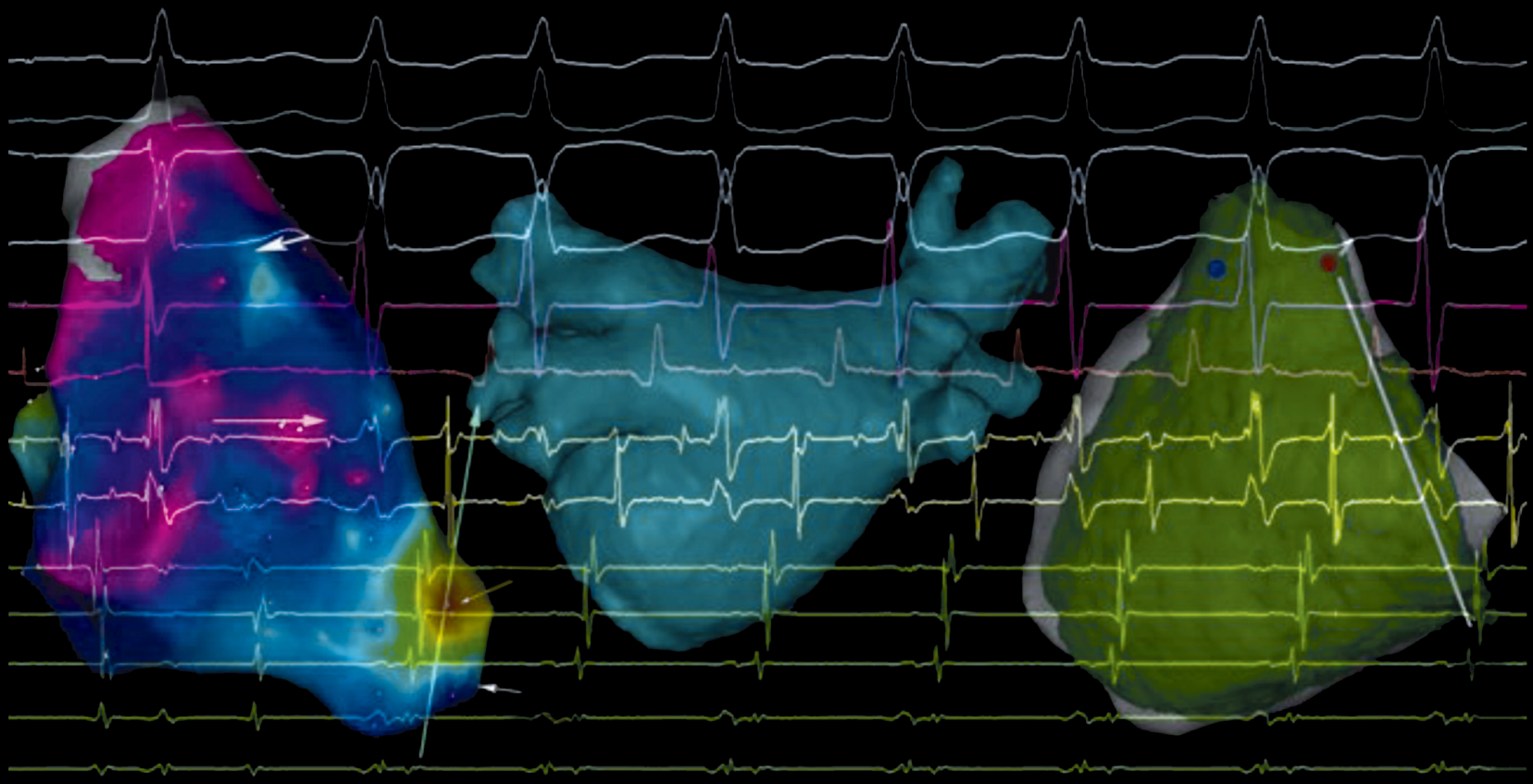
Second Edition

Cardiac Electrophysiology

A Visual Guide for Nurses, Techs, and Fellows



Paul D. Purves • George J. Klein
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Lorne J. Gula • Andrew D. Krahn

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cardiotext
PUBLISHING
Minneapolis, Minnesota

Second Edition

Cardiotext Publishing, LLC
3405 W. 44th Street
Minneapolis, Minnesota 55410
USA
www.cardiotextpublishing.com

Any updates to this book may be found at:
www.cardiotextpublishing.com/electrophysiology-heart-rhythm-mgmt
/cardiac-electrophysiology-second-edition-purves

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Cover design by Paul D. Purves; interior design by Elizabeth Edwards

Library of Congress Control Number: 2020952895

ISBN: 978-1-942909-52-1
eISBN: 978-1-942909-53-8

Printed in Canada
1 2 3 4 5 6 7 25 24 23 22 21 21

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Contents *continued*

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Preface

Cardiac Electrophysiology: A Visual Guide for Nurses, Techs, and Fellows is just that—a visual guide to electrophysiology. Written for allied health personnel, including nurses, technologists, industry personnel, and new EP fellows, this book presents the most important aspects of the EP study using pertinent images accompanied by detailed discussions of the principles involved. Topics covered include hardware connections ("connectology"), catheter placement, intracardiac signals, normal electrogram sequences associated with sinus rhythm, and—given an initial diagnosis of paroxysmal supraventricular tachycardia—the methodology we employ to uncover the mechanism of the tachycardia.

Because we have chosen to focus on the beginner, many complexities are either omitted or discussed in generalities only. The commentaries that follow most of the discussions throughout the book are intended to pique the reader's interest in advanced EP principles. Readers interested in gaining a more thorough understanding of these topics are referred to more comprehensive textbooks or to the current literature.

The computer/recording system used to generate the graphics in this guide may differ from the system with which you are familiar; however, the basic principles of signal processing (digitization, amplification, and filtering) remain the same. The channels displayed and their positions on the monitor are standard in our lab and may prove initially challenging if your lab organizes the display screen in a different manner. Regardless, try to focus on the information within these channels rather than on the cosmetics of how they are displayed. It is useful to interpret EP study findings no matter the format or from which center the recordings come, especially when preparing for EP certification examinations.

Teamwork in the EP lab is critical. In our lab, it is standard practice to encourage *everyone* to be involved and engaged in the diagnostic study and to be continually aware of any ECG changes, electrogram changes, or changes in catheter position. Checks and balances of this nature and a methodical, consistent approach are crucial components in the teamwork that enables any lab to run smoothly.

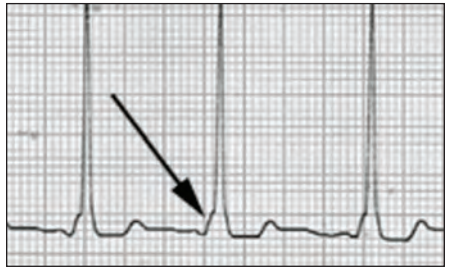
Video Descriptions

Abbreviations and Glossary

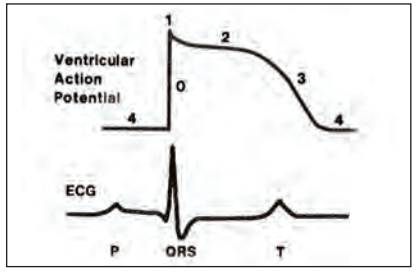
Accessory pathway
An additional electrical connection (other than the AV node) between the atria and ventricles

Accessory pathways may conduct:

- Antegrade only – generates a delta wave
- Retrograde only – no delta wave
- Bi-directional – generates a delta wave

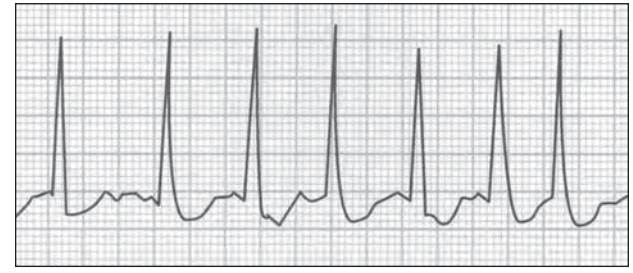


Action potential
The waveform generated by a cell's depolarization

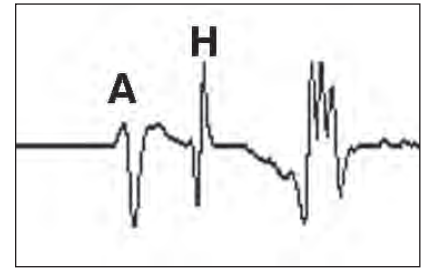


AEGM
Atrial electrogram

AF
Atrial fibrillation



A-H interval
Transit time through the AV node



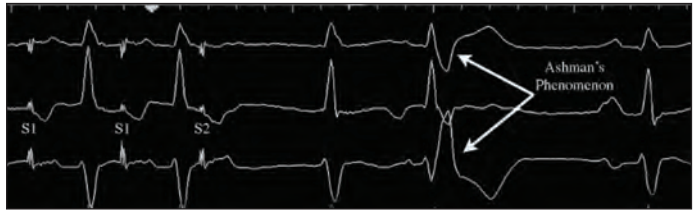
Anisotropy
The concept that conduction velocity is determined by the angle of initial depolarization

Antidromic
Denoting a wave of depolarization that is traveling retrogradely through the AV node. This term is usually used when describing the direction of the wavefront of an atrioventricular reentry circuit. In this scenario, the wave of depolarization travels down the accessory pathway and back up the AV node.

It can also denote a wave of depolarization that is traveling in the opposite direction to the predominate wave. For instance, a clockwise wave of depolarization introduced into a counterclockwise atrial flutter, therefore called an antidromic wavefront.

AP
Accessory pathway

Ashman's phenomenon
When a relatively long cycle (R-R) is followed by a relatively short R-R, the QRS associated with the short R-R often has right bundle branch block (RBBB) morphology. Often referred to a "long-shorting" the bundle branches. The right bundle branch has had insufficient time to adapt its ERP to a sudden change in heart rate. It therefore blocks.



AT
Atrial tachycardia

Atrial fibrillation
A disorganized atrial rhythmrhythm (see AF above)

Atropine
A parasympatholytic used to increase heart rate

AV
Referring to the atrioventricular node or atrioventricular conduction

AVCS
AV conduction system

AVNRT
AV nodal reentrant tachycardia

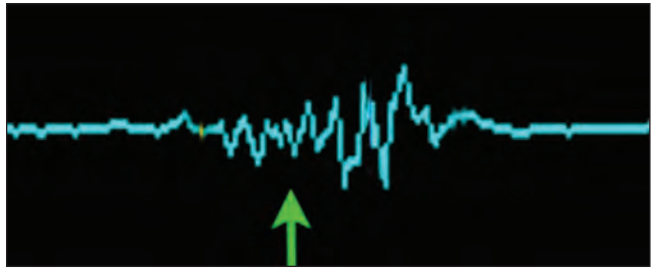
AVRT
Atrioventricular reentrant tachycardia

Bipolar
Refers to the use of two poles (a positive and a negative electrode), usually in close proximity to one another. However, a unipolar lead is just a widely spaced bipole.

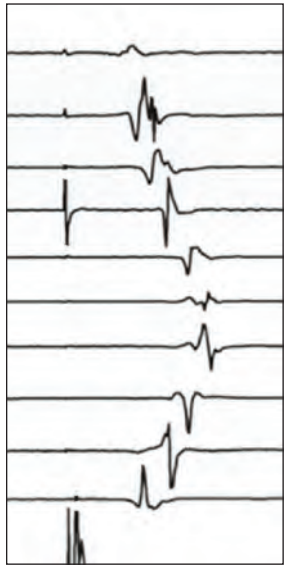
Bundle of His
Anatomic structure connecting the AV node to the bundle branches

Carto
A 3D mapping system from Biosense Webster

CFEs
Complex fractionated electrograms, usually associated with the left atrium during AF



Chevron
CS 1-2 activation occurs as early as CS 9-10 activation



Concealed
An unseen penetration of a wave of depolarization into a structure that subsequently affects the conduction properties of that structure

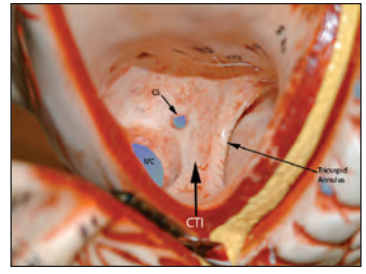
Contralateral
On the other side – it often is used as "contralateral bundle branch block"

It refers to:

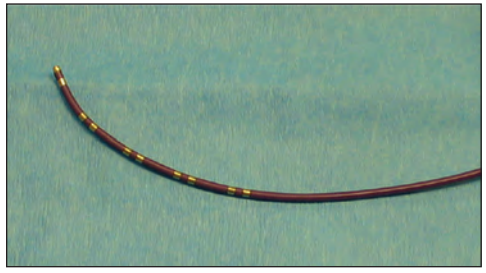
- a LBBB pattern during a right-sided tachycardia
- a RBBB during a left-sided tachycardia
- the effect of that bundle branch block on the tachycardia

CS
Coronary sinus

CTI
Cavo-tricuspid isthmus

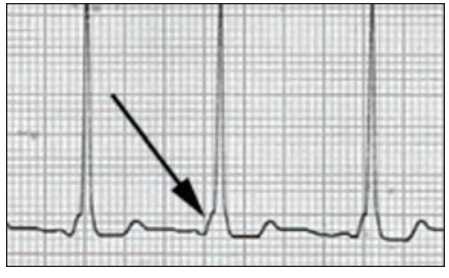


Decapolar
A 10-pole catheter, often the CS catheter



Decrement
The ability of the AV node to slow conduction

Delta wave
The initial part of this widened QRS that represents conduction directly into the ventricle

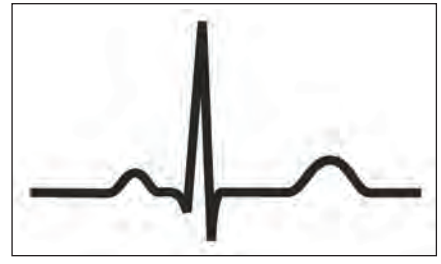


Depolarization
The process by which a cell changes on the inside from electrically negative to electrically positive

Digitization
The conversion of an analog signal to a digital signal

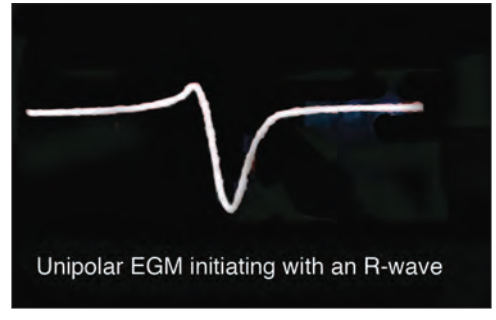
Duo-deca
A 20-pole catheter

ECG
Electrocardiogram



Echo
An unexpected return of a wave of depolarization to the chamber where the wave originated

EGM
Electrogram



Before We Begin an SVT Study

There are many reasons for patients to undergo an electrophysiology (EP) procedure. However, not every patient needs a full diagnostic study for supraventricular tachycardia (SVT), which involves incremental ventricular pacing followed by ventricular extra-stimulus pacing, atrial extra-stimulus pacing, and finally, atrial incremental pacing. Rather, the choice of catheters and equipment will change depending on the intent of the procedure. When the diagnosis has been clearly established, a more focused approach is employed. For example, if typical atrial flutter is the working diagnosis, our study will be abbreviated and focused as follows:

1. Confirm, using entrainment, that it is indeed a typical, cavo-tricuspid isthmus (CTI)–dependent flutter.
2. Proceed with a CTI ablation.

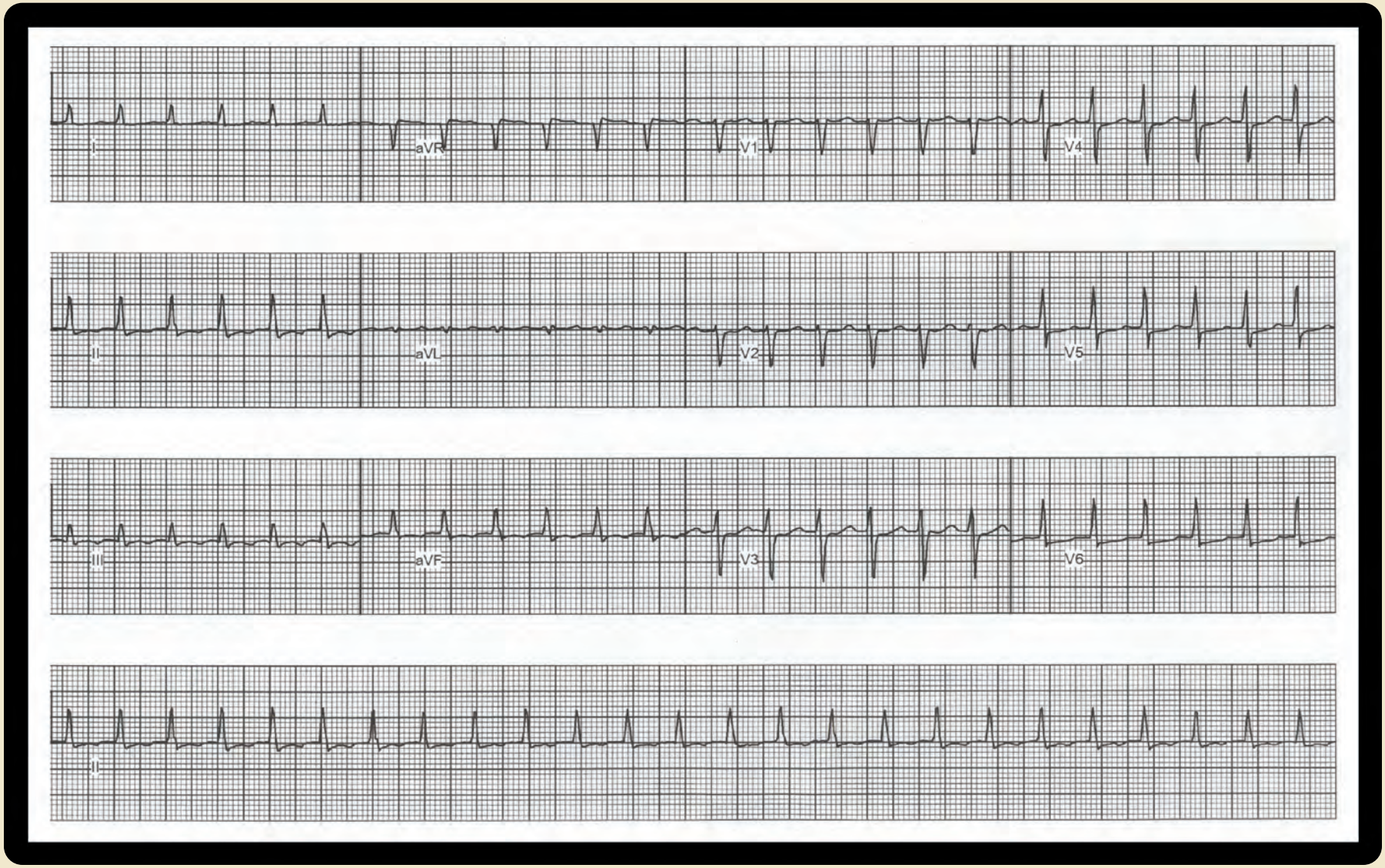
By contrast, if atrial fibrillation (AF) is the confirmed diagnosis, the diagnostic portion of the study will be very minimal to absent, and the ablation will be started after the appropriate setup.

The catheter setup and diagnostic techniques employed are unique and specific to the situation, but the SVT study forms the model for all studies and will be presented in more detail as such in the text to follow.

Before We Begin an SVT Study *continued*

In this example, the clinical diagnosis was unclear so a full diagnostic study was performed. AV nodal reentrant tachycardia (AVNRT) was the final diagnosis. Remember, just because the patient demonstrates AVNRT does not eliminate the possibility that a substrate for a different tachycardia (eg, accessory pathway [AP]) exists. Our full diagnostic study ruled this out.

Commentary: **Entrainment (mentioned on page 1) is covered later in this guide.**


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Unit 1:
The Basics

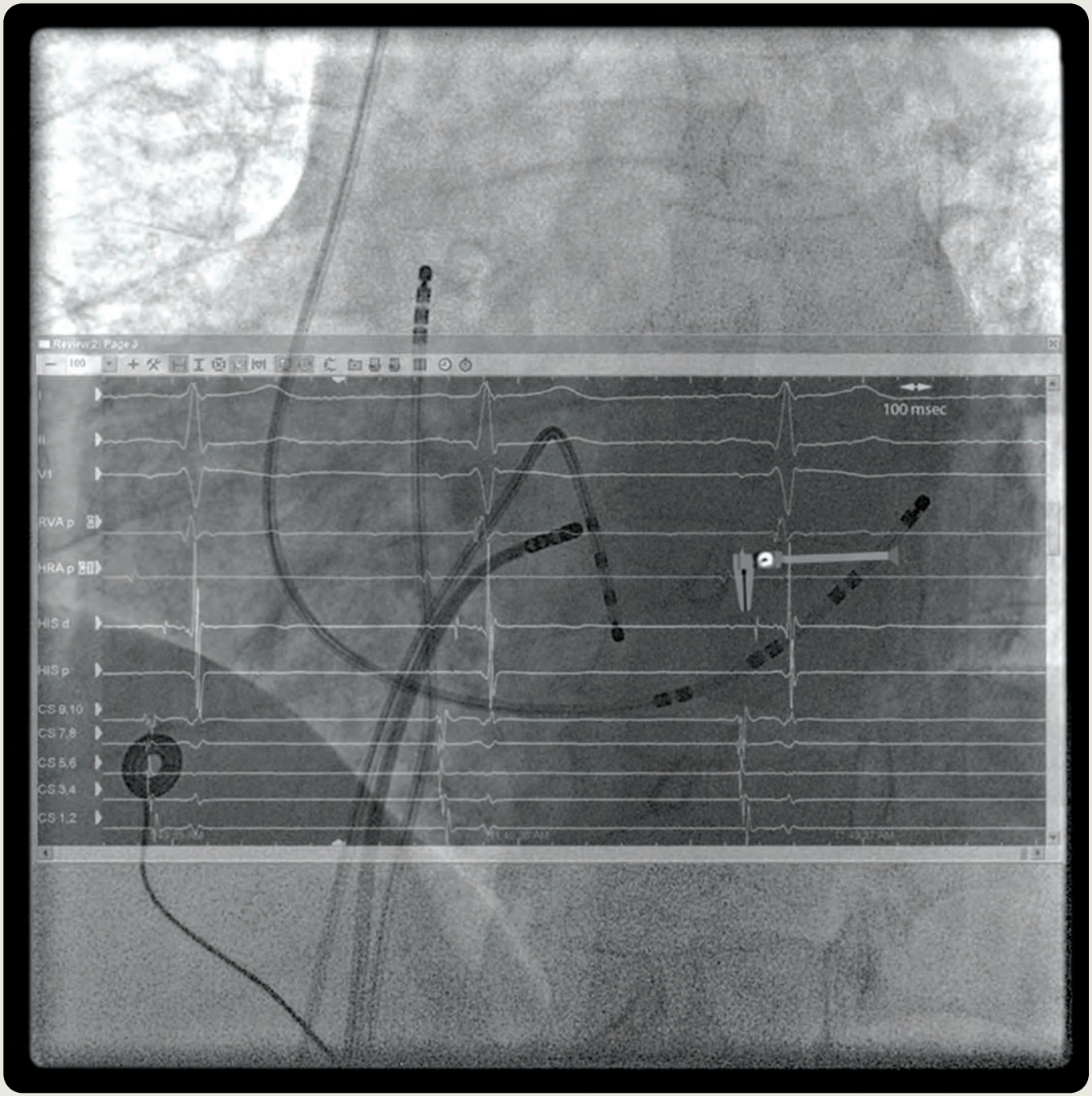
In this unit we look at catheter placement, the computer system, signal processing, signal sequence in sinus rhythm, basic conduction intervals, and two basic but critical tissue characteristics:

- Conduction velocity
- Refractoriness

Understanding these concepts is critical to understanding the mechanisms of most tachycardias.

Starting on page 18, you will find our methodology and the sequence of cardiac stimulation we use in a routine diagnostic study for SVT. Examining the normal sequence of signal conduction and measuring various basic intervals establishes a baseline for the patient. These measurements will vary depending on the clinical problem.

UNIT 1 OUTLINE


Review2 Page 3
100 msec
I
II
V1
RVA p
HRA p
HIS d
HIS p
CS 9,10
CS 7,8
CS 5,6
CS 3,4
CS 1,2

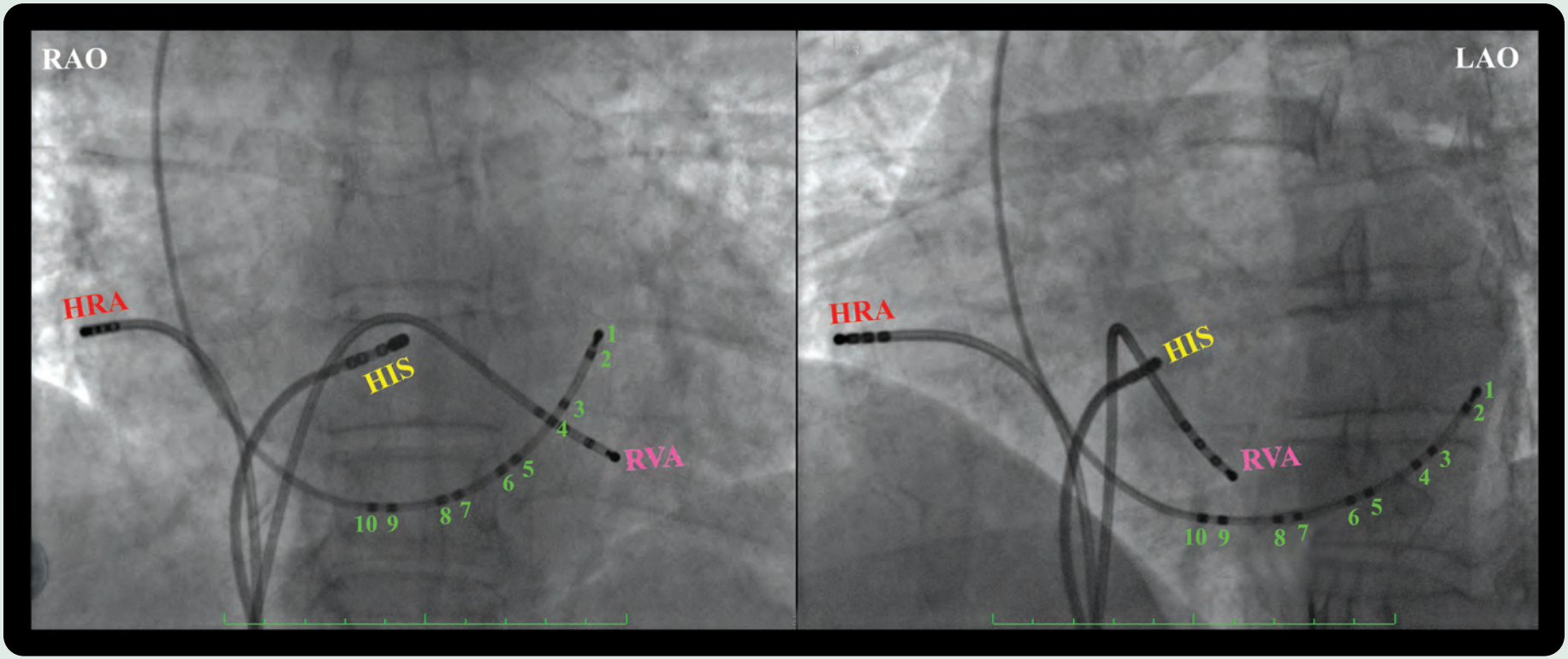
1. Catheter Placement

Multi-electrode catheters are exclusively used in clinical EP. Electrical signals are detected and recorded from the individual electrodes and from adjacent electrodes (bipole). By convention, the most distal electrode (at the tip of the catheter) is numbered 1. Subsequent electrodes are numbered in a sequential fashion, as shown in the coronary sinus (CS) decapolar catheter here (labeled in green).

The high right atrial (HRA) catheter is labeled in red. It will record an atrial (A) signal on the red HRA channel on our tracings throughout this book. The HIS catheter is labeled in yellow. It is positioned in the region of the atrioventricular (AV) node guided by both the signals and fluoroscopic images. Since the AV node is in close proximity to both the atria and the ventricles, it will have both an A signal as well as a ventricular (V) signal. In addition, the HIS catheter will have a third electrogram (EGM) representing the bundle of His, which is the electrical conduit from the AV node to the specialized conduction system within the ventricles. This EGM is often referred to simply as an "H." So the properly positioned HIS catheter will display three EGMs—atrial (A), HIS (H), and ventricular (V)—on our yellow HIS channel. The right ventricular apex (RVA) catheter is labeled in magenta. It will display a V signal on our magenta RVA channel.

The CS catheter is labeled in green. The CS is the vein situated between the left atrium and the left ventricle. Therefore, this catheter, for the most part, identifies signals from the left atrium and left ventricle when properly positioned within the CS. It is our custom that CS 9-10 should be positioned at the left edge of the spine in the left anterior oblique (LAO) projection. This "neutral" position places these poles at the junction of the right atrium and the CS opening. CS 1-2 will display signals from the lateral aspect of the left atrium. Most CS signals will have two components, A and V, on our green CS channel. Currently, we use a 10-pole (decapolar) CS catheter.

Commentary: **Atrial activation sequence proceeds from proximal to distal in the normal heart assuming a normally positioned (neutral or central) CS catheter. If the CS catheter is not in the neutral position, atrial activation during ventricular pacing may appear eccentric and thus be very misleading (ie, mimicking APs). If the CS catheter is advanced too far into the CS, the signals may appear as a chevron (ie, CS 1-2 activation is as early as CS 9-10 activation). This early activation is due to detection of Bachmann's bundle.**


RAO
HRA
HIS
RVA
1
2
3
4
5
6
7
8
9
10
LAO
HRA
HIS
RVA
1
2
3
4
5
6
7
8
9
10

4. Signal Sequence in Sinus Rhythm

1st: The HRA A electrogram (AEGM) is the earliest since this catheter is closest to the sino-atrial (SA) node. Note that it corresponds to the onset of the surface P-wave.

2nd: HIS A is the next AEGM to appear in sequence. The wave of atrial depolarization has successfully arrived at the AV node.

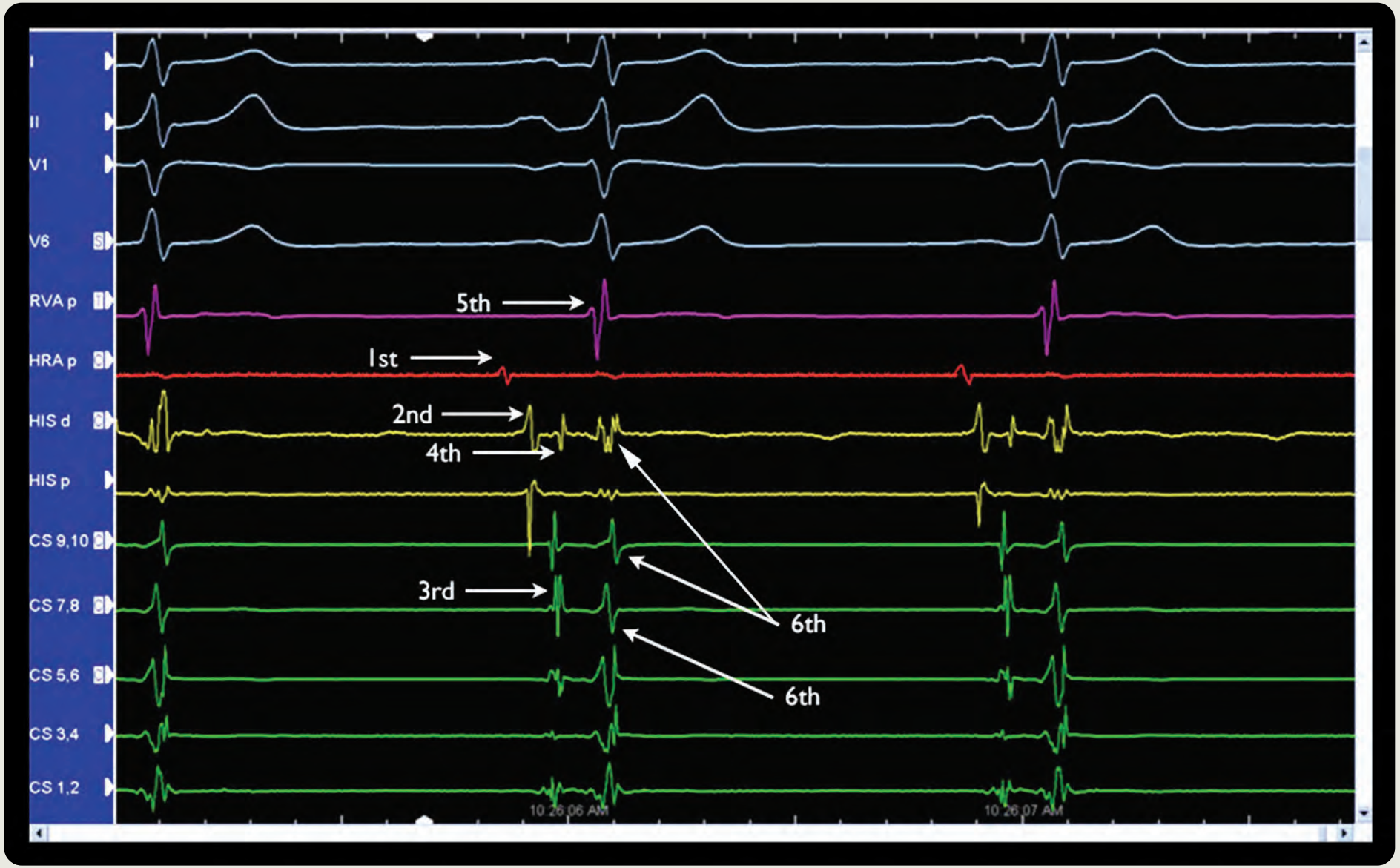
3rd: CS A signals appear next as the wave spreads from the proximal CS distally into the left atrium.

4th: The H deflection on the HIS catheter is next. This indicates that the wave has propagated over the atrioventricular (AV) node and arrived at the bundle of His.

5th: The RVA V electrogram (VEGM) is generally next as it is near to the right bundle branch exit, which is usually the first part of the ventricles to be activated. Note that it is before the HIS V or CS V signals since the wave of depolarization travels down the bundle branches, past the apex, and arrives at the base of the ventricles last.

6th: The HIS channel V and the CS V's are the last signals to occur. Remember that the HIS and CS catheters are positioned at the base of the ventricles.

Commentary: Take careful note that the RVA V signal is ahead of the HIS V and CS V signals in sinus rhythm since the right ventricle is activated early in normal depolarization without bundle branch block. On the other hand, the HIS or CS V signal may precede the right ventricle with right bundle branch block (RBBB), where the left ventricle will be activated before the right. This relationship could change if an AP is present and may give a clue as to the insertion site of the AP into the ventricles.


I
II
V1
V6
RVA p
HRA p
HIS d
HIS p
CS 9,10
CS 7,8
CS 5,6
CS 3,4
CS 1,2
5th
1st
2nd
4th
3rd
6th
6th
10:26:06 AM
10:26:07 AM

5. Basic Conduction Intervals

Routine baseline measurements should be made and recorded for every patient undergoing an EP study. The names and normal ranges are as follows:

- **P-A interval**: Measure from the onset of the P-wave on the surface ECG to the rapid deflection of the A-wave on the HIS channel. (See the red calipers.) The P-A interval is usually about 35 to 45 msec. This is the transright atrial conduction time, that is, the approximate time it takes for the electrical signal to travel from the SA node to the AV node.
- **A-H interval**: Measure on the HIS channel as the A signal to the onset of the H deflection. (See the white calipers.) The A-H interval is usually about 70 to 80 msec. This is the transnodal conduction time, or the time it takes for the electrical signal to travel through the AV node.
- **H-V interval**: Measure from the onset of the HIS deflection to the earliest onset of ventricular activation on any channel available, either intracardiac or ECG. (See the green calipers.) It is usually the onset of the QRS complex. The H-V interval is about 35 to 45 msec. This is the His to ventricular activation time, that is, the time it takes for the electrical signal to travel from the His bundle to the ventricles.

Therefore, on a surface ECG, these three measurements make up the PR interval:

PR interval = P-A + A-H + H-V
= 40 + 80 + 40
= 160 msec

Commentary: Careful measurement of these basic intervals is critical. You must know the baseline A-H in order to recognize subsequent AV nodal decrement and potential "jumps." Additionally, a long H-V interval indicates distal His-Purkinje disease.

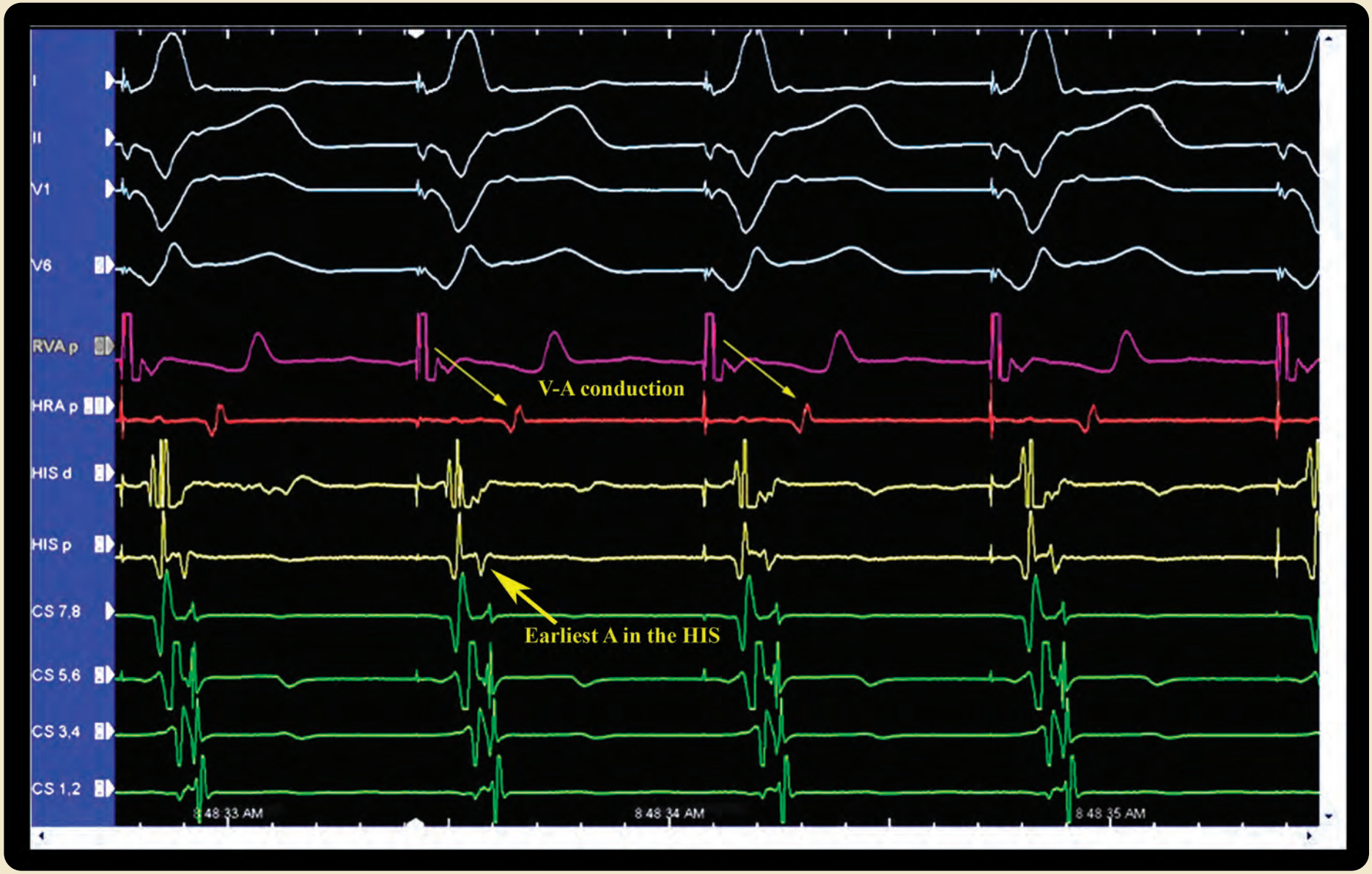

I
II
V1
RVA p
HRA p
HIS d
HIS p
CS 9,10
CS 7,8
CS 5,6
CS 3,4
CS 1,2
100 msec
11:49:35 AM
11:49:36 AM
11:49:37 AM

7. Supraventricular Tachycardia Diagnostic Study
Incremental Ventricular Pacing

Our diagnostic study begins with incremental ventricular pacing to establish the retrograde block cycle length (Wenckebach cycle length) of the A-V conduction system. Pacing begins 100 msec faster than the patient's intrinsic rate and decreases by 10 to 20 msec every eight beats until 1:1 ventriculo-atrial (V-A) conduction is no longer maintained. We do not pace faster than 250 msec because ventricular tachycardia (VT) or ventricular fibrillation (VF) may be induced at higher rates. There are four basic questions to consider:

1. **Did the pacing capture the ventricle?** Inspect the ECG and EGMs to confirm that the pacing stimulus did indeed capture the ventricle.
2. **Is there V-A conduction?** Look at the RVA channel (magenta) and the HRA channel (red) and establish the relationship between ventricular activation and atrial activation. This example shows each VEGM followed by an AEGM, or 1:1 conduction (*arrows*). At any given pacing rate, there may be no V-A conduction at all (the A signals will have no relationship to the V signals) or there may be V-A conduction that is 2:1 or some other ratio.
3. **What is the pattern of retrograde atrial activation?** In the normal heart, retrograde conduction to the atrium occurs via the AV node so that atrial activation will occur here first. This is referred to as "central" or "concentric" atrial activation and is seen as the yellow HIS A being the first A seen following the large V. It is generally slightly ahead of any CS A. The proximal CS is close to the AV node and is thus activated next. That is, the CS AEGMs activate at CS 7-8 first, followed by CS 5-6, CS 3-4, and finally CS 1-2. This central pattern is the expected pattern when retrograde conduction is through the AV node. When the earliest A signal is not at the HIS A, the pattern is referred to as "eccentric" atrial activation, which suggests that there may be another connection between the ventricles and the atrium other than the AV node (ie, an AP).
4. **Does the V-A time stay constant or become prolonged (decrements) as we increase the pacing rate?** The expected behavior of the AV node is that conduction slows at higher pacing rates. Therefore, at higher rates, the V-A time should be longer if conduction is going through the AV node (due to its decremental properties). This is readily obvious on the CS channels. Most APs do not decrement. Therefore, the absence of V-A time prolongation at higher pacing rates suggests conduction using an AP.

Commentary: **The fifth question is "What is the V-A time?" A long V-A time could indicate a retrograde slow pathway or a decremental AP. Since both demonstrate a long V-A time, making an accurate diagnosis of the tachycardia circuit may be quite challenging! More pacing maneuvers are required.**


I
II
V1
V6
RVA p
HRA p
HIS d
HIS p
CS 7,8
CS 5,6
CS 3,4
CS 1,2
V-A conduction
Earliest A in the HIS
8 48 33 AM
8 48 34 AM
8 48 35 AM

7. Supraventricular Tachycardia Diagnostic Study *continued*
Retrograde Wenckebach

This tracing illustrates the loss of 1:1 V-A conduction. The pacing interval at which this occurs is referred to as the retrograde block cycle length or the retrograde Wenckebach point.

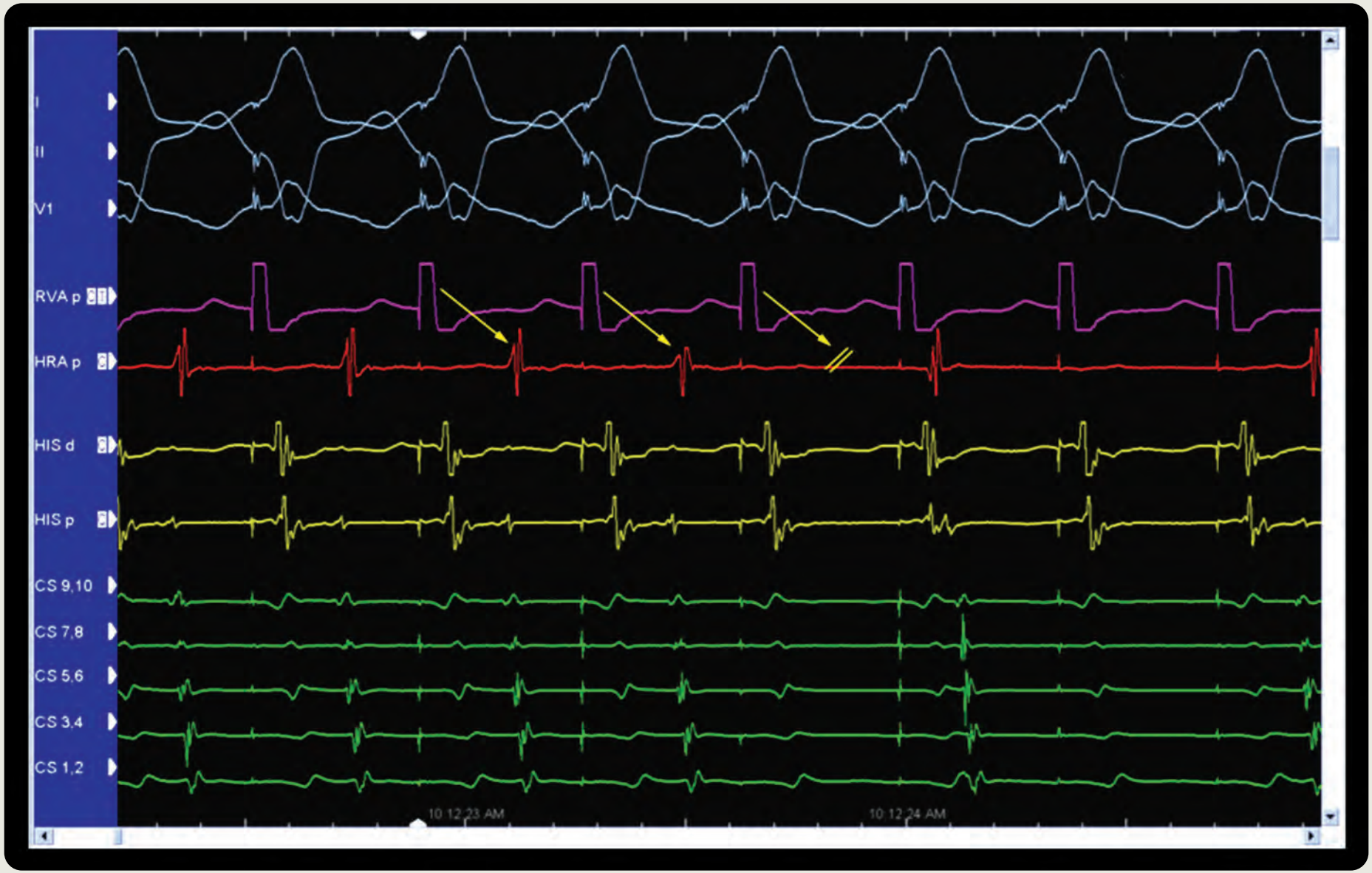
Notice that the V capture in the magenta RVA channel is followed by a red A in the HRA channel for the first three paced beats. The fourth paced beat (*third arrow*) shows a magenta V but no associated red A. This loss of an HRA A signal confirms that we have reached the Wenckebach point of the retrograde A-V conduction system.

Also notice in the yellow HIS and green CS channels that the time from the V signal to the A signal has lengthened compared with the tracing on the previous page, implying decrement in the AV node as we increase our pacing rate. The level of block with incremental pacing is generally at the AV node.

This is normal AV nodal physiology.

Commentary: **A common mistake is not appreciating minimal decrement. You must measure the V-A time at the beginning of this pacing maneuver and monitor throughout the run as well as at the end. You measure a V-A time from the onset of ventricular activation to the earliest A signal on any channel. Absence of decrement may be seen with the normal V-A conduction system but should raise the suspicion of an AP (Wolff-Parkinson-White [WPW] syndrome).**

Video 1.1: Incremental V Pacing


I
II
V1
RVA p
HRA p
HIS d
HIS p
CS 9,10
CS 7,8
CS 5,6
CS 3,4
CS 1,2
10:12:23 AM
10:12:24 AM

8. AV Nodal Reentrant Tachycardia
Typical AVNRT Pathways

AVNRT is possible in patients who have two routes over the AV node (dual AV nodal pathways). The exact anatomic substrate and location of these two pathways is not known with certainty, but it is undoubtedly true in theory and provides an excellent working concept.

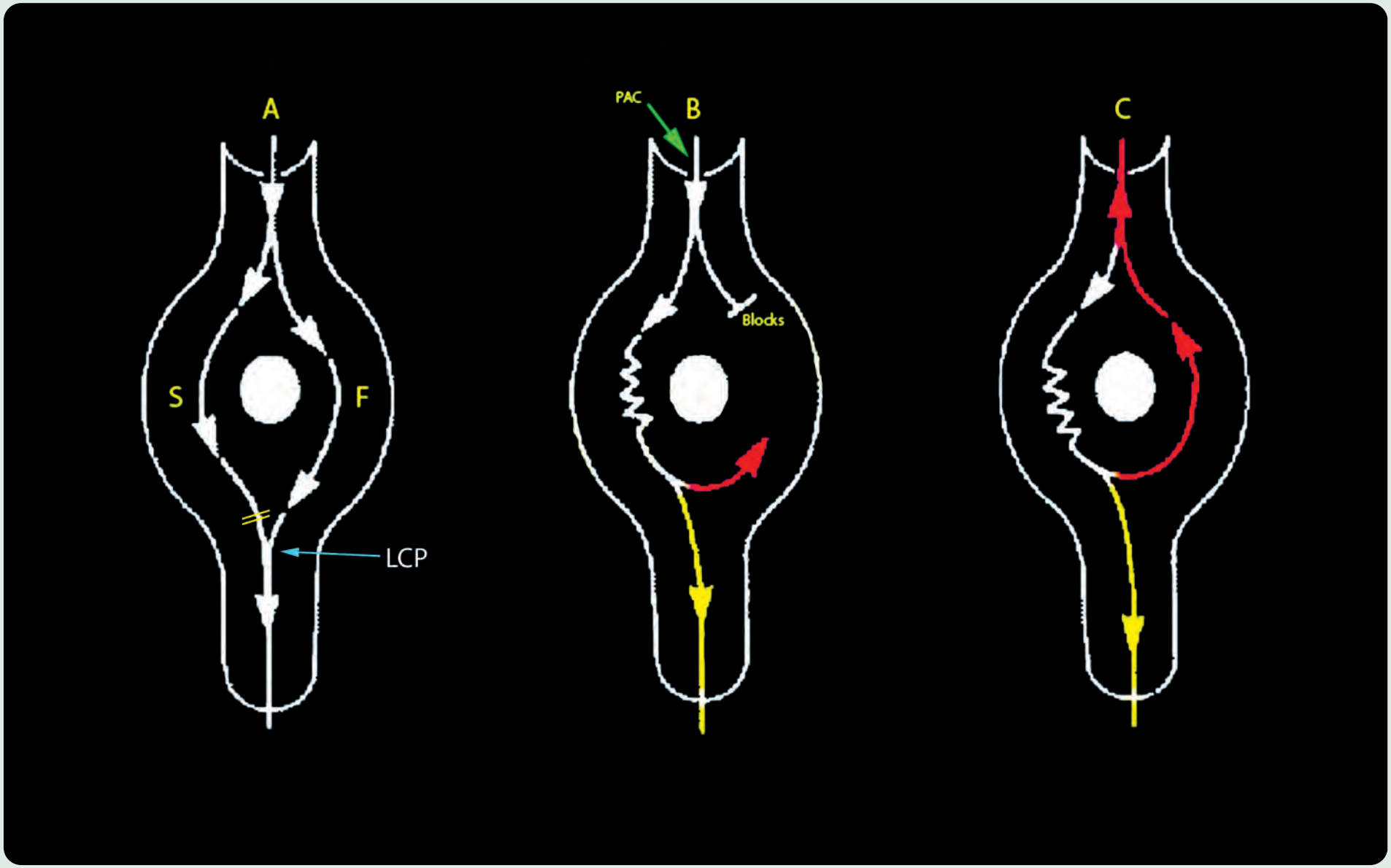
Panel A shows a representation of the AV node with two routes through it, a fast pathway (F) and a slow pathway (S). This is known as longitudinal dissociation. During sinus rhythm, the wave of depolarization will travel quickly down the fast pathway to depolarize the ventricles. The same wave also travels down the slow pathway but finds the lower common pathway (LCP) refractory due to the preceding wavefront over the fast pathway.

Panel B introduces a premature atrial contraction (PAC) (S2) that blocks in the fast pathway due to the fast pathway's longer refractory period. Therefore, the S2 conducts to the ventricle using the slow pathway. Conduction using the slow pathway rather than the fast pathway means that there will be a sudden lengthening of the A-V interval (more specifically, lengthening of the A-H interval) or a jump.

As the wave of depolarization continues toward the ventricles, it also has the opportunity to travel retrogradely back up the fast pathway (*red arrows* in panels B and C) since the fast pathway has had sufficient time to recover.

This completes the circuit and initiates AVNRT, which is the continual "spinning" down the slow pathway and up the fast pathway.

Commentary: **This is a good stylistic concept, but there are probably many mechanistic variants. For example, the diagram shows a common upper pathway entering the AV node when there is considerable evidence that there are two upper pathways entering the nodal area distinctively. An important practical point for ablation is that the slow pathway usually enters the AV node inferiorly near the CS orifice.**


A
S
F
LCP
PAC
B
Blocks
C

14. Mechanisms of Tachycardia

Tachycardias are caused by one of three underlying mechanisms: reentry, enhanced automaticity, and triggered activity. Conceptually, it helps to categorise these mechanisms into one of two major categories:

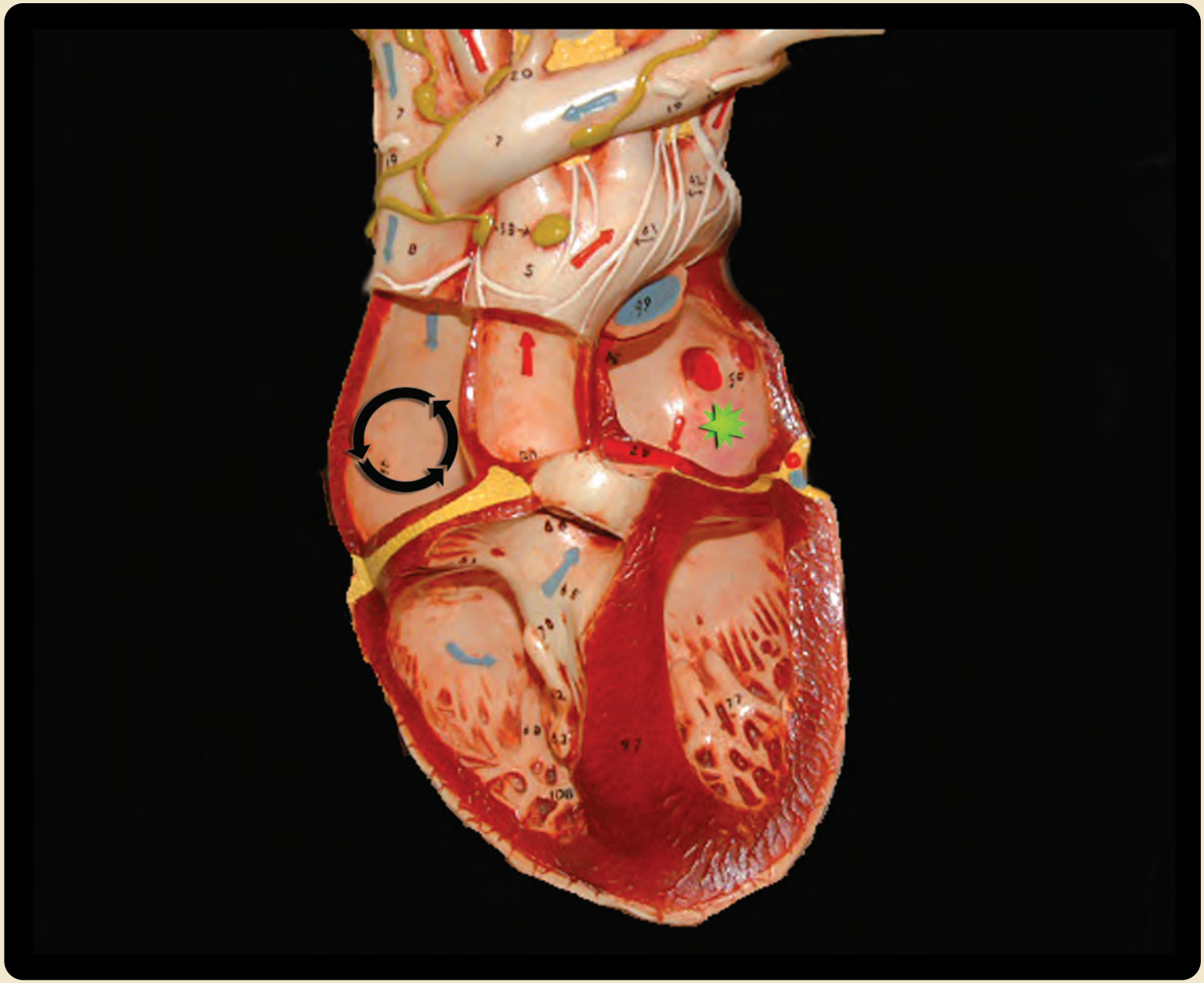
- Macro-reentrant circuits
- Focal tachycardias

Macro reentry is essentially a self-perpetuating loop of electrical activity that often incorporates a large area of the atrium, ventricle, or both. In a reentrant circuit, there is a leading "head" of depolarization and a trailing "tail" of recovery where the head is constantly chasing the tail (depicted by the *circular black arrows*). Examples include atrial flutter, AVNRT, and AVRT. These tachycardias require a zone of fast conduction and a zone of slow conduction.

In contrast, focal tachycardias emanate from a point source in the heart (depicted by the *green star*) and are not dependent on a large circuit of electrical activity. A typical example of a focal tachycardia is atrial tachycardia. Two possible mechanisms of a focal tachycardia are automaticity and triggered activity, both of which lead to abnormal spontaneous depolarizations of a myocardial cell.

Automaticity is a normal property of some specialized cells such as the sinus node. Automaticity can be an unexpected property in abnormal cells such as those found in a focal atrial tachycardia. Triggered activity is the abnormal spontaneous depolarization of cells during or immediately following the normal recovery period of the cell (called "after-depolarizations"). Triggered activity can be initiated by excess calcium in the cell.

Commentary: **The mechanism of the tachycardia is relevant to the EP study because it affects the manner in which the tachycardia is induced and studied. Macro-reentrant rhythms are more readily induced using extra-stimuli pacing, whereas triggered or automatic rhythms are less readily induced using pacing maneuvers.**



30. Vector Basics

To understand cardiac electrical axis, it is essential to understand vectors. A vector is a graphical representation of the *magnitude* and *direction* of an electrical current.

As an analogy, before buying a car that's parked in the showroom, you look at it from a variety of angles to get a complete 3D picture. The car's position does not change, but your vantage point does. Similarly, the heart's position does not change, but we have the ability to look at it from a variety of angles. This "ability" comes in the form of the 12-lead ECG. The ECG is simply looking at the stationary cardiac vector from a variety of angles.

Hypertrophy increases the magnitude of the vector and pulls it in the direction of the hypertrophy. Infarction on the other hand, decreases the magnitude of the vector and decreases the pull on the vector in the direction of the infarction.

Conduction blocks or delays will change the direction of the vector. If the sequence of ventricular activation is altered due to a delay in a fascicle, the overall electrical axis will be altered.

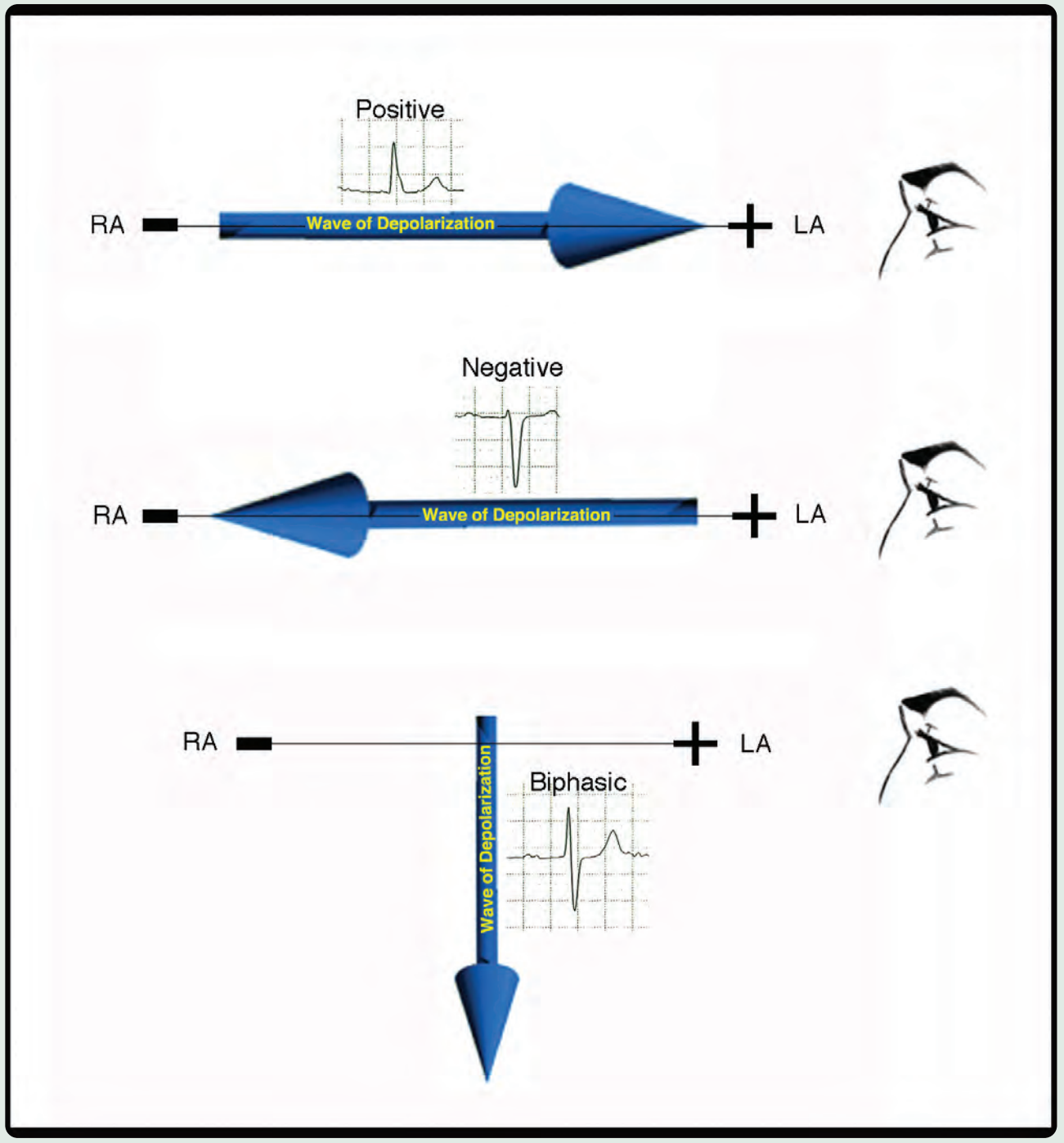
Vector Principles

Scenario 1: If a wave of depolarization is traveling parallel to and in the same direction as lead 1, you observe a very positive QRS. If the wave is not quite parallel but still in the same general direction as lead 1, the amplitude of the QRS will be reduced, but still positive.

Scenario 2: If a wave of depolarization is traveling parallel, but in the opposite direction to lead 1, you observe a very negative QRS. If the wave is not quite parallel but still generally in the opposite direction to lead 1, the amplitude of the QRS will be reduced but still negative.

Scenario 3: If a wave of depolarization is traveling perpendicular to lead 1, you observe a biphasic QRS. The amplitude of the biphasic deflection varies markedly due, in part, to the gain on the ECG machine.

Commentary: **On our EP monitor, we routinely use lead 1, lead 2, and V1. These leads give us 3 dimensions. Lead 1 looks from right to left. Lead 2 looks from superior to inferior. Lead V1 looks from posterior to anterior in the horizontal plane.**


Positive
RA
Wave of Depolarization
LA
Negative
RA
Wave of Depolarization
LA
RA
LA
Wave of Depolarization
Biphasic