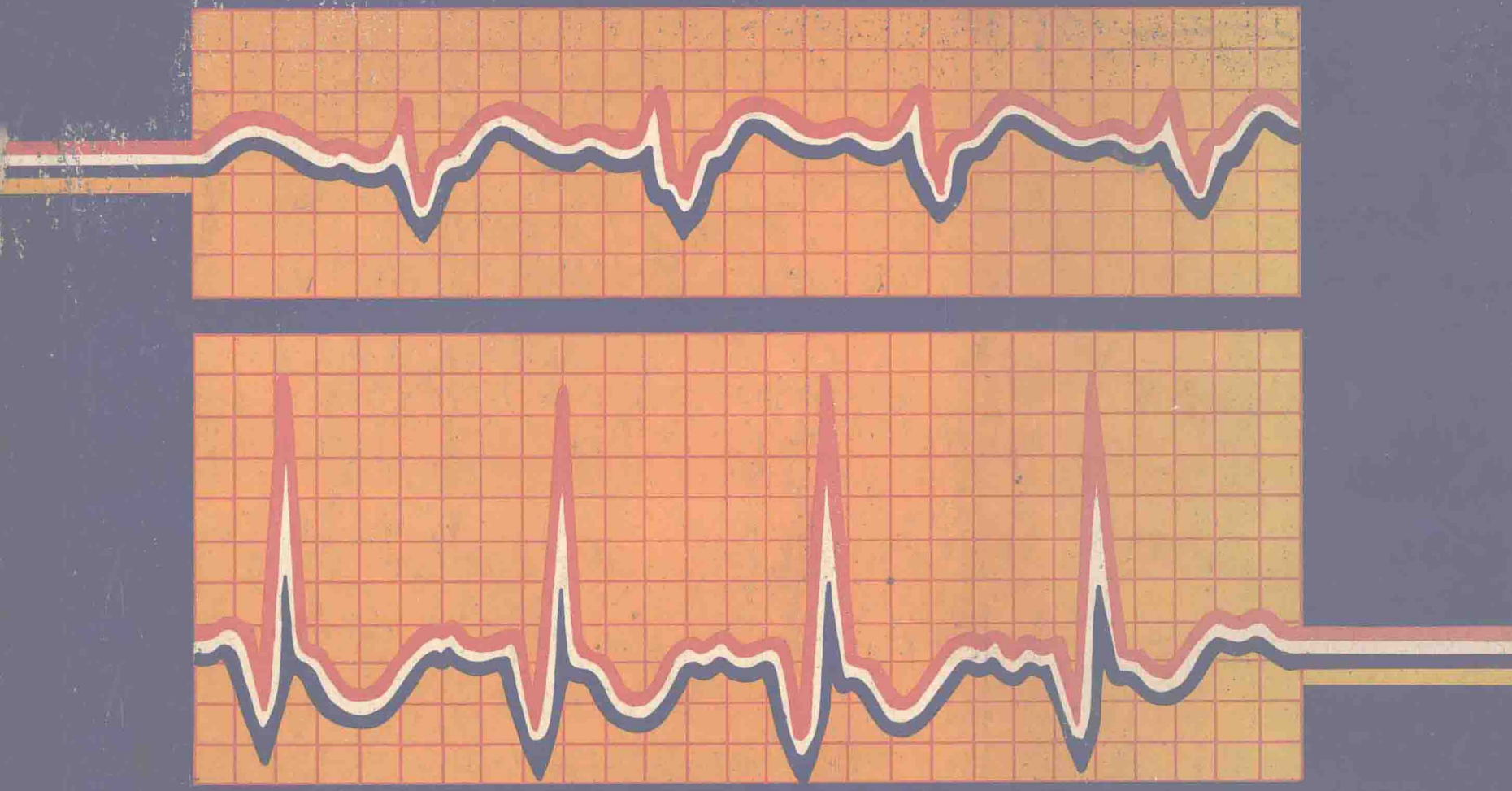
THE CARDIAC ARRHYTHMIAS

BRENDAN PHIBBS



Third edition

THE CARDIAC ARRHYTHMIAS

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Preface

The third edition of this book, like the first two, is dedicated to the principle that scientific phenomena can be explained in clear, simple, almost basic English if the writer will take the trouble to do so. A lifetime of digging through jumbled syntax to unearth simple medical facts has convinced me that most medical writing is needlessly obscure. This book is an exercise on the other face of the coin; if it has a virtue, it is the virtue of simplicity.

Don't be surprised, therefore, when you sometimes read that a pacemaker "fires" instead of "discharging," or when "antegrade propagation of the depolarizing impulse" is translated as "the wave moves down." This is deliberate. (As long as the term "wave" and the direction "down" are defined, the last two statements are identical.)

Bundle of His recordings are again mentioned briefly; these recordings are of great research and theoretical interest and have certainly illuminated many problems of conduction, but it is rare indeed for a bundle of His recording to yield any information of benefit to the patient. When a cardiologist advises a clinician that a bundle of His recording is necessary to clarify a problem of rhythm or conduction, the clinician would be well advised to call in another cardiologist, possibly more skilled in electrocardiogram interpretation. Bundle of His recording for profit and publication has been a noxious weed in the garden of cardiology these past few years; with rare exceptions, all necessary therapeutic and prognostic conclusions can be derived from the surface electrocardiogram.

Beta-blocking agents figure more largely in this third edition than in the second. After the first rush of enthusiasm for beta blockade in treatment of arrhythmias, some reasonable indications have emerged. Tachyarrhythmias associated with preexcitation probably represent the prime indication for beta blockade treatment of arrhythmias, but of course there are many other uses, as described in this text. Again, a caution; beta blockade means depressed cardiac function and at times life-threatening bronchospasm. Always consider other agents that do not pose these threats.

Finally, I would like to emphasize that this book is written in a progressive manner. The first chapters should be comprehensible to anybody with a nursing or medical degree. New information is added in blocks, and terminology is allowed to become more complex throughout the book. The Problems, Practice, and Reinforcement chapters are inserted to enable the reader to feel comfortable with the new information and terminology stage by stage.

This is a basic book for the noncardiologist, and for those who find the subject congenial I would recommend the classic, basic text by Katz, Pick, and Langendorf, as well as the extensive and always excellent writings of Marriott and Schamroth, as entrees to the more recondite aspects of the cardiac arrhythmias.

Dr. Jennifer Wing, my colleague, frequent coauthor, and fellow investigator, has provided many of the new illustrations in this third edition, and for these as well as for her thoughtful criticism and unflagging encouragement, I am greatly indebted.

Brendan Phibbs

Contents

PART ONE

Basic anatomy and physiology

1 Anatomy and physiology of the conducting tissues of the heart

ANATOMY

The essential anatomy of the conducting system of the heart is surprisingly simple: it consists of only six major parts (Fig. 1-1).

Sinoatrial (S-A) node. The S-A node is a small structure located in the right atrium near its junction with the superior vena cava. It is composed of a mass of specialized cells richly supplied with blood vessels.

Atrial syncytium. The atrial syncytium is the network of cells that forms the walls of the atria.

Atrioventricular (A-V) node. The A-V node is the structure lying in the upper portion of the interventricular septum near the entrance of the coronary sinus into the right atrium. It is composed of Purkinje fibers.

Bundle of His (atrioventricular bundle). The bundle of His is a continuation of the A-V node into the interventricular septum. The ultrastructure of the bundle of His, as seen in the electron microscope, is quite different from the structure of the A-V node.

Right and left branches of the bundle of His. The common bundle quickly divides into two bundle branches, one going down each side of the interventricular septum.

Purkinje fibers. The Purkinje fibers are scattered throughout the subendocardium of the ventricles and represent the last link in the chain of excitation.

PHYSIOLOGY OF THE NORMAL HEARTBEAT (Fig. 1-2)

The S-A node is the normal pacemaker of the heart. It sends out an electric wave that traverses the conducting tissues of the heart, producing the muscular contraction of the heartbeat. Think of the atria as a large pond. Seated at one corner of the pond, you play the role of the S-A node. You do this by dropping a rock in the pond every second: each time, a ripple spreads evenly through the water.

There is a small outlet canal from the pond that soon divides into two main branches and then into a myriad of tiny channels. You note that part of the ripple you produce travels down the outlet canal and through all the channels to their extremities. This is a good representation of the spread of the exciting impulse through the heart.

It was formerly thought that the exciting wave started by the S-A node spread uniformly throughout the atrial tissues. Newer studies suggest that there are specialized pathways of conduction from the S-A node to the A-V node. While this information is of intense theoretical interest, it has no practical significance at this time.

A part of this wave front travels down the A-V node, into the bundle of His, out the two bundle branches, and through the network of nerve fibers in the ventricles.

A restoring or repolarizing wave travels back across the heart in the same fashion.

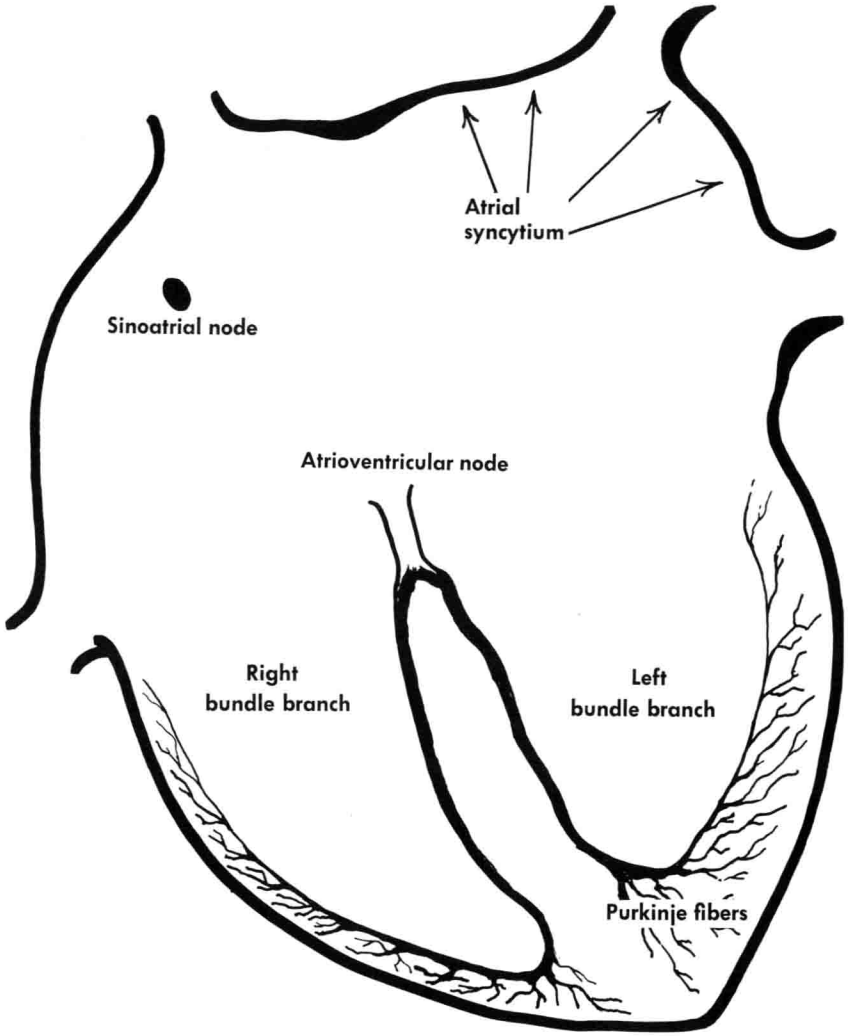


Fig. 1-1. Schema of conducting tissues of the heart.

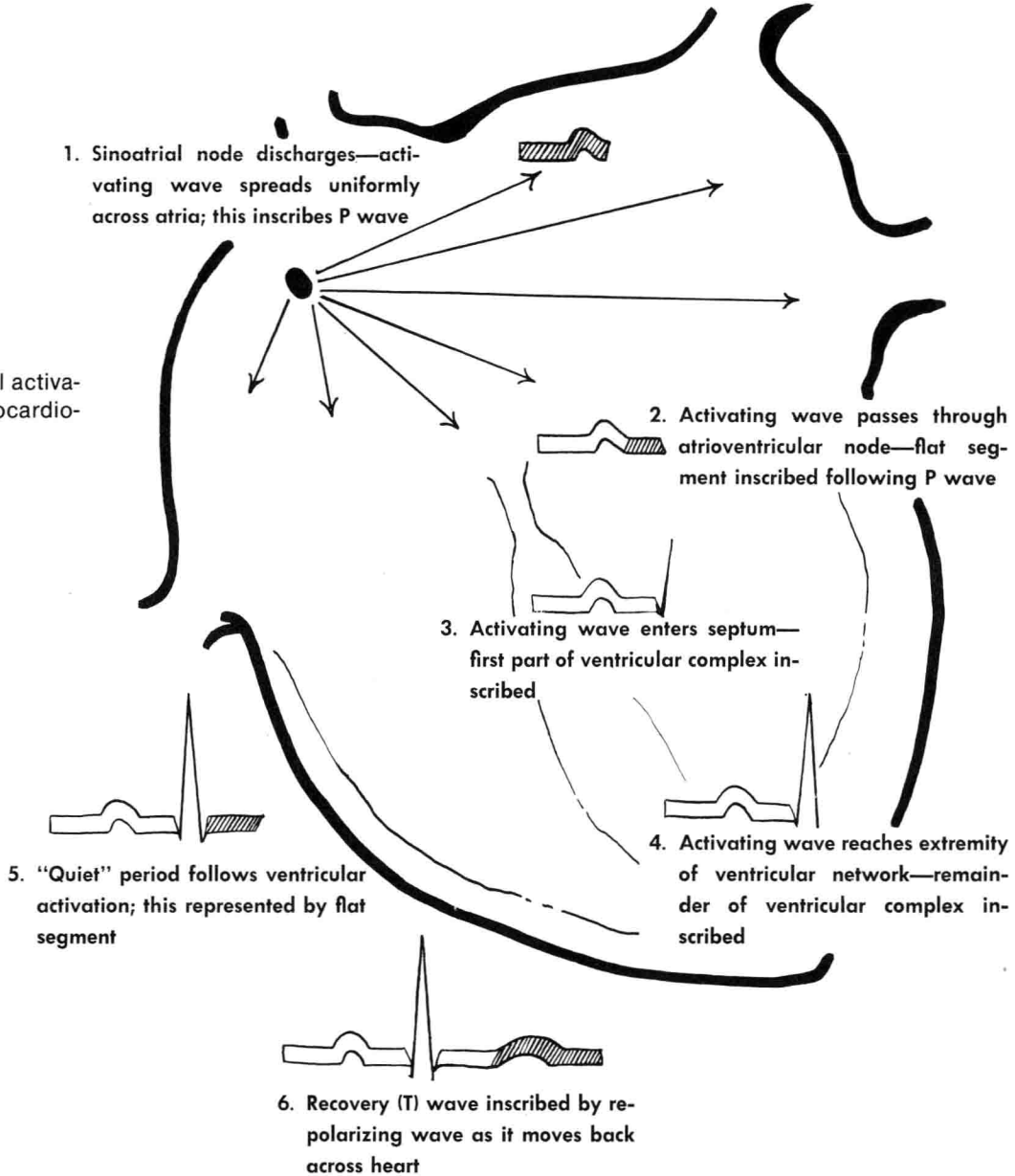


Fig. 1-2. Sequence of events in electrical activation of the heart as registered on electrocardiogram.

Correlation with waves of electrocardiogram

Fig. 1-2 shows the relation of these events to the deflections of the electrocardiogram (ECG). To diagnose arrhythmias by means of the electrocardiogram, one must be ready to use four sets of facts:

1. The S-A node discharges "silently"; no electrical activity is recorded in the surface electrocardiogram at the time of discharge of the pacemaker cells within this node or during the passage of the activating wave through the centimeter of tissue that constitutes the S-A node itself.

2. The P wave represents activation of the atria. The beginning of the upstroke of the P wave indicates the time atrial activation starts; the return of the P wave to the baseline indicates time of completion of atrial activation. In other words, the presence of a P wave indicates that atrial activation has taken place, and the width of the P wave tells the time required for completion of that activation.

3. No electrical activity is recorded at the surface of the body during passage of the activating wave through the A-V node and bundle of His. The flat segment of the electrocardiogram from the end of the P wave to the beginning of the QRS indicates this "silent" passage.

4. The ventricular conducting system begins at the "fork in the road" where the first branch of the bundle branch system leaves the common bundle of His. Inscription of the ventricular complex, or QRS complex, begins when the activating wave breaks out of the slowly conducting bundle of His and begins its swift course through the bundle branches. The QRS ends with the activation of the most remote part of the Purkinje network—the area around the crista supraventricularis in the right ventricle.

The presence of a ventricular complex indicates activation of the ventricular conducting system; the width of the ventricular complex tells how long that process takes.

EMPHASIS: Movement of an activating wave across any part of the atria or ventricles will always produce a deflection in the electrocardiogram, whether the chambers are activated in whole or in part. Movement of an activating wave through the A-V node and bundle of His is always silent, and the time required for such movement is indicated by the duration of a flat or isoelectric segment of the electrocardiogram, starting with the end of the P wave and ending with the beginning of the ventricular complex (in the case of normal activation).

Basic physiology

Nervous control of the conducting system. Both the sympathetic and the parasympathetic nerve fibers reach and influence the conducting tissues of the heart. They are basically completely opposite in their effects. Three sets of facts must be learned first:

1. Sympathetic nerve fibers are *cardioacceleratory:* stimulation through these fibers speeds the heartbeat by increasing the firing rate of the normal pacemaker (the S-A node) as well as of abnormal pacemakers (ectopic foci) in the various parts of the heart. Sympathetic stimulation also speeds the rate of conduction through the conducting system.

2. Parasympathetic fibers are *inhibitory:* stimulation of these fibers causes depression of pacemakers and slowing of conduction. Thus, with excessive parasympathetic (vagal) influence the S-A node will slow its rate of discharge; conduction through the A-V node will also be slowed as a result of the vagal effect. (This latter phenomenon is exceedingly important in terms of A-V block and of digitalis effect—note it well and remember it. You will use it often.) Vagal stimulation also depresses ectopic pacemakers in the A-V node, a fact of great importance when nodal rhythms are being studied.

3. Vagal fibers do *not* reach the conducting tissues of the ventricles; vagal fibers reach and influence the S-A node, the atrial tissues generally, the A-V node, and probably the

bundle of His, although this is not universally agreed. Sympathetic fibers, on the other hand, do reach all elements of the conducting system of the heart—atrial, A-V nodal, and ventricular.

Refractory period of conducting tissues (Fig. 1-3). Transmission of an electric wave, or potential, involves *work* on the part of cells doing the transmitting. Fatigue is produced, and the cells cannot transmit another impulse until they have recovered from this fatigue. The recovery of a particular tissue or cell takes a definite, measurable amount of time. The cell is said to have "recovered" when it is ready to transmit another impulse *normally*. It is said to have *partially recovered* when it is capable of transmission but is still somewhat fatigued so that the transmission is slow.

The time immediately following transmission of an impulse, during which a cell is not capable of any transmission at all, is called the *absolute refractory period*; in other words, the cell is absolutely incapable of transmitting during this time. Following the absolute refractory period comes an interval of time when the cell is capable of transmitting but does so *slowly*. This is the *relative refractory period*, when the rate of transmission is slow relative to the normal rate.

Diseased cells almost always have an abnormally prolonged refractory period, both relative and absolute. This is a fact of great clinical significance and will be referred to repeatedly throughout the text.

Transmitting activating impulse requires work on part of cells involved

After passage of impulse, cells fatigued; not capable of another transmission at that instant

After short period of time, cells begin to recover—

— And are soon ready for another transmission

Fig. 1-3. Refractory period, that is, period during which the cells of a conducting tissue are unable to function because of fatigue from previous transmission.

2 Basic facts and measurements in the electrocardiogram

NOMENCLATURE OF WAVES

The atrial deflection is called the P wave. An initial downward wave in the ventricular complex is a Q wave; any upward deflection is an R wave. Any downward deflection that follows an R wave is an S wave (Fig. 2-1, *A*).

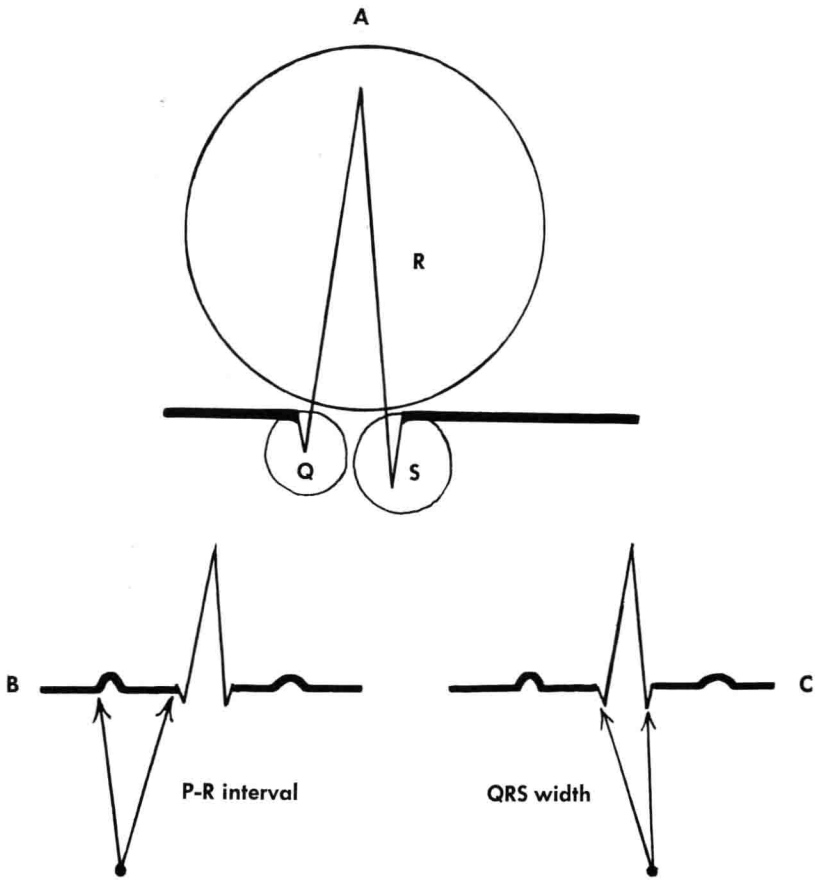
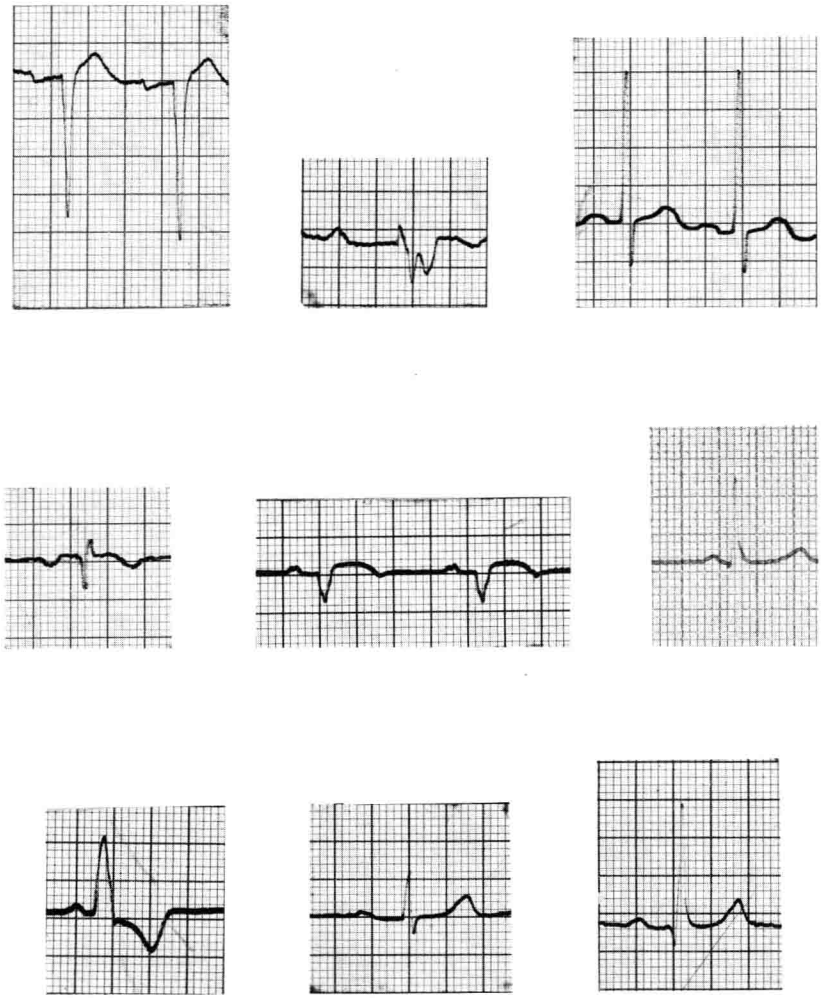


Fig. 2-1. A, Identity of ventricular deflections. **B,** Method of measuring P-R interval. **C,** Method of measuring the QRS interval.



TIME MEASUREMENTS

Each small square of the paper on which the electrocardiogram is recorded equals .04 second. Each large square contains five small ones and therefore equals .20 second.

Two measurements must be made at various times. The P-R interval, or the measurement from the first deflection of the P wave to the first deflection of the ventricular complex, is shown in Fig. 2-1, *B*. This is the time required for the activating wave to travel from the S-A node across the atria and through the A-V node and bundle of His. Normal values lie between .12 and .20 second.

Measurement of the QRS interval from the first ventricular deflection to the last (Fig. 2-1, *C*) demonstrates the time required for the spreading of the activating impulse through the ventricles from the bundle of His to the Purkinje fibers. Normal values are .09 second or less in the limb leads, .10 second or less in the precordial leads.

Some examples of electrocardiographic complexes are shown in Fig. 2-2.

Fig. 2-2. Varieties of electrocardiographic complexes. Practice measuring the time P-R and QRS intervals in each strip.

PART TWO

Simple arrhythmias

3 The sinus rhythms

"Sinus" refers to the sinoatrial node. Sinus rhythms are those rhythms that originate in the S-A node—in other words, *normal* rhythms.

As a rule, a sinus rhythm produces a regular pulse that is obvious upon a bedside examination. This is not always the case, however. Some sinus rhythms may be quite irregular and on clinical examination may be confused with more serious arrhythmias.

Before going any further, the physician must be quite sure he can recognize a sinus rhythm in the electrocardiogram. *Read and reread this chapter.*

ELECTROCARDIOGRAPHIC DIAGNOSIS OF SINUS RHYTHMS

The S-A node discharges regularly. *P waves will therefore be inscribed at regular intervals.*

Each impulse travels down the A-V node and activates the ventricles. *Each P wave will therefore be followed at a regular interval by a ventricular complex.*

The reader will instantly ask, "How *long* is this regular interval?" To answer this question it is necessary to remember only two numbers: .12 and .20. The time that elapses from the origin of the beat (the beginning of the P wave) to the first deflection of the ventricular complex will lie between these two figures in the adult heart. In infants and children this interval will frequently be shorter. In other words, an impulse cannot follow a normal pathway from the S-A node to the ventricles and arrive there in less than .12 second. On the other hand, an impulse that takes longer than .20 second to traverse this pathway must have been slowed by diseased tissue along the way, probably in the A-V node. This interval is referred to as the P-R interval. (See Fig. 2-1, *B*, for the method of measurement.)

Each activating wave follows an identical pathway across the atria and through the ventricles. Therefore, the shape or contour of all complexes will be identical. Each P wave will look like every other P wave. Each ventricular complex will look exactly like every other ventricular complex (Fig. 3-1).

In summary, a normal or "sinus" mechanism is diagnosed when—

1. P waves appear at regular intervals.
2. Each P wave is followed at a regular normal interval by a ventricular complex.
3. All P waves and ventricular complexes have the same contour and configuration (Fig. 3-1).

VARIATIONS OF SINUS RHYTHMS

Sinus tachycardia (Fig. 3-2). In sinus tachycardia the rate is over 100. It will rarely exceed 160. Exertion, thyrotoxicosis, and fever are obvious causes.

Sinus bradycardia (Fig. 3-3). In sinus bradycardia the rate is under 60. It may drop to 40 or less in extreme cases.

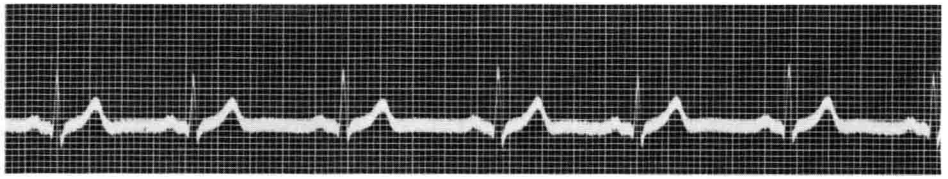


Fig. 3-1. Normal electrocardiogram.

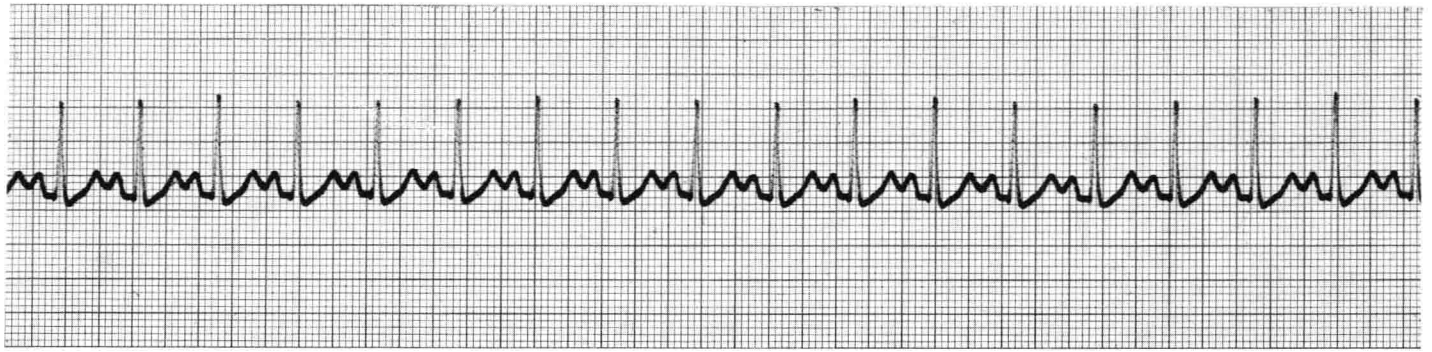


Fig. 3-2. Sinus tachycardia.

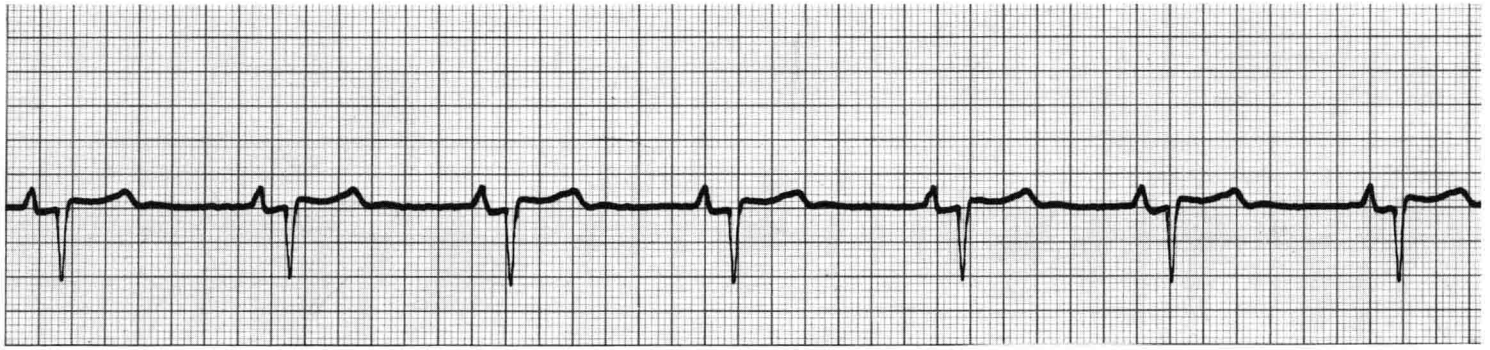


Fig. 3-3. Sinus bradycardia.

Sinus arrhythmia (Fig. 3-4). In sinus arrhythmia the rhythm varies irregularly. Commonly, the rate becomes more rapid during inspiration and slows on expiration, although this is not always true. Sinus arrhythmias may fluctuate independently of breathing. As a rule, sinus arrhythmia has no clinical significance. However, when the fluctuations in rhythm are extreme with long pauses in the slow phase, the physician should consider the possibility of sick sinus syndrome (see Chapter 14).

Note that in each case the basic criteria for a sinus mechanism are present. Whether the beats come rapidly, slowly, or irregularly, each cycle consists of a P wave followed at a normal interval by a ventricular complex. The total configuration of each "beat" is exactly like that of every other "beat."

Complex disorders of S-A node function, such as the sick sinus syndrome and various forms of S-A block, are noted in Chapter 14.

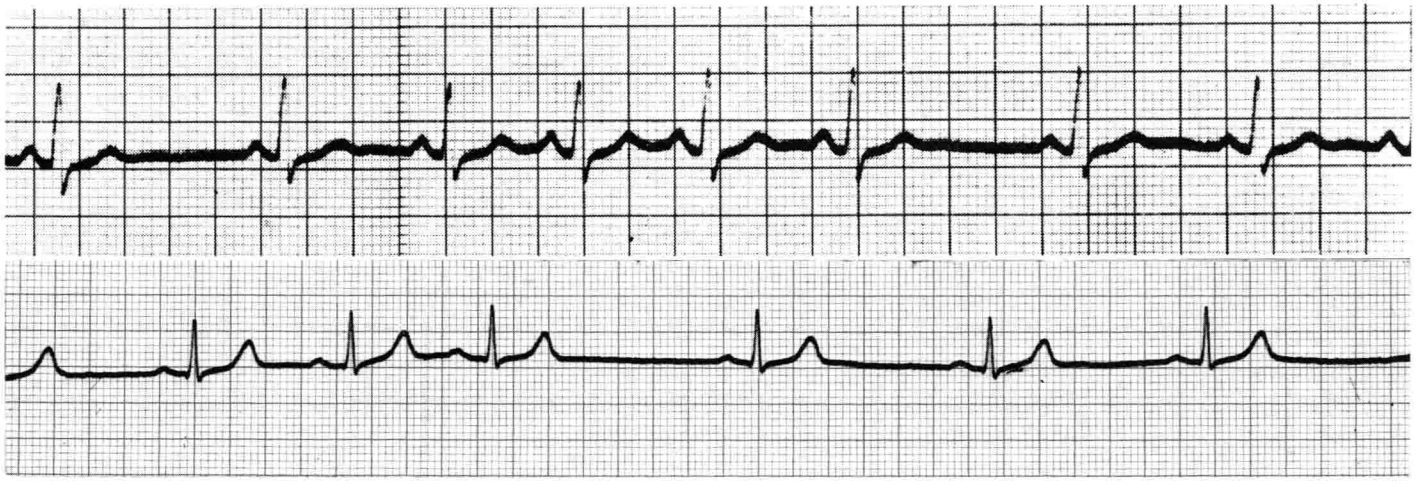


Fig. 3-4. Sinus arrhythmia.