IN THIS CHAPTER

- Understanding the causes of atherosclerosis and coronary heart disease
- Determining the causes and effects of angina
- Exploring what causes heart attacks
- Learning about arrhythmias, heart failure, and other forms of heart disease

Chapter 1
Understanding the Onset and Outcomes of Heart Disease

Your heart works harder than any other muscle in your body. Depending on your age and physical condition, a normal heart beats 60 to 90 times per minute when you're sitting and up to 150 to 200+ times per minute when you're maxing out aerobic physical activity. A healthy heart is equipped to sustain at this pace for 70 to 90 years and beyond. The key word here is *healthy.*

From the moment you're born (and even before), multiple factors related to your biology, behavior, and environment have an effect, for good or ill, on your heart and cardiovascular system. Heart disease is progressive: It starts stealthily in the coronary (and other) arteries and progresses silently for years before any detectable signs of disease emerge. Research over the last 25 years provided new insights into how heart disease begins, starting at the cellular and molecular levels. These new insights are helping to prevent heart disease, halt it, or even reverse its progress.

This chapter presents a brief overview of the heart and cardiovascular system, followed by a discussion of the silent precursors and early stages of heart disease. Then you look at *angina* and *unstable angina*, two types of chest pain that are often the first signs of heart disease for many people. Finally, you see how disease progression may result in heart attacks, arrhythmia, heart failure, and other acute problems.

Touring the Heart and Cardiovascular System

Understanding how your heart and cardiovascular system work provides a foundation for understanding heart disease and its many manifestations. These basics can help you do a better job of keeping your heart healthy.

The heart's anatomy and function

The heart is located in the center of the chest cavity, just to the left of the midline of the body. Figure 1-1 illustrates the exterior of a healthy heart and Figure 1-2 illustrates the interior. You need to understand the following important parts:

- **The heart muscle:** Called the *myocardium* (*myo* = muscle and *cardium* = heart; pronounced my-o-*car*-dee-um), this muscle contracts and relaxes to pump blood throughout the cardiovascular system.
- **The coronary arteries:** Three large coronary arteries and their many branches deliver a continuous supply of oxygenated blood to the heart. Narrowing of these arteries causes chest pain; blockage causes heart attack.
- **The pumping chambers:** The heart's job is to pump blood to the lungs to get oxygen and to pump the oxygenated blood to the rest of the body. To fulfill these tasks, the heart has a left and a right side (shown in Figure 1-2), each with one main pumping chamber called a *ventricle* located in the lower part of it. Sitting above the left and right ventricles are two small booster pumps called *atria* (or *atrium,* when you're talking about just one).

  The right ventricle pumps deoxygenated blood from the body to the lungs to receive a new supply of oxygen and back to the heart, through the left atrium to the left ventricle. The left ventricle pumps oxygenated blood through the arterial system to the rest of the body where it feeds every single living cell. Various disease conditions can damage each of these structures.

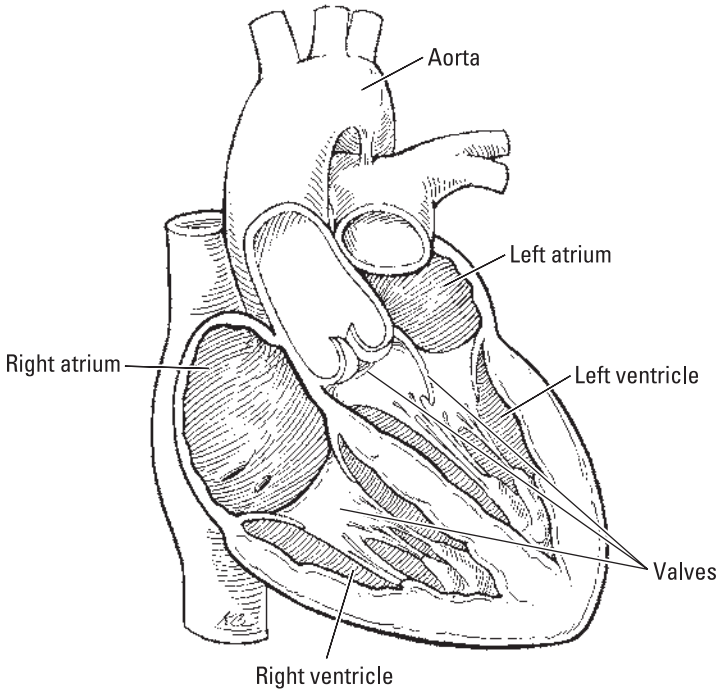

FIGURE 1-1: A typical healthy heart.

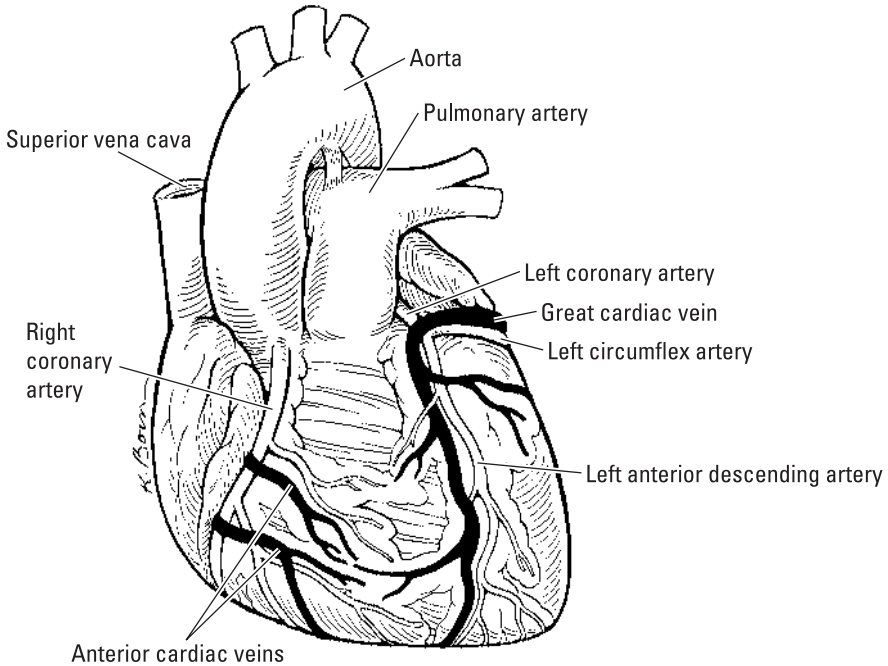

FIGURE 1-2: The interior of a normal heart.

Illustration by Kathryn Born

- **The valves:** Four valves regulate the flow of blood in and out of the heart and from chamber to chamber. They act a bit like cardiac traffic cops by directing the way blood flows, how much of it flows, and when it stops from flowing. Disease and injury can cause heart valves to leak, narrow, or otherwise malfunction, disrupting the heart's ability to pump blood efficiently.
- **The electrical system:** This electrical system is controlled by a group of specialized cells that spontaneously discharge, sending electrical currents down specialized nerves and tissues, causing the heart to contract. When any of these electrical structures becomes diseased or disordered, *arrhythmias* (ay-*rith*-mee-uhz), or heart rhythm disturbances, occur.
- **The pericardium:** The entire heart is positioned in a thin sac called the *pericardium* (*peri* = around and *cardium* = heart; pronounced per-ry-*car*-dee-um). Fluid within the sac lubricates the constantly moving surfaces. Inflammation of the pericardium from an infection or other cause causes *pericarditis*. Buildup of excess fluid inside the pericardium can cause problems with how the heart functions, a condition called *cardiac tamponade*.

Connecting every cell in your body: The cardiovascular system

A pump is useless without the rest of the plumbing, which in your body is called the *cardiovascular system.* Here's a quick look at how it all fits together and functions.

- **The lungs:** The lungs are composed of an intricate series of air sacs surrounded by a complex, highly branching network of blood vessels. Their sole purpose is to receive the deoxygenated blood from the heart, fill the red corpuscles full of fresh oxygen, and send them back to the heart for delivery to the body. The red blood cells give off waste products such as carbon dioxide at the same time they take on oxygen; the lungs then expel the carbon dioxide. This low-pressure system facilitates the rapid flow and reoxygenation of enormous amounts of blood.
- **The arteries:** As oxygenated blood returns to the left side of the heart, it is pumped out to the body through the *aorta,* the main artery of the body, and into the rest of the arterial system to feed the entire body with oxygenated blood. Although the heart exerts enough force to push oxygenated blood throughout the body, the arteries also have muscular walls that help push the blood along. The force exerted against resistance of the artery walls creates a high-pressure system that is very *elastic* to allow the arteries to expand or contract to meet the needs of various organs and muscles. Your blood pressure reading results from measuring the pressure in these arteries when contracting and at rest.

- **The capillaries:** The arterial system divides and redivides into a system of ever smaller branches to distribute nourishing blood to each individual cell, ultimately ending up in a network of microscopic vessels called *capillaries,* which deliver oxygenated blood to the working cells of every organ and muscle in the body.
- **The veins:** After oxygen leaves the capillary system, the deoxygenated blood and waste products from the cells are carried back through the body in the *veins.* The veins ultimately come together in two very large veins, called the *inferior vena cava* (*vee*-nuh *cay*-vuh) and the *superior vena cava.* The inferior vena cava drains blood from the lower part of the body and superior vena cava drains blood from the upper part of the body. These veins discharge blood into the right atrium of the heart to be pumped into the right ventricle and out to the lungs again to start the whole process over again.
- **The blood:** Although blood is not considered part of the cardiovascular system, circulating blood to every cell of the body is the reason the cardiovascular system exists. This red fluid transports oxygen and fuel to the cells and removes waste products. It's also the delivery vehicle for many specialized cells and biochemicals, including those that contribute to the development of heart disease.

Keeping the beat: How the nervous system controls heart rate

In addition to its internal electrical system, the heart has profound linkages to the nervous system that provide additional control of the heart rate. Two main branches of the involuntary nervous system interact with the heart — the sympathetic nervous system and the parasympathetic nervous system. In simple terms, the *sympathetic nervous system* helps the heart speed up, and the *parasympathetic nervous system* helps the heart slow down. They act through direct nerve links to the heart and through the release of chemical substances that reach the heart through the bloodstream.

Understanding How Heart Disease Begins and Develops

The human cardiovascular system is wondrously complex. If every element is in balance and working as it should, a state called *homeostasis,* the entire system, including the heart and blood vessels, would remain healthy. Unfortunately,

multiple factors related to your biology and lifestyle can tip the system out of balance and trigger the development of heart disease. The earliest changes typically start in childhood or adolescence and silently progress for years before producing changes that can be seen in diagnostic tests or symptoms that you experience. The most common type of cardiovascular disease is *atherosclerosis*.

Defining atherosclerosis — the most common form of cardiovascular disease

Atherosclerosis results from the gradual buildup of fatty deposits called *plaque*, or *lesions*, in the interior walls of large and medium-sized arteries. The disease process starts with small changes in the artery wall and takes years to develop to a point where the narrowing arteries may produce symptoms or negatively affect your health.

Narrowing in the heart's arteries leads to *coronary heart disease (CHD)*, also called *coronary artery disease (CAD)*. CHD gradually starves the heart muscle of the high level of oxygenated blood that it needs to function properly. A lack of adequate blood supply to the heart typically produces symptoms that range from angina and unstable angina (see "Defining unstable angina," later in this chapter) to heart attack or sudden death. Narrowing of the carotid arteries that carry blood to the brain increases your risk of stroke. Narrowed arteries in your legs or arms results in *peripheral artery disease (PAD)*.

The term a*therosclerosis* comes from two Greek words — *athero* (paste, gruel) and *sclerosis* (hardness) — that may give you a graphic image of hardened sludge. Not a pretty picture, is it? But it's an apt image for these deposits of cholesterol, other fats, cellular wastes, platelets, calcium, and other substances. These deposits typically start with fatty streaks and grow to large bumps that distort the artery and block its interior where the blood must flow. Some plaques are stable and others are unstable or vulnerable to cracking or rupturing, which often leads to an artery-blocking blood clot and subsequent heart attack.

REMEMBER

During the last 15 to 20 years, evidence from extensive population studies and clinical research has increased doctors' understanding of the many factors and pathways that contribute to the beginnings and progress of atherosclerosis. The next two sections provide an overview of medical science's best understanding right now; however, you need to remember that new studies continually add to the knowledge of this complex, multifaceted disease.

Triggering the precursors of atherosclerosis

Biological factors that contribute to the development of cardiovascular disease are present from birth and perform vital functions that enable the human body to grow and resist infection. As a consequence, all human beings are born with the potential to develop heart disease. The early precursors of atherosclerosis frequently occur in children, teens, and young adults. Fortunately, adopting a heart-healthy lifestyle can usually reverse these early manifestations. The sooner you start, the better, but it's never too late.

Current biomedical evidence has led to a consensus that atherosclerosis is a multifactorial chronic inflammatory disease that starts with the dysfunction of or injury (or both) to the endothelium, which is the inner lining of artery walls. Although only a single-cell-deep layer, the endothelium regulates the normal functioning of the arterial vessel walls. It acts as the traffic cop responding to the many blood-borne influences and biochemical signals that can modify the arterial walls. When any factor stresses or injures the endothelium, it triggers the inflammatory response that activates a variety of immune system signals and cells that rush to repair the damage.

If this process is triggered just occasionally, this immune response repairs the damaged cells and shuts down until additional injury occurs. Unfortunately, the damage produced by most risk factors is constant and chronic. Risk factors such as elevated levels of LDL cholesterol and other lipids (fats), high blood pressure, smoking, and insulin resistance and diabetes cause chronic endothelial dysfunction and inflammation, and keep the immune response stuck in the on position.

Inflammation serves as a mediator in the disease progression by recruiting various immune system fighter and repair cells. The exact pathways by which inflammation exerts its influence are emerging from current research. Scientists are looking especially for inflammation markers that may help physicians diagnose and treat people at high risk of CHD in its early stages before symptoms arise, when lifestyle and medical therapies may halt or even reverse the disease.

Progressing to fatty streaks

Among the factors causing endothelial dysfunction to progress to atherosclerotic plaque, elevated levels of the certain types of cholesterol, particularly low-density lipoprotein (LDL) cholesterol, and other lipids play a major roll.

Here's an overview of what happens:

1. **Excess LDL cholesterol is deposited on the artery walls.**

   When blood levels of cholesterol, particularly LDL cholesterol, are too high, excess LDL cholesterol is deposited on the endothelial lining of arteries where special receptor cells latch on to the LDL molecules.

2. **Trapped LDL damages the cells, triggering the body's immune system into action.**

   This trapped LDL can damage the cells by a process called *oxidation.* The oxidation attracts protective substances related to the immune system. Cells such as macrophages already in artery walls engulf the oxidized excess lipid.

3. **As the immune system tries to remove excess lipids and repair the damage, yellow fatty streaks appear on the artery walls.**

   Soon more circulating fighter cells, known as *monocytes,* enter the artery lining and transform into macrophages to gobble up more excess lipids. Other protective mechanisms such as platelets, T-cells, and growth factors for smooth muscle cells arrive and work hard to restore the damage from excess lipids. As these macrophages engulf the cholesterol, they transform into *macrophage foam cells,* which usually appear as yellow fatty streaks visible on the interior artery walls.

4. **The fatty streaks continue to grow and form scar tissue.**

   When blood cholesterol levels are lower and plenty of HDL cholesterol (the good guys) is present to carry away LDL, these fatty streaks can be halted or reversed. But when excess cholesterol or other risk factors such as the circulating platelets and other clotting factors and excess smooth muscles are present, the deposits typically continue growing.

   As the process seals off the excess lipids, it creates cholesterol-rich pockets covered with scar tissue. These lesions narrow the arteries and typically deform artery walls as they grow larger.

Growing from fatty streaks to large plaques

Decades of time and the presence of various risk factors are required for the fatty streaks to develop into intermediate (moderate-sized, symptomless) and advanced (larger, symptom-producing) plaques.

Growing to moderate, intermediate types of plaque

In the presence of normal mechanical forces, such as the effect of flowing blood against artery walls, and risk factors that can injure artery walls, many fatty streaks begin growing into larger deposits. More cholesterol and other lipid (fat) particles migrate into the artery walls. This happens particularly in areas where the intima of the artery has thickened, probably to adapt to mechanical forces exerted on the arteries.

More and more fatty substances aren't taken into macrophages or the smooth muscle cells; instead, they begin pooling between them. Some cells die and release their lipids into this core. At that point, a thin layer of intimal tissue has begun forming a cap to contain this lipid pool. Other substances such as *cytokines* (various small proteins active in the immune system) and growth factors may also play a role in forming the cap and helping it continue to grow. The formation and growth of the cap mark the transition from *intermediate* lesions to what medspeak terms *advanced* (and typically more dangerous) *lesions.*

Becoming advanced atherosclerotic plaques

As plaques continue to grow, they reach a condition and size that may produce symptoms such as angina, unstable angina, or even heart attack or stroke. The various advanced types of *atherosclerotic plaques* are characterized by a well-defined lipid core that is contained by a cap composed of layers of smooth muscle cells and other substances.

At first this cap appears to be nearly normal intimal layers. But as the plaque grows larger, the composition of the cap's layers changes, becoming more fibrous, or scarlike, as substances such as collagen and calcium enter the mix.

Some advanced plaques are stable, but others are vulnerable to cracking or rupture. When a crack or tear occurs, the lipid core is exposed to arterial blood from which sticky platelets may trigger the formation of a blood clot intended to repair the break. The clot, however, enlarges the size of the plaque. Some plaques grow larger by a cyclical process of cracking and clotting, which gradually narrows the artery. Fewer plaques may grow by a process of cap erosion rather than rupture.

WARNING

The plaques that are more vulnerable to cracking are more likely to form a clot that totally blocks the artery and causes a sudden event such as a heart attack or stroke.

Differentiating between stable and unstable plaques

As individual plaques grow to moderate size and begin exhibiting the rich lipid core and thin fibrous cap associated with the first level of advanced lesions, they appear to be more vulnerable to rupture and dangerous clot formation than larger, older, thicker plaques. Bigger doesn't necessarily mean more vulnerable. The most vulnerable plaques, which can give rise to the deadliest heart attacks, typically block the vessel by only about 40 percent to 50 percent.

Medical scientists and physicians are particularly interested in ways to accurately identify these types of vulnerable plaques, because they seem to be responsible for the majority of sudden acute cardiovascular events, including heart attack, cardiac arrest, and stroke.

Current evidence suggests that stable plaques typically have thicker, more fibrous caps with few inflammatory cells and more calcification, which make the cap tougher. Stable plaques also appear to have fewer lipids within. Although stable plaques often are large, the edges or shoulders of the lesion usually are smooth and tapered.

Unstable plaques, by contrast, are smaller in size but are very rich in cholesterol and incorporate many more inflammatory cells, which release chemicals that degrade the fibrous cap. Unstable plaques often appear structurally weak. In addition, the thinner cap may be easily ruptured or torn by a number of forces, ranging from the normal flow of blood at high stress points in the arterial system to sudden pressures such as suddenly increased blood pressure from exertion.

Researchers continue to look for tests and techniques that accurately identify and assess unstable plaque. Such tools would enable physicians to better identify individuals at greater risk of acute events and begin preventive measures.

Understanding a different type of coronary disease: Microvascular disease

Some people who experience reduced flow of blood to the heart do not have a narrowing of the larger coronary arteries caused by atherosclerotic plaque. Instead, they have coronary *microvascular disease (MVD).* MVD occurs much more often in women than men, particularly in premenopausal or younger women. In MVD,

smaller blood vessels in the heart, which range from 100 micrometers (about the size of a human hair) to 200 micrometers, constrict and prevent adequate oxygenated blood from reaching the heart muscle. As a result, people with MVD may have clear larger coronary arteries but still experience the symptoms of chest pain, although the discomfort is usually more diffuse and may last longer than with angina in CHD.

The causes of MVD are not yet clear, but chronic inflammation appears to play an important role. And the risks factors for CHD, such as high blood pressure (particularly before menopause), unhealthy cholesterol levels, smoking, and diabetes appear to contribute. Current research is also looking for possible risk factors unique to MVD as well as for more effective diagnostic techniques.

TIP

If you have symptoms of heart disease but have clear coronary arteries, ask your physician about MVD, particularly if you are a woman.

Knowing when chest pain is an emergency

WARNING

People with coronary artery disease and angina typically live with this problem for many years and discover how to manage it effectively with appropriate medicines and advice from their physicians. When angina pain changes in character, however, it can signal unstable angina or even heart attack. If you experience any of the following characteristics of chest discomfort, *you need to call 911 and be taken to a hospital immediately:*

- Pain or discomfort that is worse than you have ever experienced before
- Pain or discomfort that is not relieved by three nitroglycerin tablets in succession, each taken 5 minutes apart
- Pain or discomfort that is accompanied by fainting or lightheadedness, nausea, or cool clammy skin
- Pain or discomfort lasting longer than 20 minutes

If any of these symptoms occur, you need to call an ambulance and be taken immediately to a hospital. Under no circumstances should you drive yourself to the hospital.

Symptoms and Manifestations of Coronary Heart Disease

Because every person is an individual, physical responses to progressive coronary artery disease vary. Not every individual with heart disease has every manifestation and symptom of the condition. But these manifestations are typical:

- **Nothing:** Many people can have significant coronary atherosclerosis but experience no discomfort or other sign of the disease. That's why this condition is known in medicine as *silent ischemia. Ischemia* means lack of blood flow. People with diabetes are particularly susceptible to silent ischemia, but others can have it, too.
- **Angina:** More formally known as *angina pectoris,* angina is typified by temporary chest pain, usually during exertion. This pain usually is felt as a tightness or uncomfortable feeling across the chest or up to the neck and jaw, not as a sharp stab. Angina also may have other manifestations.

WARNING

- **Unstable angina:** Chest pain that is new, occurs when you're at rest, or suddenly grows more severe is called *unstable angina.* It's a medical emergency.
- **Heart attack:** Completely cutting off blood flow to a coronary artery causes an acute heart attack, or *myocardial infarction (MI),* the most severe result of coronary heart disease. The closure can be gradual or the result of a blood clot. A spasm in a coronary artery, particularly in the area of a narrowing, may also result in heart attack.
- **Sudden death:** The cause of sudden death from coronary heart disease often is a rhythm problem such as ventricular tachycardia or ventricular fibrillation. These rhythm problems sometimes occur in the setting of an acute heart attack. We've highlighted it here to make the point that the first indication or symptom for some people that they have CHD is a fatal cardiac arrest or heart attack. Many of these deaths happen to people in their 50s, 40s, or younger.

Angina typically is a discomfort felt in the chest, often beneath the breastbone or in nearby areas such as the neck, jaw, back, or arms.

- Individuals often describe the chest discomfort as a "squeezing sensation," "vicelike," "constricting," or "a heavy pressure on the chest." (In fact, the term *angina* comes from a Greek word that means "strangling" — a strangling pain.)

- Angina often is brought on by physical exertion or strong emotions and typically is relieved within minutes by resting or using nitroglycerin.
- Some individuals, particularly women, may experience angina as a symptom different from chest discomfort or in addition to it. Shortness of breath, nausea, faintness, abdominal pain, indigestion, or extreme fatigue may also be manifestations of angina.
- When chest pain occurs at rest, it usually is classified as *unstable angina.*

Understanding the causes of angina

You know how your muscles begin to scream when you run faster than your blood can carry adequate oxygen to them. The same thing may happen when the coronary arteries become so narrowed by atherosclerotic plaques that blood flow to the heart is inadequate to supply the heart muscle with the oxygen it needs. The temporary chest discomfort known as *angina* is your heart's way of getting your attention. It occurs when you ask your heart to work harder, and it therefore demands more blood — for instance, when you're walking briskly or running, climbing a hill or stairs, having sex, or doing housework or yardwork. Strong emotions such as fear or anger also can trigger an episode.

Angina usually does not damage the heart. It is a temporary condition — the usual episode lasts only 5 to 10 minutes. (In MVD, the episodes can last longer, about 10 minutes up to 30 minutes.) Chest discomfort makes you stop and rest, slowing the heart and lessening its demand for blood. Alternatively, most people with angina know to take a nitroglycerin tablet under the tongue when they have an angina attack. The nitroglycerin dilates the coronary arteries, enabling blood flow to the heart to increase.

WARNING

Any discomfort that doesn't stop with rest or that lasts more than 5 to 10 minutes may be a heart attack and needs to be treated as an emergency.

Diagnosing angina

A description of the discomfort a person experiences provides the most important information leading to the diagnosis of angina. However, your physician will typically order appropriate tests based on your symptoms and signs. These may range from an electrocardiogram, exercise stress test, or stress echocardiogram to nuclear stress testing and cardiac catheterization. Some of these tests can be conducted in your physician's office, but others require the resources of a hospital.

Not all chest pain is angina or involves the heart. Various conditions involving other structures in the chest can occasionally cause chest discomfort; these include spasm of the esophagus, acid reflux, hiatal hernia, and muscular pain.

People who have angina typically can live comfortably for many years with this condition by finding out how to manage the symptoms and lower their risk factors for complications.

Defining unstable angina

As the name suggests, *unstable angina* results when angina gets out of control. In unstable angina, the lack of blood flow and oxygen to the heart becomes acute and, therefore, very dangerous because the risk of complications such as heart attack is much greater.

Where stable angina has typical characteristics and predictable triggers, such as exertion or strong emotion, unstable angina is characterized by one or more of the following symptoms:

- Anginal discomfort at rest or when awakening from sleep
- A significant change in the pattern of the angina where it occurs with less exertion or is more severe than before
- A significant increase in the severity or frequency of angina
- New onset, or first experience, of anginal chest pain

If you experience any one of these characteristics, you must seek immediate medical attention.

Defining a Heart Attack

A heart attack, known medically as a *myocardial infarction (MI)*, occurs when one of the three coronary arteries that supply oxygen-rich blood to the heart muscle *(myocardium)* becomes severely or totally blocked, usually by a blood clot. When the heart muscle doesn't receive enough oxygenated blood, it begins to die. The severity of the heart attack depends on how much of the heart is injured or dies when the attack occurs.

REMEMBER

If you think you're having a heart attack, it's critical to go immediately to a hospital by ambulance where therapy can be initiated to save your heart muscle from dying. New clot-busting medicines, as well as procedures such as angioplasty, often can dissolve a clot that causes the heart attack, open the blood vessel, and save some or all of the heart muscle at risk. Although some of the heart muscle usually dies during a heart attack, the remaining heart muscle continues to function and often can compensate, to a very large degree, for the heart muscle that has died.

Understanding causes of a heart attack

Heart attack almost always is caused when a blood clot forms at the site of an existing fatty plaque that has narrowed the coronary artery. Thus, individuals are at much higher risk for heart attack if they

- Have a history of coronary heart disease
- Have experienced previous bouts of angina
- Have suffered a previous heart attack

The blockage that triggers a heart attack usually is caused by an acute blood clot. Most *acute blood clots* occur when one of the plaques or fatty deposits on the artery walls cracks or ruptures. Other, much more rare causes of acute blockages in arteries, such as a severe coronary artery spasm, can also cause heart attack.

Recognizing symptoms of a heart attack

Different people experience the symptoms of a heart attack in different ways. However, typical symptoms include some or all of the following symptoms (as described by the American Heart Association):

- Uncomfortable pressure, fullness, squeezing, or pain in the center of the chest lasting more than a few minutes
- Pain spreading to the shoulders, neck, or arms
- Chest discomfort with lightheadedness, fainting, sweating, nausea, or shortness of breath

In an individual who has angina, symptoms may be particularly difficult to differentiate from the chest discomfort of angina. However, when a heart attack is occurring, chest discomfort usually is more severe and may occur while the individual is at rest or less active than usual.

The signs of a heart attack often are subtle, particularly with individuals who have diabetes. People with diabetes may not have the classic symptoms of chest, shoulder, or arm discomfort. Chest pain experienced by many women likewise may not present the classic symptoms.

WARNING

Coronary heart disease is extremely common in men and women in the United States and particularly in men in their 40s and older and postmenopausal women. Even if you've never had a single sign of trouble, you need to call 911 and go straight to the hospital for prompt evaluation whenever you experience any of the preceding warning signs. Do not take a meeting. Do not put it off for an hour . . . *just call 911 and go!*

REMEMBER

About two-thirds of the individuals who experience an acute heart attack also experience some warning symptoms in the weeks or days preceding the acute event. They often don't realize what the warning signs were until after the event — with keen hindsight. So work on your foresight: Know the warning signs of heart attack and take them seriously.

Differentiating between heart attack and sudden cardiac arrest

Although doctors often call sudden cardiac arrest "a massive heart attack," the two technically are not the same thing. A *massive heart attack* (myocardial infarction) results from a blockage of the coronary arteries. *Sudden cardiac arrest* is caused by ventricular fibrillation, an electrical malfunction in which the heart begins to quiver rapidly, instead of contracting and pumping blood regularly. Cardiac arrest strikes without warning. Because blood flow essentially stops, victims of cardiac arrest lose consciousness and die within minutes unless emergency help is available.

Many victims of sudden cardiac arrest have underlying CHD. Sudden cardiac arrest often (but not always) occurs in the setting of an acute heart attack. It can also occur from electrical malfunction when a heart attack is not involved.

Taking Action — Immediately — for a Possible Heart Attack

Unfortunately, many people who are experiencing a heart attack either don't recognize symptoms or deny them. Doing so can be a serious or even fatal mistake, because delay

- Significantly increases the risk of sudden death from heart rhythm problems in the early phases of a heart attack.
- Increases the likelihood that a significant amount of heart muscle will die, thus increasing the likelihood and extent of the heart attack, causing disability if the individual survives.

If you or a loved one experiences any symptoms or warning signs of a heart attack, use the six-point survival plan outlined. Don't delay!

TIP

This six-point survival plan, adapted from American Medical Association recommendations, can save your life. Take these steps if you or a loved one is experiencing the symptoms of a possible heart attack:

1. **Stop what you are doing and sit or lie down.**
2. **If symptoms persist for more than two minutes, call your local emergency number or 911 and say that you may be having a heart attack.**

   Leave the phone off the hook so that medical personnel can locate your address in the event that you become unconscious.
3. **Take nitroglycerin, if possible.**

   If you have nitroglycerin tablets, take up to three pills under your tongue, one at a time, every five minutes, if your chest pain persists. If you don't have nitroglycerin, take two aspirin.
4. **Do not drive yourself (or a loved one) to the hospital if you think you are having a heart attack.**

   Ambulances have equipment and personnel who are trained to deal with individuals who are having a heart attack. Driving yourself or a loved one to the hospital is an invitation for a disaster.
5. **If the person's pulse or breathing stops, any individual trained in cardiopulmonary resuscitation (CPR) needs to immediately begin to administer it.**

   If an automated external defibrillator (AED) is available, use it. Call 911 immediately, but do not delay instituting CPR or using an AED.
6. **When you arrive at the hospital emergency room, announce clearly that you (or your loved one) may be having a heart attack and that you must be seen immediately.**

   Don't be shy about it.

Other Manifestations of Heart Disease

Atherosclerosis, angina, and heart attack aren't the only types of heart disease. This section looks briefly at four other common types of heart disease — arrhythmias (rhythm problems), heart failure, stroke, and heart valve problems.

Arrhythmias

Arrhythmias (or *dysrhythmias)* refer to problems with the electrical system that controls the heart's normal rhythm. They may occur in the context of an acute heart attack or from other causes.

WARNING

When the electrical system goes entirely haywire, it may result in a very dangerous condition called *ventricular tachycardia,* which occurs when an abnormal electrical impulse causes the heart to beat so fast that it cannot pump out adequate blood. This condition can rapidly degenerate to *ventricular fibrillation,* which occurs when the heart simply quivers and produces no blood flow. Ventricular fibrillation must be immediately terminated by an electrical shock, or *defibrillation,* administered by a medical professional.

The *atria,* the upper chambers that assist the ventricles (refer to Figure 1-2), can also have rhythm problems. One of the most common is atrial fibrillation. *Atrial fibrillation* occurs when electrical signals are chaotic, and muscles of the atria quiver rather than contract. The electrical impulses also reach the ventricles very erratically, thus producing an erratic heartbeat. This condition is common in individuals who have heart disease and can be caused by a variety of conditions including coronary heart disease, hypertension, and an elevated thyroid level.

Atrial fibrillation, by itself, is not immediately life-threatening. But because the atria are not contracting effectively, they can gather clots that can pass through the heart and into either the brain, potentially resulting in a stroke, or the lungs, potentially resulting in a very serious condition called *pulmonary embolism.* Because of the possible outcomes, treating atrial fibrillation aggressively is important. Treatment may include medications or *cardioversion* (an electrical procedure that restores normal rhythms) or both. In addition to medicines to control the heart rate, all patients who have atrial fibrillation must take blood-thinning medicines (anticoagulants) to lower the risk of blood clots generated in the atria being thrown to the brain or lungs.

Heart failure

Heart failure occurs when the heart no longer adequately pumps blood to the lungs and throughout the body. It's usually a slow process that takes place over years.

Underlying conditions, such as CHD, leakage from one of the heart valves, an acute heart attack, or various diseases of the heart muscle itself usually cause heart failure.

The heart initially compensates for small decreases in its ability to pump by doing the following:

- Enlarging *(dilatation)* to enable more blood into its pumping chambers
- Thickening the muscle walls *(hypertrophy)* to strengthen the pump and enable it to exert more force during its contraction to move more blood
- Beating faster to make up for decreased volume or power (like trying to pitch more, but smaller, pails of water on a fire)

The heart may try to compensate in these ways for years before you notice any symptoms. But when these mechanisms ultimately fail, significant heart failure occurs. By then, compensatory mechanisms often have become part of the problem.

How serious heart failure is depends on how much pumping capacity the heart has lost. A normal heart discharges about 60 to 75 percent of the blood in the main pumping chambers with each contraction, or beat. Heart failure often occurs when the amount of blood ejected per beat, called the *ejection fraction,* drops below 50 percent, and when the ejection fraction falls below 40 percent heart failure ensues. Even so, many people can survive for many years with ejection fractions of only 20 to 30 percent, or sometimes even 15 percent.

REMEMBER

All forms of heart failure are serious health problems that require medical treatment. Taking care of yourself, seeing your physician regularly, and paying scrupulous attention to recommended treatments including lifestyle modifications are important steps you can take to improve your chances of living longer. Fortunately, many effective treatments are now available for heart failure and experimental procedures such as stem cell therapy are promising.

Stroke

A stroke occurs when a blood clot or bleeding suddenly interrupts the flow of blood to an area of the brain. When deprived of blood, brain cells lose their ability to function and, if deprived for too long, die. Because brain cells and groups of brain cells have highly specialized functions, the location of stroke damage determines what loss of neurological and bodily function occurs as a result of stroke. Impairment may be temporary or permanent.

Strokes are categorized in two basic ways: ischemic stroke and hemorrhagic stroke. The causes and results of stroke depend on how and where the stroke occurs.

Ischemic stroke

An *ischemic stroke* occurs when a blood clot or other particle blocks a blood vessel in the brain and cuts off the blood supply to the portion of the brain supplied by that vessel. Without adequate oxygen, that portion of the brain suffers damage or even dies. This type of stroke is called an ischemic stroke because it's caused by *ischemia*, the medical term for lack of blood flow.

About 70 to 80 percent of all strokes are ischemic, and they occur in two basic forms:

- **Cerebral thrombosis:** This form of stroke results from progressive narrowing of arteries in the brain or sometimes in the carotid arteries in the neck. In a thrombosis, plaque (there's that cholesterol again) that narrows the artery and any clot *(thrombus)* that forms on it doesn't move, meaning the typical underlying causes for this type of blockage are atherosclerosis and high blood pressure. Before having a major stroke, many people experience a temporary lack of blood flow to the brain that's called a *transient ischemic attack* (TIA), which is a small stroke in which the effects usually last for only a few minutes or hours.

  
  WARNING

  Never ignore possible TIA symptoms; treat them as a serious warning and consult your physician.

- **Cerebral embolism:** This form of stroke occurs when a blood clot, or *embolus,* travels from somewhere else in the body to the brain. When the blood clot lodges in a vessel in the brain, it cuts off blood flow to the portion of the brain supplied by that vessel. Blood clots that cause strokes may form in and travel from a number of different locations in the body, including:
  - **Major arteries in the neck that supply the brain (the carotid arteries).** This is why your doctor often listens with a stethoscope over your neck to hear whether any narrowing of one of these arteries has occurred. The narrowing of the artery leads to turbulence that the doctor can hear.
  - **The heart — particularly in people with atrial fibrillation.** In this case, when the upper chambers, or atria, fibrillate instead of contracting normally, blood clots can form in the blood and travel directly from the left atrium to the brain.

Hemorrhagic stroke

A *hemorrhagic stroke* occurs when a blood vessel in or on the brain bursts and bleeds into the brain or into the space between the brain and skull. This type of stroke is called a hemorrhagic stroke because it's caused by a *hemorrhage* (in Greek, *hemo* means "blood" and *rhage* means "to break"). The brain is very sensitive to bleeding and pressure, which damage brain tissue, often permanently. Hemorrhagic strokes account for only about 20 percent of all strokes, but they usually are more severe and more often fatal than ischemic stroke.

Heart valve problems

As mentioned, the four heart valves serve as traffic cops of the heart, directing blood flow in the proper direction and preventing it from improperly backing up. As long as these valves open fully and shut tightly, all is well. But if any disease or injury causes valve leakage *(regurgitation)* or narrowing *(stenosis)*, major problems can result.

Significant valve leakage can overload the heart because extra blood flowing back into the heart requires an extra strong beat to eject it. A narrowed valve can cause the heart to thicken because it is being asked to pump against a much higher pressure.

A number of different conditions cause valves to leak or narrow, including:

- Congenital valvular problems (a condition you're born with).
- Damage to valve structures, such as when the structures that anchor the flaps of the mitral valve break. Heart attack can also seriously damage the muscles that control the valves.
- Progressive problems, including those that may result from the aging process, such as calcification, or those that result from an infection, such as rheumatic fever or endocarditis. If the problem becomes too severe, it may require open-heart surgery and valve replacement.

If a valve abnormality is not progressing rapidly or causing any serious problem, your physician may simply keep a close eye on it so that treatment can be initiated when and if it becomes necessary. Following a heart-healthy diet and lifestyle can also support valve health. Taking preventive antibiotics as needed when you have an underlying valve abnormality is a good idea. There are also various medical and surgical treatments for valve problems.