

# 8

## THE HEART: LABORATORY TESTS AND DIAGNOSTIC PROCEDURES

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### OBJECTIVES

After completing this chapter, the reader should be able to

- Describe the normal physiology of the heart
- Describe the electrocardiogram changes reflected by myocardial ischemia and infarction
- Explain the roles of the different biochemical markers in the diagnosis of coronary artery disease, acute coronary syndrome, and heart failure
- Given a patient's history, clinical presentation, cardiac biochemical markers, and electrocardiographic findings, assess the presence and type of acute coronary syndrome
- Given a patient case, assess the presence and type of heart failure
- Describe the role of pharmacologic agents in noninvasive imaging studies
- Describe other diagnostic procedures used for the evaluation of coronary artery disease, acute coronary syndrome, and heart failure

The heart has two basic properties: electrical and mechanical. The two work in harmony to propel blood, delivering oxygen and nutrients to all body tissues. Heart cells responsible for these properties are (1) pacemaker cells or the “electrical power” of the heart; (2) electrical conducting cells or the “hardwiring circuitry” of the heart; and (3) myocardial cells or the contractile units of the heart. Disturbances in the electrical system result in rhythm disorders, also known as *arrhythmias* or *dysrhythmias*. The pumping action is accomplished by means of striated cardiac muscle, which largely composes the myocardium. A number of cardiovascular diseases disrupt the mechanical function of the heart including coronary artery disease (CAD), acute coronary syndrome (ACS), and heart failure.

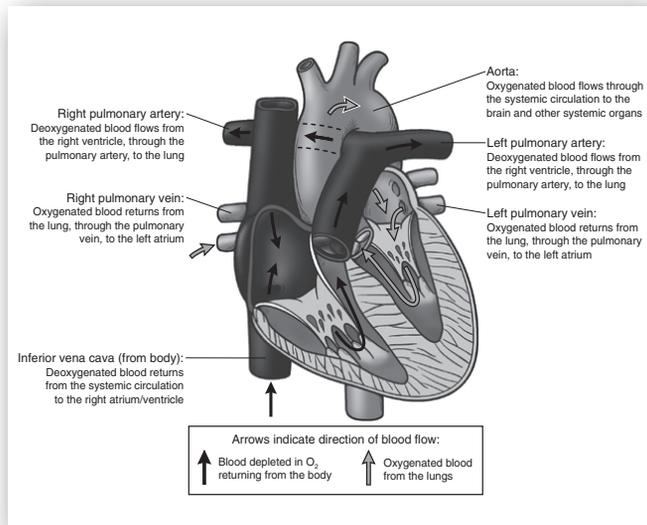
The management of ACS, heart failure, and potential complications of each contribute greatly to the overall health of and cost incurred by society. Laboratory tests are essential for establishing the diagnosis and determining the prognosis of patients. Accurate and expeditious assessment of a patient presenting with symptoms suggestive of ACS guides individualized treatment to optimize a patient's short-term and long-term outcomes. Conversely, rapid exclusion of the diagnosis permits early discharge from the coronary care unit or hospital. Laboratory and other diagnostic tests used in evaluating the patient with possible ACS and heart failure are discussed in this chapter.

### CARDIAC PHYSIOLOGY

The heart consists of two pumping units that operate in parallel, one on the right side and the other on the left side. Each is composed of an upper chamber, the atrium, and a lower chamber, the ventricle. The atrium serves as a passive portal to the ventricle and is a weak pump that helps move blood into the ventricle. The atrial contraction or kick is responsible for 20–30% of ventricular filling. The right and left ventricles supply the primary force that propels blood through the pulmonary and peripheral circulation, respectively (**Figure 8-1**).

The functional unit of the heart is comprised of a network of noncontractile cells that form the conduction system, which is responsible for originating and conducting action potentials from the atria to the ventricles. This leads to the excitation and contraction of the cardiac muscle, which is responsible for the pumping of the blood to the other organs.

The normal adult human heart contracts rhythmically at approximately 70 beats per minute (bpm). Each cardiac cycle is divided into a systolic and diastolic phase. During each cycle, blood from the systemic circulation is returned to the heart via the veins, and blood empties from the superior and inferior vena cavae into the right atrium. During the diastolic phase, blood passively fills the right ventricle through the tricuspid valve with an active filling phase by atrial contraction just prior to end-diastole. During systole, blood is then pumped from the right ventricle through the pulmonary artery to the lungs where carbon dioxide is removed and the blood is oxygenated. From the lungs, blood returns to the heart via the pulmonary veins and empties into the left atrium. Again, during diastole, blood empties from the left atrium through the mitral valve into the main pumping chamber,



**FIGURE 8-1.** Blood flow through the heart and the pulmonary and systemic circulation.

the left ventricle. With systole the left ventricle contracts and blood is forcefully propelled into the peripheral circulation via the aorta (Figure 8-1). At rest, the normal heart pumps approximately 4–6 L of blood per minute. Maintaining normal cardiac output (CO) is dependent on the heart rate (HR) and stroke volume (SV).

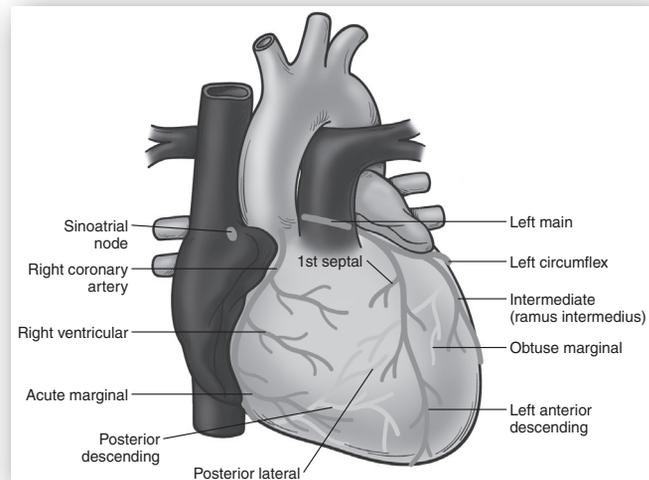
$$\text{CO (mL/min)} = \text{HR (beats/min)} \times \text{SV (mL/beat)}$$

The SV, defined as the volume of blood ejected during systole, is determined by intrinsic and extrinsic factors including myocardial contractility, preload, and afterload. The coronary arteries, the arteries supplying the heart muscle, branch from the aorta just beyond the aortic valve and are filled with blood primarily during diastole. The major coronary arteries are depicted in Figure 8-2. In the face of increased myocardial metabolic needs, the heart is able to increase coronary blood flow by vasodilation to meet myocardial oxygen demand.

## CARDIAC DYSFUNCTION

Decreased CO compromises tissue perfusion, and depending on the severity and duration, may lead to significant acute and chronic complications. A number of cardiac conditions lead to decreased CO, including hypertensive heart diseases, heart failure, valvular heart diseases, congenital heart diseases, diseases of the myocardium, conduction abnormalities, CAD, and ACS. This chapter focuses on the various tests used in the diagnosis and assessment of patients presenting with CAD, ACS, and heart failure.

Also known as *ischemic heart disease* (IHD), CAD is caused by atherosclerosis of the coronary arteries, resulting in lumen narrowing and blood flow reduction to the myocardium perfused by the affected artery. This leads to tissue ischemia and chest pain. Severe reduction in or total interruption of blood flow may lead to severe tissue ischemia or infarction, resulting in a clinical presentation as a type of ACS.



**FIGURE 8-2.** Major coronary arteries and their principal branches.

Patients with severe symptoms of myocardial ischemia or acute myocardial infarction (MI) may be experiencing one of three types of ACS: unstable angina (UA), non-ST-segment elevation MI (NSTEMI), or ST-segment elevation MI (STEMI). The most common cause for ACS is atherosclerotic plaque rupture and subsequent obstruction of the coronary lumen by thrombosis composed of platelet aggregates, fibrin, and entrapped blood cells leading to myocardial ischemia. When a coronary artery is occluded, the location, extent, rate, and duration of occlusion determine the severity of myocardial ischemia resulting in UA, NSTEMI, or STEMI.

According to the universal definition, MI may be classified into different types<sup>1</sup>:

- **Type 1:** Spontaneous MI related to ischemia due to a primary coronary event such as plaque erosion and rupture, fissuring, or dissection
- **Type 2:** MI secondary to ischemia due to an imbalance between oxygen supply and demand, such as in coronary artery spasm, coronary embolism, anemia, arrhythmias, hypertension, or hypotension
- **Type 3:** MI resulting in sudden cardiac death in the setting of absent biomarkers
- **Type 4a:** MI associated with percutaneous coronary intervention (PCI)
- **Type 4b:** MI associated with stent thrombosis as documented by angiography or at autopsy
- **Type 5:** MI associated with coronary artery bypass graft

Complications of MI include cardiogenic shock, heart failure, ventricular and atrial arrhythmias, ventricular rupture or ventricular septal defect formation, cardiac tamponade, pericarditis, papillary muscle rupture, mitral regurgitation, and embolism. Initial assessment of the patient presenting with ACS may be confounded by the presence and severity of the above complications.

Heart failure is a syndrome in which the heart is unable to pump blood at a rate sufficient to meet the demands of the body or unable to accept the fluid volume with which it is