The Circulatory System: The Heart

Objectives
In this chapter we will study
• tests commonly used in diagnosing cardiovascular disorders;
• the general symptoms and treatment of cardiac arrhythmias;
• cardiac inflammation (pericarditis, endocarditis, and rheumatic heart disease);
• cardiomyopathy; and
• myocardial infarction.

Diagnosing Cardiovascular Disorders
The diagnosis of cardiovascular diseases begins with the patient history and physical examination. A patient who complains of tightness in the chest, a burning pain worsened by coughing, difficulty breathing, weakness, lightheadedness, and fatigue may be at risk for one of many cardiovascular diseases, especially if he or she also has a family history of heart disease. Various aspects of cardiovascular function are routinely assessed in a physical examination. The pulse rate, strength, and rhythm are examined by palpation; the heart sounds are studied by auscultation with a stethoscope; and the blood pressure is measured with a sphygmomanometer.

If a cardiovascular disorder is suspected, further testing is warranted. Such tests include noninvasive and invasive techniques as well as blood analysis.

Noninvasive Tests
Reduced cardiac output affects the oxygen and glucose supply to all organs, and the central nervous system is among the most sensitive to such deficiencies. Therefore, one of the most obvious signs of cardiac dysfunction is the impairment of psychological and motor functions such as attention, consciousness, coherent thought and speech, pupillary reflexes, and visual gaze and tracking movements. Poor cardiac output also causes the blood hemoglobin to become dark red or violet in color. This effect is most easily seen in areas of the body that have a dense capillary network and thin epithelium. Thus, a cardiovascular assessment includes noting the color of the gums and other mucous membranes, conjunctivae, and nail beds. Cyanosis, or blueness of the membranes, suggests reduced cardiac function, although it also has other causes. Cyanosis therefore brings to mind several etiological hypotheses and requires further tests to narrow down its cause.

Palpation of the pulse and auscultation of the heart sounds are vital to any cardiovascular examination. More sophisticated techniques involve various forms of cardiography, the measurement and recording of cardiac functions. The best-known of these techniques is electrocardiography, the recording of an electrocardiogram (ECG). A phonocardiogram, or record of the heart sounds, is made by placing a microphone on the precordium (the chest wall anterior to the heart) and connecting it to an amplifying and recording instrument. An echocardiogram is similar in principle to a fetal sonogram. Oil is spread on the chest, and a device is placed against it that generates ultrasonic vibration and detects the echoes that come back from the heart and associated structures. The record obtained through the cardiac cycle gives information on cardiac anatomy and such functional characteristics as stroke volume and cardiac output.

Other specific diagnostic tests include the following:
• A pulse tracing is a record of the pulsation produced by blood flowing through a vessel. It is produced by placing a sensor over a blood vessel such as the common carotid artery and recording fluctuations in blood pressure over the course of the cardiac cycle. This method is used in conjunction with the ECG and phonocardiography to determine the timing of the various events in the cardiac cycle.
• A Doppler study is a technique for listening to the sounds of blood flowing in the vessels for evidence of obstructions to flow or valvular defects in the heart. The sounds are amplified by a microphone hand-held over the blood vessel.
• A stress test is a study of the ECG and blood pressure during exercise. A patient typically walks a treadmill until the maximum heart rate for his or her age and sex is reached or until he or she begins to show signs of cardiac distress such as chest or leg pain, extreme fatigue, or extreme dyspnea. The ECG and blood pressure are then examined in comparison to pre-exercise records for indicators of cardiovascular disease.

• A chest X ray is a routine part of a cardiac examination. It shows the size, contour, and position of the heart relative to surrounding structures. A sharper silhouette of the heart can be obtained if the patient first swallows a contrast medium such as barium, which makes the esophagus appear as a bright white background against which the image of the heart stands out.

**Invasive Tests**

Except for the barium swallow, the techniques just mentioned are considered noninvasive because nothing enters the body. Invasive methods entail more risk but can provide an overriding benefit in some cases by giving the diagnostician more detailed and specific information.

In cardiac catheterization, a catheter (a thin, flexible tube) is threaded into a blood vessel until it enters a heart chamber. The catheter may then be used to determine pressure within a heart chamber, to withdraw blood for measurement of blood oxygen level, or to introduce a contrast medium that enhances images of heart chamber function, valvular function, or the coronary arteries. Visualization of the coronary arteries is called coronary angiography (arteriography). A catheter is threaded from the femoral artery into the left ventricle, and a contrast dye is injected to allow filming of ventricular function for a few cardiac cycles. Then the catheter is pushed to the openings of the coronary arteries, and dye is injected into the arteries so that they can be visualized. This method is used primarily to evaluate atherosclerosis.

Other invasive methods include PET scans, injection of radioisotopes to localize myocardial infarcts or ischemic areas, and electrocardiography using electrodes introduced into the heart by way of a catheter to record from the AV bundle.

**Blood Analysis**

Blood samples also provide information about cardiac function. When cardiac muscle tissue is destroyed (as in myocardial infarction), enzymes and other cytoplasmic components leak into the blood. The enzymes of greatest interest to cardiac function are aspartate transaminase, creatine kinase (CK), and lactate dehydrogenase (LDH). All of these enzymes appear in the serum within hours of a myocardial infarction, and each enzyme is elevated at different times. Aspartate transaminase peaks in 12 to 24 hours and returns to normal in 2 to 7 days. CK peaks within 24 hours and returns to normal in 3 to 5 days. LDH rises in 12 to 24 hours, peaks within 72 hours, and returns to normal in 8 to 12 days.

Additionally, circulating sodium and potassium concentrations serve as markers in a manner similar to that observed in skeletal muscle. The ability of the heart to effectively pump blood is measured in part by the various blood gas measurements. Normal measurements for serum enzymes, ion concentrations, and blood gases are found in the Appendix of Normal Values at the end of this manual.

**Cardiac Arrhythmia**

Cardiac arrhythmia (dysrhythmia) is any disturbance of the normal heart rhythm. The signs and symptoms of arrhythmias vary from patient to patient, but in general they include palpitations (awareness of the heartbeat), dizziness and syncope (fainting), and diagnostic alterations in the ECG. However, because the patient may not experience a bout of arrhythmia while in the clinic, the physician may have the patient wear a monitor to record heart activity over a 24-hour period or longer.

Arrhythmia is treated with several techniques, including anti-arrhythmic drugs or a pacemaker. An important aspect of treatment is to reassure the patient and reduce anxiety, especially in cases that produce palpitations but pose no health risk. Precipitating factors such as exercise, alcohol, or caffeine may be identified and the patient encouraged to modify his or her behavior.

Anti-arrhythmic drugs are the most common treatment. Four classes of anti-arrhythmic drugs are available, with the choice determined by the type of arrhythmia and the side effects of the drug: Na+.
channel blockers (lidocaine, quinidine, encainidine), β-blockers (propranolol, atenolol), K⁺ channel blockers (sotalol, aminodarone), and Ca²⁺ channel blockers (verapamil, diltiazem). Pacemakers are small, battery-powered devices with electrodes that stimulate the atria or ventricles in response to events in the heart. Today’s pacemakers are programmable and can be used to regulate both tachycardia and bradycardia.

**Inflammatory Heart Diseases**

A wide variety of microorganisms can infect the tissues of the heart and trigger cardiac inflammation, or carditis. Three examples are explored here—pericarditis, endocarditis, and rheumatic heart disease.

**Pericarditis**

Conditions elsewhere in the body often lead to disorders of the pericardium. For example, infection, connective tissue diseases, and radiation therapy commonly trigger pericarditis, inflammation of the pericardium. Pericarditis produces sudden chest pain that is worsened by breathing, often making a person think he or she is having a heart attack. Other symptoms include irritability, restlessness, malaise, and difficulty swallowing. Signs found upon examination include tachycardia, a low fever, and a raspy, sandpaper-like sound called a friction rub heard at the apex and left sternal margin of the heart. The friction rub occurs when the inflamed, roughened pericardial membranes rub against each other. Pericarditis is treated with rest, analgesics, and nonsteroidal anti-inflammatory drugs.

Pericarditis usually resolves by itself in time, but some cases are complicated by pericardial effusion, the seepage of fluid into the pericardial cavity. If the fluid accumulates slowly, the pericardium can stretch to accommodate it, but if it accumulates rapidly, it can cause cardiac tamponade, a compression of the heart that prevents it from filling completely and thus reduces the stroke volume. As little as 50 to 100 ml of fluid may induce serious tamponade. Cardiac tamponade can be detected from a condition called pulsus paradoxus in which the arterial blood pressure is more than 10 mm higher when the patient exhales than when he or she inhales. Echocardiograms are the most sensitive way of confirming cardiac tamponade. If serious, pericardial effusion is treated by pericardiocentesis—puncturing the pericardium and withdrawing the fluid.

**Endocarditis**

Endocarditis is inflammation of the endocardium, especially the heart valves. It usually results from infection with bacteria, viruses, fungi, or parasites—but most often, the streptococcus and staphylococcus types of bacteria. It is often triggered by mitral valve prolapse, implantation of artificial heart valves, long-term use of cardiac catheters, I.V. drug abuse, and cardiac surgery. Males are affected twice as often as females.

Pathogenesis begins when a heart valve or other area of endocardium is “prepared” by endothelial damage to support colonization by microbes. Platelets adhere to the damaged region and produce a thrombus that can then serve as a focus of bacterial adhesion. Microbes can invade the blood from such sources as upper respiratory or skin infections, bladder catheterization, or even dental cleaning. They adhere to the thrombus and begin to proliferate, so that within 24 hours, there develops a lesion of alternating layers of bacterial colonies and clotted blood.

Typical signs of endocarditis include fever, weight loss, night sweats, a heart murmur, and abnormalities in the erythrocytes, urine, and ECG. The diagnosis is confirmed by culturing bacteria from the blood and by echocardiography. The disease is treated with antibiotics, but repetitive episodes of endocarditis may damage the valves so extensively that they require surgical replacement. Prevention of endocarditis is the reason some people are given antibiotics prior to receiving dental work.

**Rheumatic Heart Disease**

Rheumatic fever is an inflammatory disease caused by the immune response to a certain class of streptococcal bacteria. If untreated, it can lead to rheumatic heart disease, a scarring and deformity of the heart. Rheumatic fever arises most often in children from 5 to 15 years of age, developing solely as a complication of a streptococcal throat infection. If the throat infection is treated within 9 days, rheumatic fever usually does not develop. But if treatment is delayed, the infection progresses to rheumatic fever in about 3% of cases. This disease gets its name not only because it produces a fever but also because bacterial antigens bind to receptors in the synovial joints and trigger an autoimmune response, leading to widespread joint pain, among other symptoms.
About 10% of children with rheumatic fever go on to develop rheumatic heart disease. This syndrome begins with carditis in all three layers of the heart wall, but the endocarditis is the most serious. Beadlike clumps of “vegetation” develop on the heart valves and chordae tendineae. These structures become scarred and constricted, valve cusps may adhere to each other, and eventually a patient can die of cardiomegaly (enlargement of the heart), defects in electrical conduction in the heart, and left heart failure.

The first priority in treating rheumatic heart disease is to inhibit the inflammation. Aspirin (salicylate) is the treatment of choice, but cases unresponsive to aspirin are treated with corticosteroids. Penicillin G or other antibiotics are used to prevent recurrence of the streptococcus infection. Severely damaged heart valves may require surgical replacement.

**Cardiomyopathies**

*Cardiomyopathies* are structural or functional abnormalities of the myocardium. Most cases are idiopathic, but some are triggered by infectious diseases, toxins, cancer chemotherapy, alcoholism, connective tissue disease, or nutritional deficiencies. The two most common cardiomyopathies are *dilated* and *hypertrophic*.

**Dilated cardiomyopathy** is characterized by dilation of the ventricle and loss of contractility, so that the end-diastolic volume becomes greater (more blood remains behind in the heart with each beat) and the stroke volume is severely reduced. Patients commonly experience dyspnea, fatigue, palpitations, dysrhythmia, and dizziness. Dilated cardiomyopathy is treated with digitalis to stimulate the heart, diuretics to promote water excretion and lower the blood pressure, and bed rest, sometimes for extended periods. The prognosis depends on the extent of myocardial damage. Deaths from dilated cardiomyopathy usually occur within 5 years of diagnosis and most commonly result from left-ventricular failure.

**Hypertrophic cardiomyopathy** is marked by thickening of the interventricular septum. It seems to have a genetic basis. The diseased heart may appear normal in size, but thickening of the septum reduces the capacity of the ventricles. The ventricles stiffen and exhibit reduced filling and output. Angina, dizziness, palpitation, and dysrhythmia are among the signs and symptoms. Beta-blockers such as propanolol sometimes reduce ventricular stiffness and improve ventricular filling and ejection. Some cases are treated by surgically removing part of the hypertrophied myocardium. The chance of long-term survival is good with appropriate management.

Hope is now available for some patients with cardiomyopathies through heart transplants. But because donor hearts are in short supply, transplants are usually limited to patients under the age of 50 years, and even then most patients selected for transplant die before a donor heart becomes available. In the United States, it is estimated that only 10% to 12% of all patients with cardiomyopathies receive hearts annually. Successful transplantation results in a 50% to 70% 5-year survival rate.

**Myocardial Ischemia and Infarction**

Coronary atherosclerosis may cause myocardial ischemia—a prolonged deficiency of blood flow to the cardiac muscle—and lead to *myocardial infarction (MI)*, or heart attack. Frequently, an MI occurs because platelets aggregate on an atherosclerotic plaque in a coronary artery and form a thrombus (blood clot), which can build up rapidly and block the artery or break loose and block a smaller artery downstream. If half or more of the artery lumen becomes blocked, blood flow may be inadequate to meet the metabolic needs of the myocardium, especially when cardiac workload increases.

The myocardium can tolerate about 20 minutes of ischemia before tissue death begins. Within 8 seconds, the myocardial oxygen reserves are depleted and the muscle shifts to anaerobic fermentation. Fermentation generates hydrogen ions and lactic acid, lowering the tissue pH and contributing in multiple ways to cellular injury. The cells leak K⁺, Ca²⁺, and Mg²⁺; their contractility declines; and the heart’s pumping ability is compromised. MI triggers a strong inflammatory response leading, if the patient survives, to tissue repair by fibrosis. Thus, some people’s hearts exhibit scar tissue that indicates earlier infarctions of which they may have been unaware.

The first symptom of an acute MI is often severe chest pain, commonly described as a heavy, crushing sensation, “like an elephant sitting on my chest.” The pain often radiates to the neck, jaw, back, shoulder, and left arm. Yet, in some cases, pain is absent. Consequently, the MI may not be immediately diagnosed or a patient may even deny that he or she
has a life-threatening condition requiring emergency care. Patient denial is a major factor in the delay of treatment for MI and thus a major factor in mortality; 50% of deaths from acute MI occur in the first 3 to 4 hours of onset.

Other signs and symptoms of MI include restlessness, pallor, apprehension, sweating, and cyanosis. The pulse is unusually fine and difficult to feel (“thready”). An ECG often reveals arrhythmia with abnormal Q waves, changes in the S-T segment, and inverted T waves. Myocardial enzymes are elevated (see previous discussion), and within 12 hours the WBC count rises.

Diagnosis is based on the signs and symptoms just mentioned, along with the findings of imaging techniques. Patients are admitted to the hospital where the cardiac rhythm and serum enzymes can be monitored. Treatment involves the prompt administration of aspirin to minimize blood clotting and thrombolytic drugs such as tissue plasminogen activator (TPA) or streptokinase, which break up blood clots that already exist and restore myocardial perfusion in about 3 minutes. Pain relief is usually accomplished by administering sublingual nitroglycerin, a coronary vasodilator. Acute coronary care is followed by bed rest, dietary modification, and a gradual return to normal activity.

Long-term survival depends upon many factors—degree of left-ventricular ischemia and dysfunction, age, diet, and potential for ventricular dysrhythmias. About 8% to 10% of those who suffer an acute MI die within 1 year, and most of these within 3 to 4 months. The most common cause of death is ventricular fibrillation.

Case Study 19  The Hard-Working Executive

Paul is 42 years old and the president of a small company. He is just beginning his daily workout at the local gym when he notices a slight tightness in his chest. As he continues riding an exercise bike, the pain becomes more severe and radiates to his left arm, shoulder, and jaw. Paul decides he is “overdoing it” and heads for the showers, intending to go back to work for the rest of the day and see how he feels tomorrow. After returning to his office, he begins to sweat and mentions to his secretary that he has an upset stomach. He thinks he might be coming down with the flu, but needs to get a few things done before going home.

Approximately 10 minutes later, Paul’s secretary finds him unconscious on the floor of his office and calls an ambulance. When the paramedics arrive, they find that Paul is not breathing and has no detectable pulse. His skin is pale, cool, and clammy. They initiate cardiopulmonary resuscitation, reestablish breathing and a regular heartbeat, administer the clot-dissolving agent TPA, and begin transporting Paul to the hospital. On the way, Paul regains consciousness, and a paramedic gives him an aspirin to chew. They determine the following vital signs:

- Heart rate = 50 beats/min and irregular
- Blood pressure = 74/48 mmHg
- Respiratory rate = 16 breaths/min and shallow

At the hospital, Paul is promptly placed on a cardiac monitor, blood is drawn for enzyme analysis, and I.V. propanolol is given. Once Paul is stabilized, he is transported to the cardiac care unit (CCU). He is given nitroglycerin for pain relief and nasal oxygen to maintain an adequate blood O2 level; his ECG and blood pressure are closely monitored by the nursing staff.

Among the blood test results are the following:
- pH = 7.18
- Lactate = 42 mEq/L
- Creatine kinase (CK) = 82 IU/L
- Lactate dehydrogenase (LDH) = 130 IU/L

These results, coupled with the ECG, preadmission symptoms, and patient history, confirm that Paul has suffered a myocardial infarction. Paul is kept in the hospital for 7 days for monitoring and treatment. His cardiologist and his primary care physician call on Paul in the hospital. His primary care physician is well aware of Paul’s history: He is divorced and overweight, smokes up to three packs of cigarettes per day, is under treatment for atherosclerosis, and has a family history of hypertension. Paul is frightened by his hospitalization and resolves to lose weight and quit smoking. His physician reinforces these decisions, prescribes a mild tranquilizer to relieve Paul’s anxiety, and discusses
Paul’s rehabilitation with him. After 3 days of bed rest, Paul is encouraged to get up, rest in a chair, read, and walk to the bathroom as needed. He is released at the end of the week, but scheduled for frequent visits to his cardiologist for the next 6 weeks and counseled on gradual resumption of normal physical activity and on the issues of smoking, weight loss, work habits, and diet.

Based on this case study and other information in this chapter, answer the following questions.

1. What risk factors predispose Paul to a myocardial infarction?

2. Explain the physiological basis of Paul’s elevated serum lactate and lactate dehydrogenase concentrations.

3. Explain why Paul is given an aspirin to chew on the way to the hospital.

4. If Paul’s MI results from occlusion of the circumflex coronary artery only, where would you expect to find the myocardial lesion in a PET scan of the heart?

5. Explain why Paul is given propranolol in the hospital. What type of drug is this? How would it improve his condition?

6. Both nitroglycerin and streptokinase can restore perfusion of the myocardium through an obstructed coronary artery, but they do so in different ways. Contrast the mechanisms of these two drugs.

7. Why would you expect intravenous drug users to have a high incidence of endocarditis?

8. Cardiac tamponade restricts the filling and stroke volume of the right side of the heart before the left. Why do you think this is so?

9. Heart attack patients in a CCU are ideally kept in a private room and allowed few visitors for the first 2 or 3 days. The room should have a clock, calendar, and window to the outdoors, and the patient may be provided with light reading if he or she wishes, but it is better not to furnish a radio, television, or newspapers. Explain the reason for all of this.

10. At the age of 20, Oscar is diagnosed with insufficiency of the bicuspid valve. Which of the following could indicate a predisposition for this diagnosis?
   a. hypertrophic cardiomyopathy
   b. pericarditis
   c. a childhood history of rheumatic fever
   d. cardiac arrhythmia
   e. pericardial effusion

Selected Clinical Terms

- **cardiac catheterization**  Insertion of a narrow flexible tube (catheter) into the heart for blood sampling, pressure measurement, or dye injections.
- **cardiomyopathy**  Any structural or functional abnormality of the myocardium.
- **coronary angiography**  A method in which a contrast medium is injected into the coronary arteries and an X-ray made to assess arterial occlusion or other abnormalities.
- **echocardiogram**  An ultrasonic scan of the heart for the purpose of assessing cardiac anatomy and function.
- **endocarditis**  Inflammation of the endocardium, usually as a result of bacterial or other infection.
- **friction rub**  A raspy sound heard near the apex and left sternal margin of the heart when inflamed pericardial membranes rub across each other.
- **pericardial effusion**  Seepage of fluid into the pericardial cavity, presenting a risk of cardiac tamponade.
- **pericarditis**  Inflammation of the pericardium usually triggered by pathologies elsewhere in the body.
- **pulse tracing**  A method of measuring pulsations in blood pressure in a particular blood vessel over the cardiac cycle.
- **rheumatic heart disease**  Scarring and deformity of the heart, especially the endocardium, as the result of an autoimmune response to a streptococcus infection.
- **stress test**  An evaluation of cardiovascular fitness by recording the ECG and blood pressure during a defined strenuous exercise such as walking a treadmill.