Myocardial Infarction 3

Upon completion of this chapter, you will be able to:

1. Define "myocardial infarction."
2. Identify the ECG changes that may be associated with acute myocardial infarction.
3. Describe the terms "transmural" and "subendocardial" MI.
4. Describe the classic signs and symptoms that are associated with acute myocardial infarction.
5. Describe the initial management of acute myocardial infarction.
6. Explain why pain relief is a high priority in the management of acute myocardial infarction.
7. Explain the benefits of thrombolytic therapy.
8. Describe the management of each of the following:
   - Premature ventricular complexes
   - Symptomatic bradycardia
   - Supraventricular tachycardia (PSVT)
   - Ventricular tachycardia
   - Ventricular fibrillation
   - Wide-complex tachycardia of uncertain origin
   - Pulseless electrical activity
   - Asystole
   - Acute MI with hypotension
   - Acute MI with pulmonary edema
   - Acute MI with hypertension

ACUTE MYOCARDIAL INFARCTION

Definitions

Myocardial infarction

- Necrosis of some mass of heart muscle due to inadequate blood supply

ECG Changes in Acute Myocardial Infarction

- Zone of ischemia → inverted T wave due to altered repolarization
- Zone of injury → elevated ST segment due to severe ischemia
- Zone of infarction → abnormal Q wave due to lack of depolarization of necrotic tissue

Transmural MI

- Entire thickness of the myocardium is destroyed
- Often called Q-wave (producing) infarction as it usually produces, or is associated with changes in, the Q wave on the ECG
- ST segments are often elevated and T waves may be deeply inverted
- Generally accepted criteria for diagnostic Q waves:
  - More than 0.04 sec in duration
  - More than 1/4 of the height of the R wave in the same lead

Subendocardial MI

- Subendocardial layer involved, does not extend through to the epicardial wall
- Often called non-Q-wave (producing) infarction
- Less often associated with thrombosis
- Early stages of subendocardial MI often characterized by ST segment depression
- ST segments are often depressed and T waves may be inverted

Localization of MI
Indicators of Myocardial Injury

1. Anterior infarction
   - Occlusion of the left anterior descending coronary artery
   - ECG changes seen in V_1-V_4

2. Lateral infarction
   - Lateral occlusion of the left circumflex coronary artery
   - ECG changes in I, aVL, V_5, V_6
3. Diaphragmatic or inferior infarction
   - Occlusion of the right coronary artery
   - ECG changes in II, III, aVF
4. True posterior infarction
   - Occlusion of the left circumflex coronary artery
   - ECG changes in V_1-V_2 (reciprocal changes)

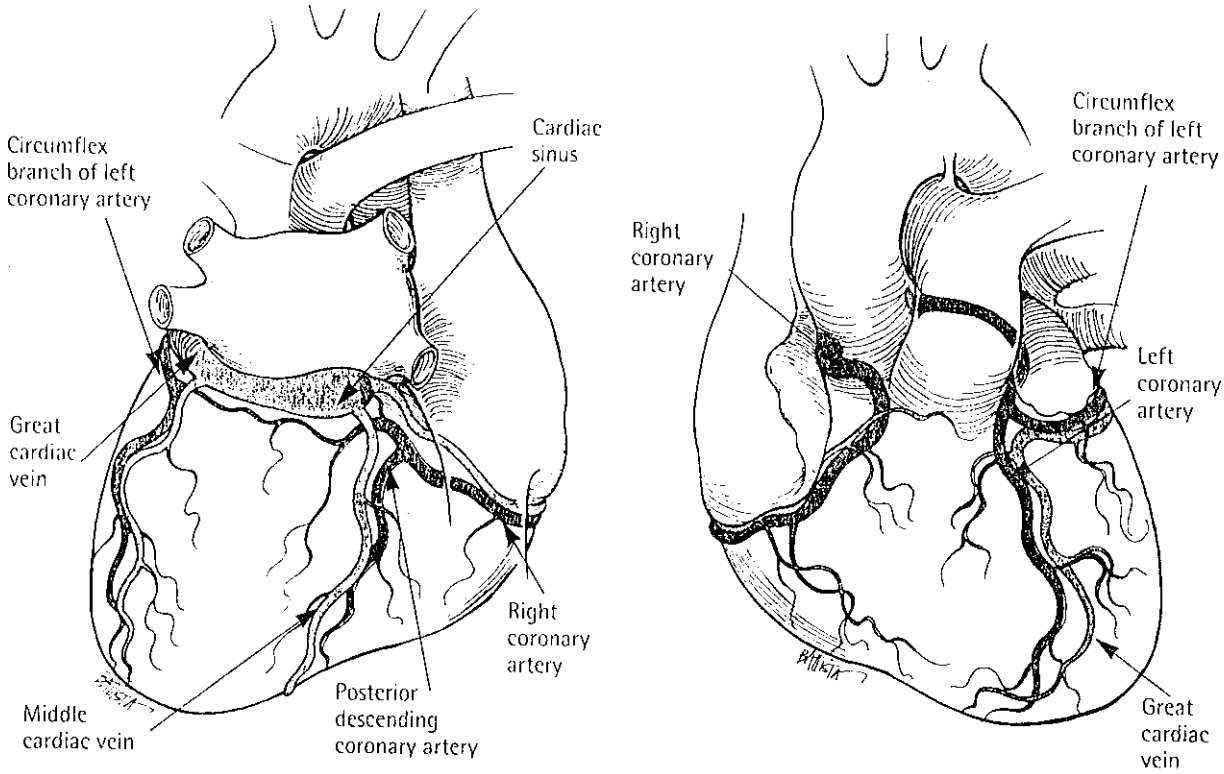

Figure 3-1 The coronary circulation.

Precipitating Events

- Rest, sleep or usual activities
- Morning hours (6:00 a.m. to noon)

Sign/Symptom Recognition

- Pain often described as crushing, squeezing, pressing, or heavy
- Pain may radiate to one (more often the left) or both shoulders and arms, neck, jaw or back
- In a high-risk person, unusual or prolonged "indigestion" should raise suspicion of MI
- Chest pain is frequently associated with MI, however, in a study of the elderly population, roughly 2/3 did not experience chest pain[1]

EARLY MANAGEMENT - GENERAL PRINCIPLES

Diagnosis and Early Treatment

1. Based on the patient's history and presenting signs and symptoms
2. Initial ECG is not conclusive
   - The initial ECG is diagnostic of acute injury in only 24-60% of patients with a final diagnosis of acute myocardial infarction[2]

Oxygen Therapy

1. Hypoxemia leads to anaerobic metabolism and metabolic acidosis, reducing the effectiveness of pharmacologic and electrical therapy
2. Supplemental O_2 decreases the extent of ST-segment changes in patients experiencing an acute MI
3. Lower O_2 flow rates (1-2 liters/min) may be necessary for COPD patients, however, "Oxygen should not be withheld for fear of suppressing respiration if hypoxemia is suspected or if significant respiratory distress is present. The rescuer should be prepared to provide assisted ventilation if necessary."[3]

ECG Monitoring

1. Should be initiated immediately
2. High incidence of VF and other serious dysrhythmias during the early hours of infarction
   - The most common cause of death within the first hours of an MI is cardiac dysrhythmias
3. ST-segment elevation of 0.1 mV or more in two or more adjacent leads is considered indicative of myocardial injury due to acute ischemia[4]

IV Access

1. Large-bore IV line of normal saline or Lactated Ringer's solution
2. Antecubital vein
   - If no IV line is in place at the time of arrest, the antecubital or external jugular veins are sites of first choice

Pain Relief

Relief of pain is a high priority

1. Benefits
   - Decreases anxiety and pain
   - May decrease BP and heart rate
   - Decreases myocardial oxygen demand
   - Decreases risk of dysrhythmias
2. Therapy
   - Sublingual nitroglycerin (if normotensive or hypertensive) 1 tablet (0.3 mg or 0.4 mg), repeated as needed every 5 minutes (maximum of 3 tablets). Nitroglycerin paste or spray may be used.
   - IV nitroglycerin for severe acute ischemic chest pain and blood pressure > 100 mm Hg
     - Increases venous capacitance (decreases preload)
     - Arteriole vasodilation (decreases afterload)
     - Coronary artery vasodilation
   - Morphine (if nitro unsuccessful or pain is severe) in small IV doses (1-3 mg), repeated every 5 min, titrated to pain relief
3. Complications
   - Hypotension
   - Dysrhythmias due to myocardial hypoperfusion or reperfusion

Thrombolytic Therapy

1. High incidence of total coronary occlusion due to coronary thrombosis early in MI
2. Should be considered for all patients with symptoms and ECG findings of acute MI (if no reason for exclusion)
3. Should be administered as soon as possible after onset of symptoms by the first physician competent in making the diagnosis of acute MI
4. Class I (definitely helpful) for:
   a. Patients with acute transmural MI
   b. Patients seen within 6 hours of symptom onset
   c. Patients with new left bundle branch block
5. Early IV β-blockade may decrease reinfarction and incidence of intracranial bleeding in patients receiving thrombolytics
   - IV β-blockers should NOT be administered to patients with hypotension, AV block, congestive heart failure, bradycardia or history of bronchospastic disease
6. After thrombolysis, use of heparin may prevent recurrence of thrombosis

Infarct Limitation

1. Diltiazem (calcium channel blocker)
   - May decrease incidence of early recurrent reinfarction in non-Q-wave MI
   - Not helpful in Q-wave MI and significant left ventricular dysfunction
2. IV Nitroglycerin
   - Increases collateral blood flow to ischemic areas with a secondary potential benefit on infarct size
   - Use in patients with persistent chest pain, without hypovolemia and without right ventricular infarction
3. β-Blockers
   - In hemodynamically STABLE patients with acute MI who did not receive thrombolytic agents, either atenolol (5 mg IV over 5 min) or metoprolol (5-10 mg slow IV push at 5 min intervals to a total of 15 mg) may be administered
   - IV β-blockers should NOT be administered to patients with hypotension, AV block, congestive heart failure, bradycardia or history of bronchospastic disease
4. Aspirin
   - Has been shown to significantly improve mortality and decrease the incidence of reinfarction when used within 24 hours of onset of chest pain[5]
   - Routine use in all MI patients strongly recommended (Class I)
   - Usual dose 150-325 mg

Prophylactic Antidysrhythmic Therapy

1. Routine use of prophylactic antidysrhythmic agents no longer recommended
   - In 1986, the AHA recommended lidocaine be administered as early as possible following the onset of symptoms, even in the absence of PVCs, and continued for at least 24 hours
   - Several studies questioned this recommendation. Lidocaine studies "not only failed to demonstrate improved prognosis, but in some cases, have cited a small but important occurrence rate of asystole and other adverse reactions after prophylactic lidocaine administration."[6]
2. Prophylactic antidysrhythmic use should be avoided in patients older than 70 years due to increased incidence of lidocaine toxicity
3. Patients seen more than six hours after onset of chest pain are less likely to develop VF, therefore routine lidocaine therapy not advisable in these patients

DYSRHYTHMIA COMPLICATIONS - PVCS

The Problem

1. PVCs common with acute MI
2. Prophylactic lidocaine administration no longer recommended

Therapy

- Lidocaine should be avoided as a routine treatment for PVCs
- In the patient with an acute MI/ischemia, treatment should be directed at rapid identification and correction of electrolyte abnormalities
  - Hypokalemia (serum K+ < 3.5 mEq/L)
  - Hypomagnesemia (serum Mg++ level < 1.4 mEq/L)
- When PVCs are seen in acute MI, first treat with oxygen, relieve pain, and consider the use of beta-blockers (if not contraindicated) and/or nitroglycerin
- If unsuccessful, or if significant ventricular ectopy present, lidocaine may be used
  - Initial dose 1-1.5 mg/kg IV bolus
  - May repeat as needed with 0.5-0.75 mg/kg every 5-10 minutes to a maximum total dose of 3 mg/kg

DYSRHYTHMIA COMPLICATIONS - BRADYCARDIAS

The Problem

1. Common during the first hours of symptoms
2. May include:
   - Sinus bradycardia
   - Junctional rhythm
   - AV block (at the level of the AV node)
   - Second or third-degree AV block at the ventricular level
   - New bundle branch block

When to Treat

1. If no symptoms, hypotension or PVCs, no treatment
2. Treatment is indicated if ventricular rate is less than 60 beats/min and:
   - symptoms (chest pain, dyspnea)
   - hypotension or
   - ventricular ectopy present

Therapy

1. Narrow-QRS bradycardia → atropine
   - Atropine is a Class I intervention in sinus bradycardia (definitely helpful)
   - Atropine is a Class IIa (probably helpful) intervention in AV block at the nodal level
2. Wide-QRS AV block bradycardia or new bundle branch block → pacing

DYSRHYTHMIA COMPLICATIONS - AV BLOCKS

First Degree AV Block

1. No treatment unless symptomatic bradycardia
2. Monitor for progression

Second Degree AV Block, Type I (Wenckebach, Mobitz I)

1. Block usually occurs at the level of the AV node
   - Resulting QRS is usually narrow (<.10 sec) since the level of the block is above the branching portion of the bundle of His
2. May occur because of:
   - Increased parasympathetic tone
   - Inferior wall MI
   - Effects of drugs such as digitalis, propranolol, verapamil
3. No treatment unless symptomatic
4. If symptomatic:
   a. Atropine 0.5-1.0 mg every 3-5 min to a maximum of 2-3 mg
   b. Transcutaneous pacing
      - Do not delay transcutaneous pacing if IV access is delayed
   c. Dopamine infusion 5-20 μg/kg/min
   d. Epinephrine infusion 2-10 μg/min
   e. Isoproterenol infusion (low dose)

Second Degree AV Block, Type II (Mobitz II)

1. Usually occurs at the level of the bundle branches
   - Resulting QRS is usually wide (>.10 sec) since the level of the block often involves the bundle of His
2. Usually associated with anteroseptal MI
3. May rapidly progress to third degree AV block without warning
4. Preparations for a transvenous pacer should be made as soon as this rhythm is identified
5. Transcutaneous pacing may be used until transvenous pacing can be accomplished
6. Atropine use *may* increase sinus node activity; however, the impulses bombarding an ischemic AV junction may result in increased AV block, further decreasing ventricular rate and blood pressure.

Third Degree (Complete) AV Block

May occur at the level of the AV node OR the bundle of His

1. If the block is at the level of the AV node → usually junctional escape pacer → narrow QRS (inferior MI)
   - If symptomatic: atropine, transcutaneous pacing, dopamine, epinephrine (possible isoproterenol)
2. If the block is at the level of the bundle of His → ventricular escape pacer → wide QRS (anterior MI)
   - If symptomatic: transcutaneous pacing until transvenous pacing can be accomplished

DYSRHYTHMIA COMPLICATIONS - SUPRAVENTRICULAR TACHYCARDIAS

The Problem

Tachycardia may increase the area of infarction or exacerbate ischemia

Sinus Tachycardia

Identify and correct cause:

- Pain = analgesia
- Hypovolemia = volume replacement
- Extensive myocardial damage = hemodynamic monitoring and drug therapy

General Management Guidelines

1. Immediate countershock is not often needed for heart rates less than 150 beats/minute
2. If the ventricular rate is greater than 150 beats per minute, and the rhythm is not sinus tachycardia:
   a. Prepare for immediate countershock
   b. A brief trial of medications may be administered based on the specific dysrhythmia

Unstable - Signs and Symptoms

- Chest pain
- Shortness of breath
- Decreased level of consciousness
- Low blood pressure
- Shock
- Pulmonary congestion
- Congestive heart failure
- Acute myocaradial infarction