CHAPTER

7

Dysrhythmia Interpretation and Management

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INTRODUCTION

The interpretation of cardiac rhythm disturbances or dysrhythmias is an essential skill for nurses employed in patient care areas where electrocardiographic monitoring occurs. The ability to rapidly analyze a rhythm disturbance as well as initiate appropriate treatment improves patient safety and optimizes successful outcomes. The critical care nurse is often the healthcare professional responsible for the continuous monitoring of the patient's cardiac rhythm, and has the opportunity to provide early intervention that can prevent an adverse clinical situation. This responsibility requires not only a mastery of interpreting dysrhythmias but also the ability to critically identify the unique monitoring needs of each patient. This chapter presents a review of basic cardiac dysrhythmias, etiology, clinical significance, and appropriate treatments to aid the novice critical care nurse in mastering dysrhythmia recognition.

The word *dysrhythmia* refers to an abnormal cardiac rhythm. People also speak of cardiac arrhythmias. Either term may be used to describe deviations from normal sinus rhythm. The term *dysrhythmia* is used throughout this text.

The goal of this chapter is to provide a basic understanding of electrocardiography for analyzing and interpreting cardiac dysrhythmias. Electrocardiography is the process of creating a visual tracing of the electrical activity of the cells in the heart. This tracing is called the *electrocardiogram* (ECG). The critical care nurse must have a clear understanding of cardiac monitoring, lead selection, and rhythm interpretation. Part of the difficulty in learning rhythm interpretation is that many of the terms used are synonymous. Throughout this chapter, those terms are clarified. This chapter discusses general concepts of dysrhythmia interpretation.

OVERVIEW OF ELECTROCARDIOGRAM MONITORING

The first ECG was recorded in 1887 by British physiologist Augustus Waller via a capillary electrometer.[9] The electrocardiogram was subsequently named, and the P Q R S T complex was described by Dr. Willem Einthoven, who proceeded to commercially produce a string galvanometer that became popularized in the early 1900s. Dr. Einthoven won the Nobel Peace prize in 1924 for inventing the electrocardiograph. The first electrical electrocardiogram machine weighed 50 pounds and was powered by a 6-volt automobile battery. Today, the 12-lead ECG machine is an essential element or mainstay of health care as a diagnostic tool.

Continuous ECG monitoring did not become common practice until the 1960s, when the first coronary care units were developed. Early cardiac monitoring consisted of monitoring for a heart rate that was too fast or too slow, and for identifying life-threatening dysrhythmias, including ventricular tachycardia, ventricular fibrillation, and asystole.[9] Today, cardiac monitoring is increasingly sophisticated. Technologies have been developed that allow continuous monitoring of 12 leads, and trending of a variety of physiological variables can be performed for any time frame. Cardiac monitoring is also performed in a variety of clinical areas outside the critical care unit. Nurse researchers have contributed a wealth of new knowledge about best practices for cardiac monitoring and

BOX 7-1 INDICATIONS FOR CARDIAC DYSRHYTHMIA MONITORING

- Resuscitated from cardiac arrest
- Early phase of acute coronary syndromes
- Newly diagnosed high-risk coronary lesions
- After cardiac surgery
- After nonurgent percutaneous coronary intervention
- After implantation of automatic defibrillator or pacemaker leads
- Temporary or transcutaneous pacemaker
- Heart block
- Dysrhythmias complicating Wolff-Parkinson-White syndrome
- Drug-induced long QT syndrome
- Intraaortic balloon counterpulsation
- Acute heart failure, pulmonary edema
- Conditions requiring critical care admission
- Procedures that require conscious sedation or anesthesia

Adapted from American Heart Association (AHA). (2011). *2010 Handbook of Emergency Cardiovascular Care for Healthcare Providers.* Dallas, Texas.

have published comprehensive standards for cardiac monitoring in hospital settings.[4,5] Box 7-1 lists priority patient populations for dysrhythmia monitoring.

CARDIAC PHYSIOLOGY REVIEW

The electrocardiogram detects a summation of electrical signals generated by specialized cells of the heart called pacemaker cells. Pacemaker cells have the property of *automaticity,* meaning these cells can generate a stimulus or an action potential without outside stimulation. This electrical signal is conducted through specialized fibers of the conduction system to the mechanical or muscle cells of the heart where a cardiac contraction is generated. Thus there must be an electrical signal for the mechanical event of contraction to occur. The coordinated electrical activity followed by a synchronous mechanical event constitutes the *cardiac cycle.*

The *cardiac cycle* (Figure 7-1) begins with an impulse that is generated from a small concentrated area of pacemaker cells high in the right atria called the sinoatrial node (SA or sinus node). The SA node has the fastest rate of discharge and thus is the dominant pacemaker of the heart. The sinus impulse quickly passes through the internodal conduction tracts and the Bachmann bundle, conductive fibers in the right and left atria. The impulse quickly reaches the atrioventricular (AV) node located in the area called the AV junction, between the atria and the ventricles. Here the impulse is slowed to allow time for ventricular filling during relaxation or ventricular *diastole.* The AV node has pacemaker properties and can discharge an impulse if the SA node fails. The electrical impulse is then rapidly conducted through the bundle of His to the ventricles via the left and right bundle branches. The left bundle branch further divides into the left anterior fascicle and the left posterior fascicle. The bundle branches divide into smaller and smaller branches, finally terminating in tiny fibers called Purkinje fibers that reach the myocardial muscle cells or myocytes. The bundle of His, the right and left bundle branches, and the Purkinje fibers are also known as the His-Purkinje system. The ventricles have pacemaker capabilities if the sinus or AV nodes cease to generate impulses.

The electrical signal stimulates the atrial muscle, called *atrial systole,* and causes the atria to contract simultaneously and eject their blood volume into the ventricles. Simultaneously, the ventricles fill with blood during *ventricular systole.* During atrial systole, a bolus of atrial blood is ejected into the ventricles. This step is called the *atrial kick,* and it contributes approximately 30% more blood to the cardiac output of the

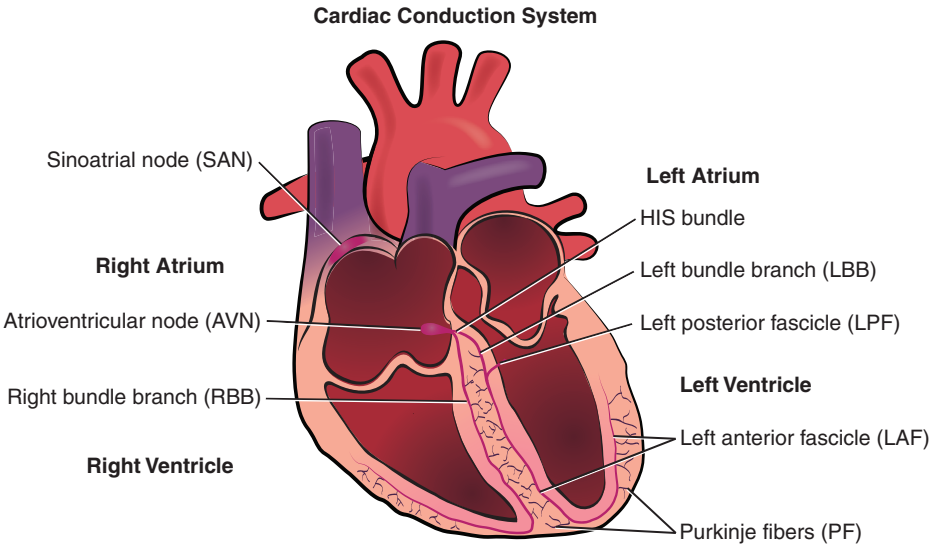


FIGURE 7-1 The electrical conduction system of the heart.

ventricles. The inflow or atrioventricular valves (tricuspid and mitral) close because of the increasing pressure of the blood volume in the ventricles. By this time the electrical impulse reaches the Purkinje fibers, and the muscle cells have become stimulated and cause ventricular contraction. The outflow valves open (aortic and pulmonic) because of increased pressure and volume in the ventricles, allowing for ejection of the ventricular blood, called ventricular systole. At the same time ventricular systole is occurring, *atrial diastole* or filling is occurring. The atria are relaxed and filling with blood from the periphery (deoxygenated) and the lungs (oxygenated). Then because of the rhythmic pacing of the heart, the muscle cells are again stimulated, the atria contract, and atrial blood is ejected once again into the ventricles. This process of electrical stimulation and mechanical response occurs rhythmically 60 to 100 times per minute in the normal heart. The coordination of the electrical and mechanical events in the upper and lower chambers of the heart result in the emptying and filling of these chambers, and the valves open and close because of pressure changes. These physiological actions result in what is known as cardiac output, which continually adjusts to the needs of the body's tissues (Figure 7-2).

Cardiac Electrophysiology

Specialized cardiac pacemaker cells possess the property of automaticity and can generate an electrical impulse on their own. Nonpacemaker or muscle cells must receive an outside stimulus in normal circumstances to generate a response. The response generated either by the pacemaker or muscle cells once stimulated is called the *action potential*. The cardiac action potential consists of phases related to depolarization, repolarization and the resting or polarized state of the cell. While this summary describes the action potential of a single

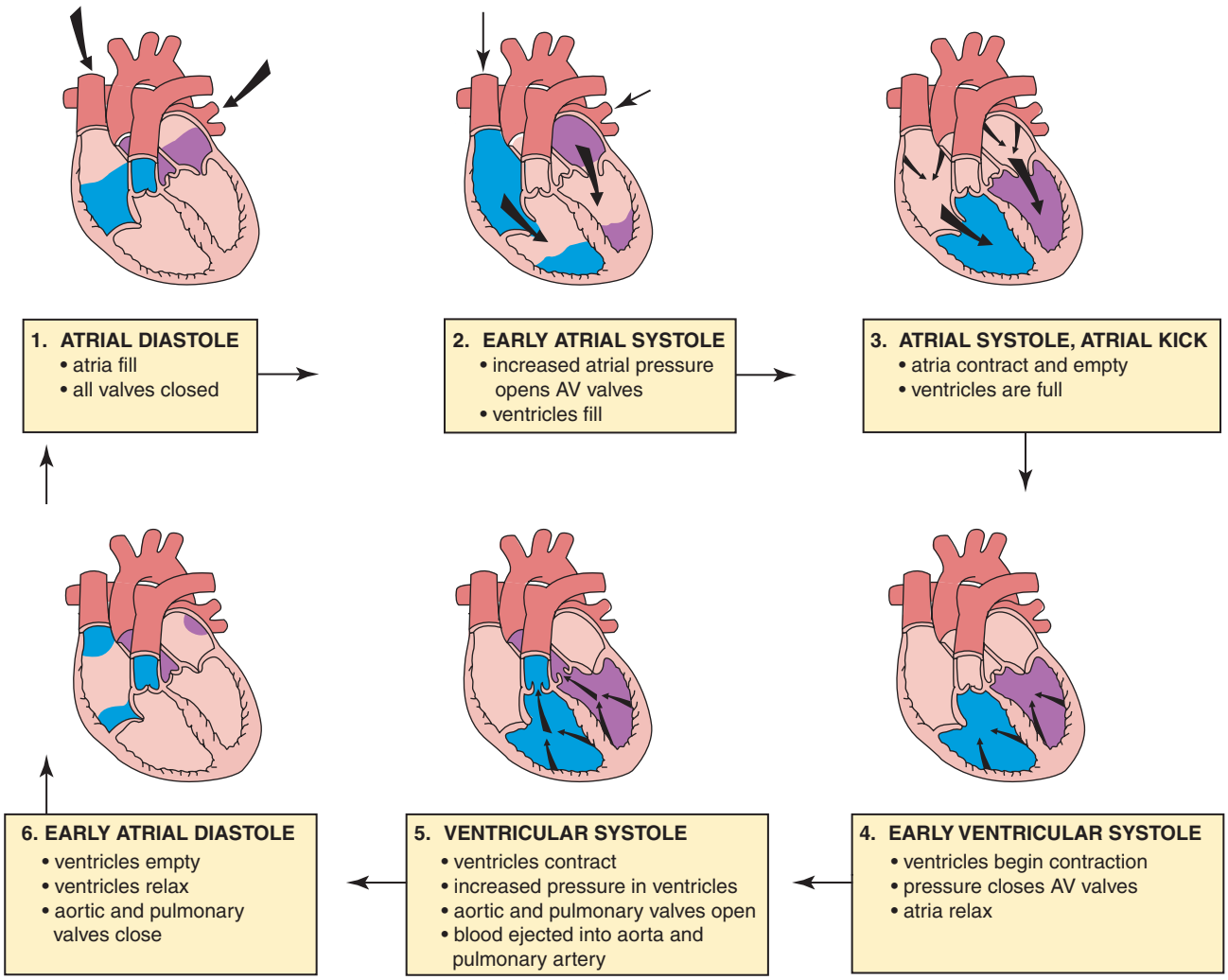


FIGURE 7-2 The cardiac cycle. (Modified from Wesley K. *Huszar's Basic Dysrhythmias and Acute Coronary Syndromes: Interpretation and Management*. 4th ed. St. Louis: Mosby JEMS; 2010.)

cell, imagine that this is occurring in millions of cardiac cells almost simultaneously resulting in a coordinated contraction of the atria and ventricles.

During the resting state of the cell, there is a difference in polarity, or charge, between the extracellular and intracellular environments that is maintained by the cell membrane. Specialized pumps prevent ions from passing through the cell membrane by diffusion. The inside of the cell is predominately negatively charged, whereas the outside is positively charged. The resting membrane potential occurs when the cell is in the polarized or resting state. The polarized cell has a higher concentration of positive ions including sodium outside the cell, causing the extracellular environment to be positive. The interior of the cell is more negative and the concentration of potassium is higher (Figure 7-3). The voltage in the interior of the cardiac muscle cell during resting membrane potential is −90 mV, whereas that of the pacemaker cells in the SA and AV nodes is −65 mV.

The stimulation of a cardiac muscle cell by an electrical impulse changes the permeability of the myocardial cell membrane. Sodium ions rush into the cell via sodium channels in the cell membrane, and potassium ions flow out of the cell, resulting in a more positively charged cell interior. The action potential describes the flow of ions inside and outside the cell as well as the voltage changes that occur. The first phase of the action potential occurs when the cell membrane becomes permeable to sodium molecules. When the membrane potential reaches −65 mV, also known as threshold, more channels in the cell membrane open up and allow sodium ions to rush into the cell; the cell interior quickly reaches +30 mV, resulting in depolarization. Following this fast phase in sodium influx, the plateau phase of the action potential occurs when calcium channels open and calcium flows into the cell. This slower phase allows for a longer period of depolarization, resulting in sustained muscle contraction. The next event of the action potential occurs when the cell returns to resting state. This process is called *repolarization* and results from ions returning to the outside (calcium and sodium) and the interior of the cell (potassium). Sodium and potassium pumps within the cell membrane maintain this concentration gradient across the cell membrane when the cell is polarized. These pumps require energy in the form of adenosine triphosphate (ATP). Now the cell has returned to its resting state with a polarity of −90 mV once again. *Depolarization* of adjacent cells occurs simultaneously as the stimulus moves across the cardiac muscle allowing for almost instantaneous depolarization of the entire muscle mass and resultant contraction (Figure 7-4).

Pacemaker cells exhibit the property of automaticity, enabling these cells to reach threshold and depolarize without an outside stimulus. The cell membrane becomes suddenly permeable to sodium during the resting state and reaches threshold, resulting in spontaneous depolarization. Resting membrane potential for these automatic cells is −65 mV, and threshold is reached at approximately −50 mV.[13] The sinus node reaches threshold at a rate of 60 to 100 times per minute. Because this is the fastest pacemaker in the heart, the SA node is the dominant pacemaker of the heart. The AV node and His-Purkinje pacemakers are latent pacemakers that reach threshold at a slower rate but can take over if the SA node fails or if sinus impulse conduction is blocked. The AV node has an inherent rate of 40 to 60 beats per minute and the His-Purkinje system can fire at a rate of 20 to 40 beats per minute.

Autonomic Nervous System

The rate of spontaneous depolarization of the pacemaker cells is influenced by the autonomic nervous system. The

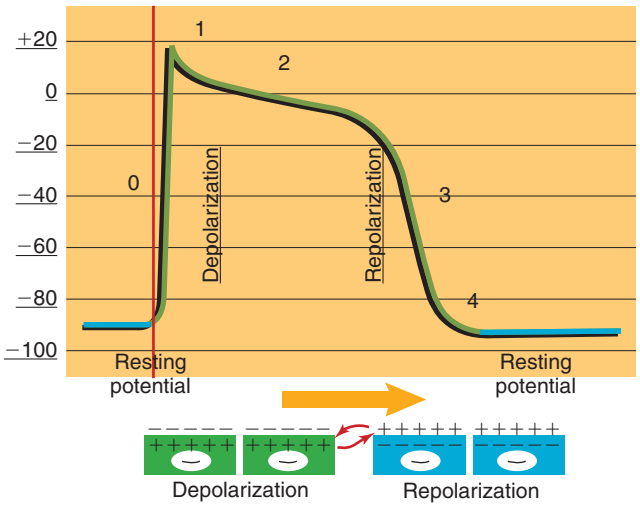


FIGURE 7-3 Cardiac action potential. (From Association of Critical-Care Nurses: *Essentials of Critical Care Orientation 2.0.*)

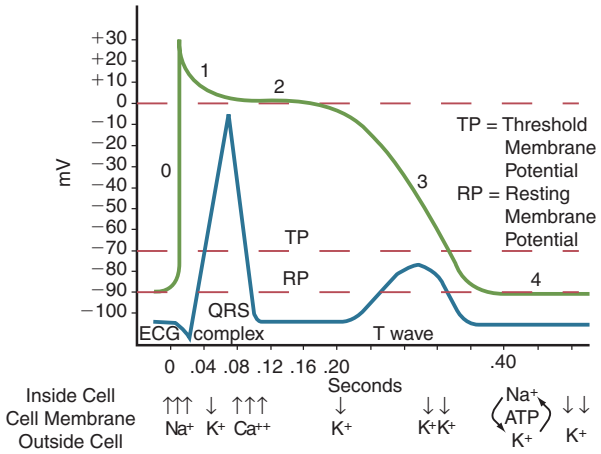


FIGURE 7-4 Cardiac action potential with the electrocardiogram and movement of electrolytes. *ATP,* Adenosine triphosphate; *Ca,* calcium; *K,* potassium; *Na,* sodium. (From American Heart Association. *Advanced Cardiac Life Support Textbook.* Dallas: Author; 1997.)

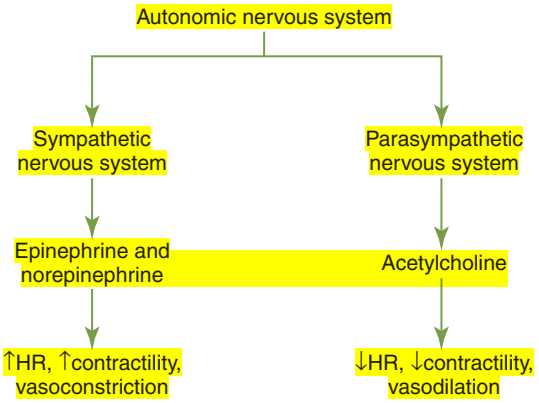


FIGURE 7-5 Autonomic nervous system.

sympathetic nervous system releases catecholamines, causing the SA node to fire more quickly in response to epinephrine and norepinephrine. The parasympathetic nervous system releases acetylcholine, which slows the heart rate. During normal circumstances these substances modulate each other, and the cardiac response allows for appropriate changes in cardiac output to meet the varying demands of the body (Figure 7-5).

THE 12-LEAD ELECTROCARDIOGRAM

The 12-lead ECG is an important diagnostic tool that provides information about myocardial ischemia, injury, cell necrosis, electrolyte disturbances, increased cardiac muscle mass (hypertrophy), conduction abnormalities, and abnormal heart rhythms.

Electrodes applied to the skin transmit the electrical signals of the movement of the cardiac impulse through the conduction system. This signal passes through skin, muscle, bone, and finally through electrodes and wires to be amplified by the ECG machine and either transcribed to ECG paper or displayed digitally. The ECG machine records the summation of the waves of depolarization and repolarization occurring during the cardiac cycle. During the polarized or resting state, a flat or *isoelectric* line is inscribed that means no current or electrical activity is occurring.

The 12-lead ECG provides a view of the electrical activity of the heart from 12 different views or angles, both frontally and horizontally. Cardiac electrical activity is not one-dimensional; thus observation in two planes provides a more complete view in the horizontal and vertical planes. When assessing the 12-lead ECG or a rhythm strip, it is helpful to understand that the electrical activity is viewed in relation to the positive electrode of that particular lead. The positive electrode is the "viewing eye" of the camera. When an electrical signal is aimed directly at the positive electrode, an upright inflection is visualized. If the impulse is going away from the positive electrode, a negative deflection is seen; and if the signal is perpendicular to the imaginary line between the positive and negative poles of the lead, the tracing is equiphasic, with equally positive and negative deflection (Figure 7-6). A tracing may be observed on a monitor, displayed digitally on a computer screen, or recorded on paper.

The electrical activity of normal conduction occurs downward between the left arm and the left leg, called the *mean cardiac vector* or direction of current flow. Thus the positive electrode reflects this electrical activity by an upright inflection if the flow of current is directed at that positive electrode, or negative deflection if moving away from that positive electrode. The vector of a lead is an imaginary line between the positive and negative electrodes. The wave of current flow of the cardiac cycle or the vector is inscribed on the ECG paper in relation to the lead vector that is being viewed. The lead reflects the magnitude and the direction of current flow (Figure 7-7).

The 12-lead ECG consists of three standard bipolar limb leads (I, II, and III), three augmented unipolar limb leads (aV_R, aV_L, and aV_F), and six precordial unipolar leads (V_1, V_2, V_3, V_4, V_5, and V_6). Bipolar leads consist of a positive and a negative lead, whereas the unipolar leads consist of a positive electrode and the ECG machine itself.

Standard Limb Leads

The standard three limb leads are I, II, and III. Limb leads are placed on the arms and legs. These leads are bipolar, meaning that a positive lead is placed on one limb and a negative lead on another.

Lead I records the magnitude and direction of current flow between the negative lead on the right arm to the positive lead on the left arm. Lead II records activity between the negative lead on the right arm and the positive lead on the left leg. Lead III records activity from the negative lead on the left arm to the positive lead on the left leg (Figure 7-8). The normal ECG waveforms are upright in these leads, with lead II producing the most upright waveforms.

The bipolar limb leads form Einthoven's triangle (Figure 7-9). This is an equilateral upside-down triangle with the heart in the center.

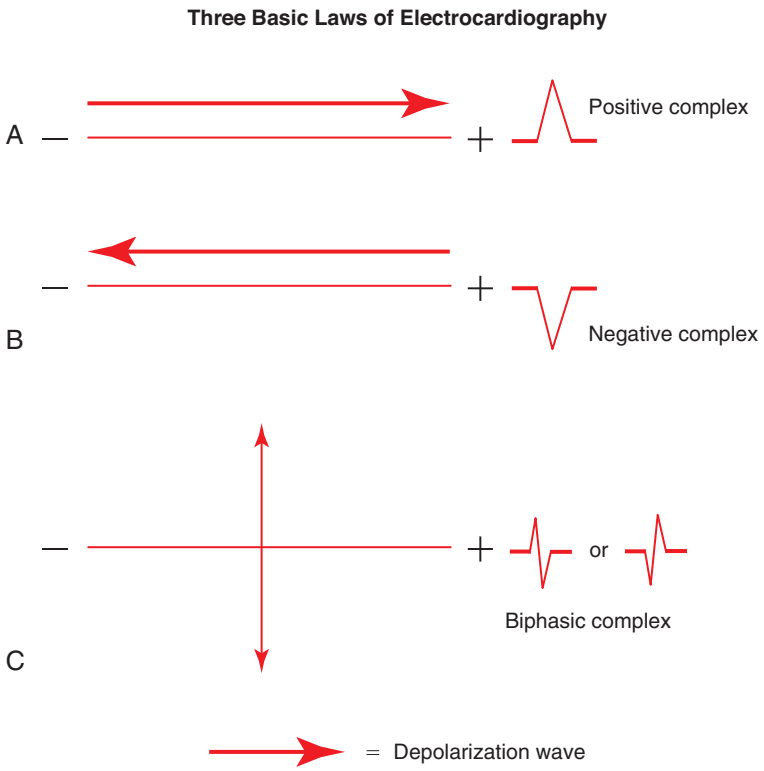


FIGURE 7-6 **A,** A positive complex is seen in any lead if the wave of depolarization spreads toward the positive pole of the lead. **B,** A negative complex is seen if the depolarization wave spreads toward the negative pole (away from the positive pole) of the lead. **C,** A biphasic (partly positive, partly negative) complex is seen if the mean direction of the wave is at right angles. These apply to the P wave, QRS complex, and T wave. (From Goldberger AL. *Clinical Electrocardiography: A Simplified Approach*. 7th ed., St. Louis: Mosby; 2006.)

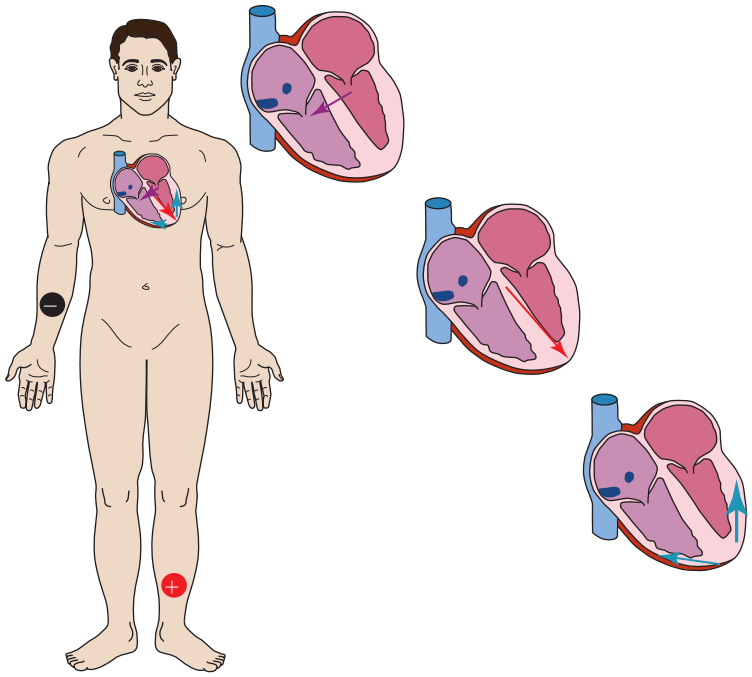


FIGURE 7-7 Direction of normal current flow through the ventricles.

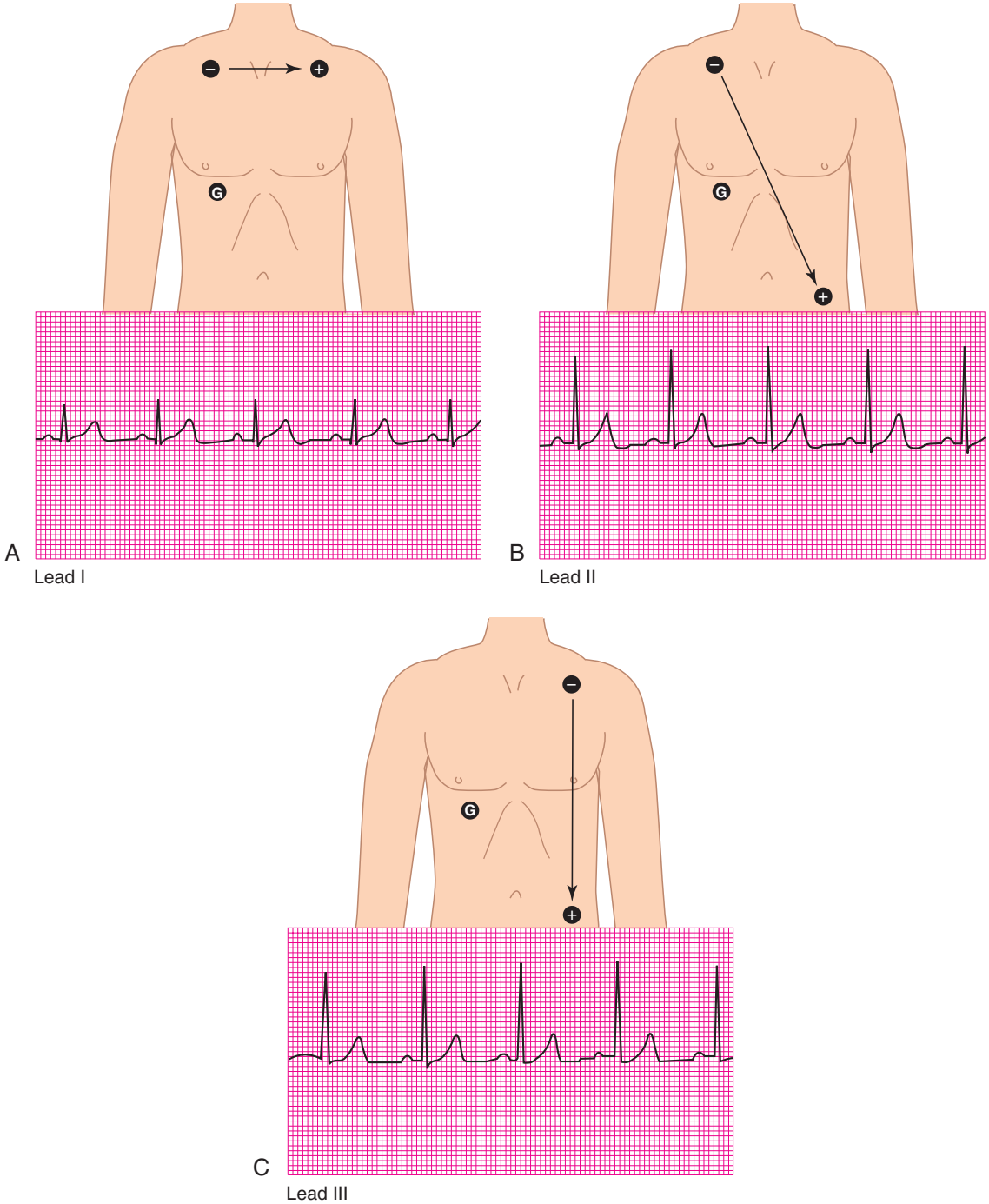


FIGURE 7-8 Standard bipolar limb leads. **A,** Lead I. **B,** Lead II. **C,** Lead III.

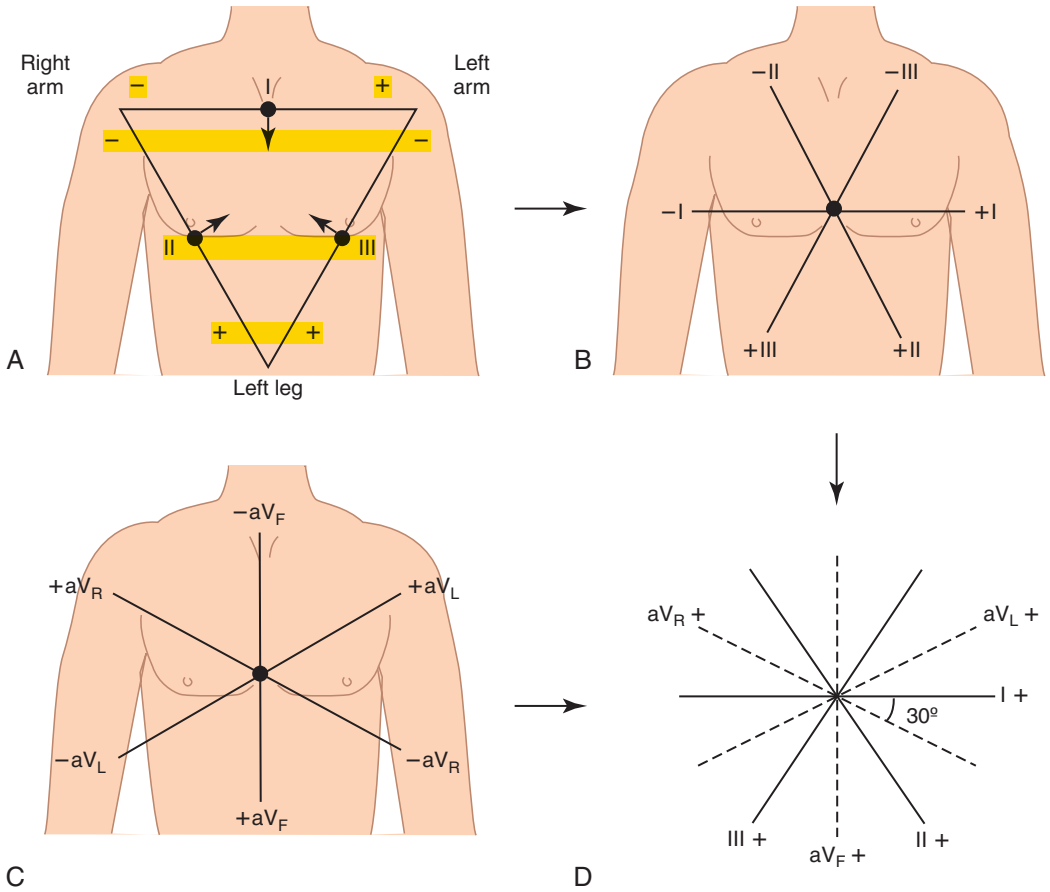


FIGURE 7-9 **A,** Einthoven's triangle. **B,** The triangle is converted to a triaxial diagram by shifting leads I, II, and III so that they intersect at a common point. **C,** Triaxial lead diagram showing the relationship of the three augmented (unipolar) leads, aV_R, aV_L, and aV_F. Notice that each lead is represented by an axis with a positive and negative pole. **D,** The hexaxial reference figure

Augmented Limb Leads

The augmented limb leads are unipolar, meaning that they record electrical flow in only one direction. A reference point is established in the ECG machine, and electrical flow is recorded from that reference point toward the right arm (aV_R), the left arm (aV_L), and the left foot (aV_F) (see Figure 7-9, *A-C*). The *a* in the names of these leads means augmented; because these leads produce small ECG complexes, they must be augmented or enlarged. The *V* means voltage and the subscripts *R, L,* and *F* stand for right arm, left arm, and left foot, where the positive electrode is located. The augmented limb leads are displayed by using the electrodes already in place for the limb leads.

The addition of the augmented limb leads to Einthoven triangle form a hexaxial reference figure when the six frontal plane leads are intersected in the center of each lead (see Figure 7-9, *D*). The figure is used to determine the exact direction of current flow called axis determination, a requisite skill of 12-lead ECG analysis. Assessment of axis deviation is an advanced skill and not addressed in this chapter. Figure 7-9 demonstrates that leads I and aV_L are close in proximity, as are leads II, III, and aV_F. Therefore the QRS patterns of leads that are close together appear similar. Because current flow is directed between the left arm and left foot, leads I, II, III, aV_L, and aV_F are usually positive if conduction is normal.

Precordial Leads

The six *precordial leads* (also called chest leads) are positioned on the chest wall directly over the heart. These leads provide a view of cardiac electrical activity from a horizontal plane rather than the frontal plane view of the limb leads. Precise placement of these leads is crucial for providing an accurate representation and for comparing with previous and future ECGs. A misplaced V lead can result in erroneous or missed diagnoses of acute coronary syndrome and lethal dysrhythmias. The precordial leads are unipolar, with a positive electrode and the AV node as a center reference (Figure 7-10). Landmarks for placement of these leads are the intercostal spaces, the sternum, and the clavicular and axillary lines. Positions for these six leads are as follows:

V_1: Fourth intercostal space, right sternal border
V_2: Fourth intercostal space, left sternal border
V_3: Halfway between V_2 and V_4
V_4: Fifth intercostal space, left midclavicular line

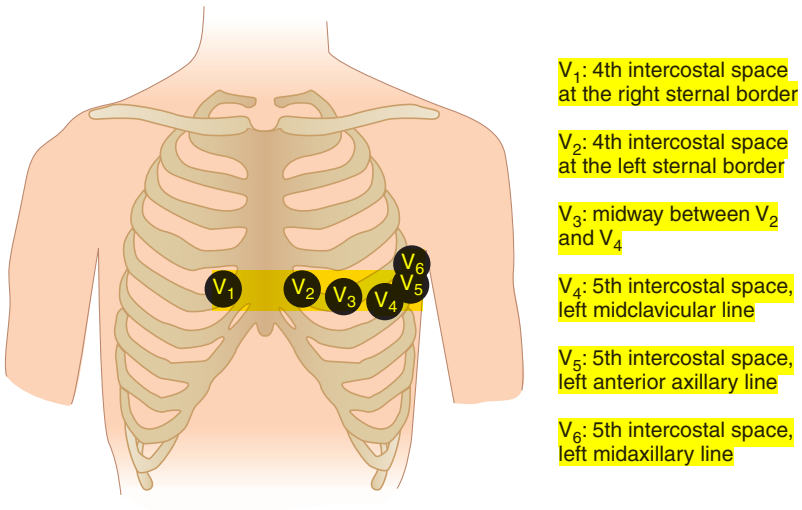


FIGURE 7-10 Precordial chest leads.

V_5: Fifth intercostal space, left anterior axillary line
V_6: Fifth intercostal space, left midaxillary line

Grouping of Leads

Each lead provides a view of the electrical activity of the heart from a different angle. Leads that view the current flow in the heart from the same angle can be grouped together. Anatomical regions are described as septal, anterior, lateral, inferior, and posterior. Septal leads are V_1 and V_2; anterior leads are V_3 and V_4; lateral leads are V_5, V_6, I, and aV_L; and inferior leads are II, III, and aV_F.[3] Assessing leads that localize these regions of the heart assists in identifying the location of myocardial ischemia, injury, and infarct. Posterior and right ventricular electrodes are not commonly part of the standard 12-lead ECG; however, if indicated, newer ECG machines can record tracings from these areas. The 15-lead ECG is an essential additional assessment that is made if the patient is suspected of having an inferior myocardial infarction, because right ventricular and posterior infarctions are common with this type of infarct (Figure 7-11).

Continuous Cardiac Monitoring

Continuous cardiac monitoring is conducted in a variety of patient care settings, including the emergency department, ambulances, high-risk obstetrical units, cardiac catheterization and electrophysiology laboratories, critical care units, operating rooms, postanesthesia care units, endoscopy suites, and progressive care units. Depending upon the sophistication of the monitoring system, any of the 12 leads can be monitored continuously. The most critical initial elements of cardiac monitoring are in skin preparation, lead placement, and appropriate lead selection.

Skin Preparation and Lead Placement

Adequate skin preparation of electrode sites requires clipping the hair, cleansing the skin, and drying vigorously. Cleansing includes washing with soap and water, or alcohol, to remove skin debris and oils.

The three-lead monitoring system depicts only the standard limb leads. These leads are marked as RA, LA, and LL. The left and right arm leads (RA and LA) are placed just below the right and left clavicle, and the leg lead (LL) is placed on the left abdominal area below the level of the umbilicus (Figure 7-12). Five-lead monitoring systems are available on many systems, and they monitor all of the limb leads and one chest lead. Instead of placing the limb leads on the arms and legs, these leads are placed just below the right and left clavicles and on the right and left abdomen below the level of the umbilicus. The precordial or chest lead is placed in the selected V lead position, usually V_1 (see Figure 7-12) Some five-lead systems have the ability to derive a 12-lead tracing (Figure 7-13).

Before application, the electrodes are checked to ensure that the gel is moist. The electrode is attached to the lead wire and placed in the designated location. Following electrode placement, the signal is assessed to insure that the waveform is clear and not disrupted by artifact. The frequency of changing electrodes is based on institutional policy. Nursing assessment must include that electrodes are placed in the correct anatomical positions at the beginning of each shift.

Lead selection is determined by the patient's diagnosis and based on risk for an ischemic cardiac event, dysrhythmia, or other factors. Typically the first lead selected is V_1 for dysrhythmia monitoring. If the system is able to simultaneously monitor a second lead, selection of this lead is based on the patient's diagnosis and individual needs. A limb lead is usually selected, such as III or II, because of the easy visualization of P waves; however, if the patient has a history of ischemia, the second lead can be based on the patient's 12-lead ECG, identifying the lead showing the greatest ischemic change. For dysrhythmia monitoring in a system allowing for continuous monitoring of two leads, V_1 and III are the standard recommendations.[1,4,5]

In most settings a 6-second strip of the patient's rhythm is obtained and documented in the patient's chart at intervals from every 4 hours to 8 hours, based on the patient acuity level and institutional policy. In addition to scheduled times, a rhythm strip is documented any time there is a change in

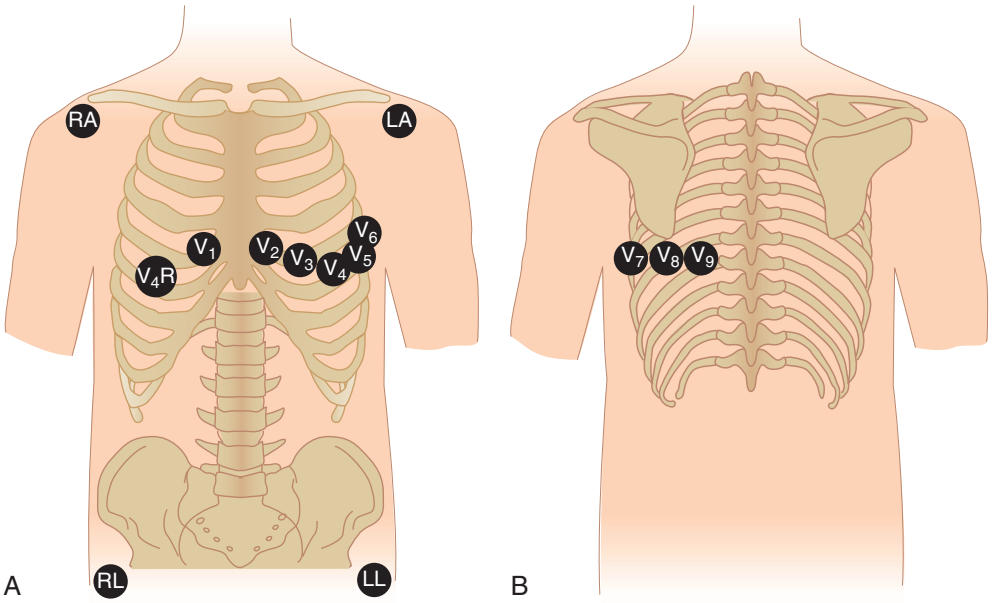


FIGURE 7-11 Lead placement for a 15-lead ECG. **A,** Anterior leads. **B,** Posterior leads.

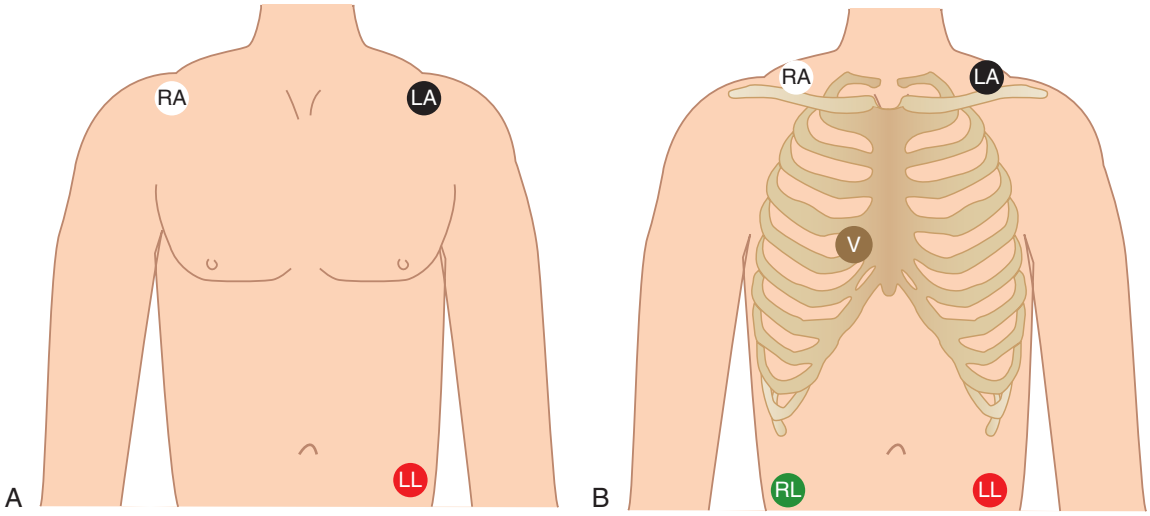


FIGURE 7-12 **A,** Three-lead ECG monitoring system. **B,** Five-lead monitoring system with V lead placed in V_1 position.

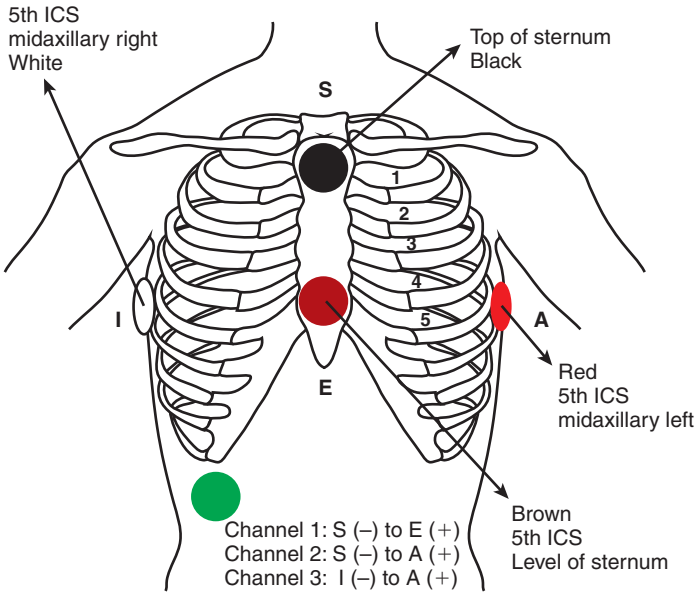


FIGURE 7-13 EASI Monitoring System. (Courtesy Philips Healthcare, Andover, Massachusetts.)

rhythm. If the patient experiences chest discomfort or other signs of myocardial ischemia or a dysrhythmia, a 12-lead ECG is performed. Many ECG machines can print a continuous 12-lead rhythm recording, allowing for assessment of a dysrhythmia from 12 different views. This is a helpful tool when diagnosing heart block, atrial dysrhythmia, or wide QRS complex tachycardia.

ST-segment monitoring allows for continuous monitoring for changes in the ST segment that may reflect myocardial ischemia.[11] Early recognition of new ST segment elevation or depression facilitates earlier intervention, thus preserving myocardium. Leads III and V_3 have been recommended as the monitoring leads best used to recognize ischemic changes in patients at risk.[2,4,6,11,12] Such patient populations include those with acute coronary syndrome, patients at risk for silent ischemia, and patients who have previously had cardiac interventions such as angioplasty and stent placement.

BASICS OF DYSRHYTHMIA ANALYSIS

Measurements

ECG paper contains a standardized grid where the horizontal axis measures time and the vertical axis measures voltage or amplitude (Figure 7-14). Larger boxes are circumscribed by

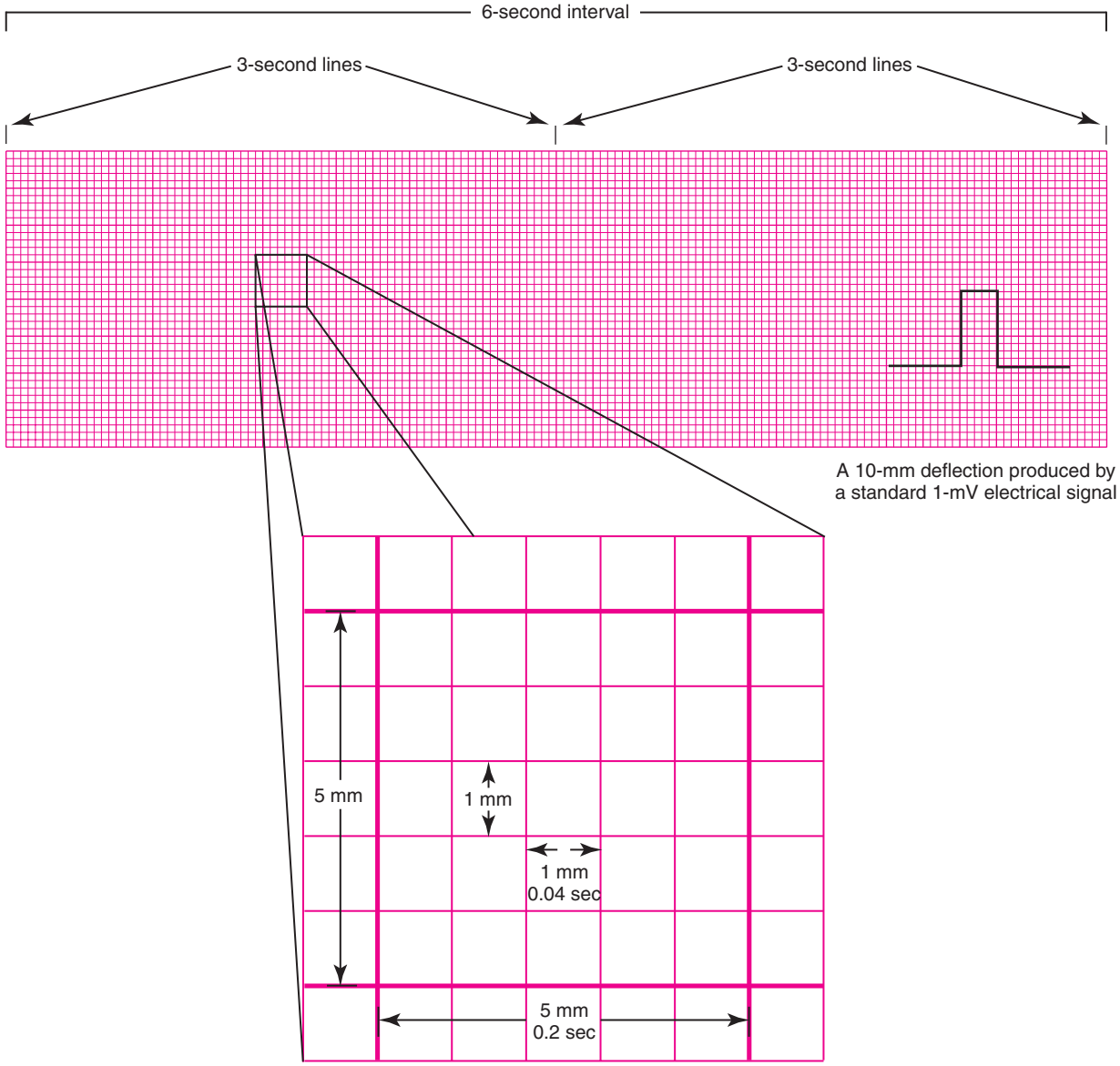


FIGURE 7-14 ECG paper records time horizontally in seconds or milliseconds. Each large box contains 25 smaller boxes with five on the horizontal axis and five on the vertical axis. Each small horizontal box is 0.04 seconds while each large box is 0.20 seconds in duration. Vertically, the graph depicts size or voltage in millivolts and in millimeters. 15 large boxes equals 3 seconds and 30 large boxes equals 6 seconds used in calculating heart rate. (From Wesley K. *Huszar's Basic Dysrhythmias and Acute Coronary Syndromes: Interpretation and Management.* 4th ed. St. Louis: Mosby JEMS; 2010.)

darker lines and the smaller boxes by lighter lines. The larger boxes contain 5 smaller boxes on the horizontal line and 5 on the vertical line for a total of 25 per large box. Horizontally, the smaller boxes denote 0.04 seconds each or 40 milliseconds; the larger box contains five smaller horizontal boxes and thus equals 0.20 seconds or 200 milliseconds. Along the uppermost aspect of the ECG paper are vertical hatch marks that occur every 15 large boxes. The area between these marks equals 3 seconds. Some ECG paper has markings every second.

The monitoring standard is to use 6-second rhythm strips for analysis and documentation of cardiac rhythms. A 6-second strip consists of two 3-second intervals or a span of three hatch marks. The measurement of time on the ECG tracing represents the speed of depolarization and repolarization in the atria and ventricles and is printed at 25 mm/sec.

Amplitude is measured on the vertical axis of the ECG paper (see Figure 7-14). Each small box is equal to 0.1 mV in amplitude. Waveform amplitude indicates the amount of electrical voltage generated in the various areas of the heart. Low-voltage and small waveforms are expected from the small muscle mass of the atria. Large-voltage and large waveforms are expected from the larger muscle mass of the ventricles.

Waveforms and Intervals

The normal ECG tracing is composed of P, Q, R, S, and T waves (Figure 7-15). These waveforms rise from a flat baseline called the *isoelectric line*.

P Wave

The P wave represents atrial depolarization. It is usually upright in leads I and II and has a rounded, symmetrical shape. The amplitude of the P wave is measured at the center of the waveform and normally does not exceed three boxes, or 3 mm, in height.

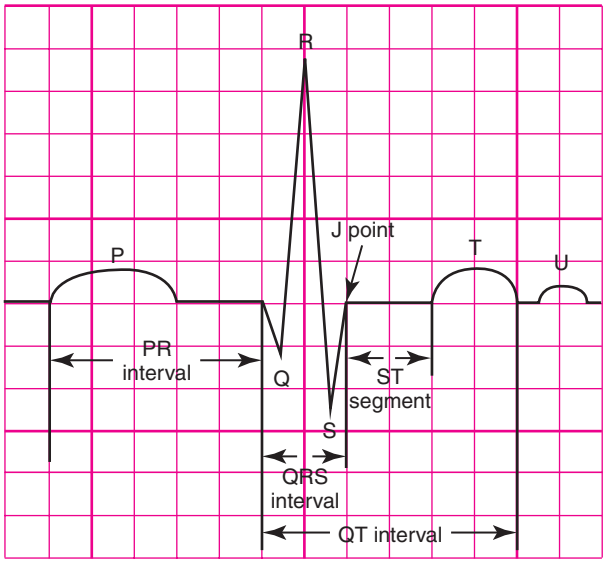


FIGURE 7-15 ECG waveforms.

Normally a P wave indicates that the SA node initiated the impulse that depolarized the atrium. However, a change in the shape of the P wave may indicate that the impulse arose from a site in the atria other than the SA node.

PR Interval

The downslope of the P wave returns to the isoelectric line for a short time before the beginning of the QRS complex. The interval from the beginning of the P wave to the next deflection from the baseline is called the PR interval. The PR interval measures the time it takes for the impulse to depolarize the atria, travel to the AV node, and dwell there briefly before entering the bundle of His. The normal PR interval is 0.12 to 0.20 seconds, three to five small boxes wide (see Figure 7-15). When the PR interval is longer than normal, the speed of conduction is delayed in the AV node. When the PR interval is shorter than normal, the speed of conduction is abnormally fast.

QRS Complex

The QRS complex represents ventricular depolarization (see Figure 7-15). Atrial repolarization also occurs simultaneously to ventricular depolarization, but because of the larger muscle mass of the ventricles, visualization of atrial repolarization is obscured by the QRS complex. The classic QRS complex begins with a negative, or downward, deflection immediately after the PR interval. The first negative deflection after the P wave is called the Q wave.

A Q wave may or may not be present before the R wave. If the first deflection from the isoelectric line is positive, or upright, the waveform is called an R wave. The size of the R wave varies across leads. The R wave is positive and tall in those leads where the direction of current is going towards the positive electrode lead. All the limb leads, with the exception of aV_R, normally have tall R waves. In the precordial leads, the R wave begins small and progressively becomes larger and more positive, going from small in V_1 to a maximal size in V_5. This change in size is termed R wave progression and occurs because the direction of current flow is moving more directly toward the positive electrode of V_4. (Figure 7-16).

The S wave is a negative waveform that follows the R wave. The S wave deflects below the isoelectric line. Some patients may have a second positive waveform in their QRS complex. If so, then that second positive waveform is called *R prime (R′)*.

The term *QRS complex* is a generic term designating the waveforms representing ventricular depolarization. In reality, the complex may be an R wave, a QS wave, or other wave, depending on the lead viewed or any abnormalities that are present. Figure 7-17 depicts the various shapes of the QRS complex and their nomenclature.

If a Q wave is present on the 12-lead ECG (not the cardiac monitor), it must be determined if it is pathological or normal. A pathological Q wave has a width of 0.04 seconds and a depth that is greater than one fourth of the R wave amplitude. Pathological Q waves are found on ECGs of individuals

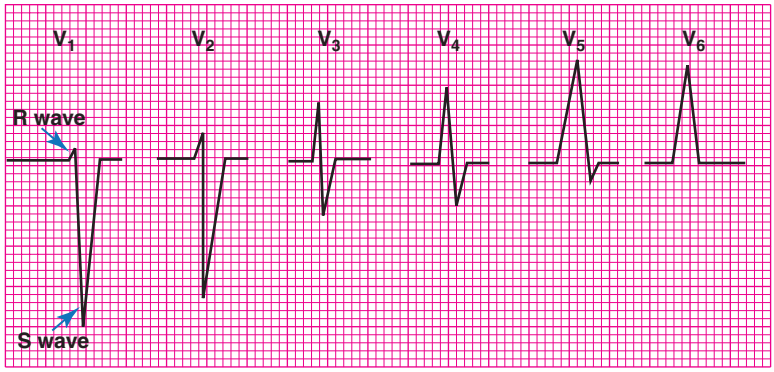


FIGURE 7-16 The normal 12-lead precordial R wave progression.

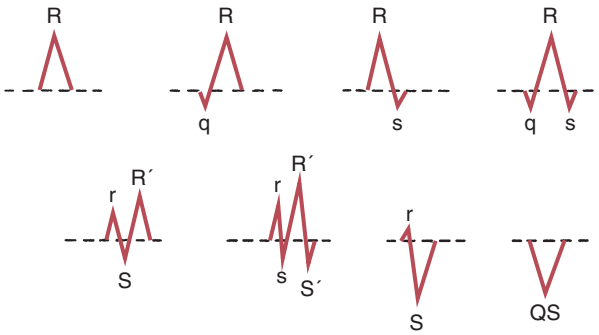


FIGURE 7-17 Nomenclature for QRS Complexes of Various Shapes. Different types of QRS complexes. An R wave is a positive waveform. A negative deflection before the R wave is a Q wave. The S wave is a negative deflection after the R wave. If the waveform is tall or deep, the letter naming the waveform is a capital letter. If the waveform is small in either direction, the waveform is labeled with a lowercase letter.

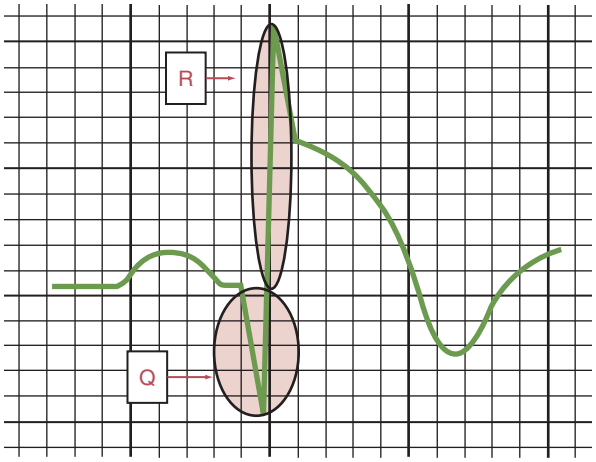


FIGURE 7-18 Pathological Q wave is greater than one fourth the height of the R wave.

who have had myocardial infarctions, and they represent myocardial muscle death (Figure 7-18).

QRS Interval

The QRS interval is measured from where it leaves the isoelectric line of the PR interval to the end of the QRS complex (see Figure 7-15). The waveform that initiates the QRS complex (whether it is a Q wave or an R wave) marks the beginning of the interval. The normal width of the QRS complex is 0.06 to 0.10 seconds. This width equals 1.5 to 2.5 small boxes.

A QRS width greater than 0.10 seconds may signify a delay in conduction through the ventricles due to a variety of factors, including myocardial infarction, atherosclerosis of the aging conduction system, or cardiomyopathy.

T Wave

The T wave represents ventricular repolarization (see Figure 7-15). T-wave amplitude is measured at the center of the waveform and is usually no higher than five small boxes, or 5 mm. In contrast to P waves, which are usually symmetrical, T waves are usually asymmetrical. Changes in T-wave amplitude or direction can indicate electrical disturbances resulting from an electrolyte imbalance, or myocardial ischemia or injury. For example, hyperkalemia can cause tall, peaked T waves, and ischemia may cause an inverted or upside-down T wave.

Some students who are novices in dysrhythmia interpretation have difficulty differentiating the P wave and the T wave. Understanding that the P wave normally precedes the QRS complex and the T wave normally follows the QRS complex aids in identification of these waveforms. In addition, the T wave usually has greater width and amplitude than the P wave, because the atria are smaller muscle masses and therefore produce smaller waveforms than do the larger ventricles.

ST Segment

The ST segment connects the QRS complex to the T wave, and is usually isoelectric, or flat. However, in some conditions the segment may be depressed (falling below baseline) or elevated (rising above baseline). The point at which the QRS complex ends and the ST segment begins is called the J (junction) point. ST-segment change is measured 0.04 seconds after the J point. To identify ST-segment elevation, use the isoelectric portion of the PR segment as a reference for baseline. Next, note whether the ST segment is level with the PR segment (see Figure 7-15). If the ST segment is above or

EVIDENCE-BASED PRACTICE

Should QT Interval Monitoring be Part of Nursing Care?

Problem
QT-interval monitoring is recommended by the American Heart Association (AHA) for several conditions: administration of medications that prolong the QT interval, overdose of medications that prolong the QT interval, electrolyte disturbances (K^+, Mg^+), and bradycardia. It is not known what proportion of patients meet these criteria and would benefit from QT interval monitoring.

Clinical Question
What proportion of critically ill patients meet the AHA criteria for QT interval monitoring?

Evidence
The researchers collected data on 1039 critically ill patients who were monitored with a system that provided continuous QT interval monitoring. A QT interval greater than 500 milliseconds for 15 minutes or longer was considered to be prolonged. All electronic data were validated by the investigators. They found that 69% of patients had at least one of the AHA criteria for monitoring. They also found that the odds for QT interval prolongation increased with the number of AHA conditions present.

Implications for Nursing
Findings of this study reinforce the need for QT interval monitoring in critically ill patients, because the majority of patients had at least one risk factor for prolonged QT intervals. As monitoring systems include more features such as QT interval monitoring, it is also important for nurses to be aware of the features available and to use them as part of routine patient monitoring.

Level of Evidence
C—Descriptive Study

Reference
Pickham, D., Helfenbein, E., Shinn, J.A., Chan, G., Funk, M., & Drew, B.J. (2010). How many patients need QT interval monitoring in critical care units? Preliminary report of the QT in practice study. *Journal of Electrocardiology,* 43, 572-576.

below the baseline, count the number of small boxes above or below at 0.04 seconds after the J point. A displacement in the ST segment can indicate myocardial ischemia or injury.[2,5,11,12] If ST displacement is noted and is a new finding, a 12-lead ECG is performed and the provider notified. The patient is assessed for signs and symptoms of myocardial ischemia.

QT Interval

The QT interval is measured from the beginning of the QRS complex to the end of the T wave (see Figure 7-15). This interval measures the total time taken for ventricular depolarization and repolarization. Abnormal prolongation of the QT interval increases vulnerability to lethal dysrhythmias, such as ventricular tachycardia and fibrillation. Normally, the QT interval becomes longer with slower heart rates and shortens with faster heart rates, thus requiring a correction of the value. Generally, the QT interval is less than half the RR interval (see Figure 7-15).

A preferred calculation that corrects for varying heart rates is a calculated QT interval, or QTc, based on the QT divided by the square root of the R to R interval. QT and QTc are routinely measured when analyzing a rhythm strip. Normal QTc is less than 0.47 seconds in males and 0.48 seconds in females. Many monitoring systems can calculate the QTc if the R to R interval is measured. QTc accuracy is based on a regular rhythm. In irregular rhythms such as atrial fibrillation, an average QTc may be necessary because the QT varies beat to beat.

Risk of a lethal heart rhythm called *torsades de pointes* occurs if QTc is prolonged greater than 0.50 seconds.[5,6] Many medications may precipitate prolonged QT, thus it is important to be vigilant about monitoring for a prolonged QT interval (see box, Evidence-Based Practice).

U Wave

A final waveform that is occasionally noted on the ECG is the U wave. If present, this waveform follows the T wave, and it represents repolarization of a small segment of the ventricles. The U wave is usually small, rounded, and less than 2 mm in height (see Figure 7-15).[13] Larger U waves may be present in patients with hypokalemia, cardiomyopathy, and digoxin toxicity.

CAUSES OF DYSRHYTHMIAS

Dysrhythmias may occur when automaticity of the normal pacemaker cells of the heart is either stimulated or suppressed. For example, if the SA node fails to fire, latent pacemakers from the AV node or ventricles may fire as a backup safety mechanism. The SA node may fire more rapidly because of the influence of circulating catecholamines. Cells either within or outside the normal conduction system may take on characteristics of pacemaker cells and begin firing because of electrolyte imbalances, ischemia, injury, necrosis, and myocardial stretch due to hypertrophy. *Ectopic beats* or *ectopic rhythms* arise from cells that normally do not have pacemaker capabilities. Slowed conduction can create alternative conductive pathways that produce abnormally fast heart rhythms. If conduction is sufficiently decreased, latent pacemakers may take over this function. Table 7-1 presents a list of antidysrhythmic drugs and their effects on changes in heart rate and rhythm.

TABLE 7-1 PHARMACOLOGY

Antidysrhythmic Drug Classifications

CLASS*	DESCRIPTION	EXAMPLES
IA	Inhibits the fast sodium channel Prolongs repolarization time Used to treat atrial and ventricular dysrhythmias	Quinidine, disopyramide, procainamide
IB	Inhibits the fast sodium channel Shortens the action potential duration Used to treat ventricular dysrhythmias only	Lidocaine, phenytoin, mexiletine, tocainide
IC	Inhibits the fast sodium channel Shortens the action potential duration of only Purkinje fibers Controls ventricular tachydysrhythmias resistant to other drug therapies Has proarrhythmic effects	Flecainide, propafenone
II	Causes beta-adrenergic blockade	Esmolol, propranolol, sotalol
III	Lengthens the action potential Acts on the repolarization phase	Amiodarone, sotalol, dofetilide; ibutilide
IV	Blocks the slow inward movement of calcium to slow impulse conduction, especially in the atrioventricular node Used for treatment of supraventricular tachycardias	Diltiazem, verapamil
IVb-like	Opens the potassium channel	Adenosine, ATP

*Class I, sodium channel blockers; Class II, beta-adrenergic blockers; Class III, potassium channel blockers; Class IV, calcium channel blockers. Adapted from Skidmore-Roth, L. (2011). *Mosby's Nursing Drug Reference.* (24th ed.). St. Louis: Elsevier Mosby.

DYSRHYTHMIA ANALYSIS

Analysis of a cardiac rhythm must be conducted systematically to correctly interpret the rhythm. Proper dysrhythmia analysis includes assessment of the following:

- Atrial and ventricular rates
- Regularity of rhythm
- Measurement of PR, QRS, and QT/QTc intervals
- Shape or morphology of waveforms and their consistency
- Identification of underlying rhythm and any noted dysrhythmia
- Patient tolerance of rhythm
- Clinical implication of the rhythm

Rate

The rate represents how fast the heart is depolarizing. Under normal conditions, the atria and the ventricles depolarize in a regular sequence. However, each can depolarize at a different rate. P waves are used to calculate the atrial rate, and QRS waves or R waves are used to calculate the ventricular rate. Rate can be assessed in various ways that are described as follows.

Six-second method: A quick and easy estimate of heart rate can be accomplished by counting the number of P waves or QRS waves within a 6-second strip to obtain atrial and ventricular heart rates per minute. This is the optimal method for irregular rhythms. Identify the lines above the ECG paper that represent 6 seconds, and count the number of P waves within the lines; then add a zero to identify the atrial heart rate estimate for 1 minute. Next, identify the number of QRS waves in the 6-second strip and again add a zero to identify the ventricular rate (Figure 7-19).

Large box method: In this method, two consecutive P and QRS waves are located. The number of large boxes between the highest points of two consecutive P waves is counted, and that number of large boxes is divided into 300 to determine the atrial rate. The number of large boxes between the highest points of two consecutive QRS waves is counted, and that number of large boxes is divided into 300 to determine the ventricular rate (Figure 7-20). This method is accurate only if the rhythm is regular. If one large box is between the two QRS waves, the rate is 300 beats per minute (300 ÷ 1 = 300); if there are two large boxes, the heart rate is 150 beats per minute (300 ÷ 2 = 150); if there are three large boxes, the heart rate is 100 beats per minute (300 ÷ 3 = 100); if there are four large boxes, the heart rate is 75 beats per minute (300 ÷ 4 = 75), and so on. A simple mnemonic can be used to simplify this method. Memorize 300-150-100-75-60-50-42-38.

Small box method: The small box method is used to calculate the *exact* rate of a regular rhythm. In this method, two consecutive P and QRS waves are located. The number of small boxes between the highest points of these consecutive P waves is counted, and that number is divided into 1500 to determine the atrial rate. The number of small boxes between the highest points of two consecutive QRS waves is counted, and that number is divided into 1500 to determine the ventricular rate (Figure 7-21). This method is accurate only if the rhythm is regular. Charts are available to calculate heart rate based on the rule of 1500.

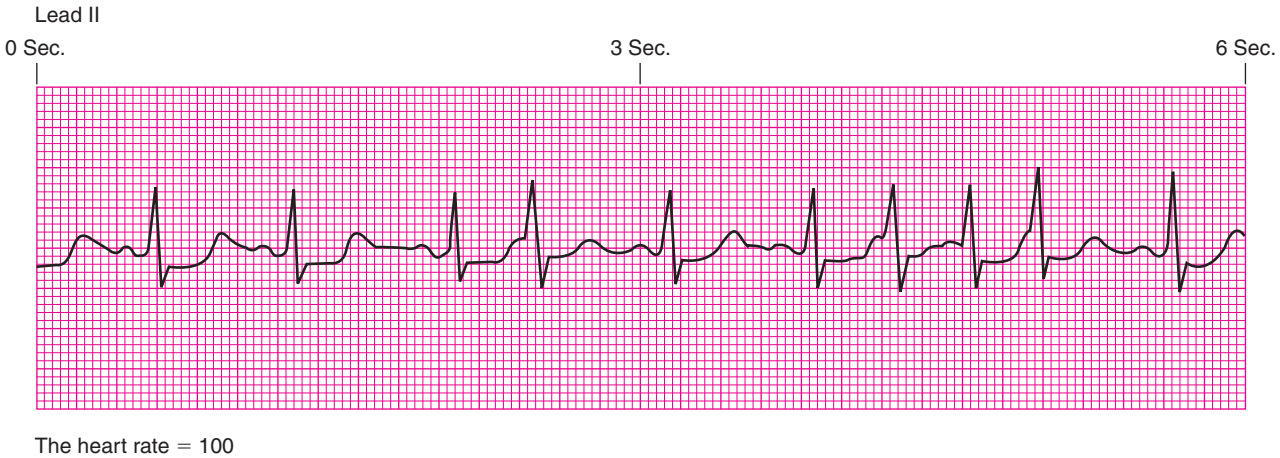
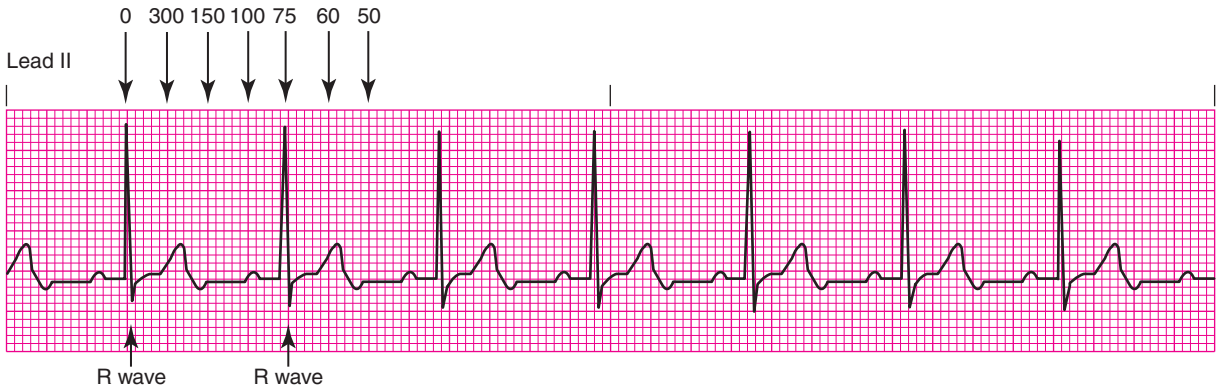


FIGURE 7-19 Six-second method of rate calculation.



Big Box Method of Heart Rate Calculation

- Identify an R wave on a solid vertical line.
- Count the number of big boxes between the first and the following R waves.
- Divide 300 by the number of big boxes between R waves or count the cadence (300…150…100…75…60) representing the big boxes between R waves.

NOTE: Since the position of the second R wave occurs with the arrow reading 75, the heart rate in this example is approximately 75 beats per minute.

FIGURE 7-20 Big box method of heart rate calculation.

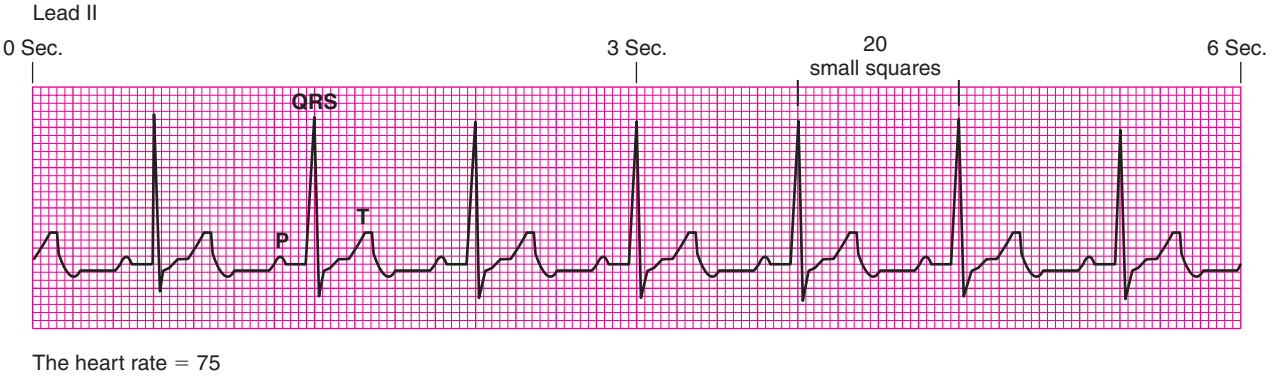


FIGURE 7-21 Small box method of heart rate calculation. (Modified from Wesley K. *Huszar's Basic Dysrhythmias and Acute Coronary Syndromes: Interpretation and Management*. 4th ed. St. Louis: Mosby JEMS; 2010.)

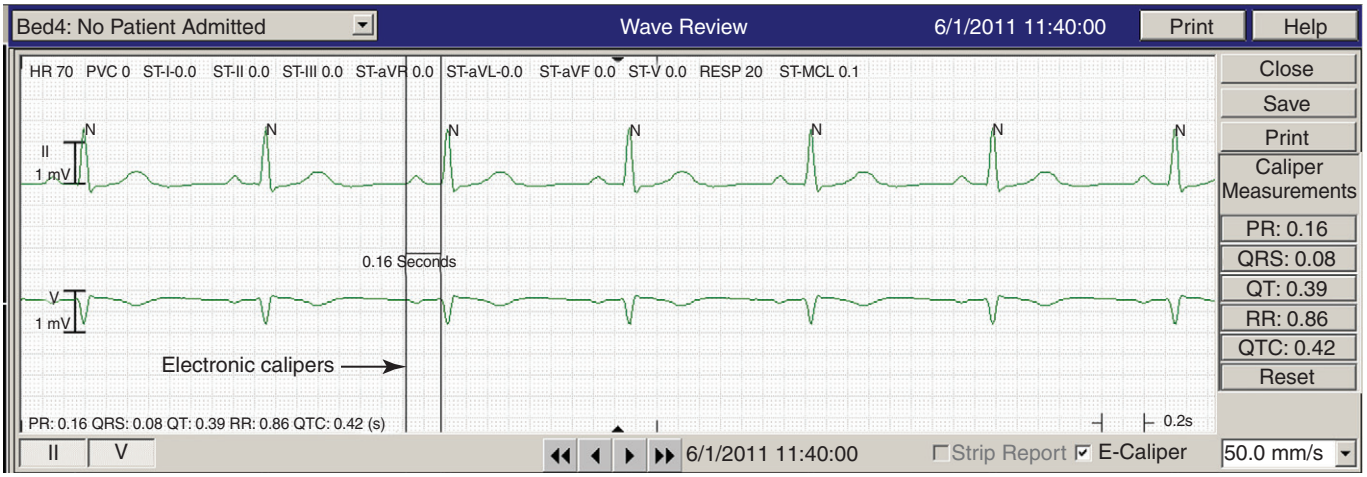


FIGURE 7-22 Electronic calipers. (Courtesy Philips Healthcare, Andover, Massachusetts.)

Cardiac monitors continuously display heart rates. However, the displayed rate should always be verified by one of the aforementioned rate calculation methods.

Regularity

Regularity is assessed by using electronic or physical calipers, or a piece of paper and pencil. To determine atrial regularity, identify the P wave and place one caliper point on the peak of the P wave. Locate the next P wave and place the second caliper point on its peak. The second point is left stationary, and the calipers are flipped over. If the first caliper point lands exactly on the next P wave, the atrial rhythm is perfectly regular. If the point lands one small box or less away from the next P wave, the rhythm is essentially regular. If the point lands more than one small box away, the rhythm is considered irregular. Electronic calipers on some monitoring systems are used the same way (Figure 7-22).

The same process can be performed with a simple piece of paper. Place the paper parallel and below the rhythm line, make a hatch mark below the first and second P waves, then move the paper over to determine if the distance between the second and third P waves is equal to the first and second. When an atrial rhythm is perfectly regular, each P wave is an equal distance from the next P wave.

This process is also used to assess ventricular regularity, except that the caliper points are placed on the peak of two consecutive R waves. One caliper point is placed on one R wave and the other caliper point on the next R wave. The second point is left stationary, and the calipers are flipped over. If the first caliper point lands exactly on the next R wave, the ventricular rhythm is perfectly regular. Paper and pencil can also be used in the same manner as previously described, placing a hatch mark on the first and second R waves, then moving the paper down the rhythm strip to determine if the subsequent R waves land on the hash mark. If the hatch mark is more than one small box away from the next R wave, the rhythm is irregular (Figure 7-23).

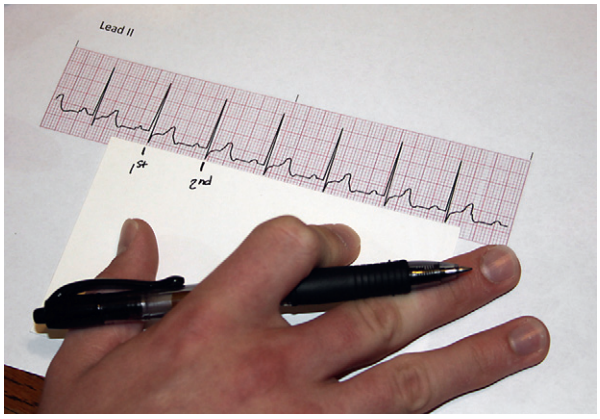


FIGURE 7-23 Use of paper and pencil to assess regularity.

Irregular rhythms can be regularly irregular or irregularly irregular. Regularly irregular rhythms have a pattern. Irregularly irregular rhythms have no pattern and no predictability. Atrial fibrillation is an example of an irregularly irregular rhythm.

Measurement of PR, QRS, QT/QTc Intervals

PR, QRS, and QT/QTc intervals are measured and documented as part of rhythm analysis. In some dysrhythmias, intervals such as the PR interval may change; thus all PR intervals are measured to ensure that they are consistent. QRS intervals can lengthen in response to new bundle branch blocks or with ventricular dysrhythmias. QT/QTc intervals can lengthen in response to certain drugs as well as electrolyte imbalances. Intervals are measured with calipers or paper and pencil as previously described by identifying the number of small boxes and multiplying by 0.04 seconds. If the end of the interval being measured falls between boxes, add 0.02 to the measurement, as this is the time allowed for half of a box of measurement.

Shape or Morphology of Waveforms

The P, QRS, and T waves of the rhythm strip are assessed for shape and consistency. All waveforms should look alike in the normal ECG. Abnormal shapes may indicate that the stimulus that caused the waveform came from an ectopic focus, or that there is a delay or block in conduction creating a bundle branch block. It is important to also confirm that a P wave precedes the QRS complex and that the T wave follows the QRS complex. Several dysrhythmias are characterized by abnormal location or sequencing of waveforms, such as the P waves in complete heart block.

Identification of Underlying Rhythm and Any Noted Dysrhythmia

The underlying rhythm is identified first. Following this step, the dysrhythmia that disrupts the underlying rhythm is determined.

Patient Tolerance of the Rhythm and Clinical Implication of the Rhythm

Once an abnormal heart rhythm is identified, the priority is to assess the patient for any symptoms that may be related to the dysrhythmia (Box 7-2). Assessment for hemodynamic deterioration includes obtaining vital signs, assessing for alterations in level of consciousness, auscultating lung sounds, and asking the patient if there are any complaints of dyspnea or chest discomfort. Instability is manifested by any of the following: hypotension, acutely altered mental status, signs of shock, ischemic chest discomfort, or acute heart failure.[3] Additionally, a 12-lead ECG is obtained to aid in identification of the dysrhythmia.

The next step is to determine if there are causes of the dysrhythmia that can be treated immediately. An example is a patient with a fast, wide complex tachycardia who has a pulse but low blood pressure. The immediate priority is to treat the patient's fast heart rhythm with a therapy such as emergent cardioversion, but the next critical step is to identify potential causes of the dysrhythmia, such as hypokalemia, hypomagnesemia, hypoxemia, or ischemia.

BOX 7-2 SYMPTOMS OF DECREASED CARDIAC OUTPUT

- Change in level of consciousness
- Chest discomfort
- Hypotension
- Shortness of breath; respiratory distress
- Pulmonary congestion; crackles
- Rapid, slow, or weak pulse
- Dizziness
- Syncope
- Fatigue
- Restlessness

BASIC DYSRHYTHMIAS

The basic dysrhythmias are classified based on their site of origin, including:

- SA node
- Atrial
- AV node or junctional
- Ventricular
- Heart blocks of the AV node

The following discussion reviews the ECG characteristics and provides examples of each dysrhythmia. Specific criteria that can be used to recognize and identify dysrhythmias are presented systematically for each one. The discussion includes typical causes, patient responses, and appropriate treatment. Medications used to treat common dysrhythmias are described in Table 7-1.

The learner who is new to identification of dysrhythmias will benefit from extensive practice reading rhythm strips and collaborating with seasoned colleagues who are adept at rhythm interpretation. Maintaining a pocket notebook (or using handheld devices with cardiac rhythm applications) with ECG criteria for each rhythm helps the learner memorize the criteria specific to common dysrhythmias. Other suggested learning aids are to complete a course in basic rhythm interpretation, either in the classroom or by computerized instruction. Finally, mastering the identification of dysrhythmias requires practice, practice, and more practice. Another essential assessment skill is recognition of hemodynamic instability related to decreased cardiac output because of the cardiac dysrhythmia (see Box 7-2).

Normal Sinus Rhythm

Normal sinus rhythm (NSR) reflects normal conduction of the sinus impulse through the atria and ventricles. Any deviation from sinus rhythm is a dysrhythmia, thus it is critical to remember and understand the criteria that determine NSR.

Sinus rhythm is initiated by an impulse in the sinus node. The generated impulse propagates through the conductive fibers of the atria, reaches the AV node where there is a slight pause, and then spreads throughout the ventricles, causing depolarization and resultant cardiac contraction in a timely and organized manner (Figure 7-24).

Rhythm Analysis

- *Rate:* Atrial and ventricular rates are the same and range from 60 to 100 beats per minute.
- *Regularity:* Rhythm is regular or essentially regular.
- *Interval measurements:* PR interval is 0.12 to 0.20 seconds. QRS interval is 0.06 to 0.10 seconds.
- *Shape and sequence:* P and QRS waves are consistent in shape. P waves are small and rounded. A P wave precedes every QRS complex, which is followed by a T wave.
- *Hemodynamic effect:* Patient is hemodynamically stable.

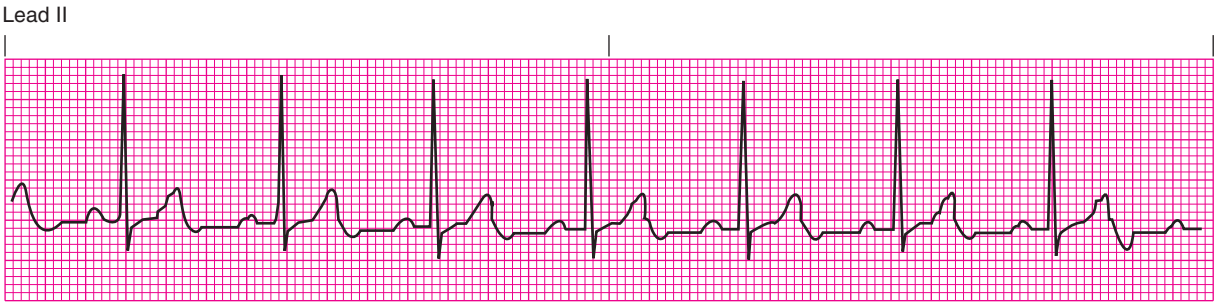


FIGURE 7-24 Normal sinus rhythm.

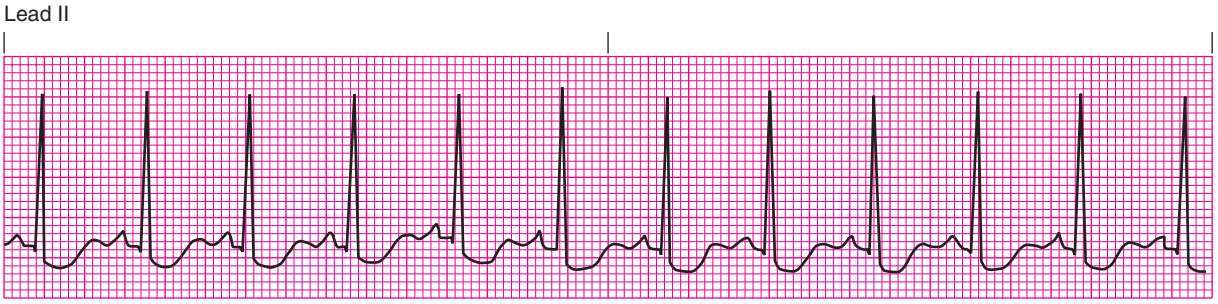


FIGURE 7-25 Sinus tachycardia.

Dysrhythmias of the Sinoatrial Node

Sinus Tachycardia

Tachycardia is defined as a heart rate greater than 100 beats per minute. Sinus tachycardia results when the SA node fires faster than 100 beats per minute (Figure 7-25). Sinus tachycardia is a normal response to stimulation of the sympathetic nervous system. Sinus tachycardia is also a normal finding in children younger than 6 years.

Rhythm analysis

- *Rate:* Both atrial and ventricular rates are greater than 100 beats per minute, up to 160 beats per minute, but may be as high as 180 beats per minute.[13]
- *Regularity:* Onset is gradual rather than abrupt. Sinus tachycardia is regular or essentially regular.
- *Interval measurements:* PR interval is 0.12 to 0.20 seconds (at higher rates, the P wave may not be readily visible). QRS interval is 0.06 to 0.10 seconds. QT may shorten.
- *Shape and sequence:* P and QRS waves are consistent in shape. P waves are small and rounded. A P wave precedes every QRS complex, which is then followed by a T wave.
- *Patient response:* The fast heart rhythm may cause a decrease in cardiac output because of the shorter filling time for the ventricles. Vulnerable populations are those with ischemic heart disease who are adversely affected by the shorter time for coronary filling during diastole.
- *Causes:* Hyperthyroidism, hypovolemia, heart failure, anemia, exercise, use of stimulants, fever, and sympathetic response to fear or pain and anxiety may cause sinus tachycardia.
- *Care and treatment:* The dysrhythmia itself is not treated, but the cause is identified and treated appropriately. For example, pain medications are administered to treat pain or antipyretics are given to treat fever.

Sinus Bradycardia

Bradycardia is defined as a heart rate less than 60 beats per minute. Sinus bradycardia may be a normal heart rhythm for some individuals such as athletes, or it may occur during sleep. Although sinus bradycardia may be asymptomatic, it may cause instability in some individuals if it results in a decrease in cardiac output. The key is to assess the patient and determine if the bradycardia is accompanied by signs of instability (Figure 7-26).

Rhythm analysis

- *Rate:* Both atrial and ventricular rates are less than 60 beats per minute.
- *Regularity:* Rhythm is regular or essentially regular.
- *Interval measurements:* Measurements are normal, but QT may be prolonged.
- *Shape and sequence:* P and QRS waves are consistent in shape. P waves are small and rounded. A P wave precedes every QRS complex, which is followed by a T wave.
- *Patient response:* The slowed heart rhythm may cause a decrease in cardiac output, resulting in hypotension and decreased organ perfusion.
- *Causes:* Vasovagal response; medications such as digoxin or AV nodal blocking agents, including calcium channel blockers and beta blockers; myocardial infarction; normal

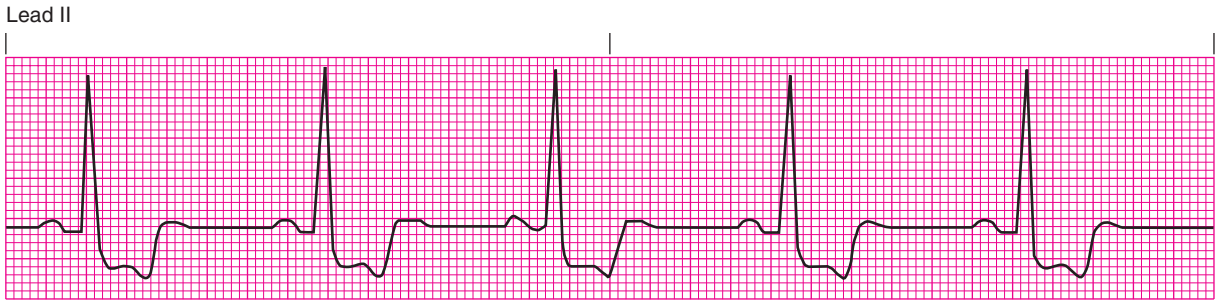


FIGURE 7-26 Sinus bradycardia.

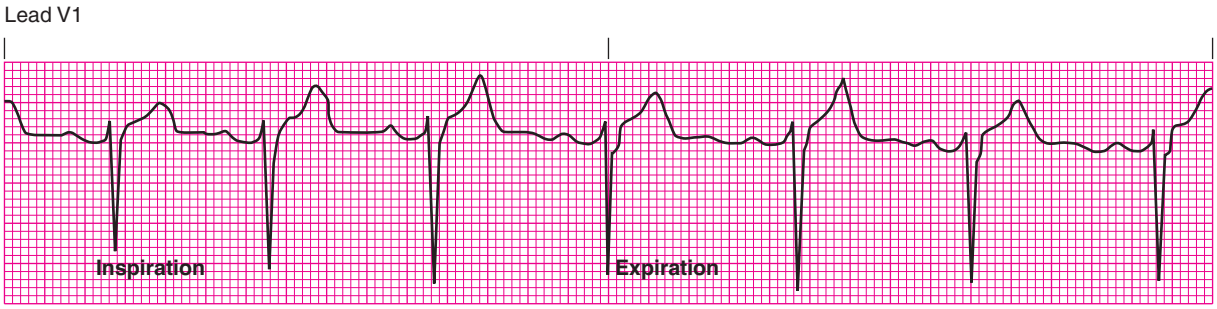


FIGURE 7-27 Sinus arrhythmia. The heart rate increases slightly with inspiration and decreases slightly with expiration.

physiological variant in the athlete; disease of the sinus node; increased intracranial pressure; hypoxemia; and hypothermia may cause sinus bradycardia.

- *Care and treatment:* Assess for hemodynamic instability related to the bradycardia. If the patient is symptomatic, interventions include administration of atropine. If atropine is not effective in increasing heart rate, then transcutaneous pacing, dopamine infusion, or epinephrine infusion may be administered.[3] Atropine is avoided for treatment of bradycardia associated with hypothermia.

Sinus Arrhythmia

Sinus arrhythmia is a cyclical change in heart rate that is associated with respiration. The heart rate slightly increases during inspiration and slightly slows during exhalation because of changes in vagal tone.[13] The ECG tracing demonstrates an alternating pattern of faster and slower heart rate that changes with the respiratory cycle (Figure 7-27).

Rhythm analysis

- *Rate:* Atrial and ventricular rates are between 60 and 100 beats per minute.
- *Regularity:* This rhythm is cyclically irregular, slowing with exhalation and increasing with inspiration.
- *Interval measurements:* Measurements are normal.
- *Shape and sequence:* P and QRS waves are consistent in shape. P waves are small and rounded. A P wave precedes every QRS complex, which is then followed by a T wave.
- *Patient response:* This rhythm is tolerated well.
- *Care and treatment:* No treatment is required.

Sinus Pauses

Sinus pauses occur when the SA node either fails to generate an impulse (sinus arrest) or the impulse is blocked and does not exit from the SA node (sinus exit block). The result of the sinus node not firing is a pause without any electrical activity.

Sinus arrest. Failure of the SA node to generate an impulse is called *sinus arrest.* The arrest results from a lack of stimulus from the SA node. The sinus beat following the arrest is not on time because the sinus node has been reset and the next sinus impulse begins a new rhythm. The end result is that no atrial or ventricular depolarization occurs for one heartbeat or more (Figure 7-28, *A*).

If the pause is long enough, the AV node or ventricular backup pacemaker may fire, resulting in *escape beats.* These beats are called junctional escape or ventricular escape beats. Typically, the sinus node resumes normal generation of impulses following the pause.

Sinus exit block. Sinus exit block also results in a pause, but the P wave following the pause in rhythm is on time or regular because the sinus node does not reset. The sinus impulse simply fails to "exit" the sinus node (Figure 7-28, *B*).

Rhythm analysis

- *Rate:* Atrial and ventricular rates are usually between 60 and 100 beats per minute, but any pause may result in a heart rate less than 60 beats per minute.
- *Regularity:* The rhythm is irregular for the period of the pause but regular when sinus rhythm resumes. In SA exit

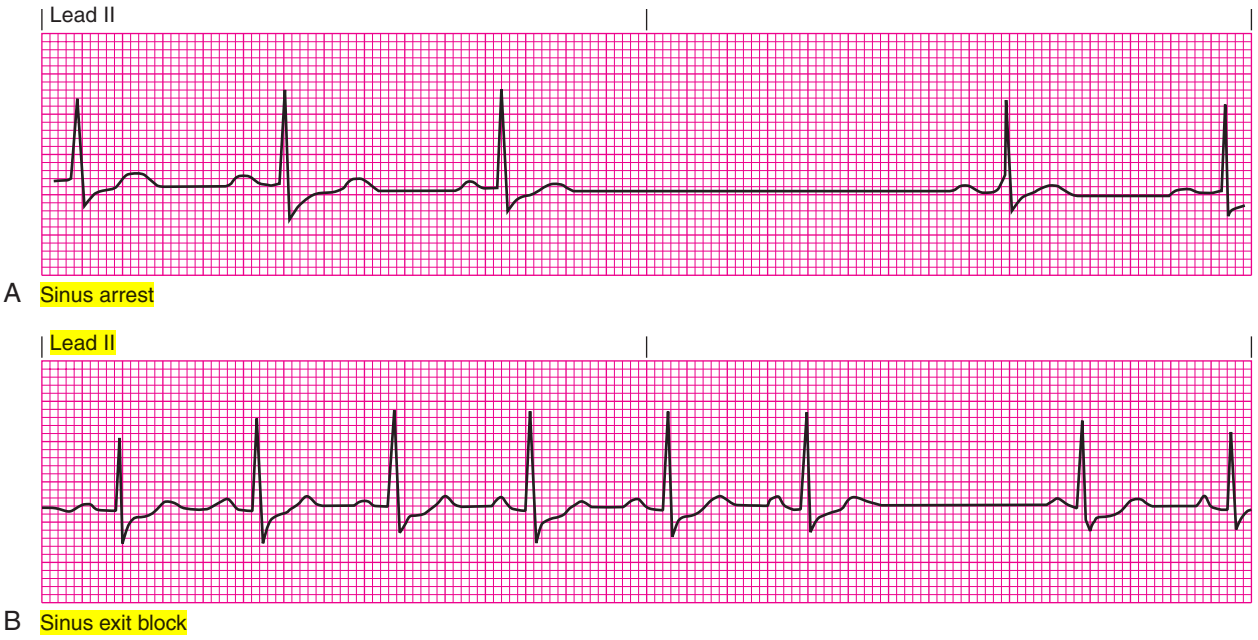


FIGURE 7-28 **A,** Sinus arrest. **B,** Sinus exit block.

block, the P wave following the pause occurs on time. In sinus arrest, the P wave following the pause is not on time.

- *Interval measurements:* Measurements of conducted beats are normal.
- *Shape and sequence:* P and QRS waves are consistent in shape. P waves are small and rounded. A P wave precedes every QRS complex, which is followed by a T wave.
- *Patient response:* Single pauses in rhythm may not be significant, but frequent pauses may result in a severe bradycardia. The patient with multiple pauses may experience signs and symptoms of decreased cardiac output (see Box 7-2).
- *Causes:* Hypoxemia; ischemia or damage of the sinus node related to myocardial infarction; AV nodal blocking medications such as beta blockers, calcium channel blockers, and digoxin; and increased vagal tone may cause sinus exit block.
- *Care and treatment:* If the patient is symptomatic, significant numbers of pauses may require treatment, including temporary and permanent implantation of a pacemaker. Causes are explored, and prescribed medications may need to be adjusted or discontinued.

Dysrhythmias of the Atria

Normally, the SA node is the dominant pacemaker initiating the heart rhythm; however, cells outside the SA node within the atria can create an ectopic focus that can cause a dysrhythmia. An ectopic focus is an abnormal beat or a rhythm that occurs outside the normal conduction system. In this case, atrial dysrhythmias arise in the atrial tissue.

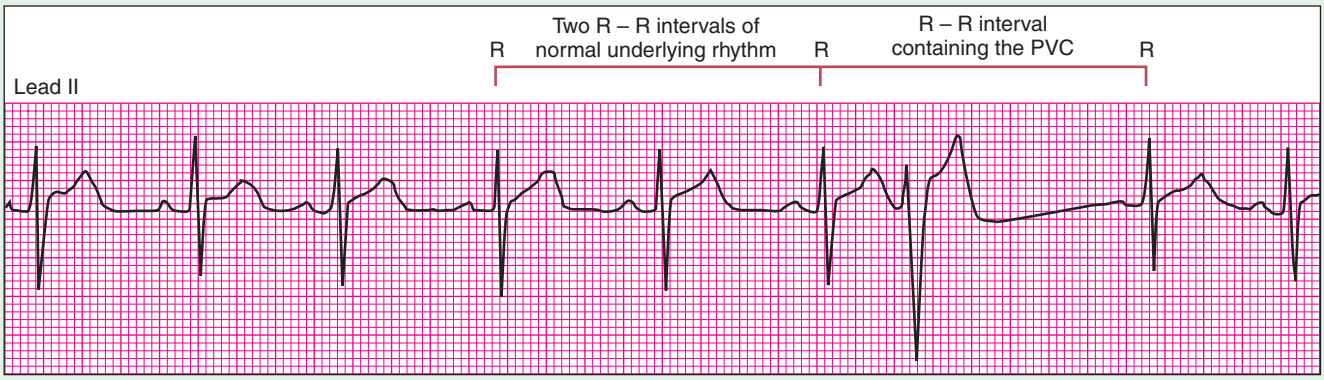
Premature Atrial Contractions

A premature atrial contraction (PAC) is a single ectopic beat arising from atrial tissue, not the sinus node. The PAC occurs earlier than the next normal beat and interrupts the regularity of the underlying rhythm. The P wave of the PAC has a different shape than the sinus P wave because it arises from a different area in the atria; it may follow or be in the T wave of the preceding normal beat. If the early P wave is in the T wave, this T wave will look different from the T wave of a normal beat. Following the PAC, a pause occurs and then the underlying rhythm typically resumes. The pause is noncompensatory, which means that when measuring the P-P intervals for atrial regularity, the P wave following the pause does not occur on time. Box 7-3 discusses how to distinguish compensatory and noncompensatory pauses. PACs are common but denote an irritable area in the atria that has developed the property of automaticity (Figure 7-29).

Nonconducted PACs are beats that create an early P wave but are not followed by a QRS complex. The ventricles are unable to depolarize in response to this early stimulus because they are not fully repolarized from the normally conducted beat preceding the PAC (see Figure 7-29). This creates a pause, but a P wave occurs either in or after

BOX 7-3 COMPENSATORY VERSUS NONCOMPENSATORY PAUSE

- A rhythm strip with a premature beat is analysed using calipers or paper and pencil.
- Two consecutive normal beats are located just before the premature beat, and the caliper points or pencil marks are placed on the R wave of each normal beat.
- The calipers are flipped over, or the paper is slid over, to where the next normal beat should have occurred. The premature beat occurs early.
- Now, with care taken not to lose placement, the calipers are flipped or the paper is slid over one more time. If the point of the calipers or the mark on the paper lands exactly on the next normal beat's R wave, the sinus node compensated for the one premature beat and kept its normal rhythm (see figure).
- If the caliper point or pencil mark does not land on the next normal beat's R wave, then the sinus node did not compensate and had to establish a new rhythm, resulting in a noncompensatory pause.



Compensatory pause; sinus rhythm with premature ventricular contraction (PVC). The pause following the PVC is compensatory. (From Paul S, Hebra JD. *The Nurse's Guide to Cardiac Rhythm Interpretation: Implications for Patient Care*. Philadelphia, Saunders; 1998.)

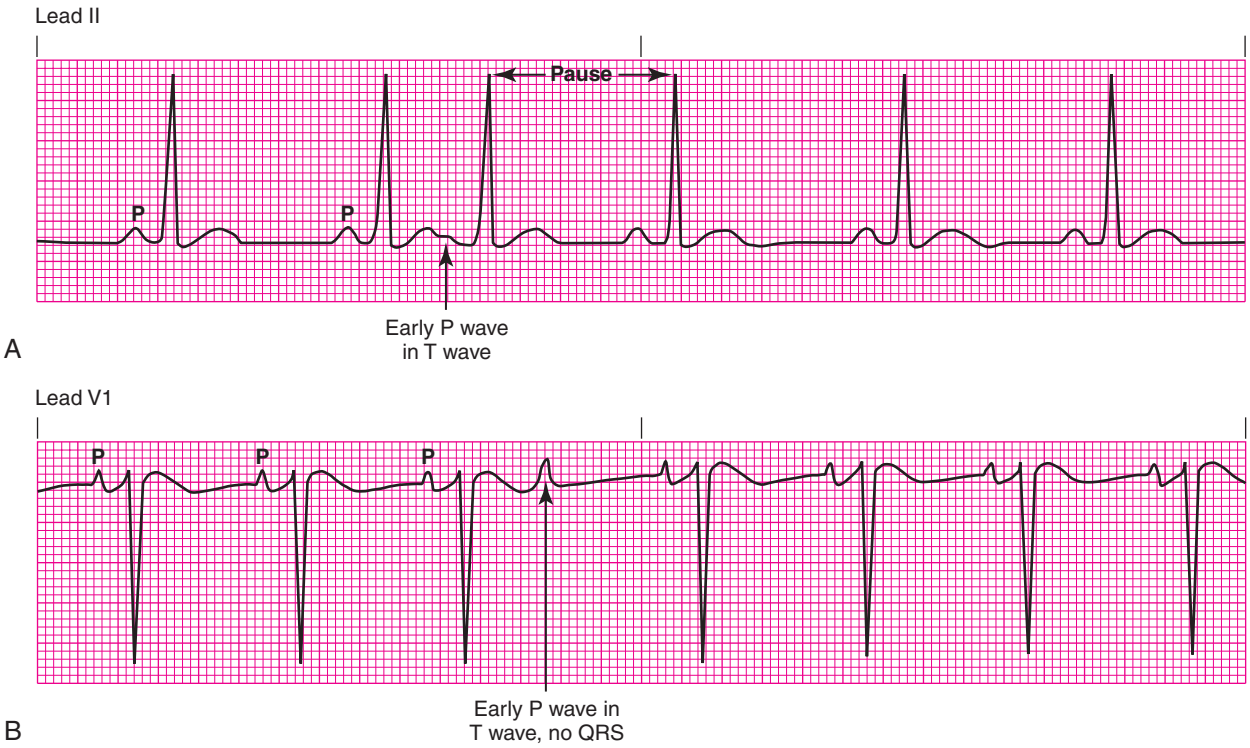


FIGURE 7-29 **A,** Premature atrial contractions (PAC) shown in the third beat. **B,** A nonconducted PAC. (Modified from Association of Critical-Care Nurses: *Essentials of Critical Care Orientation*.)

BOX 7-4 NAMES GIVEN TO EARLY BEATS

- An early beat that occurs every other beat is called bigeminy.
- An early beat that occurs every third beat is called trigeminy.
- An early beat that occurs with frequency is given the name of the underlying rhythm, and then the naming of the frequency of early beats follows that name. An example is sinus rhythm with bigeminal PACs.

the T wave. This is why comparing the shapes of the normal PQRST is a critical requirement in rhythm analysis. The frequency of occurrence of PACs varies (Box 7-4).

Rhythm analysis

- *Rate:* The rate matches that of the underlying rhythm.
- *Regularity:* The PAC interrupts the regularity of the underlying rhythm for a single beat. The PAC is followed by a noncompensatory pause (see Box 7-3).
- *Interval measurements:* The PAC may have a different PR interval than the normal sinus beat, usually shorter.
- *Shape and sequence:* The P wave of the PAC is typically a different shape than the sinus P wave. The T wave of the preceding beat may be distorted if the P wave of the PAC lies within it.
- *Patient response:* PACs are usually well-tolerated, although the patient may complain of palpitations.
- *Causes:* Stimulants such as caffeine or tobacco, myocardial hypertrophy or dilatation, ischemia, lung disease, hypokalemia, and hypomagnesemia may cause PACs. It may also be a normal variant.
- *Care and treatment:* Increasing numbers of PACs may occur before atrial fibrillation or atrial flutter. No treatment is indicated for PACs.

Atrial Tachycardia

Atrial tachycardia is a rapid rhythm that arises from an ectopic focus in the atria. Because of the fast rate, atrial tachycardia can be a life-threatening dysrhythmia. The ectopic atrial focus generates impulses more rapidly than the AV node can conduct while still in the refractory phase from the previous impulse, and these impulses are not transmitted to the ventricles. Therefore more P waves may be seen than QRS complexes and T waves. This refractoriness serves as a safety mechanism to prevent the ventricles from contracting too rapidly. The AV node may block impulses in a set pattern, such as every second, third, or fourth beat. However, if the ventricles respond to every ectopic atrial impulse, it is called 1:1 conduction, one P wave for each QRS complex. Because the P wave arises outside the sinus node, the shape is different from the sinus P wave (Figure 7-30).

If an abnormal P wave cannot be visualized on the ECG but the QRS complex is narrow, the term *supraventricular tachycardia* (SVT) is often used. This is a generic term that describes any tachycardia that is not ventricular in origin; it is also used when the source above the ventricles cannot be identified, usually because the rate is too fast.

Rhythm analysis

- *Rate:* The rate ranges from 150 to 250 beats per minute.
- *Regularity:* The rhythm is regular if all P waves are conducted.
- *Interval measurements:* The PR interval is different from the sinus PR interval. If the ectopic P wave arises near the junction, the PR interval may be shortened. If close to the sinus node, it is nearer the normal PR interval in duration.
- *Shape and sequence:* The P wave shape is different from that of the sinus P wave. The QRS complex is narrow unless there is a bundle branch block. If the P wave of the ectopic rhythm occurs in the T wave, this may alter the shape.

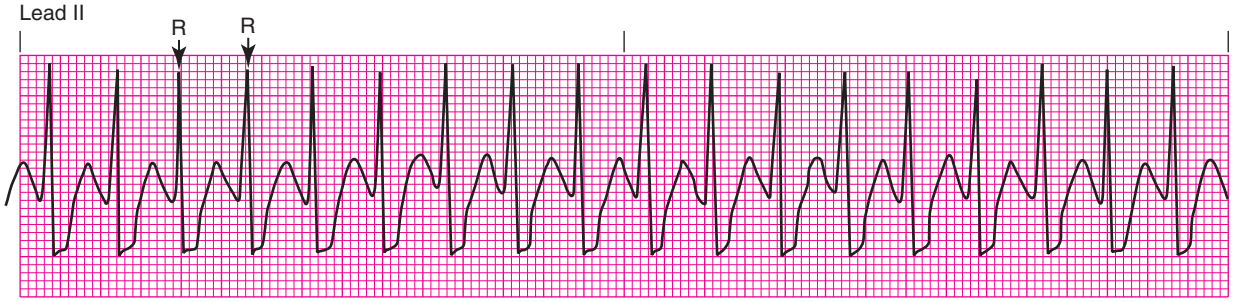


FIGURE 7-30 Atrial tachycardia with 1:1 conduction.

- *Patient response:* The faster the tachycardia, the more symptomatic the patient may become. This arises from decreased cardiac output (see Box 7-2) and resultant decreased organ perfusion.
- *Causes:* Atrial tachycardia can occur in patients with normal hearts as well as those with cardiac disease. Causes include digitalis toxicity, electrolyte imbalances, lung disease, ischemic heart disease, and cardiac valvular abnormalities.
- *Care and treatment:* Treatment is directed at assessing the patient's tolerance of the tachycardia. If the rate is over 150 beats per minute and the patient is symptomatic, emergent cardioversion is considered. Cardioversion is the delivery of a synchronized electrical shock to the heart by an external defibrillator (see Chapter 10). Medications that may be used include adenosine, beta-blockers, calcium channel blockers, and amiodarone.[3]

Wandering Atrial Pacemaker

Wandering atrial pacemaker is a dysrhythmia characterized by at least three different ectopic atrial foci followed by a QRS complex at a rate less than 100 beats per minute. At least three different P wave shapes are noted. P waves in wandering atrial pacemaker can be upright, inverted, flat, pointed, notched, and/or slanted in different directions. The PR interval varies because the impulses originate from different locations within the atria, taking various times to reach the AV node (Figure 7-31).

Rhythm analysis

- *Rate:* Rate is less than 100 beats per minute.
- *Regularity:* The rate may be slightly irregular.
- *Interval measurements:* PR intervals vary based on the sites of the ectopic foci.
- *Shape and sequence:* At least three different P shapes are noted. The QRS complex is narrow and followed by a T wave.
- *Patient response:* Patients usually tolerate this rhythm unless the rate increases.
- *Causes:* Lung disease such as chronic obstructive pulmonary diseases may cause wandering atrial pacemaker. It may also be a normal variant in the young and the elderly.
- *Care and treatment:* No treatment is usually indicated.

Multifocal Atrial Tachycardia

Multifocal atrial tachycardia is essentially the same as wandering atrial pacemaker, except the heart rate exceeds 100 beats per minute (Figure 7-32). At least three ectopic P waves are noted. This dysrhythmia is almost exclusively found in the patient with chronic obstructive pulmonary disease. Pulmonary hypertension occurs and results in increased atrial pressure and dilatation, creating irritable atrial foci.

Rhythm analysis

- *Rate:* The heart rate is greater than 100 beats per minute.
- *Regularity:* The rhythm is slightly irregular.
- *Interval measurements:* PR intervals vary.

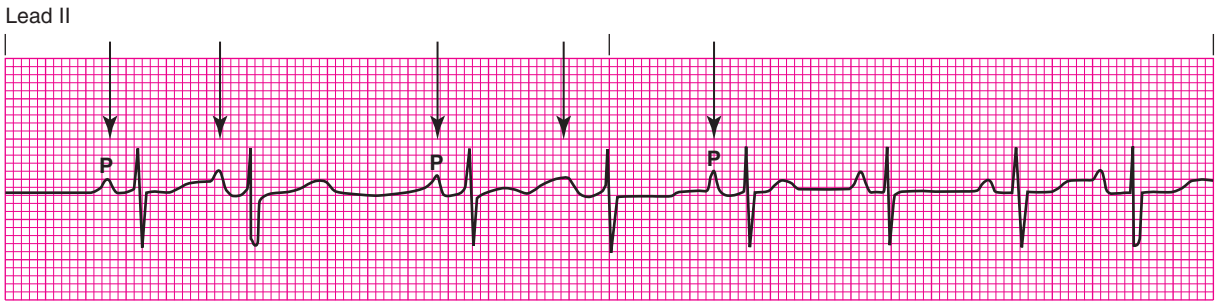


FIGURE 7-31 Wandering atrial pacemaker. Arrows indicate different shapes of P waves.

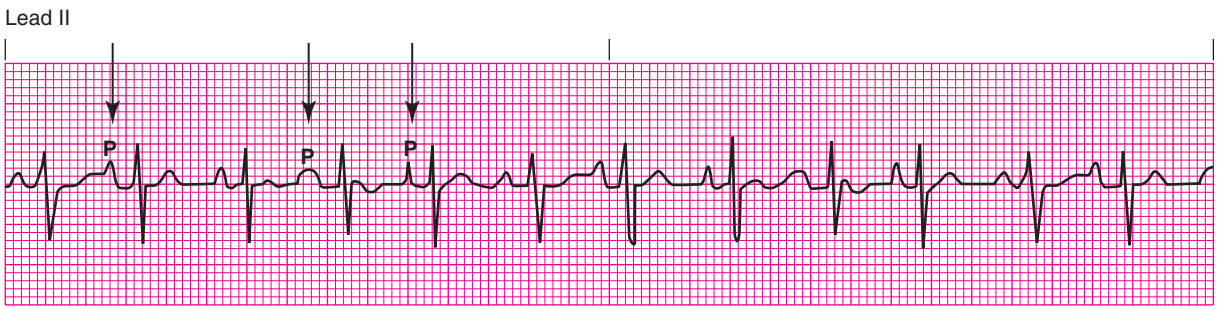


FIGURE 7-32 Multifocal atrial tachycardia. Arrows indicate different shapes of P waves.

- *Shape and sequence:* P waves differ in shape. A P wave precedes every QRS complex, which is followed by a T wave.
- *Patient response:* The response varies and is determined by the patient's tolerance of the tachycardia.
- *Causes:* Chronic obstructive pulmonary disease causes dilatation of the atria with resultant ectopic foci from the stretched tissue.
- *Care and treatment:* The treatment goal is to optimize the patient's pulmonary status.

Atrial Flutter

Atrial flutter arises from a single irritable focus in the atria. The atrial focus fires at an extremely rapid, regular rate, between 240 and 320 beats per minute.[8] The P waves are called flutter waves and have a sawtooth appearance (Figure 7-33, *A*). The ventricular response may be regular or irregular based on how many flutter waves are conducted through the AV node. The number of flutter waves to each QRS complex is called the *conduction ratio.* The conduction ratio may remain the same or vary depending on the number of flutter waves that are conducted to the ventricles. The description of atrial flutter might be constant at 2:1, 3:1, 4:1, 5:1, and so forth, or it may be variable. Flutter waves occur through the QRS complex and the T wave, and often alter their appearance (Figure 7-33). It is helpful to identify the best lead for visualizing the flutter waves in atrial flutter and use this as the second monitor lead.

Rhythm analysis

- *Rate:* Atrial rate is between 240 and 320 beats per minute but is typically 300 beats per minute (one large box between flutter waves, Figure 7-33, *B*. Ventricular rate is determined by the conduction ratio of the flutter waves.
- *Regularity:* Flutter waves are regular but the QRS complex and T waves may not be regular depending on the conduction ratio of the atrial flutter.
- *Interval measurements:* No PR interval is present. QRS and QT intervals are normal unless distorted by a flutter wave.
- *Shape and sequence:* P or flutter waves are consistent in shape and look like tines on a sawtooth blade. QRST waves are altered in shape by the flutter waves.

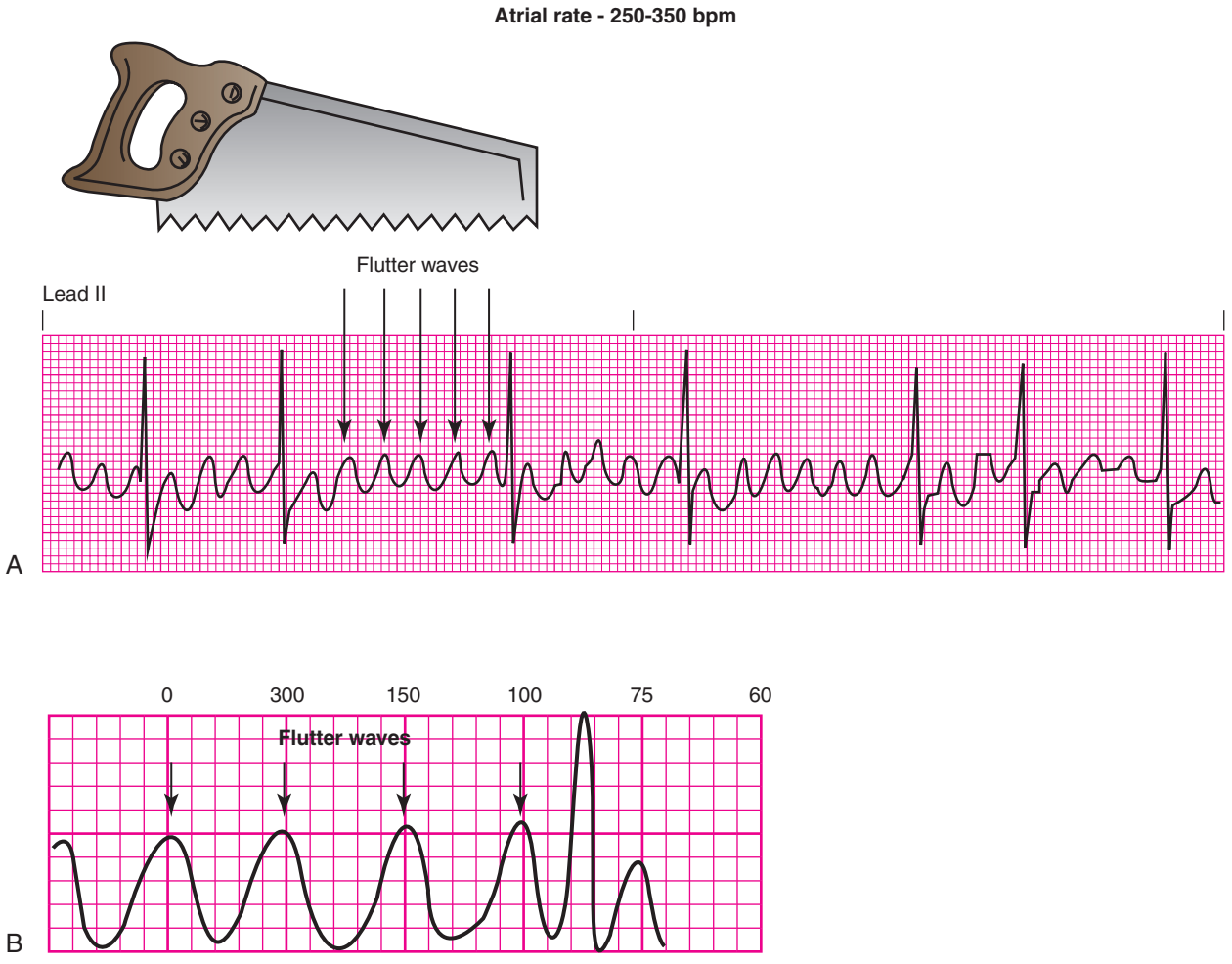


FIGURE 7-33 Atrial flutter. **A,** Flutter waves show sawtooth pattern. **B,** Enlarged view shows one large box between flutter waves.

- *Patient response:* Usually the patient is asymptomatic unless atrial flutter results in a tachycardia called rapid ventricular response (RVR). Atrial flutter with RVR occurs when atrial impulses cause a ventricular response greater than 100 beats per minute.
- *Causes:* Lung disease, ischemic heart disease, hyperthyroidism, hypoxemia, heart failure, and alcoholism can cause atrial flutter.
- *Care and treatment:* Alterations in atrial blood flow leading to blood stasis can cause clot formation. Patients identified with atrial flutter usually receive chronic antithrombotic therapy unless contraindicated. Rate control is accomplished with medications that block the AV node. Elective cardioversion may be performed once the patient has been taking anticoagulants for approximately 6 weeks. Interventional electrophysiological treatments including ablation of the irritable focus may be done in the electrophysiology lab.[8]

Atrial Fibrillation

Atrial fibrillation is the most common dysrhythmia observed in clinical practice.[8] Atrial fibrillation arises from multiple ectopic foci in the atria, causing chaotic quivering of the atria and ineffectual atrial contraction. The AV node is bombarded with hundreds of atrial impulses and conducts these impulses in an unpredictable manner to the ventricles. The atrial rate may be as high 700 and no discernible P waves can be identified, resulting in a wavy baseline and an extremely irregular ventricular response. This irregularity is called *irregularly irregular* (Figure 7-34). The ineffectual contraction of the atria results in loss of *atrial kick*. If too many impulses conduct to the ventricles, atrial fibrillation with rapid ventricular response may result and compromise cardiac output. When atrial fibrillation occurs sporadically it is called paroxysmal atrial fibrillation.

If the atrial impulse is conducted through the ventricles in a normal fashion, the QRS complex is narrow and appears normal although irregularly irregular. But if the impulse reaches one of the bundle branches before full repolarization, the QRS complex is widened in classic bundle branch block morphology. The widened QRS is due to the delay caused by the bundle branch block, and results in slowed conduction through either the right or left ventricle, depending on which bundle branch has not fully repolarized. When this event occurs, the impulse is said to be *aberrantly conducted* (Figure 7-35).

In atrial fibrillation, aberrantly conducted beats are referred to as *Ashman beats*. Ashman beats are more likely to occur when an atrial impulse arrives at the AV node just after a previously conducted impulse (see Figure 7-35). Ashman beats are often seen when the rate changes from slower to faster, referred to as a long-short cycle. Ashman beats are not clinically significant.

One complication of atrial fibrillation is thromboembolism. The blood that collects in the atria is agitated by fibrillation, and normal clotting is accelerated. Small thrombi, called *mural* thrombi, begin to form along the walls of the atria. These clots may dislodge, resulting in pulmonary embolism or stroke.

Rhythm analysis

- *Rate:* Atrial rate is uncountable; ventricular rate may vary widely.
- *Regularity:* Ventricular response is irregularly irregular, unless the patient has complete heart block. In complete heart block, the ventricular response is regular, widened, and between 20 and 40 beats per minute.
- *Interval measurements:* PR interval is absent. The QRS complex and QT interval are normal in duration unless a bundle branch block exists.
- *Shape and sequence:* No recognizable or discernible P waves are present. The isoelectric line is wavy. QRS waves are consistent in shape unless aberrantly conducted. The QRS complex is followed by a T wave.
- *Patient response:* The patient may or may not be aware of the atrial fibrillation. If the ventricular response is rapid, the patient may show signs of decreased cardiac output, or worsening of heart failure symptoms.
- *Causes:* Ischemic heart disease, valvular heart disease, hyperthyroidism, lung disease, heart failure, and aging may cause atrial fibrillation.

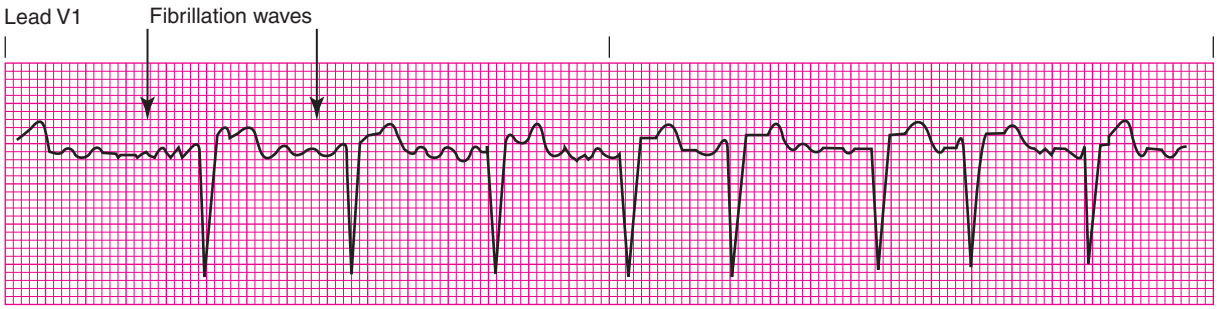


FIGURE 7-34 Atrial fibrillation.

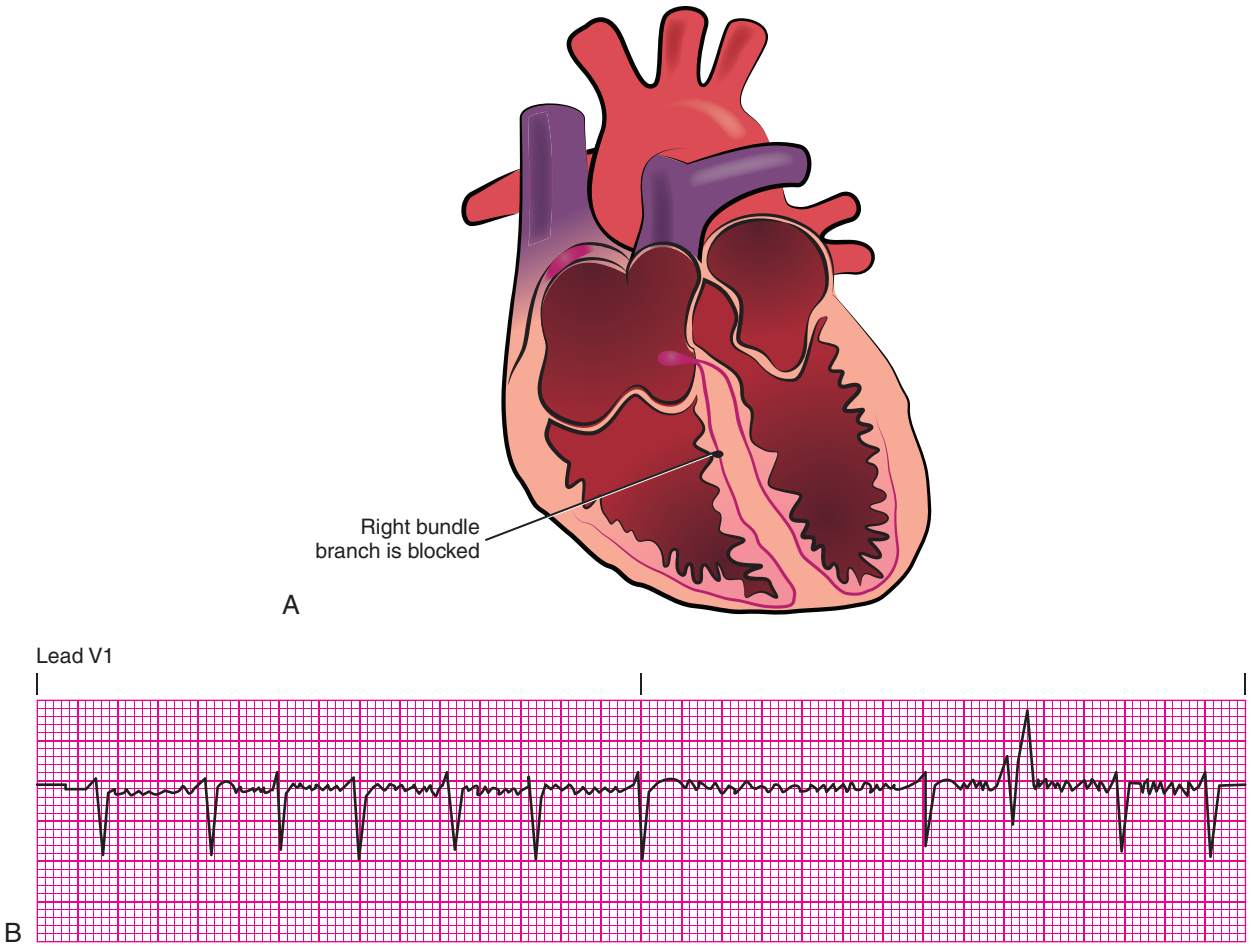


FIGURE 7-35 Atrial fibrillation with single aberrantly conducted beat (9th beat) due to right bundle branch block.

- *Care and treatment:* As with atrial flutter, alterations in blood flow and hemostasis may predispose the patient to clot formation. If there are no contraindications, the patient is prescribed anticoagulants. After at least 4 to 6 weeks of antithrombotic therapy, elective cardioversion can be considered. Amiodarone may be administered to enhance success of cardioversion. Ventricular rate control is attained by administration of AV nodal blocking agents. As with atrial flutter, ablation may be attempted. Symptomatic tachycardia is usually treated with medications because of the risk of thromboembolism. Emergent cardioversion is considered if the tachycardia is associated with hemodynamic instability.[8]

Dysrhythmias of the Atrioventricular Node

Dysrhythmias of the AV node are called *junctional rhythms,* which include junctional escape rhythm, premature junctional contractions (PJCs), accelerated junctional rhythm, junctional tachycardia, and paroxysmal supraventricular tachycardia. Several ECG changes are common to all junctional dysrhythmias. These changes include P-wave abnormalities and PR-interval changes.

P-Wave Changes

Because of the location of the AV node—in the center of the heart—impulses generated may be conducted forward, backward, or both. With the potential of forward, backward, or bidirectional impulse conduction, three different P waveforms may be associated with junctional rhythms:

1. When the AV node impulse is conducted backward, the impulse enters the atria first. Conduction back toward the atria allows for at least partial depolarization of the atria. When depolarization occurs backward, an inverted P wave is created. Once the atria have been depolarized, the impulse then moves down the bundle of His and depolarizes both ventricles normally (Figure 7-36, *A*). A short PR interval (<0.12 second) is noted.
2. When the impulse is conducted both forward and backward, *P waves may be present after the QRS complex.* In this type of conduction, the impulse first moves into the ventricles, depolarizing them and creating a QRS complex. Because the impulse is also conducted backward, some atrial depolarization occurs, and a late P wave is noted after the QRS complex (Figure 7-36, *B*).
3. When the AV node impulse moves forward, *P waves may be absent* because the impulse enters the ventricle first. The atria receives the wave of depolarization at the same time as the ventricles; thus, because of the larger muscle mass of the ventricles, there is no P wave (Figure 7-36, *C*).

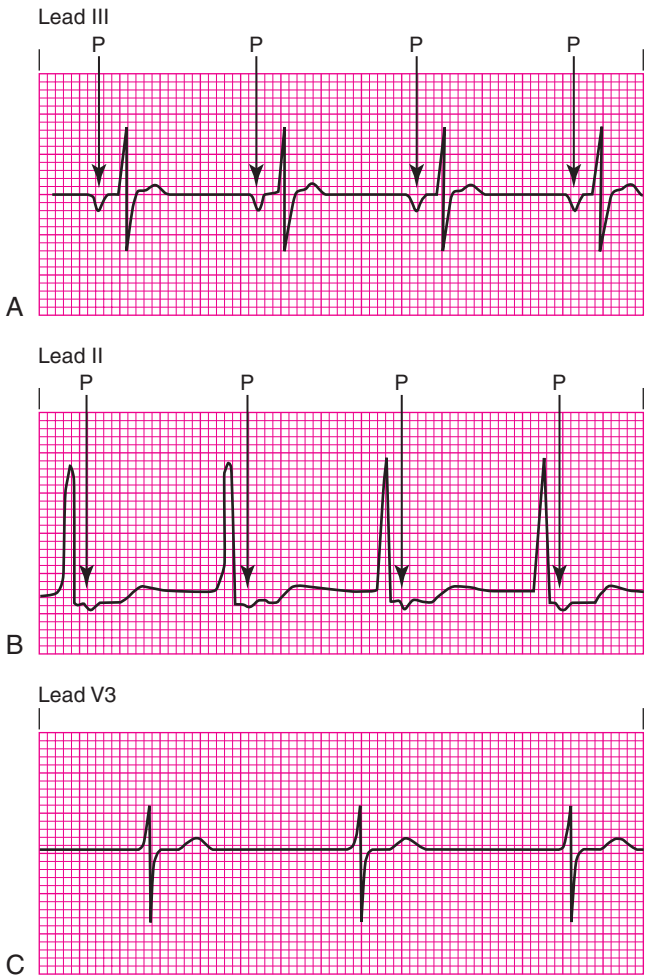


FIGURE 7-36 P waves in junctional dysrhythmias. **A,** Backward conduction with inverted P wave. **B,** Forward and backward conduction with retrograde P wave. **C,** Forward conduction with absent P wave.

Junctional Escape Rhythm

Junctional escape rhythms occur when the dominant pacemaker, the SA node, fails to fire. A junctional escape rhythm has either an inverted P wave and short PR interval preceding the QRS complex, a P wave that follows the QRS complex (retrograde), or no visible P wave. The escape rhythm may consist of many successive beats (Figure 7-37, *A*) or it may occur as a single escape beat that follows a pause, such as a sinus pause (Figure 7-37, *B*).

Rhythm analysis

- *Rate:* Heart rate is 40 to 60 beats per minute.
- *Regularity:* The rhythm is regular.
- *Interval measurements:* If a P wave is present before the QRS complex, the PR interval is shortened less than 0.12 milliseconds. QRS complex is normal.
- *Shape and sequence:* P waves may be inverted, follow the QRS complex, or be absent.
- *Patient response:* The patient is assessed for tolerance of the bradycardia.
- *Causes:* The escape rhythm results from loss of sinus node activity.
- *Care and treatment:* Determine the patient's tolerance of the bradycardia. Alert the provider of the change in rhythm. If symptomatic, administer atropine; consider transcutaneous pacing, dopamine infusion, or epinephrine infusion.[3]

Accelerated Junctional Rhythm and Junctional Tachycardia

The normal intrinsic rate for the AV node and junctional tissue is 40 to 60 beats per minute, but rates can accelerate. An accelerated junctional rhythm has a rate between 60 and 100 beats per minute (Figure 7-38, *A*) and the rate for junctional tachycardia is greater than 100 beats per minute (Figure 7-38, *B*).

Rhythm analysis

- *Rate:* Accelerated junctional rhythm is 60 to 100 beats per minute. Junctional tachycardia rhythm is greater than 100 beats per minute.
- *Regularity:* Regular
- *Interval measurements:* If a P wave is present before the QRS, the PR interval is shortened less than 0.12 ms. QRS is followed by T wave and both are normal in shape.
- *Shape and sequence:* If P wave precedes QRS, it is inverted or upside down, the P wave may not be visible or may follow the QRS.
- *Patient response:* Patient may have a decrease in cardiac output and hemodynamic instability, depending on the rate.
- *Causes:* SA node disease, ischemic heart disease, electrolyte imbalances, digitalis toxicity, and hypoxemia can be causes.
- *Care and treatment:* Assess and treat the tachycardia if the patient is hemodynamically unstable. Alert the provider of the change in rhythm.

Premature Junctional Contractions

Irritable areas in the AV node and junctional tissue can generate premature beats that are earlier than the next expected beat (Figure 7-39). These premature beats are similar to PACs but with characteristics of a junctional beat. The regularity of the underlying rhythm is interrupted by the premature junctional beat. The premature junctional contraction (PJC) is followed by a noncompensatory pause.

Rhythm analysis

- *Rate:* That of the underlying rhythm.
- *Regularity:* Underlying rhythm is interrupted by a premature beat that momentarily disrupts regularity.
- *Interval measurements:* The PJC is early, thus next to the T wave.
- *Shape and sequence:* If P wave precedes QRS, it is inverted, may not be visible, or follows the QRS. QRS is followed by T wave and both are normal in shape.
- *Patient response:* Well tolerated but patient may experience palpitations if the PJCs occur frequently.
- *Causes:* Normal variant; digitalis toxicity; ischemic or valvular heart disease; heart failure; response to endogenous or exogenous catecholamines, such as epinephrine.
- *Care and treatment:* No treatment indicated.

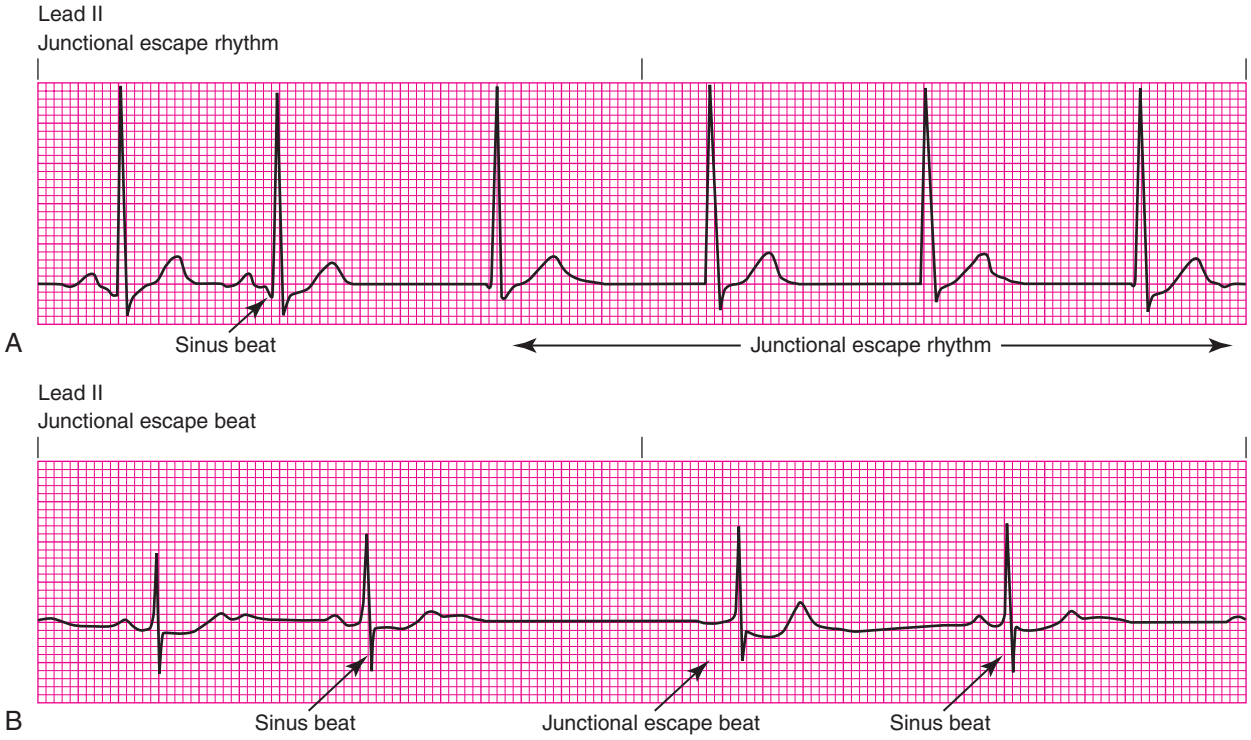


FIGURE 7-37 **A,** Junctional escape rhythm. **B,** Junctional escape beat.

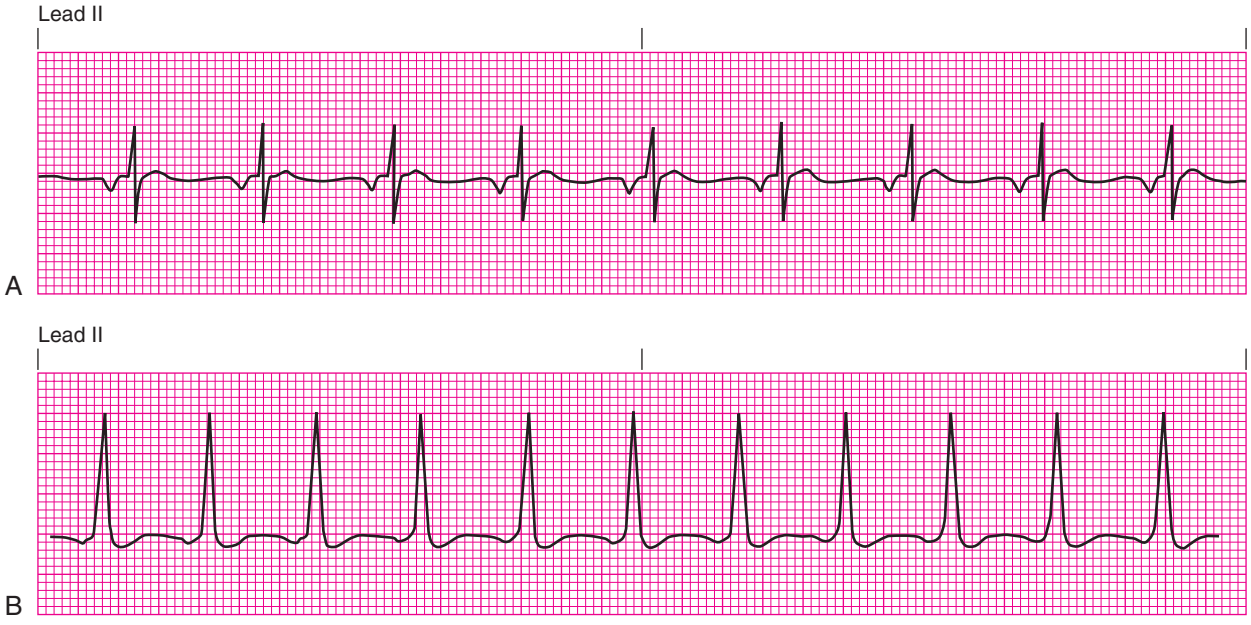


FIGURE 7-38 **A,** Accelerated junctional rhythm. **B,** Junctional tachycardia.

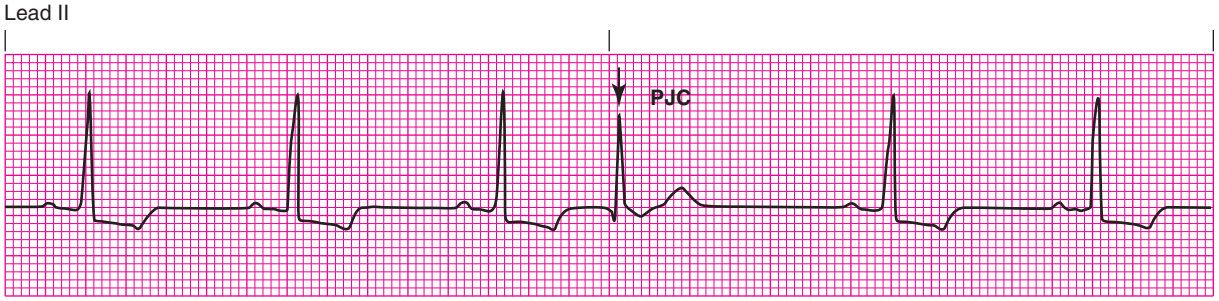


FIGURE 7-39 Sinus rhythm with premature junctional contraction (PJC).

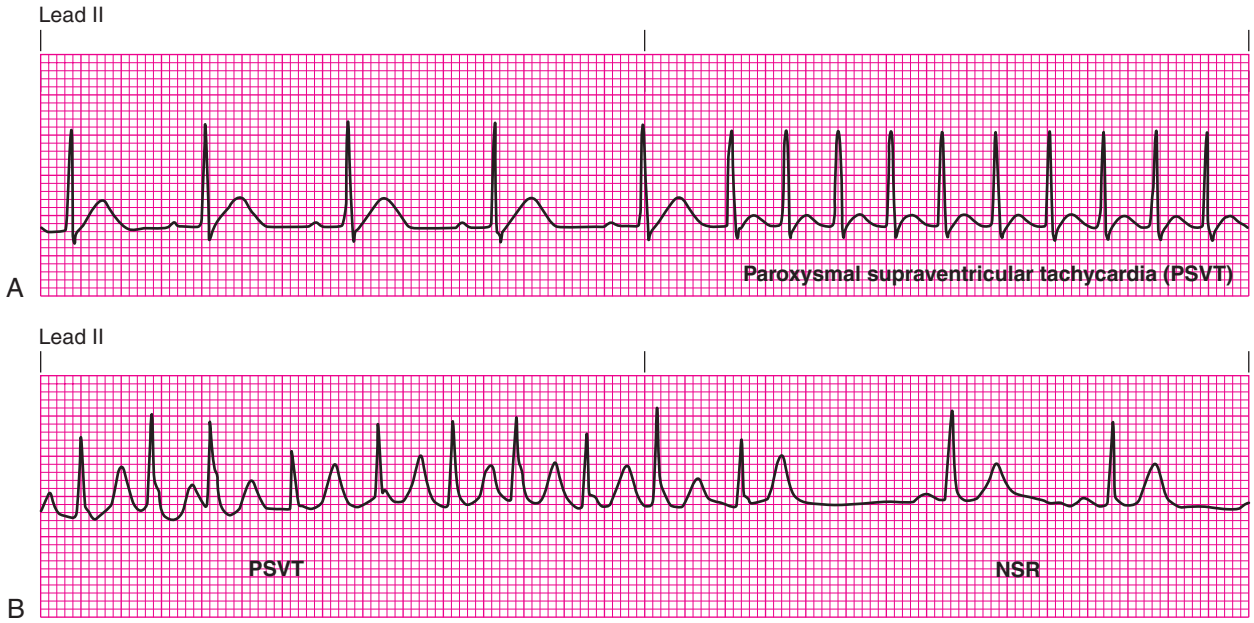


FIGURE 7-40 Paroxysmal supraventricular tachycardia (PSVT). **A,** The abrupt onset initiated by a PJC. **B,** The abrupt cessation of the PSVT.

Paroxysmal Supraventricular Tachycardia

Paroxysmal supraventricular tachycardia (PSVT) occurs above the ventricles, and it has an abrupt onset and cessation. It is initiated by either a PAC or a PJC. An abnormal conduction pathway through the AV node or an accessory pathway around the AV node results in extreme tachycardia. The QRS complex is typically narrow and a P wave may or may not be present. The primary criteria are those of the abrupt onset and cessation of the dysrhythmia (Figure 7-40, *A-B*).

Rhythm analysis

- *Rate:* Heart rate is 150 to 250 beats per minute.
- *Regularity:* The rhythm is regular.
- *Interval measurements:* If the P wave is present, the PR interval is shortened. Other intervals are normal.
- *Shape and sequence:* P wave (if present) and QRS complex are consistent in shape. The QRS complex is narrow and followed by a T wave.
- *Patient response:* The patient may be asymptomatic or symptomatic.
- *Causes:* PSVT often occurs in healthy, young adults without structural heart disease. It may be precipitated by increased catecholamines, stimulants, heart disease, electrolyte imbalances, and anatomical abnormality.
- *Care and treatment:* If the patient is asymptomatic, vagal maneuvers may be attempted. If the patient is symptomatic and the heart rate is over 150 beats per minute, emergent cardioversion is considered. Adenosine or AV nodal blocking agents are usually administered. Once stabilized, the patient is referred for further evaluation by an electrophysiologist.

Dysrhythmias of the Ventricle

Ventricular dysrhythmias arise from ectopic foci in the ventricles. Because the stimulus depolarizes the ventricles in a slower, abnormal way, the QRS complex appears widened and has a bizarre shape. The QRS complex is wider than 0.12 seconds and often wider than 0.16 seconds. The polarity of the T wave is opposite that of the QRS complex.

Depolarization from abnormal ventricular beats rarely activates the atria in a retrograde fashion. Therefore most ventricular dysrhythmias have no apparent P waves. However, if a P wave is present, it is usually seen in the T wave of the following beat; or it has no relationship to the QRS complex and is dissociated from the ventricular rhythm. Ventricular dysrhythmias can be life-threatening, thus fast recognition and intervention is imperative.

Premature Ventricular Contractions

Premature ventricular contractions (PVCs) are a common ventricular dysrhythmia. PVCs are early beats that interrupt the underlying rhythm; they can arise from a single ectopic focus or from multiple foci within the ventricles. A single ectopic focus produces PVC waveforms that look alike, called *unifocal PVCs* ((Figure 7-41, *A, C*). Waveforms of PVCs arising from multiple foci are not identical and are called *multifocal PVCs* (Figure 7-41, *B*). PVCs do not generally reset the sinus node, so the next sinus beat following the pause occurs on time. This is called a compensatory pause.

PVCs may occur in a predictable pattern, such as every other beat, every third beat, or every fourth beat. Box 7-4 lists the nomenclature for early beats. *Bigeminal* PVCs are noted in Figure 7-41, *A*. PVCs can also occur sequentially. Two PVCs in

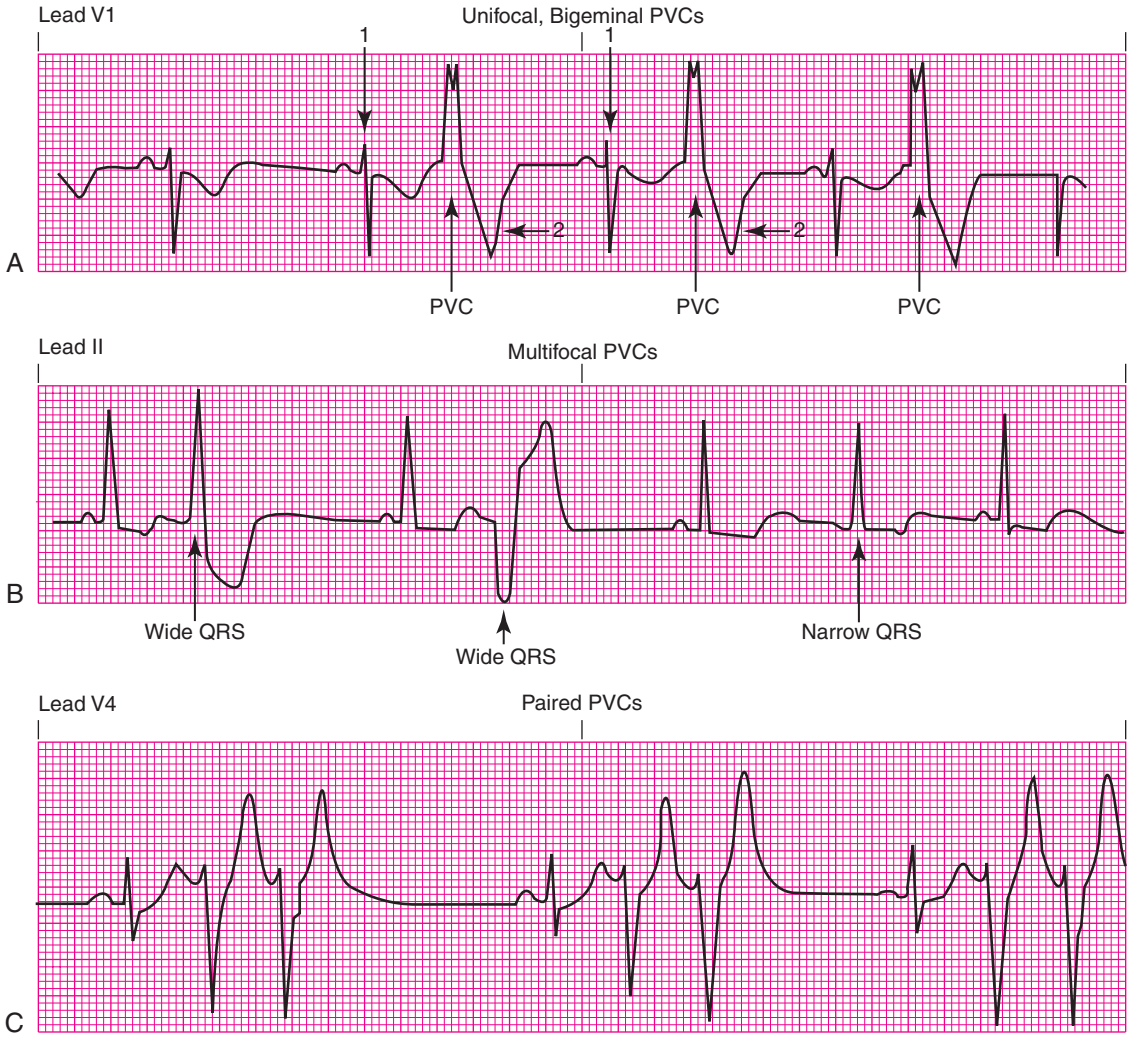


FIGURE 7-41 Premature ventricular contractions (PVCs). **A,** Sinus rhythm with unifocal PVCs. 1 is the sinus beat; 2 points to the presence of an inverted T wave. **B,** Sinus rhythm with multifocal PVCs; note the different configuration of the PVCs, indicating generation from more than one focus. **C,** The PVCs are in pairs and look the same, indicating that they are from the same foci.

a row are called a pair (Figure 7-41, *C*), and three or more in a row are called nonsustained ventricular tachycardia.

The peak of the T wave through the downslope of the T wave is considered the *vulnerable period*, which coincides with partial repolarization of the ventricles. If a PVC occurs during the T wave, ventricular tachycardia may occur. When the R wave of PVC falls on the T wave of a normal beat, it is referred to as the *R-on-T phenomenon* (Figure 7-42, *A*, *B*).

PVCs may occur in healthy individuals and usually do not require treatment. The nurse must determine if PVCs are increasing in number by evaluating the trend. If PVCs are increasing, the nurse should evaluate for potential causes such as electrolyte imbalances, myocardial ischemia or injury, and hypoxemia. Runs of nonsustained ventricular tachycardia may be a precursor to development of sustained ventricular tachycardia.

Rhythm analysis

- *Rate:* The rate matches the underlying rhythm.
- *Regularity:* The rhythm is interrupted by the premature beat.
- *Interval measurements:* There is no PR interval and the QRS complex is greater than 0.12 seconds.
- *Shape and sequence:* The QRS complex of the PVC is wide and bizarre looking. The T wave may be oriented opposite to the direction of the QRS complex of the PVC.
- *Patient response:* PVCs may be experienced as palpitations. Patients may become symptomatic if the PVCs occur frequently.
- *Causes:* Hypoxemia, ischemic heart disease, hypokalemia, hypomagnesemia, acid-base imbalances, and increased catecholamine levels can cause PVCs.
- *Care and treatment:* Treat the cause if PVCs are increasing in frequency.

Ventricular Tachycardia

Ventricular tachycardia (VT) is a rapid, life-threatening dysrhythmia originating from a single ectopic focus in the ventricles. It is characterized by at least three PVCs in a row. VT occurs at a rate greater than 100 beats per minute, but the rate is usually around 150 beats per minute and may be up to

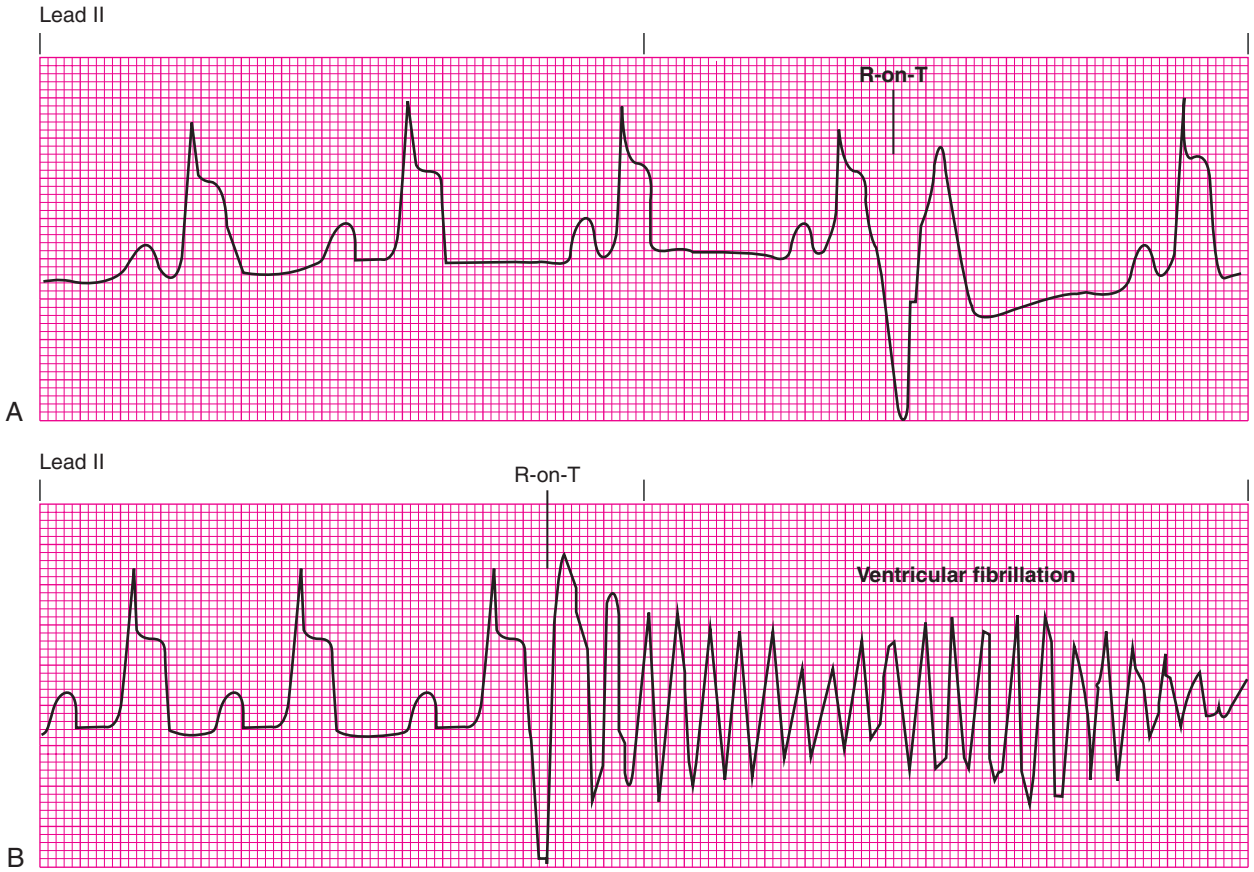


FIGURE 7-42 R-on-T. **A,** Single PVC on T wave. **B,** PVC causing ventricular fibrillation.

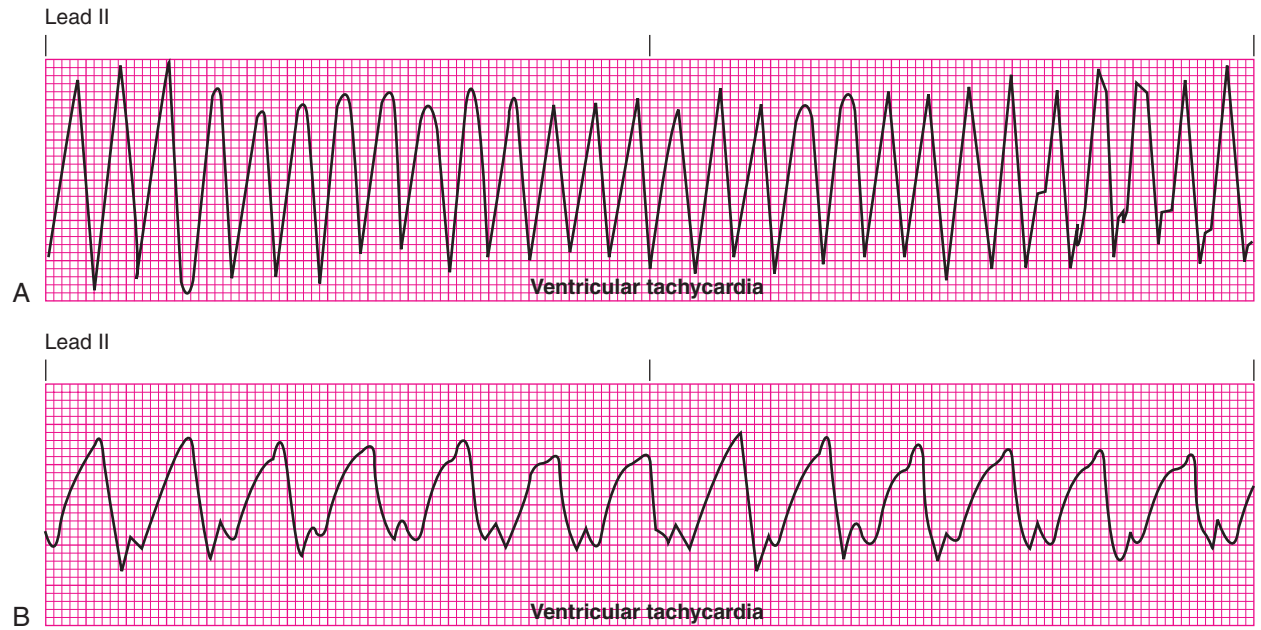


FIGURE 7-43 Ventricular tachycardia.

250 beats per minute. Depolarization of the ventricles is abnormal and produces a widened QRS complex (Figure 7-43). The patient may or may not have a pulse.

The wave of depolarization associated with ventricular tachycardia rarely reaches the atria. Therefore P waves are usually absent. If P waves are present, they have no association with the QRS complex. The sinus node may continue to depolarize at its normal rate, independent of the ventricular ectopic focus. P waves may appear to be randomly scattered throughout the rhythm, but the P waves are actually fired at a consistent rate from the sinus node. This is called *AV dissociation,* another clue that the rhythm is VT. Occasionally a P wave

will "capture" the ventricle because of the timing of atrial depolarization, interrupting the VT with a single capture beat that appears normal and narrow. Then the VT reoccurs. Capture beats are a diagnostic clue to differentiating wide complex tachycardias.

Torsades de pointes ("twisting about the point") is a type of VT that is caused by a prolonged QT interval. Unlike VT, where the QRS complex waveforms have similar shapes, torsades de pointes is characterized by the presence of both positive and negative complexes that move above and below the isoelectric line. This lethal dysrhythmia is treated as pulseless VT.[3] Magnesium deficiency is often a cause of this dysrhythmia.[3] The dysrhythmia can often be prevented by routine measurement of the QT/QTc intervals, especially if the patient is receiving drugs that prolong the QT interval. Increases in QT/QTc interval are reported to the provider, potential drug-related causes are explored, and magnesium levels are monitored and corrected (Figure 7-44).[6]

Rhythm analysis

- *Rate:* The heart rate is 110 to 250 beats per minute.
- *Regularity:* The rhythm is regular unless capture beats occur and momentarily interrupt the VT.
- *Interval measurements:* There is no PR interval. The QRS complex is greater than 0.12 seconds and often wider than 0.16 seconds
- *Shape and sequence:* QRS waves are consistent in shape but appear wide and bizarre. The polarity of the T wave is opposite to that seen in the QRS complex.
- *Patient response:* If enough cardiac output is generated by the VT, a pulse and blood pressure are present. If cardiac output is impaired, the patient has signs and symptoms of low cardiac output; the patient may experience a cardiac arrest.
- *Causes:* Hypoxemia, acid-base imbalance, exacerbation of heart failure, ischemic heart disease, cardiomyopathy, hypokalemia, hypomagnesemia, valvular heart disease, genetic abnormalities, and QT prolongation are all possible causes of VT.
- *Care and treatment:* Determine whether the patient has a pulse. If no pulse is present, provide emergent basic and advanced life support interventions, including defibrillation.[3] If a pulse is present and the blood pressure is stable, the patient can be treated with intravenous amiodarone or lidocaine. Cardioversion is used as an emergency measure in patients who become hemodynamically unstable but continue to have a pulse.

Ventricular Fibrillation

Ventricular fibrillation (VF) is a chaotic rhythm characterized by a quivering of the ventricles, which results in total loss of cardiac output and pulse. VF is a life-threatening emergency, and the more immediate the treatment, the better the survival will be. VF produces a wavy baseline without a PQRST complex (Figure 7-45).

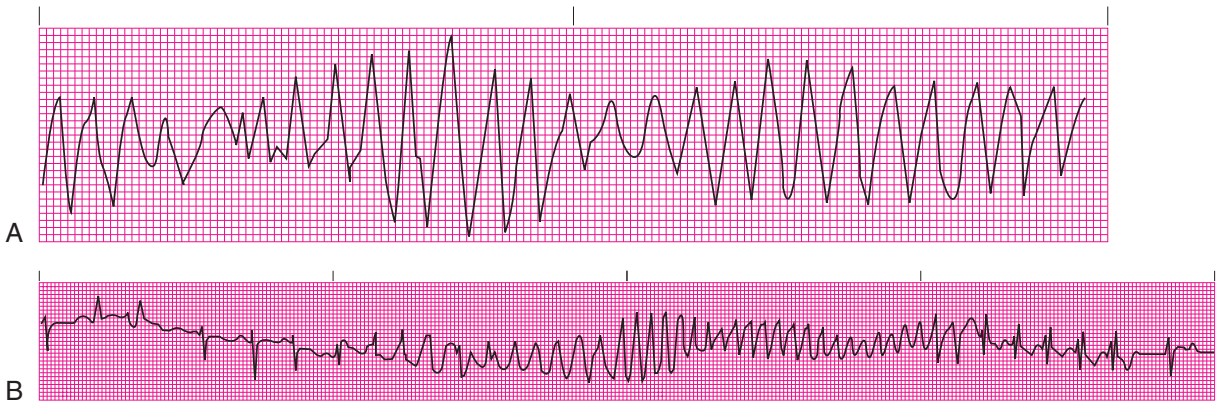
Because a loose lead or electrical interference can produce a waveform similar to VF, it is always important to immediately assess the patient for pulse and consciousness.

Rhythm analysis

- *Rate:* Heart rate is not discernible.
- *Regularity:* Heart rhythm is not discernible.
- *Interval measurements:* There are no waveforms.
- *Shape and sequence:* The baseline is wavy and chaotic, with no PQRST complexes.
- *Patient response:* The patient is in cardiac arrest.
- *Causes:* VF can be caused by ischemic and valvular heart disease, electrolyte and acid-base imbalances, and QT prolongation.
- *Care and treatment:* Immediate BLS and ACLS interventions are required.

Idioventricular Rhythm or Ventricular Escape Rhythm

Idioventricular rhythm is an escape rhythm that is generated by the Purkinje fibers. This rhythm emerges only when the SA and AV nodes fail to initiate an impulse. The Purkinje fibers are capable of an intrinsic rate of 20 to 40 beats per minute. Because this last pacemaker is located in the



A is presented at 100% for a 6-second strip. B has been reduced to 55% of actual size to be able to see a longer waveform over 12 seconds, showing how a patient goes into and comes out of rhythm.

FIGURE 7-44 Torsades de pointes.

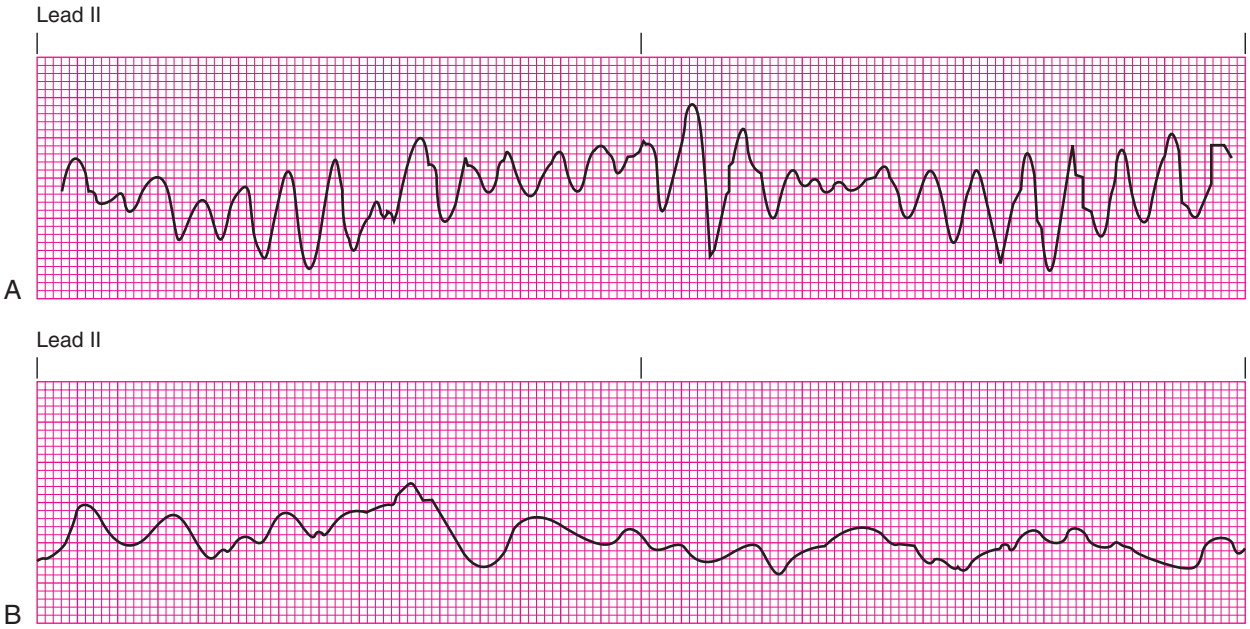


FIGURE 7-45 Ventricular fibrillation. **A,** Course. **B,** Fine.

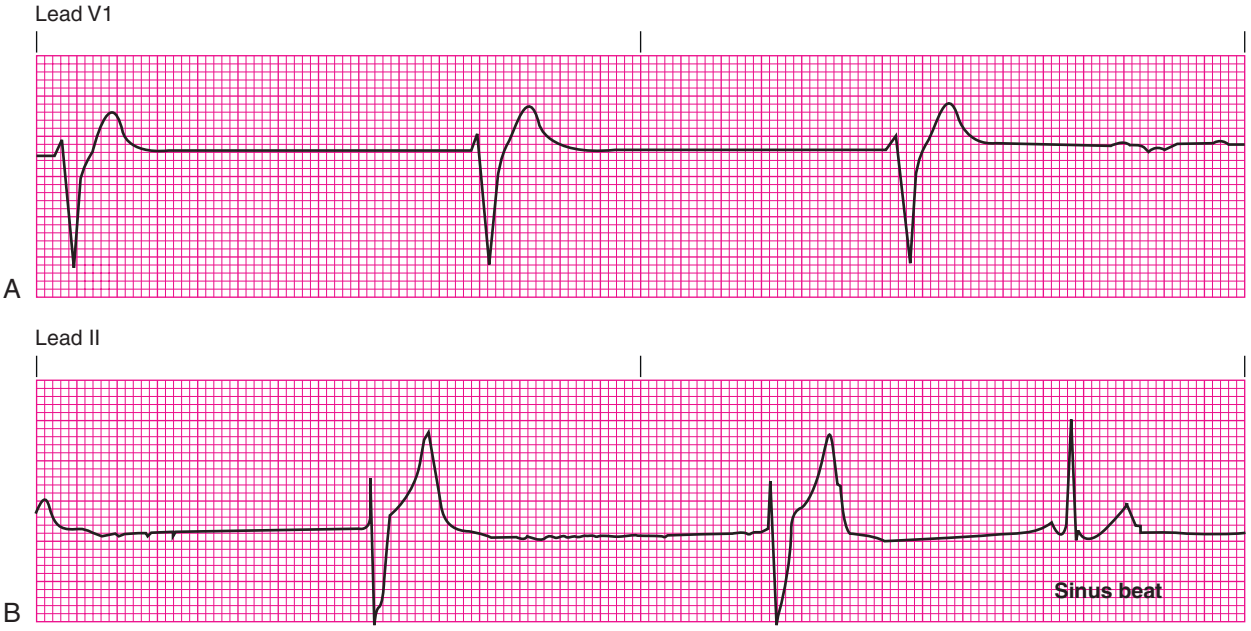


FIGURE 7-46 **A,** Ventricular escape rhythm or idioventricular rhythm. **B,** Two ventricular escape beats followed by sinus rhythm.

ventricles, the QRS complex appears wide and bizarre with a slow rate (Figure 7-46, *A-B*).

An idioventricular rhythm is considered a lethal dysrhythmia because the Purkinje fiber pacemakers may cease to fire, resulting in asystole. A single ventricular escape beat may occur following a pause if the junctional escape pacemaker does not fire (see Figure 7-46, *A-B*).

If the rate is between 40 and 100 beats per minute, this rhythm is called accelerated idioventricular rhythm (AIVR). This wide complex rhythm is often seen following reperfusion of a coronary artery by thrombolytics; percutaneous coronary interventions, such as angioplasty or stent placement; and cardiac surgery (Figure 7-47).

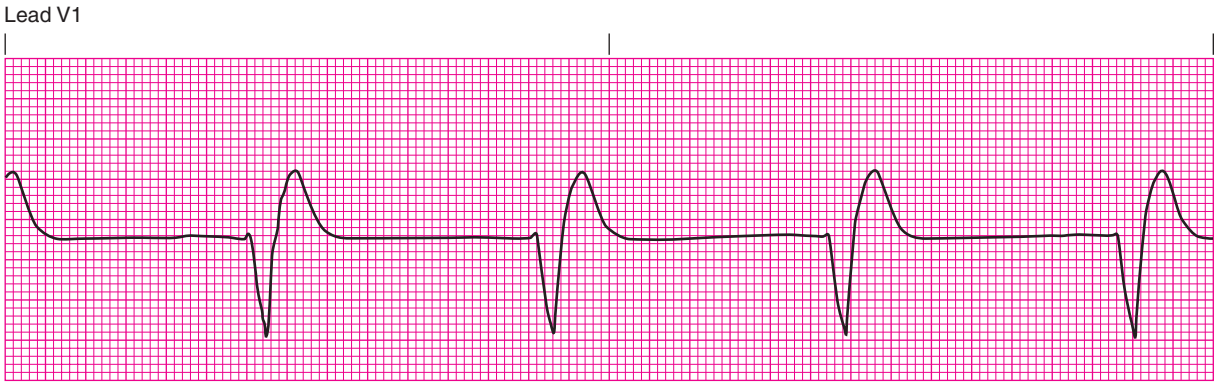


FIGURE 7-47 Accelerated idioventricular rhythm (AIVR).

Rhythm analysis

- *Rate:* The rate of idioventricular rhythm is 20 to 40 beats per minute, and the rate of AIVR is 40 to 100 beats per minute.
- *Regularity:* The rhythm is regular.
- *Interval measurements:* No P waves are present, and the QRS complex is greater than 0.12 seconds.
- *Shape and sequence:* QRS waves are wide and bizarre in shape. The QRS complex is followed by a T wave of opposite polarity.
- *Patient response:* The extreme bradycardia may cause the same symptoms as any severe bradycardia. This mechanism is the last backup pacemaker and asystole may occur.
- *Causes:* Failure of the SA and AV nodal pacemakers causes idioventricular rhythm.
- *Care and treatment:* Initiate BLS and ACLS protocols. Consider emergent transcutaneous pacing.

Asystole

Asystole is characterized by complete cessation of electrical activity. A flat baseline is seen, without any evidence of P, QRS, or T waveforms. A pulse is absent and there is no cardiac output; cardiac arrest has occurred (Figure 7-48, *A*).

Asystole often occurs following VF or ventricular escape rhythm. Following a ventricular escape rhythm this rhythm is referred to as *ventricular standstill* (Figure 7-48, *B*). Pulse should be immediately assessed because a lead or electrode coming off may mimic this dysrhythmia. During cardiac arrest situations, if asystole occurs when another rhythm has been monitored, a check of two leads should occur to confirm asystole.

Rhythm analysis

- *Rate:* Heart rate is absent.
- *Regularity:* Heart rhythm is absent.

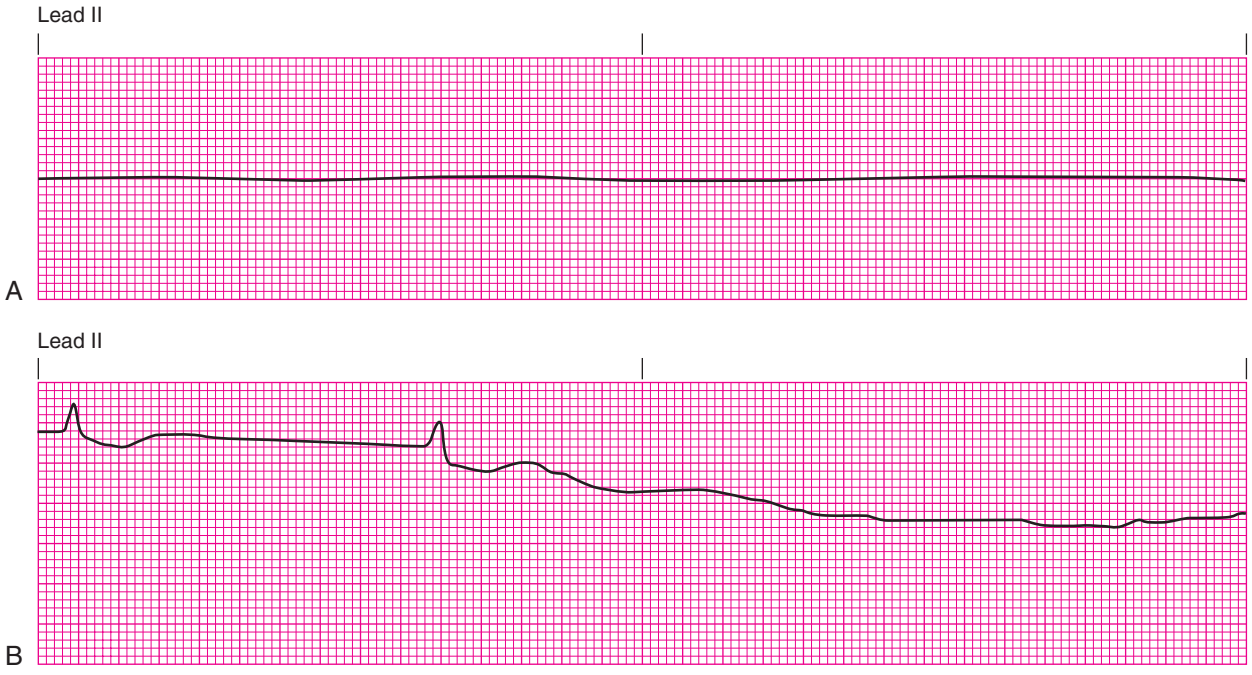


FIGURE 7-48 **A,** Asystole. **B,** Ventricular standstill.

- *Interval measurements:* PQRST waveforms are absent.
- *Shape and sequence:* Waveform is a flat or undulating line on the monitor.
- *Patient response:* The patient is in cardiac arrest.
- *Causes:* Asystole is usually preceded by another dysrhythmia such as VF or ventricular escape rhythm.
- *Care and treatment:* BLS and ACLS protocols are initiated for asystole.

Atrioventricular Blocks

AV block, which is also known as heart block, refers to an inability of the AV node to conduct sinus impulses to the ventricles in a normal manner. AV blocks can cause a delay in conduction from the SA node through the AV node, or completely block conduction intermittently or continuously. AV blocks may arise from normal aging of the conduction system or be caused by damage to the conduction system from ischemic heart disease.

Four types of AV block exist, each categorized in terms of degree. The four types of block are first-degree, second-degree type I, second-degree type II, and third-degree. The greater the degree of block, the more severe are the consequences. First-degree block has minimal consequences, whereas third-degree block may be life-threatening.

First-Degree AV Block

First-degree AV block describes consistent delayed conduction through the AV node or the atrial conductive tissue. It is represented on the ECG as a prolonged PR interval. It is a common dysrhythmia in the elderly and in patients with cardiac disease. As the normal conduction pathway ages or becomes diseased, impulse conduction becomes slower than normal (Figure 7-49).

Rhythm analysis

- *Rate:* Heart rate is determined by the underlying rhythm.
- *Regularity:* The underlying rhythm determines regularity.
- *Interval measurements:* PR interval is prolonged and is greater than 0.20 seconds. QRS complex and QT/QT_c measurements are normal.
- *Shape and sequence:* P and QRS waves are consistent in shape. P waves are small and rounded. A P wave precedes every QRS complex, which is followed by a T wave.
- *Patient response:* AV block is well tolerated.
- *Causes:* Aging and ischemic and valvular heart disease can cause AV block.
- *Care and treatment:* No treatment is required.

Second-Degree Heart Block

Second-degree heart block refers to AV conduction that is intermittently blocked. Two types of second-degree block may occur, and each has specific diagnostic criteria for accurate diagnosis.

Second-degree AV block type I. Also called Mobitz I or Wenckebach phenomenon, second-degree AV block type I is represented on the ECG as a progressive lengthening of the PR interval until there is a P wave without a QRS complex. The AV node progressively delays conduction to the ventricles resulting in a longer PR intervals until finally a QRS complex is dropped. The PR interval following the dropped QRS complex is shorter than the PR interval preceding the dropped beat. By not conducting this one beat, the AV node recovers and is able to conduct the next atrial impulse (Figure 7-50). If dropped beats occur frequently, it is useful to describe the conduction ratio, such as 2:1, 3:1, or 4:1.

Rhythm analysis

- *Rate:* The rate is slower than the underlying rhythm because of the dropped beat.
- *Regularity:* P-P intervals stay the same but the R-R intervals shorten until the dropped beat.
- *Interval measurements:* The PR interval becomes progressively longer until a QRS complex is dropped. The PR interval before the dropped QRS is longer than the PR interval of the next conducted PQRST waveforms.
- *Shape and sequence:* P and QRS waves are consistent in shape. P waves are small and rounded. A P wave precedes every QRS complex, which is followed by a T wave.
- *Patient response:* This rhythm is usually well tolerated unless there is an underlying bradycardia, or frequent dropped beats.
- *Causes:* Aging, AV nodal blocking drugs, acute inferior wall myocardial infarction or right ventricular infarction, ischemic heart disease, digitalis toxicity, and excess vagal response are all possible causes of second-degree AV block type I.
- *Care and treatment:* This type of block is usually well tolerated and no treatment is indicated unless the dropped beats occur frequently. If the patient is symptomatic,

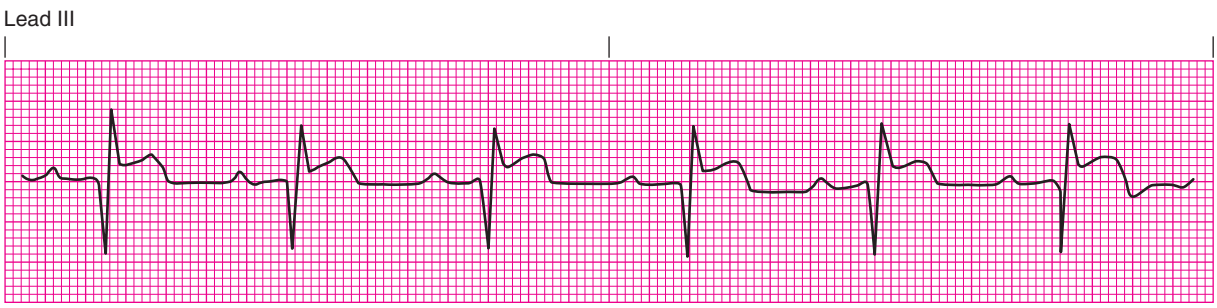


FIGURE 7-49 First-degree AV block.

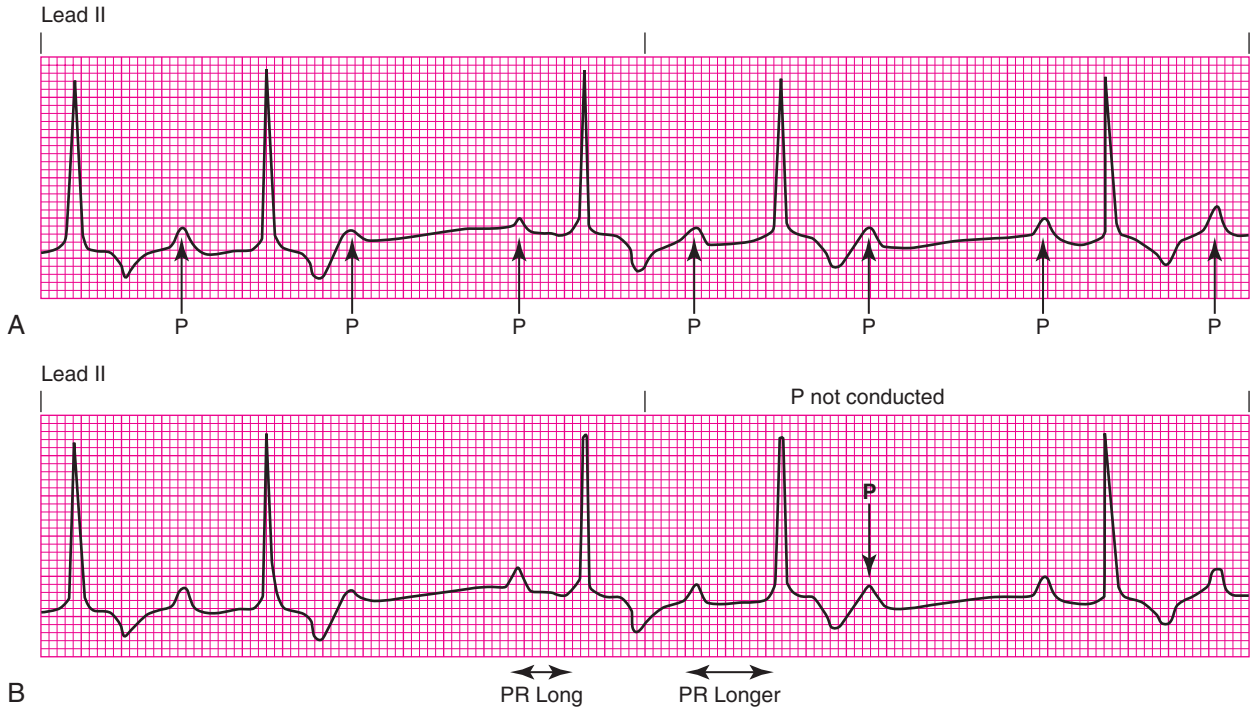


FIGURE 7-50 Second-degree AV block type I (Mobitz I, Wenckebach).

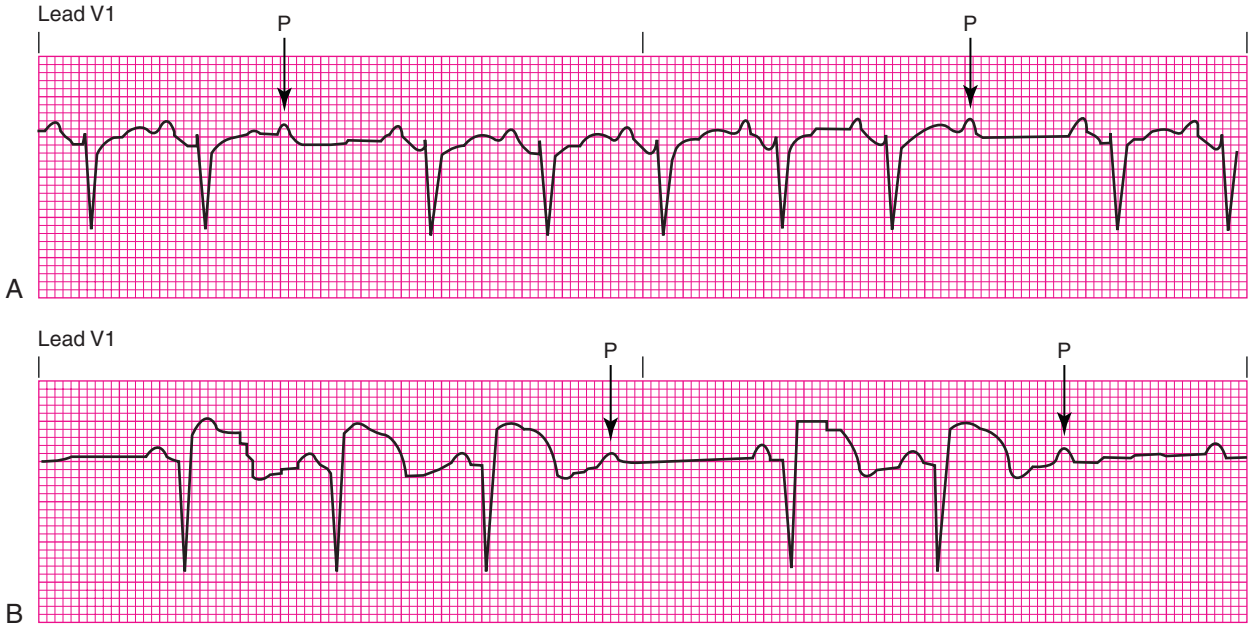


FIGURE 7-51 Second-degree AV block type II (Mobitz II). **A,** The PR interval is constant; at the 3rd and 9th P waves, there is no QRS following the P wave. **B,** No QRS follows the 4th and 7th P waves.

drugs that may contribute to the rhythm are discontinued, and potentially a permanent pacemaker may be considered in selected individuals (rare).

Second-degree AV block type II. Second-degree AV block type II (Mobitz II) is a more critical type of heart block that requires early recognition and intervention. The conduction abnormality occurs below the AV node, either in the bundle of His or the bundle branches. A P wave is generated but is not conducted to the ventricles for one or more beats. The PR interval remains the same throughout with the exception of the dropped beat(s) (Figure 7-51). Second-degree block type II is often associated with a bundle branch block and

a corresponding widened QRS complex; however, narrow QRS complexes may be observed. Second-degree block type II can progress to the more clinically significant third-degree block and may cause the patient to be symptomatic.

Rhythm analysis

- *Rate:* Heart rate is slower than the underlying rhythm because of the dropped beats.
- *Regularity:* P waves are regular, but QRS complexes are occasionally absent.
- *Interval measurements:* Intervals are constant for the underlying rhythm. PR intervals of the conducted beats do not change. QRS complexes may be widened because of a bundle branch block.
- *Shape and sequence:* P and QRS waves are consistent in shape. P waves are small and rounded. QRS complexes are missing.
- *Patient response:* The patient may tolerate one missed beat, but symptoms may occur if frequent beats are missed.
- *Causes:* Heart disease, increased vagal tone, conduction system disease, ablation of the AV node, and inferior and right ventricular myocardial infarctions are possible causes of second-degree AV block type II.
- *Care and treatment:* Patient may require a pacemaker, administration of atropine, and transcutaneous or transvenous pacing for emergent treatment.[3]

Third-Degree Block

Third-degree block is often called *complete heart block* because no atrial impulses are conducted through the AV node to the ventricles. The block in conduction can occur at the level of the AV node, the bundle of His, or the bundle branches.

In complete heart block, the atria and ventricles beat independently of each other because the AV node is completely blocked to the sinus impulse and it is not conducted to the ventricles. An escape rhythm arises from the junctional tissue or the ventricles. The atria beat at one rate, and the ventricles beat at a different rate. The atrial rate is dictated by the sinus node. The ventricular rate is slow, and usually only a ventricular or junctional escape rhythm is present. No communication exists between the atria and ventricles. Third-degree block is a type of AV dissociation (Figure 7-52).

One hallmark of third-degree heart block is that the P waves have no association with the QRS complexes and appear throughout the QRS waveform. Both the P-P and R-R intervals are regular, but the rates for each are different because they have no relationship to each other. Whenever a rhythm strip appears to have no consistent, predictable relationship between P waves and QRS complexes, third-degree block is considered.

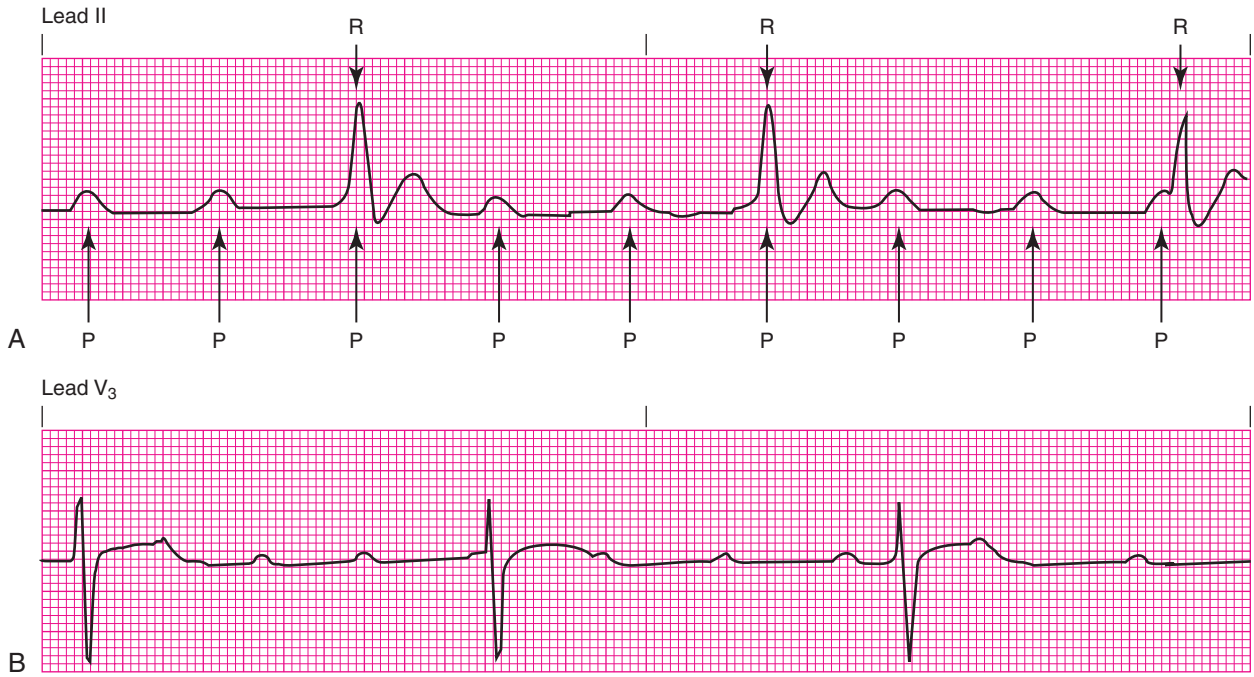


FIGURE 7-52 Third-degree AV block (complete heart block). **A,** P waves regular and present throughout at an atrial rate of 94 beats per minute and ventricular rhythm of 30 beats per minute. The P waves are not associated with the QRS complexes. **B,** Similar tracing; atrial rate 100 and ventricular rate of 30.

Rhythm analysis

- *Rate:* The atrial rate is greater than the ventricular rate.
- *Regularity:* P-P intervals are regular and R-R intervals are regular, but they not associated with each other.
- *Interval measurements:* There is no PR interval in the absence of conduction. The QRS complex is often widened greater than 0.12 seconds with a ventricular escape rhythm.
- *Shape and sequence:* P and QRS waves are consistent in shape. P waves are small and rounded. The QRS complex is followed by a T wave. There is no relationship between the P waves and QRS complexes.
- *Patient response:* Patients may become symptomatic because of the bradycardia of the escape rhythm.
- *Causes:* Ischemic heart disease, acute myocardial infarction, and conduction system disease are possible causes of third-degree heart block.
- *Care and treatment:* Treatments include transcutaneous or transvenous pacing and implanting a permanent pacemaker.

CARDIAC PACEMAKERS

A cardiac pacemaker delivers electrical current to the myocardium to stimulate depolarization when the heart rate is too slow or the heart is unable to initiate and/or conduct a native beat. A pacemaker is often implanted to treat symptomatic bradycardia, which may occur from a number of different pathophysiological conditions. These include second-degree AV block type II, third-degree AV block, and sick sinus syndrome. The need for a pacemaker may be temporary (e.g., after an acute myocardial infarction or cardiac surgery) or permanent. Battery-operated, external pulse generators are used to provide electrical energy for temporary transvenous pacemakers. Implanted permanent pacemakers are used to treat chronic conditions. These devices have a battery life of up to 10 years, which varies based on the manufacturer's recommendations.

It is important that patients be assessed for the need for pacing. Unnecessary pacing may lead to worsened outcomes, including heart failure, rehospitalization, increased mortality, and new onset of atrial fibrillation.[7]

Temporary Pacemakers

Types of temporary pacemakers include the following:

- *Transcutaneous:* Electrical stimulation is delivered through the skin via external electrode pads connected to an external pacemaker (a defibrillator with pacemaker functions; see Chapter 10).
- *Transvenous:* A pacing catheter is inserted (Figure 7-53) percutaneously into the right ventricle, where it contacts the endocardium near the ventricular septum. It is connected to a small external pulse generator (Figure 7-54) by electrode wires. Note the electrical ports on the pacing catheter which are covered by black caps (see Figure 7-53). These are connected to the pulse generator (see Figure 7-54) whereupon pacing thresholds are set for each specific patient.

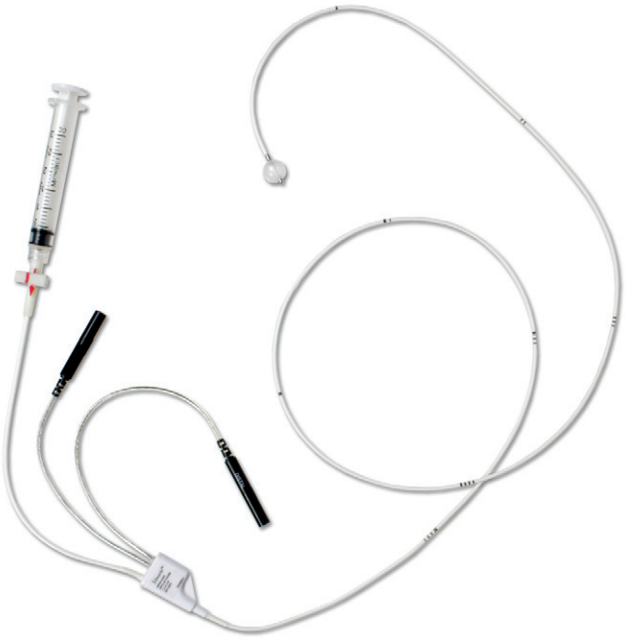


FIGURE 7-53 Balloon-tipped bipolar lead wire for transvenous pacing. (From Wiegand DLM: *AACN Procedure Manual for Critical Care*. 6th ed. St. Louis: Elsevier, 2011.)

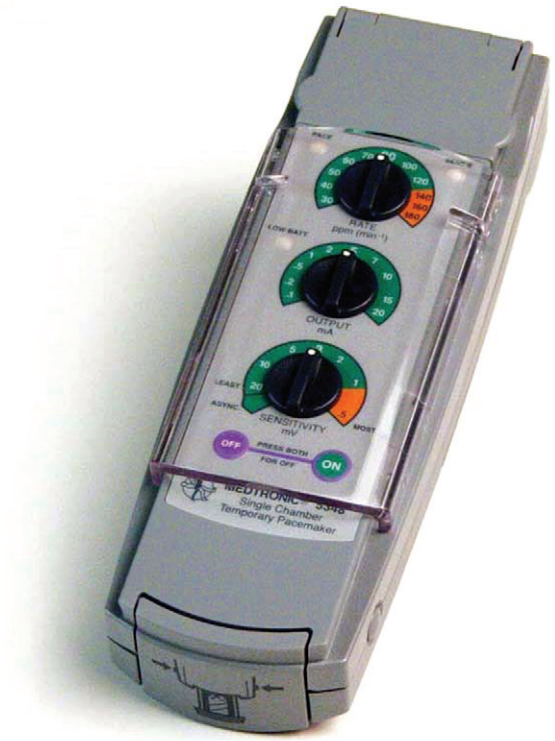


FIGURE 7-54 Single-chamber temporary pulse generator. (Courtesy Medtronic USA, Inc. Minneapolis, Minnesota).

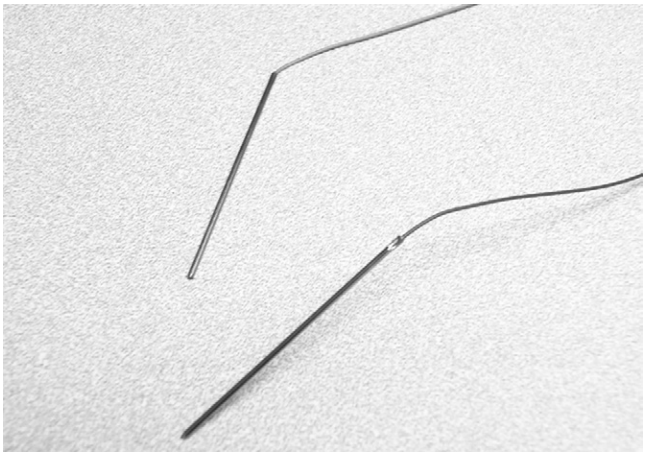


FIGURE 7-55 Epicardial wires. (From Wiegand DLM: *AACN Procedure Manual for Critical Care.* 6th ed. St. Louis: Elsevier, 2011.)

- *Epicardial:* Pacing wires are inserted into the epicardial wall of the heart during cardiac surgery (Figure 7-55); wires are brought through the chest wall and can be connected to a pulse generator if needed (Figure 7-56). Note that there are only two pacing wires shown in Figure 7-55; however, in cardiac bypass surgery patients, there are often four wires placed through the chest wall of the patient, two wires from the atrium and two wires from the ventricles. These four wires are connected to the temporary pacemaker (see Figure 7-56), and pacing thresholds are set for each patient.

FIGURE 7-56 Dual-chamber temporary pulse generator. (Courtesy Medtronic USA, Inc. Minneapolis, Minnesota).

Permanent Pacemakers

Permanent pacemakers have electrode wires that are typically placed transvenously through the cephalic or subclavian vein into the heart chambers (Figure 7-57). The leads are attached to the pulse generator, placed in a surgically created pocket just below the left clavicle.

Pacemakers may be used to stimulate the atrium, ventricle, or both chambers (dual-chamber pacemakers). Atrial pacing is used to mimic normal conduction and to produce atrial contraction, thus providing atrial kick. Ventricular pacing stimulates ventricular depolarization and is commonly used in emergency situations or when pacing is required infrequently. Dual-chamber pacing allows for stimulation of both atria and ventricles as needed to synchronize the chambers and mimic the normal cardiac cycle.

Permanent pacemakers may be programmed in a variety of ways, and a standardized code is used to determine the pacing mode that is programmed. The North American Society of Pacing and Electrophysiology (NASPE) and the British Pacing and Electrophysiology Group have revised the standardized generic code for pacemakers; it is described in Chapter 12. It is important to know the programming information for the pacemaker to assess proper functioning on the rhythm strip.

Terms for Pacemaker Function

Other terms used in describing pacemaker function are *mode, rate, electrical output, sensitivity, sense-pace indicator,* and *AV interval.*

Mode. Pacemakers can be operated in a *demand* mode or *fixed rate (asynchronous)* mode. The demand mode paces the heart when no intrinsic or native beat is sensed. For example, if the rate control is set at 60 beats per minute, the pacemaker will only pace if the patient's heart rate drops to less than 60. The fixed rate mode paces the heart at a set rate, independent of any activity the patient's heart generates. The fixed rate mode may compete with the patient's own rhythm and deliver an impulse on the T wave (R on T), with the potential for producing ventricular tachycardia or fibrillation. The demand mode is safer and is the mode of choice.

Rate. The rate control determines the number of impulses delivered per minute to the atrium, the ventricle, or both. The rate is set to produce effective cardiac output and to reduce symptoms.

Electrical output. The electrical output is the amount of electrical energy needed to stimulate depolarization. The output is measured in *milliamperes (mA)*, of which it varies depending on the type of pacing. Transcutaneous pacing requires higher milliamperes than transvenous or epicardial pacing, because the electrical energy must be delivered through the chest wall.

Sensitivity. The sensitivity is the ability of the pacemaker to recognize the body's intrinsic or native electrical activity. It is measured in *millivolts (mV)*. Some temporary pacemakers

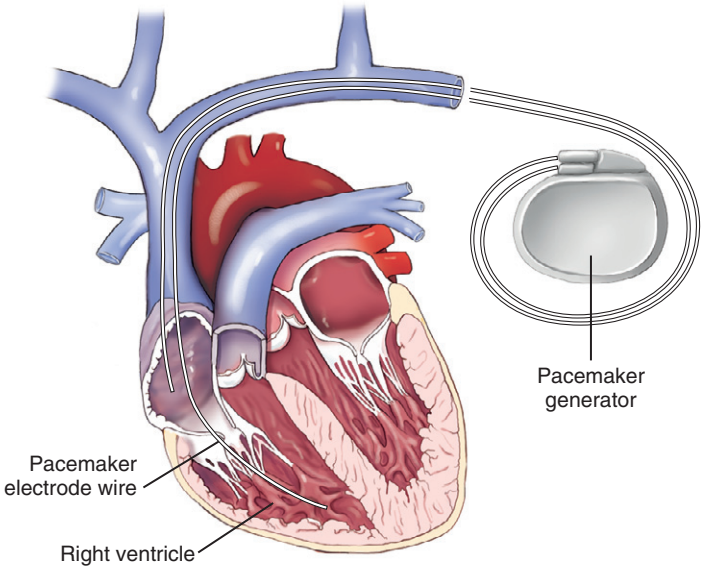


FIGURE 7-57 Permanent dual chamber (A-V) pacemaker. (From Wesley K. *Huszar's Basic Dysrhythmias and Acute Coronary Syndromes: Interpretation and Management*. 4th ed. St. Louis: Mosby JEMS; 2010.)

have a *sense-pace indicator.* If the generator detects the patient's own beat, the "sense" indicator lights. When the generator delivers a paced beat, the "pace" light comes on. Temporary pacemakers have dials or key pads for adjusting sensitivity.

AV interval indicator. The AV interval indicator is used to determine the interval between atrial and ventricular stimulation. It is used only in dual-chamber pacemakers.

Pacemaker Rhythms

Pacemaker rhythms are usually easy to identify on the cardiac monitor or rhythm strip. The electrical stimulation is noted by an electrical artifact called the *pacer spike*. If the atrium is paced, the spike appears before the P wave (Figure 7-58). If the ventricle is paced, the spike appears before the QRS complex (Figure 7-59). If both the atrium and ventricle are paced,

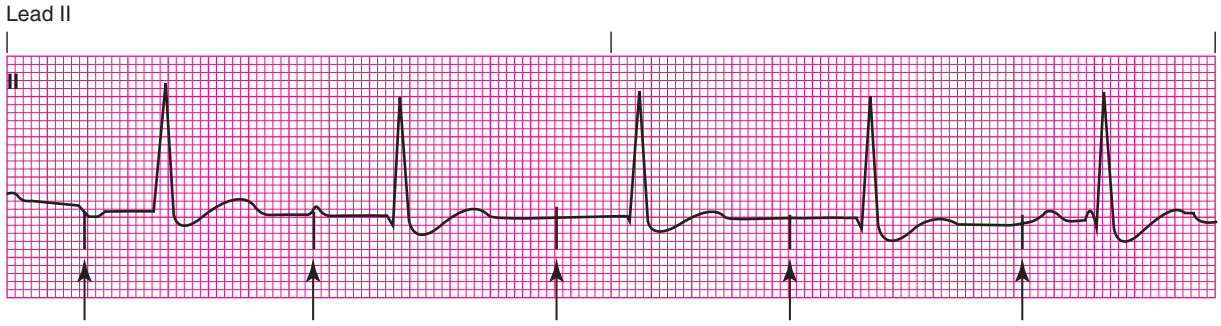


FIGURE 7-58 Atrial paced rhythm.

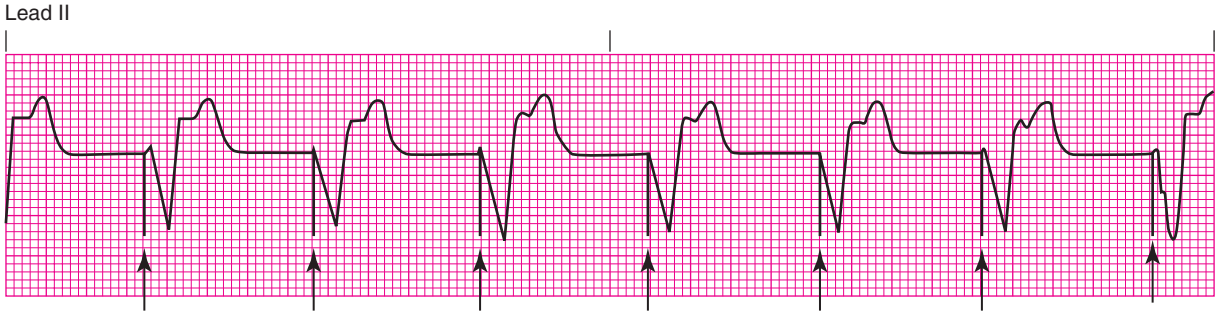


FIGURE 7-59 Ventricular paced rhythm.

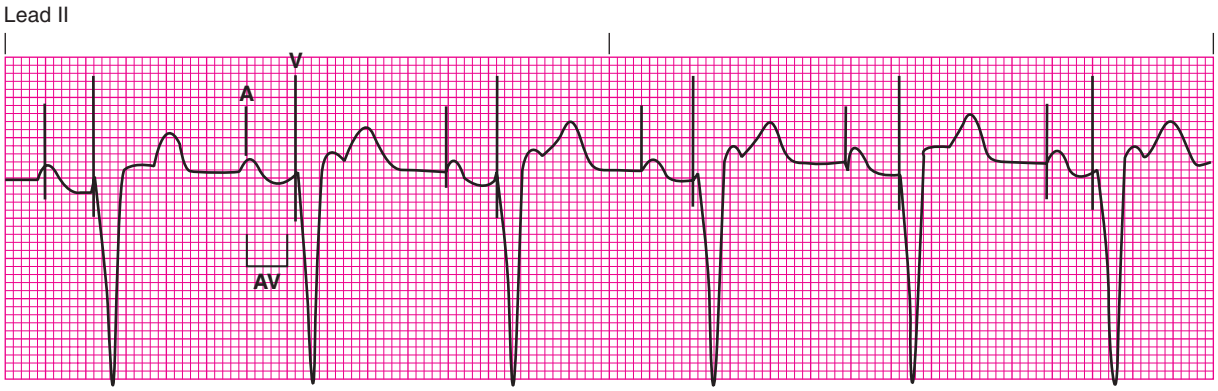


FIGURE 7-60 Dual-chamber (AV) paced rhythm. *A,* Atrial pacer spike; *AV,* AV pager spike interval; *V,* Ventricular paced spike.

spikes are noted before both the P wave and the QRS complex (Figure 7-60). The heart rate is carefully assessed on the rhythm strip. The heart rate should not be lower than the rate set on the pacemaker.

The pacemaker spike is usually followed by a larger-than-normal P wave in atrial pacing or a widened QRS complex in ventricular pacing. Sometimes the P wave is not seen even though an atrial pacer spike is present. Because the heart is paced in an artificial or abnormal fashion, the path of depolarization is altered, resulting in waveforms and intervals that are also altered.

Pacemaker Malfunction

Three primary problems can occur with a pacemaker. These problems include *failure to pace* (also called failure to fire), *failure to capture*, and *failure to sense*. If troubleshooting does not resolve pacemaker malfunction, emergency transcutaneous pacing may be needed to ensure an adequate cardiac output.

Failure to pace. *Failure to pace or fire* occurs when the pacemaker fails to initiate an electrical stimulus when it should fire. The problem is noted by absence of pacer spikes on the rhythm strip. Causes of failure to pace include battery or pulse generator failure, fracture or displacement of a pacemaker wire, or loose connections (Figure 7-61).

Failure to capture. When the pacemaker generates an electrical impulse (pacer spike) and no depolarization is noted, it is described as *failure to capture*. On the ECG, a pacer spike is noted, but it is not followed by a P wave (atrial pacemaker) or a QRS complex (ventricular pacemaker) (Figure 7-62). Common causes of failure to capture include output (milliamperes) set too low, or displacement of the

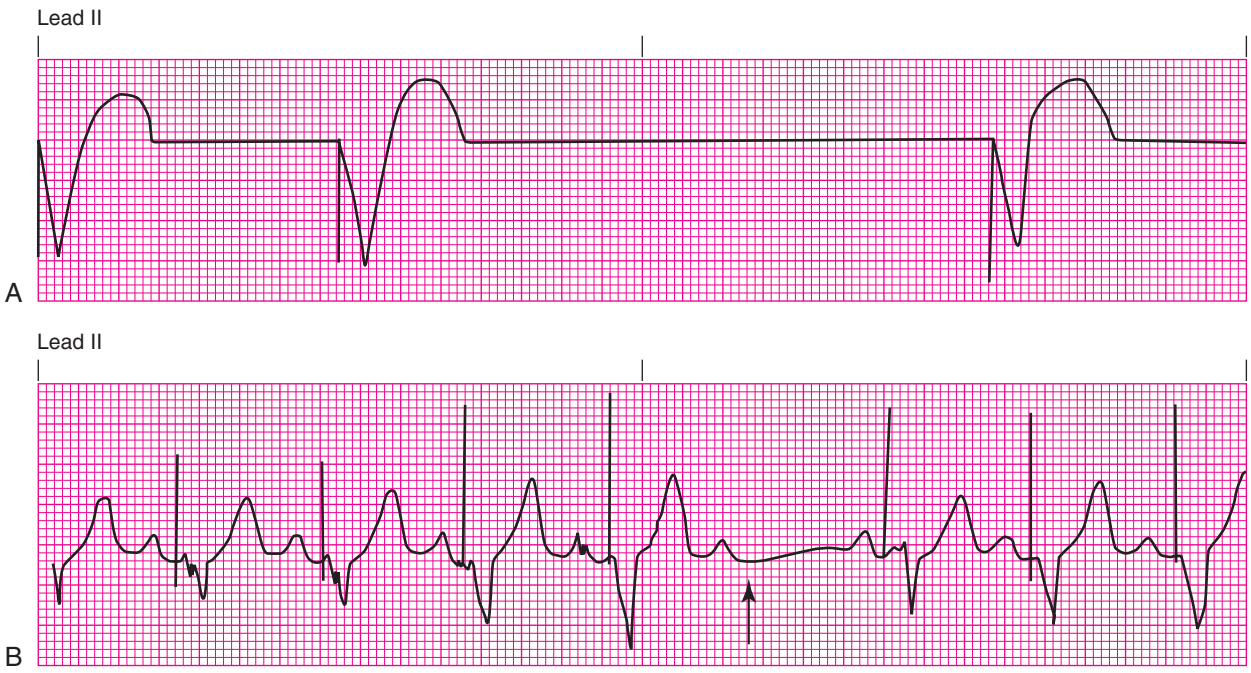


FIGURE 7-61 Failure to pace/fire.

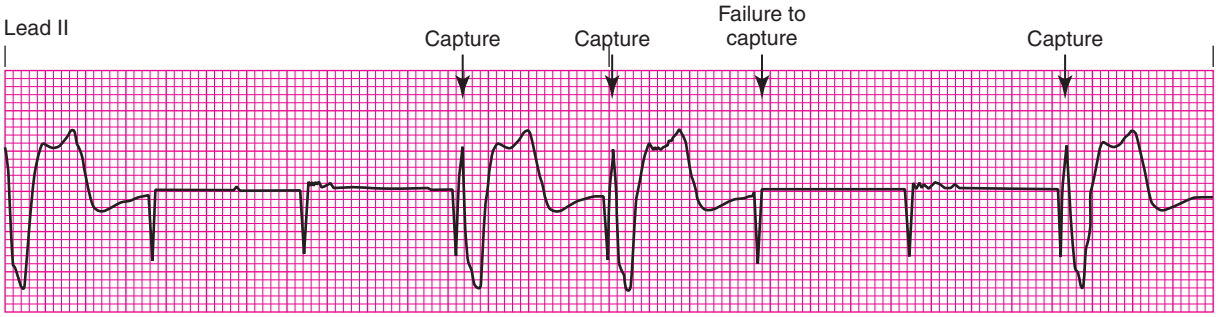


FIGURE 7-62 Failure to capture: ventricular pacemaker.

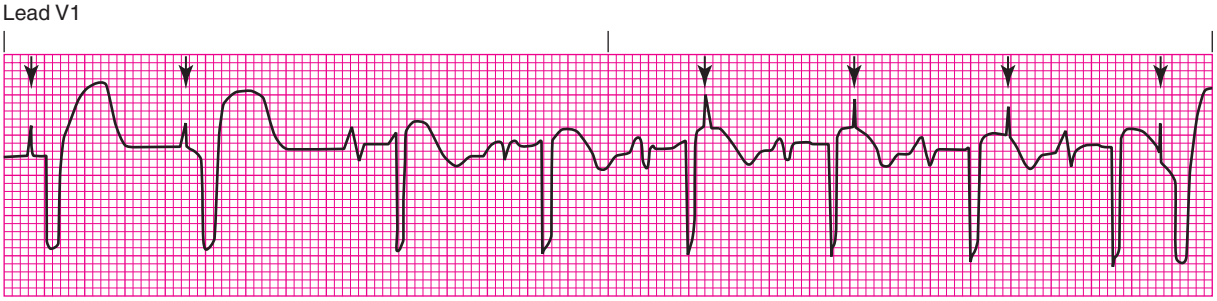


FIGURE 7-63 Failure to sense.

pacing lead wire from the myocardium (transvenous or epicardial leads). Other causes of failure to capture include battery failure, fracture of the pacemaker wire, or increased pacing threshold as a result of medication or electrolyte imbalance. Adjusting the output if the patient has a temporary pacemaker, and placing the patient on his or her left side are nursing interventions to treat failure to capture. Turning the patient onto the left side facilitates contact of a transvenous pacing wire with the endocardium and septum.

Failure to sense. When the pacemaker does not sense the patient's own cardiac rhythm and initiates an electrical impulse, it is called *failure to sense*. Failure to sense manifests as pacer spikes that fall too closely to the patient's own rhythm, earlier than the programmed rate (Figure 7-63). The most common cause is displacement of the pacemaker electrode wire. Turning the patient to the left side and adjusting the sensitivity (temporary pacemaker) are nursing interventions to use when failure to sense occurs.

OTHER DEVICES WITH PACEMAKER CAPABILITIES

All implantable cardioverter-defibrillators (ICDs) have pacemaker capabilities. The pacemaker feature of these devices is used to treat fast heart rhythms, such as VT, with antitachycardia pacing, as well as slow heart rhythms that may occur following defibrillation. Antitachycardia pacing is a short, fast burst of pacing impulses that attempt to terminate the tachycardia.[10]

Biventricular pacemakers and ICDs have an additional electrode wire placed through the coronary sinus into the left ventricle. Additional pacing wires are in the atria and the ventricle. Pacing both ventricles simultaneously improves heart function in a certain number of heart failure patients. Synchronous depolarization of both ventricles improves cardiac output and ejection fraction.[10] Many patients with ICDs benefit from telemonitoring technologies (see box, "QSEN Exemplar").

QSEN EXEMPLAR

Informatics

Integrating Clinical Informatics with Emerging Critical Care Technologies

Use of internal cardioverter-defibrillators (ICDs) is becoming more commonplace in the primary and secondary prevention of sudden cardiac death. Patients who receive these devices require evaluation during the immediate postimplantation period and frequent ongoing assessment for the duration of the implantation. Remote patient-oriented telemonitoring using a team approach may augment the clinical management and safety of patients with ICDs. Remote monitoring has the potential to diagnose dysrhythmias and device-related performance issues in real time. Shirato presented an overview of remote ICD telemonitoring methods, advantages and disadvantages of this form of telehealth, and legal and ethical concerns related to this emerging technology.

Reference

Shirato, S. (2009). The use of remote monitoring for internal cardioverter defibrillators (ICDs): The infusion of information technology and medicine. *Online Journal of Nursing Informatics, 13*(3), 1-16.

CASE STUDY

Mr. P. is a 56-year-old man who was successfully extubated (endotracheal tube removed) 4 hours after coronary artery bypass graft surgery. However, 2 hours later, the patient complains of his heart racing, and it is determined that he has palpitations. The heart rate on the bedside monitor is 168 beats per minute, blood pressure is 100/60 mm Hg, and respiratory rate is 26 breaths per minute. The ECG shows an irregularly irregular rhythm, a change from the sinus rhythm noted at the last assessment.

Questions

1. Based on this description, what is your interpretation of the rhythm?
2. What complications could occur as a result of this rhythm?
3. What clinical data would lead you to believe this complication could occur?
4. What data are you going to give to the provider?
5. What orders do you expect to get?
6. What nursing actions do you need to take and why?
7. What are some of the etiologies of this dysrhythmia?

SUMMARY

Interpretation of cardiac rhythms is an essential skill that is developed through practice and clinical experience. For the beginning student, the critical criteria for diagnosis provide the structure by which rhythms are analyzed. The initial effort is focused on learning these criteria. It is hoped that this chapter is a valuable reference in the delivery of high-quality care to patients with cardiac dysrhythmias and to their families. Care for a patient with dysrhythmias is summarized in the nursing care plan.

NURSING CARE PLAN

Patient with Dysrhythmias

NURSING DIAGNOSIS

Risk for Decreased Cardiac Tissue Perfusion related to altered perfusion.

PATIENT OUTCOMES

Adequate tissue perfusion

- Strong peripheral pulses
- Blood pressure within normal limits for patient
- Skin warm and dry
- Lungs clear bilaterally
- Urine output greater than 30 mL/hr
- Regular cardiac rhythm

NURSING INTERVENTIONS	RATIONALES
• Assess patient for tachycardia, bradycardia and/or irregularity of cardiac rhythm; monitor vital signs for change from baseline	• Early recognition and treatment will prevent further deterioration of patient condition and end organ damage
• Assess for signs of reduced cardiac output: rapid, slow, or weak pulse; hypotension; dizziness; syncope; shortness of breath; chest discomfort; fatigue; change in level of consciousness or restlessness	• Detect subtle signs of decreased cardiac output because many dysrhythmias affect cardiac output
• Determine if dysrhythmia is acute or chronic	• Assess and guide treatment
• Review history and assess for causative factors	• Identify factors causing dysrhythmias that can be eliminated or corrected, such as cardiac ischemia, hypoxemia, and electrolyte imbalance
• Determine specific type of dysrhythmia	• Guide assessment and treatment according to current guidelines
• Determine appropriate lead selection(s) based on current monitoring standards and individualized to patient's diagnosis	• Differentiate atrial from ventricular and lethal from nonlethal dysrhythmias; detect changes in ST segment that may indicate ischemia, injury, or infarct
• Obtain baseline QT/QTc measurements and regularly monitor with other intervals	• Many current medications can cause QT prolongation, placing the patient at risk for torsades de pointes
• Provide psychological support to the patient and family; provide education as to the purpose of ECG monitoring	• Decrease stressors of the hospitalized patient
• Provide oxygen therapy	• Relieve dysrhythmias associated with hypoxemia and myocardial ischemia
• If the patient has a new-onset acute dysrhythmia, assess the patient immediately, then obtain a 12-lead ECG to document the rhythm; use rhythm recording of all 12 leads if available on bedside monitor	• Capture elusive dysrhythmias that are intermittent, or disclose P waves in SVTs, which will guide diagnosis and treatment
• Assess urine output hourly	• Assess adequacy of perfusion to kidneys

ECG, Electrocardiogram.

Based on data from Gulanick M and Myers JL. *Nursing Care Plans: Diagnoses, Interventions, and Outcomes,* 7th ed. St. Louis; Mosby; 2011.

CRITICAL THINKING EXERCISES

1. You are working in the critical care unit and your patient's heart rate suddenly decreases from 88 to 50 beats per minute. What may be some of the reasons for the decreased heart rate? What assessments will you make?
2. Discuss why patients with pulmonary disease are prone to atrial dysrhythmias.
3. A 65-year-old woman with type 2 diabetes presents to the emergency department; she is short of breath and complaining of neck and shoulder pain. Her blood pressure is 185/95 mm Hg, and her heart rate is 155 beats per minute. How will you initially manage this patient? What medical intervention would you anticipate? List serious signs and symptoms of hemodynamic instability in a patient with a tachydysrhythmia.
4. Why does tachycardia sometimes lead to heart failure?

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