Cardiac emergencies

Just the facts

In this chapter, you'll learn:
- emergency assessment of the cardiovascular system
- diagnostic tests and procedures for cardiovascular emergencies
- cardiovascular disorders in the emergency department and their treatments.

Understanding cardiac emergencies

The cardiovascular system is a major control system in the body, playing a key role in cellular nutrition and circulation. It's responsible for carrying life-sustaining oxygen and nutrients via the blood to all cells of the body. When faced with an emergency involving the cardiovascular system, you must assess the patient thoroughly, always being alert for subtle changes that might indicate a potential deterioration in the patient's condition. A thorough nursing assessment forms the basis for your interventions, which must be instituted quickly to minimize potentially life-threatening risks to the patient.

Assessment

Assessment of a patient's cardiovascular system includes a health history and physical examination. If you can't interview the patient because of his condition, you may gather history information from the patient's family members, the patient's primary nurse or other health care providers, or the emergency medical response team.
Health history

To obtain the health history of a patient’s cardiovascular system, begin by introducing yourself and then obtain information on the patient’s chief complaint, personal and family health, and chest pain, if any.

Chief complaint

Use the seven attributes of a symptom, listed below, to obtain details about the patient’s chief complaint:
- location (Where is it? Does it radiate?)
- quality (What’s it like?)
- quantity or severity (How bad is it on a 1-to-10 scale?)
- timing (When does it start? How long does it last? How often does it occur?)
- setting or environmental factors (including personal activities and contributing factors, such as climbing stairs and exercising)
- factors that make it better or worse
- associated manifestations.

Personal and family health

Ask the patient for details about his family history and past medical history. Also ask about:
- current health habits, such as smoking, alcohol intake, caffeine intake, exercise, and dietary intake of fat and sodium
- stressors in the patient’s life and coping strategies he uses to deal with them
- environmental or occupational factors
- activities of daily living
- drugs the patient is taking, including prescription drugs, over-the-counter drugs, and herbal preparations
- menopause (if applicable)
- previous surgeries.

Complaints of chest pain

Many patients with cardiovascular problems complain of chest pain. Use the seven attributes of a symptom to get a complete picture of the patient’s pain.
Cardiac questions

To thoroughly assess your patient’s cardiac function, be sure to ask these questions:
• Are you in pain?
• Where’s 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?

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.)

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 precipitating, exacerbating, or relieving factors to obtain an accurate description of chest pain. (See Differentiating chest pain, page 100.)

Physical examination

Cardiac emergencies affect people of all ages, ethnicities, and cultures and can take many forms. To best identify abnormalities, use a consistent, methodical approach to the physical examination. Because of the emergency nature of the patient’s condition, remember that you may need to limit your examination to specific problem areas or stop your examination entirely to intervene if the patient exhibits signs or symptoms that his condition is deteriorating. If your initial screening indicates a cardiac problem, you may need to conduct a more detailed assessment.

The heart of it

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

- inspection
- palpation
### Stay on the ball

#### Differentiating chest pain

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

<table>
<thead>
<tr>
<th>What it feels like</th>
<th>Where it's located</th>
<th>What makes it worse</th>
<th>What causes it</th>
<th>What makes it better</th>
</tr>
</thead>
</table>
| 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; lasts 1/2 hour to 2 hours | Typically across chest but may radiate to jaw, neck, arms, or back | Exertion, anxiety | Acute myocardial infarction | Nitroglycerin and opioid analgesics such as morphine |
| Sharp and continuous; may be accompanied by friction rub; sudden onset | Substernal; may radiate to neck or left arm | Deep breathing, 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, severe pain; sometimes accompanied by dyspnea, increased pulse rate, decreased breath sounds, or deviated trachea | Lateral thorax | Normal respiration | Pneumothorax | Analgesics, chest tube insertion |
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.

Check the chest
Next, inspect the chest. Note landmarks you can use to describe your findings as well as structures underlying the chest wall. Look for pulsations, symmetry of movement, retractions, or heaves (strong outward thrusts of the chest wall that display during systole).

Arms and legs, too
Also inspect the patient's arms or legs, noting color, hair distribution, and lesions, ulcers, or edema.

Light the way
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 location 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 indicates how well the left ventricle is working because it corresponds to the apex of the heart. To find the apical impulse in a woman with large breasts, displace the breasts during the examination.

Neck next
Continue your inspection by observing the vessels in the neck. Note the carotid artery pulsations, which should be brisk and localized and don't decrease when the patient is upright, when he inhales, or when palpated. Also inspect the jugular veins. The internal jugular vein has a softer, undulating pulsation, which changes in response to position, breathing, and palpation.
Then go for the jugular

Check the jugular venous pulse by having 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. Normally, the highest pulsation takes place no more than $1\frac{3}{4}$" (3.8 cm) above the sternal notch. If pulsations appear higher, it indicates elevation in central venous pressure (CVP) and jugular vein distention.

Abnormal findings

Here are some of the abnormal findings you may note on inspection and what such findings tell you:
- Cyanosis, pallor, or cool or cold skin 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.
- 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.
- 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 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) or the use of accessory muscles to breathe typically result from a respiratory disorder, but a congenital heart defect or heart failure may also cause them.

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 Palpating the apical impulse.)

Palpate the potentials

Also palpate the sternoclavicular, aortic, pulmonic, tricuspid, and epigastric areas for abnormal pulsations. Pulsations aren't usually felt in these 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.
Palpating the 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 1/4” to 3/4” (1.5 to 2 cm) in diameter.

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.

Refill, please

Check capillary refill and time 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.” If you are unable to obtain capillary refill and time because of patient injuries or disease, firmly press in the sternal area and assess for blanching in 3 seconds.

And compare

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.

Regular and equal

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, commonly found 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, possibly indicating 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

What a thrill!

• 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 myocardial infarction (MI), or another disorder.

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.
Heart sound sites

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

Erb and friends

First, identify the auscultation sites, including 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.)

Auscultate for heart sounds with the patient in three positions:
- lying on his back with the head of the bed raised 30 to 45 degrees
- sitting up
- 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 di-
rection. Be sure to listen over the entire precordium, not just over the valves. Note the patient's heart rate and rhythm.

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 closed. The closure produces the first heart sound, S₁.
- At the end of ventricular contraction, the aortic and pulmonic valves snap shut. This snap produces the second heart sound, S₂.
- Always identify S₁ and S₂, and then listen for adventitious sounds, such as the third and fourth heart sounds (S₃ and S₄).
- Also listen for murmurs (vibrating, blowing, or rumbling sounds) and rubs (harsh, scratchy, scraping, or squeaking sounds).

Listen for the "dub"

Start auscultating at the aortic area where the S₂ is loudest. An S₂ 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." Its sound is shorter, higher-pitched, and louder than S₁. When the pulmonic valve closes later than the aortic valve during inspiration, you hear a split S₂.

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 S₁ is the loudest.

An S₁ is best heard at the apex of the heart. It results from closure of the mitral and tricuspid valves and is generally described as sounding like "lub." It's low-pitched and dull. An S₁ occurs at the beginning of ventricular systole. It may be split if the mitral valve closes just before the tricuspid valve.

Major auscultation, man!

Also auscultate the major arteries, such as the carotid, femoral, and popliteal arteries, using the bell of the stethoscope to assess for bruits.

Abnormal findings

On auscultation, you may detect S₁ and S₂ heart sounds that are accentuated, diminished, or inaudible. Other abnormal heart sounds—such as S₃, S₄, and murmurs—may result from pressure
changes, valvular dysfunctions, and conduction defects. (See Interpreting abnormal heart sounds.)

**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.

**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 sounds during early diastole to mid-diastole, 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.

**Fourth heart sound**
The fourth heart sound, or $S_4$, is abnormal and occurs late in diastole, just before the pulse upstroke. It immediately precedes the $S_1$ of the next cycle. Known as the atrial or presystolic gallop, 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 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
- anemia
- angina
- aortic stenosis
- cardiomyopathy
- coronary artery disease (CAD)
- elevated left ventricular pressure
- hypertension.

If the $S_4$ sound persists, it may indicate impaired ventricular compliance or volume overload.

**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.
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.

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

If you detect a murmur, identify where it’s loudest, pinpoint when it sounds during the cardiac cycle, and describe its pitch, pattern, quality, and intensity. (See Identifying heart murmurs.)

Location, location, and... timing

Murmurs can start in any cardiac auscultatory site and may radiate from one site to another. To identify the radiation area, auscultate from the site where the murmur seems loudest to the farthest site where it’s still heard. Note the anatomic landmark of this farthest site.

Pinpoint its presence

Determine whether the murmur happens during systole (between S₁ and S₂) or diastole (between S₂ and the next S₁). Then pinpoint when in the cardiac cycle the murmur takes place—for example, during mïddiastole 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 mur-
murmur. Occasionally, murmurs run through both portions of the cycle (continuous murmur).

**Pitch**

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

**Pattern**

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

- grade I—extremely faint; barely audible even to the trained ear
- grade II—soft and low; easily audible to the trained ear
- grade III—moderately loud; about equal to the intensity of normal heart sounds
- grade IV—loud with a palpable thrill at the murmur site
- grade V—very loud with a palpable thrill; audible with the stethoscope in partial contact with the chest
- 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.
Rubbed the wrong way

Listen for a harsh, scratchy, scraping, or squeaking sound throughout systole, diastole, or both. To enhance the sound, have the patient sit upright and lean forward or exhale. A rub usually indicates pericarditis.

Bruit

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

Bothersome bruits

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 cardiac emergencies. For example, in some patients, echocardiography—a noninvasive, risk-free test—can provide as much diagnostic information on valvular heart disease as cardiac catheterization, an invasive, high-risk test.

Cardiac monitoring

Cardiac monitoring is a form of electrocardiography (ECG) that enables continuous observation of the heart’s electrical activity. It’s an essential assessment tool in the emergency department (ED) and is used to continually monitor the patient’s cardiac status to enable rapid identification and treatment of abnormalities in rate, rhythm, or conduction.

A test with 12 views

The 12-lead ECG measures the heart’s electrical activity and records it as waveforms. It’s one of the most valuable and commonly used diagnostic tools; however, it isn’t 100% diagnostic and is used in conjunction with other tests. 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. (See *Precordial lead placement*, page 112.)

Up, down, and across...

Scanning up, down, and across the heart, each lead transmits information about a different area. The waveforms obtained from each lead vary depending on the location of the lead in relation to the wave of electrical stimulus, or depolarization, passing through the myocardium.

...from top to bottom...

The six limb leads record electrical activity in the heart's frontal plane. This plane is a view through the middle of the heart from top to bottom. Electrical activity is recorded from the anterior to the posterior axis.

...and, finally, horizontal

The six precordial leads provide information on electrical activity in the heart's horizontal plane, a transverse view through the middle of the heart, dividing it into upper and lower portions. Electrical activity is recorded from a superior or an inferior approach.

**Practice pointers**

- Use a systematic approach to interpret the ECG recording. Compare the patient's previous ECG with the current one, if available, to help identify changes.
- 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.
- 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
Precordial lead placement

To record a 12-lead ECG, place electrodes on the patient's arms and left leg and place a ground lead on the patient's right leg. The three standard limb leads (I, II, and III) and the three augmented leads (aVf, aVL, and aVF) are recorded using these electrodes. Then, to record the precordial chest leads, place electrodes as follows:

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

Right precordial lead placement

Right precordial leads can provide specific information about the function of the right ventricle. Place the six leads on the right side of the chest in a mirror image of the standard precordial lead placement, as follows:

- $V_{1R}$: fourth ICS, left sternal border
- $V_{2R}$: fourth ICS, right sternal border
- $V_{3R}$: halfway between $V_{3R}$ and $V_{4R}$
- $V_{5R}$: fifth ICS, right midclavicular line
- $V_{5R}$: fifth ICS, right anterior axillary line
- $V_{6R}$: fifth ICS, right midaxillary line.

Posterior lead placement

Posterior leads can be used to assess the posterior side of the heart. To ensure an accurate reading, make sure the posterior electrodes $V_7$, $V_8$, and $V_9$ are placed at the same horizontal level as the $V_5$ lead at the fifth intercostal space. Place lead $V_7$ at the posterior axillary line, lead $V_5$ at the paraspinal line, and lead $V_9$ halfway between leads $V_7$ and $V_9$. 

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alarm. Excessively tall, flat, or inverted T waves accompanying such symptoms as chest pain may indicate ischemia.
• A normal Q wave generally has a duration of less than 0.04 second. An abnormal Q wave has 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 aVR normally has a large Q wave, so disregard this lead when searching for abnormal Q waves.

Cardiac marker studies

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

Release those 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 monitor the progress of healing.

Heart enzymes

Cardiac enzymes include creatine kinase (CK), CK's isoenzyme MB (found specifically in heart muscle), lactate dehydrogenase (LD), and LD's isoenzymes LD₁ and LD₂ that are found in heart muscle. (See Release of cardiac enzymes and proteins, page 114.)

Troponin T and I and myoglobin are more specific tests of cardiac muscle and can be used to detect damage more quickly, allowing faster and more effective treatment.

Practice pointers

• Before CK measurement, make certain that the patient hasn’t ingested alcohol, aminocaproic acid, or lithium. If the patient has recently taken these substances, note this on the laboratory request.
• 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
Release of cardiac enzymes and proteins

Because they’re released by damaged tissue, serum proteins and isoenzymes (catalytic proteins that vary in concentration in specific organs) can help identify the compromised organ and assess the extent of damage. After an acute myocardial infarction (MI), cardiac enzymes and proteins rise and fall in characteristic patterns, as shown in the graph.

**What’s it all mean?**

Here’s what the results of cardiac marker studies indicate:
- **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.
- **LD₁** and **LD₂** levels increase 8 to 12 hours after acute MI, peak in 24 to 48 hours, and return to normal in 10 to 14 days, if tissue necrosis doesn’t persist.
- **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.
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 echocardiography techniques are motion mode (M-mode) and two-dimensional.

**Motion mode**

In *M-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. Patients who come to the ED with atrial fibrillation who require an echocardiogram may have mildly distorted images because of the rapid motion of the heart.

**TEE combination**

In *transesophageal echocardiography (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).

**Practice pointers**

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

Hemodynamic monitoring is an invasive procedure used to assess cardiac function and determine the effectiveness of therapy by measuring:
- blood pressure
- cardiac output
- intracardiac pressures
- mixed oxygen saturation. (See Putting hemodynamic monitoring to use.)

Getting involved

Hemodynamic monitoring involves insertion of a catheter into the vascular system. The types of hemodynamic monitoring include:
- arterial blood pressure monitoring
- pulmonary artery pressure monitoring (PAP), using the internal and external jugular and subclavian veins. (Femoral and antecubital veins may be used but aren’t the sites of choice.)

Controversial contraindications

As an invasive procedure, hemodynamic monitoring remains controversial in some EDs because of the risks involved, including sepsis, pneumothorax, air embolism, and pulmonary artery infarction.

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.

Pulmonary artery pressure monitoring

Continuous PAP and intermittent pulmonary artery wedge pressure (PAWP) measurements provide important information about left ventricular function and preload. Use this information for monitoring, 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
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.

<table>
<thead>
<tr>
<th>Pressure and description</th>
<th>Normal values</th>
<th>Causes of increased pressure</th>
<th>Causes of decreased pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Central venous pressure or right atrial pressure</strong>&lt;br&gt;The central venous pressure or right atrial pressure shows right ventricular function and end-diastolic pressure.</td>
<td>Normal mean pressure ranges from 1 to 6 mm Hg (1.34 to 8 cm H$_2$O).</td>
<td>• Right-sided heart failure&lt;br&gt;• Volume overload&lt;br&gt;• Tricuspid valve stenosis or insufficiency&lt;br&gt;• Constrictive pericarditis&lt;br&gt;• Pulmonary hypertension&lt;br&gt;• Cardiac tamponade&lt;br&gt;• Right ventricular infarction</td>
<td>• Reduced circulating blood volume</td>
</tr>
<tr>
<td><strong>Right ventricular pressure</strong>&lt;br&gt;Typically, the doctor measures right ventricular pressure only when initially inserting a pulmonary artery 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.</td>
<td>Normal systolic pressure ranges from 20 to 30 mm Hg; normal diastolic pressure, from 0 to 5 mm Hg.</td>
<td>• Mitral stenosis or insufficiency&lt;br&gt;• Pulmonary disease&lt;br&gt;• Hypoxemia&lt;br&gt;• Constrictive pericarditis&lt;br&gt;• Chronic heart failure&lt;br&gt;• Atrial and ventricular septal defects&lt;br&gt;• Patent ductus arteriosus</td>
<td>• Reduced circulating blood volume</td>
</tr>
<tr>
<td><strong>Pulmonary artery pressure</strong>&lt;br&gt;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.</td>
<td>Systolic pressure normally ranges from 20 to 30 mm Hg. The mean pressure usually ranges from 10 to 15 mm Hg.</td>
<td>• Left-sided heart failure&lt;br&gt;• Increased pulmonary blood flow (left or right shunting, as in atrial or ventricular septal defects)&lt;br&gt;• Any condition causing increased pulmonary arteriolar resistance (such as pulmonary hypertension, volume overload, mitral stenosis, or hypoxia)</td>
<td>• Reduced circulating blood volume</td>
</tr>
<tr>
<td><strong>Pulmonary artery wedge pressure</strong>&lt;br&gt;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.</td>
<td>The mean pressure normally ranges from 6 to 12 mm Hg.</td>
<td>• Left-sided heart failure&lt;br&gt;• Mitral stenosis or insufficiency&lt;br&gt;• Pericardial tamponade</td>
<td>• Reduced circulating blood volume</td>
</tr>
</tbody>
</table>
are receiving multiple or frequently administered cardioactive drugs
are experiencing shock, trauma, pulmonary or cardiac disease, or multiple organ dysfunction syndrome.

PAP’s parts

A pulmonary artery (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.

PAP and PAWP procedures

In PAP or PAWP measurement, the practitioner 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. When the catheter is in the pulmonary artery, PAWP measurement is possible through an opening at the catheter’s tip. The catheter is then deflated and 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.

Practice pointers

Nursing considerations depend on the type of hemodynamic monitoring conducted.

Arterial blood pressure monitoring

- Explain the procedure to the patient and his family, if possible.
- After catheter insertion, observe the pressure waveform to assess arterial pressure.
- Assess the insertion site for signs of infection, such as redness and swelling. Notify the practitioner immediately if you note such signs.
- 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.
PAP monitoring
- 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 prolonged wedging could lead to a pulmonary infarction.
- After each PAWP reading, flush the line per facility policy. If you encounter difficulty, notify the practitioner.
- Maintain 300 mm Hg pressure in the pressure bag to permit a flush flow of 3 to 6 ml/hour.
- Make sure that 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.

Irritation prevention
- 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.
- 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.
- Document the date and time of catheter insertion, the practitioner who performed the procedure, the catheter insertion site, pressure waveforms and values for the various heart chambers, the balloon inflation volume required to obtain a wedge tracing, arrhythmias that took place during or after the procedure, the type of flush solution used and its heparin concentration (if any), the type of dressing applied, and the patient’s tolerance of the procedure.

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/minute. The most widely used method for monitoring cardiac output is the bolus thermodilution technique. Other methods include the Fick method and the dye dilution test.

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 the patient's status.

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.

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 ranges from 2.5 to 4.2 L/minute/m² for adults or 3.5 to 6.5 L/minute/m² for pregnant women.

Physiologic changes can affect the cardiac output and the cardiac index; they include:
- decreased preload
- increased preload
- vasoconstriction (changes in afterload)
- vasodilation (changes in afterload)
- hypothermia.

Practice pointers
- 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 cardiac emergencies. 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 adrenergics, adrenergic blockers, antianginals, antiarrhythmics, anticoagulants, antihypertensives, cardiac glycosides and phosphodiesterase (PDE) inhibitors, diuretics, and thrombolytics.

Adrenergics

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

Classified by chemical

Adrenergic drugs are classified in two groups based on their chemical structure—catecholamines (naturally occurring and synthetic) and noncatecholamines. (See Understanding adrenergics, page 122.)

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 norepinephrine and epinephrine

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. They 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 and adverse reactions associated with these drugs.

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Indications</th>
<th>Adverse reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Catecholamines</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dobutamine</td>
<td>• Increase cardiac output in short-term treatment of cardiac decompensation from depressed contractility</td>
<td>• Headache</td>
</tr>
<tr>
<td>(Dobutrex)</td>
<td></td>
<td>• Tachycardia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Cardiac arrhythmias (premature ventricular contractions)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Hypertension</td>
</tr>
<tr>
<td>dopamine</td>
<td>• Adjunct in shock to increase cardiac output, blood pressure, and urine flow</td>
<td>• Dyspnea</td>
</tr>
<tr>
<td>epinephrine</td>
<td>• Anaphylaxis</td>
<td>• Bradycardia</td>
</tr>
<tr>
<td></td>
<td>• Bronchospasm</td>
<td>• Palpitations</td>
</tr>
<tr>
<td></td>
<td>• Hypersensitivity reactions</td>
<td>• Tachycardia</td>
</tr>
<tr>
<td></td>
<td>• Restoration of cardiac rhythm in cardiac arrest</td>
<td>• Cardiac arrhythmias (ventricular)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Widened QRS</td>
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<td></td>
<td></td>
<td>• Angina</td>
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<tr>
<td>norepinephrine (Levophed)</td>
<td>• GI bleeding</td>
<td>• Restlessness</td>
</tr>
<tr>
<td></td>
<td>• Maintain blood pressure in acute hypotensive states</td>
<td>• Anxiety</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Headache</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Tachycardia</td>
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<tr>
<td></td>
<td></td>
<td>• Palpitations</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Cardiac arrhythmias (ventricular fibrillation)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Precordial pain (in patients with ischemic heart disease)</td>
</tr>
<tr>
<td><strong>Noncatecholamines</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ephedrine</td>
<td>• Maintain blood pressure in acute hypotensive states, especially with spinal anesthesia</td>
<td>• Restlessness</td>
</tr>
<tr>
<td></td>
<td>• Treatment of orthostatic hypotension and bronchospasm</td>
<td>• Anxiety</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Dizziness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Headache</td>
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<tr>
<td></td>
<td></td>
<td>• Cardiac arrhythmias (ventricular fibrillation)</td>
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<tr>
<td></td>
<td></td>
<td>• Nausea</td>
</tr>
</tbody>
</table>
### Understanding adrenergics (continued)

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Indications</th>
<th>Adverse reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Noncatecholamines</td>
<td></td>
<td></td>
</tr>
<tr>
<td>phenylephrine (Neo-Synephrine)</td>
<td>• Maintain blood pressure in hypotensive states, especially hypotensive emergencies with spinal anesthesia</td>
<td>• Restlessness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Anxiety</td>
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<tr>
<td></td>
<td></td>
<td>• Dizziness</td>
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<td></td>
<td></td>
<td>• Headache</td>
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<td></td>
<td></td>
<td>• Palpitations</td>
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<tr>
<td></td>
<td></td>
<td>• Cardiac arrhythmias</td>
</tr>
</tbody>
</table>

### 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 heart rate, and dilate the bronchi. They can be manufactured in the body or in a laboratory.

### Excitatory or inhibitory

Catecholamines primarily act directly. When catecholamines combine with alpha- or beta-receptors, they cause 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. The heart beats faster because catecholamines increase the depolarization rate of pacemaker cells in the sinoatrial (SA) node of the heart. 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 premature ventricular contractions 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 (Vasoxyl) and phenylephrine. Those that selectively exert beta₂ activity include:
• albuterol (Proventil)
• isoetharine
• metaproterenol (Alupent).
Ephedrine is a dual-acting noncatecholamine that combines both actions.

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

Impending impulses
Adrenergic blockers 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
# 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 and adverse reactions associated with these drugs.

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Indications</th>
<th>Adverse reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Alpha-adrenergic blockers</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>phentolamine and prazosin (Minipress)</td>
<td>• Hypertension</td>
<td>• Orthostatic hypotension</td>
</tr>
<tr>
<td></td>
<td>• Peripheral vascular disorders</td>
<td>• Severe hypertension</td>
</tr>
<tr>
<td></td>
<td>• Pheochromocytoma</td>
<td>• Bradycardia</td>
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<tr>
<td></td>
<td></td>
<td>• Tachycardia</td>
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<tr>
<td></td>
<td></td>
<td>• Edema</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Difficulty breathing</td>
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<tr>
<td></td>
<td></td>
<td>• Light-headedness</td>
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<td></td>
<td></td>
<td>• Flushing</td>
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<td></td>
<td></td>
<td>• Arrhythmias</td>
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<td></td>
<td></td>
<td>• Angina</td>
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<tr>
<td></td>
<td></td>
<td>• Heart attack</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Shock</td>
</tr>
<tr>
<td><strong>Beta-adrenergic blockers</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Nonselective</strong></td>
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<td></td>
</tr>
<tr>
<td>carvedilol (Coreg), labetalol (Normodyne), nadolol (Corgard), penbutolol (Levatol), pindolol (Visken), propranolol (Inderal), sotalol (Betapace), and timolol (Blocadren)</td>
<td>• Prevention of complications after myocardial infarction, angina, hypertension, supraventricular arrhythmias, anxiety, essential tremor, cardiovascular symptoms associated with thyrotoxicosis, migraine headaches, pheochromocytoma</td>
<td>• Hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Bradycardia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Peripheral vascular insufficiency</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Heart failure</td>
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<tr>
<td></td>
<td></td>
<td>• Bronchospasm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Sore throat</td>
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<tr>
<td></td>
<td></td>
<td>• Atrialventricular block</td>
</tr>
</tbody>
</table>

- preventing the action of cholinergic drugs.

### Classified information

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

Alpha-adrenergic blockers work by interrupting the actions of sympathomimetic drugs at alpha-adrenergic receptors. This interruption 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
- 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:

- They interfere with or block the synthesis, storage, release, and reuptake of norepinephrine by neurons.
- They antagonize epinephrine, norepinephrine, or adrenergic (sympathomimetic) drugs at alpha receptor sites.

Not very discriminating

Alpha receptor sites are either alpha\textsubscript{1} or alpha\textsubscript{2} receptors. Alpha-adrenergic blockers include drugs that block stimulation of alpha\textsubscript{1} receptors and that may block alpha\textsubscript{2} stimulation.

Reducing resistance

Alpha-adrenergic blockers occupy alpha receptor sites on the smooth muscle of blood vessels, which 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\textsubscript{1}-receptor sites (located mainly in the heart)
- beta₂-receptor sites (located in the bronchi, blood vessels, and uterus).

Nonselective beta-adrenergic drugs include carvedilol, timolol, nadolol, penbutolol, labetalol, pindolol, sotalol, and propranolol.

**Highly discriminating**

Selective beta-adrenergic drugs primarily affect the beta₁-adrenergic sites. They include atenolol, esmolol, acebutolol, and metoprolol.

**Intrinsically sympathetic**

Some beta-adrenergic blockers, such as pindolol and acebutolol, have intrinsic sympathetic activity. This sympathetic activity 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 beta₁ receptor sites, reduce stimulation of the heart. They're commonly called cardioselective beta-adrenergic blockers.

Nonselective beta-adrenergic blockers, which block beta₁ and beta₂ receptor sites, reduce stimulation of the heart and cause the bronchioles of the lungs to constrict. This constriction causes bronchospasm in patients with chronic obstructive lung disorders.

**Antianginals**

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.

Anti-angina effect

Nitrates cause the smooth muscle of the veins and, to a lesser extent, the arteries to relax and dilate. Here’s what happens:
- When the veins dilate, less blood returns to the heart.
- Decreased blood return 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 as much to pump blood. This reduction in size and tension 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.
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 and adverse reactions associated with these drugs.

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Indications</th>
<th>Adverse reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nitrate</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>isosorbide dinitrate</td>
<td>• Relief and prevention of angina</td>
<td>• Dizziness</td>
</tr>
<tr>
<td>(Isordil), isosorbide</td>
<td></td>
<td>• Headache</td>
</tr>
<tr>
<td>mononitrate (Imdur),</td>
<td></td>
<td>• Hypotension</td>
</tr>
<tr>
<td>and nitroglycerin</td>
<td></td>
<td>• Increased heart rate</td>
</tr>
<tr>
<td>(Nitro-Bid)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Beta-adrenergic blockers</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>atenolol (Tenormin),</td>
<td>• First-line therapy for hypertension</td>
<td>• Angina</td>
</tr>
<tr>
<td>metoprolol (Lopressor),</td>
<td>• Long-term prevention of angina</td>
<td>• Arrhythmias</td>
</tr>
<tr>
<td>nadolol (Corgard),</td>
<td></td>
<td>• Bradycardia</td>
</tr>
<tr>
<td>and propranolol</td>
<td></td>
<td>• Bronchial constriction</td>
</tr>
<tr>
<td>(Inderal)</td>
<td></td>
<td>• Diarrhea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Fainting</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Fluid retention</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Heart failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Nausea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Shock</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Vomiting</td>
</tr>
<tr>
<td><strong>Calcium channel blockers</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>amlodipine (Norvasc),</td>
<td>• Long-term prevention of angina (especially</td>
<td>• Arrhythmias</td>
</tr>
<tr>
<td>diltiazem (Cardizem),</td>
<td>Prinzmetal's angina)</td>
<td>• Dizziness</td>
</tr>
<tr>
<td>nicardipine (Cardene),</td>
<td></td>
<td>• Flushing</td>
</tr>
<tr>
<td>nifedipine (Procardia),</td>
<td></td>
<td>• Headache</td>
</tr>
<tr>
<td>and verapamil (Calan)</td>
<td></td>
<td>• Heart failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Orthostatic hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Persistent peripheral edema</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Weakness</td>
</tr>
</tbody>
</table>

**Down with everything**

Beta-adrenergic blockers decrease blood pressure and block beta-adrenergic receptor sites in the heart muscle and conduction system. These actions decrease the heart rate and reduce 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 nitrates or beta-adrenergic blockers. Some calcium channel blockers are also used as antiarrhythmics.

**Preventing passage**
Calcium channel blockers prevent the passage of calcium ions across the myocardial cell membrane and vascular smooth-muscle cells, causing dilation of the coronary and peripheral arteries. This dilation in turn 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.

**Conduction reduction**
Calcium channel blockers also reduce the heart rate by slowing conduction through the SA and atrioventricular (AV) nodes. A slower heart rate reduces the heart’s need for oxygen.

**Antiarrhythmics**
Antiarrhythmics 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 must 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 group is the largest group of antiarrhythmic drugs. Class I agents are commonly subdivided into classes IA, IB, and IC.
Understanding antiarrhythmics

Antiarrhythmics are used to restore normal heart rhythm in patients with arrhythmias. Use this table to learn about the indications and adverse reactions associated with these drugs.

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Indications</th>
<th>Adverse reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Class IA antiarrhythmics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>disopyramide (Norpace), procainamide</td>
<td>• Atrial fibrillation</td>
<td>• Abdominal cramping</td>
</tr>
<tr>
<td>(Procanbid), quinidine sulfate, and quinidine gluconate</td>
<td>• Atrial flutter</td>
<td>• Anorexia</td>
</tr>
<tr>
<td></td>
<td>• Paroxysmal atrial tachycardia</td>
<td>• Bitter taste</td>
</tr>
<tr>
<td></td>
<td>• Ventricular tachycardia</td>
<td>• Diarrhea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Nausea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Vomiting</td>
</tr>
<tr>
<td><strong>Class IB antiarrhythmics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lidocaine (Xylocaine) and mexiletine</td>
<td>• Ventricular tachycardia, ventricular fibrillation</td>
<td>• Bradycardia</td>
</tr>
<tr>
<td>(Mexitil)</td>
<td></td>
<td>• Drowsiness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Light-headedness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Paresthesia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Sensory disturbances</td>
</tr>
<tr>
<td><strong>Class IC antiarrhythmics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>flecainide (Tambocor), moricizine (Ethmozine), and propafenone (Rythmol)</td>
<td>• Ventricular tachycardia, ventricular fibrillation, supraventricular arrhythmias</td>
<td>• Bronchospasm (propafenone)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• New arrhythmias</td>
</tr>
<tr>
<td><strong>Class II antiarrhythmics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>acebutolol (Sectral), esmolol (Brevibloc), and propranolol (Inderal)</td>
<td>• Atrial flutter, atrial fibrillation, paroxysmal atrial tachycardia</td>
<td>• Arrhythmias</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Bradycardia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Bronchoconstriction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Diarrhea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Heart failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Nausea and vomiting</td>
</tr>
<tr>
<td><strong>Class III antiarrhythmics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>amiiodarone (Cordarone)</td>
<td>• Life-threatening arrhythmias resistant to other antiarrhythmics</td>
<td>• Aggravation of arrhythmias</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Anorexia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Severe pulmonary toxicity</td>
</tr>
</tbody>
</table>

(continued)
Understanding antiarrhythmics (continued)

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Indications</th>
<th>Adverse reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Class IV antiarrhythmics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>diltiazem (Cardizem) and verapamil (Calan)</td>
<td>Supraventricular arrhythmias</td>
<td>Atrioventricular block</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bradycardia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dizziness</td>
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<tr>
<td></td>
<td></td>
<td>flushing (with diltiazem)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>peripheral edema</td>
</tr>
<tr>
<td><strong>Miscellaneous antiarrhythmics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>adenosine (Adenocard)</td>
<td>Paroxysmal supraventricular tachycardia</td>
<td>Chest discomfort</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dyspnea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>facial flushing</td>
</tr>
<tr>
<td></td>
<td></td>
<td>shortness of breath</td>
</tr>
</tbody>
</table>

**Class IA antiarrhythmics**

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

**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, a class IB antiarrhythmic, is one of the antiarrhythmics commonly used in treating patients with acute ventricular arrhythmias. Another IB antiarrhythmic is mexiletine.
Class IB drugs work by blocking the rapid influx of sodium ions during the depolarization phase of the heart's depolarization-repolarization cycle. This blocking action results in a decreased refractory period, which reduces the risk of arrhythmia.

Make a IB-line for the ventricle
Because class IB antiarrhythmics especially affect the Purkinje fibers 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, moricizine, and propafenone.

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 decrease 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.

Receptor blockers
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.

Strength reducers
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 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.
**Miscellaneous antiarrhythmics**
The class IV antiarrhythmics include 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 and diltiazem. Miscellaneous antiarrhythmics also include adenosine.

**Adenosine**
Adenosine is an injectable antiarrhythmic drug indicated for acute treatment of 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.

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

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

**Low is good**
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 A2. Dipyridamole may inhibit platelet aggregation.

**Anticlumping**
Clopidogrel inhibits platelet aggregation by blocking adenosine diphosphate receptors on platelets, thereby preventing the clumping of platelets.

**Once does it**
Sulfinpyrazone appears to inhibit several platelet functions. At dosages of 400 to 800 mg/day, it lengthens platelet survival; dosages of more than 600 mg/day prolong the patency of arteriovenous shunts used for hemodialysis. A single dose rapidly inhibits platelet aggregation.
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 and adverse reactions associated with these drugs.

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Indications</th>
<th>Adverse reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Heparins</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| heparin and low-molecular-weight heparins, such as dalteparin (Fragmin) and enoxaparin (Lovenox) | • Deep vein thrombosis  
• Disseminated intravascular coagulation  
• Embolism prophylaxis  
• Prevention of complications after myocardial infarction (MI) | • Bleeding         |
| **Oral anticoagulants**      |                                                                             |                   |
| warfarin (Coumadin)          | • Atrial arrhythmias  
• Deep vein thrombosis prophylaxis  
• Prevention of complications of prosthetic heart valves or diseased mitral valves | • Bleeding (may be severe) |
| **Antiplatelet drugs**       |                                                                             |                   |
| aspirin, dipyridamole (Persatine), sulfinpyrazone (Anturane), ticlopidine (Ticlid), and clopidogrel (Plavix) | • Decreases the risk of death after MI  
• Patients at risk for ischemic events (clopidogrel)  
• Patients with acute coronary syndrome (clopidogrel)  
• Prevention of complications of prosthetic heart valves | • Bleeding  
• GI distress  
• Headache (clopidogrel) |

Broken bindings

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

**Heparin**

Heparin, prepared commercially from animal tissue, is used to prevent clot formation. Low-molecular-weight heparin, such as dalteparin and enoxaparin, prevents deep vein thrombosis (a blood clot in the deep veins, usually of the legs) in surgical patients. Be aware, however, that a patient placed on any form of heparin is at risk for developing heparin-induced thrombocytopenia. While the risk of severe adverse effects is low, you must moni-
tor the patient’s platelet count. A decrease in platelet count is cause for alarm and should be addressed and closely monitored.

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. 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.

**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.

**Antihypertensives**

Antihypertensive drugs act to reduce blood pressure. They’re used to treat patients with hypertension, a disorder characterized by high systolic blood pressure, high diastolic blood pressure, or both.
Know the program

Although treatment for hypertension begins with beta-adrenergic blockers and diuretics, antihypertensives are used if those drugs aren’t effective. Antihypertensive therapy includes the use of sympatholytics (other than beta-adrenergic blockers), vasodilators, angiotensin-converting enzyme (ACE) inhibitors, and angiotensin receptor blockers alone or in combination. (See Understanding antihypertensives, page 138.)

**Sympatholytics**

Sympatholytic drugs include several different types of drugs. However, all of these drugs 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.

**Where and how**

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

- central-acting sympathetic nervous system inhibitors, such as clonidine, guanabenz, guanfacine, and methyldopa
- alpha blockers, such as doxazosin, prazosin, and terazosin
- mixed alpha- and beta-adrenergic blockers such as labetalol
- norepinephrine depletors, such as guanadrel and guanethidine.

**Vasodilators**

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 relaxation prevents the contraction of vascular smooth muscle.

**Direct dial**

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

Hydralazine and minoxidil are usually used to treat patients with resistant or refractory hypertension. Diazoxide and nitroprusside are reserved for use in hypertensive crisis.
### Understanding antihypertensives

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

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Indications</th>
<th>Adverse reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sympatholytic drugs</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Central-acting sympathetic nervous system inhibitors</strong>&lt;br&gt;clonidine (Catapres), guanabenz (Wytensin), guanfacine (Tenex), and methyldopa (Aldomet)</td>
<td>• Hypertension</td>
<td>• Depression&lt;br&gt;• Drowsiness&lt;br&gt;• Edema&lt;br&gt;• Hypotension (alpha blockers)&lt;br&gt;• Vertigo (central-acting drugs)</td>
</tr>
<tr>
<td><strong>Alpha blockers</strong>&lt;br&gt;doxazosin (Cardura), phentolamine, prazosin (Minipress), and terazosin (Hytrin)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mixed alpha- and beta-adrenergic blockers</strong>&lt;br&gt;labetalol (Normodyne)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Norepinephrine depletors</strong>&lt;br&gt;guanadrel (Hylorel) and guanethidine (Ismelin)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Vasodilators</strong></td>
<td>• Used in combination with other drugs to treat moderate to severe hypertension</td>
<td>• Angina&lt;br&gt;• Breast tenderness&lt;br&gt;• Edema&lt;br&gt;• Fatigue&lt;br&gt;• Headache&lt;br&gt;• Palpitations&lt;br&gt;• Rash&lt;br&gt;• Severe pericardial effusion&lt;br&gt;• Tachycardia&lt;br&gt;• Vasoconstriction</td>
</tr>
<tr>
<td>hydralazine (Apresoline), minoxidil (Rogaine), and nitroprusside (Nitropress)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Angiotensin-converting enzyme inhibitors</strong></td>
<td>• Heart failure&lt;br&gt;• Hypertension</td>
<td>• Angioedema&lt;br&gt;• Fatigue&lt;br&gt;• Headache&lt;br&gt;• Increased serum potassium concentrations&lt;br&gt;• Persistent cough</td>
</tr>
<tr>
<td>benazepril (Lotensin), captopril (Capoten), enalapril (Vasotec), fosinopril (Monopril), lisinopril (Zestril), quinapril (Accupril), and ramipril (Altace)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
ACE inhibitors
ACE inhibitors reduce blood pressure by interrupting the renin-angiotensin-aldosterone system.

Without ACE inhibition
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 inhibition
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. Less sodium and water reduces the amount of blood the heart needs to pump, resulting in lower blood pressure.

Angiotensin II receptor antagonists
Unlike ACE inhibitors, which prevent production of angiotensin, angiotensin II receptor antagonists block the action of angiotensin II, a major culprit in the development of hypertension, by attaching to tissue-binding receptor sites.

Cardiac glycosides and PDE inhibitors
Cardiac glycosides and PDE inhibitors increase the force of the heart's contractions. Increasing the force of contractions is known as a positive inotropic effect, so these drugs are also called inotropic agents (effecting the force or energy of muscular contractions). (See Understanding cardiac glycosides and PDE inhibitors, page 140.)

Slower rate
Cardiac glycosides, such as digoxin, also slow the heart rate (called a negative chronotropic effect) and slow electrical impulse conduction through the AV node (called a negative dromotropic effect).
Understanding cardiac glycosides and PDE inhibitors

Cardiac glycosides and phosphodiesterase (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 and adverse reactions associated with these drugs.

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Indications</th>
<th>Adverse reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiac glycoside</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>digoxin (Lanoxin)</td>
<td>- Heart failure, supraventricular arrhythmias</td>
<td>- Digoxin toxicity (abdominal pain, arrhythmias, depression, headache, insomnia, irritability, nausea, vision disturbances)</td>
</tr>
<tr>
<td><strong>PDE inhibitors</strong></td>
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<tr>
<td>inamrinone (Inocor) and milrinone (Primacor)</td>
<td>- Heart failure refractory to digoxin, diuretics, and vasodilators</td>
<td>- Arrhythmias</td>
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<td>- Chest pain</td>
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<td>- Hypokalemia</td>
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<td>- Mild increase in heart rate</td>
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<td>- Nausea</td>
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<td>- Thrombocytopenia</td>
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<td>- Vomiting</td>
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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. By directly relaxing vascular smooth muscle, they also decrease peripheral vascular resistance (afterload) and the amount of blood returning to the heart (preload).

Diuretics

Diuretics are used to promote the excretion of water and electrolytes by the kidneys. By doing so, diuretics play a major role in
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 and adverse reactions associated with these drugs.

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Indications</th>
<th>Adverse reactions</th>
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<tbody>
<tr>
<td><strong>Thiazide and thiazide-like diuretics</strong></td>
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</tbody>
</table>
| bendroflumethiazide (Naturetin), chlorothalidone (Hygroton), hydrochlorothiazide (HydroDIURIL), hydroflumethiazide, and indapamide (Lozol) | • Edema  
• Hypertension | • Hypokalemia  
• Hyponatremia  
• Orthostatic hypotension |
| **Loop diuretics** | | |
| bumetanide (Bumex), ethacrylate sodium (Edecrin sodium), ethacrynic acid (Edecrin), and furosemide (Lasix) | • Edema  
• Heart failure  
• Hypertension | • Dehydration  
• Hyperuricemia  
• Hypocalcemia  
• Hypochloremia  
• Hypokalemia  
• Hypomagnesemia  
• Hyponatremia  
• Orthostatic hypotension |
| **Potassium-sparing diuretics** | | |
| amiloride (Midamor), spironolactone (Aldactone), and triamterene (Dyrenium) | • Cirrhosis  
• Diuretic-induced hypokalemia in patients with heart failure  
• Edema  
• Hypertension  
• Nephrotic syndrome | • Hyperkalemia |

treating hypertension and other cardiovascular conditions. (See *Understanding diuretics.*)

The major diuretics used as cardiovascular drugs include:
- loop diuretics
- potassium-sparing diuretics
- thiazide and thiazide-like diuretics.

**Loop diuretics**

Loop (high-ceiling) diuretics are highly potent drugs.
High potency, big risk

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

In 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.

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.

Thiazide and thiazide-like diuretics

Thiazide and thiazide-like diuretics are sulfonamide derivatives.

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 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 therapy is maintained, cardiac output stabilizes but plasma fluid volume decreases.

Thrombolytics

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 ly-
Understanding thrombolitics

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 and adverse reactions associated with these drugs.

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Indications</th>
<th>Adverse reactions</th>
</tr>
</thead>
</table>
| alteplase (Activase), reteplase (Retavase), and streptokinase (Streptase) | • Acute ischemic stroke  
• Acute myocardial infarction  
• Arterial thrombosis  
• Catheter occlusion  
• Pulmonary embolus | • Allergic reaction  
• Bleeding |

(See *Understanding thrombolitics.*)

Some commonly used thrombolytic drugs include:
• reteplase
• alteplase
• streptokinase.

Surgery

Types of surgery used to treat cardiovascular system disorders include coronary artery bypass graft (CABG), vascular repair, and insertion of a ventricular assist device (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 by the results of cardiac catheterization and patient symptoms.

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. Newer surgical techniques, such as the mini-CABG and direct coronary artery bypass, can re-
duce the risk of cerebral complications 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 heart-lung bypass machine, which increases recovery time but decreases complications.

**Nursing considerations**
When caring for a patient who's undergoing CABG, your major roles include patient instruction and caring for the patient's changing cardiovascular needs:
- Check and record vital signs and hemodynamic parameters frequently, possibly every 5 to 15 minutes depending on the patient's condition.
- Administer medications and titrate according to the patient's response as ordered.
- Monitor ECGs continuously for disturbances in heart rate and rhythm.
- Reinforce the practitioner's explanation of the surgery.
- Explain the complex equipment and procedures used in the critical care unit (CCU) or postanesthesia care unit (PACU).
- Explain to the patient that he'll awaken 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 have in place a nasogastric tube, a chest tube, an indwelling urinary catheter, arterial lines, epicardial pacing wires and, possibly, a PA catheter. Tell him that discomfort is minimal and that the equipment is removed as soon as possible.
- Make sure that the patient or a responsible family member has signed a consent form.
- Assist with PA catheterization and insertion of arterial lines. Some facilities insert PA catheters and arterial lines in the operating room before surgery.

**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.

**Nursing considerations**

- Make sure the patient and his family understand the practitioner'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.
- 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). Check capillary refill time by blanching the fingernail or toenail; normal refill time is less than 3 seconds.
- 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.
- Withhold food according to the surgeon's orders and facility policy.
- If the patient is awaiting surgery for aortic aneurysm repair, be on guard for signs and symptoms of acute dissection or rupture. Notify the practitioner immediately if the patient experiences especially sudden severe pain in the chest, abdomen, or lower back; severe weakness; diaphoresis; tachycardia; or a precipitous drop in blood pressure.

**PTCA**

Percutaneous transluminal coronary angioplasty (PTCA) is a non-surgical 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 caused by angina and myocardial ischemia.

**Through one artery and into another**

After coronary angiography confirms the presence and location of the occlusion, the practitioner 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 practitioner carefully introduces a double-lumen balloon into the catheter and through the lesion, where a marked increase in the pressure gradient is obvious. The practitioner alternately inflates and deflates the balloon until arteriography verifies successful arterial dilation and a decrease in the pressure gradient. With balloon inflation, the plaque is compressed against the vessel wall, allowing coronary blood to flow more freely.

**Nursing considerations**

- Describe the procedure to the patient and his family, and tell them the procedure 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 angina during the procedure.
- Explain that the practitioner 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 practitioner.

**Take two aspirin and call me...**

- Give 650 mg of aspirin before the procedure, as ordered, to prevent platelet aggregation.
- Make sure the patient signs an informed consent form.
- Restrict food and fluids before the procedure.
• Make sure that the results of coagulation studies, complete blood count (CBC), serum electrolyte studies, blood typing and crossmatching, blood urea nitrogen (BUN), and serum creatinine are available.
• Obtain baseline vital signs and assess peripheral pulses; continue to assess the patient's vital signs and oxygen saturation frequently.
• Apply ECG electrodes and insert an I.V. line if not already in place. Monitor I.V. infusions as indicated.
• Administer oxygen through a nasal cannula.
• Give the patient a sedative as ordered.

Other treatments

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

Synchronized cardioversion

Synchronized cardioversion (synchronized countershock) is an elective or emergency procedure used to treat unstable tachyarrhythmias (such as atrial flutter, atrial fibrillation, and supraventricular tachycardia and 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.

The procedure causes immediate depolarization, interrupting reentry circuits (abnormal impulse conduction resulting 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 this elective procedure to the patient and make sure an informed consent is obtained.
• Obtain a baseline 12-lead ECG.
• Withhold food beginning as soon as possible.
Stay on the ball

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 recommended energy doses used for cardioversion are:

- 50, 100 joules for unstable supraventricular tachycardia
- 100, 200 joules (with a monophasic waveform) for atrial fibrillation
- 100, 120 joules (with a biphasic waveform) for unstable atrial flutter.

- Give a sedative as ordered.
- Apply conductive gel to the paddles or 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.
- Turn on the defibrillator and select the ordered energy level, usually between 50 and 100 joules. (See Choosing the correct cardioversion energy level.)
- Activate the synchronized mode by depressing the synchronizer switch.
- Check that the machine is sensing the R wave correctly.
- Place the paddles on the chest and apply firm pressure.
- Charge the paddles.
- Instruct other personnel to stand clear of the patient and the bed to avoid the risk of an electric shock by stating “all clear.”
- Discharge the current by pushing both paddles’ DISCHARGE buttons simultaneously.

Repeat, repeat, and repeat again

- 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.
- When using handheld paddles, continue to hold the paddles on the patient’s chest until the energy is delivered.
In sync

- 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, the amperage used, and how the patient tolerated the procedure.

Defibrillation

In defibrillation, electrode paddles are used to direct an electric current 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.

The electrode paddles delivering the current may be placed on the patient’s chest or, during cardiac surgery, directly on the myocardium.

One or both

Defibrillators can be monophasic or biphasic. Monophasic defibrillators deliver a single current of electricity that travels in one direction between the two pads or paddles on the patient’s chest. A large amount of electrical current is needed for effective monophasic defibrillation.

Positively speaking

A biphasic defibrillator delivers the electrical current 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. 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. For example, instead of using 200 joules, an initial shock of 150 joules is commonly effective.

Adjustable

Additionally, the biphasic defibrillator is able to adjust for differences in impedance or the resistance of the current through the chest, thereby reducing the number of shocks needed to terminate ventricular fibrillation. Also, damage to the myocardial muscle is reduced because of the lower energy levels used and fewer shocks needed.

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.

**Nursing considerations**

- Assess the patient to determine if he lacks a pulse. If so, call for help and perform cardiopulmonary resuscitation (CPR) until the defibrillator and other emergency equipment arrive.
- 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 conductive pads at the paddle placement positions. (See Defibrillator paddle placement.)
- Turn on the defibrillator and, if performing external defibrillation, set the energy level at 360 joules for an adult patient.

**Charge!!**

- Charge the paddles by pressing the charge buttons, which are located on the machine or paddles.
- Place the paddles over the conductive pads and press firmly against the patient’s chest, using 25 lb (11.3 kg) of pressure.
- Reassess the patient’s cardiac rhythm in two leads.
- If the patient remains in ventricular fibrillation or pulseless ventricular tachycardia, 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.

**And discharge!**

- Discharge the current by pressing both paddle discharge buttons simultaneously.
- Reassess the patient’s pulse, and give 2 minutes of CPR. Reassess his cardiac rhythm.
- If necessary, prepare to defibrillate a second time at 360 joules. Announce that you’re preparing to defibrillate, and follow the procedure described above.
- Reassess the patient and continue CPR.
- If the patient still has no pulse after the first two cycles of defibrillation and CPR, give supplemental oxygen, and begin administering appropriate medications such as epinephrine. Also, consider possible causes for failure of the patient’s rhythm to convert, such as acidosis and hypoxia.

**Rhythm restoration**

- If defibrillation restores a normal rhythm, assess the patient. Obtain baseline ABG levels and a 12-lead ECG. Provide supple-
Defibrillator paddle placement

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

Anterolateral placement
For anterolateral placement, place one paddle to the right of the upper sternum, just below the right clavicle. Place the other over the fifth or sixth intercostal space at the left anterior axillary line.

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

- Prepare the patient for possible insertion of an implantable cardioverter-defibrillator.

Transcutaneous pacemaker
A transcutaneous pacemaker, also referred to as external or non-invasive pacing, is a temporary pacemaker that's used in an emergency. The device consists of an external, battery-powered pulse generator and a lead or electrode system.
Dire straits

In a life-threatening situation, a transcutaneous pacemaker works by sending an electrical impulse from the pulse generator to the patient’s heart by way of two electrodes that 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 practitioner can institute transvenous pacing.

**Nursing considerations**

- Attach monitoring electrodes to the patient in the lead I, II, or III position. Do so even if the patient is already on telemetry monitoring, because you must connect the electrodes to the pacemaker. If you select the lead II position, adjust the left leg (LL) electrode placement to accommodate the anterior pacing electrode and the patient’s anatomy.
- Plug the patient cable into the ECG input connection on the front of the pacing generator. Set the selector switch to the monitor on position.
- You should see the ECG waveform on the monitor. Adjust the R-wave beeper volume to a suitable level and activate the alarm by pressing the alarm on button. Set the alarm for 10 to 20 beats lower and 20 to 30 beats higher than the intrinsic rate.
- Press the start/stop button for a printout of the waveform.
- Now you’re ready to apply the two pacing electrodes.

**Proper placement**

- First, make sure the patient’s skin is clean and dry to ensure good skin contact.
- Pull off the protective strip from the posterior electrode (marked back) and apply the electrode on the left side of the back, just below the scapula and to the left of the spine.
- The anterior pacing electrode (marked front) has two protective strips—one covering the jellied area and one covering the outer ring. Expose the jellied area and apply it to the skin in the anterior position—to the left of the precordium in the usual $V_2$ to $V_5$ position. Move this electrode around to get the best waveform. Then expose the electrode’s outer rim and firmly press it to the skin. (See Proper electrode placement.)

**Now to pacing**

- After making sure the energy output in milliamperes (mA) is on, connect the electrode cable to the monitor output cable.
- Check the waveform, looking for a tall QRS complex in lead II.
Proper electrode placement

Place the two pacing electrodes for a transcutaneous pacemaker at heart level on the patient's chest and back (as shown). This placement ensures that the electrical stimulus must travel only a short distance to the heart.

- Next, turn the selector switch to PACER ON. Tell the patient that he may feel a thumping or twitching sensation. Reassure him that you'll give him medication if he can't tolerate the discomfort.

Set the beat

- Now set the rate dial to 10 to 20 beats higher than the patient's intrinsic rate. Look for pacer artifact or spikes, which will appear as you increase the rate. If the patient doesn't have an intrinsic rhythm, set the rate at 60.
- Slowly increase the amount of energy delivered to the heart by adjusting the OUTPUT MA dial. Do so until capture is achieved; you'll see a pacer spike followed by a widened QRS complex that
resembles a permanent ventricular contraction. This setting is the pacing threshold. To ensure consistent capture, increase output by 10%. Don't go higher because you could cause the patient needless discomfort.
• With full capture, the patient's heart rate should be approximately the same as the pacemaker rate set on the machine. The usual pacing threshold is between 40 and 60 mA.

Them bones, them bones
• Don't place the electrodes over a bony area because bone conducts current poorly. For female patients, place the anterior electrode under the patient's breast but not over her diaphragm. If the practitioner inserts the electrode through the brachial or femoral vein, immobilize the patient's arm or leg to avoid putting stress on the pacing wires.

Check back with the vitals
• After placement of a transcutaneous 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. If possible, also obtain a rhythm strip before, during, and after pacemaker placement; any time that 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.

Common disorders

In the ED, you're likely to encounter patients with common cardiac emergencies, especially acute coronary syndrome, aortic aneurysm, cardiac arrest, cardiac arrhythmias, cardiac contusion, cardiac tamponade, heart failure, and hypertensive crisis. Regardless of the disorder, the priorities are always to ensure vital functioning—that is, airway, breathing, and circulation.

Acute coronary syndrome

Patients with acute coronary syndrome have some degree of coronary artery occlusion. The degree of occlusion defines whether the acute coronary syndrome is:
• unstable angina
• non-ST-segment elevation MI (non-STEMI)
• ST-segment elevation MI (STEMI).

**Plaque’s place**

The development of 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 thrombin activation.

**What causes it**

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

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

**How it happens**

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:

• 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 a *non-STEMI*. 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:
Ages and stages

Identifying symptoms of MI

The cardinal symptom of a myocardial infarction (MI) is persistent, intense substernal pain that may radiate to the left arm, jaw, neck, or shoulder blades. This pain is unrelieved by rest or nitroglycerin and may last several hours. Some patients with an MI, such as elderly patients and those with diabetes, may not experience pain at all. Other patients experience only mild pain; for example, female patients who experience atypical chest pain with an MI may present with complaints of indigestion and fatigue. Any patient may experience atypical chest pain, but it’s more common in women.

• burning
• squeezing
• crushing tightness in the substernal or precordial chest that may radiate to the left arm, neck, jaw, or shoulder blade. (See Identifying symptoms of MI.)

It hurts when I do this

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

Angina has four major forms:

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

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

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 non-STEMI and STEMI. (See Pinpointing infarction, page 158.)
- 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, and myoglobin.
- With a Q-wave MI, 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. 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.
Pinpointing infarction

The site of myocardial infarction (MI) depends on the vessels involved:
- Occlusion of the circumflex branch of the left coronary artery causes a lateral wall infarction.
- Occlusion of the anterior descending branch of the left coronary artery leads to an anterior wall infarction.
- True posterior or inferior wall infarctions generally result from occlusion of the right coronary artery or one of its branches.
- Right ventricular infarctions can also result from right coronary artery occlusion, and may accompany inferior infarctions, and may cause right-sided heart failure.
- In an ST-segment elevation MI, tissue damage extends through all myocardial layers; in a non-ST-segment elevation MI, damage occurs only in the innermost layer.

- Obstructive lesions may necessitate CABG or PTCA. Other alternatives include laser angioplasty, minimally invasive surgery, rotational 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 3 hours of the onset of symptoms (unless contraindications exist). Thrombolytic therapy involves administration of streptokinase, alteplase, or reteplase.
- PTCA is an option for opening blocked or narrowed arteries.
- 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.
- Morphine is administered as analgesia because pain stimulates the sympathetic nervous system, leading to an increase in heart rate and vasoconstriction.
- Aspirin is administered to inhibit platelet aggregation.

Patency protection

- 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 to relieve chest pain.
• Glycoprotein IIb/IIIa inhibitors (such as abciximab [ReoPro]) are administered to patients with continued unstable angina, with 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 contractility and to 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 to prevent remodeling.
• Laser angioplasty, atherectomy, stent placement, or transmyocardial revascularization may be initiated.
• Lipid-lowering drugs are administered to patients with elevated low-density lipoprotein and cholesterol levels.

What to do
• On admission, 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. (See Acute coronary syndrome algorithm, pages 160 and 161.)
• 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

(Text continues on page 162.)
Acute coronary syndrome algorithm

The algorithm below shows the American Heart Association's guidelines for treating a patient with acute coronary syndrome.

**Chest discomfort suggestive of ischemia**

**EMS assessment and care and hospital preparation**
- Monitor and support airway, breathing, and circulation (ABCs). Be prepared to provide CPR and defibrillation.
- Administer oxygen, aspirin, nitroglycerin, and morphine if needed.
- If available, obtain 12-lead ECG. If ST-segment elevation:
  - Notify receiving hospital with transmission or interpretation.
  - Begin fibrinolytic checklist.
- Notified hospital should mobilize hospital resources to respond to ST-segment elevation myocardial infarction (STEMI).

**Immediate ED assessment (< 10 min)**
- Check vital signs; evaluate oxygen saturation.
- Establish I.V. access.
- Obtain and review 12-lead ECG.
- Perform brief, targeted history and physical examination.
- Review or complete fibrinolytic checklist; check contraindications.
- Obtain initial cardiac marker levels and initial electrolyte and coagulation studies.
- Obtain portable chest X-ray (< 30 min).

**Immediate ED general treatment**
- Start oxygen at 4 L/minute; maintain O₂ sat > 90%.
- Aspirin 160 to 325 mg (if not given by EMS)
- Nitroglycerin sublingual, spray, or I.V.
- Morphine I.V. if pain isn't relieved by nitroglycerin

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.
- 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.

Aortic aneurysm

An aortic aneurysm is a localized outpouching or an abnormal dilation in a weakened arterial wall. Aortic aneurysm is typically found 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:
- pregnancy
- Marfan syndrome
- long-standing history of systemic hypertension and a preexisting aneurysm (in advanced age)
- trauma.

How it happens

Aneurysms arise from a defect in the middle layer of the arterial wall (tunica media, or medial layer). When 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

When 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 stress and pressure 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 an MI, renal calculi, lumbar disk disease, or duodenal compression.

When symptoms arise

Usually, the patient exhibits symptoms if rupture, expansion, embolization, thrombosis, or pressure from the mass on surrounding structures exists. Rupture is more common if the patient also has hypertension or if the aneurysm is larger than 6 cm. If the patient has a suspected thoracic aortic aneurysm, assess for:

- difficulty breathing
- complaints of sudden, excruciating, tearing pain that moves from the anterior to the posterior
- hoarseness or coughing
- nausea and vomiting
- diaphoresis
- hematemesis
- dysphagia
- aortic insufficiency murmur
- hemoptysis
- palpable pulsations at the left sternoclavicular joint
- tachycardia
- unequal blood pressure and pulse when measured in both arms.

Acute expansion

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

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

What tests tell you
No specific laboratory test diagnoses an aortic aneurysm; however, several other tests may be helpful:
• If blood is leaking from the aneurysm, leukocytosis and a decrease in hemoglobin and hematocrit may be noted.
• 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, length, 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 magnetic resonance imaging (MRI) can disclose the aneurysm's size and effect on nearby organs.
• Serial ultrasonography at 6-month intervals reveals growth of small aneurysms.
• Aortography is used to determine the aneurysm's approximate size and the patency of 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.
Emergency measures

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
• 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 appear 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.

ABGs and arterial lines

• 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 beta blockers to decrease blood pressure, heart rate, and left ventricular contractility.
• Administer I.V. morphine as ordered, to relieve pain if present.
• Administer nitroprusside (Nipride) I.V. only after beta blockers have been initiated because the heart rate can increase and potentially extend the dissection.
• Observe the patient for signs of rupture, which may be immediately fatal. Watch closely for signs of acute blood loss, such as decreasing blood pressure, increasing pulse and respiratory rates, restlessness, decreased LOC, and cool, clammy skin.
Rupture response

- If rupture does take place, insert a large-bore I.V. catheter, begin fluid resuscitation, and administer nitroprusside I.V. as ordered, usually to maintain a mean arterial pressure (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/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.
- Inform the patient and his family of possible transfer to the CCU after surgery.

Cardiac arrest

Cardiac arrest is the absence of mechanical functioning of the heart muscle. The heart stops beating or beats abnormally and doesn’t pump effectively. If blood circulation isn’t restored within minutes, cardiac arrest can lead to the loss of arterial blood pressure, brain damage, and death. (See Adult BLS cardiac arrest algorithm. See also ACLS pulseless arrest algorithm, pages 168 and 169.)

What causes it

Cardiac arrest can be caused by a wide variety of conditions, including acute MI, ventricular fibrillation, ventricular tachycardia, severe trauma, hypovolemia, metabolic disorders, brain injury, respiratory arrest, drowning, or drug overdose.

How it happens

In cardiac arrest, myocardial contractility stops, resulting in a lack of cardiac output. An imbalance in myocardial oxygen supply and demand follows, leading to myocardial ischemia, tissue necrosis, and death.

What to look for

The patient experiencing a cardiac arrest suddenly loses consciousness. Spontaneous respirations are absent, and the patient has no palpable pulse.

What tests tell you

No specific diagnostic tests are used to confirm a cardiac arrest. However, cardiac monitoring or ECG may reveal an underlying cardiac arrhythmia, such as ventricular fibrillation or asystole.
Adult BLS cardiac arrest algorithm

This algorithm shows the basic life support (BLS) steps to follow when you suspect cardiac arrest in an adult patient.

- No movement or response
  - PHONE 911 or emergency number. Get AED or send second rescuer (if available) to do this.
  - Open AIRWAY, check BREATHING.
  - If not breathing, give 2 BREATHS that make chest rise.
  - If no response, check pulse. Do you DEFINITELY feel pulse within 10 seconds?
    - No pulse
      - Give cycles of 30 COMPRESSIONS and 2 BREATHS until AED/defibrillator arrives, ALS providers take over, or victim starts to move.
        - Push hard and fast (100/min) and release completely.
        - Minimize interruptions in compressions.
      - AED/defibrillator ARRIVES
        - Check rhythm. Shockable rhythm?
          - Shockable
            - Give 1 shock. Resume CPR immediately for 5 cycles.
          - Not Shockable
            - Resume CPR immediately for 5 cycles. Check rhythm every 5 cycles; continue until ALS providers take over or victim starts to move.


How it's treated

Treatment of cardiac arrest involves basic and advanced cardiac life support measures in conjunction with treating the underlying

(Text continues on page 170.)
ACLS pulseless arrest algorithm

This algorithm shows the American Heart Association’s guidelines for treating a patient in pulseless cardiac arrest.

1. **PULSELESS ARREST**
   - BLS algorithm: call for help, give CPR.
   - Give oxygen when available.
   - Attach monitor and defibrillator when available.

2. **Check rhythm.**
   - **Shockable rhythm?**

3. **Ventricular fibrillation or Ventricular tachycardia**

4. **Give 1 shock.**
   - Manual biphasic; device specific, typically 120 to 200 joules (Note: If unknown, use 200 joules.)
   - AED: Device specific
   - Monophasic: 360 joules
   - Resume CPR immediately.

5. **Not Shockable**

6. **Asystole/PEA**

7. **Resume CPR immediately for 5 cycles.**
   - When I.V./I.O. available, give vasopressor.
   - Epinephrine 1 mg I.V./I.O.
   - Repeat every 3 to 5 min OR
   - May give 1 dose of vasopressin 40 U I.V./I.O. to replace first or second dose of epinephrine.
   - Consider atropine 1 mg I.V./I.O. for asystole or slow PEA rate; repeat every 3 to 5 min (up to 3 doses).

**Common Disorders**

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**Give 5 cycles of CPR.**

5. Check rhythm. **Shockable rhythm?**

   - **Shockable**
     - Continue CPR while defibrillator is charging.
     - Give 1 shock.
     - Manual biphasic; device specific; same as the first choice or higher dose. *(Note: If unknown, use 200 joules.)*
     - AED: Device specific
     - Monophasic: 360 joules
     - Resume CPR immediately after the shock.
     - When I.V./I.O. available, give vasopressor during CPR.
     - Epinephrine 1 mg I.V./I.O.
     - Repeat every 3 to 5 min OR
     - May give 1 dose of vasopressin 40 units I.V./I.O. to replace first or second dose of epinephrine.

   - **Not Shockable**
     - Give 5 cycles of CPR.

5. Check rhythm. **Shockable rhythm?**

   - **Shockable**
     - If asystole, go to Step 10.
     - If electrical activity, check pulse; if no pulse, go to Step 10.
     - If pulse present, begin postresuscitation care.

6. Continue CPR while defibrillator is charging. Give 1 shock.

   - Manual biphasic; device specific; same as the first choice or higher dose. *(Note: If unknown, use 200 joules.)*
   - AED: Device specific
   - Monophasic: 360 joules
   - Resume CPR immediately after the shock.

   When I.V./I.O. available, give vasopressor during CPR.

   - Epinephrine 1 mg I.V./I.O.
   - Repeat every 3 to 5 min OR

   - May give 1 dose of vasopressin 40 units I.V./I.O. to replace first or second dose of epinephrine.

---

**Give 5 cycles of CPR.**

7. Check rhythm. **Shockable rhythm?**

   - **Shockable**
     - Consider anti-arrhythmics; give during CPR (before or after the shock).
     - Amiodarone (300 mg I.V./I.O. once), then consider additional 150 mg I.V./I.O. once, then consider additional 150 mg I.V./I.O. once or lidocaine (1 to 1.5 mg/kg first dose, then 0.5 to 0.75 mg/kg/I.V./I.O. maximum 3 doses or 3 mg/kg).
     - Consider magnesium, loading dose 1 to 2 g I.V./I.O. for torsades de pointes.
     - After 5 cycles of CPR, go to Step 5.

---

**Give 5 cycles of CPR.**

8. Continue CPR while defibrillator is charging. Give 1 shock.

   - Manual biphasic; device specific; same as the first shock or higher dose. *(Note: If unknown, use 200 joules.)*
   - AED: Device specific
   - Monophasic: 360 joules
   - Resume CPR immediately after the shock.

---

**During CPR**

- Search for and treat possible contributing factors:
  - hypovolemia
  - hypoxia
  - hydrogen ion (acidosis)
  - hypo-hyperkalemia
  - hypoglycemia
  - hypothermia
  - toxins
  - tamponade, cardiac
  - tension pneumothorax
  - thrombosis (coronary or pulmonary)
  - trauma.

- Push hard and fast (100/minute).
- Ensure full chest recoil.
- Minimize interruptions in chest compressions.
- One cycle of CPR: 30 compressions then 2 breaths; 5 cycles = 2 minutes.
- Avoid hyperventilation.
- Secure the airway and confirm placement.
- Rotate compression every 2 minutes with rhythm checks.

---

**Note:** After an advanced airway is placed, rescuers no longer deliver “cycles” of CPR. Give continuous chest compressions without pauses for breaths. Give 8 to 10 breaths/minute. Check rhythm every 2 minutes.
cause of the arrest. The ultimate goal of treatment is to restore the patient’s cardiac rhythm and function.

**What to do**
- Determine responsiveness and notify the practitioner and resuscitation team.
- Initiate CPR.
- Monitor cardiac rhythm.
- Assist with ET intubation and mechanical ventilation.
- Follow advanced cardiac life support (ACLS) protocols; administer medications as ordered.
- Assist with defibrillation for ventricular fibrillation or pulseless ventricular tachycardia.

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

How it happens
Cardiac arrhythmias may result from:
• abnormal electrical conduction
• escape beats (additional abnormal heart beats resulting from a very slow heart rate)
• enhanced automaticity
• reentry.

What to look for
When a patient presents with a history of symptoms suggesting cardiac arrhythmias or has been treated for a cardiac arrhythmia, be alert for:
• reports of precipitating factors, such as exercise, smoking, sleep patterns, 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, ventricular tachycardia.

That’s not all
Additional findings may include:
• dizziness
• weakness
• chest pains
• cold and clammy extremities
• hypotension
• dyspnea
• pallor
• reduced urine output
• syncope (with severely impaired cerebral circulation).
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 174 to 181.)
- Laboratory testing may reveal electrolyte abnormalities, hypoxemia or acid-base abnormalities (with ABG analysis), or drug toxicities as the cause of 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 for cardiac arrhythmias 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
- management of the underlying disorder, such as correction of hypoxia
- temporary or permanent placement of a pacemaker to maintain heart rate
- Valsalva's maneuver
- implantable cardioverter-defibrillator (ICD), if indicated
- surgical removal or cryotherapy of an irritable ectopic focus to prevent recurring arrhythmias.

What to do

- Evaluate the patient’s ECG frequently 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. Follow ACLS protocol to treat specific life-threatening arrhythmias.
- 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 appropri-
Cardiac contusion

Cardiac contusion refers to the bruising of the myocardium. It's the most common type of injury sustained from blunt trauma to the chest.

What causes it

A cardiac contusion typically results from blunt trauma. This trauma can be related to vehicular collisions or falls. The right ventricle is the most common site of injury because it's located directly behind the sternum.

How it happens

During deceleration injuries, the myocardium strikes the sternum when the heart and aorta move forward. In addition, the aorta may be lacerated by shearing forces. Direct force may also be applied to the sternum, causing injury. Crushing and compressive forces may result in contusion as the heart is compressed between the sternum and vertebral column.

What to look for

Cardiac contusion should be suspected after any blow to the chest. Be alert for these signs and symptoms of trauma:

- shortness of breath
- bruising on the chest
- murmurs
- bradycardia or tachycardia
- precordial chest pain.

And also...

Keep these signals in mind as well:

(Text continues on page 180.)
Understanding cardiac arrhythmias

Here's an outline of many common cardiac arrhythmias and their features, causes, and treatments. Use a normal electrocardiogram 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/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 and 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/minute
- Ventricular rate variable, depending on degree of AV block (usually 60 to 100 beats/minute)
- No P waves; atrial activity appears as flutter waves (F waves); saw-tooth configuration common in lead II
- QRS complexes are uniform in shape but commonly irregular in rhythm
<table>
<thead>
<tr>
<th>Causes</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Normal physiologic response to fever, exercise, anxiety, pain, dehydration; may also accompany shock, left-sided heart failure, cardiac tamponade, hyperthyroidism, anemia, hypovolemia, pulmonary embolism (PE), and anterior wall myocardial infarction (MI)</td>
<td>• Correction of underlying cause</td>
</tr>
<tr>
<td>• May also occur with atropine, epinephrine, isoproterenol, quinidine, caffeine, alcohol, cocaine, amphetamine, and nicotine use</td>
<td>• Beta-adrenergic blockers or calcium channel blocker</td>
</tr>
<tr>
<td>• Normal in a well-conditioned heart, as in an athlete</td>
<td>• Correction of underlying cause</td>
</tr>
<tr>
<td>• Increased intracranial pressure; increased vagal tone due to strain during defecation, vomiting, intubation, or mechanical ventilation; sick sinus syndrome (SSS); hypothyroidism; and inferior-wall MI</td>
<td>• For low cardiac output, dizziness, weakness, altered level of consciousness, or low blood pressure, advanced cardiac life support (ACLS) protocol for administration of atropine</td>
</tr>
<tr>
<td>• May also occur with anticholinesterase, beta-adrenergic blocker, digoxin, and morphine use</td>
<td>• Transcutaneous or permanent pacemaker</td>
</tr>
<tr>
<td></td>
<td>• Dopamine or epinephrine infusion</td>
</tr>
<tr>
<td>• Intrinsic abnormality of atrioventricular (AV) conduction system</td>
<td>• If patient is unstable, immediate cardioversion</td>
</tr>
<tr>
<td>• Physical or psychological stress, hypoxia, hypokalemia, cardiomyopathy, congenital heart disease, MI, valvular disease, Wolff-Parkinson-White syndrome, cor pulmonale, hyperthyroidism, and systemic hypertension</td>
<td>• If QRS complex is narrow and regular and patient is stable, perform vagal maneuvers or administer adenosine</td>
</tr>
<tr>
<td>• Digoxin toxicity; use of caffeine, marijuana, or central nervous system stimulants</td>
<td>• If QRS complex is narrow and irregular, control the rate using calcium channel blockers or beta-adrenergic blockers</td>
</tr>
<tr>
<td>• Heart failure, tricuspid or mitral valve disease, PE, cor pulmonale, inferior-wall MI, and pericarditis</td>
<td>• If QRS complex is wide and irregular, administer antiarrhythmics such as amiodarone; if ineffective, then magnesium</td>
</tr>
<tr>
<td>• Digoxin toxicity</td>
<td>• If patient is unstable with a ventricular rate &gt; 150 beats/minute, immediate cardioversion</td>
</tr>
<tr>
<td></td>
<td>• If patient is stable, follow ACLS protocol for cardioversion and drug therapy, which may include calcium channel blockers, beta-adrenergic blockers, amiodarone, or digoxin</td>
</tr>
<tr>
<td></td>
<td>• Anticoagulation therapy possibly also needed</td>
</tr>
</tbody>
</table>
|                                                                      | • Radiofrequency ablation to control rhythm                  | (continued)
### Understanding cardiac arrhythmias (continued)

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

**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
<table>
<thead>
<tr>
<th>Causes</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart failure, chronic obstructive pulmonary disease, thyrotoxicosis, constrictive pericarditis, ischemic heart disease, sepsis, PE, rheumatic heart disease, hypertension, mitral stenosis, atrial irritation, or complication of coronary bypass or valve replacement surgery</td>
<td>If patient is unstable with a ventricular rate &gt; 150 beats/minute, immediate cardioversion</td>
</tr>
<tr>
<td>Nifedipine and digoxin use</td>
<td>If patient is stable, follow ACLS protocol and drug therapy, which may include calcium channel blockers, beta-adrenergic blockers, amiodarone, or digoxin</td>
</tr>
<tr>
<td></td>
<td>Anticoagulation therapy possibly also needed</td>
</tr>
<tr>
<td></td>
<td>In some patients with refractory atrial fibrillation uncontrolled by drugs, radiofrequency catheter ablation</td>
</tr>
<tr>
<td>Inferior-wall MI or ischemia, hypoxia, vagal stimulation, and SSS</td>
<td>Correction of underlying cause</td>
</tr>
<tr>
<td>Acute rheumatic fever</td>
<td>Atropine for symptomatic slow rate</td>
</tr>
<tr>
<td>Valve surgery</td>
<td>Pacemaker insertion if patient doesn’t respond to drugs</td>
</tr>
<tr>
<td>Digoxin toxicity</td>
<td>Discontinuation of digoxin, if appropriate</td>
</tr>
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<td></td>
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<tr>
<td>Possible in healthy persons</td>
<td>Correction of underlying cause</td>
</tr>
<tr>
<td>Inferior-wall MI or ischemia, hypothyroidism, hypokalemia, and hyperkalem</td>
<td>Possibly atropine if severe symptomatic bradycardia develops</td>
</tr>
<tr>
<td>Digoxin toxicity; use of quinidine, procainamide, beta-adrenergic blockers, calcium channel blockers, or amiodarone</td>
<td>Cautious use of digoxin, calcium channel blockers, and beta-adrenergic blockers</td>
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<tr>
<td></td>
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<tr>
<td>Inferior-wall MI, cardiac surgery, acute rheumatic fever, and vagal stimulation</td>
<td>Treatment of underlying cause</td>
</tr>
<tr>
<td>Digoxin toxicity; use of propranolol, quinidine, or procainamide</td>
<td>Atropine or transcutaneous pacemaker for symptomatic bradycardia</td>
</tr>
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<td></td>
<td>Discontinuation of digoxin, if appropriate</td>
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<tr>
<td>Severe coronary artery disease (CAD), anterior-wall MI, and acute myocarditis</td>
<td>Temporary or permanent pacemaker</td>
</tr>
<tr>
<td>Digoxin toxicity</td>
<td>Atropine, dopamine, or epinephrine for symptomatic bradycardia</td>
</tr>
<tr>
<td></td>
<td>Discontinuation of digoxin, if appropriate</td>
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</tbody>
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(continued)
### Understanding cardiac arrhythmias (continued)

#### Arrhythmia and features

<table>
<thead>
<tr>
<th>Arrhythmia and features</th>
<th>Third-degree AV block (complete heart block)</th>
<th>Premature ventricular contraction (PVC)</th>
<th>Ventricular tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Atrial rhythm regular</td>
<td>• Atrial rhythm regular</td>
<td>• Ventricular rate 100 to 220 beats/minute; rhythm usually regular</td>
</tr>
<tr>
<td></td>
<td>• Ventricular rhythm regular and rate slower than atrial rate</td>
<td>• Ventricular rhythm irregular</td>
<td>• QRS complexes wide, bizarre, and independent of P waves</td>
</tr>
<tr>
<td></td>
<td>• No relation between P waves and QRS complexes</td>
<td>• QRS complex premature, usually followed by a complete compensatory pause</td>
<td>• P waves not discernible</td>
</tr>
<tr>
<td></td>
<td>• No constant PR interval</td>
<td>• QRS complex wide and distorted, usually &gt; 0.12 second</td>
<td>• May start and stop suddenly</td>
</tr>
<tr>
<td></td>
<td>• QRS duration normal (junctional pacemaker) or wide and bizarre (ventricular pacemaker)</td>
<td>• Premature QRS complexes occurring alone, in pairs, or in threes, alternating with normal beats; focus from one or more sites</td>
<td></td>
</tr>
</tbody>
</table>
Causes

- Inferior- or anterior-wall MI, congenital abnormality, rheumatic fever, hypoxia, postoperative complication of mitral valve replacement, post-procedure complication of radiofrequency ablation in or near AV nodal tissue, Lev’s disease (fibrosis and calcification that spreads from cardiac structures to the conductive tissue), and Lenègre’s disease (conductive tissue fibrosis)
- Digoxin toxicity

- Heart failure; old or acute MI, ischemia, or contusion; myocardial irritation by ventricular catheter or a pacemaker; hypercapnia; hypokalemia; hypocalcemia; and hypomagnesemia
- Drug toxicity (digoxin, aminophylline, tricyclic antidepressants, beta-adrenergic blockers, isoproterenol, or dopamine)
- Caffeine, tobacco, or alcohol use
- Psychological stress, anxiety, pain, or exercise

- Myocardial ischemia, MI, or aneurysm; CAD; rheumatic heart disease; mitral valve prolapse; heart failure; cardiomyopathy; ventricular catheters; hypokalemia; hypercalcemia; hypomagnesemia; and PE
- Digoxin, procainamide, epinephrine, or quinidine toxicity
- Anxiety

Treatment

- Atropine, dopamine, or epinephrine for symptomatic bradycardia
- Transcutaneous or permanent pacemaker

- 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

- If regular QRS rhythm (monomorphic), administer amiodarone (follow ACLS protocol); if drug is unsuccessful, cardioversion
- If irregular QRS rhythm (polymorphic) and QT interval is prolonged, stop medications that may prolong QT interval; correct electrolyte imbalance; administer magnesium; if ineffective, cardioversion
- If irregular QRS rhythm (polymorphic) and QT interval is normal, stop medications that may prolong QT interval; correct electrolyte balance; administer amiodarone; if ineffective, cardioversion
- If the patient with monomorphic or polymorphic QRS complexes becomes unstable, immediate defibrillation
- If pulseless, initiate CPR; follow ACLS protocol for defibrillation, endotracheal (ET) intubation, and administration of epinephrine or vasopressin, followed by amiodarone or lidocaine and, if ineffective, magnesium sulfate or procainamide
- Implantable cardioverter-defibrillator (ICD) if recurrent ventricular tachycardia

(continued)
Understanding cardiac arrhythmias (continued)

Arrhythmia and features

**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

- Arrhythmias due to ventricular irritability
- Cardiac tamponade
- Hemodynamic instability
- Pericardial friction rub.

**What tests tell you**
- ECG will reveal rhythm disturbances, such as premature ventricular contractions, premature atrial contractions, ventricular tachycardia, atrial tachycardia, and ventricular fibrillation, along with nonspecific ST-segment or T-wave changes occurring within 24 to 48 hours after the injury.
- Echocardiogram will show evidence of abnormal ventricular wall movement and decreased ejection fraction.
- Multiple-gated acquisition scan will show decreased ability of effective heart pumping.
- Cardiac enzyme levels will show elevations of CK-MB to greater than 8% of total CK within 3 to 4 hours after the injury.
- Cardiac troponin I levels may be elevated 24 hours after the injury.

**How it’s treated**
Maintaining hemodynamic stability and adequate cardiac output are key. I.V. fluid therapy may be necessary. Continuous ECG mon-
<table>
<thead>
<tr>
<th>Causes</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Myocardial ischemia, MI, untreated ventricular tachycardia, R-on-T</td>
<td>• CPR; follow ACLS protocol for defibrillation, ET intubation,</td>
</tr>
<tr>
<td>phenomenon, hypokalemia, hyperkalemia, hypercalcemia, hypoxemia,</td>
<td>and administration of epinephrine or vasopressin, amiodarone, or</td>
</tr>
<tr>
<td>alkalosis, electric shock, and hypothermia</td>
<td>lidocaine and, if ineffective, magnesium sulfate or procainamide</td>
</tr>
<tr>
<td>• Digoxin, epinephrine, or quinidine toxicity</td>
<td>• ICD if risk of recurrent ventricular fibrillation</td>
</tr>
<tr>
<td>• Myocardial ischemia, MI, aortic valve disease, heart failure,</td>
<td>• Continue CPR; follow ACLS protocol for ET intubation,</td>
</tr>
<tr>
<td>hypoxia, hypokalemia, severe acidosis, electric shock, ventricular</td>
<td>temporary pacing, and administration of epinephrine or vasopressin</td>
</tr>
<tr>
<td>arrhythmia, AV block, PE, heart rupture, cardiac tamponade,</td>
<td>and atropine</td>
</tr>
<tr>
<td>hyperkalemia, and electromechanical dissociation</td>
<td></td>
</tr>
<tr>
<td>• Cocaine overdose</td>
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</table>

Monitoring is used to detect arrhythmias. Lidocaine may be administered to treat ventricular arrhythmias, and digoxin may be given to treat pump failure. Inotropic agents maybe used to assist with improving cardiac output and ejection fraction.

Close watch

The patient with a cardiac contusion must be monitored closely for signs and symptoms of cardiopulmonary compromise because trauma leading to cardiac contusion is commonly associated with pulmonary trauma. Supplemental oxygen therapy may be necessary. If the extent of pulmonary trauma is great, ET intubation and mechanical ventilation may be necessary.

No if hypo

I.V. morphine may be used to treat severe pain, unless the patient is hypotensive. In the latter case, other, less potent analgesics may be used.

What to do

• Assess the patient's cardiopulmonary status at least hourly, or more frequently if indicated, to detect signs and symptoms of possible injury.
• Auscultate breath sounds at least hourly, reporting signs of congestion or fluid accumulation. Evaluate peripheral pulses and capillary refill to detect decreased peripheral tissue perfusion.
- Monitor heart rate and rhythm, heart sounds, and blood pressure every hour for changes; institute hemodynamic monitoring, including central venous pressure, PAWP, and cardiac output as indicated, at least every 1 to 2 hours.
- Administer fluid replacement therapy, including blood component therapy as prescribed, typically to maintain systolic blood pressure above 90 mm Hg.
- Monitor urine output every hour, notifying the practitioner if output is less than 30 ml/hour.
- Institute continuous cardiac monitoring to detect arrhythmias or conduction defects. If arrhythmias appear, administer antiarrhythmic agents as ordered.
- Assess the patient's degree of pain and administer analgesic therapy as ordered, monitoring him for effectiveness. Position the patient comfortably, usually with the head of the bed elevated 30 to 45 degrees.
- Prepare the patient and his family for surgery, if indicated.
- Anticipate transfer of the patient to a critical care unit when appropriate.

Cardiac tamponade

Cardiac tamponade is a rapid, unchecked increase in pressure in the pericardial sac. This increased pressure 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. (See Understanding cardiac tamponade.)

What causes it

Cardiac tamponade may result from:
- viral or postirradiation pericarditis
- acute MI
- chronic renal failure requiring dialysis
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 of the heart, also known as the epicardial layer, clings to 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 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 cardiac output. Without prompt treatment, low cardiac output can be fatal.

- connective tissue disorders (such as rheumatoid arthritis, systemic lupus erythematosus, rheumatic fever, vasculitis, and scleroderma)
- effusion (from cancer, bacterial infections, tuberculosis and, rarely, acute rheumatic fever)
- hemorrhage due to nontraumatic causes (such as anticoagulant therapy in patients with pericarditis or rupture of the heart or great vessels)
- hemorrhage due to trauma (such as gunshot or stab wounds of the chest)
- idiopathic causes (such as Dressler's syndrome)
- drug reaction from procainamide (Procanbid), hydralazine (Apresoline), minoxidil (Rogaine), isoniazid (Nydrazid), penicillin (Bicillin), or daunorubicin (DaunoXome).
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.

What to look for
Cardiac tamponade has three classic features known as Beck's triad:
- elevated CVP with jugular vein distention
- muffled heart sounds
- pulsus paradoxus (inspiratory drop in systemic blood pressure greater than 15 mm Hg).

That's not all
Other signs include:
- restlessness
- anxiety
- cold, clammy skin
- cyanosis
- diaphoresis
- orthopnea
- decreased arterial pressure, decreased systolic blood pressure, and narrow pulse pressure
- tachycardia and a weak, thready pulse.

What tests tell you
- Chest X-ray shows a slightly widened mediastinum and an enlarged cardiac silhouette.
- ECG may show a low-amplitude QRS complex and electrical alternans, or 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 right atrial pressure, right ventricular diastolic pressure, and CVP.
- 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,
which can be done three different ways:

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

When pressure is low

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

Additional treatments

Additional treatment may necessary, depending on the cause. Ex-
amples of such causes and treatments are:

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

What to do

- Monitor the patient’s cardiovascular status frequently, at least
every hour, noting the extent of jugular vein distention, quality of
heart sounds, and blood pressure.
- Assess hemodynamic status, including CVP, right atrial pres-
sure, and PAP, and determine cardiac output.
- Monitor for pulsus paradoxus.
- Be alert for ST-segment and T-wave changes on the ECG. Note
rate and rhythm, and report evidence of arrhythmias.

Keep an eye on the increase

- Watch closely for signs of increasing tamponade, increasing dys-
pnea, and arrhythmias; report them immediately.
- Infuse I.V. solutions and inotropic drugs, such as dopamine, 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.
- 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.
- Anticipate transfer of the patient to a CCU when appropriate.

Heart failure

Heart failure results when the heart can’t pump enough blood to meet the metabolic needs of the body. It 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:
- left-sided heart failure
- right-sided heart failure
- systolic dysfunction
- 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:

• hypertension
• aortic and mitral valve stenosis
• left ventricular infarction.

When right goes wrong

Right-sided heart failure results from ineffective right ventricular contractile function. 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.

Just can’t pump enough

Systolic dysfunction results 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 and dilated cardiomyopathy.

It all goes to swell from here

Diastolic dysfunction results 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, or restrictive cardiomyopathy. This type of heart failure is less common than heart failure resulting from 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. These 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. Angiotensin I 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 increased volume stretches cardiac muscle fibers so that the ventricle can accept the increased volume. Eventually, the muscle becomes stretched beyond optimal 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 and atrial natriuretic factor—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 hormone
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. Atrial natriuretic factor works to counteract the negative effects of sympathetic nervous system stimulation and the renin-angiotensin-aldosterone system by producing vasodilation and diuresis.

What to look for
Early signs and symptoms of left-sided heart failure include:
- fatigue
- nonproductive cough
- orthopnea
- dyspnea
- paroxysmal nocturnal dyspnea.

Later, on the left
Later clinical manifestations of left-sided heart failure may include:
- cool, pale skin
- restlessness and confusion
- displacement of the PMI toward the left anterior axillary line
- hemoptysis
- crackles on auscultation
- S₃ heart sound
- S₄ heart sound
- tachycardia.

On the right side
Clinical manifestations of right-sided heart failure include:
- weight gain
- anorexia, fullness, and nausea
- edema
• ascites or anasarca
• hepatojugular reflux and hepatomegaly
• jugular vein distention
• nocturia
• right upper quadrant pain.

What tests tell you
• 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 and extrasystoles.
• Laboratory testing may reveal abnormal liver function and elevated BUN and creatinine levels.
• ABG analysis may reveal hypoxemia from impaired gas exchange and respiratory alkalosis because the patient blows off more carbon dioxide as the respiratory rate increases in compensation.
• Echocardiography may reveal left ventricular hypertrophy, dilation, and abnormal contractility.
• Pulmonary artery monitoring typically demonstrates elevated PAP and PAWP, left ventricular end-diastolic pressure 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:
• ACE inhibitors for patients with left ventricular dysfunction to reduce production of angiotensin II, resulting in preload and afterload reduction
• beta-adrenergic blockers to prevent remodeling in patients with mild to moderate heart failure caused by left ventricular systolic dysfunction
• CABG surgery or angioplasty for patients with heart failure due to CAD
• digoxin (Lanoxin) 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 to reduce fluid volume overload, venous return, and preload
• diuretics, nitrates, morphine, and oxygen to treat pulmonary edema
• heart transplantation in patients receiving aggressive medical treatment but still experiencing limitations or repeated hospitalizations
• lifestyle modifications, 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 to reduce symptoms
• other surgery or invasive procedures, such as cardiomyoplasty, insertion of an intra-aortic balloon pump (IABP), partial left ventriculectomy, use of a mechanical VAD, and implantation of an ICD or a biventricular pacemaker
• treatment of the underlying cause, if known.

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.
• 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, at least hourly, noting trends.
• Administer medications as ordered. Check the apical heart rate before administering digoxin.

Pump up the potassium
• Expect to administer electrolyte replacement therapy (especially potassium) after the administration of diuretics to prevent such imbalances as hypokalemia and the arrhythmias that they may cause.
• Assess respiratory status frequently—at least every hour. Auscultate lungs for abnormal breath sounds, such as crackles, wheezes, and rhonchi. Encourage coughing and deep breathing.
• Obtain a baseline weight and observe for peripheral edema.
• Assess hourly urine output. Also, monitor fluid intake, including I.V. fluids.
Teaching about heart failure

For a patient with heart failure, education about the disorder and treatments is essential to prevent complications and minimize the effects of this condition on quality of life. Additionally, a thorough understanding of the condition may help prevent future admission to the emergency department (ED).

Although admission to the ED is commonly filled with activity, it's an opportune time to begin your teaching. Consider these points:

- Explain underlying problems associated with the patient's heart failure and typical signs and symptoms.
- Review the medications prescribed to treat heart failure.
- Review suggested lifestyle changes, including diet and energy conservation measures.
- Teach the patient about avoiding foods high in sodium, and provide him with a list of foods and their sodium content, indicating which to avoid and which to include in his diet.
- Instruct how to replace potassium lost through diuretic therapy (if appropriate) by taking prescribed potassium supplements or eating potassium-rich foods, such as bananas and apricots, and drinking orange juice.
- Encourage the patient to weigh himself daily and keep a record of his weights; urge the patient to report a weight gain or loss of 2 lb (0.9 kg) or more in 3 or 4 days.
- Stress the importance of taking medications as prescribed; instruct the patient in possible adverse effects and signs and symptoms of toxicity.
- Instruct the patient on how to monitor pulse rate; advise him to report a pulse rate that's unusually irregular or less than 60 beats/minute.
- Review the danger signs and symptoms to report to his practitioner, such as dizziness, blurred vision, shortness of breath, persistent dry cough, palpitations, increased fatigue, swelling of the ankles, and decreased urine output.
- Encourage adherence to medical follow-up, including checkups and periodic blood tests.

- 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.
- Prepare the patient for surgical intervention or insertion of an IABP or ICD, or transfer to the critical care unit if indicated.
- Begin patient teaching related to heart failure and measures to reduce the risk of complications. (See Teaching about heart failure.)
Hypertensive crisis

A hypertensive emergency, commonly called hypertensive crisis, refers to the abrupt, acute, marked increase in blood pressure from the patient’s baseline that ultimately leads to acute and rapidly progressing end-organ damage.

Rapid rise

Typically, in a hypertensive crisis, the patient’s diastolic blood pressure is greater than 120 mm Hg. The increased blood pressure value, although important, is probably less important than how rapidly the blood pressure increases.

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 or Cushing’s syndrome, 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:

- sympathetic nervous system
- antidiuretic hormone
- autoregulation
- renin-angiotensin system.

Up with pressure

The renin-angiotensin system increases blood pressure in several 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 secretion 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, in which blood vessels gradually dilate when blood pressure increases, reducing peripheral resistance
• capillary fluid shift, in which 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.

**Regulating reabsorption**

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. (See *What happens in hypertensive crisis.*)

**Strain for the brain**

In hypertensive crisis, the blood pressure–regulating mechanism is disturbed, causing cerebral vasodilation. 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:

• dizziness
What happens in hypertensive crisis

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

- Abnormal renal function
- Eclampsia
- Hypertensive encephalopathy
- Intracerebral hemorrhage
- Monoamine oxidase inhibitor interactions

Causes of hypertensive crisis
- Myocardial ischemia
- Pheochromocytoma
- Withdrawal of antihypertensive drugs (abrupt)

Prolonged hypertension

Inflammation and necrosis of arterioles

Narrowing of blood vessels

Restriction of blood flow to major organs

Organ damage

Renal
- Decreased renal perfusion
- Progressive deterioration of nephrons
- Decreased ability to concentrate urine
- Increased serum creatinine and blood urea nitrogen
- Increased renal tubule permeability with protein leakage into tubules
- Renal insufficiency
- Uremia
- Renal failure

Cardiac
- Decreased cardiac perfusion
- Coronary artery disease
- Angina or myocardial infarction
- Increased cardiac workload
- Left ventricular hypertrophy
- Heart failure

Cerebral
- Decreased cerebral perfusion
- Increased stress on vessel wall
- Arterial spasm
- Ischemia
- Transient ischemic attacks
- Weakening of vessel intima
- Aneurysm formation
- Intracranial hemorrhage
• confusion, somnolence, or stupor
• irritability
• nausea
• vomiting
• anorexia
• edema
• acute retinopathy and hemorrhage, retinal exudates, and papilledema
• angina
• dyspnea on exertion, orthopnea, or paroxysmal nocturnal dyspnea
• possible left ventricular heave palpated at the mitral valve area
• severe, throbbing headache in the back of the head
• S4 heart sound
• vision loss, blurred vision, or diplopia.

**Check the head**
If the patient has hypertensive encephalopathy, you may note:
• disorientation
• decreased LOC
• seizures.

**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—when obtained several times at an interval of at least 2 minutes, revealing an elevated diastolic pressure greater than 120 mm Hg—confirms the diagnosis of hypertensive crisis.
• RBC count may be decreased secondary to hematuria if the kidneys are involved.
• If the kidneys are involved, BUN may be greater than 20 mg/dl and the serum creatinine level may be greater than 1.3 mg/dl.
• ECG may reveal ischemic changes or left ventricular hypertrophy. ST-segment depression and T-wave inversion suggest repolarization problems from endocardial fibrosis associated with left ventricular hypertrophy.
• Echocardiography may reveal increased wall thickness with or without an increase in left ventricle 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 renal impairment occurs; then specific gravity is low (less than 1.010), and 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.

**How it’s treated**
Treatment of hypertensive crisis immediately focuses on reducing the patient’s blood pressure with I.V. antihypertensive therapy. However, you must take care not to reduce the patient’s blood pressure too rapidly because his autoregulatory control is impaired.

**Slow pressure cuts**
The current recommendation is to reduce blood pressure by no more than 25% of the MAP over the first 2 hours. The next several days should bring further reductions. Here are some additional guidelines:

- 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 enables the blood pressure to increase almost immediately.
- Other agents that may be used include labetalol (Normodyne), nitroglycerin (Nitro-Bid) (the drug of choice for treating hypertensive crisis when myocardial ischemia, acute MI, or pulmonary edema is present), and hydralazine (Apresoline) (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.
- 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; titrate I.V. antihypertensive medications according to the desired response and parameters set by the practitioner.
- If using nitroprusside (Nipride), 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.
- If the patient is receiving nitroprusside (Nitropress) therapy, assess for signs and symptoms of thiocyanate toxicity, such as fatigue, nausea, tinnitus, blurred vision, and delirium. Nitroprusside is metabolized to thiocyanate, which is excreted by the kidneys. If signs are present, obtain a serum thiocyanate level; if it's greater than 10 mg/dl, notify the practitioner.

**Much monitoring**
- 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 ECG and institute treatment as indicated if you find arrhythmias. Auscultate the patient's heart, noting signs of heart failure such as S₃ or S₄ heart sounds.
- Assess the patient's neurologic status frequently—every 15 to 30 minutes initially and then every hour, based on the patient's response to therapy.

**Check in on output**
- 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.
- Administer other antihypertensives as ordered. 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.
- Anticipate transfer of the patient to the CCU as indicated.
- Provide support to the patient and his family; begin patient teaching related to the condition and measures to reduce the risk of complications as the patient's condition begins to stabilize. (See Teaching about hypertensive crisis.)
Teaching about hypertensive crisis

Hypertensive crisis is an emergency situation that most commonly results from inadequately controlled hypertension or untreated hypertension. As a result, you need to educate the patient about measures to control his hypertension to reduce the risk of complications and a recurrence of the crisis. As the patient’s condition begins to stabilize and time permits, begin your teaching. Consider these points:

- Explain the underlying events associated with the patient’s current crisis.
- Review the medications being used to treat this acute condition.
- Reinforce all aspects of blood pressure control, such as diet, medications, and lifestyle changes.
- Stress the need for adherence to medication therapy and frequent medical follow up.
- Explain the prescribed medication regimen, including dosage, frequency, adverse effects, and when to notify the practitioner.
- Reinforce necessary lifestyle changes and the need for regular exercise.
- Instruct the patient in signs and symptoms associated with possible complications, such as changes in levels of alertness, headache, vision changes, reduced urine output, and weight gain, along with the need to notify the practitioner if any occur.

Quick quiz

1. Which sign or symptom would the nurse expect to assess in a patient who’s admitted to the ED with a diagnosis of cardiac tamponade?
   
   A. Shortness of breath
   B. Pulsus paradoxus
   C. Holosystolic murmur
   D. Bounding peripheral pulse

   Answer: B. Pulsus paradoxus (inspiratory drop in systemic blood pressure greater than 15 mm Hg) is one of the three classic signs of cardiac tamponade. The other classic signs are elevated CVP with jugular vein distention and muffled heart sounds.
2. A patient receiving I.V. nitroprusside for treatment of hypertensive crisis develops blurred vision and tinnitus. The nurse also notes that his LOC has decreased. Which action is most appropriate?

A. Increase the rate of nitroprusside infusion.
B. Obtain an order for an antiarrhythmic.
C. Obtain a serum thiocyanate level.
D. Increase the flow rate of supplemental oxygen.

*Answer:* C. The patient is exhibiting signs and symptoms of thiocyanate toxicity, which include fatigue, nausea, tinnitus, blurred vision, and delirium. Therefore, the nurse must obtain a serum thiocyanate level and notify the practitioner if the level is greater than 10 mg/dl.

3. Which assessment finding would the nurse expect to find elevated in a client admitted with right-sided heart failure?

A. CVP
B. Left-ventricular end-diastolic pressure
C. PAWP
D. Cardiac output

*Answer:* A. CVP is elevated in right-sided heart failure.

4. When performing synchronized cardioversion, the nurse understands that the electrical charge is delivered at which point?

A. Initiation of the QRS complex
B. During the ST segment
C. At the peak of the R wave
D. Just before the onset of the P wave

*Answer:* C. Synchronized cardioversion delivers an electrical charge to the myocardium at the peak of the R wave.

**Scoring**

⭐⭐⭐ If you answered all four questions correctly, let your heart swell with pride. You’re tops when it comes to cardiac emergencies!

⭐⭐ If you answered three questions correctly, congratulations on all your “heart” work. You’re a member of the cardiac emergency team!

⭐ If you answered fewer than three questions correctly, don’t be heartbroken. Just go back and review the chapter!