

Clinical Cardiac Electrophysiology Fellowship Teaching Objectives for the New Millennium

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Introduction

The field of cardiac electrophysiology has grown and changed dramatically since the last guideline on fellowship training objectives was published in 1988. Not only has the ability to diagnose arrhythmic diseases improved, but also the knowledge of genetic contributions and mechanisms of these disorders has expanded. Moreover, the treatment of these arrhythmias, including ablation and implantable cardioverter defibrillators (ICDs), has forever altered patient management. The purpose of this updated fellowship teaching objective is to outline the basic science, clinical components, and procedural aspects of electrophysiology and to provide relevant references that can be used as an educational guide. This work represents a compilation by experts in the field of electrophysiology but is not intended to give guidelines regarding fellowship requirements outlined in American Board of Internal Medicine (ABIM) and ACGME documents.

This article is organized according to the following sections: (1) historical aspects of electrophysiology; (2) disease mechanisms, including basic electrophysiology and the genetic basis of arrhythmias; (3) diagnosis of arrhythmias, including surface ECG, invasive electrophysiologic evaluation, and laboratory safety; (4) treatment of arrhythmias, including basic pharmacokinetics and pharmacodynamics, ablation of supraventricular tachycardia, atrial fibrillation and flutter, ablation of ventricular tachycardia (VT), pacemakers, and ICDs; and (5) syncope and sudden cardiac death.

Historical Aspects

Fellows should be familiar with the early works of the investigators who laid the foundation and ideas for clinical electrophysiology and whose ideas were the cradle for the future generations of cardiac electrophysiologists.¹⁻³

- A. External and internal pacemaker introduction in the 1960s by Zoll, Bakken, and Furman.

- B. Scherlag and co-workers in 1969 reported on the catheter technique to record His-bundle activity in man.
- C. Wellens published the classic text on the use of programmed electrical stimulation of the heart by intracardiac catheters in the diagnosis and treatment of tachycardias.
- D. Gallagher and Sealy developed and widely applied the endocardial surgical approach to the treatment of patients with the Wolff-Parkinson-White (WPW) syndrome.
- E. Josephson and Harken studying patients with ischemic heart disease and VT developed the technique of endocardial excision, guided by preoperative and intraoperative mapping.
- F. Guiraudon and Klein developed an epicardial approach to the surgical treatment of patients with WPW syndrome.
- G. In 1980, Mirowski and co-workers reported on the implantation of an automatic defibrillator for the management of malignant ventricular arrhythmias in humans.
- H. Intracardiac ablation developed in early 1980s by Scheinman and Gallagher.

References

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3. Zipes DP, Wellens HJ: What have we learned about cardiac arrhythmias? *Circulation* 2000;102:IV-52-IV-57.

Disease Mechanisms

I. Basic Electrophysiology

Fellows should possess an understanding of the basic cellular and molecular mechanisms of electrophysiology. These concepts include the cellular and molecular bases for electrical function of the heart under normal and pathophysiologic conditions, the mechanisms responsible for the development of arrhythmias, and the actions of antiarrhythmic drugs.

- A. Determinants of the normal action potential and normal cardiac rhythm
 1. Genesis of the resting potential in excitable cells¹
 2. Channels and ionic currents responsible for the action potential²

3. Genes encoding for cardiac ion channels, exchangers, and pumps³
4. Electrical heterogeneity⁴
5. Mechanisms of automaticity in nodal and Purkinje pacemakers
- B. Determinants of normal conduction
 1. Structure and function of gap junctions
 2. Passive membrane properties and electrotonic interactions
 3. Anisotropy⁵
 4. Modulation of these factors by sympathetic and parasympathetic agonists
- C. Cellular basis for the inscription of the ECG⁴
 1. QRS
 2. T wave and ST segment
 3. J wave
 4. U wave
- D. Genesis of cardiac arrhythmias^{6,7}
 1. Mechanisms of cardiac arrhythmia
 - a. Abnormalities of impulse formation
 - i. Abnormal automaticity
 - a) Enhanced pacemaker activity
 - b) Protected pacemaker activity (parasystole)
 - ii. Triggered activity
 - a) Early afterdepolarizations
 - b) Delayed afterdepolarizations
 - b. Reentrant arrhythmias
 - i. Circus movement arrhythmia
 - c) Anatomic obstacle (ring model)
 - d) Functional block (leading circle, figure of eight, spiral/rotor)
 - ii. Reflection
 - iii. Phase 2 reentry
 2. Congenital and acquired arrhythmia syndromes
 - c. Gene mutations in ion channels, exchangers, pumps
 - i. Long QT syndrome (LQTS)⁸
 - ii. Brugada syndrome⁹
 - iii. Conduction system disease
 - d. Drug-induced arrhythmia syndromes
 - e. Autonomic nervous system-mediated arrhythmias
 - f. Ischemia- and infarction-related arrhythmias
- E. Antiarrhythmic drug actions
 1. Mechanisms of action of antiarrhythmic drugs
 - a. Drug-receptor interactions
 - b. Effects on conduction, repolarization, automaticity, and contractility¹⁰
 2. Gene-specific pharmacologic therapy³
 3. Modulation of drug effects by metabolic alterations (e.g., pH, and K⁺), rate, or changes in resting potential

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4. Antzelevitch C, Dumaine R: Electrical heterogeneity in the heart: Physiological, pharmacological and clinical implications. In Page E, Fozzard HA, Solaro RJ, eds: *Handbook of Physiology. The Heart*. Oxford University Press, New York, 2000.
5. Spach MS: Anisotropy of cardiac tissue: A major determinant of conduction? *J Cardiovasc Electrophysiol* 1999;10:887-890.
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II. Genetic Basis of Arrhythmias

Identification of the molecular basis of inherited arrhythmias represents one of the most significant advances in cardiovascular research in the past 25 years and illustrates the symbiotic relationship between basic and clinical sciences.¹⁻³ Unraveling the molecular basis of several inherited arrhythmias was instrumental in characterizing certain ionic currents that control cardiac repolarization. To date, most of the focus in genetic research of cardiac arrhythmias is on the monogenetic disorders in which single mutations result in arrhythmias. The much more common issue may be the role of gene polymorphisms in arrhythmia susceptibility and adverse response to drugs.

- A. The fellow should be familiar with the following background materials:
 1. Original descriptions of the LQTS⁴⁻⁶ and Brugada syndromes⁷
 2. Role of specific ion channels in the generation of the normal action potential
 3. Regional differences in the cardiac action potential
 4. Relationship between the action potential and the surface ECG
- B. Role of specific ion channel mutations in the genesis of arrhythmias
 1. Inherited defects in potassium channel and their subunits as a basis for LQTS⁶
 2. Inherited defects in sodium channels as a basis for LQT3 and Brugada syndrome¹⁻³
- C. Additional areas of investigation that are under active research.
 1. Lev-Lenegre disease
 2. Atrial fibrillation⁸
 3. WPW syndrome⁹

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4. Wide QRS complex tachycardia with specific attention paid to distinguishing VT from supraventricular tachycardia with aberrant ventricular conduction and ventricular preexcited tachycardias. Specific ventricular arrhythmias include:
 - a. Ventricular ectopy with specific attention to bigeminy and parasystole.
 - b. Nonsustained VT (monomorphic or polymorphic) with or without associated long QT intervals
 - c. Sustained VT with particular attention to idiopathic VT versus tachycardia associated with organic heart disease
 - d. Sustained polymorphic VT with or without long QT intervals
 - e. Bidirectional VT and other drug-induced tachycardias

E. Miscellaneous arrhythmogenic situations

1. LQTS; specific patterns of T waves related to gene defect
2. Brugada syndrome
3. Arrhythmogenic right ventricular dysplasia
4. Idiopathic ventricular fibrillation

Diagnosis of Arrhythmias

I. Surface ECG of Arrhythmias

Fellows should meet minimum competency requirements of a general cardiology fellow for ECG interpretation as outlined by the American College of Cardiology Task Force.¹ Fellows are expected to be fully familiar with the indications and performance of a variety of noninvasive ECG tests.

- A. Surface ECG interpretation
 1. Evaluation of normal and abnormal intervals
 2. Recognition of myocardial infarction and evidence of ischemia
 3. Metabolic/drug effects
 4. Conduction disturbances
 5. Identification of accessory AV connection location
- B. Noninvasive ECG tests
 1. Ambulatory ECG recordings
 2. Continuous on-line ECG monitoring (telemetry)
 3. Manually and automatically activated event recorders
 4. Analysis of signal-averaged ECGs
 5. Exercise testing for the presence of myocardial ischemia and/or arrhythmias
 6. Heart rate variability as a risk stratifier
 7. T wave alternans as a risk stratifier
- C. Bradyarrhythmias
 1. Abnormalities of sinus node function, including inappropriate sinus bradycardia, sinus arrest, and sinus exit block
 2. Abnormalities of AV conduction and their localization to the AV node, His bundle, or His-Purkinje system
- D. Tachyarrhythmias
 1. Recognition of atrial tachycardia, reentrant arrhythmias using manifest or concealed AV connection pathways, and AV nodal reentrant tachycardias (AVNRT)
 2. Typical and reverse typical (isthmus dependent) atrial flutter, lesion reentry, left atrial, and atypical flutters²
 3. Atrial fibrillation with particular reference to focal origins

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II. Invasive Electrophysiologic Evaluation

Electrophysiology fellows should be able to list the indications for invasive electrophysiologic studies; perform a comprehensive electrophysiologic study; interpret the data derived from such studies; indicate the sensitivity and specificity of these findings; and integrate these findings into the clinical care of the patient.

- A. Principles of recording
 1. Genesis of the intracardiac recording
 2. Types of recordings
 - a. Unipolar
 - b. Bipolar
 - i. Effect of electrode spacing
 - ii. Effect of electrode orientation (linear, orthogonal)
 3. Filtering
 - a. Bandpass and its effect on signal content
 - b. Notch filter
 4. Gain and clipping of recorded signal
 5. Electrode field and concept of local activation time
- B. Principles of stimulation
 1. Concept of threshold
 2. Types of stimulation and implications for pace mapping
 - a. Unipolar
 - b. Bipolar
- C. Sinus node function

1. Obtaining and evaluating the sinus node recovery time, sinus atrial conduction time, and sinus node electrogram^{1,2}
- D. AV node and AV nodal-dependent tachycardias
 1. Intra-atrial versus AV nodal conduction disease³
 2. AV nodal function and dysfunction in response to atrial overdrive pacing⁴
 3. AVNRT
 4. AV reentrant tachycardia
- E. His-Purkinje system
 1. Diagnose conduction delay or block as within the AV node or His-Purkinje system, and integrate this information with regard to need for permanent cardiac pacing⁵
- F. Ventricular arrhythmias
 1. Detail the number of sites and standard stimulation protocol for induction of VT
 2. Recognize the value of His-bundle and atrial recordings in the diagnosis of VT and/or bundle branch reentry
 3. Detail the sensitivity and specificity of induction protocols with respect to clinical presentation (VT, sustained and nonsustained, or ventricular fibrillation) and underlying myocardial substrate⁶
- G. Principles of resetting of tachycardias
 1. Single-beat resetting methods
 2. Resetting curves and their interpretation
 - a. Flat
 - b. Increasing
 - c. Mixed flat and increasing
 - d. Decreasing
 3. Use of resetting in mapping
- H. Principles of entrainment of tachycardias^{7,8}
 1. Definition of entrainment⁸⁻¹⁰
 - a. Constant fusion during pacing at a constant rate faster than the tachycardia except for the last captured beat that is not fused
 - b. Progressive fusion
 - c. Interruption of tachycardia by overdrive pacing associated with localized conduction block to a site followed by activation of that site by the next pacing impulse from a different direction and with a shorter conduction time
 - d. Change in conduction time and electrogram morphology at one recording site when pacing from another site at two different constant pacing rates, each of which is faster than the spontaneous rate of the tachycardia, but fails to interrupt it (electrogram equivalent of progressive fusion)
 2. Techniques of entrainment
 - a. Pacing technique and assurance of capture
 - b. Types of ECG/intracardiac fusion and their implications
 - i. Fixed fusion
 - ii. Progressive fusion
 - iii. Concealed entrainment
 3. Implications of entrainment
 - a. Presence of reentry
 - b. Significance for mapping

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III. Laboratory Safety

In the laboratory, procedures utilize radiation and include the potential for transmission of disease. Therefore, electrophysiology fellows should be aware of laboratory safety as related to radiation exposure to patients, self, and other staff, and the risks and prevention of transmissible disease.

- A. Radiation exposure¹⁻³
 1. Principles of radiation safety
 2. Reduction of exposure to medical personnel
 3. Precautions for medical employees during pregnancy
 4. Radiation risks for patients
 - a. Cancer
 - b. Genetic defects
 - c. Radiation-induced skin injury
- B. Precautions for transmissible diseases
 1. Hepatitis, human immunodeficiency virus, spongiform encephalopathy
 2. Shielding from bodily fluids
 3. Needle precautions
 4. Disposal of contaminated supplies
 5. Treatment and follow-up of medical personnel after exposure to bodily fluids

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Treatment of Arrhythmias

I. Basic Pharmacokinetics and Pharmacodynamics

Pharmacokinetics and pharmacodynamics are essential concepts in understanding therapeutic drug delivery. Although drugs are less commonly used clinically as a first-line basis for ventricular arrhythmias, they frequently are utilized as adjunctive therapy for ventricular arrhythmias. In addition, antiarrhythmic agents will likely continue to be frequently utilized for atrial fibrillation.

- A. Basic pharmacokinetic principles^{1,2}
 1. Absorption of drugs from the gastrointestinal tract and from intramuscular sites
 - a. Effects of lipid solubility/ionic charge
 - b. Effects of drugs that alter gastrointestinal blood on drug absorption
 2. Distribution of drugs after administration
 - a. Concepts of apparent volumes of distribution and absence of their relationships to anatomic volumes
 - b. Effects of tissue and plasma protein binding
 3. Metabolism of drugs by hepatic and extrahepatic mechanisms
 - a. First-pass hepatic metabolism and its effects of oral medications
 - b. Role of the cytochrome P450 system and other metabolic pathways in drug metabolism
 - c. Pharmacogenetic patterns that may influence drug metabolism
 - d. Induction and inhibition of drug metabolism by diet, alcohol, and other drugs
 4. Elimination of drugs by liver, kidney, and lungs
 - a. Relationship between elimination rate constant and $t_{1/2}$
 - b. Clinical relevance of dose-dependent (or concentration-dependent) kinetics (saturable processes)
 - c. Role of P glycoprotein and other drug transport systems
- B. Clinical pharmacokinetics³⁻⁶
 1. Dosing recommendations and therapeutic decisions based on the following concepts:
 - a. Clearance as a determinant of steady-state plasma concentration (renal, nonrenal, and systemic)
 - b. Half-life time relationship to time to steady state
 - c. Basic concepts of compartmental analysis
 - d. Use and abuse of loading doses
 - e. Principles of therapeutic drug level monitoring
 - f. Drug interactions
 2. Information necessary to consider the effect of the following factors on pharmacokinetics in selecting drugs and drug dosages
 - a. Age
 - b. Gender
 - c. Renal function
 - d. Hepatic function
 - e. Cardiac disease

- C. Pharmacodynamics: Basic concepts of drug actions
 1. Receptor theory
 2. Log dose-response relationships
 3. Agonists
 4. Antagonists (competitive and noncompetitive)
 5. Interactions of drugs with ionic channels
- D. Pharmacodynamics and pharmacokinetics of antiarrhythmic drugs
 1. Utility and limitations of various classification schemes: Vaughan-Williams, Sicilian Gambit, etc.⁷
 2. Effects of drugs on cardiac ion channels
 3. Differential effects of drugs on conduction and refractoriness of atrial and ventricular myocardium, bypass tracts, and His-Purkinje tissue
 4. Effects of drugs in situations with potentially abnormal electrophysiology, e.g., abnormal repolarization syndromes, ischemia, hypertrophy
 5. Effects of drugs on atrial and ventricular defibrillation thresholds
 6. The following information for individual antiarrhythmic agents in normal subjects and in patients with renal, hepatic, or heart failure:
 - a. Indications
 - b. Contraindications
 - c. Dosages
 - d. Drug interactions
 - e. Diagnosis and management of potential adverse effects
 - f. Pathways for metabolism and elimination
 - g. Range of expected elimination $t_{1/2}$
- E. Drug-device interactions
 1. The trainee should be familiar with the effects of cardiovascular drugs on both sensing and therapeutic functions of pacemakers and ICDs.⁸

References

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8. Movsowitz C, Marchlinski FE: Interactions between implantable cardioverter-defibrillators and class III agents. *Am J Cardiol* 1998; 82(4A):41I-48I.

II. Ablation of Supraventricular Tachycardia

A knowledge of which patients should undergo radiofrequency ablation and of the techniques and endpoints for ablation should be well known to clinical electrophysiology fellows.¹ In addition, the techniques of ablation, including mapping, knowledge of newer mapping technologies, ret-

rograde access to the left atrium and ventricle, and trans-septal puncture should be familiar to the fellows.

- A. Patient selection and preablation work-up, including cardiac monitoring, exercise stress testing, echocardiography, and even cardiac catheterization in selected individuals
- B. Equipment
 1. Catheters types and associated curves
 2. Sheaths, including curves and uses
 3. Energy sources
- C. Anatomy²
 1. Epicardial coronary vessels
 2. Atrial anatomy
 - a. AV node and His bundle
 - b. Triangle of Koch
 - c. Isthmus
 - d. Crista terminalis
 - e. Pulmonary veins
 - f. Coronary sinus
 - g. Sinus node
- D. Techniques and endpoints
 1. AVNRT^{3,4}
 - a. Types
 - i. Slow/fast
 - ii. Fast/slow
 - iii. Slow/slow
 - iv. Other variants
 - b. Ablation of AVNRT
 - i. Slow pathway (preferred method)
 - ii. Fast pathway
 - c. Mapping techniques for radiofrequency ablation
 - i. Slow potential guided
 - ii. Anatomically guided
 - d. Relative accuracy of endpoints of ablation
 - i. Noninducibility
 - ii. Junctional AV beats during ablation
 - iii. Elimination of slow pathway
 2. AV reentrant tachycardia^{5,7}
 - a. Types
 - i. Orthodromic
 - ii. Antidromic
 - iii. Mahaim fibers
 - b. Mapping techniques
 - i. Anterograde activation mapping of the earliest ventricular activity
 - ii. Retrograde activation mapping of the earliest atrial activity
 - c. Left-sided approaches
 - i. Transseptal
 - ii. Transaortic valve retrograde
 3. Atrial tachycardia⁸
 - a. Locations are frequently pulmonary vein insertions and crista terminalis
 - b. Mapping
 - c. Endpoints
 4. Atrial flutter and atrial fibrillation (see appropriate sections)
- E. Maneuvers to distinguish supraventricular tachycardia mechanism⁹
 1. Classification schemes
 - a. Long RP versus short RP
 - b. Wide QRS complex versus narrow QRS complex

2. Maneuvers

- a. His refractory ventricular pacing (for wide complex tachycardia) and atrial pacing (for narrow QRS complex)
 - b. Changes in tachycardia cycle length and VA time with bundle branch block
 - c. Para-Hisian pacing
- F. Complications related to supraventricular tachycardia ablation
 1. Risk of heart block
 2. Risk of stroke
 3. Pericardial tamponade

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III. Atrial Fibrillation and Flutter

Fellows should possess a sound understanding of atrial fibrillation and flutter. The understanding and therapy of atrial fibrillation is an area of intense research and fellows are expected to be aware of developments as they relate to clinical practice. Technical skills in atrial fibrillation ablation would be an ideal goal.

A. Atrial fibrillation

1. Mechanisms^{1,2}

- a. Multiple reentrant wavelets
- b. Fibrillatory conduction from a single rapid focus or reentrant circuit of very short cycle length
- c. Atrial remodeling

2. Epidemiology

- a. Age-associated prevalence
- b. Associated clinical states (hypertension, valvular disease, congestive heart failure, coronary artery disease, post open heart surgery, etc.)
3. Associated potential risks
 - a. Systemic embolism and stroke
 - b. Tachycardia-mediated cardiomyopathy
 - c. Increased mortality
4. Diagnosis
5. Therapy^{3,4}
 - a. Anticoagulation
 - i. Five primary prevention trials and their meta-analysis^{5,6}
 - ii. Warfarin to achieve international normalized ratio (INR) of 2 to 3; risks and benefits
 - iii. Questionable value of aspirin
 - b. Antiarrhythmic drugs (suppression)⁷
 - c. Ventricular rate control
 - i. Beta-adrenergic blocking agents, calcium channel blockers, digoxin
 - ii. AV node/His-bundle ablation
 - d. Devices
 - i. Atrial pacing
 - ii. Atrial defibrillation⁸
 - e. Ablation
 - i. Surgical maze
 - ii. Endocardial catheter techniques
 - a) Maze equivalent and its variants (linear ablations)
 - b) Focal ablation (pulmonary vein, superior vena cava, crista terminalis, etc.)^{9,10}
 - c) Pulmonary vein isolation^{11,12}
 - f. Cardioversion
 - i. Transthoracic
 - ii. Intrathoracic
 - iii. Pre and post anticoagulation

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- initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. *N Engl J Med* 1998;339:659-666.
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- B. Atrial flutter
 1. Nomenclature and mechanisms¹⁻⁴
 - a. Typical
 - b. Reverse typical
 - c. Lesion
 - d. Left atrial
 - e. Atypical (upper loop, etc.)
 2. Epidemiology
 3. Associated potential risks
 - a. Systemic embolism and stroke⁵
 - b. Tachycardia-mediated cardiomyopathy
 - c. Development of atrial fibrillation
 4. Diagnosis
 5. Therapy
 - a. Anticoagulation
 - i. Five primary prevention trials and their meta-analysis
 - ii. Warfarin: INR 2 to 3; risks and benefits
 - iii. Questionable value of aspirin
 - b. Antiarrhythmic drugs (suppression)
 - c. Ventricular rate control
 - i. Drugs
 - ii. AV node/His-bundle ablation
 - d. Devices
 - e. Ablation⁶⁻⁸
 - i. Mapping techniques to identify nature of atrial flutter, its anatomically vulnerable site, and demonstration of postablation efficacy
 - ii. Importance of subeustachian isthmus block for isthmus-dependent flutter
 - f. Cardioversion
 - g. Antitachycardia pacing

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IV. Ablation of VT

Fellows should recognize the indications, patient selection, and risks and anticipated benefits of catheter ablation in VT associated with or without structural heart disease. It is reasonable to expect cure for the majority of patients with truly idiopathic VT, whereas catheter ablation usually is palliative in organic heart disease, especially in advanced ischemic heart disease.

A. Idiopathic outflow tract VT¹⁻³

1. Sites
 - a. Right ventricular outflow tract
 - b. Left ventricular outflow tract
 - c. Anterobasal left ventricle
2. Mapping techniques
 - a. Activation sequence mapping; requires nonsustained or sustained VT
 - b. Pace mapping
 - c. Left ventricular outflow tract mapped via retrograde transaortic or transseptal approach

B. Idiopathic left VT^{2,3}

1. Distinct ECG pattern of "right bundle, left axis" with site in an inferior/septal/apical area
2. Mechanism: Reentry incorporating portions of the His-Purkinje system
3. Principles of mapping and ablation
 - a. Activation mapping: Purkinje potential
 - b. Entrainment mapping
 - c. Pace mapping

C. Bundle branch reentry⁴

1. Generally occurs in patients with diffuse conduction system disease and nonischemic cardiomyopathies
2. Role of different maneuvers, comparison of HH and VV intervals

D. Catheter ablation of postmyocardial infarction VT⁵⁻¹¹

1. Most appropriate as palliative therapy for frequent VT in patients with implantable defibrillators
2. Mechanism is reentry using pathways within and around large scars
3. Optimal approach is entrainment mapping during hemodynamically tolerated VT
4. Significance, sensitivity, and specificity of the following parameters:
 - a. Mid-diastolic potentials not dissociable from VT
 - b. Concealed entrainment
 - c. Postpacing intervals
 - d. Stimulus-QRS/local electrogram: QRS comparisons during entrainment and VT

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V. Pacemakers

Knowledge of indications for temporary and permanent pacing and the technical ability to place both temporary and permanent pacemakers are critical elements of the training of a clinical electrophysiologist.^{1,2}

A. Temporary pacing

1. Indications³
2. Techniques

B. Indications for permanent pacemaker implantation³

C. Natural history of various bradyarrhythmias

D. Evaluation techniques

E. Effects of medications on sinus and AV node function

F. Implantation (while minimal requirements of training program do not necessitate provision of implantation experience sufficient to perform pacemaker implantation, exposure adequate to assure a thorough understanding of concepts is critical)

1. Anatomic considerations

- a. Normal venous anatomy
- b. Abnormal venous anatomy
 - i. Persistent left superior vena cava syndrome
 - ii. Subclavian occlusion

2. Pocket location options

3. Venous access techniques

4. Cardiac sites for pacing

5. Lead testing

6. Pacing system analyzers

7. Sensing evaluation

8. Stimulation thresholds, including strength duration curves
9. Impedance measurements
- G. Follow-up
 1. Transtelephonic
 2. Clinic
 3. Troubleshooting
- H. Pacing system technology
 1. Leads (including electrodes, fixation, insulation, conductors, and connectors)
 2. Pulse generators (including batteries, circuitry, sensors, and function, including monitoring)
 3. Modes (including codes)
- I. Hemodynamics of pacing⁴
- J. Complications of pacing (including management)
- K. Lead extraction
 1. Indication
 2. Techniques
 3. Complications
- L. Device-device, device-drug, device-environmental interactions

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VI. Implantable Cardioverter Defibrillators

A training program in clinical cardiac electrophysiology should include sufficient technical and clinical instruction in ICDs to allow the fellow to provide highly competent clinical assessment, patient selection, ICD implantation, and follow-up of patients with cardiac arrhythmias.

- A. Technologic and engineering principles¹⁻⁴
 1. Leads and programmers as applied to the practice of cardiac electrophysiology
 2. Basic design and function of the ICD
 3. Defibrillation waveforms
 4. Lead systems
 5. Detection enhancements
 6. Diagnostic storage capabilities
- B. Indications and implantation guidelines for the ICD and appropriate integration of results of prospective trials into clinical decision-making⁵⁻⁹
- C. American College of Cardiology/American Heart Association (ACC/AHA), North American Society of Pacing and Electrophysiology (NASPE) guidelines for implantation of ICDs⁸
- D. Implantation techniques and testing of ICD system function at implantation¹⁻⁴
- E. Indication and implantation of coronary sinus leads to enable biventricular pacing

- F. Drug-ICD and pacemaker-ICD interaction¹⁻⁴
- G. Prevention of pacemaker-ICD interactions
- H. Complications¹⁻⁴
 - I. Follow-up and troubleshooting¹⁻⁴
 - J. Recommendations for driving for the ICD patient¹⁰
- K. Cost-effectiveness of the ICD^{2,4}
- L. Quality of life and psychosocial impact of the ICD^{2,4}

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Special Conditions

I. Syncope

It is critical for the fellow to know that cardiac syncope carries a worse prognosis than other causes of syncope, and that syncope in the presence of cardiac disease may be a harbinger of impending sudden cardiac death.¹⁻³ Also important is the distinction between syncope occurring in individuals with and those without structural heart disease.

- A. Value of history, physical examination, and work-up for structural heart disease^{2,4}
- B. Neurocardiogenic syncope
 1. Diagnosis⁵
 2. Sensitivity and specificity of tilt table test⁶
 3. Treatment⁷
 - a. Behavioral
 - b. Salt and fluids
 - c. Tilt training

- d. Pharmacologic
- e. Pacemaker
- C. Bradyarrhythmias
 1. Poor sensitivity of invasive electrophysiologic testing for diagnosis of bradycardias^{8,9}
 2. Usefulness of implantable event monitors and long-term rhythm monitoring
- D. Tachyarrhythmias
 1. Sensitivity and specificity of invasive electrophysiologic testing in various types of structural heart disease^{8,10}
 - a. Ischemic heart disease
 - b. Idiopathic dilated cardiomyopathy
 - c. Hypertrophic cardiomyopathy
 - d. Arrhythmogenic right ventricular dysplasia
 - e. No structural heart disease
- E. Indications for permanent pacemakers¹¹
- F. Indications for ICDs¹¹

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- A. Definition and magnitude of the problem^{1,2}
 1. Epidemiology
 2. Risk factors
 - B. Arrhythmic mechanisms
 1. VT and ventricular fibrillation³
 2. Torsades de pointes⁴
 3. Genetic ion channel abnormalities
 - a. LQTS⁵
 - b. Brugada syndrome⁶
 4. Bradyarrhythmias (especially in patients with marked heart failure)⁷
 - C. Precipitants^{1,2}
 1. Myocardial ischemia
 2. Electrolyte abnormalities
 3. Autonomic nervous system abnormalities⁸
 4. Congestive heart failure
 5. Medications and role of proarrhythmia^{4,9}
 - D. Management
 1. ACLS guidelines¹⁰
 2. Evaluation and therapy of structural heart disease and ischemia, including echocardiography, exercise testing, cardiac catheterization, and indications for revascularization
 3. Role, sensitivity, and specificity of electrophysiologic testing to guide therapy
 4. Role of ICD therapy¹¹
 5. Role of pharmacologic therapy
 6. Role of ablation and surgical therapies
 7. Role of ancillary therapies (e.g., beta-blockers, angiotensin-converting enzyme inhibitors, anti-ischemic therapies, etc.)
 - E. Risk stratification
 1. Role, sensitivity, and specificity of programmed ventricular stimulation in patients with coronary artery disease, reduced left ventricular ejection fraction, and asymptomatic nonsustained VT^{12,13}
 2. Role, sensitivity, and specificity of programmed ventricular stimulation in patients with nonischemic cardiomyopathy
 3. Role, sensitivity, and specificity of heart rate variability, signal-averaged ECG, T wave alternans, and QT dispersion to prognosticate risk in specific patient populations¹
 4. Specific issues in athletes, including patient evaluation and limitation of activities¹⁴
 5. Specific considerations in patients with hypertrophic cardiomyopathies¹⁵
 6. Provocative maneuvers in patients with possible genetic ion channel abnormalities
 - F. Completed clinical trials
 1. Antiarrhythmics Versus Implantable Defibrillators Trial (AVID)¹⁶
 2. Canadian Implantable Defibrillator Study (CIDS)¹⁷
 3. Cardiac Arrest Study Hamburg (CASH)
 4. Multicenter Automatic Defibrillator Implantation Trial (MADIT)¹³
 5. Multicenter Unsustained Tachycardia Trial (MUSTT)¹²
 6. Congestive Heart Failure Survival Trial of Antiarrhythmic Therapy (CHF-STAT)¹⁸
 - G. Clinical trials in progress
 1. Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT)

II. Sudden Cardiac Death

Electrophysiology fellows should be cognizant of the arrhythmic mechanisms that can cause sudden death; precipitants for arrhythmogenesis; evidence-based management of patients experiencing sudden death; role of pharmacologic and nonpharmacologic therapies; and principles of risk stratification and prophylactic approaches to patients who have not yet experienced sudden death.

2. Defibrillators in Nonischemic Cardiomyopathy Treatment Evaluation (DEFINITE)
3. Multicenter Automatic Defibrillator Implantation Trial (MADIT) II

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