Principles of Cardiac Arrhythmias

Fourth Edition

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Accurate indications, adverse reactions, and dosage schedules for drugs are provided in this book, but it is possible that they may change. The reader is urged to review the package information data of the manufacturers of the medications mentioned.

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To My Wife, Lisa
and
To Linda and Christopher

Preface

In recent years, many new antiarrhythmic agents have been introduced into clinical medicine, and many other new experimental agents are still under investigation. Thus, a chapter dealing with *Antiarrhythmic Drug Therapy* (Chapter 24) has been completely rewritten and expanded considerably.

In addition, *Electrophysiologic Studies* (Chapter 17) has been totally revised and has grown markedly because of the extensive investigative work at various medical centers in the past decade. I am grateful to my colleague, Dr. Arnold Greenspon, for preparation of this chapter.

New terms such as *"nonsustained"* versus *"sustained"* ventricular tachycardia have been added whenever applicable in various chapters dealing with ventricular tachyarrhythmias.

The whole text has been revised considerably, although its aims and the basic design are essentially unchanged. It can be said that the unique feature of this book is its practical approach with clinical applications and in-depth discussion of the fundamental mechanisms responsible for the production of various cardiac arrhythmias.

The endless cooperation of the publisher, Williams & Wilkins, in the preparation of this book is greatly appreciated.

Lastly, I will always owe deep gratitude and appreciation to my father, Dr. Il-Chun Chung, who has always provided guidance and inspiration.

Edward K. Chung, M.D.
Bryn Mawr, Pennsylvania

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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

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 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%. 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 is dependent on correct diagnosis and interpretation. Digitalis is 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 16. Management of the common cardiac arrhythmias is described in Chapters 22 through 26.

Etiology

The most common cause of cardiac arrhythmias is coronary artery disease. It has

been shown that 90 to 95% 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. Recently it has been well documented that ventricular tachycardia may occur suddenly following re-perfusion of myocardial infarction zone when coronary circulation has been reestablished by various means (e.g., coronary angioplasty) in patients with acute myocardial infarction. However, the re-perfusion arrhythmias are usually transient in most cases.

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 nonparoxysmal A-V junctional tachycardia, especially in the presence of preexisting atrial fibrillation.

In the past decade or so, a relatively newly recognized entity, "mitral valve prolapse syndrome," seems to have very high incidence of various cardiac arrhythmias which include ventricular premature contractions, paroxysmal atrial fibrillation and tachycardia. Of course, this syndrome is much more common in women than men.

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% 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 in 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 (usually Wenckebach type) 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 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.

Remember that many healthy individuals may experience a variety of cardiac arrhythmias, especially following severe emotional distress, excessive physical activities, excessive consumption of coffee, cola drink, alcohol, heavy cigarette smoking and use of various habit-forming drugs. At times, various cardiac arrhythmias may occur in healthy people without any explainable reason. Perfectly healthy young adults and children may have first degree or even Wenckebach A-V block.

Incidence

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

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 to 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 term "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 of the former (45 to 60 per minute) is faster than the inherent rate of the latter (30 to 40 per minute). The heart rate may occasionally be accelerated beyond the usual escape rate ranges in the A-V junctional or ventricular escape rhythm. A single passive beat from the A-V junction or ventricle is called an "A-V junctional escape beat" or a "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.

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

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. On rare occasions, the ectopic impulse may originate from the His bundle or a fascicle of the bundle branch system (see Chapters 9 and 17). 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." Recently, new

terms have been used frequently in the literatures. The term "nonsustained ventricular tachycardia" is used when 6 or more VPCs occur consecutively up to 29 seconds. On the other hand, the term "sustained ventricular tachycardia" is used when ventricular tachycardia lasts more than 30 seconds (see Chapters 9 and 17). 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

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 the same 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 longstanding 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 persist even if the congestive heart failure resolves. Ventricular tachyarrhythmias predominantly occur in older patients with coronary heart disease. Of course, sick sinus syndrome is almost always found in elderly individuals.

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/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 to 250 per 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. Advanced (high degree) A-V block should be considered when the ventricular rate is below 80 beats per minute (particularly less then 60 beats/minute) in atrial fibrillation or flutter.

Carotid sinus pressure is a very important diagnostic and therapeutic procedure and is discussed in detail in Chapters 16 and 22.

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. A pulse deficit is a rule rather than exception in atrial fibrillation, especially when the ventricular rate is fast. The pulse deficit may often be found as a result of VPCs. 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

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 do 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 20). 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 arrhythmias, particularly a clarification of the exact site of heart block (see Chapter 17). The 24 to 48 hour Holter monitor ECG is necessary when detailed information regarding any suspected cardiac arrhythmia is desired (see Chapter 18).

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 any unusual personal habits including the use of any habit-forming drugs, alcohol, etc., previous presence of a 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 16), 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 29) 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 to 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 mechanism 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, particularly 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 wave is a true P wave and not a wave such as atrial fibrillation or flutter, T wave, U wave or even an artifact which looks like a P wave. If a true P wave is definitely present, one should determine whether the P wave and QRS complex are related or are 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. In general, lead II is recommended to measure the P-R interval. When the P-R interval is extremely short, the atrial and ventricular activities may be unrelated, so that A-V dissociation is produced (see Chapter 6). 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 P wave is not of sinus origin, it must be originating from an ectopic focus in the atria or A-V junction or, rarely, in the ventricles. The P wave which originates in the atria may resemble or at times be almost identical to the sinus P wave, but the rate is usually faster when impulses originate in the atria. The P wave may be conducted in a retrograde fashion if it originates from either the A-V junction or ventricles. If this occurs, the P wave will be inverted in lead II but upright in lead aVR, and thus it will have a direction almost opposite to that of a sinus P wave. In addition, the P waves may also be conducted in a retrograde fashion when the cardiac impulses arise from the posterior and inferior portion of the left atrium (see Chapter 5). When the atria are activated in a retrograde fashion from the A-V junction, the P wave may appear before or after the QRS complex, depending upon whether the atria or ventricles are activated first. If the atria and ventricles are activated simultaneously, the P wave will be superimposed on the QRS complex, leading to an absent P wave. When the P wave configuration changes from beat to beat in the presence of a constant P-R interval, a wandering pacemaker is present in the sinus node. The pacemaker may also wander in the atria. This may be diagnosed when one observes a changing P-R interval with fluctuations in the P wave configuration. A wandering pacemaker is present between the sinus node and A-V junction when the P wave configuration changes from upright to inverted in the same lead with or without a changing P-R interval. Respiration may slightly change the P wave configuration in some leads and these changes should be distinguished from a wandering atrial pacemaker. Respiration may also affect the QRS complex and T wave in a similar fashion. An electrocardiogram taken during sustained inspiration or expiration will eliminate this problem. On rare occasions, the P wave configuration may change only after ectopic beats. This is due to aberrant atrial conduction (Chung's phenomenon; see Chapter 15). A varying P wave configuration is also observed when atrial fusion beats of varying degree appear on the electrocardiogram. This is seen with atrial parasystole or atrial dissociation (see Chapters 12 and 13).

An irregular P-P cycle is usually due to sinus arrhythmia but may also occur with intermittent S-A block, sinus arrest or even sinus premature beats. Ectopic atrial beats which

appear during sinus rhythm also result in an irregular P-P cycle, but the configuration of the ectopic P wave is usually different from that of the sinus P wave. Knowing the rate of the P wave allows the cardiac rhythm to be determined with relative accuracy. A P wave rate between 60 and 100 per minute with the P wave conducted in a forward direction is usually indicative of normal sinus rhythm. Nonparoxysmal A-V junctional tachycardia often produces the same rate as normal sinus rhythm but the P wave during the former is conducted in a retrograde fashion. In general, nonparoxysmal A-V junctional tachycardia produces retrograde P waves with a rate between 70 and 130 per minute. When the rate of the P wave is between 180 and 250 per minute, the ectopic rhythm is usually either paroxysmal atrial tachycardia or paroxysmal A-V junctional tachycardia. The P wave is conducted in a forward fashion in the former but in a retrograde fashion in the latter. A-V junctional escape rhythm may produce retrograde P waves, but at a much slower rate (40 to 60 per minute) than A-V junctional tachycardia. Rarely, idioventricular rhythm (ventricular escape rhythm) may show retrograde P waves. These P waves usually occur after the QRS complex. It is not uncommon to find a retrograde P wave following artificial pacemaker-induced ventricular rhythm. A retrograde P wave also can be produced in reciprocating rhythm or tachycardia (see Chapter 6).

The final step in this subgroup is to appreciate the relationship between atria and ventricles. If the P-R or R-P intervals are constant, all the above mentioned diagnostic possibilities (normal sinus rhythm, sinus bradycardia, sinus tachycardia, atrial tachycardia, A-V junctional tachycardia, A-V junctional escape rhythm and idioventricular rhythm) should be considered. This list should be narrowed after careful observation of the direction and rate of P waves. If the P-R interval varies, one must be certain whether or not the atria and ventricles are related in the cardiac cycle. The degree and frequency of the above relationship must be determined. When no relationship exists between the atria and ventricles, complete A-V dissociation is said to be present. The underlying disorders responsible for A-V dissociation may be 1) a marked slowing of sinus impulse formation, 2) acceleration of ectopic impulse formation in the A-V junction or ventricles and 3) complete A-V block (see Chapter 8). Complete A-V dissociation need not always be present, for the atria and ventricles may at times become related (atrial or ventricular captured beats) and this rhythm is known as incomplete A-V dissociation. Various complex mechanisms, including supernormal A-V conduction, concealed conduction and unidirectional block, are often responsible for the production of captured beats, particularly when A-V block exists (see Chapter 8).

When the atrial rate is found to be a multiple of the ventricular rate, second degree or advanced (high degree) A-V block, such as 2:1, 3:1, etc., is usually present. Wenckebach A-V block (Mobitz type-I) is characterized by a gradual lengthening of the P-R interval until a P wave is present without a QRS complex after it. This pattern may then repeat itself. Thus, the atrial to ventricular ratio becomes 3:2, 4:3, etc. The less common form of second degree A-V block, namely Mobitz type-II, also produces a 3:2, 4:3, etc. A-V block which may resemble the Wenckebach type, but the P-R intervals remain constant except when a blocked (nonconducted) P wave occurs.

First degree A-V block has a constant but prolonged (0.21 second or more) P-R interval. The relationship between atria and ventricles may occasionally be reversed; Wenckebach retrograde ventriculoatrial block may be the resulting abnormality.

Absence of P Wave

When the P wave is not discernible, one should determine whether: 1) the P wave is truly absent or 2) the P wave is falsely absent.

It is not uncommon to observe a P wave superimposed on a portion of the QRS complex, S-T segment or T wave of the preceding or subsequent cycle. This occurs frequently during atrial tachycardia or A-V junctional tachycardia and may occasionally be observed during sinus tachycardia. Various maneuvers such as carotid sinus pressure, breath-holding, etc. may enable the physician to delineate the P wave from other complexes by reducing the heart rate.

If after the above techniques the P wave is still not observed, the atrial mechanism must be determined. The most common cause of an absent P wave is atrial fibrillation; a less common cause is atrial flutter. In these cases, an atrial fibrillation or flutter wave is present in place of the P wave. Untreated atrial fibrilla-

tion usually has a rapid ventricular rate (more than 120 to 160 per minute), unless a significant A-V conduction defect is present. In untreated atrial flutter, the ventricular rate is usually one-half that of the atrial. This block is not anatomic in nature. Its presence is due to an inability of the A-V junction to conduct the very rapid atrial rate. Thus, the ventricular rate is frequently around 150 to 175 per minute. In A-V junctional tachycardia or escape rhythm, the P wave may be totally superimposed on the QRS complex, leading to absent P waves. This occurs when atrial and ventricular depolarization occur simultaneously. In rare circumstances, notably in severely diseased hearts, atrial activity may be completely absent because of atrial standstill.

Determination of the Origin of the QRS Complex When Atrial and Ventricular Activities Are Independent

When the P wave and QRS complex are either temporarily or continuously independent, incomplete or complete A-V dissociation occurs. When this occurs, either the atrial or ventricular mechanism may equally dominate. In other words, the atria may be controlled by the sinus node or any other atrial ectopic focus (atrial fibrillation, flutter or tachycardia), whereas the ventricles may be controlled by either the A-V junction or a ventricular ectopic focus.

When the QRS complex is unrelated to the P wave, the fundamental genesis of the impulse which activates the ventricles should be determined. It is essential to determine whether the QRS complex is produced by active or passive impulse formation. In addition, it should be determined whether the QRS complex originates from the A-V junction or the ventricles; if there is active impulse formation, A-V junctional or ventricular tachycardia, respectively, will result. In contrast to this, passive impulse formation from the A-V junction or ventricles results in either A-V junctional or ventricular escape rhythm, respectively. The QRS complex which originates from the A-V junction is ordinarily of normal configuration, although it may be slightly bizarre and wider than usual because of aberrant ventricular conduction. Aberrant ventricular conduction may occur when there is an extremely rapid rate or Ashman's phenomenon (see Chapter 15). The QRS complex which originates in a ventricle is usually wide and has a bizarre configuration. When the cardiac impulse originates from the His bundle, the QRS configuration is identical to the normal sinus beat. When the QRS contour demonstrates incomplete right bundle branch block *pattern* in the presence of A-V dissociation, the cardiac impulse is considered to be arising from one of the fascicles of the left bundle branch system.

Determination of the Nature and Origin of Beats Occurring Prematurely or Later Than Usual

Various fundamental mechanisms may produce a P wave or QRS complex which occurs prematurely. The most common example of this is the ordinary premature beat (extrasystole) which may originate from the atria, A-V junction or ventricles, and rarely from the sinus node or bundle branch system. When a premature beat is found, one should determine its origin. The origin of a premature P wave is determined by the electrical axis and configuration of the P wave and its relationship to the QRS complex. The configuration of the P wave of a sinus premature beat is identical to the P wave of the underlying sinus rhythm, whereas the P wave of an atrial premature beat is usually slightly different in configuration from the sinus P wave. An atrial premature beat is ordinarily followed by a normal QRS complex. When a short coupling interval (interval from the ectopic beat to the preceding beat of the basic rhythm) and/or Ashman's phenomenon are present, the QRS complex following an atrial premature beat may appear bizarre because of aberrant ventricular conduction. At times, a premature P wave may not be followed by a QRS complex and this is termed a nonconducted or blocked atrial premature contraction. When the P wave is conducted in a retrograde fashion, the relationship of the P wave to the preceding or the following QRS complex or both should be determined. The P wave of an A-V junctional premature beat may be preceded or followed by the QRS complex, depending upon whether the atria or ventricles were activated first. When a retrograde P wave is related to both the preceding and the following QRS complex and becomes placed between them, a reciprocal beat is usually present (see Chapter 6).

The QRS complex of A-V junctional premature or reciprocal beats may show a bizarre