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Cardiovascular Care

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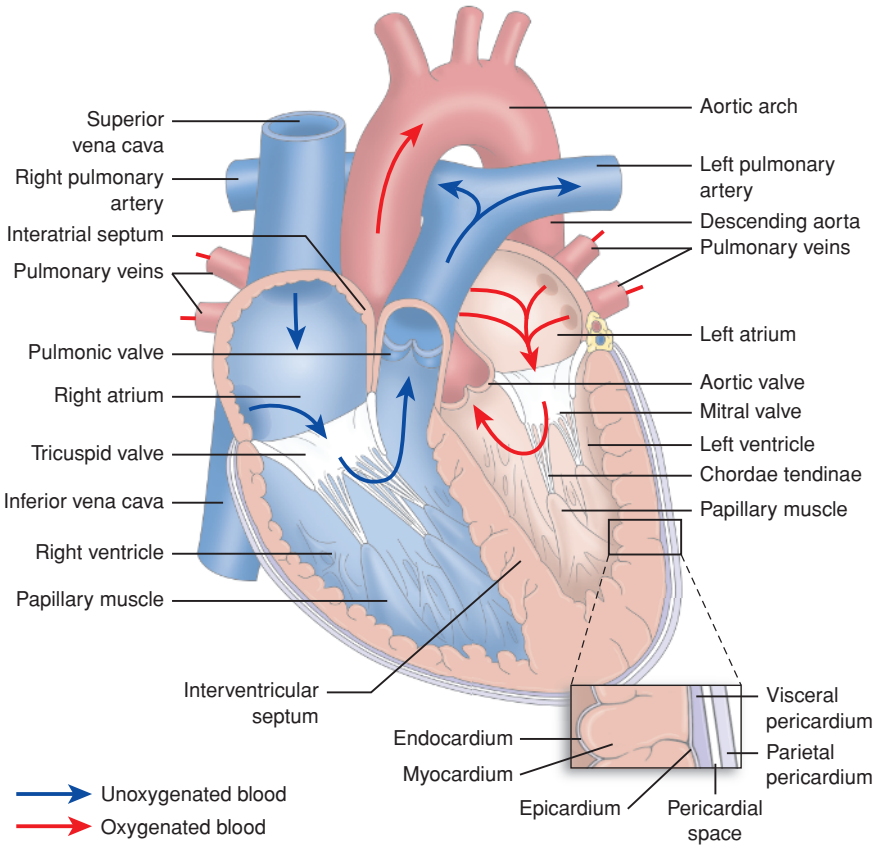
NORMAL ANATOMY AND CONDUCTION SYSTEM OF THE HEART

Blood enters the heart through the superior vena cava and inferior vena cava. The blue arrows show the blood flow though the right atrium and right ventricle to the lungs and the red arrows show the blood flow from the lungs through the left atrium and left ventricle to the aorta. Normal heart sounds are created by closure of the four valves of the heart. S_1, the first heart sound, the "lub," occurs as a result of the closure of the tricuspid and mitral valves. S_2, the second heart sound, the "dub," occurs as a result of the closure of the pulmonic and aortic valves.



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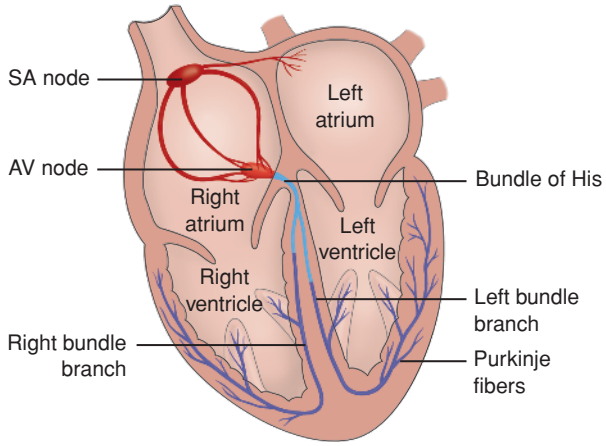
Electrical impulses originate in the SA node, travel to the AV node, down the bundle of His to the Purkinje fibers. Blood flow through the heart and the body is dependent on synchronization of the electrical system.



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TESTS

SERUM STUDIES

Electrolytes

- Potassium level—3.5 to 5.0 mEq/L
  - Excessive levels can lead to cardiac depression, dysrhythmias, and cardiac arrest
- Calcium level—8.2 to 10.3 mEq/dL
  - Increased levels can cause shortening of the QT interval, bradycardia, and heart block
  - Low levels result from loop diuretics. Clinical manifestations include cardiac changes such as prolonged ST and QT intervals, and CHF
- Magnesium level—1.3 to 2.1 mg/dL
  - Decreased levels can cause cardiac dysrhythmias: ventricular fibrillation (VF), torsades de pointes
  - Excessive levels can lead to bradycardia and conduction system blocks
- Sodium level—135 to 145 mEq/L
  - Etiology of low levels include CHF, diuretic therapy; clinical manifestations include peripheral and pulmonary edema
- Chloride level—97 to 107 mEq/L
  - Low levels caused by diuretics
  - Etiology of high levels include CHF
- Carbon dioxide level—23 to 29 mEq/L
  - Reduced by thiazide diuretics

Lipid Profile

Test	Measures	Level (mg/dL)	Classification
Total Cholesterol	Measures all of the cholesterol in all the lipoprotein particles.	<200	Desirable
		200–239	Borderline high
		≥240	High
High-Density Lipoprotein Cholesterol (HDL-C)	Measures the cholesterol in HDL particles; often called "good cholesterol" because it removes excess cholesterol and carries it to the liver for removal.	≤40	Low
		≥60	High
Low-Density Lipoprotein Cholesterol (LDL-C)	Calculates the cholesterol in LDL particles; often called "bad cholesterol" because it deposits excess cholesterol in walls of blood vessels, which can contribute to atherosclerosis. Usually, the amount of LDL-C is calculated using the results of total cholesterol, HDL-C, and triglycerides	<100	Optimal
		100–129	Near optimal/above optimal
		130–159	Borderline high
		160–189	High
		≥190	Very high
Triglycerides	Measures all the triglycerides in all the lipoprotein particles; most is in the very low-density lipoproteins (VLDLs).	Less than 150	Normal
		150–199	Borderline high
		200–499	High
		>500	Very high

Evaluating Lipid Test Results

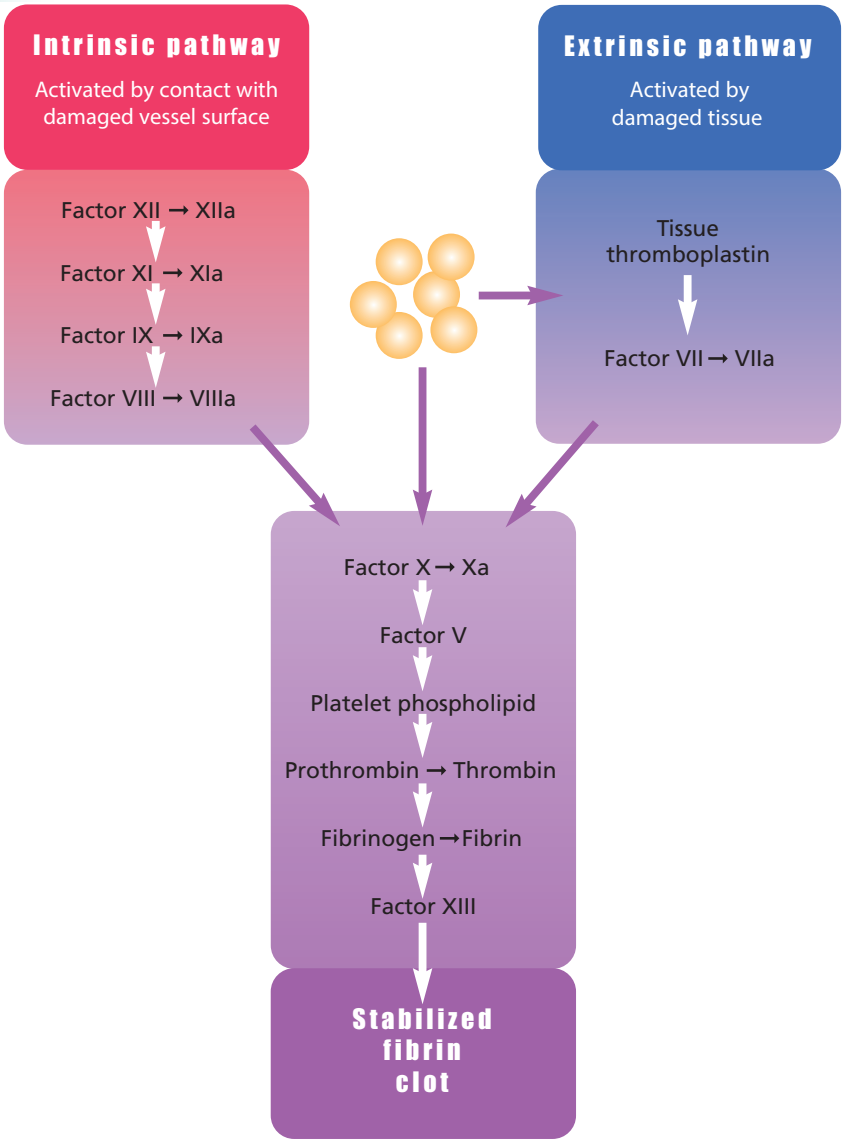
COAGULATION TESTS

Test	Action	Elevated Levels	Normal Range
International Normalized Ratio (INR) INR is the preferred test and best standardized measurement of PT.	The INR system was established to reduce the interlaboratory variation in prothrombin time. The INR is calculated as a ratio of the patient's PT to a control PT obtained using an international reference thromboplastin reagent developed by the World Health Organization (WHO). Used for monitoring warfarin (Coumadin) treatment.	Increased INR values may indicate disseminated intravascular coagulation (DIC), liver disease, antiphospholipid antibodies, vitamin K deficiency, or uncontrolled oral anticoagulation	In healthy people an INR of 1.1 or below is considered normal. An INR range of 2.0 to 3.0 is generally an effective therapeutic range for people taking warfarin for disorders such as atrial fibrillation or a blood clot in the leg or lung. In certain situations, such as having a mechanical heart valve, you might need a slightly higher INR.
Prothrombin time test (PT)	Assesses the clotting ability of blood. A prothrombin time within this range indicates that the patient has normal amounts of clotting factors VII and X.	A prolonged PT time is considered abnormal	11–15 sec
Activated Partial Thromboplastin Time (aPTT)	aPTT is sensitive to the deficiencies or abnormalities of both intrinsic and common coagulation factors, i.e., Factors I, II, V, X, VIII, IX, XI, XII, Fletcher factor, and Fitzgerald factor. The activated partial thromboplastin time (aPTT, PTT) measures the time it takes plasma to clot when exposed to substances that activate the contact factors, which assesses the intrinsic and common pathways of coagulation	When the aPTT is prolonged, there is an inhibitor present in patient's plasma.	29–35 sec
Thrombin Time (TT)	The thrombin time (TT) measures the final step of coagulation, the conversion of fibrinogen to fibrin	Thrombin time is prolonged in the presence of heparin, hypofibrinogenemia, dysfibrinogenemia, and fibrin degradation product	15–17 sec

Understanding Clotting

PICTURING PATHO

Clotting is initiated through two different pathways.



Tests to Identify Myocardial Infarction

After myocardial infarction (MI), myocardial cell death can be recognized by the appearance, in the blood, of different proteins released into the circulation from the damaged myocytes: myoglobin, cardiac troponin T and I, CK, and LDH.

Myoglobin

- Elevated
- First marker of cardiac injury after acute MI

CK-MB

- Returns to normal quickly
- Most reliable when reported as a percentage of total creatine kinase (CK) (relative index)

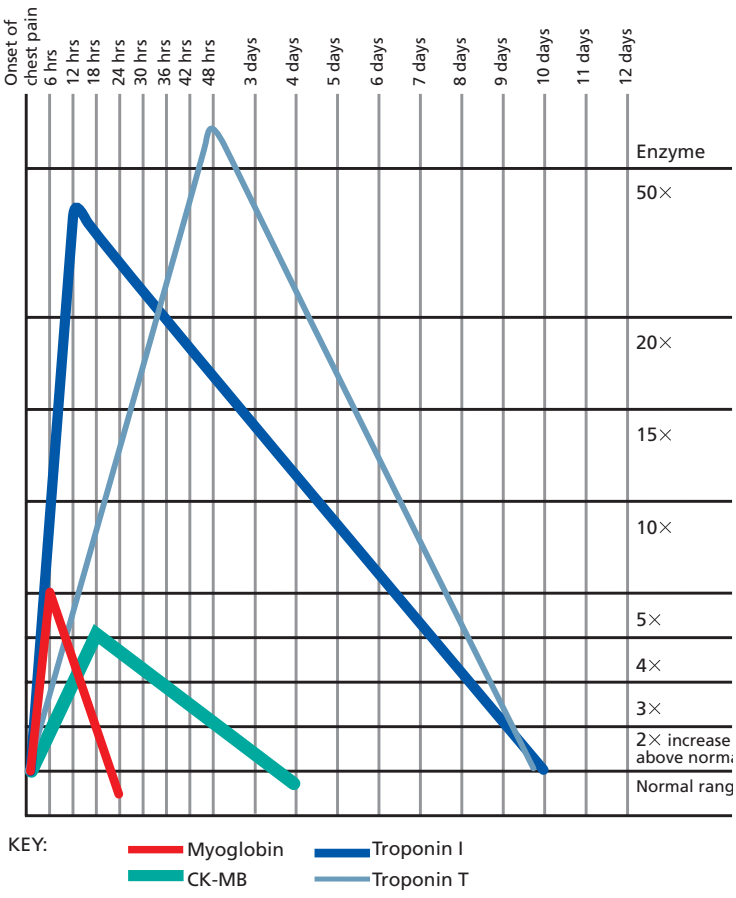
Troponin I

- Isotypes of troponin found only in myocardium
- Elevated
- Specific to myocardial damage

Troponin T

- Isotype of troponin that is less specific to myocardial damage (can indicate renal failure)
- Elevated
- Determined quickly at bedside

Release of Cardiac Enzymes and Proteins



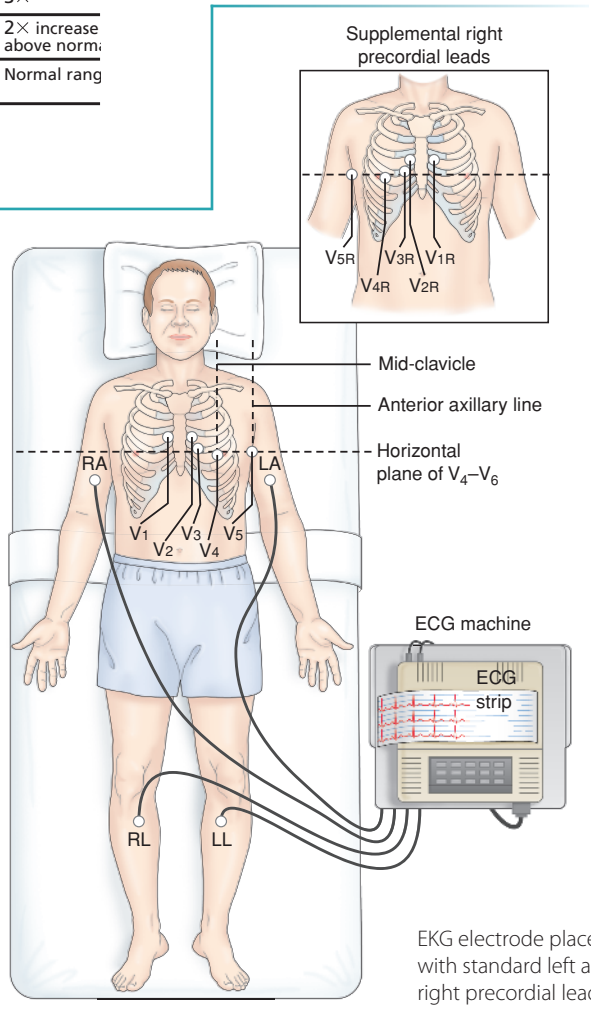
Exercise Stress Testing

A stress test, sometimes called a treadmill test or exercise test, is done while the patient walks or runs on a motorized treadmill. The test is based on the principle that exercise increases myocardial demand and coronary artery blood supply which may be inadequate during exercise and stress which can result in myocardial ischemia. Exercise stress testing is a nonevasive procedure that assesses the heart's response to an increased workload. The test can demonstrate if the blood supply is reduced in the arteries that supply the heart during exercise and identify appropriate exercise levels for the patient.

CARDIAC TESTS

Electrocardiography

The electrocardiogram (ECG) is a graphical representation (time versus amplitude of electrical vector projection) of the electrical activity of the heart. While imperfect as a diagnostic or prognostic tool, it provides a significant amount of information and is a first-line test necessary for the proper care of the patient with potential cardiovascular disease. The standard 12-lead ECG is of great value in identifying myocardial ischemia, MI, rhythm and conduction disturbances, chamber enlargement, electrolyte imbalances, and drug toxicity.



EKG electrode placement with standard left and right precordial leads.

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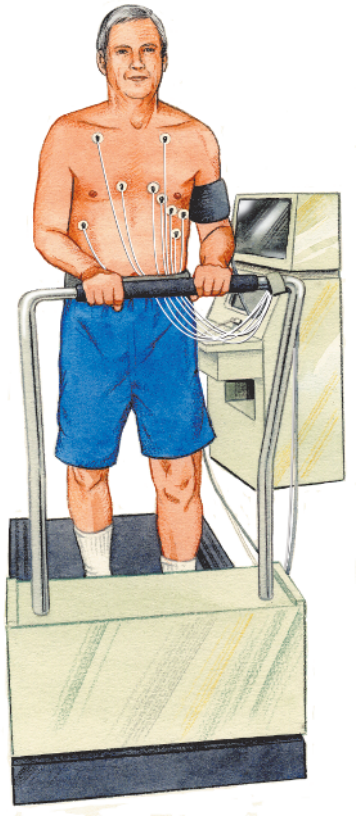


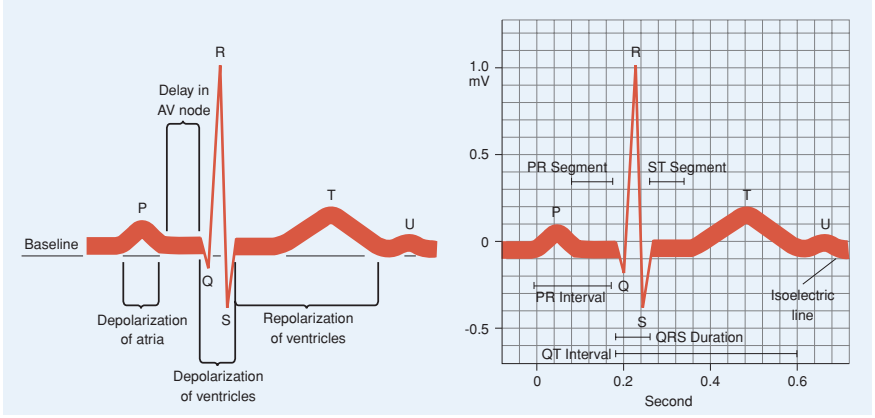


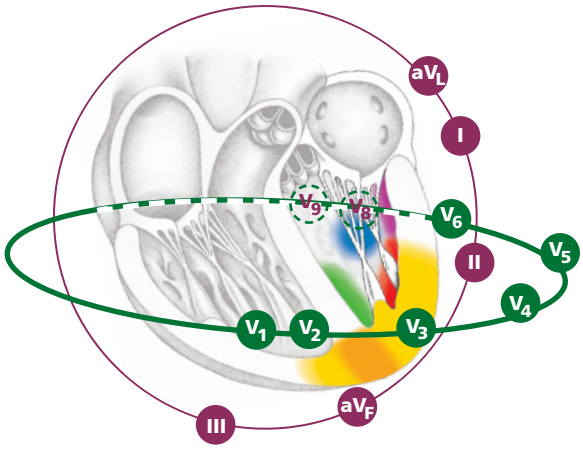
Diagram of the EKG (lead II). Illustrates the depolarization and repolarization of the atria and ventricles. The P wave represents atrial depolarization; the QRS represents complex ventricular depolarization; and the T wave represents ventricular repolarization. Atrial repolarization occurs during ventricular depolarization and is hidden under the QRS complex. (Reprinted with permission from Porth C. *Essentials of Pathophysiology*. 4th ed. Philadelphia: Wolters Kluwer; 2015.)

Drug-Induced Stress Testing

When a patient cannot tolerate physical activity, medications such as adenosine or dobutamine can be administered to cause the heart to react as if the person were exercising. The drug is given intravenously along with thallium (a radioactive substance known as tracer). The areas of the heart that lack adequate blood supply pick up the tracer very slowly, if at all. A nuclear scanner records an initial set of images and then a second set of images taken 3 to 4 hours later. A cardiologist uses these images to determine areas of heart muscle with diminished blood supply or permanent damage from MI.

Utilization of drug-induced stress testing depends on many factors, including, but not limited to:

- Ability to perform adequate exercise
- Resting ECG
- Clinical indication for performing the test
- Patient's body habitus
- History of prior coronary revascularization



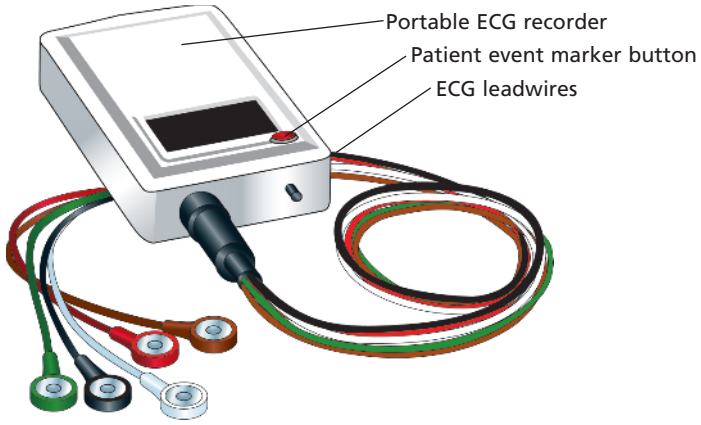
KEY:
Anteroseptal wall
Anterior wall
Lateral wall
Inferior wall
Posterior wall

Wall Affected	Leads	Artery Involved	Reciprocal Changes
Antero-septal	V_1, V_2, V_3, V_4	Left anterior descending (LAD)	None
Anterior	V_2, V_3, V_4	Left coronary artery (LCA)	II, III, aV_F
Antero-lateral	I, aV_L, V_3, V_4, V_5, V_6	LAD and diagonal branches, circumflex, and marginal branches	II, III, aV_F
Lateral	I, aV_L, V_5, V_6	Circumflex branch of LCA	II, III, aV_F
Inferior	II, III, aV_F	Right coronary artery (RCA)	I, aV_L
Posterior	V_8, V_9	RCA or circumflex	V_1, V_2, V_3, V_4 (R greater than S in V_1 and V_2, ST-segment depression, elevated T waves)

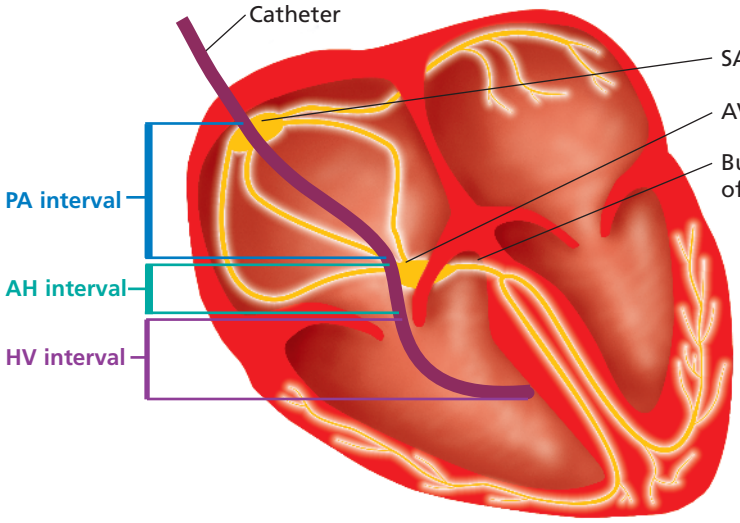
Holter Monitoring (Ambulatory ECG Monitoring)

Ambulatory ECG monitoring records the heart's activity as the patient follows his normal routine. The patient wears a small electronic recorder connected to electrodes placed on his/her chest and keeps a diary of his activities and associated symptoms. This test can be worn from 24 to 48 hours and for as long as months to years offering the ability to monitor cardiac ECG data during normal routine activity, including any physical and psychological stresses.

A Look at a Holter Monitor



Normal Conduction Intervals in Adults



PA interval = conduction from sinatrial (SA) node to atrioventricular (AV) node
= 20 to 40 msec

AH interval = conduction from AV node to bundle of His
= 4 to 150 msec

HV interval = conduction from bundle of His to ventricles
= 35 to 55 msec

ELECTROPHYSIOLOGY STUDIES

Electrophysiology studies are used to help determine the cause of an arrhythmia and the best treatment for it. A bipolar or tripolar electrode catheter is threaded into a vein, through the right atrium, and across the septal leaflet of the tricuspid valve. The femoral vein is the most common choice for the catheter insertion. However, the subclavian, internal jugular, or brachial vein may also be used. The heart's usual conduction is recorded first. The catheter sends electrical signals to the heart to change the heart rate and initiate an arrhythmia. Various drugs are then tried to terminate the arrhythmia.

Also, sometimes the cardiologist can induce an arrhythmia and then immediately treat it using radiofrequency ablation, a pacemaker, or an implantable cardioverter defibrillator (ICD).

IMAGING TESTS

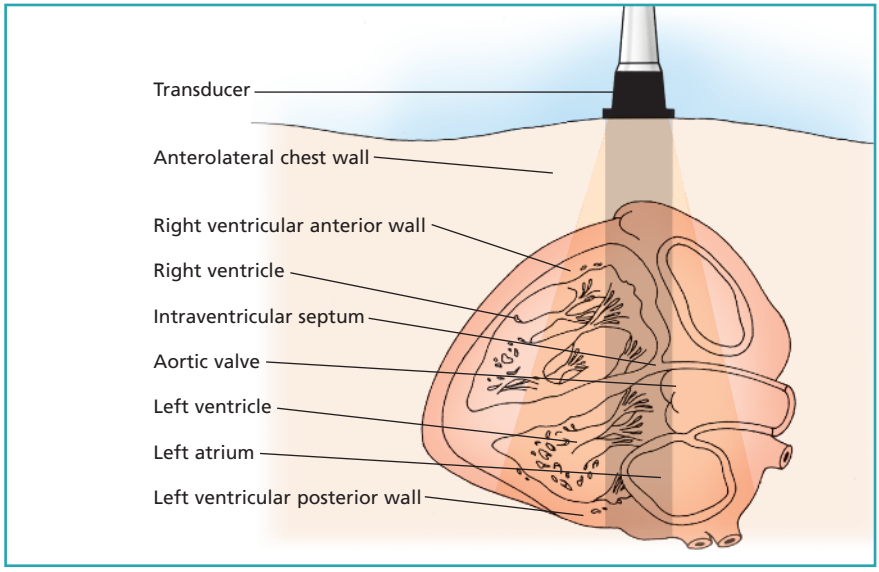
Various imaging and radiographic tests are used to help visualize heart structures and blood vessels throughout the cardiovascular system. Although many of these tests are noninvasive and quick to perform, some require the insertion of a cardiac catheter, injection of a contrast medium, or nuclear medicine to further enhance the image.

Cardiac Catheterization

Cardiac catheterization (cardiac catheterization or heart catheterization) is an invasive procedure to examine how well the heart is working. A catheter is inserted into a large blood

vessel that leads to the heart. A contrast dye visible in x-rays is injected through the catheter and flows through the heart arteries to search for narrowed or blocked coronary arteries called a coronary angiography or coronary arteriography. Cardiac catheterization is performed to:

- Identify diseases of the heart muscle, valves, or coronary (heart) arteries
- Measure the pressure in the chambers of the heart
- Measure the oxygen content in the chambers of the heart
- Evaluate the ability of the pumping chambers to contract
- Look for defects in the valves or chambers of the heart
- Myocardial biopsy

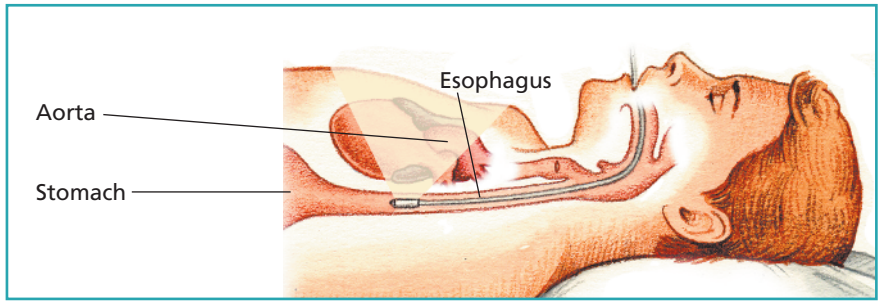


Echocardiography

Echocardiography is an excellent real-time imaging technique with a high degree of clinical accuracy. Echocardiograph uses ultra-high frequency sound waves to help examine the size, shape, and motion of the heart's structures. A special transducer is placed over the patient's chest over an area where bone and tissues are absent. It directs sound waves to the heart structures and converts them to electrical impulses. These electrical impulses are sent to the echocardiograph and displayed on a screen. The image is then recorded on a strip or videotaped.

Transesophageal Echocardiography

Transesophageal echocardiography (TEE) produces pictures of the heart using high-frequency sound waves (ultrasound). Unlike a standard echocardiogram, the echo transducer that produces the sound waves for TEE is attached to a thin tube that passes through the mouth and down the throat into the esophagus. Because the esophagus is so close to the upper chambers of the heart, very clear images of those heart structures and valves can be obtained. TEE is used when more detailed information is required than a standard echocardiogram can give them. The sound waves sent to the heart by the probe in the esophagus are translated into pictures on a video screen. TEE visualizes the heart's structure and function and provide clearer pictures of the upper chambers of the heart, and the valves between the upper and lower chambers of the heart, than standard echocardiograms.



Cardiac Magnetic Resonance Imaging

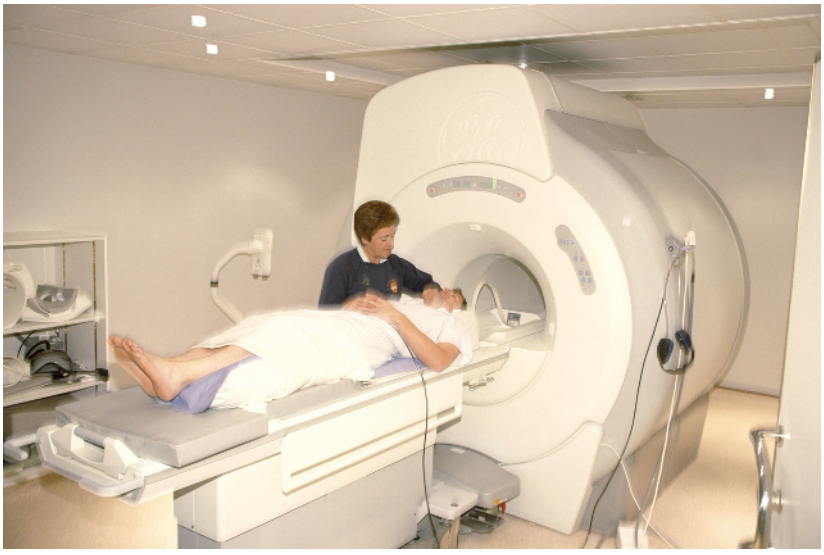
Magnetic resonance imaging (MRI) is a noninvasive test that uses a magnetic field and radiofrequency waves to create detailed pictures of organs and structures. It can be used to examine the heart and blood vessels, and to identify areas of the brain affected by stroke. MRI is also sometimes called nuclear magnetic resonance (NMR) imaging. MRI uses a powerful magnetic field, radiofrequency waves, and a computer to create detailed cross-sectional (2-dimensional) and 3-dimensional images of the inside of the body without using ionizing radiation (like x-rays, computed tomography, or nuclear imaging). This test can identify the heart's structure (muscle, valves, and chambers) and how well blood flows through the heart and major vessels. MRI can identify damage to the heart from an MI, or if there is lack of blood flow to the heart muscle because of narrowed or blocked arteries.

MRI is useful in identifying:

- Tissue damage
- Reduced blood flow in the heart muscle
- Aneurysms
- Diseases of the pericardium
- Heart muscle diseases, such as heart failure (HF) or enlargement of the heart, and tumors
- Heart valve disorders
- Congenital heart disorders
- Success of surgical repair

Individuals with any type of metal device inside the body should not have an MRI unless is certified as MRI safe. These devices include:

- Pacemakers and ICDs
- Inner ear (cochlear) implants
- Neuromuscular stimulators such as those used for pain management or muscle rehabilitation
- Implanted drug infusion pumps
- Intrauterine devices (IUDs)
- Brain aneurysm clips that are not approved for MRI
- Some dental implants (check with your dentist to make sure they are not magnetic)
- You should avoid MRI if you have metal fragments in your body. Metal fragments in the eyes can be especially dangerous because the magnet may move the metal, causing eye damage or blindness.



Multiple-Gated Acquisition Scanning

Radionuclide ventriculography (RVG, RNV) or radionuclide angiography (RNA) is often referred to as a multiple-gated acquisition (MUGA) scan. This test can view how well the heart's ventricles are pumping. During a MUGA scan, a small amount of a radioactive substance or tracer (called a radionuclide) is put into the blood which attaches to red blood cells. A gamma camera takes pictures of the heart. Pictures are taken at the same time during each heartbeat (ECG-gated) which is computer analyzed.

POSITRON EMISSION TOMOGRAPHY

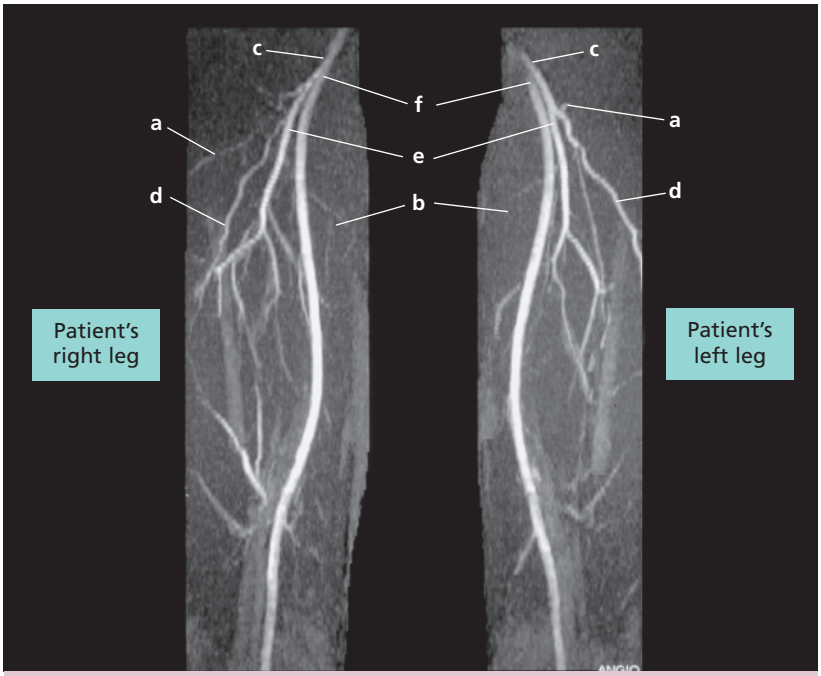
A positron emission tomography (PET) scan of the heart is a noninvasive nuclear imaging test. It uses radioactive tracers (called radionuclides) to produce pictures of the heart and diagnose coronary artery disease (CAD). PET scans can show healthy and damaged heart muscle and if there will be benefit from a percutaneous coronary intervention (PCI) such as angioplasty and stenting, coronary artery bypass surgery (CABG), or any other procedure. PET scans use radioactive material called tracers which mix with blood and are taken up by the heart muscle. A special "gamma" detector that circles the chest picks up signals from the tracer. A computer converts the signals into pictures of the heart at work. A PET scan determines if the heart is getting enough blood or if blood flow is reduced because of narrowed arteries. It also shows scaring from a prior heart attack.

A radioactive tracer is injected into the bloodstream. The tracers used for PET are mostly natural body compounds such as glucose, water, or ammonia, which are labeled or "tagged" with a small amount of radioactive material. Inside the body the radioactive tracer produces a type of energy called a gamma ray. Gamma rays are detected by a gamma detector and are used to produce a series of clear images of your heart. Images of thin slices made all the way through the heart can be produced from all different directions and angles. Computer graphics can be used to create a 3-dimensional image of your heart from the thin-slice images. Viable heart tissue will take in more of the tracer than the tissue that is no longer viable. Different colors or degrees of brightness on the PET scan show different levels of tissue function.

PERIPHERAL ARTERIOGRAPHY

A peripheral angiogram is a test that uses x-rays and dye to identify narrowed or blocked areas in one or more of the arteries that supply blood to the legs. The test is also called a peripheral arteriogram.

The angiogram helps determine if a surgical procedure is needed to open the blocked arteries. Peripheral angioplasty uses a balloon catheter to open the blocked artery from the inside. A stent, a small wire mesh tube, is generally placed in the artery after angioplasty to help keep it open. Bypass surgery is another procedure. It reroutes blood around the blocked arteries.



KEY:
a = Lateral circumflex femoral artery
b = Medial circumflex femoral artery
c = Femoral artery
d = Descending branch of the profunda femoris artery
e = Profunda femoris artery
f = Femoral artery

DOPPLER ULTRASONOGRAPHY

Duplex Doppler ultrasonography involves the use of high-frequency sound waves to image vessels and evaluate blood flow in the major vessels of the trunk (heart and intra-abdominal organs) and extremities (arms and legs) and in the extracranial cerebrovascular system (neck). This noninvasive test shows the speed, direction, and patterns of blood flow and is used to detect narrowing or blockages in arteries and veins.

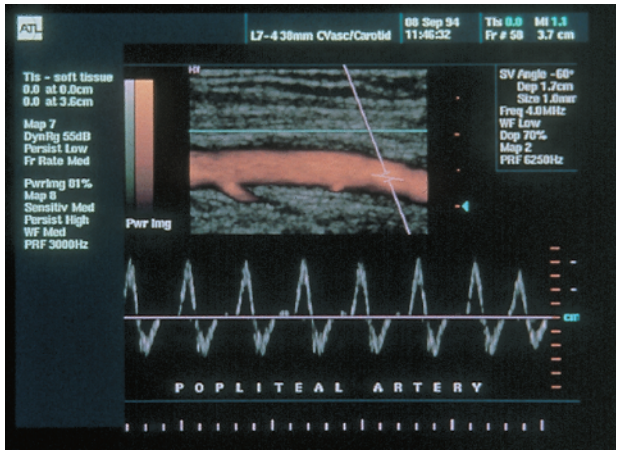
A handheld transducer directs high-frequency sound waves to the artery or vein being tested. The sound waves strike moving red blood cells and are reflected back to the transducer at frequencies that correspond to blood flow velocity through the vessel. The transducer then amplifies the sound waves to permit direct listening and graphic recording of blood flow patterns.

Pulse volume recorder testing may be performed along with duplex Doppler ultrasonography to yield a quantitative recording of changes in blood pressure in an extremity.

Normally, venous blood flow fluctuates with respiration, so observing changes in sound wave frequency during respiration helps detect venous occlusive disease. Compression maneuvers can help detect occlusion of the veins as well. Abnormal images and Doppler signals may indicate plaque, stenosis, occlusion, dissection, aneurysm, carotid body tumor, arteritis, and venous thrombosis.

Doppler of Popliteal Artery

The image at right shows a color flow duplex image of a popliteal artery with normal triphasic Doppler signal.



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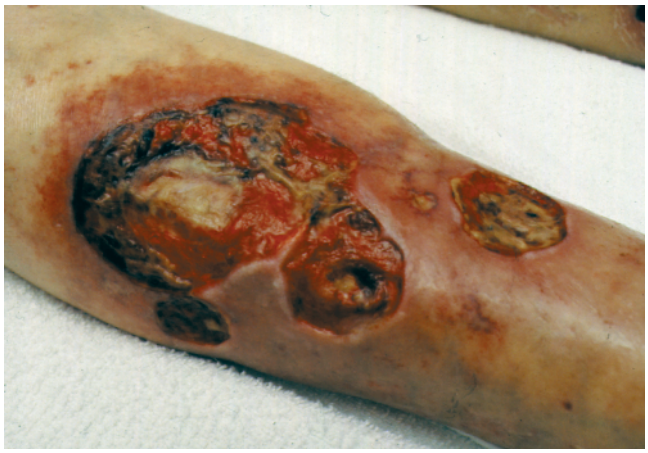
DISEASES

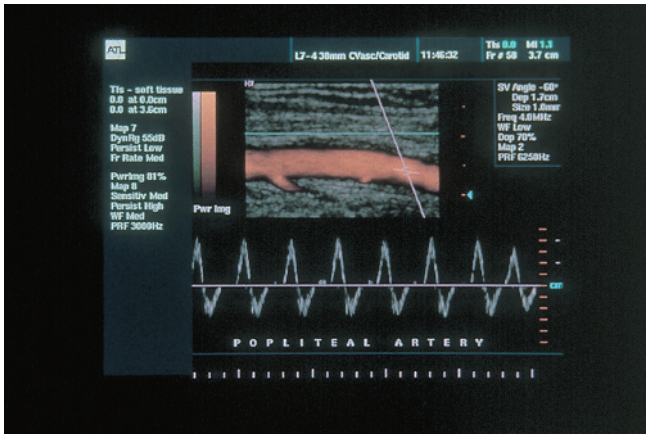
VASCULAR DISORDERS AND PROBLEMS WITH PERIPHERAL CIRCULATION

Peripheral Artery Disease

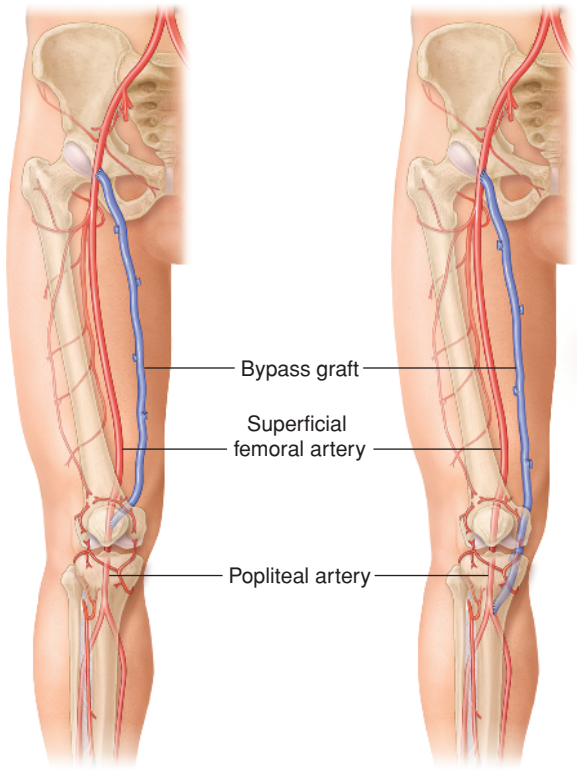
Peripheral artery disease (PAD) is used to describe the stenosis or occlusion of upper or lower extremities due to atherosclerosis or thromboembolic disease. Obstruction or narrowing of the lumen can be both asymptomatic and symptomatic. As the disease progresses and blood vessels narrow, decreased arterial flow to the lower extremities occurs resulting in symptoms that are manifested as classis intermittent claudication (IC) or atypical claudication or leg discomfort. IC is a leg muscle discomfort relieved with rest, while atypical claudication is defined as an extremity discomfort that is exertional but does not resolve with rest. PAS is systemic atherosclerotic process similar to atherosclerotic disease of the coronary vessels.

PICTURING PATHO





Color flow duplex image of popliteal artery with normal triphasic Doppler flow.



Bypass surgery provides alternate route restore adequate circulation to the distal part of the leg. (Reprinted with permission from Kupinski AM. *The Vascular System*. 2nd ed. Philadelphia: Wolters Kluwer Health; 2017.)

Risk Factors

- Male gender
- Age
- Diabetes
- Smoking
- Hypertension
- High cholesterol
- Renal insufficiency

Signs and Symptoms of Peripheral Artery Disease

- Diminished pulses
- Arterial bruits
- Decreased capillary refill
- Pallor on elevation
- Trophic changes
- Ulceration or gangrene of the toes

Treatment

- Smoking cessation
- Management of diabetes
- Statin therapy
- Antiplatelet agents
- Angiotensin converting–enzyme inhibitors
- Phosphodiesterase inhibitor
- Aortoiliac angioplasty and stenting
- Revascularization of limbs

Nursing Considerations

- Assess the legs and feet for any open areas and to report them to the healthcare practitioner.
- Position patient with feet below heart level to promote blood flow.
- Keep room temperature warm and keep patient warm, including the use of warm drinks.
- Teach to avoid caffeine, smoking, emotional stress and cold; causes vasoconstriction.

Teaching About PAD

- Teach the patient about peripheral arterial disease and to recognize acute changes in circulation, such as change in color, change in sensation, and acute pain. After surgery, teach the patient to check the pulses in his affected limb daily.
- Review a graduated exercise plan to increase walking distance over time. Refer the patient to a physical therapist as indicated.
- Teach the patient about all medications prescribed and the importance of complying with the treatment plan for existing disorders, such as hypertension and diabetes.
- Teach the patient the importance of controlling modifiable risk factors such as smoking. Refer the patient to a smoking cessation program if indicated.
- Teach the patient about atherosclerosis.
- Teach the patient the necessity of maintaining a low-fat diet, reducing weight, and maintaining a regular exercise program.
- Teach patient to recognize acute changes in circulation, such as change in color, change in sensation, and acute pain.
- Teach the patient to avoid constricting clothes such as socks.
- Teach importance of foot care.

TIP: IC or atypical claudication leg discomfort is relieved with rest.

VENOUS THROMBOSIS

Venous thrombosis is an acute condition characterized by inflammation and the formation of thrombus within a vein. In venous thrombosis, damage to the epithelial lining of the vein wall causes platelets to aggregate and releases clotting factors that cause fibrin in the blood to form a clot.

Venous thrombosis can occur within the superficial veins or the deep veins of the leg. Superficial venous thrombi typically occur in the saphenous veins in the setting of varicosities. While these thrombi can cause local congestion, swelling, pain, and tenderness, they rarely embolize. However, the local edema and impaired venous drainage to predispose the overlying skin to infections from slight trauma and to the development of varicose ulcers.

Deep venous thrombosis (DVT) is the larger leg veins at or above the knee (e.g., popliteal, femoral, and iliac veins) which are more serious because the thrombi more often embolize to the lungs and give rise to pulmonary infarction.

A venous *thromboembolus* occurs when a portion of a clot breaks off (generally from a deep vein) and travels to a distant site.

A *pulmonary embolus* (PE) occurs when a thrombus dislodges (most commonly from the leg) and travels through the venous system and through the heart, where it lodges in a branch of the pulmonary artery. Once there, the thrombus obstructs blood flow to the lung. A large PE may cause respiratory failure, right-sided HF, and death.

Virchow Triad—is a theory delineating the pathogenesis of venous thromboembolism (VTE), often called Virchow triad which proposes that VTE occurs as a result of:

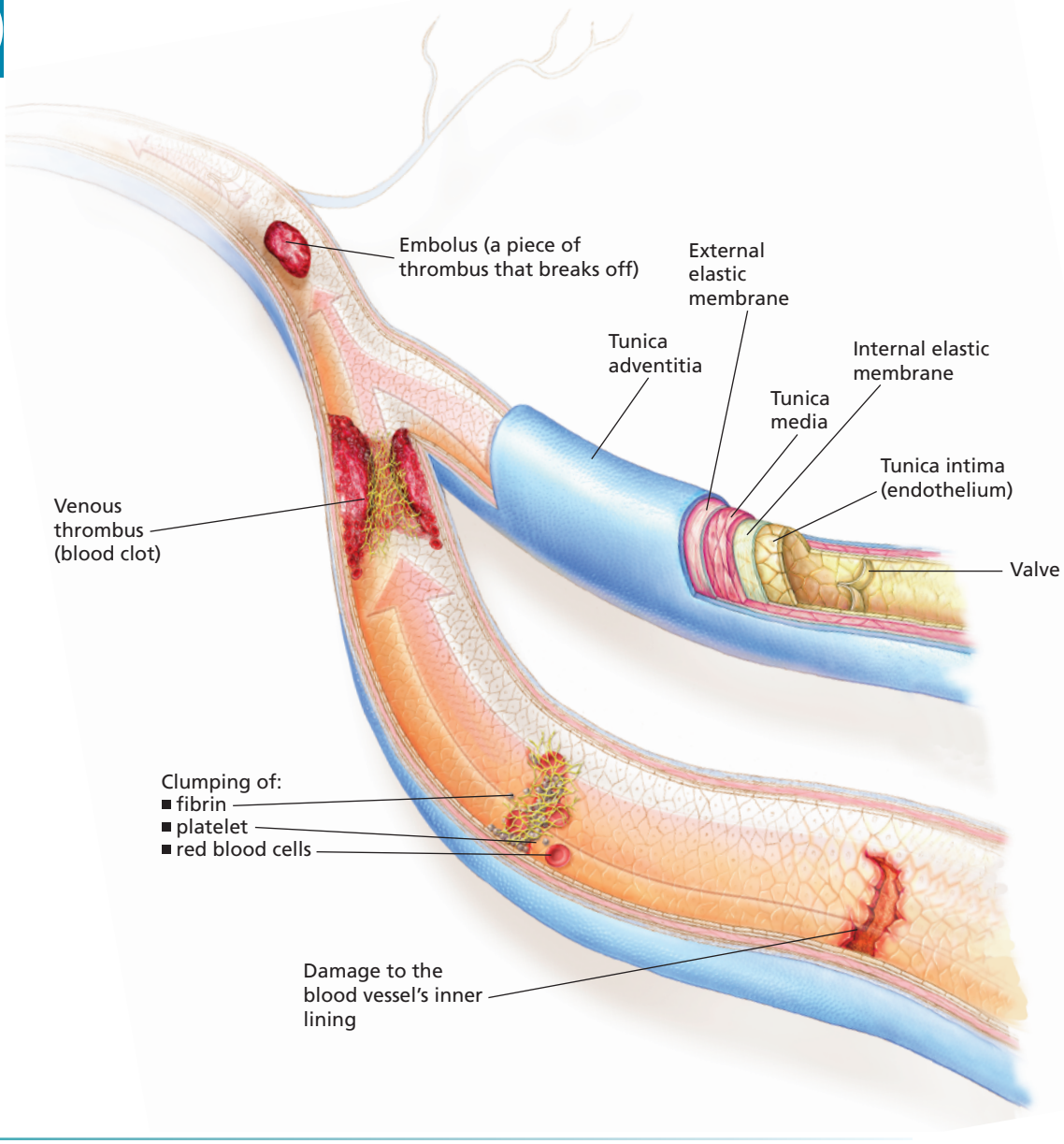
- Alterations in blood flow (i.e., stasis)
- Vascular endothelial injury
- Alterations in the constituents of the blood (i.e., inherited or acquired hypercoagulable state)

Characteristics of Patients Who Develop Venous Thromboembolism

- More than 48 hours of immobility in the preceding month
- Hospital admission in the past 3 months
- Surgery in the past 3 months
- Malignancy in the past 3 months
- Infection in the past 3 months

Looking at Venous Thrombosis

PICTURING PATHO



Signs and Symptoms

Superficial Thrombophlebitis

- Palpable induration of the affected vein
- Heat and redness along the vein
- Pain and tenderness along the vein

Deep Venous Thrombosis

- Fever, chills, and malaise
- Severe pain in the affected extremity
- Sudden nonpitting edema of the affected extremity
- Prominent superficial veins
- Erythema of the affected extremity
- Cool, pale, edematous extremity (in advanced DVT)

Treatment

- Anticoagulants, such as heparin, warfarin (Coumadin), or low-molecular-weight heparin (enoxaparin [Lovenox]) for DVT or PE
- Thrombolytics (alteplase) to dissolve the clot (in extensive PE)
- Vena cava filter to prevent PE
- Bed rest and elevation of the extremity
- Warm, moist soaks to the area
- Analgesics as needed
- Thrombectomy

Nursing Considerations

- Perform a risk assessment for DVT on admission and at each shift to direct treatment. Patients at higher risk will receive prophylactic medication such as enoxaparin; patients with a lower risk may need antiembolism or compression stockings.
- Administer anticoagulants and oxygen therapy as ordered.
- Measure the girth of the affected extremity daily to detect worsening venous outflow obstruction and possible clot extension.
- Monitor patients with a diagnosis of DVT for signs and symptoms of PE (shortness of breath, chest pain, and respiratory distress).
- Encourage ambulation when appropriate, or limb exercises for immobile patients.
- Elevate affected limb and administer analgesics, if needed. Assess for effects of treatment.
- Monitor coagulation studies for effectiveness of treatment; observe for signs and symptoms of bleeding.

Teaching About Venous Thrombosis

- Teach the patient prescribed medications, signs and symptoms that should be reported, and the importance of regular coagulation laboratory tests if taking warfarin.
- Explain all tests and treatments.
- Teach the patient to apply compression hose, elevate the affected limb, and report worsening edema, pain, or dyspnea.
- Teach the patient to exercise his limbs and minimize immobility.

HYPERTENSION

Hypertension is reported by the Eighth Joint National Committee (JNC 8), as the most common condition seen in primary care which leads to MI, stroke, renal failure, and death if not detected early and treated appropriately. Hypertension remains one of the most important preventable contributors to disease and death.

Major Risk Factors for Hypertension

- Age (older than 55 for men, 65 for women)
- Diabetes mellitus
- Elevated LDL (or total) cholesterol or low HDL cholesterol
- Estimated GFR <60 mL/min
- Family history of premature cardiovascular disease (men aged <55 or women aged <65)
- Microalbuminuria obesity (body mass index ≥30 kg/m^2)
- Physical inactivity
- Tobacco usage, particularly cigarettes

Complications of Hypertension

- Target organ damage for individuals with hypertension
  - Heart:
    - Left ventricular hypertrophy
    - Angina/prior MI
    - HF
  - Brain:
    - Stroke or transient ischemic attack
    - Dementia
- Chronic kidney disease
- Peripheral arterial disease
- Retinopathy

Classification of Blood Pressure for Adults

Category	SBP (mm Hg)		DBP (mm Hg)
Normal	<120	and	<80
Prehypertension	120–139	or	80–89
Hypertension, stage 1	140–159	or	90–99
Hypertension, stage 2	≥160	or	≥100

KEY: SBP, systolic blood pressure; DBP, diastolic blood pressure.

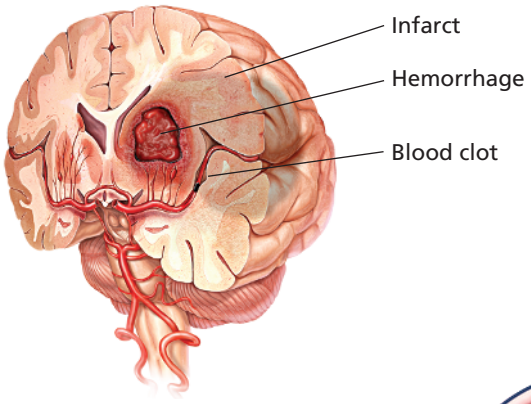
The Silent Killer

Although patients may feel healthy, untreated or poorly controlled hypertension can damage their major organs. Organs at greatest risk are the brain, eyes, and kidneys.

BRAIN

Stroke

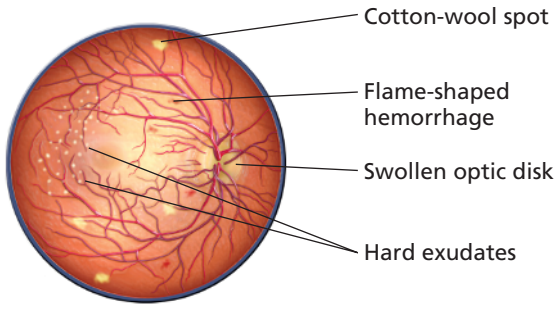
Stroke from blood clots occluding narrowed blood vessels or from hemorrhage of a weakened vessel wall (aneurysm) can be disabling or fatal.



EYE

Hypertensive retinopathy

Effects on the blood vessels within the retina can lead to hemorrhage, hard exudates, and swelling of the optic disk that may result in blindness.

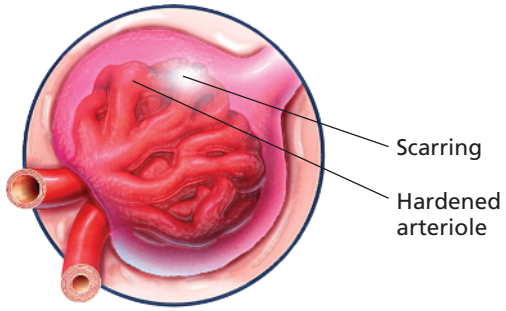


RETINA

KIDNEY

Nephrosclerosis

Arterioles in the kidney harden and restrict oxygenation of the glomeruli, causing scarring and kidney failure.



GLOMERULUS

Recommended Screening

- Adults 40 years or older should have their blood pressure measured at least annually.
- Adults between 18 and 39 years should also be screened at least annually if they have risk factors for hypertension (i.e., obesity) or if their previously measured blood pressure was 130 to 139/85 to 89 mm Hg.
- Adults between 18 and 39 years whose latest blood pressure was <130/80 mm Hg and have no risk factors for hypertension should be screened at least every 3 years.

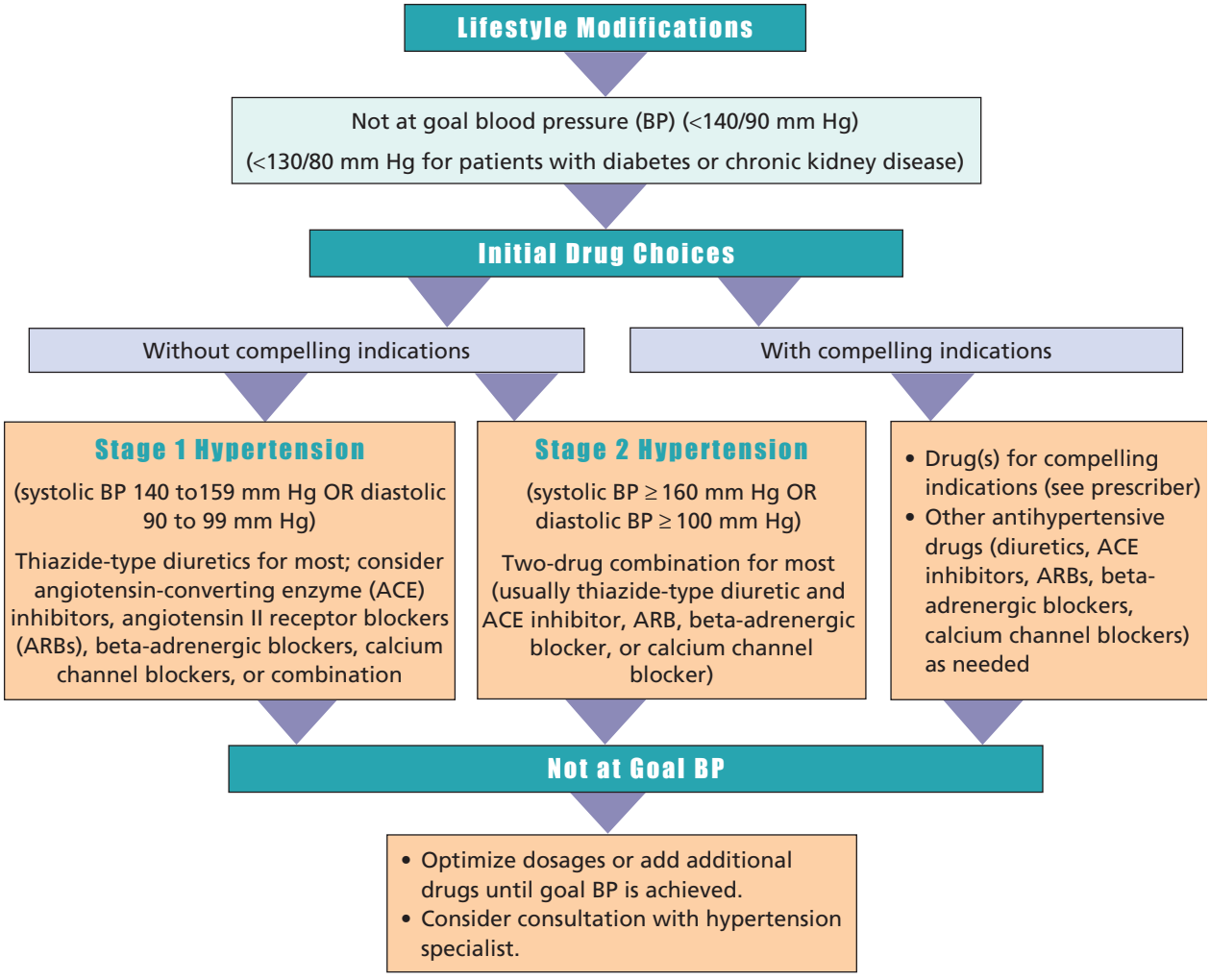
TIP: Hypertension or high blood pressure can affect the heart, brain, kidneys, eyes, and peripheral circulation.

Nursing Considerations

- When routine blood pressure screening reveals elevated pressure, make sure the sphygmomanometer cuff size is appropriate for the patient's upper arm circumference.
- Ask the patient if they smoked, had a beverage containing caffeine, or was emotionally upset before the reading.
- Advise patient to return for blood pressure testing at frequent regular intervals.

Treatment of Hypertension

The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure has developed an innovative flow chart to guide the treatment of patients with hypertension.



Source: Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. U.S. Department of Health and Human Services, NIH, NHLBI; 2004.

Teaching About Hypertension

LESSON PLANS

- Teach the patient to use a self-monitoring blood pressure cuff and to record the reading at least twice weekly in a journal for review by the physician at every office appointment.
- Tell the patient and family to keep a record of drugs used in the past, noting especially which ones are or are not effective.
- To encourage compliance with antihypertensive therapy, suggest establishing a daily routine for taking medication. Tell patient to report any adverse reactions to prescribed drugs. Advise patient to avoid high-sodium antacids and over-the-counter cold and sinus medications containing harmful vasoconstrictors.
- Help the patient examine and modify their lifestyle. Suggest stress-reduction groups, dietary changes, and an exercise program, particularly aerobic walking, to improve cardiac status and reduce obesity and serum cholesterol levels.
- Encourage a change in dietary habits. Help obese patients plan a reducing diet. Advise against intake of high-sodium foods (such as pickles, potato chips, canned soups, and cold cuts), table salt, and foods high in cholesterol and saturated fat.
- Teach the patient and family that this is a lifelong treatment. Warn the patient and family about complications that may occur from noncompliance and uncontrolled blood pressure, such as stroke and heart attack.

CORONARY VASCULAR DISORDERS

CORONARY ARTERY DISEASE OR ISCHEMIC HEART DISEASE

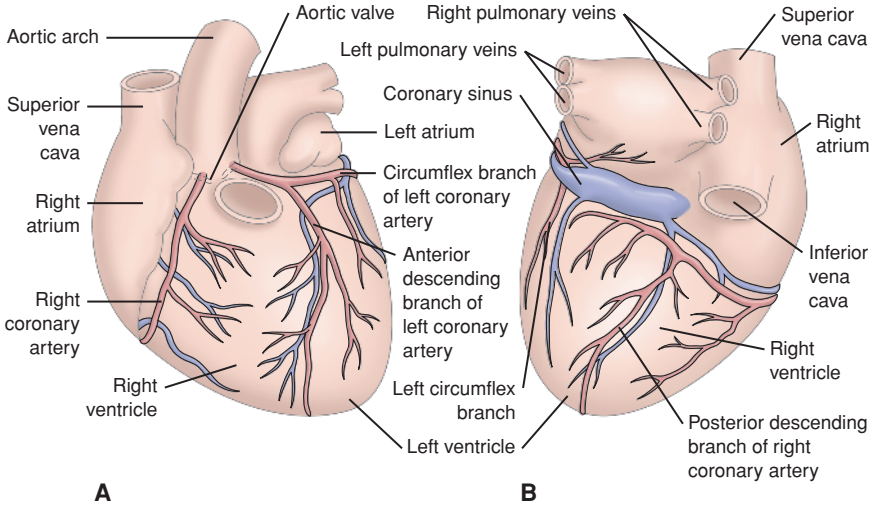
Ischemic heart disease (IHD) is often termed CAD, which is generally caused by a diminished blood flow to the myocardium and is the most common manifestation of CAD.

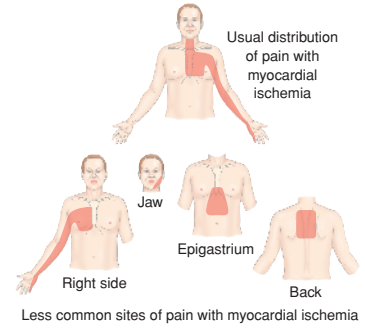
The term *angina pectoris* includes a group of symptoms attributable to myocardial ischemia. Angina pectoris which means "chest pain" is characterized by acute recurrent attacks of substernal or precordial chest discomfort. Angina is generally caused by a diminished blood flow to the myocardium and is the most common manifestation of CAD. Angina can also occur in other cardiac problems, such

Illustration of Assessment Areas of Chest Pain Based on Point of Origin

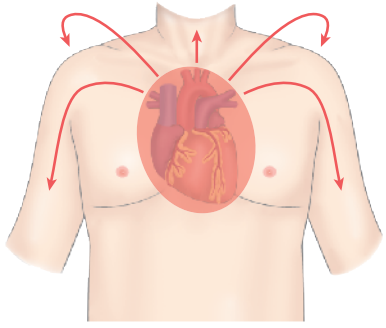
PICTURING PATHO

The coronary arteries supply the heart muscle with oxygenated blood, adjusting the flow according to metabolic needs. **A.** Anterior view of the heart. **B.** Posterior view of the heart.

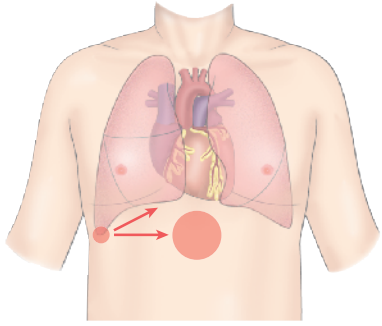




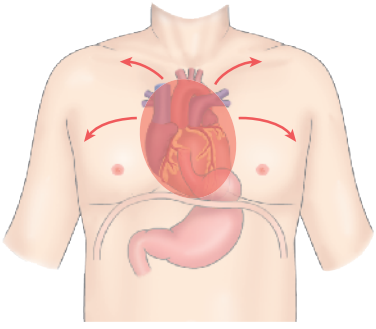
Angina Pectoris, ACS (unstable angina, MI)



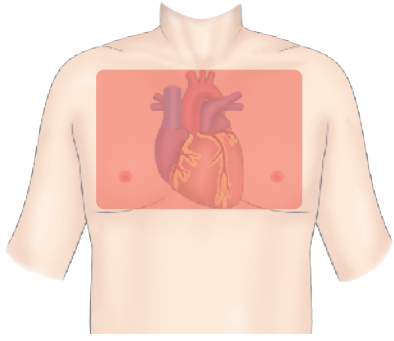
Pericarditis



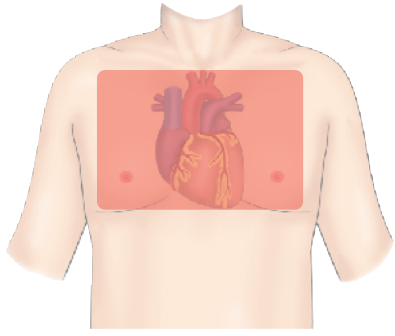
Pulmonary Disorders (pneumonia, pulmonary embolism)



Esophageal Disorders (hiatal hernia, reflux esophagitis or spasm)



Anxiety and Panic Disorders



Musculoskeletal Disorders (costochondritis

Reprinted with permission from Hinkle JL, Cheever KH. *Brunner & Suddarth's Textbook of Medical-Surgical Nursing*. 13th ed. Philadelphia: Wolters Kluwer; 2013.

as arterial spasm, aortic stenosis, cardiomyopathy, or uncontrolled hypertension. Noncardiac causes include anemia, fever, thyrotoxicosis, and anxiety/panic attacks.

Clinical Manifestations of Angina Pectoris

- Chest pain or discomfort induced by physical exertion or emotional stress which is relieved by rest and nitroglycerin
- Mild or severe pain which crescendos in discomfort and then decrescendos to relief
- Substernal chest pain, pressure, heaviness, or discomfort such as a squeezing, aching, burning, choking, strangling, and/or cramping pain
- Exertional shortness of breath
- Nausea
- Diaphoresis
- Fatigue
- Numbness or weakness in arms, wrists, or hands
- Women are more likely to have "atypical" symptoms such as dyspnea and fatigue
- Diabetics may have atypical, minimal or no symptoms

Patterns of Angina Pectoris—are caused by varying combinations of increased myocardial demand, decreased myocardial perfusion, and coronary arterial pathology identified as:

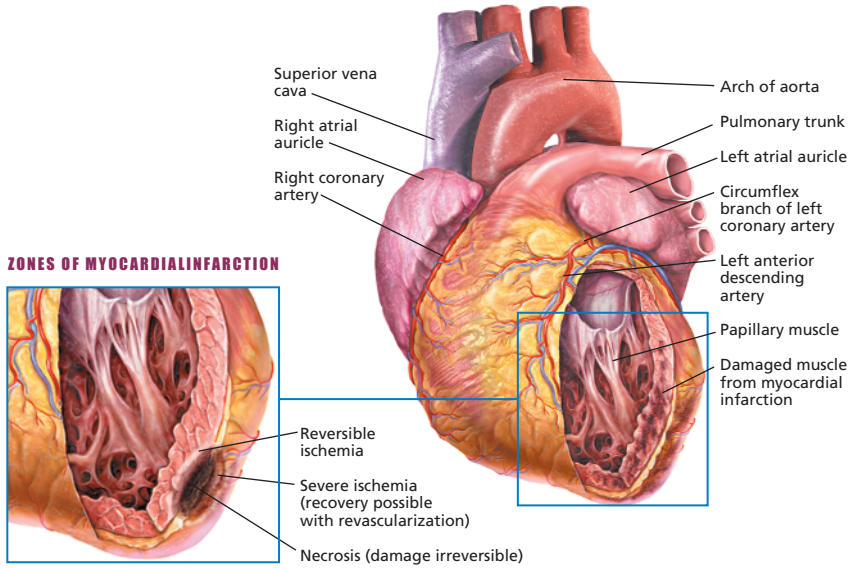
- **Stable or Typical Angina**—imbalance in coronary perfusion demand
- **Prinzmetal Variant Angina**—coronary artery spasm
- **Unstable Angina (UA)**—pattern of increasing pain, prolonged duration of pain, or pain occurring at rest

TIP: Early recognition and treatment of UA is imperative to prevent complication such as sudden death.

ACUTE CORONARY SYNDROME

Acute coronary syndrome (ACS) is a term used to describe a group of clinical symptoms which result from underlying acute myocardial ischemia. ACS includes UA, non-ST elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI), depending on the degree of coronary artery occlusion. These conditions are characterized by differences in severity, risk, etiology, pathophysiology, presentation, and management. Angina is considered unstable when a patient experiences prolonged symptoms at rest.

ACS Tissue Destruction



Clinical Manifestations of Acute Coronary Syndrome

- Chest pain
- Pressure
- Tightness or heaviness
- Pain that radiates to neck, jaw, shoulders, back, or one or both arms
- Indigestion or heartburn
- Nausea and/or vomiting associated with chest discomfort
- Persistent shortness of breath
- Weakness
- Dizziness
- Lightheadedness
- Loss of consciousness

CAUSES OF UNSTABLE ANGINA OR NSTEMI

- Thrombus or thromboembolism, usually arises from disrupted or eroded plaque
  - Occlusive thrombus, usually with collateral vessels
  - Subtotal occlusive thrombus on pre-existing plaque
  - Distal microvascular thromboembolism from plaque-associated thrombus
- Thromboembolism from plaque erosion
  - Nonplaque-associated coronary thromboembolism
  - Dynamic obstruction (coronary spasm or vasoconstriction) of epicardial and/or microvascular vessels
- Progressive mechanical obstruction to coronary flow
- Coronary arterial inflammation
- Secondary UA
- Coronary artery dissection

Treatment of UA/NSTEMI

- Oxygen
- Nitrates
- Morphine
- Beta blockers
- Heparin

- Beta blockers
- Calcium channel blockers
- ACE inhibitors
- Antiplatelet therapy
  - Aspirin
  - ADP receptor antagonists

Nursing Considerations

- During anginal episodes, monitor blood pressure and heart rate. Obtain a 12-lead ECG before administering nitroglycerin or other nitrates. Record the duration of pain, the amount of medication required to relieve it, and the accompanying symptoms.
- Instruct the patient to call whenever he feels chest, arm, or neck pain.
- Ask the patient to grade the severity of his pain on a scale of 0 to 10.
- After cardiac catheterization, review the expected course of treatment with the patient and family members. Monitor the catheter site for bleeding and check for distal pulses.
- After rotational ablation, monitor the patient for chest pain, hypotension, coronary artery spasm, and bleeding from the catheter site. Provide heparin and antibiotic therapy for 24 to 48 hours as ordered.
- After bypass surgery, monitor blood pressure, intake and output, breath sounds, chest tube drainage, and cardiac rhythm, watching for signs of ischemia and arrhythmias. Monitor capillary glucose, electrolyte levels, and arterial blood gases (ABGs). Follow weaning parameters while patient is on a mechanical ventilator. Medications such as epinephrine, nitroprusside, albumin, potassium, and blood products may be necessary. The patient may also need temporary epicardial pacing.

Teaching About CAD

- Help the patient determine which activities precipitate episodes of pain. Help patient identify and select more effective coping mechanisms to deal with stress.
- Encourage the need to follow the prescribed drug regimen.
- Discuss the need to maintain diets low in sodium and start a low-calorie diet as well.
- Explain that recurrent angina symptoms after PTCA or rotational ablation may signal reocclusion.
- Encourage regular, moderate exercise. Refer the patient to a cardiac rehabilitation center or cardiovascular fitness program near his home or workplace.
- Reassure the patient that he can resume sexual activity and that modifications can allow for sexual fulfillment without fear of overexertion, pain, or reocclusion.
- Refer the patient to a smoking cessation program.
- If the patient is scheduled for surgery, explain the procedure, when possible provide a tour of the intensive care unit, introduce patient to the staff, and discuss postoperative care.

MYOCARDIAL INFARCTION

MI is one of the manifestations of ACS commonly known as a "heart attack" resulting from death of cardiac muscle related to prolonged severe ischemia. Generally, one or more areas of the heart have prolonged decrease or cessation in oxygen supply related to insufficient coronary blood flow which results in the necrosis of myocardial tissue in the affected areas. Onset can be sudden or gradual with progression to cell death which generally occurs in 3 to 6 hours. MI results from reduced coronary blood flow:

- Most commonly, a sudden change in atheromatous plaque (interplaque hemorrhage, erosion or ulceration, rupture or fissuring).
- Vasospasm associated with platelet aggregation or cocaine abuse.
- Emboli associated with atrial fibrillation (AF), left-sided mural thrombus, vegetations of infective endocarditis (IE), intracardiac prosthetic material, or paradoxical emboli.
- Demand ischemia not due to the above mechanisms—when stress situations increase myocardial oxygen demand that cannot be met with the available blood supply.

The exact location, size, and specific morphologic features of an acute MI depend on:

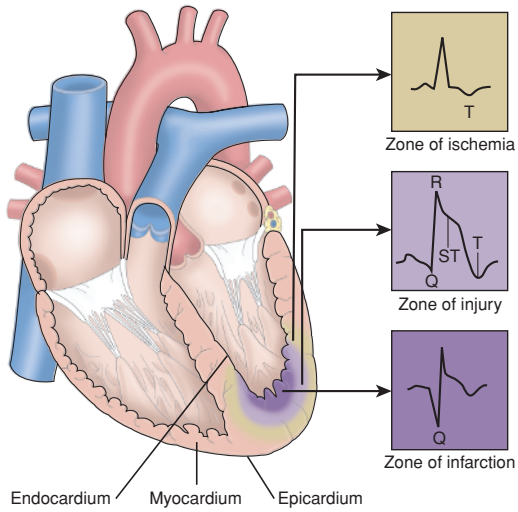
- The location, severity, and rate of development of coronary obstructions due to atherosclerosis and thrombosis
- The size of the vascular bed perfused by the obstructed vessels
- The duration of the occlusion
- The metabolic/oxygen needs of the myocardium at risk
- The extent of collateral blood supply
- The location and severity of coronary arterial spasm
- Heart rate, cardiac rhythm, and blood oxygenation

After MI, myocardial cell death can be recognized by the appearance in the blood of different proteins released into the circulation from the damaged myocytes: myoglobin, cardiac troponin T and I, CK, LDH.

TIP: Women are more likely to have "atypical" symptoms such as dyspnea and fatigue.

Effects of Ischemia, Injury and Infarction on an EKG Recording

PICTURING PATHO



Ischemia causes inversion of the T wave because of altered repolarization. Cardiac muscle injury causes elevation of the ST segment. Later Q waves develop because of the absence of the depolarization current from the necrotic tissue and opposing currents from other parts of the heart. (Reprinted with permission from Hinkle JL, Cheever KH. *Brunner & Suddarth's Textbook of Medical-Surgical Nursing*. 13th ed. Philadelphia: Wolters Kluwer; 2013.)

Comparing Signs and Symptoms of Angina and MI

	Angina	Myocardial Infarction
Character, Location, and Radiation	• Pressure, heaviness, squeezing, constriction, choking, burning, tightness (knot in throat or chest), rather than pain (sharp, stabbing, pins and needles-like).	• Severe, diffuse, steady substernal pain; described as pressure-like, squeezing, or dull.
Duration of Pain	• 2–5 min	• More than 15 min
Precipitating Events	• Elicited by activities and situations that increase myocardial oxygen demand. • Gradual in onset and offset. • Physical activity, cold, emotional stress, sexual intercourse, meals, or lying down (which results in an increase in venous return and increase in wall stress). • Once present, it may be constant and does not change with position or respiration.	• Occurs spontaneously • May be sequela to unstable angina
Relieving Measures	• Rest, nitroglycerin, oxygen may be effective.	• Not relieved by rest or nitroglycerin but requires opioids (i.e., Morphine)
Associated Symptoms	• Shortness of breath • Nausea • Diaphoresis • Belching • Nausea • Indigestion • Dizziness, • Lightheadedness, • Clamminess, • Fatigue	• Feeling of impending doom • Fatigue • Bradycardia • Tachycardia • Nausea, vomiting, hiccups • Shortness of breath • Dizziness • Palpitations • Anxiety, fear • Hypotension or hypertension • Palpable precordial pulse • Arrhythmias • Diaphoresis; cool, clammy skin; facial pallor
Cardiac Biomarkers	• Usually within normal range	• Elevated

Treatment of Myocardial Infarction

- Pain relief
- Stabilization of heart rhythm
- Revascularization of the coronary artery
- Preservation of myocardial tissue
- Reduction of cardiac workload
- Thrombolytic therapy
- PCI such as coronary angioplasty with stenting opens the narrowed or blocked segments of a coronary artery
- PCI.

Pharmacologic Therapy for Myocardial Infarction: MONA

1. M (morphine)—Relieves chest pain.
2. O (oxygen)—Increases oxygenation to ischemic heart muscle.
3. N (nitrates)—Vasodilator reduces preload by decreasing blood return to the heart and decreasing oxygen demand.
4. A (aspirin)—Immediate dosing by mouth (chewed) is recommended to halt platelet aggregation.

Nursing Assessment for Patients with Chest Pain

- Determine intensity of patient's pain by asking the patient to describe pain intensity on a scale of 0 (no pain) to 10 (worst pain).
- Access precipitating causes of pain and pain quality.
  - After eating? After certain physical activities? After emotional stress?
  - Where is the pain located? Does it radiate?
  - Is the onset sudden? Gradual?
  - How long did the pain last?
  - Is the pain steady? Consistent in quality?
  - Is the pain associated with other symptoms? Sweating? Light-headedness? Nausea? Palpitations? Shortness of breath?
  - Is there anything that makes it worse?
  - How is the pain relieved?
- Obtain a 12-lead ECG
- Observe patient for signs and symptoms, including diaphoresis, shortness of breath, protective body posture, dusky facial color, and/or changes in level of consciousness (LOC).
- Position patient in a comfortable position such as Fowler position which promotes ventilation.
- Administer oxygen, if indicated.
- Obtain BP, apical heart rate, and respiratory rate.
- Administer antiangina drug(s), as prescribed.
- Monitor for relief of pain and the duration of the angina episode.
- Monitor vital sights every 5 to 20 minutes until angina pain subsides.

TIP: Remember the acronym: MONA.

Nursing Considerations

- Monitor blood pressure, heart rate, and breath sounds.
- Observe for associated symptoms: dyspnea, nausea and vomiting, dizziness, palpitations.
- Assess pain and treat appropriately as ordered.
- Record the severity, location, type, duration, and relief of pain.
- Continuously monitor ECG rhythm strips to detect rate changes and arrhythmias; treat according to facility protocol.
- Analyze rhythm strips, and place a representative strip in the patient's chart if any new arrhythmias are identified, if chest pain occurs, or at least every shift or according to facility protocol.
- Assess for crackles, cough, tachypnea, and edema, which may indicate impending left-sided HF. Carefully monitor daily weight, intake and output, respiratory rate, serum enzyme levels, ECG readings, and blood pressure. Auscultate for adventitious breath sounds periodically (patients on bed rest typically have atelectatic crackles, which may disappear after coughing) and for S_3 or S_4 gallops.
- Provide a stool softener to prevent straining during defecation, which causes vagal stimulation and may slow heart rate.
- After thrombolytic therapy, administer continuous heparin as ordered. Monitor the partial thromboplastin time every 6 hours and monitor the patient for evidence of bleeding.
- Place patient at complete rest during anginal episodes.
- Stay with patient who is experiencing pain or appears anxious.
- Provide emotional support to help reduce stress and anxiety.
- Maintain quiet, comfortable environment.
- Administer oxygen as ordered.

Teaching About Acute Coronary Syndrome

- Explain dosages and therapy to promote compliance with the prescribed medication regimen and other treatment measures.
- Review dietary restrictions with patient and family.
- Encourage the patient to participate in a cardiac rehabilitation exercise program.
- Counsel the patient to resume sexual activity progressively.
- Advise the patient about appropriate responses to new or recurrent symptoms.
- Advise the patient to report typical or atypical chest pain.
- Stress the need to stop smoking. If necessary, refer the patient to a support group.

STRUCTURAL DISORDERS

HEART FAILURE

Heart failure (HF) is a common clinical syndrome caused by any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood. HF may be caused by disease of the myocardium, pericardium, endocardium, heart valves, vessels, or by metabolic disorders.

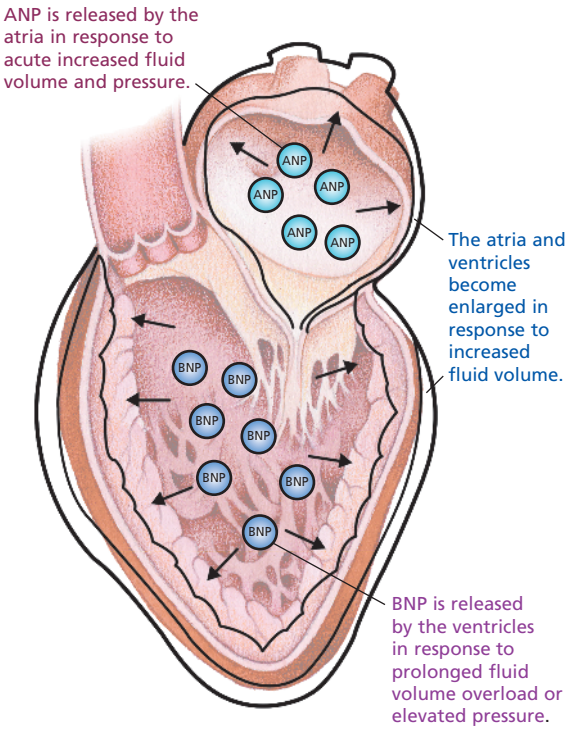
**The diagnosis of HF requires that two major or one major and two minor criteria be present concurrently. Minor criteria were acceptable only if they could not be attributed to another medical condition.*

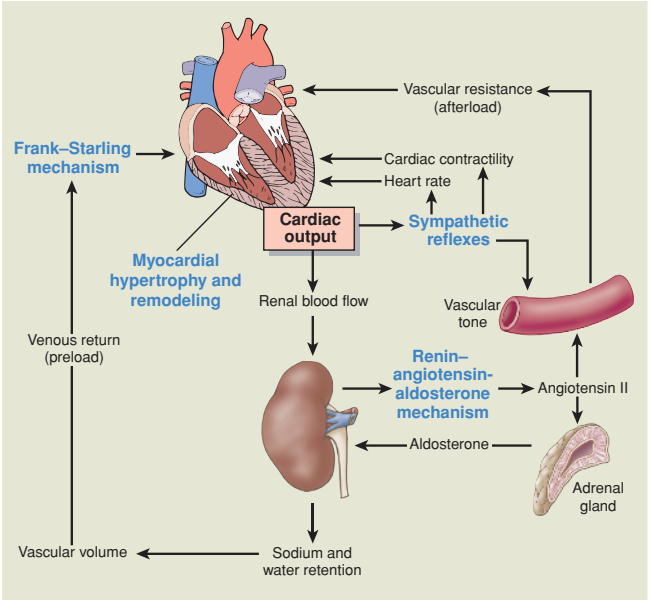
Major Criteria for Diagnosis of CHF	Minor Criteria for the Diagnosis of CHF
• Paroxysmal nocturnal dyspnea • Neck vein distension • Pulmonary rales • Cardiomegaly on chest x-ray • Acute pulmonary edema • Third sound gallop • Increased central venous pressure (>l6 cm water at the right atrium) • Hepatojugular reflux • Pulmonary edema • Visceral congestion or cardiomegaly at autopsy • Weight loss ≥4.5 kg in 5 days in response to treatment of CHF	• Bilateral ankle edema nocturnal cough • Dyspnea on ordinary exertion • Hepatomegaly • Pleural effusion • Decrease in vital capacity by 33% from maximal value recorded • Tachycardia (rate ≥l20 beats/min)

Type of HF	Causes	Morphologic Changes	Symptoms
Left Sided Heart Failure	• Ischemic Heart Disease • Hypertension • Aortic and mitral valvular diseases • Myocardial diseases	• Congestion of the pulmonary circulation • Stasis of blood in the left-sided chambers • Hypoperfusion of tissues leading to organ dysfunction	• Cough • Dyspnea (initially with exertion and later at rest) • Orthopnea • Paroxysmal nocturnal dyspnea • Feeling of suffocation • Decreased cardiac output • Decreased renal perfusion (prerenal azotemia) • Hypoxic encephalopathy
Right Sided Heart Failure	• Left sided failure • Variety of disorders that affect the lung • Cor pulmonale • Primary pulmonary hypertension • Recurrent pulmonary thromboembolism • Chronic sleep apnea • Altitude sickness	• Hypertrophy and dilation of the right side of the heart	• Minimal pulmonary congestion • Systemic and portal venous system congestion

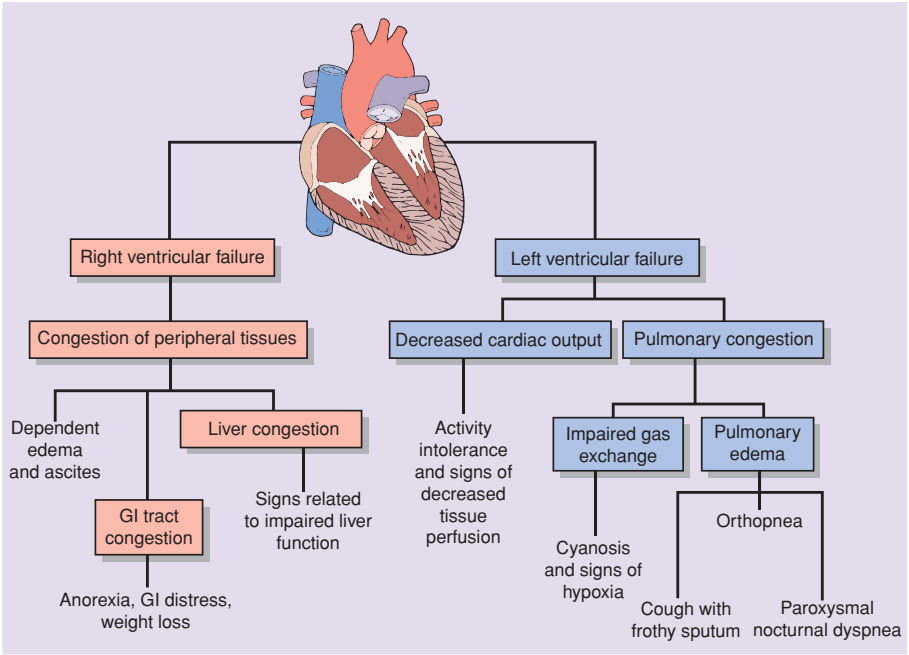
Understanding Congestive Heart Failure

PICTURING PATHO





Compensatory mechanisms in heart failure. The Frank–Starling mechanism, sympathetic reflexes, renin-angiotensin-aldosterone mechanism, and myocardial hypertrophy function in maintaining cardiac output for the failing heart. (Reprinted with permission from Porth C. *Essentials of Pathophysiology*. 4th ed. Philadelphia: Wolters Kluwer; 2015.)



Manifestations of right and left ventricular failure. (Reprinted with permission from Porth C. *Essentials of Pathophysiology*. 4th ed. Philadelphia: Wolters Kluwer; 2015.)

Treatment of Congestive Heart Failure

Control the contributing factors and associated conditions such as hypertension, myocardial ischemia or infarction, diabetes mellitus, thyroid dysfunction, and infection. In addition other treatment modalities should include lifestyle modification, pharmacologic therapy, device therapy if indicated, cardiac rehabilitation, and preventive care.

Lifestyle Modifications should Include

- Cessation of smoking
- Restriction of or abstinence from alcohol consumption
- Avoidance of illicit drugs
- Salt restriction is commonly recommended
- Fluid restriction (1.5 to 2 L/day)
- Avoidance of obesity
- Daily weight monitoring
- **Pharmacologic therapy:**
  - Improvement in symptoms can be achieved by diuretics, beta blockers, ACE inhibitors, ARBs, ARNI, hydralazine plus nitrate, digoxin, and aldosterone antagonists.
  - Prolongation of patient survival has been documented with beta blockers, ACE inhibitors, ARNI, hydralazine plus nitrate, and aldosterone antagonists. More limited evidence of survival benefit is available for diuretic therapy.
- **Tests to identify the risk of HF:**
  - **A-type natriuretic peptide (ANP):** Atrial natriuretic peptide (ANP) is predominantly secreted by the cardiac atria and is present in the plasma
  - **B-type natriuretic peptide (BNP):** BNP is a protein released from the left ventricle in response to volume expansion and pressure overload. It is the first whole blood marker for the identification of individuals with CHF. Normal value <100 pg/mL.

Teaching About Heart Failure

- Advise the patient to avoid foods high in sodium, such as canned or commercially prepared foods, to curb fluid overload.
- Stress the importance of taking prescribed medications.
- Teach the necessity of maintaining fluid restrictions.
- Encourage the patient to participate in outpatient cardiac rehabilitation.
- Stress the need for regular checkups.
- Tell the patient to notify the practitioner promptly for:
  - Irregular pulse rate or pulse rate less than 60 beats/min
  - Dizziness
  - Blurred vision
  - Shortness of breath
  - Persistent dry cough
  - Palpitations
  - Increased fatigue
  - PND
  - Swollen ankles
  - Decreased urine output
  - Rapid weight gain (3 to 5 lb [1.5 to 2.5 kg]) in 1 week.
- Discuss the importance of smoking cessation

TIP: Daily weight monitoring is recommended to detect fluid accumulation before the patient becomes symptomatic.

Nursing Considerations

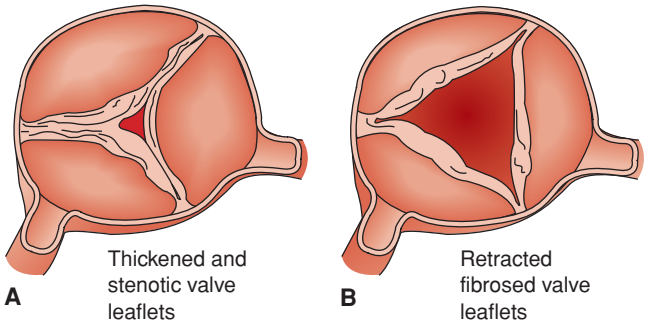
- Assess mental status (restlessness, severe anxiety, and confusion).
- Check vital signs (heart rate and blood pressure).
- Assess heart sounds, noting gallops, S3, S4.
- Assess lung sounds and determine any occurrence of paroxysmal nocturnal dyspnea (PND) or orthopnea.
- Routinely assess skin color and temperature.
- Administer medication as prescribed noting response and watching for side effects and toxicity.
- Administer stool softeners as needed (straining for a bowel movement further impairs cardiac output).
- Explain drug regimen, purpose, dose, and side effects.
- Maintain adequate ventilation and perfusion (place patient in semi- to high-Fowler position or supine position).
- Assess response to increased activity and help patient in daily activities.
- Place the patient in Fowler position and provide supplemental oxygen to facilitate breathing. Organize all activity to provide maximum rest periods.
- Weigh the patient daily (the best indicator of fluid retention), and check for peripheral edema.
- Monitor intake and urine output (especially if the patient is receiving a diuretic).
- Frequently monitor blood urea nitrogen, serum creatinine, potassium, sodium, chloride, and magnesium levels.
- Provide continuous cardiac monitoring during acute and advanced stages to identify and treat arrhythmias promptly.
- To prevent deep vein thrombosis from vascular congestion, help the patient with range-of-motion exercises and utilization of antiembolism stockings as needed. Check for calf pain and tenderness.

VALVULAR HEART DISEASE

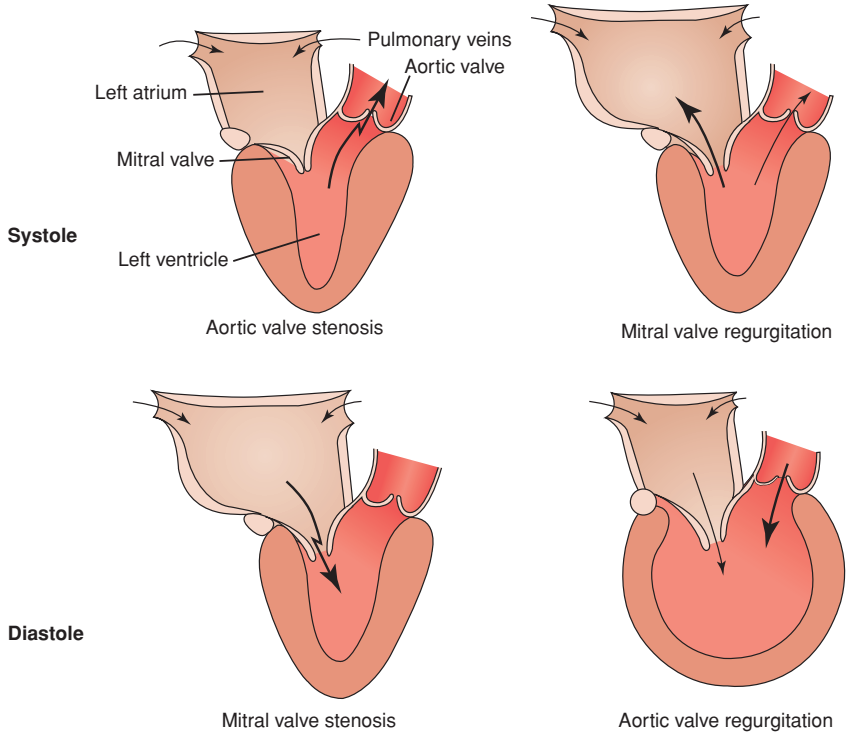
Causes of valvular heart disease results from stenosis, insufficiency (regurgitation or incompetence) or both.

- Stenosis is failure of a valve to open completely, resulting in impendence of forward flow.
- Insufficiency, results from failure of the valve to close completely, also called regurgitation, incompetence or a leaky valve.

PICTURING PATHO



Disease of the aortic valve as viewed from the aorta. **A.** Stenosis of the valve opening. **B.** An incompetent valve that is unable to close completely. (Reprinted with permission from Porth C. *Essentials of Pathophysiology*. 4th ed. Philadelphia: Wolters Kluwer; 2015.)



Alterations in hemodynamic function that accompany aortic valve stenosis, mitral valve regurgitation, mitral valve stenosis, and aortic valve regurgitation. The *thin arrows* indicate direction of normal flow and the *thick arrows* indicate the direction of abnormal flow. (Reprinted with permission from Porth C. *Essentials of Pathophysiology*. 4th ed. Philadelphia: Wolters Kluwer; 2015.)

Treatment

Treatment depends on affected valve:

Affected Valve	Causes
Aortic stenosis	Bicuspid aortic valve (or other congenital valve anomaly) Aortic valve sclerosis
Aortic regurgitation	Dilation of the ascending aorta, usually related to hypertension and age
Mitral stenosis	Rheumatic heart disease
Mitral regurgitation	Myxomatous degeneration (mitral valve prolapse)
Tricuspid regurgitation	Rheumatic heart disease Prolapse Congenital (Ebstein's) IE Radiation Carcinoid Blunt chest wall Trauma RV endomyocardial biopsy-related trauma Intra-annular RV pacemaker Implantable cardioverter-defibrillator leads
Tricuspid Stenosis	Rheumatic heart disease
Pulmonic Stenosis	Congenital disorder Less common etiologies include carcinoid and obstructing vegetations or tumors
Pulmonic regurgitation	Primary follows childhood surgery for tetralogy of Fallot or other congenital lesions that may progress insidiously

Symptoms	Treatment	LV Preload	HR	Contractility	SVR
Exertional dyspnea or decreased exercise tolerance Exertional angina Syncope Presyncope HF Angina	Management of hypertension Vasodilator therapy Surgical and transcatheter AVR Percutaneous aortic balloon dilation Statin therapy	Increase	Decrease Sinus	Maintain	Increase
Exertional dyspnea Angina Signs of heart failure	Management of hypertension Vasodilators ACE inhibitors ARBs Beta blockers	Maintain or increase	Increase	Maintain (may need support)	Decrease
Decreased exercise tolerance Exertional dyspnea	Anticoagulation therapy Heart rate control Mitral valve surgery (repair, commissurotomy, or valve replacement)	Increase	Decrease	Maintain	Maintain
Decreased exercise tolerance Exertional dyspnea LV dysfunction Pulmonary hypertension	For primary MR either surgical mitral valve repair (MVR)	Maintain	Increase	Maintain (may need support)	Decrease
Fatigue, palpitations, dyspnea, abdominal bloating, anorexia, edema, progressive hepatic dysfunction	Loop diuretics Measures to reduce pulmonary artery pressures and/or pulmonary vascular resistance Tricuspid valve surgery	Maintain or Increase	Increase	Increase	Maintain
Variable and dependent on severity of associated valve disease and degree of obstruction	Diuretics Tricuspid valve surgery	Increase	Decrease	Maintain	Maintain or Increase
None or variable and dependent on severity of obstruction	Pulmonic valve commissurotomy and valve replacement	Increase	Decrease	Maintain	Maintain
None or variable and dependent on cause of PR and RV function	Surgery when signs and symptoms of RV dysfunction occur and PR is severe	Increase	Increase	Increase	Maintain or decrease

TIP: Valvular insufficiency is also known as regurgitation, incompetence, or a leaky valve.

Nursing Considerations

- Stress the importance of adequate rest. Assist with bathing if necessary. Provide a bedside commode because using a commode puts less stress on the heart than using a bedpan. Offer the patient diversionary, physically undemanding activities.
- Alternate periods of activity with periods of rest to prevent extreme fatigue and dyspnea.
- To reduce anxiety, allow the patient to express his concerns about the effects of activity restrictions on his responsibilities and routines.
- Keep the patient's legs elevated while he sits in a chair to improve venous return to the heart.
- Place the patient in an upright position to relieve dyspnea, if necessary, and administer oxygen to prevent tissue hypoxia.
- Maintain the patient on a low-sodium diet.
- Monitor the patient for signs of HF, pulmonary edema, and adverse reactions to drug therapy.

Teaching About Valvular Disease

PICTURING PATHO

- Advise the patient to plan for periodic rest in his daily routine to prevent undue fatigue.
- Teach the patient about diet restrictions, medications, symptoms that should be reported, and the importance of consistent follow-up care.
- Tell the patient to elevate his legs whenever he sits.
- Explain all tests and treatments.
- Make sure the patient and his family understand the need to comply with prolonged antibiotic therapy and follow-up care, and the need for an additional antibiotic during dental procedures.
- Tell the patient to stop medications immediately and notify the physician if he develops a rash, fever, chills, or other signs or symptoms of allergy at any time taking antibiotics.
- Instruct the patient and his family to watch for and report early signs and symptoms of HF, such as dyspnea and a hacking, nonproductive cough.
- Teach the patient about diet restrictions, medications, symptoms that should be reported, and the importance of consistent follow-up care.
- Make sure the patient and family understand the need to comply with prolonged antibiotic therapy and follow-up care.
- Make sure the patient and family understand the need for an additional antibiotic during dental or other surgical procedures.

ABDOMINAL AORTIC ANEURYSM

Abdominal aortic aneurysm (AAA) is a dilatation of the abdominal aorta with no general agreement on how to define an AAA. Proposed definitions exist and are based on the diameter of the abdominal aorta. An AAA is classified by location, primarily with regard to its proximal extent. Factors that are important when evaluating the aortic diameter include age, gender, and body size. AAA is a common and potentially life-threatening condition.

TIP: Ruptured AAA, without repair, is nearly always fatal.

AAAs are described relative to the involvement of the renal or visceral vessels.

Type of Aneurysm	Location
Infrarenal	• Originates below the renal arteries • Most common
Juxtarenal	• Aneurysm extends to but does not involve the renal orifices
Pararenal	• Aneurysm is limited to the abdomen • Involves the visceral vessels • Involves the aorta at the level of the renal arteries • Renal artery involvement
Paravisceral	• Renal and visceral artery involvement

Sizing of Aneurysms

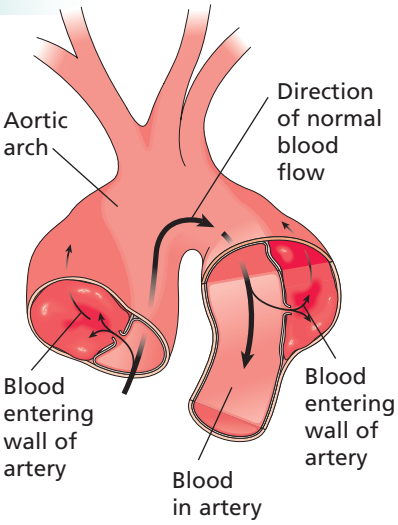
Sizing of aneurysms: >3.0 cm considered aneurysmal in most adult patients

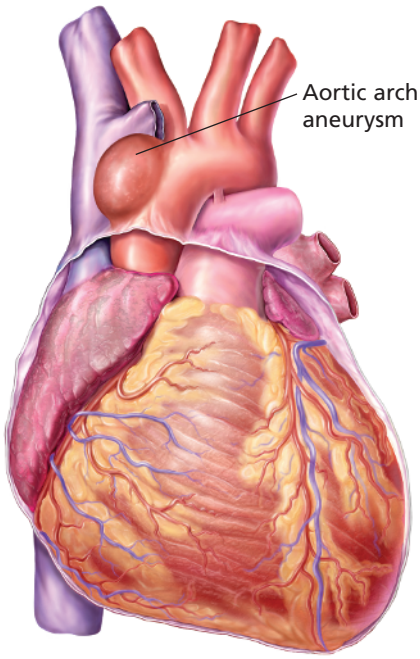
Sizing of Aneurysms	Vessel Diameter (cm)
Small	<4.0
Medium	4.0–5.5
Large	≥5.5
Very large	≥6.0

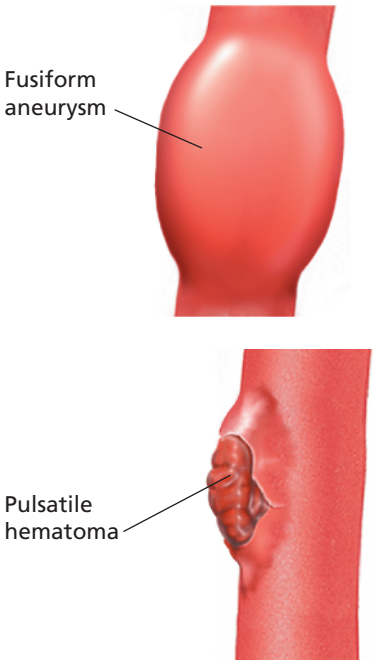
Risk Factors for AAA Development	Risk Factors for AAA Expansion	Risk Factors for AAA Rupture
• Tobacco use • Hypercholesterolemia • Hypertension • Male gender • Family history (male predominance)	• Advanced age • Severe cardiac disease • Previous stroke • Tobacco use • Cardiac or renal transplant	• Female gender • Decreased FEV1 • Aneurysm diameter and rate of growth • COPD • Higher mean blood pressure • Current tobacco use • Cardiac or renal transplant • Critical wall stress–wall strength relationship • Elevate mean arterial pressure

Determining the Types of Aortic Aneurysms

PICTURING PATHO







Treatment

- Small fusiform aneurysms, less than 4.0 cm maximum diameter, are at low risk of rupture and should be monitored
- Fusiform aneurysms greater than 5.4 cm in maximum diameter should be repaired in a healthy patient.
- Elective repair is also reasonable for patients that present with a saccular aneurysm

Clinical Manifestations

- Most AAAs do not produce any symptoms.
- Only 30% to 40% of aneurysms are noted on physical examination with detection dependent on aneurysm size.
- Detection is limited by truncal obesity.

ANEURYSMS WITH GENETIC PREDISPOSITION

Thoracic aneurysms are usually asymptomatic and detected incidentally of chest radiograph.

Clinical Manifestations

- Deep, aching back pain

Compression of respiratory structures and of the recurrent laryngeal nerve can cause:

- Dyspnea
- Hoarseness
- Coughing

TIP: Thoracic aneurysms that present as rupture as the initial manifestation is usually fatal.

Genetically mediated thoracic aortic aneurysm and dissection (TAAD) are part of a syndrome such as Marfan syndrome, Loeys–Dietz syndrome, vascular Ehlers–Danlos syndrome, Turner syndrome, or nonsyndromic, as with familial TAAD and bicuspid aortic valve. These TAAs tend to rupture at smaller aortic diameters. Decision making in patients with genetically mediated TAAD, especially in patients younger than 60 years old must consider the diameter of the ascending aorta/proximal arch but also the diameter of the aortic root and aortic valve function.

Ehlers–Danlos syndrome is an inherited connective tissue disorder known for hyperextensibility of skin, hypermobility of joints, easy bruising, and arterial aneurysms. The most common cause of death in patients with Ehlers–Danlos syndrome is arterial rupture.

Marfan syndrome is an autosomal dominant inherited disorder affecting connective tissue. Although the majority of patients with Marfan syndrome have a family history of the disease, approximately 25% of patients represent sporadic mutations. Characteristics of Marfan syndrome involve the cardiovascular, ocular, and skeletal systems. Common presentation includes aortic aneurysms and dissection, mitral valve prolapse and regurgitation, ectopia lentis, myopia, chest-wall deformities, joint laxity, and long arms and fingers.

Loeys–Dietz syndrome is an autosomal dominant aortic aneurysm syndrome characterized by the triad of arterial tortuosity and aneurysms, hypertelorism, and bifid uvula or cleft palate. Loeys–Dietz syndrome is caused by heterozygous mutations in the genes and have a high risk of aortic dissection or rupture at an early age. Mean age at death is 26 years, with thoracic aortic dissection as the leading cause of death.

Nursing Considerations

- Allow the patient to express his fears and concerns. Help him identify effective coping strategies as he attempts to deal with his diagnosis.
- Before elective surgery, weigh the patient, insert an indwelling urinary catheter and an IV line, and assist with insertion of the arterial line and pulmonary artery catheter to monitor hemodynamic balance.

In an Acute Situation

- Obtain multiple large-bore intravenous access to facilitate blood replacement.
- Prepare the patient for impending surgery.
- As ordered, obtain blood samples for kidney function tests (such as blood urea nitrogen, creatinine, and electrolyte levels), a complete blood count with differential, blood typing and cross-matching, and ABG levels.
- Monitor the patient's cardiac rhythm and vital signs.
- Assist with insertion of a pulmonary artery line and arterial line to monitor for hemodynamic status.
- Administer ordered medications, such as an antihypertensive and a beta-adrenergic blocker to control aneurysm progression and an analgesic to relieve pain.
- Be alert for signs of rupture, which may be fatal. Watch closely for any signs of acute blood loss (such as decreasing blood pressure, increasing pulse and respiratory rates, restlessness, decreased sensorium, and cool, clammy skin).
- If rupture occurs, transport the patient to surgery as soon as possible. Medical anti-shock trousers may be used while transporting to surgery.

Teaching About Aortic Aneurysm

- Explain the surgical procedure and the expected postoperative care in the intensive care unit for patients undergoing open, complex abdominal surgery (intravenous access, central line access, endotracheal intubation, and mechanical ventilation).
- Instruct the patient to take all medications as prescribed and to carry a list of medications at all times, in case of an emergency.
- Tell the patient not to push, pull, or lift heavy objects until the physician indicates that it is okay to do so.

TIP: Aneurysm size remains the most important predictor of aneurysm rupture.

INFECTIOUS/INFLAMMATORY DISORDERS

PERICARDIAL DISEASES

The pericardial space normally contains 50 mL of thin, clear, straw colored fluid in the pericardial sac, which lubricates the layers of the heart and reduces friction when the heart contracts.

Pericarditis is an inflammation of the pericardium, the membranous sac enveloping the heart. Pericarditis is the most common disease of the pericardium encountered in clinical practice. Diseases of the pericardium present clinically in one of several ways:

- Acute, subacute pericarditis
- Chronic, recurrent pericarditis
- Pericardial effusion
- Cardiac tamponade
- Constrictive pericarditis
- Effusive-constrictive pericarditis
- Cardiac masses

Clinical Manifestations of Pericarditis

- Pleuritic chest pain
- Pericardial rubs
- Widespread ST-segment elevation
- Pericardial effusion

Treatment of Pericarditis

- Targeted at the specific cause as much as possible.
- Aspirin or a nonsteroidal anti-inflammatory drug (NSAID) should be considered as first-line therapy in most cases.

PERICARDIAL EFFUSION

Pericardial effusion is an abnormal accumulation of fluid in the pericardial cavity. Since there is limited space in the pericardial cavity, fluid accumulation leads to an increased pressure which can negatively affect heart function. Pericardial disorders include any process that inflames, injures, or reduces lymphatic drainage of the pericardium.

The prognosis for individuals with pericardial effusion depends on the cause and size.

Pericardial tissue damaged by bacteria or other substances releases chemical mediators of inflammation into the surrounding tissue.

↓

Friction occurs as the inflamed pericardial layers rub against each other.

↓

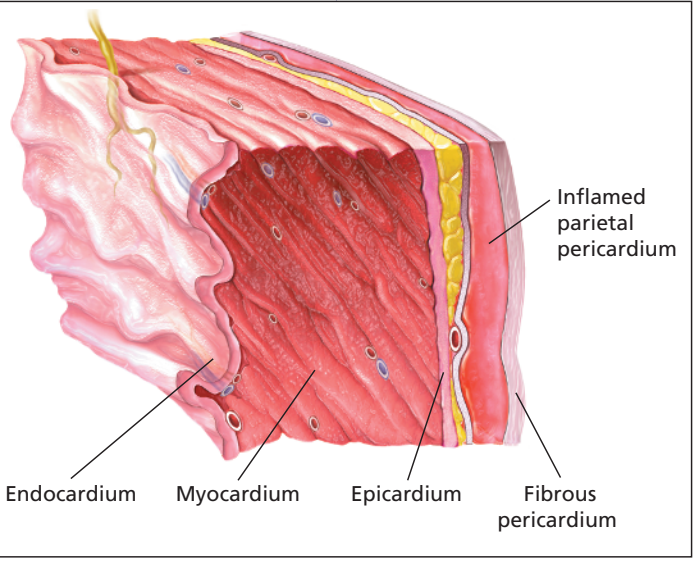
Histamines and other chemical mediators dilate vessels and increase vessel permeability.

↓

Fluids and proteins (including fibrinogen) leak into the tissue causing extracellular edema.
Macrophages, neutrophils, and monocytes in the tissue begin to phagocytose the invading bacteria.

↓

Gradually, the space fills with an exudate composed of necrotic tissue, dead neutrophils, and macrophages.
These products are eventually absorbed into healthy tissue.



Clinical Manifestations

- Chest pain—Typically sharp and pleuritic, improved by sitting up and leaning forward
- Pericardial friction rub
- ECG changes—New widespread ST elevation or PR depression
- Pericardial effusion

Nursing Considerations

- Stress the importance of adequate rest and rest periods in between activities of daily living.
- To reduce anxiety, allow the patient to express his concerns about the effects of activity

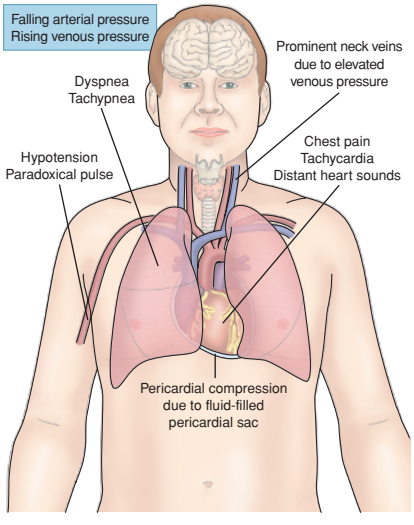
Causes of Pericardial Disease

Idiopathic (presumed to be viral, postviral, or immune-mediated)	In most case series, the majority of patients are not found to have an identifiable cause of pericardial disease. Frequently such cases are presumed to have a viral or autoimmune etiology.
Infectious	Viral (influenza, coxsackie virus, HIV), bacterial (staphylococcus, meningococcus, streptococcus, pneumococcus, gonococcus, Mycobacterium tuberculosis), fungal, parasitic, infective endocarditis with valve ring abscess
Noninfectious	Autoimmune and autoinflammatory • Systemic inflammatory diseases, especially lupus, rheumatoid arthritis, scleroderma, Sjögren syndrome, vasculitis, mixed connective disease • Autoinflammatory diseases (especially familial Mediterranean fever and tumor necrosis factor associated periodic syndrome [TRAPS]) • Postcardiac injury syndromes (immune-mediated after cardiac trauma in predisposed individuals) • Other—Granulomatosis with polyangiitis (Wegener's), polyarteritis nodosa, sarcoidosis, inflammatory bowel disease (Crohn's, ulcerative colitis), Whipple's, giant cell arteritis, Behçet disease, rheumatic fever
Neoplasm	• Metastatic—Lung or breast cancer, Hodgkins disease, leukemia, melanoma • Primary—Rhabdomyosarcoma, teratoma, fibroma, lipoma, leiomyoma, angioma • Paraneoplastic
Cardiac	• Early infarction pericarditis • MI; early, 24–72 hr; late postcardiac injury syndrome (Dressler syndrome), also seen in other settings (e.g., postmyocardial infarction and postcardiac surgery) • Myocarditis • Dissecting aortic aneurysm • Following cardiac surgery
Other	Trauma Metabolic Radiation

restrictions on his responsibilities and routines.
- Monitor pain.
- Provide an analgesic to relieve pain and oxygen to prevent tissue hypoxia.
- Before giving an antibiotic, obtain a patient history of allergies. Administer the prescribed antibiotic on time to maintain a consistent drug level in the blood.
- Assess cardiovascular status frequently, and observe for signs and symptoms of left-sided HF, such as dyspnea, hypotension, tachycardia, tachypnea, crackles, and weight gain. Check for changes in cardiac rhythm or conduction.
- Administer oxygen and evaluate ABG levels, as needed, to ensure adequate oxygenation.
- Assess cardiovascular status frequently, watching for signs of cardiac tamponade.
- Place the patient in an upright position to relieve dyspnea and chest pain.
- Monitor the patient's renal status (including blood urea nitrogen levels, creatinine clearance, and urine output) to check for signs of renal emboli and drug toxicity.
- Observe for progression to cardiac tamponade.

Pericardial Effusion

PICTURING PATHO



Assessment findings in cardiac tamponade resulting from pericardial effusion include chest pain or fullness, dyspnea, tachycardia, jugular vein distention, hypotension, paradoxical pulse, tachycardia, and distant heart sounds. (Reprinted with permission from Hinkle JL, Cheever KH. *Brunner & Suddarth's Textbook of Medical-Surgical Nursing*. 13th ed. Philadelphia: Wolters Kluwer; 2013.)

CARDIAC TAMPONADE

Cardiac tamponade is a life-threatening compression of the heart due to the pericardial accumulation of fluids as a result of inflammation, infection, trauma, and rupture of the heart or aortic dissection. Common causes of cardiac tamponade include:

- Viral pericarditis and effusion
- Ascending aortic dissection rupturing into the pericardium
- LV free wall rupture after a large anterior MI
- Trauma
- Neoplasm/malignancy
- Iatrogenic (invasive procedure-related, postcardiac surgery)

Uncommon causes of cardiac tamponade:

- Collagen vascular diseases (systemic lupus erythematosus, rheumatoid arthritis, scleroderma)
- Radiation induced
- Uremia
- Bacterial infection
- Tuberculosis
- Pneumopericardium

Clinical Manifestations

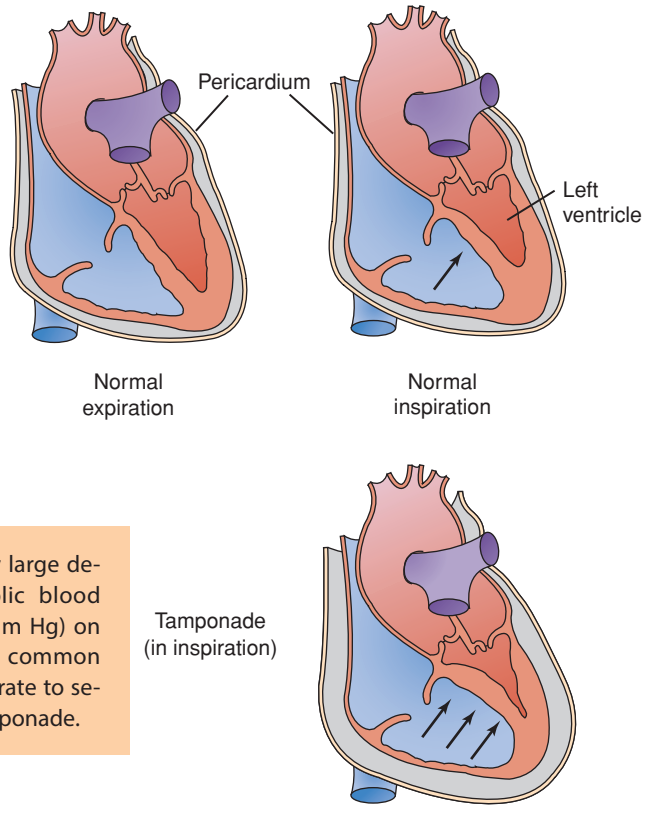
- Tachycardia
- Hypotension
- Pulsus paradoxus
- Raised jugular venous pressure
- Muffled heart sounds
- Decreased electrocardiographic voltage with electrical alternans
- Enlarged cardiac silhouette on chest x-ray
- Pain in anterior chest, worsened by motion, may vary from mild to sharp and severe chest. Generally located in precordial area and may be relieved by leaning forward

Treatment

- Removal of the pericardial fluid (pericardiocentesis)

Effects of Respiration and Cardiac Tamponade on Ventricular Filling and Cardiac Output

PICTURING PATHO



TIP: Abnormally large decrease in systolic blood pressure (>10 mm Hg) on inspiration is a common finding in moderate to severe cardiac tamponade.

Effects of respiration and cardiac tamponade on ventricular filling and cardiac output. During inspiration, venous flow into the right heart increases, causing the interventricular septum to bulge into the left ventricle. This produces a decrease in left ventricular volume, with a subsequent decrease in stroke volume output. In cardiac tamponade, the fluid in the pericardial sac produces further compression of the left ventricle, causing an exaggeration of the normal inspiratory decrease in stroke volume and systolic blood pressure. (Reprinted with permission from Porth C. *Essentials of Pathophysiology*. 4th ed. Philadelphia: Wolters Kluwer; 2015.)

INFECTIVE ENDOCARDITIS

IE refers to infection of the endocardial surface of the heart; infection of one or more heart valves, or infection of a cardiac device.

Risk Factors

- History of prior IE
- Presence of a prosthetic valve or cardiac device
- History of valvular or congenital heart disease (CHD)

Noncardiac Factors

- Intravenous drug use
- Intravenous catheter
- Immunosuppression, or a recent dental or surgical procedure
- Age >60, and men

Population at Risk for Infective Endocarditis

Patients with:

- Prosthetic valve or with prosthetic material used for cardiac valve repair
- Transcatheter-implanted prostheses and homografts
- Previous IE
- Untreated cyanotic CHD and those with CHD who have postoperative palliative shunts, conduits or other procedures

Clinical Manifestations

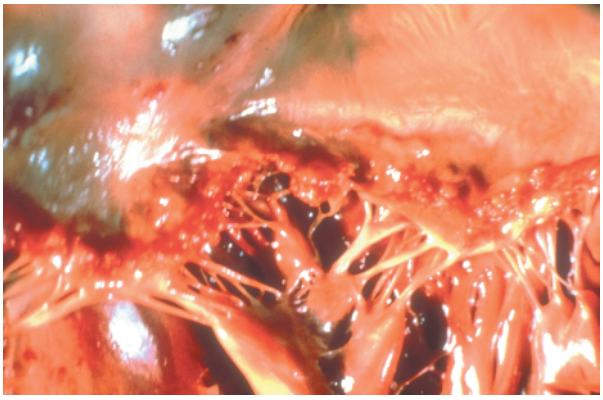
- Malaise
- Headache
- Myalgia
- Arthralgia
- Night sweats
- Loss of weight
- Flu-like syndromes
- Abdominal pain
- Dyspnea
- Cough
- Splinter hemorrhages
- Cardiac murmurs
- Petechiae
- Pleuritic pain
- Low-grade fever and nonspecific symptoms

Treatment

- Bactericidal agents
- Corrective surgery if refractory HF develops or heart structures are damaged
- Replacement of infected prosthetic valve

Libman–Sacks Endocarditis

PICTURING PATHO



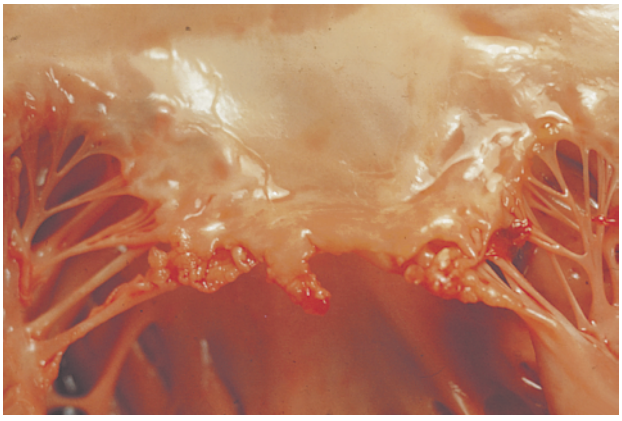
From Rubin E, Farber JL. *Pathology*. 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 1999.

Nursing Considerations

- Watch for signs and symptoms of embolization such as hematuria, pleuritic chest pain, left upper quadrant pain, and paresis.
- Monitor the patient's renal status including blood urea nitrogen levels, creatinine clearance levels, and urine output.
- Assess cardiovascular status frequently and watch for signs of left ventricular failure such as dyspnea, hypotension, tachycardia, tachypnea, crackles, neck vein distention, edema, and weight gain.
- Check for changes in cardiac rhythm or conduction.
- Evaluate ABG values as needed to ensure adequate oxygenation.
- Observe for signs of infiltration or inflammation at the venipuncture site.
- Stress the importance of taking the medication and restricting activities for as long as the doctor orders.
- Tell patient to watch closely for fever, anorexia, and other signs of relapse for about 2 weeks after treatment stops.
- Teach the patient how to recognize symptoms of endocarditis, and tell him to notify the doctor immediately if such symptoms occur.
- Stress the importance of dental hygiene to prevent caries and possible recurrent endocarditis.
- Before giving an antibiotic, obtain a patient history of allergies. Administer the prescribed antibiotic on time to maintain a consistent drug level in the blood.

Bacterial Endocarditis

PICTURING PATHO



From Rubin E, Farber JL. *Pathology*. 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 1999.

Teaching About Endocarditis

LESSON PLANS

- Teach the patient about the prescribed antibiotics. Stress the importance of taking prescribed medication and restricting activities for as long as the physician orders.
- Teach the patient to watch closely for fever, anorexia, and other signs and symptoms of relapse after treatment stops.
- Make sure the patient understands the need for a prophylactic antibiotic before, during, and after dental work, childbirth, and genitourinary, GI, or gynecologic procedures.
- Teach the patient to brush their teeth with a soft toothbrush, rinse mouth thoroughly, and avoid flossing teeth.
- Teach the patient how to recognize symptoms of endocarditis, and to notify the physician immediately if such symptoms occur.

TIP: Antimicrobial therapy for IE should be dosed to optimize sustained bactericidal serum concentrations throughout the dosing interval as much as possible.

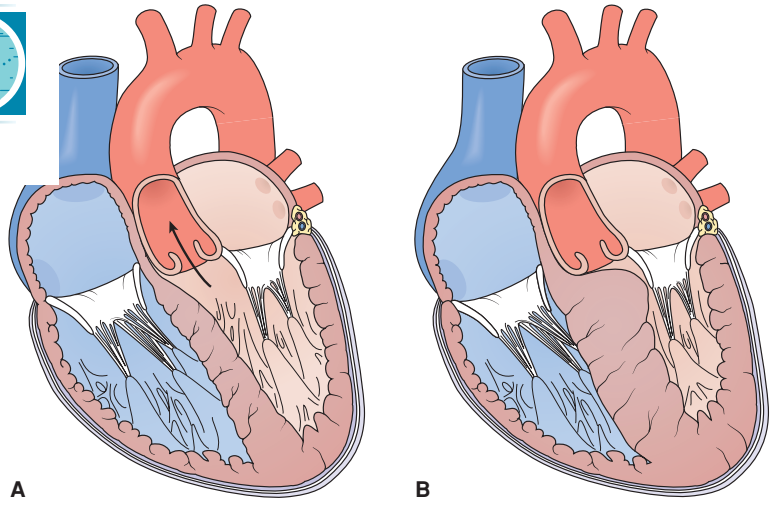
CARDIOMYOPATHY

Cardiomyopathy is a term to describe heart disease which results from an abnormality in the myocardium. Diseases of the myocardium produce abnormalities in cardiac wall thickness, chamber size, mechanical and/or electrical dysfunction, often associated with significant morbidity and mortality.

Nursing Considerations

- Alternate periods of rest with required activities of daily living and treatments. Provide personal care as needed to prevent fatigue.
- Provide active or passive range of motion exercises to prevent muscle atrophy.
- Explain the necessity of maintaining a low-sodium diet.

PICTURING PATHO



Normal heart (**A**) and hypertrophic cardiomyopathy (**B**) in which disproportionate thickening of the intraventricular septum causes intermittent left ventricular outflow obstruction. (Reprinted with permission from Porth C. *Essentials of Pathophysiology*. 4th ed. Philadelphia: Wolters Kluwer; 2015.)

Differences in Cardiomyopathies

Cardiomyopathy Type	Mechanism of Heart Failure	Characteristics	Causes of Phenotype	Treatment	Clinical Manifestations
Dilated (DCM)	Impairment of contractility (systolic dysfunction)	Dilation and impaired contraction of one or both ventricles. Dilation often becomes severe and is invariably accompanied by an increase in total cardiac mass (hypertrophy)	Myocardial infarctions, Genetic; peripartum; myocarditis; hemochromatosis; chronic anemia; toxicities (including adverse effects of chemotherapeutic agents such as doxorubicin and Adriamycin); chronic alcoholism; sarcoidosis; idiopathic	Supplemental oxygen and assisted ventilation as needed Optimizing hemodynamics Relief of symptoms Arrhythmia management Device therapy Antithrombotic therapy	Signs and symptoms of CHF (shortness of breath, easy fatigability, and poor exertional capacity) Myocardial ischemia Stroke Sudden death Dysrhythmias Cardiac murmur
Hypertrophic (HCM)	Impairment of compliance (diastolic dysfunction); intermittent ventricular outflow obstruction.		Genetic; Friedreich ataxia; storage diseases; infants of diabetic mother		
Restrictive (RCM)	Impairment of compliance (diastolic dysfunction)	Nondilated ventricles with impaired ventricular filling	Amyloidosis; radiation-induced; idiopathic		
Arrhythmogenic (ARVC)	Rhythm disturbances; ventricular tachycardia or fibrillation	Right ventricular failure Sudden death in primarily young people	Inherited		

- Monitor the patient for signs of progressive failure (decreased arterial pulses, increased jugular vein distention) and compromised renal perfusion (oliguria, increased blood urea nitrogen and serum creatinine levels, and electrolyte imbalances). Weigh the patient daily.
- Administer oxygen as needed.
- If the patient is receiving a diuretic, monitor him for signs of resolving congestion (decreased crackles and dyspnea) or too vigorous diuresis. Monitor serum potassium level for hypokalemia, especially if therapy includes a cardiac glycoside.
- Allow the patient and his family to express their fears and concerns.
- Prevent constipation and stress ulcers to reduce cardiac workload.

Teaching About Cardiomyopathy

- Before discharge, teach the patient about the illness and its treatment.
- Emphasize the need to restrict fluid and sodium intake and monitor for weight gain.
- Encourage family members to learn cardiopulmonary resuscitation because sudden cardiac arrest is possible.

ARRHYTHMIA DISORDERS

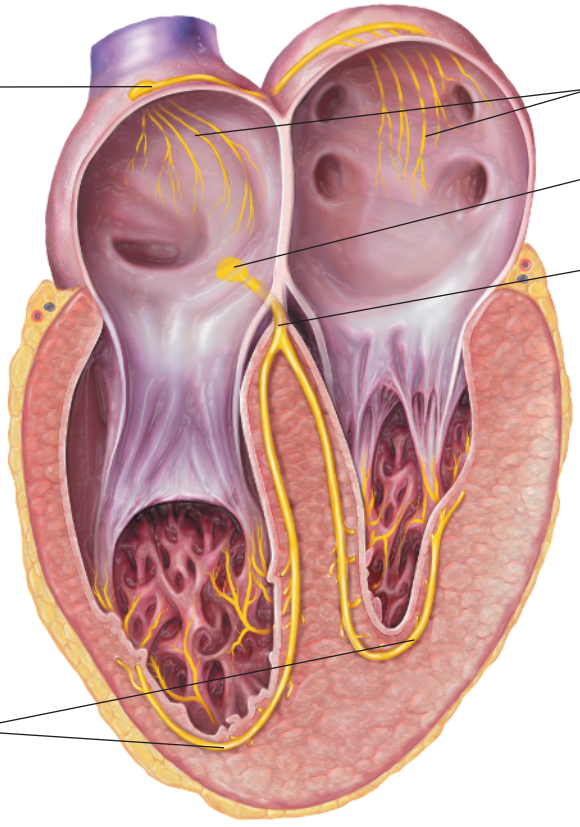
ATRIAL FIBRILLATION

AF is a rhythm that is characterized by an irregular rhythm resulting in disorganized atria contraction and lack of synchronization between the atria and ventricles. The atria rate may be as fast as 400 beats/min and the ventricular rate will be an irregular 120 to 200 beats/min.

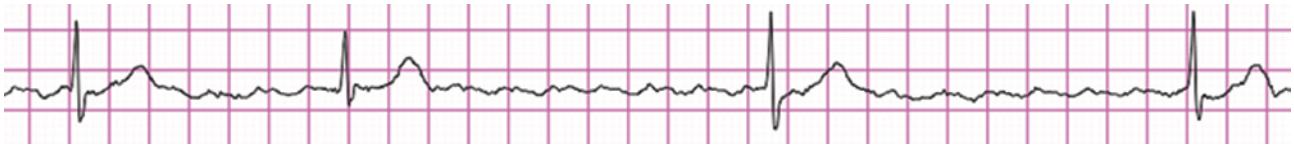
AF usually occurs in a patient who has underlying heart disease such as CAD, rheumatic heart disease, cardiomyopathy, hypertension, HF, and pericarditis. The ineffective atrial contractions and/or rapid ventricular response leads to decreased cardiac output. There is a great potential for clot formation to develop because of the blood stasis that develops in the heart as the chambers do not have enough time to empty completely between each beat. These clots can travel to the brain leading to a stroke. AF, accounts for 20% of all strokes.

On the EKG, there will be no P waves or the P waves are erratic, irregular, baseline fibrillatory waves. The QRS complexes are of uniform configuration and duration. AF is most commonly due to hypertension, HF, and increasing age.

PICTURING PATHO



In AF, electrical stimulation does not begin at the SA node but instead at other, ectopic areas within the atria or at the pulmonary vein. This causes an irregular rhythm as well as an increase in the heart rate. (Reprinted with permission from Stewart JG. *Anatomical Chart Company Atlas of Pathophysiology*. Philadelphia: Wolters Kluwer; 2018.)



In AF the heart rhythm is irregular and there is no distinct P wave since the impulses are not arising from the SA node. (Reprinted with permission from Martindale JL, Brown DFM. *A Visual Guide to ECG Interpretation*. 2nd ed. Philadelphia: Wolters Kluwer; 2016.)

Signs and Symptoms

- Shortness of breath
- Irregular palpitations
- Pulse deficit
- Weakness or problems exercising
- Chest pain
- Dizziness or fainting
- Fatigue
- Confusion

Treatment

- Anticoagulants such as warfarin (Coumadin), dabigatran (Pradaxa), rivaroxaban (Xarelto), and apixaban (Eliquis).
- Beta blockers to slow the heart rate. Example: metoprolol (Lopressor).
- Calcium channel blockers: Slows the heart rate and reduces the strength contraction. Example: diltiazem (Cardizem).
- Antiarrhythmics: slows the rate at which the electrical currents are conducted from the atria to the ventricle. Example: digoxin.
- Cardioversion: Electrodes are placed on the patient's chest to send electric shocks through the heart to establish a normal sinus rhythm.
- Radiofrequency catheter ablation: the use of radiofrequency energy to destroy a small area of heart tissue that is causing rapid and irregular heartbeats.
- Maze procedure: creates new pathways for the electrical impulses to travel easier through the heart.

Nursing Considerations

- Recognize that patients with AF are at high risk to develop a clot.
- Patients with AF may be asymptomatic and diagnosis is dependent on the EKG.
- Most accurate assessment of heart rate is the atrial rate as there may be a pulse deficit.
- Patients need to be taught that adhering to medication regimen is important to prevent complications.

TIP: Most accurate assessment of heart rate is the atrial rate as there may be a pulse deficit.

Pathophysiology

In AF, ectopic sites within the atria disrupt the normal pathway between the SA node and the AV node causing the atria to quiver. This results in incomplete emptying of the atria which may lead to the formation of clots and consequently a stroke and other cardiac complications.

Teaching About Atrial Fibrillation

- If patient has been prescribed digoxin, teach how to count the pulse rate.
- If patient has been prescribed a beta blocker, remind the patient to change positions slowly to avoid becoming dizzy.
- Remind the patient that bleeding may occur more easily due to anticoagulation therapy. Patient should be advised to use a soft-bristled toothbrush and use an electric shaver instead of a razor.
- If Coumadin has been prescribed, teach the patient to maintain a *consistent* daily intake of green leafy vegetables because of the interaction of Coumadin and foods containing Vitamin K. In addition, advise the patient to take the Coumadin as prescribed by the health care practitioner and to take the medication at the same time each day and that regular INR studies are essential to prevent an overdose of the drug which can lead to excessive bleeding.

TREATMENTS AND PROCEDURES

DRUG THERAPY

Several drug classes are critical to the treatment of cardiovascular disorders. These drug classes include:

Antianginals:
- Beta-adrenergic blockers
- Calcium channel blockers
- Nitrates

Antiarrhythmics:
- Sodium channel blockers
- Beta-adrenergic blockers
- Potassium channel blockers
- Calcium channel blockers

Antiplatelet
- Aspirin
- P2Y12 Receptor Inhibition
- Glycioriteub llb/llla Inhibitors

Anticoagulants:
- Unfractionated heparin (parenteral)
- Low-molecular-weight heparins (parenteral)
- Direct thrombin inhibitors (parenteral and oral)
- Coumadin derivatives (oral)
- Antifactor Xa inhibitors (parenteral and oral)

Antihypertensives:
- Angiotensin-converting enzymes (ACE)
- Angiotensin ll receptor blockers (ARBs)
- Beta-adrenergic blockers
- Calcium channel blockers
- Diuretics
- Selective aldosterone receptor antagonists
- Sympatholytics
- Vasodilators

Antilipemics:
- Bile-sequestering drugs
- Cholesterol absorption inhibitors
- Fibric-acid derivatives
- HMG-CoA reductase inhibitors (Statins)
- Nicotinic acid (niacin)
- PCSK9 inhibitors

Inotropics

Thrombolytics

Thrombolytics

Thrombolytics can dissolve a clot or thrombus that has caused acute MI, ischemic stroke or peripheral artery occlusion, or PE. They can also dissolve thrombi and reestablish blood flow in arteriovenous cannulas, grafts, and IV catheters. In an acute or emergency situation, they must be administered within 3 to 6 hours after the onset of symptoms. Thrombolytics include:

- Streptokinase (Streptase)
- Alteplase (tPA)
- Tenecteplase (TNKase)
- Reteplase (Retavase)

How Thrombolytics Help Restore Circulation

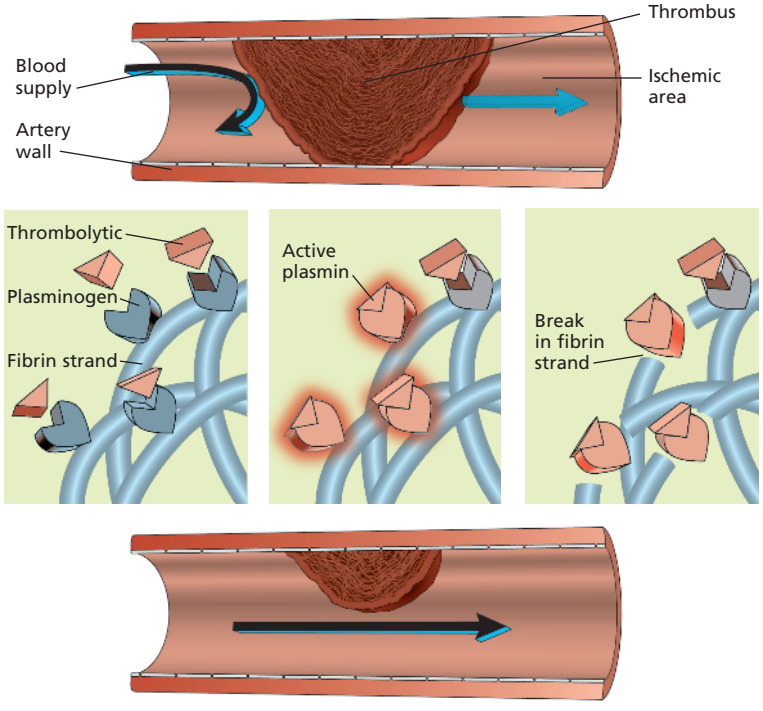
When a thrombus forms in an artery, it obstructs the blood supply, causing ischemia and necrosis. Thrombolytics can dissolve thrombi in the coronary and pulmonary arteries, restoring the blood supply to the area beyond the blockage.

OBSTRUCTED ARTERY

A thrombus blocks blood flow through the artery, causing distal ischemia.

INSIDE THE THROMBUS

The thrombolytic enters the thrombus and binds to the fibrin-plasminogen complex, converting inactive plasminogen into active plasmin. Active plasmin digests fibrin, dissolving the thrombus. As the thrombus dissolves, blood flow resumes.



SURGICAL AND OTHER PROCEDURES

Cardiac Pacing

Cardiac pacemakers are utilized when there is interruption of the electrical conduction necessitating restoration of heart rate. Pacemakers are effective in the treatment of a variety of bradycardic and tachycardia arrhythmias. Utilization of cardiac pacemakers can restore circulation and hemodynamics which are compromised by a slow heart rate.

Temporary Pacing

Temporary pacemakers are utilized in emergency situations. Temporary pacemakers contain external, battery-powered pulse generators, a lead, or electrode system which can be transcutaneous or transvenous.

Temporary cardiac pacing can be performed in a variety of ways:

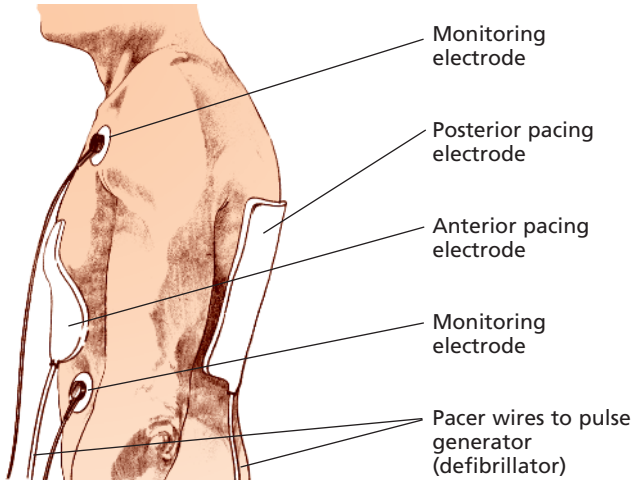
- Internally using transvenous endocardial leads
- Externally via transthoracic patches
- Internally using atrial or ventricular epicardial leads placed at the time of surgery
- Internally via an esophageal electrode, which is primarily used for atrial pacing and recording

Transvenous cardiac pacing is the preferred method to of pacing for patients with symptomatic bradycardias. Transvenous pacing requires expertise in both venous and cardiac anatomy in order to effectively access the vasculature and advance the electrode into the heart.

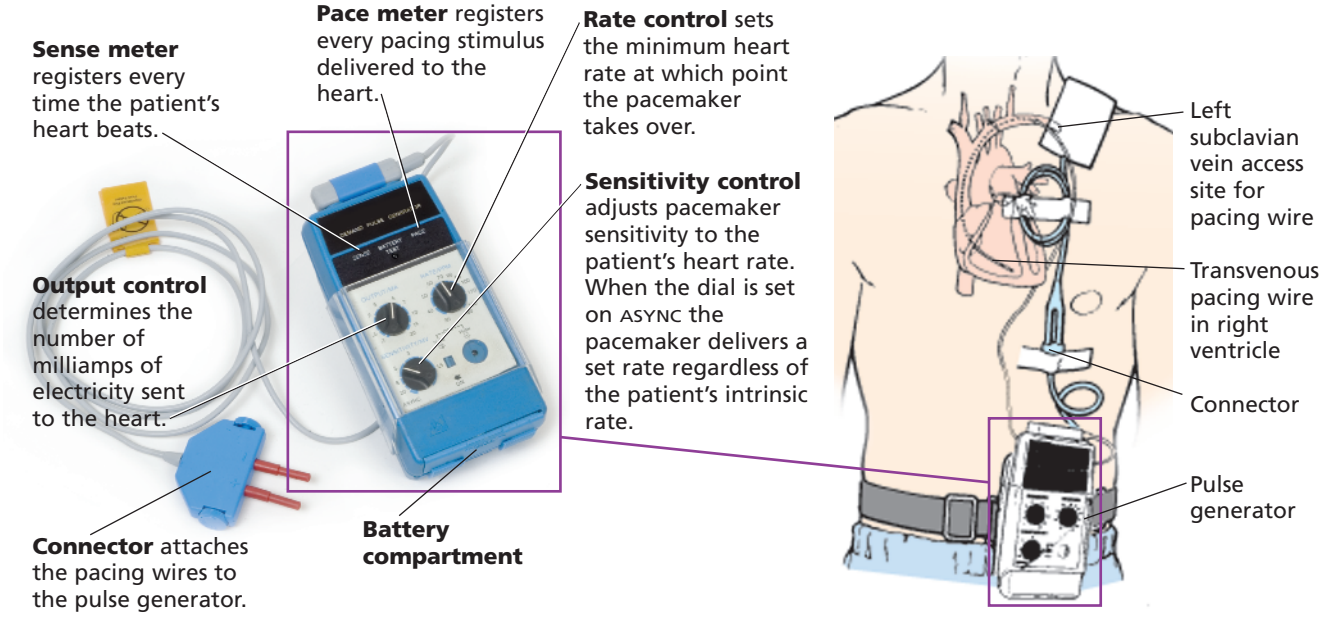
Transcutaneous Electrode Placement

HANDS ON

For a noninvasive temporary pacemaker, the two pacing electrodes are placed at heart level on the patient's chest and back, as shown. This type of pacemaker can be quickly applied in an emergency but is uncomfortable for the patient.



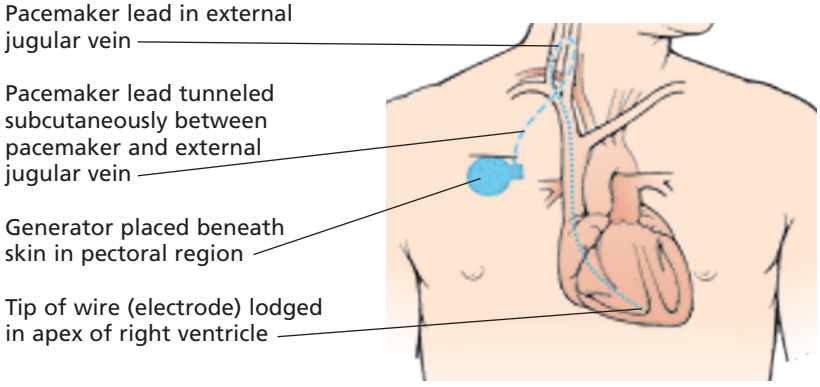
Transvenous Pulse Generator



Permanent Pacemaker Systems

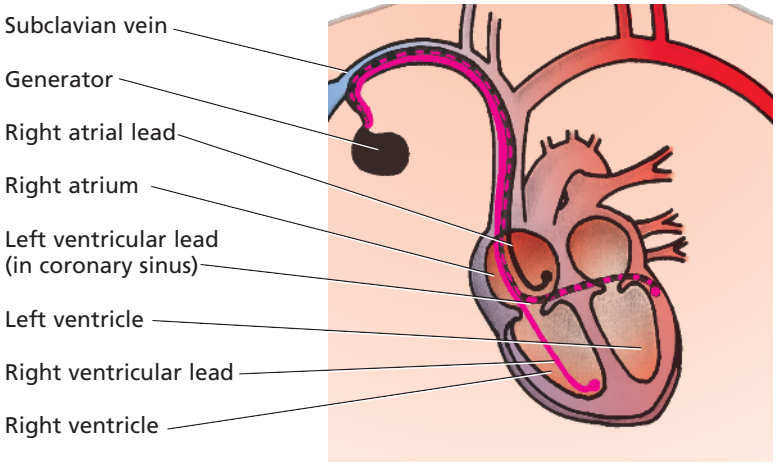
Permanent pacemakers are fully implanted in the body and connected to the heart by one or two electrode leads. The pacemaker is powered by solid-state lithium batteries, which usually last 5 to 10 years. Pacemakers are programmable, there operating characteristics can be changed by a programmer that transmits specific electromagnetic signals through the skin.

Permanent Pacemaker



Biventricular Pacemaker

A biventricular pacemaker uses three leads: one to pace the right atrium, one to pace the right ventricle, and one to pace the left ventricle. The left ventricular lead is placed in the coronary sinus. Both ventricles are paced at the same time, causing them to contract simultaneously, improving cardiac output.



Understanding Pacemaker Codes

First Letter

The first letter of a pacemaker code identifies the heart chambers that are paced:

V = Ventricle
A = Atrium
D = Dual (ventricle and atrium)
O = None

Second Letter

The second letter signifies the heart chamber where the pacemaker senses the intrinsic activity:

V = Ventricle
A = Atrium
D = Dual
O = None

Third Letter

The third letter shows the pacemaker's response to the intrinsic electrical activity it senses in the atrium or ventricle.

T = Triggers pacing
I = Inhibits pacing
D = Dual (can be triggered or inhibited depending on the mode and where intrinsic activity occurs)
O = None (the pacemaker doesn't change its mode in response to sensed activity)

Fourth Letter

The fourth letter signifies the rate modulation ability of the pacemaker (attempt to replicate the ability of a normal heart to increase heart rate in response to metabolic demand).

R = Rate modulated
O = None

Fifth Letter

The fifth letter designates the location of multisite pacing.

V = Ventricle
A = Atrium
D = Dual
O = None

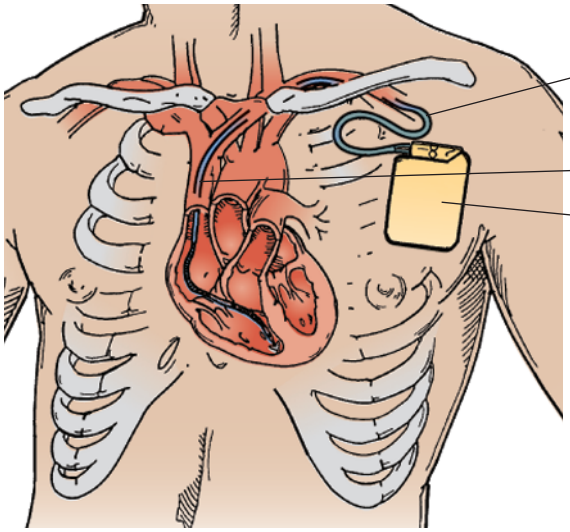
Implantable Cardioverter Defibrillators

A Look at an ICD

An ICD typically consists of a programmable pulse generator and an electrode. Most new ICDs can act as both pacemakers and defibrillator.

The subcutaneous implantable cardioverter defibrillator (S-ICD), which is being implanted throughout the world, was approved for use in the United States in September of 2012.

The S-ICD can be implanted without the use of fluoroscopy by using anatomic landmarks to guide proper positioning.



Types of ICD Therapies

VF is a common cause of sudden cardiac death (SCD) and is sometimes preceded by monomorphic or polymorphic ventricular tachycardia (VT). While cardiopulmonary resuscitation, including chest compressions and assisted ventilation, can provide temporary circulatory support for the patient with cardiac arrest, the only effective approach for terminating VF is electrical defibrillation. Development of the ICD was first implanted in humans in the 80s, which lead to the success and survival of patients with VF/VT.

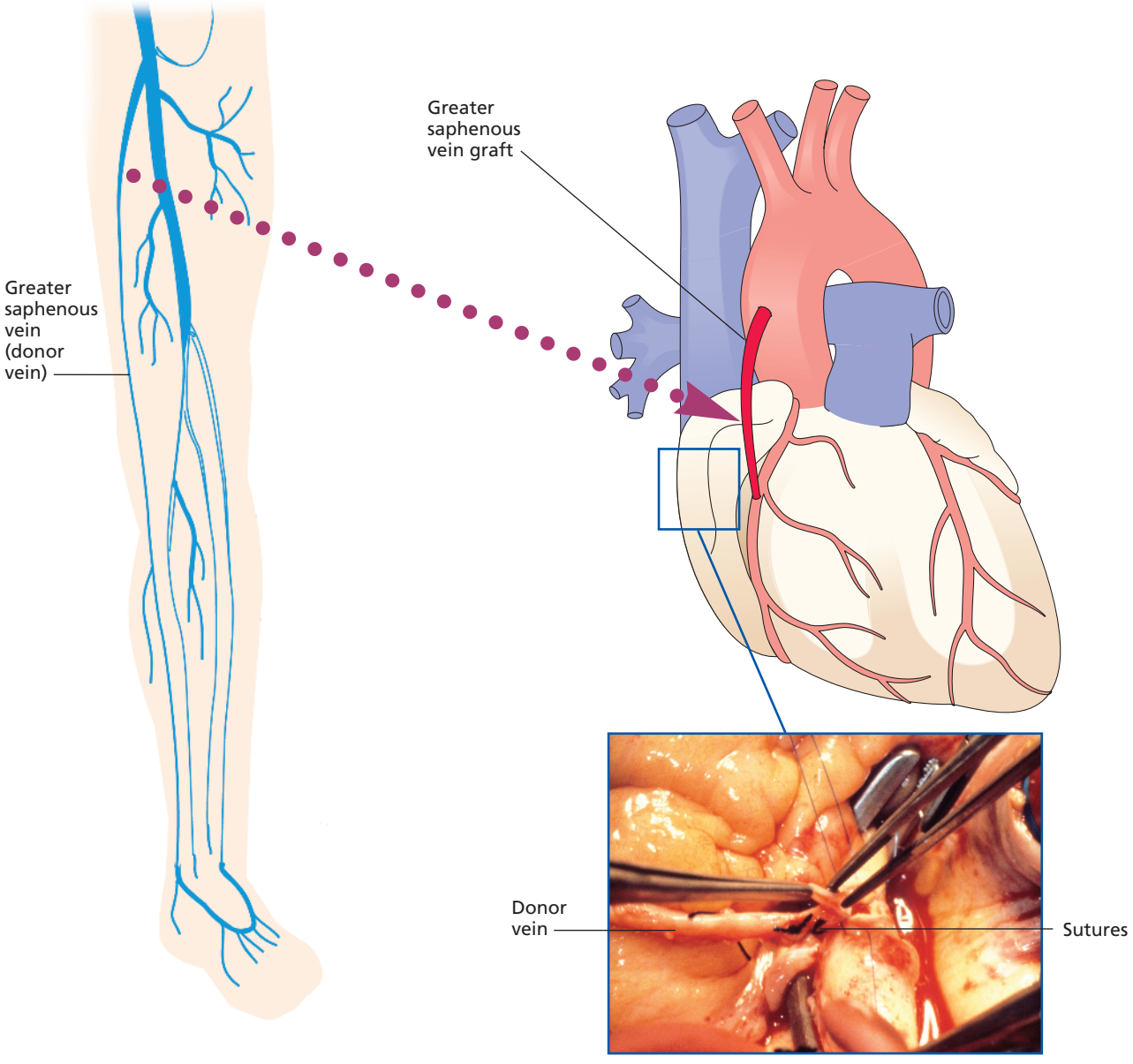
Therapy	What It Does
Antitachycardia pacing	A series of small, rapid, electrical pacing pulses are used to terminate ventricular tachycardia (VT) and return the heart to its normal rhythm.
Cardioversion	A low- or high-energy shock is timed to the R wave to terminate VT and return the heart to its normal rhythm.
Defibrillation	A high-energy shock to terminate ventricular fibrillation and return the heart to its normal rhythm.
Bradycardia pacing	Electrical pacing pulses are used when the heart's natural electrical signals are too slow. Most ICD systems can pace one chamber (VVI pacing) of the heart at a preset rate. Some systems sense and pace both chambers (DDD pacing).

Coronary Artery Bypass Graft Surgery

CABG recommended for patients with obstructive CAD whose survival will benefit compared to medical therapy or PCI. In addition, other patients with angina refractory to medical therapy may receive a recommendation for CABG if PCI cannot be performed. The procedure involves the construction of one or more grafts between the arterial and coronary circulations.

How CABG is Performed

CABG surgery circumvents an occluded coronary artery by using a segment of the saphenous vein, radial artery, or internal mammary artery to restore blood flow to the heart.

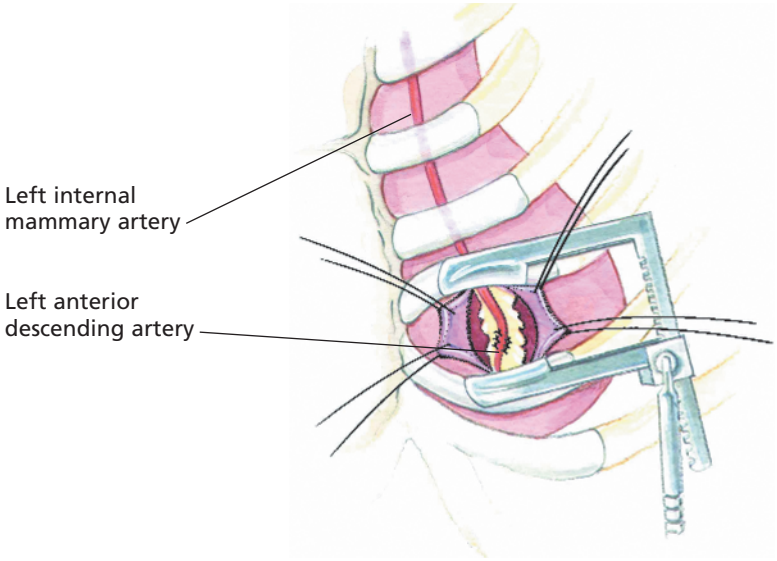


Minimally Invasive Direct Coronary Artery Bypass

Minimally invasive direct coronary artery bypass surgery (MIDCAB) also known as minimal or limited access CABG, refers to procedures that use alternative incisions to standard median sternotomy. MIDCAB is performed through a limited anterior thoracotomy and generally without cardiopulmonary bypass. In many instances, this approach typically involves only an anastomosis between the left internal mammary artery (LIMA) and left anterior descending (LAD) coronary artery or some use of a composite graft off the LIMA to other coronary arteries.

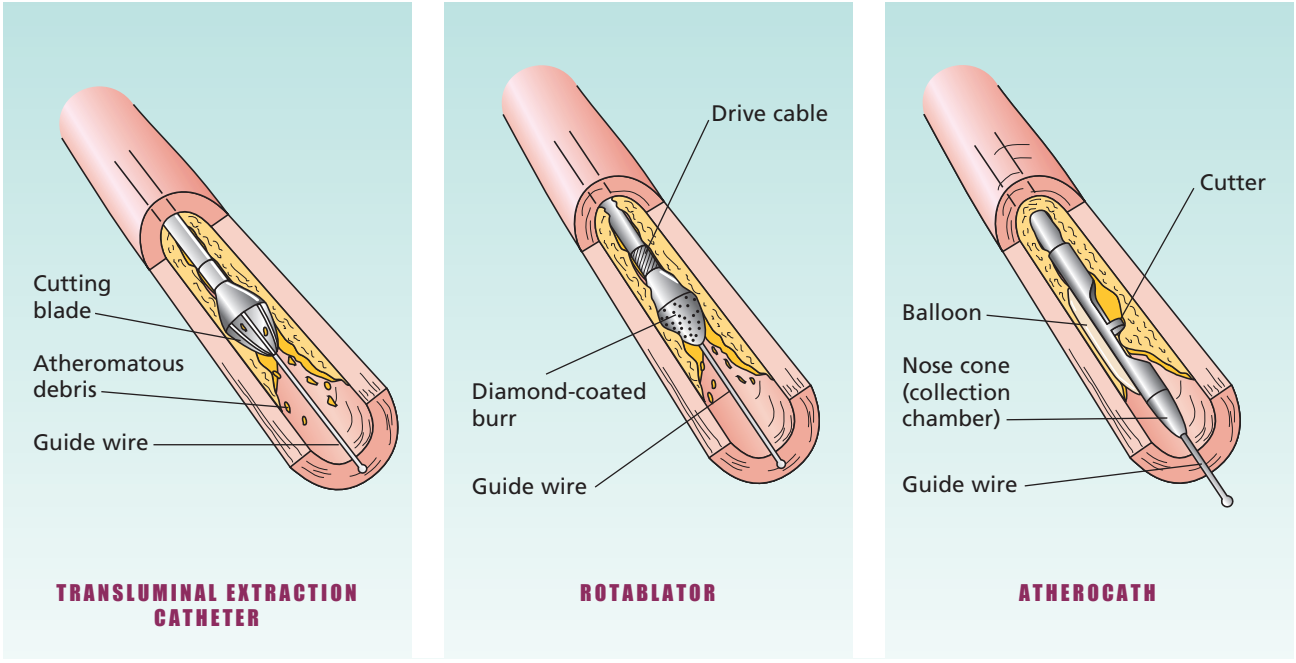
The MIDCAB Procedure

The MIDCAB procedure is performed through a short incision in the left chest cavity. The internal mammary artery is sewn to the LAD artery in the front of the heart, as shown here.



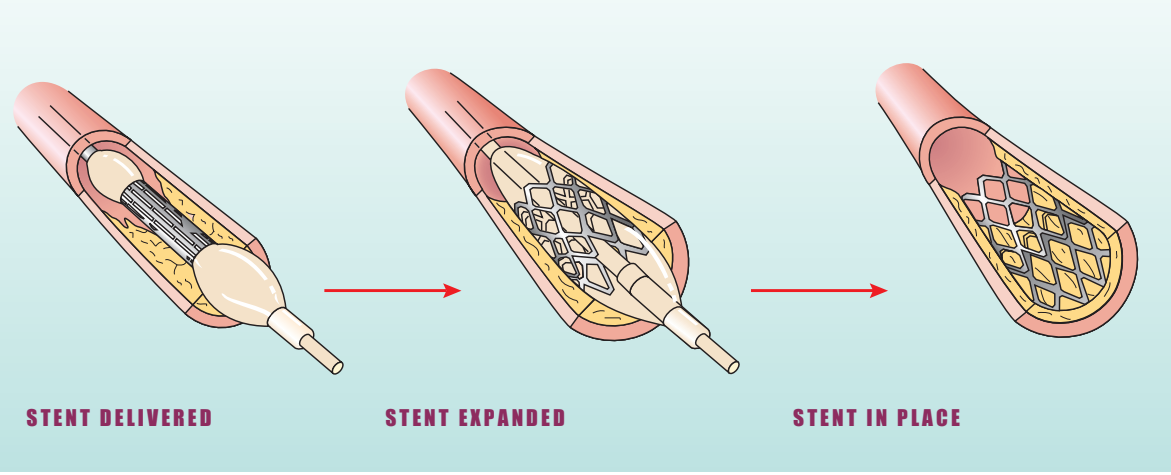
Atherectomy

Atherectomy is the use of a small, rotating knife to remove fatty deposits from blocked coronary arteries. The catheter is advanced to the arterial obstruction, the knife is positioned precisely on the fatty deposit, and then the fatty deposit is shaved off the artery wall.



Intravascular Stents

An intravascular stent may be used to hold the walls of a vessel open. Some stents are coated with a drug that is slowly released to inhibit further aggregation of fibrin or clots.



Heart Transplantation

With heart transplantation, a patient's failing heart is replaced with a donor heart. Used to treat end-stage cardiac disease in patients who have poor quality of life and are not expected to survive for more than 6 to 12 months, heart transplantation does not provide a cure.

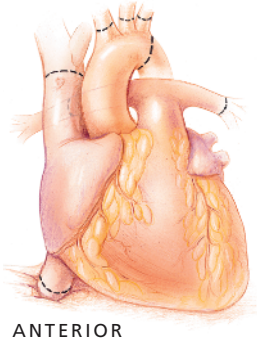
Patients who receive donor hearts must be treated for rejection with monoclonal antibodies and potent immunosuppressants that can increase the risk of life-threatening infection.

Heart Transplantation Surgery

The illustrations below outline the process of removing the donor heart and transplanting it into the recipient.

THE DONOR'S HEART

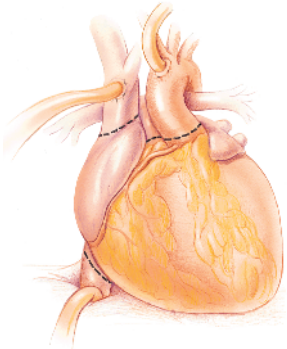
The donor's heart is removed after the surgeon cuts along these dissection lines.



ANTERIOR VIEW

THE RECIPIENT'S HEART

Before it can be removed, the recipient's heart is resected along these lines.



THE TRANSPLANTED HEART

The transplanted heart is sutured in place within the recipient.

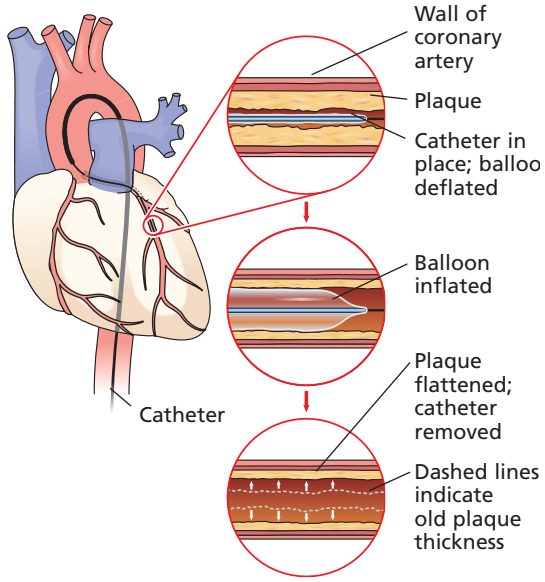
Percutaneous Transluminal Coronary Angioplasty

Percutaneous transluminal coronary angioplasty (PTCA), also called *angioplasty,* is a nonsurgical alternative to CABG. Performed in the cardiac catheterization laboratory under local anesthesia, it involves the use of a balloon-tipped catheter to dilate the blocked coronary artery. In most cases, the patients recuperate quickly, usually walking the same day and returning to work in 2 weeks.

PTCA works best when lesions are readily accessible, noncalcified, less than 10 mm, concentric, discrete, and smoothly tapered. Possible complications include vessel closure and late atherosclerosis.

Understanding PTCA

In PTCA, a guide catheter is threaded into the coronary artery by way of the femoral artery. Then, a balloon-tipped catheter is inserted through the occlusion and inflated to flatten the plaque until the vessel is opened.



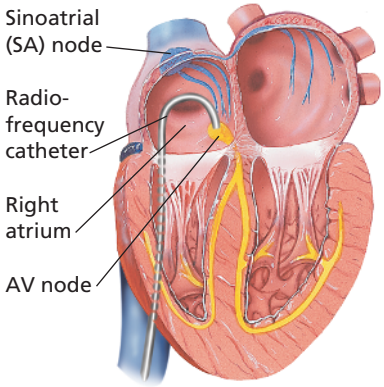
Radiofrequency Ablation

Radiofrequency ablation is used to treat arrhythmias in patients who do not respond to antiarrhythmic drugs or cardioversion. During the procedure, a special catheter is inserted in a vein and advanced to the heart. After the source of the arrhythmia is identified, radiofrequency energy destroys the abnormal electrical impulses or conduction pathway. The tissue that is destroyed can no longer conduct electrical impulses.

Types of Ablation

AV NODE ABLATION

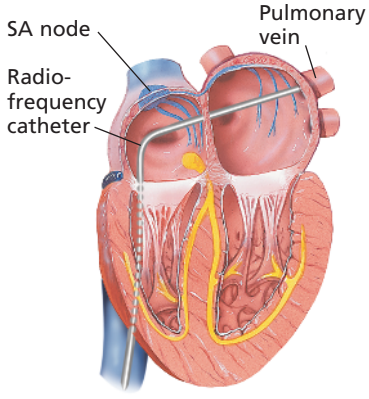
If a rapid arrhythmia originates above the AV node, the AV node may be destroyed to block impulses from reaching the ventricles.



The radiofrequency ablation catheter is directed to the base of the pulmonary vein.

PULMONARY VEIN ABLATION

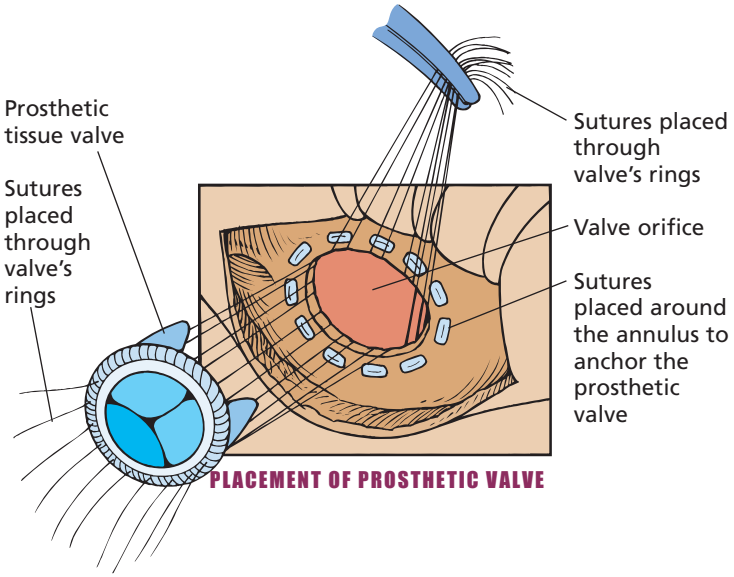
If the pulmonary vein is the source of the arrhythmia, such as in AF, radiofrequency energy is used to destroy the tissue in the area of the atrium that connects to the pulmonary vein. The scar that forms blocks impulses from firing within the pulmonary vein, preventing arrhythmias.



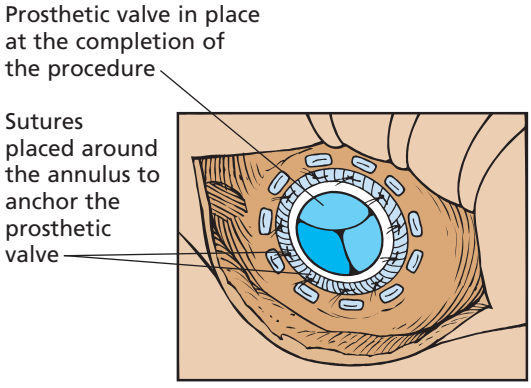
Radiofrequency energy is used to destroy the tissue where the atrium connects to the pulmonary vein.

Valve Replacement

With valve replacement, the natural heart valve is excised, and a prosthetic valve is sutured in place.



PLACEMENT OF PROSTHETIC VALVE



FINISHED VALVE REPLACEMENT

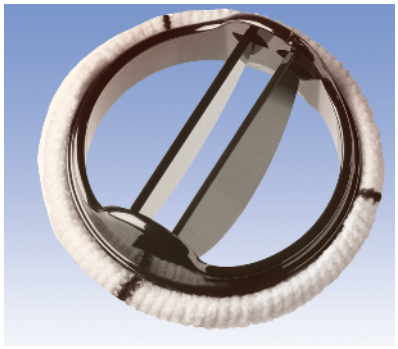
Valve Treatments

Valve treatments are used to prevent HF in patients with valvular stenosis or insufficiency accompanied by severe, unmanageable symptoms. Depending on the patient's condition, they may undergo one of three types of valve surgery. Types of valve surgery include valvuloplasty (valvular repair), commissurotomy (separation of the adherent, thickened leaflets of the mitral valve), and valve replacement (with a mechanical or prosthetic valve). When valve surgery is not an option, percutaneous balloon valvuloplasty is used to enlarge the orifice of a stenotic heart valve, improving valvular function.

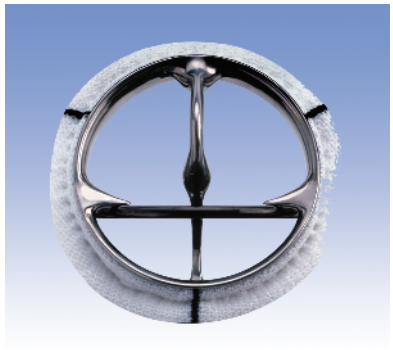
Although valve surgery carries a low risk of mortality, it can cause serious complications. Hemorrhage, for instance, may result from unligated vessels, anticoagulant therapy, or coagulopathy resulting from cardiopulmonary bypass during surgery. Stroke may result from thrombus formation caused by turbulent blood flow through the prosthetic valve or from poor cerebral perfusion during cardiopulmonary bypass. With valve replacement, bacterial endocarditis can develop within days of implantation or months later. Valve dysfunction or failure may occur as the prosthetic device wears out.

Types of Replacement Valves

Replacement valves can be mechanical or tissue.



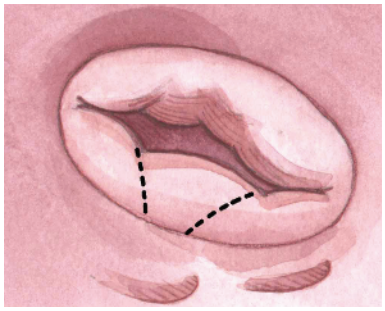
Bileaflet valve (St. Jude, mechanical)



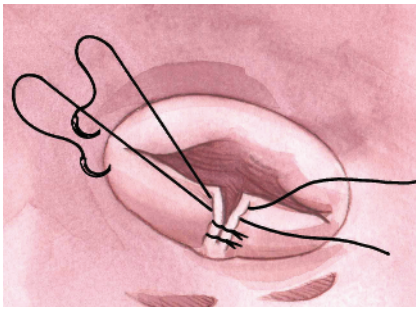
Tilting-disk valve (Medtronic-Hall, mechanical)

Porcine heterograft valve (Carpentier-Edwards, tissue)

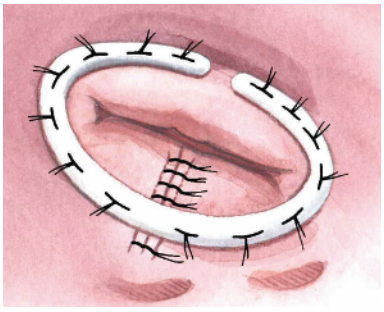
Valve Leaflet Resection and Repair



The section between the dashed line is excised.

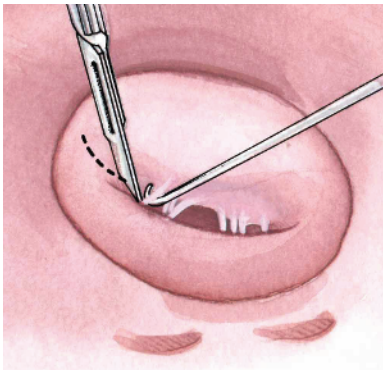


The edges are approximated and sutured.



The repair is finished off with an annuloplasty ring.

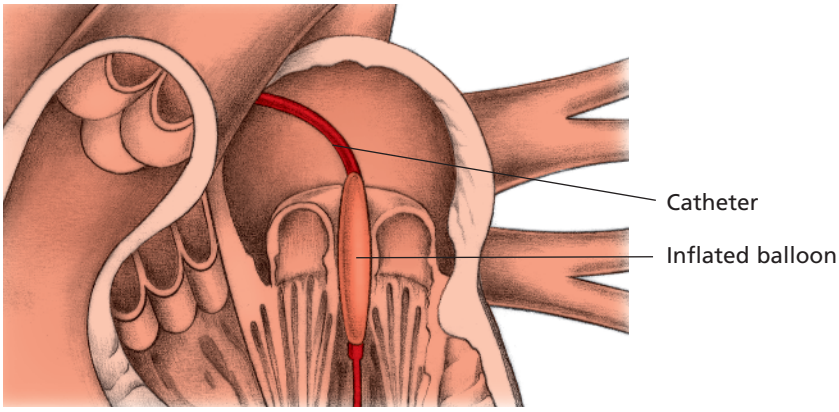
Commissurotomy of Mitral Valve



In commissurotomy, the thickened valve leaflets are surgically separated.

Percutaneous Balloon Valvuloplasty

During valvuloplasty, a surgeon inserts a small balloon catheter through the skin at the femoral vein and advances it until it reaches the affected valve. The balloon is then inflated, forcing the valve opening to widen.

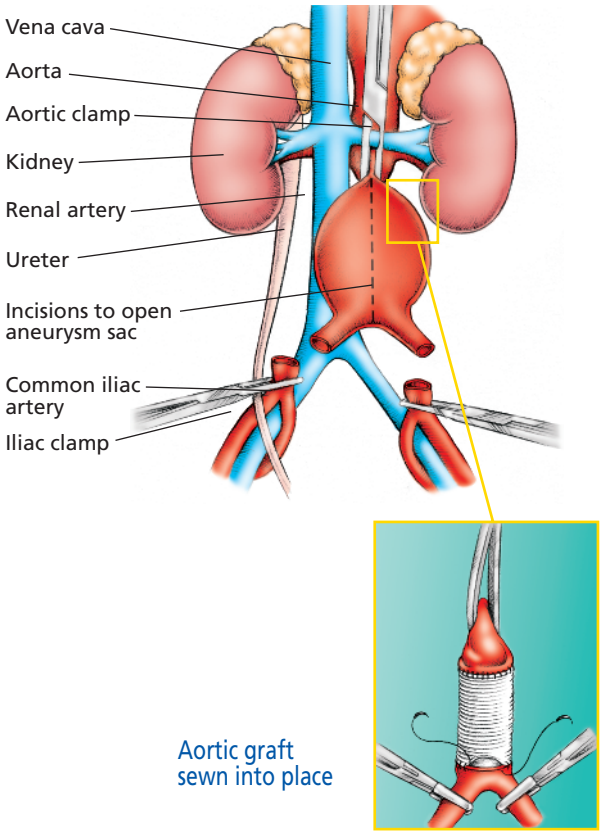


VASCULAR INTERVENTIONS

Aortic Aneurysm Resection

Aortic aneurysm resection involves removing an aneurysmal segment of the aorta. The surgeon first makes an incision to expose the aneurysm site. He then clamps the aorta, resects the aneurysm, and repairs the damaged portion of the aorta by sewing a prosthetic graft into place.

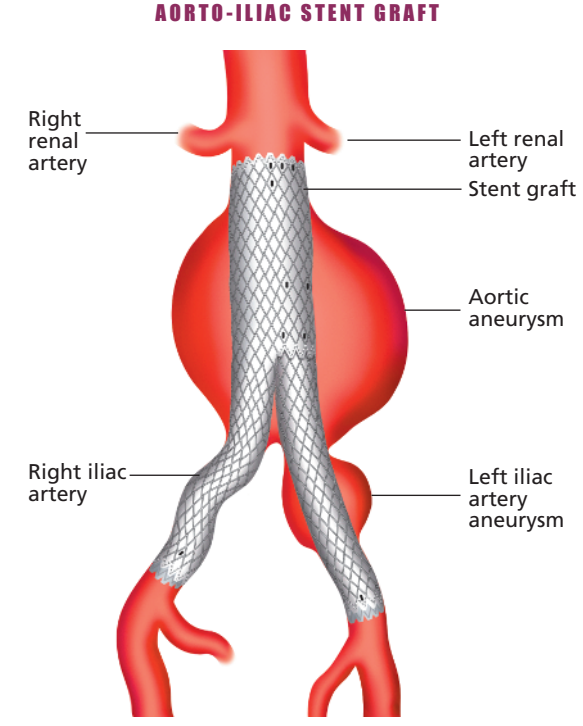
Aortic Aneurysm Resection



Aortic Endovascular Stent Graft

Endovascular stent grafting is a minimally invasive procedure used to repair AAA and other aneurysmal arteries by reinforcing the vessel walls. The surgeon uses fluoroscopic guidance to insert a delivery catheter with an attached compressed graft through a small incision into the iliac or femoral artery. The catheter is advanced into the aorta and positioned across the aneurysm below the renal arteries. The stent graft prevents blood from entering the aneurysmal area of the artery but maintains blood flow distally. This helps prevent the aneurysm from enlarging and possibly rupturing.

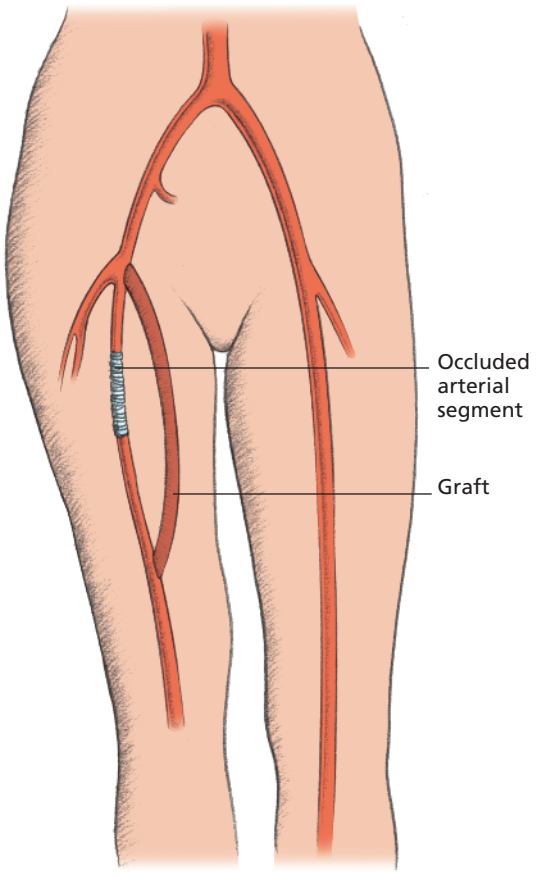
Aortic Endovascular Stent Graft



Arterial Bypass Graft

Bypass grafting serves to bypass an arterial obstruction resulting from arteriosclerosis. After exposing the affected artery, the surgeon anastomoses a synthetic or autogenous graft to divert blood flow around the occluded arterial segment.

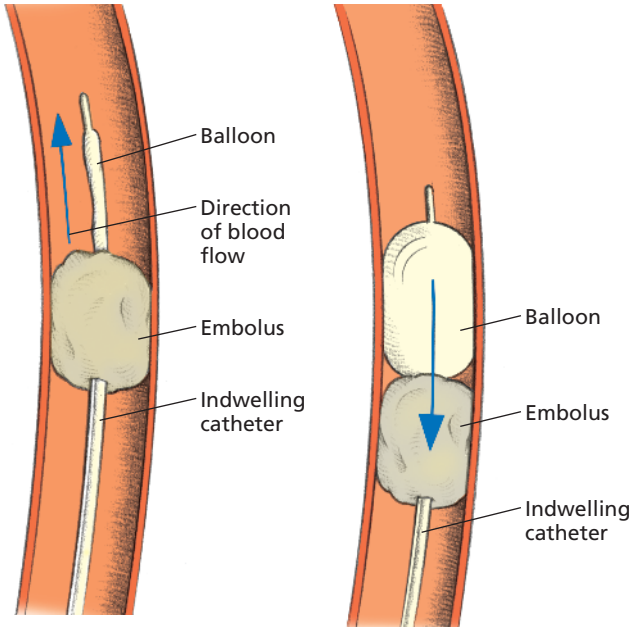
Arterial Bypass Graft



Embolectomy

To remove an embolism from an artery, a surgeon may perform an embolectomy by inserting a balloon-tipped indwelling catheter in the artery and passing it through the embolus. He then inflates the balloon and withdraws the catheter to remove the occlusion.

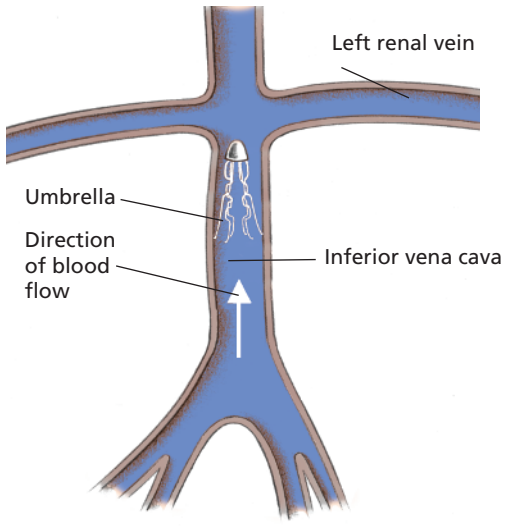
Embolectomy



Vena Caval Filter

A vena caval filter, or umbrella, traps emboli originating from the pelvis or lower extremities in the vena cava, preventing them from reaching the pulmonary vessels but allowing venous blood flow.

Vena Caval Filter



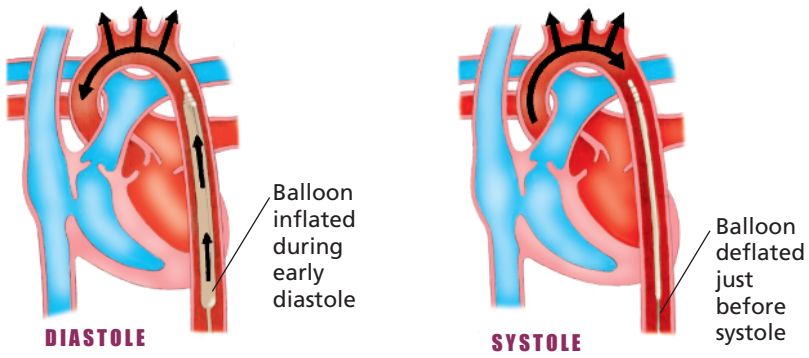
Mechanical Circulatory Assist (Support) Devices

Designed for a wide range of clinical conditions such as prophylactic insertion for high-risk invasive coronary artery procedures to manage cardiogenic shock, acute decompensated HF, or cardiopulmonary arrest.

These devices include:

- Intra-aortic balloon pump (IABP)
  - The intra-aortic balloon pump (IABP) is the most commonly used mechanical support device
  - These devices are often placed in the catheterization laboratory but in some cases, can be placed in intensive care unit.
- Intra-aortic balloon pump counterpulsation
  - Intra-aortic balloon pump (IABP) counterpulsation temporarily reduces left ventricular workload and improves coronary perfusion. It is used to treat cardiogenic shock caused by acute MI, septic shock, intractable angina before surgery, intractable ventricular arrhythmias, ventricular septal or papillary muscle ruptures, and pump failure.

Understanding an IABP

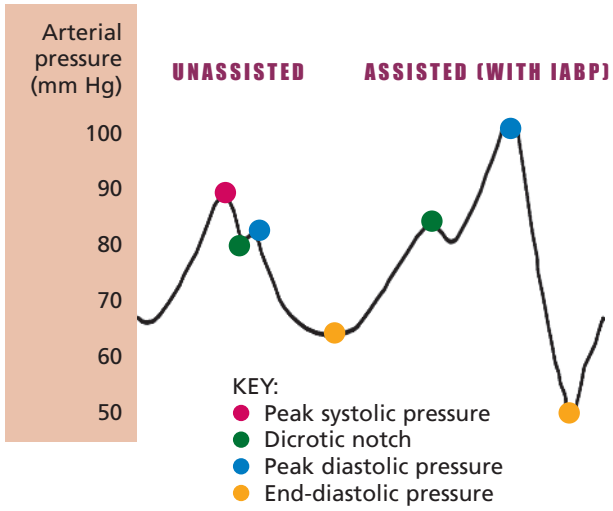


The balloon deflates rapidly at the end of diastole, creating a vacuum in the aorta. This reduces aortic volume and pressure, thereby decreasing the resistance to left ventricular ejection (afterload). This decreased workload, in turn, reduces the heart's oxygen requirements and, combined with the improved myocardial perfusion, helps prevent or diminish myocardial ischemia.

Non-IABP Percutaneous Mechanical Circulatory Assist Devices

- These devices have differing names: percutaneous ventricular assist devices (VADs), percutaneous ventricular support devices, percutaneous mechanical circulatory assist devices, percutaneous mechanical circulatory support devices, and percutaneous VADs.
  - Left ventricle to aorta assist devices—The Impella System
  - Left atrial to aorta assist devices—The Tandem Heart

Timing IABP Counterpulsation



- IABP counterpulsation is synchronized with either the ECG or the arterial waveform. Ideally, balloon inflation should begin when the aortic valve closes—at the dicrotic notch on the arterial waveform. Deflation should occur just before diastole.
- Timing of the counterpulsation is crucial. Early inflation can damage the aortic valve by forcing it closed, whereas late inflation permits most of the blood emerging from the ventricle to flow past the balloon, reducing the pump's effectiveness.
- Late deflation may cause cardiac arrest because it increases the resistance to left ventricle pumping. IABP counterpulsation boosts peak diastolic pressure and lowers peak systolic and end-diastolic pressures.

Ventricular Assist Device

A VAD is an implantable device that consists of a blood pump, cannulas, and a pneumatic or electrical drive console. The pump is synchronized to the patient's ECG and functions as the heart's ventricle. It decreases the heart's workload while increasing cardiac output.

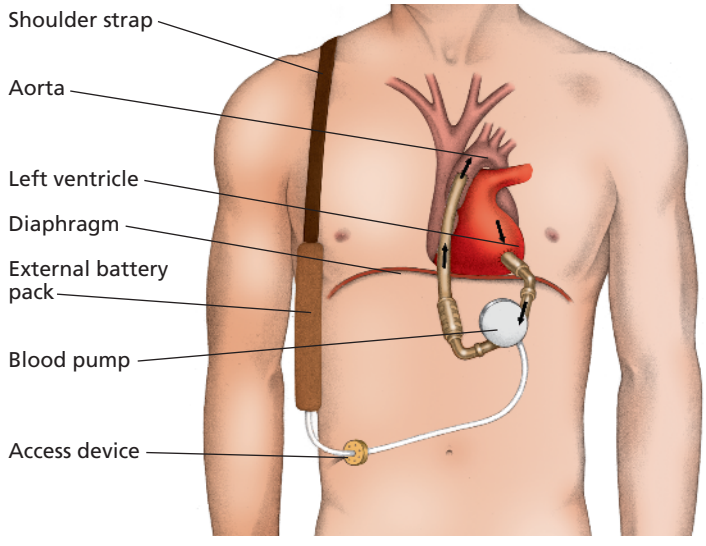
Pump Options

VADs are available as continuous flow or pulsatile pumps. A continuous flow pump fills continuously and returns blood to the aorta at a constant rate. A pulsatile pump may work in one of two ways:

- It may fill during systole and pump blood into the aorta during diastole.

Left VAD

A completely implanted left VAD is shown here.



- It may pump regardless of the patient's cardiac cycle.
- Nonpercutaneous centrifugal pumps, which are used for cardiopulmonary bypass. They are used primarily for cardiopulmonary bypass during open heart cases and thus are not placed percutaneously. They cause too much hemolysis to permit long-term use. There are presently two centrifugal pumps available for very short-term use (less than six hours).
  - the Bio-Medicus
  - Sarns
- Extracorporeal membrane oxygenator pumps

While each device has a different design and operation, the following parameters of circulatory function are improved by all devices; however the degree of improvement varies between devices and patients.

- End-organ perfusion
- Reduction in intracardiac filling pressures
- Reduction in left ventricular volumes, wall stress, and myocardial oxygen consumption
- Augmentation of coronary perfusion

As a result of treatment with these devices the following clinical parameters may be improved:

- Prevention or amelioration of cardiogenic shock
- Reduction in pulmonary congestion
- Reduction in manifestations of myocardial ischemia
- Reduction in infarct size

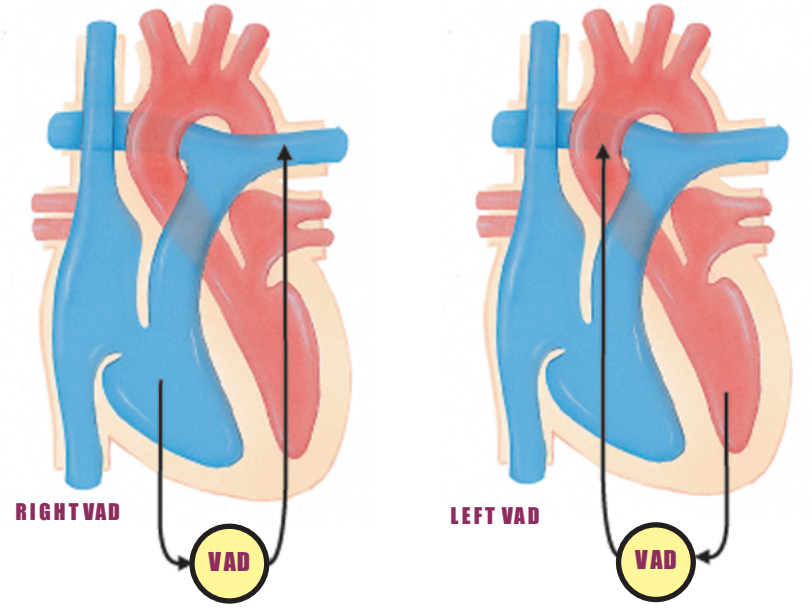
Extracorporeal Membrane Oxygenation (ECMO) is part of a larger term identified as extracorporeal life support (ECLS) which is a cardiopulmonary support system that. ECMO moves blood forward, removes carbon dioxide and adds oxygen to venous blood using an artificial membrane lung. The pulmonary circulation is bypassed and oxygenated blood returns to the patient via an arterial or venous route. With venovenous bypass, ECMO is effective primarily as a therapeutic option for patients with severe respiratory failure. With venoarterial bypass, an extracorporeal pump is utilized to support systemic perfusion, thus providing a hemodynamic support option in patients with circulatory and respiratory failure.

The ECMO systems involve placement in the central arterial and venous circulation using a large bore catheters that allow positioning of a cannula in the aorta and right atrium. Blood from the venous catheter is pumped through a heat exchanger and oxygenator and then returned to the systemic arterial circulation via the arterial cannula. The ECMO systems can be used for support for up to 30 days.

A Closer Look at VADs

There are three types of VADs.

1. A right VAD provides pulmonary support by diverting blood from the failing right ventricle to the VAD, which then pumps the blood to the pulmonary circulation via the VAD connection to the left pulmonary artery.
2. With a left VAD, blood flows from the left ventricle to the VAD, which then pumps blood back to the body via the VAD connection to the aorta.
3. When a right and a left VAD are used, it is referred to as a *biventricular VAD (BiVAD).*



Indications for Use of ECMO

- Hypoxemic respiratory failure.
- Hypercapnic respiratory failure with an arterial pH less than 7.20.
- Ventilatory support as a bridge to lung transplantation.
- Cardiac/circulatory failure/ refractory cardiogenic shock.
- Massive pulmonary embolism.
- Cardiac arrest
- Failure to wean from cardiopulmonary bypass after cardiac surgery.
- As a bridge to either cardiac transplantation or placement of a VAD.

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