II

DISORDERS

AORTIC ANEURYSM

A thoracic aortic aneurysm is an abnormal widening of the ascending, transverse, or descending part of the aorta. Aneurysm of the ascending aorta is the most common type and has the highest mortality. An abdominal aneurysm generally occurs in the aorta between the renal arteries and the iliac branches.

Causes

Aneurysm commonly results from atherosclerosis, which weakens the aortic wall and gradually distends the lumen. The exact cause is unknown, but there are factors that contribute which are included here:

- age and family history
- fungal infection (mycotic aneurysms) of the aortic arch and descending segments
- bicuspid aortic valve
- congenital disorders, such as coarctation of the aorta or Marfan syndrome
- inflammatory disorders
- trauma
- syphilis
- hypertension (in dissecting aneurysm)
- tobacco use.

AGE ALERT

Ascending aortic aneurysms, the most common type, are usually seen in hypertensive men under age 60. Descending aortic aneurysms, usually found just below the origin of the subclavian artery, are most common in elderly men with hypertension. They may also occur in younger patients after traumatic chest injury or, less commonly, after infection.

Pathophysiology

First, degenerative changes create a focal weakness in the muscular layer of the aorta (tunica media), allowing the inner layer (tunica intima) and outer layer (tunica adventitia) to stretch outward. The outward bulge is the aneurysm. The pressure of blood pulsing through the aorta progressively weakens the vessel walls and enlarges the aneurysm. As the vessel dilates, wall tension increases. This increases arterial pressure and dilates the aneurysm further.

Aneurysms may be *dissecting*, a hemorrhagic separation in the aortic wall, usually within the medial layer; *saccular*, an outpouching of the arterial wall; or *fusiform*, a spindle-shaped enlargement encompassing the entire aortic circumference.

A false aneurysm occurs when the entire wall is injured, with blood contained in the surrounding tissue. A sac eventually forms and communicates with an artery or the heart.

COMPLICATIONS

- Cardiac tamponade if aneurysm ruptures
- Dissection
- Rupture

Signs and Symptoms

Ascending Aneurysm

- Pain, the most common symptom of thoracic aortic aneurysm
- Bradycardia
- Murmur of aortic insufficiency
- Pericardial friction rub (caused by a hemopericardium)
- Unequal intensities of the right carotid and left radial pulses
- Difference in blood pressure between the right and left arms
- Jugular vein distention

Descending Aneurysm

- Pain, usually starting suddenly between the shoulder blades; may radiate to the chest
- Hoarseness
- Dyspnea and stridor
- Dysphagia
- Dry cough

Abdominal Aneurysm

Although abdominal aneurysms usually don't produce symptoms, most are evident as a pulsating mass in the periumbilical area. Other signs include:

- systolic bruit over the aorta
- tenderness on deep palpation
- lumbar pain that radiates to the flank and groin.

CLINICAL TIP

Pain caused by a dissecting aortic aneurysm:

- may be described as "ripping" or "tearing"
- commonly radiates to the anterior chest, neck, back, or abdomen
- usually has an abrupt onset.

Diagnostic Test Results

- Echocardiography shows the aneurysm and its size.
- Anteroposterior and lateral abdominal X-rays show aortic calcifications present in abdominal aortic aneurysms; posteroanterior and oblique chest X-rays will show widening of the aorta and mediastinum in thoracic aortic aneurysms.
- Computed tomography scan shows the effects on nearby organs.
- Aortography shows the size and location of the aneurysm.
- Complete blood count reveals decreased hemoglobin levels.

- Abdominal ultrasound can detect and monitor the progression of AAA.

Treatment

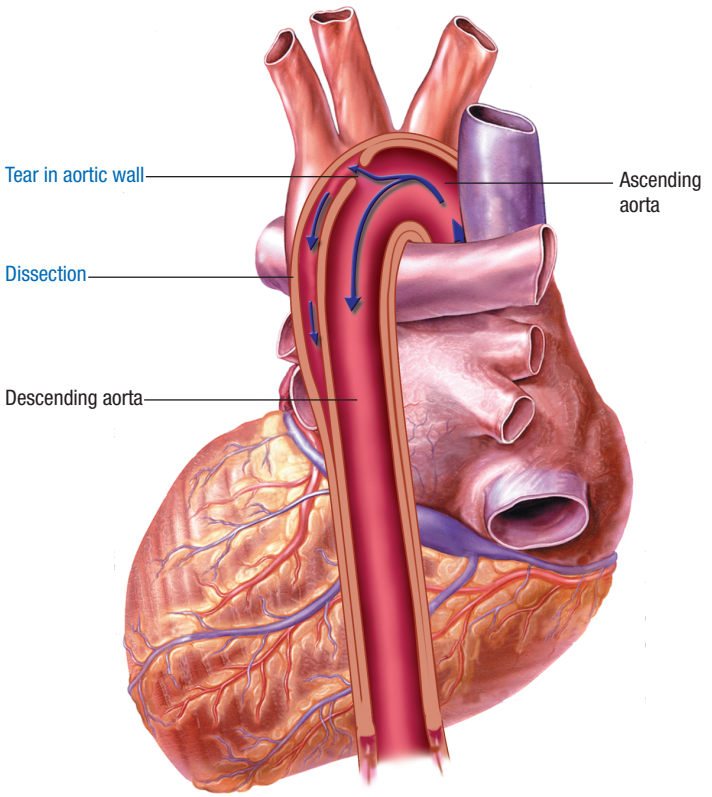
A dissecting aortic aneurysm is an emergency that requires prompt surgery and stabilizing measures. Treatment includes:

- antihypertensives such as nitroprusside
- negative inotropic agents to decrease force of contractility
- beta-adrenergic blockers
- oxygen for respiratory distress
- opioids for pain
- I.V. fluids
- possibly, whole blood transfusions.

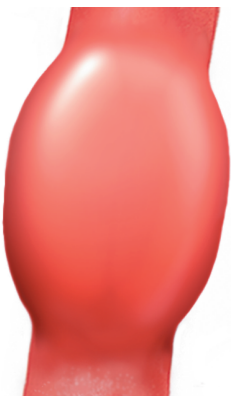
Treatment of stable AAA focuses on surveillance and tight BP control to prevent enlargement.

TYPES OF AORTIC ANEURYSMS

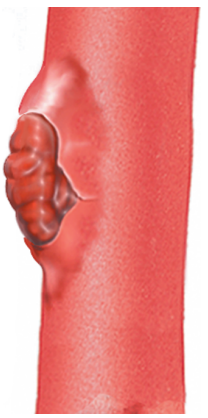
Dissecting aneurysm



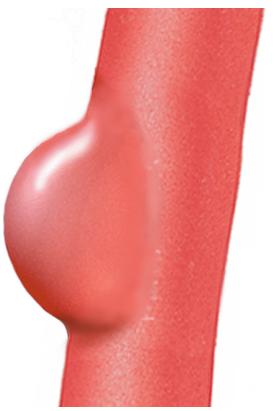
Fusiform aneurysm



False aneurysm



Saccular aneurysm



CARDIAC ARRHYTHMIAS

Abnormal electrical conduction or automaticity changes heart rate and rhythm. Arrhythmias vary in severity — from mild, producing no symptoms, and requiring no treatment (such as sinus arrhythmia, in which heart rate increases and decreases with respiration), to catastrophic ventricular fibrillation, which mandates immediate resuscitation. Arrhythmias are generally classified according to their origin (ventricular or supraventricular). Their effect on cardiac output and blood pressure, partially influenced by the site of origin, determines their clinical significance. (See the appendix "Types of cardiac arrhythmias.")

Causes

Each arrhythmia may have its own specific cause. Common causes include:

- congenital defects
- myocardial ischemia or infarction
- organic heart disease
- drug toxicity
- degeneration or obstruction of conductive tissue
- connective tissue disorders
- electrolyte imbalances
- hypertrophy of heart muscle
- acid-base imbalances
- emotional stress.

AGE ALERT

Electrocardiogram changes that occur with age include:

- longer PR, QRS, and QT intervals
- lower amplitude of QRS complex
- leftward shift of QRS axis.

Pathophysiology

Altered automaticity, reentry, or conduction disturbances may cause cardiac arrhythmias. Enhanced automaticity is the result of partial depolarization, which may increase the intrinsic rate of the sinoatrial node or latent pacemakers or may induce ectopic pacemakers to reach threshold and depolarize.

Ischemia or deformation causes an abnormal circuit to develop within conductive fibers. Although current flow is blocked in one direction within the circuit, the descending impulse can travel in the other direction. By the time the impulse completes the circuit, the previously depolarized tissue within the circuit is no longer refractory to stimulation; therefore, arrhythmias occur.

Conduction disturbances occur when impulses are conducted too quickly or too slowly.

COMPLICATIONS

- Impaired cardiac output
- Cardiac arrest in certain arrhythmias
- Stroke in prolonged atrial arrhythmias

Signs and Symptoms

Signs and symptoms of arrhythmias result from reduced cardiac output and altered perfusion to the organs and may include:

- dyspnea
- hypotension
- dizziness, syncope, and weakness
- chest pain
- cool, clammy skin
- altered level of consciousness
- reduced urinary output
- palpitations.

Diagnostic Test Results

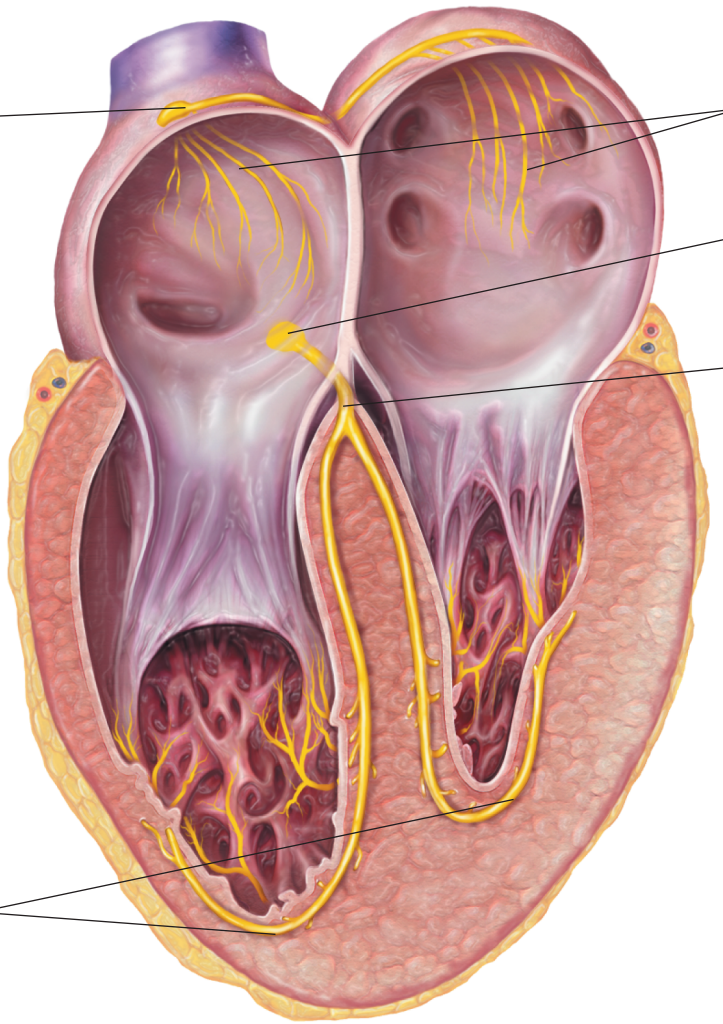
- Electrocardiography (ECG) detects arrhythmias as well as ischemia and infarction by showing prolonged or shortened intervals, elevated or depressed T waves, premature contractions, or absence of waves.
- Blood tests reveal electrolyte abnormalities, such as hyperkalemia or hypokalemia and hypermagnesemia or hypomagnesemia, as well as drug toxicities.
- Arterial blood gas analysis reveals acid-base abnormalities, such as acidemia or alkalemia.
- Holter monitoring, event monitoring, and loop recording show the presence of an arrhythmia.
- Exercise testing detects exercise-induced arrhythmias.
- Electrophysiologic testing identifies the mechanism of an arrhythmia and the location of accessory pathways; it also assesses the effectiveness of antiarrhythmic drugs, radiofrequency ablation, and implantable cardioverter–defibrillators (ICDs).

Treatment

Follow the specific treatment guidelines or protocols for each arrhythmia. Treatment generally focuses on the underlying problem and may include:

- antiarrhythmic medications
- electrolyte correction
- oxygen
- correction of acid-base balance
- cardioversion
- radiofrequency ablation
- ICD
- pacemaker
- cardiopulmonary resuscitation.

SITES OF COMMON CARDIAC ARRHYTHMIAS



Sinus node arrhythmias
- Sinoatrial block
- Sinus bradycardia
- Sinus tachycardia

Atrial arrhythmias
- Premature atrial contractions
- Atrial fibrillation
- Atrial flutter

Atrioventricular (AV) blocks
- First-degree AV block
- Second-degree AV block
- Third-degree AV block

Junctional arrhythmia
- Junctional rhythm

Ventricular arrhythmias
- Premature ventricular contractions
- Ventricular fibrillation
- Ventricular tachycardia

CARDIAC TAMPONADE

Cardiac tamponade is a rapid, unchecked rise in pressure in the pericardial sac that compresses the heart, impairs diastolic filling, and limits cardiac output. The rise in pressure usually results from blood or fluid accumulation in the pericardial sac (pericardial effusion). Even a small amount of fluid (50 to 100 mL) can cause a serious tamponade if it accumulates rapidly.

Causes

- Idiopathic
- Effusion (due to cancer, bacterial infections, tuberculosis, or, rarely, acute rheumatic fever)
- Traumatic or nontraumatic hemorrhage
- Viral or postirradiation pericarditis
- Chronic renal failure requiring dialysis
- Drug reaction (procainamide, hydralazine, minoxidil, isoniazid, penicillin, or daunorubicin)
- Heparin- or warfarin-induced tamponade
- Connective tissue disorders
- Postcardiac surgery
- Acute myocardial infarction (MI)
- Pericarditis

Pathophysiology

In cardiac tamponade, the progressive accumulation of fluid in the pericardial sac causes compression of the heart chambers. This compression obstructs filling of the ventricles and reduces the amount of blood that can be pumped out of the heart with each contraction.

Each time the ventricles contract, more fluid accumulates in the pericardial sac. This further limits the amount of blood that can fill the ventricular chambers, especially the left ventricle, during the next cardiac cycle.

The amount of fluid necessary to cause cardiac tamponade varies greatly; it may be as little as 50 to 100 mL when the fluid accumulates rapidly or more than 2,000 mL if the fluid accumulates slowly and the pericardium stretches to adapt. Prognosis is inversely proportional to the amount of fluid accumulated.

COMPLICATIONS

- Decreased cardiac output
- Cardiogenic shock
- Death if untreated

Signs and Symptoms

- Elevated central venous pressure (CVP) with jugular vein distention
- Muffled heart sounds
- Pulsus paradoxus (decreases systolic blood pressure with inspiration)
- Diaphoresis and cool, clammy skin
- Anxiety, restlessness, and syncope
- Cyanosis
- Weak, rapid pulse
- Cough, dyspnea, orthopnea, and tachypnea

CLINICAL TIP

Cardiac tamponade has three classic features, known as Beck's triad, that include:

- elevated CVP with jugular vein distention
- muffled heart sounds
- pulsus paradoxus.

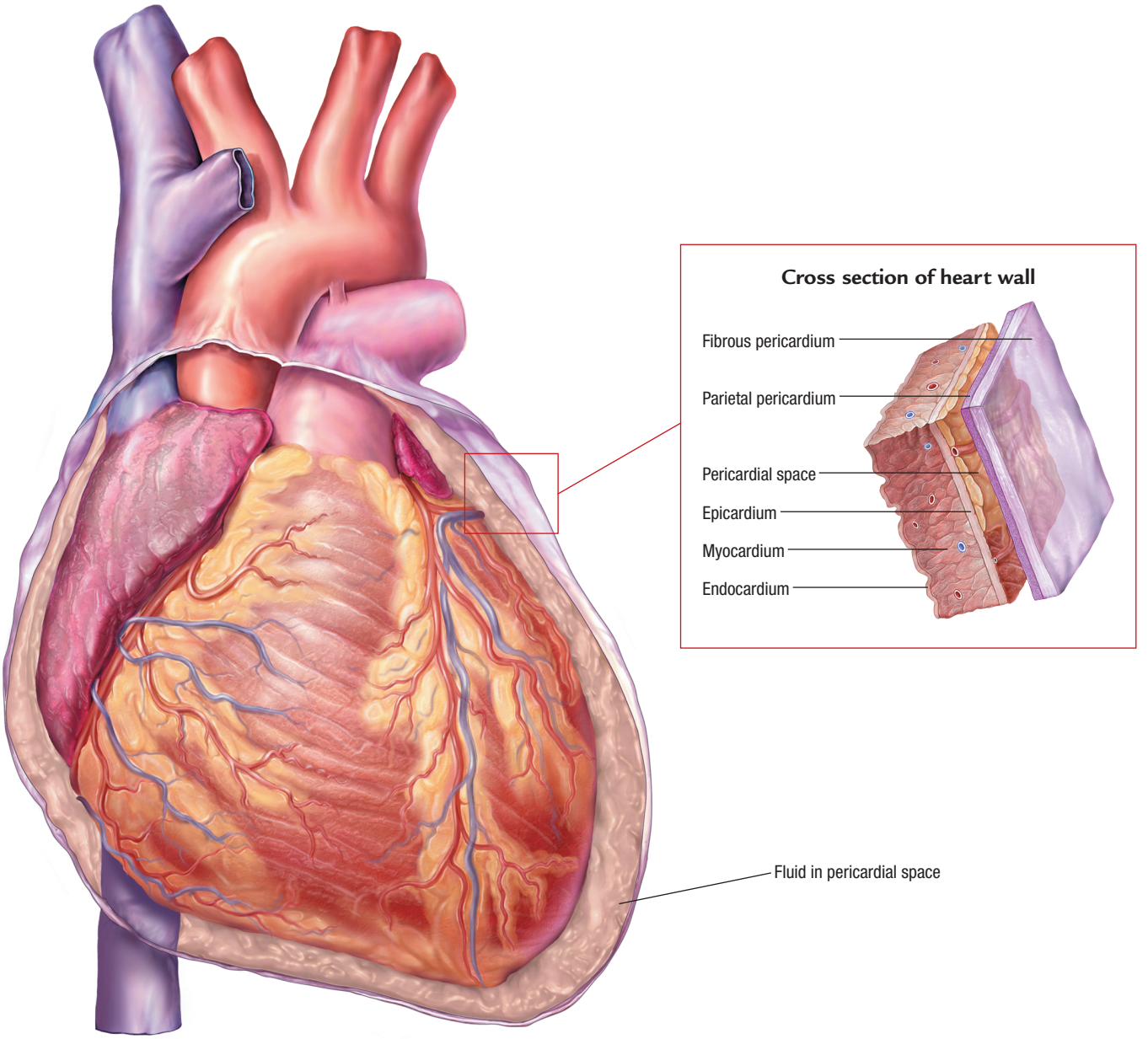
Diagnostic Test Results

- Chest X-rays show a slightly widened mediastinum and possible cardiomegaly. The cardiac silhouette may have a goblet-shaped appearance.
- ECG detects a low-amplitude QRS complex and electrical alternans, an alternating beat-to-beat change in amplitude of the P wave, QRS complex, and T wave. Generalized ST-segment elevation is noted in all leads.
- Pulmonary artery catheterization detects increased right atrial pressure, right ventricular diastolic pressure, and CVP.
- Echocardiography reveals pericardial effusion with signs of right ventricular and atrial compression.

Treatment

- Supplemental oxygen
- Continuous ECG and hemodynamic monitoring
- Pericardiocentesis
- Pericardectomy
- Resection of a portion or all of the pericardium (pericardial window)
- Trial volume loading with crystalloids
- Inotropic drugs, such as isoproterenol or dopamine
- Posttraumatic injury: blood transfusion, thoracotomy to drain reaccumulating fluid, or repair of bleeding sites may be needed
- Heparin-induced tamponade: heparin antagonist protamine sulfate to stop bleeding
- Warfarin-induced tamponade: vitamin K to stop bleeding

CARDIAC TAMPONADE



CARDIOMYOPATHY

Cardiomyopathy is classified as dilated, hypertrophic, or restrictive.

Dilated cardiomyopathy (DCM) results from damage to cardiac muscle fibers; loss of muscle tone grossly dilates all four chambers of the heart, giving the heart a globular shape.

Hypertrophic cardiomyopathy (HCM) is characterized by disproportionate, asymmetrical thickening of the interventricular septum and left ventricular hypertrophy.

Restrictive cardiomyopathy (RCM) is characterized by restricted ventricular filling due to decreased ventricular compliance and endocardial fibrosis and thickening. If severe, it's irreversible.

Causes

Most patients with cardiomyopathy have idiopathic disease, but some are secondary to these possible causes:

- viral infection
- long-standing hypertension
- ischemic heart disease or valvular disease
- chemotherapy
- cardiotoxic effects of drugs or alcohol
- metabolic disease, such as diabetes or thyroid disease.

Pathophysiology

In DCM, extensive damage to cardiac muscle fibers reduces contractility in the left ventricle. As systolic function declines, stroke volume, ejection fraction, and cardiac output fall.

COMPLICATIONS
- Heart failure
- Emboli
- Syncope
- Sudden death

In HCM, hypertrophy of the left ventricle and interventricular septum obstruct left ventricular outflow. The heart compensates for the decreased cardiac output (caused by obstructed outflow) by increasing the rate and force of contractions. The hypertrophied ventricle becomes stiff and unable to relax and fill during diastole. As left ventricular volume diminishes and filling pressure rises, pulmonary venous pressure also rises, leading to venous congestion and dyspnea.

COMPLICATIONS
- Pulmonary hypertension
- Heart failure
- Sudden death

In RCM, left ventricular hypertrophy and endocardial fibrosis limit myocardial contraction and emptying during systole as well as ventricular relaxation and filling during diastole. As a result, cardiac output falls.

COMPLICATIONS
- Heart failure
- Arrhythmias
- Emboli
- Sudden death

Signs and Symptoms

- Shortness of breath
- Peripheral edema
- Fatigue
- Weight gain
- Cough and congestion
- Nausea
- Bloating
- Palpitations
- Syncope
- Chest pain
- Tachycardia

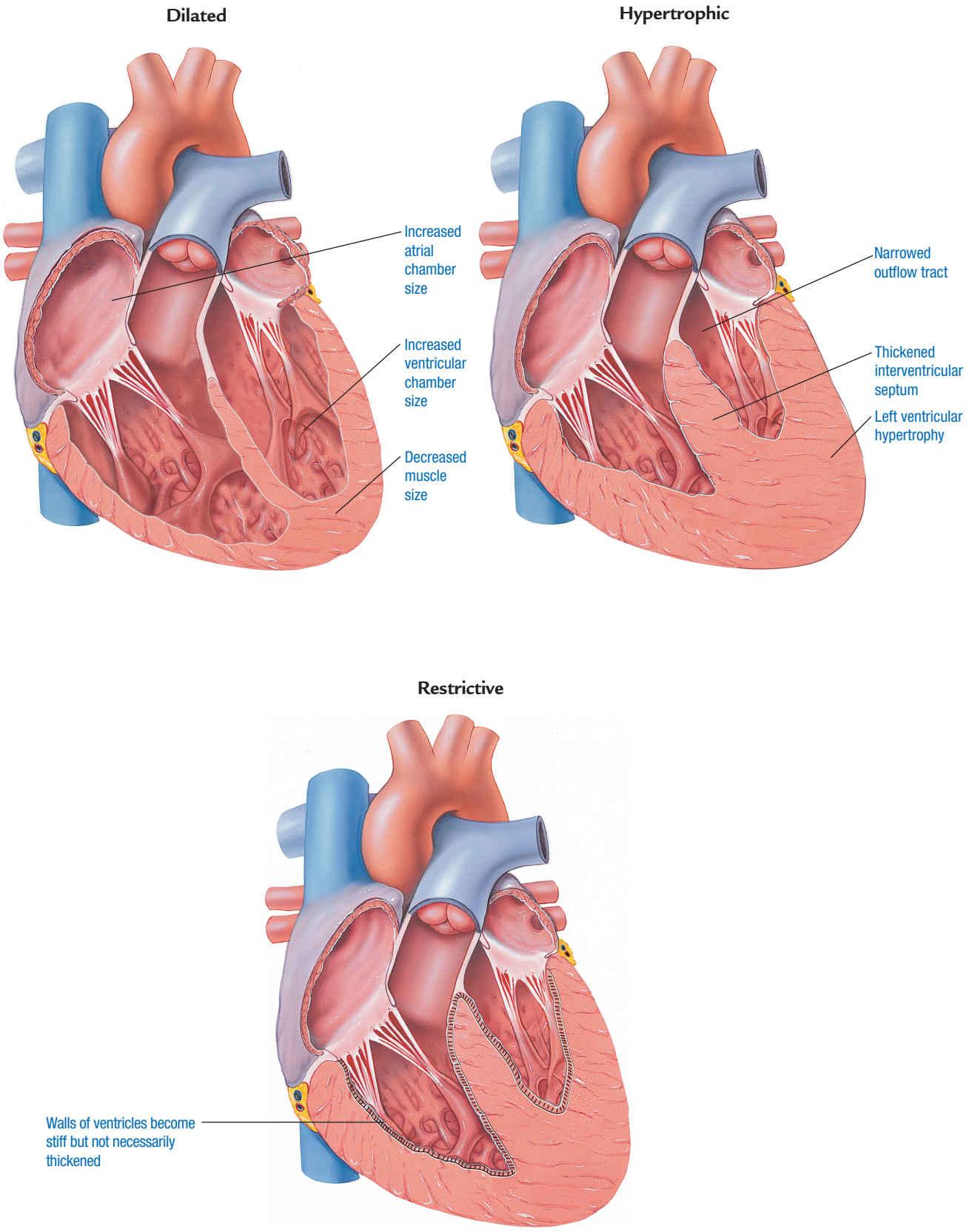
Diagnostic Test Results

- Chest X-rays show cardiomegaly and increase in heart size.
- Echocardiography reveals left ventricular dilation and dysfunction or left ventricular hypertrophy and a thick, asymmetrical intraventricular septum. It can also quantify the outlet left ventricular outflow gradient in HCM.
- Cardiac catheterization shows left ventricular dilation and dysfunction, elevated left ventricular and, commonly, right ventricular filling pressures, and diminished cardiac output.
- Thallium or cardiolite scan usually reveals myocardial perfusion defects.
- Cardiac catheterization reveals elevated left ventricular end-diastolic pressure and, possibly, mitral insufficiency.
- ECG usually shows left ventricular hypertrophy; ST-segment and T-wave abnormalities; Q waves in leads II, III, and aV_F, and in V_4 to V_6; left anterior hemiblock; left axis deviation; and ventricular and atrial arrhythmias.

Treatment

- Treatment of underlying cause
- Control of arrhythmias
- Angiotensin-converting enzyme inhibitors, diuretics, digoxin (not used in HCM), hydralazine, isosorbide dinitrate, beta-adrenergic blockers, antiarrhythmics, and anticoagulants
- Revascularization
- Valve repair or replacement
- Heart transplantation
- Lifestyle modifications, such as quitting smoking; avoiding alcohol; eating a low-fat, low-salt diet; and restricting fluids
- Ventricular myotomy or myectomy
- Mitral valve repair or replacement
- Defibrillator placement with or without biventricular pacing

TYPES OF CARDIOMYOPATHY



CONGENITAL DEFECTS

The most common congenital defects of the heart are atrial septal defect (ASD), coarctation of the aorta, patent ductus arteriosus (PDA), tetralogy of Fallot, transposition of the great arteries, and ventricular septal defect (VSD). Causes of all six defects remain unknown, although some have specific clinical associations.

ATRIAL SEPTAL DEFECT

An opening between the left and right atria permits blood flow from the left atrium to the right atrium rather than from the left atrium to the left ventricle. ASD is associated with Down syndrome.

Pathophysiology

Blood shunts from the left atrium to the right atrium because left atrial pressure is normally slightly higher than right atrial pressure. This difference forces large amounts of blood through a defect that results in right heart volume overload, affecting the right atrium, right ventricle, and pulmonary arteries. Eventually, the right atrium enlarges, and the right ventricle dilates to accommodate the increased blood volume. If pulmonary artery hypertension develops, increased pulmonary vascular resistance and right ventricular hypertrophy follow.

COMPLICATIONS

- Right-sided heart failure
- Heart rhythm abnormalities
- Pulmonary hypertension

Signs and Symptoms

- Fatigue
- Early to midsystolic murmur and low-pitched diastolic murmur
- Fixed, widely split S_2
- Systolic click or late systolic murmur at the apex
- Clubbing of nails and cyanosis with a right-to-left shunt
- Palpable pulsation of the pulmonary artery

COARCTATION OF THE AORTA

Coarctation is a narrowing of the aorta, usually just below the left subclavian artery, near the site where the ligamentum arteriosum joins the pulmonary artery to the aorta. Coarctation of the aorta is associated with Turner's syndrome and congenital abnormalities of the aortic valve.

Pathophysiology

Coarctation of the aorta may develop as a result of spasm and constriction of the smooth muscle in the ductus arteriosus as it closes. Possibly, this contractile tissue extends into the aortic wall, causing narrowing. The obstructive process causes hypertension in the aortic branches above the constriction and diminished pressure in the vessel below the constriction.

Restricted blood flow through the narrowed aorta increases the pressure load on the left ventricle and causes dilation of the proximal aorta and ventricular hypertrophy.

As oxygenated blood leaves the left ventricle, a portion travels through the arteries that branch off the aorta proximal to the coarctation. If PDA is present, the remaining blood travels through the coarctation, mixes with deoxygenated blood from the PDA, and travels to the legs. If the ductus arteriosus is closed, the legs and lower portion of the body must rely solely on the blood that circulates through the coarctation.

COMPLICATIONS

- Rupture of the aorta
- Stroke
- Cerebral aneurysm

Signs and Symptoms

- Heart failure
- Claudication and hypertension
- Headache, vertigo, and epistaxis
- Blood pressure greater in upper than in lower extremities
- Pink upper extremities and cyanotic lower extremities
- Absent or diminished femoral pulses
- Possible murmur
- Possibly, chest and arms more developed than legs

PATENT DUCTUS ARTERIOSUS

The ductus arteriosus is a fetal blood vessel that connects the pulmonary artery to the descending aorta, just distal to the left subclavian artery. Normally, the ductus closes within days to weeks after birth. In PDA, the lumen of the ductus remains open after birth. This creates a left-to-right shunt of blood from the aorta to the pulmonary artery and results in recirculation of arterial blood through the lungs. PDA is associated with premature birth, rubella syndrome, coarctation of the aorta, VSD, and pulmonic and aortic stenosis.

Pathophysiology

The ductus arteriosus normally closes as the neonate takes his first breath but may take as long as 3 months in some infants.

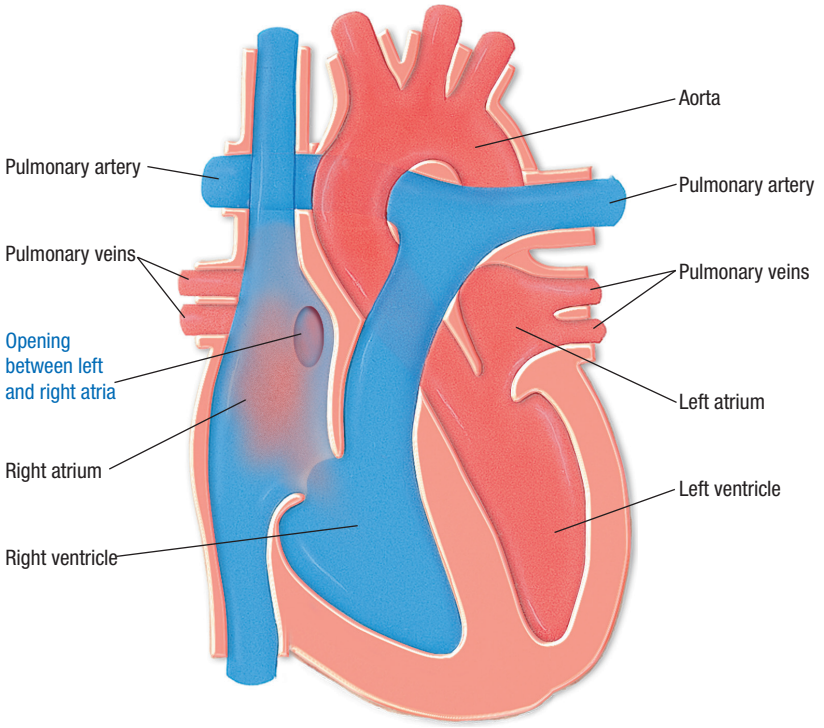
In PDA, relative resistance in pulmonary and systemic vasculature and the size of the ductus determine the quantity of blood that's shunted from left to right. Because of increased aortic pressure, oxygenated blood is shunted from the aorta through the ductus arteriosus to the pulmonary artery. The blood returns to the left side of the heart and is pumped out to the aorta once more.

Increased pulmonary venous return causes increased filling pressure and workload on the left side of the heart as well as left ventricular hypertrophy and possibly heart failure.

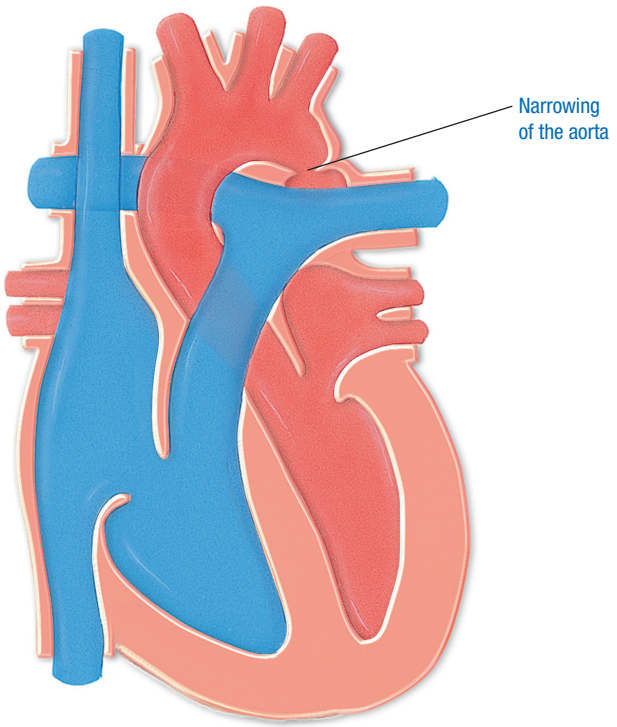
COMPLICATIONS

- Chronic pulmonary hypertension
- Cyanosis
- Left-sided heart failure

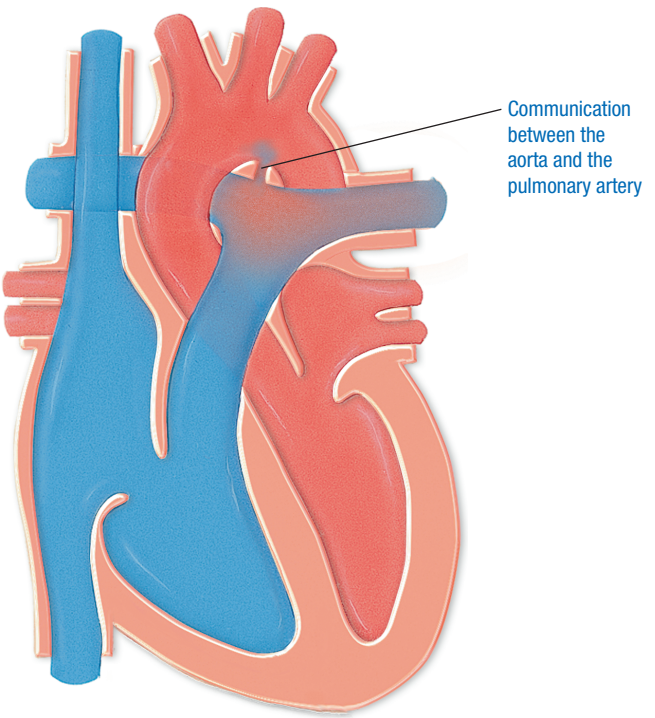
Atrial septal defect



Coarctation of the aorta



Patent ductus arteriosus



Signs and Symptoms

- Respiratory distress with signs of heart failure in infants
- Gibson murmur
- Thrill palpated at left sternal border
- Prominent left ventricular impulse
- Corrigan's pulse
- Wide pulse pressure
- Slow motor development and failure to thrive

TETRALOGY OF FALLOT

Tetralogy of Fallot is a combination of four cardiac defects: VSD, right ventricular outflow tract obstruction, right ventricular hypertrophy, and an aorta positioned above the VSD (overriding aorta). This defect is associated with fetal alcohol syndrome and Down syndrome.

Pathophysiology

Unoxygenated venous blood entering the right side of the heart may pass through the VSD to the left ventricle, bypassing the lungs, or it may enter the pulmonary artery, depending on the extent of the pulmonic stenosis. The VSD usually lies in the outflow tract of the right ventricle and is generally large enough to permit equalization of right and left ventricular pressures. However, the ratio of systemic vascular resistance to pulmonic stenosis affects the direction and magnitude of shunt flow across the VSD.

COMPLICATIONS
- Endocarditis
- Stroke

Signs and Symptoms

- Cyanosis or "blue" spells (Tet spells)
- Clubbing of digits, diminished exercise tolerance, dyspnea on exertion, growth retardation, and eating difficulties
- Squatting to reduce shortness of breath
- Loud systolic murmur and continuous murmur of the ductus
- Thrill at left sternal border
- Right ventricular impulse and prominent inferior sternum

TRANSPOSITION OF GREAT ARTERIES

The aorta rises from the right ventricle and the pulmonary artery from the left ventricle, producing two noncommunicating circulatory systems. This defect is associated with VSD, VSD with pulmonic stenosis, ASD, and PDA.

Pathophysiology

The transposed pulmonary artery carries oxygenated blood back to the lungs, rather than to the left side of the heart. The transposed aorta returns unoxygenated blood to the systemic circulation rather than to the lungs. Communication between the pulmonary and systemic circulations is necessary for survival. In infants with isolated transposition, blood mixes only at the patent foramen ovale and at the PDA, resulting in slight mixing of unoxygenated systemic blood and oxygenated pulmonary blood. In infants with concurrent cardiac defects, greater mixing of blood occurs.

COMPLICATIONS
- Heart failure
- Arrhythmias

Signs and Symptoms

- Hypoxemia, cyanosis, tachypnea, and dyspnea
- Gallop rhythm, tachycardia, hepatomegaly, and cardiomegaly
- Murmurs of ASD, VSD, or PDA; loud S_2
- Diminished exercise tolerance, fatigue, and clubbing

VENTRICULAR SEPTAL DEFECT

VSD is an opening in the septum between the ventricles that allows blood to shunt between the left and right ventricles. However, the defect is usually small and will close spontaneously. VSD is associated with Down syndrome and other autosomal trisomies, renal anomalies, prematurity, fetal alcohol syndrome, PDA, and coarctation of the aorta.

Pathophysiology

In neonates with a VSD, the ventricular septum fails to close completely by 8 weeks' gestation. VSDs are located in the membranous or muscular portion of the ventricular septum and vary in size. Some defects close spontaneously; in other defects, the septum is entirely absent, creating a single ventricle.

A VSD isn't readily apparent at birth because right and left pressures are approximately equal and pulmonary artery resistance is elevated. Alveoli aren't yet completely opened, so blood doesn't shunt through the defect. As the pulmonary vasculature gradually relaxes, between 4 and 8 weeks after birth, right ventricular pressure decreases, allowing blood to shunt from the left to the right ventricle. Initially, large VSD shunts cause left atrial and left ventricular hypertrophy.

COMPLICATIONS
- Right ventricular hypertrophy
- Heart failure
- Endocarditis

Signs and Symptoms

- Failure to thrive
- Loud, harsh systolic murmur (along the left sternal border at the third or fourth intercostal space) and palpable thrill
- Loud, widely split pulmonic component of S_2
- Displacement of point of maximal impulse to left or down
- Prominent anterior chest, cyanosis, and clubbing
- Liver, heart, and spleen enlargement
- Diaphoresis, tachycardia, and rapid, grunting respirations

Diagnostic Test Results

- Chest X-ray reveals cardiomegaly and ventricular and aortic enlargement.
- ECG may be normal or may reveal ventricular hypertrophy or axis deviation.
- Echocardiography detects the presence and size of a defect.
- Fetal echocardiogram can reveal a defect before birth.
- Cardiac catheterization confirms the diagnosis and damage.

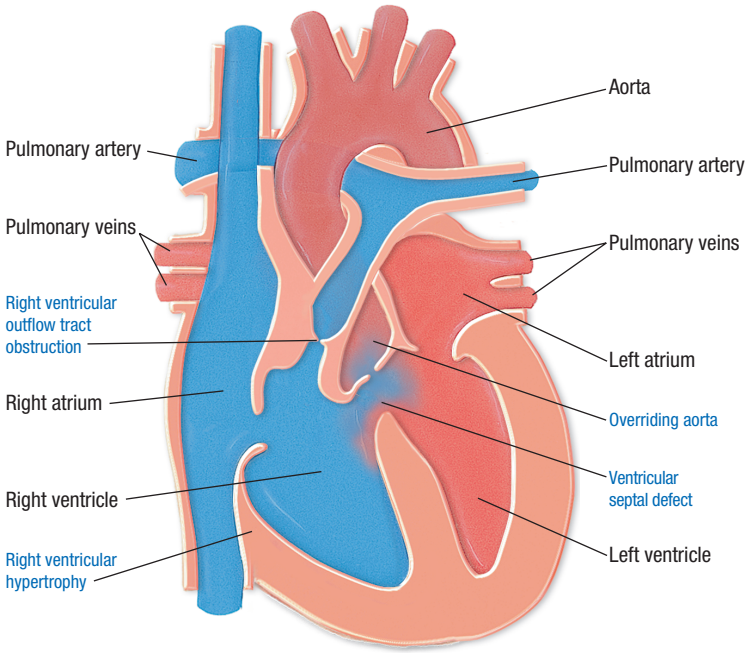
- Arterial blood gas analysis reveals hypoxemia and acid-base disturbances.
- Atrial balloon septostomy (for transposition of the great arteries).

Treatment

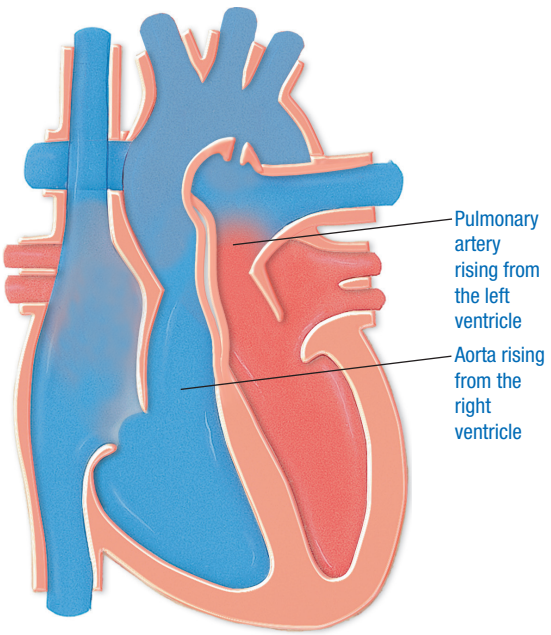
- Surgery
- Medications, such as diuretics, angiotensin-converting enzyme inhibitors, indomethacin (for PDA), and prostaglandin
- Oxygen therapy
- Antibiotic prophylaxis
- Atrial balloon septostomy (for transposition of the great arteries)
- Treatment of complications

CONGENITAL HEART DEFECTS *(continued)*

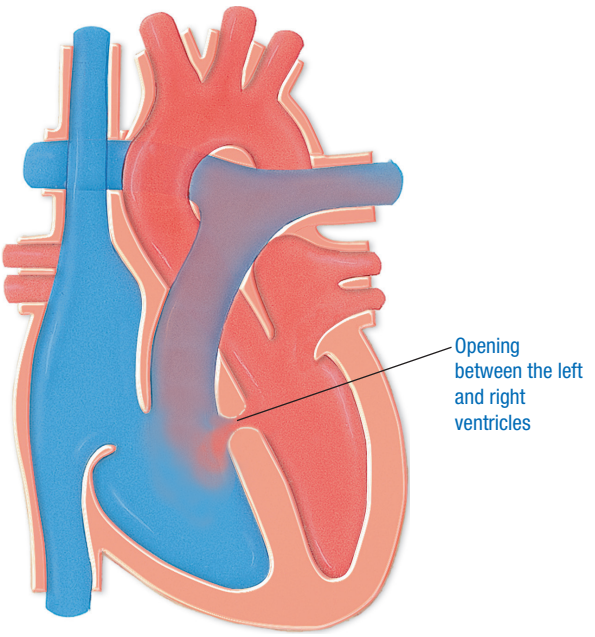
Tetralogy of Fallot



Transposition of great arteries



Ventricular septal defect



CORONARY ARTERY DISEASE

Coronary artery disease (CAD) results from the narrowing of the coronary arteries over time because of atherosclerosis. The primary effect of CAD is a diminished supply of oxygen and nutrients to myocardial tissue because of decreased blood flow.

AGE ALERT

The lifetime risk of CAD after age 40 is 49% for men and 32% for women. As women age, their risk increases.

Causes

- Atherosclerosis (most common)
- Dissecting aneurysm
- Infectious vasculitis
- Syphilis
- Congenital abnormalities
- Radiation to the chest

Pathophysiology

Fatty, fibrous plaques progressively occlude the coronary arteries, reducing the volume of blood that can flow through them and leading to myocardial ischemia.

As atherosclerosis progresses, luminal narrowing is accompanied by vascular changes that impair the ability of the diseased vessel to dilate. The consequent precarious balance between myocardial oxygen supply and demand threatens the myocardium distal to the lesion. When oxygen demand exceeds what the diseased vessel can supply, the result is localized myocardial ischemia.

Myocardial cells become ischemic within 10 seconds after coronary artery occlusion. Transient ischemia causes reversible changes at the cellular and tissue levels, depressing myocardial function. Within several minutes, oxygen deprivation forces the myocardium to shift from aerobic to anaerobic metabolism, leading to accumulation of lactic acid and reduction of cellular pH. Without intervention, this sequence of events can lead to tissue injury or necrosis.

The combination of hypoxia, reduced energy availability, and acidosis rapidly impairs left ventricular function. As the fibers become unable to shorten normally, the force of contractions and velocity of blood flow in the affected myocardial region become inadequate. Moreover, wall motion in the ischemic area becomes abnormal and each contraction ejects less blood from the heart. Restoring blood flow through the coronary arteries restores aerobic metabolism and contractility.

COMPLICATIONS

- Angina pectoris
- Myocardial infarction
- Cardiac arrest

Signs and Symptoms

- Angina (pain may be described as burning, squeezing, or tightness that radiates to the left arm, neck, jaw, or shoulder blade)
- Nausea and vomiting
- Cool extremities and pallor
- Diaphoresis caused by sympathetic stimulation
- Fatigue and dyspnea
- Xanthelasma (fat deposits on the eyelids)

AGE ALERT

The older adult with CAD may be asymptomatic because the sympathetic response to ischemia is impaired. In an active older adult, dyspnea and fatigue are two key signals of ischemia.

Diagnostic Test Results

- ECG shows ischemic changes during anginal episode.
- Stress testing detects ST-segment changes during exercise or pharmacologic stress.
- Coronary angiography reveals the location and degree of coronary artery stenosis or obstruction, collateral circulation, and the condition of the artery beyond the narrowing.
- Myocardial perfusion imaging with thallium 201 or technetium 99m (Cardiolite) may be performed during treadmill exercise to detect ischemic areas of the myocardium.
- Stress echocardiography shows abnormal wall motion in ischemic areas.
- Electron beam computed tomography identifies calcium deposits in coronary arteries.
- Cardiac catheterization reveals blockage in the coronary arteries.
- Lipid profile shows elevated cholesterol levels.

CLINICAL TIP

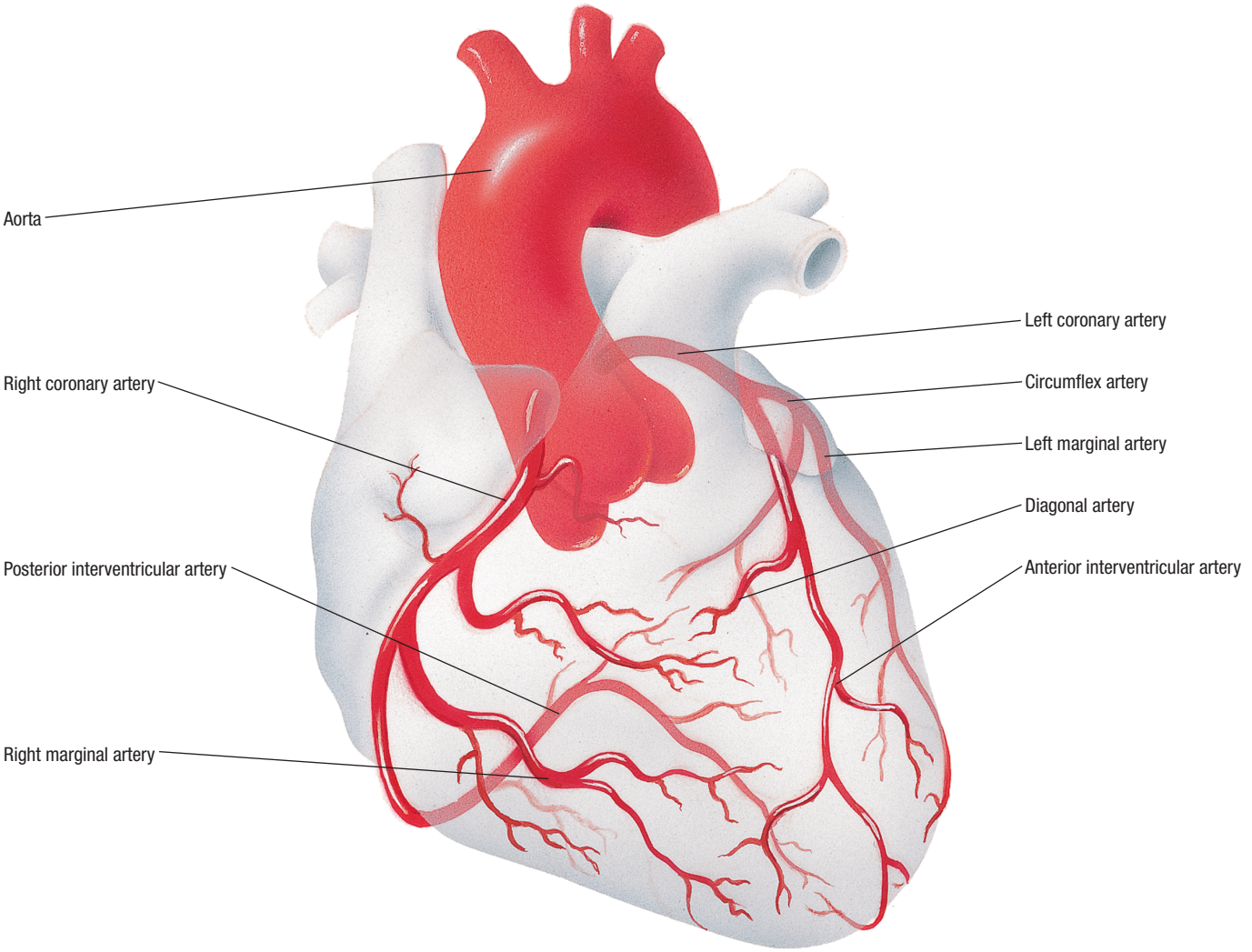
The lipid profile consists of these components:

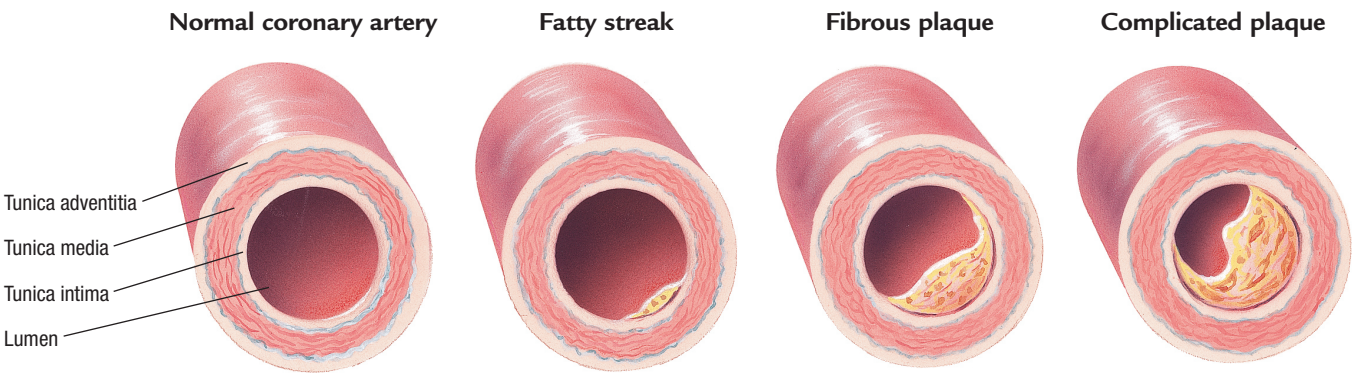
- low-density lipoprotein (LDL) — "bad" lipoprotein; carries most of the cholesterol molecules
- high-density lipoprotein (HDL) — "good" lipoprotein; removes lipids from cells
- apolipoprotein B — major component of LDL
- apolipoprotein A-1 — major component of HDL
- lipoprotein a — one of the most atherogenic lipoproteins.

Treatment

- Drug therapy: angiotensin-converting enzyme inhibitors, thrombolytics, diuretics, glycoprotein IIb/IIIa inhibitors, nitrates, and beta-adrenergic or calcium channel blockers; antiplatelet, antilipemic, and antihypertensive drugs
- Coronary artery bypass graft (CABG) surgery
- "Keyhole" or minimally invasive surgery, an alternative to traditional CABG
- Angioplasty and stent placement
- Atherectomy
- Lifestyle modifications to limit progression of CAD: stopping smoking, exercising regularly, maintaining ideal body weight, and eating a low-fat, low-sodium diet

CORONARY ARTERIES





DEEP VEIN THROMBOSIS

An acute condition characterized by inflammation and thrombus formation, deep vein thrombosis (DVT) mainly refers to thrombosis in the deep veins of the legs. Without treatment, this disorder is typically progressive and can lead to potentially lethal pulmonary embolism. DVT commonly begins with localized inflammation alone (phlebitis), which rapidly provokes thrombus formation. Rarely, venous thrombosis develops without associated inflammation of the vein.

Causes

- Idiopathic
- Endothelial damage
- Accelerated blood clotting
- Reduced blood flow, stasis
- Virchow's triad

Predisposing Risk Factors

- Prolonged bed rest
- Trauma, especially hip fracture
- Surgery, especially hip, knee, or gynecologic surgery
- Childbirth
- Hormonal contraceptives such as estrogens
- Age over 40
- Obesity
- Cancer

Pathophysiology

A thrombus forms when an alteration in the epithelial lining causes platelet aggregation and consequent fibrin entrapment of red and white blood cells and additional platelets. Thrombus formation is more rapid in areas where blood flow is slower, because contact between platelets increases and thrombin accumulates. The rapidly expanding thrombus initiates a chemical inflammatory process in the vessel epithelium, which leads to fibrosis (narrowing of the blood vessel). The enlarging clot may occlude the vessel lumen partially or totally, or it may detach and embolize to lodge elsewhere in the systemic circulation.

COMPLICATIONS

- Pulmonary embolism
- Chronic venous insufficiency

Signs and Symptoms

- Vary with site and length of the affected vein (may produce no symptoms)
- Pain or tenderness
- Fever and chills
- Malaise
- Edema (unilateral edema is most common sign and may be only sign of DVT)
- Redness and warmth over the affected area
- Palpable vein
- Surface veins more visible
- Lymphadenitis

CLINICAL TIP

Some patients may display signs of inflammation.

Diagnostic Test Results

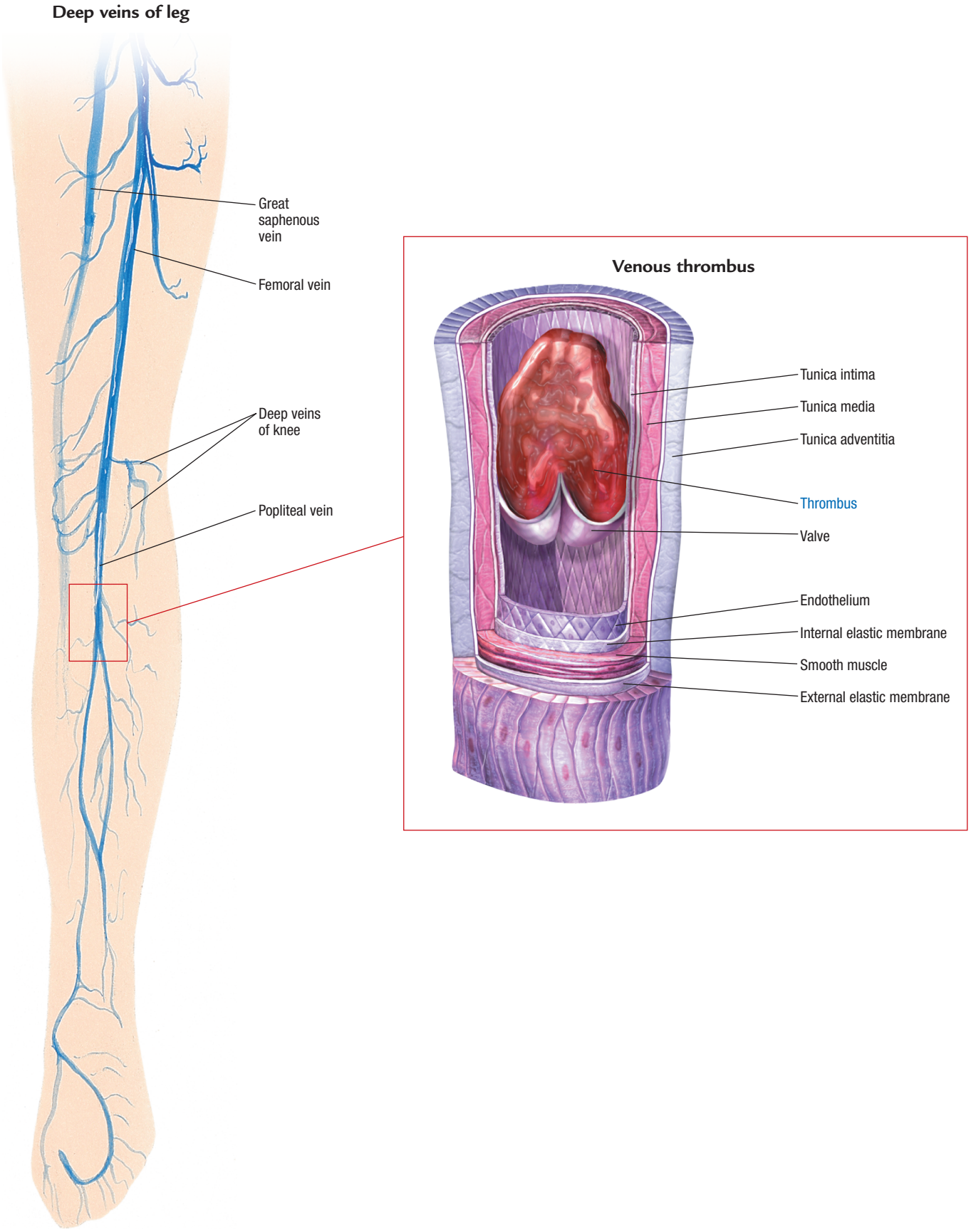
- Duplex Doppler ultrasonography reveals sluggish blood flow.
- Impedance plethysmography shows a difference in blood pressure between the arms and the legs.
- Impedance phlebography shows decreased blood flow.
- Coagulation studies reveal an elevated prothrombin time in the presence of a hypercoagulable state.
- Clotting factor deficiencies can be identified on blood work.
- CT scan is more accurate in identifying presence of DVT.

Treatment

The goals of treatment are to control thrombus development, prevent complications, relieve pain, and prevent recurrence of the disorder. Treatment includes:

- bed rest with elevation of the affected arm or leg
- warm, moist soaks over the affected area
- analgesics
- antiembolism stockings
- anticoagulants (initially, heparin; later, warfarin) — this is most important
- streptokinase
- simple ligation to vein plication, or clipping
- embolectomy and insertion of a vena caval umbrella or filter.

VENOUS THROMBUS



ENDOCARDITIS

Endocarditis, also known as *infective* or *bacterial endocarditis*, is an infection of the endocardium, heart valves, or cardiac prosthesis resulting from bacterial or fungal invasion.

Causes

- I.V. drug abuse
- Prosthetic heart valves
- Mitral valve prolapse
- Rheumatic heart disease

Other Predisposing Conditions

- Congenital abnormalities — coarctation of aorta and tetralogy of Fallot
- Subaortic and valvular aortic stenosis
- Ventricular septal defects
- Pulmonary stenosis
- Marfan syndrome
- Degenerative heart disease
- Syphilis
- Prior history of endocarditis
- Pregnancy
- Arteriovenous dialysis catheters

Native Valve Endocarditis (Non-I.V. Drug Abusers)

- Streptococci, especially *Streptococcus viridans*
- Staphylococci
- Enterococci
- Fungi (rare)

I.V. Drug Abusers

- *Staphylococcus aureus*
- Streptococci
- Enterococci
- Gram-negative bacilli
- Fungi

Prosthetic Valve Endocarditis (Within 60 Days of Insertion)

- Staphylococcal infection
- Gram-negative aerobic organisms
- Fungi
- Streptococci
- Enterococci
- Diphtheroids

Pathophysiology

In endocarditis, bacteremia — even transient bacteremia following dental or urogenital procedures — introduces the pathogen into the bloodstream. This infection causes fibrin and platelets to aggregate on the heart valve tissue and engulf circulating bacteria or fungi that flourish and form friable, wartlike vegetative growths on the valves, the endocardial lining of a heart chamber, or the epithelium of a blood vessel.

COMPLICATIONS

- Left-sided heart failure
- Valvular stenosis
- Myocardial erosion
- Vascular insufficiency
- Embolic events (CVA, arterial thrombosis) from embolism of vegetations

Signs and Symptoms

- Malaise, weakness, and fatigue
- Weight loss and anorexia
- Arthralgia
- Intermittent fever, night sweats, and chills
- Valvular insufficiency
- Loud, regurgitant murmur
- Suddenly changing murmur or new murmur in the presence of fever
- Splenic infarction — left upper quadrant pain radiating to left shoulder and abdominal rigidity
- Renal infarction — hematuria, pyuria, flank pain, and decreased urine output
- Cerebral infarction — hemiparesis, aphasia, and other neurologic deficits
- Pulmonary infarction — cough, pleuritic pain, pleural friction rub, dyspnea, and hemoptysis
- Peripheral vascular occlusion — numbness and tingling in an arm, leg, finger, or toe

Diagnostic Test Results

- Positive blood cultures identify the causative organism.

CLINICAL TIP

Three or more blood cultures in a 24- to 48-hour period (each from a separate venipuncture) identify the causative organism in up to 90% of patients. Blood cultures should be drawn from three different sites with at least 1 to 3 hours between each draw.

- Complete blood count shows normal or elevated white blood cell counts.
- Blood smear shows abnormal histiocytes (macrophages).
- Erythrocyte sedimentation rate is elevated.
- Anemia panel reveals normocytic, normochromic anemia.
- Urinalysis shows proteinuria and microscopic hematuria.
- Serum rheumatoid factor is positive in about one-half of all patients after endocarditis is present for 6 weeks.
- Echocardiography (particularly transesophageal) identifies valvular damage.
- Electrocardiogram shows atrial fibrillation or other arrhythmias.
- Chest X-ray shows the presence of pulmonic emboli.

Treatment

- Penicillin and an aminoglycoside, usually gentamicin

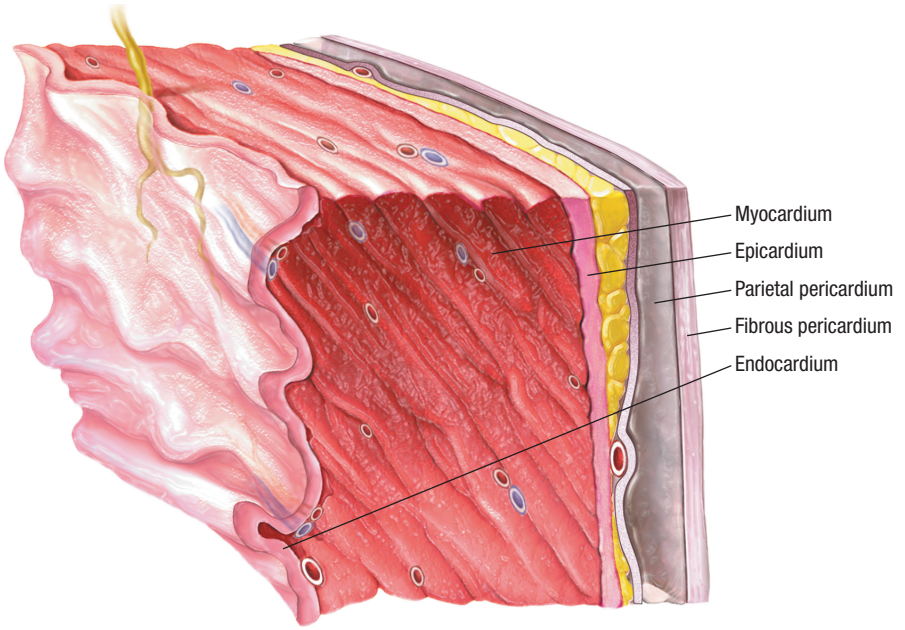
CLINICAL TIP

Any patient who's susceptible to endocarditis, such as those with valvular defects or another predisposing factor, should have prophylactic antibiotics prior to dental or other invasive procedures.

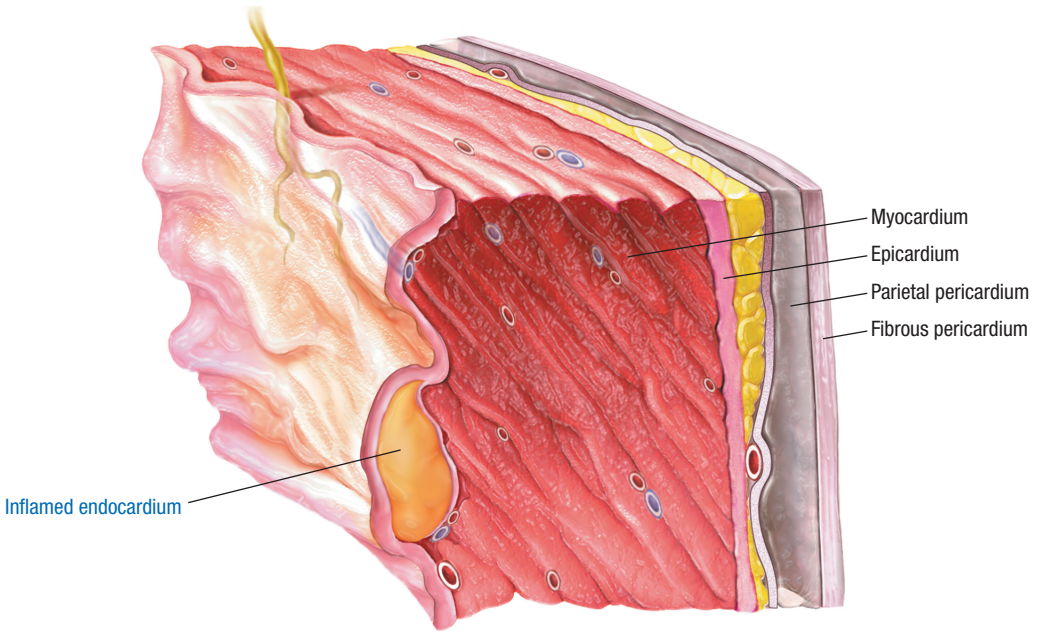
- Bed rest
- NSAIDs or acetaminophen for fever and aches
- Sufficient fluid intake
- Corrective surgery, if refractory heart failure develops or if damage to heart structures occurs
- Replacement of an infected prosthetic valve

TISSUE CHANGES IN ENDOCARDITIS

Normal heart wall



Endocarditis



HEART FAILURE

A syndrome rather than a disease, heart failure occurs when the heart can't pump enough blood to meet the metabolic needs of the body. Heart failure results in intravascular and interstitial volume overload and poor tissue perfusion.

Causes

Abnormal Cardiac Muscle Function

- Myocardial infarction (MI)
- Cardiomyopathy

Abnormal Left Ventricular Volume

- Valvular insufficiency
- High-output states: chronic anemia, arteriovenous fistula, thyrotoxicosis, pregnancy, septicemia, and hypervolemia

Abnormal Left Ventricular Pressure

- Hypertension
- Pulmonary hypertension
- Chronic obstructive pulmonary disease
- Aortic or pulmonic valve stenosis

Abnormal Left Ventricular Filling

- Mitral valve stenosis
- Tricuspid valve stenosis
- Constrictive pericarditis
- Atrial fibrillation
- Hypertension

Pathophysiology

Heart failure may be classified according to the side of the heart affected or by the cardiac cycle involved.

- *Left-sided heart failure:* decreased left ventricular contractile function. Cardiac output falls, and blood backs up into the left atrium and then into the lungs.
- *Right-sided heart failure:* ineffective right ventricular contractile function. Blood backs up into the right atrium and into the peripheral circulation.
- *Systolic dysfunction:* left ventricle can't pump enough blood out to the systemic circulation during systole; the ejection fraction falls. Blood backs up into the pulmonary circulation, pressure rises in the pulmonary venous system, and cardiac output falls.
- *Diastolic dysfunction:* left ventricle can't relax and fill during diastole. The stroke volume falls.

All causes of heart failure eventually reduce cardiac output and trigger compensatory mechanisms that improve cardiac output at the expense of increased ventricular work.

- Increased sympathetic activity enhances peripheral vascular resistance, contractility, heart rate, and venous return. It also restricts blood flow to the kidneys, causing them to secrete renin, which, in turn, converts angiotensinogen to angiotensin I to angiotensin II — a potent vasoconstrictor.
- Angiotensin causes the adrenal cortex to release aldosterone, leading to sodium and water retention and an increase in circulating blood volume. If the renal mechanism persists unchecked, it can aggravate heart failure.
- The increase in end-diastolic ventricular volume causes increased stroke work and volume during contraction, stretching cardiac muscle fibers. The muscle becomes stretched beyond optimum limits and contractility declines.

In heart failure, the body produces counterregulatory substances (prostaglandins, atrial natriuretic factor, and brain natriuretic peptide [BNP]) to reduce the negative effects of volume overload and vasoconstriction.

When blood volume increases in the ventricles, the heart makes these compensations:

- *Short-term:* as the end-diastolic fiber length increases, the ventricular muscle dilates and increases the force of contraction
- *Long-term:* ventricular hypertrophy increases the heart muscles' ability to contract and push its volume of blood into the circulation.

With heart failure, compensation may occur for a long time before signs and symptoms develop.

COMPLICATIONS

- Pulmonary edema
- MI
- Decreased perfusion to major organs

Signs and Symptoms

Left-Sided Heart Failure

- Dyspnea, orthopnea, and paroxysmal nocturnal dyspnea
- Nonproductive cough and crackles
- Hemoptysis
- Tachycardia; S_3 and S_4 heart sounds
- Cool, pale skin

Right-Sided Heart Failure

- Jugular vein distention
- Hepatojugular reflux and hepatomegaly
- Right upper quadrant pain
- Anorexia, fullness, and nausea
- Weight gain, edema, ascites, or anasarca
- Dyspnea, orthopnea, and paroxysmal nocturnal dyspnea

Diagnostic Test Results

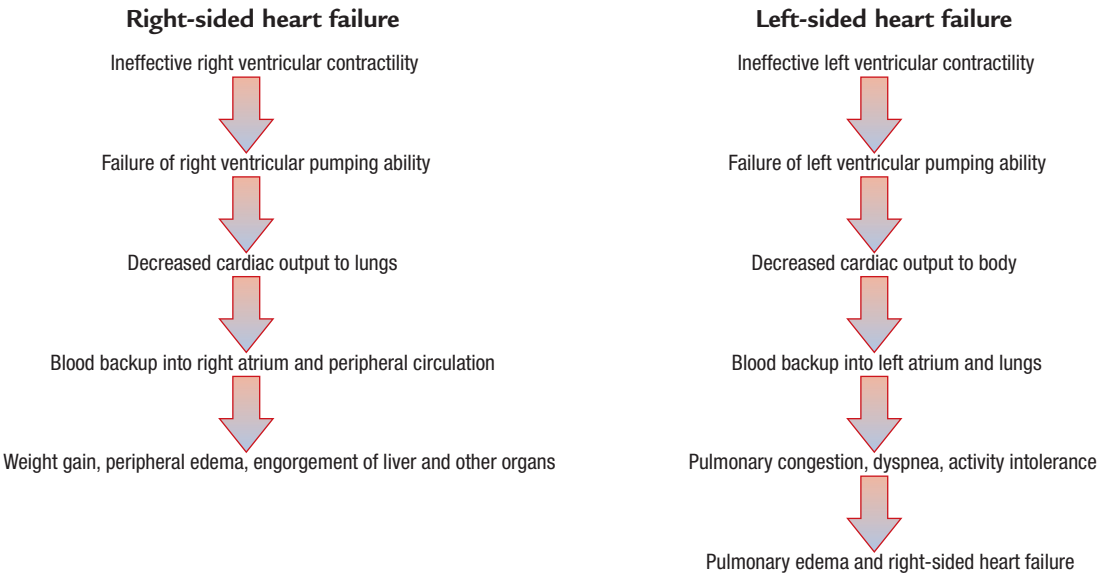
- Chest X-rays show increased pulmonary vascular markings, interstitial edema, or pleural effusion and cardiomegaly.
- ECG shows hypertrophy, ischemic changes, or infarction and may also reveal tachycardia and extrasystoles.
- BNP assay, a blood test, may show elevated levels.
- Echocardiography reveals left ventricular hypertrophy, dilation, and abnormal contractility. Echo can also show valvular abnormalities and inability to relax (diastolic dysfunction).
- Pulmonary artery monitoring typically shows elevated pulmonary artery and pulmonary artery wedge pressures (PAWP), left ventricular end-diastolic pressure in left-sided failure, and right atrial pressure or CVP in right-sided failure.

- Radionuclide ventriculography reveals an ejection fraction less than 40%; in diastolic dysfunction, the ejection fraction may be normal.

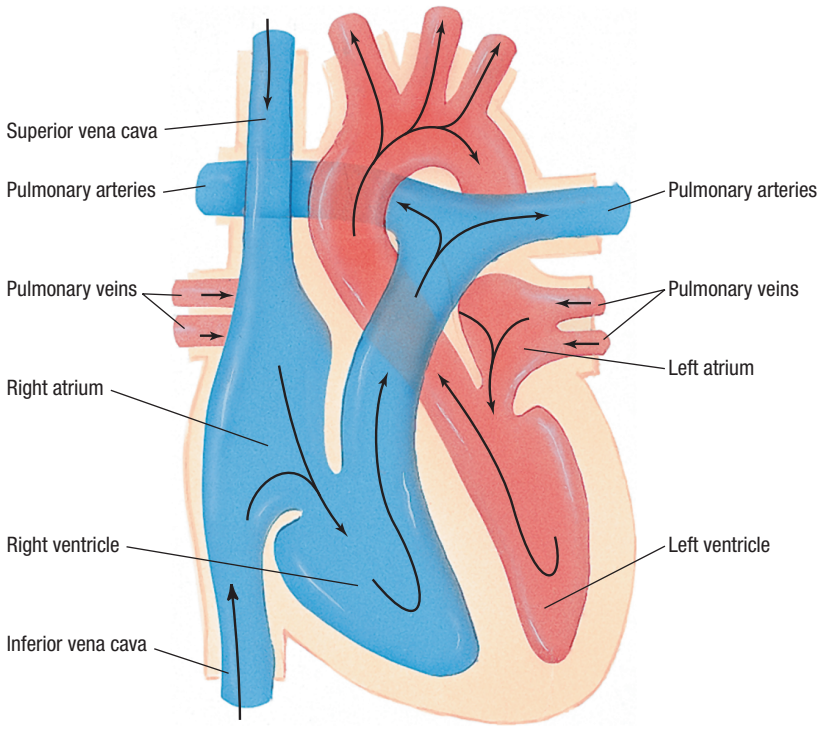
Treatment

- Treatment of the underlying cause, if known
- Angiotensin-converting enzyme inhibitors or ARBs (for patients with left ventricular dysfunction), specific beta-adrenergic blockers (for patients with left ventricular dysfunction), diuretics, digoxin, nitrates, morphine, or oxygen
- Dobutamine, milrinone, and nesiritide (for refractory HF)
- Lifestyle modifications to reduce risk factors
- Coronary artery bypass surgery (if caused by CAD), angioplasty, or heart transplantation
- Placement of prophylactic ICD (with or without Bivent pacing) for patients with low EF

TYPES OF HEART FAILURE



NORMAL CARDIAC CIRCULATION



HYPERTENSION

Hypertension, an elevation in diastolic or systolic blood pressure, occurs as two major types: *primary* (idiopathic), which is the most common, and *secondary*, which results from renal disease or another identifiable cause. Malignant hypertension is a severe, fulminant form of either type.

Causes

Risk Factors for Primary Hypertension

- Family history
- Advancing age
- Race (most common in blacks)
- Obesity
- Tobacco use
- High intake of sodium or saturated fat
- Excessive alcohol consumption
- Sedentary lifestyle and stress

Causes of Secondary Hypertension

- Excess renin
- Mineral deficiencies (calcium, potassium, and magnesium)
- Diabetes mellitus
- Coarctation of the aorta
- Renal artery stenosis or parenchymal disease
- Brain tumor, quadriplegia, and head injury
- Pheochromocytoma, Cushing's syndrome, and hyperaldosteronism
- Thyroid, pituitary, or parathyroid dysfunction
- Hormonal contraceptives, cocaine, epoetin alfa, sympathetic stimulants, monoamine oxidase inhibitors taken with tyramine, estrogen replacement therapy, and nonsteroidal anti-inflammatory drugs
- Pregnancy

Pathophysiology

Arterial blood pressure is a product of total peripheral resistance and cardiac output. Cardiac output is increased by conditions that increase heart rate or stroke volume, or both. Peripheral resistance is increased by factors that increase blood viscosity or reduce the lumen size of vessels.

Several mechanisms may lead to hypertension, including:

Cause of primary hypertension is largely unknown but several mechanisms that may lead to HTN are identified below:

- changes in the arteriolar bed causing increased peripheral vascular resistance
- abnormally increased tone in the sympathetic nervous system that originates in the vasomotor system centers, causing increased peripheral vascular resistance
- increased blood volume resulting from renal or hormonal dysfunction
- arteriolar thickening caused by genetic factors, leading to increased peripheral vascular resistance
- abnormal renin release, resulting in the formation of angiotensin II and aldosterone, which constricts the arteriole and increases blood volume.

Prolonged hypertension increases the workload of the heart as resistance to left ventricular ejection increases. To increase contractile force, the left ventricle hypertrophies, raising the oxygen demand and workload of the heart.

The pathophysiology of secondary hypertension is related to the underlying disease or medication.

COMPLICATIONS

- Stroke
- Myocardial infarction
- Heart failure
- Arrhythmias
- Retinopathy
- Encephalopathy
- Renal failure

Signs and Symptoms

- Generally produces no symptoms
- Serial blood pressure readings classify hypertension:
  - Prehypertension: Systolic blood pressure greater than 120 mm Hg but less than 140 mm Hg or diastolic blood pressure greater than 80 mm Hg but less than 90 mm Hg
  - Stage 1 hypertension: Systolic blood pressure greater than 139 mm Hg but less than 160 mm Hg or diastolic blood pressure greater than 89 mm Hg but less than 100 mm Hg
  - Stage 2 hypertension: Systolic blood pressure greater than 159 mm Hg or diastolic blood pressure greater than 99 mm Hg

Treatment for HTN should begin based on the following guidelines (JNC-8 guidelines):

General population greater than 140/90 mm Hg

Population greater than 60 years old greater than 150/90 mm Hg

Diabetics regardless of age greater than 140/90 mm Hg

- Occipital headache
- Epistaxis possibly due to vascular involvement
- Bruits (renal artery bruits present if renal artery stenosis is the cause)
- Dizziness, confusion, and fatigue
- Blurry vision
- Nocturia
- Edema

Diagnostic Test Results

- Serial blood pressure measurements show elevation. Must be elevated on two separate visits for diagnosis of HTN.
- Urinalysis shows protein, casts, red blood cells, or white blood cells, suggesting renal disease; presence of catecholamines associated with pheochromocytoma; or glucose, suggesting diabetes.
- Blood chemistry reveals elevated blood urea nitrogen and serum creatinine levels suggestive of renal disease or hypokalemia indicating adrenal dysfunction.
- Excretory urography may reveal renal atrophy, indicating chronic renal disease.
- ECG detects left ventricular hypertrophy or ischemia.
- Chest X-rays show cardiomegaly.
- Echocardiography reveals left ventricular hypertrophy, which indicates target organ damage.
- Renal ultrasound identifies renal artery stenosis.

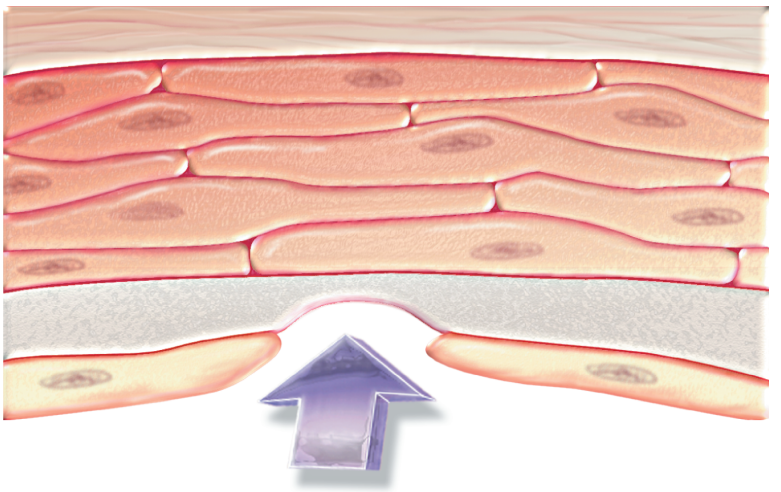
Treatment

Goal is to avoid target organ damage and complications.

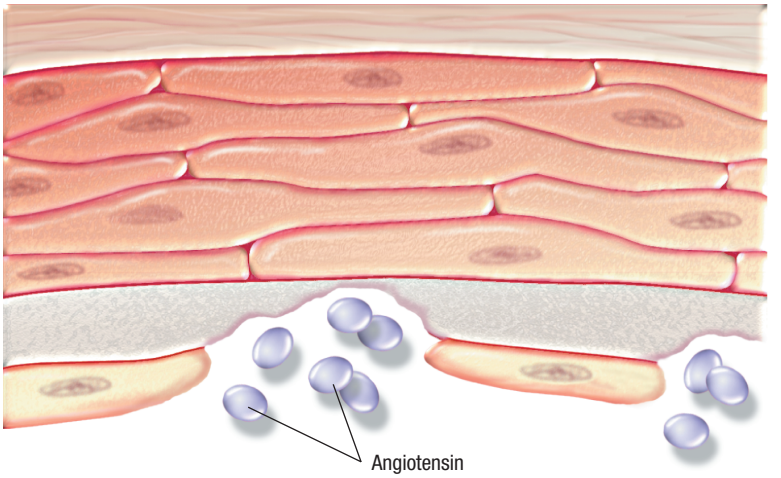
- Treatment of underlying cause if secondary HTN
- Lifestyle modifications to reduce risk factors
- Diuretics
- Angiotensin-converting enzyme inhibitors
- Alpha-receptor agonists
- Beta-adrenergic blockers

BLOOD VESSEL DAMAGE IN HYPERTENSION

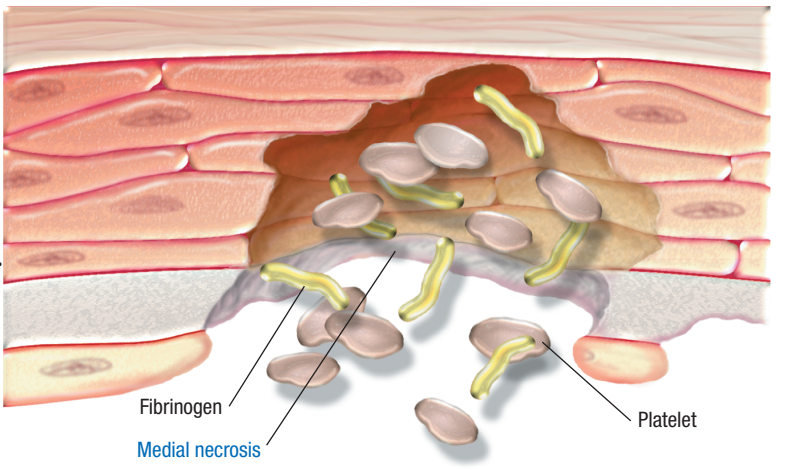
Increased intra-arterial pressure damages the endothelium.



Angiotensin II induces endothelial wall contraction, allowing plasma to leak through interendothelial spaces.



Plasma constituents deposited in the vessel wall cause medial necrosis.



MITRAL VALVE PROLAPSE

Mitral valve prolapse is also called *systolic click-murmur syndrome* and *floppy mitral valve syndrome*. It's probably a congenital abnormality.

Causes

- Autosomal dominant inheritance
- Inherited connective tissue disorders, such as Marfan syndrome, Ehlers-Danlos syndrome, and osteogenesis imperfecta
- Genetic or environmental interruption of valve development during week 5 or 6 of gestation

Pathophysiology

The cusps of the mitral valve are enlarged, thickened, and scalloped, possibly secondary to collagen abnormalities. The chordae tendineae may be longer than usual, allowing the cusps to stretch upward.

COMPLICATIONS

- Mitral regurgitation
- Infective endocarditis
- Arrhythmias

Signs and Symptoms

- Commonly produces no symptoms
- Late systolic regurgitant murmur
- Midsystolic click
- Palpitations, arrhythmias, and tachycardia
- Light-headedness or syncope
- Fatigue, especially in the morning; lethargy; weakness
- Dyspnea and hyperventilation
- Chest tightness and atypical chest pain
- Anxiety, panic attacks, and depression

CLINICAL TIP

The high incidence of mitral valve prolapse (3% to 8% of adults) suggests that it may be a normal variant. It occurs more often in women than in men. Although severe sequelae may occur (such as ruptured chordae tendineae, ventricular failure, emboli, bacterial endocarditis, and sudden death), mortality and morbidity are low. Most affected persons experience no physical limitations. The psychological effects of the diagnosis may be more disabling than the disease process itself.

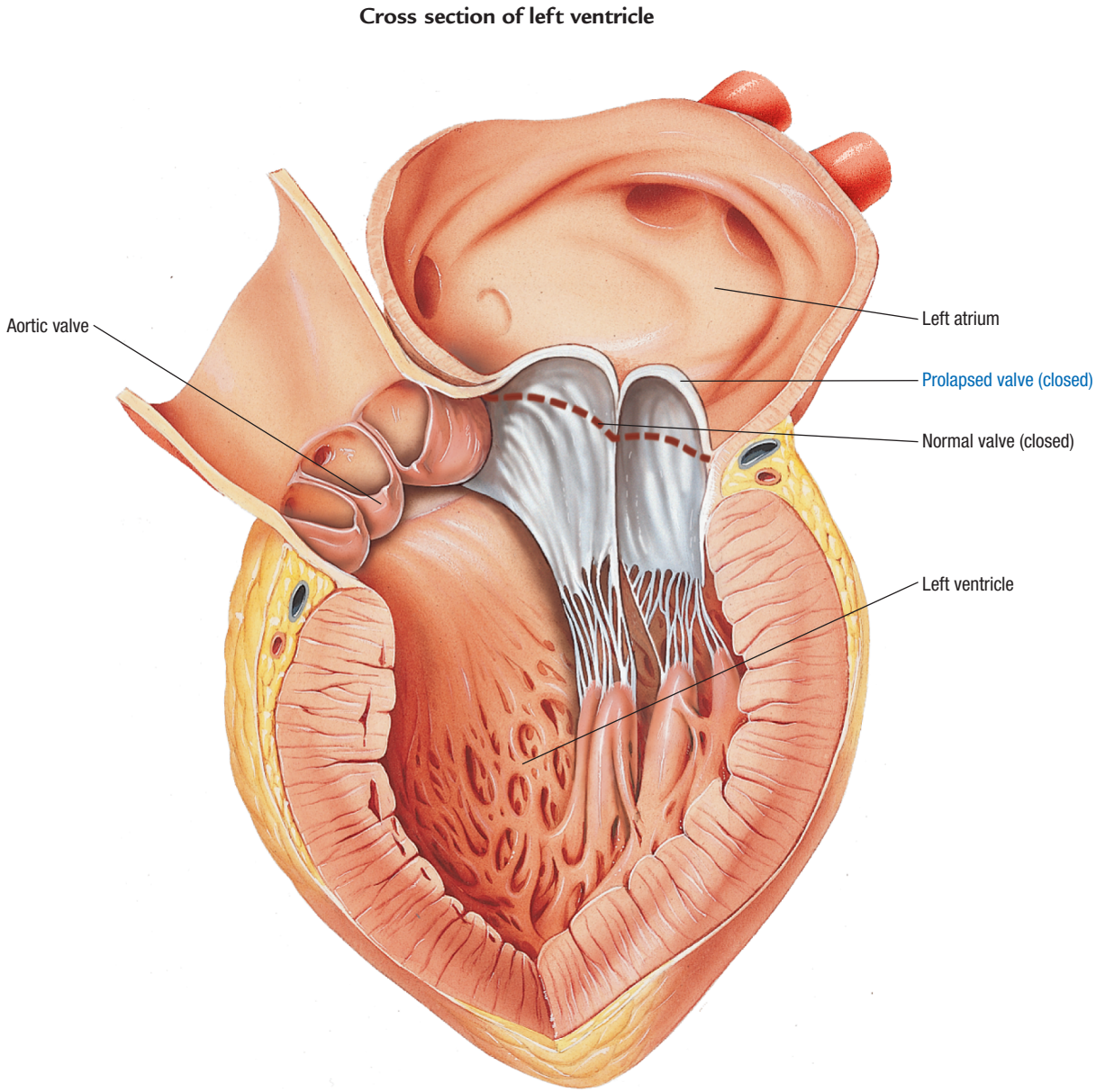
Diagnostic Test Results

- Echocardiography reveals mitral valve prolapse with or without mitral insufficiency.
- ECG (resting and exercise) is usually normal but may show atrial or ventricular arrhythmia.
- Holter monitor detects arrhythmias.

Treatment

- Corresponds to degree of mitral regurgitation
- In the presence of regurgitation, antibiotic prophylaxis before invasive procedures to prevent infective endocarditis (considered moderate risk for SBE)
- Beta-adrenergic blockers
- Measures to prevent hypovolemia, such as avoidance of diuretics, because hypervolemia can decrease ventricular volume, thereby increasing stress on the prolapsed mitral valve
- Surgical repair or valve replacement with severe mitral regurgitation

VALVE POSITION IN MITRAL VALVE PROLAPSE



MYOCARDIAL INFARCTION

In MI, a form of acute coronary syndrome, reduced blood flow through one or more coronary arteries initiates myocardial ischemia and necrosis. (See also "Coronary artery disease," page 60.)

Causes

- Thrombosis
- Coronary artery stenosis or spasm

Predisposing Risk Factors

- Family history of heart disease
- Atherosclerosis, hypertension, diabetes mellitus, and obesity
- Elevated serum triglyceride, total cholesterol, and LDL levels
- Excessive intake of saturated fats, carbohydrates, or salt
- Sedentary lifestyle and tobacco smoking
- Drug use, especially cocaine and amphetamines

Pathophysiology

If coronary artery occlusion causes prolonged ischemia, lasting longer than 30 to 45 minutes, irreversible myocardial cell damage and muscle death occur. Nonocclusive coronary atheromas can rupture and cause thrombus or emboli causing complete occlusion of coronary artery and infarct.

Occlusion of the circumflex branch of the left coronary artery causes a lateral wall infarction; occlusion of the anterior descending branch of the left coronary artery, an anterior wall infarction. True posterior or inferior wall infarctions generally result from occlusion of the right coronary artery or one of its branches.

Right ventricular infarctions can also result from right coronary artery occlusion, can accompany inferior infarctions, and may cause right-sided heart failure. In ST-elevation (transmural) MI, tissue damage extends through all myocardial layers; in non–ST-elevation (subendocardial) MI, damage occurs only in the innermost and, possibly, the middle layers.

All infarcts have a central area of necrosis surrounded by an area of potentially viable hypoxic injury, which may be salvaged if circulation is restored or may progress to necrosis. The zone of injury is surrounded by viable ischemic tissue.

The infarcted myocardial cells release cardiac enzymes and proteins. Within 24 hours, the infarcted muscle becomes edematous and cyanotic. During the next several days, leukocytes infiltrate the necrotic area and begin to remove necrotic cells, thinning the ventricular wall. Scar formation begins by the 3rd week after MI; by the 6th week, scar tissue is well established.

The scar tissue that forms on the necrotic area inhibits contractility. Compensatory mechanisms try to maintain cardiac output. Ventricular dilation may also occur in a process called *remodeling*. MI may cause reduced contractility with abnormal wall motion, altered left ventricular compliance, reduced stroke volume, reduced ejection fraction, and elevated left ventricular end-diastolic pressure.

COMPLICATIONS

- Arrhythmias
- Cardiogenic shock
- Heart failure
- Valve problems

Signs and Symptoms

- Persistent, crushing substernal chest pain that may radiate to the left arm, jaw, neck, or shoulder blades
- Cool extremities, perspiration, anxiety, and restlessness
- Shortness of breath
- Fatigue and weakness
- Nausea and vomiting
- Jugular vein distention

CLINICAL TIP

Signs and symptoms of MI in women may be different or less noticeable than MI in men and may include abdominal pain or "heartburn," back pain, jaw or teeth discomfort, shortness of breath, clammy skin, light-headedness, and unusual or unexplained fatigue.

Diagnostic Test Results

- Serial 12-lead ECG may reveal ST-segment depression or elevation. An ECG also identifies the location of MI, arrhythmias, hypertrophy, and pericarditis. (Non–Q-wave MIs may not have any ECG changes.)
- Serial cardiac enzymes and proteins show a characteristic rise and fall — specifically, CK-MB, the proteins troponin T and I, and myoglobin. Troponin is the most sensitive to cardiac damage.
- Complete blood count and other blood tests show elevated white blood cell count, C-reactive protein level, and erythrocyte sedimentation rate due to inflammation.
- Blood chemistry shows increased glucose levels following the release of catecholamines.
- Echocardiography shows ventricular wall motion abnormalities and detects septal or papillary muscle rupture.
- Chest X-rays show left-sided heart failure or cardiomegaly.
- Nuclear imaging scanning identifies areas of infarction and viable muscle cells.
- Cardiac catheterization identifies the involved coronary artery and provides information on ventricular function and volumes within the heart.

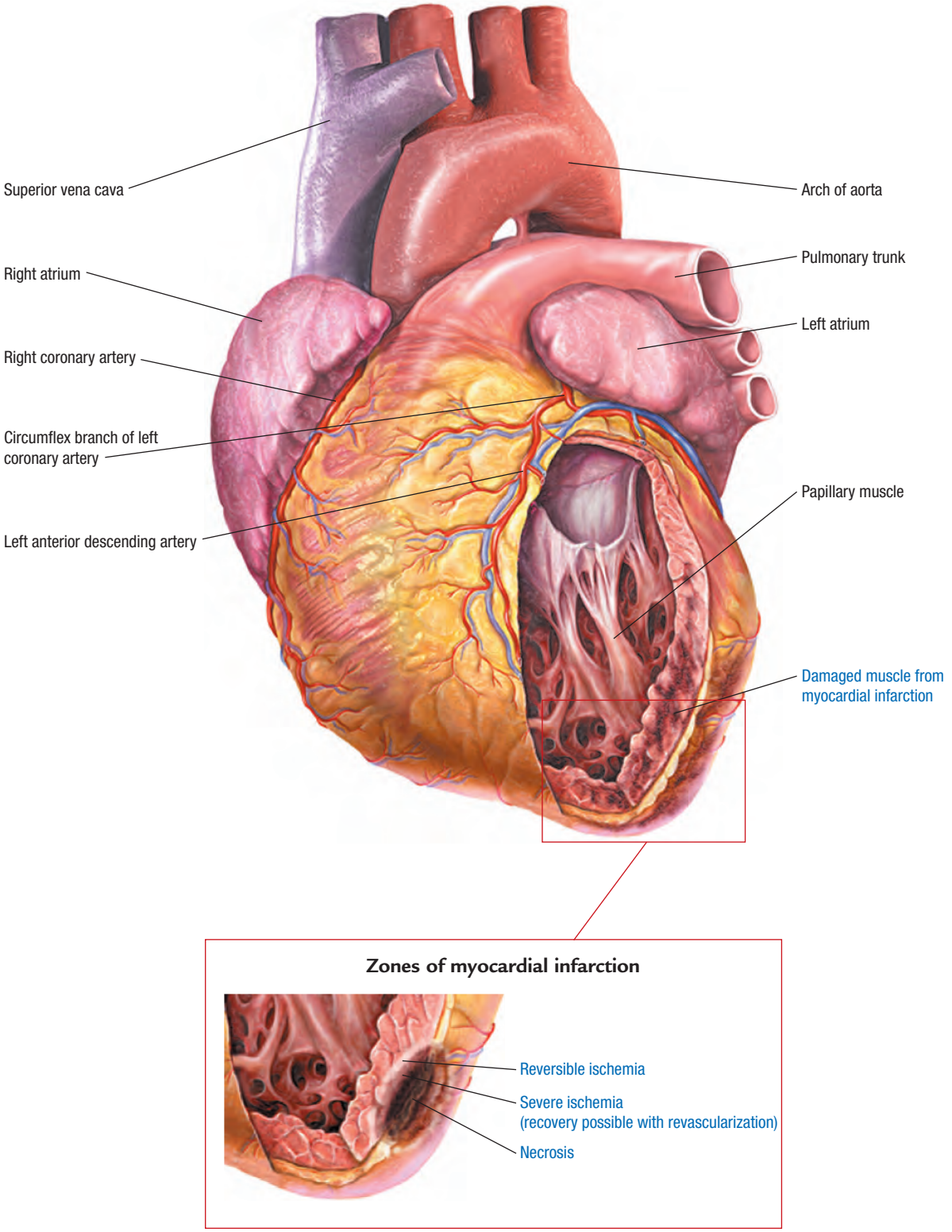
Treatment

Goal of treatment is to intervene to prevent permanent damage to myocardium. Time is muscle.

- Assessment of patients with chest pain in the emergency department within 10 minutes of symptom onset
- Oxygen
- Nitroglycerin
- Morphine
- Aspirin
- Continuous cardiac monitoring
- I.V. fibrinolytic therapy if primary coronary intervention not available
- Glycoprotein IIb/IIIa receptor blockers
- I.V. heparin

- Percutaneous transluminal coronary angioplasty with or without stent placement
- Atropine, lidocaine, transcutaneous pacing patches or a transvenous pacemaker, a defibrillator, and epinephrine
- Beta-adrenergic blockers, angiotensin-converting enzyme inhibitors, and magnesium sulfate

TISSUE DESTRUCTION IN MYOCARDIAL INFARCTION



MYOCARDITIS

Myocarditis is focal or diffuse inflammation of the cardiac muscle (myocardium). It may be acute or chronic and can occur at any age. In many cases, myocarditis causes neither specific cardiovascular symptoms nor electrocardiogram abnormalities, and recovery is usually spontaneous without residual defects.

Causes

- Infections: viral, bacterial, parasitic-protozoan, fungal, or helminthic (such as trichinosis)
- Hypersensitive immune reactions, such as acute rheumatic fever or postcardiotomy syndrome
- Radiation therapy or chemotherapeutic agents
- Toxins, such as lead, chemicals, or cocaine
- Chronic alcoholism
- Systemic autoimmune disorders, such as systemic lupus erythematosus and sarcoidosis

Pathophysiology

Damage to the myocardium occurs when an infectious organism triggers an autoimmune, cellular, or humoral reaction; noninfectious causes can lead to toxic inflammation. In either case, the resulting inflammation may lead to hypertrophy, fibrosis, and inflammatory changes of the myocardium and conduction system. The heart muscle weakens, and contractility is reduced. The heart muscle becomes flabby and dilated, and pinpoint hemorrhages may develop.

COMPLICATIONS

- Left-sided heart failure (occasionally)
- Cardiomyopathy (rare)
- Recurrence of myocarditis
- Chronic valvulitis
- Arrhythmias
- Thromboembolism

Signs and Symptoms

- Fatigue, dyspnea, and palpitations
- Fever
- Chest pain or mild, continuous pressure or soreness in the chest
- Tachycardia and S_3 and S_4 gallops
- Murmur of mitral insufficiency and pericardial friction rub
- Right-sided and left-sided heart failure (jugular vein distention, dyspnea, edema, pulmonary congestion, persistent fever with resting or exertional tachycardia disproportionate to the degree of fever, and supraventricular and ventricular arrhythmias)

CLINICAL TIP

To auscultate for a pericardial friction rub, have the patient sit upright, lean forward, and exhale. Listen over the third intercostal space on the left side of the chest. A pericardial rub has a scratchy, rubbing quality. If you suspect a rub and have difficulty hearing one, have the patient hold his breath.

Diagnostic Test Results

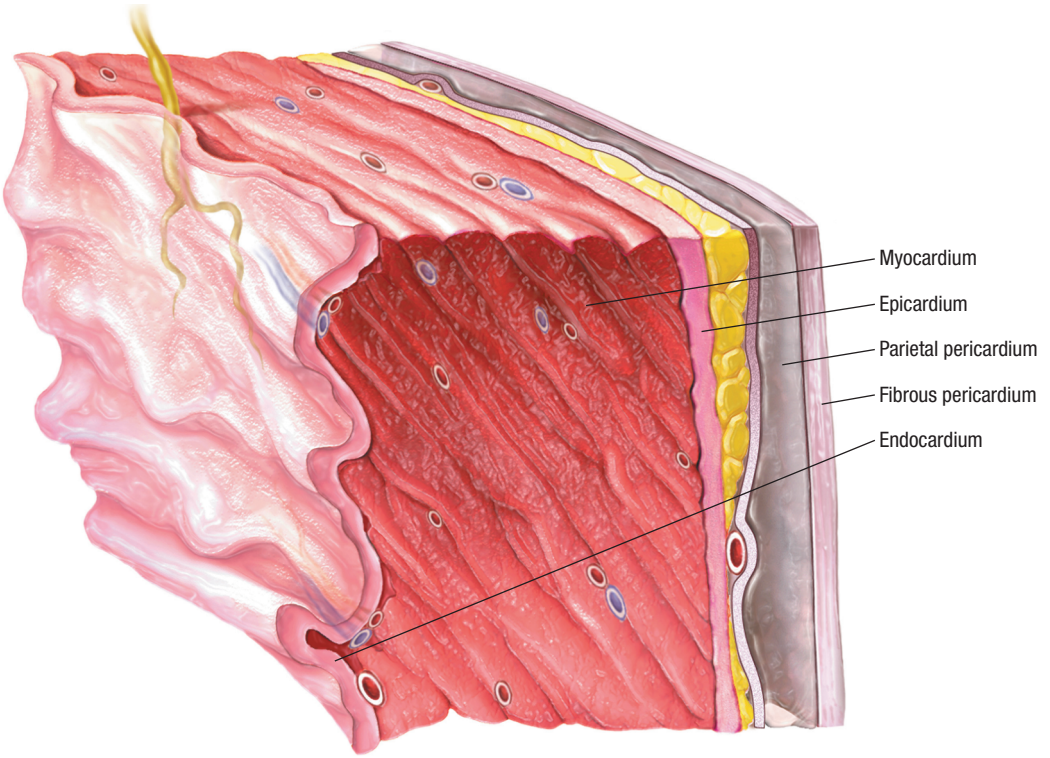
- Blood testing shows elevated levels of creatine kinase (CK), CK-MB, troponin I, troponin T, aspartate aminotransferase, and lactate dehydrogenase. Also, inflammation and infection cause elevated white blood cell count and erythrocyte sedimentation rate.
- Antibody titers are elevated, such as antistreptolysin-O titer, in rheumatic fever.
- Electrocardiogram illustrates diffuse ST-segment and T-wave abnormalities, conduction defects (prolonged PR interval, bundle-branch block, or complete heart block), supraventricular arrhythmias, and ventricular extrasystoles.
- Chest X-rays show an enlarged heart and pulmonary vascular congestion.
- Echocardiography demonstrates some left ventricular dysfunction.
- Radionuclide scanning identifies inflammatory and necrotic changes characteristic of myocarditis.
- Laboratory cultures of stool, throat, and other body fluids identify bacterial or viral causes of infection.
- Endomyocardial biopsy shows damaged myocardial tissue and inflammation.

Treatment

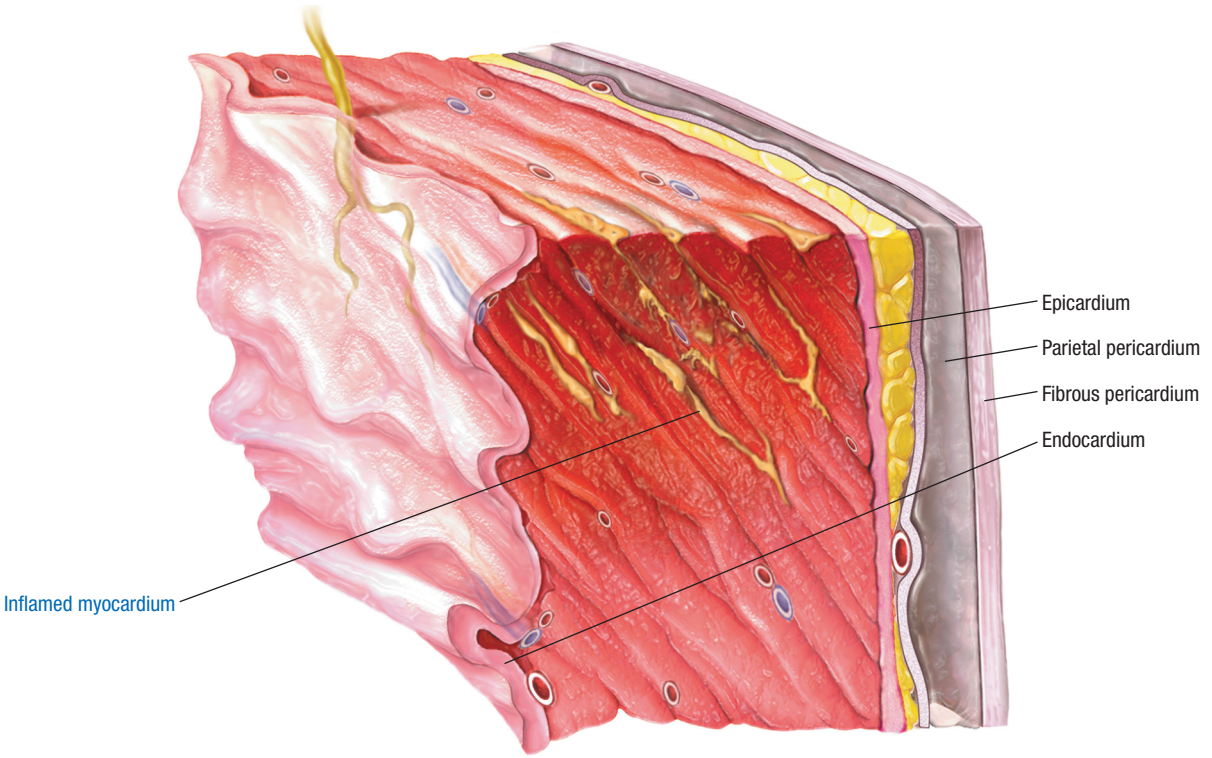
- No treatment for benign self-limiting disease
- Antibiotics
- Antipyretics
- Restricted activity
- Supplemental oxygen therapy
- Sodium restriction and diuretics
- Angiotensin-converting enzyme inhibitors
- Beta-adrenergic blockers
- Digoxin
- Antiarrhythmic drugs, such as quinidine or procainamide
- Temporary pacemaker
- Anticoagulants
- Corticosteroids and immunosuppressants
- Cardiac assist devices or heart transplantation

TISSUE CHANGES IN MYOCARDITIS

Normal heart wall



Myocarditis



PERICARDITIS

Pericarditis is inflammation of the pericardium — the fibroserous sac that envelops, supports, and protects the heart. Acute pericarditis can be fibrinous or effusive, with purulent, serous, or hemorrhagic exudate. Chronic constrictive pericarditis is characterized by dense fibrous pericardial thickening. The prognosis depends on the underlying cause but is generally good in acute pericarditis, unless constriction occurs.

Causes

- Bacterial, fungal, or viral infection
- Neoplasm
- High-dose radiation to the chest
- Uremia
- Hypersensitivity or autoimmune disease
- Previous cardiac injury, such as MI, trauma, or surgery (postcardiotomy syndrome)
- Drugs, such as hydralazine or procainamide
- Idiopathic factors
- Aortic aneurysm
- Myxedema

AGE ALERT

Pericarditis most commonly affects men ages 20 to 50, generally following respiratory illness. It can also occur in children.

Pathophysiology

Pericardial tissue damaged by bacteria or other substances releases chemical mediators of inflammation (prostaglandins, histamines, bradykinins, and serotonin) into the surrounding tissue, thereby initiating the inflammatory process. Friction occurs as the inflamed pericardial layers rub against each other. Histamines and other chemical mediators dilate vessels and increase vessel permeability. Vessel walls then leak fluids and protein (including fibrinogen) into tissues, causing extracellular edema. Macrophages already present in the tissue begin to phagocytize the invading bacteria and are joined by neutrophils and monocytes. After several days, the area fills with an exudate composed of necrotic tissue and dead and dying bacteria, neutrophils, and macrophages. If the cause of pericarditis isn't infection, the exudate may be serous (as with autoimmune disease) or hemorrhagic (as seen with trauma or surgery). Eventually, the contents of the cavity autolyze and are gradually reabsorbed into healthy tissue.

Chronic constrictive pericarditis develops if the chronic or recurrent pericarditis makes the pericardium thick and stiff, encasing the heart in a stiff shell and preventing proper filling during diastole. Consequently, left- and right-side filling pressures rise as stroke volume and cardiac output fall.

COMPLICATIONS

- Pericardial effusion
- Cardiac tamponade
- Shock
- Cardiovascular collapse

Signs and Symptoms

- Pericardial friction rub
- Sharp and (commonly) sudden pain, usually starting over the sternum and radiating to the neck, shoulders, back, and arms
- Shallow, rapid respirations
- Mild fever
- Dyspnea, orthopnea, and tachycardia
- Heart failure
- Muffled, distant heart sounds (if effusion present)
- Pallor, clammy skin, hypotension, pulsus paradoxus, jugular vein distention — indicates tamponade
- Possible progression to cardiovascular collapse
- Fluid retention, ascites, and hepatomegaly
- Pericardial knock in early diastole along the left sternal border produced by restricted ventricular filling
- Kussmaul's sign (increased jugular vein distention on inspiration caused by restricted right-sided filling)

CLINICAL TIP

The pain in pericarditis is commonly pleuritic, increasing with deep inspiration and decreasing when the patient sits up and leans forward, pulling the heart away from the diaphragmatic pleurae of the lungs.

Diagnostic Test Results

- Twelve-lead ECG reveals diffuse ST-segment elevation in the limb leads and most precordial leads that reflect the inflammatory process. Downsloping PR segments and upright T waves are present in most leads. QRS segments may be diminished when pericardial effusion exists. Arrhythmias, such as atrial fibrillation and sinus arrhythmias, may occur. In chronic constrictive pericarditis, there may be low-voltage QRS complexes, T-wave inversion or flattening, and P mitral (wide P waves) in leads I, II, and V_6.
- Blood testing reveals an elevated erythrocyte sedimentation rate as a result of the inflammatory process and a normal or elevated white blood cell count, especially in infectious pericarditis. C-reactive protein may be elevated.
- Blood cultures identify an infectious cause.
- Antistreptolysin-O titers are positive if pericarditis is caused by rheumatic fever.
- Purified protein derivative skin tests are positive if pericarditis is caused by tuberculosis.
- Echocardiography shows an echo-free space between the ventricular wall and the pericardium and reduced pumping action of the heart.
- Chest X-rays show an enlarged cardiac silhouette with a water bottle shape caused by fluid accumulation if pleural effusion is present.
- Chest or heart magnetic resonance imaging shows enlargement of the heart and signs of inflammation.

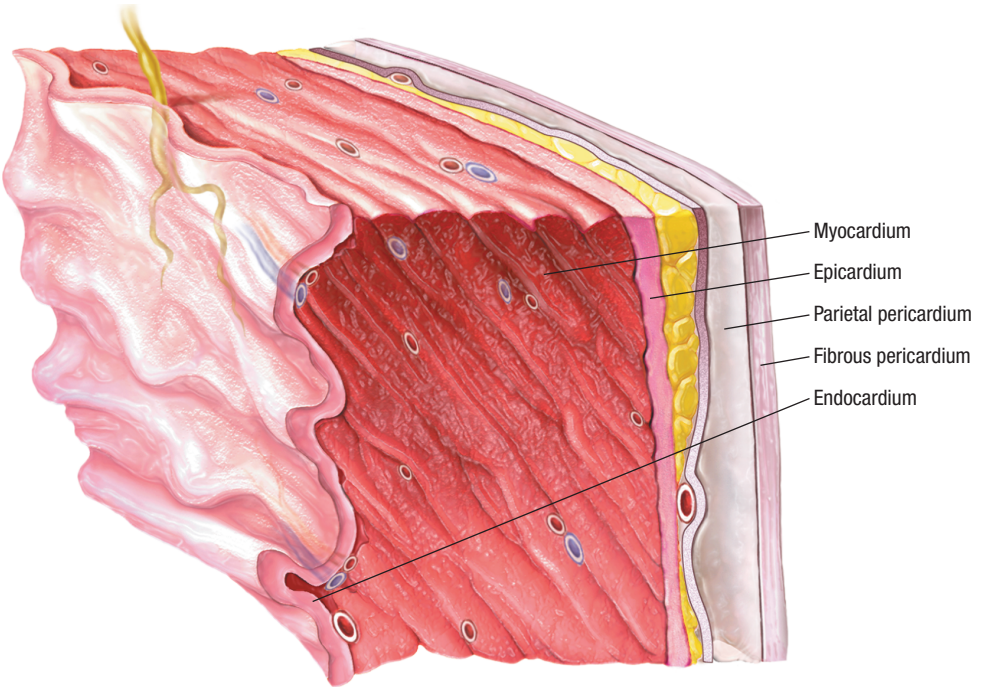
Treatment

- Bed rest as long as fever and pain persist
- Treatment of the underlying cause, if it can be identified

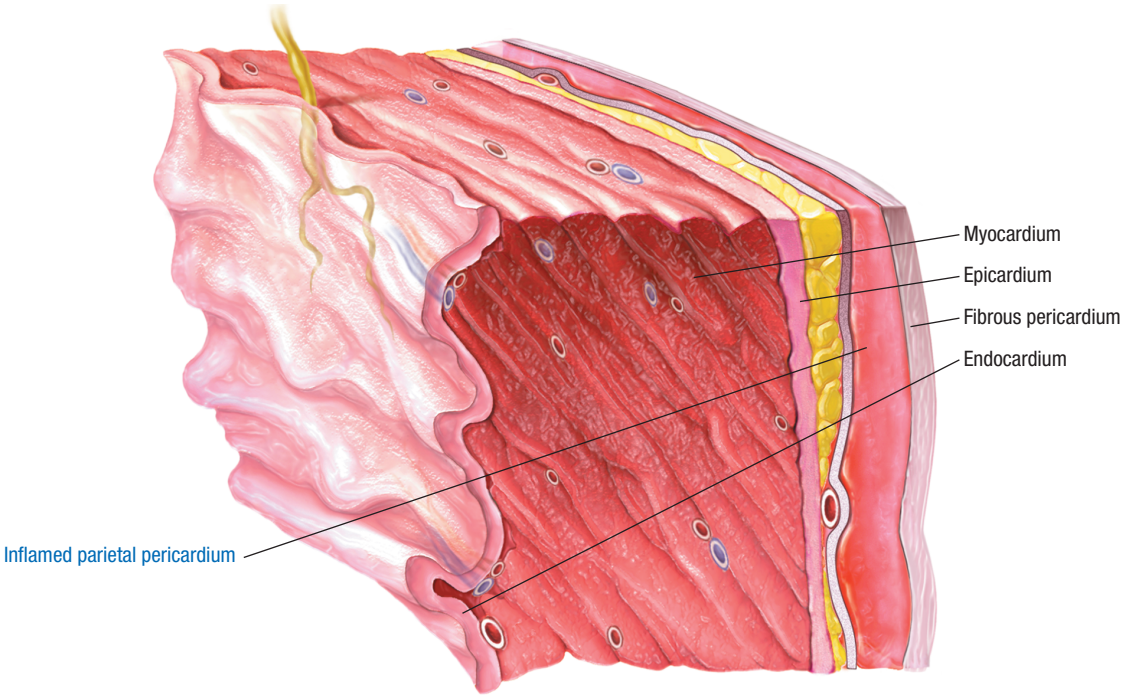
- Nonsteroidal anti-inflammatory drugs and corticosteroids
- Antibacterial, antifungal, or antiviral therapy
- Partial or total pericardectomy
- Diuretics
- Pericardiocentesis

TISSUE CHANGES IN PERICARDITIS

Normal heart wall



Pericarditis



RAYNAUD'S DISEASE

Raynaud's disease is one of several primary disorders characterized by episodic spasms of the small peripheral arteries and arterioles, precipitated by exposure to cold or stress. This condition occurs bilaterally and usually affects the hands or, less often, the feet. It's benign, requires no specific treatment, and has no serious sequelae. Raynaud's *phenomenon*, however, is secondary to any of several connective disorders — such as scleroderma, systemic lupus erythematosus, or polymyositis — and progresses to ischemia, gangrene, and amputation. Distinguishing between the two disorders is difficult because some patients experience mild symptoms of Raynaud's disease for several years and then develop overt connective tissue disease, especially scleroderma.

AGE ALERT

Raynaud's disease is most prevalent in females, particularly between puberty and age 40.

Causes

Raynaud's Disease

- Unknown; family history is a risk factor

Raynaud's Phenomenon

- Connective tissue disorders, such as scleroderma, rheumatoid arthritis, systemic lupus erythematosus, or polymyositis
- Pulmonary hypertension
- Thoracic outlet syndrome
- Arterial occlusive disease
- Myxedema
- Trauma
- Serum sickness
- Exposure to heavy metals
- Long-term exposure to cold, vibrating machinery (such as operating a jackhammer), or pressure to the fingertips (as in typists and pianists)

Pathophysiology

Raynaud's disease is a syndrome of episodic constriction of the arterioles and arteries of the extremities, resulting in pallor and cyanosis of the fingers and toes. Several mechanisms may account for the reduced digital blood flow, including:

- intrinsic vascular wall hyperactivity to cold
- increased vasomotor tone due to sympathetic stimulation
- antigen-antibody immune response (most likely because abnormal immunologic test results accompany Raynaud's phenomenon).

COMPLICATIONS

- Ischemia
- Gangrene
- Amputation

Signs and Symptoms

- Bilateral blanching (pallor) of the fingers after exposure to cold or stress:
  - Vasoconstriction or vasospasm reduces blood flow.
  - Cyanosis caused by increased oxygen extraction results from sluggish blood flow.
  - Spasm resolves, and fingers turn red (rubor) as blood rushes back into the arterioles.
- Cold and numbness
- Throbbing, aching pain, swelling, and tingling
- Trophic changes (as a result of ischemia), such as sclerodactyly, ulcerations, or chronic paronychia

Diagnostic Test Results

- Antinuclear antibody (ANA) titer identifies autoimmune disease as an underlying cause of Raynaud's phenomenon; further tests must be performed if the ANA titer is positive.
- Doppler ultrasonography shows reduced blood flow if the symptoms result from arterial occlusive disease.

Treatment

- Avoiding triggers, such as cold, and mechanical or chemical injury
- Smoking cessation and avoidance of decongestants and caffeine to reduce vasoconstriction
- Calcium channel blockers, such as nifedipine, diltiazem, and nicardipine
- Alpha-adrenergic blockers, such as phenoxybenzamine or reserpine
- Biofeedback and relaxation exercises to reduce stress and improve circulation
- Sympathectomy or amputation

PROGRESSIVE VASCULAR CHANGES

RHEUMATIC HEART DISEASE

A systemic inflammatory disease of childhood, acute rheumatic fever develops after infection of the upper respiratory tract with group A beta-hemolytic streptococci. It mainly involves the heart, joints, central nervous system, skin, and subcutaneous tissues and commonly recurs. Rheumatic heart disease refers to the cardiac manifestations of rheumatic fever and includes pancarditis during the early acute phase and chronic valvular disease later. Cardiac involvement develops in up to 50% of patients.

Rheumatic fever tends to run in families, lending support to the existence of genetic predisposition. Environmental factors also seem to be significant in the development of the disorder.

Causes

Rheumatic fever is caused by group A beta-hemolytic streptococcal pharyngitis.

Rheumatic fever appears to be a hypersensitivity reaction to a group A beta-hemolytic streptococcal infection. Because few persons (3%) with streptococcal infections contract rheumatic fever, altered host resistance must be involved in its development or recurrence.

Pathophysiology

The antigens of group A streptococci bind to receptors in the heart, muscle, brain, and synovial joints, causing an autoimmune response. Because the antigens of the streptococcus are similar to some antigens of the body's own cells, antibodies may attack healthy body cells.

Carditis may affect the endocardium, myocardium, or pericardium during the early acute phase.

COMPLICATIONS

- Chronic valvular disease
- Pericarditis
- Pericardial effusion

Signs and Symptoms

- Polyarthritis or migratory joint pain
- Erythema marginatum
- Subcutaneous nodules
- Chorea
- Streptococcal infection a few days to 6 weeks before onset of symptoms
- Fever
- New or worsening mitral or aortic murmur
- Pericardial friction rub
- Chest pain, commonly pleuritic
- Dyspnea, tachypnea, nonproductive cough, bibasilar crackles, and edema

Diagnostic Test Results

- During the acute phase, complete blood count reveals an elevated white blood cell count and an elevated erythrocyte sedimentation rate.
- Hemoglobin and hematocrit are decreased because of suppressed erythropoiesis during inflammation.
- C-reactive protein is positive, especially during the acute phase.
- Cardiac enzyme levels are increased in severe carditis.
- Antistreptolysin-O titer is elevated in 95% of patients within 2 months of onset.
- Throat cultures show the presence of group A beta-hemolytic streptococci; however, they usually occur in small numbers.
- ECG shows a prolonged PR interval.
- Chest X-rays show normal heart size or cardiomegaly, pericardial effusion, or heart failure.
- Echocardiography detects valvular damage and pericardial effusion, measures chamber size, and provides information on ventricular function.
- Cardiac catheterization provides information on valvular damage and left ventricular function.

CLINICAL TIP

Jones Criteria for diagnosis require either two major criteria or one major criterion and two minor, plus evidence of a previous group A streptococcal infection.

Major Criteria

- Carditis
- Migratory joint pain
- Sydenham's chorea
- Subcutaneous nodules, usually near tendons or bony prominences of joints, especially the elbows, knuckles, wrists, and knees
- Erythema marginatum

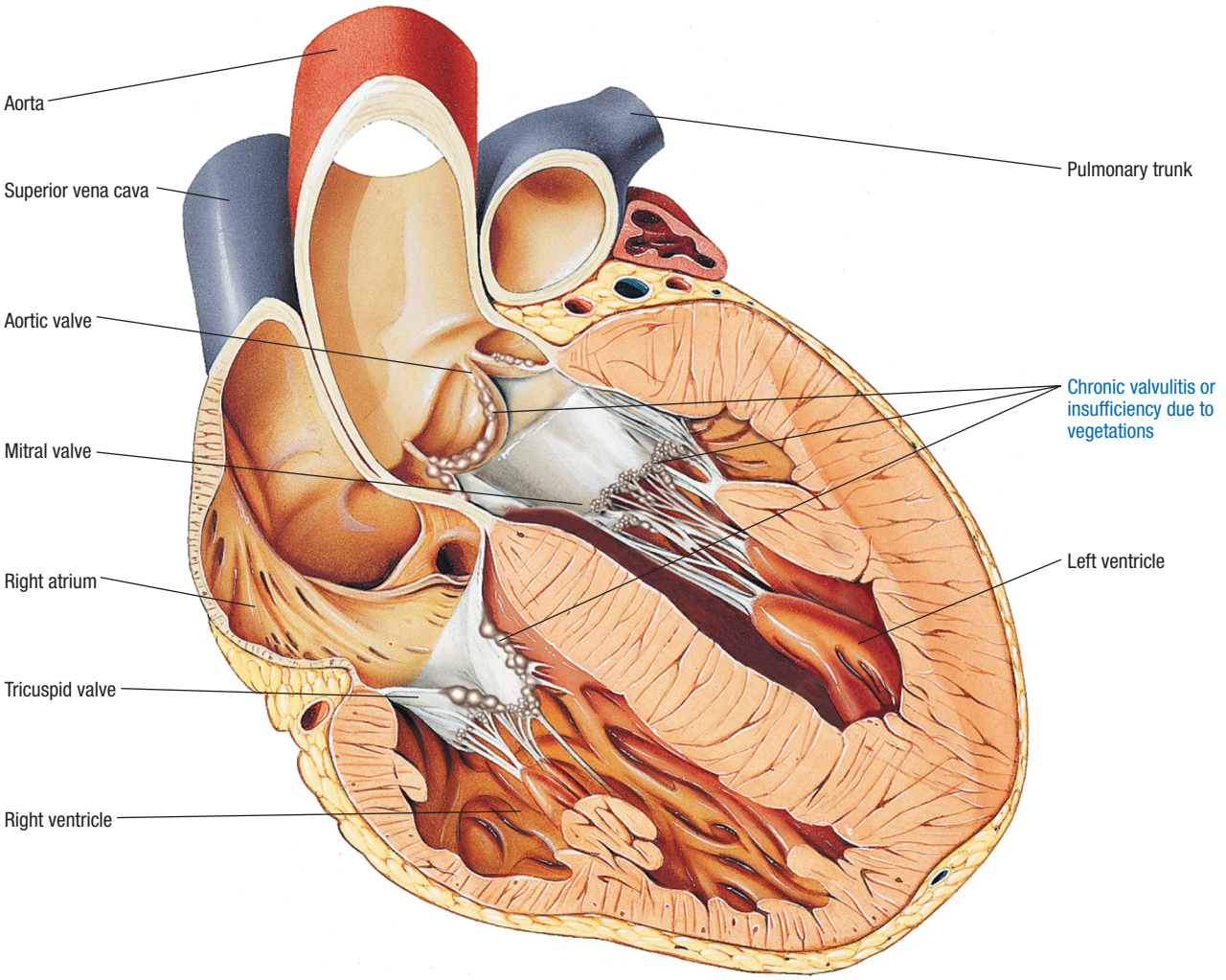
Minor Criteria

- Fever
- Arthralgia
- Elevated acute phase reactants
- Prolonged PR interval

Treatment

- Prompt treatment of all group A beta-hemolytic streptococcal pharyngitis with oral penicillin V or I.M. benzathine penicillin G; erythromycin for patients with penicillin hypersensitivity
- Salicylates
- Corticosteroids
- Strict bed rest for about 5 weeks
- Sodium restriction, angiotensin-converting enzyme inhibitors, digoxin, and diuretics
- Corrective surgery, such as commissurotomy, valvuloplasty, or valve replacement for severe mitral or aortic valvular dysfunction that causes persistent heart failure
- Secondary prevention of rheumatic fever, which begins after the acute phase subsides:
  - monthly I.M. injections of penicillin G benzathine or daily doses of oral penicillin V or sulfadiazine
  - continued treatment, usually for at least 5 years or until age 21, whichever is longer
- Prophylactic antibiotics for dental work and other invasive or surgical procedures (in the presence of valve disorders only. Rheumatic fever without valve disease does increase the risk of SBE beyond the general population

SEQUELAE OF RHEUMATIC HEART DISEASE



SHOCK

Shock is a clinical syndrome that leads to reduced perfusion of tissues and organs and organ failure. Shock can be classified into three categories: distributive (neurogenic and septic), cardiogenic, and hypovolemic.

Causes

Neurogenic Shock

- Spinal cord injury and spinal anesthesia
- Vasomotor center depression
- Severe pain
- Medications
- Hypoglycemia

Septic Shock

- Gram-negative bacteria and gram-positive bacteria
- Viruses, fungi, rickettsiae, parasites, yeast, protozoa, and mycobacteria

Cardiogenic Shock

- MI, most common cause
- Heart failure and cardiomyopathy
- Pericardial tamponade
- Pulmonary embolism

Hypovolemic Shock

- Blood loss (most common cause)
- GI fluid loss, renal loss, and fluid shifts causing severe dehydration
- Burns

Pathophysiology

Each type of shock has three stages.

Compensatory stage: When arterial pressure and tissue perfusion fall, compensatory mechanisms are activated to maintain cardiac output and perfusion to the heart and brain. As the baroreceptors in the carotid sinus and aortic arch sense a drop in blood pressure, epinephrine and norepinephrine are secreted to increase peripheral resistance, blood pressure, and myocardial contractility. Reduced blood flow to the kidney activates the renin-angiotensin-aldosterone system, causing vasoconstriction and sodium and water retention.

Progressive stage: When compensatory mechanisms can't maintain cardiac output, tissues become hypoxic. Cells switch to anaerobic metabolism and lactic acid accumulates, producing metabolic acidosis. Tissue hypoxia promotes the release of endothelial mediators, leading to venous pooling and increased capillary permeability. Sluggish blood flow increases the risk of disseminated intravascular coagulation (DIC).

Irreversible (refractory) stage: Inadequate perfusion damages cell membranes, lysosomal enzymes are released, and energy stores are depleted, leading to cell death. Lactic acid continues to accumulate, increasing capillary permeability and the movement of fluid out of the vascular space, further contributing to hypotension. Perfusion to the coronary arteries is reduced, causing myocardial depression and a further reduction in cardiac output. Circulatory failure and respiratory failure occur.

COMPLICATIONS

- Kidney or brain damage (cardiogenic and hypovolemic)
- Liver damage (cardiogenic)
- Respiratory or cardiac failure (septic) in all types of shock

Signs and Symptoms

Compensatory Stage

- Tachycardia, bounding pulse, and tachypnea
- Reduced urinary output
- Cool, pale skin (or warm, dry skin in septic shock)

Progressive Stage

- Hypotension
- Narrowed pulse pressure; weak, rapid, thready pulse
- Cold, clammy skin; cyanosis and shallow respirations

Irreversible Stage

- Unconsciousness and absent reflexes
- Rapidly falling blood pressure; weak pulse
- Slow, shallow, or Cheyne-Stokes respirations

Diagnostic Test Results

- Hematocrit is reduced in hemorrhage or elevated in other types of shock caused by hypovolemia.
- Blood, urine, and sputum cultures identify the organism responsible for septic shock.
- Coagulation studies may detect coagulopathy from DIC.
- Complete blood count reveals increased white blood cell count and erythrocyte sedimentation rate.
- Blood chemistry reveals elevated blood urea nitrogen and creatinine levels and elevated serum glucose (in early stages).
- Serum lactate increases secondary to anaerobic metabolism.
- Elevated cardiac enzymes and proteins indicate MI as a cause of cardiogenic shock.
- Arterial blood gas analysis reveals respiratory alkalosis.
- Urine specific gravity will be elevated in response to the effects of antidiuretic hormone.
- Chest X-rays will be normal in early stages; pulmonary congestion may be seen in later stages.
- ECG may show arrhythmias, ischemic changes, and an MI.
- Echocardiography reveals valvular abnormalities.

Treatment

- Identification and treatment of the underlying cause
- Maintaining a patent airway, oxygen and mechanical ventilation, and continuous cardiac monitoring
- I.V. fluids, crystalloids, colloids, or blood products

Neurogenic Shock

- Vasopressor drugs

Septic Shock

- Treatment with drotrecogin alfa (Xigris) antibiotics and inotropic and vasopressor drugs

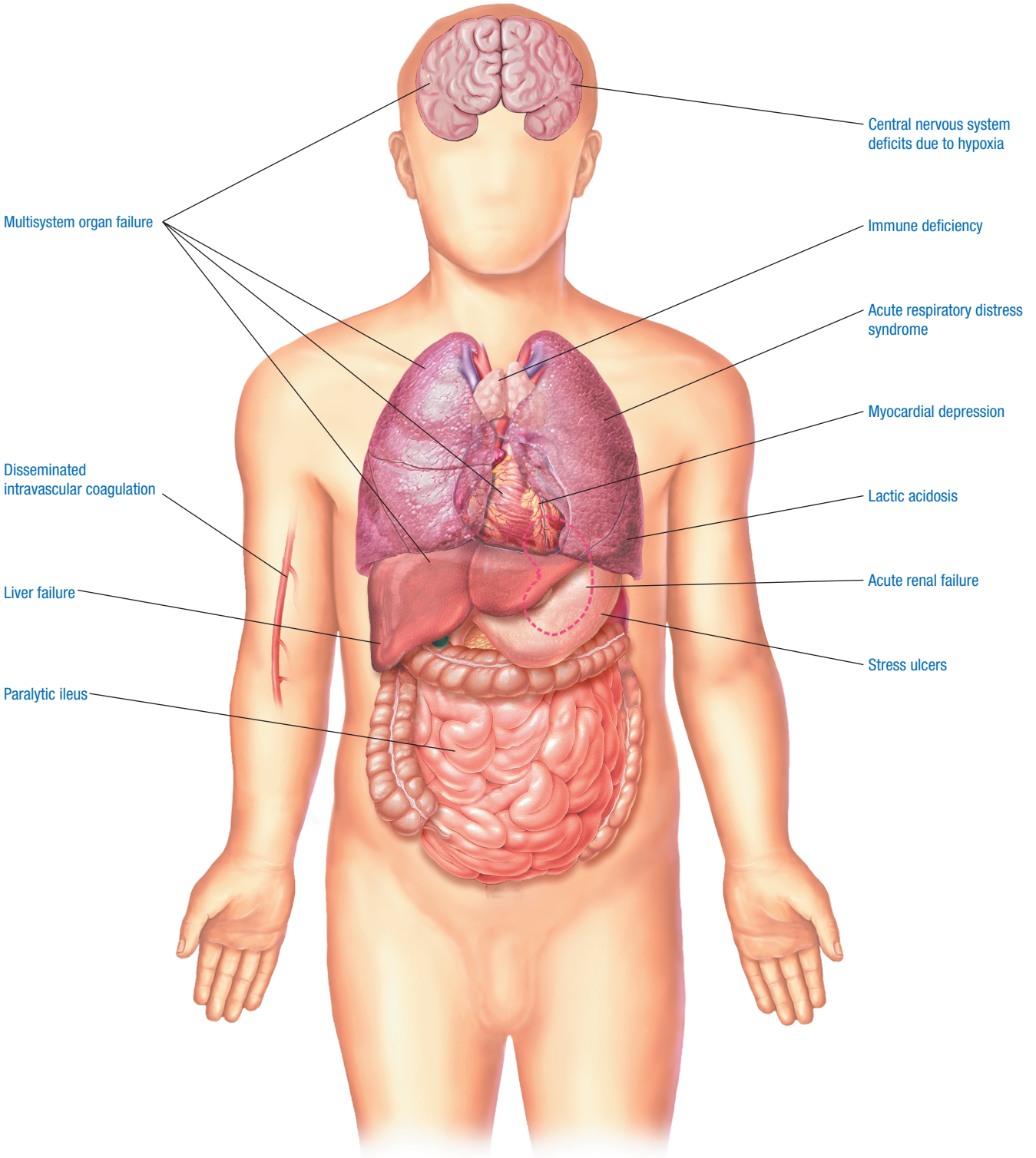
Cardiogenic Shock

- Inotropic drugs, vasodilators, and diuretics
- Intra-aortic balloon pump therapy
- Thrombolytic therapy or coronary artery revascularization
- Ventricular assist device
- Heart transplantation

Hypovolemic Shock

- Pneumatic antishock garment

MULTIORGAN SYSTEM EFFECTS OF SHOCK



VALVULAR HEART DISEASE

In valvular heart disease, three types of mechanical disruptions can occur: stenosis, or narrowing, of the valve opening (called insufficiency, incompetence, or regurgitation); incomplete closure of the valve; or prolapse of the valve.

Causes

The causes of valvular heart disease are varied and differ for each type of valve disorder.

Mitral Stenosis

- Rheumatic fever
- Congenital anomalies

Mitral Insufficiency

- Rheumatic fever
- Mitral valve prolapse
- Myocardial infarction
- Severe left ventricular failure
- Ruptured chordae tendineae
- Marfan syndrome

Aortic Insufficiency

- Rheumatic fever
- Syphilis
- Hypertension
- Endocarditis
- Marfan syndrome

Aortic Stenosis

- Congenital
- Bicuspid aortic valve
- Rheumatic fever
- Atherosclerosis

Pulmonic Stenosis

- Congenital
- Rheumatic fever (rare)

Pathophysiology

Pathophysiology of valvular heart disease varies according to the valve and the disorder.

Mitral stenosis: Structural abnormality, fibrosis, or calcification obstructs blood flow from the left atrium to the left ventricle. Left atrial volume and pressure rise, and the chamber dilates. Greater resistance to blood flow causes pulmonary hypertension, right ventricular hypertrophy, and right-sided heart failure. Inadequate filling of the left ventricle causes low cardiac output.

COMPLICATIONS
- Pulmonary edema
- Atrial fibrillation
- Pulmonary hypertension
- Right-sided heart failure
- Emboli
- Stroke

Mitral insufficiency: An abnormality of the mitral leaflets, mitral annulus, chordae tendineae, papillary muscles, left atrium, or left ventricle can lead to mitral regurgitation. Blood from the left ventricle flows back into the left atrium during systole, and the atrium enlarges to accommodate the backflow. The left ventricle also dilates to accommodate the increased volume of blood from the atrium and to compensate for diminishing cardiac output. Ventricular hypertrophy and increased end-diastolic pressure raise pulmonary artery pressure.

COMPLICATIONS
- Endocarditis
- Heart failure
- Emboli
- Stroke
- Arrhythmias

Aortic insufficiency: Blood flows back into the left ventricle during diastole, causing fluid overload in the ventricle, which dilates and hypertrophies. The excess volume causes fluid overload in the left atrium and, finally, the pulmonary system.

COMPLICATIONS
- Left-sided heart failure
- Pulmonary edema

Aortic stenosis: Over time, left ventricular pressure rises to overcome the resistance of the narrowed valvular opening. The added workload increases the demand for oxygen, and diminished cardiac output causes poor coronary artery perfusion.

COMPLICATIONS
- Ischemia of left ventricle
- Left-sided heart failure
- Arrhythmias
- Endocarditis

Pulmonic stenosis: Obstructed right ventricular outflow causes right ventricular hypertrophy, resulting in right-sided heart failure.

COMPLICATIONS
- Heart failure
- Right ventricular hypertrophy

Signs and Symptoms

The clinical manifestations vary according to valvular defects and the severity of the defect. The patient may be asymptomatic.

Common to All Valvular Disorders

- Dyspnea, weakness, and fatigue

Mitral Stenosis

- Orthopnea
- Palpitations, right-sided heart failure, crackles, and jugular vein distention
- Atrial fibrillation
- Diastolic thrill, loud S_1, and opening snap-diastolic murmur

Mitral Insufficiency

- Palpitations, angina, and tachycardia
- Left-sided heart failure, pulmonary edema, and crackles
- Split S_2; S_3; holosystolic murmur at apex
- Apical thrill

Aortic Insufficiency

- Palpitations, angina, and syncope
- Cough
- Pulmonary congestion and left-sided heart failure
- Quincke's sign
- Pulsus bisferiens and visible apical pulse
- S_3 and blowing diastolic murmur at left sternal border

Aortic Stenosis

- Palpitations, angina, and arrhythmias
- Dyspnea
- Syncope
- Pulmonary congestion and left-sided heart failure
- Diminished carotid pulses and systolic thrill (carotid)
- Decreased cardiac output
- Systolic ejection murmur that radiates to neck and S_4

Pulmonic Stenosis

- Commonly produces no symptoms
- Syncope, chest pain, and right-sided heart failure
- Systolic murmur at left sternal border and S_2 split

Diagnostic Test Results

Diagnostic test results vary with the type of valvular disease that's present. Cardiac catheterization, chest X-ray, echocardiography, and ECG are the standard diagnostic tools used to detect valvular heart disease.

Mitral Stenosis

- Cardiac catheterization reveals diastolic pressure gradient across the valve; elevated left atrial and PAWP with severe pulmonary hypertension; elevated right-sided heart pressure with decreased cardiac output; and abnormal contraction of the left ventricle.
- Chest X-ray shows left atrial and ventricular enlargement, enlarged pulmonary arteries, and mitral valve calcification.
- Echocardiography reveals left atrial and ventricular enlargement, enlarged pulmonary arteries, and mitral valve calcification.
- ECG detects left atrial hypertrophy, atrial fibrillation, right ventricular hypertrophy, and right axis deviation.

Mitral Insufficiency

- Cardiac catheterization reveals mitral regurgitation with increased left ventricular end-diastolic volume and pressure, increased atrial pressure and PAWP, and decreased cardiac output.
- Chest X-ray shows left atrial and ventricular enlargement and pulmonary venous congestion.
- Echocardiography shows abnormal valve leaflet motion and left atrial enlargement.
- ECG may show left atrial and ventricular hypertrophy, sinus tachycardia, and atrial fibrillation.

Aortic Insufficiency

- Cardiac catheterization reveals reduction in arterial diastolic pressure, aortic regurgitation, other valvular abnormalities, and increased left ventricular end-diastolic pressure.
- Chest X-ray shows left ventricular enlargement and pulmonary vein congestion.
- Echocardiography shows left ventricular enlargement, alteration in mitral valve movement, and mitral valve thickening.
- ECG shows sinus tachycardia, left ventricular hypertrophy, and left atrial hypertrophy in severe disease.

Aortic Stenosis

- Cardiac catheterization reveals pressure gradient across valve and increased left ventricular end-diastolic pressures.
- Chest X-ray shows valvular calcification, left ventricular enlargement, and pulmonary vein congestion.
- Echocardiography shows thickened aortic valve and left ventricular wall, possibly coexisting with mitral valve stenosis.
- ECG shows left ventricular hypertrophy.

Pulmonic Stenosis

- Cardiac catheterization reveals increased right ventricular pressure, decreased pulmonary artery pressure, and abnormal valve orifice.
- ECG shows right ventricular hypertrophy, right axis deviation, right atrial hypertrophy, and atrial fibrillation.

Treatment

- Digoxin, anticoagulants, nitroglycerin, beta-adrenergic blockers, diuretics, vasodilators, and angiotensin-converting enzyme inhibitors
- Low-sodium diet
- Oxygen
- Prophylactic antibiotics for invasive procedures, such as dental cleanings, endoscopies, and other procedures where the risk of introducing bacteria into the bloodstream is present. Not indicated for all valvular dysfunctions. See SBE guidelines
- Cardioversion
- Open or closed commissurotomy
- Annuloplasty or valvuloplasty
- Prosthetic valve for mitral or aortic valve disease

VARICOSE VEINS

Varicose veins are dilated, tortuous veins, engorged with blood and resulting from poor venous valve function. They can be primary, originating in the superficial veins, or secondary, occurring in the deep veins.

Causes

Primary Varicose Veins

- Congenital weakness of valves or vein wall
- Prolonged venous stasis or increased intra-abdominal pressure, as in pregnancy, obesity, constipation, or wearing tight clothes
- Standing for an extended period of time
- Family history

Secondary Varicose Veins

- Deep vein thrombosis
- Venous malformation
- Arteriovenous fistulas
- Venous trauma
- Occlusion

Pathophysiology

Veins are thin-walled, distensible vessels with valves that keep blood flowing in one direction. Any condition that weakens, destroys, or distends these valves allows the backflow of blood to the previous valve. If a valve can't hold the pooling blood, it can become incompetent, allowing even more blood to flow backward. The increasing volume of blood in the vein raises pressure and distends the vein. As the veins are stretched, their walls weaken and lose their elasticity, and they become lumpy and tortuous. Rising hydrostatic pressure forces plasma into the surrounding tissues, resulting in edema.

People who stand for prolonged periods may also develop venous pooling because there's no muscular contraction in the legs, forcing blood back up to the heart. If the valves in the veins are too weak to hold the pooling blood, they begin to leak, allowing blood to flow backward.

COMPLICATIONS

- Phlebitis
- Leg ulcers

Signs and Symptoms

- Dilated, tortuous, purplish, ropelike veins, particularly in the calves
- Edema of the calves and ankles
- Leg heaviness that worsens in the evening and in warm weather
- Dull aching in the legs after prolonged standing or walking
- Aching during menses

AGE ALERT

As a person ages, veins dilate and stretch, increasing susceptibility to varicose veins and chronic venous insufficiency. Because the skin becomes friable and can easily break down, ulcers caused by chronic venous insufficiency may take longer to heal.

Diagnostic Test Results

CLINICAL TIP

Manual compression test detects a palpable impulse when the vein is firmly occluded at least 8 inches (20.3 cm) above the point of palpation, indicating incompetent valves in the vein.

Trendelenburg's test (retrograde filling test) detects incompetent valves when the vein is occluded with the patient in the supine position and the leg is elevated 90 degrees. When the person stands (still with the vein occluded), the saphenous veins should fill slowly from below in about 30 seconds.

- Photoplethysmography characterizes venous blood flow by noting changes in the skin's circulation.
- Doppler ultrasonography detects the presence or absence of venous backflow in deep or superficial veins.
- Venous outflow and reflux plethysmography detects deep vein occlusion; this test is invasive and not routinely used.
- Ascending and descending venography demonstrate venous occlusion and patterns of collateral flow.

Treatment

- Treatment of underlying cause (if possible), such as abdominal tumor or obesity
- Antiembolism stockings or elastic bandages
- Regular exercise
- Injection of a sclerosing agent into small- to medium-sized varicosities
- Surgical stripping and ligation of severe varicose veins
- Phlebectomy (removing the varicose vein through small incisions in the skin)

VASCULAR CHANGES IN VARICOSE VEINS