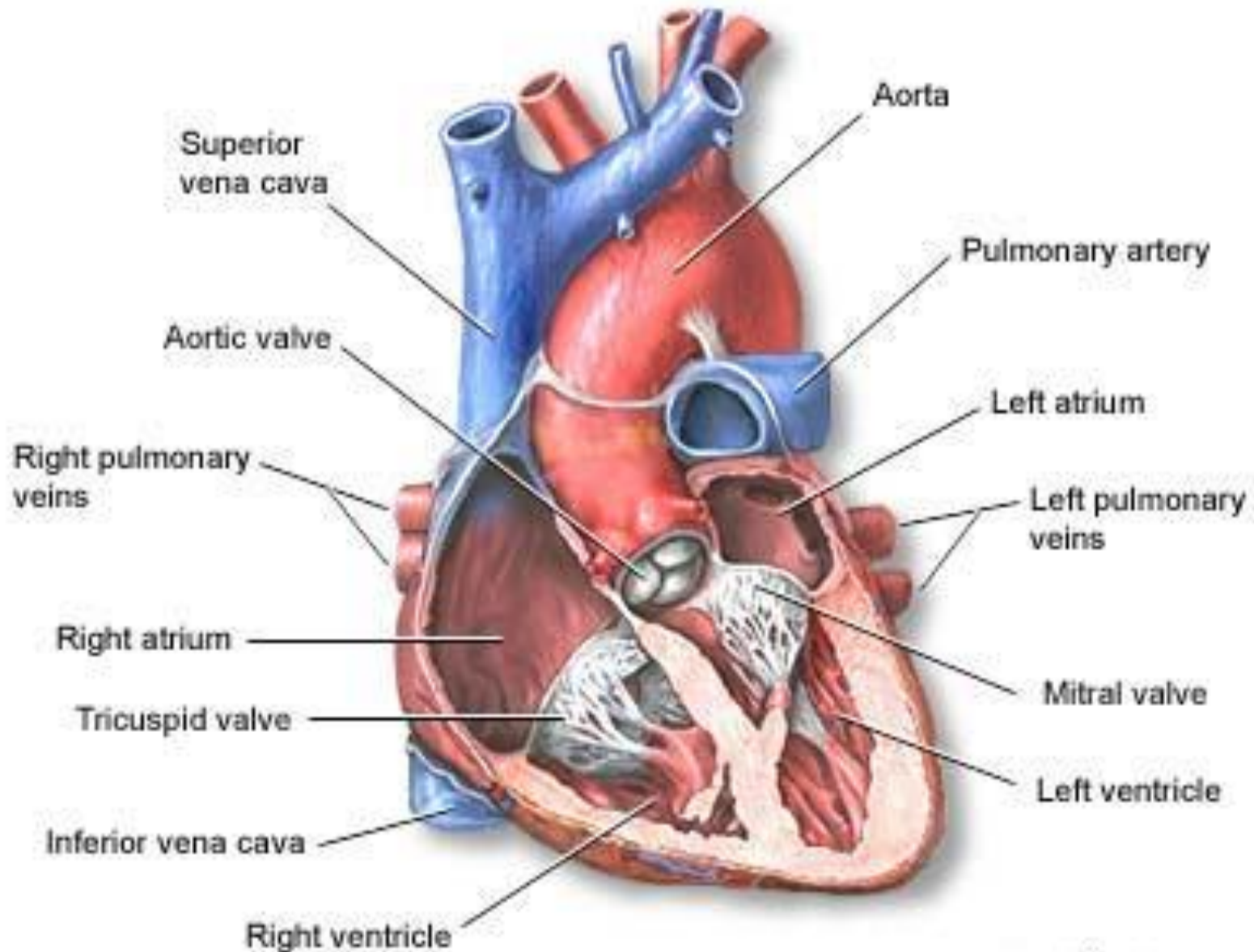



# Normal Cardiac Anatomy



# Heart Failure

- ▶ Impaired cardiac pumping
  - ▶ Results in vasoconstriction & fluid retention
  - ▶ Characterized by ventricular dysfunction, reduced exercise tolerance, diminished quality of life, & shortened life expectancy
  - ▶ Affects 5 million people
  - ▶ Most common reason for hospital admission in adults over age 65
- 

# Etiology

- ▶ Causes of HF may be divided into two subgroups:
  - Primary
  - Precipitating
  
- ▶ Classified : systolic or diastolic failure (or dysfunction): Right or left

# Heart Failure

## Systolic Heart Failure

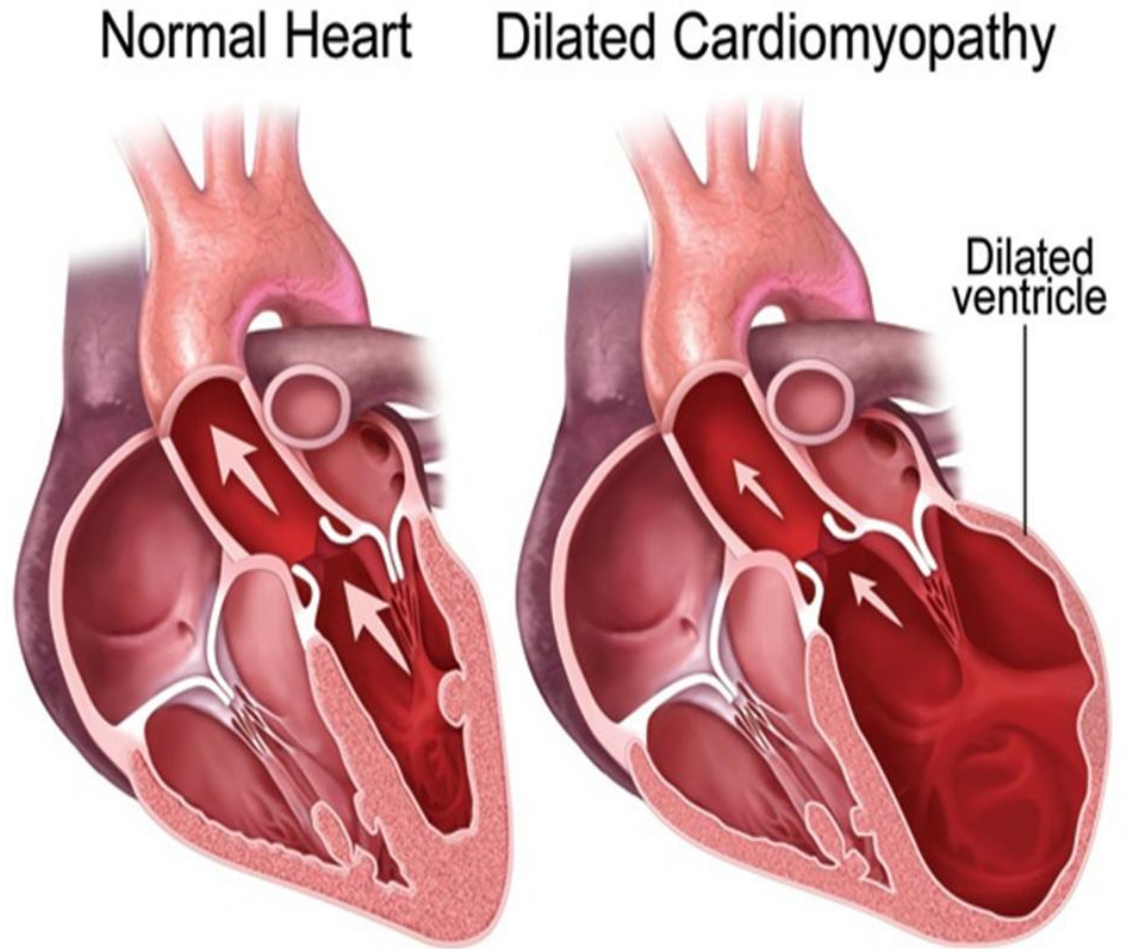
- ▶ LV: decreased function
  - Inability to pump blood forward
  - Thin-walled, dilated, hypertrophied
  - **Ejection Fraction decreased\***
    - Normal 60–70%
    - HF < 40%
- ▶ Causes: MI, CAD, HTN, CMP, Valvular dysfunction

## Diastolic Heart Failure

- ▶ Impaired relaxation/filling of ventricles in diastole ▶  
↓ SV ▶ ↓ CO; Stiff, non-compliant ventricles
  - **Normal EF**, Pulmonary congestion, Pulmonary HTN, LV hypertrophy(LVH)
- ▶ Causes: HTN, AS, HCM
  - ▶ Older adults, women

# Heart Failure: Mixed

- ▶ Poor systolic function
- ▶ Dilated ventricle
- ▶ Unable to relax
- ▶ Low EFs (<35%)
- ▶ High pulmonary pressures
- ▶ Biventricular failure
- ▶ ↓ BP, ↓ CO, ↓ renal perfusion, poor exercise tolerance
- ▶ dysrhythmias



# Pathophysiology

Compensatory mechanisms: **why?**

1. Sympathetic Nervous Activation (SNA)
2. Neurohormonal Response
3. Ventricular Dilation
4. Ventricular Hypertrophy

# 1. Sympathetic nervous system (SNS) activation

- *First and least effective mechanism*
- Release of catecholamines (epinephrine and norepinephrine)



- Increased heart rate (HR)
- Increased myocardial contractility
- Peripheral vasoconstriction

## 2. Neurohormonal responses

Kidneys release renin : **WHY**

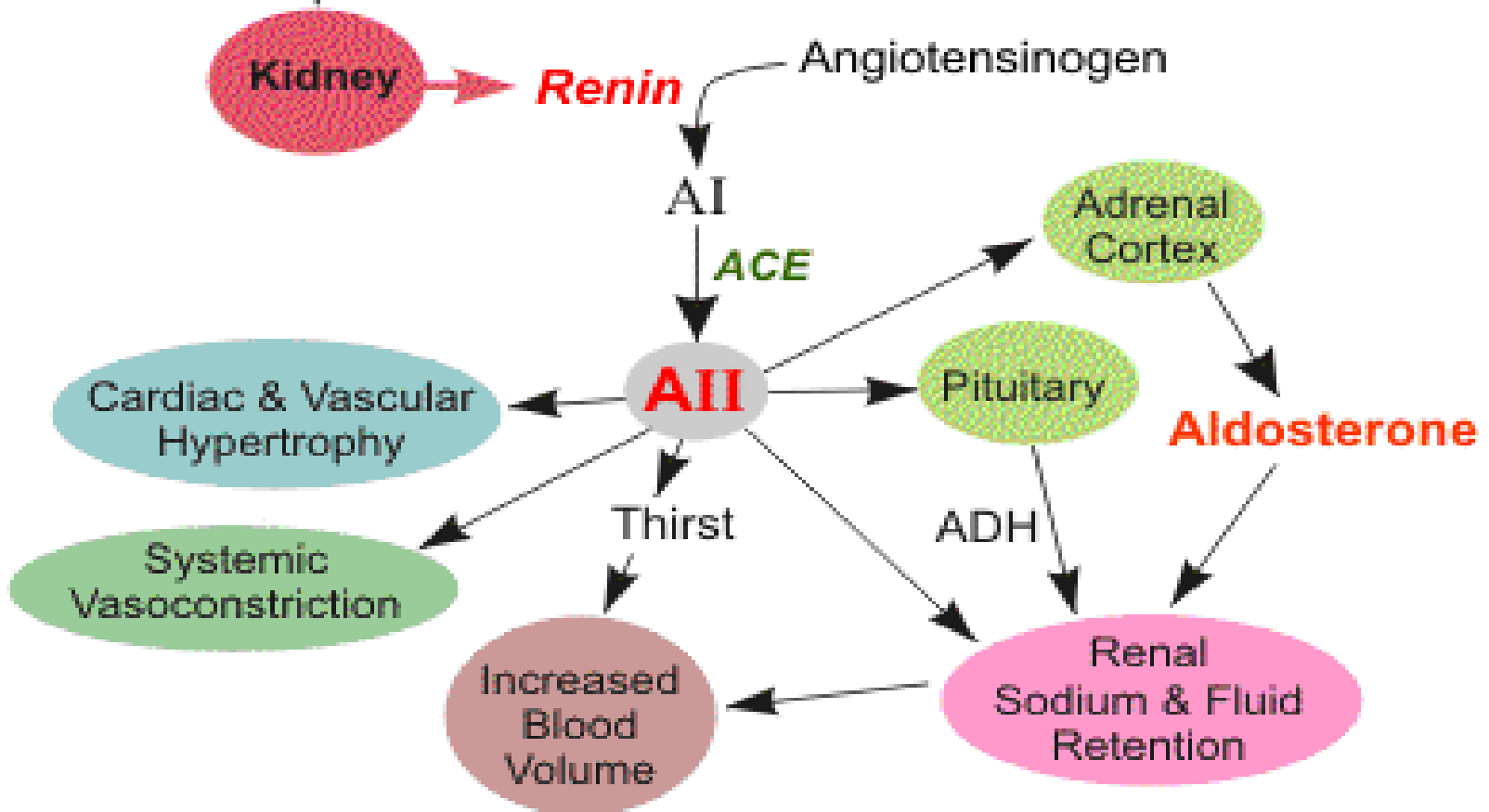
- Renin converts angiotensinogen to angiotensin II by a converting enzyme made in the lungs.

### *Angiotensin II Action:*

- Adrenal cortex to release aldosterone (sodium and water retention)
- Increased peripheral vasoconstriction (increases BP)

# Renin-Angiotensin-Aldosterone System- RAAS

Sympathetic Stimulation  
Hypotension  
Decreased Sodium Delivery



# 3. Ventricular Dilation

- Enlargement of the chambers of the heart that occurs when pressure in the left ventricle is elevated
- Initially an adaptive mechanism
- Eventually this mechanism becomes inadequate, and CO decreases.

# 4. Ventricular Hypertrophy

- *Increase* in *muscle mass* and cardiac wall *thickness* in response to chronic dilation:



- Poor contractility
- Higher O<sub>2</sub> needs
- Poor coronary artery circulation
- Risk for ventricular dysrhythmias

# Dilated & Hypertrophied Heart Chambers

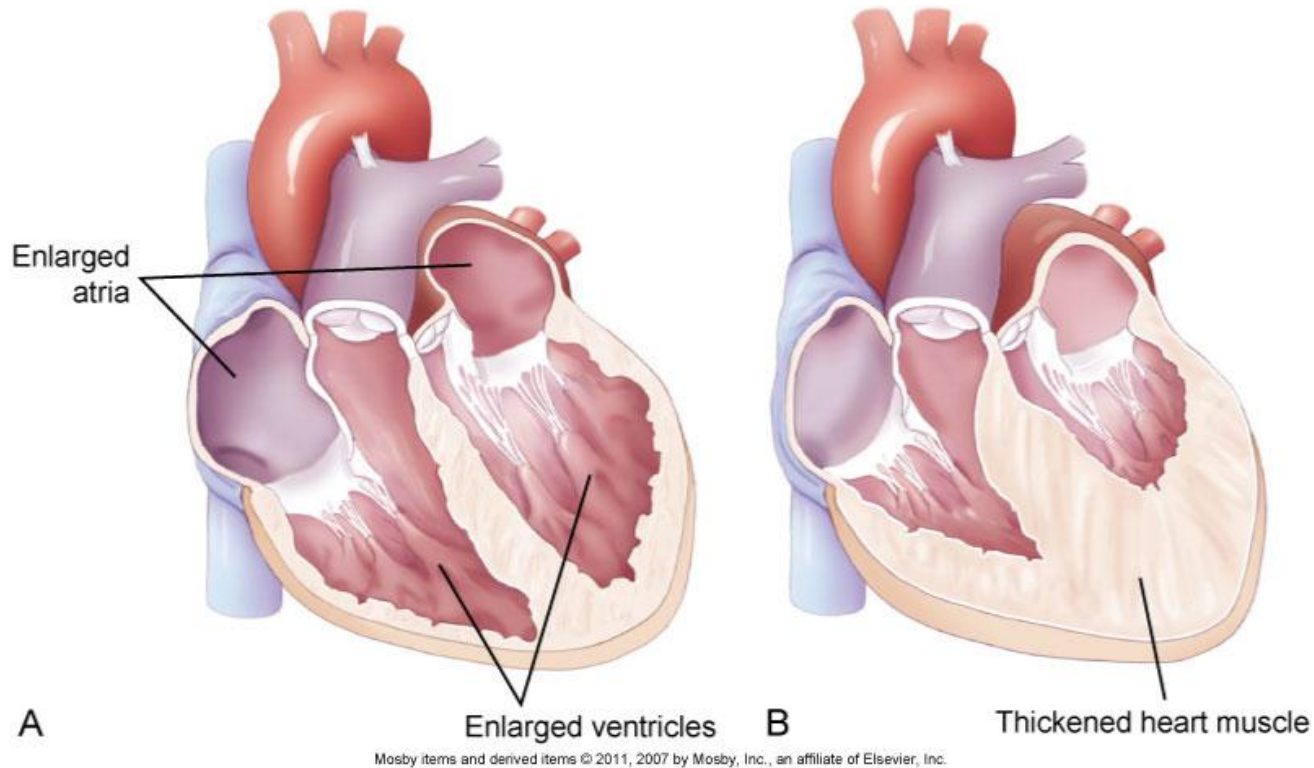


Fig. 35-1. **A**, Dilated heart chambers. **B**, Hypertrophied heart chambers.

# Counter regulatory processes

- Natriuretic peptides: Atrial natriuretic peptide (ANP), b-type natriuretic peptide (BNP) **normal = <100**

## Action:

- Promote venous and arterial vasodilation, reducing preload and afterload
  - Enhance diuresis
  - Block effects of the RAAS
  - Inhibit the development of cardiac hypertrophy with anti-inflammatory effects.
- 
- Nitric oxide (NO) from the vascular endothelium
- ## Action:
- relaxes arterial smooth muscle, resulting in vasodilation and decreased afterload.

# Types of Heart Failure

- ▶ Left-sided HF
- ▶ (most common) from left ventricular dysfunction (e.g., MI hypertension, CAD, cardiomyopathy)
  - Backup of blood into the left atrium and pulmonary veins
    - Pulmonary congestion
    - Edema

# Left Sided Heart Failure

## *Behaviors*

- ▶ Pulmonary congestion
  - Hacking cough, worse at noc
  - Dyspnea/breathlessness
  - Crackles or wheezes in lungs
  - Frothy, pink-tinged sputum
  - Tachypnea
  - S<sub>3</sub>/S<sub>4</sub> summation gallop
- ▶ Decreased CO
  - Fatigue
  - Weakness
  - Oliguria during the day
  - Nocturia during noc
  - Angina
  - Confusion, restlessness
  - Dizziness
  - Tachycardia, palpitations
  - Pallor
  - Weak peripheral pulses
  - Cool extremities

# Types of Heart Failure

- ▶ Right-sided HF from left-sided HF, Cor Pulmonale, right ventricular MI
  - Backup of blood into the right atrium and venous systemic circulation

# Right-Sided Heart Failure

## *Behaviors*

- ▶ Jugular venous distention
- ▶ Splenomegaly and Hepatomegaly
- ▶ Dependent edema (legs and sacrum)
- ▶ Ascites
- ▶ Swollen hands and feet
- ▶ Polyuria at noc
- ▶ Weight gain
- ▶ Increased BP (from excess volume)
- ▶ Decreased BP (from failure)

# Classification Systems

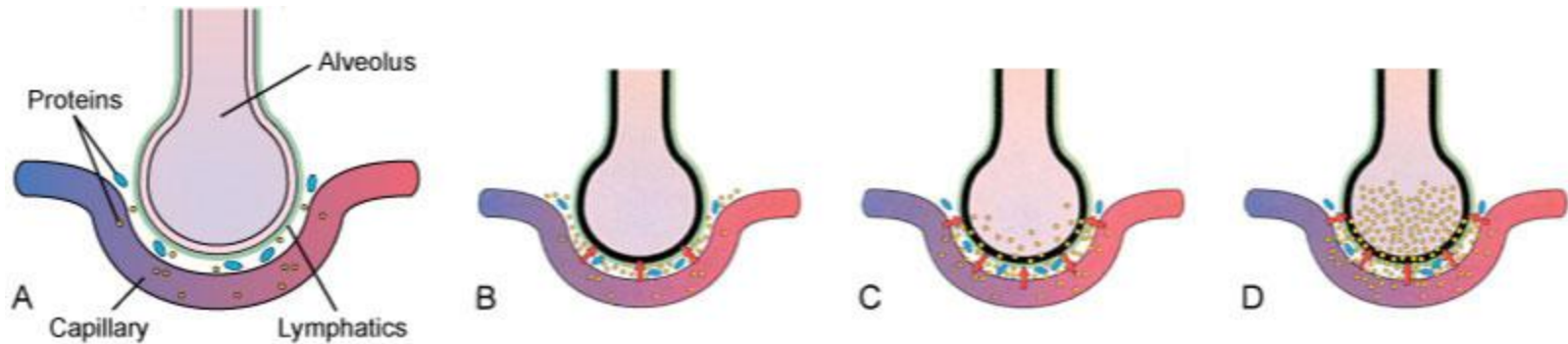
- ▶ New York Heart Association *Functional* Classification of HF
  - Classes I to IV
- ▶ ACC/AHA Stages of HF
  - Stages A to D

# Heart Failure

## *Acute Decompensation*

- ▶ Manifest as *Pulmonary Edema*
- ▶ Acute, life-threatening condition in which *alveoli fill with sero-sanguinous fluid*
- ▶ Engorgement of PV system & interstitial edema (V/Q mismatch)
- ▶ Lungs becomes less compliant w/increased resistance in small airways

# Pulmonary Edema



Modified from Urden LD, Stacy KM, Lough ME: *Thelan's critical care nursing: diagnosis and management*, ed 5, St Louis, 2006, Mosby.

Fig. 35-3. As pulmonary edema progresses, it inhibits oxygen and carbon dioxide exchange at the alveolar-capillary interface. **A**, Normal relationship. **B**, Increased pulmonary capillary hydrostatic pressure causes fluid to move from the vascular space into the pulmonary interstitial space. **C**, Lymphatic flow increases in an attempt to pull fluid back into the vascular or lymphatic space. **D**, Failure of lymphatic flow and worsening of left heart failure result in further movement of fluid into the interstitial space and into the alveoli.

# Clinical Manifestations: ADHF

- ▶ Physical findings
  - Orthopnea
  - Dyspnea, tachypnea
  - Use of accessory muscles
  - Cyanosis
  - Cool and clammy skin

# Clinical Manifestations: ADHF

- ▶ Physical findings
  - Cough with frothy, blood-tinged sputum
  - Breath sounds: Crackles, wheezes, rhonchi
  - Tachycardia
  - Hypotension or hypertension