The ability to recognize normal and abnormal cardiac rhythms, called dysrhythmias, is an essential skill for the nurse.

Four properties of cardiac cells (automaticity, excitability, conductivity, and contractility) enable the conduction system to initiate an electrical impulse, transmit it through the cardiac tissue, and stimulate the myocardial tissue to contract.

- A normal cardiac impulse begins in the sinoatrial (SA) node in the upper right atrium.
- The signal is transmitted over the atrial myocardium via Bachmann’s bundle and internodal pathways, causing atrial contraction.
- The impulse then travels to the atrioventricular (AV) node through the bundle of His and down the left and right bundle branches, ending in the Purkinje fibers, which transmit the impulse to the ventricles, resulting in ventricular contraction.

The autonomic nervous system plays an important role in the rate of impulse formation, the speed of conduction, and the strength of cardiac contraction.

- Components of the autonomic nervous system that affect the heart are the right and left vagus nerve fibers of the parasympathetic nervous system and fibers of the sympathetic nervous system.

**ECG MONITORING**

- The electrocardiogram (ECG) is a graphic tracing of the electrical impulses produced in the heart.

- ECG waveforms are produced by the movement of charged ions across the semipermeable membranes of myocardial cells.

- There are 12 recording leads in the standard ECG.
  - Six of the 12 ECG leads measure electrical forces in the frontal plane (leads I, II, III, aVR, aVL, and aVF).
  - The remaining six leads (V₁ through V₆) measure the electrical forces in the horizontal plane (precordial leads).
  - The 12-lead ECG may show changes that are indicative of structural changes, damage such as ischemia or infarction, electrolyte imbalance, dysrhythmias, or drug toxicity.

- Continuous ECG monitoring is done using leads II, V₁, and MCL₁.
  - MCL₁ is a modified chest lead that is similar to V₁ and is used when only three leads are available for monitoring.
  - Monitoring leads should be selected based on the patient’s clinical situation.

- The ECG can be visualized continuously on a monitor oscilloscope, and a recording of the ECG (i.e., rhythm strip) can be obtained on ECG paper attached to the monitor.

- ECG leads are attached to the patient’s chest wall via an electrode pad fixed with electrical conductive paste.

- **Telemetry monitoring** involves the observation of a patient’s HR and rhythm to rapidly diagnose dysrhythmias, ischemia, or infarction.
Normal sinus rhythm refers to a rhythm that originates in the SA node and follows the normal conduction pattern of the cardiac cycle.

- The P wave represents the depolarization of the atria (passage of an electrical impulse through the atria), causing atrial contraction.
- The PR interval represents the time period for the impulse to spread through the atria, AV node, bundle of His, and Purkinje fibers.
- The QRS complex represents depolarization of the ventricles (ventricular contraction), and the QRS interval represents the time it takes for depolarization.
- The ST segment represents the time between ventricular depolarization and repolarization. This segment should be flat or isoelectric and represents the absence of any electrical activity between these two events.
- The T wave represents repolarization of the ventricles.
- The QT interval represents the total time for depolarization and repolarization of the ventricles.

MECHANISMS OF DYSRHYTHMIAS
- Normally the main pacemaker of the heart is the SA node, which spontaneously discharges 60 to 100 times per minute. Disorders of impulse formation can cause dysrythmias.
- A pacemaker from another site can lead to dysrhythmias and may be discharged in a number of ways.
  - Secondary pacemakers may originate from the AV node or His-Purkinje system.
  - Secondary pacemakers can originate when they discharge more rapidly than the normal pacemaker of the SA node.
  - Triggered beats (early or late) may come from an ectopic focus (area outside the normal conduction pathway) in the atria, AV node, or ventricles.

EVALUATION OF DYSRHYTHMIAS
- Dysrhythmias result from various abnormalities and disease states, and the cause of a dysrhythmia influences the treatment.
- Several diagnostic tests are used to evaluate cardiac dysrhythmias and the effectiveness of antidysrhythmia drug therapy.
  - Holter monitoring records the ECG while the patient is ambulatory and performing daily activities.
  - Event monitors have improved the evaluation of outpatient dysrhythmias.
  - Signal-averaged ECG (SAECG) is a high-resolution ECG used to identify the patient at risk for developing complex ventricular dysrhythmias.
  - Exercise treadmill testing is used for evaluation of cardiac rhythm response to exercise.
  - An electrophysiologic study (EPS) identifies different mechanisms of tachydysrhythmias, heart blocks, bradydysrhythmias, and causes of syncope.

TYPES OF DYSRHYTHMIAS
- **Sinus bradycardia** has a normal sinus rhythm, but the SA node fires at a rate less than 60 beats/minute and is referred to as absolute bradycardia.
  - Clinical associations. Sinus bradycardia may be a normal sinus rhythm (e.g., in aerobically trained athletes), and it may occur in response to carotid sinus massage, Valsalva maneuver, hypothermia, and administration of parasympathomimetic drugs.
  - Disease states associated with sinus bradycardia are hypothyroidism, increased
intracranial pressure, obstructive jaundice, and inferior wall myocardial infarction (MI).

- Treatment consists of administration of atropine (an anticholinergic drug) for the patient with symptoms. Pacemaker therapy may be required.

- **Sinus tachycardia** has a normal sinus rhythm, but the SA node fires at a rate greater than 100 beats/minute as a result of vagal inhibition or sympathetic stimulation.
  - Clinical associations. Sinus tachycardia is associated with physiologic and psychologic stressors such as exercise, fever, pain, hypotension, hypovolemia, anemia, hypoxia, hypoglycemia, myocardial ischemia, heart failure (HF), hyperthyroidism, anxiety, and fear. It can also be an effect of certain drugs.
  - Angina may result from sinus tachycardia due to the increased myocardial oxygen consumption that is associated with an increased HR.
  - Treatment is based on the underlying cause. For example, if a patient is experiencing tachycardia from pain, tachycardia should resolve with effective pain management.

- **Premature atrial contraction** (PAC) is a contraction originating from an ectopic focus in the atrium in a location other than the sinus node. A PAC may be stopped (nonconducted PAC), delayed (lengthened PR interval), or conducted normally through the AV node.
  - Clinical associations. PACs can result from emotional stress or physical fatigue; from the use of caffeine, tobacco, or alcohol; from hypoxia or electrolyte imbalances; and from disease states such as hyperthyroidism, chronic obstructive pulmonary disease (COPD), and heart disease including coronary artery disease (CAD) and valvular disease.
  - In healthy persons, isolated PACs are not significant. In persons with heart disease, frequent PACs may indicate enhanced *automaticity* of the atria or a reentry mechanism and may warn of or initiate more serious dysrhythmias.
  - Treatment depends on the patient’s symptoms. For example, withdrawal of sources of stimulation such as caffeine or sympathomimetic drugs may be warranted.

- **Paroxysmal supraventricular tachycardia** (PSVT) is a dysrhythmia originating in an ectopic focus anywhere above the bifurcation of the bundle of His.
  - PSVT occurs because of a reentrant phenomenon (reexcitation of the atria when there is a one-way block) and is usually triggered by a PAC.
  - In the normal heart, PSVT is associated with overexertion, emotional stress, deep inspiration, and stimulants such as caffeine and tobacco. It is also associated with rheumatic heart disease, digitalis toxicity, CAD, and cor pulmonale.
  - Prolonged PSVT with HR greater than 180 beats/minute may precipitate a decreased CO, resulting in hypotension, dyspnea, and angina.
  - Treatment for PSVT includes vagal stimulation and drug therapy (i.e., IV adenosine).

- **Atrial flutter** is an atrial tachydysrhythmia identified by recurring, regular, sawtooth-shaped flutter waves that originate from a single ectopic focus in the right atrium.
  - Atrial flutter is associated with CAD, hypertension, mitral valve disorders, pulmonary embolus, chronic lung disease, cor pulmonale, cardiomyopathy, hyperthyroidism, and the use of drugs such as digoxin, quinidine, and epinephrine.
  - High ventricular rates (over 100/minute) and the loss of the atrial “kick” (atrial contraction reflected by a sinus P wave) can decrease CO and cause serious consequences such as chest pain and HF.
  - Patients with atrial flutter are at increased risk of stroke because of the risk of thrombus formation in the atria from the stasis of blood.
The primary goal in treatment of atrial flutter is to slow the ventricular response by increasing AV block.

- **Atrial fibrillation** is characterized by a total disorganization of atrial electrical activity due to multiple ectopic foci resulting in loss of effective atrial contraction.
  - Atrial fibrillation usually occurs in the patient with underlying heart disease, such as CAD, rheumatic heart disease, cardiomyopathy, hypertensive heart disease, HF, and pericarditis. It can be caused by thyrotoxicosis, alcohol intoxication, caffeine use, electrolyte disturbances, stress, and cardiac surgery.
  - Atrial fibrillation can often result in a decrease in CO, and thrombi may form in the atria as a result of blood stasis. An embolized clot may develop and pass to the brain, causing a stroke.
  - The goals of treatment include a decrease in ventricular response and prevention of cerebral embolic events.

- **Junctional dysrhythmias** refer to dysrhythmias that originate in the area of the AV node, primarily because the SA node has failed to fire or the signal has been blocked. In this situation, the AV node becomes the pacemaker of the heart.
  - Junctional premature beats are treated in a manner similar to that for PACs.
  - Other junctional dysrhythmias include junctional escape rhythm, accelerated junctional rhythm, and junctional tachycardia. These dysrhythmias are treated according to the patient’s tolerance of the rhythm and the patient’s clinical condition.
  - Junctional dysrhythmias are often associated with CAD, HF, cardiomyopathy, electrolyte imbalances, inferior MI, and rheumatic heart disease. Certain drugs (e.g., digoxin, amphetamines, caffeine, nicotine) can also cause junctional dysrhythmias.
  - Treatment varies according to the type of junctional dysrhythmia.

- **First-degree AV block** is a type of AV block in which every impulse is conducted to the ventricles but the duration of AV conduction is prolonged.
  - First-degree AV block is associated with MI, CAD, rheumatic fever, hyperthyroidism, vagal stimulation, and drugs such as digoxin, β-adrenergic blockers, calcium channel blockers, and flecainide.
  - First-degree AV block is usually not serious but can be a precursor of higher degrees of AV block. Patients with first-degree AV block are asymptomatic.
  - There is no treatment for first-degree AV block. Patients should continue to be monitored for any new changes in heart rhythm.

- **Second-degree AV block**, Type I (Mobitz I or Wenckebach heart block), is a gradual lengthening of the PR interval. It occurs because of a prolonged AV conduction time until an atrial impulse is nonconducted and a QRS complex is blocked (missing).
  - Type I AV block may result from use of drugs such as digoxin or β-adrenergic blockers. It may also be associated with CAD and other diseases that can slow AV conduction.
  - Type I AV block is usually a result of myocardial ischemia or infarction. It is almost always transient and is usually well tolerated. However, it may be a warning signal of a more serious AV conduction disturbance.
  - If the patient is symptomatic, atropine is used to increase HR, or a temporary pacemaker may be needed.

- **Second-degree AV block**, Type II (Mobitz II heart block), involves a P wave that is nonconducted without progressive antecedent PR lengthening. This almost always occurs...
when a block in one of the bundle branches is present.

- Type II second-degree AV block is a more serious type of block in which a certain number of impulses from the SA node are not conducted to the ventricles.
- Type II AV block is associated with rheumatic heart disease, CAD, anterior MI, and digitalis toxicity.
- Type II AV block often progresses to third-degree AV block and is associated with a poor prognosis. The reduced HR often results in decreased CO with subsequent hypotension and myocardial ischemia.
- Temporary treatment before the insertion of a permanent pacemaker may be necessary if the patient becomes symptomatic (e.g., hypotension, angina) and involves the use of a temporary transvenous or transcutaneous pacemaker.

- **Third-degree AV block**, or complete heart block, constitutes one form of AV dissociation in which no impulses from the atria are conducted to the ventricles.
  - Third-degree AV block is associated with severe heart disease, including CAD, MI, myocarditis, cardiomyopathy, and some systemic diseases such as amyloidosis and progressive systemic sclerosis (scleroderma).
  - Third-degree AV block almost always results in reduced CO with subsequent ischemia, HF, and shock. Syncope from third-degree AV block may result from severe bradycardia or even periods of asystole.
  - Treatment. For symptomatic patients, a transcutaneous pacemaker is used until a temporary transvenous pacemaker can be inserted.

- **Premature ventricular contraction** (PVC) is a contraction originating in an ectopic focus in the ventricles. It is the premature occurrence of a QRS complex, which is wide and distorted in shape compared with a QRS complex initiated from the normal conduction pathway.
  - PVCs are associated with stimulants such as caffeine, alcohol, nicotine, aminophylline, epinephrine, isoproterenol, and digoxin. They are also associated with electrolyte imbalances, hypoxia, fever, exercise, and emotional stress. Disease states associated with PVCs include MI, mitral valve prolapse, HF, and CAD.
  - PVCs are usually a benign finding in the patient with a normal heart. In heart disease, depending on frequency, PVCs may reduce the CO and precipitate angina and HF.
  - Treatment is often based on the cause of the PVCs (e.g., oxygen therapy for hypoxia, electrolyte replacement). Drugs that can be considered include β-adrenergic blockers, procainamide, amiodarone, or lidocaine (Xylocaine).

- **Ventricular tachycardia** (VT) is a run of three or more PVCs. It occurs when an ectopic focus or foci fire repetitively and the ventricle takes control as the pacemaker.
  - VT is a life-threatening dysrhythmia because of decreased CO and the possibility of deterioration to ventricular fibrillation, which is a lethal dysrhythmia.
  - VT is associated with MI, CAD, significant electrolyte imbalances, cardiomyopathy, mitral valve prolapse, long QT syndrome, digitalis toxicity, and central nervous system disorders.
  - VT can be stable (patient has a pulse) or unstable (patient is pulseless).
  - Treatment. Precipitating causes must be identified and treated (e.g., electrolyte imbalances, ischemia).

- **Ventricular fibrillation** (VF) is a severe derangement of the heart rhythm characterized on ECG by irregular undulations of varying shapes and amplitude. Mechanically the ventricle is simply “quivering,” and no effective contraction, and consequently no CO, occurs.
VF occurs in acute MI and myocardial ischemia and in chronic diseases such as CAD and cardiomyopathy.

- VF results in an unresponsive, pulseless, and apneic state. If not rapidly treated, the patient will die.
- Treatment consists of immediate initiation of CPR and advanced cardiac life support (ACLS) measures with the use of defibrillation and definitive drug therapy.

**Asystole** represents the total absence of ventricular electrical activity. No ventricular contraction occurs because depolarization does not occur.

- Asystole is usually a result of advanced cardiac disease, a severe cardiac conduction system disturbance, or end-stage HF.
- Patients are unresponsive, pulseless, and apneic.
- Asystole is a lethal dysrhythmia that requires immediate treatment consisting of CPR with initiation of ACLS measures (e.g., intubation, transcutaneous pacing, and IV therapy with epinephrine and atropine).

**Pulseless electrical activity** (PEA) describes a situation in which electrical activity can be observed on the ECG, but there is no mechanical activity of the ventricles and the patient has no pulse.

- Prognosis is poor unless the underlying cause can be identified and quickly corrected.
- Treatment begins with CPR followed by intubation and IV therapy with epinephrine.

**SUDDEN CARDIAC DEATH**

- **Sudden cardiac death** (SCD) refers to death from a cardiac cause.
- The majority of SCDs result from ventricular dysrhythmias, specifically ventricular tachycardia or fibrillation.

**PRODYRHYTHMIA**

- Antidysrhythmia drugs may cause life-threatening dysrhythmias similar to those for which they are administered. This concept is termed prodysrhythmia.
  - The patient who has severe left ventricular dysfunction is the most susceptible to prodysrhythmias.
  - Digoxin and some antidysrhythmia drugs can cause a prodysrhythmic response.

**DEFIBRILLATION**

- Defibrillation is the most effective method of terminating VF and pulseless VT.

- Defibrillation is accomplished by the passage of a DC electrical shock through the heart to depolarize the cells of the myocardium. The intent is that subsequent repolarization of myocardial cells will allow the SA node to resume the role of pacemaker.

- Rapid defibrillation can be performed using a manual or automatic device.
  - Manual defibrillators require health care providers to interpret cardiac rhythms, determine the need for a shock, and deliver a shock.
  - **Automatic external defibrillators** (AEDs) are defibrillators that have rhythm detection capability and the ability to advise the operator to deliver a shock using hands-free defibrillator pads.

**SYNCHRONIZED CARDIOVERSION**

- Synchronized cardioversion is the therapy of choice for the patient with hemodynamically unstable ventricular or supraventricular tachydysrhythmias.
A synchronized circuit in the defibrillator is used to deliver a countershock that is programmed to occur on the R wave of the QRS complex of the ECG.

- The synchronizer switch must be turned on when cardioversion is planned.

- The procedure for synchronized cardioversion is the same as for defibrillation, with some exceptions.

**IMPLANTABLE CARDIOVERTER-DEFIBRILLATOR (ICD)**

- The ICD is used for patients who (1) have survived SCD, (2) have spontaneous sustained VT, (3) have syncope with inducible ventricular tachycardia/fibrillation during EPS, and (4) are at high risk for future life-threatening dysrhythmias (e.g., have cardiomyopathy).

- The ICD consists of a lead system placed via a subclavian vein to the endocardium.

- A battery-powered pulse generator is implanted subcutaneously, usually over the pectoral muscle on the patient’s nondominant side.
  - The ICD sensing system monitors the HR and rhythm and identifies VT or VF.
    - Approximately 25 seconds after the sensing system detects a lethal dysrhythmia, the defibrillating mechanism delivers a shock to the patient’s heart.
    - If the first shock is unsuccessful, the generator recycles and can continue to deliver shocks.

- In addition to defibrillation capabilities, ICDs are equipped with antitachycardia and anti-bradycardia pacemakers.

- Education of the patient who is receiving an ICD is of extreme importance.

**PACEMAKERS**

- The artificial **cardiac pacemaker** is an electronic device used to pace the heart when the normal conduction pathway is damaged or diseased.

- Pacemakers were initially indicated for symptomatic bradydysrhythmias. They now provide antitachycardia and overdrive pacing.

- A permanent pacemaker is one that is implanted totally within the body.

- A specialized type of cardiac pacing has been developed for the management of HF.
  - Cardiac resynchronization therapy (CRT) is a pacing technique that resynchronizes the cardiac cycle by pacing both ventricles, thus promoting improvement in ventricular function.
  - Several devices are available that have combined CRT with an ICD for maximum therapy.

- A temporary pacemaker is one that has the power source outside the body. There are three types of temporary pacemakers: transvenous, epicardial, and transcutaneous pacemakers.

- Patients with temporary or permanent pacemakers will be ECG monitored to evaluate the status of the pacemaker.

- Complications of invasive temporary (i.e., transvenous) or permanent pacemaker insertion include infection and hematoma formation at the site of insertion of the pacemaker power source or leads, pneumothorax, failure to sense or capture with possible symptomatic
bradycardia, perforation of the atrial or ventricular septum by the pacing lead, and appearance of “end-of-life” battery parameters on testing the pacemaker.

**RADIOFREQUENCY CATHETER ABLATION THERAPY**

- Radiofrequency catheter ablation therapy is a relatively new development in the area of antidysrhythmia therapy. Ablation therapy is done after EPS has identified the source of the dysrhythmia.

- An electrode-tipped ablation catheter is used to “burn” or ablate accessory pathways or ectopic sites in the atria, AV node, and ventricles.

- Catheter ablation is considered the nonpharmacologic treatment of choice for AV nodal reentrant tachycardia or for reentrant tachycardia related to accessory bypass tracts, and to control the ventricular response of certain tachydyssrhythmias.

- The ablation procedure is a successful therapy with a low complication rate. Care of the patient following ablation therapy is similar to that of a patient undergoing cardiac catheterization.

**ECG CHANGES ASSOCIATED WITH ACUTE CORONARY SYNDROME**

- The 12-lead ECG is the primary diagnostic tool used to evaluate patients presenting with ACS.

- There are definitive ECG changes that occur in response to ischemia, injury, or infarction of myocardial cells and will be seen in the leads that face the area of involvement.

- Typical ECG changes seen in myocardial ischemia include ST-segment depression and/or T wave inversion.

- The typical ECG change seen during myocardial injury is ST-segment elevation.

- An ST-segment elevation and a pathologic Q wave may be seen on the ECG with myocardial infarction.

- Patient monitoring guidelines for patients with suspected ACS include continuous, multilead ECG and ST-segment monitoring. The leads selected for monitoring should minimally include the leads that reflect the area of ischemia, injury, or infarction.

**SYNCOPE**

- Syncope, a brief lapse in consciousness accompanied by a loss in postural tone (fainting), is a common diagnosis of patients coming into the emergency department.

- The causes of syncope can be categorized as cardiovascular or noncardiovascular.
  - Common cardiovascular causes of syncope include (1) neurocardiogenic syncope or “vasovagal” syncope (e.g., carotid sinus sensitivity) and (2) primary cardiac dysrhythmias (e.g., tachycardias, bradycardias).
  - Noncardiovascular causes can include hypoglycemia, hysteria, unwitnessed seizure, and vertebrobasilar transient ischemic attack.

- The diagnostic workup for a patient with syncope from a suspected cardiac cause begins with ruling out structural and/or ischemic heart disease.
- Echocardiography and stress testing are performed.
- In the older patient, who is more likely to have ischemic and structural heart disease, EPS is used to diagnose atrial and ventricular tachydysrhythmias, as well as conduction system disease causing bradydysrhythmias.
- In patients without structural heart disease or in whom EPS testing is not diagnostic, head-upright tilt table testing may be performed.
- Other diagnostic tests for syncope include various recording devices.
  - Holter monitors and event monitors can be used.
  - A subcutaneously implanted loop recording device can also be used to record the ECG during presyncopal and syncopal events.