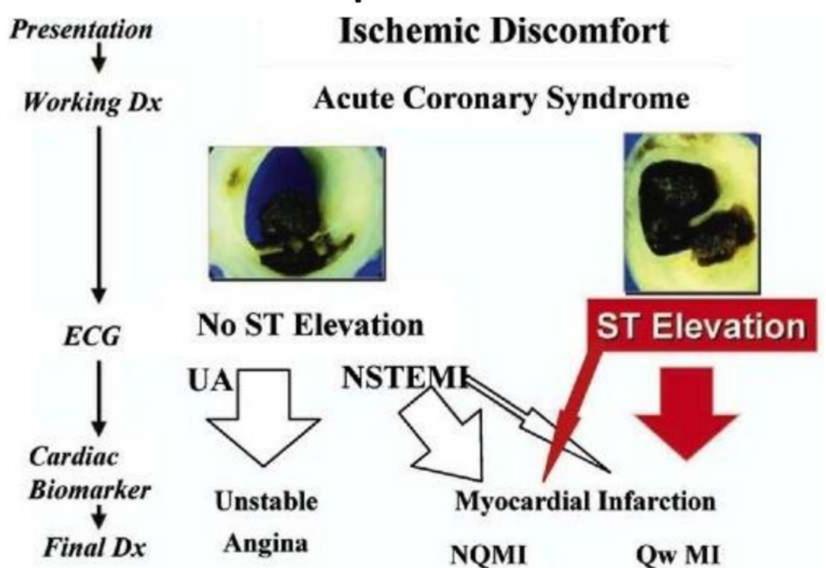


STEMI

- ACS and STEMI
- History and physical, differential diagnosis
- ECGs
- Initial Therapy and management
- Reperfusion therapy
- Complications of acute STEMI

ACS Spectrum







Hospitalizations in the U.S. Due to Acute Coronary Syndromes (ACS)

Acute Coronary
Syndromes*

1.57 Million Hospital Admissions - ACS

UA/NSTEMI†

1.24 million

Admissions per year

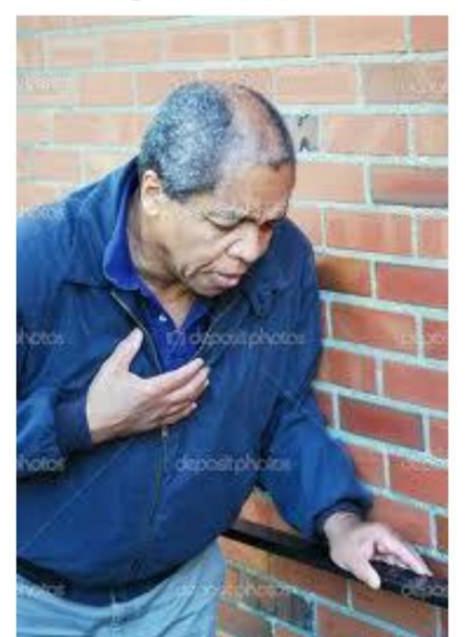
STEMI

.33 million

Admissions per year

Heart Disease and Stroke Statistics – 2007 Update. Circulation 2007; 115:69-171. *Primary and secondary diagnoses. †About 0.57 million NSTEMI and 0.67 million UA.

STEMI



Case

- A 56 year old male with no PMH presents with sudden onset of severe crushing retrosternal chest pain that woke him from sleep. It radiated down his left arm.
 - It was accompanied with sweating, and shortness of breath

Physical Examination

- HR 70/min, BP 130/80, RR 22/min
- JVP not raised
- Chest clear
- Normal S1 and S2, ?S3
- Soft non tender abdomen
- No LE edema

Differential Diagnosis of Acute Chest Pain

Cardiac

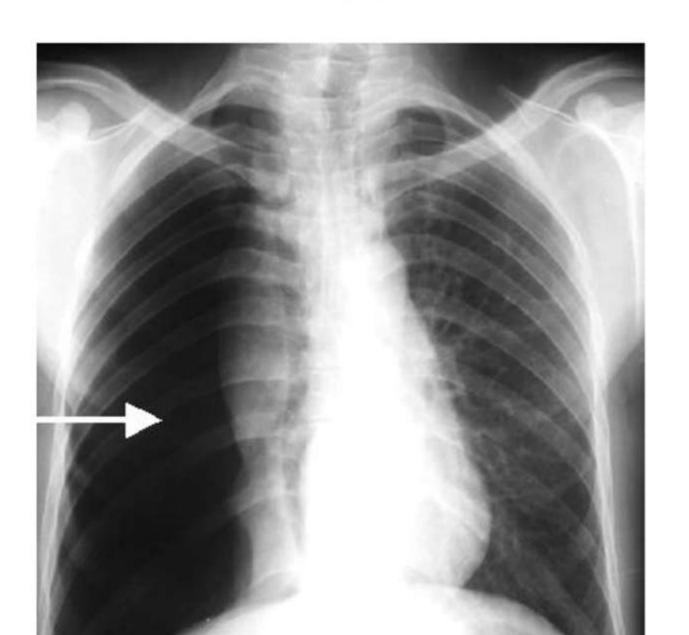
- ACS
- Aortic Dissection*
- Pericarditis

Pulmonary

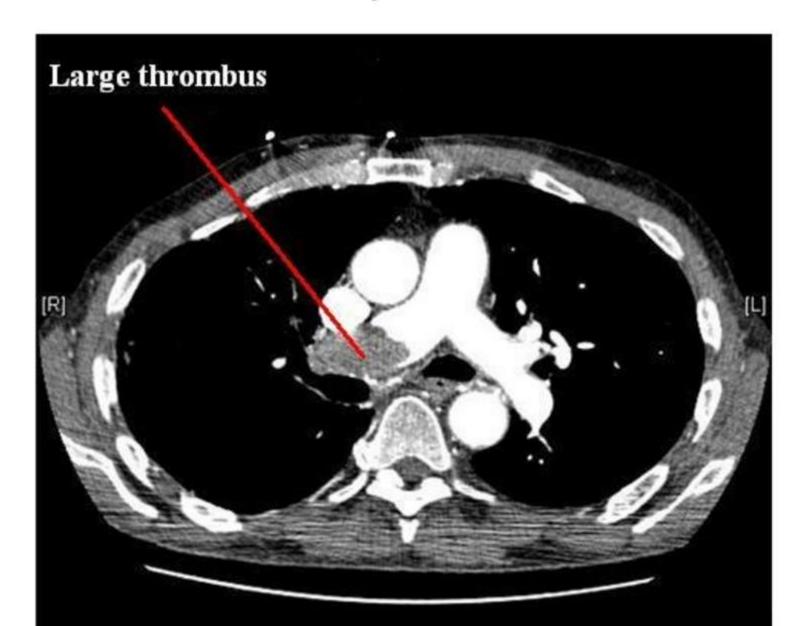
- Pulmonary Embolism*
- Pneumonia
- Pneumothorax*

- Chest wall
 - Rib fracture
 - Costochondritis
 - Herpes zoster (before rash)
- Gastrointestinal
 - Biliary
 - Esophageal
 - Spasm
 - Rupture
 - Pancreatitis
 - Peptic Ulcer*

Pneumothorax



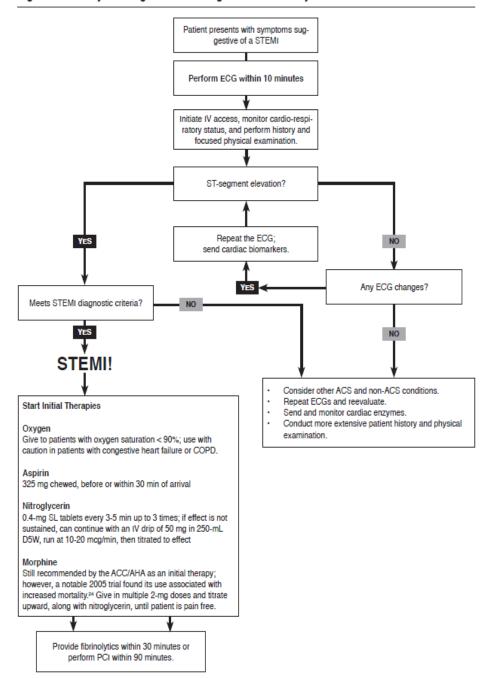
Pulmonary Embolism



Aortic Dissection



Figure 3. Pathway For Diagnosis Of ST-Segment Elevation Myocardial Infarction



ECG Criteria for STEMI

New ST elevation

- ->0.1 mV in 2 contiguous leads
- Any 2 (II, III, aVF) or (V_2 - V_6 , I, aVL)
- Not aVR or V1

In $V_2 \& V_3$

- >= 0.2 mV in men
- >= 0.15mV in women

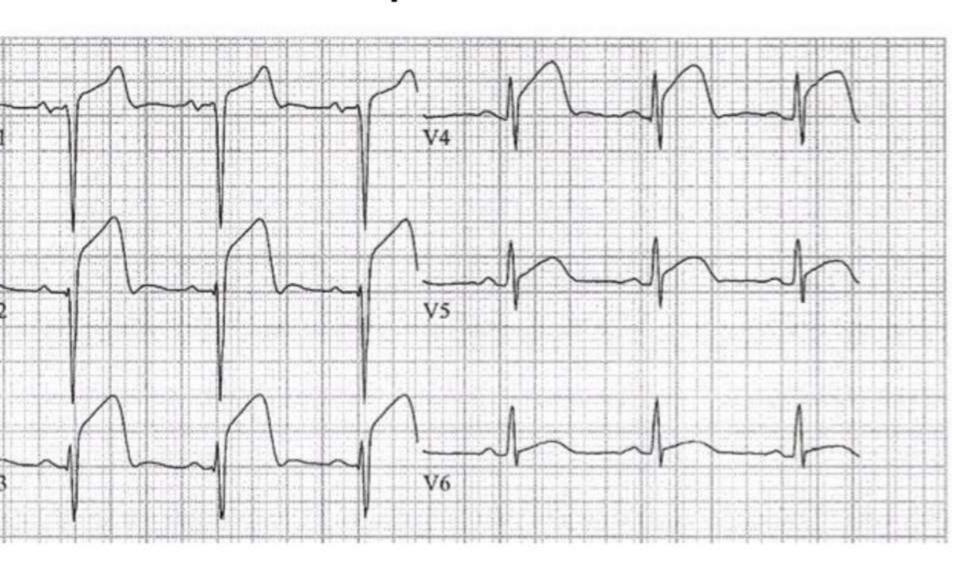
New LBBB

Thygsen et al. Universal Definition of MI

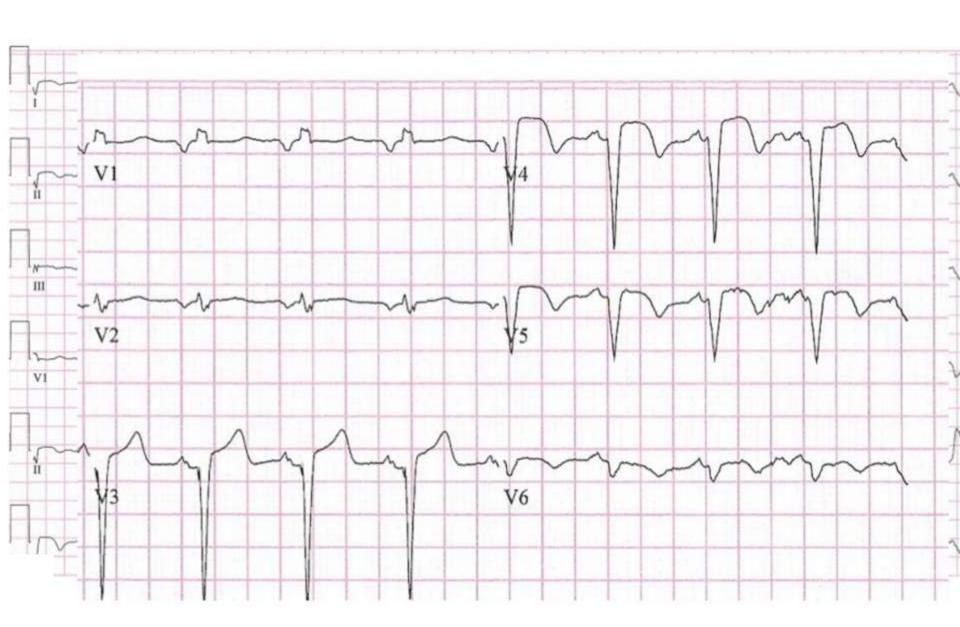
Proposed Criteria to determine who gets ECG in ER STAT

- >30 with chest pain
- >50 with dyspnea, altered mental status, upper extremity pain, syncope or weakness
- >80 with abdominal pain, nausea and vomiting
- DOESN'T REPLACE CLINICAL JUDGEMENT

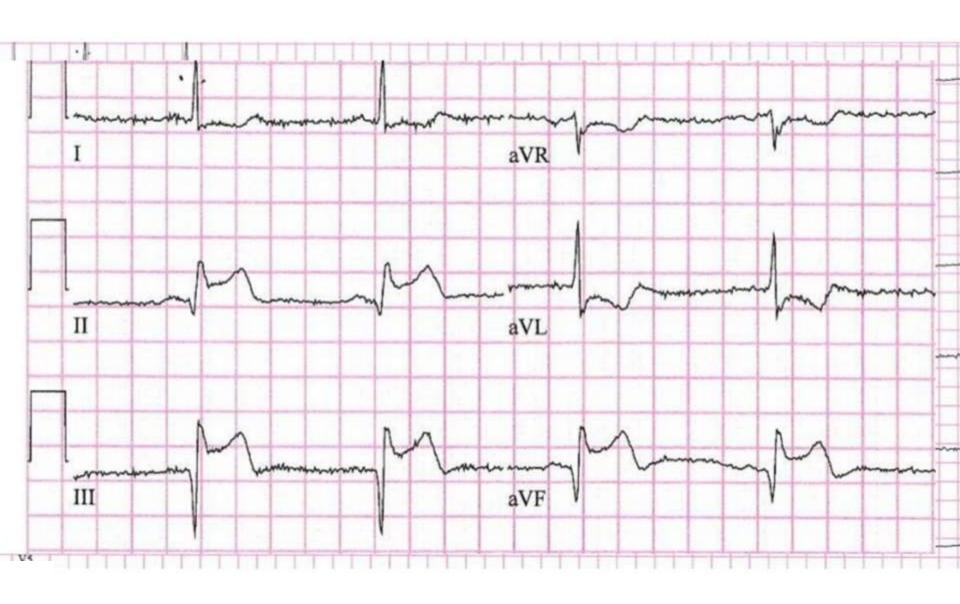
Anteroseptal wall STEMI



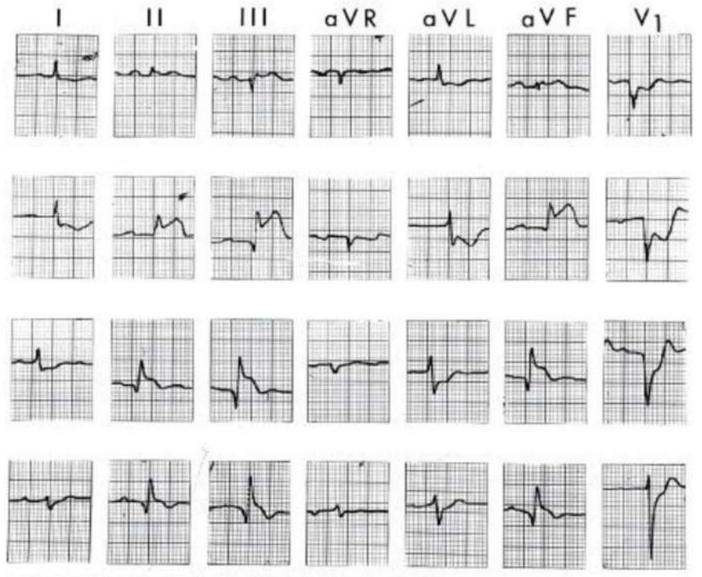
Anterolateral STEMI



Inferior Wall STEMI



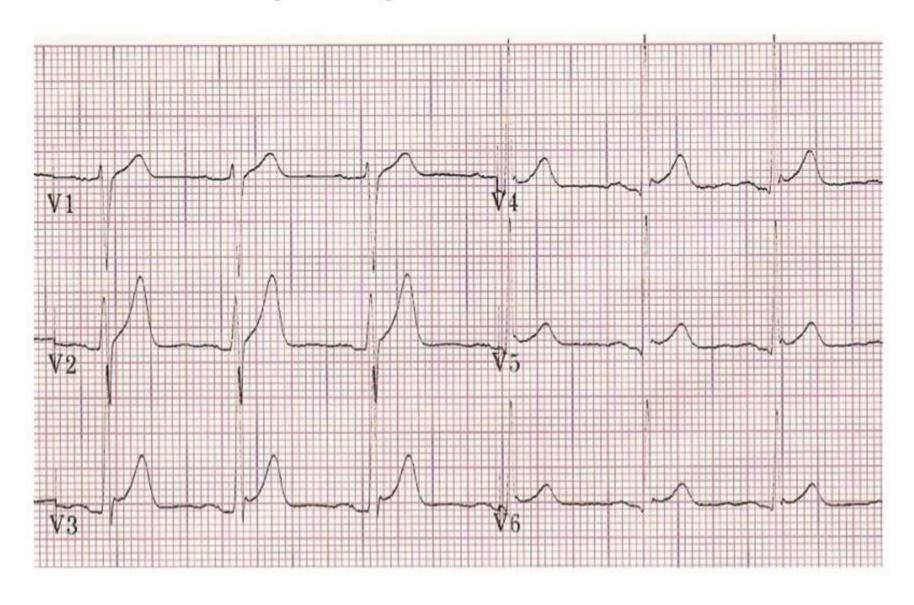
Evolution of ECG changes in STEMI



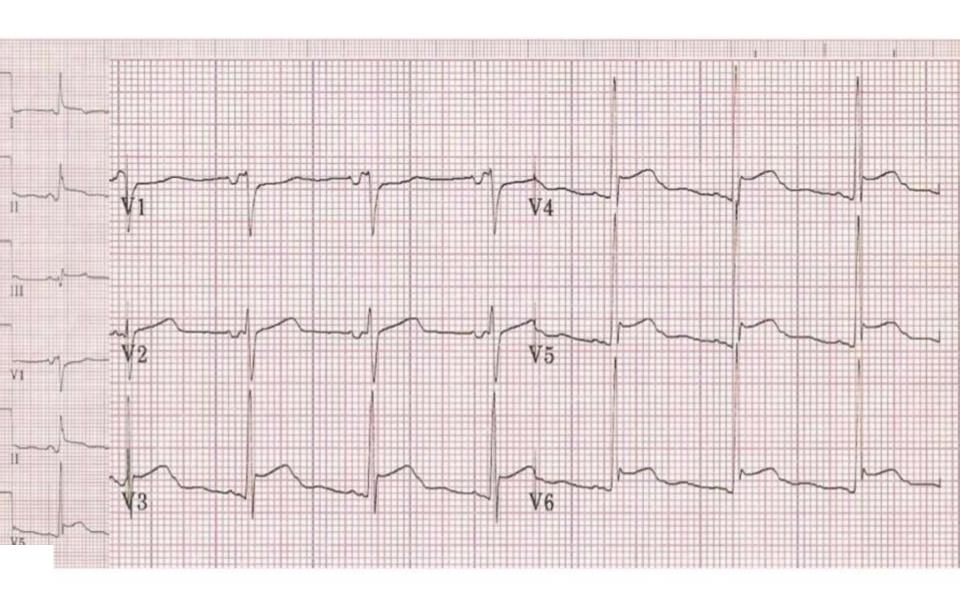
Source: Fuster V. O'Rourke RA. Walsh RA. Doole-Wilson

Not Every ST Elevation is a STEMI!!!

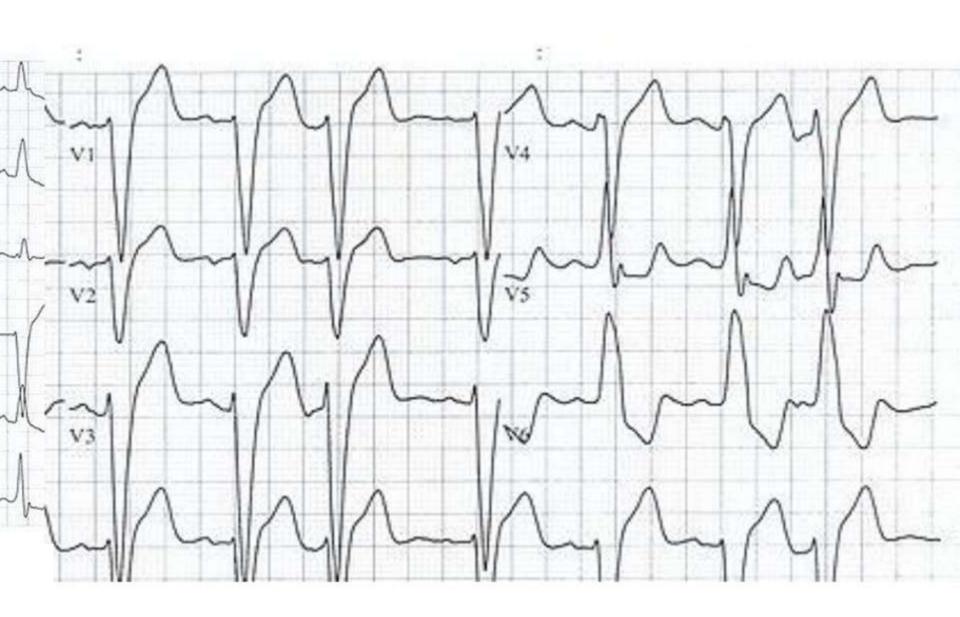
Early Repolarization



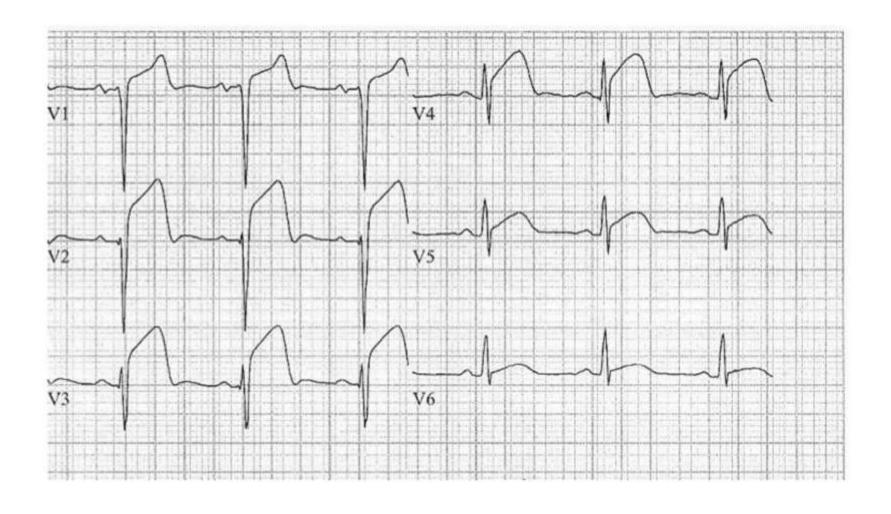
Pericarditis



Left Bundle Branch Block



Back to our patient - ECG



PATIENT HAS A STEMI!!!

STEMI PRE HOSPITAL RX GOALS

- Delivery patients to an appropriate health facility as quickly as possible
- Preventing sudden death and controlling arrhythmia using ACLS when necessary
- Initiating and continuing management of patients during inter facility transfer

Important direct signout from EMS team

- Person who initiated EMS involvement and why
- Complaints at the scene
- Vital signs, physical exam, and notable changes
- Therapies prior to arrival and patient response
- ECGs from outside and en route
- Code status
- Family contact for supplemental information

Management

Initial measures

- IV access
- Continuous cardiac monitoring
- Oxygen

Reperfusion therapy

- Fibrinolysis
- Primary PCI
- Bypass Surgery

Medication

- Antiplatelet Agents
- Anticoagulants
- Beta Blockers
- Statin



General Therapy

MONA

- Morphine (q 5-15 min CLASS I)
- Oxygen (pulse ox>90% CLASS I)
- Nitroglycerin (0.4 mg SL NTG x 3 for ischemic
 - pain CLASS I)
- Aspirin

Aspirin

- Aspirin should be chewed by patients who have not taken aspirin before presentation with STEMI. The initial dose should be 162 mg (Level of Evidence: A) to 325 mg (Level of Evidence: C). Class I
 - In a dose of 162 mg or more, aspirin produces a rapid clinical antithrombotic effect caused by immediate and near-total inhibition of thromboxane A2 production. (ISIS-2->ASA led to 23% reduction in mortality):

- 1. Antithrombotic Trialists' Collaboration. Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. *BMJ*. 2002; 324: 71–86.
- 2. ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. Randomised trial of intravenous streptokinase, oral aspirin, both, or neither among 17187 cases of suspected acute myocardial infarction. Lancet 1988;ii:349-60.



Beta-Blockers

Recommendations - Class la (B)

- ORAL beta-blocker therapy SHOULD BE initiated in the first 24 hours for patients who <u>DO NOT</u> have any of the following:
 - 1) signs of heart failure,
 - 2) evidence of a low output state,
 - 3) increased risk for cardiogenic shock, or
 - 4) relative contraindications to beta blockade
 - > 1AVB > 0.24 sec,
 - 2nd- or 3rd-degree heart block
 - reactive airway disease
- ** There is no study evaluating oral beta blockers alone

Beta-Blockers

Recommendations - Class IIa (B)

- It is reasonable to administer an IV BETA BLOCKER at the time of presentation to STEMI patients who are HYPERTENSIVE and who do not have any of the following:
 - 1) signs of heart failure,
 - 2) evidence of a low output state,
 - 3) increased risk for cardiogenic shock, or
 - 4) relative contraindications to beta blockade
 - > 1AVB > 0.24 sec,
 - > 2nd- or 3rd-degree heart block
 - reactive airway disease

*Risk factors for cardiogenic shock :heart failure, age > 70 , systolic blood pressure < 120, sinus tachycardia > 110 or heart rate < 60, increased time since onset of STEMI symptoms

Beta-Blockers

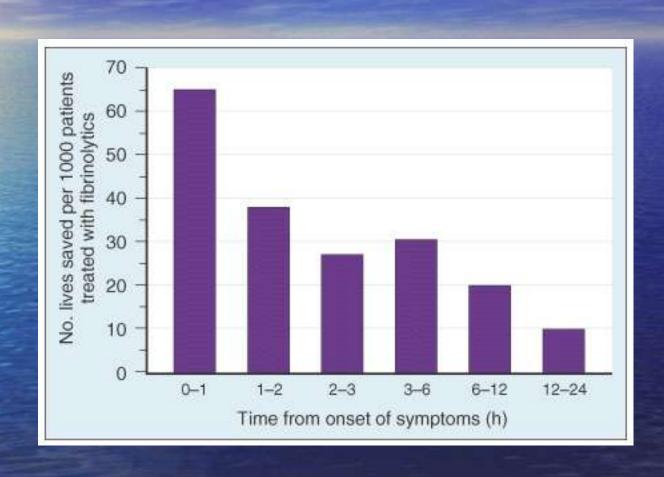
Recommendations - Class III (A)

- IV beta blockers SHOULD NOT be administered to STEMI patients who have any of the following:
 - 1) signs of heart failure
 - 2) evidence of a low output state
 - 3) increased risk* for cardiogenic shock
 - 4) relative contraindications to beta blockade
 - > 1AVB > 0.24 sec,
 - 2nd- or 3rd-degree heart block
 - reactive airway disease

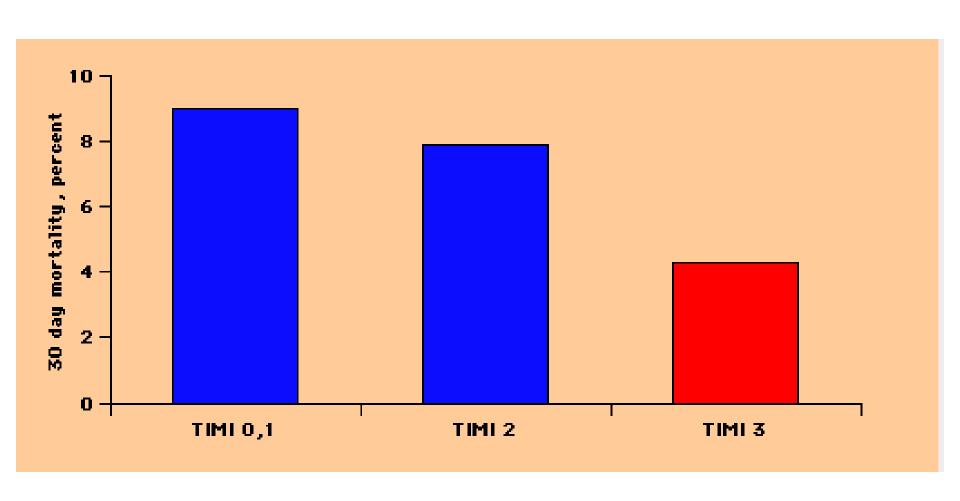
*Risk factors for cardiogenic shock :heart failure, age > 70 , systolic blood pressure < 120, sinus tachycardia > 110 or heart rate < 60, increased time since onset of STEMI symptoms



"Time is Muscle"



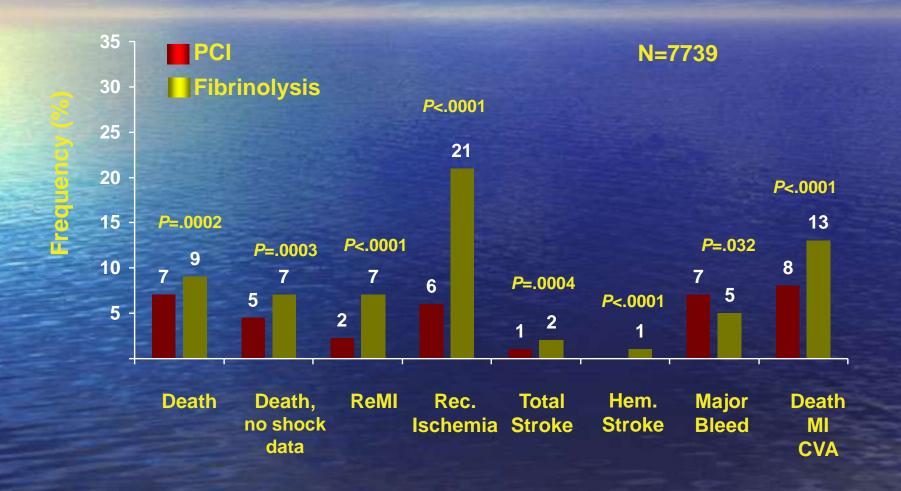
Mortality with TIMI Flow



Reperfusion

- STEMI patients presenting to a hospital with PCI capability should be treated with primary PCI within 90 min of first medical contact as a systems goal. Class la
- STEMI patients presenting to a hospital without PCI capability, and who cannot be transferred to a PCI center and undergo PCI within 90 min of first medical contact, should be treated with fibrinolytic therapy within 30 min of hospital presentation as a systems goal, unless fibrinolytic therapy is contraindicated. Class Ib

PCI vs Fibrinolysis for STEMI: Short-Term Clinical Outcomes



Brief Review of Thrombolytic Trials

GISSI-1: Streptokinase 18% reduction in mortality at 21 d

GUSTO-1: tPA. 15% reduction in 30-day mortality compared to Streptokinase

GUSTO-3: Reteplase had no benefit over tPA but is easier to use (double bolus)

ASSENT: TNKase is similar to tPA but with less non-cerebral bleeding and better mortality with symptoms>4 hrs: Single bolus, fibrin selective, resistance to PAI-1

*Overall risk of ICH is 0.7%; Strokes occurred in 1.4%

Contraindications

Absolute Contraindications

- Intracranial neoplasm
- Recent (<3 months) intracranial surgery or trauma
- recent (<3 months) ischemic stroke
- h/o hemorrhagic stroke
- Active or recent bleeding

Relative Contraindications

- -BP > 180 systolic
- H/o ischemic stroke
- Recent (<4 weeks) internal bleeding
- Thrombocytopenia

Additional Notes

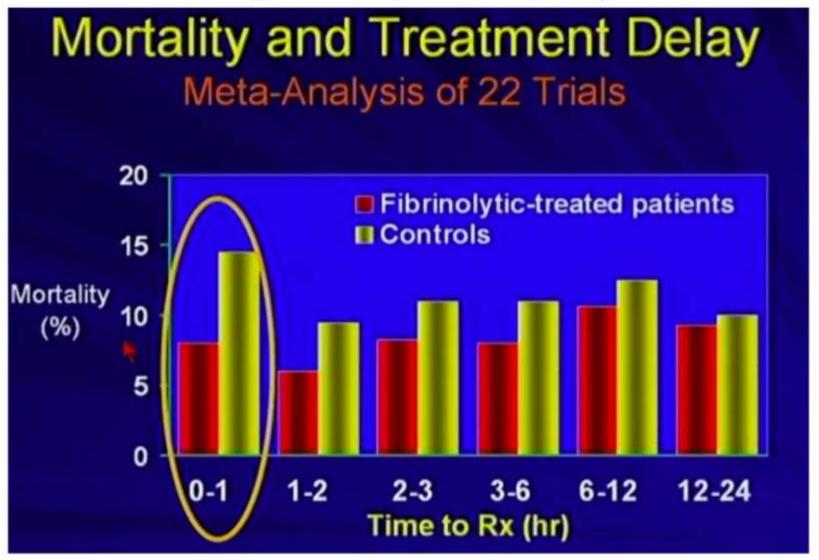
Treatment window

- Within 12 hours of onset of chest pain
- Never give after 24 hours
- If ongoing chest pain after 12 hours and low risk of bleeding may give thrombolysis

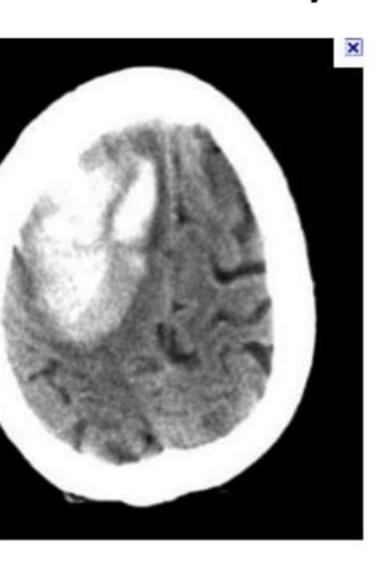
Success of thrombolysis is assessed by

- Resolution of Chest pain
- >50% reduction in ST elevation
- Development of accelerated idioventricular rhythm

50% reduction in mortality with lytics if given promptly



Fibrinolytics-Risk of ICH



- Elderly
- < 70kg
- Uncontrolled hypertension
- Lowest risk with streptokinase

Anticoagulants

- Patients undergoing reperfusion with fibrinolytics should receive anticoagulant therapy for a minimum of 48 hours (unfractionated heparin) or up to 8 days
- Anticoagulant regimens with established efficacy include:
- UFH (LOE: C)
- Enoxaparin (LOE:A)
- Fondaparinux (LOE:B)

Primary Percutaneous Coronary Intervention

- Superior to thrombolysis in most cases
- Less reinfarction, death
- Less stroke, bleeding

2013 STEMI guideline

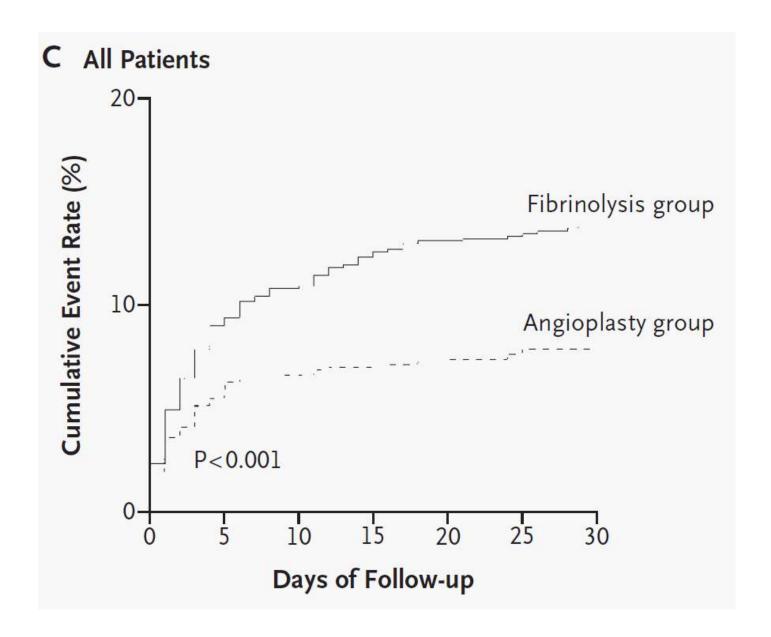
- Primary PCI is the preferred treatment for STEMI
 - At a hospital with PCI, the goal is PCI within 90 minutes
 - At a hospital without PCI, the goal is transfer and PCI within 120 minutes
 - If transfer for PCI within 120 minutes is not feasible,
 use fibrinolytic therapy unless contraindicated, with a door-to-needle time less than 30 minutes

2013 STEMI guideline

 Patients with cardiogenic shock or severe HF should be transferred immediately for PCI

 Patients who have failed to reperfuse with fibrinolytic therapy should be transferred urgently for PCI

 Therapeutic hypothermia is recommended for comatose patients with STEMI and cardiac arrest



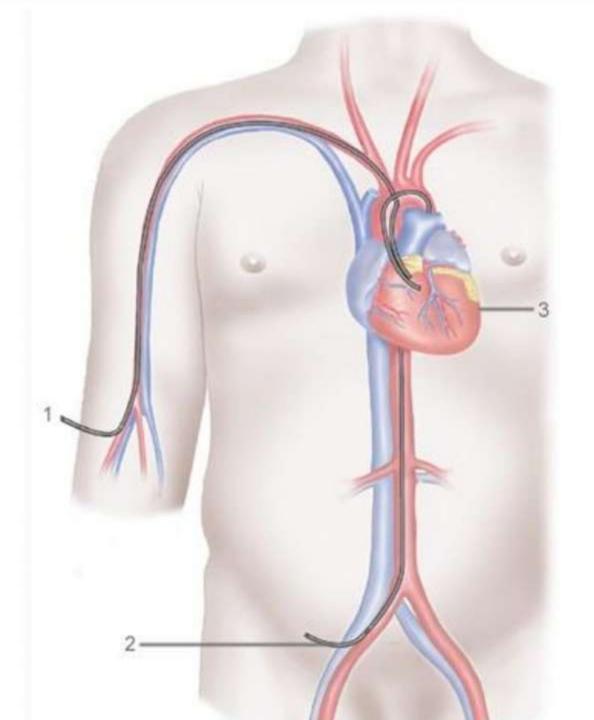
Andersen HR, et al. DANAMI-2 trial. NEJM 2003; 349: 733-742.

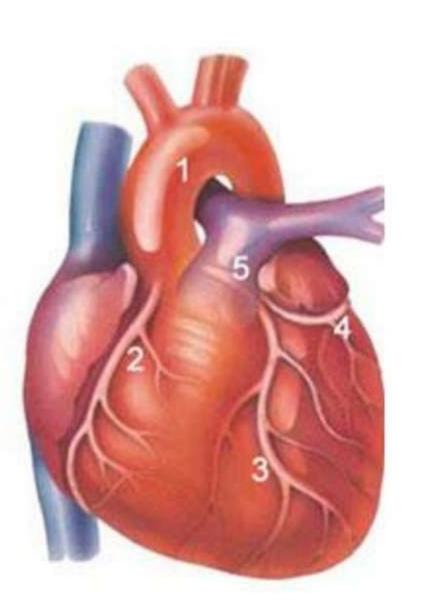
Table 8. Measures To Improve Door-To-Balloon Times¹¹⁶

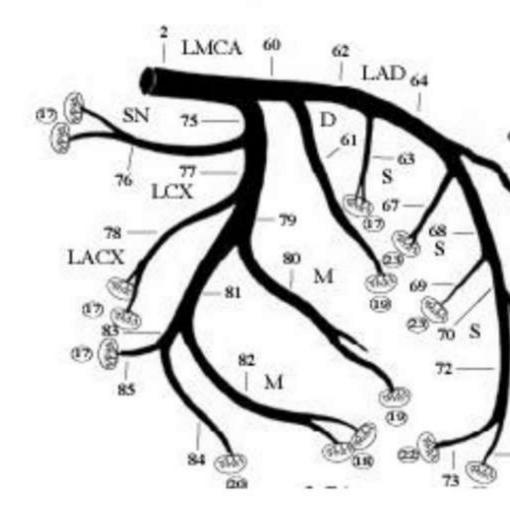
Strategy	Time Saved (min)
Emergency medicine clinician activates the catheterization laboratory.	8.2
Single call to a central page operator activates the laboratory.	13.8
Emergency department staff activates the catheterization laboratory while the patient is en route to the hospital.	15.4
Staff members are expected to be in the catheterization laboratory within 20 minutes after being paged (vs 30 minutes).	19.3
An attending cardiologist is on-site at all times.	14.6
The hospital gives real-time feedback on the door- to-balloon times to the emergency department and catheterization laboratory staffs.	8.6

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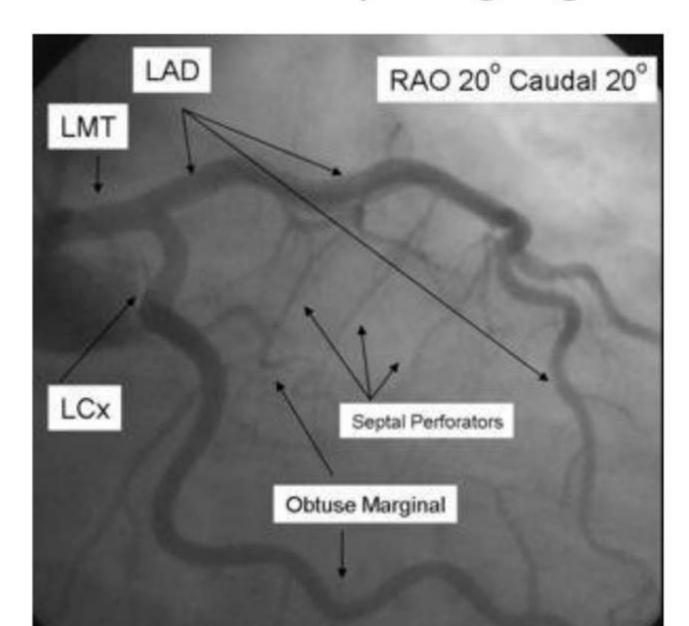
Coronary Angiography

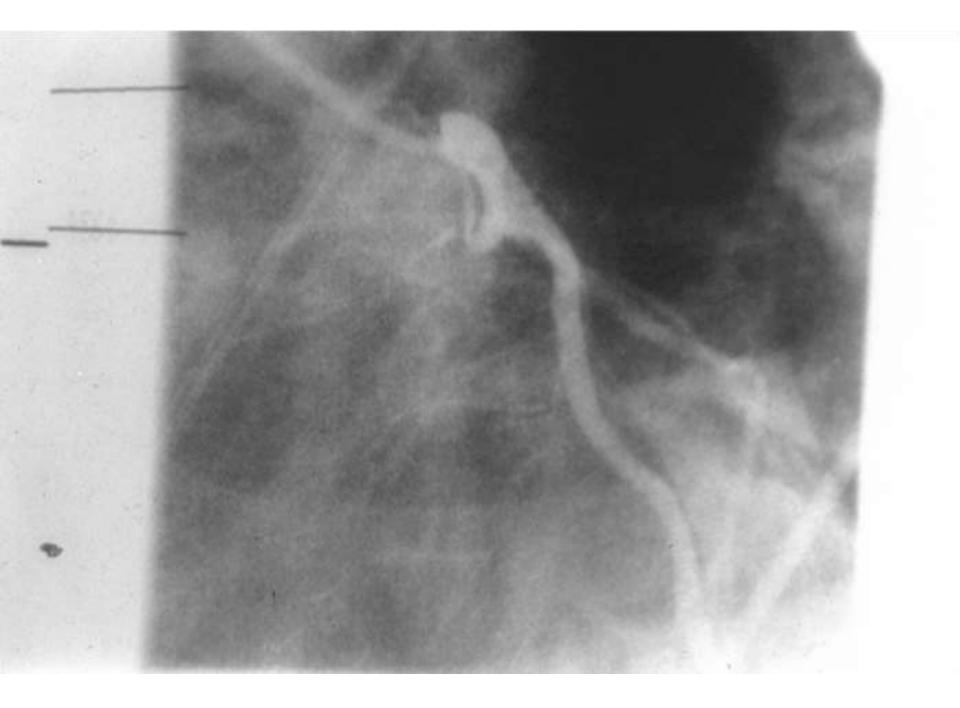


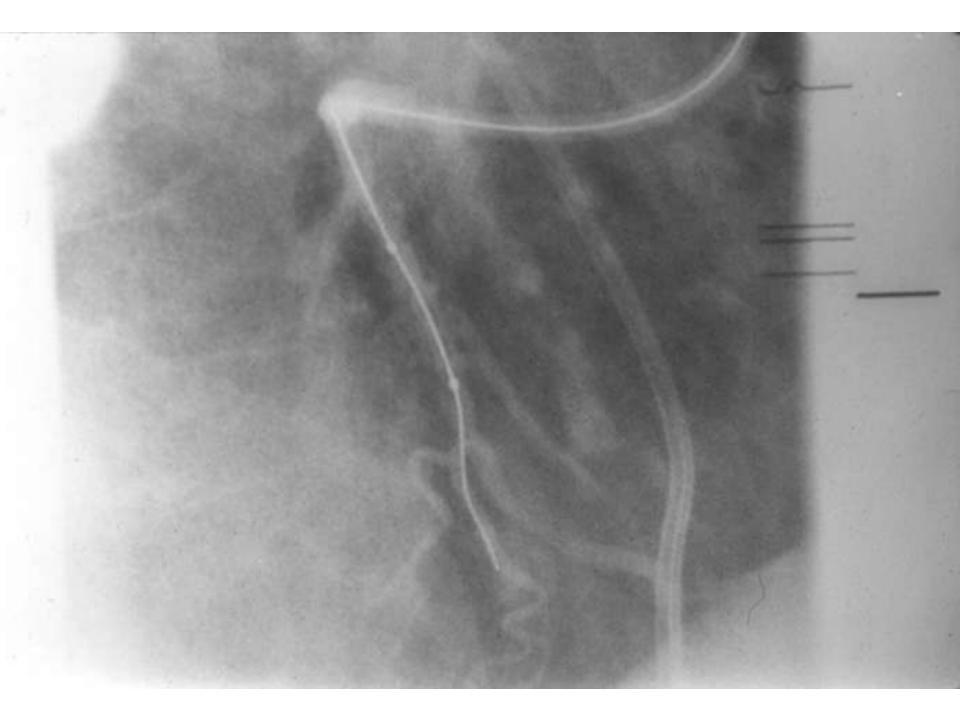


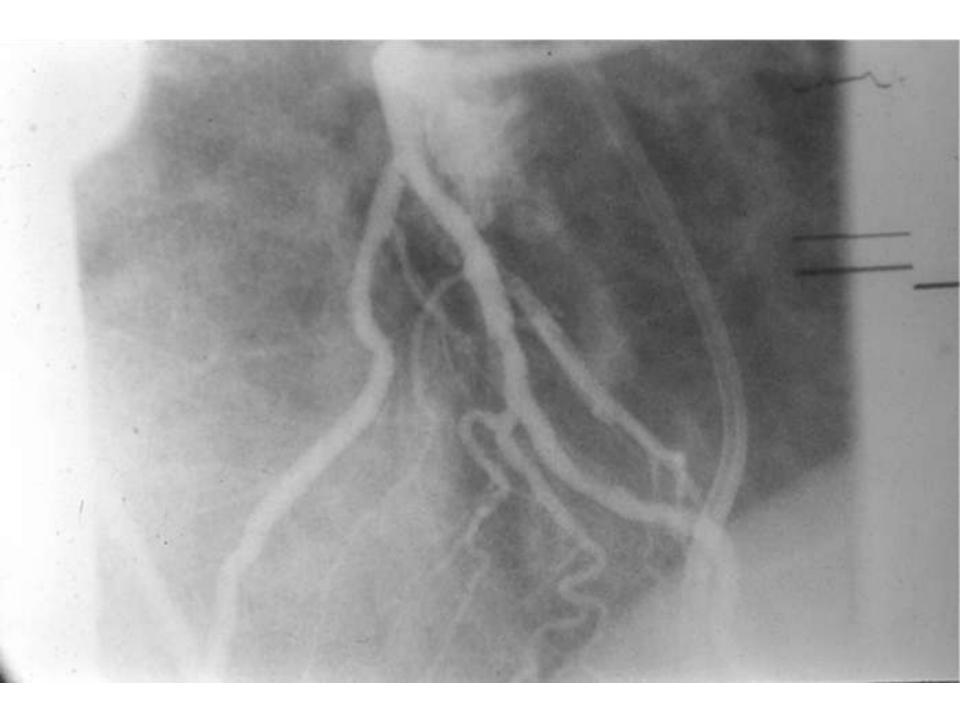


Normal Coronary Angiogram









After STEMI Care

- All patients should be admitted to a bed with continuous cardiac monitoring
- All patients should be given (if no contraindications)
 - Beta Blocker (lifelong)
 - ACE inhibitor (lifelong)
 - Statin (lifelong)
- Additional medication
 - Spironolactone (if low EF, diabetic)

Post STEMI Risk Assessment

Coronary Angiography after STEMI

- Patients who fail thrombolysis (continued chest pain, failure of ST segment resolution)
- Patients who have high risk features
 - Heart failure (either clinical or Low EF)
 - Serious Arrhythmias

Patients who don't have high risk features after STEMI should undergo Exercise ECG stress testing for risk stratification

Complications of Myocardial Infarction

- Arrhythmias
- Ventricular Septal Perforation
- Ischemic Mitral Regurgitation, Papillary Muscle Rupture
- Ventricular Free Wall Rupture
- Systemic Embolism
- Ventricular Aneurysm
- Pericarditis
- Cardiogenic Shock (another lecture)

Ventricular Arrhythmias

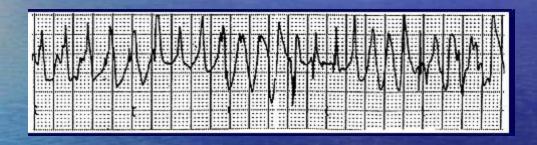


- 60-110 BPM; Up to 20% STEMI patients have this
- Usually a result of reperfusion; no specific therapy needed if HD stable. Otherwise, atropine or even atrial pacing may increase sinus rate to overdrive pace the AIVR
- Routine post-MI management with B-blockers, ACE, etc.

PVC's

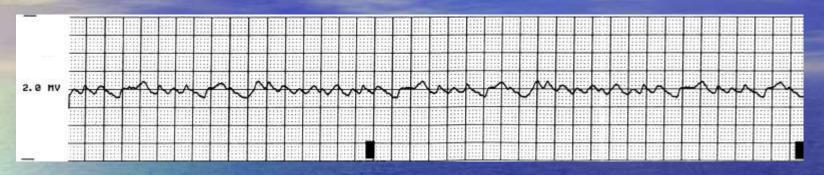
- Extremely common, along with short runs of NSVT
- Amiodarone won't increase mortality, other antiarrhythmics (other than B-blockers) do.
- B-blockers, electrolytes
- Best if no antiarrhythmics are used

Not So Benign Rhythm



- Ischemic VT is often polymorphic; HR>100-110 BPM
- Higher risk with more LV damage and in first 2 days after MI
- Treat: DCCV, cath lab (if needed), electrolyte correction, amiodarone, lidocaine, B-Blockers

If That Didn't Make You Nervous...



Primary VF: Sudden event with no warning--10% STEMI patients before lytics. MUCH MUCH less now

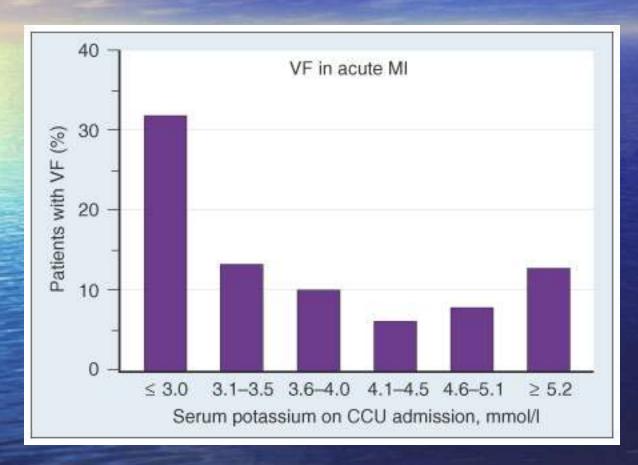
Secondary VF: Occurring in setting HF or shock

Late VF: >48 hrs after MI-->Increased risk with IVCD, anterior wall MI, persistent SVT early in course, and RV infarction requiring pacing

***Have to worry about structural complication (free wall rupture)/ischemia

Treat: Non-synced DCCV, electrolyte correction

Why get worked up about electrolytes?



NOTE: Pre-lytic study

Nordrehaug JE, van der Lippe G: Hypokalemia and ventricular fibrillation in acute myocardial infarction. Br Heart J 50:525, 1983.

Sinus Bradycardia/Junctional Escape Rhythm

- 4-5% of STEMI patients have a bradyarrhythmia
- Sinus node ischemia-Blood supply to SA node is: 65% RCA, 25% LCX, 10% dual supply.
- Most commonly seen in Inferior/posterior MI's.
- Often induced by vagal reaction that may be protective

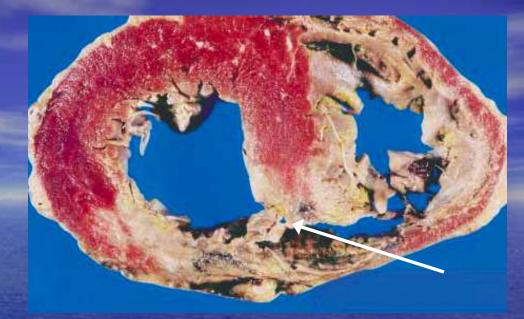
Atrioventricular Block

First-Degree: Usually the RCA and does not require treatment.

Hold the B-blocker for PR>240 ms

- Second-Degree: Usually RCA disease and does not require treatment unless HR less than 50 and arrhythmia or symptoms. Otherwise, atropine or pace
- Third-Degree: Can be from any location of infarct. Can be preceded by Mobitz II Block
 - Pace for symptoms and for hemodynamic support. Usually not needed in inferior MI's as block is transient (pace for HR<40-50)

Post-MI VSD



- ~2% of acute MI's prior to reperfusion era
- ~0.2% in GUSTO-I streptokinase trial
- Without reperfusion, usually occurs within first week
 - Day 1--Large intramural hematomas that dissect
 - Day 3-5--Coagulation necrosis
- 24 hr or less if receive lysis--Lytics reduce infarct size but may promote hemorrhagic dissection of myocardium

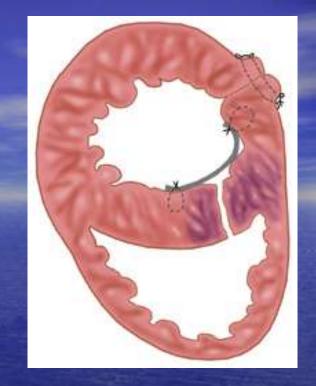
Symptoms, Exam, and Diagnosis

- Chest pain, dyspnea
- PE: Harsh, holosystolic murmur along sternal border radiating to base/apex/R parasternum; thrill in 1/2 patients; S3; Loud P2; TR.
- Compared to acute MR, murmur is loud. Up to 20% of patients may have MR as well though



CCU Management

- IABP
- Ventilation
- Diuresis/HF Management
- Inotropes (can increase shunt)
- Nitroprusside if tolerated (can cause hypotension)
- Mortality with conservative management is HIGH (24%, 46%, 67-82% at 24 hrs, 1 wk, and 2 months, respectively)
- Ultimately, mechanical closure needed (surgery vs. percutaneous)-TIMING is questionable but clinical status should not preclude this



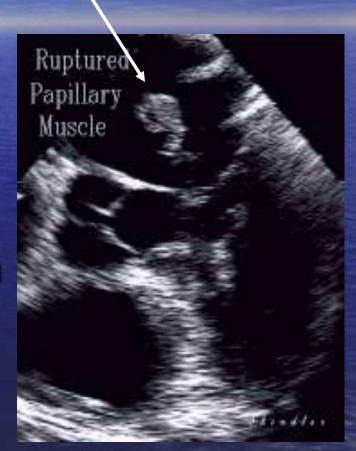
Acute Mitral Regurgitation

- Caused by papillary muscle ischemia or rupture (less likely). Rupture is usually partial since total is essentially incompatible with life
- Usually in setting of inferior MI involving the posteromedial papillary muscle (single PDA blood supply as opposed to anterolateral)
- Rupture usually occurs 3-5 days post-MI and in 1% of MI's and requires emergent operative repair (50% mortality in 24 hrs)
- Accounts for 7% of cardiogenic shock and 5% of mortality associated with acute MI
- Area of infarction does NOT have to be large

Symptoms, Exam, Diagnosis

Symptoms: Those of heart failure

PE: May or may not hear loud systolic murmur (need a gradient)



CCU Management

- Mechanical ventilation if needed
- IABP—especially for hypotension
- PCI if papillary m. ischemia (not rupture)
- Afterload reduction (nitroprusside if possible) to MAP of 70-80 mm Hg
- Since mortality is 90% with medical therapy alone, surgery is the major therapy of choice
 - Perioperative mortality 20-25%
 - Overall surgical mortality is even higher

Free Wall Rupture

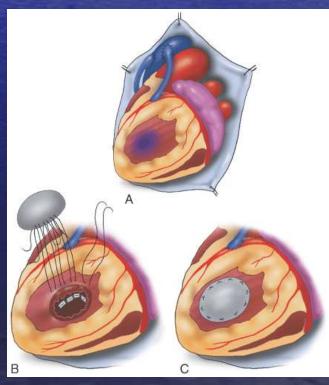
- ~10% of patients who die in hospital from STEMI
- Most commonly between 1 and 4 days (up to 3 weeks)
- Caused by tear or dissecting hematoma
- More common with fibrinolysis compared to PCI
- More common in patients without previous infarction

Symptoms, Exam, Diagnosis

- Acute symptoms include sudden chest pain (esp with cough, strain) and sudden death
- Subacute symptoms: Pericarditis-like symptoms (chest pain, nausea, vomiting)
- Exam (think HF and tamponade): JVD, pulsus, diminished heart sounds, rub, possibly a new murmur

Treatment

- Pericardiocentesis if time
- Surgical repair is the only treatment
- Mortality is reasonable if patient gets to the OR in time
- 90% mortality without surgery



Summary of Acute STEMI Complications

- Much more rare in the reperfusion era
 - Look for them especially in delayed presentation
- Arrhythmias are most common complication and may require emergent treatment
- VSD's, papillary muscle rupture, and free wall ruptures carry a VERY high mortality and require emergent surgical consultation
 - Support mechanically until patient receives operation

Treatment of ACS

UA/NSTEMI

- Usually due to plaque rupture with partial occlusion
- Treatment options:
 - Early PCI (< 24 hrs)</p>
 - Delayed PCI (> 24 hrs)
 - Medical therapy

STEMI

 Usually due to plaque rupture with total occlusion

- Treatment options:
 - Primary PCI is preferred
 - Fibrinolytic therapy
 - Medical therapy

Initial treatment of ACS

- Aspirin at initial presentation
- P2Y₁₂ inhibitor (clopidogrel, ticagrelor, . . .)
- Statin (atorvastatin 80 mg daily)
- Anticoagulation (heparin, enoxaparin, . . .)
- Nitrates / morphine for chest pain
- Revascularization
- Scenarios:
 - STEMI with PCI available
 - STEMI with PCI unavailable
 - High-risk UA/NSTEMI
 - Low-risk UA/NSTEMI

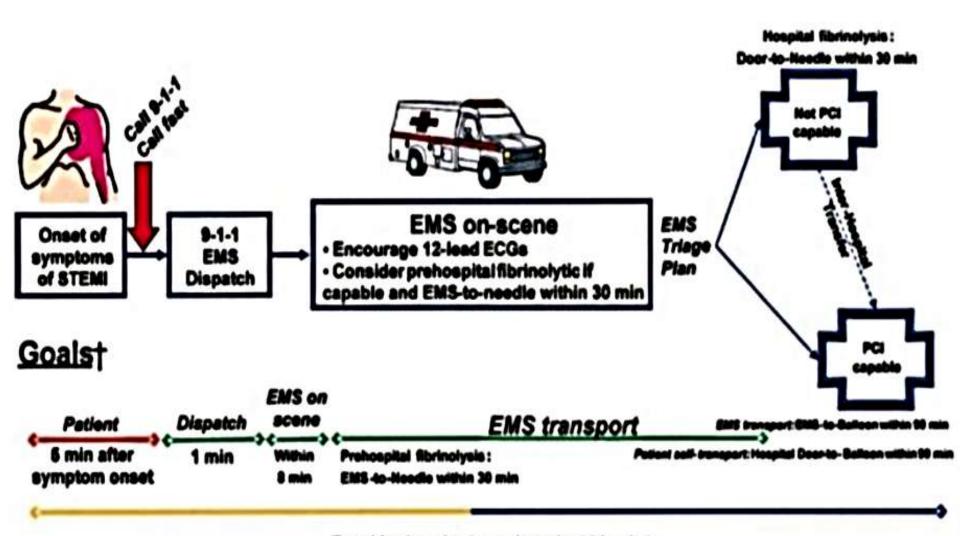
Initial treatment for STEMI with PCI available

- PCI with 90 minutes, or transfer for PCI within 120 min
- Aspirin 325 mg
- P2Y₁₂ inhibitor
 - Ticagrelor 180 mg
 - Prasugrel 60 mg
 - Clopidogrel 600 mg
- Anticoagulant
 - Heparin 70 units/kg
 - Bivalirudin (usually started in the cath lab)
- High dose statin (atorvastatin 80 mg)

Initial treatment for STEMI with PCI unavailable

- Fibrinolytic therapy within 30 minutes
- Aspirin 325 mg
- Clopidogrel
 - initial dose 300 mg for patients < 75 years old
 - initial dose 75 mg for patients ≥ 75 years old
- Enoxaparin, fondaparinux, or heparin
- High dose statin

Targets



Total ischemic time: Within 120 min*

*Golden Hour = First 60 minutes