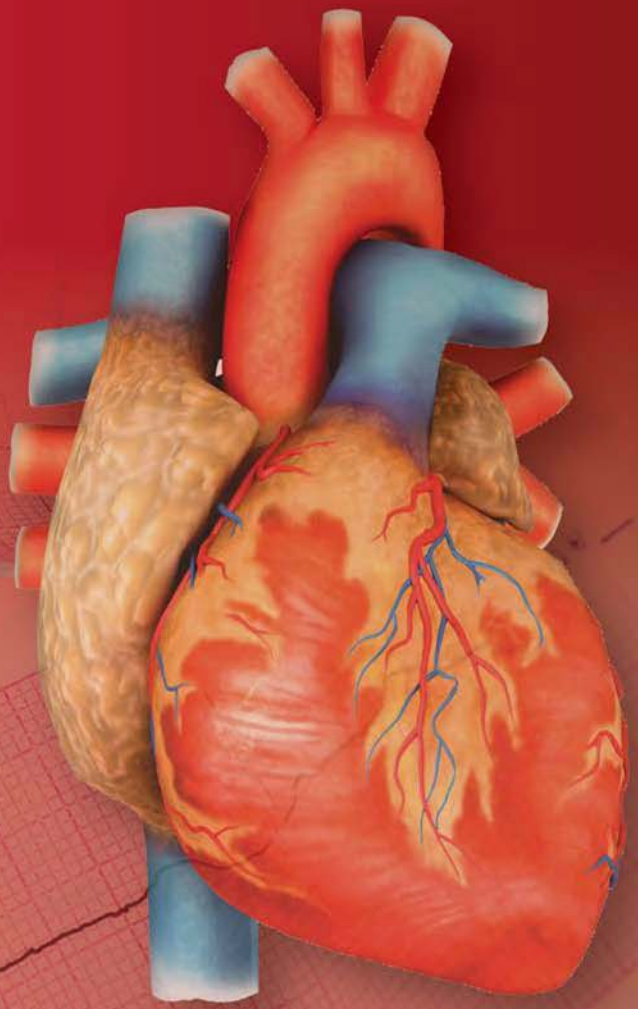
CARDIAC ARRHYTHMIAS

Interpretation, Diagnosis, and Treatment



SECOND EDITION

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GEORGE J. KLEIN
JAMES P. DAUBERT

Mc Graw Hill

Cardiac Arrhythmias: Interpretation, Diagnosis, and Treatment

Second Edition

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Cardiac Arrhythmias: Interpretation, Diagnosis, and Treatment

Second Edition

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To my wife Bonnie, my constant source of love and support for over 50 years.
To my sons David and Daniel, whose lives have filled me with pride and joy, and their wives, Malia and Beth, who are the daughters we never had.
And for my grandchildren, Dylan, Laila, Amber, and Noah, who always bring boundless joy and sunshine into our lives.
And in loving memory of my parents, Drs. Rose and Milton Prystowsky, compassionate physicians who taught me that being a physician is not a gift to be wasted.

—Eric N. Prystowsky, MD

To my wife and best friend, Klara.
To my son Ben and daughter-in-law Elissa and their children Adam, Leah, Beth, and Talya, and to my daughter Anna and son-in-law Mark and their children Lucy, Nate, and Sari, who are all my real source of pride and joy.
To the memory of my unselfish and giving parents, Paul and Clara.

—George J. Klein, MD

To the memory of my late parents, Dr. James J. Daubert and Irene M. Daubert, recognizing their inspiration for and support of my education.
To my wife Amy for her tremendous support and wise counsel throughout my medical career.
And to our children, Patrick, Thomas (MD), Mairin, and James.

—James P. Daubert, MD

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Contents

Preface

Twenty-five years ago, Drs. Prystowsky and Klein authored the first edition of this textbook. Our purpose was to "write a comprehensive textbook on an integrated approach to cardiac arrhythmias for individuals without a background in clinical electrophysiology." While we hoped clinical electrophysiologists would find our book useful, our intent was to educate the broader field of clinicians, for example, medical students, house staff, primary care physicians, cardiologists, and nurses, on the diagnosis and treatment of patients with cardiac arrhythmias. Thousands of copies of the book were sold, and we hope those readers found it useful. We received much feedback from our colleagues and trainees over the years and discussed with each other some alterations to consider when we wrote a second edition. While other textbooks on cardiac arrhythmias are available, we felt there remained a void in providing our practical application of electrophysiological principles and electrocardiographic correlations to the broader community involved in caring for patients as well as the clinical electrophysiologist. This motivated us to pursue a second edition of our book. We welcome our new co-author, Dr. James Daubert, also a Duke-trained electrophysiology fellow, and long-standing friend and colleague.

There has been a sea change in the last 25 years to our therapy of cardiac arrhythmias. Surgery to cure arrhythmias is nearly extinct, and new antiarrhythmic drug development has been stagnant. At the same time, there have been remarkable advances in catheter ablation technology and its application to treat a wide variety of cardiac arrhythmias, as well as development of newer implantable cardiac electrical devices and indications for their use. The content of our second edition reflects these changes in our field, and we have deleted specific chapters on arrhythmias in acute myocardial infarction, pharmacologic therapy, operative therapy of arrhythmias, and noninvasive methods. Important information on drug therapy and noninvasive methods is covered in other chapters. Further, we have added new chapters on atrial fibrillation, catheter ablation of supraventricular tachycardia, catheter ablation of ventricular tachycardia, and provide a major update in the chapters on sudden cardiac death and device therapy. Many new figures and information have been added to the existing chapters. To allow for easier reading, there is some deliberate overlap of information between various chapters. Key references of classic and up-to-date articles are provided for each chapter.

Like the first edition, this book represents our personal approach to patients with cardiac arrhythmias. Nonetheless, it would be unrealistic to expect 3 electrophysiologists to have entirely uniform approaches to diagnosis and therapy. Hence, the primary author's views in individual chapters prevailed when there were minor differences in approach. Each of us reviewed all the chapters and made recommendations for change. In the end, we did not feel there were any substantial differences in our approach to patient care and were comfortable in signing off on the total content.

The book is divided into 4 parts. *Part I* is Basic Electrocardiographic Observations and Clinical Electrophysiologic Correlates. There are in-depth discussions of cardiac conduction with major additions on the effects of alterations in autonomic tone, effects of premature atrial and ventricular ectopy on conduction and automaticity, concealed conduction and apparent paradoxical conduction, bundle branch block, and mechanisms of tachycardia. *Part II* focuses on specific arrhythmias. It begins with an in-depth chapter on a clinical classification of supraventricular tachycardia and includes chapters on the preexcitation syndrome (Wolff-Parkinson-White), ventricular tachycardia, atrial fibrillation, and bradycardia. Diagnostic information from patient history, physical examination, and laboratory tests is given, as well as an approach to therapy for each arrhythmia. *Part III* includes chapters on common clinical presentations of arrhythmias. An approach is given for patients who are asymptomatic but have electrocardiographic abnormalities, and for those who present with a narrow QRS tachycardia, wide QRS tachycardia, syncope, dizziness, palpitations, or who have survived a cardiac arrest. *Part IV* consists of chapters on diagnostic techniques and therapeutic modalities. Included are methods of electrophysiologic testing and diagnostic pacing maneuvers, catheter ablation of supraventricular tachycardia, catheter ablation of ventricular tachycardia, and device therapy.

Acknowledgements: Dr. Prystowsky thanks Mrs. Jane Gilmore, CMI, for her outstanding artwork.

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PART I

Basic Electrocardiographic Observations and Clinical Electrophysiologic Correlates

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1

Cardiac Conduction

Eric N. Prystowsky, MD

GENERAL CONCEPTS

The normal cardiac impulse originates in the sinus node, a crescent-shaped structure approximately 9 to 15 mm long that is located at the juncture of the superior vena cava and right atrium (**Figure 1-1**).[1] After the electrical impulse exits the sinus node, it proceeds to activate the right and left atria. Activation of the atria is responsible for the P wave recorded on the electrocardiogram (ECG) (Figure 1-1, orange color). Activation of the normal human atria takes approximately 90 to 100 ms, the right atrium being activated within approximately 65 ms.[2] The last area to be activated is the left atrial appendage, although atrial tissue near the left inferior pulmonary vein can also be activated very late. It should be noted that as the spread of atrial activation occurs, some sections of the right and left atria are activated at the same time.[2]

A controversy has existed for decades concerning the existence of specialized internodal pathways for conduction between the sinoatrial (SA) node and the atrioventricular (AV) node. The essence of this controversy is whether preferential atrial conduction between the SA and AV nodes occurs over specialized pathways or whether the activation wavefront proceeds through nonspecialized or ordinary atrial myocardium.[1,3] Most data strongly suggest that no specialized pathways of conduction exist. The observed preferential conduction of atrial impulses along various anatomical routes can be explained adequately by various factors, such as the complex anatomy of the right atrium, which includes multiple "holes" such as the orifices of the inferior and superior venae cavae and coronary sinus ostium, as well as the orientation of atrial fibers in longitudinal and perpendicular directions, with conduction being faster in the longitudinal direction.[4]

The PR interval in the ECG encompasses activation in the atria, AV node, His-Purkinje system, and ventricles (Figure 1-1). The AV node is a complex structure that includes the compact node and its posterior extensions.[5] The AV node was initially considered to be subdivided into 3 functional zones: the AN, N, and NH zones.[6-8] The N zone appears to be the most common area where block occurs in the AV node. However, a more recent microelectrode mapping of the AV node suggests that it includes 6 different cell types.[9] Slow conduction through the AV node is multifactorial, in part related to the complex arrangement of nodal cells and connective tissue, reduced electrical cellular coupling, and action potentials that are dependent on the

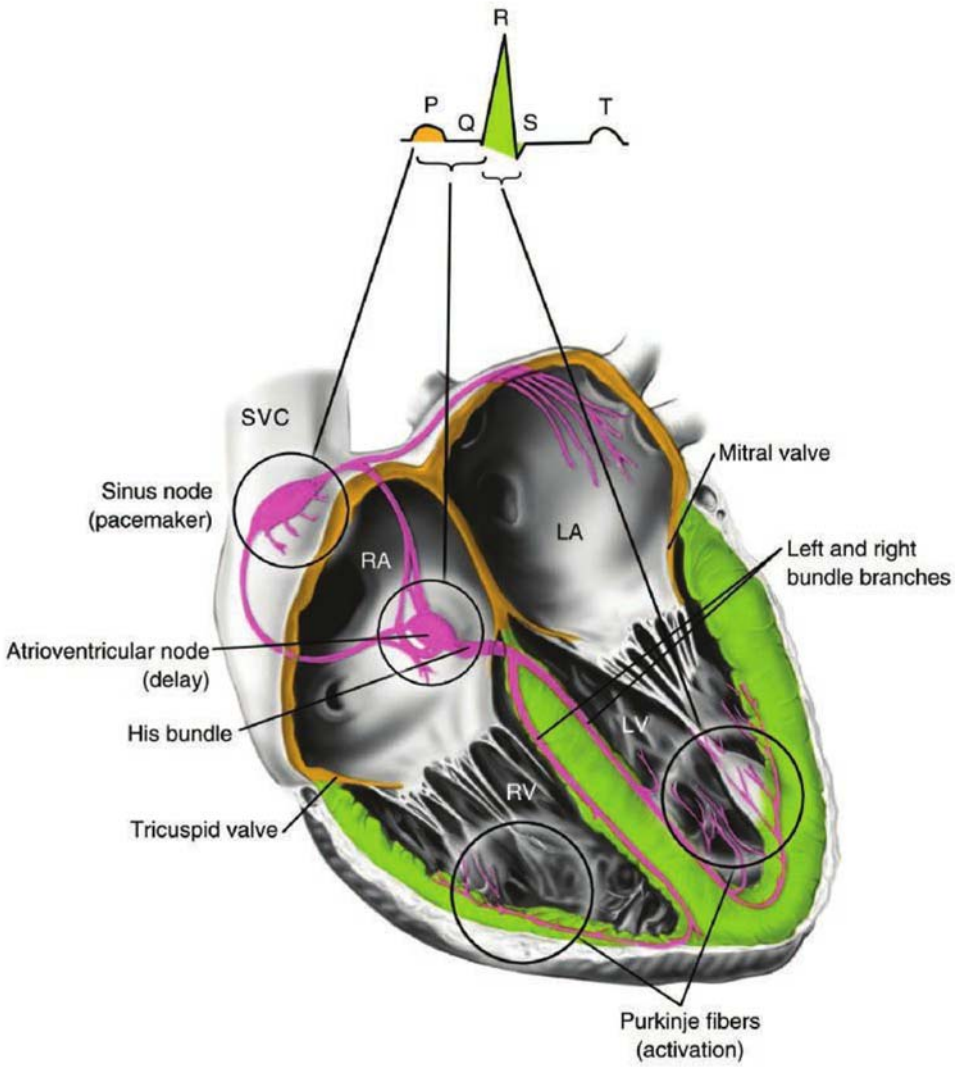


FIGURE 1-1 • Schematic of the electrocardiogram and cardiac conduction system. The pink color represents the electrical system, orange activation of the atria, and green activation of the ventricles. (See text for details.)

slow inward calcium current.[10] The impulses exit from the AV node into the ventricular specialized conduction system that has more rapid conduction properties, which consists of the His bundle, the left and right bundle branches with their subdivisions, and the peripheral network of Purkinje fibers, which terminate into ventricular myocardium. The left bundle branch is often described as having two primary functional divisions, the anterior and posterior fascicles. However, as early as 1906, Tawara[11] identified a third fascicle of the left bundle branch that enters into the left interventricular septum, an observation confirmed by others (**Figure 1-2**).[9] In a classic study by Durrer et al.[2] on excitation of the human heart, 3 endocardial areas were consistently shown to be excited synchronously 0 to 5 ms after the

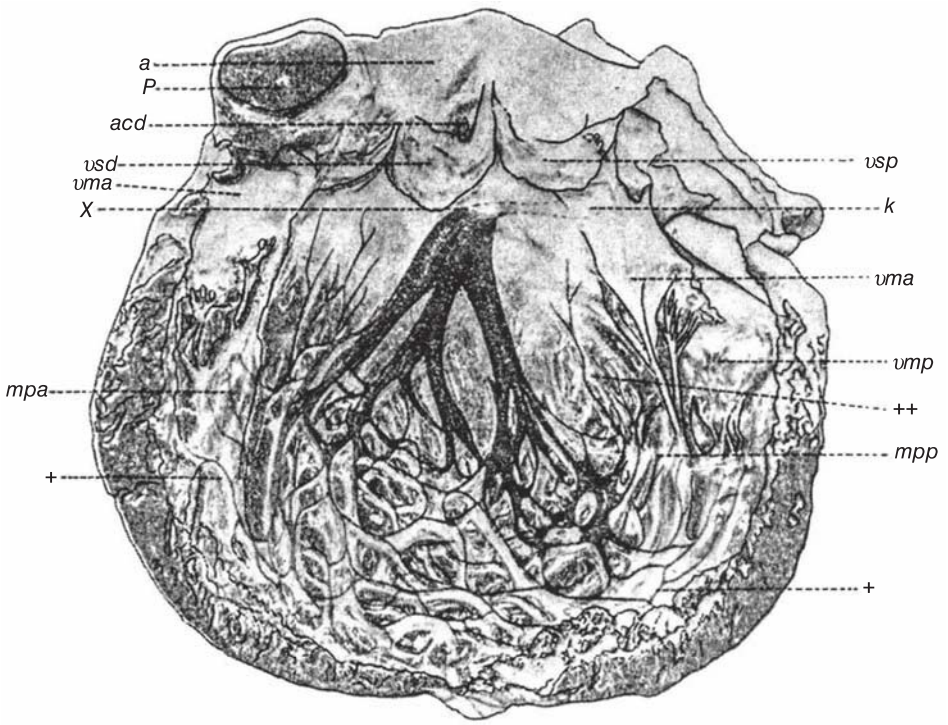


FIGURE 1-2 • Photomicrograph demonstrating the distribution of the left bundle branch in the human heart taken from the original publication of Tawara. (Reproduced with permission from Tawara S. *Das Reizleitungssystem des Saugetierherzens.* Jena, Germany: Fischer; 1906.)

beginning of the left ventricular cavity potential, giving credence to the concept of a functional trifascicular left bundle branch system.

In the absence of bundle branch block, ventricular activation is quite rapid, usually less than 100 ms, and is represented on the electrocardiogram by the QRS complex (Figure 1-1, green color). Examples of ventricular epicardial activation in normal hearts of patients undergoing surgery for ablation of the accessory pathway in the Wolff-Parkinson-White syndrome are demonstrated in **Figures 1-3** through **1-5**. These maps were computer generated during activation of 1 QRS complex, using a 56-electrode array sock. Figure 1-3 shows normal ventricular activation; right bundle branch block and left bundle branch block are noted in Figures 1-4 and 1-5, respectively.

ELECTROPHYSIOLOGIC-ELECTROCARDIOGRAPHIC CORRELATIONS

Electrophysiologic studies utilize several multipolar electrode catheters positioned at various locations in the heart (**Figure 1-6**). These catheters make it possible to record electrical potentials from within the heart and to stimulate various cardiac chambers in the diagnosis and management of cardiac arrhythmias. Whereas the electrocardiographer analyzes the surface electrocardiogram, the clinical electrophysiologist peers through this tracing to analyze its component parts (**Figure 1-7**). In essence, the more one knows about intracardiac events, the easier it is to analyze the surface electrocardiogram.

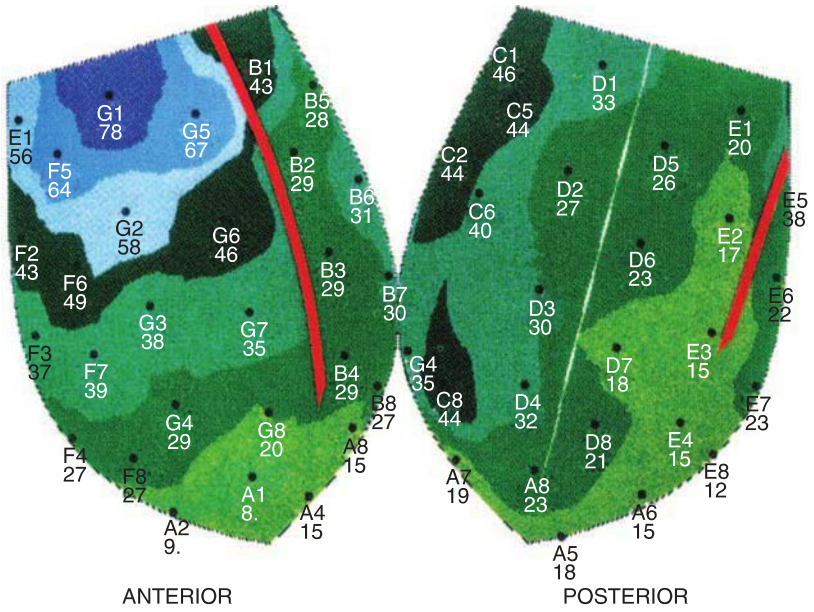


FIGURE 1-3 • Normal epicardial ventricular activation. The format for Figures 1-3, 1-4, and 1-5 is the same. At the time of surgery, a 56-electrode array sock was positioned on the epicardial surface of the heart; during 1 QRS complex, ventricular activation was obtained at all sites. Each site is noted by a letter and number that designate the electrode position and, underneath, the local activation time, using the initiation of the surface QRS complex as time 0. The left anterior descending (LAD) coronary artery is schematically represented in the anterior view and the posterior descending (PDA) coronary artery is represented similarly in the posterior view. These are approximate positions as noted during surgery. In this figure, note the rapid activation of the entire right and left ventricles. The last area activated is the base of the right ventricle, and activation time is complete within 78 ms. The right ventricle in the anterior view is to the left of the LAD and in the posterior view to the right of the PDA. This method of displaying the heart assumes that the posterior crux of the heart is divided and the ventricles are then laid out flat. The base of the heart with the atria removed is at the top of the figure and the apex (electrodes A1 through A8) is at the bottom. It is not that critical to analyze each point but rather to realize that the entire epicardial surface of the ventricle is activated relatively quickly.

PR Interval

Figure 1-8 demonstrates intracardiac electrophysiologic events.[13,14] The top tracing is surface ECG lead V_1; simultaneous intracardiac recordings are from 1 catheter in the high right atrium (HRA) and from another positioned across the tricuspid valve to record electrical signals coming from the AV junction near the His bundle electrogram (HBE). This was a quadripolar catheter, and the proximal 2 electrodes (HBE PROX) are closer to the atrial side of this juncture, recording a large atrial (A) depolarization along with a His bundle deflection (H) and a ventricular (V) electrogram that represents activation of the high ventricular septum. The distal bipolar electrode pair (HBE DIST) is situated more into the ventricle, and this enabled the recording of a right bundle branch (RB) potential.

The PA interval, a measure of atrial conduction, is taken from the onset of the surface P wave to local atrial activation in the His bundle electrogram. In our opinion, this interval is of minimal value, since it only records atrial activation through a part of the right atrium. More important measurements are the atrio-His (AH) interval, which is an approximation of AV nodal conduction time, and the His-ventricle (HV) interval, which reflects conduction through the His-Purkinje system. Measurement of the AH interval is taken from the first rapid deflection in the

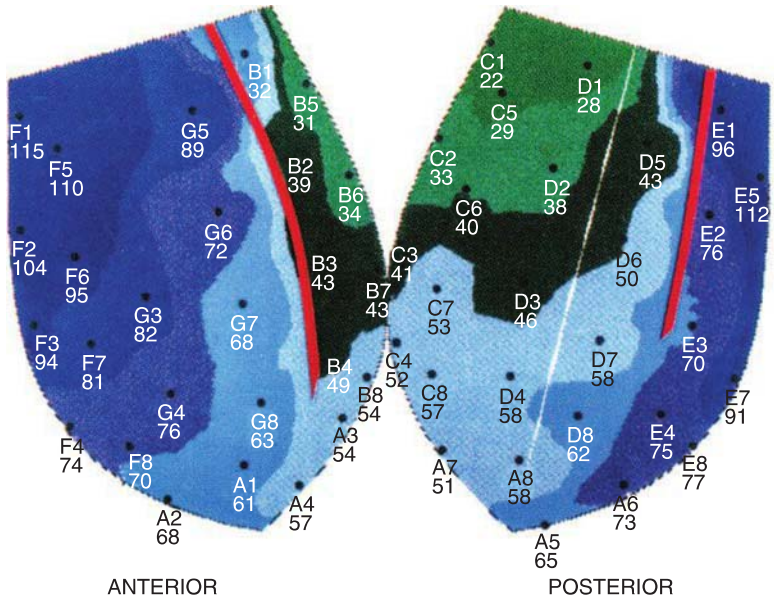


FIGURE 1-4 • Epicardial ventricular activation during right bundle branch block. (See Figure 1-3 for details of mapping methods.) Note that the left ventricle, represented by electrodes B through D, is activated in a normal fashion and relatively quickly. However, the right ventricle, represented by electrodes E through G, is activated late, as would be expected in right bundle branch block. Point F_1 is activated 115 ms after the initiation of the surface QRS complex. Compare this activation with that noted in Figure 1-3.

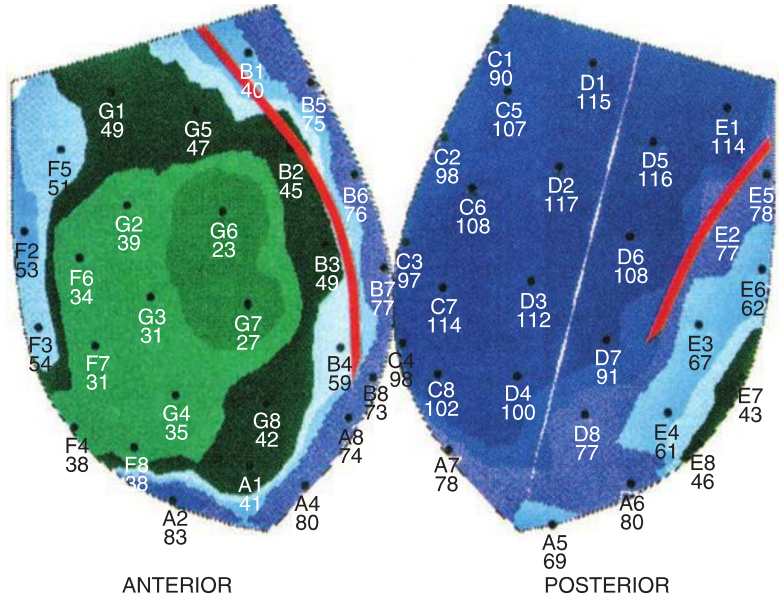


FIGURE 1-5 • Epicardial ventricular activation in left bundle branch block. (See Figure 1-3 for details of mapping.) In contrast to Figure 1-4, the earliest activated sites in this patient are on the right ventricle and the latter activated points on the left ventricle, as expected with left bundle branch block. Right ventricular points are primarily noted by G and E, which are left of the LAD in the anterior view and right of the PDA in the posterior view. Note that the last area to be activated is on the left ventricle at D2 (117 ms), in contrast to the last area activated during right bundle branch block, which is on the right ventricular epicardial surface (see Figure 1-4).

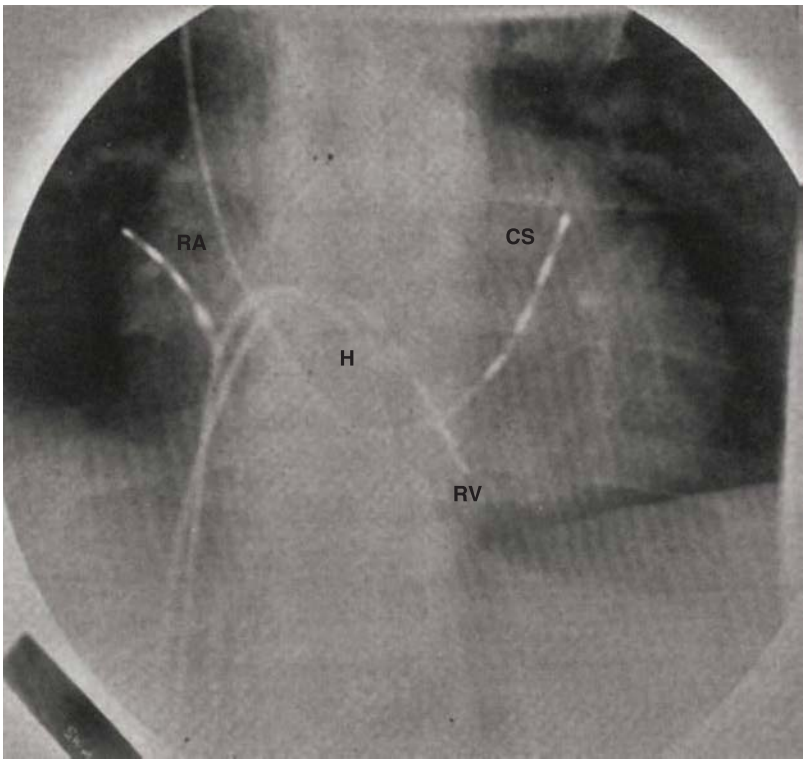


FIGURE 1-6 • Radiograph of endocardial catheter placement. (Key: RA = right atrium; CS = coronary sinus; RV = right ventricle; H = His bundle area.)

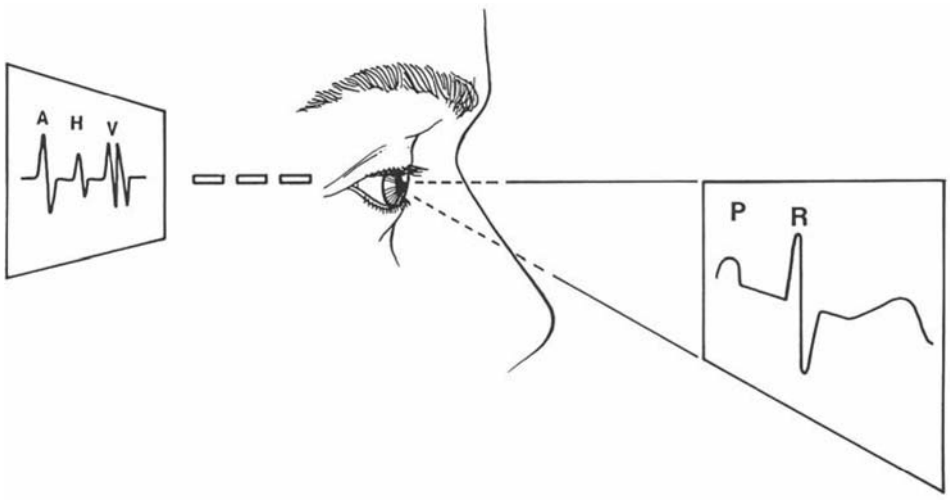


FIGURE 1-7 • A clinical electrophysiologist's view of the electrocardiogram.

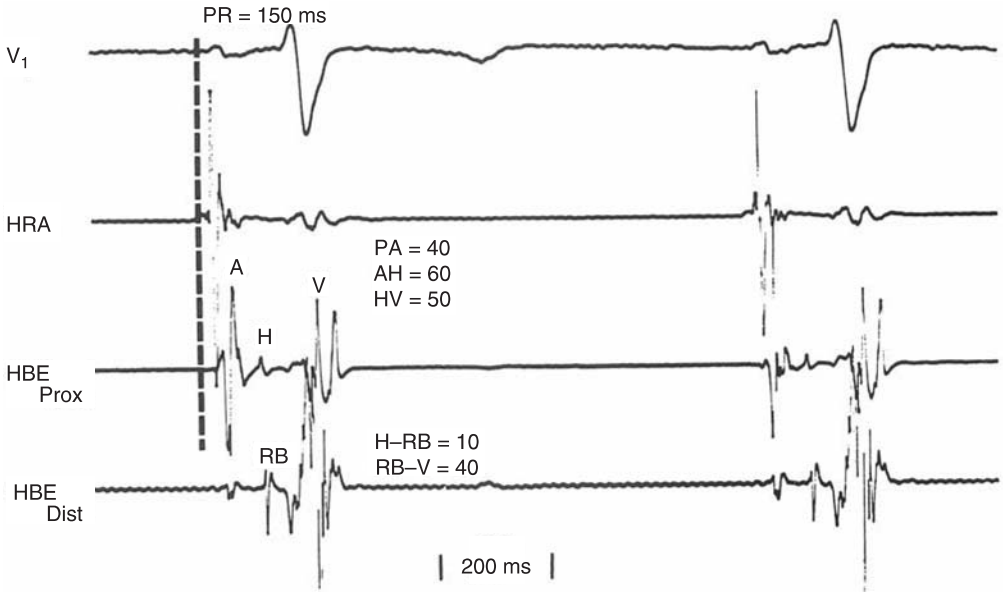


FIGURE 1-8 • Intracardiac electrophysiologic conduction intervals during the PR interval. (See text for details.) (Key: HRA = high right atrium; HBE (Prox) = proximal His bundle electrode pair; HBE (Dist) = distal His bundle electrode pair; A = atrial; H = His; V = ventricle; RB = right bundle.)

atrial electrogram on the His bundle tracing to the onset of the His bundle deflection.[14] The first rapid deflection represents local atrial activation in the vicinity of the AV node, but the earliest identifiable His bundle activity is chosen because the measurement of interest is conduction time through the AV node—not conduction time to a specific local area in the His bundle. In other words, when the His bundle activity is seen, conduction through the AV node is confirmed. In contrast, the HV interval is measured from the earliest identifiable His deflection to the earliest onset of ventricular activity recorded from either an intracardiac electrogram or any surface QRS complex. For this measurement the desired conduction time is from the earliest evidence of activity in the His bundle to any point confirming ventricular activity, regardless of the area from which this activity is generated. We consider an AH interval between 60 and 120 ms and an HV interval between 35 and 55 ms recorded during sinus rhythm as normal for our laboratories.

Abnormalities of AV conduction are discussed in more detail in Chapters 3 and 10, but some examples are offered here to illustrate the usefulness of intracardiac recordings in defining the location of conduction disturbances. In **Figure 1-9**, first-degree AV block (since no atrial activation is actually "blocked" from conducting to the ventricles, we prefer the term first-degree conduction delay) is demonstrated by a PR interval of 220 ms (0.22 s) and conduction of every P wave. The QRS duration is 90 ms, which is normal. The cause of first-degree conduction delay is noted in the His bundle electrogram. The AH interval measured 155 ms, which is markedly prolonged. However, in **Figure 1-10**, the prolonged PR interval in a patient with first-degree AV conduction delay and left bundle branch block is due to a severe conduction abnormality in the His-Purkinje system, as demonstrated by an HV interval of 165 ms. This latter situation represents a more critical cardiac conduction disturbance, as discussed in more detail in Chapter 10. A third example of first-degree AV conduction delay is shown in **Figure 1-11**. This patient

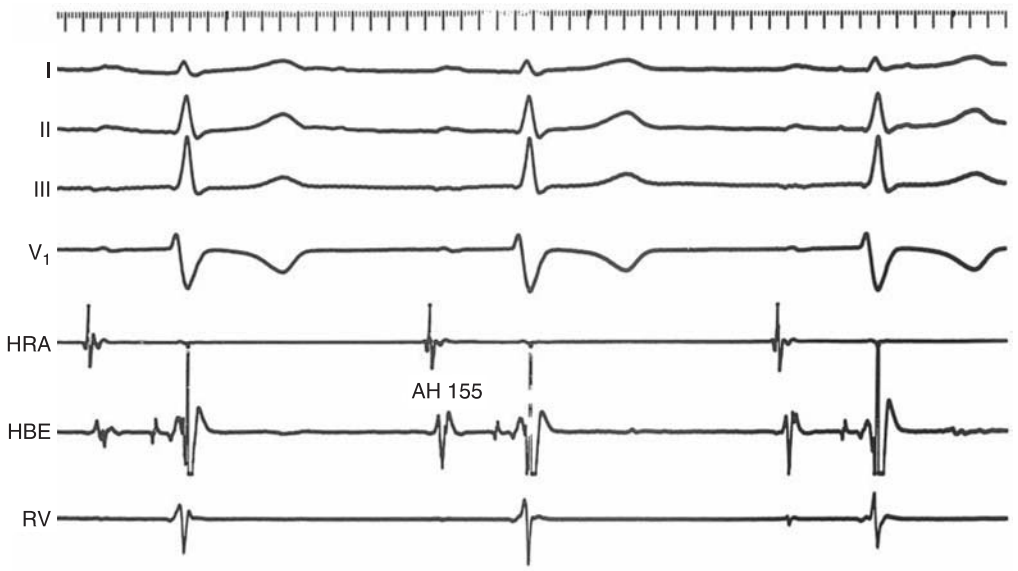


FIGURE 1-9 • First-degree AV block due to abnormal conduction in the AV node. (Key: RV = right ventricle. See Figure 1-8 for other abbreviations. Time lines: each major division = 50 ms.)

has a PR interval of 210 ms and left bundle branch block. However, the HV interval is only moderately prolonged to 65 ms, but a combination of all conduction times still yields an ECG pattern of first-degree AV conduction delay. Finally, first-degree AV conduction delay can be due to conduction abnormalities in both the AV node and His-Purkinje system (**Figure 1-12**).

The PR interval recorded during sinus rhythm usually demonstrates minimal variability unless marked changes in autonomic tone occur (see "Autonomic Nervous System Effects," later in chapter). Thus, the appearance of 2 distinctly different stable PR intervals during electrocardiographic recording

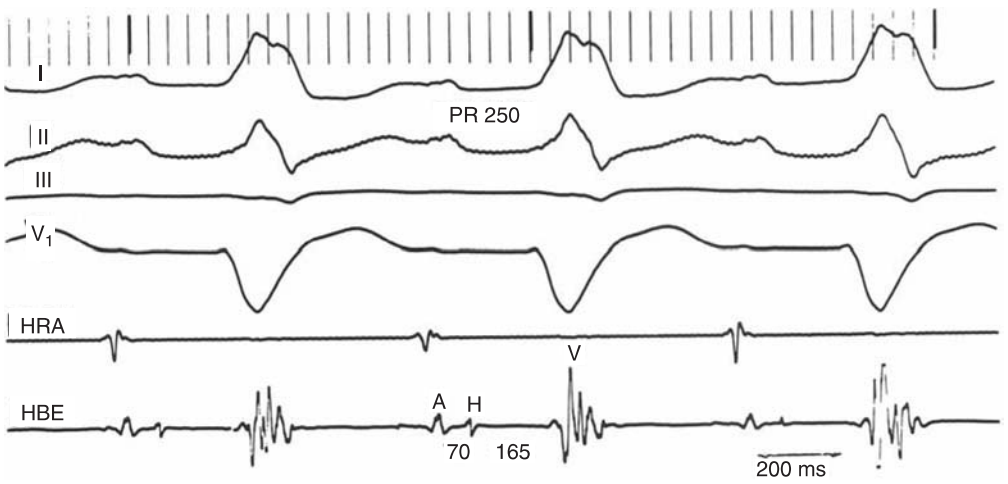


FIGURE 1-10 • First-degree AV block and left bundle branch block with a markedly prolonged HV interval. (See text for details.)

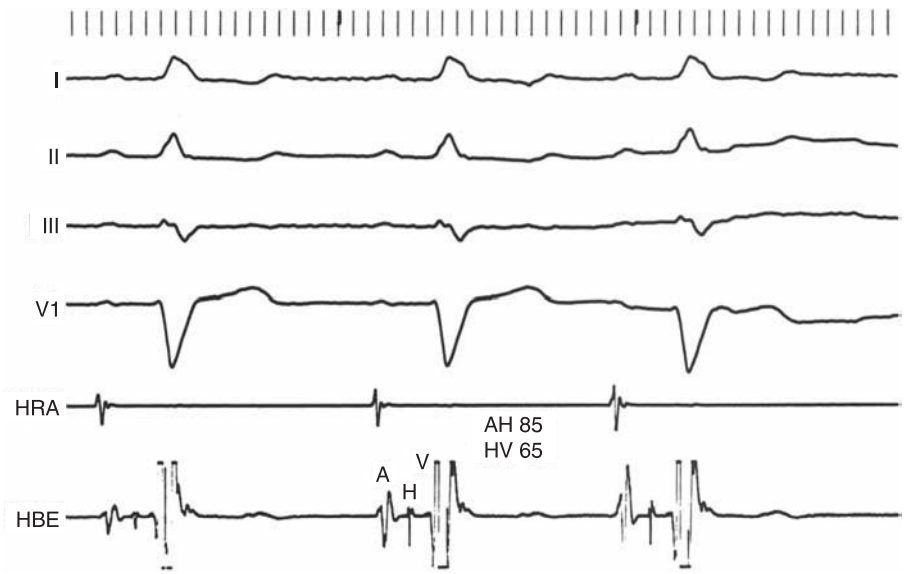


FIGURE 1-11 • First-degree AV block with left bundle branch block and minimal intracardiac conduction abnormalities.

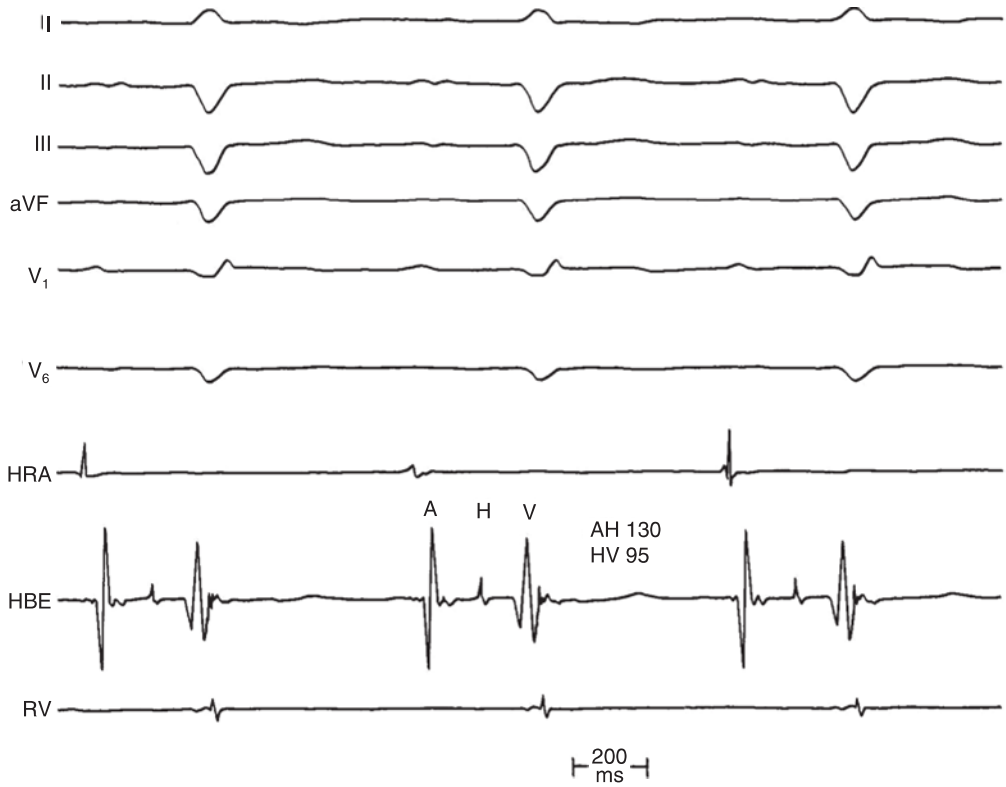
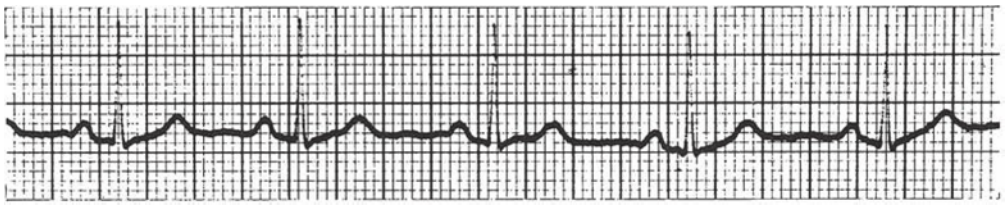
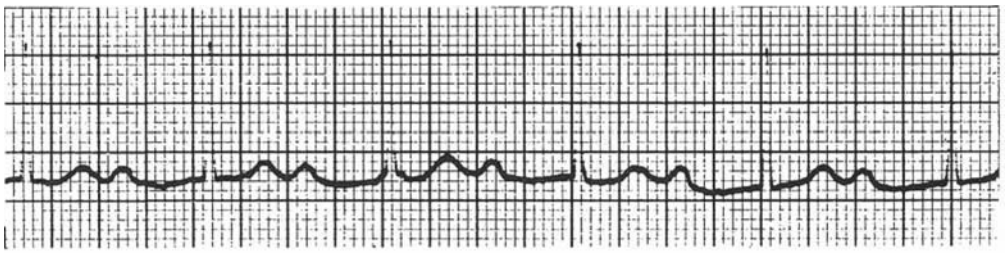


FIGURE 1-12 • First-degree AV block with combined AV nodal and His-Purkinje conduction abnormalities.



Panel A



Panel B

FIGURE 1-13 • Two separate and stable PR intervals. This patient had stable PR intervals that were either normal (A) or long (B). (Reproduced with permission from Kelley WN, ed. *Textbook of Internal Medicine.* Philadelphia, PA: Lippincott; 1989.)

strongly suggests the existence of 2 functionally separate anterograde conduction pathways, both presumably in the AV node (**Figure 1-13**). The PR interval in panel A is 180 ms and in panel B 400 ms. This patient had dual AV nodal physiology, as illustrated in **Figure 1-14**.[15-19] In this figure, the first 3 PR intervals represent AV nodal conduction over the slowly conducting pathway, and the

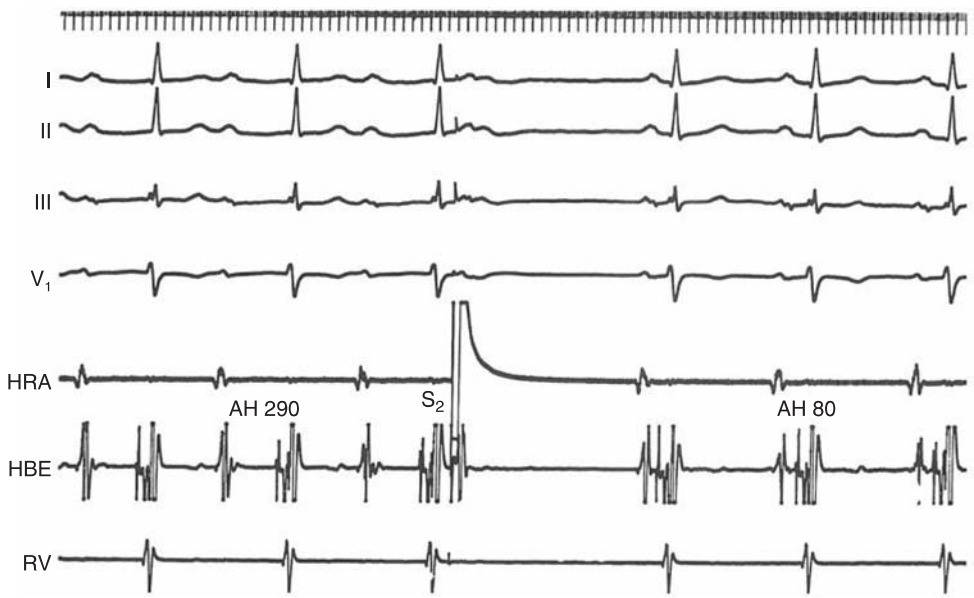


FIGURE 1-14 • Dual AV nodal physiology demonstrated at electrophysiologic study. (See text for details.) (Reproduced with permission from Prystowsky EN, Page RL. *Electrophysiology of the Sino-Atrial and Atrioventricular Nodes.* New York, NY: Alan R. Liss; 1988.)

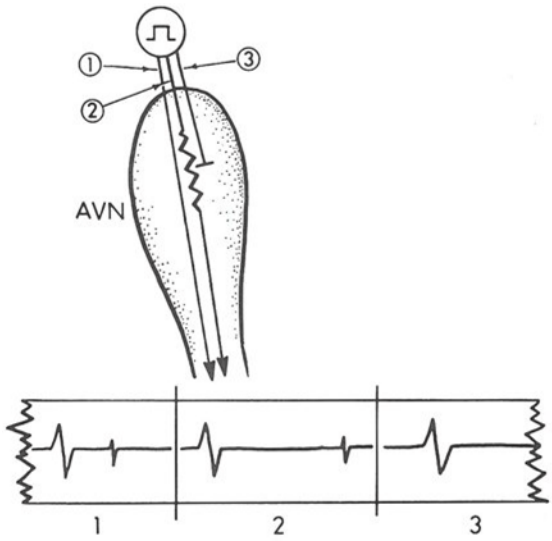


FIGURE 1-15 • Schematic of Wenckebach AV nodal block during incremental atrial pacing.

AH interval is 290 ms. A premature atrial stimulus (S_2) was introduced into the high right atrium and blocked in the AV node (no His depolarization after atrial premature complex). Atrioventricular nodal conduction resumed over the fast conducting pathway, as shown in the last 3 complexes on this tracing and demonstrated by a normal PR interval and an AH conduction time of 80 ms.

Atrioventricular nodal block

Incremental atrial pacing is used to evaluate AV nodal function in the electrophysiology laboratory. Atrial pacing is started at a rate slightly faster than the spontaneous sinus rate and is progressively increased until AV nodal block occurs. The AH interval increases with faster-paced rates as shown schematically in **Figure 1-15**. This is an example of 3:2 Wenckebach block. Complex 1 is the baseline interval and conducts with relatively minimal AV nodal delay; complex 2 shows a substantial increase in the AH interval; and complex 3 fails to conduct to the His bundle. Records from a patient are demonstrated in **Figure 1-16**. Note in ECG lead II a progressive increase in the PR interval for the first 3 paced beats, followed by a P wave without conduction to the ventricle; conduction resumes with the last paced beat, and the PR interval is shorter than the PR interval of the last conducted beat prior to block. The first PR increment is the largest of the sequence (30 vs 10 ms), consistent with Wenckebach behavior. This pattern of conduction abnormality is referred to as *Wenckebach, or Mobitz type I second-degree, AV block*. The changes in PR interval in this example occur within the AV node, as demonstrated by the progressive increase in the AH interval from 70 to 110 ms without change in the HV interval. Note that in the fourth paced beat the atrial depolarization in the HBE tracing is not followed by a His bundle deflection, confirming block within the AV node. Conduction resumes with an AH interval of 70 ms.

In very unusual instances it is difficult to detect a measurable increase in the PR interval prior to block. An example of this is noted in **Figure 1-17**. This patient had spontaneous episodes while awake of nonconducted P waves with minimal or no prior PR prolongation. Figure 1-17A was recorded upon electrophysiologic study at a routine ECG paper speed of 25 mm/s. There is only

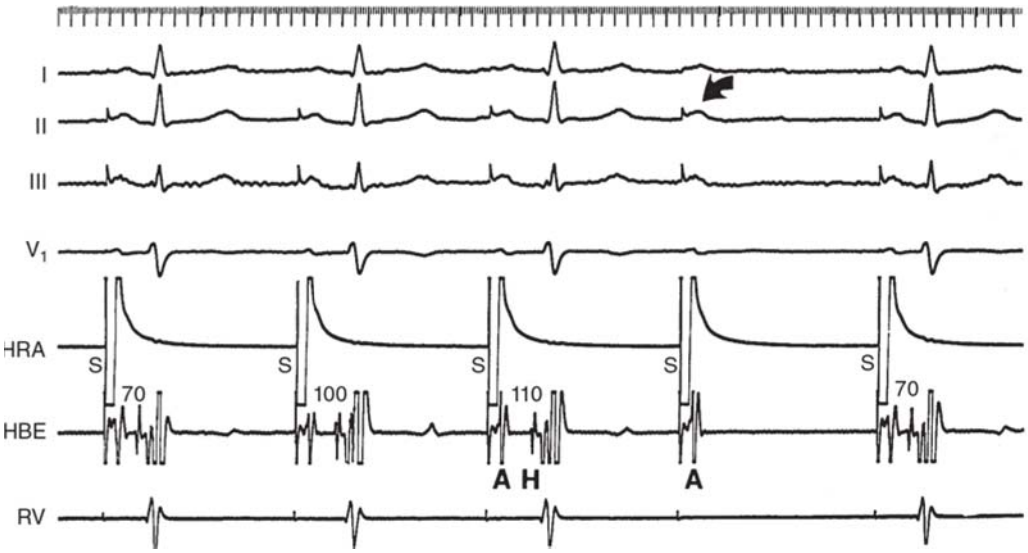
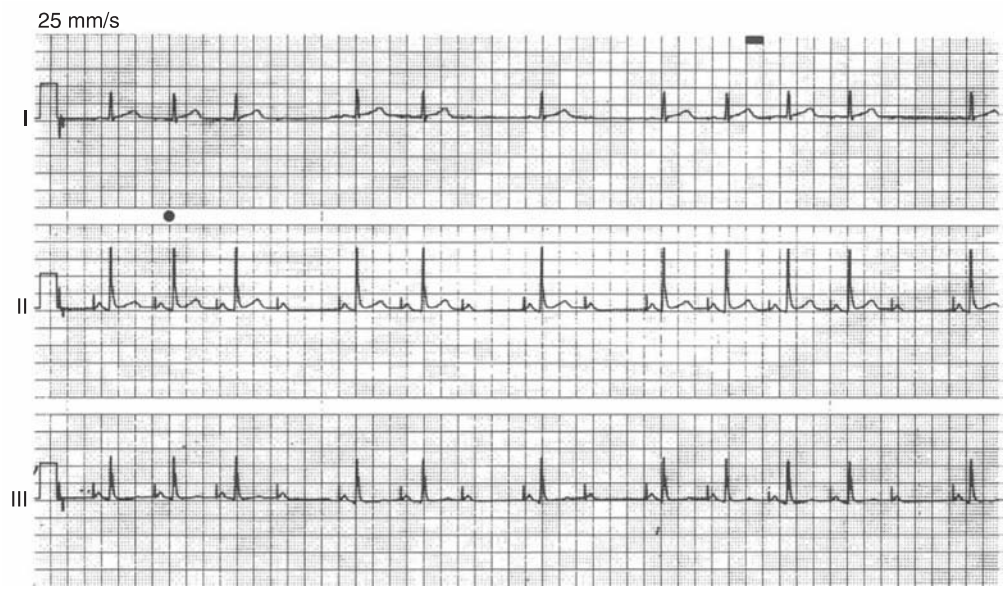


FIGURE 1-16 • Wenckebach AV nodal block. (See text for details.) Arrow points to nonconducted P wave.

a minimal change in the PR interval measurable in this example. Analysis of the intracardiac electrograms reveals conduction block in the AV node, with a mere 10 to 15 ms increase in the AH interval prior to the nonconducted P wave (Figure 1-17B). This recording was done at a 100-mm/s paper speed, where more precise measurements can be obtained. The normal



Panel A

FIGURE 1-17 • Wenckebach AV nodal block with minimal PR prolongation prior to block. (A) Wenckebach block during routine ECG recording. (B) Wenckebach AV nodal block demonstrated at electrophysiologic study. (See text for details.)

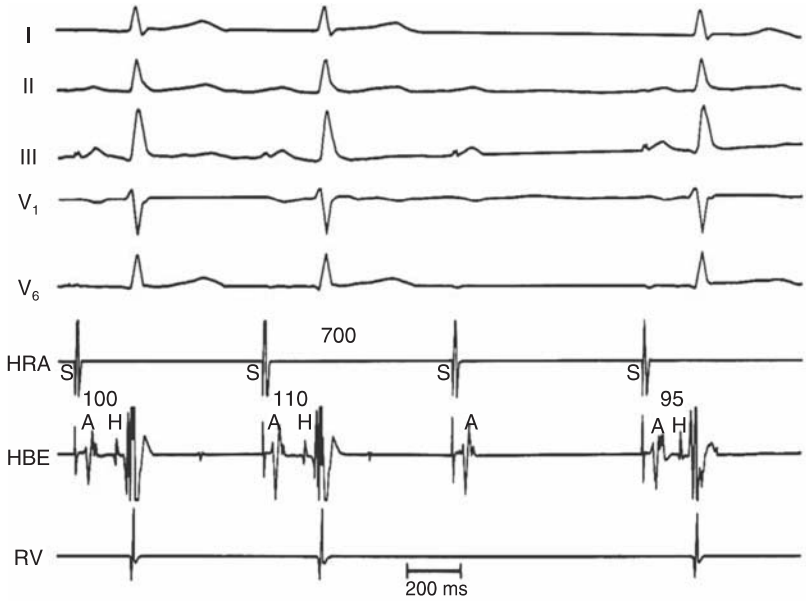
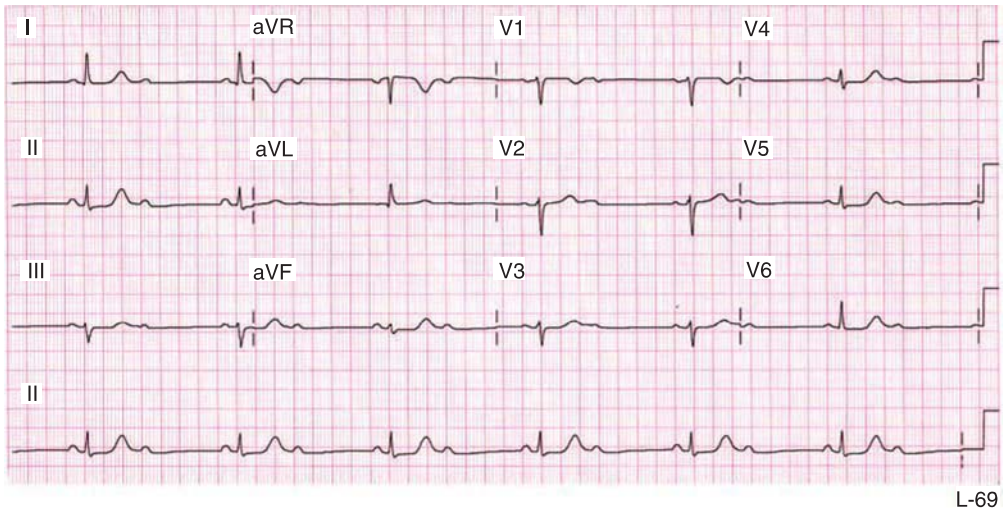


FIGURE 1-17 • (*Continued*)

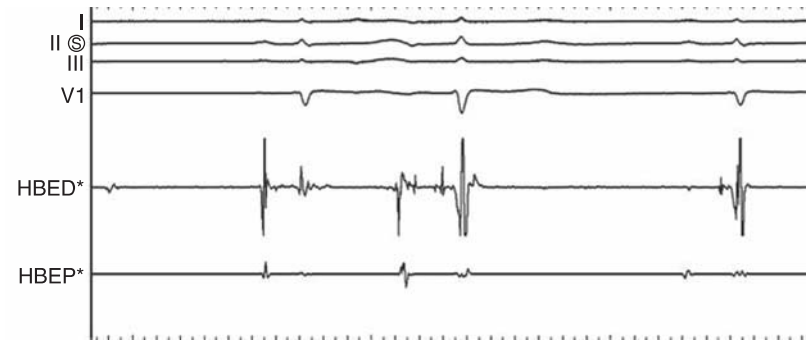
QRS complex strongly suggests that block will be in the AV node or His bundle, not below the His bundle. However, one should suspect block within the His bundle in cases of Wenckebach or 2:1 block with baseline normal PR and QRS intervals with minimal changes in PR interval before block (**Figure 1-18A**). Note in this example of 2:1 block the normal PR and QRS intervals on the conducted complexes, and the intracardiac electrograms showing a split His potential with block after the first His deflection, or intraHis block (**Figure 1-18B,C**).

On occasion, a Wenckebach conduction pattern can occur in the His-Purkinje system (**Figure 1-19**). At electrophysiologic study during incremental atrial pacing, there was a progressive increase in the PR interval, as noted in ECG lead II. Analysis of conduction times in the His bundle electrogram shows a progressive increase in the HV interval from 130 to 165 ms prior to a nonconducted P wave. The AH interval remained constant. Note that the fifth paced atrial complex demonstrates activation of the His bundle but no conduction beyond this point. Thus, conduction block is infra-His, which represents a severe form of conduction disturbance. Note that the patient has a right bundle branch block pattern. Even with a bundle branch block pattern, a Wenckebach conduction sequence most commonly implies conduction delay and block in the AV node.

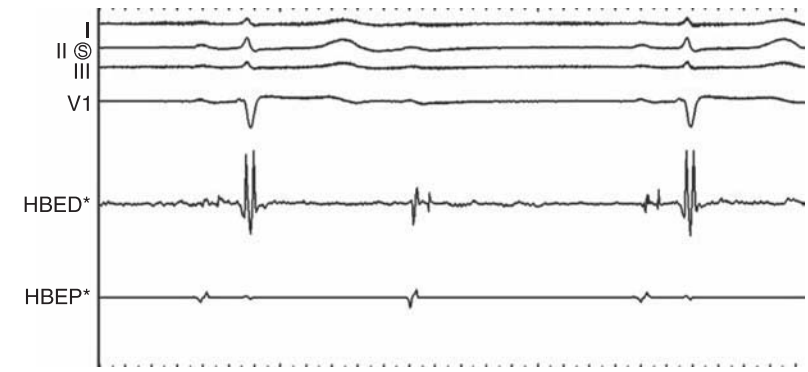
Testing of the AV nodal conduction system with incremental atrial pacing in the electrophysiology laboratory yields a wide range of atrial paced cycle lengths at which Wenckebach AV nodal block occurs. In the resting state during electrophysiologic testing, we consider AV nodal conduction to be normal if 1:1 conduction is present to a right atrial paced cycle length of less than 505 ms.[20] Of particular interest is that even though Wenckebach block may occur at a wide range of cycle lengths, the AV nodal function curve is physiologically similar for all patients. This is demonstrated in **Figure 1-20**, which shows the lengthening of the AH interval, noted on the ordinate,



Panel A



Panel B



Panel C

FIGURE 1-18 • (A) ECG with 2:1 AV block with normal PR and QRS intervals. (B) Simultaneous tracings of ECG leads 1-3 and V1, and intracardiac electrograms from the distal (HBED) and proximal (HBEP) His bundle areas from same patient. The second complex is premature and shows a split His after the atrial electrogram. (C) Block would always occur after the first His electrogram as seen in the middle atrial complex without conduction to the second His electrogram, or intraHis block. (See text for more details.)

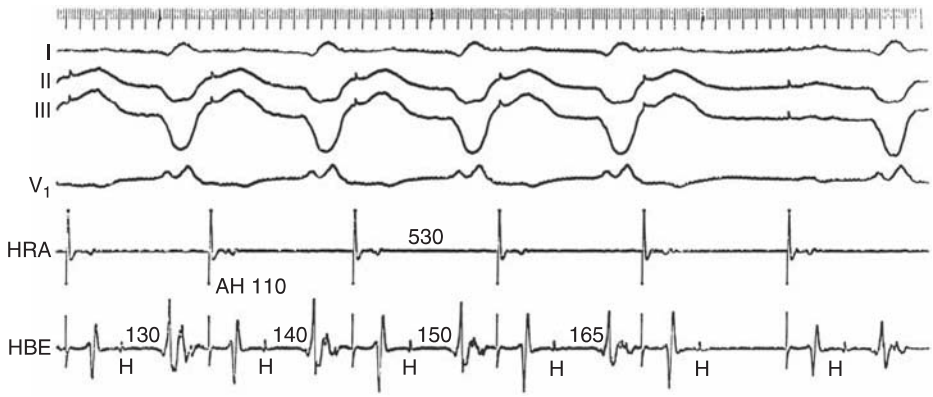


FIGURE 1-19 • Wenckebach block conduction pattern with infra-His block. (See text for details.)

as a function of shortening of the atrial pacing cycle length (increased heart rate), noted on the abscissa.[21] Each line represents a group of patients with Wenckebach block occurring at different cycle lengths. For example, the curve on the far right includes patients in whom Wenckebach block occurs at cycle lengths greater than 500 ms, whereas the curve on the far left represents patients in whom Wenckebach occurs at paced cycle lengths less than 300 ms. Each curve is divided into separate segments, with the most distal segment represented by *A*. Analysis of these data showed that the curves were progressively shifted to the left as shorter atrial paced cycle lengths were required to yield Wenckebach block, but the slopes of each part of the curves were similar for all curves. In essence, only minimal to moderate prolongation of the AH interval occurs until the Wenckebach cycle length is approached. As a corollary, drugs that demonstrate negative dromotropic effects,

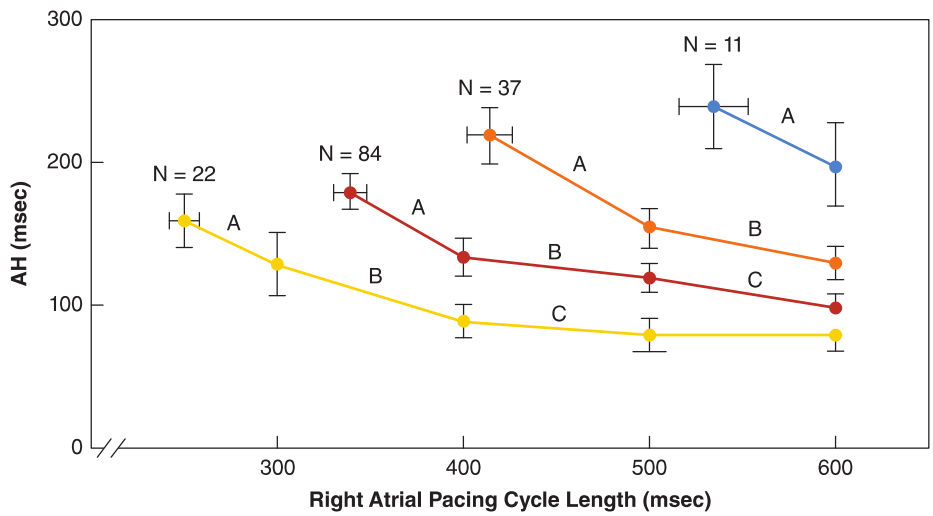


FIGURE 1-20 • Family of AV nodal function curves obtained during electrophysiologic testing. From top to bottom the curves represent patients who had shortest 1:1 atrial pacing cycle length <600 ms but ≥500 ms; <500 ms but ≥400 ms; <400 ms but ≥300 ms; and <300 ms. (Reproduced with permission from Jackman WM, Prystowsky EN, Naccarelli GV, et al. Reevaluation of enhanced atrioventricular nodal conduction: evidence to suggest a continuum of normal atrioventricular nodal physiology. *Circulation*. 1983;67:441.)

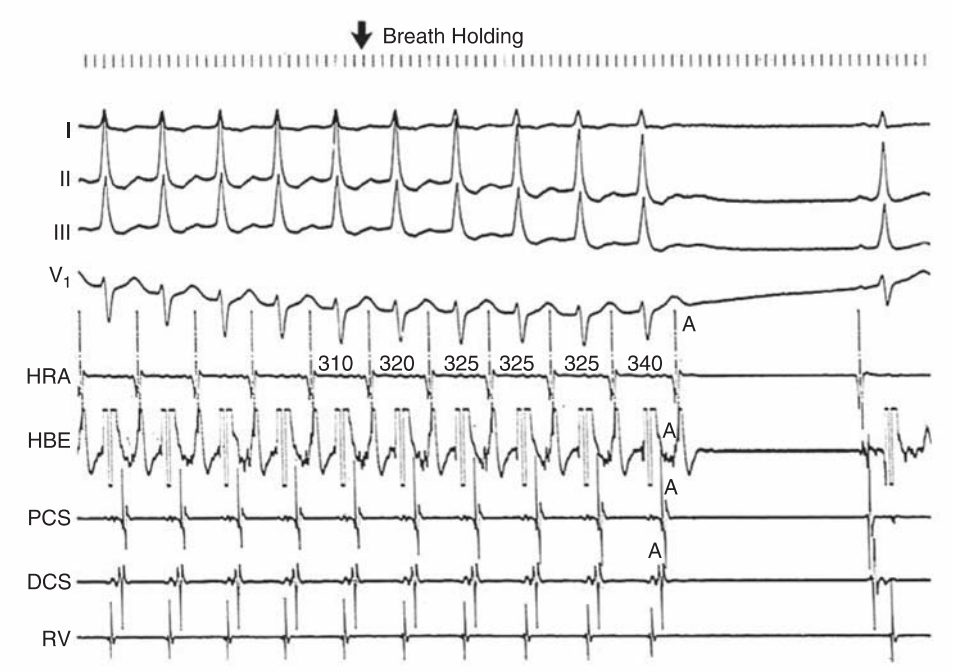


FIGURE 1-21 • Effect of increased vagal tone with breath holding to terminate atrioventricular re-entry with retrograde conduction over a left free wall accessory pathway. Note tachycardia terminates with an atrial electrogram (A) without conduction over the AV node. Simultaneous tracings are ECG leads 1-3 and V1; high right atrium (HRA); HBE; proximal (PCS) and distal (DCS) coronary sinus; and right ventricle (RV).

that is, depress conduction, on the AV node—such as verapamil or beta-adrenergic blockers—minimally affect condition on the flat portion of these curves but have a marked effect on the stressed portion (part *A*) of the curve. Since AV re-entry and AV node re-entry conduct near the Wenckebach point during tachycardia, it is not surprising that a brief increase in vagal tone with a maneuver such as breath holding (**Figure 1-21**) can terminate the arrhythmia.

RETROGRADE CONDUCTION

Incremental ventricular pacing and introduction of premature ventricular extrastimuli are used to evaluate retrograde conduction during electrophysiologic testing. **Figure 1-22** shows a schematic of possible paths of ventriculoatrial (VA) conduction. Eccentric retrograde atrial activation, that is, VA conduction during ventricular pacing or tachycardia that occurs earliest in the right or left atria typically reflects conduction over a right- or left-sided accessory pathway (AP), respectively, whereas concentric VA conduction can be over an AP in the septum or over the normal VA conduction system (His-Purkinje system and AV node, VACS). Incremental ventricular pacing typically generates VA curves seen in **Figure 1-23**. Conduction over an AP normally has minimal conduction delay but may show some delay at fast rates. However, many patients with conduction over the normal VACS show only 30 to 40 ms delay in this curve, and thus incremental pacing is not the best technique to differentiate concentric retrograde conduction over an AP from the normal VACS.