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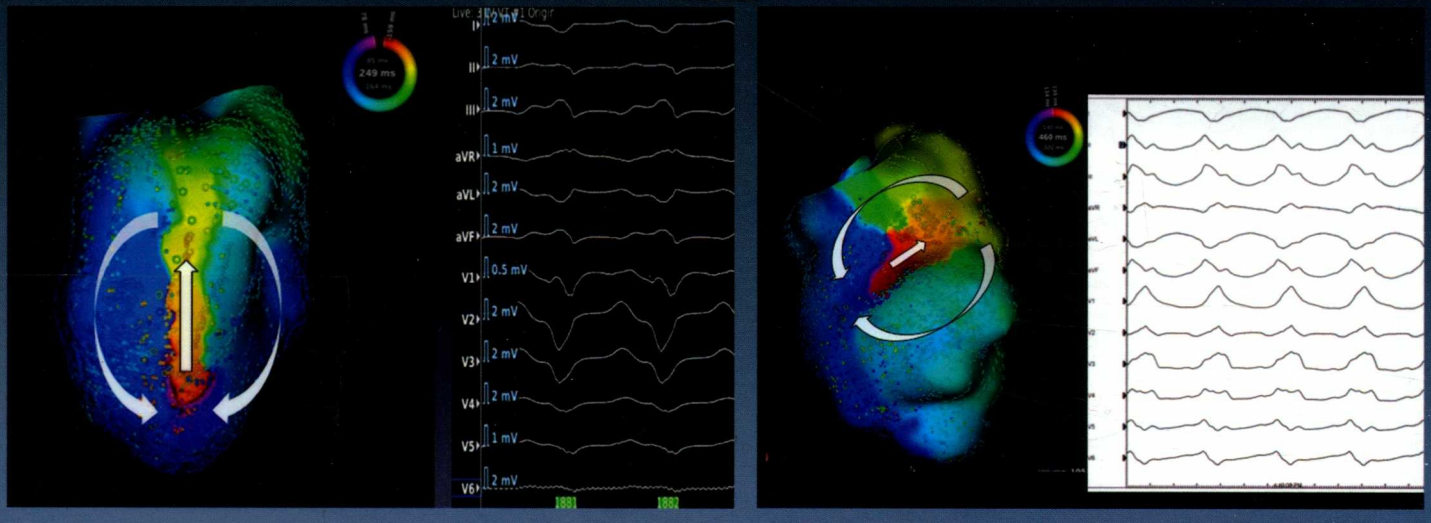
MARK E. JOSEPHSON

Josephson's Clinical Cardiac Electrophysiology

TECHNIQUES AND INTERPRETATIONS

FIFTH EDITION



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Fifth Edition

JOSEPHSON'S Clinical Cardiac Electrophysiology

Techniques and Interpretations

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This book is dedicated to my family—Sylvie, Elan, Sydney, Rachel, Todd, Stephanie, Jesse, and particularly, to my wife Joan—for their love, support, and understanding. Joan, you have been the wind beneath my wings.

FOREWORD

Historical Perspectives

The study of the heart as an electrical organ has fascinated physiologists and physicians for nearly a century and a half. Matteucci[1] studied electrical current in pigeon hearts, and Kölliker and Müller[2] studies discrete electrical activity in association with each cardiac contraction in the frog. Study of the human electrocardiogram awaited the discoveries of Waller[3] and, most important, Einthoven,[4] whose use and development of the string galvanometer permitted the standardization and widespread use of that instrument. Almost simultaneously, anatomists and pathologists were tracing the atrioventricular (A-V) conduction system. Many of the pathways, both normal and abnormal, still bear the names of the men who described them. This group of men included His,[5] who discovered the muscle bundle joining the atrial and ventricular septae that is known as the common A-V bundle or the bundle of His.

During the first half of the 20th century clinical electrocardiography gained widespread acceptance, and, in feats of deductive reasoning, numerous electrocardiographers contributed to the understanding of how the cardiac impulse in man is generated and conducted. Those researchers were, however, limited to observation of atrial (P wave) and ventricular (QRS complex) depolarizations and their relationships to one another made at a relatively slow recording speed (25 mm/s) during spontaneous rhythms. Nevertheless, combining those carefully made observations of the anatomists and the concepts developed in the physiology laboratory, these researchers accurately described, or at least hypothesized, many of the important concepts of modern electrophysiology. These included such concepts as slow conduction, concealed conduction, A-V block, and the general area of arrhythmogenesis, including abnormal impulse formation and reentry. Some of this history was reviewed by the late Langendorf.[6] Even the mechanism of pre-excitation and circus movement tachycardia were accurately described and diagrammed by Wolferth and Wood from the University of Pennsylvania in 1933.[7] The diagrams in that manuscript are as accurate today as they were hypothetical in 1933. Much of what has followed the innovative work of investigators in the first half of the century has confirmed the brilliance of their investigations.

In the 1940s and 1950s, when cardiac catheterization was emerging, it became increasingly apparent that luminal catheters could be placed intravascularly by a variety of routes and safely passed to almost any region of the heart, where they could remain for a substantial period of time. Alanis et al. recorded the His bundle potential in an isolated perfused animal heart,[8] and Kottmeier et al. recorded the His bundle potential in man during open heart surgery.[9] Giraud et al. were the first to record electrical activity from the His bundle by a catheter;[10] however, it was the report of Scherlag et al.,[11] detailing the electrode catheter techniques in dogs and humans, to reproducibly record His bundle electrogram, which paved the way for the extraordinary investigations that have occurred over the past two and a half decades.

At about the time Scherlag et al.[11] were detailing the catheter technique of recording His bundle activity, Durrer et al. in Amsterdam and Coumel and his associates in Paris independently developed the technique of programmed electrical stimulation of the heart in 1967.[12,13] This began the first decade of clinical cardiac electrophysiology. Although the early years of intracardiac recording in man were dominated by descriptive work exploring the presence and timing of His bundle activation (and that of a few other intracardiac sites) in a variety of spontaneously occurring physiologic and pathologic states, a quantum leap occurred when the technique of programmed stimulation was combined with intracardiac recordings by Wellens.[14] Use of these techniques subsequently furthered our understanding of the functional component of the A-V specialized conducting system, including the refractory periods of the atrium, A-V node, His bundle, Purkinje system, and ventricles and enables us to assess the effects of pharmacologic agents on these parameters, to induce and terminate a variety of tachyarrhythmias, and, in a major way, has led to a greater understanding of the electrophysiology of the human heart. Shortly thereafter, enthusiasm and inquisitiveness led to placement of an increasing number of catheters for recording and stimulation to different locations with the heart, first in the atria and thereafter in the ventricle. This first led to development of endocardial catheter mapping techniques to define the location of bypass tracts and the mechanisms of supraventricular tachyarrhythmias.[15,16]

Beginning in the mid-1970s, Josephson and his colleagues at the University of Pennsylvania were the first to use vigorous, systematic, multisite programmed stimulation in the study of sustained ventricular tachycardia (VT) resulting from myocardial infarction, which allowed induction of VT in more than 90% of the patients in whom this rhythm occurred spontaneously.[17–19] Subsequent investigators sought to establish a better understanding of the methodology used in the electrophysiology study to induce arrhythmias. Several studies validated the sensitivity and specificity of programmed stimulation for induction of uniform tachycardias, and the nonspecificity of polymorphic arrhythmias induced with vigorous programmed stimulation was recognized.[19,20]

In the same time period, Josephson et al.[21–23] developed the technique of endocardial catheter mapping of VT, which for the first time demonstrated the safety and significance of placing catheters in the left ventricle. This led to the recognition of the subendocardial origin of the majority of ventricular

tachyarrhythmias, associated with coronary artery disease and the development of subendocardial resection as a therapeutic cure for this arrhythmia.[24]

For the next decade, electrophysiologic studies continued to better understand the mechanisms of arrhythmias in man by comparing the response to programmed stimulation in man in the response to in vitro and in vivo studies of abnormal automaticity, triggered activity caused by delayed and early afterdepolarizations, and anatomical functional reentry. These studies, which used programmed stimulation, endocardial catheter mapping, and response of tachycardias to stimulation and drugs, have all suggested that most sustained paroxysmal tachycardias were due to reentry. The reentrant substrate could be functional or fixed or combinations of both. In particular, the use of entrainment and resetting during atrial flutter and VT were important techniques used to confirm the reentrant nature of these arrhythmias.[25–30] Resetting and entrainment with fusion became phenomena that were diagnostic of reentrant excitation. Cassidy et al.[31] using left ventricular endocardial mapping during sinus rhythm, for the first time described an electrophysiologic correlate of the pathophysiologic substrate of VT in coronary artery disease—a low-amplitude fragmented electrograms of long duration and late potentials.[31,32] Fenoglio, Wit, Josephson, and their colleagues from the University of Pennsylvania documented for the first time that these arrhythmogenic areas were associated with viable muscle fibers separated by and imbedded in scar tissue from the infarction.[33] They demonstrated that the quality and quantity of abnormal electrograms (and, hence, the pathophysiologic substrate) differed for sustained monomorphic VT, nonsustained VT, and ventricular fibrillation in patients with prior infarction and cardiomyopathy. Experimental studies by Gardner et al.[34] demonstrated that these fractionated electrograms resulted from poorly coupled fibers that were viable and maintained normal action potential characteristics but that exhibited salutatory conduction and caused by nonuniform anisotropy. Further exploration of contributing factors (triggers), such as the influence of the autonomic nervous system or ischemia, will be necessary to further enhance our understanding of the genesis of the arrhythmias. This initial decade or so of electrophysiology could be likened to an era of discovery.

Subsequently, and overlapping somewhat with the era of discovery, was the development of the concept and use of programmed stimulation as a tool for developing therapy for arrhythmias. The ability to reproducibly initiate and terminate arrhythmias led to the development of serial drug testing to assess antiarrhythmic efficacy.[35] The ability of an antiarrhythmic drug to prevent initiation of a tachycardia that we reliably initiated in the control state appeared to predict freedom from the arrhythmia in the 2- to 3-year follow-up. This was seen in many nonrandomized clinical trials from laboratories in the early 1980.

The persistent inducibility of an arrhythmia universally predicted an outcome that was worse than that in patients in whom tachycardias were made noninducible. The natural history of recurrences of ventricular tachyarrhythmias (or other arrhythmias for that matter) and the changing substrate for arrhythmias were recognized potential imitations of drug testing. It was recognized very early that programmed stimulation was not useful in selecting drugs to treat ventricular tachyarrhythmias in patients without coronary artery disease (i.e., cardiomyopathy).[36] Despite the fact that all studies showed that patients with spontaneous VT whose arrhythmias were rendered noninducible by antiarrhythmic agents far better than patients with persistently inducible arrhythmias, the inability to accurately predict freedom from recurrence led to abandonment of programmed stimulation as a modality to select antiarrhythmic agents. The ESVEM study,[37] although plagued by limitations in protocol and patient selection, put the nail in the coffin for programmed stimulation as a method of selecting antiarrhythmic therapy of arrhythmias.

With the known limitation of EP-guided therapy to predict outcomes uniformly and correctly, as well as the potentially lethal proarrhythmic effect of antiarrhythmic agents demonstrated in the CAST study,[38] the desire for nonpharmacologic approaches to therapy grew. Surgery had already become a gold standard therapy for Wolff–Parkinson–White syndrome, and innovative surgical procedures for VT had grown from our understanding of the pathophysiologic substrate of VT and coronary disease and the mapping of VT from the Pennsylvania group. However, surgery was considered a rather drastic procedure for patients with a relatively benign disorder (supraventricular tachycardia and the Wolff–Parkinson–White syndrome), and although successful for VT for coronary artery disease, was associated with a high operative mortality. These limitations have led to two major areas of nonpharmacologic therapy that have dominated the last 25 years; implantable antitachycardia/defibrillator devices and catheter ablation. These techniques were the natural evolution of our knowledge of arrhythmia mechanisms (e.g., the ability to initiate and terminate the reentrant arrhythmias by pacing and electrical conversion) and the refinement of catheter mapping techniques and the success of surgery used with these techniques.

It was Mirowski who initially demonstrated that an implantable defibrillator could convert VT or ventricular fibrillation to sinus rhythm regardless of underlying pathophysiologic substrate and prevent sudden cardiac death.[39] The initial devices were implanted epicardially via thoracotomy have been replaced by small devices with active cans and prevenous leads that are implanted pectorally similar to a pacemaker. Current devices may have single chamber, dual chamber, and biventricular pacing capability. The antitachycardia pacing modalities which evolve from clinical EP studies are widely employed and effective in terminating monomorphic gradient from VT particularly those with rates. With several major trials showing a statistical benefit of ICDs in reducing sudden death, there has been a widespread, logarithmic increase in the use of the device. I have removed the chapter on implantable devices from this edition because there are multiple texts on the topic and the electrophysiologic basis for their use is in the text.

The major thrust of the last 25 years has been the development and the use of catheterization techniques to manage cardiac arrhythmias. The concept of using a catheter to deliver energy as an antitachycardia therapeutic modality came from Dr. Melvin Scheinman[40] who was the first to demonstrate the ability to ablate the A-V junction via a catheter to control a ventricular rate in atrial fibrillation. Subsequently, the energy sources changed from a defibrillator to radiofrequency energy which is the standard at this point in time. Nonetheless, additional energy sources such as cryothermal energy, focused ultrasound, and laser energy are all currently being evaluated as modalities to be delivered by a catheter to treat arrhythmias. At the present time focal ablation using radiofrequency is the treatment of choice for all supraventricular tachyarrhythmias, including A-V nodal reentry, circus movement tachycardias using concealed or manifested accessory pathways, incessant automatic atrial tachycardia, isthmus-dependent atrial flutter as well as other macroreentrant atrial tachycardias, and VTs in both normal hearts and those associated with prior infarction.[41–55] In addition, ablation has become the treatment of choice for VPC-induced cardiomyopathy. Most exciting has been the development of the potential for ablation use in the treatment of atrial fibrillation. While the initial studies suggested that isolating the pulmonary veins to prevent the pulmonary vein foci from initiating and maintaining atrial fibrillation[56–59] have been used successful in paroxysmal atrial fibrillation, how best to treat persistent and chronic atrial fibrillation still remains unclear. We still do not understand the basic mechanisms of maintenance of atrial fibrillation, so it is not surprising that we don't know how to "fix" it. While isolations with radiofrequency energy have a reasonable acute success for paroxysmal atrial fibrillation reconnections are common and recurrences frequent, particularly if monitoring is done continuously. There has been an interest in using a variety of other lesion sets to treat persistent atrial fibrillation, but none have proved successful, and many times additional atrial tachycardias are a consequence of additional linear lesions. Most recently new high-resolution mapping systems and phase mapping using a new technology have been introduced in an attempt to improve success and understand the underpinnings of the arrhythmia.

In order to reduce stroke cool-tip radiofrequency catheters have been deployed to decrease the coagulant information resulting from noncooled-tip catheters to decrease the incidence of stroke, which remains a potential complication of this ablation. There is an interest in developing new methods to decrease strokes by use of left atrial occlusion devices one of which has just been FDA approved. How wide spread the use of these devices will be is unclear, but they are certainly reasonable for patients who can't take anticoagulation and do not wish to undergo a left atrial appendagectomy. New anticoagulants have been developed which will likely replace Coumadin.

One major concept I believe that is critical is that we need to understand the mechanism of arrhythmias before we try to "cure" them with ablation. This was easily done for supraventricular arrhythmias. The ability to accurately define reentrant circuits causing VT and even the underlying mechanism of atrial fibrillation needs further work. Although much has been accomplished, much work still remains. We must not let technology lead the way. We electrophysiologists must maintain our interest in understanding the mechanisms of arrhythmias to that we can devise nonpharmacologic or even pharmacologic approaches that would be more effective and safe to manage these arrhythmias. New molecular approaches may be forthcoming in the near future. The world of molecular biology has seen the recognition of ion channelopathies such as long QT syndrome, Brugada syndrome, idiopathic ventricular fibrillation, and catecholaminergic polymorphic VT. Early understanding of these disorders has led to potential ablative therapy, particularly in the Brugada syndrome, and the reintroduction of old fashioned drugs like quinidine and programmed stimulation to treat the short QT syndrome, Brugada syndrome, and idiopathic ventricular fibrillation.[60–62] Cardiovascular genomics will play an important role in risk stratification of arrhythmias in the future and new fields of proteomics and metabolomics will be essential if we are to develop specifically targeted molecules to treat arrhythmias.

The past 45 years have seen a rapid evolution of electrophysiology, from one of understanding the simple mechanisms to one of developing therapeutic interventions. The future will require us to go back to the past and continue to understand more complex underlying mechanisms so that our therapeutic modalities will be more successful and safe.

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PREFACE

The past 45 years have witnessed the birth, growth, and evolution of clinical electrophysiology, from a field whose initial goals were the understanding of arrhythmia mechanisms to one of significant therapeutic impact. The development and refinement of implantable devices and, in particular, catheter ablation have made nonpharmacologic therapy a treatment of choice for most arrhythmias encountered in clinical practice. Unfortunately, these new therapeutic tools have captured the imagination of young electrophysiologists to such an extent that terms such as *ablationist*, *defibrillationist*, or *implanter* are used to describe their practice. Their zest for the application of such therapeutic modalities has been associated with a decrease in the emphasis of understanding the mechanisms, clinical implications, and limitations of the therapeutic interventions used to treat arrhythmias. Such behavior is often associated with a lack of, or limited, critical thought that is essential to the development of a new therapeutic concept. There should be the development of a hypothesis, questioning the rationale of the hypothesis, and the testing the hypothesis prior to widespread application of the therapeutic strategy.

The purpose of this book is to provide the budding electrophysiologist with an *electrophysiologic* approach to arrhythmias, which is predicated on the hypothesis that a better understanding of the mechanisms of arrhythmias will lead to more successful and rationally chosen therapy. As such, this book will stress the methodology required to define the mechanism and site of origin of arrhythmias so that safe and effective therapy can be chosen. The techniques suggested to address these issues and specific therapeutic interventions employed represent a personal view, one that is based on experience and, not infrequently, on intuition.

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CONTENTS

CHAPTER

1 Electrophysiologic Investigation: Technical Aspects

PERSONNEL

The most important aspects for the performance of safe and valuable electrophysiologic studies are the presence and participation of dedicated personnel. The minimum personnel requirements for such studies include at least one physician, one or two nurses (two nurses for complex ablations requiring conscious sedation), a technician with radiation expertise, an anesthesiologist on standby, and an engineer on the premises to repair equipment. With the widespread use of catheter ablation, appropriate facilities and technical support are even more critical.[1,2] The most important person involved in such studies is the physician responsible for the performance and interpretation of these studies. This person should have been fully trained in clinical cardiac electrophysiology in an approved electrophysiology training program. The guidelines for training in clinical cardiac electrophysiology have undergone remarkable changes as interventional electrophysiology has assumed a more important role. The current training guidelines for competency in cardiac electrophysiology have been developed by the American College of Cardiology and the American Heart Association, and the American College of Physicians-American Society of Internal Medicine in collaboration with the Heart Rhythm Society (formerly, the North American Society for Pacing and Electrophysiology).[3,4] Based on these recommendations, criteria for certification in the subspecialty of clinical cardiac electrophysiology have been established by the American Board of Internal Medicine. Certifying exams are now given twice a year. Recertification is required every 10 years. The clinical electrophysiologist should have electrophysiology in general and arrhythmias in particular as his or her primary commitment. As such, they should have spent a minimum of 1 year, preferably 2 years, of training in an active electrophysiology laboratory and have met criteria for certification. The widespread practice of device implantation by electrophysiologists will certainly make a combined pacing and electrophysiology program mandatory for implanters. This should be a 2-year program. Recently, with the development of resynchronization therapy for heart failure, there has been an interest in developing a program to train heart failure physicians to implant devices in their patients. At the least this should be a program of 1 year, and in my opinion, should include training in basic electrophysiology. Such programs are currently available in a few centers. Sufficient training is necessary for credentialing, which will be extremely important for practice and reimbursement in the future.

One or, preferably, two nurses and a technician are the bare minimum support for simple EP studies and devices. Complex ablations (AF, VT, etc.) should be supported by two nurses and a technician. This is critical for safety, particularly with use of conscious sedation or anesthesia in patients in whom there is risk of life-threatening complications. These nurse–technicians must be familiar with all the equipment used in the laboratory and must be well trained and experienced in the area of cardiopulmonary resuscitation. We use two or three dedicated nurses and a technician in each of our electrophysiology laboratories. Their responsibilities range from monitoring hemodynamics and rhythms, using the defibrillator/cardioverter when necessary, and delivering antiarrhythmic medications and conscious sedation (nurses), to collecting and measuring data online during the study. They are also trained to treat any complications that could possibly arise during the study. An important but often unstressed role is the relationship of the nurse and the patient. The nurse is the main liaison between the patient and physician during the study—both verbally, communicating symptoms, and physically, obtaining physiologic data about the patient's clinical status. The nurse–technician may also play an invaluable role in carrying out laboratory-based research. It is essential that the electrophysiologist and nurse–technician function as a team, with full knowledge of the purpose and potential complications of each study being ensured at the outset of the study. A radiation technologist should also be available to assure proper equipment function and monitor radiation dose received by patients and laboratory personnel.

An anesthesiologist and probably a cardiac surgeon should be available on call in the event that life-threatening arrhythmias or complications requiring intubation, ventilation, thoracotomy, and potential surgery should arise. This is important in patients undergoing stimulation and mapping studies for malignant ventricular arrhythmias and, in particular, catheter ablation techniques (see Chapter 14). In addition, an anesthesiologist or nurse-anesthetist usually provides anesthesia support for ICD implantation and/or testing. We use anesthesia for all our atrial fibrillation ablations, and for ablative procedures in patients with fragile hemodynamics to

enable us to maintain smooth hemodynamic control during the procedure. Anesthesia is also extremely useful in elderly patients because of the frequent paradoxical response to standard sedation. Although conscious sedation is usually given by laboratory staff, in the substantial minority of laboratories, anesthesia (e.g., propofol) is given by the laboratory staff (nurse or physician) and not by an anesthesiologist.

A biomedical engineer and/or technician should be available to the laboratory to maintain equipment so that it is properly functioning and electrically safe. It cannot be stated too strongly that electrophysiologic studies must be done by personnel who are properly trained in and who are dedicated to the diagnosis and management of arrhythmias. This opinion is shared by the appropriate associations of internal medicine and cardiology.[1–4] Finally a radiation technologist should be available to assure that excessive radiation is not delivered to the patient or electrophysiology team.

EQUIPMENT

The appropriate selection of tools is of major importance to the clinical electrophysiologist. Although expensive and elaborate equipment cannot substitute for an experienced and careful operator, the use of inadequate equipment may prevent the maximal amount of data from being collected, and it may be hazardous to the patient. To some degree, the type of data collected determines what equipment is required. If the only data to be collected involve atrioventricular (A-V) conduction intervals (an extremely rare situation), this can be determined with a single catheter and a simple ECG-type amplifier and recorder, which are available in most cardiology units. However, a complete evaluation of most supraventricular arrhythmias, which may require activation mapping, necessarily involves the use of multiple catheters and several recording channels as well as a programmable stimulator. Thus, an appropriately equipped laboratory should provide all the equipment necessary for the most detailed study. In the most optimal of situations, a room should be dedicated for electrophysiologic studies. This is not always possible, and in many institutions, the electrophysiologic studies are carried out in the cardiac hemodynamic–angiographic catheterization laboratory. A volume of more than 100 cases per year probably requires a dedicated laboratory. The room should have air-filtering equivalent to a surgical operating room, if it is used for ICD and pacemaker implantation. This is the current practice in more than 90% of centers and is likely to be the universal practice in the future. It is important that the electrophysiology laboratory have appropriate radiographic equipment. The laboratory must have an image intensifier that is equipped for at least fluoroscopy, and, in certain instances, is capable of cine-fluoroscopy if the laboratory is also used for coronary angiography. To reduce radiation exposure, pulsed fluoroscopy or other radiation reduction adaptations are required. This has become critical in the ablation era, when radiation exposure can be prolonged and risk of malignancy increased. Currently the best systems are pulsed and digitally based, which reduces the radiation risk and allow for easy storage of acquired data. The equipment must be capable of obtaining views in multiple planes. Newer systems which markedly reduce radiation exposure enable the electrophysiologist to move catheters at a distance or in the absence of the fluoroscopic system. Examples of such systems are the Stereotaxis magnetic guided catheter positioning system and Hanson robotic system. The Stereotaxis system is available at this time; it is expensive and requires special catheters, which add to the expense. The Hanson system is currently not approved in the US, but is likely to be within the year. It is less expensive but all catheters can be used. These systems are of most value for complex ablations (e.g., atrial fibrillation or untolerated ventricular tachycardia), but seem excessive for most procedures. Other navigation systems are also being developed with the goal of reproducible three-dimensional navigation and reduction of fluoroscopy time and exposure. Currently, state-of-the-art equipment for the gamut of electrophysiologic studies includes permanent radiographic equipment of the C-arm, U-arm, or biplane varieties. It is critical that dosimetry to the patient is monitored. Guidelines for total dosage delivered during a single procedure are needed to prevent radiation-induced injury should be mandated.

Electrode Catheters

A variety of catheters is currently available with at least two ring electrodes that can be used for bipolar stimulation and/or recording. The catheter construction may be of the woven Dacron variety or of the newer extruded synthetic materials such as polyurethane. As a general all-purpose catheter, we prefer the woven Dacron catheters (Bard Electrophysiology, Billerica, MA) because of their greater durability and physical properties. These catheters come with a variable number of electrodes, electrode spacing, and curves to provide a range of options for different purposes (Fig. 1-1). Although they have superior torque characteristics, their greatest advantage is that they are stiff enough to maintain a shape and yet they soften at body temperature so that they are not too stiff for forming loops and bends in the vascular system to adapt a variety of uses. The catheters made of synthetic materials cannot be manipulated and change shapes within the body, so they are less desirable. Many companies make catheters for specific uses such as coronary sinus cannulation, His bundle recording, etc., but in most cases I believe this is both costly and unnecessary. The advantages of the synthetic catheters are that they are cheaper and can be made smaller (2 to 3 French) than the woven Dacron types. Currently, most electrode catheters are size 3 to size 8 French. The smaller sizes are used in children. In adult patients, sizes 5 to 7 French catheters are routinely used. Other diagnostic catheters have a deflectable tip (Fig. 1-2). These are useful to reach and record from specific sites (e.g., coronary sinus, crista terminalis, tricuspid valve). In most instances the standard woven Dacron catheters suffice, and they are significantly cheaper. Although special catheters

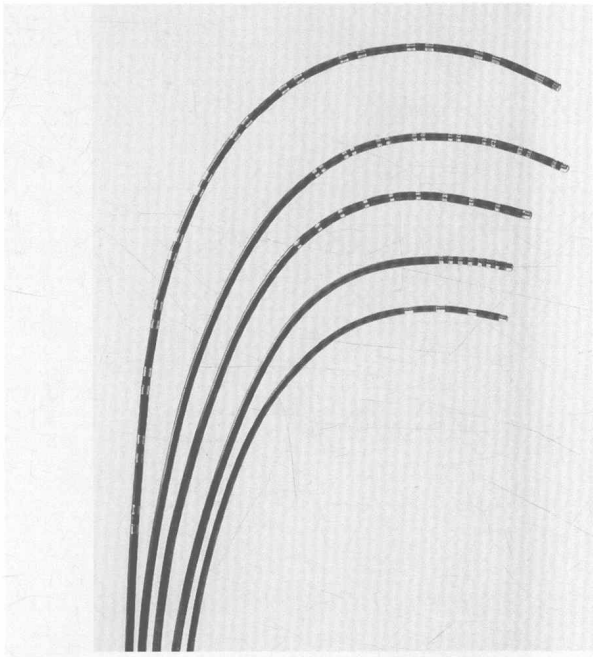

FIGURE 1-1 *Electrode catheters routinely used.* Woven Dacron catheters with varying number of electrodes and interelectrode distances.

are useful for specific indications described below, standard catheters can be used for most standard pacing and stimulation protocols. We save our hospital thousands of dollars by using standard woven Dacron catheters for all but the ablation catheter. Skilled catheterizers rarely require steerable catheters for positioning, the cost of which is often >$500 in excess of standard catheters.

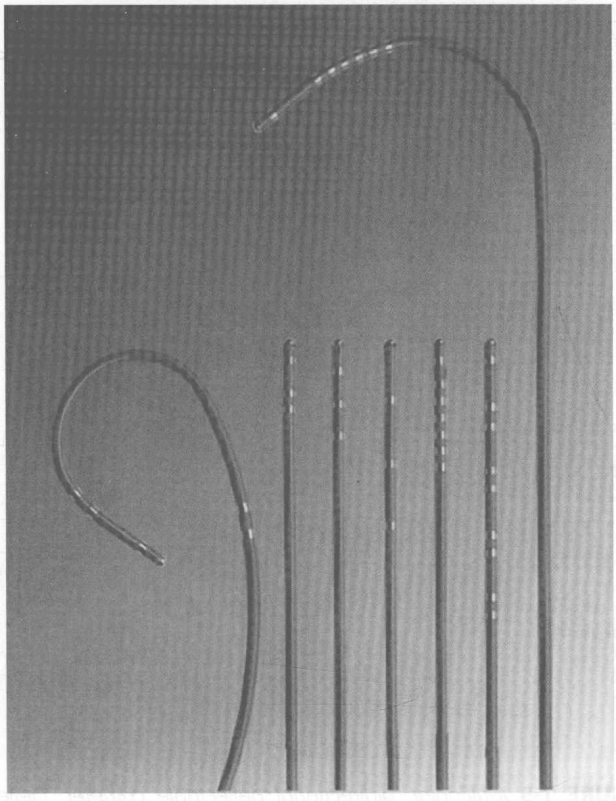

FIGURE 1-2 *Electrode catheter with deflectable tips.* Different types of catheters with deflectable tips. These are primarily made of extruded plastic.

Electrode catheters have been designed for special uses. Catheters with an end hole and a lumen for pressure measurements may be useful in: (a) electrophysiologic hemodynamic diagnostic studies for Ebstein's anomaly; (b) validation of a His bundle potential by recording that potential and the right atrial pressure simultaneously (see Chapter 2); (c) the occasional instance when it may be desirable to pass the catheter over a long guidewire or transseptal needle; and (d) electrophysiologic studies that are part of a more general diagnostic study and/or for which blood sampling from a specific site (e.g., the coronary sinus) or angiography in addition to pacing is desirable. Special catheters have also been designed to record a sinus node electrogram, although we believe that such electrograms can be obtained using standard catheters (see Chapter 3). Other catheters have been specially designed to facilitate recording of the His bundle potential using the antecubital approach, which occasionally may be useful when the standard femoral route is contraindicated. This catheter has a deflectable tip that permits it to be formed into a pronounced J-shape once it has been passed into the right atrium.

In the last decade the evolution of ablation techniques for a variety of arrhythmias necessitated the development of catheters that enhance the ability to map as well as to safely deliver radiofrequency energy. Mapping catheters fall into two general categories: (a) deflectable catheters to facilitate positioning for mapping and delivering ablative energy and (b) catheters with multiple poles (8 to 64) that allow for simultaneous acquisition of multiple activation points. The former category includes a variety of ablation catheters as well as catheters to record and pace from specific regions (e.g., coronary sinus, tricuspid annulus, slow pathway [see Chapter 8], crista terminalis [see Chapter 9]). Some ablation catheters have a cooled tip, one through which saline is infused to allow for enhanced tissue heating without superficial charring (Biosense Webster and St Jude) or internal cooling (Chili; Boston Scientific) (Fig. 1-3). Ablation catheters deliver RF energy through tips that are typically 3.5 to 5 mm in length but may be as long as 10 mm. Catheters that are capable of producing linear radiofrequency lesions are being developed to treat atrial fibrillation by compartmentalizing the atria, but currently the ability of these catheters to produce transmural linear lesions that have clinical benefit and are safe is not proven. Catheters that deliver microwave, laser, cryothermal, or pulsed-ultrasound energy to destroy tissue are currently under active investigation. The cryothermal catheters have recently been approved by the FDA for A-V nodal modification for A-V nodal tachycardia (see Chapter 14) but are also being evaluated for other uses, that is, atrial fibrillation. For this latter use a cryoballoon catheter has been developed (Arctic; Medtronic [Fig. 1-4]). In the second category are included standard catheters with up to 24 poles that can be deflected to map large and/or specific areas of the atrium (e.g., coronary sinus, tricuspid annulus, etc.) (Fig. 1-5). Of particular note are catheters shaped in the

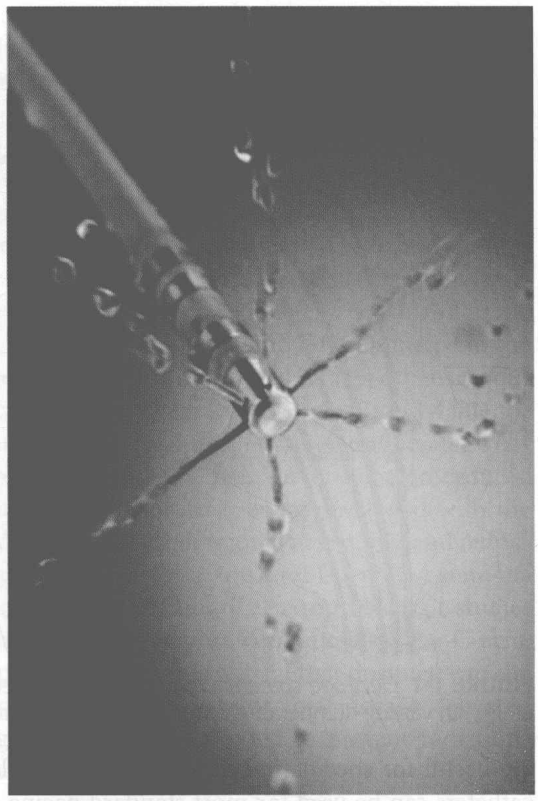

FIGURE 1-3 *Cool tip ablation catheter.* Saline spray through the catheter tip is used to maintain "low" tip temperature to prevent charring while at the same time increasing lesion size. See text for discussion.

form of a "halo" to record from around the tricuspid ring (Fig. 1-6), or a lasso catheter on a deflectable shaft to record from 10 to 20 electrodes in the pulmonary vein/ostia, (Biosense Webster and St Jude) (Fig. 1-7) and basket catheters (Fig. 1-8), which have up to 64 poles or prongs that spring open and which are used to acquire simultaneous data

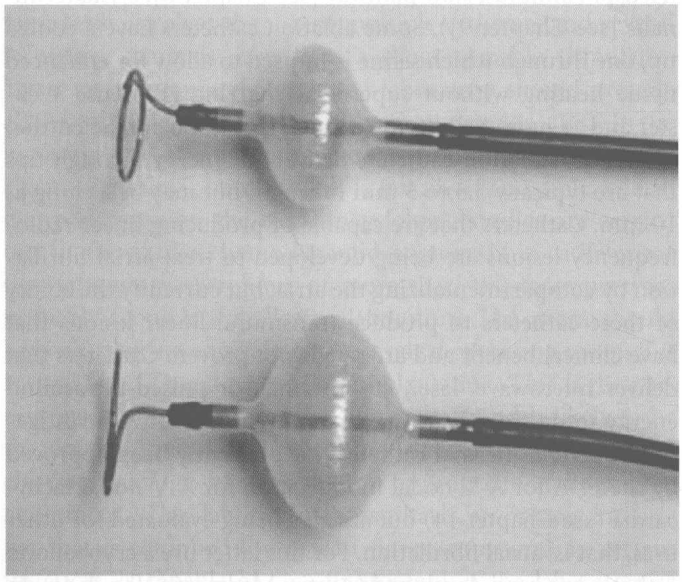

FIGURE 1-4 *Cryoballoon catheter for pulmonary vein isolation.* Two sizes of balloon catheters (24 and 28 cm) are available to deliver cryothermal lesions to the pulmonary vein ostia. A flexible lasso insets thru a lumen to identify the osyia and record pulmonary vein potentials. See Chapter 14 for further discussion.

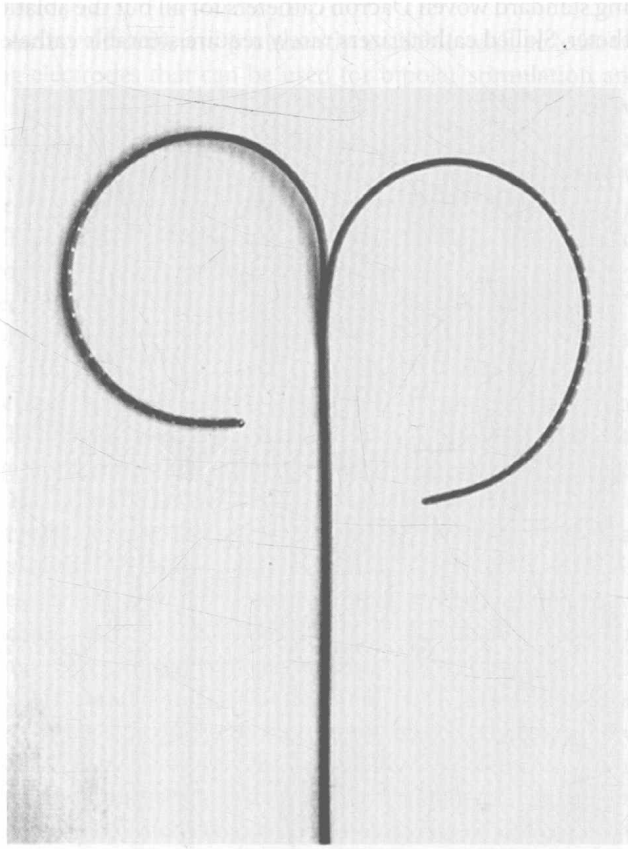

FIGURE 1-5 *Multipolar, bidirectional deflectable catheter.* Deflectable catheters with 10 to 24 poles that have bidirectional curves are useful for recording from the entire coronary sinus or the anterolateral right atrium along the tricuspid annulus.

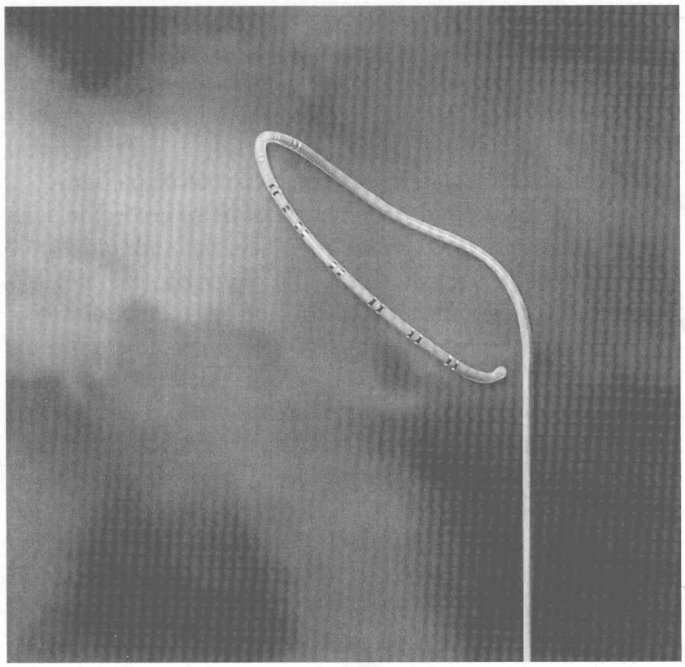

FIGURE 1-6 *Multipolar deflectable catheter for recording around the tricuspid annulus*. While standard 10 to 20 pole woven Dacron or deflectable catheters can be used to record along the anterolateral tricuspid annulus, a "halo" catheter has been specifically designed to record around the tricuspid annulus.

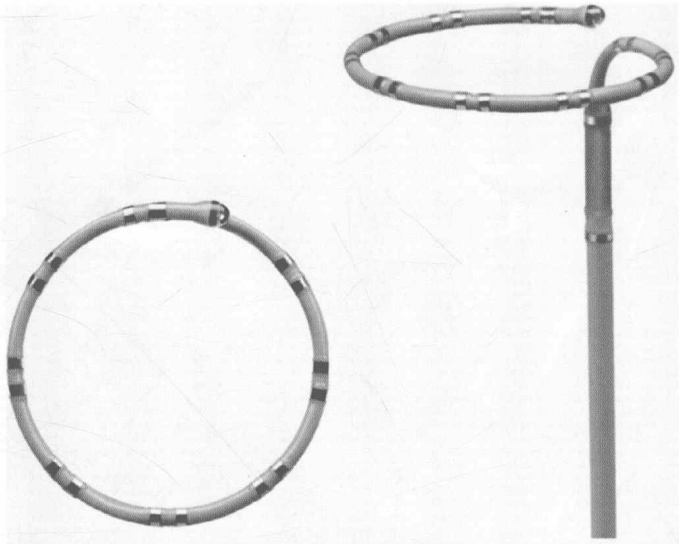

FIGURE 1-7 *Lasso catheter*. This lasso catheter is used to record from and pace inside the pulmonary vein ostia before and after pulmonary vein isolation procedures (see Chapter 14). The catheter can also be used to create an "anatomic" shell of a chamber as well as to acquire multiple simultaneous activation times.

from within a given cardiac chamber. A PentaRay catheter (Fig. 1-9) is available from Biosense Webster which has five flexible splines with 4 electrodes on each spline allowing on to acquire 20 sites of activation. The 2-mm interelectrode distance allows for high-density mapping. The floppiness of

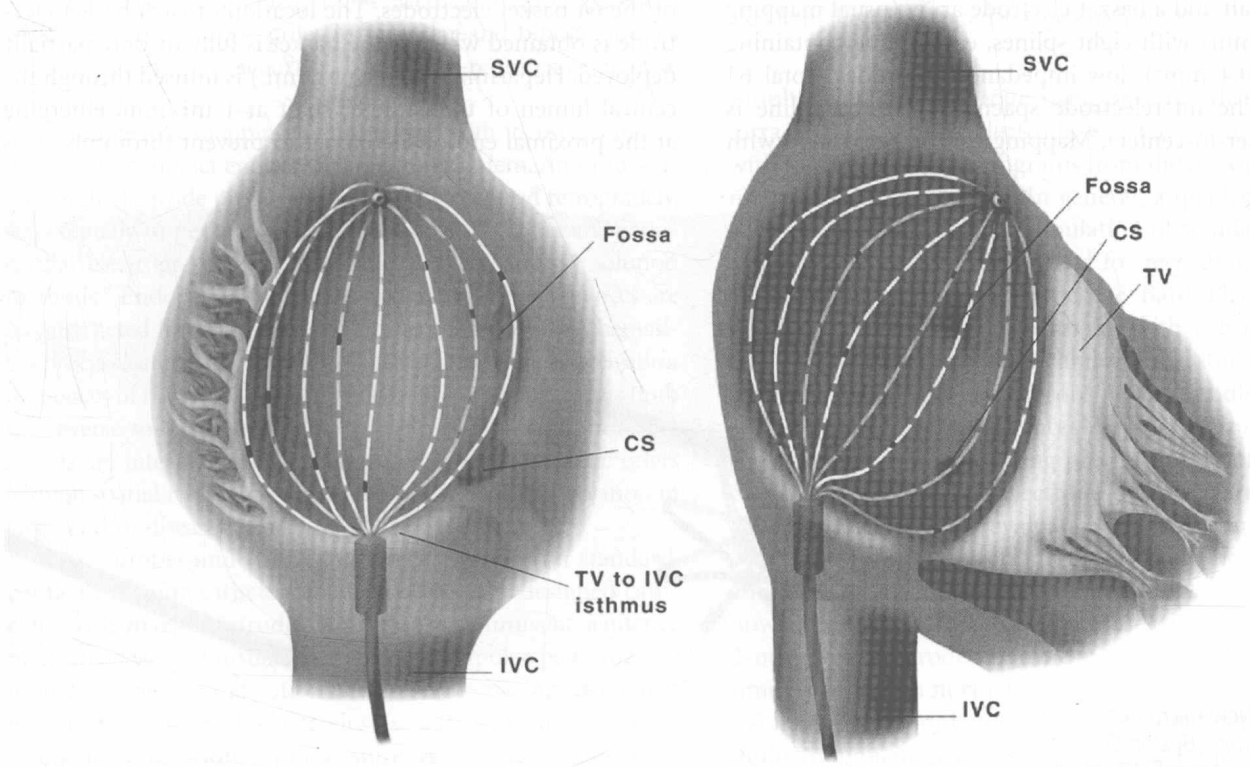

FIGURE 1-8 *Basket catheter*. A 64-pole retractable "basket" catheter with 8 splines is useful for simultaneous multisite data acquisition for an entire chamber. The schema demonstrates the catheter position in the right atrium when used for the diagnosis and treatment of atrial tachyarrhythmias.

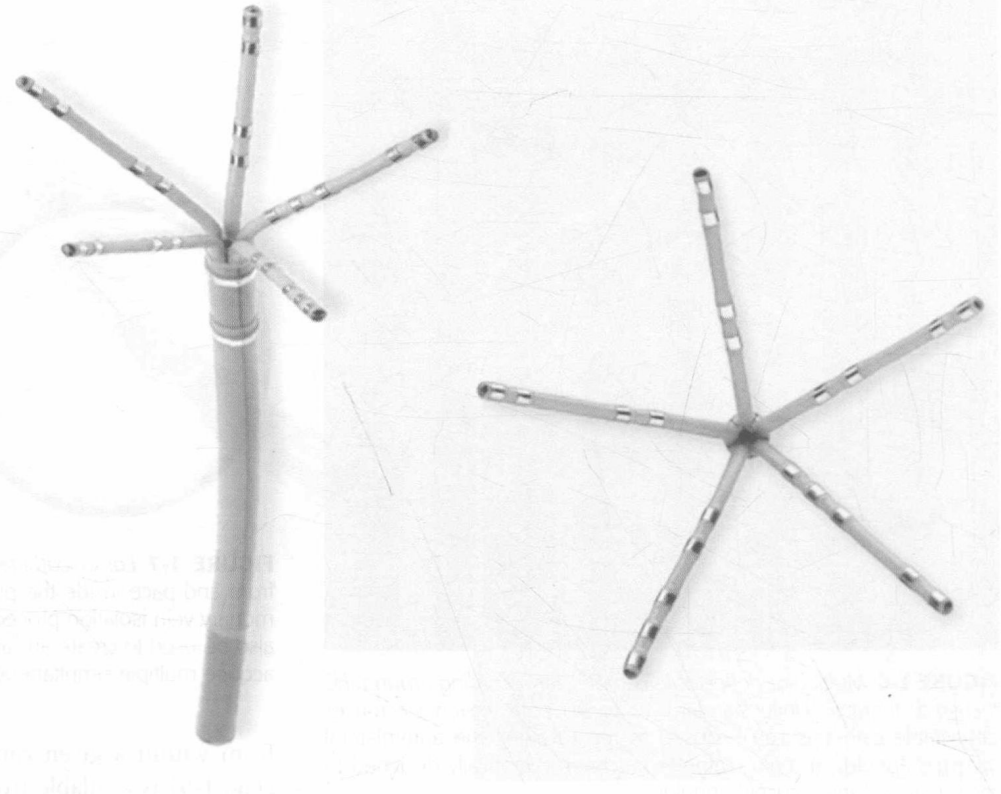

FIGURE 1-9 *PentaRay mapping catheter.* This flexible, 20 pole catheter on 5 splines allows for high-density activation mapping.

the splines sometimes makes for variable contact in misinterpretation of data. More recently Rhythmia Medical (Boston Scientific) has developed a 64 pole roving catheter (Fig. 1-10). This mapping (minibasket) catheter has an 8 F bidirectional deflectable shaft and a basket electrode array (usual mapping diameter 18 mm) with eight splines, each spline containing eight small (0.4 mm^2), low-impedance electrodes (total 64 electrodes). The interelectrode spacing along the spline is 2.5 mm (center-to-center). Mapping can be performed with the basket in variable degrees of deployment (diameter ranging 3 to 22 mm). The location of each of the 64 electrodes is identified by a combination of a magnetic sensor in the distal region of the catheter and impedance sensing on each of the 64 basket electrodes. The location of each basket electrode is obtained whether the basket is fully or only partially deployed. Heparinized saline (1 U/mL) is infused through the central lumen of the catheter shaft at 1 mL/min, emerging at the proximal end of the basket to prevent thrombus. This

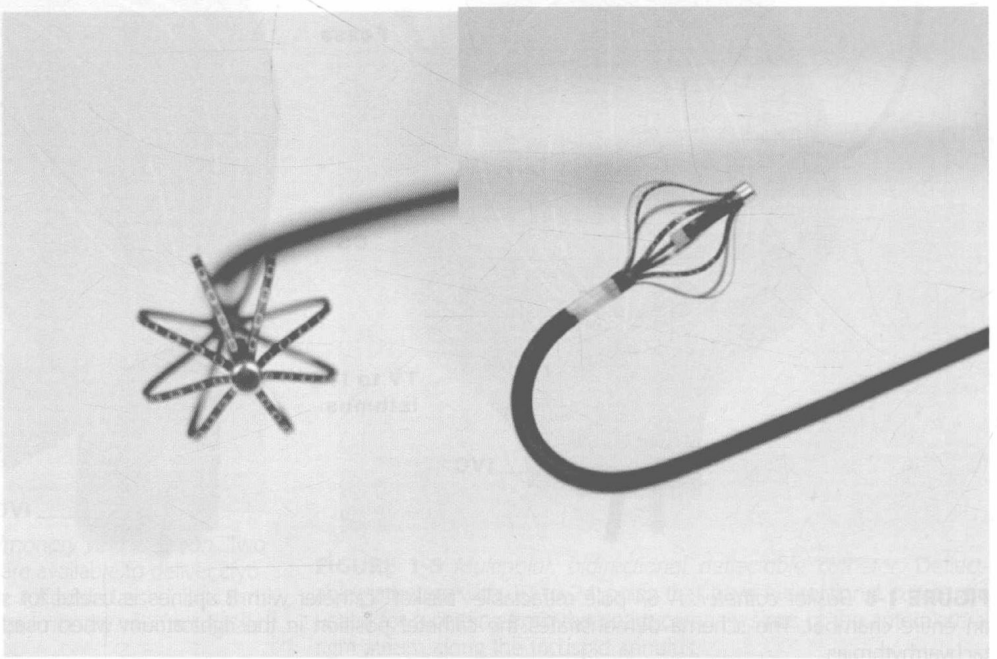

FIGURE 1-10 *New micro basket catheter from Rhythmia (Boston Scientific).* This catheter has a small, flexible basket with 64 poles on 8 splines using small (0.4 mm) low impedance electrodes. It also has bidirectional steering.