Chapter 4

Cardiovascular system

Just the facts

In this chapter, you'll learn:

- structures and functions of the cardiovascular system
- assessment of the cardiovascular system
- diagnostic tests and procedures for the cardiovascular system
- cardiovascular disorders and treatments.

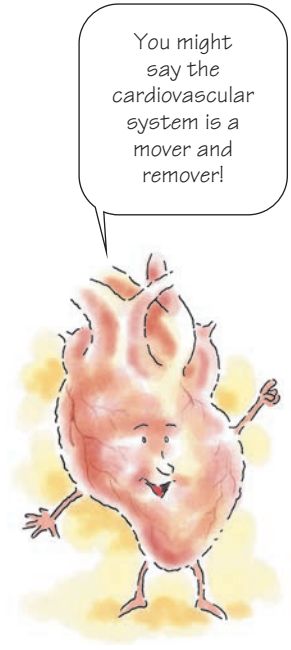
Understanding the cardiovascular system

The cardiovascular system consists of the heart and the blood vessels.

Bring it on . . . and take it away

This complex system functions to:

- carry life-sustaining oxygen and nutrients in the blood to all cells of the body
- remove metabolic waste products from the cells
- move hormones from one part of the body to another.



Heart

The heart is about the size of a closed fist. It lies beneath the sternum in the mediastinum (the cavity between the lungs), between the second and sixth ribs.

The right border of the heart aligns with the right border of the sternum. The left border aligns with the midclavicular line. The exact position of the heart varies slightly in each patient.

Pericardium

The pericardium is a sac that surrounds the heart. It's composed of an outer (fibrous) layer and an inner (serous) layer. The serous layer of the pericardium is composed of a visceral (inner) layer and a parietal (outer) layer.

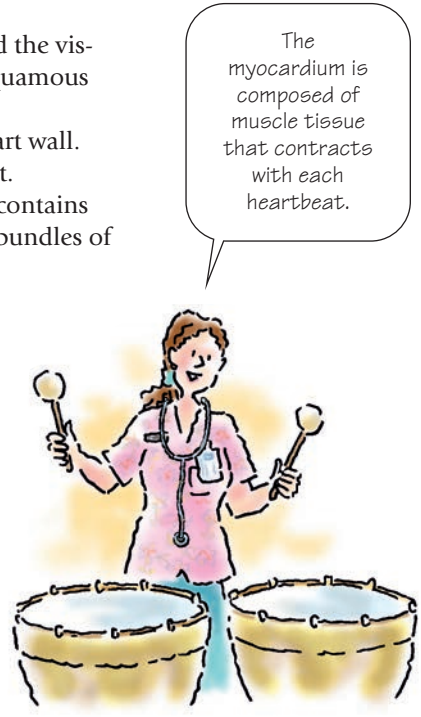
Liquid cushion

The pericardial space separates the visceral and parietal layers of the serous pericardium. This space contains 10 to 30 ml of thin, clear pericardial fluid, which lubricates the two surfaces of the serous pericardium and cushions the heart.

Heart wall

The heart's wall is composed of three layers:

1. *Epicardium* includes the outer layer of the heart wall and the visceral layer of the serous pericardium. It's made up of squamous epithelial cells overlying connective tissue.
2. *Myocardium* is the middle and largest portion of the heart wall. This layer of muscle tissue contracts with each heartbeat.
3. *Endocardium* is the innermost layer of the heart wall. It contains endothelial tissue made up of small blood vessels and bundles of smooth muscle.



Four chambers

The heart has four chambers:

- right atrium
- left atrium
- right ventricle
- left ventricle. (See *A close look at the heart.*)

Tanks for giving blood today!

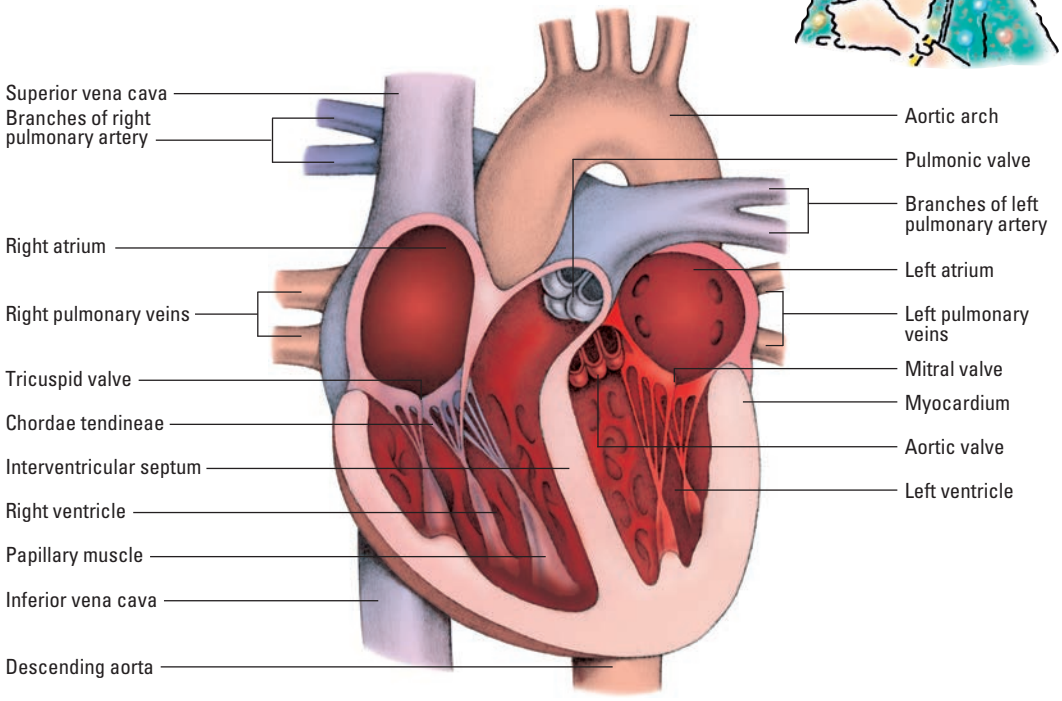
The right and left atria serve as reservoirs for blood. The right atrium receives deoxygenated blood returning from the body. The left atrium receives oxygenated blood from the lungs. Contraction of the atria forces blood into the ventricles below.

Powerful pumps

The right and left ventricles are the pumping chambers of the heart. The ventricles—which have thicker walls and are larger than the atria—are composed of highly developed muscles.

A close look at the heart

This illustration provides a detailed look at the internal structures of the heart.



The right ventricle receives blood from the right atrium and pumps it through the pulmonary arteries to the lungs, where it picks up oxygen and drops off carbon dioxide. The left ventricle receives oxygenated blood from the left atrium and pumps it through the aorta and then out to the rest of the body. The interventricular septum separates the ventricles and helps them to pump.

Heart valves

Valves in the heart keep blood flowing in one direction.

One way

Healthy valves open and close passively as a result of pressure changes in the four heart chambers. The valves prevent blood from traveling the wrong way.

Where the valves are

Valves between the atria and ventricles are called *atrioventricular (AV) valves* and include the tricuspid valve on the right side of the heart and the mitral valve on the left side. Valves between the ventricles and the pulmonary artery and the aorta are called *semilunar valves* and include the pulmonic valve on the right (between the right ventricle and the pulmonary artery) and the aortic valve on the left (between the left ventricle and the aorta).

On the cusp

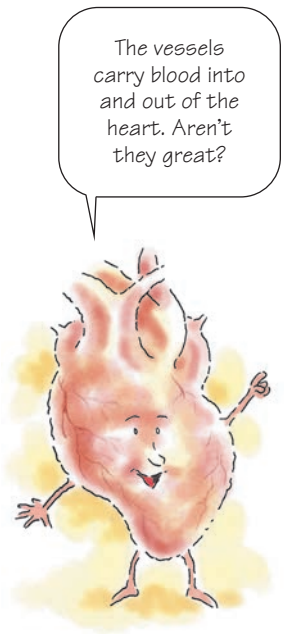
The leaflets, or cusps, of each valve keep the valves tightly closed. The tricuspid valve has three cusps. The mitral valve has two.

The cusps are anchored to the heart wall by cords of fibrous tissue called *chordae tendineae*, which are controlled by papillary muscles.

Great vessels

Leading into and out of the heart are the great vessels:

- The aorta, which carries oxygenated blood away from the left ventricle, is the main trunk of the systemic artery system.
- The inferior and superior venae cavae carry deoxygenated blood from the body into the right atrium.
- The pulmonary artery, the only artery that carries deoxygenated blood away from the heart, is a large artery that carries blood away from the right ventricle. Above the heart, it splits to form the right and left pulmonary arteries, which carry blood to the right and left lungs.
- The four pulmonary veins—two on the left and two on the right—carry oxygenated blood from the left and right lungs to the left atrium.



Coronary arteries

Like all other organs, the heart needs an adequate blood supply to survive. The coronary arteries, which lie on the surface of the heart, supply the heart muscle with blood and oxygen. (See *Heart vessels*.)

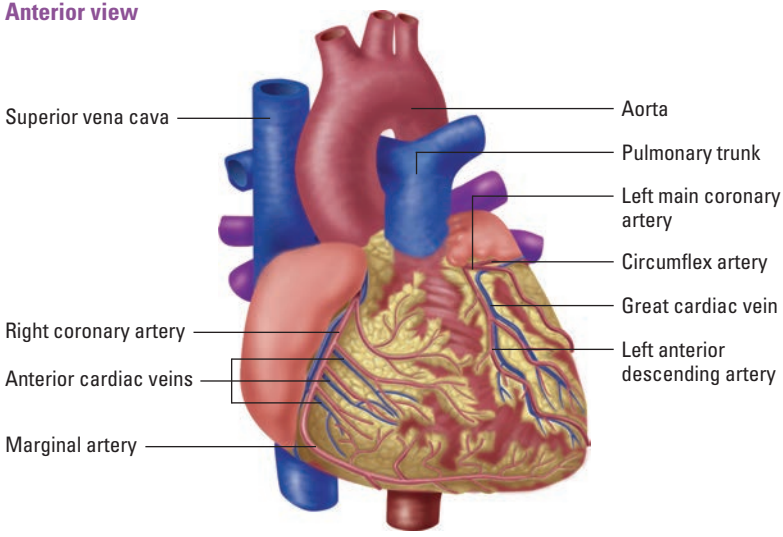
Coronary ostium

The coronary ostium is an opening in the aorta above the aortic valve. It feeds blood to the coronary arteries.

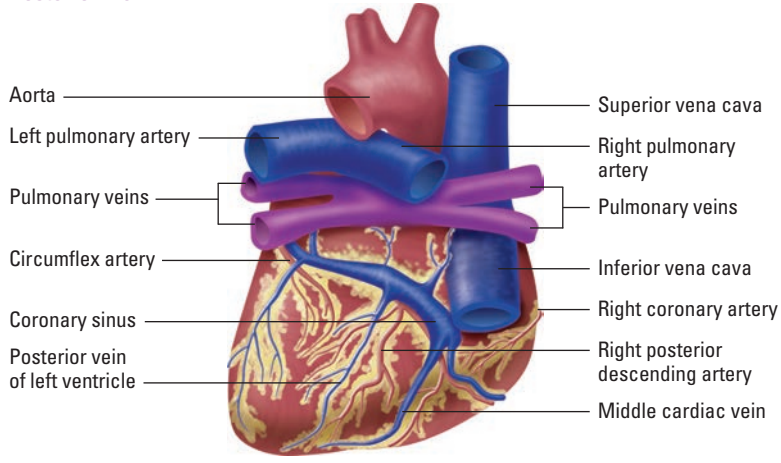
Heart vessels

These two views of the heart depict the great vessels and some of the major coronary vessels.

Anterior view



Posterior view



Ostium action

When the left ventricle is pumping blood through the aorta, the aortic valve is open and the coronary ostium is partly covered. When the left ventricle is filling with blood, the aortic valve is closed and the coronary ostium is open, enabling blood to fill the coronary arteries.

Right coronary artery

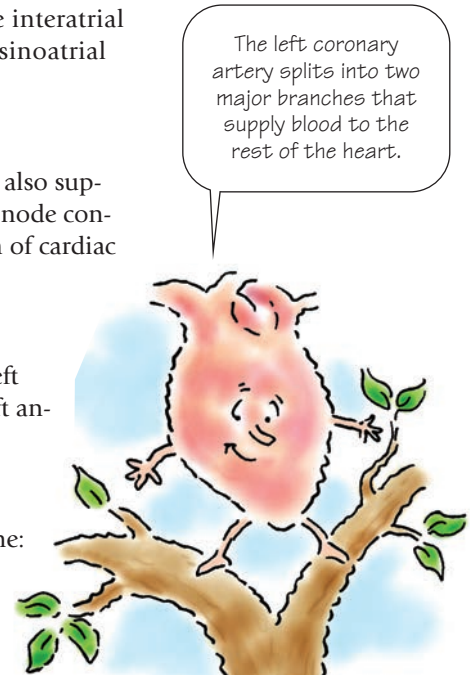
The right coronary artery supplies blood to the right atrium, the right ventricle, and part of the left ventricle. It also supplies blood to the bundle of His (muscles that connect the atria with the ventricles) and the AV node (fibers at the base of the interatrial septum that transmit the cardiac impulses from the sinoatrial [SA] node).

What do you SA about that?

In about half the population, the right coronary artery also supplies blood to the SA node of the right atrium. The SA node consists of atypical muscle fibers that establish the rhythm of cardiac contractions.

Left coronary artery

The left coronary artery runs along the surface of the left atrium, where it splits into two major branches: the left anterior descending artery and the left circumflex artery.



Left out

The left anterior descending artery supplies blood to the:

- anterior wall of the left ventricle
- interventricular septum
- right bundle branch (a branch of the bundle of His)
- left anterior fasciculus (small cluster) of the left bundle branch.

The branches of the left anterior descending artery—the septal perforators and the diagonal arteries—supply blood to the walls of both ventricles.

Circumflex-ability

The circumflex artery supplies oxygenated blood to the lateral walls of the left ventricle, the left atrium, and, in about 50% of the population, the SA node.

Circle left

In addition, the circumflex artery supplies blood to the left posterior fasciculus of the left bundle branch. This artery circles around the left ventricle and provides blood to the ventricle's posterior portion.

Veins

Like other parts of the body, the heart has veins, called *cardiac veins*, that collect deoxygenated blood from the capillaries of the myocardium. These cardiac veins join together to form an enlarged vessel called the *coronary sinus*. The right atrium receives deoxygenated blood from the heart through the coronary sinus.

Pulmonary circulation

During pulmonary circulation, blood travels to the lungs to pick up oxygen in exchange for carbon dioxide.

Heart to lungs to heart

Here's what happens during pulmonary circulation:

- Deoxygenated blood travels from the right ventricle through the pulmonary semilunar valve into the pulmonary arteries.
- Blood passes through smaller arteries and arterioles into the capillaries of the lungs.
- Blood reaches the alveoli and exchanges carbon dioxide for oxygen.
- Oxygenated blood returns through the venules and veins to the pulmonary veins.
- The pulmonary veins carry the oxygenated blood back to the left atrium of the heart.

Cardiac rhythm

Contractions of the heart occur in a rhythm that's regulated by impulses initiated at the SA node.

Nature's pacemaker

The SA node is the heart's pacemaker. Impulses initiated at the SA node are conducted from there throughout the heart. Impulses from the autonomic nervous system affect the SA node and alter its firing rate to meet the body's needs.

The cardiac cycle

The cardiac cycle consists of two phases: systole and diastole.

Out with systole, in with diastole

During systole, the ventricles contract and send blood on its outward journey. During diastole, the ventricles relax and fill with blood; the mitral and tricuspid valves are open, and the aortic and pulmonic valves are closed.

Filling and more filling

Diastole consists of ventricular filling and atrial contraction. During ventricular filling, 70% of the blood in the atria drains into the ventricles passively, by gravity. The active period of diastole, atrial contraction (also called *atrial kick*), accounts for the remaining 30% of blood that passes into the ventricles.

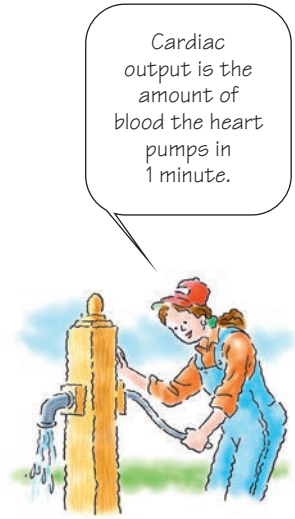
The pressure's on

When the pressure in the ventricles is greater than the pressure in the aorta and pulmonary artery, the aortic and pulmonic valves open. Blood then flows from the ventricles into the pulmonary artery, then to the lungs and into the aorta, and then to the rest of the body.

The pressure's off

At the end of ventricular contraction, pressure in the ventricles drops below the pressure in the aorta and pulmonary artery. The difference in pressure forces blood back up toward the ventricles and causes the aortic and pulmonic valves to snap shut.

As the valves shut, the atria fill with blood in preparation for the next period of diastolic filling, and the cycle begins again.



Out in a minute

Cardiac output is the amount of blood the heart pumps in 1 minute. It's equal to the heart rate multiplied by the stroke volume (the amount of blood ejected with each heartbeat).

Stroke volume depends on three major factors:

1. preload, the amount of blood volume the heart has to work with
2. afterload, the resistance the heart is working against
3. contractility. (See *Understanding preload, afterload, and contractility*.)

Understanding preload, afterload, and contractility

If you think of the heart as a balloon, it will help you understand preload, afterload, and contractility.

Blowing up the balloon

Preload is the stretching of muscle fibers in the ventricles. This stretching results from blood volume in the ventricles at end diastole. According to Starling's law, the more the heart muscles stretch during diastole, the more forcefully they contract during systole. Think of preload as the balloon stretching as air is blown into it. The more air, the greater the stretch.

The balloon's stretch

Contractility refers to the inherent ability of the myocardium to contract normally. Contractility is influenced by preload. The greater the stretch, the more forceful the contraction—or, the more air in the balloon, the greater the stretch, and the farther the balloon will fly when air is allowed to expel.

The knot that ties the balloon

Afterload refers to the pressure that the ventricular muscles must generate to overcome the higher pressure in the aorta to get the blood out of the heart. *Resistance* is the knot on the end of the balloon, which the balloon has to work against to get the air out.

Blood vessels

The vascular system is the complex network of blood vessels throughout the body that conducts systemic circulation. Blood carries oxygen and other nutrients to body cells and transports waste products for excretion.

Arteries

The major artery—the aorta—branches into vessels that supply blood to specific organs and areas of the body.

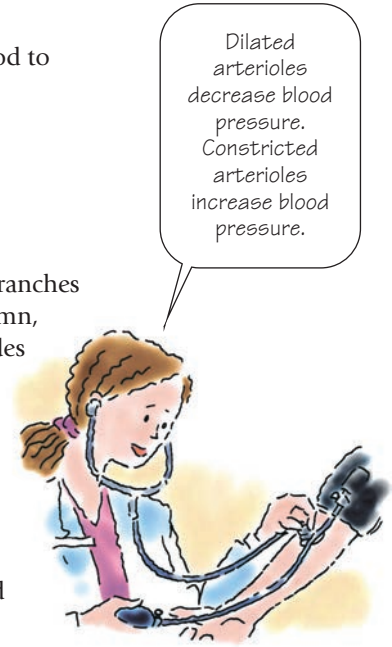
Upper blood suppliers

Three arteries arise from the arch of the aorta and supply blood to the brain, arms, and upper chest. These are the:

- left common carotid artery
- left subclavian artery
- brachiocephalic artery (also called the *innominate artery*).

Descending distribution

As the aorta descends through the thorax and abdomen, its branches supply blood to the GI and genitourinary organs, spinal column, and lower chest and abdominal muscles. Then the aorta divides into the iliac arteries, which further divide into the femoral arteries.



Arterioles

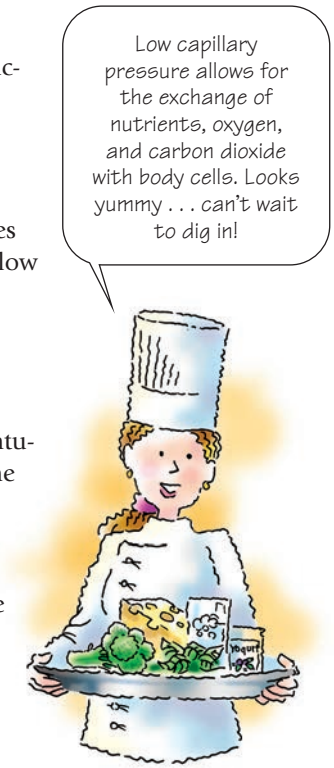
As the arteries divide into smaller units, the number of vessels increases, thereby increasing the area of perfusion. These smaller units, known as *arterioles*, can dilate to decrease blood pressure or constrict to increase blood pressure.

Capillaries

Where the arterioles end, the capillaries begin. Strong sphincters control blood flow from the capillaries into the tissues. The sphincters open to permit more flow when needed and close to shunt blood to other areas.

Small vessels, large area of distribution

Although the capillary bed contains the smallest vessels, it supplies blood to the largest area. Capillary pressure is extremely low to allow for exchange of nutrients, oxygen, and carbon dioxide with body cells.



Venules and veins

From the capillaries, returning blood flows into venules and, eventually, into veins. Valves in the veins prevent blood backflow, and the pumping action of skeletal muscles assists venous return.

Branching back to the right atrium

The veins merge until they form branches that return blood to the right atrium. The two main branches include the superior vena cava and the inferior vena cava.

Cardiovascular assessment

Assessment of a patient's cardiovascular system includes a health history and physical examination.

Health history

To obtain a health history of a patient's cardiovascular system, begin by introducing yourself and explaining what happens during the health history and physical examination. Then obtain the following information.

Chief complaint

Ask for details about the patient's chief complaint. Patients with cardiovascular problems typically cite specific complaints, including:

- chest pain
- irregular heartbeat or palpitations
- shortness of breath on exertion, when lying down, or at night
- cough
- weakness or fatigue
- unexplained weight change
- swelling of the extremities
- dizziness
- headache
- peripheral skin changes, such as decreased hair distribution, skin color changes, a thin shiny appearance to the skin, or an ulcer on the lower leg that fails to heal
- pain in the extremities, such as leg pain or cramps.

Personal and family health

Ask the patient for details about his family history and past medical history. Also ask about:

- stressors in the patient's life and coping strategies he uses to deal with them
- current health habits, such as smoking, alcohol intake, caffeine intake, exercise, and dietary intake of fat and sodium
- drugs the patient is taking, including prescription drugs, over-the-counter drugs, and herbal preparations
- previous surgeries
- environmental or occupational considerations
- activities of daily living (ADLs)
- menopause (if applicable).

Advice from the experts

Cardiac questions

To thoroughly assess your patient's cardiac function, be sure to ask these questions:

- Are you in pain?
- Where is the pain located?
- Does the pain feel like a burning, tight, or squeezing sensation?
- Does the pain radiate to your arm, neck, back, or jaw?
- When did the pain begin?
- What relieves or aggravates it?
- Are you experiencing nausea, dizziness, or sweating?
- Do you feel short of breath? Has breathing trouble ever awakened you from sleep?
- Does your heart ever pound or skip a beat? When?
- Do you ever get dizzy or faint? When?
- Do you experience swelling in your ankles or feet? When? Does anything relieve the swelling?
- Do you urinate frequently at night?
- Have you had to limit your activities?

Rating pain

Many patients with cardiovascular problems complain of chest pain. If the patient is experiencing chest pain, ask him to rate the pain on a scale of 0 to 10, in which 0 indicates no pain and 10 indicates the worst chest pain imaginable. It's vital to thoroughly assess pain.

Where, what, and why

If the patient isn't in distress, ask questions that require more than a yes-or-no response. Use familiar expressions rather than medical terms whenever possible. (See *Cardiac questions*.) The nurse can also utilize the PQRST method to help focus the cardiac pain assessment. The P stands for precipitating factors: what brings on the pain? The Q represents quality of pain: have the patient use descriptive words including tight or burning? The R stands for radiation: does the pain radiate, and where does it radiate? The S is the severity of the pain on the 0 to 10 scale as described above. Finally, the T is for treatments: what treatments has the patient tried to relieve the chest pain and are they effective?

In his own words

Let the patient describe his condition in his own words. Ask him to describe the location, radiation, intensity, and duration of pain and any precipitating, exacerbating, or relieving factors to obtain an accurate description of chest pain. (See *Understanding chest pain*.)

Understanding chest pain

Use this table to help you more accurately assess chest pain.

What it feels like	Where it's located	What makes it worse	What causes it	What makes it better
Aching, squeezing, pressure, heaviness, burning pain; usually subsides within 10 minutes	Substernal; may radiate to jaw, neck, arms, and back	Eating, physical effort, smoking, cold weather, stress, anger, hunger, lying down	Angina pectoris	Rest, nitroglycerin (*Note:* Unstable angina appears even at rest.)
Tightness or pressure; burning, aching pain, possibly accompanied by shortness of breath, diaphoresis, weakness, anxiety, or nausea; sudden onset; ½ hour to 2 hours	Typically across chest but may radiate to jaw, neck, arms, or back	Exertion, anxiety	Acute myocardial infarction (MI)	Opioid analgesics such as morphine, nitroglycerin
Sharp and continuous; may be accompanied by friction rub; sudden onset	Substernal; may radiate to neck, left arm, or back	Deep breathing (inspiration), supine position	Pericarditis	Sitting up, leaning forward, anti-inflammatory drugs
Excruciating, tearing pain; may be accompanied by blood pressure difference between right and left arm; sudden onset	Retrosternal, upper abdominal, or epigastric; may radiate to back, neck, or shoulders	Not applicable	Dissecting aortic aneurysm	Analgesics, surgery
Sudden, stabbing pain; may be accompanied by cyanosis, dyspnea, or cough with hemoptysis	Over lung area	Inspiration	Pulmonary embolus	Analgesics
Sudden and severe pain; sometimes accompanied by dyspnea, increased pulse rate, decreased breath sounds, or deviated trachea	Lateral thorax	Normal respiration	Pneumothorax	Analgesics, chest tube insertion

Physical examination

Cardiovascular disease affects people of all ages and can take many forms. To best identify abnormalities, use a consistent, methodical approach to the physical examination.

First things first

Before you begin the physical examination, wash your hands thoroughly. Obtain a stethoscope with a bell and a diaphragm, an appropriate-sized blood pressure cuff, and a penlight. Also, make sure the room is quiet.

Ask the patient to remove all clothing except his underwear and to put on an examination gown. Have the patient lie on his back, with the head of the bed at a 30- to 45-degree angle.

Memory jogger

To remember the order in which you should perform assessment of the cardiovascular system, just think, "**I**'ll **P**roperly **P**erform **A**ssessment."

Inspection

Palpation

Percussion

Auscultation

The heart of it

When performing an assessment of a patient's heart health, proceed in this order:

1. inspection
2. palpation
3. percussion
4. auscultation.

Inspection

First, take a moment to assess the patient's general appearance.

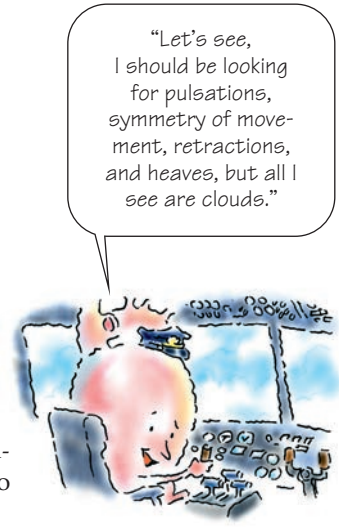
First impressions

Is the patient too thin or obese? Is he alert? Does he appear anxious? Note the patient's skin color. Are his fingers clubbed? (Clubbing is a sign of chronic hypoxia caused by a lengthy cardiovascular or respiratory disorder.) If the patient is dark-skinned, inspect his mucous membranes for pallor. The nurse can also inspect the skin noting if it is warm or dry or if the patient appears diaphoretic.

Check the chest

Next, inspect the chest. Note landmarks you can use to describe your findings as well as structures underlying the chest wall. (See *Cardiovascular landmarks*.)

Look for pulsations, symmetry of movement, retractions, or heaves (strong outward thrusts of the chest wall that occur during systole).



Inspecting the impulse

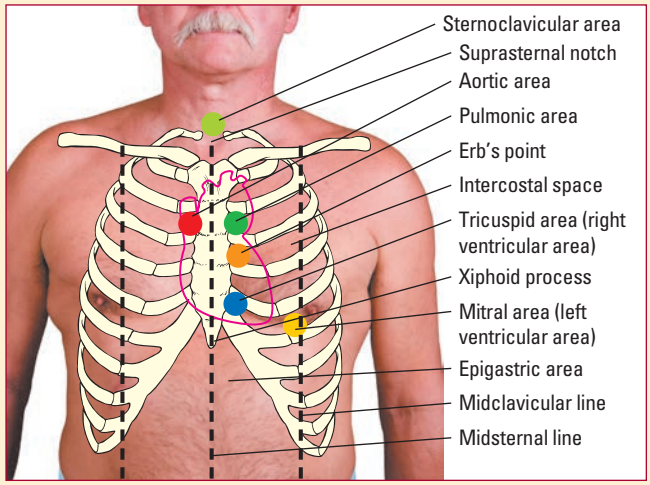
Then position a light source, such as a penlight, so that it casts a shadow on the patient's chest. Note the location of the apical impulse. This is also usually the point of maximal impulse (PMI) and should be located in the fifth intercostal space medial to the left midclavicular line.

The apical impulse gives an indication of how well the left ventricle is working because it corresponds to the apex of the heart. To

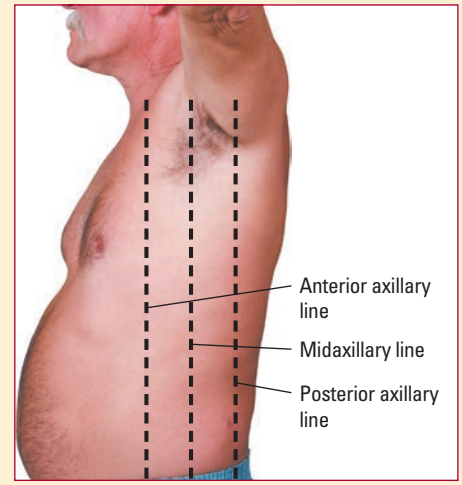
Cardiovascular landmarks

Here's a guide to finding the critical landmarks used in cardiovascular assessment.

Anterior thorax



Lateral thorax



find the apical impulse in a woman with large breasts, displace the breasts during the examination.

Abnormal findings on inspection

Here are some of the abnormal findings you may note on inspection and what such findings tell you:

- Inspection may reveal cyanosis, pallor, or cool or cold skin, which may indicate poor cardiac output and tissue perfusion.
- Skin may be flushed if the patient has a fever.
- Absence of body hair on the arms or legs may indicate diminished arterial blood flow to those areas. (See *Assessing arterial and venous insufficiency*, page 156.)
- Swelling, or edema, may indicate heart failure or venous insufficiency. It may also be caused by varicosities or thrombophlebitis.
- Chronic right-sided heart failure may cause ascites and generalized edema.

Edema is a telltale sign of possible heart failure, venous insufficiency, varicosities, or thrombophlebitis.

A chest of clues

- Inspection may reveal barrel chest (rounded thoracic cage caused by chronic obstructive pulmonary disease), scoliosis (lateral curvature of the spine), or kyphosis (convex curvature of the thoracic

Assessing arterial and venous insufficiency

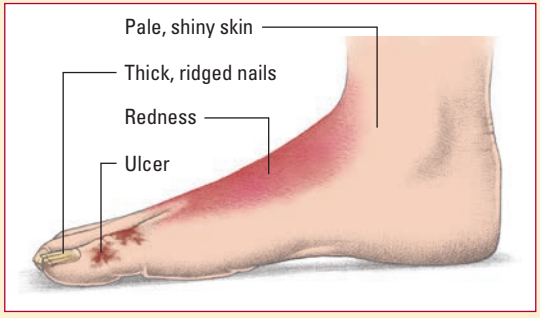
You should be aware of how assessment findings differ between healthy patients and those with arterial insufficiency or chronic venous insufficiency.

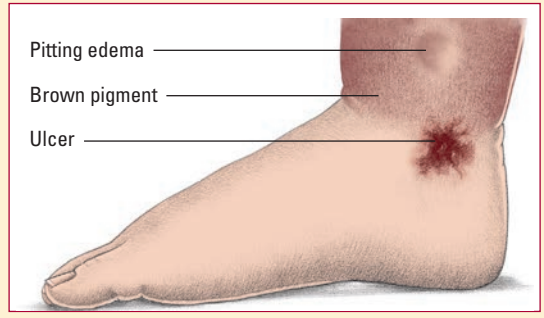
Arterial insufficiency

In a patient with arterial insufficiency, pulses may be decreased or absent. The skin is cool, pale, and shiny, and the patient may have pain in his legs and feet. Ulcerations typically occur in the area around the toes, and the foot usually turns deep red when dependent. Nails may be thick and ridged.

Chronic venous insufficiency

In a patient with chronic venous insufficiency, check for ulcerations around his ankle. Pulses are present but may be difficult to find because of pitting edema. The foot may become cyanotic when dependent, and you may see a brown pigmentation and thickening of the skin around the ankle.





spine). If severe enough, these conditions can impair cardiac output by preventing chest expansion and inhibiting heart muscle movement.

- Retractions (visible indentations of the soft tissue covering the chest wall) and the use of accessory muscles to breathe typically result from a respiratory disorder but may also occur with a congenital heart defect or heart failure.

Palpation

Note skin temperature, turgor, and texture. Using the ball of your hand and then your fingertips, gently palpate over the precordium to find the apical impulse. Note heaves or thrills (fine vibrations that feel like the purring of a cat). (See *Assessing apical impulse.*)

Elusive impulse

The apical impulse may be difficult to palpate in patients who are obese or pregnant and in patients with thick chest walls. If it's difficult to palpate with the patient lying on his back, have him lie on his left side or sit upright.

Plus, palpate

Also palpate the sternoclavicular, aortic, pulmonic, tricuspid, and epigastric areas for abnormal pulsations. Pulsations aren't usually felt in those areas. However, an aortic arch pulsation in the sternoclavicular area or an abdominal aorta pulsation in the epigastric area may be a normal finding in a thin patient.

Percussion

Percussion is less useful than other assessment methods, but it may help you locate the cardiac borders.

Border patrol

Begin percussing at the anterior axillary line and continue toward the sternum along the fifth intercostal space. The sound changes from resonance to dullness over the left border of the heart, normally at the midclavicular line. The right border of the heart is usually aligned with the sternum and can't be percussed.

Auscultation

You can learn a great deal about the heart by auscultating for heart sounds. Cardiac auscultation requires a methodical approach and lots of practice.

Here's the plan

First, identify the auscultation sites, which include the sites over the four cardiac valves, at Erb's point, and at the third intercostal space at the left sternal border. Use the bell to hear low-pitched sounds and the diaphragm to hear high-pitched sounds. (See *Heart sound sites*, page 158.)

Auscultate for heart sounds with the patient in three positions:

1. lying on his back with the head of the bed raised 30 to 45 degrees
2. sitting up
3. lying on his left side.

Upward, downward, zigward, zagward

Use a zigzag pattern over the precordium. Start at the apex and work upward or at the base and work downward. Whichever approach you use, be consistent.

Use the diaphragm to listen as you go in one direction; use the bell as you come back in the other direction. Be sure to listen over the entire precordium, not just over the valves. Note the patient's heart rate and rhythm.

Advice from the experts

Assessing apical impulse

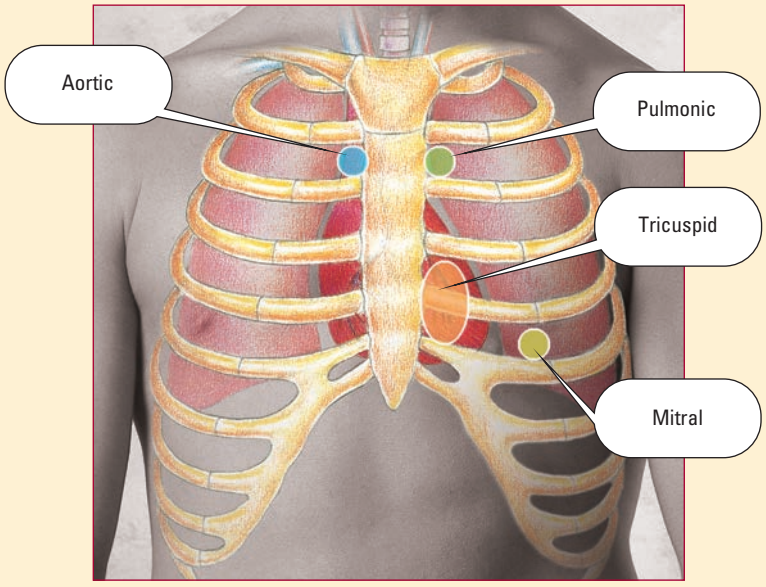
The apical impulse is associated with the first heart sound and carotid pulsation. To ensure that you're feeling the apical impulse and not a muscle spasm or some other pulsation, use one hand to palpate the patient's carotid artery and the other to palpate the apical impulse. Then compare the timing and regularity of the impulses. The apical impulse should roughly coincide with the carotid pulsation.

Note the amplitude, size, intensity, location, and duration of the apical impulse. You should feel a gentle pulsation in an area about ½" to ¾" (1.5 to 2 cm) in diameter.

Heart sound sites

When auscultating for heart sounds, place the stethoscope over the four different sites illustrated below.

Normal heart sounds indicate events in the cardiac cycle, such as the closing of heart valves, and are reflected to specific areas of the chest wall. Auscultation sites are identified by the names of heart valves but aren't located directly over the valves. Rather, these sites are located along the pathway blood takes as it flows through the heart's chambers and valves.



1, 2, 3, 4, and more

Systole is the period of ventricular contraction. As pressure in the ventricles increases, the mitral and tricuspid valves snap close. The closure produces the first heart sound, S_1.

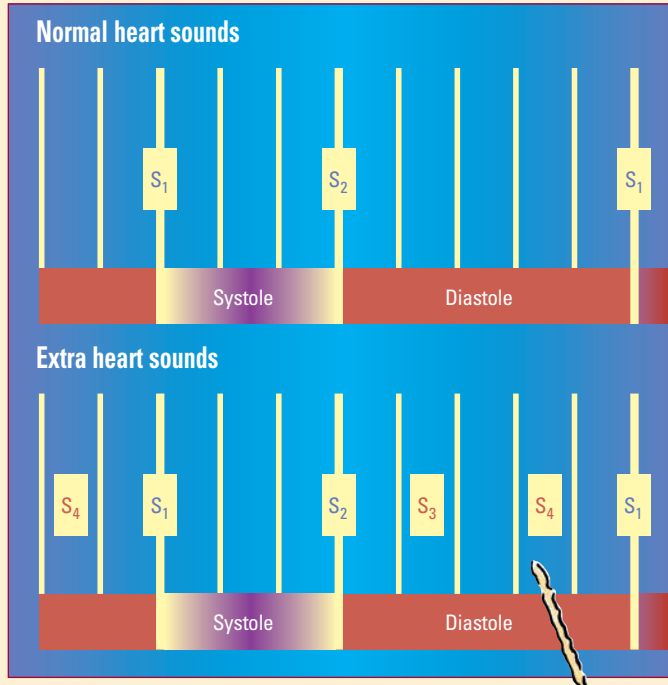
At the end of ventricular contraction, the aortic and pulmonic valves snap shut. This produces the second heart sound, S_2.

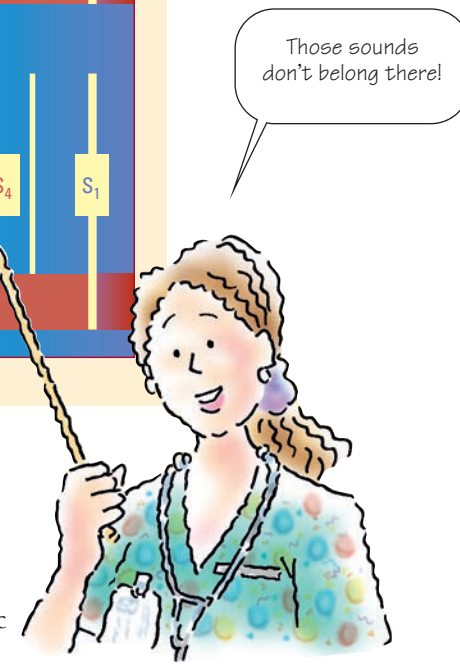
Always identify S_1 and S_2 and then listen for adventitious sounds, such as third and fourth heart sounds (S_3 and S_4). (See *Extra heart sounds in the cardiac cycle*.)

Also listen for murmurs (vibrating, blowing, or rumbling sounds) and rubs (harsh, scratchy, scraping, or squeaking sounds).

Extra heart sounds in the cardiac cycle

To understand where extra heart sounds fall in relation to systole, diastole, and normal heart sounds, compare the illustrations of normal and extra heart sounds below.





Listen for the "dub"

Start auscultating at the aortic area where the S_2 is loudest.

An S_2 is best heard at the base of the heart at the end of ventricular systole. It occurs when the pulmonic and aortic valves close and is generally described as sounding like "dub." It's a shorter, higher pitched, and louder sound than S_1.

When the pulmonic valve closes later than the aortic valve during inspiration, you hear a split S_2.

Listen for the "lub"

From the base of the heart, move to the pulmonic area and then down to the tricuspid area. Then move to the mitral area, where S_1 is the loudest.

An S_1 is best heard at the apex of the heart. It occurs with closure of the mitral and tricuspid valves and is generally described as sounding like "lub." It's low-pitched and dull.

An S_1 occurs at the beginning of ventricular systole. It may be split if the mitral valve closes just before the tricuspid valve.

Abnormal findings on heart auscultation

On auscultation, you may detect S_1 and S_2 heart sounds that are accentuated, diminished, or inaudible. Other abnormal heart sounds—such as S_3, S_4, and murmurs—may result from pressure changes, valvular dysfunctions, and conduction defects.

Third heart sound

The third heart sound—known as S_3 or *ventricular gallop*—is a low-pitched noise best heard by placing the bell of the stethoscope at the apex of the heart.

A ventricular gallop in a young person—even a 3-year-old filly—may be normal. But in someone older than age 30, it usually indicates a disorder.

Kentucky galloper

Its rhythm resembles a horse galloping, and its cadence resembles the word "Ken-tuc-ky" (lub-dub-by). Listen for S_3 with the patient in a supine or left lateral decubitus position.

An S_3 usually occurs during early diastole to middiastole, at the end of the passive-filling phase of either ventricle. Listen for this sound immediately after S_2. It may signify that the ventricle isn't compliant enough to accept the filling volume without additional force.

Age-related adversity

An S_3 may occur normally in a child or young adult. In a patient older than age 30, however, it usually indicates a disorder, such as:

- right-sided heart failure
- left-sided heart failure
- pulmonary congestion
- intracardiac shunting of blood
- MI
- anemia
- thyrotoxicosis
- mitral insufficiency
- tricuspid insufficiency.

Fourth heart sound

The fourth heart sound, or S_4, is an abnormal, low-frequency sound that occurs late in diastole, just before the pulse upstroke. It imme-

diately precedes the S_1 of the next cycle. It's known as the *atrial* or *presystolic gallop*, and it occurs during atrial contraction.

Tennessee walker

An S_4 shares the same cadence as the word "Ten-nes-see" (le-lub-dub). It's heard best on expiration with the bell of the stethoscope and with the patient in the supine position.

What S_4 says

An S_4 may indicate cardiovascular disease, such as:

- acute MI
- hypertension
- coronary artery disease (CAD)
- cardiomyopathy
- angina
- anemia
- elevated left ventricular pressure
- aortic stenosis
- left ventricular hypertrophy
- pulmonary hypertension
- pulmonary embolism.

If the S_4 sound persists, it may indicate impaired ventricular compliance or volume overload. S_4 commonly appears in elderly patients with age-related systolic hypertension and aortic stenosis.

Murmurs

A murmur, which is longer than a heart sound, makes a vibrating, blowing, or rumbling noise. Just as turbulent water in a stream babbles as it passes through a narrow point, turbulent blood flow produces a murmur.

If you detect a murmur, identify where it's loudest, pinpoint the time it occurs during the cardiac cycle, and describe its pitch, pattern, quality, and intensity. (See *Identifying heart murmurs*, page 162.)

Location and timing

Murmurs can occur in any cardiac auscultatory site and may radiate from one site to another.

Marking murmurs

To identify the radiation area, auscultate from the site where the murmur seems loudest to the farthest site it's still heard. Note the anatomic landmark of this farthest site.

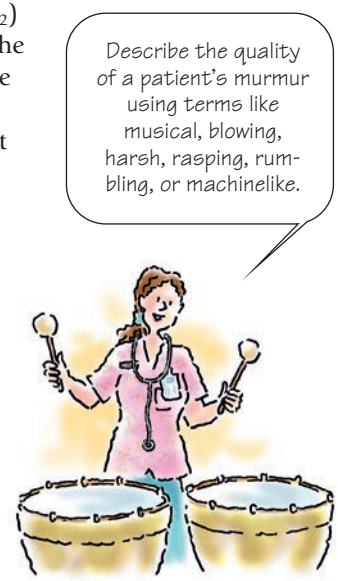
Identifying heart murmurs

To identify a heart murmur, first listen closely to determine its timing in the cardiac cycle. Then determine its other characteristics, including quality, pitch, and location as well as possible causes.

Timing	Quality and pitch	Location	Possible causes
Midsystolic (systolic ejection)	Harsh, rough with medium to high pitch	Pulmonic	Pulmonic stenosis
	Harsh, rough with medium to high pitch	Aortic and suprasternal notch	Aortic stenosis
Holosystolic (pansystolic)	Harsh with high pitch	Tricuspid	Ventricular septal defect
	Blowing with high pitch	Mitral, lower left sternal border	Mitral insufficiency
	Blowing with high pitch	Tricuspid	Tricuspid insufficiency
Early diastolic	Blowing with high pitch	Midleft sternal edge (not aortic area)	Aortic insufficiency
	Blowing with high pitch	Pulmonic	Pulmonic insufficiency
Middiastolic to late diastolic	Rumbling with low pitch	Apex	Mitral stenosis
	Rumbling with low pitch	Tricuspid, lower right sternal border	Tricuspid stenosis

Pinpoint its presence

Determine if the murmur occurs during systole (between S_1 and S_2) or diastole (between S_2 and the next S_1). Then pinpoint when in the cardiac cycle the murmur occurs—for example, during middiastole or late systole. A murmur heard throughout systole is called a *holosystolic* or *pansystolic* murmur, and a murmur heard throughout diastole is called a *pandiastolic* murmur. Occasionally, murmurs occur during both portions of the cycle (continuous murmur).



Pitch

Depending on the rate and pressure of blood flow, pitch may be high, medium, or low. A low-pitched murmur can be best heard with the bell of the stethoscope, a high-pitched murmur with the diaphragm, and a medium-pitched murmur with both.

Pattern

Crescendo occurs when the velocity of blood flow increases and the murmur becomes louder. Decrescendo occurs when velocity decreases and the murmur becomes quieter.

Up and down

A crescendo–decrescendo pattern describes a murmur with increasing loudness followed by increasing softness.

Quality

The volume of blood flow, the force of the contraction, and the degree of valve compromise all contribute to murmur quality. Terms used to describe quality include *musical*, *blowing*, *harsh*, *rasping*, *rumbling*, or *machinelike*.

Intensity

Use a standard, six-level grading scale to describe the intensity of the murmur:

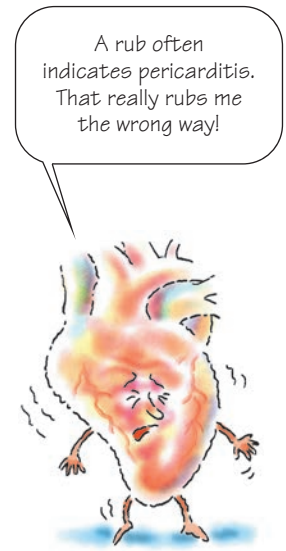
1. grade I—extremely faint; barely audible even to the trained ear
2. grade II—soft and low; easily audible to the trained ear
3. grade III—moderately loud; about equal to the intensity of normal heart sounds
4. grade IV—loud with a palpable thrill at the murmur site
5. grade V—very loud with a palpable thrill; audible with the stethoscope in partial contact with the chest
6. grade VI—extremely loud, with a palpable thrill; audible with the stethoscope over, but not in contact with, the chest.

Rubs

To detect a pericardial friction rub, use the diaphragm of the stethoscope to auscultate in the third left intercostal space along the lower left sternal border.

Rubs the wrong way

Listen for a harsh, scratchy, scraping, or squeaking sound that occurs throughout systole, diastole, or both. To enhance the sound, have the patient sit upright and lean forward or exhale. A rub usually indicates pericarditis, but it may also occur in infections or neoplasms or after cardiac surgery.



Assessing the vascular system

Assessment of the vascular system is an important part of a full cardiovascular assessment.

Inspection

Start your assessment of the vascular system the same way you start an assessment of the cardiac system—by making general observations.

To arms! . . . and legs!

Examination of the patient's arms and legs can reveal arterial or venous disorders. Examine the patient's arms when you take his vital signs. Are the arms equal in size? Evaluate the legs when the patient is standing. Are the legs symmetrical? Check the legs later during the physical examination as well, with the patient lying on his back.

Skimming the skin

Inspect the patient's skin color. Note how body hair is distributed. Note lesions, scars, clubbing, and edema of the extremities. If the patient is confined to bed, be sure to check the sacrum for swelling. Examine the fingernails and toenails for abnormalities.

Checking the neck

Continue your inspection by observing vessels in the neck. Inspection of these vessels can provide information about blood volume and pressure in the right side of the heart.

Picturing pulsations

Check the carotid artery pulsations. The carotid artery should have a brisk, localized pulsation—not weak or bounding. The carotid pulsation doesn't decrease when the patient is upright, when he inhales, or when you palpate the carotid artery.

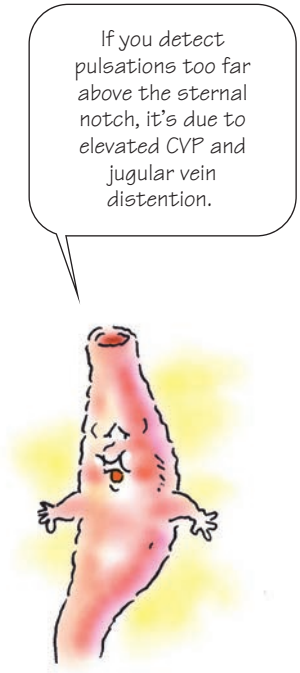
Inspect the jugular veins. The internal jugular vein has a softer, undulating pulsation. The internal jugular pulsation changes in response to position, breathing, and palpation.

Going for the jugular

To check the jugular venous pulse, have the patient lie on his back. Elevate the head of the bed 30 to 45 degrees and turn the patient's head slightly away from you.

Pulsations a notch above the notch

Normally, the highest pulsation occurs no more than ½" (4 cm) above the sternal notch. If pulsations appear higher, it indicates elevation in central venous pressure (CVP) and jugular vein distention.



Palpation

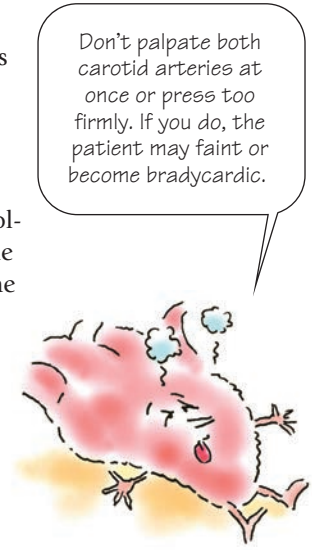
The first step in palpation is to assess skin temperature, texture, and turgor.

Note nail beds

Next, check capillary refill by assessing the nail beds on the fingers and toes. Refill time should be no more than 3 seconds or long enough to say "capillary refill."

Palpate pulses

Palpate for the pulse on each side of the neck, comparing pulse volume and symmetry. Don't palpate both carotid arteries at the same time or press too firmly. If you do, the patient may faint or become bradycardic.



Making the grade

All pulses should be regular in rhythm and equal in strength. Pulses are graded on a scale from 0 to 4+:

- 4+ is bounding.
- 3+ is increased.
- 2+ is normal.
- 1+ is weak.
- 0 is absent.

Abnormal findings

Abnormal findings on palpation may reveal:

- weak pulse, indicating low cardiac output or increased peripheral vascular resistance such as in arterial atherosclerotic disease (Note that elderly patients commonly have weak pedal pulses.)
- strong bounding pulse, which occurs in hypertension and in high cardiac output states, such as exercise, pregnancy, anemia, and thyrotoxicosis
- apical impulse that exerts unusual force and lasts longer than one-third of the cardiac cycle—a possible indication of increased cardiac output
- displaced or diffuse impulse, which is a possible indication of left ventricular hypertrophy
- pulsation in the aortic, pulmonic, or right ventricular area, which is a sign of chamber enlargement or valvular disease
- pulsation in the sternoclavicular or epigastric area, which is a sign of an aortic aneurysm
- palpable thrill, which is an indication of blood flow turbulence and is usually related to valvular dysfunction; determine how far the thrill radiates and make a mental note to listen for a murmur at this site during auscultation
- heave along the left sternal border, which is an indication of right ventricular hypertrophy
- heave over the left ventricular area, which is a sign of a ventricular aneurysm; a thin patient may experience a heave with exercise,

fever, or anxiety because of increased cardiac output and more forceful contraction
- displaced PMI, which is a possible indication of left ventricular hypertrophy caused by volume overload from mitral or aortic stenosis, septal defect, acute MI, or other disorder.

Percussion

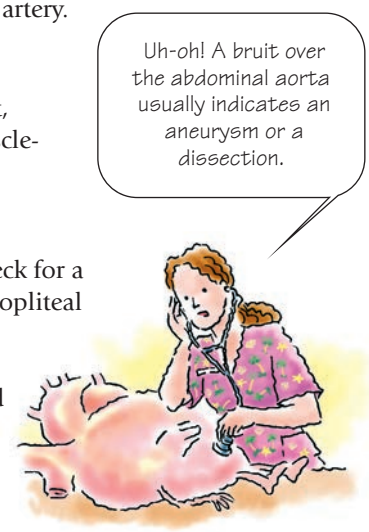
Percussion isn't used when assessing the vascular system.

Auscultation

After vascular palpation, use the bell of the stethoscope to begin auscultation. Follow the palpation sequence and listen over each artery.

Abnormal findings

Sounds aren't normally heard over the carotid arteries. A bruit, which sounds like buzzing or blowing, could indicate arteriosclerotic plaque formation.



Brutish bruits

When you auscultate for the femoral and popliteal pulses, check for a bruit or other abnormal sounds. A bruit over the femoral or popliteal artery usually indicates narrowed vessels.

During auscultation of the central and peripheral arteries, you may notice a continuous bruit, caused by turbulent blood flow. A bruit over the abdominal aorta usually indicates an aneurysm (weakness in the arterial wall that allows a sac to form) or a dissection (a tear in the layers of the arterial wall).

Diagnostic tests

Advances in diagnostic testing allow for earlier and easier diagnosis and treatment of cardiovascular disorders. For example, in some patients, echocardiography—a noninvasive and risk-free test—can provide as much diagnostic information on valvular heart disease as can cardiac catheterization—an invasive and high-risk test.

12-Lead electrocardiogram

The 12-lead electrocardiogram (ECG) measures the heart's electrical activity and records it as waveforms. It's one of the most valuable and commonly used diagnostic tools.

A test with 12 views

The standard 12-lead ECG uses a series of electrodes placed on the patient's extremities and chest wall to assess the heart from 12 different views (leads). The 12 leads include three bipolar limb leads (I, II, and III), three unipolar augmented limb leads (aV_R, aV_L, and aV_F), and six unipolar precordial limb leads (V_1 to V_6). The limb leads and augmented leads show the heart from the frontal plane. The precordial leads show the heart from the horizontal plane. If the patient presents to the emergency department with chest pain, the ECG needs to be done right away!

ECG can be used to identify myocardial ischemia and infarction, rhythm and conduction disturbances, chamber enlargement, electrolyte imbalances, and drug toxicity.

In addition to the 12-lead ECG, two other ECGs may be used for diagnostic purposes, the right chest-lead ECG and posterior-lead ECG. (See *Understanding right chest-lead and posterior-lead ECGs*.)

Nursing considerations

- Use a systematic approach to interpret the ECG recording. (See *Normal ECG waveforms*, page 168.) Compare the patient's previous ECG with the current one, if available. This will help you identify changes.

Understanding right chest-lead and posterior-lead ECGs

The right chest-lead ECG and posterior-lead ECG use chest leads to assess areas that standard 12-lead ECGs can't.

Checking out the right chest

The usual 12-lead ECG evaluates only the left ventricle. If the right ventricle needs to be assessed for damage or dysfunction, the doctor may order a right chest-lead ECG. For example, a patient with an inferior wall MI might have a right chest-lead ECG to rule out right ventricular involvement.

With this type of ECG, the six chest leads are placed on the right side of the chest in a mirror image of the standard precordial lead placement. Electrodes start at the left sternal border and swing down the right side of the breast area.

Seeing behind your back

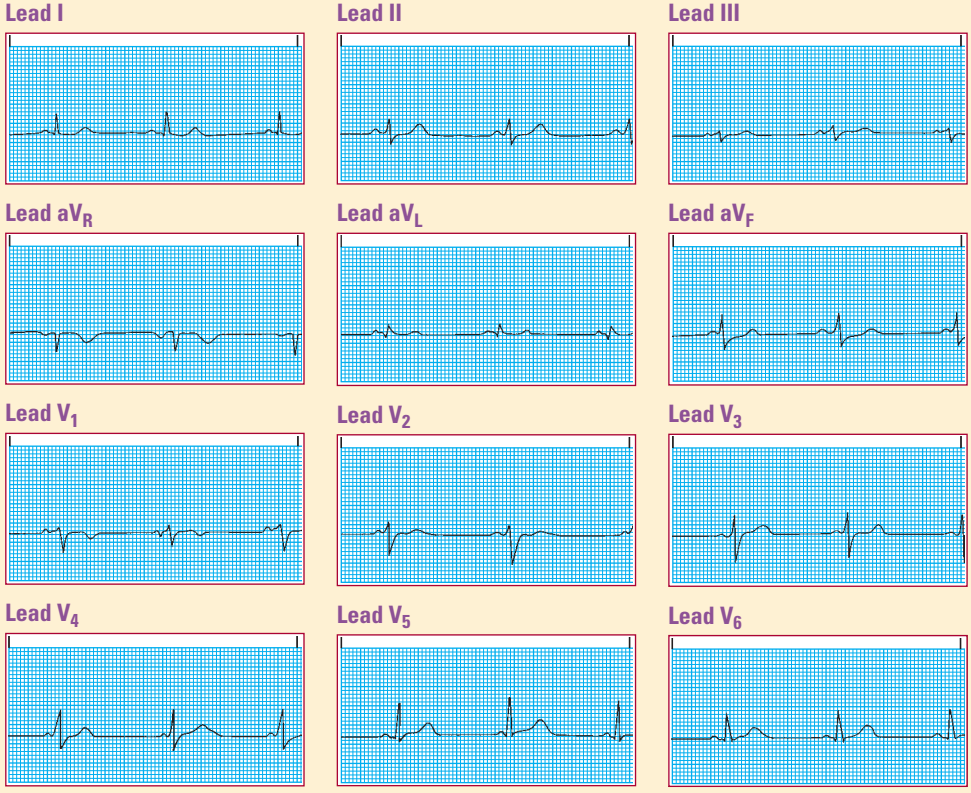
Because of lung and muscle barriers, the usual chest leads can't "see" the heart's posterior surface to record myocardial damage there. Some doctors add three posterior leads to the 12-lead ECG: leads V_7, V_8, and V_9. These leads are placed opposite the anterior leads V_4, V_5, and V_6 on the left side of the patient's back following the same horizontal line. V_7 is placed at the posterior axillary line, lead V_9 at the paraspinal line, and lead V_8 halfway between leads V_7 and V_9.

Normal ECG waveforms

Each of the 12 standard leads of an ECG takes a different view of heart activity, and each generates its own characteristic tracing. The tracings shown here represent a normal heart rhythm viewed from each of the 12 leads. Keep these facts in mind:

- An upward (positive) deflection indicates that the wave of depolarization flows toward the positive electrode.
- A downward (negative) deflection indicates that the wave of depolarization flows away from the positive electrode.
- An equally positive and negative (biphasic) deflection indicates that the wave of depolarization flows perpendicularly to the positive electrode.

Each lead represents a picture of a different anatomic area; when you find abnormal tracings, compare information from the different leads to pinpoint areas of cardiac damage.



Waves of waves

- P waves should be upright; however, they may be inverted in lead aV_R or biphasic or inverted in leads III, aV_L, and V_1.
- A P wave should be present and look the same before each QRS complex.
- PR intervals should always be constant, just like QRS-complex durations.
- QRS-complex deflections vary in different leads. Observe for pathologic Q waves.
- ST segments should be isoelectric or have minimal deviation.
- ST-segment elevation greater than 1 mm above the baseline and ST-segment depression greater than 0.5 mm below the baseline are considered abnormal. Leads facing an injured area have ST-segment elevations, and leads facing away show ST-segment depressions.
- The T wave normally deflects upward in leads I, II, and V_3 through V_6. It's inverted in lead aV_R and variable in the other leads. T-wave changes have many causes and aren't always a reason for alarm. Excessively tall, flat, or inverted T waves occurring with symptoms, such as chest pain, may indicate ischemia.
- A normal Q wave generally has a duration less than 0.04 second. An abnormal Q wave has either a duration of 0.04 second or more, a depth greater than 4 mm, or a height one-fourth of the R wave. Abnormal Q waves indicate myocardial necrosis, developing when depolarization can't follow its normal path because of damaged tissue in the area.
- Remember that aV_R normally has a large Q wave, so disregard this lead when searching for abnormal Q waves.

Holter monitor

This test is used to detect suspected dysrhythmias. The patient is connected to a small portable recorder with 3 to 5 electrodes. The recorder is worn for 24 to 48 hours, and the patient will engage in normal activities keeping a log of anytime symptoms are felt. The recordings are then analyzed for abnormalities with the documented activities and symptoms.

Cardiac marker studies

Analysis of cardiac markers (proteins) aids diagnosis of acute MI.

Release the enzymes!

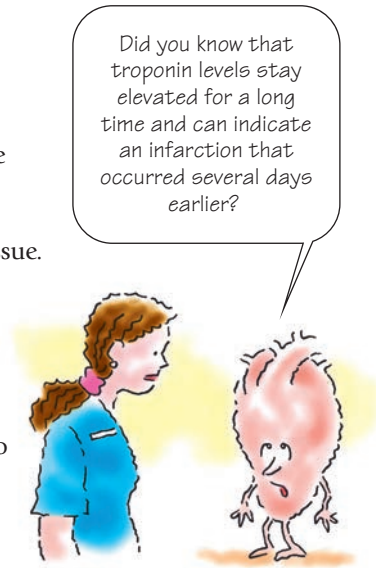
After infarction, damaged cardiac tissue releases significant amounts of enzymes into the blood. Serial measurement of enzyme levels reveals the extent of damage and helps to monitor the progress of healing.

Heart-zymes

The cardiac enzymes include creatine kinase (CK) and its isoenzyme MB (found specifically in heart muscle).

Tests for troponin T and I and myoglobin are more specific to cardiac muscle and can be used to detect damage more quickly, allowing faster and more effective treatment.

- Ischemia modified albumin (IMA) measures changes in serum albumin when it comes in contact with ischemic tissue. IMA rises faster than any other cardiac enzyme.



Meaning in markers

Here's what the results of cardiac marker studies mean:

- CK-MB levels increase 4 to 8 hours after the onset of acute MI, peak after 20 hours, and may remain elevated for up to 72 hours.
- Troponin levels increase within 3 to 6 hours after myocardial damage. Troponin I peaks in 14 to 20 hours, with a return to baseline in 5 to 7 days. Troponin T peaks in 12 to 24 hours, with a return to baseline in 10 to 15 days. Because troponin levels stay elevated for a long time, they can be used to detect an infarction that occurred several days earlier.
- Myoglobin levels may increase within 30 minutes to 4 hours after myocardial damage, peak within 6 to 7 hours, and return to baseline after 24 hours. However, because skeletal muscle damage may cause myoglobin levels to increase, it isn't specific to myocardial injury.
- IMA levels rise within minutes of myocardial ischemia. Levels peak in 6 hours and return to baseline within 12 hours. IMA levels are best interpreted when used in conjunction with troponin, myoglobin, and CK-MB levels.

Nursing considerations

- Before CK measurement, withhold alcohol, aminocaproic acid (Amicar), and lithium (Eskalith) as ordered. If the patient must continue taking these substances, note this on the laboratory request. Inform your patient that serial blood tests are necessary.
- Avoid administering I.M. injections because they can cause muscle damage and elevate some cardiac markers.
- After any cardiac enzyme test, handle the collection tube gently to prevent hemolysis and send the sample to the laboratory immediately. A delay can affect test results.

Echocardiography

Echocardiography is used to examine the size, shape, and motion of cardiac structures. It's done using a transducer placed at an acoustic window (an area where bone and lung tissue are absent) on the patient's chest. The transducer directs sound waves toward cardiac structures, which reflect these waves.

Echo, echo

The transducer picks up the echoes, converts them to electrical impulses, and relays them to an echocardiography machine for display on a screen and for recording on a strip chart or videotape. The most commonly used echocardiographic techniques are M-mode and two-dimensional.

Motion mode

In M-mode (motion mode) echocardiography, a single, pencil-like ultrasound beam strikes the heart, producing an "ice pick," or vertical, view of cardiac structures. This mode is especially useful for precisely viewing cardiac structures.

Echo in 2-D

In two-dimensional echocardiography, the ultrasound beam rapidly sweeps through an arc, producing a cross-sectional, or fan-shaped, view of cardiac structures; this technique is useful for recording lateral motion and providing the correct spatial relationship between cardiac structures. In many cases, both techniques are performed to complement each other.

TEE combination

In TEE, ultrasonography is combined with endoscopy to provide a better view of the heart's structures. (See *A closer look at TEE*.)

Echo abnormalities

The echocardiogram may detect mitral stenosis, mitral valve prolapse, aortic insufficiency, wall motion abnormalities, and pericardial effusion (excess pericardial fluid).

Nursing considerations

- Explain the procedure to the patient and advise him to remain still during the test because movement can distort results. Tell him that conductive gel is applied to the chest, and a quarter-sized transducer is placed directly over it. Because pressure is exerted to

A closer look at TEE

In transesophageal echocardiography (TEE), ultrasonography is combined with endoscopy to provide a better view of the heart's structures. Similar to other endoscopic procedures, a TEE will require the patient to have conscious sedation.

How it's done

A small transducer is attached to the end of a gastroscope and inserted into the esophagus so that images of the heart's structure can be taken from the posterior of the heart. This test causes less tissue penetration and interference from chest wall structures and produces high-quality images of the thoracic aorta (except for the superior ascending aorta, which is shadowed by the trachea).

And why

TEE is used to diagnose:

- thoracic and aortic disorders
- endocarditis
- congenital heart disease
- intracardiac thrombi
- tumors.

It's also used to evaluate valvular disease or repairs.

keep the transducer in contact with the skin, warn the patient that he may feel minor discomfort.
- After the procedure, remove the conductive gel from the skin.

Stress Testing

Exercise stress testing

This is a noninvasive test in which the patient is connected to an ECG while exercising. As physical stress causes an increase in myocardial oxygen consumption, ischemia may result showing changes to the ECG. If ischemia is noted during the test, the provider should stop the test.

Pharmacologic stress testing

If a patient is physically unable to perform an exercise stress test, a pharmacologic stress test can be done. This is done with radionuclide echocardiography. Medications are used because they cause vasodilation of normal coronary arteries, which will show ischemia if stenosis is occurring.

Cardiac magnetic resonance imaging

Magnetic resonance imaging (MRI) is a noninvasive test that evaluates tissues, structures, and blood flow. The images from the MRI are fed into a computer that reconstructs the image that will differentiate between healthy and ischemic tissue. It can be used to diagnose CAD, aortic aneurysm, congenital heart disease, left ventricular function, cardiac tumors, thrombus, valvular disease, and pericardial disorders. MRI is contraindicated in patients with pacemakers, defibrillators, brain clips, and cochlear implants.

Cardiac catheterization

Cardiac catheterization involves passing a catheter into the right, left, or both sides of the heart.

A multipurpose procedure

Cardiac catheterization permits measurement of blood pressure and blood flow in the chambers of the heart. It also allows the doctor to visualize the coronary arteries and determine the

presence of any narrowing or occlusions. It's used to determine valve competence and cardiac wall contractility and to detect intracardiac shunts.

Left sided catheterization is completed to visualize the coronary arteries and note extent of lesions within native vessels as well as bypass grafts. Balloon catheter treatments, such as angioplasty or placement of a coronary stent, may be done with cardiac catheterization.

Right-sided catheterization is performed by placing a pulmonary artery (PA) catheter in the femoral or brachial vein and then advancing it into the right atrium, ventricle, and pulmonary artery. This enables the health care provider to measure pressures in the right atrium, pulmonary artery, and also the pulmonary artery occlusion pressure.

The procedure also enables collection of blood samples and taking of diagnostic films of the ventricles (contrast ventriculography) and arteries (coronary arteriography or angiography).

Cardiac calculations

Use of thermodilution catheters allows calculation of cardiac output. Such calculations are used to evaluate valvular insufficiency or stenosis, septal defects, congenital anomalies, myocardial function and blood supply, and heart wall motion.

Confirming common problems

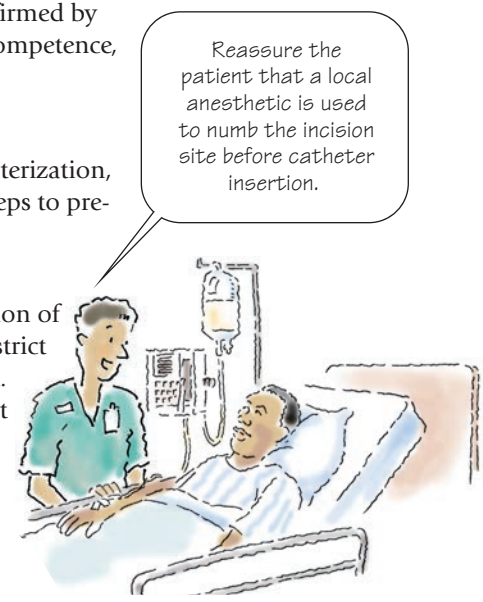
Common abnormalities and defects that can be confirmed by cardiac catheterization include CAD, myocardial incompetence, valvular heart disease, and septal defects.

Nursing considerations

When caring for a patient undergoing a cardiac catheterization, describe the procedure and events after it and take steps to prevent postoperative complications.

Before the procedure

- Explain that this test is used to evaluate the function of the heart and its vessels. Instruct the patient to restrict food and fluids for at least 6 hours before the test. Tell him the procedure takes 1 to 2 hours and that he may receive a mild sedative during the procedure.
- Tell the patient that the catheter is inserted into an artery or vein in the arm or leg. Tell him he'll experience a transient stinging sensation when a



Cardiac catheterization complications

Cardiac catheterization carries more patient risk than most other diagnostic tests. Although infrequent, complications can become life-threatening. Observe the patient carefully during the procedure. Notify the practitioner promptly and carefully document complications such as those listed here.

Left- or right-sided catheterization

- Cardiac tamponade
- Arrhythmias
- Hematoma or blood loss at insertion site
- Hypovolemia
- Infection (systemic or local)
- MI
- Pulmonary edema
- Reaction to contrast medium

Left-sided catheterization

- Arterial embolus or thrombus in limb
- Stroke or transient ischemic attack

Right-sided catheterization

- Pulmonary embolism
- Thrombophlebitis
- Vagal response
- Vagus nerve endings irritated in SA node, atrial muscle tissue, or atrioventricular junction

local anesthetic is injected to numb the incision site for catheter insertion.

- Inform the patient that injection of the contrast medium through the catheter may produce a hot, flushing sensation or nausea that quickly passes; instruct him to follow directions to cough or breathe deeply. Explain that medication will be given if he experiences chest pain during the procedure and that he may also be given nitroglycerin (Nitrostat) periodically to dilate coronary vessels and aid visualization. Reassure him that complications, such as MI and thromboembolism, are rare. (See *Cardiac catheterization complications*, page 174.)
- Make sure that the patient or a responsible family member has signed a consent form. Check for and tell the practitioner about hypersensitivity to shellfish, iodine, or contrast media used in other diagnostic tests.
- The patient may require anticoagulant therapy to be discontinued to reduce the risk for complications from bleeding.
- Contrast dye needed for the procedure can result in a decline in kidney function. Check the patient's renal function tests (blood urea nitrogen [BUN] and creatinine) and notify the practitioner of abnormalities.
- Make sure the patient has two patent I.V. access sites.

- Review activity restrictions that may be required of the patient after the procedure, such as lying flat with the limb extended for 4 to 6 hours and use of sandbags, if a femoral sheath is used.
- Document the presence of peripheral pulses, noting their intensity. Mark the pulses so they may be easily located after the procedure.

After the procedure

- Determine if a hemostatic device, such as a collagen plug or suture closure system, was used to close the vessel puncture site. A hemostatic bandage may also be used, and there are other commercial devices, including the FemoStop, which can help with pressure for the first 15 to 30 minutes. With any method, inspect the site for bleeding or oozing, redness, swelling, or hematoma formation. Maintain the patient on bed rest for 1 to 2 hours.
- Enforce bed rest for 8 hours if no hemostatic device was used. If the femoral route was used for catheter insertion, keep the patient's leg extended for 6 to 8 hours; if the antecubital fossa route was used, keep the arm extended for at least 3 hours.
- Monitor vital signs every 15 minutes for 2 hours, then every 30 minutes for the next 2 hours, and then every hour for 4 hours. If no hematoma or other problems arise, check every 4 hours. If signs are unstable, check every 5 minutes and notify the practitioner.
- Continually assess the insertion site for a hematoma or blood loss and reinforce the pressure dressing as needed.
- Check the patient's color, skin temperature, and peripheral pulse below the puncture site.
- Administer I.V. fluids as ordered (usually 100 ml/hour) to promote excretion of the contrast medium. Monitor for signs of fluid overload.
- Watch for signs of chest pain, shortness of breath, abnormal heart rate, dizziness, confusion, diaphoresis, nausea or vomiting, or extreme fatigue. Notify the practitioner immediately if these complications occur.

Electrophysiology studies

Electrophysiology studies (EPS) are used to diagnose and treat abnormal heart rhythms. The procedure involves passing two to four temporary electrode catheters into multiple heart chambers. The

electrodes are usually positioned in the right atrium, the AV node, the bundle of His region, and the apex of the right ventricle. The electrodes stimulate (pace) the heart and record the heart's electrical conduction.

Normal conduction intervals in adults are as follows: HV interval (measured from the earliest onset of the bundle of His deflection to the earliest registered surface or intracardiac ventricular activation), 35 to 55 msec; AH interval (represents the interval from the earliest rapid deflection of the atrial recording to the earliest onset of the bundle of His deflection), 45 to 150 msec; and PA interval (measured from the onset of the earliest registered surface P wave to the onset of the atrial deflection on the bundle of His catheter recording), 20 to 60 msec.

Nursing considerations

- Explain to the patient that EPS evaluate the heart's conduction system. Instruct him to restrict food and fluids for at least 6 hours before the test. Inform him that the studies take 1 to 3 hours.
- Have the patient void before the test.
- Monitor the patient's vital signs, as ordered. If they're unstable, check them every 15 minutes and alert the doctor. Observe for shortness of breath, chest pain, pallor, or changes in pulse rate, cardiac rhythm, or blood pressure. Enforce bed rest for 4 to 6 hours.
- Check the catheter insertion site for bleeding; apply a pressure bandage and sandbag to the site until bleeding stops.

Hemodynamic monitoring

Hemodynamic monitoring is used to assess cardiac function and determine the effectiveness of therapy by measuring:

- cardiac output
- mixed venous blood
- oxygen saturation
- intracardiac pressures
- blood pressure. (See *Putting hemodynamic monitoring to use*.)

The methods behind the monitoring

Follow your facility's procedure for setting up, zero referencing, calibrating, maintaining, and troubleshooting equipment. Common uses of hemodynamic monitoring include arterial blood pressure

Putting hemodynamic monitoring to use

Hemodynamic monitoring provides information on intracardiac pressures, arterial pressure, and cardiac output. To understand intracardiac pressures, picture the heart and vascular system as a continuous loop with constantly changing pressure gradients that keep the blood moving. Hemodynamic monitoring records the gradients within the vessels and heart chambers. Cardiac output indicates the amount of blood ejected by the heart each minute.

Pressure and description	Normal values	Causes of increased pressure	Causes of decreased pressure
Central venous pressure or right atrial pressure The CVP or right atrial pressure shows right ventricular function and end-diastolic pressure.	Normal mean pressure ranges from 1 to 6 mm Hg (1.34 to 8 cm H_2O).	• Right-sided heart failure • Volume overload • Tricuspid valve stenosis or insufficiency • Constrictive pericarditis • Pulmonary hypertension • Cardiac tamponade • Right ventricular infarction	• Reduced circulating blood volume • Vasodilation
Right ventricular pressure Typically, the doctor measures right ventricular pressure only when initially inserting a PA catheter. Right ventricular systolic pressure normally equals pulmonary artery systolic pressure; right ventricular end-diastolic pressure, which reflects right ventricular function, equals right atrial pressure.	Normal systolic pressure ranges from 20 to 30 mm Hg; normal diastolic pressure, from 0 to 5 mm Hg.	• Mitral stenosis or insufficiency • Pulmonary disease • Hypoxemia • Constrictive pericarditis • Chronic heart failure • Atrial and ventricular septal defects • Patent ductus arteriosus	• Reduced circulating blood volume • Vasodilation
Pulmonary artery pressure Pulmonary artery systolic pressure shows right ventricular function and pulmonary circulation pressures. Pulmonary artery diastolic pressure reflects left ventricular pressures, specifically left ventricular end-diastolic pressure, in a patient without significant pulmonary disease.	Systolic pressure normally ranges from 20 to 30 mm Hg. The mean pressure usually ranges from 10 to 15 mm Hg.	• Left-sided heart failure • Increased pulmonary blood flow (left or right shunting, as in atrial or ventricular septal defects) • Any condition causing increased pulmonary arteriolar resistance (such as pulmonary hypertension, volume overload, mitral stenosis, or hypoxia)	• Reduced circulating blood volume • Vasodilation
Pulmonary artery wedge pressure Pulmonary artery wedge pressure (PAWP) reflects left atrial and left ventricular pressures, unless the patient has mitral stenosis. Changes in PAWP reflect changes in left ventricular filling pressure.	The mean pressure normally ranges from 6 to 12 mm Hg.	• Left-sided heart failure • Mitral stenosis or insufficiency • Pericardial tamponade	Reduced circulating blood volume

Understanding minimally and noninvasive hemodynamic monitoring

Minimally and noninvasive hemodynamic monitoring techniques are easier to use and have been shown to provide reproducible, valid results. Minimally invasive techniques include esophageal Doppler hemodynamic monitoring and arterial pressure-based cardiac output (APCO) monitoring. Impedance cardiography is a noninvasive alternative for tracking hemodynamic status.

Esophageal Doppler hemodynamic monitoring

Esophageal Doppler hemodynamic monitoring uses ultrasound to measure heart function. It involves placement of a probe into the esophagus. By measuring blood flow through the heart valves or ventricular outflow tracks, this monitoring system can monitor:

- cardiac output (CO)
- stroke volume (SV)
- cardiac index (CI)
- systemic vascular resistance (SVR)
- SVR index.

This type of monitoring is appropriate for sedated critically ill patients with difficult fluid management or for use during and after cardiac surgery.

Transducer probe placement for esophageal Doppler hemodynamic monitoring is similar to inserting a nasogastric (NG) or orogastric tube and typically can be performed at the bedside. When the probe is positioned properly, it's ready to measure blood flow in the descending thoracic arch. The normal waveform should show good capture of blood flow. The monitor automatically measures such values as heart rate, peak velocity (PV), and flow time corrected (FTc). Other hemodynamic monitoring parameters are then derived from these measurements including CO, CI, SV, SV index, and SVR.

Arterial pressure-based cardiac output monitoring

In APCO, a patient's existing arterial catheter is used to continuously calculate and display CO. The arterial catheter and line are connected to a sensor, transducer, and specialized monitor preprogrammed with a clinically validated algorithm for determining CO.

Three devices are currently available. One system requires that the patient's age, gender, height, and weight be entered into the computer but no external calibration. Two others require an external calibration method. APCO is very useful in helping to determine a patient's fluid status and his potential response to a fluid challenge before he has significant changes in blood pressure.

Factors that may interfere with APCO include:

- incorrect leveling of transducer and sensor
- incorrect zeroing
- intra-aortic balloon pump (IABP)
- arrhythmias
- artificial heart or ventricular assist device (VAD)
- dampened pressure waveforms
- air bubbles in the fluid line.

Impedance cardiography

Impedance cardiography provides a noninvasive alternative for tracking hemodynamic status. This technique provides information about a patient's CI, preload, afterload, contractility, CO, and blood flow by measuring low-level electricity that flows harmlessly through the body from electrodes placed on the patient's thorax. These electrodes detect signals elicited from the changing volume and velocity of blood flow through the aorta. The signals are interpreted by the impedance monitor as a waveform. CO is computed from this waveform and the ECG.

Impedance cardiography monitoring eliminates the patient's risk for infection, bleeding, pneumothorax, emboli, and arrhythmias associated with traditional invasive hemodynamic monitoring. The accuracy of results obtained by this method is comparable to that obtained by thermodilution. In addition, the impedance cardiography monitor automatically updates information every 2nd to 10th heartbeat, providing real-time data.

monitoring, CVP monitoring, and pulmonary artery pressure (PAP) monitoring.

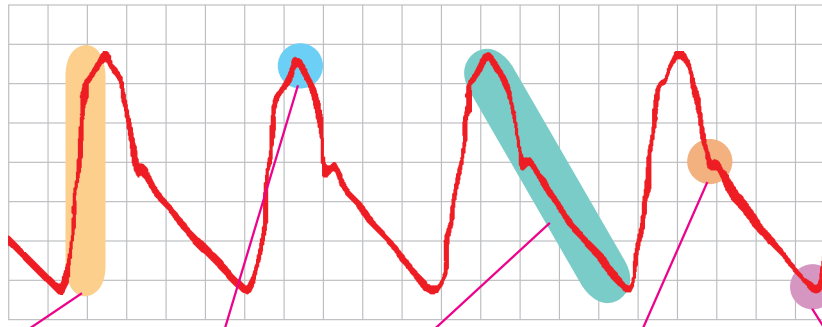
Minimally and noninvasive hemodynamic monitoring techniques are also proving to be reliable, safe options that are easier to use and can be applied in many clinical settings. (See *Understanding minimally and noninvasive hemodynamic monitoring*.)

Arterial blood pressure monitoring

In arterial blood pressure monitoring, the practitioner inserts a catheter into the radial or femoral artery to measure blood pressure or obtain samples of arterial blood for diagnostic tests such as arterial blood gas (ABG) studies.

A transducer transforms the flow of blood during systole and diastole into a waveform, which appears on an oscilloscope. The waveform has five distinct components. (See *Normal arterial waveform*.)

Normal arterial waveform



Anacrotic limb
The *anacrotic limb* marks the waveform's initial upstroke, which occurs as blood is rapidly ejected from the ventricle through the open aortic valve into the aorta.

Systolic peak
Arterial pressure then rises sharply, resulting in the *systolic peak*—the waveform's highest point.

Dicrotic limb
As blood continues into the peripheral vessels, arterial pressure falls and the waveform begins a downward trend, called the *dicrotic limb*. Arterial pressure usually keeps falling until pressure in the ventricle is less than the pressure in the aortic root.

Dicrotic notch
When ventricular pressure is lower than aortic root pressure, the aortic valve closes. This event appears as a small notch on the waveform's downside, called the *dicrotic notch*.

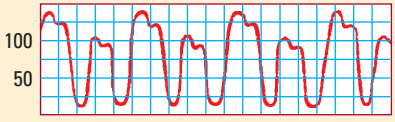
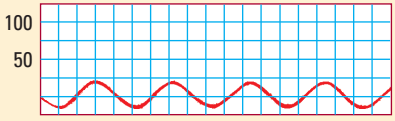
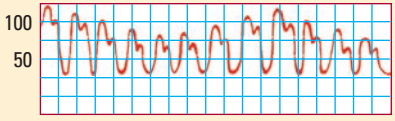
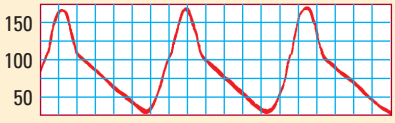
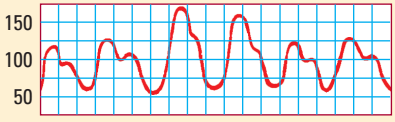
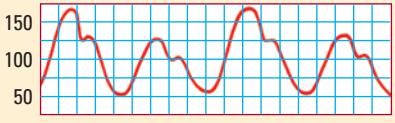
End diastole
When the aortic valve closes, diastole begins, progressing until aortic root pressure gradually falls to its lowest point. On the waveform, this is known as *end diastole*.

Nursing considerations

- Explain the procedure to the patient and his family, including the purpose of arterial pressure monitoring.
- After catheter insertion, observe the pressure waveform to assess arterial pressure. (See *Recognizing abnormal arterial waveforms*.)
- The four major components of validating the accuracy of the hemodynamic arterial line are patient positioning, zeroing the transducer, leveling the air–fluid interface to the phlebostatic axis, and assessing dynamic responsiveness with the square wave test.

Recognizing abnormal arterial waveforms

Use this chart to help you recognize and resolve waveform abnormalities.

Waveform	Abnormality	Possible causes
	Alternating high and low waves in a regular pattern	Ventricular bigeminy
	Flattened waveform	Overdamped waveform or hypotensive patient
	Slightly rounded waveform with consistent variations in systolic height	Patient on ventilator with positive end-expiratory pressure
	Slow upstroke	Aortic stenosis
	Diminished amplitude on inspiration	Pulsus paradoxus, possibly from cardiac tamponade, constrictive pericarditis, or lung disease
	Alteration in beat-to-beat amplitude (in otherwise normal rhythm)	Pulsus alternans, which may indicate left ventricular failure

- Assess the insertion site for signs of infection, such as redness and swelling. Notify the practitioner immediately if you note such signs.
- Ensure all connections are tightly closed and stopcock is in the correct position to prevent arterial bleeding
- Maintain 300 mm Hg pressure in the pressure bag to allow a flush flow of 3 to 6 ml per hour.
- Document the date and time of catheter insertion, catheter insertion site, type of flush solution used, type of dressing applied, and patient's tolerance of the procedure.

Nursing interventions

- Check the patient's ECG to confirm ventricular bigeminy. The tracing should reflect premature ventricular contractions (PVCs) every second beat.

- Check the patient's blood pressure with a sphygmomanometer. If you obtain a higher reading, suspect overdamping. Correct the problem by trying to aspirate the arterial line. If you succeed, flush the line. If the reading is very low or absent, suspect hypotension.

- Check the patient's systolic blood pressure regularly. The difference between the highest and lowest systolic pressure reading should be less than 10 mm Hg. If the difference exceeds that amount, suspect pulsus paradoxus, possibly from cardiac tamponade.

- Check the patient's heart sounds for signs of aortic stenosis. Also notify the practitioner, who will document suspected aortic stenosis in his notes.

- Note systolic pressure during inspiration and expiration. If inspiratory pressure is at least 10 mm Hg less than expiratory pressure, call the practitioner.
- If you're also monitoring PAP, observe for a diastolic plateau. This abnormality occurs when the mean CVP (right atrial pressure), mean PAP, and mean PAWP (pulmonary artery obstructive pressure) are within 5 mm Hg of one another.

- Observe the patient's ECG, noting any deviation in the waveform.
- Notify the practitioner if this is a new and sudden abnormality.

Central venous pressure

In CVP monitoring, the doctor inserts a catheter through a vein and advances it until its tip lies in or near the right atrium. Because no major valves lie at the junction of the vena cava and right atrium, pressure at end diastole reflects back to the catheter. In critically ill patients, when connected to the transducer or manometer, the catheter measures CVP, an index of right ventricular function, and central venous blood volume.

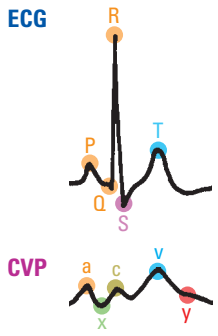
Nursing considerations

- Explain the procedure, including the purpose of CVP monitoring, to the patient and his family.
- After catheter insertion, observe the waveform to assess CVP. (See *Recognizing abnormal CVP waveforms*.)

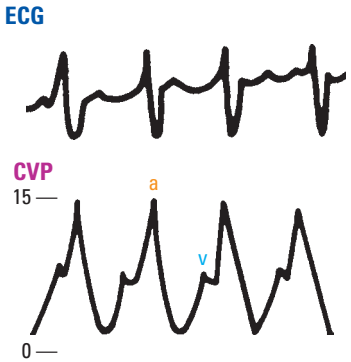
Recognizing abnormal CVP waveforms

These illustrations show a normal CVP waveform and abnormal CVP waveforms, along with possible causes of abnormal waveforms.

Normal CVP waveform



Elevated *a* wave



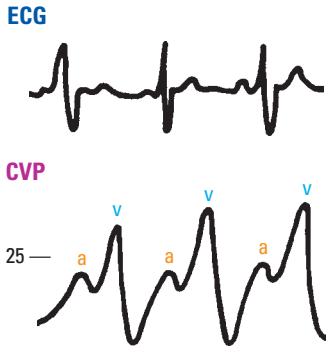
Physiologic causes

- Increased resistance to ventricular filling
- Increased atrial contraction

Associated conditions

- Heart failure
- Tricuspid stenosis
- Pulmonary hypertension

Elevated *v* wave



Physiologic cause

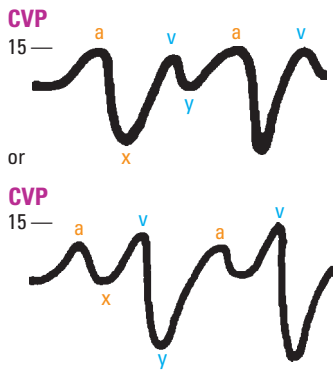
- Regurgitant flow

Associated conditions

- Tricuspid insufficiency
- Inadequate closure of the tricuspid valve due to heart failure

- Monitor the patient for infection. The U.S. Centers for Disease Control and Prevention estimates that 250,000 catheter-related bloodstream infections occur annually within the United States.
- Monitor for complications including pneumothorax, air embolism, and thrombosis. Notify the practitioner immediately if you notice such complications.
- Adhere to your facility's policy for dressing, tubing, catheter, and flush changes; use caution to prevent infection when changing dressing, tubing, and catheters.
- Document the date and time of catheter insertion, catheter insertion site, type of flush solution used, type of dressing applied, and patient's tolerance of the procedure.
- Document the CVP per your facility's policy or as ordered.

Elevated *a* and *v* waves



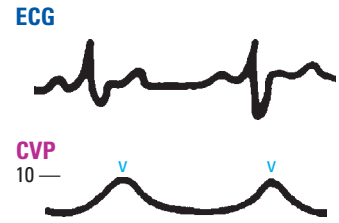
Physiologic causes

- Increased resistance to ventricular filling, which causes an elevated *a* wave
- Functional regurgitation, which causes an elevated *v* wave

Associated conditions

- Cardiac tamponade (smaller *y* descent than *x* descent)
- Constrictive pericardial disease (*y* descent exceeds *x* descent)
- Heart failure
- Hypervolemia
- Atrial hypertrophy

Absent *a* wave



Physiologic cause

- Decreased or absent atrial contraction

Associated conditions

- Atrial fibrillation
- Junctional arrhythmias
- Ventricular pacing

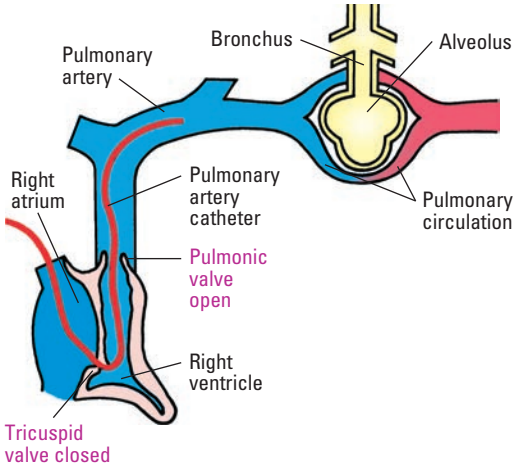
Understanding pulmonary artery pressures

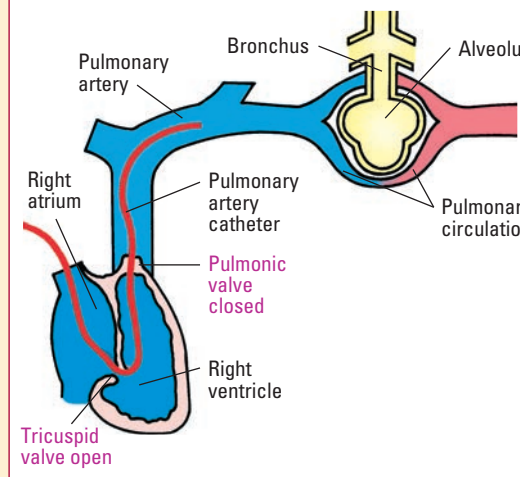
PA systolic pressure

PA systolic pressure measures right ventricular systolic ejection or, simply put, the amount of pressure needed to open the pulmonic valve and eject blood into the pulmonary circulation. When the pulmonic valve is open, PA systolic pressure should be the same as right ventricular pressure.

PA diastolic pressure

PA diastolic pressure represents the resistance of the pulmonary vascular bed as measured when the pulmonic valve is closed and the tricuspid valve is open. To a limited degree (under absolutely normal conditions), PA diastolic pressure also reflects left ventricular end-diastolic pressure.





Pulmonary artery pressure monitoring

Continuous PAP and intermittent PAWP measurements provide important information about left ventricular function and preload. (See *Understanding pulmonary artery pressures*.) Use this information for monitoring and for aiding diagnosis, refining assessment, guiding interventions, and projecting patient outcomes.

PAP purposes

PAP monitoring is indicated for patients who:

- are hemodynamically unstable
- need fluid management or continuous cardiopulmonary assessment
- are receiving multiple or frequently administered cardioactive drugs.

PAP monitoring is also crucial for patients experiencing shock, trauma, pulmonary or cardiac disease, or multiple organ dysfunction syndrome.

PAP's parts

A PA catheter has up to six lumens that gather hemodynamic information. In addition to distal and proximal lumens used to measure pressures, a PA catheter has a balloon inflation lumen that inflates the balloon for PAWP measurement and a thermistor connector lumen that allows cardiac output measurement.

Some catheters also have a pacemaker wire lumen that provides a port for pacemaker electrodes and measures continuous mixed venous oxygen saturation. (See *PA catheter ports*.)

PAP and PAWP procedures

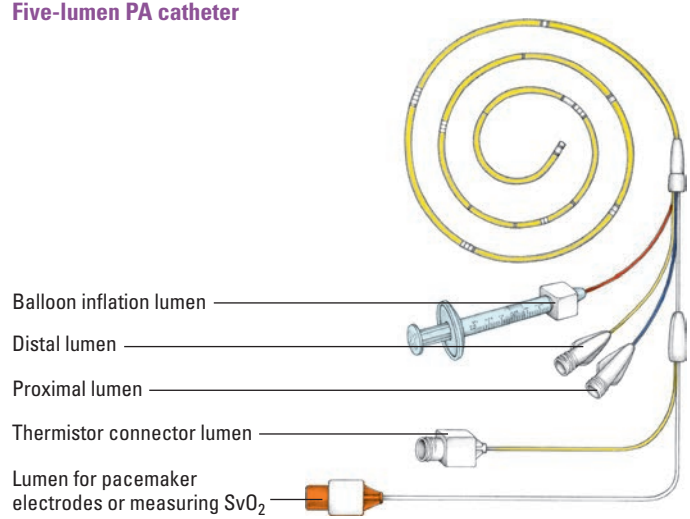
The doctor inserts the balloon-tipped, multilumen catheter into the patient's internal jugular or subclavian vein. When the catheter reaches the right atrium, the balloon is inflated to float the catheter through the right ventricle into the pulmonary artery. This permits PAWP measurement through an opening at the catheter's tip. Thermodilution PA catheters that have the ability to obtain PAPs and cardiac output measurements are now considered the gold standard to hemodynamic monitoring.

PA catheter ports

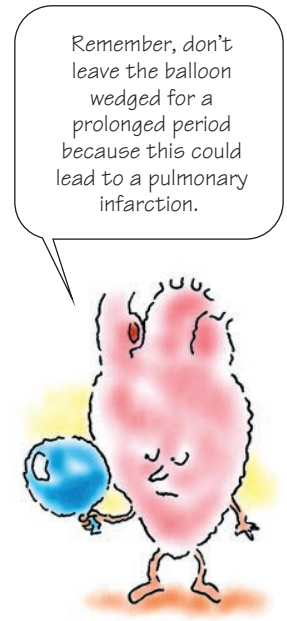
A PA catheter contains several lumen ports to allow various catheter functions:

- The balloon inflation lumen inflates the balloon at the distal tip of the catheter for PAWP measurement.
- A distal lumen measures PAP when connected to a transducer and measures PAWP during balloon inflation. It also permits drawing of mixed venous blood samples.
- A proximal lumen measures right atrial pressure (CVP).
- The thermistor connector lumen contains temperature-sensitive wires, which feed information into a computer for CO calculation.
- Another lumen may provide a port for pacemaker electrodes or measurement of mixed venous oxygen saturation (SvO_2).

Five-lumen PA catheter



The deflated catheter rests in the pulmonary artery, allowing diastolic and systolic PAP readings. The balloon should be totally deflated except when taking a PAWP reading because prolonged wedging can cause pulmonary infarction. (See *Normal PA waveforms*.)



Nursing considerations

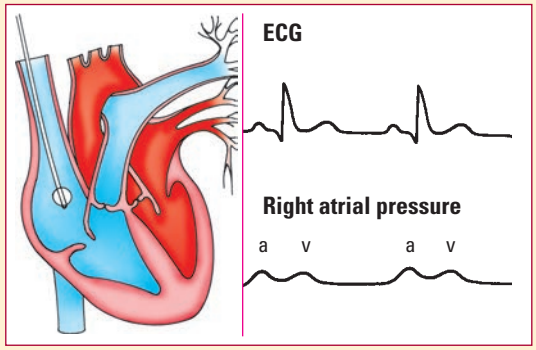
- Inform the patient he'll be conscious during catheterization and he may feel temporary local discomfort from the administration of the local anesthetic. Catheter insertion takes about 15 to 30 minutes.
- After catheter insertion, you may inflate the balloon with a syringe to take PAWP readings. Be careful not to inflate the balloon with more than 1.5 cc of air. Overinflation could distend the pulmonary artery causing vessel rupture. Don't leave the balloon wedged for a prolonged period because this could lead to a pulmonary infarction.
- After each PAWP reading, flush the line; if you encounter difficulty, notify the practitioner.
- Maintain 300 mm Hg pressure in the pressure bag to allow a flush flow of 3 to 6 ml per hour.
- If fever develops when the catheter is in place, inform the practitioner; he may remove the catheter and send its tip to the laboratory for culture.
- Make sure stopcocks are properly positioned and connections are secure. Loose connections may introduce air into the system or cause blood backup, leakage of deoxygenated blood, or inaccurate pressure readings. Also make sure the lumen hubs are properly identified to serve the appropriate catheter ports.
- Because the catheter can slip back into the right ventricle and irritate it, check the monitor for a right ventricular waveform to detect this problem promptly. Be aware that running a continuous infusion through the distal lumen will interfere with your ability to monitor this waveform for changes.
- To minimize valvular trauma, make sure the balloon is deflated whenever the catheter is withdrawn from the pulmonary artery to the right ventricle or from the right ventricle to the right atrium.
- Adhere to your facility's policy for dressing, tubing, catheter, and flush changes.
- Document the date and time of catheter insertion, the doctor who performed the procedure, the catheter insertion site, pressure waveforms and values for the various heart chambers, balloon inflation volume required to obtain a wedge tracing, arrhythmias that occurred during or after the procedure, type of flush solution used and its heparin concentration (if any), type of dressing applied, and the patient's tolerance of the procedure.

Normal PA waveforms

During PA catheter insertion, the waveforms on the monitor change as the catheter advances through the heart.

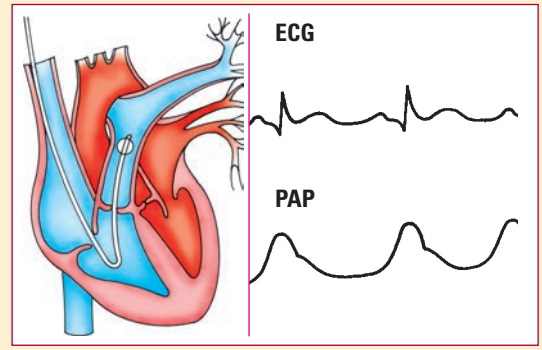
Right atrium

When the catheter tip enters the right atrium, the first heart chamber on its route, a waveform like the one shown below appears on the monitor. Note the two small upright waves. The *a* waves represent the right ventricular end-diastolic pressure; the *v* waves, right atrial filling.



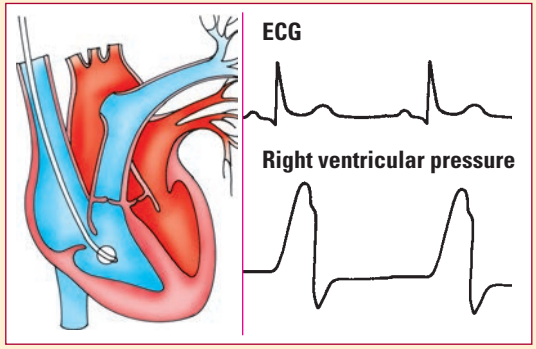
Pulmonary artery

The catheter then floats into the pulmonary artery, causing a PAP waveform such as the one shown below. Note that the upstroke is smoother than on the right ventricle waveform. The dicrotic notch indicates pulmonic valve closure.



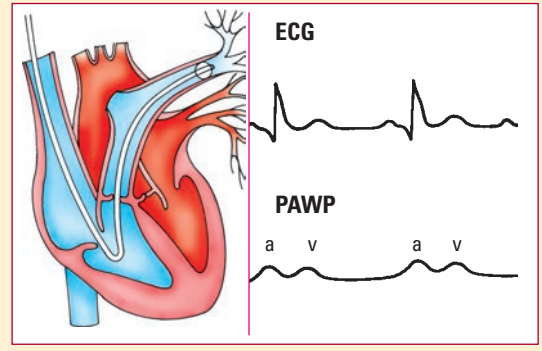
Right ventricle

As the catheter tip reaches the right ventricle, you'll see a waveform with sharp systolic upstrokes and lower diastolic dips, as shown below.



PAWP

Floating into a distal branch of the pulmonary artery, the balloon wedges where the vessel becomes too narrow for it to pass. The monitor now shows a PAWP waveform, with two small upright waves, as shown below. The *a* wave represents left ventricular end-diastolic pressure, the *v* wave, left atrial filling. The balloon is then deflated, and the catheter is left in the pulmonary artery.



Cardiac output monitoring

Cardiac output—the amount of blood ejected by the heart in 1 minute—is monitored to evaluate cardiac function. The normal range for cardiac output is 4 to 8 L per minute.

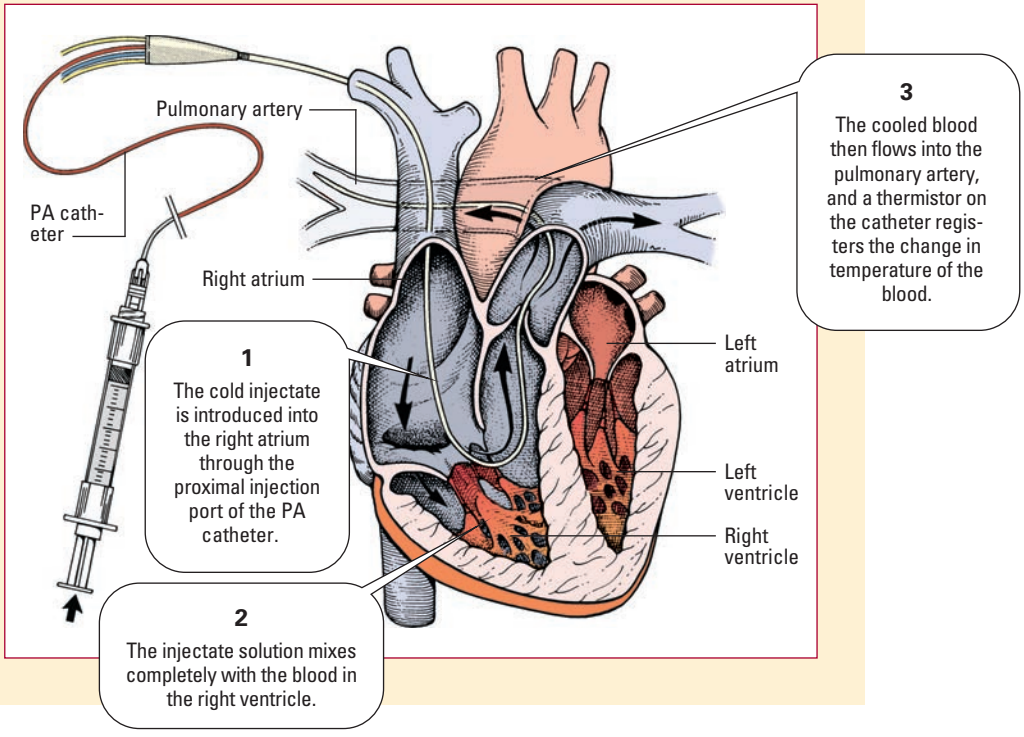
The most widely used method for monitoring cardiac output is the intermittent bolus thermodilution technique. (See *A closer look at the intermittent bolus thermodilution method.*) The ability to continuously monitor cardiac output is also available. (See *A closer look at the continuous cardiac output method.*)

On the rocks or room temperature

To measure cardiac output, a solution is injected into the right atrium through a port on a PA catheter. Iced or room-temperature injectant may be used depending on your facility's policy and on the patient's status.

A closer look at the intermittent bolus thermodilution method

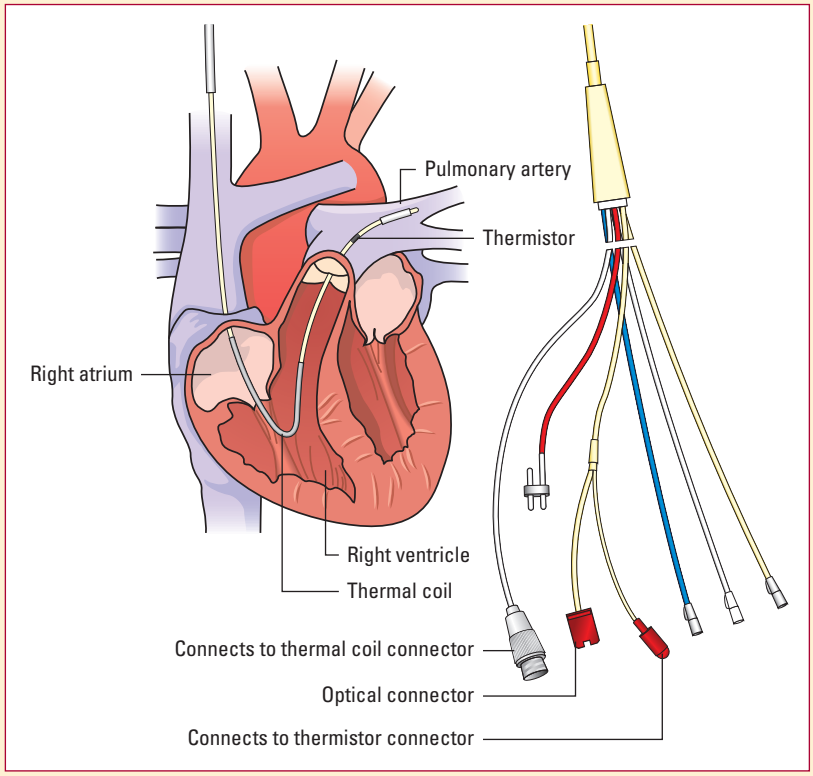
This illustration shows the path of the injectate solution through the heart during intermittent bolus thermodilution CO monitoring.



A closer look at the continuous cardiac output method

Measuring CO using a continuous cardiac output (CCO) system requires a modified PA catheter and CO computer. Rather than using a cooler-than-blood injectant as the input signal, the CCO system relies on a thermal filament on the catheter's outer surface. The thermal filament creates an input signal by emitting pulses of low heat energy, warming blood as it flows by; a thermistor then measures the temperature downstream. A computer algorithm identifies when the pulmonary artery temperature change matches the temperature of the input signal and produces a thermodilution washout curve and the CO value.

The monitor measures CO about every 30 to 60 seconds and displays a continuously updated CO value, averaged from the previous 3 to 6 minutes of data collected.



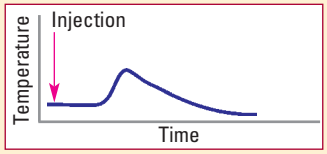
This indicator solution mixes with the blood as it travels through the right ventricle into the pulmonary artery, and a thermistor on the catheter registers the change in temperature of the flowing blood. A computer then plots the temperature change over time as a curve and calculates flow based on the area under the curve. (See *Analyzing thermodilution curves*, page 190.)

Analyzing thermodilution curves

The thermodilution curve provides valuable information about CO, injection technique, and equipment problems. When studying the curve, keep in mind that the area under the curve is inversely proportionate to CO: The smaller the area under the curve, the higher the CO; the larger the area under the curve, the lower the CO.

Besides providing a record of CO, the curve may indicate problems related to technique, such as erratic or slow injectate instillations, or other problems, such as respiratory variations or electrical interference. The curves below correspond to those typically seen in clinical practice.

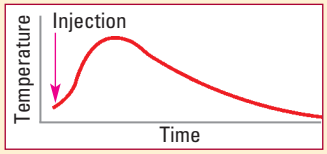
Normal thermodilution curve



With an accurate monitoring system and a patient who has adequate CO, the thermodilution curve begins with a smooth, rapid upstroke and is followed by a smooth, gradual downslope. The curve shown below indicates that the injectate instillation time was within the recommended 4 seconds and that the temperature curve returned to baseline blood temperature.

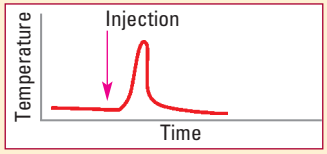
The height of the curve will vary, depending on whether you use a room temperature or an iced injectate. Room-temperature injectate produces an upstroke of lower amplitude.

Low CO curve



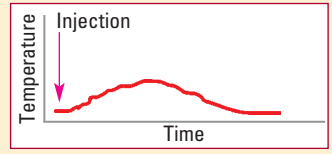
A thermodilution curve representing low CO shows a rapid, smooth upstroke (from proper injection technique). However, because the heart is ejecting blood less efficiently from the ventricles, the injectate warms slowly and takes longer to be ejected from the ventricle. Consequently, the curve takes longer to return to baseline. This slow return produces a larger area under the curve, corresponding to low CO.

High CO curve



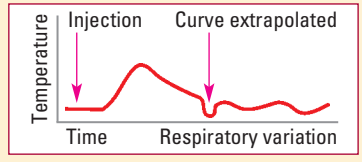
Again, the curve has a rapid, smooth upstroke from proper injection technique. But because the ventricles are ejecting blood too forcefully, the injectate moves through the heart quickly, and the curve returns to baseline more rapidly. The smaller area under the curve suggests higher CO.

Curve reflecting poor technique



This curve results from an uneven and too slow (taking more than 4 seconds) administration of injectate. The uneven and slower than normal upstroke and the larger area under the curve erroneously indicate low CO. A kinked catheter, unsteady hands during the injection, or improper placement of the injectate lumen in the introducer sheath may also cause this type of curve.

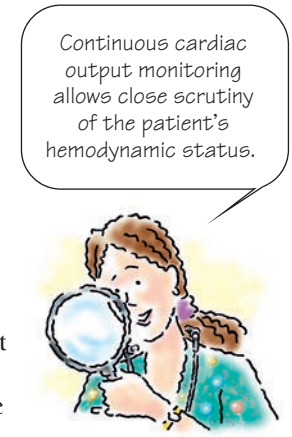
Curve associated with respiratory variations



To obtain a reliable CO measurement, you need a steady baseline pulmonary artery blood temperature. If the patient has rapid or labored respirations or if he's receiving mechanical ventilation, the thermodilution curve may reflect inaccurate CO values. The curve shown below from a patient receiving mechanical ventilation reflects fluctuating pulmonary artery blood temperatures. The thermistor interprets the unsteady temperature as a return to baseline. The result is a curve erroneously showing a high CO (small area under the curve). (*Note:* In some cases, the equipment senses no return to baseline at all and produces a sine-like curve recorded by the computer as 0.00.)

To be continued

Some PA catheters contain a filament that permits continuous cardiac output monitoring. Using such a device, an average cardiac output value is determined over a 3-minute span; the value is updated every 30 to 60 seconds. This type of monitoring allows close scrutiny of the patient's hemodynamic status and prompt intervention in case problems arise.



Better assessor

Cardiac output is better assessed by calculating cardiac index, which takes body size into account. To calculate the patient's cardiac index, divide his cardiac output by his body surface area, a function of height and weight. The normal cardiac index for adults ranges from 2.5 to 4.2 L per minute per m^2; for pregnant women, 3.5 to 6.5 L per minute per m^2. There are several other measurements of cardiac function that combine cardiac output values with other values obtained from a PA catheter and an arterial line. (See *Measuring cardiac function*.)

Measuring cardiac function

Listed here are several common measures of cardiac function that are based on information obtained from a PA catheter. Most CO systems will compute these values automatically.

	Normal values	Formula for calculation	Causes of increased values	Causes of decreased values
Stroke volume Volume of blood pumped by the ventricle in one contraction	60 to 130 ml per beat	SV = CO × 1,000/HR	• Sepsis • Hypervolemia • Inotrope administration	• Arrhythmias • Hypovolemia • Decreased contractility • Increased afterload
Stroke volume index Determines if the SV is adequate for patient's body size	30 to 65 ml/beat/m^2	SVI = SV/BSA or SVI = CI/HR	• Same as SV	• Same as SV
Systemic vascular resistance Degree of left ventricular resistance, or afterload	800 to 1,400 dynes/sec/cm^{-5}	SVR = MAP − CVP/CO × 80	• Hypothermia • Hypovolemia • Vasoconstriction	• Vasodilation • Vasodilators • Shock (anaphylactic, neurogenic, or septic)
Pulmonary vascular resistance	20 to 200 dynes/sec/cm^{-5}	PVR = MPAP − PAWP/CO × 80	• Hypoxemia • Pulmonary embolism • Pulmonary hypertension	• Pulmonary vasodilating drugs (morphine)

Nursing considerations

- Make sure your patient doesn't move during the procedure because movement can cause an error in measurement.
- Perform cardiac output measurements and monitoring at least every 2 to 4 hours, especially if the patient is receiving vasoactive or inotropic agents or if fluids are being added or restricted.
- Discontinue cardiac output measurements when the patient is hemodynamically stable and weaned from his vasoactive and inotropic medications.
- Monitor the patient for signs and symptoms of inadequate perfusion, including restlessness, fatigue, changes in level of consciousness (LOC), decreased capillary refill time, diminished peripheral pulses, oliguria, and pale, cool skin.
- Add the fluid volume injected for cardiac output determinations to the patient's total intake.
- Record the patient's cardiac output, cardiac index, and other hemodynamic values and vital signs at the time of measurement. Note the patient's position during measurement.

Treatments

Many treatments are available for patients with cardiovascular disease; the dramatic ones, such as heart transplantation and the artificial heart, have received a lot of publicity. Commonly used treatment measures include drug therapy; surgery; balloon catheter treatments; and other treatments, such as defibrillation, synchronized cardioversion, and pacemaker insertion.

Drug therapy

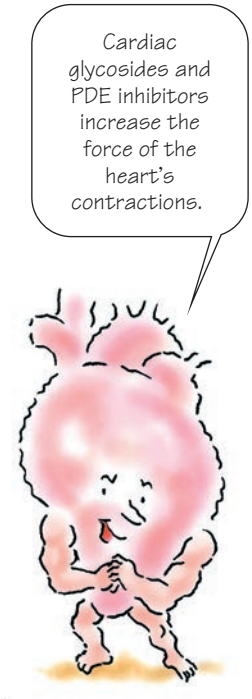
Types of drugs used to improve cardiovascular function include cardiac glycosides and phosphodiesterase (PDE) inhibitors, antiarrhythmic drugs, antianginal drugs, antihypertensive drugs, diuretic drugs, adrenergic drugs, and beta-adrenergic blockers.

Cardiac glycosides and PDE inhibitors

Cardiac glycosides and PDE inhibitors increase the force of the heart's contractions.

More force

Increasing the force of contractions is known as a *positive inotropic effect*, so these drugs are also called *inotropic agents* (affecting the force or energy of muscular contractions). (See *Understanding cardiac glycosides and PDE inhibitors*.)



Understanding cardiac glycosides and PDE inhibitors

Cardiac glycosides and PDE inhibitors have a positive inotropic effect on the heart, meaning they increase the force of contraction. Use this table to learn about the indications, adverse reactions, and practice pointers associated with these drugs.

Drugs	Indications	Adverse reactions	Practice pointers
Cardiac glycoside			
Digoxin (Lanoxin)	Heart failure, supraventricular arrhythmias	• Digoxin toxicity (nausea, abdominal pain, headache, irritability, depression, insomnia, vision disturbances, arrhythmias) • Arrhythmias • Anorexia	• If immediate effects are required (as with a supraventricular arrhythmia), a loading dose of digoxin is required. • Check apical pulse for 1 minute before administration; report pulse less than 60 beats/minute. • Therapeutic serum levels are 0.5 to 2 ng per ml.
PDE inhibitors			
Inamrinone, milrinone	Heart failure refractory to digoxin, diuretics, and vasodilators	• Arrhythmias • Nausea • Vomiting • Headache • Fever • Chest pain • Hypokalemia • Thrombocytopenia	• These drugs are contraindicated in patients in the acute phase of MI and after an MI. • Serum potassium levels should be within normal limits before and during therapy.

Slower rate

Cardiac glycosides, such as digoxin (Lanoxin), also slow the heart rate (called a *negative chronotropic effect*) and slow electrical impulse conduction through the AV node (called a *negative dromotropic effect*).

The short and long of it

PDE inhibitors, such as inamrinone and milrinone, are typically used for short-term management of heart failure or long-term management in patients awaiting heart transplant surgery.

Boosting output

PDE inhibitors improve cardiac output by strengthening contractions. These drugs are thought to help move calcium into the cardiac cell or to increase calcium storage in the sarcoplasmic reticulum.

Understanding antiarrhythmics

Antiarrhythmics are used to restore normal heart rhythm in patients with arrhythmias. Check this table for information about the indications, adverse reactions, and practice pointers associated with these drugs.

Drugs	Indications	Adverse reactions	Practice pointers
Class IA antiarrhythmics			
Disopyramide (Norpace), procainamide, quinidine sulfate, quinidine gluconate	• Ventricular tachycardia • Atrial fibrillation • Atrial flutter • Paroxysmal atrial tachycardia	• Diarrhea • Nausea • Vomiting • Arrhythmias • ECG changes • Hepatotoxicity • Respiratory arrest	• Check apical pulse rate before therapy. If you note extremes in pulse rate, hold the dose and notify the practitioner. • Use cautiously in patients with asthma.
Class IB antiarrhythmics			
Lidocaine (Xylocaine), mexiletine	• Ventricular tachycardia, ventricular fibrillation	• Drowsiness • Hypotension • Bradycardia • Arrhythmias • Widened QRS complex	• IB antiarrhythmics may potentiate the effects of other antiarrhythmics. • Administer I.V. infusions using an infusion pump.
Class IC antiarrhythmics			
Flecainide (Tambocor), propafenone (Rythmol)	• Ventricular tachycardia, ventricular fibrillation, supraventricular arrhythmias	• New arrhythmias • Heart failure • Cardiac death	• Correct electrolyte imbalances before administration. • Monitor the patient's ECG before and after dosage adjustments.
Class II antiarrhythmics			
Acebutolol (Sectral), esmolol (Brevibloc), propranolol (Inderal)	• Atrial flutter, atrial fibrillation, paroxysmal atrial tachycardia • Ventricular arrhythmias	• Arrhythmias • Bradycardia • Heart failure • Hypotension • Nausea and vomiting • Bronchospasm	• Monitor apical heart rate and blood pressure. • Abruptly stopping these drugs can exacerbate angina and precipitate MI.

Understanding antiarrhythmics

Drugs	Indications	Adverse reactions	Practice pointers
Class III antiarrhythmics			
Amiodarone (Cordarone), ibutilide fumarate (Corvert)	• Life-threatening arrhythmias resistant to other antiarrhythmics	• Aggravation of arrhythmias • Hypotension • Anorexia • Severe pulmonary toxicity (amiodarone) • Hepatic dysfunction	• Amiodarone increases the risk of digoxin toxicity in patients also taking digoxin. • Monitor blood pressure, heart rate, and rhythm for changes. • Monitor for signs of pulmonary toxicity (dyspnea, nonproductive cough, and pleuritic chest pain).
Class IV antiarrhythmics			
Diltiazem (Cardizem), verapamil (Calan)	• Supraventricular arrhythmias	• Peripheral edema • Hypotension • Bradycardia • AV block • Flushing (with diltiazem) • Heart failure • Pulmonary edema	• Monitor heart rate and rhythm and blood pressure carefully when initiating therapy or increasing dose. • Calcium supplements may reduce effectiveness.
Miscellaneous			
Adenosine (Adenocard)	• Paroxysmal supraventricular tachycardia	• Facial flushing • Shortness of breath • Dyspnea • Chest discomfort	• Adenosine must be administered over 1 to 2 seconds, followed by a 20 ml flush of normal saline solution. • Record rhythm strip during administration.

By directly relaxing vascular smooth muscle, they also decrease peripheral vascular resistance (afterload) and the amount of blood returning to the heart (preload).

Antiarrhythmics

Antiarrhythmic drugs are used to treat arrhythmias, which are disturbances of the normal heart rhythm. (See *Understanding antiarrhythmics*.)

Benefits vs. risks

Unfortunately, many antiarrhythmic drugs can worsen or cause arrhythmias, too. In any case, the benefits of antiarrhythmic therapy need to be weighed against its risks.

Four classes plus . . .

Antiarrhythmics are categorized into four major classes: I (which includes IA, IB, and IC), II, III, and IV. The mechanisms of action of antiarrhythmic drugs vary widely, and a few drugs exhibit properties common to more than one class. One drug, adenosine, doesn't fall into any of these classes.

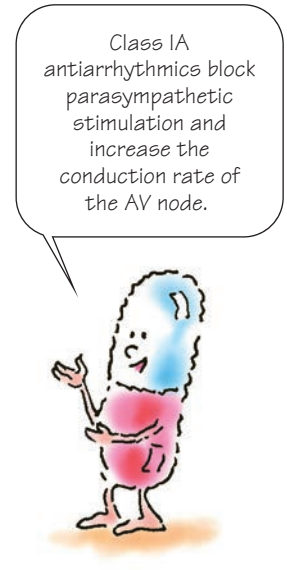
Class I antiarrhythmics

Class I antiarrhythmics are sodium channel blockers. This is the largest group of antiarrhythmic drugs. Class I agents are commonly subdivided into classes IA, IB, and IC. With the development of newer drugs, the use of this class of antiarrhythmics is decreasing.

Class IA antiarrhythmics

Class IA antiarrhythmics control arrhythmias by altering the myocardial cell membrane and interfering with autonomic nervous system control of pacemaker cells. Class IA antiarrhythmics include:

- disopyramide (Norpace)
- procainamide
- quinidine sulfate
- quinidine gluconate.



No (para)sympathy

Class IA antiarrhythmics also block parasympathetic stimulation of the SA and AV nodes. Because stimulation of the parasympathetic nervous system causes the heart rate to slow down, drugs that block the parasympathetic nervous system increase the conduction rate of the AV node.

Rhythmic risks

This increase in the conduction rate can produce dangerous increases in the ventricular heart rate if rapid atrial activity is present, as in a patient with atrial fibrillation. In turn, the increased ventricular heart rate can offset the ability of the antiarrhythmics to convert atrial arrhythmias to a regular rhythm.

Class IB antiarrhythmics

Lidocaine (Xylocaine), a class IB antiarrhythmic, is one of the antiarrhythmics commonly used in treating patients with acute ventricular arrhythmias. Mexiletine is another drug in this class.

Class IB drugs work by blocking the rapid influx of sodium ions during the depolarization phase of the heart's depolarization–repolarization cycle, resulting in a decreased refractory period, which reduces the risk of arrhythmia.

Make an IB-line for the ventricle

Because class IB antiarrhythmics especially affect the Purkinje fibers (fibers in the conducting system of the heart) and myocardial cells in the ventricles, they're used only in treating patients with ventricular arrhythmias.

Class IC antiarrhythmics

Class IC antiarrhythmics are used to treat patients with certain severe, refractory (resistant) ventricular arrhythmias. Class IC antiarrhythmics include flecainide (Tambocor) and propafenone (Rythmol).

Slowing the seeds of conduction

Class IC antiarrhythmics primarily slow conduction along the heart's conduction system. Moricizine decreases the fast inward current of sodium ions of the action potential. This depresses the depolarization rate and effective refractory period.

Class II antiarrhythmics

Class II antiarrhythmics include the beta-adrenergic antagonists, also known as *beta-adrenergic blockers*. Beta-adrenergic blockers used as antiarrhythmics include:

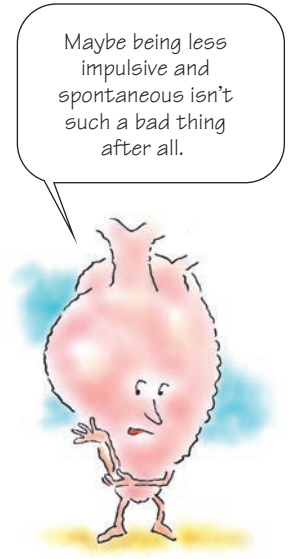
- acebutolol (Sectral)
- esmolol (Brevibloc)
- propranolol (Inderal).

Don't be so impulsive

Class II antiarrhythmics block beta-adrenergic receptor sites in the conduction system of the heart. As a result, the ability of the SA node to fire spontaneously (automaticity) is slowed. The ability of the AV node and other cells to receive and conduct an electrical impulse to nearby cells (conductivity) is also reduced.

Sometimes weaker is better

Class II antiarrhythmics also reduce the strength of the heart's contractions. When the heart beats less forcefully, it doesn't require as much oxygen to do its work.



Class III antiarrhythmics

Class III antiarrhythmics are used to treat patients with ventricular arrhythmias. Amiodarone (Cordarone) is the most widely used class III antiarrhythmic.

One-way to two-way

Although the exact mechanism of action isn't known, class III antiarrhythmics are thought to suppress arrhythmias by converting a

unidirectional block to a bidirectional block. They have little or no effect on depolarization.

Class IV antiarrhythmics

The class IV antiarrhythmics include the calcium channel blockers. These drugs block the movement of calcium during phase 2 of the action potential and slow conduction and the refractory period of calcium-dependent tissues, including the AV node. The calcium channel blockers used to treat patients with arrhythmias are verapamil (Calan) and diltiazem (Cardizem).

Adenosine

Adenosine (Adenocard) is an injectable antiarrhythmic drug indicated for acute treatment for paroxysmal supraventricular tachycardia.

Depressing the pacemaker

Adenosine depresses the pacemaker activity of the SA node, reducing the heart rate and the ability of the AV node to conduct impulses from the atria to the ventricles.

Antianginal drugs

When the oxygen demands of the heart exceed the amount of oxygen being supplied, areas of heart muscle become ischemic (not receiving enough oxygen). When the heart muscle is ischemic, a person experiences chest pain. This condition is known as *angina* or *angina pectoris*.

Reduce demand, increase supply

Although angina's cardinal symptom is chest pain, the drugs used to treat angina aren't typically analgesics. Instead, antianginal drugs correct angina by reducing myocardial oxygen demand (the amount of oxygen the heart needs to do its work), increasing the supply of oxygen to the heart, or both.

The top three

The three classes of commonly used antianginal drugs include:

- nitrates (for acute angina)
- beta-adrenergic blockers (for long-term prevention of angina)
- calcium channel blockers (used when other drugs fail to prevent angina). (See *Understanding antianginal drugs*.)

Nitrates

Nitrates are the drug of choice for relieving acute angina. Nitrates commonly prescribed to correct angina include:

- isosorbide dinitrate (Isordil)

Understanding antianginal drugs

Antianginal drugs are effective in treating patients with angina because they reduce myocardial oxygen demand, increase the supply of oxygen to the heart, or both. Use this table to learn about the indications, adverse reactions, and practice pointers associated with these drugs.

Drugs	Indications	Adverse reactions	Practice pointers
Nitrates			
Isosorbide dinitrate (Isordil), isosorbide mononitrate (Imdur), nitroglycerin (Nitro-Bid)	• Relief and prevention of angina	• Headache • Hypotension • Dizziness • Increased heart rate	• Only sublingual and translingual forms should be used to treat an acute angina attack. • Monitor the patient's blood pressure before and after administration. • Avoid administering nitrates to patients taking erectile dysfunction drugs due to the risk for severe hypotension.
Beta-adrenergic blockers			
Atenolol (Tenormin), carvedilol (Coreg), metoprolol (Lopressor), propranolol (Inderal)	• Long-term prevention of angina • First-line therapy for hypertension • Stable heart failure due to decreased left ventricle ejection fraction	• Bradycardia • Fainting • Fluid retention • Heart failure • Arrhythmias • Nausea • Diarrhea • AV blocks • Bronchospasm • Hypoglycemia	• Monitor apical pulse rate before administration. Monitor blood pressure, ECG, and heart rate and rhythm frequently. • Signs of hypoglycemic shock may be masked; watch diabetic patients for sweating, fatigue, and hunger. • Monitor patients with a history of respiratory problems for breathing difficulty if using a nonselective beta-adrenergic blocker.
Calcium channel blockers			
Amlodipine (Norvasc), diltiazem (Cardizem), nifedipine (Adalat), verapamil (Calan)	• Long-term prevention of angina (especially Prinzmetal's angina) • Hypertension	• Orthostatic hypotension • Heart failure • Hypotension • Arrhythmias • Dizziness • Headache • Persistent peripheral edema • Pulmonary edema	• Monitor cardiac rate and rhythm and blood pressure carefully when initiating therapy or increasing the dose. • Calcium supplementation may decrease the effects of calcium channel blockers.

- isosorbide mononitrate (Imdur)
- nitroglycerin.

Anti-angina effect

Nitrates cause the smooth muscle of the veins and, to a lesser extent, the arteries to relax and dilate. This is what happens:

- When the veins dilate, less blood returns to the heart.
- This, in turn, reduces the amount of blood in the ventricles at the end of diastole, when the ventricles are full. (This blood volume in the ventricles just before contraction is called *preload*.)
- By reducing preload, nitrates reduce ventricular size and ventricular wall tension so the left ventricle doesn't have to stretch much to pump blood. This, in turn, reduces the oxygen requirements of the heart.
- As the coronary arteries dilate, more blood is delivered to the myocardium, improving oxygenation of the ischemic tissue.

Reducing resistance

The arterioles provide the most resistance to the blood pumped by the left ventricle (called *peripheral vascular resistance*). Nitrates decrease afterload by dilating the arterioles, reducing resistance, easing the heart's workload, and easing oxygen demand.

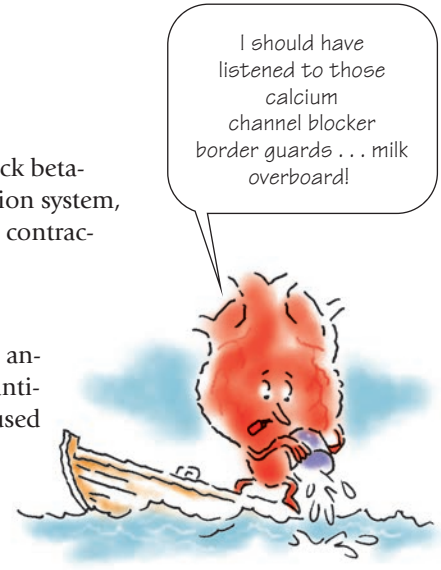
Beta-adrenergic blockers

Beta-adrenergic blockers are used for long-term prevention of angina and are one of the main types of drugs used to treat hypertension. Beta-adrenergic blockers include:

- atenolol (Tenormin)
- carvedilol (Coreg)
- metoprolol (Lopressor)
- propranolol (Inderal).

Down with everything

Beta-adrenergic blockers decrease blood pressure and block beta-adrenergic receptor sites in the heart muscle and conduction system, decreasing heart rate and reducing the force of the heart's contractions, resulting in lower demand for oxygen.



Calcium channel blockers

Calcium channel blockers are commonly used to prevent angina that doesn't respond to drugs in either of the other antianginal classes. Some calcium channel blockers are also used as antiarrhythmics.

Calcium channel blockers include:

- amlodipine (Norvasc)
- diltiazem (Cardizem)

- nifedipine (Adalat)
- verapamil (Calan).

Preventing passage

Calcium channel blockers prevent the passage of calcium ions across the myocardial cell membrane and vascular smooth muscle cells. This causes dilation of the coronary and peripheral arteries, which decreases the force of the heart's contractions and reduces the workload of the heart.

Rate reduction

By preventing arterioles from constricting, calcium channel blockers also reduce afterload. In addition, decreasing afterload decreases oxygen demands of the heart.

Calcium channel blockers also reduce the heart rate by slowing conduction through the SA and AV nodes. A slower heart rate reduces the heart's need for oxygen.

Antihypertensives act to reduce blood pressure. Treatment for hypertension begins with beta-adrenergic blockers and diuretics and may require the use of additional drugs if these treatments are ineffective.

Antihypertensive drugs

Antihypertensive drugs, which act to reduce blood pressure, are used to treat patients with hypertension, a disorder characterized by high systolic blood pressure, diastolic blood pressure, or both.

Know the program

Treatment for hypertension begins with beta-adrenergic blockers and diuretics. If those drugs aren't effective, treatment continues with sympatholytic drugs (other than beta-adrenergic blockers), vasodilators, angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers, or a combination of drugs. (See *Understanding antihypertensives*, page 202.)

Sympatholytic drugs

The sympatholytic drugs include several different types of drugs but work by inhibiting or blocking the sympathetic nervous system, which causes dilation of the peripheral blood vessels or decreases cardiac output, thereby reducing blood pressure.

The sympatholytic drugs are classified by their site or mechanism of action and include:

- central-acting sympathetic nervous system inhibitors, such as clonidine (Catapres), guanabenz, guanfacine (Tenex), and methyldopa
- alpha blockers, such as doxazosin (Cardura), phentolamine, prazosin (Minipress), and terazosin (Hytrin)
- mixed alpha- and beta-adrenergic blockers such as labetalol (Trandate)
- norepinephrine depletors, such as guanadrel (Hylorel).

We sympatholytic drugs reduce blood pressure by blocking the sympathetic nervous system. Just try to get by me!

Understanding antihypertensives

Antihypertensives are prescribed to reduce blood pressure in patients with hypertension. Use this table to learn about the indications, adverse reactions, and practice pointers associated with these drugs.

Drugs	Indications	Adverse reactions	Practice pointers
Sympatholytic drugs			
• *Central-acting sympathetic nervous system inhibitors* (such as clonidine [Catapres], guanabenz, guanfacine [Tenex], and methyldopa) • *Alpha blockers* (such as doxazosin [Cardura], phentolamine, prazosin [Minipress], and terazosin [Hytrin]) • *Mixed alpha- and beta-adrenergic blockers* (such as labetalol [Trandate]) • *Norepinephrine depletors* (such as guanadrel [Hylorel])	• Hypertension	• Hypotension (alpha blockers) • Depression • Drowsiness • Edema • Vertigo (central-acting drugs) • Bradycardia • Hepatic necrosis • Arrhythmias	• Monitor blood pressure and pulse before and after administration.
Vasodilators			
• Hydralazine, minoxidil, nitroprusside (Nipride)	• Used in combination with other drugs to treat moderate to severe hypertension • Hypertensive crisis	• Tachycardia • Palpitations • Angina • Fatigue • Headache • Severe pericardial effusion • Hepatotoxicity • Nausea • Stevens-Johnson syndrome	• Monitor blood pressure and pulse before and after administration. • Monitor patient receiving nitroprusside for signs of cyanide toxicity.
Angiotensin-converting enzyme inhibitors			
• Benazepril (Lotensin), captopril (Capoten), enalapril (Vasotec), lisinopril (Prinivil), quinapril (Accupril), ramipril (Altace)	• Hypertension • Heart failure	• Angioedema • Persistent cough • Rash • Renal insufficiency	• Monitor blood pressure and pulse before and after administration.
Angiotensin-converting enzyme inhibitors			
• Candesartan (Atacand), irbesartan (Avapro), losartan (Cozaar), olmesartan (Benicar), valsartan (Diovan)	• Hypertension • Heart failure resistant to ACE inhibitors	• Fatigue • Abdominal pain • Rash • Hypotension	• Monitor blood pressure and pulse before and after administration.

Vasodilating drugs

The two types of vasodilating drugs include calcium channel blockers and direct vasodilators. These drugs decrease systolic and diastolic blood pressure.

Calcium stoppers

Calcium channel blockers produce arteriolar relaxation by preventing the entry of calcium into the cells. This prevents the contraction of vascular smooth muscle.

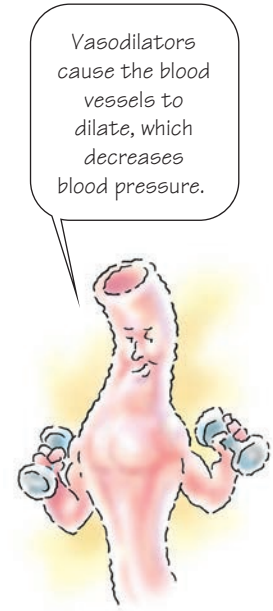
Direct dilators

Direct vasodilators act on arteries, veins, or both. They work by relaxing peripheral vascular smooth muscles, causing the blood vessels to dilate. This decreases blood pressure by increasing the diameter of the blood vessels, reducing total peripheral resistance.

The direct vasodilators include:

- hydralazine
- minoxidil
- nitroprusside (Nitropress).

Hydralazine and minoxidil are usually used to treat patients with resistant or refractory hypertension. Nitroprusside is reserved for use in hypertensive crisis.

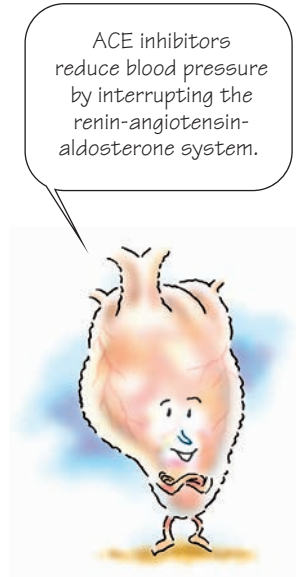


ACE inhibitors

ACE inhibitors reduce blood pressure by interrupting the renin-angiotensin-aldosterone system. These drugs are the prime choice in preventing heart failure in a patient with a recent MI.

Commonly prescribed ACE inhibitors include:

- benazepril (Lotensin)
- captopril (Capoten)
- enalapril (Vasotec)
- lisinopril (Prinivil)
- quinapril (Accupril)
- ramipril (Altace).



Without ACE interference

Here's how the renin-angiotensin-aldosterone system works:

- Normally, the kidneys maintain blood pressure by releasing the hormone renin.
- Renin acts on the plasma protein angiotensinogen to form angiotensin I.
- Angiotensin I is then converted to angiotensin II.
- Angiotensin II, a potent vasoconstrictor, increases peripheral resistance and promotes the excretion of aldosterone.
- Aldosterone, in turn, promotes the retention of sodium and water, increasing the volume of blood the heart needs to pump.

With ACE interference

ACE inhibitors work by preventing the conversion of angiotensin I to angiotensin II. As angiotensin II is reduced, arterioles dilate, reducing peripheral vascular resistance.

Less water, less work

By reducing aldosterone secretion, ACE inhibitors promote the excretion of sodium and water, reducing the amount of blood the heart needs to pump, resulting in a lowered blood pressure.

Angiotensin II receptor blockers

Unlike ACE inhibitors, which prevent production of angiotensin, angiotensin II receptor blockers (ARBs) inhibit the action of angiotensin II by attaching to tissue-binding receptor sites.

Commonly prescribed ARBs include:

- candesartan (Atacand)
- irbesartan (Avapro)
- losartan (Cozaar)
- olmesartan (Benicar)
- valsartan (Diovan).

Diuretics

Diuretics are used to promote the excretion of water and electrolytes by the kidneys. By doing so, diuretics play a major role in treating hypertension and other cardiovascular conditions. (See *Understanding diuretics*.)

The major diuretics used as cardiovascular drugs include:

- thiazide and thiazide-like diuretics
- loop diuretics
- potassium-sparing diuretics.

Thiazide and thiazide-like diuretics

Thiazide and thiazide-like diuretics are sulfonamide derivatives. Thiazide diuretics include hydrochlorothiazide, hydroflumethiazide (Saluron), and methyclothiazide (Enduron). Thiazide-like diuretics include indapamide.

Sodium stoppers

Thiazide and thiazide-like diuretics work by preventing sodium from being reabsorbed in the kidney. As sodium is excreted, it pulls water along with it. Thiazide and thiazide-like diuretics also increase the

Understanding diuretics

Diuretics are used to treat patients with various cardiovascular conditions. They work by promoting the excretion of water and electrolytes by the kidneys. Use this table to learn about the indications, adverse reactions, and practice pointers associated with these drugs.

Drugs	Indications	Adverse reactions	Practice pointers
Thiazide and thiazide-like diuretics			
Hydrochlorothiazide hydroflumethiazide (Saluron), indapamide, methyclothiazide (Enduron)	• Hypertension • Edema	• Hypokalemia • Orthostatic hypotension • Hyponatremia • Dizziness • Nausea	• Monitor serum potassium levels. • Monitor intake and output. • Monitor blood glucose values in diabetic patients. Thiazide diuretics can cause hyperglycemia.
Loop diuretics			
Bumetanide (Bumex), ethacrynic acid (Edecrin), furosemide (Lasix)	• Hypertension • Heart failure • Edema	• Dehydration • Orthostatic hypotension • Hyperuricemia • Hypokalemia • Hyponatremia • Dizziness • Muscle cramps • Rash	• Monitor for signs of excess diuresis (hypotension, tachycardia, poor skin turgor, and excessive thirst). • Monitor blood pressure, heart rate, and intake and output. • Monitor serum electrolyte levels.
Potassium-sparing diuretics			
Amiloride (Midamor), spironolactone (Aldactone), triamterene (Dyrenium)	• Edema • Diuretic-induced hypokalemia in patients with heart failure • Cirrhosis • Nephrotic syndrome • Hypertension	• Hyperkalemia • Headache • Nausea • Rash	• Monitor ECG for arrhythmias. • Monitor serum potassium levels. • Monitor intake and output.

excretion of chloride, potassium, and bicarbonate, which can result in electrolyte imbalances.

Stability with time

Initially, these drugs decrease circulating blood volume, leading to a reduced cardiac output. However, if the therapy is maintained, cardiac output stabilizes, but plasma fluid volume decreases.

Loop diuretics

Loop (high-ceiling) diuretics are highly potent drugs. They include:

- bumetanide (Bumex)
- ethacrynic acid (Edecrin)
- furosemide (Lasix).

High potency, big risk

The loop diuretics are the most potent diuretics available, producing the greatest volume of diuresis (urine production). They also have a high potential for causing severe adverse reactions.

Bumetanide is the shortest acting diuretic. It's even 40 times more potent than another loop diuretic, furosemide.

Loop diuretics are the most potent diuretics, producing the most amount of urine. But they also carry the highest risk for severe adverse reactions.

Locating the loop

Loop diuretics receive their name because they act primarily on the thick ascending loop of Henle (the part of the nephron responsible for concentrating urine) to increase the secretion of sodium, chloride, and water. These drugs may also inhibit sodium, chloride, and water reabsorption.

Potassium-sparing diuretics

Potassium-sparing diuretics have weaker diuretic and antihypertensive effects than other diuretics, but they have the advantage of conserving potassium.

The potassium-sparing diuretics include:

- amiloride (Midamor)
- spironolactone (Aldactone)
- triamterene (Dyrenium).

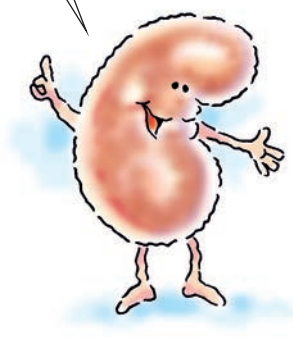
Potassium-sparing effects

The direct action of the potassium-sparing diuretics on the distal tubule of the kidneys produces:

- increased urinary excretion of sodium and water
- increased excretion of chloride and calcium ions
- decreased excretion of potassium and hydrogen ions.

These effects lead to reduced blood pressure and increased serum potassium levels.

Potassium-sparing diuretics have weaker diuretic and antihypertensive effects than other diuretics, but they conserve potassium.



Aping aldosterone

Spironolactone, one of the main potassium-sparing diuretics, is structurally similar to aldosterone and acts as an aldosterone antagonist.

Aldosterone promotes the retention of sodium and water and loss of potassium; spironolactone counteracts these effects by competing with aldosterone for receptor sites. As a result, sodium, chloride, and water are excreted, and potassium is retained.

Understanding anticoagulants

Anticoagulants reduce the blood's ability to clot and are included in the treatment plans for many patients with cardiovascular disorders. Use this table to learn about the indications, adverse reactions, and practice pointers associated with these drugs.

Drugs	Indications	Adverse reactions	Practice pointers
Heparins			
Heparin and low-molecular-weight heparins, such as dalteparin (Fragmin) and enoxaparin (Lovenox)	• Deep vein thrombosis (treatment and prevention) • Embolism prophylaxis • Disseminated intravascular coagulation (heparin) • Prevention of complications after MI	• Bleeding • Hemorrhage • Thrombocytopenia	• Monitor thromboplastin time; the therapeutic range is 1½ to 2½ times the control. • Monitor the patient for signs of bleeding. • Concomitant administration with nonsteroidal anti-inflammatory drugs (NSAIDs), iron dextran, or an antiplatelet drug increases the risk of bleeding. • Protamine sulfate reverses the effects of heparin.
Factor Xa inhibitor			
Fondaparinux (Arixtra)	• Deep vein thrombosis (treatment and prevention) • Acute pulmonary embolism	• Hemorrhage • Thrombocytopenia • Nausea • Fever	• This drug is not interchangeable with heparin or low-dose heparins. • Monitor the patient for signs of bleeding. • Monitor complete blood count (CBC) and platelet count. • Monitor anti-Xa results; the goal for prophylaxis is 0.2 to 0.4 anti-Xa units/ml; the goal for therapy is 0.5 to 1.0 anti-Xa units/ml.
Oral anticoagulants			
Warfarin (Coumadin)	• Deep vein thrombosis prophylaxis • Prevention of complications of prosthetic heart valves or diseased mitral valves • Atrial arrhythmias	• Bleeding (may be severe) • Hepatitis • Diarrhea	• Monitor prothrombin time and International Normalized Ratio. • Monitor the patient for signs of bleeding. • The effects of oral anticoagulants can be reversed with phytonadione (vitamin K_1).
Antiplatelet drugs			
Aspirin (Ecotrin), dipyridamole (Persantine), ticlopidine (Ticlid), clopidogrel (Plavix)	• Decreases the risk of death post MI • Prevention of complications of prosthetic heart valves • Reduction of risk of MI • Prevention of reocclusion in coronary revascularization procedures	• GI distress • Bleeding • Thrombocytopenia • Angioedema	• Monitor the patient for signs of bleeding. • Aspirin and ticlopidine should be taken with meals to prevent GI irritation. • Dipyridamole should be taken with a full glass of fluid at least 1 hour before meals.

Anticoagulants

Anticoagulants are used to reduce the ability of the blood to clot. (See *Understanding anticoagulants*, page 207.) Major categories of anticoagulants include heparin, oral anticoagulants, and antiplatelet drugs.

Heparin

Heparin, prepared commercially from animal tissue, is used to prevent clot formation. Low-molecular-weight heparin, such as dalteparin (Fragmin) and enoxaparin (Lovenox), prevents deep vein thrombosis (a blood clot in the deep veins, usually of the legs) in surgical patients.

No new clots

Because it doesn't affect the synthesis of clotting factors, heparin can't dissolve already formed clots. It does prevent the formation of new thrombi, though. Here's how it works:

- Heparin inhibits the formation of thrombin and fibrin by activating antithrombin III.
- Antithrombin III then inactivates factors IXa, Xa, XIa, and XIIa in the intrinsic and common pathways. The end result is prevention of a stable fibrin clot.
- In low doses, heparin increases the activity of antithrombin III against factor Xa and thrombin and inhibits clot formation. Much larger doses are necessary to inhibit fibrin formation after a clot has formed. This relationship between dose and effect is the rationale for using low-dose heparin to prevent clotting.
- Whole blood clotting time, thrombin time, and partial thromboplastin time are prolonged during heparin therapy. However, these times may be only slightly prolonged with low or ultra-low preventive doses.

Circulate freely

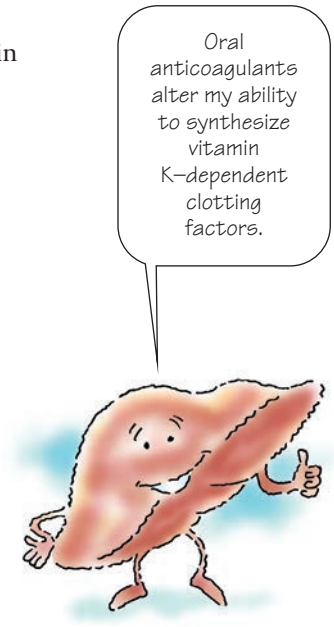
Heparin can be used to prevent clotting when a patient's blood must circulate outside the body through a machine, such as a cardiopulmonary bypass machine or hemodialysis machine.

Factor Xa inhibitors

Factor Xa inhibitors are new class of anticoagulants. At this time, the only drug in this class is fondaparinux (Arixtra). Fondaparinux works by inhibiting only factor Xa—the common point in the intrinsic and extrinsic clotting pathways. Inhibition of factor Xa prevents the formation of thrombin and the formation of a clot.

Oral anticoagulants

Oral anticoagulants alter the ability of the liver to synthesize vitamin K–dependent clotting factors, including prothrombin and factors VII, IX, and X. Clotting factors already in the bloodstream continue to coagulate blood until they become depleted, so anticoagulation doesn't begin immediately.



Warfarin vs. coagulation

The major oral anticoagulant used in the United States is warfarin (Coumadin).

Antiplatelet drugs

Examples of antiplatelet drugs are:

- aspirin (Ecotrin)
- dipyridamole (Persantine)
- ticlopidine (Ticlid)
- clopidogrel (Plavix).

Thromboembolism prevention

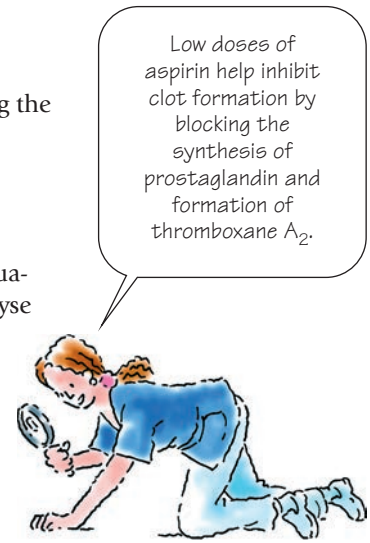
Antiplatelet drugs are used to prevent arterial thromboembolism, especially in patients at risk for MI, stroke, and arteriosclerosis (hardening of the arteries). They interfere with platelet activity in different drug-specific and dose-related ways.

Not just for "babies"

Low dosages of aspirin (81 mg/day) appear to inhibit clot formation by blocking the synthesis of prostaglandin, which in turn prevents formation of the platelet-aggregating substance thromboxane A_2. Dipyridamole and clopidogrel may inhibit platelet aggregation.

Broken bindings

Ticlopidine inhibits the binding of fibrinogen to platelets during the first stage of the clotting cascade.



Thrombolytic drugs

Thrombolytic drugs are used to dissolve a preexisting clot or thrombus and are commonly used in an acute or emergency situation. They work by converting plasminogen to plasmin, which lyse (dissolve) thrombi, fibrinogen, and other plasma proteins. (See *Understanding thrombolytics*, page 210.)

Some commonly used thrombolytic drugs include:

- alteplase (Activase)
- reteplase (Retavase)
- streptokinase (Streptase).

Understanding thrombolytics

Sometimes called *clot busters*, thrombolytic drugs are prescribed to dissolve a preexisting clot or thrombus. These drugs are typically used in acute or emergency situations. Use this table to learn about the indications, adverse reactions, and practice pointers associated with these drugs.

Drugs	Indications	Adverse reactions	Practice pointers
Thrombolytics			
Alteplase, reteplase, streptokinase	• Acute MI • Acute ischemic stroke • Pulmonary embolus • Catheter occlusion • Arterial thrombosis	• Bleeding • Allergic reaction	• Monitor partial thromboplastin time, prothrombin time, International Normalized Ratio, hemoglobin, and hematocrit before, during, and after administration. • Monitor vital signs frequently during and immediately after administration. Don't use an automatic blood pressure cuff to monitor blood pressure. • Monitor puncture sites for bleeding. Don't use a tourniquet when obtaining blood specimens. • Monitor for signs of bleeding.

Adrenergic drugs

Adrenergic drugs are also called *sympathomimetic drugs* because they produce effects similar to those produced by the sympathetic nervous system.

Classified by chemical

Adrenergic drugs are classified into two groups based on their chemical structure—catecholamines (both naturally occurring and synthetic) and noncatecholamines. (See *Understanding adrenergics*.)

Which receptor?

Therapeutic use of adrenergic drugs depends on which receptors they stimulate and to what degree. Adrenergic drugs can affect:

- alpha-adrenergic receptors
- beta-adrenergic receptors
- dopamine receptors.

Mimicking

Most of the adrenergic drugs produce their effects by stimulating alpha- and beta-adrenergic receptors. These drugs mimic the action of norepinephrine or epinephrine.

Understanding adrenergics

Adrenergic drugs produce effects similar to those produced by the sympathetic nervous system. Adrenergic drugs can affect alpha-adrenergic receptors, beta-adrenergic receptors, or dopamine receptors. However, most of the drugs stimulate the alpha- and beta-receptors, mimicking the effects of norepinephrine and epinephrine. Dopaminergic drugs act on receptors typically stimulated by dopamine.

Use this table to learn about the indications, adverse reactions, and practice pointers associated with these drugs.

Drugs	Indications	Adverse reactions	Practice pointers
Catecholamines			
Dobutamine	• Increase CO in short-term treatment of cardiac decompensation from depressed contractility.	Headache, tingling sensation, bronchospasm, palpitations, tachycardia, cardiac arrhythmias (PVCs), hypotension, hypertension and hypertensive crisis, angina, nausea, vomiting, tissue necrosis and sloughing (if catecholamine given I.V. leaks into surrounding tissue)	• Correct hypovolemia before administering drug. • Incompatible with alkaline solution (sodium bicarbonate); don't mix or give through same line; don't mix with other drugs. • Administer continuous drip on infusion pump. • Give drug into a large vein to prevent irritation or extravasation at site. • Monitor cardiac rate and rhythm and blood pressure carefully when initiating therapy or increasing the dose.
Dopamine	• Shock and correct hemodynamic imbalances. • Increase CO. • Hypotension	Headache, bradycardia, palpitations, tachycardia, conduction disturbance, cardiac arrhythmias (ventricular), hypotension, hypertension and hypertensive crisis, azotemia, angina, nausea, vomiting, gangrene of extremities in high dose, tissue necrosis and sloughing (if catecholamine given I.V. leaks into surrounding tissue), bronchospasm	• Correct hypovolemia before administering drug. • Administer continuous drip on infusion pump. • Give drug into a large vein to prevent extravasation; if extravasation occurs, stop infusion and treat site with phentolamine (Regitine) infiltrate to prevent tissue necrosis. • Monitor cardiac rate and rhythm and blood pressure carefully when initiating therapy or increasing the dose. • Monitor urine output during treatment, especially at high doses.
Epinephrine (Adrenalin)	• Bronchospasm • Hypersensitivity reactions • Anaphylaxis • Restoration of cardiac rhythm in cardiac arrest	Restlessness, anxiety, dizziness, headache, tachycardia, palpitations, cardiac arrhythmias (ventricular fibrillation), hypertension, stroke, cerebral hemorrhage, angina, increased blood glucose levels, tissue necrosis and sloughing (if catecholamine given I.V. leaks into surrounding tissue)	• Correct hypovolemia before administering drug. • Administer continuous drip on infusion pump. • Give drug into a large vein to prevent irritation or extravasation at site. • Monitor cardiac rate and rhythm and blood pressure carefully when initiating therapy or increasing the dose.

(continued)

Understanding adrenergics *(continued)*

Drugs	Indications	Adverse reactions	Practice pointers
***Catecholamines** (continued)*			
Norepinephrine (Levophed)	• Maintain blood pressure in acute hypotensive states.	Anxiety, dizziness, headache, bradycardia, cardiac arrhythmias, hypotension, hypertension, tissue necrosis and sloughing (if catecholamine given I.V. leaks into surrounding tissue), fever, metabolic acidosis, increased blood glucose levels, dyspnea	• Correct hypovolemia before administering drug. • Administer continuous drip on infusion pump. • Give drug into a large vein to prevent extravasation; if extravasation occurs, stop infusion and treat site with phentolamine infiltrate to prevent tissue necrosis. • Monitor cardiac rate and rhythm and blood pressure carefully when initiating therapy or increasing the dose.
Noncatecholamines			
Ephedrine	• Maintain blood pressure in acute hypotensive states, especially with spinal anesthesia. • Treatment of orthostatic hypotension and bronchospasm	Anxiety, dizziness, headache, palpitations, hypotension, hypertension, nausea, vomiting, tachycardia	• Correct hypovolemia before administering drug. • Give drug into a large vein to prevent irritation or extravasation at site. • Monitor cardiac rate and rhythm and blood pressure carefully when initiating therapy or increasing the dose.
Phenylephrine (Neo-Synephrine)	• Maintain blood pressure in hypotensive states, especially hypotensive emergencies with spinal anesthesia.	Restlessness, anxiety, dizziness, headache, palpitations, cardiac arrhythmias, hypertension, tissue necrosis and sloughing (if noncatecholamine given I.V. leaks into surrounding tissue)	• Correct hypovolemia before administering drug. • Administer continuous drip on infusion pump. • Give drug into a large vein to prevent extravasation; if extravasation occurs, stop infusion and treat site with phentolamine infiltrate to prevent tissue necrosis. • Monitor cardiac rate and rhythm and blood pressure carefully when initiating therapy or increasing the dose.

Doing it like dopamine

Dopaminergic drugs act primarily on receptors in the sympathetic nervous system that are stimulated by dopamine.

Catecholamines

Because of their common basic chemical structure, catecholamines share certain properties. They stimulate the nervous system, constrict

peripheral blood vessels, increase the heart rate, and dilate the bronchi. They can be manufactured in the body or in a laboratory. Common catecholamines include:

- dobutamine
- dopamine
- epinephrine (Adrenalin)
- norepinephrine (Levophed).

Direct-acting and excitatory or inhibitory

Catecholamines are primarily direct-acting. When catecholamines combine with alpha- or beta-receptors, they cause either an excitatory or inhibitory effect. Typically, activation of alpha-receptors generates an excitatory response except for intestinal relaxation. Activation of the beta-receptors mostly produces an inhibitory response except in the cells of the heart, where norepinephrine produces excitatory effects.

How heartening

The clinical effects of catecholamines depend on the dosage and the route of administration. Catecholamines are potent inotropes, meaning they make the heart contract more forcefully. As a result, the ventricles empty more completely with each heartbeat, increasing the workload of the heart and the amount of oxygen it needs to do this harder work.

Rapid rates

Catecholamines also produce a positive chronotropic effect, which means they cause the heart to beat faster. That's because the pacemaker cells in the SA node of the heart depolarize at a faster rate. As catecholamines cause blood vessels to constrict and blood pressure to increase, the heart rate decreases as the body tries to prevent an excessive increase in blood pressure.

Fascinating rhythm

Catecholamines can cause the Purkinje fibers (an intricate web of fibers that carry electrical impulses into the ventricles of the heart) to fire spontaneously, possibly producing abnormal heart rhythms, such as PVCs and fibrillation. Epinephrine is likelier than norepinephrine to produce this spontaneous firing.

Noncatecholamines

Noncatecholamine adrenergic drugs have a variety of therapeutic uses because of the many effects these drugs can have on the body, such as the local or systemic constriction of blood vessels by phenylephrine.

Alpha active . . .

Direct-acting noncatecholamines that stimulate alpha activity include methoxamine and phenylephrine.

Beta active . . .

Those that selectively exert beta$_2$ activity include:

- albuterol (Proventil)
- isoetharine
- metaproterenol.

. . . or both

Dual-acting noncatecholamines combine both actions such as ephedrine.

Adrenergic blocking drugs

Adrenergic blocking drugs, also called *sympatholytic drugs*, are used to disrupt sympathetic nervous system function. (See *Understanding adrenergic blockers*.)

Impeding impulses

These drugs work by blocking impulse transmission (and thus sympathetic nervous system stimulation) at adrenergic neurons or adrenergic receptor sites. The action of the drugs at these sites can be exerted by:

- interrupting the action of sympathomimetic (adrenergic) drugs
- reducing available norepinephrine
- preventing the action of cholinergic drugs.

Classified information

Adrenergic blocking drugs are classified according to their site of action as alpha-adrenergic blockers or beta-adrenergic blockers.

Alpha-adrenergic blocking drugs

Alpha-adrenergic blocking drugs work by interrupting the actions of sympathomimetic drugs at alpha-adrenergic receptors.

This results in:

- relaxation of the smooth muscle in the blood vessels
- increased dilation of blood vessels
- decreased blood pressure.

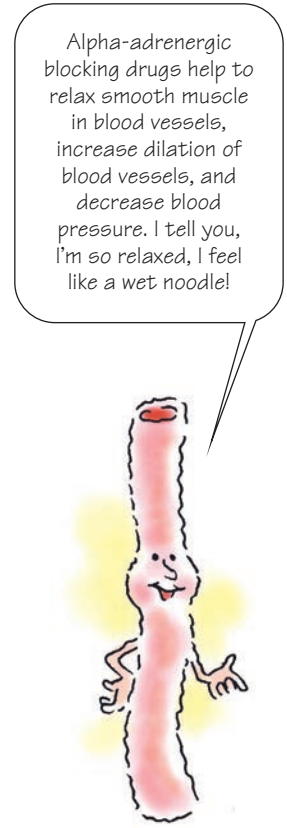
Drugs in this class include phentolamine and prazosin.

A mixed bag

Ergotamine is a mixed alpha agonist and antagonist. At high doses, it acts as an alpha-adrenergic blocker.

Alpha-adrenergic blockers work in one of two ways:

1. They interfere with or block the synthesis, storage, release, and reuptake of norepinephrine by neurons.



Understanding adrenergic blockers

Adrenergic blockers block impulse transmission at adrenergic receptor sites by interrupting the action of adrenergic drugs, reducing the amount of norepinephrine available, and blocking the action of cholinergics.

Use this table to learn the indications, adverse reactions, and practice pointers needed to safely administer these drugs.

Drugs	Indications	Adverse reactions	Practice pointers
Alpha-adrenergic blockers			
Phentolamine, prazosin (Minipress)	• Hypertension • Pheochromocytoma	Orthostatic hypotension, bradycardia, tachycardia, edema, difficulty breathing, flushing, weakness, palpitations, nausea	• Monitor vital signs and heart rhythm before, during, and after administration. • Instruct the patient to rise slowly to a standing position to avoid orthostatic hypotension.
Beta-adrenergic blockers			
Nonselective Carvedilol (Coreg), labetalol (Trandate), propranolol (Inderal), sotalol (Betapace), timolol *Selective* Acebutolol (Sectral), atenolol (Tenormin), esmolol (Brevibloc), metoprolol (Lopressor)	• Prevention of complications after MI, angina, hypertension, supraventricular arrhythmias, anxiety, essential tremor, cardiovascular symptoms associated with thyrotoxicosis, migraine headaches, pheochromocytoma	Hypotension, bradycardia, peripheral vascular insufficiency, bronchospasm (nonselective), sore throat, atrioventricular block, thrombocytopenia, hypoglycemia	• Monitor vital signs and heart rhythm frequently. • Beta-adrenergic blockers can alter the requirements for insulin and oral antidiabetic agents.

2. They antagonize epinephrine, norepinephrine, or adrenergic (sympathomimetic) drugs at alpha-receptor sites.

Not very discriminating

Alpha-receptor sites are either $alpha_1$ or $alpha_2$ receptors. Alpha-adrenergic blockers include drugs that block stimulation of $alpha_1$ receptors and that may block $alpha_2$ stimulation.

Reducing resistance

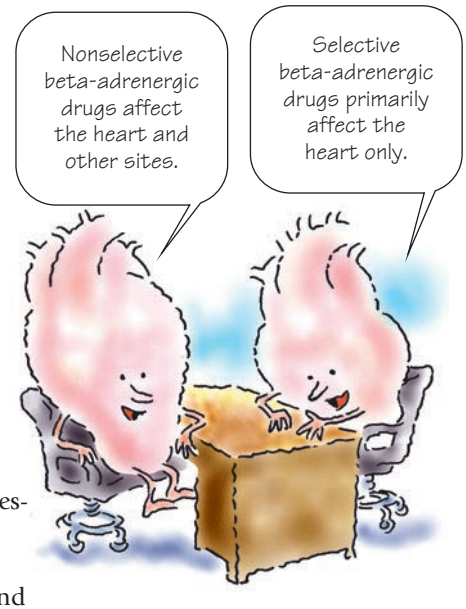
Alpha-adrenergic blockers occupy alpha-receptor sites on the smooth muscle of blood vessels.

This prevents catecholamines from occupying and stimulating the receptor sites. As a result, blood vessels dilate, increasing local blood

flow to the skin and other organs. The decreased peripheral vascular resistance (resistance to blood flow) helps to decrease blood pressure.

Beta-adrenergic blockers

Beta-adrenergic blockers, the most widely used adrenergic blockers, prevent stimulation of the sympathetic nervous system by inhibiting the action of catecholamines and other sympathomimetic drugs at beta-adrenergic receptors.



Selective (or not)

Beta-adrenergic drugs are selective or nonselective. Nonselective beta-adrenergic drugs affect:

- $beta_1$-receptor sites (located mainly in the heart)
- $beta_2$-receptor sites (located in the bronchi, blood vessels, and the uterus).

Nonselective beta-adrenergic drugs include carvedilol, labetalol, propranolol, sotalol (Betapace), and timolol.

Highly discriminating

Selective beta-adrenergic drugs primarily affect the $beta_1$-adrenergic sites. They include acebutolol, atenolol, esmolol, and metoprolol tartrate.

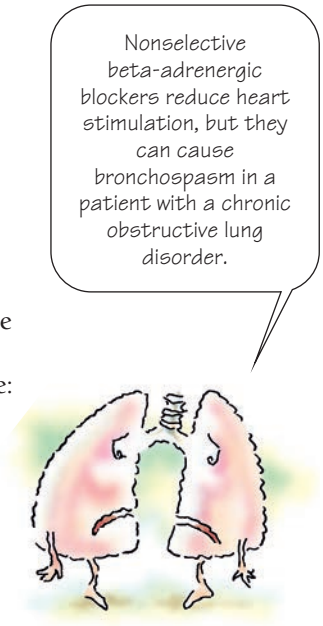
Intrinsically sympathetic

Some beta-adrenergic blockers, such as acebutolol, have intrinsic sympathetic activity. This means that instead of attaching to beta-receptors and blocking them, these beta-adrenergic blockers attach to beta-receptors and stimulate them. These drugs are sometimes classified as partial agonists.

Widely effective

Beta-adrenergic blockers have widespread effects in the body because they produce their blocking action not only at the adrenergic nerve endings but also in the adrenal medulla. Effects on the heart include:

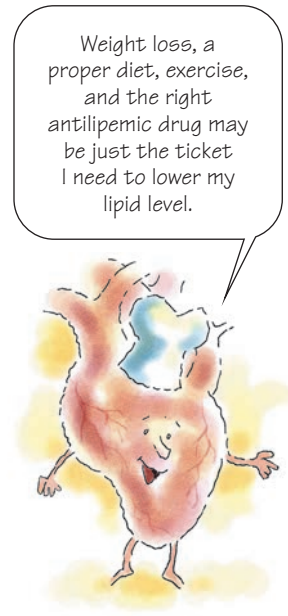
- increased peripheral vascular resistance
- decreased blood pressure
- decreased force of contractions of the heart
- decreased oxygen consumption by the heart
- slowed conduction of impulses between the atria and ventricles
- decreased cardiac output.



Selective or nonselective

Some of the effects of beta-adrenergic blocking drugs depend on whether the drug is classified as selective or nonselective. Selective beta-adrenergic blockers, which preferentially block $beta_1$ receptor sites, reduce stimulation of the heart. They're commonly called *cardioselective beta-adrenergic blockers*.

Nonselective beta-adrenergic blockers, which block both $beta_1$ and $beta_2$ receptor sites, reduce stimulation of the heart and cause the bronchioles of the lungs to constrict. This can cause bronchospasm in patients with chronic obstructive lung disorders.



Antilipemics

Antilipemic drugs are used to lower abnormally high blood levels of lipids, including cholesterol, triglycerides, and phospholipids.

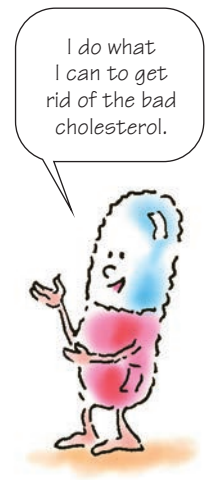
Lipid-busting combo

Antilipemics can be used in combination with lifestyle changes (proper diet, weight loss, and exercise) to lower a patient's lipid level.

A class act

Major classes of antilipemic drugs include:

- bile-sequestering drugs
- fibric acid derivatives
- HMG-CoA reductase inhibitors
- cholesterol absorption inhibitors. (See *Understanding antilipemics*, page 218.)



Bile-sequestering drugs

Bile-sequestering drugs help lower blood levels of low-density lipoproteins (LDLs, or bad cholesterol) by combining with bile acids in the intestines to form an insoluble compound that's then excreted in the feces.

Follow the exit signs, please

The decreasing level of bile acid in the gallbladder triggers the liver to synthesize more bile acids from their precursor, cholesterol. As cholesterol leaves the bloodstream and other storage areas to replace the lost bile acids, blood cholesterol levels decrease.

It's all in the family

Bile-sequestering drugs are the drugs of choice for treating familial hypercholesterolemia when the patient isn't able to reduce his LDL levels through dietary changes. Examples of bile-sequestering drugs include:

- cholestyramine (Questran)
- colesevelam (Welchol)
- colestipol hydrochloride (Colestid).

Understanding antilipemics

Antilipemics are used to lower high blood levels of lipids by combining with bile acids, reducing cholesterol formation, inhibiting enzymes, and inhibiting cholesterol absorption.

Use this table to learn the indications, adverse reactions, and practice pointers needed to safely administer these drugs.

Drugs	Indications	Adverse reactions	Practice pointers
Bile-sequestering drugs			
Cholestyramine (Questran), colesevelam (Welchol), colestipol (Colestid)	• Elevated serum cholesterol	• Constipation • Increased bleeding tendencies • Muscle and joint pain • Nausea, heartburn • Headache	• Tell the patient he'll need periodic blood tests. • Administer the drug before meals. • Don't administer the powder in dry form; mix with fluid. • Administer other medications 1 hour before or 4 to 6 hours after these drugs.
Fibric acid derivatives			
Fenofibrate (TriCor), gemfibrozil (Lopid)	• Hypercholesterolemia • Hypertriglyceridemia	• Rash, nausea, vomiting, diarrhea • Myalgia, flulike syndromes • Impotence • Dizziness, blurred vision • Abdominal pain, epigastric pain	• Tell the patient he'll need periodic blood tests. • Educate the patient on dietary and lifestyle changes to help lower cholesterol and triglyceride levels. • Administer these drugs with meals.
HMG-CoA reductase inhibitors			
Atorvastatin (Lipitor), fluvastatin (Lescol), lovastatin (Mevacor), pravastatin (Pravachol), simvastatin (Zocor), rosuvastatin (Crestor)	• Elevated cholesterol, triglyceride, and LDL levels • Prevention of cardiovascular disease in adults without clinically evident coronary disease but with multiple risk factors	• Rhabdomyolysis with acute renal failure • Headache • Flatulence, abdominal pain, constipation, nausea	• Tell the patient he'll need periodic blood tests. • Monitor periodic liver function tests. • Administer the drug at the same time each day; doesn't need to be administered with food. • Educate the patient on dietary and lifestyle changes to help lower cholesterol and triglyceride levels.
Cholesterol absorption inhibitors			
Ezetimibe (Zetia)	• Elevated cholesterol, triglyceride, and LDL levels • May be administered as adjunctive treatment with simvastatin	• Cough • Myalgia, arthralgia • Headache, dizziness	• Tell the patient he'll need periodic blood tests. • Educate the patient on dietary and lifestyle changes to help lower cholesterol and triglyceride levels. • If administering with an HMG-CoA reductase inhibitor, administer both drugs together.

Fibric acid derivatives

Fibric acid derivatives reduce high triglyceride levels and, to a lesser extent, high LDL levels.

Keeping the highs low

It isn't known exactly how these drugs work, although it's thought that they:

- reduce cholesterol production early in its formation
- mobilize cholesterol from the tissues
- increase cholesterol excretion
- decrease synthesis and secretion of lipoproteins
- decrease synthesis of triglycerides.

A common answer

Fenofibrate (TriCor) and gemfibrozil (Lopid), two commonly used fibric acid derivatives, both reduce triglyceride levels and blood cholesterol levels. Gemfibrozil also increases the high-density lipoprotein (HDL) levels in the blood and increases the serum's capacity to dissolve additional cholesterol.

HMG-CoA reductase inhibitors

Also known as *statins*, HMG-CoA reductase inhibitors lower lipid levels by interfering with cholesterol synthesis. More specifically, they inhibit the enzyme that's responsible for converting HMG-CoA to mevalonate, an early rate-limiting step in the biosynthesis of cholesterol.

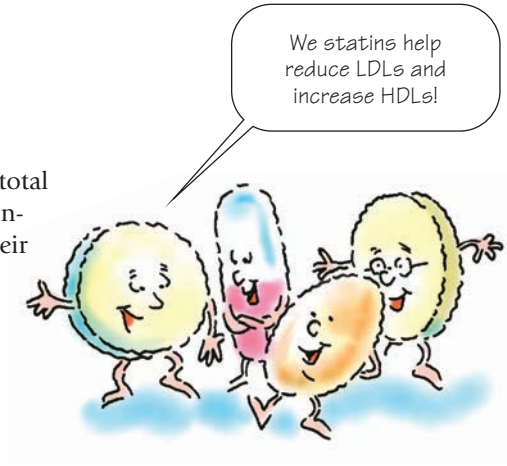
Statins with status

Commonly prescribed HMG-CoA reductase inhibitors include:

- atorvastatin (Lipitor)
- fluvastatin (Lescol)
- lovastatin (Mevacor)
- pravastatin (Pravachol)
- simvastatin (Zocor)
- rosuvastatin (Crestor).

Highs, lows, and a bonus or two

Statin drugs are used primarily to reduce LDLs and total blood cholesterol levels. They also produce a mild increase in HDLs (or good cholesterol). Because of their effect on LDL and total cholesterol, these drugs are used not only to treat hypercholesterolemia but also for primary and secondary prevention of cardiovascular events.



Cholesterol absorption inhibitors

As their name implies, cholesterol absorption inhibitors inhibit the absorption of cholesterol and related phytosterols from the intestine.

In a class by itself

At this time, ezetimibe (Zetia) is the only drug in the class. Ezetimibe reduces blood cholesterol levels by inhibiting the absorption of cholesterol by the small intestine. This leads to a decrease in delivery of intestinal cholesterol to the liver, causing a reduction in hepatic cholesterol stores and an increase in clearance from the blood.

A two-drug punch

Ezetimibe may be used alone or with statins to help lower cholesterol. There is currently one drug on the market that combines the statin simvastatin and ezetimibe (Vytorin) to help decrease total cholesterol and LDLs and increase HDLs.

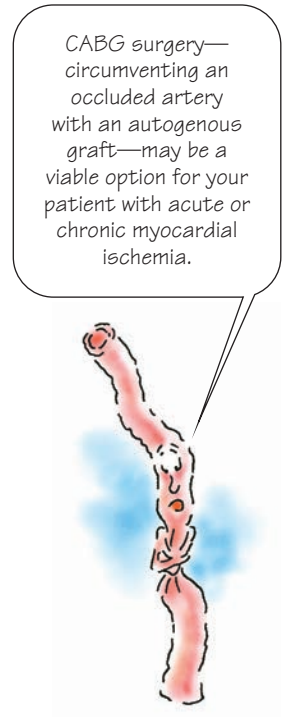
Surgery

Surgeries for treatment of cardiovascular system disorders include coronary artery bypass graft (CABG), heart transplantation, valve surgery, vascular repair, and insertion of a VAD.

Coronary artery bypass graft

CABG circumvents an occluded coronary artery with an autogenous graft (usually a segment of the saphenous vein from the leg or internal mammary artery), thereby restoring blood flow to the myocardium.

CABG is one of the most commonly performed surgeries because it's done to prevent MI in a patient with acute or chronic myocardial ischemia. The need for CABG is determined from the results of cardiac catheterization and patient symptoms. (See *Bypassing coronary occlusions*.)



Why bypass?

If successful, CABG can relieve anginal pain, improve cardiac function, and possibly enhance the patient's quality of life.

CABG varieties

CABG techniques vary according to the patient's condition and the number of arteries being bypassed.

Other surgical techniques, such as the mini-CABG and direct coronary artery bypass, can reduce the risk for cerebral complications

Bypassing coronary occlusions

After the patient receives general anesthesia, surgery begins with graft harvesting. The surgeon makes a series of incisions in the patient's thigh or calf and removes a saphenous vein segment for grafting. Most surgeons prefer to use a segment of the internal mammary artery.

Exposing the heart

After the autografts are obtained, the surgeon performs a medial sternotomy to expose the heart and then initiates cardiopulmonary bypass.

To reduce myocardial oxygen demands during surgery and to protect the heart, the surgeon induces cardiac hypothermia and standstill by injecting a cold cardioplegic solution (potassium-enriched saline solution) into the aortic root.

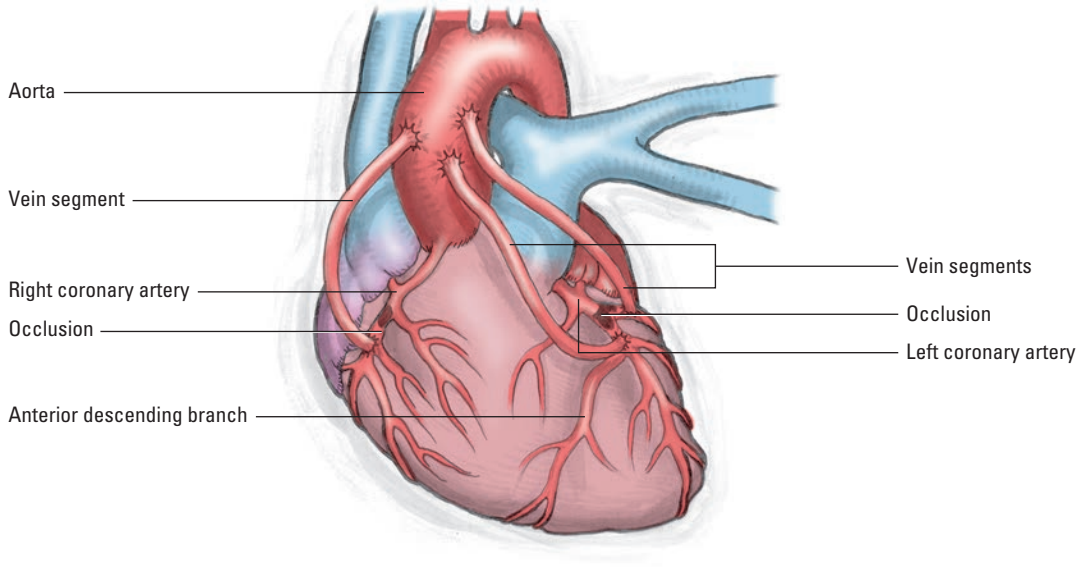
One fine sewing lesson

After the patient is prepared, the surgeon sutures one end of the venous graft to the ascending aorta and the other end to a patent coronary artery that's distal to the occlusion. The graft is sutured in a reversed position to promote proper blood flow. The surgeon repeats this procedure for each occlusion to be bypassed.

In the example depicted below, saphenous vein segments bypass occlusions in three sections of the coronary arteries.

Finishing up

After the grafts are in place, the surgeon flushes the cardioplegic solution from the heart and discontinues cardiopulmonary bypass. He then implants epicardial pacing electrodes, inserts a chest tube, closes the incision, and applies a sterile dressing.

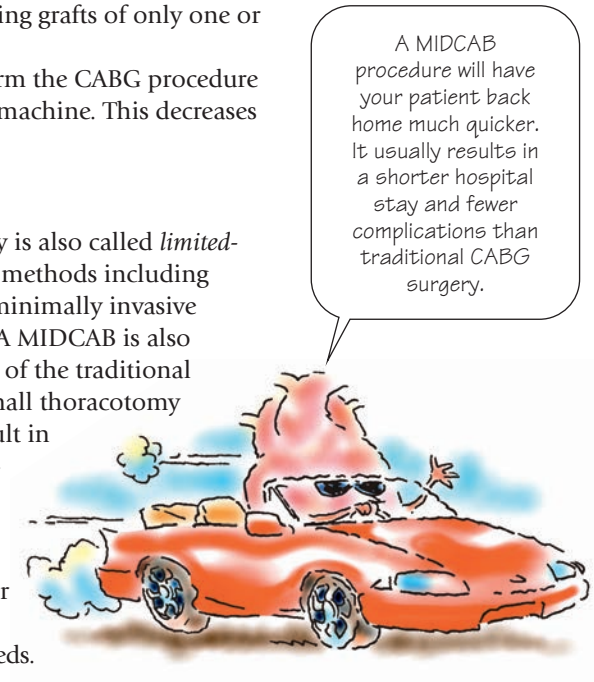


and accelerate recovery for patients requiring grafts of only one or two arteries.

In some patients, it's possible to perform the CABG procedure without using a cardiopulmonary bypass machine. This decreases recovery time and complications.

Short and sweet

Minimally invasive coronary artery surgery is also called *limited-access coronary surgery*. It has two standard methods including port-access coronary bypass (PACB) and minimally invasive direct coronary artery bypass (MIDCAB). A MIDCAB is also performed on a beating heart, but instead of the traditional midsternal incision, the surgeon uses a small thoracotomy incision. MIDCAB procedures usually result in shorter hospital stays and fewer complications than traditional CABG.



Nursing considerations

When caring for a CABG patient, your major roles include patient instruction and caring for the patient's changing cardiovascular needs.

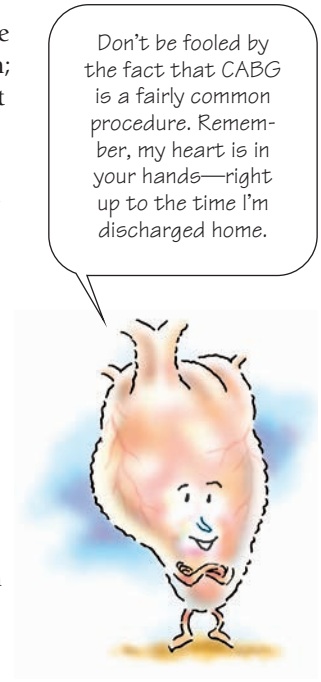
Before surgery

- Reinforce the doctor's explanation of the surgery.
- Explain the complex equipment and procedures used in the critical care unit (CCU) or postanesthesia care unit (PACU).
- Explain that the patient awakens from surgery with an endotracheal (ET) tube in place and connected to a mechanical ventilator. He'll also be connected to a cardiac monitor and may have in place an NG tube, a chest tube, an indwelling urinary catheter, arterial lines, epicardial pacing wires, and a PA catheter. Tell him that discomfort is minimal and that the equipment is removed as soon as possible.
- Review incentive spirometry techniques and range-of-motion (ROM) exercises with the patient.
- Make sure that the patient or a responsible family member has signed a consent form.
- Before surgery, prepare the patient's skin as ordered.
- Immediately before surgery, begin cardiac monitoring and then assist with PA catheterization and insertion of arterial lines. Some facilities insert PA catheters and arterial lines in the operating room before surgery.

After surgery

- After CABG, look for signs of hemodynamic compromise, such as severe hypotension, decreased cardiac output, and shock.
- Begin warming procedures according to your facility's policy.

- Check and record vital signs and hemodynamic parameters every 5 to 15 minutes until the patient's condition stabilizes. Administer medications and titrate according to the patient's response, as ordered.
- Monitor ECGs continuously for disturbances in heart rate and rhythm. If you detect serious abnormalities, notify the practitioner and be prepared to assist with epicardial pacing or, if necessary, cardioversion or defibrillation.
- To ensure adequate myocardial perfusion, keep arterial pressure within the limits set by the doctor. Usually, mean arterial pressure (MAP) less than 70 mm Hg results in inadequate tissue perfusion; pressure greater than 110 mm Hg can cause hemorrhage and graft rupture. Monitor PAP, CVP, left atrial pressure, and cardiac output as ordered.
- Frequently evaluate the patient's peripheral pulses, capillary refill time, and skin temperature and color and auscultate for heart sounds; report abnormalities.
- Evaluate tissue oxygenation by assessing breath sounds, chest excursion, and symmetry of chest expansion. Check ABG results every 2 to 4 hours and adjust ventilator settings to keep ABG values within ordered limits.
- Maintain chest tube drainage at the ordered negative pressure (usually −10 to −40 cm H_2O) and assess regularly for hemorrhage, excessive drainage (greater than 200 ml/hour), and sudden decrease or cessation of drainage.
- Monitor the patient's intake and output. Assess urine output at least hourly during the immediate postoperative period and then less frequently as the patient's condition stabilizes.
- Assess for electrolyte imbalances, especially hypokalemia and hypomagnesemia, and replace electrolytes as ordered.
- As the patient's incisional pain increases, give an analgesic as ordered. Give other drugs as ordered.
- Throughout the recovery period, assess for symptoms of stroke, pulmonary embolism, and impaired renal perfusion.
- After weaning the patient from the ventilator and removing the ET tube, provide chest physiotherapy. Start with incentive spirometry and encourage the patient to cough, turn frequently, and deep breathe. Assist with ROM exercises, as ordered, to enhance peripheral circulation and prevent thrombus formation.
- Explain that postpericardiotomy syndrome commonly develops after open heart surgery. Instruct the patient about signs and symptoms, such as fever, muscle and joint pain, weakness, and chest discomfort.
- Prepare the patient for the possibility of postoperative depression, which may not develop until weeks after discharge. Reassure him that this depression is normal and should pass quickly.



- Maintain nothing by mouth status until bowel sounds return. Then begin clear liquids and advance diet as tolerated and as ordered. Expect sodium and cholesterol restrictions. Explain that this diet can help reduce the risk of recurrent arterial occlusion.

Transmyocardial revascularization

Transmyocardial revascularization (TMR) uses a high-energy laser to create channels from the epicardial surface into the left ventricular chamber. The purpose of the TMR is to increase perfusion directly to the heart muscle. This is performed on patients who are poor candidates for CABG and whose symptoms are not responding to other medical treatments.

Heart transplantation

Heart transplantation involves the replacement of a person's heart with a donor heart. It's the treatment of choice for patients with end-stage cardiac disease that have a poor prognosis, estimated survival of 6 to 12 months, and poor quality of life. A heart transplant candidate typically has uncontrolled symptoms and no other surgical options.

No guarantee

Transplantation doesn't guarantee a cure. Serious postoperative complications include infection and tissue rejection. Most patients experience one or both of these complications postoperatively.

Rejection and infection

Rejection typically occurs in the first 6 weeks after surgery. The patient is treated with monoclonal antibodies and potent immunosuppressants. The resulting immunosuppression places the patient at risk for life-threatening infection.

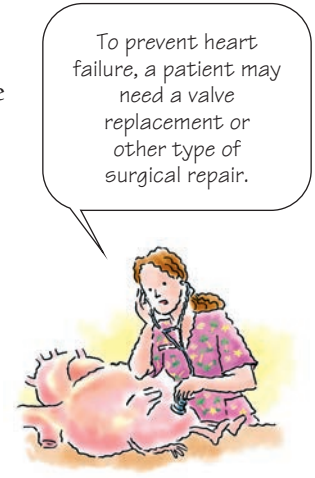
Nursing considerations

- Provide emotional support to the patient and his family. Begin to address their fears by discussing the procedure, possible complications, and the impact of transplantation and a prolonged recovery period on the patient's life.
- After surgery, maintain reverse isolation.
- Administer immunosuppressants and monitor the patient closely for signs of infection. Transplant recipients may exhibit subtle signs because immunosuppressants mask obvious signs.
- Monitor vital signs every 15 minutes until stabilized and assess the patient for signs of hemodynamic compromise, such as hypotension, decreased cardiac output, and shock.

- If necessary, administer nitroprusside during the first 24 to 48 hours to control blood pressure. An infusion of dopamine can improve contractility and renal perfusion.
- Volume replacement with normal saline, plasma expanders, or blood products may be necessary to maintain CVP.
- A patient with elevated PAP may receive prostaglandin E to produce pulmonary vasodilation and reduce right ventricular afterload.
- Monitor ECG for rhythm disturbances.
- Maintain the chest tube drainage system at the prescribed negative pressure. Regularly assess for hemorrhage or sudden cessation of drainage.
- Continually assess the patient for signs of tissue rejection (decreased electrical activity on the ECG, right axis shift, atrial arrhythmias, conduction defects, weight gain, lethargy, ventricular failure, jugular vein distention, and increased T-cell count).
- Keep in mind that the effects of denervated heart muscle or denervation (in which the vagus nerve is cut during heart transplant surgery) makes such drugs as edrophonium (Tensilon) and anticholinergics (such as atropine) ineffective.

Valve surgery

To prevent heart failure, a patient with valvular stenosis or insufficiency accompanied by severe, unmanageable symptoms may require valve replacement (with a mechanical or prosthetic valve), valvular repair, or commissurotomy. (See *Types of valve surgery*, page 226.)



Why valve surgery?

Because of the high pressure generated by the left ventricle during contraction, stenosis and insufficiency most commonly affect the mitral and aortic valves. Other indications for valve surgery depend on the patient's symptoms and affected valve:

- In aortic insufficiency, the patient undergoes valve replacement after symptoms—palpitations, dizziness, dyspnea on exertion, angina, and murmurs—have developed or if the chest X-ray and ECG reveal left ventricular hypertrophy.
- In aortic stenosis, which may be asymptomatic, the practitioner may recommend valve replacement if cardiac catheterization reveals significant stenosis.
- In mitral stenosis, surgery is indicated if the patient develops fatigue, dyspnea, hemoptysis, arrhythmias, pulmonary hypertension, or right ventricular hypertrophy.
- In mitral insufficiency, surgery is usually done when the patient's symptoms—dyspnea, fatigue, and palpitations—interfere with ADLs or if insufficiency is acute, as in papillary muscle rupture.

Types of valve surgery

When a patient with valve disease develops severe symptoms, surgery may be necessary. Several surgical procedures are available.

Commissurotomy
During commissurotomy, the surgeon incises fused mitral valve leaflets and removes calcium deposits to improve valve mobility.

Valve repair
Valve repair includes resection or patching of valve leaflets, stretching or shortening of chordae tendineae, or placing a ring in a dilated annulus (annuloplasty). Valve repair is done to avoid the complications associated with the use of prosthetic valves.

Valve replacement
Valvular replacement involves replacement of the patient's diseased valve with a mechanical or biologic valve.

In the Ross procedure, the patient's own pulmonic valve is excised and used to replace the diseased aortic valve. An allograft from a human cadaver is then used to replace the pulmonic valve. Advantages of this procedure include the potential for the pulmonary autograft to grow when used in children, anticoagulation isn't necessary, and the increased durability of the replaced valves.

Minimally invasive valve surgery
Minimally invasive valve surgery can be performed without a large median sternotomy incision to repair or replace aortic and mitral valves. Port access techniques may also be used for mitral valve surgery using endovascular cardiopulmonary bypass. Advantages of these types of surgery include a less invasive procedure, shorter hospital stays, fewer postoperative complications, reduced costs, and smaller incisions.

Nursing considerations

Provide these care measures after valve surgery.

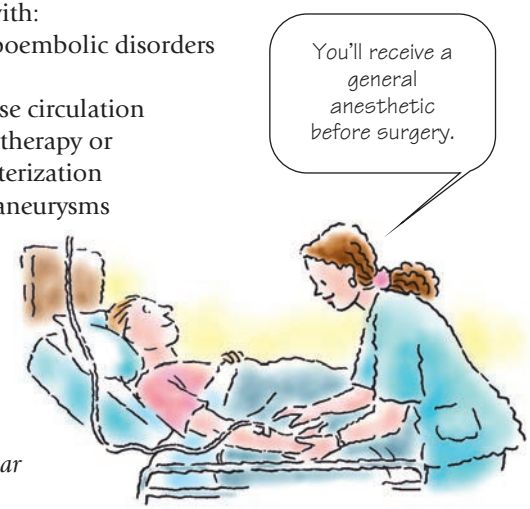
- Closely monitor the patient's hemodynamic status for signs of compromise. Watch especially for severe hypotension, decreased cardiac output, and shock. Check and record vital signs every 15 minutes until his condition stabilizes. Frequently assess heart sounds; report distant heart sounds or new murmurs, which may indicate prosthetic valve failure.
- Monitor the ECG continuously for disturbances in heart rate and rhythm, such as bradycardia, atrial fibrillation, ventricular tachycardia, and heart block. Such disturbances may signal injury of the conduction system, which may occur during valve surgery from proximity of the atrial and mitral valves to the AV node. Arrhythmias may also result from myocardial irritability or ischemia, fluid and electrolyte imbalance, hypoxemia, or hypothermia. If you detect serious abnormalities, notify the practitioner and be prepared to assist with temporary epicardial pacing.

- Take steps to maintain the patient's MAP between 70 and 100 mm Hg. Also, monitor PAP and left atrial pressure as ordered.
- Frequently assess the patient's peripheral pulses, capillary refill time, and skin temperature and color and auscultate for heart sounds. Evaluate tissue oxygenation by assessing breath sounds, chest excursion, and symmetry of chest expansion. Report any abnormalities.
- Check ABG values every 2 to 4 hours and adjust ventilator settings as needed.
- Maintain chest tube drainage at the prescribed negative pressure (usually −10 to −40 cm H_2O for adults). Assess chest tubes frequently for signs of hemorrhage, excessive drainage (greater than 200 ml/hour), and a sudden decrease or cessation of drainage.
- As ordered, administer analgesic, anticoagulant, antibiotic, antiarrhythmic, inotropic, and pressor medications as well as I.V. fluids and blood products. Monitor intake and output and assess for electrolyte imbalances, especially hypokalemia. When anticoagulant therapy begins, evaluate its effectiveness by monitoring prothrombin time and International Normalized Ratio daily.
- Throughout the patient's recovery period, observe carefully for complications.
- After weaning from the ventilator and removing the ET tube, promote chest physiotherapy. Start the patient on incentive spirometry and encourage him to cough, turn frequently, and deep breathe.

Vascular repair

Vascular repair may be needed to treat patients with:

- vessels damaged by arteriosclerotic or thromboembolic disorders trauma, infections, or congenital defects
- vascular obstructions that severely compromise circulation
- vascular disease that doesn't respond to drug therapy or nonsurgical treatments such as balloon catheterization
- life-threatening dissecting or ruptured aortic aneurysms
- limb-threatening acute arterial occlusion.



Repair review

Vascular repair methods include aneurysm resection, grafting, embolectomy, vena caval filtering, and endarterectomy. The surgery used depends on the type, location, and extent of vascular occlusion or damage. (See *Types of vascular repair*, page 228.)

Nursing considerations

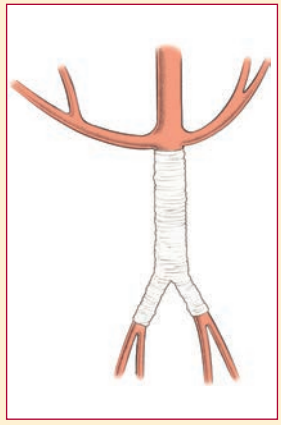
Provide care measures before and after vascular repair surgery.

Types of vascular repair

There are several surgical options to repair damaged or diseased vessels. Some of these options are aortic aneurysm repair, vena caval filter insertion, embolectomy, and bypass grafting.

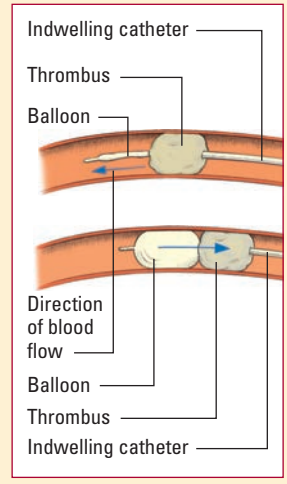
Aortic aneurysm repair

Aortic aneurysm repair is done to remove an aneurysmal segment of the aorta. The surgeon first makes an incision to expose the aneurysm site. The patient is placed on a cardiopulmonary bypass machine, if necessary. The surgeon then clamps the aorta. The aneurysm is resected, and the damaged portion of the aorta is repaired.



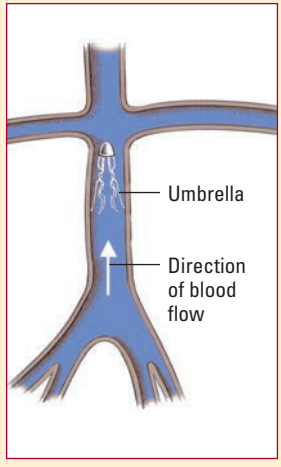
Embolectomy

An embolectomy is done to remove an embolism from an artery. During this procedure, the surgeon inserts a balloon-tipped indwelling catheter into the artery and passes it through the thrombus (top). He then inflates the balloon and withdraws the catheter to remove the thrombus (bottom).



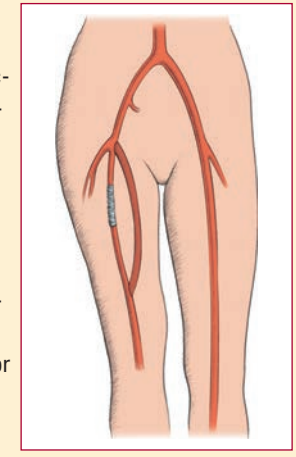
Vena caval filter insertion

A vena caval filter is inserted to trap emboli in the vena cava, preventing them from reaching the pulmonary vessels. A vena caval filter or umbrella is inserted transvenously by catheter. After in place in the vena cava, the umbrella or filter traps emboli but allows venous blood flow.



Bypass grafting

Bypass grafting is used to bypass an arterial obstruction resulting from arteriosclerosis. After exposing the affected artery, the surgeon anastomoses a synthetic or autogenous graft to divert blood flow around the occluded arterial segment. The autogenous graft may be a vein or an artery harvested from elsewhere in the patient's body. A femoropopliteal bypass is depicted.



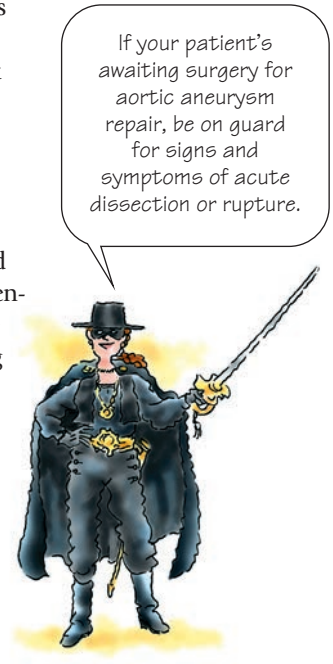
Before surgery

- Make sure the patient and his family understand the doctor's explanation of the surgery and possible complications.
- Tell the patient that he'll receive a general anesthetic and will awaken from the anesthetic in the CCU or PACU. Explain that he'll have an I.V. line in place, ECG electrodes for continuous cardiac monitoring,

and possibly an arterial line or a PA catheter to provide continuous pressure monitoring. He may also have a urinary catheter in place to allow accurate output measurement. If appropriate, explain that he'll be intubated and placed on mechanical ventilation.

Flow check

- Before surgery, perform a complete vascular assessment. Take vital signs to provide a baseline. Evaluate the strength and sound of the blood flow and the symmetry of the pulses and note bruits. Record the temperature of the extremities; their sensitivity to motor and sensory stimuli; and pallor, cyanosis, or redness. Rate peripheral pulse volume and strength on a scale of 0 (pulse absent) to 4 (bounding and strong pulse) and check capillary refill time by blanching the fingernail or toenail; normal refill time is less than 3 seconds.
- As ordered, instruct the patient to restrict food and fluids for at least 8 hours before surgery.



Be on guard!

- If the patient is awaiting surgery for aortic aneurysm repair, be on guard for signs and symptoms of acute dissection or rupture. Note especially sudden severe pain in the chest, abdomen, or lower back; severe weakness; diaphoresis; tachycardia; or a precipitous drop in blood pressure. If any of these occur, notify the practitioner immediately.

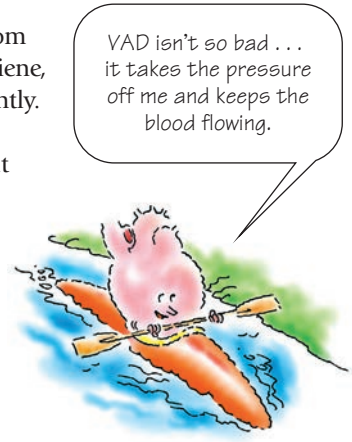
After surgery

- Check and record the patient's vital signs every 15 minutes until his condition stabilizes, then every 30 minutes for 1 hour, and hourly thereafter for 2 to 4 hours. Report hypotension and hypertension immediately.
- Auscultate heart, breath, and bowel sounds and report abnormal findings. Monitor the ECG for abnormalities in heart rate or rhythm. Also monitor other pressure readings and carefully record intake and output.
- Check the patient's dressing regularly for excessive bleeding.
- Assess the patient's neurologic and renal function and report abnormalities.
- Provide analgesics, as ordered, for incisional pain.
- Frequently assess peripheral pulses, using Doppler ultrasonography if palpation is difficult. Check all extremities bilaterally for muscle strength and movement, color, temperature, and capillary refill time.

Watch those wounds!

- Change dressings and provide incision care as ordered. Position the patient to avoid pressure on grafts and to reduce edema. Administer antithrombotics, as ordered, and monitor appropriate laboratory values to evaluate effectiveness.

- Assess for complications and immediately report relevant signs and symptoms. (See *Vascular repair complications*.)
- As the patient's condition improves, take steps to wean him from the ventilator if appropriate. To promote good pulmonary hygiene, encourage the patient to cough, turn, and breathe deep frequently.
- Assist the patient with ROM exercises, as ordered, to prevent thrombus formation. Assist with early ambulation to prevent complications of immobility.



Ventricular assist device insertion

A VAD is a device that's implanted to provide support to a failing heart. A VAD consists of:

- a blood pump
- cannulas
- pneumatic or electrical drive console.

Vascular repair complications

After a patient has undergone vascular repair surgery, monitor for these potential complications.

Complication	Signs and symptoms
Pulmonary infection	• Fever • Cough • Congestion • Dyspnea
Infection	• Redness • Warmth • Drainage • Increased pain • Fever
Renal dysfunction	• Low urine output • Elevated BUN and serum creatinine levels
Occlusion	• Reduced or absent peripheral pulses • Paresthesia • Severe pain • Cyanosis
Hemorrhage	• Hypotension • Tachycardia • Restlessness and confusion • Shallow respirations • Abdominal pain • Increased abdominal girth

More output, less work

VADs are designed to decrease the heart's workload and increase cardiac output in patients with ventricular failure.

A temporary diversion

A VAD is commonly used while a patient waits for a heart transplant. In a surgical procedure, blood is diverted from a ventricle to an artificial pump. This pump, which is synchronized to the patient's ECG, then functions as the ventricle. (See *VAD: Help for a failing heart.*)

That's shocking!

VADs are also indicated for use in patients with cardiogenic shock that doesn't respond to maximal pharmacologic therapy or inability to be weaned from cardiopulmonary bypass.

VAD: Help for a failing heart

A VAD, which is commonly called a *bridge to transplant*, is a mechanical pump that relieves the workload of the ventricle as the heart heals or until a donor heart is located.

Implantable

The typical VAD is implanted in the upper abdominal wall. An inflow cannula drains blood from the left ventricle into a pump, which then pushes the blood into the aorta through the outflow cannula.

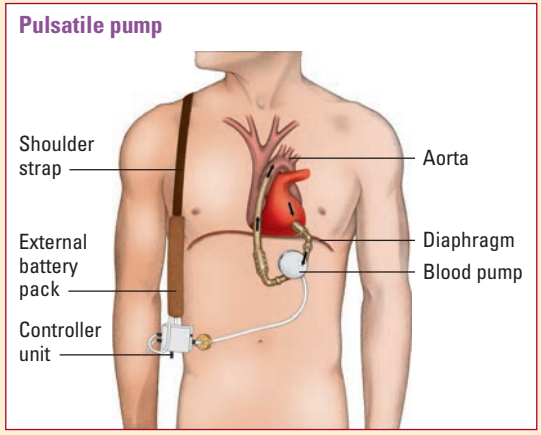
Pump options

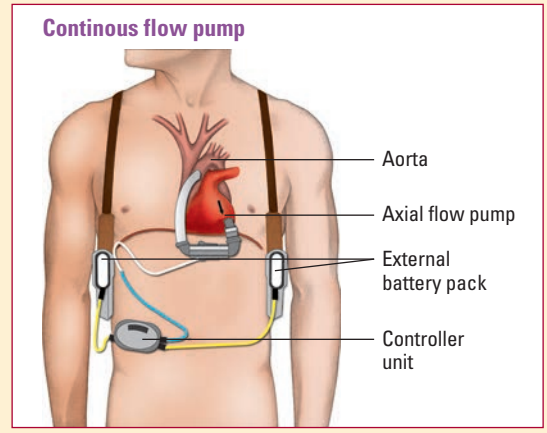
VADs are available as continuous flow (axial flow) or pulsatile pumps. A continuous flow pump fills continuously and returns blood to the aorta at a constant rate. A pulsatile pump may work in one of two ways: It may fill during systole and pump blood into the aorta during diastole, or it may pump irrespective of the patient's cardiac cycle.

Many types of VAD systems are available. The illustrations below show a pulsatile pump and a continuous flow pump. Each has an external controller and a reserve power pack.

Potential complications

Despite the use of anticoagulants, the VAD may cause thrombi formation, leading to pulmonary embolism or stroke. Other complications may include heart failure, bleeding, cardiac tamponade, or infection.





Right or left?

A VAD is used to provide systemic or pulmonary support, or both:

- A right ventricular assist device (RVAD) provides pulmonary support by diverting blood from the failing right ventricle to the VAD, which then pumps the blood to the pulmonary circulation by way of the VAD connection to the pulmonary artery.
- With a left ventricular assist device (LVAD), blood flows from the left ventricle to the VAD, which then pumps blood back to the body by way of the VAD connection to the aorta.
- When RVAD and LVAD are used, biventricular support is provided.

Nursing considerations

Follow these care measures before and after insertion of a VAD.

Before insertion

- Prepare the patient and his family for insertion, reinforcing explanations about the device, its purpose, and what to expect after insertion.
- Make sure that informed consent is obtained.
- Continue close patient monitoring, including continuous ECG, pulmonary artery and hemodynamic status, and intake and output.

After insertion

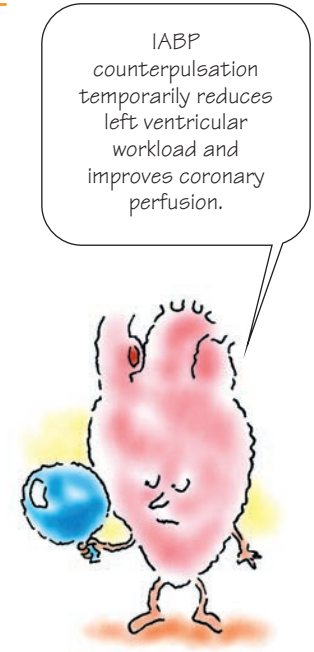
- Assess the patient's cardiovascular status at least every 15 minutes until stable and then hourly. Monitor blood pressure and hemodynamic parameters, including cardiac output and cardiac index, ECG, and peripheral pulses.
- Inspect the incision and dressing at least every hour initially and then every 2 to 4 hours as indicated by the patient's condition.
- Monitor urine output hourly and maintain I.V. fluid therapy as ordered. Watch for signs of fluid overload or decreasing urine output.
- Assess chest tube drainage and function frequently. Notify the practitioner if drainage is greater than 150 ml over 2 hours. Auscultate lungs for evidence of abnormal breath sounds. Evaluate oxygen saturation or mixed venous oxygen saturation levels and administer oxygen as needed and ordered.
- Obtain hemoglobin levels, hematocrit, and coagulation studies as ordered. Administer blood component therapy as indicated and ordered.
- Assess for signs and symptoms of bleeding.
- Turn the patient every 2 hours and begin ROM exercises when he's stable.
- Administer antibiotics prophylactically if ordered.

Balloon catheter treatments

Balloon catheter treatments of cardiovascular system disorders include IABP counterpulsation, balloon valvuloplasty, and percutaneous transluminal coronary angioplasty (PTCA).

IABP counterpulsation

IABP counterpulsation temporarily reduces left ventricular workload and improves coronary perfusion.



What for?

IABP counterpulsation may benefit patients with:

- cardiogenic shock
- septic shock
- intractable angina before surgery
- intractable ventricular arrhythmias
- ventricular septal or papillary muscle ruptures
- acute MI with left ventricular failure.

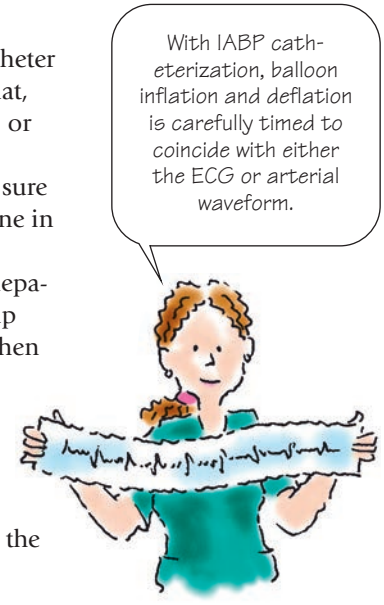
It's also used for patients who suffer pump failure before, during, or after cardiac surgery and serves as a bridge to other treatments, such as VAD, CABG, or heart transplant.

How so?

The doctor may perform balloon catheter insertion at the patient's bedside as an emergency procedure or in the operating room. (See *Understanding a balloon pump*, page 234.)

Nursing considerations

- Explain to the patient that the doctor is going to place a catheter in the aorta to help his heart pump more easily. Tell him that, while the catheter is in place, he can't sit up, bend his knee, or flex his hip more than 45 degrees.
- Attach the patient to a continuous ECG monitor and make sure he has an arterial line, a PA catheter, and a peripheral I.V. line in place.
- Gather a surgical tray for percutaneous catheter insertion, heparin, normal saline solution, the IABP catheter, and the pump console. Connect the ECG monitor to the pump console. Then prepare the femoral site.
- After the IABP catheter is inserted, select either the ECG or arterial waveform to regulate inflation and deflation of the balloon. With the ECG waveform, the pump inflates the balloon in the middle of the T wave (diastole) and deflates with the R wave (before systole). With the arterial waveform, the

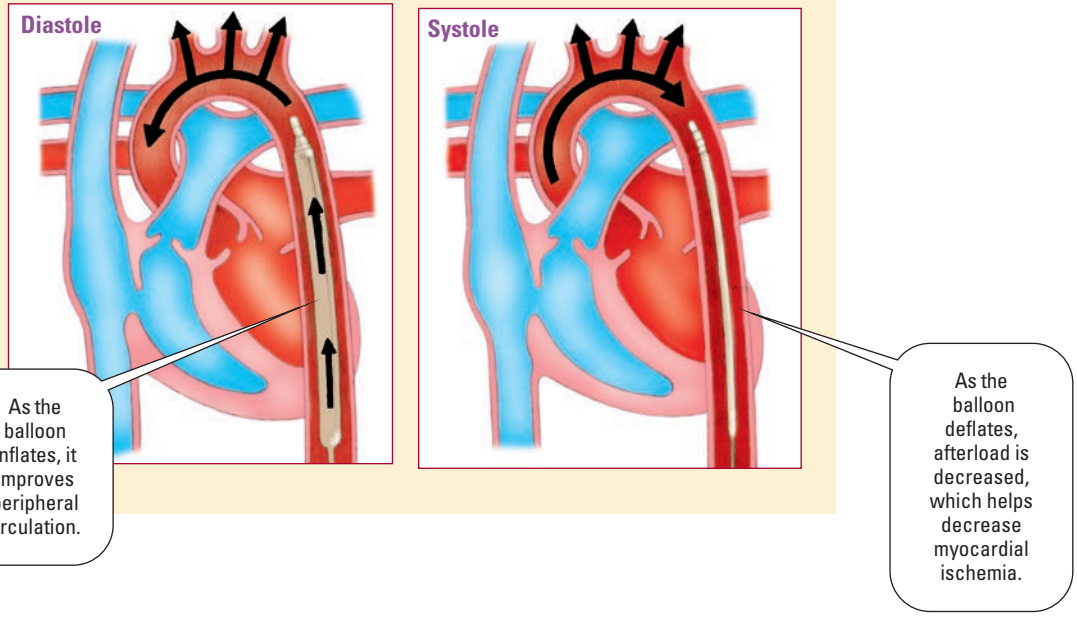


Understanding a balloon pump

An 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.

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.

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.



upstroke of the arterial wave triggers balloon inflation. (See *Timing IABP counterpulsation.*)

- Frequently assess the insertion site. Don't elevate the head of the bed more than 45 degrees to prevent upward migration of the catheter and occlusion of the left subclavian artery. If the balloon occludes the artery, you may see a diminished left radial pulse, and the patient may report dizziness. Incorrect balloon placement may also cause flank pain or a sudden decrease in urine output.

Timing IABP counterpulsation

IABP counterpulsation is synchronized with either the ECG or arterial waveform. Ideally, balloon inflation should begin just after the aortic valve closes—at the dicrotic notch on the arterial waveform. Deflation should occur just before systole.

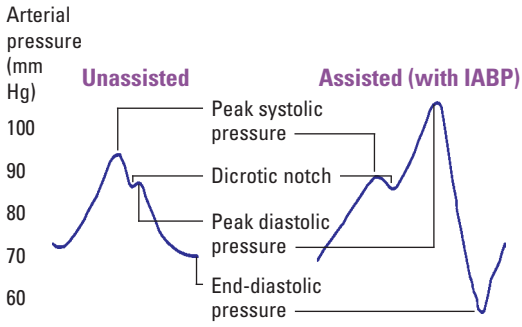
Proper timing is crucial

Early inflation can damage the aortic valve by forcing it closed, whereas late inflation permits most of the blood emerging from the ventricle to flow past the balloon, reducing pump effectiveness.

Late deflation increases the resistance against which the left ventricle must pump, possibly causing cardiac arrest.

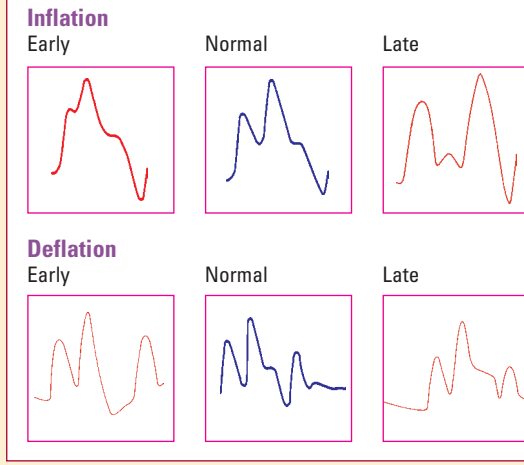
Arterial waveforms

The illustration below depicts how IABP counterpulsation boosts peak diastolic pressure and lowers peak systolic and end-diastolic pressures.



How timing affects waveforms

The arterial waveforms below show correctly and incorrectly timed balloon inflation and deflation.



- Assess distal pulses, color, temperature, and capillary refill of the patient's extremities every 15 minutes for the first 4 hours after insertion. After 4 hours, assess hourly for the duration of IABP therapy.
- Watch for signs of thrombus formation, such as a sudden weakening of pedal pulses, pain, and motor or sensory loss.
- If indicated, apply antiembolism stockings.
- Encourage active ROM exercises every 2 hours for the arms, the unaffected leg, and the affected ankle.
- Maintain adequate hydration to help prevent thrombus formation.
- If bleeding occurs, apply direct pressure and notify the doctor.
- Assess the catheter insertion site every 2 hours.

- Assess the patient's cardiovascular and respiratory status at least every 4 hours. If possible, place the IABP on standby to eliminate any extraneous sounds.
- Administer anticoagulants as ordered to help prevent thrombus formation.

Warning!

- An alarm on the console may indicate a gas leak from a damaged catheter or ruptured balloon. If the alarm sounds or you see blood in the catheter, shut down the pump console and immediately place the patient in Trendelenburg's position to prevent an embolus from reaching the brain. Then notify the doctor.

Ready to wean

- After the signs and symptoms of left-sided heart failure diminish, and the patient requires only minimal drug support, the doctor begins weaning him from IABP counterpulsation. This may be accomplished by reducing the frequency of pumping or decreasing the balloon volume; a minimum volume or pumping ratio must be maintained to prevent thrombus formation. Most consoles have a flutter function that moves the balloon to prevent clot formation. Use the flutter function when the patient has been weaned from counterpulsation but the catheter hasn't yet been removed.
- To discontinue the IABP, the doctor deflates the balloon, clips the sutures, removes the catheter, and allows the site to bleed for 5 seconds to expel clots.
- After the doctor discontinues the IABP, apply direct pressure for 30 minutes and then apply a pressure dressing. Evaluate the site for bleeding and hematoma formation hourly for the next 4 hours.

Percutaneous balloon valvuloplasty

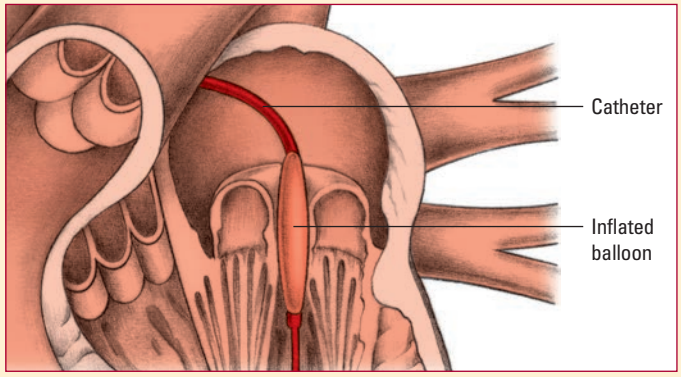
Performed in the cardiac catheterization lab, this procedure aims to improve valvular function by enlarging the orifice of a stenotic heart valve caused by a congenital defect, calcification, rheumatic fever, or aging. It involves introducing a small balloon valvuloplasty catheter through the skin at the femoral vein. (See *Percutaneous balloon valvuloplasty*.)

When surgery isn't the answer

While the treatment of choice for valvular heart disease remains surgery, percutaneous balloon valvuloplasty offers an alternative for individuals considered poor surgical candidates. Unfortunately, elderly patients with aortic disease commonly experience restenosis 1 to 2 years after undergoing valvuloplasty.

Percutaneous balloon valvuloplasty

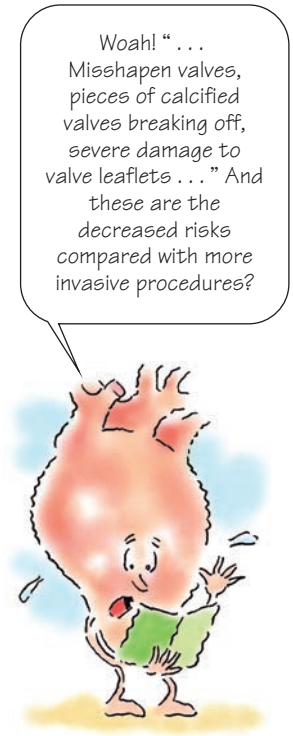
During valvuloplasty, a surgeon inserts a small balloon catheter through the skin at the femoral vein and advances it until it reaches the affected valve. The balloon is then inflated, forcing the valve opening to widen.



Those complicit complications

Despite the decreased risks as compared with some more invasive procedures, balloon valvuloplasty can lead to complications, including:

- worsening valvular insufficiency as a result of misshaping the valve so that it doesn't close completely
- pieces of the calcified valve breaking off, which may travel to the brain or lungs and cause an embolism
- severe damage to the delicate valve leaflets, requiring immediate surgery to replace the valve (rare)
- bleeding and hematoma formation at the arterial puncture site.



Nursing considerations

Provide these care measures before and after percutaneous balloon valvuloplasty.

Before the procedure

- Explain that a catheter will be inserted into an artery in the patient's groin.
- Reassure the patient that, even though he'll be awake during the procedure, he will receive sedation.

Check 'em all off

- Check the patient's history for allergies; if he has had allergic reactions to shellfish, iodine, or contrast media, notify the practitioner.

- Make sure that the results of coagulation studies, CBC, serum electrolyte studies, blood typing and crossmatching, BUN, and serum creatinine are available.
- Obtain baseline vital signs and assess peripheral pulses.
- Apply ECG electrodes and insert an I.V. line if one isn't already in place.
- Perform skin preparation according to your facility's policy.
- Give the patient a sedative as ordered.

After the procedure

- Assess the patient's vital signs and oxygen saturation every 15 minutes for the first hour and then every 30 minutes for 4 hours, unless his condition warrants more frequent checking.

Rhythms, sounds, and pulses

- Monitor his ECG rhythm continuously and assess hemodynamic parameters closely for changes. Be alert for the development of any new cardiac arrhythmias.
- Assess patient's heart and lung sounds at least every 4 hours and notify the practitioner for the development of any new murmurs or signs of valve failure.
- Assess peripheral pulses distal to the catheter insertion site as well as the color, sensation, temperature, and capillary refill time of the affected extremity.

Black and blue or bleeding

- Assess the catheter insertion site for hematoma, ecchymosis, and hemorrhage. If bleeding occurs, locate the artery and apply manual pressure; then notify the practitioner.
- Monitor the patient's neurologic status for any changes and report them to the practitioner immediately.

PTCA

A type of percutaneous coronary intervention, PTCA is a nonsurgical way to open coronary vessels narrowed by arteriosclerosis. It's usually used with cardiac catheterization to assess the stenosis and efficacy of angioplasty. It can also be used as a visual tool to direct the balloon-tipped catheter through a vessel's area of stenosis.

PTCA for pain

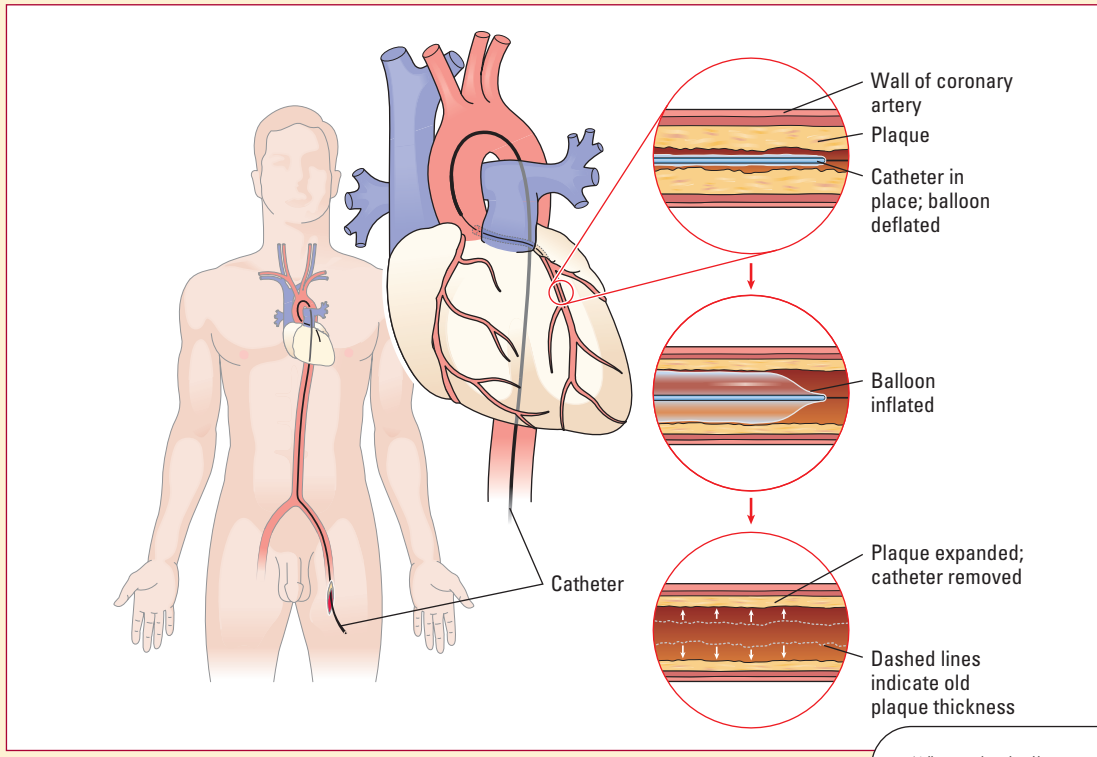
In PTCA, a balloon-tipped catheter is inserted into a narrowed coronary artery. This procedure, performed in the cardiac catheterization laboratory under local anesthesia, relieves pain due to angina and myocardial ischemia. (See *Understanding angioplasty*.)

Understanding angioplasty

PTCA is used to open an occluded coronary artery without opening the chest. This illustration shows what happens during the procedure.

First, the doctor threads the catheter. When angiography shows the guide catheter positioned at the occlusion site, the doctor carefully inserts a smaller double-lumen balloon catheter through the guide catheter and directs the balloon through the occlusion.

After the balloon is directed through the occlusion, the balloon is inflated, resulting in arterial stretching and plaque fracture. The balloon may need to be inflated and deflated several times until successful dilation occurs.



When the balloon is inflated, the plaque is compressed against the vessel wall, allowing coronary blood to flow more freely.

Through one artery and into another

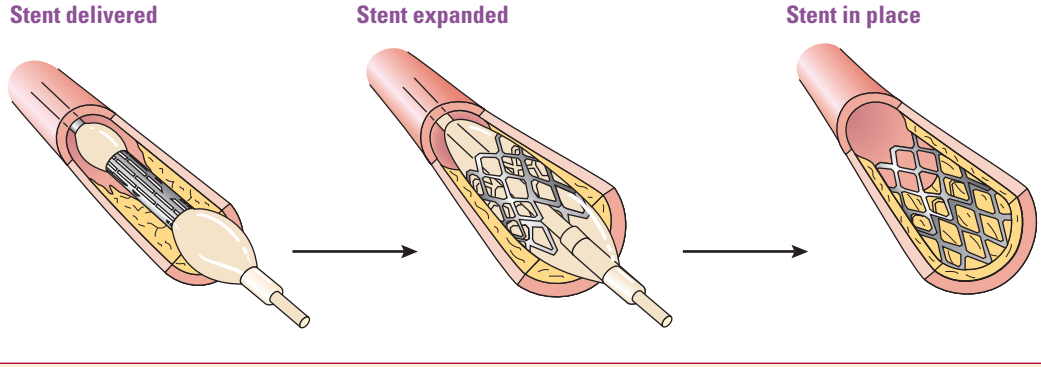
After coronary angiography confirms the presence and location of the occlusion, the doctor threads a guide catheter through the patient's femoral artery and into the coronary artery under fluoroscopic guidance.

Plaque, meet Balloon

When the guide catheter's position at the occlusion site is confirmed by angiography, the doctor carefully introduces a double-lumen balloon into the catheter and through the lesion, where a marked

Coronary artery stents

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



pressure gradient is obvious. The doctor alternately inflates and deflates the balloon until arteriography verifies successful arterial dilation and decrease in the pressure gradient. With balloon inflation, the plaque is compressed against the vessel wall, allowing coronary blood to flow more freely.

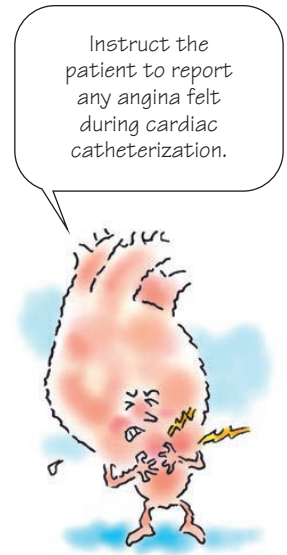
Placement of a coronary stent may also be done at the same time as an angioplasty. (See *Coronary artery stents*.)

Nursing considerations

Provide these care measures before and after cardiac catheterization.

Before the procedure

- Describe the procedure to the patient and his family and tell them it takes 1 to 4 hours to complete.
- Explain that a catheter will be inserted into an artery or a vein in the patient's groin and that he may feel pressure as the catheter moves along the vessel.
- Reassure the patient that although he'll be awake during the procedure, he'll be given a sedative. Instruct him to report any angina during the procedure.
- Explain that the doctor injects a contrast medium to outline the lesion's location. Warn the patient that he may feel a hot, flushing sensation or transient nausea during the injection.
- Check the patient's history for allergies; if he has had allergic reactions to shellfish, iodine, or contrast media, notify the doctor.



- Make sure the patient signs an informed consent form.
- Restrict food and fluids for at least 6 hours before the procedure.
- Make sure that the results of coagulation studies, CBC, serum electrolyte studies, blood typing and crossmatching, BUN, and serum creatinine are available.
- Obtain baseline vital signs and assess peripheral pulses.
- Apply ECG electrodes and insert an I.V. line if not already in place.
- Administer oxygen through a nasal cannula.
- Perform skin preparation according to your facility's policy.
- Give the patient a sedative as ordered.

After the procedure

- Assess the patient's vital signs and oxygen saturation every 15 minutes for the first hour and then every 30 minutes for 4 hours, unless his condition warrants more frequent checking. Monitor I.V. infusions, such as heparin or nitroglycerin, as indicated.
- Assess peripheral pulses distal to the catheter insertion site as well as the color, sensation, temperature, and capillary refill time of the affected extremity.
- Monitor ECG rhythm continuously and assess hemodynamic parameters closely for changes.
- Instruct the patient to remain in bed for 8 hours and to keep the affected extremity straight. Maintain sandbags in position if used to apply pressure to the catheter site. Elevate the head of the bed 15 to 30 degrees. If a hemostatic device was used to close the catheter insertion site, anticipate that the patient may be allowed out of bed in only a few hours.
- Assess the catheter site for hematoma, ecchymosis, and hemorrhage. If bleeding occurs, locate the artery and apply manual pressure; then notify the practitioner.
- Administer I.V. fluids as ordered (usually 100 ml/hour) to promote excretion of the contrast medium. Be sure to assess for signs of fluid overload.
- After the catheter is removed, apply direct pressure for at least 10 minutes and monitor the site often.
- Document the patient's tolerance of the procedure and status after it, including vital signs, hemodynamic parameters, appearance of catheter site, ECG findings, condition of the extremity distal to the insertion site, complications, and necessary interventions.

Other therapy

Other treatments for cardiovascular disorders include synchronized cardioversion, defibrillation, and pacemaker insertion.

Synchronized cardioversion

Cardioversion (synchronized countershock) is an elective or emergency procedure used to correct tachyarrhythmias (such as atrial tachycardia, atrial flutter, atrial fibrillation, and symptomatic ventricular tachycardia). It's also the treatment of choice for patients with arrhythmias that don't respond to drug therapy.

Electrifying experience

In synchronized cardioversion, an electric current is delivered to the heart to correct an arrhythmia. Compared with defibrillation, it uses much lower energy levels and is synchronized to deliver an electric charge to the myocardium at the peak R wave. (See *Choosing the correct cardioversion energy level*.)

The procedure causes immediate depolarization, interrupting reentry circuits (abnormal impulse conduction that occurs when cardiac tissue is activated two or more times, causing reentry arrhythmias) and allowing the SA node to resume control.

Synchronizing the electrical charge with the R wave ensures that the current won't be delivered on the vulnerable T wave and disrupt repolarization. Thus, it reduces the risk that the current will strike during the relative refractory period of a cardiac cycle and induce ventricular fibrillation.

Nursing considerations

- Describe the procedure to the patient and make sure an informed consent is obtained.
- Withhold all food and fluids for 6 to 12 hours before the procedure. If cardioversion is urgent, withhold food beginning as soon as possible.
- Obtain a baseline 12-lead ECG.
- Connect the patient to a pulse oximeter and blood pressure cuff.
- Ensure I.V. access.
- If the patient is awake, administer a sedative as ordered.
- Place the leads on the patient's chest and assess his cardiac rhythm.
- Attach defibrillation pads to the chest wall; position the pads so that one pad is to the right of the sternum, just below the clavicle, and the other is at the fifth or sixth intercostal space in the left anterior axillary line.

Choosing the correct cardioversion energy level

When choosing an energy level for cardioversion, try the lowest energy level first. If the arrhythmia isn't corrected, repeat the procedure using the next energy level. Repeat this procedure until the arrhythmia is corrected or until the highest energy level is reached. The initial energy dose used for cardioversion is:

- 100 joules (biphasic and monophasic) for unstable regular ventricular tachycardia with a pulse
- 120 to 200 (biphasic) or 200 (monophasic) joules for atrial fibrillation
- 50 to 100 joules (biphasic and monophasic) for atrial flutter and other supraventricular tachycardia.

Ready to jolt

- Turn on the defibrillator and select the ordered energy level, usually between 50 and 100 joules.
- Activate the synchronized mode by depressing the synchronizer switch.
- Check that the machine is sensing the R wave correctly.
- Charge the machine.
- Instruct other personnel to stand clear of the patient and the bed to avoid the risk of an electric shock.

Letting the sparks fly

- Discharge the current by pushing the DISCHARGE or SHOCK button.
- If cardioversion is unsuccessful, repeat the procedure two or three times, as ordered, gradually increasing the energy with each additional countershock.
- If normal rhythm is restored, continue to monitor the patient and provide supplemental ventilation as long as needed.
- If the patient's cardiac rhythm changes to ventricular fibrillation, switch the mode from synchronized to defibrillate and defibrillate the patient immediately after charging the machine.
- Remember to reset the SYNC MODE on the defibrillator after each synchronized cardioversion. Resetting this switch is necessary because most defibrillators automatically reset to an unsynchronized mode.
- Document the use of synchronized cardioversion, the rhythm before and after cardioversion, medication given, amperage used, and how the patient tolerated the procedure.

Defibrillation

In defibrillation, an electric current is directed through the patient's heart. The current causes the myocardium to depolarize, which in turn encourages the SA node to resume control of the heart's electrical activity. (See *Biphasic defibrillators*, page 244.)

The electrodes delivering the current may be placed on the patient's chest or, during cardiac surgery, directly on the myocardium.

Biphasic defibrillators

Monophasic defibrillators deliver a single current of electricity that travels in one direction between the two pads on the patient's chest. A large amount of electrical current is required for effective monophasic defibrillation. Biphasic defibrillators are now more popular in hospitals. Pad placement is the same as with the monophasic defibrillator. The difference is that during biphasic defibrillation, the electrical current discharged from the pads travels in a positive direction for a specified duration and then reverses and flows in a negative direction for the remaining time of the electrical discharge.

Energy efficient

The biphasic defibrillator delivers two currents of electricity and lowers the defibrillation threshold of the heart muscle, making it possible to successfully defibrillate ventricular fibrillation with smaller amounts of energy.

Adjustable

The biphasic defibrillator is able to adjust for differences in impedance or the resistance of the current through the chest. This reduces the number of shocks needed to terminate ventricular fibrillation.

Less myocardial damage

Because the biphasic defibrillator requires lower energy levels and fewer shocks, damage to the myocardial muscle is reduced. Biphasic defibrillators used at the clinically appropriate energy level may be used for defibrillation and, in the synchronized mode, for synchronized cardioversion.

Act early and quickly

Because some arrhythmias, such as ventricular fibrillation, can cause death if not corrected, the success of defibrillation depends on early recognition and quick treatment.

In addition to treating ventricular fibrillation, defibrillation may also be used to treat ventricular tachycardia that doesn't produce a pulse or polymorphic ventricular tachycardia with a pulse.

Nursing considerations

- Assess the patient to determine if he lacks a pulse. Call for help and perform cardiopulmonary resuscitation (CPR) until the defibrillator and other emergency equipment arrive. (See *Automated external defibrillator.*)
- Connect the monitoring leads of the defibrillator to the patient and assess his cardiac rhythm in two leads.
- Expose the patient's chest and apply the self-adhesive, pre-gelled conductive pads at the proper positions. (See *Defibrillator pad placement*, page 246.)
- Turn on the defibrillator and, if performing external defibrillation, set the energy level at 200 joules (biphasic) or 360 joules (monophasic) for an adult patient.

Automated external defibrillator

An automated external defibrillator (AED) has a cardiac rhythm analysis system. The AED interprets the patient's cardiac rhythm and gives the operator step-by-step directions on how to proceed if defibrillation is indicated. Most AEDs have a "quick-look" feature that allows visualization of the rhythm with the paddles before electrodes are connected.

Computer-assisted system

The AED is equipped with a microcomputer that senses and analyzes a patient's heart rhythm at the push of a button. It then audibly or visually prompts you to deliver a shock.

All models have the same basic functions but offer different operating options. For example, all AEDs communicate directions by displaying messages on a screen, giving voice commands, or both. Some AEDs simultaneously display a patient's heart rhythm.

All devices record your interactions with the patient during defibrillation, either on a cassette tape or in a solid state memory module. Some AEDs have an integral printer for immediate event documentation.

Charging and shocking once . . .

- Charge the unit by pressing the charge button located on the machine.
- Reassess the patient's cardiac rhythm in two leads.
- If the patient remains in a shockable rhythm, instruct all personnel to stand clear of the patient and the bed. Also make a visual check to make sure everyone is clear of the patient and the bed.
- Discharge the current by pressing the appropriate button on the defibrillator.
- Continue with 2 minutes of CPR. Reassess for a pulse and cardiac rhythm. Give supplemental oxygen and begin administering appropriate medications such as epinephrine.

. . . then again

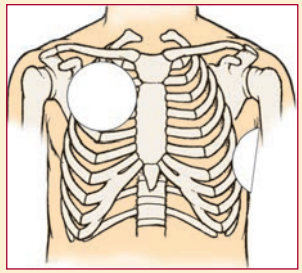
- If necessary, after the initial shock and two rounds of CPR, prepare to defibrillate a second time at the same joules. Announce that you're preparing to defibrillate and follow the procedure described previously.
- Continue CPR.
- If the patient still has no pulse after the first two cycles of defibrillation and CPR, consider possible causes for failure of the patient's rhythm to convert, such as acidosis and hypoxia.
- If defibrillation restores a normal rhythm, assess the patient. Obtain baseline ABG levels and a 12-lead ECG. Provide

Defibrillator pad placement

Here's a guide to correct pad placement for defibrillation.

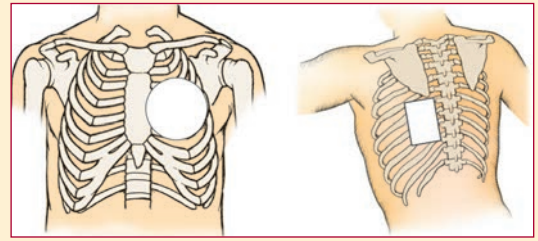
Anterolateral placement

For anterolateral placement, position one pad to the right of the upper sternum, just below the right clavicle, and the other over the fifth or sixth intercostal space at the left anterior axillary line.



Anteroposterior placement

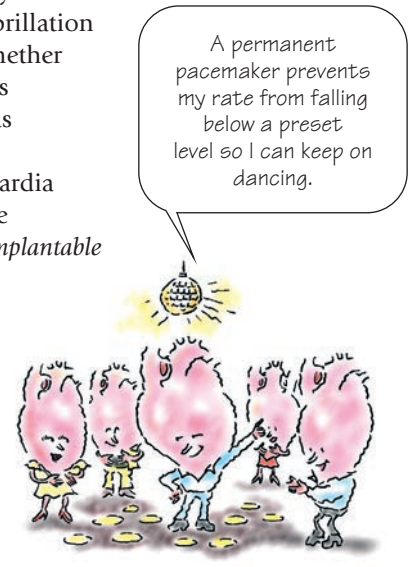
For anteroposterior placement, position the anterior pad directly over the heart at the precordium, to the left of the lower sternal border. Place the posterior pad under the patient's body beneath the heart and immediately below the scapula (but not on the vertebral column).



supplemental oxygen, ventilation, and medications as needed. Prepare the defibrillator for immediate reuse.

Document everything

- Document the procedure, including the patient's ECG rhythms before and after defibrillation; the number of times defibrillation was performed; the voltage used during each attempt; whether a pulse returned; the dosage, route, and time of any drugs administered; whether CPR was used; how the airway was maintained; and the patient's outcome.
- If the patient has recurrent episodes of ventricular tachycardia or ventricular fibrillation, the insertion of an implantable cardioverter-defibrillator (ICD) may be necessary. (See *Implantable cardioverter-defibrillator*.)



Permanent pacemaker insertion

A permanent pacemaker is a self-contained device surgically implanted in a pocket under the patient's skin. This is usually done in an operating room or cardiac catheterization laboratory.

Permanent pacemakers function in the demand mode, allowing the patient's heart to beat on its own but preventing it from falling below a preset rate.

Implantable cardioverter-defibrillator

An ICD is implanted to continually monitor a patient's heart for bradycardia, ventricular tachycardia, and ventricular fibrillation. The device also administers either shocks or paced beats. Some ICDs can provide biventricular pacing or administer therapy for atrial fibrillation.

ICDs are generally indicated when drug therapy, surgery, or catheter ablation fails to prevent the patient's dangerous arrhythmia.

What it is

An ICD system consists of a programmable pulse generator and one or more lead wires. The pulse generator is a small battery-powered computer that monitors the heart's electrical signals and delivers electrical therapy when an abnormal rhythm is identified.

It also stores information on the heart's activity before, during, and after an arrhythmia, along with tracking which treatment was delivered and the outcome of that treatment. Many devices also store electrograms (electrical tracings similar to ECGs). With an interrogation device, a practitioner can retrieve this information to evaluate ICD function and battery status and to adjust ICD system settings.

How it's programmed

When caring for a patient with an ICD, it's important to know how the device is programmed. This information is available through a status report that can be obtained and printed when the practitioner or specially trained technician interrogates the device. This involves placing a specialized piece of equipment over the implanted pulse generator to retrieve pacing function.

If the patient experiences an arrhythmia or if the device delivers a therapy, the program information is used to evaluate the functioning of the device. Program information includes:

- type and model of ICD
- status of the device (on or off)
- detection rates
- therapies that will be delivered (pacing, antitachycardia pacing, cardioversion, and defibrillation).

What you should know

- If the patient experiences cardiac arrest, initiate CPR and advanced cardiac life support.
- If the ICD delivers a shock while you're performing chest compressions, you may feel a slight shock. Wear gloves to eliminate this.
- It's safe to also externally defibrillate a patient with an ICD as long as the paddles aren't placed directly over the pulse generator. The anteroposterior paddle position is preferred.

And the nominees for insertion are . . .

Permanent pacemakers are indicated for patients with:

- persistent bradycardia
- complete heart block
- congenital or degenerative heart disease
- Stokes-Adams syndrome
- Wolff-Parkinson-White syndrome
- sick sinus syndrome.

Setting the pace

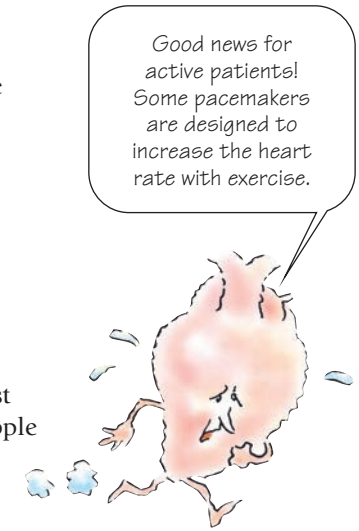
Pacing electrodes can be placed in the atria, ventricles, or both chambers (atrioventricular sequential or dual chamber). Biventricular pacemakers are also available for cardiac resynchronization therapy in some patients with heart failure. (See *Understanding pacemaker codes*, page 248.)

Understanding pacemaker codes

The capabilities of pacemakers are described by a five-letter coding system, although typically only the first three letters are used.

First letter	Second letter	Third letter	Fourth letter	Fifth letter
The first letter identifies which heart chambers are paced. Here are the letters used to signify these options: • V = ventricle • A = atrium • D = dual (ventricle and atrium) • O = none.	The second letter signifies the heart chamber where the pacemaker senses the intrinsic activity: • V = ventricle • A = atrium • D = dual • O = none.	The third letter shows the pacemaker's response to the intrinsic electrical activity it senses in the atrium or ventricle: • T = triggers pacing • I = inhibits pacing • D = dual; can be triggered or inhibited depending on the mode and where intrinsic activity occurs • O = none; the pacemaker doesn't change its mode in response to sensed activity.	The fourth letter describes rate modulation, also known as *rate responsiveness* or *rate-adaptive pacing*: • R = rate modulation (a sensor adjusts the programmed paced heart rate in response to patient activity) • O = none (rate modulation is unavailable or disabled).	The fifth letter is rarely used but specifies the location or absence of multisite pacing: • O = none (no multisite pacing is present) • A = atrium or atria (multisite pacing in the atrium or atria is present) • V = ventricle or ventricles (multisite pacing in the ventricle or ventricles is present) • D = dual site (dual site pacing in both the atria and ventricles is present).

The most common pacing codes are VVI for single-chamber pacing and DDD for dual-chamber pacing. To keep the patient healthy and active, some pacemakers are designed to increase the heart rate with exercise.



Nursing considerations

Provide care measures before and after pacemaker placement. Nursing responsibilities during surgical placement involve monitoring ECG and maintaining sterile technique.

Before surgery

- Explain the procedure to the patient.
- Before pacemaker insertion, clip the hair on the patient's chest from the axilla to the midline and from the clavicle to the nipple line on the side selected by the doctor.
- Establish an I.V. line.

- Obtain baseline vital signs and a baseline ECG.
- Provide sedation as ordered.

After surgery

- Monitor the patient's ECG to check for arrhythmias and to ensure correct pacemaker functioning.
- Check the dressing for signs of bleeding and infection.
- Change the dressing according to facility policy.
- Check vital signs and LOC every 15 minutes for the first hour, every hour for the next 4 hours, then every 4 hours.
- Provide the patient with an identification card that lists the pacemaker type and manufacturer, serial number, pacemaker rate setting, date implanted, and the doctor's name.

Temporary pacemaker insertion

A temporary pacemaker is usually inserted in an emergency. The device consists of an external, battery-powered pulse generator and a lead or electrode system.

Temporary pacemakers typically come in three types, including:

1. transcutaneous
2. transvenous
3. epicardial.

Dire straits

In a life-threatening situation, a transcutaneous pacemaker is the best choice. This device works by sending an electrical impulse from the pulse generator to the patient's heart by way of two electrodes, which are placed on the front and back of the patient's chest.

Transcutaneous pacing is quick and effective, but it's used only until the doctor can institute transvenous pacing.

More comfortable and more reliable

Besides being more comfortable for the patient, a transvenous pacemaker is more reliable than a transcutaneous pacemaker.

Transvenous pacing involves threading an electrode catheter through a vein into the patient's right atrium or right ventricle. The electrode is attached to an external pulse generator that can provide an electrical stimulus directly to the endocardium. (See *Temporary transvenous pacemaker*, page 250.)

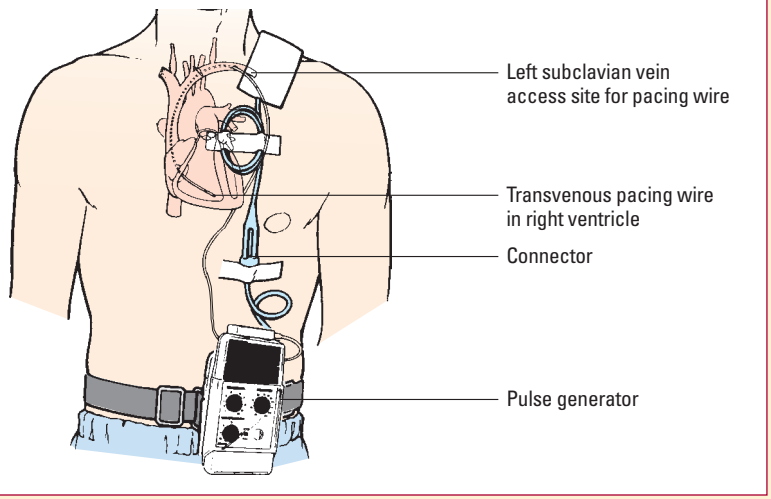
Transvenous pacemaker basics

Indications for a temporary transvenous pacemaker include:

- management of bradycardia
- presence of tachyarrhythmias
- other conduction system disturbances.

Temporary transvenous pacemaker

Transvenous pacing provides a more reliable pacing beat. This type of pacing is more comfortable for the patient because the pacing wire is inserted in the heart via a major vein.



The purpose of temporary transvenous pacemaker insertion is:

- to maintain circulatory integrity by providing for standby pacing in case of sudden complete heart block
- to increase heart rate during periods of symptomatic bradycardia
- occasionally, to control sustained supraventricular or ventricular tachycardia.

Epicardial option

During cardiac surgery, the surgeon may insert electrodes through the epicardium of the right ventricle and, if he wants to institute AV sequential pacing, the right atrium. From there, the electrodes pass through the chest wall, where they remain available if temporary pacing becomes necessary. This is called *epicardial pacing*. It uses the same equipment as temporary or transvenous pacers; it is done only after surgery.

Pacemaker no-no's

Among the contraindications to pacemaker therapy are electromechanical dissociation and ventricular fibrillation.

Nursing considerations

- Teach measures to prevent microshock; warn the patient not to use any electrical equipment that isn't grounded.
- Use other safety measures such as placing a plastic cover (supplied by the manufacturer) over the pacemaker controls to avoid an accidental setting change. If the patient needs emergency defibrillation, make sure the pacemaker can withstand the procedure. If you're unsure, disconnect the pulse generator to avoid damage.
- When using a transcutaneous pacemaker, don't place the electrodes over a bony area because bone conducts current poorly. With a female patient, place the anterior electrode under the patient's breast but not over her diaphragm.
- If the doctor inserts the transvenous pacer wire through the brachial or femoral vein, immobilize the patient's arm or leg to avoid putting stress on the pacing wires.
- After insertion of any temporary pacemaker, assess the patient's vital signs, skin color, LOC, and peripheral pulses to determine the effectiveness of the paced rhythm. Perform a 12-lead ECG to serve as a baseline and then perform additional ECGs daily or with clinical changes. Also, if possible, obtain a rhythm strip before, during, and after pacemaker placement; anytime the pacemaker settings are changed; and whenever the patient receives treatment because of a complication due to the pacemaker.
- Continuously monitor the ECG reading, noting capture, sensing, rate, intrinsic beats, and competition of paced and intrinsic rhythms. If the pacemaker is sensing correctly, the sense indicator on the pulse generator should flash with each beat.
- Record the date and time of pacemaker insertion, the type of pacemaker, the reason for insertion, and the patient's response. Note the pacemaker settings. Document any complications and the interventions taken.
- If the patient has epicardial pacing wires in place, clean the insertion site and change the dressing daily. At the same time, monitor the site for signs of infection. Always keep the pulse generator nearby in case pacing becomes necessary.

Cardiovascular system disorders

Common cardiovascular disorders include acute coronary syndromes, aneurysms, cardiac arrhythmias, cardiac tamponade, cardiogenic shock, cardiomyopathy, heart failure, hypertensive crisis, pericarditis, and valvular heart disease.

Acute coronary syndromes

Patients with acute coronary syndromes have some degree of coronary artery occlusion. The degree of occlusion defines whether the acute coronary syndrome is:

- unstable angina
- non-ST-segment elevation myocardial infarction (NSTEMI)
- ST-segment elevation myocardial infarction (STEMI).

Plaque's place

The development of any acute coronary syndrome begins with a rupture or erosion of plaque—an unstable and lipid-rich substance. The rupture results in platelet adhesions, fibrin clot formation, and activation of thrombin.

What causes it

Patients with certain risk factors appear to face a greater likelihood of developing an acute coronary syndrome. These factors include:

- family history of heart disease
- obesity
- smoking
- high-fat, high-carbohydrate diet
- sedentary lifestyle
- menopause
- stress
- diabetes
- hypertension
- hyperlipoproteinemia.

How it happens

An acute coronary syndrome most commonly results when a thrombus progresses and occludes blood flow. (An early thrombus doesn't necessarily block blood flow.) The effect is an imbalance in myocardial oxygen supply and demand.

Degree and duration

The degree and duration of blockage dictate the type of infarct that occurs:

- If the patient has unstable angina, a thrombus partially occludes a coronary vessel. This thrombus is full of platelets. The partially occluded vessel may have distal microthrombi that cause necrosis in some myocytes.
- If smaller vessels infarct, the patient is at higher risk for MI, which may progress to NSTEMI. Usually, only the innermost layer of the heart is damaged.
- STEMI results when reduced blood flow through one of the coronary arteries causes myocardial ischemia, injury, and necrosis. The damage extends through all myocardial layers.

What to look for

A patient with angina typically experiences:

- burning
- squeezing
- crushing tightness in the substernal or precordial chest that may radiate to the left arm, neck, jaw, or shoulder blade.

A woman thing?

Any patient may experience atypical chest pain, but it's more common in women. (See *Atypical chest pain in women*.)

It hurts when I do this

Angina most frequently follows physical exertion but may also follow emotional excitement, exposure to cold, or a large meal. Angina is commonly relieved by nitroglycerin and rest. It's less severe and shorter lived than the pain of acute MI.

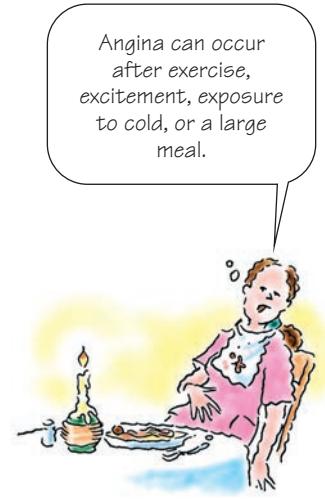
Four forms

Angina has four major forms:

1. stable—predictable pain, in frequency and duration, which can be relieved with nitrates and rest
2. unstable—increased pain, which is easily induced
3. Prinzmetal's or a variant—pain from unpredictable coronary artery spasm
4. microvascular—angina-like chest pain due to impairment of vasodilator reserve in a patient with normal coronary arteries.

Atypical chest pain in women

Women with CAD may experience typical chest pain but commonly experience atypical chest pain, vague chest pain, or a lack of chest pain. They're more likely than men to experience a toothache or pain in the arm, shoulder, jaw, neck, throat, back, breast, or stomach.



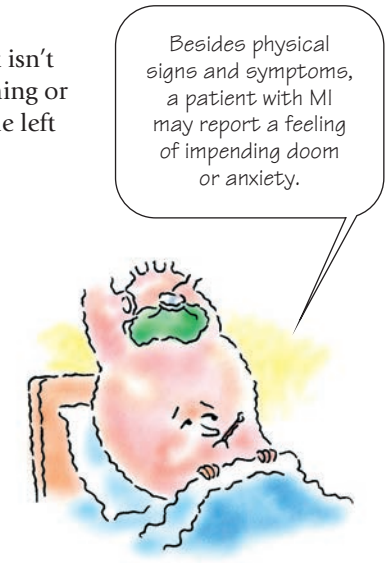
My, my, MI pain

A patient with MI experiences severe, persistent chest pain that isn't relieved by rest or nitroglycerin. He may describe pain as crushing or squeezing. The pain is usually substernal but may radiate to the left arm, jaw, neck, or shoulder blades.

And many more

Other signs and symptoms of MI include:

- a feeling of impending doom
- fatigue
- nausea and vomiting
- shortness of breath
- cool extremities
- perspiration
- anxiety
- hypotension or hypertension
- palpable precordial pulse
- muffled heart sounds.



What tests tell you

These tests are used to diagnose CAD:

- ECG during an anginal episode shows ischemia. Serial 12-lead ECGs may be normal or inconclusive during the first few hours after an MI. Abnormalities include serial ST-segment depression in NSTEMI and ST-segment elevation and Q waves, representing scarring and necrosis, in STEMI. (See *Locating myocardial damage*.)
- Coronary angiography reveals coronary artery stenosis or obstruction and collateral circulation and shows the condition of the arteries beyond the narrowing.
- Myocardial perfusion imaging with thallium-201 during treadmill exercise discloses ischemic areas of the myocardium, visualized as "cold spots."
- With MI, serial serum cardiac marker measurements show elevated CK, especially the CK-MB isoenzyme (the cardiac muscle fraction of CK), troponin T and I, myoglobin, and IMA.
- C-reactive protein (CRP) levels help measure cardiac risk. Patients with chest pain and a higher CRP level have an increased risk of CAD. The PLAC test is a new test that also helps identify patients at a higher risk for CAD.
- With STEMI, echocardiography shows ventricular wall dyskinesia.

How it's treated

For patients with angina, the goal of treatment is to reduce myocardial oxygen demand or increase oxygen supply.

Locating myocardial damage

After you've noted characteristic lead changes of an acute MI, use this chart to identify the areas of damage. Match the lead changes in the second column with the affected wall in the first column and the artery involved in the third column. Column four shows reciprocal lead changes.

Wall affected	Leads	Artery involved	Reciprocal changes
Anterior	V_2 to V_4	Left coronary artery, left anterior descending (LAD) artery	II, III, aV_F
Anterolateral	I, aV_L, V_3 to V_6	LAD artery, circumflex artery	II, III, aV_F
Anteroseptal	V_1 to V_4	LAD artery	None
Inferior (diaphragmatic)	II, III, aV_F	Right coronary artery	I, aV_L
Lateral	I, aV_L, V_5, V_6	Circumflex artery, branch of left coronary artery	II, III, aV_F
Posterior	V_8, V_9	Right coronary artery, circumflex artery	V_1 to V_4
Right ventricular	V_{4R}, V_{5R}, V_{6R}	Right coronary artery	None

These treatments are used to manage angina:

- Nitrates reduce myocardial oxygen consumption.
- Beta-adrenergic blockers may be administered to reduce the workload and oxygen demands of the heart.
- If angina is caused by coronary artery spasm, calcium channel blockers may be given.
- Antiplatelet drugs minimize platelet aggregation and the danger of coronary occlusion.
- Antilipemic drugs can reduce elevated serum cholesterol or triglyceride levels.
- Obstructive lesions may necessitate CABG or PTCA. Other alternatives include laser angioplasty, minimally invasive surgery, atherectomy, or stent placement.

MI relief

The goals of treatment for MI are to relieve pain, stabilize heart rhythm, revascularize the coronary artery, preserve myocardial tissue, and reduce cardiac workload.

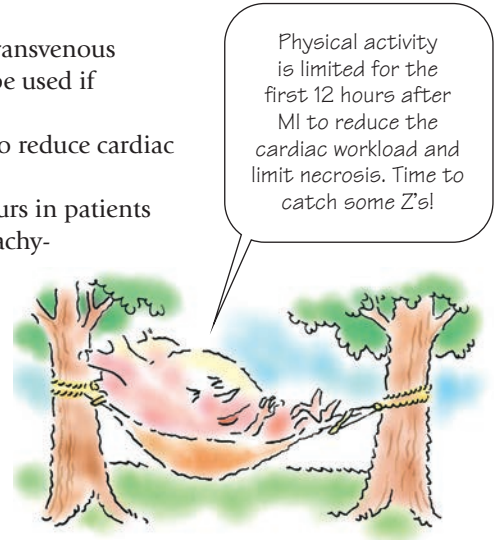
Here are some guidelines for treatment:

- Thrombolytic therapy should be started within 6 hours of the onset of symptoms (unless contraindications exist). Thrombolytic therapy involves administration of streptokinase (Streptase), alteplase (Activase), or reteplase (Retavase).

- PTCA or stent placements are options for opening blocked or narrowed arteries. Primary percutaneous coronary intervention is the preferred method in management of acute MI. In a patient presenting with STEMI, the patient's coronary artery should be opened with percutaneous coronary intervention within 90 minutes or with a target time of less than 60 minutes.
- Oxygen is administered to increase oxygenation of the blood.
- Nitroglycerin is administered sublingually to relieve chest pain, unless systolic blood pressure is less than 90 mm Hg or heart rate is less than 50 or greater than 100 beats/minute.

Heartache

- Morphine is administered as analgesia because pain stimulates the sympathetic nervous system, leading to an increase in heart rate and vasoconstriction.
- Aspirin and antiplatelet drugs are administered to inhibit platelet aggregation.
- I.V. heparin is given to patients who have received tissue plasminogen activator to increase the chances of patency in the affected coronary artery.
- Lidocaine, transcutaneous pacing patches (or a transvenous pacemaker), defibrillation, or epinephrine may be used if arrhythmias are present.
- Physical activity is limited for the first 12 hours to reduce cardiac workload, thereby limiting the area of necrosis.
- I.V. nitroglycerin is administered for 24 to 48 hours in patients without hypotension, bradycardia, or excessive tachycardia, to reduce afterload and preload and relieve chest pain.
- Glycoprotein IIb/IIIa inhibitors (such as abciximab [ReoPro]) are administered to patients with continued unstable angina or acute chest pain, or following invasive cardiac procedures, to reduce platelet aggregation.
- I.V. beta-adrenergic blocker is administered early to patients with evolving acute MI; it's followed by oral therapy to reduce heart rate and contractibility and reduce myocardial oxygen requirements.
- ACE inhibitors are administered to those with evolving MI with ST-segment elevation or left bundle-branch block to reduce afterload and preload and prevent remodeling.
- Laser angioplasty, atherectomy, or stent placement may be initiated.
- Lipid-lowering drugs are administered to patients with elevated LDL and cholesterol levels.

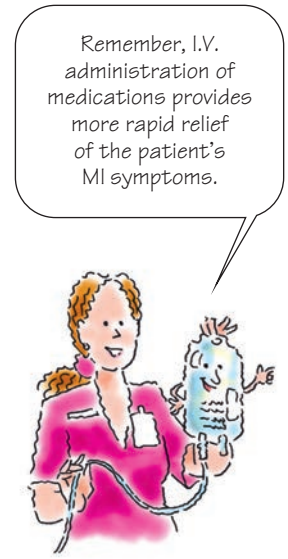


What to do

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

CCU, ECG, and more!

- On admission to the coronary care unit, monitor and record the patient's ECG, blood pressure, temperature, and heart and breath sounds. Also, assess and record the severity, location, type, and duration of pain.
- Obtain a 12-lead ECG and assess heart rate and blood pressure when the patient experiences acute chest pain.
- Monitor the patient's hemodynamic status closely. Be alert for indicators suggesting decreased cardiac output, such as decreased blood pressure, increased heart rate, increased PAP, increased PAWP, decreased cardiac output measurements, and decreased right atrial pressure.
- Assess urine output hourly.
- Monitor the patient's oxygen saturation levels and notify the practitioner if oxygen saturation falls below 90%.
- Check the patient's blood pressure after giving nitroglycerin, especially the first dose.
- During episodes of chest pain, monitor ECG, blood pressure, and PA catheter readings (if applicable) to determine changes.
- Frequently monitor ECG rhythm strips to detect heart rate changes and arrhythmias.
- Obtain serial measurements of cardiac enzyme levels as ordered.
- Watch for crackles, cough, tachypnea, and edema, which may indicate impending left-sided heart failure. Carefully monitor daily weight, intake and output, respiratory rate, serum enzyme levels, ECG waveforms, and blood pressure. Auscultate for S_3 or S_4 gallops.
- Prepare the patient for reperfusion therapy as indicated.
- Administer and titrate medications as ordered. Avoid giving I.M. injections; I.V. administration provides more rapid symptom relief.



I need a break

- Organize patient care and activities to allow rest periods. If the patient is immobilized, turn him often and use intermittent compression devices. Gradually increase the patient's activity level as tolerated.
- Provide a clear liquid diet until nausea subsides. Anticipate a possible order for a low-cholesterol, low-sodium diet without caffeine.
- Provide a stool softener to prevent straining during defecation.

Aortic aneurysm

An aortic aneurysm is a localized outpouching or an abnormal dilation in a weakened arterial wall. Aortic aneurysm typically occurs in the aorta between the renal arteries and the iliac branches, but the abdominal, thoracic, or ascending arch of the aorta may be affected.

What causes it

The exact cause of an aortic aneurysm is unclear, but several factors place a person at risk, including:

- advanced age
- history of hypertension
- smoking
- atherosclerosis
- connective tissue disorders
- diabetes
- trauma.

How it happens

Aneurysms arise from a defect in the middle layer of the arterial wall (tunica media, or medial layer). Once the elastic fibers and collagen in the middle layer are damaged, stretching and segmental dilation occur. As a result, the medial layer loses some of its elasticity, and it fragments. Smooth muscle cells are lost, and the wall thins.

Thin and thinner

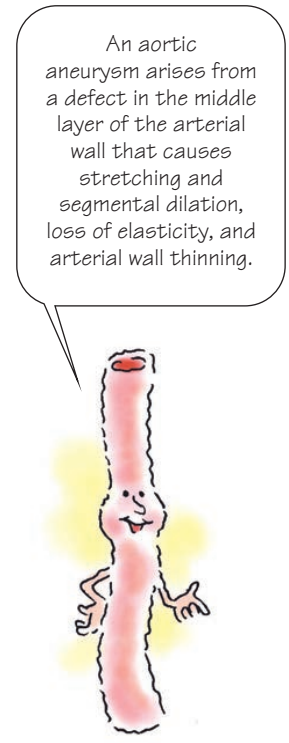
The thinned wall may contain calcium deposits and atherosclerotic plaque, making the wall brittle. As a person ages, the elastin in the wall decreases, further weakening the vessel. If hypertension is present, blood flow slows, resulting in ischemia and additional weakening.

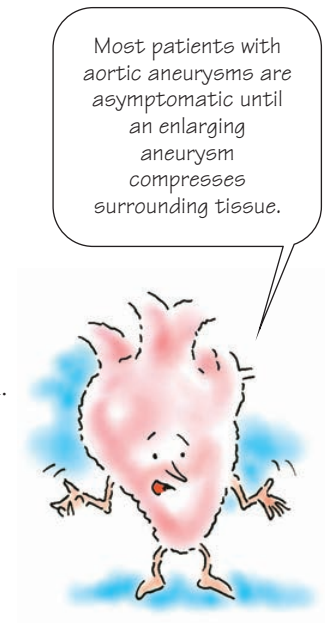
Wide vessel, slow flow

After an aneurysm begins to develop, lateral pressure increases, causing the vessel lumen to widen and blood flow to slow. Over time, mechanical stressors contribute to elongation of the aneurysm.

Blood forces

Hemodynamic forces may also play a role, causing pulsatile stresses on the weakened wall and pressing on the small vessels that supply nutrients to the arterial wall. In aortic aneurysms, this causes the aorta to become bowed and tortuous.





What to look for

Most patients with aortic aneurysms are asymptomatic until the aneurysms enlarge and compress surrounding tissue.

A large aneurysm may produce signs and symptoms that mimic those of MI, renal calculi, lumbar disc disease, and duodenal compression.

Some of the classic symptoms of a thoracic aortic aneurysm include substernal pain, hoarseness or coughing, difficulty swallowing, difficulty breathing, aortic murmur, and unequal blood pressure and pulses when measured in both arms.

When symptoms arise

Usually, if the patient exhibits symptoms, it's because of rupture, expansion, embolization, thrombosis, or pressure from the mass on surrounding structures. Rupture is more common if the patient also has hypertension or if the aneurysm is larger than 6 cm.

Thoracic aortic aneurysm

If the patient has a suspected thoracic aortic aneurysm, assess for:

- complaints of substernal pain possibly radiating to the neck, back, abdomen, or shoulders
- hoarseness or coughing
- difficulty swallowing
- difficulty breathing
- unequal blood pressure and pulse when measured in both arms
- aortic insufficiency murmur.

Acute expansion

When there's an acute expansion of a thoracic aortic aneurysm, assess for:

- severe hypertension
- neurologic changes
- a new murmur of aortic sufficiency
- right sternoclavicular lift
- jugular vein distention
- tracheal deviation.

Abdominal aortic aneurysm

The patient with an abdominal aortic aneurysm may experience:

- dull abdominal pain
- lower back pain that's unaffected by movement
- gastric or abdominal fullness
- pulsating mass in the periumbilical area (if the patient isn't obese)
- systolic bruit over the aorta on auscultation of the abdomen
- hypotension (with aneurysm rupture).

What tests tell you

No specific laboratory test to diagnose an aortic aneurysm exists. However, these tests may be helpful:

- If blood is leaking from the aneurysm, leukocytosis and a decrease in hemoglobin and hematocrit may be noted.

Telltale TEE

- TEE allows visualization of the thoracic aorta. It's commonly combined with Doppler flow studies to provide information about blood flow.
- Abdominal ultrasonography or echocardiography can be used to determine the size, shape, and location of the aneurysm.
- Anteroposterior and lateral X-rays of the chest or abdomen can be used to detect aortic calcification and widened areas of the aorta.
- Computed tomography (CT) scan and MRI can disclose the aneurysm's size and effect on nearby organs.
- Serial ultrasonography at 6-month intervals reveals any growth of small aneurysms.
- ECG will be absent of any signs of MI.
- Aortography is used in determining the aneurysm's approximate size and patency of the visceral vessels.

How it's treated

Aneurysm treatment usually involves surgery and appropriate drug therapy. Aortic aneurysms usually require resection and replacement of the aortic section using a vascular or Dacron graft. However, keep these points in mind:

- If the aneurysm is small and produces no symptoms, surgery may be delayed, with regular physical examination and ultrasonography performed to monitor its progression.
- Large or symptomatic aneurysms are at risk for rupture and need immediate repair.
- Endovascular grafting may be an option for a patient with an abdominal aortic aneurysm. This procedure, which can be done using local or regional anesthesia, is a minimally invasive procedure whereby the walls of the aorta are reinforced to prevent expansion and rupture of the aneurysm.
- Medications to control blood pressure, relieve anxiety, and control pain are also prescribed.

Rush to respond to rupture

Rupture of an aortic aneurysm is a medical emergency requiring prompt treatment, including:

- resuscitation with fluid and blood replacement
- I.V. propranolol to reduce myocardial contractility

- I.V. nitroprusside to reduce blood pressure and maintain it at 90 to 100 mm Hg systolic
- analgesics to relieve pain
- an arterial line and indwelling urinary catheter to monitor the patient's condition preoperatively.

What to do

- Assess the patient's vital signs, especially blood pressure, every 2 to 4 hours or more frequently, depending on the severity of his condition. Monitor blood pressure and pulse in extremities and compare findings bilaterally. If the difference in systolic blood pressure exceeds 10 mm Hg, notify the practitioner immediately.
- Assess cardiovascular status frequently, including heart rate, rhythm, ECG, and cardiac enzyme levels. MI can occur if an aneurysm ruptures along the coronary arteries.
- Obtain blood samples to evaluate kidney function by assessing BUN, creatinine, and electrolyte levels. Measure intake and output, hourly if necessary, depending on the patient's condition.
- Monitor CBC for evidence of blood loss, including decreased hemoglobin, hematocrit, and red blood cell (RBC) count.
- Send blood to the laboratory to be typed and crossmatched in case the patient needs a blood transfusion.
- If the patient's condition is acute, obtain an arterial sample for ABG analysis, as ordered, and monitor cardiac rhythm. Assist with arterial line insertion to allow for continuous blood pressure monitoring. Assist with insertion of a PA catheter to assess hemodynamic balance.
- Administer ordered medications to control aneurysm progression. Provide analgesics to relieve pain, if present.
- Observe the patient for signs of rupture, which may be immediately fatal. Watch closely for any signs of acute blood loss: decreasing blood pressure; increasing pulse and respiratory rates; cool, clammy skin; restlessness; and decreased LOC.

Rupture response

- If rupture occurs, insert a large-bore I.V. catheter, begin fluid resuscitation, and administer nitroprusside I.V. as ordered, usually to maintain a MAP of 70 to 80 mm Hg. Also administer propranolol I.V. (to reduce left ventricular ejection velocity) as ordered until the heart rate ranges from 60 to 80 beats per minute. Expect to administer additional doses every 4 to 6 hours until oral medications can be used.
- If the patient is experiencing acute pain, administer morphine I.V. as ordered.
- Prepare the patient for emergency surgery.

After surgery

- Administer nitroprusside or nitroglycerin and titrate to maintain a normotensive state.
- Provide analgesics to relieve pain.
- Administer anticoagulants, such as heparin, to help prevent formation of thrombi.
- Continue to monitor ECG for changes. Assess the patient's hemodynamic status at least every 4 hours; he may have a decreased CVP, PAP, and PAWP.
- Administer I.V. fluids as ordered.
- Monitor the patient for signs of bleeding, such as hypotension and decreased hemoglobin and hematocrit.
- Perform meticulous pulmonary hygiene measures, including suctioning, chest physiotherapy, and deep breathing.
- Assess urine output hourly.
- Maintain NG tube patency to ensure gastric decompression.
- Assist with serial Doppler examination of all extremities to evaluate the adequacy of vascular repair and presence of embolization.
- Assess for signs of poor arterial perfusion, such as pain, paresthesia, pallor, pulselessness, paralysis, and poikilothermy (coldness).

Cardiac arrhythmias

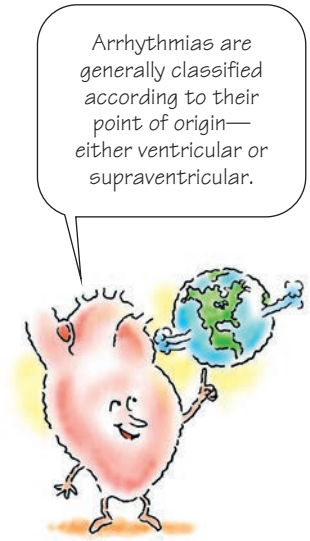
In cardiac arrhythmia, abnormal electrical conduction or automaticity changes heart rate and rhythm.

Asymptomatic to catastrophic

Cardiac 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 requires immediate resuscitation.

Organized by origin and effects

Cardiac arrhythmias are generally classified according to their origin (ventricular or supraventricular). Their effect on cardiac output and blood pressure, partially influenced by the site of origin, determines their clinical significance. Lethal arrhythmias, such as ventricular tachycardia and ventricular fibrillation, are a major cause of sudden cardiac death.



What causes it

Common causes of cardiac arrhythmias include:

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

How it happens

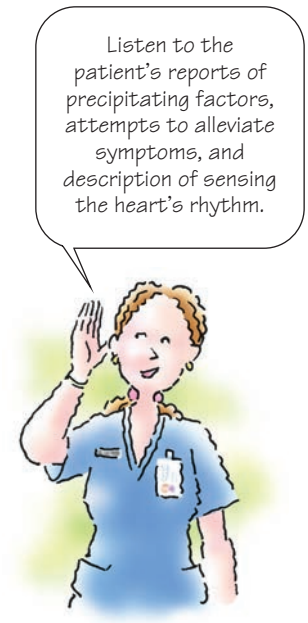
Cardiac arrhythmias may result from:

- enhanced or depressed automaticity
- altered conduction pathways
- abnormal electrical conduction.

What to look for

When a patient presents with a history of symptoms suggestive of cardiac arrhythmias, or has been treated for a cardiac arrhythmia, be alert for:

- reports of precipitating factors, such as exercise, smoking, sleep, emotional stress, exposure to heat or cold, caffeine intake, position changes, or recent illnesses
- attempts to alleviate the symptoms, such as coughing, rest, medications, or deep breathing
- reports of sensing the heart's rhythm, such as palpitations, irregular beating, skipped beats, or rapid or slow heart rate.



A matter of degree

Physical examination findings vary depending on the arrhythmia and the degree of hemodynamic compromise.

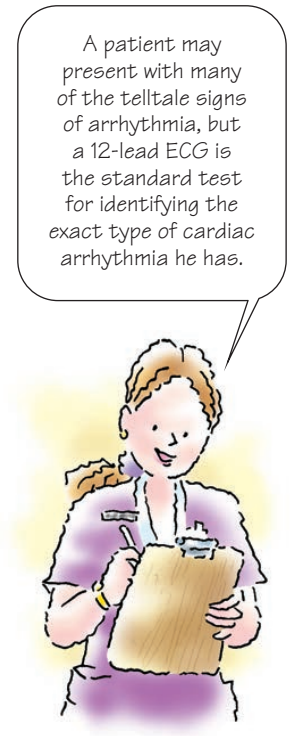
Circulatory failure along with an absence of pulse and respirations is found with asystole, ventricular fibrillation, and sometimes with ventricular tachycardia.

That's not all

Additional findings may include:

- pallor
- cold and clammy extremities
- reduced urine output
- dyspnea

- hypotension
- weakness
- chest pains
- dizziness
- syncope
- anxiety
- fatigue
- auscultation of S_3.



What tests tell you

- A 12-lead ECG is the standard test for identifying cardiac arrhythmias. A 15-lead ECG (in which additional leads are applied to the right side of the chest) or an 18-lead ECG (in which additional leads are also added to the posterior scapular area) may be done to provide more definitive information about the patient's right ventricle and posterior wall of the left ventricle. (See *Understanding cardiac arrhythmias*, pages 266 to 271.)
- Laboratory testing may reveal electrolyte abnormalities, hypoxemia or acid–base abnormalities (with ABG analysis), or drug toxicities as the cause of arrhythmias.
- Exercise testing may reveal exercise-induced arrhythmias.
- Electrophysiologic testing may be used to identify the mechanism of an arrhythmia and location of accessory pathways and to assess the effectiveness of antiarrhythmic drugs.

How it's treated

The goals of treatment are to return pacer function to the sinus node, increase or decrease ventricular rate to normal, regain AV synchrony, and maintain normal sinus rhythm.

Treatments to correct abnormal rhythms include therapy with:

- antiarrhythmic drugs
- electrical conversion with defibrillation and cardioversion
- Valsalva's maneuver
- temporary or permanent placement of a pacemaker to maintain heart rate
- ICD if indicated
- surgical removal or cryotherapy of an irritable ectopic focus to prevent recurring arrhythmias
- management of the underlying disorder such as correction of hypoxia.

What to do

Care for the patient experiencing a cardiac arrhythmia as follows:

- Evaluate the patient's ECG regularly for arrhythmia and assess hemodynamic parameters as indicated. Document arrhythmias and notify the practitioner immediately.

- When life-threatening arrhythmias develop, rapidly assess the patient's LOC, pulse and respiratory rates, and hemodynamic parameters. Monitor his ECG continuously. Be prepared to initiate CPR if indicated.
- Administer oxygen to help improve myocardial oxygen supply.
- Administer analgesics, as appropriate, and help the patient decrease anxiety.
- Assess the patient for predisposing factors, such as fluid and electrolyte imbalance, and signs of drug toxicity, especially with digoxin.
- Administer medications as ordered; monitor for adverse effects; and monitor vital signs, hemodynamic parameters (as appropriate), and appropriate laboratory studies. Prepare to assist with or perform cardioversion or defibrillation if indicated.
- If you suspect drug toxicity, report it to the practitioner immediately and withhold the next dose.
- If a temporary pacemaker needs to be inserted, make sure that a fresh battery is installed to avoid temporary pacemaker malfunction and carefully secure the external catheter wires and the pacemaker box.
- After pacemaker insertion, monitor the patient's pulse rate regularly and watch for signs of pacemaker failure and decreased cardiac output.

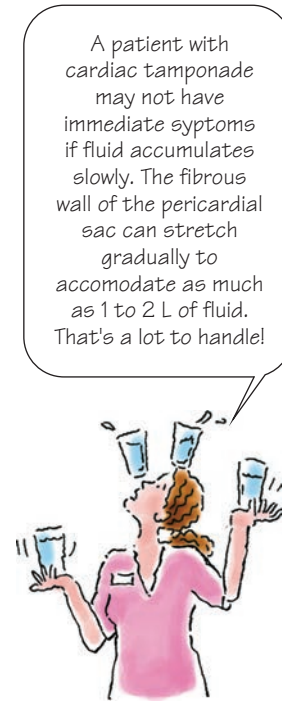
Cardiac tamponade

Cardiac tamponade is a rapid, unchecked increase in pressure in the pericardial sac. This compresses the heart, impairs diastolic filling, and reduces cardiac output.

Pericardial pressure

The increase in pressure usually results from blood or fluid accumulation in the pericardial sac. Even a small amount of fluid (50 to 100 ml) can cause a serious tamponade if it accumulates rapidly.

If fluid accumulates rapidly, cardiac tamponade requires emergency lifesaving measures to prevent death. A slow accumulation and increase in pressure 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.



What causes it

Cardiac tamponade may result from:

- idiopathic causes (such as Dressler's syndrome)
- effusion (from cancer, bacterial infections, tuberculosis, and, rarely, acute rheumatic fever)
- hemorrhage due to trauma (such as gunshot or stab wounds of the chest)

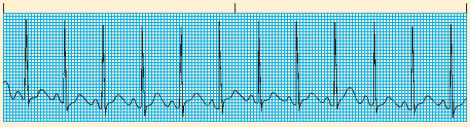
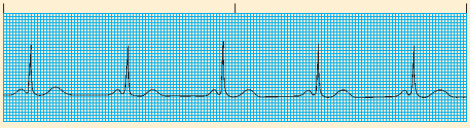
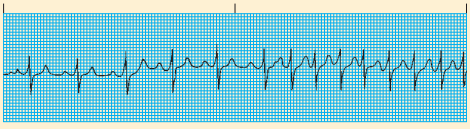
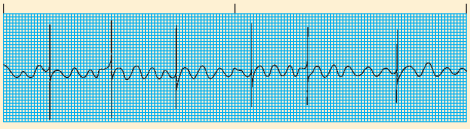
(Text continues on page 272.)

Understanding cardiac arrhythmias

Here's an outline of many common cardiac arrhythmias and their features, causes, and treatments. Use a normal ECG strip, if available, to compare normal cardiac rhythm configurations with the rhythm strips shown here.

Characteristics of normal sinus rhythm include:

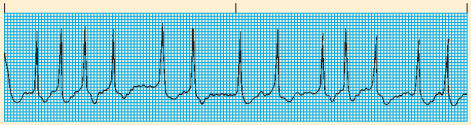
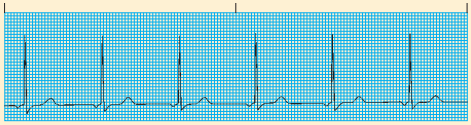
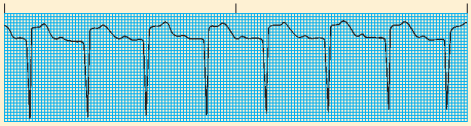
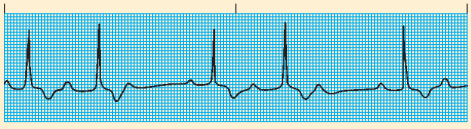
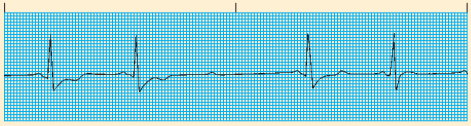
- ventricular and atrial rates of 60 to 100 beats per minute
- regular and uniform QRS complexes and P waves
- PR interval of 0.12 to 0.20 second
- QRS duration <0.12 second
- identical atrial and ventricular rates, with constant PR intervals.

Arrhythmia	Features
Sinus tachycardia 	• Atrial and ventricular rhythms regular • Rate >100 beats/minute; rarely, >160 beats/minute • Normal P waves preceding each QRS complex
Sinus bradycardia 	• Atrial and ventricular rhythms regular • Rate <60 beats/minute • Normal P waves preceding each QRS complex
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 waves • P waves preceding each QRS complex • Sudden onset and termination of arrhythmia
Atrial flutter 	• Atrial rhythm regular; rate 250 to 400 beats per minute • Ventricular rate variable, depending on degree of AV block (usually 60 to 100 beats per minute) • No P waves; atrial activity appears as flutter waves (f waves); sawtooth configuration common in lead II • QRS complexes are uniform in shape but often irregular in rhythm.

Causes	Treatment
• Normal physiologic response to fever, exercise, anxiety, pain, dehydration; may also accompany shock, left-sided heart failure, hyperthyroidism, anemia, hypovolemia, pulmonary embolism, and anterior wall MI • May also occur with atropine, epinephrine, isoproterenol (Isuprel), aminophylline, caffeine, alcohol, cocaine, amphetamine, and nicotine use	• Correction of underlying cause • Beta-adrenergic blockers or calcium channel blocker
• Normal, in well-conditioned heart, as in an athlete, or during sleep • Increased intracranial pressure, vagal stimulation, vomiting, sick sinus syndrome, hypothyroidism, inferior wall MI, and hypothermia • May also occur with calcium channel blockers, beta-adrenergic blocker, digoxin (Lanoxin), and morphine use	• Correction of underlying cause • For low CO, dizziness, weakness, altered LOC, or low blood pressure; advanced cardiac life support (ACLS) protocol for administration of atropine • Temporary or permanent pacemaker • Dopamine (Intropin) or epinephrine infusion
• Stress, hypoxia, hypokalemia, cardiomyopathy, MI, valvular disease, Wolff-Parkinson-White syndrome, cor pulmonale, hyperthyroidism, anxiety, hypoxia, rheumatic heart disease • May also occur with digoxin toxicity; use of caffeine, marijuana, central nervous system stimulants, nicotine, or alcohol	• If patient is unstable, immediate cardioversion • If patient is stable, vagal stimulation, Valsalva's maneuver, and carotid sinus massage or adenosine • After rhythm converts, use calcium channel blockers or beta-adrenergic blockers.
• Heart failure, tricuspid or mitral valve disease, pulmonary embolism, cor pulmonale, pericarditis, and hyperthyroidism • May also occur with digoxin toxicity or alcohol use	• If patient is unstable with a ventricular rate >150 beats/minute, immediate cardioversion • If patient is stable, follow ACLS protocol for cardioversion and drug therapy, which may include calcium channel blockers, beta-adrenergic blockers, amiodarone, or digoxin. • Anticoagulation therapy may also be necessary. • Radio frequency ablation to control rhythm

(continued)

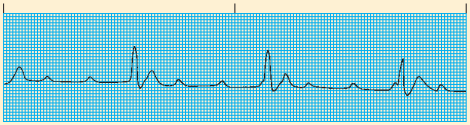
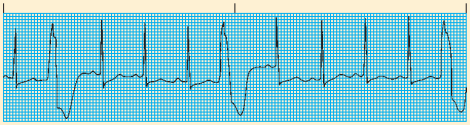
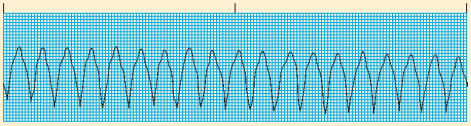
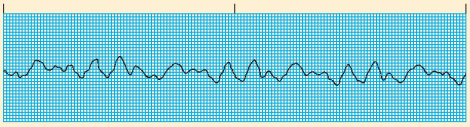
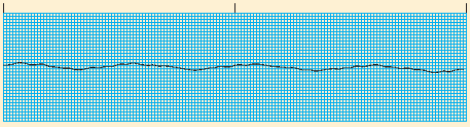
Understanding cardiac arrhythmias *(continued)*

Arrhythmia	Features
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, atrial activity appears as erratic, irregular, baseline fibrillatory waves (f waves)
Junctional rhythm 	• Atrial and ventricular rhythms regular; atrial rate 40 to 60 beats per minute; ventricular rate usually 40 to 60 beats per minute (60 to 100 beats per 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
First-degree AV block 	• Atrial and ventricular rhythms regular • PR interval >0.20 second • P wave precedes QRS complex. • QRS complex normal
Second-degree AV block *Mobitz I (Wenckebach)* 	• Atrial rhythm regular • Ventricular rhythm irregular • Atrial rate exceeds ventricular rate. • PR interval progressively longer with each cycle until QRS complex disappears (dropped beat); PR interval shorter after dropped beat
Second-degree AV block *Mobitz II* 	• Atrial rhythm regular • Ventricular rhythm regular or irregular, with varying degree of block • PR interval constant for conducted beats • P waves normal size and shape, but some aren't followed by a QRS complex

Causes	Treatment
• Heart failure, chronic obstructive pulmonary disease, thyrotoxicosis, pericarditis, ischemic heart disease, pulmonary embolus, hypertension, mitral stenosis, atrial irritation, or complication of coronary bypass or valve replacement surgery • May also occur with nifedipine, digoxin, or alcohol use	• If patient is unstable with a ventricular rate >150 beats/minute, immediate cardioversion • If patient is stable, follow ACLS protocol and drug therapy, which may include calcium channel blockers, beta-adrenergic blockers, amiodarone, or digoxin. • Anticoagulation therapy may also be necessary. • In some patients with refractory atrial fibrillation uncontrolled by drugs, radio frequency catheter ablation
• MI or ischemia, hypoxia, vagal stimulation, and sick sinus syndrome • Valve surgery • May also occur with digoxin toxicity	• Correction of underlying cause • Atropine for symptomatic slow rate • Pacemaker insertion if patient doesn't respond to drugs • Discontinuation of digoxin if appropriate
• May be seen in healthy persons • MI or ischemia, hyperkalemia, complication of coronary bypass or valve surgery • May also occur with digoxin toxicity; use of beta-adrenergic blockers, calcium channel blockers, or amiodarone	• Correction of underlying cause • Possibly atropine if severe symptomatic bradycardia develops • Cautious use of digoxin, calcium channel blockers, and beta-adrenergic blockers
• Inferior wall MI, cardiac surgery, conduction system defects, and vagal stimulation • May also occur with digoxin toxicity; use of beta-adrenergic blockers or calcium channel blockers	• Treatment of underlying cause • Temporary pacemaker for symptomatic bradycardia (atropine usually not helpful) • Discontinuation of digoxin if appropriate
• Severe CAD, anterior wall MI, acute myocarditis, hypertension, conduction system defects, and complication of cardiac surgery	• Temporary or permanent pacemaker • Dopamine, or epinephrine for symptomatic bradycardia (atropine usually not helpful)

(continued)

Understanding cardiac arrhythmias *(continued)*

Arrhythmia	Features
Third-degree AV block *(complete heart block)* 	• 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 duration normal (junctional pacemaker) or wide and bizarre (ventricular pacemaker)
Premature ventricular contraction (PVC) 	• Atrial rhythm regular • Ventricular rhythm may be regular except for aberrant beats. • QRS complex premature, usually followed by a complete compensatory pause • QRS complex wide and distorted, usually >0.12 second; conducted in opposite direction • Premature QRS complexes occurring alone, 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
Ventricular tachycardia 	• Ventricular rate 100 to 250 beats per minute, rhythm usually regular • QRS complexes wide, bizarre, and independent of P waves • P waves not discernible • May start and stop suddenly
Ventricular fibrillation 	• Ventricular rhythm and rate chaotic and rapid • QRS complexes wide and irregular; no visible P waves
Asystole 	• No atrial or ventricular rate or rhythm • No discernible P waves, QRS complexes, or T waves

Causes	Treatment
• Inferior or anterior wall MI, hypoxia, postoperative complication of cardiac surgery, postprocedure complication of radiofrequency ablation in or near AV nodal tissue, and potassium imbalance • May also occur with digoxin toxicity	• Atropine, dopamine, or epinephrine for symptomatic bradycardia • Temporary or permanent pacemaker
• Heart failure; old or acute MI, ischemia, or contusion; myocardial irritation by ventricular catheter or a pacemaker; hypokalemia; hypocalcemia; hypomagnesemia; cardiomyopathy; hypoxia; and acidosis • May also occur with drug toxicity (digoxin, aminophylline, epinephrine, isoproterenol, or dopamine) • Caffeine, tobacco, or alcohol use • Psychological stress, anxiety, pain, or exercise	• If warranted, procainamide, amiodarone, or lidocaine 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
• Myocardial ischemia, MI, or aneurysm; CAD; mitral valve prolapse; cardiomyopathy; ventricular catheters; hypokalemia; hypocalcemia; hypomagnesemia; myocardial reperfusion; acidosis; and hypoxia • May also occur with digoxin, procainamide, epinephrine, or quinidine toxicity • Anxiety	• If pulseless, initiate CPR; follow ACLS protocol for defibrillation, administration of epinephrine or vasopressin followed by amiodarone (lidocaine may be considered if amiodarone isn't available), and advanced airway placement; magnesium sulfate only for torsades de pointes • If regular wide-complex QRS rhythm (monomorphic) present, administer adenosine (follow ACLS protocol); if drug is unsuccessful, cardioversion • If polymorphic (irregular) ventricular tachycardia present, immediate defibrillation • ICD if recurrent ventricular tachycardia
• Myocardial ischemia, MI, untreated ventricular tachycardia, R-on-T phenomenon, hypokalemia, hypomagnesemia, hypoxemia, alkalosis, electric shock, and hypothermia • May also occur with digoxin, epinephrine, or tricyclic antidepressant toxicity	• CPR; follow ACLS protocol for defibrillation, ET intubation, and administration of epinephrine or vasopressin, and amiodarone • ICD if risk for recurrent ventricular fibrillation
• Myocardial ischemia, MI, heart failure, hypoxia, hypokalemia, severe acidosis, shock, ventricular arrhythmia, AV block, pulmonary embolism, heart rupture, hyperkalemia • May also occur with cocaine overdose	• Continue CPR and follow ACLS protocol for ET intubation and administration of epinephrine or vasopressin.

- hemorrhage due to nontraumatic causes (such as anticoagulant therapy in patients with pericarditis or rupture of the heart or great vessels)
- viral or postirradiation pericarditis
- chronic renal failure requiring dialysis
- drug reaction from procainamide, hydralazine, minoxidil, isoniazid (INH), penicillin, or daunorubicin (Cerubidine)
- connective tissue disorders (such as rheumatoid arthritis, systemic lupus erythematosus, rheumatic fever, vasculitis, and scleroderma)
- acute MI.

How it happens

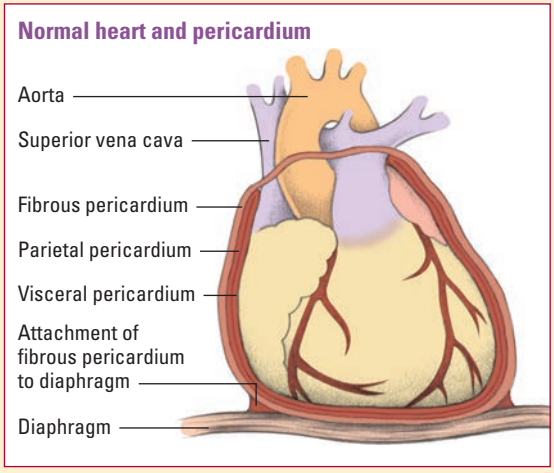
In cardiac tamponade, accumulation of fluid in the pericardial sac causes compression of the heart chambers. This compression obstructs blood flow into the ventricles and reduces the amount of blood that can be pumped out of the heart with each contraction. (See *Understanding cardiac tamponade.*)

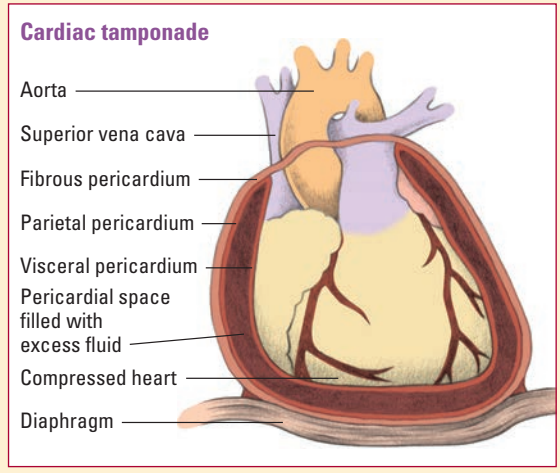
Understanding cardiac tamponade

The pericardial sac, which surrounds and protects the heart, is composed of several layers:

- The fibrous pericardium is the tough outermost membrane.
- The inner membrane, called the *serous membrane*, consists of the visceral and parietal layers.
- The visceral layer clings to the heart and is also known as the *epicardial layer* of the heart.
- The parietal layer lies between the visceral layer and the fibrous pericardium.
- The pericardial space—between the visceral and parietal layers—contains 10 to 30 ml of pericardial fluid. This fluid lubricates the layers and minimizes friction when the heart contracts.

In cardiac tamponade, shown below to the right, blood or fluid fills the pericardial space, compressing the heart chambers, increasing intracardiac pressure, and obstructing venous return. As blood flow into the ventricles decreases, so does CO. Without prompt treatment, low CO can be fatal.





What to look for

Cardiac tamponade has three classic features known as *Beck's triad*:

1. elevated CVP with jugular vein distention
2. muffled heart sounds
3. drop in systolic blood pressure.

That's not all

Other signs include:

- narrowed pulse pressure
- orthopnea
- anxiety
- restlessness
- jugular vein distention with inspiration
- mottling
- clear breath sounds (this helps distinguish cardiac tamponade from heart failure).

What tests tell you

- Chest X-ray shows a slightly widened mediastinum and an enlarged cardiac silhouette.
- ECG may show low-amplitude QRS complex and electrical alternans, an alternating beat-to-beat change in amplitude of the P wave, QRS complex, and T wave. Generalized ST-segment elevation is noted in all leads. An ECG is used to rule out other cardiac disorders; it may reveal changes produced by acute pericarditis.
- PA catheterization discloses increased CVP, right ventricular diastolic pressure, PAWP, and decreased cardiac output/cardiac index.
- Echocardiography may reveal pericardial effusion with signs of right ventricular and atrial compression.
- CT scan or MRI may be used to identify pericardial effusions or pericardial thickening caused by constrictive pericarditis.

How it's treated

The goal of treatment is to relieve intrapericardial pressure and cardiac compression by removing accumulated blood or fluid. This can be done three different ways:

1. pericardiocentesis (needle aspiration of the pericardial cavity)
2. surgical creation of an opening, called a *pericardial window*
3. insertion of a drain into the pericardial sac to drain the effusion.

When pressure's low

If the patient is hypotensive, trial volume loading with crystalloids such as I.V. normal saline solution may be used to maintain systolic blood pressure. An inotropic drug, such as dobutamine, may be necessary to improve myocardial contractility until fluid in the pericardial sac can be removed.

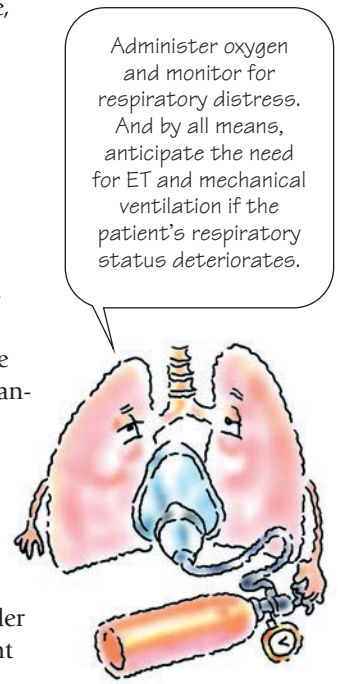
Additional treatments

Additional treatment may be necessary, depending on the cause. Examples of such causes and treatments are:

- traumatic injury—blood transfusion or a thoracotomy to drain reaccumulating fluid or to repair bleeding sites
- heparin-induced tamponade—administration of the heparin antagonist protamine sulfate
- warfarin-induced tamponade—vitamin K administration
- renal failure–induced tamponade—hemodialysis.

What to do

- Monitor the patient's cardiovascular status frequently, at least every hour, noting extent of jugular vein distention, quality of heart sounds, and blood pressure.
- Assess hemodynamic status, including CVP, right atrial pressure, PAP, and PAWP and determine cardiac output.
- Monitor for pulsus paradoxus.
- Be alert for ST-segment and T-wave changes on ECG. Note rate and rhythm and report evidence of any arrhythmias.
- Watch closely for signs of increasing tamponade, increasing dyspnea, and arrhythmias and report immediately.
- Infuse I.V. solutions and inotropic drugs, such as dobutamine, as ordered to maintain the patient's blood pressure.
- Administer oxygen therapy as needed and assess oxygen saturation levels. Monitor the patient's respiratory status for signs of respiratory distress, such as severe tachypnea and changes in the patient's LOC. Anticipate the need for ET intubation and mechanical ventilation if the patient's respiratory status deteriorates.
- Prepare the patient for pericardiocentesis or thoracotomy.



Under pressure

- If the patient has trauma-induced tamponade, assess for other signs of trauma and institute appropriate care, including the use of colloids, crystalloids, and blood component therapy under pressure or by rapid volume infuser if massive fluid replacement

is needed; administration of protamine sulfate for heparin-induced tamponade; and vitamin K administration for warfarin-induced tamponade.
- Assess renal function status closely, monitoring urine output every hour and notifying the practitioner if output is less than 0.5 mg/kg/hour.
- Monitor capillary refill time, LOC, peripheral pulses, and skin temperature for evidence of diminished tissue perfusion.

Cardiogenic shock

Cardiogenic shock is a condition of diminished cardiac output that severely impairs tissue perfusion. It's sometimes called *pump failure*.

Shocking stats

Cardiogenic shock is a serious complication in nearly 15% of all patients hospitalized with acute MI. It typically affects patients whose area of infarction involves 40% or more of left ventricular muscle mass; in such patients, mortality may exceed 85%.

What causes it

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
- cardiomyopathy
- chronic or acute heart failure
- acidosis.

Other offenders

Other causes include myocarditis and depression of myocardial contractility after cardiac arrest and prolonged cardiac surgery.

Mechanical abnormalities of the ventricle, such as acute mitral or aortic insufficiency or an acutely acquired ventricular septal defect or ventricular aneurysm, may also result in cardiogenic shock.

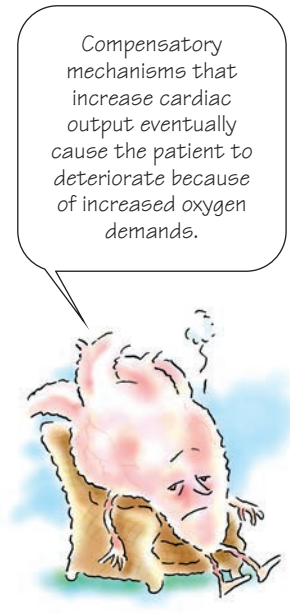
How it happens

Regardless of the cause, here's what happens:
- Left ventricular dysfunction initiates a series of compensatory mechanisms that attempt to increase cardiac output and, in turn, maintain vital organ function.
- As cardiac output falls, baroreceptors in the aorta and carotid arteries initiate responses in the sympathetic nervous system. These

responses, in turn, increase heart rate, left ventricular filling pressure, and afterload to enhance venous return to the heart.
- These compensatory responses initially stabilize the patient but later cause the patient to deteriorate as the oxygen demands of the already compromised heart increase.

Lower and lower output

- The events involved in cardiogenic shock comprise a vicious cycle of low cardiac output, sympathetic compensation, myocardial ischemia, and even lower cardiac output.



What to look for

Cardiogenic shock produces signs of poor tissue perfusion, such as:
- cold, pale, clammy skin
- drop in systolic blood pressure to 30 mm Hg below baseline or a sustained reading below 90 mm Hg that isn't attributable to medication
- tachycardia
- rapid respirations
- oliguria (urine output less than 20 ml/hour)
- anxiety
- confusion
- narrowing pulse pressure
- crackles heard in lungs
- neck vein distention
- S_3, faint heart sounds, and possibly a holosystolic murmur.

What tests tell you

- PAP monitoring reveals increased CVP, PAP, PAWP, and SVR, reflecting an increase in left ventricular end-diastolic pressure (preload) and heightened resistance to left ventricular emptying (afterload) caused by ineffective pumping and increased peripheral vascular resistance. Thermodilution catheterization reveals a reduced cardiac index.
- Invasive arterial pressure monitoring shows systolic arterial pressure less than 90 mm Hg caused by impaired ventricular ejection.
- ABG analysis may show metabolic and respiratory acidosis and hypoxia.
- ECG demonstrates possible evidence of acute MI, ischemia, or ventricular aneurysm and arrhythmias.

- Echocardiography is used to determine left ventricular function and reveals valvular abnormalities.
- Serum enzyme measurements display elevated levels of CK, aspartate aminotransferase, and alanine aminotransferase, which indicate MI or ischemia and suggest heart failure or shock. CK-MB (an isoenzyme of CK that occurs in cardiac tissue) and troponin isoenzyme levels may confirm acute MI.
- Brain natriuretic peptide (BNP) levels are elevated, indicating ventricular overload.
- Cardiac catheterization and echocardiography may reveal other conditions that can lead to pump dysfunction and failure, such as cardiac tamponade, papillary muscle infarct or rupture, ventricular septal rupture, pulmonary emboli, venous pooling (associated with venodilators and continuous or intermittent positive-pressure breathing), hypovolemia, and acute heart failure.

How it's treated

The goal of treatment is to enhance cardiovascular status by increasing cardiac output, improving myocardial perfusion, and decreasing cardiac workload. Treatment consists of administering a combination of cardiovascular drugs and mechanical-assist techniques.

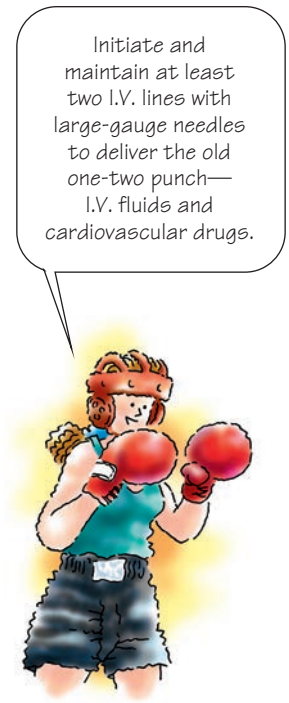
Treatment ABCs

Treatment begins with these measures:

- maintaining a patent airway; preparing for intubation and mechanical ventilation if the patient develops respiratory distress
- supplemental oxygen to increase oxygenation
- continuous cardiac monitoring to detect changes in heart rate and rhythm; administration of antiarrhythmics, as necessary
- initiating and maintaining at least two I.V. lines with large-gauge needles for fluid and drug administration
- I.V. fluids, crystalloids, colloids, or blood products, as necessary, to maintain intravascular volume.

Cardiovascular drugs

Drug therapy may include I.V. dopamine, phenylephrine, or norepinephrine to increase blood pressure and blood flow to kidneys. Inamrinone or dobutamine—inotropic agents that increase myocardial contractility and cardiac output—are commonly used.



Decrease resistance and pressure

A vasodilator, nitroglycerin or nitroprusside, may be used with a vasopressor to further improve cardiac output by decreasing afterload (SVR) 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.

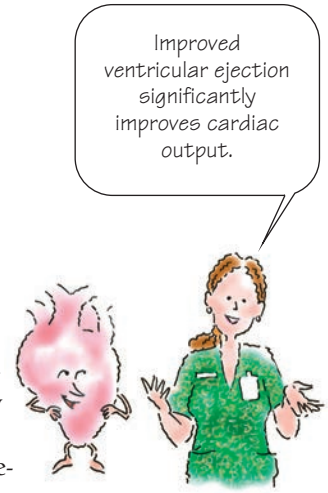
Overloaded and out of control

Diuretics also may be used to reduce preload (PAWP) in patients with fluid volume overload. Antiarrhythmics may also be used to prevent or control arrhythmias that may reduce cardiac output.

Mechanical assistance

Treatment may also include mechanical assistance by IABP to improve coronary artery perfusion and decrease cardiac workload. The IABP is 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 therefore reduce cardiac workload.

Improved ventricular ejection significantly improves cardiac output. Subsequent vasodilation in the peripheral vessels leads to lower preload volume and reduced workload of the left ventricle. This is because of decreasing SVR.



End-stage effort

When drug therapy and IABP insertion fail, a VAD may be inserted to assist the pumping action of the heart. When all other medical and surgical therapies fail, heart transplantation may be considered.

More measures

Additional treatment measures for cardiogenic shock may include:

- thrombolytic therapy or coronary artery revascularization to restore coronary artery blood flow, if cardiogenic shock is due to acute MI
- emergency surgery to repair papillary muscle rupture or ventricular septal defect, if either is the cause of cardiogenic shock.

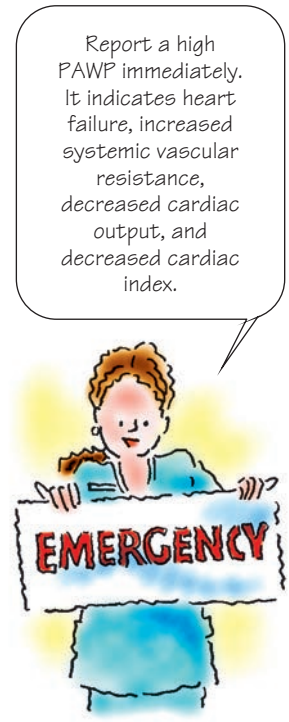
What to do

- Begin I.V. infusions of normal saline solution using a large-bore (14G to 18G) catheter, which allows easier administration of later blood transfusions.

- Administer oxygen by facemask or artificial airway to ensure adequate oxygenation of tissues. Adjust the oxygen flow rate to a higher or lower level, as ABG measurements indicate. Many patients need 100% oxygen, and some require 5 to 15 cm H_2O of positive end-expiratory or continuous positive airway pressure ventilation.

Monitor, record, and then monitor more

- Monitor and record blood pressure, pulse, respiratory rate, and peripheral pulses every 1 to 5 minutes until the patient stabilizes. Monitor cardiac rhythm continuously. Systolic blood pressure less than 80 mm Hg usually results in inadequate coronary artery blood flow, cardiac ischemia, arrhythmias, and further complications of low cardiac output.
- Using a PA catheter, closely monitor CVP, PAP, PAWP, SVR, and cardiac output. High CVP and PAWP readings indicate heart failure, increased SVR, decreased cardiac output, and decreased cardiac index and should be reported immediately.

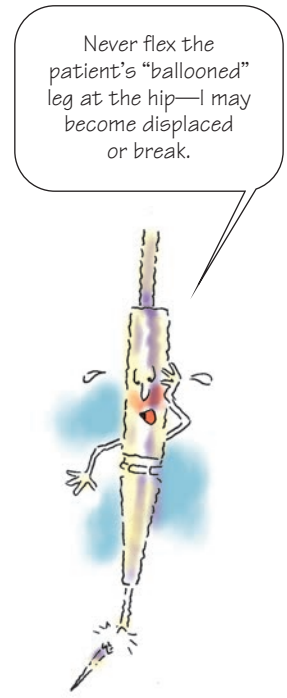


Watch all those fluids

- Determine how much fluid to give by checking blood pressure, urine output, CVP, or PAWP. Whenever the fluid infusion rate is increased, watch for signs of fluid overload, such as an increase in PAWP. If the patient is hypovolemic, preload may need to be increased, typically accomplished with I.V. fluids. However, I.V. fluids must be given cautiously, being increased gradually while hemodynamic parameters are closely monitored. In this situation, diuretics aren't given.
- Insert an indwelling urinary catheter 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 PAWP. Notify the practitioner if urine output doesn't improve.
- Administer a diuretic, such as furosemide, as ordered, to decrease preload and improve stroke volume and cardiac output.
- Monitor ABG values, CBC, and electrolyte levels. Expect to administer sodium bicarbonate by I.V. push if the patient is acidotic. Administer electrolyte replacement therapy as ordered.
- During therapy, assess skin color and temperature and note any changes. Cold, clammy skin may be a sign of continuing peripheral vascular constriction, indicating progressive shock.

Don't move!

- If your patient is on the IABP, move him as little as possible. Never flex the patient's "ballooned" leg at the hip because this may displace or fracture the catheter. Never place the patient in a sitting position for any reason (including chest X-rays) while the balloon is inflated; the balloon will tear through the aorta and result in immediate death.
- During use of the IABP, assess pedal pulses and skin temperature and color to ensure adequate peripheral circulation. Check the dressing over 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.



When to wean

- If the patient becomes hemodynamically stable, gradually reduce the frequency of balloon inflation to wean him from the IABP.
- When weaning the patient from the IABP, watch for ECG changes, chest pain, and other signs of recurring cardiac ischemia as well as for shock.
- Prepare the patient for possible emergency cardiac catheterization to determine eligibility for PTCA or CABG to reperfuse (restore blood flow to) areas with reversible injury patterns.
- To ease emotional stress, plan care measures to allow frequent rest periods and provide as much privacy as possible. Allow family members to visit and comfort the patient as much as possible.

Cardiomyopathy

Cardiomyopathy generally refers to disease of the heart muscle fibers. It takes three main forms:

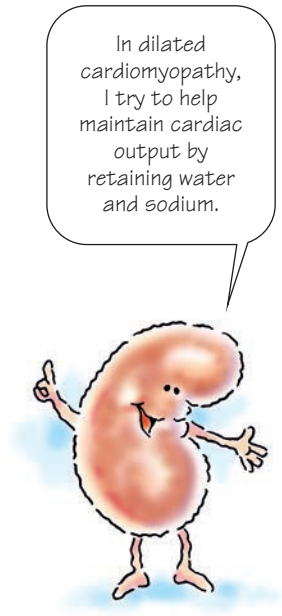
1. dilated
2. hypertrophic
3. restrictive (extremely rare).

Cardiomyopathy is the second most common direct cause of sudden death; CAD is first. Because dilated cardiomyopathy usually isn't diagnosed until its advanced stages, the prognosis is generally poor.

What causes it

Most patients with cardiomyopathy have idiopathic, or primary, disease, but some cases are secondary to identifiable causes. Hypertrophic cardiomyopathy is almost always inherited as a non-sex-linked autosomal dominant trait.

Males and blacks are at greatest risk for cardiomyopathy; other risk factors include hypertension, pregnancy, viral infections, and alcohol use.



How it happens

The disease course in cardiomyopathy depends on the special type, as outlined here.

Dilated cardiomyopathy

Dilated cardiomyopathy primarily affects systolic function. It results from extensively damaged myocardial muscle fibers. Consequently, contractility in the left ventricle decreases.

Poor compensation

As systolic function declines, stroke volume, ejection fraction, and cardiac output decrease. As end-diastolic volumes increase, pulmonary congestion may occur. The elevated end-diastolic volume is a compensatory response to preserve stroke volume despite a reduced ejection fraction.

The sympathetic nervous system is also stimulated to increase heart rate and contractility.

Kidneys kick in

The kidneys are stimulated to retain sodium and water to maintain cardiac output, and vasoconstriction occurs as the renin-angiotensin system is stimulated. When these compensatory mechanisms can no longer maintain cardiac output, the heart begins to fail.

Detrimental dilation

Left ventricular dilation occurs as venous return and SVR increase. The stretching of the left ventricle eventually leads to mitral insufficiency. Subsequently, the atria also dilate, as more work is required to pump blood into the full ventricles. Cardiomegaly is a consequence of dilation of the atria and ventricles. Blood pooling in the ventricles increases the risk of emboli.

Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy primarily affects diastolic function. The features of hypertrophic cardiomyopathy include:

- asymmetrical left ventricular hypertrophy
- hypertrophy of the intraventricular septum
- rapid, forceful contractions of the left ventricle
- impaired relaxation
- obstruction of left ventricular outflow.

Fouled-up filling

The hypertrophied ventricle becomes stiff, noncompliant, and unable to relax during ventricular filling. Consequently, ventricular filling is reduced and left ventricular filling pressure rises, causing increases in left atrial and pulmonary venous pressures and leading to venous congestion and dyspnea.

The increase in venous pressures and venous congestion leads to tachycardia, which causes a decrease in left ventricular filling time. Reduced ventricular filling during diastole and obstruction to ventricular outflow lead to low cardiac output.

Hypertrophy hazards

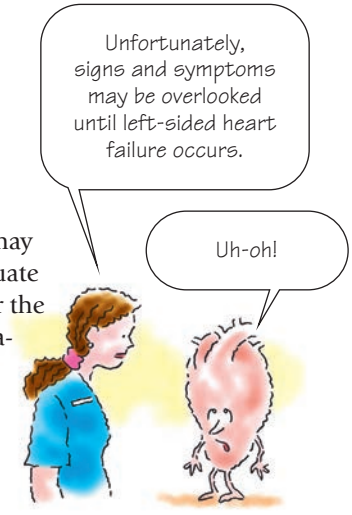
If papillary muscles become hypertrophied and don't close completely during contraction, mitral insufficiency occurs. Moreover, intramural coronary arteries are abnormally small and may not be sufficient to supply the hypertrophied muscle with enough blood and oxygen to meet the increased needs of the hyperdynamic muscle.

Restrictive cardiomyopathy

Restrictive cardiomyopathy is characterized by stiffness of the ventricle caused by left ventricular hypertrophy and endocardial fibrosis and thickening. The ability of the ventricle to relax and fill during diastole is reduced. Furthermore, the rigid myocardium fails to contract completely during systole. As a result, cardiac output decreases.

What to look for

Generally, for patients with dilated or restrictive cardiomyopathy, the onset is insidious. As the disease progresses, exacerbations and hospitalizations are frequent regardless of the type of cardiomyopathy.



Dilated cardiomyopathy

For a patient with dilated cardiomyopathy, signs and symptoms may be overlooked until left-sided heart failure occurs. Be sure to evaluate the patient's current condition and then compare it with that over the past 6 to 12 months. Signs and symptoms of dilated cardiomyopathy may include:

- shortness of breath, orthopnea, dyspnea on exertion, fatigue
- peripheral edema, hepatomegaly, jugular vein distention
- tachycardia, palpitations
- pansystolic murmur associated with mitral and tricuspid insufficiency

- S_3 and S_4 gallop rhythms
- irregular pulse if atrial fibrillation exists
- crackles in lungs.

Hypertrophic cardiomyopathy

Signs and symptoms vary widely among patients with hypertrophic cardiomyopathy. The presenting symptom is commonly syncope or sudden cardiac death. Other possible signs and symptoms include:

- angina
- dyspnea and orthopnea
- fatigue
- systolic ejection murmur along the left sternal border and apex
- ventricular arrhythmias
- irregular pulse with atrial fibrillation, palpitations
- S_4 and possible S_3 gallop rhythms, split S_2 heart sound.

Restrictive cardiomyopathy

A patient with restrictive cardiomyopathy presents with signs of heart failure and other signs and symptoms, including:

- fatigue and weakness
- dyspnea
- orthopnea
- chest pain
- hepatomegaly
- peripheral edema
- S_3 or S_4 gallop rhythms
- systolic murmurs
- heart blocks.

What tests tell you

These tests are used to diagnose cardiomyopathy:

Dilated cardiomyopathy

- Chest X-ray shows an enlarged heart and pulmonary edema.
- An ECG will show biventricular enlargement and, commonly, atrial fibrillation.
- Echocardiogram will show decreased ventricular movement and ejection fraction. It will also demonstrate an increase in atrial and ventricular chamber size and abdomen wall motion. It may also demonstrate mitral valve insufficiency.
- Hemodynamic monitoring will show an increased PAWP and PAP and a decreased cardiac output/cardiac index. In late stages, the CVP may also be elevated.

Hypertrophic cardiomyopathy

- Chest X-ray shows an enlarged heart with pronounced left atrial dilation. Pulmonary congestion may also be seen.
- An ECG will show left atrial enlargement and left ventricular hypertrophy. ST and T-wave changes may be seen. Atrial fibrillation and ventricular arrhythmias, such as ventricular tachycardia and ventricular fibrillation, are also common.
- An echocardiogram will show an enlarged left atrium and hypertrophy of the intraventricular septum. Left ventricular outflow narrowing, if present, can also be seen. Abnormal wall motion may also be present.
- Cardiac catheterization with heart biopsy can provide definitive diagnosis.

Restricted cardiomyopathy

- Chest X-ray shows an enlarged heart and pulmonary edema.
- An ECG will demonstrate low QRS complex voltage. AV heart blocks are commonly seen.
- Echocardiogram will show atrial enlargement. The walls of the ventricles will be thickened but the interior chamber size will be decreased.
- Hemodynamic monitoring will show increased PAP and PAWP. Left and right end-diastolic pressures will also be elevated.

How it's treated

There's no known cure for cardiomyopathy. Treatment is individualized based on the type of cardiomyopathy and the patient's condition.

Dilated cardiomyopathy

For a patient with dilated cardiomyopathy, treatment may involve:

- management of the underlying cause, if it's known
- ACE inhibitors and ARBs to reduce afterload through vasodilation and increase cardiac output
- diuretics, taken with ACE inhibitors, to reduce fluid retention
- digoxin, for patients not responding to ACE inhibitor and diuretic therapy, to improve myocardial contractility
- hydralazine and isosorbide dinitrate, in combination, to produce vasodilation
- beta-adrenergic blockers for patients with mild or moderate heart failure
- antiarrhythmics, such as amiodarone, used cautiously to control arrhythmias
- cardioversion to convert atrial fibrillation to sinus rhythm

- pacemaker insertion to correct arrhythmias
- anticoagulants to reduce the risk of emboli
- revascularization, such as CABG surgery, if dilated cardiomyopathy is due to ischemia
- valvular repair or replacement, if dilated cardiomyopathy is due to valve dysfunction
- lifestyle modifications such as smoking cessation; low-fat, low-sodium diet; physical activity; and abstinence from alcohol
- heart transplantation in patients resistant to medical therapy
- inotropes, such as dobutamine, to improve myocardial contractility and improve heart failure.

Hypertrophic cardiomyopathy

For a patient with hypertrophic cardiomyopathy, treatment may involve:

- beta-adrenergic blockers to slow the heart rate, reduce myocardial oxygen demands, and increase ventricular filling by relaxing the obstructing muscle, thereby increasing cardiac output
- antiarrhythmic drugs, such as amiodarone, to reduce arrhythmias
- cardioversion to treat atrial fibrillation
- anticoagulation to reduce the risk for systemic embolism with atrial fibrillation
- verapamil and diltiazem to reduce ventricular stiffness and elevated diastolic pressures
- ablation of the AV node and implantation of a dual-chamber pacemaker (controversial), in patients with obstructive hypertrophic cardiomyopathy and ventricular tachycardias, to reduce the outflow gradient by altering the pattern of ventricular contraction
- ICD to correct ventricular arrhythmias
- ventricular myotomy or myectomy (resection of the hypertrophied septum) to ease outflow tract obstruction and relieve symptoms
- mitral valve replacement to correct mitral insufficiency
- heart transplantation for intractable symptoms.

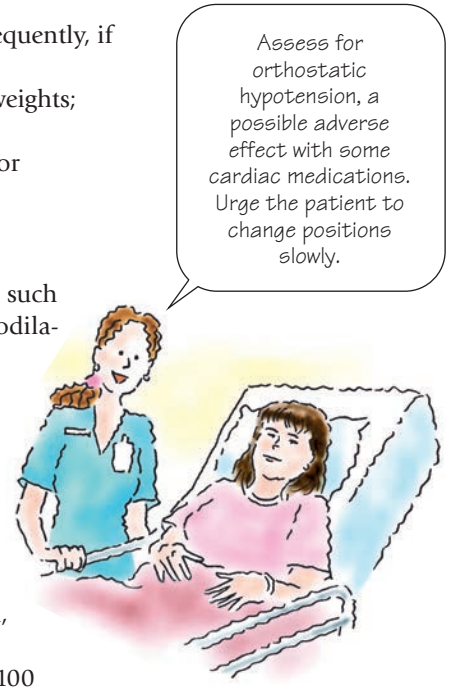
Restrictive cardiomyopathy

For a patient with restrictive cardiomyopathy, treatment may involve:

- management of the underlying cause such as administering deferoxamine to bind iron in restrictive cardiomyopathy due to hemochromatosis
- digoxin, diuretics, and a restricted sodium diet to ease the symptoms of heart failure, although no therapy exists for patients with restricted ventricular filling
- oral vasodilators to control intractable heart failure.

What to do

- Administer drugs, as ordered, to promote adequate heart function.
- Assess hemodynamic status every 2 hours or more frequently, if necessary.
- Monitor intake and output closely and obtain daily weights; institute fluid restrictions as ordered.
- Institute continuous cardiac monitoring to evaluate for arrhythmias.



No sudden moves

- Assess the patient for possible adverse drug reactions, such as orthostatic hypotension associated with use of vasodilators, diuretics, or ACE inhibitors. Urge the patient to change positions slowly.
- Be aware that patients with hypertrophic cardiomyopathy should not receive medication that may decrease preload (diuretics, nitrates) or dopamine or digoxin because the increase in myocardial contractility may worsen the outflow obstruction.
- Auscultate heart and lung sounds, being alert for S_3 and S_4 heart sounds or murmurs, or crackles, rhonchi, and wheezes indicative of heart failure. Monitor vital signs for changes, especially a heart rate greater than 100 beats/minute, respiratory rate greater than 20 breaths per minute, and a systolic blood pressure less than 90 mm Hg, all of which suggest heart failure.
- Assist the patient with ADLs to decrease oxygen demand.

Oxygen orders

- Administer supplemental oxygen as ordered. Assess for changes in LOC, such as restlessness or decreased responsiveness, indicating diminished cerebral perfusion. If the patient has a PA catheter in place, evaluate mixed venous oxygen saturation levels; if not, monitor oxygen saturation levels using pulse oximetry.
- Organize care to promote periods of rest for the patient.
- Prepare the patient, as indicated, for insertion of pacemaker, ICD, IABP, or cardiac transplantation.

Heart failure

Heart failure occurs when the heart can't pump enough blood to meet the metabolic needs of the body. The American Heart

Association and American College of Cardiology developed a classification system for heart failure staging patients from stage A to D based on physical examination, diagnostic tests, and clinical symptoms.

Heart failure results in intravascular and interstitial volume overload and poor tissue perfusion. An individual with heart failure experiences reduced exercise tolerance, a reduced quality of life, and a shortened life span.

What causes it

The most common cause of heart failure is CAD, but it also occurs in infants, children, and adults with congenital and acquired heart defects.

How it happens

Heart failure may be classified into four general categories:

1. left-sided heart failure
2. right-sided heart failure
3. systolic dysfunction
4. diastolic dysfunction.

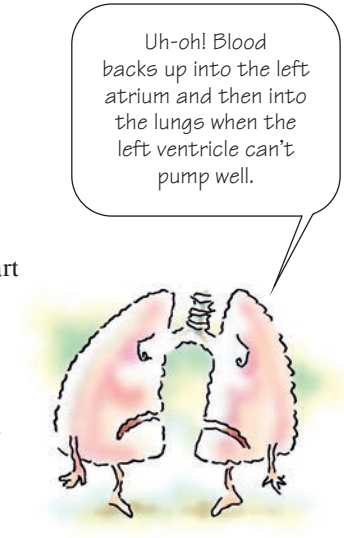
When the left loses its faculties

Left-sided heart failure is a result of ineffective left ventricular contractile function.

As the pumping ability of the left ventricle fails, cardiac output drops. Blood is no longer effectively pumped out into the body; it backs up into the left atrium and then into the lungs, causing pulmonary congestion, dyspnea, and activity intolerance.

If the condition persists, pulmonary edema and right-sided heart failure may result. Common causes include:

- left ventricular infarction
- hypertension
- aortic and mitral valve stenosis.



When right goes wrong

Right-sided heart failure results from ineffective right ventricular contractile function. The most common cause for right-sided heart failure is left-sided heart failure; however, it can result from a right ventricular MI.

When blood isn't pumped effectively through the right ventricle to the lungs, blood backs up into the right atrium and into the peripheral circulation. The patient gains weight and develops peripheral edema and engorgement of the kidney and other organs.

Blame it on the left

Right-sided heart failure may be due to an acute right ventricular infarction or a pulmonary embolus. However, the most common cause is profound backward flow due to left-sided heart failure.

Other causes of right-sided heart failure include:

- arrhythmias
- volume overload
- mitral and pulmonic valve stenosis
- cardiomyopathy.

Just can't pump enough

Systolic dysfunction occurs when the left ventricle can't pump enough blood out to the systemic circulation during systole and the ejection fraction falls. Consequently, blood backs up into the pulmonary circulation and pressure increases in the pulmonary venous system. Cardiac output decreases; weakness, fatigue, and shortness of breath may occur.

Causes of systolic dysfunction include:

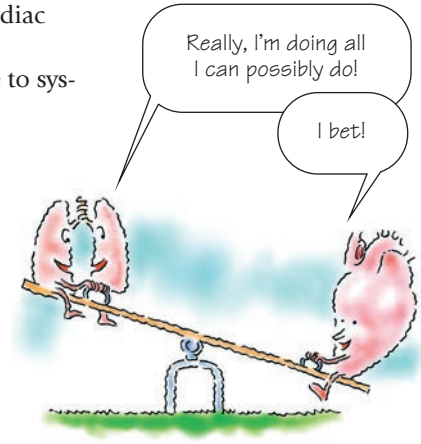
- MI
- dilated cardiomyopathy
- arrhythmias
- aortic valve insufficiency
- acute rheumatic fever.

It all goes to swell from here.

Diastolic dysfunction occurs when the ability of the left ventricle to relax and fill during diastole is reduced and the stroke volume falls. Therefore, higher volumes are needed in the ventricles to maintain cardiac output. Consequently, pulmonary congestion and peripheral edema develop.

Diastolic dysfunction may occur as a result of left ventricular hypertrophy, hypertension, cardiomyopathy, MI, or cardiac tamponade.

This type of heart failure is less common than that due to systolic dysfunction, and treatment isn't as clear.



Compensatory mechanisms

All types of heart failure eventually lead to reduced cardiac output, which triggers compensatory mechanisms that improve cardiac output at the expense of increased ventricular work. The compensatory mechanisms include:

- increased sympathetic activity
- activation of the renin-angiotensin-aldosterone system
- ventricular dilation
- ventricular hypertrophy.

Increased sympathetic activity

Increased sympathetic activity—a response to decreased cardiac output and blood pressure—enhances 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.

Renin-angiotensin-aldosterone system

Increased sympathetic activity also restricts blood flow to the kidneys, causing them to secrete renin which, in turn, converts angiotensinogen to angiotensin I, which then becomes angiotensin II—a potent vasoconstrictor. Angiotensin causes the adrenal cortex to release aldosterone, leading to sodium and water retention and an increase in circulating blood volume.

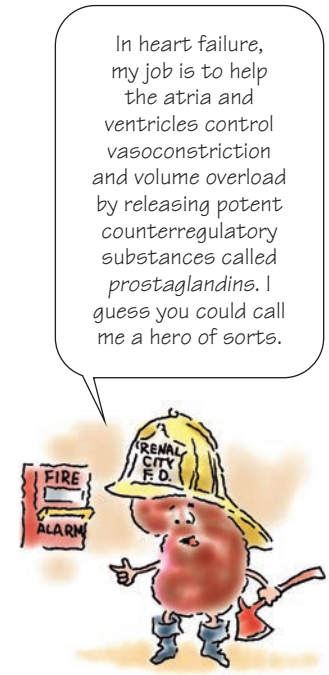
This renal mechanism is helpful; however, if it persists unchecked, it can aggravate heart failure, as the heart struggles to pump against the increased volume.

Ventricular dilation

In ventricular dilation, an increase in end-diastolic ventricular volume (preload) causes increased stroke work and stroke volume during contraction. This stretches cardiac muscle fibers so that the ventricle can accept the increased volume. Eventually, the muscle becomes stretched beyond optimum limits and contractility declines.

Ventricular hypertrophy

In ventricular hypertrophy, an increase in ventricular muscle mass allows the heart to pump against increased resistance to the outflow of blood, improving cardiac output. However, this increased muscle mass also increases the myocardial oxygen requirements.



Compromising situation

An increase in the 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.

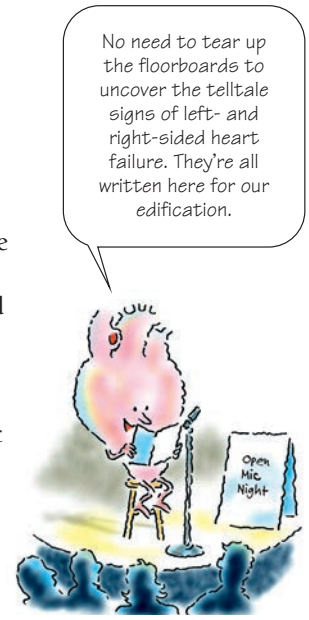
Counterregulatory substances

In heart failure, counterregulatory substances—prostaglandins, atrial natriuretic factor, and BNP—are produced in an attempt to reduce the negative effects of volume overload and vasoconstriction caused by the compensatory mechanisms.

Kidneys' contributions

The kidneys release the prostaglandins prostacyclin and prostaglandin E2, which are potent vasodilators. These vasodilators also

act to reduce volume overload produced by the renin-angiotensin-aldosterone system by inhibiting sodium and water reabsorption by the kidneys.



Counteracting hormones

Atrial natriuretic factor is a hormone that's secreted mainly by the atria in response to stimulation of the stretch receptors in the atria caused by excess fluid volume. This hormone works to counteract the negative effects of sympathetic nervous system stimulation and the renin-angiotensin-aldosterone system by producing vasodilation and diuresis.

BNP is another hormone that's secreted by the ventricle in response to increased ventricular pressures. BNP works in the same manner as atrial natriuretic factor to help counteract the sympathetic nervous system and the renin-angiotensin-aldosterone system.

What to look for

Learn to recognize the signs and symptoms of both right- and left-sided heart failure to ensure that your patient receives attention promptly.

Left-sided heart failure

Look for these early and later signs of disease.

Early bird specials

Early signs and symptoms of left-sided heart failure include:

- dyspnea
- orthopnea
- paroxysmal nocturnal dyspnea
- fatigue
- nonproductive cough.

Late night leftovers

Later clinical manifestations of left-sided heart failure may include:

- crackles on auscultation
- hemoptysis
- displacement of the PMI toward the left anterior axillary line
- tachycardia
- S_3 heart sound
- S_4 heart sound
- cool, cyanotic skin
- confusion.

Right-sided heart failure

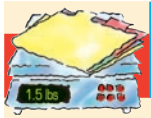
Look for these clinical manifestations of right-sided heart failure:

- neck vein distention
- hepatojugular reflux and hepatomegaly
- right upper quadrant pain
- anorexia and nausea
- nocturia
- weight gain
- pitting edema
- ascites or anasarca
- S_3 heart sound.

What tests tell you

These tests are used to diagnose heart failure:

- Chest X-ray shows increased pulmonary vascular markings, interstitial edema, or pleural effusion and cardiomegaly.
- ECG may indicate hypertrophy, ischemic changes, or infarction and may also reveal tachycardia.
- Laboratory testing may reveal abnormal liver function, elevated BUN and creatinine levels, and elevated BNP levels. (See *BNP: A potent predictor.*)
- ABG analysis may reveal hypoxemia from impaired gas exchange and respiratory alkalosis because the patient blows off more carbon dioxide as respiratory rate increases in compensation.
- Echocardiography may reveal left ventricular hypertrophy, dilation, and abnormal contractility.



Weighing the evidence

BNP: A potent predictor

It has been shown that elevated levels of B-type natriuretic peptide (BNP) can predict sudden death in patients with heart failure. In a follow-up study, researchers sought to determine the best predictors of mortality by comparing BNP levels with other established mortality predictors: peak oxygen consumption, BUN levels, systolic blood pressure, and pulmonary capillary wedge pressure. They analyzed data from 1,215 congestive heart failure patients and determined that BNP was the most robust predictor of mortality. They concluded that analyzing BNP levels could be useful in determining the urgency and timing of cardiac transplantation.

Source: Sachdeva, A., Horwich, T. B., & Fonarow, G. C. (2010). Comparison of usefulness of each of five predictors of mortality and urgent transplantation in patients with advanced heart failure. *American Journal of Cardiology, 106*(6), 830–835.

- Pulmonary artery monitoring typically demonstrates elevated PAP and PAWP, left ventricular end-diastolic pressure and decreased cardiac output/cardiac index in left-sided heart failure, and elevated right atrial pressure or CVP in right-sided heart failure.
- Radionuclide ventriculography may reveal an ejection fraction less than 40%; in diastolic dysfunction, the ejection fraction may be normal.

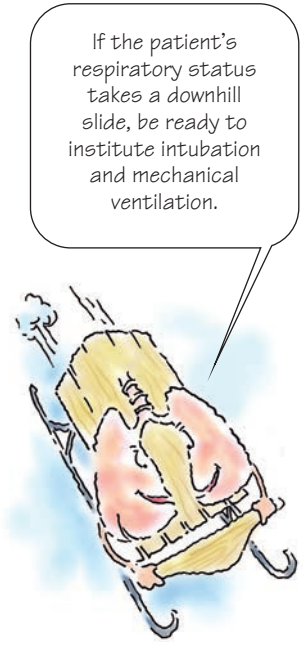
How it's treated

The goal of therapy is to improve pump function. Correction of heart failure may involve:

- treatment of the underlying cause, if it's known
- diuretics to reduce fluid volume overload, venous return, and preload
- ACE inhibitors for patients with left ventricle dysfunction to reduce production of angiotensin II, resulting in preload and afterload reduction
- beta-adrenergic blockers in patients with mild to moderate heart failure caused by left ventricular systolic dysfunction to prevent remodeling
- digoxin for patients with heart failure due to left ventricular systolic dysfunction to increase myocardial contractility, improve cardiac output, reduce the volume of the ventricle, and decrease ventricular stretch
- diuretics, nitrates, morphine, and oxygen to treat pulmonary edema
- administration of synthetic BNP medications, such as nesiritide (Natrecor), to help increase contractility
- lifestyle modifications to reduce symptoms of heart failure, such as weight loss, if obese; limited sodium (to 2 g/day) and alcohol intake; reduced fat intake; smoking cessation; stress reduction; and development of an exercise program
- CABG surgery or angioplasty for patients with heart failure due to CAD
- heart transplantation in patients receiving aggressive medical treatment but still experiencing limitations or repeated hospitalizations
- other surgery or invasive procedures, such as cardiomyoplasty, insertion of an IABP, partial left ventriculectomy, use of a mechanical VAD, and implantation of an ICD or a biventricular pacemaker.

What to do

- Place the patient in Fowler's position to maximize chest expansion and give supplemental oxygen, as ordered, to ease his breathing. Monitor oxygen saturation levels and ABGs as indicated. If respiratory status deteriorates, anticipate the need for ET intubation and mechanical ventilation.



Feel the rhythm

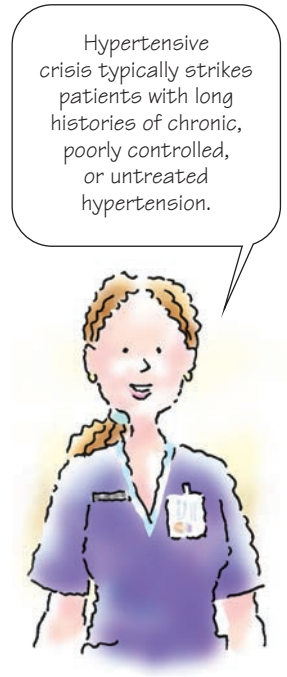
- Institute continuous cardiac monitoring and notify the practitioner of changes in rhythm and rate. If the patient develops tachycardia, administer beta-adrenergic blockers as ordered; if atrial fibrillation is present, administer anticoagulants or antiplatelet agents as ordered to prevent thrombus formation.
- If the patient develops a new arrhythmia, obtain a 12-lead ECG immediately.
- Monitor hemodynamic status, including cardiac output, cardiac index, and pulmonary and systemic vascular pressures closely, at least hourly, noting trends. If available, institute continuous cardiac output monitoring.
- Administer medications as ordered. Check apical heart rate before administering digoxin.
- Assess respiratory status frequently, at least every 1 to 2 hours. Auscultate lungs for abnormal breath sounds, such as crackles, wheezes, and rhonchi. Encourage coughing and deep breathing.
- Obtain daily weights and observe for peripheral edema.
- Assess hourly urine output. Also, monitor fluid intake, including I.V. fluids.
- Frequently monitor BUN and serum creatinine; liver function studies; and serum potassium, sodium, chloride, magnesium, and BNP levels daily.

Event planner

- Organize all activities to provide maximum rest periods. Assess for signs of activity intolerance, such as increased shortness of breath, chest pain, increased arrhythmias, heart rate greater than 120 beats/minute, and ST-segment changes, and have the patient stop activity.
- To prevent deep vein thrombosis caused by vascular congestion, assist the patient with ROM exercises. Enforce bed rest and apply antiembolism stockings or intermittent compression devices.
- Prepare the patient for surgical intervention or insertion of IABP or ICD if indicated.

Hypertensive crisis

A hypertensive emergency, commonly called *hypertensive crisis*, refers to the abrupt, acute, and marked increase in blood pressure from the patient's baseline that ultimately leads to acute and rapidly progressing end-organ damage.



Rapid rise

Typically, the patient's diastolic blood pressure is greater than 120 mm Hg, and his MAP is greater than 150 mm Hg. The increased blood pressure value, although important, is probably less important than how rapidly the blood pressure increases. Arterial lines will be utilized to titrate therapy to the desired result of tissue perfusion and preservation of organ function.

What causes it

Most patients who develop hypertensive crisis have long histories of chronic, poorly controlled, or untreated primary hypertension. Conditions that cause secondary hypertension, such as pheochromocytoma, Cushing's syndrome, or autonomic dysreflexia, may also be responsible.

How it happens

Arterial blood pressure is a product of total peripheral resistance and cardiac output:

- Cardiac output is increased by conditions that increase heart rate, stroke volume, or both.
- Peripheral resistance is increased by factors that increase blood viscosity or reduce the lumen size of vessels, especially the arterioles.

Faulty mechanisms

Hypertension may result from a disturbance in one of the body's intrinsic mechanisms, including:

- renin-angiotensin system
- autoregulation
- sympathetic nervous system
- antidiuretic hormone.

Up with pressure

The renin-angiotensin system increases blood pressure in these ways:

- Sodium depletion, reduced blood pressure, and dehydration stimulate renin release.

- Renin reacts with angiotensinogen, a liver enzyme, and converts it to angiotensin I, which increases preload and afterload.
- Angiotensin I converts to angiotensin II in the lungs; angiotensin II is a potent vasoconstrictor that targets the arterioles.
- Circulating angiotensin II increases preload and afterload by stimulating the adrenal cortex to secrete aldosterone. This increases blood volume by conserving sodium and water.

Maintaining flow

In autoregulation, several intrinsic mechanisms together change an artery's diameter to maintain tissue and organ perfusion despite fluctuations in systemic blood pressure.

These mechanisms include stress relaxation and capillary fluid shifts:

- In stress relaxation, blood vessels gradually dilate when blood pressure increases, reducing peripheral resistance.
- In capillary fluid shift, plasma moves between vessels and extravascular spaces to maintain intravascular volume.

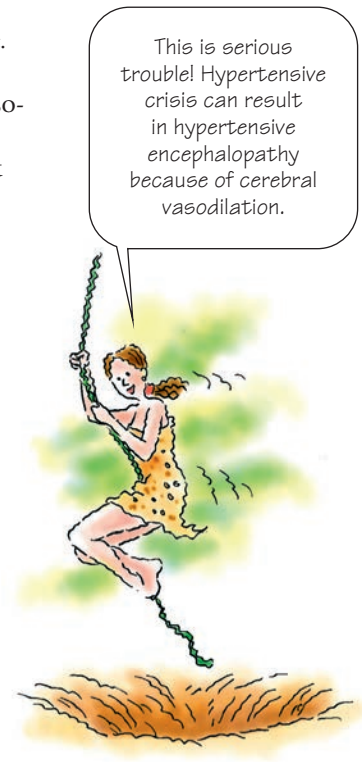
Taking control

Sympathetic nervous system mechanisms control blood pressure. When blood pressure decreases, baroreceptors in the aortic arch and carotid sinuses decrease their inhibition of the medulla's vasomotor center.

Consequent increases in sympathetic stimulation of the heart by norepinephrine increases cardiac output by:

- strengthening the contractile force
- raising the heart rate
- augmenting peripheral resistance by vasoconstriction.

Stress can also stimulate the sympathetic nervous system to increase cardiac output and peripheral vascular resistance. The release of antidiuretic hormone can regulate hypotension by increasing reabsorption of water by the kidney. In reabsorption, blood plasma volume increases, thus raising blood pressure. In hypertensive crisis, one or more of these regulating mechanisms is disrupted.



Strain for the brain

Hypertensive crisis can result in hypertensive encephalopathy because of cerebral vasodilation from an inability to maintain autoregulation. Blood flow increases, causing an increase in pressure and subsequent cerebral edema. This increase in pressure damages the intimal and medial lining of the arterioles.

What to look for

Your assessment of a patient in hypertensive crisis almost always reveals a history of hypertension that's poorly controlled or hasn't been treated. Signs and symptoms may include:

- severe, throbbing headache
- vomiting
- irritability
- confusion
- blurred vision or diplopia
- dyspnea on exertion, orthopnea, or paroxysmal nocturnal dyspnea
- angina
- possible left ventricular heave palpated at the mitral valve area
- S_4 heart sound
- acute retinopathy with retinal exudates.

Check the head

If the patient has hypertensive encephalopathy, you may note:

- decreased LOC
- disorientation
- seizures
- focal neurologic deficits, such as hemiparesis, and unilateral sensory deficits
- papilledema
- temporary vision loss.

Kidney-related consequences

If the hypertensive emergency has affected the kidneys, you may note reduced urine output as well as elevated BUN and creatinine levels.

What tests tell you

- Blood pressure measurement confirms the diagnosis of hypertensive emergency. Blood pressure measurement, obtained several times at an interval of at least 2 minutes, reveals an elevated diastolic pressure greater than 120 mm Hg.
- If there's renal involvement, BUN may be greater than 20 mg/dl and serum creatinine level may be greater than 1.3 mg/dl.
- ECG may reveal ischemic changes or left ventricular hypertrophy.
- Echocardiography may reveal increased wall thickness with or without an increase in left ventricular size.
- Chest X-ray may reveal enlargement of the cardiac silhouette with left ventricular dilation or pulmonary congestion and pleural effusions with heart failure.

- Urinalysis results may be normal unless there's renal impairment; then specific gravity is low (less than 1.010); hematuria, casts, and proteinuria may also be found. If the patient's condition is due to a disease condition, such as pheochromocytoma, a 24-hour urine test reveals increases in vanillylmandelic acid and urinary catecholamines.
- Renal ultrasound may reveal renal artery stenosis.
- CT or MRI of the brain may show cerebral edema or hemorrhage.

How it's treated

Treatment is focused immediately on reducing the patient's blood pressure with I.V. antihypertensive therapy. However, care must be taken not to reduce the patient's blood pressure too rapidly because the patient's autoregulatory control is impaired.

Slow pressure cuts

The current recommendation is to reduce the blood pressure by no more than 25% of the MAP over the first 2 hours. Further reductions should occur over the next several days.

More measures

- Sodium nitroprusside given as an I.V. infusion and titrated according to the patient's response is the drug of choice. It has a rapid onset of action, and its effects cease within 1 to 5 minutes of stopping the drug. Thus, if the patient's blood pressure drops too low, stopping the drug almost immediately allows the blood pressure to increase.
- Other agents that may be used include labetalol, nitroglycerin (the drug of choice for treating hypertensive emergency when myocardial ischemia, acute MI, or pulmonary edema is present), and hydralazine (specifically indicated for treating hypertension in pregnant women with preeclampsia).
- Lifestyle changes may include weight reduction, smoking cessation, exercise, and dietary changes.
- After the acute episode is controlled, maintenance pharmacotherapy to control blood pressure plays a key role.

What to do

- Immediately obtain the patient's blood pressure.
- If not already in place, institute continuous cardiac and arterial pressure monitoring to assess blood pressure directly; determine the patient's MAP.
- Assess ABGs. Monitor the patient's oxygen saturation level using pulse oximetry; if you're monitoring the patient hemodynamically,

assess mixed venous oxygen saturation. Administer supplemental oxygen, as ordered, based on the findings.

- Administer I.V. antihypertensive therapy as ordered; if using nitroprusside, wrap the container in foil to protect it from the light and titrate the dose based on specified target ranges for systolic and diastolic pressures. Immediately stop the drug if the patient's blood pressure drops below the target range.
- Monitor blood pressure every 1 to 5 minutes while titrating drug therapy, then every 15 minutes to 1 hour as the patient's condition stabilizes.
- Continuously monitor ECGs and institute treatment as indicated if arrhythmias occur. Auscultate the patient's heart, noting signs of heart failure, such as S_3 or S_4 heart sounds.
- Assess the patient's neurologic status every hour initially and then every 4 hours as the patient's condition stabilizes.
- Monitor urine output every hour and notify the practitioner if output is less than 0.5 ml/kg/hour. Evaluate BUN and serum creatinine levels for changes and monitor daily weights.
- Obtain serum thiocyanate levels after 48 hours of therapy and then regularly thereafter while the patient is receiving nitroprusside.
- Administer other antihypertensives as ordered. As the patient's condition stabilizes, expect to begin oral antihypertensive therapy while gradually weaning I.V. drugs to prevent hypotension. If the patient is experiencing fluid overload, administer diuretics as ordered.
- Assess the patient's vision and report changes, such as increased blurred vision, diplopia, or loss of vision.
- Administer analgesics as ordered for headache; keep your patient's environment quiet, with low lighting.

Pericarditis

Pericarditis is an inflammation of the pericardium, the fibroserous sac that envelops, supports, and protects the heart. It occurs in 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.

What causes it

Pericarditis may result from:

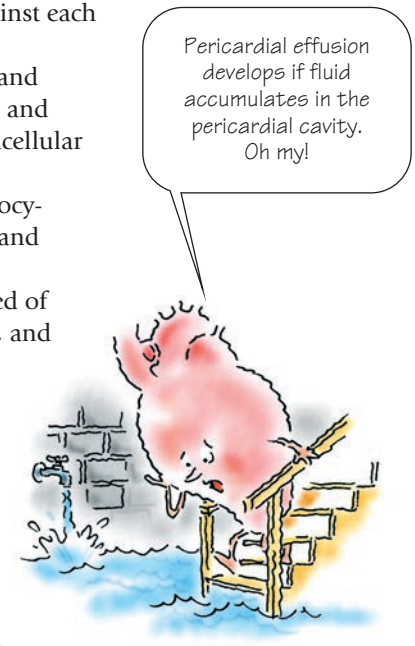
- idiopathic factors (most common in acute pericarditis)
- bacterial, fungal, or viral infection (infectious pericarditis)

- neoplasms (primary disease or metastases from lungs, breasts, or other organs)
- high-dose radiation to the chest
- uremia
- hypersensitivity or autoimmune disease, such as acute rheumatic fever (the most common cause of pericarditis in children), systemic lupus erythematosus, and rheumatoid arthritis
- previous cardiac injury, such as MI (Dressler's syndrome), trauma, or surgery (postcardiotomy syndrome) that leaves the pericardium intact but causes blood to leak into the pericardial cavity
- drugs, such as hydralazine, procainamide, or daunorubicin.

How it happens

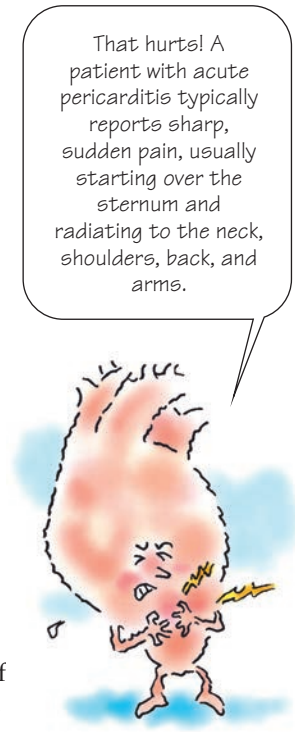
Here's what happens in pericarditis:

- Pericardial tissue damaged by bacteria or other substances results in the release of chemical mediators of inflammation (prostaglandins, histamines, bradykinins, and serotonin) into the surrounding tissue, thereby initiating the inflammatory process.
- Friction occurs as the inflamed pericardial layers rub against each other.
- Histamines and other chemical mediators dilate vessels and increase vessel permeability. Vessel walls then leak fluids and protein (including fibrinogen) into tissues, causing extracellular edema.
- Macrophages already present in the tissue begin to phagocytize the invading bacteria and are joined by neutrophils and monocytes.
- After several days, the area fills with an exudate composed of necrotic tissue and dead and dying bacteria, neutrophils, and macrophages.
- Eventually, the contents of the cavity autolyze and are gradually reabsorbed into healthy tissue.
- Pericardial effusion develops if fluid accumulates in the pericardial cavity.
- Cardiac tamponade results when there's a rapid accumulation of fluid in the pericardial space, compressing the heart and preventing it from filling during diastole, and resulting in a drop in cardiac output.
- Chronic constrictive pericarditis develops if the pericardium becomes thick and stiff from chronic or recurrent pericarditis, encasing the heart in a stiff shell and preventing the heart from properly filling during diastole. This causes an increase in both left- and right-sided filling pressures, leading to a drop in stroke volume and cardiac output.



What to look for

- The patient with acute pericarditis typically complains of sharp, sudden pain, usually starting over the sternum and radiating to the neck, shoulders, back, and arms. The pain is usually pleuritic, increasing with deep inspiration and decreasing when the patient sits up and leans forward. This decrease occurs because leaning forward pulls the heart away from the diaphragmatic pleurae of the lungs. A pericardial friction rub may be heard over the left lateral sternal border.



Cardiac complications

- Pericardial effusion, the major complication of acute pericarditis, may produce effects of heart failure, such as dyspnea, orthopnea, and tachycardia. It may also produce ill-defined substernal chest pain and a feeling of chest fullness.
- If fluid accumulates rapidly, cardiac tamponade may occur, causing pallor, clammy skin, hypotension, pulsus paradoxus, 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, including fluid retention, ascites, and hepatomegaly.

What tests tell you

These tests are used to diagnose pericarditis:

- ECG may reveal diffuse ST-segment elevation in the limb leads and most precordial leads that reflect the inflammatory process. Upright T waves are present in most leads. QRS segments may be diminished when pericardial effusion exists. Arrhythmias, such as atrial fibrillation and sinus arrhythmias, may occur. In chronic constrictive pericarditis, there may be low-voltage QRS complexes, T-wave inversion or flattening, and P mitral waves (wide P waves) in leads I, II, and V_6.
- Laboratory testing may reveal an elevated erythrocyte sedimentation rate as a result of the inflammatory process or a normal or elevated white blood cell (WBC) count, especially in infectious pericarditis; BUN may point to uremia as a cause of pericarditis. CRP levels may be elevated, indicating inflammation.
- Blood cultures may be used to identify an infectious cause.
- Antistreptolysin-O titers may be positive if pericarditis is due to rheumatic fever.
- Purified protein derivative skin test may be positive if pericarditis is due to tuberculosis.

- Echocardiography may show an echo-free space between the ventricular wall and the pericardium and reduced pumping action of the heart. It may also help identify if a pleural effusion is present. It may also help identify a pleural effusion.
- Chest X-rays may be normal with acute pericarditis. The cardiac silhouette may be enlarged, with a water bottle shape caused by fluid accumulation, if pleural effusion is present.

How it's treated

Treatment for a patient with pericarditis is done to:

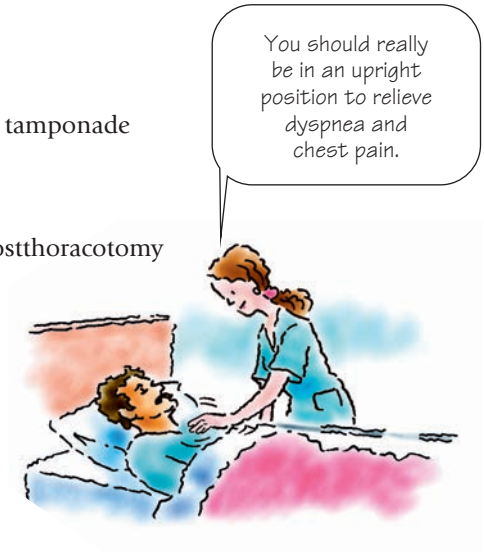
- relieve symptoms
- prevent or correct pericardial effusion and cardiac tamponade
- manage the underlying disease.

Bed rest and drug therapy

In idiopathic pericarditis, post MI pericarditis, and postthoracotomy pericarditis, treatment is twofold, including:

- bed rest as long as fever and pain persist
- administration of NSAIDs to relieve pain and reduce inflammation.

If symptoms continue, the practitioner may prescribe corticosteroids to provide rapid and effective relief. Corticosteroids must be used cautiously because pericarditis may recur when drug therapy stops.



Further treatments

When infectious pericarditis results from disease of the left pleural space, mediastinal abscesses, or septicemia, the patient requires antibiotics, surgical drainage, or both.

If cardiac tamponade develops, the doctor may perform emergency pericardiocentesis and may inject antibiotics directly into the pericardial sac.

Heavy-duty treatments

Recurrent pericarditis may necessitate partial pericardiectomy, which creates a window that allows fluid to drain into the pleural space. In constrictive pericarditis, total pericardiectomy may be necessary to permit the heart to fill and contract adequately.

What to do

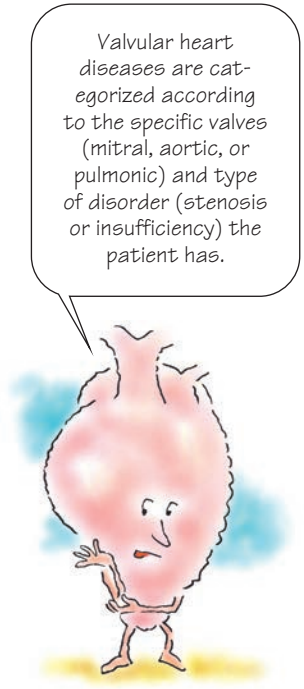
- Maintain the patient on bed rest until fever and pain diminish. Assist the patient with bathing if necessary. Provide a bedside commode to reduce myocardial oxygen demand.

- Place the patient in an upright position to relieve dyspnea and chest pain. Auscultate lung sounds at least every 2 hours. Administer supplemental oxygen as needed based on oxygen saturation or mixed venous oxygen saturation levels.
- Administer analgesics to relieve pain and NSAIDs, as ordered, to reduce inflammation. Administer steroids if the patient fails to respond to NSAIDs.
- If your patient has a PA catheter, monitor hemodynamic status. Assess the patient's cardiovascular status frequently, watching for signs of cardiac tamponade.
- Administer antibiotics on time to maintain consistent drug levels in the blood.
- Institute continuous cardiac monitoring to evaluate for changes in ECG. Look for the return of ST segments to baseline with T-wave flattening by the end of the first 7 days.
- Keep a pericardiocentesis set available if pericardial effusion is suspected and prepare the patient for pericardiocentesis as indicated.
- Provide appropriate postoperative care, similar to that given after cardiothoracic surgery.

Valvular heart disease

In valvular heart disease, three types of mechanical disruption can occur:

1. stenosis, or narrowing, of the valve opening
2. incomplete closure of the valve
3. prolapse of the valve.



What causes it

Valvular heart disease in children and adolescents most commonly results from congenital heart defects. In adults, rheumatic heart disease is a common cause.

Other causes are grouped according to the type of valvular heart disease and include the following:

Mitral insufficiency

- Hypertrophic cardiomyopathy
- Papillary muscle dysfunction
- Left ventricle dilation from left ventricle failure

Mitral stenosis

- Endocarditis
- Left atrium tumors
- Mitral annulus calcification

Aortic insufficiency

- Calcification
- Endocarditis
- Hypertension
- Drugs, especially appetite suppressants

Aortic stenosis

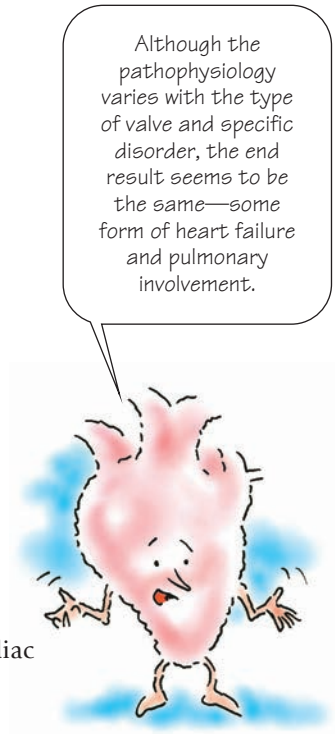
- Calcification

Pulmonic stenosis

- Carcinoid syndrome

How it happens

Valvular heart disease may result from numerous conditions, which vary and are different for each type of valve disorder. Pathophysiology of valvular heart disease varies according to the valve and the disorder.



Mitral insufficiency

In mitral insufficiency, 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 PAP, eventually leading to left-sided and right-sided heart failure.

Mitral stenosis

In mitral stenosis, the valve narrows as a result of valvular abnormalities, fibrosis, or calcification. This obstructs blood flow from the left atrium to the left ventricle. Consequently, left atrial volume and pressure increase 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.

Aortic insufficiency

In aortic insufficiency, blood flows back into the left ventricle during diastole, causing fluid overload in the ventricle which, in turn, 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

In aortic stenosis, elevated left ventricular pressure tries to overcome the resistance of the narrowed valvular opening. The added workload increases the demand for oxygen, and diminished cardiac output causes poor coronary artery perfusion, ischemia of the left ventricle, and left-sided heart failure.

Pulmonic stenosis

In pulmonic stenosis, obstructed right ventricular outflow causes right ventricular hypertrophy in an attempt to overcome resistance to the narrow valvular opening. The ultimate result is right-sided heart failure.

What to look for

The history and physical examination findings vary according to the type of valvular defects.

Mitral insufficiency

Signs and symptoms of mitral insufficiency include:

- orthopnea
- dyspnea
- fatigue
- angina (rare)
- palpitations
- right-sided heart failure (jugular vein distention, peripheral edema, hepatomegaly)
- systolic murmur
- split S_2, S_3, and S_4 heart sounds.

Mitral stenosis

Signs and symptoms of mitral stenosis include:

- dyspnea on exertion, paroxysmal nocturnal dyspnea, orthopnea
- fatigue, weakness
- right-sided heart failure
- crackles on auscultation
- palpitations
- loud S_1 and S_2
- middiastolic murmur.

Aortic insufficiency

Signs and symptoms of aortic insufficiency include:

- dyspnea
- cough
- left-sided heart failure
- pulsus biferiens (rapidly rising and collapsing pulses)
- blowing diastolic murmur or S_3
- chest pain with exertion
- crackles on auscultation.

Aortic stenosis

Signs and symptoms of aortic stenosis include:

- dyspnea and paroxysmal nocturnal dyspnea
- fatigue
- syncope
- angina
- palpitations and cardiac arrhythmias
- left-sided heart failure
- systolic murmur at the base of the carotids
- chest pain with exertion
- split S_1 and S_2.

Pulmonic stenosis

Although a patient with pulmonic stenosis may be asymptomatic, possible signs and symptoms include:

- dyspnea on exertion
- right-sided heart failure
- systolic murmur.

What tests tell you

The diagnosis of valvular heart disease can be based on the results of:

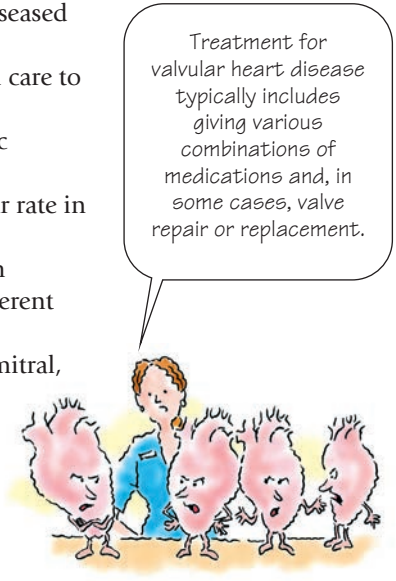
- cardiac catheterization
- chest X-rays
- echocardiography
- ECG.

How it's treated

Treatments for patients with valvular heart disease commonly include:

- digoxin, a low-sodium diet, diuretics, vasodilators, and especially ACE inhibitors to correct left-sided heart failure
- oxygen administration in acute situations to increase oxygenation

- anticoagulants to prevent thrombus formation around diseased or replaced valves
- prophylactic antibiotics before and after surgery or dental care to prevent endocarditis
- nitroglycerin to relieve angina in conditions such as aortic stenosis
- beta-adrenergic blockers or digoxin to slow the ventricular rate in atrial fibrillation or atrial flutter
- cardioversion to convert atrial fibrillation to sinus rhythm
- open or closed commissurotomy to separate thick or adherent mitral valve leaflets
- balloon valvuloplasty to enlarge the orifice of a stenotic mitral, aortic, or pulmonic valve
- annuloplasty or valvuloplasty to reconstruct or repair the valve in mitral insufficiency
- valve replacement with a prosthetic valve for mitral and aortic valve disease.



What to do

- Assess the patient's vital signs, ABG values, pulse oximetry, intake and output, daily weights, blood chemistry studies, chest X-rays, and ECG.
- Place the patient in an upright position to relieve dyspnea if needed. Administer oxygen to prevent tissue hypoxia as needed and indicated by ABGs and pulse oximetry.
- Institute continuous cardiac monitoring to evaluate for arrhythmias; if any occur, administer appropriate therapy according to facility policy and the practitioner's order.
- For a patient with aortic insufficiency, observe the ECG for arrhythmias, which can increase the risk of pulmonary edema, and for fever and infection.
- If the patient has mitral stenosis, watch closely for signs of pulmonary dysfunction caused by pulmonary hypertension, tissue ischemia caused by emboli, and adverse reactions to drug therapy.
- For a patient with mitral insufficiency, observe for signs and symptoms of left-sided heart failure, pulmonary edema, and adverse reactions to drug therapy.

Quick quiz

1. The nurse obtains a rhythm strip on a patient who has had a myocardial infarction and makes the following analysis: P wave not apparent, ventricular rate 170, RR interval not measurable with a wide and distorted QRS complex. The nurse interprets this rhythm as:

A. Sinus bradycardia
B. Junctional escape rhythm
C. Atrial fibrillation
D. Ventricular tachycardia

Answer: D. The key variables when evaluating this rhythm is that there are no P waves present, and the QRS complex is wide and distorted.

2. A patient with dilated cardiomyopathy has developed atrial fibrillation that is unresponsive to drug therapy. The nurse anticipates the patient may need teaching about:

A. Cardiac catheterization
B. Insertion of an implantable cardioverter-defibrillator
C. Electrical cardioversion
D. Lifestyle modifications

Answer: C. After assessing for blood clots, there is a potential the patient could be a candidate for electrical cardioversion. The patient will also need to be anticoagulated with Coumadin to ensure no complications of emboli arise from the arterial fibrillation.

3. Which action should the nurse take first when preparing a patient for cardioversion with stable supraventricular tachycardia who is alert and oriented?

A. Turn the synchronizer to the "off" position.
B. Start a peripheral I.V. and ensure patency.
C. Set the defibrillator to 300 joules.
D. Place the patient on 100% nonrebreather.

Answer: B. A peripheral I.V. is needed in case the cardioversion puts the patient in a potential lethal dysrhythmia. It will also be needed to administer sedative medications.

4. Which parameter is often measured in right-sided heart failure to ensure appropriate fluid volume status within the patient?

A. CVP
B. Left-ventricular end-diastolic pressure
C. PAWP
D. Cardiac output

Answer: A. CVP is elevated in right-sided heart failure and is directly related to preload.

5. A patient whose cardiac monitor shows sinus bradycardia at the rate of 55 beats/minute; upon assessment, the nurse finds the patient is apneic with no palpable pulses. What should the nurse do first?

A. Defibrillate.
B. Give 100% oxygen per nonrebreather.
C. Administer epinephrine.
D. Start CPR and initiate a code blue.

Answer: D. The patient is in pulseless electrical activity and will need immediate lifesaving interventions. The nurse will need to start CPR and call a code blue.

Scoring

☆☆☆ If you answered all five questions correctly, you're all heart! (You'd have to be to make it through this cardiovascular workout!)

☆☆ If you answered four questions correctly, take heart. You have all the blood and gumption you need to succeed.

☆ If you answered fewer than four questions correctly, have yourself a heart-to-heart, then try again. You'll do better next time.

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