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CLINICAL ELECTROCARDIOGRAPHY AND ARRHYTHMIAS

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CLINICAL ELECTROCARDIOGRAPHY

1. Introduction

The electrocardiogram had been used as a diagnostic test in clinical medicine for more than 70 years, and is now a routine part of any complete medical evaluation. Many cardiac abnormalities alter the heart's electrical activity, and cause changes in the ECG. Since the electrical activity of both atria and ventricles is reflected in the ECG, the test is of particular value in defining cardiac rhythm. Diseases which result in changes in the myocardial muscle mass will alter the ECG. For example, an increase in ventricular muscle mass (hypertrophy) usually results in larger QRS amplitudes. Diseases which cause death of heart muscle and replacement by scar tissue (such as myocardial infarctions) will be reflected in characteristic changes in morphology of the QRS complex. Inadequate blood supply to heart muscle resulting from coronary artery disease may cause alterations in the repolarization of muscle cells, which will be reflected in characteristic changes in the ST-T wave portion of the electrocardiogram.

On the other hand, the ECG is insensitive to some cardiac abnormalities, and many ECG changes are non-specific. Small, localized regions of heart muscle damage may exist but not be visible in the routine clinical ECG. The ECG may be completely normal in patients with severe coronary artery disease, as long as adequate myocardial blood flow is present at the time of the recording. Increased sensitivity may be obtained by recording the detailed distribution of potentials on the surface of the torso using large numbers (up to hundreds) of electrodes. Such "body surface mapping" requires extensive instrumentation and computation making it impractical for routine clinical use at the present time. Transient abnormalities such as coronary artery spasm, or sporadic rhythm disturbances may be completely missed in a routine ECG recording which documents less than one minute of data.

The usefulness of the ECG has been extended by technology for recording 24 hours of ECG data while the patient engages in his usual daily activities. Such ambulatory ECG ("Holter") recordings are particularly useful in documenting sporadic events. The combination of ECG recordings and graded physical exercise ("stress testing") increases the sensitivity of the ECG in detecting underlying coronary artery disease. This chapter will discuss only clinical scalar electrocardiography, and will focus on the general principles relating features of the ECG to underlying physiologic phenomena. We will cover arrhythmias in moderate detail, and will provide illustrative examples of the ECG correlates of myocardial hypertrophy, ischemia, and infarction.

2. The Normal Electrocardiogram

A typical clinical 12-lead scalar ECG from a normal subject is shown in Figure 1A. It consists of a short sample of ECG (usually one to three beats) from each of the six frontal plane leads and the six precordial leads.

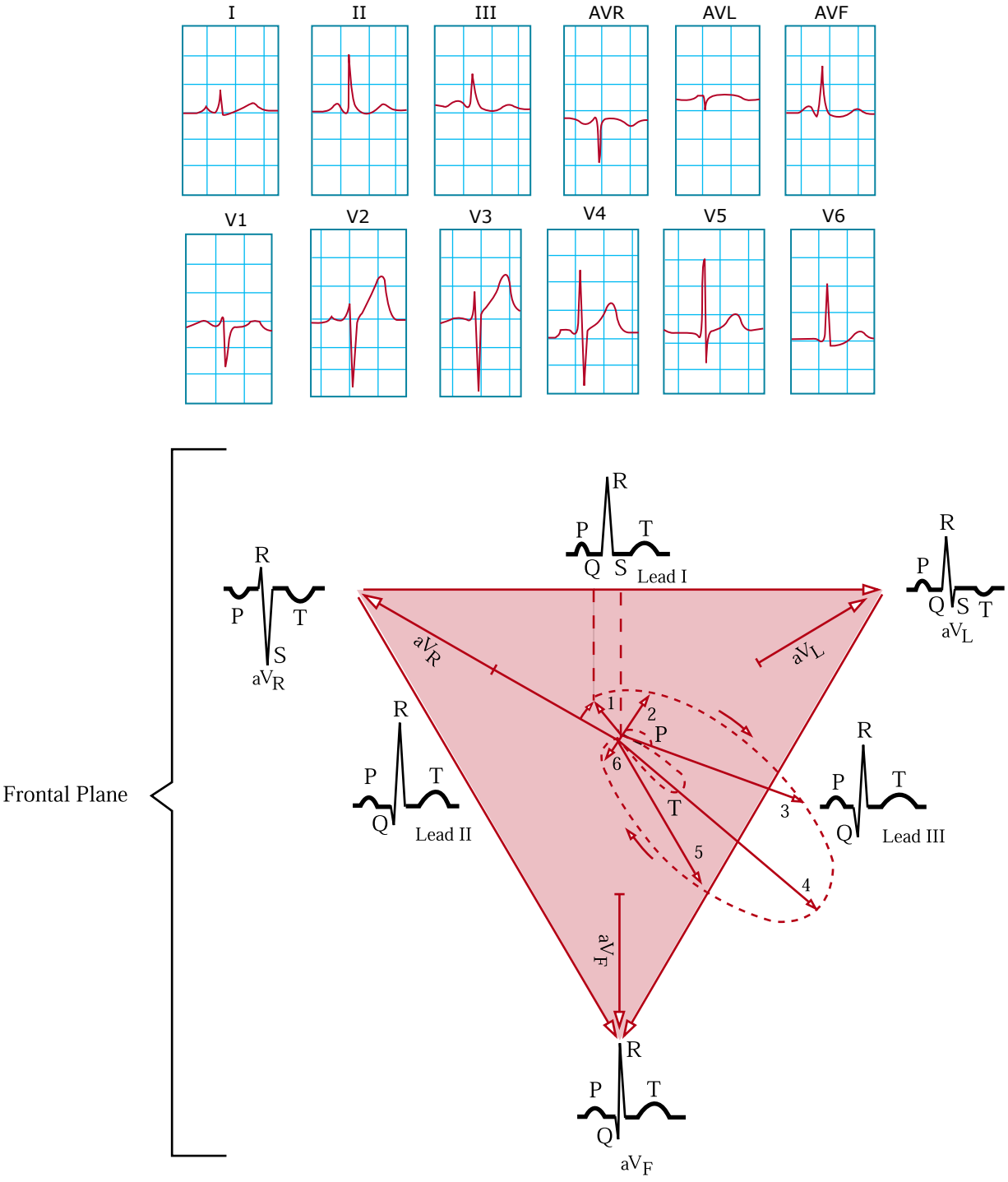
Figure 1B illustrates a normal vectorcardiogram showing the locus of the tip of the heart vector during depolarization of atria (P-loop) and ventricles (QRS loop), and the repolarization of the ventricles (T-loop).

Figure 2 shows one cycle of a typical electrocardiogram in detail, together with a rhythm strip: a longer strip (usually Lead II in order to show clear P-waves) is added to provide information about cardiac rhythm. The conventions regarding the voltage and time calibrations are indicated on the figure. Electrocardiographic equipment is usually calibrated so that the vertical distance between two adjacent lines represents 0.1 millivolt and the horizontal distance between two adjacent lines represents 0.04 seconds. The lines on standard graph paper are one millimeter apart. For example, a one millivolt waveform lasting 0.2 seconds will stand 10 millimeters tall and 5 millimeters wide.

Figure 1 - Normal Electrocardiogram and Vectorcardiogram

A. A normal 12-lead scalar electrocardiogram

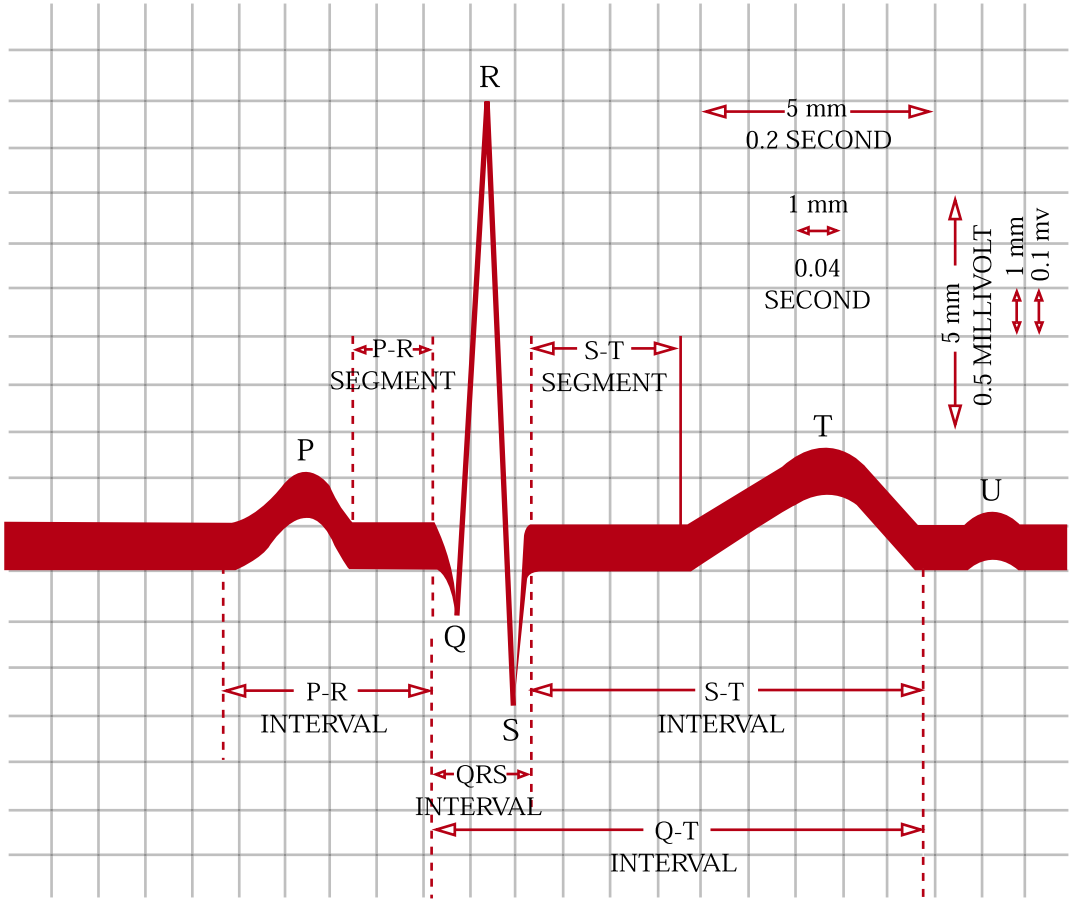
B. A normal vectorcardiogram in the frontal plane, showing also the standard scalar lead vectors.



Normal Vector-Cardiographic Loop and Derivation of the Electrocardiogram in Various Leads

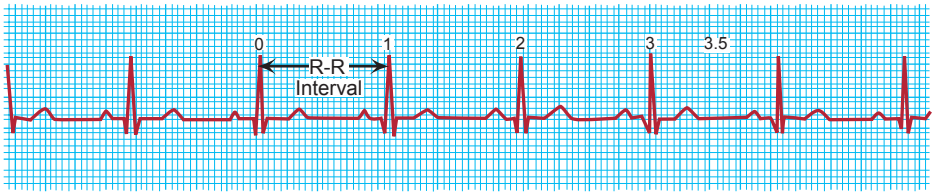
Figure by MIT OCW. After p. 56 (top) in Netter, Frank H. A Compilation of Paintings on the Normal and Pathologic Anatomy and Physiology, Embryology, and Diseases of the Heart, edited by Fredrick F. Yonkman. Vol. 5 of *The Ciba Collection of Medical Illustrations*. Summit, N.J.: Ciba Pharmaceutical Company, 1969.

Figure 2



Recording Conventions, Waveform Nomenclature, and Normal Values for the Electrocardiogram.

		P-R Interval	QRS Interval	Rate	Q-T Interval	S-T Segment
Normal Ranges	Adults	0.18 to 0.20 Second	0.07 to 0.10 Second	60	0.33 to 0.43 Second	0.14 to 0.16 Second
	Children	0.15 to 0.18 Second		70	0.31 to 0.41 Second	0.13 to 0.15 Second
				80	0.29 to 0.38 Second	0.12 to 0.14 Second
	Children			90	0.28 to 0.36 Second	0.11 to 0.13 Second
				100	0.27 to 0.35 Second	0.10 to 0.11 Second
				120	0.25 to 0.32 Second	0.06 to 0.07 Second



Calculation of Rate: Count number of R-R intervals (3.5) in 3 seconds (15 time spaces of 0.2 second each) ↓ Multiply 3.5 by 20 to give rate per minute (70 in this case)

Figures by MIT OCW. After p. 50 in Netter, Frank H. A Compilation of Paintings on the Normal and Pathologic Anatomy and Physiology, Embryology, and Diseases of the Heart, edited by Fredrick F. Yonkman. Vol. 5 of *The Ciba Collection of Medical Illustrations*. Summit, N.J.: Ciba Pharmaceutical Company, 1969.

From left to right the first deflection seen in Figure 2 is labeled a P-wave. It represents the voltage change at the body's surface caused by the depolarization of the atria. Atrial depolarization is usually complete in about 0.1 seconds. Therefore, the P-wave usually spans about 2.5 millimeters. A P-wave typically stands 1-2 millimeters tall (0.1-0.2 millivolts).

The P-wave on the vectorcardiogram is represented by a low-amplitude loop (Fig. 1). The mean axis of the P-wave is +60˚ in the frontal plane and +30˚ in the horizontal plane. Thus, its electrical axis roughly parallels the mean direction of spread of depolarization from the SA node to the AV junction. P-wave amplitude, therefore, will be maximum in Leads II, and aVF.

Once the P-wave is completed, an isoelectric segment follows during which no surface potential is visible using ordinary equipment. During this time the cardiac action potential passes through the AV node and ventricular conduction system. The interval from the beginning of the P-wave to the beginning of the QRS complex is called the PR interval and is normally no longer than 0.2 seconds in adults. Action potentials in the conduction system may be documented only by using catheter electrodes placed within the heart in close proximity to the common bundle of His (see Figure 3).

A wave corresponding to atrial repolarization occurs, but is ordinarily buried in the QRS complex, and is not identifiable in the ECG.

The QRS wave complex follows the PR segment. It represents ventricular depolarization, lasts approximately 80-100 milliseconds, and has an amplitude of 0.5-1.0 millivolts. The QRS vector initially points to the right and anteriorly as septal depolarization is initiated. Then the vector sweeps leftward and posteriorly as the remainder of the ventricular myocardium is depolarized. (See Figure 21 of previous chapter.) The QRS depolarization is much greater in amplitude than the P-wave complex because of the greater muscular mass of the ventricle, and the greater synchronization of depolarization by the high speed ventricular conduction system. The most common mean QRS

axis is between +30 and +90 degrees in the frontal plane and 0 to –30 degrees in the horizontal plane. Thus, the normal mean QRS vector points downward to the left, and posteriorly.

Figure 3 - Bundle of His Recording
His bundle diagram. Note electrophysiologic events in relation to the surface electrocardiogram.

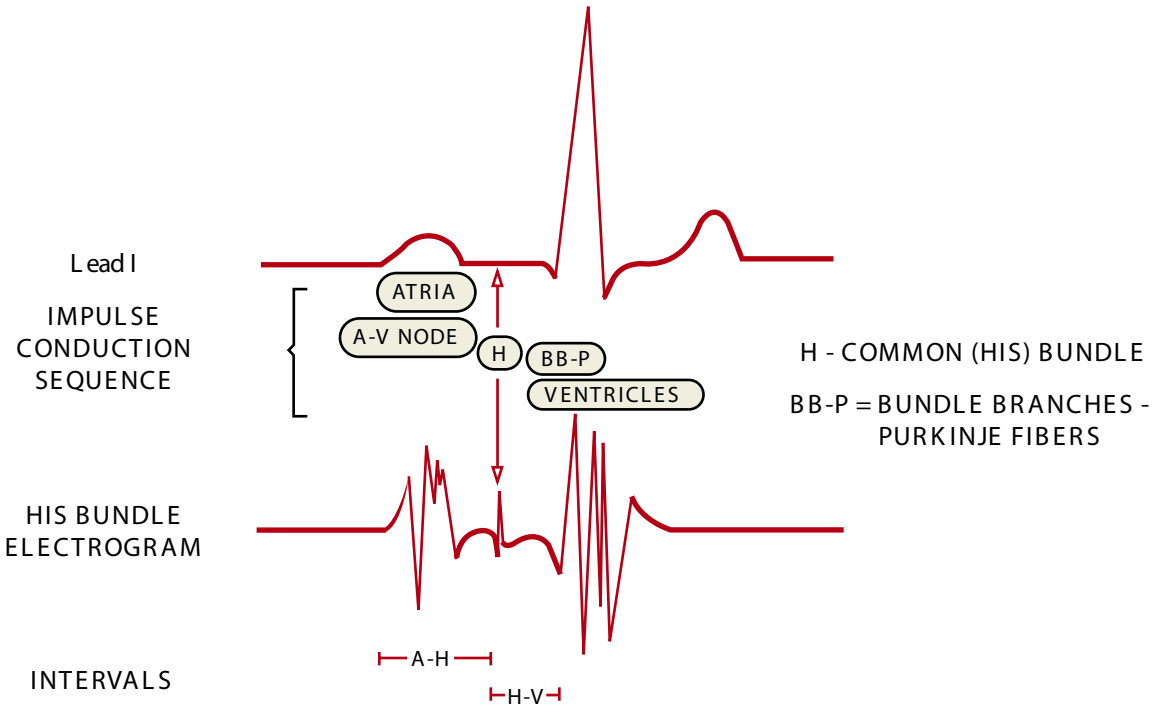


Figure by MIT OCW. After p. 168 in Phillips, Raymond E., and Mary K. Feeney. *The Cardiac Rhythms: A Systematic Approach to Interpretation.* 2nd ed. Philadelphia: W.B. Saunders Company, 1980.

At the completion of the QRS complex, another segment of zero voltage normally follows: the ST segment. It corresponds to the plateau period of the action potential during which the ventricles remain depolarized. It typically lasts 150 milliseconds, but is a function of heart rate. (See Figure 2.)

The T-wave follows the ST-segment, and corresponds to ventricular repolarization. Cellular repolarization is a much slower process than depolarization. Also, repolarization does not appear to

propagate from cell to cell. Rather, individual cells repolarize independently depending on their individual plateau duration. Whereas depolarization spreads from endocardium to epicardium, repolarization normally proceeds in the opposite direction (the action potentials of epicardial cells are shorter than those of endocardial cells). Because of the reversed directions of propagation of repolarization and depolarization, the mean axis of the T-wave loop is roughly parallel to the QRS axis. (See Figure 1.) Thus, in scalar leads showing a primarily positive QRS deflection, the T-wave should also be upright, and vice versa. The T-wave is of smaller amplitude and longer duration than the QRS complex. Normal T-wave duration is 0.15-0.20 seconds, and normal amplitude in the limb leads is less than 0.6 millivolts. The normal angle between the mean QRS vector and the mean T-wave vector is less than 40˚.

The QT interval is measured from the beginning of the QRS complex to the end of the T-wave. The QT interval is used as a measure of the duration of the action potential of ventricular muscle cells, and is a function of the heart rate. (See Figure 2.)

3. Pathophysiology of Arrhythmias

3.1 Introduction

The rhythm of the heart is determined by the generation of an impulse by some pacemaker cell, and the conduction of that impulse to the rest of the heart. Disturbances in cardiac rhythm are a result of abnormalities in impulse initiation, or conduction, or both. There are a number of mechanisms that can cause these abnormalities, and in this section we will review many of them.

3.2 Arrhythmias Caused by Abnormal Impulse Generation

3.2.1 Normal Automaticity

The property of autorhythmicity is shared by cells in the SA node, some parts of the atria, the AV junction, and the ventricular conduction system. In the normal heart, there is a gradient of automaticity as one moves from the SA node down to the Purkinje system. The rate of impulse generation is normally highest at the SA node (70 to 80 per minute), lower in the AV junction (50 to

60 per minute), and lowest (30 to 40 per minute) in the ventricular conduction system. In the normal heart, the rate of impulse formation in the sinus node is sufficiently rapid that potentially automatic cells (latent pacemakers) in other regions of the heart are reset before they reach threshold. Thus, heart rate is established by the fastest pacemaker site.

A number of factors can affect automaticity. The autonomic nervous system has a major influence on the cells of the SA node and AV junction. Vagal stimulation slows the rate of firing, and sympathetic stimulation increases the rate. Increased temperature causes increased rate of firing and vice versa (Fig. 4). Hypoxia and hypercapnia cause an increase in the slope of the phase 4 depolarization, and increase the firing rate of pacemaker cells. Cardiac dilation increases the rate of firing. Automaticity of cells is affected by electrolyte imbalance and myocardial injury, and these mechanisms will be discussed in the next section.

Figure 4 - The Effect of Temperature on Firing Rate of Automatic Cells.
The figure shows pacemaker potentials from sheep Purkinje fibers at different bath temperatures. (From Weidman, 1956.)

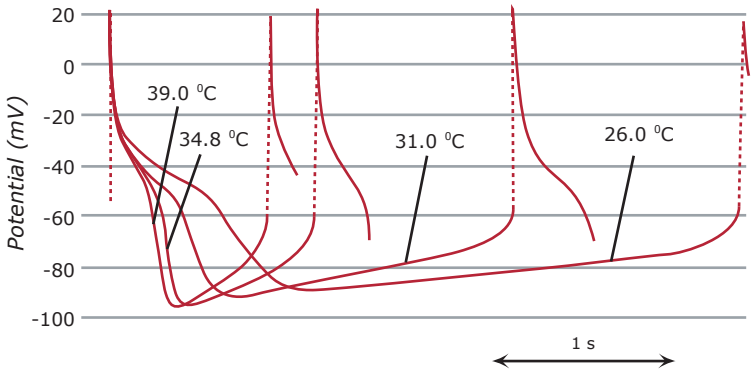


Figure by MIT OCW. After fig. 7.1, p. 86 in Noble, Denis. *The Initiation of the Heartbeat*, 2nd ed. Oxford: Clarendon Press, 1979.

If for some reason, a higher pacemaker center fails to generate an impulse, or if the impulse is not properly conducted, a pacemaker lower in the cardiac conduction system will have time to depolarize to its threshold potential and generate an impulse. Such a beat is called an *escape beat*. A sustained sequence of such beats is called an *escape rhythm*. On the other hand, if a lower pacemaker site prematurely discharges because of local increased automaticity, the resultant beat is called an *ectopic beat*. A series of such beats would thus be an *ectopic rhythm*.

3.2.2 Abnormal Automaticity

Under normal conditions, atrial and ventricular myocardiac cells do not exhibit spontaneous diastolic depolarization. Under abnormal conditions, however, automaticity may be observed in these cells. If such cells are experimentally depolarized to a membrane potential more positive than about –60 millivolts, spontaneous automaticity may occur and cause repetitive impulse generation. This phenomenon is called *abnormal automaticity*. It has also been observed in purkinje cells. Conditions which might lead to such abnormal partial depolarization of cardiac cells might be found in myocardial injury. Severely injured cells (as in myocardial ischemia and/or infarction) would be expected to release large amounts of potassium into the immediate extracellular vicinity of nearby viable cells. This is particularly the case with subendocardial Purkinje fibers which may remain viable even in a zone of infarcted tissue because of oxygen diffusing directly from the intracavitary blood pool. The action potentials generated by such partially depolarized cells often show the slow upstrokes characteristic of the slow inward current (Figure 5a).

Myocardial fibers with low resting potentials will not fire spontaneously if the sinus node drives them faster than their intrinsic rate. The abnormal focus may manifest itself, however, when the sinus rate decreases.

It is possible for these abnormal pacemaker sites to be functionally isolated form the rest of the heart by surrounding tissue which is electrically unexcitable. Action potentials from the normal myocardium are prevented from reaching the independent pacer site (“entrance block”), and similarly, impulses generated by the ectopic focus are unable to propagate into the rest of the heart (“exit block”). Figure 5b demonstrates this phenomenon in an experimental situation. It is also possible to have an abnormal pacer site which can propagate its impulses into the rest of the heart, but which is not reset from the outside because of entrance block. Such a pacemaker site is called a “parasystolic” pacemaker.

Figure 5 - Abnormal Automaticity, Entrance Block and Exit Block in Purkinje Fibers that Survived Myocardial Infarction.

Intracellular recordings of infracted and non-infarcted ventricular tissue from an isolated dog heart after anterior descending coronary artery ligation. A. Comparison of Purkinje fibers in non-infarcted (lower trace) and infracted (upper trace) areas. The infracted fiber exhibits a slow rising low amplitude action potential that results in spontaneous depolarization of the surrounding tissue. B. Non-infarcted Purkinje fibers (traces 1 and 2) artificially stimulated with surface electrodes (left of arrow) are unrelated to depolarizations of an infracted Purkinje fiber (trace 3) indicating entrance block. The lack of unstimulated non-infarcted Purkinje fiber depolarization (traces 1 and 2, right of arrow) despite spontaneous depolarization of the infracted fiber (trace 3) indicates exit block. (After M. E. Josephson, *Ventricular Tachycardia, Mechanisms and Management*. New York: Futura Publishing Co., 1982, p. 59.)

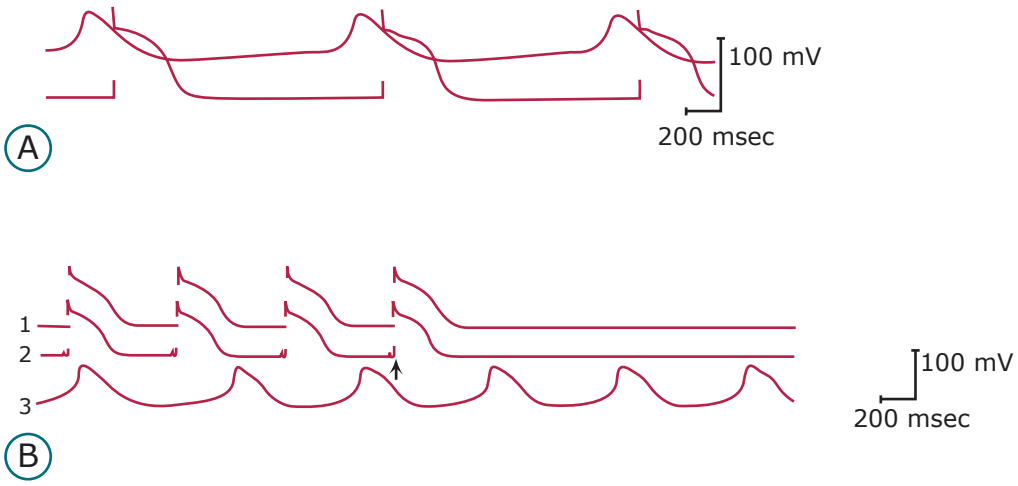


Figure by MIT OCW. After p. 59 Josephson, Mark E. *Ventricular Tachycardia: Mechanisms and Management*. Mount Kisco, N.Y.: Futura Publishing Company, 1975.

Another important mechanism which may lead to abnormal automaticity is the phenomenon know as *triggered activity*. Triggered activity is impulse generation caused by after-depolarization. An after-depolarization is a second subthreshold depolarization that occurs either during the repolarization phase (*early* after-depolarization), or after repolarization is complete or nearly complete (*delayed* after-depolarization).

Early after-depolarizations usually occur during the repolarization phase of an action potential that has been initiated from a high level of membrane potential (usually between –75 and

–90 millivolts). This is illustrated in Figure 6a. Under some conditions, early after-depolarizations can lead to second upstrokes which may reach the threshold potential for activating the slow inward current. This results in a second action potential which occurs prior to the complete repolarization of the first. This second action potential may also be followed by a train of additional action potentials all occurring at the low level of membrane potential characteristic of the plateau or Phase 3 (Figures 6b, 6c).

Figure 6 - Early Afterdepolarizations

A. Example of early depolarization, a depolarizing afterpotential that occurs in the setting of incomplete repolarization and results in absence or delay of normal repolarization.
B. Sustained rhythmic activity from a low resting potential (-60mV) that eventually repolarizes to the original high resting potential (-90mV).
C. Abnormal automaticity resulting from artificially decreased membrane potentials -40mV (trace 1), -45mV (trace 2), and -30mV (trace 3). The amount of automatic activity increases as membrane potential is reduced. (After Wit A L, Friedman P F. Bases for ventricular arrhythmias accompanying myocardial infarction. *Arch. Intern. Med.* 1975; **135**: 459.)

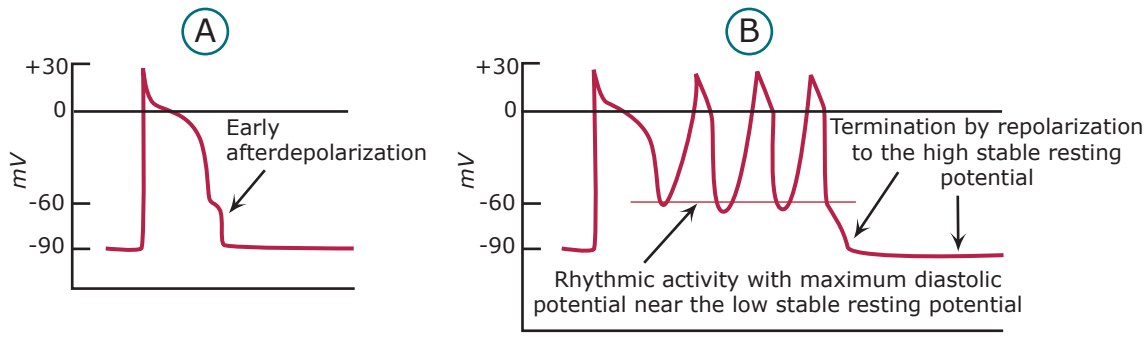


Figure by MIT OCW. After fig. 1-8, p. 12 and fig. 1-15, p. 17 in Cranefield, Paul F., and Ronald S. Aronson. *Cardiac Arrhythmias: The Role of Triggered Activity and Other Mechanisms*. Mount Kisco, N.Y.: Futura Publishing Company, 1988.

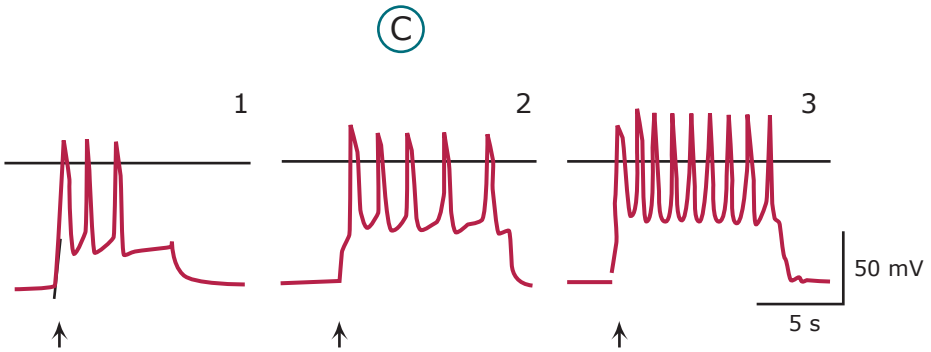


Figure by MIT OCW. After fig. 20.5, p. 808 in Macfarlane, Peter W., and T.D. Veitch Lawrie, ed. *Comprehensive Electrocardiology: Theory and Practice in Health and Disease.* vol. 2. New York: Pergamon Press, 1989.

Early after-depolarizations which lead to triggered activity in isolated cardiac preparations may be caused by factors which are present *in vivo* under some pathologic conditions. These factors include hypoxia, high pCO_2, and high concentrations of catecholamines. Since these conditions may be present in an ischemic or infarcted region of the ventricles, it is conceivable that early after-depolarizations may cause some of the arrhythmias that occur soon after myocardial ischemia. It also has been suggested that mechanical injury may predispose to early after-depolarization. Mechanical injury might occur clinically in the area of an infarct or aneurysm.

Delayed after-depolarizations are transient or oscillatory depolarizations which occur immediately after the terminal repolarization of an action potential. Delayed after-depolarizations are illustrated in Figure 7. This phenomenon is observed under a number of conditions in which there is a significant increase in the intracellular calcium. One of the most widely recognized causes is digitalis toxicity. Cardiac glycosides inhibit the sodium-potassium pump, thus leading to an increase in intracellular sodium. The intracellular sodium is then probably extruded from the cell in exchange for calcium by a sodium/calcium exchange mechanism. Catecholamines can cause delayed after-depolarizations. In addition, they have sometimes been observed in the presence of low membrane potentials.

Delayed after-depolarizations may not reach threshold, in which case triggered activity does not occur. The amplitude of the delayed after-depolarization tends to increase as the rate of firing of the cell is increased, or as the coupling interval of a premature stimulation is decreased. (See Figure 8.) Once the delayed after-depolarization reaches threshold, a prolonged train of triggered impulses may result. Triggered activity can be terminated by either premature or overdrive electrical stimulation.

Figure 7 - Delayed Afterdepolarizations

After depolarizations and triggering in coronary sinus fibers A) Stimulated action potentials (horizontal line) lead to progressively increasing in amplitude afterdepolarizations and eventual triggering of rapid sustained rhythmic activity (end of horizontal line). During rhythmic activity, membrane potential, action potential amplitude and action potential upstroke velocity decrease. Rhythmic activity is shown at a more rapid sweep speed (right). The last action potential is followed by afterdepolarization and then quiescence. Eventually, the membrane potential then returns to the level present prior to triggering. B) The effect of decreasing stimulus cycle length on afterdepolarization amplitude and triggering. At left of panel, fiber was stimulated at cycle length of 1500 msec (underlined by middle horizontal bar). Afterdepolarization following last driven impulse had an amplitude of 17 mV. At a cycle length of 1200 msec, triggering of sustained rhythmic activity is observed, with decrease in action potential amplitude. (After A. L. Wit and P. F. Cranefield, "Triggered and automatic activity in the canine coronary sinus." *Circ Res* 41:435, 1977.)

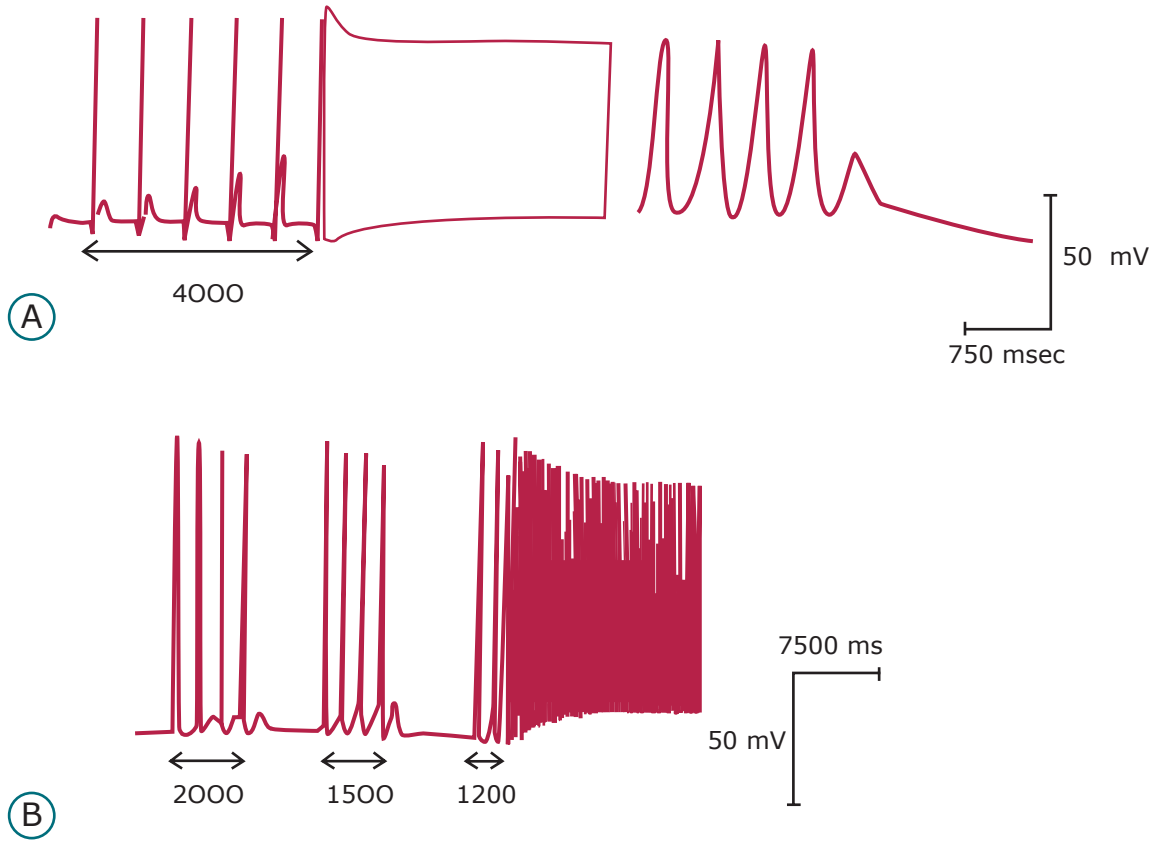


Figure by MIT OCW. After fig. 26-11, p. 840 in Hurst...

Figure 8 - Delayed Afterdepolarizations

Effect of premature stimulation on afterdepolarization amplitude and triggering. Premature impulses (arrows) were generated after a series of 10 impulses driven at 4000ms. Coupling intervals between pulse train and premature premature impulse generation was progressively decreased from trace A to trace C resulting in increases in impulse magnitude and induction of triggered activity in trace C.

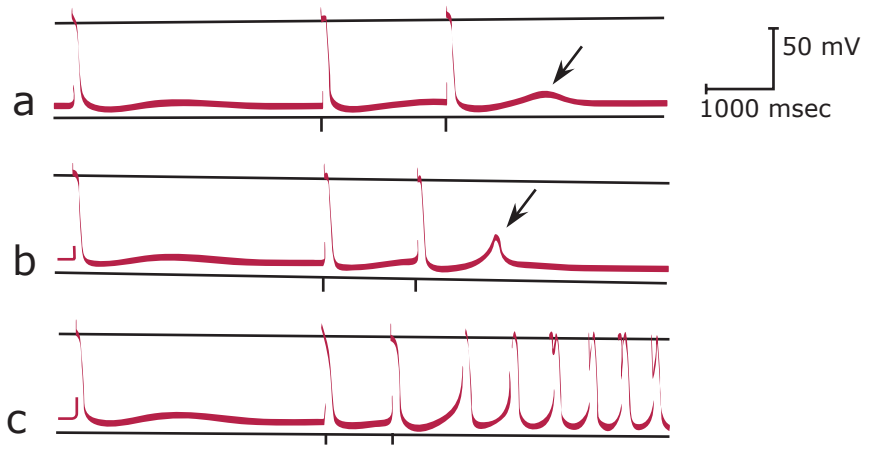


Figure by MIT OCW. After p. 1464 in Fozzard...

3.3 Abnormal Impulse Conduction

3.3.1 Decremental Conduction and Block

A number of arrhythmias are caused by failure of the cardiac impulse to propagate properly throughout the heart. Some disease processes, such as infarction and fibrosis, may permanently interrupt portions of the cardiac conduction system. The result may be a fixed barrier to conduction in one of the major bundle branches (bundle branch block), or even complete electrical separation of the atria and ventricles (complete heart block).

More commonly, functional changes in the electrophysiological properties of the conduction system modify impulse conduction. Slowing of conduction may occur in any part of the conduction system, and is referred to as “decremental conduction”. (See Figure 9.) It results from partial inactivation of the fast sodium channels. If this current is sufficiently inactivated, cells my be left with only calcium channels to support the action potential which then propagates quite slowly.

Decremental conduction may occasionally lead to complete failure of conduction. Sometimes the block is one-way, preventing conduction in one direction, but not the other.

Figure 9 - Decremental Conduction

Effect of decremental conduction on action potential shape. When impulses being transmitted through cardiac muscle reach an area of decremental conduction (shaded), the action potential magnitude is decreased and the upstroke is slowed. If the impulse is transmitted, the normal action potential is regenerated, but arrives delayed. (After A. Katz, *Physiology of the Heart*, Raven Press.)

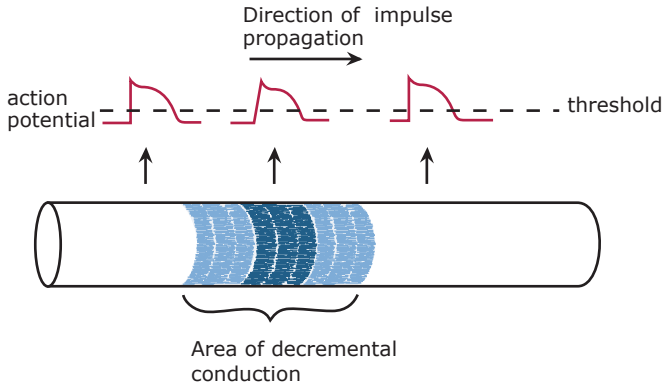


Figure by MIT OCW. After fig. 21.4, p. 523 in Katz, Arnold M. *Physiology of the Heart.* 2nd ed. New York: Raven Press, 1992.

The velocity of conduction of a cardiac action potential is determined by several factors including:

1) the amplitude and rate of rise of the phase 0 depolarizing current;

2) The radius of the conducting fiber; and

3) the internal resistance, r_i, of the myocardial bundle.

Decreasing the amplitude or the rise-time of the action potential slows conduction velocity. Such changes may result from partial depolarization of the cell as may occur in areas of myocardial injury where the extracellular potassium concentration rises. Certain drugs, particularly some anti-arrhythmics, slow the rate of rise of the action potential by partially inhibiting the fast inward sodium current.

The velocity of propagation is proportional to the square root of the fiber radius. The very slow conduction velocity in the AV node may thus be explained on the basis of both the slow rise-

time of the action potential and also the small cell size. Furthermore, in regions of the SA node and AV node the density of tight junctions (nexi) is reduced, with the effect of increasing the longitudinal intracellular resistance, r_i. This change also tends to slow impulse conduction.

A decrease in conduction velocity with eventual block may occur when an impulse arrives at cells that have not completely recovered excitability from a previously conducted action potential. An action potential triggered during the relative refractory phase, for example, will have a lower amplitude and slower rise time than normal. This results in slow propagation and possible block. (For example, if atrial premature beats occur early enough, they may be blocked at the level of the AV node and never depolarize the ventricle.) Since the refractory period of the right bundle branch is typically longer than that of the left bundle, some atrial premature beats which are successfully propagated across the AV node may be blocked in the right bundle branch resulting in a QRS complex which is abnormally shaped. This phenomenon is called “aberrant conduction.” Although most common in the right bundle, it may also occur in the left bundle branch.

3.3.2 Reentry

In the presence of slow conduction and/or unidirectional block, it is possible to establish in the myocardium a so-called “reentrant loop” of excitation. There are many possible geometric arrangements for such loops, which may exist in many locations of the heart. In all configurations, however, it is required that an action potential pathway exist such that the wave-front of activation returns to previously excited tissue after a delay long enough to permit that tissue to have recovered its excitability. Some workers have subdivided the reentrant mechanism into two categories, “random reentry” and “ordered reentry.” Random reentry is most associated with atrial or ventricular fibrillation, whereas ordered reentry can cause most other types of arrhythmias. The main distinction between the two is that during random reentry, propagation occurs over reentrant pathways that continuously change their size and location with time. Ordered reentry, on the other hand, implies a relatively fixed reentrant pathway. Despite the differences, similar prerequisite electrophysiologic conditions are required for either kind of reentrant excitation. The wave-length of

the impulse in the reentrant circuit (conduction velocity × refractory period) must be shorter than the length of the circuit, so that the tissue into which the impulse is reentering has had time to recover excitability. Because of this requirement, it is clear that the relationship among path length, conduction velocity, and refractory period is crucial. Reentry can be promoted by slowing conduction velocity, by shortening the refractory period, or by a combination of both.

Reentry may occur in a variety of geometric configurations such as loops of Purkinje fiber bundles in a distal conduction system, bundles of surviving muscle fibers in healed myocardial infarcts, or in fibrotic regions of the atria or ventricles (Figure 10).

Figure 10 - Re-entry

Possible mechanisms for peripheral purkinje system reentry. The left panel shows a main bundle of Purkinje fibers (MB) dividing into two branches (A and B), before terminating on ventricular muscle (VM). Unidirectional antegrade conduction block (hatched) occurs in a severely depressed area of branch B. Action potentials from each region are shown below. Action potential I in the main bundle trace (MB) corresponds to the antegrade impulse conducted from the main bundle (arrow I in the diagram) to both branches, A and B. Conduction is blocked in B at the solid black line, but continues through A (see action potential in trace A) into the ventricular muscle. The impulse also continues into B in a retrograde direction where it conducts back through the area of unidirectional block in B (see action potential in trace B) and reenters the main bundle (arrow II in the diagram and action potential II in trace MB). The bottom of the panel shows how such events might appear on the ECG. QRS I corresponds to antegrade propagation of impulse I into VM and QRS II corresponds to the reentrant premature depolarization after retrograde propagation of the reentrant impulse II. The right panel shows two parallel fibers in an unbranched bundle of Purkinje fibers. The entire shaded area is depressed with the more severe unidirectional conduction block indicated by the darker area. Action potentials can only propagate from left to right in the bottom fiber. The impulse traverses the top fiber in the reverse direction, re-entering the bottom fiber again to cause a second depolarization.

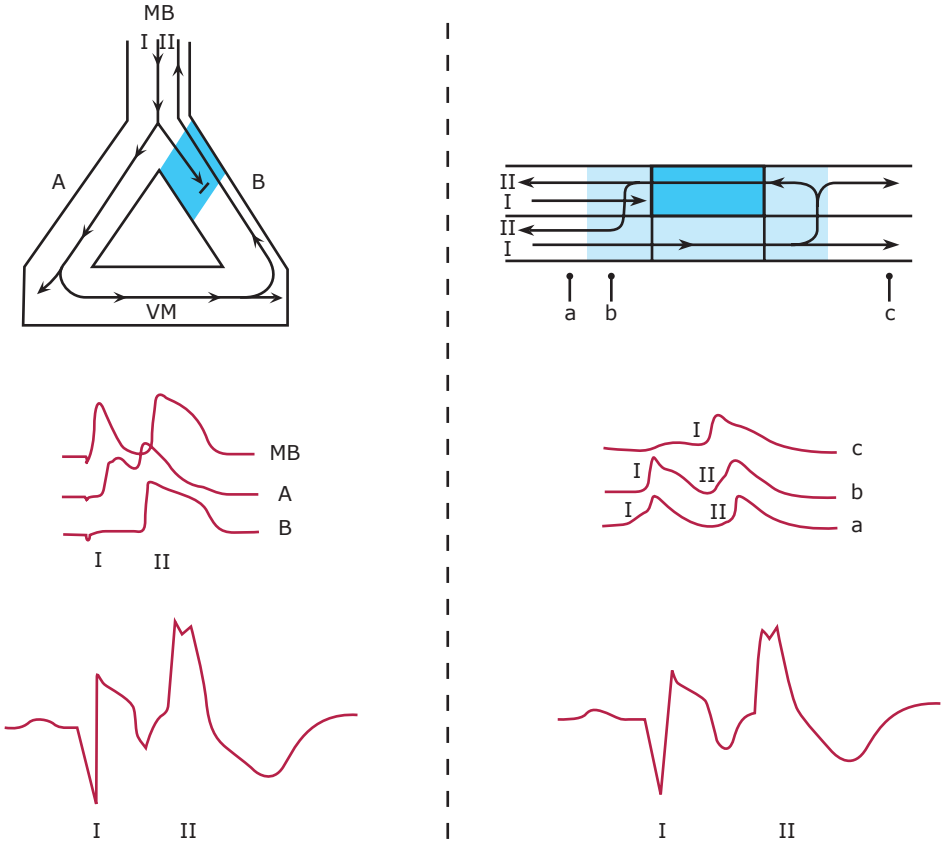


Figure by MIT OCW. After p. 48 in Josephson, Mark E. *Ventricular Tachycardia: Mechanisms and Management.* Mount Kisco, N.Y.: Futura Publishing Company, 1975.

Reentry may be supported in the AV node, if it is functionally dissociated into two parallel conduction bundles with different properties (Fig. 11a, b). The action potential may propagate down one parallel pathway while being unidirectionally blocked in the other. If the propagation times are slow enough, a reflected wave of depolarization may traverse back up the previously blocked

fascicle to institute a reentrant beat. This mechanism is thought to be responsible for many supraventricular arrhythmias.

Figure 11 - Mechanism of AV Nodal Re-entry

Mechanism of paroxysmal supraventricular tachycardia due to AV nodal reentry. PSVT due to AV nodal reentry results from dual AV nodal pathways having different conduction properties and refractory periods.

A: During normal sinus rhythm in the presence of dual AV nodal pathway, the fast pathway (generally having a longer refractory period) is primarily responsible for AV transmission.

B: A premature atrial impulse is blocked by the the fast pathway due to its longer refractory period, and therefore propagates down the slow pathway. As a result, the PR interval is prolonged and retrograde invasion of the fast pathway can occur.

C: Echo beats or AV nodal reentrant tachycardia occur when the temporal relationship between slow pathway conduction and recovery of fast pathway excitability allow the impulse to reenter the slow pathway after retrograde fast pathway transmission. The atria can also be activated by retrograde conduction. In a much less common form of AV nodal reentry, a shorter refractory period in the fast pathway reverses the loop, with antegrade conduction through the fast pathway and retrograde conduction up the slow pathway. Radio frequency ablation therapy is often applied to the slow pathway at the site indicated by the asterisk (*).

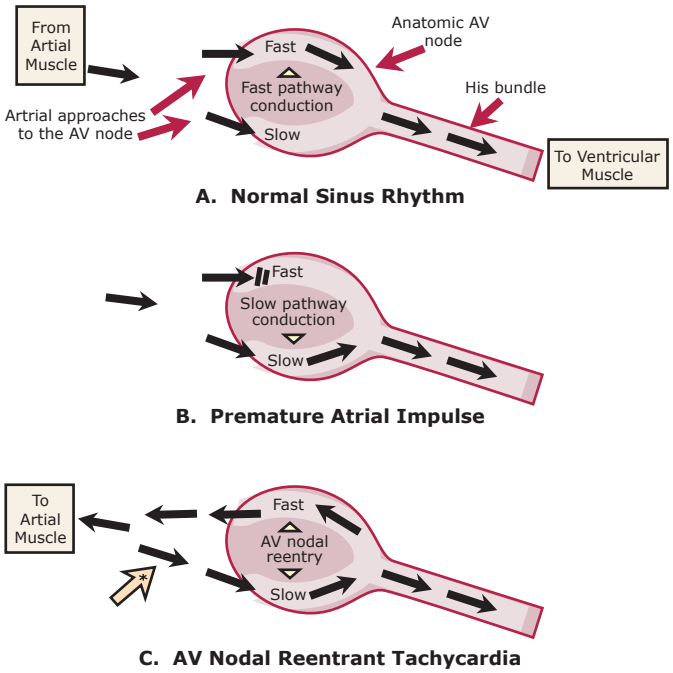


Figure by MIT OCW. After fig. 27-11, p. 886 in Hurst...

Patients with so-called “pre-excitation syndrome” (or Wolff-Parkinson-White syndrome) have abnormal accessory pathways of conduction which connect the atria and ventricles in parallel with the AV node (Fig. 12a). Such patients are likely to develop reentrant loops composed of the

AV node and the accessory pathway (Fig. 12b). (This mechanism is felt to be the cause of the high incidence of supraventricular arrhythmias in these patients.) The impulse may travel from atrium to ventricle via the AVN, and then back retrogradely to the atrium via the accessory pathway (orthodromic conduction). Alternatively, the impulse may travel rapidly form atrium to ventricle via the accessory connection, and then travel retrogradely back to the atrium via the AV node (antidromic conduction).

Figure 12 - Bypass Tracts and Re-entry

A. Composite diagram of four possible pathways for anomalous conduction. They may produce ventricular pre-excitation and be responsible for reentrant tachycardias. Bypass tracts: A, atrioventricular (Kent); B, atrio-His (James); C, Intranodal; D, nodoventricular (Mahaim).

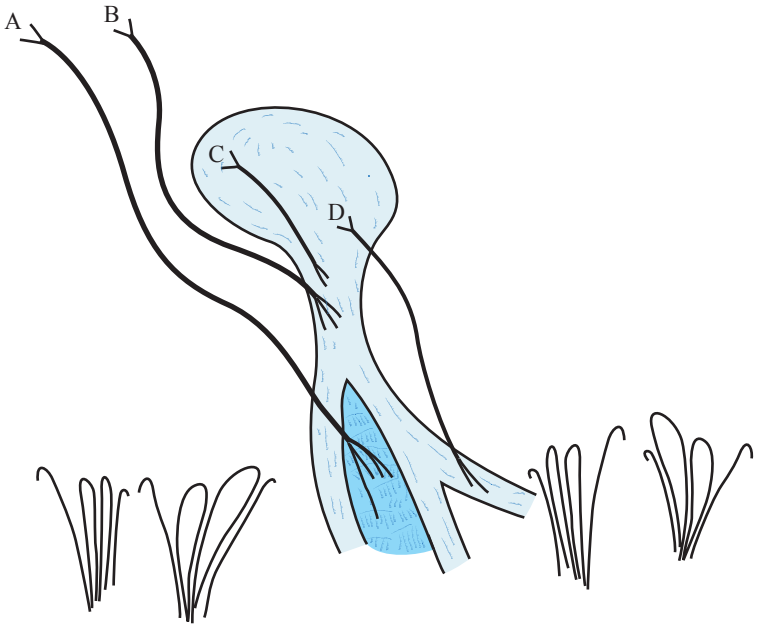


Figure by MIT OCW. After fig. 5-8, p. 174 in Phillips, Raymond E., and Mary K. Feeney. *The Cardiac Rhythms: A Systematic Approach to Interpretation.* 2nd ed. Philadelphia: W.B. Saunders Company, 1980.

B. Reentry phenomenon involving a bypass tract

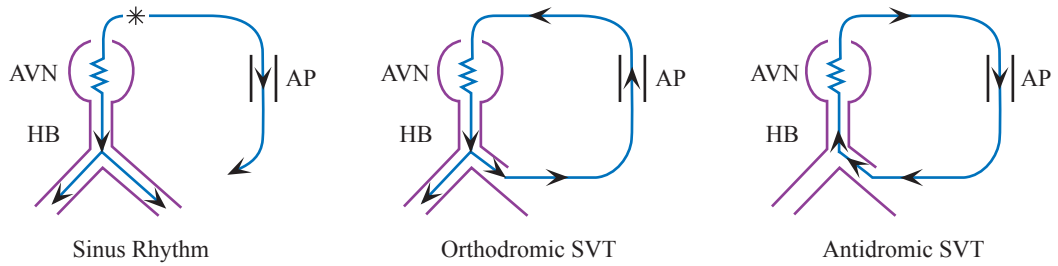


Figure by MIT OCW. After fig. 10-1 p. 191 in El-Sherif, Nabil, and Philip Samet. *Cardiac Pacing and Electrophysiology.* 3rd ed. Philadelphia: Harcourt Brace Jovanovich, Inc.,W.B. Saunders Company, 1991.

Reentrant excitation may also result from the anisotropic structure of cardiac muscle. Its anatomic and biophysical properties vary according to the direction of measurement. For example, conduction velocities in a direction parallel to the myocardial fiber orientation are more rapid than in a direction perpendicular to the long axis. Additional anisotropic behavior is introduced in the presence of diffuse fibrosis, or regional ischemia. It has been demonstrated that such anisotropic properties can lead to reentrant loops (Fig. 13). It is this type of milieu which could lead to the development of the chaotic electrical activity of ventricular fibrillation.

Figure 13 - Epicardial activation maps 3.5 min after coronary artery occlusion in the porcine heart

Unipolar electrograms recorded from the ischemic and non-ischemic zones of the right ventricle are shown (top). The activation maps during the last basic beat (B on the electrogram recordings and left activation map), and the first two impulses of a rapid ventricular tachycardia (impulses 1 and 2 on the electrogram recordings and middle and right activation maps) culminate in ventricular fibrillation. Activation map isochrones show the time each region is activated. Arrows indicate the direction and pattern of impulse conduction. (From M. E. Josephson, *Ventricular Tachycardia Mechanisms and Management*. New York: Futura Publishing Co., 1982. p. 45.)

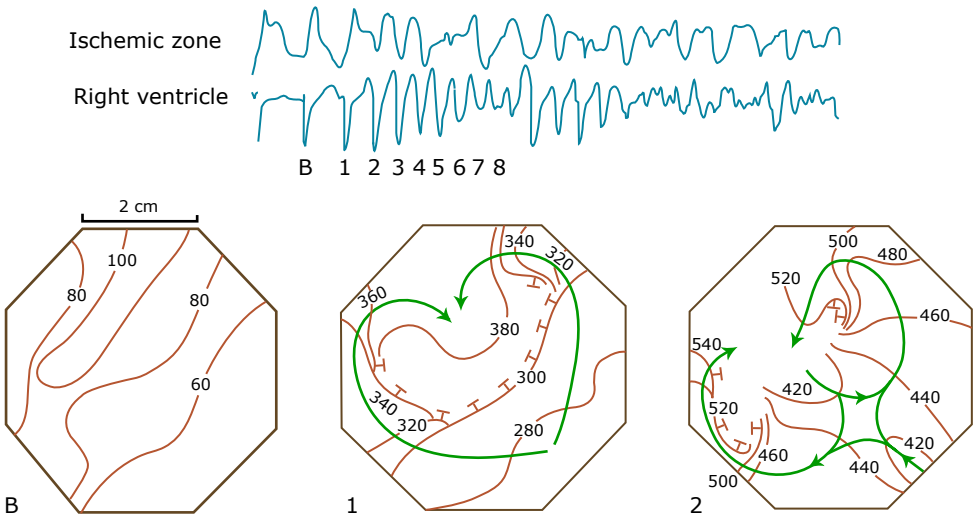


Figure by MIT OCW.

4. Clinical Examples of Arrhythmias

4.1 An Approach to ECG Rhythm Analysis

In this section we will present a number of examples of arrhythmias. In analyzing the clinical electrocardiogram for rhythm, it is important to use a systematic approach.

First, identify the QRS complexes. The following observations should be made:

1) What is the ventricular rate?

2) Are the QRS complexes spaced at regular intervals? If not, what is the nature of the irregularity?

3) Are the QRS complexes identical in shape in a given lead? Are they of normal size and morphology?

Next, identify the P-waves. In some cases this will require careful observation, and more than one lead axis may be necessary. The following questions should be explored:

1) Is there a one-to-one relationship between P-waves and QRS complexes? If not, is there a definable pattern?

2) Is the PR interval of normal duration?

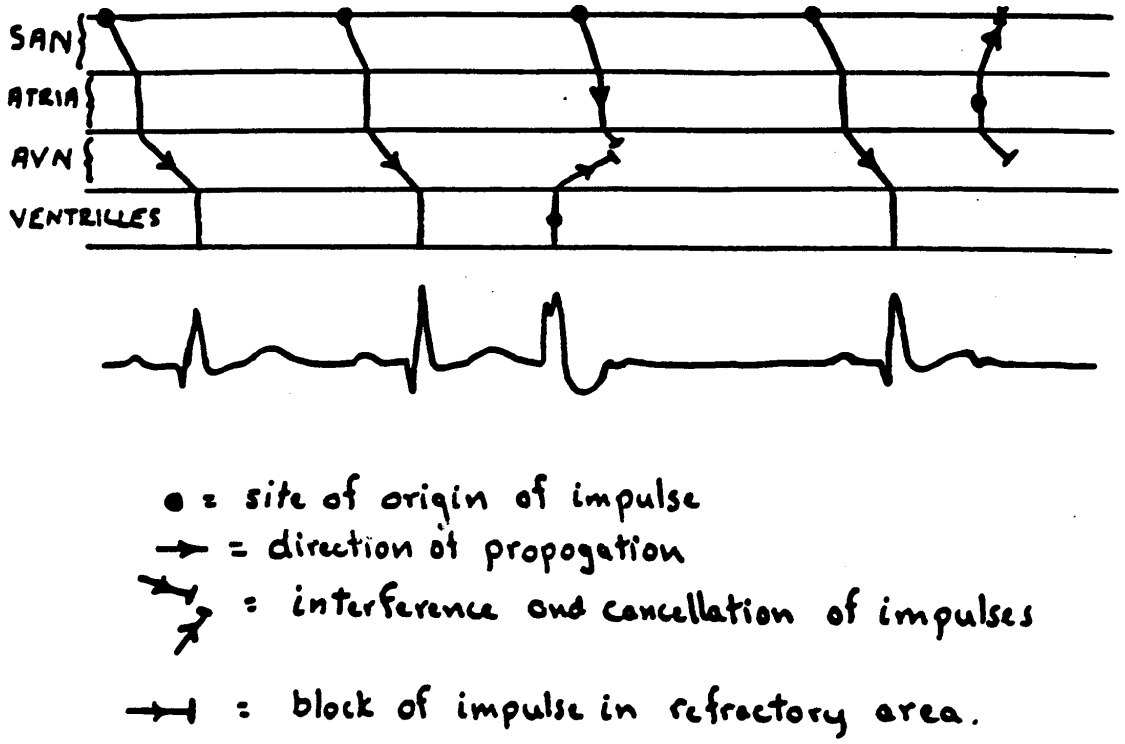
3) What is the atrial rate?

4) Are the P-waves identical in shape in a given lead? Are they of normal size and shape?

Based on the above analysis, it should be possible to identify the mechanism of the rhythm in most cases. The so-called “ladder diagram” is a very useful method for representing the detailed rhythm, including the site of impulse origin and its propagation pathway through the heart. Figure 14 illustrates the technique. The vertical axis of the diagram is divided into four separate regions

corresponding to different sections of the cardiac conduction system. The top space represents the region of the SA node, the second space is the atria, the third space is the AV junction and ventricular conduction system, and fourth space represents the ventricles. The horizontal axis represents time. Electrical activation of the heart is represented by tracing the propagation pathway vertically as shown in the figure. Arrows indicate direction of propagation, and a dot indicates the point of origin of the depolarization.

Figure 14 - Ladder Diagram



The figure illustrates several electrophysiologic phenomena. The first two beats are normal sinus beats. Electrical activity begins in the SA node, and progagates slowly until it exits into the atrial tissue. Atrial depolarization is represented as an instantaneous process (vertical line) to reflect the notion that it is essentially an “all-or-none” phenomenon. The impulse travels slowly through the AV junction (sloped line) and depolarizes the ventricles (again represented by a vertical line). The third beat in the figure is abnormal. The electrical activity originates in two sites. The ventricular depolarization originates from a site in the ventricles and spreads throughout these structures and in

a retrograde direction through the AV junction. In the meantime, the SA node fired, the impulse depolarized the atria, and also invaded the AV junction. The two impulses collide in the AV junction and "destructively interfere" since each meets refractory tissue in its path. The fourth beat is a normal one. The fifth beat represents electrical activity originating from somewhere in the atria and spreading to both the SA node (where it resets the pacemaker) and to the AV junction, where it fails to propagate (perhaps because the tissue was still refractory from a previous beat, or because of disease). This phenomenon is termed "AV block".

By extension of these principles virtually any arrhythmia may be represented on the ladder diagrams.

4.2 Arrhythmias Due to Disturbances of Impulse Formation

4.2.1 Normal Sinus Rhythm and Variants

Normal sinus rhythm is characterized by a regular cardiac rate with normal QRS complexes whose duration must be less than 100 milliseconds (Fig. 15). The P-waves are normal in shape, and are synchronized with the QRS complexes. The PR interval must be less than 0.2 seconds. Heart rates may range from 60-100 bpm.

There are a number of variant types of sinus rhythm. "Sinus tachycardia" (Fig. 16) refers to rates above 100 bpm. "Sinus bradycardia" means heart rates less than 60 (Fig. 17). "Sinus arrhythmia" (Fig. 18) is a normal rhythm in which heart rate varies periodically, usually with the respiratory cycle. There is an acceleration of rate during inspiration, and a slowing of rate during expiration. A "sinus pause" refers to a P-P interval which is clearly longer than the usual, but less than 3 seconds. A sinus pause extending beyond three seconds is generally referred to as "sinus arrest".

Figure 15 - Normal Sinus Rhythm—Rate 85

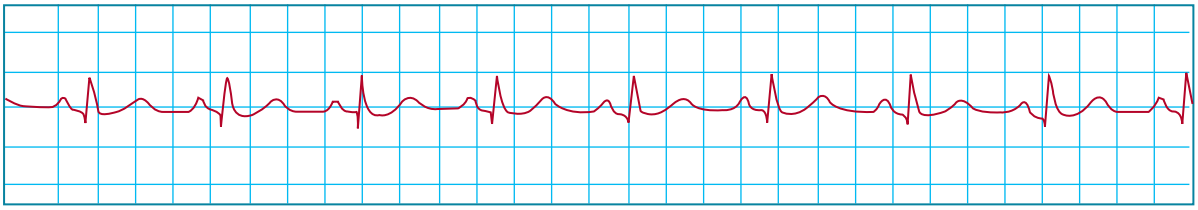


Figure by MIT OCW.

Figure 16 - Sinus Tachycardia—Rate 122

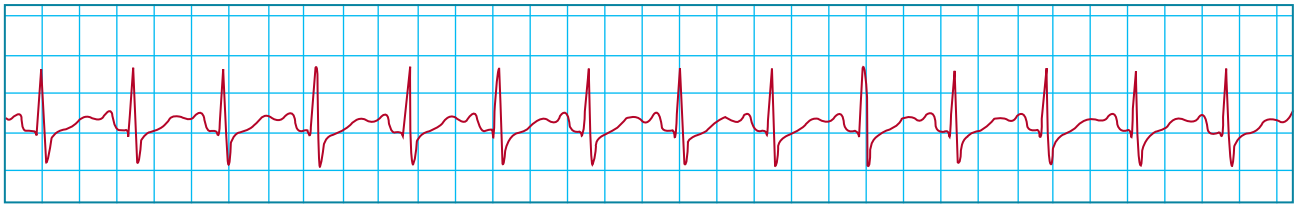


Figure by MIT OCW.

Figure 17 - Sinus Bradycardia—Rate 48

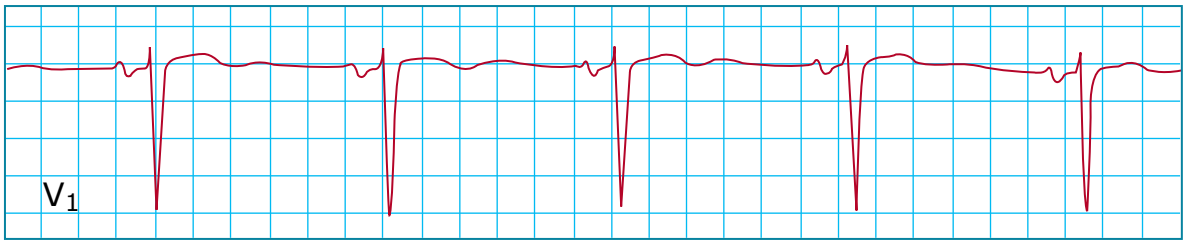


Figure by MIT OCW.

Figure 18 - Sinus Arrhythmia

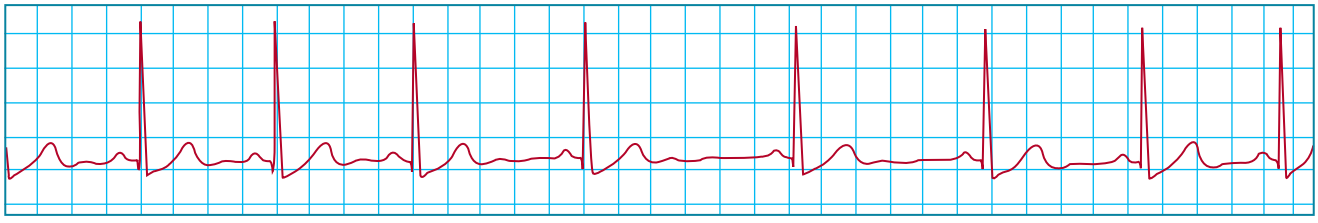


Figure by MIT OCW.

4.2.2 Escape Beats and Rhythms

Escape beats arise from lower (normally latent) pacemakers outside of the sinus node that fire because of either depressed sinus node function or blocked conduction of sinus impulses. Escape beats may originate at any pacemaker site below the sinus node.

Atrial Escape Beats

If the SA node slows sufficiently (perhaps due to vagal tone), other latent pacemaker sites in the atrium may emerge to establish heart rate. The P-wave resulting from these beats is usually different in shape from the normal, and in many cases is inverted in polarity. This reflects the fact that the beats originate low in the atrium. Such beats are sometimes referred to as “low atrial” or “coronary sinus” beats (or rhythms if sustained). (See Figs. 19, 20.)

Figure 19 - Atrial Escape Beat

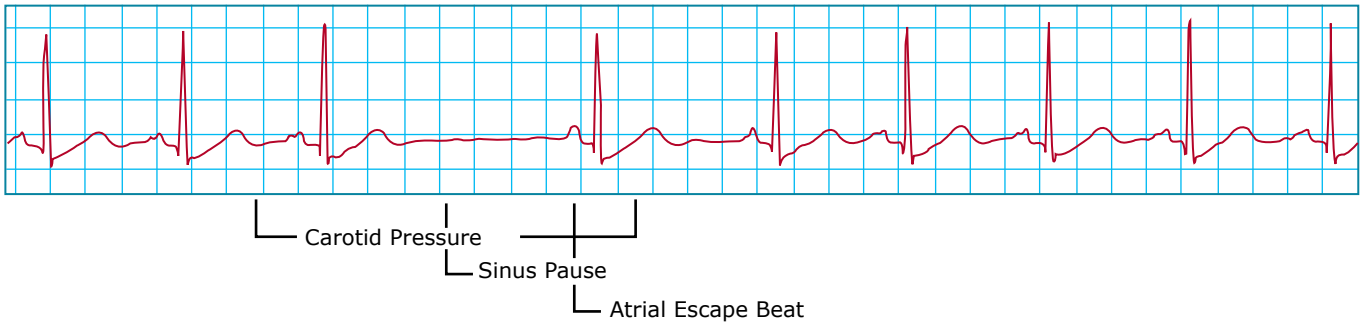


Figure 20 - Atrial Escape Rhythm Emerges as Sinus Rate Slows

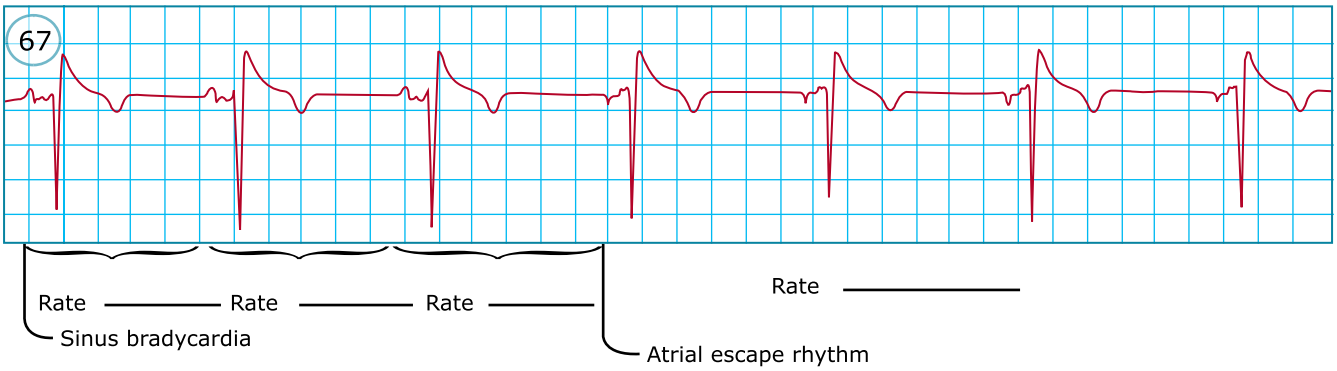


Figure by MIT OCW.

AV Nodal Escape Beats

AV-nodal escape beats often terminate prolonged sinus pauses. The QRS complex is normal because the impulse is conducted normally to the ventricles (Fig. 21). The P-wave is either not visible at all, or may be found just prior to or immediately following the QRS. In general the P-wave is abnormal in shape since it is retrogradely conducted. If the P-wave immediately precedes the QRS complex, the beat is referred to as a "high nodal beat". Conversely, if the P-wave follows the QRS, the beat is called a "low nodal beat". A sequence of nodal beats makes up a nodal rhythm (Figs. 22, 23). The rhythm is be called an escape rhythm if its rate is clearly less than that of the usual sinus rate.

Figure 21 - Nodal Escape Beats.
The last two beats are nodal escape beats which appear as sinus pacemaker slows.

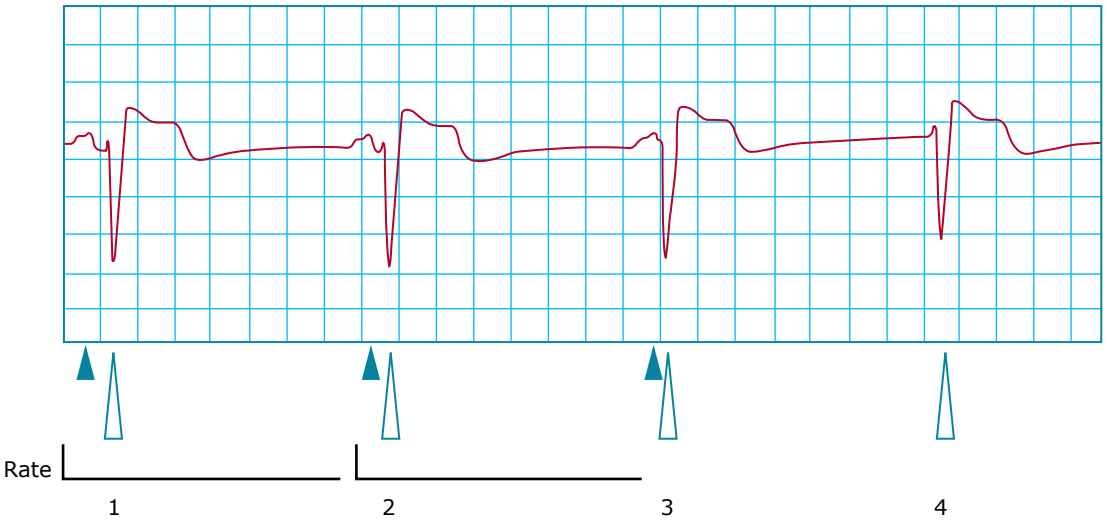


Figure by MIT OCW.

Figure 22 - Nodal Rhythm in Complete AV Block

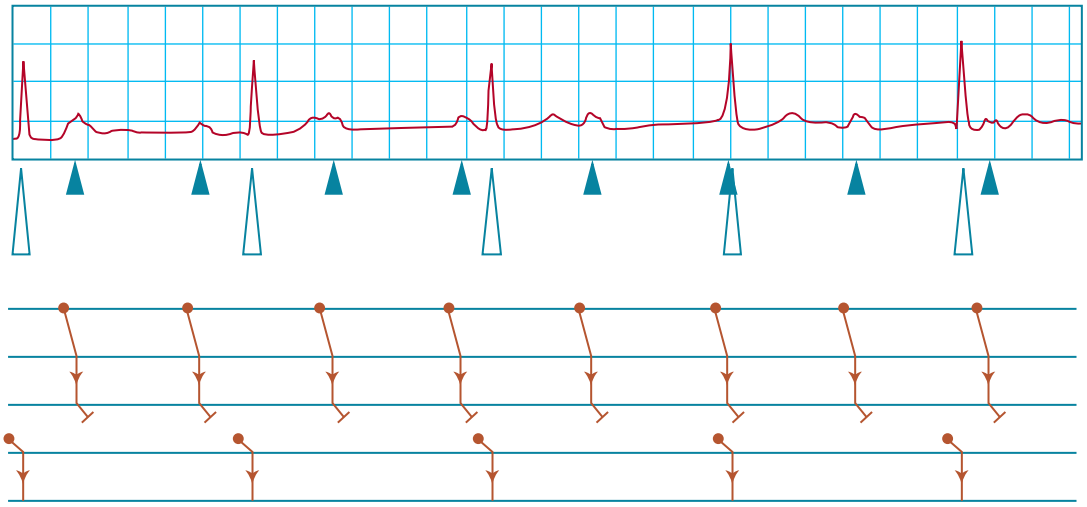


Figure by MIT OCW.

Figure 23 - Low Nodal Rhythm

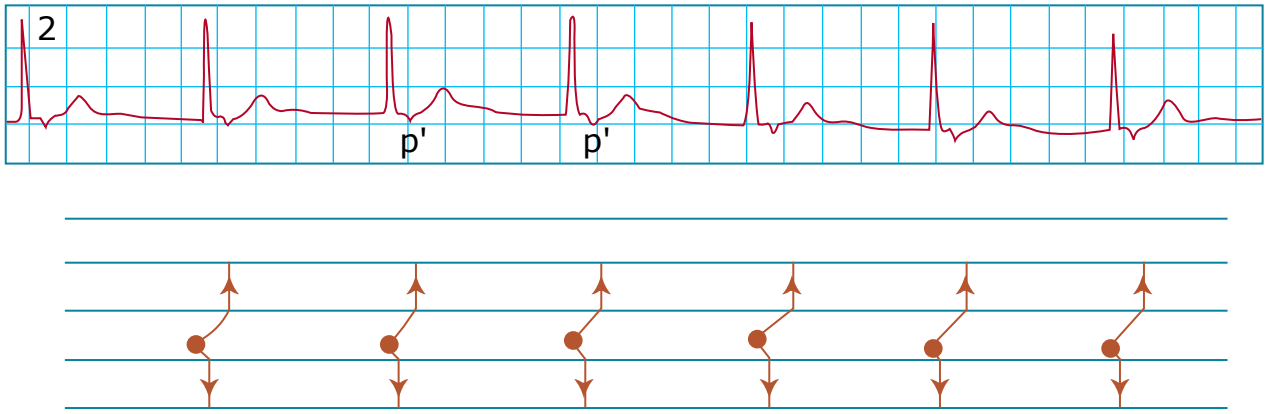


Figure by MIT OCW.

Ventricular Escape Beats

Ventricular escape beats protect the heart against asystole in the event of AV block (either fixed or transitory). They are characterized by a wide and usually bizarre QRS complex. The cardiac impulse originates in the ventricular Purkinje system. It is generally conducted with a slow propagation speed (0.5 meter/second) through the myocardium, thus leading to a wide QRS complex (usually greater than 120 msec) (Fig. 24). Ventricular escape rhythms (idioventricular rhythms) are common in cases of complete heart block, and have rates of about 40 per minute. (See Fig. 51 below.)

4.2.3 Ectopic Beats and Rhythms

Ectopic beats arise from pacemakers outside the sinus node as a result of an abnormal increase in rhythmicity.

Atrial Premature Beats (APBs)

APBs are seen frequently in normal individuals and have little clinical significance. They are also seen in heart disease, and when frequent, may be an early sign of atrial irritability which may progress to more serious atrial dysrhythmias. In APBs the QRS complexes are normal since they

propagate normally through the ventricles via the conduction system (Fig. 25). The P-waves are generally slightly abnormal since they originate from an abnormal focus, and propagate in an abnormal pattern. The impulse generally invades the area of the SA node and resets the sinus pacemaker.

APBs occurring quite early following the previous beat may be aberrantly conducted, frequently with a right bundle branch block configuration. Aberrant conduction is particularly likely when the APB follows a long RR interval (the Ashman phenomenon) (Fig. 26). If an APB is extremely early it may run into refractory tissue in the AV node and be non-conducted (Fig. 27).

Paroxysmal Atrial Tachycardia (PAT)

This is an absolutely regular supraventricular tachycardia with heart rates which range between 120 and 220 bpm (fig. 28). The QRS complexes are normal, although on some occasions aberrant conduction simulates ventricular tachycardia. (The differential diagnosis of supraventricular tachycardia with wide QRS complexes from ventricular tachycardia may be extremely difficult.) P-waves may be slightly abnormal, since they are arising from an ectopic focus. The PR interval may be normal or prolonged. If the atrial rate is quite high, it is likely that some atrial beats will be non-conducted, resulting in 2:1, 3:1, or 4:1 AV block. The mechanism of PAT and other supraventricular tachy-arrhythmias is probably a reentrant loop involving the AV junction

PAT may often be seen in otherwise normal individuals. It also occurs in patients with heart disease, including coronary artery disease, valvular disease and thyrotoxicosis. The attacks come suddenly, may last for seconds, minutes, hours or even days. If the heart rate is high enough, congestive heart failure may ensue, particularly if there is concomitant structural heart disease. The treatment is to slow conduction through the AV node. This may be accomplished by increasing vagal tone by means of carotid sinus pressure, or by using drugs such as digitalis or calcium channel blocking agents such as diltiazens or verapamil.

Figure 24 - Ventricular Escape Beat

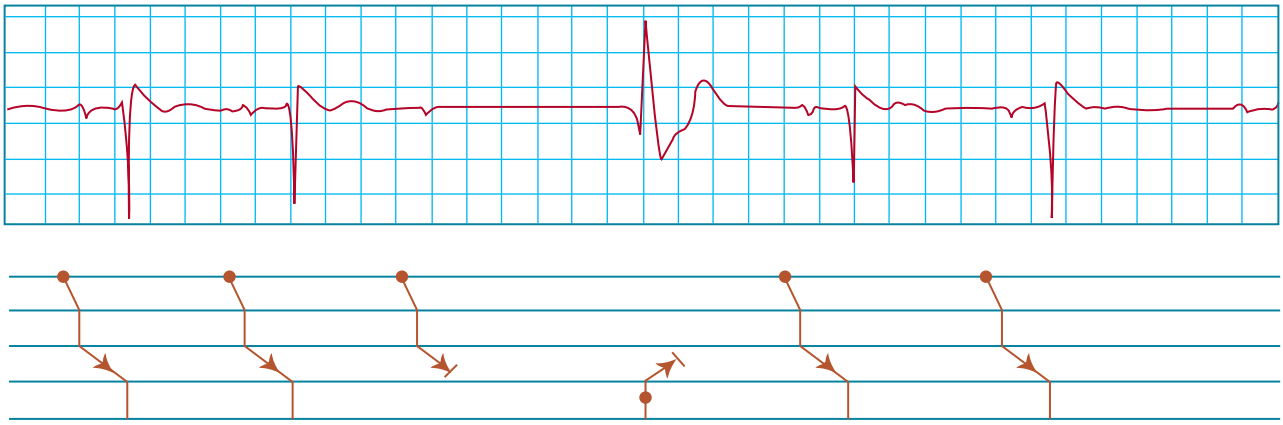


Figure by MIT OCW.

Figure 25 - Atrial Premature Contractions

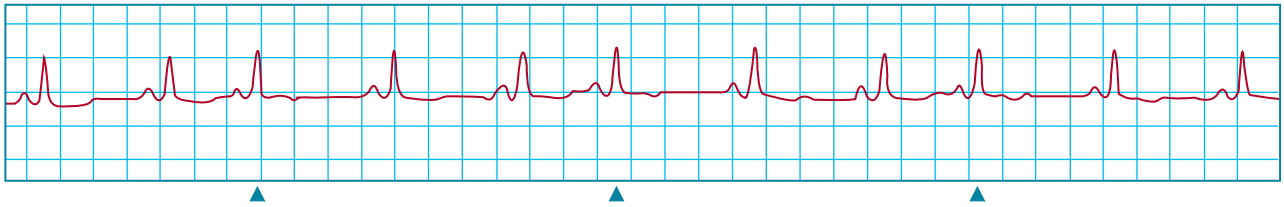


Figure by MIT OCW.

Figure 26 - Aberrantly Conducted APBs (Ashman Phenomenon)

Figure by MIT OCW.

Figure 27 - Non-conducted (Blocked) APBs

Figure by MIT OCW.

Figure 28 - Atrial Tachycardia

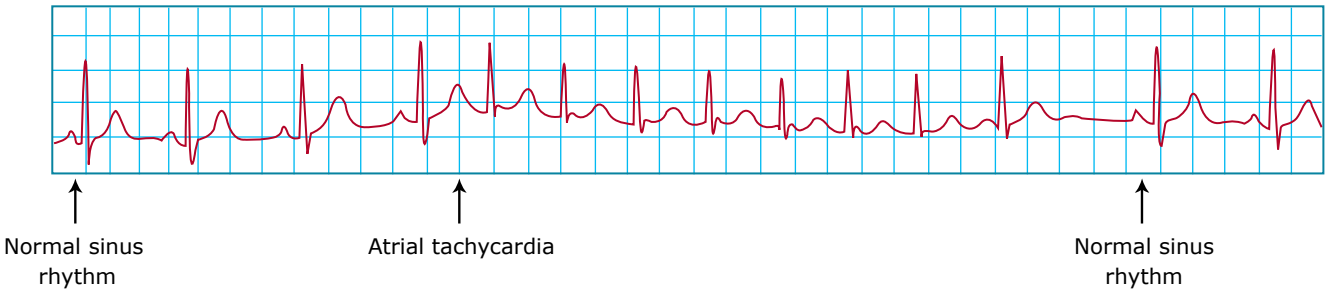


Figure by MIT OCW.

AV Junctional Premature Contractions

Nodal, or AV junctional premature contractions arise from an ectopic focus in the AV nodal area. The impulse is generally conducted in two directions simultaneously. Retrograde conduction activates the atria (with abnormal P-waves) and antegrade conduction activates the ventricles (normal QRS). The P-wave may precede (high nodal)(Fig. 29) or follow (low nodal) the QRS (Fig. 23). Frequently, no P-wave is seen, either because retrograde conduction is blocked or because the P-wave falls in the midst of the QRS complex (Fig. 30).

"AV junctional tachycardia" is produced by sustained activity of the ectopic junctional pacemaker. Since the intrinsic rate of nodal automaticity is 40-60 beats/minute, an "accelerated

junctional rhythm" is considered at rates above 60 (Fig. 31). AV junctional tachycardia usually refers to rates above 100 (although there is no universal agreement on criteria).

The mechanism of junctional tachyarrhythmias may well be reentry in the AV node, a possible manifestation of functional longitudinal dissociation (recall Figure 11b).

AV junctional tachycardia may occur as a paroxysmal arrhythmia in otherwise normal individuals. Nodal tachycardia may also be a manifestation of digitalis toxicity, since that drug increases automaticity in the AV junctional tissue.

At very rapid rates it is often impossible to differentiate sinus tachycardia, atrial tachycardia, and AV junctional tachycardia, since the P-waves and T-waves tend to blend together indistinguishable. When a definite diagnosis cannot be made from the surface ECG, the term "supraventricular tachycardia" is used (Figure 32).

Figure 29 - High Junctional Premature Beat

Figure by MIT OCW.

Figure 30 - AV Junctional Premature Contraction (no P-wave)

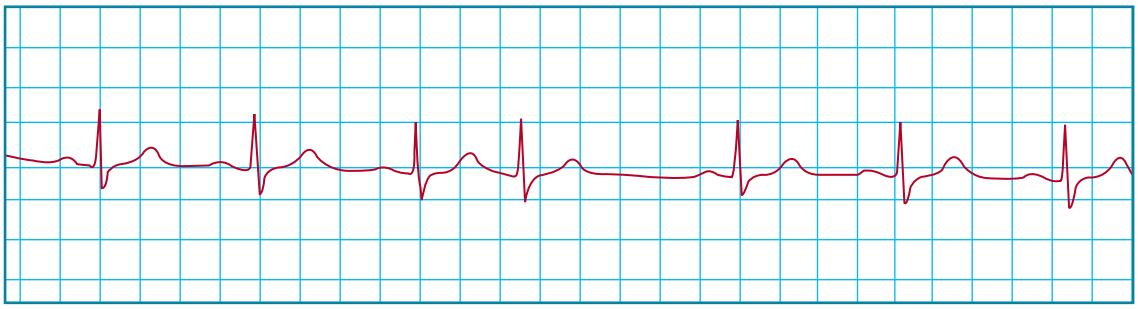


Figure by MIT OCW.

Figure 31 - Accelerated Junctional Rhythm

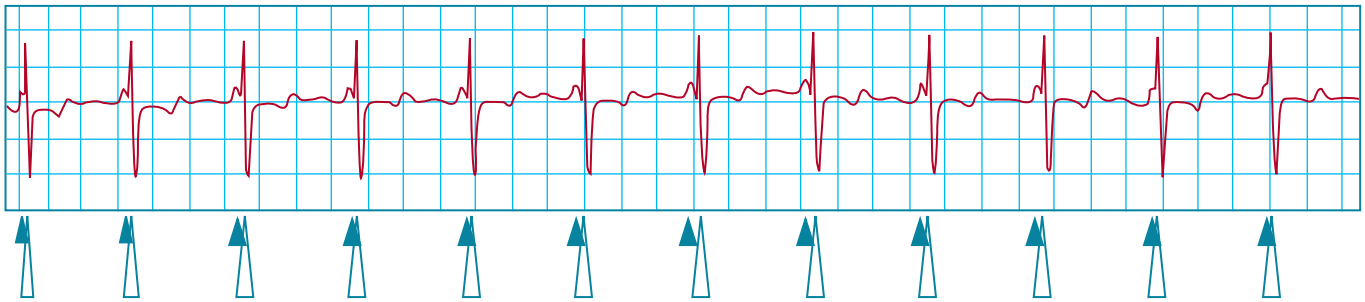


Figure by MIT OCW.

Figure 32 - Supraventricular Tachycardia

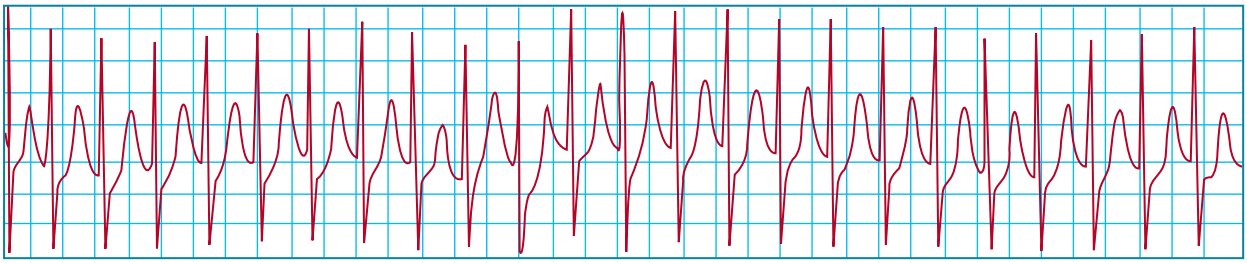


Figure by MIT OCW.

Atrial Flutter

In atrial flutter, there is a regular, rapid atrial rhythm at a rate of 220-330 bpm. The most common atrial rate is about 300 bpm. There will always be some AV block, generally 2:1 but on some occasions 4:1 or variable AV block may be seen (Figs. 33 and 34). Hence, the ventricular rate may be regular or irregular. The QRS complex is usually normal. In leads II and AVF one frequently sees the characteristic saw-tooth pattern of the atrial depolarizations. The mechanism of this arrhythmia is likely a reentrant mechanism in the atrium.

This arrhythmia is usually associated with underlying heart disease such as coronary artery disease, rheumatic valve disease, cardiomyopathy, etc. It is a difficult rhythm to eradicate. It

sometimes responds to drugs such as quinidine, procainamide, verapamil, or amiodarone; but often electrical cardioversion is required. Chronic recalcitrant, symptomatic atrial flutter may be managed with RF catheter ablation with some success. The rhythm is usually tolerated well as long as the ventricular response rate can be kept below about 100 bpm.

Atrial Fibrillation

In this arrhythmia, the atrial activity is chaotic and very rapid. The AV node is bombarded with a high frequency of atrial impulses. These impulses are transmitted through the node in a probabilistic manner leading to "irregularly irregular" ventricular responses at rates which may vary widely. Ventricular response rates in untreated atrial fibrillation may range from as low as 50 to as high as 200 bpm depending upon the state of function of the AV junction (Fig. 35). The QRS complexes are generally normal in morphology. However, aberrant conduction does occur, particularly with very short R-R intervals. The P-waves are not present as discrete depolarizations. In some cases, a small irregular oscillation of the baseline may be seen, but in other cases the baseline appears flat.

This is a common arrhythmia clinically, and is usually associated with underlying heart disease. Rapid ventricular response rates may lead to decreased cardiac output, angina, or congestive heart failure.

Treatment is usually focused on increasing the degree of AV block to slow the ventricular response rate. Drugs such as digitalis or calcium-channel blockers (verapamil or diltiazem) will accomplish this objective. Conversion of the rhythm to normal sinus is desirable whenever possible, and may be attempted using either drugs or electric cardioversion. More agressive approaches include implantable atrial defibrillators, RF ablation procedures, or ablation of the AV junction with permanent cardiac pacing.

Ventricular Premature Beats (VPBs)

These ectopic beats originate from somewhere in the ventricles. The QRS complex is wide (greater than 0.12 seconds) and bizarre (Fig. 36). VPBs may exhibit fixed coupling to previous normal beats. They may occur early or late in the cycle.

The mechanism for PVCs may be reentry or triggered activity as discussed previously. Some VPBs appear to show no fixed coupling to preceding normal beats. If they show a regular rhythm of their own, they may result from a parasystolic focus (Fig. 37). Note that some parasystolic depolarizations experience “exit block” and do not result in ventricular excitation. Parasystolic ventricular ectopic beats are usually considered relatively benign.

Most VPBs are followed by a pause. The pause is usually compensatory—meaning that the coupling interval to the preceding normal beat plus the pause following the VPB comprise an interval equal to twice the normal R-R interval (Fig. 38). An interpolated VPB is one which is sandwiched between two normal QRS complexes which arrive on time (Fig. 39).

Figure 33 - Atrial Flutter (atrial rate—300)

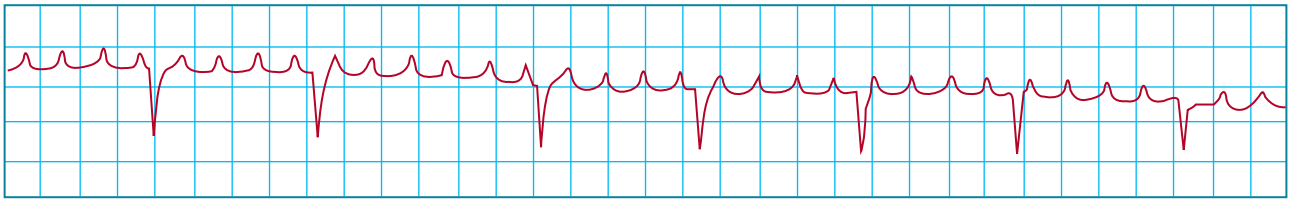


Figure by MIT OCW.

Figure 34 - Atrial Flutter (2:1 conduction, atrial rate—300)

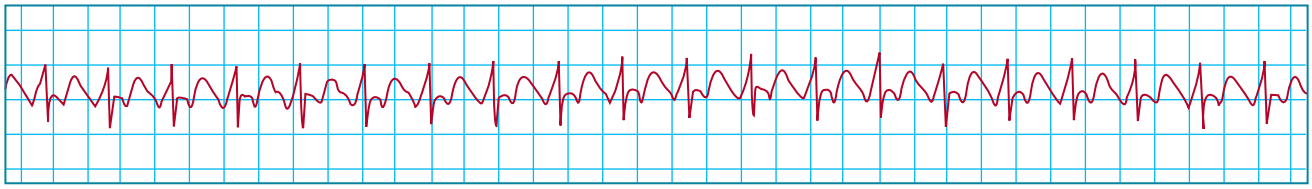


Figure by MIT OCW.

Figure 35 - Atrial Fibrillation (2 examples)

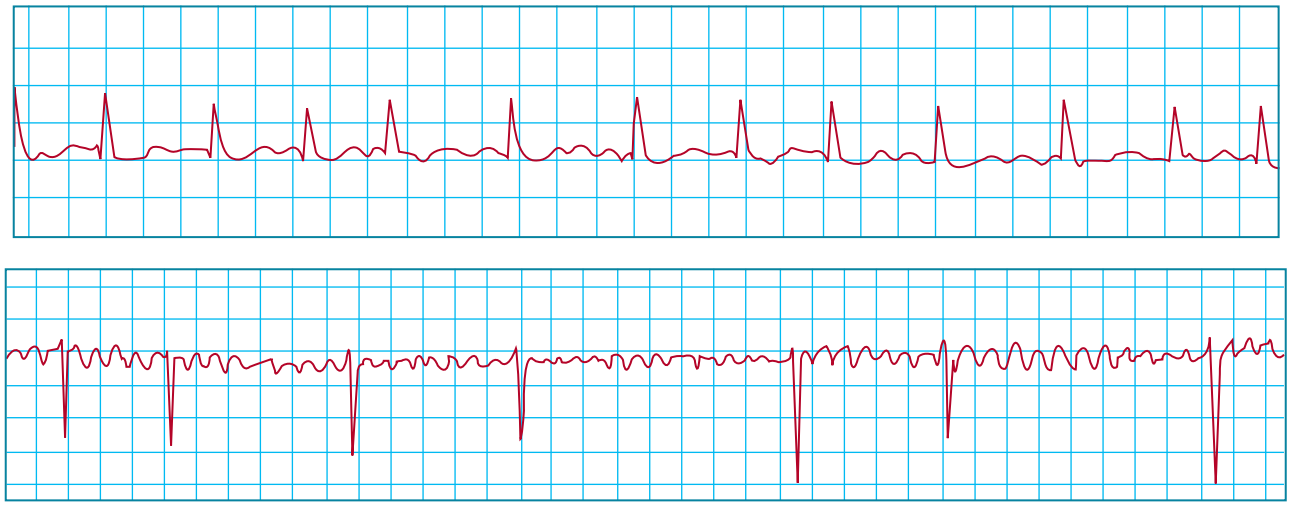


Figure by MIT OCW.

Figure 36 - Ventricular Premature Contractions

Figure 37 - Apparent and Concealed VPBs in Parasystole

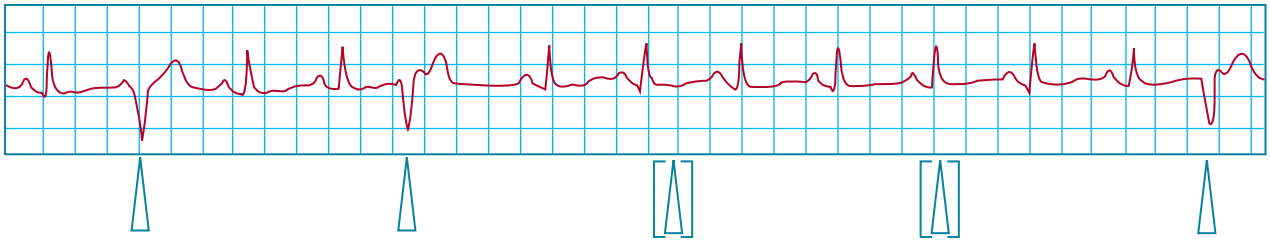


Figure by MIT OCW.

Figure 38 - Compensatory Pause Following VPB

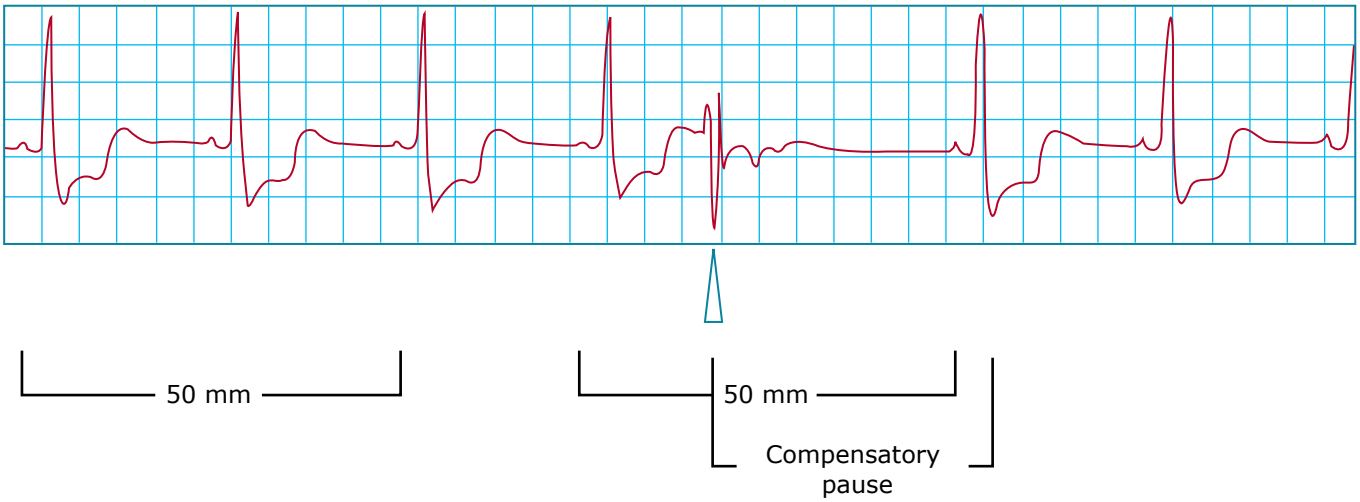


Figure by MIT OCW.

VPBs are often found in otherwise normal individuals and probably have little significance if they are infrequent. In heart disease, VPBs may be a risk factor for increased incidence of more serious ventricular arrhythmias and sudden death. VPBs may occur singly or in groups and the following ordering of increasing severity of ventricular ectopic activity has been proposed:

Occasional:	less than 30 per hour VPBs of the same morphology (Fig. 36)
Frequent:	greater than 30 per hour uniform VPBs or bigeminy where every other beat is a VPB (Fig. 40)
Multiform PVCs:	different QRS morphologies (Fig. 41)
Couplets:	pairs of consecutive VPBs (Fig. 42)
Ventricular Tachycardia:	runs of three or more VPBs (Fig. 43)
Ventricular Flutter:	rapid ventricular tachycardia with a sinusoidal configuration caused by merging of QRSs and Ts (Fig. 44)
Ventricular Fibrillation	chaotic electrical activity without definite QRS complexes (Fig. 45)

VPBs which occur very early in the cardiac cycle such that they fall on the T-wave of the previous beat are considered particularly dangerous. At the time corresponding to the peak of the T-wave, the ventricular myocardium is just beginning to repolarize. Some cells may be in the relatively refractory period, while others may be more fully recovered, and still others quite refractory. The electrical properties of the myocardium are thus quite varied, and conditions favoring reentrant loops are likely. Thus, an extra stimulus in the form of an isolated VPB which is very early-cycle may trigger a repetitive ventricular ectopic rhythm such as ventricular tachycardia or ventricular fibrillation. (The period near the T-wave peak is often referred to as the “vulnerable period”.) Figure 46 shows example of an early cycle “R-on-T” VPB initiating ventricular tachycardia.

Proper characterization of ventricular ectopic activity requires long-term (24-hour) ECG monitoring. This is usually accomplished with small battery-operated tape recorders. The ECG is recorded throughout the patient’s normal daily activities. The tape is scanned at high speed after the recording period, and summary reports are prepared. New technology has led to considerable automation in the scanning process, and has even made possible real-time arrhythmia analysis using portable microprocessors.

The treatment for ventricular ectopic activity is generally restricted to patients who have demonstrated structural heart disease, or patients who have major symptoms (episodes of light-headedness or syncope). Suppression of VPBs in individuals with otherwise normal hearts is generally not indicated because: (1) ventricular ectopic activity in such individuals is not associated with increased risk of death, and (2) many anti-arrhythmic drugs have been shown to increase the risk of death. Patients with high-grade and/or symptomatic VEA (ventricular ectopic activity) who have known structural heart disease, may be candidates for therapy. Beta adrenergic blockers have a substantial benefit on mortality, and may suppress VEA in many patients. Patients who are at high risk of sudden death due to ventricular tachyarrhythmias are best treated with implantable defibrillators.

Ventricular Fibrillation

This rhythm is manifested by a random oscillation of potential on the surface ECG, with no QRS complexes (Fig. 45). There is no coherent, synchronous electrical or mechanical activity. There is no cardiac output, and this rhythm is fatal if untreated. Treatment is by electrical countershock together with other resuscitative measures.

Figure 39 - Interpolated VPB

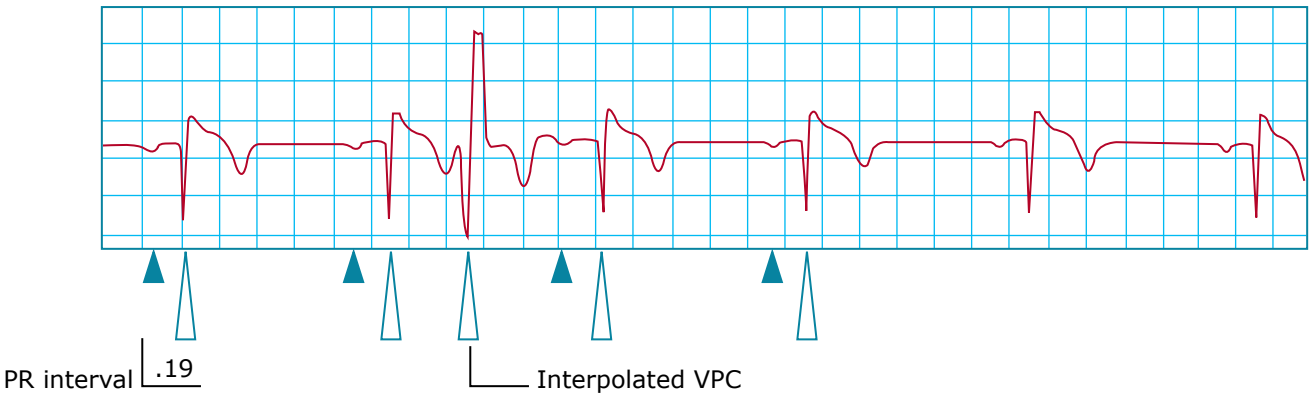


Figure by MIT OCW.

Figure 40 - Bigemeny

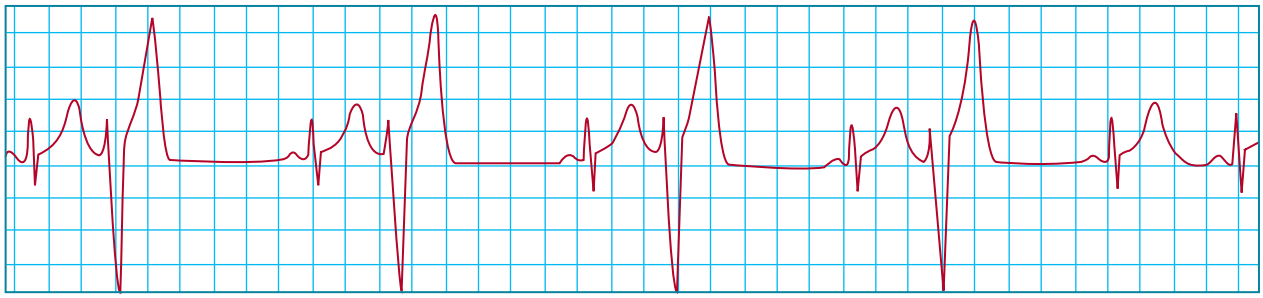


Figure by MIT OCW.

Figure 41 - Multiform VPBs

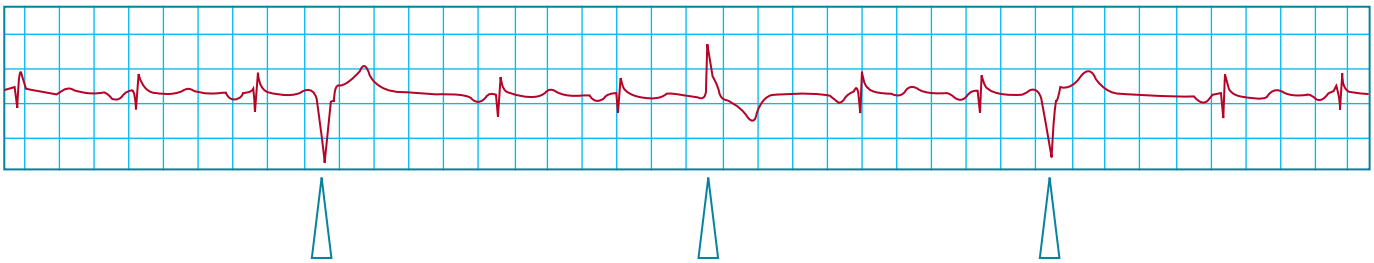


Figure by MIT OCW.

Figure 42 - Ventricular Couplets

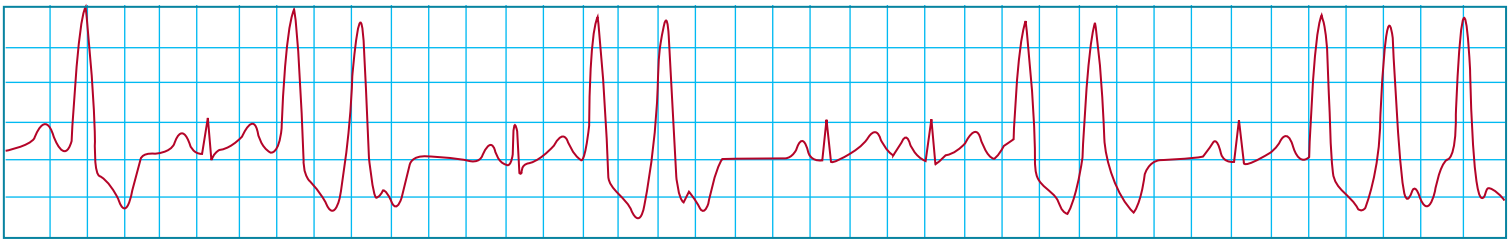


Figure by MIT OCW.

Figure 43 - Short Bursts of Ventricular Tachycardia

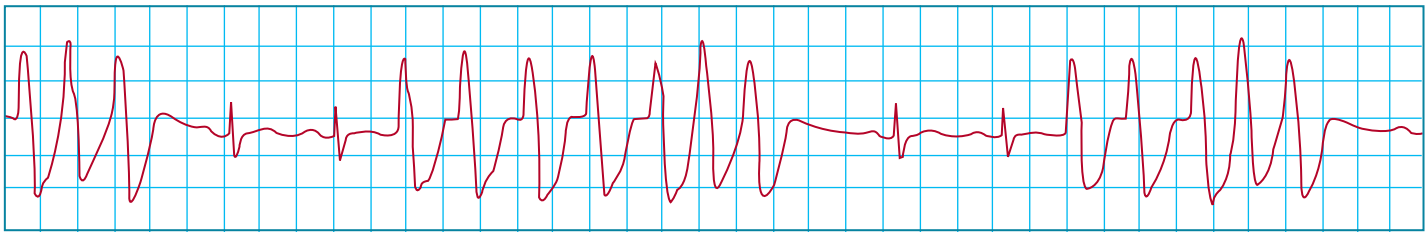


Figure by MIT OCW.

Figure 44 - Ventricular Flutter

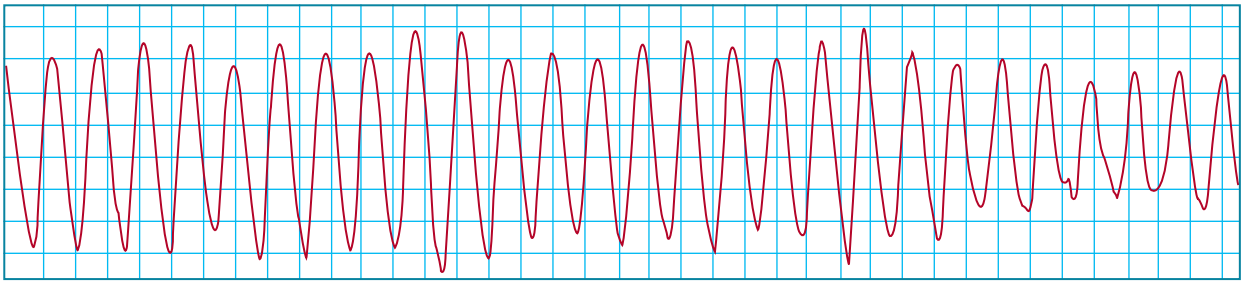


Figure by MIT OCW.

Figure 45 - Three Examples of Ventricular Fibrillation

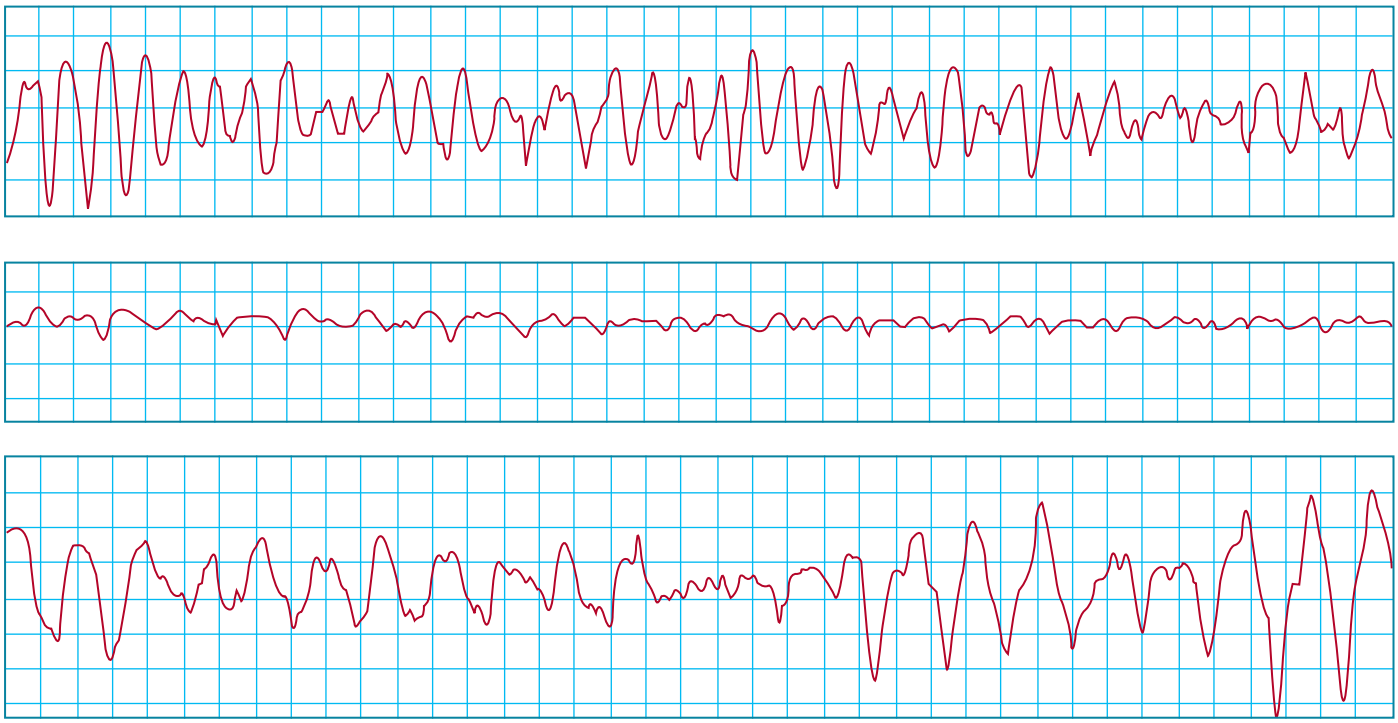


Figure by MIT OCW.

Figure 46 - R-on-T VPB Initiates Ventricular Flutter

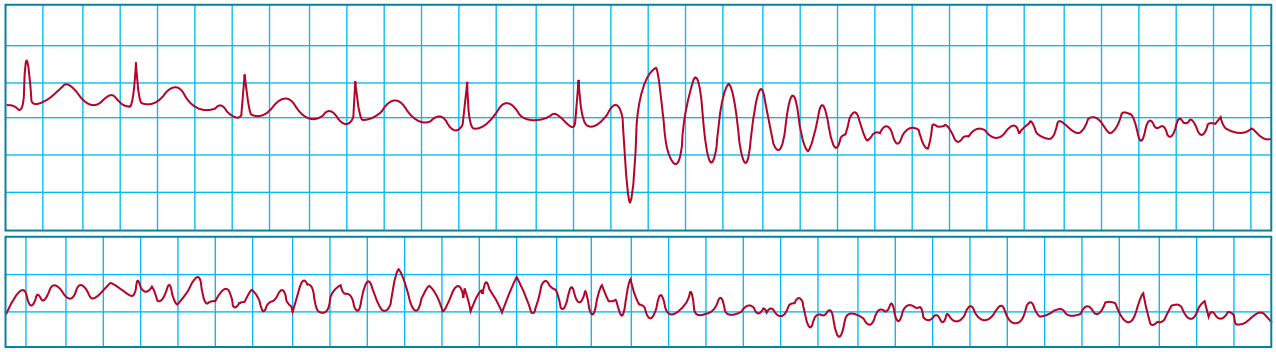


Figure by MIT OCW.

4.3 Arrhythmias Due to Abnormal Conduction

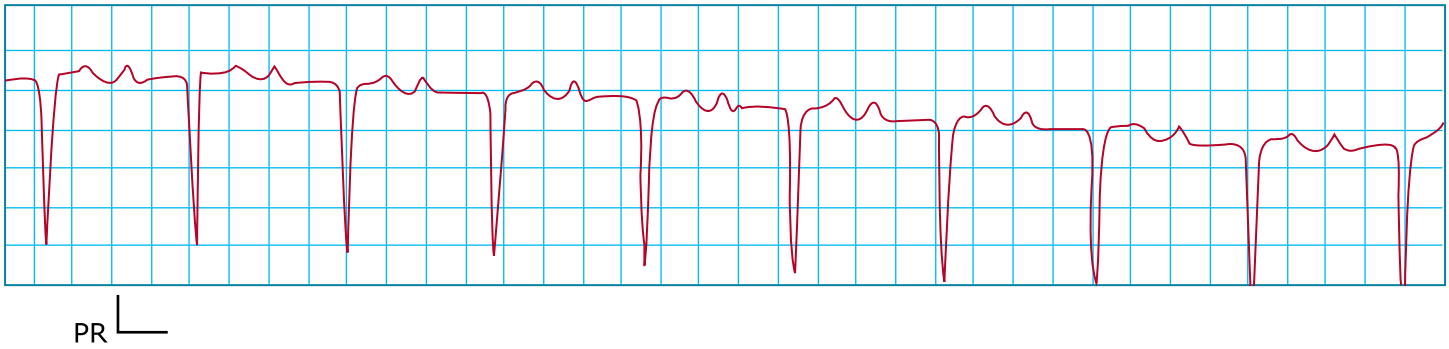
4.3.1 Atrio-Ventricular Conduction Defects

A variety of important rhythm disturbances are related to decremental conduction and block in the AV junction.

First Degree AV Block

This arrhythmia is simply a prolongation of propagation through the AV node. It is manifested on the ECG as a PR-interval longer than 0.20 seconds (Figure 47). All atrial depolarizations are conducted to the ventricles. The arrhythmia does not cause hemodynamic problems of significance, although in some cases optimal AV phasing is lost. The rhythm may reflect underlying disease of the AV junction, or may be a result of drugs such as digoxin, beta-blockers, or calcium antagonists.

Figure 47 - First Degree A-V Block



Second Degree AV Block

In this situation, some atrial impulses are not propagated to the ventricles. The block is either in the AV junction or in the ventricular conduction system. Second degree block may manifest itself in many forms, some with more clinical significance than others.

An increase in vagal tone may result in prolongation of the PR-interval, and in some cases will cause transient block in AV propagation. Transient second degree heart block may be seen in normal individuals especially during sleep or in episodes of intense vagal activity such as vomiting. Significant second degree AV block may also be a manifestation of chronic or acute heart disease such as fibrosis, ischemia, infarction, or inflammation. It may be transient (as with acute inferior myocardial infarction) or permanent.

Second degree AV block has been subdivided into two types:

- Mobitz type I (Wenckebach) (Fig. 48). In this form, the PR-interval grows progressively longer until finally an atrial beat is non-conducted. This results in a typical group beating pattern, with a fixed or variable ratio of atrial to ventricular beats (4:3, 6:5, etc.). The AV node conduction velocity seems to slow with successive beats until one impulse fails to be conducted. After a period of rest, the AV node recovers and the cycle repeats.

Figure 48 - Second Degree A-V Block (Wenckebach or Mobitz Type I)

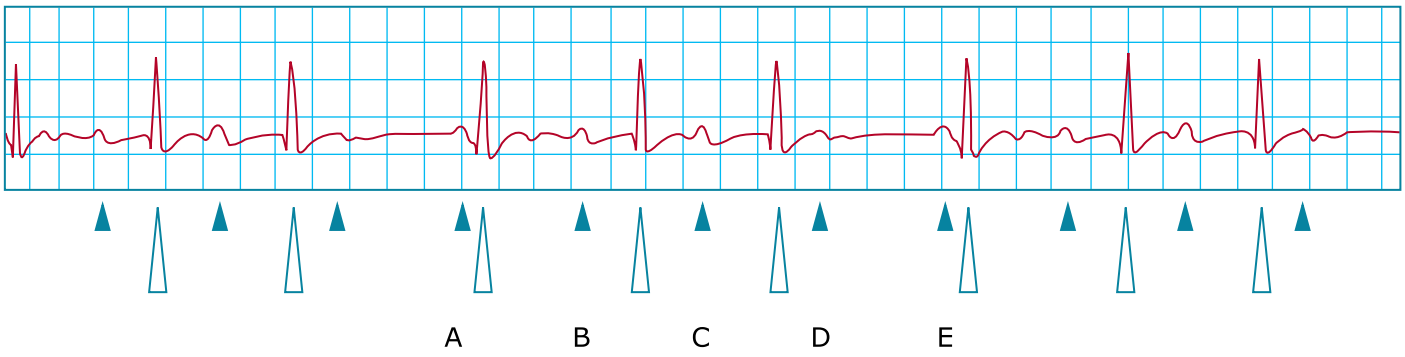


Figure by MIT OCW.

- Mobitz type II (Fig. 49). In this type of second degree AV block the PR intervals do not change, but remain constant. Occasional atrial beats are non-conducted or “dropped”.

Figure 49 - Second Degree A-V Block (Mobitz II)

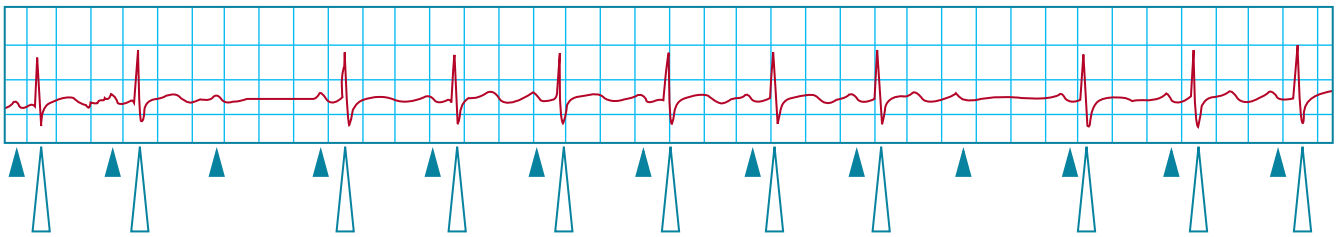


Figure by MIT OCW.

The two forms of second degree AV block have important clinical distinctions. The form with constant PR-intervals is usually associated with organic disease of the ventricular conduction system. Recordings from within the heart demonstrate a prolongation in the conduction time from the bundle of His to the ventricle. The implication is that the major ventricular conduction pathways have been significantly damaged. This exposes the patient to significant risk of complete heart block.

The Wenckebach type second degree heart block is more often transient and is seen either in association with an acute process (such as myocardial infarction), or as a result of changing vagal tone. Wenckebach-type block is usually associated with the AV node itself, and is generally not considered a precursor of progressive AV node conduction failure.

Third Degree (Complete) AV Block

Third degree AV block implies complete block of all atrial impulses. There is no communication between the atria and the ventricles, and AV synchrony is lost. Since the ventricles receive no supraventricular impulses, an escape rhythm usually ensues. If the conduction block is above the AV node, then a junctional escape rhythm may develop (Fig. 50). If the AV node is severely depressed, or the conduction system is interrupted below the AV node, then an idioventricular escape rhythm develops (Fig. 51). Patients with complete heart block who are

untreated tend to die suddenly as a result of either cardiac standstill or ventricular fibrillation. Even with the use of oral sympathomimetic drugs, the annual mortality rate was approximately 50%. The treatment of choice is a permanent cardiac pacemaker (Fig. 52). This technology has been extremely successful in treating complete heart block, essentially eliminating the increased risk of dying prematurely.

Figure 50 - Complete A-V Block with Junctional Escape Rhythm

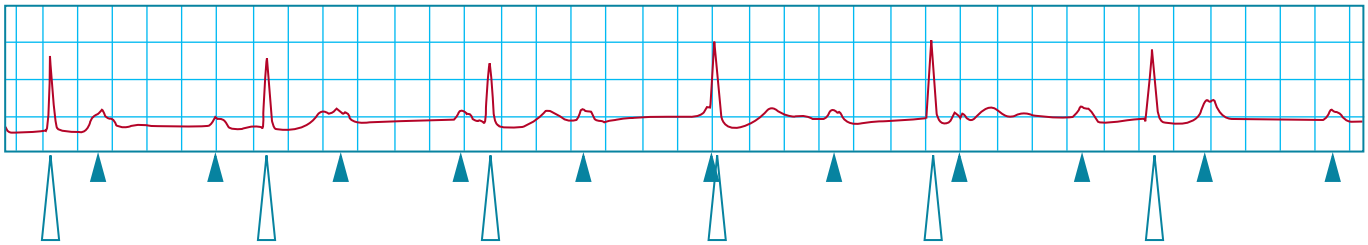


Figure by MIT OCW.

Figure 51 - Complete Heart Block with Idioventricular Escape Rhythm

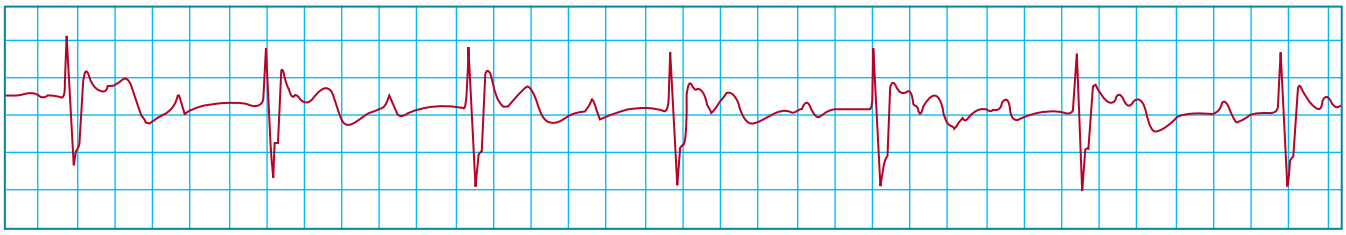


Figure by MIT OCW.

Figure 52 - Pacemaker Rhythm

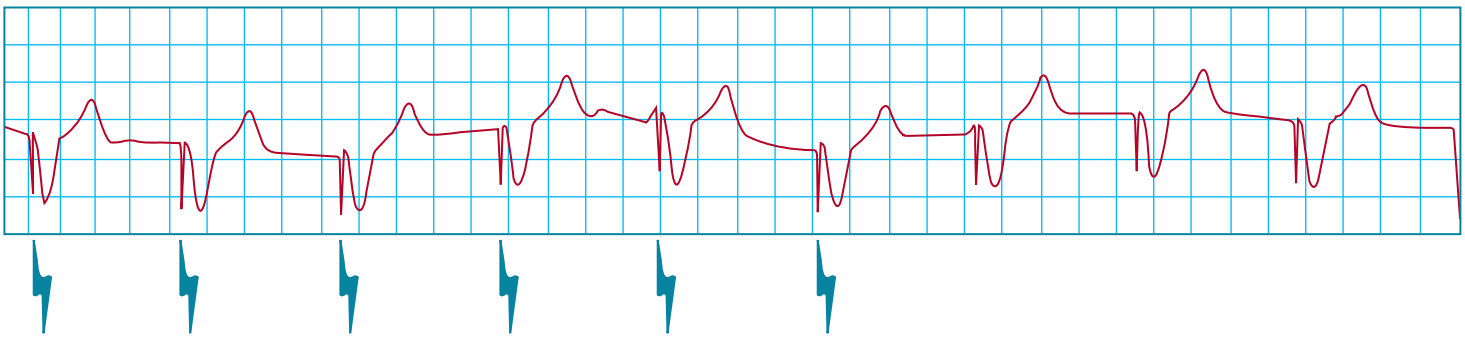


Figure by MIT OCW.

4.3.2 Ventricular Conduction Defects

The ventricular conduction pathways are illustrated in Figure 53. After leaving the AV node, electrical activity passes through the bundle of His. The conduction system then divides distally: the right ventricle is supplied by the right bundle branch, and the left ventricle is supplied by branches from the left bundle. There are several types of ventricular conduction defects. A **bundle branch block** is an ECG abnormality resulting from failure to conduct in either the right bundle branch or the main division of the left bundle branch. A **hemi-block** is an ECG pattern which is associated with failure to conduct through one of the two fascicles of the left bundle branch.

Figure 53
Diagrammatic Illustration of the Intraventricular Conduction System

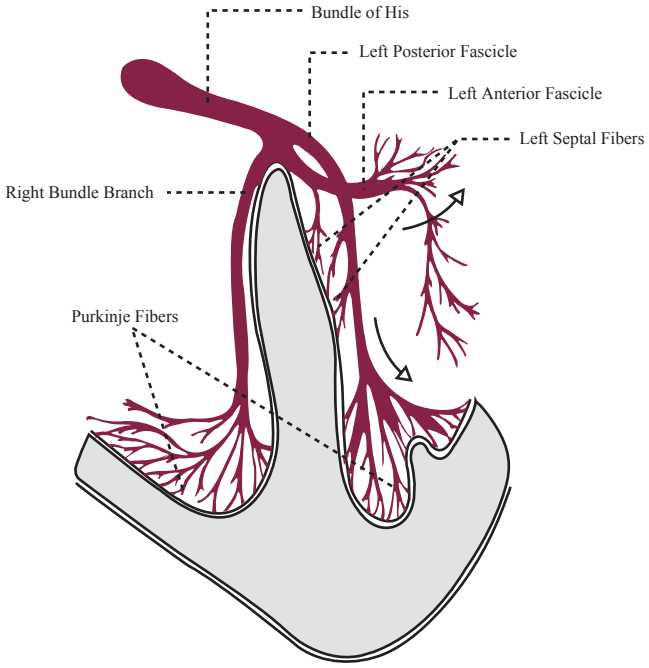


Figure by MIT OCW.

Right Bundle Branch Block

Right bundle branch block (RBBB) is fairly common and is not diagnostic of any heart disease as such. (See Figure 54.) While it may be seen in a number of types of heart disease (coronary heart disease, hypertensive heart disease, congenital heart disease, right ventricular hypertrophy, etc.), it may also be found in normal individuals with no evidence of heart disease. Figure 55 illustrates the abnormal sequence of ventricular depolarization in RBBB. Note that septal

depolarization is normal since it originates from the intact left bundle branch. Left ventricular depolarization spreads normally. Right ventricular depolarization is delayed because the conduction velocity through muscle is much slower than that through the conduction system. Figure 56 shows the typical pattern of RBBB. Note the wide QRS complexes, with the relatively normal initial waves, but the abnormal late deflections reflecting the late right ventricular depolarization. This leads to a late positive (R′) wave in the anterior precordial leads and a late negative S wave in the lateral precordial leads and lead I.

Figure 54 - Right Bundle Branch Block

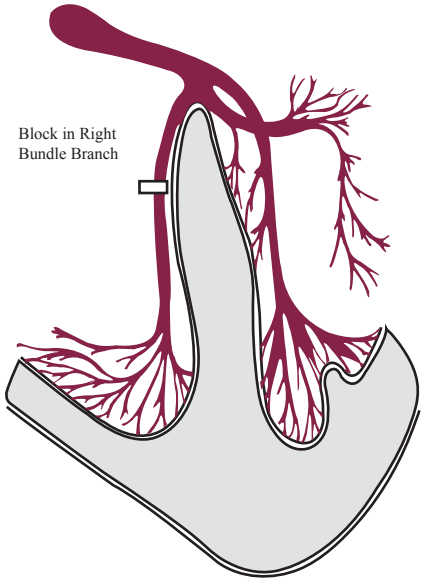


Figure by MIT OCW. After Goldman (1973), Fig. 9-2.

Figure 55 - Sequence of Septal-Ventricular Activation in Right Bundle Branch Block

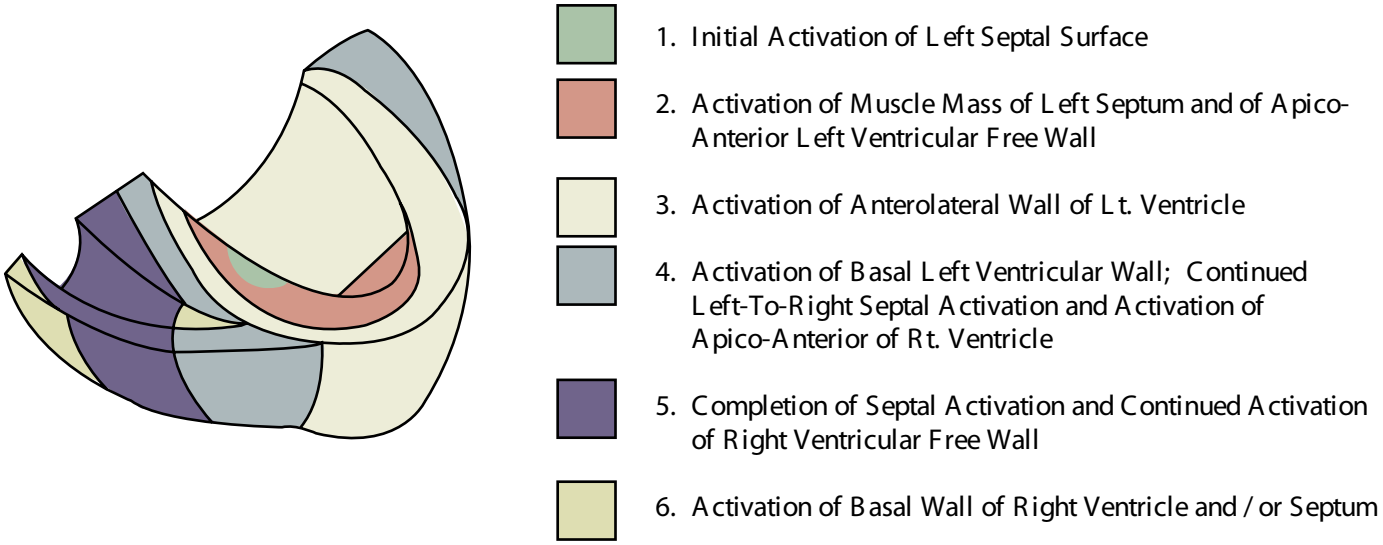


Figure by MIT OCW.

Figure 56 - ECG Pattern of RBBB

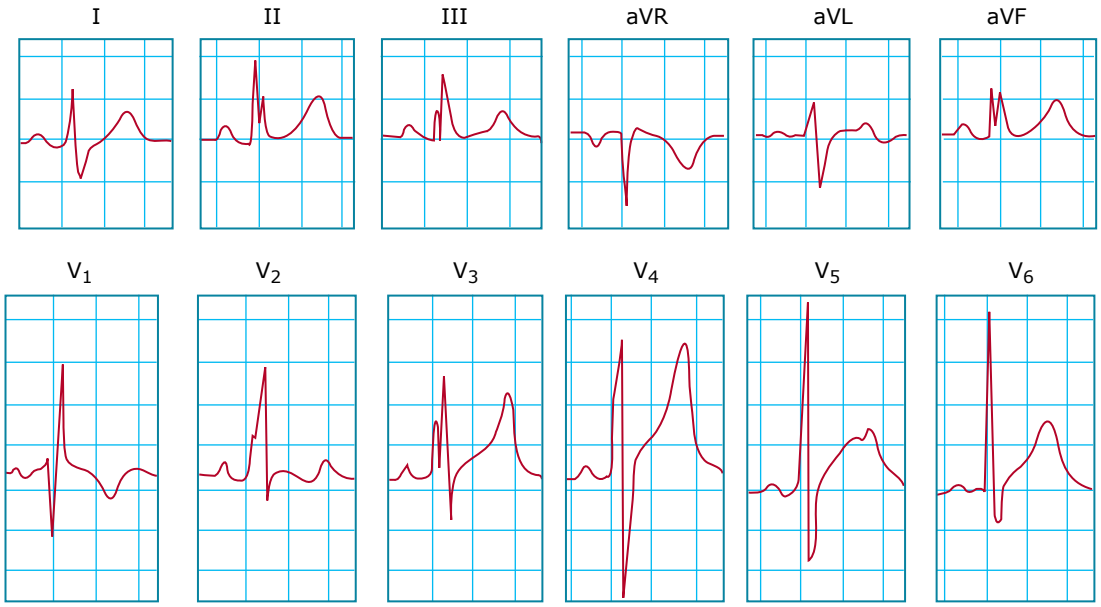


Figure by MIT OCW.

Left Bundle Branch Block

Left bundle branch block (LBBB) is illustrated in Figure 57. It is rarely seen in individuals with healthy hearts. It may occur in association with a wide variety of clinical entities including coronary artery disease, hypertensive heart disease, aortic valvular disease, and others. The sequence of ventricular depolarization begins at the apex of the right ventricle and spreads across the septum from right to left (opposite from normal) and later across the entire left ventricle (Fig. 58). The ECG in LBBB is illustrated in Figure 59. Note the wide QRS complexes, the absence of the initial (septal) r-wave in V_l, and the abnormal ST and T waves.

Figure 57 - Left Bundle Branch Block

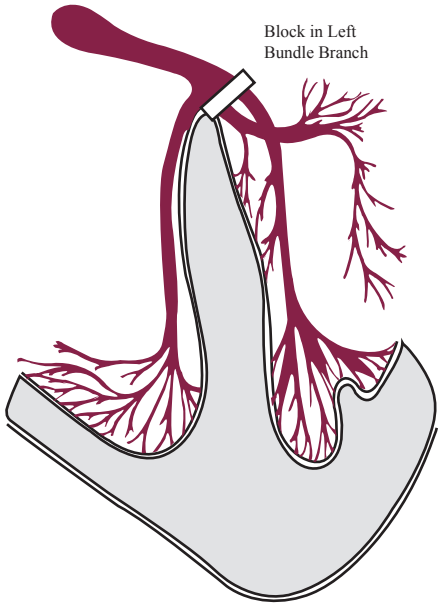


Figure by MIT OCW. After Goldman (1973), Fig. 9-11.

Figure 58 - Sequence of Septal-Ventricular Activation in Left Bundle Branch Block

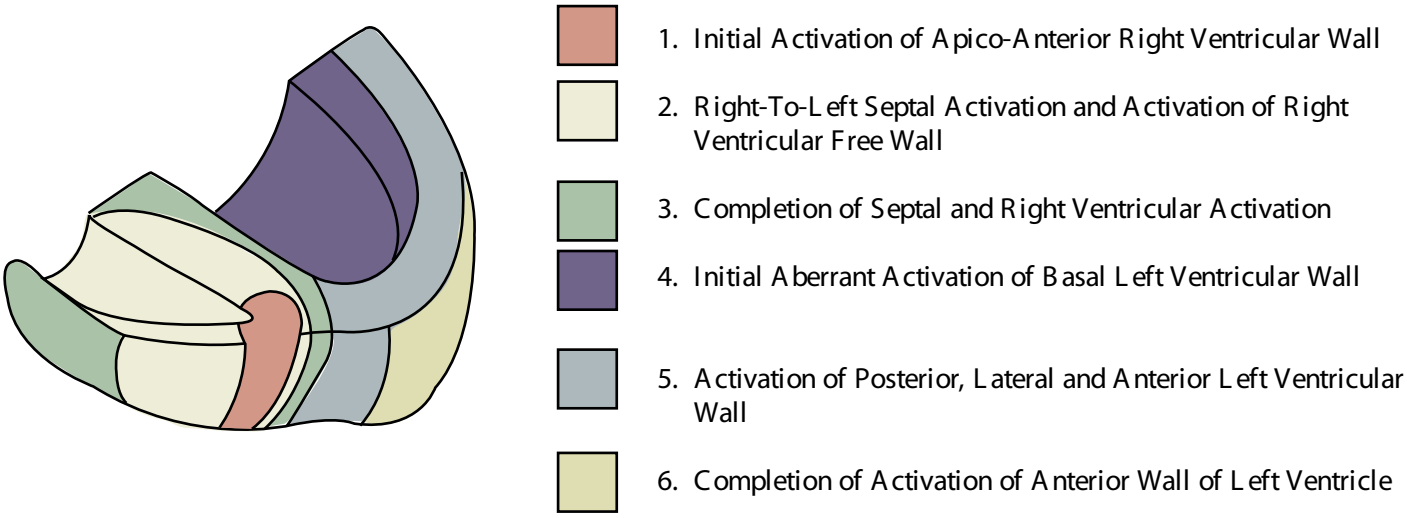


Figure by MIT OCW.

Figure 59 - Left Bundle Branch Block

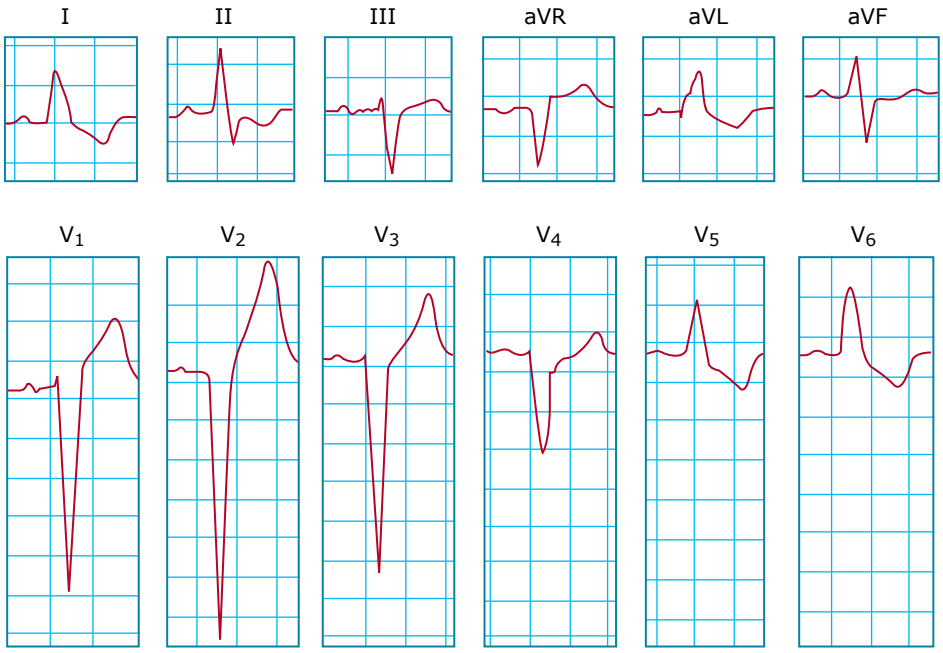


Figure by MIT OCW.

The Hemiblocks

The anterior division of the left bundle branch supplies the anterior and superior portions of the left ventricle. The posterior division supplies the posterior and inferior portions. If one fascicle is blocked, depolarization occurs via the other fascicle and spreads more slowly into the "block" area. The major effect of this change in depolarization sequence is a shift in the electrical axis of the heart. The QRS complex is not significantly widened. In left anterior hemiblock (Fig. 60) the posterior portion of the left ventricle depolarizes first, followed by the anterior and superior portions. The resultant shift in the temporal course of the heart vector causes a significant shift in the mean electrical axis toward the left. The axis is usually more negative than –30 degrees—often more than –60 degrees. (See Figure 61.)

Figure 60 - Left Anterior Hemiblock

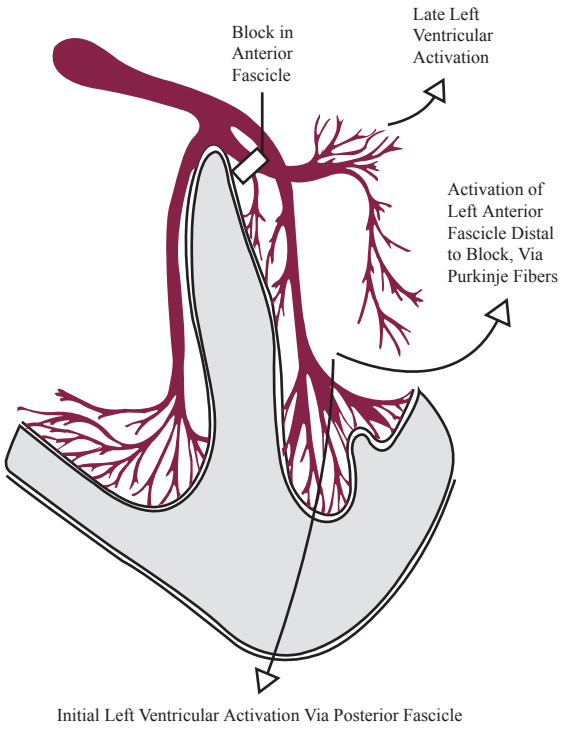


Figure by MIT OCW. After Goldman (1973), Fig. 9-20.

Figure 61 - Left Anterior Fascicular Block.
The mean QRS axis = –50 degrees. The QRS interval = 0.1 second. The R voltage in aVL = 14 mm. There is ST depression in I, aVL, and V_6. Persistent S waves in V_{5-6} (clockwise rotation) are the result of the superiorly directed late QRS forces. Clinical diagnosis: Hypertensive cardiovascular disease.

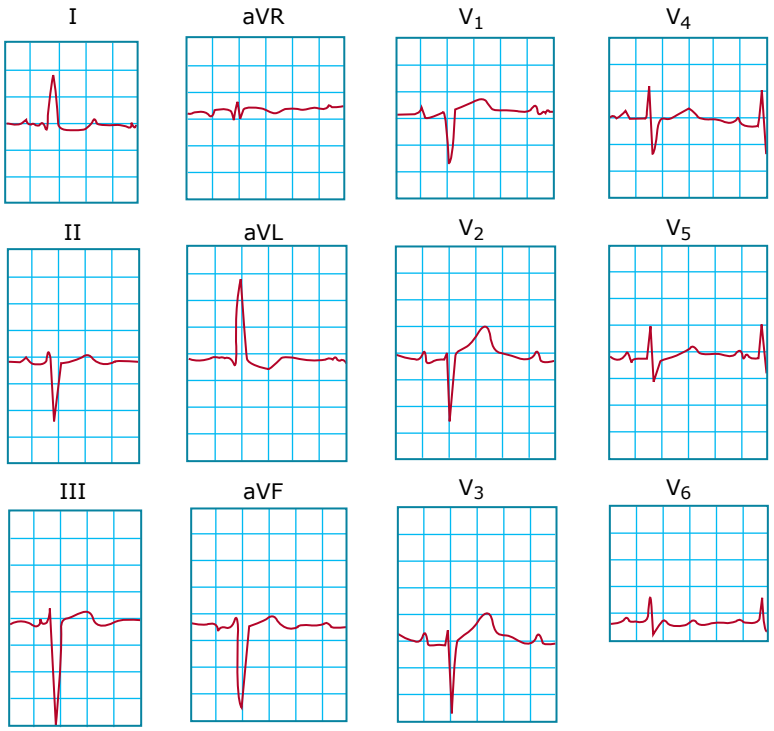


Figure by MIT OCW.

In left posterior hemiblock (Figs. 62 and 63), the mean electrical axis gets shifted to the right (greater than +110 degrees). This QRS axis may be in the wide range of normal, and can be a manifestation of right ventricular hypertrophy or lateral wall myocardial infarction. Hence, the ECG diagnosis of left posterior hemiblock is difficult, and requires exclusion of possible right ventricular hypertrophy or old infarction.

Bifascicular block is used to indicate the ECG finding of complete right bundle branch block in combination with left axis deviation which implies left anterior hemiblock (see Figure 64). In this situation two of the three major portions of the ventricular conduction system are blocked.

Trifascicular block is used to describe the ECG finding of bilateral bundle branch block and a prolonged PR interval indicating first degree AV block. Although the latter could represent delay in the AV node, it more likely represents incomplete block in the remaining intact fascicle of the conduction system (Figures 65 and 66). Patients with this finding are at increased risk of developing complete AV block.

Figure 62 - Left Posterior Hemiblock

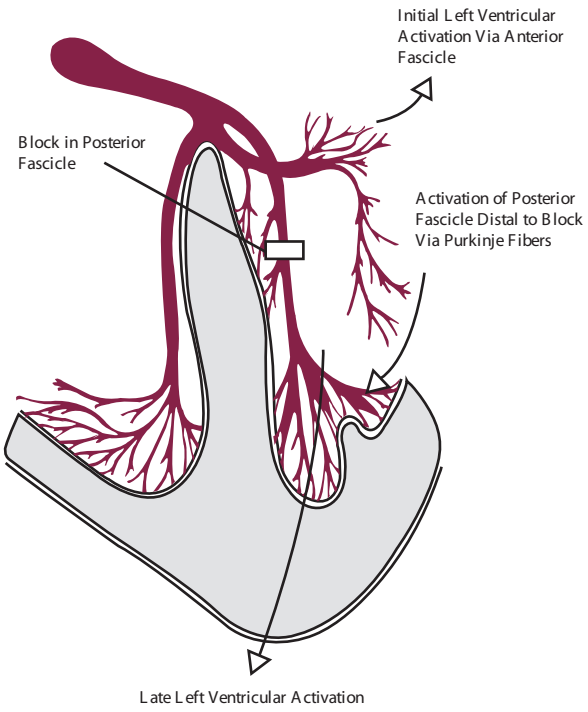


Figure by MIT OCW. After Goldman (1973), Fig. 9-22.

Figure 63 - Left Posterior Fascicular Block.

Mean frontal plane QRS axis is +110 degrees. QRS interval is 0.1 second. P waves in leads II, III, and aVF are tall. While this is consistent with right atrial and right ventricular hypertrophy, cardiac catheterization revealed normal right heart pressures. This example emphasizes the need to exclude right ventricular hypertrophy before electrocardiographic diagnosis of posterior fascicular block. In addition, precordial leads indicate an anterior myocardial infarction. Clinical diagnosis: Arteriosclerotic heart disease with old myocardial infarction.

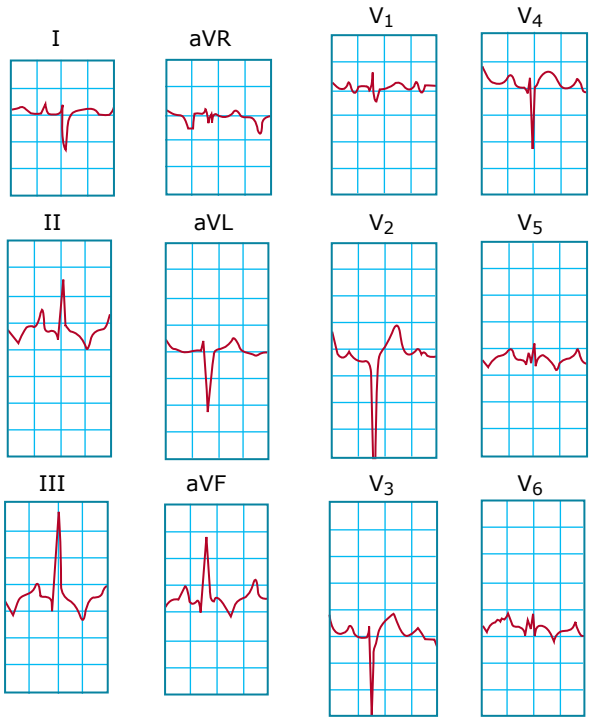


Figure by MIT OCW.

Figure 64 - Bilateral bundle disease.

The initial 0.05 second QRS is directed leftward and superiorly (frontal plane axis = –60 degrees) indicating a lesion in the left bundle anterior fascicle. The terminal portion of the QRS is delayed and oriented to the right and anteriorly (wide S in I and V_{5-6}: wide R′ in V_{1-2}), typical of right bundle branch block. This combination suggests bilateral bundle disease.

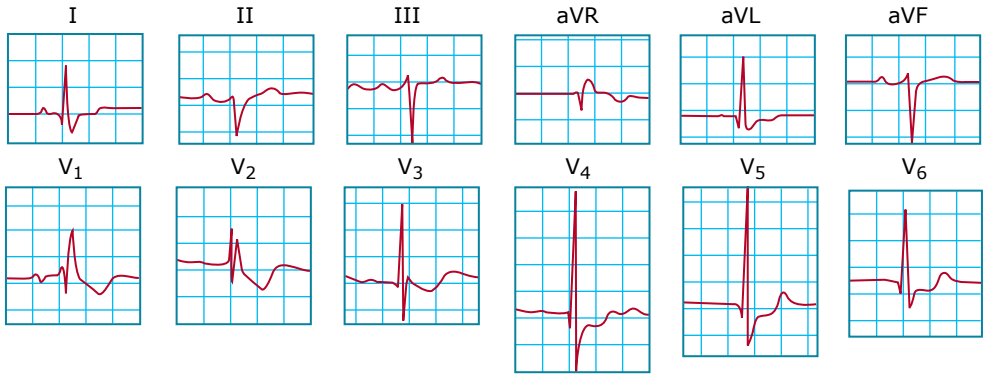


Figure by MIT OCW.

Figure 65 - Diagram of trifascicular block.

Complete blocks in right bundle branch and left anterior fascicle; incomplete block in left posterior fascicle which prolongs the P-R interval.

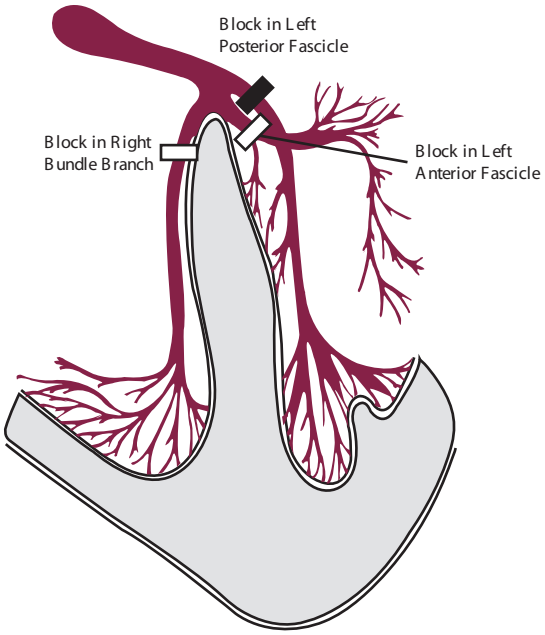


Figure by MIT OCW. After Goldman (1973), Fig 9-31.

Figure 66 - Trifascicular block.

The QRS interval is 0.13 seconds. The wide S waves in I and wide R′ waves in V_{1-3} indicate right bundle branch block. The initial 0.06 second frontal plane QRS vector is –60 degrees, indicating a left anterior fascicular block. The P-R interval is 0.35 seconds, indicating \first degree heart block, possibly due to incomplete block in the left posterior fascicle. The q waves in V_{2-3} do not necessarily indicate anterior wall infarction.

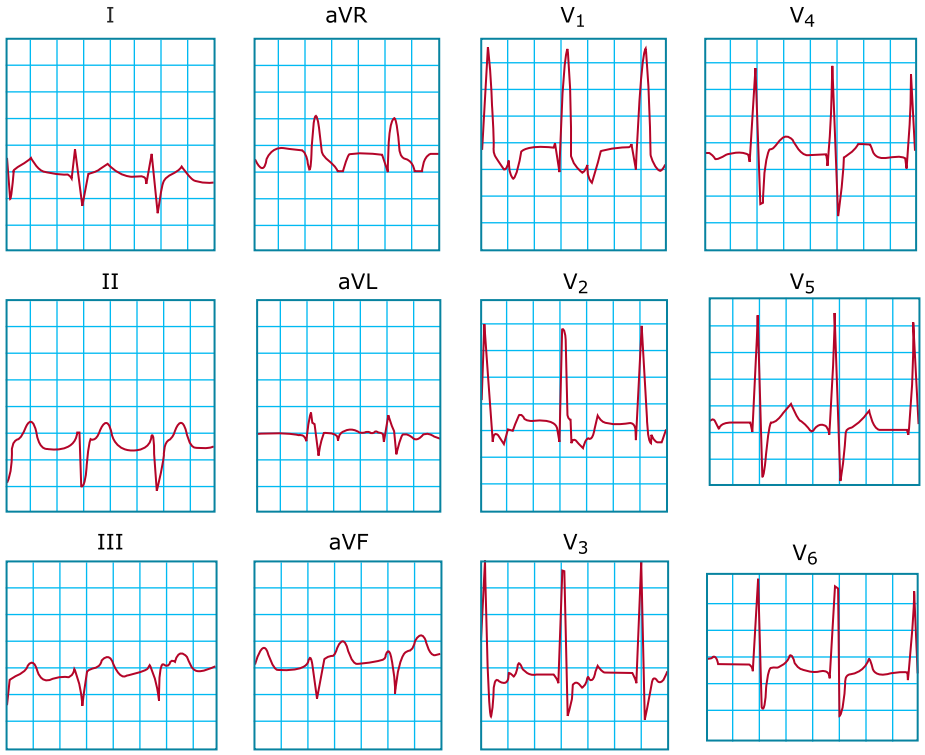


Figure by MIT OCW.

5. Survey of additional ECG Abnormalities

5.1 Hypertrophy Patterns

In some pathological conditions certain chambers of the heart may be required to perform increased levels of mechanical work. For example, with hypertension (high blood pressure) the left ventricle must generate abnormally high pressures. In mitral stenosis (a narrowing of the mitral valve) the left atrium must generate increased pressure to maintain an adequate blood flow through the narrow valve. In response to the increased work load, the muscle cells of the affected chamber increase in volume and in contractile elements. This hypertrophy of muscle is reflected in the electrocardiogram by an increased magnitude of the heart vector. In addition, conduction times may be increased since the muscle mass and volume increases. Hypertrophy of specific chambers is reflected in characteristic changes in the ECG waveforms.

Left Atrial Hypertrophy (P-mitrale)

As the excitation wave of the heart travels from the SA node to the AV node, the right atrium is depolarized before the left atrium. The P-wave is the electrical sum of both depolarizations. In left atrial hypertrophy the left atrial contribution becomes more pronounced, resulting in a wide (greater than 0.11 sec) notched P-wave. In lead V_1, the wave is diphasic with a large terminal negative sweep. Figure 67 illustrates the classical picture of the left atrial hypertrophy in a case of mitral stenosis.

Figure 67 - Left atrial hypertrophy.
Note the broad, notched P waves in standard and extremity leads and V_{4-6}. The characteristic diphasic P wave with a wide negative component is seen in V_1. Clinical diagnosis: Mitral stenosis.

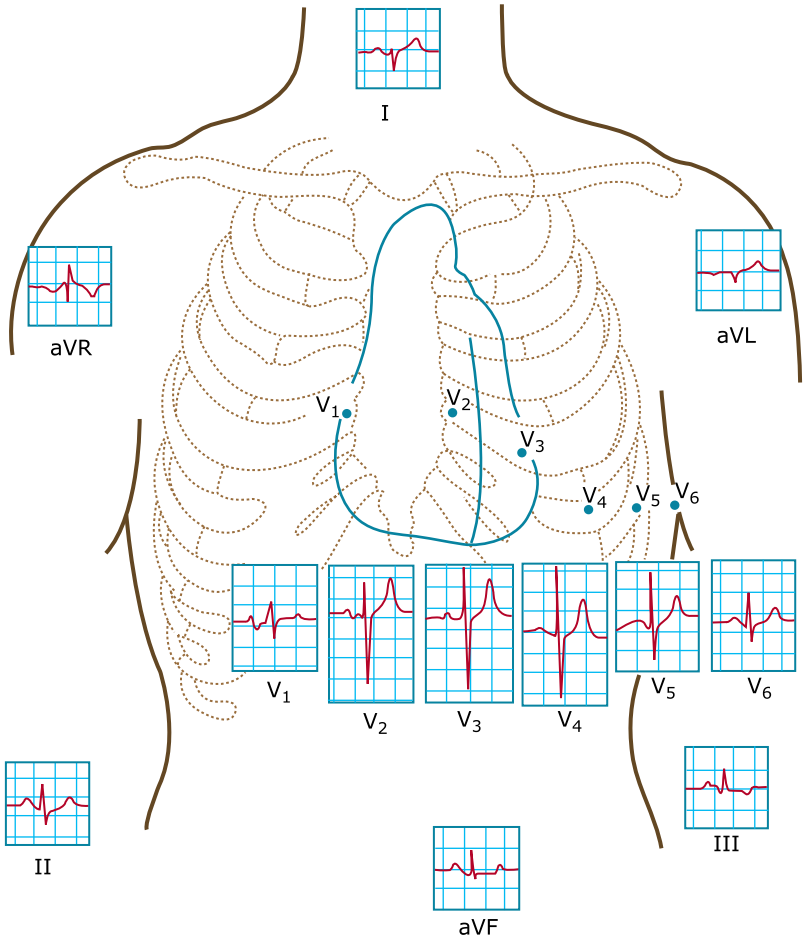


Figure by MIT OCW.

Right Atrial Hypertrophy (P-Pulmonale)

Tall, slender, peaked P-waves in leads II, III, aVF are characteristic of right atrial hypertrophy. The P-waves in lead V_1 may be prominent, peaked, diphasic or inverted. Right atrial hypertrophy may be seen in tricuspid stenosis or regurgitation, congenital heart defects such as atrial septal defect, or secondary to right ventricular overload. Right atrial hypertrophy is often seen in patients with severe lung disease and increased pulmonary artery pressure—hence the name "P-pulmonale". Figure 68 illustrates right atrial hypertrophy.

Figure 68 - Right atrial hypertrophy.

There are tall, peaked P waves in II, III, and aVF. A deeply inverted and slender P wave is seen in V_1. The QRS and T abnormalities are indicative of right ventricular hypertrophy. Clinical and autopsy diagnosis: Severe chronic obstructive lung disease; right atrial and ventricular hypertrophy.

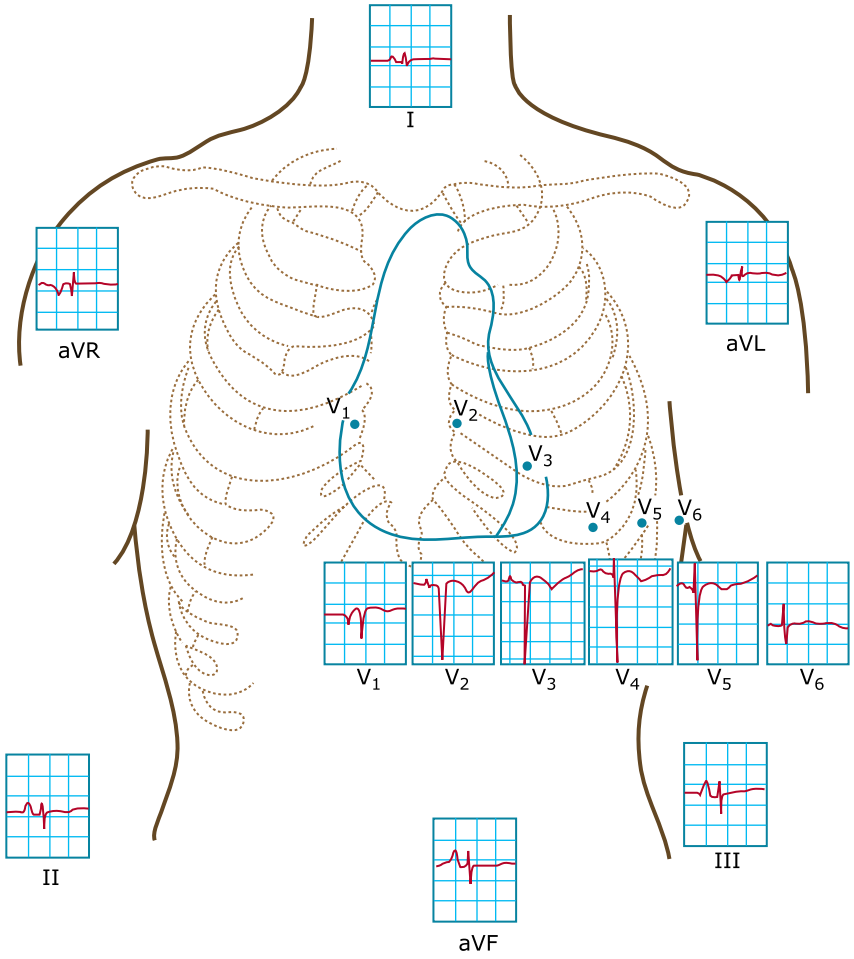


Figure by MIT OCW.

Left Ventricular Hypertrophy

Left ventricular hypertrophy (LVH) leads to changes in the amplitude of the QRS complex, the duration of the QRS, the electrical axis, and in ST-T wave morphology. The amplitude of the QRS is increased and the ECG diagnosis of LVH requires one or more of the following criteria:

a. The sum of the largest S-wave in V_1 or V_2 and the largest R-wave in V_5 or $V_6 \geq 3.5$ mV.

b. The largest S-wave in V_1 or $V_2 \geq 2.5$ mV.

c. The largest R-wave in V_5 or $V_6 \geq 2.5$ mV.

d. The largest R-wave in a limb lead ≥ 1.3 mV.

e. The sum of the R in lead I and the S in lead III ≥ 2.5 mV.

The duration of the QRS complex is usually slightly prolonged, but generally not beyond 0.12 seconds.

The electrical axis is usually shifted to the left beyond –30˚.

ST-T wave changes are usually seen in LVH. ST-segment depression and T-wave inversion are common.

Figure 69 illustrates the ECG findings of LVH. This condition may result from a number of clinical states including: hypertension, aortic stenosis of regurgitation, mitral regurgitation, long-standing coronary artery disease, and certain congenital diseases such as patent ductus arteriosus and coartation of the aorta.

Figure 69 - LVH

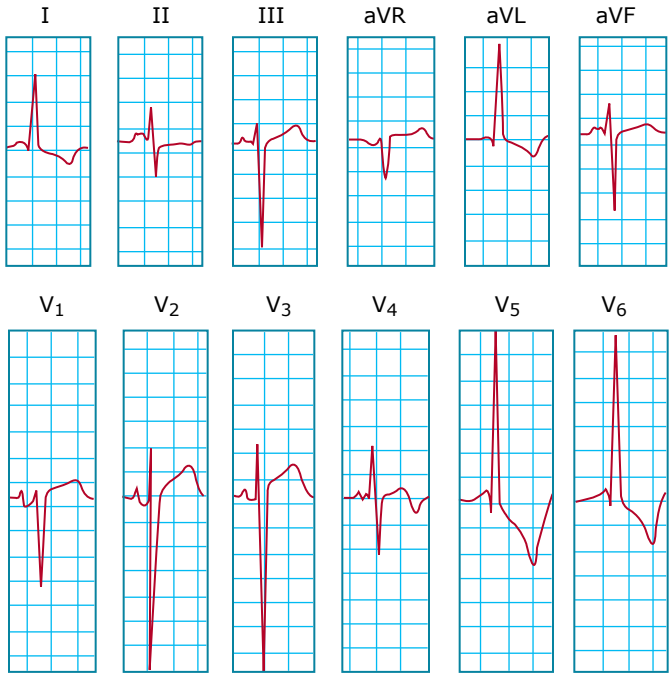


Figure by MIT OCW.

Right Ventricular Hypertrophy

Right ventricular hypertrophy (RVH) may occur with mitral stenosis, pulmonary hypertension, pulmonic stenosis, severe pulmonary disease and certain forms of congenital heart disease. The ECG changes seen with RVH are often more difficult to detect than those associated with LVH. The changes are related to the QRS vector: it is increased in magnitude and its course is skewed in the direction of the right ventricle. Any of the following criteria suggest RVH:

a. Right axis deviation $\geq +110^{\circ}$.

b. R wave in $V_1 \geq 0.7$ mV.

c. S wave in V_5 or $V_6 \geq 0.7$ mV.

d. R/S ratio in $V_1 > 1.0$.

e. R/S ratio in $V_6 < 1.j$

Figure 70 illustrates RVH.

Figure 70 - RVH

Electrocardiogram of right ventricular hypertrophy in a 23 year old woman with tetralogy of Fallot. (1) There is right-axis deviation of the A QRS with an abnormally wide frontal plane QRS-T angle (90˚). (2) There is a reversed precordial QRS transition with tall R_{V_1} and delayed onset of the intrinsicoid deflection (0.05 second) in lead V_1. (3) Initial R waves (without normal septal Q waves) in leads I, aVL, and V_6 suggest that initial septal activation produces forces directed to the left and posteriorly (a not infrequent finding in right ventricular hypertrophy).

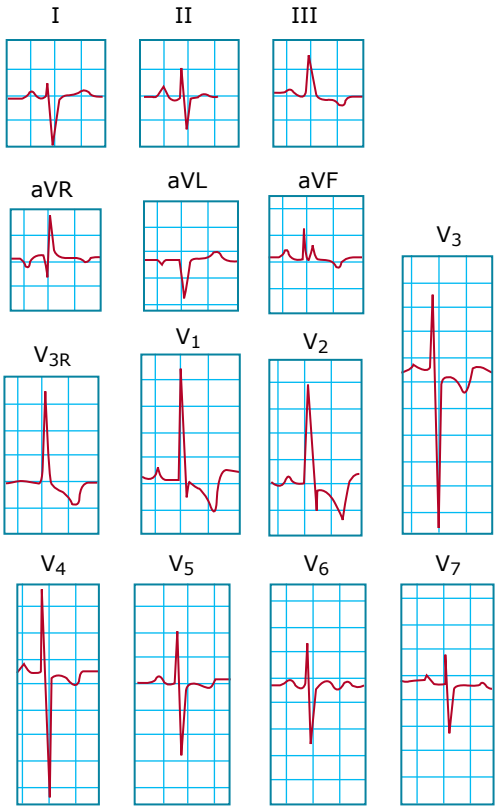


Figure by MIT OCW.

5.2 Ischemia, Injury, and Infarction

Ischemia of myocardial tissue causes shortening of the action potential, particularly in the endocardial areas. As a result, repolarization begins at the endocardial surface and propagates toward epicardium (exactly the opposite of normal). The change of repolarization is reflected in T-wave inversion, and in some cases ST-segment straightening and depression. Thus, ischemia may be manifested as T-waves of opposite polarity to QRS complexes. This change unfortunately is not specific, since other conditions lead to T-wave abnormalities (LVH, bundle branch blocks, etc.). On

the other hand, if serial ECGs show systematic T-wave inversions, the evidence for ischemia may be stronger.

Injury of myocardial cells refers to damage such that groups of cells partially depolarize. This damage may be reversible or may proceed to death of cells (infarction). Injury of subendocardial cells leads to ST-segment depression. Subepicardial injury leads to ST-segment elevation. Figure 71 presents a model to explain these observations. During phase 4 there is a DC potential ("injury potential") created by virtue of the boundary between partially depolarized cells and normal myocardium. Since ECG machines are AC coupled, this shift is not registered. During the plateau of the action potential all myocardial cells are depolarized and the potential of the electrode falls to zero. This leads to an apparent depression of the ST-segment. The opposite argument holds for why subepicardial injury leads to ST-segment elevation.

Infarcted cells are dead, and electrically unexcitable. Absence of the ability to conduct the action potential in a region of infarction causes a dramatic change in the magnitude and direction of the equivalent heart vector. In particular, positive R-waves may disappear and be replaced by negative Q-waves. In actual myocardial infarctions a sequence of injury followed by infarction is usually seen. ST-segment elevation is seen early (hours-day), followed by loss of R-wave voltage and Q-wave development (day-days) and T-wave inversion. As time goes on, the ST-segment shift returns to baseline (days to weeks), and still later the T-waves may normalize. The Q-waves persist permanently, however, reflecting replacement of normal muscle by scar tissue. (See Fig. 72.)

Figure 71 - Systolic current of injury concept of the disappearance of the diastolic current of injury.

An exploring electrode of a unipolar lead is positioned over the epicardial surface of a wedge of ventricular muscle. A) Subendocardial injury (dotted area) leads to partial depolarization of the injured zone (positively charged with respect to the outer fully polarized muscle). As a result, a diastolic current of injury flows between the two regions. This can be treated as a current vector, *i*, originating from *E*, resulting in measurement of a positive potential at the recording electrode. Althought, the baseline is deflected upward, clinical ECG machines are AC coupled, and do not record DC shifts. After depolarization, the wedge of muscle is electrically neutral, the diastolic injury current disappears, and the electrocardiographic tracing returns to its preinjury base line. Later, as the muscle repolarizes, the injury current reappears and the tracing gradually returns to the postinjury level. On the electrocardiogram, this results in a depressed S-T segment. (B) Subepicardial injury, in contrast, leads to a reverse polarity diastolic injury vector (*I*): before depolarization the original base line is depressed, while after depolarization the S-T segment is apparently elevated.

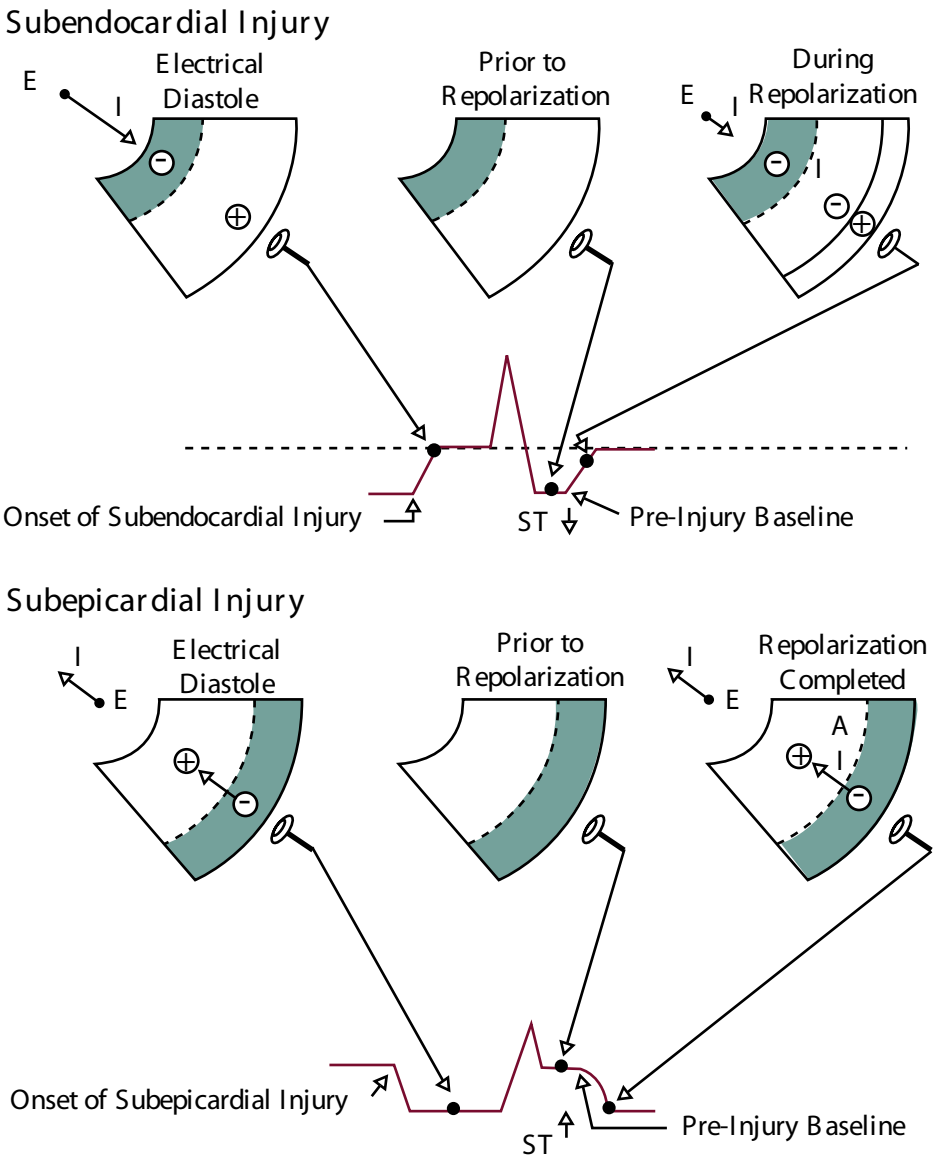
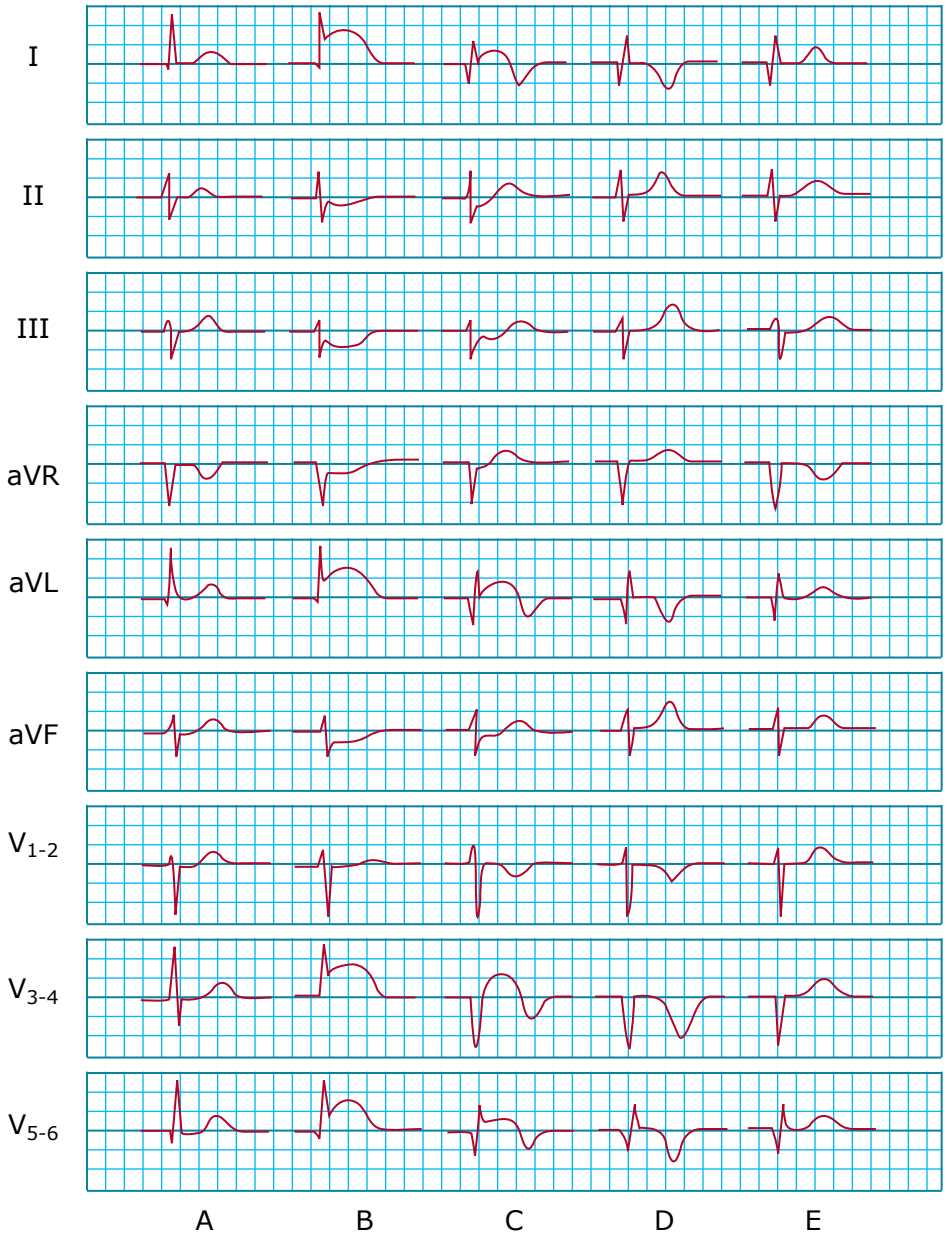


Figure by MIT OCW.

Figure 72 - Diagrammatic Illustration of Serial Electrocardiographic Patterns in Anterior Infarction

A: Normal tracing.
B: Early pattern (hours after infarction): leads I, aVL, and V_{3-6} exhibit ST segment elevation while leads II, III, and aVF exhibit reciprocal ST depression.
C: Later pattern (many hours to a few days): Q waves have appeared in I, aVL, and V_{5-7}, and QS complexes are present in V_{3-4}, indicating that the major transmural infarction is underlying the area recorded by V_{3-4}; ST segment changes persist, but are diminished, and T waves are beginning to invert in leads with elevated ST segments.
D: Late established pattern (many days to weeks): Q waves and QS complexes persist; ST segments are isoelectric; T waves are symmetric and deeply inverted in leads with previous ST elevation and tall in leads with previous ST depression. This pattern may persist for the remainder of the patient's life.
E: Very late pattern: This may occur many months to years after the infarct. Abnormal Q waves and QS complexes persist while the T waves have gradually returned to normal.



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Sources for Figures:

EKG strip charts from PhysioNet were obtained specifically from the PhysioNet MIT-BIH Arryhthmia Database Directory at http://www.physionet.org/physiobank/database/mitdb/. For more information on PhysioNet, please see:

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