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# 1

## Cardiovascular disorders

### Introduction

The cardiovascular system begins its activity when the fetus is barely a month old and is the last body system to cease activity at the end of life. This system is so vital that its activity defines the presence of life.

### ***Life-giving transport system***

The heart, arteries, veins, and lymphatics form the cardiovascular network that serves as the body's transport system, bringing life-supporting oxygen and nutrients to cells, removing metabolic waste products, and carrying hormones from one part of the body to another. Often called the *circulatory system*, it may be divided into two branches: *pulmonary circulation*, in which blood picks up new oxygen and liberates the waste product carbon dioxide; and *systemic circulation* (including coronary circulation), in which blood carries oxygen and nutrients to all active cells while transporting waste products to the kidneys, liver, and skin for excretion.

Circulation requires normal functioning of the heart, which propels blood through the system by continuous rhythmic contractions. Located behind the sternum, the heart is a muscular organ the size of a man's fist. It has three layers: the *endocardium* —the smooth inner layer; the *myocardium* —the thick, muscular middle layer that contracts in rhythmic beats; and the *epicardium*—the thin, serous membrane, or outer surface of the heart. Covering the entire heart is a saclike membrane called the *pericardium*, which has two layers: a *visceral* layer that's in contact with the heart and a *parietal*, or outer, layer. To prevent irritation when the heart moves against this layer during contraction, fluid lubricates the parietal pericardium.

The heart has four chambers: two thin-walled chambers called *atria* and two thick-walled chambers called *ventricles*. The atria serve as reservoirs during ventricular contraction (systole) and as booster pumps during ventricular relaxation (diastole). The left ventricle propels blood through the systemic circulation. The right ventricle, which forces blood through the pulmonary circulation, is much thinner than the left because it meets only one-sixth the resistance.

#### **ELDER TIP**

*As a person's body ages, the ventricular and aortic walls stiffen, decreasing the heart's pumping action.*

### **Heart valves**

Two kinds of valves work inside the heart: *atrioventricular* and *semilunar*. The atrioventricular valve between the right atrium and ventricle has three leaflets, or cusps, and three papillary muscles; hence, it's called the *tricuspid valve*. The atrioventricular valve between the left atrium and ventricle consists of two cusps shaped like a bishop's miter and two papillary muscles and is called the *mitral valve*. The tricuspid and mitral valves prevent blood backflow from the ventricles to the atria during ventricular contraction. The leaflets of both valves are attached to the ventricles' papillary muscles by thin, fibrous bands called *chordae tendineae*; the leaflets separate and descend funnellike into the

ventricles during diastole and are pushed upward and together during systole to occlude the mitral and tricuspid orifices. The valves' action isn't entirely passive because papillary muscles contract during systole and prevent the leaflets from prolapsing into the atria during ventricular contraction.

The two semilunar valves, which resemble half moons, prevent blood backflow from the aorta and pulmonary arteries into the ventricles when those chambers relax and fill with blood from the atria. They're referred to as the *aortic valve* and *pulmonic valve* for their respective arteries.

#### ELDER TIP

*In elderly people, fibrotic and sclerotic changes thicken heart valves and reduce their flexibility. These changes lead to rigidity and incomplete closure of the valves, which may result in systolic or diastolic murmurs.*

### **The cardiac cycle**

**Diastole** is the phase of ventricular relaxation and filling. As diastole begins, ventricular pressure falls below arterial pressure, and the aortic and pulmonic valves close. As ventricular pressure continues to fall below atrial pressure, the mitral and tricuspid valves open, and blood flows

rapidly into the ventricles. Atrial contraction then increases the volume of ventricular filling by pumping 15% to 25% more blood into the ventricles. When **systole** begins, the ventricular muscle contracts, raising ventricular pressure above atrial pressure and closing the mitral and tricuspid valves. When ventricular pressure finally becomes greater than that in the aorta and pulmonary artery, the aortic and pulmonic valves open, and the ventricles eject blood. Ventricular pressure continues to rise as blood is expelled from the heart. As systole ends, the ventricles relax and stop ejecting blood, and ventricular pressure falls, closing both valves.

$S_1$  (the first heart sound) is heard as the ventricles contract and the atrioventricular valves close.  $S_1$  is loudest at the heart's apex, over the mitral area.  $S_2$  (the second heart sound), which is normally rapid and sharp, occurs when the aortic and pulmonic valves close.  $S_2$  is loudest at the heart's base (second intercostal space on both sides of the sternum).

Normally, with inspiration, a split  $S_2$  will be auscultated. With expiration, the splitting becomes closer or may become single. However, a fixed split  $S_2$  will be heard if the patient has a right bundle-branch block.

Ventricular distention during diastole, which can occur in heart failure, creates low-frequency vibrations that may be heard as a third heart sound ( $S_3$ ), or ventricular gallop. An atrial gallop ( $S_4$ ) may appear at the end of diastole, just before  $S_1$ , if atrial filling is forced into a ventricle that has become less compliant or overdistended or has a decreased ability to contract. A pressure rise and ventricular vibrations cause this sound.

### **Cardiac conduction**

The heart's conduction system is composed of specialized cells capable of generating and conducting rhythmic electrical impulses to stimulate heart contraction. This system includes the sinoatrial (SA) node, the atrioventricular (AV) junction, the bundle of His and its bundle branches, and the ventricular conduction tissue and Purkinje fibers.

Normally, the SA node controls the heart rate and rhythm at 60 to 100 beats/minute. Because the SA node has the lowest resting potential, it's the heart's pacemaker. If it defaults, another part of the system takes over. The AV junction may emerge at 40 to 60 beats/minute; the bundle of His and bundle branches at 30 to 40 beats/minute; and ventricular conduction tissue at 20 to 30 beats/minute.

#### ELDER TIP

*As the myocardium of the aging heart becomes more irritable, extrasystoles may occur along with sinus arrhythmias and sinus bradycardias. In addition, increased fibrous tissue infiltrates the SA nodes and internodal atrial tracts, which may cause atrial fibrillation and flutter.*

## **Cardiac output**

Cardiac output—the amount of blood pumped by the left ventricle into the aorta each minute—is calculated by multiplying the stroke volume (the amount of blood the left ventricle ejects during each contraction) by the heart rate (number of beats/minute). When cellular demands increase, stroke volume or heart rate must increase.

Many factors affect the heart rate, including exercise, pregnancy, and stress. When the sympathetic nervous system releases norepinephrine, the heart rate increases; when the parasympathetic system releases acetylcholine, it slows. As a person ages, the heart rate takes longer to normalize after exercise.

Stroke volume depends on the ventricular blood volume and pressure at the end of diastole (preload), resistance to ejection (afterload), and the myocardium's contractile strength (inotropy). Changes in preload, afterload, or inotropic state can alter the stroke volume.

### **ELDER TIP**

*Exercise cardiac output declines slightly with age. A decrease in maximum heart rate and contractility may cause this change.*

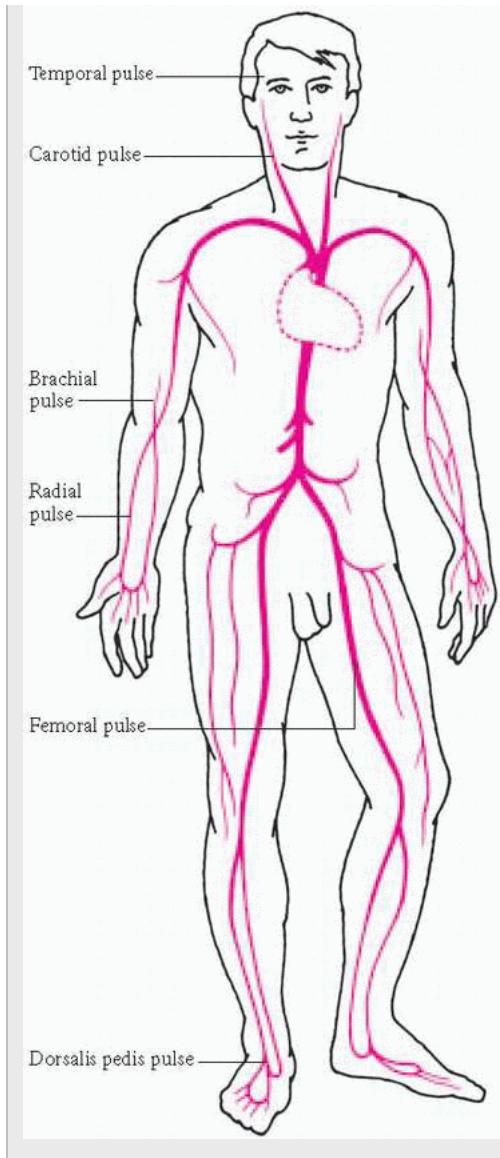
## **Circulation and pulses**

Blood circulates through three types of vessels: *arteries*, *veins*, and *capillaries*. The sturdy, pliable walls of the arteries adjust to the volume of blood leaving the heart. The major artery branching out of the left ventricle is the aorta. Its segments and subbranches ultimately divide into minute, thin-walled (one-cell thick) capillaries. Capillaries pass the blood to the veins, which return it to the heart. In the veins, valves prevent blood backflow.

### **PULSE POINTS**

Peripheral pulse rhythm should correspond exactly to the auscultatory heart rhythm. The pulse's character may offer useful information. For example, *pulsus alternans*, a strong beat followed by a weak one, can mean myocardial weakness. A *waterhammer* (or *Corrigan's*) pulse, a forceful bounding pulse best felt in the carotid arteries or in the forearm, accompanies increased pulse pressure—commonly with capillary pulsations of the fingernails (*Quincke's sign*). This pulse usually indicates patent ductus or aortic insufficiency.

*Pulsus biferiens*, a double peripheral pulse for every apical beat, can signal aortic stenosis, hyperthyroidism, or some other disease. *Pulsus bigeminus* is a coupled rhythm; you feel its beat in pairs. *Pulsus paradoxus* is exaggerated waxing and waning of the arterial pressure ( 15 mm Hg decrease in systolic blood pressure during inspiration).



### ELDER TIP

*Aging contributes to arterial and venous insufficiency as the strength and elasticity of blood vessels decrease.*

Pulses are felt best wherever an artery runs near the skin and over a hard structure. (See *Pulse points*.) Easily found pulses are:

- *radial artery*—anterolateral aspect of the wrist
- *temporal artery*—in front of the ear, above and lateral to the eye
- *common carotid artery*—neck (side)
- *femoral artery*—groin.

The lymphatic system also plays a role in the cardiovascular network. Originating in tissue spaces, the lymphatic system drains fluid and other plasma components that build up in extravascular spaces and

reroutes them back to the circulatory system as lymph, a plasmalike fluid. Lymphatics also extract bacteria and foreign bodies.

## **Cardiovascular assessment**

Physical assessment provides vital information about cardiovascular status.

- Check for underlying cardiovascular disorders, such as central cyanosis (impaired gas exchange), edema (heart failure or valvular disease), and clubbing (congenital cardiovascular disease).
- Palpate the peripheral pulses bilaterally and evaluate their rate, equality, and quality on a scale of 0 (absent) to +4 (bounding). (See *Pulse amplitude scale*.)
- Inspect the carotid arteries for equal appearance. Auscultate for bruits; then palpate the arteries individually, one side at a time, for thrills (fine vibrations due to irregular blood flow).
- Check for pulsations in the jugular veins (more easily seen than felt). Watch for jugular vein distention—a possible sign of right-sided heart failure, valvular stenosis, cardiac tamponade, or pulmonary embolism. Take blood pressure readings in both arms while the patient is lying, sitting, and standing.
- Palpate the precordium for any abnormal pulsations, such as lifts, heaves, or thrills. Use the palms (at the base of the fingertips) or the fingertips. The normal apex will be felt as a light tap and extends over 1" (2.5 cm) or less.
- Systematically auscultate the anterior chest wall for each of the four heart sounds in the aortic area (second intercostal space at the right sternal border), pulmonic area (second intercostal space at the left sternal border), right ventricular area (lower half of the left sternal border), and mitral area (fifth intercostal space at the midclavicular line). However, don't limit your auscultation to these four areas. Valvular sounds may be heard all over the precordium. Therefore, inch your stethoscope in a Z pattern, from the base of the heart across and down and then over to the apex, or start at the apex and work your way up. For low-pitched sounds, use the bell of the stethoscope; for high-pitched sounds, the diaphragm. Carefully inspect each area for pulsations, and palpate for thrills. Check the location of apical pulsation for deviations in normal size (3/8" to 3/4" [1 to 2 cm]) and position (in the mitral area)—possible signs of left ventricular hypertrophy, left-sided valvular disease, or right ventricular disease.
- Listen for the vibrating sound of turbulent blood flow through a stenotic or incompetent valve. Time the murmur to determine where it occurs in the cardiac cycle—between S<sub>1</sub> and S<sub>2</sub> (systolic), between S<sub>2</sub> and the following S<sub>1</sub> (diastolic), or throughout systole (holosystolic). Finally, listen for the scratching or squeaking of a pericardial friction rub.

### **PULSE AMPLITUDE SCALE**

To record your patient's pulse amplitude, use this standard scale:

**0:** Pulse isn't palpable.

**+1:** Pulse is thready, weak, difficult to find, may fade in and out, and disappears easily with pressure.

**+2:** Pulse is constant but not strong; light pressure must be applied or pulse will disappear.

**+3:** Pulse considered normal. Is easily palpable, doesn't disappear with pressure.

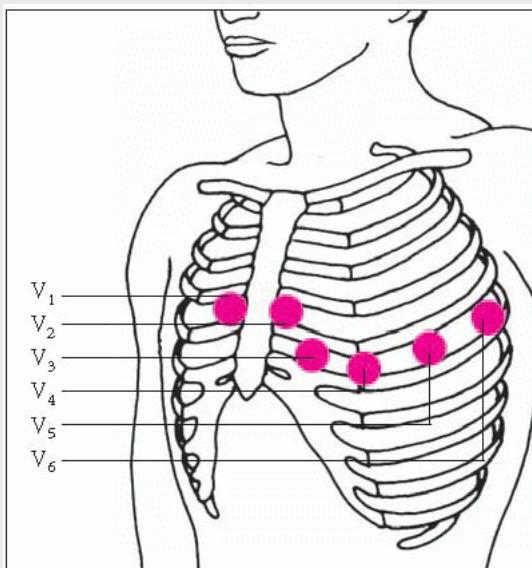
**+4:** Pulse is strong, bounding, and doesn't disappear with pressure.

### **POSITIONING CHEST ELECTRODES**

To record the 12-lead electrocardiogram, place electrodes on the patient's arms and legs (with the ground lead on the patient's right leg). The three standard limb leads (I, II, III) and the three augmented leads (aVR, aVL, aVF) are recorded using these electrodes.

To record the precordial (chest) leads, place the electrodes as follows:

- $V_1$ —fourth intercostal space (ICS), right sternal border
- $V_2$ —fourth ICS, left sternal border
- $V_3$ —midway between  $V_2$  and  $V_4$
- $V_4$ —fifth ICS, left midclavicular line
- $V_5$ —fifth ICS, left anterior axillary line
- $V_6$ —fifth ICS, left midaxillary line.



## Special cardiovascular tests

Electrocardiography (ECG) measures electrical activity by recording currents transmitted by the heart. It can detect ischemia, injury, necrosis, bundle-branch blocks, fascicular blocks, conduction delay, chamber enlargement, and arrhythmias. In Holter monitoring, a tape recording tracks as many as 100,000 cardiac cycles over a 12 or 24-hour period. This test may be used to assess the effectiveness of antiarrhythmic drugs or to evaluate arrhythmia symptoms. A signal-averaged ECG will identify afterpotentials, which are associated with a risk of ventricular arrhythmias. (See *Positioning chest electrodes*.)

Chest X-rays may reveal cardiac enlargement and aortic dilation. They also assess pulmonary circulation. When pulmonary venous and arterial pressures rise, characteristic changes appear, such as dilation of the pulmonary venous shadows. When pulmonary venous pressure exceeds oncotic pressure of the blood, capillary fluid leaks into lung tissues, causing pulmonary edema. This fluid may settle in the alveoli, producing a butterfly pattern, or the lungs may appear cloudy or hazy; in the interlobular septa, sharp linear densities (Kerley's lines) may appear.

Exercise testing using a bicycle ergometer or treadmill determines the heart's response to physical stress. This test measures blood pressure and ECG changes during increasingly rigorous exercises. Myocardial ischemia, abnormal blood pressure response,

or arrhythmias indicate the circulatory system's failure to adapt to exercise.

Cardiac catheterization evaluates chest pain, the need for coronary artery surgery or angioplasty, congenital heart defects, and valvular heart disease and determines the extent of heart failure. Right-sided catheterization involves threading a pulmonary artery thermodilution catheter, which can measure cardiac output, through a vein into the right side of the heart, pulmonary artery, and its branches in the lungs to measure right atrial, right ventricular, pulmonary artery, and pulmonary artery wedge pressures. Left-sided catheterization entails retrograde catheterization of the left ventricle or transseptal catheterization of the left atrium. Ventriculography during left-sided catheterization involves injecting radiopaque dye into the left ventricle to measure ejection fraction and to disclose abnormal heart wall motion or mitral valve incompetence.

In coronary arteriography, radiopaque material injected into coronary arteries allows cineangiographic visualization of coronary arterial narrowing or occlusion.

Digital subtraction angiography evaluates the coronary arteries through the use of X-ray images that are digitally subtracted by computer. Time-based color enhancement shows blood flow in nearby areas.

Echocardiography uses echoes from pulsed high-frequency sound waves (ultrasound) to evaluate cardiac structures. Mmode echocardiography, in which a single, stationary ultrasound beam strikes the heart, produces a vertical view of cardiac structures. Two-dimensional echocardiography (most common), in which an ultrasound beam rapidly sweeps through an arc, produces a cross-sectional or fan-shaped view of cardiac structures. Both M-mode and two-dimensional echocardiography may use contrast agents for enhancement. Doppler echocardiography records blood flow within the cardiovascular system. Color Doppler echocardiography shows the direction of blood flow, which provides information about the degree of valvular insufficiency. Transesophageal echocardiography combines ultrasound with endoscopy to better view the heart's structures. This procedure allows images to be taken from the heart's posterior aspect.

Echocardiography provides information about valve leaflets, size and dimensions of heart chambers, and thickness and motion of the septum and the ventricular walls. It can also reveal intracardiac masses, detect pericardial effusion, diagnose hypertrophic cardiomyopathy, and estimate cardiac output and ejection fraction. This test can also evaluate possible aortic dissection when it involves the ascending aorta.

In multiple-gated acquisition scanning, a radioactive isotope in the intravascular compartment allows measurement of stroke volume, wall motion, and ventricular ejection fraction. Myocardial imaging usually uses the radioactive agent thallium201 or Tc-99m sestamibi (Cardiolite) to detect abnormalities in myocardial perfusion. This agent concentrates in normally perfused areas of the myocardium but not in ischemic areas ("cold spots"), which may be permanent (scar tissue) or temporary (from transient ischemia). These tests can be done as exercise studies or can be combined with drugs, such as adenosine or Persantine, in patients unable to exercise.

Acute infarct imaging documents muscle viability (not perfusion) through the use of technetium-labeled pyrophosphate. Unlike thallium, technetium accumulates only in irreversibly damaged myocardial tissue. Areas of necrosis appear as "hot spots" and can be detected only during an acute myocardial infarction (MI). This test determines the size and location of an infarction but can produce false results.

## Blood tests

Cardiac enzymes (cellular proteins released into blood after cell membrane injury) confirm acute MI or severe cardiac trauma. All cardiac enzymes—creatinine kinase (CK), lactate dehydrogenase, and aspartate aminotransferase, for example—are also found in other cells. Fractionation of enzymes can determine the source of damaged cells. For example, three fractions of CK are isolated, one of which

(an isoenzyme called *CK-MB*) is found only in cardiac cells. *CK-MB* in the blood indicates injury to myocardial cells.

Measurement of a cardiac protein called *troponin* is the most precise way to determine if a patient has experienced an MI. Some 6 hours after an MI, a blood test can detect two forms of troponin: T and I. Troponin T levels peak about 2 days after an MI and return to normal about 16 days later. Troponin I levels reach their peak in less than 1 day after an MI and return to normal in about 7 days.

Peripheral arteriography consists of a fluoroscopic X-ray after arterial injection of a contrast medium. Similarly, phlebography defines the venous system after injection of a contrast medium into a vein. Impedance plethysmography evaluates the venous system to detect pressure changes transmitted to lower leg veins.

Doppler ultrasonography evaluates the peripheral vascular system and assesses peripheral artery disease when combined with sequential systolic blood pressure readings.

Endomyocardial biopsy can detect cardiomyopathy, infiltrative myocardial diseases, and transplant rejection.

Electrophysiologic studies help diagnose conduction system disease and serious arrhythmias. Electronic induction and termination of arrhythmias aid drug selection. Endocardial mapping detects an arrhythmia's focus using a finger electrode. Epicardial mapping uses a computer and a fabric sock with electrodes that's slipped over the heart to detect arrhythmias.

Magnetic resonance imaging can investigate cardiac structure and function. Positron emission tomography and magnetic resonance spectroscopy are used to assess myocardial metabolism.

Electron beam computed tomography, also known as ultrafast computed tomography, is used to detect microcalcifications in the coronary arteries. This test is useful for identifying early coronary artery disease.

## ***Managing cardiovascular disease***

Patients with cardiovascular disease pose a tremendous challenge. Their sheer numbers alone compel a thorough understanding of cardiovascular anatomy, physiology, and pathophysiology. Anticipate a high anxiety level in cardiac patients, and provide support and reassurance, especially during procedures such as cardiac catheterization.

Cardiac rehabilitation programs are widely prescribed and offer education and support along with exercise instruction. Rehabilitation programs begin in health care facilities and continue on an outpatient basis. Helping the patient resume a satisfying lifestyle requires planning and comprehensive teaching. Inform the patient about health care facilities and organizations that offer cardiac rehabilitation programs.

## **CONGENITAL ACYANOTIC DEFECTS**

### ***Ventricular septal defect***

In ventricular septal defect (VSD), the most common congenital heart disorder, an opening in the septum between the ventricles allows blood to shunt between the left and right ventricles. This disease accounts for up to 30% of all congenital heart defects. The prognosis is good for defects that close spontaneously or are correctable surgically but poor for untreated defects, which are sometimes fatal by age 1, usually from secondary complications.

### ***Causes and incidence***

In neonates with VSD, the ventricular septum fails to close completely by the eighth week of gestation, as it would normally. VSD occurs in some neonates with fetal alcohol syndrome, but a

causal relationship hasn't been established. Although most children with congenital heart defects are otherwise normal, in some, VSD coexists with additional birth defects, especially Down syndrome and other autosomal trisomies, renal anomalies, and such cardiac defects as patent ductus arteriosus and coarctation of the aorta. 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 entire septum is absent, creating a single ventricle.

VSD isn't readily apparent at birth, because right and left ventricular pressures are about equal, so blood doesn't shunt through the defect. As the pulmonary vasculature gradually relaxes, 4 to 8 weeks after birth, right ventricular pressure decreases, allowing blood to shunt from the left to the right ventricle.

Less than 1% of neonates are born with VSD. In 80% to 90% of neonates who are born with this disorder, the hole is small and will usually close spontaneously. In the remaining 10% to 20% of neonates, surgery is needed to close the hole.

## ***Complications***

- Right arterial and ventricular hypertrophy
- Heart failure
- Pulmonary hypertension

## ***Signs and symptoms***

Clinical features of VSD vary with the defect's size, the shunting's effect on the pulmonary vasculature, and the infant's age. In a small VSD, shunting is minimal, and pulmonary artery pressure and heart size remain normal. Such defects may eventually close spontaneously without ever causing symptoms.

Initially, large VSD shunts cause left atrial and left ventricular hypertrophy. Later, an uncorrected VSD will cause right ventricular hypertrophy due to increasing pulmonary vascular resistance. Eventually, biventricular heart failure and cyanosis (from reversal of shunt direction) occur. Resulting cardiac hypertrophy may make the anterior chest wall prominent. A large VSD increases the risk of pneumonia.

Infants with large VSDs are thin and small and gain weight slowly. They may develop heart failure with dusky skin; liver, heart, and spleen enlargement because of systemic venous congestion; diaphoresis; feeding difficulties; rapid, grunting respirations; and increased heart rate. They may also develop severe pulmonary hypertension. Fixed pulmonary hypertension may occur much later in life with right-to-left shunt (Eisenmenger's syndrome), causing cyanosis and clubbing of the nail beds.

The typical murmur associated with a VSD is blowing or rumbling and varies in frequency. In the neonate, a moderately loud early systolic murmur may be heard along the lower left sternal border. About the second or third day after birth, the murmur may become louder and longer. In infants, the murmur may be loudest near the heart's base and may suggest pulmonary stenosis. A small VSD may produce a functional murmur or a characteristic loud, harsh systolic murmur. Larger VSDs produce audible murmurs (at least a grade 3 pansystolic), loudest at the fourth intercostal space, usually with a thrill; however, a large VSD with minimal pressure gradient may have no audible murmur. In addition, the pulmonic component of  $S_2$  sounds loud and is widely split. Palpation reveals displacement of the point of maximal impulse to the left. When fixed pulmonary hypertension is present, a diastolic murmur may be audible on auscultation, the systolic murmur becomes quieter, and  $S_2$  is greatly accentuated.

## ***Diagnosis***

Diagnostic findings include:

- Chest X-ray is normal in small defects; in large VSDs, it shows cardiomegaly, left atrial and left ventricular enlargement, and prominent pulmonary vascular markings.
- Electrocardiography (ECG) is normal in children with small VSDs; in large VSDs, it shows left and right ventricular hypertrophy, suggesting pulmonary hypertension.
- Echocardiography may detect a large VSD and its location in the septum, estimate the size of a left-to-right shunt, suggest pulmonary hypertension, and identify associated lesions and complications.

## **Q CONFIRMING DIAGNOSIS**

*Cardiac catheterization determines the VSD's size and exact location, calculates the degree of shunting by comparing the blood oxygen saturation in each ventricle, determines the extent of pulmonary hypertension, and detects associated defects.*

## **Treatment**

In mild cases, no treatment is needed, although the infant should be closely followed to make sure that the hole closes properly as he grows. Large defects usually require early surgical correction before

heart failure and irreversible pulmonary vascular disease develop.

For small defects, surgery consists of simple suture closure. Moderate to large defects require insertion of a patch graft, using cardiopulmonary bypass. In patients with heart failure, digoxin and diuretics may be prescribed to control symptoms. In patients who develop increased pulmonary resistance and irreversible pulmonary vascular changes that produce a reversible right-to-left shunt (Eisenmenger's syndrome), a heart-lung transplant may be required.

If the child has other defects and will benefit from delaying surgery, pulmonary artery banding normalizes pressures and flow distal to the band and prevents pulmonary vascular disease, allowing postponement of surgery. (Pulmonary artery banding is done only when the child has other complications.) A rare complication of VSD repair is complete heart block from interference with the bundle of His during surgery. (Heart block may require temporary or permanent pacemaker implantation.)

Before surgery, treatment consists of:

- digoxin, sodium restriction, and diuretics to prevent heart failure
- careful monitoring by physical examination, X-ray, and ECG to detect increased pulmonary hypertension, which indicates a need for early surgery
- measures to prevent infection (prophylactic antibiotics, for example, to prevent infective endocarditis).

Generally, postoperative treatment includes a brief period of mechanical ventilation. The patient will need analgesics and may also require diuretics to increase urine output, continuous infusions of nitroprusside or adrenergic agents to regulate blood pressure and cardiac output and, in rare cases, a temporary pacemaker.

## **Special considerations**

Although the parents of an infant with VSD often suspect something is wrong with their child before diagnosis, they need psychological support to help them accept the reality of a serious cardiac disorder. Because surgery may take place months after diagnosis, parent teaching is vital to prevent

complications until the child is scheduled for surgery or the defect closes. Thorough explanations of all tests are also essential.

- Instruct parents to watch for signs of heart failure, such as poor feeding, sweating, and heavy breathing.
- If the child is receiving digoxin or other medications, tell the parents how to give it and how to recognize adverse effects. Caution them to keep medications out of the reach of all children.
- Teach parents to recognize and report early signs of infection and to avoid exposing the child to people with obvious infections.
- Encourage parents to let the child engage in normal activities.
- Tell parents to follow-up with their pediatrician. Also tell them that child life therapy may be appropriate if their child displays delayed growth and development or failure to thrive.
- Stress the importance of prophylactic antibiotics before and after surgery.

After surgery to correct VSD:

- Monitor vital signs and intake and output. Maintain the infant's body temperature with an overbed warmer. Give catecholamines, nitroprusside, and diuretics, as ordered; analgesics as needed.
- Monitor central venous pressure, intra-arterial blood pressure, and left atrial or pulmonary artery pressure readings. Assess heart rate and rhythm for signs of conduction block.
- Check oxygenation, particularly in a child who requires mechanical ventilation. Suction to maintain a patent airway and to prevent atelectasis and pneumonia, as needed.
- Monitor pacemaker effectiveness if needed. Watch for signs of failure, such as bradycardia and hypotension.
- Reassure parents and allow them to participate in their child's care.

## ***Atrial septal defect***

In an atrial septal defect (ASD), an opening between the left and right atria allows shunting of blood between the chambers. *Ostium secundum defect* (most common)

occurs in the region of the fossa ovalis and, occasionally, extends inferiorly, close to the vena cava; *sinus venosus defect* occurs in the superior-posterior portion of the atrial septum, sometimes extending into the vena cava, and is almost always associated with abnormal drainage of pulmonary veins into the right atrium; *ostium primum defect* occurs in the inferior portion of the septum primum and is usually associated with atrioventricular valve abnormalities (cleft mitral valve) and conduction defects.

ASD accounts for about 10% of congenital heart defects and appears almost twice as often in females as in males, with a strong familial tendency. Although ASD is usually a benign defect during infancy and childhood, delayed development of symptoms and complications makes it one of the most common congenital heart defects diagnosed in adults. The prognosis is excellent in asymptomatic patients but poor in those with cyanosis caused by large, untreated defects.

## ***Causes and incidence***

The cause of ASD is unknown. In this condition, blood shunts from left to right because left atrial pressure normally is slightly higher than right atrial pressure; this pressure difference forces large amounts of blood through a defect. The left-to-right shunt 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 because of the shunt (rare in children), increased pulmonary vascular

resistance and right ventricular hypertrophy will follow. In some adult patients, irreversible (fixed) pulmonary artery hypertension causes reversal of the shunt direction, which results in unoxygenated blood entering the systemic circulation, causing cyanosis.

ASD is present in 4 of every 100,000 people. Symptoms usually develop before age 30. When no other congenital defect exists, the patient—especially children—may be asymptomatic.

## ***Complications***

- Unoxygenated blood in systemic circulation
- Right and left ventricular hypertrophy
- Atrial arrhythmias
- Heart failure
- Emboli

## ***Signs and symptoms***

ASD commonly goes undetected in preschoolers; such children may complain about feeling tired only after extreme exertion and may have frequent respiratory tract infections but otherwise appear normal and healthy. However, they may show growth retardation if they have large shunts. Children with ASD seldom develop heart failure, pulmonary hypertension, infective endocarditis, or other complications. However, as adults, they usually manifest pronounced symptoms, such as fatigability and dyspnea on exertion, frequently to the point of severe limitation of activity (especially after age 40).

In children, auscultation reveals an early to midsystolic murmur, superficial in quality, heard at the second or third left intercostal space. In patients with large shunts (resulting from increased tricuspid valve flow), a low-pitched diastolic murmur is heard at the lower left sternal border, which becomes more pronounced on inspiration. Although the murmur's intensity is a rough indicator of the size of the left-to-right shunt, its low pitch sometimes makes it difficult to hear and, if the pressure gradient is relatively low, a murmur may not be detectable. Other signs include a fixed, widely split  $S_2$ , caused by delayed closure of the pulmonic valve, and a systolic click or late systolic murmur at the apex, resulting from mitral valve prolapse, which occasionally affects older children with ASD.

In older patients with large, uncorrected defects and fixed pulmonary artery hypertension, auscultation reveals an accentuated  $S_2$ . A pulmonary ejection click and an audible  $S_4$  may also be present. Clubbing and cyanosis become evident; syncope and hemoptysis may occur with severe pulmonary vascular disease.

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## ***Diagnosis***

A history of increasing fatigue and characteristic physical features suggest ASD. The following findings confirm it:

- Chest X-ray shows an enlarged right atrium and right ventricle, a prominent pulmonary artery, and increased pulmonary vascular markings.
- Electrocardiography may be normal but usually shows right axis deviation, prolonged PR interval, varying degrees of right bundle-branch block, right ventricular hypertrophy, atrial fibrillation (particularly in severe cases after age 30) and, in ostium primum defect, left axis deviation.
- Echocardiography measures right ventricular enlargement, may locate the defect, and shows volume overload in the right side of the heart. (Other causes of right ventricular enlargement must be ruled out.)

## CONFIRMING DIAGNOSIS

*Two-dimensional echocardiography with color Doppler flow, contrast echocardiography, or both have supplanted cardiac catheterization as the confirming tests for ASD. Cardiac catheterization is used if inconsistencies exist in the clinical data or if significant pulmonary hypertension is suspected.*

### **Treatment**

Operative repair is advised for all patients with uncomplicated ASD with evidence of significant left-to-right shunting. Ideally, this is performed when the patient is between ages 2 and 4. Operative treatment shouldn't be performed on patients with small defects and trivial left-to-right shunts. Because ASD seldom produces complications in infants and toddlers, surgery can be delayed until they reach preschool or early school age. A large defect may need immediate surgical closure with sutures or a patch graft.

Physicians have developed a new procedure, referred to as catheter closure or transcatheter closure of the atrial septal defect, that uses wires or catheters that can close ASD without surgery. In this procedure, the surgeon makes a tiny incision in the groin to introduce the catheters. Then, he advances the catheters into the heart and places the closure device across the ASD. This procedure may not be applicable to all patients.

### **Special considerations**

- Before cardiac catheterization, explain pretest and posttest procedures to the child and her parents. If possible, use drawings or other visual aids to explain it to the child.
- As needed, teach the patient about prophylactic antibiotics to prevent infective endocarditis. (They may be administered before dental or other invasive procedures.)
- If surgery is scheduled, teach the child and her parents about the intensive care unit and introduce them to the staff. Show parents where they can wait during the operation. Explain postoperative procedures, tubes, dressings, and monitoring equipment.
- After surgery, closely monitor the patient's vital signs, central venous and intra-arterial pressures, and intake and output. Watch for atrial arrhythmias, which may remain uncorrected.

### **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 (the remnant of the ductus arteriosus, a fetal blood vessel) joins the pulmonary artery to the aorta. Coarctation may occur with aortic valve stenosis (usually of a bicuspid aortic valve) and with severe cases of hypoplasia of the aortic arch, patent ductus arteriosus, and ventricular septal defect. Generally, the prognosis for coarctation of the aorta depends on the severity of associated cardiac anomalies; the prognosis for isolated coarctation is good if corrective surgery is performed before this condition induces severe systemic hypertension or degenerative changes in the aorta.

### **Causes and incidence**

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 (arteries that supply the arms, neck, and head) and diminished pressure in the vessels 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. Untreated, this condition may lead to left-sided heart failure and, rarely, to cerebral hemorrhage and aortic rupture. If ventricular septal defect accompanies coarctation, blood shunts left to right, straining the right side of the heart. This leads to pulmonary hypertension and, eventually, right-sided heart hypertrophy and failure.

Coarctation of the aorta occurs in 1 of every 10,000 people and is usually diagnosed in children or adults younger than age 40. It accounts for about 7% of all congenital heart defects in children and is twice as common in males as in females. When it occurs in females, it's commonly associated with Turner's syndrome, a chromosomal disorder that causes ovarian dysgenesis.

## **Complications**

- Infective endocarditis
- Pulmonary hypertension
- Right ventricular hypertrophy
- Right-sided heart failure

## **Signs and symptoms**

Clinical features vary with age. During the first year of life, when aortic coarctation may cause heart failure, the infant displays tachypnea, dyspnea, pulmonary edema, pallor, tachycardia, failure to thrive, cardiomegaly, and hepatomegaly. In most cases, heart sounds are normal unless a coexisting cardiac defect is present. Femoral pulses are absent or diminished.

If coarctation is asymptomatic in infancy, it usually remains so throughout adolescence, as collateral circulation develops to bypass the narrowed segment. During adolescence, this defect may produce dyspnea, claudication, headaches, epistaxis, and hypertension in the upper extremities despite collateral circulation. It commonly causes resting systolic hypertension and wide pulse pressure; high diastolic pressure readings are the same in both the arms and legs. Coarctation may also produce a visible aortic pulsation in the suprasternal notch, a continuous systolic murmur, an accentuated S<sub>2</sub>, and an S<sub>4</sub>.

## **Diagnosis**

### **CONFIRMING DIAGNOSIS**

*The cardinal signs of coarctation of the aorta are resting systolic hypertension, absent or diminished femoral pulses, and wide pulse pressure.*

The following tests support this diagnosis:

- Chest X-ray may demonstrate left ventricular hypertrophy, heart failure, a wide ascending and descending aorta, and notching of the undersurfaces of the ribs, due to extensive collateral circulation. (See *Recognizing coarctation of the aorta*, page 14.)
- Electrocardiography may eventually reveal left ventricular hypertrophy.
- Echocardiography may show increased left ventricular muscle thickness, coexisting aortic valve abnormalities, and the coarctation site.
- Doppler ultrasound and cardiac catheterization evaluate collateral circulation and measure pressure in the right and left ventricles and in the ascending and descending aortas (on both sides of the obstruction).
- Aortography locates the site and extent of coarctation.

## **Treatment**

For an infant with heart failure caused by coarctation of the aorta, treatment consists of medical management with digoxin, diuretics, oxygen, and sedatives. If medical management fails, surgery may be needed.

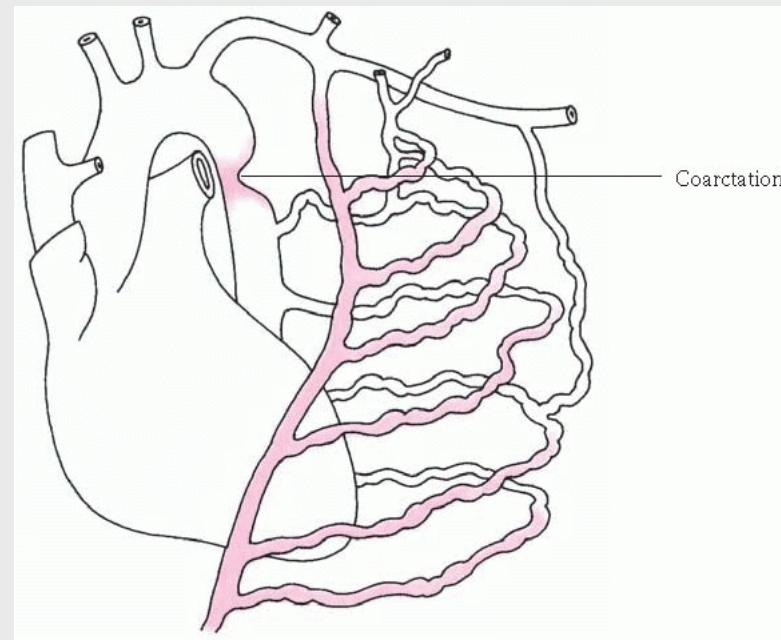
The child's condition usually determines the timing of surgery. Signs of heart failure or hypertension may call for early surgery. If these signs don't appear, surgery usually occurs during the preschool years.

Before the operation, the child may require endocarditis prophylaxis or, if he's older and has previously undetected coarctation, antihypertensive therapy. During surgery, the surgeon uses a flap of the left

subclavian artery to reconstruct an unobstructed aorta.

## **RECOGNIZING COARCTATION OF THE AORTA**

Collateral circulation develops to bypass the occluded aortic lumen, and can be seen on X-ray as notching of the ribs. By adolescence, palpable, visible pulsations may be evident.



Balloon therapy may be indicated for some patients as an alternative to surgical repair. It uses a technique similar to that used to open the coronary arteries, but is performed on the aorta.

## **Special considerations**

- Palpate the pulses in the legs in newborns and at well-baby visits to detect absent or diminished pulses.
- When coarctation in an infant requires rapid digitalization, monitor vital signs closely and watch for digoxin toxicity (poor feeding and vomiting).
- Balance intake and output carefully, especially if the infant is receiving diuretics with fluid restriction.

- Because the infant may not be able to maintain proper body temperature, regulate environmental temperature with an overbed warmer if needed.
- Monitor blood glucose levels to detect possible hypoglycemia, which may occur as glycogen stores become depleted.
- Offer the parents emotional support and an explanation of the disorder. Also explain diagnostic procedures, surgery, and drug therapy. Tell parents what to expect postoperatively.
- For an older child, assess the blood pressure in his extremities regularly, explain any exercise restrictions, stress the need to take medications properly and to watch for adverse effects, and teach him about tests and other procedures.

After corrective surgery:

- Monitor blood pressure closely, using an intra-arterial line. Measure blood pressure in arms and legs. Monitor intake and output.
- If the patient develops hypertension and requires nitroprusside or trimethaphan, administer it, as ordered, by continuous I.V. infusion, using an infusion pump. Watch for severe hypotension and regulate the dosage carefully.
- Provide pain relief and encourage a gradual increase in activity.
- Promote adequate respiratory functioning through turning, coughing, and deep breathing.
- Watch for abdominal pain or rigidity and signs of GI or urinary bleeding.
- If an older child needs to continue antihypertensives after surgery, teach him and his parents about them.
- Stress the importance of continued endocarditis prophylaxis.

## ***Patent ductus arteriosus***

The ductus arteriosus is a fetal blood vessel that connects the pulmonary artery to the descending aorta. In patent ductus arteriosus (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. Initially, PDA may produce no clinical effects, but in time it can precipitate pulmonary vascular disease, causing symptoms to appear by age 40. The prognosis is good if the shunt is small or surgical repair is effective. Otherwise, PDA may advance to intractable heart failure, which may be fatal.

## ***Causes and incidence***

Normally, the ductus closes within days to weeks after birth. Failure to close is most prevalent in premature neonates, probably as a result of abnormalities in oxygenation or the relaxant action of prostaglandin E, which prevents ductal spasm and contracture necessary for closure. PDA commonly accompanies rubella syndrome and may be associated with other congenital defects, such as coarctation of the aorta, ventricular septal defect, and pulmonary and aortic stenoses.

In PDA, relative resistances in pulmonary and systemic vasculature and the size of the ductus determine the amount of left-to-right shunting. The left atrium and left ventricle must accommodate the increased pulmonary venous return, in turn increasing filling pressure and workload on the left side of the heart and possibly causing heart failure. In the final stages of untreated PDA, the left-to-right shunt leads to chronic pulmonary artery hypertension that becomes fixed and unreactive. This causes the shunt to reverse; unoxygenated blood thus enters systemic circulation, causing cyanosis.

PDA is found in 1 of every 2,500 to 5,000 infants and is the most common congenital heart defect found in adults. It affects twice as many females as males.

## **Complications**

- Left-sided heart failure
- Pulmonary artery hypertension
- Respiratory distress (children)

## **Signs and symptoms**

In neonates, especially those who are premature, a large PDA usually produces respiratory distress, with signs of heart failure due to the tremendous volume of blood shunted to the lungs through a patent ductus and the increased workload on the left side of the heart. Other characteristic features may include heightened susceptibility to respiratory tract infections, slow motor development, and failure to thrive. Most children with PDA have no symptoms except cardiac ones. Others may exhibit signs of heart disease, such as physical underdevelopment, fatigability, and frequent respiratory tract infections. Adults with undetected PDA may develop pulmonary vascular disease and, by age 40, may display fatigability and dyspnea on exertion. About 10% of them also develop infective endocarditis.

Auscultation reveals the classic machinery murmur (Gibson murmur): a continuous murmur (during systole and diastole) best heard at the heart's base, at the second left intercostal space under the left clavicle in 85% of children with PDA. This murmur may obscure S<sub>2</sub>. However, with a right-to-left shunt, such a murmur may be absent. Palpation may reveal a thrill at the left sternal border and a prominent left

ventricular impulse. Peripheral arterial pulses are bounding (Corrigan's pulse); pulse pressure is widened because of an elevation in systolic blood pressure and, primarily, a drop in diastolic pressure.

## **Diagnosis**

- Chest X-ray may show increased pulmonary vascular markings, prominent pulmonary arteries, and left ventricle and aorta enlargement.
- Electrocardiography (ECG) may be normal or may indicate left atrial or ventricular hypertrophy and, in pulmonary vascular disease, biventricular hypertrophy.
- Echocardiography detects and helps estimate the size of a PDA. It also reveals an enlarged left atrium and left ventricle or right ventricular hypertrophy from pulmonary vascular disease.

### **CONFIRMING DIAGNOSIS**

*Cardiac catheterization shows pulmonary arterial oxygen content higher than right ventricular content because of the influx of aortic blood. Increased pulmonary artery pressure indicates a large shunt or, if it exceeds systemic arterial pressure, severe pulmonary vascular disease. Catheterization allows calculation of blood volume crossing the ductus and can rule out associated cardiac defects. Dye injection definitively demonstrates PDA.*

## **Treatment**

Asymptomatic infants with PDA require no immediate treatment. Those with heart failure require fluid restriction, diuretics, and cardiac glycosides to minimize or control symptoms. If these measures can't control heart failure, surgery is necessary to ligate the ductus. If symptoms are mild, surgical correction is usually delayed until the infant is between ages 6 months to 3 years, unless problems develop. Before surgery, children with PDA require antibiotics to protect against infective endocarditis.

Other forms of therapy include cardiac catheterization to deposit a plug or coil in the ductus to stop shunting or administration of indomethacin I.V. (a prostaglandin inhibitor that's an alternative to surgery in premature neonates) to induce ductus spasm and closure.

### ***Special considerations***

PDA necessitates careful monitoring, patient and family teaching, and emotional support.

- Watch carefully for signs of PDA in all premature neonates.
- Be alert for respiratory distress symptoms resulting from heart failure, which may develop rapidly in a premature neonate. Frequently assess vital signs, ECG, electrolyte levels, and intake and output. Record response to diuretics and other therapy. Watch for signs of digoxin toxicity (poor feeding and vomiting).
- If the infant receives indomethacin for ductus closure, watch for possible adverse effects, such as diarrhea, jaundice, bleeding, and renal dysfunction.
- Before surgery, carefully explain all treatments and tests to parents. Include the child in your explanations. Arrange for the child and her parents to meet the intensive care unit staff. Tell them about expected I.V. lines, monitoring equipment, and postoperative procedures.
- Immediately after surgery, the child may have a central venous pressure catheter and an arterial line in place. Carefully assess vital signs, intake and output, and arterial and venous pressures. Provide pain relief as needed.
- Before discharge, review instructions to the parents about activity restrictions based on the child's tolerance and energy levels. Advise parents not to become overprotective as their child's tolerance for physical activity increases.
- Stress the need for regular follow-up examinations. Advise parents to inform any practitioner who treats their child about his history of surgery for PDA—even if the child is being treated for an unrelated medical problem.

## **CONGENITAL CYANOTIC DEFECTS**

### ***Tetralogy of Fallot***

Tetralogy of Fallot is a combination of four cardiac defects: ventricular septal defect

(VSD), right ventricular outflow tract obstruction (pulmonary stenosis), right ventricular hypertrophy, and dextroposition of the aorta, with overriding of the VSD. Blood shunts right to left through the VSD, permitting unoxygenated blood to mix with oxygenated blood, resulting in cyanosis. Tetralogy of Fallot sometimes coexists with other congenital heart defects, such as patent ductus arteriosus or atrial septal defect.

### ***Causes and incidence***

The cause of tetralogy of Fallot is unknown, but it results from embryologic hypoplasia of the outflow tract of the right ventricle. Multiple factors, such as Down syndrome, have been associated with its presence. Prenatal risk factors include maternal rubella or other viral illnesses, poor prenatal nutrition, maternal alcoholism, mother older than age 40, and diabetes.

Tetralogy of Fallot occurs in about 5 of every 10,000 infants and accounts for about 10% of all congenital heart diseases. It occurs equally in boys and girls. Before surgical advances made correction possible, about one-third of these children died in infancy.

### ***Complications***

- Cerebral abscess
- Pulmonary thrombosis
- Venous thrombosis
- Cerebral embolism
- Infective endocarditis

## ***Signs and symptoms***

The degree of pulmonary stenosis, interacting with the VSD's size and location, determines the clinical and hemodynamic effects of this complex defect. 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 pulmonary stenosis affects the direction and magnitude of shunt flow across the VSD. Severe obstruction of right ventricular outflow produces a right-to-left shunt, causing decreased systemic arterial oxygen saturation, cyanosis, reduced pulmonary blood flow, and hypoplasia of the entire pulmonary vasculature. Increased right ventricular pressure causes right ventricular hypertrophy. Milder forms of pulmonary stenosis result in a left-to-right shunt or no shunt at all.

Generally, the hallmark of the disorder is cyanosis, which usually becomes evident within several months after birth but may be present at birth if the neonate has severe pulmonary stenosis. Between ages 2 months and 2 years, children with tetralogy of Fallot may experience cyanotic or "blue" spells. Such spells result from increased right-to-left shunting, possibly caused by spasm of the right ventricular outflow tract, increased systemic venous return, or decreased systemic arterial resistance.

Exercise, crying, straining, infection, or fever can precipitate blue spells. Blue spells are characterized by dyspnea; deep, sighing respirations; bradycardia; fainting; seizures; and loss of consciousness. Older children may also develop other signs of poor oxygenation, such as clubbing, diminished exercise tolerance, increasing dyspnea on exertion, growth retardation, and eating difficulties. These children habitually squat when they feel short of breath; this is thought to decrease venous return of unoxygenated blood from the legs and increase systemic arterial resistance.

Children with tetralogy of Fallot also risk developing cerebral abscesses, pulmonary thrombosis, venous thrombosis or cerebral embolism, and infective endocarditis.

In females with tetralogy of Fallot who live to childbearing age, incidence of spontaneous abortion, premature births, and low birth weight rises.

## ***Diagnosis***

In a patient with tetralogy of Fallot, auscultation detects a loud systolic heart murmur (best heard along the left sternal border), which may diminish or obscure the pulmonic component of  $S_2$ . In a patient with a large patent ductus, the continuous murmur of the ductus obscures the systolic murmur. Palpation may reveal a cardiac thrill at the left sternal border and an obvious right ventricular impulse. The inferior sternum appears prominent.

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The results of special tests also support the diagnosis:

- Chest X-ray may demonstrate decreased pulmonary vascular marking, depending on the pulmonary obstruction's severity, and a boot-shaped cardiac silhouette.
- Electrocardiography shows right ventricular hypertrophy, right axis deviation and, possibly, right atrial hypertrophy.

- Echocardiography identifies septal overriding of the aorta, the VSD, and pulmonary stenosis and detects the hypertrophied walls of the right ventricle.
- Laboratory findings reveal diminished arterial oxygen saturation and polycythemia (hematocrit may be more than 60%) if the cyanosis is severe and long-standing, predisposing the patient to thrombosis.

## CONFIRMING DIAGNOSIS

*Cardiac catheterization confirms the diagnosis by visualizing pulmonary stenosis, the VSD, and the overriding aorta and ruling out other cyanotic heart defects. This test also measures the degree of oxygen saturation in aortic blood.*

## Treatment

Effective management of tetralogy of Fallot necessitates prevention and treatment of complications, measures to relieve cyanosis, and palliative or corrective surgery. During cyanotic spells, the knee-chest position and administration of oxygen and morphine improve oxygenation. Propranolol (a beta-adrenergic blocking agent) may prevent blue spells.

Palliative surgery is performed on infants with potentially fatal hypoxic spells. The goal of surgery is to enhance blood flow to the lungs to reduce hypoxia; this is often accomplished by joining the subclavian artery to the pulmonary artery (Blalock-Taussig procedure). Supportive measures include prophylactic antibiotics to prevent infective endocarditis or cerebral abscess administered before, during, and after bowel, bladder, or any other surgery or dental treatments. Management may also include phlebotomy in children with polycythemia.

Complete corrective surgery to relieve pulmonary stenosis and close the VSD, directing left ventricular outflow to the aorta, requires cardiopulmonary bypass with hypothermia to decrease oxygen utilization during surgery, especially in young children. An infant may have this corrective surgery without prior palliative surgery. It's usually done when progressive hypoxia and polycythemia impair the quality of his life, rather than at a specific age. However, most children require surgery before they reach school age.

## Special considerations

- Explain tetralogy of Fallot to the parents. Inform them that their child will set his own exercise limits and will know when to rest. Make sure they understand that their child can engage in physical activity, and advise them not to be overprotective.
- Teach the parents to recognize serious hypoxic spells, which can dramatically increase cyanosis; deep, sighing respirations; and loss of consciousness. Tell them to place their child in the knee-chest position and to report such spells immediately. Emergency treatment may be necessary.
- Instruct the parents on ways to prevent overexerting their child, such as feeding him slowly and providing smaller and more frequent meals. Tell them that remaining calm may decrease his anxiety and that anticipating his needs may minimize crying. Encourage the parents to recruit other family members in the care of the child to help prevent their own exhaustion.
- To prevent infective endocarditis and other infections, warn the parents to keep their child away from people with infections. Urge them to encourage good dental hygiene, and tell them to watch for ear, nose, and throat infections and dental caries, all of which necessitate immediate treatment. When dental care, infections, or surgery requires prophylactic antibiotics, tell the parents to make sure the child completes the prescribed regimen.
- If the child requires medical attention for an unrelated problem, advise the parents to inform the practitioner immediately of the child's history of tetralogy of Fallot because any treatment must take this serious heart defect into consideration.

- During hospitalization, alert the staff to the child's condition. Because of the right-to-left shunt through the VSD, treat I.V. lines like arterial lines. A clot dislodged from a catheter tip in a vein can cross the VSD and cause cerebral embolism. The same thing can happen if air enters the venous lines.

After palliative surgery:

- Monitor oxygenation and arterial blood gas (ABG) values closely in the intensive care unit.
- If the child has undergone the Blalock-Taussig procedure, don't use the arm on the operative side for measuring blood pressure, inserting I.V. lines, or drawing blood samples, because blood perfusion on this side diminishes greatly until collateral circulation develops. Note this on the child's chart and at his bedside.

After corrective surgery:

- Watch for right bundle-branch block or more serious disturbances of atrioventricular conduction and for ventricular ectopic beats.
- Be alert for other postoperative complications, such as bleeding, right-sided heart failure, and respiratory failure. After surgery, transient heart failure is common and may require treatment with digoxin and diuretics.
- Monitor left atrial pressure directly. A pulmonary artery catheter may also be used to check central venous and pulmonary artery pressures.
- Frequently check color and vital signs. Obtain ABG measurements regularly to assess oxygenation. Suction to prevent atelectasis and pneumonia, as needed. Monitor mechanical ventilation.
- Monitor and record intake and output accurately.
- If atrioventricular block develops with a low heart rate, a temporary external pacemaker may be necessary.
- If blood pressure or cardiac output is inadequate, catecholamines may be ordered by continuous I.V. infusion. To decrease left ventricular workload, administer nitroprusside, if ordered, and provide analgesics, as needed.
- Keep the parents informed about their child's progress. After discharge, the child may require digoxin, diuretics, and other drugs. Stress the importance of complying with the prescribed regimen and make sure the parents know how and when to administer these medications. Teach the parents to watch for signs of digoxin toxicity (anorexia, nausea, and vomiting). Prophylactic antibiotics to prevent infective endocarditis will still be required. Advise the parents to avoid becoming overprotective as the child's tolerance for physical activity rises.

## ***Transposition of the great arteries***

In this congenital heart defect, the great arteries are reversed: the aorta arises from the right ventricle and the pulmonary artery from the left ventricle, producing two noncommunicating circulatory systems (pulmonary and systemic). Transposition accounts for about 5% of all congenital heart defects and often coexists with other congenital heart defects, such as ventricular septal defect (VSD), VSD with pulmonary stenosis (PS), atrial septal defect (ASD), and patent ductus arteriosus (PDA). It affects two to three times more males than females.

## ***Causes and incidence***

Transposition of the great arteries results from faulty embryonic development, but the cause of such development is unknown. In transposition, oxygenated blood returning to the left side of the heart is

carried back to the lungs by a transposed pulmonary artery; unoxygenated blood returning to the right side of the heart is carried to the systemic circulation by a transposed aorta.

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.

Transposition of the great arteries occurs in about 40 of every 100,000 infants.

## **Complications**

- Chronic heart failure
- Poor oxygenation
- Arrhythmias
- Right-sided heart failure

## **Signs and symptoms**

Within the first few hours after birth, neonates with transposition of the great arteries and no other heart defects generally show cyanosis and tachypnea, which worsen with crying. After several days or weeks, such neonates usually develop signs of heart failure (gallop rhythm, tachycardia, dyspnea, hepatomegaly, and cardiomegaly). S<sub>2</sub> is louder than normal because the anteriorly transposed aorta is directly behind the sternum; in many cases, however, no murmur can be heard during the first few days of life. Associated defects (ASD, VSD, or PDA) cause their typical murmurs and may minimize cyanosis but may also cause other complications (especially severe heart failure). VSD with PS produces a characteristic murmur and severe cyanosis.

As infants with this defect grow older, cyanosis is their most prominent abnormality. However, they also develop diminished exercise tolerance, fatigability, coughing, clubbing, and more pronounced murmurs if ASD, VSD, PDA, or PS is present.

## **Diagnosis**

- Chest X-rays are normal in the first days of life. Within days to weeks, right atrial and right ventricular enlargement characteristically cause the heart to appear oblong. X-rays also show increased pulmonary vascular markings, except when pulmonary stenosis coexists.
- Electrocardiography typically reveals right axis deviation and right ventricular hypertrophy but may be normal in a neonate.
- Arterial blood gas (ABG) measurements indicate hypoxia and secondary metabolic acidosis.

### **CONFIRMING DIAGNOSIS**

*Echocardiography demonstrates the reversed position of the aorta and pulmonary artery and records echoes from both semilunar valves simultaneously, due to aortic valve displacement. It also detects other cardiac defects. Cardiac catheterization reveals decreased oxygen saturation in left ventricular blood and aortic blood; increased right atrial, right ventricular, and pulmonary artery oxygen saturation; and right ventricular systolic pressure equal to systemic pressure. Dye injection reveals the transposed vessels and the presence of any other cardiac defects.*

## **Treatment**

An infant with transposition may undergo atrial balloon septostomy (Rashkind procedure) during cardiac catheterization. This procedure enlarges the patent foramen ovale, which improves oxygenation by allowing greater mixing of the pulmonary and systemic circulations. Atrial balloon septostomy requires passage of a balloon-tipped catheter through the foramen ovale and subsequent inflation and withdrawal across the atrial septum. This procedure alleviates hypoxia to a certain degree. Afterward, digoxin and diuretics can lessen heart failure until the infant is ready to withstand corrective surgery (usually between birth and age 1).

One of three surgical procedures can correct transposition, depending on the defect's physiology. The Mustard procedure replaces the atrial septum with a Dacron or pericardial partition that allows systemic venous blood to be channeled to the pulmonary artery—which carries the blood to the lungs for oxygenation—and oxygenated blood returning to the heart to be channeled from the pulmonary veins into the aorta. (See *Mustard procedure*.) The Senning procedure accomplishes the same result, using the atrial septum to create partitions to redirect blood flow. In the arterial switch, or Jantene procedure, transposed arteries are surgically anastomosed to the correct ventricle. For this procedure to be successful, the left ventricle must be used to pump at systemic pressure, as it does in neonates or in children with a left ventricular outflow obstruction or a large VSD. Surgery also corrects other heart defects.

## **Special considerations**

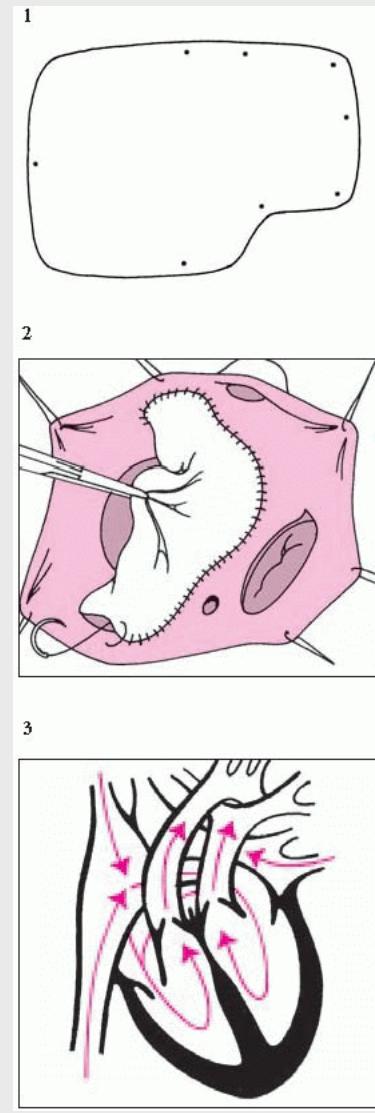
- Explain cardiac catheterization and all necessary procedures to the parents. Offer emotional support.
- Monitor vital signs, ABG values, urine output, and central venous pressure, watching for signs of heart failure. Give digoxin and I.V. fluids, being careful to avoid fluid overload.
- Teach the parents to recognize signs of heart failure and digoxin toxicity (poor feeding and vomiting). Stress the importance of regular checkups to monitor cardiovascular status.
- Teach the parents to protect their infant from infection and to give antibiotics.
- Tell the parents to let their child develop normally. They need not restrict activities; he'll set his own limits.
- If the patient is scheduled for surgery, explain the procedure to the parents and child, if old enough. Teach them about the intensive care unit and introduce them to the staff. Also explain postoperative care.
- Preoperatively, monitor ABG values, acid-base balance, intake and output, and vital signs.

After corrective surgery:

- Monitor cardiac output by checking blood pressure, skin color, heart rate, urine output, central venous and left atrial pressures, and level of consciousness. Report abnormalities or changes.
- Carefully monitor ABG levels and report changes in trends.
- To detect supraventricular conduction blocks and arrhythmias, monitor the patient closely. Watch for signs of atrioventricular blocks, atrial arrhythmias, and faulty sinoatrial function.
- After Mustard or Senning procedures, watch for signs of baffle obstruction such as marked facial edema.
- Encourage parents to help their child assume new activity levels and independence. Teach them about postoperative antibiotic prophylaxis for endocarditis.

## **MUSTARD PROCEDURE**

In the Mustard procedure, a Dacron patch (1) is sutured in the excised atrial septum (2) to divert pulmonary venous return to the tricuspid valve and systemic venous return to the mitral valve (3).



## ACQUIRED INFLAMMATORY HEART DISEASE

### ***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 fails to produce specific cardiovascular symptoms or electrocardiogram (ECG) abnormalities, and recovery is usually spontaneous, without residual defects. Occasionally, myocarditis is complicated by heart failure; in rare cases, it leads to cardiomyopathy.

### ***Causes and incidence***

Myocarditis may result from:

- bacterial infections—diphtheria; tuberculosis; typhoid fever; tetanus; and staphylococcal, pneumococcal, and gonococcal infections
- chemical poisons—such as chronic alcoholism
- helminthic infections—such as trichinosis
- hypersensitive immune reactions—acute rheumatic fever and postcardiotomy syndrome
- parasitic infections—especially South American trypanosomiasis (Chagas' disease) in infants and immunosuppressed adults; also toxoplasmosis
- radiation therapy—large doses of radiation to the chest in treating lung or breast cancer
- viral infections (most common cause in the United States and western Europe)— coxsackievirus A and B strains and, possibly, poliomyelitis, influenza, rubeola, rubella, and adenoviruses and echoviruses.

Myocarditis occurs in 1 to 10 of every 100,000 people in the United States. The median age for this disorder is 42, and incidence is equal between males and females. Children, especially neonates, and persons who are immunocompromised or pregnant (especially pregnant black women) are at higher risk for developing this disorder.

## ***Complications***

- Arrhythmias
- Thromboembolism
- Chronic valvulitis (when disease results from rheumatic fever)
- Recurrence of disease
- Left-sided heart failure (occasional)
- Cardiomyopathy (rare)

## ***Signs and symptoms***

Myocarditis usually causes nonspecific symptoms—such as fatigue, dyspnea, palpitations, and fever—that reflect the accompanying systemic infection. Occasionally, it may produce mild, continuous pressure or soreness in the chest (unlike the recurring, stress-related pain of angina pectoris). Although myocarditis is usually self-limiting, it may induce myofibril degeneration that results in right- and left-sided heart failure, with cardiomegaly, jugular vein distention, dyspnea, persistent fever with resting or exertional tachycardia disproportionate to the degree of fever, and supraventricular and ventricular arrhythmias. Sometimes myocarditis recurs or produces chronic valvulitis (when it results from rheumatic fever), cardiomyopathy, arrhythmias, and thromboembolism.

## ***Diagnosis***

Patient history commonly reveals recent febrile upper respiratory tract infection, viral pharyngitis, or tonsillitis. Physical examination shows supraventricular and ventricular arrhythmias,  $S_3$  and  $S_4$  gallops, a faint  $S_1$ , possibly a murmur of mitral insufficiency (from papillary muscle dysfunction) and, if pericarditis is present, a pericardial friction rub.

Laboratory tests can't unequivocally confirm myocarditis, but the following findings support this diagnosis:

- cardiac enzymes: elevated creatine kinase (CK), CK-MB, aspartate aminotransferase, and lactate dehydrogenase levels
- increased white blood cell count and erythrocyte sedimentation rate
- elevated antibody titers (such as antistreptolysin-O titer in rheumatic fever).

## CONFIRMING DIAGNOSIS

*Endomyocardial biopsy is rarely performed to diagnose myocarditis; the procedure is invasive and costly. A negative*

*biopsy doesn't exclude the diagnosis, and a repeat biopsy may be needed.*

ECG typically shows diffuse ST-segment and T-wave abnormalities as in pericarditis, conduction defects (prolonged PR interval), and other supraventricular arrhythmias. Echocardiography demonstrates some degree of left ventricular dysfunction, and radionuclide scanning may identify inflammatory and necrotic changes characteristic of myocarditis.

Stool and throat cultures may identify bacteria.

## *Treatment*

Treatment includes antibiotics for bacterial infection, modified bed rest to decrease heart workload, and careful management of complications. Inotropic support of cardiac function with amrinone, dopamine, or dobutamine may be needed. Heart failure requires restriction of activity to minimize myocardial oxygen consumption, supplemental oxygen therapy, sodium restriction, diuretics to decrease fluid retention, and cardiac glycosides to increase myocardial contractility. However, cardiac glycosides should be administered cautiously because some patients with myocarditis may show a paradoxical sensitivity to even small doses. Arrhythmias necessitate prompt but cautious administration of antiarrhythmics because these drugs depress myocardial contractility.

Thromboembolism requires anticoagulation therapy. Treatment with corticosteroids or other immunosuppressants may be used to reduce inflammation, but they haven't been shown to change the progression of myocarditis infections. Nonsteroidal anti-inflammatory drugs are contraindicated during the acute phase (first 2 weeks) because they increase myocardial damage.

Surgical treatment may include left ventricular assistive devices and extra corporeal membrane oxygenation for support of cardiogenic shock. Cardiac transplantation has been beneficial for giant cell myocarditis.

## *Special considerations*

- Assess cardiovascular status frequently, watching for signs of heart failure, such as dyspnea, hypotension, and tachycardia. Check for changes in cardiac rhythm or conduction.
- Observe for signs of digoxin toxicity (anorexia, nausea, vomiting, blurred vision, and cardiac arrhythmias) and for complicating factors that may potentiate toxicity, such as electrolyte imbalance or hypoxia.
- Stress the importance of bed rest. Assist with bathing, as necessary; provide a bedside commode because this stresses the heart less than using a bedpan. Reassure the patient that activity limitations are temporary. Offer diversional activities that are physically undemanding.
- During recovery, recommend that the patient resume normal activities slowly and avoid competitive sports.

## PREVENTION

- *Instruct patient to obtain prompt treatment of causative disorders.*
- *Instruct patient to practice good hygiene, including thorough hand-washing.*

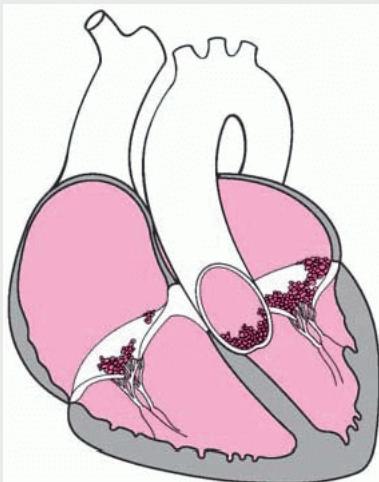
- Tell patient to thoroughly wash and cook food.

## **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. This invasion produces vegetative growths on the heart valves, endocardial lining of a heart chamber, or endothelium of a blood vessel that may embolize to the spleen, kidneys, central nervous system, and lungs. In endocarditis, fibrin and platelets aggregate on the valve tissue and engulf circulating bacteria or fungi that flourish and produce friable verrucous vegetations. (See *Degenerative changes in endocarditis*, page 24.) Such vegetations may cover the valve surfaces, causing ulceration and necrosis; they may also extend to the chordae tendineae, leading to their rupture and subsequent valvular insufficiency. Untreated endocarditis is usually fatal, but with proper treatment, 70% of patients recover. The prognosis is worst when endocarditis causes severe valvular damage, leading to insufficiency and heart failure, or when it involves a prosthetic valve.

### **DEGENERATIVE CHANGES IN ENDOCARDITIS**

This illustration shows typical vegetations on the endocardium produced by fibrin and platelet deposits on infection sites.



### **Causes and incidence**

Most cases of endocarditis occur in I.V. drug abusers, patients with prosthetic heart valves, and those with mitral valve prolapse (especially males with a systolic murmur). These conditions have surpassed rheumatic heart disease as the leading risk factor. Other predisposing conditions include coarctation of the aorta, tetralogy of Fallot, subaortic and valvular aortic stenosis, ventricular septal defects, pulmonary stenosis, Marfan syndrome, degenerative heart disease (especially calcific aortic stenosis) and, rarely, syphilitic aortic valve. However, some patients with endocarditis have no underlying heart disease.

Infecting organisms differ among these groups. In patients with native valve endocarditis who aren't I.V. drug abusers, causative organisms usually include—in order of frequency—streptococci (especially *Streptococcus viridans*), staphylococci, or enterococci. Although many other bacteria occasionally cause the disorder, fungal causes are rare in this group. The mitral valve is involved most commonly, followed by the aortic valve.

In patients who are I.V. drug abusers, *Staphylococcus aureus* is the most common infecting organism. Less commonly, streptococci, enterococci, gram-negative bacilli, or fungi cause the disorder. The tricuspid valve is involved most commonly, followed by the aortic and then the mitral valve.

In patients with prosthetic valve endocarditis, early cases (those that develop within 60 days of valve insertion) are usually due to staphylococcal infection. However, gram-negative aerobic organisms, fungi, streptococci, enterococci, or diphtheroids may also cause the disorder. The course is usually fulminant and is associated with a high mortality. Late cases (occurring after 60 days) present similarly to native valve endocarditis.

In the United States, endocarditis affects 1.4 to 4.2 people out of every 100,000. Males are twice as likely as females to acquire this infection, and the mean age of onset is 50. Mortality is associated with increased age, infection of the aortic valve, heart failure and underlying heart disease, and central nervous system complications; mortality rates vary with the infecting organism.

## ***Complications***

- Left-sided heart failure
- Valvular stenosis or insufficiency
- Myocardial erosion

## ***Signs and symptoms***

Early clinical features of endocarditis are usually nonspecific and include malaise, weakness, fatigue, weight loss, anorexia, arthralgia, night sweats, chills, valvular insufficiency and, in 90% of patients, an intermittent fever that may recur for weeks. A more acute onset is associated with organisms of high pathogenicity such as *S. aureus*. Endocarditis commonly causes a loud, regurgitant murmur typical of the underlying heart lesion. A suddenly changing murmur or the discovery of a new murmur in the presence of fever is a classic physical sign of endocarditis.

In about 30% of patients, embolization from vegetating lesions or diseased valvular tissue may produce typical features of

splenic, renal, cerebral, or pulmonary infarction or of peripheral vascular occlusion:

- splenic infarction—pain in the left upper quadrant, radiating to the left shoulder; and abdominal rigidity
- renal infarction—hematuria, pyuria, flank pain, and decreased urine output
- cerebral infarction—hemiparesis, aphasia, or other neurologic deficits
- pulmonary infarction (most common in right-sided endocarditis, which commonly occurs among I.V. drug abusers and after cardiac surgery)—cough, pleuritic pain, pleural friction rub, dyspnea, and hemoptysis
- peripheral vascular occlusion—numbness and tingling in an arm, leg, finger, or toe, or signs of impending peripheral gangrene.

Other signs may include splenomegaly; petechiae of the skin (especially common on the upper anterior trunk) and the buccal, pharyngeal, or conjunctival mucosa; and splinter hemorrhages under the nails. Rarely, endocarditis produces Osler's nodes (tender, raised, subcutaneous lesions on the fingers or toes), Roth's spots (hemorrhagic areas with white centers on the retina), and Janeway lesions (purplish macules on the palms or soles).

## ***Diagnosis***

### **CONFIRMING DIAGNOSIS**

*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 1 hour between each draw.*

The remaining 10% may have negative blood cultures, possibly suggesting fungal infection or infections that are difficult to diagnose such as *Haemophilus parainfluenzae*.

Other abnormal but nonspecific laboratory test results include:

- normal or elevated white blood cell count
- abnormal histiocytes (macrophages)
- elevated erythrocyte sedimentation rate
- normocytic, normochromic anemia (in 70% to 90% of patients)
- proteinuria and microscopic hematuria (in about 50% of patients)
- positive serum rheumatoid factor (in about 50% of patients after endocarditis is present for 3 to 6 weeks).

Echocardiography (particularly, transesophageal) may identify valvular damage; electrocardiography may show atrial fibrillation and other arrhythmias that accompany valvular disease.

## **Treatment**

The goal of treatment is to eradicate the infecting organism with appropriate antimicrobial therapy, which should start promptly and continue over 4 to 6 weeks. Selection of an antibiotic is based on identification of the infecting organism and on sensitivity studies. While awaiting results, or if blood cultures are negative, empiric antimicrobial therapy is based on the likely infecting organism.

Supportive treatment includes bed rest, aspirin for fever and aches, and sufficient fluid intake. Severe valvular damage, especially aortic or mitral insufficiency, may require corrective surgery if refractory heart failure develops, or in cases requiring that an infected prosthetic valve be replaced.

## **Special considerations**

- Before giving antibiotics, obtain a patient history of allergies. Administer antibiotics on time to maintain consistent antibiotic blood levels.
  - Observe for signs of infiltration or inflammation at the venipuncture site, possible complications of long-term I.V. drug administration. To reduce the risk of these complications, rotate venous access sites.
  - Watch for signs of embolization (hematuria, pleuritic chest pain, left upper quadrant pain, or paresis), a common occurrence during the first 3 months of treatment. Tell the patient to watch for and report these signs, which may indicate impending peripheral vascular occlusion or splenic, renal, cerebral, or pulmonary infarction.
  - Monitor the patient's renal status (blood urea nitrogen levels, creatinine clearance, and urine output) to check for signs of renal emboli or evidence of drug toxicity.
- 
- Observe for signs of heart failure, such as dyspnea, tachypnea, tachycardia, crackles, jugular vein distention, edema, and weight gain.
  - Provide reassurance by teaching the patient and his family about this disease and the need for prolonged treatment. Tell them to watch closely for fever, anorexia, and other signs of relapse about 2 weeks after treatment stops. Suggest quiet diversionary activities to prevent excessive physical exertion.

- Make sure susceptible patients understand the need for prophylactic antibiotics before, during, and after dental work, childbirth, and genitourinary, GI, or gynecologic procedures.
- Teach patients how to recognize symptoms of endocarditis and tell them to notify the practitioner at once if such symptoms occur. (See *Preventing endocarditis*.)

## **PREVENTION ENDOCARDITIS**

Any patient who is at risk for or susceptible to endocarditis, such as those with valvular defects, murmurs, or other predisposing factors, should have prophylactic antibiotics before dental or other invasive procedures.

In addition, the patient should practice good hygiene, including thoroughly washing his hands and washing fruits and vegetables and thoroughly cooking all food to prevent introducing organisms into his system. Maintaining good oral health by daily brushing and flossing and having regular dental checkups can also prevent infection. Be sure to advise the patient to notify his family practitioner as well as his dentist or another specialist that he has a condition that places him at high risk for endocarditis.

## ***Pericarditis***

Pericarditis is an inflammation of the pericardium, the fibroserous sac that envelops, supports, and protects the heart. It occurs in both acute and chronic forms. 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 and incidence***

Common causes of this disease include:

- bacterial, fungal, or viral infection (infectious pericarditis)
- neoplasms (primary or metastatic from lungs, breasts, or other organs)
- high-dose radiation to the chest
- uremia
- hypersensitivity or autoimmune disease, such as acute rheumatic fever (most common cause of pericarditis in children), systemic lupus erythematosus, and rheumatoid arthritis
- postcardiac injury such as myocardial infarction (MI), which later causes an autoimmune reaction (Dressler's syndrome) in the pericardium; trauma; or surgery that leaves the pericardium intact but causes blood to leak into the pericardial cavity
- drugs, such as hydralazine or procainamide
- idiopathic factors (most common in acute pericarditis).

Less common causes include aortic aneurysm with pericardial leakage, and myxedema with cholesterol deposits in the pericardium.

Pericarditis most commonly affects men ages 20 to 50, but it can also occur in children following infection with an adenovirus or coxsackievirus.

## ***Complications***

- Pericardial effusion

- Cardiac tamponade

- Shock

- 
- Cardiovascular collapse

- Death

## **PATTERNS OF CARDIAC PAIN**

Although pain perception is individualistic, specific characteristics are associated with different types of cardiac pain, as shown below.

### **Pericarditis**

#### ***Onset and duration***

- Sudden onset; continuous pain lasting for days; residual soreness

#### ***Location and radiation***

- Substernal pain to left of midline; radiation to back or subclavicular area

#### ***Quality and intensity***

- Mild ache to severe pain, deep or superficial; "stabbing," "knifelike"

#### ***Signs and symptoms***

- Precordial friction rub; increased pain with movement, inspiration, laughing, coughing; decreased pain with sitting or leaning forward (sitting up pulls heart away from diaphragm)

#### ***Precipitating factors***

- Myocardial infarction or upper respiratory tract infection; invasive cardiac trauma

### **Angina**

#### ***Onset and duration***

- Gradual or sudden onset; pain usually lasts less than 15 minutes and not more than 30 minutes (average: 3 minutes)

#### ***Location and radiation***

- Substernal or anterior chest pain, not sharply localized; radiation to back, neck, arms, jaws, even upper abdomen or fingers

#### ***Quality and intensity***

- Mild-to-moderate pressure; deep sensation; varied pattern of attacks; "tightness," "squeezing," "crushing," "pressure"

#### ***Signs and symptoms***

- Dyspnea, diaphoresis, nausea, desire to void, belching, apprehension

#### ***Precipitating factors***

- Exertion, stress, eating, cold or hot and humid weather

### **Myocardial infarction**

#### ***Onset and duration***

- Sudden onset; pain lasts 30 minutes to 2 hours; waxes and wanes; residual soreness 1 to 3 days

### **Location and radiation**

- Substernal, midline, or anterior chest pain; radiation to jaws, neck, back, shoulders, or one or both arms

### **Quality and intensity**

- Persistent, severe pressure; deep sensation; "crushing," "squeezing," "heavy," "oppressive"

### **Signs and symptoms**

- Nausea, vomiting, apprehension, dyspnea, diaphoresis, increased or decreased blood pressure; gallop heart sound, "sensation of impending doom"

### **Precipitating factors**

- Occurrence at rest or during physical exertion or emotional stress

## **Signs and symptoms**

Acute pericarditis typically produces a sharp and often sudden pain that usually starts over the sternum and radiates to the neck, shoulders, back, and arms. However, unlike the pain of MI, pericardial pain is often 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.

Pericardial effusion, the major complication of acute pericarditis, may produce effects of heart failure (such as dyspnea, orthopnea, and tachycardia), ill-defined substernal chest pain, and a feeling of fullness in the chest. (See *Patterns of cardiac pain*.)

#### **ALERT**

*If the fluid accumulates rapidly, cardiac tamponade may occur, resulting in pallor, clammy skin, hypotension, pulsus paradoxus (a decrease in systolic blood pressure of 15 mm Hg or more*

*during slow inspiration), jugular vein distention and, eventually, cardiovascular collapse and death.*

Chronic constrictive pericarditis causes a gradual increase in systemic venous pressure and produces symptoms similar to those of chronic right-sided heart failure (fluid retention, ascites, and hepatomegaly).

## **Diagnosis**

Because pericarditis commonly coexists with other conditions, diagnosis of acute pericarditis depends on typical clinical features and elimination of other possible causes. The pericardial friction rub, a classic symptom, is a grating sound heard as the heart moves. It can usually be auscultated best during forced expiration, while the patient leans forward or is on his hands and knees in bed. It may have up to three components, corresponding to the timing of atrial systole, ventricular systole, and the rapid-filling phase of ventricular diastole. Occasionally, this friction rub is heard only briefly or not at all. Nevertheless, its presence, together with other characteristic features, is diagnostic of acute pericarditis. In addition, if acute pericarditis has caused very large pericardial effusions, physical examination reveals increased cardiac dullness and diminished or absent apical impulse and distant heart sounds.

Chest X-ray, echocardiogram, chest magnetic resonance imaging (MRI), heart MRI, heart computed tomography scan, and radionuclide scanning can detect fluid that has accumulated in the pericardial

sac. They may also show enlargement of the heart and signs of inflammation or scarring, depending on the cause of pericarditis.

In patients with chronic pericarditis, acute inflammation or effusions don't occur—only restricted cardiac filling.

Laboratory results reflect inflammation and may identify its cause:

- normal or elevated white blood cell count, especially in infectious pericarditis
- elevated erythrocyte sedimentation rate
- slightly elevated cardiac enzyme levels with associated myocarditis
- culture of pericardial fluid obtained by open surgical drainage or cardiocentesis (sometimes identifies a causative organism in bacterial or fungal pericarditis)
- electrocardiography showing the following changes in acute pericarditis: elevation of ST segments in the standard limb leads and most precordial leads without significant changes in QRS morphology that occur with MI, atrial ectopic rhythms such as atrial fibrillation and, in pericardial effusion, diminished QRS voltage.

Other pertinent laboratory data include blood urea nitrogen levels to check for uremia, antistreptolysin-O titers to detect rheumatic fever, and a purified protein derivative skin test to check for tuberculosis. In pericardial effusion, echocardiography is diagnostic when it shows an echo-free space between the ventricular wall and the pericardium.

## ***Treatment***

The goal of treatment is to relieve symptoms and manage the underlying systemic disease. In acute idiopathic pericarditis and postthoracotomy pericarditis, treatment consists of bed rest as long as fever and pain persist, and nonsteroidal drugs, such as aspirin and indomethacin, to relieve pain and reduce inflammation. Post-MI patients should avoid nonsteroidal anti-inflammatory drugs and steroids because they may interfere with myocardial scar formation. If these drugs fail to relieve symptoms, corticosteroids may be used. Although corticosteroids produce rapid and effective relief, they must be used cautiously because episodes may recur when therapy is discontinued.

Infectious pericarditis that results from disease of the left pleural space, mediastinal abscesses, or septicemia requires antibiotics (possibly by direct pericardial injection), surgical drainage, or both. Cardiac tamponade may require pericardiocentesis. Signs of tamponade include pulsus paradoxus, jugular vein distention, dyspnea, and shock.

Recurrent pericarditis may necessitate partial pericardectomy, which creates a “window” that allows fluid to drain into the pleural space. In constrictive pericarditis, total pericardectomy to permit adequate filling and contraction of the heart may be necessary. Treatment must also include

management of rheumatic fever, uremia, tuberculosis, and other underlying disorders.

## ***Special considerations***

A patient with pericarditis needs complete bed rest. In addition, health care includes:

- assessing pain in relation to respiration and body position to distinguish pericardial pain from myocardial ischemic pain
- placing the patient in an upright position to relieve dyspnea and chest pain; providing analgesics and oxygen; and reassuring the patient with acute pericarditis that his condition is temporary and treatable
- monitoring for signs of cardiac compression or cardiac tamponade, possible complications of pericardial effusion (Signs include decreased blood pressure, increased central venous pressure,

and pulsus paradoxus. Because cardiac tamponade requires immediate treatment, keep a pericardiocentesis set handy whenever pericardial effusion is suspected.)

- explaining tests and treatments to the patient. (If surgery is necessary, he should learn deep-breathing and coughing exercises beforehand. Postoperative care is similar to that given after cardiothoracic surgery.)

## ***Rheumatic fever and rheumatic heart disease***

Acute rheumatic fever is a systemic inflammatory disease of childhood, in many cases recurrent, that follows a group A betahemolytic streptococcal infection. Rheumatic heart disease refers to the cardiac manifestations of rheumatic fever and includes pancarditis (myocarditis, pericarditis, and endocarditis) during the early acute phase and chronic valvular disease later. Long-term antibiotic therapy can minimize the recurrence of rheumatic fever, reducing the risk of permanent cardiac damage and eventual valvular deformity. However, severe pancarditis occasionally produces fatal heart failure during the acute phase. Of the patients who survive this complication, about 20% die within 10 years.

### ***Causes and incidence***

Rheumatic fever appears to be a hypersensitivity reaction to a group A betahemolytic streptococcal infection, in which antibodies manufactured to combat streptococci react and produce characteristic lesions at specific tissue sites, especially in the heart and joints. Because very few persons (3%) with streptococcal infections ever contract rheumatic fever, altered host resistance must be involved in its development or recurrence. Although rheumatic fever tends to be familial, this may merely reflect contributing environmental factors. For example, in lower socioeconomic groups, incidence is highest in children between ages 5 and 15, probably as a result of malnutrition and crowded living conditions. This disease strikes generally during cool, damp weather in the winter and early spring. In the United States, it's most common in the northern states.

### ***Complications***

- Destruction of mitral and aortic valves
- Severe pancarditis
- Pericardial effusion
- Fatal heart failure

### ***Signs and symptoms***

In 95% of patients, rheumatic fever characteristically follows a streptococcal infection that appeared a few days to 6 weeks earlier. A temperature of at least 100.4° F (38° C) occurs, and most patients complain of migratory joint pain or polyarthritis. Swelling, redness, and signs of effusion usually accompany such pain, which most commonly affects the knees, ankles, elbows, or hips. In 5% of patients (generally those with carditis), rheumatic fever causes skin lesions such as erythema marginatum, a nonpruritic, macular, transient rash that gives rise to red lesions with blanched centers. Rheumatic fever may also produce firm, movable, nontender, subcutaneous nodules about 3 mm to 2 cm in diameter, usually near tendons or bony prominences of joints (especially the elbows, knuckles, wrists, and knees) and less often on the scalp and backs of the hands. These nodules persist for a few days to several weeks and, like erythema marginatum, often accompany carditis.

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Later, rheumatic fever may cause transient chorea, which develops up to 6 months after the original streptococcal infection. Mild chorea may produce hyperirritability, a deterioration in handwriting, or an inability to concentrate. Severe chorea (Sydenham's chorea) causes purposeless, nonrepetitive,

involuntary muscle spasms; poor muscle coordination; and weakness. Chorea always resolves without residual neurologic damage.

The most destructive effect of rheumatic fever is carditis, which develops in up to 50% of patients and may affect the endocardium, myocardium, pericardium, or the heart valves. Pericarditis causes a pericardial friction rub and, occasionally, pain and effusion. Myocarditis produces characteristic lesions called Aschoff bodies (in the acute stages) and cellular swelling and fragmentation of interstitial collagen, leading to formation of a progressively fibrotic nodule and interstitial scars. Endocarditis causes valve leaflet swelling, erosion along the lines of leaflet closure, and blood, platelet, and fibrin deposits, which form beadlike vegetations. Endocarditis affects the mitral valve most often in females; the aortic, most often in males. In both females and males, endocarditis affects the tricuspid valves occasionally and the pulmonic only rarely.

Severe rheumatic carditis may cause heart failure with dyspnea; right upper quadrant pain; tachycardia; tachypnea; a hacking, nonproductive cough; edema; and significant mitral and aortic murmurs. The most common of such murmurs include:

- a systolic murmur of mitral insufficiency (high-pitched, blowing, holosystolic, loudest at apex, possibly radiating to the anterior axillary line)
- a midsystolic murmur due to stiffening and swelling of the mitral leaflet
- occasionally, a diastolic murmur of aortic insufficiency (low-pitched, rumbling, almost inaudible). Valvular disease may eventually result in chronic valvular stenosis and insufficiency, including mitral stenosis and insufficiency, and aortic insufficiency. In children, mitral insufficiency remains the major sequela of rheumatic heart disease.

## ***Diagnosis***

Diagnosis depends on recognition of one or more of the classic symptoms (carditis, rheumatic fever without carditis, polyarthritis, chorea, erythema marginatum, or subcutaneous nodules) and a detailed patient history. Laboratory data support the diagnosis:

- White blood cell count and erythrocyte sedimentation rate may be elevated (during the acute phase); blood studies show slight anemia due to suppressed erythropoiesis during inflammation.
- C-reactive protein is positive (especially during acute phase).
- Cardiac enzyme levels may be increased in severe carditis.
- Antistreptolysin-O titer is elevated in 95% of patients within 2 months of onset.
- Electrocardiogram changes aren't diagnostic; but PR interval is prolonged in 20% of patients.
- Chest X-rays show normal heart size (except with myocarditis, heart failure, or pericardial effusion).
- Echocardiography helps evaluate valvular damage, chamber size, and ventricular function.
- Cardiac catheterization evaluates valvular damage and left ventricular function in severe cardiac dysfunction.

## ***Treatment***

Effective management eradicates the streptococcal infection, relieves symptoms, and prevents recurrence, reducing the chance of permanent cardiac damage. During the acute phase, treatment includes penicillin, sulfadiazine, or erythromycin. Salicylates such as aspirin relieve fever and minimize joint swelling and pain; if carditis is present or salicylates fail to relieve pain and inflammation, corticosteroids may be used. Supportive treatment requires strict bed rest for about 5 weeks during the acute phase with active carditis, followed by a progressive increase in physical activity, depending on clinical and laboratory findings and the response to treatment.

After the acute phase subsides, low-dose antibiotics may be used to prevent recurrence. Such preventive treatment usually continues for 5 years or until age 21 (whichever is longer). Heart failure necessitates

continued bed rest and diuretics. Severe mitral or aortic valve dysfunction that causes persistent heart failure requires corrective valvular surgery, including commissurotomy (separation of the adherent, thickened leaflets of the mitral valve), valvuloplasty (inflation of a balloon within a valve), or valve replacement (with prosthetic valve). Such surgery is seldom necessary before late adolescence.

### ***Special considerations***

Because rheumatic fever and rheumatic heart disease require prolonged treatment, the care plan should include comprehensive patient teaching to promote compliance with the prescribed therapy.

- Before giving penicillin, ask the patient or his parents if he has ever had a hypersensitive reaction to it. If he hasn't, warn that such a reaction is possible. Tell them to stop the drug and call the practitioner immediately if he develops a rash, fever, chills, or other signs of allergy *at any time* during penicillin therapy.
- Instruct the patient and his family to watch for and report early signs of heart failure, such as dyspnea and a hacking, nonproductive cough.
- Stress the need for bed rest during the acute phase and suggest appropriate, physically undemanding diversions. After the acute phase, encourage his family and friends to spend as much time as possible with the patient to minimize boredom. Advise his parents to secure tutorial services to help the child keep up with schoolwork during the long convalescence.
- Help his parents overcome any guilt feelings they may have about their child's illness. Tell them that failure to seek treatment for streptococcal infection is common because this illness often seems no worse than a cold. Encourage the child and his parents to vent their frustrations during the long, tedious recovery. If the child has severe carditis, help them prepare for permanent changes in his lifestyle.
- Teach the patient and his family about this disease and its treatment. Warn parents to watch for and immediately report signs of recurrent streptococcal infection—sudden sore throat, diffuse throat redness and oropharyngeal exudate, swollen and tender cervical lymph glands, pain on swallowing, temperature of 101° to 104° F (38.3° to 40° C), headache, and nausea. Urge them to keep the child away from people with respiratory tract infections.
- Promote good dental hygiene to prevent gingival infection. Make sure the patient and his family understand the need to comply with prolonged antibiotic therapy and follow-up care and the need for additional antibiotics during dental surgery or procedures. Arrange for a home health nurse to oversee home care if necessary.
- Teach the patient to follow current recommendations of the American Heart Association for prevention of bacterial endocarditis. Antibiotic regimens used to prevent recurrence of acute rheumatic fever are inadequate for preventing bacterial endocarditis.

## **VALVE DISORDERS**

### ***Valvular heart disease***

In valvular heart disease, three types of mechanical disruption can occur: stenosis, or narrowing, of the valve opening; incomplete closure of the valve; and prolapse of the valve. A combination of these three in the same valve may also occur. They can result from such disorders as endocarditis (most common), congenital defects, and inflammation, and they can lead to heart failure.

Valvular heart disease occurs in varying forms, described below. Additional information is provided in *Types of valvular heart disease*, pages 32 through 35.

- **Mitral insufficiency:** In this form, blood from the left ventricle flows back into the left atrium during systole, causing the atrium to enlarge to accommodate the backflow. As a result, 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 result in increased pulmonary artery pressure, eventually leading to left- and right-sided heart failure.

## TYPES OF VALVULAR HEART DISEASE

Causes and incidence	Signs and symptoms	Diagnostic measures
<b>Aortic insufficiency</b>		
<ul style="list-style-type: none"> <li>▪ Results from rheumatic fever, syphilis, hypertension, endocarditis, or may be idiopathic</li> <li>▪ Associated with Marfan syndrome</li> <li>▪ Most common in males</li> <li>▪ Associated with ventricular septal defect, even after surgical closure</li> </ul>	<ul style="list-style-type: none"> <li>▪ Dyspnea, cough, fatigue, palpitations, angina, syncope</li> <li>▪ Pulmonary venous congestion, heart failure, pulmonary edema (left-sided heart failure), “pulsating” nail beds</li> <li>▪ Rapidly rising and collapsing pulses (pulsus bifidus), cardiac arrhythmias, wide pulse pressure in severe insufficiency</li> <li>▪ Auscultation: reveals S<sub>3</sub> and diastolic blowing murmur at left sternal border</li> <li>▪ Palpation and visualization of apical impulse in chronic disease</li> </ul>	<ul style="list-style-type: none"> <li>▪ Cardiac catheterization: reduction in arterial diastolic pressures, aortic insufficiency, other valvular abnormalities, and increased left ventricular end-diastolic pressure</li> <li>▪ X-ray: left ventricular enlargement, pulmonary vein congestion</li> <li>▪ Echocardiography: left ventricular enlargement, alterations in mitral valve movement (indirect indication of aortic valve disease), and mitral thickening</li> <li>▪ Electrocardiography (ECG): sinus tachycardia, left ventricular hypertrophy, and left atrial hypertrophy in severe disease</li> </ul>
<b>Aortic stenosis</b>		
<ul style="list-style-type: none"> <li>▪ Results from congenital aortic bicuspid valve (associated with coarctation of the aorta), congenital stenosis of valve cusps, rheumatic fever, or atherosclerosis in elderly persons</li> <li>▪ Most common in males</li> </ul>	<ul style="list-style-type: none"> <li>▪ Dyspnea on exertion, paroxysmal nocturnal dyspnea, fatigue, syncope, angina, palpitations</li> <li>▪ Pulmonary venous congestion, heart failure, pulmonary edema</li> <li>▪ Diminished carotid pulses, decreased cardiac output, cardiac arrhythmias; may have pulsus alternans</li> <li>▪ Auscultation: reveals systolic murmur at base or</li> </ul>	<ul style="list-style-type: none"> <li>▪ Cardiac catheterization: pressure gradient across valve (indicating obstruction), increased left ventricular end-diastolic pressures</li> <li>▪ X-ray: valvular calcification, left ventricular enlargement, and pulmonary venous congestion</li> <li>▪ Echocardiography: thickened aortic valve and left ventricular wall</li> <li>▪ ECG: left ventricular hypertrophy</li> </ul>

in carotids and, possibly, S<sub>4</sub>

#### **Mitral insufficiency**

- Results from rheumatic fever, hypertrophic cardiomyopathy, mitral valve prolapse, myocardial infarction, severe left-sided heart failure, or ruptured chordae tendineae
- Associated with other congenital anomalies such as transposition of the great arteries
- Rare in children without other congenital anomalies
- Orthopnea, dyspnea, fatigue, angina, palpitations
- Peripheral edema, jugular vein distention (JVD), hepatomegaly (right-sided heart failure)
- Tachycardia, crackles, pulmonary edema
- Auscultation: reveals holosystolic murmur at apex, possible split S<sub>2</sub>, and S<sub>3</sub>
- Cardiac catheterization: mitral insufficiency with increased left ventricular end-diastolic volume and pressure, increased atrial pressure and pulmonary artery wedge pressure (PAWP); and decreased cardiac output X-ray: left atrial and ventricular enlargement, pulmonary venous congestion
- Echocardiography: abnormal valve leaflet motion, left atrial enlargement
- ECG: left atrial and ventricular hypertrophy, sinus tachycardia, and atrial fibrillation

#### **Mitral stenosis**

- Results from rheumatic fever (most common cause)
- Most common in females
- May be associated with other congenital anomalies
- Dyspnea on exertion, paroxysmal nocturnal dyspnea, orthopnea, weakness, fatigue, palpitations
- Peripheral edema, JVD, ascites, hepatomegaly (right-sided heart failure in severe pulmonary hypertension)
- Crackles, cardiac arrhythmias (atrial fibrillation), signs of systemic emboli
- Auscultation: reveals loud S<sub>1</sub> or opening snap and diastolic murmur at apex
- Cardiac catheterization: diastolic pressure gradient across valve; elevated left atrial pressure and PAWP (> 15 mm Hg) with severe pulmonary hypertension and pulmonary artery pressures (PAPs); elevated right-sided heart pressure; decreased cardiac output; and abnormal contraction of the left ventricle
- X-ray: left atrial and ventricular enlargement, enlarged pulmonary arteries, and mitral valve calcification
- Echocardiography: thickened mitral valve leaflets, left atrial enlargement
- ECG: left atrial hypertrophy, atrial fibrillation, right ventricular hypertrophy, and right-axis deviation

#### **Mitral valve prolapse syndrome**

- Cause unknown; researchers speculate that metabolic or neuroendocrine factors cause constellation of signs and symptoms
- Most commonly affects young women but may occur in both sexes and in all age-groups
- May produce no signs
- Chest pain, palpitations, headache, fatigue, exercise intolerance, dyspnea, lightheadedness, syncope, mood swings, anxiety, panic attacks
- Auscultation: typically reveals mobile, midsystolic click, with or without mid-to-late systolic murmur
- Two-dimensional echocardiography: prolapse of mitral valve leaflets into left atrium
- Color-flow Doppler studies: mitral insufficiency
- Resting ECG: ST-segment changes, biphasic or inverted T-waves in leads II, III, or AV
- Exercise ECG: evaluates chest pain and arrhythmias

#### **Pulmonic insufficiency**

- May be congenital or may result from pulmonary hypertension
- Dyspnea, weakness, fatigue, chest pain
- Cardiac catheterization: pulmonic insufficiency, increased right ventricular pressure, and associated cardiac defects
- X-ray: right ventricular and pulmonary arterial enlargement

<ul style="list-style-type: none"> <li>May rarely result from prolonged use of pressure monitoring catheter in the pulmonary artery</li> </ul>	<ul style="list-style-type: none"> <li>Peripheral edema, JVD, hepatomegaly (right-sided heart failure)</li> <li>Auscultation: reveals diastolic murmur in pulmonic area</li> </ul>	<ul style="list-style-type: none"> <li>ECG: right ventricular or right atrial enlargement</li> </ul>
<b>Pulmonic stenosis</b>		
<ul style="list-style-type: none"> <li>Results from congenital stenosis of valve cusp or rheumatic heart disease (infrequent)</li> <li>Associated with other congenital heart defects such as tetralogy of Fallot</li> </ul>	<ul style="list-style-type: none"> <li>Asymptomatic or symptomatic with dyspnea on exertion, fatigue, chest pain, syncope</li> <li>May lead to peripheral edema, JVD, hepatomegaly (right-sided heart failure)</li> <li>Auscultation: reveals systolic murmur at left sternal border, split S<sub>2</sub> with delayed or absent pulmonic component</li> </ul>	<ul style="list-style-type: none"> <li>Cardiac catheterization: increased right ventricular pressure, decreased PAP, and abnormal valve orifice</li> <li>ECG: may show right ventricular hypertrophy, right axis deviation, right atrial hypertrophy, and atrial fibrillation</li> </ul>
<b>Tricuspid insufficiency</b>		
<ul style="list-style-type: none"> <li>Results from right-sided heart failure, rheumatic fever and, rarely, trauma and endocarditis</li> <li>Associated with congenital disorders</li> <li>Associated with I.V. drug abuse and infective endocarditis manifesting as tricuspid valve disease</li> </ul>	<ul style="list-style-type: none"> <li>Dyspnea and fatigue</li> <li>May lead to peripheral edema, JVD, hepatomegaly, and ascites (right-sided heart failure)</li> <li>Auscultation: reveals possible S<sub>3</sub> and systolic murmur at lower left sternal border that increases with inspiration</li> </ul>	<ul style="list-style-type: none"> <li>Right-sided heart catheterization: high atrial pressure, tricuspid insufficiency, decreased or normal cardiac output</li> <li>X-ray: right atrial dilation, right ventricular enlargement</li> <li>Echocardiography: shows systolic prolapse of tricuspid valve, right atrial enlargement</li> <li>ECG: right atrial or right ventricular hypertrophy, atrial fibrillation</li> </ul>
<b>Tricuspid stenosis</b>		
<ul style="list-style-type: none"> <li>Results from rheumatic fever</li> <li>May be congenital</li> <li>Associated with mitral or aortic valve disease</li> <li>Most common in women</li> </ul>	<ul style="list-style-type: none"> <li>May be symptomatic with dyspnea, fatigue, syncope</li> <li>Possibly peripheral edema, JVD, hepatomegaly, and ascites (right-sided heart failure)</li> <li>Auscultation: reveals diastolic murmur at lower left sternal border that increases with inspiration</li> </ul>	<ul style="list-style-type: none"> <li>Cardiac catheterization: increased pressure gradient across valve, increased right atrial pressure, decreased cardiac output</li> <li>X-ray: right atrial enlargement</li> <li>Echocardiography: leaflet abnormality, right atrial enlargement</li> <li>ECG: right atrial hypertrophy, right or left ventricular hypertrophy, and atrial fibrillation</li> </ul>

- Mitral stenosis:** Narrowing of the valve by valvular abnormalities, fibrosis, or calcification obstructs blood flow from the left atrium to the left ventricle. Consequently, 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. Also, inadequate filling of the left ventricle produces low cardiac output.
- Mitral valve prolapse (MVP):** One or both valve leaflets protrude into the left atrium. MVP is the term used when the anatomic prolapse is accompanied by signs and symptoms unrelated to the

valvular abnormality.

- 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. Left-sided heart failure and pulmonary edema eventually result.
- Aortic stenosis: Increased left ventricular pressure tries to overcome the resistance of the narrowed valvular opening. The added workload increases the demand for oxygen, whereas diminished cardiac output causes poor coronary artery perfusion, ischemia of the left ventricle, and left-sided heart failure.
- Pulmonic insufficiency: Blood ejected into the pulmonary artery during systole flows back into the right ventricle during diastole, causing fluid overload in the ventricle, ventricular hypertrophy and, finally, right-sided heart failure.
- Pulmonic stenosis: Obstructed right ventricular outflow causes right ventricular hypertrophy, eventually resulting in right-sided heart failure.
- Tricuspid insufficiency: Blood flows back into the right atrium during systole, decreasing blood flow to the lungs and the left side of the heart. Cardiac output also lessens. Fluid overload in the right side of the heart can eventually lead to right-sided heart failure.
- Tricuspid stenosis: Obstructed blood flow from the right atrium to the right ventricle causes the right atrium to dilate and hypertrophy. Eventually, this leads to right-sided heart failure and increases pressure in the vena cava.

## **Treatment**

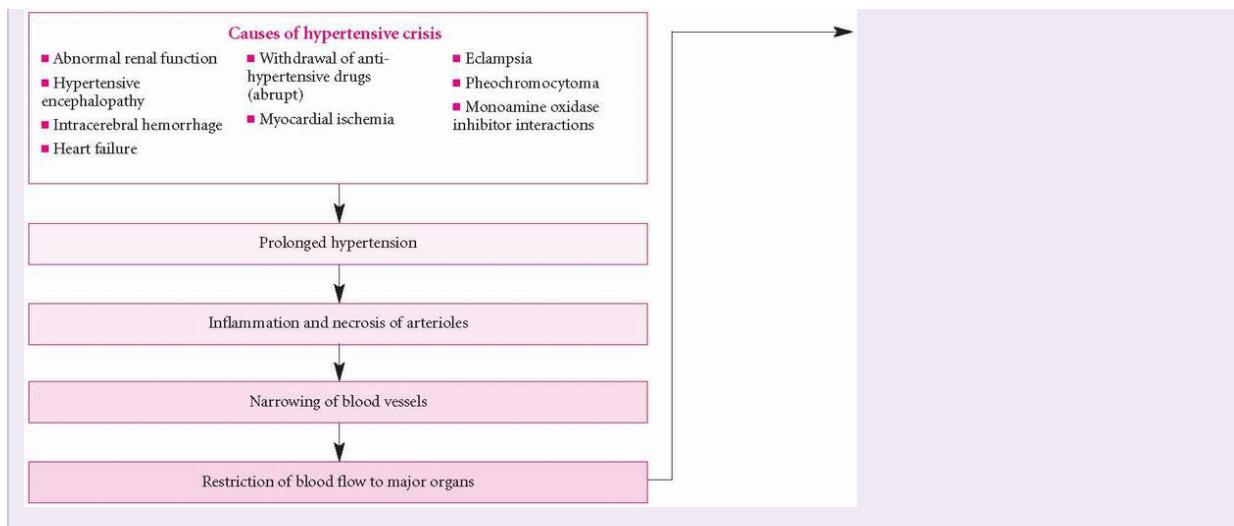
Treatment depends on the nature and severity of associated symptoms. For example, heart failure requires digoxin, diuretics, a sodium-restricted diet and, in acute

cases, oxygen. Other measures may include anticoagulant therapy or antiplatelet medications to prevent thrombus formation around diseased or replaced valves, prophylactic antibiotics before and after surgery or dental care, and valvuloplasty. An intra-aortic balloon pump may be used temporarily to reduce backflow by enhancing forward blood flow into the aorta.

## **PATHOPHYSIOLOGY**

### **WHAT HAPPENS IN HYPERTENSIVE CRISIS**

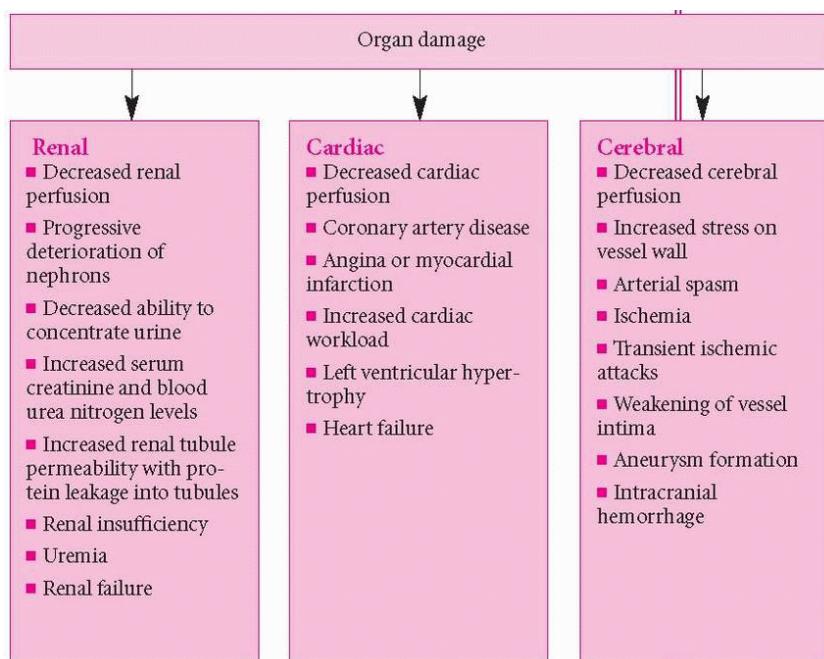
Hypertensive crisis is a severe rise in arterial blood pressure caused by a disturbance in one or more of the regulating mechanisms. If left untreated, hypertensive crisis may result in renal, cardiac, or cerebral complications and, possibly, death.



If the patient has severe signs and symptoms that can't be managed medically, open heart surgery using cardiopulmonary bypass for valve repair or replacement is indicated.

## ***Special considerations***

- Watch closely for signs of heart failure or pulmonary edema and for adverse effects of drug therapy.
- Teach the patient about diet restrictions, medications, and the importance of consistent follow-up care.
- If the patient has surgery, watch for hypotension, arrhythmias, and thrombus formation. Monitor vital signs, arterial blood gas values, intake, output, daily weight, blood chemistries, chest X-rays, and pulmonary artery catheter readings.



## **DEGENERATIVE CARDIOVASCULAR DISORDERS**

## **Hypertension**

Hypertension, an intermittent or sustained elevation in diastolic or systolic blood pressure, occurs as two major types: essential (idiopathic) hypertension, the most common, and secondary hypertension, which results from renal disease or another identifiable cause. Malignant hypertension is a severe, fulminant form of hypertension common to both types. Hypertension is a major cause of stroke, cardiac disease, and renal failure. The prognosis is good if this disorder is detected early and treatment begins before complications develop. Severely elevated blood pressure (hypertensive crisis) may be fatal. (See *What happens in hypertensive crisis.*)

## **Causes and incidence**

Hypertension affects 25% of adults in the United States. If untreated, it carries a high mortality. Risk factors for hypertension include family history, race (most common in blacks), stress, obesity, a diet high in saturated fats or sodium, tobacco use, sedentary lifestyle, and aging.

Secondary hypertension may result from renal vascular disease; pheochromocytoma; primary hyperaldosteronism; Cushing's syndrome; thyroid, pituitary, or parathyroid dysfunction; coarctation of the aorta; pregnancy; neurologic disorders; and use

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of hormonal contraceptives or other drugs, such as cocaine, epoetin alfa (erythropoietin), and cyclosporine.

Cardiac output and peripheral vascular resistance determine blood pressure. Increased blood volume, cardiac rate, and stroke volume as well as arteriolar vasoconstriction can raise blood pressure. The link to sustained hypertension, however, is unclear. Hypertension may also result from failure of intrinsic regulatory mechanisms:

- Renal hypoperfusion causes release of renin, which is converted by angiotensinogen, a liver enzyme, to angiotensin I. Angiotensin I is converted to angiotensin II, a powerful vasoconstrictor. The resulting vasoconstriction increases afterload. Angiotensin II stimulates adrenal secretion of aldosterone, which increases sodium reabsorption. Hypertonic-stimulated release of antidiuretic hormone from the pituitary gland follows, increasing water reabsorption, plasma volume, cardiac output, and blood pressure.
- Autoregulation changes an artery's diameter to maintain perfusion despite fluctuations in systemic blood pressure. The intrinsic mechanisms responsible include stress relaxation (vessels gradually dilate when blood pressure rises to reduce peripheral resistance) and capillary fluid shift (plasma moves between vessels and extravascular spaces to maintain intravascular volume).
- When the blood pressure drops, baroreceptors in the aortic arch and carotid sinuses decrease their inhibition of the medulla's vasomotor center, which increases sympathetic stimulation of the heart by norepinephrine. This, in turn, increases cardiac output by strengthening the contractile force, increasing the heart rate, and augmenting peripheral resistance by vasoconstriction. Stress can also stimulate the sympathetic nervous system to increase cardiac output and peripheral vascular resistance.

## **Complications**

- Stroke
- Coronary artery disease
- Angina
- Myocardial infarction
- Heart failure

- Arrhythmias
- Sudden death
- Cerebral infarction
- Hypertensive encephalopathy
- Hypertensive retinopathy
- Renal failure

## ***Signs and symptoms***

Hypertension usually doesn't produce clinical effects until vascular changes in the heart, brain, or kidneys occur. Severely elevated blood pressure damages the intima of small vessels, resulting in fibrin accumulation in the vessels, development of local edema and, possibly, intravascular clotting. Symptoms produced by this process depend on the location of the damaged vessels:

- brain – stroke
- retina – blindness
- heart – myocardial infarction
- kidneys – proteinuria, edema and, eventually, renal failure.

Hypertension increases the heart's workload, causing left ventricular hypertrophy and, later, left- and right-sided heart failure and pulmonary edema.

## ***Diagnosis***

Serial blood pressure measurements are obtained and compared to previous readings and trends to reveal an increase in diastolic and systolic pressures. (See *Classifying blood pressure readings*.)

Auscultation may reveal bruits over the abdominal aorta and the carotid, renal, and femoral arteries; ophthalmoscopy reveals arteriovenous nicking and, in hypertensive encephalopathy, papilledema. Patient history and the following additional tests may show predisposing factors and help identify an underlying cause such as renal disease:

- Urinalysis: Protein levels and red and white blood cell counts may indicate glomerulonephritis.
  - Excretory urography: Renal atrophy indicates chronic renal disease; one kidney more than 5/8" (1.5 cm) shorter than the other suggests unilateral renal disease.
  - Serum potassium: Levels less than 3.5 mEq/L may indicate adrenal dysfunction (primary hyperaldosteronism).
- 
- Blood urea nitrogen (BUN) and serum creatinine: BUN level that's normal or elevated to more than 20 mg/dl and serum creatinine level that's normal or elevated to more than 1.5 mg/dl suggest renal disease.

### **CLASSIFYING BLOOD PRESSURE READINGS**

The National Institutes of Health, which used to classify blood pressure according to severity categories — mild, moderate, severe, and very severe — has replaced this classification system with a system based on stages.

The following revised categories are based on the average of two or more readings taken on separate visits after an initial screening. They apply to adults age 18 and older who aren't taking antihypertensives, aren't acutely ill, and don't have other health conditions, such as diabetes and kidney disease. (If the

systolic and diastolic pressures fall into different categories, use the higher of the two pressures to classify the reading. For example, a reading of 160/92 mm Hg should be classified as stage 2.)

Normal blood pressure with respect to cardiovascular risk is a systolic reading below 120 mm Hg and a diastolic reading below 80 mm Hg. In general, hypertension is defined as a systolic blood pressure of 140 mm Hg or higher or a diastolic pressure above 90 mm Hg. (For patients with diabetes or chronic kidney disease, hypertension is defined as a reading of 130/80 mm Hg or higher.)

Category	Systolic	Diastolic
<i>Normal</i>	< 120 mm Hg	and < 80 mm Hg
<i>Pre-hypertension</i>	120 to 139 mm Hg	or 80 to 89 mm Hg
<i>Hypertension</i>		
Stage 1	140 to 159 mm Hg	or 90 to 99 mm Hg
Stage 2	160 mm Hg	or 100 mm Hg

In addition to classifying stages of hypertension based on average blood pressure readings, clinicians should also take note of target organ disease and any additional risk factors. For example, a patient with diabetes, left ventricular hypertrophy, and a blood pressure reading of 144/98 mm Hg would be classified as "stage I hypertension with target-organ disease (left ventricular hypertrophy) and another major risk factor (diabetes)." This additional information is important to obtain a true picture of the patient's cardiovascular health.

Other tests help detect cardiovascular damage and other complications:

- Electrocardiography may show left ventricular hypertrophy or ischemia.
- Chest X-ray may show cardiomegaly.
- Echocardiography may show left ventricular hypertrophy.

## **Treatment**

The National Institutes of Health recommends the following approach for treating primary hypertension:

- First, help the patient start needed lifestyle modifications, including weight reduction, moderation of alcohol intake, regular physical exercise, reduction of sodium intake, and smoking cessation.
- If the patient fails to achieve the desired blood pressure or make significant progress, continue lifestyle modifications and begin drug therapy.
- For stage 1 hypertension (systolic [SBP] blood pressure 140 to 159 mm Hg, or diastolic blood pressure [DBP] 90 to 99 mm Hg) in the absence of compelling indications (heart failure, postmyocardial infarction, high coronary disease risk, diabetes, chronic kidney disease, or recurrent stroke prevention), give most patients

thiazide-type diuretics. Consider using an angiotensin-converting enzyme (ACE) inhibitor, beta-

adrenergic blocker, calcium channel blocker (CCB), angiotensinreceptor blocker (ARB), or a combination.

- For stage 2 hypertension (SBP 160 mm Hg, or DBP 100 mm Hg) in the absence of compelling indications, give most patients a two-drug combination (usually a thiazide-type diuretic and an ACE inhibitor, ARB, CCB, or beta-adrenergic blocker).
- If the patient has one or more compelling indications, base drug treatment on benefits from outcome studies or existing clinical guidelines. Treatment may include the following, depending on indication:
  - Heart failure – diuretic, beta-adrenergic blocker, ACE inhibitor, ARB, or aldosterone antagonist
  - High coronary disease risk – diuretic, beta-adrenergic blocker, ACE inhibitor, or CCB
  - Diabetes – diuretic, beta-adrenergic blocker, ACE inhibitor, or CCB
  - Chronic kidney disease – ACE inhibitor or ARB
  - Postmyocardial failure – ACE inhibitor, beta-adrenergic blocker, or aldosterone antagonist
  - Recurrent stroke prevention – diuretic or ACE inhibitor.

Give other antihypertensive drugs as needed.

- If the patient fails to achieve the desired blood pressure, continue lifestyle modifications and optimize drug dosages or add drugs until the goal blood pressure is achieved. Also, consider consultation with a hypertension specialist.

Treatment of secondary hypertension focuses on correcting the underlying cause and controlling hypertensive effects.

Typically, hypertensive emergencies require parenteral administration of a vasodilator or an adrenergic inhibitor or oral administration of a selected drug, such as nifedipine, captopril, clonidine, or labetalol, to rapidly reduce blood pressure. The initial goal is to reduce mean arterial blood pressure by no more than 25% (within minutes to hours) then to 160/110 within 2 hours while avoiding excessive falls in blood pressure that can precipitate renal, cerebral, or myocardial ischemia.

Examples of hypertensive emergencies include hypertensive encephalopathy, intracranial hemorrhage, acute left-sided heart failure with pulmonary edema, and dissecting aortic aneurysm. Hypertensive emergencies are also associated with eclampsia or severe gestational hypertension, unstable angina, and acute myocardial infarction.

Hypertension without accompanying symptoms or target-organ disease seldom requires emergency drug therapy.

## ***Special considerations***

- To encourage adherence to antihypertensive therapy, suggest that the patient establish a daily routine for taking his medication. Warn that uncontrolled hypertension may cause stroke and heart attack. Tell him to report adverse drug effects. Also, advise him to avoid high-sodium antacids and over-the-counter cold and sinus medications, which contain harmful vasoconstrictors.
- Encourage a change in dietary habits. Help the obese patient plan a weight reduction diet; tell him to avoid high-sodium foods (pickles, potato chips, canned soups, and cold cuts) and table salt.
- Help the patient examine and modify his lifestyle (for example, by reducing stress and exercising regularly).
- If a patient is hospitalized with hypertension, find out if he was taking his prescribed medication. If he wasn't, ask why. If he can't afford the medication, refer him to appropriate social service agencies. Tell the patient and his family to keep a record of drugs used in the past, noting

especially which ones were or weren't effective. Suggest recording this information on a card so that the patient can show it to his practitioner.

- When routine blood pressure screening reveals elevated pressure, first make sure the cuff size is appropriate for the patient's upper arm circumference. Take the pressure in both arms in lying, sitting, and standing positions. Ask the patient if he smoked, drank a beverage containing caffeine, or was emotionally upset before the test. Advise him to return for blood pressure testing at frequent and regular intervals.

- To help identify hypertension and prevent untreated hypertension, participate in public education programs dealing with hypertension and ways to reduce risk factors. Encourage public participation in blood pressure screening programs. Routinely screen all patients, especially those at risk (blacks and people with family histories of hypertension, stroke, or heart attack). (See *Preventing hypertension*.)

## PREVENTING HYPERTENSION

Certain risk factors for hypertension can't be changed, such as family history, race, and aging; but lifestyle modifications can help prevent hypertension. Based on American Heart Association recommendations, advise your patient to do the following:

### **Maintain a healthy weight**

Maintain a normal weight or lose weight if overweight. Weight loss lowers blood pressure.

### **Reduce salt**

Salt intake should be reduced to about 1.5 g per day. Reducing salt intake can lower blood pressure in individuals with and without hypertension.

### **Increase potassium**

Patients should eat 8 to 10 servings of fruits and vegetables per day to increase potassium intake. Potassium reduces blood pressure in individuals with and without hypertension. Those with kidney disease or heart failure should contact their practitioner before increasing their potassium intake.

### **Limit alcohol intake**

Studies have shown a correlation with alcohol intake and increased blood pressure, especially in individuals who drink more than 2 drinks per day.

### **Include exercise**

Regular physical activity is defined by the American Heart Association as moderate to vigorous exercise for 30 to 60 minutes a day on most or all days of the week. A lack of physical activity can lead to obesity and increase the risk of hypertension, heart attack, and stroke.

### **Manage stress**

Stress can lead to increased alcohol consumption, smoking, overeating, and other activities that increase the risk of heart attack or stroke. Daily relaxation for short periods during the workday and on weekends can also lower blood pressure.

### **Stop smoking**

Smoking even filtered and light or ultra cigarettes can lead to atherosclerosis. Quitting or not starting is the only way to prevent this major risk factor for heart

attack and stroke.

### Follow the DASH diet

The dietary approaches to stopping hypertension (DASH) diet encourages vegetables, fruits, and low-fat dairy as well as whole grains, fish, poultry, and nuts. Discourage the eating of fats, red meat, sweets, and sugar-containing beverages. However, individuals with reduced kidney function should always consult their practitioners before starting this diet; it's rich in potassium which isn't recommended for individuals with these disorders.

## Coronary artery disease

Coronary artery disease (CAD) occurs when the arteries that supply blood to the heart muscle harden and narrow. The result is the loss of oxygen and nutrients to myocardial tissue because of diminished coronary blood flow. This reduction in blood flow can also lead to coronary syndrome (angina or myocardial infarction).

### Causes and incidence

Atherosclerosis is the usual cause of CAD. In this form of arteriosclerosis, fatty, fibrous plaques, possibly including calcium deposits, narrow the lumen of the coronary arteries, reduce the volume of blood that can flow through them, and lead to myocardial ischemia. Plaque formation also predisposes to thrombosis, which can provoke myocardial infarction (MI).

Atherosclerosis usually develops in highflow, high-pressure arteries, such as those in the heart, brain, kidneys, and in the aorta, especially at bifurcation points. It has been linked to many risk factors: family history, male gender, age (risk increased in those aged 65 or older), hypertension, obesity, smoking, diabetes mellitus, stress, sedentary lifestyle, high serum cholesterol (particularly high low-density lipoprotein cholesterol) or triglyceride levels, low highdensity lipoprotein cholesterol levels, high blood homocysteine levels, menopause and, possibly, infections producing inflammatory responses in the artery walls.

Uncommon causes of reduced coronary artery blood flow include dissecting aneurysms, infectious vasculitis, syphilis, and congenital defects in the coronary vascular system. Coronary artery spasms may also impede blood flow. (See *Coronary artery spasm*.)

Coronary artery disease is the leading cause of death in the United States. According to the American Heart Association, someone in the United States suffers a coronary heart event approximately every 29 seconds, and someone dies from such an event about every 60 seconds.

### Signs and symptoms

The classic symptom of CAD is angina, the direct result of inadequate oxygen flow to the myocardium. Anginal pain is usually described as a burning, squeezing, or tight feeling in the substernal or precordial chest that may radiate to the left arm, neck, jaw, or shoulder blade. Typically, the patient clenches his fist over his chest or rubs his left arm when describing the pain, which may be accompanied by nausea, vomiting, fainting, sweating, and cool extremities. Anginal episodes most often follow physical exertion but may also follow emotional excitement, exposure to cold, or a large meal. Some patients, particularly those with diabetes, may not experience typical anginal pain but may have dyspnea, fatigue, diaphoreses, or more vague symptoms.

Angina has four major forms: *stable* (pain is predictable in frequency and duration and can be relieved with nitrates and rest), *unstable* (pain increases in frequency and duration and is more easily induced), *Prinzmetal's* or *variant* (from unpredictable coronary artery spasm), and *microvascular* (in which impairment of vasodilator reserve causes angina-like chest pain in a patient with normal

coronary arteries). Severe and prolonged anginal pain generally suggests MI, with potentially fatal arrhythmias and mechanical failure.

## **Diagnosis**

The patient history – including the frequency and duration of angina and the presence of associated risk factors – is crucial in evaluating CAD. Additional diagnostic measures include the following:

- Electrocardiogram (ECG) during angina may show ischemia and, possibly, arrhythmias such as premature ventricular contractions. ECG is apt to be normal when the patient is pain-free. Arrhythmias may occur without infarction, secondary to ischemia.
- Treadmill or exercise stress test may provoke chest pain and ECG signs of myocardial ischemia.
- Coronary angiography reveals coronary artery stenosis or obstruction, possible collateral circulation, and the arteries' condition beyond the narrowing.
- Myocardial perfusion imaging with thallium-201, Cardiolite, or Myoview during treadmill exercise detects ischemic areas

of the myocardium, visualized as “cold spots.”

- Stress echocardiography may show wall motion abnormalities.
- Electron-beam computed tomography identifies calcium within arterial plaque; the more calcium seen, the higher the likelihood of CAD.

## **CORONARY ARTERY SPASM**

In coronary artery spasm, a spontaneous, sustained contraction of one or more coronary arteries causes ischemia and dysfunction of the heart muscle. This disorder also causes Prinzmetal's angina and even myocardial infarction in patients with unoccluded coronary arteries. Its cause is unknown but possible contributing factors include:

- altered flow of calcium into the cell
- intimal hemorrhage into the medial layer of the blood vessel
- hyperventilation
- elevated catecholamine levels
- fatty buildup in lumen

## **Signs and symptoms**

The major symptom of coronary artery spasm is angina. But unlike classic angina, this pain often occurs spontaneously and may not be related to physical exertion or emotional stress; it's also more severe, usually lasts longer, and may be cyclic, frequently recurring every day at the same time. Such ischemic episodes may cause arrhythmias, altered heart rate, lower blood pressure and, occasionally, fainting due to diminished cardiac output. Spasm in the left coronary artery may result in mitral insufficiency, producing a loud systolic murmur and, possibly, pulmonary edema, with dyspnea, crackles, hemoptysis, or sudden death.

## **Treatment**

After diagnosis by coronary angiography and electrocardiography (ECG), the patient may receive calcium channel blockers (verapamil, nifedipine, or

diltiazem) to reduce coronary artery spasm and vascular resistance; and nitrates (nitroglycerin or isosorbide dinitrate) to relieve chest pain.

When caring for a patient with coronary artery spasm, explain all necessary procedures and teach him how to take his medications safely. For calcium antagonist therapy, monitor blood pressure, pulse rate, and ECG patterns to detect arrhythmias. For nifedipine and verapamil therapy, monitor digoxin levels and check for signs of digoxin toxicity. Because nifedipine may cause peripheral and periorbital edema, watch for fluid retention.

Because coronary artery spasm is commonly associated with atherosclerotic disease, advise the patient to stop smoking, avoid overeating, maintain a low-fat diet, use alcohol sparingly, and maintain a balance between exercise and rest.

## **Treatment**

The goal of treatment in patients with angina is to either reduce myocardial oxygen demand or increase oxygen supply. Therapy consists primarily of nitrates such as nitroglycerin (given sublingually, orally, transdermally, or topically in ointment form) to dilate coronary arteries and improve blood supply to the heart. Glycoprotein IIb-IIIa inhibitors and antithrombin drugs may be used to reduce the risk of blood clots. Beta-adrenergic blockers may be used to decrease heart rate and lower the heart's oxygen use. Calcium channel blockers may be used to relax the coronary arteries and all systemic arteries, reducing the heart's workload. Angiotensin-converting enzyme inhibitors, diuretics, or other medications may be used to lower blood pressure.

Percutaneous transluminal coronary angioplasty (PTCA) may be performed during cardiac catheterization to compress fatty deposits and relieve occlusion in patients with no calcification and partial occlusion.

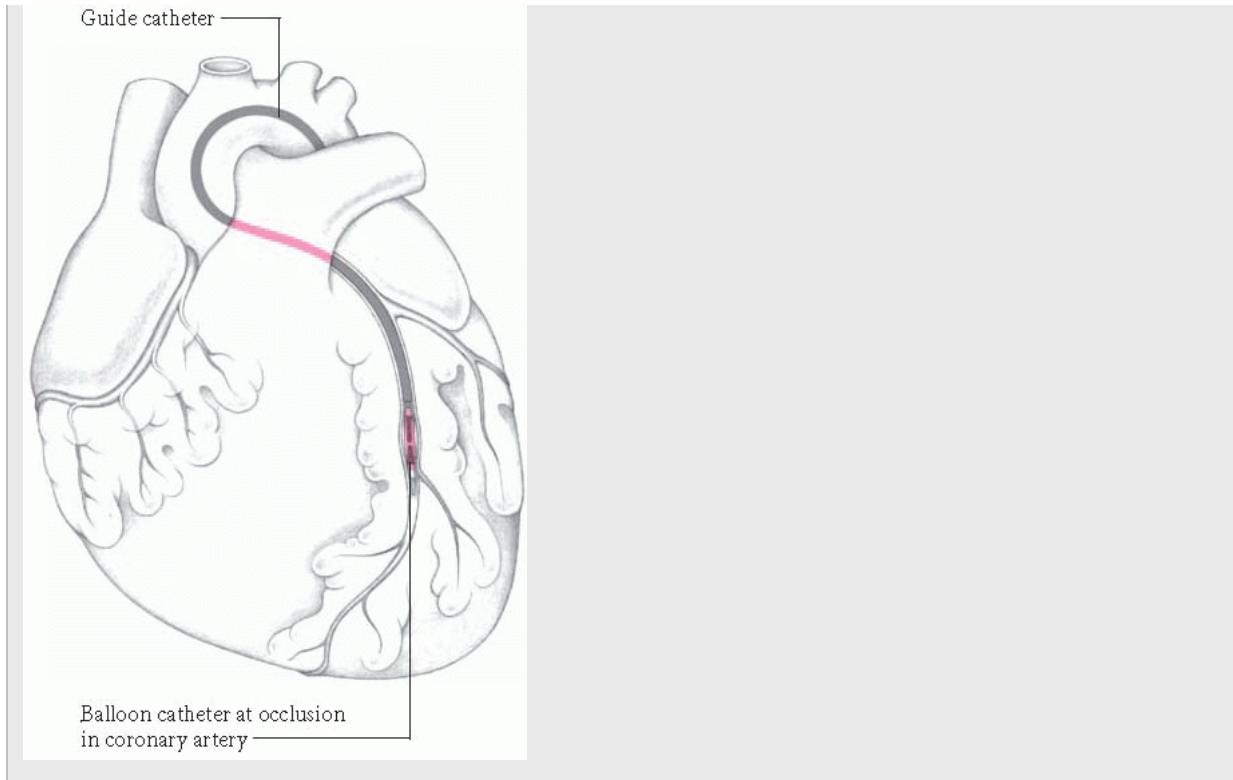
PTCA carries a certain risk but its morbidity is lower than that for surgery. (See *Relieving occlusions with angioplasty*.)

## **RELIEVING OCCLUSIONS WITH ANGIOPLASTY**

Percutaneous transluminal coronary angioplasty can open an occluded coronary without opening the chest — an important advantage over bypass surgery. First, coronary angiography must confirm the presence and location of the arterial occlusion. Then, the physician threads a guide catheter through the patient's femoral artery into the coronary artery under fluoroscopic guidance, as shown at right.

When angiography shows the guide catheter positioned at the occlusion site, the physician carefully inserts a smaller double-lumen balloon catheter through the guide catheter and directs the balloon through the occlusion (opposite page, left). A marked pressure gradient will be obvious.

The physician alternately inflates and deflates the balloon until an angiogram verifies successful arterial dilation (opposite page, right) and the pressure gradient has decreased.

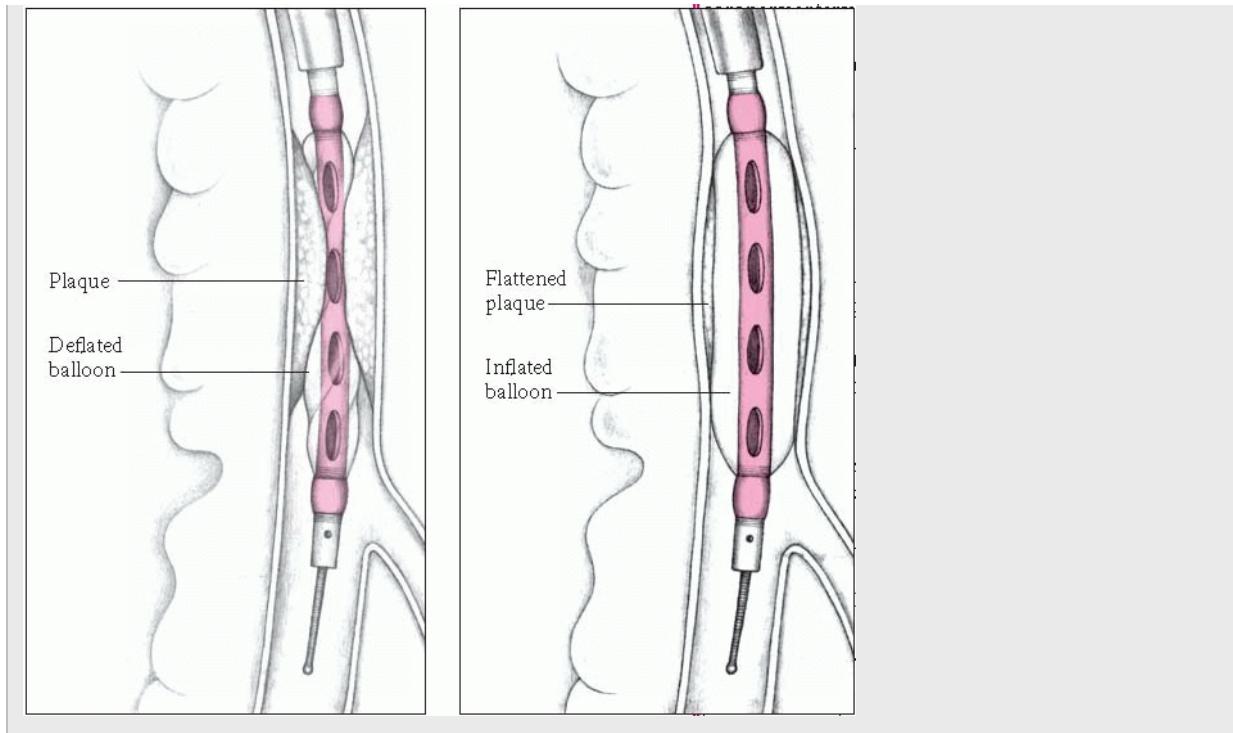


PTCA is an alternative to grafting in elderly patients or in others who can't tolerate cardiac surgery. However, patients who have a left main coronary artery occlusion, lesions in extremely tortuous vessels, or occlusions older than 3 months aren't candidates for PTCA.

PTCA can be done along with coronary stenting, or stents may be placed alone. Stents provide a framework to hold an artery open by securing the flaps of the tunica media against an artery wall. Intravascular coronary artery stenting is done to reduce the incidence of restenosis. Prosthetic cylindrical stents made of stainless steel coil are positioned at the site of occlusion. Drug-eluting stents coated with sirolimus or paclitaxel reduce restenosis rates by about 9%.

Laser angioplasty corrects occlusion by vaporizing fatty deposits with the eximeter or hot-tipped laser device. Percutaneous myocardial revascularization, or PMR, uses a laser to create channels in the heart muscle to improve perfusion to the myocardium. A carbon dioxide laser is used to create transmural channels from the epicardium to the myocardium, extending into the left ventricle. This technique is also known as transmyocardial revascularization and appears to be effective for severe symptoms. In addition, a stent may be placed in the artery to act as a scaffold to hold the artery open. Another procedure is rotational atherectomy, which removes arterial plaque with a high-speed burr. Obstructive lesions

may necessitate coronary artery bypass graft (CABG) surgery and the use of vein grafts.



A surgical technique available as an alternative to traditional CABG surgery is minimally invasive coronary artery bypass surgery, also known as “keyhole” surgery. This procedure requires a shorter recovery period and has fewer postoperative complications. Instead of sawing open the patient's sternum and spreading the ribs apart, several small cuts are made in the torso through which small surgical instruments and fiber-optic cameras are inserted. This procedure was initially designed to correct blockages in just one or two easily reached arteries; it may not be suitable for more complicated cases.

Coronary brachytherapy, which involves delivering beta or gamma radiation into the coronary arteries, may be used in patients who've undergone stent implantation in a coronary artery but then developed such problems as diffuse in-stent restenosis. Brachytherapy is a promising technique, but its use is restricted to the treatment of stentrelated problems because of complications and the unknown long-term effects of the radiation. However, in some facilities, brachytherapy is being studied as a first-line treatment of coronary disease.

## **PREVENTION**

*Because CAD is so widespread, prevention is of great importance. Encourage dietary restrictions aimed at reducing intake of calories (in obesity) and salt, saturated fats, and cholesterol, which serves to minimize the risk, especially when supplemented with regular exercise. Also, encourage patient to stop smoking and to reduce stress. Other preventive actions to encourage include control of hypertension, control of elevated serum cholesterol or triglyceride levels (with antilipemics), and measures to minimize platelet aggregation and the danger of blood clots (with aspirin or other antiplatelet drugs).*

## **Special considerations**

- During anginal episodes, monitor blood pressure and heart rate. Take an ECG during anginal episodes and before administering nitroglycerin or other nitrates. Record duration of pain, amount of medication required to relieve it, and accompanying symptoms.

- Keep nitroglycerin available for immediate use. Instruct the patient to call immediately whenever he feels chest, arm, or neck pain.
- Before cardiac catheterization, explain the procedure to the patient. Make sure he knows why it's necessary, understands the risks, and realizes that it may indicate a need for surgery.
- After catheterization, review the expected course of treatment with the patient and his family. Monitor the catheter site for bleeding. Also, check for distal pulses. To counter the dye's diuretic effect, make sure the patient drinks plenty of fluids. Assess potassium levels.
- If the patient is scheduled for surgery, explain the procedure to him and his family. Give them a tour of the intensive care unit and introduce them to the staff.
- After surgery, monitor blood pressure, intake and output, breath sounds, chest tube drainage, and ECG, watching for signs of ischemia and arrhythmias. Also, observe for and treat chest pain and possible dye reactions. Give vigorous chest physiotherapy and guide the patient in removal of secretions through deep-breathing, coughing, and expectoration of mucus.
- Before discharge, stress the need to follow the prescribed drug regimen (antihypertensives, nitrates, and antilipemics, for example), exercise program, and diet. Encourage regular, moderate exercise. Refer the patient to a self-help program to stop smoking.

## ***Myocardial infarction***

Myocardial infarction (MI), commonly known as a *heart attack* and part of a broader category of disease known as *acute coronary syndrome*, results from prolonged myocardial ischemia due to reduced blood flow through one of the coronary arteries. In cardiovascular disease, the leading cause of death in the United States and western Europe, death usually results from the cardiac damage or complications of MI. (See *Complications of myocardial infarction*.) Mortality is high when treatment is delayed, and almost one-half of sudden deaths due to an MI occur before hospitalization, within 1 hour of the onset of symptoms. The prognosis improves if vigorous treatment begins immediately.

## ***Causes and incidence***

Predisposing risk factors include:

- diabetes mellitus
- drug use, especially cocaine
- elevated serum triglyceride, total cholesterol, and low-density lipoprotein levels
- hypertension
- obesity or excessive intake of saturated fats, carbohydrates, or salt
- positive family history
- sedentary lifestyle
- smoking
- stress or a type A personality.

The site of the MI depends on the vessels involved. 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 Q-wave (transmural) MI, tissue damage extends through all myocardial layers; in non-Qwave (subendocardial) MI, only in the innermost and possibly the middle layers.

Incidence is high: About 1 million patients visit the hospital each year with an MI and another 200,000 to 300,000 people die from MI-related complications without seeking medical care. Men and postmenopausal women are more susceptible to MI than premenopausal women, although incidence is rising among females, especially those who smoke and take hormonal contraceptives.

## **Signs and symptoms**

The cardinal symptom of MI is persistent, crushing substernal pain that may radiate to the left arm, jaw, neck, or shoulder blades. Such pain is usually described as heavy, squeezing, or crushing, and may persist for 12 hours or more. However, in some MI patients – particularly elderly people or those with diabetes – pain may not occur at all; in others, it may be mild and confused with indigestion. In patients with coronary artery disease, angina of increasing frequency, severity, or duration (especially if not provoked by exertion, a heavy meal, or cold and wind) may signal impending infarction.

## **COMPLICATIONS OF MYOCARDIAL INFARCTION**

Complication	Diagnosis	Treatment
Arrhythmias	<ul style="list-style-type: none"> <li>Electrocardiogram (ECG) shows premature ventricular contractions, ventricular tachycardia, or ventricular fibrillation; in inferior wall myocardial infarction (MI), bradycardia and junctional rhythms or atrioventricular block; in anterior wall MI, tachycardia or heart block.</li> </ul>	<ul style="list-style-type: none"> <li>Antiarrhythmics, atropine, and pacemaker; cardioversion for tachycardia</li> </ul>
Heart failure	<ul style="list-style-type: none"> <li>In left-sided heart failure, chest X-rays show venous congestion, cardiomegaly, and Kerley's B lines.</li> <li>Catheterization shows increased pulmonary artery pressure (PAP) and central venous pressure.</li> </ul>	<ul style="list-style-type: none"> <li>Diuretics, angiotensin-converting enzyme inhibitors, vasodilators, inotropic agents, cardiac glycosides, and beta-adrenergic blockers</li> </ul>
Cardiogenic shock	<ul style="list-style-type: none"> <li>Catheterization shows decreased cardiac output and increased PAP and pulmonary artery wedge pressure (PAWP).</li> <li>Signs include hypertension, tachycardia, S<sub>3</sub>, S<sub>4</sub>, decreased levels of consciousness, decreased urine output, jugular vein distension, and cool, pale skin.</li> </ul>	<ul style="list-style-type: none"> <li>I.V. fluids, vasodilators, diuretics, cardiac glycosides, intra-aortic balloon pump (IABP), and beta-adrenergic stimulants</li> </ul>
Rupture of left ventricular papillary muscle	<ul style="list-style-type: none"> <li>Auscultation reveals an apical holosystolic murmur. Inspection of jugular vein pulse or hemodynamic monitoring shows increased v waves.</li> <li>Dyspnea is prominent.</li> <li>Color-flow and Doppler echocardiogram show mitral insufficiency. Pulmonary artery catheterization shows increased PAP and PAWP.</li> </ul>	<ul style="list-style-type: none"> <li>Nitroprusside (Nitropress)</li> <li>IABP</li> <li>Surgical replacement of the mitral valve with possible concomitant myocardial revascularization (in patients with significant coronary artery disease)</li> </ul>
Ventricular septal rupture	<ul style="list-style-type: none"> <li>In left-to-right shunt, auscultation reveals a holosystolic murmur and thrill.</li> <li>Catheterization shows increased PAP and PAWP.</li> <li>Confirmation is by increased oxygen saturation of the right ventricle and pulmonary artery.</li> </ul>	<ul style="list-style-type: none"> <li>Surgical correction, IABP, nitroglycerin, nitroprusside, low-dose inotropic agents, or pacemaker</li> </ul>
Pericarditis or Dressler's syndrome	<ul style="list-style-type: none"> <li>Auscultation reveals a friction rub.</li> <li>Chest pain is relieved by sitting up.</li> </ul>	<ul style="list-style-type: none"> <li>Aspirin</li> </ul>
Ventricular aneurysm	<ul style="list-style-type: none"> <li>Chest X-ray may show cardiomegaly.</li> <li>ECG may show arrhythmias and persistent ST-segment elevation.</li> </ul>	<ul style="list-style-type: none"> <li>Cardioversion, defibrillation, antiarrhythmics, vasodilators, anticoagulants, cardiac glycosides, and</li> </ul>

	<ul style="list-style-type: none"> <li>Left ventriculography shows altered or paradoxical left ventricular motion.</li> </ul>	diuretics (If conservative treatment fails, surgical resection is necessary.)
Thromboembolism	<ul style="list-style-type: none"> <li>Severe dyspnea and chest pain or neurologic changes.</li> <li>Nuclear scan shows ventilation/perfusion mismatch.</li> <li>Angiography shows arterial blockage.</li> </ul>	<ul style="list-style-type: none"> <li>Oxygen and heparin</li> </ul>

Other clinical effects include a feeling of impending doom, fatigue, nausea, vomiting, and shortness of breath. Some patients may have no symptoms. The patient may experience catecholamine responses, such as coolness in extremities, perspiration, anxiety, and restlessness. Fever is unusual at the onset of an MI, but a low-grade temperature elevation may develop during the next few days. Blood pressure varies; hypotension or hypertension may be present.

The most common post-MI complications include recurrent or persistent chest pain, arrhythmias, left-sided heart failure (resulting in heart failure or acute pulmonary edema), and cardiogenic shock. Unusual but potentially lethal complications that may develop soon after infarction include thromboembolism; papillary muscle dysfunction or rupture, causing mitral insufficiency; rupture of the ventricular septum, causing ventricular septal defect; rupture of the myocardium; and ventricular aneurysm. Up to several months after infarction, Dressler's syndrome may develop (pericarditis, pericardial friction rub, chest pain, fever, leukocytosis and, possibly, pleurisy or pneumonitis).

## Diagnosis

### CONFIRMING DIAGNOSIS

*Persistent chest pain, elevated ST segment on electrocardiogram (ECG), and elevated total creatine kinase (CK) and CK-MB levels over a 72-hour period usually confirm MI. Troponin T or troponin I is also used in the diagnosis because both are specific to cardiac necrosis, and levels rise 6 to 8 hours after onset of ischemia.*

Auscultation may reveal diminished heart sounds, gallops and, in papillary dysfunction, the apical systolic murmur of mitral insufficiency over the mitral valve area.

When clinical features are equivocal, assume that the patient had an MI until tests rule it out. Diagnostic laboratory results include:

- serial 12-lead ECG – ECG abnormalities may be absent or inconclusive during the first few hours after an MI. When present, characteristic abnormalities include serial ST-segment depression in non-Q-wave (subendocardial) MI and ST-segment elevation in Q-wave (transmural) MI.
- serial serum enzyme levels – CK levels are elevated, specifically, the CK-MB isoenzyme.
- echocardiography – may show ventricular wall motion abnormalities in patients with a Q-wave (transmural) MI.
- nuclear ventriculography scans (multiple gated acquisition or radionuclide ventriculography) – using I.V. radioactive substance, can identify acutely damaged muscle by picking up radioactive nucleotide, which appears as a “hot spot” on the film; useful in localizing a recent MI.

## Treatment

The goals of treatment are to relieve chest pain, stabilize heart rhythm, reduce cardiac workload, revascularize the coronary artery, and preserve myocardial tissue. Arrhythmias, the predominant problem during the first 48 hours after the infarction, may require antiarrhythmics, possibly a pacemaker and, rarely, cardioversion. Arrhythmias are best detected using a 12-lead ECG.

To preserve myocardial tissue in ST elevation MI, fibrinolytic therapy should be started I.V. within 30 minutes of arrival in the emergency department, if not contraindicated. Fibrinolytic therapy includes a choice of streptokinase, alteplase, urokinase, tenecteplase, or reteplase. (See *Comparing thrombolytics*, pages 50 and 51.)

Primary percutaneous transluminal coronary angioplasty is a Class I recommendation as an alternative to thrombolytic therapy only if performed in a timely manner by physicians skilled in the procedure and supported by experienced personnel in high-volume centers.

Other treatments consist of:

- lidocaine, vasopressin, or amiodarone for ventricular arrhythmias, or other drugs, such as procainamide, quinidine, or disopyramide
- antiplatelet therapy with glycoprotein IIb-IIIa inhibitors, such as ticlopidine and clopidogrel for non-ST-elevation MI
- atropine I.V. or a temporary pacemaker for heart block or bradycardia
- nitroglycerin (sublingual, topical, transdermal, or I.V.); calcium channel blockers, such as nifedipine, verapamil, or diltiazem (sublingual, oral, or I.V.); or isosorbide dinitrate (sublingual, oral, or I.V.) to relieve pain by redistributing blood to ischemic areas of the myocardium, increasing cardiac output, and reducing myocardial workload
- heparin I.V. (usually follows thrombolytic therapy)
- morphine I.V. for pain and sedation
- bed rest with bedside commode to decrease cardiac workload
- oxygen administration at a modest flow rate for 2 to 3 hours (a lower concentration is necessary if the patient has chronic obstructive pulmonary disease)
- angiotensin-converting enzyme inhibitors for patients with large anterior wall MIs and for those with an MI and a left ventricular ejection fraction less than 40%
- drugs to increase myocardial contractility or blood pressure
- beta-adrenergic blockers, such as propranolol or atenolol, after acute MI to help prevent reinfarction by reducing the heart's workload
- aspirin to inhibit platelet aggregation (should be initiated immediately and continued for years)
- pulmonary artery catheterization to detect left- or right-sided heart failure and to monitor the patient's response to treatment.

## ***Special considerations***

Care for patients who have suffered an MI is directed toward detecting complications, preventing further myocardial damage, and promoting comfort, rest, and emotional well-being. Most MI patients receive treatment in the intensive care unit (ICU), where they're under constant observation for complications.

- On admission to the ICU, monitor and record the patient's ECG, blood pressure, temperature, and heart and breath sounds.
- Assess and record the severity and duration of pain, and administer analgesics. Avoid I.M. injections; absorption from the muscle is unpredictable and bleeding is likely if the patient is receiving thrombolytic therapy.
- Check the patient's blood pressure after giving nitroglycerin, especially the first dose.
- Frequently monitor the ECG to detect rate changes or arrhythmias. Place rhythm strips in the patient's chart periodically for evaluation.

- During episodes of chest pain, obtain 12-lead ECG (before and after nitroglycerin therapy as well), blood pressure, and

pulmonary artery catheter measurements and monitor them for changes.

- Watch for signs and symptoms of fluid retention (crackles, cough, tachypnea, and edema), which may indicate impending heart failure. Carefully monitor daily weight, intake and output, respirations, serum enzyme levels, and blood pressure. Auscultate for adventitious breath sounds periodically (patients on bed rest frequently have atelectatic crackles, which disappear after coughing), for  $S_3$  or  $S_4$  gallops, and for new-onset heart murmurs.
- Organize patient care and activities to maximize periods of uninterrupted rest.
- Initiate a cardiac rehabilitation program. This usually includes education regarding heart disease, exercise, and emotional support for the patient and his family.
- Ask the dietary department to provide a clear liquid diet until nausea subsides. A low-cholesterol, low-sodium, low-fat, high-fiber diet may be prescribed.

- Provide a stool softener to prevent straining during defecation, which causes vagal stimulation and may slow the heart rate. Allow use of a bedside commode and provide as much privacy as possible.
- Assist with range-of-motion exercises. If the patient is completely immobilized by a severe MI, turn him often. Antiembolism stockings help prevent venostasis and thrombophlebitis.
- Provide emotional support and help reduce stress and anxiety; administer tranquilizers as needed. Explain procedures and answer questions. Explaining the ICU environment and routine can ease anxiety. Involve the patient's family in his care as much as possible.

## **COMPARING THROMBOLYTICS**

If your patient has suffered a myocardial infarction (MI), you must intervene promptly to minimize cardiac damage and avert death. If appropriate, prepare the patient for thrombolytic therapy as ordered.

Thrombolytic drugs enhance the body's natural ability to dispose of blood clots. To lyse (dissolve) fibrin, the essential component of a clot, tissue activators convert plasminogen to plasmin. A nonspecific protease, plasmin degrades fibrin, fibrinogen, and procoagulant factors (such as factors V, VII, and XII). Candidates for thrombolytic therapy include patients with acute ST-segment elevation and chest pain that has lasted no more than 6 hours. Timely use of thrombolytic agents can restore myocardial perfusion and prevent further injury. When effective, thrombolytic agents relieve chest pain, restore the ST segment to baseline, and induce reperfusion arrhythmias within 30 to 45 minutes.

Contraindications to thrombolytic therapy include surgery within the past 2 months, active bleeding, a history of stroke, intracranial neoplasm, arteriovenous malformation, aneurysm, or uncontrolled hypertension.

Here's how selected thrombolytics open occluded coronary arteries in patients with an acute MI.

### **Alteplase (Activase)**

This naturally occurring enzyme has been cloned and produced as a drug, alteplase (tissue plasminogen activator). Binding to plasminogen, it catalyzes the conversion of plasminogen to plasmin in the presence of fibrin. Because of

its strong affinity for fibrin, alteplase concentrates at the clot site, resulting in a minimal decrease in the fibrinogen level.

This thrombolytic has a half-life of 5 minutes, so maintaining coronary artery patency depends on continued anticoagulation with heparin. Alteplase doesn't induce antigenic responses; doses may be repeated at any time.

### **Reteplase (Retevase)**

Reteplase, recombinant plasminogen activator, has a half-life of 13 to 16 minutes. Its longer half-life allows it to be administered as a bolus. Two boluses are required.

### **Anistreplase (Eminase)**

Anistreplase is a partially synthetic thrombolytic drug that's composed of a complex of streptokinase bound to human plasminogen.

This complex binds to fibrin and promotes plasminogen conversion to plasmin. But the dose needed to lyse coronary artery clots can cause systemic clot lysis, characterized by fibrinogen depletion. This results in bleeding complications.

Anistreplase has the longest half-life (90 minutes). Because it's partially composed of streptokinase, a foreign protein, anistreplase is antigenic and may cause an allergic reaction. The per-dose cost is less than that for alteplase.

This drug's main advantage is ease of administration: only a single bolus is required.

### **Streptokinase (Streptase)**

Streptokinase, a thrombolytic, is a bacterial protein that binds to circulating plasminogen and catalyzes plasmin formation. Its low specificity for fibrin induces a systemic lytic state and increases the risk of bleeding.

The half-life is approximately 20 minutes. Like anistreplase, streptokinase is antigenic.

### **Tenecteplase (TNKase)**

Tenecteplase is a modified form of human tissue plasminogen activator that binds to fibrin and converts plasminogen to plasmin. It's given as a single bolus dose.

### **Urokinase (Abbokinase)**

Naturally produced by the human kidney, urokinase promotes thrombolysis by directly activating the conversion of plasminogen to plasmin.

With a serum half-life of 10 to 20 minutes, urokinase is rapidly cleared by the kidneys and liver. Unlike anistreplase and streptokinase, it doesn't induce an antigenic response. Urokinase isn't given through a peripheral I.V. line to treat an acute MI, but patients who undergo cardiac catheterization may receive it directly in a coronary artery.

To prepare the patient for discharge:

- Thoroughly explain dosages and therapy to promote compliance with the prescribed medication regimen and other treatment measures. Warn about drug adverse effects and advise the patient to watch for and report signs of toxicity (anorexia, nausea, vomiting, and yellow vision, for example, if the patient is receiving digoxin).

- Review dietary restrictions with the patient. If he must follow a low-sodium or low-fat and low-cholesterol diet, provide a list of foods that he should avoid. Ask the dietitian to speak to the patient and his family.
- Counsel the patient to resume sexual activity progressively.
- Advise the patient to report typical or atypical chest pain. Postinfarction syndrome may develop, producing chest pain that must be differentiated from recurrent MI, pulmonary infarct, or heart failure.
- If the patient has a Holter monitor in place, explain its purpose and use.
- Stress the need to stop smoking.
- Encourage participation in a cardiac rehabilitation program.
- Review follow-up procedures, such as office visits and treadmill testing, with the patient.

## PREVENTION

- *Instruct patient to practice healthy-heart living, with a hearthealthy diet, regular exercise, stress reduction and preventive care, maintaining a healthy weight, smoking cessation, and abstinence from alcohol and illegal drugs, especially cocaine.*
- *Suggest a daily aspirin regimen for patients with coronary artery disease or history of an MI.*

## **Heart failure**

Heart failure is a syndrome characterized by myocardial dysfunction that leads to impaired pump performance (diminished cardiac output) or to frank heart failure and abnormal circulatory congestion. Congestion of systemic venous circulation may result in peripheral edema or hepatomegaly; congestion of pulmonary circulation may cause pulmonary edema, an acute life-threatening emergency. Pump failure usually occurs in a damaged left ventricle (left-sided heart failure) but may occur in the right ventricle (right-sided heart failure) either as a primary disorder or secondary to left-sided heart failure. Sometimes, leftand right-sided heart failure develop simultaneously. (See *What happens in heart failure*.)

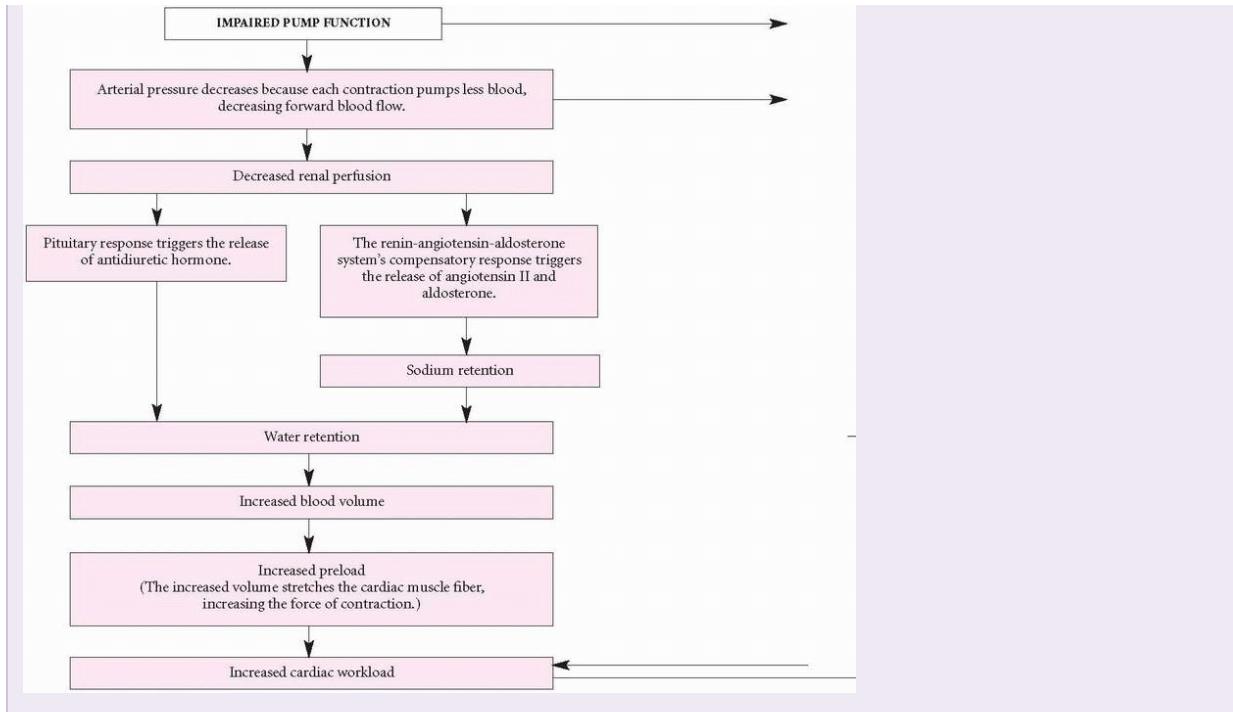
Although heart failure may be acute (as a direct result of myocardial infarction [MI]), it's generally a chronic disorder associated with sodium and water retention

by the kidneys. Advances in diagnostic and therapeutic techniques have greatly improved the outlook for patients with heart failure, but the prognosis still depends on the underlying cause and its response to treatment.

## PATHOPHYSIOLOGY

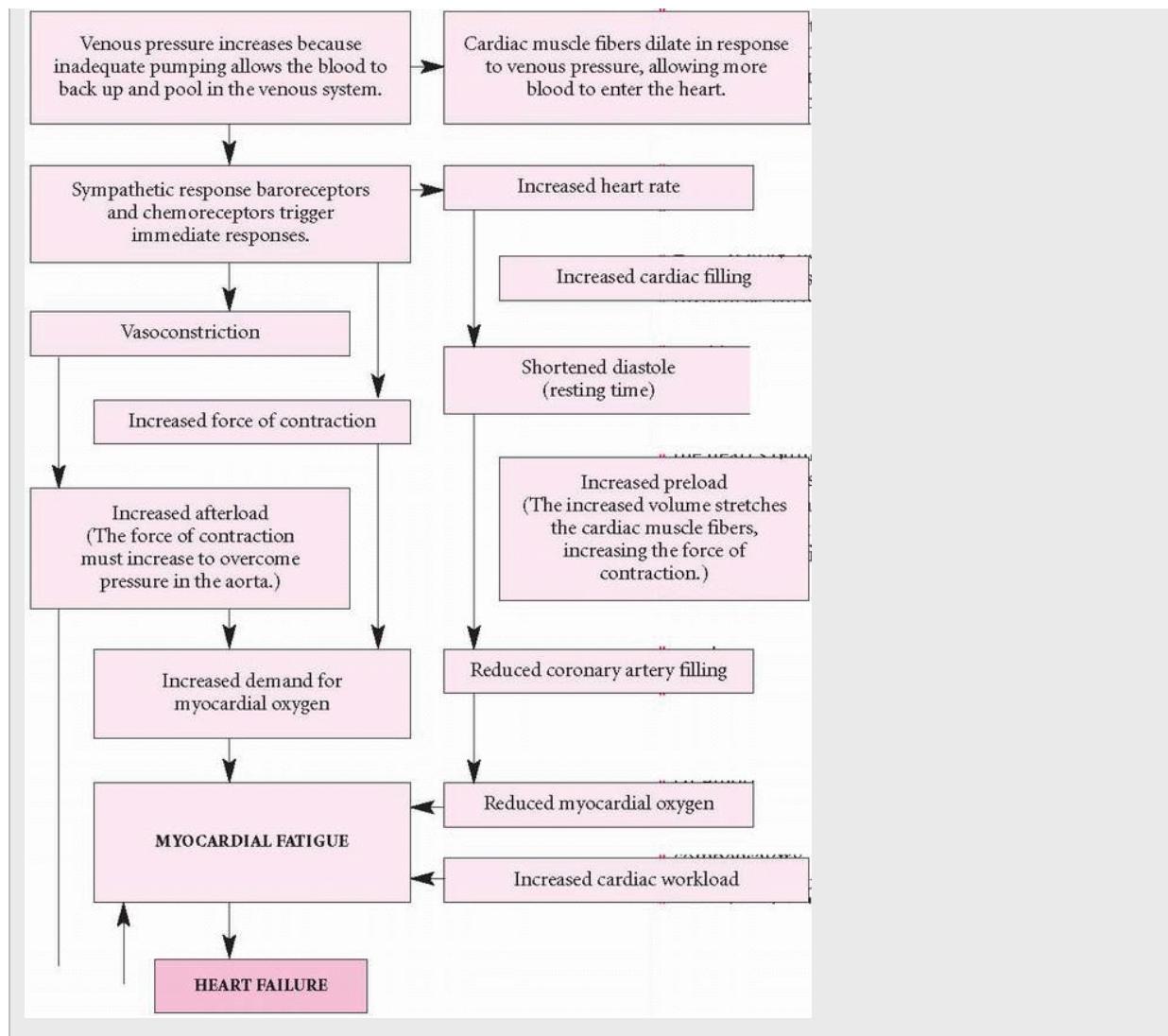
### **WHAT HAPPENS IN HEART FAILURE**

Heart failure occurs when cardiac output is inadequate to meet the body's needs. The pathophysiology of heart failure is shown in the flow chart below.



## ***Causes and incidence***

Heart failure may result from a primary abnormality of the heart muscle such as an infarction, inadequate myocardial perfusion due to coronary artery disease, or cardiomyopathy. Other causes include:



- diastolic dysfunction, preserved ejection fraction, impairment of ventricular filling by diminished relaxation or reduced compliance seen with hypertrophic cardiomyopathy, myocardial hypertrophy, and pericardial restriction
- mechanical disturbances in ventricular filling during diastole when there's too little blood for the ventricle to pump, as in mitral stenosis secondary to rheumatic heart disease or constrictive pericarditis and atrial fibrillation
- systolic hemodynamic disturbances, such as excessive cardiac workload due to volume overloading or pressure overload that limit the heart's pumping ability. These disturbances can result from mitral or aortic insufficiency, which causes volume overloading, and aortic stenosis or systemic hypertension, which result in increased resistance to ventricular emptying. Reduced cardiac output triggers compensatory mechanisms, such as ventricular dilation, hypertrophy, increased sympathetic activity, and activation of the renin-angiotensinaldosterone system. These mechanisms improve cardiac output at the expense of increased ventricular work. In cardiac dilation, an increase in end-diastolic ventricular volume (preload) causes increased stroke work and stroke volume during contraction, stretching cardiac muscle fibers beyond optimum

limits and producing pulmonary congestion and pulmonary hypertension, which in turn lead to right-sided heart failure.

In ventricular hypertrophy, an increase in muscle mass or diameter of the left ventricle allows the heart to pump against increased resistance (impedance) to the outflow of blood. An increase in ventricular diastolic pressure necessary to fill the enlarged ventricle may compromise diastolic coronary blood flow, limiting the oxygen supply to the ventricle and causing ischemia and impaired muscle contractility.

Increased sympathetic activity occurs as a response to decreased cardiac output and blood pressure by enhancing peripheral vascular resistance, contractility, heart rate, and venous return. Signs of increased sympathetic activity, such as cool extremities and clamminess, may indicate impending heart failure. Increased sympathetic activity also restricts blood flow to the kidneys, which respond by reducing the glomerular filtration rate and increasing tubular reabsorption of salt and water, in turn expanding the circulating blood volume. This renal mechanism, if unchecked, can aggravate congestion and produce overt edema.

Chronic heart failure may worsen as a result of respiratory tract infections, pulmonary embolism, stress, increased sodium or water intake, or failure to adhere to the prescribed treatment regimen.

Heart failure affects about 2 of every 100 people between ages 27 and 74. It becomes more common with advancing age.

## ***Complications***

- Pulmonary edema
- Multi-organ failure
- Myocardial infarction

## ***Signs and symptoms***

Left-sided heart failure primarily produces pulmonary signs and symptoms; right-sided heart failure, primarily systemic signs and symptoms. However, heart failure often affects both sides of the heart.

Clinical signs of left-sided heart failure include dyspnea, orthopnea, crackles, possibly wheezing, hypoxia, respiratory acidosis, cough, cyanosis or pallor, palpitations, arrhythmias, elevated blood pressure, and pulsus alternans.

Clinical signs of right-sided heart failure include dependent peripheral edema, hepatomegaly, splenomegaly, jugular vein distention, ascites, slow weight gain, arrhythmias, positive hepatojugular reflex, abdominal distention, nausea, vomiting, anorexia, weakness, fatigue, dizziness, and syncope.

### **ALERT**

*Excessive fluid can accumulate in the pericardium, requiring removal through pericardiocentesis.*

## ***Diagnosis***

- Electrocardiography may reflect heart strain or enlargement, ischemia, or old MI. It may also reveal atrial enlargement, tachycardia, and extrasystoles.
- Chest X-ray shows increased pulmonary vascular markings, interstitial edema, or pleural effusion and cardiomegaly.
- Pulmonary artery pressure monitoring typically demonstrates elevated pulmonary artery and pulmonary artery wedge pressures, left ventricular end-diastolic pressure in left-sided heart failure, and elevated right atrial pressure or central venous pressure in right-sided heart failure.
- B-type natriuretic peptide (BNP) is a neuro-hormone produced predominantly by the heart ventricle and is released in response to blood volume expansion or pressure overload. Blood concentrations

greater than 100 pg/ml are an accurate predictor of heart failure. (See *Linking BNP levels to heart failure symptom severity*.)

- Echocardiogram may demonstrate wall motion abnormalities and chamber dilation.

Other tests that may also demonstrate enlargement of the heart or decreased functioning include chest computed tomography scan, cardiac magnetic resonance imaging, or nuclear scans, such as multiple-gated acquisition scanning and radionuclide ventriculography.

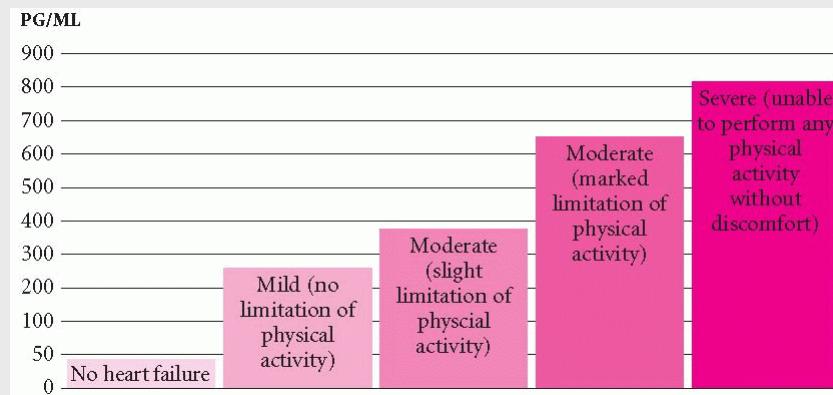
## Treatment

The goal of therapy is to improve pump function by reversing the compensatory mechanisms producing the clinical effects, underlying disorders, and precipitating factors.

Heart failure can be quickly controlled by treatment consisting of:

### LINKING BNP LEVELS TO HEART FAILURE SYMPTOM SEVERITY

This chart shows the level of B-type natriuretic peptide (BNP) levels and its correlation with symptoms of heart failure. The higher the level of BNP, the more severe the symptoms.



- angiotensin-converting enzyme inhibitors to decrease peripheral vascular resistance
- antiembolism stockings to reduce the risk of venostasis and thromboembolus formation
- bed rest for acute heart failure
- carvedilol, a nonselective beta-adrenergic blocker with alpha-receptor blockade to reduce mortality and improve quality of life
- digoxin or dopamine to strengthen myocardial contractility
- diuresis to reduce total blood volume and circulatory congestion
- inotropic agents, such as dobutamine and milrinone, given I.V. to improve the heart's ability to pump
- nesiritide, a recombinant form of endogenous human B-type natriuretic peptide, to reduce sodium through its diuretic action
- vasodilators to increase cardiac output by reducing the impedance to ventricular outflow (afterload).

Excess fluid can be removed through dialysis if necessary. Circulatory assistance can be provided by implanted devices, such as the intra-aortic balloon pump and the left ventricular assist device, but they're only temporary solutions.

Left ventricular remodeling surgery may also be performed. This surgical technique involves cutting a wedge about the size of a small slice of pie out of the left ventricle of an enlarged heart. The remainder of the heart is sewn together. The result is a smaller organ that's able to pump blood more efficiently. This procedure offers promising results, especially for those whose only alternative may be a heart transplant.

Watch for and treat complications, which typically may include pulmonary edema (see *Pulmonary edema: How to intervene*); venostasis, with predisposition to thromboembolism (associated primarily with prolonged bed rest); cerebral insufficiency; and renal insufficiency, with severe electrolyte imbalance.

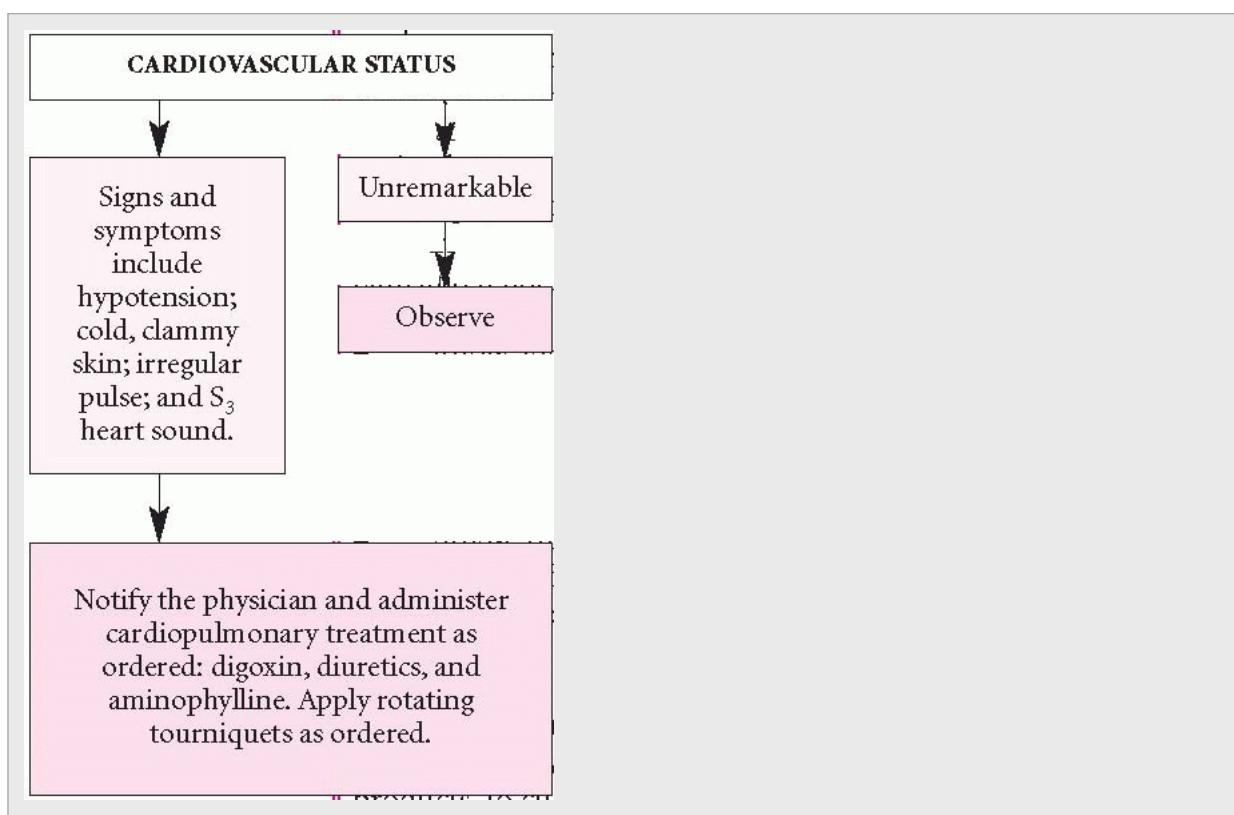
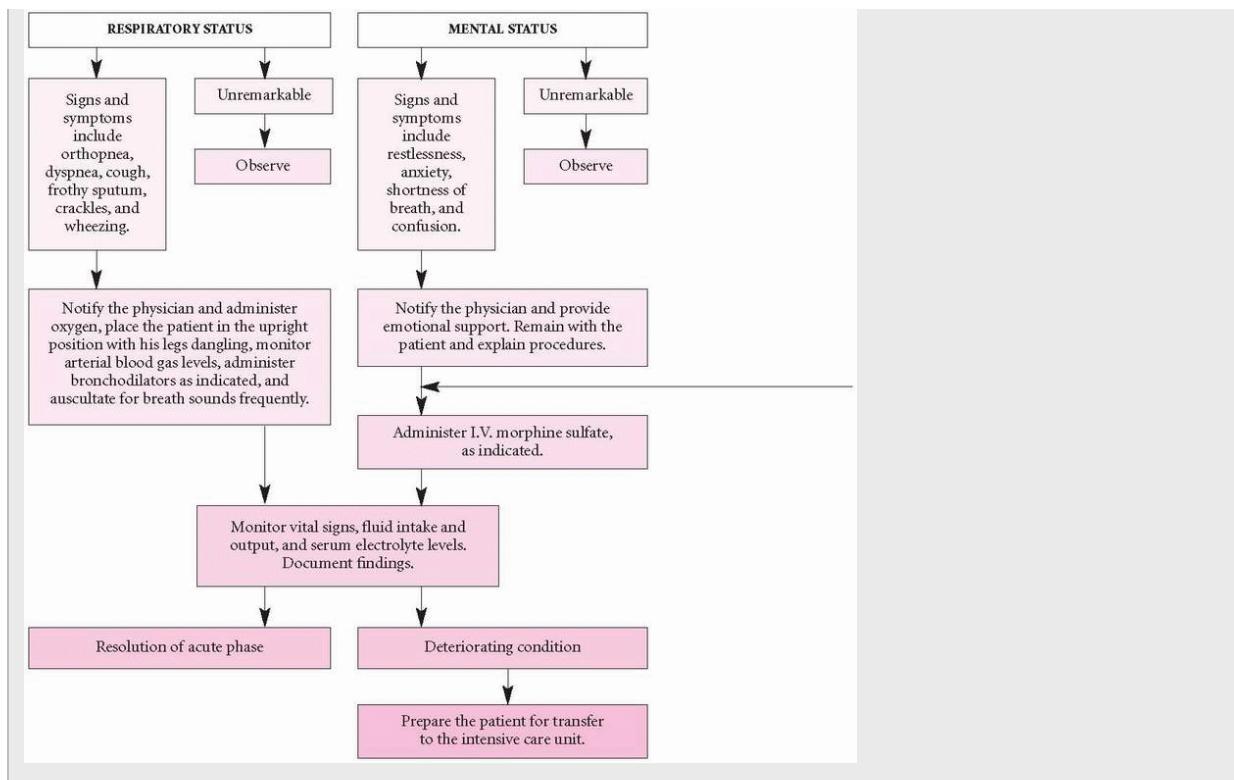
### ***Special considerations***

During the acute phase of heart failure:

- Place the patient in Fowler's position and give him supplemental oxygen to help him breathe more easily.
- Weigh the patient daily and check for peripheral edema. Carefully monitor I.V. intake and urine output, vital signs, and mental status. Auscultate the heart for abnormal sounds (S3 gallop) and the lungs for crackles or rhonchi. Report changes at once.
- Frequently monitor blood urea nitrogen, creatinine, and serum potassium, sodium, chloride, and magnesium levels.
- Make sure the patient has continuous cardiac monitoring during acute and advanced stages to identify and treat arrhythmias promptly.
- To reduce the risk of deep vein thrombosis due to vascular congestion, assist the patient with range-of-motion exercises. Enforce bed rest and apply antiembolism stockings. Check regularly for calf pain and tenderness.
- Allow adequate rest periods.

### **PULMONARY EDEMA: HOW TO INTERVENE**

Obtain the patient history, assist with diagnostic tests, and assess respiratory, mental, and cardiovascular status.



To prepare the patient for discharge:

- Advise the patient to avoid foods high in sodium, such as canned or commercially prepared foods and dairy products, to curb fluid overload.
- Encourage participation in an outpatient cardiac rehabilitation program.
- Explain to the patient that the potassium he loses through diuretic therapy may need to be replaced by taking a prescribed potassium supplement and eating highpotassium foods, such as bananas and apricots.
- Stress the need for regular checkups.
- Stress the importance of taking digoxin exactly as prescribed. Tell the patient to watch for and immediately report signs of toxicity, such as anorexia, vomiting, and yellow vision.
- Tell the patient to notify the practitioner promptly if his pulse is unusually irregular or measures less than 60 beats/minute; if he experiences dizziness, blurred vision, shortness of breath, a persistent dry cough, palpitations, increased fatigue, paroxysmal nocturnal dyspnea, swollen ankles, or decreased urine output; or if he notices rapid weight gain (3 to 5 lb [1.4 to 2.3 kg] in 1 week).

## PREVENTION

- *Instruct patient to make lifestyle modifications, including regular exercise, weight loss, smoking cessation, stress reduction, and reduced sodium, alcohol, and fat intake.*
- *Instruct patient to practice compliance with and timely administration of maintenance doses of diuretics and cardiac drugs.*

## **Dilated cardiomyopathy**

Dilated cardiomyopathy results from extensively damaged myocardial muscle fibers. This disorder interferes with myocardial metabolism and grossly dilates all four chambers of the heart, giving the heart a globular appearance and shape. In this disorder, hypertrophy may be present. Dilated cardiomyopathy leads to intractable heart failure, arrhythmias, and emboli. Because this disease isn't usually diagnosed until it's in the advanced stages, the patient's prognosis is generally poor.

## **Causes and incidence**

The cause of most cardiomyopathies is unknown. Occasionally, dilated cardiomyopathy results from myocardial destruction by toxic, infectious, or metabolic agents, such as certain viruses, endocrine and electrolyte disorders, and nutritional deficiencies. Other causes include muscle disorders (myasthenia gravis, progressive muscular dystrophy, and myotonic dystrophy), infiltrative disorders (hemochromatosis and amyloidosis), and sarcoidosis.

Cardiomyopathy may also be a complication of alcoholism. In such cases, it may improve with abstinence from alcohol but recurs when the patient resumes drinking. How viruses induce cardiomyopathy is unclear, but researchers suspect a link between viral myocarditis and subsequent dilated cardiomyopathy, especially after infection

with poliovirus, coxsackievirus B, influenza virus, or human immunodeficiency virus.

Metabolic cardiomyopathies are related to endocrine and electrolyte disorders and nutritional deficiencies. Thus, dilated cardiomyopathy may develop in patients with hyperthyroidism, pheochromocytoma, beriberi (thiamine deficiency), or kwashiorkor (protein deficiency).

Cardiomyopathy may also result from rheumatic fever, especially among children with myocarditis.

Antepartal or postpartal cardiomyopathy may develop during the last trimester or within months after delivery. Its cause is unknown, but it occurs most frequently in multiparous women older than age 30, particularly those with malnutrition or preeclampsia. In these patients, cardiomegaly and heart

failure may reverse with treatment, allowing a subsequent normal pregnancy. If cardiomegaly persists despite treatment, the prognosis is poor.

Dilated cardiomyopathy occurs in 2 of every 100 people and affects all ages and sexes. It's most common in adult men.

## ***Complications***

- Heart failure
- Arrhythmias
- Emboli
- Ventricular arrhythmias
- Syncope
- Sudden death

## ***Signs and symptoms***

In dilated cardiomyopathy, the heart ejects blood less efficiently than normal. Consequently, a large volume of blood remains in the left ventricle after systole, causing signs of heart failure—both left-sided (shortness of breath, orthopnea, dyspnea on exertion, paroxysmal nocturnal dyspnea, fatigue, and an irritating dry cough at night) and right-sided (edema, liver engorgement, and jugular vein distention). Dilated cardiomyopathy also produces peripheral cyanosis and sinus tachycardia or atrial fibrillation at rest in some patients secondary to low cardiac output. Auscultation reveals diffuse apical impulses, pansystolic murmur (mitral and tricuspid insufficiency secondary to cardiomegaly and weak papillary muscles), and  $S_3$  and  $S_4$  gallop rhythms.

## ***Diagnosis***

No single test confirms dilated cardiomyopathy. Diagnosis requires elimination of other possible causes of heart failure and arrhythmias.

- Electrocardiography (ECG) and angiography rule out ischemic heart disease; ECG may also show biventricular hypertrophy, sinus tachycardia, atrial enlargement and, in 20% of patients, atrial fibrillation and bundle-branch block.
- Chest X-ray shows cardiomegaly—usually affecting all heart chambers—and may demonstrate pulmonary congestion, pleural or pericardial effusion, or pulmonary venous hypertension.
- Chest computed tomography scan or echocardiography identifies left ventricular thrombi, global hypokinesia, and degree of left ventricular dilation.
- Nuclear heart scans, such as multiple-gated acquisition scanning and ventriculography, show heart enlargement, lung congestion, heart failure, and decreased movement or functioning of the heart.

## ***Treatment***

Therapeutic goals include correcting the underlying causes and improving the heart's pumping ability with digoxin, diuretics, oxygen, and a sodium-restricted diet. Other options may involve bed rest and steroids. Vasodilators reduce preload and afterload, thereby decreasing congestion and increasing cardiac output. Acute heart failure requires vasodilation with nitroprusside or nitroglycerin I.V. Long-term treatment may include prazosin, hydralazine, isosorbide dinitrate, angiotensin-converting enzyme inhibitors, and anticoagulants.

When these treatments fail, therapy may require a heart transplant for carefully selected patients. Cardiomyoplasty, which wraps the latissimus dorsi muscle around the ventricles, assists the ventricle

to effectively pump blood. A cardiomystimulator delivers bursts of electrical impulses during systole to contract the muscle.

## ***Special considerations***

In the patient with acute failure:

- Monitor for signs of progressive failure (increasing crackles and dyspnea and increased jugular vein distention) and compromised renal perfusion (oliguria, elevated blood urea nitrogen and creatinine levels, and electrolyte imbalances). Weigh the patient daily.
- If the patient is receiving vasodilators, check blood pressure and heart rate. If he becomes hypotensive, stop the infusion and place him in a supine position, with legs elevated to increase venous return and to ensure cerebral blood flow.
- If the patient is receiving diuretics, monitor for signs of resolving congestion (decreased crackles and dyspnea) or too vigorous diuresis. Check serum potassium level for hypokalemia, especially if therapy includes digoxin.
- Therapeutic restrictions and an uncertain prognosis usually cause profound anxiety and depression, so offer support and let the patient express his feelings. Be flexible with visiting hours.
- Before discharge, teach the patient about his illness and its treatment. Emphasize the need to avoid alcohol, smoking, to restrict sodium intake, to watch for weight gain (a weight gain of 3 lb [1.4 kg] over 1 to 2 days indicates fluid accumulation), and to take digoxin as prescribed, and watch for its adverse effects (anorexia, nausea, vomiting, and yellow vision).
- Encourage family members to learn cardiopulmonary resuscitation.

## ***Hypertrophic cardiomyopathy***

This primary disease of cardiac muscle, also called *idiopathic hypertrophic subaortic stenosis*, is characterized by disproportionate, asymmetrical thickening of the interventricular septum, particularly in the left ventricle's free wall. In hypertrophic cardiomyopathy, cardiac output may be low, normal, or high, depending on whether stenosis is obstructive or nonobstructive. If cardiac output is normal or high, the disorder may go undetected for years; but low cardiac output may lead to potentially fatal heart failure. The disease course varies; some patients progressively deteriorate; others remain stable for years.

## ***Causes and incidence***

Despite being designated as idiopathic, hypertrophic cardiomyopathy may be inherited as a non-sex-linked autosomal dominant trait in almost all cases. Most patients have obstructive disease, resulting from effects of ventricular septal hypertrophy and the movement of the anterior mitral valve leaflet into the outflow tract during systole. Eventually, left ventricular dysfunction, from rigidity and decreased compliance, causes pump failure.

This disorder affects 2 to 5 of every 1,000 people and is usually the cause of sudden death, particularly in otherwise healthy athletes.

## ***Complications***

- Pulmonary hypertension
- Heart failure
- Ventricular arrhythmias
- Sudden death

## **Signs and symptoms**

Clinical features of the disorder may not appear until it's well advanced, when atrial dilation and, possibly, atrial fibrillation abruptly reduce blood flow to the left ventricle. Reduced inflow and subsequent low output may produce angina pectoris, arrhythmias, dyspnea, orthopnea, syncope, heart failure, and death. Auscultation reveals a medium-pitched systolic ejection murmur along the left sternal border and at the apex; palpation reveals a peripheral pulse with a characteristic double impulse (pulsus bifidus) and, with atrial fibrillation, an irregular pulse.

## **Diagnosis**

Diagnosis depends on typical clinical findings and these test results:

- Echocardiography (most useful) shows increased thickness of the intra-ventricular septum and abnormal motion of the anterior mitral leaflet during systole, occluding left ventricular outflow in obstructive disease.
- Cardiac catheterization reveals elevated left ventricular end-diastolic pressure and, possibly, mitral insufficiency.
- Electrocardiography usually shows left ventricular hypertrophy, T-wave inversion, left anterior hemiblock, Q waves in precordial and inferior leads, ventricular arrhythmias and, possibly, atrial fibrillation.
- Auscultation confirms an early systolic murmur.

## **Treatment**

The goals of treatment are to relax the ventricle and to relieve outflow tract obstruction. Agents such as propranolol, a beta-adrenergic blocker, slow heart rate and increase ventricular filling by relaxing the obstructing muscle, thereby reducing angina, syncope, dyspnea, and arrhythmias. However, propranolol may aggravate symptoms of cardiac decompensation. Atrial fibrillation necessitates cardioversion to treat the arrhythmia and, because of the high risk of systemic embolism, anticoagulant therapy until fibrillation subsides. Because vasodilators such as nitroglycerin reduce venous return by permitting pooling of blood in the periphery, decreasing ventricular volume and chamber size, and may cause further obstruction, they're contraindicated in patients with hypertrophic cardiomyopathy. Also contraindicated are sympathetic stimulators such as isoproterenol, which enhance cardiac contractility and myocardial demands for oxygen, intensifying the obstruction. Although quinidine is used to suppress ventricular arrhythmia, disopyramide is preferred because of its negative inotropic properties. Patients with potentially lethal arrhythmias may need an implantable-cardioverter defibrillator to prevent sudden death.

If drug therapy fails, surgery is indicated. Ventricular myotomy (resection of the hypertrophied septum) or ventricular myectomy (removal of the hypertrophied septum) alone or combined with mitral valve replacement may ease outflow tract obstruction and relieve symptoms. However, ventricular myotomy may cause complications, such as complete heart block and ventricular septal defect.

## **Special considerations**

- Because syncope or sudden death may follow well-tolerated exercise, warn such patients against strenuous physical activity such as running.
- Administer medications as prescribed. *Caution:* Avoid nitroglycerin, digoxin, and diuretics because they can worsen obstruction. Warn the patient not to stop taking propranolol abruptly, because doing so may increase myocardial demands. To determine the patient's tolerance for an increased

dosage of propranolol, take his pulse to check for bradycardia. Also take his blood pressure while he's supine and standing (a drop in blood pressure [more than 10 mm Hg] when standing may indicate orthostatic hypotension).

- Before dental work or surgery, tell the patient to discuss prophylaxis for subacute infective endocarditis with his health care provider.
- Provide psychological support. If the patient is hospitalized for a long time, be flexible with visiting hours and encourage occasional weekends away from the hospital, if possible. Refer the patient for psychosocial counseling to help him and his family accept his restricted lifestyle and poor prognosis.
- If the patient is a child, have his parents arrange for him to continue his studies in the health care facility.
- Because sudden cardiac arrest is possible, urge the patient's family to learn cardiopulmonary resuscitation.

## CARDIAC COMPLICATIONS

### ***Hypovolemic shock***

In hypovolemic shock, reduced intravascular blood volume causes circulatory dysfunction and inadequate tissue perfusion. Without sufficient blood or fluid replacement, hypovolemic shock syndrome may lead to irreversible cerebral and renal damage, cardiac arrest and, ultimately, death. Hypovolemic shock requires early recognition of signs and symptoms and prompt,

aggressive treatment to improve the prognosis. (See *What happens in hypovolemic shock*, page 62.)

#### ***Causes and incidence***

Hypovolemic shock usually results from acute blood loss—about one-fifth of total volume. Such massive blood loss may result from GI bleeding, internal hemorrhage (hemothorax and hemoperitoneum), external hemorrhage (accidental or surgical trauma), or from any condition that reduces circulating intravascular plasma volume or other body fluids such as in severe burns. Other underlying causes of hypovolemic shock include intestinal obstruction, peritonitis, acute pancreatitis, ascites and dehydration from excessive perspiration, severe diarrhea or protracted vomiting, diabetes insipidus, diuresis, or inadequate fluid intake.

#### ***Complications***

- Acute respiratory distress syndrome
- Acute tubular necrosis
- Disseminated intravascular coagulation
- Multiple-organ-dysfunction syndrome

#### ***Signs and symptoms***

Hypovolemic shock produces a syndrome of hypotension, with narrowing pulse pressure; decreased sensorium; tachycardia; rapid, shallow respirations; reduced urine output (less than 25 ml/hour); and cold, pale, clammy skin. Metabolic acidosis with an accumulation of lactic acid develops as a result of tissue anoxia, as cellular metabolism shifts from aerobic to anaerobic pathways. Disseminated intravascular coagulation (DIC) is a possible complication of hypovolemic shock.

#### ***Diagnosis***

No single symptom or diagnostic test establishes the diagnosis or severity of shock. Characteristic laboratory findings include:

- elevated potassium, serum lactate, and blood urea nitrogen levels
- increased urine specific gravity (more than 1.020) and urine osmolality
- decreased blood pH and partial pressure of arterial oxygen and increased partial pressure of arterial carbon dioxide.

In addition, gastroscopy, aspiration of gastric contents through a nasogastric tube, computed tomography scan, and X-rays identify internal bleeding sites; coagulation studies may detect coagulopathy from DIC. Echocardiography or right heart catheterization can help differentiate between hypovolemic and cardiogenic shock.

## Treatment

Emergency treatment measures must include prompt and adequate blood and fluid replacement to restore intravascular volume and raise blood pressure. Saline solution or lactated Ringer's solution, then possibly plasma proteins (albumin) or other plasma expanders, may produce adequate volume expansion until whole blood can be matched. A rapid solution infusion system can provide these crystalloids or colloids at high flow rates. Application of a pneumatic antishock garment may be helpful. (See *Using a pneumatic antishock garment*.) Dopamine, dobutamine, epinephrine, and norepinephrine can help increase blood pressure and cardiac output after fluid resuscitation measures are done. Treatment may also include oxygen administration, identification of bleeding site, control of bleeding by direct measures (such as application of pressure and elevation of a limb) and, possibly, surgery.

## Special considerations

Management of hypovolemic shock necessitates prompt, aggressive supportive measures and careful assessment and monitoring of vital signs. Follow these priorities:

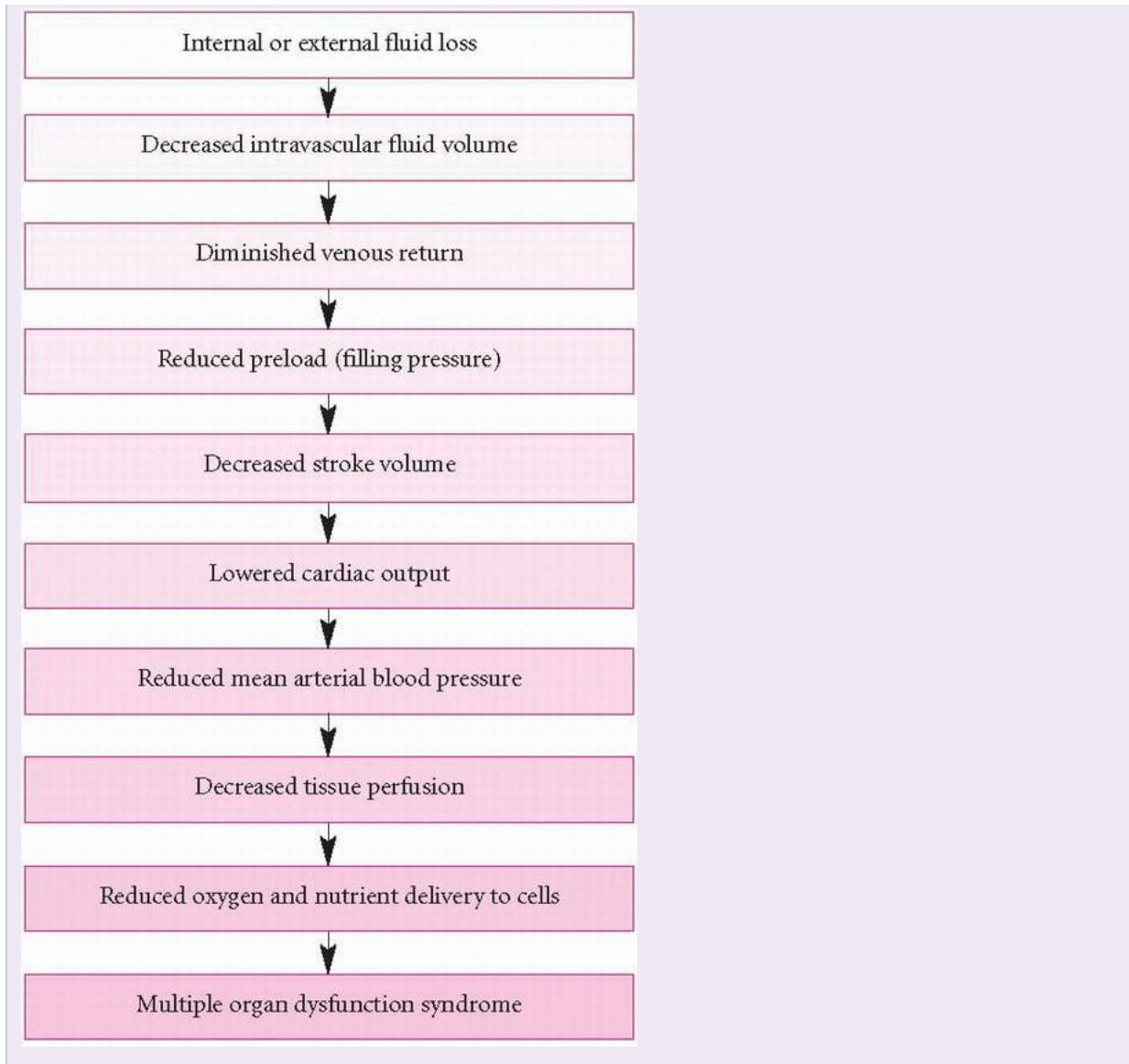
- Check for a patent airway and adequate circulation. If blood pressure and heart rate are absent, start cardiopulmonary resuscitation.
- Record blood pressure, pulse rate, peripheral pulses, respiratory rate, and other vital signs every 15 minutes and the electrocardiograph continuously. Systolic blood pressure lower than 80 mm Hg usually results in inadequate coronary artery blood flow, cardiac ischemia, arrhythmias, and further complications of low cardiac output. When blood pressure drops below 80 mm Hg, increase the oxygen flow rate and notify the practitioner immediately.

progressive drop in blood pressure, accompanied by a thready pulse, generally signals inadequate cardiac output from reduced intravascular volume. Notify the practitioner and increase the infusion rate.

## PATHOPHYSIOLOGY

### WHAT HAPPENS IN HYPOVOLEMIC SHOCK

In hypovolemic shock, vascular fluid volume loss causes extreme tissue hypoperfusion. Internal fluid losses can result from hemorrhage or third space fluid shifting. External fluid loss can result from severe bleeding or from severe diarrhea, diuresis, or vomiting. Inadequate vascular volume leads to decreased venous return and cardiac output. The resulting drop in arterial blood pressure activates the body's compensatory mechanisms in an attempt to increase vascular volume. If compensation is unsuccessful, decompensation and death may occur.

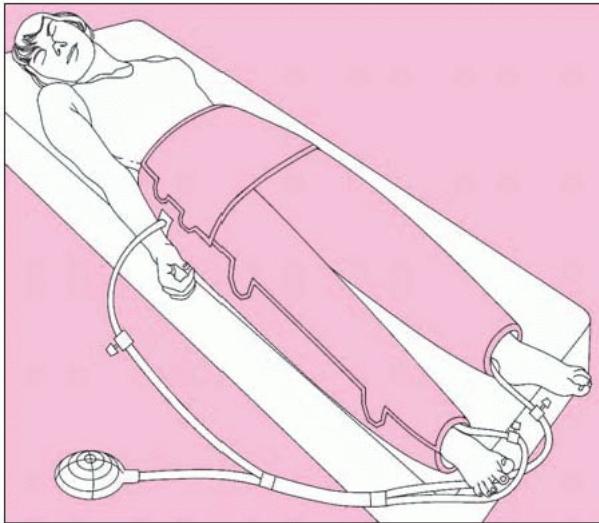


- Start I.V. lines with normal saline or lactated Ringer's solution, using a large-bore catheter (14 G), which allows easier administration of later blood transfusions. (*Caution:* Don't start I.V. lines in the legs of a patient in shock who has suffered abdominal trauma, because infused fluid may escape through the ruptured vessel into the abdomen.)
- An indwelling urinary catheter may be inserted to measure hourly urine output. If output is less than 30 ml/hour in adults, increase the fluid infusion rate, but watch for signs of fluid overload such as an increase in pulmonary artery wedge pressure (PAWP). Notify the practitioner if urine output doesn't improve. An osmotic diuretic such as mannitol may be ordered to increase renal blood flow and urine output.

Determine how much fluid to give by checking blood pressure, urine output, central venous pressure (CVP), or PAWP. (To increase accuracy, CVP should be measured at the level of the right atrium, using the same reference point on the chest each time.)

## **USING A PNEUMATIC ANTISHOCK GARMENT**

A pneumatic antishock garment counteracts bleeding and hypovolemia by slowing or stopping arterial bleeding; by forcing any available blood from the lower body to the heart, brain, and other vital organs; and by preventing return of the available circulating blood volume to the legs.



### **Do**

- While the patient is wearing a pneumatic antishock garment, monitor blood pressure, apical and radial pulse rates, and respirations; check extremities for pedal pulses, color, warmth, and numbness; and make sure the garment isn't too constricting.
- Remove the garment only when a physician is present, fluids are available for transfusion, and anesthesia and surgical teams are available. The compartments are deflated slowly and only one section at a time while the patient is monitored closely.
- To clean, wash with warm soap and water, air-dry, and store.

### **Don't**

- Don't apply the garment if positions or wounds show or suggest major intrathoracic or intracranial vascular injury or if the patient has open extremity bleeding, pulmonary edema, or trauma above the application site. It may be used during pregnancy; however, the abdominal compartment should *not* be inflated.
- When cleaning, don't autoclave or use solvents.

- Draw an arterial blood sample to measure blood gas levels. Administer oxygen by face mask or airway to ensure adequate oxygenation of tissues. Adjust the oxygen flow rate to a higher or lower level, as blood gas measurements indicate.
- Draw venous blood for complete blood count and electrolyte, type and crossmatch, and coagulation studies.
- During therapy, assess skin color and temperature, and note changes. Cold, clammy skin may be a sign of continuing peripheral vascular constriction, indicating progressive shock.
- Watch for signs of impending coagulopathy (petechiae, bruising, and bleeding)

or oozing from gums or venipuncture sites).

- Explain procedures and their purpose. Throughout these emergency measures, provide emotional support to the patient and his family.

## PREVENTION

- *Recognize patients with conditions that reduce blood volume as at-risk patients.*
- *Estimate fluid loss and replace, as necessary, to prevent hypovolemic shock.*

## **Cardiogenic shock**

Sometimes called *pump failure*, cardiogenic shock is a condition of diminished cardiac output that severely impairs tissue perfusion. It reflects severe left-sided heart failure and occurs as a serious complication in 5% to 10% of all patients hospitalized with acute myocardial infarction (MI).

Historically, mortality for cardiogenic shock had been 80% to 90%, but recent studies indicate that the rate has dropped to 56% to 67% due to the advent of thrombolytics, improved interventional procedures, and better therapies. Mortality is expected to decline even further.

## **Causes and incidence**

Cardiogenic shock can result from any condition that causes significant left ventricular dysfunction with reduced cardiac output, such as MI (most common), myocardial ischemia, papillary muscle dysfunction, or end-stage cardiomyopathy. Regardless of the underlying cause, left ventricular dysfunction sets into motion a series of compensatory mechanisms that attempt to increase cardiac output and, in turn, maintain vital organ function. (See *What happens in cardiogenic shock*.) As cardiac output falls in left ventricular dysfunction, aortic and carotid baroreceptors initiate sympathetic nervous responses, which increase heart rate, left ventricular filling pressure, and peripheral resistance to flow, to enhance venous return to the heart. These compensatory responses initially stabilize the patient but later cause deterioration with rising oxygen demands of the already compromised myocardium. These events comprise a vicious circle of low cardiac output, sympathetic compensation, myocardial ischemia, and even lower cardiac output.

Incidence of cardiogenic shock is higher in men than in women because of their higher incidence of coronary artery disease. However, among people with MI, women have a higher incidence of cardiogenic shock than men.

## **Signs and symptoms**

Cardiogenic shock produces signs of poor tissue perfusion: cold, pale, clammy skin; a decrease in systolic blood pressure to 30 mm Hg below baseline, or a sustained reading below 80 mm Hg not attributable to medication; tachycardia; rapid, shallow respirations; oliguria (less than 20 ml/ hour); restlessness; mental confusion and obtundation; narrowing pulse pressure; and cyanosis. Although many of these clinical features also occur in heart failure and other shock syndromes, they're usually more profound in cardiogenic shock.

## **Diagnosis**

Auscultation may detect gallop rhythm, faint heart sounds and, possibly, if the shock results from rupture of the ventricular septum or papillary muscles, a holosystolic murmur.

- Pulmonary artery pressure (PAP) monitoring may show increased PAP, and increased pulmonary artery wedge pressure (PAWP), reflecting a rise in left ventricular end-diastolic pressure (preload) and increased resistance to left ventricular emptying (afterload) due to ineffective pumping and

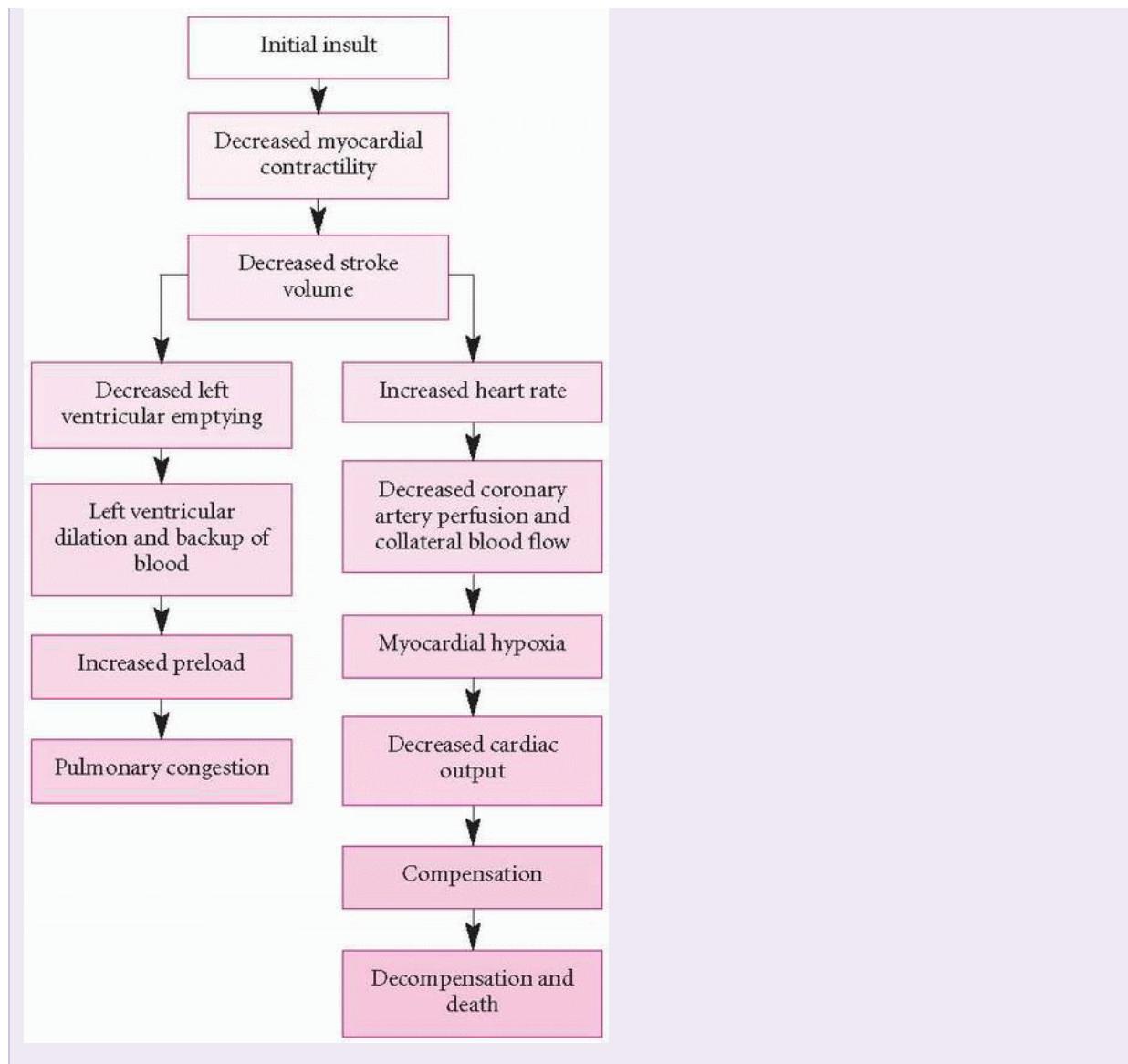
increased peripheral vascular resistance. Thermodilution technique measures decreased cardiac output.

- Invasive arterial pressure monitoring may indicate hypotension due to impaired ventricular ejection.
  - Arterial blood gas (ABG) analysis may show metabolic acidosis and hypoxia.
  - Electrocardiography may show possible evidence of acute MI, ischemia, or ventricular aneurysm.
  - Echocardiography can determine left ventricular function and reveal valvular abnormalities.
- 
- Enzyme levels may show elevated creatine kinase, lactate dehydrogenase, aspartate aminotransferase, and alanine aminotransferase, which point to MI or ischemia and suggest heart failure or shock. Troponin I, troponin T, and isoenzyme values may confirm acute MI.

## PATHOPHYSIOLOGY

### **WHAT HAPPENS IN CARDIOGENIC SHOCK**

When the myocardium can't contract sufficiently to maintain adequate cardiac output, stroke volume decreases and the heart can't eject an adequate volume of blood with each contraction. The blood backs up behind the weakened left ventricle, increasing preload and causing pulmonary congestion. In addition, to compensate for the drop in stroke volume, the heart rate increases in an attempt to maintain cardiac output. As a result of the diminished stroke volume, coronary artery perfusion and collateral blood flow decrease. All of these mechanisms increase the heart's workload and enhance left-sided heart failure. The result is myocardial hypoxia, further decreased cardiac output, and a triggering of compensatory mechanisms to prevent decompensation and death.



Additional tests determine other conditions that can lead to pump dysfunction and failure, such as cardiac arrhythmias, cardiac tamponade, papillary muscle infarct or rupture, ventricular septal rupture, pulmonary emboli, venous pooling (associated with vasodilators and continuous intermittent positive-pressure breathing), and hypovolemia.

## **Treatment**

The aim of treatment is to enhance cardiovascular status by increasing cardiac output,

improving myocardial perfusion, and decreasing cardiac workload with combinations of various cardiovascular drugs and mechanical-assist techniques. Myocardial reperfusion can be accomplished by percutaneous transluminal coronary angioplasty, stents, thrombolytic therapy, or bypass grafting. Drug therapy may include dopamine I.V., a vasoconstrictor that increases cardiac output, blood pressure, and renal blood flow; amrinone or dobutamine I.V., inotropic agents that increase myocardial contractility; norepinephrine, when a more potent vasoconstrictor is necessary; and nitroprusside I.V., a vasodilator that may be used with a vasoconstrictor to further improve cardiac output by decreasing peripheral vascular resistance (afterload) and reducing left ventricular end-diastolic pressure

(preload). However, the patient's blood pressure must be adequate to support nitroprusside therapy and must be monitored closely.

The intra-aortic balloon pump (IABP) is a mechanical-assist device that attempts to improve coronary artery perfusion and decrease cardiac workload. (See *Understanding the IABP*.) The inflatable balloon pump is percutaneously or surgically inserted through the femoral artery into the descending thoracic aorta. The balloon inflates during diastole to increase coronary artery perfusion pressure and deflates before systole (before the aortic valve opens) to reduce resistance to ejection (afterload) and reduce cardiac workload. Improved ventricular ejection, which significantly improves cardiac output, and a subsequent vasodilation in the peripheral vasculature lead to lower preload volume.

When drug therapy and IABP insertion fail, treatment may require the use of a ventricular assist device. This device (which may be either temporary or permanent) diverts systemic blood flow from a diseased ventricle into a centrifugal pump. It assists the heart's pumping action rather than replaces it.

## **Special considerations**

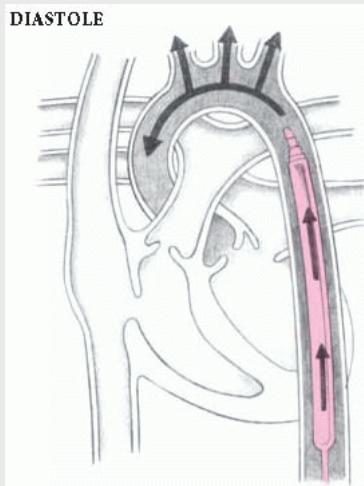
- At the first sign of cardiogenic shock, check the patient's blood pressure and heart rate. If the patient is hypotensive or is having difficulty breathing, ensure a patent I.V. line and a patent airway, and provide oxygen to promote tissue oxygenation. Notify the practitioner immediately.
- Monitor ABG values to measure oxygenation and detect acidosis from poor tissue perfusion. Increase oxygen delivery as indicated. Check complete blood count and electrolyte levels.
- After diagnosis, monitor cardiac rhythm continuously and assess skin color, temperature, and other vital signs often. Watch for a drop in systolic blood pressure to less than 80 mm Hg (usually compromising cardiac output further). Report hypotension immediately.
- An indwelling urinary catheter may be inserted to measure urine output. Notify the practitioner if output drops below 30 ml/ hour.
- Using a pulmonary artery catheter, closely monitor PAP, PAWP and, if equipment is available, cardiac output. A high PAWP indicates heart failure and should be reported.
- When a patient is on the IABP, reposition him often and perform passive range-of-motion exercises to prevent skin breakdown. However, don't flex the patient's "ballooned" leg at the hip because this may displace or fracture the catheter. Assess pedal pulses and skin temperature and color to make sure circulation to the leg is adequate. Check the dressing on the insertion site frequently for bleeding and change it according to facility protocol. Also, check the site for hematoma or signs of infection, and culture any drainage.
- After the patient becomes hemodynamically stable, the frequency of balloon inflation is gradually reduced to wean him from the IABP. During weaning, carefully watch for monitor changes, chest pain, and other signs of recurring cardiac ischemia and shock.
- Provide psychological support and reassurance because the patient and his family may be anxious about the intensive care unit, IABP, and other tubes and devices. To ease emotional stress, plan your care to allow frequent rest periods, and provide as much privacy as possible.

### **UNDERSTANDING THE IABP**

An intra-aortic balloon pump (IABP) consists of a polyurethane balloon attached to an external pump console by means of a large-lumen catheter. It's inserted percutaneously through the femoral artery and positioned in the descending aorta, just distal to the left subclavian artery and above the renal arteries.

**Push...**

This external pump works in precise counterpoint to the left ventricle, inflating the balloon with helium early in diastole and deflating it just before systole. As the balloon inflates, it forces blood toward the aortic valve, thereby raising pressure in the aortic root and augmenting diastolic pressure to improve coronary perfusion. It also improves peripheral circulation by forcing blood through the brachiocephalic, common carotid, and subclavian arteries arising from the aortic trunk.



### ...and pull

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



### PREVENTION

*Emphasize the fact that prevention requires timely, thorough, and aggressive identification and treatment of causative disorders.*

## **Ventricular aneurysm**

A ventricular aneurysm is an outpouching, almost always of the left ventricle, that produces ventricular wall dysfunction in about 20% of patients after myocardial infarction (MI). Ventricular aneurysm may develop within weeks after MI. Untreated ventricular aneurysm can lead to arrhythmias, systemic embolization, or heart failure, and may cause sudden death. Resection improves the prognosis in patients with heart failure or refractory patients who have developed ventricular arrhythmias.

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### **Causes and incidence**

When MI destroys a large muscular section of the left ventricle, necrosis reduces the ventricular wall to a thin sheath of fibrous tissue. Under intracardiac pressure, this thin layer stretches and forms a separate noncontractile sac (aneurysm). Abnormal muscular wall movement accompanies ventricular aneurysm and includes akinesia (lack of movement), dyskinesia (paradoxical movement), asynergia (decreased and inadequate movement), and asynchrony (uncoordinated movement). During systolic ejection, the abnormal muscular wall movements associated with the aneurysm cause the remaining normally functioning myocardial fibers to increase the force of contraction in order to maintain stroke volume and cardiac output. At the same time, a portion of the stroke volume is lost to passive distention of the noncontractile sac.

### **Complications**

- Ventricular arrhythmias
- Cerebral embolism
- Heart failure
- Death

### **Signs and symptoms**

Ventricular aneurysm may cause arrhythmias—such as premature ventricular contractions or ventricular tachycardia—palpitations, signs of cardiac dysfunction (weakness on exertion, fatigue, and angina) and, occasionally, a visible or palpable systolic precordial bulge. This condition may also lead to left ventricular dysfunction, with chronic heart failure (dyspnea, fatigue, edema, crackles, gallop rhythm, and jugular vein distention); pulmonary edema; systemic embolization; and, with left-sided heart failure, pulsus alternans. Ventricular aneurysms enlarge but seldom rupture.

### **Diagnosis**

Persistent ventricular arrhythmias, onset of heart failure, or systemic embolization in a patient with left-sided heart failure and a history of MI strongly suggests ventricular aneurysm. Indicative tests include the following:

- Left ventriculography reveals left ventricular enlargement, with an area of akinesia or dyskinesia (during cineangiography) and diminished cardiac function.
- Electrocardiography may show persistent ST-T wave elevations after infarction.
- Chest X-ray may demonstrate an abnormal bulge distorting the heart's contour if the aneurysm is large; the X-ray may be normal if the aneurysm is small.
- Noninvasive nuclear cardiology scan may indicate the site of infarction and suggest the area of aneurysm.
- Echocardiography shows abnormal motion in the left ventricular wall.

## ***Treatment***

Depending on the aneurysm's size and the complications, treatment may necessitate only routine medical examination to follow the patient's condition or aggressive measures for intractable ventricular arrhythmias, heart failure, and emboli.

Emergency treatment of ventricular arrhythmia includes antiarrhythmics I.V. or cardioversion. Preventive treatment continues with oral antiarrhythmics, such as procainamide, quinidine, or disopyramide.

Emergency treatment for heart failure with pulmonary edema includes oxygen, cardiac glycosides I.V., furosemide I.V., morphine I.V. and, when necessary, nitroprusside I.V. and intubation.

Maintenance therapy may include nitrates, prazosin, and oral hydralazine. Systemic embolization requires anticoagulation therapy or embolectomy. Refractory ventricular tachycardia, heart failure, recurrent arterial embolization, and persistent angina with coronary artery occlusion may necessitate surgery, of which the most effective procedure is aneurysmectomy with myocardial revascularization.

## ***Special considerations***

- If ventricular tachycardia occurs, administer a prescribed antiarrhythmic such as lidocaine. Monitor blood pressure and heart rate. If cardiac arrest develops, initiate cardiopulmonary resuscitation (CPR) and call for assistance, resuscitative equipment, and medication.
- In a patient with heart failure, closely monitor vital signs, heart sounds, intake

and output, fluid and electrolyte balances, and blood urea nitrogen and creatinine levels. Because of the threat of systemic embolization, frequently check peripheral pulses and the color and temperature of extremities. Be alert for sudden changes in sensorium that indicate cerebral embolization and for any signs that suggest renal failure or progressive MI.

- If arrhythmias necessitate cardioversion, use a sufficient amount of conducting jelly to prevent chest burns. If the patient is conscious, give diazepam or methohexitol I.V., as ordered, before cardioversion. Explain that cardioversion is a lifesaving method using brief electroshock to the heart. If the patient is receiving antiarrhythmics, check appropriate laboratory tests. For instance, if the patient takes procainamide, check antinuclear antibodies because this drug may induce symptoms that mimic lupus erythematosus.

If the patient is scheduled to undergo resection:

- Before surgery, explain expected postoperative care in the intensive care unit (including use of such things as endotracheal tube, ventilator, hemodynamic monitoring, chest tubes, and drainage bottle).
- After surgery, monitor vital signs, intake and output, heart sounds, and pulmonary artery catheter. Watch for signs of infection, such as fever and drainage.

To prepare the patient for discharge:

- Teach him how to check for pulse irregularity and rate changes. Encourage him to follow his prescribed medication regimen—even during the night—and to watch for adverse effects.
- Because arrhythmias can cause sudden death, refer the family to a community-based CPR training program.
- Provide psychological support for the patient and his family.

## ***Cardiac tamponade***

In cardiac tamponade, a rapid, unchecked rise in intrapericardial pressure impairs diastolic filling of the heart. The rise in pressure usually results from blood or fluid accumulation in the pericardial sac. If fluid accumulates rapidly, this condition requires emergency lifesaving measures to prevent death. A slow accumulation and rise in pressure, as in pericardial effusion associated with malignant tumors, may not produce immediate symptoms, because the fibrous wall of the pericardial sac can gradually stretch to accommodate as much as 1 to 2 L of fluid.

## ***Causes and incidence***

Increased intrapericardial pressure and cardiac tamponade may be idiopathic (Dressler's syndrome) or may result from:

- effusion (in cancer, bacterial infections, tuberculosis and, rarely, acute rheumatic fever)
- hemorrhage from trauma (such as gunshot or stab wounds of the chest and perforation by catheter during cardiac or central venous catheterization or postcardiac surgery)
- hemorrhage from nontraumatic causes (such as rupture of the heart or great vessels or anticoagulant therapy in a patient with pericarditis)
- acute myocardial infarction (MI)
- end stage lung cancer
- heart tumors
- radiation therapy
- hypothyroidism
- systemic lupus erythematosus
- uremia.

Cardiac tamponade occurs in 2 of every 10,000 people.

## ***Complications***

- Cardiogenic shock
- Death

## ***Signs and symptoms***

Cardiac tamponade classically produces increased venous pressure with jugular vein distention, reduced arterial blood pressure, muffled heart sounds on auscultation, and pulsus paradoxus (an abnormal inspiratory drop in systemic blood pressure greater than 15 mm Hg). The absence of a preexisting pericardial friction rub may suggest an increase in fluid in the pericardial space. These classic symptoms represent failure of physiologic compensatory mechanisms to override the effects of rapidly rising pericardial pressure, which limits diastolic filling of the ventricles and reduces

stroke volume to a critically low level. Generally, ventricular end-systolic volume may drop because of inadequate preload. The increasing pericardial pressure is transmitted equally across the heart cavities, producing a matching rise in intracardiac pressure, especially atrial and end-diastolic ventricular pressures. Cardiac tamponade may also cause dyspnea, diaphoresis, pallor or cyanosis, anxiety, tachycardia, narrow pulse pressure, restlessness, and hepatomegaly, but the lung fields will be clear. The patient typically sits upright and leans forward.

## ***Diagnosis***

- Chest X-ray shows slightly widened mediastinum and cardiomegaly.

- Electrocardiography (ECG) is rarely diagnostic of tamponade but is useful in ruling out other cardiac disorders. It may reveal changes produced by acute pericarditis.
- Pulmonary artery catheterization detects increased right atrial pressure, right ventricular diastolic pressure, and central venous pressure (CVP).
- Echocardiography, computed tomography scan, or magnetic resonance imaging shows pericardial effusion with signs of right ventricular and atrial compression.

## ***Treatment***

The goal of treatment is to relieve intrapericardial pressure and cardiac compression by removing accumulated blood or fluid. Pericardiocentesis (needle aspiration of the pericardial cavity) or surgical creation of an opening (pericardiectomy or pericardial window) dramatically improves systemic arterial pressure and cardiac output with aspiration of as little as 25 ml of fluid. Such treatment necessitates continuous hemodynamic and ECG monitoring in the intensive care unit. Trial volume loading with temporary I.V. normal saline solution with albumin, and perhaps an inotropic drug, such as isoproterenol or dopamine, is necessary in the hypotensive patient to maintain cardiac output. Although these drugs normally improve myocardial function, they may further compromise an ischemic myocardium after MI.

Depending on the cause of tamponade, additional treatment may include:

- in traumatic injury—blood transfusion or a thoracotomy to drain reaccumulating fluid or to repair bleeding sites
- in heparin-induced tamponade—the heparin antagonist protamine sulfate
- in warfarin-induced tamponade—vitamin K.

Resection of a portion or all of the pericardium to allow full communication with the pleura may be needed if repeated pericardiocentesis fails to prevent recurrence.

## ***Special considerations***

If the patient needs pericardiocentesis:

- Explain the procedure to him. Keep a pericardial aspiration needle attached to a 50-ml syringe by a three-way stopcock, an ECG machine, and an emergency cart with a defibrillator at the bedside. Make sure the equipment is turned on and ready for immediate use. Position him at a 45- to 60-degree angle. Connect the precordial ECG lead to the hub of the aspiration needle with an alligator clamp and connecting wire, and assist with fluid aspiration. When the needle touches the myocardium, you'll see an ST-segment elevation or premature ventricular contractions.
- Monitor blood pressure and CVP during and after pericardiocentesis. Infuse I.V. solutions, as prescribed, to maintain blood pressure. Watch for a decrease in CVP and a concomitant rise in blood pressure, which indicate relief of cardiac compression.
- Watch for complications of pericardiocentesis, such as ventricular fibrillation, vasovagal response, or coronary artery or cardiac chamber puncture. Closely monitor ECG changes, blood pressure, pulse rate, level of consciousness, and urine output.

If the patient needs thoracotomy:

- Explain the procedure to him. Tell him what to expect postoperatively (chest tubes, drainage bottles, and oxygen administration). Teach him how to turn, deep breathe, and cough.
- Give antibiotics, protamine sulfate, or vitamin K, as ordered.
- Postoperatively, monitor critical parameters, such as vital signs and arterial

blood gas values, and assess heart and breath sounds. Give pain medication as ordered. Maintain the chest drainage system and be alert for complications, such as hemorrhage and arrhythmias.

## **PREVENTION**

*Instruct patient to practice heart-healthy living, with a heart-healthy diet, stress reduction, regular exercise, and preventive care, maintaining a healthy weight, smoking cessation, and abstinence from alcohol.*

## **Cardiac arrhythmias**

In cardiac arrhythmias (sometimes called *cardiac dysrhythmias*), abnormal electrical conduction or automaticity changes heart rate and rhythm. (See *Normal cardiac conduction*, page 72.) Arrhythmias vary in severity, from those that are mild, asymptomatic, and require no treatment (such as sinus arrhythmia, in which heart rate increases and decreases with respiration) to catastrophic ventricular fibrillation, which necessitates 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 origin site, determines their clinical significance. (See *Types of cardiac arrhythmias*, pages 73 to 80.)

## **Causes and incidence**

Arrhythmias may be congenital or they may result from one of several factors, including myocardial ischemia, myocardial infarction, or organic heart disease. Drug ingestion (cocaine, amphetamines, caffeine, beta-blockers, psychotropics, sympathomimetics), drug toxicity, or degeneration of the conductive tissue necessary to maintain normal heart rhythm (sick sinus syndrome) can sometimes precipitate arrhythmias. People with imbalances of blood chemistries or those with a history of cardiac conditions (coronary artery disease or heart valve disorders) are at higher risk for developing arrhythmias.

## **Complications**

- Impaired cardiac output

## **Signs and symptoms**

Signs and symptoms of cardiac arrhythmias include palpitations, fainting, lightheadedness, dizziness, chest pain, shortness of breath, changes in pulse patterns, paleness, and the temporary absence of breathing. However, the patient with a cardiac arrhythmia may be asymptomatic until the development of sudden cardiac arrest.

## **Diagnosis**

Diagnosis is made by tests that reveal the arrhythmia, such as 12-lead electrocardiography. Ambulatory cardiac monitoring (Holter monitoring), echocardiography, electrophysiology studies, and coronary angiography may also confirm or rule out suspected causes of arrhythmias and help determine treatment.

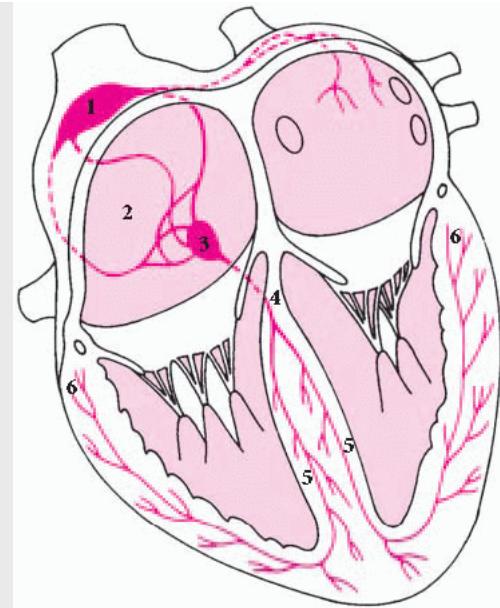
## **Special considerations**

- Assess an unmonitored patient for rhythm disturbances.
- If the patient's pulse is abnormally rapid, slow, or irregular, watch for signs of hypoperfusion, such as altered level of consciousness (LOC), hypotension, and diminished urine output.
- Document arrhythmias in a monitored patient, and assess for possible causes and effects.

- When life-threatening arrhythmias develop, rapidly assess LOC, respirations, and pulse rate.
  - Initiate cardiopulmonary resuscitation if indicated.
  - Evaluate the patient for altered cardiac output resulting from arrhythmias.
- 
- Administer medications as ordered and prepare to assist with medical procedures, if indicated (for example, cardioversion).
  - Monitor patient for predisposing factors —such as fluid and electrolyte imbalance—and signs of drug toxicity, especially with digoxin. If you suspect drug toxicity, report such signs to the practitioner immediately and withhold the next dose.
  - To prevent arrhythmias in a postoperative cardiac patient, provide adequate oxygen and reduce the heart's workload while carefully maintaining metabolic, neurologic, respiratory, and hemodynamic status.
  - Consider sedation for transcutaneous pacing if appropriate.
  - To avoid temporary pacemaker malfunction, install a fresh battery before each insertion. Carefully secure the external catheter wires and the pacemaker box. Assess the threshold daily. Watch closely for premature contractions, a sign of myocardial irritation.
  - To avert permanent pacemaker malfunction, restrict the patient's activity after insertion as ordered. Monitor the pulse rate regularly and watch for signs of decreased cardiac output.
  - If the patient has a permanent pacemaker, warn him about environmental hazards, as indicated by the pacemaker manufacturer. Although hazards may not present a problem, in doubtful situations, 24-hour Holter monitoring may be helpful. Tell the patient to report light-headedness or syncope and stress the importance of regular checkups.
  - Compare the patient's cardiac status (pulse, blood pressure, and cardiac output) with the cardiac rhythm before and after treatments.

### **NORMAL CARDIAC CONDUCTION**

Each electrical impulse travels from the sinoatrial node (1) through the intra-atrial tracts (2), producing atrial contraction. The impulse slows momentarily as it passes through the atrioventricular junction (3) to the bundle of His (4). Then, it descends the left and right bundle branches (5) and reaches the Purkinje fibers (6), stimulating ventricular contraction.



## PREVENTION

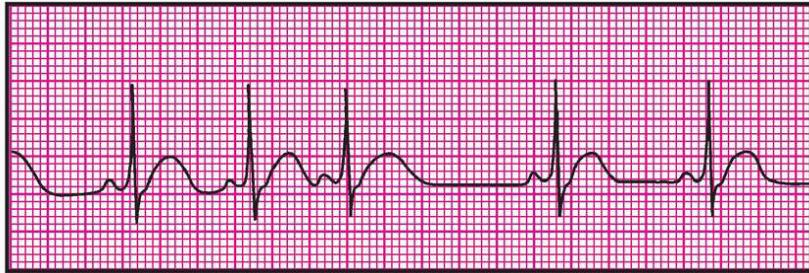
- Maintain adequate oxygenation.
- Maintain normal fluid, acid-base, and electrolyte balance (especially potassium, magnesium, and calcium).
- Maintain normal drug levels.

## TYPES OF CARDIAC ARRHYTHMIAS

This chart reviews many common cardiac arrhythmias and outlines their features, causes, and treatments. Use a normal electrocardiogram strip, if available, to compare normal cardiac rhythm configurations with the rhythm strips below. Characteristics of normal rhythm include:

- ventricular and atrial rates of 60 to 100 beats/minute
- regular and uniform QRS complexes and P waves
- PR interval of 0.12 to 0.2 second
- QRS duration <0.12 second
- identical atrial and ventricular rates, with constant PR interval.

Arrhythmia and features	Causes	Treatment
<i>Sinus arrhythmia</i>	• A normal sinus rhythm with a prolonged PR interval.	• Atropine if rate is slow.



- Irregular atrial and ventricular rhythms
- Normal P wave preceding each QRS complex

variation of normal sinus rhythm in athletes, children, and elderly people  
 • Also seen in digoxin toxicity and inferior wall myocardial infarction (MI)

decreases below 40 beats/ minute and patient is symptomatic (for example, has hypotension)

### *Sinus tachycardia*

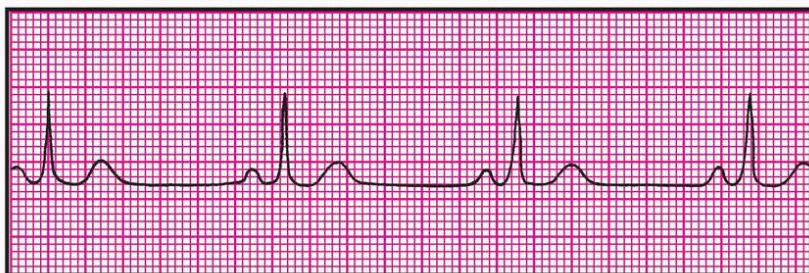


- Atrial and ventricular rhythms regular
- Rate > 100 beats/minute; rarely, >160 beats/minute
- Normal P wave preceding each QRS complex

• Normal physiologic response to fever, exercise, anxiety, pain, dehydration; may also accompany shock, left-sided heart failure, cardiac tamponade, hyperthyroidism, anemia, hypovolemia, pulmonary embolism, anterior wall MI  
 • May also occur with atropine, epinephrine, isoproterenol, quinidine, caffeine, alcohol, and nicotine use

• Correction of underlying cause  
 • Beta-adrenergic blockers or calcium channel blockers for symptomatic patients

### *Sinus bradycardia*



- Regular atrial and ventricular rhythms
- Rate < 60 beats/minute
- Normal P wave preceding each QRS complex

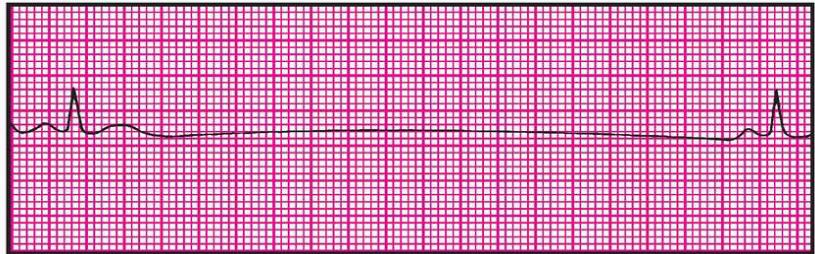
• Normal in well-conditioned heart, as in an athlete  
 • Increased intracranial pressure; increased vagal tone due to straining during defecation, vomiting, intubation, mechanical ventilation; sick sinus syndrome;

• For low cardiac output, dizziness, weakness, altered level of consciousness, or low blood pressure: follow advanced cardiac life support (ACLS) protocol for administration of atropine  
 • Temporary pacemaker; may

hypothyroidism;  
inferior wall MI  
▪ May also occur  
with  
anticholinesterase,  
beta blocker,  
digoxin, and  
morphine use

need to be  
evaluated for  
permanent  
pacemaker at a  
later time

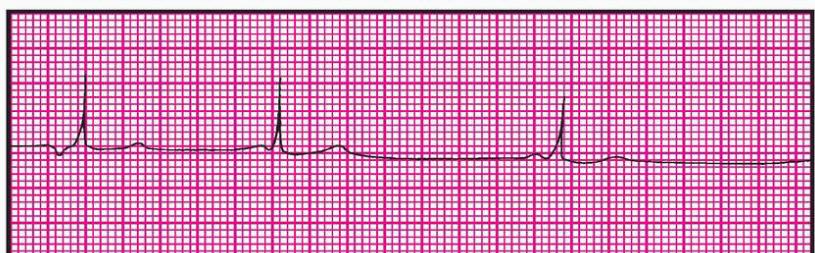
#### **Sinoatrial (SA) arrest or block (sinus arrest)**



- Atrial and ventricular rhythms normal except for missing complex
- Normal P wave preceding each QRS complex
- Pause not equal to a multiple of the previous sinus rhythm

- Acute infection
- Coronary artery disease,  
degenerative heart disease, acute  
inferior wall MI
- Vagal stimulation,  
Valsalva's maneuver, carotid sinus massage
- Digoxin, quinidine, or salicylate toxicity
- Pesticide poisoning
- Pharyngeal irritation caused by endotracheal (ET) intubation
- Sick sinus syndrome
- Treat symptoms with atropine I.V.
- Temporary pacemaker; consider permanent pacemaker for repeated episodes

#### **Wandering atrial pacemaker**

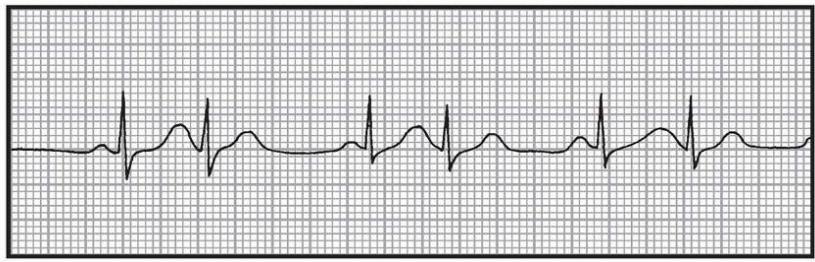


- Atrial and ventricular rhythms vary slightly
- Irregular PR interval
- P waves irregular with changing configuration, indicating that they aren't all from SA node or single atrial focus; may appear after the QRS complex
- QRS complexes uniform in shape but irregular in rhythm

- Rheumatic carditis due to inflammation involving the SA node
- Digoxin toxicity
- Sick sinus syndrome
- No treatment if patient is asymptomatic
- Treatment of underlying cause if patient is symptomatic

#### **Premature atrial contraction (PAC)**

- Coronary or valvular heart disease, atrial ischemia, coronary
- Usually no treatment is needed



- Premature, abnormal-looking P waves that differ in configuration from normal P waves
- QRS complexes after P waves, except in very early or blocked PACs
- P wave often buried in the preceding T wave or identified in the preceding T wave

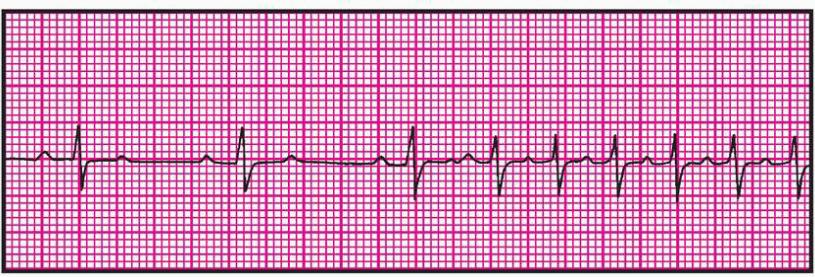
atherosclerosis, heart failure, acute respiratory failure, chronic obstructive pulmonary disease (COPD), electrolyte imbalance, and hypoxia

- Treatment of underlying cause if patient is symptomatic

Digoxin toxicity; use of aminophylline, adrenergics, or caffeine

- Anxiety

#### *Paroxysmal supraventricular tachycardia*



- Atrial and ventricular rhythms regular
- Heart rate > 160 beats/minute; rarely exceeds 250 beats/minute
- P waves regular but aberrant; difficult to differentiate from preceding T wave
- P wave preceding each QRS complex
- Sudden onset and termination of arrhythmia
- When a normal P wave is present, it's called *paroxysmal atrial tachycardia*; when a normal P wave isn't present, it's called *paroxysmal junctional tachycardia*

Intrinsic abnormality of atrioventricular (AV) conduction system

- Physical or psychological stress, hypoxia, hypokalemia, cardiomyopathy, congenital heart disease, MI, valvular disease, Wolff-Parkinson-White syndrome, cor pulmonale, hyperthyroidism, systemic hypertension
- Digoxin toxicity; use of caffeine, marijuana, or central nervous system stimulants

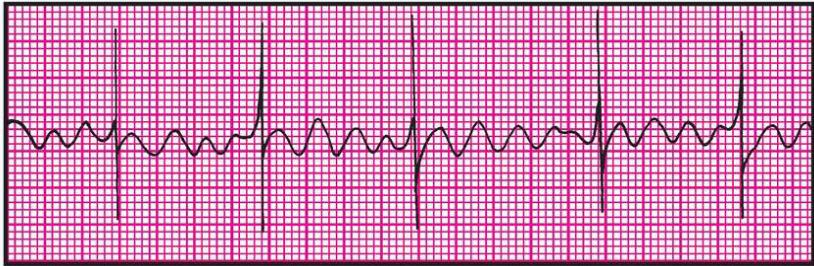
If patient is unstable, prepare for immediate cardioversion

- If patient is stable, vagal stimulation, Valsalva's maneuver, carotid sinus massage
- Adenosine by rapid I.V. bolus injection to rapidly convert arrhythmia
- If patient has a normal ejection fraction, consider calcium channel blockers, beta-adrenergic blockers, or amiodarone
- If patient has an ejection fraction less than 40%, consider amiodarone

#### *Atrial flutter*

- Heart failure, tricuspid or mitral valve disease,

If patient is unstable with a ventricular rate



- Atrial rhythm regular; rate, 250 to 400 beats/minute
- Ventricular rate variable, depending on degree of AV block (usually 60 to 100 beats/minute)
- Sawtooth P wave configuration possible (F waves)
- QRS complexes uniform in shape but often irregular in rate

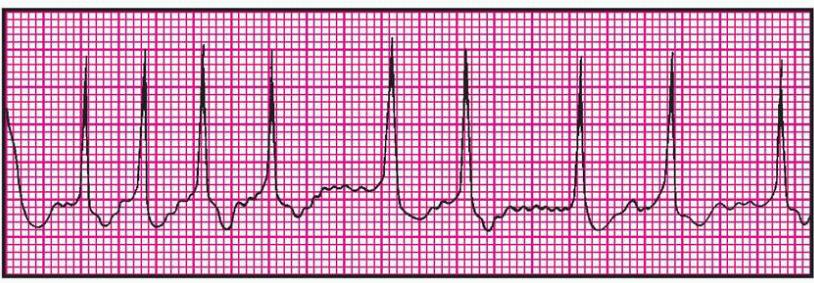
pulmonary embolism, cor pulmonale, inferior wall MI, carditis

• Digoxin toxicity

> 150 beats/minute, prepare for immediate cardioversion

- If patient is stable, drug therapy may include calcium channel blockers, beta-adrenergic blockers, or antiarrhythmics
- Anticoagulation therapy may be necessary

#### *Atrial fibrillation*



- Atrial rhythm grossly irregular; rate > 400 beats/minute
- Ventricular rhythm grossly irregular
- QRS complexes of uniform configuration and duration
- PR interval indiscernible
- No P waves, or P waves that appear as erratic, irregular, baseline fibrillatory waves

Heart failure, COPD, thyrotoxicosis, constrictive pericarditis, ischemic heart disease, sepsis, pulmonary embolus, rheumatic heart disease, hypertension, mitral stenosis, atrial irritation, complication of coronary bypass or valve replacement surgery

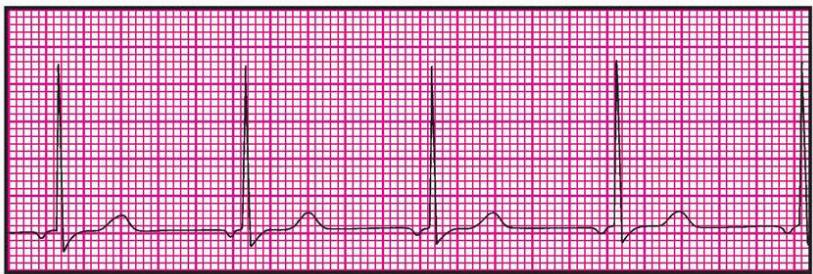
If patient is unstable with a ventricular rate > 150 beats/minute, prepare for immediate cardioversion

- If patient is stable, drug therapy may include calcium channel blockers, beta-adrenergic blockers, digoxin, procainamide, quinidine, ibutilide, or amiodarone
- Consider anticoagulation to prevent emboli
- Dual chamber atrial pacing, implantable atrial pacemaker, or surgical maze procedure may also be used

#### *Junctional rhythm*

Inferior wall MI

Correction of



- Atrial and ventricular rhythms regular
- Atrial rate 40 to 60 beats/ minute
- Ventricular rate usually 40 to 60 beats/minute (60 to 100 beats/ minute is accelerated junctional rhythm)
- P waves preceding, hidden within (absent), or after QRS complex; usually inverted if visible
- PR interval (when present) < 0.12 second
- QRS complex configuration and duration normal, except in aberrant conduction

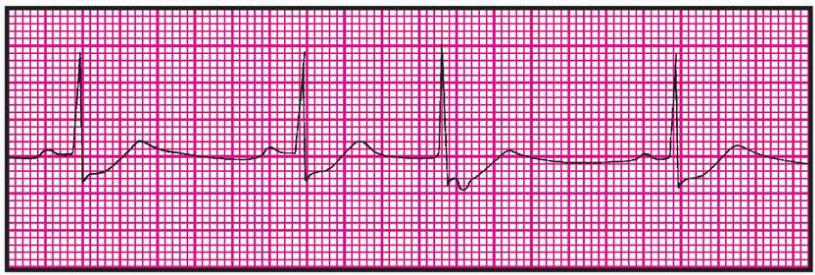
or ischemia,  
hypoxia, vagal  
stimulation, sick  
sinus syndrome

- Acute rheumatic fever
- Valve surgery
- Digoxin toxicity

underlying cause

- Atropine for symptomatic slow rate
- Pacemaker insertion if patient is refractory to drugs
- Discontinuation of digoxin if appropriate

#### *Premature junctional contractions*

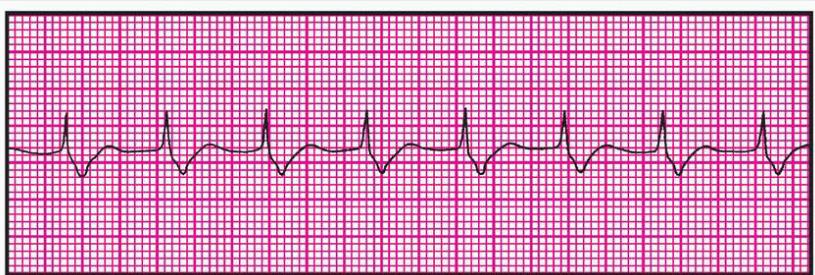


- Atrial and ventricular rhythms irregular
- P waves inverted; may precede, be hidden within, or follow QRS complex
- PR interval < 0.12 second if P wave precedes QRS complex
- QRS complex configuration and duration normal

- MI or ischemia
- Digoxin toxicity and excessive caffeine or amphetamine use

- Correction of underlying cause
- Discontinuation of digoxin if appropriate

#### *Junctional tachycardia*

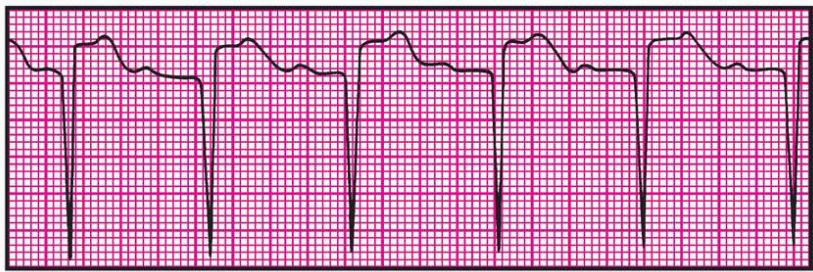


- Atrial rate > 100 beats/minute; however, P wave may be absent, hidden in QRS complex, or preceding T wave
- Ventricular rate > 100 beats/ minute
- P wave inverted
- QRS complex configuration and duration normal
- Onset of rhythm often sudden, occurring in bursts

- Myocarditis, cardiomyopathy, inferior wall MI or ischemia, acute rheumatic fever, complication of valve replacement surgery
- Digoxin toxicity

- Cardioversion if ventricular rate is > 150 or if patient is symptomatic
- Amiodarone, beta-adrenergic blockers, or calcium channel blockers if patient is stable
- Discontinuation of digoxin if appropriate

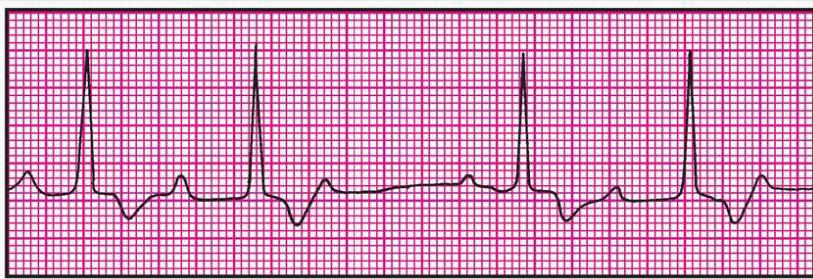
#### *First-degree AV block*



- Atrial and ventricular rhythms regular
- PR interval > 0.20 second
- P wave preceding each QRS complex
- QRS complex normal

- Inferior wall MI or ischemia or infarction, hypothyroidism, hypokalemia, hyperkalemia
- Digoxin toxicity; use of quinidine, procainamide, beta-adrenergic blockers, calcium channel blockers, or amiodarone
- Correction of underlying cause
- Possibly atropine if PR interval exceeds 0.26 second or symptomatic bradycardia develops
- Cautious use of digoxin, calcium channel blockers, and beta-adrenergic blockers

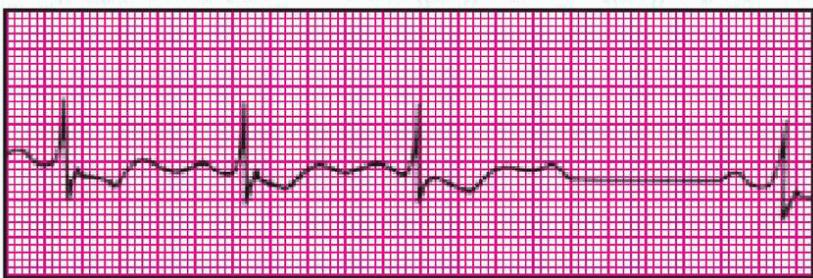
#### **Second-degree AV block Mobitz I (Wenckebach)**



- Atrial rhythm regular
- Ventricular rhythm irregular
- Atrial rate exceeds ventricular rate
- PR interval progressively, but only slightly, longer with each cycle until QRS complex disappears (dropped beat); PR interval shorter after dropped beat

- Inferior wall MI, cardiac surgery, acute rheumatic fever, and vagal stimulation
- Digoxin toxicity; use of propranolol, quinidine, or procainamide
- Treatment of underlying cause
- Atropine or temporary pacemaker for symptomatic bradycardia
- Discontinuation of digoxin if appropriate

#### **Second-degree AV block Mobitz II**

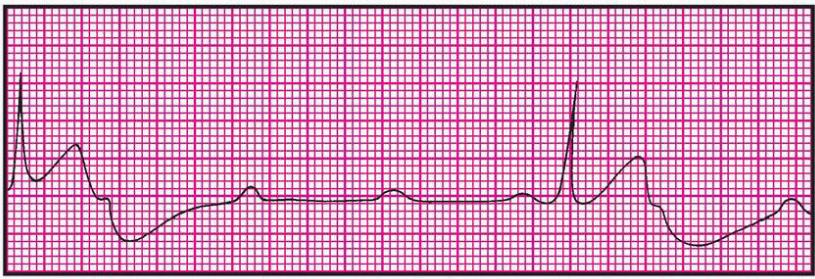


- Atrial rhythm regular
- Ventricular rhythm regular or irregular, with varying degree of block
- P-P interval constant
- QRS complexes periodically absent

- Severe coronary artery disease, anterior wall MI, acute myocarditis
- Digoxin toxicity
- Atropine, epinephrine, and dopamine for symptomatic bradycardia
- Temporary or permanent pacemaker for symptomatic bradycardia
- Discontinuation of digoxin if appropriate

#### **Third-degree AV block (complete heart block)**

- Inferior or anterior wall MI, congenital abnormality,
- Atropine, epinephrine, and dopamine for

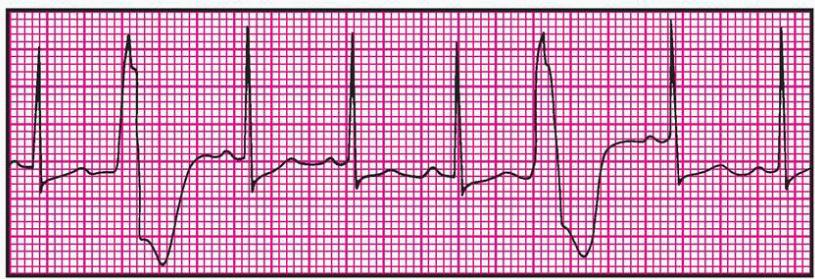


- Atrial rhythm regular
- Ventricular rhythm regular and rate slower than atrial rate
- No relation between P waves and QRS complexes
- No constant PR interval
- QRS interval normal (nodal pacemaker) or wide and bizarre (ventricular pacemaker)

rheumatic fever, hypoxia, postoperative complication of mitral valve replacement, Lev's disease (fibrosis and calcification that spreads from cardiac structures to the conductive tissue), Lenegre's disease (conductive tissue fibrosis)  
 ▪ Digoxin toxicity

symptomatic bradycardia  
 ▪ Temporary or permanent pacemaker for symptomatic bradycardia

#### **Premature ventricular contraction (PVC)**



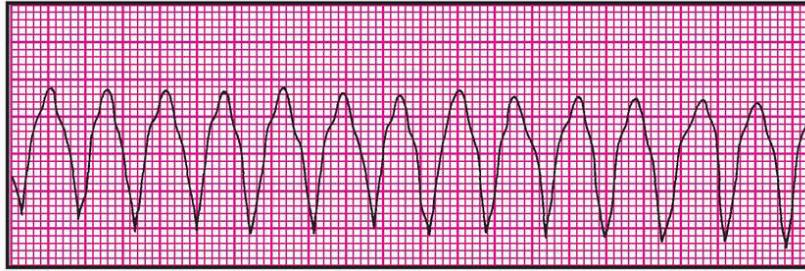
- Atrial rhythm regular
- Ventricular rhythm irregular
- QRS complex premature, usually followed by a complete compensatory pause
- QRS complex wide and distorted, usually > 0.14 second
- Premature QRS complexes occurring singly, in pairs, or in threes; alternating with normal beats; focus from one or more sites
- Ominous when clustered, multifocal, with R wave on T pattern

▪ Heart failure; old or acute myocardial ischemia, infarction, or contusion; myocardial irritation by ventricular catheter such as a pacemaker; hypercapnia; hypokalemia, hypocalemia  
 ▪ Drug toxicity (cardiac glycosides, aminophylline, tricyclic antidepressants, beta-adrenergics [bisoprolol or dopamine])  
 ▪ Caffeine, tobacco, or alcohol use  
 ▪ Psychological stress, anxiety, pain, exercise

▪ If warranted, procainamide, lidocaine, or amiodarone I.V.  
 ▪ Treatment of underlying cause  
 ▪ Discontinuation of drug causing toxicity  
 ▪ Potassium chloride I.V. if PVC induced by hypokalemia  
 ▪ Magnesium sulfate I.V. if PVC induced by hypomagnesemia

#### **Ventricular tachycardia (VT)**

- Myocardial ischemia, infarction, or aneurysm; coronary artery disease; rheumatic
- Pulseless: Initiate cardiopulmonary resuscitation (CPR); follow ACLS protocol



- Ventricular rate 140 to 220 beats/minute, regular or irregular
- QRS complexes wide, bizarre, and independent of P waves
- P waves not discernible
- May start and stop suddenly

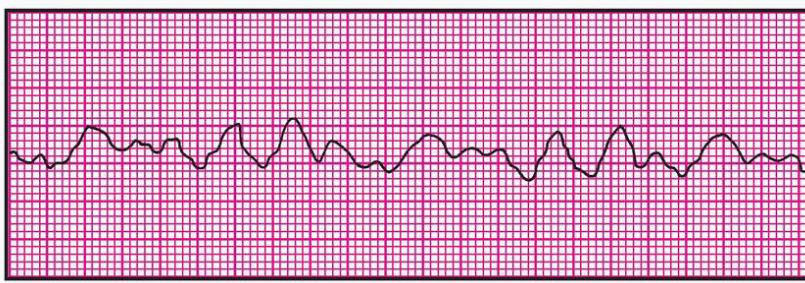
heart disease; mitral valve prolapse; heart failure; cardiomyopathy; ventricular catheters; hypokalemia; hypercalcemia; pulmonary embolism

- Digoxin, procainamide, epinephrine, or quinidine toxicity
- Anxiety

for defibrillation, ET intubation, and administration of epinephrine or vasopressin, followed by amiodarone or lidocaine; if ineffective, consider magnesium sulfate

- With pulse: If hemodynamically stable, follow ACLS protocol for administration of amiodarone; if ineffective, initiate synchronized cardioversion
- If polymorphic VT, consult an expert in arrhythmia management

#### *Ventricular fibrillation*



- Ventricular rhythm and rate rapid and chaotic
- QRS complexes wide and irregular; no visible P waves

• Myocardial ischemia or infarction, R-on-T phenomenon, untreated ventricular tachycardia, hypokalemia, hyperkalemia, hypercalcemia, alkalosis, electric shock, hypothermia

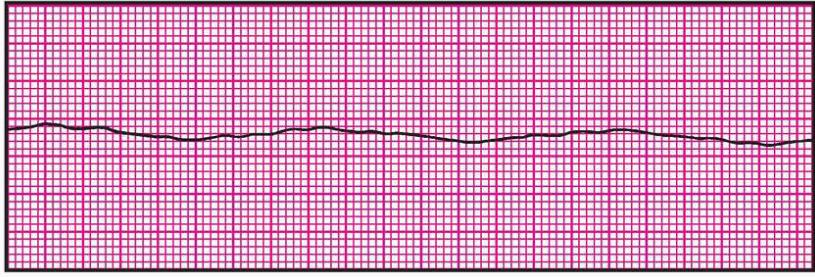
- Digoxin, epinephrine, or quinidine toxicity

• Pulseless: Start CPR; follow ACLS protocol for defibrillation, ET intubation, and administration of epinephrine or vasopressin, lidocaine, or amiodarone; if ineffective, consider magnesium sulfate

#### *Asystole*

• Myocardial ischemia or infarction, aortic valve disease, heart failure, hypoxemia,

• Start CPR; follow ACLS protocol for ET intubation, transcutaneous pacing, and



- No atrial or ventricular rate or rhythm
- No discernible P waves, QRS complexes, or T waves

hypokalemia, severe acidosis, electric shock, ventricular arrhythmias, AV block, pulmonary embolism, heart rupture, cardiac tamponade, hyperkalemia, electromechanical dissociation  
• Cocaine overdose

administration of epinephrine or vasopressin; and consider atropine

## VASCULAR DISORDERS

### *Thoracic aortic aneurysm*

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. Aneurysms may be *dissecting*, a hemorrhagic separation in the aortic wall, usually within the medial layer; *saccular*, an outpouching of the arterial wall, with a narrow neck; or *fusiform*, a spindle-shaped enlargement encompassing the entire aortic circumference. (See *Types of aortic aneurysms*, page 82.) Some aneurysms progress to serious and, eventually, lethal complications, such as rupture of an untreated thoracic dissecting aneurysm into the pericardium, with resulting tamponade.

### *Causes and incidence*

Thoracic aortic aneurysms commonly result from atherosclerosis, which weakens the aortic wall and gradually distends the lumen. An intimal tear in the ascending aorta initiates dissecting aneurysm in about 60% of the patients. Regardless of causation, these aneurysms affect 6 out of every 100,000 people.

#### ELDER TIP

*Ascending aortic aneurysms, the most common type, are usually seen in hypertensive men younger than age 60. Descending aortic aneurysms, usually found just below the origin of the subclavian artery, are most common in elderly men who are hypertensive.*

Descending aortic aneurysms are also seen in younger patients with a history of traumatic chest injury; less often in those

with infection. Transverse aortic aneurysms are the least common type.

Other causes include:

- fungal infection (mycotic aneurysms) of the aortic arch and descending segments
- congenital disorders, such as coarctation of the aorta and Marfan syndrome
- trauma, usually of the descending thoracic aorta, from an accident that shears the aorta transversely (acceleration/deceleration injuries)
- syphilis, usually of the ascending aorta (uncommon because of antibiotics)
- hypertension (in dissecting aneurysm).

## **Complications**

- Rupture into pericardium
- Cardiac tamponade

## **Signs and symptoms**

The most common symptom of thoracic aortic aneurysm is pain. With ascending aneurysm, the pain is described as severe, boring, and ripping and extends to the neck, shoulders, lower back, or abdomen but seldom radiates to the jaw and arms. Pain is more severe on the right side.

Other signs of ascending aneurysm may include bradycardia, aortic insufficiency, pericardial friction rub caused by a hemopericardium, unequal intensities of the right carotid and left radial pulses, and a difference in blood pressure between the right and left arms. These signs are absent in descending aneurysm. If dissection involves the carotids, an abrupt onset of neurologic deficits may occur.

With descending aneurysm, pain usually starts suddenly between the shoulder blades and may radiate to the chest; it's described as sharp and tearing. Transverse aneurysm causes a sudden, sharp, tearing pain radiating to the shoulders. It may also cause hoarseness, dyspnea, dysphagia, and a dry cough because of compression of surrounding structures in this area. (See *Clinical characteristics of thoracic dissection*, page 83.)

## **Diagnosis**

Diagnosis relies on patient history, clinical features, and appropriate tests. In an asymptomatic patient, diagnosis often occurs accidentally when chest X-rays show widening of the aorta. Other tests help confirm aneurysm:

- Aortography, the most definitive test, shows the lumen of the aneurysm, its size and location, and the false lumen in dissecting aneurysm.
- Electrocardiography (ECG) helps distinguish thoracic aneurysm from myocardial infarction.
- Echocardiography may help identify dissecting aneurysm of the aortic root.
- Hemoglobin levels may be normal or low, due to blood loss from a leaking aneurysm.
- Computed tomography scan can confirm and locate the aneurysm and may be used to monitor its progression.
- Magnetic resonance imaging may aid diagnosis.
- Transesophageal echocardiography is used to diagnose and size an aneurysm in either the ascending or the descending aorta.

## **Treatment**

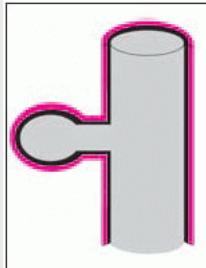
Dissecting aortic aneurysm is an emergency that requires prompt surgery and stabilizing measures: antihypertensives such as nitroprusside; negative inotropic agents that decrease contractility force such as propranolol; oxygen for respiratory distress; opioids for pain; I.V. fluids and, possibly, whole blood transfusions.

Surgery consists of resecting the aneurysm, restoring normal blood flow through a Dacron or Teflon graft replacement and, with aortic valve insufficiency, replacing the aortic valve. Groin catheter placement may be used for aortic stenting. This procedure, which may be used for aneurysms of the descending aorta, eliminates the need for a chest incision.

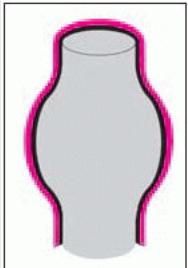
Postoperative measures include careful monitoring and continuous assessment in the intensive care unit, antibiotics, endotracheal (ET) and chest tubes, ECG monitoring, and pulmonary artery catheterization.

Long-term management includes treatment of underlying conditions, such as heart disease and diabetes.

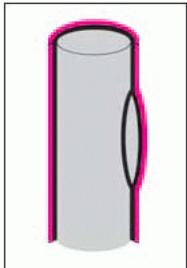
## TYPES OF AORTIC ANEURYSMS



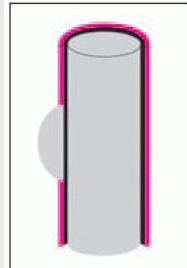
**Saccular**  
Unilateral pouchlike bulge with a narrow neck



**Fusiform**  
A spindle-shaped bulge encompassing the vessel's entire diameter



**Dissecting**  
A hemorrhagic separation of the medial layer of the vessel wall, which creates a false lumen



**False aneurysm**  
A pulsating hematoma resulting from trauma; usually seen in the femoral artery after catheterization

## *Special considerations*

- Monitor blood pressure, pulmonary artery wedge pressure (PAWP), and central venous pressure (CVP). Assess pain, breathing, and carotid, radial, and femoral pulses.
- Make sure laboratory tests include complete blood count, differential, electrolyte levels, type and crossmatching for whole blood, arterial blood gas studies, and urinalysis.
- Insert an indwelling urinary catheter. Administer dextrose 5% in water or lactated Ringer's solution, and antibiotics, as ordered. Carefully monitor nitroprusside I.V.; use a separate I.V. line for infusion. Adjust the dose by slowly increasing the infusion rate. Meanwhile, check blood pressure every 5 minutes until it stabilizes. With suspected bleeding from aneurysm, give whole blood transfusion.
- Explain diagnostic tests. If surgery is scheduled, explain the procedure and expected postoperative care (I.V. lines, ET and drainage tubes, cardiac monitoring, and ventilation).

After repair of thoracic aneurysm:

- Assess level of consciousness. Monitor vital signs; pulmonary artery pressure, PAWP, and CVP; pulse rate; urine output; and pain.
- Check respiratory function. Carefully observe and record type and amount of chest-tube drainage, and frequently assess heart and breath sounds.
- Monitor I.V. therapy.
- Give medications as appropriate.
- Watch for signs of infection, especially fever, and excessive wound drainage.
- Assist with range-of-motion exercises of legs to prevent thromboembolic phenomenon due to venostasis during prolonged bed rest.
- After stabilization of vital signs and respiration, encourage and assist the patient in turning, coughing, and deep breathing. If necessary, provide intermittent positive-pressure breathing to promote lung expansion. Help the patient walk as soon as he's able.
- Before discharge, ensure compliance with antihypertensive therapy by explaining the need for such drugs and the expected adverse effects. Teach the patient how to monitor his blood pressure. Refer

him to community agencies for continued support and assistance, as needed.

- Throughout hospitalization, offer the patient and his family psychological support. Answer all of their questions honestly and provide reassurance.

## **CLINICAL CHARACTERISTICS OF THORACIC DISSECTION**

Ascending aorta	Descending aorta	Transverse aorta
<b><i>Character of pain</i></b>		
Severe, boring, ripping, extending to neck, shoulders, lower back, or abdomen (rarely to jaw and arms); more severe on right side	Sudden onset, sharp, tearing, usually between the shoulder blades; may radiate to the chest; most diagnostic feature	Sudden onset, sharp, boring, tearing, radiates to shoulders
<b><i>Other symptoms and effects</i></b>		
If dissection involves carotids, abrupt onset of neurologic deficit (usually intermittent); bradycardia, aortic insufficiency, and hemopericardium detected by pericardial friction rub; unequal intensity of right and left carotid pulses and radial pulses; difference in blood pressure, especially systolic, between right and left arms	Aortic insufficiency without murmur, hemopericardium, or pleural friction rub; carotid and radial pulses and blood pressure in both arms tend to stay equal	Hoarseness, dyspnea, pain, dysphagia, and dry cough resulting from compression of surrounding structures
<b><i>Diagnostic features</i></b>		
<b><i>Chest X-ray</i></b>		
Best diagnostic tool; shows widening of mediastinum, enlargement of ascending aorta	Shows widening of mediastinum, descending aorta larger than ascending	Shows widening of mediastinum, descending aorta larger than ascending, widened transverse arch
<b><i>Aortography</i></b>		
Shows false lumen; narrowing of lumen of aorta in ascending section	Shows false lumen; narrowing of lumen of aorta in descending section	Shows false lumen, narrowing of lumen of aorta in transverse arch
<b><i>Treatment</i></b>		
This is a medical emergency requiring immediate, aggressive treatment to reduce blood pressure (usually with nitroprusside or trimethaphan). Surgical repair is also required.	Surgical repair is required but less urgent than for the ascending dissection. Nitroprusside and propranolol may be used to control hypertension if bradycardia and heart failure are absent.	Immediate surgical repair (mortality as high as 50%) and control of hypertension are required.

## **Abdominal aneurysm**

Abdominal aneurysm, an abnormal dilation in the arterial wall, generally occurs in the aorta between the renal arteries and iliac branches. Rupture—in which the aneurysm breaks open, resulting in profuse bleeding—is a common complication that occurs in larger aneurysms. Dissection occurs when the artery's lining tears, and blood leaks into the walls.

## **Causes and incidence**

Abdominal aortic aneurysms result from arteriosclerosis, hypertension, congenital weakening, cystic medial necrosis, trauma, syphilis, and other infections. In children, this disorder can result from blunt abdominal injury or Marfan syndrome. These aneurysms develop slowly. First, a focal weakness in the muscular layer of the aorta (tunica media), due to degenerative changes, allows the inner layer (tunica intima) and outer layer (tunica adventitia) to stretch outward. Blood pressure within the aorta progressively weakens the vessel walls and enlarges the aneurysm.

This disorder is four times more common in men than in women and is most prevalent in whites ages 40 to 70. Less than 50% of people with a ruptured abdominal aortic aneurysm survive.

## **Complications**

- Rupture
- Hemorrhage
- Shock

## **Signs and symptoms**

Although abdominal aneurysms usually don't produce symptoms, most are evident (unless the patient is obese) as a pulsating mass in the periumbilical area, accompanied by a systolic bruit over the aorta. Some tenderness may be present on deep palpation. A large aneurysm may produce symptoms that mimic renal calculi, lumbar disk disease, and duodenal compression. Abdominal aneurysms rarely cause diminished peripheral pulses or claudication, unless embolization occurs.

Lumbar pain that radiates to the flank and groin from pressure on lumbar nerves may signify enlargement and imminent rupture. A rare but recognized symptom is unrelenting testicular pain with no other cause. If the aneurysm ruptures into the peritoneal cavity, it causes severe, persistent abdominal and back pain, mimicking renal or ureteral colic. Signs of hemorrhage—such as weakness, sweating, tachycardia, and hypotension—may be subtle because rupture into the retroperitoneal space produces a tamponade effect that prevents continued hemorrhage. Patients with such rupture may remain stable for hours before shock and death occur, although 20% die immediately.

## **Diagnosis**

Because abdominal aneurysms seldom produce symptoms, they're commonly detected accidentally as the result of an X-ray or a routine physical examination.

### **CONFIRMING DIAGNOSIS**

Several tests can confirm a suspected abdominal aneurysm. Serial ultrasound (sonography) can accurately determine the aneurysm's size, shape, and location. Anteroposterior and lateral X-rays of the abdomen can detect aortic calcification, which outlines the mass, at least 75% of the time. Aortography shows the condition of vessels proximal and distal to the aneurysm and the aneurysm's extent but may underestimate aneurysm diameter because it visualizes only the flow channel and not the surrounding clot. Computed tomography scan is used to diagnose and size the aneurysm. Magnetic resonance imaging can be used as an alternative to aortography.

## **Treatment**

Usually, abdominal aneurysm requires resection of the aneurysm and replacement of the damaged aortic section with a Dacron graft. (See *Abdominal aneurysms: Before and after surgery*, page 86. Also see *Endovascular grafting for repair of an AAA*, page 87.) If the aneurysm is small and asymptomatic, surgery may be delayed and the aneurysm may be followed and allowed

to expand to a certain size because of possible surgical complications; however, small aneurysms may also rupture. Because of this risk, surgical repair or replacement is recommended for symptomatic patients or for patients with aneurysms greater than 5 cm in diameter.

Stenting is also a treatment option. It can be performed without an abdominal incision by introducing the catheters through arteries in the groin. However, not all patients with abdominal aortic aneurysms are candidates for this treatment.

Regular physical examination and ultrasound checks are necessary to detect enlargement, which may forewarn rupture. Large aneurysms or those that produce symptoms pose a significant risk of rupture and necessitate immediate repair. In patients with poor distal runoff, external grafting may be done.

Risk factor modification is fundamental in the medical management of abdominal aneurysm, including control of hypocholesterolemia and hypertension. Beta-adrenergic blockers are commonly prescribed to reduce the risk of aneurysm expansion and rupture.

## **Special considerations**

Abdominal aneurysm requires meticulous preoperative and postoperative care, psychological support, and comprehensive patient teaching. Following diagnosis, if rupture isn't imminent, elective surgery allows time for additional preoperative tests to evaluate the patient's clinical status.

- Monitor vital signs, and type and crossmatch blood.
- Use only gentle abdominal palpation.
- As ordered, obtain renal function tests (blood urea nitrogen, creatinine, and electrolyte levels), blood samples (complete blood count with differential), electrocardiogram and cardiac evaluation, baseline pulmonary function tests, and arterial blood gas (ABG) analysis.
- Be alert for signs of rupture, which may be immediately fatal. Watch closely for signs of acute blood loss (decreasing blood pressure; increasing pulse and respiratory rate; cool, clammy skin; restlessness; and decreased sensorium).
- If rupture does occur, the first priority is to get the patient to surgery immediately. A pneumatic antishock garment may be used while transporting him to surgery. Surgery allows direct compression of the aorta to control hemorrhage. Large amounts of blood may be needed during the resuscitative period to replace blood loss. In such a patient, renal failure caused by ischemia is a major postoperative complication, possibly requiring hemodialysis.
- Before elective surgery, weigh the patient, insert an indwelling urinary catheter and an I.V. line, and assist with insertion of an arterial line and pulmonary artery catheter to monitor fluid and hemodynamic balance. Give prophylactic antibiotics as ordered.
- Explain the surgical procedure and the expected postoperative care in the intensive care unit (ICU) for patients undergoing complex abdominal surgery (I.V. lines, endotracheal [ET] and nasogastric [NG] intubation, and mechanical ventilation).
- After surgery, in the ICU, closely monitor vital signs, intake and hourly output, neurologic status (level of consciousness, pupil size, and sensation in arms and legs), and ABG values. Assess the depth, rate, and character of respirations and breath sounds at least every hour.

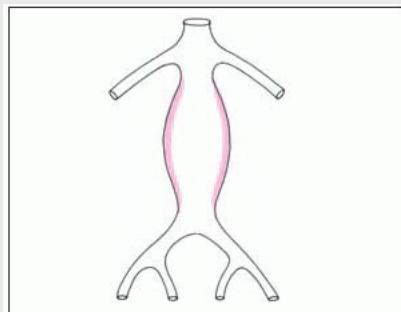
- Watch for signs of bleeding (increased pulse and respiratory rates and hypotension) and back pain, which may indicate the graft is tearing. Check abdominal dressings for excessive bleeding or drainage. Be alert for temperature elevations and other signs of infection. After NG intubation for intestinal decompression, irrigate the tube frequently to ensure patency. Record the amount and type of drainage.
- Suction the ET tube often. If the patient can breathe unassisted and has good breath sounds and adequate ABG values, tidal volume, and vital capacity 24 hours after surgery, he will be extubated and will require oxygen by mask.
- Weigh the patient daily to evaluate fluid balance.
- Help the patient walk as soon as he's able (generally the second day after surgery).
- Provide psychological support for the patient and his family. Help ease their fears

about the ICU, the threat of impending rupture, and surgery by providing appropriate explanations and answering all questions.

## **ABDOMINAL ANEURYSMS: BEFORE AND AFTER SURGERY**

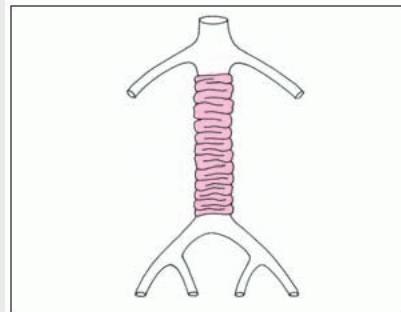
During surgery, a prosthetic graft replaces or encloses the weakened area.

Before surgery



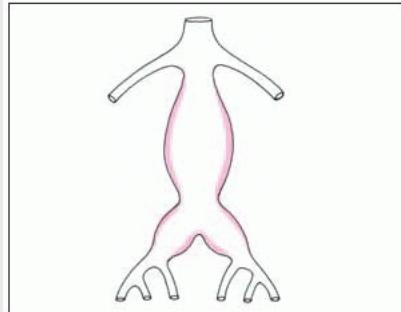
Aneurysm below renal arteries and above bifurcation

After surgery



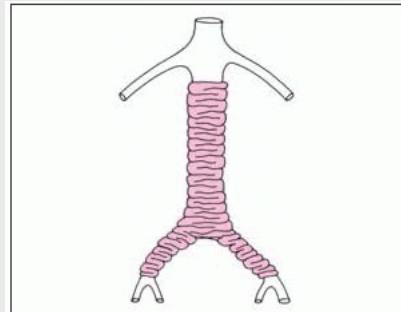
The prosthesis extends distal to the renal arteries to above the aortic bifurcation.

Before surgery



Aneurysm below renal arteries involving the iliac branches

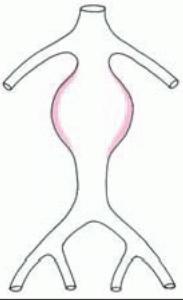
After surgery



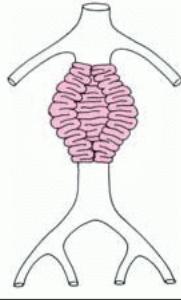
The prosthesis extends to the common femoral arteries.

Before surgery

After surgery



Small aneurysm in a patient with poor distal runoff (poor risk)



The external prosthesis encircles the aneurysm and is held in place with sutures.

## Femoral and popliteal aneurysms

Femoral and popliteal aneurysms (sometimes called *peripheral arterial aneurysms*) are the end result of progressive atherosclerotic changes occurring in the walls (medial layer) of these major peripheral arteries. These aneurysmal formations may be *fusiform* (spindle-shaped) or *saccular* (pouchlike); the fusiform type is three times more common. They may be singular or multiple segmental lesions, often affecting both legs, and may accompany other arterial aneurysms located in the abdominal aorta or iliac arteries. (See *Arteries of the leg*.)

This condition occurs most frequently in men older than age 50. The clinical course is usually progressive, eventually ending in thrombosis, embolization, and gangrene. Elective surgery before complications arise greatly improves the prognosis.

## Causes and incidence

Femoral and popliteal aneurysms are usually secondary to atherosclerosis. Rarely, they result from congenital weakness in the arterial wall. They may also result from trauma (blunt or penetrating), bacterial infection, or peripheral vascular reconstructive surgery (which causes “suture line” aneurysms, or false aneurysms, in which a blood clot forms a second lumen).

## Complications

- Amputation of thrombosis
- Emboli
- Gangrene

## Signs and symptoms

Popliteal aneurysms may cause pain in the popliteal space when they're large enough to compress the medial popliteal nerve and edema and venous distention if the vein is compressed. Femoral and popliteal aneurysms can produce symptoms of severe ischemia in the leg or foot due to acute

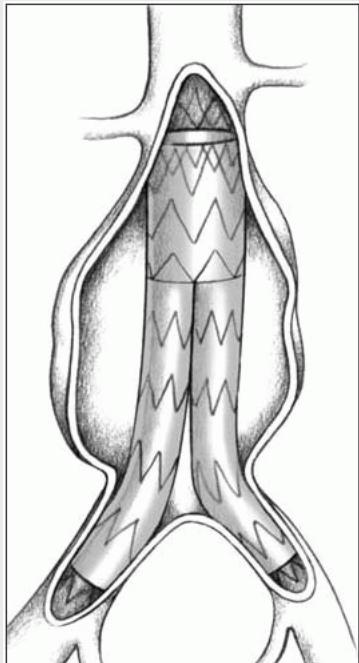
thrombosis within the aneurysmal sac, embolization of mural thrombus fragments and, rarely, rupture. Symptoms of acute aneurysmal thrombosis include severe pain, loss of pulse and color, coldness in the affected leg or foot, and gangrene. Distal petechial hemorrhages may develop from aneurysmal emboli.

## ENDOVASCULAR GRAFTING FOR REPAIR OF AN AAA

Endovascular grafting is a minimally invasive procedure for the repair of an abdominal aortic aneurysm (AAA). This procedure reinforces the walls of the aorta to prevent rupture and prevent expansion of the aneurysm.

Endovascular grafting is performed with fluoroscopic guidance: Using a guide wire, a delivery catheter with an attached compressed graft is inserted through a small incision into the femoral or iliac artery. The delivery catheter is advanced into the aorta, where it's positioned across the aneurysm. A balloon on the catheter expands the graft and affixes it to the vessel wall.

The procedure generally takes 2 to 3 hours to perform. Patients are instructed to walk the first day after surgery and are generally discharged from the facility in 1 to 3 days.



## ***Diagnosis***

Diagnosis is usually confirmed by bilateral palpation that reveals a pulsating mass above or below the inguinal ligament in femoral aneurysm. When thrombosis has occurred, palpation detects a firm, nonpulsating mass. Arteriography or ultrasound may be indicated in doubtful situations. Arteriography may also detect associated aneurysms, especially those in the abdominal aorta and the iliac arteries. Ultrasound may be helpful in determining the size of the popliteal or femoral artery.

## ***Treatment***

Femoral and popliteal aneurysms require surgical bypass and reconstruction of the artery, usually with an autogenous saphenous vein graft replacement. Arterial occlusion that causes severe ischemia and gangrene may require leg amputation.

## ***Special considerations***

Before corrective surgery:

- Assess and record circulatory status, noting the location and quality of peripheral pulses in the affected arm or leg.
- Administer prophylactic antibiotics or anticoagulants, as ordered.
- Discuss postoperative procedures and review the explanation of the surgery.

After arterial surgery:

- Monitor carefully for early signs of thrombosis or graft occlusion (loss of pulse, decreased skin temperature and sensation, and severe pain) and infection (fever).
- Palpate distal pulses at least every hour for the first 24 hours and then as frequently as ordered. Correlate these findings with preoperative circulatory assessment. Mark the sites on the patient's skin where pulses are palpable to facilitate repeated checks.
- Help the patient walk soon after surgery to prevent venostasis and possible thrombus formation.

To prepare the patient for discharge:

- Tell the patient to immediately report any recurrence of symptoms because the saphenous vein graft replacement can fail or another aneurysm may develop.
- Explain to the patient with popliteal artery resection that swelling may persist for some time. If antiembolism stockings are ordered, make sure they fit properly and teach the patient how to apply them. Warn against wearing constrictive apparel.
- If the patient is receiving anticoagulants, suggest measures to prevent bleeding such as using an electric razor. Tell him to report any signs of bleeding immediately (bleeding gums, tarry stools, and easy bruising). Explain the importance of follow-up blood studies to monitor anticoagulant therapy. Warn him to avoid trauma, tobacco, and aspirin.

## ***Thrombophlebitis***

An acute condition characterized by inflammation and thrombus formation, thrombophlebitis may occur in deep (intermuscular or intramuscular) or superficial (subcutaneous) veins. Deep vein thrombosis (DVT) or thrombophlebitis affects small veins, such as the soleal venous sinuses, or large veins, such as the vena cava and the femoral, iliac, and subclavian veins, causing venous insufficiency. (See *Chronic venous insufficiency*.) This disorder is typically progressive, leading to pulmonary embolism, a potentially lethal complication. Superficial thrombophlebitis is usually self-limiting and seldom leads to pulmonary embolism. Thrombophlebitis often begins with localized inflammation alone (phlebitis), but such inflammation rapidly provokes thrombus formation. Rarely, venous thrombosis develops without associated inflammation of the vein (phlebothrombosis).

## ***Causes and incidence***

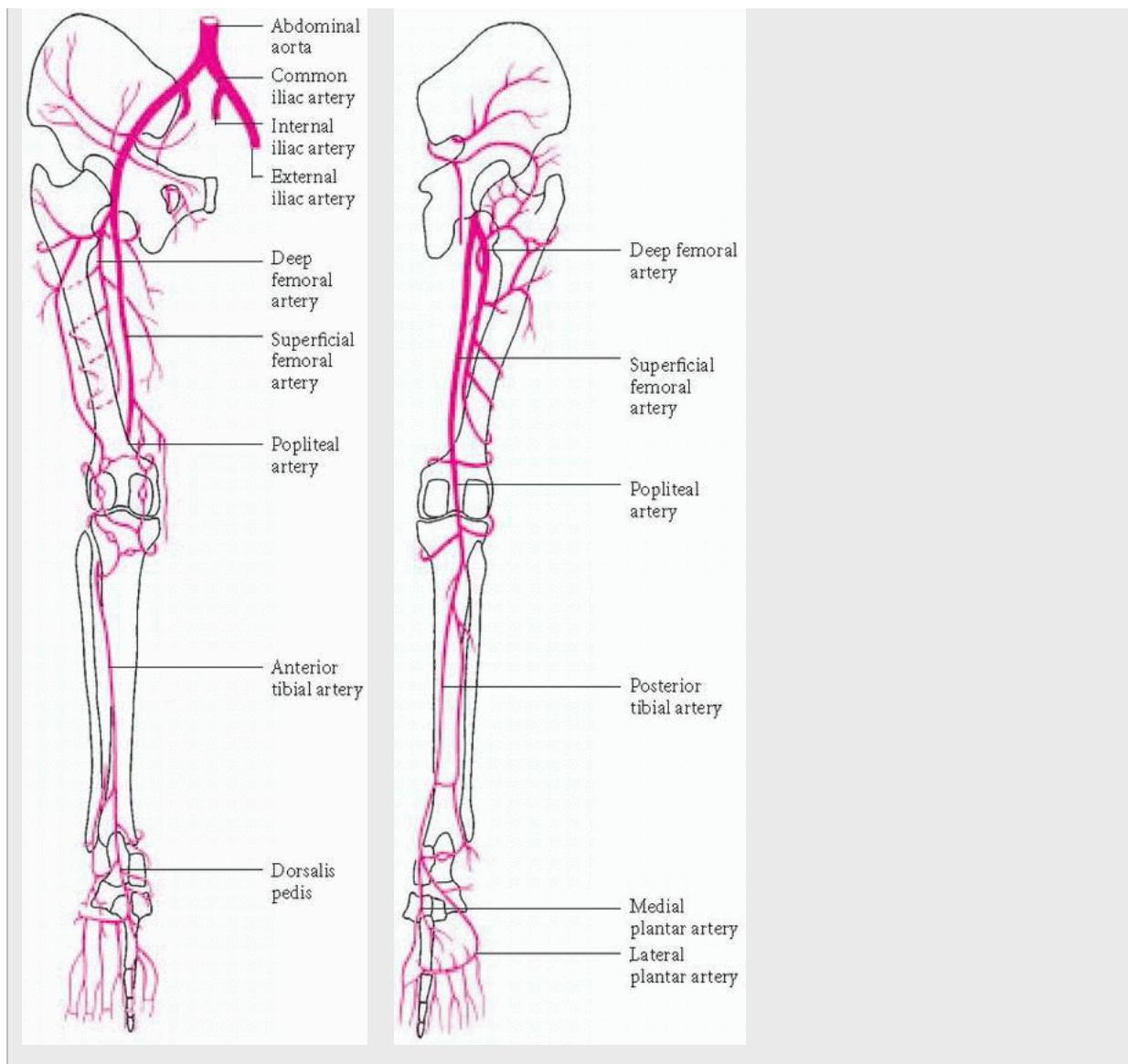
A thrombus occurs when an alteration in the epithelial lining causes platelet aggregation

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, due to greater contact between platelet and thrombin accumulation. The rapidly expanding thrombus initiates a chemical inflammatory process in the vessel epithelium, which leads to fibrosis. The enlarging clot may occlude the vessel lumen partially or totally, or it may detach and embolize to lodge elsewhere in the systemic circulation.

## **ARTERIES OF THE LEG**

FRONT VIEW

BACK VIEW



## CHRONIC VENOUS INSUFFICIENCY

Chronic venous insufficiency results from the valvular destruction of deep vein thrombophlebitis, usually in the iliac and femoral veins, and occasionally the saphenous veins. It's often accompanied by incompetence of the communicating veins at the ankle, causing increased venous pressure and fluid migration into the interstitial tissue. Clinical effects include chronic swelling of the affected leg from edema, leading to tissue fibrosis, and induration; skin discoloration from extravasation of blood in subcutaneous tissue; and stasis ulcers around the ankle.

Treatment of small ulcers includes bed rest, elevation of the legs, warm soaks, and antimicrobial therapy for infection. Treatment to counteract increased venous pressure, the result of reflux from the deep venous system to surface veins, may include compression dressings, such as a sponge rubber pressure

dressing or a zinc gelatin boot (Unna's boot). This therapy begins after massive swelling subsides with leg elevation and bed rest.

Large stasis ulcers unresponsive to conservative treatment may require excision and skin grafting. Patient care includes daily inspection to assess healing. Other care measures are the same as for varicose veins.

DVT may be idiopathic, but it usually results from endothelial damage, accelerated blood clotting, and reduced blood flow known as Virchow's triad. Predisposing factors are prolonged bed rest, trauma, surgery, childbirth, and use of hormonal contraceptives such as estrogens. It occurs in about 80 of every 100,000 people; 1 of every 20 persons is affected at some point during his lifetime. Males are at slightly greater risk than females. People older than age 40 are also at increased risk.

Causes of superficial thrombophlebitis include trauma, infection, I.V. drug abuse, and chemical irritation due to extensive use of the I.V. route for medications and diagnostic tests.

### ***Complications***

- Pulmonary embolism
- Chronic venous insufficiency

### ***Signs and symptoms***

In both types of thrombophlebitis, clinical features vary with the site and length of the affected vein. Although DVT may occur asymptotically, it may also produce severe pain, fever, chills, malaise and, possibly, swelling and cyanosis of the affected arm or leg. Superficial thrombophlebitis produces visible and palpable signs, such as heat, pain, swelling, rubor, tenderness, and induration along the length of the affected vein. Varicose veins may also be present. (See *Varicose veins*.) Extensive vein involvement may cause lymphadenitis.

### ***Diagnosis***

Some patients may display signs of inflammation and, possibly, a positive Homans' sign (pain on dorsiflexion of the foot) during physical examination; others are asymptomatic. Physical findings are usually non-specific and not reliable for making the diagnosis of DVT. Essential laboratory tests include:

- Duplex Doppler ultrasonography and impedance plethysmography make it possible to noninvasively examine the major veins (but not calf veins).
- Plethysmography shows decreased circulation distal to the affected area; this test is more sensitive than ultrasound in detecting DVT.

### ***VARICOSE VEINS***

Varicose veins are dilated, tortuous veins, usually affecting the subcutaneous leg veins—the saphenous veins and their branches. They can result from congenital weakness of the valves or venous wall, diseases of the venous system such as deep vein thrombophlebitis, conditions that produce prolonged venostasis such as pregnancy, or occupations that necessitate standing for an extended period.

Varicose veins may be asymptomatic or produce mild to severe leg symptoms, including a feeling of heaviness; cramps at night; diffuse, dull aching after prolonged standing or walking; aching during menses; fatigability; palpable nodules and, with deep-vein incompetency, orthostatic edema and stasis pigmentation of the calves and ankles.

## **Treatment**

In mild to moderate varicose veins, antiembolism stockings or elastic bandages counteract pedal and ankle swelling by supporting the veins and improving circulation. An exercise program such as walking promotes muscular contraction and forces blood through the veins, thereby minimizing venous pooling. Severe varicose veins may necessitate stripping and ligation or, as an alternative to surgery, injection of a sclerosing agent into small affected vein segments.

To promote comfort and minimize worsening of varicosities:

- Discourage the patient from wearing constrictive clothing.
- Advise the patient to elevate his legs above heart level whenever possible and to avoid prolonged standing or sitting.

After stripping and ligation or after injection of a sclerosing agent:

- To relieve pain, administer analgesics as ordered.
- Frequently check circulation in toes (color and temperature) and observe elastic bandages for bleeding. When ordered, rewrap bandages at least once a shift, wrapping from toe to thigh, with the leg elevated.
- Watch for signs of complications, such as sensory loss in the leg (which could indicate saphenous nerve damage), calf pain (thrombophlebitis), and fever (infection).

## **□ CONFIRMING DIAGNOSIS**

*Phlebography, which shows filling defects and diverted blood flow, usually confirms the diagnosis.*

Diagnosis must also rule out peripheral artery disease, lymphangitis, cellulitis, and myositis.

Diagnosis of superficial thrombophlebitis is based on physical examination (redness and warmth over the affected area, palpable vein, and pain during palpation or compression).

## **Treatment**

The goals of treatment are to control thrombus development, prevent complications, relieve pain, and prevent recurrence of the disorder. Symptomatic measures include bed rest, with elevation of the affected arm or leg; warm, moist soaks to the affected area; and analgesics. After the acute episode of DVT subsides, the patient may resume activity while wearing antiembolism stockings that were applied before he got out of bed.

Treatment also includes anticoagulants (initially, heparin; later, warfarin) to prolong clotting time. Low-molecular-weight (LMW) heparin has been shown to be effective in treating DVT. Although LMW heparin is more expensive, it doesn't require monitoring for its anticoagulant effect. Full anticoagulant doses must be discontinued during any operative period because of the risk of hemorrhage. After some types of surgery, especially major abdominal

or pelvic operations, prophylactic doses of anticoagulants may reduce the risk of DVT and pulmonary embolism. For lysis of acute, extensive DVT, treatment should include streptokinase. Rarely, DVT may cause complete venous occlusion, which necessitates venous interruption through simple ligation to vein plication, or clipping. Embolectomy and insertion of a vena caval umbrella or filter may also be done.

Therapy for severe superficial thrombophlebitis may include an anti-inflammatory drug such as indomethacin, antiembolism stockings, warm soaks, and elevation of the leg.

## **Special considerations**

Patient teaching, identification of high-risk patients, and measures to prevent venostasis can prevent DVT; close monitoring of anticoagulant therapy can prevent serious complications such as internal hemorrhage.

- Enforce bed rest as ordered, and elevate the patient's affected arm or leg. If you plan to use pillows for elevating the leg, place them so they support the entire length of the affected extremity to prevent possible compression of the popliteal space.
- Apply warm soaks to increase circulation to the affected area and to relieve pain and inflammation. Give analgesics to relieve pain, as ordered.
- Measure and record the affected arm or leg's circumference daily and compare this measurement to the other arm or leg. To ensure accuracy and consistency of serial measurements, mark the skin over the area and measure at the same spot daily.
- Administer heparin I.V., as ordered, with an infusion monitor or pump to control the flow rate if necessary.
- Measure partial thromboplastin time regularly for the patient on heparin therapy; prothrombin time and international normalized ratio (INR) for the patient on warfarin (therapeutic anticoagulation values are 1½ to 2 times control values for prothrombin time and an INR of 2 to 3). Watch for signs and symptoms of bleeding, such as dark, tarry stools; coffee-ground vomitus; and ecchymoses. Encourage the patient to use an electric razor and to avoid medications that contain aspirin.
- Be alert for signs of pulmonary emboli (crackles, dyspnea, hemoptysis, sudden changes in mental status, restlessness, and hypotension).

To prepare the patient with thrombophlebitis for discharge:

- Emphasize the importance of follow-up blood studies to monitor anticoagulant therapy.
- If the patient is being discharged on heparin therapy, teach him or his family how to give subcutaneous injections. If he requires further assistance, arrange for a home health nurse.
- Tell the patient to avoid prolonged sitting or standing to help prevent recurrence.
- Teach the patient how to properly apply and use antiembolism stockings. Tell him to report any complications such as cold, blue toes. (See *Preventing thrombophlebitis*.)

## **Raynaud's disease**

Raynaud's disease is one of several primary arteriospastic disorders characterized by episodic vasospasm in 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. Raynaud's disease is most prevalent in females, particularly those between puberty and age 40. It's a benign condition, requiring no specific treatment and causing no serious sequelae.

Raynaud's phenomenon, however, a condition commonly associated with several connective tissue disorders—such as scleroderma, systemic lupus erythematosus (SLE), or polymyositis—has a progressive course, leading to ischemia, gangrene, and amputation. Distinguishing between the two disorders is difficult because some patients who experience mild symptoms of Raynaud's disease for several years may later develop overt connective tissue disease—especially scleroderma.



### **PREVENTION**

#### **PREVENTING THROMBOPHLEBITIS**

To prevent thrombophlebitis in a high-risk patient, perform range-of-motion exercise while the patient is on bedrest, use intermittent pneumatic calf

massage during lengthy surgical or diagnostic procedures, apply antiembolism stockings postoperatively, and encourage early ambulation.

After some types of surgery, especially major abdominal or pelvic operations, prophylactic doses of anticoagulants may reduce the risk of deep vein thrombosis and pulmonary embolism.

## ***Causes and incidence***

Although the cause is unknown, several theories account for the reduced digital blood flow: intrinsic vascular wall hyperactivity to cold, increased vasomotor tone due to sympathetic stimulation, and antigen-antibody immune response (the most likely theory because abnormal immunologic test results accompany Raynaud's phenomenon). Risk factors include associated diseases (Buerger's disease, atherosclerosis, rheumatoid arthritis, scleroderma, and SLE) and smoking.

This disorder affects females more often than males.

## ***Complications***

- Ischemia
- Gangrene
- Amputation

## ***Signs and symptoms***

After exposure to cold or stress, the skin on the fingers typically blanches and then becomes cyanotic before changing to red and before changing from cold to normal temperature. Numbness and tingling may also occur. These symptoms are relieved by warmth. In long-standing disease, trophic changes, such as sclerodactyly, ulcerations, or chronic paronychia, may result. Although it's extremely uncommon, minimal cutaneous gangrene necessitates amputation of one or more phalanges.

## ***Diagnosis***

Clinical criteria that establish Raynaud's disease include skin color changes induced by cold or stress; bilateral involvement; absence of gangrene or, if present, minimal cutaneous gangrene; normal arterial pulses; and patient history of clinical symptoms of longer than 2 years' duration. Diagnosis must also rule out secondary disease processes, such as chronic arterial occlusive or connective tissue disease.

## ***Treatment***

Initially, treatment consists of avoidance of cold, mechanical, or chemical injury; cessation of smoking; and reassurance that symptoms are benign. Because adverse drug effects, especially from vasodilators, may be more bothersome than the disease itself, drug therapy is reserved for unusually severe symptoms. Such therapy may include phenoxybenzamine or reserpine; low doses of nifedipine have been shown to be effective. Sympathectomy may be helpful when conservative modalities fail to prevent ischemic ulcers and becomes necessary in less than 25% of patients.

## ***Special considerations***

- Warn the patient against exposure to the cold. Tell him to wear mittens or gloves in cold weather or when handling cold items or defrosting the freezer.
- Advise the patient to avoid stressful situations and to stop smoking.

- Instruct the patient to inspect the skin frequently and to seek immediate care for signs of skin breakdown or infection.
  - Teach the patient about drugs, their use, and their adverse effects.
  - Provide psychological support and reassurance to allay the patient's fear of amputation and disfigurement.
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## ***Buerger's disease***

Buerger's disease (sometimes called *thromboangiitis obliterans*)—an inflammatory, nonatheromatous occlusive condition—causes segmental lesions and subsequent thrombus formation in the small and medium arteries (and sometimes the veins), resulting in decreased blood flow to the feet and legs. This disorder may produce ulceration and, eventually, gangrene.

### ***Causes and incidence***

Buerger's disease is caused by vasculitis, an inflammation of blood vessels, primarily of the hands and feet. The vessels become constricted or totally blocked, reducing blood flow to the tissues and resulting in pain and, eventually, damage.

This disorder occurs in 6 of every 10,000 people. Incidence is highest among males ages 20 to 40 who have a history of smoking or chewing tobacco. It may be associated with a history of Raynaud's disease and may occur in people with autoimmune disease.

### ***Complications***

- Gangrene
- Muscle atrophy
- Ulceration

### ***Signs and symptoms***

Buerger's disease typically produces intermittent claudication of the instep, which is aggravated by exercise and relieved by rest. During exposure to low temperature, the feet initially become cold, cyanotic, and numb; later, they redden, become hot, and tingle. Occasionally, Buerger's disease also affects the hands, possibly resulting in painful fingertip ulcerations. Associated signs and symptoms may include impaired peripheral pulses, migratory superficial thrombophlebitis and, in later stages, ulceration, muscle atrophy, and gangrene.

### ***Diagnosis***

Patient history and physical examination strongly suggest Buerger's disease. Supportive diagnostic tests include:

- Doppler ultrasonography to show diminished circulation in the peripheral vessels
- plethysmography to help detect decreased circulation in the peripheral vessels
- angiography or arteriography to locate lesions and rule out atherosclerosis.

### ***Treatment***

The primary goals of treatment are to relieve symptoms and prevent complications. Such therapy may include an exercise program that uses gravity to fill and drain the blood vessels or, in severe disease,

a lumbar sympathectomy to increase blood supply to the skin. Aspirin and vasodilators may also be used. Amputation may be necessary for nonhealing ulcers, intractable pain, or gangrene.

### ***Special considerations***

- Strongly urge the patient to stop smoking to enhance the treatment's effectiveness. Symptoms may disappear if he stops his tobacco use. If necessary, refer him to a self-help group to stop smoking.
- Warn the patient to avoid precipitating factors, such as emotional stress, exposure to extreme temperatures, and trauma.
- Teach the patient proper foot care, especially the importance of wearing well-fitting shoes and cotton or wool socks. Show him how to inspect his feet daily for cuts, abrasions, and signs of skin breakdown, such as redness and soreness. Remind him to seek medical attention at once after any trauma.
- If the patient has ulcers and gangrene, enforce bed rest and use a padded footboard or bed cradle to prevent pressure from bed linens. Protect the feet with soft padding. Wash them gently with a mild soap and tepid water, rinse thoroughly, and pat dry with a soft towel.
- Provide emotional support. If necessary, refer the patient for psychological counseling to help him cope with restrictions imposed by this chronic disease. If he has undergone amputation, assess rehabilitative needs, especially regarding changes in body image. Refer him to physical therapists, occupational therapists, and social service agencies, as needed.

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### ***Peripheral artery disease***

Peripheral artery disease (PAD), referred to as *arterial occlusive disease*, is the obstruction or narrowing of the lumen of the aorta and its major branches, causing an interruption of blood flow, usually to the legs and feet. PAD may affect the carotid, vertebral, innominate, subclavian, mesenteric, and celiac arteries. Occlusions may be acute or chronic and commonly cause severe ischemia, skin ulceration, and gangrene.

The prognosis depends on the occlusion's location, the development of collateral circulation to counteract reduced blood flow and, in acute disease, the time elapsed between occlusion and its removal.

### ***Causes and incidence***

Peripheral artery disease is a common complication of atherosclerosis. The occlusive mechanism may be endogenous, due to emboli formation or thrombosis, or exogenous, due to trauma or fracture. Predisposing factors include smoking; aging; such conditions as hypertension, hyperlipidemia, and diabetes; and a family history of vascular disorders, myocardial infarction, or stroke.

Peripheral artery disease has no racial predilection. Men older than 50 are at increased risk for intermittent claudication, a common sign of peripheral artery disease.

### ***Complications***

- Severe ischemia
- Skin ulceration
- Gangrene
- Limb loss

### ***Signs and symptoms***

The signs and symptoms of peripheral artery disease depend on the site of the occlusion. (See *Types of peripheral artery disease*, page 96.)

## **Diagnosis**

Diagnosis of peripheral artery disease is usually indicated by patient history and physical examination.

Pertinent supportive diagnostic tests include the following:

- Arteriography demonstrates the type (thrombus or embolus), location, and degree of obstruction and the collateral circulation. It's particularly useful in chronic disease or for evaluating candidates for reconstructive surgery.
- Doppler ultrasonography and plethysmography are noninvasive tests that show decreased blood flow distal to the occlusion in acute disease.
- Ophthalmodynamometry helps determine the degree of obstruction in the internal carotid artery by comparing ophthalmic artery pressure to brachial artery pressure on the affected side. More than a 20% difference between pressures suggests insufficiency.
- EEG and computed tomography scan may be necessary to rule out brain lesions.

## **Treatment**

Treatment depends on the obstruction's cause, location, and size. For mild chronic disease, supportive measures include elimination of smoking, hypertension control, and walking exercise. For carotid artery occlusion, antiplatelet therapy may begin with ticlopidine or clopidogrel and aspirin. For intermittent claudication of chronic occlusive disease, pentoxifylline and cilostazol may improve blood flow through the capillaries, particularly for patients who are poor candidates for surgery.

Acute peripheral artery disease usually requires surgery to restore circulation to the affected area, for example:

- Atherectomy—Excision of plaque using a drill or slicing mechanism.
  - Balloon angioplasty—Compression of the obstruction using balloon inflation.
  - Bypass graft—Blood flow is diverted through an anastomosed autogenous or Dacron graft past the thrombosed segment.
  - Combined therapy—Concomitant use of any of the above treatments.
  - Embolectomy—A balloon-tipped Fogarty catheter is used to remove thrombotic material from the artery. Embolectomy is used mainly for mesenteric, femoral, or popliteal artery occlusion.
  - Laser angioplasty—Use of excision and hot tip lasers to vaporize the obstruction.
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- Lumbar sympathectomy—An adjunct to surgery, depending on the sympathetic nervous system's condition.
  - Patch grafting—This procedure involves removal of the thrombosed arterial segment and replacement with an autogenous vein or Dacron graft.
  - Stents—Insertion of a mesh of wires that stretch and mold to the arterial wall to prevent reocclusion. This new adjunct follows laser angioplasty or atherectomy.
  - Thromboendarterectomy—Opening of the occluded artery and direct removal of the obstructing thrombus and the medial layer of the arterial wall; usually performed after angiography and commonly used with autogenous vein or Dacron bypass surgery (femoral-popliteal or aortofemoral).
  - Thrombolytic therapy—Lysis of any clot around or in the plaque by urokinase, streptokinase, or alteplase.

## TYPES OF PERIPHERAL ARTERY DISEASE

Site of occlusion	Signs and symptoms
<b>Carotid arterial system</b> ▪ Internal carotids ▪ External carotids	<ul style="list-style-type: none"> <li>Absent or decreased pulsation with an auscultatory bruit over the affected vessels</li> <li>Neurologic dysfunction: transient ischemic attacks (TIAs) due to reduced cerebral circulation producing unilateral sensory or motor dysfunction (transient monocular blindness, and hemiparesis), possible aphasia or dysarthria, confusion, decreased mentation, and headache (These are recurrent features that usually last 5 to 10 minutes but may persist up to 24 hours and may herald a stroke.)</li> </ul>
<b>Vertebrobasilar system</b> ▪ Vertebral arteries ▪ Basilar arteries	<ul style="list-style-type: none"> <li>Neurologic dysfunction: TIAs of the brain stem and cerebellum producing binocular vision disturbances, vertigo, dysarthria, and “drop attacks” (falling down without loss of consciousness); less common than carotid TIA</li> </ul>
<b>Innominate</b> ▪ Brachiocephalic artery	<ul style="list-style-type: none"> <li>Indications of ischemia (claudication) of the right arm</li> <li>Neurologic dysfunction: signs and symptoms of vertebo-basilar occlusion</li> <li>Possible bruit over the right side of the neck</li> </ul>
<b>Subclavian artery</b>	<ul style="list-style-type: none"> <li>Clinical effects of vertebrobasilar occlusion and exercise-induced arm claudication</li> <li>Subclavian steal syndrome (characterized by the backflow of blood from the brain through the vertebral artery on the same side as the occlusion, into the subclavian artery distal to the occlusion)</li> <li>Possibly gangrene (usually limited to the digits)</li> </ul>
<b>Mesenteric artery</b> ▪ Superior (most commonly affected) ▪ Celiac axis ▪ Inferior	<ul style="list-style-type: none"> <li>Bowel ischemia, infarct necrosis, and gangrene</li> <li>Diarrhea</li> <li>Leukocytosis</li> <li>Nausea and vomiting</li> <li>Shock due to massive intraluminal fluid and plasma loss</li> <li>Sudden, acute abdominal pain</li> </ul>
<b>Aortic bifurcation</b> (saddle block occlusion, a medical emergency associated with cardiac embolization)	<ul style="list-style-type: none"> <li>Sensory and motor deficits (muscle weakness, numbness, paresthesias, and paralysis) in both legs</li> <li>Signs of ischemia (sudden pain and cold, pale legs with decreased or absent peripheral pulses) in both legs</li> </ul>
<b>Iliac artery</b> (Leriche's syndrome)	<ul style="list-style-type: none"> <li>Absent or reduced femoral or distal pulses</li> <li>Impotence</li> <li>Intermittent claudication of the lower back, buttocks, and thighs, relieved by rest</li> <li>Possible bruit over femoral arteries</li> </ul>
<b>Femoral and popliteal artery</b> (associated with aneurysm formation)	<ul style="list-style-type: none"> <li>Gangrene</li> <li>Intermittent claudication of the calves on exertion</li> <li>Ischemic pain in feet</li> <li>Leg pallor and coolness; blanching of the feet on elevation</li> <li>No palpable pulses in the ankles and feet</li> <li>Pretrophic pain (heralds necrosis and ulceration)</li> </ul>

Amputation becomes necessary with failure of arterial reconstructive surgery or with the development of gangrene, persistent infection, or intractable pain.

Other therapy includes heparin to prevent emboli (for embolic occlusion) and bowel resection after restoration of blood flow (for mesenteric artery occlusion).

### ***Special considerations***

- Provide comprehensive patient teaching, including proper foot care. Explain diagnostic tests and procedures. Advise the patient to stop smoking and to follow the prescribed medical regimen.

Preoperatively, during an acute episode:

- Assess the patient's circulatory status by checking for the most distal pulses and by inspecting his skin color and temperature.
- Provide pain relief as needed.
- Administer heparin by continuous I.V. drip as ordered. Use an infusion monitor or pump to ensure the proper flow rate.
- Wrap the patient's affected foot in soft cotton batting and reposition it frequently to prevent pressure on any one area. Strictly avoid elevating or applying heat to the affected leg.
- Watch for signs of fluid and electrolyte imbalance, and monitor intake and output for signs of renal failure (urine output less than 30 ml/hour).
- If the patient has carotid, innominate, vertebral, or subclavian artery occlusion, monitor him for signs of stroke, such as numbness in an arm or leg and intermittent blindness.

Postoperatively:

- Monitor the patient's vital signs. Continuously assess his circulatory function by inspecting skin color and temperature and by checking for distal pulses. In charting, compare earlier assessments and observations. Watch closely for signs of hemorrhage (tachycardia and hypotension) and check dressings for excessive bleeding.
- In carotid, innominate, vertebral, or subclavian artery occlusion, assess neurologic status frequently for changes in level of consciousness or muscle strength and pupil size.
- In mesenteric artery occlusion, connect the nasogastric tube to low intermittent suction. Monitor intake and output (low urine output may indicate damage to renal arteries during surgery). Check bowel sounds for return of peristalsis. Increasing abdominal distention and tenderness may indicate extension of bowel ischemia with resulting gangrene, necessitating further excision, or it may indicate peritonitis.
- In saddle block occlusion, check distal pulses for adequate circulation. Watch for signs of renal failure and mesenteric artery occlusion (severe abdominal pain), and for cardiac arrhythmias, which may precipitate embolus formation.
- In iliac artery occlusion, monitor urine output for signs of renal failure from decreased perfusion to the kidneys as a result of surgery. Provide meticulous catheter care.
- In both femoral and popliteal artery occlusions, assist with early ambulation, but discourage prolonged sitting.
- After amputation, check the patient's stump carefully for drainage and record its color and amount, and the time. Elevate the stump as ordered, and administer adequate analgesic medication. Because phantom limb pain is common, explain this phenomenon to the patient.
- When preparing the patient for discharge, instruct him to watch for signs of recurrence (pain, pallor, numbness, paralysis, and absence of pulse) that can result

from graft occlusion or occlusion at another site. Warn him against wearing constrictive clothing.

## Selected references

Alspach, J., ed. *AACN Core Curriculum for Critical Care Nursing*, 6th ed. Philadelphia: W.B. Saunders Co., 2006.