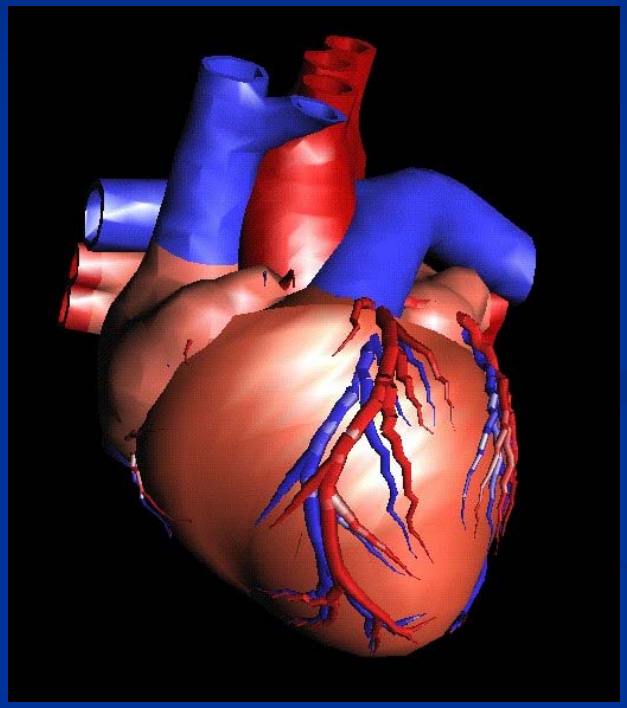
Acute Coronary Syndrome

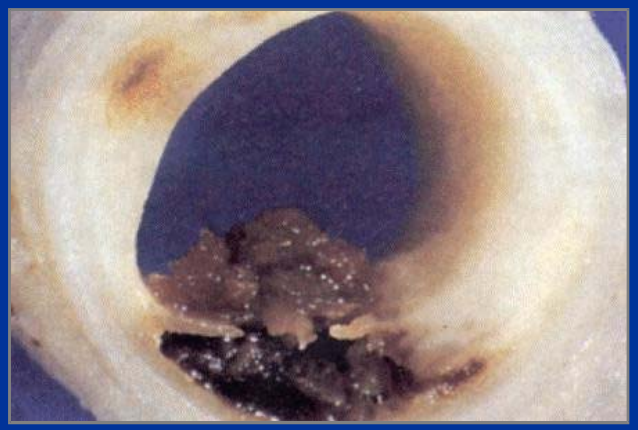


Dr. Annie Sun

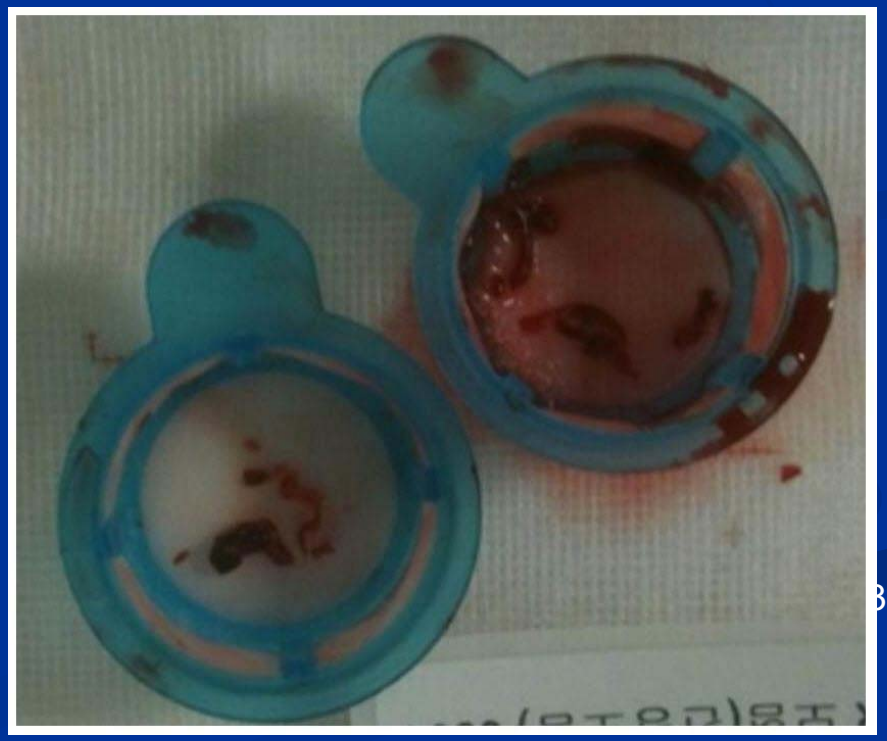
What is ACS?

- unstable angina
- non- ST elevation MI (NSTEMI)
- ST elevation MI (STEMI)

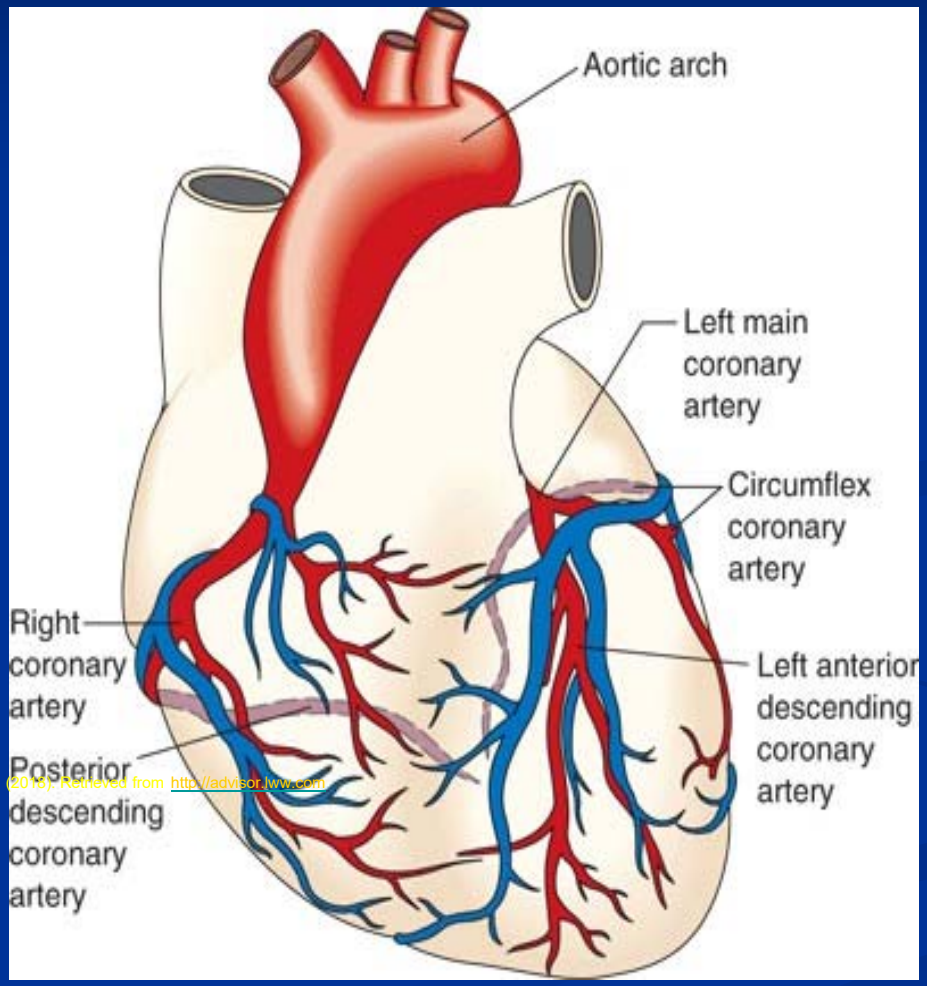
ACS/ STEMI Review



90% of acute MIs caused by thrombus formation from rupture of unstable plaques

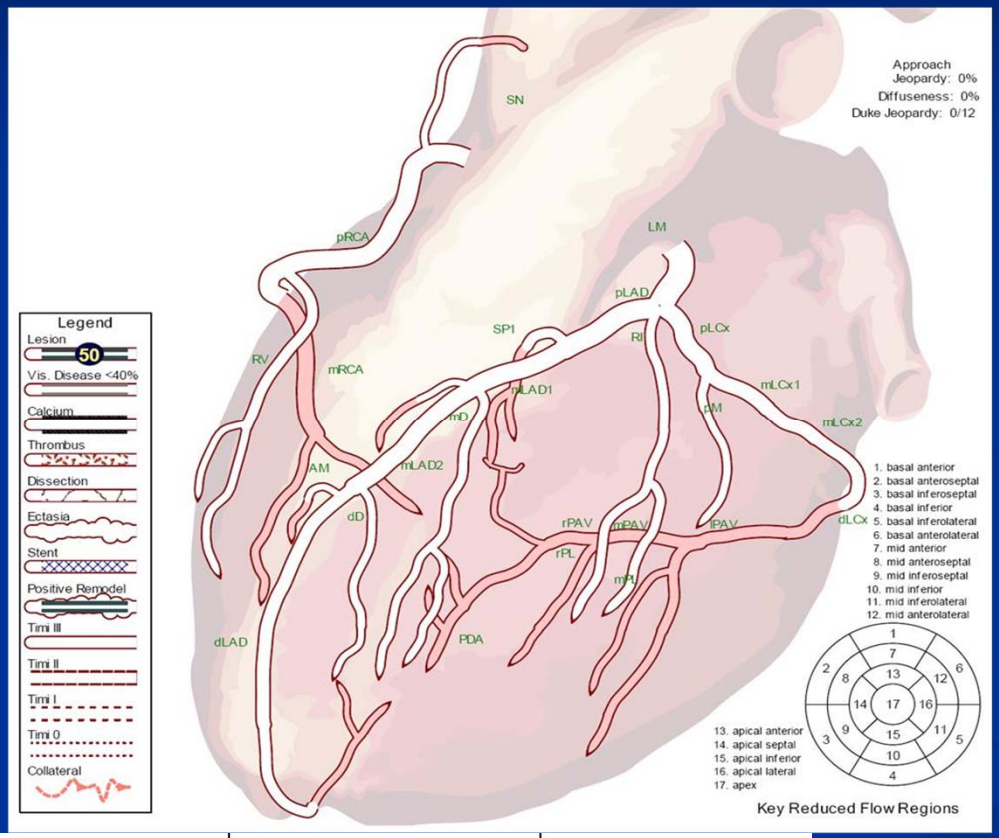


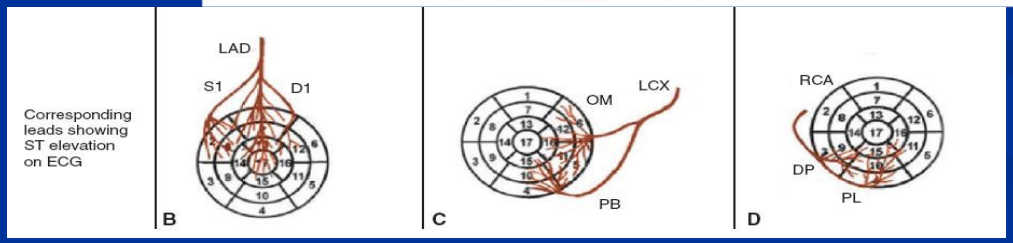
Coronary Circulation



Lippincott advisor. (2010). Retrieved from http://advisor.lww.com

CARAT Diagram

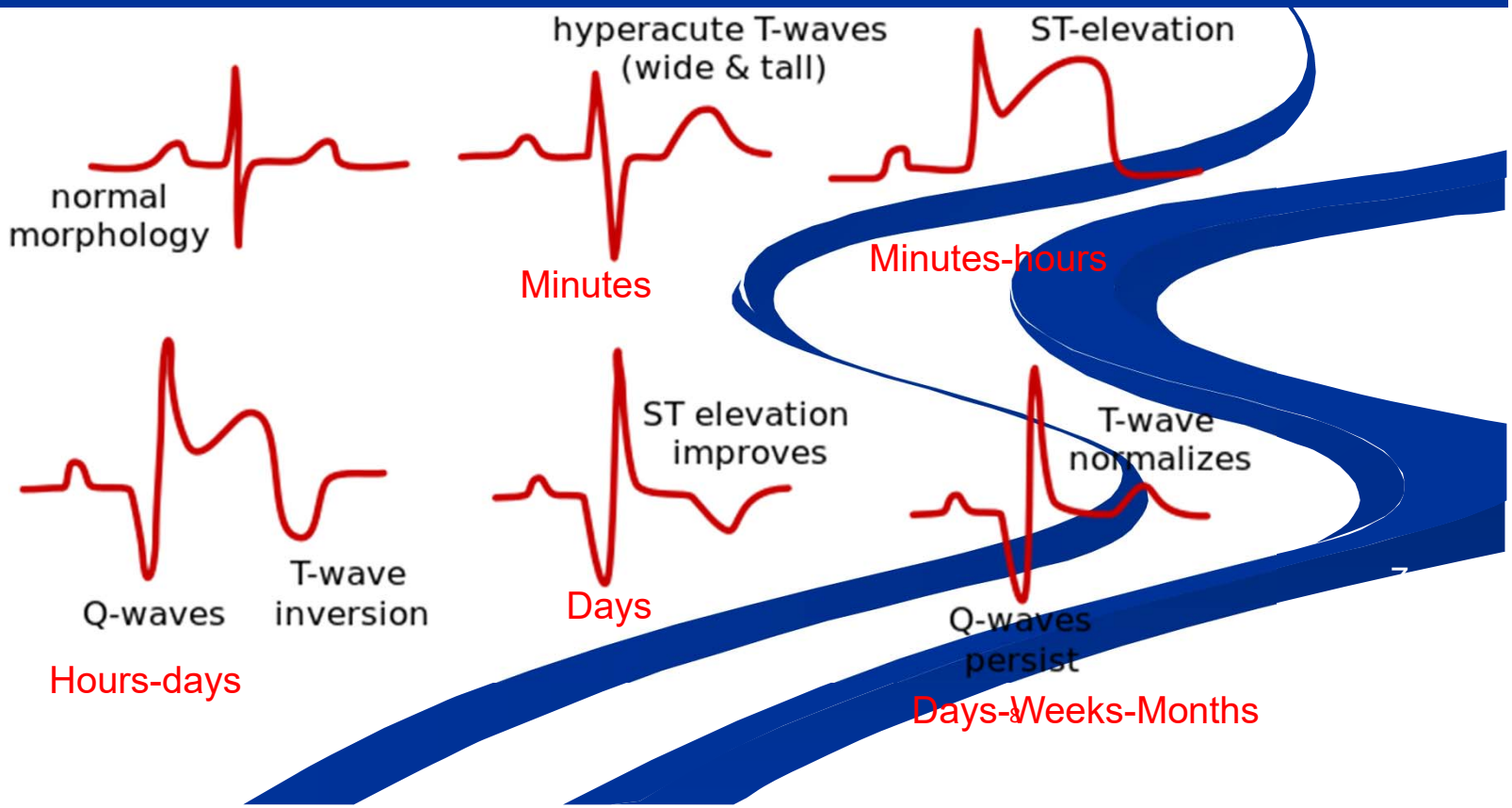




ACS SUMMARY

	STEMI	NON-STEMI	ANGINA
Chest Pain	Greater than or equal to 20 minute duration	Greater than or equal to 20 minute duration	Usually 3-5 minute duration
ST Segment	Elevation of at least 1mm in 2 contiguous leads	Depression for up to 24 hours	Transient depression possible
T Waves	Peaked / Elevated	Inversion	Transient inversion possible
Cardiac Markers	Elevated	Elevated	Not elevated

EVOLUTION OF ISCHEMIA



Unstable Angina

- ischemic chest pain occurring at rest or with minimal exertion, rapid deterioration of previously stable angina (crescendo angina), or new onset severe angina without positive Marker.

NSTEMI

- the development of heart muscle necrosis results from an acute interruption of blood supply to a part of the heart which is demonstrated by an elevation of cardiac markers (CK-MB or Troponin) in the blood and the absence of ST-segment elevation in ECG

ST Elevation MI (STEMI)

- the development of cardiac muscle necrosis results from an acute interruption of blood supply to a part of the heart that is demonstrated by the presence of ST-segment elevation in electrocardiography (ECG) and an elevation of cardiac markers (CK-MB or Troponin) in the blood

Risk Factors: Non-Modifiable

- Age
  - Age = Risk
- Race
- Gender
  - Men > Women before menopause
  - Women's risk after menopause; almost = Men
- Positive Family History: first degree relative (ie, parent or sibling) prior to age 50 (males) or 60 (females)

Risk Factors: Modifiable

- Major Risk Factors are
  - Smoking
  - Moderate alcohol intake
  - Sedentary Lifestyle
  - Obesity
  - Stress
  - Diet
  - Hypertension
  - Hypercholesteremia
  - Diabetes
  - CKD

Risk is assessed

- Low: normal ECG (or nonspecific changes), Troponin T negative
- Intermediate: nonspecific ECG changes, Troponin T borderline, ongoing chest pain
- High: transient ST elevation (> 1 mm) or depression (> 1 mm, or sustained ST depression (> 2 mm), T wave inversion, Troponin positive

Risk assessment tools

GRACE

TIMI

TIMI Risk Score for UA / NSTEMI

Historical	Points
Age >= 65	1
>= 3 coronary artery disease (CAD) risk factors (family history, hypertension, elevated blood cholesterol, diabetes mellitus, smoker)	1
Known CAD (stenosis >=50%)	1
ASA use in past 7 days	1
Presentation	
Recent (<= 24 hrs) severe angina	1
Elevated cardiac markers	1
ST deviation >= 0.5mm	1
Risk score = Total Points (0-7)	

Cardiac Events (%) by Risk Score

Risk Score	30 Day Mortality (%)
0	0.8
1	1.6
2	2.2
3	4.4
4	7.3
5	12
6	16
7	23
8	27
> 8	36

GRACE "ACS" RISK CALCULATOR

(In-Hospital Death Basic) Ver: 4.7

GRACE Risk Score Calculator

Killip Class* 1	Risk Points	SBP (mmHg)	Risk Points	Heart Rate	Risk Points	Age (yrs)	Risk Points	Creatinine Level (umol/L)	Risk Points	Other Risk Factors	Risk Points	
I	0	≤ 80	58	≤ 50	0	≤ 30	0	0-34	1	Cardiac Arrest	39	
II	20	80-99	53	50-69	3	30-39	8	35-70	4	ST-Segment Deviation	39	
III	39	100-119	43	70-89	9	40-49	25	71-105	7	Cardiac Enzyme ↑	14	
IV	59	120-139	34	90-109	15	50-59	41	106-140	10			
		140-159	24	110-	24	60-69	58	141-176	13			
		160-199	10	150-	38	70-79	75	177-353	21			
		≥200	0	≥200	46	80-89	91	>354	28			
						≥90	100					GRACE Risk Score
		+		+		+		+		+		

_____/_____/_____
Completed by Date Time

Low Risk				*1-108*
Intermediate Risk				*109-140*
High Risk				*>140*

* If using web based calculator record score in Grace Risk Score column *

**A photocopy of this document should be faxed with the patient angiogram referral and accompany chart on transfer

*1 Killip Classes:
I = no clinical signs of heart failure
II = basal crackles (mild pulmonary congestion), an S3 & elevated JVP
III = extensive crackles (frank acute pulmonary edema)
IV = cardiogenic shock (systolic BP less than 90 mm Hg, hypo perfusion & evidence of peripheral vasoconstriction– oliguria, cyanosis, sweating)

Website for GRACE RISK calculator: http://www.outcomes-umassmed.org/grace/acs_risk/acs_risk_content.html

Assessment of Chest Pain

P - *P*recipitating factors, provoking, preventable

Q - *Q*uality, quantity

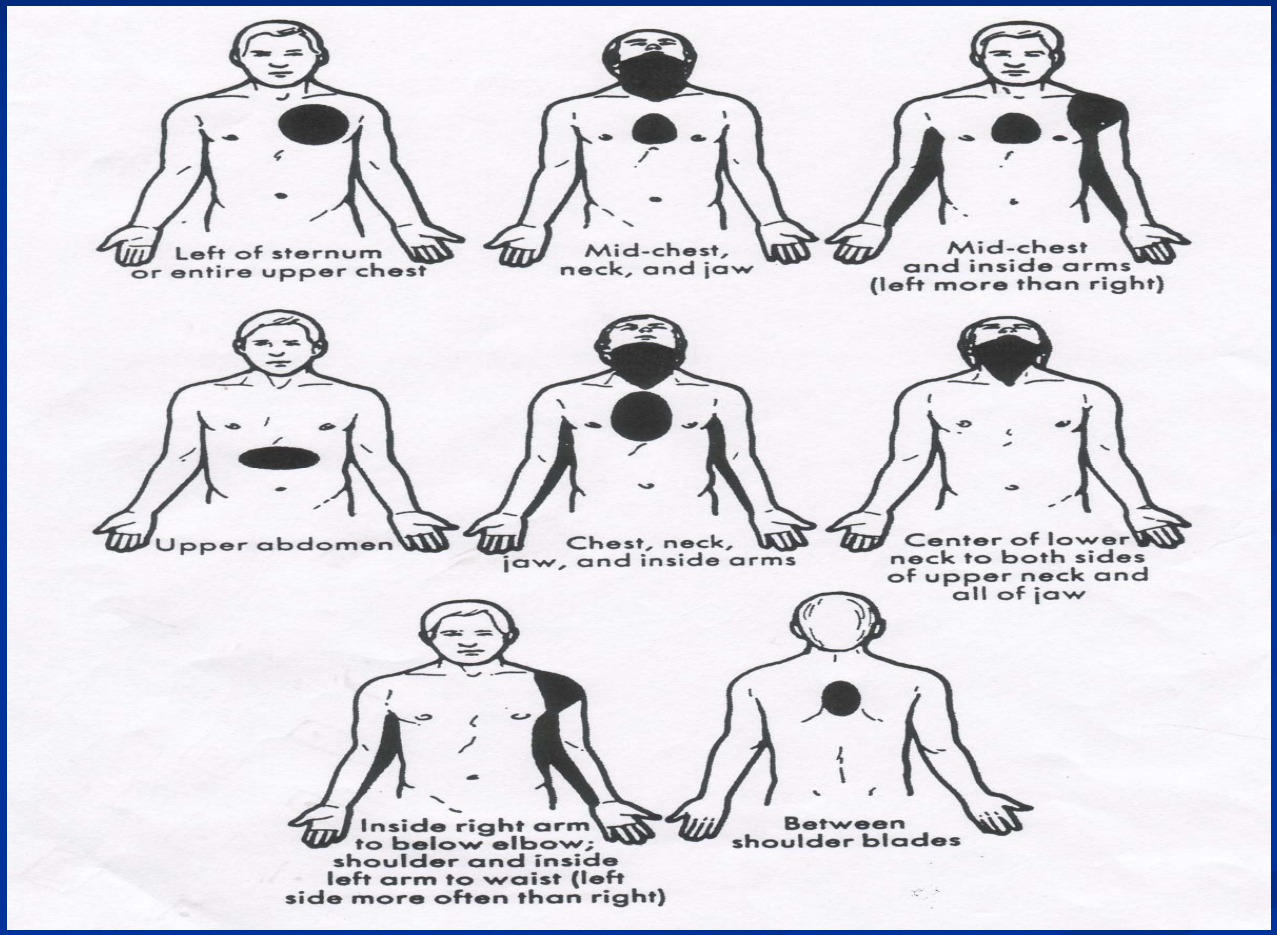
R - *R*adiation, reproducible, relief

S - *S*ymptoms associated with pain

Assessment of Chest Pain

- Onset
- Location
- Radiation
- Intensity
- Type
- Precipitating Factors
- Aggravating Factors
- Relieving Factors
- Associated symptoms
- Reproducible

Location of Myocardial Pain



Associated S & S of Cardiac Pain

- Dyspnea, SOB
- Fatigue
- Diaphoresis
- Nausea and vomiting
- Numbness, tingling
- Poor Pallor

Differential Diagnosis

- PE
- Aortic Dissection
- Tension Pneumothorax
- Pericardia tamponade
- Esophageal rupture
- Pulmonary causes
- Gastrointestinal causes
- Musculoskeletal causes
- Psychiatric causes
- Other conditions: i.e. Function

Diagnostic Investigation

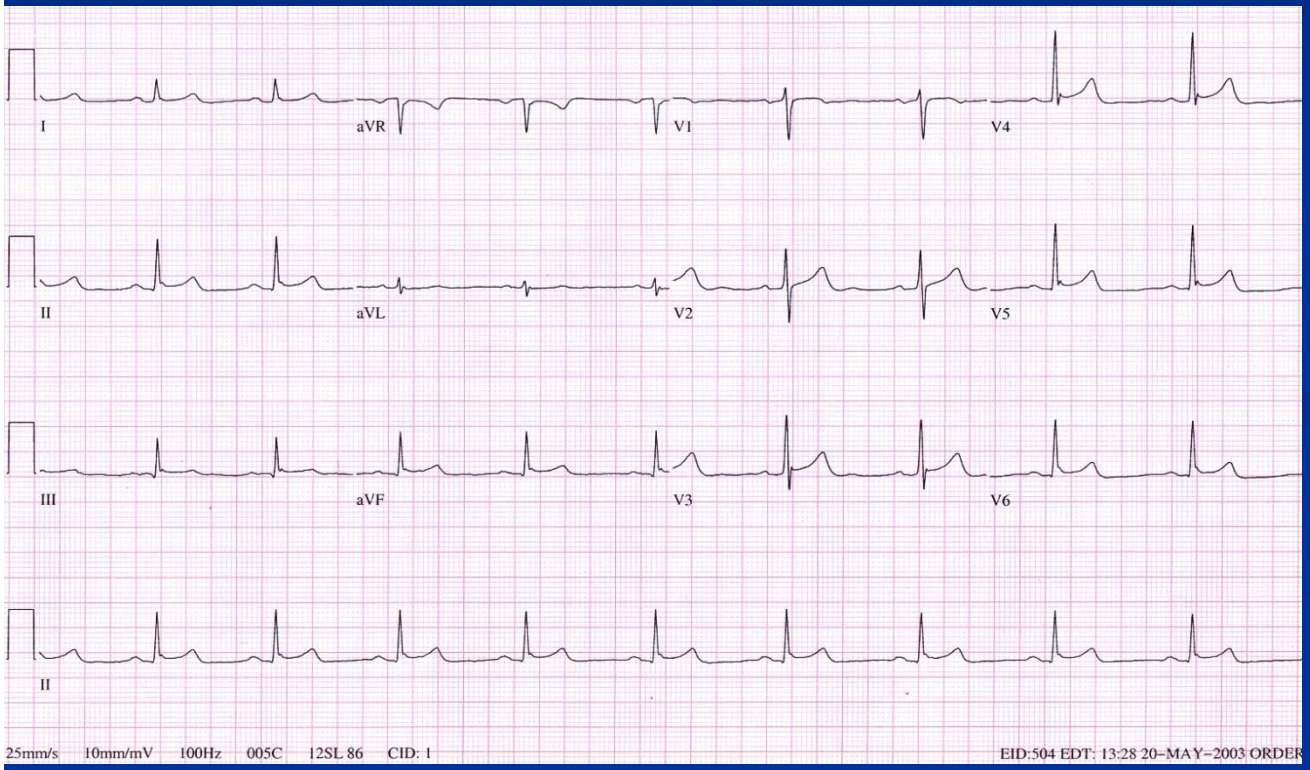
1. Blood Work
2. ECG
3. CXR
4. Echo
5. MPI
6. Stress Test
7. Angiogram

Troponin T- High Sensitivity

Troponin T HS

- 1-14 ng/L negative
- 15-49 ng/L non-specific/non-diagnostic- repeat in 2-4 hrs
- 50-109 ng/L borderline elevation- repeat in 2-4 hrs
- >/= 110 ng/L positive marker

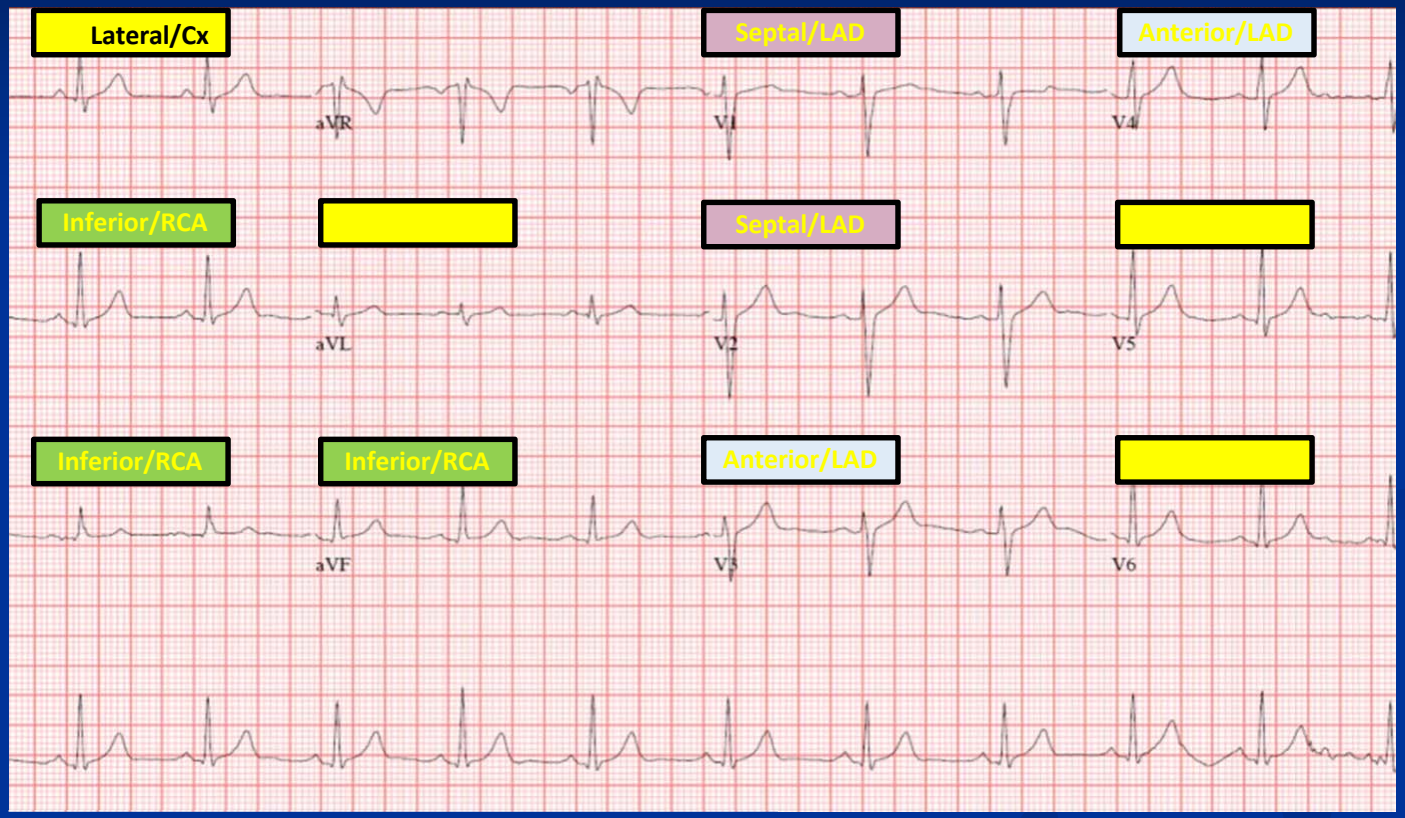
2. Electrocardiogram (ECG):



Views the electrical activity of the heart

- Useful in assessing for ischemia or infarct as well as heart rate and rhythm

12 Lead ECG

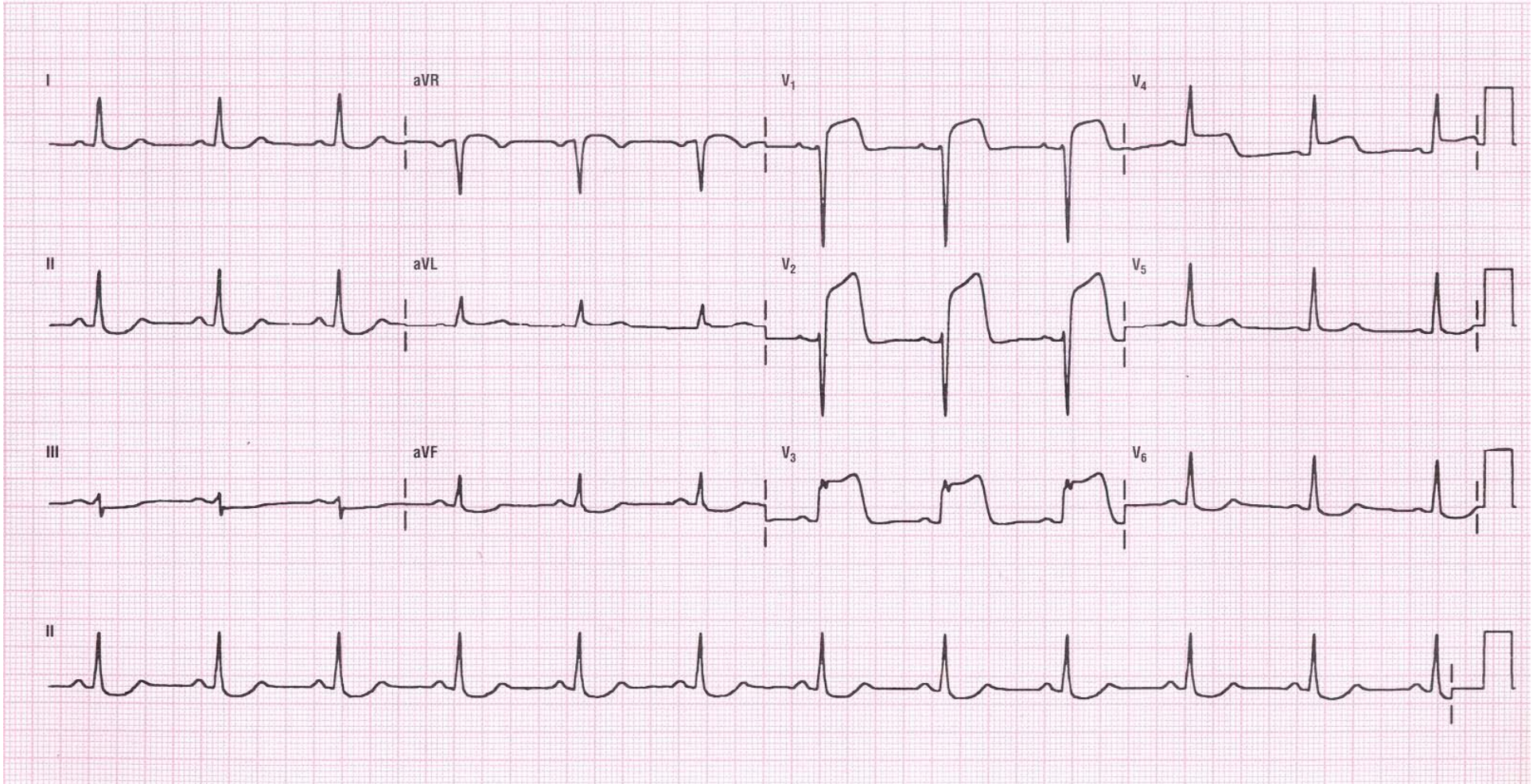


Note: the above Lead II strip is 10 seconds in duration

ECG zone of injury

- • S- septal- V1, V2
- • A- anterior- V3, V4
- • L- lateral- V5, V6, I,avL
- • I- inferior- II,III, AvF

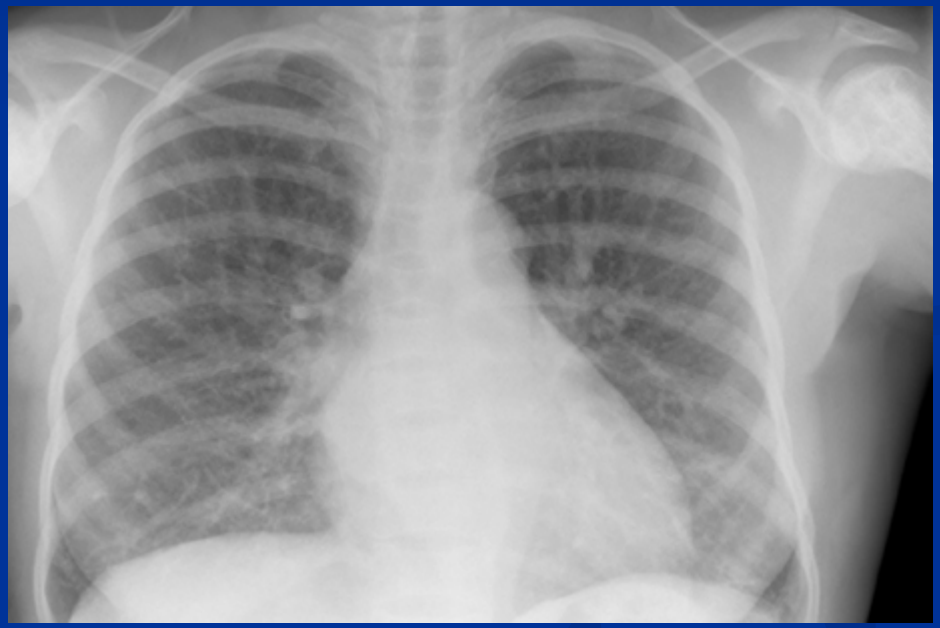
ST Elevated MI (STEMI)



3. CXR:

- Used to see if cardiac patients have an enlarged heart or fluid accumulating in the lungs

- Also useful to help differentiate whether SOB is related to Heart Failure or Pneumonia



4. Echocardiography (ECHO):

- Echocardiography is the use of ultrasound to visualize cardiac structures.

- This technique can assess the anatomy, motion and function of the cardiac valves and chambers non invasively, thus aid in the diagnosis of a variety of cardiac abnormalities.

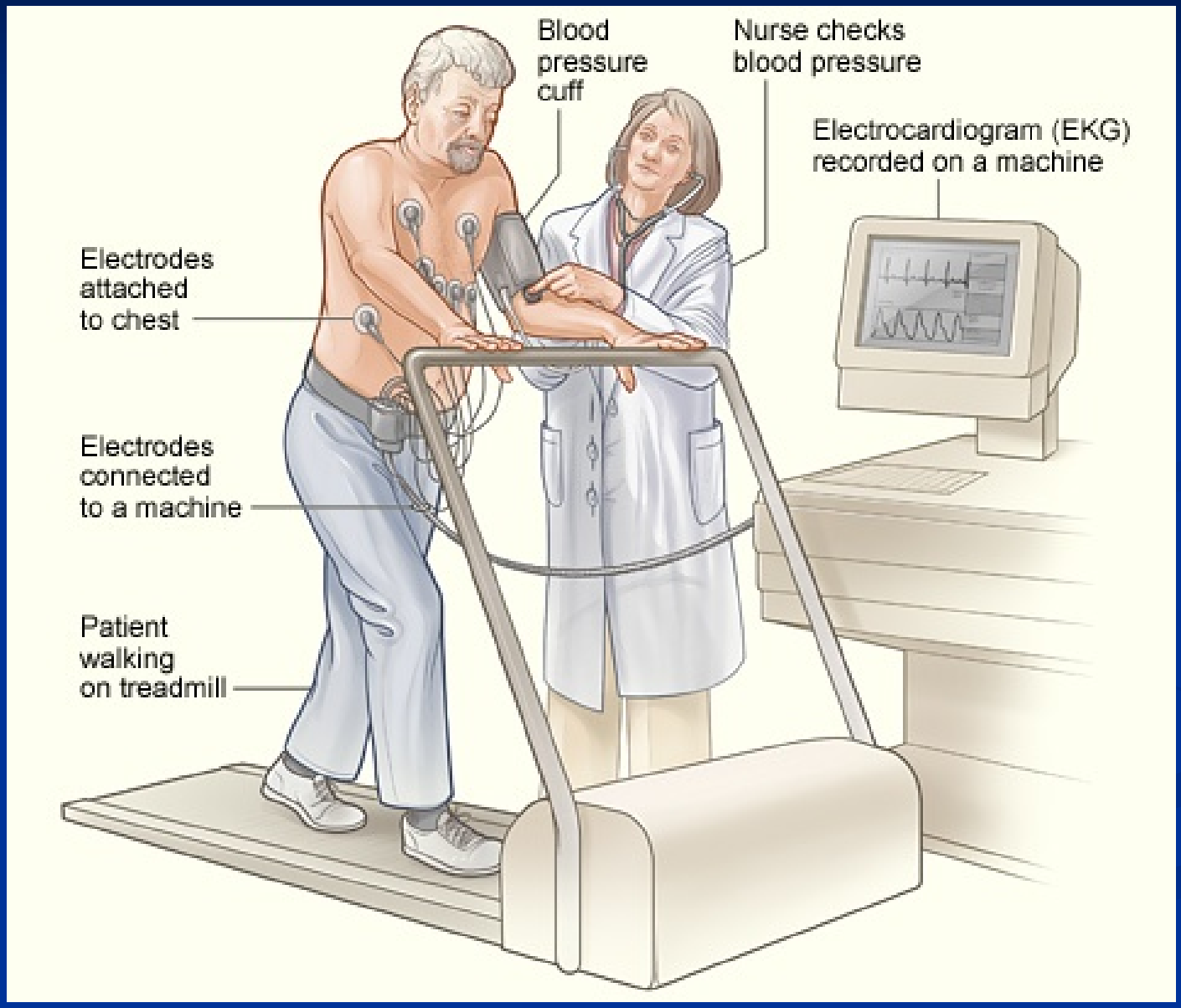
5. MPI

- **MPI (myocardial perusion imaging) Scan:**
  - Involves injection of thallium-201 & 2nd a radioactive isotope which attaches to RBC
  - useful to assess blood flow or perfusion
- **MPI involves stress component- either by exercise or drugs**
  - to induce ischemia if no ischemia at rest

MPI Prep

- No Beta Blockers, Calcium channel blockers or nitrates 24hrs before test- why?
  - Patient's heart rate and blood pressure needs to be elevated during the test, these medications would prevent it from elevating
- NPO in am- no diabetic meds to be given

5. Stress Test:



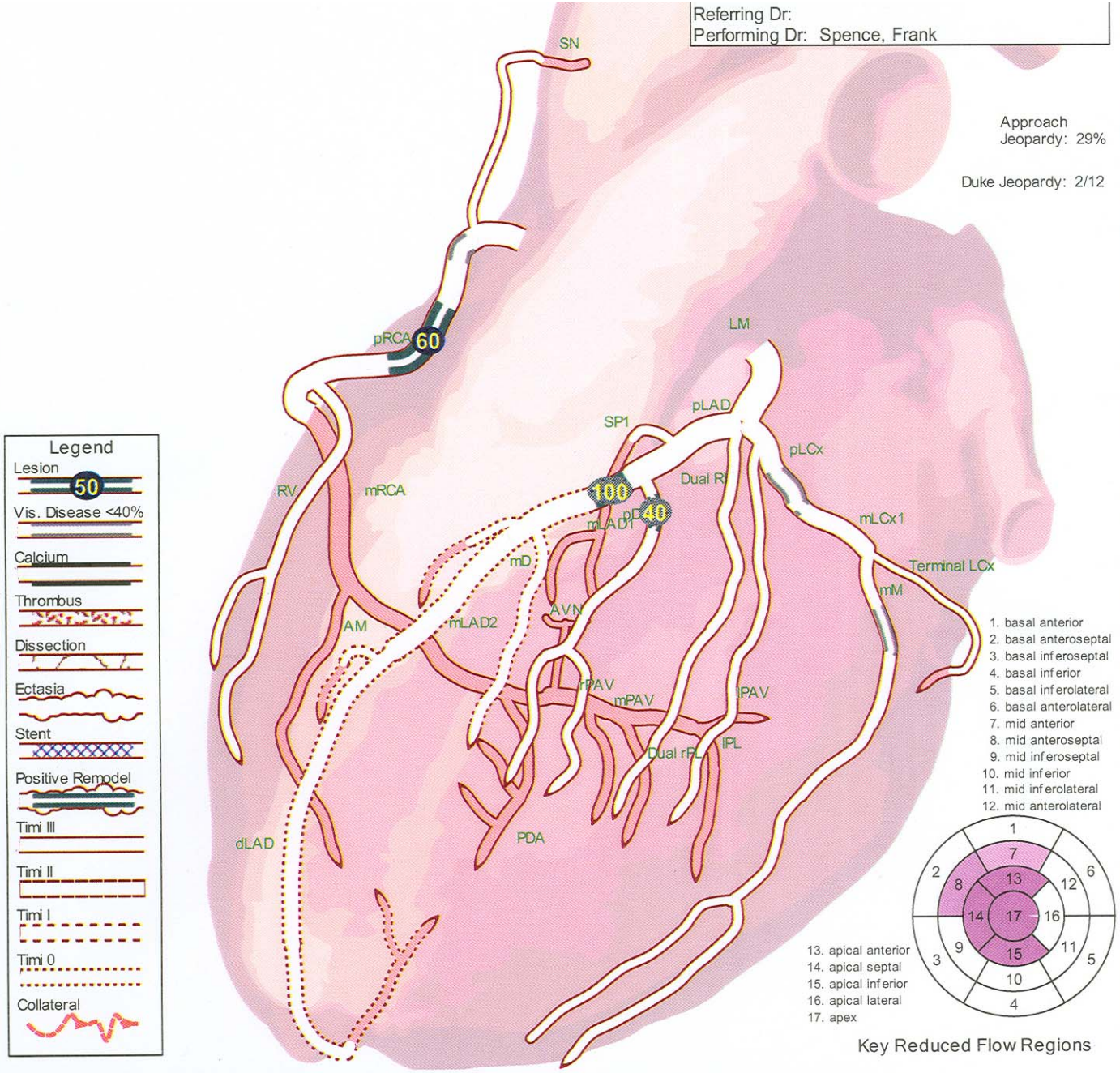
- Pass/Fail
- If patient develops chest pain, extreme SOB or has ECG changes may indicate the need for further cardiac testing

6. Coronary Angiography:

- **Angiogram**
  - invasive procedure, visualizes the chambers, valves and coronary arteries
  - catheter inserted via the arterial system then dye is injected
    - The right femoral or Radial artery are the most commonly used artery but the left femoral artery can also be accessed
- **PCI**
  - interventional procedure (dilation, stents)
  - balloon angioplasty

Angiogram Prep

- Hold anticoagulants- high risk of bleeding during and after procedure as we are accessing the femoral or radial artery



Culprit Reasons: mLAD1: Concordant ST elevation, RWM - LV gram, Lesion morphology, Lesion severity

Comments: Anterior STEMI. LV-anterolateral, apical, inferoapical, & septal akinesis, EDP=24mmHg. CAD as per CARAT diagram - culprit occluded LAD. Rec: Immediate LAD PCI.

Acute Coronary Syndrome-GOALS OF TREATMENT

- RESTORE Coronary Blood Flow
  - In the infarct related artery as early as possible
- REDUCE Size of Infarct
  - By dissolving newly formed clot before Necrosis occurs

Time is Muscle

Goal:

Door to drug within 30 minutes!

Immediate Interventions

- Oxygen, IV access
- Thorough physical assessment
- Vital signs
- ECG
- Targeted history and review of risk factors
- Cardiac markers (Troponin T)

"MONA greets all patients"
(morphine, oxygen, nitro, aspirin)

ACS Pharmacological Management FIBRINOLYTIC THERAPY

- Clot busting enzyme
  - Converts plasminogen to plasmin: breaks down fibrin thereby limiting myocardial injury

- CONSIDERATIONS
  - Tenectaplase (rTNK)
  - Administered as IV bolus dose
  - Systemic clotting effect is prolonged; avoid invasive procedures
  - Adverse effects: significant bleeding risk, CVA risk especially elderly women

Emergent Percutaneous Coronary Intervention (PCI) or Coronary Artery Bypass Grafting (CABG)

Indicated for:

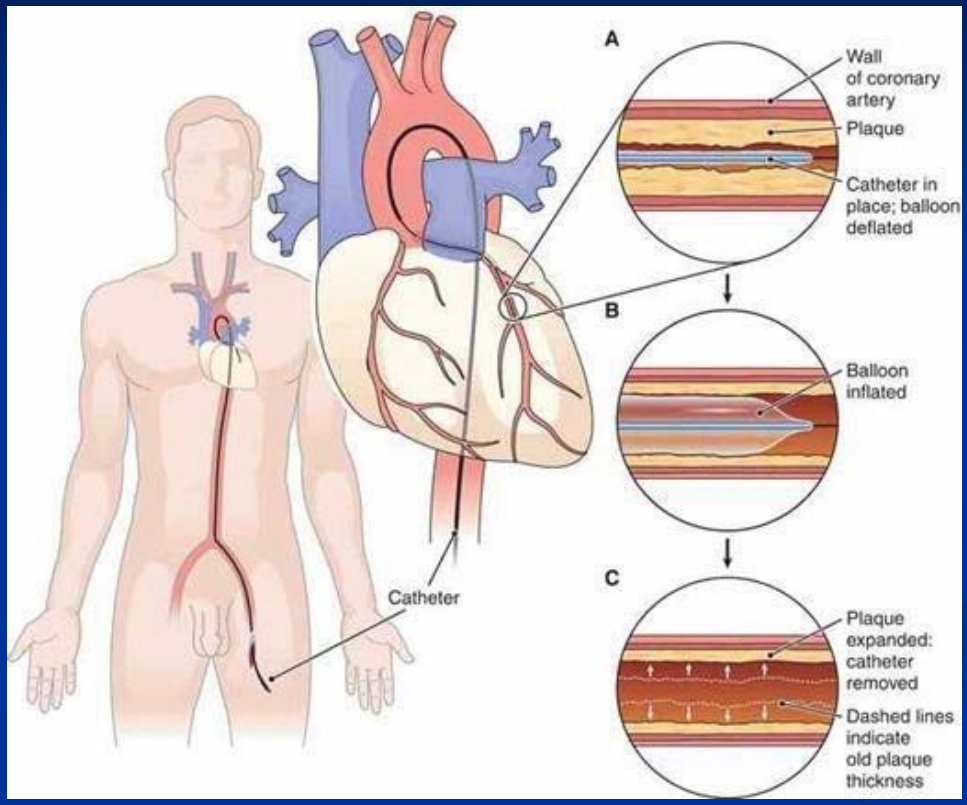
- Hemodynamic instability upon presentation
- Cardiogenic shock
- Malignant dysrhythmias

Goal: < 90 min from door to balloon inflation

PERCUTANEOUS CORONARY INTERVENTION (PCI)

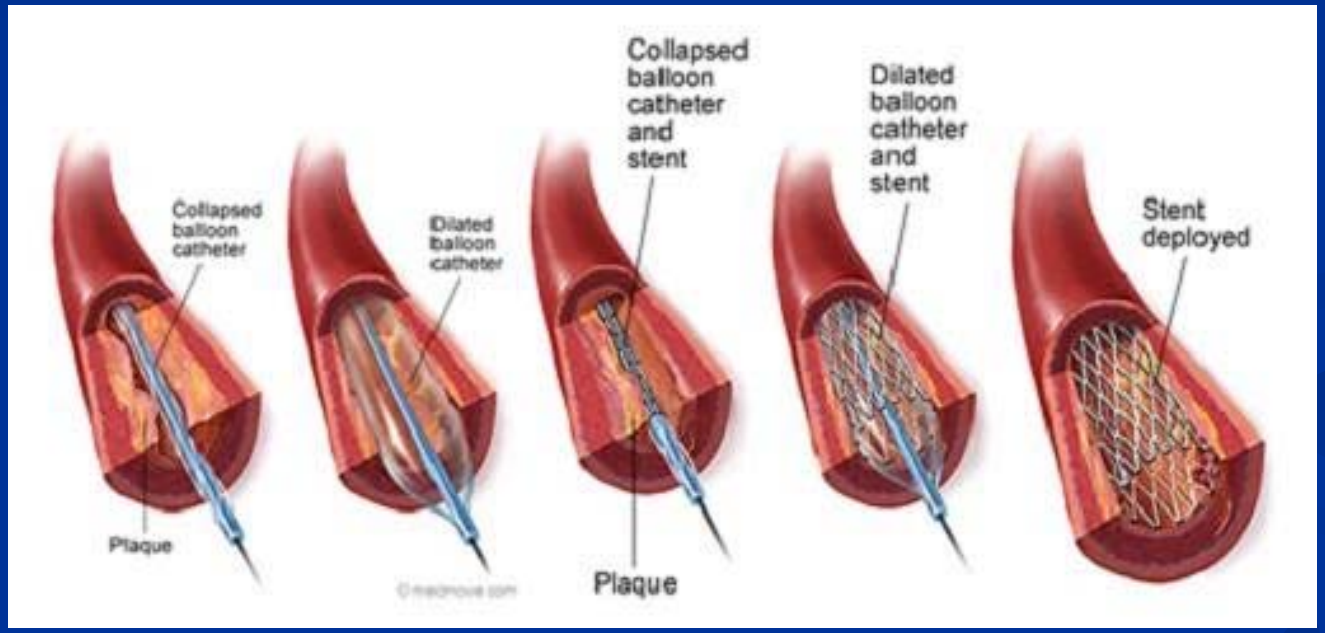
- Mechanical widening of narrowed/obstructed blood vessel
- Radiology guided procedure
  - BALLOON CATHETER inflated at point of critical lesion; crushes fatty deposit reopening blood vessel
  - Frequently stent placed to maintain vessel patency
- PRIMARY management for MI
- When PCI not feasible less than 90 minutes, Fibrinolysis should b administered if no contraindications

PCI



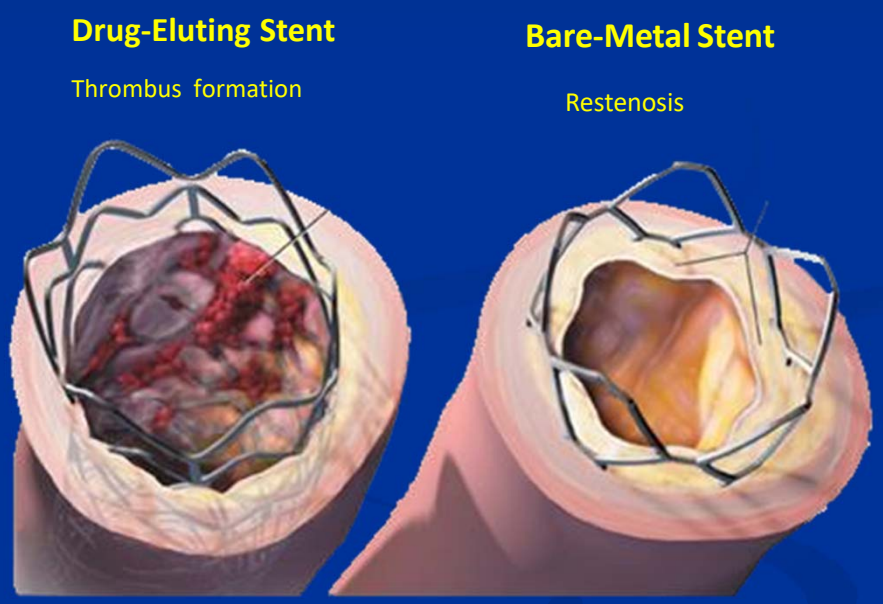
Lippincott advisor. (2018). Retrieved from http://advisor.lww.com

Stent placement



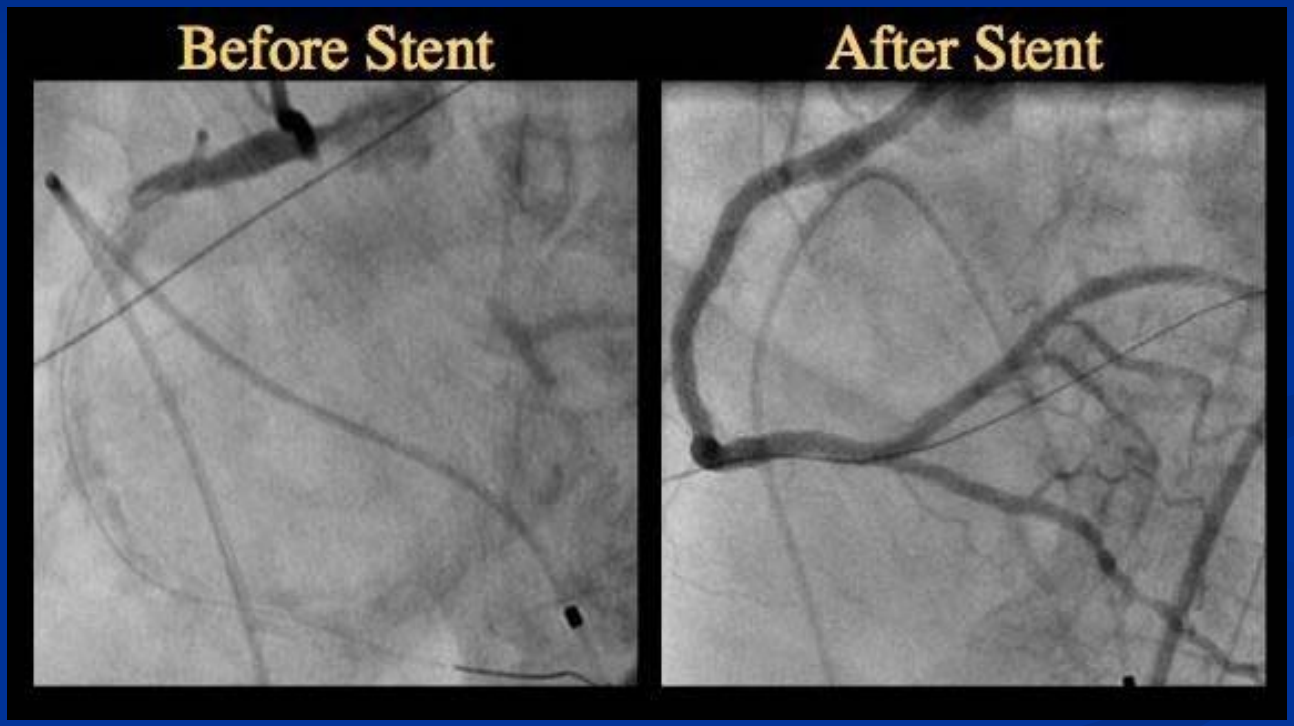
Antiplatelet Therapy- Drug Eluting Stents

- Drug-eluting stents release drug to reduce risk of restenosis NOT thrombosis
- Drug-eluting stents are much slower to be covered by endothelium than bare-metal stents
- Exposed metal of drug-eluting stent remains more thrombogenic for a longer period
- Antiplatelet therapy is more critical for a longer period to prevent stent thrombosis



Adapted from: Shuchman M. N Engl J Med. 2007;356:325–328.

Pre and POST Stent



Lippincott advisor. (2018). Retrieved from http://advisor.lww.com

17

Anticipated Medications

- ASA
- Clopidogrel or ticagrelor
- Heparin or LMWH, Fondaparinux
- Nitrates
- Beta Blockers
- ACE inhibitors or ARB
- Lipid lowering agents

- Cardiologist may follow-up with stress test (with follow-up angiography if indicated) and discuss risk factor modification

ASA

- In secondary prevention of CVD after acute myocardial infarction (MI), occlusive stroke, transient ischemic attack (TIA), stable angina, and coronary artery bypass surgery to reduce risks of MI, stroke and vascular death.
- In primary prevention of a first cardiovascular event as an individual clinical judgment for apparently health men and women at moderate to high risk.

Platelet P2Y12 Receptor Blockers: Clopidogrel or Ticagrelor

- CURE trial: combination therapy led to a significant reduction in the combined primary endpoint of cardiovascular death, nonfatal myocardial infarction (MI), or stroke
- Plato trial: randomly assigned to either ticagrelor (180 mg loading dose followed by 90 mg twice daily) or clopidogrel (300 to 600 mg loading dose followed by 75 mg daily) primary end point (first event of death from vascular causes, MI, or stroke) occurred significantly less often in patients receiving ticagrelor

Anticoagulant therapy

- Rupture of an atherosclerotic plaque is the usual initiating event in an acute coronary syndrome (ACS).
- Thrombin activity at the site of plaque rupture may result in delayed or incomplete reperfusion of occluded vessels and contributes to reocclusion.
- The heparins, including unfractionated heparin (UFH) and the low molecular weight heparins (LMWH), are indirect thrombin inhibitors that complex with antithrombin
- One potential advantage of fondaparinux over LMWH or unfractionated heparin is that the risk for heparin-induced thrombocytopenia (HIT) is substantially lower , compare with UFH, a significant reduction in the primary endpoint of death or reinfarction.

Nitrates

- Dilatation of large coronary arteries and arterioles (>100 millimicrons in diameter), which may lead to increased perfusion of ischemic zones.
- Dilatation of the venous system with decreased preload, reduction in ventricular volume, and a fall in pulmonary capillary wedge pressure.
- This effect is useful in patients with pulmonary congestion.
- Systemic arterial dilatation, which decreases afterload, also occurs but to a lesser degree. These changes lower wall stress and oxygen consumption and can reverse a restrictive filling pattern.
- Reduction of infarct size in experimental animal studies.
- Termination of an episode of variant angina.
- Enhanced collateral blood flow.

beta blocker therapy reduces infarct size and early mortality

- Decreased oxygen demand due to the reductions in heart rate, blood pressure, and contractility, and the consequent relief of ischemic chest pain.
- Decreased risk of ventricular fibrillation as suggested by experimental studies demonstrating an increase in the ventricular fibrillation threshold and by clinical trials showing a relative risk reduction in sudden cardiac death
- Bradycardia prolongs diastole and therefore improves coronary diastolic perfusion and reduces after-depolarizations and triggered activity.
- Reduction in remodeling and improvement in left ventricular hemodynamic function
- Improved left ventricular diastolic function with a less restrictive filling pattern
- Slowing of the yearly rate of progression of coronary atherosclerosis in patients with and without MI.
- Inhibition of platelet aggregation and thromboxane synthesis

(ACE) inhibitors or (ARBs)

- decrease cardiovascular mortality in post-MI patients with systolic dysfunction
- improves the left ventricular ejection fraction

Statin(PROVE IT-TIMI 22 trial)

- **The median LDL-C achieved was significantly lower with atorvastatin**
- **The primary end point (all-cause mortality, MI, unstable angina requiring rehospitalization, revascularization more than 30 days after randomization, or stroke) was significantly reduced with atorvastatin.**
- **Subset analysis revealed a trend toward benefit from atorvastatin as early as 30 days after the initiation of therapy; the risk reduction was similar to that seen at later time periods.**

Complications of an Acute MI

1. Cardiogenic shock
   - Occurs in approx. 10 % of AMI patients
   - Associated with a mortality of >85%
   - Severity directly associated with amount of myocardium damaged
   - Presentation: pale, cool, clammy, may be mottled, may be confused/disoriented due to poor cerebral perfusion, poor hemodynamic profile (low BP, low CI, high SVRI)
   - Invasive monitoring and advanced care essential (e.g. inotropes, vasoactive meds, IABP)

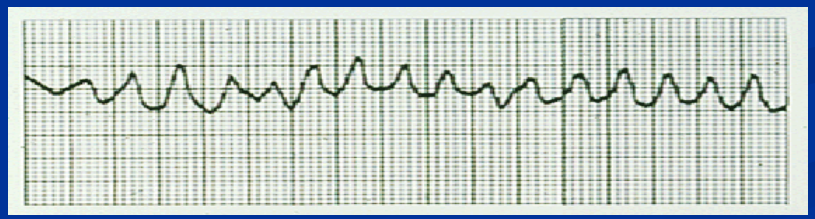
2. Ventricular Septal Defect (VSD)
   - Can be lethal
   - Loud murmur along right and left sternal border suggests rupture of interventricular septum (left to right shunt)
   - Diagnosis based on echocardiogram
3. Ventricular Aneurysm
   - Aneurysm clot formation as a result of turbulent blood flow in the aneurysm
   - Manifested by intractable ventricular dysrhythmias
   - Diagnosis based on echocardiogram
   - Ventricular remodeling

5. Papillary Muscle Dysfunction/ Rupture

- Rupture of the head of a papillary muscle causes a prominent murmur
- A new murmur must be reported to the physician immediately!
  - Can occur with ischemia of papillary muscle

6. Lethal Dysrhythmias

- Highest incidence in patients seen early following the onset of symptoms
- Particular dysrhythmia should be suspected based on the site of infarction



7. Heart Blocks

- Associated with area and size of infarct
- Blocks may progress- watch carefully

8. Pericarditis/ pericardial friction rub

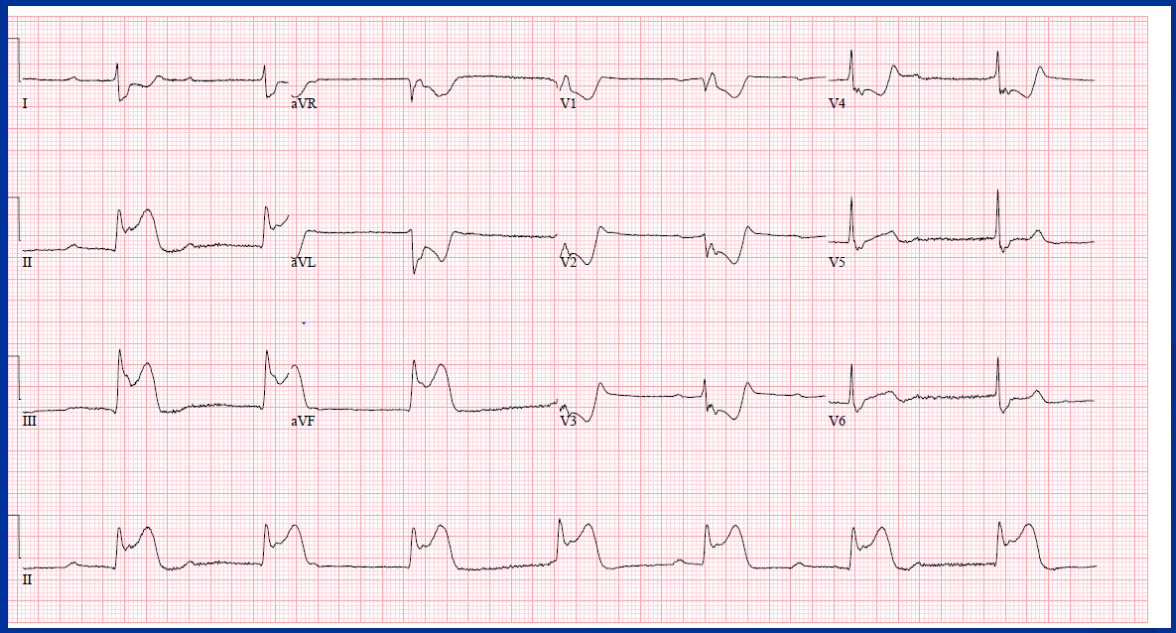
- Usually presents second or third day following AMI
- May have fever
- Pain with deep inspiration
- Pain worse when lying on left side
- Rub heard throughout the cardiac cycle; have patient lean forward in bed to appreciate sound.
- Pain is treated with NSAIDs in the absence of contraindications

Case Study

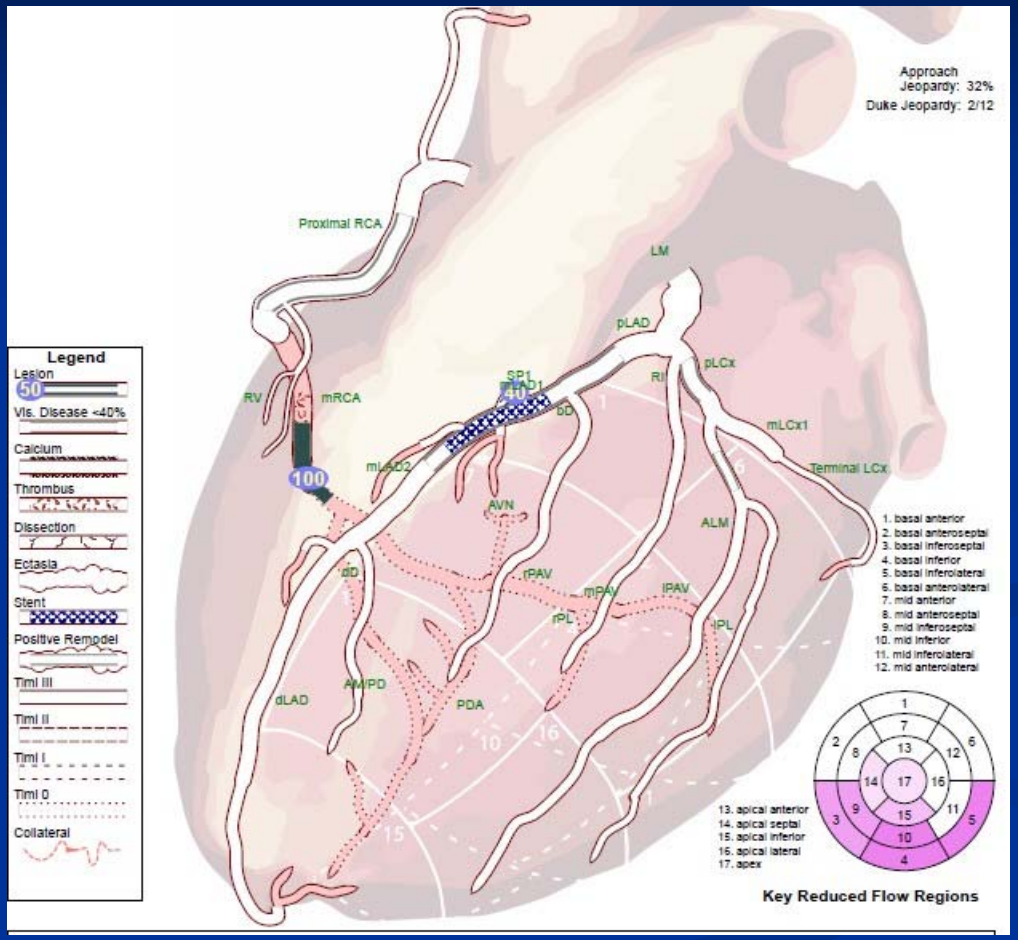
72 year old male with + history CAD with stent to LAD

- Presented to Cath lab direct via EMS. Inferior STEMI with 3rd degree heart block
- Gout
- hypertension

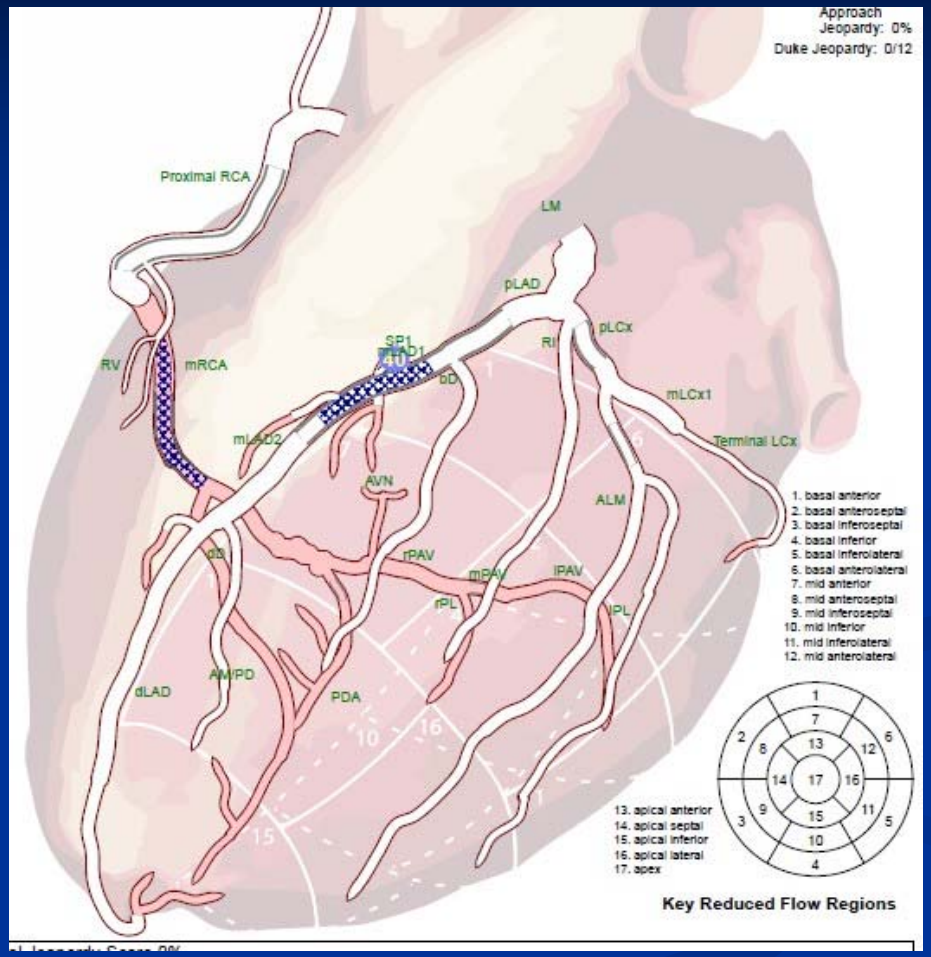
EMS
ECG 1024



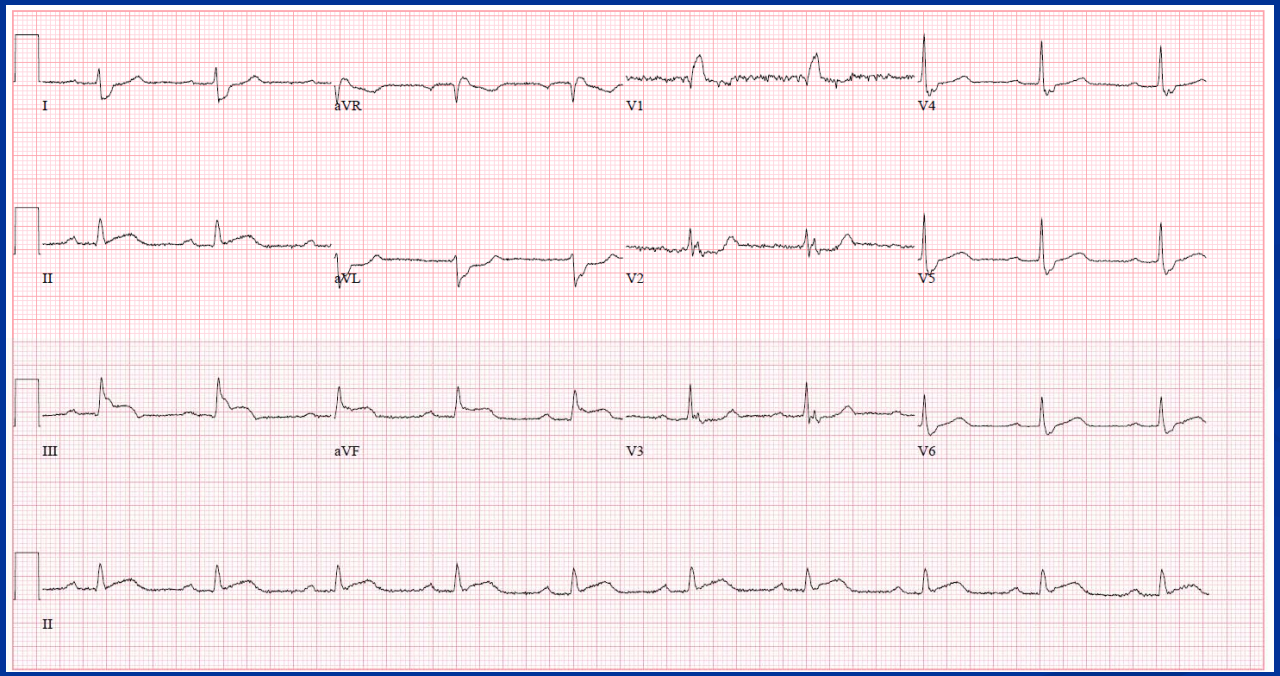
Cath


Approach Jeopardy: 32%
Duke Jeopardy: 2/12
Proximal RCA
LM
pLAD
pLCx
RI
SP1
D1
40
50
RV
mRCA
100
mLAD2
bD
mLCx1
Terminal LCx
AVN
ALM
dD
rPAV
mPAV
lPAV
rPL
lPL
dLAD
AM
PD
PDA
Legend
Lesion
Vis. Disease <40%
Calcium
Thrombus
Dissection
Ectasia
Stent
Positive Remodel
Timi III
Timi II
Timi I
Timi 0
Collateral
1. basal anterior
2. basal anteroseptal
3. basal inferoseptal
4. basal inferior
5. basal inferolateral
6. basal anterolateral
7. mid anterior
8. mid anteroseptal
9. mid inferoseptal
10. mid inferior
11. mid inferolateral
12. mid anterolateral
13. apical anterior
14. apical septal
15. apical inferior
16. apical lateral
17. apex
Key Reduced Flow Regions

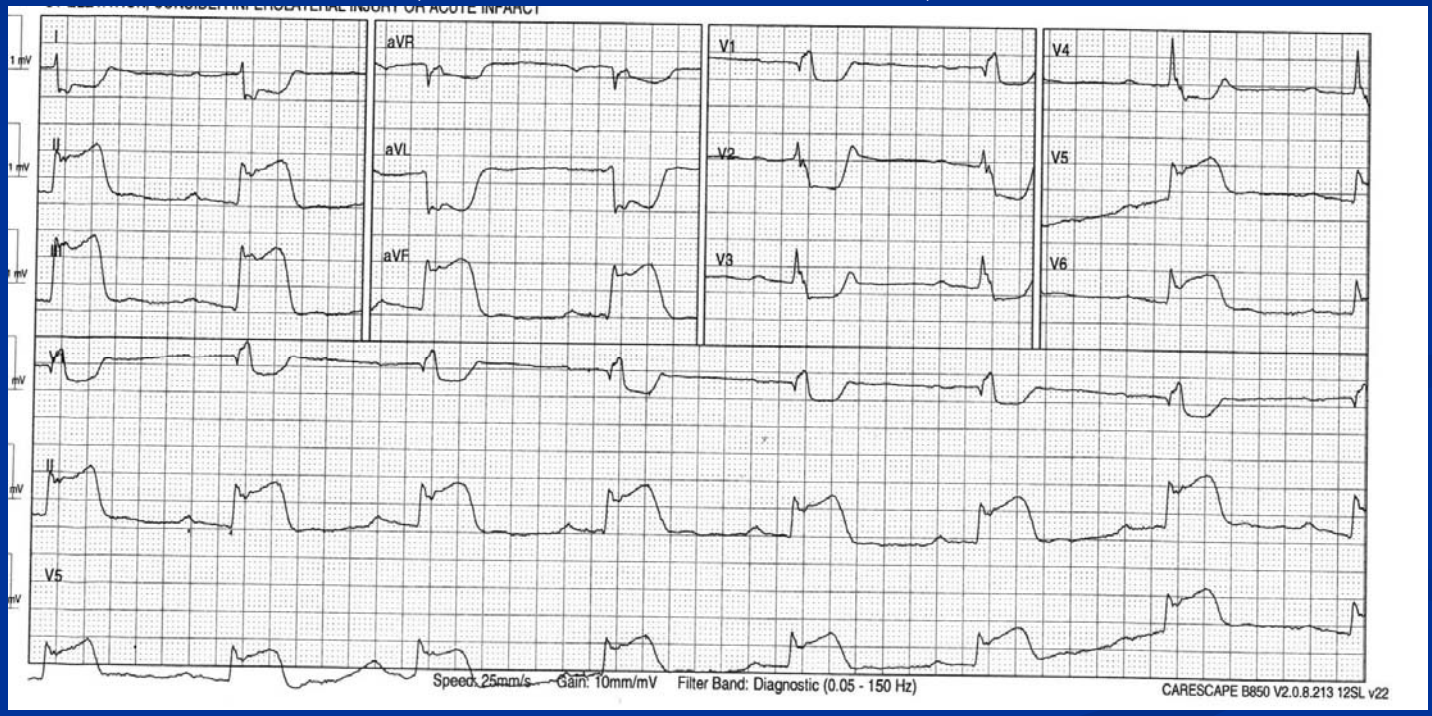
PCI


Approach
Jeopardy: 0%
Duke Jeopardy: 0/12
Proximal RCA
LM
pLAD
pLCx
RI
RV
mRCA
SP1
AD1
pD
mLCx1
mLAD2
Terminal LCx
AVN
ALM
pD
rPAV
mPAV
lPAV
rPL
lPL
dLAD
AM/PD
PDA
1. basal anterior
2. basal anteroseptal
3. basal inferoseptal
4. basal inferior
5. basal inferolateral
6. basal anterolateral
7. mid anterior
8. mid anteroseptal
9. mid inferoseptal
10. mid inferior
11. mid inferolateral
12. mid anterolateral
13. apical anterior
14. apical septal
15. apical inferior
16. apical lateral
17. apex
Key Reduced Flow Regions

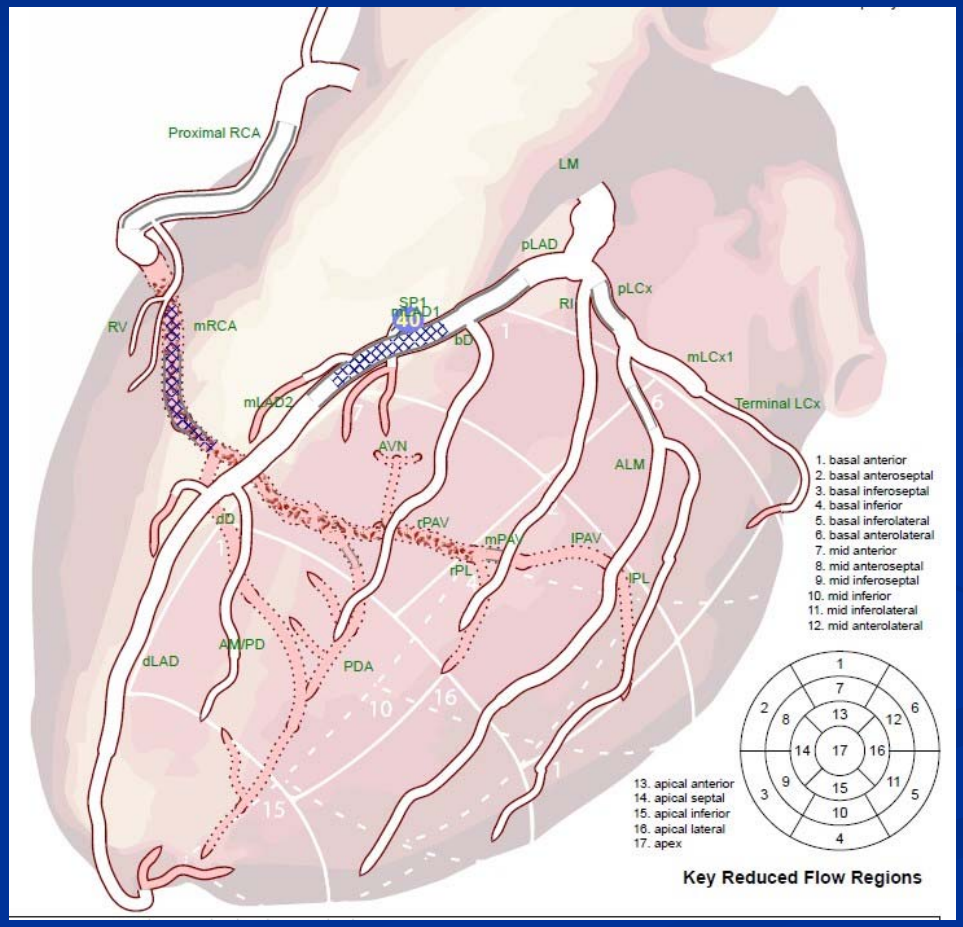
1200 (post procedure ECG)



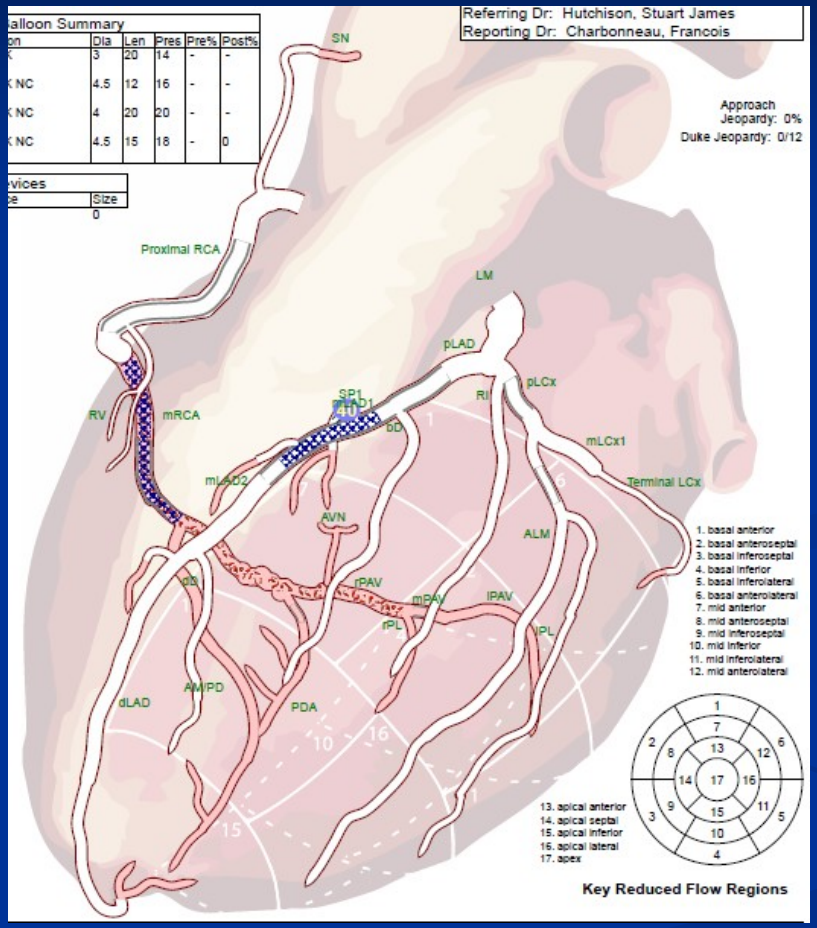
1229 (ST alarms)



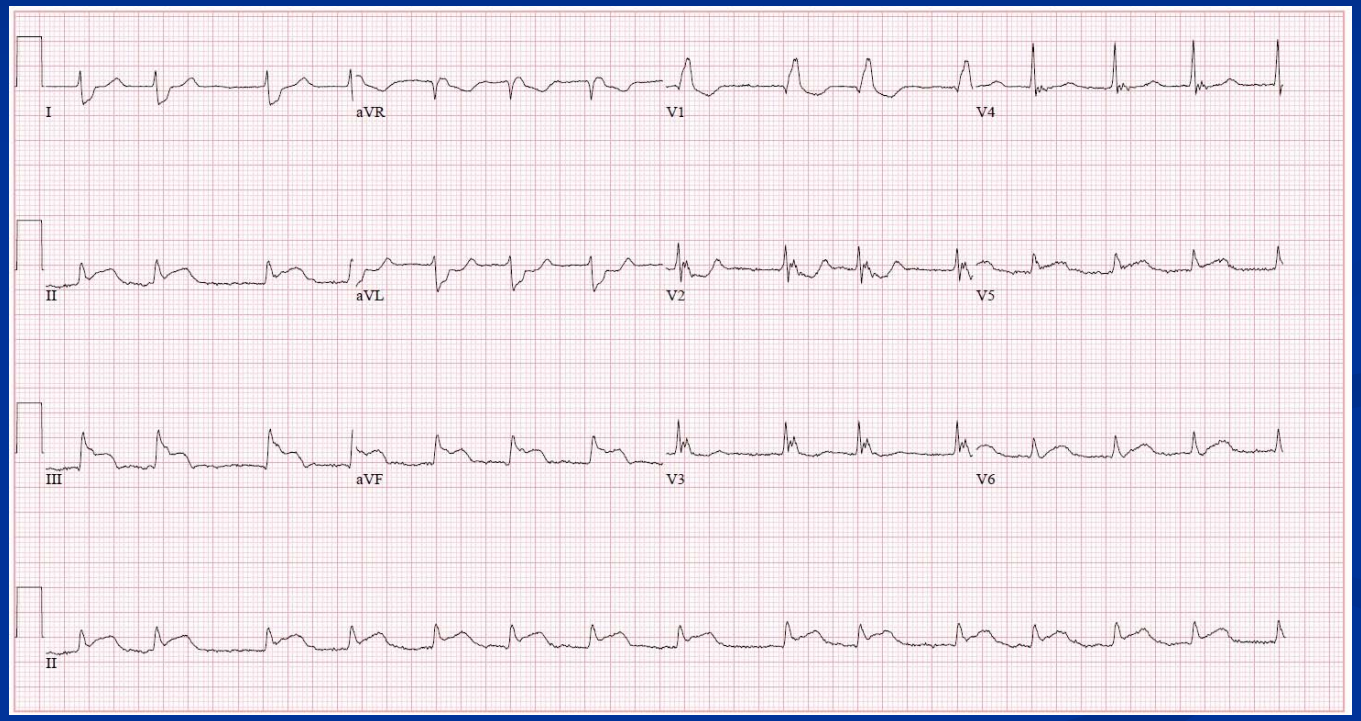
Back to the Cath lab STAT



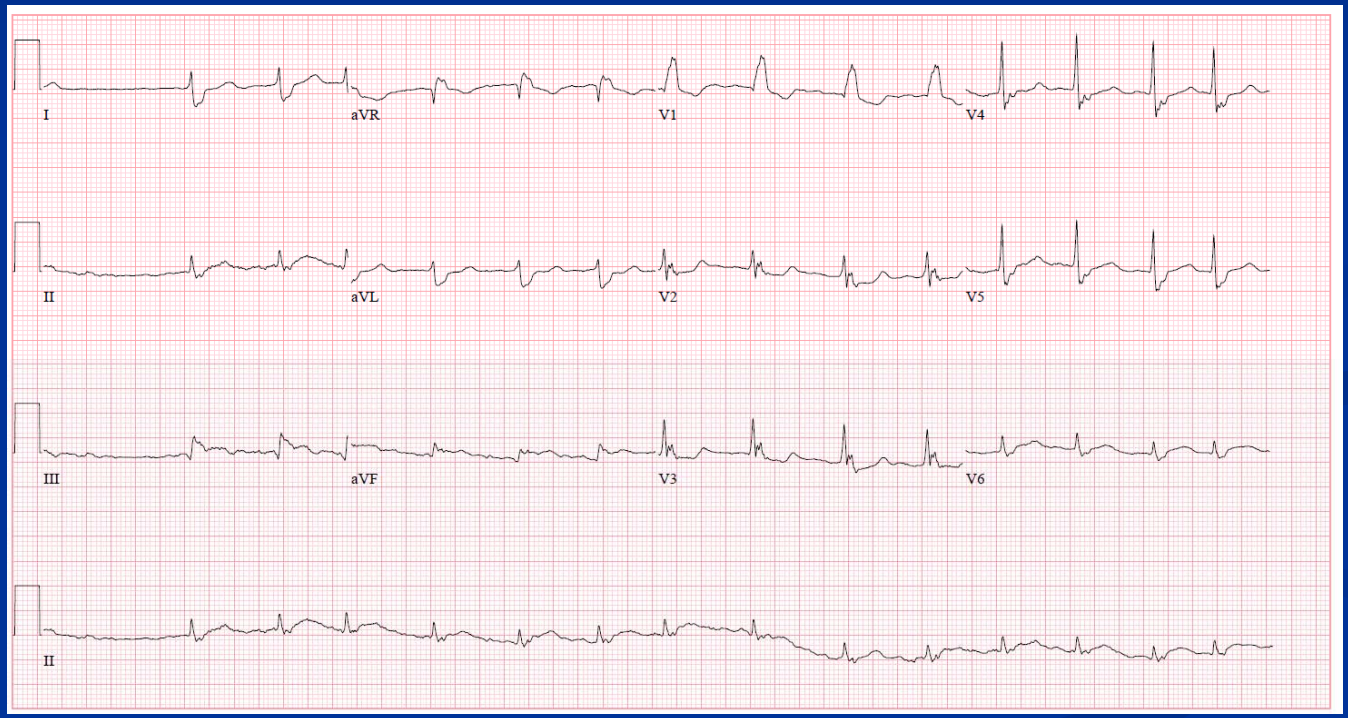
2nd PCI


alloon Summary
on | Dia | Len | Pres | Pre% | Post%
3 | 20 | 14 | - | -
NC | 4.5 | 12 | 16 | - | -
NC | 4 | 20 | 20 | - | -
NC | 4.5 | 15 | 18 | - | 0
vices
e | Size
0
Referring Dr: Hutchison, Stuart James
Reporting Dr: Charbonneau, Francois
Approach Jeopardy: 0%
Duke Jeopardy: 0/12
SN
Proximal RCA
LM
pLAD
pLCx
RI
SP1
AD1
RV
mRCA
bD
mLCx1
mLAD2
Terminal LCx
AVN
ALM
pD
rPAV
mPAV
lPAV
rPL
lPL
dLAD
AM/PD
PDA
1. basal anterior
2. basal anteroseptal
3. basal inferoseptal
4. basal inferior
5. basal inferolateral
6. basal anterolateral
7. mid anterior
8. mid anteroseptal
9. mid inferoseptal
10. mid inferior
11. mid inferolateral
12. mid anterolateral
13. apical anterior
14. apical septal
15. apical inferior
16. apical lateral
17. apex
Key Reduced Flow Regions

1418 (post procedure ECG)



2344 (8 hr ECG)



Next morning ECG 0622