Cardiac Arrhythmias: Electrophysiologic Techniques and Management

Leonard S. Dreifus, M.D. / Editor

CARDIOVASCULAR CLINICS
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F. A. DAVIS COMPANY, PHILADELPHIA

Cardiovascular Clinics, 16/1, Cardiac Arrhythmias: Electrophysiologic Techniques and Management

Printed in the United States of America

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Library of Congress Cataloging in Publication Data

Cardiovascular clinics. 1-
Philadelphia, F. A. Davis, 1969-
v. ill. 27 cm.
Editor: v. 1- A. N. Brest.
Key title: Cardiovascular clinics, ISSN 0069-0384.
1. Cardiovascular system—Diseases—Collected works. I. Brest, Albert N., ed.
[DNLM: W1 CA77N]
RC681.A1C27 616.1 70-6558
ISBN 0-8036-9227-7 MARC-S

Library of Congress 75[8307]

Cardiac Arrhythmias: Electrophysiologic Techniques and Management

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*Not Available

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Preface

The advancements in basic and clinical electrophysiology have been immense over the past few years. It is the purpose of this text to acquaint the clinician with the needs and clinical usefulness of electrophysiologic studies in the daily practice of cardiovascular medicine. While numerous textbooks adequately illustrate interesting typical and unusual examples of various cardiac arrhythmias, few have attempted to identify the methods and techniques of electrophysiologic studies and the actual clinical needs. The chapters have been divided into the various anatomic divisions of the specialized tissues and working myocardial structures of the heart.

The editors gratefully acknowledge the importance of the work of the many scientists who have offered so much to the understanding of electrophysiology of cardiac arrhythmias and who have contributed so generously to this text.

Leonard S. Dreifus, M.D.
Guest Editor

Editor's Commentary

One of the most exciting advances in clinical and investigative cardiology during recent years has been the growth and development of electrophysiology as a bona fide discipline. Information derived from the electrophysiology laboratory can be immediately useful in the recognition of complex rhythm disturbances, in the selection of antiarrhythmic drug therapy, and in the application of a variety of nonpharmacologic (pacemaker, other electrical, and surgical) therapies. This issue of CARDIOVASCULAR CLINICS provides a state-of-the-art examination of the electrophysiologic techniques and clinical applications that constitute current practice. I am extremely grateful to Leonard S. Dreifus for his guidance in the formulation of this issue, and both of us wish to express our gratitude to the individual authors for their exemplary contributions.

Albert N. Brest, M.D.
Editor-in-Chief

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Introduction to Clinical Electrophysiologic Studies

Eric L. Michelson, M.D., and Roberto P. Medina, M.D.

Tachy- and bradyarrhythmias are a major cause of mortality and morbidity. Although most frequently associated with coronary artery disease and cardiomyopathy, serious arrhythmias may also occur in patients with mitral valve prolapse, congenital heart disease, electrolyte imbalance, primary and secondary diseases of the sinus and atrioventricular nodes and/or the cardiac conduction system, as well as in individuals without identifiable structural abnormalities of the heart. Unfortunately, when therapeutic drug regimens are selected empirically for patients with the most malignant arrhythmias and at high risk of sudden death, without objective measures to predict efficacy, there appears to be only marginal effect on prognosis.[1,2]

The lack of uniformly effective and safe antiarrhythmic drugs and the search for a more rational approach to the management of patients at high risk of life-threatening arrhythmias have given great impetus to the recent development of safe and reliable techniques for the evaluation of the electrophysiologic properties of the heart. Routinely, the susceptibility of the heart to a variety of tachy- and bradyarrhythmias and the response to antiarrhythmic drugs and different pacing modalities can be evaluated. The question of indiscriminate use of costly resources has made the medical community the subject of increasing criticism,[3] while at the same time a physician's decision to defer costly diagnostic studies or withhold therapy may also have serious medicolegal consequences.[4] Admittedly, clinical electrophysiologic studies are invasive, potentially costly, time consuming, and not without risk.[5] Thus, their practical clinical application calls for a rational approach to the procedure and appropriate selection of patients most likely to receive benefit.

The application of invasive electrophysiologic techniques has made important contributions to our understanding of the mechanisms of arrhythmias and the mode of action of antiarrhythmic drugs. These advances are reviewed in detail in other chapters in this book. Our focus will be on the practical, clinical application of electrophysiologic testing.

The field of clinical electrophysiology began as recently as 1969 with the recording of the His bundle electrogram in human beings, using electrode catheter techniques.[6] The early years of electrophysiologic investigation were dominated by descriptive work exploring the timing of electrical activation of the different parts of the conduction system and myocardium in a variety of spontaneously occurring physiologic and pathologic conditions. The development of the technique of programmed stimulation[7] added a new perspective to the understanding of the electrophysiology of the human heart, with the possibility of stressing the conduction system, determining refractory

periods, evaluating the response of automatic pacemaker tissue to overdrive suppression, and attempting the induction of tachyarrhythmias in the laboratory. The possibility of initiating and terminating arrhythmias in a controlled situation provided a rational basis for the evaluation of therapeutic modalities in patients at risk of serious or life-threatening arrhythmias, when the tailoring of a protective pharmacologic regimen is crucial. Current developments focus on longitudinal studies using electrophysiologic testing to assess prognosis and the value of specific interventions, including drugs, devices, and antiarrhythmic surgery, as well as the introduction of noninvasive electrocardiographic signal-averaging techniques.

TECHNIQUES AND BASELINE DETERMINATIONS

Catheterization and Intracardiac Recordings[8,9]

Although some success has been reported in recording His bundle electrograms from the body surface using signal averaging, the technique is generally limited to recording spontaneous events and does not allow for the use of programmed electrical stimulation. To record intracardiac electrograms, electrode catheters are introduced into different locations in the heart, including the right atrium, coronary sinus, right atrioventricular junction (to record the His bundle electrogram), and right ventricle. More recently, left ventricular stimulation has also been used.[10] Detailed mapping studies may require recording from multiple sites simultaneously, including the left ventricle, but the introduction of two to four catheters in the right heart chambers is sufficient for most clinical applications.

The transvenous route is used, except for left ventricular stimulation. Catheters are introduced by the percutaneous Seldinger technique, most commonly using the femoral or occasionally the brachial approach. The His bundle electrogram is better recorded from the femoral approach, and the coronary sinus is more easily entered from the left brachial, subclavian, or jugular route. The performance of a venous cutdown is seldom necessary. The left subclavian or jugular approach is well suited to serial studies done over days to weeks requiring stable catheter position. Systemic heparinization is mandated only when the arterial system is entered for left ventricular stimulation, and the possible complications of the procedure are similar to those of a routine catheterization procedure. However, the possible thromboembolic complications of introducing multiple intravenous catheters may be limited by heparinization during right heart studies as well, particularly during prolonged catheterization. Size 5 to 7 French electrode catheters are advanced under fluoroscopic guidance. These catheters usually have electrodes separated by a distance of 1 cm; either bipolar, quadripolar, or hexapolar catheters are commonly used for most recording and stimulation techniques.

The intracardiac electrograms are usually displayed simultaneously with three or more surface electrocardiographic recordings on an oscilloscopic screen and recorded on a strip-chart for on-line evaluation and/or tape recorder for later analysis. The signal processor, oscilloscope, and recorder are often incorporated as a single unit, similar to those used for routine hemodynamic studies. A programmable stimulator is used for the pacing studies, capable of pacing over a wide range of cycle lengths, from several sites simultaneously, and introducing accurately timed single or multiple extrastimuli synchronized to intrinsic or paced rhythms. The stimulator is a constant current pacemaker, and the current output is usually set at two to three times the patient's late diastolic excitability threshold at a given site and with a pulse width of 1 to 2 msec.

The right atrium may be approached from any venous route, and the most common site for stimulation and recording is the high right atrium posteriorly at the junction of the superior vena cava, near the region of the sinus node. The left atrium is approached indirectly through the coronary sinus from the right atrium, and the left brachial route is most often successful. The right ventricular apex and/or other locations in this chamber are easily accessible from any venous site. The left ventricle is not usually entered, but when necessary, the femoral arterial approach is preferred. The His bundle electrogram is recorded by introduction of a catheter into the right ventricle, which is slowly withdrawn to the region of the tricuspid valve while clockwise torque is maintained to keep the electrode in contact with the interventricular septum. This maneuver is performed while the electrogram is continuously monitored until a satisfactory tracing incorporating atrial and ventricular electrograms of approximately equal size is obtained. A sharp biphasic or triphasic deflection representing the His bundle electrogram appears at this point between the atrial and ventricular potentials. This technique is successfully performed in most patients using proper signal filtering (40–500 Hz) by an experienced operator, although a stable position of the catheter is difficult to obtain in some cases. Validation of the His bundle potential may be obtained by pacing from the same catheter used for recording.[11] When pacing the His bundle, the interval between the pacing impulse artifact and the onset of the QRS deflection on the surface electrocardiogram should be the same as the onset of the His bundle potential to the QRS prior to pacing. In addition, the configuration of the QRS complex should be the same during sinus rhythm and His bundle pacing, confirming that the catheter is not positioned distal to the His bundle, which would then result in ventricular capture.[11] The technique of His bundle pacing is difficult, and there are exceptions to the rules mentioned above. Therefore, it is seldom performed during most clinical studies.

The induction of arrhythmias during programmed electrical stimulation is common, and this is often the purpose of the study. These arrhythmias are often terminated by pacing techniques as well; at other times they revert spontaneously to sinus rhythm. If the arrhythmia is poorly tolerated, such as with rapid ventricular tachycardia and ventricular fibrillation, or with atrial fibrillation in patients who have rapidly conducting atrioventricular-bypass tracts, electrical cardioversion or defibrillation is mandatory. Accordingly, every electrophysiologic catheterization laboratory must have both a defibrillator, which is checked prior to each study, with ready access to a backup unit, and at least one person in the laboratory who is continuously assessing the clinical status of the patient and is ready to use the defibrillator when indicated. The incidence of induction, in the laboratory, of life-threatening arrhythmias requiring electrical cardioversion or defibrillation varies according to the specific studies performed; incidence is highest when the induction of ventricular tachycardia is attempted in patients with previous episodes of hemodynamically significant sustained ventricular tachycardia or sudden death. Even during these studies, the incidence varies according to the techniques employed. Although it has been reported to be as high as 52 percent,[12] figures around 20 to 30 percent[13] are more in accordance with the experience of most laboratories.

The laboratory personnel should include an experienced catheterizer, as well as a properly trained and experienced electrophysiologist, technician, and nurse, as a minimum for most studies. Experience with sick patients, resuscitation techniques, serious arrhythmias, pacemakers, and antiarrhythmic drugs is mandatory. Studies can be done in a properly equipped catheterization laboratory or procedure room. Under special circumstances, studies may also be done at the bedside or in the cardiothoracic operating room using "portable" equipment. No matter where the study is done, the

requirements for an optimal study are a well-selected and well-prepared patient, trained personnel, and proper equipment.[8]

Timing of Intracardiac Events

Certain baseline measurements are performed routinely in patients undergoing electrophysiologic studies. These include the standard surface electrocardiographic determinations, including the basic cycle length, the duration of the P wave, the P-R interval, the QRS duration, and the Q-T interval, that can often be more accurately measured because of the faster paper speeds used (100 to 200 mm/sec versus 25 mm/sec of the standard electrocardiogram).

The timing of certain intracardiac events during the electrophysiologic study is affected by the autonomic tone of the patient.[14] A quiet environment, dim lights, and a relaxed atmosphere provide conditions as close to baseline as possible. The intervals usually measured include the following: (a) P-A interval or (right) intra-atrial conduction time, (b) interatrial conduction time if a coronary sinus catheter is introduced, (c)

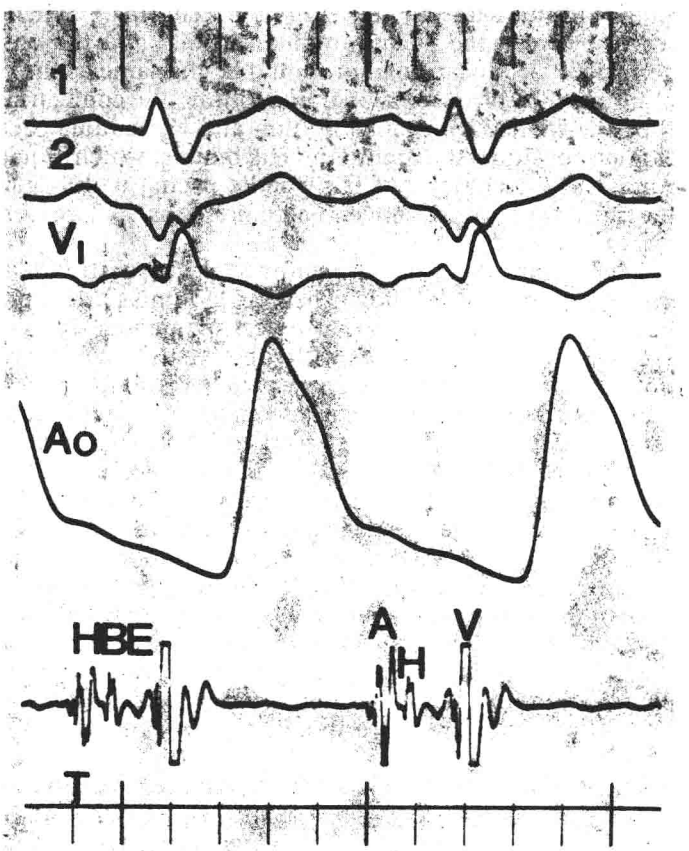

Figure 1. Simultaneous recording of surface electrocardiographic leads 1, 2, and V_1, along with the aortic pressure (Ao), intracardiac His bundle electrogram (HBE), and time lines (T, 100 msec, and 500 msec) in a patient with right bundle branch block, marked left axis deviation, and syncope. The A-H interval, from the onset of the atrial electrogram (A) on the HBE to the onset of His activation (H) represents conduction time through the atrioventricular node; and the H-V interval, from onset of the His to the earliest evidence of ventricular activation (V), in this case on surface lead V_1, indicates the infranodal conduction time.

A-H interval, or conduction time through the atrioventricular node, and (d) H-V interval, or conduction time in the His–bundle branch–Purkinje system (Fig. 1).

The (right) intra-atrial conduction time, between the onset of the surface P wave and the local atrial deflection on the His bundle electrogram (P-A interval), and the interatrial conduction time, between the local atrial deflections on the high right atrial and the coronary sinus electrograms (right-to-left atrial conduction interval), are both approximations of only limited clinical value. To assess atrial conduction more accurately in selected patients, endocardial mapping of the atria is performed. The normal antegrade atrial activation sequence begins in the high- or mid-lateral right atrium, spreading to the atrioventricular junction, and then to the left atrium.[15] When retrograde ventriculoatrial conduction is mapped, retrograde atrial activation normally proceeds from the atrioventricular junction to the left and right atria. This information is important, regardless of the specific conduction times, for the identification of bypass tracts and the evaluation of supraventricular tachycardias.[16]

The A-H interval is measured from the earliest atrial deflection on the His bundle electrogram to the earliest His deflection and represents both the conduction time from the low right atrium to the His bundle, through the atrioventricular node, and, conventionally, the AV nodal conduction time. Normal values vary widely, ranging from approximately 45 to 145 msec, shortening with sympathetic influences and lengthening with enhanced vagal tone.[14] The H-V interval, measured from the earliest His deflection on the His bundle electrogram to the earliest onset of ventricular activation on any surface electrocardiographic lead or intracardiac recording, represents the conduction time from the proximal His bundle to the ventricular myocardium through the His Purkinje system. Normal values are narrowly defined by most investigators as ranging between 35 and 55 msec,[8] and the H-V interval is not usually affected discernibly by variations in autonomic tone during the study. Its stability provides the basis for prospective studies on conduction system disease, inasmuch as the H-V interval should be reproducible during subsequent studies in the absence of pharmacologic interventions or progressive conduction system disease.

Programmed Electrical Stimulation[7,8,9]

Pacing techniques during electrophysiologic studies are used for three main purposes: (1) to characterize the electrical properties of atria, ventricles, and the atrioventricular conduction system; (2) to induce and analyze the mechanisms of arrhythmias; and (3) to evaluate the effects of pharmacologic, electrical, and surgical interventions on those physiologic properties and on the inducibility of arrhythmias. The techniques used are incremental pacing from different sites, and the introduction of single or multiple extrastimuli during spontaneous or paced basic rhythms. Pacing is usually done using a stimulation current that is adjusted in relation to the late diastolic threshold (for example, two or three times diastolic threshold). The relationship between the threshold and the pacing current should remain constant throughout the study and during longitudinal studies of the same patient or population, and the diastolic threshold should be determined again after each intervention (for example, after administration of a drug) or when the pacing site is changed.

Atrial pacing is performed to determine the electrophysiologic properties of the sinus node and the atrioventricular conduction system, and to induce and/or terminate supraventricular tachycardias. Incremental pacing is begun at a cycle length just below (i.e., rate slightly faster than) that of the spontaneous rhythm, with progressive shortening in 50- to 100-msec decrements until atrioventricular nodal block (the Wenckebach point) occurs. Often pacing is done for 1 minute, and the various AV conduction intervals are also assessed at each paced cycle length. The sinus node recovery time

(described below) is also of interest and can be determined from the interval immediately following the offset of pacing. After all parameters have returned to baseline, pacing at the next shorter cycle length is initiated. Ventricular pacing is performed with similar techniques to provide information about ventriculoatrial conduction and bypass tracts, and to induce or terminate ventricular tachyarrhythmias.

Extrastimuli are introduced in the atrium or ventricle, scanning diastole at progressively shorter coupling intervals. For example, after every eight or 10 spontaneous or paced beats, an extrastimulus is introduced at a coupling interval programmed to either the last atrial or ventricular beat; after a preset pause, the eight- or 10-beat cycle repeats, and the extrastimulus can be introduced at a shorter coupling interval. This technique allows for the determination of different electrophysiologic properties of the sinus node, the atrial and ventricular myocardium, and the atrioventricular conduction system. For example, the refractory periods of the different cardiac tissues are determined by the extrastimulus technique, introducing extrastimuli at progressively shorter coupling intervals until a response is no longer elicited. The relative refractory period is reached when delayed conduction of the impulse results, without complete block. The effective (or absolute) refractory period is the longest coupling interval of an impulse that completely fails to propagate through the tissue studied. Refractory periods are a function not only of the electrophysiologic properties of the tissue studied, but also of the basic driving cycle length used and the intensity of the current delivered. Therefore, a constant cycle length is used throughout a study for comparing refractory periods. The effective refractory period of any tissue is inversely related to the current strength used; within the physiologic range, the greater the current the shorter the refractory period. The relation between refractory period and the driving cycle length is more complex and depends on the tissue studied. The refractory period of atrial or ventricular muscle or of the His Purkinje system tends to decrease with decreasing cycle length, whereas that of the atrioventricular node behaves in the opposite fashion: the effective refractory period increases with decreasing driving cycle lengths.[17] Furthermore, both atrioventricular nodal refractoriness and conduction are markedly affected by autonomic tone.[3]

THE PATIENT WITH BRADYCARDIA

Bradycardia, arbitrarily defined as a heart rate of less than 60 beats per minute, is usually not a pathologic finding. It has been frequently reported in healthy athletes as an adaptative mechanism to cardiovascular conditioning,[18,19] and physiologic bradycardia has been associated with the normal aging process.[20] The electrophysiologic abnormalities found in patients with bradycardia can be divided into those of an altered impulse formation (sinus node dysfunction) and those of impaired impulse conduction. This division is somewhat arbitrary, and abnormalities of impulse formation and conduction are frequently observed in the same individual, but these abnormalities are evaluated separately in the electrophysiologic laboratory using specific techniques described below. Abnormal sensitivity to alterations in autonomic tone may also have profound effects on both automaticity and conduction.

Evaluation of the Sinus Node

Although sinus bradycardia is frequent in the general population and heart rates as slow as 35 to 45 beats per minute may occur in healthy individuals such as long distance runners,[18] sinus node dysfunction is the primary indication for approximately 50 percent of the permanent pacemaker implantations in this country.[21] Nevertheless, the survival of patients with the sick sinus syndrome is reportedly similar to that of the