Principles of Cardiac Arrhythmias

SECOND EDITION

Edward K. Chung

Principles of Cardiac Arrhythmias

SECOND EDITION

Edward K. Chung
M.D., F.A.C.P., F.A.C.C.

Professor of Medicine
Jefferson Medical College of
Thomas Jefferson University
and
Director of the Heart Station and
Attending Physician (Cardiologist)
Thomas Jefferson University Hospital
Philadelphia, PA. 19107

Fellow, American College of Cardiology
Former Governor for West Virginia, American College of Cardiology
Fellow, American College of Physicians
Member, American Federation for Clinical Research
Member, American Heart Association
Member, American Medical Association

Editorial Board Member for *Cardiology, The Journal of Electrocardiology, Heart and Lung, Drug Therapy, Primary Cardiology,* and *Journal of International Physicians*

The Williams & Wilkins Company
BALTIMORE

Made in the United States of America

Library of Congress Cataloging in Publication Data

Chung, Edward K
Principles of cardiac arrhythmias.

Includes bibliographical references and index.
1. Arrhythmia. I. Title. [DNLM: 1. Arrhythmia. 2. Electrophysiology. WG330 C559p]
RC685.A65C48 1977 616.1'28 76-51351
ISBN 0-683-01572-9

First Edition, 1971
Reprinted 1973

Italian Edition, 1975

Composed and printed at the
Waverly Press, Inc.
Mt. Royal and Guilford Aves.
Baltimore, Md. 21202, U.S.A.

Preface to the Second Edition

Since the first edition of this book was published in 1971, extensive electrophysiologic investigations have been carried out, particularly in the field of His bundle electrocardiography. This technique has provided valuable information regarding the mechanisms of various cardiac arrhythmias, especially the identification of the exact site of block and genesis of tachyarrhythmias. Thus, a new chapter entitled His Bundle Electrocardiography has been added. For the same reasons, the chapters dealing with the Wolff-Parkinson-White syndrome and the management of cardiac arrhythmias have been significantly expanded.

A new chapter entitled Holter Monitor Electrocardiography has been added because this test, at present, is one of the most valuable noninvasive cardiac diagnostic methods to detect intermittent or transient cardiac arrhythmias and to clarify the etiology of many symptoms, especially fainting episodes and palpitations.

Another new chapter entitled Intraventricular Conduction Disturbances has been added because these disturbances are extremely important clinically. Hemiblocks, bifascicular block and trifascicular block are described in detail. This information is extremely valuable for the understanding of A-V block and the indications for use of artificial pacemakers.

The sections on digitalis and digitalis intoxication have also been markedly expanded by adding discussions of the clinical applications of serum digitalis level determinations.

The whole text has been revised considerably, although the aim and the basic design of this book are essentially unchanged. It can be said that the unique feature of this book is the practical approach with in-depth discussions of the fundamental mechanisms of various arrhythmias. Thus, the clinical significance as well as the therapeutic approaches for various arrhythmias are discussed in detail.

Many illustrative electrocardiograms have been replaced and many new ones added. The author is indebted to the physicians who have been extremely courteous in giving their permission to reproduce some of the electrocardiograms in this book. This is acknowledged in each illustration so used. The author would like to express his sincere appreciation to Onkar S. Narula, M.D., Anthony N. Damato, M.D., Kenneth M. Rosen, M.D. and Douglas P. Zipes, M.D. for providing many valuable illustrations for this book.

Sincere thanks and appreciation are also expressed to Mr. Frank Wright, a medical student at the Thomas Jefferson University, for making excellent reproductions of many of the electrocardiograms for this book.

The secretarial burden was carried out

cheerfully by Miss Theresa McAnally, the personal secretary to the author. Her able assistance and effort have been most valuable in the completion of this book. The endless cooperation of the publisher, The Williams & Wilkins Company, in the preparation of this book is greatly appreciated.

Edward K. Chung, M.D.
Philadelphia, PA.

Preface to the First Edition

The purpose of this textbook is to discuss in depth and to review extensively every known type of abnormal impulse formation and conduction in the human heart. All discussions include electrophysiological principles of the heart as they apply to clinical medicine. The fundamental mechanisms responsible for the production of various arrhythmias are emphasized, as are clinical presentations and therapy when pertinent.

Until the advent of coronary care units and artificial pacemakers, many, if not most, cardiac arrhythmias were only superficially appreciated. These electrical events were considered an isolated subject belonging only to the electrocardiographers. It was not known until recently that arrhythmias occur in nearly 90 per cent of patients during acute myocardial infarction. A full knowledge of cardiac arrhythmias is not only essential for the electrocardiographer, but these disorders must be completely familiar to all who participate in the complete care of the cardiac patient. This includes cardiac surgeons, cardiologists, internists, house staff and specialized nursing personnel. The specialized nurses have become of late, extremely important in the rapid identification and treatment of arrhythmias.

Needless to say, the most common cause of cardiac arrhythmias is coronary heart disease, particularly acute myocardial infarction. Accordingly, the cardiac arrhythmias associated with acute myocardial infarction are discussed in detail.

Among all available drugs, digitalis is still the most valuable in treating various heart diseases and supraventricular tachyarrhythmias. As with all valuable and potent drugs, toxic effects will be manifested if an individual receives an excessive amount of the drug, or if an individual is unable to handle the drug properly because of his genetic makeup or a concomitant disease process. Although the expressions of digitalis toxicity are multifarious, the most important and most frequent manifestation of digitalis intoxication is the development of digitalis-induced cardiac arrhythmias. In fact, digitalis toxicity is one of the most common etiologies in the genesis of cardiac arrhythmias. Therefore, digitalis-induced arrhythmias are fully discussed.

Cardiac arrhythmias related to, or induced by, artificial pacemakers are considered in detail. These arrhythmias not only are clinically important, but may be an extremely valuable tool in understanding the electrophysiology of abnormal cardiac events.

This author is aware that differences of opinion exist in the interpretation of some of the complex cardiac arrhythmias. This may be especially true regarding the

application of some fundamental concepts to interpretation. Terminology also varies in this relatively new field. For these reasons, less commonly used terms are included in parentheses following the more commonly used terms.

The arrangement of the text and illustrations is based upon the author's experience in teaching cardiac arrhythmias to physicians and advanced students.

It should be noted that some of the illustrations have been published previously by this author in reports on specific aspects of cardiac arrhythmias. Hand-drawn diagrams are frequently used in addition to actual electrocardiograms when dealing with explanations of the fundamental mechanisms involved. Pertinent references are included at the end of each chapter for those who desire further study.

The author is indebted to the physicians who have been extremely courteous in giving their permission to reproduce some of the electrocardiograms in this book. This is acknowledged in each illustration so used. My sincere thanks and appreciation are also expressed to Dr. Robert A. Woolfitt, a member of the housestaff at Vanderbilt University School of Medicine, and Mr. Ronald Scobbo, a senior student at West Virginia University, School of Medicine, for their indispensable assistance in the preparation of this book.

The secretarial burden was carried cheerfully by Mrs. Carol Johnson, Mrs. Patricia Berry, Mrs. Donna Knowlton and Miss Brenda Jackson. I also wish to express my gratitude to Dr. Robert J. Marshall, Director of the Cardiopulmonary Division, Dr. Clark K. Sleeth, Dean of the School of Medicine, and Dr. Edmund Flink, Chairman of the Department of Internal Medicine, West Virginia University Medical Center, for their valuable advice and assistance. The endless cooperation of the publisher, The Williams & Wilkins Company, in the preparation of this book is greatly appreciated.

Finally, I will always owe deep gratitude and appreciation to my teachers, Dr. Edward Massie, Director of the Heart Station, Dr. Thomas J. Walsh, Associate Director of the Heart Station, and Dr. Carl V. Moore, Chairman of the Department of Internal Medicine, Washington University School of Medicine and Barnes Hospital in St. Louis, who have always taught and inspired me.

Edward K. Chung, M.D.
Morgantown, W. Va.

Contents

chapter

1 Introduction

GENERAL CONSIDERATIONS

Introductory Remarks

Disturbances of impulse formation and conduction may occur anywhere in the human heart. Over the years, various terms such as "arrhythmia," "dysrhythmia," "ectopic rhythm" and "disorder of heart beat" have been loosely used in order to describe disturbances in cardiac rhythm. Of the above terms, "arrhythmia" is the most commonly used designation. This term has been widely accepted by most cardiologists and electrocardiographers.

Clinical Significance[1-9]

Some cardiac arrhythmias are rather benign and clinically insignificant, whereas others are truly serious and need immediate and specific therapy. The most common cardiac arrhythmia is premature beats (extrasystoles). Premature beats may be atrial, atrioventricular (A-V) junctional or ventricular in origin, and may be found in apparently healthy individuals. However, premature beats may become clinically significant if they occur too frequently, originate from multiple foci or are found in individuals with proven heart disease. Their occurrence during digitalization is certainly of clinical importance. Premature beats will usually disappear with exercise if the individual is free of significant heart disease. Detailed information regarding premature beats may be found elsewhere in this book (Chapters 5, 6 and 9).

The study of cardiac arrhythmias should no longer be of interest only to electrocardiographers or cardiologists. The diagnosis and treatment of cardiac arrhythmias are absolutely necessary skills for all physicians, surgeons and nurses who participate in cardiac care. Because of the wide acceptance of coronary care units and artificial pacemakers, a full understanding of cardiac arrhythmias has become extremely important and essential. It has become obvious that certain cardiac arrhythmias both directly and indirectly influence morbidity and mortality. The mortality rate for myocardial infarction is two to three times higher when complicated by serious arrhythmias, especially those of ventricular origin. Furthermore, the mortality rate for certain digitalis-induced arrhythmias approaches 100 per cent. Cardiac arrhythmias may be the first objective evidence of underlying heart disease; for example, one of the earliest and most common manifestations of acute rheumatic fever is first degree A-V block. The immediate and proper treatment of various ectopic tachyarrhythmias is imperative, for the arrhythmia often precipitates or may be in itself an early sign of impending heart failure. The *correct* diagnosis of various tachyarrhythmias is essential because specific therapy only follows correct diagnosis

and interpretation. Digitalis is often the drug of choice for many of the supraventricular tachyarrhythmias, particularly paroxysmal atrial fibrillation or flutter (unless digitalis-induced); however, digitalis may be contraindicated if the tachyarrhythmia originates from the ventricle. The differential diagnosis of supraventricular and ventricular tachyarrhythmias is discussed in detail in Chapter 15. Management of the common cardiac arrhythmias is described in Chapter 17.

Etiology[3, 9–14]

The most common cause of cardiac arrhythmias is coronary artery disease. It has been shown that 90 to 95 per cent of all patients with acute myocardial infarction have some associated cardiac arrhythmia. The high incidence of arrhythmias during acute myocardial infarction was observed after coronary care units became widely used. The two most common cardiac arrhythmias during an acute myocardial infarction are premature ventricular contractions and sinus tachycardia, in that order.

Digitalis intoxication is another very common cause of arrhythmias. The most common cardiac arrhythmia induced by digitalis is ventricular premature beats, particularly ventricular bigeminy. An equally common arrhythmia in this group is non-paroxysmal A-V junctional tachycardia, especially in the presence of preexisting atrial fibrillation.

The presence of cardiac arrhythmias may at times suggest a specific underlying heart disease or even noncardiac disease. For instance, atrial fibrillation is commonly found in rheumatic heart disease, hyperthyroidism and Wolff-Parkinson-White syndrome, in addition to being a well-known manifestation of coronary heart disease. Full knowledge of the Wolff-Parkinson-White syndrome is important because 70 to 75 per cent of subjects with this syndrome have an associated rapid supraventricular tachyarrhythmia such as reciprocating tachycardia, atrial flutter or atrial fibrillation. A detailed discussion of the Wolff-Parkinson-White syndrome is found elsewhere in this book (Chapter 10). Some arrhythmias are considered almost pathognomonic of certain conditions. For example, ventricular bigeminy, nonparoxysmal A-V junctional tachycardia and atrial tachycardia with A-V block appearing during digitalis therapy almost always indicate digitalis intoxication. Other heart disease such as congenital, rheumatic, luetic, hypertensive and traumatic may at times produce various cardiac arrhythmias. Congenital cardiac arrhythmias such as congenital second degree and complete A-V block have also been reported. Noncardiac conditions, which include metabolic disturbances, anemia, electrolyte imbalance, anesthesia, drug intoxication, collagen disease, central nervous system disorders and psychoneurogenic disorders, may be associated with a variety of cardiac arrhythmias.

Incidence[1, 2, 5, 7–10, 15]

It is almost impossible to determine the exact incidence in the general population of the various cardiac arrhythmias. The incidence varies at different medical institutions depending upon the nature of the institution, the type of patients seen and the ability and interest of the physicians interpreting the electrocardiograms. The highest incidence is usually found in large general hospitals where the elderly and known cardiac patients are usually seen. In general, premature beats (either atrial (auricular), A-V junctional or ventricular), sinus arrhythmia, sinus bradycardia and sinus tachycardia are the most common arrhythmias. Almost as frequent are paroxysmal atrial or A-V junctional tachycardia and first degree A-V block. Atrial fibrillation is probably the next most frequent arrhythmia recorded. Atrial flutter, second degree A-V block and A-V dissociation are relatively infrequent, and complete A-V block, sinoatrial (S-A) block and ventricular tachycardia or flutter are considerably less common. The rare cardiac arrhythmias include parasystole and atrial dissociation.

Classification and Genesis of Cardiac Arrhythmias[1, 2, 5, 15–21]

Cardiac arrhythmias have been classified in various ways, but in general they are divided into two major categories: (1) abnormal impulse formation and (2) abnormal conduction. A detailed classification of cardiac arrhythmias is shown in Table 1–1.

Abnormal Impulse Formation

Active and passive impulse formation are the two fundamental mechanisms involved in impulse formation in the human heart.

In the normal heart, the cardiac impulse originates from the sinus (S-A) node which is called the primary pacemaker. The impulse which originates from the sinus node spreads throughout the atria to produce atrial activation which forms the P wave in the electrocardiogram. After the completion of atrial depolarization (activation), the impulse reaches the A-V node. The time involved for the impulse formed in the sinus node to reach the A-V node is, by definition, the A-V conduction time (the P-R interval on the electrocardiogram). The impulse then travels through the common bundle (bundle of His), right and left bundle branches, left anterior and posterior fascicles and the Purkinje fibers to activate the ventricles and form the QRS complex. This is followed by ventricular repolarization which produces T waves on the electrocardiogram. The wave of atrial repolarization is usually too small to be recorded by the ordinary electrocardiographic apparatus. If it were recorded, it would be superimposed on the QRS complex.

The following are criteria which should be met before the term *normal sinus rhythm* may be used: (1) P wave of sinus origin (normal mean axis of P wave), (2) constant and normal P-R interval (0.12–0.20 second), (3) constant P wave configuration in a given lead, (4) heart rate between 60 and 100 per minute and (5) constant P-P (or R-R) interval. Further information on this important subject is given in Chapter 3.

A sinus rate above 100 per minute is known as sinus tachycardia, whereas a rate slower than 60 per minute is called sinus bradycardia. If the sinus node produces an impulse irregularly, this is termed sinus arrhythmia. When sinus arrhythmia is related to respiration, it is called respiratory sinus arrhythmia. Otherwise, it is known as nonrespiratory sinus arrhythmia. When the sinus node fails to produce an impulse, sinus arrest or pause will result. A finding on the electrocardiogram similar to sinus arrest is S-A block, in which the impulse from the sinus node is unable to spread to the atria because of a block at the S-A junction.

Any cardiac impulse originating from a site other than the sinus node is termed ectopic. When three or more consecutive ectopic beats appear, an ectopic rhythm is said to be present. When two ectopic beats appear consecutively, the terms group beats or paired beats may be applied. Ectopic beats or rhythm may originate from the atria, the A-V junction or the ventricles. The mechanism responsible for ectopic beats or rhythm may be active or passive.

Passive Ectopic Rhythm. Passive ectopic impulse formation is merely a physiological mechanism which maintains ventricular activity. Passive A-V junctional rhythm (commonly known as A-V junctional escape rhythm) may appear on the electrocardiogram when the sinus node produces impulses at a slower rate than usual (sinus bradycardia). This rhythm may also occur when the sinus node is unable to generate an impulse (sinus arrest) or when the sinus impulse fails to be conducted to the A-V junction. The latter may be due to either S-A block or A-V block. When the A-V junction is unable to produce an impulse under these circumstances, the ventricles may then produce a passive rhythm called ventricular escape rhythm or idioventricular rhythm. Conditions which predispose to ventricular escape rhythm are a severely diseased A-V node and bilateral bundle branch block. A-V junctional escape rhythm is much more common than ventricular escape rhythm, simply because the inherent rate

TABLE 1-1
Classification of Cardiac Arrhythmias

I. Disturbances of impulse formation
  A. Disturbance of sinus impulse formation
    1. Sinus premature beats (extrasystoles)
    2. Sinus tachycardia
    3. Sinus bradycardia
    4. Sinus arrhythmia
      a. Respiratory
      b. Nonrespiratory
      c. Ventriculophasic
    5. Wandering pacemaker in the sinus node
    6. Sinus arrest (pause or standstill)
  B. Disturbances of ectopic impulse formation
    1. Passive impulse formation
      a. Atrial escape beats and rhythm
      b. A-V junctional escape beats and rhythm
      c. Ventricular escape beats and rhythm (idioventricular beats and rhythm)
      d. Wandering pacemaker between S-A and A-V nodes
    2. Active impulse formation
      a. Atrial in origin
        (1) Atrial premature beats (extrasystoles)
        (2) Atrial tachycardia
          (a) Paroxysmal
          (b) Nonparoxysmal
        (3) Atrial flutter
        (4) Atrial impure flutter
        (5) Atrial fibrillation
        (6) Atrial flutter-fibrillation
        (7) Multifocal atrial tachycardia (chaotic atrial rhythm, wandering pacemaker in the atria)
      b. A-V junctional in origin
        (1) A-V junctional premature beats (extrasystoles)
        (2) A-V junctional tachycardia
          (a) Paroxysmal
          (b) Nonparoxysmal
        (3) Wandering pacemaker in the A-V junction
      c. Ventricular in origin
        (1) Ventricular premature beats (extrasystoles)
        (2) Ventricular tachycardia
          (a) Paroxysmal
          (b) Nonparoxysmal
        (3) Ventricular flutter
        (4) Ventricular fibrillation
        (5) Chaotic rhythm

II. Conduction disturbances
  A. Sinoatrial (S-A) block
    1. Mobitz type-I (Wenckebach)
    2. Mobitz type-II
  B. Intra-atrial block
  C. Atrioventricular (A-V) block
    1. First degree A-V block
    2. Second degree A-V block
      a. Mobitz type-I (Wenckebach)
      b. Mobitz type-II
    3. High degree (advanced) A-V block
    4. Complete (third degree) A-V block
    5. Dual A-V conduction
    6. Supernormal A-V conduction
  D. Intraventricular block
    1. Right bundle branch block
      a. Complete
      b. Incomplete
    2. Left bundle branch block
      a. Complete
      b. Incomplete
      c. Hemiblocks
        (1) Left anterior hemiblock
        (2) Left posterior hemiblock
    3. Bilateral bundle branch block (Bifascicular block, Trifascicular block)
    4. Nonspecific (diffuse) intraventricular block
  E. Exit block

III. Mixed disturbances of impulse formation and conduction, and ill-defined arrhythmias
  A. Atrioventricular (A-V) dissociation
    1. Complete
    2. Incomplete
  B. Wolff-Parkinson-White syndrome (ventricular preexcitation syndrome)
  C. Reciprocal beats and reciprocating rhythm and tachycardia
  D. Parasystole
    1. Atrial
    2. A-V junctional
    3. Ventricular
    4. Combined
  E. Atrial dissociation
  F. Electrical alternans
  G. Coronary sinus rhythm
  H. Coronary nodal rhythm
  I. Lown-Ganong-Levine syndrome
  J. Concealed conduction

IV. Artificial pacemaker-induced rhythm

of the former (45–60/minute) is faster than the inherent rate of the latter (30–40/minute). A single passive beat from the A-V junction or ventricle is called an A-V junctional escape beat or ventricular escape beat, respectively. Needless to say, a passive ectopic rhythm has a slower rate than that originating from the sinus node. Thus, ectopic escape rhythm does not appear during normal sinus rhythm or sinus tachycardia. If the impulse varies in origin from the sinus node and atria or between the sinus node and A-V junction in successive cycles, a wandering atrial pacemaker is said to be present. On rare occasions, atrial escape beats or rhythm may occur.

Active Ectopic Rhythm. Active ectopic impulse formation is caused by augmented excitability of an ectopic focus. Ectopic foci may originate from the atria, A-V junction or ventricles. These impulses, if single, are known as atrial, A-V junctional or ventricular premature beats (extrasystoles). Six or more consecutive premature beats are termed paroxysmal tachycardia. Active atrial impulse formation results in various atrial tachyarrhythmias which include atrial fibrillation, flutter and tachycardia. Active A-V junctional impulse formation results in either paroxysmal or nonparoxysmal A-V junctional tachycardia. A detailed description of the A-V junctional tachycardias is found in Chapter 6. The most common rapid ventricular rhythm is paroxysmal ventricular tachycardia. Ventricular flutter and fibrillation are less common. Occasionally, there may coexist more than one active ectopic impulse-forming center which results in multifocal tachyarrhythmias. Atrial and A-V junctional tachycardia, double A-V junctional tachycardia and bidirectional A-V junctional or ventricular tachycardia are examples of multifocal tachyarrhythmias. Chaotic rhythm is usually found preceding death. These impulses often originate from multiple ventricular foci.

Conduction Disturbances

Conduction disturbances may occur anywhere in the heart. Heart block occurring at the S-A junction is termed S-A block. If the block is present in the atria, it is termed intra-atrial (intra-auricular) block. The most common form of heart block is A-V block of varying degree. A-V block is divided into three major groups: first, second and third degree A-V block. When heart block exists in the ventricles, it is known as intraventricular block. Intraventricular block includes right and left bundle branch block, hemiblocks, bifascicular and trifascicular block and nonspecific (or diffuse) intraventricular block.

A-V dissociation may result from various combinations of abnormal impulse formation and conduction. In general, A-V dissociation is due to three major disorders: (1) slowing or failure of sinus impulse formation (sinus bradycardia and/or sinus arrhythmia and sinus pause), (2) acceleration of impulse formation in the A-V junction or ventricles (A-V junctional or ventricular tachycardia) and (3) complete A-V block and/or S-A block. A detailed description of A-V dissociation is found in Chapter 6.

Complex arrhythmias usually result from mixed disturbances of impulse formation and conduction. The genesis of some rare arrhythmias such as parasystole, electrical alternans, atrial dissociation, etc. may not be attributable to any of the above mechanisms and some aspects of these arrhythmias are still not clearly understood.

BEDSIDE DIAGNOSIS OF CARDIAC ARRHYTHMIAS[1, 2, 5, 7, 10, 14, 22, 23]

From time to time, it may become necessary to diagnose a cardiac arrhythmia at the bedside. The diagnosis can at times be made by a careful history and complete physical examination. Before performing the physical examination, the physician should avail himself of all clinical information regarding previous cardiac arrhythmias or known heart disease. A detailed history should include information pertaining to the onset, frequency and duration of the cardiac arrhythmia. Concurrent medication should of course be considered in the evaluation of these patients.

Age

Age is an important consideration since the incidence of certain arrhythmias varies among age groups. In older children and young adults, respiratory sinus arrhythmia is almost always present, whereas this arrhythmia is rather unusual in older people. In contrast to this, atrial fibrillation is relatively uncommon in children or young adults, even in the presence of some underlying process. Children with the Wolff-Parkinson-White syndrome rarely have concurrent atrial fibrillation, whereas the latter is certainly not uncommon in elderly patients with the same syndrome. In general, atrial fibrillation is one of the more common arrhythmias in the older age group. When children or young adults develop rapid heart action, it is almost always a regular supraventricular tachycardia (either paroxysmal atrial or A-V junctional tachycardia). A-V block is also uncommon in younger adults or children, except during the presence of acute rheumatic fever. Needless to say, the incidence of practically any type of cardiac arrhythmia increases with age. This is primarily attributable to the increased incidence of coronary heart disease and/or hypertensive heart disease and/or the frequent occurrence of congestive heart failure. Permanent complete A-V block is frequently found in elderly individuals because of long-standing degenerative changes in the conduction system. Atrial fibrillation with rapid ventricular response is extremely common in older patients with acute congestive heart failure. Atrial fibrillation may often persist even if the congestive heart failure resolves. Ventricular tachyarrhythmias predominantly occur in older patients with coronary heart disease.

Rate and Regularity of Cardiac Rhythm

The rate and regularity of the apical and peripheral pulse is also of help in diagnosing cardiac arrhythmias.

Regular Rhythm

A heart rate below 30 per minute is usually due to complete A-V block producing idioventricular rhythm (ventricular escape rhythm). A less likely cause is second degree A-V block. A heart rate between 40 and 60 per minute may be caused by sinus bradycardia, A-V junctional escape rhythm due to complete A-V block and second degree A-V block. In subjects with a heart rate between 60 and 100 per minute, the most common rhythm is normal sinus rhythm, especially when there is no overt heart disease. On the other hand, atrial tachycardia with 2:1 A-V block or nonparoxysmal A-V junctional tachycardia with or without A-V dissociation may also result in a heart rate between 60 and 100 per minute, particularly when digitalis toxicity or acute myocardial infarction is present. Sinus tachycardia is the most likely diagnosis when the heart rate is between 100 and 160 per minute, although atrial flutter or tachycardia with 2:1 A-V response and A-V junctional tachycardia with or without A-V dissociation are less likely possibilities. If the heart rate ranges from 180 to 250 per minute, the most common rhythm is either paroxysmal atrial tachycardia or A-V junctional tachycardia. However, atrial flutter with 2:1 A-V response or ventricular tachycardia must also be considered. Heart rates between 250 and 350 per minute are almost always due to atrial flutter with 1:1 A-V conduction. Rarely, ventricular tachycardia or flutter may also produce this rapid rate.

Irregular Rhythm

The most common cause of an irregular rhythm is premature beats (extrasystoles), either atrial, A-V junctional or ventricular in origin. When an irregular and rapid rhythm (120–250/minute) is present, atrial fibrillation is almost always responsible, although other possibilities such as atrial tachycardia or flutter with varying A-V response and multifocal atrial tachycardia should be considered. Ventricular tachycardia may show slight irregularity on the electrocardiogram but it does not produce any irregularity via auscultation. When the heart rate of an irregular rhythm is between 60 and 100 per minute, this arrhythmia is often due to atrial fibrillation

after digitalization. Less commonly, this irregularity may be due to atrial tachycardia or flutter with varying A-V block, frequent premature beats or even sinus arrhythmia. A significant degree of A-V block should be suspected when the ventricular rate in atrial fibrillation slows below 55 per minute.

Carotid sinus pressure is a very important diagnostic and therapeutic procedure and it is discussed in detail in Chapters 17 and 23.

Changing intensity of the first heart sound at the apex ("cannon" sound) accompanied by a regular rhythm often indicates A-V dissociation, particularly complete A-V block when the atrial mechanism is sinus in origin. Careful inspection of the jugular venous pulse may be helpful in diagnosis. Without question, the precise diagnosis of cardiac arrhythmias can only be made by studying an electrocardiographic tracing.

THE FUNDAMENTAL APPROACH TO INTERPRETATION OF CARDIAC ARRHYTHMIAS[1–3, 10, 15–17, 23–46]

In the analysis of cardiac rhythm disturbances, it is essential to use well-designed electrocardiographic calipers. The calipers should be of such construction that the two legs can be set in any position and the opposing surfaces of the two legs are coplanar so that any rapid or slow rate may be measured. The end of each leg should be sharply pointed.

For a detailed analysis of cardiac arrhythmias, it is desirable to have a long (about 10 feet) rhythm strip of lead II, or sometimes leads III or aVF which show the P wave most clearly. Occasionally, lead V_1 shows the P wave more clearly than the above-mentioned leads. Ideally, long rhythm strips of leads II and V_1 should be used for detailed analysis. A rhythm strip should be taken immediately following the original electrocardiogram when any cardiac arrhythmia is suspected or found, because certain arrhythmias are very transient in nature and may change within a few seconds or minutes. When the cardiac arrhythmia is a rapid one, long strips should be obtained during various procedures, including carotid sinus massage, exercise, etc., in order to clarify the fundamental mechanism involved (see Chapter 23). At times, it is necessary to take a long strip of the electrocardiogram with double speed and/or double standardization in order to magnify the P waves, if they are believed to be present. Rarely, esophageal leads or even atrial intracavitary leads may be obtained for further clarification of the P wave. Recently, His bundle electrocardiography has provided valuable information regarding the mechanisms of various arthythmias, particularly a clarification of the exact site of heart block (see Chapter 16).

In general, the following process should be followed in the analysis of any cardiac arrhythmia:

(1) Acquisition of all available clinical information.

(2) General inspection of the electrocardiogram.

(3) Determination of the dominant rhythm.

(4) Determination of the presence or absence of a P wave.

(5) Determination of the origin of the QRS complex when atrial and ventricular activities are independent.

(6) Determination of the nature and origin of beats occurring prematurely or later than usual.

(7) Concluding interpretation of the cardiac arrhythmia.

Acquisition of Clinical Information

Clinical information is of great help in interpreting cardiac arrhythmias. This information should include the patient's age, presence of a previous similar arrhythmia and its onset and frequency, presence of known heart disease, previous history of congestive heart failure and noncardiac diseases such as hyperthyroidism, drug administration, particularly digitalis, and electrolyte imbalance. A detailed history of an artificial pacemaker implantation, including the approximate date of implantation, type of pacemaker, etc., is important because malfunction of an artificial pacemaker may produce various serious arrhythmias. In addition, one must review

all available previous tracings in order to determine whether the patient had any past cardiac arrhythmia, left bundle branch block, right bundle branch block, hemiblocks or bilateral bundle branch block, Wolff-Parkinson-White syndrome, myocardial infarction, etc. This information is frequently invaluable in distinguishing between supraventricular and ventricular tachycardia (see Chapter 15), and enhances the accuracy of the diagnosis.

General Inspection of the Electrocardiogram

By a general inspection of a given tracing, it is possible to determine whether the basic rhythm is normal sinus rhythm or a type of cardiac arrhythmia. If any arrhythmia is present, one should determine whether the arrhythmia occurs occasionally, frequently, continuously, regularly or irregularly, repetitively or with various combinations. Various noncardiac artifacts which may simulate cardiac arrhythmias (see Chapter 24) must also be detected. It is also possible to determine whether the arrhythmia is simple or complex, clinically benign or serious.

Determination of Dominant Rhythm

After a general inspection of a given electrocardiogram, the dominant rhythm should be determined. The dominant rhythm may be sinus rhythm, but it could be any type of ectopic rhythm. If an ectopic rhythm is dominant, one should determine whether the ectopic rhythm is due to active or passive impulse formation (see Table 1-1). However, in most common and simple arrhythmias, the dominant rhythm is of sinus origin. The second most common dominant rhythm is atrial fibrillation; a less frequent dominant rhythm is atrial flutter. Occasionally, the dominant rhythm may change from one mechansim to another (from sinus to ectopic or vice versa or even from ectopic to ectopic) on the same electrocardiogram. At times, it is difficult to determine the dominant rhythm, particulary when dealing with complex arrhythmias. Even if the dominant rhythm is ectopic in origin, as a rule, one begins with sinus beats, if present even occasionally. It is immensely helpful to determine whether sinus beats are present.

Determination of Presence or Absence of P Waves

By knowing whether a P wave is present or absent, one can narrow the differential diagnosis significantly. When a P wave seems to be present, one should be certain that the P wave is a true P wave and not other waves such as atrial fibrillation or flutter, T wave, U wave or even artifacts which look like P waves. If a true P wave is definitely present, one should determine whether the P wave and QRS complex are related or independent. Measurement of the P-R interval (A-V conduction time) is, by definition, the interval from the onset of the P wave to the onset of the QRS complex. The P-R interval is usually constant in a single lead, but it may differ slightly in other leads because a portion of the P wave or QRS complex may be isoelectric. Therefore, it is ordinarily recommended that the longest P-R interval in the six extremity leads be measured. Dual A-V conduction or any other type of A-V conduction defect should be suspected when the P-R intervals in different leads vary significantly.

Presence of P Wave

When a P wave is present, one should determine whether the P wave is of sinus or ectopic origin. In order to reach a conclusion, the following steps should be carried out:

(1) Determination of origin of P wave (mean axis of P wave).

(2) Inspection of P wave configuration.

(3) Inspection of regularity of P-P cycle.

(4) Measurement of P wave rate.

(5) Determination of the relationship between P wave and QRS complex.

By the above process, if the P wave is found to be of sinus origin, a conclusion can be reached as to whether normal sinus rhythm (see Chapter 3), sinus tachycardia, sinus bradycardia, etc. are present. If the