2

Preventing Cardiovascular Disease

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Goals for Behavior Change

- Choose three high-fat foods that you regularly eat and replace them with low-fat healthier foods.
- Find out your total cholesterol, LDL cholesterol, and HDL cholesterol. Select three or four lifestyle behaviors to change to improve your cholesterol profile.
- Find out your blood pressure. Select two or three behaviors to change (excluding taking medication) to lower it.
- If you use tobacco products, devise a plan for quitting the tobacco habit.
- If you are not currently active, make a list of physical activities to improve your health status and level of fitness. Determine how many calories you would attempt to expend per week.
- Plan and implement three strategies to help you deal with chronic stress.

Key Terms

aneurysm
atherosclerosis
cerebral hemorrhage
cholesterol
embolus
hypertension
ischemia
lipoprotein (a)
myocardial infarction
sedentary
thrombus

Objectives

After completing this chapter, you will be able to do the following:

- Describe the gross anatomy and function of the heart.
- Trace the development of cardiovascular disease during the twentieth century in the United States.
- Identify and differentiate among several types of cardiovascular disease.
- Identify the risk factors for coronary heart disease and discuss ways to reduce them.
- Explain the lifestyle behaviors that contribute to health and longevity.

Cardiovascular disease encompasses a group of diseases that affect the heart and blood vessels. Cardiovascular disease—the leading cause of death in the United States—accounts for 41.4 percent of deaths.[1] About 25 percent of Americans (approximately 59 million people) have one or more forms of heart or blood vessel disease. Approximately 1.5 million heart attacks have occurred every year in the last few years, and about 500,000 of these end in death each year. At least 250,000 heart attack victims die within the first hour following the onset of symptoms. They fail to reach a hospital before death occurs. A significant number of these premature deaths could have been prevented with early recognition and treatment.

Of heart attack victims, 50 percent wait an average of two hours before seeking medical attention. Denying the possibility that a heart attack is occurring is the primary reason for the delay. The situation is complicated because the symptoms of a heart attack are similar to those of other physical ailments, and people are more prone to believe that it is one of the other problems than that it is a heart attack.

Although the figures are foreboding, substantial progress has occurred during the last forty-five years. The death rate for cardiovascular diseases has declined by more than 50 percent since 1950.[1] Although an impressive accomplishment, it is somewhat diminished by the fact that cardiovascular diseases remain by far the leading cause of death in the United States. Two types in particular, coronary heart disease and strokes, are the first and third leading causes of death. More than 50 percent of the premature deaths in our society are attributed to lifestyle habits.

Research has convincingly shown that following a heart-healthy lifestyle can significantly lower the risk for heart disease, and it has been a major contributor to the decline in heart disease mortality.[2] The medical profession, through the development and use of sophisticated diagnostic procedures and vastly improved after-the-fact treatments, have equally contributed to the downward trend in the death rate from cardiovascular disease.

Circulation

You can understand circulation better if you are familiar with the basic anatomy and function of the heart. The heart consists of cardiac muscle and weighs between 8 and 10 ounces. It is about the size of a fist and lies in the center of the chest. The heart is divided into two halves, or pumps, by a wall (the septum), and each half is subdivided into an upper chamber (the atrium) and a lower chamber (the ventricle). The right heart, or pulmonary pump, receives deoxygenated blood from the tissues and pumps it to the lungs so that carbon dioxide can be exchanged for a fresh supply of oxygen. From the lungs, the oxygen-rich blood is sent to the left heart, or systemic pump, so that the oxygenated blood can be pumped to all the tissues of the body. Both pumps work simultaneously. The systemic pump carries the heavier workload of the two and thus has a more muscular ventricular wall. Figure 2-1 illustrates the circulatory system.

The arteries carry oxygenated blood away from the heart while the veins carry deoxygenated blood to the heart. There are two exceptions, one in the arterial system and one in the venous system. First, the pulmonary artery carries *deoxygenated* blood from the right heart to the lungs to exchange carbon dioxide for a fresh supply of oxygen. Second, the pulmonary vein carries *fully oxygenated* blood from the lungs to the left heart for distribution throughout the body.

The primary function of circulation is to provide a constant supply of blood and nutrients to the cells while removing their waste products. Under ordinary circumstances, the interruption of blood flow for as little as four to six minutes can result in irreversible brain damage due to oxygen deprivation.

The average heart beats seventy to eighty times per minute at rest. Endurance athletes often have resting

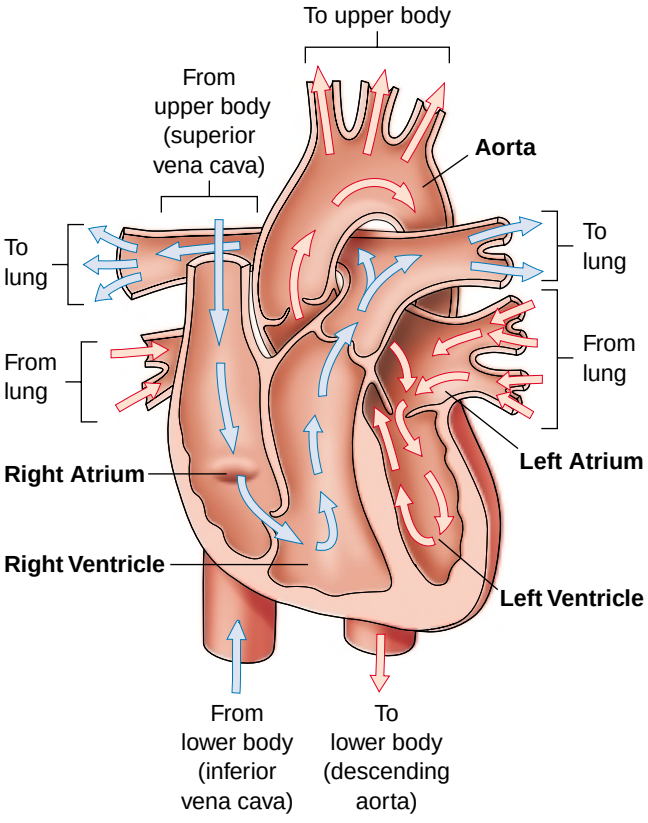


Figure 2-1 The Circulatory System

heart rates in the thirty and forty beat range, whereas some overweight and sedentary smokers have resting heart rates in the nineties. The low heart rates of endurance athletes reflect physiological adaptations to training that represent normal values for this group. The Framingham Heart Disease Study showed that a rapid resting heart rate increased the risk of death from heart attack. Mortality increased progressively with higher resting heart rates, especially among men.

The heart is self-regulating; it contains its own conduction system fully capable of establishing and maintaining the heart beat without outside neural stimulation. The heart's beating rate and rhythm are established by the sinoatrial node (SA node, or pacemaker), located in the right atrium, as shown in figure 2-2. The atria contract, forcing blood into the ventricles as the electrical impulse travels from the SA node to the atrioventricular node (AV node), located between the right atrium and right ventricle. The electrical impulse pauses for one-tenth of a second at the AV node to allow the ventricles to fill with blood and then resumes down the system and spreads throughout the ventricular walls. The ventricles contract during this time, ejecting blood from these chambers.

Blood that enters the chambers of the heart does not directly nourish the heart muscle because there are no direct circulatory routes from the heart's chambers into its muscular walls. Instead, blood must first be ejected from the heart to the aorta (the largest artery in the body) and then to the coronary arteries that supply the myocardium (heart muscle) with blood and oxygen. The majority of blood is received by the myocardium during diastole (between beats) because the blood vessels dilate during this time, increasing their capacity to accept and deliver blood.

Coronary circulation is illustrated in figure 2-3. The left coronary artery supplies a major portion of the myocardium with blood, whereas the right coronary artery serves less of it. Both vessels divide and subdivide downstream and eventually culminate in a dense network of capillaries (the smallest blood vessels in the body). Blood supply to the myocardium is so important that every muscle fiber is supplied by at least one capillary. The coronary veins return deoxygenated blood to the right atrium so that it can enter pulmonary circulation. The veins bring deoxygenated blood from all tissues back to the right atrium.

Blood plasma is a clear, yellowish fluid that carries approximately 100 chemicals. Plasma represents 55 percent of the blood content. The remaining 45 percent consists of blood solids—the erythrocytes (red blood cells), the leukocytes (white blood cells), and the blood platelets. The red blood cells are the most abundant of the blood solids, composing about 99 percent of the total. These cells carry oxygen and carbon dioxide attached to hemoglobin, an iron-rich protein pigment and a main component of red blood cells. The white blood cells are an important part of the body's defense system against invading microorganisms and other foreign substances. The blood platelets are involved in the complex processes that lead to the formation of clots for repairing damaged blood vessels.

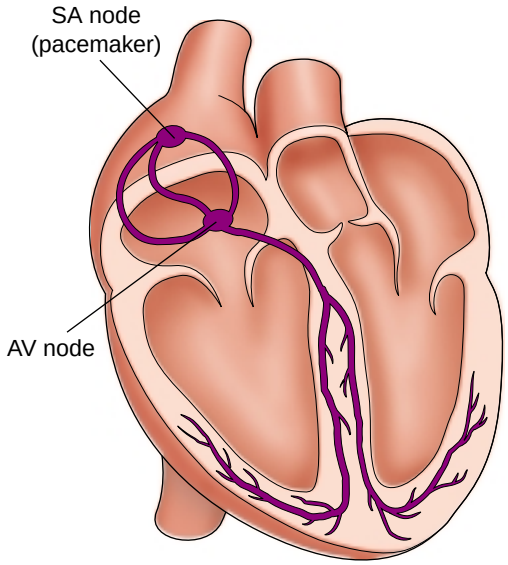


Figure 2-2 Electrical Conduction System of the Heart

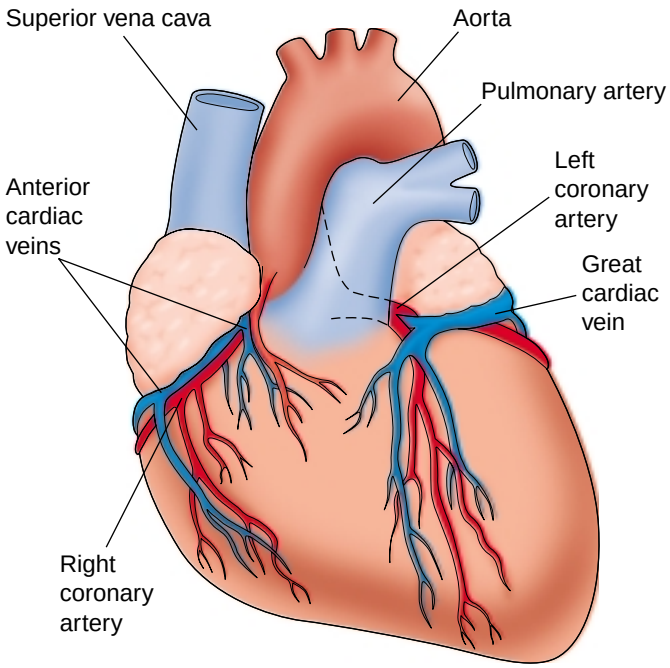


Figure 2-3 Coronary Circulation

Cardiovascular Disease: A Twentieth-Century Phenomenon

Cardiovascular disease, a relatively rare event 100 years ago, reached epidemic proportions during the middle of the twentieth century. The term *angina pectoris* (chest pain) was introduced into the medical literature by William Heberden, a British physician, in the latter part of the eighteenth century. He was unable to offer any treatment for this strange malady. It was not until 1910 that physicians made the connection between recurrent episodes of angina pectoris and heart disease. Chest pain and other manifestations of a heart attack were not identified with obstructions of the coronary arteries until the early 1900s. An American physician gave the first accurate description of the events associated with a heart attack in 1912. The illness he described, which afflicted a 55-year-old man with no previous evidence of disease, is now a common occurrence in American life. The man died three days after the onset of symptoms. A postmortem examination of the heart revealed that a clot had occluded, or blocked, one of the major coronary arteries. In 1912, this was a medical rarity.

Coronary heart disease is responsible for the majority of heart attack deaths, but other forms of heart disease contribute to disability and death. Congenital heart defects, which exist at birth, affect approximately 32,000 newborns annually. Approximately 4820 of these infants die from their defects.[1] Rheumatic heart disease, caused by a streptococcal infection of the throat or ear, is virtually 100 percent preventable. Antibiotic treatment during the infection stage arrests the processes that could lead to rheumatic heart disease. Congestive heart failure occurs when the heart muscle is so damaged that it can no longer contract with sufficient force to pump blood throughout the body. The leading causes of congestive heart failure are poorly controlled or uncontrolled long-standing hypertension or a history of previous heart attacks or both. Approximately 4.6 million Americans have congestive heart failure and about seventy-one thousand die annually of this condition.

Coronary Heart Disease

Coronary heart disease (also known as *coronary artery disease*) is a disease of the arteries that supply the heart with blood and nutrients. A diagnosis of coronary artery disease is made if any artery is narrowed by 60 percent. A heart attack, or **myocardial infarction** (death of heart muscle tissue), occurs when an obstruction or spasm disrupts or blocks blood flow to a portion of the heart muscle. The amount of heart muscle damage is determined by the location of the obstruction or spasm and the speed with which medical intervention is begun. Heart attacks of any magnitude produce irreversible injury and myocardial tissue death. It usually takes five to six weeks to form a fibrous scar around dead cardiac tissue. This area of dead tissue can no longer contribute to the pumping of blood, resulting in a less efficient heart. Massive heart attacks that cause extensive muscle damage result in death.

Although most heart attacks occur after the age of 65, the dysfunctions leading to them often begin before adolescence. These processes occur most often without symptoms and often go undetected until, without warning, a heart attack occurs. The attack is sudden, but the circumstances leading to it develop over many years. There is considerable evidence that the silent phase of coronary heart disease begins as early as childhood.[3,4]

Risk factors are genetic predispositions, lifestyle behaviors, and environmental influences that increase one's susceptibility to disease. Elevated blood pressure and blood fats (cholesterol and triglycerides) that occur during adulthood can often be traced back to childhood. See Wellness Across the Generations: Childhood Origins of Heart Disease for more information about this connection.

The ongoing Framingham Study, which began in 1949, identified the risk factors connected with heart disease.[5] Cigarette smoking, high blood pressure, elevated cholesterol levels, diabetes, obesity, stress, physical inactivity, age, gender, and family history were found to be highly related to heart attack and stroke. As the risks were discovered, the realization evolved that heart disease was not the inevitable consequence of aging or bad luck but a preventable acquired disease. After a few years, researchers realized that preventive efforts should begin in childhood.

Autopsy studies of 18-year-olds who died in accidents have shown a positive relationship between blood cholesterol levels and the prevalence of fatty streaks on the walls of the coronary arteries and aorta. The evidence indicates that the average cholesterol level in children in overfed, underexercised societies such as the United States is too high.

Autopsy studies of American combat battle casualties, whose average age was 22 years, in the Korean and Vietnam wars showed obstructions in the coronary arteries. These obstructions are caused by **atherosclerosis,** a slow, progressive disease of the arteries that can originate in childhood.[4] It is characterized by the deposition of plaque beneath the lining of the artery (figure 2-4). Plaque consists of fatty substances, cholesterol, blood platelets, fibrin, calcium, and cellular debris. The atherosclerotic process is responsible for 80 percent of the coronary heart disease deaths in the United States. The lipid oxidation theory, the current theory of the development of atherosclerosis, is explained in the section dealing with cholesterol as a risk factor.

As many as one-third of heart attacks are silent; they have no obvious signs or symptoms.[5] These patients

Wellness Across the Generations

Childhood Origins of Heart Disease

Behavior patterns established during childhood that increase the likelihood of coronary heart disease may and often do persist into adulthood. A physically inactive child is likely to become a physically inactive adult.[6] Only 50 percent of young people in the United States (ages 12 to 21 years) regularly participate in vigorous physical activity, and one-fourth report no physical activity at all.[4] Physical inactivity is a primary contributor to the unprecedented explosion of overweight and obesity among children in the United States. The percentage of overweight youngsters today has doubled since 1980, and this trend shows no signs of abating or reversing in the near future.[7] Sixty-one percent of overweight 5- to 10-year-olds have one or more risk factors for cardiovascular disease and 27 percent have two or more.[8] One of the serious consequences of early obesity has been the emergence of Type 2 diabetes—a disease with life-threatening complications that in the recent past occurred almost exclusively to middle-aged adults—which is currently occurring among American adolescents. The two major reasons for this trend are: (1) overweight/obesity and (2) physical inactivity.[7] The American Diabetic Association and the American Academy of Pediatrics recommend that at-risk children should be screened with a blood test every two years starting at the age of 10.[9] An estimated 6 million teenagers and 100,000 youngsters under the age of 13 are regular smokers.[11] Smoking rates among high school students rose from 27.5 percent in 1991 to 34.8 percent in 1995.[12] These are major risks that begin in childhood and continue into adulthood. More than 3000 children and teenagers take up the smoking habit.[4]

Leading health authorities presented a list of ten high priority public health concerns in *Healthy People 2010*. Topping the list was the need for Americans, young and old, to become more physically active. The second concern dealt with the unprecedented number of overweight and obese people in the United States.[10]

A recent study using sophisticated measuring techniques has found that children 9 to 11 years old whose cholesterol was higher than normal had blood vessels stiffer than expected for their young age. Arterial dilation was less than normal in these children while resistance to blood flow was greater than normal in response to the heart's contractions.[13]

Another recent study found that young males with elevated levels of blood cholesterol "have a much greater risk of dying from heart disease and an overall shorter life expectancy than young men with normal cholesterol levels."[14]

Attempts to prevent heart disease need to begin in childhood. Knowledgeable parents can serve as role models who practice, rather than just talk about, healthy behaviors. Active parents should be the strongest influence, but they are not the only influence. Schools need to offer quality physical education programs throughout the twelve years of precollege education, and communities should offer opportunities and provide facilities for active participation in games and recreational play.

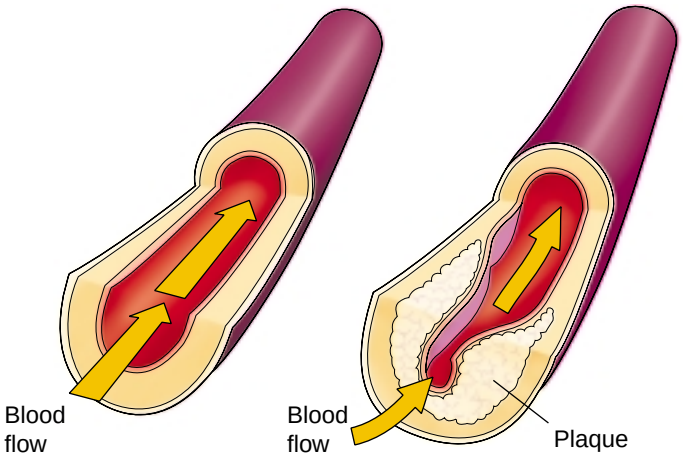


Figure 2-4 Progressive Narrowing of a Normal Coronary Artery (Atherosclerosis)

typically do not experience chest pain or chest discomfort nor do they have arm, neck, or jaw pain.[15] Those most likely to experience a silent heart attack are older people, diabetics, females, those who had a prior stroke or heart failure, and nonwhites. The absence of chest pain, a hallmark of heart attacks, can more than double the risk of dying probably because these people wait longer before seeking medical treatment. Three to four million Americans experience cardiac **ischemia** without knowing it. Ischemia (reduced blood flow) occurs as the result of arterial disease or arterial spasms that restrict blood flow to any part of the body, including the heart.[1] Silent ischemia can and does initiate heart attacks without prior warning. However, the typical heart attack is obvious, and the symptoms are pronounced. See Just the Facts: Heart Attack: The Warning Signs.

Just the Facts

Heart Attack: The Warning Signs

The Warning Signs

- Uncomfortable pressure, fullness, a squeezing sensation as if a band were being tightened around the chest, pain in the center of the chest lasting longer than 10 minutes
- Pain that spreads to the shoulders, arms, or neck
- The above warning signs accompanied by dizziness, fainting, sweating, nausea, and shortness of breath

What to Do If These Signs Appear

If you have chest discomfort lasting more than 10 minutes:

- Do not deny what may be occurring
- Call the emergency service or have a friend or family member drive you to the nearest hospital that has 24-hour emergency cardiac care
- Know in advance which hospitals have such service
- Prominently display in your home the telephone number of the emergency rescue service and also carry a copy with you

Stroke (Brain Attack)

The majority of strokes (cerebrovascular accidents, or brain attacks) follow the same sequence of events that results in coronary heart disease. A stroke is essentially the result of diseased blood vessels that supply the brain. It shares the same risk factors as coronary heart disease, and it takes years to develop.

Strokes are caused by a **thrombus** (a clot that forms and occludes an artery supplying the brain) or an **embolus** (a clot that forms elsewhere in the body and fragments, dislodges, and is transported to one of the cerebral blood vessels that is too small for its passage). **Cerebral hemorrhage** (the bursting of a blood vessel in the brain caused by trauma, arterial brittleness, or aneurysm) is also a cause of stroke. An **aneurysm** is a weak spot in an artery that forms a balloonlike pouch that can rupture. It may be a congenital defect or the result of uncontrolled or poorly controlled hypertension.

Between 70 and 80 percent of all strokes are due to a thrombus or an embolus. Brain cells once supplied by these blood vessels die and do not regenerate. As a result, the functional losses that occur (e.g., paralysis on one side of the body, difficulty speaking) cannot be fully recovered. That brain cells die also means that treatment will not be very effective. Many stroke victims are unable to return to a normal lifestyle unless the stroke was mild, in which case a full recovery is possible.

Just the Facts

Stroke: The Warning Signs

The American Heart Association suggests that people be familiar with the following warning signs:

- Temporary loss of speech or difficulty in speaking or understanding speech
- Unexplained dizziness, unsteadiness, or sudden falls
- Temporary dimness or loss of vision, particularly in one eye
- Sudden, temporary weakness or numbness of the face, arm, and leg on one side of the body
- Occurrence of a series of minor strokes, or transient ischemic attacks (TIAs)

Strokes caused by hemorrhages result in a 50 percent mortality rate. Victims die from the pressure imposed by blood leaking into the brain. Those who survive this type of stroke are likely to recover more of their normal functions than are those whose strokes were caused by a blood clot. The blood that spills in and on the brain during a hemorrhagic stroke produces pressure that gradually abates as the blood is absorbed by the body. Function is regained as the pressure relents.

On many occasions a stroke is preceded by warning signs and signals days, weeks, or months before a major stroke. These must be recognized and then acted on so that prompt medical and lifestyle interventions may be instituted to prevent or delay a stroke.

Preventing a stroke is similar to preventing coronary heart disease. Both involve blood pressure and cholesterol control, smoking cessation, weight management, exercise, and proper nutrition. See Just the Facts: Stroke: The Warning Signs, which identifies the major warning signs.

Risk Factors for Heart Disease

The major risk factors for heart disease are increasing age, male gender, heredity, and race.[1] While these risk factors cannot be changed, their impact on heart disease can be reduced by a heart-healthy lifestyle. Tobacco smoke, high blood cholesterol, high blood pressure, physical inactivity, obesity and overweight, and diabetes mellitus are major risk factors that can be modified by a heart-healthy lifestyle and medication if needed. Other contributing risk factors include an individual's response to stress, hormonal factors, birth control pills, and excessive alcohol consumption. See figure 2-5 for the major risk factors.

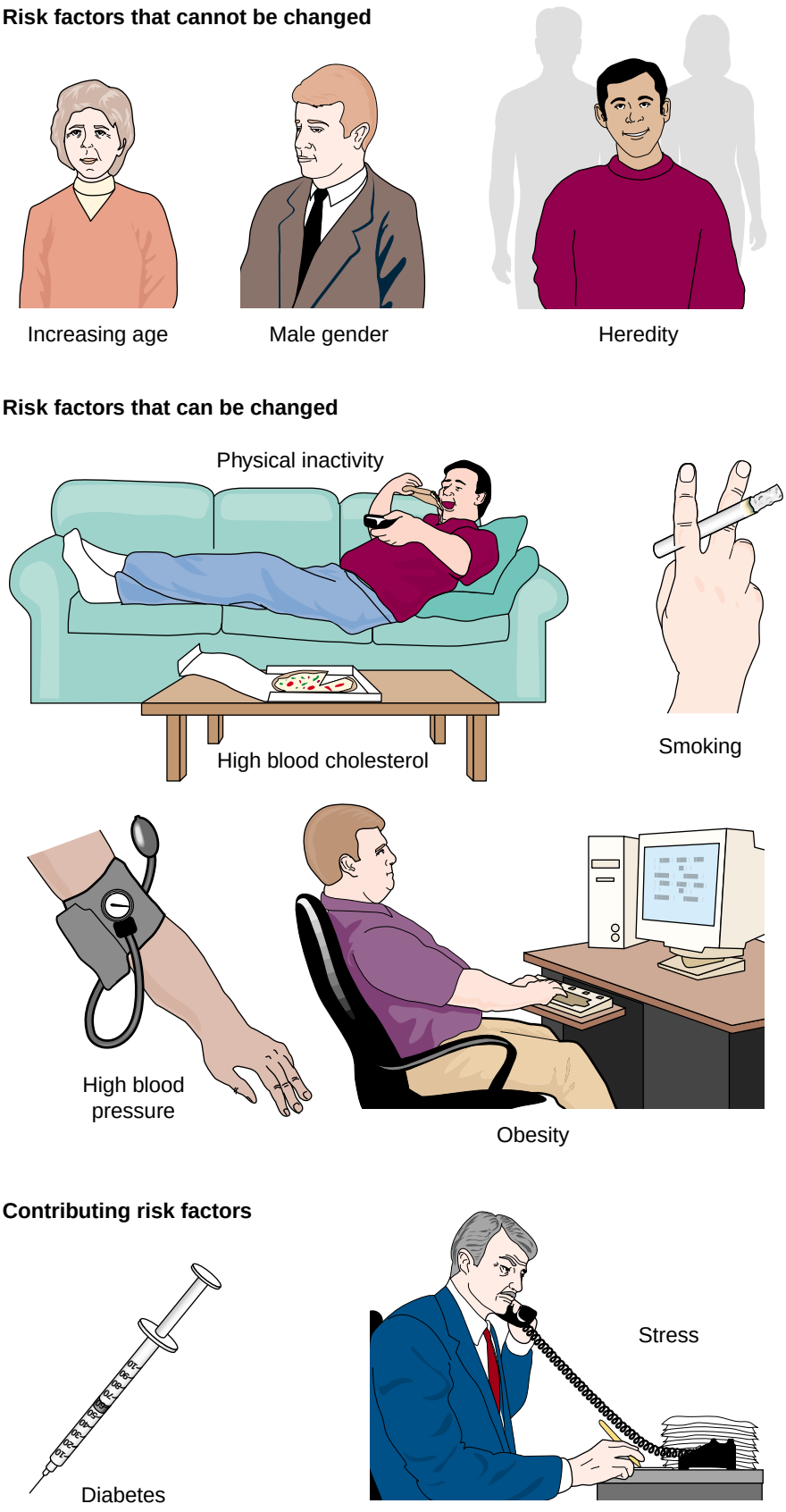


Figure 2-5 Cardiovascular Disease Risk Factors

These risk factors account for the majority of cardiovascular disease in the United States. However, there are many other factors that can be involved, some backed by a sound and growing body of evidence. These include high serum homocysteine, lipoprotein (a), high blood fibrinogen, and high blood insulin levels. Other possible risk factors, supported by inconclusive evidence, include short stature, baldness, ear lobe creases, high serum uric acid level, and others. A few of the more important of these risk factors are covered later in this chapter.

Major Risk Factors That Cannot Be Changed

Age

Approximately 55 percent of heart attacks occur in people 65 years of age or older. This age group accounts for more than 80 percent of fatal heart attacks.[1]

Male Gender

Until recently, the incidence of coronary heart disease among women was largely unexplored. Men have been the primary subjects in coronary heart disease and risk factor studies because of the high incidence of both among men. However, coronary heart disease is also the leading cause of death and disability among women, accounting for almost 250,000 deaths annually.[1] Women have less heart disease than men, particularly before menopause. See Wellness Across the Generations: Women and Coronary Disease: Pre- and Postmenopause.

An alarming trend is the increased incidence of heart attacks in premenopausal women who have been smoking cigarettes long enough for it to affect their health, especially when combined with oral contraceptive use.

Heredity and Race

According to the American Heart Association, "A tendency toward heart disease or atherosclerosis appears to be hereditary, so children of parents with cardiovascular disease are more likely to develop it themselves."[1] A history of first-degree male relatives (father, grandfather, and brothers) who have coronary heart disease or who died of coronary heart disease before the age of 55 or first-degree female relatives (mother, grandmother, and sisters) who have coronary heart disease or who died of coronary heart disease before the age of 65 indicates a strong familial tendency.[17] If the family history is positive, the modifiable risk factors must be controlled. Some minority groups have a higher rate of heart disease than would be expected. For example, African Americans have a high incidence of hypertension. One in three African-American adults is hypertensive compared to one in four adults in the general public. They also experience higher rates of morbidity (illness) and mortality (death) from consequences of hypertension such as strokes and kidney failure.[14] Other minority groups—Mexican Americans, Native Americans, Native Hawaiians, and some Asian Americans—have high rates of heart disease primarily due to rampant obesity and one of its major consequences, diabetes.[1]

Wellness Across the Generations

Women and Coronary Heart Disease: Pre- and Postmenopause

Heart attacks are relatively rare among premenopausal women because of the production and presence of estrogen (the female sex hormone responsible for the development of secondary sexual characteristics and the various phases of the menstrual cycle and essential for bone formation). Estrogen is also protective in that it lowers LDL cholesterol (the harmful form), raises HDL cholesterol (the protective form), and may increase blood flow to the heart.

During and after menopause, the production of estrogen decreases and eventually stops, and the protection from heart disease diminishes. Women tend to develop coronary heart disease about ten years later than men. By age 65 the risk for men and women equalizes.[16]

To maintain a lower risk after menopause women should consider estrogen replacement therapy (ERT), because this form of therapy can cut the risk of coronary heart disease in half. ERT is particularly beneficial for women who have had a previous heart attack or for those at high risk because of cigarette smoking, high blood pressure, high cholesterol, obesity, or physical inactivity. However, ERT may not be appropriate for all postmenopausal women because it increases the risk for breast cancer.

Women should make the decision regarding ERT replacement in consultation with their physicians who are aware of their heart disease risk and who know their family histories of breast cancer.

Although age, male gender, heredity, and race are major risks not under our direct control, we can modify their effects by living a wellness lifestyle. Abstaining from tobacco products; exercising regularly; controlling blood pressure, cholesterol, and body weight; managing stress; and maintaining a social support system can lessen the impact of these unchangeable risk factors.

Major Risk Factors That Can Be Changed

Cholesterol

Cholesterol is a steroid that is an essential structural component of neural tissue; it is used in the construction of cell walls and for the manufacture of hormones and bile (for the digestion and absorption of fats). A certain amount of cholesterol is required for good health, but high levels in the blood are associated with heart attacks and strokes.

The National Heart, Lung, and Blood Institute suggests that Americans reduce cholesterol consumption to less than 200 milligrams per day (200 mg/day), that fat intake may be increased to a maximum of 35 percent of the total calories consumed, and that saturated fat plus trans fats be reduced to no more than 6 percent of the total calories. (See Just the Facts: New Guidelines for Fat and Cholesterol.) Many authorities are convinced that limiting total fat and saturated fat is more important than being overly restrictive of cholesterol.

Data from 1987 to 1992 indicate that total fat consumption in the United States decreased by 6 percent, whereas saturated fat intake decreased by 11 percent. This parallels the decline in consumption of animal flesh and animal products.[18] Although this is a small step in the right direction, the dietary intake of saturated fat remains too high, at 12 percent of total calories.[19] Many authorities recommend that saturated fat consumption make up no more than 8 percent of total calories. In addition, the newest dietary fat recommendations emphasize monounsaturated fats (heart-healthy fats) to approximately 12 percent of total fat intake accompanied by an increase in omega-3 fatty acids from fish. Omega-3 fats are the most polyunsaturated of the fats, and they are also heart-healthy. These two fats lower total cholesterol and low density lipoproteins.[20]

Americans have made substantial progress in reducing cholesterol consumption. The average cholesterol consumed by men and women respectively in 1960 was 704 mg/day and 493 mg/day.[21] By 1994, cholesterol intake had dropped to 376 mg/day for men and 259 mg/day for women.

Cholesterol is consumed in the diet (exogenous, or dietary cholesterol), but it is also manufactured by the body from saturated fats (endogenous). Cholesterol synthesis in the body would occur even if a cholesterol-free diet were consumed. The liver manufacturers about 70 percent of endogenous cholesterol with the remainder being synthesized in the intestinal and arterial walls. The total amount synthesized varies between 500 and 2000 mg/day.[22]

If you add the typical dietary intake of 259 to 376 mg/day to this total, you can see that the body must process, use, or remove considerable amounts of cholesterol to prevent it from accumulating and clogging the arteries. The liver alone produces enough cholesterol to meet the body's needs. Therefore, consuming cholesterol is not necessary to maintain health. Table 2-1 lists some common foods that contain cholesterol and saturated fat.

Just the Facts

New Guidelines for Fat and Cholesterol

The National Cholesterol Education Program through the auspices of the National Heart, Lung, and Blood Institute released its new recommendations for detecting and lowering high cholesterol in adults. This revision, which occurred in May, 2001, represents the first major change since 1993. Table 2-2, later in this chapter, shows the new values and categories of risk. Some selected highlights recommended by a panel of twenty-seven experts appear here.

1. Physicians are strongly encouraged to pay more attention to the "metabolic syndrome," representative of a significant risk for heart disease. This syndrome is a combination of excessive abdominal fat, high blood pressure, high blood glucose, elevated triglycerides, and low HDL cholesterol.
2. HDL cholesterol is considered a major risk if it is below 40 mg/dL, instead of the previous 35 mg/dL.
3. Dietary intake of cholesterol should be less than 200 mg/day, instead of the previous 300 mg/day.
4. There should be more aggressive treatment of triglycerides.
5. Less than 7 percent of the calories should come from saturated fat, instead of the previous 8 percent.
6. Dietary fat may be increased to 35 percent of the total daily calories instead of the previous 30 percent provided that the majority of these come from monounsaturated and polyunsaturated sources.
7. The new recommendations strongly urge the necessity of attaining and maintaining normal body weight as well as consistent participation in physical activity.

Table 2-1 Sources of Dietary Cholesterol and Saturated Fat

	Cholesterol (mg)*	Saturated Fat (g)**
Meats (3 oz.)		
Beef liver	372	2.5
Veal	86	4.0
Pork	80	3.2
Lean beef	56	2.4
Chicken (dark meat)	82	2.7
Chicken (white meat)	76	1.3
Egg	274	1.7
Dairy products (1 cup; cheese, 1 oz.)		
Ice cream	59	8.9
Whole milk	33	5.1
Butter (1 tsp.)	31	7.1
Yogurt (low fat)	11	1.8
Cheddar	30	6.0
American	27	5.6
Camembert	20	4.3
Parmesan	8	2.0
Oils (1 tbsp.)		
Coconut	0	11.8
Palm	0	6.7
Olive	0	1.8
Corn	0	1.7
Safflower	0	1.2
Seafood (3 oz.)		
Squid	153	.4
Oily fish	59	1.2
Lean fish	59	.3
Shrimp (6 large)	48	.2
Clams (6 large)	36	.3
Lobster	46.4	.08
Other items of interest		
Pork brains (3 oz.)	2,169	1.8
Beef kidney (3 oz.)	683	3.8
Beef hot dog (1)	75	9.9
Prime ribs of beef (3 oz.)	66.5	5.3
Doughnut	36	4.0
Milk chocolate	0	16.3
Green or yellow vegetable or fruit	0	Trace
Peanut butter (1 tbsp.)	0	1.5
Angel food cake	0	1.96
Skim milk (1 cup)	4	.3
Cheese pizza (3 oz.)	6	.8
Buttermilk (1 cup)	9	1.3
Ice milk, soft (1 cup)	13	2.9
Turkey, white meat (3 oz.)	59	.9

*There are 1000 mg in a gram.

**There are 28 g in an ounce and 454 g in a pound.

A number of population studies during the last twenty years have indicated a positive relationship between serum cholesterol (the level of cholesterol circulating in the blood) and the development of coronary heart disease. The National Heart, Lung, and Blood Institute reviewed this evidence and concluded that high circulating levels of serum cholesterol cause heart disease.

Values of serum cholesterol above 200 milligrams per deciliter (mg/dl) of blood are higher than the average risk. Table 2-2 shows the values of risk associated with total cholesterol (TC), LDL and HDL cholesterol (to be discussed shortly), and another serum lipid (blood fat), the triglycerides.

An important collaborative study involving twelve research centers throughout the United States provided clinical evidence implicating cholesterol as a culprit in coronary heart disease.[23] Half of a group of 3806 subjects was given a cholesterol-lowering drug, and the other half was given a placebo (a substance that looked like the drug but had no medicinal properties). The subjects were followed for approximately 7.4 years, at which time the data indicated that the drug group reduced their cholesterol levels by 13 percent, suffered 19 percent fewer heart attacks, and experienced 24 percent fewer fatal heart attacks. The incidence of coronary bypass surgery and angina were also significantly reduced. The researchers concluded that each 1 percent reduction in cholesterol level results in a 2 percent reduction in the risk of coronary heart disease.

A later follow-up of this study indicated that the reduction in coronary heart disease is probably closer to 3 percent for every 1 percent that cholesterol is lowered.[24] The strategies for lowering cholesterol in the blood are presented in Real-World Wellness: Strategies for Lowering Cholesterol.

The Cholesterol Carriers. The amount of cholesterol circulating in the blood accounts for only part of the total cholesterol in the body. Unlike sugar and salt, cholesterol does not dissolve in the blood, so it is

Table 2-2 Risk Profile—Lipid and Lipoprotein Concentrations

Total Cholesterol	Category of Risk
< 200 mg/dl[a]	Desirable
200–239 mg/dl	Borderline
≥ 240 mg/dl[b]	High
LDL Cholesterol	**Category of Risk**
< 100 mg/dl	Optimal
100–129 mg/dl	Near optimal/above optimal
130–159 mg/dl	Borderline high
160–189 mg/dl	High
≥ 190 mg/dl	Very high
HDL Cholesterol	**Category of Risk**
≤ 40 mg/dl[c]	Increased risk
≥ 60 mg/dl	Heart protective
Triglycerides	**Category of Risk**
< 150 mg/dl	Normal
150–199 mg/dl	Borderline high
200–499 mg/dl	High
≥ 500 mg/dl	Very high

[a] < is less than

[b] ≥ is equal to or greater than

[c] ≤ is equal to or less than

Adapted from "Revised Cholesterol Guidelines" July, 2001, *Harvard Heart Letter* 11(11):6–7.

Real-World Wellness

Strategies for Lowering Cholesterol

My doctor told me that my cholesterol level is too high. How can I lower it to reduce my risk of having a heart attack or stroke?

First, establish a realistic goal, such as about how much you think you can lower your cholesterol. Second, identify the lifestyle changes that can lower cholesterol. Third, attempt to do all of the following that apply to you:

- Reduce your fat consumption to less than 30 percent of total calories (25 percent would be better).
- Reduce your saturated fat consumption to less than 10 percent of total calories (8 percent would be better).
- Reduce your dietary cholesterol to less than 300 mg/day (less than 200 mg/day would be better).
- Lose weight if you are overweight.
- Stop smoking cigarettes and/or stop using other tobacco products.
- Increase your consumption of soluble fiber, found in fruits, vegetables, and grains.
- Do aerobic exercise at least three to four times per week for at least 30 minutes each time.

If these strategies fail to normalize your cholesterol level in six months, you may have to consider taking cholesterol-lowering drugs.

transported by protein packages, which facilitate its solubility. These transporters are the lipoproteins manufactured by the body. They include the chylomicrons, very low-density lipoprotein (VLDL), intermediate-density lipoprotein (IDL), low-density lipoprotein (LDL), and high-density lipoprotein (HDL). Dietary cholesterol enters the body from the digestive system attached to the chylomicrons. The chylomicrons shrink as they give up their cholesterol to the cells of the body. The fragments that remain are removed by the liver and used to manufacture and secrete VLDLs, triglyceride-rich lipoproteins. The triglycerides represent 99 percent of the stored fats in the body. The VLDLs are degraded as their cargo of triglycerides is either used by the cells for energy or stored in adipose cells. The VLDL remnants may be removed by the liver or converted to LDLs (figure 2-6).

LDLs are the primary transporters of cholesterol and the most capable of producing atherosclerosis. Michael S. Brown and Joseph L. Goldstein won a 1985 Nobel Prize in medicine and physiology for discovering that the liver and the cells of the body have receptor sites that bind LDLs, removing them from circulation. The liver contains 50 to 75 percent of these sites; the remainder are in other cells of the body. When LDL concentrations are excessive, the liver sites become saturated, and further removal of them from the blood is significantly impeded. As a result, plasma levels of cholesterol rise, leading to the formation and development of atherosclerotic plaque.

The development of atherosclerosis in the coronary arteries is complex. The *lipid oxidation theory* represents the latest thinking of the scientific community regarding the formation of plaque and therefore the growth and progress of atherosclerosis. This theory proposes that the initial event in the atherosclerotic process is an injury (lesion) that occurs to the inner lining of the arteries. Such injuries occur because of prolonged exposure to tobacco smoke, high blood pressure, elevated LDL cholesterol, diabetes mellitus, high amounts of serum homocysteine, and viral and bacterial infections.[3] These injuries, which occur at multiple sites, allow LDL

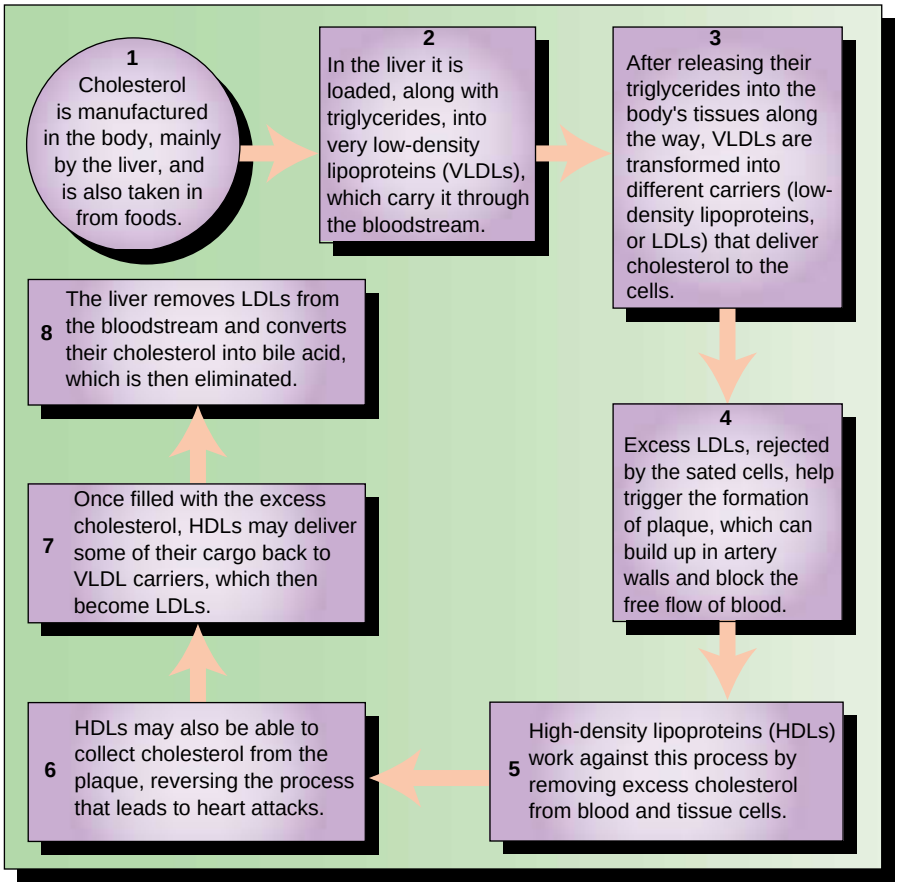
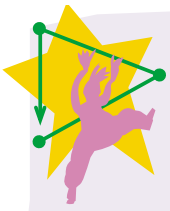


Figure 2-6 Cholesterol Carriers



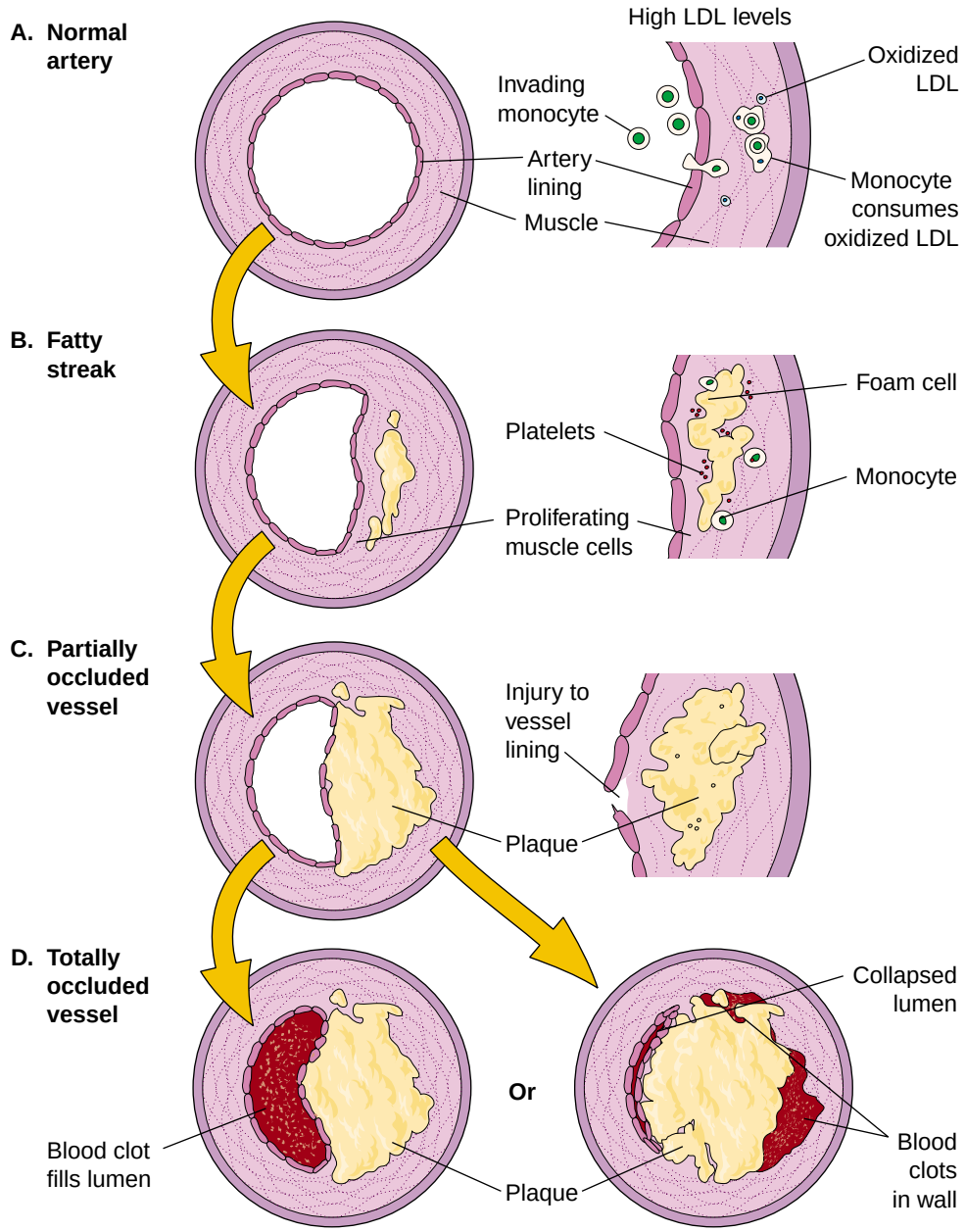
HealthQuest Activities

- The *Heart Attack Risk* and *Cardiovascular Exploration* activities in the Cardiovascular Health Module allow you to assess your risk for cardiovascular disease. In the first of these activities, *Heart Attack Risk*, answer the questions about your health history, behavioral practices, and knowledge about heart disease. *HealthQuest* will then provide the correct answers and point out any health risks indicated by your responses. In the *Cardiovascular Exploration* section, fill out all of the items and then click on the Show Me! button to find out your overall risk. You can get more specific feedback on each risk factor by clicking on the underlined words.

cholesterol to infiltrate under the artery lining, where they become oxidized (come in contact with oxygen). Oxidized LDLs become toxic and trigger the body's immune system to destroy them. The process of destroying oxidized LDLs contributes to the formation of plaque. Generally, two to three decades after the atherosclerotic process begins, plaque has progressed to the point that one or more arteries become so narrowed that a blood clot or spasm occurs, shutting off blood supply to a portion of the heart muscle. The result is a heart attack. See figure 2-7 for an illustration of this process.

Heart attacks are rare when LDL values in the blood are below 100 mg/dl. A national panel of experts has developed guidelines for safe and unsafe levels of LDL, and these appear in table 2-2. A high circulating level of LDL cholesterol is positively related to cardiovascular disease. Weight loss, a diet low in saturated fat and total fat, exercise, and medication (if needed) will lower LDL levels in the blood.

HDLs are involved in reverse transport; they accept cholesterol from the blood and tissues and transfer it to VLDLs and LDLs for transport to the liver, where it can be degraded, disposed of, or recycled. HDLs protect the arteries from atherosclerosis by clearing cholesterol from the blood. Cardiovascular health depends greatly on low levels of total cholesterol and LDLs and a high level of HDLs. Cigarette smoking, diabetes, elevated triglyceride levels, and anabolic steroids lower HDL, whereas physical exercise, weight loss, and moderate alcohol consumption raise it.



A. When a lesion occurs, LDL-Cs infiltrate below the artery lining, where they become oxidized and toxic. Monocytes follow and engulf the toxic LDLs.

B. The monocytes swell with LDL-C and become bloated. Platelets stimulate the smooth muscle cells to grow. Monocytes become foam cells. The growth bulges into the artery opening (lumen) and is recognized as a fatty streak (the first noticeable sign of atherosclerosis).

C. The bulge (now plaque) continues to grow over decades and the artery channels continue to narrow.

D. Eventually a blood clot forms and stops blood flow to the heart muscle. The result is a heart attack and death of a portion of heart muscle.

Figure 2-7 Progression of Atherosclerosis and Coronary Artery Disease

Moderate alcohol consumption (no more than two drinks per day for males and no more than one drink per day for females) increases HDL cholesterol. However, recent evidence has shown that three to four alcoholic drinks per week is sufficient to modestly raise HDL cholesterol. Caution should be exercised regarding alcohol intake because the health hazards associated with it far outweigh its few advantages. Alcohol consumption, even in moderate amounts, is not an acceptable way to raise HDL unless prescribed by a physician who is well aware of the patient's health and family history. An alcoholic drink is defined as a 5-ounce glass of wine or a 12-ounce beer or 1½ ounces of 80-proof spirits. However, alcohol is a depressant that impairs judgment and removes inhibitions so that people under its influence behave in ways they ordinarily would not while sober. Excessive alcohol intake is three drinks or more per day. Health is compromised at consumption levels above two drinks per day. The risks increase for heart disease, blood pressure, cirrhosis of the liver, breast cancer, and osteoporosis; also, the immune system weakens, red blood cell production slows down, and the brain is adversely affected. (See Chapter 10 for a more complete discussion of the effects of alcohol.)

Table 2-2 presents the current guidelines for HDL cholesterol levels in the blood. The higher the HDL, the greater the protection from cardiovascular disease. The average value for men is 45 mg/dl, and for women it is 55 mg/dl.[25] This biological difference in HDL levels between genders partly explains the lower incidence of heart disease in premenopausal women as compared with men. After menopause, HDL levels in women begin to decrease, as does their protection provided by this subfraction of cholesterol. The ratio between total cholesterol (TC) and HDL (TC/HDL) should also be considered when the risk is interpreted. This ratio is determined by dividing TC by HDL (table 2-3).[25]

Another blood fat, the serum triglycerides, is involved in the development and progression of atherosclerosis. Normal serum triglycerides range from 50 mg/dl to 150 mg/dl. Table 2-2 identifies the risk associated with various serum triglyceride levels.

High serum triglycerides usually coexist with low HDL cholesterol, a proven risk factor for cardiovascular disease. Teasing out the independent effect of serum triglyceride levels has been difficult. But recent evidence has shown that high levels are a modest independent risk factor for heart disease and a reliable predictor when coupled with low HDL cholesterol.[26] Serum triglycerides greater than 190 mg/dl increase blood viscosity (blood thickness), resulting in sluggish blood flow. Viscous blood is more difficult to circulate, so oxygen and nutrients are not delivered as efficiently to the body's tissues, and these include the heart muscle.[3]

Table 2-2 shows the relative risk posed by high serum triglycerides. Levels below 150 mg/dl are in the normal category, but evidence suggests that an optimal level is below 100 mg/dl.[27]

A number of studies have shown that sedentary people with high triglycerides can reduce serum triglycerides substantially when they participate in moderately intense aerobic exercise for 45 minutes 4 days per week.[28] Physically fit people metabolize serum triglycerides more effectively than do sedentary people and are able to clear these triglycerides from the blood more rapidly after a high-fat meal.[27]

Other strategies for lowering serum triglycerides include weight loss; reductions in dietary sugar, fat, and alcohol; and the substitution of fish for meat a couple of times during the week. Increasing fish consumption, as long as it is not fried, lowers the dietary intake of calories and saturated fat and increases the intake of omega-3 fatty acids, which tend to lower serum triglycerides.

Blood pressure

Blood pressure, recorded in millimeters of mercury (mmHg), is the force exerted against the walls of the arteries as blood travels through the circulatory system. Pressure is created when the heart contracts and pumps blood into the arteries. The arterioles (smallest arteries) offer resistance to blood flow, and if the resistance is persistently high, the pressure rises and remains high. The medical term for high blood pressure is **hypertension.**

Hypertension is a silent disease that has no characteristic signs or symptoms, so blood pressure should be checked periodically. Blood pressure can be measured quickly with a sphygmomanometer. A cuff is wrapped around the upper arm and inflated with enough air to compress the artery, temporarily stopping blood flow. A stethoscope is placed on the artery below the cuff so that the sound of blood coursing through the artery can be heard when the air in the cuff is released. The first sound represents the systolic pressure (the maximum pressure of blood flow when the heart contracts), and the last sound heard is the diastolic pressure (the minimum pressure of blood flow between heart beats). A typical pressure for young adults is 120/80. Pressures of 140/90 or greater are considered to be hypertensive.[1] The lower limit of normal is 100/60. From a health perspective, having a low to normal blood pressure and maintaining it as long as possible is advantageous.

Table 2-3 Ratio of Total Cholesterol to HDL Cholesterol

Risk	Men	Women
Very low (one-half average)	<3.4	<3.3
Low risk	4.0	3.8
Average risk	5.0	4.5
Moderate risk (two times average)	9.5	7.0
High risk (three times average)	>23	>11

Keeping your blood pressure under control helps keep your heart healthy.

Table 2-4 Classification of Blood Pressure for Adults Age 18 Years and Older

Category	Systolic (mmHg)	Diastolic (mmHg)
Normal	<130	<85
High normal	130–139	85–89
Hypertension		
Stage 1 (mild)	140–159	90–99
Stage 2 (moderate)	160–179	100–109
Stage 3 (severe)	180–209	110–119
Stage 4 (very severe)	≥210	≥120

Standards for classifying blood pressure in adults appear in table 2-4. This table represents the latest thinking among medical researchers about the stages of blood pressure values. All hypertensive values in the table represent an increased risk for cardiovascular and kidney disease. The higher the blood pressure, the greater the risk. The lower the blood pressure (within normal range), the better. For example, a 2 mmHg decrease in systolic blood pressure is estimated to reduce the risk of mortality from stroke by 6 percent and from coronary heart disease by 4 percent.[29] Recent evidence indicates that an increase in systolic blood pressure (SBP) accompanied by a decrease in the diastolic blood pressure (DBP) doubles the risk of dying from cardiovascular disease.[17] This combination seems to be more dangerous than when both pressures increase. The DBP decreases when the large arteries stiffen.

The difference between the SBP and DBP is called the "pulse pressure" (SBD minus DBP).[30] The SBP increases because age and poor lifestyle habits cause the arteries to lose their resilience. At the same time, the DBP decreases because the stiff arteries aren't as responsive to blood flow between heartbeats. A pulse pressure of 60 mmHg or more is associated with the development and progression of atherosclerosis.

Approximately 50 million American adults and children have high blood pressure, and 35,130 people die of its complications annually.[1] The cause of high blood pressure is not known in 90 to 95 percent of the cases. This is referred to as *essential hypertension*. *Essential* is a medical term that means "of unknown origin or cause." Essential hypertension cannot be cured, but it can be controlled. The other 5 to 10 percent of the cases of hypertension have a specific cause. If the cause can be determined and eliminated, blood pressure will return to normal.

The heart is adversely affected by uncontrolled or undiagnosed hypertension of long duration. Pumping blood for years against high resistance in the arteries increases the workload of the heart, and it becomes enlarged in response to the strain. The heart receives inadequate rest because the resistance to blood flow is consistently high, and this produces overly stretched muscle fibers. They progressively lose the ability to rebound. The result is that they contract less forcefully. At this point the heart loses its efficiency and weakens. If intervention does not occur early, congestive heart failure is inevitable. Hypertension also damages the arteries and accelerates atherosclerosis.

Hypertension is the most important risk factor for brain attacks (strokes) as well as a major risk for heart disease. More than 500,000 brain attacks occur every year. Certain segments of the population are at higher risk, such as African Americans, who have more than a 60 percent greater risk of death and disability from stroke than do whites; this is a direct result of pervasive hypertension.[31] Stroke mortality is highest in African-American females born before 1950 and in African-American males born after 1950.

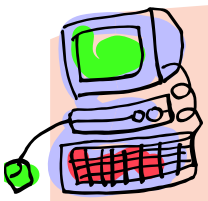
Treatment for Hypertension. Treatment for hypertension may include all or some of the following: weight loss, salt and alcohol restriction, calcium and potassium supplementation, voluntary relaxation techniques, exercise, and medication. Excess body weight increases the work of the heart because it must meet the nutrient demands of the extra tissue.

High salt intake increases the blood pressure in those who are salt sensitive. Estimates indicate that at least 30 percent of Americans are salt sensitive, but the actual number is probably higher. Salt is an acquired taste that can be modified. Curbing salt consumption while blood pressure is still normal cannot hurt and it may help.

Some calcium-deficient hypertensive people may normalize their blood pressure by taking calcium supplements. However, calcium supplementation does not work for all calcium-deficient hypertensive people, and it does not work for hypertensive people who are not calcium deficient.

Yoga, meditation, hypnotherapy, biofeedback, and other relaxation techniques can lower the pressure of hypertensive people. They can also reduce the pressure of normotensive people (those with normal blood pressure).[32] Meditating hypertensive people lowered their systolic pressure by 11 points and their diastolic pressure by 6 points more than did people in a control group counseled on the importance of losing excess weight, exercising regularly, cutting salt consumption, and drinking less alcohol.

Many people drink alcohol because it relaxes them, but ingestion of more than 2 ounces of alcohol per day raises blood pressure in some people. The same precautions regarding the use of alcohol and HDL cholesterol apply for hypertension.



Wellness on the Web Behavior Change Activities

What's Your Risk for Heart Disease?

In the battle to maintain a healthy heart, you have a powerful ally. The American Heart Association (AHA) is dedicated to educating and informing you on how to fight heart disease and stroke. Go to the OLC at www.mhhe.com/anspaugh5e, select Chapter 2, and complete the AHA's interactive risk assessment titled "What's Your Risk?" This assessment will show you what factors place you at greater risk for heart disease and allow you to take charge of your health by making more healthful lifestyle choices. Complete the 16 items and then click on the Submit button for your results. Which of your risk factors cannot be changed? Which can you change?

What Questions Should I Ask My Physician?

Often we tend to think of doctors as all-knowing gurus who can tell us everything we need to live a healthy life. The fact is, your relationship with your physician is a partnership—and for best results, you need to know what to ask when you visit your doctor. The American Heart Association reminds you that, after making healthful lifestyle changes, your second-best friend in the fight against heart disease and stroke is your doctor. For help in formulating questions for this vital partner in your health care, go to the OLC at www.mhhe.com/anspaugh5e, select Chapter 2, and read the AHA's 10 questions you should ask your doctor at your next checkup in the section titled "Ask Your Doctor." Help your physician help you live a healthier life!

The American College of Sports Medicine (ACSM) has developed a position paper on the relationship between exercise and hypertension.[33] Some of the more important conclusions follow:

1. Endurance exercises can reduce both systolic and diastolic pressures by 10 points each in people with mild essential hypertension (with systolic pressures between 140 and 180 mmHg and diastolic pressures between 90 and 105 mmHg).
2. Moderately intense endurance exercises lower the blood pressure as much as or more than vigorous exercise.
3. Endurance exercise training delays or prevents the onset of hypertension in people at high risk.
4. By itself, resistive or strength training does not seem to lower the blood pressure unless it is accompanied by aerobic training and is done in a circuit (circuit training is explained in Chapter 4).
5. Physically fit hypertensive people have a lower mortality rate than do physically unfit normotensive people.

The mechanisms through which exercise lowers blood pressure in hypertensive individuals are not fully understood. The probable mechanisms include the following:

1. Exercise contributes to weight loss, which in turn lowers blood pressure.
2. Aerobic training reduces resistance to blood flow in the arteries and at rest, lowering the force required to circulate blood.
3. Many hypertensive people have elevated levels of hormones that constrict blood vessels. Exercise metabolizes these, which results in less resistance to blood flow.
4. Exercise training sensitizes the cells to insulin, so less circulates in the blood. The kidneys respond by excreting sodium, which reduces the blood pressure.

Medications that control blood pressure have been developed. However, they have side effects that include weakness, leg cramps, stuffy nose, occasional diarrhea, heartburn, drowsiness, anemia, depression, headaches, joint pain, dizziness, impotence, and skin rash. The side effects are unique to the drugs being used to control blood pressure. Despite these side effects, many people find it easier to take the medicine than to make the difficult lifestyle changes that lower blood pressure. However, the effort to control blood pressure with medication should not negate the importance of controlling lifestyle factors that contribute to hypertension. Medicine and lifestyle efforts are not mutually exclusive—each contributes to blood pressure management.

Cigarette smoking/tobacco use

Many medical authorities consider cigarette smoking the most harmful of the preventable risk factors associated with chronic illness and premature death.[34] Cigarette smoking is responsible for one of every five deaths annually in the United States.[35] It contributes to approximately 420,000 premature, preventable deaths from cardiovascular and pulmonary diseases. About 90 percent of lung cancer, 80 percent of emphysema, 75 percent of bronchitis, and 30 percent of coronary heart disease is attributable to smoking. In 1987, lung cancer became the leading cause of cancer death among women, replacing breast cancer. In 1997, 66,000 women died of lung cancer compared to 44,000 who died of breast cancer.

Harmful Products in Cigarettes. Nicotine; carbon monoxide; and other poisonous gases, tars, and chemical additives for taste and flavor are the hazardous

products in cigarettes. Carbon monoxide and nicotine have a devastating effect on the heart and blood vessels. Nicotine is an addictive stimulant that increases the resting heart rate, blood pressure, and metabolism. For this reason, it should be reclassified as a drug and placed under the jurisdiction of the Food and Drug Administration (FDA).

A brief summary of nicotine's effects on the cardiovascular system is found in Just the Facts: Nicotine. Carbon monoxide, a poisonous gas that is a by-product of the combustion of tobacco products, displaces oxygen in the blood because it has a greater attraction to hemoglobin. The diminished oxygen-carrying capacity of the blood is partly responsible for the shortness of breath that smokers experience with mild physical exertion.

Cigarettes and other tobacco products are not regulated by the FDA because tobacco is not classified as a food or drug. The tobacco industry is under no mandate to disclose the nature and type of chemicals added to tobacco products. Some of these additives are harmful. The public has a right to know, but the tobacco industry has successfully resisted attempts by government agencies and consumer groups to force disclosure.

The harmful effects of cigarette smoking are insidious and take time to appear. The medical profession measures the damage from smoking in pack years. Smoking one pack of cigarettes per day for fifteen years is equal to 15 pack years (15 years × 1 pack per day = 15 pack years). Two packs per day for fifteen years is equal to 30 pack years (15 × 2 = 30). Medical problems become evident after 25 to 30 pack years.

The Challenge of Quitting. To quit the tobacco habit, you have to simultaneously break the addiction to nicotine and the psychological dependence on smoking. This involves changing behavior and effectively dealing with the social and situational stimuli that promote the desire to smoke. One-third of current cigarette smokers attempt to stop smoking every year but less than 10 percent of them succeed.[36] Most quitters made four to ten unsuccessful attempts and tried several stop smoking methods before finally succeeding. Quitting the smoking habit is a formidable challenge. However, 50 percent of active smokers have managed to stop smoking in the last fifty years. While exceedingly difficult, it can be done if one finds the right motivation. Since 1964, more men than women have quit, more whites than African Americans, and more non-Hispanics than Hispanics. More elderly people and more educated people also have higher quit rates.[37]

Complicating the effort to quit, particularly among young women, is the fear of gaining weight. Approximately 65 percent of those who quit do gain weight, but the physiological adaptations that occur may only account for a 7- to 8-pound weight gain. The physiological mechanisms responsible are probably associated with a slowing of metabolism, a slight increase in appetite, and slower transit time of food in the digestive system so that more is absorbed by the body. Weight gain beyond 8 pounds is probably caused by altered eating patterns rather than physiology. Food smells and tastes better when a person is not smoking. Food may substitute for a cigarette, especially during social activities. It may provide some of the oral gratification previously obtained from smoking, and it may relieve tension. Weight gain can be avoided by eating sensibly and exercising moderately and frequently.

As a group, smokers are approximately 7 percent thinner than nonsmokers, but smokers tend to distribute more fat in the abdominal area.[38] The waist-to-hip ratio (WHR) is greater in smokers even though they are thinner. This fat distribution predisposes smokers to coronary heart disease, diabetes, stroke, and some forms of cancer.

Passive Smoking and Smokeless Tobacco. Involuntary or passive smoking (inhaling the smoke of others) is associated with premature disease and death. Estimates indicate that 38,000 to 43,000 nonsmokers who are regularly exposed to environmental smoke die annually from smoking-related causes.[39] The majority of these (35,000 to 40,000) die from heart disease, and 3000 die from lung cancer. There is a dose-response effect. The more the nonsmoker is exposed to environmental smoke, the greater his or her risk for premature morbidity (illness) and mortality (death).

Children of smoking parents are more likely to experience a higher incidence of influenza, colds, bronchitis, asthma, and pneumonia. The impact of passive smoking on them can last a lifetime and may range

Just the Facts

Nicotine

Nicotine is a powerful stimulant that:

- Increases LDL and lowers HDL levels
- Causes the platelets to aggregate, increasing the probability of arterial spasms
- Increases the oxygen requirement of cardiac muscle
- Constricts blood vessels
- Produces cardiac dysrhythmias (irregular heart beat)
- Is a causative agent in the 30% of coronary heart disease deaths related to smoking
- Increases the viscosity of the blood

from delayed physical and intellectual development to the hazards associated with prolonged exposure to carcinogenic substances.

An alarming trend is the escalating sale of smokeless tobacco products. Chewing tobacco and dipping snuff have become popular among high school and college men. The World Health Organization (WHO) has described the growing use of smokeless tobacco as a new threat to society. Nicotine is an addictive drug regardless of the method of delivery, and its effects are similar whether it is inhaled, as in smoking, or absorbed through the tissues of the oral cavity, as in dipping and chewing. The incidence of oral cancer may be 50 times higher among long-term users of smokeless tobacco products than among nonusers. Smokeless tobacco is addictive and deadly, and its use is rising among adolescent males.

Cigar sales in the country had been flat for twenty-five years until 1994, when sales began to increase as the result of a marketing campaign by the magazine *Cigar Aficionado*, cigar invitation-only dinners, and celebrity endorsements of cigar smoking, characterizing it as sophisticated and glamorous. Cigar smoking is no longer looked upon as the sole dominion of males; many women have taken up the habit. Cigar sales increased from 3.4 billion in 1993 to 5.1 billion in 1997.[40]

Cigars were not specifically included in the 1984 law that required tobacco companies to place labels on packages of cigarettes warning that they were hazardous to health. As a result, cigars carry no such warning label. This does not mean that cigar smoking is not harmful to health. See Just the Facts: Cigar Smoking: A Hazard to Health for the facts on cigar smoking.

Just the Facts

Cigar Smoking: A Hazard to Health

A common assumption is that cigar smoking is not as hazardous as cigarette smoking, because people usually don't inhale cigar smoke. The facts regarding the dangers of cigar smoking are:

- Cigars contain nicotine, carbon monoxide, tars, and poisonous gases.
- Nicotine reaches the brain by being absorbed through the lining of the mouth rather than from the lungs, but the effect is the same. See Just the Facts: Nicotine for a summary of nicotine's harmful effects on the heart and blood vessels.
- Most of the same cancer-producing substances found in cigarettes are also present in cigars.
- Overall cancer deaths are 34 percent higher for men who smoke cigars than for men who do not.
- Cigar smokers have 4 to 10 times more risk of dying from mouth and throat cancers than do nonsmokers.

Physical inactivity

Physical inactivity has been officially recognized as a major risk factor for cardiovascular disease by the American Heart Association.[1] The upgrading of physical inactivity, which appeared in the AHA's 1993 report, reflects the importance of participating in physical activities regularly. The AHA made the upgrade because the weight of the evidence that has been accumulating in the last few decades shows that exercise produces many important health benefits. This is good news for those who have been physically active, and it may motivate some sedentary people to become active.

Physical inactivity (hypokinesis) is debilitating to the human body. A couple of weeks of bed rest or chair rest produce muscle atrophy, bone demineralization, and decreases in aerobic capacity and maximum breathing capacity. Your body was constructed for and thrives on physical exertion.

Two major reviews have shown that physical inactivity poses a significant risk for developing heart disease. In one review, the researchers critiqued forty-three studies and concluded that physical inactivity increased the risk for coronary heart disease by 1.5 to 2.4 times.[41] The risk associated with physical inactivity is similar to that of the other major risk factors. According to the Centers for Disease Control and Prevention, the need for regular exercise by the general public should be promoted as vigorously as efforts to control blood pressure, lower cholesterol, and stop smoking. A later review by another team of researchers concluded that inactive people have a 90 percent greater risk for developing coronary heart disease than do active people.[42]

With few exceptions, the results of later studies are consistent with the results of these two reviews. Some of the more recent studies have indicated a dose-response relationship between level of physical activity and cardiovascular disease.[43] This means that (1) men at high risk who regularly participate in light- to moderate-intensity physical activity expending about 1500 calories per week will likely lower their risk for coronary heart disease by about 25 percent to 50 percent, and (2) men who engage regularly in more intense physical activity (above the moderate level) lower their risk for coronary heart disease by 60 to 70 percent and experience greater longevity.

Several studies examined the effect on coronary heart disease of level of physical fitness rather than of total numbers of calories expended per week in physical activity. These studies corroborated the results of the calorie expenditure studies that physically fit people are less inclined to develop coronary heart disease than are unfit people.[44]

In summary, people actively engaged in leisure-time or occupational physical activity as well as those who participate in physical activities for the purpose of developing fitness are at a lower risk of death from cardiovascular disease and all-cause mortality. Other major studies have supported the view that people who regularly engage in physical activities of moderate intensity have significantly fewer heart attacks and experience fewer deaths from all causes than do people who exercise little or not at all. Moderate activity was described as the equivalent of walking 1 to 2 miles per day for a total of 5 to 10 miles per week at a speed of 3 to 4 MPH. The greatest health benefits were gained by those who expended 1500 to 2000 calories per week (15 to 20 miles of walking) in physical activity. A total of 17,000 men were followed for more than thirty years. Those who regularly walked, climbed stairs, or participated in sports activities decreased their risk from all causes of mortality. Those who expended a minimum of 500 calories per week (5 miles of walking or its equivalent) to a maximum of 3500 calories per week (35 miles of walking or its equivalent) experienced a progressive increase in longevity.

Investigators at the Cooper Institute for Aerobics Research[45] studied the relationship between physical fitness and mortality from all causes. The uniqueness of this study was twofold: first, the researchers measured the physical fitness levels of all subjects by treadmill testing, and second, more than 3000 of the 13,344 subjects were women. Because of their lower risk for cardiovascular disease, women have essentially been neglected as subjects in heart disease studies.

The results of this study indicated that a low physical fitness level increased the risk for both men and women of death from cardiovascular disease, cancer, and all other forms of disease. The difference in all-cause mortality was greatest between those in the moderately fit category and those in the low-fit category. The difference between the moderately fit and the highly fit was insignificant. For people who exercise regularly, the risk of dying from a heart attack is 35 to 55 percent less than it is for sedentary people.[46]

A study using only women as subjects investigated the physical fitness benefits versus the health benefits of three levels of walking intensity.[47] One group walked at 5 MPH, a second group walked at 4 MPH, and a third group walked at 3 MPH. The results showed that physical fitness improved on a predictable dose-response basis. The fastest walkers improved the most and the slowest walkers improved the least, but the cardiovascular risk was reduced equally among the three groups. Low-level exercise was as effective as the highest level in promoting cardiovascular health. Exercise for health does not have to be as strenuous as exercise for physical fitness.

The American College of Sports Medicine and the Centers for Disease Control and Prevention reacted to the results of these studies by jointly issuing a recommendation for exercise for Americans. The new guideline states that "every U.S. adult should accumulate 30 minutes or more of moderate intensity physical activity on most, preferably all, days of the week."[48] This is a minimum guideline for exercise and was not intended to replace previous recommendations regarding more vigorous exercise for the purpose of improving physical fitness. Instead, the recommendation is directed at that 60 percent of our population who are sedentary or marginally active. Health professionals hope that the recommendation will motivate physically inactive and underactive Americans to participate in a more active lifestyle.

The term *physical activity* refers to any physical movement that results in energy expenditure. Physical activity includes but is not limited to walking, climbing stairs, mowing the lawn (using a riding mower does not count), raking leaves without a blower, mopping and vacuuming floors, washing and waxing the car (by hand), dancing, and playing with children and grandchildren. Moderate intensity physical activity can be achieved by walking 3 to 4 miles per hour or through any other activity that burns as many calories as walking at those speeds.

People who consistently exercise above the moderate level not only receive the health benefits but also develop a higher level of physical fitness. Those who exercise in accordance with the guidelines gain about the same health benefits as those who are more physically active, but they will not attain the same degree of physical fitness. That 60 percent of the population inclined to ignore the advice to exercise regularly can become part of the estimated 250,000 premature deaths per year attributed to a **sedentary** lifestyle.[4]

The health and longevity returns from exercise and a physically active lifestyle are significant. Estimates indicate that longevity is increased by 1 minute for every minute spent walking and by 2 minutes for every minute spent jogging.[49] The potential for improving the health status of Americans through appropriate lifestyle behaviors is evident from estimates indicating that 60 percent of deaths are premature and approximately 50 to 60 percent of all illness and disabilities are preventable.[50]

Obesity

Obesity strains the heart and coexists with many of the modifiable risk factors that promote cardiovascular disease. Obese people who have no other risk factors are still more likely to develop heart disease or stroke. Obesity contributes to approximately 300,000 deaths annually in the United States.[34] Not only is obesity associated with an increased risk for heart disease, but the manner in which fat is distributed in the body might also accentuate the risk.[34] Fat that accumulates in the upper half of the body (referred to as *visceral* or *central abdominal obesity*) is likely to be accompanied by high triglycerides, low HDL cholesterol, insulin resistance, and hypertension. This cluster of factors is called *syndrome X*, and it is associated with a significant increase in the likelihood of developing cardiovascular disease.

Obese people can lower their risk with a modest weight loss of 5 to 10 percent. This is a realistic, attainable goal. But the risk remains lower only if the loss of weight is maintained. Approximately 95 percent of people who lose weight regain it in a few years. Although calorie restriction is the primary method for losing weight, regular exercise is the most effective method for maintaining the loss. Diet and exercise are not mutually exclusive; instead, they compliment each other, and both are important players in weight management.

Diabetes mellitus

Diabetes mellitus is a metabolic disorder in which the body cannot make use of sugar (glucose) as a fuel. The hormone insulin must be produced and secreted into the bloodstream so that blood sugar can be transported into the cells. The cells have receptor sites to which insulin attaches, making the cell amenable to the entrance of sugar.

In type 1 diabetes, no insulin is produced, and so it must be injected daily. Type 1, insulin-dependent diabetes mellitus (IDDM), usually occurs early in life. Type 2, or non–insulin-dependent diabetes mellitus (NIDDM), occurs in middle-aged, overweight, sedentary adults. Excessive weight is a factor because it increases cellular resistance to insulin so that more insulin than normal is required to effect the passage of sugar from the blood to the cells. In contrast, exercise decreases insulin resistance, making cellular membranes more permeable to sugar. About 90 percent of diabetes mellitus is of the type 2 variety. Data indicate that at least 75 percent of new cases of type 2 diabetes can be prevented through regular exercise and maintaining normal weight.[51]

Diabetes mellitus has numerous long-range complications. These primarily involve degenerative disorders of the blood vessels and nerves. Diabetics who die prematurely are usually the victims of cardiovascular lesions and accelerated atherosclerosis. The incidence of heart attacks and strokes is higher among diabetics than nondiabetics. Diabetes increases the risk of coronary artery disease by 2 to 3 times the normal rate in men and 3 to 7 times the normal rate in women.[34]

The arteries supplying the kidneys, eyes, and legs are particularly susceptible to atherosclerosis. Kidney failure is one of the long-term complications of diabetes. Diabetes is also the leading cause of blindness in U.S. adults. Impaired delivery of blood to the legs may lead to gangrene, necessitating amputation of the affected tissues. In addition to circulatory problems, degenerative lesions in the nervous system may result, leading to multiple diseases that result in dysfunction of the brain, spinal cord, and peripheral nerves.[52] Unfortunately, medical science has been unable to identify the biological mechanisms responsible for these long-term vascular and neural complications. However, these complications can be mitigated by leading a balanced, well-regulated life, thereby keeping diabetes under control. Control includes dietary manipulation, exercise, weight control, rest, and medication if needed.

The landmark Physician's Health Study was the first major effort to show that exercise reduced the risk of developing type 2 diabetes. The physicians participating in the study who exercised vigorously five or more times per week had a 42 percent greater reduction in the incidence of type 2 diabetes than did those who exercised less than one time per week. The reduction in risk was particularly pronounced among those at greatest risk: the obese. The researchers concluded that at least 24 percent of cases of type 2 diabetes were related to sedentary living. Even high-risk men (those who were overweight and had a parental history of diabetes) benefited from regular exercise. Every 500 calories burned per week in leisure-time physical activity reduced the risk of type 2 diabetes by 6 percent.[53]

Data collected from more than 70,000 women subjects ages 40 to 65 years indicated that those who participated in moderate exercise on a regular basis lowered their risk of developing type 2 diabetes compared to women who did not exercise regularly.[54] Accumulating evidence shows that the effect of exercise on type 2 diabetes prevention applies to both men and women.

Stress

Stress is difficult to define and quantify. Authorities agree that distress, or chronic stress, produces a complex array of physiological changes in the body. Together, these physiological events are called the *fight or flight response*. The hormones released by the body during these events produce the stress response. This includes (1) increases in heart rate, breathing rate, blood pressure; (2) the tendency for blood platelets to

Nurturing Your Spirituality

Can Stress and Depression Make You Sick?

A convincing body of evidence suggests that chronic anger, anxiety, loneliness, or depression can be catastrophic for people with coronary artery disease.[55] At the same time, emerging evidence shows that these same mood states and feelings in healthy people may increase the likelihood of developing heart disease in the future.

Scientists are beginning to unravel the connection between mood states and heart disease. Consistently high levels of stress hormones circulating in the bloodstream suppress the immune system by interfering with the normal repair and maintenance functions of the body. This increases one's vulnerability to infections and disease. Continuing high levels of cortisol and norepinephrine stimulate a prolonged fight or flight response that can eventually lead to wear and tear on the heart and arteries. Frequent and prolonged periods of stress increase blood pressure, and that usually leads to injuries of the artery walls. These injuries are the first step in the development and ultimate progression of atherosclerosis.[56] Data indicate that exaggerated responses to stress may be a triggering mechanism for heart attack and stroke.

Depression (prolonged sadness beyond a reasonable length of time) also stimulates the production of stress hormones. Depression that occurs later in life increases the risk of coronary heart disease in two ways: (1) by reducing blood flow to the heart in those whose blood vessels are narrowed and (2) by causing heart rhythm disturbances.[57]

Scientists have much to learn about the relationship between the heart and mind. Physicians are beginning to acknowledge that depression is a significant factor in producing cardiovascular complications for those who already have heart disease. The good news is that depression is treatable. Mild depression responds to regular exercise and voluntary relaxation techniques, both of which reduce the production of stress hormones. On the other hand, severe depression requires psychological counseling and medication plus regular exercise and relaxation training. Eighty percent of people with severe depression respond to treatment.

Regular exercise promotes relaxation, reduces the response to stress, enhances emotional well-being, and lowers cardiac reactivity (high heart rate, blood pressure, and resistance to blood flow). Cardiac reactivity occurs when modest stressors produce physiological responses by the heart and circulatory system that are out of proportion to the stressor. If these occur frequently, the development of atherosclerosis may well be the result.

Exercise acts as a safety valve that enables people to "let off steam" in a constructive way. Jogging, swimming, cycling, weight training, racquetball, and other physical activities focus our energies in worthwhile pursuits that rid the body of stress products that have accumulated. Exercise training, a physiological stressor, helps build tolerance to psychological and emotional stressors. In other words, the "physiological toughness" developed through exercise training enables us to cope more effectively with other types of stressors.[58]

Do stress and depression cause heart attacks? The answer is a qualified "yes." We should have a more definitive answer after a few more years of research.

aggregate (clump together); (3) blood sugar rushing to the muscles to provide more energy; and (4) the activation of the immune system. The stress response represents a significant strain on the body.

Stressors (events or situations that cause stress) may be acute or chronic. Acute stressors are situational and temporary. Taking a midterm exam or making an oral presentation are examples of events that provoke acute stress. When such an event is over, the body returns, within a short time, to its prior state of balance and harmony. However, chronic stress (characterized by prolonged elevations of stress hormones and a general feeling of uneasiness that permeates one's life) presents a much more serious problem. Constant worry about work, finances, or relationships or persistent feelings of anger and isolation are examples of chronic stress. Is chronic stress a causative agent in the development of chronic diseases in general and heart disease specifically? See Nurturing Your Spirituality: Can Stress and Depression Make You Sick?

Preventing and Reversing Heart Disease

Preventing heart disease is much preferred to treating it after the fact. Prevention includes regular exercise, maintenance of optimal body weight, sound nutritional practices, abstinence from tobacco products, nonuse of alcohol (or use in moderation), and abstinence from drugs. Dealing with stress in constructive ways, removing oneself as much as possible from destructive and disease-producing environmental conditions, and having periodic medical examinations are other aspects of prevention. It is much better physically, psychologically, and economically to make the effort to enhance health now than to reject or ignore health promotion principles and treat disease later. It is never too late to change behavior. Even patients with coronary artery disease can benefit from lifestyle changes.

Until recently, medical thinking indicated that established atherosclerotic plaques in the coronary arteries

Stress-relieving activities, such as meditation, soothe the spirit and may even lower your risk of heart disease and other chronic illnesses. How do you manage stress?

were there to stay. Progression of the disease seemed inevitable unless medical corrective procedures were employed. But evidence has surfaced indicating that reversal of the disease is possible with appropriate lifestyle behaviors.

Dean Ornish showed that comprehensive behavior changes are required to reverse established coronary artery disease. Ornish devised a program that included a vegetarian diet in which only 6.8 percent of the calories came from fat; 4.4 hours of moderate aerobic exercise per week; stress-management techniques consisting of stretching exercises, practicing of breathing techniques, meditation, progressive relaxation, and the use of imagery; smoking cessation; and attendance at regular group support meetings. The subjects in this study were evaluated against a control group who received "usual and customary" care.[59]

At the end of the first year, 82 percent of the subjects in the Ornish program showed regression of atherosclerosis compared with only 10 percent of the usual care group. Over the following four years, the Ornish subjects showed further regression, whereas the usual care group experienced progression of atherosclerosis.[60]

Other investigators have examined the effect of less stringent interventions than those advocated by the Ornish program on the regression of atherosclerosis. These attempts have been less successful than the Ornish program but more successful than the usual care program.

The main criticism of the Ornish program was that lifelong compliance would be difficult. Twenty-nine percent of the study participants dropped out during the last four years of the program. It is not easy to permanently change bad habits, but the hard work associated with following a low-fat diet, exercising consistently at a moderately intense level, and giving up smoking can make people with coronary artery disease feel better and may allow them to avoid surgery.

Refer to Assessment Activity 2-1 at the end of this chapter. Respond to each of the risk questions about factors to determine your risk status.

Other Possible Risk Factors

Iron-Enriched Blood

In 1992 a study completed in Finland found that men with high levels of iron (as measured by serum ferritin) also had a high probability of incurring heart problems. Since then, a number of studies completed in the United States have failed to confirm the high iron–high heart disease connection. These American studies found the opposite. Women with high iron levels were found to have half the heart disease risk as women with low iron stores, and men in the high iron group were 20 percent less likely to die of heart disease than were men with the lowest levels.[61] The rationale is that low iron levels lead to a reduction in oxygen-carrying hemoglobin so that less oxygen is transported to the tissues. It is possible that the decrease in oxygen transport to the cells, including the cells of the heart, is responsible for the heart problems that occur with greater frequency in those who are iron deficient.

Now that you are aware of the major risk factors for heart disease, refer to Assessment Activities 2-1 and 2-2 to test your risk and your knowledge. Be as accurate as possible when providing the necessary information for Activity 2-1. Read the case study (Assessment 2-2) and respond to all of the questions.

Homocysteine

Homocysteine is an amino acid (a building block of protein) derived from methionine metabolism.[34] Methionine is one of the essential amino acids that must be obtained through the diet protein foods. Homocysteine is carried through the bloodstream, and under normal conditions, it is broken down into two other amino acids. When that breakdown does not occur, homocysteine levels rise to present a risk for heart disease. Several studies, including the Physician's Health Study and the Framingham Heart Study, showed that the probability of having a heart attack increases to 3 times the normal level in subjects with the highest homocysteine levels. This puts homocysteine on par with the other major risk factors for coronary artery disease.[62]

Homocysteine is measured in *micromoles*, which are based on molecular weight. The normal range is 4 to 15 micromoles per deciliter of blood. Heart attacks are more likely to occur to those whose levels are above 15.[63]

Homocysteine contributes to heart disease probably by damaging the inner lining of the arteries. The damage initiates the abnormal growth of smooth muscle cells,

which in turn promotes the development and progression of atherosclerosis. It also promotes blood platelet aggregation that can lead to the formation of blood clots.

The good news is that homocysteine levels can be controlled with diet. The B vitamins (folic acid or folate, B_6, and B_{12}) are needed to split homocysteine into other amino acids. Folate is the most important of the three, but evidence indicates that optimal benefits occur when all three are available in appropriate amounts. The recommended daily amounts for these vitamins are 400 micrograms of folate, 6 micrograms of B_{12}, and 2 milligrams of B_6.

Lipoprotein (a) or Lp (a)

Lipoprotein (a) represents a group of particles that resemble LDL-C (the bad form of cholesterol). A high circulating level of Lp (a) in the blood may increase the risk for coronary artery disease and stroke, and although study results are not yet conclusive, they appear to be heading in that direction.[17] Many scientists believe that high levels of Lp (a) promote the formation of blood clots and increase the ability of LDL-C to form plaque (the material that blocks arteries).

Lp (a) levels above 30 milligrams per deciliter of blood are considered high. Some medical researchers believe that high values of Lp (a) may be as dangerous to the heart as low levels of HDL-C (the good form of cholesterol).[64] Lp (a) levels are determined primarily by one's genetic composition. Healthy behaviors such as consuming a low fat diet, regular exercise, and so on seem to have little effect on Lp (a). Thus far, the only successful treatments for reducing Lp (a) are large doses of niacin, a water-soluable vitamin, and possibly estrogen supplementation for postmenopausal women.[17] People with high levels of Lp (a) should concentrate on lowering LDL-C and total cholesterol, raising HDL, and keeping the other heart disease risk factors under control.

High Sensitivity C-Reactive Protein (hs-CRP)

C-reactive protein has been used as a diagnostic test for inflammatory rheumatoid conditions. Scientists have improved this test in the last few years by making it more sensitive to inflammation in the body. The new and improved version, high sensitivity C-reactive protein (hs-CRP) can identify inflammation earlier and at lower levels than its predecessor. Atherosclerosis, the underlying cause of 80 percent of coronary heart disease, is a low-level inflammatory process. The newer, more sensitive test shows promise as a diagnostic tool for detecting atherosclerosis early so that an intervention program can be developed and put in place early in the process.

More than 28,000 postmenopausal women free of cardiovascular disease were tested for inflammation, homocysteine level, and seven different lipid or lipoprotein risk factors.[17] The subjects were followed for three years. During that time some of them experienced one or more cardiovascular events. They were compared to age-matched subjects who had not had a cardiovascular event. The most accurate predictor of a potential event was hs-CRP. The study director stated that hs-CRP was statistically twice as strong a predictor of risk for cardiovascular problems than LDL cholesterol, the current gold standard. If these results are confirmed by other studies, this test will become a routine part of a medical exam.

Fibrinogin

An elevated fibrinogin level in the blood is positively correlated with heart disease and stroke. Fibrinogin adds to the viscosity (thickness or stickiness) of the blood and it enhances the formation of blood clots.[34] High fibrinogin levels are more common in men than women, smokers than nonsmokers, in those physically inactive compared to those who are active, and in those with high serum triglycerides compared to those with low levels.

Medical Contributions

Diagnostic Techniques

Diagnosing cardiovascular disease is becoming more sophisticated. Diagnosis begins with a medical examination and patient history. This procedure may be supplemented with a variety of tests that may confirm or refute the physician's suspicions of the presence of cardiovascular disease. Graded exercise tests (GXTs) using a motor-driven treadmill with the patient hooked to an electrocardiogram (ECG) have gained popularity in the last ten years or so. Such noninvasive tests use surface electrodes on the chest that are sensitive to the electrical actions of the heart. Mechanical abnormalities of the heart produce abnormal electrical impulses displayed on the ECG strip. These are read and interpreted by the physician.

The treadmill "road tests" the heart as it works progressively harder to meet the increasing oxygen requirement as the exercise protocol becomes more physically demanding. This test is more accurate for men than women. The gender difference in response to the treadmill test is not completely understood, but it is believed that women's breasts and extra fat tissue interfere with the reception of electrical impulses by the chest electrodes.

In some cases a thallium treadmill test is required because it is more sensitive; however, it is also much more expensive. This involves the injection of radioactive

thallium during the final minute of the treadmill test. Thallium is accepted, or taken up, by normal heart muscle but not by ischemic heart muscle. The absorption or nonabsorption of thallium can be seen on a television monitor. The thallium stress test increases diagnostic sensitivity to cardiovascular disease to approximately 90 percent.

Echocardiography is a safe, noninvasive technique that uses sound waves to determine the size of the heart, the thickness of the walls, and the function of the heart's valves. *Cardiac catheterization* is an invasive technique in which a slender tube is threaded from a blood vessel in an arm or leg into the coronary arteries. A liquid contrast dye that can be seen on X-ray film is injected into the coronary arteries. X-ray films are taken throughout the procedure to locate where and how severely the coronary arteries are narrowed.

Medical Treatment

A variety of drugs are used in cardiovascular therapies. These drugs lower blood pressure and cholesterol, minimize the likelihood of blood clotting, and dissolve clots during a heart attack.

Plain aspirin is proving to be an effective drug in the fight against heart disease. Low-dose (one-fourth of a regular aspirin) aspirin therapy consisting of a single daily dose for those who have heart disease reduces the risk of having another heart attack or dying from a subsequent attack. Low-dose aspirin therapy is more effective when supplemented with a "booster" dose of one whole aspirin on the first and fifteenth of each month. Also, regular aspirin use may prevent a first heart attack from occurring in apparently healthy people, but no one should take aspirin or any other drug without first consulting a physician.[65]

Surgical techniques have also affected the treatment of cardiovascular disease. *Coronary artery bypass surgery* is designed to shunt blood around an area of blockage by removing a leg vein and sewing one end of a leg vein into the aorta and the other end into a coronary artery below the blockage, thereby restoring blood flow to the heart muscle (figure 2-8).

The internal mammary arteries also are used for bypass grafts. Many authorities consider these to be the ideal grafts. There are two internal mammary arteries, but the one in the left side of the chest is preferable because it is nearer to the coronary arteries. Many surgeons would rather not use both arteries in the same patient because the diminished flow of blood to the chest impairs healing of the surgical wound. Also, fashioning bypass grafts out of these arteries is time-consuming precision surgery, and there are only two of them and they don't reach all parts of the heart. The advantage is that 95 percent of them remain open ten years after surgery.

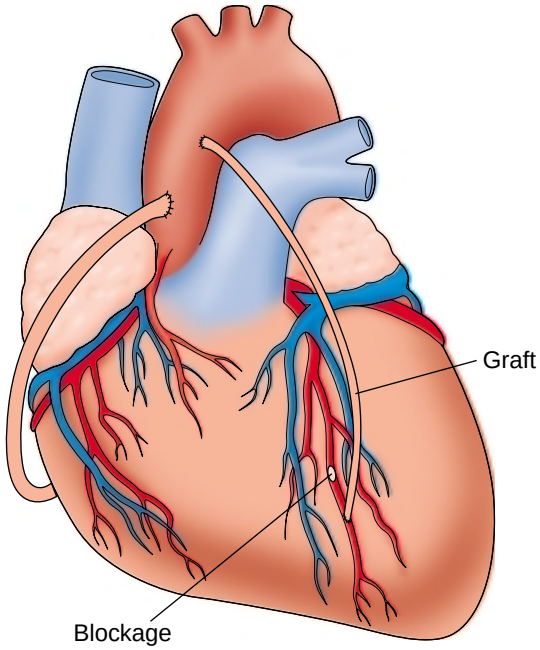


Figure 2-8 Coronary Bypass Graft

Balloon angioplasty uses a catheter with a doughnut-shaped balloon at the tip. The catheter is positioned at the narrow point in the artery, and the balloon is inflated, which cracks and compresses the plaque, stretches the artery wall, and widens the blood vessel to allow greater blood flow (figure 2-9). Laser angioplasty uses heat to burn away plaque if the catheter can be maneuvered into the correct position. This technology appears to be useful for patients with certain types of atherosclerotic narrowings or blockages. Coronary atherectomy, one of the newest techniques, uses a specially tipped catheter equipped with a high-speed rotary cutting blade to shave off plaque.

Catheterization techniques are also used to implant a coronary stent in a diseased artery. The stent is a flexible, metallic tube that functions like a scaffold to support the walls of diseased arteries, thus maintaining an open passage for blood flow (figure 2-10). Stents are positioned in such arteries by a catheter. When correctly positioned, a balloon inside the stent is inflated, causing the stent to expand. This action stretches the artery. Then the balloon is withdrawn, leaving the expanded stent behind to keep the blood vessel open.

This technique shows much promise, but there is a serious limitation associated with the procedure: It increases the risk of blood clots forming at the site of the stent. To counteract this risk, patients are given blood-thinning medications for two to three months following the stent's implantation, and then they are maintained on aspirin thereafter.

Artificial valves have been developed to replace defective heart valves, and these work well. In the summer

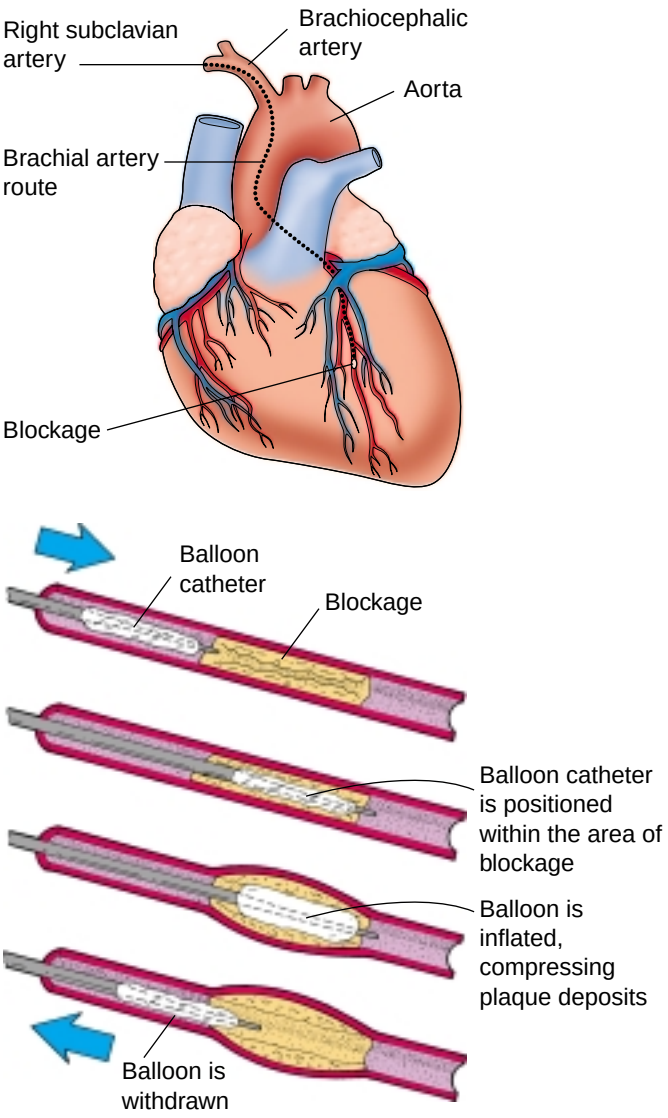


Figure 2-9 Balloon Angioplasty

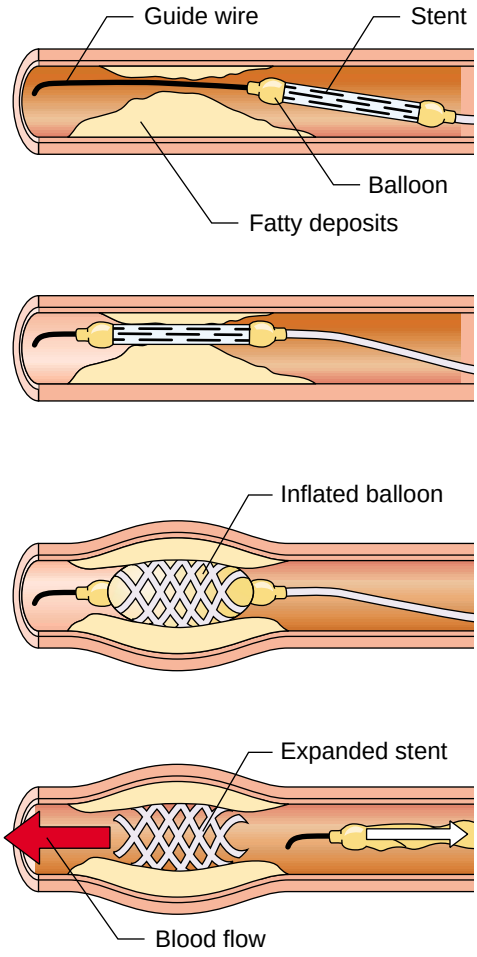


Figure 2-10 Coronary Stent

of 2001, the first successful self-contained mechanical heart was implanted in the chest of a male patient whose life expectancy was literally being measured in days. At this writing, three more patients, all of whom were extremely ill, received the same type of mechanical heart and all four patients are alive and improving. This device is considered experimental and its long-term success rate has yet to be demonstrated. However, it is a technological giant step forward compared to its predecessors, and with modifications, seems to have great potential for extending the lives of people whose hearts are so severely damaged that conventional medical treatments are ineffective.

Mechanical devices (left ventricular assist devices) have been used to aid the failing hearts of patients awaiting donor hearts. These devices take over the burden of pumping blood throughout the body and may keep patients alive for a month or more. Left ventricular assist devices suffer from the same power source problem as mechanical hearts.

Heart transplants have prolonged many lives. The outlook for patients has improved considerably because of the development and use of medicines to suppress the immune system in such patients and because of refinements in the prevention and early detection of the body's attempts to reject donor hearts. As a result, about 85 percent of transplant patients live longer than one year, and 65 percent survive for at least five years.[34] In 1968, twenty-three heart transplants were performed; in 1995, about 2350 were performed.

Candidates for transplants are those whose hearts are irreversibly damaged with disease that does not respond to conventional treatment. Without a new heart, these people will die. The main problems associated with heart transplantation are insufficient numbers of donors, the difficulty of procuring compatible donor hearts, and the constant threat of organ rejection by the recipient.

Summary

- Approximately 1.5 million heart attacks occur each year, and 500,000 of these result in death.
- The heart is two pumps in one; the pulmonary pump, which sends deoxygenated blood to the lungs, and the systemic pump, which sends oxygenated blood to all tissues of the body.
- Blood plasma is a clear, yellowish fluid that makes up about 55 percent of the blood. The remaining 45 percent consists of blood solids—red blood cells, white blood cells, and blood platelets.
- Strokes are caused by a thrombus, an embolus, or a hemorrhage.
- Coronary heart disease is a disease of the coronary blood vessels that bring nourishment and oxygen to the heart.
- Many of the risk factors for heart disease originate in childhood.
- The treatment of heart disease includes the development of appropriate lifestyle habits and medical intervention.
- The major risk factors that cannot be changed are age, male gender, and heredity.
- The major risk factors that can be changed are elevated cholesterol levels, hypertension, cigarette smoking, physical inactivity, and obesity.
- The other contributing risk factors are diabetes mellitus and stress.
- Cholesterol is a steroid that is essential for many bodily functions, but too much circulating in the blood creates a risk for cardiovascular disease.
- Low-density lipoproteins are associated with the development of atherosclerotic plaque.
- High-density lipoproteins protect the arteries from the formation of plaque.
- Blood pressure is the force exerted against the walls of the arteries as blood is pumped from the heart and travels through the circulatory system.
- *Hypertension* is the medical term for high blood pressure.
- Cigarette smoking may be the most potent of the risk factors associated with chronic illness and premature death.
- Involuntary, or passive, smoking is associated with premature disease and death.
- Obesity is a major risk factor that often coexists with many of the other risk factors for cardiovascular disease.
- Diabetes mellitus must be controlled to reduce the accompanying cardiovascular complications.
- Regular exercise significantly reduces the risk for type 2 diabetes mellitus.
- Stress predisposes a person to illness and may hasten the disease process.
- Elevated levels of homocysteine and of Lp (a) in the blood are proving to be important risk factors for heart disease.
- Medical research has made a significant contribution to reducing the incidence of heart disease through the development of sophisticated technological advances in diagnosis and treatment.

Review Questions

1. Define *pulmonary pump* and *systemic pump* and discuss the function of each.
2. Describe the advent of heart disease in the United States.
3. Identify and describe the causes of stroke.
4. What is coronary heart disease? Discuss the available treatment options.
5. What are the risk factors for heart disease and how are they categorized by the AHA?
6. What is cholesterol? LDL? HDL?
7. What is the relationship between total cholesterol and HDL?
8. What is essential hypertension?
9. What ingredients in cigarettes increase the risk for cardiovascular disease? Describe their effect on the heart and blood vessels.
10. What is the risk associated with smokeless tobacco products?
11. Defend the proposition that a moderate level of exercise improves health and increases longevity.
12. What are the cardiovascular complications of diabetes mellitus?
13. How does stress contribute to cardiovascular disease?
14. Can heart disease be prevented or at least delayed? Identify the lifestyle behaviors that may be involved in the process.
15. Can atherosclerosis be reversed? Cite evidence to support your answer.
16. What contributions has the medical profession made to decreasing the death rate from heart disease?

References

1. American Heart Association. 1998. *1999 Heart and Stroke Statistical update*. Dallas: American Heart Association.
2. Tufts University. 2000. Healthful eating habits really do protect your heart. *Tufts University Health & Nutrition Letter* 18(11): 2.
3. Squires, R. W. 2001. Coronary atherosclerosis. In *ACSM Resource Manual (*4th ed.), J. L. Roitman. Philadelphia: Lippincott Williams and Wilkins.
4. U.S. Dept. of Health and Human Services. 1996. *Physical Activity and Health: A Report of the Surgeon General.* Atlanta: U.S. Dept. of Health and Human Services, Centers for Disease Control and Prevention, National

Center for Chronic Disease Prevention and Health Promotion.
5. Kannell, W. B., et al. 1968. Epidemiology of acute myocardial infarction: The Framingham Study. *Medicine Today* 2:50.
6. Corbin, C. B., and R. P. Pangranzi. 1998. *Physical activity for children: a statement of guidelines.* NASE/AAHPERD.
7. U.S. Dept. of Health and Human Services. 2000. *Promoting Better Health for Young People Through Physical Activity and Sports*. Silver Springs, Md.: Centers for Disease Control.
8. Freedman, D. S., et al. 1999. The relation of overweight to cardiovascular risk factors among children and adolescents: the bogalusa heart study. *Pediatrics* 103:1175–1182.
9. Tufts University. 2000. More kids are getting diabetes—and more should be screened for it. *Tufts University Health & Nutrition Letter* 18(2):2.
10. U.S. Dept. of Health and Human Services. 2000. *Healthy People 2010: Understanding and Improving Health*. Washington, D.C.: U.S. Dept. of Health and Human Services, Government Printing Office.
11. Bartecchi, C. E., et al. 1994. The human cost of tobacco use. *New England Journal of Medicine* 330(13):987.
12. University of California at Berkeley. 1996. Up in smoke. *University of California at Berkeley Wellness Letter* 12(11):8.
13. Harvard Medical School. 2000. Cholesterol's effects evident in children. *Harvard Heart Letter* 10(10):7.
14. Publishers of the New England Journal of Medicine. Cholesterol concerns in the young. *HealthNews* 6(9):7.
15. Harvard Medical School. 2000. Heart attacks without chest pain: the danger is greater. *Harvard Heart Letter* 11(1):5–6.
16. Margolis, S., and S. C. Achuff. 1998. *Coronary Heart Disease.* Baltimore: The Johns Hopkins Medical Institutions.
17. Margolis, S., and G. Gerstenblith. 2001. *Coronary Heart Disease.* Baltimore: The Johns Hopkins Medical Institutions.
18. Margolis, S., and L. J. Cheskin. 1998. *Weight Control.* Baltimore: The Johns Hopkins Medical Institutions.
19. Walsh, J. 1998. Low fat, no fat, some high fat fat? Type of fat, not amount may be key. *Environmental Nutrition* 21(4):1.
20. "Heart-smart advice updated for 2000: more fat, but make it 'mono,' please." *Environmental Nutrition* 22(7):1, 4.
21. University of California at Berkeley. 1994. Fascinating facts. *University of California at Berkeley Wellness Letter* 10(6):1.
22. McArdle, W. D., F. I. Katch, and V. L. Katch. 1999. *Sports and Exercise Nutrition*. Philadelphia: Lippincott Williams and Wilkins.
23. Editors. 1984. The lipid research clinics coronary primary prevention trial results. I. Reduction in incidence of coronary heart disease. *Journal of American Medical Association* 251:351.
24. LaRosa, J. C., et al. 1990. The cholesterol facts: a summary of the evidence relating dietary facts, serum cholesterol, and coronary heart disease: a joint statement by the American Heart Association and the National Heart, Lung, and Blood Institute. *Circulation* 81:1721.
25. Steen, S. N., and G. Butterfield. 1998. Diet and nutrition. In *ACSM Resource Manual* (3rd ed.), ed. J. L. Roitman. Baltimore: Williams & Wilkins.
26. Harvard Medical School. 2000. Triglycerides and heart disease. *Harvard Heart Letter* 11(1):1–4.
27. University of California at Berkeley. 1998. The ups and downs of triglycerides. *University of California at Berkeley Wellness Letter* 14(3):4.
28. Thomas, T. R., and T. P. La Fontaine. 2001. Exercise, nutritional strategies and lipoproteins. In *ASCM Resource Manual* (4th ed.), ed. J. L. Roitman. Philadelphia: Lippincott Williams and Wilkins.
29. National High Blood Pressure Education Program. 1993. *The Fifth Report of the Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure, National Heart, Lung, and Blood Institute, National Institutes of Health,* NIH publication no. 93-1088. Bethesda, Md.: National Institutes of Health.
30. Harvard Medical School. 2001. A better way of measuring hypertension? *Harvard Health Letter* 26(4):3.
31. U.S. Dept. of Health and Human Services. 2000. *Healthy People 2010* (2nd ed.) with understanding and improving health and objectives for improving health, 2 vols. Washington, D.C.: U.S. Government Printing Office.
32. Tufts University. 1997. Meditation lowers blood pressure as well as drugs. *Tufts University Diet & Nutrition Letter* 14(111):3.
33. ACSM. 1993. Physical activity, physical fitness, and hypertension. *Medicine and Science in Sports and Exercise* 25(10):1.
34. Mooss, A., and N. F. Gordon. 2001. Conceptual basis for coronary artery disease risk factor assessment in clinical practice. In *ACSM Resource Manual* (4th ed.), ed. J. L. Roitman. Philadelphia: Lippincott Williams and Wilkins.
35. Centers for Disease Control and Prevention. 1999. Tobacco use—United States 1990–1999. *Journal of the American Medical Association* 292:2202–2204.
36. Hales, D. 2001. *An invitation to fitness and wellness.* Australia: Wadsworth.
37. Centers for Disease Control and Prevention. 1994. CDC surveillance summaries. *MMWR* 43(SS-3).
38. U.S. Dept. of Health and Human Services. 1988. *The Health Consequences of Smoking—Nicotine Addiction, a Report of the Surgeon General.* Rockville, Md.: U.S. Department of Health and Human Services.
39. American Cancer Society. 1997. *Cancer Facts and Figures—1997.* Atlanta, Ga.: American Cancer Society.
40. U.S. Dept. of Health and Human Services. 1998. *Progress Review—Tobacco, Prevention Report*. Washington, D.C.: Office of Disease Prevention and Health Promotion.
41. Powell, K. E. 1987. Physical activity and the incidence of coronary heart disease. *Annual Review of Public Health* 8:253.
42. Berlin, J. A., and G. A. Colditz. 1990. A meta-analysis of physical activity in the prevention of coronary heart disease. *American Journal of Epidemiology* 132:612.
43. Whaley, M. A., and L. A. Kaminsky. 2001. Epidemiology of physical activity, physical fitness, and selected chronic diseases. In *ACSM Resource Manual* (4th ed.), ed. J. L. Roitman. Baltimore: Lippincott, Williams and Wilkins.
44. Blair, S. N., et al. 1995. Changes in physical fitness and all-cause mortality: a prospective study of healthy and unhealthy men. *Journal of the American Medical Association* 273:1093–1098.
45. Blair, S. N., et al. 1989. Physical fitness and all-cause mortality—prospective

study of healthy men and women. *Journal of American Medical Association* 262:2395.
46. Manson, J. E., et al. 1992. The primary prevention of myocardial infarction. *New England Medical Journal* 326:1406.
47. Duncan, J. J., et al. 1995. Women walking for health and fitness. *Journal of American Medical Association* 273:402.
48. Pate, R. R., et al. 1995. Physical activity and public health. *Journal of American Medical Association* 273:402.
49. Lee, I. M., and R. S. Paffenbarger. 2000. Associations of light, moderate, and vigorous intensity physical activity with longevity: the Harvard alumni health study. *American Journal of Epidemiology* 151:293–299.
50. Pescatello, L. S., et al. 1994. Health care reform and the exercise professional. *ACSM Certified News* 4(2):1.
51. Editor. 1996. How to avoid adult onset diabetes. *Nutrition Action Health Letter* 23(7):3.
52. Campaigne, B. 2001. Exercise and diabetes mellitus. In *ACSM Resource Manual* (4th ed.), ed. J. L. Roitman. Philadelphia: Lippincott Williams and Wilkins.
53. Manson, J. E., et al. 1992. A prospective study of exercise and incidence of diabetes among U.S. male physicians. *Journal of American Medical Association* 268:63.
54. Hu, F. B., et al. 1999. Walking compared with vigorous physical activity and risk of type 2 diabetes in women: a prospective study. *Journal of the American Medical Association* 282:1433–1439.
55. Harvard Medical School. 2000. Anger and heart disease risk. *Harvard Heart Letter* 10(11):6–7.
56. Harvard University. 1998. Stress and atherosclerosis. *Harvard Health Letter* 23(4):8.
57. Harvard University. 1998. Grumpy old men: at risk for coronary disease? *Harvard Health Letter* 8(12):7.
58. Sime, W. W., and K. Hellweg. 2001. Stress and coping. In *ACSM Resource Manual* (4th ed.), ed. J. L. Roitman. Philadelphia: Lippincott Williams and Wilkins.
59. Ornish, D., et al. 1990. Can lifestyle changes reverse coronary heart disease? *The Lancet* 336:129.
60. Ornish, D., et al. 1993. Can lifestyle changes reverse atherosclerosis? Four-year results of the lifestyle heart trial. *Circulation* 88 (suppl.):2064.
61. Editors. 1997. Iron's link to heart disease. *HealthNews* 3(3):7.
62. Harvard University. 1997. Evidence mounts for heart disease marker. *Harvard Health Letter* 22(11):1.
63. Mayo Clinic. 1997. Homocysteine. *Mayo Clinic Health Letter* 15(12):7.
64. University of California at Berkeley. 1997. Ask the experts. *University of California at Berkeley Wellness Letter* 14(3):7.
65. Hennekens, C. H., M. L. Dyken, and V. Fuster. 1997. Aspirin as a therapeutic agent in cardiovascular disease—a statement for healthcare professionals from the American Heart Association. *Circulation* 96:2757.

Suggested Readings

Harvard University. 2001. Depression and heart disease. *Harvard Heart Letter* 11(8):3–5.

Discusses the differences between being depressed and actually suffering from clinical depression. Provides the major symptoms associated with clinical depression, discusses the major research that links heart disease with depression, and describes the methods by which depression is treated.

Editors of the New England Journal of Medicine. 2001. Better stroke prevention. *HealthNews* 7(4):1–2.

Discusses the causes of strokes and newer treatments that have evolved from the most recent research. Also, discusses the effects of a very old drug, aspirin, in the treatment of strokes. This article is accompanied by the comments of one of the associate editors whose medical specialty is emergency medicine.

University of California at Berkeley. 2001. A dozen ways to improve your walking workouts. *University of California at Berkeley Wellness Letter* 17(7):6.

For those who walk for heart health and other health-related reasons. Defines brisk walking and then gives twelve ways to make walking more fun and more effective.

Editors. 2001. Diabetes and heart disease: what you don't know can hurt you. *Heart Advisor* 4(3):3.

More than one-half of the diabetics in the United States are unaware that they have the disease so they are probably also unaware that diabetes is a major contributor to heart disease, stroke, kidney disease, blindness, and nerve damage. It is important to know the signs and symptoms in order to be treated early.

Editors. 2001. Strength training for heart patients. *Heart Advisor* 4(4):4–5.

Explains why strength training is important for heart patients. Discusses how heart patients can get started and then provides some tips for a successful resistance program.

Name _____ Date _____ Section _____

Assessment Activity 2-1

What's Your Heart Attack Risk?

Directions: By answering questions in the twelve items below, you can calculate your odds of having a heart attack within the next ten years. The test is based on data from four of the most extensive American studies of coronary risk. (The test is not accurate for people who already have a history of coronary disease. For definitive advice, ask your doctor.) Advice on improving your odds follows this test.

The Test

For every "yes" answer to items 1 through 9, add or subtract points as shown.

Question	Men	Women
1. Do you get little or no regular exercise?	Plus 2	Plus 6
2. Calculate your body mass index (BMI) as follows: Multiply your weight in pounds by 704. Divide the result by your height in inches. Divide that result by your height in inches again and round to the nearest whole number.		
Is your BMI from 21 to 24?	Plus 0	Plus 2
Is your BMI from 25 to 28?	Plus 2	Plus 3
Is your BMI 29 or over?	Plus 4	Plus 6
3. Do you have diabetes?	Plus 8	Plus 11
4. If you're an ex-smoker, did you quit in the past five years?	Plus 1	Plus 4
If you smoke, do you smoke fewer than 15 cigarettes a day?	Plus 2	Plus 8
Do you smoke 15 to 24 cigarettes a day?	Plus 4	Plus 15
Do you smoke more than 24 cigarettes a day?	Plus 6	Plus 18
5. Did either of your parents have a heart attack before age 60?	Plus 9	Plus 9
6. Do you take medicine to control blood pressure? (This is a sign that your pressure was once elevated.)	Plus 1	Plus 1
7. If you are a postmenopausal woman, are you currently taking estrogen alone?		Minus 5
Are you currently taking estrogen plus progestin?		Minus 3
If you don't currently take estrogen, did you previously take it (with or without progestin)?		Minus 2
8. Do you take low doses of aspirin at least every other day? (A low dose is between one quarter and one whole 325-mg tablet.)	Minus 4	Minus 4
9. Do you drink alcohol in moderation? ("Moderate" drinking is 2 to 14 drinks per week. A "drink" is 12 ounces of beer, 5 ounces of wine, or 1½ ounces of liquor.)	Minus 4	Minus 4
Add up your points so far. SUBTOTAL	_____	_____
Now calculate items 10 through 12, rounding to the nearest whole number.		
10. Multiply your systolic pressure (the higher number) by 0.14 if you are a man, by 0.15 if you are a woman.	Plus ___	Plus ___
11. Multiply your age by 0.51 if you are a man, by 0.8 if you are a woman.	Plus ___	Plus ___

12. Multiply your total cholesterol level by 0.07 if you are a man, by 0.06 if you are a woman. Multiply your HDL level by 0.25 if you are a man, by 0.3 if you are a woman. If you don't know your cholesterol levels and want to assume they're about average, you could substitute 205 for total cholesterol and 51 for HDL. Adults 20 and over should have cholesterol testing at least every 5 years.	Plus ___ Minus ___	Plus ___ Minus ___
Add up your points for items 10 through 12.	SUBTOTAL _____	_____
Add the two subtotals to get your score	TOTAL _____	_____

Probability* of Having a Heart Attack

Men

Score	1 Year	5 Years	10 Years
0–35	<0.1%	<0.4%	<1%
36–45	0.1–0.2%	0.4–1%	1–3%
46–55	0.2–0.6%	1–3%	3–7%
56–65	0.6–2%	3–8%	7–17%
66–70	2%	8–13%	17–27%
71–75	2–4%	13–20%	27–40%
76–80	4–6%	20–30%	40–56%

Women

Score	1 Year	5 Years	10 Years
0–60	<0.1%	<0.4%	<1%
61–70	0.1–0.2%	0.4–1%	1.3%
71–80	0.2–0.5%	1–3%	3–7%
81–85	0.5–1%	3–5%	7–12%
86–90	1%	5–8%	12–19%
91–95	1–2%	8–13%	19–29%
96–100	2–4%	13–20%	29–43%

*"Probability" indicates the percentage of people like yourself who will have a heart attack during the period cited. If your probability is 7% for the 10-year column, for example, it means that out of a random sampling of 100 people with the same score as yourself, seven will have a heart attack within a decade of today.

Name _____ Date _____ Section _____

Assessment Activity 2-2

A Case Study of Bill M.

Directions: To determine your understanding of cardiovascular health and wellness, read the following case study and answer the accompanying questions.

Bill, a 38-year-old man who is 5′8″ tall and weighs 205 lb, has the following history:

- His father died of a heart attack at age 48 years; his grandfather died of a heart attack at age 52 years.
- His cholesterol level is 256 mg/dl, LDL is 172 mg/dl, and HDL is 40 mg/dl.
- His blood pressure is consistently in the 150/95 range.
- He smokes one pack of cigarettes per day.
- He drinks six to eight brewed cups of coffee daily.
- He eats two eggs with bacon or sausage and buttered toast daily.
- Meat is a major part of supper; he skips lunch.
- His favorite snacks are ice cream, buttered popcorn, and salted peanuts.
- He occasionally plays tennis on Sunday afternoons.
- He owns his own business and often works 55 to 60 hours per week.

Answer the following:

What are Bill's risk factors for coronary heart disease? _____

Which of these can he control? _____

What suggestions can you give him regarding his current diet? _____

_____

What effect may a change in diet have on his coronary risk profile? _____

_____

What suggestions can you make regarding Bill's need for exercise, and how might a change in his activity level affect his coronary risk profile? _____

_____

What are the risks associated with obesity? _____

_____