

**Practical
Cardiac
Electrophysiology**

Jaypee Brothers

Practical Cardiac Electrophysiology

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Practical Cardiac Electrophysiology

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Dedicated to

*My mother, Dr Satya Bhargava, a classical singer and an author in the field of music,
a true example of dedication and determination, for being a constant source of inspiration;
my wife, Rekha Bhargava, for her patience and unconditional support and
my lovely daughters, Devpriya and Shivpriya, for allowing me to devote time
that should have been rightfully theirs.*

—Kartikeya Bhargava

*My mother-in-law, Kamala Aravamudan (née Ramanujam),
a wonderful person whose nature is to be kind and pleasant yet has the strength and
persistence to say and do what may be difficult but needed for the betterment of those around her.*

—Samuel J Asirvatham

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Foreword

Drs Kartikeya Bhargava and Samuel J Asirvatham have carefully selected a well-known group of international experts to contribute to this multi-authored, comprehensive and up-to-date textbook of cardiac electrophysiology. *Practical Cardiac Electrophysiology* is largely clinically oriented and constitutes 47 chapters covering the spectrum of clinical diagnosis and management of arrhythmias, in and out of the electrophysiology laboratory. There is extensive coverage of all our “tools” including mapping equipment, ablation catheters and lab setup. There is an excellent chapter on practical cardiac anatomy, a must read for the serious student of the electrophysiology.



The book not only covers the most current fashionable entities and procedural skills, but also covers the less glamorous but necessary areas such as sinus node function testing.

This is not a “quick read” but individual chapters can be used as an excellent starting point for studying an area of interest for the electrophysiologist be they novice or more experienced. It would also serve well as a basis for study for board review as there is virtually no area of clinical electrophysiology not covered.

Overall, a useful addition to the shelf of any serious student of electrophysiology.

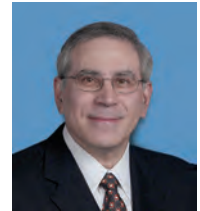
A handwritten signature in black ink, appearing to read 'G Klein', written in a cursive style.

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Jaypee Brothers

Foreword

I was asked to write a Foreword for *Practical Cardiac Electrophysiology* edited by Drs Kartikeya Bhargava and Samuel J Asirvatham. This book contains 47 chapters authored by experts from around the world and includes topics as basic as how to do an electrophysiology study to complex imaging techniques and approaches to ablation of supraventricular and ventricular arrhythmias. While I admit, I had an opportunity to do a cursory journey through the various chapters in this textbook, my role is not one of a reviewer. Rather, I will address a more fundamental question, why bother to do such a project.



I grew up in an era of medical education where we “cherished” our textbooks. The chapters were read, key sections underlined, often reread, and kept on a shelf for ready reference. It was important to read journals to keep abreast of new observations (actually, not so new by the time the journal arrived). However, during teaching rounds, quotes from Friedberg’s or Hurst’s Textbook of Cardiology reigned supreme. The years moved on and a few specialty textbooks in electrophysiology became available, including one from my co-author Dr George Klein and me. Scores of journals entered the cardiovascular space, several specializing in cardiac arrhythmias. But in the distance, a looming shadow appeared that produced a sea change in how we access information: The Internet.

What a marvelous educational tool the Internet is, constantly available at your fingertips and nearly always willing to answer your queries. A search of a topic can not only provide the latest literature on it but also an abundance of non-vetted information of questionable worth – good luck on sorting through it! There are more blogs and commentary sites than “Carter has Little Liver Pills” (you youngsters will need to search the Internet for that reference). Still, it is an incredible fountain of knowledge, the modern-day Pierian Spring.

So, I ask again, why bother assembling more than 2 score chapters from even more authors yielding hundreds of pages of information, even if it can be put into an electronic format? The reason is that reference books such as this are needed and provide a cohesive source of information for a novice or expert in clinical electrophysiology. The chapters and authors have been “vetted” by two accomplished electrophysiologists, Dr Asirvatham, who is one of the world’s leading educators and a past recipient of the Distinguished Teacher award from Heart Rhythm Society (HRS), and Dr Kartikeya Bhargava. Thus, the reader has a single reference source to answer most questions about cardiac arrhythmias. Any such textbook will be somewhat out of date by the nature of how fast our field is moving, but in my experience this accounts, mostly for changes in therapy or sometimes an ablation technique, but not in the core principles of our field. I previously stated that my responsibility is not to review the content of this thorough textbook, but I must admit that I did do more than a “peek” in some of the chapters. I was delighted to see that the authors used “AV node-dependent arrhythmias” in one of their overall sections, a term that we initially used in our textbook in 1994, and I have found this is a useful way to teach concepts of supraventricular arrhythmias.

In summary, my congratulations to the editors for compiling such a complete and excellent resource for clinical electrophysiologists. It is worth having on your electronic bookshelf.

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Preface

“Education is not the learning of facts, but the training of the mind to think.”

—Albert Einstein

The complexity of cardiac electrophysiology is simultaneously a source of never-ending challenge and ever-fulfilling satisfaction for practitioners of this art. To attempt good invasive electrophysiology practice without learning the facts and being conversant in the fundamental principles is futile. Yet, the cornerstones themselves are insufficient in guiding a practitioner through the impasse between success and complication. This textbook begins with a recognition that the basics of anatomy, physiology, biophysics, and electrocardiography require mastery before progress can be made. In addition, the focus on practical understanding and training the electrophysiologist’s mind to be able to apply these principles in real-time when confronted by challenging arrhythmias permeates the book.

There already exist outstanding textbooks of electrophysiology which are often comprehensive treatises or collected case studies. The present work, we hope, benefits all practitioners; those in the developing world may stand to benefit the most. The large number of patients, sometimes suboptimal resources, and in certain cases the lack of access to the standard books and journals have been kept in mind by keeping this book practical and easy to use.

We acknowledge the time and effort of an international panel of master electrophysiologists, who have authored the works that reflect their specific areas of expertise.

Extensive illustrations, case-based discussions, and brief summaries provided at the end of most chapters will provide a perspective on the topic covered in the chapter and guide the readers in applying this information in their daily work.

Kartikeya Bhargava
Samuel J Asirvatham

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List of Abbreviations

18-F-FDG	Flourine-18 Fluorodeoxyglucose	BrS	Brugada Syndrome
3D	Three Dimensional	BT	Bypass Tract
AAD	Anti-arrhythmic Drug	CA	Cardiac Arrest
AAV	Adeno-associated Virus	CICR	Calcium induced Calcium Release
ACE	Angiotensin Converting Enzyme	CAD	Coronary Artery Disease
ACLS	Advanced Cardiac Life Support	cAMP	Cyclic Adenosine Monophosphate
ACTN-2	Alpha Actinin-2	CASPER	Cardiac Arrest Survivors with Preserved Ejection Fraction Registry
AEF	Atrioesophageal Fistula	CCAABV	Congenital Complete Atrioventricular Block
AEGM	Atrial Electrogram	CCB	Calcium Channel Blocker
AF	Atrial Fibrillation	CCW	Counter-clockwise
AFI	Atrial Flutter	CF	Contact Force
AFP	Atriofascicular Pathway	CFAE	Complex Fractionated Atrial Electrograms
AHA	American Heart Association	CFB	Central Fibrous Body
AIV	Anterior Interventricular Vein	CHD	Congenital Heart Disease
ALARA	As Low As Reasonably Achievable	CHF	Congestive Heart Failure
AMC	Aortomitral Continuity	CL	Cycle Length
AMP	Adenosine Mono Phosphate	CMIV	Cox-Maze IV Procedure
ANS	Autonomic Nervous System	CMP	Cardiomyopathy
AP	Accessory Pathway	CMP	Cox-Maze Procedure
APL	Action Potential	CMR	Cardiac Magnetic Resonance Imaging
APC	Atrial Premature Contraction	CMRR	Common-Mode Rejection Ratio
APD	Atrial Premature Depolarization	CPVT	Catecholaminergic Polymorphic Ventricular Tachycardia
ARI	Activation Recovery Intervals	CRD	Cournard
ARP	Absolute Refractory Period	CRT	Cardiac Resynchronization Therapy
ART	Antidromic Reciprocating Tachycardia	CS	Coronary Sinus
ARVC	Arrhythmogenic Right Ventricular Cardiomyopathy	CT	Crista Terminalis
ARVD/C	Arrhythmogenic Right Ventricular Dysplasia/ Cardiomyopathy	CTB	Cardiac Tuberculosis
ASC	Aortic Sinus Cusps	CTCA	Computed Tomography Coronary Angiogram
AT	Atrial Tachycardia	CTI	Cavo-Tricuspid Isthmus
ATP	Antitachycardia Pacing	CW	Clockwise
ATT	Antitubercular Treatment	DAD	Delayed Afterdepolarization
AV	Atrioventricular	DC	Direct Current
AVN	Atrioventricular Node	DCM	Dilated Cardiomyopathy
AVNERP	Atrioventricular Nodal Effective Refractory Period	DE	Delayed Enhancement
AVNRT	Atrioventricular Nodal Reentrant Tachycardia	DPP-6	Dipeptidyl aminopeptidase-like protein-6
AVRT	Atrioventricular Reciprocating or Reentrant Tachycardia	DS	Desmosomal
AWP	Alternating Wenckebach Periods	DSC	Desmocollin
BB	Bundle Branch	DSG	Desmoglein
BBB	Bundle Branch Block	DSP	Desmoplakin
BBR	Bundle Branch Reentry	DSP	Digital Signal Processing
BBRVT	Bundle Branch Reentrant Ventricular Tachycardia	DWR	Double Wave Reentry
BLS	Basic Life Support	EAD	Early Afterdepolarization
		EAM	Electro-anatomical Map/mapping
		EAT	Ectopic Atrial Tachycardia
		ECG	Electrocardiogram

ECGI	Electrocardiographic Imaging	LAD	Left Anterior Descending
ECMO	Extra Corporeal Membrane Oxygenation	LAFB	Left Anterior Fascicular Block
EGM	Electrogram	LAO	left Anterior Oblique
E-IDC	Electrograms with Isolated Delayed Components	LASER	Light Amplification by Stimulated Emission of Radiation
EMB	Endomyocardial Biopsy	LAVA	Local Abnormal Ventricular Activity
EP	Electrophysiology	LB	Left Bundle
EPS	Electrophysiology Study	LBB	Left Bundle Branch
ER	Early Repolarization	LBBB	Left Bundle Branch Block
ERP	Early Repolarization Pattern	LCC	Left Coronary Cusp
ERP	Effective Refractory Period	LCx	Left Circumflex
ERS	Early Repolarization Syndrome	LF	Left Fascicle
ES	Electrical Storm	LGE	Late Gadolinium Enhancement
EUS	Electrically Unexcitable Scar	LICU	Low Intensity Collimated Ultrasound
FDG	Fluorodeoxyglucose	LLR	Lower Loop Reentry
FO	Fossa Ovalis	LMCA	Left Main Coronary Artery
FP	Fast Pathway	LMWH	Low Molecular Weight Heparin
fQRS	Fragmented QRS	LN	Lymph Node
FRP	Functional Refractory Period	LOM	Ligament of Marshall
GA	General Anesthesia	LP	Late Potential
GCV	Great Cardiac Vein	LPF	Low Pass Filter
Gd	Gadolinium	LPFB	Left Posterior Fascicular Block
GM	Granulomatous Myocarditis	LQTS	Long QT Syndrome
GP	Ganglionated Plexus/Plexi	LSPV	Left Superior Pulmonary Vein
GWAS	Genome Wide Association Studies	LSVC	Left Superior Vena Cava
HA	His Bundle-Atrial	LV	Left Ventricle
HB	His Bundle	LVEF	Left Ventricular Ejection Fraction
HCM	Hypertrophic Cardiomyopathy	LVOT	Left Ventricular Outflow Tract
HIFU	High Intensity Focused Ultrasound	MA	Mitral Annulus
HOP	His Overdrive Pacing	MAP	Monophasic Action Potential
HP	His-Purkinje	MAT	Multifocal Atrial Tachycardia
HPE	Histopathological Examination	MB	Moderator Band
HPF	High Pass Filter	MDCT	Multidetector Computerized Tomography
HPS	His-Purkinje System	MET	Metabolic Equivalent
HRA	High Right Atrium	MI	Myocardial Infarction
IABP	Intra-aortic Balloon Pump	MM	Monomorphic
IART	Intra-atrial Reentrant Tachycardia	MR	Magnetic Resonance
IAS	Interatrial Septum	MRI	Magnetic Resonance Imaging
ICD	Implantable Cardioverter-Defibrillator	MTB	Mycobacterium <i>tuberculosis</i>
ICE	Intracardiac Echocardiography	MW	Microwave energy
IF-VT	Interfascicular Ventricular Tachycardia	NCC	Non-coronary cusp
IHR	Intrinsic Heart Rate	NCX	Sodium-Calcium ion Exchanger
IIR	Intra-isthmus Reentry	NOAC	Novel Oral Anticoagulant
IP	Isolated Potential	NSIVCD	Non-specific Intraventricular Conduction Defect
iPSC-CM	Induced Pluripotent Stem Cell-derived Cardiac Myocytes	NSVT	Non-Sustained Ventricular Tachycardia
IST	Inappropriate sinus tachycardia	ORT	Orthodromic Reciprocating Tachycardia
IVC	Inferior vena cava	OTVT	Outflow Tract Ventricular Tachycardia
IVCD	Intraventricular Conduction Defect	OVM	Oblique Vein of Marshall
JET	Junctional Ectopic Tachycardia	P2R	Phase 2 Reentry
JPB	Junctional Premature Beat	PA	Pulmonary Artery
JSN	Josephson	PAC	Premature Atrial Complex /Contraction
JT	Junctional Tachycardia	PAF	Paroxysmal Atrial Fibrillation
JUP	Plakoglobin	PAM	Papillary Muscles
LA	Left Atrium/Atrial	PCL	Paced/Pacing cycle length
LAA	Left Atrial Appendage	PCR	Polymerase Chain Reaction

PDE	Phosphodiesterase	SF	Safety Factor
PES	Programmed Electrical Stimulation	SHD	Structural Heart Disease
PET	Position Emission Tomography	SIDS	Sudden Infant Death Syndrome
PET-CT	Positron Emission Tomography - Computed Tomography	SMT	Septomarginal Trabeculation
PI	Preexcitation Index	SMVT	Sustained Monomorphic Ventricular Tachycardia
PJRT	Permanent Junctional Reciprocating Tachycardia	SN	Sinus Node
PKP	Plakophilin	SND	Sinus Node Dysfunction
PLN	Phospholamban	SNRT	Sinus Node Recovery Time
PLVT	Pleomorphic Ventricular Tachycardia	SOO	Site of Origin
PMVT	Polymorphic Ventricular Tachycardia	SP	Slow Pathway
PNP	Phrenic Nerve Palsy	SQTS	Short QT Syndrome
POTS	Postural Orthostatic Tachycardia Syndrome	SR	Sarcoplasmic Reticulum
PPI	Postpacing Interval	SR	Sinus Rhythm
PSVT	Paroxysmal Supraventricular Tachycardia	SSFP	Steady State Free Precession
PTSD	Post-traumatic Stress Disorder	SVC	Superior Vena Cava
PV	Pulmonary Vein	SVT	Supraventricular Tachycardia
PVAC	Pulmonary Vein Ablation Catheter	TA	Tricuspid Annulus
PVC	Premature Ventricular Complex/Contraction	TB	Tuberculosis
PVI	Pulmonary Vein Isolation	TCL	Tachycardia Cycle Length
QTc	Corrected QT interval	TEE	Transesophageal Echocardiography
RA	Right Atrium	TFC	Task Force Criteria
RAA	Right Atrial Appendage	TGFβ	Transforming Growth Factor Beta
RAO	Right Anterior Oblique	TMEM43	Transmembrane Protein 43
RB	Right Bundle	TRPM4	Transient Receptor Potential Melastatin Protein 4
RBB	Right Bundle Branch	TTE	Transthoracic Echocardiogram
RBBB	Right Bundle Branch Block	TTN	Titin
RCA	Right Coronary Artery	TWI	T-wave inversion
RCC	Right Coronary Cusp	TZI	Transition Zone Index
RCT	Randomized Clinical Trials	ULR	Upper Loop Reentry
RF	Radiofrequency	VA	Ventriculoatrial
RFA	Radiofrequency Ablation	VA or VArr	Ventricular Arrhythmia
RIPV	Right Inferior Pulmonary Vein	VEGM	Ventricular Electrogram
RMS	Room Mean Squared	VES	Ventricular Extrastimulus
RRP	Relative Refractory Period	VES	Ventricular Extrasystole
RSPV	Right Superior Pulmonary Vein	VF	Ventricular Fibrillation
RV	Right Ventricle	VGLA	Visually Guided Laser Ablation
RVOT	Right Ventricular Outflow Tract	VPC	Ventricular Premature Complex/Contraction
RYR	Ryanodine Receptor	VSD	Ventricular Septal Defect
SA	Sinoatrial	VT	Ventricular Tachycardia
SA	Stimulus to Atrial	WACA	Wide Area Circumferential Ablation
SAECG	Signal Averaged Electrocardiogram	WCT	Wilson Central Terminal
SAN	Sinoatrial Node	WPW	Wolff-Parkinson-White
SCD	Sudden Cardiac Death		
SEMA3A	Semaphorin 3A		
SERCA	Sarcoplasmic Reticulum Calcium Adenosine Triphosphatase		

Contents

<i>Contributors</i>	vii-xi
<i>Forewords</i>	xiii-xv
<i>Preface</i>	xvii
<i>Acknowledgments</i>	xix
<i>List of Abbreviations</i>	xxi-xxiii

SECTION A

INTRODUCTION AND BASICS OF CARDIAC ELECTROPHYSIOLOGY

- 1. Electrophysiology Study: Indications, Hardware and Set-up, Catheter Placement** 3-30
Ajit Thachil
- 2. Electrophysiology Study: Technical Details, Electrograms, Noise and Filtering** 31-41
Katherine Duello, KL Venkatachalam
- 3. Measurements of Basic Intervals, Refractory Periods and Programmed Electrical Stimulation** 43-53
B Hygriv Rao
- 4. Sinus Node Function Evaluation and Abnormalities** 55-63
Watchara Lohawijarn, Negar Salehi, Vini Singh, Aravdeep S Jhand, Ranjan K Thakur
- 5. Atrioventricular Conduction and Block** 65-96
Kartikeya Bhargava

SECTION B

FUNDAMENTALS OF CARDIAC ANATOMY, IMAGING, MAPPING AND ABLATION

- 6. Cardiac Anatomy for Electrophysiologists** 99-118
José Angel Cabrera, Sergio Castrejón, Damián Sánchez-Quintana
- 7. Imaging in Cardiac Electrophysiology** 119-133
Gerard Loughlin, Karen Ordovas, Edward P Gerstenfeld
- 8. Conventional Mapping Techniques: Fundamentals** 135-147
Daniel Pelchovitz, Rishi Arora
- 9. Three-dimensional Mapping of Cardiac Arrhythmias: Techniques, Principles and Application** 149-167
Freddy Del-Carpio Munoz, Samuel J Asirvatham
- 10. Radiofrequency Ablation: Principles and Biophysics** 169-188
Abhishek Deshmukh, Paul Friedman
- 11. Non-Radiofrequency Sources of Ablation** 189-201
Sampath Gunda, Moustapha Atoui, Dhanunjaya Lakkireddy

SECTION C**SUPRAVENTRICULAR TACHYARRHYTHMIAS:
AV NODE-DEPENDENT TACHYCARDIAS**

- | | |
|--|----------------|
| 12. Supraventricular Arrhythmias: Classification | 205-211 |
| <i>Vikas Kalra, Mithilesh K Das</i> | |
| 13. Supraventricular Tachycardias: Approach during Electrophysiology Study | 213-222 |
| <i>Peter Cheung, Taresh Taneja, Shoei K Stephen Huang</i> | |
| 14. Supraventricular Tachycardias: Baseline Features during Sinus Rhythm and Tachycardia | 223-246 |
| <i>John William Schleifer, Komandoor Srivathsan</i> | |
| 15. Supraventricular Tachycardias: Ventricular Pacing Maneuvers | 247-264 |
| <i>Jeffrey Munro, Dan Sorajja, Win-Kuang Shen</i> | |
| 16. Supraventricular Tachycardias: Atrial Pacing Maneuvers | 265-271 |
| <i>Nishant Verma, Bradley P Knight</i> | |
| 17. Atrioventricular Nodal Reentrant Tachycardia: Classification, Electrophysiological Features, and Ablation | 273-279 |
| <i>Demosthenes G Katritsis, Mark E Josephson</i> | |
| 18. Wolff-Parkinson-White Syndrome and Atrioventricular Accessory Pathway-related Arrhythmias: Localization, Mapping and Ablation | 281-302 |
| <i>Rahul Jain, John M Miller</i> | |
| 19. Mahaim Fiber Accessory Pathways and Other Variants of Preexcitation | 303-329 |
| <i>Shomu Bohora</i> | |
| 20. Junctional Tachycardia | 331-340 |
| <i>Leonard A Steinberg, Benzy J Padanilam</i> | |

SECTION D**SUPRAVENTRICULAR TACHYARRHYTHMIAS:
ATRIAL TACHYCARDIA, FLUTTER AND FIBRILLATION**

- | | |
|--|----------------|
| 21. Focal Atrial Tachycardia and its Differentiation from Macroreentrant Atrial Tachycardia | 343-349 |
| <i>Chrishan J Nalliah, Jonathan M Kalman</i> | |
| 22. Atrial Flutter: Classification, Mechanisms and Management | 351-360 |
| <i>Yanfei Yang, Melvin Scheinman</i> | |
| 23. Atrial Fibrillation: Classification and Mechanisms of Initiation and Maintenance | 361-374 |
| <i>Ashok Shah, Nicolas Derval, Thomas Pambrun, Sana Amraoui, Seigo Yamashita, Benjamin Berte, Noora Al-Jefairi, Antonio Frontera, Darren Hooks, Arnaud Denis, Frédéric Sacher, Mélèze Hocini, Pierre Jais, Michel Haissaguerre</i> | |
| 24. Atrial Fibrillation Ablation: Pulmonary Vein Isolation Techniques, Strategies and Principles | 375-387 |
| <i>Rajiv Mahajan, Darragh Twomey, Dennis H Lau, Prashanthan Sanders</i> | |
| 25. Atrial Fibrillation Ablation: Substrate Modification and Other Strategies | 389-399 |
| <i>Tilman Maurer, Erik Wissner, Karl-Heinz Kuck</i> | |

26. Atrioventricular Junction Ablation for Rate Control in Atrial Fibrillation	401-407
<i>Amar Upadhyay, Raja J Selvaraj</i>	
27. Atrial Fibrillation Ablation: Clinical Studies, Efficacy and Complications	409-413
<i>Antonio Sorgente, Riccardo Cappato</i>	
28. Surgical Ablation for Atrial Fibrillation	415-428
<i>Matthew C Henn, Matthew R Schill, Chawannuch Ruaengsri, Richard B Schuessler, Ralph J Damiano Jr</i>	
29. Atrial Arrhythmias in Congenital Heart Disease and Postcardiac Surgery	429-440
<i>Edward P Walsh</i>	

SECTION E**VENTRICULAR TACHYARRHYTHMIAS**

30. Introduction to Ventricular Arrhythmias	443-447
<i>George Shaw, David Callans</i>	
31. Monomorphic Ventricular Tachycardia: Mechanisms and Etiology	449-464
<i>Jianqing Li, Mark E Josephson</i>	
32. Ventricular Tachycardia in Ischemic and Nonischemic Cardiomyopathy: Reentrant Circuits, Mapping Techniques and Ablation Strategies	465-487
<i>Adam Lee, Haris M Haqqani</i>	
33. Ventricular Tachycardia in Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy	489-500
<i>Hugh Calkins</i>	
34. Ventricular Tachycardia in Specific Cardiomyopathies: Sarcoidosis and Tuberculosis	501-510
<i>Daljeet Kaur Saggu, Calambur Narasimhan</i>	
35. Idiopathic Left Ventricular Tachycardia	511-530
<i>Akihiko Nogami</i>	
36. Idiopathic Outflow Tract Ventricular Tachycardia	531-545
<i>Jackson J Liang, Sanjay Dixit</i>	
37. Idiopathic Ventricular Tachycardia from the Mitral Annulus, Papillary Muscles and Other Sites	547-570
<i>Takumi Yamada</i>	
38. Bundle Branch Reentry: Mechanisms, Diagnosis and Management	571-582
<i>Zalmen Blanck, Anwer Dhala, Masood Akhtar</i>	
39. J Wave Syndromes	583-603
<i>Charles Antzelevitch, Bence Patocskaï</i>	
40. Early Repolarization Syndrome and Risk of Sudden Cardiac Death	605-611
<i>Peter A Noseworthy</i>	
41. Idiopathic Ventricular Fibrillation: Mechanisms and Management Strategies	613-626
<i>Arnon Adler, Raphael Rosso, Sami Viskin</i>	
42. Ventricular Arrhythmia Storm: Etiology, Mechanisms and Management	627-634
<i>SP Abhilash, Narayanan Namboodiri</i>	

SECTION F

MISCELLANEOUS

43. Entrainment: Principles and Clinical Applications <i>Ivan Cakulev, Albert L Waldo</i>	637-657
44. Wide QRS Complex Tachycardia: An Electrophysiologic Approach <i>Masood Akhtar</i>	659-703
45. Provocative Drug Testing in the Electrophysiology Lab <i>Venkata A Narla, Nitish Badhwar</i>	705-709
46. Catheter Ablation in Children <i>Philip Wackel, Bryan Cannon</i>	711-716
47. Epicardial Ablation: Techniques and Applications <i>Tahmeed Contractor, Roderick Tung, Noel G Boyle, Kalyanam Shivkumar</i>	717-725
<i>Index</i>	727

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CHAPTER 38

Zalmen Blanck, Anwer Dhala, Masood Akhtar

Bundle Branch Reentry: Mechanisms, Diagnosis and Management

LIST OF ABBREVIATIONS

AV	Atrioventricular	LBBB	Left Bundle Branch Block
BBR	Bundle Branch Reentry	LV	Left Ventricle
EGM	Electrogram	RBB	Right Bundle Branch
EP	Electrophysiology	RBBB	Right Bundle Branch Block
HB	His-Bundle	RV	Right Ventricle
HPS	His-Purkinje System	SMVT	Sustained Monomorphic Ventricular Tachycardia
IF-VT	Interfascicular Ventricular Tachycardia	SR	Sinus Rhythm
LBB	Left Bundle Branch	VT	Ventricular Tachycardia

INTRODUCTION

The most common mechanism of sustained monomorphic ventricular tachycardia (SMVT) is reentry related to scar tissue, usually in patients with ischemic or nonischemic cardiomyopathies. However, reentry in the His-Purkinje system (HPS), also called bundle branch reentry (BBR), accounts for approximately 6% of SMVT in patients with structural heart disease.¹ This is a unique type of VT because the reentry circuit is well defined: the His-bundle (HB), the bundle branches and transseptal myocardial conduction are the components of the reentry circuit.²⁻⁴ Although relatively uncommon, this type of VT may be more frequent than generally suspected for the following reasons:

- Syncope or sudden death are the most common manifestations of this arrhythmia,^{1,5} and 12-lead

electrocardiographic (ECG) documentation usually is not available.

- Induction of this mechanism of VT in the electrophysiology (EP) laboratory may be difficult or not reproducible, and a variety of electric stimulation techniques or pharmacologic maneuvers that might not be used routinely in EP laboratories may be required.
- An HB recording during VT is necessary for the diagnosis of this arrhythmia,⁵⁻⁸ and may not be obtained during EP studies performed solely for VT.
- In the United States and in many parts of the world, defibrillator implantation is usually performed without EP evaluation, even in patients implanted for secondary prevention of life-threatening ventricular arrhythmias.

It is important to recognize BBR as the mechanism of VT because catheter ablation of the right bundle branch

(RBB), a procedure that can be easily performed in most patients and has a high success rate,^{1,5,9-12} is curative of this type of VT.

MECHANISMS OF BUNDLE BRANCH REENTRY

Isolated BBR beats can be found in up to 50% of patients with normal intraventricular conduction undergoing EP studies; it is a finding without any prognostic significance.²⁻⁴ The QRS morphology in these beats, or when sustained tachycardia is induced, will depend upon which bundle branch is used for antegrade propagation of the electric impulse: the QRS will exhibit a left (L), or right bundle branch block (RBBB) morphology, if the impulse propagates down the RBB or the LBB, respectively. The induction of isolated BBR beats or sustained BBR tachycardia share a common mechanism, as follows (**Figure 38.1**).

During right ventricular (RV) programmed stimulation using a constant basic drive, a premature beat (S2) with a long coupling period is introduced and retrograde conduction to the HB occurs via the RBB, resulting in short V2-H2 intervals (**Figure 38.1A**). As the S2 coupling periods are shortened, progressive delay in the retrograde RBB conduction is encountered (longer V2-H2), while propagation of the impulse proceeds transseptally into the LBB (which has shorter refractoriness than the RBB). Additional shortening of the coupling periods reach the effective refractory of the RBB, resulting in retrograde conduction block (**Figures 38.1B and C**). Propagation of the stimulus continues transseptally, and via the LBB to the HB. A retrograde HB potential, inscribed after the local ventricular electrogram (EGM), becomes apparent. Further conduction delay in the LBB allows recovery of the initial site of the block in the RBB, allowing the impulse to propagate antegradely and activate the RV. This results in a wide QRS complex with a left bundle branch block (LBBB) pattern, the so-called V3 phenomenon, BBR beat, or a macro-reentrant beat. It should be noted that there is an inverse relationship between the retrograde conduction delay in the LBB (V2-H2), and the degree of recovery of the antegrade conduction in the RBB (H2-V3). Longer conduction times in the LBB (longer V2-H2), facilitate the antegrade recovery of the RBB, resulting in shorter H2-V3 intervals. On the other hand, insufficient delay in V2-H2 (i.e., longer coupling periods) may result in a longer H2-V3.²⁻⁴

It has been shown that reentry in the HPS is more likely to occur when premature beats are introduced during basic drives that incorporate short-long sequences, in contrast to constant basic drives. This is due to the cycle length dependency of the HPS refractoriness.¹³⁻¹⁵ It has been suggested that an abrupt change in cycle length (short-to-long) may result in conduction block at a more distal site in the muscle-Purkinje-RBB axis, which will allow sufficient

recovery of excitability in the RBB-Purkinje-muscle to allow antegrade conduction and reentry. This also will result in a shorter H2-V3 interval.

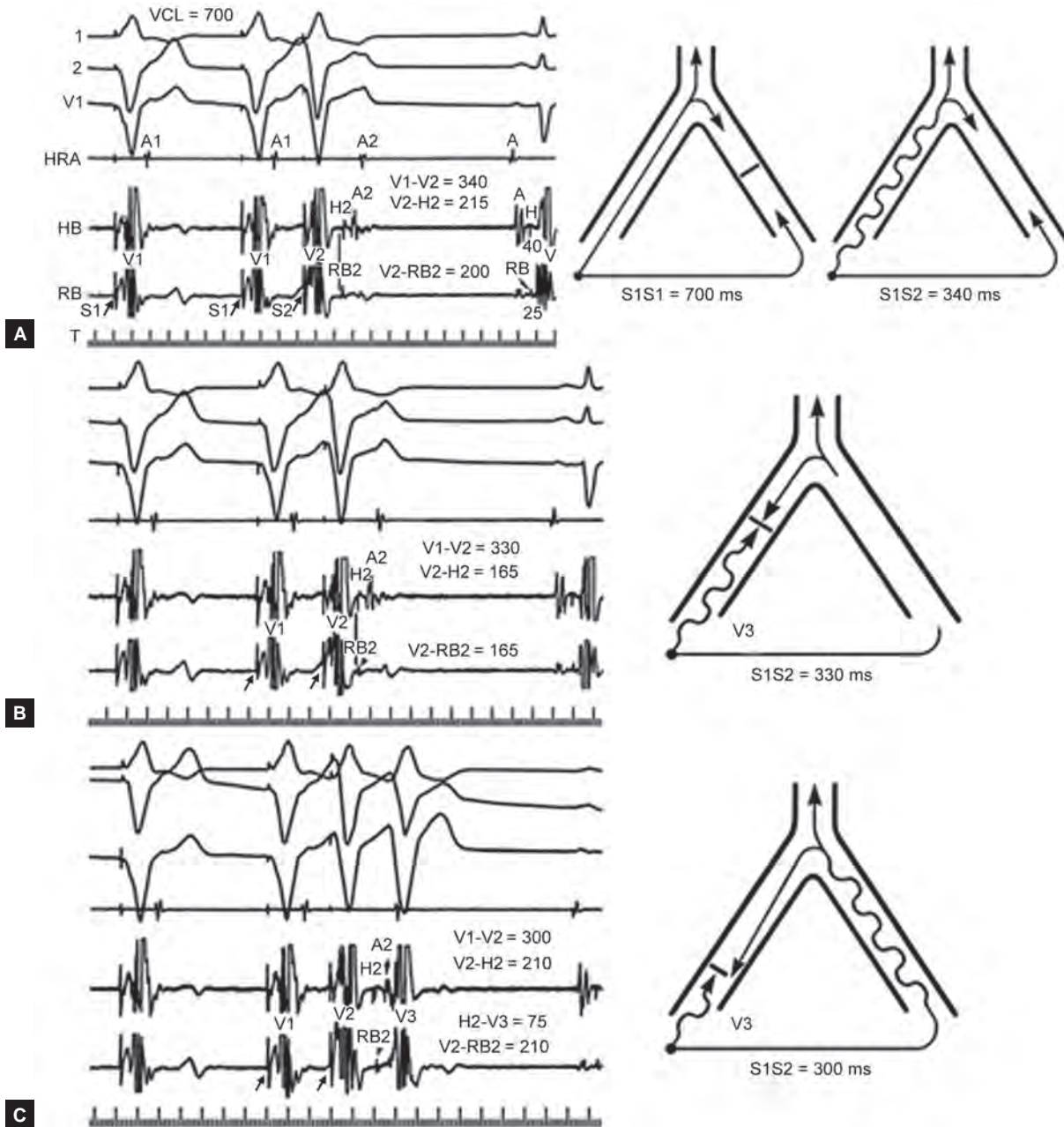
Although the most common type of BBR has an LBBB pattern, BBR with an RBBB pattern also may occur during RV stimulation. During this type of reentry, there is a retrograde LBBB and the impulse retrogradely propagates to the HB via the RBB. This can only occur when the LBB refractoriness is longer than that of the RBB or when retrograde RBB conduction resumes after a bilateral HPS block (gap phenomenon). This type of reentry also may be seen during left ventricular (LV) stimulation, as retrograde LBBB may be easier to accomplish given the proximity of the LBB to the stimulation site.

In patients with normal intraventricular conduction, BBR is a limited phenomenon, and if short-to-long pacing sequences are used, up to 3 BBR beats may be seen.^{3,13-15} In most cases, the reentry terminates in the retrograde limb of the circuit, in the muscle-Purkinje-LBB axis.¹⁶ Rarely, the reentry will terminate in the antegrade limb. The maintenance of this phenomenon is critically dependent upon the relationship between the conduction velocity and the recovery of excitability in front of the reentrant impulse. The presence of conduction abnormalities (i.e., intraventricular conduction delay) facilitates the development of clinically relevant sustained reentry.

Another, much less common, type of HPS reentry with a narrow QRS complex has been described in the presence of normal intraventricular conduction during RV stimulation.¹⁷ This occurs when there is retrograde conduction via the LBB, followed by antegrade propagation via the RBB and one of the LBB fascicles, resulting in a narrow QRS with variable axis, depending upon which fascicle is used for antegrade conduction.

CLINICAL CHARACTERISTICS OF PATIENTS WITH BBR-VT

Sustained BBR-VT usually occurs in patients with significant structural heart disease: LV dysfunction with low ejection fraction and congestive heart failure are typical findings. Although nonischemic cardiomyopathy is the underlying substrate in about 45% of these patients,^{1,5-7,9} this type of VT can also be seen in ischemic and valvular cardiomyopathies,¹⁸ and also has been reported in patients with Ebstein's anomaly,¹⁹ hypertrophic cardiomyopathy,¹¹ and any other type of structural heart disease associated with abnormal intraventricular conduction.¹⁶ Myotonic dystrophy and other types of dystrophies also can be a substrate for this VT given the involvement of the HPS in these conditions.²⁰ Rarely, patients with isolated HPS disease, without other evidence of cardiac disease, have been reported to develop sustained BBR.^{16,21} In some patients, valvular replacement surgery (aortic or mitral) predisposes them to develop



Figures 38.1A to C: In Panels A, B, and C, the tracings displayed are, from top to bottom, surface ECG leads 1, 2 and V1, and intracardiac recordings from the high right atrium (HRA), His-bundle (HB), right bundle branch (RB), and time lines (T). The three panels show the effect of premature ventricular beats introduced with progressively shorter coupling periods to a constant basic drive in the retrograde conduction in the His-Purkinje system. During the constant ventricular drive (700 ms), retrograde conduction is by way of the right bundle branch; this impulse collides with the transeptally conducted impulse in the left bundle branch (see diagram). A premature ventricular beat (coupling period 340 ms) results in slowing of the retrograde right bundle branch conduction, with subsequent emergence of the right bundle branch and HB potentials after the local ventricular electrogram. Note that the right bundle branch potential precedes the HB potential ($V2 - RB2 = 200$ ms versus $V2 - HB2 = 215$ ms) as expected with retrograde conduction proceeding via the right bundle branch. In Panel B, the introduction of a premature beat with a shorter coupling period ($S2$ 330 ms) results in (proximal) retrograde block in the right bundle branch, which allows the transeptally conducted impulse to reach the HB via the left bundle branch. Note the change in the sequence of HB activation compared to Panel A ($V2 - RB2 = 165$ ms versus $V2 - H2 = 165$ ms). The HB and the right bundle branch are simultaneously activated, as expected during retrograde conduction via the LBB. In Panel C, the coupling period of $S2$ is further shortened to 300 ms, which results in retrograde block in the distal right bundle branch. This shift in the site of right bundle branch block, and the slower transeptal and left bundle branch retrograde conduction, allow recovery of the site of block and activation of the right ventricle via the RBB (see diagram), resulting in a bundle branch reentrant beat with a left bundle branch block morphology, also called V3 phenomenon, or macro-reentrant beat

sustained BBR in the immediate postoperative period.¹⁸ This group of patients who developed BBR postoperatively had better preserved cardiac function and left ventricular ejection fraction than the typical patient with cardiomyopathy and BBR (LVEF 44%). Of course, the most important determinant of long-term survival in these patients is the degree of cardiac dysfunction.^{1,5,16,18,21}

Clinical Presentation

Sustained BBR is usually a fast tachycardia, and given the association with significant cardiac disease, it results in significant hemodynamic compromise: syncope or sudden death are the clinical presentation in up to 70% of these patients.^{1,5} Twelve-lead ECG documentation of the VT is rarely available, so the relative incidence of spontaneous VT with LBBB or RBBB morphology is unknown.

ECG Findings

The most common abnormalities include mild PR interval prolongation in sinus rhythm (SR) (average 256 ms)^{1,5} About 25% of patients have atrial fibrillation as the intrinsic rhythm. Most patients have an intraventricular conduction delay with an LBBB pattern. Rarely, an RBBB pattern is seen, a finding that does not exclude BBR as the mechanism of the VT, because the RBBB pattern may reflect antegrade conduction delay, rather than complete antegrade block, in the RBB. In the same context, a complete LBBB pattern may also be a manifestation of antegrade conduction delay, rather than complete conduction block. Even in the presence of a complete antegrade conduction block, the bundle branch may still be able to exhibit retrograde conduction, a necessary requirement for BBR to occur.¹⁶

In our experience, atrioventricular (AV) dissociation was present in nearly 100% of patients with sustained BBR.^{1,5} This may be due to the fast cycle lengths of BBR and the presence of drugs that may depress AV conduction (e.g., beta-blockers, digoxin).

ELECTROPHYSIOLOGIC CHARACTERISTICS OF PATIENTS WITH BBR-VT

The presence of conduction disease in the HPS, manifested as prolongation of the His-ventricle (HV) interval, is a cardinal finding in this patient population, regardless of the type of underlying structural substrate.^{1,5,6-12} In our experience, the HV interval ranged from 60 ms to 110 ms (average 80 ms).⁵

BBR is most commonly induced by RV stimulation. This can be accomplished by the introduction of premature ventricular stimuli to a constant basic drive, or more commonly, by the introduction of premature stimuli to a drive incorporating a pause before introducing the premature beat(s), so-called short-long-short.¹³⁻¹⁵ We routinely use a 600 ms

pause during a 400 ms drive prior to introducing premature beats. As the electric properties of the HPS may vary between patients, the use of protocols incorporating different short-long sequences may be necessary (i.e. 350–650, 400–700, etc.).¹³⁻¹⁵ Induction of BBR with an RBBB may also require LV stimulation.

In some instances, the use of class 1A anti-arrhythmic drugs (e.g., procainamide) may facilitate induction of sustained BBR when the VT is not induced in the baseline state. Procainamide prolongs the antegrade and retrograde conduction times of the HPS, and by prolonging the HV and VH intervals, allows the penetration by the reentrant impulse into a better recovered RBB or LBB, respectively.²² It should be noted that induction of BBR should be attempted during the slow administration of these drugs, as they may also abolish this type of reentry. Sometimes, isoproterenol may also be useful to induce this type of VT. However, the use of these drugs has not been systematically studied in patients with sustained BBR.

In contrast to other types of VT, BBR-VT can almost always be terminated by overdrive ventricular stimulation, regardless of the VT cycle length (unless, of course, ventricular fibrillation is induced). The rationale for this is the relative large size of the reentrant circuit, the presence of an “excitable conduction gap,” and the proximity of the RV stimulation site to the reentrant circuit. All these factors facilitate the penetration of the circuit by the propagated stimulated impulses.

Diagnostic Criteria for BBR-VT

The EP criteria diagnostic of BBR are shown in **Table 38.1**. The diagnosis of BBR-VT requires intracardiac recordings during the induced VT (i.e., HB and/or bundle branch potentials). In some cases, it may be difficult to obtain an HB recording during the VT, in which case, an RBB potential may be more stable and easier to record, and may facilitate the diagnosis.⁸

Table 38.1: Diagnostic criteria for BBR-VT

1. The VT exhibits QRS morphology that is typical of an LBBB or RBBB, consistent with ventricular depolarization via the His-Purkinje system.
2. The onset of ventricular activation is preceded by a His-bundle potential and bundle branch potentials, with an appropriate sequence of activation to the corresponding QRS morphology, and with stable HV, RB-V, or LB-V intervals.
3. Spontaneous variations in V-V intervals are preceded by similar variations in H-H intervals.
4. Induction of tachycardia is consistently dependent upon achieving a critical delay in the His-Purkinje system.
5. The VT cannot be induced after successful catheter ablation of the RBB.

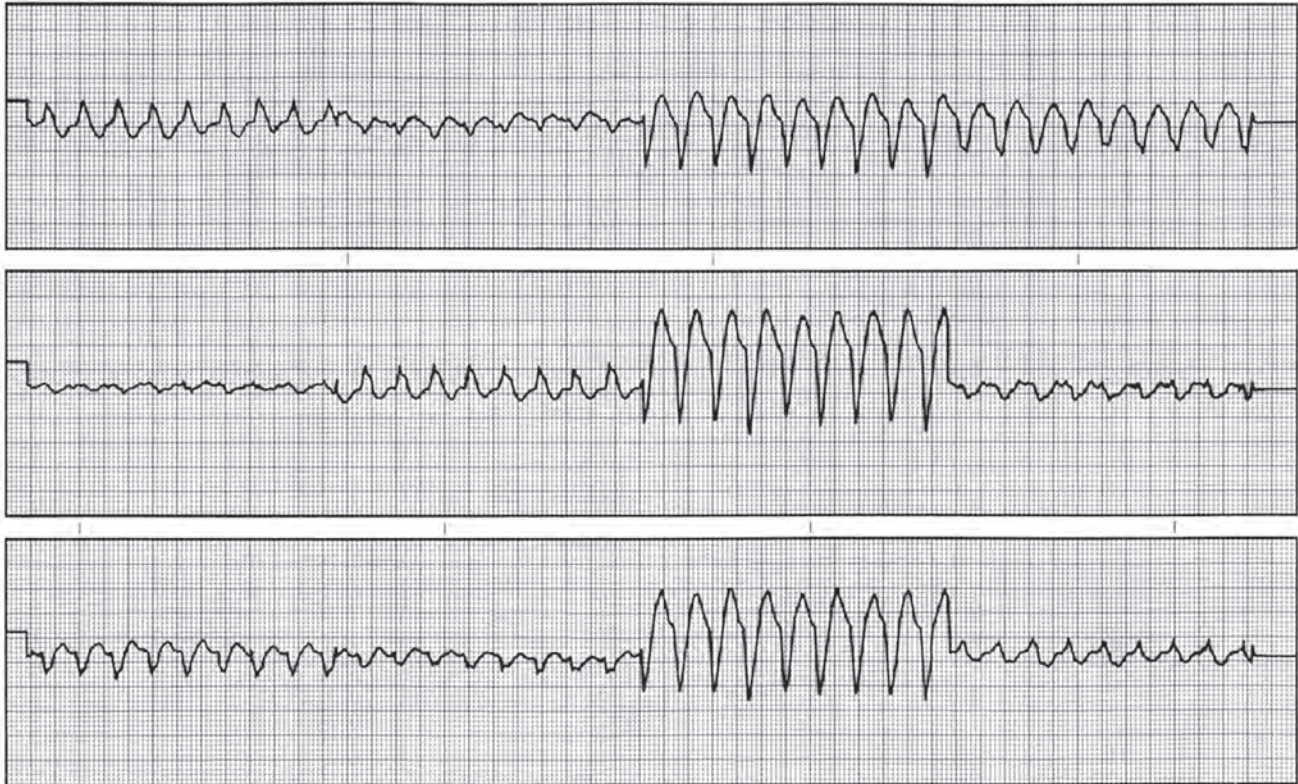
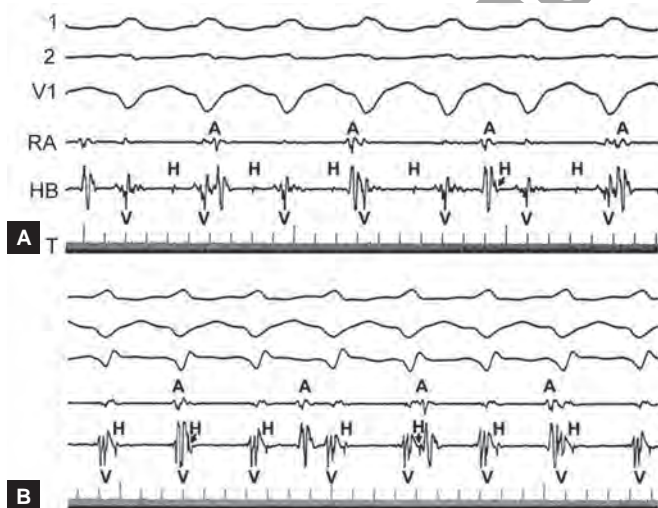


Figure 38.2: Twelve-lead surface electrocardiogram of spontaneous bundle branch reentrant tachycardia with left bundle branch QRS pattern and left-axis deviation at a rate of 215 bpm (not labeled). Because ventricular activation occurs by way of the right bundle branch, the QRS configuration is suggestive of intraventricular aberrant conduction. (Used with permission from Elsevier from Zipes DP, Jalife J. Cardiac Electrophysiology: From Cell to Bedside, 2nd edn. (2005) Saunders, Philadelphia, Penn. p. 881).



Figures 38.3A and B: Bundle branch reentry with left (A) and right (B) bundle branch block morphology. Tracings, from top to bottom in each panel, include surface ECG leads 1, 2 and V1, and intracardiac recordings from the right atrium (RA), His-bundle (HB), and time lines (T). In Panel A, bundle branch reentry tachycardia with a left bundle branch block morphology is displayed. Note the relatively slow cycle length, unusual in this type of tachycardia. The HV interval of 90 ms was identical to the one in sinus rhythm. In contrast, during tachycardia with a right bundle branch block, the HV interval is much longer, 250 ms. Antegrade activation in each tachycardia is dependent upon the RBB and the LBB, respectively, resulting in significantly different HV intervals

During BBR-VT with an LBBB pattern (**Figure 38.2**), the most common type of induced BBR-VT, the HV interval is similar to, or slightly longer than, the HV interval in SR (**Figure 38.3A**).^{1,5,6-12} Rarely, if a very proximal HB recording is obtained, a slightly shorter HV interval may be obtained

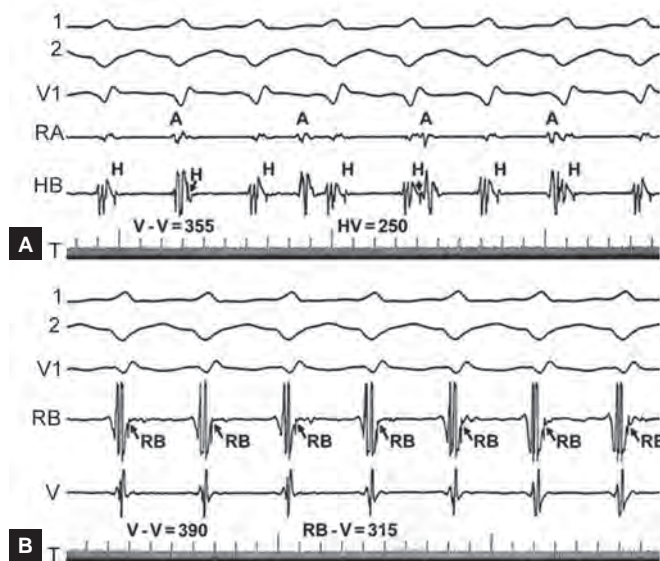
during the VT as the HB and the RBB may be simultaneously activated via the LBB.

In contrast, the induction of BBR with an RBBB pattern may result in an HV interval that is significantly different than in sinus rhythm (**Figure 38.3B**). In patients with BBR,

the HV interval during SR is generally determined by the conduction properties of the RBB. However, during VT with an RBBB pattern, the HV interval is determined by the conduction properties of the LBB. Different antegrade conduction properties of the RBB and the LBB may account for different HV intervals during intrinsic rhythm versus tachycardia.

Recording the HB potential and the bundle branch potentials can document the sequence of activation of the HPS during the VT, an important diagnostic criteria for BBR (Table 38.1, Figures 38.4, 38.5 and 38.6A). During VT with an LBBB pattern, activation of the LBB is followed by activation of the HB, which in turn is followed by activation of the RBB. The opposite sequence of activation occurs during BBR-VT with an RBBB pattern.

As ventricular activation is dependent upon the propagation of the impulse in the HPS, irregularities in the H-H cycle lengths during BBR (typically seen at the onset of the tachycardia), will precede similar irregularities in the corresponding V-V cycle lengths (Figure 38.6B). This is an important criterion to distinguish VT due to BBR from scar-related VT with incidental (retrograde) activation of the HPS.



Figures 38.4A and B: Bundle branch reentry (BBR) with right bundle branch block (RBBB) morphology. Panel A shows, from top to bottom, surface ECG leads 1, 2 and V1, and intracardiac recordings from the right atrium (RA), His-bundle, and time lines (T). Panel B shows the same surface ECG leads, and intracardiac recordings from the right bundle branch (RB), right ventricle (RV), and time lines (T). During BBR with RBBB morphology (Panel A), the HV is determined by the conduction properties of the left bundle branch, in this case, 250 ms. In contrast, during sinus rhythm, the HV interval, determined primarily by the right bundle branch, was 90 ms (not shown). In Panel B, the right bundle branch potential is shown. Note the appropriate sequence of activation: the RBB potential is recorded before the His-bundle potential, as expected in this type of BBR reentry

Merino et al.²³ described another diagnostic criterion for BBR. Given the close proximity between the BBR reentry circuit (i.e., distal RBB) and the RV apex, the post-pace interval was equal or <30 ms when RV stimulation was performed during BBR with an LBBB (compared to >100 ms for myocardial VT) (Figure 38.7). This may be particularly useful when an HB or RBB potential cannot be recorded.

BBR-VT with LBBB Pattern

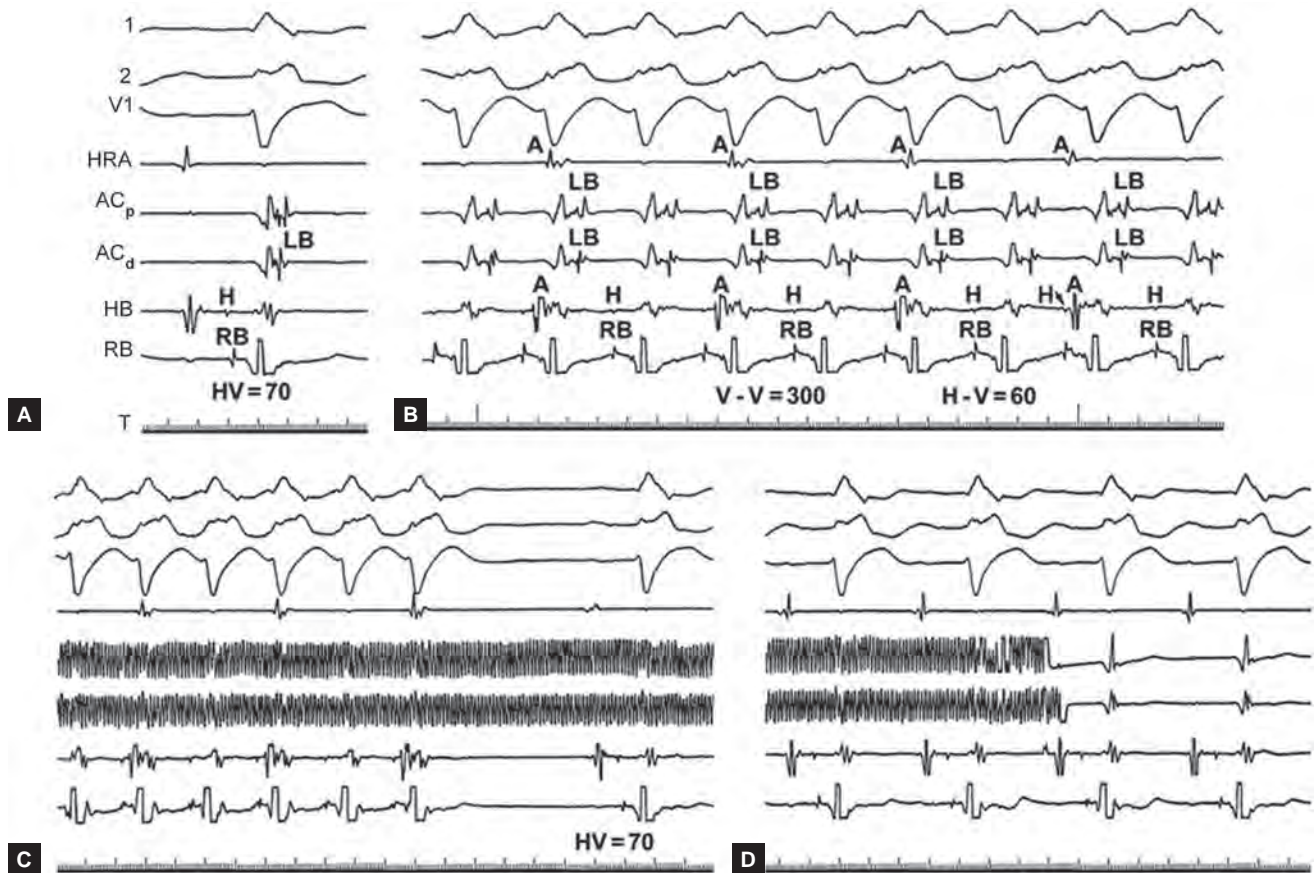
As previously mentioned, this is, by far, the most common type of HPS-related VT,^{1,5,6-12} perhaps because programmed stimulation is routinely performed from the RV. In our experience,^{1,5} induction of this VT required LV stimulation in 2 of 59 patients. The QRS morphology is suggestive of aberrant conduction (Figure 38.2) because myocardial activation is by way of the HPS, in this case the RBB. In the absence of anti-arrhythmic drugs, the cycle length of this VT is fast, ranging from 200 ms to 300 ms. The QRS axis is usually normal or leftward. Rightward axis is rare, unless the QRS in SR also is rightward. The HV interval ranges from 55 ms to 160 ms.

BBR-VT with RBBB Pattern

In this type of VT, activation of the HB is by the RBB, followed by antegrade conduction via the LBB (Figures 38.4A and B). We induced this VT in 6 of 59 patients. In 2 of the 6 patients, it was the only type of VT inducible. This type of VT, in contrast to the one with LBBB pattern, more often required LV or atrial stimulation. A functional proximal RBBB may occur during atrial pacing (or atrial fibrillation);²⁴ slow antegrade propagation over the LBB may allow recovery of the RBB, facilitating BBR. This type of VT may be less common than BBR with an LBBB pattern because LV stimulation is not routinely performed, but also due to the shorter retrograde refractoriness of the LBB (compared to the RBB), in which case, retrograde block may be more difficult to accomplish during RV pacing. The QRS axis in this type of VT may be normal, leftward, or rightward, depending upon which fascicle is used for antegrade propagation. In our experience, the cycle length of this tachycardia has ranged from 220 ms to 360 ms, and the HV interval between 65 ms and 250 ms.^{1,5,18} Although rare, this type of VT was more commonly seen in the immediate postoperative period after valvular replacement surgery, compared to patients with nonischemic cardiomyopathy.¹⁸

Interfascicular (IF)-VT

In this type of VT,²⁵⁻²⁷ the reentry circuit involves the distal LBB, the left-sided fascicles, and myocardial conduction (Figure 38.8). The RBB is not part of the reentry circuit and is activated incidentally; therefore, catheter ablation of the RBB will not eliminate this type of VT. This mechanism of VT needs to be excluded from BBR with an RBBB pattern because in both cases the QRS morphology is RBBB pattern.



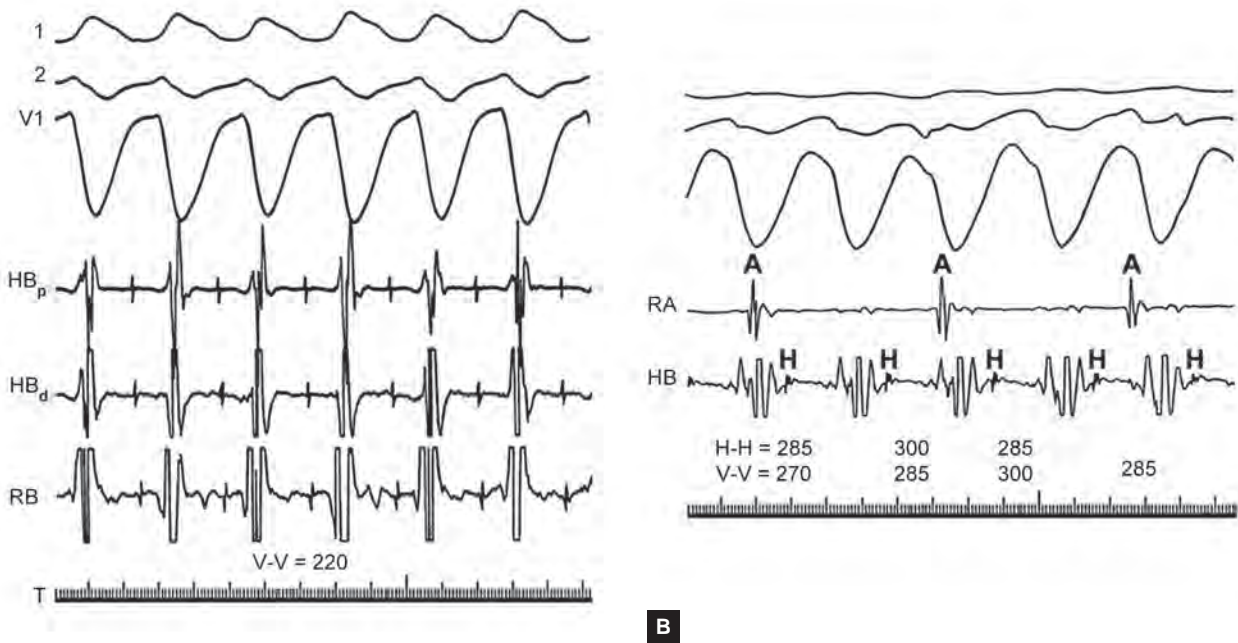
Figures 38.5A to D: Catheter ablation of the left bundle branch for bundle branch reentrant tachycardia. The four panels in this figure display, from top to bottom, surface ECG leads 1, 2 and V1 and intracardiac recordings from the high right atrium (HRA), proximal and distal ablation catheter (AC_p and AC_d), His-bundle (HB), right bundle branch (RB), and time lines (T). Panel A shows sinus rhythm with His and right bundle branch potentials. The ablation catheter is positioned in the left side of the interventricular septum and is recording a left bundle branch potential (LB) after the local ventricular electrogram. This is a retrograde (transeptally conducted impulse) potential given the complete antegrade left bundle branch block. During bundle branch reentrant tachycardia (Panel B), the same sequence of activation is displayed as in sinus rhythm, with a slightly shorter HV interval. Panel C displays delivery of radiofrequency current to the left bundle branch during bundle branch reentry, which results in termination of the tachycardia. The first escape beat has the same HV interval as before the ablation and there is no change in the QRS morphology. Note in Panel D that the LB potential is no longer recorded. (From Blanck Z, Deshpande S, Jazayeri MR, Akhtar M. Catheter ablation of the left bundle branch for the treatment of sustained bundle branch reentrant ventricular tachycardia. *J Cardiovasc Electrophysiol* 1995; 6:40-3. Used with permission from John Wiley and Sons)

The sequence of activation of the HPS, being different in these two tachycardias, may be helpful in differentiating them. During BBR with RBBB (i.e., retrograde conduction via the RBB), the RBB is activated before the HB is activated. In contrast, during IF-VT, the RBB is expected to be activated after the HB activation. Patients with IF-VT usually have concomitant BBR.^{25,26} We recently noted that an RBBB may be a prerequisite for IF reentry (spontaneous or inducible).²⁵ The RBBB may be pre-existing or occur after catheter ablation for BBR-VT. The HV interval during IF-VT is usually shorter than in SR, as the "turnaround" between the fascicles is distal to the HB. Depending on the fascicle used for antegrade conduction, the QRS during IF-VT will be rightward or

leftward. Ablation of the LBB, or one of its fascicles, is necessary to eliminate this type of VT and has been performed successfully.²⁵⁻²⁷

DIFFERENTIAL DIAGNOSIS OF BBR-VT

BBR-VT should be suspected in the presence of a wide QRS complex tachycardia with AV dissociation, where HB potentials precede ventricular activation. The diagnosis of BBR-VT requires careful analysis of the sequence of HPS activation and the relationship between changes in H-H and V-V cycle lengths. Otherwise, this mechanism may go unrecognized and be attributed to the common variety of



Figures 38.6A and B: Diagnosis of bundle branch reentry. Panel A, from top to bottom, shows surface ECG leads 1, 2 and V1, and intracardiac recordings from proximal and distal His-bundle (HB_p and HB_d), right bundle branch (RB), and time lines (T). Intracardiac recordings during bundle branch reentrant tachycardia show the His and bundle branch potentials to precede the onset of the surface ECG, the appropriate sequence of His-Purkinje system activation during tachycardia with a left bundle branch pattern (i.e., from proximal to distal), and a very short cycle length, typical of this type of reentry. Panel B, from top to bottom, shows surface ECG leads 1, 2 and V1, and intracardiac recordings from the right atrium (RA), His-bundle (HB), and time lines (T). This figure shows an important criteria for bundle branch reentrant tachycardia: during irregular cycle lengths, H-H changes will precede and dictate the corresponding V-V changes

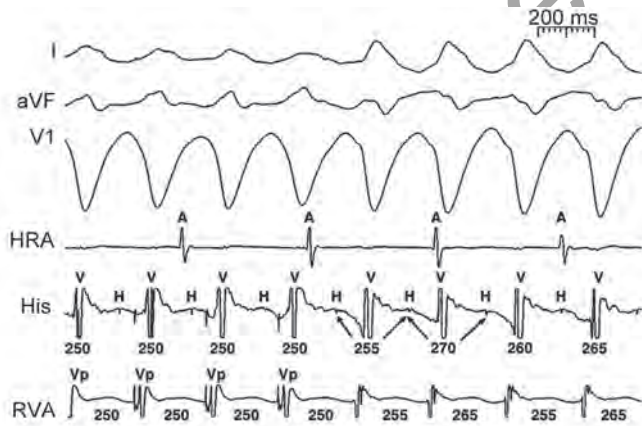


Figure 38.7: Post-pace interval during bundle branch reentry. Tracings, from top to bottom, include surface ECG leads 1, 2 and V1, and intracardiac recordings from the right atrium (RA), His-bundle (His), and right ventricular apex (RVA). This figure shows a post-pace interval (PPI) of 250 ms after a train of ventricular pacing from the RV apex (first four beats of the figure). A similar PPI from this pacing site as the cycle length of tachycardia is consistent with bundle branch reentry

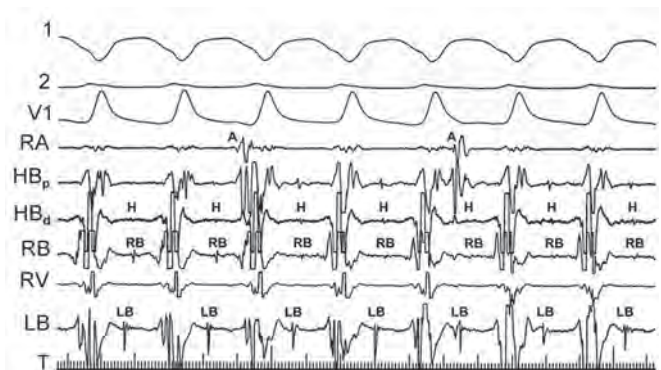


Figure 38.8: Intersfascicular reentrant tachycardia. Displayed from top to bottom are surface ECG leads 1, 2 and V1 and intracardiac recordings from the right atrium (RA), proximal and distal His-bundle (HB_p and HB_d), right ventricle (RV), left bundle branch (LB), and time lines (T). Intracardiac recordings during intersfascicular tachycardia show the appropriate sequence of His-Purkinje system activation: the left bundle branch is activated first, followed by simultaneous activation of the HB and the RB. The HV interval (not labeled) is 25 ms shorter than in sinus rhythm, a finding consistent with this mechanism of tachycardia. The QRS morphology is right bundle branch block (RBBB). In bundle branch reentry with the same QRS pattern (e.g., RBBB), the opposite sequence of activation would be expected (e.g., RB, followed by HB, followed by LB)

scar-related VT. Perhaps, the most important factor in the diagnosis of BBR-VT is to suspect it in the appropriate clinical setting.

Myocardial Scar-related VT

This type of VT, with retrograde activation of the HPS, is the most important consideration and should always be differentiated from BBR-VT. In most scar-related VTs, the HB activation is “obscured” by the local ventricular EGM, and it is not usually seen. However, in some VTs, the HB potential may be recorded before the local ventricular EGM but after the onset of the QRS in the 12-lead ECG, which rules out BBR. In other VTs, an HB or BB potential may appear to precede the onset of the surface QRS, a finding similar to BBR-VT (Figure 38.9). In these cases, and in contrast to BBR, changes in V-V intervals will precede subsequent changes in H-H intervals. In addition, given the same QRS morphology (i.e., RBBB), analysis of the sequence of HPS activation may be helpful as it may differ between myocardial VT, where the HB may be activated retrogradely by the LBB, and BBR-VT, where the HB also is activated retrogradely, but by the RBB. Finally, if myocardial VT is suspected, RV pacing during SR at the same cycle length as the VT may be helpful to demonstrate retrograde HPS activation (Figures 38.10A and B), a finding that would support a myocardial VT.

Supraventricular Tachycardia with Aberrant Conduction

Patients with BBR almost never exhibit 1:1 AV conduction during tachycardia. In addition, the sequence of activation

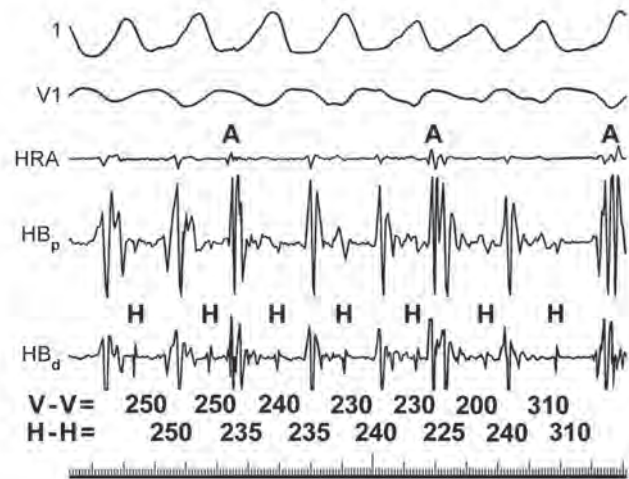
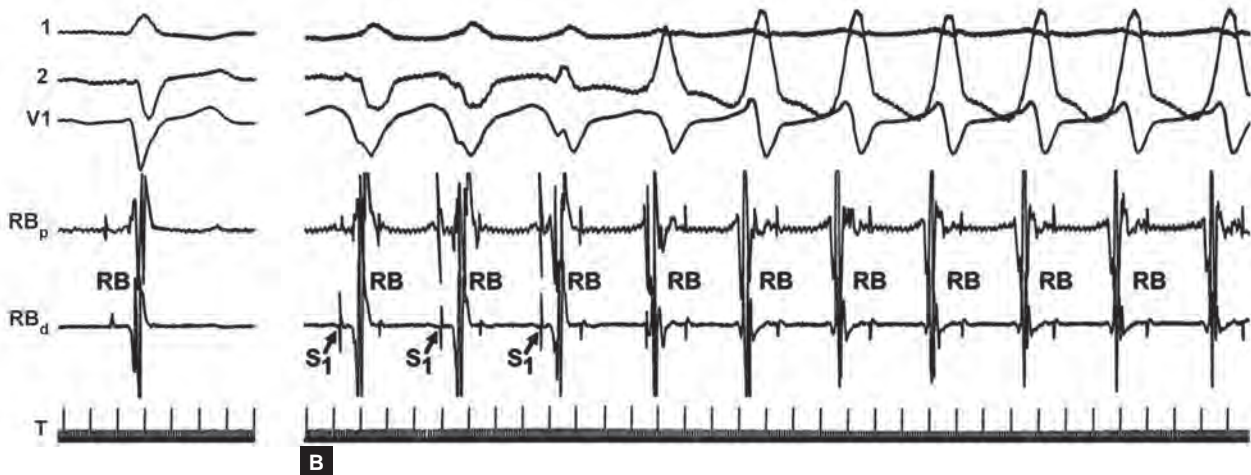


Figure 38.9: Incidental activation of the His-bundle during myocardial ventricular tachycardia initiated after catheter ablation of the right bundle branch. Tracings, from top to bottom, are surface ECG leads I and V1; high right atrium (HRA) and proximal and distal His-bundle recordings (HB_p and HB_d); and time lines (T). All intervals are in milliseconds. During induced sustained ventricular tachycardia with a left bundle branch block QRS configuration, each ventricular electrogram is preceded by a His-bundle potential. However, retrograde activation of the His-Purkinje system is coincidental, and changes in V-V intervals precede or are unrelated to changes in H-H intervals, as expected during myocardial ventricular tachycardia. In this case, activation of the His-bundle is retrograde through the left bundle branch, the conduction of which was severely impaired. (From Blanck Z, et al. Bundle Branch Reentrant Ventricular Tachycardia: Cumulative Experience in 48 Patients. *J Cardiovasc Electrophysiol* 1993;4:253-63. Used with permission from John Wiley and Sons)



Figures 38.10A and B: Tracings, from top to bottom, show surface ECG leads 1, 2 and V1, and intracardiac recordings from proximal and distal right bundle branch (RB_p and RB_d) and time lines (T). Panel A shows the RB potentials in sinus rhythm. Panel B displays the end of a ventricular pacing drive (first 4 beats) followed by ongoing ventricular tachycardia (VT). Note that the RB is captured during ventricular pacing, with a similar sequence as during VT, a finding consistent with myocardial, scar-related VT

of the HPS is different: in supraventricular tachycardia, the HPS is activated antegradely, with a similar sequence as in sinus rhythm. In contrast, during BBR, the HPS sequence of activation is retrograde usually via the LBB.

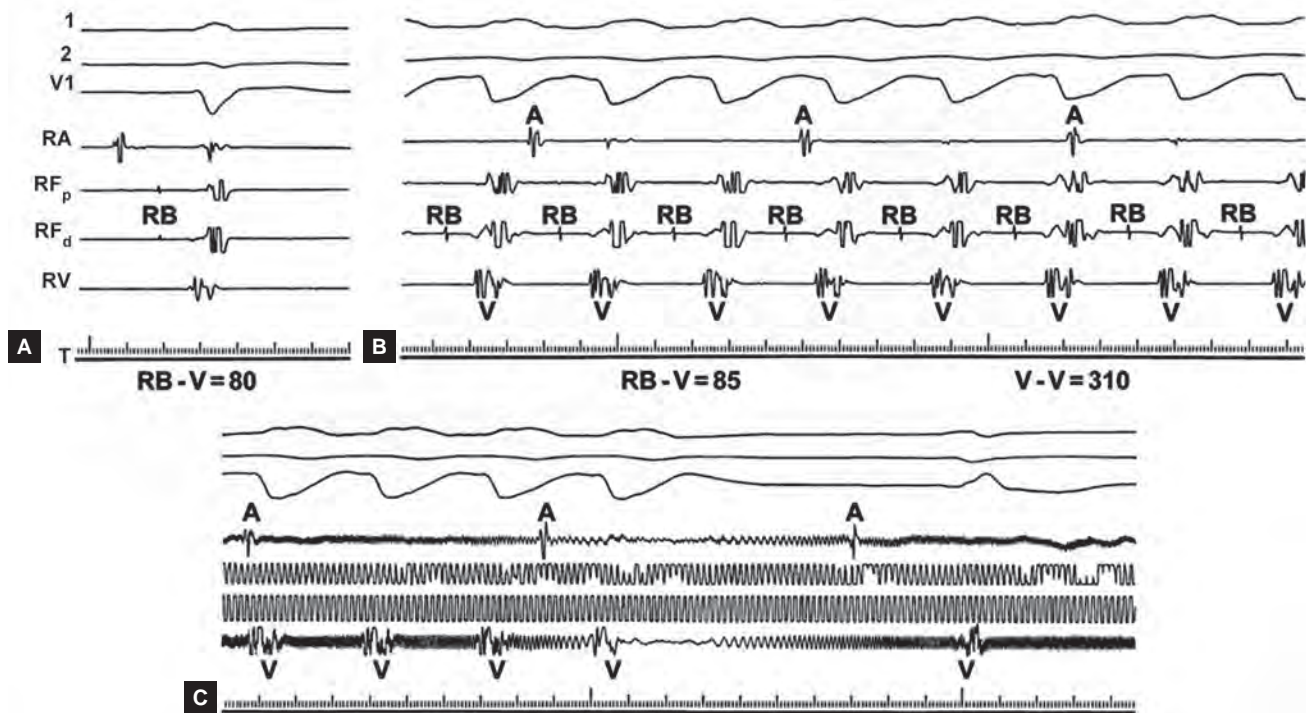
Atriofascicular Reentry

In this tachycardia, ventricular activation also is by way of the RBB,²⁸ and the HB is activated retrogradely, as in BBR. However, the sequence of HPS activation is different in both tachycardias: in BBR with an LBBB pattern, the HB is activated before the RBB, and the opposite sequence is seen in atriofascicular reentry. Also, the atrium is part of the atriofascicular reentry circuit, and most patients with atriofascicular reentry do not have structural heart disease. Atrial pacing in patients with atriofascicular reentry may show pre-excitation.

BBR-VT should always be suspected in patients with nonischemic cardiomyopathy presenting with syncope or sudden death. It also should be suspected in patients with inducible SMVT and conduction abnormalities, or when the VT has an LBBB pattern.

TREATMENT OF BBR-VT

Radiofrequency catheter ablation of the RBB is the treatment of choice for BBR-VT⁸ (Figures 38.11A to C). This procedure will eliminate both types of BBR (LBBB and RBBB) by creating complete conduction block in the RBB.^{1,5,9-12} In this ablation, a catheter is placed in the septum until an RBB potential is recorded. The nature of this potential is confirmed by the absence of an atrial EGM and an H-RB interval of at least 20 ms.^{9,10} Inadvertent ablation of the HB will result in complete AV block and persistent inducibility



Figures 38.11A to C: Termination of sustained bundle branch reentrant tachycardia during catheter ablation of the right bundle branch using radiofrequency current. Displayed from top to bottom are ECG leads 1, 2, and V1; intracardiac recordings from the right atrium (RA), proximal and distal ablating catheter (RF_p, RF_a), and right ventricle electrogram (RV); and time lines (T). All intervals are in milliseconds. In Panel A, activation of the right bundle branch is recorded in the distal bipole of the ablating catheter during sinus rhythm. Panel B shows bundle branch reentrant ventricular tachycardia with a left bundle branch block pattern and a cycle length of 310 ms. Activation of the right bundle branch is recorded in the ablating catheter. In Panel C, ablation of the right bundle branch and termination of the tachycardia occur within 6 seconds of energy application. Note the expected complete right bundle branch block QRS morphology in the first sinus beat after terminating the tachycardia. (From Blanck Z, et al. Bundle Branch Reentrant Ventricular Tachycardia: Cumulative Experience in 48 Patients. *J Cardiovasc Electrophysiol* 1993;4:253-63. Used with permission from John Wiley and Sons)

of BBR. Given the anatomic features of the RBB (relatively thin and superficially located in the sub-endocardium) this procedure is easily performed and successful in the majority of patients.²⁹

Although catheter ablation of the LBB is technically more challenging than RBB ablation, it can be attempted in select patients with BBR or in patients with IF-VT, as described previously (**Figure 38.5**).^{16,24,26,27,30} Patients with complete antegrade LBBB (i.e., QRS duration >140 ms) may benefit more from LBB ablation as this will eliminate retrograde conduction in the LBB, eliminate induction of BBR, and prevent complete AV block, a likely complication of RBB ablation in the presence of a complete LBBB.¹⁶

After RBB ablation, prophylactic pacemaker implantation was carried out only if the HV interval prolonged significantly (>90–100 ms), or infra-His block could be documented during atrial stimulation.⁵ However, with the advent of biventricular pacing, the role of prophylactic pacing and defibrillator implantation has changed over the years, and the presence of LV dysfunction and congestive heart failure are additional considerations for prophylactic device implantation in these patients. Of note, in 25% of our patients with BBR, a concomitant scar-related SMVT also was induced,^{1,5} another factor when considering device implantation post-ablation.

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EDITORS' SUMMARY

The authors who have taught the electrophysiology community about this unique and fascinating arrhythmia—bundle-branch reentrant tachycardia—provide a well-referenced and well-illustrated summary that is enjoyable to read. Although not a common arrhythmia, bundle-branch reentry is imminently treatable and is a veritable microcosm of all invasive arrhythmia diagnosis. The principles of reset, attempting to find what is in and not in a circuit of a reentrant tachycardia, identifying the driver or the critical link, and the concept of pseudo intervals (the HV during tachycardia and proximal His-V during bundle-branch reentry) are all represented and clearly discussed in this chapter. The early student of invasive electrophysiology would do well to read this chapter along with those on AV node reentry (Chapter 17) and diagnostic maneuvers both for SVT (Chapters 15 and 16) and entrainment (Chapter 43) for a comprehensive foray into the art and science of diagnostic maneuvers for arrhythmia diagnosis.

Jaypee