


EMC 451

Advanced ECG Interpretation

Unit 11: Management of Acute Coronary Syndromes

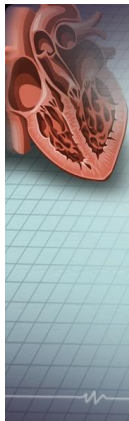
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Unit Objectives

- Upon completion of this unit, you should be able to:
 - Describe the AHA algorithm for ACS
 - Discuss the benefits of prehospital 12-lead EKG
 - Discuss the use of glycoprotein IIb/IIIa receptor inhibitors, heparin, LMH, ACE inhibitors, beta blockers, aspirin, and nitroglycerin in the management of ACS.
 - Discuss the use of PCI and fibrinolytics in the management of ACS.
 - Discuss the current recommendations regarding prehospital thrombolysis.
 - Describe the use of angiography, angioplasty, stents, and CABG in the management of ACS.

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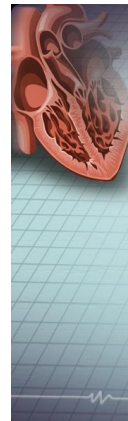


Deaths from ACS

- CVD was 251.2 per 100,000
- CVD accounted for 33.6% of all 2,243,712 deaths in 2007
- More than 150,000 Americans killed by CVD were less than 65 years of age.
- This year, 785,000 Americans will have a new MI. 470,000 will have a recurrent MI
- Most deaths occur in prehospital setting
- Most deaths result from VF or VT
- Major risk of VF/VT is within the first 4 hours of symptoms
- Reperfusion reduces risk of VF/VT

Heart Disease and Stroke Statistics—2011 Update : A Report From the American Heart Association
<http://circ.ahajournals.org/content/123/4/e18.full.pdf>

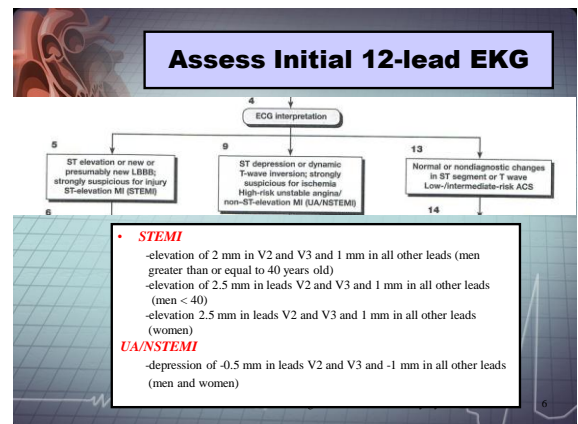
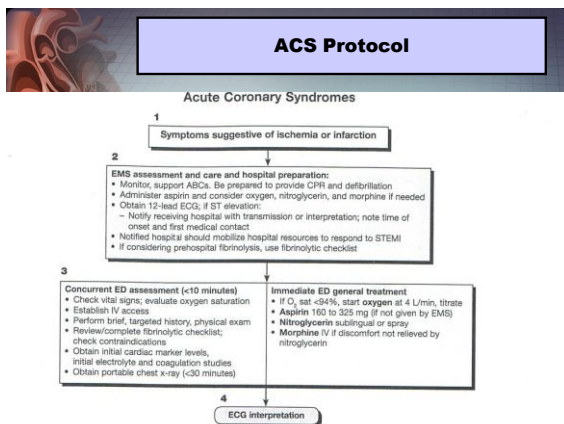
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Goals of Therapy

- Reduce the amount of myocardial necrosis that occurs in patients with AMI, preserving left ventricular function, preventing HF, and limiting other cardiovascular complications.
- Prevent major adverse cardiac events: death, non-fatal MI, need for revascularization.
- Treat acute, life threatening complications of ACS.

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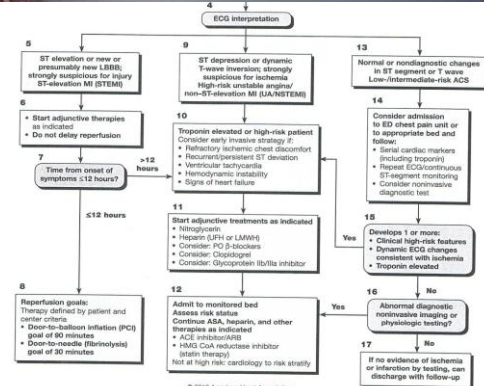
Prehospital 12-lead EKG

- Increases on-scene time by 4 minutes
- No difference in quality of information between field EKG and EKG obtained at hospital
- Reduces door-to-drug time between 20 and 55 minutes
- Improved outcome (8% mortality with a field 12-lead vs. 12% without)

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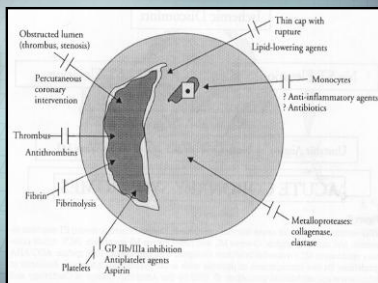
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ACS Protocol Continued



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Interventions in the Thrombogenesis Process



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Heparin

- Prevents new clots from forming by inhibiting thrombin
- Difficult to dose because patients differ in the concentrations of several proteins to which heparin binds.
- Serial coagulation levels required.
- Reduction in mortality not as high as once thought (now shown to produce only 6% reduction)
- Probably no benefit when patients are treated with ASA, beta blockers, nitrates, and ACE inhibitors
- Recommended for patients receiving alteplase, reteplase, tenecteplase

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Low Molecular Weight Heparin (LMWH)

- Longer plasma half-life and a more predictable anticoagulation response
- Shorter duration of bleeding time after platelet inhibition
- Lower incidence of thrombocytopenia
- Lower incidence of bleeding events
- Easier to administer
- No anticoagulant monitoring necessary
- Drugs
 - Lovenox
 - Fragmin
 - Fraxiparin



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Rx ONLY

Glycoprotein IIb/IIIa Receptor Inhibitors

- GP IIb/IIIa receptor is the final common pathway to platelet aggregation
- GP IIb/IIIa inhibition is one method of reducing ischemic complications after plaque fissure or rupture.
- Prevents new clots from forming
- Drugs
 - Integrelin (Eptifibatide)
 - ReoPro (Abciximab)
 - Aggrastat (Tirofiban)


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Aspirin

- 23% reduction in mortality from MI.
- Antiplatelet effect
- 160 – 325 mg




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Beta Adrenergic Blockers


- Reduce myocardial oxygen demand by reducing heart rate, system pressure, and myocardial contractility.
- Provides protective effect against progression to Q-wave MI.
- Reduces incidence of arrhythmias.
- Contraindicated in patients with symptomatic bradycardia, LV failure, heart block, COPD, IDDM

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Nitroglycerin

- Previously thought to reduce size of infarct, but recent studies are inconclusive.
- Use for treatment of ischemic chest pain in the early management of ACS.
- Not a substitute for narcotic analgesia.
- Also used in the treatment of HTN, CHF, and large anterior wall MI.
- Contraindicated in RV MI. Obtain V4R in all patients with inferior wall MI prior to administering NTG.




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ACE Inhibitors

- 6% reduction in mortality
- Reduces infarct size, increases collateral blood flow
- Indications
 - AMI
 - LV failure (< 40% EF)
 - Heart failure
- Drugs
 - Captopril
 - Enalapril
 - Quinapril




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Clopidogrel

- Irreversible blockade of ADP receptor
- Blocks activation of the GP IIb/IIIa pathway




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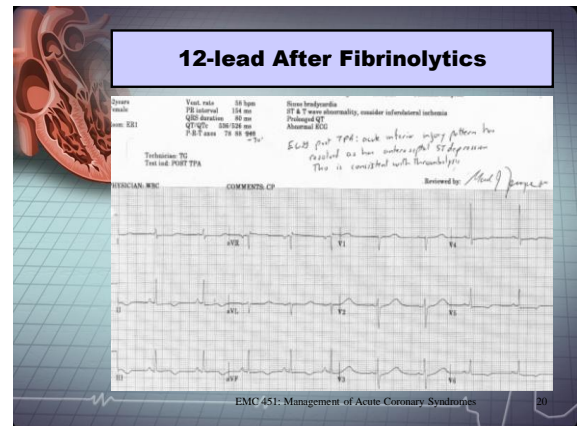
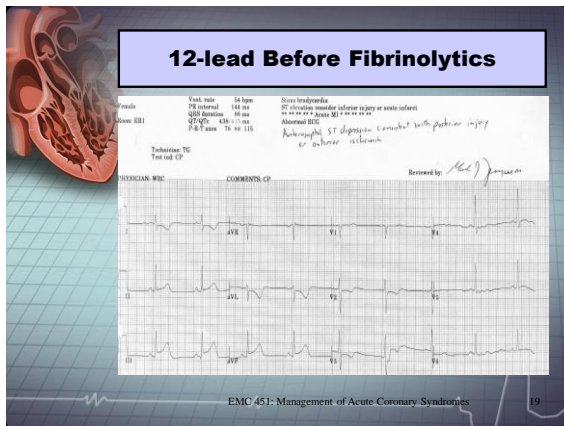


Fibrinolytics

- **Alteplase (TPA, Activase)**
 - Catalyzes the conversion of tissue plasminogen to plasmin in the presence of fibrin. Fibrin specificity produces local fibrinolysis in the area of recent clot formation.
 - 3 hour infusion
- **Streptokinase (Streptase)**
 - Bonds with plasminogen to expose plasminogen activating site
 - Converts plasminogen to plasmin
 - 1 hour infusion
- **APSAC (Anistreplase, Eminase)**
 - Activates endogenous fibrinolytic system to produce plasmin
 - 5 minute infusion
- **Retavase (Retavase)**
 - Activates conversion of plasminogen to plasmin
 - 2 minute bolus followed in 30 minutes by a second 2 minute bolus
- **Tenecteplase**
 - Single bolus over 5 seconds
 - Most fibrin specific
 - One of the lowest mortality rates



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Prehospital Thrombolysis

- Grampian Region Early Anistreplase Trial (GREAT)**
 - Thrombolysis 130 minutes sooner
 - 50% reduction in mortality
- European Myocardial Infarction Project (EMIP)**
 - Thrombolysis median of 55 minutes sooner
 - 16% reduction in mortality
- Myocardial Infarction Triage and Intervention Trial (MITI)**
 - No significant difference in mortality
 - Advanced notification reduced hospital door-to-drug times which confounded results

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