Coronary Artery Disease
and
Acute Coronary Syndrome (MI)
OXYGENATION NEEDS

• Ventilation
• Perfusion
• Diffusion

*What concept is involved?
*Your patient reports having CP, what do you do?
*What nursing physical assessments are involved?
Coronary Artery Disease (CAD)

Atherosclerosis – major cause of CAD

- Begins as soft deposits of fat that harden with age
- Referred to as “hardening of arteries”
- Characterized by lipid deposits within intima of artery
- Endothelial injury and inflammation play a major role in development

Stages of Atherosclerosis (Fig. 33-1 pg. 703)
Audience Response Question

Which patient is *most* at risk for developing coronary artery disease?

a. A hypertensive patient who smokes cigarettes
b. An overweight patient who uses smokeless tobacco
c. A patient who has diabetes and uses methamphetamines
d. A sedentary patient who has elevated homocysteine levels
Risk Factors for CAD

• Nonmodifiable risk factors
  – Age
  – Gender
  – Ethnicity
  – Family history
  – Genetic predisposition
Risk Factors for CAD

• Major modifiable risk factors
  – **Elevated serum lipids**
    • Cholesterol >200 mg/dL (5.2 mmol/L)
    • Triglycerides >150 mg/dL (3.7 mmol/L)
    • High-density lipoproteins (HDL)
    • Low-density lipoproteins (LDL)
  – **Hypertension**
  – **Tobacco use**
    • Second-hand smoke
    • All lead to vessel inflammation and thrombosis
  – **Physical inactivity**
  – **Obesity**

• Contributing modifiable risk factors
  – Diabetes
  – Metabolic syndrome
  – Psychologic states
  – Homocysteine level
  – Substance abuse

“Arterial damage”

• RN has major role in teaching health-promoting behaviors
Audience Response Question

The nurse determines that teaching about implementing dietary changes to decrease the risk of CAD has been effective when the patient says,

a. “I should not eat any red meat such as beef, pork, or lamb.”
b. “I should have some type of fish at least 3 times a week.”
c. “Most of my fat intake should be from olive oil or the oils in nuts.”
d. “If I reduce the fat in my diet to about 5% of my calories, I will be much healthier.”
Coronary Artery Disease
Etiology and Pathophysiology

• C-reactive protein (CRP)
  – Protein produced by liver
  – Nonspecific marker of inflammation
  – Increased in many patients with CAD
  – Chronic exposure to CRP linked with unstable plaques and oxidation of LDL cholesterol
Coronary Artery Disease
Etiology and Pathophysiology

- Collateral circulation
  - Arterial anastomoses within coronary circulation
  - Increased with chronic ischemia
  - May be inadequate with rapid-onset CAD
  - Old vs. young
Clinical Manifestations of CAD
Angina = Chest Pain

- Chronic and progressive disease
- Can be acute for varying reasons, **important to rule out MI**
- O2 demand > O2 supply → myocardial ischemia
- Angina = clinical manifestation
  - Occurs when arteries are blocked 70% or more
  - 50% or more for left main coronary artery
Some patients, especially women and older adults, report atypical symptoms of angina including dyspnea, nausea, and/or fatigue.
Chronic Stable Angina
Types of Angina

• **Silent ischemia**
  – Diabetic neuropathy
  – Confirmed by ECG changes
    • ST segment depression and/or T-wave inversion
• **Prinzmetal’s (variant) angina**
  – Occurs at rest
  – Spasm of a major coronary artery (calcium channel blockers)
  – CAD may or may not be present

• **Microvascular angina**
  – CP occurs in absence of significant CAD or coronary spasm of a major coronary artery
  – CP related to myocardial ischemia associated with atherosclerosis or spasm of the small distal branch vessels of the coronary microcirculation
  – Prevention and treatment follows CAD recommendations
Relationships among coronary artery disease, chronic stable angina, and acute coronary syndrome.
Clinical Manifestations of CAD
Chronic Stable Angina

• Intermittent CP that occurs over a long period with same pattern of onset, duration, and intensity of symptoms
• Provoked by physical exertion, stress, emotional upset
• Few minutes in duration, subsides when precipitating factors resolved
• Control with drugs (i.e. nitroglycerin)
Nursing and Interprofessional Care: CAD

• Physical fitness – no matter what age, but will differ
  – FITT formula: 30 minutes most days plus weight training 2 days a week
  – Regular physical activity contributes to
    • Weight reduction
    • Reduction of >10% in systolic BP
    • Increase in HDL cholesterol
Nursing and Interprofessional Care: CAD

• Nutritional therapy (Lower LDL)
  – ↓ Saturated fats and cholesterol
  – ↓ Red meat, egg yolks, whole milk
  – ↓ alcohol and simple sugars

• ↑ Complex carbohydrates and fiber
  (whole grains, fruits, vegetables, and fiber)

• ↑ Omega-3 fatty acids
  (AHA recommends eating tofu, soybean, canola, walnut, flaxseed because these products contain alpha-linolenic acid, which becomes omega-3 fatty acid in the body.)
Nursing and Interprofessional Care: CAD – DRUG THERAPY

• Lipid-lowering drug therapy – **If diet and exercise ineffective**
  – Statins – atorvastatin (Lipitor), simvastatin (Zocor)
    • Inhibit cholesterol synthesis, decrease LDL, increase HDL
    • Monitor for liver damage and myopathy

• Antiplatelet therapy
  – ASA (81 mg or 325 mg)
  – Clopidogrel 75 mg

*Explain the mechanism of action*
Chronic/Unstable Angina
Interprofessional Care: Drug Therapy

• Goal: ↓ O₂ demand and/or ↑ O₂ supply
• Short-acting nitrates SL NTG
  – Dilate peripheral and coronary blood vessels
  – Give sublingually or by spray
  – If no relief in 5 minutes, call EMS; if some relief, repeat every 5 minutes for maximum 3 doses
  – Patient teaching
  – Can use prophylactically

• Long-acting nitrates – oral, NTG ointment, Transdermal controlled-release NTG
  – To reduce angina incidence
  – Main side effects: headache (Tylenol), orthostatic hypotension
Chronic Stable Angina
Interprofessional Care: Drug Therapy

• ACE and ARBs:
  – benazepril (Lotensin)
  – enalapril (Vasotec)
  – lisinopril (Zestril)
  – losartan (Cozaar)

• β-Blockers:
  – atenolol (Tenormin)
  – metoprolol (Lopressor)

• Calcium channel blockers:
  – diltiazem (Cardizem)
  – amlodipine (Norvasc)
  – nifedipine (Procardia)

• Lipid lowering drugs
  – atorvastatin (Lipitor)
  – simvastatin (Zocor)

• Antiplatelets
  – aspirin
  – clopidogrel
Chronic Stable/Unstable Angina
Interprofessional Care: Diagnostic Studies

- Chest x-ray
- 12-lead ECG
- Laboratory studies – lipid profile, CRP, BMP, CBC coags, cardiac enzymes
- Echocardiogram
- Exercise stress test (physical/pharmacologic)
  - For patients with abnormal but nondiagnostic ECG and negative biomarkers
- Coronary CT Angiography (CCTA)
Chronic Stable/Unstable Angina Nursing/Interprofessional Care: Diagnostic Studies

- Cardiac catheterization/coronary angiography
  - Visualize blockages (diagnostic)
  - Open blockages (interventional)
    - Percutaneous coronary intervention (PCI)
    - Balloon angioplasty
    - Stent
- *For patients with a STEMI
- *Not for patients with UA or NSTEMI
Pre-PCI and Post-PCI With Stent Placement Fig 33-6 pg. 718
Nursing Management
Coronary revascularization: PCI

– Monitor for recurrent angina
– Frequent VS –per protocol, including cardiac rhythm
– Monitor catheter insertion site for bleeding
  • Want to achieve hemostasis (sandbags)
– Neurovascular assessment
– Bed rest per institutional policy
Nursing Management
Chronic Stable/Unstable Angina

• Acute Intervention
  – Upright position
  – Supplemental oxygen
  – Assess vital signs, cardiac monitor, IV access
  – 12-lead ECG
  – Administer NTG followed by an opioid analgesic, if needed
  – Assess heart and breath sounds
  – Admission to hospital for observation
    • Interventional cardiac catheterization
Nursing Management
Chronic Stable/Unstable Angina

• Ambulatory Care
  – Provide reassurance, decrease stress response
  – Patient teaching
    • CAD and angina
    • Precipitating factors for angina
    • Risk factor reduction
    • Drugs (substance abuse)
Acute Coronary Syndrome Etiology and Pathophysiology

• Result
  – Partial occlusion of coronary artery: UA or NSTEMI
  – Total occlusion of coronary artery: STEMI
Clinical Manifestations of ACS
Unstable Angina

• New in onset, occurs at rest
• Increase in frequency, duration, or with less effort
• Pain lasting ≥ 10 minutes
• Needs immediate treatment that is different from unstable angina
• Symptoms in women often under-recognized as heart related

• Severe CP not relieved by rest, position change, or nitrate administration
  • Heaviness, pressure, tightness, burning, constriction, crushing
  • Substernal or epigastric
  • May radiate to neck, lower jaw, arms, back
  – Often occurs in early morning
  – Atypical in women, elderly
  – No pain if cardiac neuropathy (diabetes)
Clinical Manifestations of ACS
Myocardial Infarction (MI)

• Result of abrupt stoppage of blood flow through a coronary artery, causing irreversible myocardial cell death (necrosis)
  – Preexisting CAD
  – STEMI - occlusive thrombus
  – NSTEMI - non-occlusive thrombus

• **ST-elevation and non-ST-elevation**
  – Changes in QRS complex, ST segment, and T wave
  – Serial ECGs reflect evolution of MI
  – Distinguish between STEMI and NSTEMI/UA for dx and tx purpose
Unstable Angina and MI Diagnostic Studies

**STEMI**
- Usually have a complete coronary occlusion.
- ST elevation is first seen on the 12-lead ECG.
- Within few hours to days, T-wave inversion and pathologic Q waves develop.

**NSTEMI or UA**
- Usually have transient thrombosis or incomplete coronary occlusion.
- These patients often develop ST depression or T wave inversion on the initial ECG.
- They usually do not develop pathologic Q waves.
Normal ECG
Myocardial Infarction From Occlusion
(Fig 33-11 pg. 724)
Clinical Manifestations of ACS
Myocardial Infarction

• Catecholamine release and stimulation of SNS
  – Diaphoresis
  – Increased HR and BP
  – Skin: ashen, clammy, and/or cool to touch

• Cardiovascular
  – Initially, ↑ HR and BP, then ↓ BP (secondary to ↓ in CO)
  – Crackles, JVD
  – Abnormal heart sounds
    • S3 or S4
    • New murmur

• Nausea and vomiting
  – Reflex stimulation of the vomiting center by severe pain
  – Vasovagal reflex

• Fever
  – Up to 100.4° F (38° C) in first 24-48 hours
  – Systemic inflammatory process caused by heart cell death
Myocardial Infarction Healing Process

- Within 24 hours, leukocytes infiltrate the area of cell death
- Neutrophils and macrophages begin to remove necrotic tissue by fourth day → thin wall
- Necrotic zone identifiable by ECG changes
- Collagen matrix laid down
- 10 to 14 days after MI, scar tissue is still weak

- **Heart muscle vulnerable to stress**
- Monitor patient carefully as activity level increases
- By 6 weeks after MI, scar tissue has replaced necrotic tissue
  - Area is said to be healed, but less compliant

- **Ventricular remodeling**
  - Normal myocardium will hypertrophy and dilate in an attempt to compensate for infarced muscle
Complications of Myocardial Infarction

• **Dysrhythmias**
  – Most common complication
  – **Can be caused by ischemia, electrolyte imbalances, or SNS stimulation**
  – VT and VF are most common cause of death in prehospitalization period

• **Heart failure**
  – Pumping power of heart has diminished
  – Left-sided HF
  – Right-sided HF

• **Cardiogenic shock**
  – Severe LV failure, papillary muscle rupture, ventricular septal rupture, LV free wall rupture, right ventricular infarction
  – Requires aggressive management
    • Associated with a high death rate
Complications of Myocardial Infarction

• **Acute pericarditis**
  – Inflammation of pericardium
  – Mild to severe chest pain
    • Increases with inspiration, coughing, movement of upper body
    • Relieved by sitting in forward position
  – Pericardial friction rub

• **Papillary muscle dysfunction or rupture**
  – Causes mitral valve regurgitation

• **Left ventricular aneurysm**
  – Myocardial wall becomes thinned and bulges out during contraction
  – Leads to HF, dysrhythmias, and angina

• **Ventricular septal wall rupture and left ventricular free wall rupture**
  – HF and cardiogenic shock
  – Emergency repair
Unstable Angina and MI Diagnostic Studies

• Cardiac Biomarkers
  – Troponin
    • Rises within 4-6 hours, peaks 10-24 hours, detected for up to 10-14 days
  – Creatine kinase (CK)
    • CK-MB cardiac specific
      • Rises in 3-6 hours, peaks in 12-24 hours, returns to baseline within 12-48 hours
  – Serial Cardiac Biomarkers + ECG (i.e. Q8 hrs)
Interprofessional Care
Acute Coronary Syndrome

• **Initial interventions**
  
  – 12-lead ECG
  – Upright position
  – Oxygen – keep \( \text{O}_2 \text{ sat} > 93\%
  – IV access
  – Nitroglycerin (SL) and ASA (PO/PR)
  – Statin
  – Morphine
Interprofessional Care
Acute Coronary Syndrome

• Ongoing monitoring – admission to hospital
  – Treat dysrhythmias
  – Frequent vital sign monitoring
  – Bed rest/limited activity for 12–24 hours

• UA or NSTEMI
  – Dual antiplatelet therapy – aspirin, clopidogrel (Plavix), and heparin
  – Cardiac catheterization with PCI once stable
  – Reperfusion therapy
Interprofessional Care
Acute Coronary Syndrome

• Emergent PCI
  – Treatment of choice for confirmed STEMI
  – Goal: 90 minutes from door to catheter laboratory
  – Balloon angioplasty + stent(s)
  – Many advantages over Coronary Artery Bypass Graft (CABG) – surgical intervention
Interprofessional Care
Acute Coronary Syndrome

- **Thrombolytic therapy**
  - Only for patients with a STEMI
    - Agencies that do not have cardiac catheterization resources
  - Given IV within 30 minutes of arrival to the ED
  - Patient selection critical due to bleeding complications
  - Draw blood and start 2–3 IV sites: *what blood studies will you draw?*
  - Administer according to protocol
  - Monitor closely for signs of bleeding
  - Assess for signs of reperfusion
    - Cardiac rhythm –return of ST segment to baseline best sign, VS, cardio/pulmonary and *neurological* assessment

- **IV heparin to prevent reocclusion**
  - Heparin gtt (25,000 units/500ml NS or D5W)
Interprofessional Care
Acute Coronary Syndrome

• Surgical revascularization if:
  – Failed medical management
  – Presence of left main coronary artery or three-vessel disease
  – Not a candidate for PCI (e.g., blockages are long or difficult to access)
  – Failed PCI with ongoing chest pain
  – History of diabetes mellitus, LV dysfunction, chronic kidney disease
A patient is admitted to the coronary care unit following a cardiac arrest and successful cardiopulmonary resuscitation. When reviewing the health care provider’s admission orders, which order should the nurse question?

a. Oxygen at 4 L/min per nasal cannula
b. Morphine sulfate 2 mg IV every 10 minutes until the pain is relieved
c. Tissue plasminogen activator (tPA) 100 mg IV infused over 3 hours
d. IV nitroglycerin at 5 mcg/minute and increase 5 mcg/minute every 3 to 5 minutes
Interprofessional Care

Acute Coronary Syndrome – Traditional coronary artery bypass graft (CABG) surgery

- Requires sternotomy and cardiopulmonary bypass (CPB)
- Uses arteries and veins for grafts
  - The internal mammary artery (IMA) is most common artery used for bypass graft
  - Radial artery is another potential graft
Nursing Management
Acute Coronary Syndrome

• Complications related to CPB
  – Bleeding and anemia from damage to RBCs and platelets
  – Fluid and electrolyte imbalances
  – Hypothermia as blood is cooled as it passes through the bypass machine
  – Infections
Nursing Management
Acute Coronary Syndrome

• CABG: postoperative nursing care
  – ICU for first 24–36 hours
  – Pulmonary artery catheter
  – Intraarterial line
  – Pleural/mediastinal chest tubes
  – Continuous ECG
  – ET tube with mechanical ventilation
  – Epicardial pacing wires
  – Urinary catheter
  – NG tube

  – Assess patient for bleeding
  – Monitor hemodynamic status
  – Assess fluid status (I&Os)
  – Replace blood and electrolytes PRN
  – Restore temperature
  – Monitor for atrial fibrillation
    (which is common)
  – Surgical site care
    • Radial artery harvest site
    • Leg incisions
    • Chest incision
  – Pain management
  – DVT prevention
  – Pulmonary hygiene
  – Cognitive dysfunction

• Begin cardiac rehabilitation
Nursing Management
Acute Coronary Syndrome

• This is a postoperative patient – basic individualized care on its own despite the type of surgical intervention done. What else will you anticipate as far the progression of this patient with regards to other body systems (i.e. diet, activity, elimination, etc.)?
Interprofessional Care
Acute Coronary Syndrome

• Drug therapy –postoperative:
  – IV nitroglycerin (NTG)
  – Analgesics –morphine
  – β-adrenergic blockers
  – ACE inhibitors
  – Antidysrhythmic drugs
  – Lipid-lowering drugs
  – Stool softeners
  – Maintenance IVF
  – Electrolyte replacement
  – Blood products
Nursing Management
Acute Coronary Syndrome

• Acute Care - Psychosocial
  – Anxiety reduction
    • Identify source and alleviate
    • Patient teaching important
  – Emotional and behavioral reaction
    • Maximize patient’s social support systems
    • Consider open visitation
Nursing Management
Chronic Stable Angina and ACS

• Planning: Overall goals
  – Relief of pain
  – Preservation of heart muscle
  – Immediate and appropriate treatment
  – Effective coping with illness-associated anxiety
  – Participation in a rehabilitation plan
  – Reduction of risk factors

• Evaluation
  – Stable vital signs
  – Relief of pain
  – Decreased anxiety
  – Realistic program of activity
  – Effective management of therapeutic regimen
Nursing Management
Acute Coronary Syndrome

• Ambulatory Care
  – Cardiac rehabilitation
  – Patient and caregiver teaching
  – Physical activity
    • Gradually increased
    • Monitor heart rate
    • Low-level stress test before discharge
    • Isometric vs isotonic activities

“To prevent a heart attack, take one aspirin every day. Take it out for a run, then take it to the gym, then take it for a bike ride...”
Nursing Management
Acute Coronary Syndrome

• Ambulatory Care
  – Resumption of sexual activity – moderate energy activity equivalent to climbing 2 flights of stairs
    • Teach when discuss other physical activity
    • Erectile dysfunction drugs contraindicated with nitrates
    • Prophylactic nitrates before sexual activity
    • When to avoid sex
    • Typically 7–10 days post MI or when patient can climb two flights of stairs