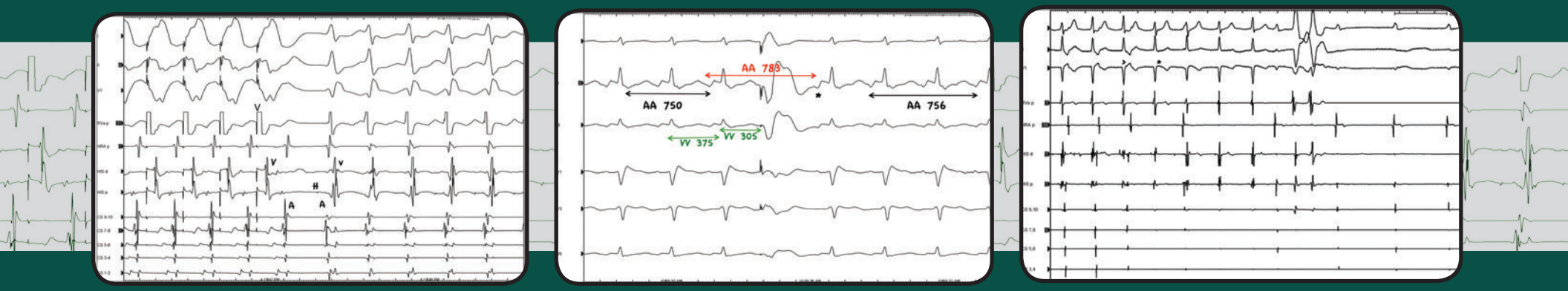


ELECTROPHYSIOLOGICAL MANEUVERS

FOR ARRHYTHMIA ANALYSIS

WRITTEN AND EDITED BY: **GEORGE J. KLEIN**

LORNE J. GULA - PETER LEONG-SIT - JAIMIE MANLUCU
PAUL D. PURVES - ALLAN C. SKANES - RAYMOND YEE

cardiotext

Electrophysiological Maneuvers for Arrhythmia Analysis

Written and Edited By:

George J. Klein, MD, FRCP(C); Professor of Medicine,
Division of Cardiology, Western University, London, Ontario, Canada

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/electrophysiological-maneuvers

Comments, inquiries, and requests for bulk sales can be directed to the publisher at: info@cardiotextpublishing.com.

This book is intended for educational purposes and to further general scientific and medical knowledge, research, and understanding of the conditions and associated treatments discussed herein. This book is not intended to serve as and should not be relied upon as recommending or promoting any specific diagnosis or method of treatment for a particular condition or a particular patient. It is the reader's responsibility to determine the proper steps for diagnosis and the proper course of treatment for any condition or patient, including suitable and appropriate tests, medications, or medical devices to be used for or in conjunction with any diagnosis or treatment.

Due to ongoing research, discoveries, modifications to medicines, equipment and devices, and changes in government regulations, the information contained in this book may not reflect the latest standards, developments, guidelines, regulations, products, or devices in the field. Readers are responsible for keeping up to date with the latest developments and are urged to review the latest instructions and warnings for any medicine, equipment, or medical device. Readers should consult with a specialist or contact the vendor of any medicine or medical device where appropriate.

Except for the publisher's website associated with this work, the publisher is not affiliated with and does not sponsor or endorse any websites, organizations, or other sources of information referred to herein.

The publisher and the authors specifically disclaim any damage, liability, or loss incurred, directly or indirectly, from the use or application of any of the contents of this book.

Unless otherwise stated, all figures and tables in this book are used courtesy of the authors.

Library of Congress Control Number: 2014937499

ISBN: 978-1-935395-89-8
eISBN: 978-1-935395-15-7

Printed in the United States of America 4 5 6 7 8 9 10 20 19 18 17

Table of Contents

Contributors

Written and Edited By:

George J. Klein, MD, FRCP(C); Professor of Medicine, Division of Cardiology, Western University, London, Ontario, Canada

Contributors:

Lorne J. Gula, MD, FRCP(C); Associate Professor of Medicine, Division of Cardiology, Western University, London, Ontario, Canada

Peter Leong-Sit, MD, FRCP(C); Assistant Professor of Medicine, Division of Cardiology, Western University, London, Ontario, Canada

Jaimie Manlucu, MD, FRCP(C); Assistant Professor of Medicine, Division of Cardiology, Western University, London, Ontario, Canada

Paul D. Purves, BSc, RCVT, CEPS; Senior Electrophysiology Technologist, Cardiac Investigation Unit, London Health Sciences Centre, London, Ontario, Canada

Allan C. Skanes, MD, FRCP(C); Professor of Medicine, Division of Cardiology, Western University, London, Ontario, Canada

Raymond Yee, MD, FRCP(C); Professor of Medicine, Director of Arrhythmia Service, Division of Cardiology, Western University, London, Ontario, Canada

Foreword

While the field of electrophysiology has grown as a discipline, much of the focus of current trainees has been on interventional electrophysiology. There has been an erosion of education related to the underpinnings of electrophysiology: pathophysiology and mechanisms of arrhythmias. *Electrophysiological Maneuvers for Arrhythmia Analysis* is a unique attempt to organize, in a practical way, methodologies that can be used to understand tachycardia mechanisms and origins. There is no other text that aims to achieve these goals. Dr. Klein is one of the senior clinical electrophysiologists in the world and a world-class educator. This book imparts Dr. Klein's vast experience in the field in which he has made seminal contributions, as well as his expertise as an educator.

One of the attributes of Dr. Klein's approach to teaching electrophysiologic principles is that he relates the physiology to specific clinical problems. He has chosen a number of coauthors, with whom he has shared years of experience, to

help provide a concise, practical, and easily understandable guide to allow physicians to approach electrophysiologic problems effectively. Despite the fact that many of these concepts are difficult, the authors have been able to make the explanations of the concepts clear and simple. They explain how stimulation, pharmacologic and physiologic perturbations can resolve important questions related to the differential diagnosis of tachycardia mechanisms. Their approach to analyzing arrhythmias is systematic and straightforward. Clinical examples are given for all the "maneuvers." The benefits and limitations of each are addressed in great detail from the opening chapter, in which Dr. Klein provides an overview of the book. The remaining chapters discuss the methods used to define sites of origin of arrhythmia, the presence or absence of preexcitation, the role of bypass tracts with an AV node in arrhythmias, and how to determine where unexpected signals arise (e.g., pulmonary vein vs. left atrial appendage).

In my opinion, this book should be on the shelf of every electrophysiologist trainee as well as every clinical cardiac electrophysiologist. It is a classic, like its editor. Dr. Klein deserves high praise for organizing his and his colleagues' clinical experiences and thought processes into a concise, practical text that should be part of all training programs in electrophysiology.

—Mark E. Josephson, MD

Chief, Division of Cardiovascular Medicine; Director, Harvard-Thorndike Electrophysiology Institute and Arrhythmia Service, Beth Israel Deaconess Medical Center; Herman Dana Professor of Medicine, Harvard Medical School, Boston, Massachusetts

Preface

The last two decades have witnessed an explosion of technology to facilitate ablation of a wide variety of arrhythmias. Much of this has been in sophisticated imaging technologies and mapping of arrhythmias. Notwithstanding this, there remains an integral role for "traditional" electrogram (EGM) analysis for the simple reason that it is useful to understand the mechanism of a tachycardia to plan an intelligent ablation strategy.

The key to understanding the mechanism of a complex tachycardia often lies in watching its behavior in the face of perturbations, and this is the fundamental rationale underpinning the "electrophysiogical maneuver." To this end, there have been many detailed and lucid publications describing these, and the purpose of this publication is not to create an encyclopedic volume of them. Our laboratory has been training electrophysiology fellows for more than 30 years, and we have watched many highly gifted (and some not so!) trainees pass

through. It is our view that the key to understanding electrophysiologic data lies in an understanding of a few relatively simple unifying concepts as we detail in chapter 1. The task is then to perform the maneuver properly and to apply a systematic approach to the understanding of what is observed. Understanding the data should not depend on an intuitive ability to visualize all complexities at a glance. We present typical examples and scenarios to illustrate this systematic approach to make the diagnosis and hopefully avoid the diagnostic pitfalls.

Abbreviations

AF	atrial fibrillation
AP	accessory pathway
AT	atrial tachycardia
AV	atrioventricular
AVN	AV node
AVNRT	AV nodal reentrant tachycardia
AVRT	AV reentrant tachycardia
CL	cycle length
CS	coronary sinus
EGM	electrogram
EP	electrophysiology
ERP	effective refractory period
HB	His bundle
HRA	high right atrium; high right atrial electrogram

JT	junctional tachycardia
LBB	left bundle branch
LBBB	left bundle branch block
LIPV	left inferior pulmonary vein
LSPV	left superior pulmonary vein
LV	left ventricle
ms	millisecond
PCL	pacing cycle length
PPI	postpacing interval
PVC	premature ventricular contraction
PV	pulmonary vein
RBB	right bundle branch
RBBB	right bundle branch block
RIPV	right inferior pulmonary vein
RSPV	right superior pulmonary vein
RV	right ventricle
RVA	right ventricular apex
S, St, Stim	stimulus
SA	sinoatrial
SVT	supraventricular tachycardia
TCL	tachycardia cycle length
TVA	tricuspid valve annulus
VA	ventriculoatrial
VF	ventricular fibrillation
VT	ventricular tachycardia
WPW	Wolff-Parkinson-White

1

Principles of the Electrophysiological Maneuver

Observations during ongoing tachycardia or during induction or termination of the arrhythmia usually expose the mechanism of a given arrhythmia during electrophysiologic study. Nonetheless, there may not be a diagnostic observation that definitively distinguishes all possibilities. In such instances, it is necessary to invoke one or more key interventions that might be expected to arbitrate among the diagnostic possibilities for a given arrhythmia.

This might be a stimulation maneuver (usually), pharmacological maneuver, or other. The maneuver might be during ongoing tachycardia or during sinus rhythm or pacing. The maneuver during tachycardia will be most definitive but maneuvers during sinus or paced rhythms may establish the physiological background that enhances or diminishes the probability of a given mechanism. For example, the para-His bundle pacing maneuver can establish the presence or absence of an accessory pathway (AP) and narrow the diagnostic possibilities for a given arrhythmia, although it doesn't directly prove that the AP is involved in the tachycardia.

Regardless of the tachycardia (wide QRS, atrial, supraventricular, etc.), the "drill" is always the same. That is, identify the universe of possibilities for a given dilemma and test the hypotheses with one or more maneuvers as necessary. There are clean "smoking gun" maneuvers but any maneuver is fallible for multiple reasons (operator error, ambiguous electrograms (EGMs), in diseased tissue, poor catheter access to a potentially key zone, capricious coincidence) and it is reassuring to verify by multiple means or to have a "tool box" of maneuvers.

The Importance of "Geography"

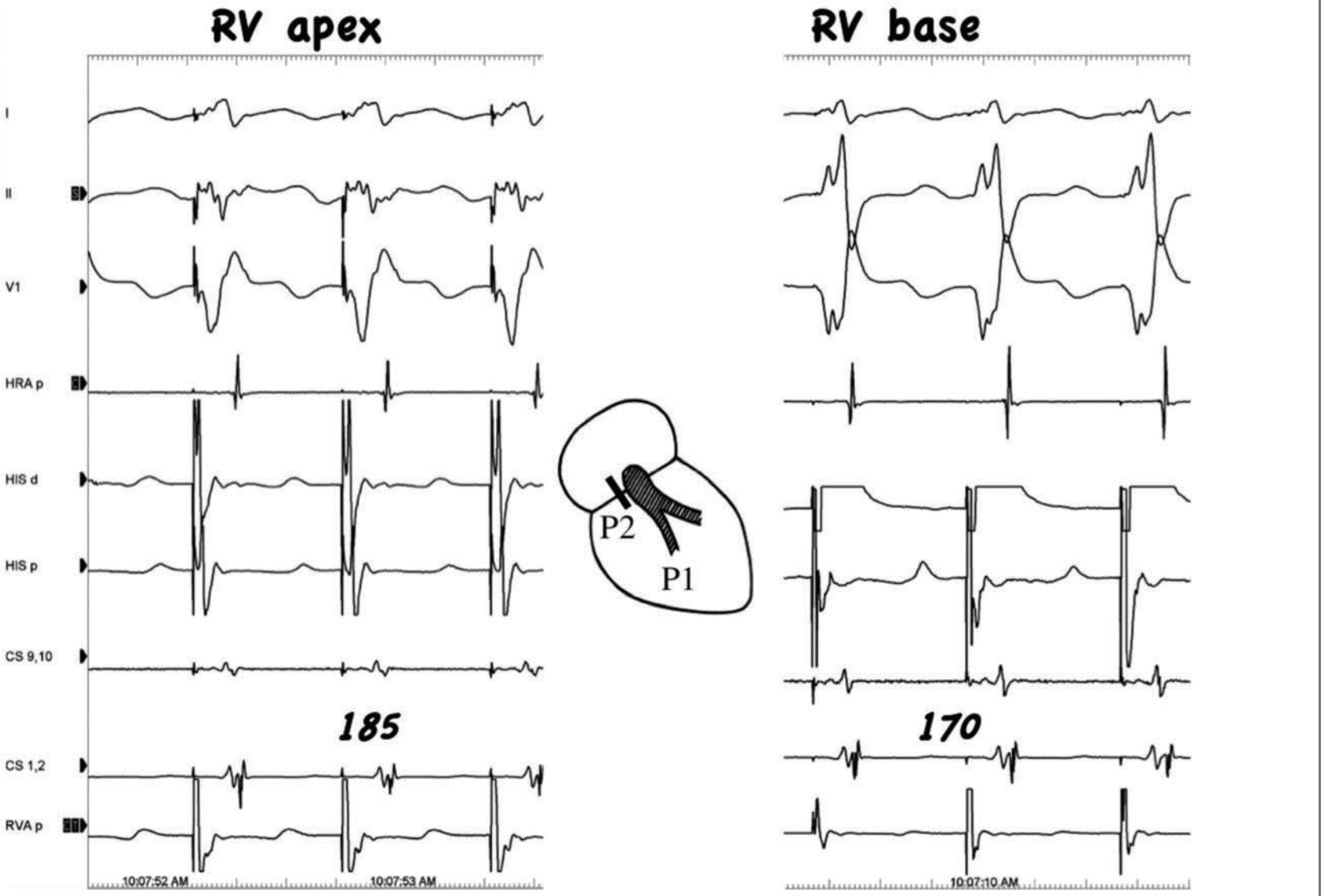
It might seem obvious but factors like distance, conduction time, conduction barriers (both relative and absolute), and substrate all need to be considered and have a critical bearing in the thought process. Two EGMs can be far apart temporally because they are physically far apart, because there is conduction slowing between them or possibly because there is a conduction barrier between them and the route from one to another involves a long detour. Simple pacing maneuvers can distinguish these, as will be illustrated subsequently.

A simple application of "geography" can be illustrated in the following example. Our problem involves a patient with persistent retrograde conduction after ablation of a manifest posteroseptal pathway. Tachycardia is no longer inducible but retrograde conduction persists and is not cycle length dependent. Is the pathway still there? What simple maneuver can be done to test this relatively common dilemma? The ventriculoatrial (VA) conduction time, given a constant cycle length and constant atrial sampling site, will vary at different ventricular pacing sites, being longer the farther the pacing site is from the "exit" of the ventricles to the atrium. Retrograde conduction over the normal VA conduction system enters via the distal bundle branches, that is, more apically. With a functioning AP of the usual type, VA conduction will be over the AP, and the VA time will be shortest from the ventricular pacing site closest to the atrioventricular (AV) ring at the location of the AP.

This is illustrated in **Figure 1.1**, which shows that VA conduction time, when pacing closer to the base of the heart nearer the expected AP site P2, provides a shorter VA time (right panel) than pacing from the apical region P1 (left panel). This is most compatible with the continuing presence of an AP, which was the case. One would expect the opposite, if retrograde conduction were proceeding over the normal AV conduction system of which entrance is more apical (i.e., the distal bundle branches). Of course, there will be other ways to verify this, as will be evident in subsequent chapters.

It is timely here to consider the possible pitfalls of this specific maneuver, since similar ones apply for virtually any pacing maneuver. Consider what these might be. Remember that proof of the existence of the AP doesn't mean that it is involved in any given tachycardia. Consider the possibility that the pathway is cycle length dependent and has a long conduction time, causing it to be "later" than conduction over the normal system. Consider that it might have a long refractory period such that it is not conducting at the cycle length chosen for pacing. Consider that the AV node may have a very short conduction time over the retrograde system and compete with the pathway to get to the atria such that the difference in pacing site VA is attenuated. Consider finally that the pacing catheters may also not be exactly where you think they are. This partial list emphasizes the need to exercise care and judgment in interpreting any maneuver.

Figure 1.1


RV apex
RV base
I
II
V1
HRA p
HIS d
HIS p
CS 9,10
CS 1,2
RVA p
185
170
P2
P1
10:07:52 AM
10:07:53 AM
10:07:10 AM

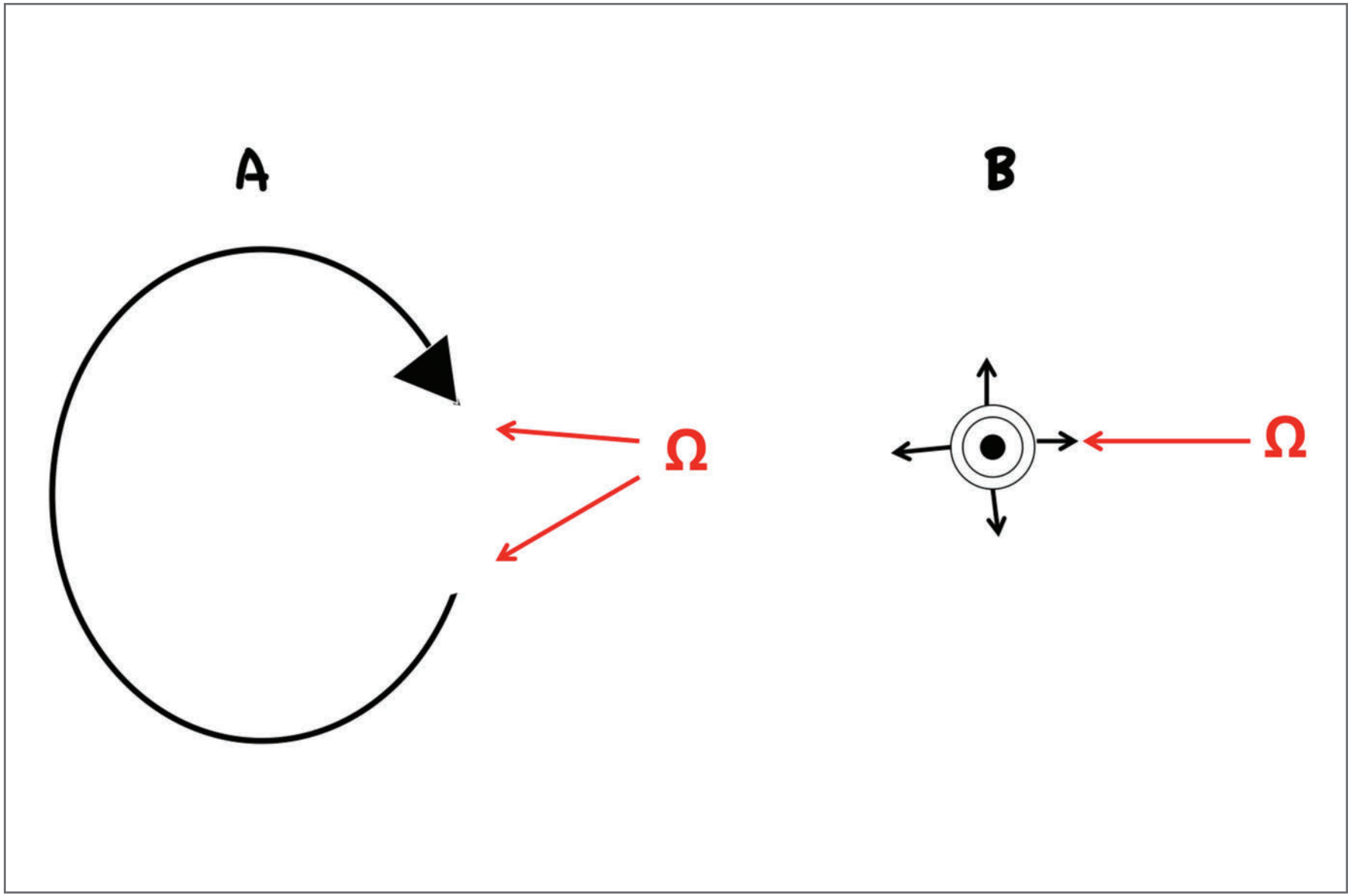
Fusion and Reset: A Key Concept

There are a plethora of EP maneuvers and "new" maneuvers continue to be published. This is much less intimidating to the novice if one considers that the great majority are derivatives of a few simple principles such as that of "geography" described previously and the concept of "fusion and reset." One might note that arguably the most compelling problem for the electrophysiologist is ascertaining the arrhythmia mechanism. This doesn't mean necessarily at a molecular or even cellular level but, for practical purposes, the electrophysiologist needs to know the mechanism well enough to define critical tissue to be ablated while limiting collateral damage to normal tissues.

Most clinical tachycardias can be thought of as being either "reentrant" or "focal" (although clearly fibrillation, torsades de pointes, and others need to be thought of differently). "Focal" is not a "mechanism" but is a descriptor for a relatively small "point source" mechanism that may be automatic, triggered, or "micro" reentrant (of secondary consideration for the purpose of ultimate ablation). This is illustrated graphically in **Figure 1.2**, which highlights the gross dissimilarity of these 2 classes of mechanism and should suggest pacing interventions to differentiate the 2 without significant difficulty.

Figure 1.2A depicts a classical reentrant circuit (in 2 dimensions here for clarity) with anatomic boundaries and an excitable gap of nonrefractory tissue between the advancing "head" and receding refractory "tail." An extrastimulus advances depolarization toward the gap, resulting in "fusion" with depolarization from the tachycardia, which may (if there is sufficient depolarization penetration by the extrastimulus) be evident on the surface ECG or intracardiac EGMs. The depolarization resulting from the extrastimulus here is unable to penetrate the circuit to conduct *antidromically* over the circuit due to refractoriness. However, the same circuit may get into the gap *orthodromically* and preexcite or advance the "tail" of the circuit. This may advance, delay, or terminate the next cycle depending on the properties of the circuit. Such a circuit is said to be "reset," although it is possible that reset is not discernable if slowing in the circuit due to prematurity balances the precocity of the extrastimulus. **One can appreciate that coincidental reset and fusion are only possible if the circuit or underlying mechanism has an excitable gap with a "separate entrance and exit."** With very rare exceptions, "fusion and reset" is only seen with "macroreentry." One can also readily predict the determinants of reset. These include size of the excitable gap (as determined by conduction time and refractoriness), proximity of the pacing site physically to the excitable gap, and physical barriers (e.g., scar) between the pacing site and the excitable gap.

Figure 1.2



Consider the same extrastimulus in the case of the focal source of tachycardia in Figure 1.2B. It is easy to envisage fusion with collision of the wave from the pacing site with the wave from the focus. However, the wave from the focus in essence creates a protective barrier of refractoriness after discharge such that the tachycardia "generator" is not susceptible to disruption (i.e., reset). That is, *both* fusion *and* reset are not possible with a focus. Conversely, one can potentially penetrate the focus (reset) but it is only when there is no ring of refractoriness around the focus from its own depolarization, i.e., one can get reset without fusion but not both at the same time.

The importance of this concept is well illustrated with a clinical example using the quintessential model of macrorentry, namely atrioventricular reentrant tachycardia (AVRT) using an accessory AV pathway as part of the circuit.

This tracing (**Figure 1.3**) demonstrates a regular, narrow QRS tachycardia with a 1:1 AV relationship. The p is negative in lead II, suggesting low to high atrial activation. Earliest atrial activation is at the proximal coronary sinus but is relatively early at the His bundle and high right atrium (HRA) EGMs. A premature ventricular contraction (PVC) from the right ventricular apex (RVA) is delivered at a time when the His bundle activation is completed, i.e., the His deflection is on time. This *delays* the next atrial cycle, a situation that is only compatible with an accessory AV pathway as the retrograde limb of an AV reentrant circuit. This is the well-known "His refractory PVC" that is a fundamental of clinical electrophysiology predicated on the fact that the subsequent atrial depolarization can't be influenced via the normal AV conduction system if the His bundle is refractory. The "reset" indicates that the extrastimulus has penetrated the circuit to alter the subsequent cycle. In the preceding example, the extrastimulus has delayed rather than advanced the next cycle since the AP in question exhibits cycle length dependent prolongation of conduction time. This is illustrated in **Figure 1.4**. Most APs have a relative constant conduction time independent of cycle length (curve C), whereas some prolong the conduction as a function of more premature coupling of the extrastimulus (curve B). Whether the reset advances (#3 for both curves), delays (#2 for curve B) or terminates (#1 for both curves), the tachycardia depends on the degree of prematurity of the extrastimulus relative to the advancing wave front to the AP.

Figure 1.3