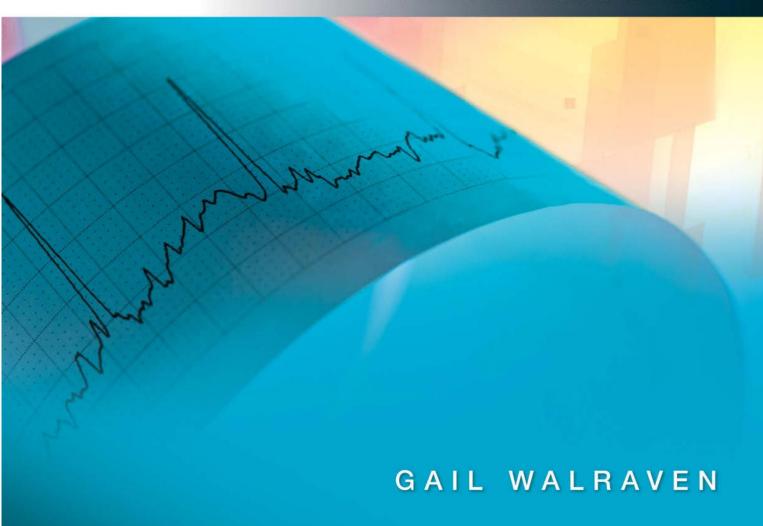
# EIGHTH EDITION Basic Arrhythmias

With 12-Lead EKGs



? Pearson

# **Basic Arrhythmias**

## **Eighth Edition**

#### Gail Walraven

Brady is an imprint of PEARSON



Vice President, Health Science and TED: Julie Levin Alexander

Vice President's Assistant: Sarah Henrich Portfolio Manager: Derril Trakalo

Portfolio Management Assistant: Lisa Narine Vice President, Content Production and

Digital Studio: Paul DeLuca

Managing Content Producer: Melissa Bashe Vice President, Product Marketing: David Gesell Senior Field Marketing Manager: Brian Hoehl Manufacturing Manager: Vatche Demirdjian Manufacturing Buyer: Mary Ann Gloriande **Cover Photo:** Lucidio Studio Inc/Photographer's Choice RF/Getty Images

Creative Director: Blair Brown
Creative Digital Lead: Mary Siener
Managing Producer, Digital Studio,
Health Science: Amy Peltier

Digital Studio Producer - e-text 2.0: Ellen Viganola

Digital Studio Producer: Lisa Rinaldi Digital Content Team Lead: Brian Prybella Composition: iEnergizer Aptara®, Ltd. Printer/Binder: Courier Kendallville Cover Printer: Phoenix Color

**Notice:** The author and the publisher of this book have taken care to make certain that the information given is correct and compatible with the standards generally accepted at the time of publication. Nevertheless, as new information becomes available, changes in treatment and in the use of equipment and procedures become necessary. The reader is advised to carefully consult the instruction and information material included in each piece of equipment or device before administration. Students are warned that the use of any techniques must be authorized by their medical advisor, where appropriate, in accordance with local laws and regulations. The publisher disclaims any liability, loss, injury, or damage incurred as a consequence, directly or indirectly, of the use and application of any of the contents of this book.

Copyright © 2017, 2011, 2006, by Pearson Education, Inc. 221 River Street, Hoboken, New Jersey 07458. All rights reserved. Printed in the United States of America. This publication is protected by Copyright, and permission should be obtained from the publisher prior to any prohibited reproduction, storage in a retrieval system, or transmission in any form by any means, electronic, mechanical, photocopying, recording, or likewise. For information regarding permission(s), write to Pearson Education, Inc. Permission Department. 221 River Street, Hoboken, New Jersey 07458

#### Library of Congress Cataloging-in-Publication Data

Names: Walraven, Gail, (Date) author. Title: Basic arrhythmias/Gail Walraven.

Description: Eighth edition. | Boston: Brady, is an imprint of Pearson, 2016. |

Includes index.

Identifiers: LCCN 2016023195 | ISBN 9780134380995 | ISBN 0134380991 Subjects: LCSH: Arrhythmia—Diagnosis—Programmed instruction. |

Electrocardiography—Programmed instruction.

Classification: LCC RC685.A65 W33 2016 | DDC 616.1/2807547—dc23 LC record available at

https://lccn.loc.gov/2016023195

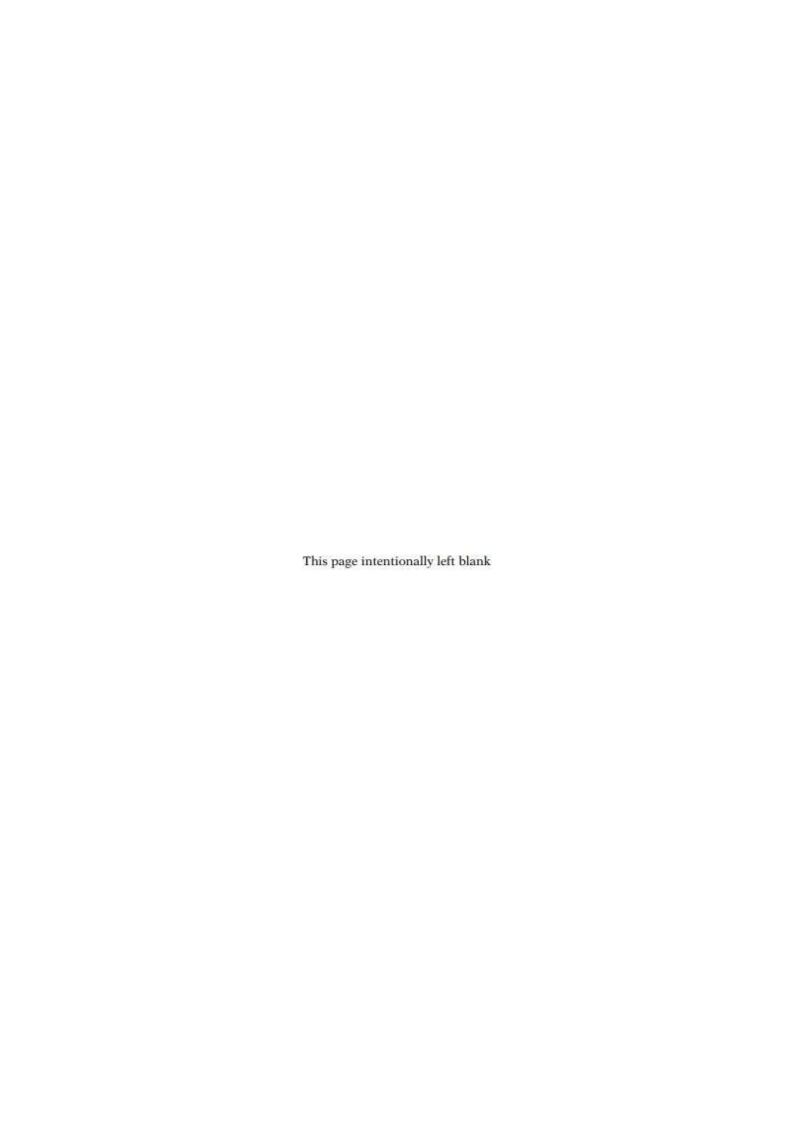


10 9 8 7 6 5 4 3 2 1

Dearson ISBN 12-078-0-12-438000

## **Dedication**

To the men in my life: Bruce, Kellen, Dustin, and Case



## **Contents**

| Preface                              | vii     | P Waves                                     | 54     |
|--------------------------------------|---------|---|--------|
| Navigating through the Text          | xi      | PR Intervals and QRS Complexes              | 55     |
| Learning Objectives                  | xiii    | Role of the AV Node                         | 56     |
| <b>1</b> Electrophysiology           | 1       | Ventricular vs. Supraventricular            | 56     |
| Licetrophtysiology                   | 1       | KEY POINTS                                  | 58     |
| Self-Instructional Unit              |         | SELF-TEST                                   | 58     |
| Electrical vs. Mechanical Function   | 1       | PRACTICE STRIPS (PART I)                    | 61     |
| Impulse Formation                    | 3       | PRACTICE STRIPS (PART II)                   | 65     |
| Polarization, Depolarization         | 4       |   |        |
| Conduction System                    | 5       | <b>4</b> Sinus Rhythms                      | 69     |
| Inherent Rates                       | 6       | Self-Instructional Unit                     |        |
| Irritability, Escape                 | 7       | Introduction                                | 69     |
| Nervous System Influence             | 8       | Normal Sinus Rhythm                         | 70     |
| KEY POINTS                           | 11      | Sinus Bradycardia                           | 72     |
| SELF-TEST                            | 11      | Sinus Tachycardia                           | 72     |
| <b>2</b> M 1 M                       | 1.1     | Sinus Arrhythmia                            | 74     |
| <b>2</b> Waves and Measurements      | 14      | Review                                      | 75     |
| Self-Instructional Unit              |         | KEY POINTS                                  | 77     |
| Introduction                         | 14      | SELF-TEST                                   | 77     |
| Electrodes                           | 15      | PRACTICE STRIPS                             | 79     |
| Rule of Electrical Flow              | 15      | INTERPRETATION OF CHAPTER 3 RHYTHM STRIPS   | 94     |
| Monitoring Leads                     | 16      | INTERNALIZATION OF CHARLETONITY TIMESTILL O | 3-     |
| Graph Paper                          | 17      | <b>5</b> Atrial Rhythms                     | 95     |
| Voltage Measurements                 | 18      |   | ,,,    |
| Time Measurements                    | 19      | Self-Instructional Unit                     |        |
| Cardiac Cycle                        | 19      | Atrial Rhythms                              | 95     |
| Waves, Intervals, Segments           | 21      | Wandering Pacemaker                         | 96     |
| P Wave and PRI                       | 21      | Ectopics                                    | 97     |
| QRS Complex                          | 22      | Premature Atrial Complex                    | 98     |
| ST Segment and T Wave                | 24      | Atrial Tachycardia                          | 99     |
| Measurements                         | 24      | Atrial Flutter                              | 101    |
| Practice                             | 24      | Atrial Fibrillation                         | 103    |
| Artifact, Interference               | 25      | KEY POINTS                                  | 106    |
| Refractory Periods                   | 26      | SELF-TEST                                   | 106    |
| KEY POINTS                           | 28      | PRACTICE STRIPS                             | 109    |
| SELF-TEST                            | 28      | C   | 4.00   |
| PRACTICE STRIPS (PART I)             | 31      | <b>6</b> Junctional Rhythms                 | 138    |
| PRACTICE STRIPS (PART II)            | 33      | Self-Instructional Unit                     |        |
| 0                                    |         | Junctional Pacemaker                        | 138    |
| <b>3</b> Analyzing EKG Rhythm Strips | 49      | Junctional P Wave                           | 140    |
| Self-Instructional Unit              |         | Premature Junctional Complex                | 142    |
| Analysis Format                      | 49      | Junctional Escape Rhythm                    | 144    |
| Regularity                           | 51      | Junctional Tachycardia                      | 145    |
| Rate                                 | 52      | Accelerated Junctional Rhythm               | 146    |
|                                      | 1100000 |   | 20,000 |

#### vi Contents

| Supraventricular Tachycardia                    | 149 | Idioventricular Rhythm  | 228        |
|---|-----|---|------------|
| KEY POINTS                                      | 151 | Asystole  |            |
| SELF-TEST                                       | 151 | Pulseless Electrical Activity (PEA)   |            |
| PRACTICE STRIPS                                 | 154 | KEY POINTS  | 232        |
| <b>7</b> Heart Blocks                           | 175 | SELF-TEST PRACTICE STRIPS   | 233<br>237 |
| Self-Instructional Unit                         |     | 0   |            |
| Conduction Through the AV Node                  | 175 | <b>9</b> Practice Makes Perfect   | 276        |
| Heart Blocks                                    | 176 | Introduction  | 276        |
| First-Degree Heart Block                        | 177 | PRACTICE STRIPS   | 278        |
| Second-Degree Heart Blocks                      | 179 | 10 5: 10: 1   | 100        |
| Type II Second-Degree Heart Block               | 181 | <b>10</b> Final Challenge   | 403        |
| Wenckebach (Type I Second-Degree Heart Block)   | 183 | Introduction  | 403        |
| Third-Degree Heart Block (Complete Heart Block) | 186 | SELF-TEST   | 404        |
| KEY POINTS                                      | 191 |   |            |
| SELF-TEST                                       | 191 | Appendix A: Cardiac Anatomy and Physiology  | 454        |
| PRACTICE STRIPS                                 | 194 | Appendix B: Pathophysiology and Clinical<br>Implications of Arrhythmias   | 462        |
| 8 Ventricular Rhythms                           | 215 | Appendix C: 12-Lead Electrocardiography   | 467        |
| Self-Instructional Unit                         |     | Appendix D: Basic 12-Lead Interpretation  | 480        |
| Ventricular Rhythms                             | 215 |   | F04        |
| Premature Ventricular Complex (PVC)             | 216 | Appendix E: Pacemakers  | 534        |
| Unifocal vs. Multifocal                         | 220 | Glossary  | 544        |
| R on T Phenomenon                               | 221 | Answers Key   | 551        |
| Runs and Couplets                               | 222 | Index of Practice Strips by Rhythm Names  | 586        |
| Grouped Beating                                 | 223 | State which will be a state of the state of |            |
| Ventricular Tachycardia (VT)                    | 225 | Subject Index   | 593        |
| Ventricular Fibrillation                        | 226 | Flash Cards   | 605        |

## **Preface**

There are many ways to learn electrocardiography and many levels of expertise within this complex field. For ease of reference, the various levels of knowledge/ability can be outlined as follows:

# Single-Lead Rhythm Interpretation

**Level III:** Ability to interpret arrhythmias that include more sophisticated features such as sinus arrest, pacemakers, aberrancy, and blocked beats.

#### 12-Lead EKGs

**Level I:** Ability to recognize a limited number of familiar patterns, usually the major life-threatening rhythms. No understanding of rules or mechanisms.

**Level II:** Basic understanding of the rules and mechanisms of common arrhythmias according to pacemaker sites. No familiarity with more sophisticated features that complicate basic arrhythmias.

**Level IV:** Familiarity with 12-lead EKGs (e.g., bundle branch block, infarction location, axis deviation).

**Level V:** Ability to distinguish subtle EKG findings (e.g., concealed conduction, reciprocal conduction, trifascicular block, His bundle recordings).

Levels I, II, and III are concerned with interpretation of patterns and use only single-lead EKG tracings. Levels IV and V are multiple leads used to examine both arrhythmias and the greater complexities of the 12-lead EKG.

It is usual for a student to proceed through these levels in a relatively logical progression from simple to complex. Unfortunately, it is also common for a potential student to be prevented from entering this fascinating field for lack of the initial training upon which to build more sophisticated understanding. *Basic Arrhythmias* is designed to provide a sound base of understanding for those interested individuals who have been unable to receive other forms of training. Its primary area of concentration is Level II, the area of basic understanding of the common, uncomplicated rhythms. It is hoped that *Basic Arrhythmias* will provide an enjoyable and interesting way for people to develop the

framework that will later support continued learning in the area of electrocardiography.

This self-instructional program is targeted toward several groups of people:

- Those who have previously approached EKGs with "pattern recognition"
- Those who have been unable to participate in more conventional EKG training
- Those who are involved in a formal EKG training program and will use this program concurrently to solidify their learning experience

Because the nature of the book is to provide a foundation for future learning, great care was taken to instill simple, basic concepts without giving the student any misconceptions or erroneous impressions. To this end, some information may have been eliminated or lightly passed over because it was considered to be more sophisticated than necessary, and thus might leave the reader confused and without immediate access to an instructor's help. It is hoped that *Basic Arrhythmias* will open the door for new learners and will provide instructors with more class time for the critical area of reinforcement and refinement.

This text was originally designed to be entirely self-instructional with Chapters 1–8 designed in a programmed format to address basic electrophysiology, waves and measurements, rhythm analysis, and the five major groups of arrhythmias. Over the years, additional sections have been appended to add extra practice strips and to cover cardiac anatomy, clinical manifestations, 12-lead electrocardiography, and cardiac pacemakers. The result is that the first half of the book is self-instructional, while the appendices are generally more traditional narrative text. Together, the two sections provide a good foundation for ongoing learning in the field of electrocardiography.

## About the Eighth Edition

Basic Arrhythmias has been well received for many years and has been used successfully by hundreds of thousands of students as they begin to study electrocardiography. It is always a challenge to update and refresh it without detracting from what makes it such a success. With this edition, we've fixed a few bugs, switched out some troublesome EKG strips, added some new illustrations and upgraded some old ones, expanded some sections, and

clarified a few ambiguous areas. But for the most part, the body of the text is not changed significantly. Please note that a compiled list of learning objectives is presented later in this front matter section. These objectives outline in depth what you can be expected to learn and do after completing each chapter.

In addition, we have expanded and improved the learning resources for the *Basic Arrhythmias* program. *MyBradyLab*, an online site dedicated to *Basic Arrhythmias*, presents unique study resources and new learning tools to supplement basic text information.

I am very excited about this eighth edition of *Basic Arrhythmias* and hope you find it as enjoyable as I do. With all its upgrades and its fresh new look, it promises to continue the long tradition of helping students embark on their studies of electrocardiography.

#### What's New in the Eighth Edition

- More 12-leads for practice
- Transition from simplified tracings to more "real-world" 12-leads
- Updated content
- · Addition of some new content for clarification
- Standardization of instructor support materials with new edition of text

## Acknowledgments

It is impossible to fully acknowledge all the people who have contributed over the years to making Basic Arrhythmias what it is today. There have been literally hundreds of people over the years from all across the country who have offered ideas and suggestions, contributed strips, provided clinical reviews, and debated interpretation with me. Without that clinical help, this book would not have achieved the reputation for excellence that it enjoys today. For the eighth edition, I want to again thank Jerrold Glassman, MD, medical director of the Department of Cardiology at Scripps Mercy Hospital, San Diego; he always makes himself available when I come up with a new question. For this edition I am also indebted to Elizabeth Ann Morrell, RN, MSN, NEA-BC, Senior Director, Patient Care, Scripps Mercy Hospitals, Chula Vista and San Diego. And for the tremendous help gathering new EKG strips, I especially want to thank James N. Phan, MBA/HCM, RDCS, RVT, FASE.

Coupled with the clinical excellence, the team at Brady Publishing has set the standard for excellence in the publication of such works. Going all the way back to the original Brady family, countless publishing professionals have added their mark to this book. Most recently, Pearson has moved us to new heights with its print and media

expertise. It is always a joy to work with such professionals. Thank you.

> GAIL WALRAVEN La Jolla, California

#### Instructor Reviewers

The reviewers of *Basic Arrhythmias* provided excellent suggestions for improving the text. Their reviews were an important aid in the revision and updating of material, and their assistance is greatly appreciated.

#### Reviewers of the 8th Edition

Ashley Cheryl, BSN, RN, Education Instructor, Saint Francis Hospital, Department of Education and Professional Development, Tulsa, OK

Scott Jones, MBA, EMT-P, EMS Professor, Victor Valley College Regional Public Safety Training Center, Yucaipa, CA

Lawrence Linder, PhD, NREMTP, Program Manager, Hillsborough Community College, Tampa, FL

#### **Reviewers of Previous Editions**

Lauri Beechler, RN, MSN, CEN, Director, Paramedic Program, Loyola University Medical Center, Maywood, IL

Jackilyn E. Cypher, RN, MSN, NREMT-P, Paramedic Program Course Director, Lead Instructor, Portland Community College, Cascade Campus, Portland, OR

Deborah Ellis, RN, MSN, NP-C Assistant Professor, Missouri Western State University, Saint Joseph, MO

Mary Fuglaar, PHRN, NREMT-B Training Lieutenant, Fort Bend County EMS Rosenberg, TX

Brian Hess, Star Technical Institute Philadelphia, PA

Bradley K. Jordan, EMT-P EMT-P/Level 1 EMS Instructor, Rockingham Community College, Wentworth, NC

Christine Markut, PhD, RN, BC, CNE, Associate Professor, Stevenson University, Stevenson, MD

Lynette McCullough, NREMT-P, MCH Program Coordinator, Paramedic Technology, Griffin Technical College, Griffin, GA

Jeff McDonald, BS, LP Program Coordinator Tarrant County College, Hurst, TX

Mike McEvoy, EMS Coordinator, Saratoga County, NY

Matthew F. Powers, RN MS CEN MICP EMS Chief, North County Fire Authority Fire/Emergency Nursing, Daly City, CA Trent Ragsdell, MBA, Life Support Program Manager/ EMS Coordinator, 55th Medical Group Education and Training Department, Offutt AFB, NE

Ken Schoch, Program Director, Yavapai College Prescott, AZ

Douglas P. Skinner, BS, NREMTP, NCEE, Training Officer, Loudoun County Fire Rescue, Leesburg, VA

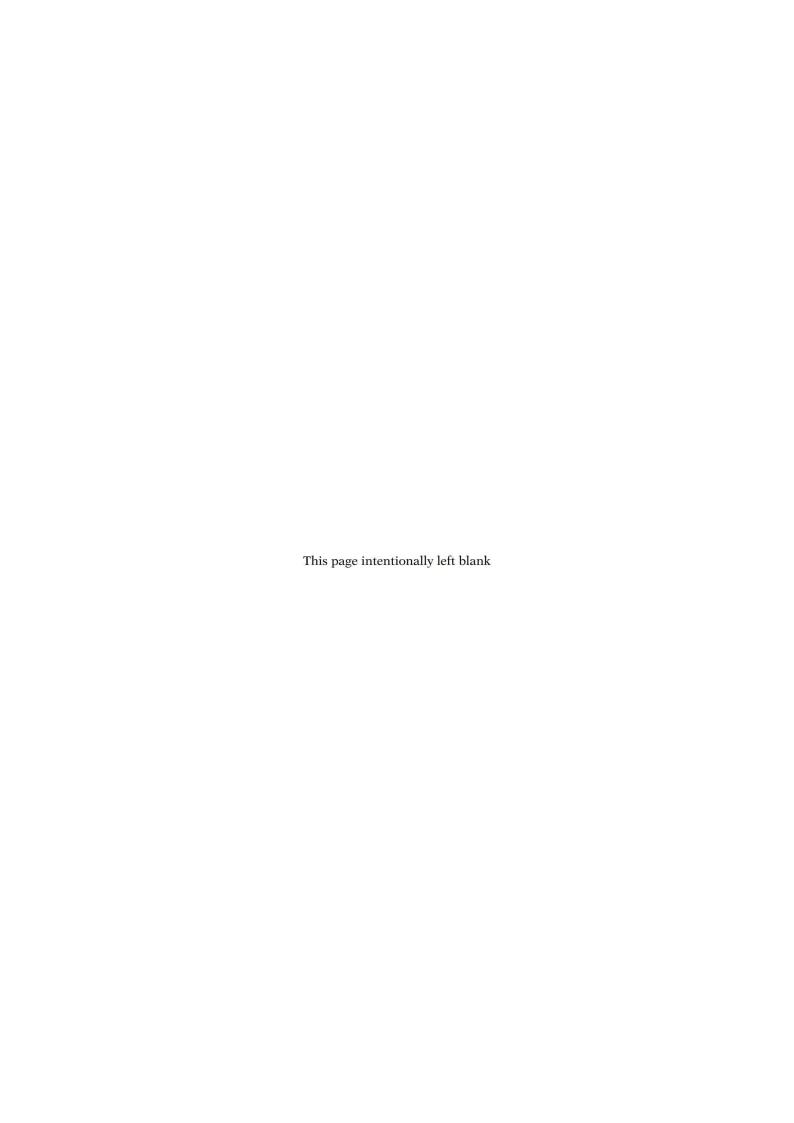
Michael Smertka, EMT-P, EMS, Assistant Instructor, Cleveland Clinic EMS Academy Cleveland, OH

Kimberly Tew, BSN, RN, University of Iowa College of Nursing, Iowa City, IA Paul S. Visich, PhD, MPH, Director of the Human Performance Laboratory, Central Michigan University Mt. Pleasant, MI

Carl Voskamp, MBA, LicP, EMS Program Coordinator, The Victoria College Victoria, TX

Michael L. Wallace, MPA, E T-P, CCEMTP, EMS Captain/Educator, Central Jackson County Fire Protection District, Blue Springs, MO

Charlotte A. Wisnewski, PhD, RN, BC, CDE, CNE, Associate Professor, University of Texas Medical Branch School of Nursing, Galveston, TX



## Navigating Through the Text

The first eight chapters of this text are structured as a self-instructional unit in a "programmed learning" format. As such, it is an entirely self-contained learning package; the only additional materials you will need are a pencil and a set of EKG calipers, which are available at most medical supply houses and medical bookstores. Everything else is provided here in a format designed to make this learning an enjoyable and worthwhile process. Before you start the program, you should know something about the format of these first eight chapters.

Most of the material has been organized into learning units of approximately similar time frames, so that you can pace yourself as you go along. The first three chapters prepare you with the basic principles of electrocardiography and explain many of the theories and concepts that are the foundation of arrhythmia interpretation. Starting with Chapter 4, you will begin systematically learning arrhythmias according to their site of origin within the heart. Finally, you will be given supplemental practice material to ensure retention of your learning and a self-test to validate your knowledge. The internal structure of each individual chapter is essentially as follows.

#### Self-Instructional Unit

This programmed narrative will teach you any rules and explanatory materials that you will need to know to interpret arrhythmias. As you read the text, you will be asked to respond to incomplete sentences or direct questions. The answers are given in the right-hand margin directly across from the question. Before reading the text, get into the habit of covering the margin with this book's Heart Rate Calculator bookmark, located on the back cover flap. Then, as you are asked to respond, write your answer in the blank space provided, and slide the bookmark down the page to reveal the desired response. If you have trouble with an answer, go back over the preceding frames to find the solution.

## **Key Points**

Near the end of each chapter, you will find a brief summary of each key point contained in that chapter. This is provided for your review as you complete the chapter and as a reference should you need to look up a point in the future.

#### Self-Tests

Each chapter closes with a self-test of the important information contained in that chapter. The format of the self-tests is very similar to the format of the text, except that each question is keyed back to the frames in the chapter that specifically provide the answer to the question. Since the self-test is intended to tell you whether or not you learned the material in the chapter, you will want to let a little time pass between completing the chapter and beginning the self-test. If you take the test immediately after finishing the chapter, you might simply be recognizing familiar terms, rather than truly understanding the information. So, once you finish the chapter, take a break from the subject for an hour or two. Then come back to take the test. If you do well on the test, go on to the next chapter. If your results indicate that you did not really learn the material, do not proceed until you remedy that. To do so will merely confuse you and may eventually prevent you from learning future subjects well.

#### **Practice Sheets**

Most of the chapters include at least one Practice Sheet of EKG rhythm strips for you to develop skill in analyzing and interpreting arrhythmias. Since practice is probably the single most important element in developing skill at arrhythmia interpretation, it is critical that you take advantage of the practice time provided.

This book is designed to enable you to teach yourself arrhythmia interpretation. However, your learning will be greatly expedited if you have access to an instructor who will guide your learning and stimulate your thinking. If possible, identify a potential instructor before you begin the program, and arrange for tutorial help should you require it. Then your questions can be answered and your horizons expanded as you progress within this self-instructional course.

#### Flash Cards

Some of the chapters will ask you to take time out from the program to memorize material such as specific rules for each arrhythmia. For your convenience, any material that must be committed to memory has been printed on

#### xii Navigating Through the Text

the flash cards that are provided with this book. This is an effective method of memorizing material in a short period of time. Whenever you are directed to memorize information, stop working on the program and use the flash cards

to accomplish this task. Then return to the text. The intext Flash Card icon, presented in the margin next to corresponding programmed narrative, reminds you to turn to the flash cards.

## Learning Objectives

The objectives below outline in depth what you can be expected to learn and do after completing each chapter.

#### Chapter 1 ELECTROPHYSIOLOGY

- 1 Describe the electrophysiologic basis of cardiac arrhythmias.
  - 1.1 Give the uses and limitations of cardiac arrhythmia monitoring.
    - 1.1.1 Distinguish between the electrical and the mechanical functions of the heart.
    - 1.1.2 Relate cardiac arrhythmia monitoring to pulse/perfusion assessment.
  - 1.2 Explain how cardiac impulses are formed.
    - 1.2.1 Briefly describe the sodium pump.
    - 1.2.2 Define polarization and describe the polarized state.
    - 1.2.3 Define depolarization and explain how it occurs.
    - 1.2.4 Define repolarization and explain how it occurs.
  - 1.3 Describe the heart's electrical conduction system.
    - 1.3.1 Identify the five major areas of electrical conduction.
    - 1.3.2 Outline the physical layout of the conduction system.
    - 1.3.3 Describe the usual pattern of electrical flow through the conduction system.
    - 1.3.4 Give the inherent rates for the SA node, the AV junction, and the ventricles.
  - 1.4 Explain the influence of the nervous system on rate of cardiac impulse formation.
    - 1.4.1 Differentiate between irritability and escape.
    - 1.4.2 Name the nervous system that exerts an influence over rate of cardiac impulse formation.
    - 1.4.3 Identify the two opposing branches of the above-named nervous system, and tell how each would influence the heart if stimulated.
    - 1.4.4 Describe the effect on the heart if one of the branches is blocked.

#### Chapter 2 WAVES AND MEASUREMENTS

- 2 Convey cardiac electrical stimuli to a visible graphic medium suitable for arrhythmia interpretation.
  - 2.1 Demonstrate the monitoring equipment used to detect cardiac electrical activity.
    - 2.1.1 Prepare equipment/materials for monitoring.

- 2.1.2 Demonstrate electrode placement for basic arrhythmia monitoring.
- 2.1.3 Optimize contact between electrode and skin.
- 2.1.4 Select a lead that gives good wave visibility for arrhythmia interpretation.
- 2.2 Cite specifications of the graph paper used to display cardiac electrical activity.
  - 2.2.1 Given the standardized speed at which EKG graph paper is run through the EKG machine, identify the time intervals associated with each of the following:
    - a. time notches in the margins
    - b. one small box
    - c. one large box
- 2.3 Relate the components of a single cardiac cycle to the electrophysiological events that created them.
  - 2.3.1 Differentiate between the following graphic deflections:
    - a. wave
    - b. segment
    - c. interval
    - d. complex
  - 2.3.2 Given a single cardiac cycle, locate each of the following components and describe the electrical events that created it:
    - a. P wave
    - b. PR segment
    - c. PR interval
    - d. Q wave
    - e. R wave
    - f. Swave
    - g. QRS complex
    - h. ST segment
    - i. T wave
  - 2.3.3 Give the normal time duration for each of the following:
    - a. PR interval
    - b. QRS complex
  - 2.3.4 Identify the two phases of the refractory period.
  - 2.3.5 Identify the vulnerable phase of the cardiac cycle.
  - 2.3.6 Recognize deflections on an EKG tracing that were created by something other than cardiac electrical activity.
- 2.4 Differentiate between a single cardiac cycle and an EKG rhythm strip.

## Chapter 3 ANALYZING EKG RHYTHM STRIPS

- 3 Utilize an organized analysis format to gather necessary data from a rhythm strip to interpret the presenting arrhythmia.
  - 3.1 Relate the use of a systematic analysis format to the eventual interpretation of an arrhythmia.
  - 3.2 Outline the five components of an organized approach to rhythm strip analysis.
    - 3.2.1 Describe the pertinent aspects of a systematic analysis of *regularity*, including R–R intervals, P–P intervals, patterns, and ectopics.
    - 3.2.2 Describe the pertinent aspects of a systematic analysis of *rate*.
    - 3.2.3 Describe the pertinent aspects of a systematic analysis of *P waves*, including location, morphology, and patterns.
    - 3.2.4 Describe the pertinent aspects of a systematic analysis of *PR intervals*, including duration, changes, and patterns.
    - 3.2.5 Describe the pertinent aspects of a systematic analysis of ORS complexes, including duration, morphology, and patterns.

#### Chapter 4 SINUS RHYTHMS

- 4 Recognize arrhythmias that originate in the sinus node.
  - 4.1 Describe the characteristics of a sinus pacemaker.
    - 4.1.1 Outline the physiologic mechanisms common to the sinus node.
    - 4.1.2 Describe the expected path of conduction for an impulse originating from a sinus pacemaker.
    - 4.1.3 Identify EKG features common to all arrhythmias in the sinus category.
  - 4.2 Outline the identifying features specific to each of the arrhythmias originating in the sinus node.
    - 4.2.1 Describe *Normal Sinus Rhythm*, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
    - 4.2.2 Describe *Sinus Bradycardia*, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
    - 4.2.3 Describe *Sinus Tachycardia*, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
    - 4.2.4 Describe *Sinus Arrhythmia*, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).

#### Chapter 5 ATRIAL RHYTHMS

- 5 Recognize arrhythmias that originate within the atria.
  - 5.1 Describe the characteristics of an atrial pacemaker.
    - 5.1.1 Outline the physiologic mechanisms common to atrial pacemakers.
    - 5.1.2 Describe the expected path of conduction for an impulse originating from within the atria.
    - 5.1.3 Identify EKG features common to all arrhythmias in the atrial category.
  - 5.2 Outline the identifying features specific to each of the arrhythmias originating within the atria.
    - 5.2.1 Describe Wandering Pacemaker, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
    - 5.2.2 Describe Premature Atrial Complexes, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
    - 5.2.3 Describe Atrial Tachycardia, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
    - 5.2.4 Describe Atrial Flutter, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
    - 5.2.5 Describe *Atrial Fibrillation*, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).

#### Chapter 6 JUNCTIONAL RHYTHMS

- 6 Recognize arrhythmias that originate in the AV junction.
  - 6.1 Describe the characteristics of a junctional pacemaker.
    - 6.1.1 Outline the physiologic mechanisms common to junctional pacemakers.
    - 6.1.2 Describe the expected path of conduction for an impulse originating in the AV junction.
    - 6.1.3 Identify EKG features common to all arrhythmias in the junctional category.
  - 6.2 Outline the identifying features specific to each of the arrhythmias originating in the AV junction.
    - 6.2.1 Describe *Premature Junctional Complexes*, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
    - 6.2.2 Describe Junctional Escape Rhythm, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).

- 6.2.3 Describe Junctional Tachycardia, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
- 6.2.4 Describe Accelerated Junctional Rhythm, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
- 6.2.5 Describe the term Supraventricular Tachycardia, and define circumstances in which it can be used appropriately.

#### Chapter 7 **HEART BLOCKS**

- 7 Recognize arrhythmias that are manifestations of conduction defects at the AV node.
  - 7.1 Describe the characteristics of the category of arrhythmias known as AV heart blocks.
    - 7.1.1 Outline the physiologic mechanisms involved in AV heart block.
    - 7.1.2 Describe the conduction defects encountered in AV heart blocks.
    - 7.1.3 Identify EKG features common to all arrhythmias in the heart block category.
  - 7.2 Outline the identifying features specific to each of the arrhythmias included in the heart block category.
    - 7.2.1 Describe First-Degree Heart Block, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
    - 7.2.2 Describe Type I Second-Degree Heart Block (Wenckebach), including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
    - 7.2.3 Describe Type II Second-Degree Heart Block, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
    - 7.2.4 Describe Third-Degree Heart Block, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).

#### **VENTRICULAR RHYTHMS** Chapter 8

- 8 Recognize arrhythmias that originate in the ventricles.
  - 8.1 Describe the characteristics of a ventricular pacemaker.
    - 8.1.1 Outline the physiologic mechanisms common to ventricular pacemakers.
    - 8.1.2 Describe the expected path of conduction for an impulse originating in the ventricles.
    - 8.1.3 Identify EKG features common to all arrhythmias in the ventricular category.

- 8.2 Outline the identifying features specific to each of the arrhythmias originating in the ventricles.
  - 8.2.1 Describe Premature Ventricular Complexes, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
  - 8.2.2 Describe Ventricular Tachycardia, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
  - 8.2.3 Describe Ventricular Fibrillation, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
  - 8.2.4 Describe Idioventricular Rhythm, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).
  - 8.2.5 Describe Asystole, including etiology, conduction, and resulting EKG features (regularity, rate, P waves, PR intervals, and QRS complexes).

#### PRACTICE MAKES PERFECT Chapter 9

- 9 Identify basic cardiac arrhythmias as presented on 6-second rhythm strips.
  - 9.1 Apply techniques learned in Chapters 1-8 to analyze each of the rhythm strips in Chapter 9.
    - 9.1.1 Use a methodical process to approach data collection (regularity, rate, P waves, PR intervals, and QRS complexes).
    - 9.1.2 Analyze collected data and apply it to rules of each arrhythmia to identify the presenting arrhythmia.

#### Chapter 10 FINAL CHALLENGE

- 10 Test yourself on your ability to identify basic cardiac arrhythmias as presented on 6-second rhythm strips.
  - 10.1 Apply techniques learned in Chapters 1-8 to analyze each of the rhythm strips in Chapter 10.
    - 10.1.1 Use a methodical process to approach data collection (regularity, rate, P waves, PR intervals, and QRS complexes).
    - 10.1.2 Analyze collected data and apply it to rules of each arrhythmia to identify the presenting arrhythmia.
  - 10.2 Demonstrate your competency by approaching all 100 strips in a self-test format, without referring to outside resources.

#### Appendix A CARDIAC ANATOMY AND **PHYSIOLOGY**

- A.1 Describe the heart's location and structure.
- A.2 Identify the four internal chambers of the heart.

- A.3 Describe the heart walls and the pericardial sac.
  - A.3.1 Name the walls between the heart's chambers.
  - A.3.2 Describe the three layers of the heart wall.
  - A.3.3 Differentiate between left and right heart pumping functions.
  - A.3.4 Describe the pumping activity in a single cardiac cycle.
- A.4 Explain the heart's valves.
  - A.4.1 Name the four valves.
  - A.4.2 Explain the valves' role in the heart's pumping cycle.
- A.5 Identify the four heart sounds.
  - A.5.1 Explain the first and second heart sounds.
  - A.5.2 Explain the third and fourth heart sounds.
  - A.5.3 Explain gallop rhythms.
  - A.5.4 Explain heart murmurs.
- A.6 Explain systole and diastole.
  - A.6.1 Describe atrial diastole and systole.
  - A.6.2 Describe ventricular diastole and systole.
- A.7 Explain coronary circulation.
  - A.7.1 State the purpose of coronary circulation.
  - A.7.2 Describe the functions of the coronary arteries, cardiac veins, and coronary sinus.
- A.8 Identify the heart's surfaces.

#### Appendix B PATHOPHYSIOLOGY AND CLINICAL IMPLICATIONS OF ARRHYTHMIAS

- B.1 Describe the clinical effects of arrhythmias.
  - B.1.1 Define cardiac output.
  - B.1.2 Give the formula for a calculated cardiac output.
  - B.1.3 Name three categories of arrhythmia that can interfere with cardiac output.
  - B.1.4 List eight symptoms of reduced cardiac output.
- B.2 Explain the general principles of treating arrhythmias.
  - B.2.1 Name additional measures that may be needed to support perfusion.
  - B.2.2 Explain the role of the American Heart Association in developing and maintaining Advanced Cardiac Life Support recommendations for treating arrhythmias.
- B.3 Explain the significance of each of the 22 basic arrhythmias in this book, and describe the clinical picture of each.

#### Appendix C 12-LEAD ELECTROCARDI-OGRAPHY

- C.1 Explain the advantage of a 12-lead EKG tracing.
- C.2 State the fundamental rules of electrocardiography.
- C.3 Explain leads and electrode placement.
  - C.3.1 Differentiate between bipolar and unipolar leads.
  - C.3.2 Define monitoring lead.
  - C.3.3 Describe the placement of leads on the frontal and horizontal planes.
- C.4 Explain the vectors and axis of electrical flow through the heart.
  - C.4.1 Define mean QRS axis.
  - C.4.2 Explain lead axes.
  - C.4.3 Explain the EKG features of R waves, Q waves, S waves, QS waves, Intrinsicoid Deflections, J Points, and QT Intervals.
  - C.4.4 Explain vector relationships.
  - C.4.5 Describe axis deviation and give the significance of left and right axis deviation.
  - C.4.6 Describe a method for quickly estimating QRS axis.
- C.5 Describe the standardized format for a printed 12-lead EKG report.
  - C.5.1 Explain the importance of learning to recognize normal in 12-lead EKGs.
- C.6 State the limitations of 12-lead EKGs.

## Appendix D BASIC 12-LEAD INTERPRETATION

- D.1 Explain interpreting myocardial damage on the EKG.
  - D.1.1 Define ischemia.
  - D.1.2 Define myocardial infarction.
  - D.1.3 Describe EKG changes associated with myocardial damage.
  - D.1.4 Describe the evolution of ischemic changes (age of infarction).
  - D.1.5 State which leads identify damage on specific surfaces of the heart.
- D.2 Explain interpreting chamber enlargement on the EKG
  - D.2.1 State causes of chamber enlargement.
  - D.2.2 Describe the appearance of chamber enlargement on the EKG.
- D.3 Explain interpreting bundle branch block on the EKG.
  - D.3.1 Define bundle branch block.

- D.3.2 Describe the appearance of bundle branch block on the EKG.
- D.3.3 Differentiate between right and left bundle branch block on the EKG.
- D.4 Explain interpreting other abnormalities on the EKG.
  - D.4.1 Describe the appearance of pericarditis on the EKG.
  - D.4.2 Describe the appearance of digitalis toxicity on the EKG.
  - D.4.3 Describe the appearance of hyperkalemia and hypokalemia on the EKG.
  - D.4.4 Describe the appearance of hypercalcemia and hypocalcemia on the EKG.
- D.5 Explain the format for analyzing a 12-lead EKG.
  - D.5.1 Name the key to easy and effective analysis of the 12-lead EKG.
  - D.5.2 Name the subjects that the summary analysis of an EKG should address.

#### Appendix E PACEMAKERS

- E.1 Describe pacemakers.
  - E.1.1 Explain the purpose of artificial pacemakers.
  - E.1.2 Define capture.

- E.1.3 Name the three components of pacemakers.
- E.2 Name the chambers of the heart that a pacemaker may pace.
- E.3 Describe a "smart" pacemaker.
- E.4 Explain the two basic ways in which pacemakers can initiate impulses.
  - E.4.1 Define triggered pacemaker.
  - E.4.2 Define inhibited pacemaker.
- E.5 Explain the three-letter code system used to classify pacemakers.
- E.6 Explain assessment of pacemaker function.
  - E.6.1 Describe the appearance of pacemakers on the EKG.
  - E.6.2 Describe the basic sequence of assessing pacemaker function.
  - E.6.3 Name the information that can be revealed by the relationship between pacemaker spikes and the patient's complexes.
- E.7 Name and describe four common types of pacemaker malfunctions.
- E.8 Explain how pacemaker malfunction is treated.



# MyBRADYLab™

MyBRADYLab gives you the power to reach students on their terms—and to teach however you like. Encourage students to immerse themselves in the Pearson eText on their own time and use your classroom sessions to workshop key concepts. Or enhance your lectures with other engaging content that brings course material to life.

Fostering engagement both within and outside the classroom, MyBRADYLab helps students better prepare for class, quizzes, and exams—resulting in improved performance in the courses.

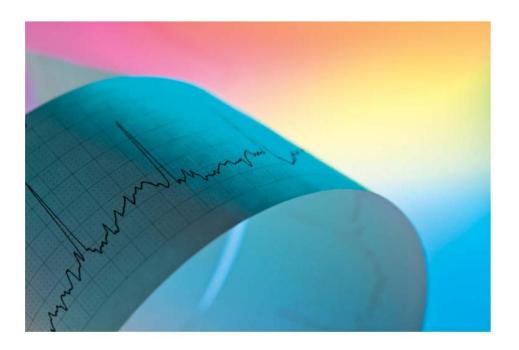
#### Features include

- Gradebook: A fully functional and customizable gradebook allows instructors to view students results by chapter, outcome, homework and more, to help identify where more classroom time is needed!
- Multimedia Library: To help you build assignments, or add an extra engaging element to your lectures, each MyBRADYLab
  course comes with a Multimedia Library.
- Pearson eText: The Pearson eText gives students access to their textbook anytime, anywhere. In addition to note taking, highlighting, and bookmarking, the Pearson eText offers interactive and sharing features.
- Rhythm Randomizer: This innovative exercise is for practice, and can be used as a test of your skills. With a database of
  nearly six hundred randomly generated strips, virtual calipers that you control, and a calculator, you have everything needed
  to practice reading strips. Test yourself and monitor your progress through the integrated "SCORE" function. There's even a
  "HINT" button if you need a little help.
- So much more, visit www.bradybooks.com to learn more!

For more information,
please contact your BRADY sales representative at 1-800-638-0220,
or visit us at www.bradybooks.com

## Chapter 1

## Electrophysiology



#### Overview

**IN THIS CHAPTER,** you'll learn how cardiac arrhythmias reflect what is actually happening electrically in the heart. You will explore the uses and limitations of cardiac arrhythmia monitoring. You will learn how cardiac impulses are formed and how the heart's electrical system conducts electrical impulses throughout the heart. You will also learn how the nervous system can influence the rate at which the heart forms electrical impulses.

#### Electrical vs. Mechanical Function

- **1.** The human heart is intended to pump blood to the rest of the body. This process has two distinct components:
  - The *electrical* impulse that tells the heart to beat
  - The mechanical beating of the heart in response to the electrical stimulation, resulting in pumping of blood

To perform these two functions, the heart has two distinct types of cells. There are electrical (*conductive*) cells, which initiate electrical activity and conduct it through the heart, and there are mechanical (*contracting*) cells, which respond to the electrical stimulus and contract to pump blood. After the \_\_\_\_\_\_ cells initiate

electrical

| the impulse and conduct it through the heart, the  | sodium                                 |
|--|--|
| cells respond by contracting and pumping blood.  | pump                                   |
| 2. The heart will respond with contraction only if it is stimulated by electrical activity. Thus, you cannot have a mechanical response if there is no stimulus.   | electrical                             |
| 3. After the electrical cells have discharged their stimuli, the mechanical cells are expected to respond by $\_\_\_$ .  | contracting                            |
| 4. Without stimulus, the mechanical cells can't be expected to contract.   | electrical                             |
| 5. Since it is not practical to see inside a living patient's heart, we must rely on external evidence to evaluate the status of both electrical and mechanical cardiac function. For a complete assessment of cardiac status, we must evaluate both and functions.  | electrical; mechanical                 |
| 6. As part of our assessment of mechanical function, we use blood pressure, pulses, and other perfusion parameters to determine whether or not the heart is pumping adequately. We must also look for external evidence to evaluate the heart's electrical activity. The best way to do this is to monitor the <i>electrocardiogram</i> (EKG). An EKG tracing is used to evaluate the activity of the heart, while the mechanical activity is evaluated by assessing and and | electrical<br>pulses<br>blood pressure |
| 7. You might occasionally encounter a situation in which the heart muscle is not able to contract in response to the electrical stimulus. In this case, you could have electrical activity but no response. If you had a functioning electrical system but a failing heart muscle, you could very likely see a viable EKG tracing but the patient might not have palpable or blood pressure.   | mechanical pulses                      |
| 8. To evaluate a patient's cardiac function, you must assess the mechanical function by examining and and evaluate electrical function by analyzing the tracing.   | pulses; blood pressure<br>EKG          |
| 9. An EKG tracing is designed to give a graphic display of the electrical activity in the heart. The pattern displayed on the EKG is called the heart rhythm. Technically, the word arrhythmia refers to an abnormal heart rhythm, although the term is often used more generally to refer to all cardiac electrical patterns. The term dysrhythmia is synonymous with arrhythmia; both are used to refer to patterns of activity within the heart.                          | electrical                             |
| 10. An EKG can't tell you about the heart's mechanical activity—you have to assess the patient's pulse and blood pressure to determine that. But an EKG can tell you about the activity, which can be a vital part of your patient assessment. This data is provided in the form of recognizable patterns, called <i>arrhythmias</i> . Arrhythmias are graphic representations of the heart's activity.  | electrical                             |
| 11. To understand and interpret arrhythmias, it is necessary to understand the electrical activity that is occurring within the heart. This is because all arrhythmias are actually graphic displays of electrical activity. The term <i>electrocardiography</i> is given to the study of arrhythmias because arrhythmias are manifestations of activity within the heart.   | electrical                             |
| 12. To help you understand and eventually be able to interpret individual arrhythmia patterns, you might want to know a little bit about the electrical processes that take place in the heart to produce the arrhythmia. To do this we'll consider the electrical   |  |

component independent of the mechanical component. For now, we are discussing only the \_\_\_\_\_\_ activity in the heart.

electrical

## Impulse Formation

13. The electrical (pacemaking) cells in the heart are distinctive in that they can create their own electrical impulses without an outside stimulus. On the cellular level, they create a change in electrical balance in the cell, causing an electrical current to form. This ability of cardiac cells to initiate electrical impulses on their own is called automaticity. Automaticity is the ability of cardiac cells to create their own impulses \_\_\_\_\_\_ an outside stimulus. It is not the whole heart that creates the charge, it's the individual pacemaking \_\_\_\_\_\_ within the heart's electrical system.

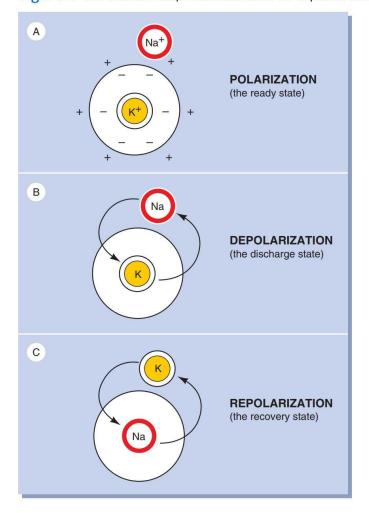
without cells

**14.** The creation of an electrical impulse is a function of electrolytes within cardiac cells, or more accurately, the way those electrolytes move across cell walls. The primary electrolytes involved in creating the heart's electrical stimulus are sodium (Na<sup>+</sup>) and potassium (K<sup>+</sup>). Sodium and \_\_\_\_\_\_\_ are the primary electrolytes that allow the heart to initiate impulses. Both carry a positive electrical charge, but they are not present in equal quantities. The sodium "outweighs" the potassium, making the potassium *relatively* negative to the sodium. It is the *difference* in that potential that allows electrolytes to move through cell membranes. It is movement of these electrolytes through the cell \_\_\_\_\_\_ that creates the electrical impulse.

potassium

membrane

Figure 1 The Sodium Pump: Chemical Basis for Impulse Formation



#### 4 Chapter 1

| <b>15.</b> In a resting cell, the potassium is on the inside and the sodium is on the outside. The outside of the cell is positive, and the inside is <i>relatively</i> negative. The charges are, so no electricity flows. (Figure 1A). As the sodium enters the cell, and the potassium leaves, an electrical charge is created. (Figure 1B) The sodium then returns to the outside of the cell and the potassium goes back in. (Figure 1C) This   | balanced  |
|--|---|
| phenomenon is commonly referred to as the <b>sodium pump</b> . The cycle is repeated for every heartbeat. The refers to the movement of electrolytes in and out of the cell to create an electrical stimulus. When the positive and negative   | sodium pump   |
| charges are balanced, no flows. When the positive and negative charges exchange places, an impulse is formed.  | electricity electrical  |
| 16. For an electrical current to form, there must be a difference between the electrical charges. In the resting cell, the charges are balanced; hence no electricity flows. This is called the <i>polarized</i> state. The cell charges are and ready for action. Polarization refers to a ready state where the electrical charges are and no current flows. When the cell is in its ready state, it is said to be When the charges exchange places in the cell, the result is formation of an current. Once the pacemaker cells provide the stimulation, the flow is passed from cell to cell along the conduction pathways until the cells are stimulated to contract. | balanced<br>balanced<br>electrical<br>polarized<br>electrical<br>cardiac muscle |
| Polarization, Depolarization   |   |
| 17. The polarized state is considered a "ready for action" phase. When the two chemical charges (sodium and potassium) trade places, the electricity flows in a wave-like motion throughout the heart. This wave of electrical flow is called <b>depolarization</b> and is how the electrical stimulus travels through the heart (Figure 1B). Polarization refers to the "ready" state, and refers to the process of electrical discharge and flow of electrical activity.   | depolarization  |
| <b>18.</b> After the cell depolarizes, the positive and negative electrical charges will again return to their original positions around the cell, and the cell will prepare itself for another discharge (Figure 1C). The process that follows depolarization, when the cell charges are returning to their original state, is called <b>repolarization</b> . Repolarization refers to the return of the electrical charges to their position. Repolarization occurs depolarization.  | original<br>after   |
| 19. If each of the positive charges on the outside of the cell is balanced by a negative charge on the inside of the cell, the electrical charges will be balanced, and there will be no movement of electricity. This state is called and can be considered a "ready" state.  | polarization  |
| <b>20.</b> The wave of electrical activity that takes place when the electrical charges surrounding the cell trade places is called, and the return of the electrical charges to their original state is called  | depolarization<br>repolarization  |
| <b>21.</b> If polarization is considered the <i>ready</i> state, and is considered the <i>discharge</i> state, then would be considered the <i>recovery</i> state.   | depolarization repolarization   |
| 22. Now let's relate this cellular activity to what is actually happening in the heart. All of the sequences described in the preceding frames are happening to single cells within the heart, but they do it in alike movement, resulting in the entire heart responding electrically to the same activity.   | wave  |

down the internodal and intraatrial

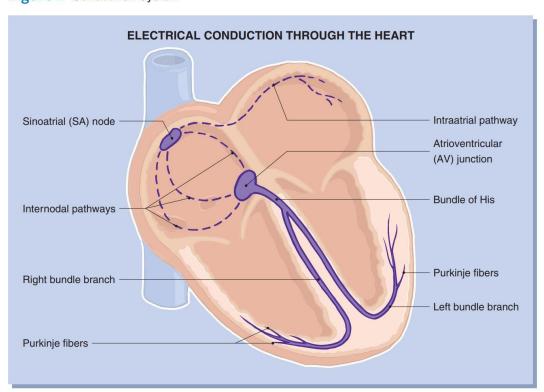
pathways

## **Conduction System**

23. The electrical cells in the heart are all arranged in a system of pathways called the conduction system. The physical layout of the conduction system is shown in Figure 2. This information is an essential part of arrhythmia interpretation and should therefore MyBRADYLab™ be memorized now. (*Note:* Flash cards are provided at the back of this book to help you with this and all other memorizing you will need to do as you learn to interpret arrhythmias. The MyKit website also includes activities to make this learning easier.) Normally, the electrical impulse originates in the SA node and travels to the ventricles by way of the AV node. Look at Figure 2 and trace a normal electrical impulse. Where would the impulse go after it left the AV node and the Bundle of His? down the left and right bundle branches and then to the Purkinje fibers 24. In the normal heart, the first impulse that starts the flow of electrical current through the heart comes from the SA node. The impulse travels through the atria by way of the intraatrial pathways and to the AV node by means of the internodal pathways. If you look microscopically at the cells along these pathways, you would not see any physical difference between them and the cells in other areas of the atria, so researchers have questioned whether they actually exist. However, current electro-

Figure 2 Conduction System

does the current go? \_



physiologic studies support the concept that these pathways do exist, if only as a preferred route by which impulses travel to the AV node. As it leaves the SA node, where

#### 6 Chapter 1

| the AV node. The AV node is ur<br>not have any pacemaker cells<br>making cells are actually locate<br>an area called the <b>AV Junction</b><br>about conduction, but the term | e tissue along the conduction pathway is at the site of aique in that it does have conductive tissue, but it does like other areas of the conduction system. The paced at the junction between the AV node and the atria, in a. Thus, the term AV Node can be used when talking an AV Junction is more accurate if you are specifically bulses. Don't let this confuse you. It is simply an explanation |                      |            |
|---|---|----------------------|------------|
| nation of what might otherwis   | e appear to be indiscriminate use of the two phrases.   |                      |            |
|   | if we're talking only about, ssing pacemaking capabilities, we will call it the AV  | conduction           |            |
|   | pacemaking capabilities, we will call it the 11v  | Junction             |            |
| 26. After leaving the area to reac are located within the right and   | Bundle of His   |                      |            |
|   | bundle branches, smaller fibers distribute the electrical stimulate contraction. These terminal fibers are called   | Purkinje             |            |
| 28. Are the muscle cells themse   | elves part of the electrical conduction system?   | No, they are made up |            |
| <b>29.</b> Rearrange the following parorder of conduction:  | rts of the conduction system to place them in the actual  | MyBRADYLab™          | FLASH CARD |
| (1)   | (a) Bundle of His   | 1. b                 |            |
| (2)   | (b) SA node   | 2. f                 |            |
| (3)   | (c) Purkinje fibers   | 3. e                 |            |
| (4)   | (d) left and right bundle branches  | 4. a                 |            |
| (5)   | (e) AV node   | 5. d                 |            |
| (6) (f) intraatrial pathways  |   | 6. c                 |            |
|   |   |                      |            |

### **Inherent Rates**

**30.** Each of the three major areas of the conduction system has its own built-in rate, called an *inherent rate*, at which it initiates impulses. An inherent rate means simply that each site has a rate range at which it usually produces impulses. A site can exceed or fall below its inherent rate, indicating that these rates are not concrete rules. But generally speaking, the sites will produce impulses at a rate within their own \_\_\_\_\_\_ rate ranges.

**31.** The inherent rate ranges of the major sites are as follows:

20-40 beats per minute

SA Node 60–100 beats per minute
AV Junction 40–60 beats per minute

Ventricle

inherent

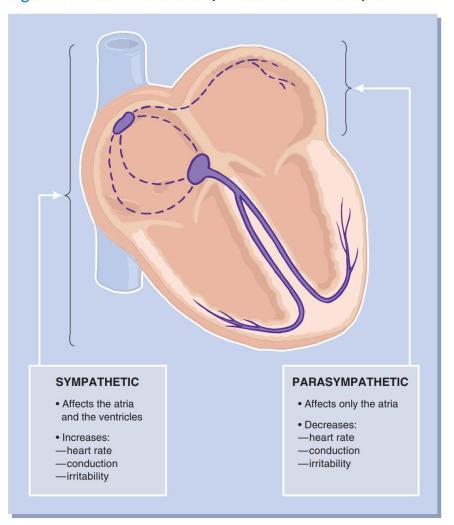


 $MyBRADYLab^{\mathsf{TM}}$ 

| This information would give you a clue that if an EKG rate was between 20 and 40 beats per minute (bpm), the electrical impulse that stimulated the rhythm probably originated in the  | ventricle<br>AV junction<br>SA node |
|--|-------------------------------------|
| <b>32.</b> These rates are often helpful clues to be used in interpreting arrhythmias, but they can be misleading unless they are understood to be mere guidelines and not concrete  | rules                               |
| 33. Generally speaking, the fastest inherent rate will become the pacemaker of the heart and override all other stimuli. The inherent rate of the SA node is the fastest and therefore keeps the heart at a rate between and bpm. Thus, the normal EKG is "sinus" in origin. The SA node is the normal pacemaker for the heart because the rate of the SA node is than the other conduction sites.   | 60<br>100<br>faster                 |
| Irritability, Escape   |                                     |
| 34. If, however, a site becomes irritable and begins to discharge impulses at a faster-than-normal rate, it can override the SA node and take over the pacemaking function for the heart. If the SA node is discharging at a rate of 72 and the AV junction begins to fire at a rate of 95, the will become the pacemaker.   | AV junction                         |
| <b>35.</b> This mechanism of an irritable site speeding up and taking over as pacemaker is called <i>irritability</i> . It is usually an undesirable occurrence, since it overrides the normal pacemaker and causes the heart to beat faster than it otherwise would. Irritability occurs when a site below the SA node and takes over the pacemaking role.  | speeds up                           |
| <b>36.</b> Something very different happens if the normal pacemaker slows down for some reason. If the SA node drops below its inherent rate, or if it fails entirely, the site with the next highest inherent rate will usually take over the pacemaking role. The next highest site is within the, so that site would become the pacemaker if the SA node should fail. This mechanism is called <b>escape</b> and is a safety feature that is built into the heart to protect it in case the normal fails. | AV junction pacemaker               |
| <b>37.</b> Escape mechanism, unlike irritability, is a safety feature to protect the heart. Would you expect an irritable rhythm to be faster or slower than an escape rhythm?   | faster                              |
| <b>38.</b> The inherent rate of different areas of the conduction system refers to the rate at which that site   | initiates impulses                  |
| 39. The SA node has an inherent rate of to bpm. This means that the normal rate of the heart will usually be within that range.  | 60;100                              |
| <b>40.</b> If the rate of an EKG is between 40 and 60, the impulse for that rhythm is probably coming from the   | AV junction                         |
| <b>41.</b> What is the inherent rate of the ventricular conductive tissues?  | 20–40 bpm                           |

| 8 Chapter 1   |   |
|---|---|
| <b>42.</b> Because these rates cannot be relied upon as firm rules, they should be viewed only as If they are used as clues, the rates will be helpful in interpreting arrhythmias, but if they are considered inflexible they will simply confuse the learner.   | guidelines  |
| <b>43.</b> A rule regarding the pacemaker function of the heart states that the site that initiates impulses at the rate will usually become the pacemaker.   | fastest   |
| <b>44.</b> In the normal heart, the initiates impulses at the fastest rate and therefore becomes the  | SA node pacemaker   |
| <b>45.</b> If the AV junction or the ventricle became irritable, either could become the pacemaker if it were able to accelerate until it   | became faster than the SA node  |
| <b>46.</b> The process described in the preceding frame is called   | irritability  |
| <b>47.</b> If the SA node failed as pacemaker, or if its rate dropped below the normal range, the would probably take over as pacemaker.  | AV junction   |
| 48. The safety mechanism described in the preceding frame is called   | escape  |
| Nervous System Influence  |   |
| <b>49.</b> In addition to the inherent rates, the heart can be influenced by the autonomic nervous system. The two branches of this nervous system oppose each other and thus keep the heart in a relative state of balance. The <i>sympathetic branch</i> influences both the atria (i.e., the SA node, the intraatrial and internodal pathways, and the AV junction) and the ventricles. If the sympathetic branch is stimulated, it will cause both the atria and ventricles to react in these ways: | MyBRADYLab™   |
| <ul> <li>Increased rate</li> <li>Increased conduction through the AV node</li> <li>Increased irritability</li> </ul>  |   |
| The <i>parasympathetic branch</i> has the opposite effects, but it influences only the atria; it has little or no effect on the ventricles. While stimulation of the parasympathetic branch causes the atria to slow down, as well as decreasing irritability and slowing conduction through the AV node, stimulation of the sympathetic branch would cause what three effects on the atria and ventricles?   | increased heart rate;<br>increased AV conduction;<br>increased irritability   |
| These nervous influences are outlined in Figure 3.  |   |
| <b>50.</b> If the vagus nerve (which is part of the parasympathetic branch) is stimulated, you would expect the heart rate to On the other hand, if both the sympathetic and the parasympathetic branches are balanced, the heart rate would remain normal. What would you expect if you blocked the normal influence of the vagus nerve?   | decrease  You would get a response similar  |
|   | to stimulation of the sympathetic<br>branch: heart rate would increase<br>as well as irritability and AV<br>conduction. |

Figure 3 Innervation of the Heart by the Autonomic Nervous System



| a drug that would either stimulate the sympathetic branch or  | block the parasympathetic branch                               |
|---|--|
| <b>52.</b> The two branches of the autonomic nervous system that influence heart rate are the branch and the branch.  | sympathetic parasympathetic                                    |
| 53. Which of these branches, when stimulated, will produce an increase in heart rate, AV conduction, and irritability?  | sympathetic  |
| <b>54.</b> One of the branches has control over the atria and the ventricles, while the other influences only the atria. Which one affects both the atria and the ventricles? | sympathetic  |
| <b>55.</b> If both branches are exerting equal influence over the heart, what will happen to the rates?   | They will stay within the ranges of the normal inherent rates. |

| <b>56.</b> What will happen if one of the branches of the autonomic nervous system is blocked?   | The heart will respond to the influence of the opposing branch. |
|--|---|
| <b>57.</b> Using the reasoning described in the preceding frame, explain what would happen to the heart rate if the parasympathetic branch were blocked  | It would increase.  |
| 58. The vagus nerve is part of the branch of the autonomic nervous system. Therefore, stimulation of the vagus nerve would cause the heart rate to, and blocking of the vagus nerve would cause the heart  | parasympathetic   |
| rate to  | increase  |
| <b>59.</b> All of this discussion is about activity and does not yet connect with mechanical activity. In order to discuss the heart contracting and produc-   | electrical  |
| ing a pulse, we must connect the electrical activity withactivity.   | mechanical  |
| 60. If the muscle cells receive an electrical stimulus, they will respond to it by contracting. Sometimes, however, the muscle itself can't contract because it is injured or chemically imbalanced. In these cases the electrical component is all right, but the component needs attention. In such a patient you would expect to find the EKG essentially normal, but the would be absent or diminished.  | mechanical pulse  |
| 61. The opposite situation is more common and is the reason you are reading this book. This is when the heart muscle is able to respond but the electrical activity is erratic. Sometimes the electrical stimuli will make the ventricles contract before the atria do, or maybe there will just be too many electrical stimuli, so that the heart is not able to respond effectively to any of them. And sometimes the electrical impulse will discharge before the ventricles have time to fill with blood, thereby causing the ventricles to contract and eject insufficient blood for an adequate pulse. In all of these conditions, the erratic electrical activity will be seen on the EKG as an | arrhythmia  |
|  |   |

#### **KEY POINTS**

- The heart has two types of cells:
  - · Electrical cells, which initiate and conduct impulses
  - Mechanical cells, which contract in response to stimulation
- Arrhythmias are graphic representations of electrical activity.
- Electrical activity precedes mechanical activity.
- Electrical activity can occur without mechanical response (pulse).
- If the electrical impulse stimulates the mechanical cells to contract, the heart is expected to contract and pump blood, thus producing a pulse.
- Polarization is when the electrical charges are balanced and ready for discharge.
- Depolarization is the discharge of energy that accompanies the transfer of electrical charges across the cell membrane.
- Repolarization is the return of electrical charges to their original state of readiness.
- Depolarization differs from contraction in that depolarization is an electrical phenomenon, whereas contraction is mechanical and is expected to follow depolarization.
- As shown in Figure 2, electrical flow in the normal heart originates in the SA Node, then travels via the intraatrial and internodal pathways to the AV Node, then through the Bundle of His to the Left and Right Bundle Branches, and finally to the Purkinje Fibers, where the mechanical cells are stimulated.

The inherent rates of the conduction system are as follows:

| SA Node     | 60-100 bpm |
|-------------|------------|
| AV Junction | 40-60 bpm  |
| Ventricles  | 20-40 bpm  |

- The site with the fastest rate will be the pacemaker.
- The SA Node is the normal pacemaker of the heart.
- Irritability is when a site speeds up and takes over as pacemaker.
- Escape is when the normal pacemaker slows down or fails and a lower site assumes pacemaking responsibility.
- The influence of the autonomic nervous system can also affect the heart:
  - Sympathetic stimulation causes:

Increased heart rate
Increased AV conduction
Increased irritability

• Parasympathetic stimulation causes:

Decreased heart rate Decreased AV conduction Decreased irritability

- The sympathetic branch influences both the atria (i.e., the SA node, the intraatrial and internodal pathways, and the AV junction) and the ventricles; the parasympathetic branch influences only the atria.
- If one branch of the autonomic nervous system is blocked, the effects of the opposing branch will prevail.

#### **SELF-TEST**

*Directions:* Complete this self-evaluation of the information you have learned from this chapter. If your answers are all correct and you feel comfortable with your understanding of the material, proceed to the next chapter. However, if you miss any of the questions, you should review

the referenced frames before proceeding. If you feel unsure of any of the underlying principles, invest the time now to go back over the entire chapter. **Do not proceed with** the next chapter until you are very comfortable with the material in this chapter.

#### Questions

- 1. Name the two types of cardiac cells and tell what type of activity each is responsible for.
- 2. How do these two types of cells work together to produce cardiac activity?
- **3.** What physical signs are used to reflect the mechanical function of the heart?

| Referenced Frames | Answers   |
|-------------------|---|
| 1, 2, 3, 4        | electrical: conduction;<br>mechanical: contraction    |
| 1, 2, 3, 4        | Electrical cells stimulate muscle cells to contract.  |
| 5, 6, 7, 8        | pulses, blood pressure,<br>other perfusion parameters |