

HEART ATTACKS AND CORONARY ARTERY DISEASE

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INTRODUCTION

Coronary artery disease has probably affected human beings throughout history, but it is only in the last century or so that it has emerged as a leading cause of death. The first description of the symptoms of coronary artery disease was written in 1768 by William Heberden, an English physician. Dr. Heberden coined the term “angina pectoris”—from the Latin, *angere*, which means to strangle or distress, and *pectoris*, “of the chest”—and his classic description still holds true today

There is a disorder of the breast, marked with strong and peculiar symptoms considerable for the kind of danger belonging to it, and not extremely rare, of which I do not recollect any mention among medical authors. The seat of it, and sense of strangling and anxiety, with which it is attended, may make it not improperly be called Angina pectoris.

Those, who are afflicted with it, are seized while they are walking and more particularly when they walk soon after eating, with a painful and most

disagreeable sensation in the breast, which seems as if it would take their life away, if it were to increase or to continue; the moment they stand still, all this uneasiness vanishes.

Although the relationship between angina pectoris and diseased coronary arteries was established just a few years later, it was not until the early 20th century that the medical profession gave widespread recognition to coronary artery disease as a major cause of death. Such recognition may have been slow in coming because the disease was not widely prevalent until around the middle of the 19th century. With the advent of improved sanitation, immunization, and other advances in public health, the death toll from infectious diseases—previously the leading cause of death—dropped. In industrialized nations, these advances in public health coincided with lifestyle changes, such as adoption of a diet high in meat and other fatty foods, an increase in cigarette smoking, and a more sedentary life-style. It was at this time that the death rate from heart attacks began to soar. (See Chapter 3.)

According to statistics compiled by the Centers for Disease Control, almost one in two Americans dies of cardiovascular disease. The total annual toll is

more than 975,000; of these, about 500,000 die of heart attacks. The large majority of heart attacks results from coronary artery disease, a condition that afflicts about 5 million Americans.

Of course, mortality statistics are only part of the story—coronary artery disease also affects life-style, productivity, and the economy. According to 1991 figures compiled by the American Heart Association and the National Center for Health Statistics, about 6 million Americans have a history of a heart attack, angina, or both. Although the likelihood of a heart attack increases with age, a large number of Americans—mostly men—are struck down during their most productive years. About 45 percent of heart attacks occur before the age of 65, with 5 percent before age 40. The American Heart Association puts the total annual cost of cardiovascular disease at \$94.5 billion, a figure that includes both direct medical costs and estimated lost productivity resulting from disability.

Fortunately, the number of deaths from coronary artery disease—while still unacceptably high—has been steadily declining since the late 1950s. In 1950, the age-adjusted death rate from heart attacks was 226 per 100,000 Americans. By 1986, this had dropped by nearly half to 129 per 100,000. For example, the five-year survival rate of patients with angina improved from 75 percent in the years 1950 to 1970, to 87 percent during 1970 to 1975. Much of this improvement is undoubtedly the result of improved medical care. But altered life-style factors such as smoking cessation and a reduction in fat consumption are also believed to lower the risk of premature death from a heart attack.

ANATOMY OF THE HEART

The normal human heart has two major coronary arteries, so named because, together with their branches, they surround the heart like a crown (or *corona*). From its branch off the aorta, the left main coronary artery quickly divides into two vessels: the left anterior descending artery and the circumflex artery. Another vessel, the right coronary artery, comes off the aorta and supplies blood to the right and bottom parts of the heart. The three vessels supply all the oxygenated blood necessary to keep the heart's muscle and electrical conduction system functioning and viable. (See Chapter 1.)

Although the body's entire volume of blood passes through the heart's chambers approximately every 60 seconds, only about 5 percent of the total amount of oxygenated blood is available for the heart's own energy needs. The coronary arteries (which are 3 to 5 millimeters or 1/8 to 1/5 of an inch in diameter) are the sole conduits for this supply. Because heart muscle (myocardium) extracts oxygen from arterial blood with maximum efficiency, any increase in the heart's workload requires an increase in the blood supply. When there is an imbalance between the available supply of blood (oxygen) and demand for blood (oxygen), the heart muscle becomes oxygen-deprived, a condition known as myocardial ischemia. Without adequate blood flow to the heart muscle, the heart itself is unable to function properly.

OXYGEN DEPRIVATION (ISCHEMIA)

For the majority of people suffering from coronary artery disease, the supply of oxygenated blood is reduced due to a progressive narrowing of the open channels (the interior lumens) of the coronary arteries. This is due to atherosclerosis, a disease in which scattered lesions, known as atherosclerotic plaques or atheromas, appear on the inner wall of the coronary artery. See Figure 11.1, a series of illustrations showing how an artery becomes blocked. (The word *atheroma* comes from the Greek for porridge, because atheromas contain a porridgelike mixture of cholesterol, fat, and fibrous or starlike tissue.)

The first signs of atherosclerosis can appear at an early age. A significant proportion of males in their teens and early 20s may already have fatty streaks and other evidence of the disease on the walls of their coronary arteries—as was first demonstrated by autopsies conducted on young American soldiers killed during the Korean War. The buildup of atherosclerotic plaque is a gradual process, however, and it may take upward of 20 years or more from the first appearance of fatty streaks before the coronary arteries are blocked enough to produce symptoms such as angina or shortness of breath. Symptoms usually do not occur until the coronary artery has been narrowed by about 50 to 70 percent. Even with significantly clogged coronary arteries causing ischemia, however, many people do not experience symptoms. This is referred to as silent ischemia.

The exact causes of buildup of atherosclerotic

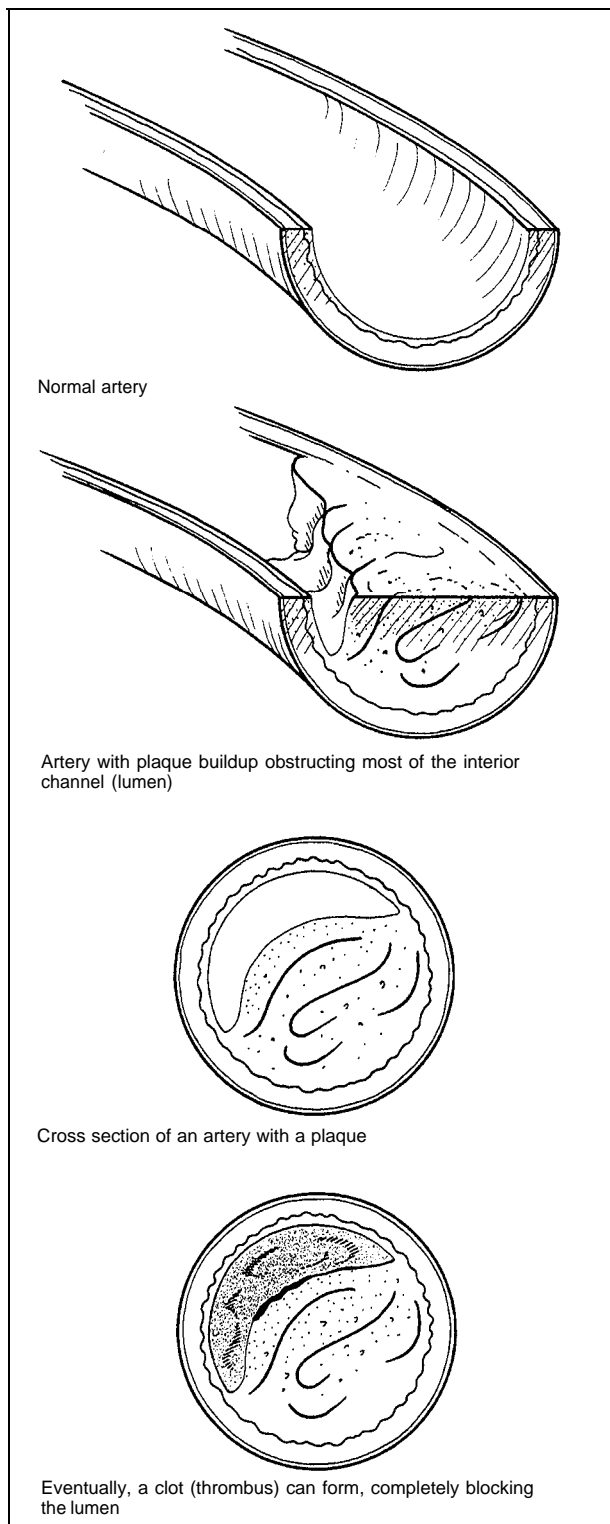


Figure 11.1
How a normal artery *may* become blocked by fatty plaque,

plaque are not understood, nor is it possible to pinpoint how they begin or what their course will be. However, evidence based on a number of long-term studies (such as the Framingham Heart Study, which has examined the health of several thousand men and women in the Boston suburb of Framingham from

1948 to the present day) has enabled us to identify which *risk factors* increase the likelihood that someone will develop atherosclerosis. These risk factors include some that are controllable, such as smoking, hypertension, and elevated blood cholesterol, as well as age, gender, family history, and other factors that are beyond our control. (See Chapter 3.)

In addition to atherosclerotic plaque buildup, spasms of the muscles that encircle the coronary arteries can also interrupt the coronary blood supply. In 85 percent of people who have coronary artery spasms, atherosclerosis is also present. In about 10 to 15 percent of people with typical anginal chest pains, spasms may be the sole cause of the oxygen deprivation (ischemia) and resulting pain.

Some people who experience angina may have normal coronary arteries. The angina some of these people experience may be caused by a constriction or narrowing of the aortic valve. In others—who may have no evidence of coronary artery spasm, heart valve disease, or left ventricular heart muscle abnormality—there is no clear reason for the angina. These people generally have an excellent overall prognosis.

An inability to deliver adequate oxygen during rest or periods of increased demand can result in ischemia manifested by angina and other symptoms. Factors affecting the heart muscles' demand for blood include blood pressure, heart rate, and the size of the left ventricle.

A sizable percentage of people suffer from chronically high blood pressure. In addition, blood pressure temporarily rises during exercise or periods of stress. (See Chapter 12.) The heart rate is increased by exertion, fever, stress, and an overactive thyroid. Enlargement of the main pumping chamber—the left ventricle—is commonly the result of hypertension or certain heart valve disorders. All of these conditions result in increased work for the heart and the need for more oxygen. If this cannot be supplied, symptoms may occur.

SYMPTOMS

The primary symptom of coronary artery disease is chest pain or *angina*, which is not itself a disease but a set of symptoms closely corresponding to Heberden's original description. A person suffering from angina may clutch a fist to the chest while describing

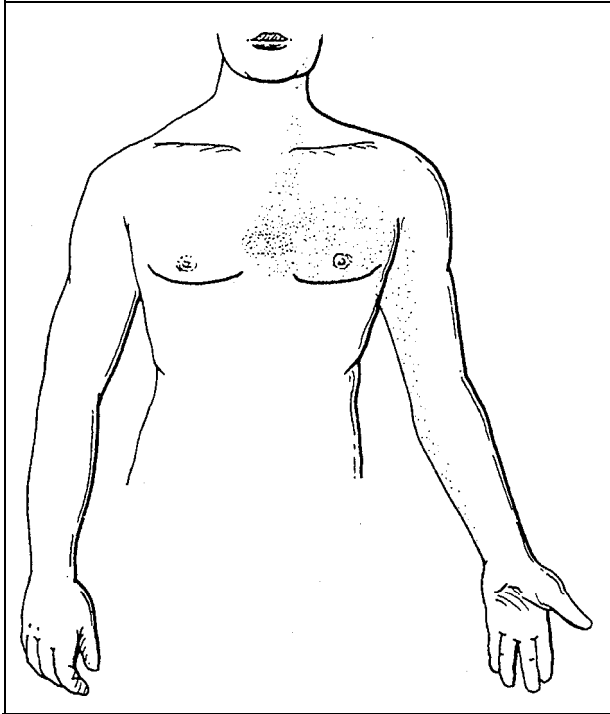


Figure 11.2
The shaded areas show locations for angina. It occurs most frequently or classically in the center of the chest (most heavily shaded area), but it may also radiate to the whole chest, neck, jaw, and down the arm, particularly on the left side. Angina can also occur in these places without occurring in the center of the chest.

a feeling of discomfort or pain, often using such words as “pressure” or “heaviness.” This pain is usually located in the center of the chest but may radiate to or occur only in the neck, shoulder, arm, or lower jaw, particularly on the left side. (See Figure 11.2.) Brief sharp stabbing or sticking pains confined to a small area of the chest are rarely caused by angina.

For most people, these symptoms almost always occur during or after physical activity and/or emotional stress and are more likely to occur following a meal or in cold weather. People who have what is known as stable angina can often predict with reasonable accuracy the amount of activity that precipitates an attack—sometimes to the point of knowing how many stairs they can climb before pain begins. Typical activities that might bring on angina include walking up several flights of stairs, climbing a hill, or other sudden vigorous activities, such as running for a bus or playing tennis. A change in anginal pattern, such as increased frequency of angina or the new onset of angina at rest, is referred to as crescendo or unstable angina.

Angina may be more likely to occur following a meal, because blood pools in the stomach and the intestinal tract during digestion, increasing the work

of the heart. During cold weather, angina may also be more frequent because vessels may go into spasm, increasing the work of the heart while simultaneously decreasing the blood supply to the heart. In general, anginal symptoms usually fade and disappear when the person ceases the particular activity that provoked them.

Ischemia may occasionally occur without symptoms of angina or other discomfort, so-called “silent ischemia.” Some patients may experience only silent episodes of ischemia, whereas others have episodes with and without angina. The potential danger of silent ischemia is that someone may not be aware of the reduced blood flow to the heart muscle and might, therefore, be less likely to cease the activity precipitating it. The diagnosis, significance, and treatment of silent ischemia are areas of active research.

DIAGNOSIS

Angina is a clinical diagnosis; however, diagnosing coronary artery disease purely on the basis of symptoms may be difficult. The discomfort of angina is not always experienced in the same way, and a patient's symptoms may be vague. Chest pain can also occur in a variety of other conditions that may exist alone or may accompany coronary artery disease. It is, therefore, important for the physician to distinguish between anginal pains and chest pain from other sources. (See Table 11.1.) Anginal pain usually begins gradually and lasts for several minutes, generally fading when the individual stops the activity that precipitated the attack or takes a medication such as nitroglycerin, which widens (dilates) the coronary arteries and increases blood supply to the heart muscle.

Angina is probably not the cause of the chest discomfort if it lasts less than 5 seconds or more than 20 minutes (provided the patient is not having a heart attack), if the pain is sharp or “stabbing,” if it is precipitated by a sudden movement or deep breath, if it is confined to a small area, if it is not relieved by rest or cessation of physical activity (again, provided the patient is not having a heart attack), or if the chest wall is tender to the touch.

The sources of chest pain that mimic angina include esophageal or stomach disorders (for example, reflux of stomach acid into the esophagus, resulting in “heartburn”), pain due to obstruction of the bile duct, inflammation of the cartilage of the chest wall, and arthritis of the bones in the neck.

Table 11.1
Identifying Causes of Chest Pain

Causes	Type of chest pain and other symptoms	Causes	Type of chest pain and other symptoms
Blood clot in the lung (pulmonary embolism)	Chest pain accompanied by breathlessness, faintness, cough bringing up bloody phlegm, blueness (cyanosis) around the mouth.	Lack of oxygen in the heart (angina)	Dull, heavy, constricting pain in the center of the chest that can spread to throat and upper jaw, back, and arms (mainly left arm). Pain appears when person is active and disappears when activity stops and person rests. Can be accompanied by difficult breathing, sweating, nausea, and dizziness.
Broken rib	Pain in or near the chest area that increases with pressure or movement; area around fracture may be swollen and bruised.	Nerve infection (shingles) in the chest area	Intense, knifelike pain in one area of the chest that precedes, by several days or less, a rash (groups of blisters on the skin—much like chicken pox—above the affected nerve); pain continues through and after rash appearance.
Collapsed lung (pneumothorax)	Usually sudden sharp chest pain on one side of the body, accompanied by breathlessness; may be discomfort rather than pain, may include pain at the bottom of the neck and tightness across chest.	Pneumonia	Respiratory illness, including cough and fever, precedes other symptoms, including chest pain, shortness of breath, chills, sweating, bloody or yellow phlegm, or delirium.
Heart attack	Crushing pain in the center of the chest, accompanied by difficult breathing, sweating, nausea, or a feeling of faintness.	Pulled muscle in the chest area	Pain, stiffness, or tenderness in the chest area as a result of overstretching a muscle (for instance, while working out); area may become swollen as a result of internal bleeding.
Heartburn and hiatus hernia	Painful burning sensation in the chest that becomes worse when person bends forward or lies down; person may also experience belching and regurgitation of acidic fluid.		
Infection of the airways in the lungs (acute bronchitis)	Pain in the upper chest that worsens when coughing; deep cough that brings up grayish or yellowish phlegm from the lungs.		

Disorders of the heart can also result in anginalike symptoms. These include an elevated pressure in the lungs (pulmonary hypertension) or a blood clot in an artery supplying the lungs (*pulmonary embolism*), resulting in lack of oxygen delivery to the lung tissue. Inflammation or infection of the tough outer sac that covers the heart (*acute pericarditis*) can produce persistent chest pain, which usually comes on suddenly and is aggravated by coughing or movement.

Further complicating the difficulty of making a diagnosis of coronary artery disease on the basis of symptoms alone is the existence of silent ischemia. Some people—who may or may not also occasionally experience anginal discomfort—can show all the clinical signs of an attack of angina and yet may not feel any discomfort at the time. This syndrome appears to be more common in persons with diabetes.

The presence of chest discomfort in someone who

has several risk factors for heart disease strongly suggests to the physician that the patient has coronary artery disease. However, accurate diagnosis of coronary artery disease in these people (and those with chest pain who do not fit the risk profile) may require some of the following tests.

ELECTROCARDIOGRAPHY

The electrocardiogram (ECG), a graphic record of the electric currents generated by the heart, is an essential tool for the diagnosis of coronary artery disease. An ECG taken while a person is resting (a resting, or baseline, ECG) will not show evidence of lack of oxygen in the heart muscle—unless the patient is having an attack of angina at the time—but it can demonstrate the presence of a previous heart attack or other changes suggesting that the heart muscle may not be receiving an adequate blood supply.

The baseline ECG can provide a considerably more useful diagnosis if the patient experiences angina during testing. The test can then not only show that inadequate oxygen is reaching the heart muscle, but also give an idea of which artery is blocked and the extent of heart muscle that maybe at risk.

The exercise ECG—popularly known as an exercise stress or tolerance test—is another useful tool for diagnosis. While being monitored, the patient engages in physical activity of progressive intensity, usually on a treadmill, stationary bicycle, or stair-climbing device. Exercise is usually continued until the heart rate reaches 85 percent of a calculated so-called maximum level—about 220 minus the person's age (for a 60-year-old person, about 160 beats per minute), or until symptoms of fatigue or chest pain or significant ECG changes are noted.

If coronary arteries are healthy, they dilate or open up to supply the extra blood and oxygen necessary to sustain the extra heart muscle workload. If this occurs, the electrocardiogram shows few changes. If the arteries are narrowed or go into spasm, however, portions of the heart muscle do not get enough blood and ECG changes will occur.

If changes occur at low work levels after only a few minutes and/or at a heart rate of only about 100 beats per minute, this suggests that coronary heart disease may be fairly severe. It is also useful to monitor a patient's blood pressure and heart rate during

the test, because a drop in blood pressure during exercise also implies that the extent of coronary artery disease may be severe. The exercise ECG can correctly identify about 65 to 75 percent of people with coronary artery disease.

Helter monitoring, or the ambulatory ECG, which is worn for 24 to 48 hours, may be a useful tool in diagnosing some cases of angina. ECG changes may be recorded during episodes of angina. Like other tests in cardiology, it may not be necessary in all cases.

RADIOISOTOPE SCANS

Thallium-201 is an isotope that is used for diagnosing coronary artery disease. This radioactive substance is injected and passes through the bloodstream into the heart muscle cells. The distribution of thallium is recorded with a gamma camera, and areas of heart muscle that are not getting sufficient oxygen show up as “cold spots” in which the blood flow did not deliver thallium or the heart muscle cells did not take it up. Combined with the exercise ECG, an exercise thallium test helps to correctly identify about 90 percent of people with coronary obstructions. This test also helps to locate the specific sites of lesions. Recently, new radioisotope agents have become available that may replace thallium-201 in the future. (See Chapter 10.)

Another diagnostic tool, multigated acquisition scan (MUGA), which uses the radioisotope technetium, can also provide information on the size and contraction pattern of the left ventricle. Contraction abnormalities that are induced by exercise can indicate coronary artery disease. Ischemic or infarcted (dead, due to a heart attack) regions of the heart usually contract abnormally.

People who are unable to tolerate an exercise test because of orthopedic problems or impaired leg circulation (see Chapter 17) can be effectively tested using thallium combined with a potent drug, dipyridamole (Persantine). Dipyridamole causes the coronary arteries to dilate (as they should to satisfy the increased demand for oxygenated blood created by exertion) and thus increases the blood flow. If there are blockages in an artery, the increase of flow does not occur and a “cold spot” is imaged. This test compares favorably with exercise thallium imaging.

ECHOCARDIOGRAPHY

Portions of the heart can be seen using an ultrasound method called echocardiography. The echocardiogram is a useful diagnostic tool for determining impaired function and increased thickness of the walls of the left ventricle as well as for helping to rule out other cardiac problems such as valve disease. (See Chapter 14.) As with the MUGA, abnormalities in ventricular contraction (wall motion abnormalities) can be documented by the echocardiogram during exercise or pharmacologic stimulation. A diagnosis of angina and/or coronary heart disease can usually be made without the use of this test, however.

CORONARY ANGIOGRAPHY

X-ray imaging of the coronary arteries can be performed in a cardiac catheterization laboratory. Here, with the patient under mild sedation, an opening is made to a blood vessel in the groin or arm, and a thin tube is threaded up through the vessel to the heart. A dye that shows up on X-ray is injected into the coronary vessels to outline their lumen, and into the left ventricle to assess its contraction. This sequence of events is captured on motion-picture X-rays (angiograms).

Coronary angiography provides the “gold standard” diagnosis of the extent and location of disease in the coronary arteries. Angiography also gives a clear indication of whether the left ventricle is functioning well. A stenotic or narrowed vessel can be identified by an indentation or narrowing in the column or channel of dye in the vessel due to the obstructing plaque or clot. The severity of the stenosis can be quantified by the percent of the narrowing of the dye channel. An occluded vessel can be identified because it contains little or no blood and hence shows little or no dye beyond the blockage.

People are sometimes fearful of this procedure because it does carry a small risk of mortality (0.1 percent) or adverse reactions such as heart attack or stroke (less than 3 percent), but the potential life-saving benefits of an accurate diagnosis may outweigh by far the modest risk. Obviously, however, all people with angina do not need catheterization. The cardiologist will usually consider whether the diag-

nostic benefits exceed the potential risks given any of the following situations:

- A patient who is under medical treatment combined with life-style changes continues to suffer from incapacitating angina. Such people can usually be relieved of their pain by coronary bypass surgery or angioplasty, and coronary angiography is necessary to determine whether their arteries are suitable for either procedure.
- An electrocardiogram and other tests suggest that a patient risks damage to a considerable portion of the heart muscle (for example, the patient who has marked electrocardiogram [ECGI changes after only a few minutes of a stress test). Certain severe anatomic subsets of coronary artery disease (as shown using coronary angiography) are better treated with coronary bypass surgery.
- Coronary angiography may sometimes be suggested to evaluate the coronary anatomy and hence better advise a patient about his or her prognosis or treatment options.

DETERMINING TREATMENT

The choice of treatment depends on both the need to relieve symptoms and the need to identify those at increased risk of death. For example, in a large study of patients with chronic stable angina who were undergoing treatment in Veterans Administration facilities, it was shown that 35 percent of those who had obstructions of the left main coronary artery died within four years with medical treatment alone. This compared with a four-year death rate of 27 percent for patients who had obstructions of three vessels, a 12 percent death rate for patients who had obstructions of two vessels, and a 2 percent death rate for patients with obstructions of one vessel. (Fortunately, obstructions of the left main coronary artery are not too common.)

It is useful in deciding treatment to determine the specific type of angina that is present. People with coronary artery disease may be affected by what seems to be either stable exertional angina or vasospastic (for example, Prinzmetal’s) angina. (See box, “Complications of Angina.”)

Complications of Angina

The medical problems that can arise from coronary artery disease are:

- *Heart rhythm disorders (arrhythmias)*, which are disturbances in the heart's electrical activity. In people with coronary artery disease, the arrhythmias are likely to have been caused by damage to certain areas of the heart muscle through lack of oxygen (ischemia) or heart attack (infarction). Electrical instability is probably the major cause of sudden death in people with coronary artery disease. When heart rhythm disorders are the major clinical manifestations of coronary artery disease, therapy focuses primarily on preventing arrhythmias using medical, electrical, or in refractory cases, surgical therapy. (See Chapter 16.)
- *Unstable angina or angina which becomes progressively more severe regardless of treatment.* A person with this kind of angina may find that the frequency and severity of chest pain increases, and attacks may occur during rest or may be provoked by less effort than usual. This type of angina may also occur in people who previously have not had angina; the attacks increase in frequency and severity and may occur during rest or be precipitated by less and less physical activity each time. This angina is not well controlled by medication.
- *Angina that cannot be controlled.* This unstable angina often serves as a forewarning of impending heart attack. People who suffer from unstable angina should be hospitalized—preferably in a coronary intensive care unit—and treated with bed rest and medication. Aspirin and the drug heparin, a blood thinner administered intravenously, have been shown to reduce the incidence of heart attacks in people with unstable angina. Coronary angiography (chest X-ray of dye-filled blood vessels) should be considered to determine the extent and location of any narrowing of the coronary arteries and to help decide whether angioplasty or bypass surgery should be performed.
- *Heart attack.*
- *Sudden death.*

Stable exertional angina is caused by an imbalance between the coronary blood supply and demand resulting from a fixed or stable obstruction of one or more of the coronary arteries. Oxygen deprivation usually occurs at about the same point during exertion, and people can generally predict the factors that provoke an attack. Pain can usually be alleviated with medication and/or by stopping the activity that pre-

cipitated the attack. In addition, medication generally helps to reduce the frequency of the attacks or, often, eliminates them by decreasing the heart's blood (oxygen) requirements or by increasing blood (oxygen) supply.

People with vasospastic angina, which is caused by arterial spasms, may often have a fixed blood vessel narrowing, but of a kind or degree in which constriction of the blood vessel also plays an important role in the onset of oxygen deprivation. Angina is less predictable in these people. They may experience days when there is little or no chest pain regardless of the amount of physical activity or days when angina is sparked by even slight exertion. In fact, angina frequently occurs even when the person is resting or asleep. Vasospastic angina usually responds to medications that alleviate or prevent vessel spasms.

DRUG THERAPY

A variety of medications are used to treat angina. (See Chapter 23.) These medications work either by reducing the oxygen demand of the heart, by helping increase the supply of blood, or by doing both. Often, two or more medications will be prescribed together because they can complement each other's actions and may reduce the necessary dose of any one drug, thus minimizing side effects.

The oldest and most frequently used coronary artery medications are the nitrates. Nitrates dilate veins, causing blood to pool in the veins and thus reducing the amount of blood returning to the heart. This has the effect of decreasing the size of the left ventricle, reducing the work of the heart (lowering heart muscle demand for oxygen), and lowering the blood pressure. Nitrates may also increase the supply of oxygenated blood by causing the coronary arteries to open more fully, thus improving blood flow. Nitrates also relieve coronary artery spasm. They do not, however, appear to decrease the strength of the heart's contraction. Nitrates are available in the form of nitroglycerin tablets, long-acting tablets, topical ointments, and time-release medicated patches that attach to the skin.

During an attack of angina, nitroglycerin tablets are taken under the tongue (sublingually), where the medication is quickly absorbed into the bloodstream. The medication begins to work within five minutes, and its beneficial effects last from 10 to 30 minutes.

Because of its rapid effect and short duration of action, sublingual nitroglycerin is generally used for relief of individual angina episodes rather than for sustained treatment.

Isosorbide dinitrate is a long-acting nitrate. It takes 3 to 15 minutes to take effect, and its benefits last from one to two hours when it is taken sublingually. The benefits of oral forms of isosorbide dinitrate last from four to six hours, depending on the size of the dose; there is also a longer-acting sustained-release form.

Transdermal nitroglycerin disks are patches worn on the skin; the nitroglycerin is absorbed into the bloodstream to provide continuous delivery of medication for up to 24 hours. However, it is generally recommended that the patch be removed during some part of each day to prevent the buildup of tolerance to the drug's effects.

BETA BLOCKERS

Beta blockers were first introduced in the early 1970s and have become one of the most useful types of drugs to treat coronary artery disease (and effort-induced angina in particular). Beta blockers work by blocking or inhibiting certain receptors in the heart.

During exercise or emotional stress, adrenalinelike products are released and normally stimulate these receptors (beta-adrenergic receptors) to transmit messages to the heart to speed up and pump harder. By blocking these beta receptors and reducing the heart's workload (lowering heart rate and strength of contraction), beta blockers effectively reduce the demand of the heart muscle for oxygen during physical activity or excitement. This helps prevent oxygen deprivation to areas of the heart muscle. Beta blockers also help to lower blood pressure, which further reduces the work of the heart.

The drugs may be used alone or in combination with others that relieve angina; the effects of beta blockers are particularly complemented by nitrate therapy. Beta blockers currently on the market include propranolol (Inderal), nadolol (Corgard), timolol (Blocadren), pindolol (Visken), betaxolol (Kerlone), metoprolol (Lopressor), atenolol (Tenormin), acebutolol (Sectral), and penbutolol (Levatol).

CALCIUM CHANNEL BLOCKERS

Calcium plays an important role in the contraction of the smooth muscle cells of both the heart and the arteries. Calcium blockers or antagonists work by

blocking the channels through which calcium would normally enter these cells. By helping to block smooth muscle contraction which causes arteries to narrow, the medication helps keep the vessels dilated, thereby improving blood flow.

The calcium antagonists commonly used in the United States are nifedipine (Procardia), nicardipine (Cardene), verapamil (Calan, Isoptin), and diltiazem (Cardizem). Although their clinical effects and chemical structures are different, they all work by reducing the ability of calcium to enter heart muscle and vascular smooth muscle cells. As a result, they are effective in treating coronary artery spasm and increasing blood flow by dilating the arteries. The heart's workload is also decreased because the drugs lower blood pressure and decrease the strength of the heart's contractions.

Calcium channel blockers may often be prescribed as an addition to a regimen consisting of a beta blocker and a nitrate, particularly for people whose anginal discomfort has persisted despite the use of the latter medications. Verapamil and diltiazem have also been used to treat heart rhythm disorders. Caution in the use of these drugs is necessary in people with any significant degree of heart block (abnormality of the heart's normal rhythm electrical conductive system) or poor left ventricle function.

COMBINATION DRUG TREATMENT

Effective treatment for people with severe angina often involves using a combination of drugs, most often a nitrate, a beta blocker, and a calcium channel blocker. For people with less severe angina, there are several options. Broadly speaking, beta blockers are often the treatment of choice for angina that is usually brought on by an increase in heart work or oxygen demand. For people with angina in which vessel spasms are likely to play a significant role, calcium channel blockers may be the drugs of choice. Nitrates are generally used in conjunction with either drug.

Aspirin therapy is also being frequently recommended for people with coronary artery disease. Aspirin has an antiplatelet effect that reduces the risk of clot formation in a coronary artery. Platelets are a type of blood cell. They are instrumental in clot formation that can occur at the site of a plaque and further decrease blood flow through a coronary artery, often resulting in a heart attack. Aspirin has been shown to be beneficial after a heart attack and for reducing the risk of a heart attack in people who suffer from unstable angina and possibly also in people who suffer from stable angina.

ANGIOPLASTY AND SURGERY

Two options for interventional or surgical treatment of angina are currently available and widely used: balloon angioplasty (also called percutaneous transluminal coronary angioplasty or PTCA) and coronary artery bypass surgery.

Angioplasty involves inserting a thin tube (catheter) with a deflated balloon on its tip through an incision into a blood vessel in the groin or the arm and threading it through the major arteries until it reaches the coronary arteries. The catheter is then positioned so that the balloon rests within the blockage, at which point the physician inflates the balloon, thereby flattening and cracking the plaque or other obstruction against the vessel wall and also stretching the vessel open with the pressure. When the balloon is deflated and removed, the blocked vessel remains less obstructed.

This procedure was first performed in humans in 1977. More than 200,000 Americans now undergo it each year, with an overall initial success rate approaching 90 percent, according to the National Heart, Lung, and Blood Institute's registry figures for 1985 and 1986. Approximately 30 percent of patients undergoing angioplasty experience a recurrence of artery narrowing (restenosis) within six months, although they may benefit from repeat angioplasty. The mortality rate is around 1 percent, with a 4 percent chance of complications that might require emergency coronary artery bypass surgery.

Care in selection of patients is important to the success of angioplasty. Ideally, the patient should have only one or two vessels obstructed (although multivessel angioplasty is increasingly being used), and the obstructions should be in sections of the artery that can be reached easily by the catheter. Improvements in technique, along with new developments in the field, make angioplasty an increasingly effective method of treating coronary artery disease. However, angioplasty is not initially successful in some cases. Studies are under way to compare angioplasty to medical therapy in patients with predominantly single vessel disease and to surgery in patients with multiple vessel disease. (See Chapter 24 for further details.)

Coronary artery bypass surgery is one of the most common and successful major surgeries performed today. Although this operation was first performed as recently as 1967, about 320,000 bypass procedures are now done in the United States each year. The

principal behind the coronary bypass operation is to provide new conduits to bypass obstructed or narrowed sections of the coronary arteries. These new conduits can be fashioned from lengths of a vein removed from the leg (the saphenous vein) or from an artery of the chest wall (the internal mammary artery). This procedure takes place in an operating room, with the patient deeply anesthetized. His or her heart is stopped, and blood is circulated through the body by a pump outside of the body (a heart-lung machine).

Coronary artery bypass surgery has a relatively low mortality rate (1 to 2 percent in people with good heart muscle function), although there is also a 5 to 10 percent risk of a heart attack during or immediately after the operation. Coronary artery bypass surgery provides complete relief from anginal pain in about 70 percent of people and partial relief in another 20 percent. The clearest indication for bypass surgery, therefore, is for patients who continue to have incapacitating angina despite being on a good medical program.

There is evidence that coronary artery bypass surgery improves the longevity of certain people— notably, those with blockages of the left main coronary artery branch and those with disease in all three coronary arteries and impaired function of the left ventricle. There is little proof that surgery improves the survival rate of people with narrowing in one or two arteries alone, but a major study showed that quality of life (symptoms) improved in patients in all categories. Because of improvements in operative technique, coronary artery bypass surgery is now an option for elderly people, as well as for people suffering from other diseases—such as diabetes mellitus—in conjunction with coronary artery disease. It is, however, an expensive operation with a significant recovery period. (See Chapter 25 for a more detailed discussion.)

LIFESTYLE MODIFICATION

Life-style changes are an important part of any treatment regimen for angina. Some changes may be useful in reducing the frequency of attacks by identifying and modifying the activities and situations that precipitate these attacks. Changes may be relatively minor, such as avoiding exertion after a heavy meal or using a golf cart instead of walking. Emotional upset should be avoided as much as possible, and air con-

ditioning maybe considered a necessity for patients with coronary artery disease who live in hot, humid climates.

Long-term changes that reduce known cardiovascular risk factors are also helpful, because they can help not only to prevent further damage to the arteries but also, in some instances, actually to reverse the damage.

For smokers, smoking cessation is the first and most efficacious life-style modification that can be undertaken. Studies have shown that if a person quits smoking altogether, the risk of a heart attack returns within 3 to 5 years to a level similar to that of non-smokers in the same age group. (See Chapter 6.)

Changes in diet are also a vital part of reducing the continued development and progression of atherosclerosis. Reducing the total calories and the intake of saturated fats and dietary cholesterol while increasing the intake of starches and high-fiber food may significantly lower blood cholesterol levels. (See Chapter 5.)

Regular aerobic exercise has many possible beneficial effects: controlling weight, lowering blood cholesterol, improving cardiovascular tone, reducing stress, and providing a general feeling of well-being. The conditioning effect of exercise also increases a person's ability to perform a greater amount of work with the use of less oxygen. (See Chapter 7.)

Exercise need not be strenuous, but it is important that it be energetic enough to gradually raise the heart rate and that it be performed regularly (a minimum of three to five days a week). Brisk walking for 30 to 45 minutes is inexpensive, requires no skill, and puts little burden on knees, back, or hips. Exercises that produce a sudden strain—such as lifting heavy weights—should be avoided or conducted under a physician's guidance. They have relatively little cardiovascular benefit, and the sudden increase of blood pressure that such activities produce may precipitate an attack of angina.

Reducing stress can be a valuable adjunct to any life-style modification. While stress has been only tenuously linked to high blood pressure and coronary artery disease, reduction of stress can benefit the body as a whole. It is important to realize that stress does not arise just from having a lot to do; rather, it comes from feelings of being overwhelmed and unable to cope, from feelings of hostility and from an inability to relax or enjoy leisure time. Regularly setting aside time to pursue an enjoyable activity (such as listening to music), meditation, and in some cases, psychological counseling, can all help reduce stress. (See Chapter 8.)

HEART ATTACKS

A heart attack, known medically as *acute* myocardial infarction or an acute MI, is a major and all too common medical emergency. Each year, there are about 1.5 million heart attacks in the United States, leading to more than 500,000 deaths. Most of these deaths—more than 300,000—are sudden, occurring before the patient even reaches the hospital.

The vast majority of heart attacks are a direct result of coronary artery disease. A blood clot or muscular spasm in a narrowed coronary vessel may suddenly block it completely, triggering an infarction in the area of the heart muscle that is normally nourished by that artery. (The myocardium is the muscular wall of the heart. Infarction is a term to describe the death of some of this vital tissue because it has been deprived of blood and oxygen.)

A myocardial infarction can be dangerous because irreparable heart damage may develop within a short time after the muscle is deprived of oxygen. An infarction that affects as little as 10 percent of the myocardium can cause death if it involves a critical area such as the papillary muscle (the muscle supporting the heart valve) or if it precipitates an irregular rhythm or perforation of the heart wall. Still, heart attack patients often survive much larger infarctions, affecting up to 30–40 percent of the myocardium, if a less critical area is involved.

The severity of the heart attack depends on several factors, including:

- *The site of the coronary artery that is blocked.* Blockages of the left main and the left anterior descending arteries are usually more life-threatening than blockage of the right coronary artery.
- *Cardiac arrhythmias.* Blockage of a coronary artery can cause a serious heartbeat irregularity (arrhythmia) that may result in sudden death. For example, blockage may cause a malfunction of the heart's electrical impulse system, leading to an inefficiently rapid beat (tachycardia) or an ineffective fluttering of heart muscle (ventricular fibrillation). Ventricular fibrillation is fatal unless blood flow is restored with cardiopulmonary resuscitation and the normal heartbeat restored with drugs or electric shock therapy (defibrillation). Serious arrhythmias may also arise later, after the acute phase of a heart at-

tack, if certain areas of the ventricular wall have been damaged. (See Chapter 16.)

- **Collateral circulation.** When a key coronary vessel slowly becomes blocked over a period of months or years, the heart muscle's demand for oxygen prompts other vessels and their branches to widen and even extend into the oxygen-deprived area to provide an alternative blood source. In effect, a gradual natural coronary bypass takes place. This is referred to as "collateral" coronary blood flow, which can be a saving grace if the original vessel becomes totally occluded. Collaterals are credited with saving many older heart attack patients. The sudden, fatal heart attacks that sometimes strike younger men or women may be more serious because the blockage occurs in a vessel serving an area for which collaterals have yet to develop.

WARNING SIGNS AND SYMPTOMS

Heart attacks vary in severity and in symptoms. The one clear rule is that whenever heart attack is suspected, the person must be taken to a hospital as quickly as possible. About 60 percent of all heart attack deaths occur within the first hour. Yet, according to the American Heart Association, at least half of people suffering a heart attack delay seeking help for two or more hours.

The initial pain of a heart attack is often intense—a crushing feeling or pressure in the middle of the chest. But in other cases it is much less severe; the pain may be no more than an unusual dull, aching sensation that persists. Or there may be a strong squeezing sensation inside the center of the chest. Some people experience burning feelings, while in some cases, they simply feel bloated. Sometimes, there is virtually no pain. (These are referred to as silent heart attacks.)

When pain occurs, it most often is focused beneath the sternum (breastbone). Or it may spread out, encompassing all of the chest, the shoulders and arms (the left arm more often than the right), and even the neck and the jaws. For some people, this chest pain seems very much like, albeit more severe than, the angina pectoris that they had previously experienced. In unusual instances, there is little or no pain, although there may be other symptoms. If an anginal

episode lasts for more than 10 or 15 minutes and it is not relieved by up to three nitroglycerin tablets (given every few minutes), it is a sign that a heart attack may be occurring.

Cold sweats are common just before or during a heart attack. The person may be dizzy or weak or may feel faint; loss of consciousness can also occur. The pulse may be rapid and shallow or irregular. Nausea, vomiting, and other gastrointestinal symptoms are common. A person having a heart attack also may be short of breath. He or she maybe weak, pale, and extremely anxious. (For information on how to help victims of a heart attack and other cardiac emergencies, see box "What You Can Do" and Chapter 27.)

Prompt emergency care not only saves many lives but it also helps minimize the damage of a heart attack. Many ambulances and other emergency vehicles are now equipped with life-saving equipment. In fact, many are actually mobile coronary care units, and the emergency medical teams are trained in administering life-saving treatment even before the patient reaches the emergency room. In most communities, emergency medical service (EMS) workers, ambulance drivers, firemen, and others are now trained to stabilize heart attack patients before and while transporting them to the emergency room. (See Chapter 27.)

Even in the face of marked symptoms of a heart attack, there is a natural tendency to wait and see if the pain or discomfort in the chest is from heartburn or some other harmless ailment. But, if it is a heart attack, irreversible damage may occur within hours if not minutes. Perhaps more important, some of the most potent new drugs that can prevent death of heart muscle work only if they are given within the first four to six hours of the heart attack. Thereafter, they may be less beneficial.

Preferably, treatment should be sought at a hospital with a 24-hour-a-day emergency room that is continuously staffed by doctors. If the choice presents itself, one should go to a hospital with an intensive care unit (ICU) or, preferably, one with a specialized ICU called a coronary care unit (CCU).

IN THE EMERGENCY DEPARTMENT

When you accompany a person with a suspected heart attack to the emergency department, ask immediately for a doctor or a nurse and clearly an-

What You Can Do

These are the American Heart Association's instructions for use with a possible heart attack victim:

Know the warning signals of a heart attack

- Uncomfortable pressure, fullness, squeezing or pain in the center of the chest lasting two minutes or longer.
- Pain spreading to the shoulders, neck, or arms.
- Severe pain, lightheadedness, fainting, sweating, nausea, or shortness of breath.
- Not all these warning signs occur in every heart attack. If some start to occur, however, don't wait. Get help immediately. Delay can be deadly.

Know what to do in an emergency

- Find out which area hospitals have 24-hour emergency cardiac care.
- Determine (in advance) the hospital or medical facility nearest home and office, and tell family and friends to call this facility in an emergency.

- Keep a list of emergency rescue service numbers next to the telephone and in a pocket, wallet, or purse.
- If any chest discomfort lasts 15 minutes or more, call the emergency rescue service.
- If getting to the hospital is faster by car, do not wait for an ambulance.

Be a heart saver

- If someone is experiencing the signs of a heart attack—and the warning signs last two minutes or longer—act immediately.
- Expect a “denial.” It's normal for someone with chest discomfort to deny the possibility of something as serious as a heart attack. But don't take “no” for an answer. Insist on taking prompt action.
- Call the emergency service, or
- Get to the nearest hospital emergency room that offers 24-hour emergency cardiac care.

nounce that a heart attack patient has arrived. After an examination, the nurses and doctors can determine whether it really is a heart attack or some other perhaps less serious problem.

Emergency department nurses and physicians often can diagnose a typical heart attack by looking at the patient. (They may see several heart attack victims each day.) Even so, looks can be deceptive, and a diagnosis must be confirmed by talking to and examining the patient and by taking an electrocardiogram (ECG) and administering a series of blood tests.

The heart may be beating too rapidly (tachycardia) or too slowly (bradycardia). The blood pressure, too, may be elevated, or more commonly it may be on the low end of normal. The ECG typically shows irregularities, particularly changes in the Q waves, ST segments, and/or T waves. (See Figure 11.1.) Doctors often can deduce from the ECG which coronary vessel is afflicted.

A blood specimen should be drawn quickly and then tested for the presence of enzymes that are secreted by heart muscle cells that may have been injured, a strong indication of muscle damage or death (infarction). Treatment starts immediately, particularly if the patient's heart has stopped or he or she is unconscious.

A defibrillator will be deployed at once if the heart is fibrillating. A jolt of electricity is passed through the heart, between paddle-shaped electrodes held against the chest. This electric shock often interrupts arrhythmias and restores the heart to normal (sinus) rhythm. If the heart has stopped, doctors will compress the chest, up and down, trying to maintain the heart's pumping action. (See Chapter 27.)

Over the past decade, the most recent innovation in the treatment of heart attack patients has been the use of clot-dissolving or thrombolytic agents, a technique called reperfusion therapy. Most heart attacks result from the formation of a blood clot within a coronary artery that is narrowed by atherosclerosis or spasm, and it is possible to restore blood flow to the heart by dissolving this clot (thrombus).

Because the lack of blood and oxygen causes progressive death of myocardial cells, it is important to administer the thrombolytic agent as soon as possible. In more than two-thirds of cases, if the thrombolytic agent is administered within 6 hours of the onset of the heart attack, the blood clots can be dissolved and the blood flow restored, thereby salvaging heart muscle. Studies have clearly shown that early administration of thrombolytic agents results in better survival and better heart function following myocardial infarction.

The earliest thrombolytic agents were streptokinase (Kabikinase, Streptase) and urokinase (Abbokinase). More recently, t-PA (tissue plasminogen activator) and anistreplase (Eminase) have been introduced for clinical use in the United States. TPA (alteplase or Activase) is a genetically engineered agent that contains a natural human substance that activates an enzyme that dissolves the blood clot. Although the agent is most effective when given early, in selected patients, the myocardium can be salvaged when the thrombolytic agent is given later. These drugs have revolutionized the care of heart attack patients and have reduced the death rates by about 20 percent.

In addition to thrombolytic agents, aspirin and heparin may also be administered. The drug heparin is given intravenously and interferes with normal blood clotting. These drugs can prevent a clot in the artery from growing larger or from reforming after it has been dissolved by the thrombolytic agent.

Additional drugs are often administered early in the course of a heart attack. Morphine may be injected or infused through an intravenous line to relieve pain. This old, powerful narcotic agent is extremely effective and is still the standard for pain relief. Oxygen may be given through a face mask or nasal prongs to improve the oxygen content of the blood still flowing to the heart. Intravenous beta blockers and nitroglycerin may also be administered in an attempt to limit the size of the heart attack. Sometimes drugs used to treat or prevent irregular heart rhythms will also be given. (See Chapter 23 for more information on cardiovascular drugs.)

The aim of all of these therapies is to restore blood flow, restore a regular heartbeat, and then give the damaged heart time to recover. For some people, however, these medical treatment methods are not effective and the blocked artery may fail to open. In some cases, further treatment, such as angioplasty or coronary bypass surgery, may be necessary.

An angiogram, an X-ray showing dye-filled blood vessels, can indicate where the blockage is located, and balloon angioplasty may be used to open the obstruction. In some patients who have extensive blockage—sometimes in two or more vessels—an emergency coronary artery bypass operation may be performed. Coronary artery bypass surgery can be performed on an emergency basis, is relatively safe, and, in most cases, is quite effective.

A variety of other procedures may be deployed in the CCU to assist heart attack victims, especially those who show signs of heart failure. In one, a balloonlike device (an intra-aortic balloon catheter) is inserted

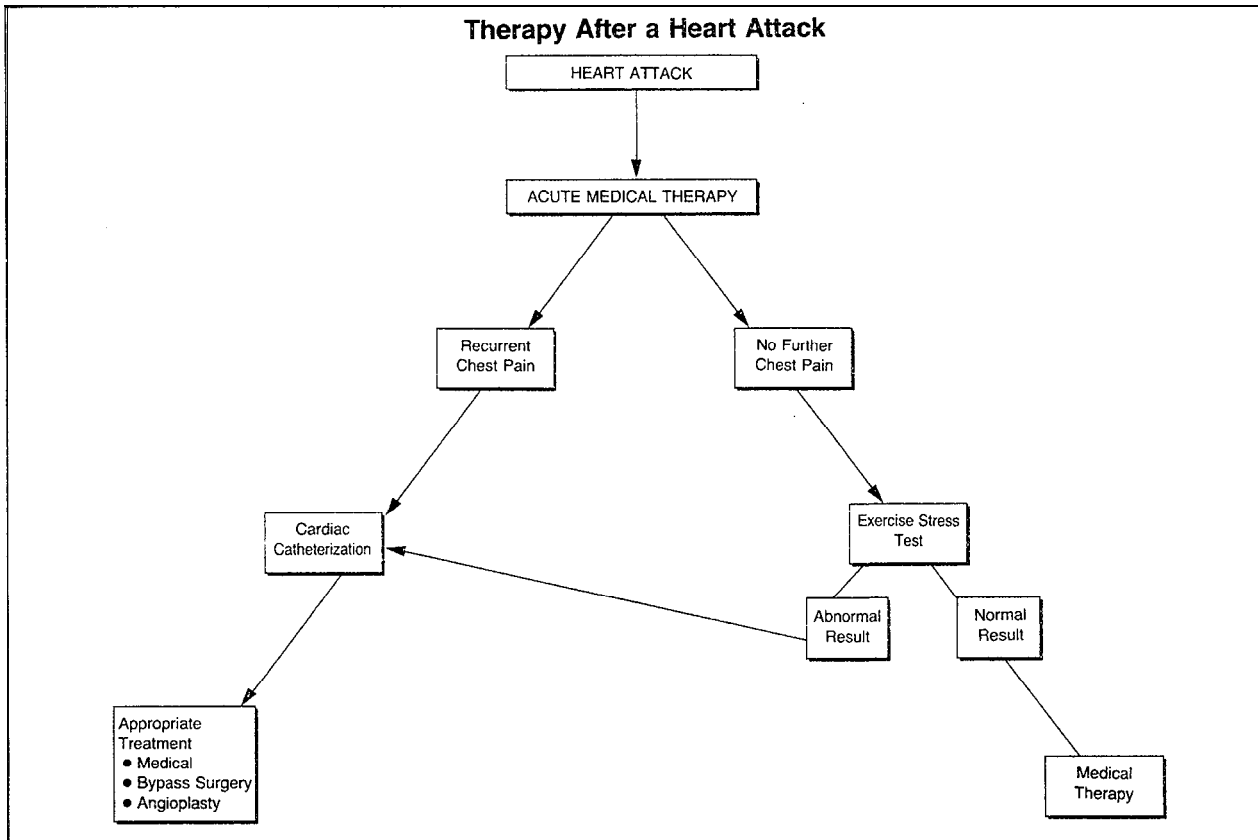
inside the aorta, the body's largest artery which rises from the heart. The balloon can be inflated rhythmically from outside the body. This forces blood into the aorta and forward through the circulatory system, thereby assisting the weakened heart. In essence, the balloon is an auxiliary pump that temporarily carries some of the load when the heart is weakened by a heart attack.

HOW CORONARY CARE UNITS HELP

Much of the recent improvement in heart attack treatment comes from specific new developments—better ways to detect heart damage and improved drugs or other treatments. Still, a major factor in decreased heart attack mortality is the development of total, integrated care, with an emphasis on monitoring heart function, that is provided through coronary care units (CCUs). These are areas in the hospital reserved for heart patients and are staffed by specially trained doctors, nurses, and technicians. CCU workers can follow a patient's status in minute detail, using sophisticated computerized electrocardiograms (ECGs) and other monitoring methods. The patient is attached on an ongoing basis to an ECG monitor that sounds an alarm when an irregular heartbeat develops. Defibrillator and other life-saving equipment are on hand and can be used within minutes if a problem suddenly arises.

When the patient's heart and other vital organs are again functioning in a stable way, usually within a few days, he or she maybe moved out of the CCU into an ordinary hospital room. The hospital stay for a heart attack can vary from one to three weeks, depending upon the severity and extent of heart damage and the occurrence of any complications. The heart begins to heal during the first several weeks after a heart attack by forming scar tissue to replace the damaged or dead heart cells. Although scar tissue strengthens the injured part of the heart muscle, it cannot contract like normal heart muscle. Therefore, the remaining heart muscle must compensate by working or contracting harder to pump blood. In addition to scar formation, collateral blood vessels (see above) may develop to bring more blood to the surrounding damaged but living heart cells in the border regions of the myocardial infarction.

The presence and extent of the heart attack can be definitively diagnosed by serial electrocardiograms,



blood tests (that measure enzymes released from dying heart cells), and possibly by an echocardiogram or radionuclide scan to image how the various regions of the heart muscle (for example, left ventricle) are pumping. A severe or large heart attack can be complicated by: rupture of the heart wall; low blood pressure; fluid buildup (congestion) in the lungs due to inability of the heart to pump adequate amounts of blood; blood clots in the heart or legs; irregular heart rhythms such as tachycardia or fibrillation; and recurrent chest pain, either due to an inflamed heart sac (pericarditis) or recurrent angina.

In this era of thrombolytic therapy in which it is frequently possible to successfully reopen a previously occluded artery, it is important to evaluate patients for their risk for another heart attack. (See box, "Therapy After a Heart Attack.") A successfully opened stenotic artery can reocclude with new clots in up to one-fourth of patients within days or months following the initial heart attack. Therefore, patients frequently have an exercise stress test prior to hospital discharge to ascertain if they are at high risk for another heart attack. Recurrent angina or a positive stress test usually leads to catheterization and evaluation for coronary artery angioplasty or bypass surgery.

The total recovery from a heart attack usually takes two to three months. During this time, the patient should try initially to reduce the strain on the heart by resting, and then to improve heart function by gradually increasing activities and starting routine exercise. The exact activity prescription for a heart attack patient depends on the size and complications of the heart attack, the level of activity before the heart attack, and how the heart responds to increased activity.

Heart attack patients are often maintained on treatment with aspirin and beta blockers, which have been shown to decrease the risk of subsequent heart attacks, as well as treatment with other antianginal drugs as needed. The goal of rehabilitation is to gradually increase one's activities to the point of resuming a reasonable life-style. (See Chapter 28.)

Initial avoidance of extreme stress or exercise and extremes of hot and cold temperatures is important to minimize the risk of putting too much stress on the heart or of precipitating angina. A physician can usually guide the resumption of activities based on the severity of the heart attack and data from an exercise stress test. Most activities of daily living, including sexual activity, can usually be resumed within three to six weeks after a heart attack. It will also be im-

portant in the rehabilitation period to decrease cardiac risk factors (see Chapter 3).

Feelings of anxiety, anger, and depression are not uncommon during the acute and chronic phases of a heart attack. It is important, however, to realize that most patients recover well from this life-threatening event.

THE PAYOFF: SALVAGED LIVES

The death rate from acute heart attack has dropped significantly over the last two or three decades. In

part, this is because there are about 25 percent fewer heart attacks. There has also been a similar 25 percent reduction in fatalities from heart attacks. A significant part of this reduction in deaths is due to newer treatments, careful observation and management of patients in CCUs, and improvement in other facets of heart attack care.

Heart attack survivors require shorter hospitalizations than before. They also tend to be healthier—less disabled—after their heart attacks because of improved treatment methods. Skillful coronary care limits the damage and disability. Most heart attack patients now can regain normal or near-normal lifestyles, and some actually enjoy better health than before their heart attacks.