CARDIOVASCULAR NURSING

THE HEART

- Hollow, muscular organ
- Weight approximately 300 g
- It occupies the space between the lungs (mediastinum) and rests on the diaphragm
- The heart pumps blood to the tissues supplying them with oxygen and other nutrients.

THREE LAYERS OF THE HEART

- Endocardium-lines the inside of the heart and valves
- Myocardium-made up of muscle fibers and is responsible for the contraction
- Epicardium- Exterior layer of the heart in which pericardium can be found
  - ✓ Outermost
  - ✓ Essential coronary arteries are located

Pericardium- thin layer of fibrous tissue that contains pericardial fluid that lubricates the lining of the heart, it consists of two layers:

- Adhering to the epicardium is the **visceral pericardium.**
- Enveloping the visceral pericardium is the **parietal pericardium**, which supports the heart in the mediastinum.

"The pumping action of the heart is accomplished by the rhythmic relaxation and contraction"

Systole- refers to the events in the heart during, contraction of the two top chambers (atria) and two lower chambers (ventricles)

Diastolic- is characterized by relaxation of the lower chambers which allows the ventricles to fill in preparation for contraction

2 CHAMBERS

UPPER

- **ATRIUM**
- Collecting/ Receiving chamber

LOWER

- **VENTRICLES**
- Pumping/ Contracting chamber

Apical impulse (also called **the point of maximal impulse [PMI])** located at the 15th intercostal space (ICS), left mid-clavicular line.

MECHANICAL PROPERTIES OF THE HEART

CARDIAC OUTPUT

- Volume of blood (liters) ejected by the heart each minute 5 L/min
- During exercise the total cardiac output may increase fourfold, to 2 L/min.
- **Cardiac Output = Heart Rate x Stroke Volume**
  - CO = HR x SV

PULSE RATE/ HEART RATE

- Number of times the ventricles contract each minute
- 60-100 beats/min
- Controlled by the ANS

STROKE VOLUME

- Volume of blood ejected by the left ventricle during each systole
- Affected by 3 factors:
  - Preload
  - Contractility
  - Afterload

PRELOAD
- ❖ Degree of myocardial stretch at the end of diastole & just before contraction
- ❖ Determined by the amount of blood returning to the heart from venous & pulmonary system

STARLING'S LAW
- ❖ The more the heart is filled during diastole, the more forcefully it contracts
- ❖ The higher the preload, the higher the stroke volume**.**

CONTRACTILITY
- ❖ Force generated by the contracting enhanced by myocardium
- ❖ Catecholamines, sympathetic activity and with medications such as the 3 D's
  - Digoxin, Dopamine, Dobutamine
- ❖ The higher the contractility, the higher the stroke volume.

AFTERLOAD
- ❖ Pressure or resistance that the ventricles must overcome to eject blood through the semi-lunar valves
- ❖ Directly proportional to the BP & Diameter of blood vessels
- ❖ The higher the afterload, the lower the stroke volume.

HEART SOUNDS

1. The first heart sound (S1) is heard as the atrioventricular valves close and is heard loudest at the apex of the heart.
2. The second heart sound (S2) is heard when the semilunar valves close and is heard loudest at the base of the heart.
3. A third heart sound (S3) may be heard if ventricular wall compliance is decreased and structures in the ventricular wall vibrate heart; this can occur in conditions such as congestive heart failure or valvular regurgitation. However, a third heart sound may be normal in individuals younger than 30 years.
4. A fourth heart sound (S4) may be heard on atrial systole if resistance to ventricular filling the is present; this is an abnormal finding, and causes include cardiac hypertrophy, disease, or injury to the ventricular wall.

CARDIAC ELECTROPHYSIOLOGY

Automaticity: ability to initiate an electrical impulse by itself
Excitability: ability to respond to an electrical impulse
Conductivity: ability to transmit an electrical impulse from one cell to another

SINOATRIAL (SA) NODE
- ❖ Location: Junction of Superior vena cava & Right Atrium
- ❖ Function: Pacemaker of heart
- ❖ Initiates 60-100 bpm

ATRIOVENTRICULAR (AV) NODE
- ❖ Location: Interatrial septum
- ❖ Delays the electric impulse to allow ventricular filling of 0.8 milliseconds
- ❖ 40-60 beats/min

BUNDLE OF HIS
- ❖ Location: Interventricular septum
- ❖ Branches out into:
  - Right main Bundle Branch
  - Left main Bundle Branch

PURKINJE FIBERS
- ❖ Location: Walls of ventricles
- ❖ Ventricular contractions
- ❖ Fastest conduction is: 20 - 40 beats/min
- ❖ It can function as a backup pacemaker if all other pacemakers fail

FACTS:
- ❖ "The parasympathetic impulses, which travel to the heart through the Vagus nerve, can slow the cardiac rate, whereas sympathetic impulses increase it."

- Baroreceptors are specialized nerve cells located in the aortic arch and in both right and left internal carotid arteries. The baroreceptors are sensitive to changes in blood pressure.

- Hypotension can result in less baroreceptor stimulation, which prompts a decrease in parasympathetic inhibitory activity in the SA node, allowing for enhanced sympathetic activity. The resultant vasoconstriction and increased heart rate elevate the blood pressure.

ELECTRICAL CONDUCTION THROUGH THE HEART

P WAVE

- The P wave represents atrial muscle depolarization. It is normally small, smoothly rounded, and no wider than 0.12 second

QRS COMPLEX

- The QRS complex represents ventricular muscle depolarization
- Normal QRS width is 0.04 to 0.10 second.
- Atrial repolarization happens simultaneously.

T WAVE

- The T wave represents ventricular repolarization
- T waves are not normal more than 5 mm

PR INTERVAL

- The PR interval is measured from the beginning of the P wave to the beginning of the QRS complex and represents the time required for the impulse to travel through atria, AV junction, and Purkinje system. The normal PR interval is 0.12 to 0.20 seconds.

QT INTERVAL

- It represents the total time for ventricular depolarization and repolarization.
- QT interval is usually 0.32 to 0.40
- If QT interval becomes prolonged, the patient may be at risk for a lethal ventricular dysrhythmia called torsades de pointes.

PP INTERVAL

- The duration between the beginning of one P wave and the beginning of the next P wave
- Used to calculate atrial rate and rhythm

RR INTERVAL

- The duration between the beginning of one QRS complex and the beginning of the next QRS complex; used to calculate ventricular rate and rhythm

U WAVE

- The part of an ECG that may reflect Purkinje fiber repolarization: usually it is not seen unless a patient's serum potassium level is low (Hypokalemia)

CORONARY ARTERY DISEASE

- Coronary artery disease (CAD) is the most prevalent type of cardiovascular disease in adults.
- Most common cause of cardiovascular disease is atherosclerosis- (abnormal accumulation of fats)

CLINICAL MANIFESTATIONS

- Symptoms and complications according to the location and degree of narrowing of the arterial lumen, if impediment to the blood flow has occurred, inadequate supply to cardiac cells will lead to a condition known as ischemia.

MODIFIABLE RISK FACTORS

- Hyperlipidemia
- Cigarette smoking, tobacco use
- Hypertension
- Diabetes mellitus

- Metabolic syndrome
- Obesity
- Physical inactivity

NON-MODIFIABLE RISK FACTORS

- Family history' of CAD (first-degree relative with cardiovascular disease at 55 years of age or younger for men and at 65 years of age or younger for women)
  - ✓ Increasing age '4 More than 45 years for men
  - ✓ More than 55 years for women
- Gender (men develop CAD at an earlier age than women)
- Race (higher incidence of heart disease in African Americans than in Caucasians)

CLINICAL MANIFESTATION

- Possibly normal asymptomatic periods
- Chest pain
- Palpitations
- Dyspnea
- Syncope
- Excessive fatigue

SURGICAL PROCEDURES

- ❖ PTCA to compress the plaque against the walls of the artery and dilate the vessel
- ❖ Laser angioplasty to vaporize the plaque
- ❖ Atherectomy to remove the plaque from the artery
- ❖ Vascular stent to prevent the artery from closing and to prevent restenosis
- ❖ Coronary Artery Bypass Grafting (CABG) to improve blood flow to the myocardial tissue at risk for ischemia or infarction because of the occluded artery

MEDICATIONS

- ❖ Nitrates to dilate the coronary arteries and decrease preload and afterload
- ❖ Calcium channel blockers to dilate coronary arteries and reduce vasospasm
- ❖ Cholesterol-lowering medications to reduce the development of atherosclerotic plaques
- ❖ Beta-Blockers to reduce the BP in individuals who are hypertensive

*All adults 20 years of age or older should have a fasting lipid profile (total cholesterol, LDL, HDL, and triglyceride I performed at least once every 5 years and more often if the profile is abnormal"

*HDL, (high density lipoprotein) is known as good cholesterol because it transports other lipoproteins such as LDL to the liver, where they can be degraded and excreted. Because of this, a high HDL level is a strong protective factor for heart disease.

*Mediterranean diet another diet that promotes the ingestion of vegetables and fish and restricts red meat, is also reported to reduce mortality from cardiovascular disease"

*Cholesterol is present in all body tissues and is a major component of low-density lipoproteins, brain and nerve cells, cell membranes, and some gallbladder stones

*Increased cholesterol levels, LDL (Low density lipoprotein) levels, and triglyceride levels place the client at risk for coronary artery disease

INSTRUCT THE CLIENT REGARDING DIET COMPOSED OF:

- ❖ Low-calorie
- ❖ Low-sodium
- ❖ Low-cholesterol
- ❖ Low-fat diet
- ❖ Increase in dietary fiber

VALUES

- Cholesterol: 140 to 199 mg/dL
- Low-density lipoproteins: Lower than 130 mg/dL
- High-density lipoproteins: 30 to 70 mg/dL
- Triglycerides: Lower than 200 mg/dL

HEART FAILURE (HF)

- HF is the inability of the heart to pump enough blood to meet the needs of tissues for oxygen and nutrients.
- Decreased heart contractility/ Pump failure
- Inadequacy of the heart to pump blood throughout the body
- Insufficient perfusion of body tissues (decreased cardiac output)

COMMON CAUSES OF HEART FAILURE

- Hypertension
- CAD
- Cardiomyopathy
- Substance abuse (Alcohol, Cocaine, Amphetamines)
- Valvular disease
- History of myocardial infarction
- Congenital defects
- Cardiac infections & inflammations
- Hyperkinetic conditions

ACC/AHA CLASSIFICATION OF HEART FAILURE

STAGE A	Patients at high risk for Developing left ventricular Dysfunction but without structural heart disease or symptoms of heart failure
STAGE B	Patients with left ventricular dysfunction or structural heart disease who have not developed symptoms of heart failure
STAGE C	Patients with left ventricular dysfunction or structural heart disease with current or prior symptoms of heart failure
STAGE D	Patients with refractory end-stage heart failure requiring specialized interventions

TWO MAJOR TYPES OF HEART FAILURE

- Systolic heart failure- alteration in ventricular contraction which is characterized by weakened heart muscle
- Diastolic heart failure- characterized by a stiff and non- compliant heart muscle making it difficult for the ventricle to fill. The signs and symptoms of HF can be related to which ventricle is affected.

LEFT-SIDED HEART FAILURE

- Pulmonary venous blood volume and pressure increase, forcing fluid from the pulmonary capillaries into the pulmonary, tissues and alveoli, causing pulmonary„' interstitial edema and impaired Bas exchange.
- Pulmonary congestion occurs
- Signs and symptoms: Pulmonary/Lung (Left=Lung)
  - Dyspnea, cough, pulmonary crackles/rales, and low oxygen saturation levels.
  - Orthopnea, difficulty breathing when lying flat.
  - Frothy, pink (blood-tinged) sputum: pulmonary congestion (pulmonary edema)
- An extra heart sound, the S3, or "ventricular gallop," may he detected on auscultation.
- The dominant feature in HF is inadequate tissue perfusion

Compensatory Mechanisms

- Compensatory mechanisms act to restore cardiac output to near-normal levels.
  - Sympathetic nervous system stimulation
  - ✓ Arterial vasoconstriction
  - ✓ Increases afterload
  - ✓ Increased left cardiac workload
  - ✓ Increased heart rate
  - ✓ Improved stroke volume
  - ✓ Arterial vasoconstriction

- Renin-angiotensin system activation

❖ A decrease in renal perfusion due to low cardiac output causes the release of renin by the kidneys.

❖ Renin promotes the formation of Angiotensin I, a benign, inactive substance.

❖ Angiotensin- converting enzyme (ACE) in the lumen of pulmonary blood vessels converts angiotensin I to angiotensin II a potent vasoconstrictor, which then increase blood pressure and afterload.

❖ Angiotensin II also stimulates the release of aldosterone from the adrenal cortex, resulting in sodium and fluid retention by the renal tubules and stimulation of antidiuretic hormone. These mechanisms lead to the fluid volume overload commonly seen in HF.

COMMON NURSING DIAGNOSES

- Impaired gas exchange related to ventilation perfusion imbalance
- Decreased CO related to altered contractility, preload & afterload
- Activity intolerance related to an imbalance between 02 supply and demand
- Potential for pulmonary edema, pneumonia, dysrhythmias

MANAGEMENT

- Patients with orthopnea usually prefer not to lie flat. They may need pillows to prop themselves up in bed, or they may sit in a chair and even sleep sitting up.
- Monitor vital signs and look for changes.
- Record fluid intake and output—weigh daily to assess for fluid overload.
- Position patient in semi-Fowler's position to oxygen as ordered because it ease breathing
- Administer oxygen as ordered because it helps to decrease workload of heart.
- Administer diuretic as prescribed.
- Tell the patient:
  ✓Eat foods low in sodium to avoid fluid retention.

RIGHT-SIDED HEART FAILURE

❖ Right side of the heart cannot eject blood and cannot accommodate all the blood that normally returns to it from the venous circulation
❖ Increased venous pressure leads to Jugular vein distention and increased capillary hydrostatic pressure throughout the venous system
❖ Edema of the lower extremities (dependent system edema)
❖ Hepatomegaly (enlargement of the liver)
❖ Ascites (accumulation of fluid in the peritoneal al cavity)
❖ Weight gain due to retention of fluid.

"Inability of the right heart to empty its blood volume results in blood backing up into the systemic circulation. LV failure is the most common cause of right ventricular (RV) failure. Sustained pulmonary hypertension also causes RV failure".

NURSING INTERVENTIONS

- Monitor heart rate and for dysrhythmias by using a cardiac monitor.
- Assess for edema in dependent areas and in the sacral, lumbar, and posterior thigh regions in the client on the bed rest.
- Avoid the unnecessary IV administration of fluids.
- Monitor weight to determine a response to treatment.
- Assess for hepatomegaly and ascites, and measure and record abdominal girth.

PHARMACOLOGIC MANAGEMENT FOR HF

Administer diuretics for symptom control resulting in patient comfort by reducing blood volume	Furosemide, bumetanide, metolazone, hydrochlorothiazide, spironolactone-be aware of electrolyte imbalance-these medications may alter the K+ level
Administer ACE inhibitors to decrease afterload	Captopril, enalapril, lisinopril
Administer beta blocker, which help to raise ejection fraction, and decrease ventricular size	Metoprolol (Lopressor, Toprol) Atenolol (Tenormin) Carvedilol (Coreg)
Administer inotrope to strengthen myocardial contractility	Digoxin
Administer vasodilator to reduce preload, relieve dyspnea	Nitroprusside, Nitroglycerin ointment

ARTERIOSCLEROSIS

- Thickening or hardening of the arterial wall

ATHEROSCLEROSIS

- Type of arteriosclerosis where a fatty plaque as formed within the arterial wall
- Leading contributor of CAD (coronary artery disease) and CVA (cerebrovascular accident)

VALVULAR HEART DISEASE

- Valvular heart disease occurs when the heart valves cannot fully open (stenosis) or closes inwards causing a leak (insufficiency or regurgitation).

TYPES:

- Mitral Stenosis: Valvular tissue thickens and narrows the valve opening, preventing blood from flowing from the left atrium to the left ventricle.
- Mitral Insufficiency, regurgitation: Valve is incompetent, preventing complete valve closure during systole.
- Mitral Valve Prolapse: Valve leaflets protrude into the left atrium during systole.
- Aortic Stenosis: Valvular tissue thickens and narrows the valve opening, preventing blood from flowing from the left ventricle into the aorta.
- Aortic Insufficiency: Valve is incompetent, preventing complete valve closure during diastole.

MITRAL STENOSIS

- Usually due to rheumatic endocarditis
- Causing valve thickening by fibrosis and calcification
- Mitral valve opening narrows

- Left atrial pressure rises and dilates
- Pulmonary artery pressure increases
- Can cause right ventricular failure

CLINICAL MANIFESTATIONS

- A Iow-pitched, rumbling, diastolic murmur is heard at the apex
- Dyspnea on exertion
- Orthopnea
  - ✓ Difficulty Breathing When Lying Flat
- Paroxysmal nocturnal dyspnea
  - ✓ Shortness of Breath that occurs suddenly during sleep

- Dyspnea and dry cough
- Hemoptysis and pulmonary edema
- Right sided heart failure may occur late
- Atrial dysrhythmias

MEDICAL MANAGEMENT

- Patients with mitral stenosis may benefit from anticoagulants to decrease the risk for developing atrial thrombus
- Surgical intervention consist of valvuloplasty
- Percutaneous transluminal valvuloplasty
- Mitral valve replacement

NURSING MANAGEMENT

- Place patient in a nigh Fowler's position to ease breathing
- **Monitor for:**
  - ✓ Pulmonary edema because it may be a complication of surgery
  - ✓ Thrombus because of a valve.
  - ✓ Arrhythmias because of an imitated heart- patient may feel palpitations, anxiety.
  - ✓ Arterial Blood Gas (ABG) to monitor for oxygenation, acidosis, alkalosis.
  - ✓ Weigh the patient daily to determine fluid balance
- **Explain to the patient:**
  - ✓ Signs and symptoms to look for and to report changes in condition.
  - ✓ Restrict diet to low-sodium and low-fat foods

MITRAL REGURGITATION (INSUFFICIENCY)

- ❖ Mitral regurgitation involves blood flowing back from the left ventricle into the left atrium during systole. Often the edges of the mitral valve leaflets do not close during systole
- ❖ Most common cause is mitral valve prolapse and rheumatic heart disease

CLINICAL MANIFESTATIONS

- Dyspnea, fatigue, and weakness are the most common symptoms.
- Palpitations, shortness of breath on exertion, and cough from pulmonary congestion also occur.
- Systolic murmur is heard as a high- pitched, blowing sound at the apex

MANAGEMENT

- Patients with mitral regurgitation and heart failure benefit from afterload reduction (arterial dilation)
- Angiotensin-converting enzyme (ACE) inhibitor
- Surgical intervention consists of mitral valvuloplasty (ie, surgical repair of the valve) or valve replacement

AORTIC REGURGITATION

- ❖ Aortic regurgitation is the flow of blood back into the left ventricle from the aorta during diastole"
- ❖ Blood from the aorta returns to the left ventricle during diastole"

ETIOLOGY

- Inflammatory lesions that deform the leaflets
- Rheumatic endocarditis,
- Congenital abnormalities
- Syphilis
- Dissecting aneurysm

"In many cases, the cause is unknown and is classified as idiopathic"

CLINICAL MANIFESTATIONS

- Patients experience forceful heart beats especially in the head and neck
- Marked arterial pulsations that are visible or palpable at the carotid or temporal arteries
- Palpable at the carotid or temporal arteries
- Exertional dyspnea and fatigue

- A diastolic murmur is heard as a high-pitched, blowing sound at the third or fourth intercostal space at the left sterna border.
- Widening of pulse pressure
- One characteristic sign of the disease is the water-hammer (Corrigan's) pulse

SURGICAL MANAGEMENT

- The treatment of choice is aortic valvuloplasty or valve replacement, preferably performed before left ventricular failure occurs.
- Surgery is recommended for any patient with left ventricular hypertrophy regardless of the presence or absence of symptoms

NURSING MANAGEMENT

- Patient is advised to avoid physical exertion
- Vasodilators such as calcium channel blockers (eg. nifedipine [Adalat, Procardia))
- Ace inhibitors (eg. Captopril, enalapril, lisinopril, ramipril). or hydralazine

AORTIC STENOSIS

- Narrowing of the orifice between the left ventricle and the aorta,

CAUSE

- Degenerative calcifications caused by inflammatory changes that occur in response to years of normal mechanical stress.

PATHOPHYSIOLOGY

- Progressive narrowing of the valve orifice occurs, the left ventricle contracts more forcefully and consumes more energy. It compensates by thickening its walls or hypertrophies.

CLINICAL MANIFESTATIONS

- Exertional dyspnea caused by increased pulmonary venous pressure
- Pulmonary edema may also occur
- Syncope and dizziness because decreased circulation to the brain
- Angina pectoris from increased demands of the left ventricle
- Loud rough systolic murmur heard over the aortic area
- Blood pressure is normal

TREATMENT

- Surgical replacement of the aortic valve or Percutaneous valvuloplasty procedures

INFECTIVE ENDOCARDITIS

- Microbial infection of the endothelial surface of the heart, it usually develops in people with prosthetic heart valves or structural heart defects

- Hospital acquired infective endocarditis occurs in patients with indwelling catheters

PATHOPHYSIOLOGY

- A deformity or injury of the endocardium brought about by infectious organisms leads to accumulation on the endocardium of fibrin and platelets. The infection may erode through the endocardium into underlying structures (valves /leaflets) causing deformity.

ASSESSMENTS

- Cluster of petechiae may be found on the body
- Small painful nodules (Osiers nodes) may be present in pads of fingers or toes
- Irregular red, purple, painless, flat macules (Janeway Lesions) may be present on the palms fingers and toes.
- Hemorrhages with pale centers in the eyes caused by emboli (Roth spots) caused by emboli may be observed in the fundi of the eyes
- Splinter hemorrhages (ie, reddish-brown lines and streaks) may be seen under the fingernails and toenails,

PREVENTION

- ❖ Antibiotic prophylaxis is recommended for high-risk patients immediately before and sometimes after the following procedures
  - Dental procedures
  - Tonsillectomy or adenoidectomy
  - Bronchoscopy
  - Cystoscopy
  - Surgery involving infected skin musculoskeletal tissue

MEDICAL MANAGEMENT

- Antibiotic therapy is usually administered parenterally in a continuous IV infusion for 2 to 6 weeks. penicillin is usually the medication of choice
- In fungal endocarditis, an antifungal agent, such as amphotericin B (eg, Abelcet, Amphocin, Fungizone), is the usual treatment

Nurse Home Care Instructions for the Client with Infective Endocarditis

- Teach the client to maintain aseptic technique during setup and administration of intravenous antibiotics.
- Instruct the client to monitor intravenous catheter sites for signs of infection and report this immediately to the physician.
- Instruct the client to record the temperature daily for up to 6 weeks and report fever.
- Encourage oral hygiene at least twice a day with a soft toothbrush and rinse well with water after brushing
- Client should avoid use of oral irrigation devices and flossing to avoid bacteremia.

MYOCARDITIS

- ❖ Myocarditis is an inflammation of the myocardium. It is usually diagnosed when it leads to significant cardiac dysfunction. Myocarditis can cause considerable morbidity and mortality
- ❖ Infection could be bacterial, protozoal, fungal parasitic
- ❖ Viral myocarditis is the most common type
- ❖ Characterized by necrosis and cell injury associated with inflammation of the heart muscle

ASSESSMENT FINDINGS

- Non-specific symptoms: fatigue, dyspnea and palpitation
- If the disease has progressed, symptoms of heart failure present, such as tachycardia, pulmonary edema, diaphoresis, neck vein distention, and cardiomegaly.
- In myocarditis, the ECG can show low-voltage QRS complexes, ST segment elevation, or heart block
- An S4 and systolic ejection murmurs may be heard on auscultation
- Patients may also sustain sudden cardiac death or quickly develop severe congestive heart failure

MEDICAL MANAGEMENT

- Patient are given specific treatment for the underlying cause if it is known (eg, penicillin for hemolytic streptococci)
- Inotropic support of cardiac function with dopamine, or dobutamine may be used Netroprusside and nitroglycerine may be used to decrease afterload
- Beta Blocker are avoided because they decrease the strength of ventricular contraction (have a negative inotropic effect)
- Sedation may be necessary to decrease cardiac workload
- Intra-aortic balloon pulsation and left ventricular assists devices have been used to improve cardiac output myocarditis

NURSING ALERT

Patients with myocarditis are sensitive to digitalis. Nurses must closely monitor these patients for digitalis toxicity, which evidenced by dysrhythmia, anorexia, nausea, vomiting, headache, and malaise,

Pericarditis

- ❖ Pericarditis refers to an inflammation of the pericardium, the membranous sac enveloping the heart. It may be a primary illness or it may develop during various medical and surgical disorders.

PATHOPHYSIOLOGY

- The inflammation process of pericarditis may lead to an accumulation of fluid in the pericardial (pericardial effusion) and increased pressure on the heart leading to cardiac tamponade
- Prolonged episodes of pericarditis may lead to thickening and decreased elasticity of pericardium. These conditions restrict the heart's ability to fill with blood (constrictive pericarditis)
- Restricted filling may result in increased systemic venous pressure

ASSESSMENTS

- Chest pain- located beneath the clavicle, in the neck or in the left scapular region, may worsen with deep inspiration and may be relieved with a forward leaning or sitting position. (Tripod Position)
- Most characteristic sign of pericarditis is a creaky or scratchy friction rub heard most clearly at the left lower sternal border (pericardial friction rub)

MEDICAL MANAGEMENT

- Administer therapy for treatment and symptom relief, and detect signs and symptoms of cardiac tamponade.
- Analgesics and nonsteroidal anti-inflammatory drugs (NSAIDs
- Indomethacin (Indocin) is contraindicated because it may decrease coronary blood flow
- A pericardial window, a small opening made in the pericardium
- May be performed to allow continuous drainage into the chest cavity.
- Surgical removal of the tough encasing pericardium (pericardiectomy) may be necessary to release both ventricles from the constrictive and restrictive inflammation scarring.

PERICARDIOCENTESIS

❖ Procedure in which some of the pericardial fluid is removed
  - Emergency resuscitation should be readily available
  - The head of the bed is elevated to 45 to 60 degrees, placing the heart In proximity to the chest wall so that the needle can be directly inserted into the pericardial sac
  - Slow iv infusion is started in case it becomes necessary to administer emergency medications or blood products
  - Ultrasound imaging is used to guide placement of the needle into the pericardial space

❖ Desired effect
  - Decrease in central venous pressure
  - Increase in blood pressure
  - Withdrawal of pulsus paradoxus
    - ✓ >10 mm Hg drop in blood pressure during inspiration
  - Disappearance of prominent neck veins due to increased venous pressure

COMPLICATIONS OF PERICARDIOCENTESIS

- Coronary artery puncture
- Myocardial trauma
- Dysrhythmias
- Pleural laceration
- Gastric puncture

NURSING MANAGEMENT

- Patients with acute pericarditis require pain management with analgesics, positioning, and psychological support caring for patients with pericarditis must be alert to cardiac tamponade
- After pericardiocentesis, the patient's heart rhythm, blood pressure, venous pressure, and heart sounds are monitored to detect possible recurrence of cardiac tamponade

NURSING ALERT

- A pericardial friction rub is diagnostic feature of pericarditis. It has a creaky or Scratchy sound and is louder at the end of exhalation.
- Nurses should monitor for the pericardial friction rub by placing the diaphragm of the stethoscope tightly against the thorax and auscultating the left sternal edge in the fourth intercostal space, the site where the pericardium comes into contact with the left chest wall.
- The rub may be heard best when a patient is sifting and leaning forward.

CARDIAC TAMPONADE

- ❖ A condition where the heart is unable to pump blood due to accumulation of fluid in the pericardial sac (pericardial effusion)
- ❖ This condition restricts ventricular filling resulting to decreased cardiac output
- ❖ Acute tamponade happens when there sudden accumulation of more than 50 ml fluid in the pericardial sac

CAUSES

- Cardiac trauma
- Complication of Myocardial infarction
- Pericarditis

ASSESSMENT FINDING

- **BECK's Triad**
  - ✓ Jugular vein distention
  - ✓ Hypotension
  - ✓ Distant/muffled heart sound
- Pulsus paradoxus
  - >10 mm Hg drop in blood pressure during inspiration
- Increased Central Venous Pressure
- Decreased cardiac output
- Anxiety
- Dyspnea

LABORATORY FINDINGS

- Echocardiogram= shows accumulation of fluid in the pericardial sac
- Chest X-ray

NURSING MANAGEMENT

- The client needs to be placed in a critical care unit for hemodynamic monitoring.
- Administer fluids intravenously as prescribed to manage decreased cardiac output.
- Prepare the client for pericardiocentesis to withdraw pericardial fluid if prescribed.
- Monitor for recurrence of tamponade following pericardiocentesis.
- If the client experiences recurrent tamponade or recurrent effusions or develops adhesions from chronic pericarditis, a portion (pericardial window) or all of the pericardium (pericardiectomy) may be removed to allow adequate ventricular filling and contraction.

ANGINA PECTORIS

- ❖ Angina pectoris is a clinical syndrome usually characterized by episodes or paroxysms of pain or pressure in the anterior chest
- ❖ The cause is insufficient coronary blood flow, resulting in a decreased oxygen supply when there is increased myocardial demand for oxygen

PATHOPHYSIOLOGY

- Angina is usually caused by atherosclerotic disease and associated with a significant obstruction of at least one major coronary artery

TYPES OF ANGINA

- ❖ Stable angina: predictable and consistent pain that occurs on exertion and is relieved by rest and/or nitroglycerin
- ❖ Unstable angina (also called pre-infarction angina or crescendo angina): symptoms increase in frequency and severity; may not be relieved with rest or nitroglycerin
- ❖ 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 pain

TRIGGERING FACTORS

- Exertion
- Exposure to cold
- 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;
  - ✓ In a severely compromised heart, shunting of blood for digestion can be sufficient to induce anginal pain
- Stress or any emotion-provoking situation, causing the release of catecholamine's, which increases blood pressure, heart rate, and myocardial workload

MANIFESTATIONS

- Heavy sensation in the upper chest that ranges from discomfort to agonizing pain
- Severe apprehension and a feeling of impending death.
- Retrosternal pain
- Pain radiates to the neck, jaw, shoulders, and inner aspects of the upper am-is, usually the left arm

"An important characteristic of angina is that it subsides with rest or administering nitroglycerin. In many patients, anginal symptoms follow a stable, predictable pattern."

ASSESSMENT AND DIAGNOSTIC FINDINGS

- ECG may show changes indicative of ischemia such as T-wave inversion
- Nuclear scan or invasive procedure (eg, cardiac catherization, coronary angiography).
- ST depression

MEDICAL MANAGEMENT

- The objectives of the medical management of angina are decrease the oxygen demand of the myocardium and to increase the oxygen supply
- Percutaneous transluminal coronary angioplasty (PTCA)
  - ✓ Balloon-tipped catheter is used to open blocked coronary vessels and resolve ischemia. The purpose of PTCA is to improve blood flow within the coronary artery by compressing and "cracking" the atheroma
- Intracoronary stents
  - ✓ A Stent is a metal mesh that provides structural support to vessel at risk of acute closure.
- Atherectomy
  - ✓ Atherectomy removes plaque from a coronary artery by the use of a cutting chamber on the inserted catheter of a rotating blade that pulverizes the plaque.
- CABG (Coronary Artery Bypass Graft)
  - ✓ Surgical procedure in which a blood vessel is grafted to an occluded artery so that blood can flow beyond the occlusion

PHARMACOLOGIC MANAGEMENT

- Nitroglycerine causes dilation of the veins the result is venous pooling of blood throughout the body. As a result, less blood returns to the heart, decreasing the cardiac workload
- Facts about nitroglycerine
  - ✓ Can be given:
    - o Sublingual tablet
    - o Spray
    - o Topical agent,
    - o Intravenous I.V. administration

MEDICATIONS USED TO TREAT ANGINA

NITRATES

- Nitroglycerin (Nitrostat, Nitro-Bid): Short-term and long-term reduction of myocardial oxygen consumption through selective vasodilation

Beta-Adrenergic Blocking Agents (beta- blockers)

- Metoprolol (Lopressor, Toprol) Reduction of myocardial oxygen consumption by blocking beta-adrenergic
- Atenolol (Tenormin) stimulation of the heart

Calcium Ion Antagonists (calcium channel blockers)
- Amlodipine (Norvasc) Negative inotropic effects; indicated in patients not responsive to beta-blockers;
- Diltiazem (Cardizem, Tiazac) used primary treatment for vasospasm
- Felodipine (Plendil)

Antiplatelet Medications
- Aspirin Prevention of platelet aggregation
- Clopidogrel (Plavix)
- Glycoprotein agents:
- Abciximab (ReoPro)
- Tirofiban (Aggrastat)
- Eptifibatide (Integrilin)

Anticoagulants
- Heparin (unfractionated): Prevention of thrombus formation
- Low-molecular-weight heparins (LMWHs): Enoxaparin (Lovenox)
- Dalteparin (Fragmin)

MYOCARDIAL INFARCTION
- ❖ In an MI, an area of the myocardium is permanently destroyed, typically because plaque rupture and subsequent thrombus formation result in complete occlusion of the artery.
- ❖ The ECG usually identifies the type and location of the MI, and other ECG indicators such as a Q wave and patient history identify the timing. Regardless of the location, the goals of medical therapy are to prevent or minimize myocardial tissue death and prevent complications

RISK FACTORS
Non-modifiable Risk Factor
- Age
  - ✓ Average age of a person having a first heart attack is 65.8 yrs (male) and 70. 4 yrs (female) - *AHA 2003*
- Family history
- Ethnic background
  - ✓ African-Americans has a higher risk for developing M.I.

Modifiable Risk Factor
- Hypertension
- Smoking
- Hyperlipidemia
- Obesity

- Impaired glucose tolerance (DM)
- Physical inactivity
- Stress

ASSESSMENT
SUBSTANTIAL CHEST PAIN
- The pain associated with an MI usually lasts longer than 30 minutes
- Radiating to the left arm, back or jaw
- Occurring w/o a cause usually in the morning
- Relieved only by opioids associated with nausea, diaphoresis, dyspnea, fear & anxiety, palpitations, fatigue, shortness of breath.
- Decreased left ventricular function
- Decreased cardiac output
- Cardiovascular system compensates by increasing heart rate (Frank-Starling law)

CG AND CARDIAC ENZYMES ASSESSMENTS
- T-wave in
- ST-segment elevation
- Abnormal Q wave

- Elevated CKMB assessed by mass assay is an indicator of acute MI
- An increase in the level of troponin in the serum can be detected within a few hours during acute MI.
- Enzymes that indicate Myocardial Infarction

ENZYMES	TIME OF ELEVATION	DESCRIPTION
MYOGLOBIN	First 1-3 hours	First enzyme to elevate due to decreased oxygenation
TROPONIN I	2-4 hours	Released at the mentioned time frame
AST (aspartate amino transferase)	8 hours	Is also used for liver damage
CK-MB	24 hours	**Cardiac- specific** isoenzyme; it is found mainly in cardiac cells and therefore increases only when there has been damage to these cells
LDH (Lactic Dehydrogenase)	72 hours	Reflects tissue breakdown and hemolysis

MEDICAL MANAGEMENT

❖ The goals of medical management are to minimize myocardial damage, preserve myocardial function, and prevent complications this can be achieved by:
- Reperfusing the area with the emergency use of thrombolytic medications
- Reducing myocardial oxygen demand and increasing oxygen supply with medications, oxygen administration, and bed rest

PHARMACOLOGIC THERAPY

❖ Drug of choice: Morphine I.V.
  - Potent vasodilator: Increases oxygen supply to myocardial tissues
  - Decreases oxygen demand

❖ (ACE) inhibitors decreases blood pressure thus decreasing the workload of the heart

❖ Thrombolytics dissolve (ie, lyse) the thrombus in a coronary artery (thrombolysis), allowing blood to flow through the coronary artery again

❖ THROMBINS2 – "the new MONA (Morphine, Oxygen, Nitroglycerin, Aspirin)"
  - Thienopyridines: Antiplatelet drugs (Clopidogrel, Prasurgel)
  - Heparin: Anticoagulant
  - RAAS Inhibitors: ACE-Inhibitors (-pril) or ARBs (-sartan)
  - Oxygen
  - Morphine
  - Beta-blockers: -olol
  - Invasive interventions
  - Nitroglycerin: vasodilator
  - Statin: reduces cholesterol levels (Atorvastatin, Rosuvastatin)
  - Salicylate: aspirin/acetylsalicylic acid (ASA)

Common Physical Presentation of the Patient with Acute Myocardial Infarction	
General	Alert anxious, Restless often fatigued
Skin	Cool, clammy; diaphoretic
Heart	Cardiovascular S3 or S4 gallop may or may not be present; dysrhythmias or murmurs. Jugular venous distention May be seen in the presence of pump failure
Lungs	Dyspnea, tachypnea, rales (crackles) suggest pulmonary congestion and heart failure
Circulatory	Peripheral pulses may be pounding or thready, regular or irregular
Gastrointestinal	Nausea and vomiting

CARDIOMYOPATHY

❖ A heart muscle disease associated with cardiac dysfunction.

TYPES
- Dilated
- Hypertrophic
- Restrictive

DILATED CARDIOMYOPATHY

- ❖ Extensive damage to the myofibrils & interference with myocardial metabolism
- ❖ Normal ventricular wall thickness but dilation of both ventricles & impairment of systolic function
- ❖ Decreased CO- inadequate heart pumping
- ❖ Dyspnea on exertion fatigue and palpitation

CAUSES

- Alcohol abuse
- Chemotherapy

ASSESSMENT

- Fatigue, weakness
- HF (left side)
- Dysrhythmias
- Moderate to severe cardiomegaly

HYPERTROPHIC CARDIOMYOPATHY

- ❖ Asymmetric ventricular hypertrophy and disarray of myocardial fibers
- ❖ LVH leads to a stiff LV that result in diastolic filling abnormalities
- ❖ Obstruction in LV outflow
- ❖ 50 % genetically inherited

ASSESSMENT

- Dyspnea
- Angina
- Fatigue, syncope, palpitations
- Mild cardiomegaly
- Ventricular dysrhythmias
- Sudden death common
- Heart failure

RESTRICTIVE CARDIOMYOPATHY

- ❖ Restriction or filling of the rigid ventricular walls
- ❖ The cause is unknown (ie, idiopathic) in most cases.
- ❖ Can be caused by endocrinal or myocardial disease and produce a clinical picture similar to constrictive pericarditis
- ❖ Fibrosed walls cannot expand or contract
- ❖ Chamber is also narrowed

ASSESSMENT

- Dyspnea & fatigue
- HF (Right side)
- Mild to moderate cardiomegaly
- Heart block

SHOCK

- ❖ Inadequate organ perfusion to meet the tissue's oxygenation demand.
- ❖ Hypoperfusion can be present in the absence of significant hypotension
- ❖ 3 Types of Shock
    - Hypovolemic
    - Cardiogenic
    - Distributive – systemic vasodilation leading to decreased blood pressure and insufficient tissue perfusion
        - Neurogenic
        - Anaphylactic
        - Septic

TYPES OF SHOCK

HYPOVOLEMIC

❖ Occurs when there is a loss of fluid (blood, plasma) resulting in inadequate tissue perfusion; caused by:
- ✓ Hemorrhage/Excessive bleeding
- ✓ Excessive diarrhea or vomiting
- ✓ Dehydration
- ✓ Fluid loss from fistulas or burns
- ✓ Massive third spacing/edema

TREATMENT
- Primary problem/underlying cause must be treated
- Whole blood, plasma (fluid and blood) Replacement and electrolytes

MANAGEMENT:
Major goals in the treatment of hypovolemic shock are to restore intravascular volume to reverse the sequence of events leading to inadequate tissue perfusion, to redistribute fluid volume, and to correct the underlying cause of the fluid loss as quickly as possible

CARDIOGENIC

❖ Occurs when pump failure causes inadequate tissue perfusion; caused by
- ✓ Congestive heart failure
- ✓ Myocardial infarction
- ✓ Cardiac tamponade

MANAGEMENT
- The goals of medical management in cardiogenic shock are to limit further myocardial damage and preserve the healthy myocardium and to improve the cardiac function by increasing cardiac contractility, decreasing ventricular afterload, or both.

NEUROGENIC

❖ Neurogenic shock develops as a result of **the loss of autonomic nervous system** function below the level of the lesion in the spinal cord which caused rapid vasodilation and subsequent pooling of blood within the peripheral vessels

MANAGEMENT
- Treatment of neurogenic shock involves restoring sympathetic tone, either through the stabilization of a spinal cord injury or, by positioning the patient properly.
- It is important to elevate and maintain the head of the bed at least 30 degrees to prevent neurogenic shock when a patient receives spinal or epidural anesthesia. Elevation of the head helps prevent the spread of the anesthetic agent up the spinal cord.

ANAPHYLACTIC

❖ Caused by an allergic/anaphylactic reaction that causes a release of histamine and subsequent systemic vasodilation

MANAGEMENT:
- Treatment of anaphylactic shock requires removing the causative antigen (eg, discontinuing an antibiotic agent), administering medications that restore vascular tone, and providing emergency support of basic life functions.
- Epinephrine is given for its vasoconstrictive action (emergency drug).
- Diphenhydramine (Benadryl) is administered to reverse the effects of histamine, thereby reducing capillary permeability.

SEPTIC

❖ Similar to anaphylaxis; the body's reaction to bacterial toxins (generally gram-negative infections) results in the leakage of plasma into tissues

MANAGEMENT
- Current treatment of sepsis and septic shock involves identification and elimination of the cause of infection.

TYPE	MECHANISM
Hypovolemic	Loss of blood or plasma
Cardiogenic	Decreased pumping capability/contractility of heart
Distributive	Systemic vasodilation
- Anaphylactic	due to severe allergic reaction
- Septic	due to severe infection
- Neurogenic	due to loss of SNS and vasomotor tone

HYPERTENSION

❖ Hypertension is defined as a systolic blood pressure greater than 140 mm Hg and a diastolic pressure greater than 90 mmHg

TYPES OF HYPERTENSION

ESSENTIAL HYPERTENSION

- No known direct cause
- Risk factor
  - ✓ Age > 60 yrs
  - ✓ Family history of hypertension
  - ✓ Excessive caloric consumption
  - ✓ Physical inactivity
  - ✓ Excessive alcohol intake
  - ✓ Hyperlipidemia
  - ✓ High salt intake or caffeine; reduced intake of potassium, calcium, or magnesium
  - ✓ Smoking

SECONDARY HYPERTENSION

- Disease
  - ✓ Renal vascular & parenchymal disease
  - ✓ Primary aldosterone
  - ✓ Pheochromocytoma
  - ✓ Cushing's disease
  - ✓ Coarctation of aorta
  - ✓ Brain tumors
  - ✓ Encephalitis

PHARMACOLOGIC THERAPY

- For patients with uncomplicated hypertension and no specific indications for another medication, the recommended initial medications include diuretics, beta blockers and angiotensin-converting enzyme (ACE)

BETA-BLOCKERS

- First line drug therapy
- Reduce BP by decreasing CO
- Decrease sympathetic stimulation
- Inhibit release of renin from the kidneys

ANGIOTENSIN -CONVERTING ENZYME (ACE) INHIBITORS

- Lower BP by reducing peripheral vascular resistance w/o reflex increase in CO, HR or contractility.
  - ✓ Captopril (Capoten)
  - ✓ Enalapril (Vasotec)
  - ✓ Lisinopril (Zestril)
- Decrease secretion of aldosterone

CENTRAL ALPHA ANTAGONIST

- Acts on CNS preventing reuptake of norepinephrine resulting in lower peripheral Vascular resistance & BP
  - ✓ Clonidine (Catapres)
  - ✓ Does not decrease renal blood flow

CALCIUM-CHANNEL BLOCKER

- Verapamil (Isoptin
- Amlodipine
- Diltiazem
- Nicardipine

DIURETICS (WATER PILL)

- Thiazide
  - ✓ Chlorothiazide (Diuril)
  - ✓ Hydrochlorothiazide (Hydrodiuril)
- Loop
  - ✓ Furosemide (Lasix)
  - ✓ Bumetanide (Bumex)
- Potassium - sparing
  - ✓ Spironolactone (Aldactone)

NURSING INTERVENTIONS

- The objective of nursing care for patients with hypertension focuses on lowering and controlling the blood pressure without adverse effects and without undue cost through:
  - ✓ Adhere to the treatment regimen
  - ✓ Implementing necessary lifestyle changes
  - ✓ Taking medications as prescribed
  - ✓ Scheduling regular follow-up appointments

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 upper extremities

- Factors
  - ✓ Men between 20-35 years of age
  - ✓ Heavy smoking and chewing tobacco

CLINICAL MANIFESTATIONS

- Foot cramps, especially of the arch (instep claudication), after exercise
- Pain is relieved by rest
- Intense rubor (reddish-blue discoloration) of the foot and absence of the pedal pulse

MEDICAL MANAGEMENT

- The main objectives are to improve circulation to the extremities, prevent the progression of the disease
- Vasodilators are rarely prescribed

RAYNAUD'S PHENOMENON

❖ Raynaud's phenomenon is a form of intermittent arteriolar vasoconstriction that results in coldness, pain, and pallor of the fingertips or toes.

Factors:

- Raynaud's phenomenon is most common in women between 16 and 40 years of age, and it occurs more frequently in cold climate

CLINICAL MANIFESTATION

- The characteristic sequence of color change of Raynaud's phenomenon is described as **white, blue, and red**.
- Numbness, tingling, and burning pain occur as the color changes.
- The manifestations tend to be bilateral and symmetric and may involve toes and fingers.

MEDICAL MANAGEMENT

- Avoiding the particular stimuli (E.g. cold, tobacco) that provoke vasoconstriction is a primary factor in controlling Raynaud's phenomenon.
- Calcium channel blockers (Nifedipine [Procardia], amlodipine [Norvasc])
- Sympathectomy (interrupting the sympathetic nerves by removing the sympathetic ganglia or dividing their branches) may help some patients.

NURSING MANAGEMENT

- Exposure to cold must be minimize
- Sweater should be available when entering air-conditioned rooms
  - ✓ Avoid smoking and all sources of nicotine like nicotine gum or patches.

VENOUS THROMBOEMBOLISM

❖ Deep vein thrombosis (DVT)
- Virchow's triad
  - ✓ Vessel wall injury
  - ✓ Venous stasis (stasis of blood)
  - ✓ Altered blood coagulation

- Risk Factors for Venous stasis
  - ✓ Bed rest or immobilization
  - ✓ Obesity
  - ✓ History of varicosities
  - ✓ Spinal cord injury
  - ✓ Age (greater than 65 years)

ASSESSMENT

- Obstruction of the deep veins comes edema and swelling of the extremity because the outflow of venous blood is inhibited
- Limb pain, a feeling of heaviness, functional impairment, ankle engorgement, and edema

PREVENTION

- Preventive measures include the application of graduated compression stockings
- In surgical patients is administration of subcutaneous unfractionated or low molecular- weight heparin (LMWH).
- Lifestyle changes as appropriate, which may include weight loss, smoking cessation, and regular exercise

MEDICAL MANAGEMENT

Anticoagulant therapy
- (Administration of a medication to delay the clotting time of blood, prevent the formation of a thrombus in postoperative patients, and forestall the extension of a thrombus after it has formed)
- Oral Anticoagulant Warfarin (Coumadin)

Thrombolytic
- Alteplase (Activase, t-PA)
- Urokinase (Abbokinase)
- Streptokinase (Streptase)

NURSING MANAGEMENT

- If the patient is receiving anticoagulant therapy, the nurse must frequently monitor the aPTT, prothrombin time (PT) and INR
- Elevation of the affected extremity, graduated compression stockings, and analgesic agents for pain relief are adjuncts the therapy. They help improve circulation and increase comfort.
- Warm, moist- packs applied to the affected extremity reduce the discomfort associated with DVT
- The patient is encouraged to walk once anticoagulation therapy has been initiated. The nurse should instruct the patient that walking is better than standing or sitting for long periods

NURSING ALERT

- For ambulatory patients, graduated compression stockings are removed at night and reapplied before the legs are lowered from the bed to the floor in the morning.

ANEURYSMS

❖ An aneurysm is a localized sac or dilation formed at a weak point in the wall of the artery.

CAUSES

- Congenital: Primary connective disorders (Marfan's syndrome)
- Mechanical (hemodynamic): Poststenotic and arteriovenous fistula and amputation related
- Traumatic (pseudoaneurysms): Penetrating arterial injuries, blunt arterial (pseudoaneurysms)
- Infectious (mycotic): Bacterial, fungal, spirochetal infections
- Pregnancy-related degenerative:
- Nonspecific, inflammatory variant
- Anastomotic (postarteriotomy) and graft aneurysms: Infection, arterial wall failure, suture failure, graft failure

TYPES

- ❖ Normal artery.
- ❖ False aneurysm—actually a pulsating hematoma. The clot and connective tissue are outside the arterial wall,
- ❖ True aneurysm. One, two, or all three layers of the artery may be involved.
- ❖ Fusiform aneurysm—symmetric, spindle shaped expansion of entire circumference of involved vessel.
- ❖ Saccular aneurysm—a bulbous protrusion of one side of the arterial wall.
- ❖ Dissecting aneurysm—this usually is a hematoma that splits the layers of the arterial wall.

MEDICAL MANAGEMENT

- Antihypertensive agents, including:
  - ✓ Diuretics,
  - ✓ Beta blockers,
  - ✓ Ace inhibitors,
  - ✓ Angiotensin II receptor antagonists, calcium channel blockers

These drugs are frequently prescribed to maintain the patient's blood pressure within acceptable limits to prevent rupture of the aneurysms

SURGICAL MANAGEMENT

- Resection of the vessel and sewing a bypass graft in place
- Endovascular grafting, which involves the transluminal placement and attachment of a sutureless aortic graft prosthesis across an aneurysm