TABLE 12-1 Selected Cardiovascular Drugs

Name	Use	Action	Adverse Effects
Nitroglycerin	Angina attacks and prophylaxis	Reduces cardiac workload, peripheral and coronary vasodilator	Dizziness, headache
Metoprolol (Lopressor)	Hypertension, angina, antiarrhythmic	Blocks beta-adrenergic receptors, slows heart rate	Dizziness, fatigue
Nifedipine (Adalat)	Angina, hypertension, peripheral vasodilator, antiarrhythmic	Calcium blockers, vasodilator	Dizziness, fainting, headache
Digoxin (Lanoxin)	Congestive heart failure and atrial arrhythmias	Slows conduction through AV node and increases force of contraction (cardiotonic) to increase efficiency	Nausea, fatigue, headache, weakness
Enalapril (Vasotec)	Hypertension	ACE inhibitor—blocks formation of angiotensin II and aldosterone	Headache, dizziness, hypotension
Furosemide (Lasix) hypertension	Edema with CHF, hypertension	Diuretic—increases excretion of water and sodium	Nausea, diarrhea, dizziness
Simvastatin (Zocor)	Hypercholesteremia (CHD)	Decreases cholesterol and LDL	Digestive discomfort
Warfarin (Coumadin)	Prophylaxis and treatment of thromboemboli	Anticoagulant—interferes with vitamin K in synthesis of clotting	Excessive bleeding (antidote: vitamin K)
ASA (aspirin)	Prophylaxis of thromboemboli	Prevents platelet adhesion, anti-inflammatory	Gastric irritation, allergy

CHD, Coronary heart disease; CHF, congestive heart failure; LDL, low-density lipoprotein.

artery. The basic problem is insufficient oxygen for the needs of the heart muscle.

A common cause of disability and death, coronary artery disease may ultimately lead to heart failure, serious dysrhythmias, or sudden death. It is the leading cause of death in men and women in the United States, causing approximately 385,000 deaths each year. Statistics for 2009 reveal that one in four deaths are the result of some form of heart disease, and the incidence for new or repeated heart attacks is 935,000 Americans. It is estimated that 13.2 million live with coronary artery disease in the United States. An additional 6 million are currently diagnosed with congestive heart failure (there is some overlap within these figures). The CDC reported that in 2008, high blood pressure was listed as a factor in 348,000 deaths and affects 68 million Americans. Males tend to develop heart disease at an earlier age than women, but women tend to have more complications likely due to later diagnosis. The current statistics show a decrease in numbers of individuals being diagnosed with heart disease, which many attribute to prevention awareness programs.

Arteriosclerosis and Atherosclerosis

Pathophysiology

Arteriosclerosis can be used as a general term for all types of arterial changes. It is best applied to degenerative changes in the small arteries and arterioles, commonly occurring in individuals over age 50 and those with diabetes. Elasticity is lost, the walls become thick and hard, and the lumen gradually narrows and may become obstructed. This leads to diffuse ischemia and necrosis in various tissues, such as the kidneys, brain, or heart.

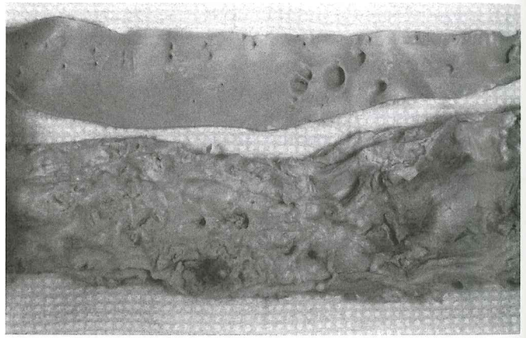


FIGURE 12-9 Comparison of a normal aorta with its smooth lining and patent openings into branching arteries (*top*) with an atherosclerotic aorta (*bottom*). Note the rough surface and blocked openings to branches. *(Courtesy of Paul Emmerson and Seneca College of Applied Arts and Technology, Toronto, Canada.)*

Atherosclerosis is differentiated by the presence of *atheromas,* plaques consisting of lipids, cells, fibrin, and cell debris, often with attached thrombi, which form inside the walls of large arteries. Note in Figure 12-9 how the unaffected artery is smooth, and the openings to branch arteries are clearly defined. By comparison, the atherosclerotic artery has a very rough, elevated surface, with loose pieces of plaque and thrombus, and the openings to branching arteries are blocked. Atheromas form primarily in the large arteries, such as the aorta and iliac arteries, the coronary arteries, and the carotid arteries, particularly at points of bifurcation,

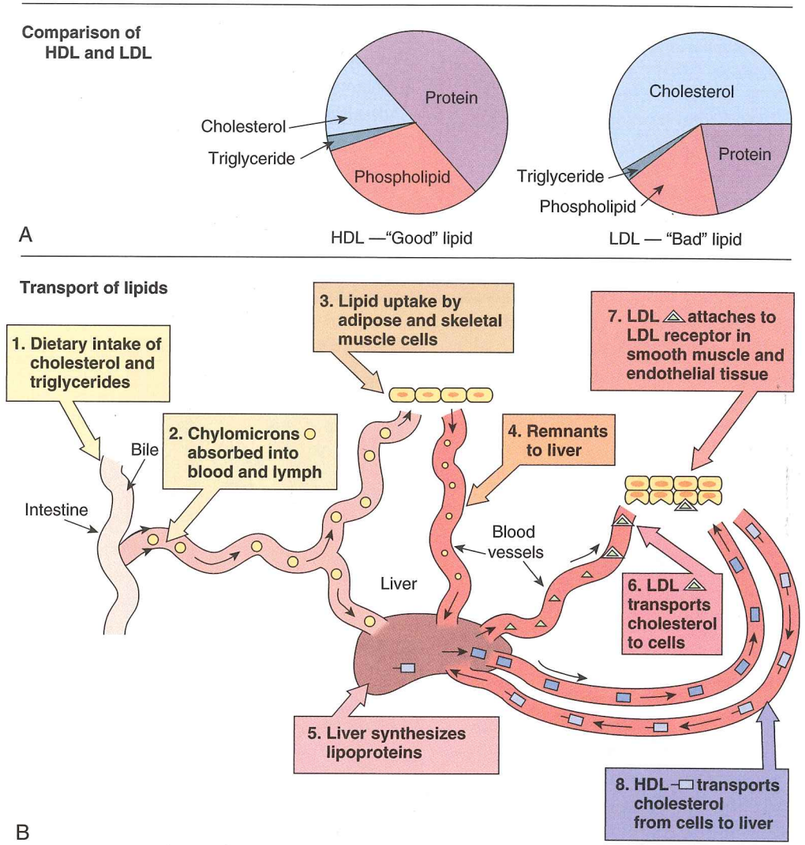


FIGURE 12-10 Composition of lipoproteins and transport of lipoproteins in blood.

where turbulent blood flow may encourage the development of atheromas.

Lipids or fats, which are usually transported in various combinations with proteins (lipoproteins), play a key role in this process (Fig. 12-10). Lipids, including cholesterol and triglycerides, are essential elements in the body and are synthesized in the liver; therefore they can never be totally eliminated from the body.

Analysis of serum lipids includes assessment of all the subgroups (total cholesterol, triglycerides, low-density lipoproteins, and high-density lipoproteins) because the proportions indicate the risk factor for the individual. The serum lipids of particular importance follow:

- Low-density lipoprotein, which has a *high* lipid content and transports cholesterol from the liver *to* cells, is the dangerous component of elevated serum levels of lipids and cholesterol. It is a major factor contributing to atheroma formation. Also, LDL binds to receptors, for example on the membranes of vascular smooth muscle cells and enters them; it is considered the "bad" lipoprotein that promotes atheroma formation.
- High-density lipoprotein is the "good" lipoprotein; it has a *low* lipid content and is used to transport cholesterol *away* from the peripheral cells to the liver, where it undergoes catabolism and excretion.

The process appears to begin with endothelial injury in the artery, often at a very young age. Endothelial injury causes inflammation in the area, leading to elevated C-reactive protein (CRP) levels. White blood cells, particularly monocytes and macrophages, and lipids accumulate in the intima, or inner lining, of the artery and in the media, or muscle layer. Smooth muscle cells proliferate or multiply (Fig. 12-11). Thus, a plaque forms and inflammation persists. Platelets adhere to the rough, damaged surface of the arterial wall, forming a thrombus and partial obstruction of the artery.

Lipids continue to build up at the site of arterial injury, along with fibrous tissue. Platelets adhere and release prostaglandins, which precipitate inflammation and vasospasm. This draws more platelets to aggregate

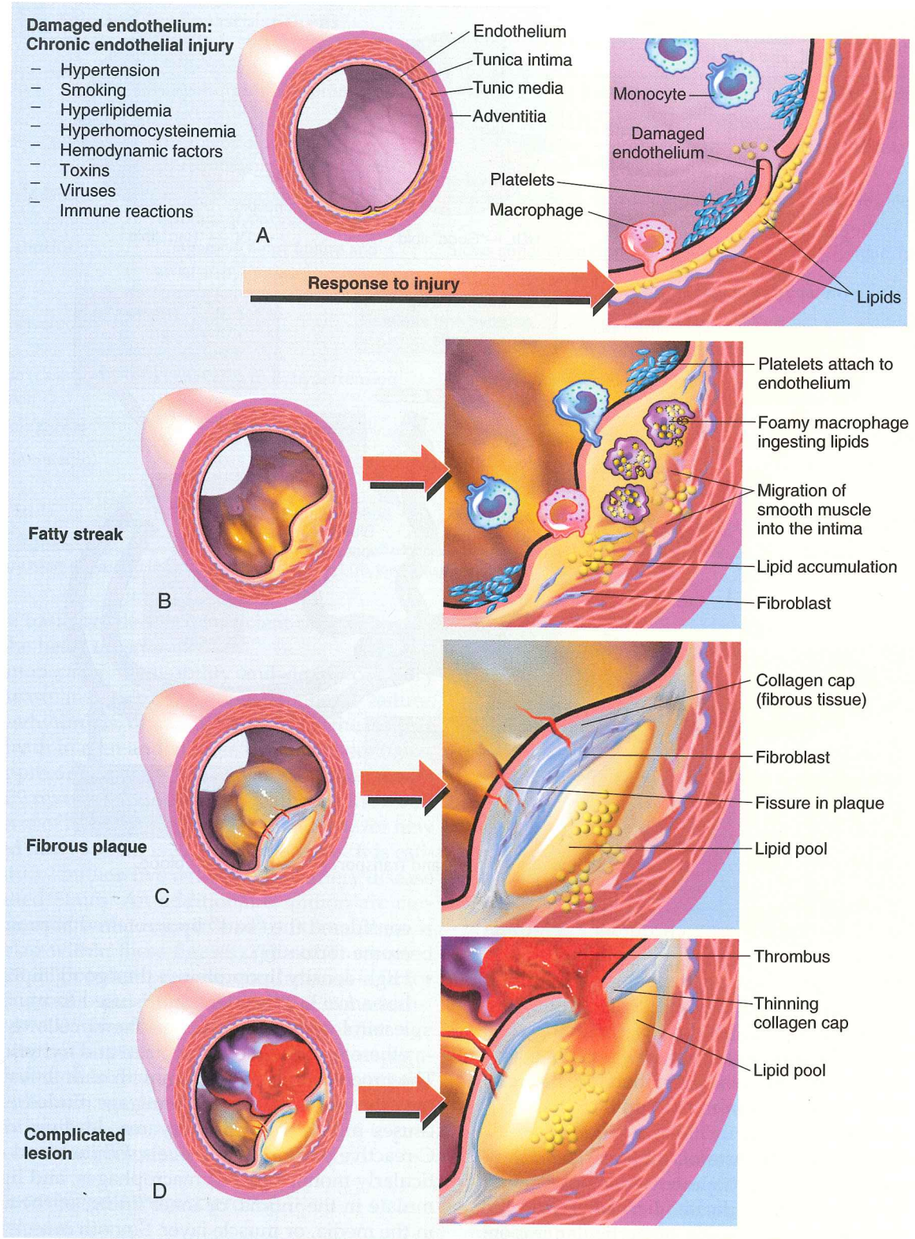


FIGURE 12-11 Progression of artherosclerosis. **A**, Damaged endothelium. **B**, Diagram of fatty streak and lipid core formation. **C**, Diagram of fibrous plaque. **D**, Diagram of completed lesion; thrombus is red; collagen is blue. Plaque is complicated by red thrombus deposition. *(From McCance KL, et al:* Pathophysiology, *ed 6, St. Louis, 2010, Mosby.)*

at the site, enlarging the thrombus. Arterial flow becomes more turbulent, again promoting thrombus formation. A vicious cycle persists. Blood flow progressively decreases as the lumen narrows. At some point, the plaque may ulcerate and break open. This may precipitate more inflammation or a thrombus may form at this site, resulting in total obstruction in a very short time. This may be the precipitating factor for myocardial infarction.

The atheroma also damages the arterial wall, weakening the structure and decreasing its elasticity. In time, atheromas may calcify, causing further rigidity of the wall. This process may lead to aneurysm, a bulge in the arterial wall (see Fig. 12-34), or to rupture and hemorrhage of the vessel.

Initially the atheroma manifests as a yellowish fatty streak on the wall. It becomes progressively larger, eventually becoming a large, firm projecting mass with an irregular surface on which a thrombus easily forms. As the atheroma increases in size and the coronary arteries are partially obstructed, angina (temporary myocardial ischemia) may occur; a total obstruction leads to myocardial infarction. Atheromas are also a common cause of strokes, renal damage, and peripheral vascular disease, which affects the legs and feet (Fig. 12-12).

Etiology

The cause of atherosclerosis appears to be multifactorial, and some of the factors are **synergistic**, enhancing the total effect. There are two groups of risk factors for atherosclerosis, one group that can be modified to some extent and one that cannot.

The factors that cannot be changed (nonmodifiable) include:

- Age, with atherosclerosis more common after age 40 years, particularly in men
- Gender, that is, women are protected by higher HDL levels until after menopause, when estrogen levels decrease.
- Genetic or familial factors seem to have a strong influence on serum lipid levels, metabolism, and cell receptors for lipids; some conditions are inherited, such as familial hypercholesterolemia, but family lifestyle factors may also have a role.
- The other group of predisposing factors are *modifiable*. These include factors such as:
  - Obesity or diets high in cholesterol and animal fat, which elevate serum lipid levels, especially LDL. The significant increase in obesity in children is of great concern with regards to a relative increase in cardiovascular disease in the coming years. The Centers for Disease Control and Prevention estimate that more than12.5 million children and adolescents under age 19 in the United States are obese and at risk of metabolic syndrome. Data collected in the United States for 2011 also indicated that 35.7% of adult men and women were clinically obese. Obesity is the primary indicator of *metabolic syndrome*, which is directly linked with the development of coronary artery disease in adulthood (see Chapter 23).
  - Cigarette smoking. The risk associated with smoking is directly related to the number of packs of cigarettes smoked per day. Smoking decreases

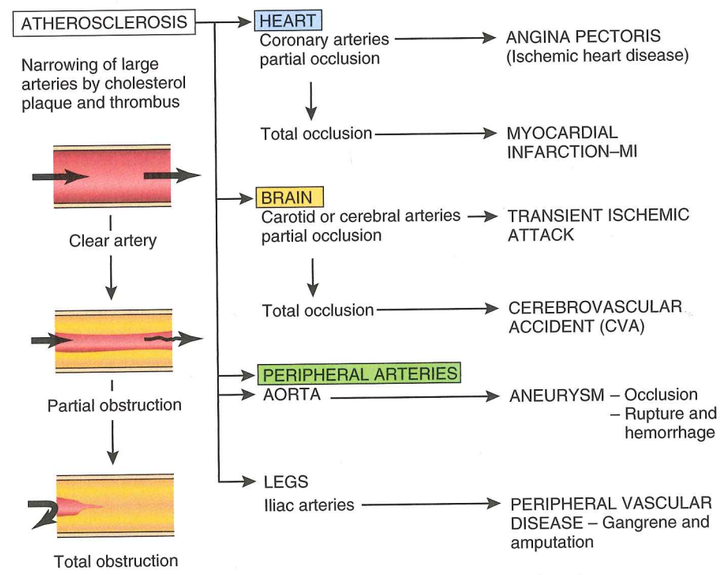


FIGURE 12-12 Possible consequences of atherosclerosis.

HDL, increases LDL, promotes platelet adhesion, and increases fibrinogen and clot formation as well as vasoconstriction.

- Sedentary lifestyle, which predisposes to sluggish blood flow and obesity. Exercise also reduces blood pressure and stress level and increases HDL while lowering LDL and cholesterol. Increasing numbers of children and adults report declining levels of physical activity.
- The presence of diabetes mellitus. In individuals with diabetes, especially those whose disease is not well controlled, serum lipid levels are increased and there is a tendency toward endothelial degeneration. The substantial increase in incidence and earlier onset in recent years of type 2 diabetes has increased the incidence of cardiovascular disease.
- Poorly controlled hypertension, which causes endothelial damage
- Combination of some oral contraceptives and smoking
- The combination of high blood cholesterol and high blood pressure in an individual has been shown to increase the risk of atherosclerosis and coronary artery disease significantly.

Diagnostic Tests

Serum lipid levels, including those of LDL and HDL, should be checked to identify the patient's risk and monitor the efficacy of treatment. Serum levels of high-sensitivity CRP indicate the presence of inflammation, indicating increased risk. However, CRP may be elevated due to other chronic inflammatory disease. Low CRP levels appear to indicate a low risk of developing cardiovascular disease. Exercise stress testing can be used for screening or to assess the degree of obstruction in arteries. Nuclear medicine studies can be used to determine the degree of tissue perfusion, the presence of collateral circulation, and the degree of local cell metabolism. To minimize risk and promote early diagnosis and treatment, the acceptable range for test results may be modified or lowered as new evidence becomes available.

APPLY YOUR KNOWLEDGE 12-2

Research is continuing to investigate the role of microbial infections in the damage of blood vessels. What are some portals of entry or potential sources from which bacteria may gain entry into the circulatory system and reach vessels such as the coronary arteries? What measures could be taken to prevent these types of infections and subsequent vessel damage?

Treatment

1. Losing weight and maintaining weight at healthy levels reduces the onset of metabolic syndrome as well as hypertension and atherosclerosis. Waist measurements below 35 in/87.5 cm in females and below 40 in/100 cm in males are considered healthy benchmarks.
2. Lowering serum cholesterol and LDL levels by substituting nonhydrogenated vegetable oils for trans fats and saturated fats has been well promoted as an effective means of slowing the progress of atherosclerosis. Vegetable oils containing linolenic acid and fish oils and other foods containing Omega 3 fatty acids are considered particularly useful. High dietary fiber intake also appears to decrease LDL levels. General weight reduction decreases the workload on the heart. Lipid-reducing (cholesterol or LDL) drugs such as probucol, clofibrate, and lovastatin may help in resistant cases. These measures may slow the progress of previously formed lesions and also prevent new ones.
3. Sodium intake should be minimized as well to control hypertension.
4. Control of primary disorders such as diabetes or hypertension is important.
5. Cease smoking.
6. Exercise appropriate for age and health status will promote collateral circulation and reduce LDL levels.
7. If thrombus formation is a concern, oral anticoagulant therapy may be required; this may include a small daily dose of ASA or warfarin (Coumadin).
8. When atheromas are advanced, surgical intervention (percutaneous transluminal coronary **angioplasty** may be required to reduce obstruction by means of invasive procedures requiring cardiac catheterization. The catheter contains an inflatable balloon that flattens the atheroma. Newer techniques use laser angioplasty, a laser beam, and fiberoptic technology with a catheter. The high-energy laser causes the obstruction to disintegrate into microscopic particles that are removed by macrophages. There appears to be less risk of recurrence with this method. Stents, small tube-like structures, may be inserted into arteries after angioplasty, to maintain an opening. Surgery such as coronary artery bypass grafting (CABG) to reroute blood flow around the obstruction, using veins or the left internal mammary artery for a graft, appears to have an improved long-term prognosis (Fig. 12-13). A graft can also be placed around an obstructed aorta. Less invasive means of CABG are currently being used in some patients.

THINK ABOUT 12-5

a. Explain three ways of reducing the risk of atherosclerosis.
b. Give three common locations of atheromas.
c. Describe two ways in which an artery can become totally obstructed.

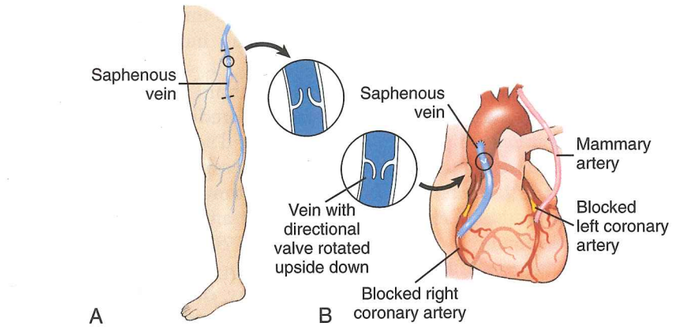


FIGURE 12-13 Coronary artery bypass graft (CABG) surgery. *(From Shiland BJ:* Medical Terminology and Anatomy for ICD-10 Coding. *St. Louis, 2012, Mosby.)*

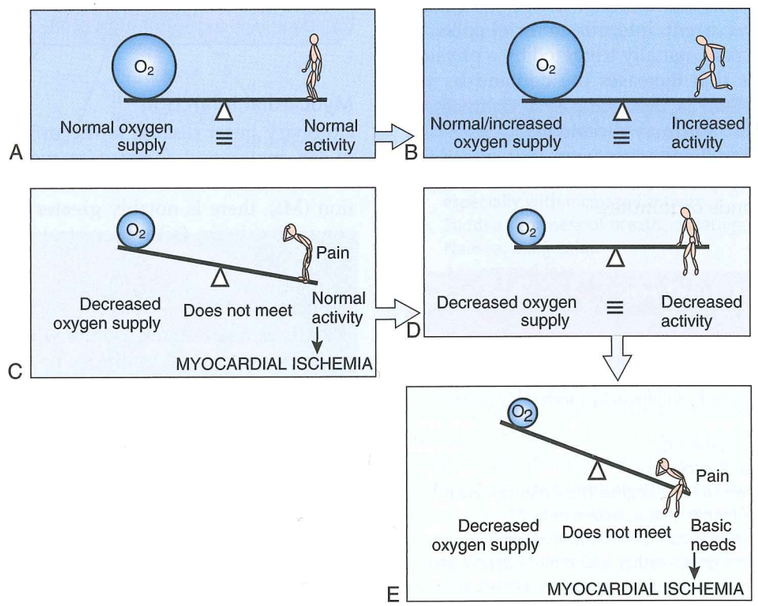


FIGURE 12-14 Angina—an imbalance between oxygen supply and demand.

Angina Pectoris

Pathophysiology

Angina, or chest pain, occurs when there is a deficit of oxygen to the heart muscle. This can occur when the blood or oxygen supply to the myocardium is impaired, when the heart is working harder than usual and needs more oxygen, or when a combination of these factors is present (Fig. 12-14). Usually the heart can adapt its blood supply to its own needs by vasodilation (**autoregulation**) unless the vessel walls are damaged or cannot relax. The reduced blood supply may be due to partial obstruction by atherosclerosis or spasm in the coronary arteries. When the supply and demand for oxygen are marginally balanced, an increase in cardiac demand with any physical or emotional exertion can cause a relative deficit of oxygen to the myocardium.

Chest pain may occur in a variety of patterns: classic or exertional angina; variant angina, in which vasospasm occurs at rest; and unstable angina, a more serious form. Unstable angina refers to prolonged pain at rest and of recent onset, perhaps the result of a break in an atheroma. This may precede a myocardial infarction. Most commonly, an episode of anginal pain occurs when the demand for oxygen increases suddenly, with exertion. In most cases, no permanent damage to the myocardium results from angina unless the episodes are frequent, prolonged, and severe.

Etiology

Insufficient myocardial blood supply is associated with atherosclerosis, arteriosclerosis, vasospasm (a localized contraction of arteriolar smooth muscle), and myocardial hypertrophy, in which the heart has outgrown its blood supply. Severe anemias and respiratory disease can also cause an oxygen deficit. Increased demands for oxygen can arise in circumstances such as tachycardia associated with hyperthyroidism or the increased force of contractions associated with hypertension.

Precipitating factors of angina attacks are related to activities that increase the demands on the heart, such as running upstairs, getting angry, respiratory infection with fever, exposure to weather extremes or pollution, or eating a large meal.

Signs and Symptoms

Angina occurs as recurrent, intermittent brief episodes of substernal chest pain, usually triggered by a physical or emotional stress that increases the demand by the heart for oxygen. Pain is described as a tightness or pressure in the chest and may radiate to the neck and left arm. Often pallor, diaphoresis (excessive sweating), and nausea are also present. Attacks vary in severity and last a few seconds or minutes.

EMERGENCY TREATMENT FOR ANGINA ATTACK

1. Let patient rest, stop activity.
2. Seat patient in an upright position.
3. Administer nitroglycerin sublingually (preferably patient's own supply).
4. Check pulse and respiration.
5. Administer oxygen if necessary.
6. For a patient known to have angina, the American Heart Association recommends that a second dose of nitroglycerin be given if pain persists more than 5 minutes. After three doses within a 10-minute period and no pain relief, the pain should be treated as a heart attack. Call for assistance and emergency medical intervention.
7. For a patient without a history of angina, emergency medical aid should be sought after 2 minutes without pain relief.

Treatment

Anginal pain is usually quickly relieved by rest and the administration of coronary vasodilators, such as nitroglycerin. The drug may relieve vasospasm in the coronary arteries but primarily acts to reduce systemic resistance, thus decreasing the demand for oxygen. Many patients carry nitroglycerin (in the correct dosage) with them at all times to be administered sublingually in an emergency (the tablet is not swallowed but dissolves under the tongue and enters the blood directly for instant effect). If chest pain persists following treatment, it is important to seek hospital care because the pain may indicate the presence of a myocardial infarction.

It is important to determine the history of angina and the factors predisposing to attacks to minimize their frequency and severity. The avoidance of sudden physical exertion, especially in cold or hot weather, marked fatigue, or strong emotional incidents, is recommended. Antianxiety and stress reduction techniques may be necessary in certain situations. Some clients use nitroglycerin in the form of a topical ointment, a skin patch, a nasal spray, or oral tablets (isosorbide) on a regular basis to reduce the number of attacks.

THINK ABOUT 12-6

a. Describe the characteristics of anginal pain.

Myocardial Infarction

Coronary heart disease is the primary cause of death in US males and females as well as a major cause of disability. For those who survive a myocardial infarction (MI), there is notably greater risk of a second MI, congestive heart failure, or stroke occurring within a short time.

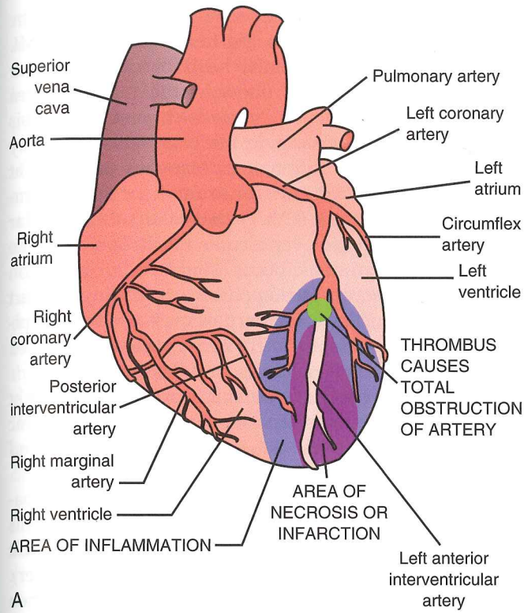
Pathophysiology

An MI, or heart attack, occurs when a coronary artery is totally obstructed, leading to prolonged ischemia and cell death, or infarction, of the heart wall (Fig. 12-15A,*B*). The most common cause is atherosclerosis, usually with thrombus attached (see previous discussion under Coronary Artery Disease).

Infarction may develop in three ways:

1. The thrombus may build up to obstruct the artery.
2. Vasospasm may occur in the presence of a partial occlusion by an atheroma leading to total obstruction.
3. Part of the thrombus may break away, forming an embolus or emboli that flow(s) through the coronary artery until lodging in a smaller branch, blocking that vessel (see Fig. 12-11). Most infarctions are transmural; that is, all three layers of the heart are involved. The majority involve the critical left ventricle. The size and location of the infarct determine the severity of the damage.

At the point of obstruction the heart tissue becomes necrotic, and an area of injury, inflammation, and ischemia develops around the necrotic zone (see Fig. 12-15*A*). With cell destruction, specific enzymes are released from the myocardium into tissue fluid and blood; these enzymes appear in the blood and are diagnostic. The functions of myocardial contractility and conduction are lost quickly as oxygen supplies are depleted. If the blood supply can be restored in the first 20 to 30 minutes, irreversible damage may be prevented. After 48 hours, the inflammation begins to subside. If sufficient blood supply has been maintained in the outer area of inflammation, function can resume. On the other hand, if treatment has not been instituted quickly or is not effective, the area of infarction may increase. Because the myocardial fibers do not regenerate, the area of necrosis is gradually replaced by fibrous (nonfunctional) tissue, beginning around the seventh day. It may take 6 to 8 weeks to form a scar, depending on the size of the infarcted area.



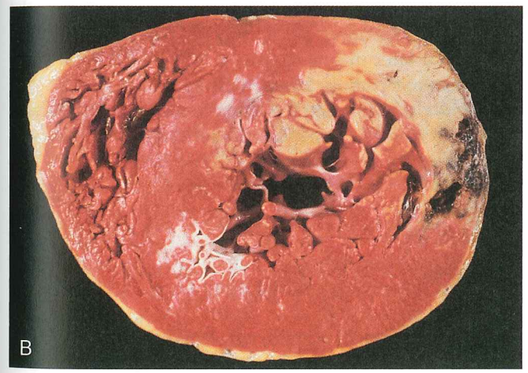


FIGURE 12-15 **A**, Damage caused by myocardial infarction. **B**, Acute myocardial infarct of the posterolateral left ventricle, shown by lack of stain in the necrotic area. Note the dark area on the right indicating hemorrhage and ventricular rupture. The white area on the lower left indicates an old infarct. *(From Kumar V, Abbas AK, Fausto M:* Robbins and Cotran Pathologic Basis of Disease, *ed 7, Philadelphia, 2005, Saunders.)*

WARNING SIGNS OF HEART ATTACK

(These signs may be intermittent initially.)

1. Feeling of pressure, heaviness, or burning in the chest, especially with increased activity
2. Sudden shortness of breath, sweating, weakness, fatigue
3. Nausea, indigestion
4. Anxiety and fear

Signs and Symptoms

It is important to seek a diagnosis and medical care as soon as these signs occur to prevent permanent heart damage or death. If thrombolytic therapy is administered within 20 minutes of the onset, blood flow can be restored, and no permanent damage occurs in the heart. Many paramedic teams can now administer fibrinolytic drugs, saving many lives. Automated external defibrillators (AEDs) may be found in many public buildings to be used in event of cardiac arrest.

As a myocardial infarction develops, the following manifestations become more evident:

- Pain: Sudden substernal chest pain that radiates to the left arm, shoulder, jaw, or neck is the hallmark of myocardial infarction. The pain is usually described as severe, steady, and crushing, and no relief occurs with rest or vasodilators. In some cases, pain is not present (*silent* myocardial infarction) or is interpreted as gastric discomfort. Women often report a milder pain, more like indigestion.
- Pallor and diaphoresis, nausea, dizziness and weakness, and dyspnea
- Marked anxiety and fear
- Hypotension: Hypotension is common, and the pulse is rapid and weak as cardiac output decreases and shock develops.
- Low-grade fever

Diagnostic Tests

1. Typical changes occur in the ECG during the course of a myocardial infarction, which confirm the diagnosis and assist in monitoring progress.
2. Serum enzymes and isoenzymes released from necrotic cells also follow a typical pattern, with elevations of lactic dehydrogenase (LDH-1), aspartate aminotransferase (AST, formerly SGOT), and creatine phosphokinase with M and B subunits (CK-MB or CPK-2) (Fig. 12-16). The particular isoenzymes, LDH-1 and CK-MB, are more specific for heart tissue.
3. Serum levels of myosin and cardiac troponin are elevated a few hours after MI, providing for an earlier confirmation. A rise in cardiac troponin levels is considered most specific for myocardial tissue damage.
4. Serum electrolyte levels, particularly potassium and sodium, may be abnormal.
5. Leukocytosis and an elevated CRP and erythrocyte sedimentation rate are common, signifying inflammation. There is evidence that high blood levels of CRP indicate a more marked inflammatory response, with plaques more inclined to rupture, thrombus to form, and ultimately a more severe heart attack.
6. Arterial blood gas measurements will be altered particularly if shock is pronounced.
7. Pulmonary artery pressure measurements are also helpful in determining ventricular function.

Complications

The following are common occurrences immediately following the infarction and also at a later time:

- Sudden death shortly after myocardial infarction occurs frequently (in about 25% of patients), usually owing to ventricular arrhythmias and fibrillation (see next section, Cardiac Dysrhythmias). This is the major cause of death in the first hour after an MI. One type of dysrhythmia, heart block, may occur when the conduction fibers in the infarcted area can no longer function. Second, an area of necrosis and inflammation outside the conduction pathway may stimulate additional spontaneous impulses at an **ectopic** site, causing, for example, *premature ventricular contractions* (PVCs) that lead to ventricular tachycardia and/or ventricular fibrillation. In some cases, dysrhythmias occur later as inflammation spreads to the conduction pathways, leading to heart block. Conduction irregularities may also be precipitated by hypoxia, by increased potassium released from necrotic cells, acidosis, and drug toxicities.
- Cocaine users may suffer fatal heart attacks, even at a young age, because cocaine interferes with cardiac conduction as well as causing vasospasm and occlusion.
- *Cardiogenic shock* may develop if the pumping capability of the left ventricle is markedly impaired. This greatly reduces cardiac output, leading to significant hypoxia (see the topic of shock later in this chapter).
- *Congestive heart failure* is a common occurrence when the contractility of the ventricle is reduced and stroke volume declines. This may occur a few days after the MI or much later as activity is resumed. (CHF is covered later in this chapter.)

Less frequent complications include:

- *Rupture* of the necrotic heart tissue, particularly in patients with a ventricular aneurysm or those with significant hypertension. This usually develops 3 to 7 days after the MI when the necrotic tissue is breaking down.
- *Thromboembolism* may result from a thrombus that develops over the infarcted surface inside the heart *(mural thrombus)* and eventually breaks off. If originating in the left side of the heart, the embolus will travel to the brain or elsewhere in the body, whereas if the source is the right ventricle, the result will be a pulmonary embolus. (A thrombus may form in the deep leg veins due to immobility and poor circulation and also cause a pulmonary embolus [see Chapter 13].)

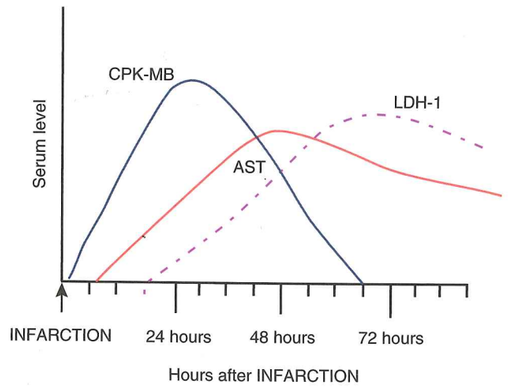


FIGURE 12-16 Serum enzymes and isoenzyme levels with myocardial infarction. *AST*, aspartate aminotransferase; *CPK-MB*, creatine phosphokinase containing M and B subunits; *LDH-1*, lactate dehydrogenase.

Treatment

As mentioned, paramedics in many areas are equipped to provide immediate life-saving treatment. Keeping the patient calm, oxygen therapy, and analgesics such as morphine for pain relief are the usual treatment modalities. Anticoagulants such as heparin or warfarin may be used, or the newer thrombolytic agents, including streptokinase, urokinase, or tissue plasminogen activator, may be administered immediately to reduce the clot in the first hours. Depending on the individual circumstances, medication to reduce dysrhythmias, defibrillation, or a pacemaker (which may be temporary) may be required. Drugs, such as digoxin, support the heart function. Specific measures may be required if shock or congestive heart failure develops. Bypass surgery may be performed. Other specific drugs are mentioned in the general treatment section.

Cardiac rehabilitation programs that offer individualized plans for regular exercise, dietary modifications, and stress reduction are useful following recovery. A schedule for the resumption of normal activities, such as climbing stairs, returning to work, and resuming sexual activities, can be established. Appropriate medications to treat any predisposing condition, as well as those to minimize the effects of the MI, are prescribed. Frequently a low dose of ASA is recommended to reduce the risk of further thrombi. The American Heart Association has organized a hospital-based program "Get With The Guidelines" to provide optimum treatment to all patients and promote patient compliance after discharge, thus improving outcomes.

The prognosis depends on the site and size of the infarct, the presence of collateral circulation, and the time elapsed before treatment. The mortality in the first year is 30% to 40% and results from complications or recurrences.

THINK ABOUT 12-7

a. Compare the causes of the chest pain that occurs with angina to that which occurs with myocardial infarction.
b. Explain why an embolus may cause a larger infarction than an atheroma with thrombus.
c. List the tests that confirm a diagnosis of myocardial infarction.
d. Explain why part of the myocardium is nonfunctional following myocardial infarction.
e. Suggest several treatment measures that may minimize the area of infarction. Why is time a critical element in treatment of MI?

Cardiac Dysrhythmias (Arrhythmias)

Deviations from normal cardiac rate or rhythm may result from damage to the heart's conduction system or systemic causes such as electrolyte abnormalities (see Chapter 2 for the effects of potassium imbalance), fever, hypoxia, stress, infection, or drug toxicity. Interference with the conduction system may result from inflammation or scar tissue associated with rheumatic fever or myocardial infarction. The ECG provides a method of monitoring the conduction system and detecting abnormalities (see Fig. 12-16). Holter monitors record the ECG over a prolonged period as a patient follows normal daily activities.

Dysrhythmias reduce the efficiency of the heart's pumping cycle. A slight increase in heart rate increases cardiac output, but a very rapid heart rate prevents adequate filling during diastole, reducing cardiac output, and a very slow rate also reduces output to the tissues, including the brain and the heart itself. Irregular contractions are inefficient because they interfere with the normal filling and emptying cycle. Among the many types of abnormal conduction patterns that exist, only a few examples are considered here.

Sinus Node Abnormalities

The SA node is the pacemaker for the heart, and its rate can be altered.

- *Bradycardia* refers to a regular but slow heart rate, less than 60 beats per minute; it often results from vagal nerve or parasympathetic nervous system stimulation. An exception occurs in athletes at rest, who may have a slow heart rate because they are conditioned to produce a large stroke volume.
- Tachycardia is a regular rapid heart rate, 100 to 160 beats per minute (Fig. 12-17). This may be a normal response to sympathetic stimulation, exercise, fever, or stress, or it may be compensation for decreased blood volume.
- *Sick sinus syndrome* is a heart condition marked by alternating bradycardia and tachycardia and often requires a mechanical pacemaker.

Atrial Conduction Abnormalities

Atrial conduction abnormalities are the most common dysrhythmias, (i.e., clinical abnormalities of heart conduction). Hospital admissions for paroxysmal atrial fibrillation have increased by 66% primarily due to aging of the population and an increase in the prevalence of coronary heart disease.

Premature atrial contractions or *beats* (PAC/PAB) are extra contractions or *ectopic* beats of the atria that usually arise from a focus of irritable atrial muscle cells outside the conduction pathway. They tend to interfere with the timing of the next beat. Ectopic beats may also develop from *re-entry* of an impulse that has been delayed in damaged tissue and then completes a circuit to re-excite the same area before the next regular stimulus arrives. Sometimes people feel *palpitations,* which are rapid or irregular heart contractions that often arise from excessive caffeine intake, smoking, or stress.

Atrial *flutter* refers to an atrial heart rate of 160 to 350 beats per minute, and atrial *fibrillation* is a rate over 350 beats per minute. With flutter, the AV node delays conduction, and therefore the ventricular rate is slower. A pulse deficit may occur because a reduced stroke volume is not felt at the radial pulse. Atrial fibrillation causes pooling of blood in the atria and is treated with anticoagulant medications to prevent clotting and potential cerebrovascular accident (stroke). Ventricular filling is not totally dependent on atrial contraction, and therefore these atrial arrhythmias are not always symptomatic unless they spread to the ventricular conduction pathways.