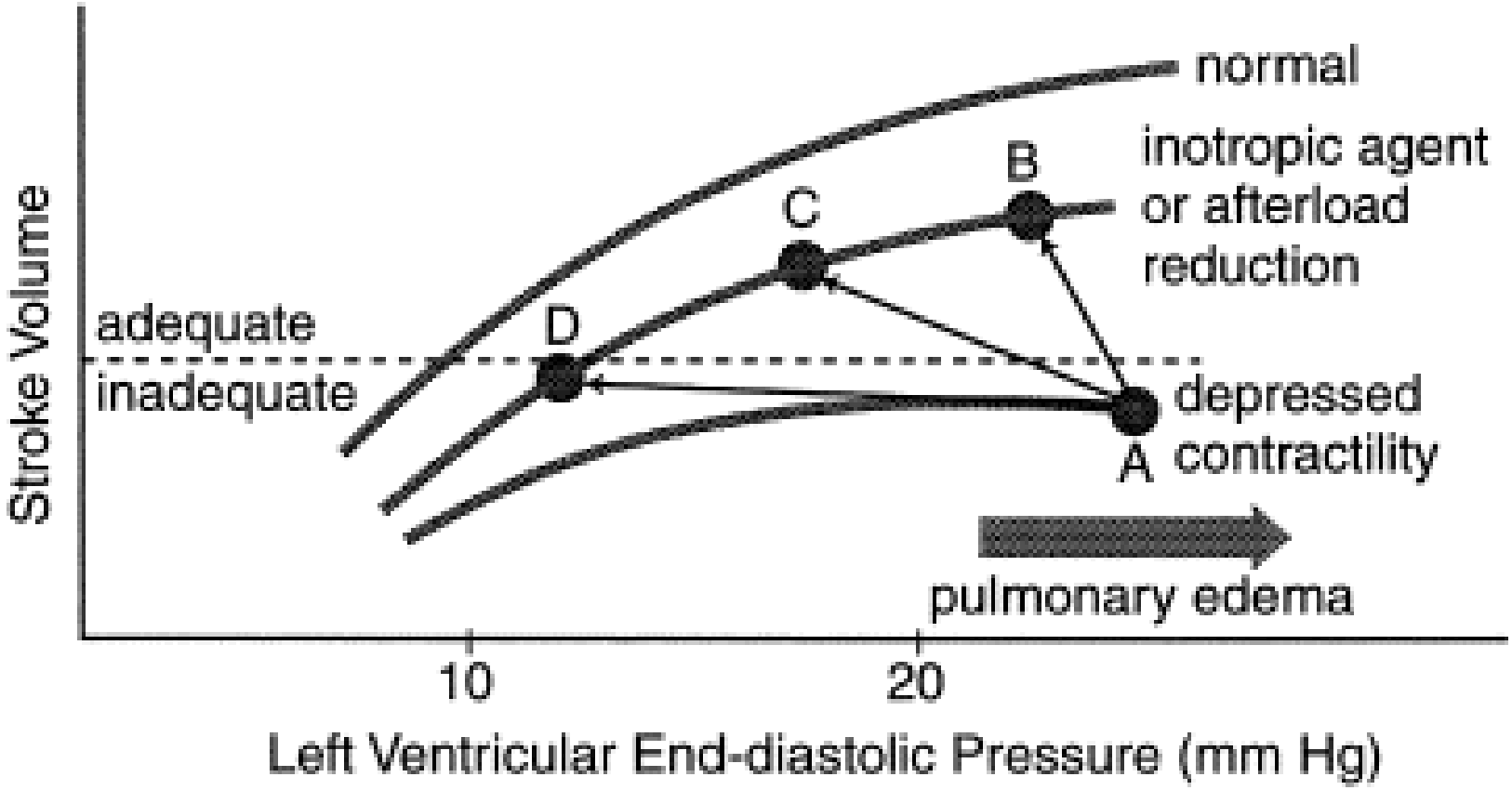
Heart Failure

Etiology

- The heart can be viewed as a pump with an output proportional to its filling volume and inversely proportional to the resistance against which it pumps.
- As ventricular end-diastolic volume increases, a healthy heart increases cardiac output until a maximum is reached and cardiac output can no longer be augmented (the Frank-Starling principle).
- Hearts working under various types of stress function along different Frank-Starling curves.


normal
inotropic agent
or afterload
reduction
B
C
D
A
adequate
inadequate
depressed
contractility
pulmonary edema
Stroke Volume
10
20
Left Ventricular End-diastolic Pressure (mm Hg)

- Heart failure occurs when the heart cannot deliver adequate cardiac output to meet the metabolic needs of the body.
- Cardiac output can be calculated as the product of heart rate and stroke volume.
- The primary determinants of stroke volume are the *afterload* (pressure work), *preload* (volume work), and *contractility* (intrinsic myocardial function).

- Decreased myocardial contractility or abnormal loading conditions may cause heart failure.
- Abnormal loading conditions may be afterload (AS, PS, CoA)or preload (VSD, PDA, valvar insufficiency).
- Abnormalities in heart rate can also compromise cardiac output and produce both bradyarrhythmias and tachyarrhythmias.

- Systemic oxygen transport is calculated as the product of cardiac output and systemic oxygen content.
- Alterations in the oxygen-carrying capacity of blood (e.g., anemia or hypoxemia) also lead to a decrease in systemic oxygen transport and, if compensatory mechanisms are inadequate, can result in decreased delivery of substrate to tissues.

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

- Differential diagnosis of heart failure depends on age of presentation
- First weeks of life, excessive afterload
- Around 2 months, left to right shunts
- Any age, cardiomyopathies

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

Clinical Manifestations

- In children, the signs and symptoms include fatigue, effort intolerance, anorexia, abdominal pain, dyspnea, and cough.
- The elevation in systemic venous pressure may be gauged by clinical assessment of jugular venous pressure and liver enlargement.
- Edema is usually discernible in dependent portions of the body, or anasarca may be present.
- Orthopnea and basilar rales are variably present.
- Cardiomegaly is invariably noted.
- A gallop rhythm is common; when ventricular dilatation is advanced, the holosystolic murmur of mitral or tricuspid valve regurgitation may be heard.

- In infants, prominent manifestations include tachypnea, feeding difficulties, poor weight gain, excessive perspiration, irritability, weak cry and noisy, labored respirations with intercostal and subcostal retractions, as well as flaring of the alae nasi; wheezing is prominent.
- Hepatomegaly usually occurs, and cardiomegaly is invariably present, a gallop rhythm can frequently be recognized.
- Edema may be generalized and usually involves the eyelids as well as the sacrum and less often the legs and feet.

- Tachypnea, orthopnea, wheezing and pulmonary edema with left sided heart failure
- Hepatomegaly, edema and distended neck veins with right sided heart failure

- Chest x-ray shows cardiac enlargement.
- Pulmonary vascularity is variable and depends on the cause of the heart failure.
- With large left-to-right shunts , exaggeration of the pulmonary arterial vessels to the periphery
- With cardiomyopathy ,a relatively normal pulmonary vascular bed early in the course
- Fluffy perihilar pulmonary markings suggestive of venous congestion , with more severe degrees of heart failure.

- Electrocardiography may show chamber hypertrophy assessing the cause of heart failure.
- Low-voltage QRS morphologic characteristics with ST-T wave abnormalities may suggest myocardial inflammatory disease or pericarditis.
- Is the best tool for evaluating rhythm disorders.

- Echocardiographic techniques are useful in assessing dilated chamber sizes, decreased myocardial function, CHDs.
- Fractional shortening, 28 and 40%
- Ejection fraction, 55-65%
- Pre-ejection: ejection period, less than 40%

Treatment

- Rest
- Diet
- Diuretics
- Afterload reducers
- Inotropes
- B blockers

Therapy	Mechanism
GENERAL CARE	
Rest	Reduces cardiac output
Oxygen	Improves oxygenation in presence of pulmonary edema
Sodium, fluid restrictions	Decreases vascular congestion; decreases preload
DIURETICS	
Furosemide	Salt excretion by way of ascending loop of Henle; reduces preload; afterload reduced 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 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
Amrinone/milrinone	Nonsympathomimetic, noncardiac glycosides with inotropic effects; may cause vasodilation
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