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About the American Heart Association

The American Heart Association is a not-for-profit, voluntary health organization funded by private contributions. Our mission is to reduce disability and death from cardiovascular diseases and stroke. These include heart attack, stroke and related disorders.

Preventing heart disease and stroke is — and always has been — our first priority. We've funded about $2 billion in heart and blood vessel research since 1949. Nearly 30 percent of our yearly expenses supports research.

Our association was founded in 1924 in New York City by six cardiologists. Today it's one of the world's premier health organizations. We have 15 regional affiliates and about 2,000 divisions and branches throughout the United States and Puerto Rico. About 22.5 million volunteers join us every year to fight heart and blood vessel diseases, the nation's No. 1 killer.

About this booklet

This booklet discusses the major types of heart and blood vessel (cardiovascular) disorders. In it, you'll learn

1) what some of the major disorders and their risk factors are.

2) what can be done to reduce risk.

3) how victims are diagnosed and treated.

A WORD OF CAUTION: Please don't use this booklet to try to diagnose individual cases. Every case is unique. Only a healthcare professional is qualified to diagnose and prescribe treatment.

A companion booklet, *Heart and Stroke Statistical Update*, contains the most recent prevalence, incidence, mortality and cost statistics available as of the publication date.

The Heart and How It Works

The normal human heart is a strong, muscular pump a little larger than a fist. **Each day an average heart "beats" (expands and contracts) 100,000 times and pumps about 2,000 gallons of blood.** In a 70-year lifetime, an average human heart beats more than 2.5 billion times.

The heart pumps blood continuously through the circulatory system. The circulatory system is the network of elastic tubes that carries blood throughout the body. It includes the heart, lungs, arteries, arterioles (small arteries) and capillaries (very tiny blood vessels). These blood vessels carry oxygen- and nutrient-rich blood to all parts of the body. The circulatory system also includes venules (small veins) and veins. These are the blood vessels that carry oxygen- and nutrient-depleted blood back to the heart and lungs. If all these vessels were laid end-to-end, they'd extend for about 60,000 miles. That's enough to encircle the earth more than twice.

The circulating blood brings oxygen and nutrients to all the body's organs and tissues, including the heart itself. It also picks up waste products from the body's cells. These waste products are removed as they're filtered through the kidneys, liver and lungs.

What is the heart's structure?

The heart has four chambers through which blood is pumped. The upper two are the right and left atria. The lower two are the right and left ventricles. Four valves open and close to let blood flow in only one direction when the heart beats:

1) The tricuspid valve is between the right atrium and right ventricle.
2) The pulmonary or pulmonic valve is between the right ventricle and the pulmonary artery.
3) The mitral valve is between the left atrium and left ventricle.
4) The aortic valve is between the left ventricle and the aorta.

Each valve has a set of flaps (also called leaflets or cusps). The mitral valve has two flaps. The others have three. Under normal conditions, the valves let blood flow in just one direction. Blood flow occurs only when there's a difference in pressure across the valves that causes them to open.

How does the heart pump blood?

The heart pumps blood by a highly organized sequence of contractions of its four chambers. The heart works as follows:

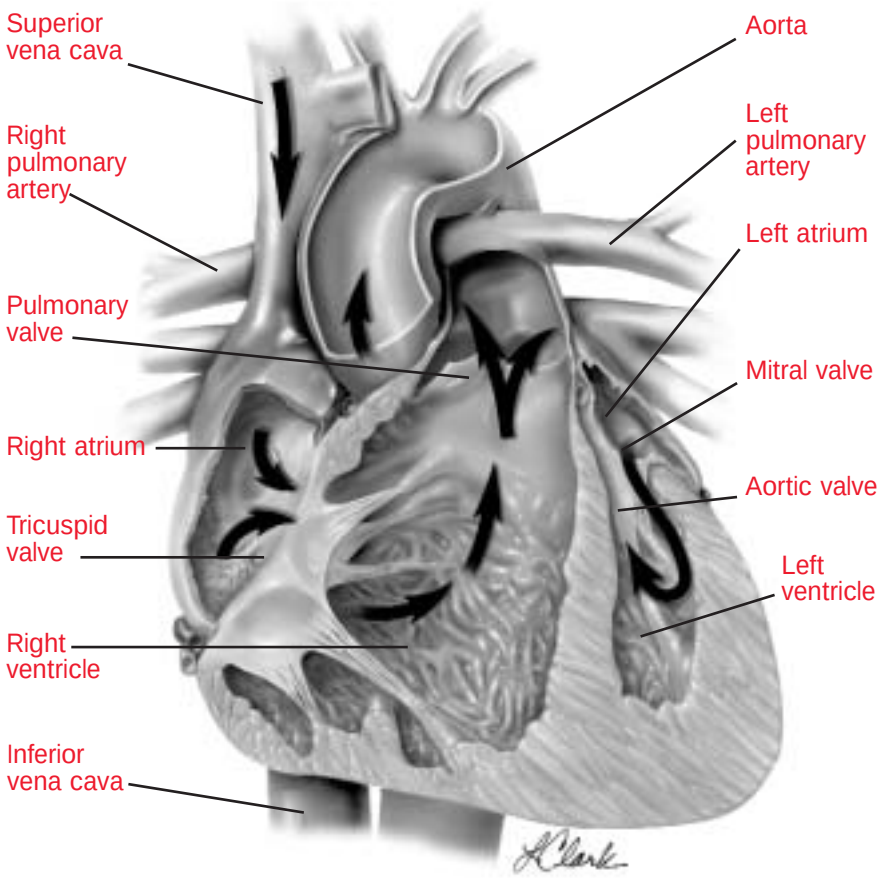
The right atrium receives blood from the veins. This blood carries little oxygen and lots of carbon dioxide. That's because it's returning from the body's tissues, where much of the oxygen was removed and the carbon dioxide added. Venous blood is darker than arterial blood because of the difference in dissolved gases.

While the heart is relaxed, venous blood flows through the open tricuspid valve to fill the right ventricle. An electrical signal starts the heartbeat by causing the atria to contract. This contraction "tops off" the filling of the ventricle. Shortly after the atrium contracts, the right ventricle contracts. As this occurs, the tricuspid valve closes and the partially deoxygenated blood is pumped through the pulmonary valve, into the pulmonary artery and on to the lungs. In the lungs the blood gives up its carbon dioxide and gets oxygen before returning to the left atrium. This newly oxygenated blood is bright red.

At the same time the right atrium contracts, the left atrium contracts, topping off the flow of oxygenated blood through the mitral valve and into the left ventricle. Then a split second later the left ventricle contracts, pumping the blood through the aortic valve, into the aorta and on to the body's tissues.

A heart's four chambers must beat in an organized way. This is governed by the electrical impulse. A chamber of the heart contracts when an electrical impulse moves across it. Such a signal starts in a small bundle of highly specialized cells in the right atrium — the sinoatrial node (SA node), also called the sinus node. A discharge from this natural "pacemaker" causes the heart to beat. This pacemaker generates electrical impulses at a given rate, but emotional reactions and hormonal factors can affect its rate of discharge. This lets the heart rate respond to varying demands.

The electrical impulses generated by the SA node move throughout the right and left atrium, causing the muscle cells to contract. Shortly after both atria have contracted, the electrical signal travels down specialized fibers throughout the ventricles. The signal's path causes the ventricles to contract together in a wringing motion, squeezing blood from them. The route of this electrical impulse is specific and produces the coordinated, sequential contraction of the heart's four chambers that's necessary for the heart to work properly.



Cross section of the heart.

Atherosclerosis: A Major Cause of Cardiovascular Disease

What is atherosclerosis?

Atherosclerosis comes from the Greek words *athero* (meaning gruel or paste) and *sclerosis* (hardness). It involves deposits of fatty substances, cholesterol, cellular waste products, calcium and other substances in the inner lining of an artery. This buildup is called plaque. It usually affects large and medium-sized arteries.

Atherosclerosis is a slow, complex disease that starts in childhood and often progresses when people grow older. In some people it progresses rapidly, even in their third decade. Many scientists think it begins with damage to the innermost layer of the artery. This layer is called the endothelium.

Three proven causes of damage to the arterial wall are 1) elevated levels of cholesterol and triglyceride in the blood, 2) high blood pressure and 3) tobacco smoke. Tobacco smoke greatly worsens atherosclerosis and speeds its growth in the coronary arteries, the aorta and arteries in the legs. (The coronary arteries bring blood to the heart muscle; the aorta is the large vessel through which the heart pumps blood that goes to the body.)

Because of the damage to the endothelium, fats, cholesterol, platelets, cellular debris, calcium and other substances are deposited in the artery wall. These may stimulate artery wall cells to produce other substances that result in further buildup of cells. These cells and the surrounding material (plaques) may become large enough to thicken the endothelium significantly. The artery's diameter shrinks and blood flow decreases, reducing the oxygen supply.

Plaques can also become unstable and rupture. Those that rupture cause a blood clot (thrombus) to form that can totally block blood flow in the artery. A blood clot that breaks off and travels to another part of the body is called an embolus. If a clot blocks a blood vessel that feeds the heart, it causes a heart attack. If it blocks a blood vessel that feeds the brain, it causes a stroke. And if blood supply to the arms or legs is reduced, it can cause difficulty walking and eventually gangrene.

What is cholesterol and where does it come from?

Cholesterol is a soft, fat-like substance. It's found in the blood and in all the body's cells. It's an important part of a healthy body because it's used to form cell membranes, some hormones and other needed tissues.

In atherosclerosis, plaque builds up in arteries over time and may become large enough to significantly reduce blood flow.

People get cholesterol in two ways. The body — mainly the liver — produces varying amounts, usually about 1,000 mg a day. Another 400 to 500 mg (or more) can come directly from foods. Foods from animals (especially egg yolks, meat, fish, poultry and whole-milk dairy products) contain it; foods from plants don't. Typically the body makes all the cholesterol it needs, so people don't need to consume it.

A high level of cholesterol in the blood is a major risk factor for coronary heart disease, heart attack and stroke. Hypercholesterolemia is the term for high levels of blood cholesterol.

How is cholesterol carried in the blood?

Cholesterol and other fats can't dissolve in blood. They have to be transported to and from the cells by special "carriers" called lipoproteins, which are created by the liver. Lipoproteins carry cholesterol and triglycerides, which are found in foods and made by the body.

The process starts when cholesterol and fats in food go to the intestine to be digested and absorbed. Chylomicrons are made in the intestinal wall. Chylomicrons are fatty particles containing mainly triglycerides, but also cholesterol, phospholipids and protein. When the chylomicrons enter the bloodstream, they contact with binding sites on capillaries. Many of their triglycerides break down and are released into the blood. The rest of the chylomicron (the "chylomicron remnant"), now richer in cholesterol, continues in circulation until it reaches the liver and is absorbed.

The liver then produces very-low-density lipoprotein (VLDL), the largest type of lipoprotein. VLDL carries triglycerides made in the liver from fatty acids, carbohydrates, alcohol and some cholesterol. VLDL is released into the bloodstream and, like chylomicrons, is carried to tissue capillaries. There the triglycerides are broken down and either used for energy or stored by muscle or fat cells.

After VLDL releases its triglycerides, what remains is a "VLDL remnant" called intermediate-density lipoprotein (IDL). Some IDL is removed from circulation by the liver; the rest is transformed into low-density lipoprotein (LDL).

What is LDL cholesterol?

LDL is the major cholesterol carrier in the blood. It carries about 60 to 80 percent of the body's cholesterol. Some of this cholesterol is used by tissues to build cells, and some is returned to the liver. If too much LDL cholesterol circulates in the blood, it can slowly build up in the walls of the arteries feeding the heart and brain. Together with other substances it can form plaque and contribute to atherosclerosis. That's why LDL cholesterol is often called "bad" cholesterol. Lower levels of LDL cholesterol reflect a lower risk of heart disease. A high level of LDL cholesterol (160 mg/dL and above) reflects an increased risk of heart disease. An optimal level of LDL cholesterol is less than 100 mg/dL, especially for people who have coronary heart disease or diabetes. (See page 38 for more information.)

What is HDL cholesterol?

About one-third to one-fourth of blood cholesterol is carried by high-density lipoprotein or HDL. It is a flat, disk-like particle produced primarily in the liver and intestines and released into the bloodstream. As VLDL and chylomicron particles release their triglycerides into the body's cells, fragments containing proteins, fats and cholesterol break away. Medical experts think HDL tends to carry cholesterol away from the arteries and back to the liver, where it's passed from the body.

Some experts believe HDL removes excess cholesterol from atherosclerotic plaques and thus slows their growth. Recent studies indicate that the antioxidant and anti-inflammatory properties of HDL also inhibit atherosclerosis.

HDL cholesterol is "good" cholesterol because a high level of it seems to protect against heart attack. The opposite is also true: People with a **low** HDL cholesterol level (less than 40 mg/dL) have a **higher** risk. A low level of HDL cholesterol also may raise stroke risk.

The levels of HDL cholesterol and LDL cholesterol in the blood are measured to evaluate the risk of atherosclerosis. That helps determine the risk of heart attack or stroke.

What is Lp(a) cholesterol?

Lp(a) is a genetic variation of plasma LDL. A high Lp(a) level is an important risk factor for developing atherosclerosis prematurely. The way an increased Lp(a) cholesterol contributes to disease isn't clear. The lesions in artery walls contain substances that may interact with Lp(a), leading to the buildup of lipids in atherosclerotic plaques.

What is known about HDL and triglyceride levels?

As a rule, women have higher HDL cholesterol levels than men. The female sex hormone estrogen tends to raise HDL cholesterol, which may help explain why premenopausal women are usually protected from developing heart disease. Estrogen production is highest during the childbearing years.

Triglyceride levels range from about 50 to 250 mg/dL, depending on age and sex. As people tend to get older, fatter or both, their triglyceride and cholesterol levels tend to rise. Women also tend to have higher triglyceride levels. Many people who have heart disease or diabetes have high triglyceride levels, called hypertriglyceridemia. A high triglyceride level combined with low HDL cholesterol or high LDL cholesterol seems to speed up atherosclerosis. The American Heart Association recommends keeping fasting triglyceride levels below 150 mg/dL.

What does research show?

Males and people with a family history of premature cardiovascular disease have an increased risk of atherosclerosis. These risk factors can't be controlled. Research shows the benefits of reducing the **controllable risk factors for atherosclerosis**:

- High blood cholesterol (especially LDL or "bad" cholesterol over 100 mg/dL)
- Cigarette smoking and exposure to tobacco smoke
- High blood pressure
- Diabetes mellitus
- Obesity
- Physical inactivity

Research to find ways to prevent or reverse atherosclerosis is now under way. One promising area of study is finding ways to control elevated levels of cholesterol and other fats in the blood.

One dramatic advance was the discovery of cell-surface receptors for LDL by 1985 Nobel laureates Drs. Joseph Goldstein and Michael Brown. These receptors bind LDL circulating through the bloodstream, allowing the LDL and its cholesterol to enter cells. Research has shown that when the amount of cholesterol within cells builds up, the number of these receptors on cell surfaces is reduced and blood levels of LDL increase. This can lead to more cholesterol being available for deposit in artery walls.

Another finding came from the Coronary Primary Prevention Trial (CPPT). It showed that lowering a high level of blood cholesterol reduces deaths from heart attack.

Another important advance was the development of a new class of cholesterol-lowering drugs. These compounds either block the synthesis of cholesterol by the body's cells, or force its elimination by preventing its absorption from the intestine.

Of course many fundamental questions remain. Medical scientists are continuing to search for answers by studying life at its most basic level — the cell.

Scientists are studying other ways in which platelets may play a role in atherosclerosis. For example, they're involved in forming a group of substances called prostaglandins, one of which may damage arteries. They also contain a substance called "platelet growth factor," which can stimulate the growth of smooth muscle cells. These cells are normally present in the artery wall. But their abnormal growth and increase is believed to be one of the earliest events in the atherosclerotic process.

One recent theory suggests that excess lipoproteins in the blood are trapped in the artery wall. When this happens and they accumulate, they become oxidized. That leads to "modified" lipoproteins that are rapidly taken up by smooth muscle cells. This, in turn, leads to foam cells forming and deposits of connective tissue cells and other elements.

The problem of high blood cholesterol isn't limited to adults.

Research also suggests that inflammation in the circulating blood may play an important role in triggering heart attacks and strokes. Inflammation is the body's response to injury, and blood clotting is often part of that response. Blood clots, as described above, can slow down or stop blood flow in the arteries.

Even though much more work needs to be done, scientists have found some answers. For instance, they've found a definite relationship between the amount of cholesterol in the bloodstream and coronary artery disease (blockage of arteries supplying blood to the heart muscle itself). A large body of scientific evidence shows that a diet high in saturated fats, *trans* fats and cholesterol can raise blood cholesterol levels and contribute to atherosclerosis.

The problem of high blood cholesterol isn't limited to adults. Millions of children also have elevated levels. That means they may be at higher risk of atherosclerosis and coronary heart disease later in life.

That's why the American Heart Association recommends that healthy adults and children aged 2 and older eat a diet that's low in saturated fats and cholesterol. Eating a proper diet helps reduce the risk of high blood cholesterol and thus the risk of heart attack.

What is homocysteine, and how is it related to cardiovascular risk?

Homocysteine is an amino acid in the blood. Too much homocysteine is related to a higher risk of coronary heart disease, stroke and peripheral vascular disease.

Some evidence suggests that homocysteine may contribute to atherosclerosis by damaging the inner lining of arteries and promoting blood clots. However, a direct causal link hasn't been found.

Folic acid and other B vitamins help break down homocysteine in the body. Homocysteine levels in the blood are strongly influenced by diet and genetic factors. Dietary folic acid and vitamins B_6 and B_{12} have the greatest effects. Several studies found that higher levels of B vitamins in the blood are related, at least partly, to lower concentrations of homocysteine. Other evidence shows that low levels of folic acid in the blood are linked with a higher risk of fatal coronary heart disease and stroke.

So far, no controlled treatment study has shown that folic acid supplements reduce the risk of atherosclerosis, or that taking these vitamins affects the development or recurrence of cardiovascular disease. Researchers are trying to determine how much folic acid, B_6 and/or B_{12} are needed to lower homocysteine levels.

Screening for blood homocysteine levels isn't widely available and may cost $200, which isn't currently covered by insurance. Still, it may be useful in patients with a personal or family history of cardiovascular disease, but who don't have the well-established risk factors (smoking, high blood cholesterol, high blood pressure, physical inactivity, obesity and diabetes).

Evidence is lacking for the benefit of lowering homocysteine levels, but **patients at high risk should be strongly advised to get enough folic acid and vitamins B_6 and B_{12} in their diet. They should eat at least five servings of fruits and green, leafy vegetables daily.** Some grain products are now fortified with folic acid.

This is just one possible risk factor. A physician taking any type of nutritional approach to reducing risk should consider a person's overall risk factor profile and total diet.

High Blood Pressure

What is blood pressure?

Blood pressure results from two forces. One is created by the heart as it pumps blood into the arteries and through the circulatory system. The other is the force of the arteries as they resist the blood flow.

When the heart pumps, it forces blood through the large arteries into the smaller arteries, the arterioles. The arterioles can contract or expand (dilate), altering the resistance to blood flow. This affects the amount of blood flow and the blood pressure. If the arterioles contract, the resistance to blood flow increases, which reduces blood flow and increases blood pressure. Expansion (dilation) of the arterioles has the opposite effect. It follows that changes in the inner diameter of the arterioles are important in regulating blood flow and determining blood pressure. If the arterioles stay constricted, they can cause high blood pressure (hypertension).

How is blood pressure measured?

Blood pressure is measured by a quick, painless test using a medical instrument called a sphygmomanometer. A rubber cuff is wrapped around a person's upper arm and inflated. It compresses a large artery in the arm, momentarily stopping the blood flow.

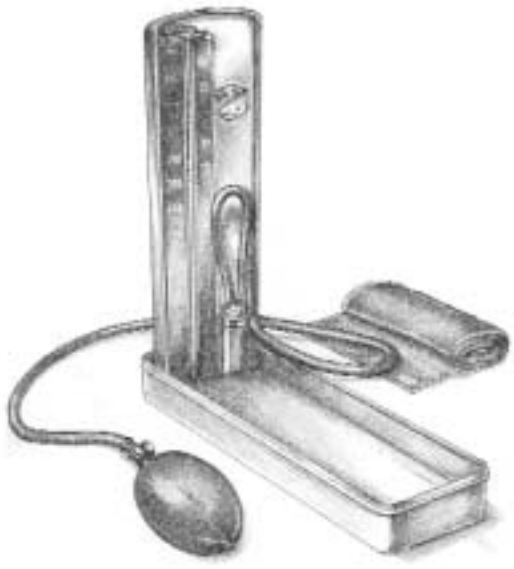
Next, air in the cuff is released, and the person measuring the blood pressure listens with a stethoscope. When the blood starts to pulse through the artery, it makes a sound. Sounds continue to be heard until the pressure in the artery exceeds the pressure in the cuff.

The person listening and watching the sphygmomanometer gauge records two measurements. Systolic pressure (the higher number) is the pressure of the blood flow when the heart beats (the pressure when the first sound is heard). Diastolic pressure is the pressure between heartbeats (the pressure when the last sound is heard). Blood pressure is measured in millimeters of mercury, which is abbreviated mm Hg.

The harder it is for blood to flow, the higher the numbers will be.

What is high blood pressure?

High blood pressure in an adult is defined as a systolic pressure of 140 mm Hg or higher and/or a diastolic pressure of 90 mm Hg or higher for an extended time. A systolic pressure of 120 to 139 mm Hg or a diastolic pressure of 80 to 89 mm Hg is "prehypertension" and needs to be watched carefully. Blood pressure of less than 120 over 80 mm Hg is considered normal for adults.



Blood pressure can be measured quickly and painlessly.

Blood pressure classification for adults age 18 and older

Blood Pressure Category	Systolic (mm Hg)		Diastolic (mm Hg)
Normal*	less than 120	and	less than 80
Prehypertension	120–139	or	80–89
Hypertension, Stage 1	140–159	or	90–99
Hypertension, Stage 2	160 or higher	or	100 or higher

*Unusually low readings should be evaluated for clinical significance.

Source: Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7 Express) NIH Publication No. 03-5233, May 2003

Hypertension is the medical term for high blood pressure. It doesn't refer to being tense, nervous or hyperactive. A person can be calm and relaxed and still have high blood pressure. High blood pressure usually has no symptoms. In fact, many people have high blood pressure for years without knowing it. That's why it's called the "silent killer."

The only way to detect high blood pressure is to have a doctor or other qualified health professional check for it. It's a dangerous disease, not to be taken lightly!

Why is high blood pressure harmful?

High blood pressure causes the heart to work harder than normal. Both the heart and arteries are then more prone to injury. High blood pressure increases the risk of heart attacks, strokes, kidney failure, eye damage, congestive heart failure and atherosclerosis.

If high blood pressure isn't treated, the heart may have to work harder and harder to pump enough blood and oxygen to the body's organs and tissues. A heart forced to work harder than normal for a long time tends to enlarge and weaken. A slightly enlarged heart may work well, but one that's enlarged a lot has a hard time doing its job.

High blood pressure also hurts arteries and arterioles. Over time they become scarred, hardened and less elastic. This may occur as people age, but high blood pressure accelerates this process, probably because it speeds atherosclerosis.

Arterial damage is bad because hardened or narrowed arteries may not be able to supply enough blood to the body's organs. And if the organs don't get enough oxygen and nutrients, they can't work properly. Another risk is that a blood clot may lodge in an artery narrowed by fatty buildups, shutting off normal blood supply to part of the body.

What causes high blood pressure?

In 90–95 percent of cases, the cause is unknown. This type of high blood pressure is called essential hypertension. Fortunately, though scientists don't fully understand the causes of this disease, they've developed both non-drug and drug treatments that treat it effectively. They've also identified some factors that contribute to higher blood pressure. These are arteriosclerosis (hardening of the arteries), thickening (hypertrophy) of the artery wall, and excess contraction of the arterioles (small arteries).

In the remaining 5–10 percent of cases, high blood pressure results from a recognizable underlying problem. This is called secondary hypertension. Some possible causes are a kidney abnormality, tumor of the adrenal gland or a congenital defect of the aorta. When the root cause is corrected, blood pressure usually returns to normal.

What can be done about high blood pressure?

Dietary and lifestyle changes help control high blood pressure. These changes are recommended for people with prehypertension.

- The American Heart Association advises people to eat lots of **fruits, vegetables, and fat-free and low-fat dairy products**. Such diets are rich in potassium, calcium, magnesium and protein, and low in saturated fat, total fat and cholesterol.
- Some people can lower their blood pressure by reducing **sodium** (salt) in their diet. This means avoiding salty foods and cutting down on salt in cooking and at the table.
- Drinking too much **alcohol** (more than one ounce of pure alcohol, or two drinks per day for men and one drink per day for women) raises blood pressure in some people and should be restricted. Alcoholic drinks are high in non-nutritious calories, so people trying to lose weight shouldn't drink alcohol.
- Many people who have high blood pressure are **overweight or obese**. Obesity is a risk factor for both high blood pressure and heart disease. When people lose weight, their blood pressure often drops, too.
- Regular **physical activity** helps control weight and lower blood pressure.

For some people, losing weight, reducing sodium and other lifestyle changes won't lower high blood pressure as much as needed. These people will probably need to take medication.

Medicines called **antihypertensives** lower high blood pressure. Some, called **diuretics**, rid the body of excess fluids and salt (sodium). Others, called **beta blockers**, reduce the heart rate and the heart's output of blood.

Angiotensin converting enzyme (ACE) **inhibitors, angiotensin II receptor blockers** and **calcium antagonists (calcium channel blockers)** are drugs used to treat high blood pressure. ACE inhibitors interfere with the body's production of angiotensin, a chemical that causes the arterioles to constrict. Angiotensin II receptor blockers block the effects of angiotensin. Calcium antagonists can slow the heart rate and relax blood vessels.

Another class of antihypertensives is called **sympathetic nerve inhibitors**. Sympathetic nerves go from the brain to all parts of the body, including the arteries and arterioles. They can cause the arterioles to

constrict, raising blood pressure. These drugs reduce blood pressure by inhibiting these nerves from constricting blood vessels.

Vasodilators are another useful group of drugs. They can cause the muscle in blood vessel walls to relax, allowing the vessel to dilate (widen). These drugs are especially effective in the arterioles.

In most cases these drugs lower blood pressure. But quite often people respond very differently to them. That's why most patients must go through a trial period to find out which medications work best with the fewest side effects.

People with high blood pressure should do two things:

1) Follow your healthcare provider's instructions.

2) Stay on your medication.

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2) Stay on your medication.

What factors contribute to high blood pressure?

Because medical science doesn't understand why most cases of high blood pressure occur, it's hard to say how to prevent it. Several factors may contribute to it.

- **Heredity.** People whose parents have high blood pressure are more likely to develop it than those whose parents don't.
- **Race.** African Americans are also more likely to have high blood pressure than Caucasians are.
- **Male sex.** Men have a greater risk of high blood pressure than women until age 55, when their respective risks are similar. At age 75 and older, women are more likely to develop high blood pressure than men are.
- **Increasing age.** Blood pressure tends to increase with age. Older people are more likely to have high blood pressure.
- **Sodium (salt) sensitivity.** Most Americans consume much more salt (sodium) than their bodies need. Heavy sodium consumption leads to high blood pressure in some people.
- **Obesity and overweight.** Studies have shown that body weight, changes in body weight over time, and skinfold thickness are related to changes in blood pressure levels. People who are overweight are more likely to have high blood pressure.
- **Sedentary or inactive lifestyle.** The American Heart Association recommends at least 30 minutes of moderate to vigorous physical activities on most days.
- **Heavy alcohol consumption.** Experts recommend that drinkers limit themselves to no more than 1–2 drinks per day.
- **Diabetes mellitus, gout** and **kidney disease.** People with these conditions are more likely to have high blood pressure.
- **Pregnancy.** Some women who've never had high blood pressure develop it during pregnancy, especially in the last three months. Both mother and baby may be in danger if it's not treated.

- Use of some **oral contraceptives**. A woman taking oral contraceptives is more likely to develop high blood pressure if she's overweight, has had high blood pressure during pregnancy, has a family history of high blood pressure or has mild kidney disease.
- Some other **medications** also can raise blood pressure and/or interfere with the effectiveness of antihypertensive drugs. People with high blood pressure should tell their doctor all of the prescribed and over-the-counter medicines they're taking. These include such drugs as steroids, non-steroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen, nasal decongestants and other cold remedies, diet pills, cyclosporine, erythropoietin, tricyclic antidepressants, and monoamine oxidase inhibitors.

What about low blood pressure?

Within limits, the lower your blood pressure reading is, the better. In most people, blood pressure isn't too low until it causes symptoms such as lightheadedness or fainting. In certain conditions or disease states, blood pressure can become too low, as in these examples:

- certain nervous system disorders (orthostatic hypotension, autonomic failure)
- endocrine disorders (parathyroid disease)
- heart attack
- prolonged bed rest
- fainting (syncope)
- decreases in blood volume due to severe bleeding (hemorrhage) or dehydration
- effects of certain drugs
- severe infections (sepsis)
- shock

Normal blood pressure for healthy adults is less than 120/80 mm Hg. However, unusually low readings should be evaluated to rule out medical causes.

Angina Pectoris and Heart Attack

What is angina pectoris?

Angina pectoris is the medical term for chest pain or discomfort due to coronary heart disease. Angina is a symptom of a condition called myocardial ischemia. It occurs when the heart muscle (myocardium) doesn't get as much blood (hence as much oxygen) as it needs. This usually happens because one or more of the heart's arteries (those that supply blood to the heart muscle) is narrowed or blocked. Insufficient blood supply is called ischemia.

Typical angina is uncomfortable pressure, fullness, squeezing or pain in the center of the chest. The discomfort also may be felt in the neck, jaw, shoulder, back or arm. Many types of chest discomfort aren't related to angina, such as that caused by acid reflux (heartburn) and lung infection or inflammation.

Angina often occurs when the heart needs more blood. Running to catch a bus, for example, could trigger an attack of angina, while walking might not. Angina may happen during physical exercise, strong emotions or extreme temperatures. Some people, such as those with a coronary artery spasm, may have angina when they're resting. (See below, unstable angina, Prinzmetal's or variant angina pectoris.)

Angina also can occur in people with valvular heart disease, hypertrophic cardiomyopathy (an enlarged heart) or uncontrolled high blood pressure — but these cases are fairly rare.

Angina is a sign that someone is at higher risk of heart attack, cardiac arrest and sudden cardiac death.

What is stable angina?

People with stable angina (or chronic stable angina) have episodes of chest discomfort that are usually predictable. They occur with exertion (such as running to catch a bus) or under mental or emotional stress. Normally the chest discomfort is relieved with rest and/or nitroglycerin.

People with episodes of chest discomfort should see their physician for an evaluation. The doctor will evaluate the person's medical history and risk factors, conduct a physical exam, order a chest X-ray and take an electrocardiogram (ECG or EKG). Some people will also need an exercise ECG (stress test), an echocardiogram or other tests to complete the diagnosis.

What is unstable angina?

People with unstable angina have unexpected chest pain that usually occurs while at rest. The discomfort may be more severe and prolonged than typical angina or be the first time a person has angina. The most common cause is reduced blood flow to the heart muscle due to coronary arteries narrowed by atherosclerosis. An artery may be abnormally constricted or partially blocked by a blood clot. Inflammation, infection and secondary causes also can lead to unstable angina. In variant or Prinzmetal's angina (see the next section), a form of unstable angina, coronary artery spasm is the cause.

Unstable angina is an acute coronary syndrome and should be treated as an emergency. People with new, worsening or persistent chest discomfort should be evaluated in a hospital emergency department or "chest pain unit" and monitored carefully. They're at increased risk for

- acute myocardial infarction (heart attack).
- severe cardiac arrhythmias. These may include ventricular tachycardia and fibrillation.
- cardiac arrest leading to sudden death.

What is variant angina pectoris (Prinzmetal's angina)?

Variant angina pectoris is also called Prinzmetal's angina. Unlike typical angina, it nearly always occurs when a person is at rest. It doesn't follow a period of physical exertion or emotional stress, either. Attacks can be very painful and usually occur between midnight and 8 a.m. It's associated with acute myocardial infarction (heart attack), severe cardiac arrhythmias (including ventricular tachycardia and fibrillation), and sudden cardiac arrest.

Variant angina is due to coronary artery spasm. About two-thirds of people with it have severe coronary atherosclerosis in at least one major vessel. The spasm usually occurs very close to the blockage.

What are ischemic heart disease and silent ischemia?

Ischemia is a condition where the flow of blood, and therefore oxygen, to a part of the body is restricted. Cardiac ischemia refers to lack of blood flow and oxygen to the heart muscle.

Ischemic heart disease refers to heart problems caused by narrowed heart arteries. When arteries are narrowed, less blood and oxygen reaches the heart. This is also called **coronary artery disease** and **coronary heart disease**. This can lead to heart attack. Ischemia often causes chest pain or discomfort.

As many as 3 to 4 million Americans may have ischemic episodes without knowing it. These people have ischemia without pain ("silent ischemia"). They may have a heart attack with no prior warning. In addition, people with angina also may have undiagnosed episodes of silent ischemia. An exercise test or a 24-hour portable monitor of the electrocardiogram (Holter monitor) are two of the various tests used to diagnose this problem.

What are angiogenesis and collateral circulation?

Angiogenesis is the creation of blood vessels. The body creates small blood vessels called "collaterals" to help compensate for reduced blood flow. Often this reduced blood flow results when a major artery, such as one in the heart, is blocked.

Collateral vessels normally aren't open. However, they grow and enlarge in some people with coronary heart disease or other blood vessel disease. While everyone has collateral vessels, at least in microscopic form, they don't open in all people.

When a collateral vessel enlarges, it lets blood flow from an open artery to either an adjacent artery or further downstream on the same artery. In this way, collateral vessels grow and form a kind of "detour" around a blockage. This collateral circulation provides alternate routes of blood flow to the heart in cases when it isn't getting the blood supply it needs (myocardial ischemia).

Scientists are trying to use gene therapy to copy this natural process by delivering genetic material to the affected areas. Then the body will take over healing itself. For patients with peripheral (leg) or coronary (heart) arteries damaged by vascular disease, the ability to "turn on" the angiogenesis gene could be a potentially powerful way to "grow" new blood vessels.

Several scientific studies exploring this possibility are now under way. The technique of delivering gene therapy to the heart is being developed. The genetic material to be used is also being tested.

What drugs are used to treat angina?

Angina pectoris can be treated with drugs that affect the heart muscle's blood supply or the heart's demand for oxygen. Drugs that affect blood supply are coronary vasodilators; they cause blood vessels to relax. When this happens, the opening inside the vessels (the **lumen**) gets bigger. Then blood flow improves, letting more oxygen and nutrients reach the heart muscle.

Nitroglycerin is the drug most often used. It relaxes the veins and the coronary arteries. By relaxing the veins, it reduces the amount of blood that returns to the heart and eases the heart's workload. By relaxing the coronary arteries, it increases the heart's blood supply.

The heart's demand for oxygen also can be modified with a drug to reduce blood pressure (see pages 10–11). This lowers resistance to flow and reduces the heart's workload and need for oxygen. Drugs that slow the heart rate have a similar effect.

What procedures are used to treat angina?

Invasive techniques that improve the heart and the heart's blood supply also may be used. One of these is **percutaneous transluminal coronary angioplasty**. It's also known as PTCA, angioplasty, balloon dilation or balloon angioplasty. Two newer techniques are **laser angioplasty** and **atherectomy**. These are often followed by a **stent procedure**. An open-heart procedure is **coronary artery bypass graft surgery**. (These procedures are discussed on pages 19–21.)

Before performing any of these procedures, a doctor must find the blocked part of the coronary artery. This requires **coronary arteriography**, which is done during **cardiac catheterization**. In this procedure a doctor guides a thin, flexible, plastic tube (a catheter) through an artery in the arm or leg and into the coronary arteries. Then the doctor injects a liquid dye visible in X-rays through the catheter. High-speed X-ray movies show the liquid as it flows through the arteries. Doctors can identify blockages by tracing the flow.

Some newer diagnostic tools evaluate how well the heart works. These tests may be done before or after a heart attack. Some are still somewhat experimental and are limited to larger medical centers.

Computer Imaging Tests Performed in Many Medical Centers

1. Radionuclide Imaging or Radionuclide Angiography (includes such tests as thallium test, MUGA scan or acute infarct scintigraphy)

These tests involve injecting radioactive substances called radionuclides into the bloodstream. Computer-generated pictures can then find them in the heart. These tests show how well the heart muscle is supplied with blood, how well the heart's chambers are working, or identify a part of the heart damaged by heart attack.

Radionuclide angiography also can be used as a nuclear brain scan. In it radioactive compounds are injected into a vein in the arm, and a machine similar to a Geiger counter creates a map showing their uptake into different parts of the head. The pictures show how the brain works rather than its structure. This test can detect blocked blood vessels and areas where the brain is damaged.

2. Magnetic Resonance Imaging (MRI), also called Nuclear Magnetic Resonance (NMR) Imaging

This test uses powerful magnets to look inside the body. Computer-generated pictures can show the heart muscle, identify damage from a heart attack, diagnose certain congenital cardiovascular defects and evaluate disease of larger blood vessels such as the aorta. It can outline the affected part of the brain and help define problems created by stroke. Unlike radiographic imaging methods,

- It's non-ionizing and has no known biological hazards.
- It can produce high-resolution images of the heart's chambers and large vessels without the need for contrast agents.
- It's intrinsically three-dimensional.
- It produces images of cardiovascular structures without interference from adjacent bone or air.
- It has high tissue contrast.

MRI is an accepted technique for evaluating diseases of the aorta such as dissection, aneurysm and coarctation; diseases of the pericardium such as constrictive pericarditis or hematoma; congenital cardiac lesions before or after surgical repair; heart muscle diseases, including those affecting the right ventricle such as dysplasia; and cardiac masses such as intracardiac tumor or invasive lung malignancy.

Other proven but less-common uses of MRI include evaluating cardiac chamber morphology such as ventricular mass; global or regional ventricular function; and valve regurgitation.

Other potential applications are now under active investigation. These include evaluating coronary artery anatomy and flow; evaluating myocardial blood flow; assessing myocardial viability with pharmacologic stress; and assessing myocardial metabolism by spectroscopic techniques.

In summary, MRI provides clinically relevant anatomic and functional information noninvasively and with minimal risk, if the well-known contraindications (such as pacemakers) and potential hazards (such as attraction of metallic objects) are avoided.

3. Digital Cardiac Angiography (DCA) or Digital Subtraction Angiography (DSA)

This modified form of computer imaging records pictures of the major blood vessels to the heart or brain. It lets a doctor know if there are any blockages, how severe they are, and what can be done about them. In this test, a dye is injected into a vein in the arm, and an X-ray machine quickly takes a series of pictures of the chest or head and neck.

4. Cardiac Computed Tomography (CT), Computerized Axial Tomographic Scan (CAT scan)

X-ray computed tomography, including conventional, helical and electron-beam ("Ultrafast®") forms, provides cross-sectional images of the chest, including the heart and great vessels. In general, cardiac tomography (also called CT scan and coronary artery scanning) is useful to evaluate aortic disease (such as aortic dissection), cardiac masses and pericardial disease.

CT provides clinically relevant anatomic and functional information. It's also relatively noninvasive and has very low short- and long-term risks (if the well-known potential hazards are avoided).

Computerized axial tomographic scan is also used to examine how the brain looks, functions and gets its blood supply. This test can outline the affected part of the brain and help define the problem a stroke creates.

5. Electron-Beam Computed Tomography (EBCT or Ultrafast® CT)

EBCT is an especially fast form of X-ray imaging technology. It's particularly useful to

- evaluate bypass graft patency, intra- and congenital cardiac lesions, and
- quantify right and left ventricular muscle mass, chamber volumes, and systolic and diastolic function (such as cardiac output and ejection fraction).

Electron-beam CT also can measure calcium deposits in the coronary arteries. The amount of calcium EBCT detects is related to the amount of underlying coronary atherosclerosis.

A high coronary calcium score, derived from EBCT scans of the coronary arteries, predicts the occurrence of cardiac events. These include fatal and nonfatal heart attacks or the need for coronary bypass surgery or coronary (balloon) angioplasty in the next one or two years. A negative calcium score implies a very low risk for obstructing coronary lesions and coronary events. It has a high negative predictive value for coronary events.

The increased predictive value of EBCT of the coronary arteries compared to traditional risk factor assessment isn't yet completely defined.

EBCT isn't a substitute for cardiac catheterization. EBCT measurement of coronary calcium isn't helpful for patients who've already had a heart attack or undergone coronary bypass surgery or coronary angioplasty.

6. Cardiac Positron Emission Tomography (PET)

Positron emission tomography of the heart allows heart tissue function to be studied and quantified. Its use in research has provided novel observations in cardiac physiology and pathophysiology. PET combines

- tomographic imaging with radionuclide tracers of blood flow, metabolism and receptors, and

- tracer kinetic principles for non-invasively quantifying regional myocardial blood flow, substrate fluxes, biochemical reaction rates and neural control.

Clinical studies suggest an important role for PET in diagnosing patients, describing disease and developing treatment strategy. Two areas of clinical application have emerged:

- PET is a highly accurate, noninvasive way to detect, localize and describe coronary artery disease that impairs blood flow to the myocardium (heart muscle).
- PET accurately identifies injured but viable myocardium, such as reversible ventricular dysfunction.

Technological improvements have occurred in PET scanners, cyclotron production of tracer labels and radiotracer synthesis. These have greatly enhanced the performance of cardiac PET studies, which now appear to be feasible. Cardiac PET studies also can be done without an on-site cyclotron. They use generator-produced isotopes such as rubidium-82 and/or tracers of metabolism produced off-site.

7. Single Photon Emission Computed Tomography (SPECT)

SPECT of the heart is a well-established nuclear imaging technique. It involves taking a series of pictures around the chest after injecting a radioactive tracer into the blood. Then computer graphics are used to create images of slices through the heart. This technique has been applied to the heart for myocardial perfusion (blood flow) imaging with agents like thallium-201 and the technetium-based myocardial perfusion tracers. These agents are injected either at rest or with exercise or pharmacologic stress.

Cardiac SPECT was introduced for myocardial blood flow imaging to overcome some of the limitations of planar imaging and to improve how blood flow defects are localized and quantified. Cardiac SPECT makes it easier to detect and localize myocardial blood flow defects at rest and during stress. SPECT can localize coronary artery disease and assess the extent and severity of blood flow abnormalities better than planar imaging. As a result, SPECT imaging is now widely used in nuclear cardiology laboratories across the country.

Several large, published studies have demonstrated the quantitative methods of interpretation. When SPECT is used to image the technetium-based myocardial blood flow tracers, global and regional function of the ventricle can be obtained in addition to regional perfusion. SPECT imaging of the heart also can be used along with newer agents that evaluate metabolism, but these applications are now investigational.

In summary, SPECT myocardial blood flow imaging is a well-established, clinically useful way to diagnose coronary artery disease and manage patients who have it.

How is variant angina or Prinzmetal's angina treated?

Calcium antagonists are extremely effective in preventing coronary spasms of variant or Prinzmetal's angina. These drugs, along with nitrates, are the mainstays of treatment. Prinzmetal's angina tends to be cyclic, appearing for a time, then going away. Because of this, after six to 12 months of treatment, the calcium antagonists may be gradually reduced. In some cases PTCA is used, when a blockage is thought to cause the spasm.

What is balloon angioplasty or PTCA?

PTCA is an established and effective therapy used to dilate (widen) narrowed coronary arteries. It's a less traumatic and less expensive alternative to bypass surgery for some patients with coronary artery disease.

A doctor inserts a thin, flexible plastic tube called a **guiding catheter** into the narrowed part of the coronary artery. Next, a very thin wire is threaded across the blockage. Over this wire, a thin, expandable balloon is passed to the blockage. Then the balloon is inflated. The balloon "opens up" the blockage by compressing the plaque and stretching the artery open. Now blood can flow more easily. Then the balloon is deflated and the wire and catheter are removed. Typically balloon angioplasty reduces an artery blockage to less than 20 to 30 percent narrowed.

In 10 to 20 percent of patients who've had PTCA, the dilated part of the artery narrows again within six months after the procedure **(restenosis)**. They may require either another PTCA or coronary artery bypass surgery.

Complications from angioplasty can occur in some patients, but major complications are unusual. A very small percentage of patients needs emergency coronary bypass surgery when the procedure fails to open the artery.

Many advances have been made in this procedure. New devices and medications increase long-term success and decrease complications. One of these advances is the **coronary stent**. Stents are being used more and more to prevent renarrowing.

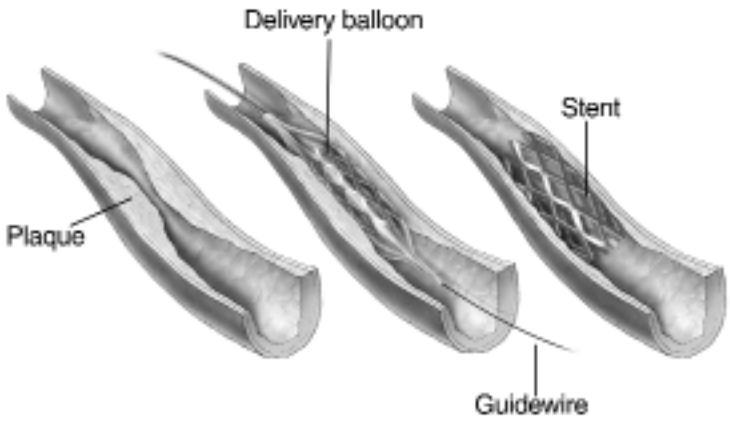
In PTCA, a balloon-tipped catheter is placed where the artery is narrowed. The balloon is inflated, compressing the plaque.

What is a stent procedure?

This procedure uses a wire mesh tube (a stent) to prop open an artery that's recently been cleared using angioplasty. The stent is collapsed to a small diameter and put over a balloon catheter. It's then moved into the area of the blockage. When the balloon is inflated, the stent expands, locks in place and forms a scaffold. This holds the artery open. The stent stays in the artery permanently, holds it open, improves blood flow to the heart muscle and relieves symptoms (usually chest pain).

This is a common procedure. About 70–90 percent of PTCA procedures involve stents. A stent may be used instead of — or along with — angioplasty. Stent use depends on certain features of the artery blockage. This includes the size of the artery and where the blockage is.

In certain patients, stents reduce the renarrowing that occurs after balloon angioplasty or other procedures using catheters. Stents also help restore normal blood flow and keep open an artery that's been torn or injured by a balloon catheter.



The stent procedure uses a wire mesh tube (a stent) to prop open an artery that's recently been cleared using angioplasty or atherectomy.

Reclosure (restenosis) is also a problem with the stent procedure. In recent years doctors have used new types of stents. Some of these are covered with drugs that help keep the blood vessel from reclosing. These new stents have shown promise for improving the long-term success of this procedure.

What is laser angioplasty?

This is a technique used to open coronary arteries blocked by plaque. A catheter with a laser at its tip is inserted into an artery. Then it's advanced through the artery to the blockage. When the laser is in position, it emits pulsating beams of light that vaporize the plaque.

This procedure has been used alone and with balloon angioplasty. The first laser device (the "eximer laser") for opening coronary arteries was approved by the Food and Drug Administration in 1992. It's used in many major U.S. medical centers.

What is atherectomy?

Atherectomy is a procedure to remove plaque from arteries. Coronary atherectomy removes plaque from the arteries supplying blood to the heart muscle.

As in angioplasty, an ultra-thin wire is threaded through a special catheter into the coronary artery and across the blockage. Several devices then may be used. One of them, a special "burr," is like a drill bit coated with very fine diamonds. It's threaded over this wire to the blocked area and gently advanced into the plaque. Rotating at high speed, the burr grinds the plaque into very tiny pieces.

In directional coronary atherectomy (usually called "DCA"), a small rotating cutter "shaves off" pieces of the blockage. Another device used is a laser catheter that vaporizes the plaque. See "What is laser angioplasty?" on page 20.

After an atherectomy, the artery is usually opened more with balloon angioplasty. A stent is often inserted after that.

What is coronary artery bypass surgery?

This is a type of heart surgery. It reroutes, or "bypasses," blood around clogged arteries and improves the supply of blood and oxygen to the heart. It's sometimes called CABG (for coronary artery bypass graft) or "cabbage."

In bypass surgery, surgeons take a blood vessel from another part of the body and make a detour around the blocked part of the coronary artery. An artery may be detached from the chest wall and the open end attached to the coronary artery below the blocked area. Or a segment of a long vein in the leg may be taken and used. One end is sewn onto the large artery leaving the heart (the aorta). The other end of the vein is attached or "grafted" to the coronary artery below the blocked area. Either way, blood can use this new channel to flow freely to the heart muscle.

Whether to use coronary artery bypass surgery, atherectomy, balloon angioplasty, a stent or the laser depends on where the blockage is, how many blockages there are, the extent of the blockage(s) and other factors. Patients should discuss their options with their physicians to determine the best approach for their situation.

If you have angina, you should also modify your other heart attack risk factors. This means you must not smoke. Work with your healthcare provider to control high blood pressure, high blood cholesterol and diabetes. Modify your diet to reduce the saturated fat, *trans* fat and cholesterol you eat. Finally, try to walk or perform other physical activity on most days to help maintain a healthy weight and reduce your overall risk.

What are warning signs of important changes in angina?

If you have stable angina and start getting chest pain more easily, more often, at night, etc., this could signal an important change in your condition. See your doctor right away.

What is a heart attack?

The medical term for heart attack is **myocardial infarction**. A heart attack is also sometimes called a **coronary thrombosis** or **coronary occlusion**.

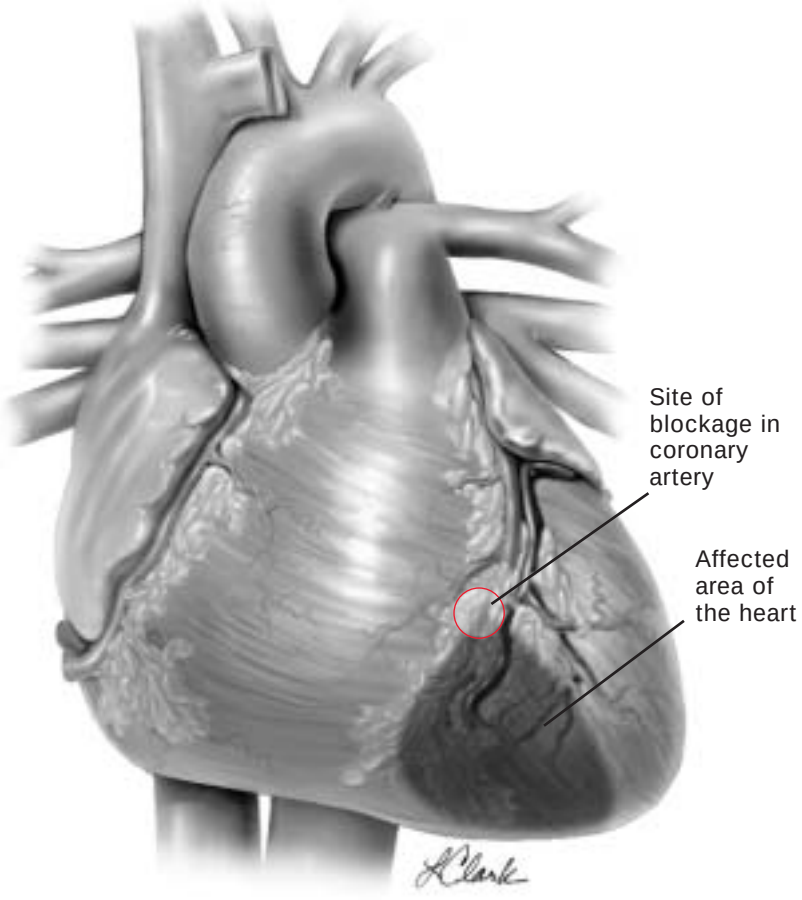
Heart attacks (also called coronary attacks) result from coronary heart disease (CHD) — disease of the blood vessels that feed the heart muscle. Coronary artery disease (CAD) and ischemic heart disease are other names for coronary heart disease.

A heart attack occurs when the blood supply to part of the heart muscle (the myocardium) is severely reduced or stopped. The reduction or stoppage happens when one or more of the coronary arteries supplying blood to the heart muscle is blocked. This is usually caused by atherosclerosis, which leads to a buildup of plaque (deposits of fat-like substances) inside the artery wall.

The plaque can eventually burst, tear or rupture, creating a “snag” where a blood clot forms and blocks the artery. This leads to a heart attack.

If the blood supply is cut off for more than a few minutes, muscle cells suffer permanent injury and die. This can kill or disable someone, depending on how much heart muscle is damaged.

Sometimes a coronary artery temporarily contracts or goes into spasm. When this happens the artery narrows and blood flow to part of the heart muscle decreases or stops. Scientists aren’t sure what causes a spasm. But a spasm can occur in normal-appearing blood vessels as well as vessels partly blocked by atherosclerosis. A severe spasm can cause a heart attack.



When the heart’s blood supply is blocked, a heart attack can occur.

How is a coronary (heart) attack diagnosed?

Heart attacks often have warning signs. These are outlined on page 35.

To diagnose a heart attack, a physician must study the results of several tests. Evaluating people with chest discomfort and a potentially life-threatening heart problem commonly includes four steps:

1) Medical history, including relevant risk factors for coronary atherosclerosis
2) Physical examination
3) Electrocardiogram (ECG or EKG) to look for abnormalities caused by damage to the heart
4) Blood tests to detect abnormal levels of certain enzymes in the bloodstream

Blood tests confirm (or refute) suspicions raised in the early stages of evaluation that may occur in an emergency room, intensive care unit or urgent care setting. These tests are sometimes called heart damage markers or cardiac enzymes.

A blood test often used to confirm heart muscle damage is the creatine kinase, or **CK** for short. A small fraction of the CK enzyme, **CK-MB**, is often measured, too. CK-MB shows an increase above normal in a person’s blood test about six hours after a heart attack starts. It reaches its peak level in about 18 hours and returns to normal in 24 to 36 hours. The peak level and the return to normal can be delayed in people who’ve had a large heart attack, especially if they don’t get early and aggressive treatment.

Tests have been developed to measure the level of other cardiac muscle proteins called troponins, specifically troponin T (cTnT) and troponin I (cTnI). These proteins control the interactions between actin and myosin, which contracts or squeezes the heart muscle. Troponins specific to heart muscle have been found, allowing the development of blood tests (assays) that can detect heart muscle injury with great sensitivity and specificity. Normally the level of cTnT and cTnI in the blood is very low. It increases substantially within four to six hours (on average) of muscle damage. It peaks at 10 to 24 hours and can be detected for a week or more after.

Several studies have identified a measurable relationship between cardiac troponin levels and long-term outcome after an episode of chest discomfort. They suggest that these tests may be particularly useful to evaluate levels of risk. In other words, it's possible that the results of a troponin test could be used to identify people at low risk or high risk for later, serious heart problems. It remains to be proven whether more cost-effective methods of treatment and, eventually, a better outcome will result from routine troponin testing.

How is a heart attack treated?

When a heart attack occurs, it's critical to recognize the signs and respond **immediately**. **Time is critical.** When a coronary artery is blocked, the heart muscle doesn't die instantly. But damage increases the longer an artery stays blocked. Delay may increase the damage to the heart and reduce the chance of survival. It also lessens the chance of preserving heart muscle. This increases the risk of disability.

Anyone who has the warning signs of a heart attack should be rushed to the nearest hospital with 24-hour emergency cardiovascular care. People who pass out before reaching the emergency room may receive cardiopulmonary resuscitation (CPR).

Most communities have an emergency cardiovascular care system that can respond quickly. This prompt care dramatically reduces heart damage. In fact, 88 percent of heart attack survivors under age 65 can return to their usual work. Prompt care isn't the only reason so many heart attack survivors recover so quickly, but it's an important one.

If a victim gets to an emergency room fast enough, reperfusion therapy may be done. This increases blood supply to the heart muscle. It's done with drugs to dissolve clots, balloon angioplasty (PTCA), or surgery. The sooner any of these techniques occur, the more likely the patient will benefit.

Thrombolysis involves injecting a clot-dissolving agent, such as streptokinase, urokinase or tPA (tissue plasminogen activator), to dissolve a clot in a coronary artery and restore some blood flow. For best effect, these drugs must be used within a few hours (usually 3) of a heart attack.

If this treatment isn't done immediately after a heart attack, PTCA or coronary artery bypass surgery (CABG) may be done later to improve blood supply to the heart muscle. Once part of the heart muscle dies, its function can't be restored. However, function may be restored to areas with decreased blood flow.

Is there any way to reduce the chance of a heart attack?

Many scientific studies show that certain characteristics increase the risk of coronary heart disease, which causes heart attack. These are called risk factors. Some of them can be modified, treated or controlled, and some can't. The major modifiable risk factors are high blood pressure, tobacco smoke, high blood cholesterol, physical inactivity, obesity and diabetes mellitus. These and other risk factors are discussed on pages 36–43.

The American Heart Association strongly urges Americans to control their modifiable risk factors. Also, people with angina should take episodes of chest pain seriously and see their doctor before their atherosclerosis leads to a heart attack.

Arrhythmias and Sudden Cardiac Death

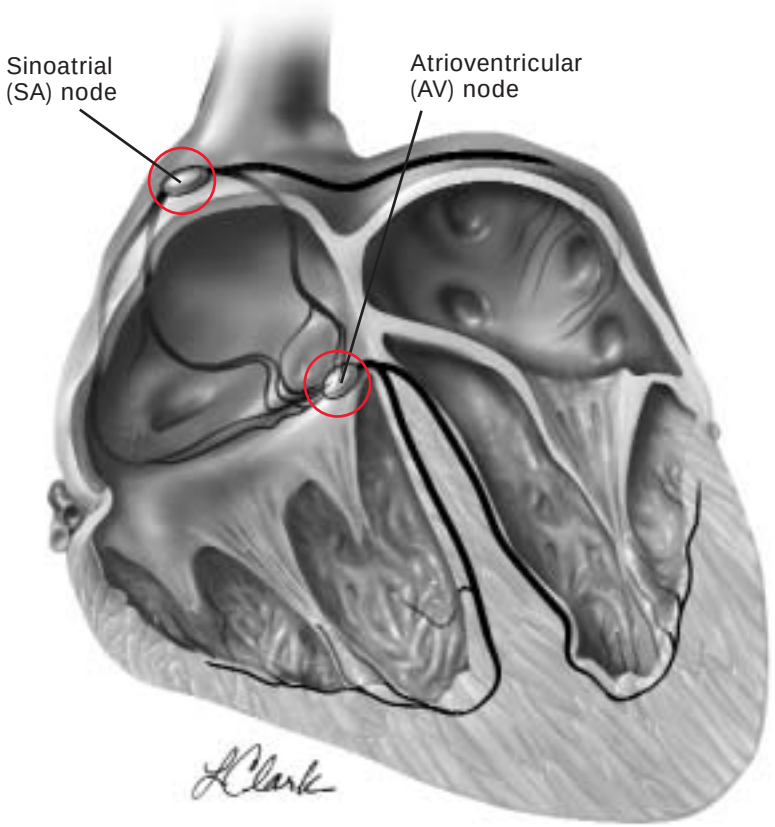
What are arrhythmias?

Arrhythmias or dysrhythmias are abnormal heart rhythms. They can cause the heart to pump less effectively.

The heart has four chambers. The top two are the atria, and the lower two are the ventricles. Normally the heartbeat starts in the right atrium when a special group of cells sends an electrical signal. (These cells are called the sinoatrial or SA node, the sinus node or the heart's "pacemaker.") This signal spreads throughout the atria and to the atrioventricular (AV) node. The AV node connects to a group of fibers in the ventricles that conduct the electric signal. The impulse travels down these specialized fibers (the His-Purkinje system) to all parts of the ventricles. The electrical signal must follow this exact route for the heart to pump properly.

The heart contracts (beats) as the electrical impulse moves through it. This normally occurs 60 to 100 times a minute. The atria contract a split second before the ventricles. This lets them empty their blood into the ventricles before the ventricles contract.

On an electrocardiogram (ECG or EKG), the P wave shows the impulse passing through the atria. The QRS wave shows the impulse passing through the ventricles. As long as the impulse is transmitted normally, the heart pumps and beats at a regular pace.



The SA and AV nodes are responsible for normal cardiac rate and rhythm.

The term arrhythmia refers to any change from this normal sequence of starting and conducting impulses. Some arrhythmias are so brief (for example, a temporary pause or premature beat) that the overall heart rate or rhythm isn't greatly affected. But if arrhythmias last for some time, they may cause the heart rate to be too slow or too fast, or the heart rhythm to be erratic.

The term **bradycardia** describes a rate of less than 60 beats per minute. **Tachycardia** usually refers to a heart rate of more than 100 beats per minute.

What causes arrhythmias?

Under some conditions almost all heart tissue can start a heartbeat. In other words, another part of the heart can become the pacemaker. Cells in the heart's conduction system can fire automatically and start electrical activity. Normally, the heart's most rapidly firing cells are in the sinus node, making that area a natural pacemaker. Secondary pacemakers elsewhere in the heart provide a "back-up" rhythm when the sinus node doesn't work properly or when impulses are blocked somewhere in the conduction system.

An arrhythmia occurs...

- when the heart's natural pacemaker develops an abnormal rate or rhythm.
- when the normal conduction pathway is interrupted.
- when another part of the heart takes over as pacemaker.

What is heart block?

Sometimes the signal from the upper to lower chambers is impaired or doesn't transmit. This is "heart block" or "AV block." This does **not** mean that blood flow or blood vessels are blocked. Heart block is classified according to the level of impairment — first-degree heart block, second-degree heart block or third-degree (complete) heart block.

First-degree heart block, or first-degree AV block, is when the electrical impulse moves through the AV node more slowly than normal. The time it takes for the impulse to get from the atria to the ventricles (the PR interval) should be less than about 0.2 seconds. If it takes longer than this, it's called first-degree heart block.

Heart rate and rhythm are normal, and there may be nothing wrong with the heart. Certain heart medicines such as digitalis can slow conduction of the impulse from the atria to the ventricles and cause first-degree AV block.

In second-degree heart block, some signals from the atria don't reach the ventricles. This causes "dropped beats." On an electrocardiogram, the P wave isn't followed by the QRS wave, because the ventricles weren't activated.

Third-degree or complete heart block (complete AV block) means that the heart's electrical signal doesn't pass from the upper to the lower chambers. When this occurs, an independent pacemaker in the lower chambers takes over. The ventricles can contract and pump blood, but at a slower rate than the atrial pacemaker. On the electrocardiogram, there's no normal relationship between the P and QRS waves.

Complete heart block is most often caused in adults by heart disease or as a side effect of drug toxicity. Heart block also can be present at — or even before — birth. (This is called congenital heart block.) It also may result from an injury to the electrical conduction system during heart surgery. When the pacemaker in the ventricles isn't fast enough or reliable enough, an artificial pacemaker is put in.

What causes tachycardias?

Under certain conditions, the automatic firing rate of secondary pacemaker tissue may become too fast. If such an abnormal "focus" fires faster than the sinus node, it may take over control of the heart rhythm and cause tachycardia.

In another type of abnormal conduction, impulses get caught in a merry-go-round-like sequence. This process, called reentry, is a common cause of tachycardias. Regardless of their cause, tachycardias are classified by where they arise. Thus, ventricular tachycardias begin in the heart's lower chambers. Supra-ventricular tachycardias arise higher in the heart — either in the upper chambers (atria) or the middle region (AV node or the very beginning portion of the His-Purkinje system).

What are the symptoms of arrhythmias?

Arrhythmias can produce a broad range of symptoms, from barely perceptible to cardiovascular collapse and death. For example, a single premature beat may feel like a "palpitation" or "skipped beat." Premature beats that occur often or in rapid succession may cause a greater awareness of heart palpitations. They may cause a "fluttering" sensation in the chest or neck.

When arrhythmias last long enough to affect how well the heart works, more serious symptoms may develop. If the heart rate is too slow (bradycardia), the heart may not be able to pump enough blood to the body. This can cause fatigue, dizziness, lightheadedness, fainting or near-fainting spells.

Rapid heart beating (called tachycardia or tachyarrhythmia) can reduce the heart's ability to pump and circulate blood effectively. This can cause palpitations, rapid heart action, dizziness, lightheadedness, fainting or near fainting. Heartbeats may be either regular or irregular in rhythm.

Cardiac arrest, collapse and sudden death follow unless medical help is provided immediately.

When rapid heart beating starts in the ventricles — called ventricular tachycardia — it can be life-threatening. The most serious cardiac rhythm disturbance is ventricular fibrillation. The lower chambers quiver and the heart can't pump any blood. **Cardiac arrest, collapse and sudden death follow unless medical help is provided immediately.** If treated in time, ventricular tachycardia and ventricular fibrillation can be converted into normal rhythm with electrical shock.

Tachycardias also can cause serious injury to other organs. For example, the brain, kidneys, lungs or liver may be damaged because the heart can't pump blood effectively during cardiac arrest. Blood clots can form in the heart's upper chambers because of atrial fibrillation. In this disorder, the atria quiver instead of beating effectively. Blood isn't pumped completely out of them when the heart beats, so the blood pools and clots. If a piece of a blood clot in the atria breaks free, it can lodge in an artery in the brain, causing a stroke. Such clots can also damage other organs.

Who is prone to arrhythmias?

Arrhythmias occur throughout the population. Their severity varies widely. The heart rate speeds up during physical activity, stress or excitement, and slows down during sleep. Beyond these daily changes, probably everyone at some time has premature atrial or ventricular beats. In fact, during a 24-hour period about one-fifth of healthy adults are likely to have frequent or multiple types of ventricular premature beats. (This includes short episodes of ventricular tachycardia in a small percentage of monitored people.)

The prevalence of atrial and ventricular arrhythmias tends to increase with age, even when there's no overt sign of heart disease. Certain congenital conditions may make a person prone to arrhythmias. For example, an incompletely developed conduction system can cause chronic heart block and bradycardia. People born with extra conduction pathways, either near the AV node or bridging the atria and ventricles, are prone to reentrant supraventricular tachycardias.

Still, **acquired heart disease is the most important factor making a person prone to arrhythmias.** The main causes are atherosclerosis, high blood pressure and inflammatory or degenerative conditions. The scarring or abnormal tissue deposits found with these diseases can cause bradycardias by interfering with the work of the sinus node or overall AV conduction. Likewise, they can cause tachycardias, originating in either the atria or ventricles. They may cause cells to fire abnormally or create islands of electrically inert tissue. (Impulses circulate in a reentrant fashion around these areas.)

A variety of other factors may lead some people to develop arrhythmias. Among them is the part of the autonomic nervous system that's involved in cardiovascular regulation. One element of this control system slows the sinus rate and depresses AV nodal conduction. (These effects occur during sleep or in well-trained athletes.) The opposing element of the autonomic nervous system tends to speed up the firing rate of the sinus node and other pacemaker tissue in the heart. Further, it may also make it easier for reentrant tachycardias to occur.

Many chemical agents may cause arrhythmias, sometimes with serious consequences. Known factors include high or low blood and tissue concentrations of a variety of minerals. Some examples are potassium, magnesium and calcium. These play a vital role in starting and conducting normal impulses in the heart. Alcohol, cigarettes and recreational drugs can provoke arrhythmias. So can various cardiac medications. Even drugs used to treat an arrhythmia may cause another arrhythmia.

How are arrhythmias diagnosed?

An electrocardiogram is the standard clinical tool for diagnosing arrhythmias. It records the relative timing of atrial and ventricular electrical events. It can be used to measure how long it takes for impulses to travel through the atria, AV conduction system and ventricles.

An arrhythmia is considered documented if it can be recorded on an ECG. Often, though, the ECG of someone who complains of symptoms that suggest arrhythmia doesn't show anything. That's because of the fleeting nature of arrhythmias.

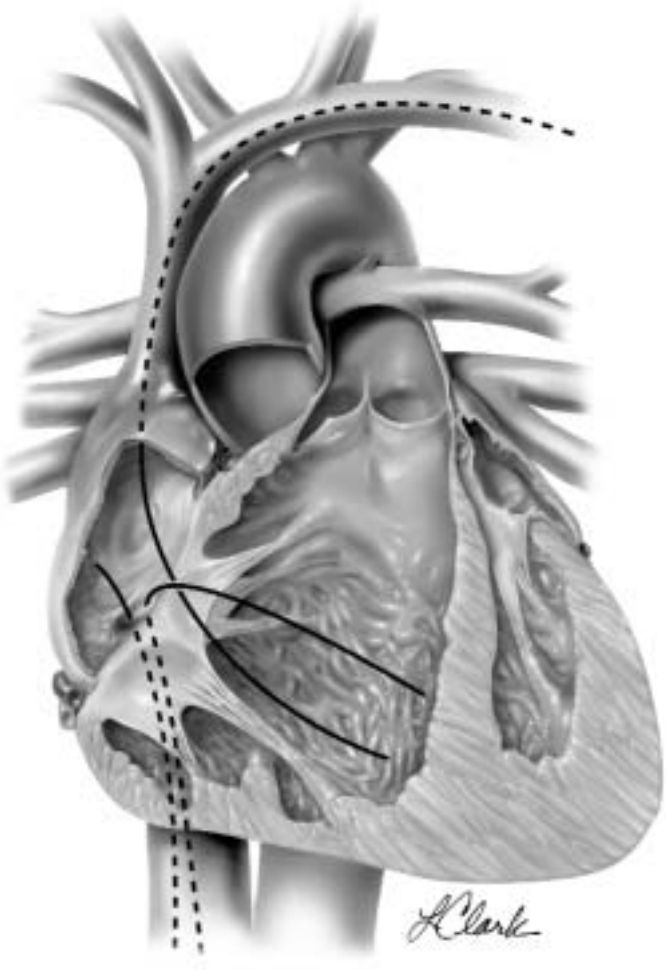
Suspected arrhythmias sometimes may be documented by using a small, portable ECG recorder, called a Holter monitor. This can record 24 hours of continuous electrocardiographic signals. For suspected arrhythmias that occur less than daily, a patient can wear an event monitor. It has a continuously updated memory loop and can let the heart be monitored by telephone.

These techniques are passive; they require an arrhythmia to occur spontaneously. Other options are used that provoke arrhythmias and make their diagnosis (and thus their proper treatment) easier. For example, treadmill testing may be used for people whose suspected arrhythmias are clearly exercise-related. In patients prone to passing out, tilt table studies may be used. These reproduce the faint when it's due to abnormal nervous system reflexes that cause the heart rate to slow down and the blood pressure to drop.

Electrophysiologic testing has become extremely valuable for provoking known but infrequent arrhythmias and for unmasking suspected arrhythmias. This procedure is done using local anesthesia. In it, temporary electrode catheters are placed through peripheral veins or arteries into the heart using a fluoroscope. Then these catheters are positioned in the atria, ventricles or both, and at strategic locations along the conduction system. Their purpose is to record cardiac electrical signals and "map" the spread of electrical impulses during each beat.

This technique shows where the heart block is (AV node vs. His-Purkinje system). It also shows where tachycardia originates (supraventricular vs. ventricular) far better than an ECG usually does. The ability to electrically stimulate the heart at programmed rates and induce precisely timed premature beats lets a doctor assess electrical properties of the heart's conduction system. Most significantly, it also triggers latent tachycardia or bradycardia. Induced tachycardias can usually be stopped by rapid pacing via the electrode catheters. Sometimes an externally applied shock may be required if the patient loses consciousness during the tachycardia.

Being able to "turn on" and "turn off" tachycardias during electrophysiologic studies allows antiarrhythmic drugs to be tested quickly for effectiveness. This can be done during a single study using intravenous therapy or during short follow-up studies with oral medication. Electrophysiologic testing has been performed safely worldwide; complications only rarely occur.



Cross section of the heart showing various catheter positions for recording electrical activity.

When should arrhythmias be treated?

Once an arrhythmia has been documented, it's important to try to find out where it originates. It's also necessary to find out whether it's abnormal or merely reflects the heart's normal physiologic processes. The arrhythmia must be abnormal and clinically significant before it warrants an intervention. That means it must either cause symptoms or put a person at risk for more serious arrhythmias or complications of arrhythmias in the future.

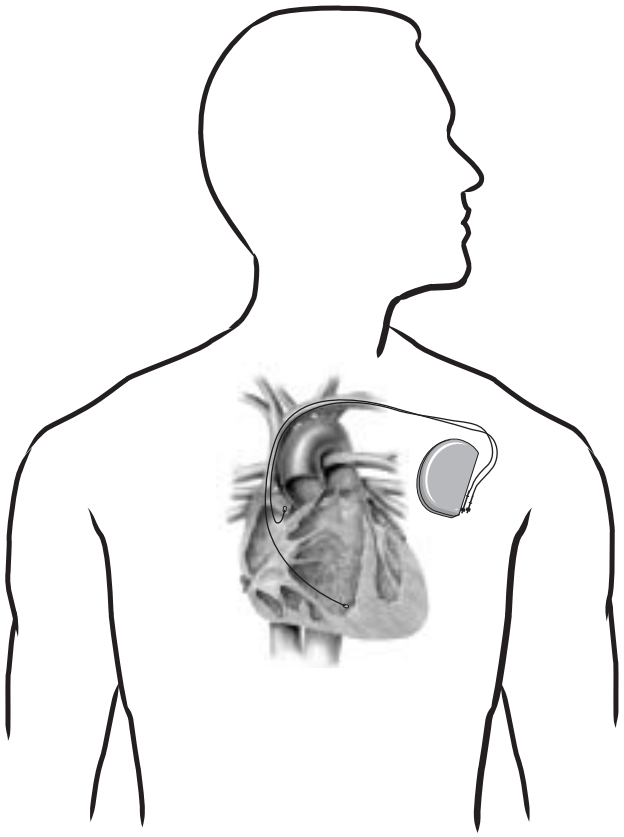
In some patients whose symptoms suggest arrhythmias, tachycardias or bradycardias may be found during diagnostic (particularly electrophysiologic) tests. In these cases, a doctor must judge whether the arrhythmia is a likely enough explanation for the patient's original symptoms to justify therapy. The risks and benefits of the intervention also must be taken into account.

How are bradycardias treated?

Potentially life-threatening bradycardias may be treated acutely with medication. Such medication increases the automatic firing rate of cardiac pacemaker tissue and improves the transmission of impulses through the conduction system.

Another way to maintain the cardiac rhythm is to insert a temporary pacemaker. This involves using a thin, flexible electrode wire. One end is positioned inside the heart or next to the heart wall. The other is connected to an external, temporary pulse generator. It uses batteries to send electrical impulses to the heart via the wire.

If symptomatic bradycardia persists or is likely to recur, despite eliminating reversible causes, then implanting a permanent pacemaker makes sense. This device consists of a pulse generator, which can be as small as a silver dollar. The pulse generator is hooked up to one or two pacemaker leads that are permanently affixed to a ventricular or atrial site, or to both.



Common placement of a permanent pacemaker.

Permanent pacemakers deliver electrical stimuli to the heart when the heart's spontaneous rate falls below a set value. Many of these devices have sensors to vary the pacemaker's rate based on the body's needs. These are called demand pacemakers.

The pacemaker generator is implanted under the skin below the collarbone. It may work for 8 to 12 years before it needs to be replaced.

How are tachycardias treated?

Symptomatic tachycardias and premature beats may be treated with a variety of antiarrhythmic drugs. These may be given intravenously on an acute basis, or orally for long-term treatment. These drugs either suppress the abnormal firing of pacemaker tissue or depress the transmission of impulses in tissues that either conduct too rapidly or participate in reentry. In patients with atrial fibrillation, a blood thinner (anticoagulant) is often added to reduce the risk of blood clots and stroke.

When tachycardias or premature beats occur often, the effectiveness of antiarrhythmic drug therapy may be gauged by ECG monitoring in a hospital, by using a 24-hour Holter monitor or by serial drug evaluation with electrophysiologic testing.

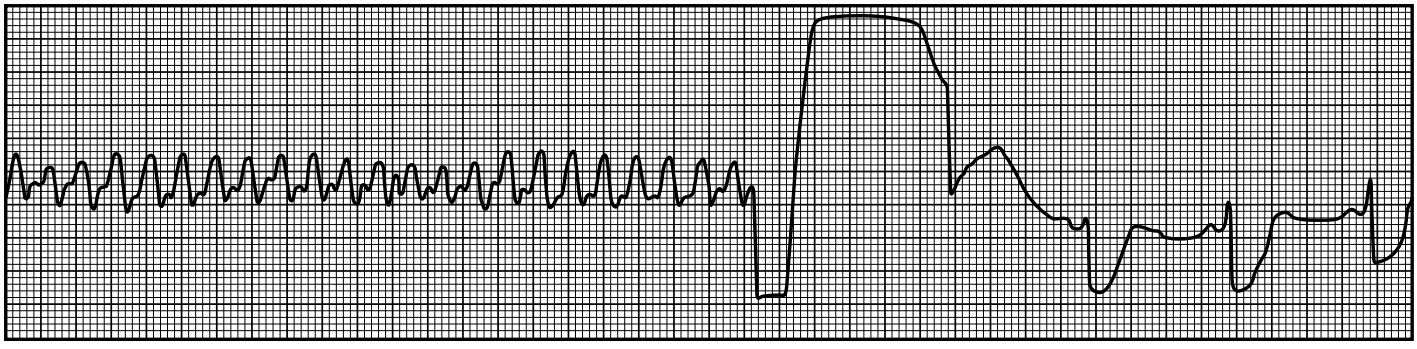
The relative simplicity of antiarrhythmic drug therapy must be balanced against two disadvantages. One is that the drugs must be taken daily and indefinitely. The other is the risk of side effects. While side effects are a risk of all medication, those associated with antiarrhythmic drugs can be very hard to manage. They include proarrhythmia, which is the more-frequent occurrence of preexisting arrhythmias or the appearance of new arrhythmias as bad as or worse than those being treated.

What non-drug treatments are used for tachycardias?

Several non-drug therapies are now being used to treat patients with tachycardias. One involves using **adenosine**, a natural substance produced in humans that helps protect against ischemia (lack of blood flow). It's produced in blood vessels, heart and skeletal muscle, and other organs. Adenosine has a depressant or arrythmogenic effect on sinoatrial node activity. This makes adenosine effective in treating tachycardias involving the SA node. Adenosine is also used in diagnosing and treating supraventricular tachycardias.

Ablative techniques physically destroy the heart tissue that causes or contributes to a tachycardia. In this surgical approach, the problem tissue is removed or destroyed by local heating or cooling. Until recently, such therapy was only feasible through surgery (often an open-heart procedure).

Newer advances now permit therapeutic ablations using a transcatheter approach. In this technique, an electrode catheter inserted through a vein or artery during electrophysiologic studies is used to perform targeted electrocautery in the heart. A patient may be cured of tachycardia through ablative therapy, so antiarrhythmic medication is no longer needed. Transcatheter ablation is rapidly becoming the treatment of choice for many supraventricular tachycardias.



Conversion of ventricular fibrillation to a regular heartbeat.

Other types of electrical therapy are also used to treat tachycardias. On an acute basis, many life-threatening tachycardias can be stopped by an electric shock delivered to the heart or by rapid "overdrive" pacing with an electrode catheter. Implantable devices can provide automatic electrical therapy on a chronic basis for patients with recurrent tachycardias.

The greatest advance in this area is the **implantable cardioverter defibrillator**. It's used in patients at risk for recurrent, sustained ventricular tachycardia or fibrillation.

The device is connected to leads positioned inside the heart or on its surface. These leads are used to deliver electrical shocks, sense the heart's rhythm and sometimes pace the heart, as needed. The various leads are tunneled to a pulse generator, which is implanted in a pouch beneath the skin of the chest or abdomen. These generators are typically a little larger than a wallet. They have electronics that automatically monitor and treat abnormal heart rhythms. Newer devices can be installed through blood vessels. This eliminates the need for open-chest surgery.

When an implantable cardioverter defibrillator detects ventricular tachycardia or fibrillation, it shocks the heart to restore the normal rhythm. New devices also provide overdrive pacing to electrically convert a sustained ventricular tachycardia, and "backup" pacing if bradycardia occurs. They also offer other sophisticated functions. These include storing detected arrhythmic events and the ability to do "noninvasive" electrophysiologic testing.

Implantable cardioverter defibrillators have been very useful in preventing sudden death in patients with known, sustained ventricular tachycardia or fibrillation. Studies are now being done to find out how best to use them and whether they may help prevent cardiac arrest in patients who haven't had, but are at high risk for, life-threatening ventricular arrhythmias. One study now in progress, the AVID Trial, compares implantable defibrillators to medical treatment. This will help show how effective defibrillators are in increasing patients' long-term survival.

What is sudden cardiac death (SCD)?

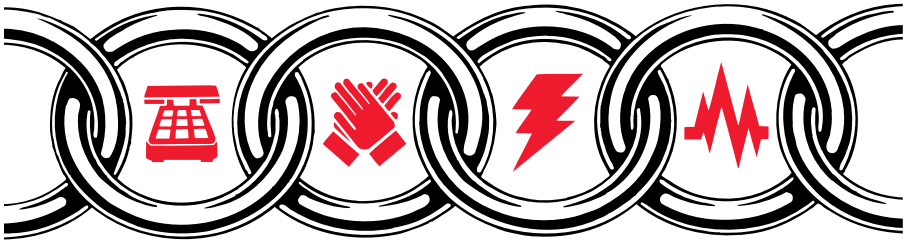
Also called sudden death, SCD occurs when the heart stops abruptly (cardiac arrest). The victim may or may not have diagnosed heart disease. The time and mode of death are **unexpected**. It can occur within minutes after symptoms appear, or there may be no symptoms before collapse. The most common underlying reason that patients die suddenly from cardiac arrest is coronary heart disease.

Sudden death from cardiac arrest is a major health problem that has received much less publicity than heart attack. Although the direct medical costs are much less than for lingering illnesses, its economic and social impacts are huge. Sudden cardiac death occurs on average at about 60 years of age, claims many people during their most productive years, and devastates unprepared families.

What causes sudden cardiac death?

Most known heart diseases can lead to cardiac arrest and sudden cardiac death. Most cases of cardiac arrest that lead to SCD occur when the heart's electrical impulses become rapid (ventricular tachycardia) and then chaotic (ventricular fibrillation or VF). This irregular heart rhythm (arrhythmia) causes the heart to suddenly stop pumping blood. A small number of cardiac arrests are caused by extreme slowing of the heart (bradycardia).

Other causes of cardiac arrest include respiratory arrest, electrocution, drowning, choking and trauma. Cardiac arrest also can occur without any known cause.



Each link in the chain of survival is critical.

The term "massive heart attack" is often mistakenly used in the media to describe sudden cardiac death. The terms "heart attack" or "myocardial infarction" refer to death of heart muscle tissue due to the loss of blood supply, not necessarily resulting in a cardiac arrest or the death of the heart attack victim. A heart attack may cause cardiac arrest and sudden cardiac death, but the terms aren't synonymous.

Most heart attack victims do **not** develop cardiac arrest. However, because cardiac arrest may complicate a heart attack, anyone with heart attack warning signs should call 9-1-1. (See warning signs on page 35.)

Can the cardiac arrest that causes SCD be reversed?

If cardiac arrest victims receive no treatment, brain damage can start to occur in just four to six minutes after the heart stops pumping blood. If cardiac arrest victims receive immediate cardiopulmonary resuscitation (CPR), it will keep blood flowing to the heart and brain until definitive treatment is provided. CPR consists of mouth-to-mouth rescue breathing and chest compressions.

VF cardiac arrest can be reversed if the victim is treated with an electric shock to the heart within a few minutes. The electric shock can stop the abnormal rhythm and allow a normal rhythm to resume. This process, called defibrillation, is done using a defibrillator.

A victim's chances of survival after VF cardiac arrest are reduced by 7 to 10 percent with every minute that passes without treatment. Few attempts at resuscitation succeed after 10 minutes have elapsed.

In-hospital survival after cardiac arrest in heart attack patients improved dramatically when the DC defibrillator and bedside monitoring were developed. Later it also became clear that cardiac arrest could be reversed outside a hospital by properly staffed emergency rescue teams trained to give CPR and to defibrillate. Thus, the problem isn't the ability to reverse cardiac arrest, but reaching the victim in time to do so. **The American Heart Association strongly supports implementing the "chain of survival" to rescue people who suffer sudden cardiac arrest in the community.**

To strengthen the chain of survival, communities should have the following four elements in place:

1) **An enhanced 9-1-1 system.**
2) **CPR training programs available to all citizens.**
3) **An automated external defibrillator (AED) program to deliver CPR and early defibrillation within minutes of a cardiac arrest.**
4) **Mobile advanced life support (paramedic) units available 24 hours a day.**

Who's at risk for sudden cardiac death?

In 90 percent of adult victims of sudden cardiac death, two or more major coronary arteries are narrowed by atherosclerosis. Scarring from a prior heart attack is found in two-thirds of victims. It's not surprising, then, that predisposing factors for sudden cardiac death are similar to risk factors for atherosclerotic heart disease. They include cigarette smoking and high blood pressure.

A heart that's scarred or enlarged from any cause is prone to develop life-threatening ventricular arrhythmias. The first six months after a heart attack is a particularly high-risk period for sudden cardiac death in patients with atherosclerotic heart disease. A thickened heart muscle from any cause (typically high blood pressure or valvular heart disease) — especially when there's congestive heart failure, too — is an important risk factor for sudden cardiac death.

Under certain conditions, various heart medications and other drugs — as well as illegal drug abuse — can lead to abnormal heart rhythms that cause sudden cardiac death. So-called "antiarrhythmic" drugs, even at normally prescribed doses, sometimes may produce lethal ventricular arrhythmias ("proarrhythmic" effect). Regardless of whether there's heart disease, significant changes in blood levels of potassium and magnesium (from using diuretics, for example) also can cause life-threatening arrhythmias and cardiac arrest.

When sudden cardiac death occurs in young adults, other heart abnormalities are more likely causes. Many of these young victims have a thickened heart muscle (hypertrophic cardiomyopathy) without having high blood pressure. (See page 64.)

Certain electrical abnormalties within the heart also may cause sudden cardiac death in the young. One is the Wolff-Parkinson-White syndrome, in which there's a rapid conduction pathway between the heart's upper and lower chambers. The electrical signal from the atria may arrive at the ventricles too soon. This sometimes can allow dangerously rapid rates to develop in the ventricles when there's a rapid rhythm disturbance in the atria.

Another problem is the long Q-T syndrome, an infrequent, hereditary disorder that can occur in otherwise-healthy people. It usually affects children or young adults. The Q-T interval represents the time for electrical activation and inactivation (recovery) of the ventricles. When the electrical recovery after each heartbeat is prolonged, it may set the stage for fatal ventricular arrhythmias.

Less often, inborn blood vessel abnormalities, particularly in the coronary arteries and aorta, may be present in young sudden death victims. Adrenaline released during intense athletic or physical activity often triggers sudden cardiac death when these abnormalities are present.

What treatments are available for survivors of cardiac arrest?

If a cardiac arrest is due to ventricular tachycardia or ventricular fibrillation, survivors are at risk for another arrest, especially if they have underlying heart disease. Patients whose cardiac arrest is associated with atherosclerotic heart disease are at risk of recurrent cardiac arrests when the first cardiac arrest episode occurs without a new heart attack. That's because this implies a persistent tendency toward electrical instability.

Survivors of cardiac arrest must have all causes corrected to prevent future episodes. Possible causes include myocardial ischemia, arrhythmia, etc. This often requires cardiac catheterization (to show the heart and coronary blood vessels) and electrophysiologic testing. It's also necessary to find out the possible role of reversible causes and remove or correct them. Such factors may include excessive

doses of various cardiac drugs, the presence of antiarrhythmic agents, and abnormal blood levels of various minerals, especially potassium.

For example, cardiac arrest survivors with the Wolff-Parkinson-White syndrome (with otherwise-normal hearts) may be satisfactorily treated with a catheter ablation procedure that destroys the rapid conduction pathway between the upper and lower heart chambers. At the other extreme, a heart transplant may be advised for patients whose cardiac arrest was caused by severe structural heart disease.

In cardiac arrest survivors with atherosclerotic heart disease but without a new heart attack, treatment focuses on both the narrowing in the coronary arteries and the ventricular tachycardia and fibrillation that occurs during electrophysiologic testing. Therapy limited to reversing or blunting the effects of reduced blood supply to the heart (through bypass surgery, angioplasty or medication) is likely to protect only a minority of these patients from recurrent cardiac arrest. Such treatments alone don't stabilize the electrical abnormalities in scarred heart muscle that can lead to recurrent cardiac arrest.

Patients with atherosclerotic heart disease are at risk of recurrent cardiac arrests when the first, aborted sudden death episode occurs without a new heart attack.

Many therapies exist to control potentially life-threatening ventricular tachyarrhythmias that result from diseased or scarred heart muscle. Antiarrhythmic medication may protect against subsequent sudden death in certain subsets of cardiac arrest survivors. (They include persons whose hearts pump well who are given a drug that suppresses ventricular tachycardia induced during electrophysiologic testing.) However, antiarrhythmic medication is limited by the need for life-long dosing and the potential for intolerable or lethal side effects. As a result, implantable cardioverter defibrillators are being used more and more. They can automatically detect ventricular tachycardia or fibrillation when it occurs and, within seconds, deliver a lifesaving electrical shock to restore a normal rhythm.

Rapid heart rhythms account for the great majority of sudden cardiac deaths. Still, very slow rhythms due to conduction system failure sometimes cause cardiac arrest. People resuscitated from this uncommon type of cardiac arrest are treated with a permanent pacemaker after acute reversible causes, such as drug toxicity, have been ruled out.

How can the public prepare for cardiac emergencies?

- **Know the warning signs of heart attack and cardiac arrest.** During cardiac arrest, a victim becomes unresponsive, stops normal breathing, and loses pulse or other signs of circulation.
- **Call 9-1-1 immediately** to access the emergency medical services (EMS) if you see any warning signs of heart attack or if someone collapses suddenly and is unresponsive.
- **Begin cardiopulmonary resuscitation (CPR) immediately.** This will help keep the cardiac arrest victim alive until emergency help arrives. CPR keeps blood flowing to the heart and brain until defibrillation can be provided.
- **Provide early defibrillation with an automated external defibrillator (AED).** Public access defibrillation programs can train lay rescuers to perform CPR and use an AED in the community.

Death from cardiac arrest isn't inevitable. If more people react quickly by calling 9-1-1 and doing CPR, more lives can be saved.

Heart Attack Warning Signs and Actions

Some heart attacks are sudden and intense — the "movie heart attack," in which a person dramatically gasps, clutches his heart and drops to the ground, and no one doubts what's happening. But most heart attacks start slowly, with mild pain or discomfort. Often the people affected aren't sure what's wrong and wait too long before getting help. Here are some of the signs that can mean a heart attack is happening.

- **Chest discomfort.** Most heart attacks involve discomfort in the center of the chest that lasts more than a few minutes, or that goes away and comes back. It can feel like uncomfortable pressure, squeezing, fullness or pain.
- **Discomfort in other areas of the upper body.** Symptoms can include pain or discomfort in one or both arms, the back, neck, jaw or stomach.
- **Shortness of breath.** This feeling often comes along with chest discomfort. But it can occur before the chest discomfort.
- **Other signs.** These may include breaking out in a cold sweat, nausea or light-headedness.

If you or someone you're with has chest discomfort, especially with one or more of the other signs, don't wait longer than a few minutes (no more than 5) before calling for help. **Call 9-1-1...Get to a hospital right away.**

Calling 9-1-1 is almost always the fastest way to get lifesaving treatment. Emergency medical services (EMS) staff can begin treatment when they arrive — up to an hour sooner than if someone gets to the hospital by car. The staff are also trained to revive someone whose heart has stopped. You'll also get treated faster in the hospital if you come by ambulance.

If you can't access the EMS, have someone drive you to the hospital right away. If you're the one having symptoms, don't drive yourself, unless you have absolutely no other option.

Chest or arm pain may be a sign of heart attack. Don't ignore it.

Risk Factors for Heart Disease

Risk factors are traits and lifestyle habits that increase the risk of disease. Extensive clinical and statistical studies have identified several risk factors for coronary heart disease and heart attack. Most of them can be modified, treated or controlled. Some can't. The more risk factors a person has, the higher the chances that he or she will develop heart disease. The best way to prevent a heart attack is to reduce heart disease risk factors.

What risk factors for heart disease can be controlled or treated?

High Blood Pressure — High blood pressure makes the heart work harder than normal, causing it to enlarge and weaken over time. Both the heart and arteries are then more prone to injury. High blood pressure raises the risk of heart attacks, strokes, kidney failure, eye damage, congestive heart failure and atherosclerosis. When high blood pressure exists with obesity, smoking, high blood cholesterol levels or diabetes, the risk of heart attack increases several times.

Blood pressure tends to increase with age. Men have a greater risk of high blood pressure than women until age 55, when their respective risks are similar. At age 75 and older, women are more likely to develop high blood pressure than men are.

High blood pressure usually has no specific symptoms and no early warning signs. It's truly a "silent killer." But a simple, quick, painless test can detect it. (See page 8.)

If you have high blood pressure, work with your healthcare provider to control it. Eating a proper diet, losing weight, exercising regularly, restricting salt (sodium) intake and following a medication regimen may all be prescribed to lower blood pressure.

Tobacco Smoke — Smoking is the single most preventable cause of death in the United States. Smokers' risk of heart attack is more than twice that of nonsmokers. Smokers who have a heart attack are more likely to die and die suddenly (within an hour) than are nonsmokers.

The nicotine and carbon monoxide in tobacco smoke reduce the amount of oxygen in the blood. They also damage blood vessel walls, causing plaque to build up. Tobacco smoke may trigger blood clots to form, too. Smoking also promotes heart disease by reducing HDL ("good") cholesterol. (See page 38 for more about HDL cholesterol.)

Smoking greatly increases your risk for heart disease.

Smoking is the biggest risk factor for peripheral vascular disease, which is the narrowing of blood vessels carrying blood to leg and arm muscles. In fact, this condition occurs almost exclusively in smokers. Smokers with peripheral vascular disease are also more likely to develop gangrene and require leg amputation.

People who smoke cigars or pipes seem to have a higher risk of death from coronary heart disease (and possibly stroke) than people who don't smoke tobacco. However, their risk isn't as great as cigarette smokers'. This is probably because they're less likely to inhale the smoke.

Smoking may disturb the heart rhythm in people who have chest pain or who've had a heart attack. This can lead to sudden cardiac arrest, in which the heart stops pumping. Death follows within minutes after symptoms appear. Cigarette smoking is the biggest risk factor for sudden cardiac arrest.

Constant exposure to other people's smoke raises the risk of heart disease and stroke even for nonsmokers.

If you don't smoke, don't start. **If you do smoke, get help to quit NOW!** Many effective programs, nicotine patches and other medications are available to help you quit. As soon as you stop smoking, your risk of heart disease starts to drop. In time your risk will be about the same as if you'd never smoked.

High Blood Cholesterol — The risk of coronary heart disease rises as blood cholesterol levels increase. When other risk factors (such as high blood pressure and tobacco smoke) are present, this risk increases even more.

High cholesterol has no symptoms, and many people have it without knowing it. It's important to find out what your cholesterol levels are so you can lower them if you need to.

Everyone age 20 and older should have their cholesterol measured at least once every five years. It's best to have a blood test called a fasting "lipoprotein profile" to find out your cholesterol numbers. This test is done after a 9–12-hour fast. It gives information about total cholesterol, LDL ("bad") cholesterol, HDL ("good") cholesterol and triglycerides. Researchers have established healthy ranges for each of these. They're given in the tables that follow.

If you can't have a lipoprotein profile, knowing your total and HDL cholesterol can give you a good idea of your cholesterol levels. However, if your total cholesterol is 200 mg/dL or more — or if your HDL cholesterol is less than 40 mg/dL — you need to have a lipoprotein profile done.

If your cholesterol is high or you have other risk factors, your healthcare provider will likely want to monitor your cholesterol more closely. Then you should follow your provider's advice about how often to have your cholesterol tested.

Total Cholesterol Level	Category
Less than 200 mg/dL	Desirable level that puts you at lower risk for heart disease. A cholesterol level of 200 mg/dL or higher raises your risk.
200–239 mg/dL	Borderline high
240 mg/dL and above	High blood cholesterol. A person with this level has more than twice the risk of heart disease as someone whose cholesterol is below 200 mg/dL.

Cholesterol levels are measured in milligrams (mg) of cholesterol per deciliter (dL) of blood.

Most cholesterol in the blood is carried in a protein "package" called low-density lipoprotein (LDL). LDL cholesterol is "bad" cholesterol because it's deposited in artery walls, increasing the buildup of plaque. High levels of LDL cholesterol **raise** your risk of coronary heart disease and stroke.

LDL Cholesterol Level	Category
Less than 100 mg/dL	Optimal
100–129 mg/dL	Near or above optimal
130–159 mg/dL	Borderline high
160–189 mg/dL	High
190 mg/dL and above	Very High

mg/dL = milligrams per deciliter of blood

Your LDL cholesterol goal depends on how many other risk factors you have.

- If you don't have coronary heart disease or diabetes and have one or no risk factors, your LDL goal is less than 160 mg/dL.
- If you don't have coronary heart disease or diabetes and have two or more risk factors, your LDL goal is less than 130 mg/dL.
- If you do have coronary heart disease or diabetes, your LDL goal is less than 100 mg/dL.

HDL (high-density lipoprotein) **cholesterol** is the "good" cholesterol. A high level of it **lowers** your risk of heart disease and heart attack. HDL cholesterol tends to carry excess cholesterol back to the liver, where it's removed from the body. People with a **low** level of HDL cholesterol (less than 40 mg/dL) have a higher risk of heart attack.

HDL Cholesterol Level	Category
Less than 40 mg/dL	A major risk factor for heart disease.
40–59 mg/dL	The higher your HDL level, the better.
60 mg/dL and above	An HDL of 60 mg/dL and above is considered protective against heart disease.

mg/dL = milligrams per deciliter of blood

Triglyceride is the most common type of fat in the body. Many people who have heart disease or diabetes have high triglyceride levels.

Normal triglyceride levels vary by age and sex. A high triglyceride level combined with low HDL cholesterol or high LDL cholesterol seems to speed up atherosclerosis. Talk with your healthcare provider about your triglyceride level at regular check-ups, and how to reduce it if it's too high.

Triglyceride Level	Category
Less than 150 mg/dL	Normal
150–199 mg/dL	Borderline high
200–499 mg/dL	High
500 mg/dL and above	Very high

mg/dL = milligrams per deciliter of blood

The body needs a certain amount of cholesterol to build cell membranes, etc. However, the liver makes enough cholesterol to meet these needs. That's why diet is important. A diet high in saturated fats, *trans* fats and cholesterol tends to raise total blood cholesterol and LDL cholesterol. A diet low in saturated fat, *trans* fat and cholesterol helps lower blood cholesterol levels.

Dietary cholesterol is only found in foods from animals. Foods from plants, such as fruits and vegetables, don't have cholesterol.

Saturated fats are found in foods from animals. Some plant oils, commonly used in commercially baked goods and other food products, also are high in saturated fats. Many foods high in saturated fats are also high in cholesterol.

Trans fats result from adding hydrogen to vegetable oils used in commercial baked goods and for cooking in most restaurants and fast-food chains.

On the whole, Americans should reduce the amount of saturated fat, *trans* fat, cholesterol and total fat in their diet. For information about the types of dietary fat (saturated, polyunsaturated, monounsaturated, *trans*) and how to reduce dietary fat and cholesterol, see "An Eating Plan for Healthy Americans: Our American Heart Association Diet." It's available at American Heart Association offices, by calling 1-800-AHA-USA1 (1-800-242-8721), or on our Web site, **americanheart.org.**

If you have high blood cholesterol, eating a healthy diet, maintaining a healthy weight and getting regular physical activity are very important to lower your risk. If you still need drugs to reduce your blood cholesterol, a healthy diet will help lower your cholesterol and improve your overall cardiovascular health.

Physical Inactivity — An inactive lifestyle is a risk factor for coronary heart disease. When physical inactivity is combined with overeating, then excess weight, higher blood cholesterol levels and diabetes can result. All of these raise the risk of heart disease.

Regular, moderate-to-vigorous exercise is important to reduce the risk of heart and blood vessel disease. Exercise can help control blood cholesterol, diabetes and obesity as well as help lower blood pressure in some people.

For most healthy people, the American Heart Association recommends 30–60 minutes of physical activity on most days of the week to condition the heart and lungs.

Moderate activities such as walking, gardening, housework and dancing for at least 30 minutes on most days can help the heart. The time may be broken into three 10-minute, one 10-minute and one 20-minute, or two 15-minute periods per day. People who've been inactive can start with 10 minutes, then work up to more.

Before starting a vigorous exercise program, it's smart to get your doctor's OK. This is especially important if you're middle-aged or older; have heart disease, have had a stroke or another medical problem; and have been inactive a long time.

Obesity and overweight — People who have excess body fat — especially if a lot of it is in the waist area — are at higher risk for health problems. These problems include high blood pressure, high blood cholesterol, high triglycerides, diabetes, heart disease and stroke. People with excess body fat are at higher risk of heart disease even if they don't have other risk factors.

The **waist measurement** and the **body mass index** (BMI) are the recommended ways to estimate a person's body fat. A high-risk waistline is **more than 35 inches** for women, **more than 40 inches** for men.

The body mass index assesses a person's body weight relative to height. It correlates highly with body fat in most people. To find your BMI risk level, weigh and measure yourself wearing very little clothing and no shoes. Find your height in feet and inches in the BMI table. The range of weights that corresponds to minimal risk, moderate risk (overweight) and high risk (obese) is shown for each height.

- BMI values less than 18.5 are considered underweight. **BMI values from 18.5 to 24.9 are healthy.**
- **Overweight is defined as a BMI of 25.0–29.9.** A BMI of 25 corresponds to about 10 percent over ideal body weight.
- **Obesity is defined as a BMI of 30.0 or greater,** or about 30 or more pounds overweight. Extreme obesity is defined as a BMI of 40 or greater.

(To calculate your exact BMI number, multiply your weight in pounds by 705, divide by your height in inches, then divide again by your height in inches.)

Body Mass Index (BMI) Risk Levels (for adults)

Height	Minimal Risk (BMI under 25)	Moderate risk (BMI 25–29.9) Overweight	High risk (BMI 30 and above) Obese
4'10"	118 lbs. or less	119–142 lbs.	143 lbs. or more
4'11"	123 or less	124–147	148 or more
5'0"	127 or less	128–152	153 or more
5'1"	131 or less	132–157	158 or more
5'2"	135 or less	136–163	164 or more
5'3"	140 or less	141–168	169 or more
5'4"	144 or less	145–173	174 or more
5'5"	149 or less	150–179	180 or more
5'6"	154 or less	155–185	186 or more
5'7"	158 or less	159–190	191 or more
5'8"	163 or less	164–196	197 or more
5'9"	168 or less	169–202	203 or more
5'10"	173 or less	174–208	209 or more
5'11"	178 or less	179–214	215 or more
6'0"	183 or less	184–220	221 or more
6'1"	188 or less	189–226	227 or more
6'2"	193 or less	194–232	233 or more
6'3"	199 or less	200–239	240 or more
6'4"	204 or less	205–245	246 or more

(Adapted from *Obesity Education Initiative: Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults*, National Institutes of Health, National Heart, Lung, and Blood Institute, Preprint June 1998)

Some well-trained people with dense muscle mass may have a high BMI score but very little body fat. For them the waist measurement may be a better indicator.

Many overweight and obese people have difficulty losing weight. Even modest weight loss (5 to 10 percent of body weight) can help reduce high blood pressure and total blood cholesterol. It can also help control diabetes in some people. If you can't lose weight on your own, talk to a doctor, registered dietitian (R.D.), or nutritionist licensed or certified by the state (an L.D. or C.N.).

If you're overweight, losing 10 to 20 pounds can help lower your heart disease risk.

Try to reach a healthy weight — and stay there. To lose weight, most women should eat 1200 to 1500 calories a day, but not less than 1200. Most men should eat 1500 to 1800 calories a day, but not less than 1500. Losing one to two pounds per week is considered a healthy weight loss. (One pound of fat equals 3500 calories.)

Diabetes Mellitus — Diabetes is a disease in which the body doesn't make or respond properly to the hormone **insulin**. The body needs insulin to convert sugar, starches and other foods into energy. Diabetes is defined as a fasting plasma glucose (blood sugar) of 126 mg/dL or more measured on two occasions.

Type 2 diabetes, the most common form, usually appears in adults, often in middle age. It's becoming an increasing problem in children and adolescents, though. Obesity and physical inactivity are two risk factors for type 2 diabetes. In a mild form, it can go undetected for many years. Untreated diabetes can lead to a host of serious medical problems, including heart and blood vessel disease.

The other form of diabetes mellitus is type 1 or juvenile diabetes. It typically begins early in life. People with type 1 diabetes have a primary insulin deficiency. They must take insulin to stay alive.

Even when glucose levels are under control, diabetes greatly increases the risk of heart disease and stroke. In fact, most people with diabetes die of some form of heart or blood vessel disease. One reason for this is that diabetes is usually linked with low HDL ("good") cholesterol and high triglyceride levels. It also affects the blood vessels themselves. Many people with diabetes also have high blood pressure, increasing their risk even more.

It's critically important for people with diabetes to have regular medical check-ups to help control it. Work with your healthcare provider to change eating habits, control your weight and get regular exercise. Sometimes even drugs are needed. Take steps to control any other risk factors to reduce your risk. For example, blood pressure for people with diabetes should be **lower than 130/85 mm Hg.**

What risk factors can't be controlled?

Increasing Age — Heart attack can strike at any age. But the older you get, the more likely you are to develop heart disease or have a heart attack.

Male Sex — Men have a greater risk of heart attack than women, and they have attacks earlier in life. Women's risk rises after menopause but never reaches men's risk levels.

Race — African Americans have more severe high blood pressure than Caucasians and a higher risk of heart disease. Compared with Caucasians, heart disease risks are high among Mexican Americans, American Indians and native Hawaiians. This is partly due to higher rates of obesity and diabetes.

Heredity (Family History) — If your brother, father or grandfather had a heart attack before age 55, or your sister, mother or grandmother had one before age 65, you may be at risk, too. If you've had a heart attack, you're at higher risk of having a second attack.

Some families have a genetic condition that raises blood cholesterol or triglyceride levels. High blood pressure or diabetes may run in the family. Of course, a family's lifestyle also may contribute to heart disease risk. For example, family members may be overweight, physically inactive, or smoke or eat large amounts of foods high in cholesterol and saturated fat.

Most people with a strong family history of heart disease have at least one other risk factor. **Because you can't control your family history, it's even more important to treat and control any other risk factors you have.**

What other factors can affect the risk of heart disease?

Individual Response to Stress — We all feel stress, but we feel it in different amounts and react in different ways. Too much stress over a long time, and unhealthy responses to it, may create health problems in some people. For example, people under stress may overeat, start smoking or smoke more than they otherwise would.

Find healthy ways to handle stress. If you think your reaction to stress may be hurting your health or increasing your risk factors, do something about it. Work with your healthcare provider to monitor and control your risk factors. Make a major effort to stop smoking, stay at a healthy weight, and eat foods low in saturated fat, *trans* fat and cholesterol.

Excessive Alcohol — Drinking too much alcohol raises blood pressure, can cause heart failure and can lead to stroke.

Women should not have an average of more than one alcoholic drink a day. Men should not have more than two drinks a day. One drink is defined as $1^1/_2$ fluid ounces (fl oz) of 80-proof spirits (such as bourbon, Scotch, vodka, gin, etc.), 1 fl oz of 100-proof spirits, 4 fl oz of wine, or 12 fl oz of beer.

If you don't drink, don't start. And if you drink, do so in moderation. To help prevent birth defects, **pregnant women should not drink alcohol in any form.**

Some Illegal Drugs — Intravenous drug abuse carries a high risk of infections of the heart (endocarditis) and stroke. Cocaine use has been linked to strokes and heart attacks. Some have been fatal even in first-time users.

Aging and Menopause — As women age, their risk of heart disease and stroke begins to rise and keeps rising. If menopause is caused by surgery to remove the uterus and ovaries, the risk rises more sharply. If menopause occurs naturally, the risk rises more slowly.

For decades, millions of women have used estrogen replacement therapy (ERT) after menopause. ERT is approved to help relieve symptoms like "hot flashes" and to protect against osteoporosis, a crippling bone disease. Progestin is usually added to prevent uterine cancer in women who still have a uterus. This combination is called hormone replacement therapy (HRT).

For women who don't have cardiovascular disease, many observational studies have suggested that ERT and HRT may help reduce the risk of heart disease. However, ERT and HRT can have some negative effects, and some studies suggest increased risk. Major clinical trials now under way will help us better understand the benefits and risks of hormone use in healthy women.

If you've gone through menopause or had your ovaries removed, you may be considering ERT or HRT. It's important to consult your healthcare provider. You need to make sure your decision is based on the best currently available information about the health effects of these therapies.

For women who already have cardiovascular disease

- Estrogen replacement therapy (ERT) or hormone replacement therapy (HRT) should not be started to prevent another cardiovascular event.
- The decision to continue or stop ERT or HRT in women with CVD who have been undergoing long-term estrogen therapy should be based on established noncoronary benefits and risks and patient preference.
- If a woman develops an acute CVD event, such as a heart attack or stroke, or is immobilized while undergoing ERT or HRT, there's an increased risk of blood clots. To minimize this risk, it's prudent to consider discontinuing ERT/HRT or to consider anticoagulant treatment while she is hospitalized. Reinstating ERT or HRT should be based on established noncoronary benefits and risks, as well as patient preference.

For women who do not have cardiovascular disease

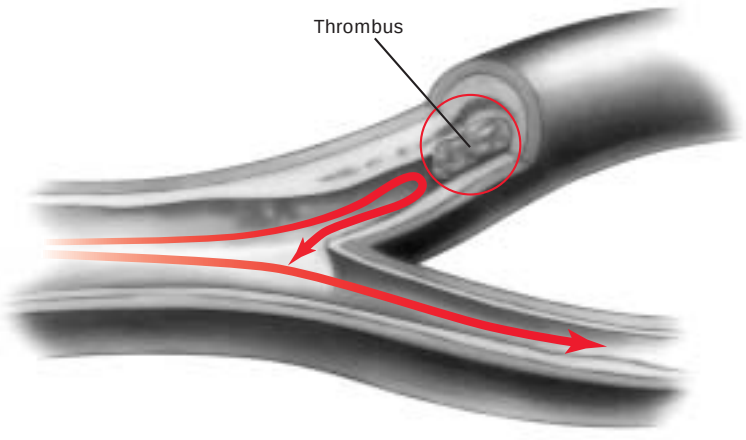
- Firm clinical recommendations for primary prevention await the results of ongoing, randomized clinical trials.
- There's not enough data to suggest that ERT or HRT should be started for the sole purpose of preventing the first CVD event.
- Starting and continuing ERT or HRT should be based on established noncoronary benefits and risks, possible coronary benefits and risks, and patient preference.

Stroke

What is a stroke?

Stroke is a cardiovascular disease. It affects the blood vessels that supply blood to the brain.

A stroke occurs when a blood vessel that brings oxygen and nutrients to the brain bursts or is clogged by a blood clot or some other particle. Because of this rupture or blockage, part of the brain doesn't get the blood and oxygen it needs. Deprived of oxygen, nerve cells in the affected area of the brain can't work and die within minutes. And when nerve cells can't work, the part of the body they control can't work either. The devastating effects of stroke are often permanent, because dead brain cells aren't replaced.



When an artery to the brain becomes blocked, a stroke can occur.

What are the types of stroke?

There are four main types of stroke. Two are caused by blood clots or other particles (ischemic strokes), and two by bleeding (hemorrhage). **Cerebral thrombosis** and **cerebral embolism** are caused by clots or particles that plug an artery. They account for about 70–80 percent of all strokes. Ruptured blood vessels cause **cerebral** and **subarachnoid hemorrhages**. These (bleeding) strokes have a much higher fatality rate than strokes caused by clots.

Cerebral thrombosis is the most common stroke. It occurs when a blood clot (thrombus) forms and blocks blood flow in an artery bringing blood to part of the brain. Blood clots usually form in arteries damaged by fatty buildups, called atherosclerosis.

Cerebral thrombotic strokes often occur at night or first thing in the morning, when blood pressure is low. They're often preceded by a transient ischemic attack, also called a TIA or "mini-stroke." (For more information about TIAs, see page 47.)

Cerebral embolism occurs when a wandering clot or some other particle (an embolus) forms away from the brain, usually in the heart. The bloodstream carries the clot until it lodges in an artery leading to or in the brain and blocks the flow of blood.

Most of these emboli are blood clots that form during atrial fibrillation. In this disorder the two small upper chambers of the heart (the atria) quiver instead of beating effectively. Some blood isn't pumped completely out of them when the heart beats, so it pools and clots. When a blood clot enters the circulation and lodges in a narrowed artery of the brain, a stroke occurs.

A subarachnoid hemorrhage occurs when a blood vessel on the brain's surface ruptures and bleeds into the space between the brain and the skull (but not into the brain itself).

Another type of stroke occurs when a defective artery in the brain bursts, flooding the surrounding tissue with blood. This is a cerebral hemorrhage.

Hemorrhage (or bleeding) from an artery in the brain can be caused by a head injury or a burst aneurysm. Aneurysms are blood-filled pouches that balloon out from weak spots in the artery wall. They're often caused or made worse by high blood pressure. If an aneurysm bursts in the brain, it causes a hemorrhagic stroke.

Strokes affect people in different ways, depending on the type of stroke, the area of the brain affected and the extent of the brain injury.

When a cerebral or subarachnoid hemorrhage occurs, the loss of a constant blood supply means some brain cells can no longer work. Accumulated blood from the burst artery also may press on the surrounding brain tissue and interfere with how the brain works. Severe or mild symptoms can result, depending on the amount of pressure.

The amount of bleeding determines the severity of cerebral hemorrhages. In many cases, people with cerebral hemorrhages die of increased pressure on their brains. But those who live tend to recover much more than those who've had strokes caused by a clot. The reason is that when a blood vessel is blocked, part of the brain dies — and the brain doesn't regenerate. But when a blood vessel in the brain bursts, pressure from the blood compresses part of the brain. If the person survives, gradually the pressure goes away. Then the brain may regain some of its former function.

What are the effects of stroke?

Stroke affects different people in different ways. It depends on the type of stroke, the area of the brain affected and the extent of the brain injury. Brain injury from a stroke can affect the senses, motor activity, speech and the ability to understand speech. It can also affect behavioral and thought patterns, memory and emotions. Paralysis or weakness on one side of the body is common.

A stroke survivor may cry easily or have sudden mood swings, often for no apparent reason. This is called **emotional lability**. Laughing uncontrollably also may occur but isn't as common as crying. Depression is common, as stroke survivors may feel less than "whole."

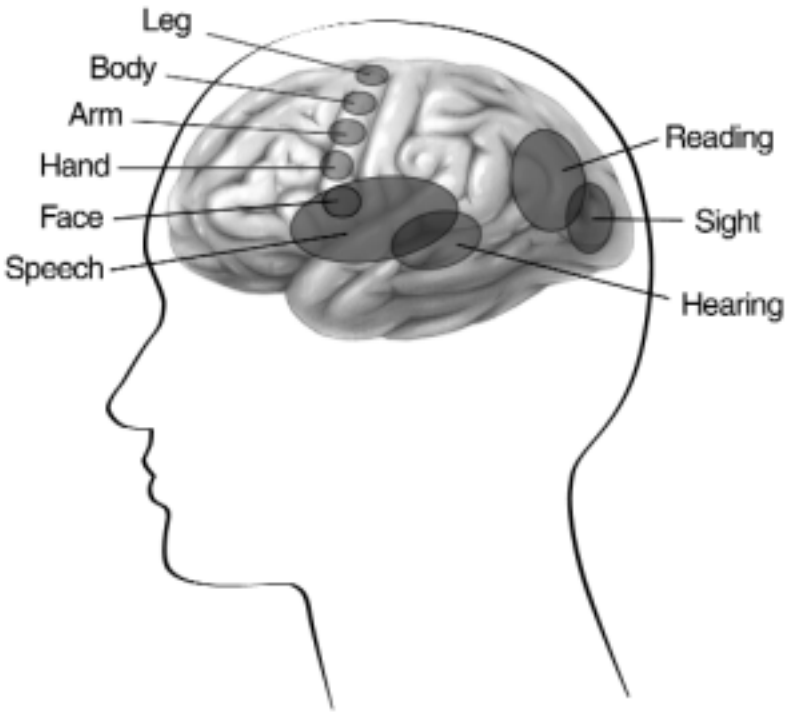
Stroke often causes people to lose mobility and/or feeling in an arm or leg, or suffer dimness of sight on one side. The loss of feeling or visual field results in a loss of awareness, so stroke survivors may forget or ignore their weaker side. This problem is called **neglect**. As a result, they may ignore items on their affected side and have trouble reading. They also may dress only one side of their bodies and think they're fully dressed. Bumping into furniture or door jambs is also common. One-sided neglect is most common in those with injury to the brain's right hemisphere.

A stroke can affect seeing, touching, moving and thinking, so a person's perception of everyday objects may be changed. Stroke survivors may not be able to recognize and understand familiar objects the way they did before. When vision is affected, objects may look closer or farther away than they really are. This causes survivors to have spills at the table or collisions when they walk.

Stroke usually doesn't cause hearing loss, but survivors may have problems understanding speech. They also may have trouble saying what they're thinking. This is called **aphasia**. Aphasia affects the ability to talk, listen, read and write. It's most common when a stroke weakens the body's right side.

A related problem is that a stroke can affect muscles used in talking (those in the tongue, palate and lips). Speech can be slowed, slurred or distorted, so stroke survivors can be hard to understand. This is called **dysarthria**. It may require the help of a speech expert. Chewing and swallowing food also can be a problem. This is called **dysphagia**, and it can occur when one side of the mouth is weak. One or both sides of the mouth can lack feeling, increasing the risk of choking.

Finally, a stroke can affect the ability to think clearly. It may be hard to plan and carry out even simple activities. Stroke survivors may not know how to start a task, confuse the sequence of logical steps in tasks, or forget how to do tasks they've done many times before.



Different parts of the brain control different functions.

Stroke Warning Signs and Actions

Know the warning signs of stroke.

- Sudden numbness or weakness of the face, arm or leg, especially on one side of the body.
- Sudden confusion, trouble speaking or understanding.
- Sudden trouble seeing in one or both eyes.
- Sudden trouble walking, dizziness, loss of balance or coordination.
- Sudden, severe headache with no known cause.

Take action in an emergency.

If a stroke occurs, here are the most important things to remember:

- Not all the warning signs occur in every stroke. Don't ignore signs of stroke, even if they go away!
- Check the time. When did the first symptom start? You'll be asked this important question later.
- If you or someone with you has one or more stroke symptoms that last more than a few minutes, don't delay! Immediately call 9-1-1 or the emergency medical services (EMS) number so an ambulance (ideally with advanced life support) can quickly be sent for you.
- Many clinical trials led to advances in stroke prevention and treatment. For example, tissue plasminogen activator, a clot-busting drug known as tPA, is a major advance. If it's given within three hours of the onset of an ischemic stroke, it can reduce long-term disability.
- **Stroke is a medical emergency. Every second counts!**

Responding quickly and properly will allow you to benefit from the stroke chain of survival.

- **Rapid recognition and reaction to stroke warning signs.** Recognize the warning signs and note when they began. Call 9-1-1 immediately!
- **Rapid start of pre-hospital care.** Receive early assessments and pre-hospital care by emergency medical personnel.
- **Rapid Emergency Medical Services system transport and hospital pre-notification.**
- **Rapid diagnosis and treatment in the hospital.**

A sudden, severe headache may be a sign of stroke.

What is a TIA or transient ischemic attack?

Any of the above symptoms may be temporary and last only a few minutes. This may be due to a transient ischemic attack (TIA). A TIA is a "little stroke" or "mini-stroke" that produces stroke-like symptoms but no lasting damage.

Most strokes aren't preceded by TIAs. However, more than a third of the people who've had one or more TIAs will later have a stroke. In fact, a person who's had one or more TIAs is many times more likely to have a stroke than someone of the same age and sex who hasn't.

TIAs are more useful for predicting **if** a stroke will occur than **when** one will happen. They can occur days, weeks or even months before a major stroke. In about half of the cases, the stroke occurs within one year of the TIA.

TIAs occur when a blood clot temporarily clogs an artery, and part of the brain doesn't get the blood it needs. The symptoms occur rapidly and last a relatively short time. Most TIAs last less than five minutes. The average is about a minute. Unlike stroke, when a TIA is over, there's no injury to the brain.

The usual TIA symptoms are the same as those of stroke, only temporary.

It's very important to recognize the warning signs of a TIA or stroke. The short duration of these symptoms and lack of permanent brain injury is the main distinction between TIA and stroke.

TIAs are extremely important predictors of stroke. **Don't ignore them!** If symptoms appear, CALL 9-1-1 TO GET MEDICAL ATTENTION IMMEDIATELY. A doctor should determine if a TIA or stroke has occurred, or if it's another medical problem with similar symptoms. Some examples are seizure, fainting, migraine headache, or general medical or cardiac condition. Prompt medical or surgical attention to these symptoms could prevent a fatal or disabling stroke from occurring.

Risk Factors for Stroke

Risk factors are traits and lifestyle habits that increase the risk of disease. Extensive clinical and statistical studies have identified several factors that increase the risk of stroke. Most of them can be modified, treated or controlled. Some can't. The more risk factors a person has, the higher the chances that he or she will have a stroke. The best way to prevent a stroke is to reduce stroke risk factors.

Controlling high blood pressure significantly reduces the risk of stroke. Often blood pressure can be controlled just by eating a healthier diet and maintaining a healthy weight. Drugs to control blood pressure are also available.

What risk factors for stroke can be controlled or treated?

High Blood Pressure — High blood pressure **(140/90 mm Hg or higher)** is the most important risk factor for stroke. It usually has no specific symptoms and no early warning signs. That's why everybody should have their blood pressure checked regularly.

Tobacco Use — Cigarette smoking is the No. 1 preventable risk factor for stroke. The nicotine and carbon monoxide in tobacco smoke reduce the amount of oxygen in the blood. They also damage blood vessel walls, making clots more likely to form. Using some kinds of birth control pills combined with smoking cigarettes greatly increases stroke risk. **If you smoke, get help to quit NOW!**

Diabetes Mellitus — Diabetes is defined as a fasting plasma glucose (blood sugar) of 126 mg/dL or more measured on two occasions. While diabetes is treatable, having it still increases a person's risk of stroke. Many people with diabetes also have high blood pressure, high blood cholesterol and are overweight. This increases their risk even more.

If you have diabetes, work closely with your doctor to manage it and control your other risk factors. For example, blood pressure for people with diabetes should be **lower than 130/85 mm Hg**.

Carotid or Other Artery Disease — The carotid arteries in the neck supply blood to the brain. A carotid artery narrowed by fatty deposits from atherosclerosis may become blocked by a blood clot. Carotid artery disease is also called carotid artery stenosis.

People with **peripheral artery disease** have a higher risk of carotid artery disease, which raises their risk of stroke. Peripheral artery disease is the narrowing of vessels carrying blood to leg and arm muscles. It's caused by fatty buildups in arteries (atherosclerosis).

Atrial Fibrillation — This heart rhythm disorder raises the risk for stroke. The heart's upper chambers quiver instead of beating effectively, which lets the blood pool and clot. If a clot breaks off, enters the bloodstream and lodges in an artery leading to the brain, a stroke results. Patients with atrial fibrillation can be treated with drugs that keep clots from forming.

Other Heart Disease — People with coronary heart disease or heart failure have more than twice the risk of stroke as those with hearts that work normally. Dilated cardiomyopathy (an enlarged heart), heart valve disease and some types of congenital heart defects also raise stroke risk.

Transient Ischemic Attacks (TIAs) — TIAs are "mini-strokes" that produce stroke-like symptoms but no lasting damage. Recognizing and treating TIAs can reduce your risk of a major stroke. It's very important to recognize the warning signs of a TIA or stroke. Call 9-1-1 to get medical help immediately if they occur! People with TIAs are usually treated with drugs that keep clots from forming.

Certain Blood Disorders — A high red blood cell count thickens the blood and makes clots more likely. This raises the risk of stroke. Doctors may treat this problem by removing blood cells or prescribing "blood thinners."

Sickle cell anemia is a genetic disorder that mainly affects African Americans. "Sickled" red blood cells are less able to carry oxygen to the body's tissues and organs. They also tend to stick to blood vessel walls. This can block arteries to the brain and cause a stroke.

High Blood Cholesterol — A high blood cholesterol level (240 mg/dL or higher) is bad because cholesterol can build up in the walls of arteries. And narrowed arteries are more likely to become blocked, causing a heart attack or stroke. **LDL ("bad") cholesterol** is deposited in artery walls, increasing the buildup of plaque. High levels of LDL cholesterol (greater than 100 mg/dL) and triglycerides (blood fats) directly increase the risk of stroke in people with previous coronary heart disease, ischemic stroke or transient ischemic attack (TIA). Low levels of **HDL ("good") cholesterol** (less than 40 mg/dL) also may raise stroke risk.

Physical Inactivity and Obesity — Being inactive, obese or both can increase your risk of high blood pressure, high blood cholesterol, diabetes, heart disease and stroke. So go on a brisk walk, take the stairs, and do whatever you can to make your life more active. Try to get a total of at least 30 minutes of physical activity on most days.

Excessive Alcohol — Drinking an average of more than one alcoholic drink a day for women or more than two drinks a day for men raises blood pressure, can cause heart failure and can lead to stroke. One drink is defined as 1 1/2 fluid ounces (fl oz) of 80-proof spirits (such as bourbon, Scotch, vodka, gin, etc.), 1 fl oz of 100-proof spirits, 4 fl oz of wine, or 12 fl oz of beer.

Some Illegal Drugs — Intravenous drug abuse carries a high risk of stroke and endocarditis (infection of the heart's lining or valves). Cocaine use has been linked to strokes and heart attacks. Some have been fatal even in first-time users.

What risk factors can't be controlled?

Increasing Age — People of all ages, including children, have strokes. But the older you are, the greater your risk for stroke. The chance of having a stroke more than doubles for each decade of life after age 55.

Sex (Gender) — Stroke is more common in men than in women. In most age groups, more men than women will have a stroke in a given year. However, women account for more than half of all stroke deaths. Women who are pregnant have a higher stroke risk. So do women taking birth control pills who also smoke or have high blood pressure or other risk factors.

Heredity (Family History) and Race — Your stroke risk is greater if a parent, grandparent, sister or brother has had a stroke. African Americans have a much higher risk of death from a stroke than Caucasians do. This is partly because blacks have higher risks of high blood pressure, diabetes and obesity.

Prior Stroke or Heart Attack — Someone who has had a stroke is at much higher risk of having another one. If you've had a heart attack, you're at higher risk of having a stroke, too.

What other factors can affect the risk of stroke?

Individual Response to Stress — We all feel stress, but we feel it in different amounts and react in different ways. Too much stress over a long time, and unhealthy responses to it, may create health problems in some people. For example, people under stress may overeat, start smoking or smoke more than they otherwise would.

> Find healthy ways to handle stress. Make a major effort to stop smoking, stay at a healthy weight, and eat foods low in saturated fat, *trans* fat, cholesterol and sodium.

Aging and Menopause — See the discussion of aging and menopause on pages 42–43.

Birth Control Pills — Today's low-dose oral contraceptives carry a much lower risk of heart disease and stroke than the early Pill did. However, women on the Pill who smoke or have high blood pressure or other risk factors are at higher risk.

Taking oral contraceptives and smoking greatly increases the risk of stroke.

> If you take oral contraceptives, get regular check-ups. Birth control pills can be taken for years with no harmful effects. Still, yearly checks of blood pressure, triglycerides and glucose are very important.

Diagnosing Stroke

What information does a doctor need?

When someone has shown symptoms of a TIA or stroke, a doctor must first gather data to make a diagnosis. He or she will take a careful history of recent events as well as a general medical history for diabetes, hypertension, heart and blood vessel disease and other neurological diseases. He or she will measure blood pressure in both arms, test the pulse, check the heart, listen for bruits (abnormal sounds) over the neck and collarbone, check the eyes and give a neurological exam.

A doctor might use many tests in a neurological exam. For example, doctors may test patients' level of consciousness, orientation, memory and emotional control. Some doctors test patients by having them stand motionless with feet together, arms outstretched and eyes closed. Testing for facial paresis (paralysis) by having patients bare their gums and stick out their tongues is also sometimes done. Hearing might be tested by rubbing the thumb and forefinger together about 12 inches from the ear. Vision is tested by having patients read newsprint using one eye at a time. Visual fields can be tested by having a person cover one eye and look into the doctor's opposite eye. (The doctor will then bring a thumb or small object from the side and ask the patient to signal when it becomes visible.) The perception of pain and light touch, muscular strength and deep tendon reflexes are also commonly tested.

Different doctors use different tests; the tests listed here are only examples of what **might** be done.

After these basic tests, many doctors will request laboratory tests showing a complete blood count, blood sugar, urea and electrolytes. Some people may be given an electrocardiogram.

What tests reveal a stroke?

Identifying stroke warning signals is one way to diagnose a stroke. But proper diagnosis doesn't stop there. Other tests may be run, because the symptoms may not necessarily result from a stroke. For example, a brain tumor can produce similar symptoms. A doctor must eliminate other possibilities before making a diagnosis.

Remarkable advances in modern technology now make it possible to examine how the brain looks, works and gets its blood supply. These tests can outline the affected part of the brain and help define the problem created by stroke. Most of these tests are safe, painless and can be done as an outpatient.

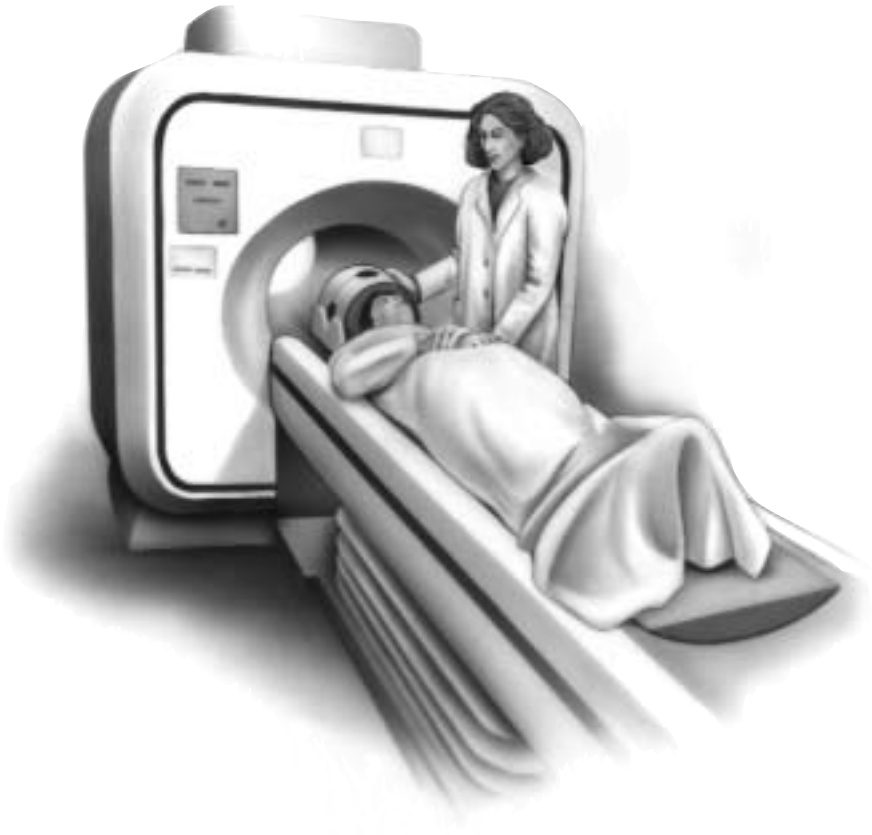
These tests fall into three categories. Tests that image the brain create images that look similar to ordinary X-rays. Tests that measure the electrical activity of the brain give useful information about how it's working and pinpoint areas where it's working abnormally. Finally, blood flow tests measure flow and detect blockages in blood vessels. They're useful in revealing areas of significant atherosclerosis in carotid arteries. A doctor must decide on a case-by-case basis whether such tests will be useful, and if so, which ones to use.

What brain imaging tests are done?

The **computerized axial tomographic** (CT or CAT) **scan** uses X-rays to generate an image of the brain. Doctors use CT to find out if a stroke has occurred and, if so, what kind it was: **ischemic** (result of blockage) or **hemorrhagic** (result of bleeding). CT scanning takes from 20 minutes to an hour to complete.

Magnetic resonance imaging (MRI) **scanning** uses a giant magnet to generate an image. The patient is placed in a magnetic field, then the head is subjected to bursts of energy of a known frequency. The brain cells' response to these bursts of energy is detected as signals that ultimately generate an image of the brain. MRI can give very accurate images of the brain. It's used to determine the presence, location and size of aneurysms and arterio-venous malformations, which are potential sources for hemorrhagic stroke.

In **radionuclide angiography** (nuclear brain scan), radioactive compounds are injected into a vein in the arm; the bloodstream then carries them toward the head. As the radioactive compound circulates in the blood, it constantly emits bursts of radiation. When the radioactive compound reaches the brain, these bursts of radiation are detected and used to form an image of the brain. This imaging procedure can show areas where the brain has been deprived of blood flow and is damaged.



Magnetic resonance imaging.

What tests show the brain's electrical activity?

Two basic tests show the electrical activity of the brain: an electroencephalogram (EEG) and an evoked response test. In an **electro-encephalogram**, small metal disks (electrodes) are placed at strategic locations on a person's scalp. The electrodes detect the electrical activity (impulses), which are then transcribed to paper. The test shows such characteristics as intensity (the size of the impulse), duration (the width of the impulse), frequency (how often impulses occur during a given time) and location (what region of the brain produces these impulses). By observing these characteristics on the EEG, a physician can gain valuable information about underlying problems in the brain.

Evoked responses measure the brain's ability to process and react to different sensory stimuli. A doctor evokes a visual response by flashing a light or checkerboard pattern in front of a patient. For auditory evoked responses, a doctor makes a sound in one of the patient's ears. For bodily evoked responses, one of the nerves in an arm or leg is electrically stimulated. The responses from any of these sensory stimuli can indicate abnormal areas of the brain.

What tests show blood flow?

The **Doppler ultrasound** test uses high-frequency sound waves to detect blockages in the carotid artery. A Doppler probe or instrument capable of generating ultrasound waves is placed on the neck, very close to the carotid artery. Ultrasound

waves from the probe travel through the neck and bounce off the moving blood cells. The reflected sound wave, now returning to the probe at a different frequency, is then detected by the same probe. The change in frequency of the sound waves relates to the speed of the blood cells and thus the blood flow.

In **carotid phonoangiography**, a sensitive microphone is placed on the neck, very close to the carotid artery, to record sounds. In a normal artery, blood flows in a smooth and controlled manner. However, blockages, such as those caused by atherosclerosis, cause the blood flow to become turbulent. This turbulent blood flow can create a sound, called a **bruit** (BROO-ee), that can be detected and registered by the microphone. A bruit indicates a blockage in the carotid artery and is cause for more tests.

Finally, **digital subtraction angiography** (DSA) images the brain's major blood vessels. A thin plastic tube (a catheter) is inserted into a major leg artery and advanced through the body's major vessels until it reaches the brain's blood vessels. A contrast dye is injected through the catheter and allowed to circulate in the bloodstream. Then an X-ray machine quickly takes a series of pictures of the head and neck. The images track the contrast dye as it moves through the brain's blood vessels. This imaging technique lets the doctor identify and localize the source of stroke.

Treatment and Rehabilitation

How are strokes treated?

Surgery, drugs, acute hospital care and rehabilitation are all accepted stroke treatments.

When a neck artery is partially blocked by a fatty buildup, surgery might be used to remove the plaque. This is called **carotid endarterectomy.**

Cerebral angioplasty is a new, experimental technique. Balloons, stents and coils are used to treat some types of problems with the brain's blood vessels. Its widespread use will require more study of its safety and effectiveness.

The Food and Drug Administration has approved the clot-busting drug **tissue plasminogen activator** (tPA) to treat stroke. This is a major advance. Now physicians have an approved treatment for acute strokes caused by blood clots, which constitute 70–80 percent of all strokes. Not every stroke patient should be treated with tPA, particularly those having a hemorrhagic stroke. tPA carries a risk of bleeding in the brain, but its benefits outweigh the risks when an experienced doctor uses it properly.

tPA is effective only if given promptly. **For maximum benefit, the therapy must be started within three hours of the onset of stroke symptoms. That's why it's so critical that medical professionals and the public recognize stroke as a medical emergency and respond immediately.**

Sometimes treating a stroke means treating the heart. The reason is that various kinds of heart disease can contribute to the risk of stroke. For example, damaged heart valves may need to be treated surgically or with anti-clotting drugs to reduce the chance of clots forming around them. Blood clots also can form in the hearts of people with atrial fibrillation. This is a type of arrhythmia (abnormal heart rhythm). If clots form, there's a chance they could travel to the brain and cause a stroke.

Can stroke survivors be rehabilitated?

Stroke is the No. 3 killer in the United States. It's also a leading cause of serious, long-term disability. Many survivors are left with mental and physical disabilities.

Most gains in a person's ability to function in the first 30 days after a stroke are due to spontaneous recovery. Still, rehabilitation is important. For the most part, successful rehabilitation depends on

- the extent of the brain injury.
- the survivor's attitude.
- the rehabilitation team's skill.
- the cooperation of family and friends.

People with the least impairment are likely to benefit the most. Even when improvement is slight, rehabilitation may still mean the difference between returning home or staying in an institution.

The goal of rehabilitation is to reduce dependence and improve physical abilities. Often old skills have been lost and new ones are needed. It's also important to maintain and improve a person's physical condition when possible.

Rehabilitation begins early as nurses and other hospital personnel work to prevent such secondary problems as stiff joints, bedsores and pneumonia. These can result from being in bed for a long time.

A person's family has a key role in rehabilitation. A caring and able spouse or partner can be one of the most important positive factors in rehabilitation. Family members' knowledge also helps a lot. Family members need to understand what the stroke survivor has been through and how disabilities can affect the person. They can handle the situation more easily if they know what to expect and how to handle problems that arise after the person leaves the hospital.

For a stroke survivor, the goal of rehabilitation is to be as independent and productive as possible.

Congenital Heart and Blood Vessel Defects

What is a congenital heart or blood vessel defect?

Congenital means inborn or existing at birth. Among the terms you may hear are congenital heart defect, congenital heart disease and congenital cardiovascular disease. The word "defect" is more accurate than "disease." A congenital heart defect occurs when the heart or blood vessels near the heart don't develop normally before birth.

Congenital heart defects are present in almost one percent of live births. They're the most common congenital malformations in newborns. **In most cases scientists don't know why they occur.** Sometimes a viral infection causes serious problems. German measles (rubella) is an example. If a woman contracts German measles while pregnant, it can interfere with how the baby's heart develops or produce other malformations. Other viral diseases also may cause congenital defects.

Heredity sometimes has a role in congenital cardiovascular defects. More than one child in a family may have a congenital cardiovascular defect, but this is rare. Certain conditions affecting multiple organs, such as Down's syndrome, can involve the heart, too. Some prescription drugs and over-the-counter medicines, as well as alcohol and "street" drugs, may increase the risk of having a baby with a heart defect. Researchers are studying other factors.

What are the types of congenital defects?

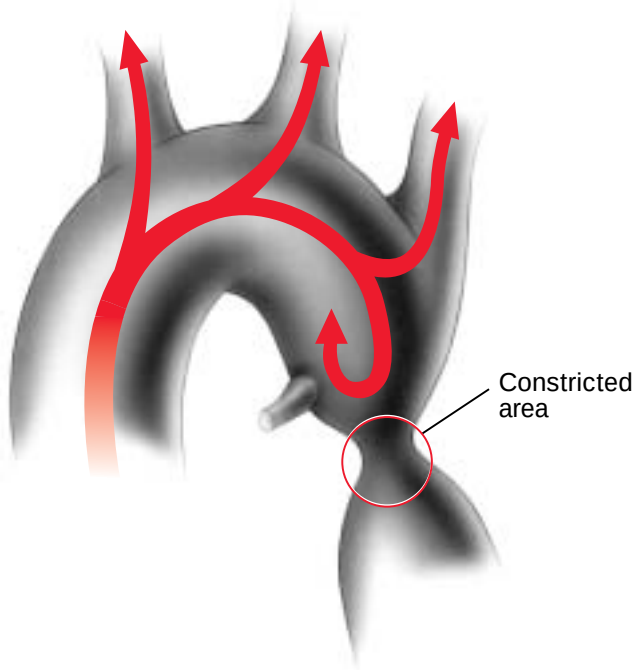
Most heart defects either obstruct blood flow in the heart or vessels near it, or cause blood to flow through the heart in an abnormal

way. Rarely defects occur in which only one ventricle (single ventricle) is present, or both the pulmonary artery and aorta arise from the same ventricle (double outlet ventricle). A third rare defect occurs when the right or left side of the heart is incompletely formed (hypoplastic heart).

Several congenital defects of each type are described here. For detailed information and more examples, see our Web site, **americanheart.org**. Information in Spanish for some defects is also available there.

Patent ductus arteriosus (PDA)

This defect lets blood mix between the pulmonary artery and the aorta. Before birth there's an open passageway (the ductus arteriosus) between these two blood vessels. Normally this closes within a few hours of birth. When this doesn't happen, some blood that should flow through the aorta and on to nourish the body returns to the lungs. A ductus that doesn't close is common in premature infants but rare in full-term babies.



Coarctation of the aorta restricts blood flow to the lower body.

Obstruction defects

An obstruction is a narrowing that partially or completely blocks the flow of blood. Obstructions called **stenoses** can occur in heart valves, arteries or veins.

The three most common forms of obstructed blood flow are pulmonary valve stenosis, aortic valve stenosis and coarctation of the aorta.

In **pulmonary stenosis (PS)** the pulmonary or pulmonic valve is defective and doesn't open properly. This valve is between the right ventricle and the pulmonary artery. Normally it opens to let blood flow from the right ventricle to the lungs. A defective pulmonary valve that doesn't open properly is called **stenotic**. This forces the right ventricle to pump harder than normal to overcome the obstruction.

In **aortic stenosis (AS)** the aortic valve, between the left ventricle and the aorta, is narrowed. The heart has difficulty pumping blood to the body. Aortic stenosis occurs when the aortic valve didn't form properly. A normal valve has three leaflets (cusps) but a stenotic valve may have only one cusp (unicuspid) or two cusps (bicuspid), which are thick and stiff.

In **coarctation of the aorta ("Coarct")** the aorta is pinched or constricted. This obstructs blood flow to the lower body and increases blood pressure above the constriction.

Septal defects

Some congenital heart defects let blood flow between the heart's right and left chambers. This happens when a baby is born with an opening between the wall **(septum)** that separates the right and left sides of the heart. This defect is sometimes called "a hole in the heart."

The two most common types of such openings are atrial septal defect and ventricular septal defect. Two variations are Eisenmenger's complex and atrioventricular canal defect.

In **atrial septal defect (ASD)** an opening exists between the heart's two upper chambers. This lets some blood from the left atrium (blood that's already been to the lungs) return via the hole to the right atrium instead of flowing through the left ventricle, out the aorta and to the body.

In **ventricular septal defect (VSD)**, an opening exists between the heart's two lower chambers. Some blood that's returned from the lungs and been pumped into the left ventricle flows to the right ventricle through the hole instead of being pumped into the aorta. Because the heart has to pump extra blood and is overworked, it may enlarge.

Eisenmenger's complex is a ventricular septal defect coupled with pulmonary high blood pressure, the passage of blood from the right side of the heart to the left (right to left shunt), an enlarged right ventricle and a latent or clearly visible bluish discoloration of the skin called cyanosis. It may also include a malpositioned aorta that receives ejected blood from both the right and left ventricles (an overriding aorta).

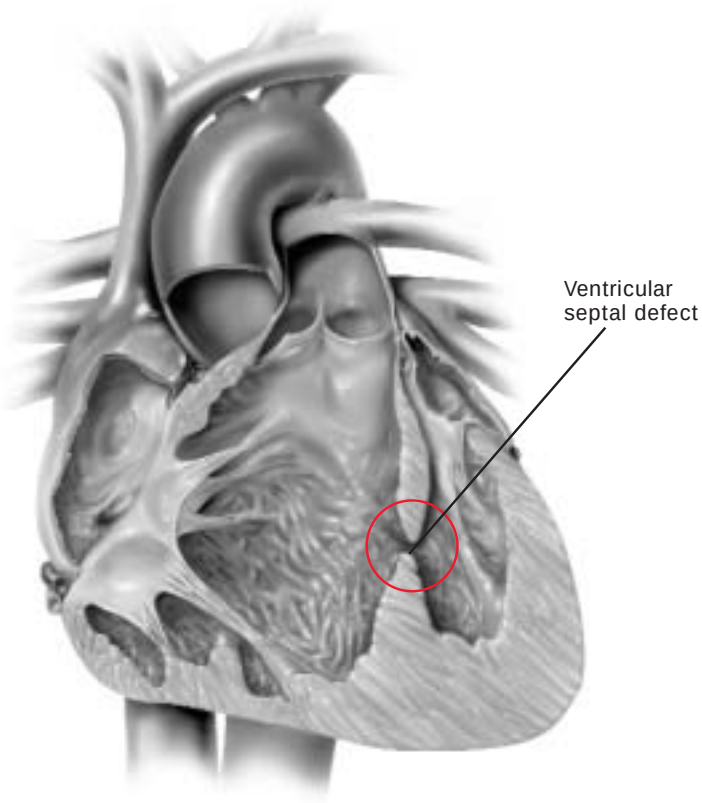
A less-common type of opening is the **atrioventricular (AV) canal defect**, also called **endocardial cushion defect** or **atrioventricular septal defect**. A large hole in the center of the heart exists where the wall between the upper chambers joins the wall between the lower chambers. Also, the tricuspid and mitral valves that normally separate the heart's upper and lower chambers aren't formed as individual valves. Instead, a single large valve forms that crosses the defect.

Cyanotic defects

Another type of heart defect is **congenital cyanotic heart defects**. In these defects, blood pumped to the body contains less oxygen than normal. This causes a condition called cyanosis, a blue discoloration of the skin. The term "blue babies" is often applied to infants with cyanosis.

Examples of cyanotic defects are tetralogy of Fallot, transposition of the great arteries, tricuspid atresia, pulmonary atresia, truncus arteriosus and total anomalous pulmonary venous connection.

Tetralogy of Fallot has four components. The two major ones are a large hole, or ventricular septal defect, that lets blood pass from the right to the left ventricle without going through the lungs; and a narrowing (stenosis) at



VSD allows blood to leak from one side of the heart to the other.

or just beneath the pulmonary valve. This narrowing partially blocks the blood flow from the heart's right side to the lungs. The other two components are the right ventricle is more muscular than normal; and the aorta lies directly over the ventricular septal defect.

In **transposition of the great arteries**, the positions of the pulmonary artery and the aorta are reversed. The aorta is connected to the right ventricle, so most of the blood returning to the heart from the body is pumped back out without first going to the lungs. The pulmonary artery is connected to the left ventricle, so most of the blood returning from the lungs goes back to the lungs again.

Infants born with transposition survive only if they have one or more connections that let oxygen-rich blood reach the body. One such connection may be an atrial septal defect or a ventricular septal defect. (See page 55.) Another may be a patent ductus arteriosus (PDA). (See page 54.)

In **tricuspid atresia**, there's no tricuspid valve. That means no blood can flow from the right atrium to the right ventricle. As a result, the right ventricle is small and not fully developed. The child's survival depends on there being an atrial septal defect and usually a ventricular septal defect. (See page 55.) Because the circulation is abnormal, the blood can't get enough oxygen, and the child looks blue (cyanotic).

In **pulmonary atresia**, no pulmonary valve exists, so blood can't flow from the right ventricle into the pulmonary artery and on to the lungs. The right ventricle acts as a blind pouch that may stay small and not well developed. The tricuspid valve is often poorly developed, too.

An opening in the atrial septum lets blood exit the right atrium. That allows venous (bluish) blood to mix with the oxygen-rich (red) blood in the left atrium. The left ventricle pumps this mixture of oxygen-poor blood into the aorta and out to the body. The baby appears blue (cyanotic) because there's less oxygen in the blood circulating through the arteries. The only source of lung blood flow is the patent ductus arteriosus (PDA). (See page 54.) If the PDA narrows or closes, the lung blood flow is reduced to critically low levels. This can cause very severe cyanosis.

Truncus arteriosus is a complex malformation where only one artery arises from the heart and forms the aorta and pulmonary artery. Surgery for this condition usually is required early in life. It includes closing a large ventricular septal defect within the heart, detaching the pulmonary arteries from the large common artery, and connecting the pulmonary arteries to the right ventricle with a tube graft. Children with truncus arteriosus need lifelong follow-up to see how well the heart is working.

Children with truncus arteriosus need lifelong follow-up to see how well the heart is working.

Total anomalous pulmonary venous (P-V) connection is another cyanotic defect. The pulmonary veins that bring oxygen-rich (red) blood from the lungs back to the heart aren't connected to the left atrium. Instead, the pulmonary veins drain through abnormal connections to the right atrium. In the right atrium, oxygen-rich (red) blood from the pulmonary veins mixes with venous (bluish) blood from the body. Part of this mixture passes through the atrial septum (atrial septal defect) into the left atrium. From there it goes into the left ventricle, to the aorta and out to the body. The rest of the poorly oxygenated mixture flows through the right ventricle, into the pulmonary artery and on to the lungs. The blood passing through the aorta to the body doesn't have enough oxygen, which causes the child to look blue (cyanotic).

Hypoplastic left heart syndrome

In hypoplastic left heart syndrome, the left side of the heart is underdeveloped — including the aorta, aortic valve, left ventricle and mitral valve. Blood returning from the lungs must flow through an atrial septal defect. (See page 55.) The right ventricle pumps the blood into the pulmonary artery, and blood reaches the aorta through a patent ductus arteriosus. (See page 54.)

Babies with this problem often seem normal at birth, but will come to medical attention within a few days as the ductus closes. They become ashen, have rapid and difficult breathing and have trouble feeding. Without treatment, this heart defect is usually fatal within the first days or months of life.

How does a doctor detect a heart defect?

Serious congenital heart defects are usually diagnosed at birth or during infancy. Sometimes a doctor hears an abnormal sound (a murmur) in the heart. In other babies, cyanosis is present.

Special tests are often needed. A chest X-ray gives information about a child's lungs and the heart's size and shape. An electrocardiogram (ECG or EKG) can show an abnormal heartbeat rhythm.

A Doppler echocardiogram is also usually used. An echocardiogram is a painless test that uses high-frequency sound waves to image the heart's internal structures. A Doppler test uses sound waves to measure blood flow. By combining these two tests, a doctor can learn about the heart's structure and function.

Sometimes an in-hospital test called a cardiac catheterization is required. Here a doctor inserts a catheter into a blood vessel in the groin and slowly advances it under X-ray guidance until it reaches the heart. This test can measure blood pressure and how much oxygen is in the blood of the heart chambers and vessels. It gives other information, too. A special fluid visible by X-ray also can be injected into the blood vessels or heart, and an X-ray motion picture can be made. This procedure can help define the heart defect.

How can a congenital heart defect be treated?

Many children with congenital heart and blood vessel defects may need medical treatment such as diuretics, digoxin or other drugs. Diuretics help the body excrete water and salts by promoting urination. Digoxin strengthens the heart's contractions, slows the heart rate and helps remove extra fluid from body tissues.

Some children may need surgery. The goal of surgery is to repair the defect as completely as possible and make circulation as normal as possible. Some children may need more than one surgical procedure.

The malformed part of the heart or blood vessel may be surgically repaired in several ways. Here are some examples:

- A ductus arteriosus can be closed by tying it.
- Stenotic valves can be widened, either by a balloon procedure during cardiac catheterization or by surgery.
- A narrowed segment of a blood vessel can be removed.
- Septal defects can be closed by sewing the defect shut or by sewing a patch (made of durable, synthetic material) over the hole.
- In babies with transposition of the great arteries, the major arteries can be switched.
- A shunt can be used to form a passage between blood vessels to divert blood from one part of the heart to another.
- In some cases, treatment with special equipment in the cardiac catheterization laboratory is effective.

For detailed information and more examples, see our Web site, **americanheart.org**.

Some congenital heart defects don't require surgery. Drugs may be used to prevent complications, relieve symptoms or both. Sometimes medical treatment is used for awhile and surgery performed later.

Most people with congenital heart defects, before and after treatment, are at risk for getting an infection on the heart's inner lining, valves or blood vessels (endocarditis). (See page 62.) To help prevent this, they'll need to take antibiotic drugs before certain dental and surgical procedures.

Rheumatic Heart Disease

What is rheumatic heart disease?

Rheumatic heart disease is a condition in which the heart valves are damaged by rheumatic fever. Rheumatic fever begins with a strep throat from streptococcal infection.

Rheumatic fever is an inflammatory disease. It can affect many of the body's connective tissues — especially those of the heart, joints, brain or skin. When rheumatic fever permanently damages the heart, the damage is called rheumatic heart disease.

Anyone can get acute rheumatic fever, but it usually occurs in children 5 to 15 years old. The rheumatic heart disease that results can last for life.

How can you tell if you have strep throat?

The most common symptoms of strep throat are the sudden onset of a sore throat (particularly with painful swallowing), fever, and tender, swollen glands under the jaw angle.

Laboratory tests can confirm inflammation and identify a streptococcal infection. There's no specific lab test for rheumatic fever. Strep throat infections can be detected by throat cultures or more rapid laboratory antigen detection tests.

Throat cultures, blood antibody tests and other blood tests are also used to find out if a recent strep infection could have triggered rheumatic fever.

What happens in acute rheumatic fever?

The first symptoms are a high fever that lasts 10 to 14 days and arthritic pain and soreness that moves from one joint to another. In acute attacks of rheumatic fever, joints often swell and become red and hot.

A child suffering from rheumatic fever may have shortness of breath or chest pains. These symptoms indicate the heart is affected. Other signs include tiring easily, eating poorly or paleness. A doctor examining the child may hear an abnormal heart murmur or find that the heart is enlarged. Acute inflammation of the heart is a serious condition. It requires direct and some-times lengthy medical care.

What tests are used to detect heart damage?

Chest X-rays and an electrocardiogram are two common tests to find out if the heart has been affected.

Echocardiography sends sound waves into the chest to rebound from the heart's walls and valves. The recorded waves show the shape, texture and movement of the valves. They also show the size of the heart chambers and how well they're working. This technique doesn't hurt or pose a risk to people.

Doctors also use cardiac catheterization to study heart damage. (See pages 15 and 57.)

What happens when rheumatic heart disease damages a heart valve?

A damaged heart valve either doesn't fully close **(insufficiency)** or doesn't fully open **(stenosis)**.

A heart valve that doesn't close properly lets blood leak back into the chamber from which it was pumped. This is called **regurgitation** or leakage. With the next heartbeat, regurgitated blood flows through the valve and mixes with blood that flows normally. This extra volume of blood puts more strain on the heart muscle. A doctor can diagnose an insufficient heart valve by listening to the heart and verify it by echocardiography.

When a valve doesn't open enough, the heart must pump harder than normal to force blood through the narrowed opening. Usually there are no symptoms until the valve opening becomes very narrow. With modern diagnostic tools, however, doctors can discover valves that can't open fully many years before people complain of discomfort.

People with mitral or aortic valves that don't fully close find that their heart becomes overactive during vigorous work or play, or during emotional excitement.

What are the symptoms of rheumatic heart disease?

Symptoms vary greatly. Often the damage to heart valves isn't immediately noticeable. Some people have no problem or feel only mild discomfort for years.

Eventually, damaged heart valves can cause serious, even disabling, problems. These problems depend on how bad the damage is and which heart valve is affected.

Valve abnormalities on the heart's left side (the **mitral** and **aortic** valves) usually cause symptoms earlier than abnormalities on the right side. The reason is that blood pressures are higher on the heart's left side. (The **tricuspid** valve is usually the only valve affected by rheumatic heart disease on the right side of the heart. The **pulmonic** valve is almost never affected.)

People with mitral or aortic valves that don't fully close find that their heart becomes overactive during vigorous work or play, or during emotional excitement. Increased physical or emotional activity puts more strain on a heart that's already overworked because of the leaking valve.

As a result, the left ventricle gradually gets bigger to make up for the added volume of blood. Eventually this becomes counterproductive, and the heart can't pump enough blood. Pressure builds in the ventricle and causes blood to back up into the lungs, causing shortness of breath. It can result in an inadequate blood supply to the body and fatigue. The body also may retain fluid.

The lungs of people with **mitral valve stenosis** are under more pressure. This puts an extra burden on the heart's right side, since it must pump against the raised pressure. The added pressure in the lungs also causes fluid retention **(pulmonary edema)** and shortness of breath.

In **aortic valve stenosis**, the extra pressure and larger size of the left ventricle means the heart muscle itself needs more blood. If the coronary arteries don't supply enough blood to the heart tissue, angina pectoris can occur. (See page 13.) Dizziness or fainting during exertion, shortness of breath, fatigue and palpitations are other symptoms of the same problem. The most advanced condition is congestive heart failure. (See page 61.)

How can rheumatic heart disease be prevented?

The best defense against rheumatic heart disease is to prevent rheumatic fever from ever occurring. By treating strep throat with penicillin or other antibiotics, doctors can usually stop acute rheumatic fever from developing.

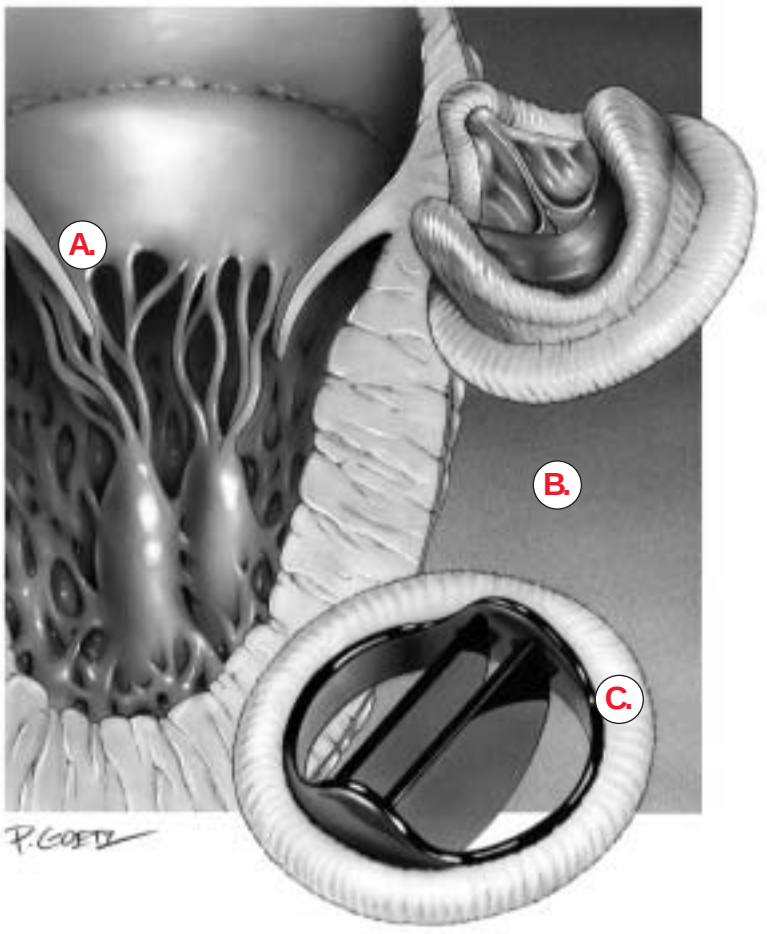
Often a doctor will wait for the result of a throat culture to be sure a strep infection is present and antibiotics are warranted. Most sore throats are caused by viral infections that aren't helped by antibiotic treatment. Strep throats are bacterial infections, so antibiotics help them.

People who've had rheumatic fever are at risk for more attacks and heart damage. That's why they're given continuous monthly or daily antibiotic treatment, maybe for life. If their heart has been damaged by rheumatic fever, they're also given a different antibiotic when they undergo dental or surgical procedures. This helps prevent bacterial endocarditis, a dangerous infection of the heart's lining or valves. (See page 62.)

What can be done about a bad heart valve?

When heart valves are damaged by rheumatic fever, they may not open or close properly. If they don't open properly, they block the forward flow of blood. If they don't close properly, blood may leak backwards.

When these problems occur, valve replacement surgery may be recommended. In this surgery the diseased valve is replaced with a metal or plastic valve, or with a specially prepared valve from another human heart (cadaver valve) or from an animal such as a pig (porcine valve). Most people who have valve replacement surgery improve markedly.



A. Normal mitral valve; B. Porcine replacement valve; C. Artificial replacement valve.

Congestive Heart Failure

What is congestive heart failure?

Congestive heart failure (or heart failure) is a condition in which the heart can't pump enough blood to the body's other organs. It occurs because the heart muscle is damaged or overworked. This can result from

- narrowed arteries that supply blood to the heart muscle — coronary artery disease.
- past heart attack (myocardial infarction) with scar tissue that interferes with the heart muscle's normal work.
- high blood pressure.
- heart valve disease due to past rheumatic fever or other causes.
- primary disease of the heart muscle itself, called cardiomyopathy.
- heart or blood vessel defects present at birth — congenital heart disease.
- infection of the heart valves (endocarditis) and/or heart muscle itself (myocarditis).

The "failing" heart keeps working but not as well as it should. People with heart failure can't exert themselves, because they become short of breath and tired.

As blood flow out of the heart slows, blood returning to the heart through the veins backs up, causing congestion in the tissues. Often swelling (edema) results. Most often the legs and ankles swell, but other parts of the body can swell, too. Sometimes fluid collects in the lungs and interferes with breathing, causing shortness of breath, especially when a person is lying down.

Heart failure also affects the kidneys' ability to dispose of sodium and water. The retained water increases the edema.

How is congestive heart failure diagnosed and treated?

The most common signs of congestive heart failure are swollen legs or ankles or difficulty breathing. Another symptom is weight gain when fluid builds up.

> Congestive heart failure usually requires a treatment program of rest, proper diet, modified daily activities and prescribed drugs.

Various drugs are used to treat congestive heart failure. They perform different functions. **ACE (angiotensin converting enzyme) inhibitors** and **vasodilators** expand blood vessels and decrease resistance. This lets blood flow more easily and makes the heart's work easier or more efficient. **Beta blockers** can improve how well the heart's left lower chamber (left ventricle) works. **Digitalis** strengthens the heart's pumping action. **Diuretics** help the body eliminate excess salt and water.

When a specific cause of congestive heart failure is discovered, it should be treated or, if possible, corrected. For example, some cases of congestive heart failure can be treated by treating high blood pressure. If the congestive heart failure is caused by an abnormal heart valve, the valve can be surgically replaced.

Most cases of mild and moderate congestive heart failure are treatable. Proper medical supervision can prevent such people from becoming invalids.

If the heart becomes so damaged that it can't be repaired, a more drastic approach should be considered. A heart transplant could be an option.

What about heart transplants?

A heart may be irreversibly damaged by long-lasting heart disease or viral infection. People with long-term heart failure, heart muscle disease, or other irreversible heart injury from coronary artery disease and multiple heart attacks that can't be treated by any other medical or surgical means may be candidates for heart transplants.

When the heart can no longer adequately work and a person is at risk of dying, a heart transplant may be indicated. It involves removing a diseased heart and replacing it with a healthy human heart. Cardiac transplantation is recognized as a proven procedure in appropriately selected patients.

When do infants and children need heart transplants?

Children with complex forms of congenital heart defects and children with dilated cardiomyopathy (see page 63) are most likely to be potential recipients.

Hypoplastic left heart syndrome is the most common, complex congenital heart defect for which a heart transplant is a potentially useful treatment. In this condition the heart's lower left pumping chamber (left ventricle) and the large artery that carries blood to the body (ascending aorta) are too small to support normal blood flow.

Other Heart and Blood Vessel Diseases

What is bacterial endocarditis?

Bacterial endocarditis is an infection of the heart's inner lining (endocardium) or the heart valves. This can damage or even destroy the heart valves. It occurs when bacteria in the bloodstream (bacteremia) lodge on abnormal heart valves or other damaged heart tissue.

Certain bacteria normally live on parts of your body, such as the mouth and upper respiratory system, the intestinal and urinary tracts, and the skin. Some surgical and dental procedures cause a brief bacteremia. Bacteremia is common after many invasive procedures, but only certain bacteria commonly cause endocarditis.

Endocarditis rarely occurs in people with normal hearts. People who have these preexisting heart conditions are at risk for developing endo-carditis when a bacteremia occurs:

- artificial (prosthetic) heart valves
- a history of previous endocarditis
- heart valves damaged (scarred) by rheumatic or other heart disease
- congenital heart or heart valve defects
- hypertrophic cardiomyopathy (See page 64.)

People who've had endocarditis before are at risk of getting it again, even when they don't have heart disease. Some congenital heart defects, including a ventricular septal defect, an atrial septal defect, or a patent ductus arteriosus, can be successfully repaired surgically. (See pages 54, 55 and 57.) There's no longer an increased risk for endocarditis after that.

How can bacterial endocarditis be prevented?

Not all cases of endocarditis can be prevented, because we don't always know when a bacteremia occurs. However, under certain circumstances, **the American Heart Association recommends antibiotics for patients whose heart condition puts them at risk for developing endocarditis. For example, antibiotics are prescribed before certain dental or surgical procedures.** These include procedures most likely to produce bacteremia with organisms that commonly cause endocarditis:

- professional teeth cleaning
- tonsillectomy or adenoidectomy
- examination of the respiratory passageways with a rigid bronchoscope
- certain types of surgery on the respiratory passageways, the gastrointestinal tract or the urinary tract
- gallbladder or prostate surgery

Antibiotics may be recommended for other types of procedures if the tissue is infected. This preventive step normally involves taking a dose of antibiotics an hour before the procedure.

The American Heart Association has issued an endocarditis wallet card for you to carry. You can get it from your doctor or on our Web site, **americanheart.org**. It's also important to maintain good oral health through regular brushing and flossing and regular visits to the dentist.

What is cardiomyopathy?

Cardiomyopathy is a serious disease in which the heart muscle becomes inflamed and doesn't work as well as it should. There may be multiple causes including viral infections.

Cardiomyopathy can be classified as primary or secondary. Primary cardiomyopathy can't be attributed to a specific cause, such as hypertension, heart valve disease, artery diseases or congenital heart defects. Secondary cardiomyopathy is due to specific causes and is often associated with diseases involving other organs as well as the heart. There are three principal types of cardiomyopathy — dilated, hypertrophic and restrictive.

What is dilated (congestive) cardiomyopathy?

This is the most common form. The heart cavity is enlarged and stretched (cardiac dilation). The heart is weak and doesn't pump normally, and most patients develop congestive heart failure. Arrhythmias and disturbances in the heart's electrical conduction also may occur.

Since blood flows more slowly through an enlarged heart, blood clots easily form. A blood clot is also called a **thrombus**. A clot that breaks free, circulates in the bloodstream and blocks a small blood vessel is called an **embolus**. It can cause life-threatening problems in many parts of the body.

- Clots that stick to the inner lining of the heart are called mural thrombi.
- If the clot breaks off in the right ventricle, it can be carried into the pulmonary circulation in the lung, forming pulmonary emboli.
- Blood clots formed in the heart's left side may be dislodged and carried into the body's circulation. They can form cerebral emboli in the brain, renal emboli in the kidney, peripheral emboli in the arms or legs or even coronary artery emboli in the heart.

A condition known as Barth syndrome, a rare and relatively unknown genetically linked heart disease, can cause dilated cardiomyopathy. This syndrome affects male children, usually during their first year of life, but it can be diagnosed later. In these young patients the heart condition is often associated with

changes in the skeletal muscles, short stature and an increased likelihood of bacterial infections. They also have a condition known as neutropenia, which is a reduced number of white blood cells (neutrophils). There are clinical signs of the cardiomyopathy in the newborn child or within the first months of life. These children also have metabolic and mitochondrial abnormalities.

How is dilated or congestive cardiomyopathy treated?

A person with cardiomyopathy may suffer an embolus before any other symptom of cardiomyopathy appears, and anti-clotting (anticoagulant) drug therapy may be needed. Arrhythmias may require antiarrhythmic drugs. More rarely, "heart block" may develop, requiring an artificial pacemaker. (See pages 25 and 29.) Therapy for dilated cardiomyopathy is sometimes disappointing. If the person is young and otherwise healthy, and if the disease gets worse and worse, a heart transplant may be considered.

When cardiomyopathy results in marked heart dilation, the leaflets of the mitral and tricuspid valves may not be able to close properly, resulting in murmurs. Blood pressure may rise because of increased sympathetic nerve activity. These nerves also can cause arteries to narrow. This mimics high blood pressure. That's why some people have high blood pressure readings. Because the level of blood pressure determines the heart's workload and oxygen needs, one treatment approach is to use vasodilators (drugs that "relax" the arteries). They lower blood pressure and thus the left ventricle's workload.

What is hypertrophic cardiomyopathy?

In this condition, the muscle mass of the left ventricle enlarges or "hypertrophies." In one form of the disease, the wall between the two ventricles (septum) becomes enlarged and obstructs the blood flow from the left ventricle. Besides obstructing blood flow, the thickened wall sometimes distorts one leaflet of the mitral valve, causing it to leak.

The syndrome is known as hypertrophic obstructive cardiomyopathy (HOCM) or asymmetric septal hypertrophy (ASH). It's also called idiopathic hypertrophic subaortic stenosis (IHSS). In the other form of the disease, non-obstructive hypertrophic cardiomyopathy, the enlarged muscle doesn't obstruct blood flow.

In over half the cases, the disease is hereditary. Close blood relatives (parents, children or siblings) of such persons often have enlarged septums, although they may have no symptoms.

The symptoms of hypertrophic cardiomyopathy include shortness of breath on exertion, dizziness, fainting and angina pectoris (chest pain or discomfort caused by reduced blood supply to the heart muscle). Some people have cardiac arrhythmias, abnormal heart rhythms that in some cases can lead to sudden death. The obstruction to blood flow from the left ventricle increases the ventricle's work, and a heart murmur may be heard.

How is hypertrophic cardiomyopathy treated?

A drug (a beta blocker or a calcium channel blocker) is the usual treatment. If a person has an arrhythmia, an antiarrhythmic drug may also be used. If the drug treatment fails, sometimes surgery is done on the obstructive form of hypertrophic cardiomyopathy.

Alcohol ablation is another nonsurgical treatment being developed for hypertrophic obstructive cardiomyopathy. It involves injecting alcohol down a small branch of one of the heart arteries to the extra heart muscle. This destroys the extra heart muscle without having to cut it out surgically.

People undergoing this procedure usually suffer chest pain during the alcohol injection. The alcohol also can disrupt normal heart

rhythms and require the insertion of a pacemaker. Alcohol ablation is a relatively new procedure being done at only a few specialized centers in the United States. However, it's too soon to know whether this treatment will result in long-term benefit. **It's still considered experimental.**

What is restrictive cardiomyopathy?

This is the least common type in the United States. The myocardium of the ventricles becomes excessively "rigid," so it's harder for the ventricles to fill with blood between heartbeats. A person with restrictive cardiomyopathy often complains of being tired, may have swollen hands and feet, and may have difficulty breathing on exertion. This type of cardiomyopathy is usually due to another disease process.

What is Kawasaki disease?

Kawasaki disease is a children's illness. It's also known as Kawasaki syndrome or mucocutaneous lymph node syndrome. It and acute rheumatic fever are the two leading causes of acquired heart disease in children in the United States.

About 80 percent of people with Kawasaki disease are under age 5. Children over age 8 are rarely affected. The disease occurs more often among boys (about 1.5 times as often as in girls) and among those of Asian ancestry. But it can occur in every racial and ethnic group.

The symptoms of Kawasaki disease include fever, rash, swollen hands and feet, redness of the whites of the eyes, swollen lymph glands in the neck, and irritation and inflammation of the mouth, lips and throat. Doctors don't know what causes Kawasaki disease, but it doesn't appear to be hereditary or contagious. Scientists who've studied the disease think the evidence strongly suggests it's caused by an infectious agent such as a virus. It's very rare for more than one child in a family to develop Kawasaki disease.

In as many as 20 percent of the children with Kawasaki disease, the heart is affected. The coronary arteries or the heart muscle itself can be damaged.

How does Kawasaki disease affect the heart?

The coronary arteries are most often affected. Part of a coronary wall can be weakened and balloon (bulge out) in an aneurysm. A blood clot can form in this weakened area and block the artery, sometimes leading to a heart attack. The aneurysm also can burst, but this rarely happens.

Other changes include inflammation of the heart muscle (myocarditis) or the sac surrounding the heart (pericarditis). Arrhythmias or abnormal working of some heart valves also can occur.

Usually all the heart problems go away in five or six weeks, and there's no lasting damage. But sometimes coronary artery damage persists.

An arrhythmia or damaged heart muscle can be detected using an electrocardiogram (ECG). An echocardiogram (or "echo") is used to look for possible damage to the heart or coronary arteries.

How is Kawasaki disease treated?

Even though the cause of Kawasaki disease is unknown, certain medicines do help. Aspirin is often used to reduce fever, rash, joint inflammation and pain, and to help prevent blood clots from forming. Another medicine, **intravenous gamma globulin**, can reduce the risk of developing coronary artery abnormalities when given early in the illness.

What is peripheral vascular disease?

Peripheral vascular disease (PVD) refers to diseases of blood vessels outside the heart and brain. It's often a narrowing of vessels that carry blood to leg and arm muscles. There are two types of these circulation disorders:

- **Functional** peripheral vascular diseases don't have an organic cause. They don't involve defects in blood vessels' structure. They're usually short-term effects and can come and go. Raynaud's disease (or Raynaud's phenomenon) is an example. It can be triggered by cold temperatures, emotional stress, work with vibrating machinery or smoking.
- **Organic** peripheral vascular diseases are caused by structural changes in the blood vessels, such as inflammation and tissue damage. Peripheral artery disease is an example. It's caused by fatty buildups in arteries (atherosclerosis).

What is peripheral artery disease?

Peripheral artery disease (PAD) is a condition similar to coronary artery disease and carotid artery disease. In PAD, fatty deposits build up along artery walls and affect blood circulation, mainly in arteries leading to the legs and feet. In its early stages, a common symptom is cramping or fatigue in the legs and buttocks during activity. Such cramping subsides when the person stands still. This is called "intermittent claudication." People with PAD have a higher risk of death from stroke and heart attack, due to the risk of blood clots.

How is peripheral artery disease diagnosed and treated?

The techniques used to diagnose PAD are similar to those used to diagnose carotid artery disease and cardiovascular disease. They include Doppler ultrasound, X-ray angiography and magnetic resonance imaging (MRI). (See pages 16–17 and 51–52.)

PAD is treated with drugs called "anti-thrombotics." These medicines prevent blood from clotting. Two types of anti-thrombotics are anti-platelet agents and anticoagulants.

A type of angioplasty can be used to dilate narrowed peripheral arteries. Often a stent is used to hold the artery open.

Peripheral vascular disease (PVD) refers to diseases of blood vessels outside the heart and brain.

A type of surgery for PAD involves bypassing the diseased part of the artery. A vein from another part of the body or a synthetic blood vessel is used. Another surgical procedure is atherectomy. In this case, the diseased vessel is cut open and the fatty deposit is removed. (See pages 20–21 for more information on these procedures.)

Glossary

Ablation — A group of therapeutic methods that physically destroy the cardiac tissue that causes or contributes to some types of tachycardia (fast heartbeat). May be done through surgery or using a transcatheter approach with an electrode catheter.

Abnormal Glucose Tolerance — The inability to metabolize (use) sugar adequately.

Adenosine — A substance produced in humans that helps protect against ischemia (lack of blood flow). It's produced in blood vessels, heart and skeletal muscle, and other organs.

Aneurysm — A ballooning-out of the wall of an artery, a vein or the heart due to weakening of the wall by disease, injury or an abnormality present at birth.

Angina Pectoris — Medical term for chest pain or discomfort due to coronary heart disease. A condition in which the heart muscle doesn't get enough blood, resulting in chest pain.

Angiocardiography — An X-ray examination of the blood vessels or chambers of the heart. A special fluid (contrast medium or dye) visible by X-ray is injected into the bloodstream. Tracing the course of this fluid produces X-ray pictures called angiograms.

Angiogenesis — The creation of blood vessels. The body creates small blood vessels called "collaterals" to help compensate for reduced blood flow.

Angiogram — An X-ray picture of blood vessels or chambers of the heart that shows the course of a special fluid (contrast medium or dye) injected into the bloodstream.

Angioplasty — A procedure sometimes used to dilate (widen) narrowed arteries. A catheter with a deflated balloon on its tip is passed into the narrowed artery segment, the balloon inflated, and the narrowed segment widened. Then the balloon is deflated and the catheter is removed.

Angiotensin — A chemical the body produces that causes the arterioles to constrict.

Angiotensin Converting Enzyme (ACE) Inhibitors — A class of drugs used to treat high blood pressure and heart failure. ACE inhibitors interfere with the body's production of angiotensin.

Angiotensin II Receptor Blockers — A class of drugs used to treat high blood pressure and heart failure. They block the effects of angiotensin.

Anticoagulant — A blood-thinning drug used to reduce the risk of clots.

Antihypertensive — A group of drugs used to treat high blood pressure.

Aorta — The large artery that receives blood from the heart's left ventricle and distributes it to the body.

Aortic Stenosis (AS) — A congenital heart defect in which the aortic valve, between the left ventricle and the aorta, is narrowed.

Aortic Valve — The heart valve between the left ventricle and the aorta. It has three flaps (cusps).

Aphasia — The inability to speak, write or understand spoken or written language because of brain injury or disease.

Arrhythmia (or Dysrhythmia) — An abnormal rhythm of the heart.

Arteriography — A testing procedure in which a dye visible to X-rays is injected into the bloodstream, then X-ray pictures are taken and studied to see if the arteries are damaged.

Arterioles — Small, muscular branches of arteries. When they contract, they increase resistance to blood flow, and blood pressure in the arteries increases.

Arteriosclerosis — Commonly called hardening of the arteries, this includes a variety of conditions that cause artery walls to thicken and lose elasticity. Some hardening of arteries often occurs when people grow older.

Artery — One of a series of vessels that carry blood from the heart to the various parts of the body. Arteries have thick, elastic walls that expand as blood flows through them.

Atherectomy — A procedure to remove plaque from arteries. An ultra-thin wire is threaded through a special catheter into the blocked artery. Several devices then may be used. One is a high-speed rotating "burr" that grinds the plaque into very tiny pieces. Another is a small rotating cutter that "shaves off" pieces of the blockage. Another is a laser catheter that vaporizes the plaque. (See Laser Angioplasty.)

Atherosclerosis — A form of arteriosclerosis in which the inner layers of artery walls become thick and irregular due to deposits of fat, cholesterol and other substances. This buildup is called "plaque." As the interior walls of arteries become lined with these deposits, the arteries become narrowed, reducing the blood flow through them.

Atria — The heart's two upper chambers.

Atrial Fibrillation — A disorder in which the heart's two small, upper chambers quiver instead of beating effectively. Blood that isn't pumped completely out of the atria when the heart beats may pool and clot. Then a blood clot may enter the bloodstream, become a cerebral embolus and cause an ischemic stroke.

Atrial Septal Defect (ASD) — A congenital heart defect in which an opening exists between the heart's two upper chambers.

Atrioventricular (AV) Canal Defect (or Atrioventricular Septal Defect) — A congenital heart defect in which a large hole in the center of the heart exists where the wall between the upper chambers joins the wall between the lower chambers. Also, a single large valve crosses the defect.

Atrioventricular (AV) Node — A small mass of specialized conducting tissue at the bottom of the right atrium. The electrical impulse stimulating the heart to contract must pass through this node to reach the ventricles.

Atrium — Either of the heart's two upper chambers in which blood collects before being passed to the ventricles.

Bacterial Endocarditis — See Endocarditis.

Balloon Angioplasty — See Angioplasty.

Barth Syndrome — A rare and relatively unknown genetically linked heart disease that can cause dilated cardiomyopathy.

Beta Blocker — A medicine to treat high blood pressure and some other heart conditions by reducing the heart rate and the heart's output of blood.

Bicuspid Aortic Valve — A congenital heart defect in which the aortic valve has only two flaps (cusps) instead of the normal three.

Blood Clot — A jelly-like mass of blood tissue formed by clotting factors in the blood. Clots stop the flow of blood from an injury. Blood clots also can form inside an artery whose walls are damaged by atherosclerotic buildup, or inside the atria if blood isn't pumped out completely and pools. Blood clots that block an artery can cause a heart attack or stroke.

Blood Pressure — The force or pressure exerted by the heart in pumping blood; the pressure of blood in the arteries.

"Blue Babies" — Babies who have a blue tinge to their skin (cyanosis) resulting from not enough oxygen in the arterial blood; this often indicates a heart defect.

Body Mass Index (BMI) — A formula to assess a person's body weight relative to height. It correlates highly with body fat in most people. Weight in kilograms is divided by height in meters squared (kg/m^2).

Bradycardia — Slowness of the heart rate (less than 60 beats per minute).

Calcium Antagonists (Calcium Channel Blockers) — A class of drugs used to treat high blood pressure and some other heart conditions. They can slow the heart rate and relax blood vessels.

Capillaries — Microscopically small blood vessels between arteries and veins that distribute oxygenated blood to the body's tissues.

Cardiac — Pertaining to the heart.

Cardiac Arrest — The stopping of the heartbeat, usually because of interference with the electrical signal (often associated with coronary heart disease).

Cardiac Catheterization — The process of examining the heart by introducing a thin tube (catheter) into a vein or artery and passing it into the heart.

Cardiac Computed Tomography (CT), Computerized Axial Tomographic Scan (CAT scan) — An X-ray imaging technique that provides cross-sectional images of the chest, including the heart and great vessels, or the brain. It's used to evaluate certain heart diseases and define the areas affected by stroke.

Cardiac Positron Emission Tomography (PET) — A test that lets physicians study and quantify how the heart tissue works. PET combines tomographic imaging with radionuclide tracers and tracer kinetic principles.

Cardiology — The study of the heart and its functions in health and disease.

Cardiomyopathy — A serious disease affecting the heart. It involves an inflammation and reduced function in heart muscle. There are multiple causes including viral infections.

Cardiopulmonary Resuscitation (CPR) — A combination of chest compression and mouth-to-mouth breathing. This technique is used during cardiac arrest to keep oxygenated blood flowing to the heart muscle and brain until advanced cardiac life support can be started or an adequate heartbeat resumes.

Cardiovascular — Pertaining to the heart and blood vessels. ("Cardio" means heart; "vascular" means blood vessels.) The circulatory system of the heart and blood vessels is the cardiovascular system.

Carotid Artery — One type of major artery in the neck carrying blood from the heart to the brain. The other type is vertebral artery.

Carotid Artery Disease (Carotid Artery Stenosis) — A carotid artery narrowed by a buildup of plaque. A type of atherosclerosis. Carotid artery disease is a major risk factor for ischemic stroke.

Carotid Bruit — An abnormal sound in the neck of a person with carotid artery disease, created by the blood as it flows through the diseased artery.

Carotid Phonoangiography — A test using a sensitive microphone placed on the neck, very close to the carotid artery. It records sounds and detects blockages, such as those caused by carotid artery disease.

Cerebral — Pertaining to the brain.

Cerebral Angioplasty — An experimental technique to treat some types of cerebrovascular problems. See Angioplasty.

Cerebral Embolism — A blood clot formed in one part of the body, then carried by the bloodstream to the brain, where it blocks an artery. A type of stroke.

Cerebral Hemorrhage — Bleeding within the brain, resulting from a ruptured aneurysm or a head injury. A type of stroke.

Cerebral Thrombosis — Formation of a blood clot in an artery that supplies part of the brain, blocking the flow of blood. A type of stroke.

Cerebrovascular — Pertaining to the brain and its major blood vessels.

Cerebrovascular Accident (CVA) — Also called apoplexy or stroke, this describes an impeded blood supply to some part of the brain, resulting in injury to brain tissue.

Cerebrovascular Occlusion — The obstruction or closing of a blood vessel leading to or within the brain, resulting in a stroke.

Cholesterol — A fat-like substance found in the blood and produced by the liver. Also found in animal tissue and present only in foods from animal sources such as whole-milk dairy products, meat, fish, poultry, animal fats and egg yolks.

Chylomicrons — Fatty particles in the blood containing mainly triglycerides, but also cholesterol, phospholipids and protein. They're made in the intestinal wall as foods are digested.

Cineangiography — The technique of taking moving pictures to show how a dye visible by X-ray passes through blood vessels.

Circulatory System — Pertaining to the heart, blood vessels and the blood's circulation.

Coarctation of the Aorta ("Coarct") — A congenital heart defect in which the aorta is pinched or constricted. This obstructs blood flow to the lower part of the body and increases blood pressure above the constriction.

Collateral Circulation — The process in which a system of small, normally closed arteries opens up and starts to carry blood to part of the heart when a coronary artery is blocked, or to part of the brain when a cerebral artery is blocked. They can serve as alternate routes of blood supply.

Congenital — Refers to conditions existing at birth.

Congenital Heart and Blood Vessel Defects (Congenital Cardiovascular Defects, Congenital Cardiovascular Disease) — Malformation of the heart or its major blood vessels present at birth.

Congestive Heart Failure — The inability of the heart to pump out all the blood that returns to it. This results in blood backing up in the veins that lead to the heart and sometimes in fluid accumulating in various parts of the body.

Coronary Arteries — Two arteries arising from the aorta that arch down over the top of the heart, branch and provide blood to the heart muscle.

Coronary Artery Disease (CAD) — Conditions that cause narrowing of the coronary arteries, reducing blood flow to the heart muscle. A type of atherosclerosis.

Coronary Bypass Surgery — Surgery that reroutes, or "bypasses," blood around clogged coronary arteries and improves the supply of blood and oxygen to the heart muscle. It's sometimes called CABG (for coronary artery bypass graft) or "cabbage."

Coronary Care Unit (CCU) — A specialized facility in a hospital or emergency mobile unit that's equipped with monitoring devices and staffed with trained personnel. It's designed specifically to treat heart patients.

Coronary Heart Disease (CHD) — Disease of the heart caused by atherosclerotic narrowing of the coronary arteries likely to produce angina pectoris or heart attack.

Coronary Occlusion — An obstruction of a coronary artery that hinders blood flow to some part of the heart muscle. A cause of heart attack.

Coronary Thrombosis — Formation of a clot in one of the arteries that conduct blood to the heart muscle. Also called coronary occlusion.

Creatine Kinase (CK) — A blood enzyme. Tests for CK and the fraction CK-MB are used to confirm the existence of heart muscle damage.

Cyanosis — Blueness of skin caused by insufficient oxygen in the blood.

Defibrillator — An electronic device that helps reestablish normal contraction rhythms in a malfunctioning heart.

Diabetes — The inability of the body to produce or respond properly to insulin. The body needs insulin to convert sugar and starch into the energy needed in daily life. The full name for this condition is diabetes mellitus; defined as a fasting blood glucose of 126 mg/dL or more measured on two occasions.

Diastolic Blood Pressure — The lowest blood pressure measured in the arteries, it occurs when the heart muscle relaxes between beats. In a typical blood pressure reading, such as 120/78, the lower number is diastolic blood pressure.

Digital Cardiac Angiography (DCA), Digital Subtraction Angiography (DSA) — A modified form of computer imaging that records pictures of the major blood vessels to the heart or brain. It shows blockages, how severe they are and what can be done about them.

Digitalis (also Digoxin, Digitoxin) — A drug that strengthens and slows the heart's pumping action, and helps eliminate fluid from body tissues when heart failure is present. It's also used to treat certain heart rhythm abnormalities.

Diuretic — A drug that increases the rate at which urine forms by promoting the excretion of water and salts. It's used to treat high blood pressure, congestive heart failure and some congenital heart defects.

Doppler Ultrasound — A test that uses high-frequency sound waves to detect blockages in an artery.

Dysarthria — Speech that is slowed, slurred or distorted due to muscular problems caused by damage to the brain or nervous system.

Dysphagia — Difficulty chewing and swallowing food that occurs when one side of the mouth is weak due to stroke or other brain injury. One or both sides of the mouth can lack feeling, increasing the risk of choking.

Dysrhythmia — See Arrhythmia.

Ebstein's Anomaly — A congenital downward displacement of the tricuspid valve (located between the heart's upper and lower chambers on the right side) into the heart's right bottom chamber (or right ventricle). It's usually associated with an atrial septal defect.

Echocardiography — A diagnostic method in which pulses of sound are transmitted into the body. The echoes returning from the surfaces of the heart and other structures are electronically plotted and recorded to produce a "picture" of the heart's size, anatomic shape and movements.

Edema — Swelling due to an abnormally large amount of fluid in body tissues.

Eisenmenger's Complex — A congenital heart defect. A ventricular septal defect is coupled with pulmonary high blood pressure, the passage of blood from the right side of the heart to the left (right to left shunt), an enlarged right ventricle and a latent or clearly visible bluish discoloration of the skin (cyanosis).

Electrocardiogram (ECG or EKG) — A graphic record of electrical impulses produced by the heart.

Electroencephalogram (EEG) — A graphic record of the electrical impulses produced by the brain.

Electron-Beam Computed Tomography (EBCT or Ultrafast® CT) — The high-speed form of X-ray imaging technology. It's used to evaluate various structures and functions in the heart and to measure calcium deposits in the coronary arteries.

Electrophysiologic Testing — A procedure used to provoke known but infrequent arrhythmias and to unmask suspected arrhythmias. Using local anesthesia, temporary electrode catheters are positioned in the heart's atria, ventricles or both, and at strategic locations along the conduction system. They record cardiac electrical signals and "map" the spread of electrical impulses during each heartbeat.

Embolus — A blood clot or other particle that forms in one part of the body, then is carried to another part of the body.

Emotional Lability — An effect of stroke in which a survivor cries or laughs for no apparent reason.

Endarterectomy — Surgical removal of plaque deposits or blood clots in an artery.

Endocarditis — An infection of the heart lining or valves, usually caused by bacteria. People with prosthetic heart valves, abnormal heart valves, a history of endocarditis or congenital heart defects are at increased risk of this disease.

Endothelium — The smooth inner lining of some body structures, including the heart (endocardium) and blood vessels.

Enzyme — A complex chemical that can speed up specific biochemical processes in the body.

Estrogen — A hormone produced in a woman's body. Synthetic forms of estrogen are used in oral contraceptives and hormone replacement therapy.

Exercise Stress Test (or Treadmill Test) — A diagnostic test in which a person walks on a treadmill or pedals a stationary bicycle while hooked up to equipment to monitor the heart. The test monitors heart rate, breathing, blood pressure, electrical activity (on an electrocardiogram) and the person's level of tiredness. It shows if the heart's blood supply is sufficient and if the heart rhythm is normal.

Fibrillation — Fast, uncoordinated contractions of individual heart muscle fibers. The heart chamber involved can't contract all at once and pumps blood ineffectively, if at all.

HDL Cholesterol — Often called "good" cholesterol because a high level of it seems to protect against heart attack. People with a low HDL cholesterol level (less than 40 mg/dL) have a higher heart disease risk. A low level of HDL cholesterol also may raise stroke risk. (See High-Density Lipoprotein.)

Heart Attack — Death of or damage to part of the heart muscle due to an insufficient blood supply. The medical term for heart attack is myocardial infarction. It's also sometimes called a coronary thrombosis or coronary occlusion.

Heart Block (or AV Block) — An impaired electrical signal from the heart's upper to lower chambers (through the AV or sinus node).

Heart-Lung Machine — An apparatus that oxygenates and pumps blood to the body while a person's heart is opened for surgery.

Heart Murmur — An abnormal sound in the heart caused by defective heart valves or holes in the heart walls. Can also be caused by pregnancy, fever and other conditions.

Hemorrhage — Severe bleeding leading to excessive blood loss.

Hemorrhagic Stroke — The injury to brain cells caused by ruptured blood vessels in the brain. Cerebral hemorrhages bleed inside the brain; subarachnoid hemorrhages bleed into the space between the brain and the skull.

Heredity — The passing of a genetic quality or trait from parent to offspring.

High Blood Pressure — A chronic increase in blood pressure above its normal range (systolic pressure of 140 mm Hg or higher and/or diastolic pressure of 90 mm Hg or higher).

High-Density Lipoprotein (HDL) — A type of protein believed to transport cholesterol away from the tissues and to the liver, where it can be removed from the bloodstream. (See HDL Cholesterol.)

Homocysteine — An amino acid. Too much homocysteine in the blood is related to a higher risk of coronary heart disease, stroke and peripheral vascular disease.

Hypercholesterolemia — High levels of blood cholesterol, a major risk factor for coronary heart disease, heart attack and stroke.

Hypertension — Same as High Blood Pressure.

Hypertriglyceridemia — High levels of triglycerides in the blood. A high triglyceride level combined with low HDL cholesterol or high LDL cholesterol seems to speed up atherosclerosis (fatty buildups of plaque in the arteries).

Hypoplastic Heart — A rare congenital defect in which the right or left side of the heart is incompletely formed.

Hypotension — Low blood pressure.

Implantable Cardioverter Defibrillator — A device used in patients at risk for recurrent, sustained ventricular tachycardia or fibrillation. Leads positioned inside the heart or on its surface are used to deliver electrical shocks, sense the cardiac rhythm and pace the heart, as needed. The leads are tunneled to a pulse generator implanted in a pouch beneath the skin of the chest or abdomen.

Incidence — The number of new cases of a disease that develop in a population during a one-year period. For some statistics, new and recurrent attacks or cases are combined.

Inferior Vena Cava — A major vein that carries blood from the lower body (legs and abdomen) to the heart.

Intermittent Claudication — Cramping or fatigue in the legs and buttocks during activity that subsides when a person stands still. It's a common, early symptom of peripheral artery disease.

Ischemia — Reduced blood flow to an organ, usually due to a constricted or blocked artery.

Ischemic Heart Disease (Coronary Artery Disease, Coronary Heart Disease) — This term is applied to heart ailments caused by narrowed coronary arteries and characterized by a reduced blood supply to the heart.

Ischemic Stroke — The death of or injury to brain cells caused when a blood clot or other particle blocks a cerebral artery. Cerebral thrombosis and cerebral embolism are ischemic strokes.

Kawasaki Disease — An acute children's illness of unknown cause that's characterized by fever, rash, swelling and inflammation of various parts of the body. The coronary arteries or other parts of the heart are affected in up to 20 percent of children with this disease.

Kawasaki Syndrome — See Kawasaki Disease.

LDL Cholesterol — Often called "bad" cholesterol. A high level of LDL cholesterol (160 mg/dL and above) reflects an increased risk of heart disease and stroke. (See Low-Density Lipoprotein.)

Laser Angioplasty — A technique used to open coronary arteries blocked by plaque. A catheter with a laser at its tip is inserted into an artery. Then it's advanced through the artery to the blockage. When the laser is in position, it emits pulsating beams of light that vaporize the plaque.

Lipid — A fatty substance insoluble in blood.

Lipoprotein — The combination of lipid (fat) surrounded by a protein; the protein makes the fat soluble in blood.

Low-Density Lipoprotein (LDL) — A type of protein that transports "harmful" cholesterol in the blood. It's the major cholesterol carrier in the blood. (See LDL Cholesterol.)

Lp(a) Cholesterol — A genetic variation of LDL cholesterol. A high level of Lp(a) is an important risk factor for developing atherosclerosis prematurely.

Lumen — The open space within a tube, such as a blood vessel.

Magnetic Resonance Imaging (MRI), Nuclear Magnetic Resonance (NMR) Imaging — An imaging procedure that uses powerful magnets to look inside the body. Computer-generated pictures can image the heart muscle and evaluate various heart problems. It can outline the affected part of the brain and help define problems created by stroke.

Mitral Valve — The heart valve between the left atrium and left ventricle. It has two flaps (cusps).

Monounsaturated Fats — A type of fat found in many oils but mostly in canola, olive and peanut oil, nuts and avocados.

Mortality — The total number of deaths from a given disease in a population during an interval of time, usually a year.

Mucocutaneous Lymph Node Syndrome — See Kawasaki Disease.

Myocardial Infarction — The damaging or death of an area of the heart muscle (myocardium) resulting from a blocked blood supply to that area; medical term for a heart attack.

Myocardial Ischemia — Deficient blood flow to part of the heart muscle.

Myocarditis — Inflammation of the heart muscle (myocardium).

Myocardium — The muscular wall of the heart. It contracts to pump blood out of the heart and then relaxes as the heart refills with returning blood.

Nitroglycerin — A drug that causes blood vessels to dilate and is often used to treat angina pectoris (chest pain or discomfort).

Obesity — The condition of being significantly overweight. It's defined as a body mass index (BMI) of 30.0 or greater, or about 30 pounds or more over ideal body weight. Extreme obesity is defined as a BMI of 40.0 or more.

Occluded Artery — An artery in which blood flow has been impaired by a blockage.

Open-Heart Surgery — Surgery performed on the opened heart while the bloodstream is diverted through a heart-lung machine.

Overweight — Defined as a body mass index (BMI) of 25.0–29.9 kg/m^2. A BMI of 25 corresponds to about 10 percent over ideal body weight.

Pacemaker — The "natural" pacemaker of the heart is called the sinus node. It's a small group of specialized cells in the top of the heart's right atrium. It produces the electrical impulses that travel down to the ventricular muscle, causing the heart to contract. An "artificial pacemaker" is an electrical device that can substitute for a defective natural pacemaker or conduction pathway. The artificial pacemaker controls the heart's beating by emitting a series of rhythmic electrical discharges.

Paramedical — Pertaining or closely related to the art or practice of medicine. This term is often applied to personnel whose work supports, or is closely related to, that of practicing physicians.

Patent Ductus Arteriosus (PDA) — A congenital heart defect in which an open passageway allows blood to mix between the pulmonary artery and the aorta.

Percutaneous Transluminal Coronary Angioplasty (PTCA) — See Angioplasty.

Perfusion — Blood flow.

Pericarditis — Inflammation of the outer membrane surrounding the heart.

Pericardium — The outer fibrous "sac" that surrounds the heart.

Peripheral Artery Disease — A type of peripheral vascular disease that affects blood circulation, primarily in arteries leading to the legs and feet. It's caused by atherosclerosis.

Peripheral Vascular Disease — Diseases of blood vessels outside the heart and brain or diseases of the lymph vessels. Often a narrowing of vessels carrying blood to leg and arm muscles.

Plaque — Also called atheroma, this is a deposit of fatty (and other) substances in the inner lining of the artery wall characteristic of atherosclerosis.

Plasma Lipid — The lipid (fatty particles) carried in blood.

Platelets — An element in blood that aids in blood clotting.

Phospholipid — A type of fat (lipid) that contains phosphorous. It splits into fatty acids, glycerin and a nitrogen compound by the addition of water.

Polyunsaturated Fats — A type of fat found mainly in vegetable oils such as corn, safflower, sunflower and soybean oils. They're usually liquid at room temperature.

Prevalence — The total number of cases of a given disease in a population at a specific time. Prevalence is sometimes expressed as a percentage of population.

Prinzmetal's Angina — See Variant Angina Pectoris.

Progestin — A synthetic form of the female hormone progesterone, used in oral contraceptives and hormone replacement therapy.

Prophylaxis — Preventive treatment.

Prostaglandins — A group of substances formed in the blood, one of which may damage arteries.

Pulmonary — Pertaining to the lungs.

Pulmonary Atresia — A congenital heart defect in which no pulmonary valve exists. Blood can't flow from the right ventricle into the pulmonary artery and on to the lungs. This results in a blue discoloration of the skin (cyanosis).

Pulmonary Stenosis (PS) — A congenital heart defect in which the pulmonary or pulmonic valve is defective and doesn't open properly.

Pulmonic (Pulmonary) Valve — The heart valve between the right ventricle and the pulmonary artery. It has three flaps (cusps).

Radionuclide Imaging (Radionuclide Angiography) — Tests using substances called radionuclides injected into the bloodstream. Computer-generated pictures show the heart working and any damage from a heart attack. As a nuclear brain scan, this test can detect areas where the brain is damaged.

Reentry — A type of abnormal conduction in which electrical impulses get caught in a merry-go-round-like sequence. This is a common cause of tachycardias.

Regurgitation — The leakage that results when a heart valve that doesn't close properly lets blood leak back into the chamber from which it was pumped.

Reperfusion Therapy — One or more techniques to restore blood flow to part of the heart muscle damaged during a heart attack, or part of the brain injured during a stroke. It may include clot-dissolving drugs (thrombolysis), balloon angioplasty or surgery.

Restenosis — The reclosure of an opening, such as a blood vessel or heart valve, after an angioplasty, stent or other procedure has been performed to open it.

Rheumatic Heart Disease — Damage done to the heart, particularly the heart valves, by one or more attacks of rheumatic fever.

Risk Factor — An element or condition involving certain hazard or danger. When referring to the heart and blood vessels, a *positive* risk factor is associated with an *increased* chance of developing cardiovascular disease including stroke. A *negative* risk factor is associated with a *reduced* chance of developing heart and blood vessel disease.

Rubella — Commonly known as German measles.

Saturated Fats — Types of fat found in all foods from animals and from some plants, like coconut, palm and palm kernel oils. They're typically solid at room temperature.

Septum — The muscular wall dividing the chambers on the heart's left side from the chambers on the right.

Sickle Cell Anemia — A genetic blood disorder that mainly affects African Americans. "Sickled" red blood cells are less able to carry oxygen to the body's tissues and organs. They also tend to stick to blood vessel walls. This can block arteries to the brain and cause a stroke.

Silent Ischemia — Episodes of ischemia that aren't accompanied by pain.

Single Photon Emission Computed Tomography (SPECT) — A nuclear imaging technique that involves injecting a radioactive liquid into the blood, then taking a series of pictures around the chest.

Sinoatrial (SA) or Sinus Node — See Pacemaker.

Sodium — A mineral essential to life found in nearly all plant and animal tissue. Table salt (sodium chloride) is nearly half sodium.

Spasm — The sudden, temporary or prolonged contraction of a muscle or artery.

Sphygmomanometer — An instrument for measuring blood pressure.

Stable Angina — Predictable chest discomfort that usually occurs on exertion (such as running to catch a bus) or under mental or emotional stress. Normally the chest discomfort is relieved with rest or nitroglycerin or both.

Stenosis — The narrowing or constriction of an opening, such as a blood vessel or heart valve.

Stent Procedure — Using a wire mesh tube (a stent) to prop open an artery that's recently been cleared using angioplasty.

Stethoscope — An instrument for listening to sounds within the body.

"Strep" Infection (Streptococcal Infection) — An infection, usually in the throat, resulting from streptococcus bacteria.

Stress — Bodily or mental tension resulting from a person's response to physical, chemical or emotional factors. Stress can refer to physical exertion as well as mental anxiety.

Stroke (also called Apoplexy, Brain Attack or Cerebrovascular Accident) — Loss of muscle function, mental function, vision, sensation or speech resulting from brain cell injury caused by an insufficient supply of blood to part of the brain.

Subaortic Stenosis — A congenital heart defect. The left ventricle is narrowed just below the aortic valve, which blood passes through to go into the aorta.

Subarachnoid Hemorrhage — Bleeding from a blood vessel on the surface of the brain into the space between the brain and the skull. A type of stroke.

Sudden Cardiac Death, Sudden Death — Death resulting from the abrupt loss of heart function (cardiac arrest). The victim may or may not have diagnosed heart disease. The death occurs within minutes after symptoms appear. The most common underlying reason that patients die suddenly from cardiac arrest is coronary heart disease.

Superior Vena Cava — A major vein that carries blood from the upper body (head, neck, chest and arms) to the heart.

Supraventricular Tachycardia — A condition in which heart tissue in either the upper chambers (atria) or the middle region (above the ventricles) develops pacemaker activity, resulting in an abnormally fast heartbeat.

Sympathetic Nerve Inhibitors — A class of antihypertensive drugs that reduce blood pressure by inhibiting the sympathetic nerves from constricting blood vessels.

Systolic Blood Pressure — The highest blood pressure measured in the arteries. It occurs when the heart contracts with each heartbeat. In a typical blood pressure reading, such as 120/78, the upper number is systolic blood pressure.

Tachycardia — An abnormally fast heartbeat (more than 100 beats per minute).

Tetralogy of Fallot — A complex, congenital heart defect with four components: ventricular septal defect, pulmonary valve stenosis, muscular right ventricle and the aorta directly over the ventricular septal defect. Blood pumped to the body contains less-than-normal amounts of oxygen. This results in cyanosis, a blue discoloration of the skin.

Thrombolysis — The breaking up of a blood clot.

Thrombosis — The formation or presence of a blood clot (thrombus) inside a blood vessel or chamber of the heart.

Thrombus — A blood clot that forms inside a blood vessel or chamber of the heart.

Tissue Plasminogen Activator (tPA) — One of several clot-dissolving drugs used during a heart attack or stroke to restore blood flow in a blocked artery.

Total Anomalous Pulmonary Venous (P-V) Connection — A congenital heart defect. The pulmonary veins that bring oxygen-rich (red) blood from the lungs back to the heart aren't connected to the left atrium. Instead, the pulmonary veins drain through abnormal connections to the right atrium. The blood passing through the aorta to the body doesn't have enough oxygen, which causes the child to look blue (cyanotic).

***Trans* Fats** — A type of fat that results from adding hydrogen to vegetable oils used in commercial baked goods and for cooking in most restaurants and fast-food chains.

Transient Ischemic Attack (TIA) — A temporary stroke-like event that lasts for only a short time and is caused by a temporarily blocked blood vessel leading to or within the brain. Also called a "little stroke" or "mini-stroke," it's an extremely important stroke indicator.

Transposition of the Great Arteries — A congenital heart defect in which the positions of the pulmonary artery and the aorta are reversed. Blood pumped to the body contains less-than-normal amounts of oxygen. This results in cyanosis, a blue discoloration of the skin.

Tricuspid Atresia — A congenital heart defect in which there's no tricuspid valve. That means no blood can flow from the right atrium to the right ventricle. This results in a blue discoloration of the skin (cyanosis).

Tricuspid Valve — The heart valve between the right atrium and the right ventricle. It has three flaps (cusps).

Triglyceride — The most common type of fat in the body. The body gets triglyceride directly from some foods (fatty acids) and makes it in the liver from other energy sources (carbohydrates, alcohol and some cholesterol).

Troponins — Proteins found in heart muscle. Blood tests for troponins can detect heart muscle injury.

Truncus Arteriosus — A complex congenital heart defect where only one artery arises from the heart and forms the aorta and pulmonary artery.

Ultrafast® CT — See Electron-Beam Computed Tomography.

Ultrasound — High-frequency sound vibrations, not audible to the human ear, used in medical diagnosis.

Unstable Angina — Chest pain or discomfort that's unexpected and usually occurs while at rest. The discomfort may be more severe and prolonged than typical angina or be the first time a person has angina.

Variant Angina Pectoris (or Prinzmetal's Angina) — Attacks of chest pain due to coronary artery spasm that occur almost exclusively when a person is at rest.

Vascular — Pertaining to blood vessels.

Vasodilators — A group of drugs that cause the muscle in the walls of the blood vessels (especially the arterioles) to relax, allowing the vessels to dilate.

Vein — One of a series of vessels that carries blood from various parts of the body back to the heart.

Ventricle — One of the two lower chambers of the heart.

Ventricular Fibrillation — A condition in which the ventricles contract in an extremely fast, chaotic fashion so no blood is pumped from the heart.

Ventricular Septal Defect (VSD) — A congenital heart defect in which an opening exists between the heart's two lower chambers.

Ventricular Tachycardia — A condition in which an area of the ventricle muscle develops pacemaker activity, resulting in a very fast, abnormal heartbeat.

Venules — Small veins, the blood vessels that carry blood back to the heart and lungs.

Vertebral Artery — One type of major blood vessel in the neck carrying blood from the heart to the brain. The other type is carotid artery.

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