THE

CARDIAC ARRHYTHMIAS

An Approach to Their Electrocardiographic Recognition

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Preface to the Second Edition

IN THE NEARLY 5 YEARS that have elapsed since the publication of our first edition, we have seen the birth of an era of enormous advances in electrocardiography. His bundle recordings have uncovered a wealth of knowledge and in many cases have confirmed or disproved decades-old theories that attempted to elucidate the mechanism of production of various arrhythmias. In addition, the widespread uses of intracavitary recordings in surgery, intensive care units and cardiac catheterization laboratories have opened a significant number of new avenues in practical electrophysiology.

We have tried in this second edition to incorporate as much of the new knowledge as is possible without departing from our original purpose, stated in the preface to the first edition, of serving primarily the needs of residents or interns in medicine and of nurses in coronary care units. When possible, new tracings have been chosen to replace those which, in our opinion, could be improved. The section on test tracings has been revised extensively, and approximately 30% of the records shown are new.

The section on pre-excitation has been considerably revised as a result of new knowledge on the subject, and new tracings are incorporated. A discussion of isorhythmic ventricular rhythm has been added, accompanied by appropriate examples. A new section has been introduced, Secondary Mechanisms, which describes those arrhythmias that appear only as a consequence of other (primary) disorders of cardiac rhythm. The term "wide QRS tachycardia" is applied to rapid, regular rhythms of unclear origin, avoiding the much-abused label "ventricular tachycardia" (empiric in most clinical situations, anyway).

A large number of boxed definitions in the text have been revised in order to make them more precise and easier to understand.

Nomenclature has also been upgraded. Measurements of intervals are now given mostly in milliseconds and the terms "beat" and "contraction" are avoided when possible. They have been replaced in most cases by "depolarization" when used in reference to phenomenona that are essentially electrical in nature.

The lead shown in the tracings that accompany the text is usually V_1 or a bipolar precordial that closely resembles it. If other leads are used, they are labeled accordingly.

The authors wish to extend their appreciation to Dr. Minor Duggan, Director of the Medical Illustrations and Publications Department of the Miami Heart Institute, for reviewing the manuscript, and to Ms. Louise Jeskey for the many, many hours spent at the typewriter.

A.E.L.
A.B.

Preface to the First Edition

IT IS OUR PURPOSE in this manual to present a detailed, illustrated classification of the arrhythmias commonly seen in clinical cardiology in the hope that it may aid the moderately advanced electrocardiographer to recognize and identify rhythm disorders.

The serious student of arrhythmias has several excellent textbooks available to him, notably the works of Katz and Pick[1] and Scherf and Cohen.[2] At the other extreme, the cardiac nurse trainee and the medical student have access to many simplified approaches to arrhythmia diagnosis. But the needs of the resident or intern in medicine, who frequently is confronted with problems in arrhythmia diagnosis, particularly in coronary care units, have been largely neglected. It is in an effort to meet these needs that the authors have sought to provide a training manual which consists of an amply illustrated atlas of arrhythmias together with their classification and the basic diagnostic criteria for their precise definition. Although designed primarily for the medical resident or intern, this manual can also be used effectively for advanced study by cardiac nurse specialists and practitioners who wish to progress beyond the confines of elementary textbooks.

The bulk of the manual is devoted to a systematic classification of arrhythmias with appropriate illustrations on the left-hand page so that easy correlation may be made with the textual material and diagnostic criteria given on the right-hand page. In setting up this material we have used a classification that was originally designed for teaching ECGs by the digital computer. We have not attempted to present the more controversial or highly complex arrhythmias because they are handled competently elsewhere. On the other hand, in order that all abnormalities of impulse formation and impulse conduction may be represented, we have included certain types of conduction disturbance (i.e., bundle branch block, intra-atrial block, etc.), even though they produce no abnormality in cardiac rhythm. We have also included a description of the more commonly used modes of artificial cardiac pacing complemented by a few commonly seen rhythm disturbances caused by electronic pacemakers.

In our classification we have emphasized the consideration of morphologic criteria, and have referred to mechanisms only when the ones at play are basic, well known and accepted by most authorities in the field. Etiology, therapy, complications, associated conditions and prognosis are also not taken into consideration since the different abnormal rhythms are taken out of any assumed clinical context. There can be no "specific therapy" for any individual arrhythmia unless the clinical setting is thoroughly understood.

We have used the term "AV junctional" in preference to "AV nodal," since automatic impulse formation in the AV node has been repeatedly questioned.[3,4] We have stressed the extreme difficulty, if not practical impossibility, of determining with certainty the ventricular origin of a

tachycardia with wide QRS complexes in the routine standard 12 lead electrocardiogram. We use the term "fourth degree heart block" for defining those cases of AV dissociation in which there is complete heart block.

An additional section of the manual outlines a diagnostic approach to arrhythmia interpretation using a "flow diagram" much like that which one would use to program a digital computer for recognizing arrhythmias. Through the use of this flow diagram one can approach an arrhythmia in a logical manner, seeking a series of "yes-no" answers to basic questions derived from electrocardiographic observations and measurements. This allows the student to learn the value of certain sets of criteria for arrhythmia diagnosis rather than to rely on rote pattern recognition alone.

The Test Tracings (p. 122) may be used for self-instruction or by an instructor. A key to these tracings will be found on page 144. ECG tracings with all grades of complexity are included.

Finally, the manual offers a series of references for the student whose interest in any particular rhythm goes beyond the material presented.

We are grateful to the Inter-Mountain Regional Medical Program (Coordinator, Dr. C. Hilmon Castle) for an initial trial printing of this manual so that it could be used extensively in teaching, both in Salt Lake City and in Miami, before final revision. Drs. Agustin Castellanos, Jr., and Antonio Gomez of Miami kindly supplied some of the tracings used in the section on artificial pacemakers. Dr. J. A. Abildskov reviewed some of the material in the manual, and we are thankful for his suggestions and criticisms. We must acknowledge the enormous contribution of the ECG technicians and cardiac nurse specialists at the Latter-day Saints Hospital, Salt Lake City, whose sharp eyes and curiosity led to the obtaining of many of these tracings. Finally, we thank Barbara Lindsay, without whose encouragement this book would not have been conceived, and without whose forbearance and enthusiasm it could not have been consummated.

A.E.L.
A.B.

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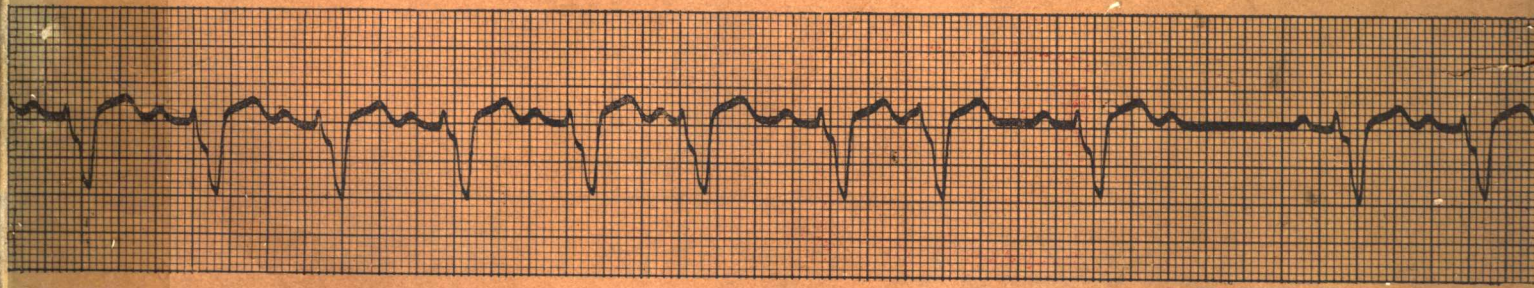
The Cardiac Arrhythmias



Measurement of Heart Rate

HEART RATE may be measured precisely by dividing the R-R intervals (in seconds) into 60. For instance, if the R-R interval is 0.8 sec, the heart rate would be:

$$\frac{60}{0.8} = 75 \text{ per minute}$$

Figure 1 illustrates a simple way of approximating heart rate. Although not exact, it will enable the interpreter to tell at a glance whether the heart rate is over 100 but less than 150, or less than 60 but more than 50.

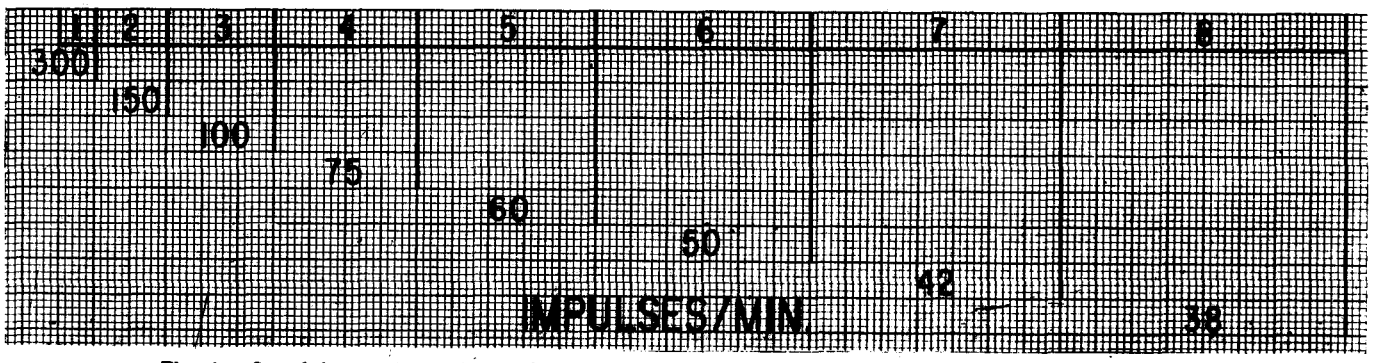


Fig. 1.—A quick way to estimate heart rate. The top line shows the number of large (200 msec or 0.20 sec) squares for the indicated heart rate.

Commonly Used Abbreviations

SA:	Sinoatrial
AV:	Atrioventricular
PAD:	Premature Atrial Depolarization
PVD:	Premature Ventricular Depolarization
IVCD:	Intraventricular Conduction Defect
AVC:	Aberrant Ventricular Conduction
RBBB:	Right Bundle Branch Block
LBBB:	Left Bundle Branch Block
IVR:	Isorhythmic Ventricular Rhythm

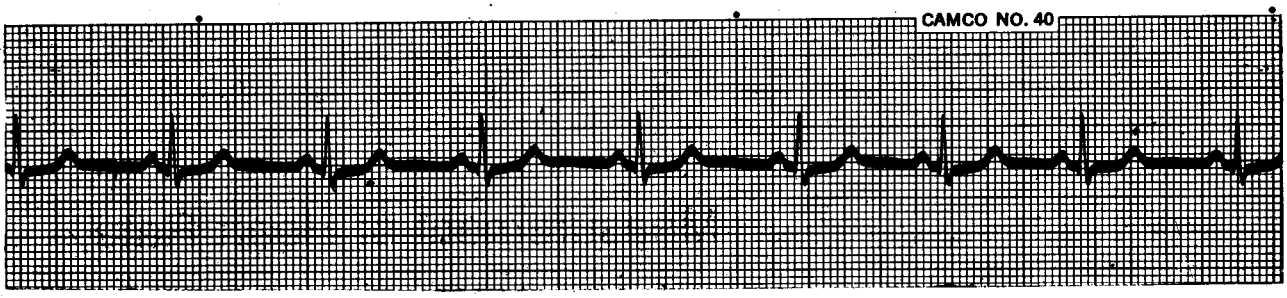


Fig. 2.—Normal sinus rhythm (lead 2). The heart rate is 69 and the P-R interval is 150 msec. QRS duration of 80 msec indicates normal intraventricular conduction.

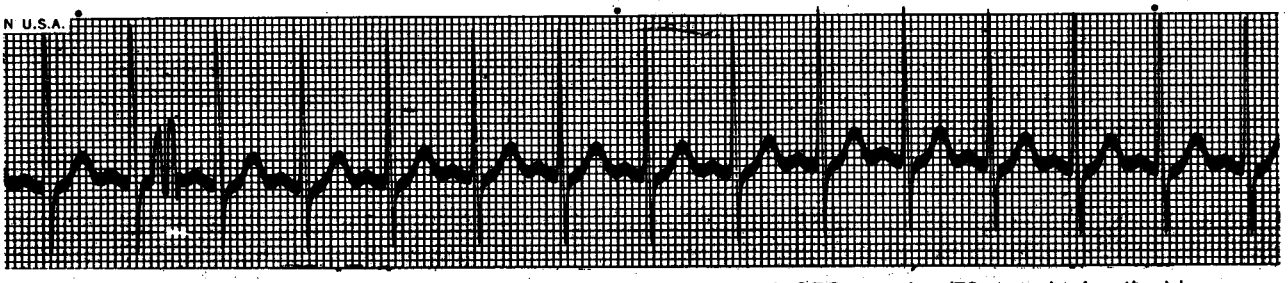


Fig. 3.—Sinus tachycardia (lead 2). The heart rate is 125; each QRS complex (70 msec in duration) is preceded by a P wave with a fixed P-R interval of 130 msec.

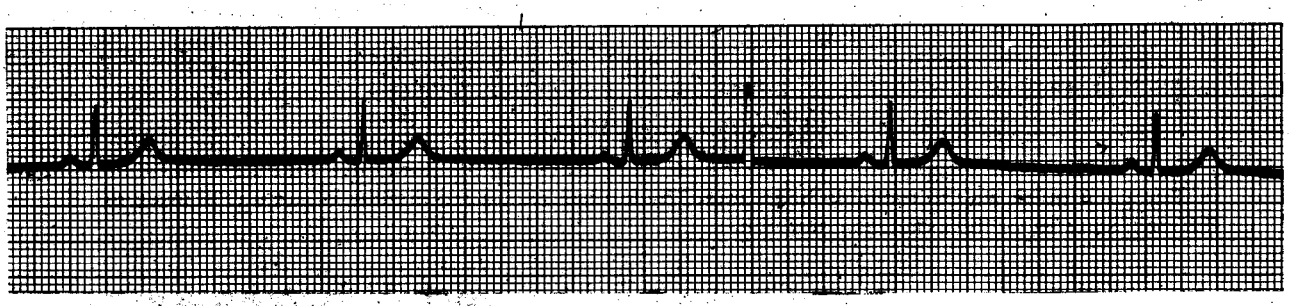


Fig. 4.—Sinus bradycardia (lead 2). The heart rate is 41. Each QRS complex (60 msec in duration) is preceded by a P wave with a fixed P-R interval of 160 msec.

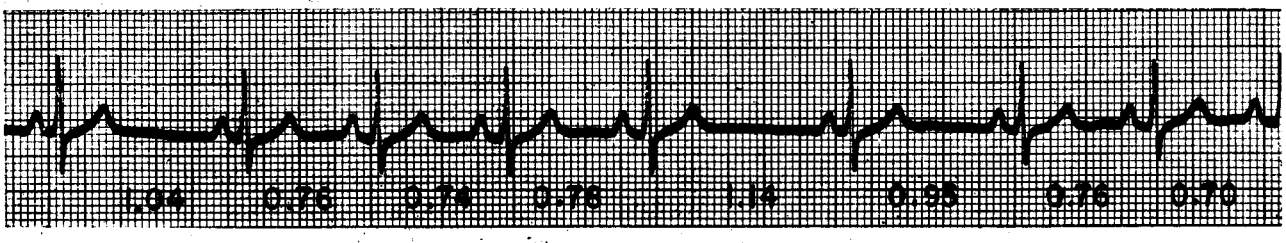


Fig. 5.—Sinus arrhythmia (lead 3). Cyclic variation in heart rate is present, i.e., the R-R intervals vary from 700 msec to 1.14 sec. The P-R intervals are constant.

Basic Definitions

Normal Sinus Mechanism: QRS complexes follow P waves at fixed intervals, the P-R interval ranging from 120–200 msec at normal heart rates in adults. Upright regular P waves are seen in leads 1 and 2, and inverted P waves are present in lead aVR. The QRS complexes are 100 msec or less in duration and of normal configuration in the absence of bundle branch block or aberrant ventricular conduction (AVC).

Figures 2–5 show examples of ***sinus rhythms.*** In sinus arrhythmia, the variation in R-R intervals is more than 10% of the average R-R interval.

Tachycardia: The rate of effective impulse formation of the qualifying focus (sinus, atrial, AV junctional or ventricular) is 100 per minute or more.

Bradycardia: The rate of effective impulse formation of the qualifying focus is 60 per minute or less.

Arrhythmia: In its pure sense, the term "arrhythmia" denotes a lack of rhythm, and as such is improperly applied to many disorders of the heart beat. Obviously, most atrial tachycardias, with their remarkable regularity, are not arrhythmic, whereas atrial fibrillation, with its completely unpredictable R-R intervals, is arrhythmic. Perhaps, a general term "dysrhythmia" should be applied to all abnormalities of impulse formation or impulse conduction, regardless of rate or regularity.

Ectopic Depolarizations: Depolarization of the atria or ventricles, or both, occurs at a time not anticipated from extrapolation of the basic underlying rhythm. Ectopic activity, which may arise in the atria, AV junction or ventricles, may assume one of two forms:

1. *Premature depolarizations,* which follow the preceding QRS complex by an interval shorter than the prevailing R-R interval. These may be further subdivided into:
   a. *Extrasystoles:* Premature depolarizations that follow the preceding QRS complex by a fixed interval (fixed coupling).
   b. *Parasystole:* Premature depolarizations that originate in an independent and protected focus and follow the preceding QRS complex by varying intervals (nonfixed coupling).
2. *Escape depolarizations,* which follow the preceding QRS complex by an interval longer than the prevailing R-R interval.

Two special features of ectopic activity should be observed carefully:

1. *Coupling interval:* The time between the onset of the preceding QRS complex and the onset of the ectopic depolarization.
2. *Post-extrasystolic pause:* The pause following an extrasystole is considered to be fully compensatory when the R-R interval of the two complexes surrounding the premature complex is twice the prevailing R-R interval (complete compensatory pause).

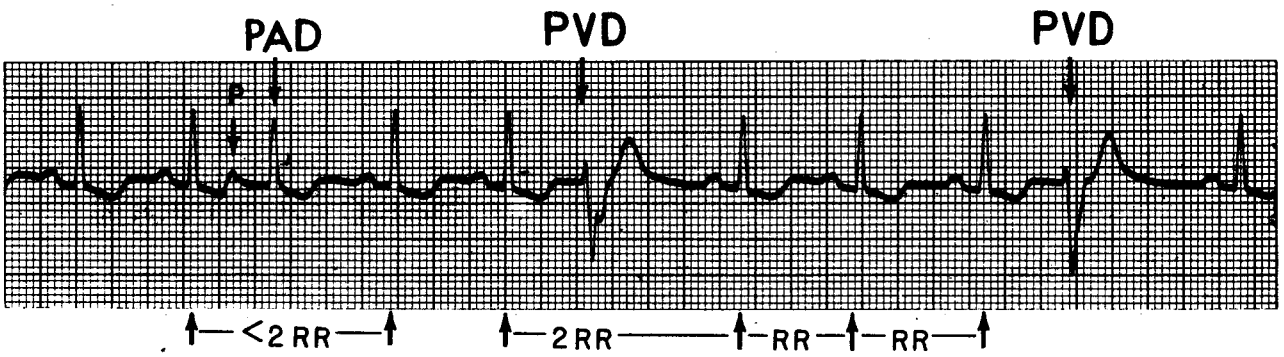


Fig. 6.—Sinus rhythm with an atrial and two ventricular extrasystoles. Complete and incomplete compensatory pauses are illustrated (see text).

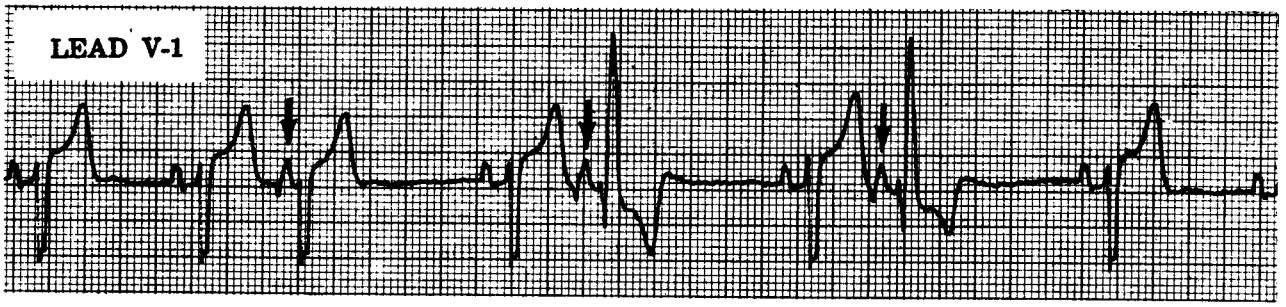


Fig. 7.—Aberrant ventricular conduction (AVC). The 2d and 3d premature P waves (arrows) are followed by QRS complexes with aberrant conduction.

Figure 6 shows that the compensatory post-extrasystolic pauses following the premature ventricular depolarizations (PVDs) are complete, whereas the pause following the premature atrial depolarization (PAD) is partially compensatory and is less than the sum of two prevailing R-R intervals. The partially compensatory pause is caused by resetting of the sinoatrial (SA) node, a common result of premature atrial stimulation. It is very important to look for P waves hidden in the T wave preceding any premature ectopic depolarization. Failure to differentiate between PADs with AVC (see below) and PVDs often leads to therapeutic errors.

Interpolation: Interpolation occurs when an extrasystole is enclosed or "sandwiched" between two normally timed P-QRS sequences. The prevailing rhythm is not disturbed except, for example, when the P-R interval following a ventricular interpolation is slightly prolonged because of partial refractoriness of the AV conduction pathway (retrograde concealed conduction—see below).

Wenckebach Phenomenon: In the AV Wenckebach phenomenon, a progressive lengthening of conduction time between the atria and ventricles occurs. This is recognized in the ECG as a progressive prolongation of the P-R interval for a number of cycles until conduction fails and ventricular depolarization does not occur. After a pause, the sequence repeats itself producing group beating. Although usually seen as a disorder of AV conduction, the Wenckebach phenomenon may affect any segment of the conduction pathways. Thus, we may see it in the bundle branches, the SA junction, etc. In the AV junction, the Wenckebach phenomenon may occur during sinus mechanism or with any other type of supraventricular focus.

Aberrant Ventricular Conduction (AVC)[5]: This term describes temporary alteration of the QRS morphology under conditions where a normal QRS complex might be expected. It is used, for instance, to describe the broad QRS complex of an atrial extrasystole when ventricular depolarization occurs very early and part of the conduction system, usually the right bundle branch, is still refractory. *Figure 7* shows three PADs. The first follows the preceding P wave by 600 msec, and its QRS complex does not differ from the prevailing QRS morphology. The coupling time of the 2d and 3d premature P waves is shorter (480 msec); their QRS complexes are widened and exhibit the triphasic right bundle branch block (RBBB) morphology (rSR′) often seen in AVC.

Atrioventricular Dissociation: In AV dissociation, the atria and ventricles are activated separately, each set of chambers by its own focus (AV junctional or ventricular for the ventricles) with or without anatomic interruption of the AV conduction pathway. AV dissociation is not in itself a primary arrhythmia, but a phenomenon always secondary to some other disturbance of impulse formation or conduction, i.e., slowing of the SA node, acceleration of a junctional or ventricular focus, AV block, etc.

In cases of AV dissociation in which two foci coexist, either of which is capable of stimulating the entire heart or any single chamber, the term *dominant pacemaker* is used to refer to the focus which initiates ventricular depolarization. This focus is usually the more rapid of the two potential pacemakers. In AV dissociation, for instance, the dominant pacemaker may be a ventricular or AV junctional focus, yet the subsidiary pacemaker (sinus or ectopic atrial) remains active.

The term *capture* is used to designate temporary control of ventricular activation by a focus which at that time is not the dominant pacemaker. Capture may be seen in wide-QRS tachycardia, where occasional normally conducted depolarizations (normal QRS preceded by a P wave) occur. Capture may also occur in a retrograde fashion, i.e., the atria may be activated by an impulse arising in the AV junction or lower and thus are "captured from below."

Concealed Conduction (Partial Penetration)[6,7]**:** Under certain conditions, a stimulus fails to traverse the AV junction completely. This creates a conduction disturbance in the junctional tissues and delays the passage of the next stimulus. For example, in AV dissociation a supraventricular stimulus originating in the focus activating the atria is occasionally able to penetrate the AV junction partially and to depolarize the lower focus before it fires the next scheduled QRS *(antegrade concealed conduction)*. This resetting of the lower focus will be seen in the tracing as an unexpected pause. *Retrograde concealed conduction* can be inferred from the surface ECG under special circumstances, such as from the prolonged P-R interval following interpolated ventricular extrasystoles (p. 95). Concealed conduction may be observed in areas of the conduction system other than the AV junction.

Fusion Beats: Fusion beats arise when stimuli originating in two different foci concurrently depolarize a cardiac chamber. The resultant complex is a fusion or "combination" of the different morphologies of the two depolarizations had they occurred separately, depending upon the relative arrival times of the two depolarization waves. Thus, the ventricles may be activated through normal AV conduction pathways at the same time that a late ventricular extrasystole occurs. A fusion QRS, resembling both the normal QRS and the QRS ordinarily produced by the extrasystole, would then result. Fusion beats may occur at the atrial level, but are much less common.

Wide-QRS Tachycardia: This term is used to define rapid, regular rhythms of uncertain origin in which the QRS complexes are 110 msec or longer in duration. The site of origin of the tachycardia (i.e., AV junctional with AVC or ventricular) cannot be accurately determined from the surface ECG alone.

Parasystole: An independent and protected ectopic focus (atrial, AV junctional or ventricular in origin) coexists with the dominant cardiac pacemaker and produces premature complexes whose interectopic intervals have a common denominator. Fusion beats and nonfixed coupling of the premature complexes are usual characteristics of this arrhythmia. Although the parasystolic complexes may alter the dominant rhythm, the parasystolic focus itself is protected in some way and its rate of impulse formation is not altered by the normal activation forces.

Laddergram: A schematic representation used to illustrate the presumed sequence of events during cardiac activation as shown in *Figure 8*. Anatomic level is depicted vertically and time horizontally; the slope of the AV level is proportional to the length of the P-R interval. Arrows indicate direction of depolarization; dots indicate an ectopic focus.

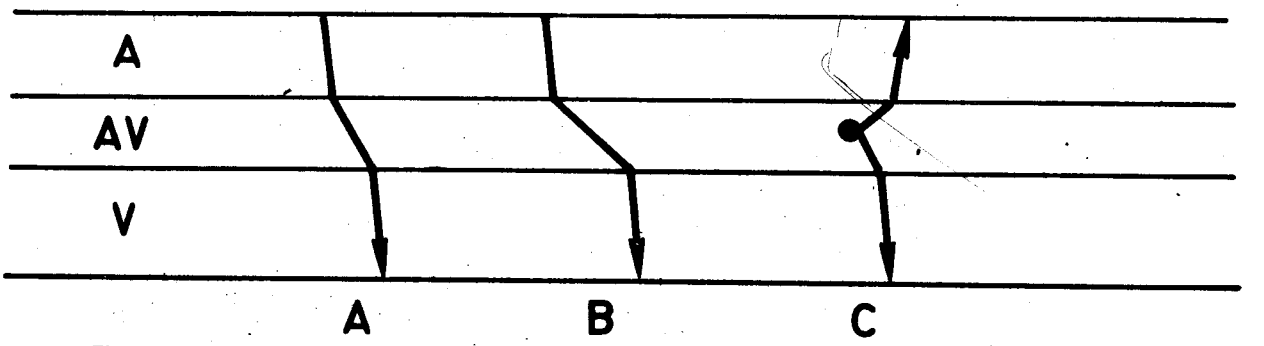


Fig. 8. – (A) The course of a **normal sinus depolarization.** (B) An example of **prolonged P-R interval.** (C) An **ectopic depolarization of AV junctional origin** with antegrade conduction to the ventricles and retrograde conduction to the atria.

Classification and Atlas of Arrhythmias

I. Rhythms Originating Above the Atrioventricular Junction

A. Abnormalities of Impulse Formation

1. ISOLATED OR INTERMITTENT

a. SINUS ARREST

> SINUS ARREST: The sinus node fails to stimulate the surrounding atrial tissue and consequently a P wave fails to appear at the expected time. This causes a pause that is not a multiple of the prevailing sinus interval. The pause is often terminated by an escape depolarization originating in a lower focus.

Figure 9 illustrates *sinus arrest*. Pauses occur because of failure of the SA node to fire on schedule. After variable intervals of time, escape depolarizations terminate the pauses; their normal QRS morphology indicates their AV junctional origin (p. 53). In the upper tracing, the interval between the 2d and 3d P waves is not a multiple of the prevailing P-P interval, thus differentiating this abnormality from SA block (p. 45). In the lower tracing, however, the interval between the 2d and 3d P waves (note that the 3d P wave barely precedes the escape depolarization) is three times the prevailing P-P interval. This may be coincidental or occur because of 3:1 SA block.

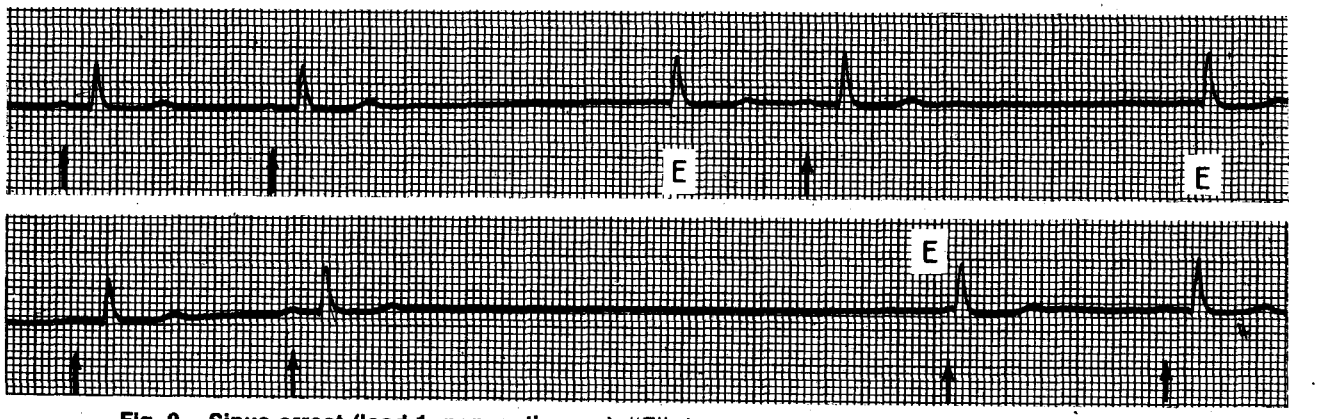


Fig. 9.—Sinus arrest (lead 1, noncontinuous). "E" denotes escape depolarizations; arrows indicate P waves.