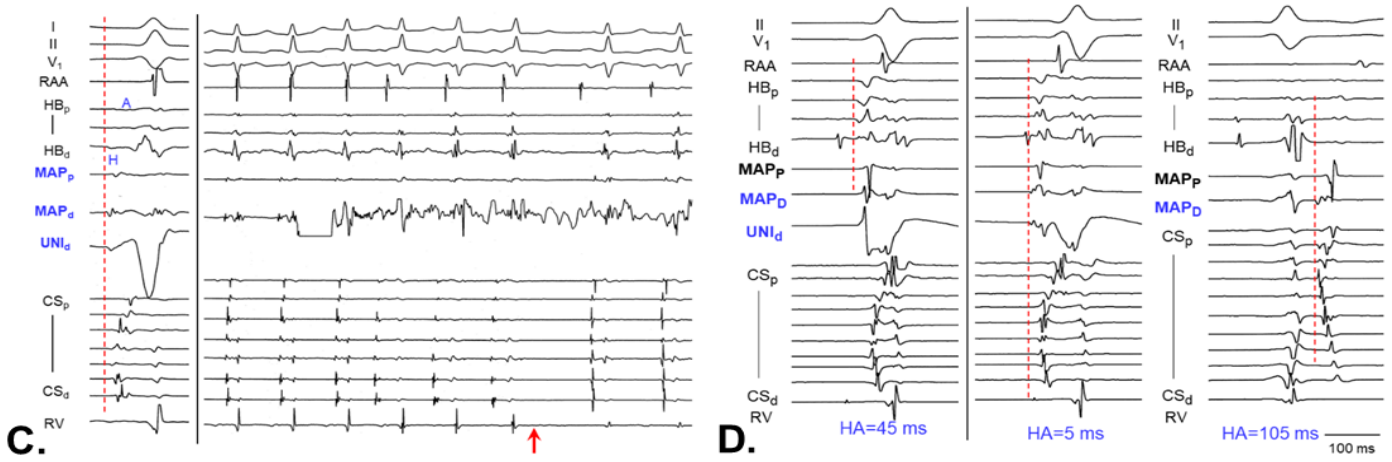


Figure 7.12. Slow/slow AVNRT with a very short HA interval. **A.** CS burst pacing induced AVNRT after an AH jump. **B.** This slow/slow has a very short HA interval, but the site of earliest activation was in the proximal CS. **C. Left panel.** Mapping catheter was positioned at the site of earliest atrial activation in the inferior triangle of Koch where the RIE is located. **Right panel.** Ablation there quickly caused retrograde slow pathway conduction block (red arrow) and termination of AVNRT. **D**. HA interval during slow/fast vs. slow/slow AVNRT in another patient. Mapping catheter was positioned at the RIE area for all 3 panels. **Left panel.** HA=45 ms during slow/fast AVNRT **Middle panel.** Shorter HA interval (5 ms) during slow/slow AVNRT. **Right panel.** HA interval = 105 ms during another slow/slow AVNRT in the same patient.

The following list summarizes the most important, practical observations that Dr. Jackman made over the past three decades:

1. *AVNRT has an atrial component. If single atrial extra-stimuli are delivered close to the atrial component of the AVNRT circuit, AVNRT can easily be reset.*

2. *The site of earliest atrial activation during retrograde fast pathway conduction is not in the triangle of Koch but rather at a site behind the Tendon of Todaro, 0.5-1 cm posterior and inferior to the site recording a HB potential (**Figure 7.2A**).*

3. *AVNRT with a short HA interval does not indicate that the retrograde limb is the fast pathway (**Figure 7.12**). A short HA interval can be caused by a long lower common pathway. There is a substantial overlap of the HA interval between retrograde slow pathway conduction and fast pathway conduction. A short HA interval is a common observation in slow/slow AVNRT as well; sometimes, the HA interval can be negative! If the HA interval is short and local ventricular activation obscures the site of earliest atrial activation, single ventricular extra-stimuli can expose the local atrial activation timing (**Figure 7.2B**).*

4. *In contrast to slow/fast AVNRT, both fast/slow and slow/slow AVNRT have a long lower common pathway. If ventricular extra-stimuli (VES) or RV overdrive pacing is used to diagnose fast/slow or slow/slow AVNRT, it is important to appreciate that very early VES or sufficiently long RV overdrive pacing will be required to reset or entrain fast/slow and slow/slow AVNRT. Many slow/slow or fast/slow AVNRTs were mistakenly diagnosed as atrial tachycardia because the duration of RV overdrive pacing was too short.*

5. *If slow pathway ablation at the conventional location (inferior triangle of Koch and CS ostium) fails to eliminate 1:1 antegrade slow pathway conduction and slow/fast AVNRT, targeting the LIE or left atrial slow pathway based on the resetting response carries a significantly lower risk of AVN injury than delivering RF applications progressively superiorly along the triangle of Koch.*

6. *Fast/slow AVNRT with a very long HA interval usually uses the RIE for retrograde conduction. The best ablation target is usually located at the lower edge of the CS ostium or inferior triangle of Koch. If RV pacing does not produce 1:1 retrograde slow pathway conduction with the same atrial activation sequence as AVNRT, ablation may have to be performed during AVNRT.*

Catheter Ablation of AVNRT

What Dr. Jackman would do in preparation for AVNRT ablation

1. Attempt to induce sustained AVNRT to verify the diagnosis of AVNRT.

2. Map the RA septum and CS in order to determine which type of AVNRT it is, based upon the site of earliest retrograde atrial activation. If the atrial activation sequence is identical between AVNRT and anteroseptal RV pacing, mapping can be performed during RV pacing as well.

3. Deliver decremental RA pacing to evaluate 1:1 antegrade slow pathway and fast pathway conduction as a reference to both ablation efficacy and safety.

4. Deliver decremental RV pacing to evaluate 1:1 retrograde slow pathway and fast pathway conduction. The occurrence of very rapid junctional automaticity during RF application or loss of retrograde fast pathway conduction has been reported as a prelude to AVN injury. However, loss of 1:1 retrograde fast pathway conduction can occur without injury to the AV node or fast pathway when the rate of the junctional rhythm exceeds the retrograde conduction capability of the fast pathway. In addition, in approximately half of the patients with slow/slow AVNRT, there was no retrograde fast pathway conduction at all. Slow pathway ablation in sinus rhythm may lead to junctional automaticity without VA conduction. Immediate termination of RF application may not be necessary. With this in mind, operators should be prepared to immediately initiate atrial pacing to monitor the AH interval when AVNRT termination and VA block occur during ablation of slow/slow or fast/slow AVNRT. As noted, this is because ablation in AVNRT is often required for lack of 1:1 retrograde slow pathway conduction during RV pacing. For these reasons, Dr. Jackman delivers decremental ventricular pacing immediately before ablation to evaluate retrograde conduction in order to set the stimulator in advance.

The author prefers to do a quick map of the CS ostium to know the location of its roof and floor, followed by evaluating the distance between the middle of the CS ostium and the HB potential to assess the risk of AVN injury. For a low-sitting HB potential (e.g. in elderly patients with a tortuous ascending aorta), the ablation site may need to be slightly lower than the middle level of the CS ostium to avoid AVN injury. For this type of higher risk ablation, the author surely will implement the resetting technique to find and target the slow pathway participating in AVNRT (see discussion below).

Ablation of common slow/fast AVNRT (RIE as the antegrade slow pathway)

The ablation target of common slow/fast AVNRT is the RIE. Dr. Jackman prefers to create a short linear lesion between the tricuspid annulus and the edge of the CS ostium, *at the level of the middle of the CS ostium* (**Figure 7.13A**). Junctional automaticity (sometimes only 1-2 beats) occurs in >95% patient with successful elimination of the RIE. If RF applications to the RIE fail to elicit junctional automaticity, the operator should presume that these RF lesions were not effective due to poor electrode-issue contact. The RF application (30-35 watts) is maintained at each site producing junctional automaticity until 15-20 sec after cessation or marked slowing of the junctional automaticity. The RF pullback is continued until the ablation electrode reaches the edge of CS ostium. On approaching the margin of the CS ostium during the

pullback, RF power is reduced to 20-25 Watts. The tip electrode is then positioned within the edge of the CS ostium. Dr. Jackman avoids delivering RF energy near the floor of the CS ostium to prevent coronary artery injury (see **Chapter 6** for detail).

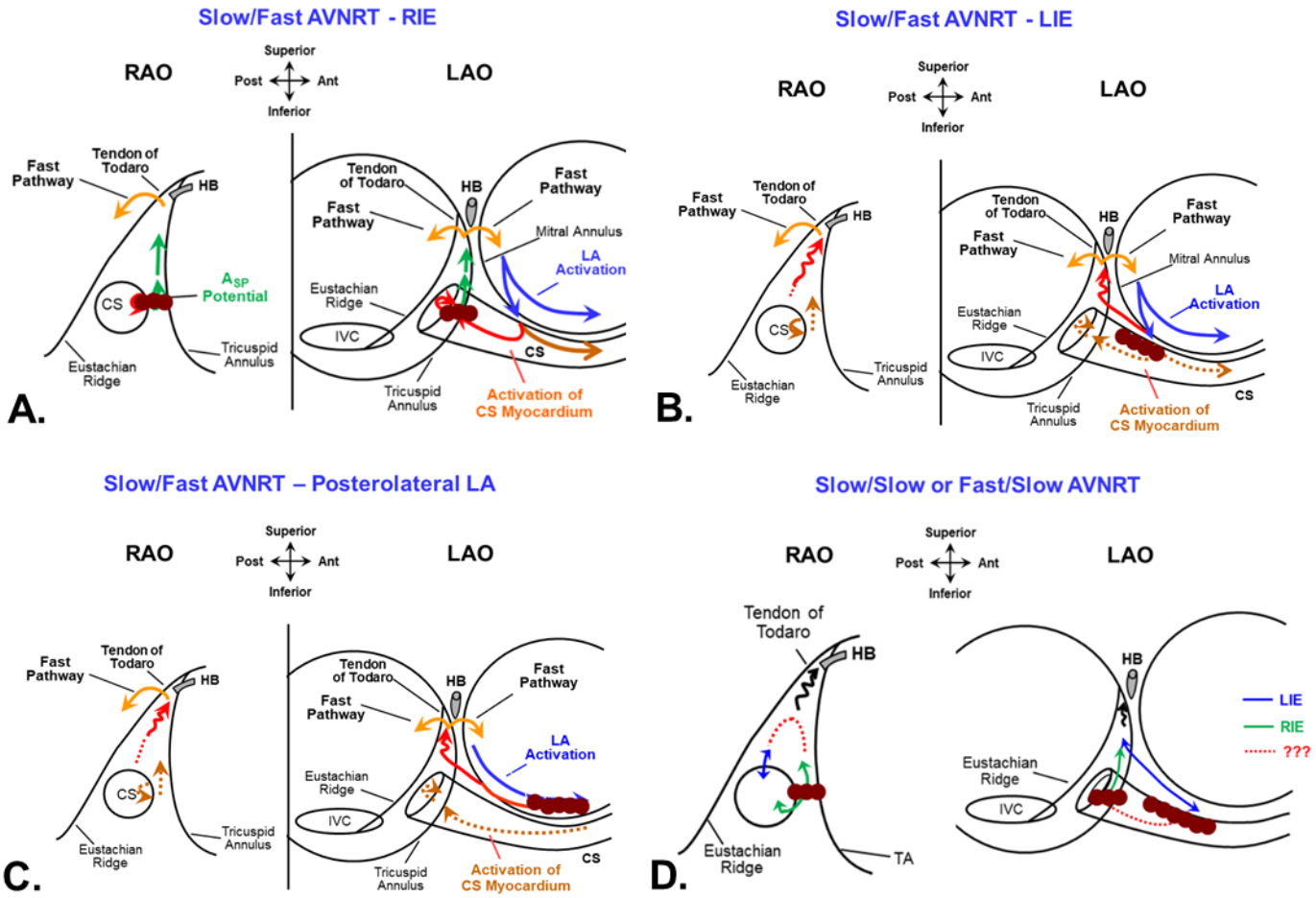


Figure 7.13. Successful ablation sites of different forms of AVNRT. A. Slow/fast AVNRT using the RIE for antegrade conduction. Successful ablation site is usually in the inferior triangle of Koch or at the CS ostium. **B.** Slow/fast AVNRT using the LIE for antegrade conduction. Successful ablation site is usually along the roof of the proximal CS, 2-4 cm from the ostium. Ablation usually does *not* elicit junctional automaticity. **C**. Slow/fast AVNRT using the left atrial slow pathway for antegrade conduction. Successful ablation site is usually at the posterolateral LA not far from to the mitral annulus. **D**. Ablation of slow/slow and fast/slow AVNRT often requires targeting both the RIE and LIE. *Courtesy of Dr. Jackman*

The author has never seen Dr. Jackman ablate an RIE superior to the superior margin of the CS ostium. The incidence of AVN injury using his approach is <0.5%, including many patients with multiple prior AVNRT ablations. Moreover, Dr. Jackman never uses cryoablation to treat AVNRT. If ablation up to the superior margin of the CS ostium still fails to eliminate the antegrade slow pathway and AVNRT, it indicates that the antegrade slow pathway participating in the AVNRT is likely located in the CS (LIE) or in the left atrium (the left atrial variant of slow/fast AVNRT). Resetting with single atrial extra-stimuli (see description below) will be employed to localize the antegrade slow pathway participating in the reentrant circuit.

The author prefers to start RIE ablation at a site on the tricuspid annulus at the level of the middle of the CS ostium where the distal electrode of the ablation catheter records *a very small or no* atrial potential but the proximal electrode pair records a relatively large atrial potential (**Figure 7.14**). This approach is to ensure that the most annular aspect of the inferior triangle of Koch is not missed. The author adopted this approach by virtue of experiences in which many referring electrophysiologists thought they had done extensive ablation in the inferior triangle of Koch. However, the most annular aspect of the triangle of Koch was missed. The author's anecdotal experience has been that if the antegrade slow pathway has been injured by ablation but 1:1 antegrade conduction continues and slow/fast AVNRT is now maintained by a

very long AH interval, the site in the triangle of Koch that was missed is often the site in close proximity to the tricuspid annulus as shown in **Figure 7.14**.

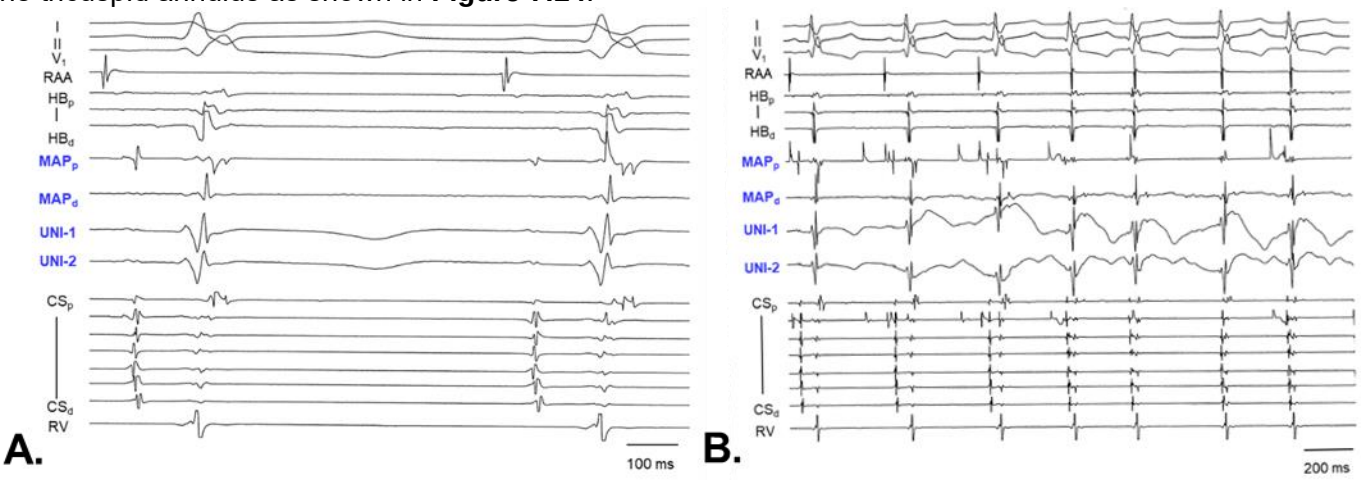


Figure 7.14. Ablating the RIE from the annular side of the inferior triangle of Koch. A. Note that the mapping catheter recorded nearly no atrial potential on both the distal and proximal unipolar electrode (UNI-1 and UNI-2). It appears that the tip of the ablation catheter was in the RV. The proximal electrode pair recorded a large atrial potential, indicating that the tip electrode is not deep in the ventricle. **B.** Ablation there elicited junctional automaticity. The RIE was badly injured but not eliminated after this RF application. A few more RF applications between here and CS ostium eliminated both antegrade RIE conduction and AVNRT.

In some patients, a slow pathway potential (Asp) can be recorded in the inferior triangle of Koch (**Figure 7.15; 7.3C**). In most cases, Asp is a relatively sharp potential preceded by a rounded, far-field atrial potential that represents far-field atrial activation posterior to the tendon of Todaro (**Figure 7.15B**). In other cases, the atrial potential and Asp form a multi-component complex potential (**Figure 7.15C**). Ablation targeting the Asp usually elicits junctional automaticity and eliminates the RIE.

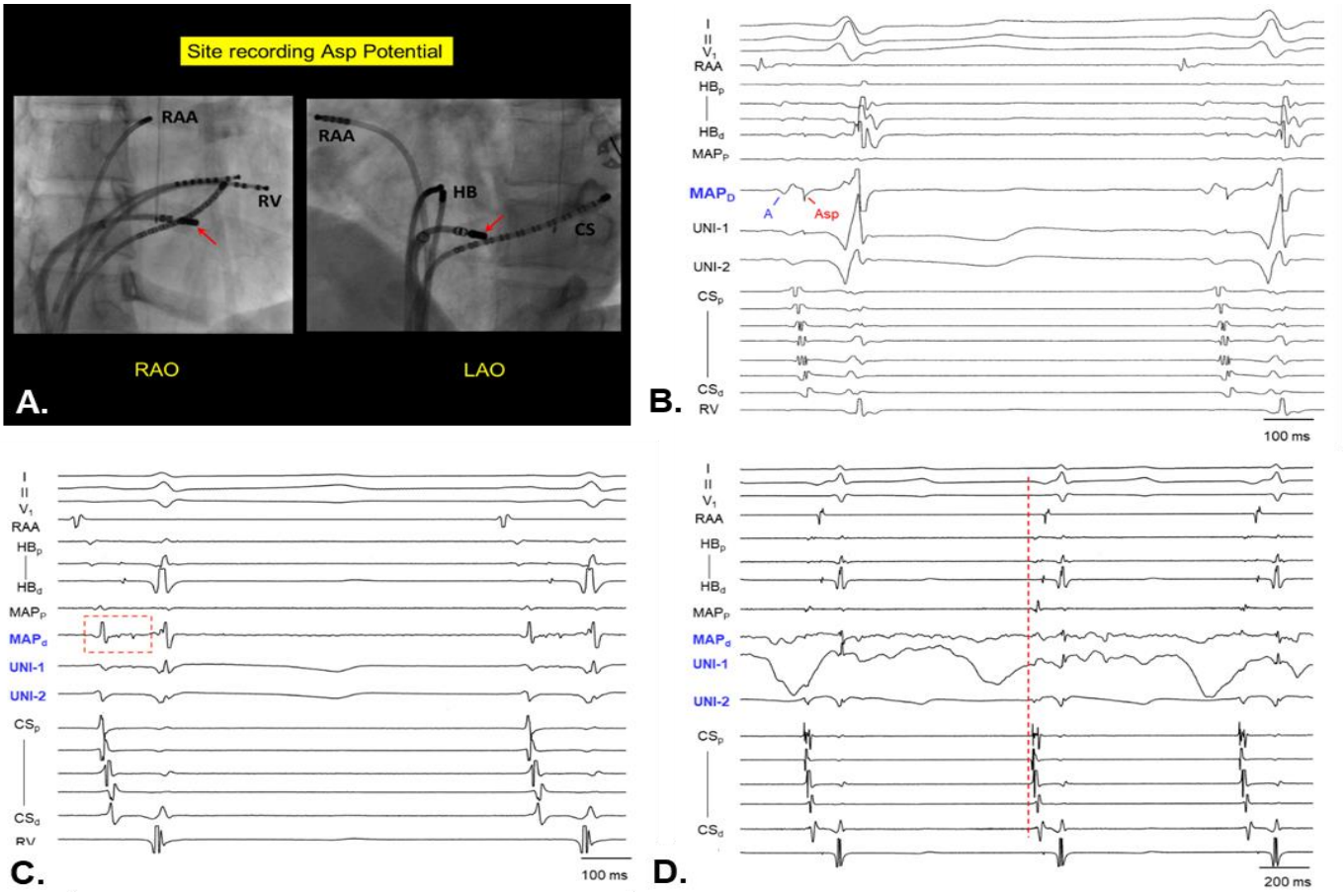


Figure 7.15. Ablation of RIE by targeting the slow pathway potential (Asp). A. In a patient with a large funnel-shaped CS ostium, radiographs of the typical location (red arrow) recording an Asp in the inferior triangle of Koch are shown. **B.** Asp is often preceded by a far-field atrial potential that is caused by atrial activation behind the tendon of Todaro. **C.** In some cases, the Asp and atrial potential form a multi-component complex potential (in red box). **D.** Ablation there elicited junctional automaticity. Vertical red line: earliest atrial activation.

The most common reason for failure to eliminate the RIE is poor electrode-tissue contact. This is usually due to catheter being pushed away from the septum by a prominent Eustachian ridge. This problem is recognized fluoroscopically in the LAO projection as a rightward movement of the catheter tip (**Figure 7.16A**). Obviously, if the operator ablates sites superior to the roof of the CS ostium, a prominent Eustachian ridge is no longer a concern but the risk of AVN injury is higher. An SL2 sheath can be used to get around a prominent Eustachian ridge. In this situation, the operator may need to impart gentle counterclockwise torque on the SL2 sheath but impart clockwise torque to the ablation catheter in order to provide good electrode-tissue contact (**Figure 7.16B**). This allows the SL2 sheath to bypass the Eustachian ridge in order for the ablation catheter to touch the inferior triangle of Koch This approach sounds a bit counterintuitive but is practically very effective. To determine electrode-tissue contact, one should use the LAO view, not the RAO view.

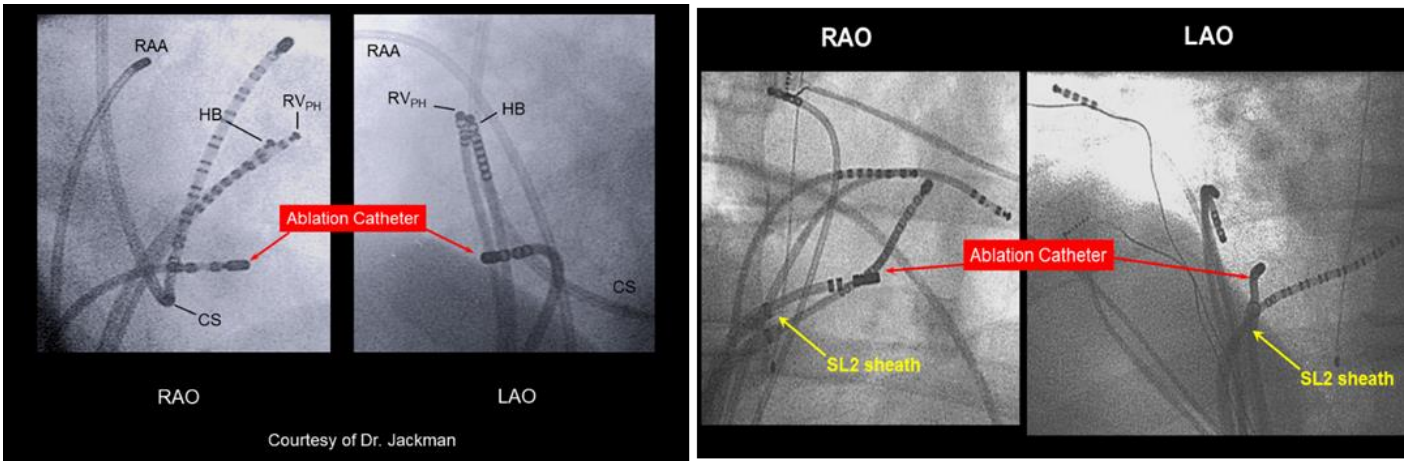


Figure 7.16. **Poor electrode-tissue contact in RIE ablation. A**. The ablation catheter appeared to have stable contact with the inferior triangle of Koch in the RAO view but the LAO view revealed that the ablation catheter was pushed away from the septum by a prominent Eustachian ridge. Courtesy of Dr. Jackman. **B**. To get around the problem, an SL2 sheath was used to bypass the ridge in another patient.

Catheter Ablation of "Leftward Inferior Extension" Slow/Fast AVNRT

In approximately 5% of patients with slow/fast AVNRT, ablation between the tricuspid annulus and CS ostium produces junctional automaticity, suggesting injury to the RIE but fails to eliminate AVNRT because the RIE is a bystander. Dr. Jackman will deliver atrial extra-stimuli from the anatomical location of the LIE along the roof of the proximal CS to verify that the LIE is the antegrade limb of the reentrant circuit (see discussion below). Then, RF applications are delivered to the site approximately 4 cm from the CS ostium, continuing proximally toward the roof of CS ostium (**Figure 7.4; 7.13B**). Ablation is usually discontinued when the tip electrode is approximately 2 cm distal to CS ostium to avoid AVN injury because the ablation catheter positioned at this location tends to push upward. Notably, ablation of the LIE can terminate this type of AVNRT but junctional automaticity during LIE ablation is usually *not* observed.

Catheter Ablation of "Left Atrial" Slow/Fast AVNRT

In less than 1% of the patients with slow/fast AVNRT, ablation between the tricuspid annulus and the CS ostium (RIE) and along the roof of the proximal CS (LIE) fails to eliminate AVNRT. In most of these patients, the slow pathway participating in slow/fast AVNRT is located in the left atrium close to the inferolateral mitral annulus (but not on the annulus). Basically, the entire reentrant circuit is located in the LA. However, one cannot differentiate this rare form of slow/fast AVNRT from the common form of AVNRT (RIE) by EGMs alone (**Figure 7.11A, 7.17A**).

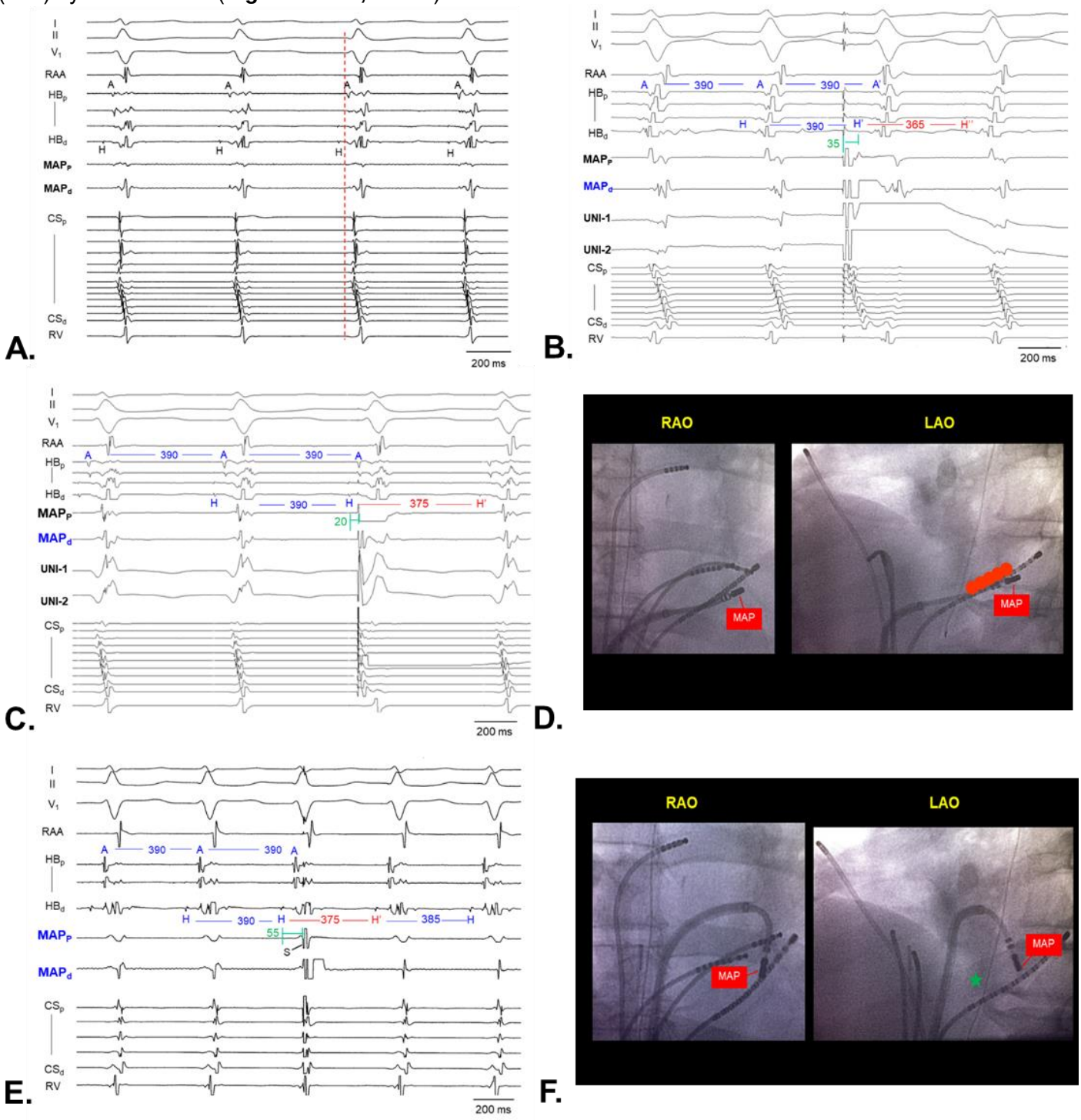


Figure 7.17. Ablation of slow/fast AVNRT using a left atrial slow pathway for antegrade conduction. A. This slow/fast AVNRT looks no different from a regular slow/fast AVNRT using the RIE for antegrade ablation. However, prior ablations were not successful after extensive ablation in the inferior triangle of Koch. **B.** In AVNRT, a series of single atrial extra-stimuli (AES) were delivered to the inferior triangle of Koch where the RIE is located. AES could not reset the tachycardia until it was 35 ms *before* the HB potential. Note that the atrial activation timing recorded in the HBp was not affected by the AES (A-A' interval = 390 ms),

indicating that AES perturbed the tachycardia through the antegrade slow pathway but RIE was not the slow pathway participating in AVNRT. **C-D.** The mapping catheter was positioned in posterolateral CS adjacent to the presumed location of the LIE. The latest AES that could reset the tachycardia was 20 ms *after* the HB potential, indicating that this site is closer to the antegrade slow pathway than the RIE. However, a pull-back RF application (red dots) targeting the LIE did not affect AVNRT. **E-F.** The mapping catheter was positioned in posterolateral LA. The latest AES that could reset the tachycardia was 55 ms *after* the HB potential, indicating that this site is in close proximity to the antegrade slow pathway of the tachycardia. Ablation here elicited junctional automaticity and eliminated antegrade slow pathway conduction and AVNRT. In the OU-EP laboratory, this rare form of slow pathway was successfully ablated by targeting its septal aspect (green asterisk) in 2/14 patients.

Using resetting responses to localize the left atrial slow pathway

This pacing maneuver is based on the rationale that at a site close to the reentrant circuit, even a late atrial extra-stimulus (AES) will easily engage the reentrant circuit and reset the tachycardia. On the other hand, a site distant from the circuit will require a very early AES to engage the reentrant circuit. A late AES (e.g. after the onset of the HB potential) is delivered to the inferolateral mitral annulus. A legitimate AES is the one that does not affect the timing of the atrial activation recorded in the HB area to ensure that the retrograde fast pathway is not perturbed (**Figure 7.17B-E**). An advance in the next HB potential by ≥5-10 ms and resetting of the tachycardia indicate that the AES site is close to the antegrade slow pathway of the reentrant circuit. After comparing the resetting responses elicited from different sites in the LA, the site where the latest AES can reset AVNRT is chosen as the ablation target. Ablation at this site frequently produces junctional automaticity with retrograde fast pathway conduction very similar to ablation of the RIE. The left atrial slow pathway and AVNRT are usually eliminated afterwards. The anecdotal experience of the OU-EP group is that the left atrial slow pathway tends to be located at the 4 o'clock position along the mitral annulus as viewed in the LAO projection. Of note, this type of slow pathway is not located on the mitral annulus as a left free wall AP. It is not clear if the ligament of Marshall is related to this type of slow pathway. There were two other cases in which the left atrial antegrade slow pathway was successfully ablated at more septal sites (green asterisk in **Figure 7.17F**), probably by eliminating the septal aspect of this rare form of slow pathway. However, ablation at this septal site carries a higher risk of AVN injury.

Catheter Ablation of Slow/Slow AVNRT

Dr. Jackman's approach to ablation of slow/slow AVNRT is to first map the retrograde slow pathway, which is usually but not always the LIE (**Figure 7.18**). RF applications are delivered to the site of earliest retrograde atrial activation, which is usually located along the roof of the proximal CS (LIE) and less commonly in the inferior triangle of Koch (RIE). He may target the retrograde slow pathway first, followed by anatomically ablating the antegrade slow pathway (RIE). In other instances, he may anatomically ablate the RIE first. One may ablate retrograde slow pathway conduction during ventricular pacing, rather than during tachycardia, to avoid upward movement of the ablation catheter when AVNRT terminates. However, if 1:1 retrograde slow pathway conduction cannot be maintained by RV pacing, ablation may have to be performed during AVNRT with precaution. Since retrograde fast pathway conduction is either absent or poor in many patients with slow/slow AVNRT, junctional automaticity with VA block may be observed. With this in mind, operators should have atrial pacing set up before ablation in order to monitor antegrade fast pathway conduction during ablation as soon as accelerated junctional rhythm with VA block occurs. After the retrograde slow pathway (usually LIE) conduction is eliminated, ablation of the RIE in the inferior triangle of Koch is performed to eliminate the antegrade limb of the reentrant circuit.

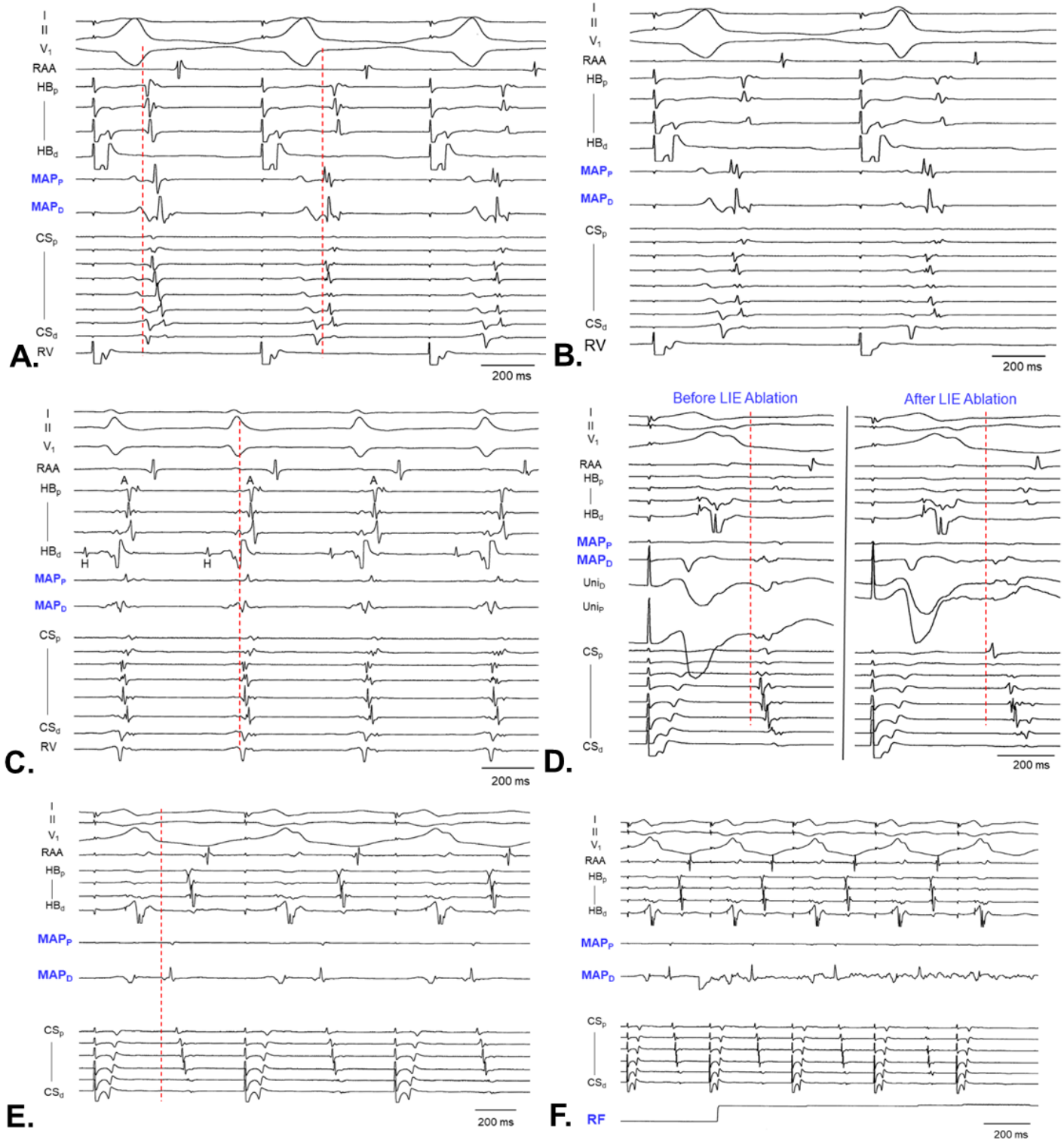


Figure 7.18. Ablation of slow/slow AVNRT. A. Decremental RV pacing resulted in changes in site of earliest atrial activation from the anterior septum to posterior septum. Mapping catheter was positioned along the roof of the proximal CS near the LIE. Vertical red line: earliest atrial activation timing. **B.** Parahisian pacing verified that the 2nd atrial activation was retrograde AVN slow pathway. **C.** Slow/slow AVNRT was then induced. The atrial activation sequence was identical to that during parahisian pacing. Ventricular extra-stimuli also proved that this was an AVNRT (not shown). **D. Left panel.** Mapping inside the CS and in the inferior triangle of Koch identified that the site of earliest atrial activation was along the roof of the proximal CS, 3 cm distal to the CS ostium, indicating that retrograde conduction was mediated by the LIE. **Right panel.** After ablation of the LIE, the atrial activation sequence changed. The floor of the CS ostium was now earliest. **E.** Mapping showed that the site of earliest atrial activation was at the CS ostium (RIE). **F.** Ablation at that site quickly caused VA block (last beat). AVNRT was no longer inducible. There was no antegrade or retrograde slow pathway conduction after ablation.

Elimination of retrograde slow pathway conduction and the inducibility of AVNRT can be achieved in almost all patients with slow/slow AVNRT. In Dr. Jackman's early experience, the recurrence rate after RIE ablation alone was high (8%). The recurrent tachycardia was usually slow/fast AVNRT using another slow pathway for antegrade conduction. These results led to Dr. Jackman's current practice of ablating both the antegrade and retrograde slow pathway participating in slow/slow AVNRT; in other words, both the RIE and LIE.

Catheter Ablation of Fast/Slow AVNRT

Similar to slow/slow AVNRT, the target for ablation in fast/slow AVNRT is the retrograde slow pathway (usually but not always the RIE) used in the tachycardia, followed by ablation of the antegrade slow pathway (usually the LIE). RF energy is delivered to the site of earliest retrograde atrial activation, between the tricuspid annulus and CS ostium, where the RIE is located. In some patients, a retrograde slow pathway potential (A_{SP} potential) can be recorded. When present, ablation there has the greatest likelihood of eliminating retrograde slow pathway conduction. The amplitude of the Asp recorded here is usually low and may have multiple component as a result of the far-field potential from the RA posterior to the tendon of Todaro. With this approach, the recurrence rate is approximately 1-2%. Again, ablation along the floor of the CS should be avoided to prevent coronary artery injury.

Endpoint of AVNRT ablation

Dr. Jackman's endpoint for successful ablation includes: 1) elimination of AVNRT. Single slow/fast atrial echo beats are acceptable because they may be indicative of another slow pathway (e.g. left atrial slow pathway) not participating in the reentrant circuit. 2) elimination of 1:1 antegrade slow pathway conduction during decremental atrial pacing. For slow/slow and fast/slow AVNRT, 1:1 retrograde slow pathway conduction should be eliminated as well.

Dr. Jackman's approach to difficult AVNRT ablation (including failed prior ablation)

1. Verify the diagnosis first

a. Septal AT misdiagnosed as slow/fast AVNRT or vice versa.

b. Orthodromic AVRT using a concealed anteroseptal AP for retrograde conduction was misdiagnosed as slow/fast AVNRT or vice versa

C. Orthodromic AVRT using a concealed posteroseptal AP for retrograde conduction was misdiagnosed as slow/slow or fast/slow AVNRT or vice versa.

D. Fascicular VT or high septal VT misdiagnosed as AVNRT with aberrant conduction.

E. Atrio-fascicular AVRT (Mahaim) misdiagnosed as AVNRT with aberrant conduction.

2. Map the site of earliest atrial activation to determine which type of AVNRT it is

This is a very important step of treating challenging cases of AVNRT. Knowing the type of AVNRT provides operators the roadmap to the ablation target. Having a full grasp of the AVNRT reentrant circuits that Dr. Jackman has proposed may be a challenging task; however, these hypothesized circuits are based on his three decades of experience in eliminating difficult AVNRT. If it is slow/slow or fast/slow AVNRT, mapping and targeting the retrograde slow pathway conduction has the highest likelihood of eliminating the tachycardia. One of the most common mistakes is to interpret slow/slow AVNRT as slow/fast AVNRT because the HA interval is short. One of the lessons that the author has learned is that if the atrial activation

timing in the HB area is clearly earlier than that of proximal CS, it is mostly likely slow/fast AVNRT. If the activation timing in the HB area is slight earlier than that in the proximal CS, spending a few minutes mapping the fast pathway area and the RIE and LIE area is well worth the time.

3. Use resetting responses in slow/fast AVNRT to determine which slow pathway participates in this slow/fast AVNRT.

a. Start with delivering late AES from the LIE, followed by the RIE area. The area that the resetting response should cover is similar to the area that ablation of RIE or LIE should cover as described earlier. The site where the latest AES can reset the AVNRT is in close proximity to the slow pathway serving as the antegrade limb of the slow/fast AVNRT. Typically, the target site is where an AES is at least 30 ms *later* than the timing of the HB potential (**Figure 7.17**). Good electrode-tissue (RIE) contact must be achieved in the presence of a prominent Eustachian ridge.

b. If *late* AES cannot reset the AVNRT from either the RIE or LIE, trans-septal puncture is performed to assess the resetting response along the posterolateral mitral annulus.

Figure 7.19 and 7.20 illustrate representative examples of using the resetting responses to find the antegrade slow pathway participating in slow/fast AVNRT that is difficult to ablate. Sometimes, elderly patients have a prolonged AH interval in the baseline state. Dr. Jackman's experience has been that if the retrograde fast pathway conduction is normal, ablating the antegrade slow pathway does not cause a higher degree of AV block. Certainly, using the resetting response to identify the culprit slow pathway is a plausible approach in this situation.

The author expedites the resetting response in the following way:

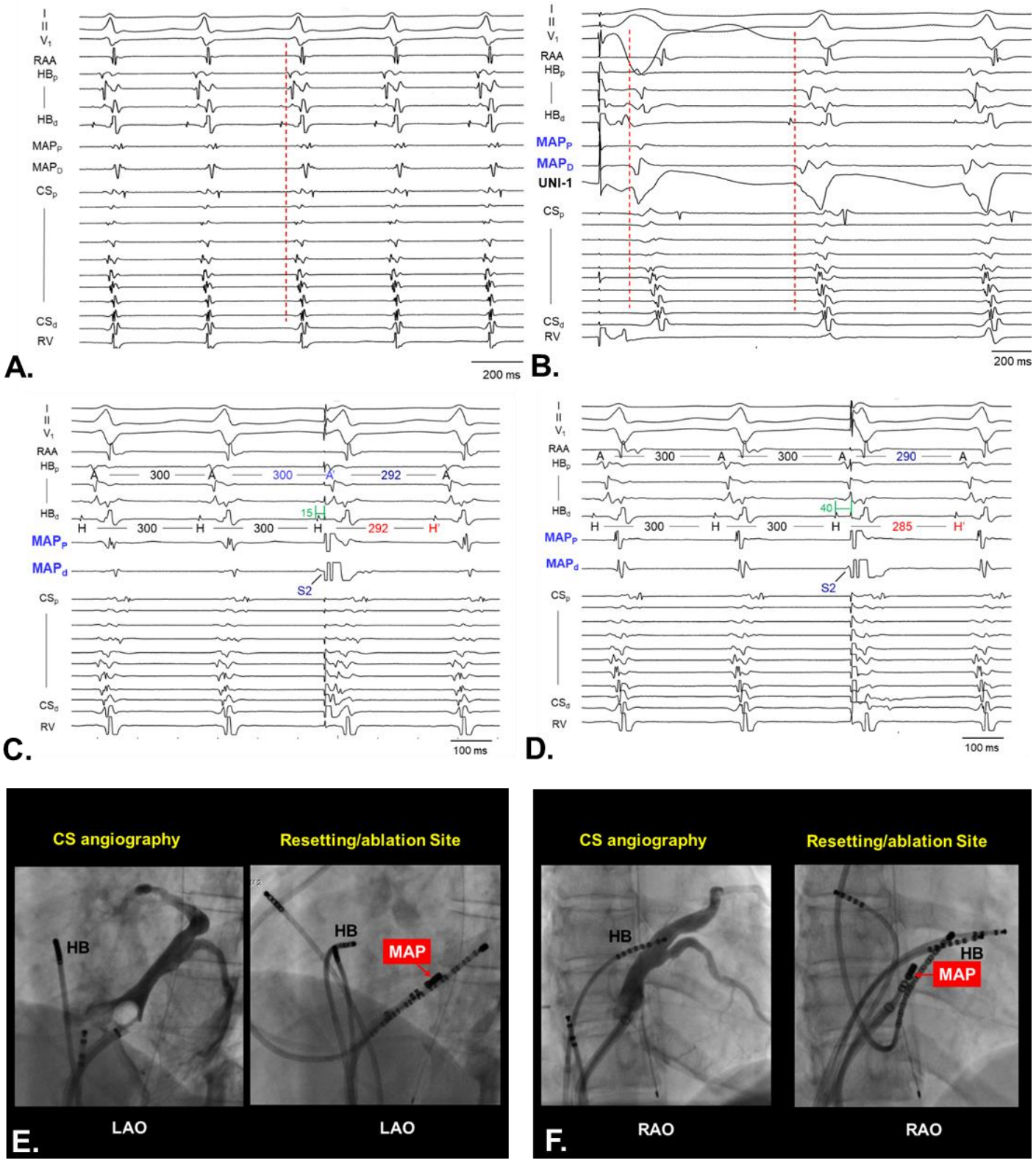
a. After verifying that it is slow/fast AVNRT, single AES are delivered to the LIE first, followed by the RIE. The stimulator is programmed to deliver the 1st stimulus 40 ms after the onset of the HB potential, followed by progressively earlier AES by a decrement of 5 ms. If AES 20 ms after the inscription of the HB potential still fails to reset AVNRT, it is not likely that the LIE or the left atrial slow pathway is used as the antegrade limb of this slow/fast AVNRT. Single AES will then be delivered to the RIE area. The anecdotal experience of the OU-EP group is that if the site of interest is in close proximity to the culprit slow pathway, it is rare to require an AES that is within 30 ms following the inscription of the HB potential.

b. If RIE resets better than LIE, ablation targeting the RIE is then performed. If the patient had prior failed ablation or ablation carries a higher risk of AVN injury (e.g. short distance between the CS ostium and HB potential), resetting responses are evaluated at several sites in the RIE to pinpoint the slow pathway. It is very common that only one RF application is needed to eliminate the last strand of RIE in patients who had extensive RIE ablation in prior ablation procedures.

c. If LIE resets better than RIE, a few more sites in the LIE area are tested to pinpoint the location of the LIE. If LIE ablation fails to eliminate AVNRT, trans-septal puncture to map the posterolateral LA is the next step.

d. The OU-EP group has had only limited experience (1 or 2 cases) in the superior form of slow/fast AVNRT. Assessing resetting responses may or may not work for this form of AVNRT.

4. Target the site of earliest atrial activation during AVNRT or RV pacing to treat slow/slow or fast/slow AVNRT as already discussed. Dr. Jackman would ablate both the RIE and LIE.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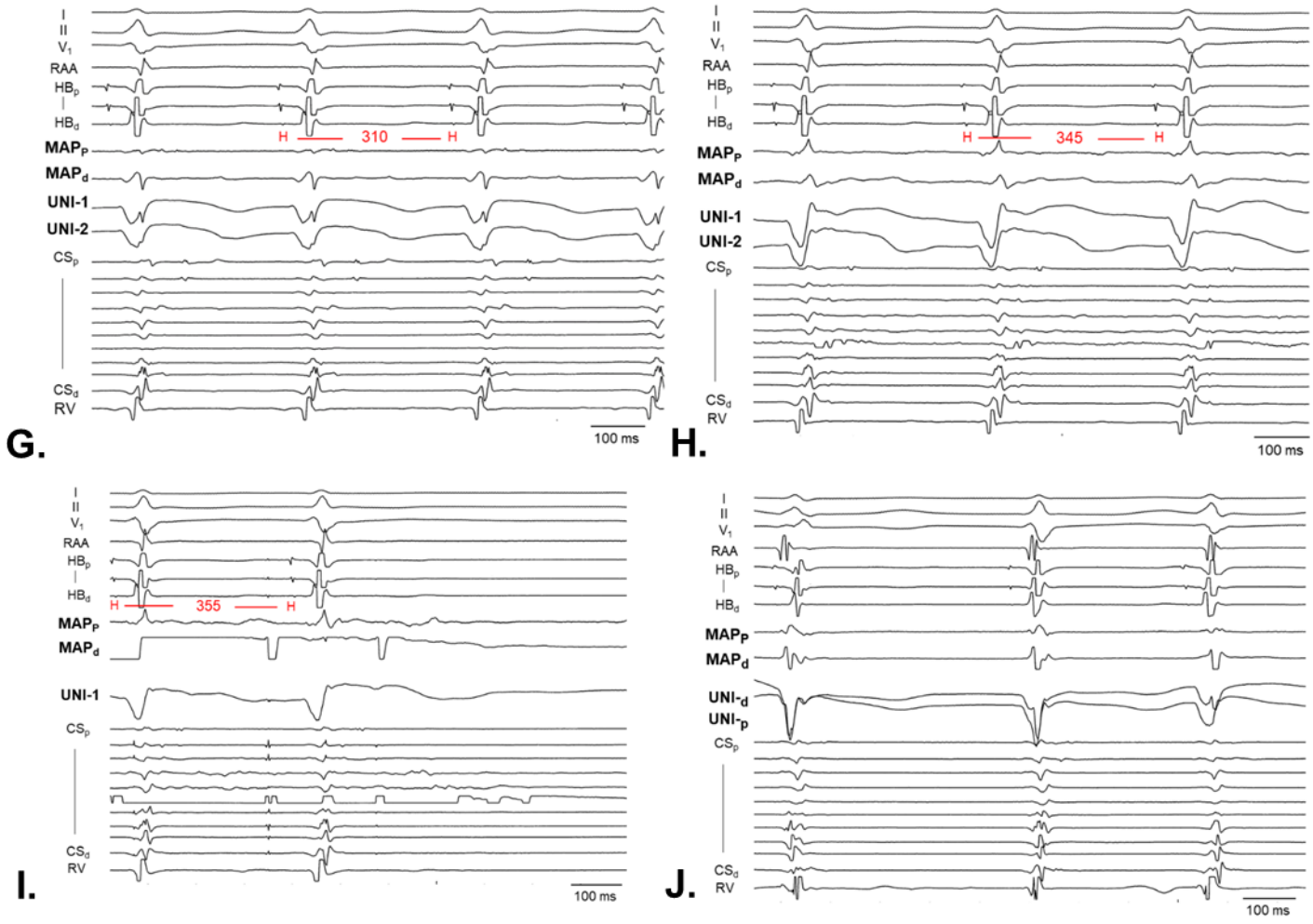
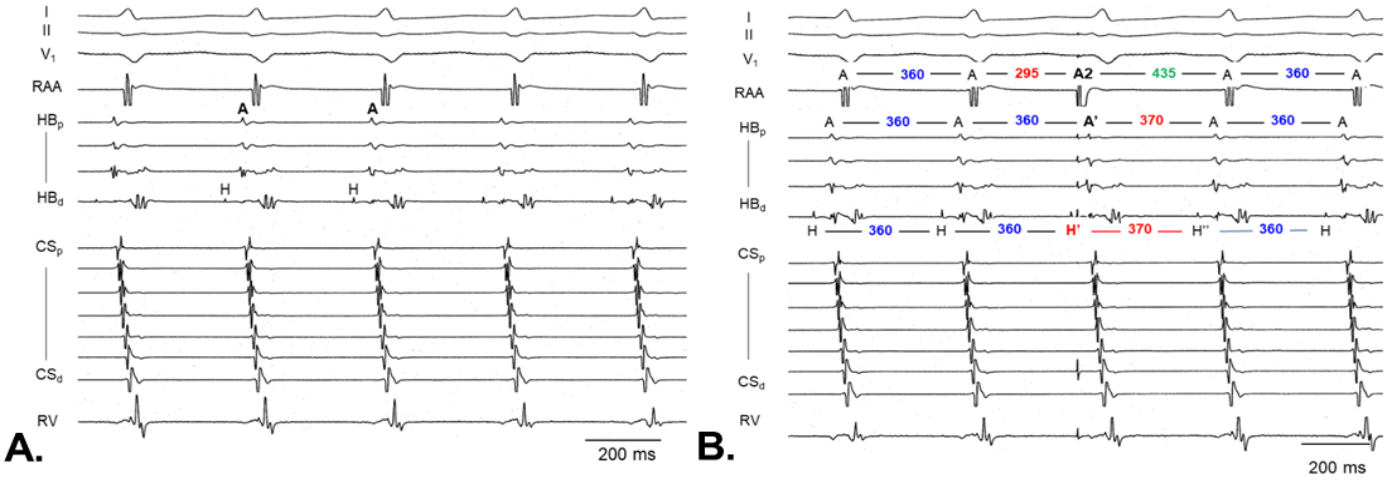
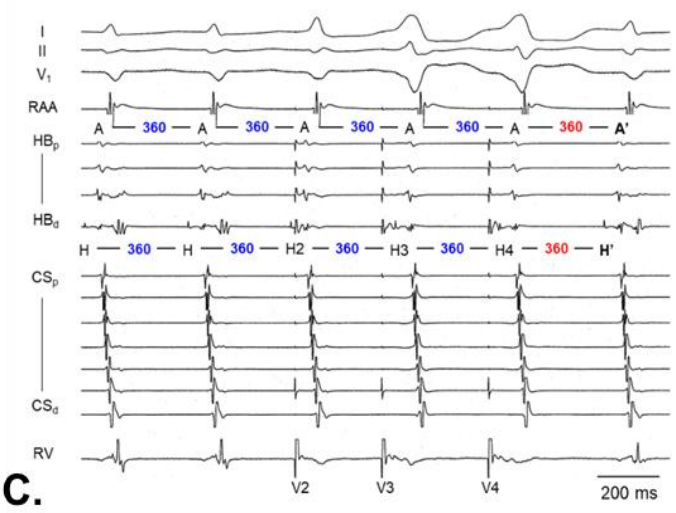


Figure 7.19. A young woman with two prior AVNRT ablations. A. An SVT was easily induced. Ventricular extra-stimuli and RV overdrive pacing verified that the SVT was AVNRT (not shown). Site of earliest activation (vertical red line) was in the HB area. **B.** Mapping during AVNRT showed that the site of earliest atrial activation was in the fast pathway area behind the tendon of Todaro (same figure as **Figure 7.2B**), consistent with slow/fast AVNRT. **C.** The *latest* AES delivered from the RIE that reset the tachycardia was 15 ms after the HB potential. Note that the atrial timing recorded on the HBp bipolar electrodes was not affected by the AES (A-A'=300 ms), indicating that retrograde fast pathway conduction was not perturbed by AES. AES advanced the next HB potential by 8 ms (H-H'=292 ms). **D.** The *latest* AES delivered from the LIE that reset the tachycardia was 40 ms after the HB potential. AES advanced the next HB potential by 15 ms (H-H'=285 ms). **E-F.** Radiographs demonstrate the position of the mapping catheter at the LIE. **G-I.** Before ablation of the LIE, the AH interval was 310 ms, which progressively lengthened to 315 ms and 345 ms before AVNRT was terminated. **J.** After AVNRT termination, ablation induced junctional automaticity. Of note, ablation of the LIE usually does *not* elicit junctional automaticity.

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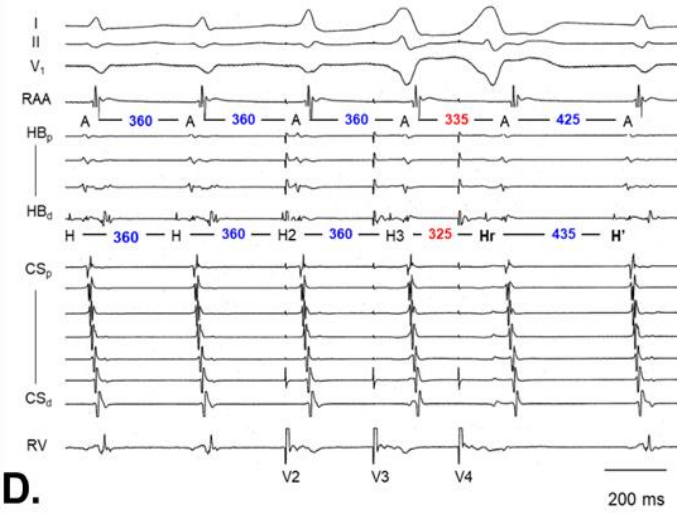
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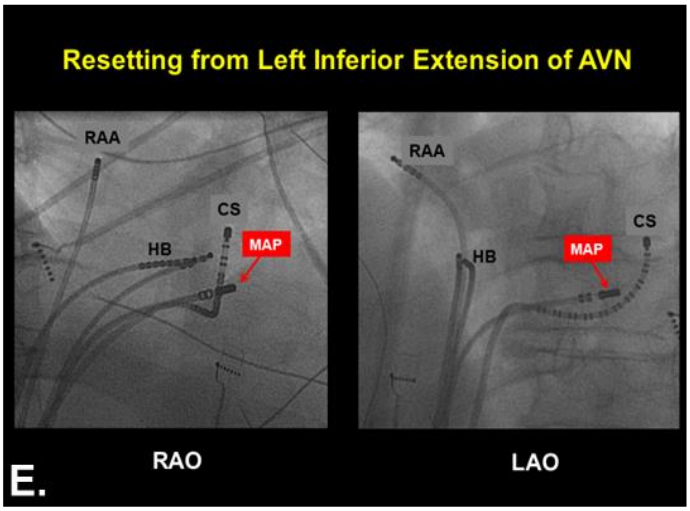
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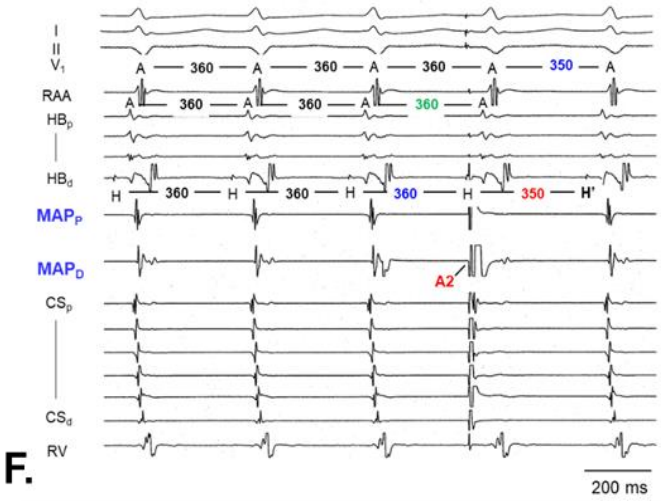
E.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A2
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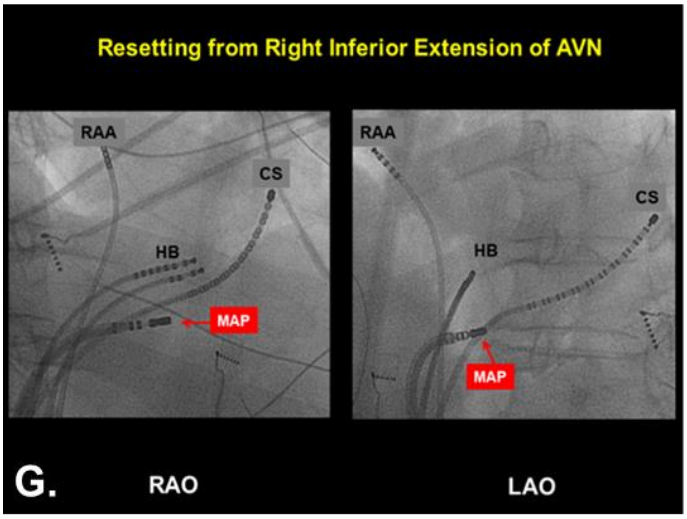
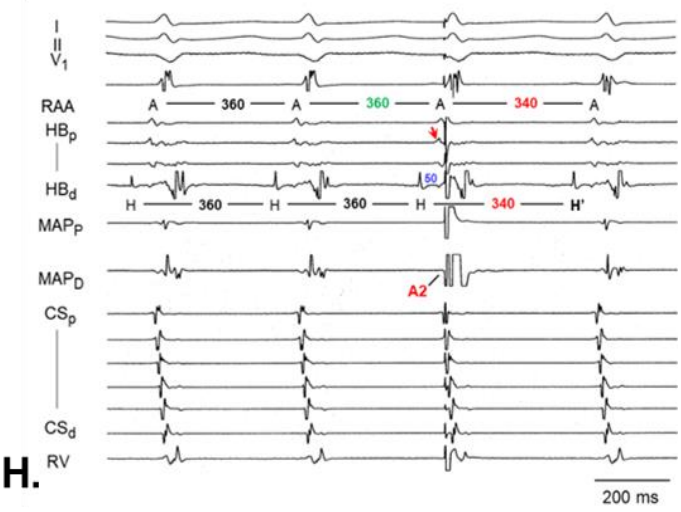
F.


Resetting from Right Inferior Extension of AVN
RAA
CS
HB
MAP
RAO
LAO

G.


I
II
V1
RAA
HBp
HBd
MAPP
MAPD
CSp
CSd
RV
A 360 A 360 A 340 A
H 360 H 360 H 340 H'
50
A2
200 ms

H.

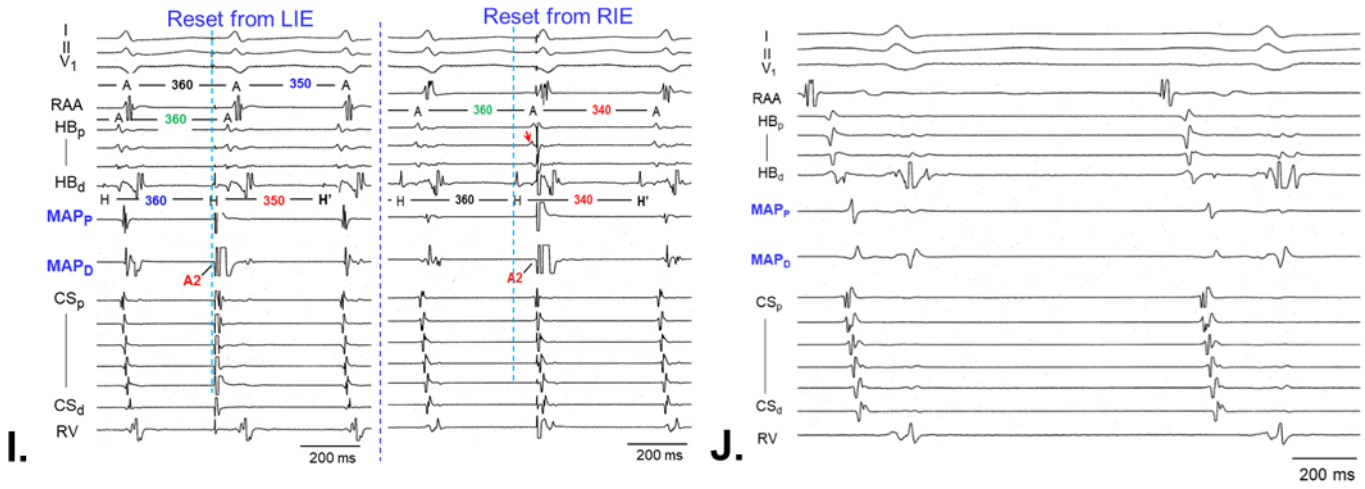


Figure 7.20. Stepwise approach to difficult slow/fast AVNRT ablation. This patient had prior slow/fast AVNRT ablation. A. An SVT (CL=360 ms) was easily induced by RA burst pacing. The VA interval measured at HBp electrodes was zero, excluding AVRT using a concealed anteroseptal AP for retrograde conduction. **B.** An atrial extra-stimulus (AES) was delivered to the RAA 65 ms earlier. Note that AES did not affect the timing of the atrial activation (A') recorded on the HB catheter (A-A'=360 ms) as well as the timing of the HB potentials (H-H'=360 ms). However, A2 delayed the next HB potential (H'') by 10 ms (H'-H''=370 ms) and reset the tachycardia, excluding the diagnosis of junctional tachycardia. It is noteworthy that A2 also makes the diagnosis of focal atrial tachycardia very unlikely because the timing of the earliest atrial activation (recorded by the HB catheter) was not affected by A2 but the tachycardia was reset. This pacing maneuver is helpful in differentiating focal atrial tachycardia from AVNRT with persistent 2:1 block. **C.** Three ventricular extra-stimuli (VES) were delivered to the anteroseptal RV near the site of the earliest atrial activation. Despite significantly advanced the local ventricular activation time, V3 and V4 did not affect the timing of the HB potential. Note that H2, H3 and H4 were all resulted from antegrade HB activation. **D.** V2 and V3 advanced the local ventricular activation but the HB potentials (H2 and H3) were still resulted from antegrade HB conduction. In contrast, V4 advanced the retrograde HB timing (Hr) by 35 ms which then advanced the atrial activation timing by 25 ms. The Hr-H' timing was 435 ms, indicating that V4 had reset the SVT and proved that this SVT was AVNRT. **E-F.** Radiographs of the mapping catheter positioned at the LIE. Progressively earlier AES (A2) were delivered to the LIE without affecting the tachycardia. Finally, an AES delivered immediately after the HB potential advanced the next HB potential (H') by 10 ms, producing a positive resetting response. However, LIE was not likely the slow pathway participating in AVNRT. **G.** Radiographs of the mapping catheter positioned at the RIE. **H.** From the RIE, an AES was delivered at the inferior TOK, 50 ms after the HB potential. This late AES advanced the next HB potential by 20 ms and reset the tachycardia. Note that the atrial timing near the HB potential (red arrow) was not affected by A2, indicating that the antegrade slow pathway, not the retrograde slow pathway, was perturbed by the AES. This observation also indicated that the RIE was in close proximity to the antegrade slow pathway of the AVNRT. **I.** Side-by-side comparison of the resetting response from the LIE and RIE. Blue vertical line: beginning of the HB potential. **J.** At the site of the RIE where a positive resetting response was elicited, the EGM recorded on the ablation catheter (MAPd) did not look unique but ablation there eliminated the slow pathway (RIE) and AVNRT.

Chapter 8:
Ablation of Atrial Fibrillation

The mechanisms underlying the initiation, maintenance and progression of atrial fibrillation (AF) remain poorly understood. The sniper's approach therefore would not work for AF unless ablation targets are better defined. Set aside your sniper riffle, draw an AK-47 to sweep the target!

Chapter 9:

Ablation of Focal Atrial Tachycardia and Premature Ventricular Contraction

As described in **Chapter 5**, Dr. Jackman's preferred ablation target for accessory pathway (AP) ablation is where the distal ablation electrode records an AP potential. The grand strategy that Dr. Jackman maps a focal atrial tachycardia (AT) or premature ventricular contractions (PVC) is similar and can be summarized in two words: unipolar electrogram (EGM). Using minimally filtered unipolar EGMs (0.05 or 0.2 Hz to 400-500 Hz) is an integral part of Dr. Jackman's mapping strategy for all arrhythmias. The author has never seen Dr. Jackman mapping an arrhythmia without taking advantage of the timing and morphology of the unipolar EGM.

Basically, Dr. Jackman uses bipolar EGM to find the target and distal unipolar EGM (UNI-1) to fine-tune the target. The EGM at the origin of a focal arrhythmia should fulfill the following criteria (**Figure 9.1**):

1. There is no other site recording earlier near-field or far-field activation.

2. The distal *unipolar* EGM (UNI-1) and the distal *bipolar* EGM of the mapping catheter begin simultaneously.

3. At this site, the distal unipolar EGM begins with a steep, negative component (a QS or qS pattern). That is, there is no far-field potential earlier than the beginning of the distal unipolar EGM.

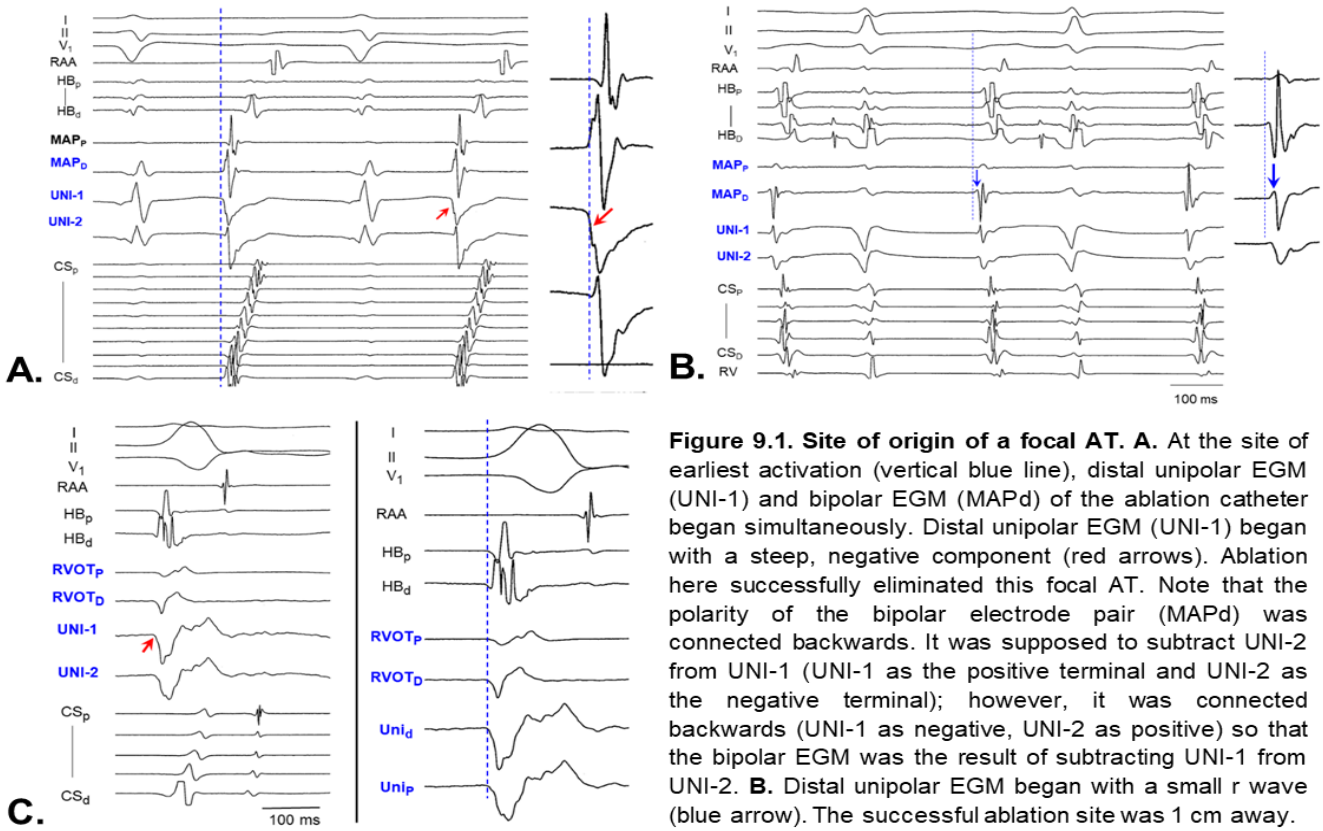


Figure 9.1. Site of origin of a focal AT. A. At the site of earliest activation (vertical blue line), distal unipolar EGM (UNI-1) and bipolar EGM (MAPd) of the ablation catheter began simultaneously. Distal unipolar EGM (UNI-1) began with a steep, negative component (red arrows). Ablation here successfully eliminated this focal AT. Note that the polarity of the bipolar electrode pair (MAPd) was connected backwards. It was supposed to subtract UNI-2 from UNI-1 (UNI-1 as the positive terminal and UNI-2 as the negative terminal); however, it was connected backwards (UNI-1 as negative, UNI-2 as positive) so that the bipolar EGM was the result of subtracting UNI-1 from UNI-2. **B.** Distal unipolar EGM began with a small r wave (blue arrow). The successful ablation site was 1 cm away. **C.** The distal unipolar EGM exhibits a QS pattern (**left panel**). However, the timing of both the bipolar and unipolar EGM at the earliest site in the RVOT area was only simultaneous with the onset of the QRS complex. The early far-field component of the distal unipolar EGM might be a low-frequency signal (<1 Hz) and was filtered out (filter setting: 1-400 Hz in this case). **Right panel**: enlarged view of the left panel.

As discussed in **Chapter 2**, the distal unipolar EGM and distal bipolar EGM of the mapping catheter are filtered differently. In the OU-EP laboratory, the high-pass and low-pass filter for the *unipolar* EGMs are set at 0.1-1 and 400-500 Hz respectively. The high-pass and low-pass filter for the *bipolar* EGMs are set at 25-30 and 250-400 Hz, respectively. The discrepancy of filter setting can lead to significant confusion when the operator interprets the beginning of the unipolar and bipolar EGM. It is not uncommon that the operator identified a site where the unipolar EGM showed a QS pattern but the timing of the bipolar EGM was not early (**Figure 9.1C**). This is because the low frequency far-field signal in the unipolar EGM was filtered out if the high-pass filter was set at 1 Hz. The remaining higher frequency components happened to start with a QS morphology. Another possibility is that the gain of the unipolar EGM was too low to recognize a far-field potential with a very small amplitude. The author prefers to set the unipolar EGM filter to be 0.1-500 Hz when mapping of a focal arrhythmia is expected in order to avoid filtering out the low frequency potential.

As mentioned in **Chapter 1**, Dr. Jackman relies heavily on the triggered sweep function of the Bard recording system (now Boston Scientific) to map a focal arrhythmia (**Figure 1.1)**. Although electro-anatomical mapping can be very helpful, Dr. Jackman maps a focal arrhythmia mainly based on the timing and morphology of the EGM to select the ablation target. Electro-anatomical mapping only serves as a tool to remind him of the location of each EGM. When Dr. Jackman uses CARTO to map a focal arrhythmia, the local timing of each point is determined by the *first*, sharp down-stroke component (largest dV/dt) of the distal unipolar EGM of the mapping catheter, not the earliest timing of the bipolar or unipolar EGM (**Figure 9.2**). This practice is to ensure that the timing of each point reflects the true local activation timing. After detailed mapping, the true site of earliest activation will be evident. Sometimes, there is a large area of early activation. After carefully examining these “early” points, if there is no point fulfilling the 3 criteria listed above, the origin of this focal arrhythmia is unlikely to be on the endocardial surface in this area. Dr. Jackman will consider mapping the epicardial surface or the neighboring chamber. For example, if the earliest EGM in the posterior aspect of the RVOT does not fulfill the 3 criteria, Dr. Jackman will map the coronary cusp before ablating the RVOT target (**Figure 9.3**). Of note, if both unipolar electrodes (UNI-1 and UNI-2) record the same potential, this potential is most likely to be far-field (**Figure 9.4A**).

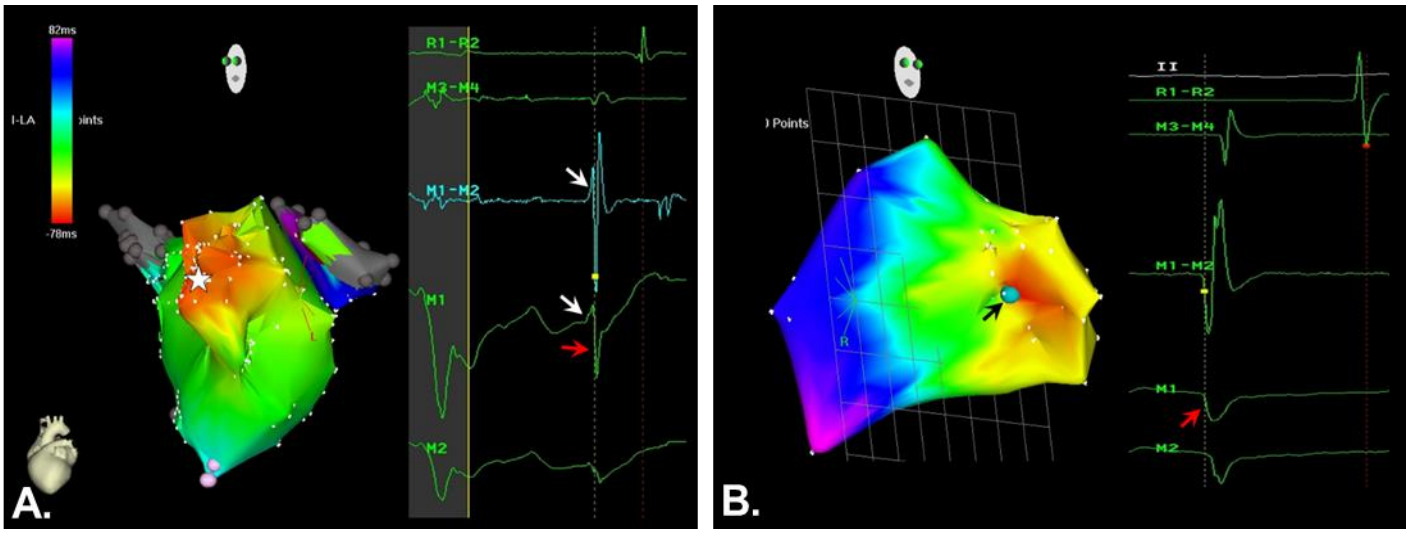


Figure 9.2. Using unipolar EGM to select ablation target. A. In a young woman with a focal AT originating from the epicardial surface of the LA appendage, endocardial mapping of the LA appendage showed a large area of “early” activation. All the points with an early activation timing began with a far-field potential (white arrows) preceding the first, sharp negative component of the distal unipolar EGM (red arrow). **Right panel.** EGMs in the right panel correspond to the white star on the CARTO map. **B.** Epicardial mapping identified the site of earliest activation. Distal unipolar EGM and bipolar EGM of the mapping catheter began simultaneously. Distal unipolar EGM showed a QS pattern (red arrow). Ablation here immediately eliminated the tachycardia.

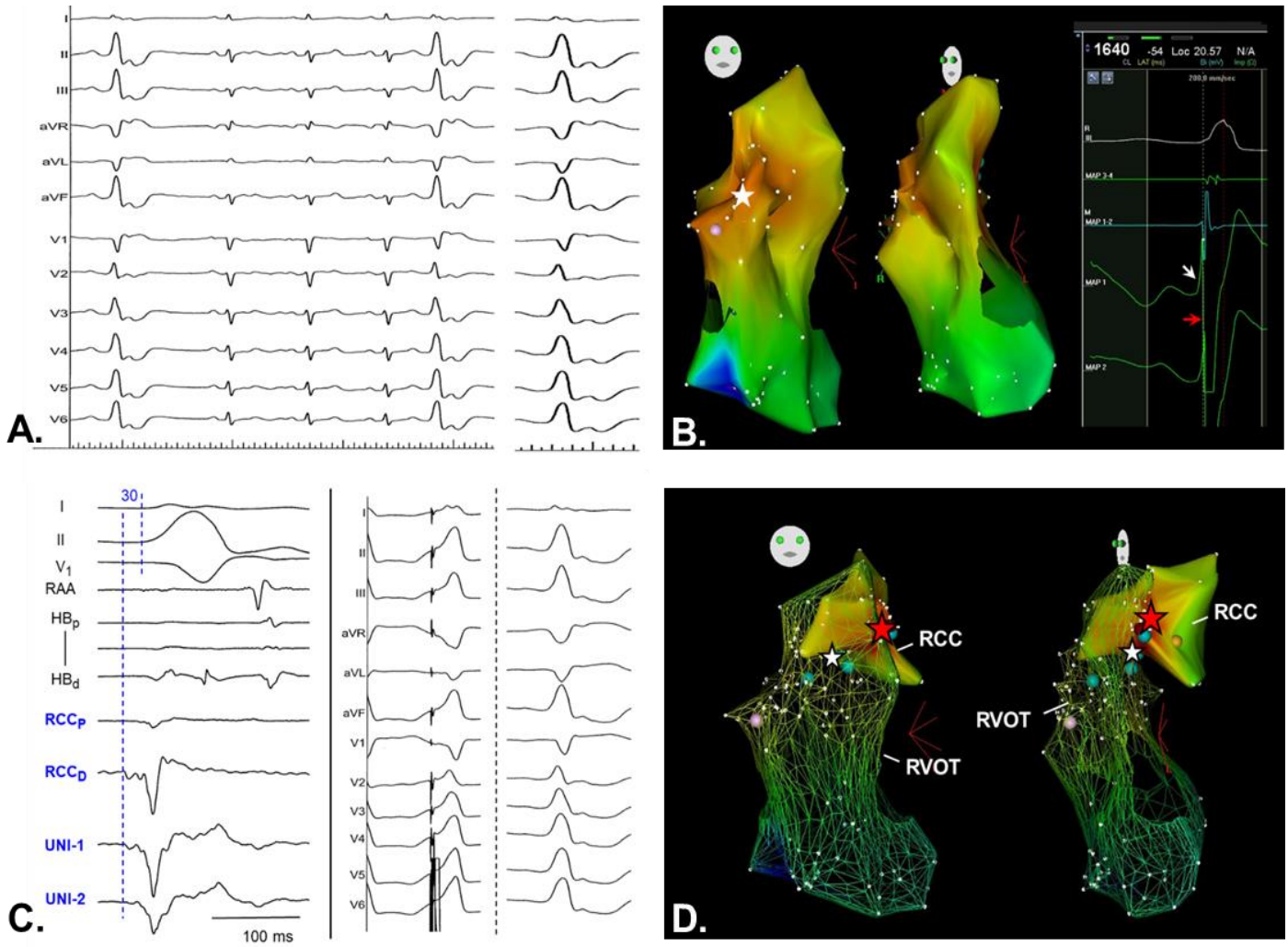


Figure 9.3 PVCs originated from the right coronary cusp (RCC). A. PVCs had a low-amplitude, M-shaped R wave in lead I. Precordial transition of PVCs was earlier than that in sinus rhythm. Note ST elevation in V1 and deep S wave in II, III, aVF and V3-V6. The ECG filter was set to be 1-100 Hz by mistake. **B.** Activation mapping of the RVOT area identified an area that was diffusely early. The distal unipolar EGM of the "earliest" site (white star) began with a far-field potential (white arrow). The local activation timing (red arrow) was 10 ms later than the beginning of the far-field potential. **C. Left panel.** At the site of successful ablation in the RCC, both the bipolar and distal unipolar EGM were 30 ms earlier than the onset of the QRS complex. The distal unipolar EGM began with a small q wave. **Right Panel.** Pace mapping at the successful ablation site only produced 90% match. **D.** Site of earliest activation recorded from the RVOT (white star) and from the RCC (red star).

The sharp component of the distal unipolar EGM, representing the activation of the tissue beneath it, should greatly diminish or be eliminated if ablation is effective. If most of the amplitude of the bipolar EGM is contributed by the proximal unipolar EGM (UNI-2), ablation at that site may not affect the amplitude of the bipolar EGM and may mislead the operator to deliver longer RF applications. Therefore, the OU-EP group almost never uses reduction of the amplitude of the *bipolar* EGM to evaluate ablation efficacy (**Figure 9.4**). If the sharp component of the distal unipolar EGM does not change after ablation, it suggests poor electrode-tissue contact or epicardial or intramural origin of the focal arrhythmia.

Depending on the nature of the focal arrhythmia, the distal unipolar EGM at the site of successful ablation may or may exhibit a qs/QS pattern. If the tip electrode is in contact with the source of an RVOT PVC, the distal unipolar EGM probably will begin with a negative component (qs/QS pattern) because the activation wave front begins beneath the tip electrode. If the tip electrode is in contact with a band of myocardium above the pulmonary valve that is the source of the PVC, it is unlikely that the distal unipolar EGM will begin with a negative component because the distal electrode simply records the activation wave front passing through the tissue beneath it. However, ablation there still can eliminate the PVC. This is similar

to recording an accessory pathway potential, the unipolar EGM of which rarely exhibits a qs/QS pattern. However, ablation anywhere recording an AP potential can eliminate the AP. Occasionally, the tip electrode is in contact with the beginning of the band of myocardium responsible for the PVC; the pre-potential recorded on the distal unipolar electrode exhibits a qs/QS pattern.

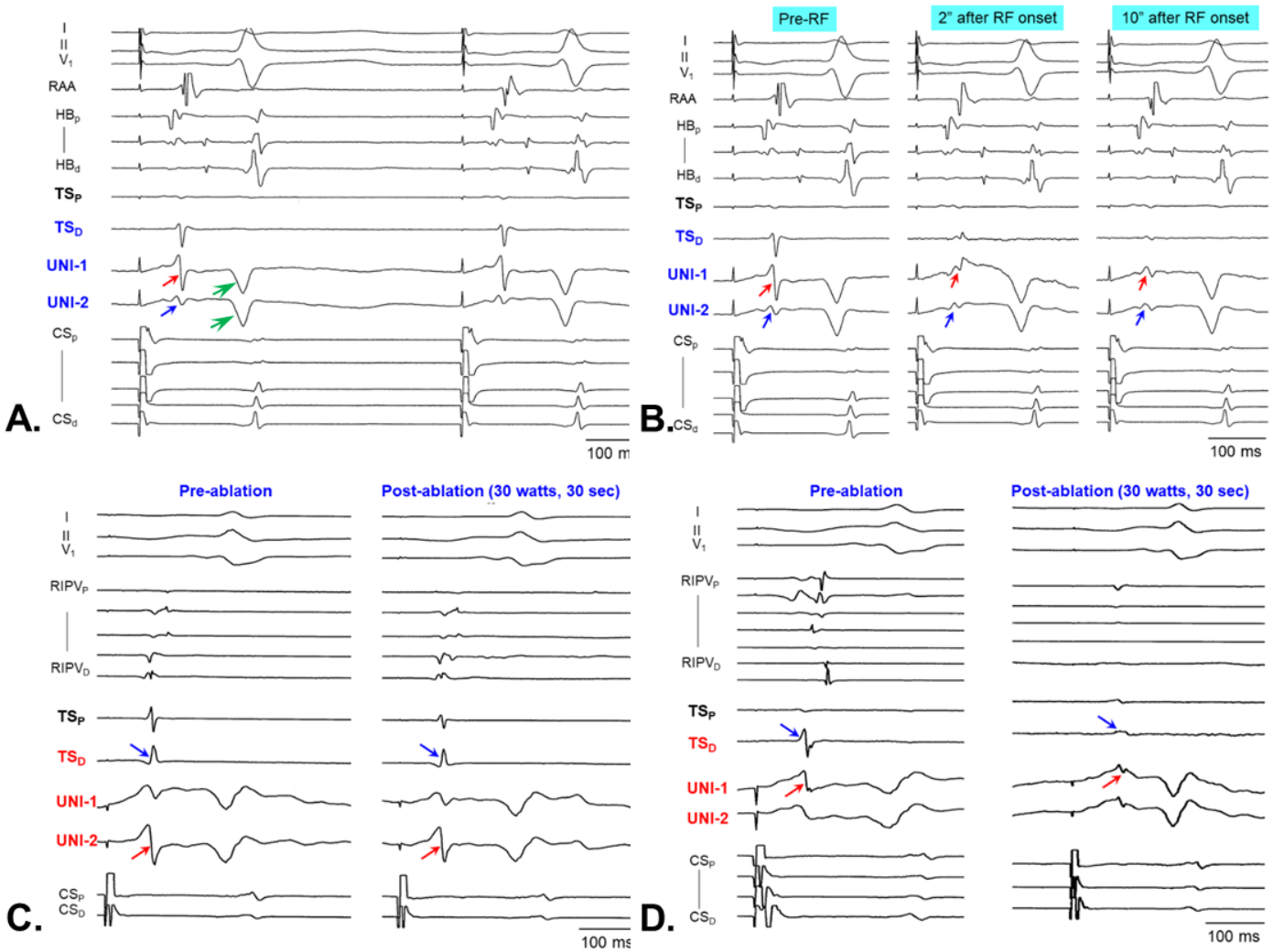


Figure 9.4. Using distal unipolar EGM to assess ablation effect. A. Before PV isolation, the sharp component of the bipolar EGM mainly came from the distal unipolar EGM (UNI-1; red arrow). Proximal unipolar EGM (UNI-2) was rounded and appeared to be far-field. Note that the ventricular potential (green arrows) of the distal and proximal unipolar EGM looked identical, indicating that it was a far-field potential. **B.** Ablation here (30 watts) quickly abolished the sharp, near-field EGM (red arrows) but the UNI-2 EGM remained unchanged (blue arrows). **C.** At a different site, the sharp component of the UNI-2 EGM (red arrow) constituted most of the bipolar EGM. In contrast, the UNI-1 EGM was rounded and appeared to be far field. After RF application, neither the unipolar EGMs nor bipolar EGM changed. **D.** After the ablation catheter was repositioned, the sharp component of the bipolar EGM now came from the UNI-1 EGM. Ablation there quickly abolished both the UNI-1 EGM and bipolar EGM. These examples demonstrate the importance of analyzing both the UNI-1 and UNI-2 EGM for mapping/ablation.

After exhaustive search, if the EGM at the site(s) of earliest endocardial activation is always preceded by a far-field potential, the origin of the PVC may be intramural, epicardial or in the neighboring chamber (**Figure 9.5**). To deliver higher power and/or longer RF applications will depend on the location of the site of "earliest' activation. For posterior RVOT locations, it is advisable to map the coronary cusps and LVOT first. For ventricular septum site, it may require ablation from both the RV and LV side. The author had heard plenty stories about catastrophic tamponade because operators did not use the unipolar EGM to localize the source of PVCs. Higher power of RF applications were delivered to the RVOT after ablation transiently suppressed the PVC. Most of such complications could have been prevented if the operator had analyzed the unipolar EGM carefully to realize that the target was not on the endocardial surface of the RVOT. If the ablation target

is in a scar, unipolar recordings may not be helpful in selecting ablation target. Local abnormal ventricular activities (LAVA) may be embedded deep in dense scars. The distal unipolar EGM may not show any sharp potential (local activation) at all. The bipolar EGM may better represent the local ventricular activation for selecting ablation targets and monitoring ablation efficacy (**Figure 9.6**).

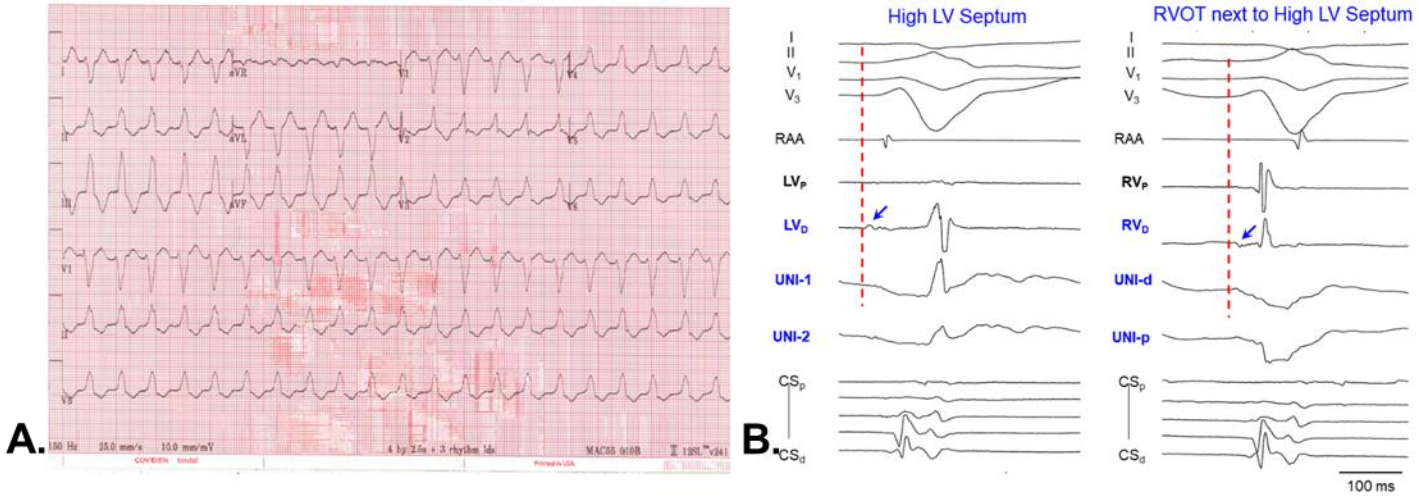


Figure 9.5. Intramural high septal VT. A. 12-lead ECG of VT. **B. Left panel:** EGM of the site of earliest activation (vertical red line) recorded from the high LV septum showed a small, rounded pre-potential, 30 ms before the onset of the PVC. Note that this pre-potential was barely visible on the distal unipolar EGM (UNI-1). This type of EGM strongly suggests that the distal ablation electrode is on top of an intramural focus. **Right panel:** EGM of the site of earliest activation recorded from the RVOT, directly opposite to the site recording the EGM shown in the left panel. The small, rounded pre-potential was 20 ms before the onset of the PVC. These findings indicate that the origin of the PVC/VT was intramural. When RF applications (40-45 watts) were delivered to the high LV septum, PVC/VT did not disappear until 2 minutes later. After several high power, long RF applications delivered to high LV septum, PVC/VT became scanty but required ablation from the RVOT (right panel) to completely eliminate the PVC/VT.

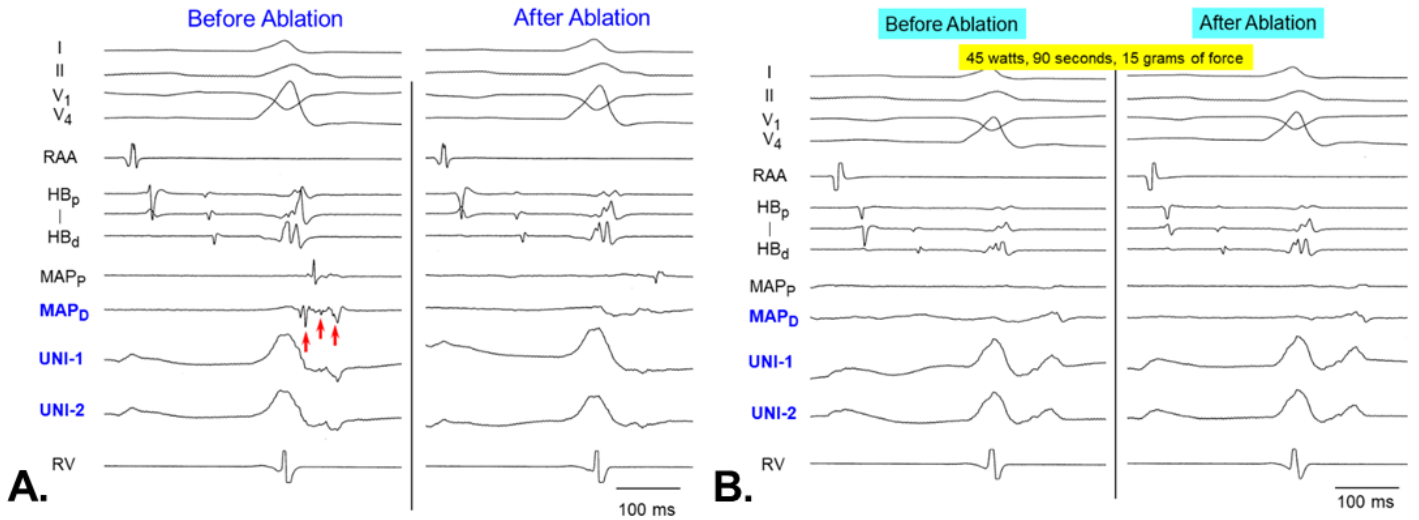


Figure 9.6. Ablation targeting local abnormal ventricular activity (LAVA) in a patient with ischemic cardiomyopathy. A. Distal bipolar EGM recorded 3 sharp components (red arrows). After ablation (40 watts, 60"), these 3 components disappeared. **B.** At another site, rounded, far-field EGMs were recorded on the bipolar and both unipolar electrodes. After ablation (40 watts, 90"), there was no change in any of the EGM. Note that both UNI-1 and UNI-2 showed exactly the same EGM, indicating that these EGMs were far-field.

In the OU-EP laboratory, focal AT or PVC ablation procedures are started without any sedation or anesthesia to maximize arrhythmia inducibility. Dr. Jackman always instructs patients to proactively report symptoms; he avoids repeatedly asking patients about symptoms when arrhythmia appears. This practice is to avoid priming the patient to report equivocal symptoms that can mislead the operator to chase an arrhythmia that is minimally symptomatic. For patients referred for focal AT or PVC ablation, the author prefers

to acquire a 2-minute 12-lead ECG in the preparation area. Correlation between symptoms and arrhythmia as well as prediction of the origin of arrhythmia can be assessed more accurately in this way because in the presence of patches for the defibrillator and electro-anatomical mapping system, the position of the ECG patches in the EP laboratory, particularly the precordial leads, is often different from that of a routine 12-lead ECG (**Figure 9.7**). Using the 12-lead ECG acquired in the EP laboratory for arrhythmia localization is prone to error.

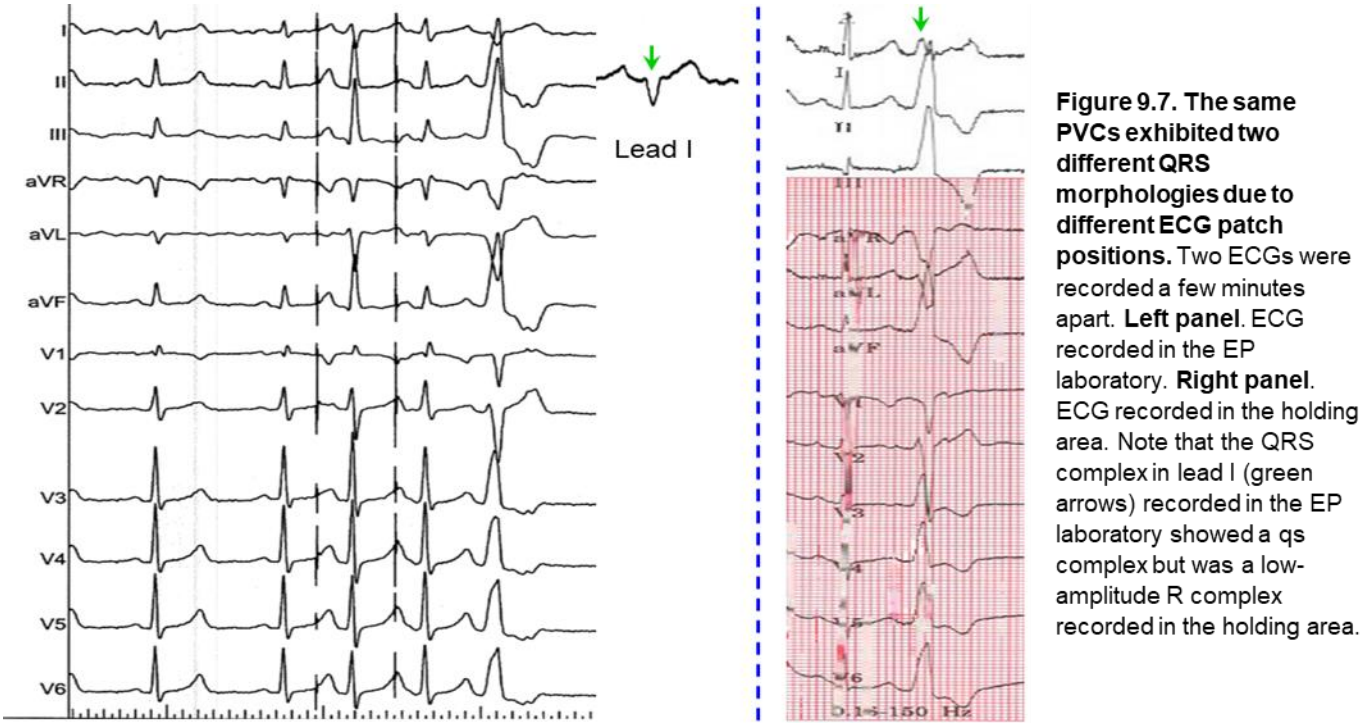


Figure 9.7. The same PVCs exhibited two different QRS morphologies due to different ECG patch positions. Two ECGs were recorded a few minutes apart. **Left panel**. ECG recorded in the EP laboratory. **Right panel**. ECG recorded in the holding area. Note that the QRS complex in lead I (green arrows) recorded in the EP laboratory showed a qs complex but was a low-amplitude R complex recorded in the holding area.

Which chamber to map first?

For a focal AT, the author uses the ECG criteria published by Drs. Peter Kistler and Jonathan Kalman to decide which atrium is more likely to be the origin of the focal AT (JACC 2006;48[5]:1010). Of note, these ECG criteria are less accurate in patients who had prior AF ablation because circumferential PV isolation or linear lesion sets create many zones of slow conduction or conduction block in the atrium, rendering the algorithm published by Drs. Kistler and Kalman less accurate. To map PVCs, the author uses the algorithms published in a series of papers by Dr. Frank Marchlinski's group.

Focal AT without prior AF ablation

ATs originating from the crista terminalis are the most common form of focal AT. The P wave morphology of a crista AT is similar to that of the sinus rhythm. Dr. Jackman prefers to position a multi-electrode catheter along the crista terminalis in preparation for mapping a focal AT. The tip of the catheter is positioned in the proximal SVC to ensure that this catheter covers the superior-posterior wall of the RA where cristal terminalis is located. Some of the electrodes on this crista catheter should record double potentials to ensure that the crista catheter is indeed positioned along the crista terminals.

Right superior pulmonary vein (RSPV) is situated behind the RA posterior wall. An RSPV tachycardia can masquerade as a crista AT. An important clue that Dr. Jackman is always looking for to differentiate a PV or LA tachycardia from a crista tachycardia is the timing and morphology of the distal unipolar EGM (UNI-1) when the mapping catheter is pointing at the septal-posterior wall of the RA. If the site of earliest activation along the crista terminalis is preceded by a significant far-field potential on the unipolar EGM, Dr. Jackman has a low threshold for mapping the LA and right-sided PVs before ablating the earliest activation site in the RA (**Figure 9.8; Figure 9.9**). Another important clue is the relative timing of the septal vs. the lateral aspect

of the crista terminalis. If the activation timing of the lateral aspect is earlier, it is very unlikely that this AT is an LA or RSPV tachycardia. On the contrary, if the activation timing of the septal aspect of the crista terminalis is earlier, Dr. Jackman has a low threshold of mapping the LA and right-sided PVs.

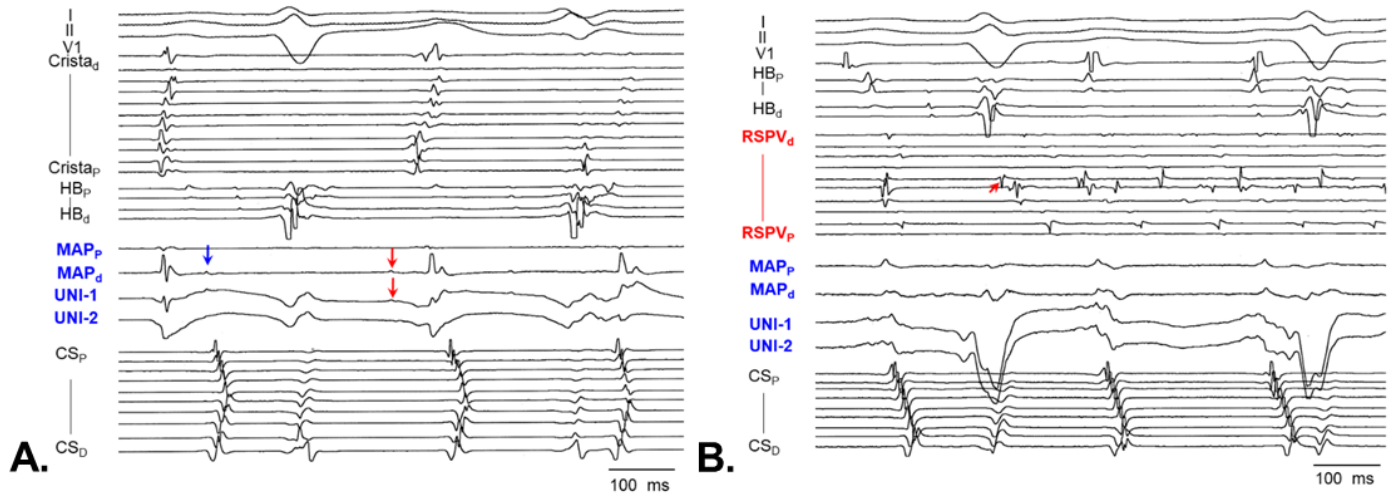


Figure 9.8. Far-field potential recorded at the site of earliest activation in the RA. **A.** Mapping catheter was positioned at the septal aspect of high RA posterior wall. A small, far-field potential was visible (red arrow). RF current was not delivered to this site due to its far-field characteristics. Note that in sinus rhythm, this far-field potential was late (blue arrow). **B.** A LASSO catheter was positioned inside the RSPV and showed RSPV firing (red arrow).

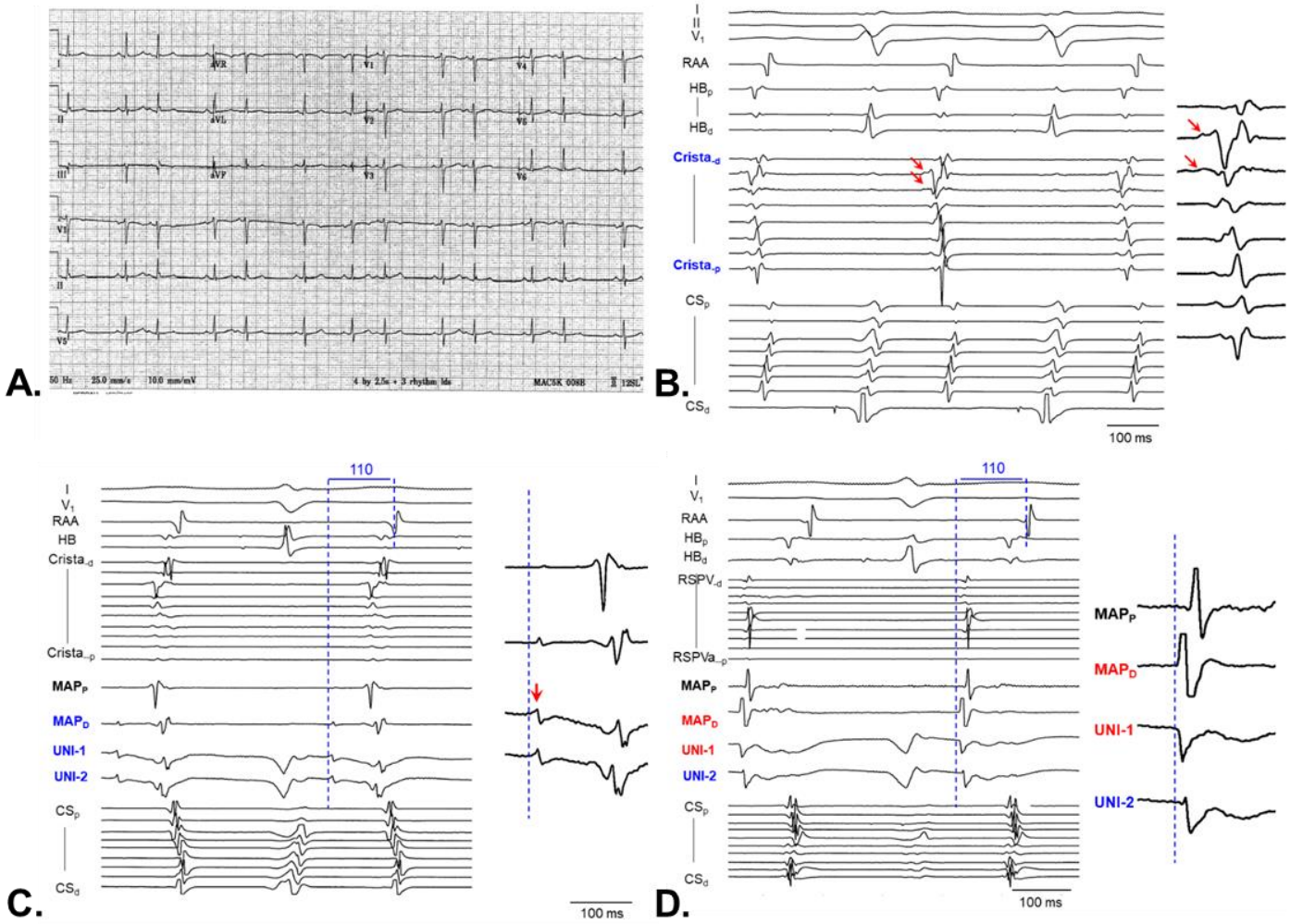


Figure 9.9. RSPV tachycardia. A. 12-lead ECG. **B.** The site of earliest activation in the RA was recorded along the septal aspect of the crista terminalis but the earliest potential appeared to be far-field (red arrows). Tip of the CS catheter was at the distal CS-AIV

junction. **C.** Mapping catheter in the RSPV recorded an even earlier potential (110 ms before RAA) but the UNI-1 EGM began with an r wave (red arrow). **D.** At the site of earliest activation inside the RSPV, the UNI-1 EGM began with a q wave. To avoid PV stenosis by targeting the site of earliest activation, RSPV antrum was isolated to treat this RSPV tachycardia.

LA tachycardia can conduct to the RA through three major routes: Bachmann's bundle (**Figure 9.10**), inter-atrial septum and CS in decreasing order of prevalence. LA tachycardias preferentially conducting to the RA through the CS often originate in the septal aspect of the LA. Dr. Jackman maps the 3 areas to determine if activation timing is earlier there. If it is, Dr. Jackman will proceed with trans-septal puncture to map the LA and PVs.

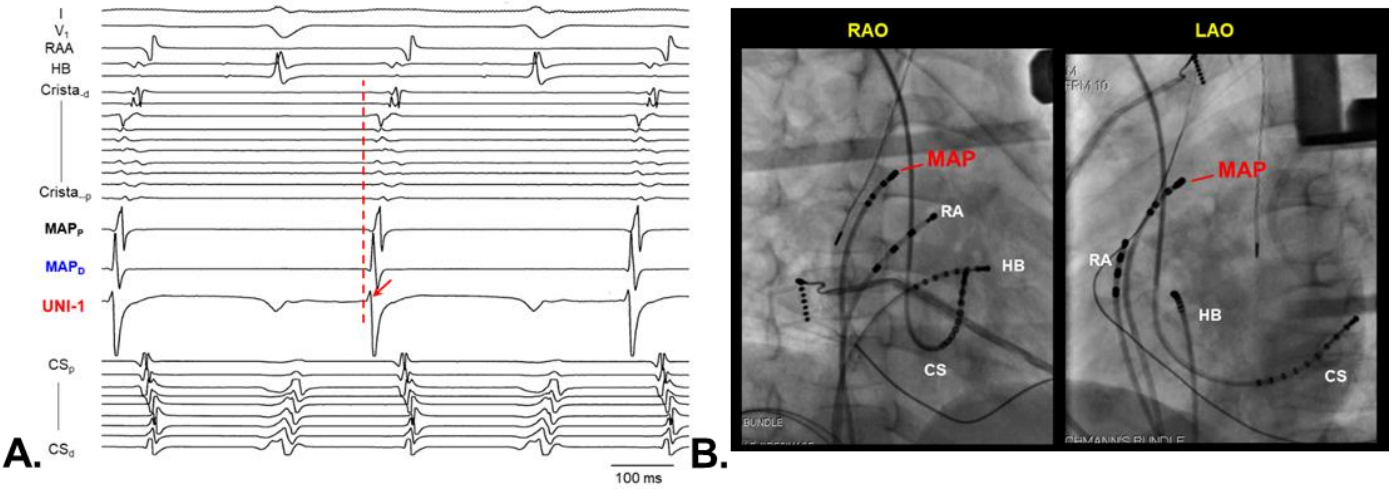


Figure 9.10. RSPV tachycardia conducts to RA through Bachmann's bundle. A. Mapping catheter was positioned at the Bachmann's bundle. The far-field activation timing (red arrow) was as early as the far-field potential on the Crista catheter (vertical dotted line). These findings indicate that it was a left atrial tachycardia. **B.** Location of Bachmann's bundle. The mapping catheter was positioned just below the RA-SVC junction, pointing toward the tricuspid annulus on the RAO view and pointing leftward on the LAO view.

Septal AT

The anecdotal experience of the OU-EP group indicates that many of the septal right atrial tachycardias originate in the area slightly anterior and superior to the fossa ovalis. They are usually located superior to the HB; ablation here does not carry a high risk of AVN injury because AVN is situated inferior to the HB. If the timing of the RA septum appears very early but the distal unipolar EGMs does not exhibit a qs/QS pattern, Dr. Jackman would map the LA septum before attempting to ablate the "earliest" site along the RA septum to avoid the risk of AV block (**Figure 9.11**). It is noteworthy that the CS catheter typically only covers the inferior and inferolateral aspect of the LA adjacent to the mitral annulus. The anterior wall of the LA is not covered by a catheter in a routine EP study. A focal AT originating in the anterior wall of the LA can easily conduct to the interatrial septum much earlier than to the CS and create a false impression that the atrial timing of the HB region is earliest and CS activation shows a proximal- to-distal pattern. For this reason, the practice of the OU-EP group to treat an "apparent" RA septal tachycardia is to map the LA before attempting to ablate the site of earliest activation on the RA septum to minimize the risk of AVN injury. It is not uncommon to find out that the site of earliest atrial activation was on the anterior wall of the LA, far away from the septum; ablation there does not carry a risk of AV block. In a true septal AT, if the timing of the bipolar EGM is equally early between the RA and LA septum, RF application should be delivered to the site where the distal unipolar EGM begins with a qs/QS pattern (**Figure 9.11**). If the distal unipolar EGM at the site of earliest activation begins with a far-field potential on both the left and right inter-atrial septum, the next step is to map the coronary cusp.

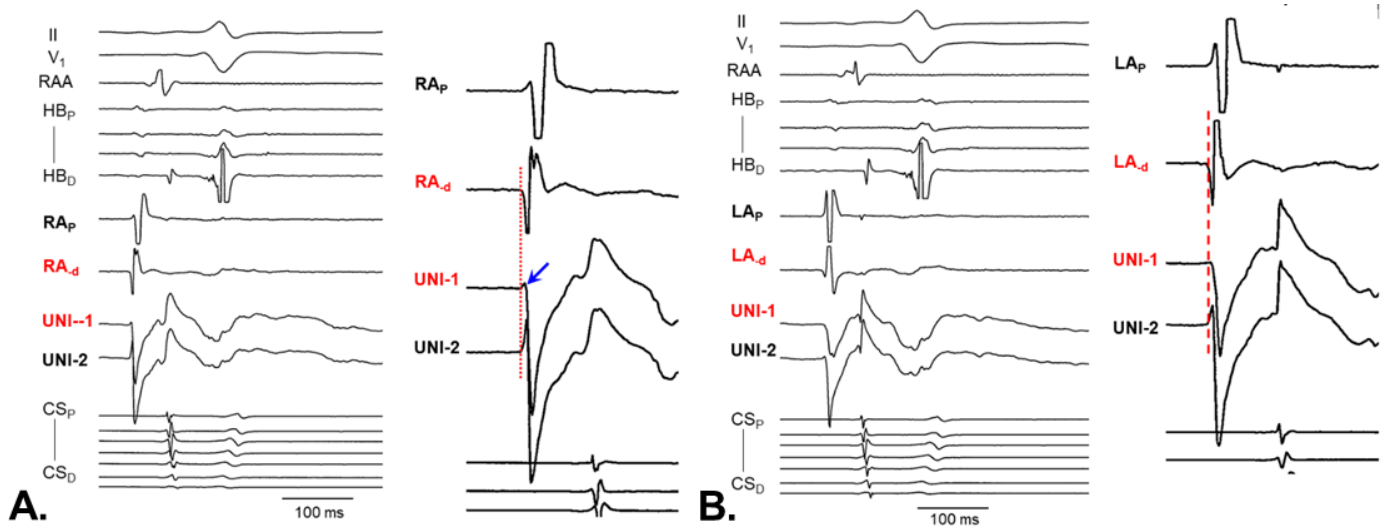
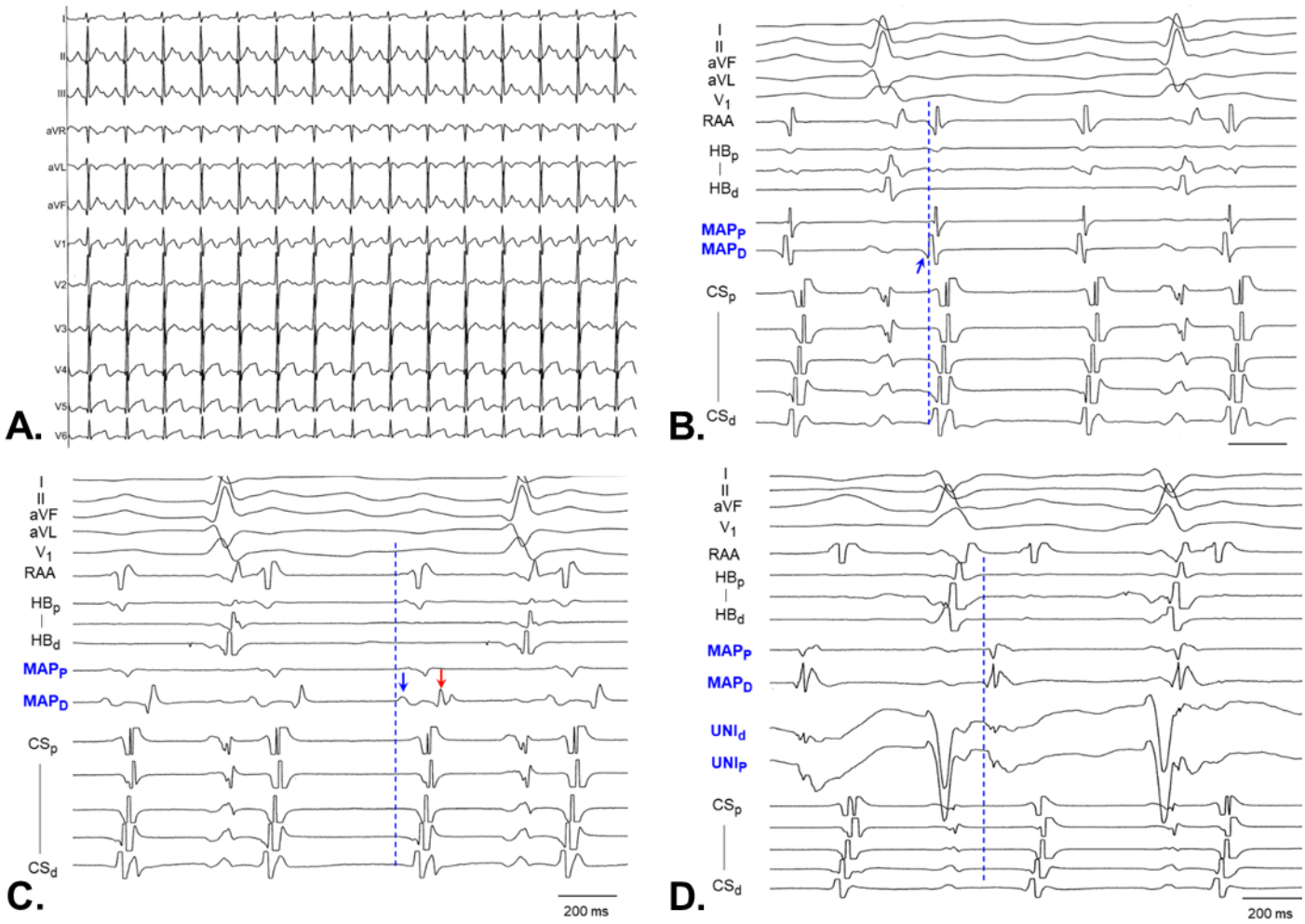


Figure 9.11. Using unipolar EGM to select ablation target of a septal AT. A. EGM at the site of earliest activation in the RA was recorded on the RA septum. Not that the distal unipolar EGM began with a small r wave (blue arrow), indicating that the origin of the tachycardia was elsewhere. **B.** EGM of the site of earliest activation recorded from the LA septum. Note that the distal unipolar EGM began with a QS morphology. Ablation there quickly eliminated the tachycardia.

If the atrial timing at the His region and distal CS are very similar, Dr. Jackman will map the left fibrous trigone. ATs in this region often exits to the RA through the Bachmann's bundle. Atrial timing recorded from the HB area appears to be early but the unipolar EGM shows a long far-field potential (**Figure 9.12**).



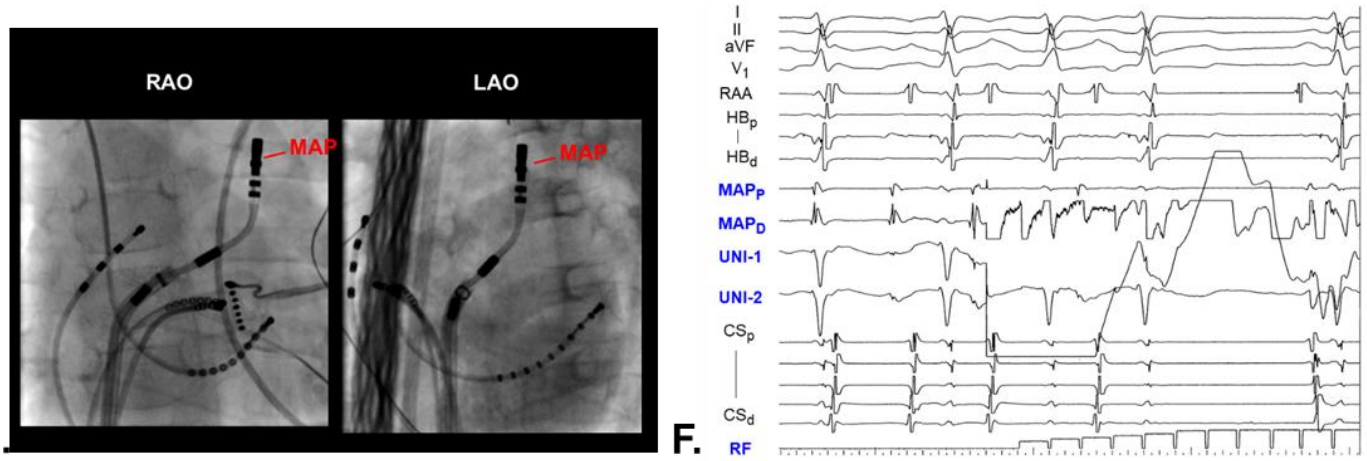


Figure 9.12. Atrial tachycardia originating from the left fibrous trigone. A. 12-lead ECG of tachycardia. **B.** Atrial activation timing of the anteroseptal area and anterolateral CS were similar. The blue vertical line indicates similar earlier activation recorded on the mapping catheter positioned at the fossa ovalis, indicating that none of these sites (HB, CSd and fossa) was near the origin of the tachycardia. **C.** Mapping catheter was positioned at the Bachmann's bundle where a double potential was recorded. The first component (blue arrow) was rounded and appeared to be a far-field potential from the LA. This potential was simultaneous with the far-field potential recorded on the HB catheter. The second component (red arrow) was sharp and was the local RA activation timing. **D-E.** Mapping catheter was positioned under the left fibrous trigone where earliest atrial activation was recorded; the distal unipolar EGM (UNId) began with a qS pattern. **F.** Ablation there eliminated AT.

Pulmonary Vein Tachycardia

As discussed earlier, a right PV tachycardias can be difficult to differentiate from a high crista AT. A left PV tachycardia can be difficult to differentiate from a LAA tachycardia. An important maneuver to map a PV tachycardia is to know the tachycardia originates from the PV (inside-out) or from the PV antrum (outside-in). The former can be cured by PV isolation (either segmental ostial isolation of the culprit PV or circumferential isolation of both PVs on the same side). The latter will require detailed mapping to identify it origin; empirical PV isolation may or may not eliminate this type of tachycardia. The author prefers to put a LASSO catheter deep into the culprit PV and slowly pulls it back. If the timing of distal PV is earlier than that of the antrum, PV isolation is then performed. On the other hand, if the timing of distal PV is late, more mapping around the PV-LA junction and LA will be performed to identify the true origin of the "PV" tachycardia.

Young females with multiple focal ATs

Patients with this type of arrhythmia often have other co-existing arrhythmias such as inappropriate sinus tachycardia, vasovagal syncope or postural orthostatic tachycardia syndrome, suggesting that dysfunction of the autonomic nervous system may underlie these arrhythmias. The standard practice of the OU-EP group to treat this type of patients is to start the procedure without any sedation or anesthesia to maximize the chance of inducing focal ATs. Risks of SA nodal or AV nodal injury and phrenic nerve injury are explained to patients in great detail before ablation. Before administering isoproterenol, the author prefers to quickly map the RA to identify the location of the sinus node and phrenic nerve to minimize the risk of injuring these structures. Extensive RA and CS burst pacing as well as multiple does of isoproterenol are used to induce focal ATs. If the procedure is acutely successful, patients tend to have symptom relief for only a few months; recurrent AT(s) usually originates in a different focus. After multiple ablation procedures, many patients remain symptomatic; some patient continue to have debilitating symptoms despite SA nodal or AV nodal injury. The author often recommends yoga, Tai-Chi or meditation to the patients to help reduce sympathetic outflow as adjuvant therapies to ablation.

Mapping PVCs or Focal VT

Using the triggered sweep function provided by the Bard recording system to map RVOT/LVOT PVCs, Dr. Jackman sets the trigger at the tall R wave in lead II, III or aVF. With this approach, the only QRS complex showing up on the live and review screens are the targeted PVC beats (**Figure 9.13**). Of note, the slope of the R wave of PVCs may show subtle beat-to-beat variations; timing reference set on the R wave may not be accurate. Dr. Jackman typically selects a stable intraventricular EGM (e.g. AIV or ventricular branch of the CS) as the intraventricular timing reference **(Figure 9.13**). When mapping catheter is positioned in the zone of early activation, this intraventricular reference is used for accurate timing comparison. This intraventricular reference can be anywhere in the RV or LV as long as the catheter does not move and EGM is stable regardless of the cardiac cycle or respiration.

The frequency of PVCs often needs to be maintained by adrenergic stimulants such as isoproterenol. Because some PVCs only occurs when the sinus rate is relatively slow, sinus tachycardia induced by isoproterenol would suppress PVCs. Despite discontinuation of isoproterenol, the sinus rate remains too fast for PVCs to recur. To avoid this problem, the author's preference is to start with a moderate dose of phenylephrine to induce hypertension, which should trigger the baro-reflex to lower the sinus rate. If the frequency of PVC is not affected by phenylephrine, isoproterenol is washed in after blood pressure returns to the normal range.

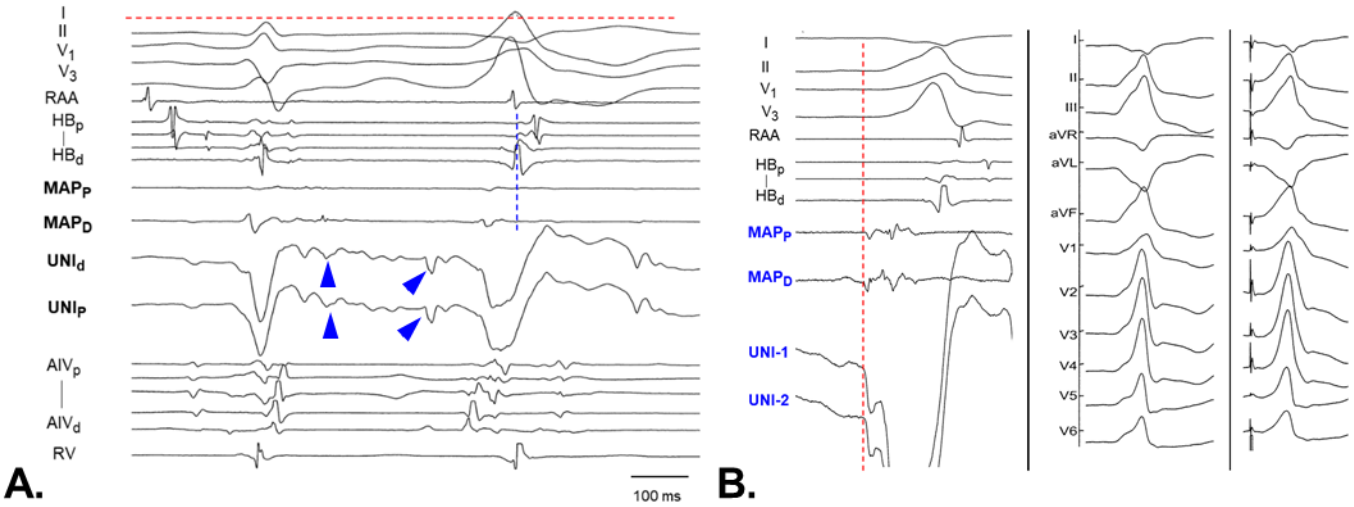


Figure 9.13. Mapping PVCs. A. The Bard triggered sweeps are set to sense the potential when it crosses the horizontal red line in ECG lead II. In this way, the review screen only recalls the beats that have a tall R complex (PVCs). Because QRS complexes may vary slightly beat by beat, a stable ventricular EGM is used for accurate intracardiac timing reference (vertical blue line). Blue arrowheads denote artifacts. Note that both the distal and proximal unipolar electrodes recorded the same potential, indicating that they were far-field potentials. **B**. **Left panel.** Site of successful ablation at the junction of the great cardiac vein and anterior inter-ventricular vein. The bipolar and unipolar EGM began simultaneously; the distal unipolar EGM (UNI-1) began with a qs wave. **Right panel.** Pace mapping showed 97% match. Ablation here eliminated PVCs.

Often, the same site tagged by electro-anatomical mapping during PVC and during sinus rhythm is > 1cm apart. After PVCs are eliminated by ablation, reinforcement ablation during sinus rhythm around the successful ablation site may appear to be at a distance from the successful site tagged during PVCs. To avoid this problem, the author prefers to take a location-only point on the CARTO during sinus rhythm immediately before ablation to serve as a reference point in sinus rhythm to deliver effective reinforcement ablation lesions.

If one cannot find any early activation site where the distal unipolar EGM begins with a qs/QS pattern, it suggests intramural or epicardial origin. If no qs/QS EGM can be found after mapping the neighboring chamber or epicardium, Dr. Jackman ablates the sites showing the earliest unipolar EGM. It may require higher power and longer RF applications to eliminate this intramural focus. If a pre-potential is the ablation

target (e.g. in the coronary cusp), the distal unipolar EGM may not begin with a qs/QS pattern because the tip electrode may be in contact with the body, not the origin of the muscle bundle that is the source of the PVC. As long as the distal unipolar electrode records the pre-potential, it is a good ablation target. This is similar to an AP ablation. As long as the tip electrode records the AP potential, ablation there should work.

The OU-EP group has not adopted the practice of using cryoablation to treat PVCs that originate from locations difficult to stabilize electrode-tissue contact. The idea behind using cryoablation is that it can stick to the target and appears to provide better electrode-tissue contact. However, for cryoablation to work, it requires good electrode-tissue contact to allow the tissue temperature to drop to -40 to -50^{o}C. If contact is suboptimal, the blood between the electrode and tissue forms an ice ball. Similar to an igloo, ice ball serves as an insulator that only allows the tissue temperature to reach 0^{o}C. Therefore, for cryoablation to work in PVC ablation, the electrode-tissue contact must be very good in the first 30-40 seconds to allow the tip electrode to be stuck to the targeted tissue without leaving a space between the targeted tissue and electrode to form an ice ball.

"Whack-a-mole" ablation

It is not uncommon to encounter a situation in which PVCs of different origins appear randomly like the "whack-a-mole" game. Operators end up not being able to finish mapping any of the PVCs. In this difficult situation, the author carefully selects some ECG leads that can discern different forms of PVCs. These ECG leads are displayed in both the Bard recording system and CARTO mapping system. While mapping PVC1, if the local timing is early for PVC2, the author instructs an assistant to write down "CARTO point 43, early for PVC2" in the Bard recording system. This approach allows the operator to focus on mapping one form of PVC but gain insight into the site of early activation of other forms of PVCs. On a luck day, after PVC1 was eliminated, the author has a rough idea about the site of early activation of other forms of PVCs as well. Detailed mapping can then be proceeded. Manufactures of all the mapping systems are developing new algorithms to allow the operator to map multiple PVCs at the same time. In the near future, we may not need to play the "whack-a-mole" game anymore.

Myocardial origin vs. Purkinje origin?

The conduction velocity of the Purkinje fibers is 2-3 M/sec, approximately 10 times faster than that of myocardium. Unless the peripheral Purkinje system is significantly diseased, arrhythmias originating in the Purkinje system usually conducts quickly back to the His bundle. The VH interval of Purkinje-related arrhythmias is usually shorter than 40 ms. For example, the VH interval is typically 20-30 ms in atrio-fascicular AVRT (Mahaim) and in VT originating in the left posterior fascicle. Because of fast conduction in the Purkinje system, the wave front of arrhythmias originating in the Purkinje system propagates quickly to both ventricles through the peripheral Purkinje system. For arrhythmias originating from the LBB or its fascicle, the initial vector of septal activation is usually preserved, retaining the small, sharp r wave (<40 ms) in V1 and/or V2 lead. Therefore, the presence of a typical RBBB pattern (V1 and/or V2 begins with a small, sharp r wave) should alert the operator that this arrhythmia may originate from the left-sided Purkinje system, not myocardium. For arrhythmias originating from the RBB system, the V1 and/or V2 lead can begin with a small, sharp r wave or exhibit a QS pattern, depending on how the proximal ventricular septum is activated. If it is activated by the RBB, it most likely will exhibit a QS pattern because the septal activation vector is pointing away from the V1 and/or V2 lead. If the proximal septum is activated by the septal branches of the LBB, it most likely will begin with a small, sharp r wave in V1 and/or V2 because the septal vector is pointing toward the V1 and/or V2 lead. As discussed in **Chapter 4** and **Chapter 11**, the author views the small, sharp r wave in lead V1 and/or V2 as a finding with a high positive predictive value of ventricular activation through the Purkinje system; however, the absence of it does not exclude arrhythmias of His-Purkinje origin because the refractory period of the septal branches of the LBB may be longer than the coupling interval of the arrhythmia.

The author uses a two-step process to make an educated guess based on the electrophysiological properties of the His-Purkinje system.

Step-1: examine lead V1 and V2 to determine if it exhibits typical bundle branch block or not.

Step-2: measure the VH interval of the PVC or VT.

a. If it exhibits *typical* LBBB or RBBB pattern and the VH interval is fixed and <40 ms, the arrhythmia most likely originates from the His-Purkinje system. The author will target the Purkinje potential.

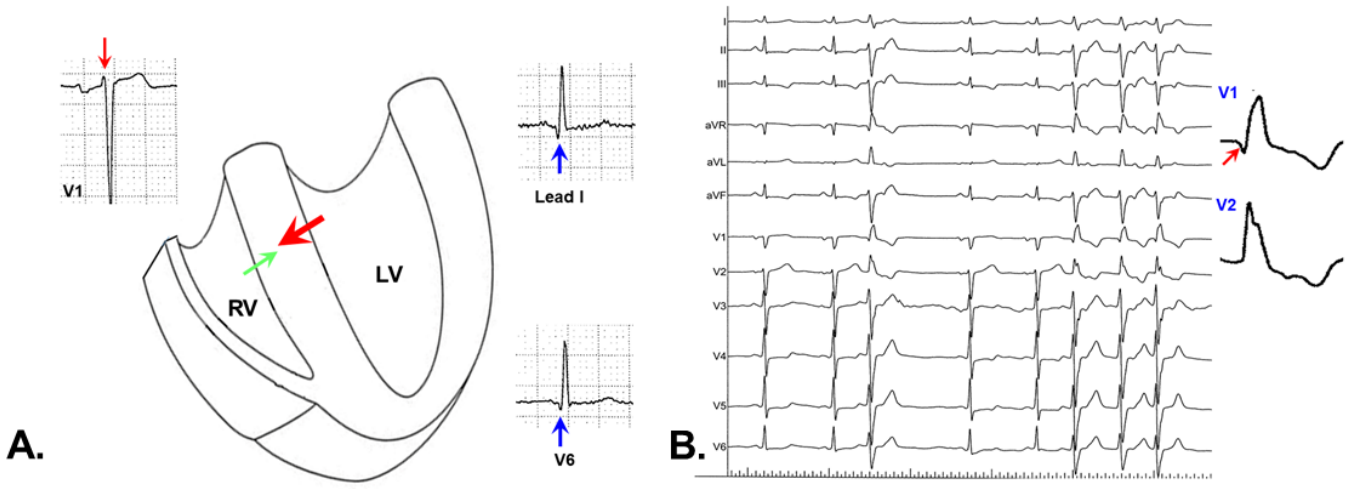
b. If it exhibits *atypical* LBBB or RBBB pattern and the HB potential is dissociated from the ventricular potential (the VH interval is not fixed) or the VH interval is very long (e.g. >60 ms), this arrhythmia is unlikely to originate from the Purkinje system. The author will search for the earliest myocardial potential.

c. For PVCs exhibiting *atypical* BBB but with a long, fixed VH (e.g. 60 ms), it suggests that the arrhythmia does not originate from the Purkinje system but quickly engages the Purkinje system. For example, PVCs originating in the papillary muscle with a stable VH interval of 70 ms may suggest that the origin of the PVC might not be deep in the papillary muscle.

d. If clinical or induced arrhythmia showed multiple morphologies consistent with LBBB or RBBB with hemi-block, it strongly suggests that this arrhythmia originates in the His-Purkinje system and has multiple exits.

d. These educated guesses are based on the assumption that the patient does not have diffusely diseased His-Purkinje system. However, if a patient has a very long HV interval (e.g. >80 ms), the presence of diffusely diseased His-Purkinje system makes the odds of Purkinje-related arrhythmia even higher.

Figure 9.14 illustrates some of the examples correctly predicted by the authors educated guesses. Readers may dig out some past papillary muscle PVC or fascicular VT cases to see if this two-step process correctly predict the origin of the arrhythmia.



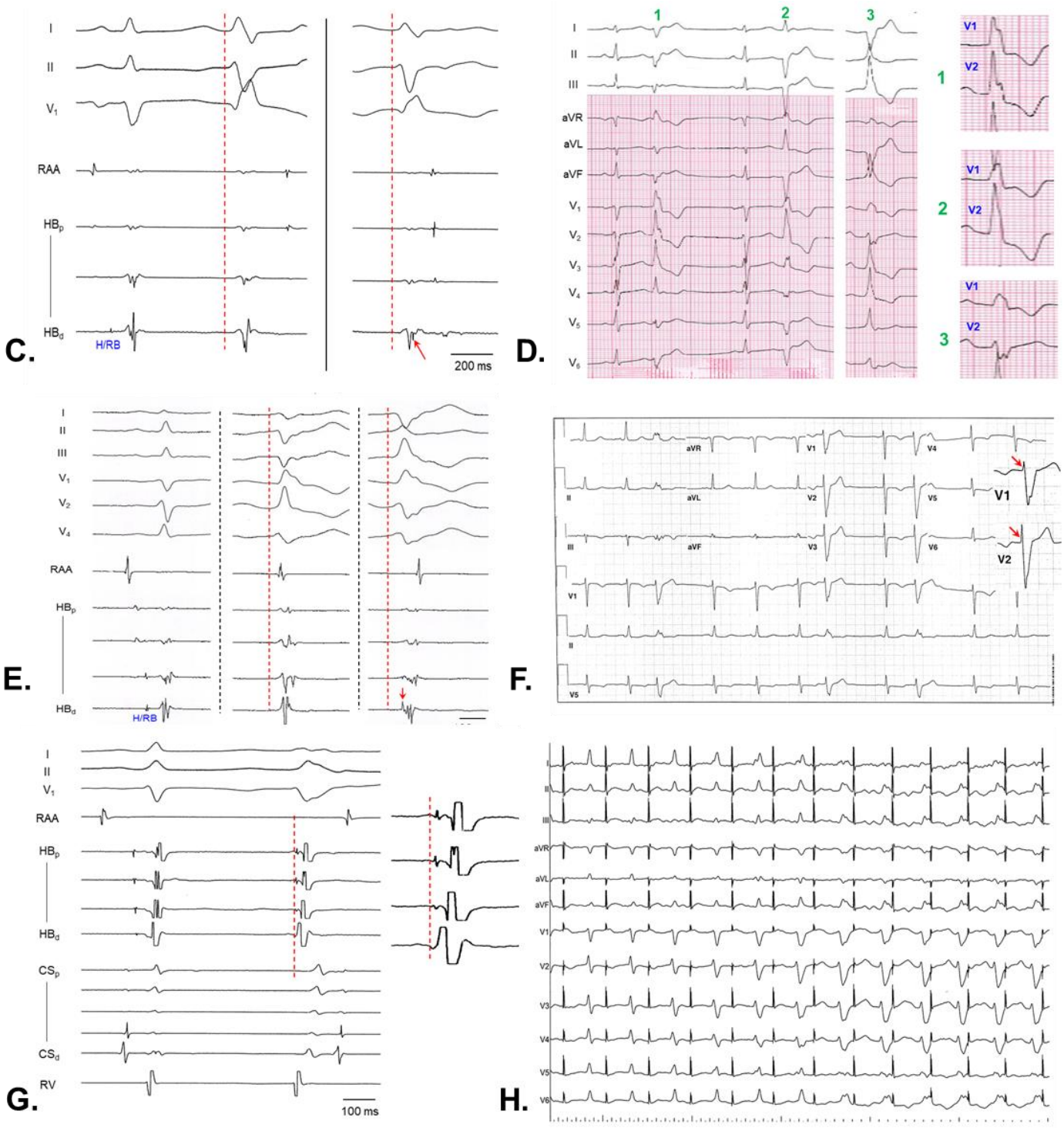


Figure 9.14. PVCs originated from papillary muscle vs. Purkinje system. A. Ventricular septum is the first part of the ventricle that is activated by the Purkinje network of the left ventricle (red arrow). Approximately 5 ms later, the septal branch of the RBB activates the septum (green arrow). Ventricular septum is activated within 20-30 ms. The net vector is therefore left to right, posterior to anterior, creating a small, sharp r wave in V1 and/or V2 (thin red arrow) as well as a q wave (blue arrows) in lateral leads such as lead I and V6**. B.** PVCs originated from the posterior-medial papillary muscle. Note that the QRS complex in lead V1 began with a q wave, consistent with an atypical RBBB morphology. **C.** There was no visible HB/RBB potential in the PVC shown in the left panel. In another PVC complex, a late HB/RBB potential (red arrow) was visible. These findings strongly suggest that PVCs did not originate from the Purkinje system. It required multiple, high power, long RF applications to eliminate this PVC originated deep in the posterior-medial papillary muscle. Vertical red line: beginning of the QRS complex. **D.** In another patient, three different PVCs with RBBB morphology were identified. Note atypical RBBB of all the 3 different PVCs. **E. Left penal:** sinus rhythm; **middle panel**: PVC1. There was no visible HB/RBB potential. This PVC

originating deep in the posterior-medial papillary muscle was eliminated by multiple high power, long RF applications. **Right panel:** PVC3. A VH interval of 63 ms was noted in most PVC3 complexes, suggesting that PVC3 may not be deep in the anterior-lateral papillary muscle. PVC3 was easily eliminated. Red arrow: HB potential. **F.** In another patient referred for PVC ablation, ECG showed typical LBBB, suggesting that this arrhythmia originated from the RBB system. **G.** A fixed VH interval of 20 ms was visible. Note retrograde RBB to HB conduction. **G.** During decremental atrial pacing, the stimulus-QRS interval increased progressively and typical LBBB became more prominent. These responses are typical for an atrio-fascicular accessory pathway (Mahaim). This “PVC” was automaticity originating from the accessory AVN. Ablation targeting the accessory pathway potential on the tricuspid annulus eliminated this “PVC”.

Chapter 10:

Mapping and Ablation of Reentrant Atrial Tachycardia

Atrial tachycardias (AT) occurring after surgical correction of congenital heart diseases or AF ablation are probably the most difficult arrhythmia to map and ablate. In both situations, focal or reentrant ATs hide within extensive atrial scars. For ATs occurring after AF ablation, the most challenging cases are the ones with prior extensive CFAE ablation which produces numerous non-transmural lesions, creating perfect substrates for macro-reentrant, micro-reentrant or focal ATs. Diffuse low-voltage areas, caused by atrial fibrosis in advanced stage of AF or by scars created by prior ablation, pose great challenges to operators. Making things worse, local electrograms (EGMs) in these low voltage areas tend to have multiple components; annotating the timing of these low voltage areas can be very challenging.

In general, AT can be classified into three major forms: macro-reentrant AT, micro-reentrant AT (generally, reentrant circuit<2 cm in diameter) and focal AT (**Figure 10.1**). The latter two forms of AT may show a centrifugal pattern of activation on the electro-anatomical mapping system; to differentiate between the two forms of ATs can be very difficult, depending on the density of mapping and responses to entrainment. Occasionally, a focal AT can be differentiated from a micro-reentrant AT if a multi-electrode mapping catheter happens to be positioned in the center of the apparent centrifugal activation but this is a rarity. With closely-spaced 64 electrodes, the Rhythmia mapping system is probably the best tool to differentiate a focal AT from a micro-reentrant AT but it may still require re-annotation and entrainment to differentiate between the two forms of AT.

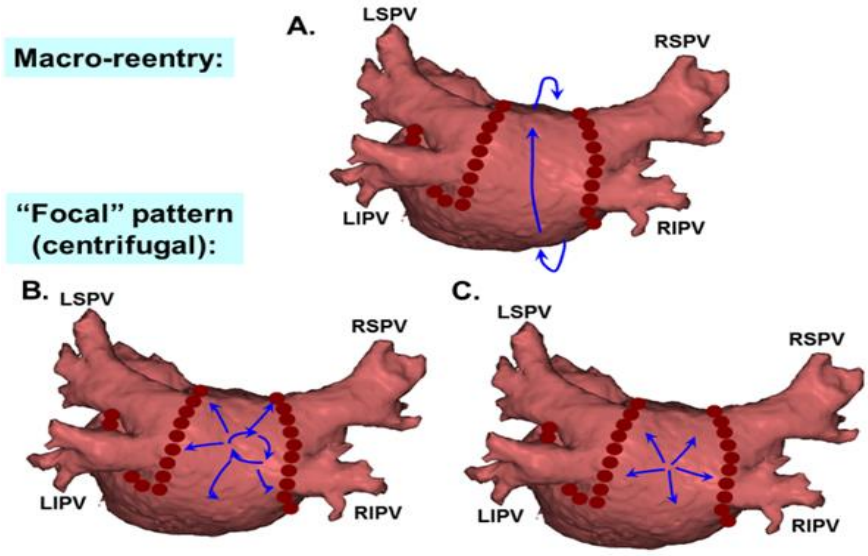


Figure 10.1 Three common types of atrial tachycardia (AT). A. macro-reentrant AT. **B.** micro-reentrant (small-circuit) AT. **C.** focal AT. On a low density electro-anatomical map, **B and C** may look identical. Macro-reentrant ATs, micro-reentrant ATs and focal ATs account for approximately 50%, 30% and 20% of the ATs after AF ablation, respectively.

Ablation of cavo-tricuspid isthmus (CTI)

Ablation of the CTI is considered a "bread and butter" type of ablation. However, difficult CTI ablation is not uncommon, usually because of thick pectinate muscle, a prominent Eustachian ridge or a pouch in the CTI. In the late 90's when the CARTO mapping system was still in its infancy, Dr. Hiroshi Nakagawa came up with an idea of using voltage amplitude to correlate with the thickness of the pectinate muscle in the CTI. The study was designed to create a voltage map of the CTI during atrial flutter (AFL) or during CS pacing after the diagnosis of CTI-dependent AFL had been verified by entrainment. Based on the voltage in CTI, the ablation line was customized to avoid high voltage areas (>3.5 mV) which Drs. Nakagawa and Jackman hypothesized to represent thick pectinate muscle (**Figure 10.2)**. Over the past 20 years, the OU-EP group continued to use this voltage mapping strategy to guide ablation. The recurrence rate was very low. The few failed cases all had multiple prior ablations, creating a thick layer of endocardial scars in the CTI. Ablation lesions simply could not penetrate deep enough to eliminate the surviving epicardial myocardium in the CTI.

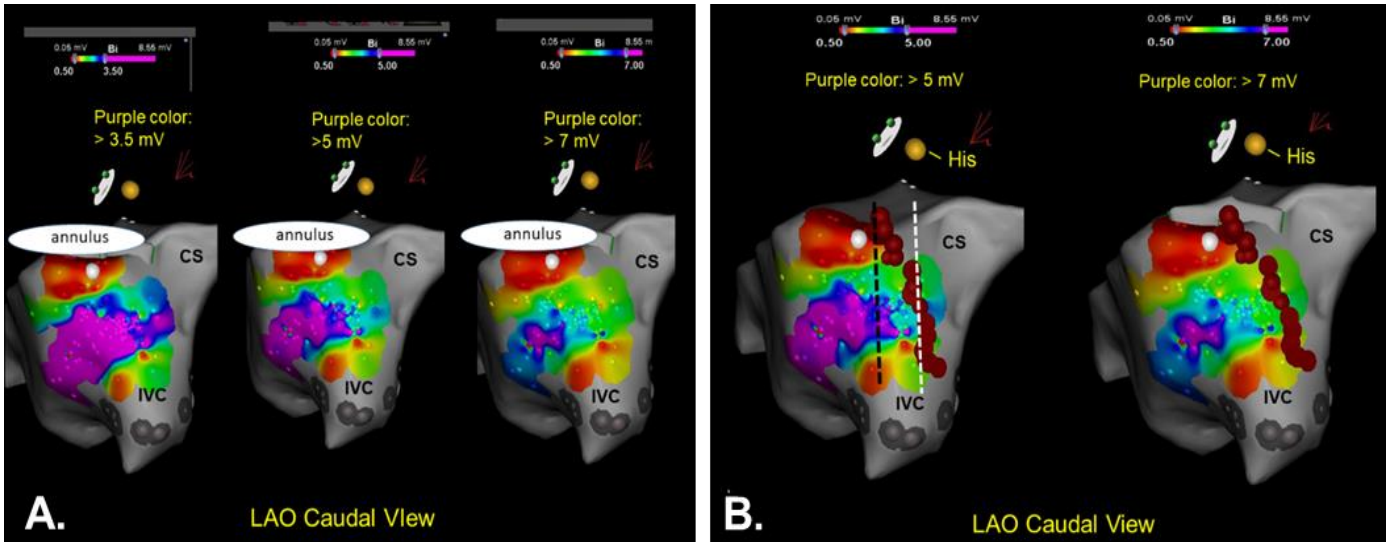


Figure 10.2 Isthmus-dependent right atrial flutter. A. Mapping of the cavo-tricuspid isthmus (CTI) revealed an area in the middle of the CTI that had very high voltage. The CARTO voltage map was set to display purple color if the voltage is larger than 3.5, 5 and 7 mV for the left, middle and right panel, respectively. **B.** The ablation line was customized to be more septal to circumvent the high voltage area. Higher power/time/force was applied to sites with higher voltage to achieve transmural necrosis. One linear ablation lesion made conduction block across the CTI. White dotted line indicates septum. Black dotted line indicates the location of a typical CTI ablation line without the knowledge of the voltage map. This line would have to transect a high voltage area with a significant risk of ablation failure.

The importance of CTI voltage mapping prior to ablation is to identify sites with high voltage, a pouch or tall Eustachian ridge as well as to ensure that the ablation catheter can touch all the points along the designed ablation line. Certainly, intracardiac echo can visualize a tall Eustachian ridge or a pouch but is not sensitive enough to estimate the thickness of pectinate muscle. There have been many patients referred to the OU-EP group after multiple failed AFL ablations. It is not uncommon to identify an area along the ablation line that had never been touched in prior ablations (**Figure 10.3)**. Typically, they are located on the tricuspid annulus, in a CTI pouch or in front of a prominent Eustachian ridge. The latter requires a very tight curve of the ablation catheter to reach the myocardium there. Non-transmural lesions are often located in the area with high voltage. Knowing the site of higher voltage also helps adjust ablation power/force/time to achieve transmural necrosis.

In the OU-EP laboratory, the conduction time across the CTI line was never used as a criterion to determine if conduction block has been achieved. First, the length of CTI is usually >3 cm. The recording range of a HALO catheter only covers a small portion of the CTI. If the HALO catheter is positioned adjacent to the tricuspid valve, it can easily miss a long gap in front of the Eustachian ridge. Second, the conduction time through a non-transmural ablation site can be as long as 150-200 ms, leading to a long conduction time across the CTI line and giving a false impression of conduction block. This type of gap typically supports an AFL with a longer CL or a clockwise AFL. In the OU-EP laboratory, the standard practice after AFL ablation is to remap the CTI during proximal CS pacing; the pacing site is as close to the CTI line as possible. The most typical activation pattern is that the paced wave front propagates around the IVC and then toward the annulus. The timing along the lateral side of the CTI line should be progressively later toward the annulus. Any point that breaks this pattern is potentially a gap. The author prefers not to take any point ipsilateral to the pacing site so that on the other side of the line, the color spectrum (from red to purple) on CARTO represents only 30-50 ms of the activation time. In this way, a gap can easily be identified. In **Figure 10.3C**, the entire color spectrum only accounts for 37 ms of activation time. If a point near the CS ostium had been taken, all the points on the lateral side of the CTI line would display dark blue or purple; it would be more difficult to recognize a gap. Pacing close to the linear lesion set and mapping the contralateral side is the strategy that the OU-EP laboratory implements to verify conduction block across any linear lesion set.

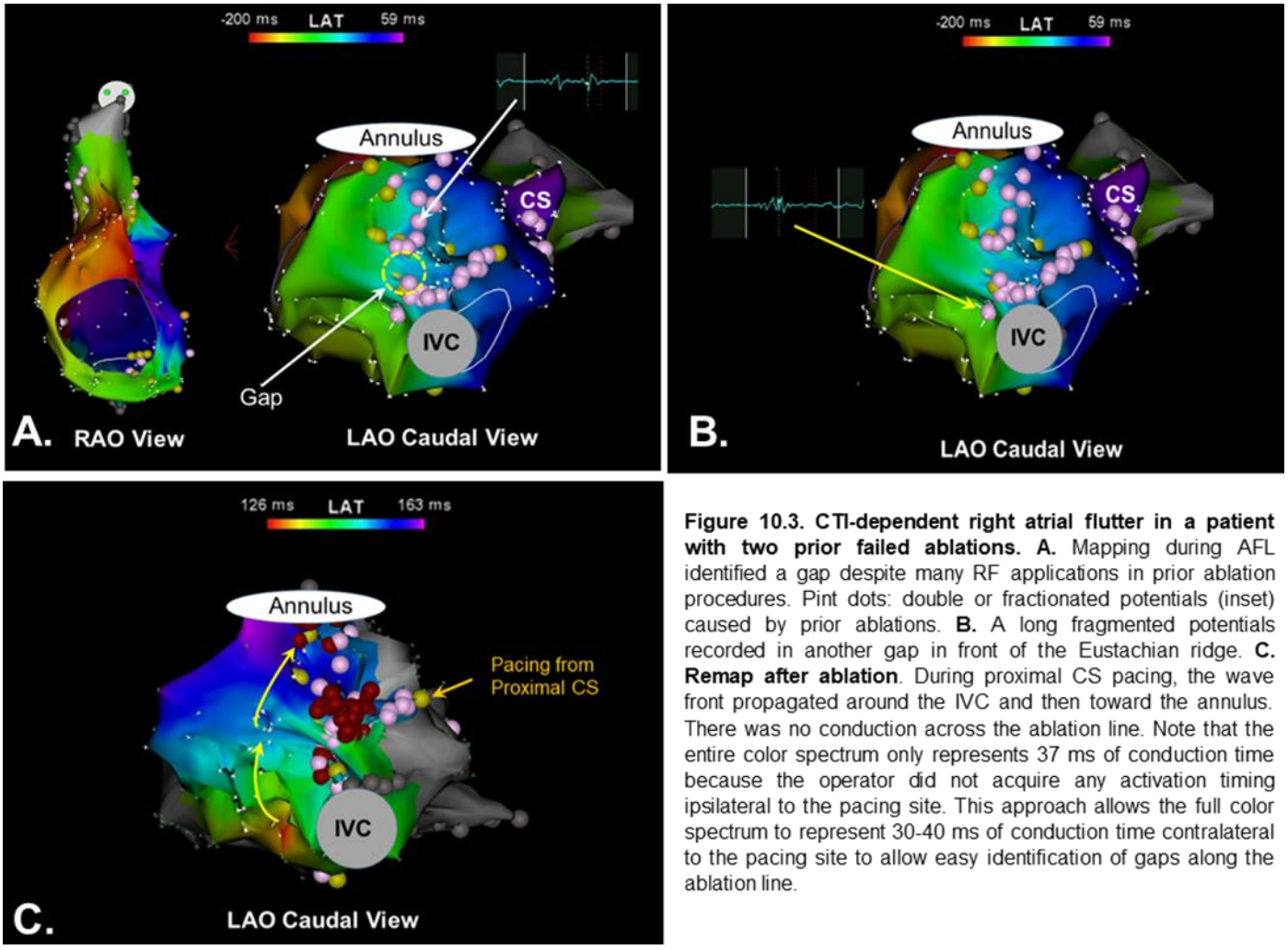


Figure 10.3. CTI-dependent right atrial flutter in a patient with two prior failed ablations. A. Mapping during AFL identified a gap despite many RF applications in prior ablation procedures. Pint dots: double or fractionated potentials (inset) caused by prior ablations. **B.** A long fragmented potentials recorded in another gap in front of the Eustachian ridge. **C. Remap after ablation**. During proximal CS pacing, the wave front propagated around the IVC and then toward the annulus. There was no conduction across the ablation line. Note that the entire color spectrum only represents 37 ms of conduction time because the operator did not acquire any activation timing ipsilateral to the pacing site. This approach allows the full color spectrum to represent 30-40 ms of conduction time contralateral to the pacing site to allow easy identification of gaps along the ablation line.

Macro-reentrant AT after AF ablation

The most common forms are mitral annular AT, roof-dependent AT and AT propagating through gaps along the PVI ablation lines (e.g. propagating horizontally around the PV carina). Macro-reentrant ATs are usually easier to map but creating a durable linear lesion set can be a challenging task due to unpredictable variations of the thickness of the atrial muscle along the line. For example, the LA roof is usually thin near the RSPV-roof junction but may be thick in the middle of the roof. Choosing a safe and effective power/force/time of RF application is always a balancing act.

The arrhythmogenicity of the RA and LA appears to be very different. Based on the experience of the OU-EP group in mapping patients with corrected congenital heart diseases, it is rare to see a macro-reentrant AT in the RA if the width of a potentially arrhythmogenic channel is longer than 3 cm. In contrast, much wider channels in the LA can be arrhythmogenic. For example, mitral annular AT can occur when the distance between the mitral annulus and LIPV is >5 cm. The author hypothesizes that the discrepancy of arrhythmogenesis between the RA and LA may rest on the fact that the LA is more richly innervated by the cardiac autonomic nervous system. Disruptions of autonomic innervation (particularly vagal) by PVI can easily lead to marked ERP dispersion and help initiate and maintain macro-reentrant ATs.

Micro-reentrant AT after AF ablation

The reentrant circuit of micro-reentrant ATs is typically <2 cm in diameter and frequently iatrogenic, caused by non-transmural ablation lesions at sites such as PV-LA junction, base of LA appendage, LA septum

and anterior wall of LA. Non-transmural lesions create slow conduction zones or protected isthmuses, facilitating the formation and maintenance of micro-reentrant ATs. Entrainment or resetting delivered to the small reentrant circuit should produce concealed fusion and a short PPI. However, the risk of tachycardia termination can be very high if one attempts to entrain the tachycardia in a slow conduction zone. Given the wide distribution of long, fractionated EGMs in the atria after prior extensive ablation, using entrainment or resetting responses to search a small reentrant circuit may be unrealistic unless a presumed reentrant circuit has been identified by mapping. In the OU-EP laboratory, the grand strategy is to map the tachycardia first. Resetting or entrainment is utilized only after activation mapping is completed but it is not clear if an area in the atrium is in the reentrant circuit or not. In this scenario, entrainment may be delivered to such a site to refine the activation map. If entrainment happens to terminate the tachycardia, the activation map is still sufficient to devise an ablation strategy.

Focal AT after AF ablation

Drs. Kisler and Kalman had published a series of manuscripts of localizing focal ATs based on the P wave morphology (*JACC* 2006;48(5):1010-7). However, in patients with prior AF ablation, the atrial activation wave front is greatly altered by ablation lesion sets. Using the P wave morphology to predict the origin of a focal AT may not be accurate. On a lucky day, a focal AT is diagnosed when the tachycardia terminates and re-initiates multiple times or the tachycardia cycle length (CL) wobbles >15%. However, in the vast majority of cases, it is difficult to differentiate a focal AT from a reentrant AT without mapping. Unlike reentrant tachycardia in which similar PPIs are produced by the same pacing CL, attempts to entrain a focal AT with the same pacing CL often result in great variations of PPI because a focal AT cannot be entrained.

Electro-anatomical mapping is the most accurate, albeit difficult, method to make a correct diagnosis of a focal AT. In the most typical cases, a centrifugal activation pattern is identified and a large percentage of the tachycardia CL is also missing. This feature can distinguish a focal AT from a micro-reentrant AT; the entire tachycardia CL of the latter can be found and a large portion of the CL is confined to a small area which often exhibits long fractionated potentials. Occasionally, a focal AT happens to originate in a site adjacent to a line of conduction block. Conduction time to the other side of the line happens to be long enough to be similar to the tachycardia CL, giving the operator the false impression that the entire tachycardia CL has been mapped out and mistakenly diagnosed the AT as a macro-reentrant AT (**Figure 10.4**).

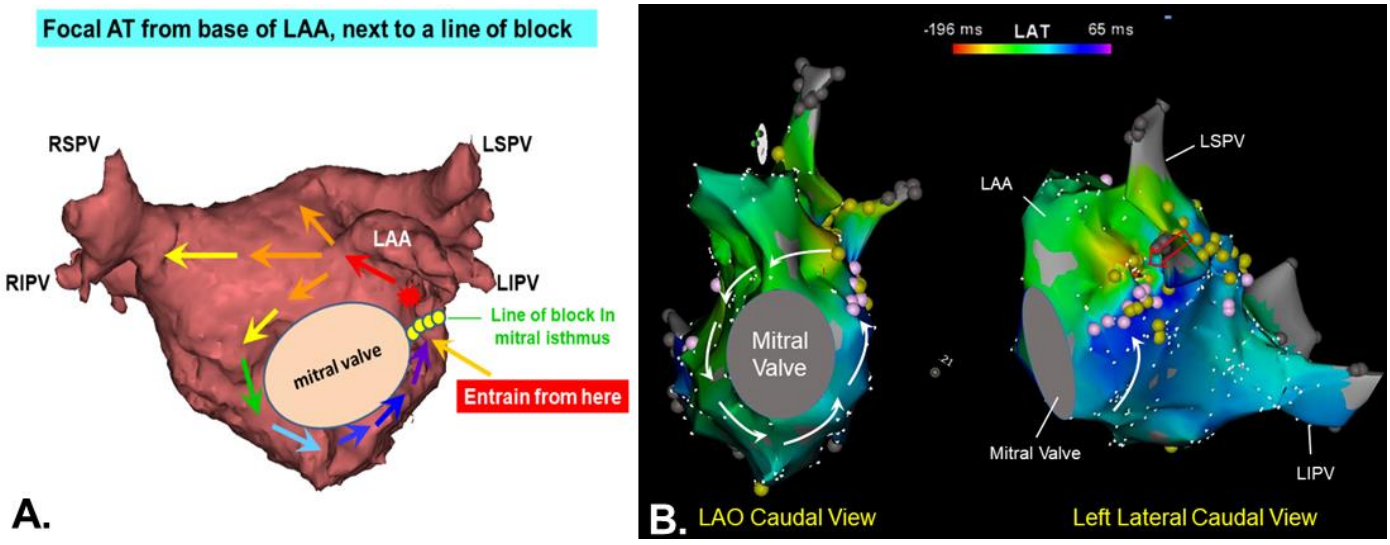


Figure 10.4. Focal AT masquerading as a reentrant AT. **A.** In this schema, there is a focal AT originating from the base of the LAA, above a line of conduction block in the mitral isthmus. The propagation time around the mitral annulus happens to be similar to the AT cycle length, masquerading as a mitral annular AT. Overdrive pacing delivered from the site below the mitral isthmus line (brown arrow) should result in manifest fusion and a long and inconsistent PPI or cannot suppress the AT at all. **B.** A patient with prior PVI and mitral isthmus line. This AT appeared to be a counterclockwise mitral annular AT. Conduction

across the mitral isthmus was indeed blocked. This AT was a focal AT from the lateral aspect of the LAA base. Higher density mapping was then performed at the site where the “head-meets-tail” appeared to be (red arrows). Activation mapping still missed 30 ms (9%) of the total tachycardia cycle length. This example also underscores the importance of setting the CL window to be 100% of the tachycardia CL, not 90%. Pink dots: double potential. Gold dots: fractionated potential.

How to Map an Atrial Tachycardia?

Foremost, what is the targeted arrhythmia?

In patients who have had prior AF ablation, the 12-lead ECG of a stable AT may look like AF (irregular RR interval, invisible P waves). Even a typical right atrial flutter may look atypical. Blindly re-isolating the PVs probably will not eliminate the clinical AT unless the AT depends on the gaps along the PVI line. The CS catheter is often the catheter helping operators to differentiate between AT and AF. Notably, the proximal CS, particularly from the ostium to Vieussens valve, is covered by CS myocardium. During stable AT, it is not uncommon to see dissociation of the atrial and CS myocardial potentials. Far-field ventricular potentials, dissociated CS and atrial potentials may create irregularly irregular EGMs, leading to the wrong diagnosis of AF **(Figure 10.5**). In the OU-EP laboratory, the standard practice is to push the CS catheter into the great cardiac vein (beyond the Vieussens valve) to minimize the interference from dissociated CS myocardial potentials. In **Figure 10.5D**, it is evident that after pushing the CS catheter farther into the great cardiac vein, the clinical arrhythmia was an AT, not AF. AT was successfully ablated after high-density CARTO mapping.

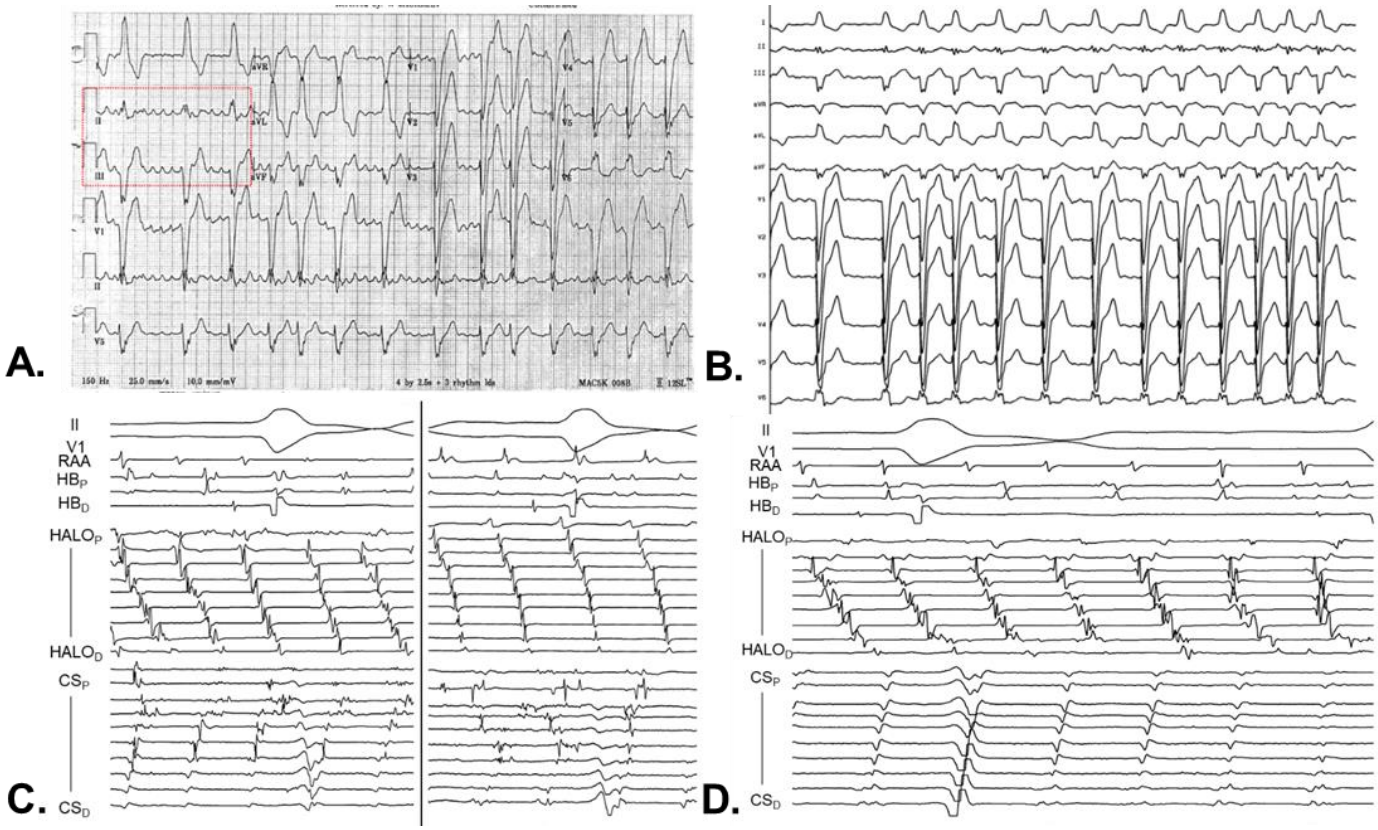


Figure 10.5. Recurrence of atrial tachyarrhythmia after two prior ablation procedures for paroxysmal AF. **A.** Regular P waves (lead II, III) of the clinical tachycardia suggested AT. **B.** ECG recorded in the EP laboratory suggested AF. **C.** The EGMs recorded on the HALO catheter positioned along the tricuspid annulus suggested AT or flutter; however, the CS EGM of both panels suggested AF. **D.** When the CS catheter was advanced beyond the Vieussens valve, it was evident that this arrhythmia was an AT.

Entrainment first or mapping first?

In the OU-EP laboratory, the choice is always activation mapping over entrainment. The reason is obvious; activation mapping provides substantially more information about the AT and potential ablation targets. Attempts of entrainment can easily terminate the AT and render it non-inducible. The author remembers vividly a live case presented in the 2007 Boston AF symposium. A patient presented with persistent AT after AF ablation. The operator elected not to map the AT but to attempt entrainment mapping to figure out the whereabouts of the reentrant circuit. Attempts of entrainment quickly terminated the AT and it could not be re-induced. For ATs in patients with repaired congenital heart diseases (e.g. Fontan procedure), attempt to entrain the tachycardia runs a very high risk of losing the tachycardia. In addition, many Fontan patients have focal ATs as well; activation mapping is always a much better choice.

Figure 10.6 illustrates the value of activation mapping. One can create a crude map of the posterior wall and assess the direction of the wave front. In a roof-dependent AT, the atrial activation sequence of the LA posterior wall is either roof-to-bottom or bottom-to-roof, depending on the direction of the wave front propagation. In a peri-mitral AT, wave front collision would collide on the LA posterior wall. The caveat of this rapid assessment is illustrated in **Figure 10.6C** in which entrainment at the roof will produce a short PPI. However, without more detailed activation mapping, the narrow channel of the inferior-posterior wall cannot be identified. Instead of ablating this narrow channel, a long roof line will need to be made to stop this AT. The recurrence rate is also much higher if a long roof line has to be made.

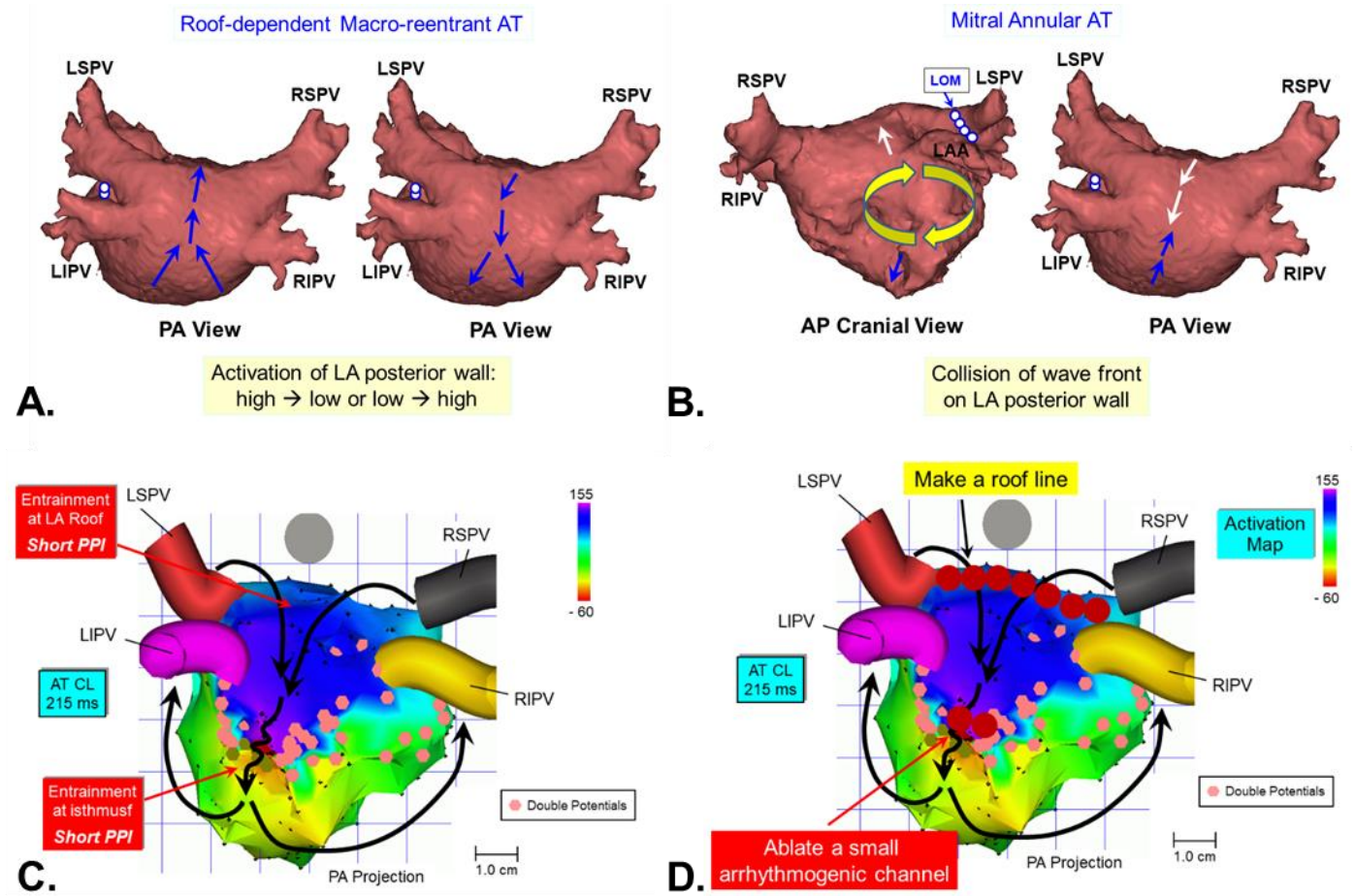


Figure 10.6. A-B. Quick assessment of peri-mitral and roof-dependent AT. A. In a roof-dependent AT, the atrial activation sequence of the LA posterior wall is either roof-to-bottom or bottom-to-roof, depending on the direction of the wave front propagation. **B.** In a peri-mitral AT, wave front collision on the posterior wall of LA is expected. **C-D.** Entrainment at the roof as well as the narrow channel on the posterior-inferior wall produced short PPI. It is obvious that the small arrhythmogenic channel is a better ablation target. Pink dots and red dots indicate double/fractionated potentials and ablation sites, respectively. *Courtesy of Drs. Jackman and Nakagawa.*

In the OU-EP laboratory, entrainment is usually performed *after* activation mapping. Sometimes, entrainment is used to determine which atrium to map first. In order not to terminate the targeted AT, the author prefers to deliver entrainment to the atrium *less likely* to house the AT. For example, in a patient undergoing multiple AF ablations, the clinical AT is more likely to be an LA-AT (e.g. tall, positive P waves in lead V1). If entrainment from the RA produces manifest fusion with a long PPI, activation mapping of the LA is then performed. The author usually delivers RA entrainment *twice* to the lateral RA free wall, approximately 2 cm lateral to the 9 o'clock position of the tricuspid annulus (**Figure 10.7A**). This particular site is in the reentrant circuit of an AT propagating around an RA free wall scar as well as in the circuit of a CTI-dependent atrial flutter. Entrainment here produces a short PPI for either tachycardia. If entrainment was delivered to the CTI during an RA free wall AT, manifest fusion and a long PPI may give operators the wrong impression that this AT originates in the LA. The reason to entrain the AT twice at each pacing CL is to help differentiate a reentrant AT from a focal AT. The former should produce very similar PPIs. The latter tends to produce different PPI by the same pacing CL because the tachycardia was only suppressed, not entrained. **Figure 10.7B-D** illustrates a typical example of a double-loop RA tachycardia in a patient *without* prior history of cardiac surgery. Entrainment from the CTI showed concealed fusion and a very short PPI, indicating that this was a typical RA flutter. However, activation mapping identified two reentrant loops. CTI ablation alone is unlikely to terminate this AT, which may lead to excessive ablation in the CTI and cause tamponade. For this reason, if entrainment verified the diagnosis of CTI-dependent AFL, the author prefers to quickly map the RA free wall.

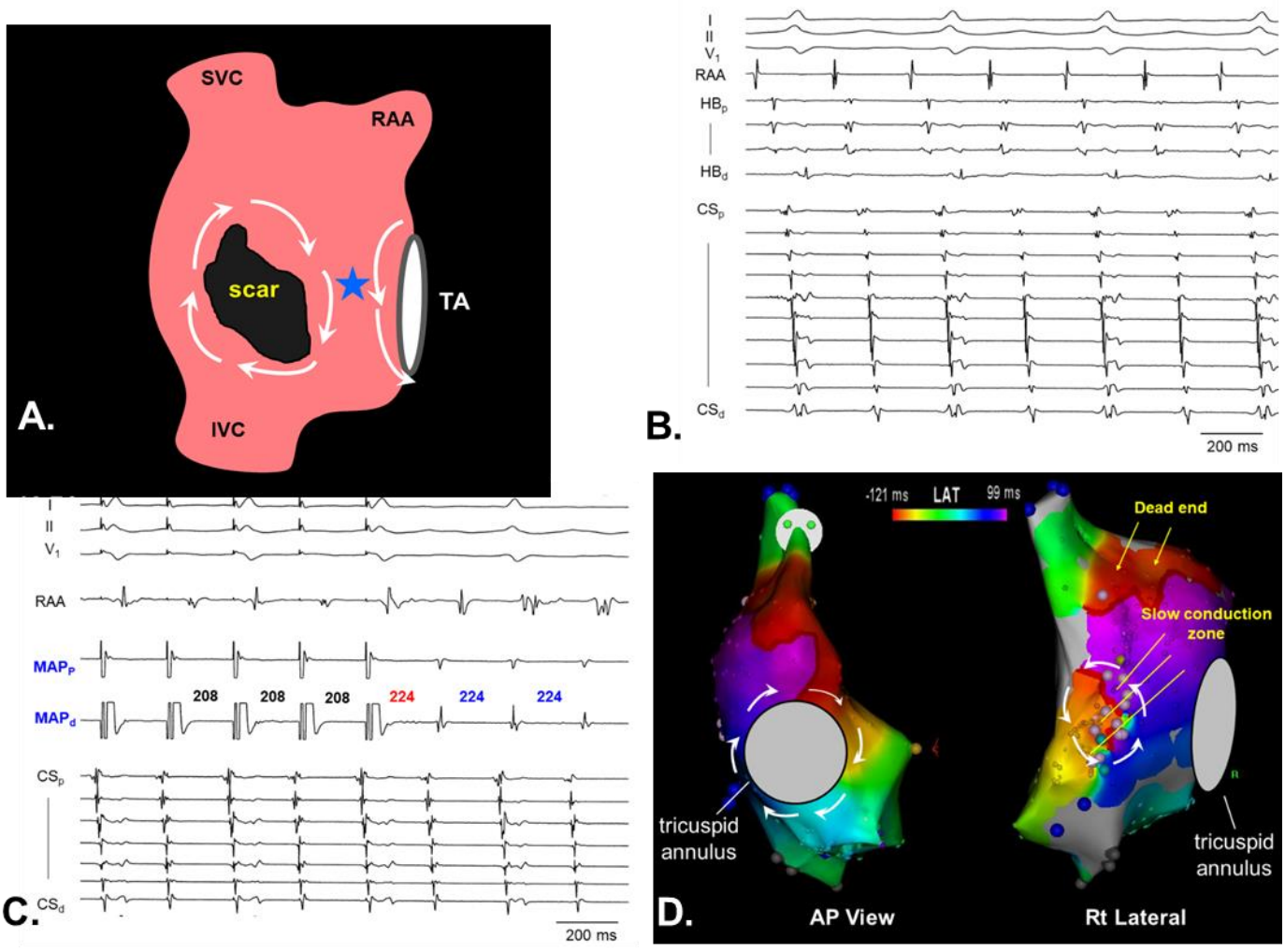


Figure 10.7. Entrainment of an RA macro-reentrant tachycardia. This patient did not have prior cardiac surgery. **A.** Lateral free wall 2 cm from the annulus (blue star) is in the circuit of a RA free wall reentrant AT and a CTI-dependent AFL. **B.** Intracardiac recordings of a patient presented with an atrial tachycardia, consistent with a clockwise atrial flutter. **C.** Entrainment from the CTI showed concealed fusion and a PPI equaled the tachycardia CL. **D.** Activation mapping showed that this was a

double-loop reentrant AT. One loop was a clockwise AFL (left panel). The other loop propagated around a linear scar on the RA free wall. Note that the activation timing of the RA-SVC junction appeared to be early but it was indeed very late and fell into the window of the next tachycardia cycle (dead end).

Which atrium to map first?

A left atrial tachycardia (LA-AT) can conduct to the RA through three major routes: Bachmann's bundle, inter-atrial septum and CS in decreasing order of prevalence (**Figure 10.8A**). LA-ATs preferentially conducting to the RA through the CS often originate in the septal aspect of the LA (**Figure 10.8B**). In addition to the 3 aforementioned major routes, direct myocardial connection between the RA and RSPV or between the RA and LA outside the inter-atrial septum have been reported. It is not clear how often these myocardial fibers connecting the RA and LA contribute to conduction between the RA and LA during ATs.

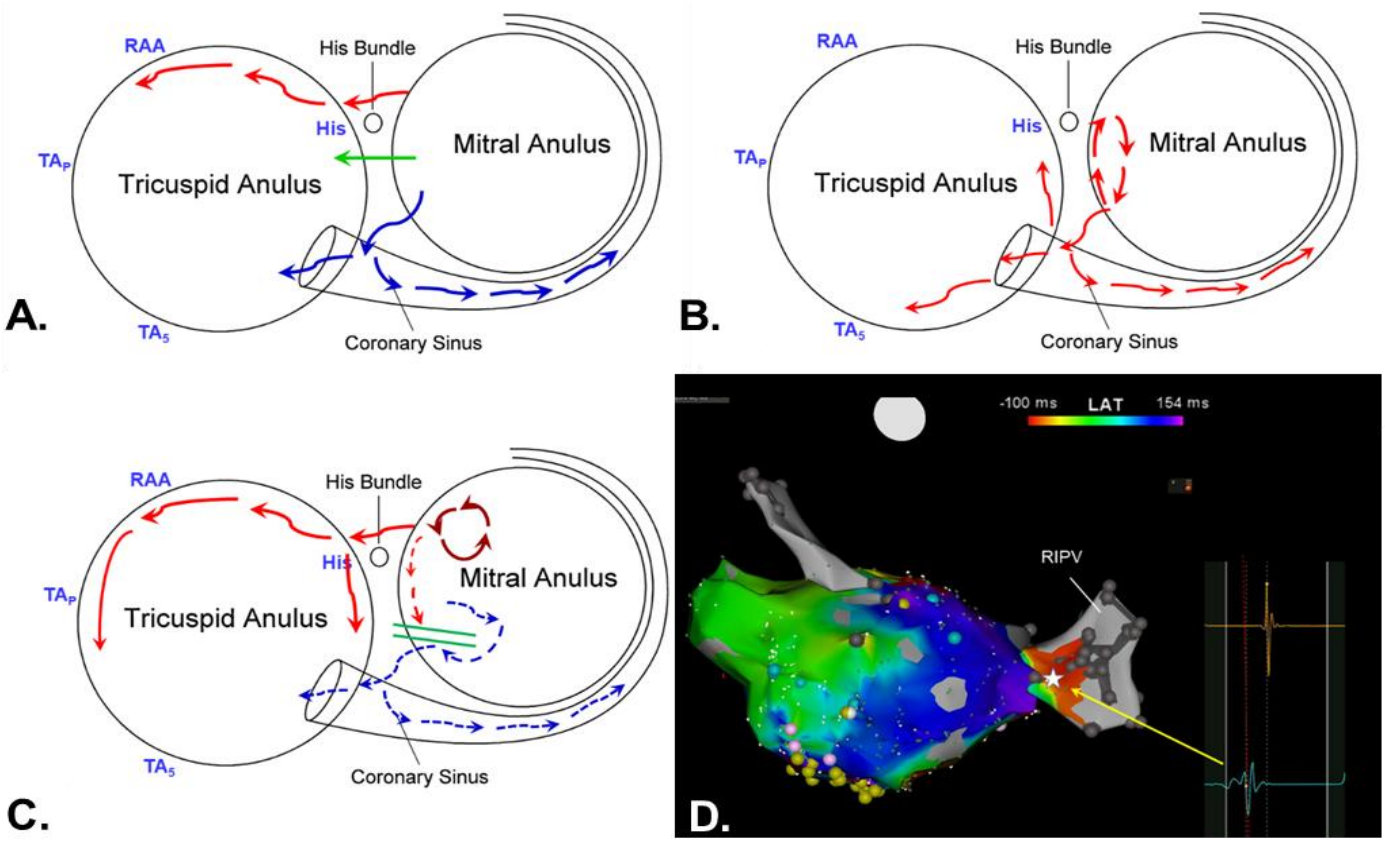


Figure 10.8. Conduction from LA to RA. A. Three major routes of conduction from LA to RA: Bachmann's bundle (red arrows), inter-atrial septum (green arrow) and CS (blue arrows). **B.** CS is the least common route of LA to RA conduction but it often occurs in the presence of a septal LA-AT. **C.** In the presence of extensive LA ablation, conduction time from the LA septum to CS is either blocked (double green lines) or markedly slowed (dotted blue lines). A septal LA-AT may preferentially conduct to the RA through the Bachmann's bundle (red arrows), creating a false impression that wobbling of the CL in the RA precedes that of the CS and leading to the wrong conclusion that this is an RA-AT. **D.** An area in the RIPV showed "early" timing (white star), suggesting a focal AT from the RIPV. However, the timing of all the points outside RIPV were very late, indicating that the activation timing of the points in the RIPV were also very late, falling into the window of the next tachycardia cycle and appeared early.

The first step in mapping an AT is to decide which chamber to map first. Atrial activation sequence of the AT may provide some help. For example, if the earliest atrial activation is bracketed at posterolateral CS, it is unlikely a right atrial tachycardia (RA-AT). However, both RA-AT and LA-AT can produce an activation pattern of CSp to CSd or CSd to CSp. If the CS catheter is positioned in the distal great cardiac vein, a septal RA-AT conducting to the LA through the Bachmann's bundle can produce a CSd to CSp activation sequence, masquerading as an LA-AT. Before operators attempt to entrain or map the AT, it is prudent to detect any wobbling of the tachycardia CL. In general, if CL wobbling in the LA precedes that in the RA, it suggests an

LA-AT. On the contrary, if CL wobbling in the RA precedes that in the LA, it suggests an RA-AT but with one major caveat. When the LA was ablated extensively and many zones of slow conduction or conduction block were created, an LA-AT located on the septal-anterior wall may preferentially conduct to the RA through the Bachmann's bundle. At the same time, conduction toward the CS is impeded by these slow conduction zones, creating a false impression that CL wobbling in the RA precedes that in the CS and leading to the wrong conclusion that this is an RA-AT (**Figure 10.8C**). Importantly, the CS catheter only covers the inferior to anterolateral LA adjacent to the mitral annulus. The activation timing of the anterior wall and posterior wall of the LA may not be reflected by the activation timing on the CS catheter. If wobbling of the tachycardia CL and the atrial activation sequence do not help determine the origin of the AT and 12-lead ECG does not suggest an RA-AT, the standard practice of the OU-EP group is to map the LA without entrainment if the patient had AF ablation before. If entrainment needs to be done before mapping, the author delivers overdrive pacing from the chamber where it is *less* likely to house the AT to avoid premature termination of the AT. A typical response of entraining an LA-AT from the RA is to entrain the entire RA without affecting the tachycardia.

Specific issues regarding mapping an atrial tachycardia

Mapping an AT can be a daunting task because annotating the local activation timing can be confusing in the following situations: (1) wobbling of the tachycardia CL, (2) multiple low-voltage areas, (3) numerous slow conduction zones, (4) double or fractionated EGMs and (5) very late potentials in dead ends masquerading as the "head" of the reentrant wave front or the origin of a focal AT (**Figure 10.8D**). The best example of such "dead end" sites is the PV potentials in an LA-AT after prior PVI. The PV potentials are passively activated by the LA-AT and usually very late due to slow conduction into the PV. PV potentials may fall into the window of the next AT cycle, creating false "early timing" on the activation map

As mentioned in the beginning of this book, the OU-EP laboratory does not have the Ensite mapping system. However, the principles of electro-anatomical mapping should hold true for the CARTO, EnSite and Rhythmia systems. With the rapid advances of technology, it is expected that all the three mapping systems will have similar accuracy in the near future. What is described in this chapter should be applicable to all the three mapping systems.

1. Reference selection

Selection of a stable timing reference is a prerequisite for mapping. A good reference EGM is either monophasic or biphasic and is stable regardless of the respiratory or cardiac cycle (**Figure 10.9)**. A multi-phasic EGM creates problems of sensing and triggering by the mapping system and should be avoided if possible. In patients with advanced stages of AF or with extensive scars from prior surgery or ablation, the CS may have diffusely low amplitude, multi-phasic EGMs, making them not suitable for reference. In this scenario, placing a circular catheter in the LA appendage may be a good alternative. Ideally, the reference should not be too far away from the targeted arrhythmia; however, the highest priority is reference stability. For example, the RA appendage may not be suitable to be the reference to map an LA-AT. If there are multiple zones of slow conduction or conduction block between the location of the reference electrode and the arrhythmia, the reference may be one CL behind the LA-AT. Points on the activation map come from different cardiac cycles, causing the map to be very confusing, particularly when CL wobbles.

Another problem that operators often encounter is the interference from far-field ventricular activation. Somehow the algorithm of CARTO-3 tends to select the atrial beat concurrent with a QRS complex, leading to inaccurate annotation of the local atrial timing (**Figure 10.9A**). The author prefers to select an atrial beat not overlapped with a far-field ventricular potential. In the presence of a slow AT with 1:1 AV conduction, it can be challenging to differentiate a local atrial potential from a local ventricular potential. In this situation, single ventricular extra-stimuli can move the ventricular potential away to expose the true local atrial activation timing (**Figure 10.9B**).

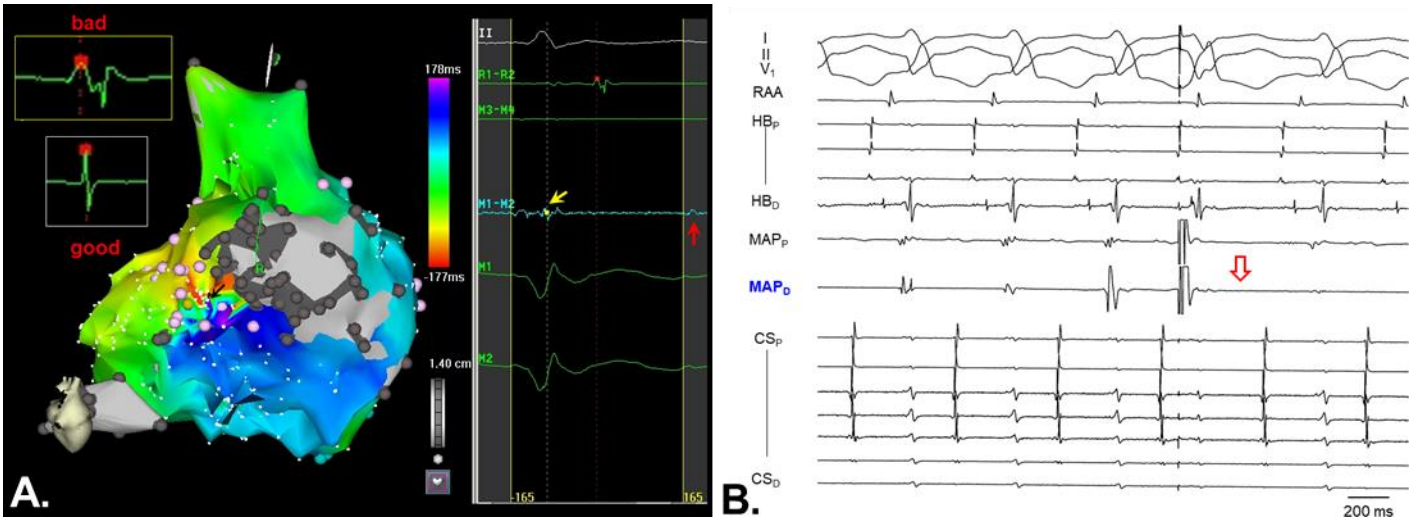


Figure 10.9. Common problems during mapping. A. Two reference EGMs are displayed in the left upper corner. The bad reference has multiple components; the good reference has a biphasic EGM with a large amplitude. The timing selected by the computer algorithm (red dot) in the bad reference EGM is suboptimal due to its multiple components. Another problem is to take the atrial timing in the middle of a far-field ventricular potential. The algorithm of the CARTO system selected a potential that was indeed a far-field ventricular potential (yellow arrow, right panel). In the next CL window (without far-field ventricular EGM), it is evident where the correct timing should have been selected (red arrow). **B.** In an AT with 1:1 AV conduction, the activation timing of the sites along the AV annulus can be very confusing due to interference from far-field ventricular potentials. In this situation, single ventricular extra-stimuli can move the far-field ventricular potential away to reveal the true local atrial timing. In this example, all the potentials on the mapping catheter were far-field ventricular potentials. The empty red arrow denotes the absence of atrial potential at this site.

2. Selection of the window of tachycardia CL

For unfounded reasons, Biosense representatives had been teaching electrophysiologists to select the window of tachycardia CL only amounting to 90% of the true tachycardia CL. This practice, in the opinion of the OU-EP group, is a big mistake as it will miss critical information about the tachycardia and potentially create a false “head meets tail” site. The standard practice of the OU-EP group is to set the widow exactly the same as the tachycardia CL. Sometimes tachycardia CL shows fixed wobbling (e.g. alternating between 220 and 234 ms), which often suggests the presence of a reentrant tachycardia conducting at its maximal capacity. An explanation for the short-long CL wobbling is like a person running along a circular track at his/her maximal capacity. S/he runs slower when tired but faster after getting some rest by running slower. In this scenario, operators may set the CL window as the shorter CL (e.g. 220 ms) to minimize the problem of double counting in which one of the two immediate neighboring points is assigned to very early activation timing but the other point is assigned to very late timing.

The author prefers to set the left margin of the CL window 100 ms before the onset of the p waves in order to capture a focal tachycardia. For reentrant AT, it is a continuous activity. Therefore, the timing of each point in reference to the p wave is not important. However, it is unknown to the operator if the targeted AT is focal or reentrant before mapping is completed. Operators can to set the left margin of the CL window significantly earlier (e.g. 100 ms) before the onset of the p wave to make it easier to recognize a focal AT.

3. How to annotate the timing of an EGM

There are multiple ways to annotate the timing of an EGM in the electro-anatomical mapping system, such as rapid down stroke of the distal unipolar EGM also known as max dV/dt, beginning of bipolar EGM, maximal voltage of bipolar EGM and minimal voltage of bipolar EGM. When the EGM is monophasic or biphasic, assigning local activation timing is straightforward as the component with the largest slope (max dV/dt), maximal voltage or minimal voltage can easily be identified. When the EGM has multiple components,

annotating the local activation timing becomes very challenging. In fact, the local activation timing of each point depends on the activation timing of the points surrounding it. One can easily appreciate that a smart computer algorithm would work much better than a human brain to deal with a network of activation timing. During the developmental phase of the Rhythmia mapping system, the inventors of the system spent a great deal of time observing how Drs. Jackman and Nakagawa annotated complex EGMs, similar to the way IBM Watson learned to play chess. The algorithm of annotating complex EGMs in the Rhythmia mapping system is based on the concept that each local activation timing is dependent on the timing surrounding it. A summary of how the OU-EP group annotates the activation timing is listed below.

1. At first, make a crude map during tachycardia in the chamber of interest, taking points approximately 2 cm apart. This crude map will provide a "bird's eye view" of the tachycardia (**Figure 10.10**). Monophasic or biphasic electrograms that the operator is confident in annotating the activation timing will provide important insight into the big picture of the activation map. These straightforward points (**Figure 10.11A**) indeed serve as the "anchor" to help annotate the adjacent confusing points. Often, a crude map is enough to identify the normal conduction zones, which are usually not the sites where ablation can terminate the AT. However, they help identify the direction of wave front and where to find the slow conduction zones. Operators may set the left pane of the mapping screen as an activation map and the right pane as voltage map to provide complementary information to help identify critical arrhythmogenic channels (**Figure 10.10**).

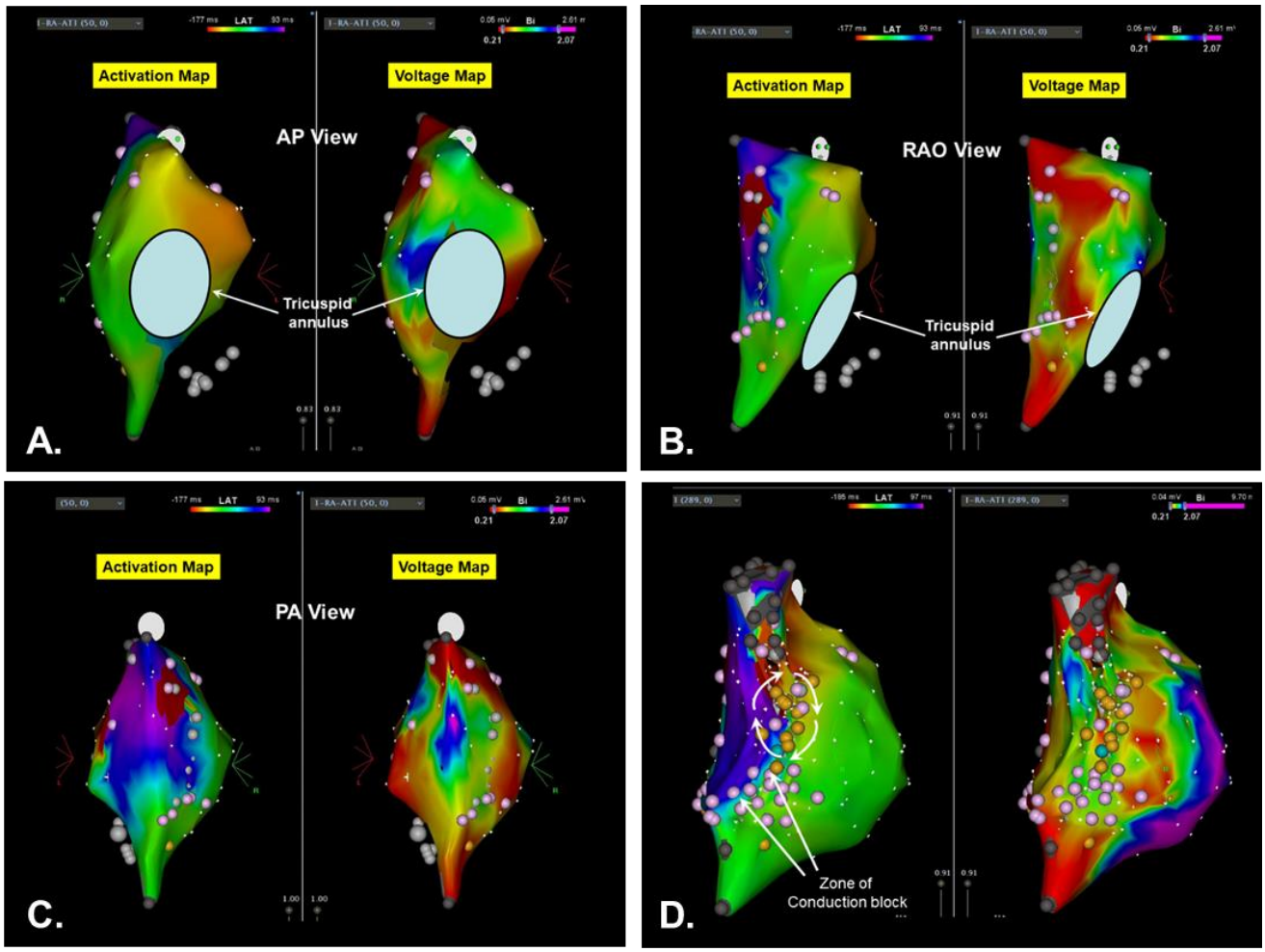


Figure 10.10. Mapping of an RA-AT: left panel: activation map; right panel: voltage map. A. At first, make a crude map, taking points approximately 2 cm apart, to get a "bird's eye view" of the tachycardia. It is evident that there was no low voltage area on the anterior and anterolateral wall adjacent to the tricuspid annulus and this AT was not a CTI-dependent atrial flutter. **B.** The RAO view showed at least one low voltage area on the free wall with slow conduction. **C.** The PA view showed slow conduction, indicating that the business end of this AT is somewhere along

the posterolateral aspect of the RA free wall. **D.** High-density mapping showed a reentrant AT propagating around a slow conduction zone (gold dots). This patient had common AV canal and underwent 3 cardiac surgeries before. Pink dots: wide double potentials indicative of conduction block during AT. Gold dots: continuous EGM or EGM with multiple components.

Points with double potentials or fractionated potentials are tentatively assigned with timing based on the corresponding sharp component on the distal unipolar EGM (UNI-1) or timing of surrounding points (**Figure 10.11**). These points are tagged with different colors (pink points for double potentials, gold points for fractionated potentials as practiced by the OU-EP group). After a more detailed map is acquired, activation timing of these pink and gold points will be adjusted, based on the timing of the surrounding points. In addition, a voltage map also provides critical information about the location of the low voltage areas. Importantly, low voltage areas do not always equate to scars. The author prefers to assign a point as a scar only if the contact force is higher than 10 grams and no local EGM is recorded. Similar to myocardial bundles in an infarct scar in patients with ischemic cardiomyopathy, low voltage areas in AF patients may harbor potential arrhythmogenic channels. If the noise level of an EP lab is 0.1 mV, all the arrhythmogenic channels with a voltage less than 0.1 mV will be missed. In the OU-EP laboratory, the average noise level, using ThermoCool catheters, is 0.02-0.03 mV. However, we rarely select ablation targets based on a voltage map. The voltage map is to provide complementary information to the activation map. Blind homogenization of a low voltage area is almost never performed in the OU-EP laboratory. This practice potentially can introduce more non-transmural lesions and set the stage for more micro-reentrant AT or focal AT.

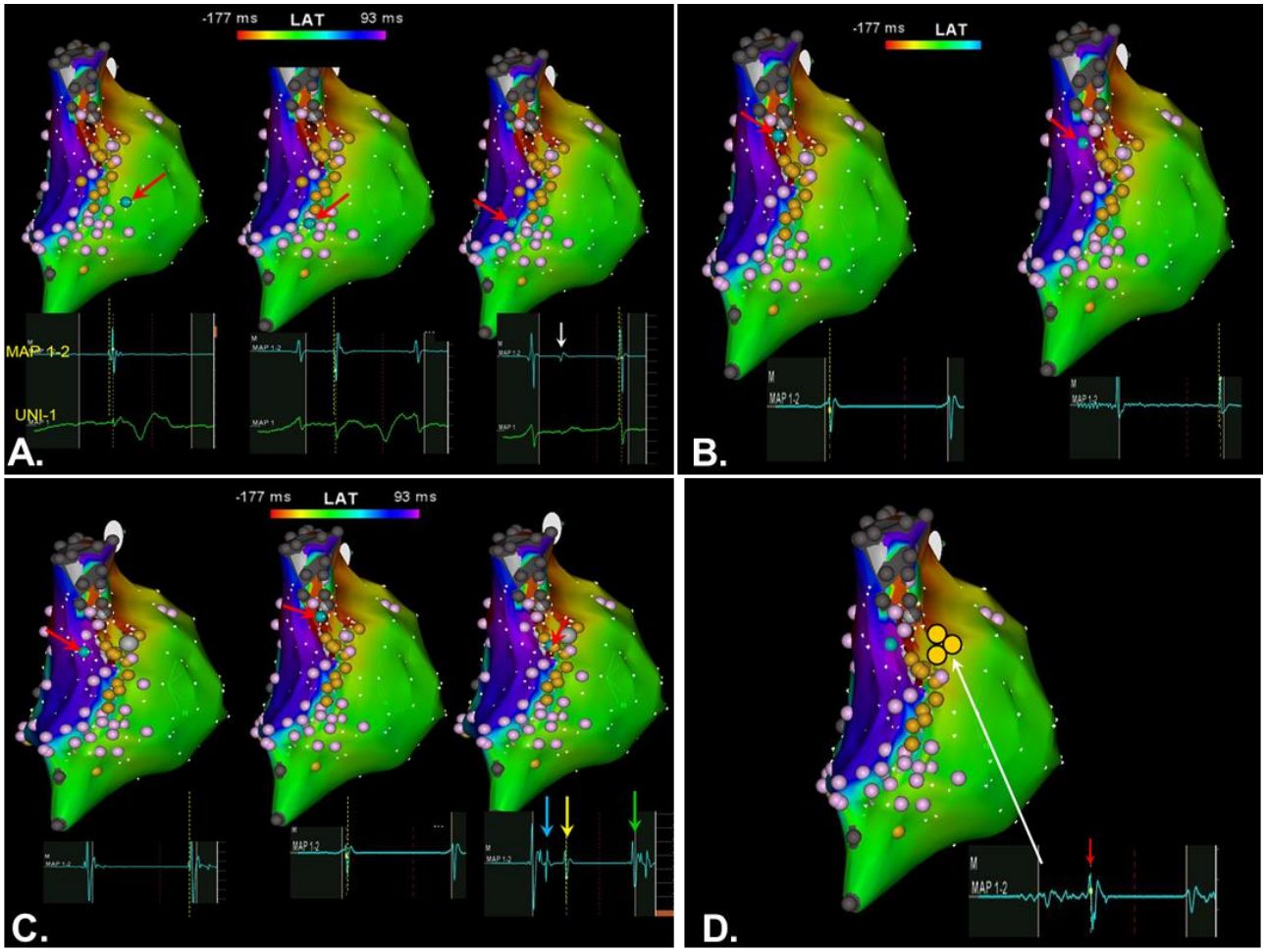


Figure 10.11. Assigning local activation timing (the same patient as in Figure 10.10). A. After the direction of wave front has been figured out, the timing of double potentials can be re-adjusted based on the timing of the unipolar EGM and the timing of surrounding points. **Left panel.** A biphasic atrial potential that the local activation timing can be selected easily. This point can be used as the “anchor” to help determine the activation timing of a neighboring

point showing a double potential (**middle panel**). Note that the sharp unipolar potential in the right panel helped select the local activation timing. The point displayed in the right panel (on the other side of the surgical incision line) also helped assign the timing of the double potential in the middle panel. The white arrow indicates a far-field potential corresponding to the near-field potential in the middle panel. **B.** At the site where "head meets tail", operators can adjust the timing to very early (left panel) or very late (right panel) after mapping is completed, based on the direction of wave front propagation and timing of surrounding points. Note that very early or very late timing does not affect the map anymore because the entire CL has been found. **C.** The EGM displayed in the right panel had 3 components. From the neighboring points displayed in the left and middle panel, it is evident that the late component (green arrow) was a far-field potential from the other side of the line of conduction block. There was another point (not shown) slightly inferior to the point displayed in the middle panel that had the same timing as the first component (blue arrow) of the right panel. Since the wave front propagated inferiorly from the point in the middle panel, the timing of the point displayed in the right panel was assigned to the 2nd component (yellow arrow). Note that the interval between the yellow and green arrow was 148 ms, indicating conduction block in tachycardia between the two potentials. Therefore, the business end of this tachycardia must be more upstream from this point. **D**. At sites with continuous low-amplitude potential (three gold dots), timing of this EGM is not important because the surrounding points indicate that the activation wave front passes through this slow conduction zone to reach the point displayed on the right panel in **C**. The timing indicated by the red arrow was the same as the timing indicated by the yellow arrow in the right panel in **C.** Ablation here terminated AT.

2. High-density mapping focusing on areas of interest (e.g. slow conduction zone or sites of prior ablation) is conducted. For activation timing straddling the edge of the CL window**,** it is challenging to assign it as very early or very late. Again, the operator should look at the surrounding points to determine the direction of the activation wave front to help assign the correct activation timing (**Figure 10.11**). If this point is truly located at a site in the reentrant circuit, the points surrounding it should show progressively earlier timing in one direction and later in the other direction. Later, "purple" points can be adjusted to "red" points and "red" points can be adjusted to "purple" points to make sense of the map. For a reentrant AT, what is very early or very late is not important as long as the operator knows that the wave front propagates from one side of the "head-meets-tail" region to the other side. Operators need to be aware that an "early" point may be in a blind alley, though. If the author is not sure about the activation time of a given point, that point can be tagged as a "location only" point to be evaluated after more points are acquired. It would better not have a point than have a bad point.

3. Areas showing 2:1 or Wenckebach conduction during AT should be taken as scar tissue to avoid confusion because activation timing of these points can be random (**Figure 10.12**) and these sites do not participate in the tachycardia. However, operators need to keep in mind that this area is not a true scar and can participle in another tachycardia with a longer CL. The author prefers to annotate in the Bard recording system as "CARTO point xyz, Wenckebach conduction" as a reminder that this point is not a true scar. In addition, points with concurrent far-field ventricular activation should be avoided to prevent assigning far-field ventricular timing as local atrial activation timing (**Figure 10.9B**).

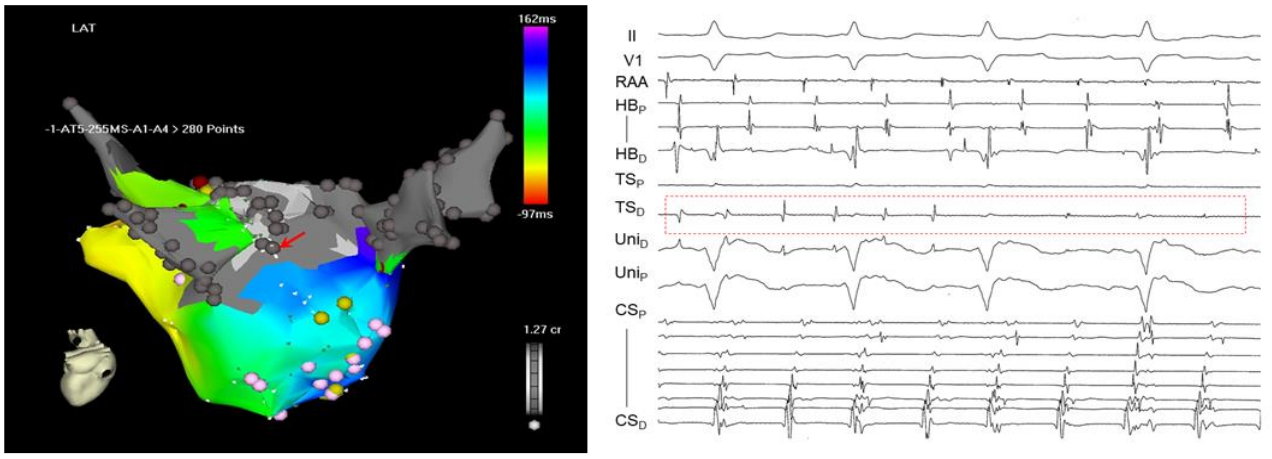


Figure 10.12. A site with activation timing that was dissociated from the tachycardia should be assigned as a scar or location-only point to avoid contamination of the activation map.

4. Confusion may arise when a very late point falls into the window of the next CL and appears to be “early” in the next window and can cause multiple “head-meets- tail” areas. **Figure 10.13A** illustrates an area with early activation timing outside the reentrant circuit. This is a typical example of a dead end or blind alley where late potentials fall into the next tachycardia cycle and appeared to be early on the CARTO map. Confusion also can be caused by residual atrial-PV conduction. During sinus rhythm, these PV potentials may be >100 ms later than the adjacent antral potential. During AT, these potentials can be so late that they fall into the next window and appear early on the activation map (**Figure 10.8D**). However, one should not automatically assign residual PV potential as very late timing in patients with a history of prior PV isolation to avoid missing PVI gap-dependent ATs. The timing of adjacent antral points should provide the clue.

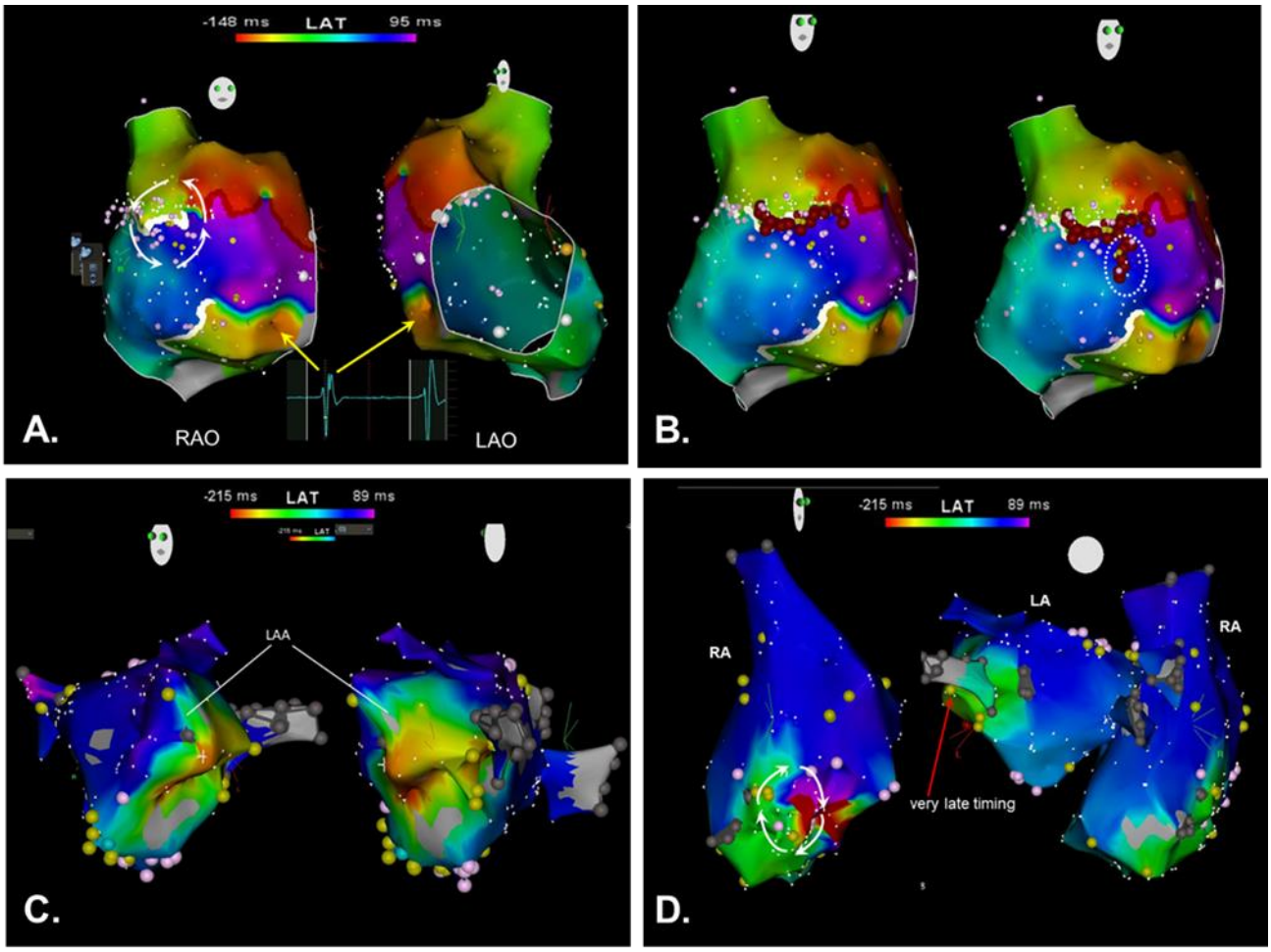


Figure 10.13. AT in a young patient with prior ASD repair. A. Left panel. A reentrant tachycardia propagating around high anterolateral RA wall in a counterclockwise direction was evident. In the inferolateral RA, there was an area showing “early” activation timing (yellow arrow). This area indeed represented very late activation so that the timing of the local EGM fell into the window of the next tachycardia cycle. **Right panel.** Because of this very late activation time in the inferolateral RA adjacent to the annulus, it was evident that there was *no* concurrent clockwise atrial flutter. **B. Left panel.** The author’s stupidity led to the making of a linear ablation lesion set along a line of widely-spaced double potentials. AT was not terminated by this linear lesion set because conduction block already occurred in this area during tachycardia. The linear lesion was not extended more laterally to avoid SAN injury. **Right panel.** A short vertical linear lesion to target another slow conduction zone in the circuit (inside the white dotted circle) terminated the AT. **C.** In another patient with prior LA ablation, mapping of the LA suggested that it was a focal AT originating in the LAA. Note that the yellow and blue color on the activation map was extrapolated by the CARTO algorithm. There were barely any points around the LA appendage, misleading the operator to believe that this was a LA appendage tachycardia. **D. Left panel**. Mapping of the RA showed that this AT was a reentrant AT propagating around the RA free wall in a clockwise direction. Ablation there terminated the AT. **Right panel.** A bi-atrial view indicates that the “early timing” of the LAA indeed was very late, falling into the window of the next tachycardia cycle.

Sometimes, an isolated area with “early timing” may be caused by a concomitant focal AT coexisting with a reentrant AT. If ablation targeting the reentrant AT prolongs the tachycardia CL but fails to terminate the AT, the operator may consider remapping the tachycardia to see if the current tachycardia is a focal AT. If one looks into the original map carefully, one may find centrifugal activation from this isolated early area but activation collides with the reentrant wave front.

5. Sometimes, there appears to be two competing ATs. The activation sequence of each AT is stable for seconds or minutes, which was replaced by the other atrial activation sequence for seconds or minutes. The two alternating activation sequences suggest two competing ATs, not uncommon in AF patients taking dofetilide or amiodarone that organize AF into 2 competing ATs. To map this type of ATs, the operator may call up specific EGMs in the mapping system to differentiate the two ATs. For example, if AT1 shows CSd to CS2 conduction while AT2 shows CS2 to CSd conduction. The operator can map AT1 first (the predominant AT) and ignore all atrial beats with an activation sequence from CS2 to CSd **(Figure 10.14).** After AT1 is successfully ablated, AT2 can then be mapped.

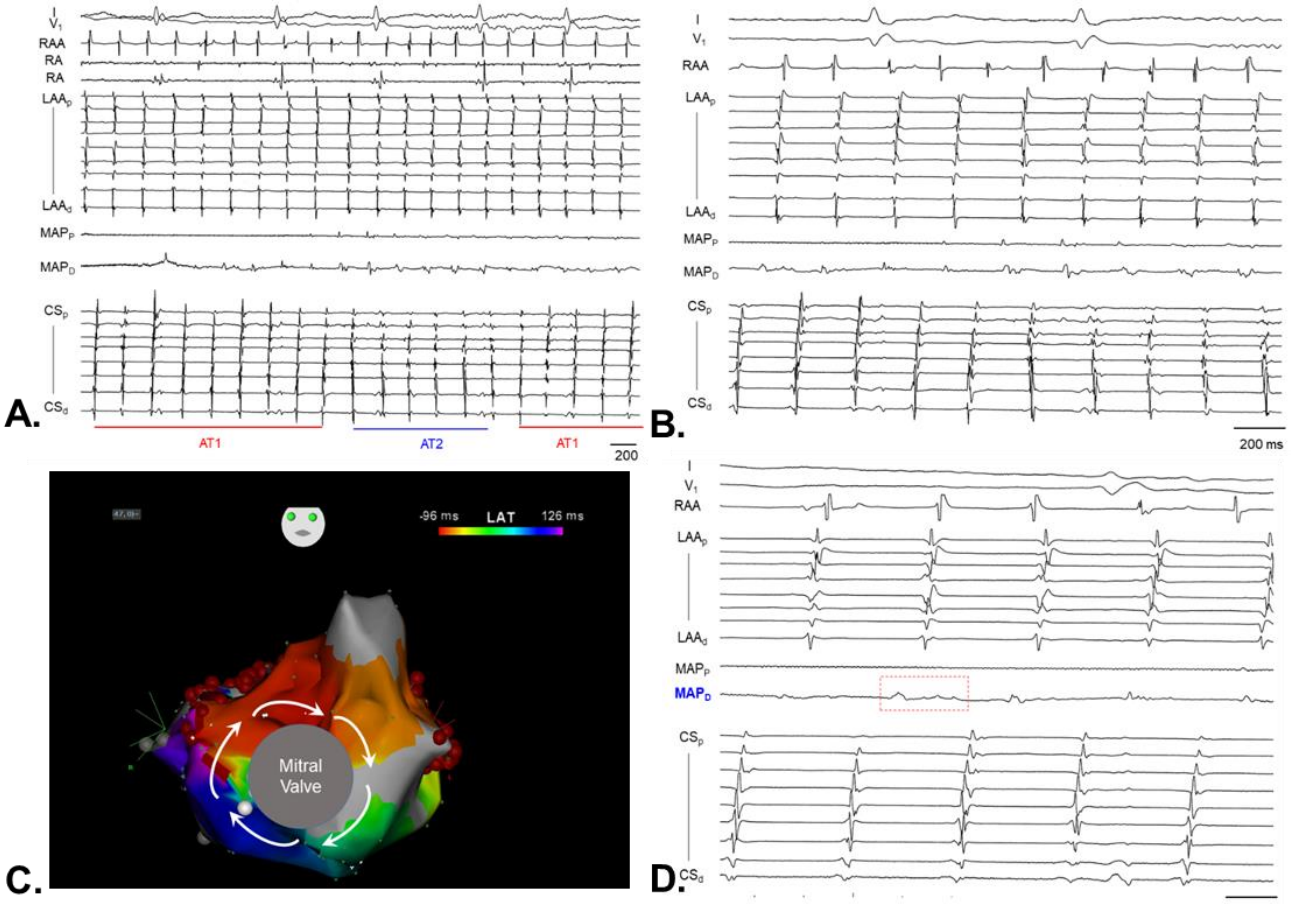


Figure 10.14. Two competing ATs in a patient with two prior ablations. A. AT1 and AT2 had different CS activation patterns. **B.** EGM with a higher sweep speed. **C.** By ignoring AT2, a low-density CARTO map suggested that AT1 was a clockwise mitral annular AT. **D.** More detailed mapping of AT1 verified that it was a clockwise mitral annular AT. Mapping catheter was positioned at one of the gaps along the mitral isthmus line made in prior ablations. AT1 was not inducible after conduction block across the mitral isthmus line was accomplished.

6. After a high-density map is acquired, the operator readjusts the timing of the pink and gold points (double and fractionated potential, respectively) as well as the red and purple points (e.g. very early vs. very late) to make sense of the map. The definition of a reentrant tachycardia in electro-anatomical mapping is (1) continuous activity with head-meets-tail and (2) total activation time equals the tachycardia CL. If the activation map identified the critical element of the tachycardia but cannot

differentiate a micro-reentrant tachycardia from a focal tachycardia, the standard practice of OU-EP is not to attempt to entrain or reset the tachycardia but to ablate it. This is because resetting or entrainment delivered to a slow conduction zone carries a high risk of terminating the tachycardia.

7. Sometimes, high-density mapping still misses 10-15% CL of a *reentrant AT* or the entire tachycardia CL is found but a portion of the activation timing is missing. This problem often occurs in patients after extensive ablation, creating endocardial scars but preserved epicardial conduction. This is particularly challenging in patients after 28-mm cryoballoon ablation, which can lead to a large area of epicardial conduction If a significant portion of tachycardia CL is missing, one should concentrate on the area between the “early” and “late” timing to look for the missing CL as this area may harbor the missing CL. For example, a small slow conduction zone with long fractionated potentials straddling the edge of the CL window may account for the missing CL.

4. Multi-electrode fast mapping

CARTO, EnSite and Rhythmia mapping systems all provide multi-electrode fast mapping. To date, Rhythmia is probably the most accurate one to differentiate a focal AT from a reentrant AT thanks to its closely-spaced small electrodes and computer algorithm. When the CARTO system is used to map an AT, the OU-EP group continues to use point-by-point activation mapping because a map with <300 good points is usually enough to find the AT; erroneous or uncertain timing assignment can easily be found and corrected using the methods already discussed. At the same time, multi-electrode fast mapping using a PentaRay catheter is being cautiously adopted. Our main concern is that multi-electrode fast mapping may acquire too many points that are annotated erroneously by the algorithm. It is difficult to find and correct so many erroneously assigned points to make sense of the map. The algorithms of CONFIDENSE and RIPPLE mapping are steadily improving and are expected to be substantially more reliable in the near future.

The author had many opportunities of chairing sessions of difficult ablation cases in various meetings. A recurrent scenario is that the presenter showed an activation map of a complex AT acquired by multi-electrode mapping. There were >3000 points on the map, but the map made no sense at all because of numerous bad points with erroneous activation timing assigned by the automated algorithm. Operators did not know where the bad points were and there were too many to be corrected. It appears that operators were not actively thinking when they performed multi-electrode mapping and passively expected that computer would tell them the critical elements in the tachycardia. The quality of multi-electrode mapping often depends on how the atrial wall was “painted” by the multi-electrode catheter. Sometimes, the most critical area only had scanty points but areas not important had hundreds of points. Sometimes, critical small, fractionated potentials were erroneously rejected by the algorithm. Therefore, operators should actively look for areas important for tachycardia while performing multi-electrode mapping to ensure these areas of interest are covered well, rather than passively sweep across the atrial wall and expect the answer will come out of the mapping system.

5. The role of double potentials, fractionated potentials

The timing of a potential within the tachycardia CL provides little insight into the role of that point in the tachycardia. Before CARTO was available, Dr. Jackman’s experience was that at the site where ablation terminated the tachycardia, the timing of 1/3 of the sites was presystolic, 1/3 was systolic (within the P wave) and 1/3 was diastolic. Similarly, 1/3 sites showed single, double and fractionated potentials, respectively. Many successful ablation sites exhibit fractionated potentials, often representing diseased myocardium with slow conduction. However, most of the fractionated potentials do not participate in AT; it is not advisable to

ablate these potentials to treat an AT without mapping. This practice more likely will produce more non-transmural lesions and set the stage for more focal or reentrant ATs. **Figure 10.15** showed two EGMs, a biphasic one with normal amplitude and a fractionated one with low amplitude. Many operators would choose to ablate the later because it appears to reflect diseased myocardium and slow conduction. However, the former may represent a site that prior ablation completely missed; the latter was a bystander site. Without activation mapping, blindly ablating fractionated potentials may only introduce more non-transmural lesions.

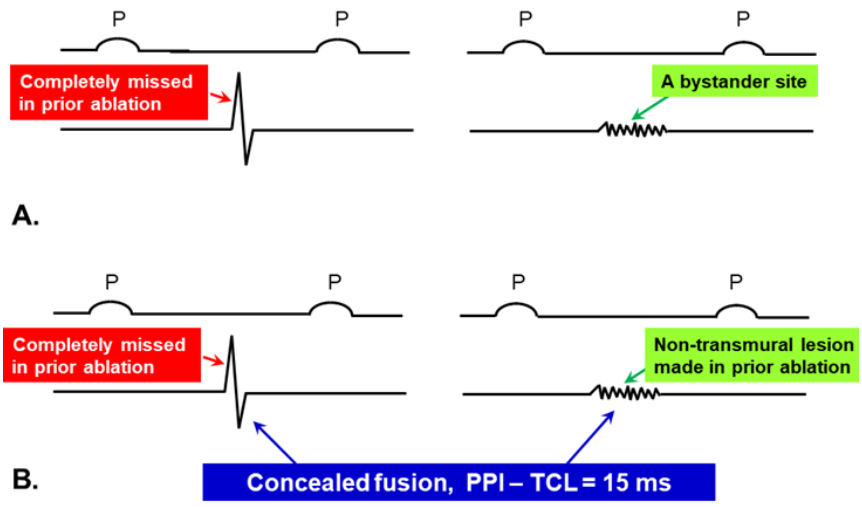


Figure 10.15. Two diastolic potentials. Their roles in the reentrant circuit can only be defined by activation mapping. **A.** Using CTI-dependent atrial flutter as an example, the EGM in the left panel may represent a site that was in the CTI but was completely missed in prior ablations. The EGM in the right panel may be a bystander site, not a critical element in the circuit (e.g. RA free wall). Ablation should not target the fractionated potential. **B.** If entrainment from both sites produced concealed fusion and a PPI 15 ms longer than the tachycardia CL, both points are located in the AFL reentrant circuit. The EGM of the left panel may represent a point that was completely missed in prior ablation. The EGM of the right panel may represent a non-transmural lesion made in prior ablation. Ablation should target both sites.

A site showing wide double potentials typically represents conduction block *in tachycardia* at these specific sites; ablation at these sites usually does not affect the tachycardia because conduction block already occurred. **Figure 10.13B** (left panel) illustrates that the author mistakenly made a linear lesion set along a line of wide double potential, which did not affect the tachycardia at all. What the author should have done is to carefully map the area exhibiting double potentials to look for continuous potentials (e.g. shown in **Figure 10.11D**, which often represent slow conduction sneaking through an “apparent” line of conduction block. If no such potential is found, it indicates that this is truly a line of conduction block in tachycardia. Ablation there would not affect the tachycardia.

However, wide double potentials in tachycardia may represent functional conduction block; it may be able to support another AT with longer CL. The standard practice of the OU-EP group is to identify the entire reentrant circuit first. After successful elimination of the tachycardia and gaps along linear lesion set, remapping during long pacing CL (600-700 ms) is performed to determine if conduction block across these double potential sites is present or not. If in doubt, RF applications will be delivered to these sites with double potentials until conduction block is verified by remapping at a longer pacing CL.

6. The role of entrainment mapping

As discussed earlier, the standard practice of the OU-EP group is to use entrainment primarily to determine which atrium to map first as well as help clarify the tachycardia mechanism *after* activation mapping is completed. Our default approach is activation mapping. Entrainment delivered along the mitral annulus or on the LA roof is easy to do and may demonstrate the site of entrainment is located within the reentrant circuit but provides little information about the most effective ablation target. **Figure 10.6C-D** illustrates a good example of selecting an ablation target based on activation map (ablating a small channel vs. making a roof line). Another problem with entrainment mapping is that it is difficult to determine concealed vs. manifest fusion. Unlike entrainment mapping of VT, 12-lead ECG is not very helpful to determine manifest vs. concealed fusion in ATs because of low-amplitude P waves, particularly in patients with congenital heart diseases or after AF ablation. With a limited number of catheters to cover both atria, a wrong conclusion of

concealed fusion may be drawn simply because the 2 or 3 diagnostic catheters failed to reveal manifest fusion.

PPI is therefore the measurement that most operators rely on, which only provides the information of the proximity between the reentrant circuit and the pacing site. Several problems need to be addressed. First, if the reentrant circuit has a short excitable gap, shorter CL of overdrive pacing may produce a PPI >40 ms longer than the tachycardia CL ("increasing resetting response" coined by Dr. Mark Josephson; **Figure 10.16A**), misleading the operator that the pacing site is distant from the reentrant circuit. Based on Dr. Josephson's publications, the author usually starts with pacing CL 7% shorter than the tachycardia CL to minimize this problem. Second, ablation at a site with a short PPI does not guarantee termination of the AT. Using entrainment mapping of VT as an analogy, entrainment delivered to the outer loop of the VT circuit would produce a short PPI but ablation there would not terminate the VT. Similarly, if one entrains a typical RA flutter from the 9 o'clock position of the tricuspid annulus, the PPI will be short but ablation there is unlikely to terminate the flutter. Third, depending on the origin of the AT and the area of the atria covered by catheters, concealed vs. manifest fusion is very difficult to distinguish. Antidromic fusion during entrainment also introduces confusion and can easily be interpreted as manifest fusion. One can imagine what the outcome would be if one performs entrainment mapping for VT without a 12-lead ECG and chooses ablation target only based on PPI. Fourth, entrainment mapping requires multiple attempts of entrainment from different sites, which often leads to change or termination of the AT. Fifth, PPI can be significantly longer in patients taking antiarrhythmic drugs that slow down conduction (e.g. flecainide, amiodarone) or if there is a slow conduction zone in the reentrant circuit that also has decremental conduction properties (**Figure 10.16B-C**). Sixth, stable capture may be difficult to accomplish if the pacing site is severely diseased. If pacing only captured 2 beats, the first non-captured beat may be used to calculate the PPI but measurement may be prone to error.

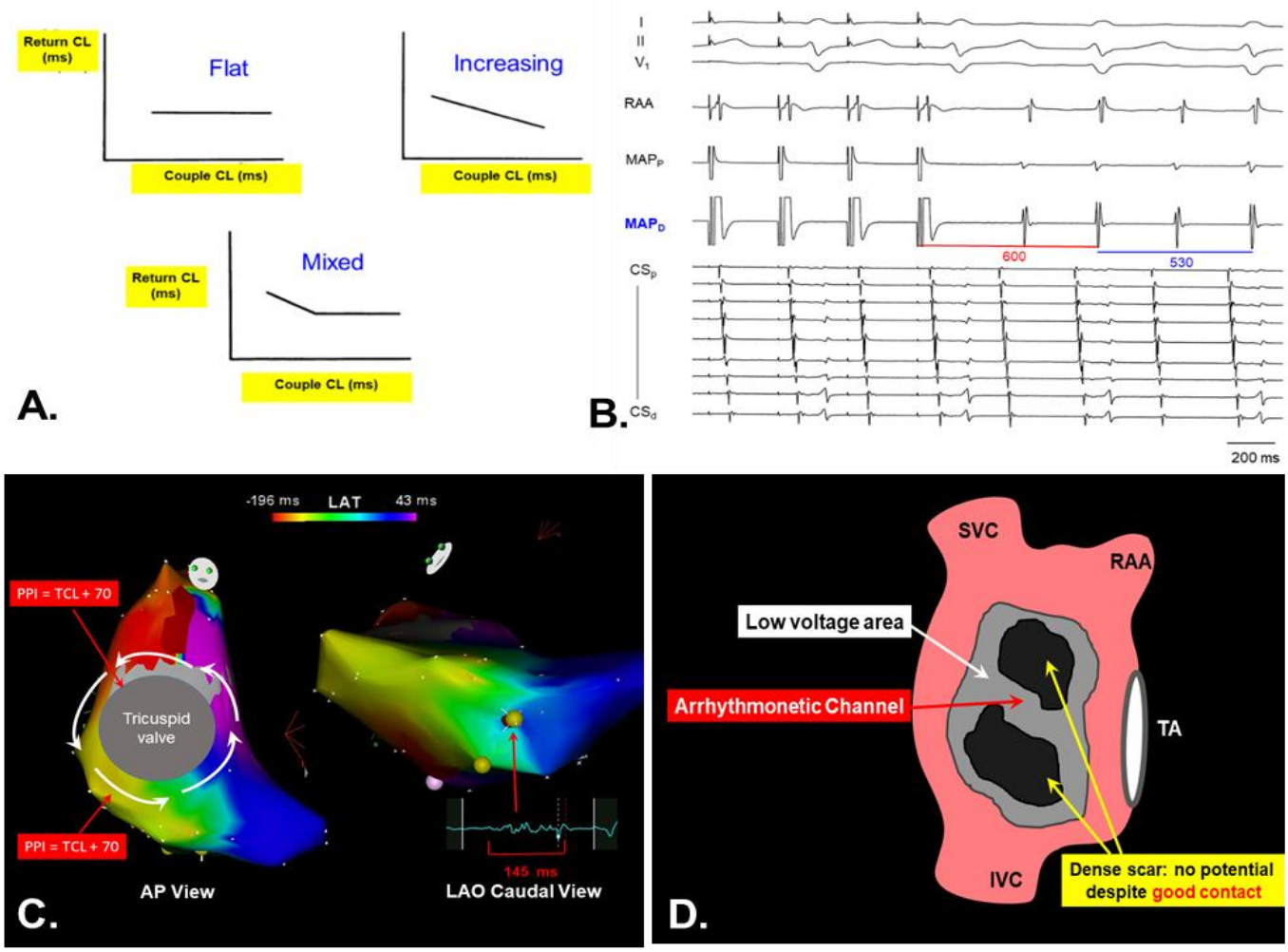


Figure 10.16. A. Three possible resetting responses. The coupling interval of the extra-stimulus is shown along the abscissa, and the return cycle is shown along the ordinate. The flat response usually occurs if there is a large excitable gap in the reentrant circuit. When the coupling interval of the extra-stimulus is short enough, it encroaches upon the refractory period of the tail of the previous beat, producing an increasing response. A mixed response may be the most common response in ATs; therefore, the pacing CL of entrainment should not be too short to cause an increasing response and a long PPI. **B.** In a patient with CTI-dependent AFL (tachycardia CL=265 ms), entrainment (240 ms) delivered from posterolateral tricuspid annulus produced a PPI 70 ms longer than the flutter CL, suggesting that it was not CTI-dependent AFL. **C. Left panel.** Two sites along the flutter reentrant circuit both produced a PPI 70 ms longer than the flutter CL. CARTO map indicated counterclockwise AFL. **Right panel.** It took 145 ms to conduct through a gap in the CTI. Long PPI was caused by slow or decremental conduction through this area. **D.** Schematic representation of an arrhythmogenic channel within an RA free wall scar. If the noise level in the EP laboratory is too high, the entire arrhythmogenic channel will be buried in noise and missed.

8. Low voltage zone vs. scar

With the advent of contact force catheters, it is less likely to misinterpret an area as a low voltage zone or scar due to poor electrode-tissue contact. However, it remains challenging to differentiate a dense scar from a low voltage zone housing areas of slow conduction. The noise level of the EP laboratory plays a determinant role in this. When Dr. Jackman studied ATs in patients with corrected congenital heart diseases (e.g. after Fontan operation), the median amplitude of the arrhythmogenic channels was 0.37 mV. Some potentials were as small as 0.04 mV. If the baseline noise of the ablation catheter is 0.1 mV, all the slow conduction zone with potentials <0.1 mV will be mistakenly labelled as “scar”. In this case, 37% of the arrhythmogenic channels will be missed (**Figure 10.16D**). Electrophysiologists may refer to **Chapter 2** in this book for the details of how to reduce the noise level in the EP laboratory. Connecting two low voltage areas to eliminate the potential arrhythmogenic channels only makes sense if the two low voltage areas are indeed dense scars (no electrogram in the presence of good contact force). If the low voltage areas themselves have numerous arrhythmogenic channels, connecting the two areas is not likely to make any impact on the arrhythmogenic substrate.

Sometimes a very small potential is recorded on the mapping catheter but the operator is not sure if this potential is real or is an artifact. Dr. Jackman uses the following methods to determine the validity of this type of small potentials. First, a real signal will occur at the same timing during each cardiac cycle. Second, the timing of the points adjacent to this small potential should be slightly earlier or later.

9. Patients with prior cardiac surgery

Operators or patients should make good efforts to acquire a copy of the operating report. Knowing how the atria were opened provides enormous help. The author encountered a patient with a very remote history of “cardiac surgery” in her infancy; the operative report was not available. There was no low voltage area in either atrium. The clinical tachycardia was AVNRT and a focal AT. The puzzle was solved when her mother showed up the next day and told us that the “cardiac surgery” was to treat patent ductus arteriosus. If the inter-atrial groove (Waterston’s groove) was incised to allow for mitral valve repair, the clinical tachycardia is often a reentrant AT propagating (1) around the incision site on the septal-anterior wall of the LA or (2) around the right pulmonary veins with or without a concomitant mitral annular tachycardia.

How to verify conduction block across a linear ablation lesion set

Making a complete linear lesion set is sometimes very difficult due to thick myocardium, overlying endocardial scars or poor electrode-tissue contact. Verifying conduction block across the line can be challenging as well. It is well known that gaps along a linear lesion set are very arrhythmogenic. A good example is the STAR AF-II trial in which conduction block across linear lesion sets at the end of the index ablation procedure was achieved in only 74% patients. If one considers the high incidence of conduction resumption after acute success, it is safe to presume that at least half of the linear lesions eventually became arrhythmogenic substrates in the STAR AF-II study, which may account for the results that adding linear lesion sets to PVI did not improve the long-term success of persistent AF ablation.

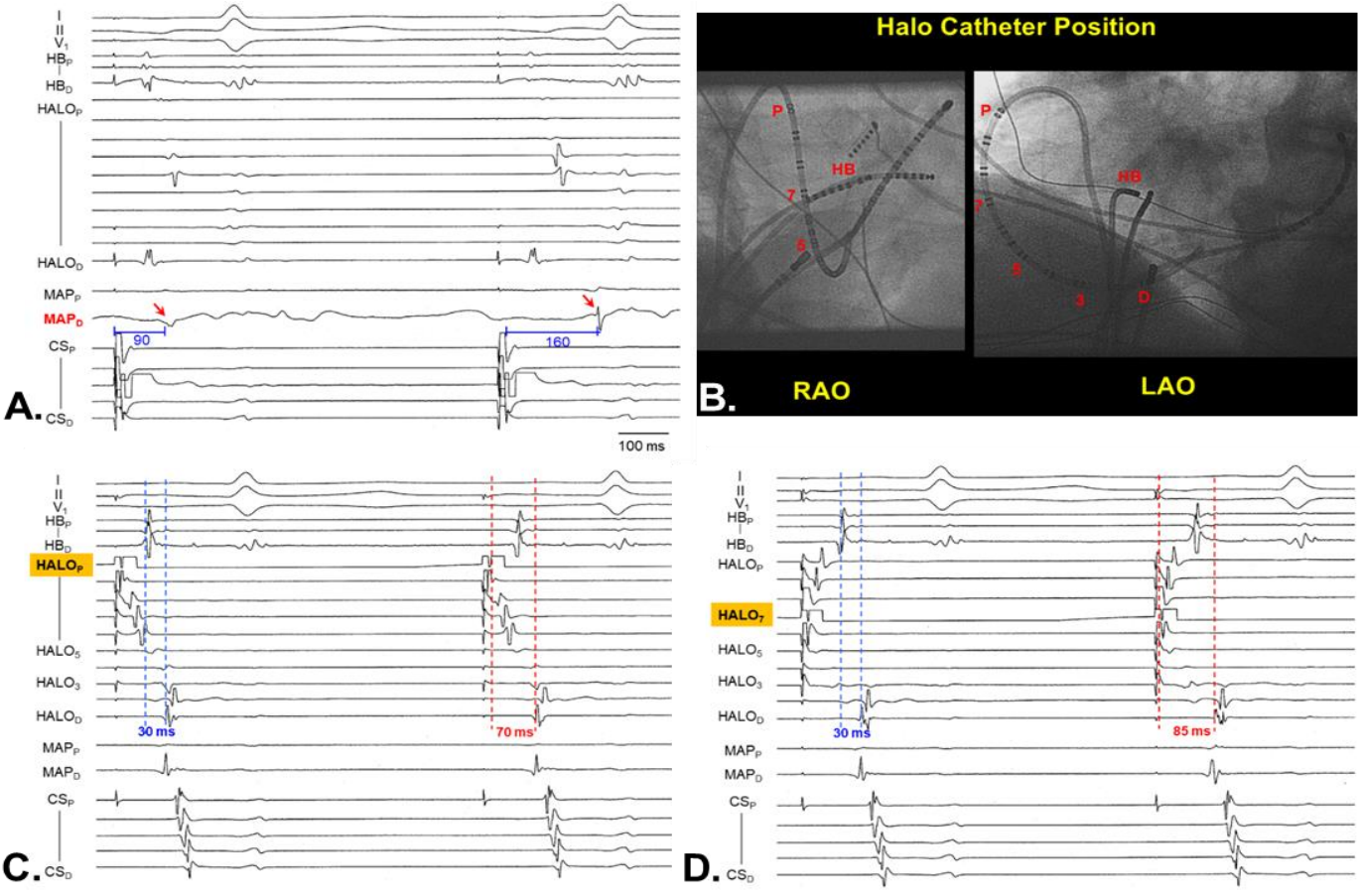
Widely-spaced double potential means conduction block?

During tachycardia, a line of widely-spaced double potentials often suggests conduction block *at that site and at that CL* but provides little information if this line of conduction is functional or anatomical. The former may support another AT with a longer CL. As discussed in previous chapters, the OU-EP group rarely uses an interval to rule in or rule out a diagnosis. Therefore, we do not make the assumption of conduction block based on how wide the double potentials are. The standard practice is to leave these widely-spaced double potentials alone in the first place and target the slow conduction zone believed to be the critical element in the reentrant circuit based on activation mapping. **Figure 10.13B** illustrates an example that a line of widely-spaced double potentials should not have been targeted first because conduction block during tachycardia already occurred. Ablation there did not affect the tachycardia at all.

After the AT was terminated, pacing at a long pacing CL (600-700 ms) from a site in close proximity to the line of conduction block is performed. Electro-anatomical mapping *contralateral* to the pacing site usually can provide a definitive answer if conduction across these double potentials exists or not. If it does, it indicates functional conduction block during tachycardia. RF applications are delivered to the sites where pacing at long CLs revealed conduction. The end point of ablation is conduction block across these double potentials.

Selection of pacing site

Selection of a good pacing site is a requisite for examining conduction block across a linear lesion set. In general, the pacing site needs to be as close to the linear lesion set as possible. For example, CS ostium is an ideal pacing site to examine conduction block across the CTI line. **Figure 10.17** illustrates an example of how pacing sites can affect the judgment of conduction block in a CTI linear lesion set. Pacing too far away from the ablation line may lead the operator to erroneously conclude that conduction block is achieved or not achieved depending on how the paced wave front conducts to both sides of the linear lesion set.



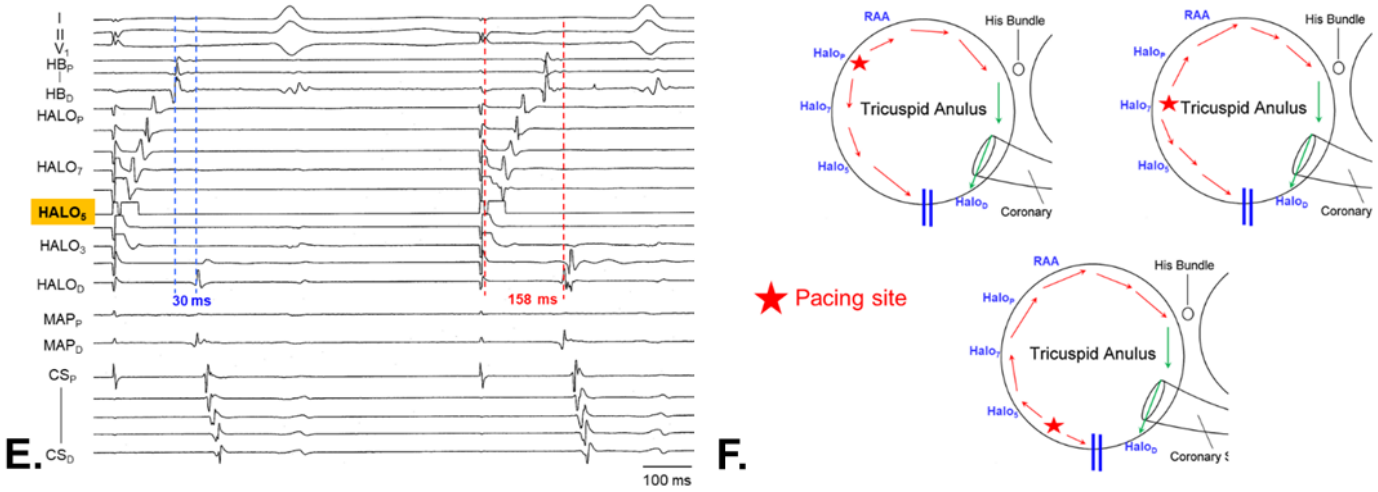


Figure 10.17. Effects of pacing site on judgment of conduction block across a linear lesion set. A. A CTI line was made during proximal CS pacing. Suddenly, the interval between the pacing artifact to the EGM recorded on the ablation catheter (MAPd) increased from 90 ms to 160 ms. **B**. Catheter position. **C.** Pacing from HALO-p, the stimulus-HALOd EGM interval was only 70 ms, suggesting no conduction block. The interval between the atrial timing of the HB catheter and HALO-d was 30 ms. **D.** Pacing from HALO-7, the stimulus-HALOd EGM interval was only 85 ms, suggesting no conduction block. The interval between the atrial timing of the HB catheter and HALO-d was still 30 ms. **E.** Pacing from HALO5, the stimulus-HALOd EGM interval was 158 ms. The interval between the atrial timing of the HB catheter and HALO-d was still 30 ms. **F.** Conduction block across CTI was indeed achieved but the pacing site was too far from the CTI line, giving the impression of no conduction block. Note that the conduction time from the HB area to HALO-d (green arrow) remained stable at 30 ms despite different pacing sites.

There are a number of common mistakes that operators can make while attempting to verify conduction block across a linear lesion set. The absence of double potentials across the line essentially proves that conduction block is absent. However, the presence of "widely-separated" double potentials during atrial pacing does not guarantee conduction block; it only indicates that conduction at that site is significantly delayed. A common mistake is to declare conduction block after seeing a few "widely-separated" double potential along the line (**Figure 10.18**). The interval between the pacing artifact and the 2nd component of the double potential often indicates the conduction time from the pacing site to the other side of the line; the length of this interval often correlates with the chance of conduction block but a long interval between the double potential still does not guarantee conduction block because a gap may exist at a site distant from the sites where a wide double potential is recorded.

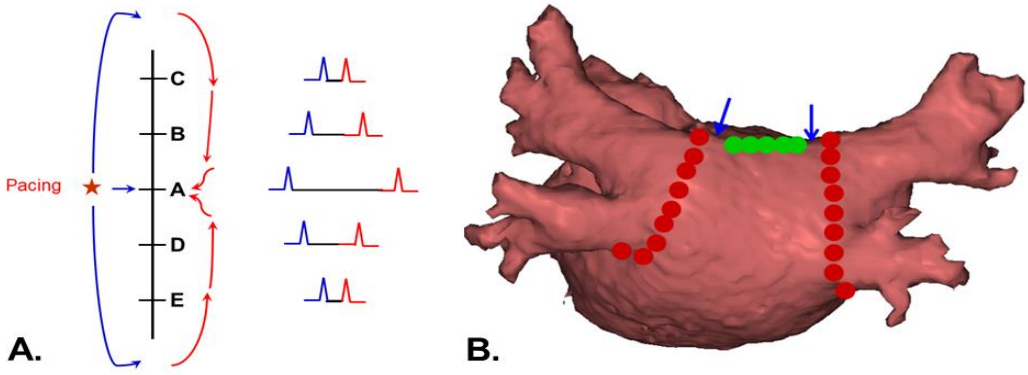


Figure 10.18. A wide double potential in the center of a linear lesion set but gaps exit at both ends of the line. A. In the center of the line, the interval between the two components of a double potential can be very long. If the operator only mapped this point, it would give the false impression that conduction block across the line has been accomplished. **B.** This scenario can easily occur when an LA roof line has gaps on both ends (blue arrows).

The interval between the pacing artifact and the first component of the double potential, indicating the conduction time from the pacing site to the ipsilateral side of the line, is also important. If the interval between the pacing artifact and the first component of the double potential is long as well, one cannot determine if the 2nd component of the double potential is a result of conduction block or slow conduction through a gap (**Figure**

10.19). This problem often occurs when operators attempt to verify conduction block across the LA roof line or mitral isthmus line during LAA pacing in patients who had extensive ablation adjacent to the base of the LAA. In this scenario, both the first and 2nd components of the double potentials along the ablation line are late. The first component is late because of slow conduction zones around the base of LAA created by ablation. One cannot verify if the 2nd potential is late because of conduction block or slow conduction through a gap along the line. **Figure 10.19** illustrates a series of examples of what appeared to be conduction block across the mitral isthmus line but indeed gaps along the mitral isthmus line were still present and mitral annular AT was still easily inducible.

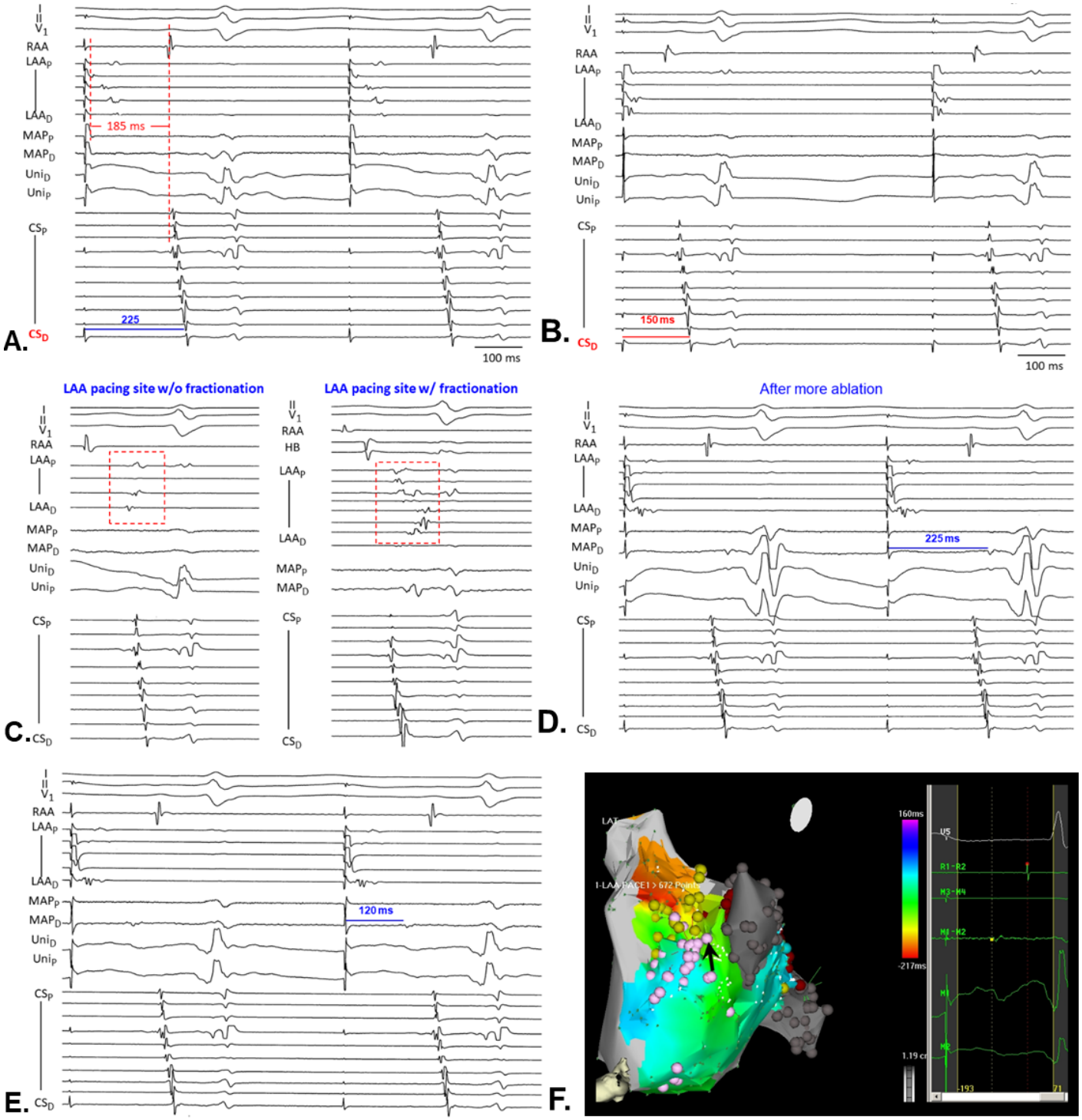


Figure 10.19. Effects of pacing site on judgment of conduction block in a patient with 3 prior AF ablations. A. After ablation of the mitral isthmus between the LPV carina and annulus, remapping below the ablation line was performed during LAA pacing. CSd was below the ablation line. The interval between the stimulus artifact and CSd EGM was 225 ms; activation pattern was CSp to CSd, indicative of conduction block across the mitral isthmus line. Note that the interval from the pacing stimulus to CSp EGM was 185 ms, indicating very slow intra-atrial conduction. **B.** After changing the LAA pacing site, the interval between the pacing artifact and CSd EGM shortened to 150 ms, suggesting the presence of gap along the line. **C.** Different stimulus-EGM intervals between (**A**) and (**B**) were caused by different degrees of slow conduction around the pacing site. In (**A**), pacing was delivered to a site where extensive ablation had been performed (long fractionated potentials). In (**B**), the Lasso catheter was moved proximal to the pacing site in (**A**). More mapping verified that conduction block across the mitral isthmus line was not achieved. Mitral annular AT was still inducible. **D.** After more ablation in the mitral isthmus, remapping below the line, *near the annulus*, identified a late potential (225 ms after the pacing artifact), a promising finding. **E and F**. However, another point near the LIPV was early and was ablated. Conduction block across the mitral isthmus line was eventually accomplished (not shown here).

If the atrial activation time in front of the roof line is late during LAA pacing (**Figure 10.20**), the author prefers to pace the Bachmann's bundle from the high septal RA (**Figure 10.21**). If the interval between the pacing artifact and the atrial activation timing in front of the roof line is significantly shorter than that of LAA pacing, Bachmann's bundle pacing is used to verify conduction block across the roof line. Another approach is to pace from the high posterior wall and posterolateral CS to examine the roof line and mitral isthmus line, respectively. Before mapping the linear lesion set, the author prefers to sample 2-3 points along the line *ipsilateral* to the pacing site to ensure that conduction from the pacing site to the ablation line is not significantly delayed. **Figure 10.22** illustrates 2 examples of pacing in close proximity to the ablation line to verify conduction block.

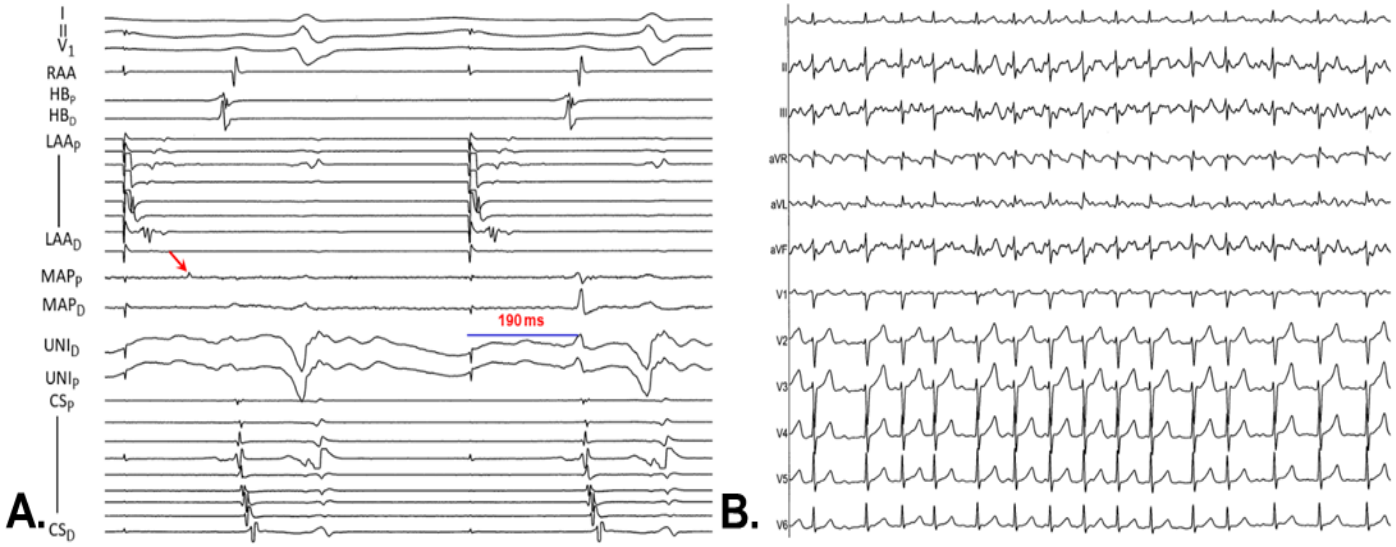


Figure 10.20. Slow conduction in the atrium affects judgment of conduction block in the same patient as Figure 10.19. A. During LAA pacing, the mapping catheter was positioned at the high posterior wall, immediately behind the roof line. The interval between the pacing stimulus and EGM was 190 ms, an encouraging finding. However, the potential in front of the line was also very late (red arrow), seriously affecting the accuracy of remapping during LAA pacing. **B.** A roof-dependent AT was soon induced. When LAA pacing is not suitable for remapping the roof line, pacing can be delivered from either the high posterior wall of the LA or from the Bachmann's bundle. As long as the atrial timing ipsilateral to the pacing site is early, this pacing site is legitimate.

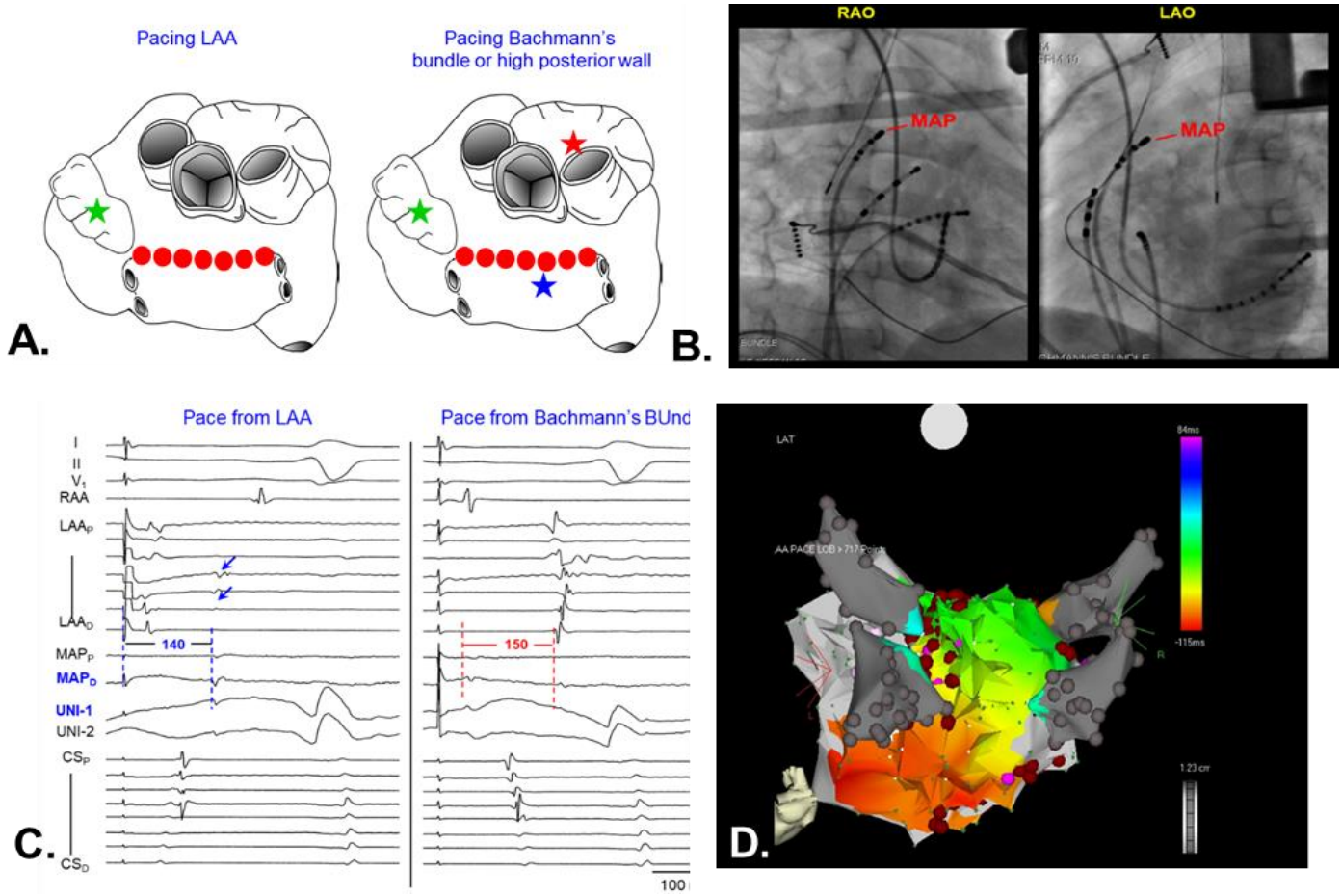


Figure 10.21. Pacing sites to verify conduction block across the ablation line. A. Left panel. LAA pacing (green star) is the first choice for both mitral isthmus line and roof line. **Right panel.** If slow conduction is present in front of the roof line due to extensive LAA ablation, pacing from Bachmann's bundle (red star) or at high posterior wall (blue star) may circumvent this problem. **B.** Radiographs showed that the mapping catheter was positioned at the Bachmann's bundle just below the RA-SVC junction, pointing leftward. **C.** In a patient with prior extensive AF ablation, pacing from the LAA was performed to examine conduction across the roof line. **Left panel.** A potential 140 ms after the pacing stimulus was identified, suggestive of conduction block across the roof line. However, the mapping catheter was positioned *in front of* the roof line. Also note that the LAA itself had severe conduction delay (blue arrows) due to prior ablation of the base of the LAA. Therefore, LAA pacing could not be used to verify conduction block across the roof line. **Right panel.** Pacing from the Bachmann's bundle similar to a site shown in (**B**), a wide double potential (150 ms) was evident, suggestive of conduction block. Note that the first potential was early. **D.** CARTO mapping showed low to high on the posterior wall, indicating conduction block across the roof line.

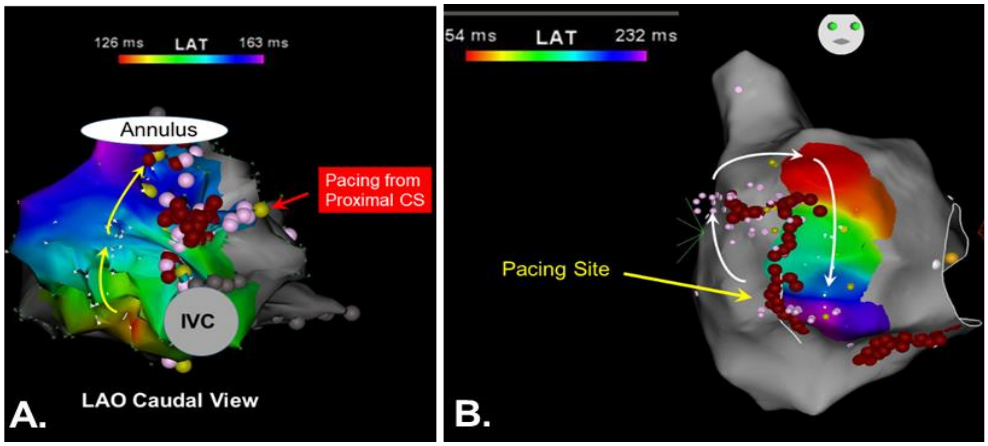


Figure 10.22. Pacing site to examine conduction block. A. For CTI line, pacing from CS ostium is the first choice. **B.** The same patient as shown in **Figure 10.13**. After extending the linear lesion set to the IVC and making a CTI line, pacing from inferolateral RA verified conduction block across the vertical ablation line.

Endocardial vs. epicardial (CS) conduction block across the mitral isthmus line

In the mitral isthmus, both endocardial conduction and epicardial conduction (e.g. CS or ligament of Marshall) play a critical role in the maintenance of mitral annular AT. Making a mitral isthmus line between the LPV and annulus often requires ablation within the CS to produce conduction block. A common mistake is failure to deliver enough LA endocardial ablation before moving to CS ablation, particularly the area near the LIPV where the leftward extension of the Bachmann's bundle may contribute to the increased thickness of the myocardium. After insufficient endocardial ablation and CS ablation, mitral annular AT remains inducible. The operator then re-ablates the LA endocardium and CS back and forth in hope of achieving conduction block. With progressively worsening of edema, conduction block becomes more and more difficult to accomplish. One of the reasons to infuse ethanol into the vein of Marshall is to eliminate the epicardial conduction of the thick myocardium in this region as well as to eliminate the epicardial conduction through the ligament of Marshal (LOM)l. LOM is an epicardial structure and can be localized by drawing an imaginary line between the LIPV-LAA ridge to the Vieussens valve. The top segment of the LOM courses along the epicardial surface of the LAA-LPV ridge. The author usually delivers higher power, long RF applications to the LAA-LPV ridge in hopes that ATs using LOM for conduction can be minimized. It is the author's opinion that effective *transmural* endocardial ablation probably can eliminate most of the ablation in the great cardiac vein because great cardiac vein ablation serves two purposes: (1) eliminate epicardial conduction of the mitral isthmus and (2) eliminate the LA-CS connection. The former is probably more important than the latter.

The author's practice is to ablate the LA endocardium with enough power/force/time during mitral annular AT. After AT is terminated, ablation continued during LAA pacing. After wide double potentials were identified along the *entire* ablation line, remapping below the line is performed. Endocardial activation should be inferior-to-superior below the line and the timing of the 2nd component of the double potential at all sites along the line should be similar. If it is, it indicates endocardial conduction block. Of note, mapping needs to include the area 3-4 cm below the isthmus line, particularly the area near the LIPV, to identify residual epicardial conduction (e.g. through the LOM). If CS conduction remains distal to proximal, RF applications are then delivered within the CS slightly superior to the endocardial ablation line. However, if catheter manipulation within the CS is not too challenging, mapping of CS first is preferred to identify the site of earliest conduction, the presumed site of connection between the LA endocardium and CS myocardium. With this approach, CS conduction block can usually be accomplished. Notably, ablation in the CS carries a risk of injuring the circumflex artery. While ablating within the CS, the author prefers to set the live screen to be 12-lead ECG at 25 mm/second sweep speed to monitor the ST-T change to detect myocardial ischemia. Finally, during LAA pacing, posterolateral LA is mapped to search for epicardial conduction through the LOM or leftward extension of the Bachmann's bundle. After undergoing several painful cases, the author would map the area above the mitral isthmus line during distal CS pacing (pacing directly below the ablation line) as well. Mapping should include the lateral anterior wall and LAA to ensure bidirectional block across the mitral isthmus line.

The most commonly used EP criterion to verify conduction block across the mitral isthmus is to demonstrate a CS-p to CS-d conduction pattern during LAA pacing. This criterion does not guarantee endocardial conduction block, though. **Figure 10.19** illustrates a series of examples in which prior extensive, non-transmural mitral isthmus ablations led to gaps buried in scars manifesting as far-field, rounded potentials at all the sites with early activation timing. CS conduction was proximal to distal all the time, indicating the presence of CS conduction block; however, mitral annular AT was independent of CS. **Figure 10.23** illustrates a mitral annular AT in the presence of CS conduction block. Note that the bracketed CS activation pattern during tachycardia did not support the diagnosis of mitral annular AT. For the aforementioned reasons, the author would remap above and below the mitral isthmus line after CS ablation to ensure that both endocardial and epicardial conduction block is accomplished.

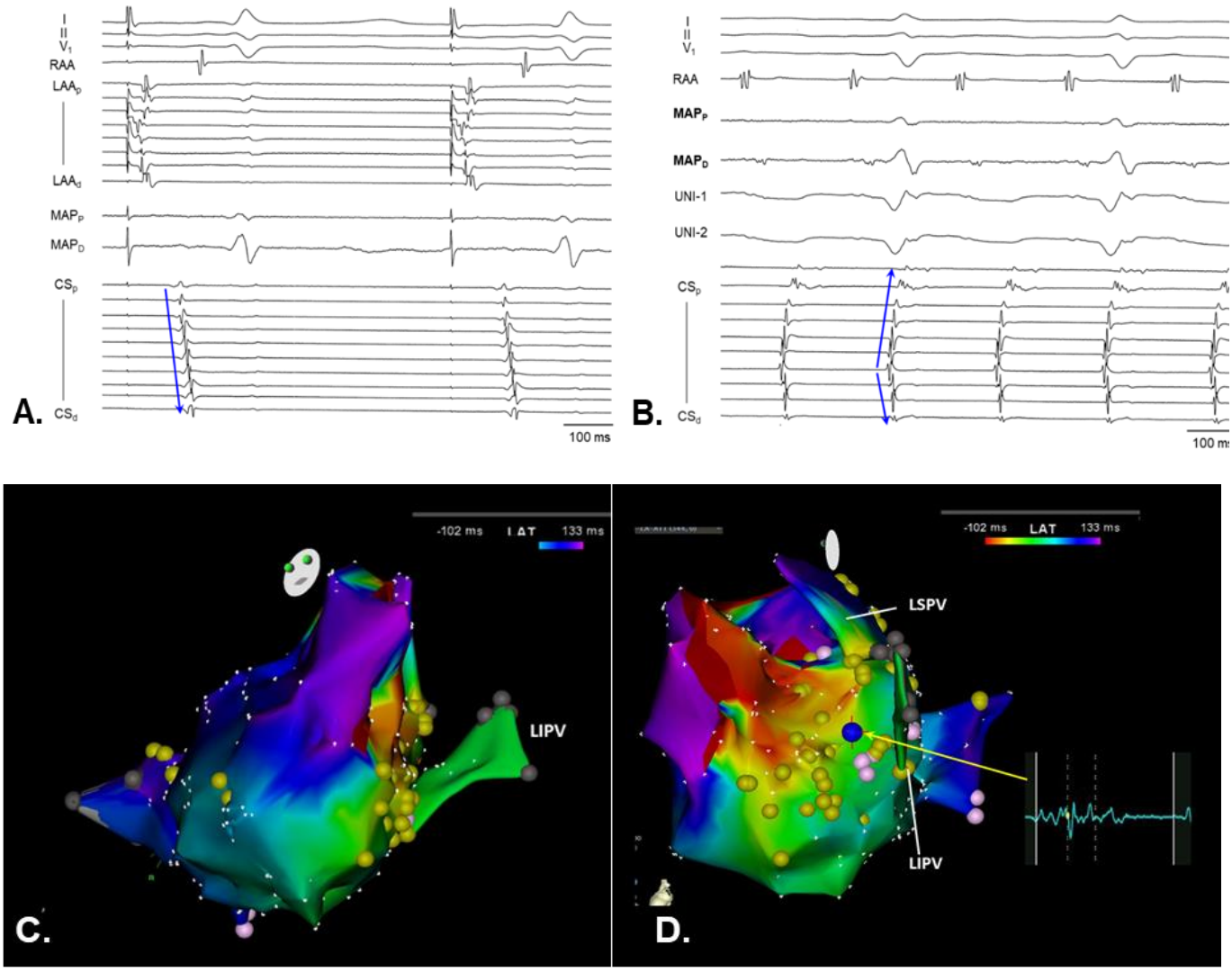


Figure 10.23. A 57 y/o male with one prior AF ablation (PVI, mitral isthmus line and roof line). A. LAA pacing showed that CS activation was proximal to distal (blue arrow), suggesting conduction block across the mitral isthmus line created in prior ablation. **B.** AT was easily induced. The CS activation pattern did not support the diagnosis of a mitral annular AT. **C**. CARTO map showed a clockwise mitral annular AT. **D.** A long fractionated potential (125 ms, inset) at one of the gaps along the mitral isthmus line. RF application there immediately terminated the tachycardia.

Mitral isthmus line is probably the most difficult linear ablation lesion set to make. The OU-EP group follows a simple rule: if one decides to make any linear lesion set, conduction block across the ablation line must be accomplished after ablation; otherwise this incomplete ablation line is a perfect setup for future reentrant arrhythmia. It is not uncommon for us to spend 2-3 hours to make a linear lesion set. If the CS catheter is difficult to be advanced to the mitral isthmus area, the author would obtain a CS angiogram to delineate the GCV anatomy and make sure the ablation can be advanced to the mitral isthmus if CS ablation is needed. If the CS anatomy prevents the ablation catheter to be advanced to the mitral isthmus area, one should seriously consider alternative approaches (e.g. an anterior mitral line) with the understanding that the Bachmann's bundle is difficult to ablate as well. If endocardial mitral isthmus ablation cannot achieve conduction block, not being able to ablate the CS may render this incomplete mitral isthmus line a very arrhythmogenic substrate. If the operator then makes an anterior mitral line, it carries a very high risk of isolating the LAA, which requires an LAA occlusion device to prevent the severe consequences of LAA thrombus. Similarly, if a paroxysmal AF patient has equivocal history of right atrial flutter but atrial flutter was not induced after AF ablation, the author would map the CTI to evaluate if an empirical CTI line should be

made. If CTI mapping shows high voltage (thick pectinate muscle), tall Eustachian ridge, deep pouch or other difficult anatomy, the author may avoid making an empirical CTI line because an incomplete line is very arrhythmogenic.

ATs after surgery for congenital heart diseases

A landmark paper published by Drs. Jackman and Nakagawa (*Circulation 2001 Feb 6;103(5):699-709*) described a cohort of patients with complex ATs after congenital heart disease surgeries. CARTO was still in its infancy when the study was conducted. Drs. Jackman and Nakagawa figured out how to maximize the benefit of that new mapping system. This is indeed the source of the discussions in this chapter. They proposed an idea of “no channel, no tachycardia”, emphasizing the importance of identifying and eliminating arrhythmogenic channels. The same principle was later expanded to ablations of complex ATs after AF ablation. A prerequisite of treating this type of AT is to minimize the noise in the EP laboratory. The potentials in many of the arrhythmogenic channels buried in scars were only 0.03-0.04 mV. In the presence of electrical noise, these channels may be completely overlooked.

Of note, the atria of patients with corrected congenital heart diseases can be very diseased and EGMs are diffusely small. Very small atrial potentials are often difficult to capture by pacing but fortunately easy to be eliminated by ablation. One cannot determine if a small potential represents live or dead tissue or if the small potential plays a role in the tachycardia based on whether that small potential can be captured by overdrive pacing or not. Dr. Jackman heavily relies on using the triggered sweeps as described in **Chapter 1** to map these small potentials because they will show up at the same timing in each cardiac cycle. EGMs recorded from dense scar often have multiple components; the amplitude as well as the sharpness of each component recorded on the distal unipolar EGM (UNI-1) may not help the operator choose the correct activation time. As already discussed, Dr. Jackman prefers to choose one component to time it based on the timing of the points surrounding it and re-assign the activation timing later when more points are acquired. Another important issue related to patients with congenital heart diseases is that many of them, particularly after the Fontan procedure, have focal ATs as well. Simply connecting the scars in sinus rhythm may not eliminate the clinical tachycardia. ATs in Fontan patients are notorious for being terminated by entrainment. Operators should always attempt activation mapping first.

Chapter 11:

Ablation of Ventricular Tachycardia

In the author's opinion, the most important contribution that Dr. Jackman made to VT ablation was to propose the concepts of (1) performing substrate mapping and targeting isolated late potentials to treat infarct scar-related VT and (2) targeting early pre-systolic Purkinje potentials to treat VT originating in the left posterior fascicle. He proposed the former concept in the late 1990s based on observations made by pioneers such as Drs. Andy Witt, Mark Josephson, JM deBakker and Benjamin Scherlag that abnormal local ventricular EGMs (e.g. isolated late potentials, fractionated potentials) represent critical elements in the VT reentrant circuit. Original research by de Bakker et al using a Langendorff perfusion setup elucidated the mechanisms and circuits for infarct scar related VT. Within the scar, multiple discrete presystolic or diastolic EGMs of low amplitude can be recorded. Histology studies revealed that separate bundles of surviving myocardium give rise to these low amplitude EGMs (**Figure 11.1A**). These surviving myocardial bundles can be intramural, sub-endocardial or sub-epicardial. Slow conduction velocity through these isolated tracts suggested that reentry may occur between these myocardial bundles. Dr. Jackman viewed *isolated* late potentials (**Figure 11.1B**) as the most important elements in the reentrant circuit and hypothesized that the VT reentrant circuit can be interrupted when most, if not all, of the isolated late potentials are eliminated. He presented promising results in many conferences in the early 2000s but somehow never bothered to publish the results. Thanks to Dr. Jackman's pioneering work, to date, substrate mapping and ablation are being performed to treat almost all types of VT in the presence of structural heart disease. Local abnormal ventricular activity (LAVA), which includes isolated late potentials, is now the preferred target in substrate-based VT ablation.

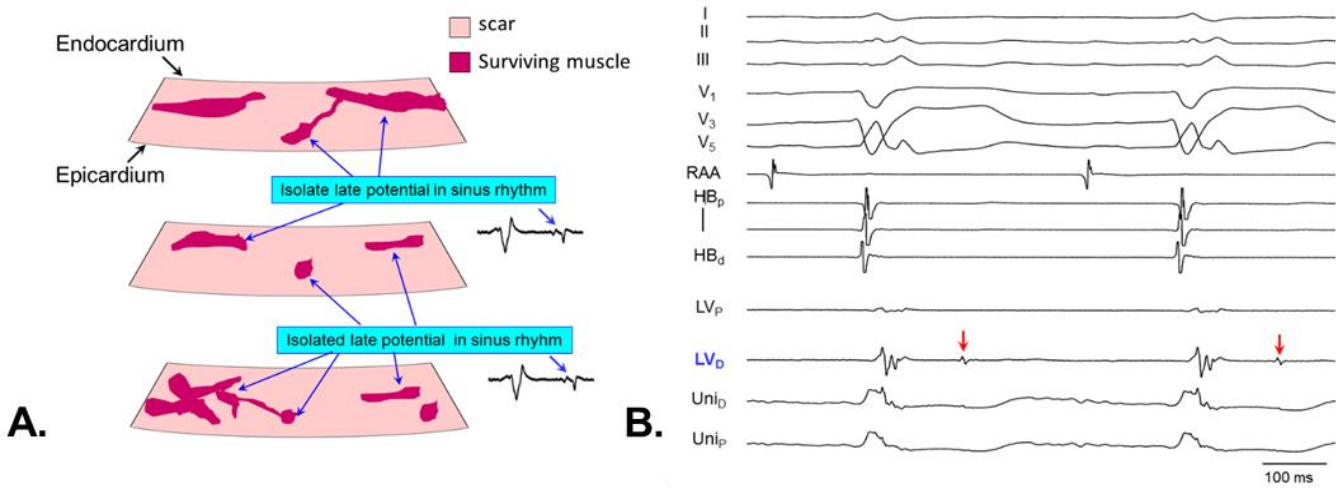


Figure 11.1. Surviving myocardial bundles generate low amplitude, presystolic or diastolic potentials. A. Three adjacent histologic slices illustrate that surviving myocardial bundles form a 3-D network. These myocardial bundles have been shown to generate local abnormal ventricular activities (LAVA). **B.** An isolated late potential (red arrows) separated from the local ventricular activation by an iso-electrical interval.

When ablation of fascicular VT originating in the left posterior fascicle was still in its infancy, the mechanism operating in fascicular VT had not yet been elucidated. Dr. Jackman pioneered an effective ablation strategy of targeting early pre-systolic Purkinje potentials in the territory of the left posterior fascicle (Circulation 1993; 88:2607-2617). Two decades after Dr. Jackman's pioneering work, reentry has been established to be the mechanism underlying fascicular VT. The earliest Purkinje potential that Dr. Jackman targeted is indeed adjacent to the site where the antegrade and retrograde limb of the reentrant circuit

connect. Targeting the earliest Purkinje potential, as Dr. Jackman originally proposed, still works for most left posterior fascicular VT.

VT in patients with structural heart disease

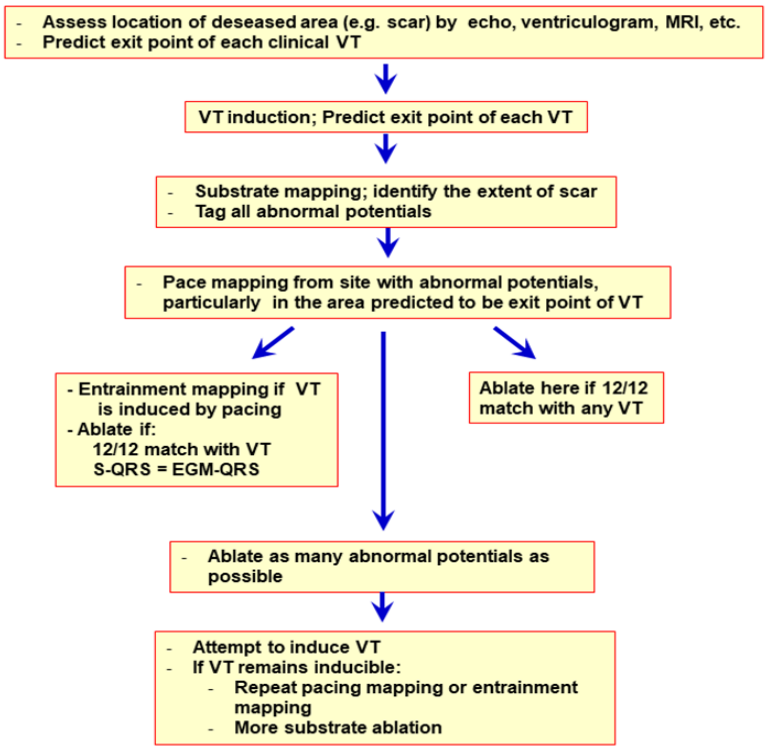


Figure 11.2. Workflow of VT ablation in patients with structural heart diseases

In the past two decades, many groups have proposed different mapping and ablation strategies to treat VT. Currently, abnormal ventricular EGMs caused by abnormal local ventricular conduction in various diseases such as myocardial infarction, ARVC and non-ischemic cardiomyopathy are collectively identified as LAVA. The idea of substrate mapping and targeting LAVA has been extended to ablation for nearly all types of VT in the presence of structural heart disease with myocardial bundles traversing the diseased area (e.g. scar or fibro-fatty tissue). This chapter focuses on Dr. Jackman's approach to treating VT in patients with structural heart disease. **Figure 11.2** is a summary of the work flow in the OU-EP laboratory for VT ablation in patients with structural heart disease.

In preparation for VT ablation

In the OU-EP laboratory, reviewing prior imaging studies (e.g. echocardiography, cardiac MRI) and 12-lead ECGs of all clinical VTs constitutes the most important pre-ablation assessment. Segments with regional wall motion abnormalities (e.g. hypokinesis, akinesis, dyskinesis and/or aneurysm) along with associated wall thickness are carefully evaluated in order to correlate the diseased area identified by imaging studies with the VT exit point identified by ECG. The exit point of each clinical VT is localized based on a series of publications from the University of Pennsylvania group over the past 4 decades. Localization of the exit point of each VT serves as the road map to guide pace mapping and entrainment mapping.

A high-quality echocardiogram provides an immense amount of information about the VT substrate. A mistake that we electrophysiologists often make is to rely on the echo report detailing the areas with wall motion abnormalities without personally reviewing the images. Wall motion abnormalities can be caused by scar or hibernating myocardium; the latter typically has normal or increased wall thickness and is unlikely to be the endocardial ablation target. The author does not allow EP fellows to scrub in until s/he has reviewed the imaging studies and identified abnormal substrate. If ischemic cardiomyopathy is defined as cardiomyopathy caused by prior myocardial infarction, a discrete, thin-walled scar caused by prior infarct should be visible by at least one imaging modality. In patients with severe coronary artery disease, hypokinesis/akinesis with *normal* wall thickness should alarm the operator to search for intra-mural or epicardial arrhythmogenic substrate, because many of these patients indeed have non-ischemic cardiomyopathy in the presence of coronary artery disease. Since the advent of primary PCI in late 1990s to treat STEMI, the substrate of scar-related VT has changed substantially. Large infarct scars with numerous late potentials have gradually been replaced by smaller scars with early diastolic or late systolic LAVA. With

non-transmural infarcts, much of the arrhythmogenic substrate is intramural and requires higher power, longer RF applications to eliminate the intramural substrate.

VT induction

Today, substrate mapping and ablation are gaining popularity; some electrophysiologists adopt substrate ablation as the sole approach to VT ablation. The low voltage area or LAVA sites are "homogenized" by RF applications without making efforts to induce VT. However, it is of paramount importance to induce VT, carefully examine its morphology and make an effort to record a His bundle (HB) potential to determine the relationship between VT and the HB potential. In patients with non-ischemic cardiomyopathy, the most common origins of VTs are the basal lateral/inferolateral LV and anterior septum. The former usually requires epicardial ablation; the latter endocardial ablation. If an outflow tract-like VT is reproducibly induced in a patient with non-ischemic cardiomyopathy, starting the procedure with endocardial mapping would be more fruitful. **Figure 11.3** illustrates an example of a patient with non-ischemic cardiomyopathy. The VT leading to multiple ICD shocks was a focal (or small reentrant) VT located 1 cm below the right coronary cusp. If VT had not been induced and activation mapping had not been attempted, substrate ablation alone probably would not eradicate this VT. It is known that up to 50% of the LAVA in septal intramural scars can be missed by endocardial substrate mapping. That is to say, without the guidance of the morphology of a 12-lead ECG in VT and/or entrainment mapping, the success rate of endocardial substrate mapping/ablation may be <50% in these cases.

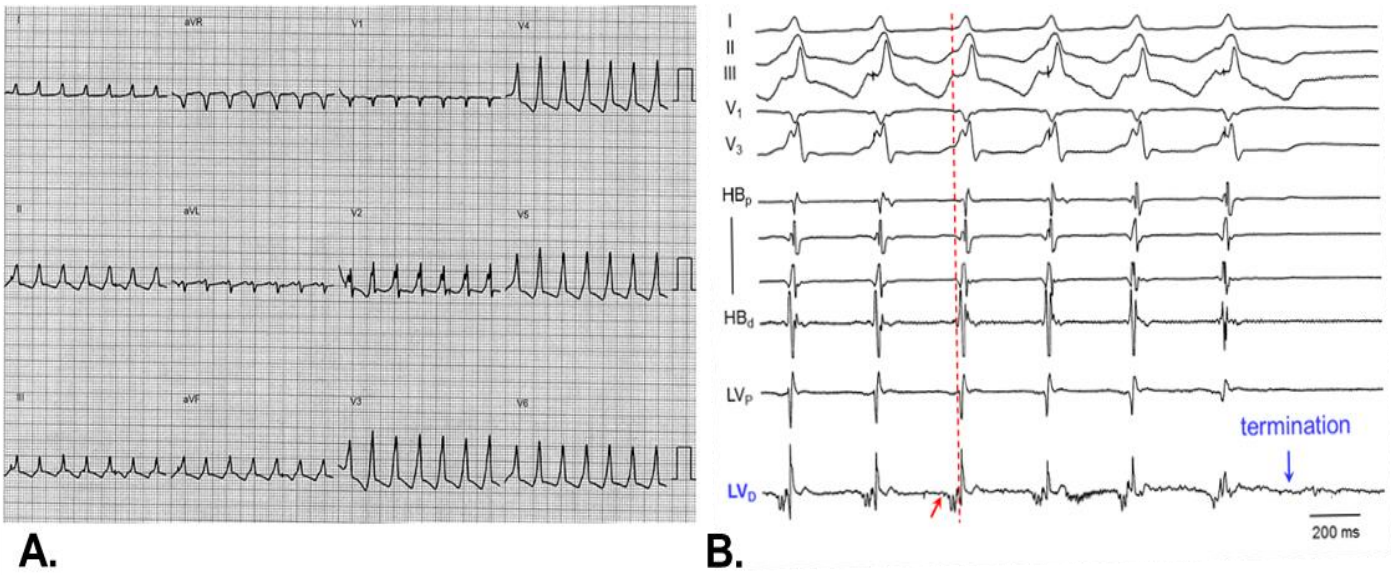


Figure 11.3. Importance of VT induction prior to substrate mapping. A. 12-lead ECG of VT induced in a patient with non-ischemic cardiomyopathy. In prior ablation, LV endocardial mapping found no low voltage area. No ablation was done. The original plan was to start with epicardial ablation. The morphology of this VT suggests that the site of origin (either focal or reentrant VT) is in high interventricular septum. LV endocardial ablation was therefore started. **B.** A pre-potential (red arrow) 40 ms earlier than the onset of the QRS complex was found in the high inter-ventricular septum, 1 cm below the right coronary cusp. Ablation there terminated VT in 2 seconds.

If the induced VT shows typical RBBB or LBBB morphology and the HV interval is fixed (1:1 relationship between the HB potential and VT), this observation should prompt the operator to search for a His-Purkinje related VT (e.g. fascicular VT, bundle-branch reentrant VT), not a VT of myocardial origin. **Figure 11.4** illustrates an example of a patient with non-ischemic cardiomyopathy. Echocardiography showed diffuse, severe hypokinesis. By odds, the author was inclined to attempt epicardial ablation only since the patient was very ill and obese (170 kg). Induction of bundle-branch reentrant VT turned a difficult procedure into an easy, successful one by ablating the right bundle branch. Bundle-branch reentrant VT will be discussed later in this chapter.

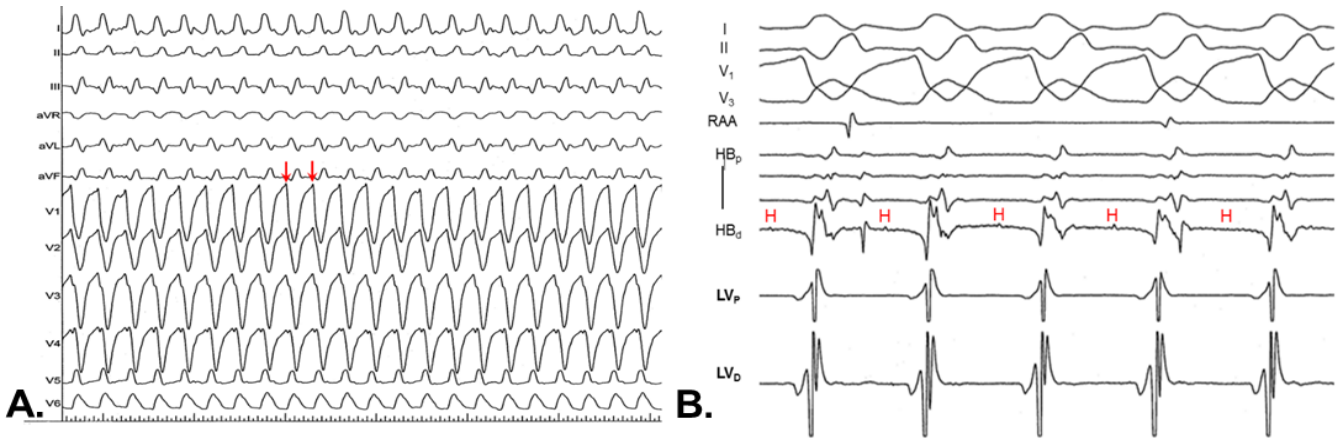


Figure 11.4. Bundle-branch reentrant VT in a very ill, obese patient with non-ischemic cardiomyopathy. A. VT showed typical LBBB morphology with a small, sharp r wave in V1, suggestive of Purkinje-related VT. **B.** During VT, the HV interval was fixed at 75 ms. VT was terminated by ablating the RBB.

The OU-EP group attempts to induce VT as soon as diagnostic catheters are in place. The burst pacing or programmed stimulation protocols are not too aggressive to begin with in order to avoid inducing VF. The author usually stops induction if 3 different VTs have been induced because pace mapping later and catheter manipulation often induce more VTs. The morphologies of these VTs are carefully examined to predict their exit points. During substrate mapping, if the mapping catheter is in the area of a predicted VT exit point, pace mapping will be performed there. If pace-mapping produces a good match with VT, concentrated RF applications will be delivered to this area. It is important to note that the results of pace mapping tend to have a high positive predictive value, but a low negative predictive value for identifying locations within the VT circuit. One may think of the 3-D network formed by the surviving muscle bundles in a scar as a maze. In VT, the wave front only exits through one specific point (**Figure 11,5, 11.6**). During pacing in sinus rhythm, there may be many other possible exit points, creating different QRS complexes. For this reason, Dr. Jackman's original hypothesis was to ablate all late potentials to eradicate all combinations and permutation of the potential reentrant circuits.

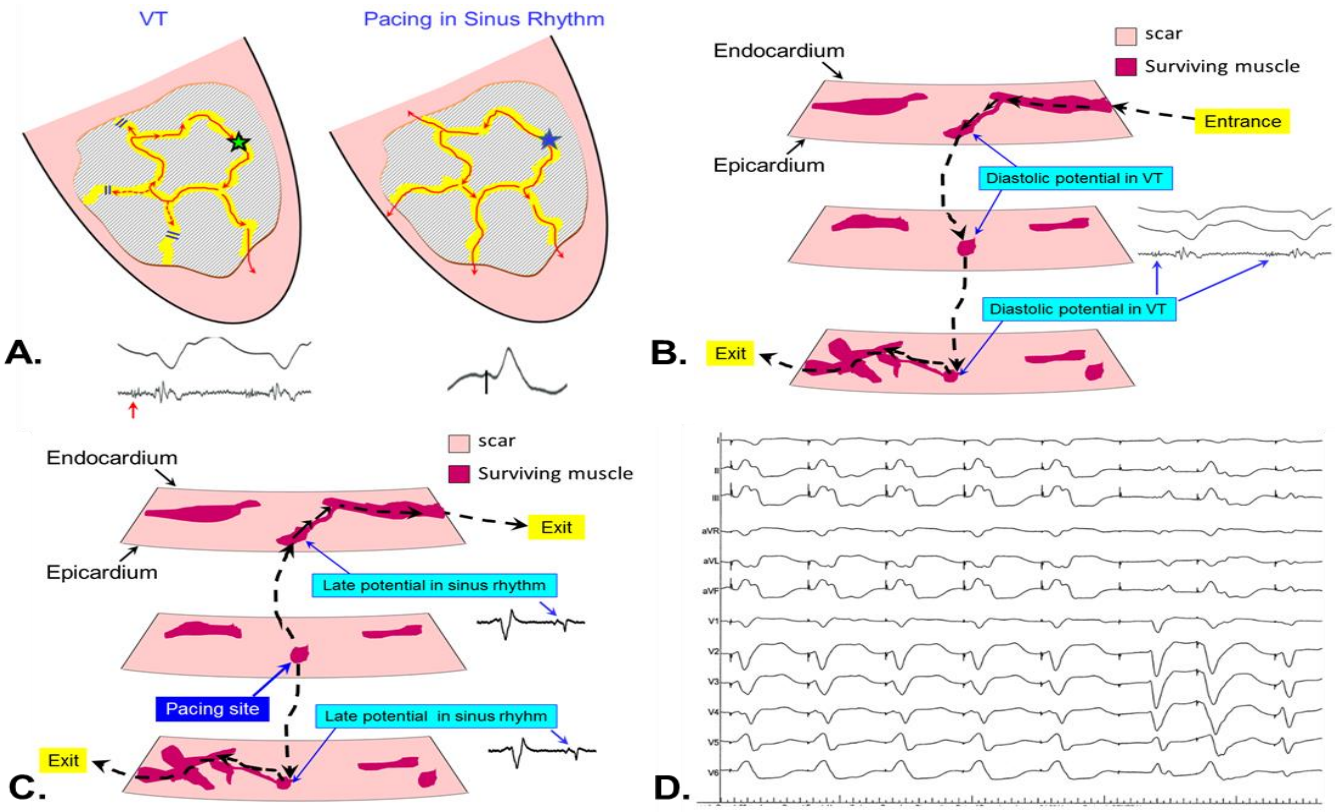


Figure 11.5. In sinus rhythm, pacing from sites within the protected VT isthmus may produce a completely different QRS morphology as compared with the VT morphology. A. Left panel. In VT, a site in the protected isthmus (green star) recorded a pre-systolic (or diastolic) potential (red arrow). The VT wave front encounters refractory period (double bars) of all possible exits but one, which becomes the VT exit point and produces the VT morphology. **Right panel. Pacing the same site (blue star) in sinus rhythm**. Paced wave front can exit the scar through multiple sites, producing different QRS morphologies. Similar concepts are explained in a 3-D illustration for VT (**B**) and pacing (**C**). **D.** In a patient with a large inferior wall infarct scar, pacing from inferior-apical LV produced a QRS complex suggestive of a basal-superior exit.

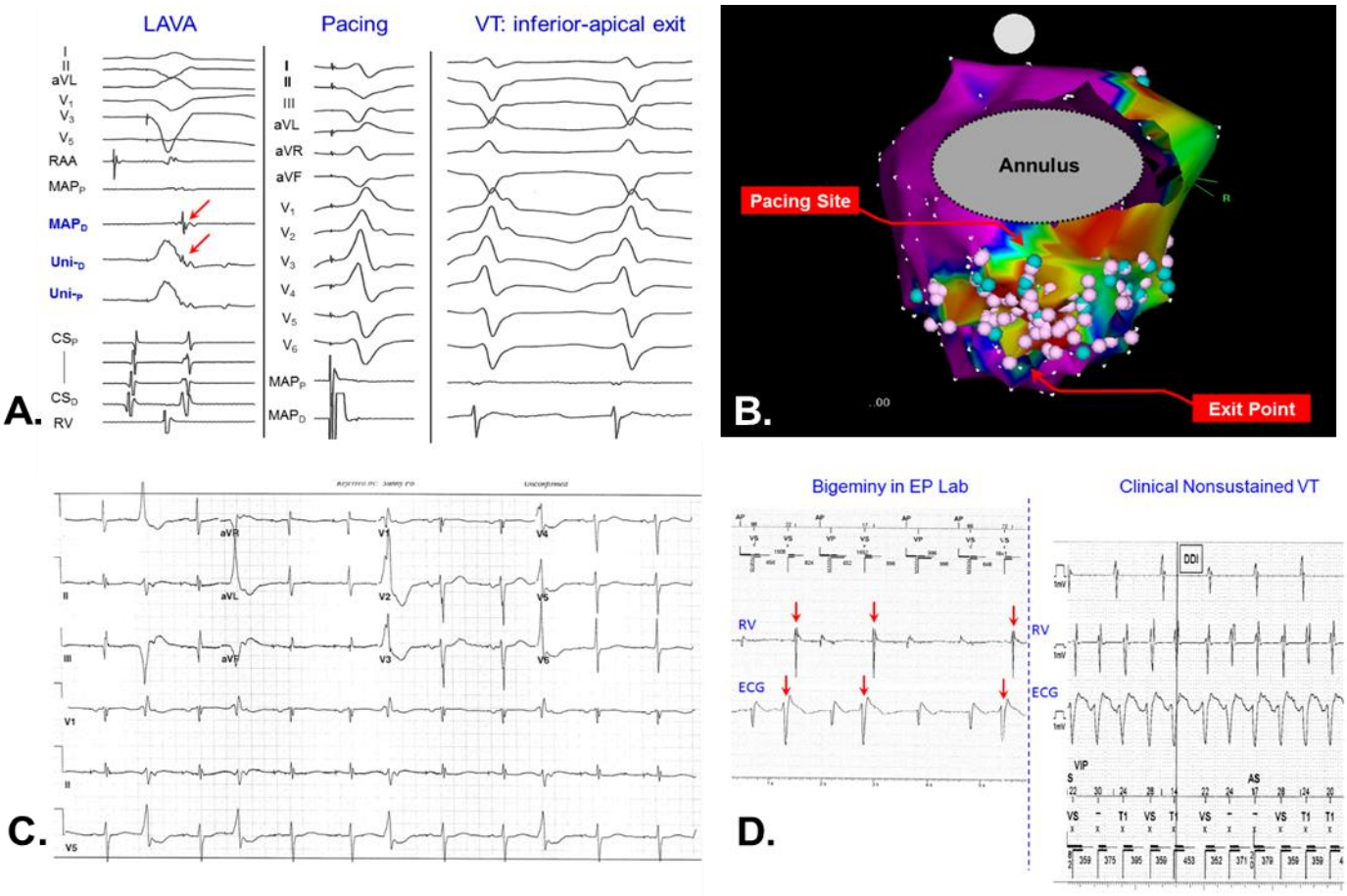


Figure 11.6. A. In a patient with a large infarct scar, despite the inferior-basal location of the pacing site, it produced the QRS morphology predicted to be an inferior-apical exit, nearly identical to VT. This finding suggests the presence of a large network of surviving myocardial bundles spanning from the base to the apex of the LV. Red arrows indicate LAVA. **B.** Pacing site and predicted exit point displayed on CARTO. **C-D.** In another patient with a small inferior-basal scar, the clinical VT was frequent, nonsustained VT. **C.** Morphology of frequent PVCs in the EP laboratory. **D.** When bigeminy PVCs were recorded in the EP laboratory, the morphology of their near-field and far-field EGMs recorded by the ICD was very similar to that of the nonsustained VT captured by ICD. It was presumed that nonsustained VT might be caused by this PVCs. Ablation of this PVC was performed before substrate ablation.

It is important for operators to know how ECG patches are positioned in his/her EP laboratory. In the presence of defibrillator patches, mapping system patches as well as preparation for epicardial access, the ECG patch position is often different from that when the clinical VT was recorded. Deviations from standard ECG recordings need to be factored in when one attempts to correlate induced VT with clinical VT or attempts to determine the exit point of the induced VT. EGMs and far-field ECGs recorded by an ICD can provide helpful information to correlate induced VT with clinical VT. In patients with frequent nonsustained VT, it is particularly helpful to compare the EGM and far-field ECG captured by the ICD with PVCs or nonsustained VT recorded in the EP laboratory because in some patients with structural heart disease, the clinical VT is a focal VT from a site that also produces frequent PVCs. **Figure 11.6C-D** illustrates a patient with a small inferior-basal infarct scar and frequent nonsustained VT. He presented to the EP laboratory with ventricular bigeminy. The morphology of PVCs was very similar to the clinical VT captured by his ICD. The ablation strategy was to target the monomorphic PVCs first, followed by substrate mapping and ablation.

Activation mapping

If the patient tolerates VT well, electro-anatomic activation mapping can help define the VT reentrant circuit. As part of the reentrant circuit may be mid-myocardial or epicardial, a complete reentrant circuit often cannot be obtained; critical elements in the reentrant circuit have to be identified by entrainment mapping. With advances in multi-electrode mapping, activation mapping has become more feasible for hemodynamically tolerable VTs. In poorly tolerated VTs, activation mapping is not possible without LV hemodynamic support devices such as Impella, TandemHeart or ECMO. For unmappable VTs, a substrate based approach in sinus rhythm or ventricular pacing is widely accepted as an alternative approach.

Substrate mapping

Substrate mapping searches for arrhythmogenic substrate in sinus rhythm or ventricular pacing. The EGM amplitude (voltage) is the most useful parameter to define diseased myocardium. In the vast majority of healthy left ventricular myocardium, bipolar voltage larger than 1.5 mV is considered normal. Many EP laboratories define a low-voltage zone as an area with a bipolar voltage <0.5 mV. It is of paramount importance to emphasize that a zone with very low-voltage (e.g. <0.1 mV) does not equal a dense scar where only fibrotic tissue exists. Many of the arrhythmogenic myocardial bundles generate voltages in the range of 0.03-0.05 mV. If the noise level of the EP lab is 0.06 mV, many arrhythmogenic myocardial bundles will be missed. This problem underscores the importance of reducing the noise in the EP laboratory as discussed in **Chapter 2**. All of the LAVA in these areas are tagged on the electro-anatomic map and used to guide ablation. During this process, care must also be taken to ensure adequate electrode-tissue contact to avoid an underestimate of voltage. The author does not tag a site as “scar” unless there is no EGM recorded with contact force>10 grams.

LAVA within the scar are often viewed as the electrophysiological signatures of viable myocardial bundles and important elements in the VT reentrant circuit. They are typically low voltage signals (e.g. 0.03-0.30 mV). In comparison to the term “late potential” or “diastolic potential”, LAVA is more inclusive, and includes abnormal potentials in the systolic phase as well (**Figure 11.7**). These abnormal EGMs, including multi-component EGMs, isolated diastolic potentials and fractionated EGMs, are usually abnormal potentials occurring near or after the end of the QRS complex. However, alternation in the ventricular wave front entering the scar can have a great impact on the morphology and timing of LAVA. Some LAVA appear within the QRS complex and are obscured by the local ventricular EGM (**Figure 11.7B**). This is similar to the oblique course of an accessory pathway in which an AP potential can be obscured or exposed depending on how the wave front engages the AP. Therefore, the distribution of LAVA within a given scar may be different between ventricular-sensed rhythm and ventricular-paced rhythm. In patients with a large infarct scar, if one only found a small number of LAVA, one should consider changing ventricular conduction by pacing from a different site or delivering single ventricular extra-stimuli to expose LAVA.

In addition to the direction of wave front propagation, the orientation between the electrode and tissue as well as the size of the recording electrode can affect the morphology and amplitude of LAVA. As discussed in **Chapter 2**, bipolar EGMs look different between perpendicular and parallel orientation. Compared to closely-spaced small electrode catheters (e.g. PentaRay), bipolar EGMs recorded by an ablation catheter (e.g. 3.5-2-2 mm spacing) tend to be more rounded (far-field signals). If substrate mapping is conducted using a PentaRay catheter, LAVA may look sharper compared to the EGMs recorded by an ablation catheter at the same site. This difference is caused by the smaller, closely spaced electrodes of the PentaRay catheter as well as parallel orientation between the bipolar electrodes and the myocardium. Operators may find that an ablation catheter does not record the abnormal EGMs that the PentaRay catheter recorded.

Although LAVA within the scar have been shown to be critical elements in the VT reentrant circuits, they alone have poor specificity for identifying the arrhythmogenic channels. Most of the LAVA identified within a scar are actually not part of the critical isthmus of VT. Two characteristics can improve the specificity of these findings. First, pace-mapping from LAVA sites (particularly late potentials) that produces a long stimulus-QRS interval with 12/12 match to the VT morphology improves specificity, because the pacing site

is probably located in a protected isthmus where slow conduction leads to a long stimulus-QRS interval. Second, a long, isolated late potential improves specificity. In the original study conducted by the OU-EP group on patients with ischemic cardiomyopathy, there was almost no VT recurrence within a year after ablation if the low voltage area (<0.5 mV) was larger than 30 cm^2 and the latest diastolic potential was >230 ms later than the QRS complex. In essence, if plentiful *late* potentials exist in a large scar, the outcome of substrate ablation is excellent. For this reason, if a late potential is recorded, the OU-EP group would conduct pace mapping at that site. It is not uncommon to observe a good match to induced VT from a pacing site distant from the predicted exit point of VT (**Figure 11.6**) due to the presence of a large network of surviving myocardial bundles.

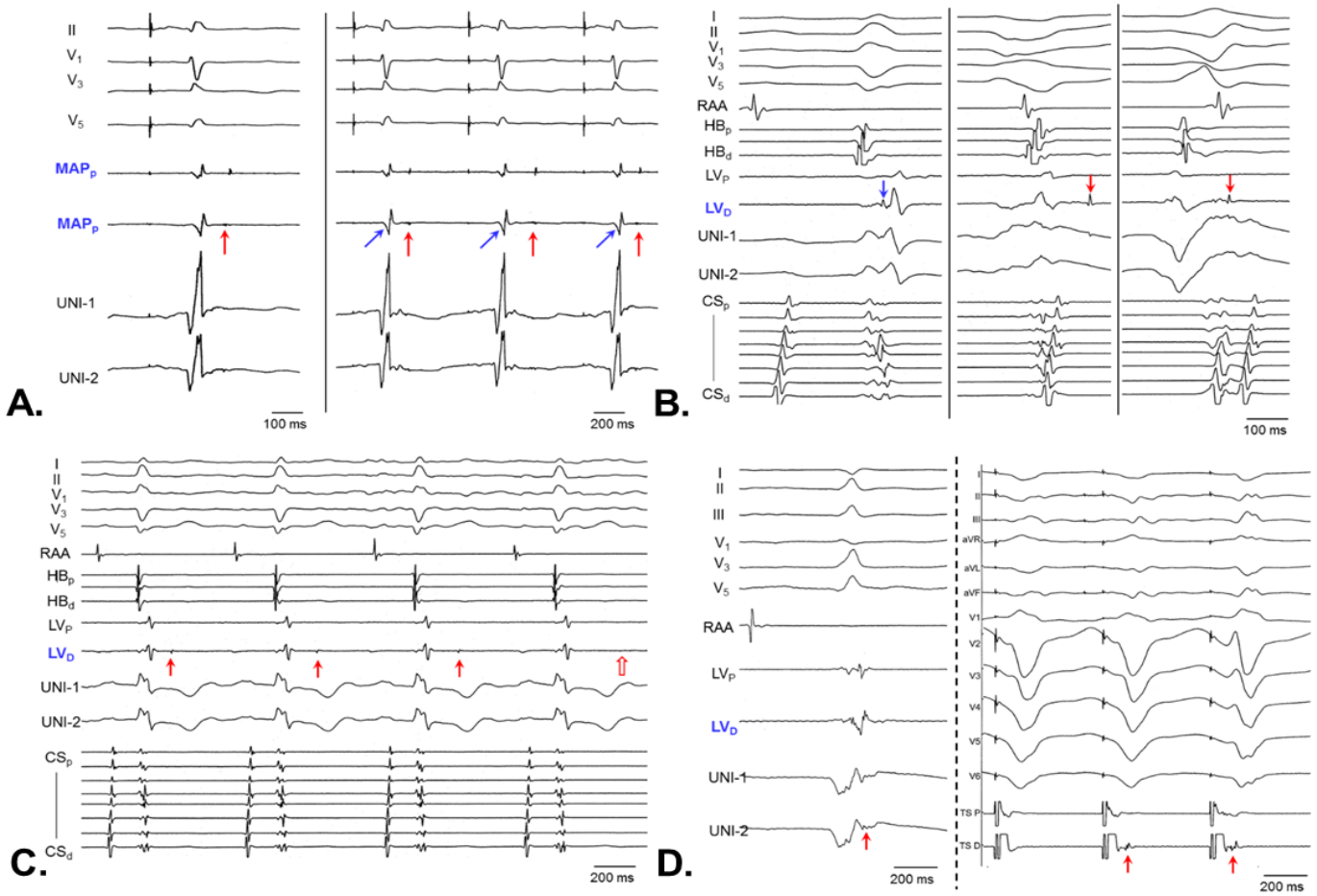


Figure 11.7. Various EP characteristics of LAVA. A. An isolated late potential with slow conduction property (red arrow). Note different interval between the local ventricular activation (blue arrow) and the late potential. **B. Left panel.** In another patient with ischemic cardiomyopathy, LAVA (blue arrow) within the local ventricular activation was noted. **Middle and right panel**. PVCs, altering the directions of wave front propagation into the infarct scar, made this LAVA more visible (red arrow). **C.** Wenckebach conduction of an isolate late potential (red arrows). **D.** In another patient with ischemic cardiomyopathy, pacing from a LAVA site produced different QRS morphologies, depending on the current output (**right panel**). At higher output, LAVA (red arrow) was captured (first beat, right panel). The morphology of the QRS complex changed as pacing output was decreased and part of the LAVA was no longer captured by pacing.

Some LAVA may appear not to participate in a given VT circuit but may form critical elements in the reentrant circuit of another VT. For this reason, Dr. Jackman's original proposal was to carefully tag all the late potentials to serve as ablation targets. However, ablation does not start until the network of isolated late potentials has been mapped out. Premature ablation, unless necessitated by deterioration of the patient's condition, may produce conduction block to the downstream surviving myocardial bundles, leading to an underestimate of the potential VT circuits and possibly a less favorable ablation outcome.

Pace mapping

Pace mapping in sinus rhythm provides critical information about the proximity between the pacing site and the VT exit point. A longer stimulus-QRS interval (e.g. >40-50 ms) indicates that the pacing site is in a slow conduction zone, but this is a very nonspecific finding as any bystander myocardial bundle may have such characteristics as well. Scar borders often house the exit points of multiple VTs and require more detailed pace mapping. Pace mapping has three essential elements: capture, paced QRS morphology and stimulus-QRS interval (indication of abnormal or slow conduction). Ideally, the lowest current that captures tissue should be used in order to minimize the amount of captured myocardium. Prior reports identified unexcitable scar as tissue where pacing output >10mA at a 2-ms pulse width failed to capture. However, this definition needs to be interpreted with caution because some LAVA located in a dense scar cannot be captured at maximal current despite being a critical element in the reentrant circuit.

In pace mapping, a 12/12 match to a clinical VT usually indicates that the pacing site is in proximity to the exit point of the VT reentrant circuit. However, a discrepancy between the paced QRS morphology and clinical VT does not exclude the possibility that the pace mapping site is in the reentrant circuit because pacing at a given site in the network of surviving muscle bundles can produce multiple morphologies, depending on the size of tissue captured, regions of conduction block and multiple possible exits (**Figure 11.5A**). It is not uncommon to observe that the paced QRS morphology from the successful ablation site failed to match the VT morphology but entrainment mapping during VT produced concealed fusion. It is worthwhile to induce VT to attempt entrainment mapping to determine if the LAVA recorded in an area expected to be the VT exit point is indeed in the protected isthmus of the reentrant circuit.

Entrainment mapping

In a reentrant circuit, terms such as “presystolic” and “late diastolic” are meaningless because the activation wave front propagates in circular motion. EGM timing alone in a given VT cycle length is a poor indicator of ablation target selection, as a bystander pathway (e.g. blind alley) may record presystolic or diastolic potentials as well. Entrainment mapping helps determine if the EGM of interest is located in the protected isthmus of a reentrant circuit. The VT reentrant circuit has several components, including entrance, protected isthmus (common pathway), exit, outer loop, inner loop and bystander sites (**Figure 11.8A**). A protected isthmus is the most vulnerable component of the VT reentrant circuit and is usually formed by viable myocardial bundles with slow conduction properties. It is protected by a conduction barrier such as scar that allows the VT wave front to leave the isthmus only through the exit point. If a protected isthmus is found, there is a very high likelihood that ablation there will terminate VT. Entrainment mapping is a form of activation mapping. Instead of delineating the entire VT reentrant circuit like one would pursue to map an atrial tachycardia, entrainment mapping is used to search for the most critical element (protected isthmus) in the reentrant circuit. It is important to point out that there can be a great discrepancy between the QRS morphology of pacing in sinus rhythm and pacing in VT; it is not uncommon that the site of the critical isthmus is a site where pace mapping showed a poor match with the VT (**Figure 11.8B**). Entrainment mapping is the best technique for ablation target selection. Dr. William Stevenson wrote several wonderful review articles on this subject. A recent review was published in J Cardiovasc Electrophysiol *(2016 Dec;27(12):1437-1447*).

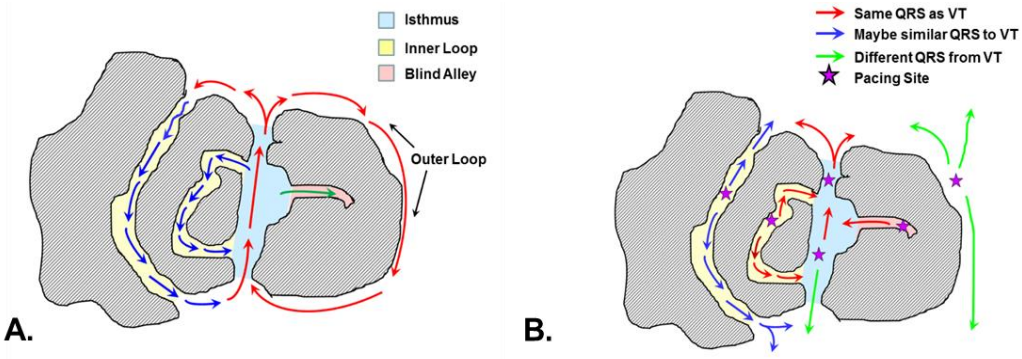


Figure 11.8. Schematic representation of various components of a reentrant circuit in scar-related VT. A. Entrainment delivered from the isthmus (common pathway) produces concealed fusion. Pacing from sites in a blind alley or an inner loop that also connects to the isthmus can produce concealed fusion as well. However, in these bystander sites, the stim-QRS interval is significantly longer because the paced wave front has to travel additional length in the blind alley or inner loop to enter the isthmus. **B.** The QRS morphology of pacing during sinus rhythm depends on how the wave front exits the scar. Despite pacing in the middle of the isthmus, it can produce two very different QRS morphologies.

To assess any entrainment response, Dr. Jackman follows a 3-step approach:

1. Make sure the last 4 beats are captured. The interval between the EGM of interest measured on consecutive beats should follow the pacing CL. For any tachycardia that Dr. Jackman attempts to entrain, he carefully examines all intracardiac EGMs and the ECG to decide which EGM or ECG lead he will be watching to determine if tachycardia has been entrained. As soon as the tachycardia is entrained by 4 paced beats, he stops pacing to avoid termination of the tachycardia. A common mistake that operators can make is to deliver overdrive pacing much longer than needed, resulting in termination of the tachycardia.

2. Compare the activation sequence (for SVT or atrial tachycardia) or QRS morphology (for VT) between tachycardia and paced beats.

3. Measure the post-pacing interval (PPI).

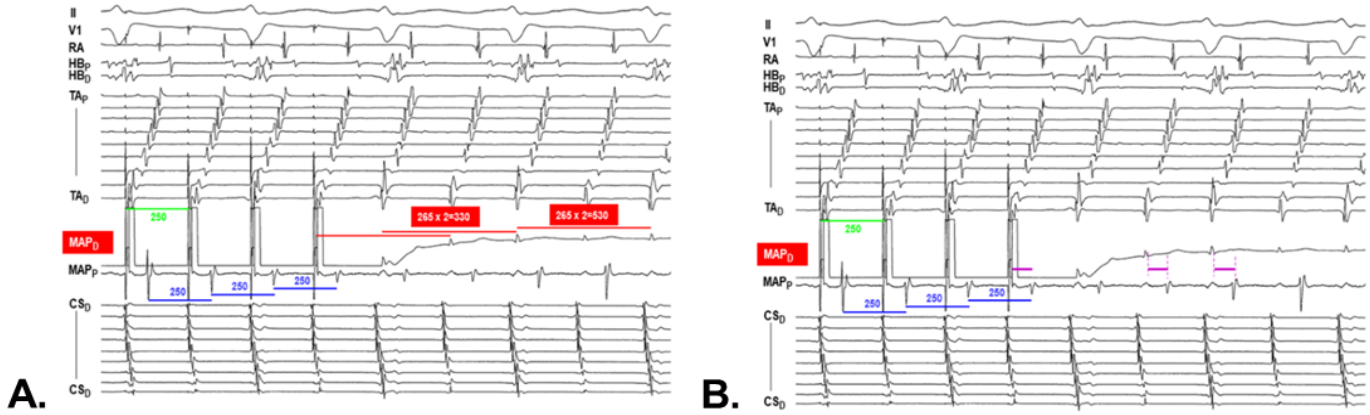


Figure 11.9. Two techniques to measure PPI if local EGM is obscured by amplifier saturation. This tachycardia was a clockwise right atrial flutter (AFL CL 265 ms; pacing CL 250 ms). **A**. Measure multiple tachycardia cycle length (red lines; 2x cycle length in this case) and march the caliper backwards to find the timing of the EGM of interest. PPI (265 ms) equals the flutter cycle length. **B**. Measure the relative timing (short purple lines) between the proximal and distal pair of the ablation catheter to find the timing of the EGM of interest on the distal electrode pair (MAP_D).

Sometimes, the EGM of interest is obscured by the pacing artifact (amplifier saturation), precluding accurate measurement of the PPI. Dr. Jackman uses two methods to measure the EGM of interest in this situation: (1) use the “march back” or “n+1” technique in which the width of a caliper is set to be multiples of the tachycardia CL and marched backwards to where the EGM of interest is supposed to be (**Figure 11.9A**) and (2) measure the relative EGM timing between the proximal and distal electrode pair of the ablation catheter as the reference interval (**Figure 11.9B**). Importantly, the second technique requires a stable relationship between the proximal and distal pair of the ablation catheter. A short PPI (<30ms) suggests that the pacing catheter is in close proximity to the VT circuit. It is important to note that interpretation of a PPI is based on an assumption that overdrive pacing does not slow the conduction time in the reentrant circuit. If the patient takes antiarrhythmic drugs (e.g. amiodarone) that slow conduction, a longer PPI (e.g. 50 ms) does not exclude the pacing site’s close proximity to the reentrant circuit. In general, the shorter the pacing cycle length, the higher the likelihood of causing a falsely prolonged PPI due to the paced wave front encroaching upon the excitable gap of the reentrant circuit (increasing response; **Figure 10.16A**). The author prefers to set the pacing CL 7% faster than the tachycardia CL to avoid tachycardia termination as well as avoiding false prolongation of the PPI (increasing response).

Figure 11.10 to 11.13 demonstrate several examples of entrainment mapping and related issues. With a 3.5-mm electrode catheter, the distal bipolar electrodes may record several far-field potentials. In essence, if a diastolic or presystolic potential is located in a protected isthmus, overdrive pacing from that site during VT should be able to entrain the VT and produce the same QRS morphology as the VT morphology (concealed fusion) because the wave front cannot leave the isthmus except through its exit point. In addition to QRS morphology and PPI, entrainment mapping requires comparison of the stimulus to QRS (stim-QRS) interval during overdrive pacing and EGM to QRS (EGM-QRS) interval during VT. If one uses the onset of

the QRS complex as the moment when the VT wave front exits the protected isthmus, the interval of stim-QRS during overdrive pacing should be very similar to the interval of EGM-QRS during VT if the catheter is located within the VT circuit. The stim-QRS interval is often slightly longer than the EGM-QRS interval because pacing faster than the tachycardia in the isthmus (a slow conduction zone) can prolong conduction to the exit point by 20-30 ms. Although entrainment with concealed fusion may occur by pacing inside the protected isthmus, pacing from bystander sites such as blind alleys can also produce concealed fusion (albeit with a longer PPI) (**Figure 11.8**). Ablation at these bystander sites has a low likelihood of terminating the VT. If entrainment produces manifest fusion, it indicates that the pacing site is not in the protected isthmus. Ablation there is not likely to terminate the VT.

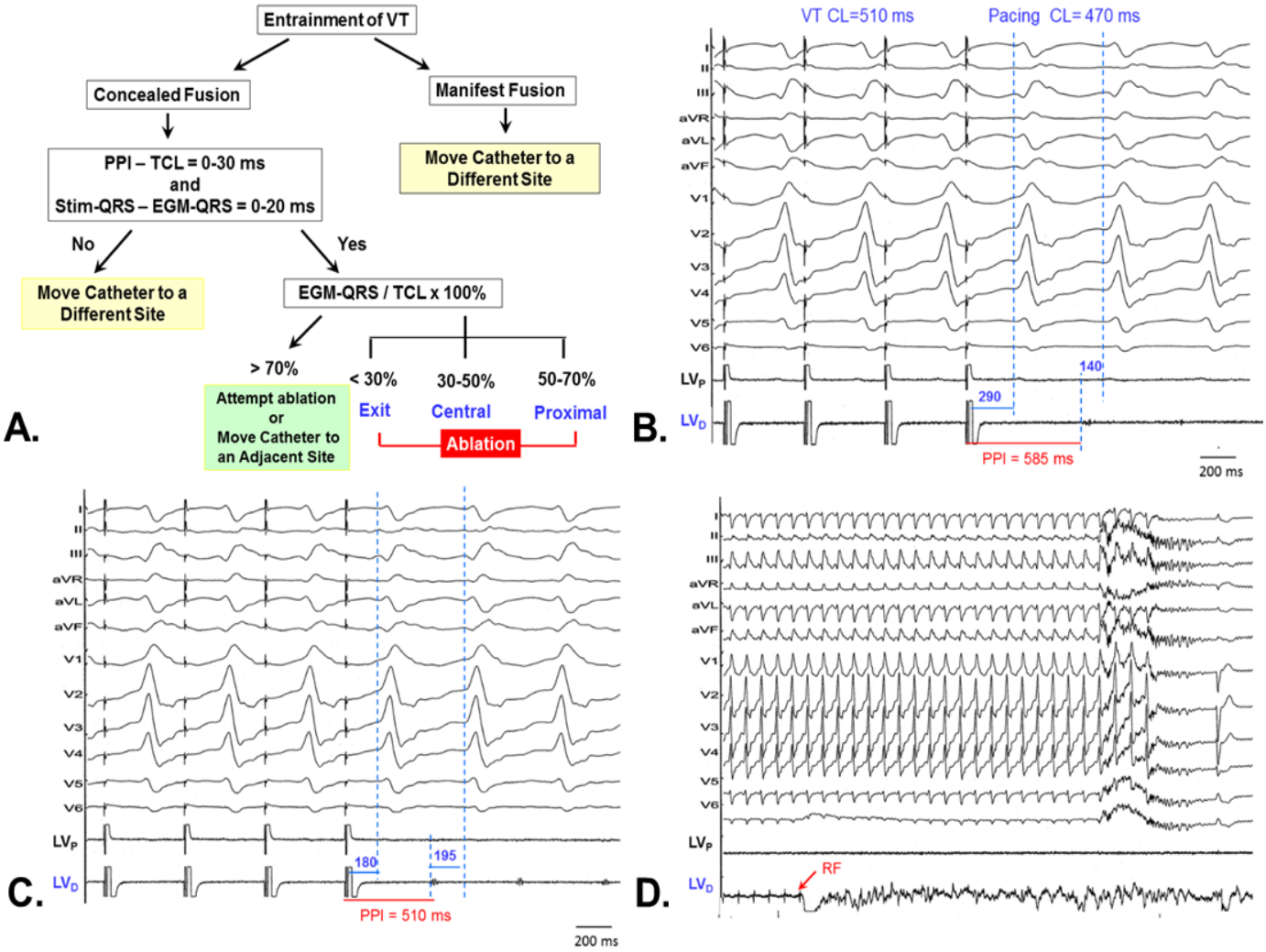


Figure 11.10. Example of entrainment mapping. A. Algorithm of entrainment mapping in the OU-EP laboratory. This algorithm is based on Dr. William Stevenson's publications. If manifest fusion is seen, the pacing site is either in an outer loop or a remote site. Ablation would not affect the VT. If the stim-QRS/TCL ratio is >0.7, the author still attempts to ablate this site because if this is site is in an inner loop, it may be close to the proximal segment of the protected isthmus and worthwhile to ablate there. **B.** In a patient with an inferior wall infarct scar, entrainment mapping there produced concealed fusion. There was a large discrepancy between the stim-QRS interval (290 ms) and EGM-QRS interval (140 ms). The PPI was 75 ms longer than the VT cycle length. This site was most likely in an inner loop or blind alley. **C.** At another site, entrainment mapping produced concealed fusion and a short PPI. The stim-QRS and EGM-QRS interval were similar**. D.** Ablation there terminated VT.

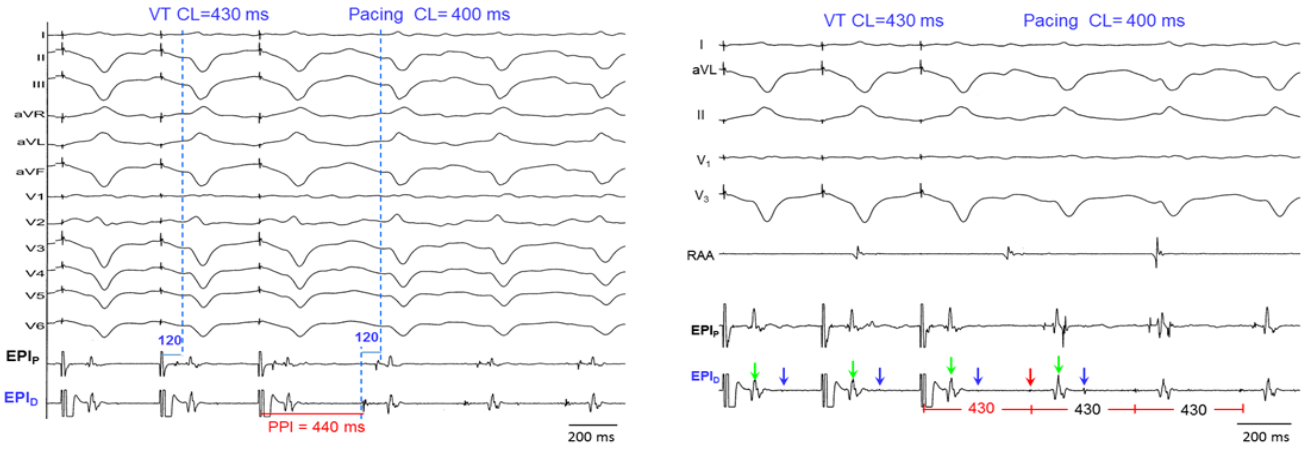


Figure 11.11. Example of epicardial entrainment mapping in a patient with non-ischemic cardiomyopathy. A. Entrainment mapping delivered there produced concealed fusion and a short PPI (440 ms), 10 ms longer than the tachycardia CL. Stim-QRS interval and EGM-QRS interval were identical (120 ms, 27% VT cycle length), suggesting that the entrainment site was in the vicinity of the exit point of the protected isthmus. **B.** At another site, 3 potentials were recorded. Entrainment captured the first potential (red arrow). The other two potentials (blue and green arrows) were not captured by entrainment, indicative of being far-field potentials. Note that the largest potential (green arrows) was a far-field potential.

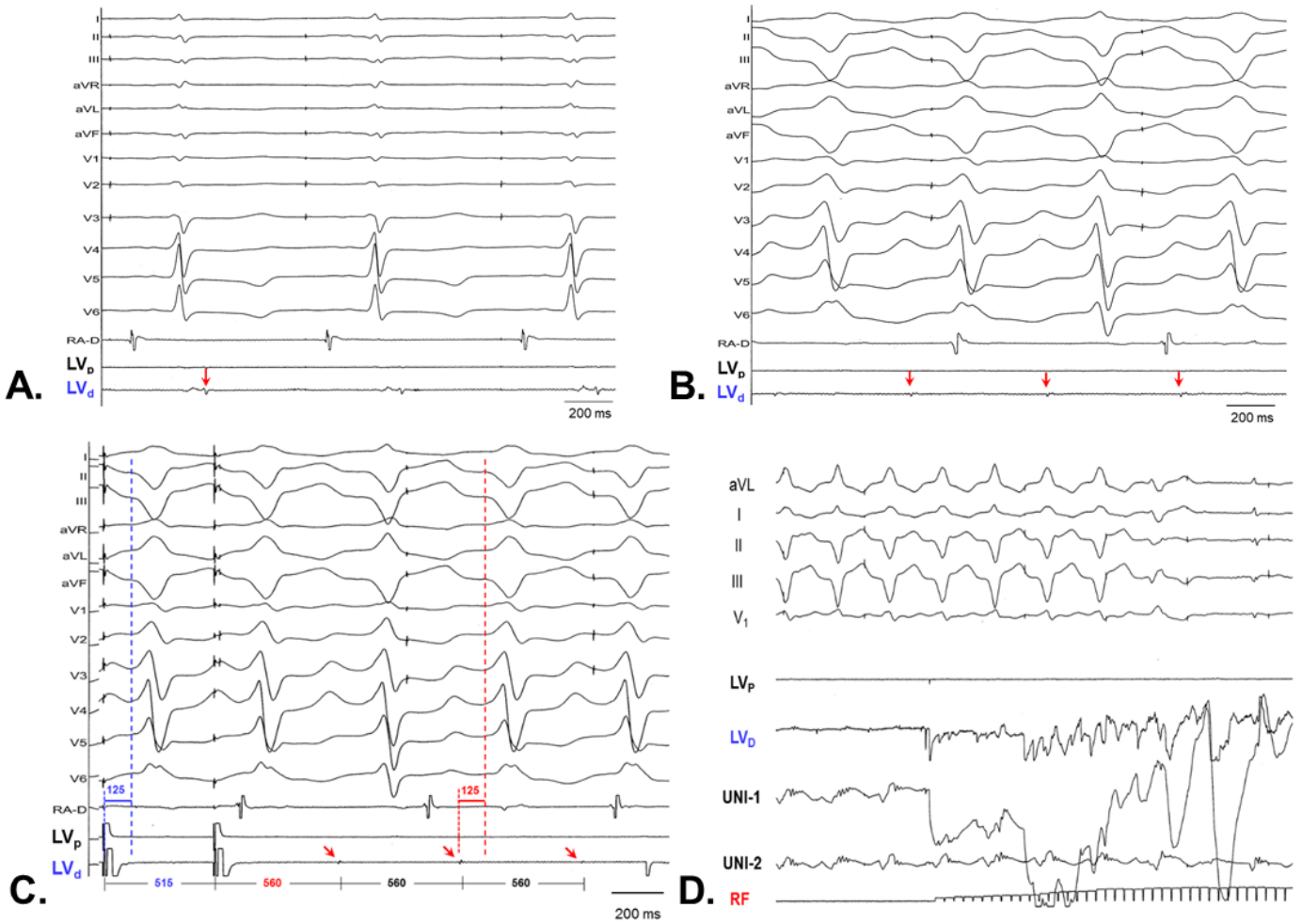


Figure 11.12. Examples of entrainment mapping in a patient with ischemic cardiomyopathy. A. A late potential (0.08 mV, red arrow) in a low voltage area. **B.** The same late potential (red arrow) was recorded during VT. Although the second half of the QRS complex of the 3rd beat was different from others due to ventricular fusion from antegrade AVN conduction, the morphology of the onset of the QRS complex was identical among the 4 beats of VT, indicating that each beat was activated

by the VT wave front. **C.** Entrainment delivered from the site shown in (**B**) produced concealed fusion. The PPI was identical to the VT cycle length (560 ms). The stim-QRS interval and EGM-QRS interval were both equal (125 ms; 18% of the tachycardia CL), suggesting that this site was located in the isthmus adjacent to its exit. **D.** Ablation there terminated VT in 3 seconds. VT was not induced after a 90-second RF application.

In the presence of concealed fusion, longer stim-QRS and EGM-QRS intervals suggest a more proximal location, or entrance of the isthmus; shorter stim-QRS and EGM-QRS intervals suggest a more distal location or exit of the isthmus. One can use the stim-QRS or EGM-QRS interval to estimate the location of an EGM in the protected isthmus. If the stim-QRS or EGM-QRS accounts for <30%, 30-50% and 50-70% of the VT cycle length, the EGM of interest is considered to be in the vicinity of the exit, middle, and entrance area of the isthmus, respectively (**Figure 11.10A**). If it is >70% of the VT cycle length, the catheter is likely to be located in an inner loop; ablation at an inner loop will not terminate the tachycardia. However, the author might still ablate there because an inner loop may be in close proximity to the protected isthmus. With the motion of the heart in VT, the ablation electrode often slides across a relatively large area. If ablation at a presumed inner loop site fails to terminate VT, gentle movement of the catheter to an adjacent site may fall onto the isthmus.

As noted above, LAVA may have multiple components. Operators must discern which component is captured by pacing (presumably the near-field potential) and should not make the mistake of selecting a far-field potential to measure the EGM-QRS interval and PPI (**Figure 11.11 to 11.13**). One may use the "n+1" or "march back" technique to trace each component of the EGM back to the last paced beat to determine which component was captured. Components that are not captured are considered far-field potentials. However, small potentials in the protected isthmus may or may not be captured by entrainment mapping. At a given site, if pace mapping in sinus rhythm showed 12/12 match but entrainment mapping fails to capture (**Figure 11.12A-C**), it may be worthwhile to deliver an RF application there with the understanding that this site may be in an inner loop or blind alley.

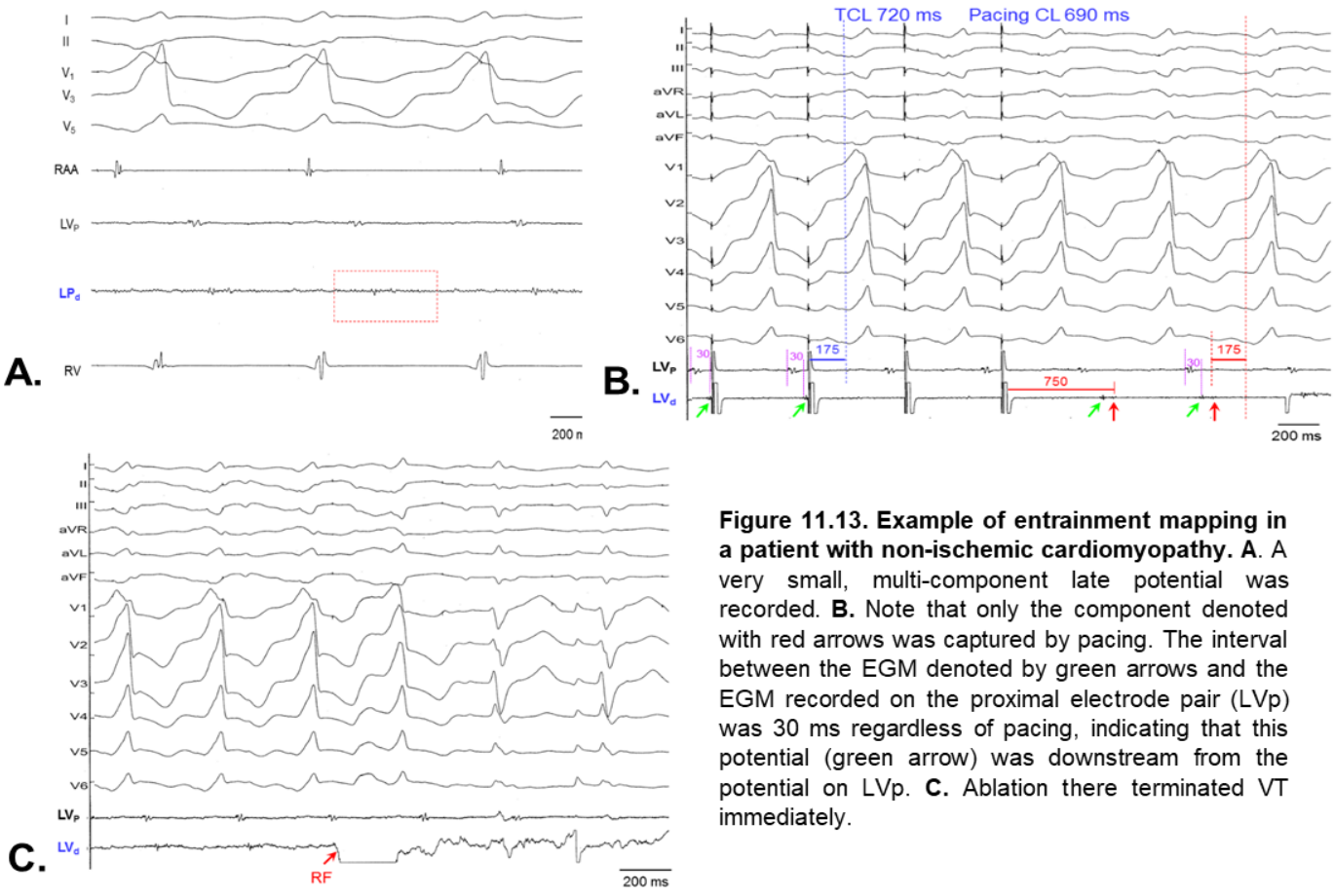
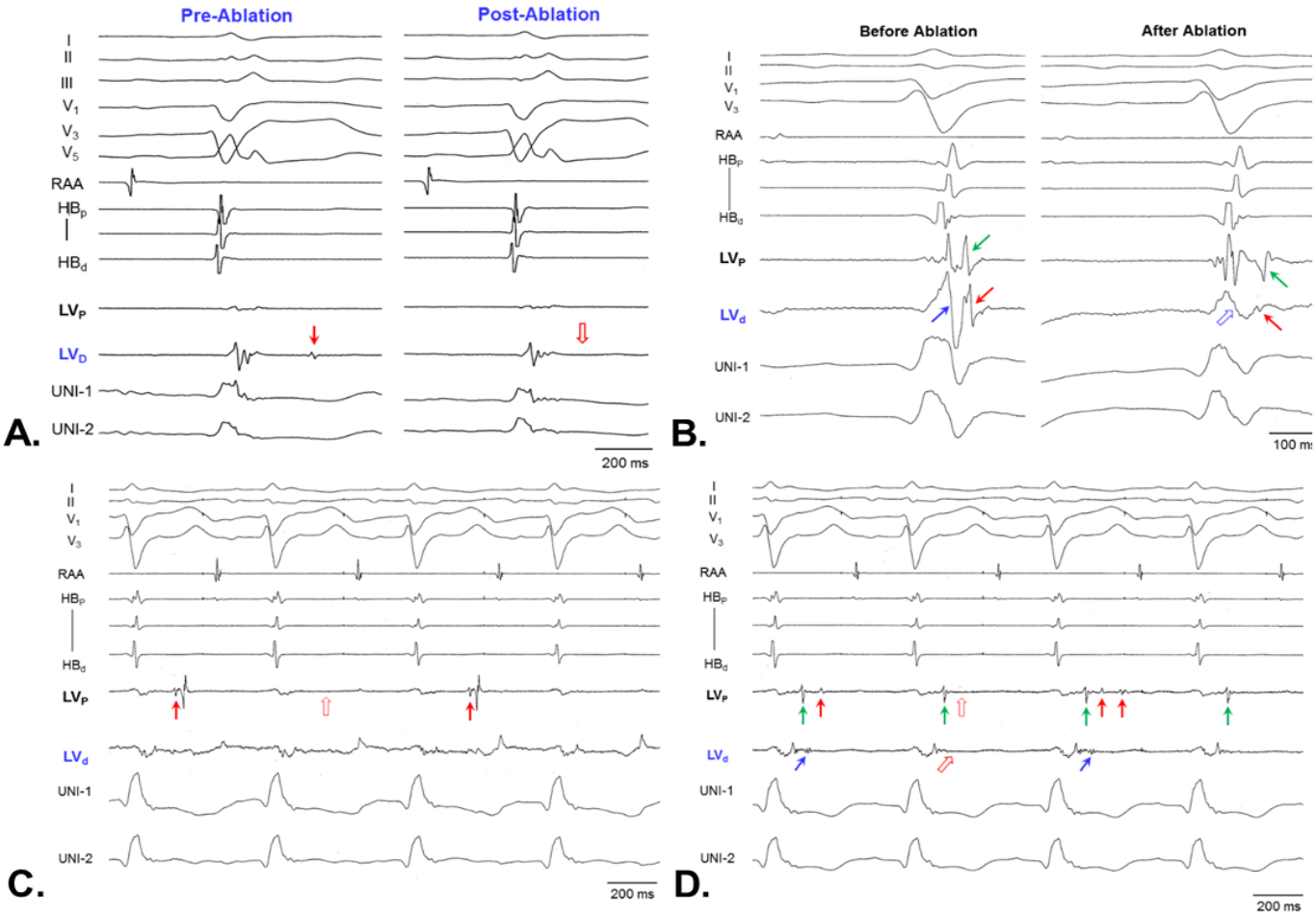


Figure 11.13. Example of entrainment mapping in a patient with non-ischemic cardiomyopathy. A. A very small, multi-component late potential was recorded. **B.** Note that only the component denoted with red arrows was captured by pacing. The interval between the EGM denoted by green arrows and the EGM recorded on the proximal electrode pair (LVp) was 30 ms regardless of pacing, indicating that this potential (green arrow) was downstream from the potential on LVp. **C.** Ablation there terminated VT immediately.

Ablation

In the OU-EP laboratory, if LAVA are recorded at a site in the vicinity of a predicted VT exit point, pace mapping is then performed there. If the stim-QRS interval is long (indicating that this site is located in a slow conduction zone), and the QRS morphology is very similar or identical to that of VT, an attempt will be made to induce VT for entrainment mapping to identify the critical isthmus of the VT circuit. Because LAVA in a diseased area are thought to represent slow conduction within a network of surviving muscle bundles, ablation of the LAVA upstream in the conduction pathway may eliminate all the downstream LAVA. For this reason, with the exception of hemodynamic instability in sinus rhythm, the OU-EP group does not start ablation until the entire low voltage area has been mapped and LAVA/late potentials have been tagged. The OU-EP group extends this practice to ablation of all VTs in structural heart disease, including non-ischemic cardiomyopathy and ARVC. The goal is to ablate most, if not all, tagged LAVA. Importantly, if a site tagged with “LAVA” before ablation does not show any LAVA after ablation elsewhere, the author will still ablate this site since the myocardial bundles there may be part of the reentrant circuit of another VT. It is not uncommon to observe that the disappeared LAVA are re-exposed by changing the direction of the ventricular wave front propagating into the diseased area.

LAVA (or some of its components) may represent far-field potentials. It is therefore important to observe the effect of ablation on LAVA. Ideally, the targeted LAVA should disappear or the amplitude of LAVA should diminish significantly if enough power, force and time are applied to the target (**Figure 11.14**). If there is no change in the morphology of LAVA after ablation with good power/force/time, it suggests that the targeted LAVA may be a far-field potential buried in dense scar or is an intramural or epicardial potential. Operators may have to apply higher power and longer RF applications to the target again in hope that the RF current may penetrate deeper. Intracardiac echo is a useful tool in this situation to allow the operator to monitor lesion formation for both efficacy and safety.



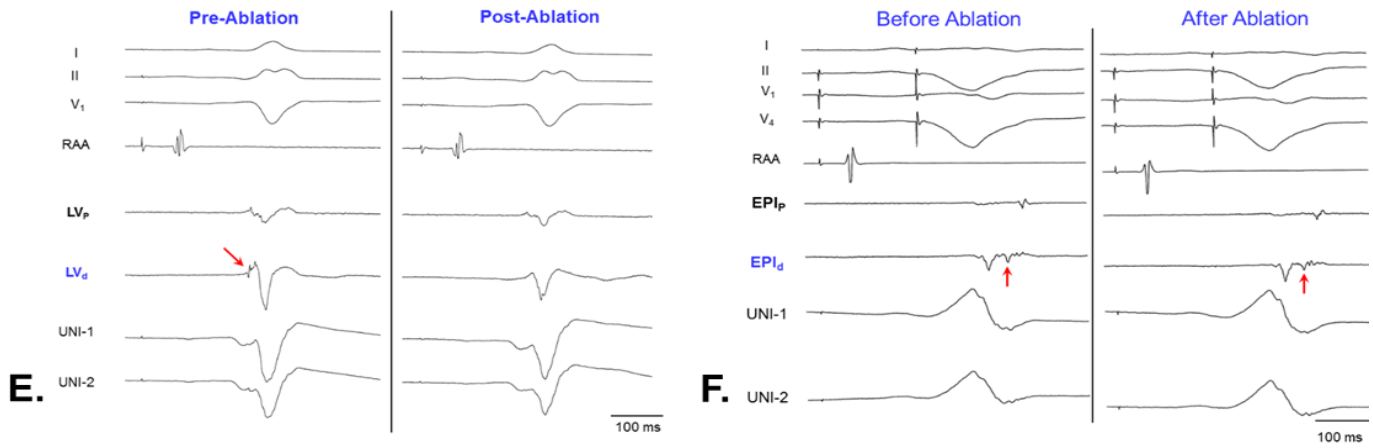


Figure 11.14. Effects of ablation on LAVA. A. The ideal response to ablation is eradication of LAVA. The red arrow and red empty arrow indicate LAVA before and after ablation, respectively. **B.** Note that the local ventricular activation (the steep negative deflection denoted by blue arrow) as well as the targeted LAVA (red arrow) were nearly eliminated. The LAVA (green arrow) on the proximal electrode pair was delayed by ablation. **C.** While RF noise on the distal pair of ablation electrode (LV_d) made assessment of ablation effect difficult, it was evident that conduction to the proximal electrode pair became 2:1. **D.** The small potential denoted by blue arrows connects to the small potential (red arrow) on the proximal electrode pair (LV_p). Disappearance of the former led to disappearance of the latter. The potential (green arrow) on the proximal electrode pair was not connected to the potential denoted by blue arrows. **E**. The timing of the targeted LAVA (red arrow) was early systolic but it was eliminated by ablation. This observation underscores the importance of not just targeting diastolic potentials because different wave fronts into the diseased area can change the morphology and timing of LAVA. **F.** LAVA (red arrow) was not affected by ablation (40 watts, 90 seconds, 15 grams of force), suggesting that this LAVA was not an endocardial potential.

Effective elimination of LAVA is also important if operators adopt the strategy of "homogenization" of the scar. Scar homogenization requires a large number of RF applications to cover the whole area. A common mistake is failure to deliver enough power/time/force at each site, and ablation only "scratches the surface" of the scar. In this case, the scar is covered with numerous ablation tags, but the LAVA network is not destroyed. **Figure 11.15** illustrates the CARTO map of a patient with ischemic cardiomyopathy who underwent VT ablation in the OU-EP laboratory. At that time, we just started using the Stereotaxis-ThermoCool catheter to ablate scar-related VT and did not increase the power and/or duration of ablation to compensate for the very soft ablation catheter. The scar was covered with ablation tags but the patient had VT recurrence a few months later. In the 2nd ablation procedure, there were still numerous LAVA in the scar. Since then, the OU-EP group has increased the power and duration of RF applications when using the Stereotaxis system and has not encountered the same issue.

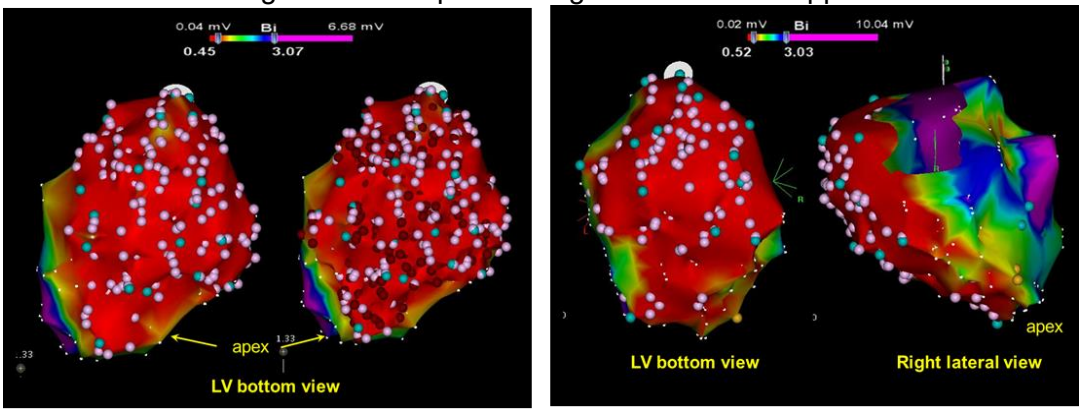


Figure 11.15. A patient with ischemic cardiomyopathy who underwent two VT ablations in the OU-EP laboratory. A. Numerous LAVA (pink dots) were identified and ablated using a Stereotaxis-ThermoCool catheter. However, power and duration of ablation were not increased to compensate for a very soft ablation catheter. **B.** When the patient returned a few months later for a repeat VT ablation, there were still numerous LAVA in the scar.

Purkinje-related VT

As already discussed, a 12-lead ECG provides the most important clue to the diagnosis of VT. Unless the rate of VT is very fast (e.g. >180 bpm), Purkinje-related VTs tend to exhibit typical RBBB or LBBB morphology. In other words, lead V1 and/or V2 begins with a small, sharp (<40 ms wide) r wave that represents the septal activation vector (left to right, posterior to anterior; **Figure 11.16A**).

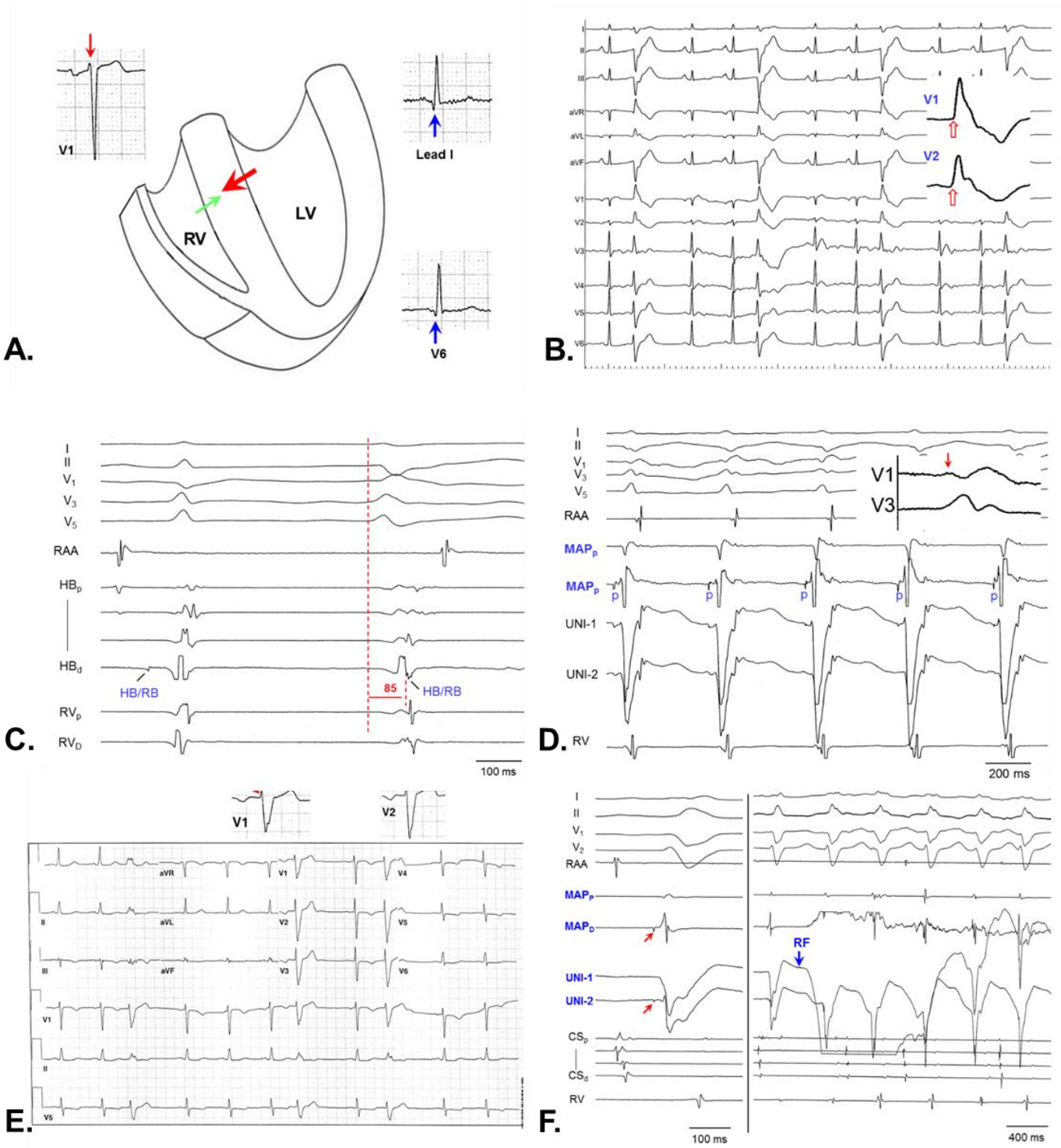


Figure 11.16. Ventricular septal activation in sinus rhythm, LBBB and RBBB. A. Ventricular septum is the first part of the ventricle that is activated by the Purkinje network of the left ventricle (red arrow). Approximately 5 ms later, the septal branch of the RBB activates the septum (green arrow). Ventricular septum is activated within 20-30 ms. The net vector is therefore left to right, posterior to anterior, creating a small, sharp r wave in V1 and/or V2 (thin red arrow) as well as a q wave (blue arrows) in lateral leads such as lead I and V6**. B.** ECG of PVCs with a RBBB morphology and superior axis, suggesting that the site of origin may be the posterior-medial papillary muscle or left posterior fascicle. However, neither lead V1 nor V2 began with a

small, sharp r wave (blank red arrow), suggestive of non-Purkinje origin. **C.** A HB potential was only occasionally recorded. When it was recorded, it was 85 ms after the onset of the QRS complex, indicating that the site of origin is unlikely to be the left posterior fascicle. This PVC originated in the posterior-medial papillary muscle. **D.** In another VT in the same patient, the small, sharp r wave was visible in lead V1 (red arrow in inset). All QRS complexes were preceded by a Purkinje potential (P) with a fixed P-V interval, indicating that this VT was a Purkinje-related tachycardia. **E**. In another patient referred for PVC ablation, the QRS complex of both V1 and V2 began with a sharp r wave (red arrow), suggesting that PVC originated in the His-Purkinje system. In this patient, "PVCs" were caused by automaticity from an atrio-fascicular accessory pathway (Mahaim fiber). Decremental atrial pacing led to progressive increment in the magnitude of preexcitation and a long AV interval, consistent with an atrio-fascicular AP (not shown). **F.** Left panel shows the EGM at the site of successful ablation. A sharp Purkinje potential (red arrow), 28 ms before the onset of QRS, was recorded on the ablation catheter. **Right panel.** Ablation induced automaticity.

When the VT rate is fast, it may reach the refractory period of septal activation; V1 and/or V2 loses its small r and become R/qR (RBBB morphology) or QS (LBBB morphology). In some patients with proximal LBB disease, the Purkinje fibers activating the LV septum may be diseased and V1 and/or V2 shows a QS pattern. The author uses the small, sharp r wave in V1 and/or V2 as an important clue to differentiate Purkinje-related VT from myocardium-related VT. However, absence of this small, sharp r wave does not exclude Purkinje-related VT at fast heart rates; diseases in the Purkinje system also can make the small, sharp r wave not visible. In the author's opinion, a small, sharp r wave in V1 and/or V2 during tachycardia has a very high positive predictive value indicating that the ventricle is activated by the His-Purkinje system (e.g. SVT with aberrancy, atrio-fascicular AVRT, bundle branch reentrant VT or fascicular VT; **Figure 11.16**). In the presence of such an r wave, the author searches for evidence to support the diagnosis of Purkinje-related tachycardia. The first evidence to look for is the relationship between the HB potential and VT as well as the HV interval. If the HV interval is fixed, it suggests a Purkinje-related tachycardia. **Figure 11.16E-F** illustrate an example of PVCs resulting from automaticity from an atrio-fascicular AP (Mahaim pathway). Typical LBBB with a small, sharp r wave in V1 and V2 clued the operator in to search for a target preceded by a Purkinje potential.

Sometimes, it is difficult to differentiate a VT originating in the posterior-medial papillary muscle from a VT originating in the left posterior fascicle. While the former usually exhibits a qR or R morphology in V1, the HB potential is usually dissociated from the VT. As both papillary muscles are covered with Purkinje network as well, some papillary PVCs or VTs can engage the peripheral Purkinje network. In this scenario, the *VH* interval is usually long (**Figure 11.16C**). For example, VT with an VH interval of 60 ms is unlikely to be a fascicular VT; operators should search for a myocardial target in this case (e.g. earliest local ventricular activation). The same assessment can be applied to atrio-fascicular (Mahaim) APs in which the HV interval during AVRT is usually -20 to -30 ms. If the HV interval has a significantly more negative value (e.g. -50 ms), two possibilities exist. First, RBBB is present (**Figure 5.25**). Second, the tachycardia is an atrio-ventricular form of Mahaim tachycardia in which the accessory AVN connects to basal ventricular myocardium, rather than the peripheral RBB network, increasing the conduction time to the HB. Local ventricular activation near the tricuspid annulus is early in this situation, similar to an ordinary AP (**Figure 5.27F**)

Left Fascicular VT originating in the left posterior fascicle (LPF-VT)

Left fascicular VTs are more prevalent in Asia. Fascicular VTs originating in the left anterior fascicle are relatively rare in the Western Hemisphere. Salient work of Asian electrophysiologists established reentry as the mechanism responsible for LPF-VT. Most cardiologists have been educated to view the left anterior and posterior fascicles as the only two branches of the LBB. Important findings by Dr. Tawara and Dr. Demoulin (Demoulin JC, British Heart Journal 1972;34:807-814) that the LV endocardium is covered with extensive Purkinje network were overlooked. A septal bundle, which is present in many people, is seldom taught. In the author's opinion, a diseased septal Purkinje network, including the septal fascicle(s), may constitute the reentrant circuit of LPF-VT. In the author's simplified view, one can consider the reentrant circuit of LPF-VT as similar to slow/fast AVNRT. The diseased septal Purkinje fibers (slow conduction zone) forms the antegrade limb of the reentrant circuit; the healthy Purkinje fibers of the LPF form the retrograde limb (**Figure 11.17A**). Potentials from diseased Purkinje fibers are termed P1 while potentials from healthy LPF fibers are termed P2. In sinus rhythm, the normal (P2) and diseased (P1) Purkinje fibers are activated in the

same direction; therefore, P1 potentials are obscured by local ventricular activation and are usually not visible (**Figure 17B**). In LPF-VT, using a 20-pole electrode catheter, one may record a series of P1 potentials conducting from the base to apex (antegrade limb) and another series of potentials (P2) conducting in the opposite direction (retrograde limb; **Figure 11.17**). Ablation targeting distal P1, junction of P1 and P2 or earliest P2 can eliminate LPF-VT. The ablation target that Dr. Jackman originally proposed (early Purkinje potential, usually 20-40 ms before the onset of QRS) is probably either distal P1 or proximal P2 in the vicinity of the site where P1 and P2 connect.

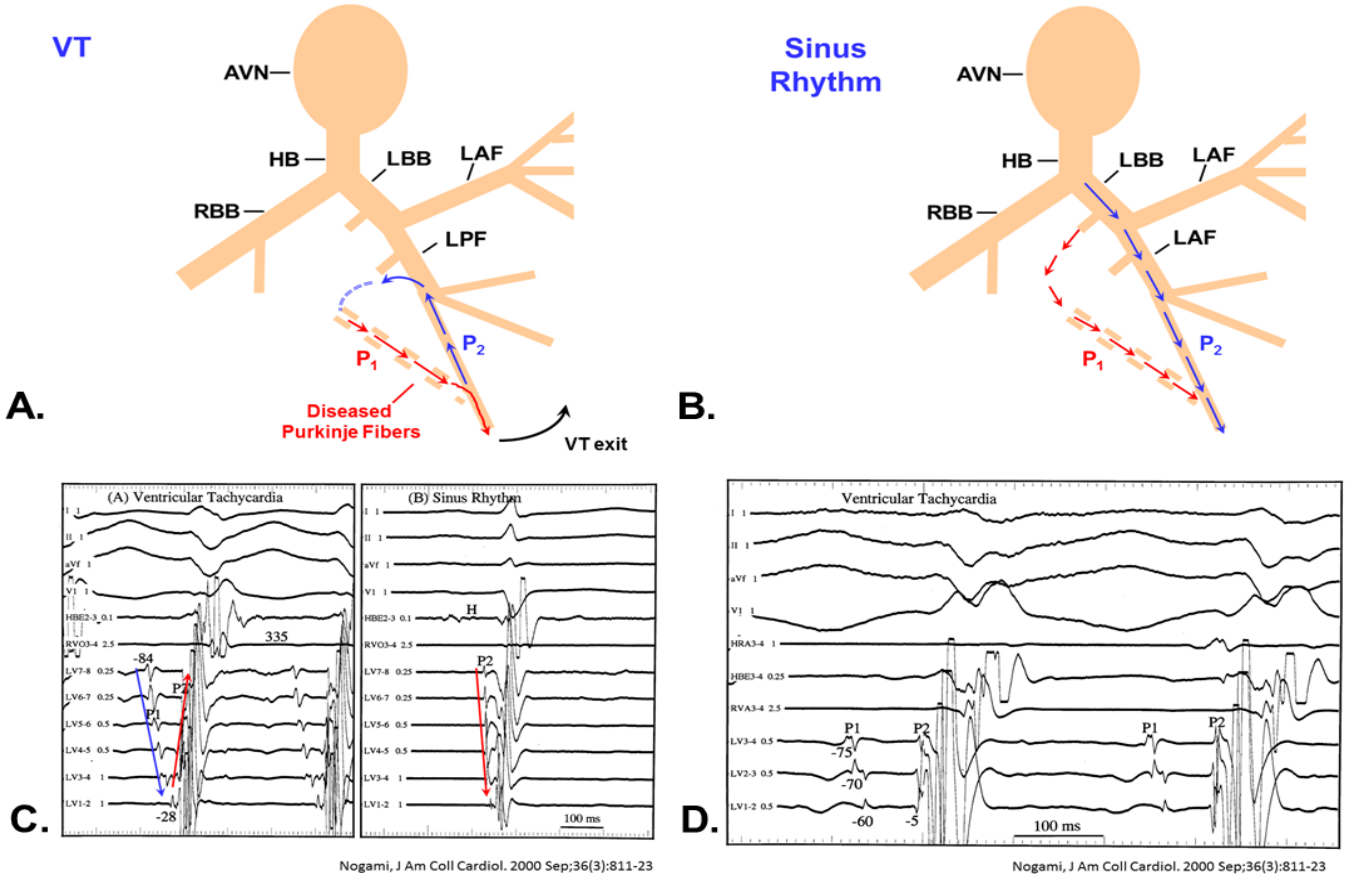


Figure 11.17. Reentrant circuit of LPF-VT. A. The diseased Purkinje fibers (slow conduction zone) form the antegrade limb of the reentrant circuit (the P1 potential); the healthy Purkinje fibers of the LFP form the retrograde limb (the P2 potential). **B.** In sinus rhythm, the normal (P2) and diseased (P1) Purkinje fibers are activated in the same direction; therefore, diseased Purkinje potentials (P1) with slow conduction are obscured by local ventricular activation and are usually not visible. **C. Left panel.** During VT, a diastolic potential (P1; blue arrow) and a presystolic Purkinje potential (P2; red arrow) were recorded. While P1 was recorded earlier from the proximal than the distal electrodes, P2 was recorded earlier from the distal than the proximal electrodes. **Right panel.** During sinus rhythm, recording at the same site showed the P2 (red arrow), which was recorded before the onset of the QRS complex. **D.** Recordings at the site of successful ablation during VT. P1 and P2 were recorded in the midseptal area. *Modified from Nogami et al. J Am Coll Cardiol 2000;36(3):811-823 with permission.*

Deploying a closely-spaced multi-electrode catheter to record P1 and P2 certainly provides great insight into the location of the reentrant circuit. However, it is very technically challenging to position such a catheter along the LV septum without traumatizing the Purkinje network constituting the reentrant circuit. Dr. Jackman often jokes about himself as a perfectionist, but there are two arrhythmias for which perfection is the enemy of good enough: fascicular VT and atrio-fascicular AP (Mahaim). Both arrhythmias are caused by the Purkinje tissue situated superficially on the endocardial surface which is easily mechanically traumatized. After a few failed attempts to record P1 and P2, the primary targets sought by the OU-EP group to treat LPF-VT are still early Purkinje potentials or double Purkinje potentials (**Figure 11.18**). The latter may represent

the site proximal to where P1 and P2 connect (recording a distal P1 and a proximal P2 potential); ablation there potentially may eliminate both the antegrade and retrograde limbs.

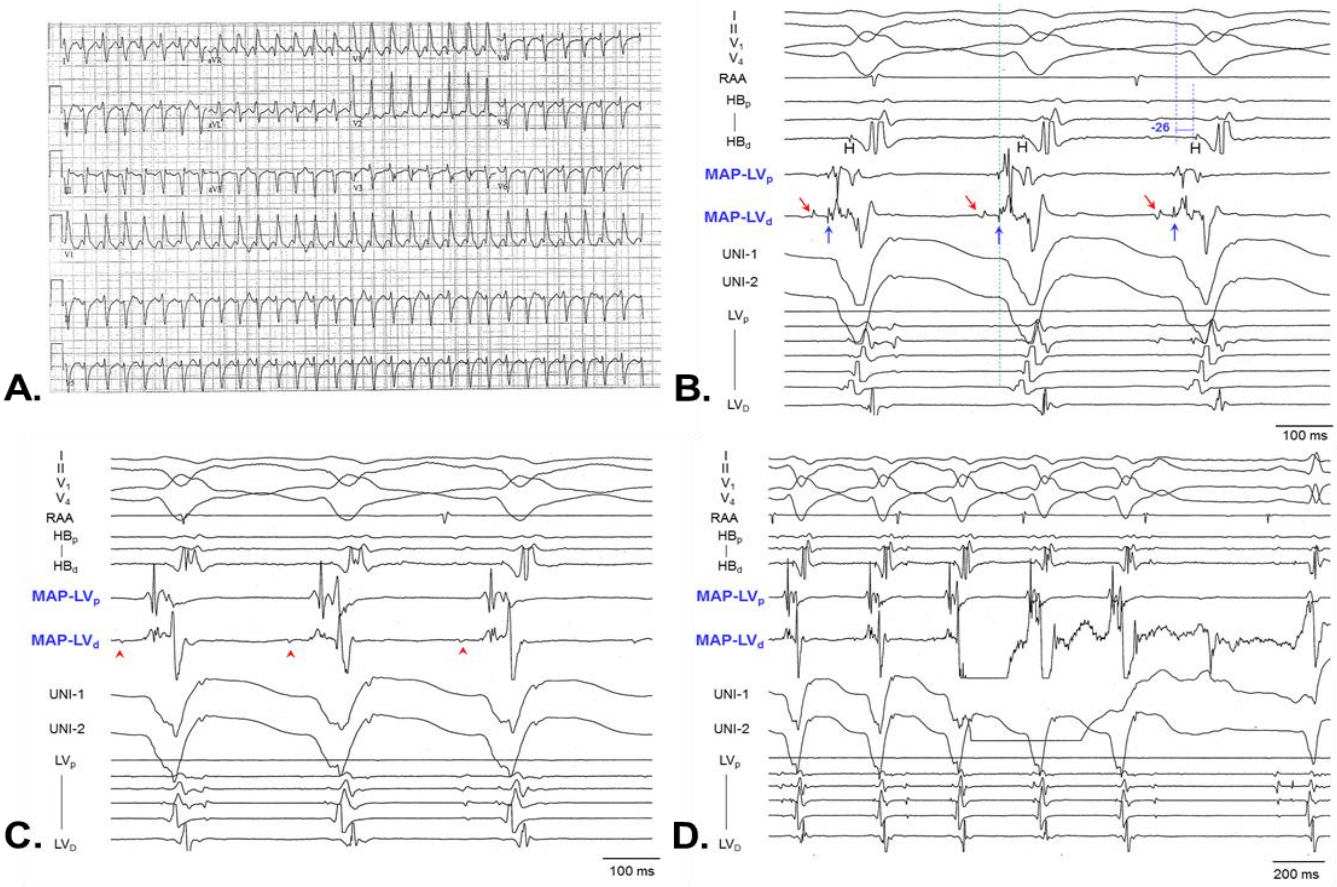


Figure 11.18. A young woman with prior unsuccessful ablation for LPF-VT. A. Note that lead V1 or V2 did not show a small, sharp r wave in this patient. Absence of the septal activation vector does not exclude fascicular VT; the next clue is to look for a fixed HV interval. **B.** The HV interval was stable at -26 ms, consistent with a Purkinje-related VT (e.g. fascicular VT). Note that two potentials (red and blue arrows) were recorded on the mapping catheter positioned on the LV septum. The second potential (blue arrow) was simultaneous with the onset of the QRS complex and also immediately followed by a local ventricular potential. VT was terminated by mechanical trauma. The author hypothesized that this site was slightly proximal to the connection between P1 and P2. The first potential might be a P1 (red arrow); the 2nd potential might be a P2, in close proximity to the exit point of the reentrant circuit. **C.** VT was reinduced. Note that the first potential was not as sharp as the one shown in (**B**). An example of "perfection is the enemy of good enough". This was probably a P1 potential. **D.** Ablation there terminated VT.

The HV interval during a Purkinje-related VT provides important clues to the location of the site of origin. If the exit point is in distal Purkinje network, it reaches ventricular myocardium quickly to produce a QRS complex but the wave front has to travel a longer distance in the Purkinje network to the HB. The HV interval is usually in the range of -20 to -40 ms (**Figure 4.21**). By contrast, if the exit point is in the proximal Purkinje network, the wave front travels a shorter distance to the HB but travels a longer distance to reach ventricular myocardium; thus, the HV interval is usually less negative (e.g. -10 to 0 ms). If the HV interval is a positive value, the site of origin is very proximal in the His-Purkinje system (e.g. distal HB). The author has used this assessment for longer than a decade to approximate the site of origin of Purkinje-related arrhythmias, and it has worked quite well.

Fascicular VT is notorious for being difficult to induce and easily mechanically traumatized. Operators should make every attempt to induce VT before putting a catheter into the LV. If VT was induced earlier but was not re-inducible for mapping, another useful technique is to estimate the location of the reentrant circuit of the LPF-VT by measuring the HV interval during sinus rhythm and VT (**Figure 11.19**). This approach was derived from a similar technique that evaluates the conduction time of the lower common pathway in the AVNRT reentrant circuit by comparing the HA interval during AVNRT and RV pacing (**Figure 7.5**). By

measuring the HV interval during sinus rhythm and the HV interval in LPF-VT, one can approximate the interval between the earliest P2 and the onset of QRS complex. The site generating the earliest P2 during VT is in close proximity to where P1 and P2 connect. If LPF-VT is no longer inducible, operators can search for a site in the LPF territory with that specific Purkinje-V interval and empirically ablate this site. Another approach in the event of non-inducibility is to map the septal Purkinje potentials in sinus rhythm to delineate the courses of the septal Purkinje fibers. Linear RF lesions are then delivered from the midseptal LV to the junction of the septum and inferior wall in order to transect the reentrant circuit. The length of this line is usually shorter than 2 cm.

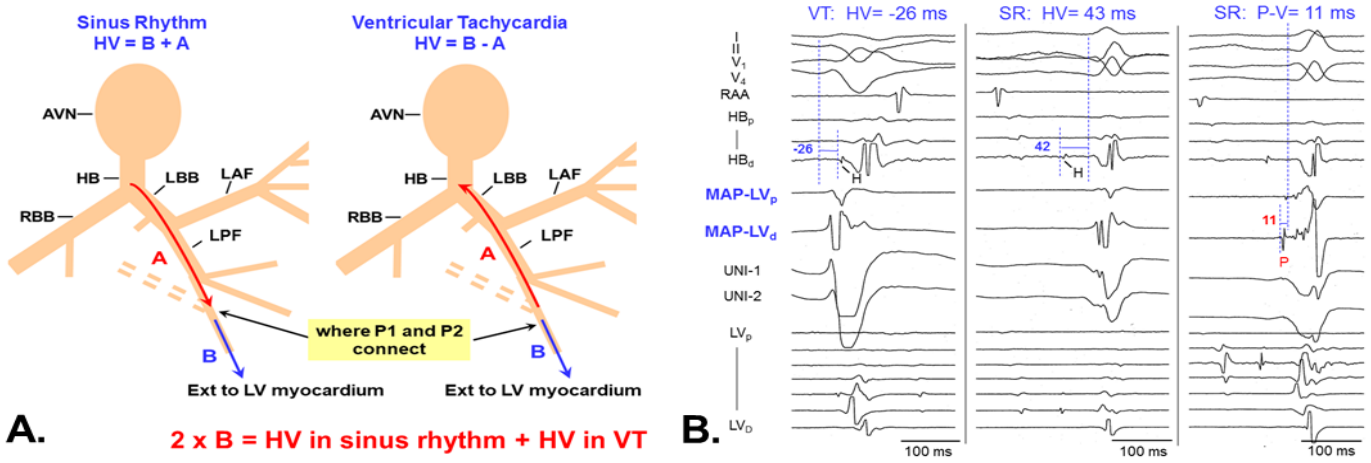


Figure 11.19. Calculation of the timing of earliest P2 in sinus rhythm. A. In sinus rhythm, the conduction time from HB potential to the onset of QRS (HV interval) equals A + B. Interval A represents the conduction time from the HB potential to P2; interval B represents the conduction time from P2 to LV. In VT, the HV interval equals B – A. One can use the HV interval in sinus rhythm and VT to calculate the conduction time of B in sinus rhythm. **B**. In this example, the HV interval in VT and sinus rhythm was -26 ms and 43 ms, respectively. This predicts that the interval from the earliest P2 to LV would be 9 ms ([43-26] / 2). **Right panel.** The P-V interval at the site of successful ablation was 11 ms.

Bundle-branch reentrant VT

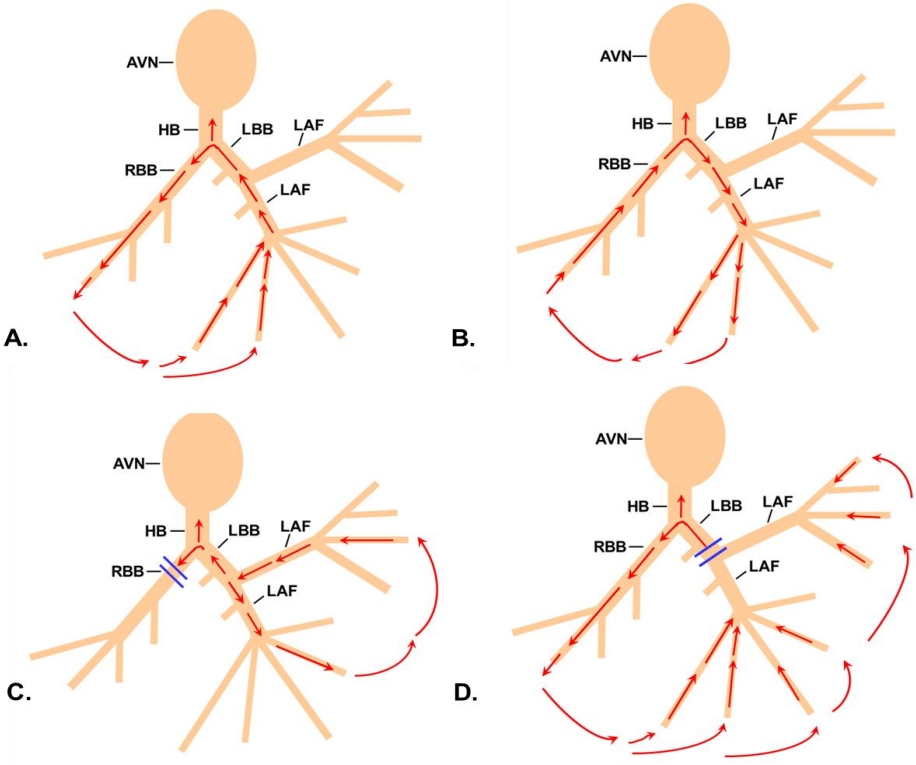


Figure 11.20. Schematic representation of BBR-VT. A. In the most common type of BBR-VT, RBB and LBB serves as the antegrade and retrograde limb of the circuit, respectively. VT exhibits a LBBB morphology. **B.** In this type of BBR-VT, the antegrade and retrograde limb of the reentrant circuit is formed by LBB and RBB, respectively. VT exhibits a RBBB morphology. **C.** After RBB ablation to treat BBR-VT, the left anterior and posterior fascicle form the reentrant circuit and create inter-fascicular VT. **D.** Before RBB ablation, the left anterior and posterior fascicles may be retrograded invaded by the trans-septal ventricular wave front, preventing inter-fascicular VT from occurring.

Bundle-branch reentrant VT (BBR-VT) usually occurs in patients with severely diseased Purkinje systems in which delay in the Purkinje system is significant enough to set up reentry. Most patients with BBR-VT have baseline LBBB and a long HV interval. A PVC (e.g. from the RV) retrogradely invades the RBB and conducts trans-septally to the LV to engage the peripheral LBB (**Figure 11.20**). Subsequently, the wave front propagates retrogradely to the bifurcation point of LBB and RBB and then conducts antegradely to the RBB. The wave front propagates continuously between the LBB (retrograde limb) and RBB (antegrade limb). This is the most common form of BBR-VT, which has a typical LBBB morphology. Another form of BBR-VT propagates in the opposite direction, using the LBB as the antegrade limb and RBB as the retrograde limb. Even though the His-Purkinje system is severely diseased in these patients, rapid conduction in the LBB and RBB leads to fast VT (often >200 bpm).

BBR-VT has the following EP characteristics (**Figure 11.21**). First, the HV interval in VT is ≥ that during sinus rhythm. Second, if the VT cycle length wobbles, the changes in the H-H interval lead the changes in the V-V interval. Third, entrainment delivered from the RV apex produces a short PPI because the RV apex is in close proximity to the reentrant circuit (RBB). However, the QRS morphology during pacing is usually very similar, but not identical to that in VT (i.e. some degree of manifest fusion) because RV apex entrainment captures RV myocardium as well. Dr. Jackman rarely positions the RV catheter at the RV apex for resetting or entrainment. BBR-VT is the only exception because the RV apex is in close proximity to the reentrant circuit.

Operators need to be aware of a rare form of BBR-VT called inter-fascicular VT in which the VT circuit is formed by left anterior and posterior fascicles (**Figure 11.20C-D**). Most of the reported cases share the same scenario: VT occurred after successful ablation of the RBB to treat BBR-VT. In patients with severely diseased Purkinje systems with baseline complete LBBB, ventricular activation is initiated by the RBB; the wave front then conducts trans-septally to the LV and possibly retrogradely invades the peripheral left anterior and posterior fascicles. After the RBB was ablated, antegrade conduction over the diseased LBB activates the left-sided Purkinje network. If there is a significant discrepancy in conduction velocity between the left anterior and posterior fascicle, a premature beat can set up reentry between the two fascicles. Of note, in inter-fascicular VT, the HV interval during VT is expected to be shorter than that in sinus rhythm. However, there may not be a 1:1 relationship between the HB potential and VT because the His-Purkinje system is severely diseased. The wave front may or may not be able to conduct retrogradely to the HB.

Two important points about His-Purkinje related tachycardias need to be addressed.

1. The HB potential recorded in VT is a retrograde HB potential. The HV interval in tachycardia is determined by the wave front propagating antegradely to exit to the ventricle and the wave front propagating retrogradely to the HB. That is, the two wave fronts propagate in opposite directions. The HV interval in tachycardia can be affected by the relative speed of conduction in two directions. Therefore, on rare occasions, the HV interval can be shorter in BBR-VT than that in sinus rhythm.

2. If clinical VTs showed both LBBB morphology and RBBB morphology with left anterior or posterior fascicular block, these findings should alert the operators to seek Purkinje-related VT. Multiple VT morphologies are caused by different Purkinje reentrant circuits and therefore different exit points. Of note, this type of tachycardia is usually fast; typical LBBB or RBBB pattern may or may not be observed.

Purkinje-trigger polymorphic VT or VF

In some patients with idiopathic VF or idiopathic polymorphic VT, tachycardias are triggered by firing from the Purkinje system, papillary muscles or outflow tracts. In the case of Purkinje firing, the QRS complex of the leading beat of the polymorphic VT or VF is often relatively narrow and exhibits *typical* bundle branch block or hemi-block morphology (**Figure 11.22**). The leading beat is preceded by a Purkinje potential, and the ablation target is the leading Purkinje potential. Because Purkinje fibers form a network on the endocardial surface, ablation needs to cover a relatively large area in order to destroy the targeted Purkinje network to prevent the triggered beat from conducting to the rest of the Purkinje system. Ablation of Purkinje triggers

may initiate VF, but RF application must be continued to destroy the targeted Purkinje trigger. The author only had a handful of cases with this arrhythmia but would ablate for at least 20 more seconds before defibrillating the patient. Because Purkinje fibers are superficial, it does not require long RF applications to eliminate Purkinje targets. Electrode-tissue contact may be more stable in VF due to lack of ventricular contraction. It would be a mistake to discontinue RF application immediately after VF occurs; the RF application may not be long enough to make a good lesion.

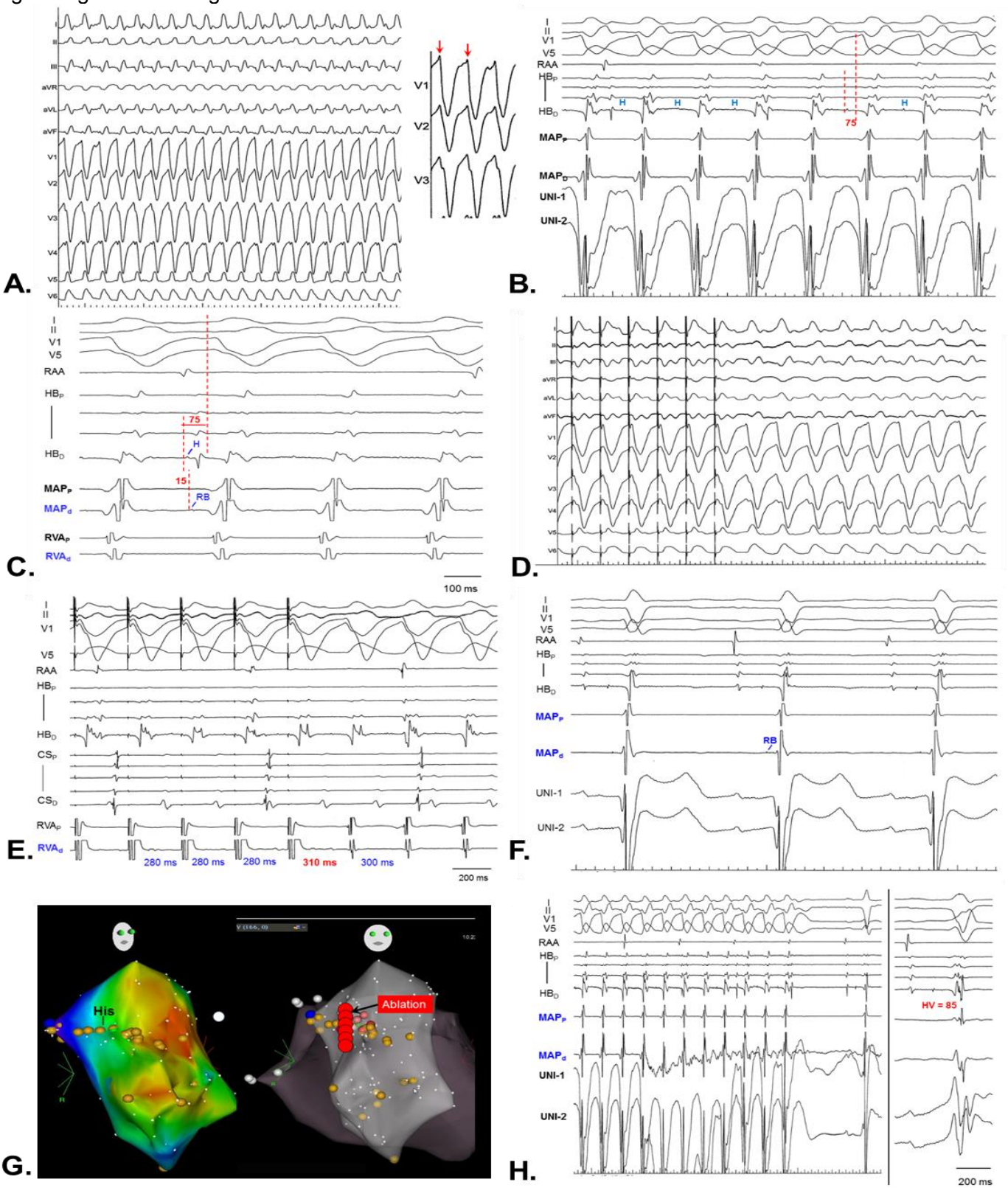


Figure 11.21. Bundle-branch reentrant VT in a patient with non-ischemic cardiomyopathy. A. VT showed typical LBBB morphology. Note that leads V1 and V2 began with a small, sharp r wave (red arrows in inset). **B.** In VT, HV interval was fixed at 75 ms, 5 ms longer than the HV interval in sinus rhythm, indicating that this was a Purkinje-related VT, most likely BBR-VT. **C.** The right bundle potential (RB) was 15 ms later than the HB potential, indicating antegrade conduction over the RBB. **D and E**. Entrainment delivered at the RV apex showed similar but not identical morphology to that of VT. PPI was only 10 ms longer than the VT cycle length indicating that RV apex was close to the reentrant circuit. **F.** A right bundle potential was selected as the ablation target**. G. Left panel.** RB potential (gold dots) were labeled. **Right panel.** A vertical ablation line transected the right bundle. **H. Left panel**. RF application there terminated BBR-VT immediately. **Left panel.** After ablation, the HV interval was prolonged from 70 ms to 85 ms with RBBB morphology.

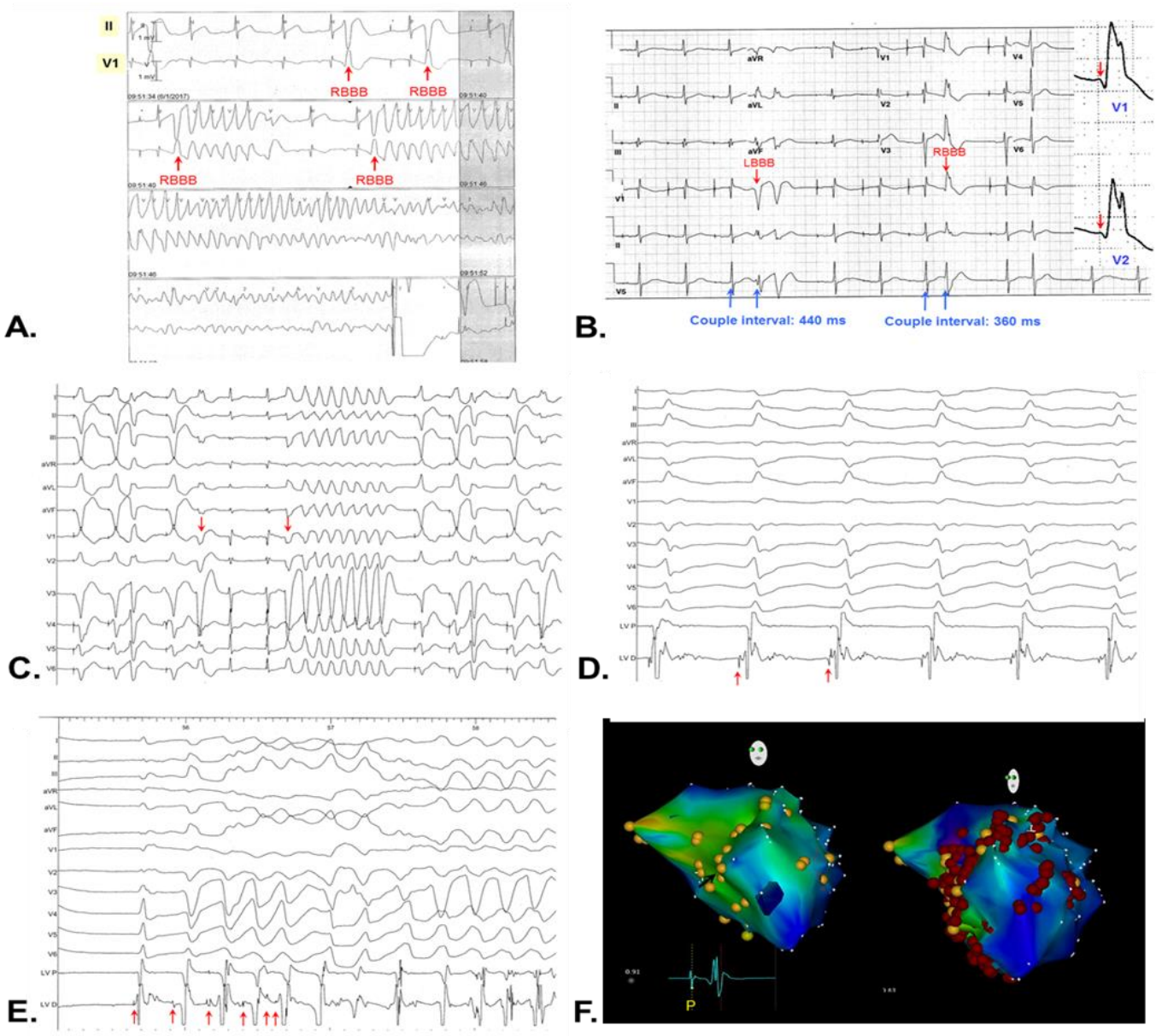


Figure 11.22. Recurrent VF triggered by Purkinje firing in a 55 y/o male without structural heart disease. A. VF was repeatedly triggered by the same PVC with RBBB morphology and superior axis. **B.** Short-coupled PVCs with either LBBB or RBBB morphology were captured. Insets showed that the QRS complex in V1 and V2 began with a small sharp r wave, suggesting that PVC originated from the His-Purkinje system. Note that in (**A**) and (**B**), PVCs showed RBBB with either superior or inferior axis, suggesting that peripheral Purkinje fibers of both the left anterior and posterior fascicles were involved. **C.** Occasionally, polymorphic VT was triggered by a PVC with LBBB morphology. **D.** Purkinje-related VT was induced. Red arrows: Purkinje potentials. **E.** VF was triggered by Purkinje firing. Note that Purkinje potentials (red arrows) led the first few beats of polymorphic VT before degenerating into VF. **F.** In the first procedure, firing only occurred a few times, not sufficient to localize the source of firing. Empirical ablation of the Purkinje potential in the left anterior fascicle failed to eliminate the

VT/VF. The patient continued to suffer from daily ICD shocks despite being placed under general anesthesia. In the second procedure, no firing could be induced. After mapping the LV Purkinje potentials (gold dots; **left panel**), RF applications were delivered to peripheral Purkinje potentials (distal toward proximal) in order to destroy the entire network. The working hypothesis was to preserve the proximal Purkinje system to allow the peripheral Purkinje potentials to be identified and ablated. If ablation leads to complete LBBB, that indicates interruption of the peripheral Purkinje system (**right panel**). The proximal Purkinje system (e.g. proximal left anterior and posterior fascicles) were ablated last. The patient did not have any VT during a 12-month period.