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].)

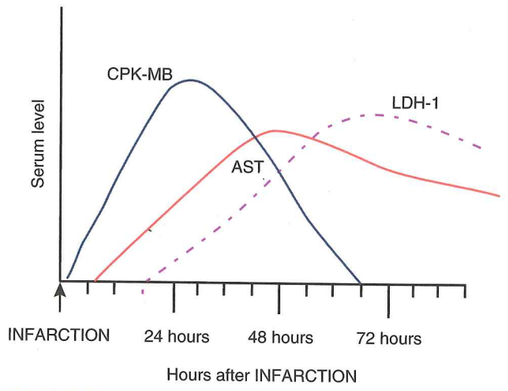

FIGURE 12-16 Serum enzymes and isoenzyme levels with myocardial infarction. *AST*, aspartate aminotransferase; *CPK-MB*, creatine phosphokinase containing M and B subunits; *LDH-1*, lactate dehydrogenase.

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.

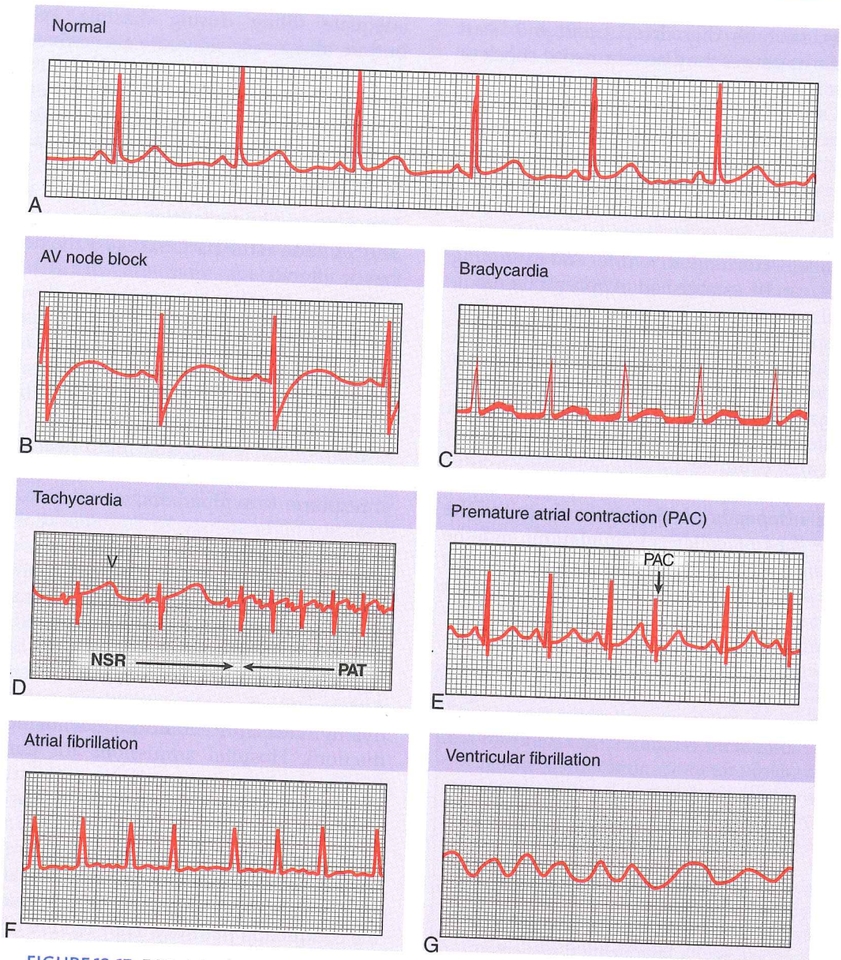

FIGURE 12-17 ECG strip chart recordings. **A**, Normal ECG. **B**, AV node block. Very slow ventricular contraction (25 to 45 beats/min at rest); P waves widely separated from peaks of QRS complexes. **C**, Bradycardia. Slow heart rhythm (less than 60 beats/min); no disruption of normal rhythm pattern. **D**, Tachycardia. Rapid heart rhythm (greater than 100 beats/min); no disruption of normal rhythm pattern. NSR, Normal sinus rhythm; PAT, paroxysmal (sudden) atrial tachycardia. **E**, Premature atrial contraction (PAC). Unexpected, early P wave that differs from normal P waves; PR interval may be shorter or longer than normal; normal QRS complex; more than 6 PACs per minute may precede atrial fibrillation. **F**, Atrial fibrillation. Irregular, rapid atrial depolarizations; P wave rapid (greater than 300/min) with irregular QRS complexes (150 to 170 beats/min). **G**, Ventricular fibrillation. Complete disruption of normal heart rhythm. *(From Patton KT, Thibodeau GA:* Anatomy & Physiology, *ed 8, St. Louis, 2013, Mosby.)*

Atrioventricular Node Abnormalities—Heart Blocks

Heart block occurs when conduction is excessively delayed or stopped at the AV node or bundle of His.

Partial blocks may be:

1. First-degree, in which the conduction delay prolongs the PR interval, the time between the atrial and ventricular contractions
2. Second-degree, in which a longer delay leads periodically to a missed ventricular contraction
3. *Total,* or third-degree, blocks occur when there is no transmission of impulses from the atria to the ventricles. The ventricles contract spontaneously at a slow rate of 30 to 45 beats per minute, totally independent of the atrial contraction, which continues normally (see Fig. 12-17C). In this case, cardiac output is greatly reduced, sometimes to the point of fainting (**syncope**), causing a *Stokes-Adams* attack or cardiac arrest.

Ventricular Conduction Abnormalities

1. *Bundle branch block* refers to interference with conduction in one of the bundle branches. This usually does not alter cardiac output but does appear on the ECG as a wide QRS wave.
2. Ventricular *tachycardia* is likely to reduce cardiac output because the filling time is reduced and the force of contraction is reduced.
3. In ventricular *fibrillation* the muscle fibers contract independently and rapidly (uncoordinated quivering) and therefore are ineffective in ejecting blood (see Fig. 12-17*D*). The lack of cardiac output causes severe hypoxia in the myocardium, and contraction ceases.
4. *Premature ventricular contractions (PVCs)* are additional beats arising from a ventricular muscle cell or ectopic pacemaker. Occasional PVCs do not interfere with heart function, but increasing frequency, multiple ectopic sites, or paired beats are of concern because ventricular fibrillation can develop from these, leading to cardiac arrest.

A summary of these abnormalities may be found in Table 12-2.

Treatment of Cardiac Dysrhythmias

The cause of the dysrhythmia should be determined and treated. Easily correctable problems include those caused by drugs, such as digitalis toxicity, bradycardia due to beta-blockers, or potassium imbalance related to some diuretics. In these examples a change in dosage or drug may eliminate the dysrhythmia.

Antiarrhythmic drugs are effective in many cases of heart damage. Beta$_1$-adrenergic blockers and calcium channel blockers are discussed earlier in this chapter. Atrial dysrhythmias often respond to digoxin, which slows AV node conduction and strengthens the contraction, thus increasing efficiency.

Sinoatrial nodal problems or total heart block requires a pacemaker, either a temporary attachment or a device that is permanently implanted in the chest; such a device provides electrical stimulation through electrodes directly to the heart muscle (Fig. 12-18). Pacemakers may stimulate a heart contraction only as needed or take over total control of the heart rate. Caution is required with the use of some electronic equipment when certain types of pacemakers are in place. Serious life-threatening dysrhythmias may require the use of defibrillators and cardioversion devices that transmit an electric shock to the heart to interrupt the disorganized electrical activity that occurs with fibrillation, for example, and then

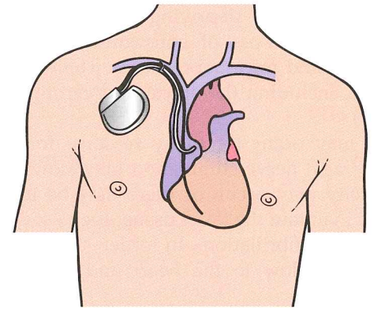

FIGURE 12-18 Permanent pacemaker implanted in the chest. *(From deWit S, Kumagai C:* Medical-Surgical Nursing: Concepts and Practice, *Philadelphia, 2013, Saunders.)*

TABLE 12-2 Cardiac Dysrhythmias

Name	Conduction change	Effect
Bradycardia	Rate regular, slower than 60/minute	Stroke volume increased Possibly reduced cardiac output
Tachycardia	Rate regular, fast, 100-160/minute	Possibly reduced cardiac output
Atrial flutter	Rate 160-350/min	Less filling time Often reduced cardiac output
Fibrillation	Rate over 300/min; uncoordinated muscle contractions	No filling, no output—cardiac standstill
Premature ventricular contractions	Additional ectopic beats	May induce fibrillation
Bundle Branch Block	Delayed conduction in one bundle branch, wide QRS wave	No effect
Heart Block 1° (partial)	Delays conduction in A-V node, prolongs PR interval	No effect
Heart Block 2° (partial)	Delays conduction in A-V node, gradually increasing PR until one contraction missed	Periodic decrease in output
Total Heart Block	No conduction in A-V node, ventricles slowly contract independent of atrial contraction	Marked decrease in output, causing syncope

allows the SA node to take control again, returning the heart to sinus rhythm. These devices may be external or implanted internally. Newer devices have electronic memory, which can be downloaded to assess cardiac function and efficiency of the device.

THINK ABOUT 12-8

a. Compare PVCs, atrial flutter, atrial fibrillation, and total heart block.
b. Using one type of dysrhythmia as an example, explain how cardiac output may be reduced.
c. Explain the absence of peripheral pulses in ventricular fibrillation.

Cardiac Arrest or Standstill (Asystole)

Cardiac arrest is the cessation of all activity in the heart. There is no conduction of impulses, and the ECG shows a flat line. Lack of contractions means that no cardiac output occurs, thus depriving the brain and heart itself of oxygen. Loss of consciousness takes place immediately, and respiration ceases. There is no pulse at any site, including the apical and carotid sites (see Fig. 12-17).

Arrest may occur for many reasons; for example, excessive vagal nerve stimulation may slow the heart, drug toxicity may occur, or there may be insufficient oxygen to maintain the heart tissue due to severe shock or ventricular fibrillation. In order to resuscitate a person, blood flow to the heart and brain must be maintained.

EMERGENCY TREATMENT FOR CARDIAC ARREST

1. Call for emergency medical help and begin CPR.
2. Commence use of an automatic electrical defibrillator if one is available. (These are located in public buildings and marked with a red symbol showing an electrical flash through a heart. The letters AED appear on the cover [Fig. 12-19].)
3. Continue CPR if no AED device is available or if instructed to do so by the device.

FIGURE 12-19 The universal AED symbol indicates presence and location of automatic electrical defibrillator. Symbol may be red or green in color.

Congestive Heart Failure

Pathophysiology

Congestive heart failure occurs when the heart is unable to pump sufficient blood to meet the metabolic needs of the body. Usually CHF occurs as a complication of another condition. It may present as an acute episode but usually is a chronic condition. It may result from a problem in the heart itself, such as infarction or a valve defect; it may arise from increased demands on the heart, such as those imposed by hypertension or lung disease; or it may involve a combination of factors. Depending on the cause, one side of the heart usually fails first, followed by the other side. For example, an infarction in the left ventricle or essential hypertension (high blood pressure) affects the left ventricle first, whereas pulmonary valve stenosis or pulmonary disease affects the right ventricle first. It is helpful in the early stages to refer to this problem as left-sided CHF or right-sided CHF.

Initially various compensation mechanisms maintain cardiac output (Fig. 12-20, top part). Unfortunately, these mechanisms often aggravate the condition instead of providing assistance:

- The reduced blood flow into the systemic circulation and thus the kidneys leads to increased renin and aldosterone secretion. The resulting vasoconstriction (increased afterload) and increased blood volume (increased preload) add to the heart's workload.
- The SNS response also increases heart rate and peripheral resistance. Increased heart rate may decrease the efficiency of the heart and impede filling, as well as increasing work for the heart.
- The chambers of the heart tend to dilate (enlarge), and the cardiac muscle becomes hypertrophied (**cardiomegaly**), with the wall of the ventricle becoming thicker. This process demands increased blood supply to the myocardium itself, and eventually some myocardial cells die, to be replaced with fibrous tissue.

There are two basic effects when the heart cannot maintain its pumping capability:

1. *Cardiac output or stroke volume decreases,* resulting in less blood reaching the various organs and tissues, a "forward" effect. This leads to decreased cell function, creating fatigue and lethargy. Mild acidosis develops, which is compensated for by increased respirations (see Chapter 2). Because the affected ventricle cannot pump its load adequately, the return of blood to that side of the heart is also impaired.
2. *"Backup" congestion develops* in the circulation behind the affected ventricle (Fig. 12-21). The output from the ventricle is *less* than the inflow of blood.

For example, if the left ventricle cannot pump all of its blood into the systemic circulation, the normal volume of blood returning from the lungs cannot enter the left side of the heart. This eventually causes congestion in the pulmonary circulation, increased capillary

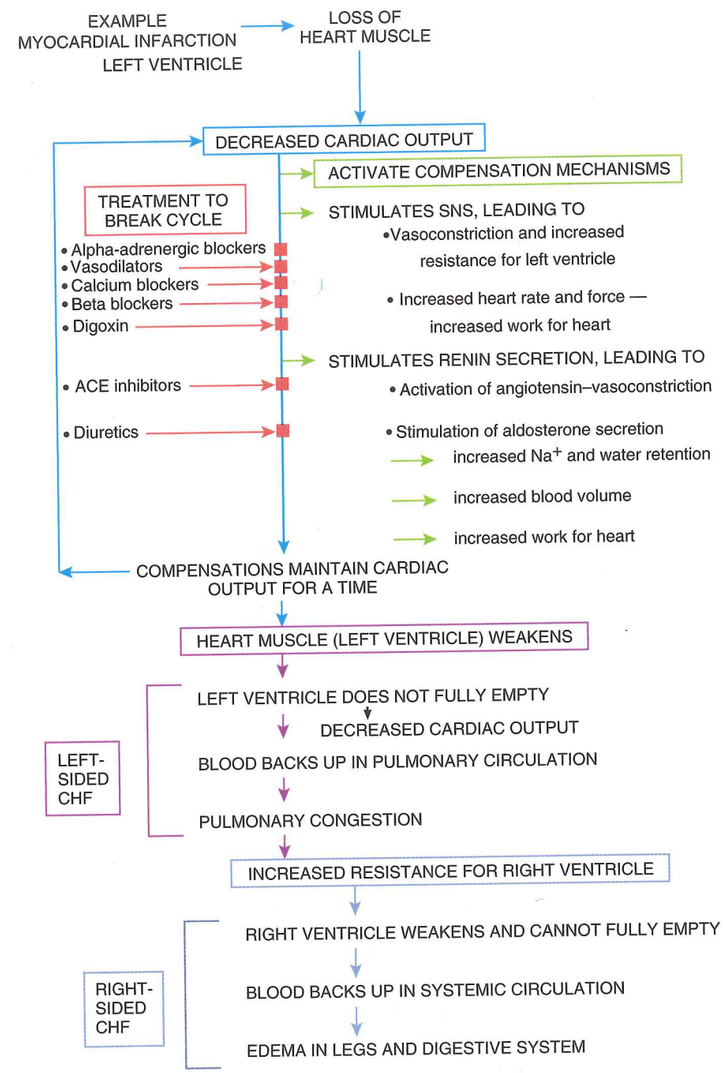

FIGURE 12-20 Course of congestive heart failure.

pressure, and possible pulmonary edema, in which fluid is forced into the alveoli. This situation is termed left-sided CHF.

In right-sided CHF, the right ventricle cannot maintain its output, so less blood proceeds to the left side of the heart and the systemic circulation (forward effect). The backup effect, or congestion, is apparent in the systemic circulation, as shown by increased blood volume and congestion in the legs and feet and eventually also in the portal circulation (liver and digestive tract) and neck veins. Right- and left-sided cardiac failures are compared in Table 12-3.

Etiology

Infarction that impairs the pumping ability or efficiency of the conducting system, valvular changes, or congenital heart defects may cause failure of the affected side. Presently coronary artery disease is the leading cause of CHF. Increased demands on the heart cause heart failure that may take various forms, depending on the ventricle most adversely affected. For example, essential hypertension increases diastolic blood pressure, requiring the left ventricle to contract with more force to open the aortic valve and eject blood into the aorta. The left ventricle hypertrophies and eventually