CARDIAC ARRHYTHMIAS

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With 10 Contributors

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Preface

In the mid 1960's, Andrew G. Wallace established the Cardiac Care Unit at Duke University Medical Center and began to do basic research in electrophysiology in Will Sealey's laboratory. These activities have formed the basis for the training of all of the contributors to this book and for many other cardiologists and cardiovascular surgeons who have had a continuing major interest in cardiac arrhythmias. Andy's first Fellows were Rick Schaal, currently a cardiac electrophysiologist at Ohio State University, and Doug Zipes, a cardiac electrophysiologist at the University of Indiana. Others who have subsequently trained at Duke and are continuing to pursue the study of cardiac arrhythmias are Menashe Waxman, at the University of Toronto; Barry Ramo, at the New Mexico Heart Institute and the University of New Mexico; John Boineau, at the Medical College of Georgia; Marcel Gilbert, at the Cardiology Institute of Laval in Quebec; Andrew Tonkin, at Flinders Medical Center in Adelaide, Australia; Hugh Miller, at the Royal Infirmary of Edinburgh; Ronald Campbell, at the University of Newcastle upon Tyne; Eric Prystowsky, at the University of Indiana; Robert Svenson, at the Heineman Research Institute in Charlotte, North Carolina; George Klein, at the University of Western Ontario in London; Warren Smith, at Green Lane Hospital in Aukland, New Zealand; Charles Kerr, at the University of British Columbia; Dwight Davis, at Pennsylvania State University at Hershey; Steve Hammill, at the Mayo Clinic; Mike Reiter, at the University of Colorado; Tom Guarnieri, at Johns Hopkins; Richard Sterba, at the Cleveland Clinic; Dave Benditt, James Dorsey, Woody Benson, and Robert Anderson, at the University of Minnesota; and John Gallagher, Ed Pritchett, Gus Grant, Jim Cox, and Larry German, at Duke Medical Center. Others, such as Mike Rotman, Bob Waugh, and I, have remained active in the pursuit of understanding and the teaching of cardiac arrhythmias. The Duke program has been responsible for major advances in the surgical management of cardiac arrhythmias, particularly in the Wolff–Parkinson–White syndrome. The laboratories of John Gallagher, Ed Pritchett, David Shand, Ray Ideker, and Harold Strauss continue to train the next generation of investigators in the area of cardiac arrhythmias.

One of our group, Barry Ramo, had learned of the Pick and Langendorf method of conducting cardiac arrhythmia seminars while he was a House Officer at the University of Chicago. On Barry's recommendation, many of us attended one of the yearly courses at the Michael Reese Hospital in Chicago and further developed our interests in the teaching of cardiac arrhythmias. As we developed our own arrhythmia courses, workbooks of increasing sophistication were produced, forming the basis for the current text.

We had discussed a collaborative effort from our various locations throughout North America for some time. The Duke Medical School curriculum makes it possible for medical students to pursue diverse and individual activities for basic science credits during their third year. Malcolm Thaler took this opportunity to co-author a text in medical immunology and other medical tests. From his position as a medical editor at Churchill Livingstone, Mal stimulated us to organize our concepts of the teaching of cardiac arrhythmias into a text. He helped design the format of the book and rewrote much of the material into a more coh-

esive unit. We were able to complete the work just as Mal moved back into his medical internship.

Our goal was a book which would be very different from existing arrhythmia texts. It would be written primarily by and for noncardiac electrophysiologists. It would not attempt to cover all aspects of cardiac arrhythmias. It would emphasize a "How to" approach regarding the history, physical examination, manipulation of the autonomic nervous system, and use of ECG monitoring techniques. Barry Ramo's comments about the use of the patient's history reflect his years of experience using the patient history for the understanding of arrhythmias. Bob Waugh continues to investigate optimal ways of using the physical examination to teach medical students about cardiovascular physiology. Menashe Waxman and his colleagues at the University of Toronto have developed methods for fine-tuning the balance between sympathetic and parasympathetic aspects of the autonomic nervous system for the diagnosis and management of cardiac arrhythmias. The editors thought it was most important to include the extensive chapter on the use of the autonomic nervous system in a format that was somewhat different from that of the rest of the book because of its unique contribution to our understanding of arrhythmias. Bob Waugh and I have relied heavily on Wanda Bride and Marguerite English, the head nurse and assistant head nurse of our Cardiac Care Unit, for preparing the section on ECG monitoring.

The terminology regarding cardiac arrhythmias has been simplified as much as possible and practical classification systems have been presented. There are only a small number of major problems in cardiac arrhythmias and each is presented, using a common format, in Chapters 6 through 12. There are extensive illustrations that have been reproduced, where possible, at full size. Also, contrasting examples are presented in a single figure to provide the most dramatic example of a particularly important aspect of the understanding of arrhythmias.

The chapter on pharmacologic management is the result of Mike Rotman's years of experience in treating patients who have cardiac arrhythmias. We have been able to weave into Mike's experience the expertise of our Duke cardiac pharmacologist, Wayne Stargel. The chapter on arrhythmia management in the common settings of an acute myocardial infarct and the perioperative period represents the personal experiences of Drs. Ramo and Rotman.

We believe this book will be useful for anyone who has responsibility for patients with cardiac arrhythmias and has the goal of developing a practical understanding of the principles governing diagnosis and management.

I wish to acknowledge the work of Judy Berry, Gail McKinnis, Jo White, and Diana Visconti in preparing the manuscript, the stimulation of Rick Klausner in bringing Mal Thaler and me together to plan the project, the support of Bill Schmitt at Churchill Livingstone in guiding the manuscript through its various stages, the helpful review of the manuscript by Tom Hinohara, the medical illustrations of Francine Mehler, and the photography of Dave Huggett.

Galen S. Wagner

Contents

Techniques of Arrhythmia Diagnosis

1

The Physiology of Normal and Abnormal Rhythms

GALEN S. WAGNER, M.D. BARRY W. RAMO, M.D.

Cardiac cells are electrically polarized in the resting state with the inside of the cell negative with respect to the outside. Activation of these cells requires them to be "depolarized" or lose their negativity. This electrical activation initiates the mechanical action of shortening or contraction of the cardiac cell. The cell remains in the depolarized state for a period of time; during this time it cannot be further activated and is therefore termed "refractory." Following this refractory period, the cardiac cell "repolarizes" and is then capable of being depolarized once again.

Some cardiac cells can depolarize spontaneously and are referred to as pacemaker cells. Other cells (working myocardial cells) need an external source of electrical stimulation. The *action potential* is the "electrocardiogram" of a single cardiac cell and is obtained by putting a microelectrode inside that cell. Figure 1.1 shows both an action potential from a single cell and the surface ECG that is the sum of the action potentials from all of the cardiac cells. The action potential is divided into four phases. Phase 0 occurs when the cell is initially depolarized and the recording from inside the cell indicates a change from −90 to +20 mV. During phases 1 and 2 the cell remains depolarized. During phase 3 repolarization or recovery occurs. During repolarization the cell gradually resumes its resting or recovered state. The ECG correlation with the action potential is shown; the QRS complex corresponds to phase 0, the ST segment to phases 1 and 2, and the T wave to phase 3. During phases 1 and 2 the cell is absolutely refractory and no depolarization can occur. During phase 3 the cell is only relatively refractory. In this phase the cell can be depolarized further, but the action potential produced is smaller and conduction of electricity through the cell is slowed.

Phase 4 of the action potential is the resting period for myocardial cells. For pacemaker cells, a slow gradual depolarization occurs during phase 4 until the cell reaches a threshold potential, when phase 0 begins. It is this capability of spontaneous diastolic depolari-

zation during phase 4 that characterizes pacemaker or "automatic" cells within the heart (see Chapter 4).

Normal Rhythms

The normal cardiac rhythm is initiated by the spontaneous activation of cells within the sinus node located high in the right atrium. This event is silent on the surface ECG. The P wave begins only when the electrical impulse starts to spread through the right atrial myocardium. By the time approximately one half of the P wave has been inscribed and the impulse has just begun to advance through the left atrium, the atrioventricular (AV) node is entered. The latter half of the P wave is produced solely by left atrial activation (Figure 1.2).

In the normal heart the electrical impulse can exit from the atria only through the AV node. The conduction velocity within the AV node is extremely slow, thereby permitting optimal time for atrial contraction to contribute to ventricular filling.

Approximately 0.04 sec prior to the onset of the QRS complex, the impulse has already emerged from the AV node and has entered the His (or common) bundle. His bundle activation can be recorded through an intracavitary electrode positioned across the tricuspid valve. This "spike" is clearly separated from the subsequent activation of the ventricular muscle. This delay indicates that the electrical impulse does not enter the muscle either directly from the AV node or His bundle, but only after it passes through the proximal aspects of both right and left bundle branches. Ventricular contraction would be very inefficient if the sequence progressed from base to apex, because the outflow valves are located at the base. Optimal efficiency of ventricular contraction requires that the electrical impulse spread rapidly along the endocardial surfaces of both ventricles and that the basal areas of the ventricles are the last to be activated. Despite the efficiency with which this elaborate conduction system provides for myocardial activation, its complexity leaves it vulnerable to the rhythm disturbance of AV block at several sites: (1) the AV node, (2) the

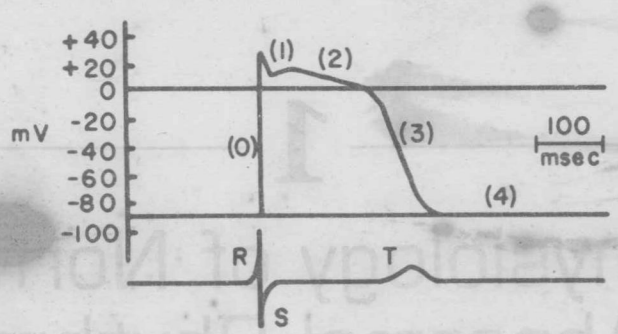


Figure 1.1.

The various phases of depolarization and repolarization of the cardiac cell in relation to the surface ECG are depicted. The surface ECG is the electrical sum of all the activity of the heart's depolarizing and repolarizing cells. During phases 1 and 2 the cell is refractory to further stimulation (absolute refractory period), and during phase 3 a greater than normal stimulus is required to elicit a second depolarization (relative refractory period). During phase 4 the cell is fully covered.

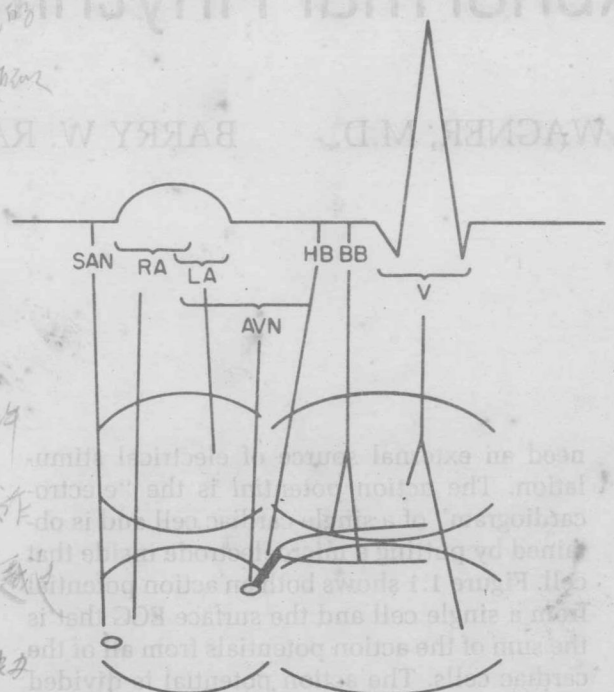
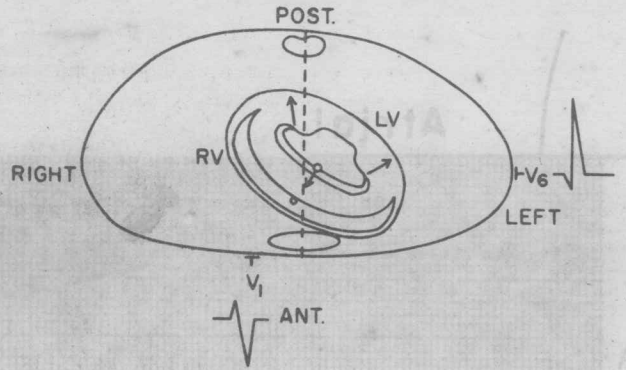


Figure 1.2.

The depolarization of the SA node, SAN, is "silent." During the time from the beginning of the P wave to the onset of the QRS complex several areas of the heart are activated: right atrium, RA; left atrium, LA; AV node, AVN; His (common) bundle, HB; and the bundle branches, BB. An electrical impulse enters the LA and the AVN almost simultaneously, but a much longer time is required to traverse the AVN. In the absence of a bundle branch block, activation of the ventricles, V, is relatively synchronous.

Figure 1.3.

The transverse plane of the body is shown at the level of the ventricles. The dashed line indicates the midline. The small circles on either side of the interventricular septum indicate the right and left bundle branches. The right bundle branch remains a single structure in its course toward the right ventricular apex, while the left bundle divides into septal, anterior, and posterior fascicles. Normal activation therefore results in initial anterior forces in lead V_1 (small R wave) and initial negative forces in lead V_6 (small Q wave).



His bundle, or (3) the proximal aspects of both bundle branches.

The AV node has a posterior location within the AV connective tissue. Purkinje fibers exit from the node forming the His bundle. As the His bundle moves anteriorly, some of the fibers exit to form the posterior fascicle of the left bundle, which travels toward the posterior papillary muscle of the mitral valve. Some of the remaining fibers on the left side continue anteriorly to form the anterior fascicle of the left bundle, which extends toward the anterior papillary muscle of the mitral valve, while others move directly into the midportion of the left side of the interventricular septum (Figure 1.3). It is these latter fibers that initiate ventricular activation and produce the left to right depolarization of the septum, which causes an initial positive (R) wave in lead V_1 and an initial negative (Q) wave in lead V_6.

The right bundle separates from the fibers of the anterior aspect of the left bundle at the top of the interventricular septum. It proceeds as an intact bundle toward the apex of the right ventricle. Normally, the thinner-walled right ventricle is completely activated about halfway through the QRS complex, exerting minimal, if any, effect on the shape of this complex. Just as the latter half of the P wave is produced solely by left atrial activation, so the latter half of the QRS complex is produced solely by left ventricular activation.

Abnormal Rhythms

An abnormality in the electrical activation of the heart can result in (1) a change in the appearance of the P wave or QRS complex, (2) an altered relationship between the P wave and QRS complex, or (3) a bradycardia or tachycardia. These abnormalities can be categorized as problems of either impulse formation or impulse conduction.

Problems of Impulse Formation

Although impulse formation could occur in the sinus node in the absence of the autonomic nervous system, the rate of impulse formation is continually modulated by the balance between sympathetic and parasympathetic tone. Impulse formation may therefore be slowed if there is either an intrinsic abnormality of the sinus node or an increase in parasympathetic tone. Increased impulse formation in the sinus node is usually not due to an intrinsic abnormality, but rather to an increase in sympathetic tone.

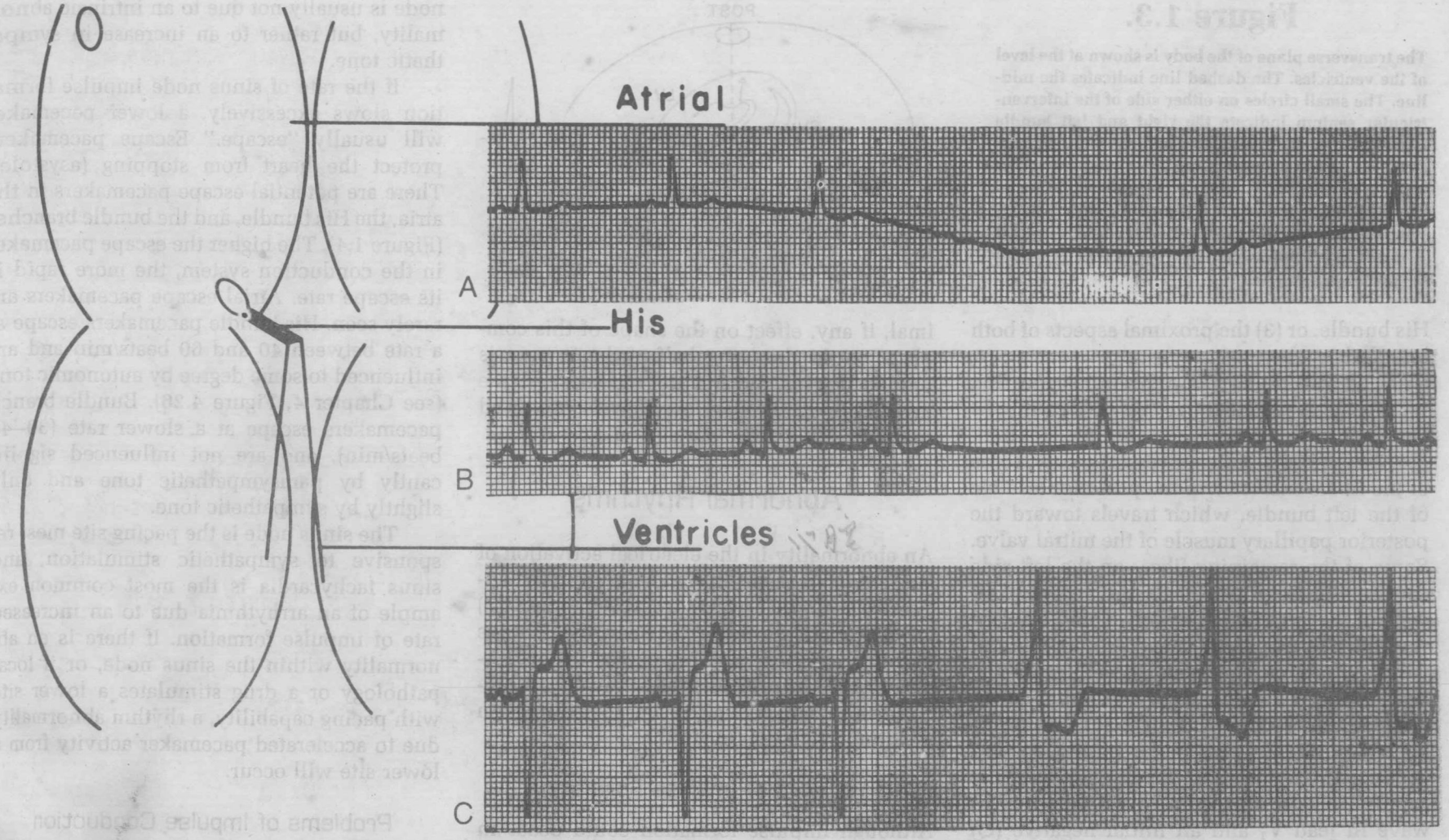
If the rate of sinus node impulse formation slows excessively, a lower pacemaker will usually "escape." Escape pacemakers protect the heart from stopping (asystole). There are potential escape pacemakers in the atria, the His bundle, and the bundle branches (Figure 1.4). The higher the escape pacemaker in the conduction system, the more rapid is its escape rate. Atrial escape pacemakers are rarely seen. His bundle pacemakers escape at a rate between 40 and 60 beats/min and are influenced to some degree by autonomic tone (see Chapter 4, Figure 4.26). Bundle branch pacemakers escape at a slower rate (30–40 beats/min), and are not influenced significantly by parasympathetic tone and only slightly by sympathetic tone.

The sinus node is the pacing site most responsive to sympathetic stimulation, and sinus tachycardia is the most common example of an arrhythmia due to an increased rate of impulse formation. If there is an abnormality within the sinus node, or if local pathology or a drug stimulates a lower site with pacing capability, a rhythm abnormality due to accelerated pacemaker activity from a lower site will occur.

Problems of Impulse Conduction

An abnormality of impulse conduction may take one of two forms: (1) failure of an impulse to move from a proximal to a more distal area, or (2) "reentry" of an impulse back into a previously activated area.

Failure of Impulse Conduction. Failure of impulse conduction can occur between a



pacemaker, such as the sinus node, and the surrounding myocardium (exit block) or between the atria and the ventricles (AV block). As noted in the discussion of normal rhythms, both the AV node and the His bundle and its branches lie between the atrial myocardium and the ventricular myocardium. Block within the His bundle is very uncommon, and therefore AV block usually occurs either in the AV node or in both the left and right bundle branches.

AV block is characterized by two features; degree and type. The amount of AV block is indicated by the term "degree." First-degree indicates slow conduction of all impulses resulting in a prolonged (>0.2 sec) P-R interval.

Figure 1.4.

The three rhythm strips show atrial pauses that are terminated by escape beats located in the atrium (A), indicated by the small inverted P wave before the normal-appearing QRS terminating the pause, the His bundle (B), indicated by the absence of atrial activity before the normal-appearing QRS terminating the pause, and from the ventricle (C), indicated by the wide QRS with no preceding atrial activity.

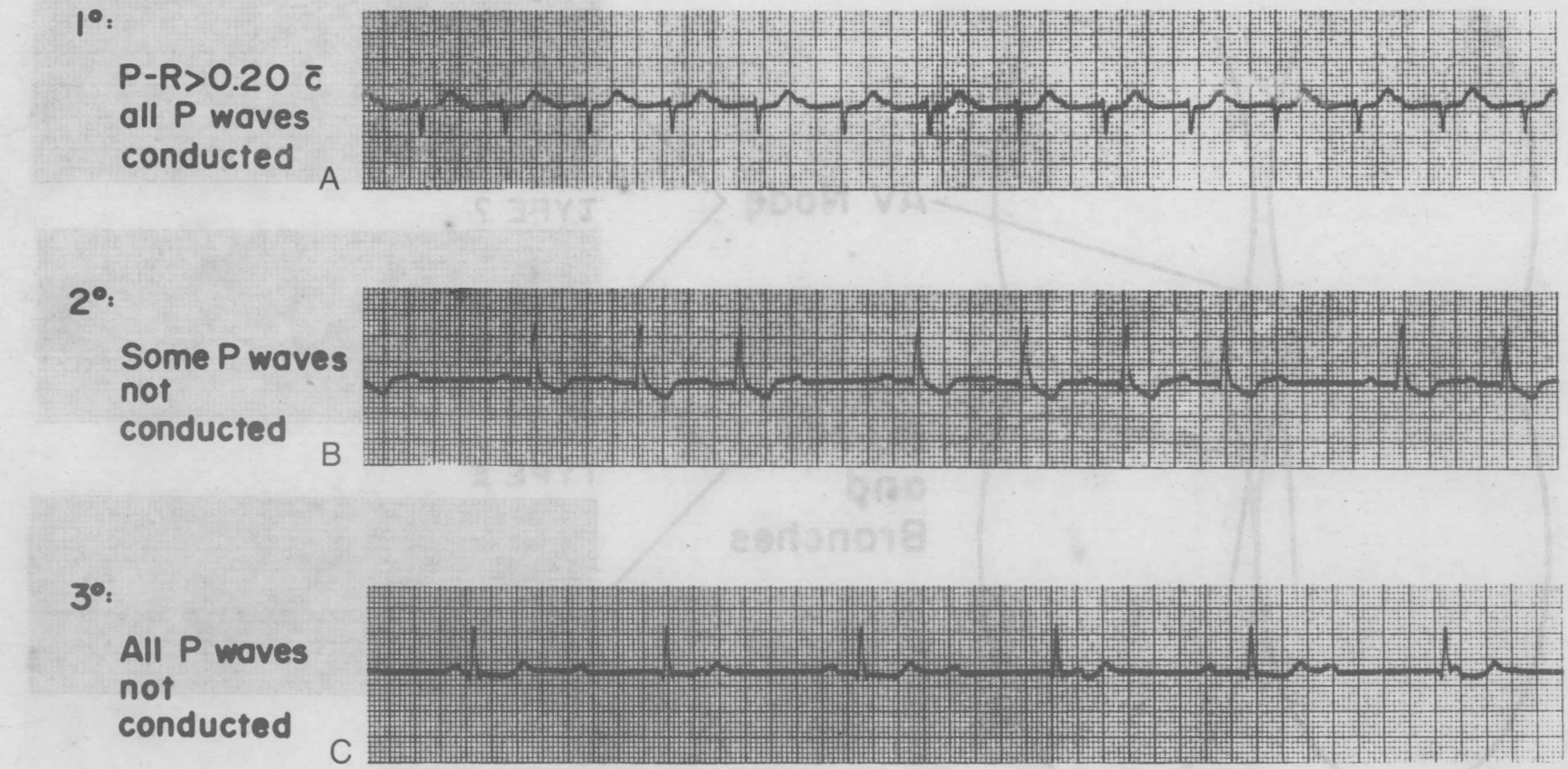


Figure 1.5.

The (A) rhythm strip shows first-degree AV block (all P waves conducted but with a greater than normal P-R interval). Second-degree AV block is shown in the (B) strip. The varying P-R and R-R intervals with a constant QRS morphology define AV *a*ssociation with conduction of at least some of the P waves. In the (C) rhythm strip the P-R intervals are again varying, but the R-R intervals are regular, defining AV *dis*sociation. Since some of the P waves should have been conducted (i.e., not occurring during the preceding T wave and greater than 0.2 secs in front of the following QRS), third-degree AV block is present.

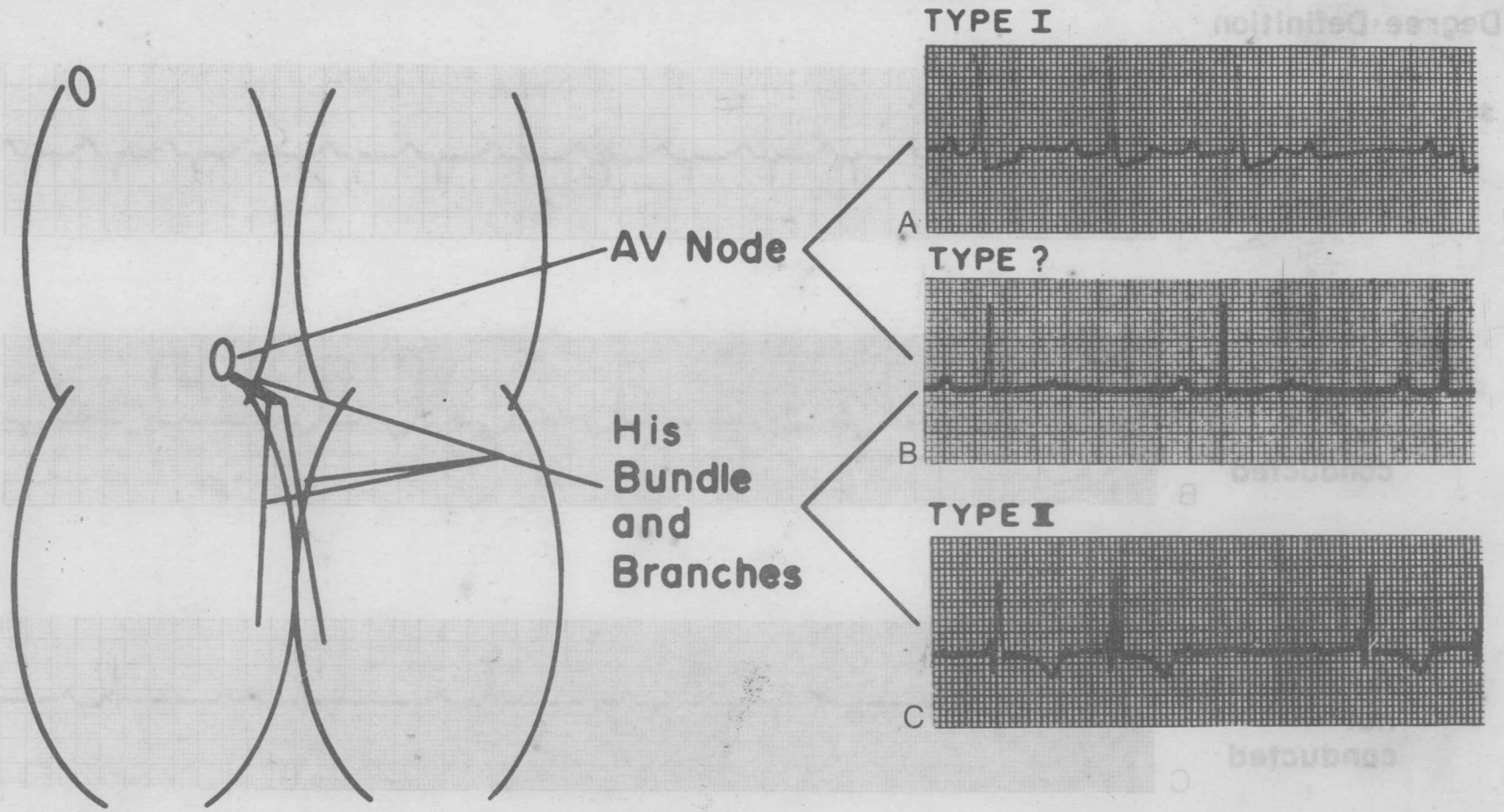


Figure 1.6.

(A) The AV node typically causes Type I block, defined by a varying P-R interval; (B) but it can also cause a constant P-R interval when the recovery times (from last conducted QRS to the next conducted P wave) are constant. (C) The His bundle and bundle branches typically cause Type II block, defined by a constant P-R interval despite a varying recovery time. The P-R interval after the pause is identical to the other P-R intervals in the strip. When both the R-P and P-R intervals are fixed (strip B) it is impossible to identify the Type of AV block, and either an intervention or observation of further rhythm strips is necessary.

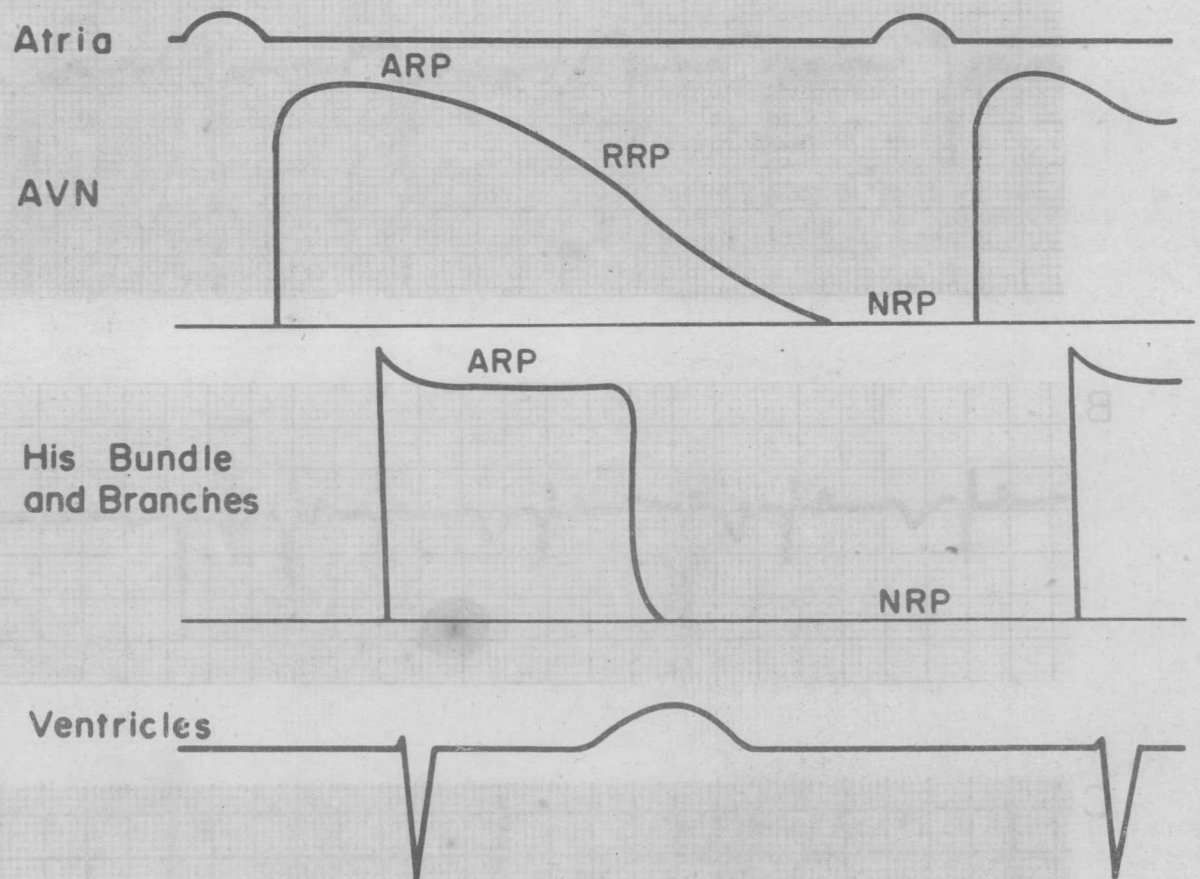


Figure 1.7.

There is an absolute refractory period, ARP, in both the AV node, AVN, and the His bundle and its branches. A relative refractory period, RRP, is essentially absent in the His bundle and bundle branches but constitutes a significant portion of the action potential in the AV node. Activation during the relative refractory period results in slowed conduction, producing the typical variation in the P-R interval that is characteristic of AV node conduction. NRP = non-refractory period.

Second-degree indicates conduction of only some impulses. Third-degree indicates no conduction at all (Figure 1.5).

The second feature of AV block is the type (Mobitz Type). The concept of "type" is used to help localize the site of the conduction disturbance (Figure 1.6). Type I block occurs in the AV node because the AV node can vary its conduction time producing measurable changes in the P-R interval. This variability is possible because of the long relative refractory period in the AV node (Figure 1.7). This relative refractory period, and thereby the AV nodal conduction time, varies with changes in autonomic tone: sympathetic tone speeds conduction and parasympathetic tone slows conduction. AV nodal conduction also depends on the length of the recovery period following the previous beat. For example, if an atrial premature beat is only slightly premature, it will be conducted with a normal P-R interval. If it is more premature, it will be conducted with first-degree AV block. If it is quite premature, it will not be conducted at all (Figure 1.8).

Type II block occurs in the common bundle and its branches because this area cannot vary its conduction time sufficiently to permit measurable variation in the P-R interval. This inability to vary conduction occurs as a result of the extremely short relative refractory pe-

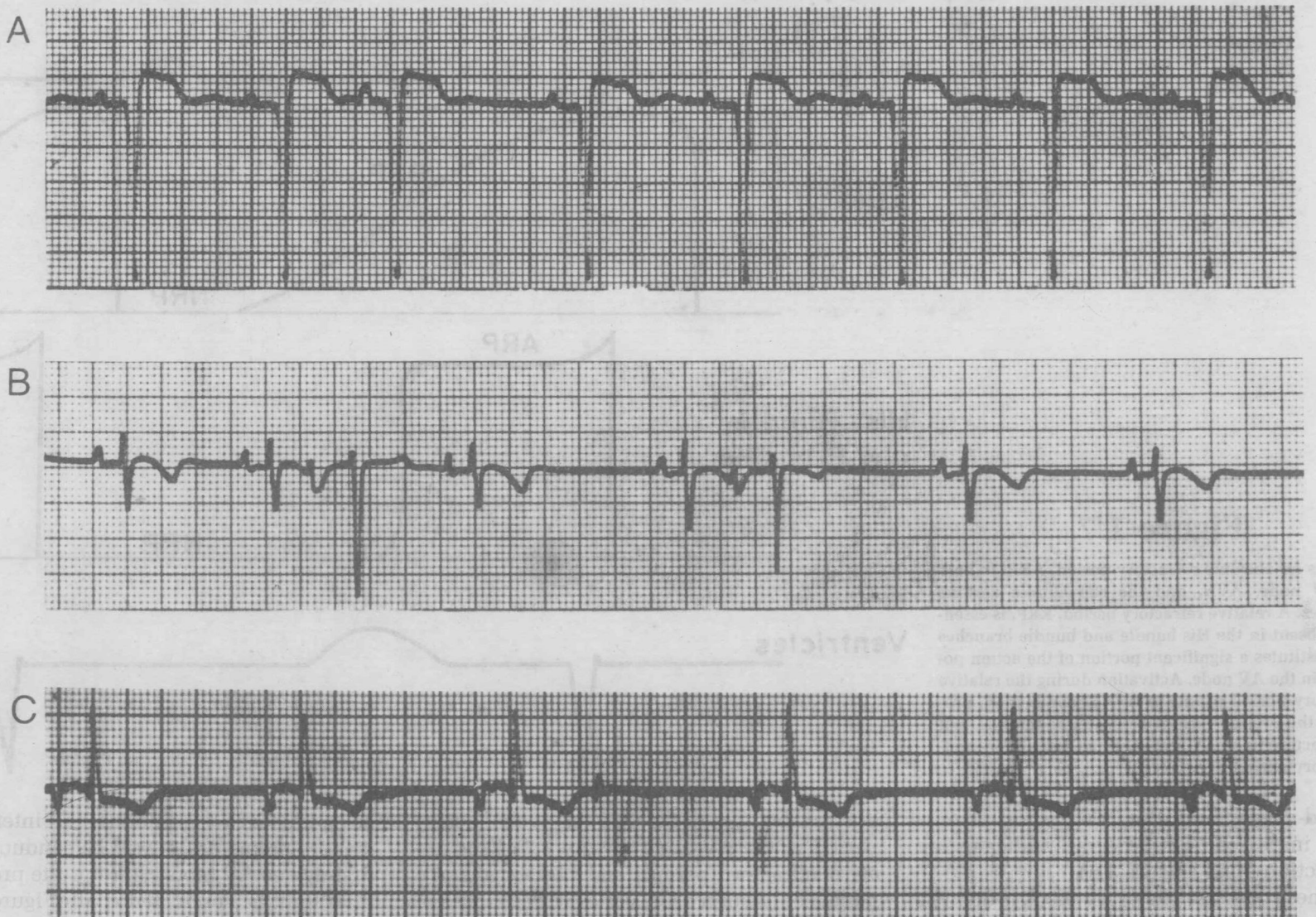


Figure 1.8.

In (A) the third P wave is premature, occurs well after the preceding QRS, and conducts with a P-R interval that is little changed from the P-R interval of the sinus beats. In (B) the third and sixth P waves are premature and occur much earlier in the cycle in comparison to the premature P waves in rhythm strip (A). Consequently, the P-R interval of these premature beats is prolonged. In rhythm strip (C) there is a premature P wave after the third cycle that is completely blocked. The blocked P wave is less premature than those in rhythm strip (B), illustrating that in this particular patient parasympathetic tone was higher and/or there was intrinsic AV node disease.

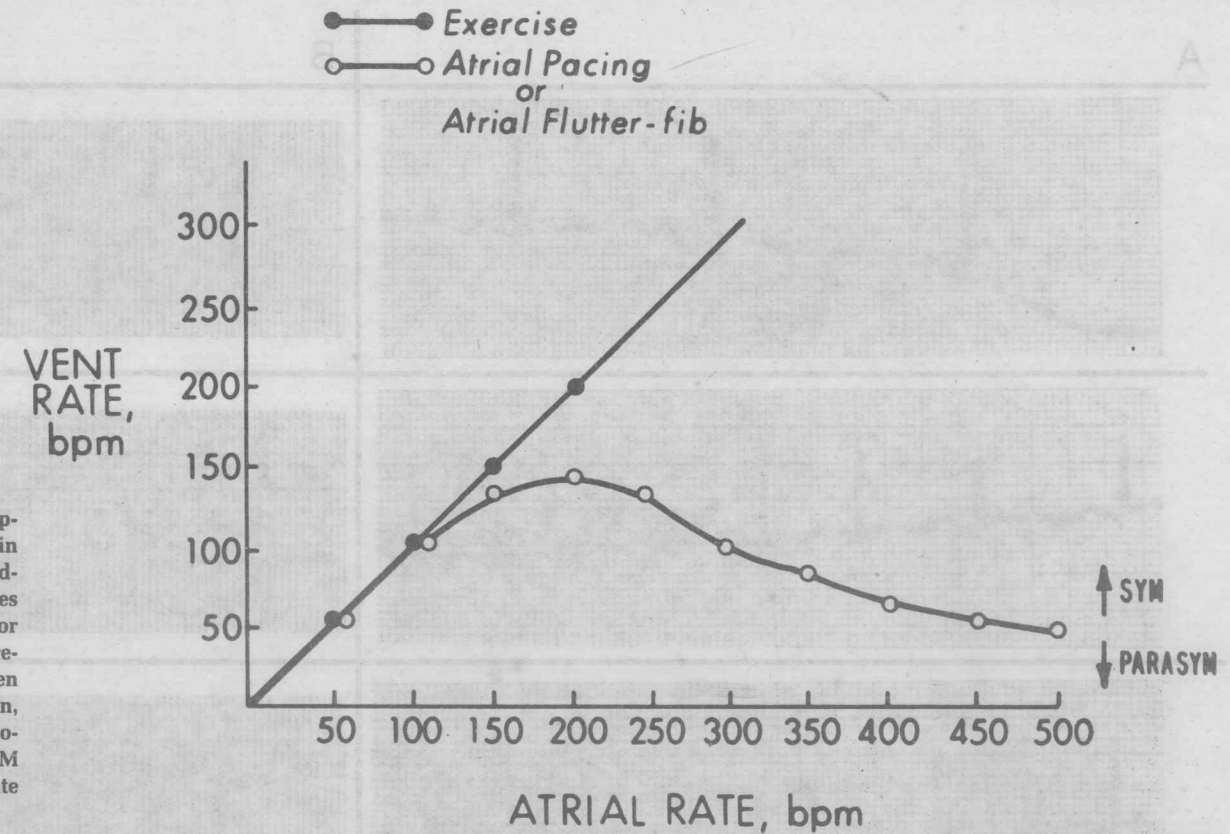


Figure 1.9.

The line of identity (closed circles) identifies the typical 1:1 AV relationship that exists when increases in atrial rate are mediated through exercise or other adrenergic stimulation. When the atrial rate increases through other mechanisms, such as atrial pacing or atrial flutter-fibrillation (e.g., open circles), a 1:1 relationship exists for the lower atrial rates, but when the atrial rate exceeds approximately 150 beats/min, AV nodal block typically occurs. Changes in autonomic tone (SYM = sympathetic tone and PARASYM = parasympathetic tone) can, respectively, facilitate or impair AV conduction.

riod of the His bundle and its branches (Figure 1.7). This short relative refractory period does not vary with changes in autonomic tone. Thus the conduction time through the His bundle and its branches remains constant despite progressively more premature activation. When, with extremely premature stimulation, the absolute refractory period is encountered, no conduction will result (see Chapter 4, Figure 4.14). The His bundle and its branches thereby exhibit "all-or-none" conduction capability (Figure 1.6C).

Type I block can be easily recognized by observing a *variation* in the P-R interval (Figure 1.6A). Wenckebach block is the classical example of Type I block. Wenckebach block rarely occurs in its pure form (increasing P-R, decreasing R-R, and pause less than twice the shortest R-R) because of intervening variation in autonomic tone. It is important to realize that "typing" block requires observation of conduction times and therefore is possible in the presence of either first- or second-degree block. Type II block is much more difficult to identify. It is possible to diagnose Type II AV block only when the P-R interval remains constant despite variations in the preceding resting period (Figure 1.6C). When the constant P-R interval occurs only in the presence of a constant resting period, typing of the AV block is impossible. This occurs when 2:1 AV block is present as in Figure 1.6B. In this situation there is a great temptation to diagnose "Type II" block simply because the P-R is constant. However, since there is no variation in the resting period that precedes the P-R interval, one would expect a constant AV nodal conduction time as well as a constant His bundle or bundle branch conduction time. This 2:1 pattern of second-degree block therefore is not diagnostic of Type II block (see Chapter 4, Figures 4.23, 4.24, and 4.27).