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Chapter 6
Myocardial Ischemia, Injury, & Infarction

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Cardiovascular Diseases (CVD)

- Include:
  - Coronary heart disease
  - Stroke (brain attack)
  - Hypertension
  - Rheumatic heart disease

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1997 Statistics

- 59,700,000 Americans have one or more forms of cardiovascular disease
- Cardiovascular disease claimed 953,110 lives in the United States (41.2% of all deaths)
- About one sixth of the individuals that die of CVD are under the age of 65
- Coronary heart disease is the single leading cause of death in America today

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Acute Coronary Syndromes

- Include:
  - Unstable angina
  - Non-ST-segment elevation MI
  - ST-segment elevation MI
- Sudden cardiac death may occur with each of these syndromes

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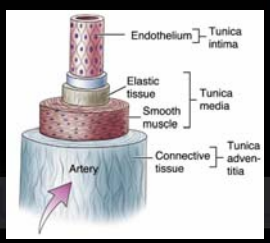
Structure of Arteries

- Arteries consist of three layers
  - Outer layer = Tunica adventitia
    - Consists of flexible fibrous connective tissue
    - Helps hold the vessel open



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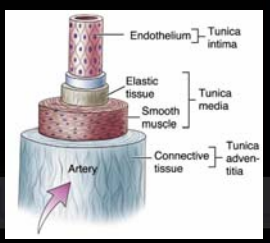
Arterial Structure

- Middle layer = Tunica media
  - Encircled by smooth muscle
  - Innervated by fibers of the autonomic nervous system
  - Allows constriction and dilation



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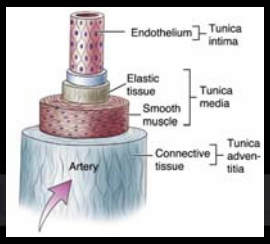
Arterial Structure

- Innermost layer = Tunica intima
  - Made up of endothelium that lines the vascular system
  - Endothelium is a single layer of cells in direct contact with the blood



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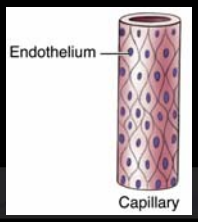
Functions of Normal Endothelium

- Present a barrier between the blood and the arterial wall
- Antithrombotic properties prevent platelets from sticking and aggregating and forming clots
- Produce vasodilating factors



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Functions of Normal Endothelium

- Produce vasoconstricting factors
- Release platelet factors that promote thrombus formation
- Prevent migration and proliferation of smooth muscle cells
- Prevent inflammation

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Pathogenesis of Acute Coronary Syndromes

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Arteriosclerosis

- A chronic disease of the arterial system characterized by abnormal thickening and hardening of the vessel walls

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Atherosclerosis

- A form of arteriosclerosis
- Accumulation of fibrofatty deposits in the intimal lining of large and middle-sized muscular arteries
- Buildup results in decreased blood flow
  - Ischemia

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Atherosclerosis

- Any artery in the body can develop atherosclerosis
- Coronary artery involvement
  - Angina (chest pain)
- Involvement of arteries of legs
  - Peripheral vascular disease
- Involvement of carotid arteries
  - TIA

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Coronary Artery Pathology

- Usual cause of an acute coronary syndrome is the rupture of an atherosclerotic plaque
- Atherosclerotic lesions
  - Fatty streak
  - Fibrous plaque
  - Advanced (complicated) lesion

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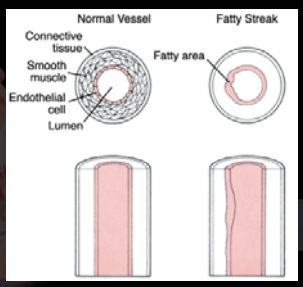
Fatty Streaks



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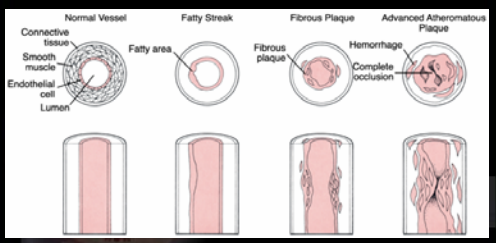
Fibrous Plaques/Advanced Lesions



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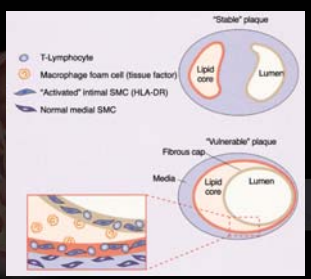
Comparison Of "Stable" and "Vulnerable" Plaques



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Vulnerable Plaques

- "Vulnerable" plaques are prone to rupture
  - Soft
  - Primarily consist of lipid-rich atheromatous material
    - Lipid pool or "gruel"
  - Have a thin cap of fibrous tissue that separates it from the vessel lumen
- Determinants of a plaque's vulnerability to rupture are thought to include:
  - The size of the atheromatous core
  - The thickness of the fibrous cap

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Vulnerable Plaques

- Contributing factors to plaque rupture may include:
  - Shear stress (the frictional force from blood flow)
  - Coronary spasm at the site of the plaque
  - Internal plaque changes
  - Effects of risk factors (e.g., hypertension, hyperlipidemia)

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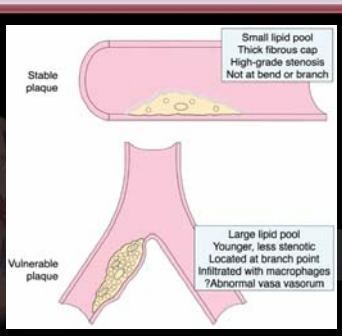
Vulnerable Plaques



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Plaque Rupture

- If the fibrous cap ruptures:
  - Plaque contents are exposed to flowing blood
    - Promotes platelet adhesion and aggregation
  - Coagulation cascade then begins
    - Results in additional platelet aggregation and thrombosis

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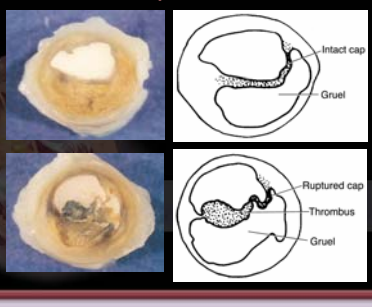
View of a Vulnerable Plaque



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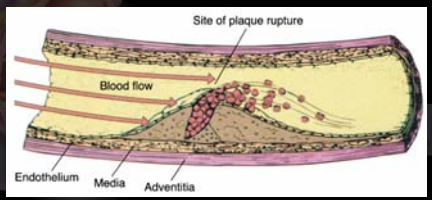
Plaque Rupture

- When an atherosclerotic plaque ruptures or erodes:
  - Platelets adhere to the damaged lining of the vessel and to each other within 1 to 5 seconds, forming a plug



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Plaque Rupture

- "Sticky platelets" secrete several chemicals including thromboxane A2
  - Stimulates vasoconstriction
- Aspirin
  - Blocks synthesis of thromboxane A2
  - Inhibits platelet aggregation

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Plaque Rupture

- Once platelets are activated:
  - Glycoprotein IIb/IIIa receptors appear on the surface of the platelet
    - Essential for platelet aggregation
  - Fibrinogen molecules bind to these receptors
    - Form bridges between platelets, allowing them to aggregate

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Plaque Rupture

- Glycoprotein IIb/IIIa receptor inhibitors
  - Prevent fibrinogen binding and platelet aggregation

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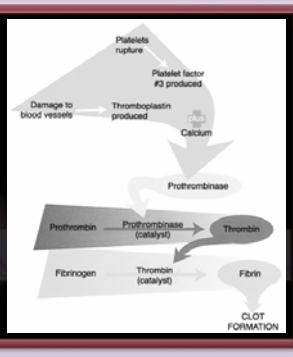
Plaque Rupture

- As the process continues:
  - Fibrinogen cross-links platelets
  - Thrombin is then generated
  - Fibrin is formed, ultimately producing a thrombus



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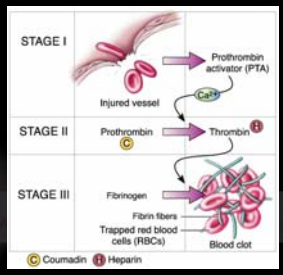
Plaque Rupture

- Heparin
  - Can inhibit further clotting
    - Inactivates thrombin
    - Prevents conversion of fibrinogen to fibrin



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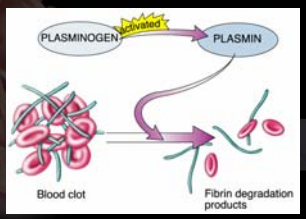
Fibrinolysis

- Fibrinolytics ("clot-busters")
  - Stimulate conversion of plasminogen to plasmin, which dissolves the clot



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Acute Coronary Syndromes

- Complete occlusion of the coronary artery
  - Q-wave MI (ST-elevation MI)
  - Sudden death
- Incomplete occlusion
  - No clinical signs and symptoms (silent)
  - Unstable angina
  - Non-Q-wave MI (non-ST-segment elevation MI)
  - Sudden death

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Coronary Occlusion

The patient's clinical presentation and outcome depend on the:

- Amount of myocardium supplied by the affected artery
- Severity and duration of myocardial ischemia
- Electrical instability of the ischemic myocardium
- Degree and duration of coronary obstruction
- Presence (and extent) or absence of collateral coronary circulation

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Myocardial Ischemia

- Ischemia results when the heart's demand for oxygen exceeds its supply from the coronary circulation

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Myocardial Ischemia – ECG Changes

- Myocardial ischemia delays repolarization
- ECG changes include temporary changes in the ST-segment and T wave

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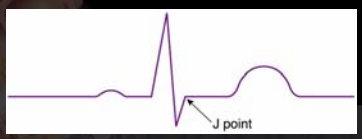
Myocardial Ischemia – ECG Changes

- On the ECG, the J-point is the point where the QRS ends and the ST segment begins



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Acute Coronary Syndrome

- "Although typical characteristics substantially raise the probability of CAD, features not characteristic of chest pain, such as sharp stabbing pain or reproduction of pain on palpation, do not exclude the possibility of acute coronary syndrome."

Braunwald E, Antman EM, Beasley JW, Califf RM, Cheitlin MD, Hochman JS, Jones RH, Kereiakes D, Kupersmith J, Levin TN, Pepine CJ, Schaeffer JW, Smith EE III, Steward DE, Theroux P. ACC/AHA guidelines for the management of patients with unstable angina and non–ST-segment elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients With Unstable Angina). *J Am Coll Cardiol* 2000;36:970 –1062.

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Myocardial Injury

- Ischemia prolonged more than just a few minutes results in myocardial injury
- Injured myocardial cells can live or die depending on how quickly blood flow is restored to the affected tissue

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Myocardial Injury

- Methods to restore blood flow may include:
  - Administration of fibrinolytic agents
  - Coronary angioplasty
  - Coronary artery bypass graft (CABG)
  - Other interventions

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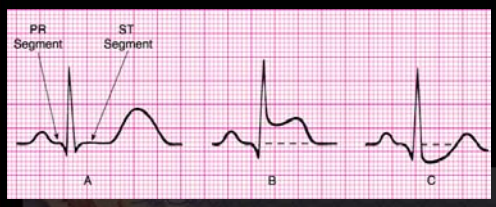
Myocardial Injury – ECG Changes



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Myocardial Infarction

- MI is death of myocardial cells due to prolonged ischemia
- Possible causes
  - Coronary thrombus
  - Coronary spasm
  - Coronary embolism (rare)

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Diagnosis of MI

Diagnosis of MI is based on the presence of at least two of the following three criteria:

- Clinical history of ischemic type chest discomfort
- Changes on serially obtained ECGs
- Rise and fall in serum cardiac markers

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Classification of MI

- Size
  - Microscopic
  - Small
    - < 10% of left ventricle
  - Medium
    - 10% to 30%
  - Large
    - > 30%
- Location
  - Anterior
  - Lateral
  - Inferior
  - Posterior
  - Septal
  - Combination of locations

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Classification of MI

- ST-segment elevation or non-ST-segment elevation infarction
- Terminology
  - Acute MI = 6 hours to 7 days
  - Healing MI = 7 to 28 days
  - Healed MI = 29 days or more

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Acute Coronary Syndromes

Clinical Manifestations

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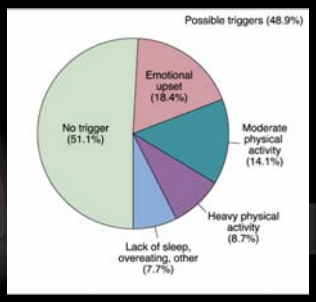
History - Precipitating Factors

- A precipitating factor is present in approximately 50% of patients experiencing MI



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Precipitating Factors

- Approximately 2/3 of patients describe the new onset of angina or a change in their anginal pattern in the month preceding infarction
- Cocaine use may be a factor
- Inquire about the use of cocaine in patients with suspected acute coronary syndromes, particularly in patients < 40 years of age

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Circadian Rhythmicity

- Frequency of ischemic episodes increases shortly after awakening
  - Secondary peak may occur in early evening hours

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MI – Clinical Presentation

- Chest discomfort present in 75% to 80% of patients with acute MI
  - Single most common symptom of infarction
- May begin in the central or left chest and then radiate to the arm, jaw, back, or shoulder

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MI – Clinical Presentation

- May be accompanied by:
  - Unexplained nausea and vomiting
  - Persistent shortness of breath due to LVF
  - Unexplained:
    - Weakness
    - Dizziness
    - Sweating
    - Anxiety
    - Lightheadedness or syncope
  - Combination of these symptoms
- Symptoms may occur with or without associated chest discomfort

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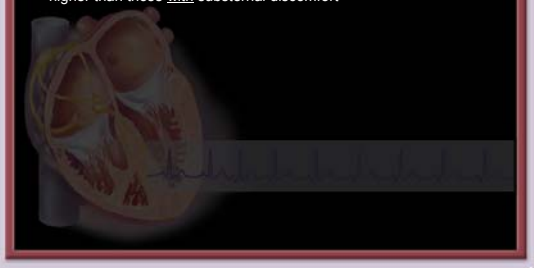
MI – Clinical Presentation

- Overall mortality rate of patients without chest pain is substantially higher than those with substernal discomfort



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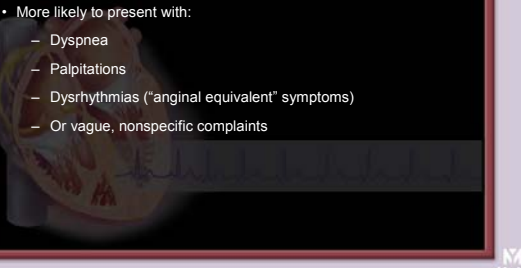
Atypical Presentation

- Chest discomfort absent in ~ 20% of patients
- More likely to present with:
  - Dyspnea
  - Palpitations
  - Dysrhythmias (“anginal equivalent” symptoms)
  - Or vague, nonspecific complaints



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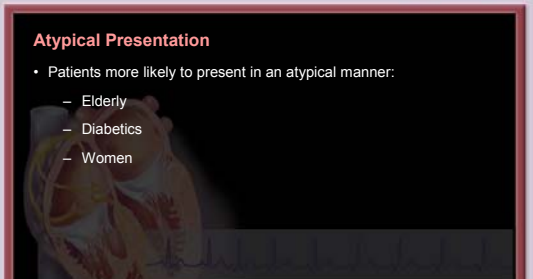
Atypical Presentation

- Patients more likely to present in an atypical manner:
  - Elderly
  - Diabetics
  - Women



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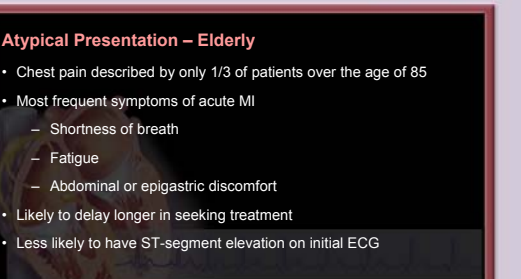
Atypical Presentation – Elderly

- Chest pain described by only 1/3 of patients over the age of 85
- Most frequent symptoms of acute MI
  - Shortness of breath
  - Fatigue
  - Abdominal or epigastric discomfort
- Likely to delay longer in seeking treatment
- Less likely to have ST-segment elevation on initial ECG



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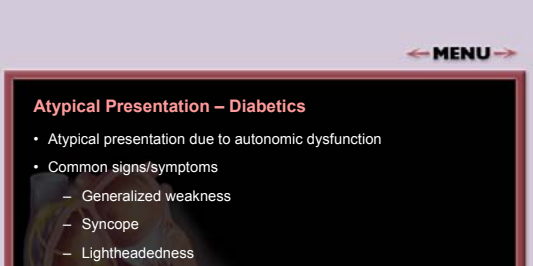
Atypical Presentation – Diabetics

- Atypical presentation due to autonomic dysfunction
- Common signs/symptoms
  - Generalized weakness
  - Syncope
  - Lightheadedness
  - Change in mental status



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Atypical Presentation - Women

- May present with typical symptoms
- Some may have only vague chest discomfort that tends to come and go with no known aggravating factors

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Atypical Presentation - Women

- Others do not have chest pain but have other signs and symptoms that may include:
  - Diaphoresis
  - Lightheadedness
  - Shortness of breath
  - Back pain
  - Nausea/vomiting

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MI – Symptom Recognition

- Most patients do not seek medical care for 2 hours or more
  - Many wait 12 hours or more
- Women often delay longer than men do in seeking hospital medical treatment

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MI – Reperfusion Therapy

- Major advantage of reperfusion therapy is time dependent
- Greatest benefit for eligible patients when initiated at or close to the onset of acute MI symptoms
- Generally, reperfusion therapy beyond 12 hours may offer little benefit

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MI - Mortality

- Mortality from acute MI decreases as the interval between symptom onset and initiation of treatment decreases
- Each hour of delay = 1% mortality

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MI – Delay in Treatment

Factors affecting prehospital delay in patients with signs and symptoms of acute myocardial infarction

Factors Contributing to Increased Delay	Factors Contributing to Decreased Delay
Older age	Hemodynamic instability
Female gender	Large infarct size
African-American race	Sudden onset of severe chest pain
Low socioeconomic status	Recognition by patient that symptoms are heart-related
Low emotional or somatic awareness	Consulting a friend, co-worker, or stranger
History of angina, diabetes, or both	
Consulting a spouse or other relative	
Consulting a physician	
Self-treatment	

Source: Educational Strategies To Prevent Prehospital Delay In Patients At High Risk For Acute Myocardial Infarction. NIH Publication No. 97-3787 September 1997 National Institutes Of Health National Heart, Lung, And Blood Institute.

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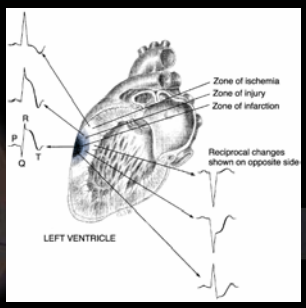
Zones of Ischemia, Injury, Infarction



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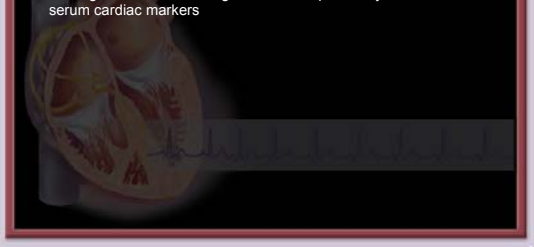
Non-ST-Segment Elevation MI

- ST-segment depression in leads facing the affected area
- MI diagnosed if ECG changes are accompanied by elevations of serum cardiac markers



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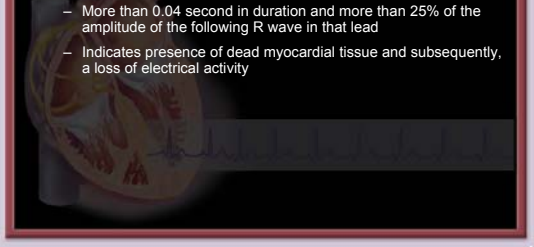
ST-Segment Elevation MI

- Most patients with ST-segment elevation MI will develop Q-wave MI
- Abnormal (pathologic) Q wave
  - More than 0.04 second in duration and more than 25% of the amplitude of the following R wave in that lead
  - Indicates presence of dead myocardial tissue and subsequently, a loss of electrical activity



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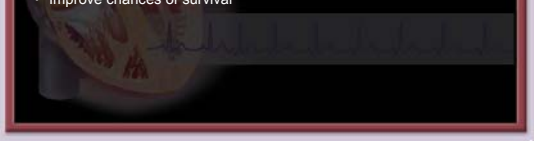
ACS Management Goals

- Minimize infarct size
- Salvage ischemic myocardium
- Alleviate vasoconstriction
- Reduce myocardial oxygen demand
- Prevent and manage complications
- Improve chances of survival



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Initial Recognition and Management of ACS

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Acute MI Protocol

- Emergency departments should have an acute MI protocol that yields:
  - Targeted clinical examination and a 12-lead ECG within 10 minutes
  - Door-to-drug time < 30 minutes

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Immediate Management

- Immediate management of all acute coronary syndromes is generally the same

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Initial Assessment

- Obtain a brief, targeted history/physical examination
- Age
- Gender
- Signs and symptoms
  - Location of pain
  - Duration
  - Quality
  - Relation to effort
  - Time of symptom onset
- History of CAD
- CAD risk factors present?
- History of Viagra use?

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Possible Acute Coronary Syndrome

- Recent episode of chest discomfort at rest not entirely typical of ischemia but pain free at time of initial evaluation
- Normal or unchanged ECG
- No serum cardiac marker elevation
- Managed by either observation in emergency department chest pain unit or admitted to monitored hospital bed

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Definite Acute Coronary Syndrome

- Recent episode of typical ischemic discomfort that is either of new onset or severe or exhibits an accelerating pattern of previous stable angina (especially if it occurred at rest or within 2 weeks of previously documented MI)
- Triage based on 12-lead ECG

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ACS – Initial Interventions

- If findings are consistent with possible or definite acute coronary syndrome, perform the following interventions (including obtaining and reviewing 12-lead ECG), within 10 minutes of patient presentation

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Initial Interventions

- Targeted history/physical exam
  - Use checklist (yes-no)
  - Focus on eligibility for reperfusion therapy
- Assess vital signs
- Determine oxygen saturation
- Establish IV access, ECG monitoring
- Administer aspirin 162 to 325 mg (chewed) if no reason for exclusion
- Obtain baseline serum cardiac marker levels

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Initial Interventions

- Obtain 12-lead ECG (physician to review) – categorize patient into one of three groups
  - ST-segment elevation or new or presumably new LBBB
  - ST-segment depression/transient ST-segment/T wave changes
  - Normal or nondiagnostic ECG
- Serial ECGs in patients with Hx suggesting MI and nondiagnostic ECG
- Obtain lab specimens (CBC, lipid profile, electrolytes)
- Portable chest x-ray

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- M = Morphine
- O = Oxygen
- N = Nitroglycerin
- A = Aspirin

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Routine Measures

- Oxygen
- Aspirin
  - 162 to 325 mg – chewed

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Routine Measures – Pain Relief

- Pain relief is a priority
- Benefits
  - Decreases anxiety and pain
  - May decrease BP and heart rate
  - Decreases myocardial oxygen demand
  - Decreases risk of dysrhythmias
- Possible complications - hypotension

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Routine Measures – Nitroglycerin SL or Spray

- Ensure IV access, SBP > 90 mm Hg
- Avoid nitroglycerin administration
  - Presence of marked bradycardia (<50 beats/min)
  - Suspected right ventricular infarction
  - Viagra use within 24 hours
- Avoid long-acting oral nitrate preparations in the early management of acute MI

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Routine Measures – Morphine

- Morphine 2 to 4 mg IV (if discomfort is not relieved with NTG)
  - May repeat every 5 min
  - Ensure SBP > 90 mm Hg
- If respiratory depression occurs, treat with IV naloxone

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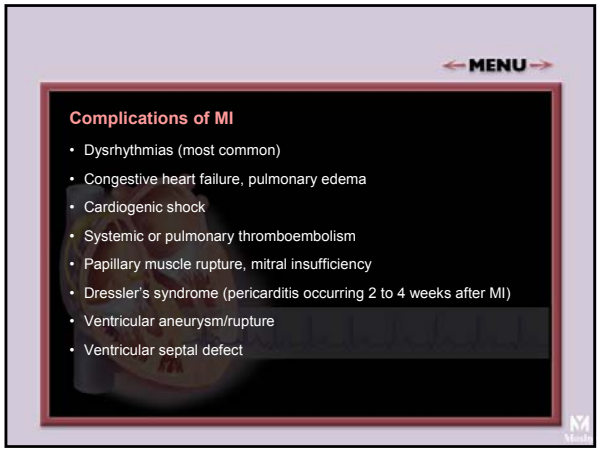
Complications of MI

- Primary electrical complication of acute MI
  - Cardiac dysrhythmias
- Primary mechanical complications of acute MI
  - CHF and shock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Complications of MI
• Dysrhythmias (most common)
• Congestive heart failure, pulmonary edema
• Cardiogenic shock
• Systemic or pulmonary thromboembolism
• Papillary muscle rupture, mitral insufficiency
• Dressler's syndrome (pericarditis occurring 2 to 4 weeks after MI)
• Ventricular aneurysm/rupture
• Ventricular septal defect