Cadiovascular Disorders

community health nursing (Kenya Medical Training College)

CARDIOVASCULAR DISEASES

Signs and Symptoms Cardiovascular Disorders:

- Chest pain or discomfort (angina pectoris, MI, valvular heart disease)
- Shortness of breath or dyspnea (MI, left ventricular failure, HF)
- Edema and weight gain (right ventricular failure, HF)
- Palpitations (dysrhythmias resulting from myocardial ischemia, valvular heart disease, ventricular aneurysm, stress, electrolyte imbalance)
- Fatigue (earliest symptom associated with several cardiovascular disorders)
- Dizziness and syncope or loss of consciousness (postural hypotension, dysrhythmias, vasovagal effect, cerebrovascular disorders)

HEART'S CONDUCTION

- The heart's conducting system consists of the sinoatrial node (SA node), atrioventicular node (AV node), the bundle of His, the bundle branches and the Purkinje fibers.
- The electrical impulse that causes rhythmic contraction of heart muscles arises in the SA node which is the intrinsic pacemaker of the heart. From the SA node, the impulse spreads over the atrial muscles causing atrial contraction. The impulse is also conducted to the atrioventicular (AV) node.
- From the AV node the electrical impulse is conducted to ventricular muscles via the bundle of His, the bundle branches and the Purkinje fibers. The bundle branches and the Purkinje fibers are collectively called the **ventricular conduction system**.

THE S.A NODE

The SA node under the influence of the autonomic nervous system. The sympathetic system innervates the heart and causes increases the heart rate via B1 adrenergic receptors, for instance in fight or fright. The parasympathetic system, via the vagus nerve, slows the heart rate and establishes the resting heart rate of about 60-70 beats per minute. If parasympathetic actiivty is blocked by anti-cholinergic drugs or the vagal nerve is cut, the heart rate increases. If parasympathetic stimulation is increased, for instance by massaging the carotid sinus (baroreceptors), the heart rate decreases.

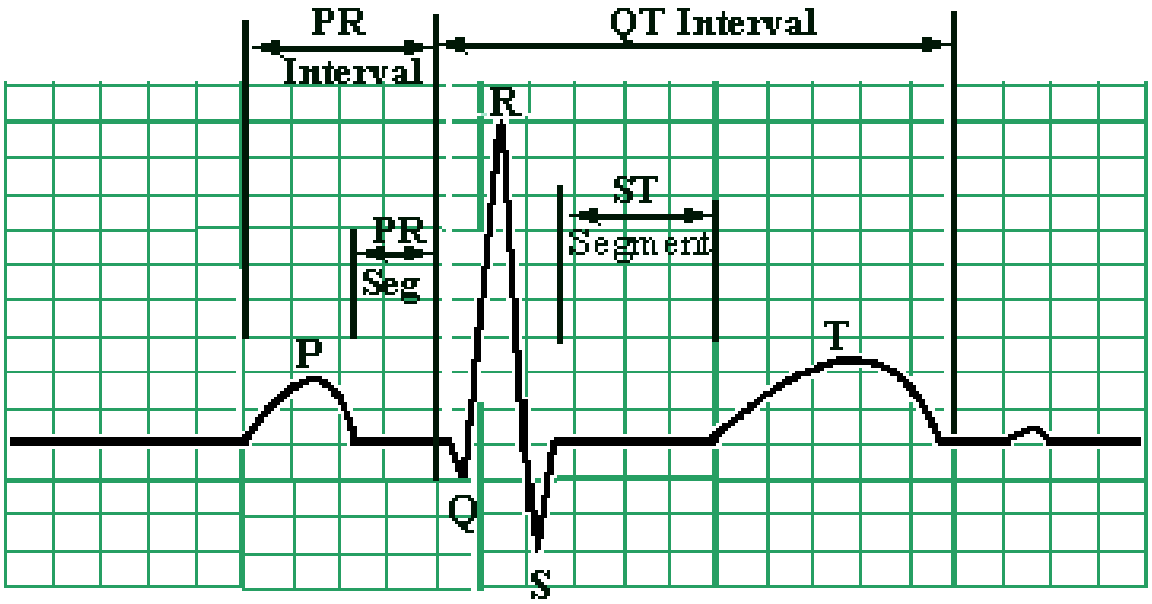
Why does the rhythm originate in the SA node?

The rhythm originates from the SA node because the SA node depolarizes more frequently (60-100 beats per minute) than the AV node (40-60 beats per minute) and ventricular conducting system (30-40 beats per minute) so the AV node and ventricular conducting system are ***'captured' by the sinus impulse and driven at 60-100 beast per minute.***

- ***The SA node spontaneously depolarize to initiate an action impulse that is rapidly propagated through the atria (causing atrial contract), then slowly through the AV***

node and rapidly via the bundle branches and Purkinje system to the ventricles, causing ventricular contraction.

- ***The electrical activity of the heart can be recorded at the surface of the body using an electrocardiogram.***
- ***The electrical impulse that travels through the heart can be viewed by means of electrocardiography, the end product of which is an electrocardiogram (ECG)***



- The **P wave** is caused by atrial depolarization. The P wave is usually smooth and positive. The P wave duration is normally less than 0.12 sec.
- The **PR interval** is the portion of the EKG/ECG wave from the beginning of the P wave (onset of atrial depolarization) to the beginning of the QRS complex (onset of ventricular depolarization). It is normally 0.12 - 0.20 seconds.
- The **PR segment** is the portion on the EKG wave from the end of the P wave to the beginning of the QRS complex. The PR segment corresponds to the time between the end of atrial depolarization to the onset of ventricular depolarization. It is an isoelectric segment, during which the impulse travels from the AV node through the conducting tissue (bundle branches, and Purkinje fibers) towards the ventricles.
- The **QRS complex** represents the time it takes for depolarization of the ventricles. - due to ventricular depolarization. The normal QRS interval range is from 0.04 sec - 0.12 sec measured from the first deflection to the end of the QRS complex.

- The **ST Segment** represents the period of ventricular muscle contraction before repolarization. The ST segment is normally isoelectric (no electrical activity is recorded). however, the ventricles are contracting.
- The **QT interval** begins at the onset of the QRS complex and to the end of the T wave. It represents the time of ventricular depolarization until venticular repolarization.
- The **T wave** due to ventricular repolarization. The wave is normally rounded and positive.

INTRODUCTION TO CARDIAC ARRYTHMIAS

Arrythmias/dysrhythmias are disorders of the formation or conduction (or both) of the electrical impulse within the heart. Most cardiac arrhythmias result from disorders of impulse formation, impulse conduction or a combination of both. Arrhythmias can be recognized by evaluating the EKG in a systematic manner, ie: determine rate; determine regularity of rhythm; identify P wave & its shape; identify QRS complex; determine r/ship btn P waves & QRS complexes.

It is important to remember to treat the patient not the EKG. Find out the Patient's history and use it in conjunction with the EKG.

The following EKG clues can be used to recognize cardiac arrythmias in non-sinus rhythm EKGs i.e Sinus bradycardia or **Sinus tachycardia**

1. Sinus Bradycardia (*brady* - slow)

- Sinus bradycardia occurs when the hearts rate is slower than 60 beats per minute.
- The sinus bradycardia rhythm is similar to normal sinus rhythm, except that the RR interval is longer. Each P wave is followed by a QRS complex in a ratio of 1:1. The PR interval is often slightly prolonged and occasionally, the P-waves might be abnormally wide.
- The symptoms of sinus bradycardia include dyspnea, dizziness, and extreme fatigue. Bradycardia may be accompanied by an increase in stroke volume due to greater end diastolic pressure (preload).

2. Sinus Tachycardia (*tachy* - fast)

- Sinus tachycardia occurs when the sinus rhythm is faster than 100 beats per minute.
- The rhythm is similar to normal sinus rhythm with the exception that the RR interval is shorter, less than 0.6 seconds. P waves are present and regular and each P-wave is followed by a QRS complex in a ratio of 1:1.
- Sinus tachycardia results from increased automaticity of the SA node, for instance, due to increased sympathetic stimulation of the heart, fever or cardiac toxicity.

If the rhythm is irregular, check for the following: Atrial flutter Or Atrial fibrillation.

3. Atrial Flutter

- Atrial flutter occurs when the atria are stimulated to contract at 200-350 beats per minute usually because electrical impulses are traveling in a circular fashion around and around the atria.
- Often the impulses are traveling around an obstacle like the mitral valve, tricuspid valve or the openings of the superior or inferior vena cavae.

- Atrial flutter is usually associated with mitral valve disease, pulmonary embolism, thoracic surgery, hypoxia, electrolyte disturbances and hypercalcaemia.

4. Atrial Fibrillation

- Atrial fibrillation occurs when the atria depolarize repeatedly and in an irregular uncontrolled manner usually at at atrial rate greater than 350 beats per minute. As a result, there is no concerted contraction of the atria.
- No P-waves are observed in the EKG due to the chaotic atrial depolarization. The chaotic atrial depolarization waves penetrate the AV node in an irregular manner, resulting in irregular ventricular contractions.
- The QRS complexes have normal shape, due to normal ventricular conduction. However the RR intervals vary from beat to beat. The ventricular rate may increase to greater than 150 beats per minute if uncontrolled.

If there are no P-waves, it could be an indication of either Atrial fibrillation. No P-waves will observable. Rather, a wavy base-line is recorded. OR Sinus arrest with junctional or ventricular escape. (Refer to Smeltzer, C.,Bare, B. 2004)

5. Ventricular Tachycardia (VT)

Ventricular tachycardia occurs when electrical impulses originating either from the ventricles cause rapid ventricular depolarization (140-250 beats per minute). Since the impulse originates from the ventricles, the QRS complexes are wide and bizarre. Ventricular impulses can be sometimes conducted backwards to the atria. in which case, P-waves may be inverted. Otherwise, regular normal P waves (60-100 beats per minute) may be present but not associated with QRS complexes (AV dissociation). The RR intervals are usually regular. Ventricular tachycardia is often due to some form of heart disease. Ventricular tachycardia can occur rarely in response to exercise or anxiety. In this case, the electrical impulses and rhythmic beats is similar is a normal beat but at a much faster rate.

6. Ventricular Fibrillation

Ventricular fibrillation occurs when parts of the ventricles depolarize repeatedly in an erratic, uncoordinated manner. The EKG in ventricular fibrillation shows random, apparently unrelated waves. Electrical defibrillation, by passage of current at high voltage, may be successful in restoration of a normal regular rhythm. The electrical current stimulates each myocardial cell to depolarize simultaneously. Following synchronous repolarization of all ventricular cells, the SA node assumes the role of pacemaker and the ventricular myocardial

cells can resume the essentially simultaneous depolarization of normal sinus rhythm. Ventricular fibrillation is associated with drug toxicity, electrocution, drowning and myocardial infarction.

7. Wolff-Parkinson-White Syndrome

- Normally, the AV node is the only conduction pathway for impulses from the atria to the ventricles. Wolff-Parkinson-White syndrome is characterized by the presence of an accessory atrioventicular pathway located between the wall of the right or left atria and the ventricles, known as the Bundle of Kent.
- Consists of episodes of of paroxysmal tachyarrythmia characterized by a shortened PR interval and widened QRS complex.
- Its caused by a faulty development of the AV ring of fibrous tissue. A conduction pathway called the bundle of Kent is formed.
- It is commonly associated with congenital heart abnormalities like Tetrology of Fallot, coarctation of the aorta, tricuspid atresia and transposition of the great vessels.
- In severe cases, treatment would involve surgical removal or ablation of one of the pathways.

NB: For Nursing Management Of Patients With Dyshythmias, Refer To: Smeltzer, C.,Bare, B. 2004 Page 698-705

CONDUCTION ABNORMALITIES

First-Degree Atrioventricular Block. First-degree heart block occurs when all the atrial impulses are conducted through the AV node into the ventricles at a rate slower than normal.
Second-Degree Atrioventricular Block, Type I. Second-degree, type I heart block occurs when all but one of the atrial impulses are conducted through the AV node into the ventricles. Each atrial impulse takes a longer time for conduction than the one before, until one impulse is fully blocked
Second-Degree Atrioventricular Block, Type II. Second-degree,type II heart block occurs when only some of the atrial impulses are conducted through the AV node into the ventricles
Third-Degree Atrioventricular Block. Third-degree heart block occurs when no atrial impulse is conducted through the AV node into the ventricles.

PACEMAKER THERAPY

A pacemaker is an electronic device that provides electrical stimuli to the heart muscle. Pacemakers are usually used when a patient has a slower-than-normal impulse formation or a conduction disturbance that causes symptoms.

CARDIOVERSION AND DEFIBRILLATION

Cardioversion and defibrillation are treatments for tachydysrhythmias. They are used to deliver an electrical current to depolarize a critical mass of myocardial cells.

NURSING ALERT

When performing defibrillation or cardioversion, the nurse should remember these key points:assignment!!!!

Cardioversion

Cardioversion involves the delivery of a "timed" electrical current to terminate a tachydysrhythmia. In cardioversion, the defibrillator is set to synchronize with the ECG on a cardiac monitor so that the electrical impulse discharges during ventricular depolarization (QRS complex

Defibrillation

Defibrillation is used in emergency situations as the treatment of choice for ventricular fibrillation and pulseless VT. Defibrillation depolarizes a critical mass of myocardial cells at once; when they repolarize, the sinus node usually recaptures its role as the pacemaker. The electrical voltage required to defibrillate the heart is usually greater than that required for cardioversion

CARDIAC CONDUCTION SURGERY

Atrial tachycardias and ventricular tachycardias that do not respond to medications and are not suitable for antitachycardia pacing may be treated by methods other than medications and devices.

Endocardial Isolation

Endocardial isolation involves making an incision into the endocardium that separates the area where the dysrhythmia originates from the surrounding endocardium.

Endocardial Resection

In endocardial resection, the origin of the dysrhythmia is identified, and that area of the endocardium is peeled away.

Catheter Ablation Therapy

Catheter ablation destroys specific cells that are the cause or central conduction method of a tachydysrhythmia.

CORONARY VASCULAR DISORDERS

CORONARY ARTERY DISEASE

Coronary artery disease (CAD) is the most prevalent type of cardiovascular disease. For this reason, it is important for nurses to become familiar with the various types of coronary artery conditions and the methods for assessing, preventing, and treating these disorders medically and surgically.

Coronary Atherosclerosis

- It is which is an abnormal accumulation of lipid, or fatty, substances and fibrous tissue in the vessel wall.
- The substances create blockages or narrow the vessel in a way that reduces blood flow to the myocardium.
- Studies (Mehta et al., 1998) indicate that atherosclerosis involves a repetitious inflammatory response to artery wall injury and an alteration in the biophysical and biochemical properties of the arterial walls.

Pathophysiology

Atherosclerosis begins as fatty streaks, lipids that are deposited in the intima of the arterial wall.The reason why some fatty streaks continue to develop is unknown, although genetic and environmental factors are involved. The continued development of atherosclerosis involves an inflammatory response. These deposits, called **atheromas** or plaques, protrude

into the lumen of the vessel, narrowing it and obstructing blood flow . The thrombus may obstruct blood flow, leading to sudden cardiac death or an acute **myocardial infarction (MI)**, which is the death of heart tissue.

Clinical Manifestations

- **Angina pectoris** refers to chest pain that is brought about by myocardial ischemia. Angina pectoris usually is caused by significant coronary atherosclerosis. dysfunction.
- Other clinical manifestations of CAD may be abnormalities signaled by changes on the electrocardiogram (ECG), high levels of cardiac enzymes,dysrhythmias, and sudden death.

Factors for Coronary Artery Disease

Nonmodifiable Risk Factors

- Family history of coronary heart disease
- Increasing age
- Gender (heart disease occurs three times more often in men than
- in premenopausal women)
- Race (higher incidence of heart disease in African Americans than
- in Caucasians)

Modifiable Risk Factors

- High blood cholesterol level
- Cigarette smoking, tobacco use
- Hypertension
- Diabetes mellitus
- Lack of estrogen in women
- Physical inactivity
- Obesity

ANGINA PECTORIS

Angina pectoris is a clinical syndrome usually characterized by episodes or paroxysms of pain or pressure in the anterior chest.

Pathophysiology

Angina is usually caused by atherosclerotic disease. Almost invariably, angina is associated with a significant obstruction of a major coronary artery. Several factors are associated with typical anginal pain:

- Physical exertion, which can precipitate an attack by increasing myocardial oxygen demand
- Exposure to cold, which can cause vasoconstriction and an elevated blood pressure, with increased oxygen demand
- Eating a heavy meal, which increases the blood flow to the mesenteric area for digestion, thereby reducing the blood supply available to the heart muscle heart, shunting of blood for digestion can be sufficient to induce anginal pain.)
- Stress or any emotion-provoking situation, causing the release of adrenaline and increasing blood pressure, which may accelerate the heart rate and increase the myocardial workload

Types of Angina

Stable angina: predictable and consistent pain that occurs on exertion and is relieved by rest

Unstable angina (also called preinfarction angina or crescendo angina): symptoms occur more frequently and last longer than stable angina. The threshold for pain is lower, and pain ma occur at rest.
Intractable or refractory angina: severe incapacitating chest pain
Variant angina (also called Prinzmetal's angina): pain at rest with reversible ST-segment elevation; thought to be caused by coronary artery vasospasm
Silent ischemia: objective evidence of ischemia (such as electrocardiographic changes with a stress test), but patient reports no symptoms

Assessment and Diagnostic Findings

- The diagnosis of angina is often made by evaluating the clinica manifestations of ischemia and the patient's history
- The patient may undergo an exercise or pharmacologic
- C-reactive protein (CRP) is a marker for inflammation of vascular endothelium.

Medical Management

- Revascularization procedures to restore the blood supply to the myocardium include **percutaneous coronary interventional (PCI)** procedures (eg, **percutaneous transluminal coronary angioplasty [PTCA]**, intracoronary stents, and atherectomy), CABG, and percutaneous transluminal myocardial revascularization (PTMR).

PHARMACOLOGIC THERAPY
Among medications used to control angina are nitroglycerin, beta-adrenergic blocking agents, calcium channel blockers, and antiplatelet agents.
Oxygen Administration. Oxygen therapy is usually initiated at the onset of chest pain in an attempt to increase the amount of oxygen delivered to the myocardium and to decrease pain.
Nursing Management of Patients With Coronary Vascular Disorders
NURSING DIAGNOSES
• Ineffective myocardial tissue perfusion secondary to CAD, as evidenced by chest pain or equivalent symptoms
• Anxiety related to fear of death
• Deficient knowledge about the underlying disease and methods for avoiding complications
• Noncompliance, ineffective management of therapeutic regimen related to failure to accept necessary lifestyle changes

MYOCARDIAL INFARCTION

Pathophysiology

- MI refers to the process by which areas of myocardial cells in the heart are permanently destroyed.
- A profound imbalance exists between myocardial oxygen supply and demand.
- As the cells are deprived of oxygen, ischemia develops, cellular injury occurs, and over time, the lack of oxygen results in infarction, or the death of cells.

Signs and Symptoms of an Acute Myocardial Infarction (MI) or Acute Coronary Syndrome (ACS)

Cardiovascular: Chest pain or discomfort, palpitations.
Respiratory: Shortness of breath, dyspnea, tachypnea
Gastrointestinal: Nausea and vomiting.

Genitourinary: Decreased urinary output may indicate cardiogenic shock.
Skin: Cool, clammy, diaphoretic, and pale
Neurologic: Anxiety, restlessness, light-headedness may indicate

Assessment and Diagnostic Findings

PATIENT HISTORY

The patient history has two parts: the description of the presenting symptom (eg, pain) and the history of previous illnesses and family health history, particularly of heart disease.

ELECTROCARDIOGRAM

LABORATORY TESTS

- **creatine kinase (CK)**,
- lactic dehydrogenase (LDH) levels.
- **Myoglobin.** Myoglobin is a heme protein that helps to transport oxygen.
- **Troponin**. **Troponin**, a protein found in the myocardium, regulates the myocardial contractile process.

Medical Management

PHARMACOLOGIC THERAPY

Thrombolytics. The purpose of thrombolytics is to dissolve and lyse the thrombus in a coronary artery (thrombolysis), allowing blood to flow through the coronary artery again (reperfusion), minimizing the size of the infarction, and preserving ventricular function.

- The thrombolytic agents used most often are **streptokinase** (Kabikinase, Streptase), alteplase (Activase), and reteplase (r-PA, TNKase

Angiotensin-Converting Enzyme Inhibitors (ACE-I

ACE inhibitors (ACE-I) prevent the conversion of angiotensin from I to II

EMERGENT PERCUTANEOUS CORONAR INTERVENTION (PCI)

The patient in whom an acute MI is suspected may be referred for an immediate PCI. PCI may be used to open the occluded coronary artery in an acute MI and promote reperfusion to the area that has been deprived of oxygen.

NURSING PROCESS: THE PATIENT WITH MYOCARDIAL INFARCTION

NURSING DIAGNOSES

• Ineffective cardiopulmonary tissue perfusion related to reduced coronary blood flow from coronary thrombus and atherosclerotic plaque
• Potential impaired gas exchange related to fluid overload from left ventricular dysfunction
• Potential altered peripheral tissue perfusion related to decreased cardiac output from left ventricular dysfunction
• Anxiety related to fear of death

STRUCTURAL, INFECTIOUS, AND INFLAMMATORY CARDIAC DISORDERS

A. Acquired Valvular Disorders

- The valves of the heart control the flow of blood through the heart into the pulmonary artery and aorta by opening and closing in response to the blood pressure changes as the heart contracts and relaxes through the cardiac cycle.
- The valves include **tricuspid valve, mitral valve, pulmonic valve , and aortic valve.**

- When valves do not close completely, blood flows backward through the valve in a process called **regurgitation**.
- When valves do not open completely, a condition called **stenosis**, the flow of blood through the valve is reduced.

MITRAL VALVE PROLAPSE

Mitral valve prolapse, formerly known as mitral prolapse syndrome, is a deformity that usually produces no symptoms.

Pathophysiology

- In mitral valve prolapse, a portion of a mitral valve leaflet balloons back into the atrium during systole.
- Blood then regurgitate from the left ventricle back into the left atrium

Clinical Manifestations

- fatigue, shortness of breath, light-headedness, dizziness, syncope, palpitations, chest pain, and anxiety

Medical Management

- Heart failure is treated the same as it would be for any other patient with heart failure
- In advanced stages of disease, mitral valve repair or replacement may be necessary.

MITRAL REGURGITATION

Mitral regurgitation involves blood flowing back from the left ventricle into the left atrium during systole. Often, the margins of the mitral valve cannot close during systole.

Pathophysiology

- Mitral regurgitation may be caused by problems with one or more of the leaflets, the chordae tendineae, the annulus, or the papillary muscles.
- The annulus may be stretched by heart enlargement or deformed by calcification.
- The papillary muscle may be unable to contract because of ischemia.
- With each beat of the left ventricle, some of the blood is forced back into the left atrium.

Clinical Manifestations

Chronic mitral regurgitation is often asymptomatic, but acute mitral regurgitation (eg, that resulting from a myocardial infarction) usually manifests as severe congestive heart failure.

Assessment and Diagnostic Findings

Echocardiography is used to diagnose and monitor the progression of mitral regurgitation.

Medical Management

- Management of mitral regurgitation is the same as that for congestive heart failure.
- Surgical intervention consists of mitral valve replacement or valvuloplasty (ie, surgical repair of the heart valve).

MITRAL STENOSIS

Mitral stenosis is an obstruction of blood flowing from the left atrium into the left ventricle. It is most often caused by rheumatic endocarditis, which progressively thickens the mitral valve leaflets and chordae tendineae. The leaflets often fuse together.

Clinical Manifestations

- Breathing difficulty (ie, dyspnea) on exertion as a result of pulmonary venous hypertension.

- Patients with mitral stenosis are likely to show progressive fatigue as a result of low cardiac output.
- They may expectorate blood (ie, hemoptysis), cough, and experience repeated respiratory infections.

Assessment and Diagnostic Findings

Echocardiography is used to diagnose mitral stenosis.

Medical Management

- Antibiotic prophylaxis therapy is instituted to prevent recurrence of infections.
- Surgical intervention consists of valvuloplasty, usually a commissurotomy to open or rupture the fused commissures of the mitral valve.

AORTIC REGURGITATION

Aortic regurgitation is the flow of blood back into the left ventricle from the aorta during diastole. This valvular defect also may result from endocarditis, congenital abnormalities, diseases such as syphilis, a dissecting aneurysm that causes dilation or tearing of the ascending aorta, or deterioration of an aortic valve replacement.

Clinical Manifestations

- Exertional dyspnea and fatigue
- orthopnea, paroxysmal nocturnal dyspnea

Assessment and Diagnostic Findings

Diagnosis may be confirmed by echocardiogram, radionuclide imaging, ECG, magnetic resonance imaging, and cardiac catheterization.

Medical Management

- Before the patient undergoes invasive antibiotic prophylaxis is needed to prevent endocarditis.
- Aortic valvuloplasty or valve replacement is the treatment of choice preferably performed before left ventricular failure.

AORTIC STENOSIS

Aortic valve stenosis is narrowing of the orifice between the left ventricle and the aorta. In adults, the stenosis may involve congenital leaflet malformations or an abnormal number of leaflets (ie, one or two rather than three), or it may result from rheumatic toms or changes in symptoms to the health care provider.

Valve Repair and Replacement Procedures

Homografts. **Homografts**, or **allografts** (ie, human valves), are obtained from cadaver tissue donations.

Autografts. Autografts (ie, autologous valves) are obtained by excising the patient's own pulmonic valve and a portion of the pulmonary artery for use as the aortic valve.

Cardiomyopathy is a heart muscle disease associated with cardiac dysfunction.

Infectious Diseases of the Heart

Among the most common infections of the heart are infective endocarditis, myocarditis, and pericarditis.

RHEUMATIC FEVER

- Rheumatic fever is an inflammatory disease that follows infection with certain strains of group A streptococci.
- Acute rheumatic fever (ARF) is a delayed, nonsuppurative sequela of a pharyngeal infection with the group A streptococcus (GAS).
- Following the initial pharyngitis, a latent period of two to three weeks occurs before the first signs or symptoms of ARF appear

Epidemiology

- Worldwide, there are 470,000 new cases of rheumatic fever and 233,000 deaths
- Most major outbreaks occur under conditions of impoverished overcrowding where access to antibiotics is limited.

Etiology

- Epidemiologic studies suggest an individual propensity to develop rheumatic fever, a nonsuppurative complication of group A streptococcal infection of the upper respiratory tract must occur, which occurs most commonly in children 5 to 15 years of age.
- *Streptococcus pyogenes* (β-hemolysis group A streptococci) cause both superficial infections (e.g. pharyngitis, impetigo) and invasive diseases.
- Environmental factors (latitude, altitude, humidity), nutrition, crowding, and age appear to influence the incidence of rheumatic fever, probably because the same factors influence the incidence of streptococcal infection.

Pathogenesis

- Role of the streptococcus:
  - Outbreaks of rheumatic fever closely follow epidemics of streptococcal pharyngitis or scarlet fever with associated pharyngitis
  - Adequate treatment of a documented streptococcal pharyngitis markedly reduces the incidence of subsequent rheumatic fever
  - Appropriate antimicrobial prophylaxis prevents the recurrence of disease in patients who have had ARF
  - Most patients with ARF have elevated antibody titers to at least one of (if not all) three antistreptococcal antibodies (streptolysin "O", hyaluronidase, and streptokinase), whether or not they recall an antecedent sore throat
- Importance of pharyngitis: Streptococcal pharyngitis has been the only streptococcal infection associated with ARF.
- RF may arise from GAS pyoderma or from pharyngitis due to non-group A streptococcal strains that inherited certain group A streptococcal antigens or enzymes that are important for initiating ARF
- Molecular mimicry: As a result of molecular mimicry, antibodies directed against GAS antigens crossreact with host antigens
- In addition to the role of antibody, observations suggest a role for cellular immunity in molecular mimicry in ARF.

- The earliest and most common feature is a painful migratory arthritis, which is present in approximately 80% of patients
- Carditis: Streptococcal M protein and N-acetyl-beta-D-glucosamine (NABG, the immunodominant carbohydrate antigen of GAS) share epitopes with myosin
- Chorea: Molecular mimicry may also be involved in the development of Sydenham chorea
- In Sydenham chorea, the antibodies bind to lysoganglioside on the neuronal cell surface, where they are capable of triggering a signaling cascade
- From a pathophysiologic viewpoint, the available data suggest involvement of the basal ganglia and cortical structures

Clinical features

- Pharyngitis usually occurs two to four weeks before the onset of ARF symptoms.
- The onset of the disease usually is characterized by an acute febrile illness that may manifest itself in one of several ways:
    1) Migratory arthritis predominantly involving the large joints
    2) Carditis and valvulitis
    3) Central nervous system involvement (e.g. Sydenham chorea)
    4) Rash
    5) Some combination of the above
- **Arthritis:** In the classic, untreated case, the arthritis of rheumatic fever affects several joints in quick succession, each for a short time
- **Carditis:** Rheumatic fever produces a pancarditis affecting the pericardium, epicardium, myocardium, and endocardium.
- **Chorea:** Sydenham chorea (SC), also known as St. Vitus dance, St. Johannis' chorea, chorea minor, and rheumatic chorea is a neurologic disorder consisting of abrupt, purposeless, nonrhythmic involuntary movements, muscular weakness, and emotional disturbances
- **Subcutaneous nodules:** These skin lesions in ARF have many identifying characteristics
- The nodules are firm and painless.
- **Erythema marginatum:** Erythema marginatum is an evanescent, non-pruritic rash, pink or faintly red, usually affecting the trunk and sometimes the proximal parts or the limbs, but not the face

Investigations

Laboratory Studies

- Group A streptococcal antigen detection tests are specific but not very sensitive
- Acute-phase reactants such as C-reactive protein and ESR are usually elevated and helpful in monitoring disease activity

Imaging Studies

- Echocardiography is more sensitive than standard auscultation for helping detect regurgitant lesions, but the prognostic significance of these subauscultory findings is unclear
- Chest radiograph may reveal cardiomegaly

- ECG is helpful for diagnosing carditis and may reveal a prolonged PR interval, but this finding is not necessarily associated with later cardiac sequelae

Diagnosis

Jones criteria

- If supported by evidence of a preceding group A streptococcal infection, the presence of **two major** manifestations or of **one major** and **two minor** manifestations is indicative of a high probability of acute rheumatic fever
- **Major manifestations:** The five major manifestations are:
  1. Carditis
  2. Polyarthritis
  3. Chorea
  4. Erythema marginatum
  5. Subcutaneous nodules
- **Minor manifestations:** The four minor manifestations are:
- Clinical findings
  1. Arthralgia
  2. Fever
- Laboratory findings
  1. Elevated acute phase reactants (erythrocyte sedimentation rate, C-reactive protein)
  2. Prolonged PR interval
- **Evidence of preceding streptococcal infection:** Any one of the following is considered adequate evidence of infection.
  1. Positive throat culture for group A beta-hemolytic streptococci or positive rapid streptococcal antigen test
  2. Elevated or rising streptococcal antibody titer, most often antistreptolysin O

Treatment

- Three major goals in the treatment of acute rheumatic fever are:
  1. Symptomatic relief of acute disease manifestations
  2. Eradication of the group A beta-hemolytic streptococcus
  3. Prophylaxis against future infection to prevent recurrent cardiac disease
- There is no therapy that slows progression of valvular damage in patients with ARF.

Symptomatic relief

- **Arthritis:** The mainstay of treatment for acute rheumatic fever remains antiinflammatory agents, most commonly aspirin
- Usually 80 to 100 mg/kg per day in children and 4 to 8 g/day in adults are required.
- **Carditis:** Severe carditis is marked by the presence of significant cardiomegaly, congestive heart failure, or third-degree heart block.

- Patients with severe carditis are often treated with corticosteroids, but studies of the effects of corticosteroids in the treatment of rheumatic carditis have shown conflicting results
- When corticosteroids are used, the usual dose is 2 mg/kg per day of oral prednisone for the first one to two weeks.
- Depending upon the clinical and laboratory response, the dose is then tapered over the next two weeks.
- Digoxin can be useful in patients with severe carditis, but its use should be monitored closely because of the possibility of heart block
- Valve surgery may be necessary when heart failure is due to regurgitant lesions that cannot be adequately managed with medical therapy
- **Sydenham chorea:** We recommend corticosteroids as the initial treatment: prednisone 1 mg/kg per day as a single dose for two weeks and then tapered over two to three weeks.
- Protracted Sydenham chorea has responded to haloperidol
- Chorea requires long-term antimicrobial prophylaxis, even if no other manifestations of rheumatic fever evolve

Antibiotic therapy

- Antibiotic therapy with penicillin should be started and maintained for at least 10 days, regardless of the presence or absence of pharyngitis at the time of diagnosis.
- The dose of oral penicillin V is that recommended for the eradication of streptococcal pharyngitis: 250 mg two to three times daily for children 500 mg two to three times daily for adults.
- A depot penicillin, such as benzathine penicillin G, in one single intramuscular dose should be given if compliance is an issue:
  - 600,000 units for children who weigh ≤ 27 kg
  - 1.2 million units for children who weigh >27 kg and adults
- Individuals who are allergic to penicillin can be treated with erythromycin 40 mg/kg per day (maximum dose of 1000 mg) divided into two to four doses.

Antibiotic prophylaxis

- The goal of prophylaxis against group A streptococcal infection is to prevent recurrence of ARF.
- Recurrence is most common within two years of the original attack but can happen at any time.
- Recurrence rates decrease with increasing age
- Acceptable oral regimens include:
  . Penicillin V potassium 400,000 units (250 mg) twice per day
  . Sulfadiazine 500 mg per day for children ≤ 27 kg and 1000 mg per day for children >27 kg and adults.
- The classic parenteral regimen is benzathine penicillin G 1.2 million units intramuscularly every four weeks
- Individuals who are allergic to penicillin and sulfadiazine can be treated with oral erythromycin 250 mg twice daily
- Duration of prophylaxis: The duration of prophylaxis is not well defined, and depends upon the number of previous attacks, the time lapsed since the last attack, the risk of exposure to streptococcal infections, the age of the patient, and the presence or absence of cardiac involvement

- WHO guidelines: The World Health Organization (WHO) published guidelines for secondary prevention of ARF in 2001
- The WHO recommendations for the duration of secondary prophylaxis are:
  . Patients without proven carditis — At least five years of antibiotic prophylaxis following diagnosis of ARF or until age 18 (whichever is longer)
  . Patients with mild mitral regurgitation — At least ten years of prophylaxis or until age 25 (whichever is longer)
  . Patients with severe valve disease and/or after valve surgery — Life-long prophylaxis

Rheumatic Heart Disease

- Rheumatic heart disease is the most serious complication of rheumatic fever.
- Acute rheumatic heart disease often produces a pancarditis characterized by endocarditis, myocarditis, and pericarditis.
- Endocarditis is manifested as valve insufficiency.
- The mitral valve is most commonly and the mitral valve alone is affected in 50-60% of cases, and combined lesions of the aortic and mitral valves occur in 20%; Tricuspid involvement occurs only in association with mitral or aortic disease in about 10% of cases.
- The pulmonary valve is rarely affected.
- Rheumatic mitral stenosis has a delayed onset compared to rheumatic mitral regurgitation (MR)
- Chronic rheumatic heart disease results from single or repeated attacks of rheumatic fever that produce rigidity and deformity of valve cusps, fusion of the commissures, or shortening and fusion of the chordae tendineae.
- The first clue to organic valvular disease is a murmur.
- The inflammatory process in the valve leaflets is thought to be initiated by cross-reactivity between streptococcal antigen and the valve tissue; there is no evidence for active infection of the valve leaflets
- Although the incidence of rheumatic heart disease is variable after an episode of acute rheumatic fever, approximately 50 percent of those with evidence of carditis develop organic valvular damage.
- In addition, up to 75 percent of patients with documented recurrences of rheumatic fever have some form of valvular disease after 45 years of follow-up
- Although rheumatic valve disease often results in MR in the first two decades of life, mitral stenosis (MS) and mixed MS/MR are more often seen in adults

RHEUMATIC ENDOCARDITIS

Acute rheumatic fever, which occurs most often in school-age children, follows 0.3% to 3% of cases of group A beta-hemolytic streptococcal pharyngitis (Chin, 2001). Prompt treatment of strep throat with antibiotics can prevent the development of rheumatic fever

Pathophysiology

- Leukocytes accumulate in the affected tissues and form nodules, which eventually are replaced by scar tissue.

- The myocardium is certain to be involved in this inflammatory process; rheumatic myocarditis develops, which temporarily weakens the contractile power of the heart.
- Rheumatic endocarditis, results in permanent and often crippling side effects.

Clinical Manifestations

- A few patients with rheumatic fever become critically ill with intractable heart failure, serious dysrhythmias, and pneumonia.

Prevention

Rheumatic endocarditis is prevented through early and adequate treatment of streptococcal infections.

Medical Management

Long-term antibiotic therapy is therecommended treatment, and penicillin administered parenterally remains the medication of choice.

Nursing Management

A key nursing role in rheumatic endocarditis is teaching patients about the disease, its treatment, and the preventive steps needed to avoid potential complications.

INFECTIVE ENDOCARDITIS

Infective endocarditis is an infection of the valves and endothelial surface of the heart.

.

Pathophysiology

- Infective endocarditis is most often caused by direct invasion of the endocardium by a microbe (eg, streptococci, enterococci, pneumococci, staphylococci).
- The infection usually causes deformity of the valve leaflets, but it may affect other cardiac structures such as the chordae tendineae.

Clinical Manifestations

The patient exhibits signs and symptoms similar to those described in rheumatic endocarditis

Assessment and Diagnostic Findings

- The general manifestations, which may be mistaken for influenza, include vague complaints of malaise, anorexia, weight loss, cough, and back and joint pain.
- Fever is intermittent and may be absent in patients who are receiving antibiotics or corticosteroids or in those who are elderly or have heart failure or renal failure.

Risk Factors for Infective Endocarditis

High Risk

- Prosthetic cardiac valves
- History of bacterial endocarditis (even without heart disease)
- Complex cyanotic congenital malformations
- Surgically constructed systemic or pulmonary shunts or conduits

Moderate Risk

- Mitral valve prolapse with valvular regurgitation or thickened
- leaflets
- Hypertrophic cardiomyopathy
- Acquired valvular dysfunction

Prevention

Antibiotic prophylaxis is recommended for highrisk patients immediately before and sometimes after the following
procedures:

Medical Management

PHARMACOLOGIC THERAPY

Antibiotic therapy is usually administered parenterally in a continuous intravenous infusion for 2 to 6 weeks.

SURGICAL MANAGEMENT

- Surgical valve replacement greatly improves the prognosis for patients with severe symptoms from damaged heart valves.
- Aortic or mitral valve excision and replacement are required for patients who develop congestive heart failure despite adequate medical treatment, patients who have more than one serious systemic embolic episode,and patients with uncontrolled infection, recurrent infection, or fungal endocarditis

MYOCARDITIS

Myocarditis is an inflammatory process involving the myocardium.

Pathophysiology

- Myocarditis usually results from a viral, bacterial, mycotic, parasitic, protozoal, or spirochetal infection.
- It also may occur in patient after acute systemic infections such as rheumatic fever, in those receiving immunosuppressive therapy, or in those with infective endocarditis.

Clinical Manifestations

- The patient may be asymptomatic, and the infection resolves on its own. The patient may develop mild to moderate symptoms and seek medical attention.
- The patient with mild to moderate symptoms often complains of fatigue and dyspnea, palpitations, and occasional discomfort in the chest and upper abdomen.

Prevention

Prevention of infectious diseases by means of appropriate immunizations (eg, influenza, hepatitis) and early treatment appears to be important in decreasing the incidence of myocarditis

Medical Management

- The patient receives specific treatment for the underlying cause if it is known (eg, penicillin for hemolytic streptococci) and is placed on bed rest to decrease the cardiac workload.
- Bed rest also helps to decrease myocardial damage and the complications of myocarditis.

Nursing Management

- The nurse assesses the patient's temperature to determine whether the disease is subsiding.
- Elastic compression stockings and passive and active exercises should be used, because embolization from venous thrombosis and mural thrombi can occur.

PERICARDITIS

Pericarditis refers to an inflammation of the pericardium, the membranous sac enveloping the heart.

Pathophysiology

• Idiopathic or nonspecific causes
• Infection: usually viral (eg, Coxsackie, influenza); rarely bacterial (eg, streptococci, staphylococci, meningococci, gonococci); and mycotic (fungal)
• Disorders of connective tissue: systemic lupus erythematosus, rheumatic fever, rheumatoid arthritis, polyarteritis
• Hypersensitivity states: immune reactions, medication reactions, serum sickness

• Disorders of adjacent structures: myocardial infarction, dissecting aneurysm, pleural and pulmonary disease (pneumonia)
• Neoplastic disease: caused by metastasis from lung cancer or breast cancer, leukemia, and primary (mesothelioma) neoplasms
• Radiation therapy
• Trauma: chest injury, cardiac surgery, cardiac catheterization, pacemaker implantation
• Renal failure and uremia
• Tuberculosis

Clinical Manifestations

- chest pain, although pain also may be located beneath the clavicle, in the neck, or in the left scapula region.
- Other signs may include mild fever, increased white blood cell count, and increased erythrocyte sedimentation rate (ESR).

Assessment and Diagnostic Findings

- patient's history,
- An echocardiogram may detect inflammation

Medical Management

- Analgesics and NSAIDs such as aspirin or ibuprofen may be prescribed for pain relief during the acute phase.
- Corticosteroids (eg, prednisone) may be prescribed if the pericarditis is severe or if the patient does not respond to NSAIDs
- Pericardiocentesis, a procedure in which some of the pericardial fluid is removed, may be performed to assist in the identification of the causative agent.
- Surgical removal of the tough encasing pericardium (pericardiectomy) may be necessary to release both ventricles from the constrictive and restrictive inflammation.

Nursing Management

- The nurse caring for the patient with pericarditis must be alert to the possibility of cardiac tamponade.
- Patients with acute pericarditis require pain management with analgesics, positioning, and psychological support.

HEART DISEASE

HEART FAILURE

HF, often referred to as **congestive heart failure (CHF)**, is the inability of the heart to pump sufficient blood to meet the needs of the tissues for oxygen and nutrients.

CHRONIC HEART FAILURE

As with coronary artery disease, the incidence of HF increases with age. However, the rate of coronary artery disease is decreasing and just the opposite is true for HF. There are two types of HF, which are identified by assessment of left ventricular functioning: an alteration in ventricular filling (**diastolic heart failure**) and an alteration in ventricular contraction(**systolic heart failure**).

Pathophysiology

HF results from a variety of cardiovascular diseases but leads to some common heart abnormalities that result in decreased contraction (systole), decreased filling (diastole), or

both.The sympathetic stimulation and the decrease in renal perfusion by the failing heart cause the release of renin by the kidney. Angiotensin-converting enzyme (ACE) in the lumen of blood vessels converts angiotensin I to angiotensin II, a vasoconstrictor that also causes the release of aldosterone. Aldosterone promotes sodium and fluid retention and stimulates the thirst center. Aldosterone causes additional detrimental effects to the myocardium and exacerbates myocardial fibrosis. Angiotensin, aldosterone, and other neurohormones (eg, atrial natriuretic factor, endothelin, andprostacyclin) lead to an increase in preload and afterload, which increases stress on the ventricular wall.

Etiology

- Coronary artery disease, cardiomyopathy, hypertension, or valvular disorders.
- Ischemia causes myocardial dysfunction because of resulting hypoxia and acidosis from the accumulation of lactic acid.
- Myocardial infarction causes focal heart muscle necrosis, the death of heart muscle cells, and a loss of contractility; the extent of the infarction correlates with the severity of HF.

New York Heart Association (NYHA)
Classification of Heart Failure

CLASSIFICATION	SYMPTOMS	PROGNOSIS
I	Ordinary physical activity does not cause undue fatigue, dyspnea, palpitations, or chest pain No pulmonary congestion or peripheral hypotension Patient is considered asymptomatic Usually no limitations of activities of daily living (ADLs)	Good
II	Slight limitation on ADLs Patient reports no symptoms at rest but increased physical activity will cause symptoms Basilar crackles and S3 murmur may be detected	Good
III	Marked limitation on ADL Patient feels comfortable at rest but less than ordinary activity will cause symptoms	Fair
IV	Symptoms of cardiac insufficiency at rest	Poor

Clinical Manifestations

LEFT-SIDED HEART FAILURE

- Pulmonary congestion occurs when the left ventricle cannot pump the blood out of the ventricle to the body.
- These effects of left ventricular failure have been referred to as *backward failure.*.

- Dyspnea, or shortness of breath, may be precipitated by minimal to moderate activity (**dyspnea on exertion [DOE]**); dyspnea also can occur at rest.
- O**rthopnea**, difficulty in breathing when lying flat
- Sudden attacks of orthopnea at night, a condition known as **paroxysmal nocturnal dyspnea (PND)**.
- Dry hacking cough that may be mislabeled as asthma or chronic obstructive pulmonary disease (COPD). Large quantities of frothy sputum, which is sometimes pink (blood tinged), may be produced, usually indicating severe pulmonary congestion (pulmonary edema).
- Blood flow to the kidneys decreases, causing decreased perfusion and reduced urine output (**oliguria**

RIGHT-SIDED HEART FAILURE

This occurs because the right side of the heart cannot eject blood and cannot accommodate all the blood that normally returns to it from the venous circulation. The increase in venous pressure leads to jugular vein distention (JVD). The clinical manifestations that ensue include

- edema of the lower extremities (dependent edema), hepatomegaly (enlargement of the liver), distended jugular veins, ascites (accumulation of fluid in the peritoneal cavity), weakness, anorexia and nausea, and paradoxically, weight gain due to retention of fluid.

.

Assessment and Diagnostic Findings

- An echocardiogram is usually performed to confirm the diagnosis of HF.
- Laboratory studies usually completed in the initial workup include serum electrolytes, blood urea nitrogen (BUN), creatinine, B-type natriuretic peptide (BNP), thyroid-stimulating hormone (TSH), a complete blood cell count (CBC), and routine rinalysis.

Medical Management

PHARMACOLOGIC THERAPY

Angiotensin-Converting Enzyme Inhibitors. ACE-Is stimulate the kidneys to excrete sodium and fluid (while retaining potassium), thereby reducing left ventricular filling pressure and decreasing pulmonary congestion.

Angiotensin II Receptor Blockers (ARBs). Although their action is different than that of ACE-Is, ARBs (eg, losartan [Cozaar]) have a similar hemodynamic effect as ACE-Is: lowered blood pressure and lowered systemic vascular resistance

Hydralazine and Isosorbide Dinitrate. Nitrates (eg, isosorbide dinitrate) cause venous dilation, which reduces the amount of blood return to the heart and lowers preload. Hydralazine lowers systemic vascular resistance and left ventricular afterload. It has also been shown to help avoid the development of nitrate tolerance.

Beta-Blockers. such as carvedilol (Coreg), metoprolol (Lopressor, Toprol), or bisoprolol (Zebeta), have been found to reduce mortality and morbidity

Diuretics. Diuretics are medications used to increase the rate of urine production and the removal of excess extracellular fluid from the body.

Digitalis. The most commonly prescribed form of digitalis for patients with HF is digoxin (Lanoxin). The medication increases the force of myocardial contraction and slows conduction through the AV node. It improves contractility, increasing left ventricular output. The medication also enhances diuresis, which removes fluid and relieves edema.

A key concern associated with digitalis therapy is digitalis toxicity.
Calcium Channel Blockers. Eg. verapamil , nifedipine (Adalat), cause vasodilation, reducing systemicvascular resistance.
NUTRITIONAL THERAPY
A low-sodium (≤2 to 3 g/day) diet and avoidance of excessive amounts of fluid are usually recommended.

Nursing Management
• Keeping an intake and output record to identify a negative balance (more output than input)
• Weighing the patient daily at the same time and on the same scale
• Auscultating lung sounds at least daily to detect an increase or decrease in pulmonary crackles
• Determining the degree of JVD
• Identifying and evaluating the severity of dependent edema
• Monitoring pulse rate and blood pressure, as well as monitoring for postural hypotension and making sure that the patient does not become hypotensive from dehydration
• Examining skin turgor and mucous membranes for signs of dehydration
• Assessing symptoms of fluid overload (eg, orthopnea, paroxysmal nocturnal dyspnea, and dyspnea on exertion) and evaluating changes

Digoxin Use and Toxicity in Heart Failure

Preparations
Digoxin
• Tablets: 0.125, 0.25, 0.5 mg (Lanoxin)
• Capsules: 0.05, 0.1, 0.2 mg (Lanoxicaps)
• Elixir: 0.05 mg/mL (Lanoxin Pediatric elixir)
• Injection: 0.25 mg/mL, 0.1 mg/mL (Lanoxin)

Digoxin Toxicity
A serious complication of digoxin therapy is toxicity. Diagnosis of digoxin toxicity is based on the patient's clinical symptoms, which include the following:
• Fatigue, depression, malaise, anorexia, nausea, and vomiting (early effects of digitalis toxicity)
• Changes in heart rhythm: new onset of regular rhythm or new onset of irregular rhythm
• ECG changes indicating SA or AV block; new onset of irregular rhythm indicating ventricular dysrhythmias; and atrial tachycardia with block, junctional tachycardia, and ventricular tachycardia

Reversal of Toxicity
- If the toxicity is severe, digoxin immune FAB (Digibind) may be prescribed.
- Digibind binds with digoxin and makes it unavailable for use.

Nursing Considerations and Actions
NB: Before administering digoxin, it is standard nursing practice to assess apical heart rate. When the patient's rhythm is atrial fibrillation and the heart rate is less than 60, or the rhythm becomes regular, the nurse may withhold the medication and notify the physician, because these signs indicate the development of AV conduction block. Although withholding digoxin is a common practice, the medication does not need to be withheld for a heart rate of less than 60 if the patient is in sinus rhythm because digoxin does not affect sinoatrial node automaticity.

NURSING PROCESS: THE PATIENT WITH HEART FAILURE

NURSING DIAGNOSES

Based on the assessment data, major nursing diagnoses for the patient with HF may include the following:

• Activity intolerance (or risk for activity intolerance) related to imbalance between oxygen supply and demand because of decreased CO
• Excess fluid volume related to excess fluid or sodium intake and retention of fluid because of HF and its medical therapy
• Anxiety related to breathlessness and restlessness from inadequate oxygenation
• Powerlessness related to inability to perform role responsibilities because of chronic illness and hospitalizations
• Noncompliance related to lack of knowledge

POTENTIAL COMPLICATIONS

• Cardiogenic shock
• Dysrhythmias
• Thromboembolism
• Pericardial effusion and cardiac tamponade

ACUTE HEART FAILURE (PULMONARY EDEMA)

Pulmonary edema is the abnormal accumulation of fluid in the lungs. The fluid may accumulate in the interstitial spaces or in the alveoli.

Pathophysiology

Pulmonary edema is an acute event that results from HF. It can occur acutely, such as with myocardial infarction, or it can occur as an exacerbation of chronic HF. With increased resistance to left ventricular filling, the blood backs up into the pulmonary circulation.

Clinical Manifestations

- Sudden onset of breathlessness and a sense of suffocation
- patient's hands become cold and moist, the nail beds become cyanotic (bluish), and the skin turns ashen (gray).
- The pulse is weak and rapid, and the neck veins are distended

Assessment and Diagnostic Findings

Most often, a chest x-ray is obtained to confirm that the pulmonary veins are engorged.

Medical Management

PHARMACOLOGIC THERAPY

Various treatments and medications are prescribed for pulmonary edema, among them oxygen, morphine, diuretics, and various intravenous medications.

Oxygen Therapy. Oxygen is administered in concentrations adequate to relieve hypoxemia and dyspnea.

Morphine. Morphine is administered intravenously in small doses(2 to 5 mg) to reduce peripheral resistance and venous return so that blood can be redistributed from the pulmonary circulation to other parts of the body.

Diuretics. Diuretics promote the excretion of sodium and water by the kidneys. Furosemide (Lasix), for example, is administered intravenously to produce a rapid diuretic effect.

Dobutamine(Dobutrex)**.** A catecholamine, dobutamine stimulates the beta1-adrenergic receptors. Its major action is to increase cardiac contractility.

Milrinone. Milrinone (Primacor) is a phosphodiesterase inhibitor that delays the release of calcium from intracellular reservoirs and prevents the uptake of extracellular calcium by the cells. This promotes vasodilation.

Nursing Management

POSITIONING THE PATIENT TO PROMOTE CIRCULATION: The patient is positioned upright, preferably with the legs dangling over the side of the bed. This has the immediate effect of decreasing venous return, lowering the output of the right ventricle, and decreasing lung congestion.

PROVIDING PSYCHOLOGICAL SUPPORT

CARDIOGENIC SHOCK

Cardiogenic shock occurs when the heart cannot pump enough blood to supply the amount of oxygen needed by the tissues.

Pathophysiology

- The degree of shock is proportional to the extent of left ventricular dysfunction.
- The heart muscle loses its contractile power, resulting in a marked reduction in SV and CO, which is sometimes called forward failure.
- The damage to the myocardium results in a decrease in CO, which reduces arterial blood pressure and tissue perfusion in the vital organs (heart, brain, lung, kidneys).

Clinical Manifestations

- Cerebral hypoxia (restlessness, confusion, agitation)
- low blood pressure, rapid and weak pulse, cold and clammy skin, increased respiratory crackles,
- hypoactive bowel sounds, and decreased urinary output.

Assessment and Diagnostic Findings

- Use of a PA catheter to measure left ventricular pressures and CO is important in assessing the severity of the problem and planning management.
- Arterial blood gas analysis shows metabolic acidosis, and all laboratory test results indicate organ dysfunction

Medical Management

PHARMACOLOGIC THERAPY

Vasopressors, or pressor agents, are medications used to raise blood pressure and increase CO.eg catecholamines, such as norepinephrine (Levophed) and dopamine (Intropin

OTHER TREATMENTS

Other therapeutic modalities for cardiogenic shock include use of circulatory assist devices. The most frequently used mechanical support device is the intra-aortic balloon pump (IABP).

Nursing Management

- The patient in cardiogenic shock requires constant monitoring and intensive care.
- The critical care (intensive care) nurse must carefully assess the patient, observe the cardiac rhythm, monitor hemodynamic parameters, and record fluid intake and urinary output

THROMBOEMBOLISM

- The decreased mobility of the patient with cardiac disease and the impaired circulation that accompany these disorders contribute to the development of intracardiac and intravascular thrombosis.

- Intracardiac thrombus is especially common in patients with atrial fibrillation, because the atria do not contract forcefully and blood flow slows through the atrium, increasing thrombus formation.
- Intracardiac thrombus is detected by an echocardiogram and treated with anticoagulants, such as heparin and warfarin (Coumadin).
- A part of the thrombus may become detached (embolus) and may be carried to the brain, kidneys, intestines, or lungs.
- The symptoms of pulmonary embolism include chest pain, cyanosis, shortness of breath, rapid respirations, and hemoptysis (bloody sputum).

PERICARDIAL EFFUSION AND CARDIAC TAMPONADE

Pathophysiology

Pericardial effusion refers to the accumulation of fluid in the pericardial sac. This occurrence may accompany pericarditis, advanced HF, metastatic carcinoma, cardiac surgery, trauma, or nontraumatic hemorrhage.

A rapidly developing effusion, however, can stretch the pericardium to its maximum size and, because of increased pericardial pressure, reduce venous return to the heart and decrease CO. The result is cardiac tamponade (compression of the heart).

Clinical Manifestations

- Venous pressure tends to rise, as evidenced by engorged neck veins
- Systolic blood pressure that is detected during exhalation but not heard with inhalation is called **pulsus paradoxus**. Pulsus paradoxus exceeding 10 mm Hg is abnormal.
- The cardinal signs of cardiac tamponade are falling systolic blood pressure, narrowing pulse pressure, rising venous pressure (increased jugular venous distention), and distant (muffled) heart sounds

Assessment and Diagnostic Findings

An echocardiogram may be performed to confirm the diagnosis.

Medical Management

PERICARDIOCENTESIS : puncture of the pericardial sac to aspirate pericardial fluid)

Complications of pericardiocentesis include ventricular or coronary artery puncture, dysrhythmias, pleural laceration, gastric puncture, and myocardial trauma.

PERICARDIOTOMY: pericardial window

.

CARDIAC ARREST

Cardiac arrest occurs when the heart ceases to produce an effective pulse and blood circulation. It may be caused by a cardiac electrical event, as when the HR is too fast (especially ventricular tachycardia or ventricular fibrillation) or too slow (bradycardia or AV block) or when there is no heart rate at all (asystole).

Clinical Manifestations

- Consciousness, pulse, and blood pressure are lost immediately.
- Ineffective respiratory gasping may occur.

- The pupils of the eyes begin dilating within 45 seconds.

Emergency Management: Cardiopulmonary Resuscitation

The ABCDs of basic cardiopulmonary resuscitation (CPR) are *a*irway, *b*reathing, *c*irculation, and *d*efibrillation

Resuscitation consists of the following steps:

1. Airway: maintaining an open airway
2. Breathing: providing artificial ventilation by rescue breathing
3. Circulation: promoting artificial circulation by external cardiac compression
4. Defibrillation: restoring the heartbeat

The chest compression rate is 80 to 100 times per minute. If only one rescuer is available, the rate is two ventilations to every 15 cardiac compressions. When two rescuers are available, the first person performs the cardiac compressions, pausing after the fifth compression, when the second rescuer gives one ventilation over 1.5 to 2 seconds and at a tidal volume of less than 1 L.

Medications Used in Cardiopulmonary Resuscitation

VASCULAR DISORDERS AND PROBLEMS OF PERIPHERAL CIRCULATION

ARTERIOSCLEROSIS AND ATHEROSCLEROSIS

- **Arteriosclerosis** is the most common disease of the arteries; the term means *hardening of the arteries.*
- It is a diffuse process whereby the muscle fibers and the endothelial lining of the walls of small arteries and arterioles become thickened.
- **Atherosclerosis** involves a different process, affecting the intima of the large and mediumsized arteries.
- These changes consist of the accumulation of lipids, calcium, blood components, carbohydrates, and fibrous tissue on the intimal layer of the artery. These accumulations are referred to as atheromas or plaques.

Risk Factors

- age or gender
- genetic factors
- Tobacco use may be one of the strongest risk factors in the development of atherosclerotic lesions. Nicotine decreases blood flow to the extremities and increases heart rate and blood pressure by stimulating the sympathetic nervous system, causing vasoconstriction.
- Many other factors such as obesity, stress, and lack of exercise have been identified as

Medical Management

SURGICAL MANAGEMENT

Vascular surgical procedures **angioplasty**, also called percutaneous transluminal angioplasty (PTA)

PERIPHERAL ARTERIAL OCCLUSIVE DISEASE

Arterial insufficiency of the extremities is usually found in individuals older than 50 years of age, most often in men. The legs are most frequently affected; however, the upper extremities may be involved.

Clinical Manifestations

The hallmark is intermittent claudication. This pain may be described as aching, cramping, fatigue, or weakness that is consistently reproduced with the same degree of exercise or activity and relieved with rest.

Assessment and Diagnostic Findings

- When the extremity is examined, it may feel cool tothe touch and look pale when elevated or ruddy and cyanotic when placed in a dependent position.
- Skin and nail changes, ulcerations, gangrene, and muscle atrophy may be evident.
- The diagnosis of peripheral arterial occlusive disease may be made using CW Doppler and ankle-brachial indices (ABIs), treadmill testing for claudication, duplex ultrasonography

Medical Management

PHARMACOLOGIC THERAPY

- Pentoxifylline (Trental) increases erythrocyte flexibility and reduces blood viscosity, and it is therefore thought to improve the supply of oxygenated blood to the muscle.
- Cilostazol (Pletal) works by inhibiting platelet aggregation, inhibiting smooth muscle cell proliferation, and increasing vasodilation.
- Antiplatelet aggregating agents such as aspirin, ticlopidine (Ticlid), and clopidogrel (Plavix) are thought to improve circulation throughout diseased arteries or prevent intimal hyperplasia leading to stenosis.

SURGICAL MANAGEMENT

Bypass grafts are performed to reroute the blood flow around the stenosis or occlusion.

THROMBOANGIITIS OBLITERANS (BUERGER'S DISEASE)

- Buerger's disease is characterized by recurring inflammation of the intermediate and small arteries and veins of the lower and (in rare cases) upper extremities. It results in thrombus formation and occlusion of the vessels.
- It is differentiated from other vessel diseases by its microscopic appearance.
- In contrast to atherosclerosis, Buerger's disease is believed to be an autoimmune disease that results in occlusion of distal vessels. The cause of Buerger's disease is unknown, but it is believed to be an autoimmune vasculitis. It occurs most often in men between the ages of 20 and 35 years, and it has been reported in all races and in many areas of the world.

Clinical Manifestations

- Pain is the outstanding symptom of Buerger's disease
- Cold sensitivity of the Raynaud type is found in one half the patients and is frequently confined to the hands.

Assessment and Diagnostic Findings

- Duplex ultrasonography is used to document patency of the proximal vessels and to visualize the extent of distal disease.
- Contrast angiography is performed to demonstrate the diseased portion of the anatomy.

Management

The treatment of Buerger's disease is essentially the same as that for atherosclerotic peripheral arterial disease.

AORTITIS

- Aortitis is inflammation of the aorta, particularly of the aortic arch. Two types are known to occur: Takayasu's disease and syphilitic aortitis.
- Takayasu's disease, or occlusive thromboaortopathy, is uncommon; today, syphilitic aortitis is rare.

AORTIC ANEURYSM

- An aneurysm is a localized sac or dilation formed at a weak point in the wall of the aorta.
- The most common forms of aneurysms are saccular or fusiform.
- A saccular aneurysm projects from one side of the vessel only.
- If an entire arterial segment becomes dilated, a fusiform aneurysm develops.

THORACIC AORTIC ANEURYSM

- Approximately 85% of all cases of thoracic aortic aneurysm are caused by atherosclerosis.
- They occur most frequently in men between the ages 40 and 70 years.

Clinical Manifestations

Symptoms are variable and depend on how rapidly the aneurysm dilates and how the pulsating mass affects surrounding intrathoracic structures.

Assessment and Diagnostic Findings

Diagnosis of a thoracic aortic aneurysm is principally made by chest x-ray, transesophageal echocardiography, and CT.

Medical Management

- In most cases, an aneurysm is treated by surgical repair.
- The goal of surgery is to repair the aneurysm and restore vascular continuity with a vascular graft

.

ABDOMINAL AORTIC ANEURYSM

- The most common cause of abdominal aortic aneurysm is atherosclerosis.
- Untreated, the eventual outcome may be rupture and death.

Pathophysiology

- All aneurysms involve a damaged media layer of the vessel.
- This may be caused by congenital weakness, trauma, or disease.
- After an aneurysm develops, it tends to enlarge.
- Risk factors include genetic predisposition, smoking (or other tobacco use), and hypertension; more than one half of patients with aneurysms have hypertension.

Clinical Manifestations

- Some patients complain that they can feel their heart beating in their abdomen when lying down, or they may say they feel an abdominal mass or abdominal throbbing.

.

Medical Management

.

SURGICAL MANAGEMENT

The standard treatment for abdominal aortic aneurysm repair has
been open surgical repair of the aneurysm by resecting the vessel and sewing a bypass graft in place.

Nursing Management

Before surgery, nursing assessment is guided by anticipating a rupture and by recognizing that the patient may have cardiovascular, cerebral, pulmonary, and renal impairment from atherosclerosis.

.

DISSECTING AORTA

Occasionally, in an aorta diseased by arteriosclerosis, a tear develops in the intima or the media degenerates, resulting in a dissection

Pathophysiology

- Arterial dissections (separations) are commonly associated with poorly controlled hypertension .
- Dissection is caused by rupture in the intimal layer.
- As the separation progresses, the arteries branching from the involved area of the aorta shear and occlude.
- The tear occurs most commonly in the region of the aortic arch

Clinical Manifestations

- Onset of symptoms is usually sudden. Severe and persistent pain, described as tearing or ripping, may be reported.
- The pain is in the anterior chest or back and extends to shoulders, epigastric area, or abdomen.

.

Assessment and Diagnostic Findings

Arteriography, CT, transesophageal echocardiography, duplex ultrasonography, and magnetic resonance imaging aid in the diagnosis.

Medical Management

Medical or surgical treatment of a dissecting aneurysm depends on the type of dissection present and follows the general principles outlined for the treatment of thoracic aortic aneurysms.

Nursing Management

A patient with a dissecting aorta requires the same nursing care as a patient with an aortic aneurysm requiring surgical intervention.

RAYNAUD'S DISEASE

- Raynaud's disease is a form of intermittent arteriolar vasoconstriction that results in coldness, pain, and pallor of the fingertips or toes.
- The cause is unknown, although many patients with the disease seem to have immunologic disorders.
- Symptoms may re sult from a defect in basal heat production that eventually decreases the ability of cutaneous vessels to dilate.
- Episodes may be triggered by emotional factors or by unusual sensitivity to cold.

- The disease is most common in women between 16 and 40 years of age, and it occurs more frequently in cold climates and during the winter.
- The term *Raynaud's phenomenon* is used to refer to localized, intermittent episodes of vasoconstriction of small arteries of the feet and hands that cause color and temperature changes.

Clinical Manifestations

The classic clinical picture reveals pallor brought on by sudden vasoconstriction.

Medical Management

- Avoiding the particular stimuli (eg, cold, tobacco) that provoke vasoconstriction is a primary factor in controlling Raynaud's disease.
- Calcium channel blockers may be effective in relieving symptoms

Nursing Management

- The nurse teaches patients to avoid situations that may be stressful or unsafe.
- Stress management classes may be helpful.
- Exposure to cold must be minimized, and in areas where the fall and winter months are cold, the patient should remain indoors asmuch as possible .

VENOUS THROMBOSIS, DEEP VEIN THROMBOSIS (DVT), THROMBOPHLEBITIS, AND PHLEBOTHROMBOSIS

Although the terms *venous thrombosis, deep vein thrombosis (DVT), thrombophlebitis,* and *phlebothrombosis* do not necessarily reflect identical disease processes, for clinical purposes, they are often used interchangeably.

Pathophysiology

- Deep and superficial veins have valves that permit unidirectional flow back to the heart.
- The valves lie at the base of a segment of the vein that is expanded into a sinus.
- This arrangement permits the valves to open without coming into contact with the wall of the vein, permitting rapid closure when the blood starts to flow backward.

Virchow's triad of DVT

- stasis of blood (venous stasis)
- vessel wall injury
- and altered blood coagulation

Clinical Manifestations

- The entire extremity becomes massively swollen, tense, painful, and cool to the touch.
- Homans' sign (pain in the calf after the foot is sharply dorsiflexed) is not specific for deep vein thrombosis because it can be elicited in any painful condition of the calf.
- Thrombosis of superficial veins produces pain or tenderness, redness, and warmth in the involved area.

Prevention

- Preventive measures include the application of elastic compression stockings, the use of intermittent pneumatic compression devices, and special body positioning and exercise
- A further method to prevent venous thrombosis in surgical patients is administration of subcutaneous unfractionated or low molecular weight heparin.

Medical Management

Anticoagulation Therapy

Thrombolytic Therapy.

Surgical Management:A thrombectomy (removal of the thrombosis) is the procedure of choice.

Nursing Management

If the patient is receiving anticoagulant therapy, the nurse must frequently monitor the partial thromboplastin time, prothrombin time, hemoglobin and hematocrit values, platelet count, and fibrinogen level

VARICOSE VEINS

- Varicose veins (varicosities) are abnormally dilated, tortuous superficial veins caused by incompetent venous valves.
- Most commonly, this condition occurs in the lower extremities, the saphenous veins, or the lower trunk; however, it can occur elsewhere in the body, such as esophageal varices

Pathophysiology

- Varicose veins may be considered primary (without involvement of deep veins) or secondary (resulting from obstruction of deep veins).
- A reflux of venous blood in the veins results in venous stasis

Clinical Manifestations

- of dull aches, muscle cramps, and increased muscle fatigue in the lower legs.
- Ankle edema and a feeling of heaviness of the legs may occur.
- Nocturnal cramps

Assessment and Diagnostic Findings

Diagnostic tests for varicose veins include the duplex scan

Prevention

The patient should avoid activities that cause venous stasis, such as

- wearing tight socks or a constricting panty girdle, crossing the legs at the thighs, and sitting or standing for long periods.

Medical Management

.

SCLEROTHERAPY

In sclerotherapy, a chemical is injected into the vein, irritating the venous endothelium and producing localized phlebitis and fibrosis, thereby obliterating the lumen of the vein.

Nursing Management

- Bed rest is maintained for 24 hours
- Elastic compression stockings are used to maintain compression of the leg.
- The nurse assists the patient to perform exercises and move the legs.
- The foot of the bed should be elevated.

LYMPHANGITIS AND LYMPHADENITIS

- Lymphangitis is an acute inflammation of the lymphatic channels.
- Usually, the infectious organism is a hemolytic *Streptococcus.*
- The characteristic red streaks that extend up the arm or the leg from an infected wound outline the course of the lymphatic vessels as they drain.
- The lymph nodes located along the course of the lymphatic channels also become enlarged, red, and tender (acute lymphadenitis).
- They can also become necrotic and form an abscess (suppurative lymphadenitis)

LYMPHEDEMA AND ELEPHANTIASIS

- Lymphedemas are classified as primary (congenital malformations) or secondary (acquired obstructions).
- Tissue swelling occurs in the extremities because of an increased quantity of lymph that results from obstruction of lymphatic vessels.
- Lymphatic obstruction caused by a parasite (filaria) is seen frequently in the tropics.
- When chronic swelling is present, there may be frequent bouts of acute infection characterized by high fever and chills and increased residual edema after the inflammation has resolved.
- This condition, in which chronic swelling of the extremity recedes only slightly with elevation, is referred to as elephantiasis.

Medical Management

- External compression devices milk the fluid proximally from the foot to the hip or from the hand to the axilla.
- PHARMACOLOGIC THERAPY: As initial therapy, the diuretic furosemide (Lasix) is prescribed as needed to prevent the fluid overload that can result from the mobilization of extracellular fluid.
- SURGICAL MANAGEMENT: excision of the affected subcutaneous tissue and fascia, with skin grafting to cover the defect OR surgical relocation of superficial lymphatic vessels into the deep lymphatic system by means of a buried dermal flap to provide a conduit for lymphatic drainage.

Nursing Management

- Prophylactic antibiotics may be prescribed for 5 to 7 days.
- Constant elevation of the affected extremity and observations for complications are essential.
- Complications may include flap necrosis, hematoma or abscess under the flap, and cellulitis.

HYPERTENSION

Hypertension is a systolic blood pressure greater than 140 mm Hg and a diastolic pressure greater than 90 mm Hg over a sustained period, based on the average of two or more blood pressure measurements taken in two or more contacts with the health care provider after an initial screening.

Primary Hypertension

- Between 90% and 95% have **primary hypertension**, meaning that the reason for the elevation in blood pressure cannot be identified.

The remaining 5% to 10% of this group have high blood pressure related to specific causes, such as narrowing of the renal arteries, renal parenchymal disease, hyperaldosteronism (mineralocorticoid hypertension) certain medications, pregnancy, and coarctation of the aorta (Kaplan, 2001).

- **Secondary hypertension** is the term used to signify high blood pressure from an identified cause.
- Prolonged blood pressure elevation eventually damages blood vessels throughout the body, particularly in target organs such as the heart, kidneys, brain, and eyes.
- The usual consequences of prolonged, uncontrolled hypertension are myocardial infarction, heart failure, renal failure, strokes, and impaired vision.

Pathophysiology

Hypertension may be caused by one or more of the following:

- Increased sympathetic nervous system activity related to dysfunction of the autonomic nervous system
- Increased renal reabsorption of sodium, chloride, and water related to a genetic variation in the pathways by which the kidneys handle sodium
- Increased activity of the renin-angiotensin-aldosterone system, resulting in expansion of extracellular fluid volume and increased systemic vascular resistance
- Decreased vasodilation of the arterioles related to dysfunction of the vascular endothelium
- Resistance to insulin action, which may be a common factor linking hypertension, type 2 diabetes mellitus, hypertriglyceridemia, obesity, and glucose intolerance

Clinical Manifestations

- High blood pressure.
- Retinal changes such as hemorrhages, exudates (fluid accumulation), arteriolar narrowing, and cottonwool spots (small infarctions) occur
- Pathologic changes in the kidneys (indicated by increased blood urea nitrogen [BUN] and creatinine levels) may manifest as nocturia.
- stroke or transient ischemic attack (TIA), manifested by alterations in vision or speech, dizziness, weakness, a sudden fall, or temporary paralysis on one side (hemiplegia).

Assessment and Diagnostic Evaluation

- Routine laboratory tests include urinalysis, blood chemistry (ie, analysis of sodium, potassium, creatinine, fasting glucose, and total and high-density lipoprotein [HDL] cholesterol levels), and a 12-lead electrocardiogram.
- Renal damage may be suggested by elevations in BUN and creatinine levels or by microalbuminuria or macroalbuminuria.
- Additional studies, such as creatinine clearance, renin level, urine tests, and 24-hour urine protein, may be performed.

Medical Management

The goal of hypertension treatment is to prevent death and complications by achieving and maintaining the arterial blood pressure at 140/90 mm Hg or lower.
Dietary Approaches to Stop Hypertension (DASH) diet.

The DASH (Dietary Approaches to Stop Hypertension) Diet*

FOOD GROUP	NO. SERVINGS PER DAY
Grains and grain products	7–8
Vegetables	4–5
Fruits	4–5
Lowfat or fat-free dairy foods	2–3
Meat, fish, and poultry	2 or fewer
Nuts, seeds, and dry beans	4–5 weekly

PHARMACOLOGIC THERAPY

- the recommended initial medications include diuretics, beta-blockers, or both.

NURSING PROCESS: THE PATIENT WITH HYPERTENSION

NURSING DIAGNOSES

Based on the assessment data, nursing diagnoses for the patient may include the following:
• Deficient knowledge regarding the relation between the treatment regimen and control of the disease process
• Noncompliance with therapeutic regimen related to side effects of prescribed therapyare needed to determine the effectiveness of medication therapy and to detect any changes in blood pressure that indicate the need for a change in the treatment plan.
****Medication Therapy for Hypertension****

HYPERTENSIVE CRISES

A) Hypertensive Emergency

- **Hypertensive emergency** is a situation in which blood pressure must be lowered mmediately (not necessarily to less than 140/90 mm Hg) to halt or prevent damage to the target organs.
- Conditions associated with hypertensive emergency include acute myocardial infarction, dissecting aortic aneurysm, and intracranial hemorrhage.
- The medications of choice in hypertensive emergencies are those that have an immediate effect.
- Intravenous vasodilators, including sodium nitroprusside (Nipride, Nitropress), nicardipine hydrochloride (Cardene), fenoldopam mesylate (Corlopam), enalaprilat (Vasotec I.V.), and nitroglycerin (Nitro-Bid IV, Tridil), have an immediate action that is short lived (minutes to 4 hours), and they are therefore used as the initial treatment.

HYPERTENSIVE URGENCY

- **Hypertensive urgency** is a situation in which blood pressure must be lowered within a few hours.
- Severe perioperative hypertension is considered a hypertensive urgency.
- Hypertensive urgencies are managed with oral doses of fast-acting agents such as loop diuretics (bumetanide [Bumex], furosemide [Lasix]), beta-blockers propranolol (Inderal), metoprolol (Lopressor), nadolol (Corgard), angiotensin-converting enzyme inhibitors (benazepril [Lotensin], captopril [Capoten], enalapril [Vasotec]), calcium antagonists (diltiazem[Cardizem], verapamil [Isoptin SR, Calan SR, Covera HS]), or alpha2-agonists, such as clonidine (Catapres) and guanfacine

✓ Extremely close hemodynamic monitoring of the patient's blood pressure and cardiovascular status is required during treatment of hypertensive emergencies and urgencies.
✓ The nurse may think that taking vital signs every 5 minutes is appropriate if the blood pressure is changing rapidly or may check vital signs at 15 or 30 minutes intervals if the situation is more stable.