Etiology and Pathophysiology

- **Myocardial Infarction**
  - **Subendocardial MI**
    - **The damage has not penetrated through the entire thickness**

Etiology and Pathophysiology

- **Myocardial Infarction**
  - Infarctions are described by the area of occurrence

Etiology and Pathophysiology
Healing Process

- **Within 24 hours, leukocytes infiltrate the area of cell death**

- **Enzymes are released from the dead cardiac cells (important indicators of MI)**

Etiology and Pathophysiology
Healing Process

- **Proteolytic enzymes of neutrophils and macrophages remove all necrotic tissue by 2nd or 3rd day**

- **Development of collateral circulation improves areas of poor perfusion**

Etiology and Pathophysiology
Healing Process

- **Necrotic zone identifiable by ECG changes and nuclear scanning**
- **10 to 14 days after MI, scar tissue is still weak**

Etiology and Pathophysiology
Healing Process

- **By 6 weeks after MI, scar tissue has replaced necrotic tissue**
  - **Area is said to be healed**

Etiology and Pathophysiology
Healing Process

- *Ventricular remodeling*
  - In an attempt to compensate for the infarcted muscle, the normal myocardium will hypertrophy and dilate

Types of Angina
Silent Ischemia

- Up to 80% of patients with myocardial ischemia are asymptomatic
- Associated with diabetes mellitus and hypertension

Types of Angina
Prinzmetal's Angina

- **When spasm occurs:**
  - Pain
  - Marked, transient ST segment elevation with angina (unlike with AMI; ↑ST = MI)
  - May occur during REM sleep
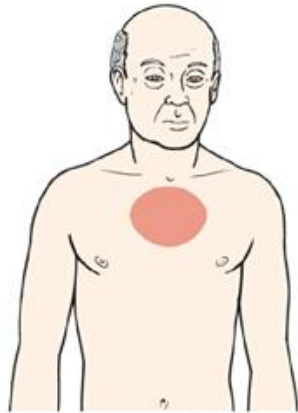
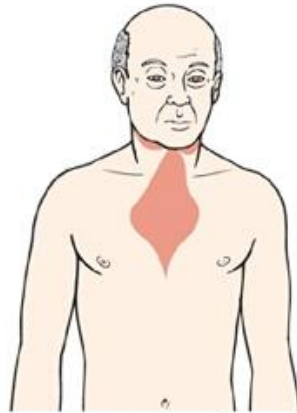# Clinical Manifestations
Myocardial Infarction

- **Pain**
  - Severe, immobilizing chest pain not relieved by rest, position change, or nitrate administration
    - **The hallmark of an MI**

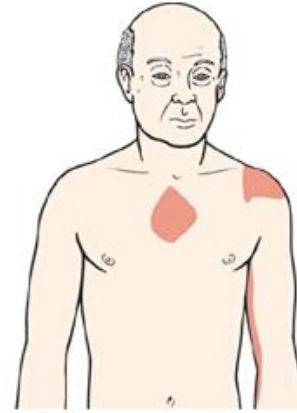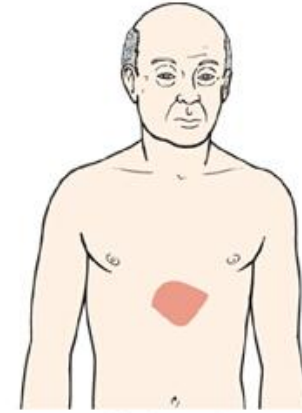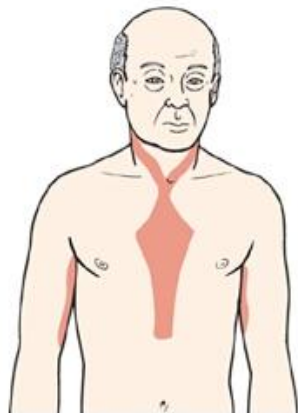# Location of Chest Pain

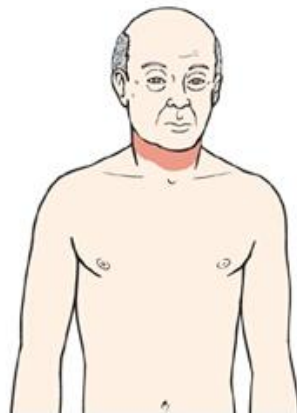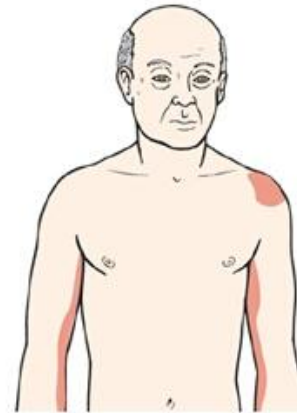Upper chest

Substernal radiating to neck and jaw

Substernal radiating down left arm

Epigastric

Epigastric radiating to neck, jaw, and arms

Neck and jaw

Left shoulder and down both arms
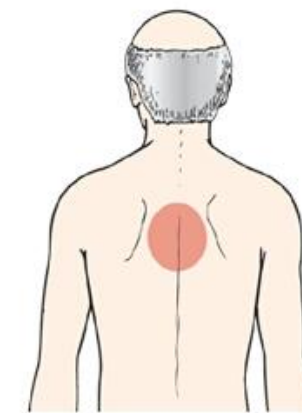
Intrascapular

Fig. 33-12

Clinical Manifestations
Myocardial Infarction

- **Nausea and vomiting**
  - Can result from reflex stimulation of the vomiting center by the severe pain

Clinical Manifestations
Myocardial Infarction

- **Sympathetic nervous system stimulation**
  - ↑ catecholamines released during initial phases of MI

Clinical Manifestations
Myocardial Infarction

- **Fever**
  - May ↑ within 1ˢᵗ 24 hours up to 100.4°
  - May last as long as 1 week

Clinical Manifestations
Myocardial Infarction

- **Fever**
  - Systemic manifestation of the inflammatory process caused by cell death

Clinical Manifestations
Myocardial Infarction

- **Cardiovascular manifestations**
  - $\uparrow$ BP and heart rate initially
  - Later the BP may drop from $\downarrow$ CO

Clinical Manifestations
Myocardial Infarction

- **Cardiovascular manifestations**
  - ↓ urine output
  - Crackles
  - Hepatic engorgement
  - Peripheral edema

Complications of Myocardial Infarction

- **Arrhythmias**
  - Most common complication
  - Present in 80% of MI patients
  - Most common cause of death in the prehospital period

Complications of Myocardial Infarction

- **Congestive heart failure**
  - A complication that occurs when the pumping power of the heart has diminished

Complications of Myocardial Infarction

- **Cardiogenic shock**
  - Occurs when inadequate oxygen and nutrients are supplied to the tissues because of severe LV failure
  - Requires aggressive management

Complications of Myocardial Infarction

- **Papillary muscle dysfunction**
  - Causes mitral valve regurgitation
  - Condition aggravates an already compromised LV

Complications of Myocardial Infarction

- **Ventricular aneurysm**
  - Results when the infarcted myocardial wall becomes thinned and bulges out during contraction

Complications of Myocardial Infarction

- **Pericarditis**
  - An inflammation of the visceral and/or parietal pericardium
  - May result in cardiac compression, ↓ LV filling and emptying, and cardiac failure

Complications of Myocardial Infarction

- **Pulmonary embolism**
  - Source of the thrombus may be the roughened endocardium or leg veins

Diagnostic Studies
Myocardial Infarction

- **History of pain**
- **Risk factors**
- **Health history**
- **ECG: ST elevation, greater than 1 mm above PR Interval; T Wave inversion (flipped T Waves); Pathological Q-wave (Q wave greater than ¼ size of R wave)**
- **Serum cardiac markers:**
  - ➤ **CK-MB: indicates muscle damage (rises 3-12 hours post AMI – returns to normal 2-3 days)**
  - ➤ **Triponen: is a myocardial muscle protein (rises as quickly as CK; remains elevated for 2 weeks)**
  - ➤ **Myoglobin: rises 3 hours after AMI; lacks cardiac specificity**

Women and
Coronary Artery Disease

- **Diabetes mellitus found to be the single most powerful predictor of CAD in women**