

### N102-Manifestations of CAD

Disorder	Etiology	Signs & Symptoms	Collaborative Care Meds-P. 744 Table 34-11 P. 743 Figure 34-5	Complications
<b>Stable Angina</b>	Decreased O2 supply vs. demand Lack of O2 supply is temporary & reversible (ischemia) <b>Chest pain that occurs over long period with same pattern of onset, duration, and intensity</b>	<b>Chest pain or discomfort</b> lasting 5-15 minutes, Constrictive or squeezing, Vague sensation, Ache in chest, almost never sharp or stabbing, may complain of indigestion or burning <b>Predictable</b> pattern, avoid triggers <b>Provoked</b> by exertion & relieved by rest	<b>Goal</b> -increase O2 supply & relieve pain <b>Meds</b> -Antiplatelet/ Anticoagulant Nitrates- Beta blockers Calcium Channel Blockers(CCB)-if BB poorly tolerated or Prinzmetal's Angina ACE Inhibitors/ARBs Lipid Lowering Drugs <b>Diet</b> -low salt, cholesterol, & saturated fat <b>Manage risk factors</b> for CAD <b>Education &amp; Exercise</b> <b>Flu Vaccine</b> <b>PCI or CABG</b>	Stable Angina becomes unstable and progresses to ACS (unstable angina, NSTEMI, STEMI)
<b>Unstable Angina (ACS)</b> (ischemia)	Ischemia <b>prolonged not immediately reversible</b> Deterioration of once stable atherosclerotic plaque by rupture, platelet aggregation, or thrombus <b>Partial occlusion</b> by thrombus of coronary artery	<b>Chest pain</b> is new in onset, occurs at rest, or worsens, <b>unpredictable</b> <b>Progresses</b> in past few hours, days, weeks, often ends in pain @ rest Previous stable angina progresses changes pattern <b>Occurs @ rest or with minimal exertion</b> <b>Women</b> often report fatigue, SOB, anxiety, or indigestion	<b>Goal</b> -increase O2 supply & relieve pain Oxygen therapy, IV, ECG <b>Meds</b> -Antiplatelet/ Anticoagulant Nitrates, Morphine Beta blockers Calcium Channel ACE Inhibitors/ARBS Lipid Lowering Drugs <b>Diet</b> -low salt, cholesterol, & saturated fat <b>Manage risk factors</b> for CAD <b>Education , Exercise, Flu Vaccine, PCI</b>	Unstable angina progresses to infarction with myocardial cell death

Disorder	Etiology	Signs & Symptoms	Collaborative Care Meds-P. 744 Table 34-11 P. 743 Figure 34-5	Complications
<p><b>Myocardial Infarction (ACS)</b> <b>NSTEMI</b> (ischemia) &amp; <b>STEMI</b> (infarction)</p>	<p><b>Sustained ischemia &gt;20 minutes causes cell death</b> Degree of altered function depends on <b>size and area of infarction &amp; pre-established collateral circulation</b> Described by <b>area of occurrence</b>(anterior, inferior, posterior, lateral) Enzymes infiltrate area of cell death within 24 hrs. Proteolytic enzymes of neutrophils &amp; macrophages remove necrotic tissue by 2<sup>nd</sup> or 3<sup>rd</sup> day Development of collateral circulation improves areas of poor perfusion Necrotic zone identified by ECG changes 10-14 days necrotic tissues still weak <b>By 6 weeks</b> scar tissue has replaced necrotic tissues-area said to be healed</p>	<p><b>Pain</b> described as heaviness, constriction, tightness, pressure, or crushing <b>not relieved by rest, position change, or nitrates</b> <b>Some women</b> may complain of SOB or fatigue although women more similar with men in MI symptoms <b>Common locations</b> substernal, retrosternal, or epigastric, may radiate <b>SNS stimulation</b>-glycogen, diaphoresis, peripheral vasoconstriction, skin ashen, clammy, &amp;/or cool to touch <b>CV</b>-increase HR BP then decrease BP due to decrease CO, crackles, JVD, peripheral edema, S3 S4, new Murmur <b>N&amp;V</b> from reflex stimulation of vomiting center or vasovagal reflex <b>Fever</b> systemic inflammatory process, temp may increase first 24 hrs. up to 100.4 &amp; may last up to 1 wk.</p>	<p><b>Goal-</b></p> <ul style="list-style-type: none"> <li>• <b>increase O2 supply</b></li> <li>• <b>decrease workload</b></li> <li>• <b>preserve myocardium</b></li> <li>• <b>relieve pain</b></li> </ul> <p>Oxygen therapy 12 Lead ECG IV access <b>Meds-</b> Antiplatelet/Anticoagulation Nitrates- Morphine Beta blockers ACE Inhibitors/ARBS- Anti-dysrhythmics Lipid Lowering Drugs Stool softeners <b>Diet</b>-low salt, cholesterol, &amp; saturated fat <b>Manage risk factors</b> for CAD <b>Education &amp; Exercise</b> <b>Cardiac Rehab</b> <b>Flu Vaccine</b> <b>Emergent Reperfusion Therapy-</b> PCI, Thrombolytics, CABG</p>	<p><b>Ventricular Remodeling</b> <b>Dysrhythmias</b>-most common complication <b>Heart Failure</b> <b>Cardiogenic Shock</b> <b>Ventricular Aneurysm</b> <b>Acute Pericarditis</b> <b>Dressler Syndrome</b> <b>Pulmonary Embolism-</b></p>

- **PQRST Assessment of Angina (page 741)**
  - Precipitating events-events activities precipitated pain i.e. argument, exercise, and rest?
  - Quality of pain-What does pain feel like i.e. squeezing, tight, heaviness?
  - Radiation of Pain-Where is the pain located-does it radiate to other areas? P. 741
  - Severity of Pain-On 0-10 scales what number would you rate the pain?
  - Timing-When did it begin, has it changed over time, have you had it before?
- **Precipitating Factors of Angina**
  - Physical exertion
  - Temperature extremes
  - Strong emotions
  - Heavy meal consumption
  - Sexual activity
  - Stimulants i.e. cocaine, amphetamines, for some caffeine
  - Circadian Rhythm Patterns-tends to occur early am after awakening
- **CAD**
  - Major cause atherosclerosis, CRP
  - Developmental stages
    - Fatty Streak-lipid filled smooth muscle cells earliest lesions
    - Fibrous Plaque-Collagen covers the fatty streak, vessel lumen narrows reduces blood flow, fissures can develop
    - Complicated Lesion-Plaque rupture, thrombus formation, further narrowing or total occlusion of vessel
  - Risk Factors
    - Non-modifiable-age, gender, ethnicity, genetic predisposition
    - Modifiable-Serum Lipids, BP, Diabetes, Hypertension, Tobacco, Obesity, Inactivity