

CHAPTER 145

Heart Failure

ETIOLOGY AND EPIDEMIOLOGY

The force generated by the cardiac muscle fiber depends on its contractile status and basal length, which is equivalent to

the preload. As the preload (fiber length, left ventricular filling pressure, or volume) increases, myocardial performance (stroke volume and wall tension) increases up to a point (the normal Starling curve). The relationship is the **ventricular function curve** (Fig. 145.1). Alterations in the contractile state of the muscle lower the relative position of the curve but retain the relationship of fiber length to muscle work. The cardiac output equals stroke volume times the heart rate; thus heart rate is another important determinant of cardiac work. Additional factors also affect cardiac performance (Table 145.1).

Heart failure occurs when the heart is unable to pump blood at a rate commensurate with metabolic needs (inadequate oxygen delivery). It may be due to a change in **myocardial contractility** that results in low cardiac output or to **abnormal loading conditions** being placed on the myocardium. The abnormal loading conditions may be **afterload** (pressure overload, such as with aortic stenosis, pulmonary stenosis, or coarctation of the aorta) or **preload** (volume overload, such as in ventricular septal defect [VSD], patent ductus arteriosus [PDA], or valvular insufficiency). Volume overload is the most common cause of heart failure in children.

The age of presentation is helpful in creating the differential diagnosis (Table 145.2). In the first weeks of life, excessive afterload being placed on the myocardium is most common. Heart failure presenting around 2 months of age is usually due to increasing left-to-right shunts that become apparent as the pulmonary vascular resistance decreases. Acquired heart disease, such as myocarditis and cardiomyopathy, can present at any age.

CLINICAL MANIFESTATIONS

Heart failure presents in infants as poor feeding, failure to thrive, tachypnea, and diaphoresis with feeding. Older children may present with shortness of breath, easy fatigability, and edema. The physical examination findings depend on whether pulmonary venous congestion, systemic venous

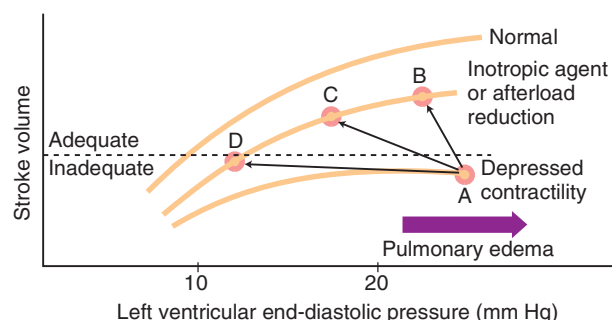


FIGURE 145.1 Ventricular function curve illustrating the effect of inotropic agents or arterial vasodilators. In contrast to diuretics, the effect of digitalis or arterial vasodilator therapy in a patient with heart failure is movement onto another ventricular function curve, which is intermediate between the normal and the depressed curves. When the patient's ventricular function moves from A to B by the administration of one of these agents, the left ventricular end-diastolic pressure may also decrease because of improved cardiac function; further administration of diuretics or venodilators may shift the function further to the left along the same curve from B to C and eliminate the risk of pulmonary edema. A vasodilating agent that has arteriolar and venous dilating properties (e.g., nitroprusside) would shift this function directly from A to C. If this agent shifts the function from A to D because of excessive venodilation or the administration of diuretics, the cardiac output may decrease too much, even though the left ventricular end-diastolic pressure would be normal (10 mm Hg) for a normal heart. Left ventricular end-diastolic pressures of 15–18 mm Hg are usually optimal in the failing heart to maximize cardiac output, but to avoid pulmonary edema. (From Andreoli TE, Carpenter CCJ, Griggs RC, et al., eds. *Cecil Essentials of Medicine*. 5th ed. Philadelphia: Saunders; 2001.)

TABLE 145.1 Factors Affecting Cardiac Performance

PRELOAD (LEFT VENTRICULAR DIASTOLIC VOLUME)

Total blood volume
Venous tone (sympathetic tone)
Body position
Intrathoracic and intrapericardial pressure
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*
Circulating catecholamines*
Digitalis, calcium, other inotropic agents*
Increased heart rate or postextrasystolic augmentation*
Anoxia, acidosis†
Pharmacologic depression†
Loss of myocardium†
Intrinsic depression†

HEART RATE

Autonomic nervous system
Temperature, metabolic rate

*Increases contractility.

†Decreases contractility.

From Andreoli TE, Carpenter CCJ, Griggs RC, et al. *Cecil Essentials of Medicine*. 5th ed. Philadelphia: Saunders; 2001.

TABLE 145.2 Etiology of Heart Failure by Age Group

FETUS

Severe anemia (hemolysis, fetal-maternal transfusion, hypoplastic anemia)
Supraventricular tachycardia
Ventricular tachycardia
Complete heart block
Atrioventricular valve insufficiency
High-output cardiac failure (arteriovenous malformation, teratoma)

PREMATURE NEONATE

Fluid overload
PDA
VSD
Cor pulmonale (BPD)

FULL-TERM NEONATE

Asphyxial cardiomyopathy
Arteriovenous malformation (vein of Galen, hepatic)
Left-sided obstructive lesions (coarctation of aorta, hypoplastic left heart, critical aortic stenosis)
Transposition of great arteries
Large mixing cardiac defects (single ventricle, truncus arteriosus)
Viral myocarditis
Anemia
Supraventricular tachycardia
Complete heart block

INFANT/TODDLER

Left-to-right cardiac shunts (VSD)
Hemangioma (arteriovenous malformation)
Anomalous left coronary artery
Metabolic cardiomyopathy
Acute hypertension (hemolytic uremic syndrome)
Supraventricular tachycardia
Kawasaki disease
Postoperative repair of congenital heart disease

CHILD/ADOLESCENT

Rheumatic fever
Acute hypertension (glomerulonephritis)
Viral myocarditis
Thyrotoxicosis
Hemochromatosis/hemosiderosis
Cancer therapy (radiation, doxorubicin)
Sickle cell anemia
Endocarditis
Cor pulmonale (cystic fibrosis)
Arrhythmias
Chronic upper airway obstruction (cor pulmonale)
Unrepaired or palliated congenital heart disease
Cardiomyopathy

BPD, Bronchopulmonary dysplasia; PDA, patent ductus arteriosus; VSD, ventricular septal defect.

congestion, or both are present. Tachycardia, a gallop rhythm, and thready pulses may be present with either cause. If left-sided failure is predominant, tachypnea, orthopnea, wheezing, and pulmonary edema are seen. Hepatomegaly, edema, and distended neck veins are signs of right-sided failure.

IMAGING STUDIES

The absence of cardiomegaly on a chest x-ray usually rules out the diagnosis of heart failure. An echocardiogram assesses the

heart chamber sizes, measures myocardial function, and diagnoses congenital heart defects when present.

TREATMENT

Initial treatment is directed at improving myocardial function and optimizing preload and afterload. Diuretics, inotropic support, and, often, **afterload reduction** are employed (Table 145.3). Long-term therapy usually consists of diuretics

followed by afterload reduction. Long-term therapy with **β blockers** also may be beneficial, although this remains somewhat controversial in pediatric patients. Spironolactone is usually added to the medical regimen because of its effect on cardiac remodeling.

PEARLS FOR PRACTITIONERS

See *The Cardiovascular System: Pearls for Practitioners* at the end of this section.

TABLE 145.3 Treatment of Heart Failure

THERAPY	MECHANISM
GENERAL CARE	
Rest	Reduces cardiac output
Oxygen	Improves oxygenation in the presence of pulmonary edema
Sodium, fluid restrictions	Decreases vascular congestion; decreases preload
DIURETICS	
Furosemide	Salt excretion at ascending loop of Henle; reduces preload; afterload reduces with control of hypertension; may also cause venodilation
Combination of distal tubule and loop diuretics	Greater sodium excretion
INOTROPIC AGENTS	
Digitalis	Inhibits membrane Na^+ , K^+ -ATPase and increases intracellular Ca^{2+} ; improves cardiac contractility, increases myocardial oxygen consumption
Dopamine	Releases myocardial norepinephrine plus a direct effect on β -receptor; may increase systemic blood pressure; at low infusion rates, dilates renal artery, facilitating diuresis
Dobutamine	β_1 -receptor agent; often combined with dopamine
Milrinone	Phosphodiesterase 3 inhibitor with positive inotropic properties and decreases vascular resistance/afterload
AFTERLOAD REDUCTION	
Hydralazine	Arteriolar vasodilator
Nitroprusside	Arterial and venous relaxation; venodilation reduces preload
Captopril/enalapril	Inhibition of angiotensin-converting enzyme; reduces angiotensin II production
OTHER MEASURES	
Mechanical counterpulsation	Improves coronary flow, afterload
Transplantation	Removes diseased heart
Extracorporeal membrane oxygenation	Bypasses heart
Carvedilol	β -blocking agent