TABLE 2-1 NORMAL VALUES FOR COMMON HEMODYNAMIC PARAMETERS

Heart rate	60-100 beats/min
PRESSURES (mm Hg)	
Central venous	≤9
Right atrial	≤9
Right ventricular	
Systolic	15-30
End-diastolic	≤9
Pulmonary arterial	
Systolic	15-30
Diastolic	3-12
Pulmonary capillary wedge	≤12
Left atrial	≤12
Left ventricular	
Systolic	100-140
End-diastolic	3-12
Aortic	
Systolic	100-140
Diastolic	60-90
RESISTANCE	
Systemic vascular resistance	800-1500 dynes-sec/cm^{-5}
Pulmonary vascular resistance	30-120 dynes-sec/cm^{-5}
Cardiac output	4-6 L/min
Cardiac index	2.5-4 L/min

TABLE 2-2 FACTORS AFFECTING CARDIAC PERFORMANCE

PRELOAD (LEFT VENTRICULAR DIASTOLIC VOLUME)
Total blood volume
Venous (sympathetic) tone
Body position
Intrathoracic and intrapericardial pressures
Atrial contraction
Pumping action of skeletal muscle
AFTERLOAD (IMPEDANCE AGAINST WHICH THE LEFT VENTRICLE MUST EJECT BLOOD)
Peripheral vascular resistance
Left ventricular volume (preload, wall tension)
Physical characteristics of the arterial tree (elasticity of vessels or presence of outflow obstruction)
CONTRACTILITY (CARDIAC PERFORMANCE INDEPENDENT OF PRELOAD OR AFTERLOAD)
Sympathetic nerve impulses
Increased contractility
Circulating catecholamines
Digitalis, calcium, other inotropic agents
Increased heart rate or post-extrasystolic augmentation
Anoxia, acidosis
Decreased contractility
Pharmacologic depression
Loss of myocardium
Intrinsic depression
HEART RATE
Autonomic nervous system
Temperature, metabolic rate
Medications, drugs

The cardiac index is a way of normalizing the CO to body size. It is the CO divided by the body surface area and is measured in L/min/m^2. The normal CO is 4 to 6 L/min at rest and can increase fourfold to sixfold during strenuous exercise.

The main determinants of SV are preload, afterload, and contractility (Table 2-2). *Preload* is the volume of blood in the ventricle at the end of diastole; it is primarily a reflection of venous return. Venous return is determined by the plasma volume and the venous compliance. Clinically, intravenous fluids increase preload, whereas diuretics or venodilators such as nitroglycerin decrease preload. When the preload is increased, the ventricle stretches, and the ensuing ventricular contraction becomes more rapid and forceful, because the increased sarcomere length facilitates actin and myosin cross-bridge kinetics by means of an increased sensitivity of troponin C to calcium. This phenomenon is known as the Frank-Starling relationship. Ventricular filling pressure (ventricular end-diastolic pressure, atrial pressure, or pulmonary capillary wedge pressure) is frequently used as a surrogate measure of preload.

Afterload is the force against which the ventricles must contract to eject blood. The main determinants of afterload are the arterial pressure and the dimensions of the left ventricle. As the arterial blood pressure increases, the amount of blood that can be ejected into the aorta decreases. Wall stress, an often overlooked determinant of afterload, is directly proportional to the size of the ventricular cavity and inversely proportional to the ventricular wall thickness (Laplace's law). Therefore, ventricular wall hypertrophy is a compensatory mechanism to reduce afterload. Drugs such as angiotensin-converting enzyme (ACE) inhibitors and hydralazine reduce blood pressure (BP) by reducing afterload. Diuretics decrease left ventricular volume and size, which can reduce wall stress–mediated afterload.

Contractility, or inotropy, represents the force of ventricular contraction in the presence of constant preload and afterload. Inotropy is regulated at a cellular level through stimulation of cathecholminergic (epinephrine, norepinephrine, and dopamine) receptors, intracellular signaling cascades (phosphodiesterase inhibitors), and intracellular calcium levels (affected by levosimendan and, indirectly, by digoxin). Many antihypertensive medications (e.g., β-blockers, calcium channel antagonists) interfere with adrenergic receptor activation or intracellular calcium levels, which can decrease the strength of ventricular contractions. Please refer to Chapter 53, "Cardiac Function and Circulatory Control," in *Goldman-Cecil Medicine*, 25th Edition.

Physiology of the Coronary Circulation

The normally functioning heart maintains equilibrium between the amount of oxygen delivered to myocytes and the amount of oxygen consumed by them (myocardial oxygen consumption, or Mvo_2). If a myocyte works harder because it is contracting with increased frequency (HR), with increased intensity (contractility), or against an increased load (wall stress), then it will use more oxygen and its Mvo_2 will increase. In order to meet this increase in demand for more oxygen, the heart will have to either increase blood flow or increase its efficiency in extracting oxygen. The heart is unique in that its oxygen extraction is almost maximal at resting conditions. Therefore, increasing blood flow is the only reasonable means of increasing oxygen supply.

Microvascular blood flow in the coronary circulation is impaired during systole because the intramyocardial blood vessels are compressed by contracting myocardium. Therefore, most coronary flow occurs during diastole. Accordingly, the diastolic pressure is the major pressure driving flow within the coronary circulation. Systolic pressure impedes intramyocardial