Acute MI: Diagnosis to Discharge

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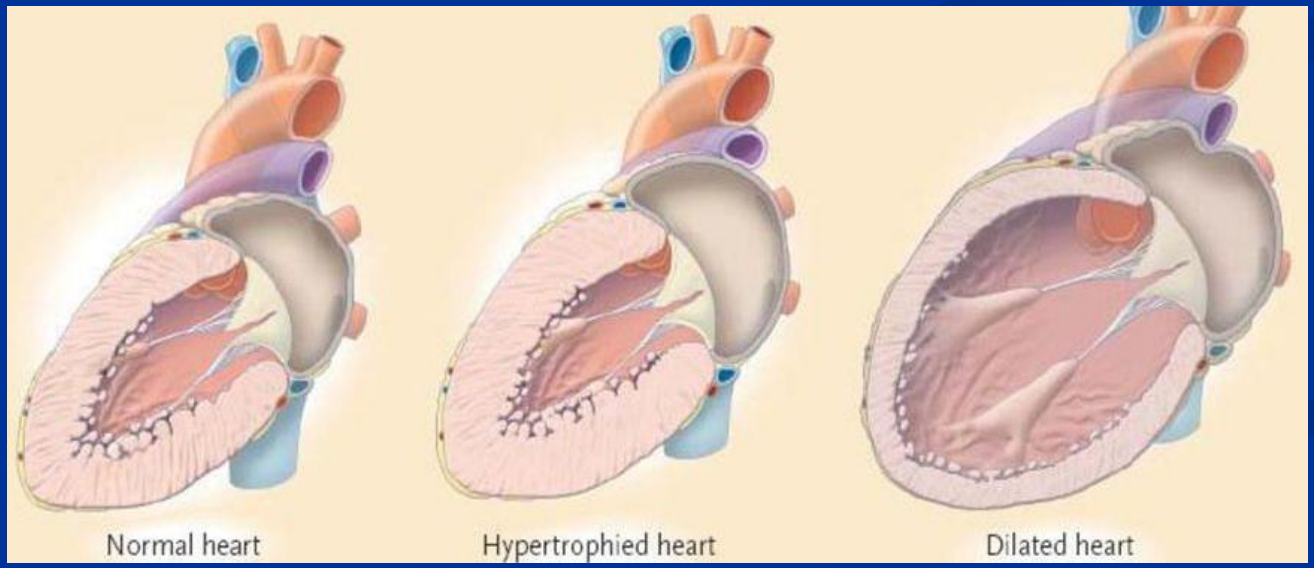
Objectives

- Discuss the need for immediate treatment (STEMI) versus non-emergent treatment (nstemi), including symptoms, lab values and EKG changes.
- Identify treatments such as PCI or surgery and discuss complications of MI based on area of infarct.
- Outline the medical treatment and follow-up care of MI patients needed at discharge.

Acute MI

- Occurs when one of the coronary arteries becomes blocked causing damage to the myocardium
- Extensive myocardial remodeling with fibrous tissue in infarcted and non-infarcted myocardium, damaging tissue structure, increasing tissue stiffness leading to ventricular dysfunction



File: heart ventricular hypertrophy commons.wikimedia.org

AMI

- One of the leading causes of death in the developing world
- Affects 3 M people worldwide
- > 700,000 deaths in US annually
- One third die before arriving at a hospital
- 5 to 10% die within the first 12 months after MI
- Prognosis depends on the extent of myocardial damage*

AMI etiology

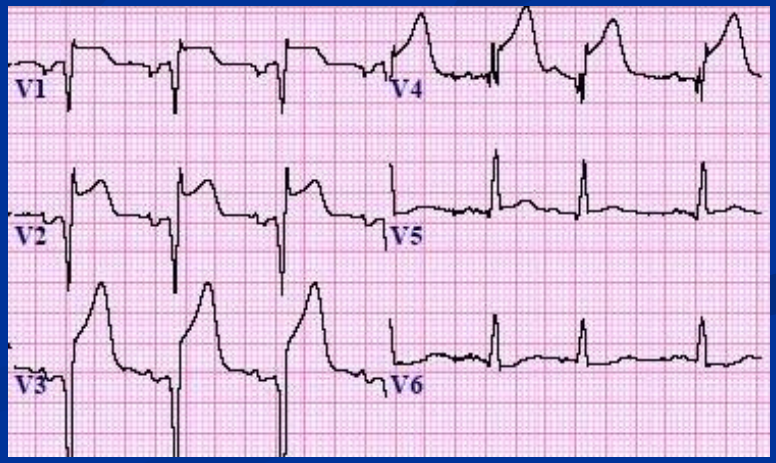
- Atherosclerotic plaque rupture
- Inflammatory response
- Thrombus formation
- Platelet aggregation
- Decreased O2 delivery through the coronary arteries
- Decreased O2 to the myocardium
- Ischemic cascade (sequence of events)
- Cell death to the myocardium
- MI

Risk factors for CAD

- Smoking
- HTN
- Dyslipidemia
- DM
- Obesity
- Presence of PVD
- Sedentary lifestyle
- Poor oral hygiene
- Non-modifiable – age, sex, family history

Acute Coronary Syndrome

- Unstable angina – similar to nstemi but troponins are not elevated, may have a "normal" EKG
- NSTEMI – no ST elevation on EKG but troponins are elevated, may have a "normal" EKG
- STEMI – ST segment elevation MI, initially diagnosed by EKG



Type II MIs

- Type II MI or demand-mediated MI
- Due to an oxygen supply/demand mismatch
- Most cases triggered by non-coronary causes; acute blood loss anemia, acute hypoxia, shock states
- Type 2A – with fixed obstructive CAD
- Type 2B – without fixed obstructive CAD

5 types of MI

- Type 1 related to atherosclerotic disease
- Type 2 secondary to oxygen supply/demand
- Type 3 sudden cardiac death likely secondary to ischemia
- Type 4 associated with PCI (4b r/t stent thrombosis)
- Type 5 associated with cardiac surgery

  Both type 4 & 5 have elevated troponins after procedure

Let's talk Troponins

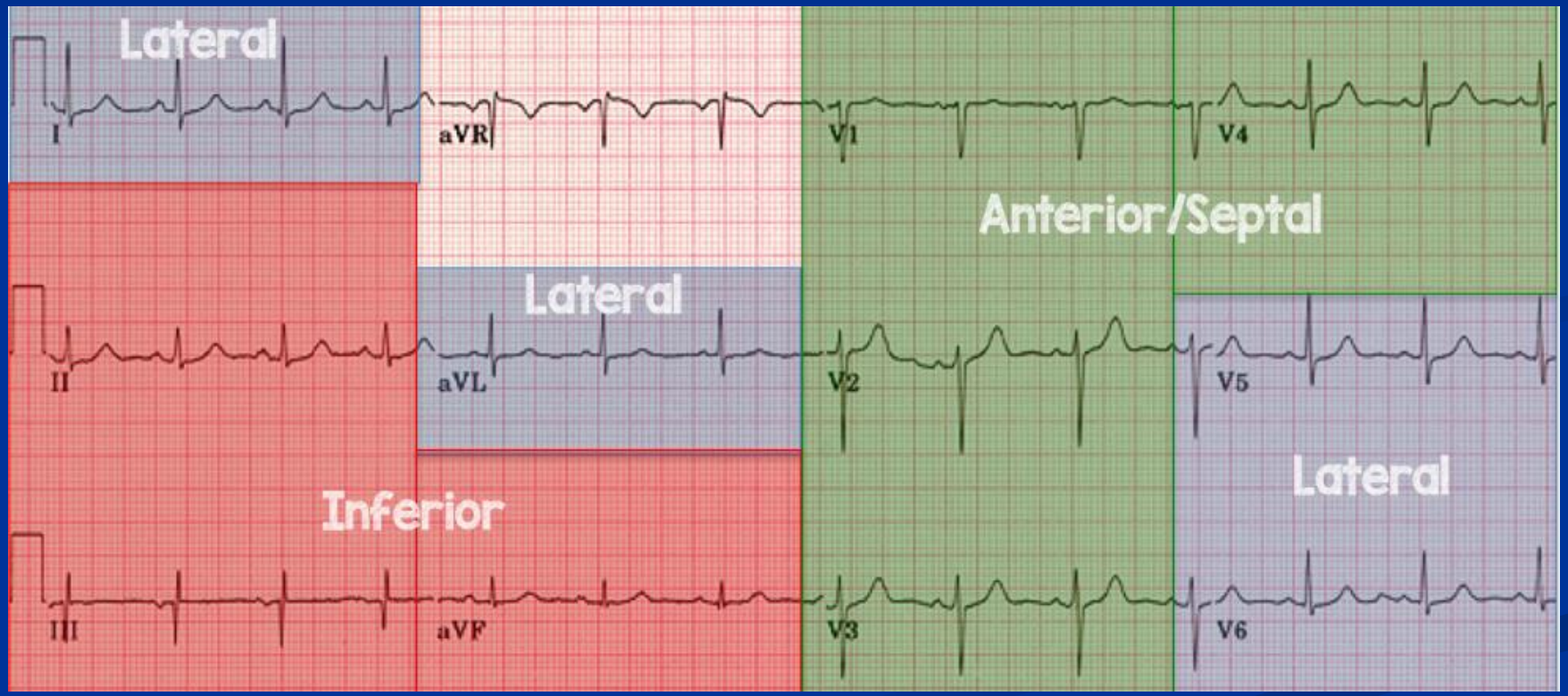
- A protein found in the body, specifically in heart muscle cells
- If the heart muscle is oxygen deprived, they start to die
- Troponin proteins in those cells spill into the bloodstream
- High-sensitivity cardiac troponin (hs-cTn) normal value is 14 ng/L.

  > 14 - heart damage or MI is very likely

Increased troponins other than MI

- CKD
- CHF
- Sepsis
- Pulmonary embolism
- Myocarditis
- Aortic dissection
- Blunt chest trauma
- Cardiotoxic chemotherapy

II, III, aVF -inferior
I, aVL -lateral
SAL



File: 12leadECG
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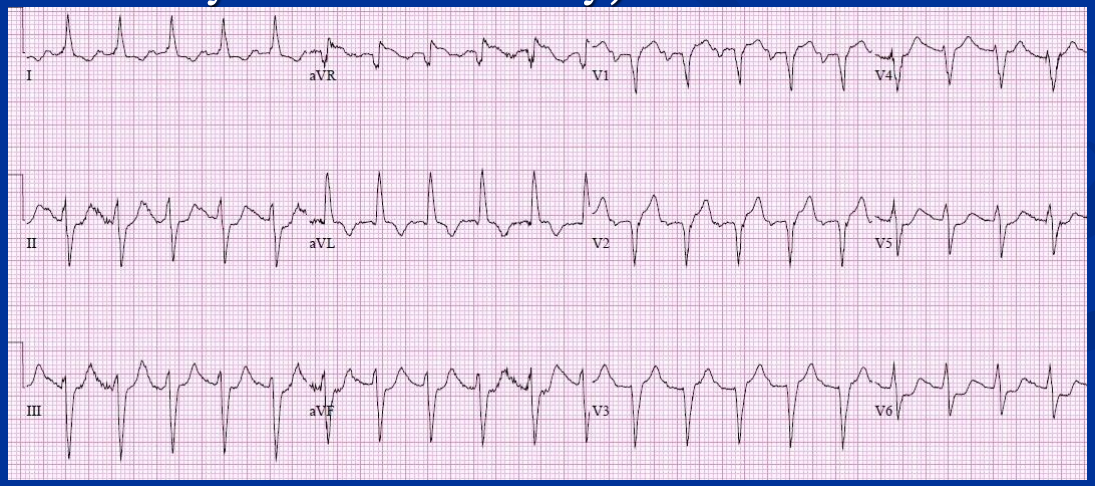
Lead aVR

- Initially thought to provide "indirect" information or information already seen in other leads
- In acute anterior MI, ST elevation in aVR predicts LAD occlusion proximal to the first septal perforator
- aVR ST segment elevation greater than the elevation in V1 can predict acute left main coronary artery occlusion (80% specificity & sensitivity)



MAJOR CORONARY ARTERIES

Left main artery

Left anterior descending (LAD)

-diagonal branches

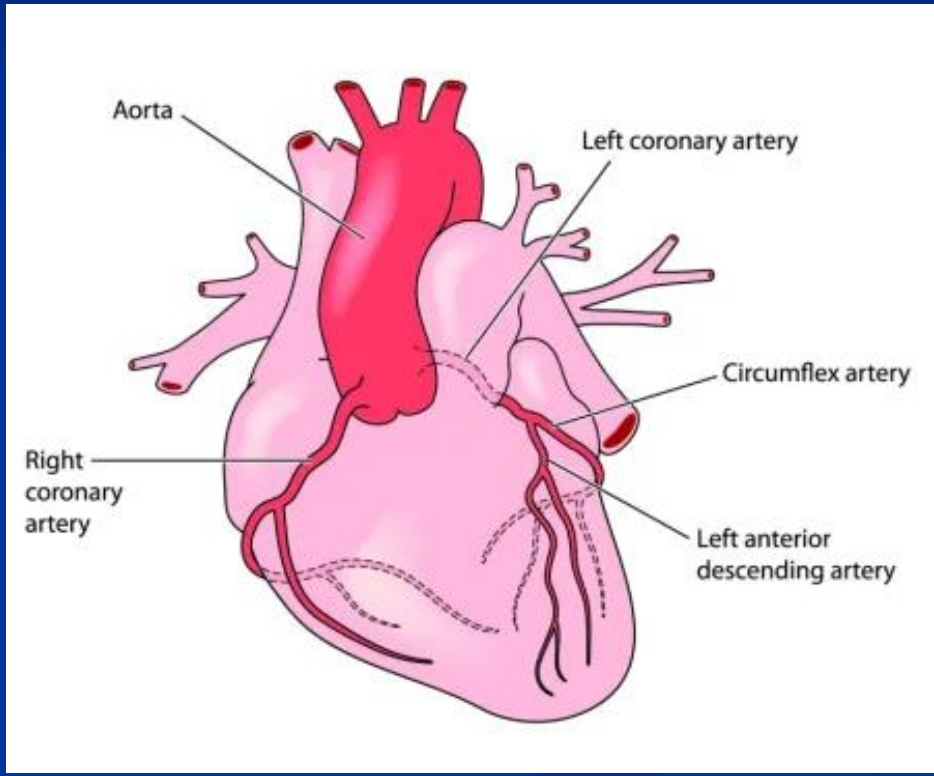
Circumflex (Cx)

-obtuse marginal

Right coronary artery (RCA)

-posterior descending (PDA)

-acute marginal



File: coronary arteries pt.svg
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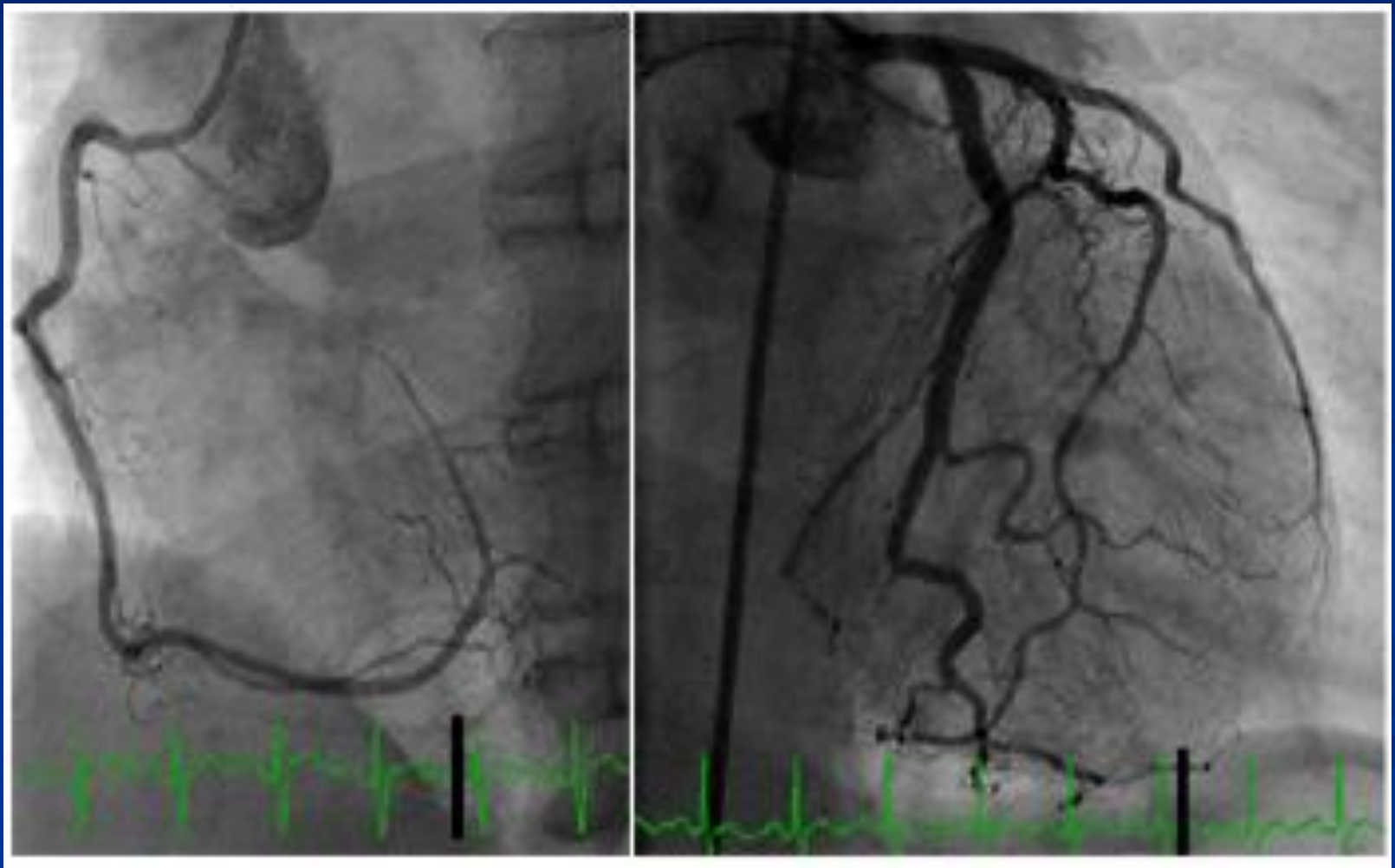
Coronary Arteries



File: Angiography coronary arteries
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What artery feeds what part of the myocardium

- LM – LV, LA (Also "feeds" the LAD & Cx)
- LAD – anterior wall
- Cx – lateral wall and back
- RCA – RV, RA, SA node, AV node

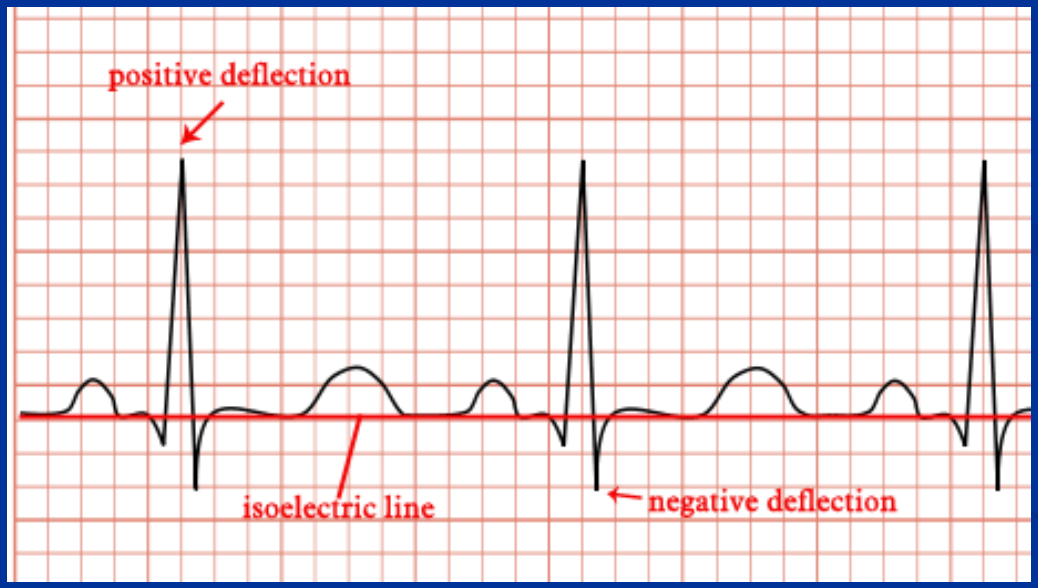
  Acute marginal - posterior & inferior walls, RV & LV

  LAD & RCA - septum

Complications depend on injury

- Anterior wall MI (V3, V4) –more myocardial damage, LV dysfunction, cardiogenic shock
- Inferior wall MI (II, III, aVF) –bradyarrhythmias, heart block, c-shock, RV infarction, <10% mortality
- Lateral wall MI (I, aVL, V5, V6) –usually seen with other infarctions. Isolated lateral MI's can be seen with occlusion of obtuse marginal or diagonal branch occlusions. LV free-wall rupture can occur

Remember Normal Sinus Rhythm (NSR)



File: ECG normal sinus rhythm
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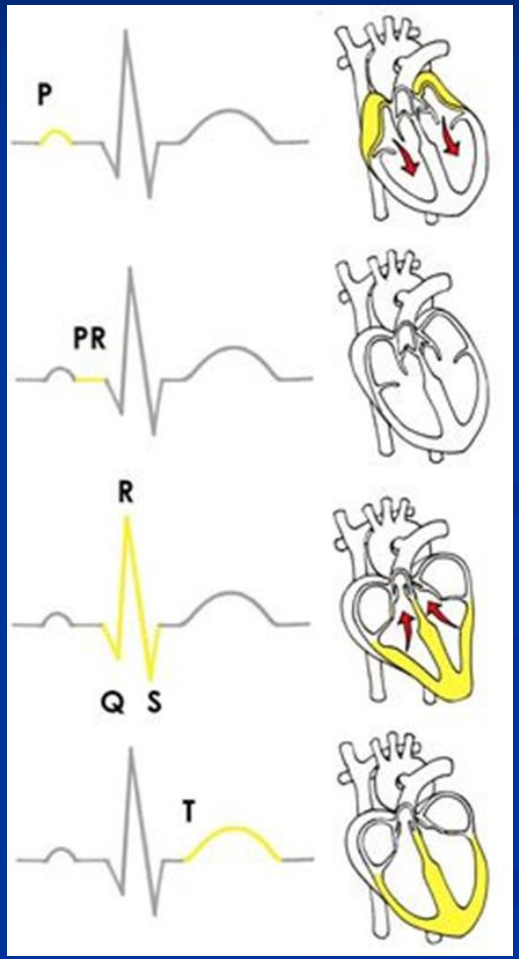
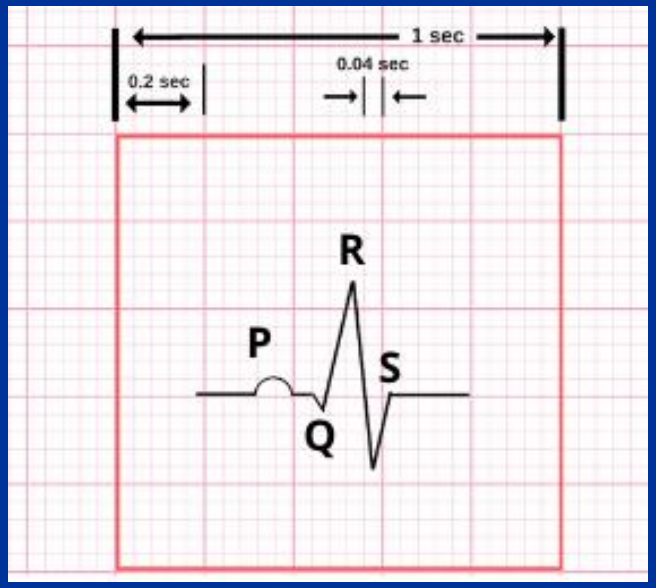
PQRST



Measurement
STEMI – 2 mm or > elevation



File: PQRST
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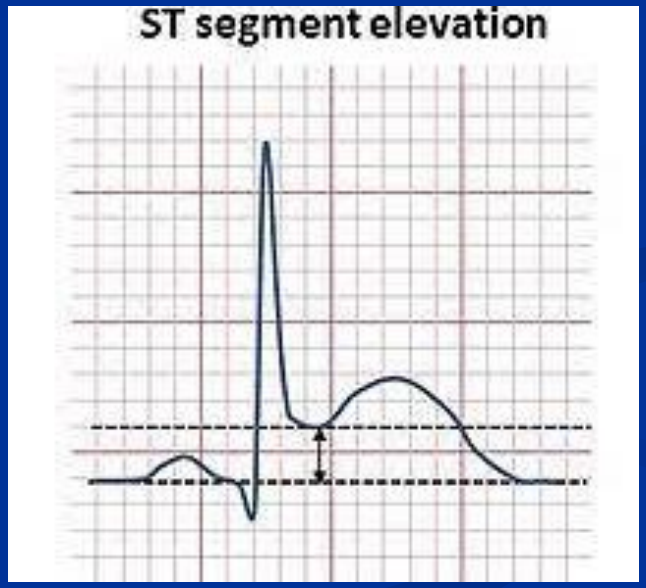

File: ecg st-elevation
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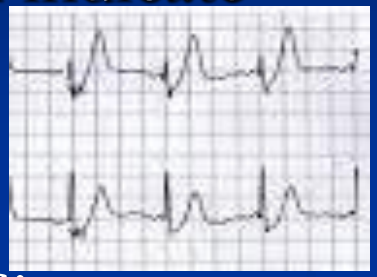
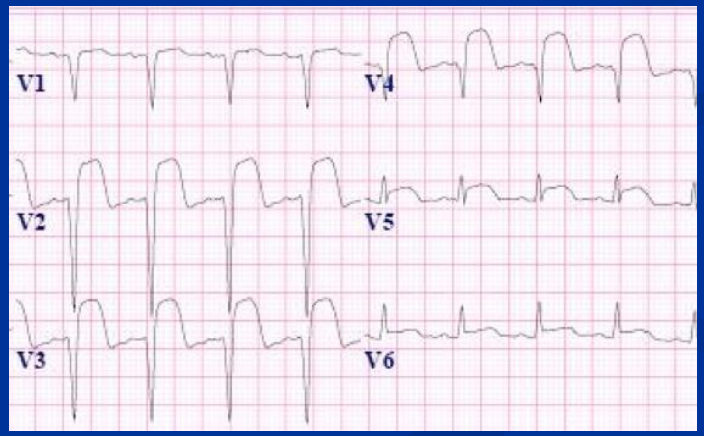
12-lead EKG

- Peaked T waves (hyperacute T waves) can indicate early ischemia, may progress to STEMI



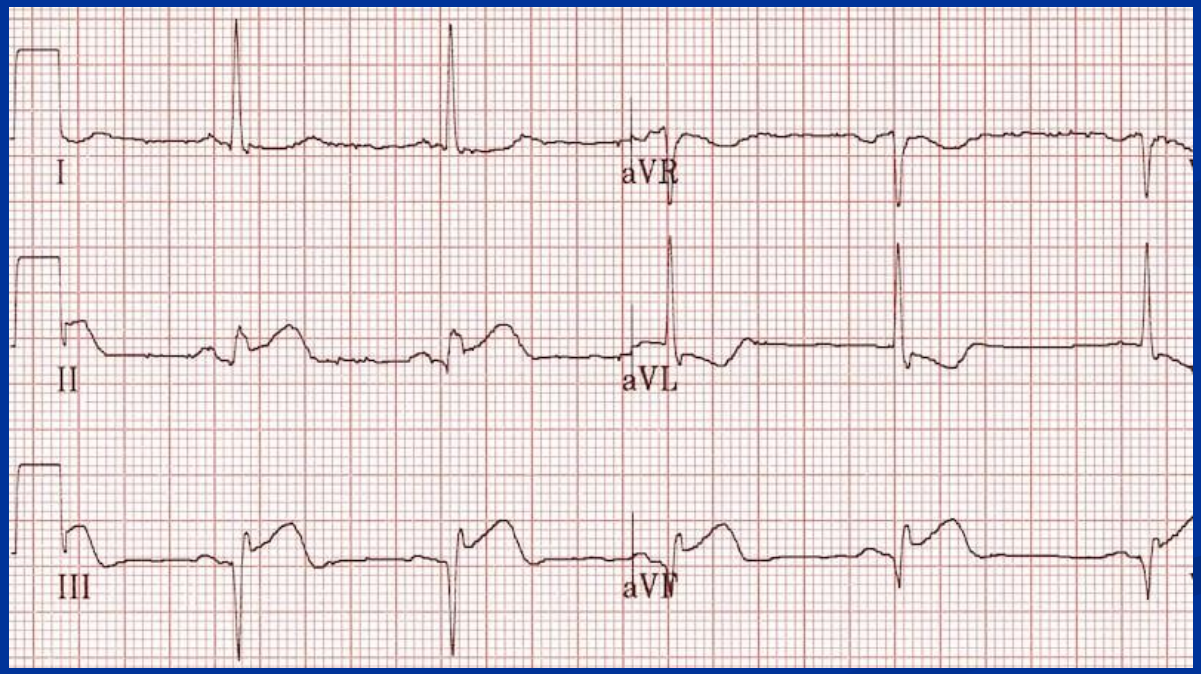
- ST elevation > 2mm in 2 contiguous leads:
  - Inferior II, III, AVF
  - Septal V1, V2
  - Anterior V3, V4
  - Lateral I, AVL, V5, V6



You will often see ST depression in leads in opposite anatomical regions of the myocardium (reciprocal changes)

12-lead EKG

- Q waves - Pathologic Q waves are a sign of **previous MI**, generally taking several hours to days to develop, usually permanent
- >1 mm wide, >2mm deep, >25% size of QRS complex



File: ECG inferior ST elevation
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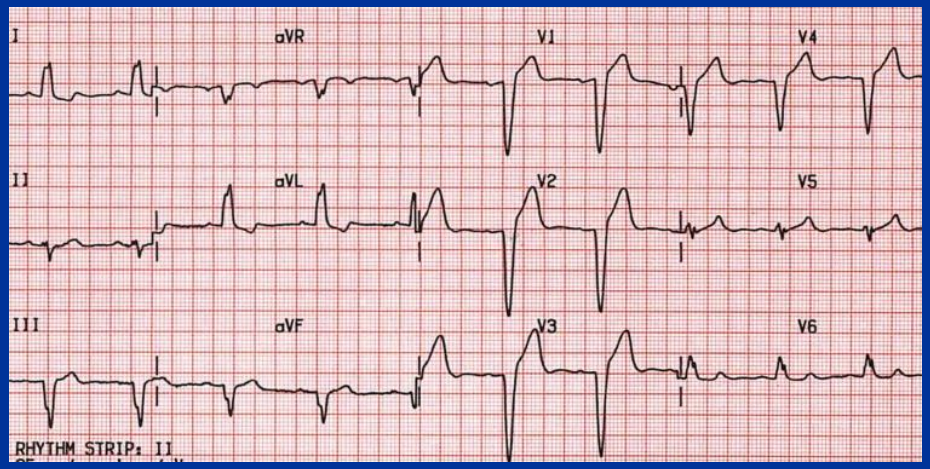
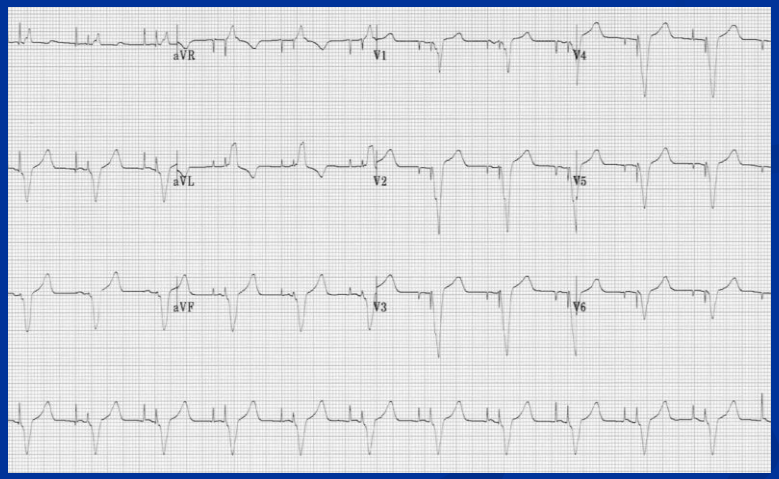
12-lead EKG

- ST segment elevation may be difficult to diagnose with:

LBBB



Pacemaker



Diagnosing CAD

- Stress test (nuclear) for patients (neg MI)

  -sensitivity of 81%, missing 19% of high-grade stenosis

- 2D echocardiogram (TTE) shows pumping function (LVEF), heart size, valve function, patterns of blood flow through the heart = wall motion.

  WMA – most often caused by ischemia

- Coronary angiogram (Left heart cath) – the Gold standard of testing for CAD, can include IVUS, FFR

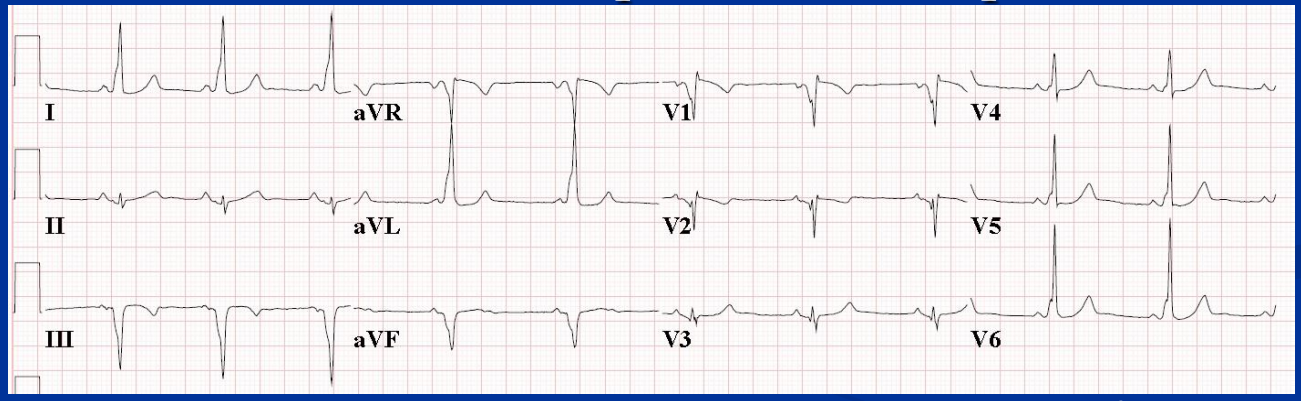
  Risks (<2%) injury to the catheterized artery, arrhythmias, allergic reactions to the dye, kidney damage from the dye

Diagnosing AMI

- Look and Listen
- EKG within 10 minutes of arrival, lab work; CBC, chemistry, troponins, CXR
- Compare EKG with prior EKG if possible
- P.E. - good history, medications
- ? Contraindications for blood thinners
- ? Contraindications for emergent angiogram

Case study 1

- Walter – 65 yo WM, hx HTN, DL, DM II, +fam hx,
- Hospitalized recently at outlying hospital for "mild CHF", hx "bad flu" 9 mos & 6 mos PTA, echo LVEF 25%, treated, sent home with LifeVest, meds – Lopressor, Lisinopril, Lasix



- Admitted after LifeVest fired x2, trops flat, BP 140/88, SR 90s occ. PVCs, CXR ok
- Cathed –significant multi-vessel CAD, EF <15%, CTS consulted for CABG

Case study 1

- Thallium myocardial viability scan showed almost no viability, CABG would provide no benefit.
- Treatment: optimization of GDMT, ICD, eval for BiV-ICD, possible evaluation for LVAD, transplant
- *VS on admit & during hospitalization – BP 140s/80s, SR 90s with PVCs – pt needs optimization of his meds *prior to discharge*; increased BB, change to EB BB (coreg, toprol XL, bisoprolol), increase ace or change to Entresto, add other meds as BP tolerates.

Case study 2

- Betty, 87 yo, hx HTN, DL, DM, O2-dependent COPD, s/p right total hip arthroplasty, being transferred to SAR
- Before discharge, c/o palpitations, nausea, weakness
- EKG shows Afib VR 100, NS ST-Ts, first trop 48, echo EF 40%, +WMA anteroseptal walls
- Diagnosed with type II nstemi
- Angio showed TVD, CTS consulted for CABG
- Betty refused CABG, PCI of the mid LAD was done, GDMT for CAD

MI Presentation:

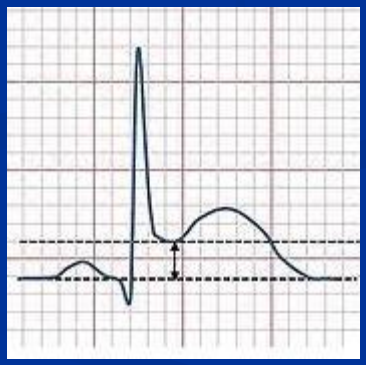
- Chest pain ("pressure", "discomfort", "tightness") which may or may not radiate to the neck, shoulder, teeth, jaw, back or abdomen

Diff dx: PE, pneumothorax, pericarditis, aortic dissection, myocarditis, acute cholecystitis

- Diabetics may not have pain (neuropathy)
- Dyspnea with or without cough
- Lightheadedness, dizziness, syncope
- Nausea, vomiting, GI complaints
- "I just don't feel right"

EKG Findings

- Ischemia – T wave inversion, peaked T waves, ST depression or completely "normal"
- Injury – ST elevation. The injury is happening NOW
- Where are the EKG Abnormalities?



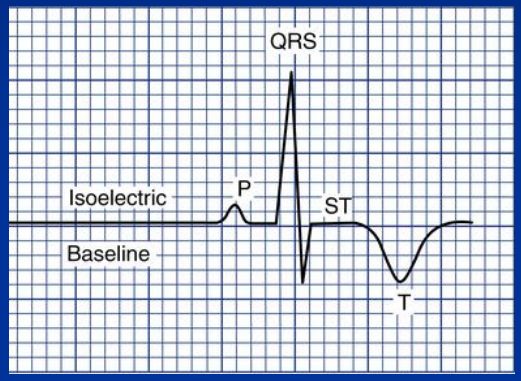
Leads I & AVL, V5 & V6 – Lateral wall

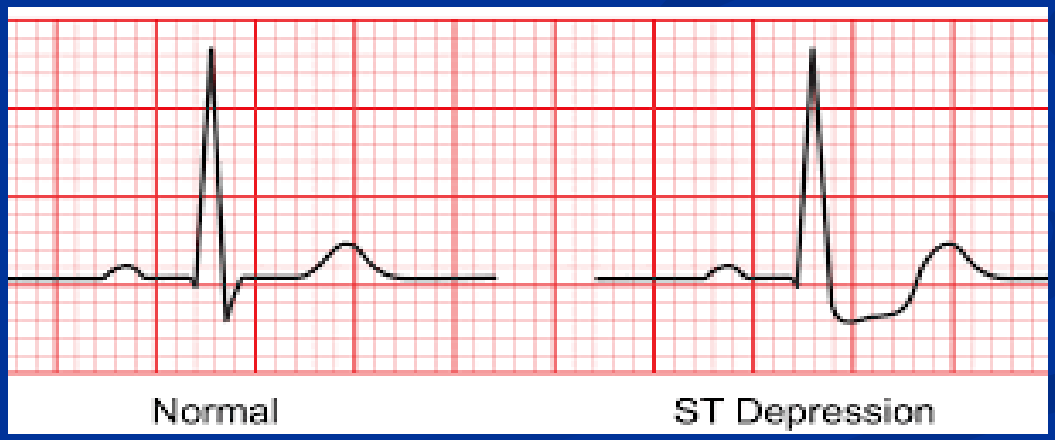
Leads II, III, AVF – Inferior wall

Leads V1, V2 – Septal wall

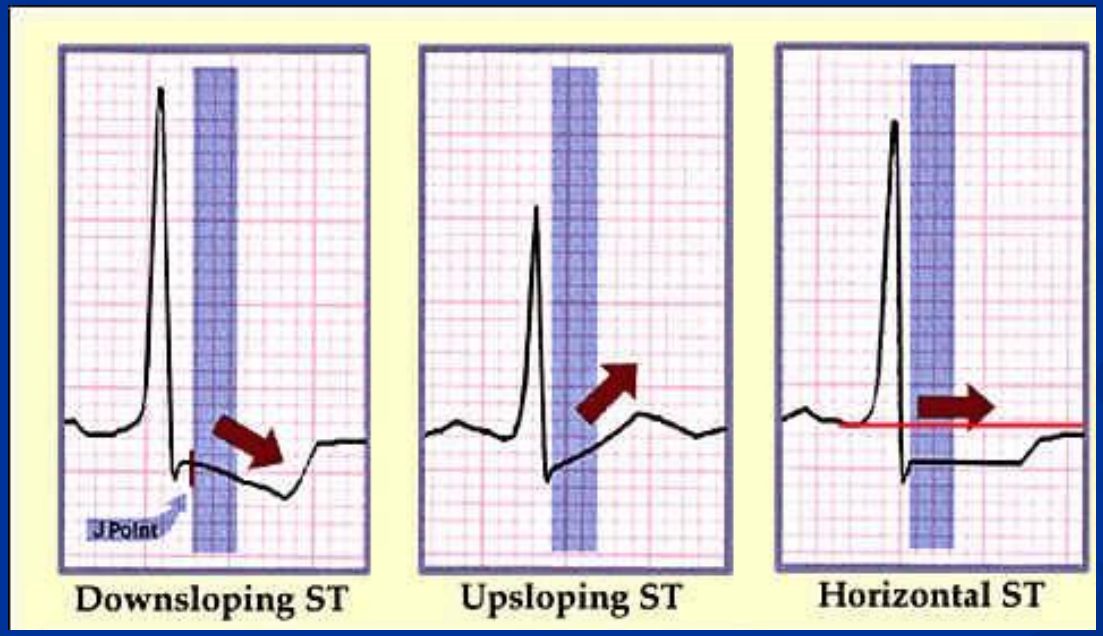
Leads V3, V4 – Anterior wall

ST depression, T wave inversion
It's trying to tell you something





ST segment depression = Ischemia



File: ecg ST depression
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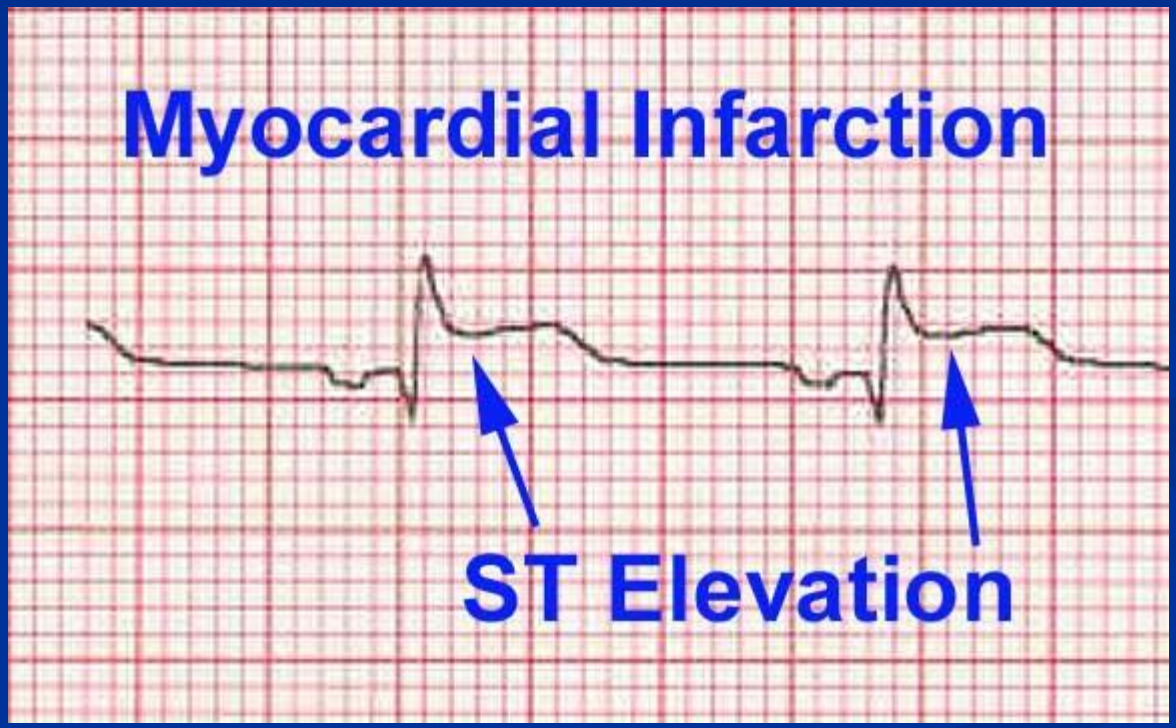
ST Elevation – INJURY and it's happening NOW



File: ecg ST elevation
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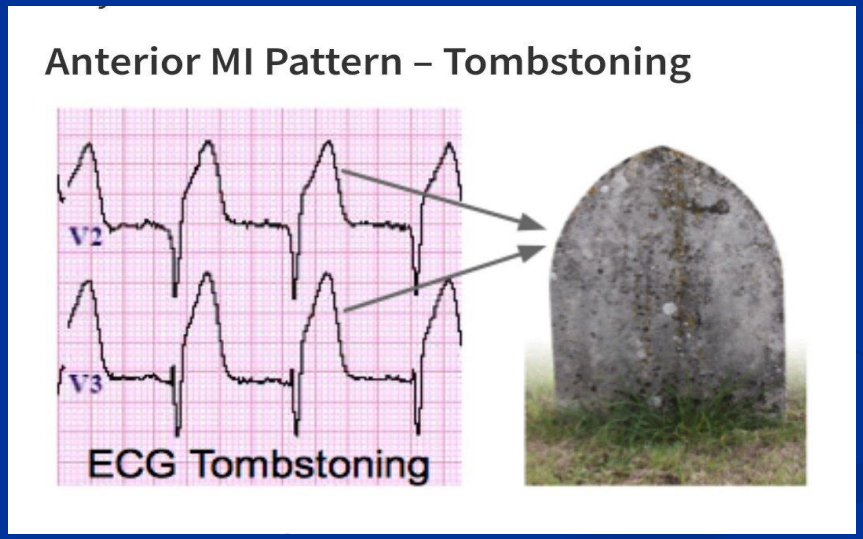
Tombstone?

- An old term "Tombstoning"
- ST elevation high and wide



File: ecg anteriorMI
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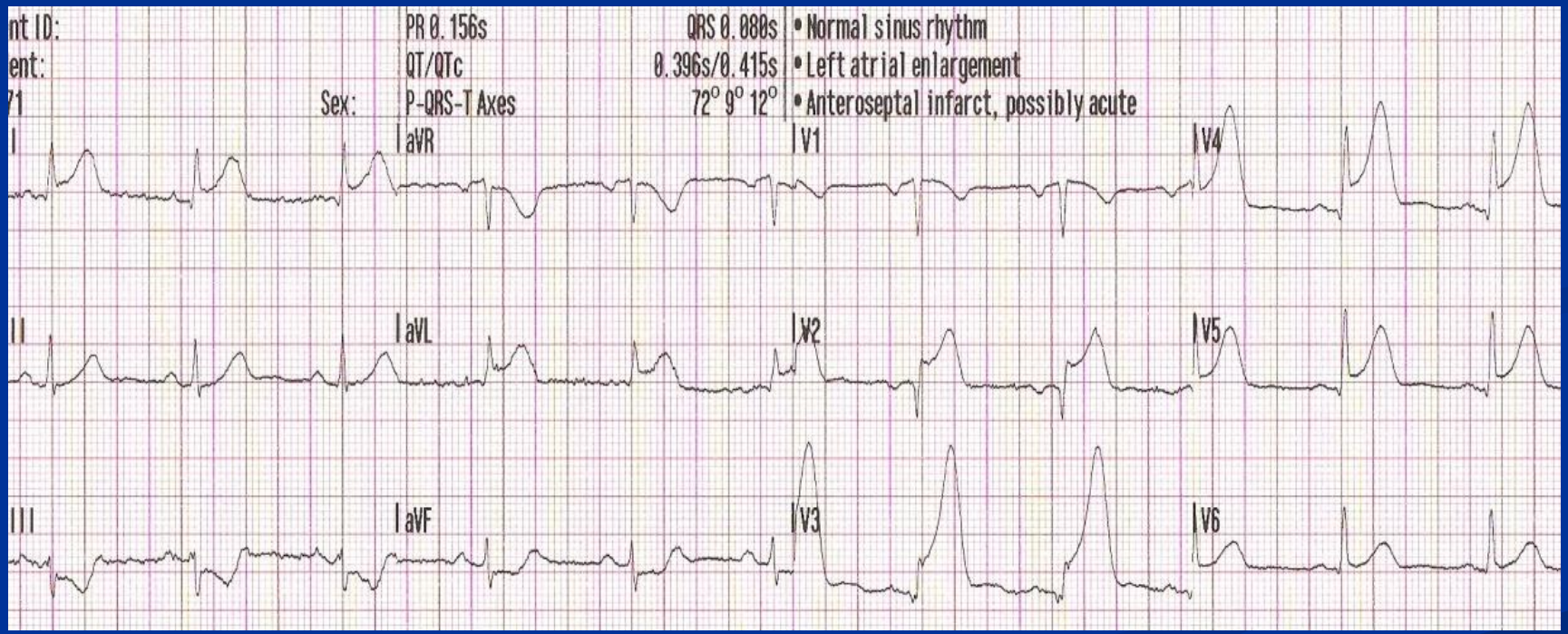
Trying to make some sense out of it all…

- Get into a habit of looking at EKGs in a certain way
- Make sure you look at each part of the heart on the EKG - inferior II, III, AVF, etc. Look for changes in 2 leads of an area "contiguous leads", this is significant.
- You can see opposite (reciprocal) changes (ST depression) in the other leads with a STEMI
- ST elevation in all leads can indicate acute pericarditis

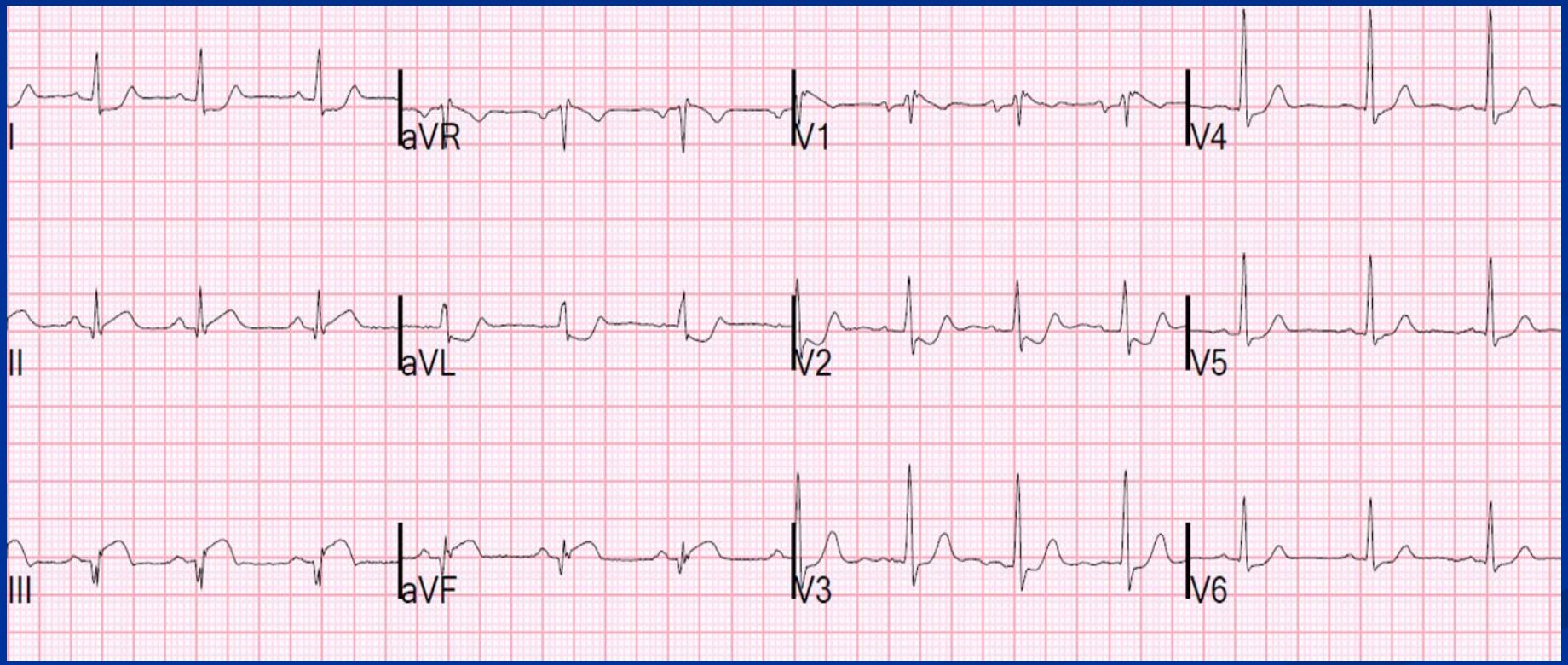
For the following EKGs:

- Get into the habit of looking at EKGs the same way each time
- Look at the leads of each area of the heart
- Check for ST elevation
- Check for signs of ischemia
- Check for arrhythmias

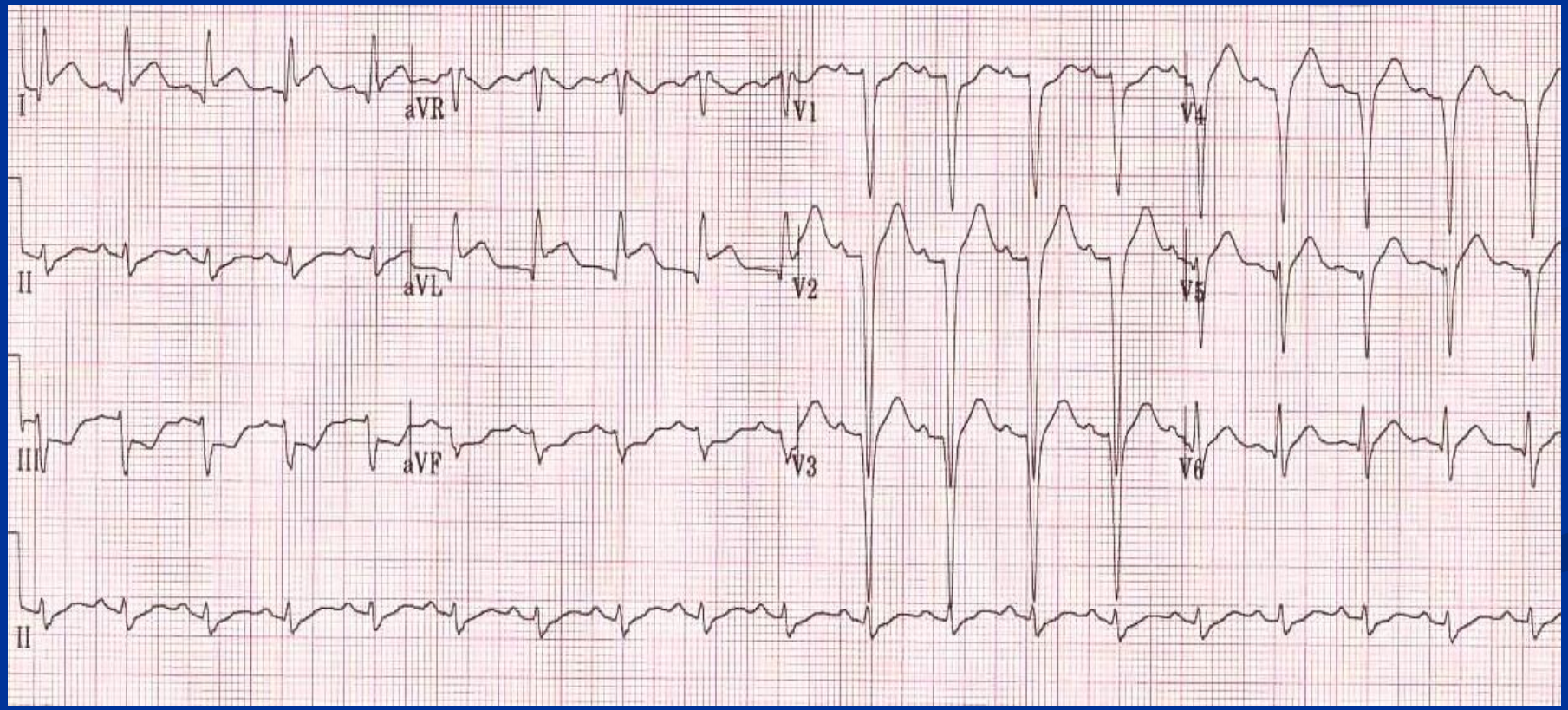
Antero-septal ST elevation MI with lateral involvement
Note: reciprocal changes inferiorly



Acute inferior STEMI
(2 contiguous leads)

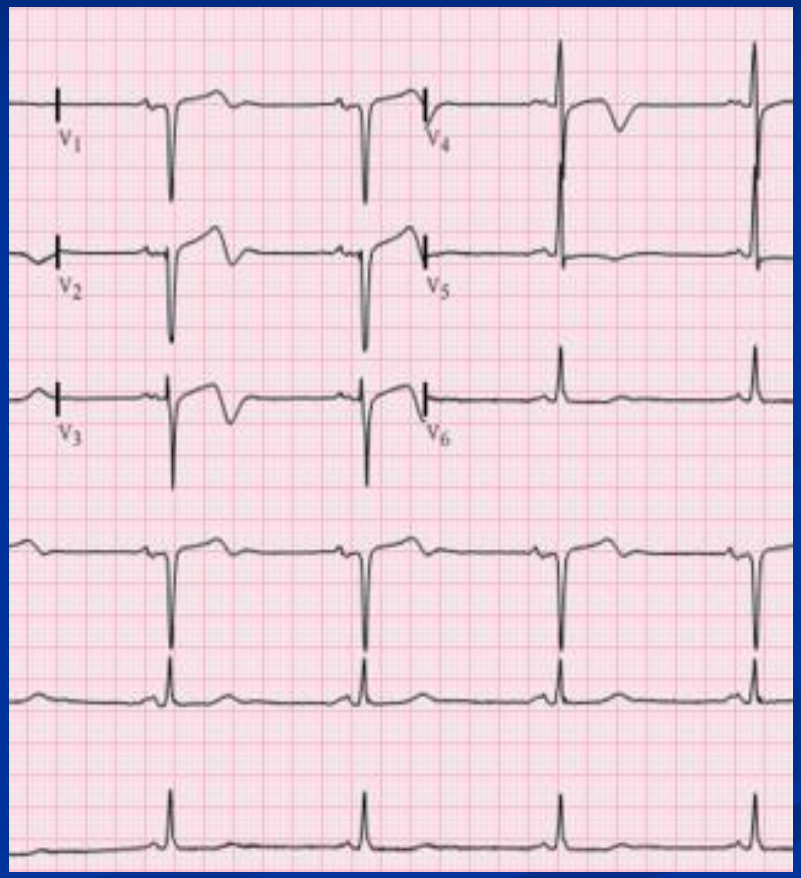


Anterior Lateral wall STEMI



Wellen's syndrome/sign

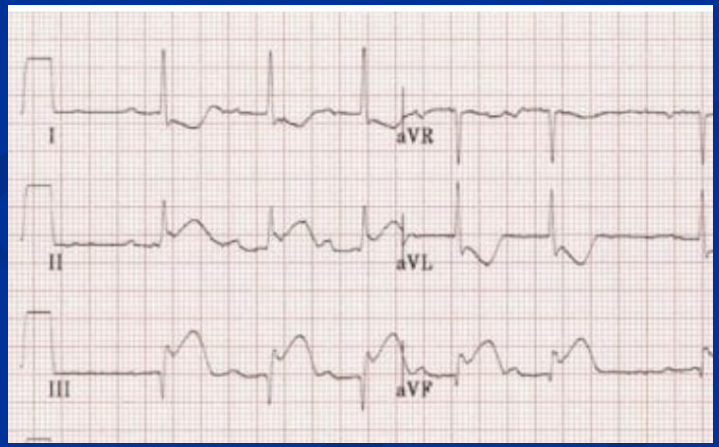
- Progressive symmetrical deep T wave inversion in leads V2 and V3
- Slope of inverted T waves generally at 60°-90°
- Discrete or no ST segment elevation
- No loss of precordial R waves
- EKG manifestation of ***critical proximal LAD stenosis***



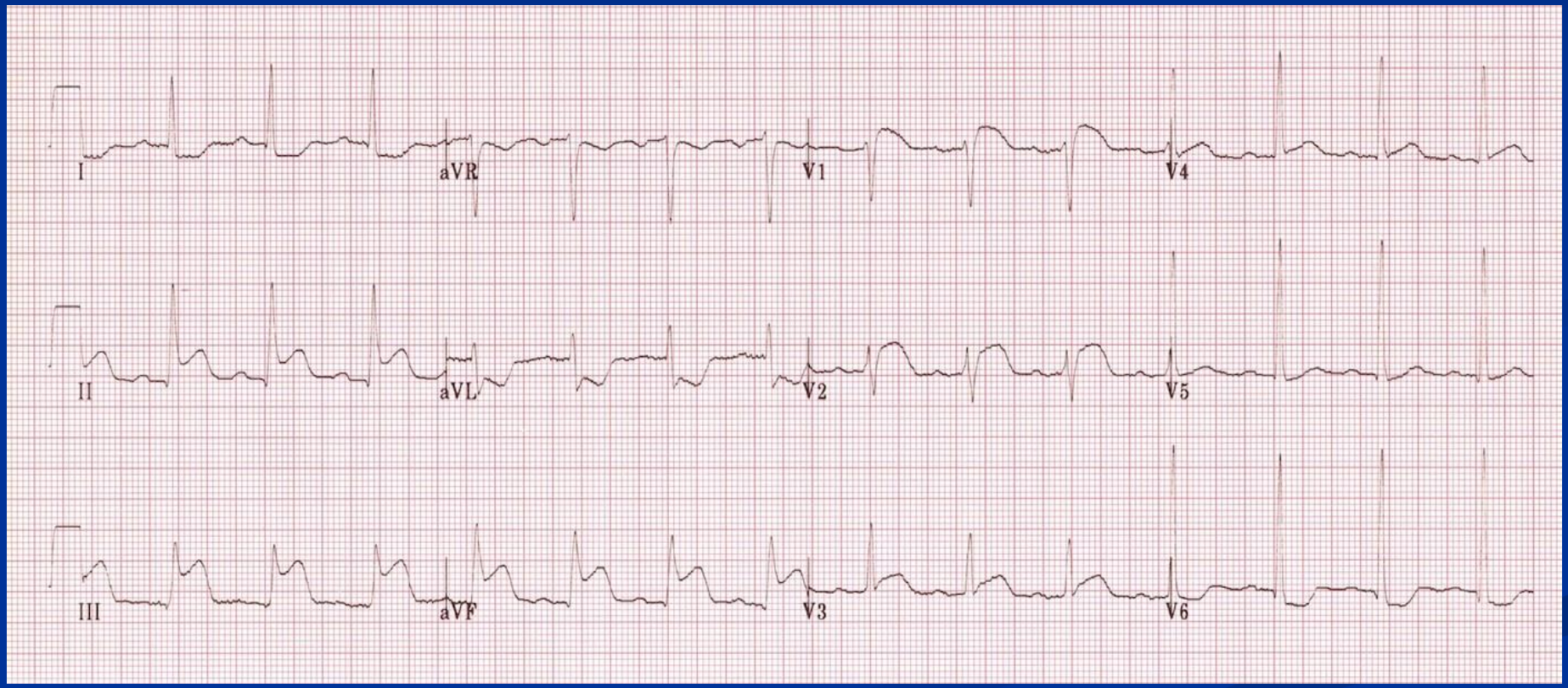
RV infarction

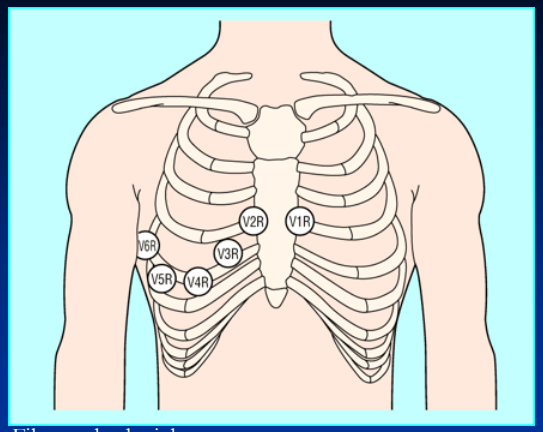
- Isolated RV infarctions are rare <10%
- Occurs in 10-50% of inferior STEMIs
- Tx: IVFs, pre-load dependent
- ST elevation in lead III > II
- Right-sided EKG

  ST elevation lead V4



Note: ST elevation V1 & V2 as well as Inferior STEMI





File: ecg leads right
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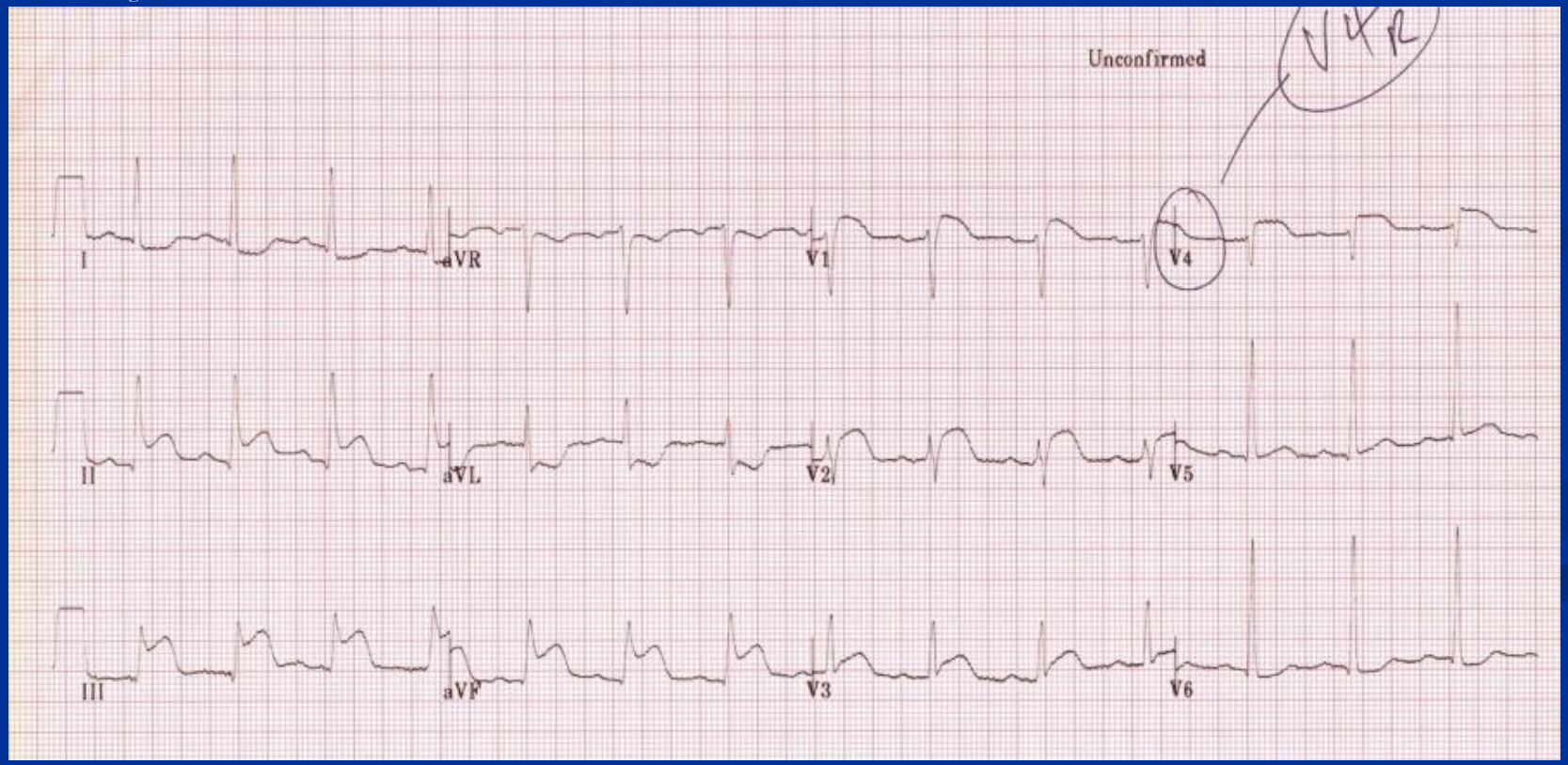
RV Infarct

Right-sided EKG

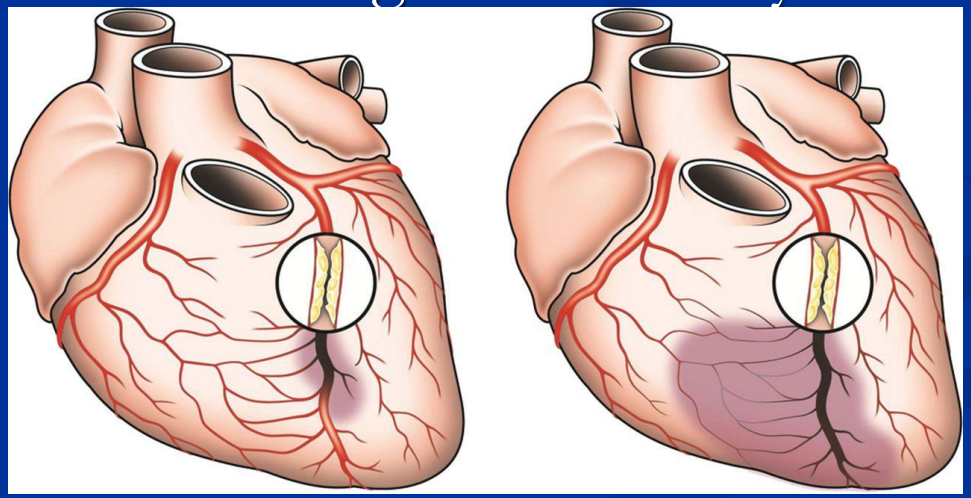


RV infarct

- Fluids!
- Pre-load dependent
- No NTG, no diuretics, no vasodilators
- No morphine or anything to decrease pre-load
- Treatment is reperfusion
- Once revascularized, may give meds as appropriate

Collateralization

- Collaterals –progressive ischemic CAD can result in the growth of new vessels (angiogenesis) & collateralization within the myocardium
- Increases myocardial blood supply by increasing number of vessels
- Often insufficient during increased myocardial O2 demands



File: heart occluded artery
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Chronic Total Occlusions (CTO)

- Onset is gradual. Body adapts creating small blood vessels that circumvent the blockage. The "collaterals" form a natural bypass, connecting the area that gets enough blood flow to the end of the vessel that is chronically occluded.
- This re-routed blood flow helps deliver blood to the heart, but collateral circulation often does not supply enough blood to meet increased demand during exercise.
- Can be treated by experienced interventionalist by PCI or bypass

Posterior MI

- Posterior wall myocardial infarction occurs when circulation becomes disrupted to the posterior heart, commonly occurs with inferior MI (PDA of RCA thrombus) can lead to significant area of infarction
- Symptoms may be classic, but due to more subtle EKG changes, it is often missed or misdiagnosed
- ST segment depression in the septal & anterior leads (V1-V4), because these leads see the MI backwards; the leads are placed anteriorly, but the myocardial injury is posterior
- Goals of therapy remain the same - reperfusion

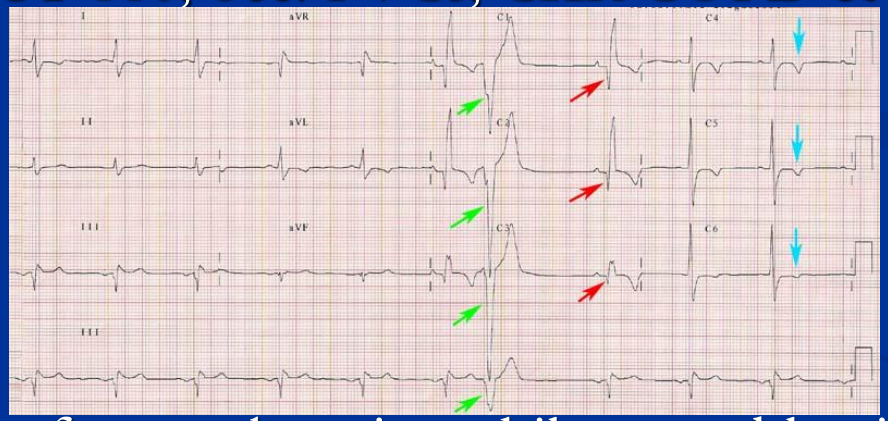
Dominance

- In 70% of the population, the right coronary artery (RCA) supplies the posterior descending artery (PDA), which supplies the posterior circulation. Known as "right dominant" circulation.
- In 10% of the population, the PDA originates from the LCx artery, known as "left dominant" circulation.
- In the remaining 20% of the population, the RCA and LCx both supply the posterior descending artery, known as co-dominant circulation.

Case study 3

- Mitch, 49 yo, hx HTN, DL, "borderline DM", smokes 1.5 ppd, +Fam hx, also hx CAD having had PCI to LAD 2 yrs before, on *no medications*
- Admitted to ER with c/o dyspnea & "a little CP"

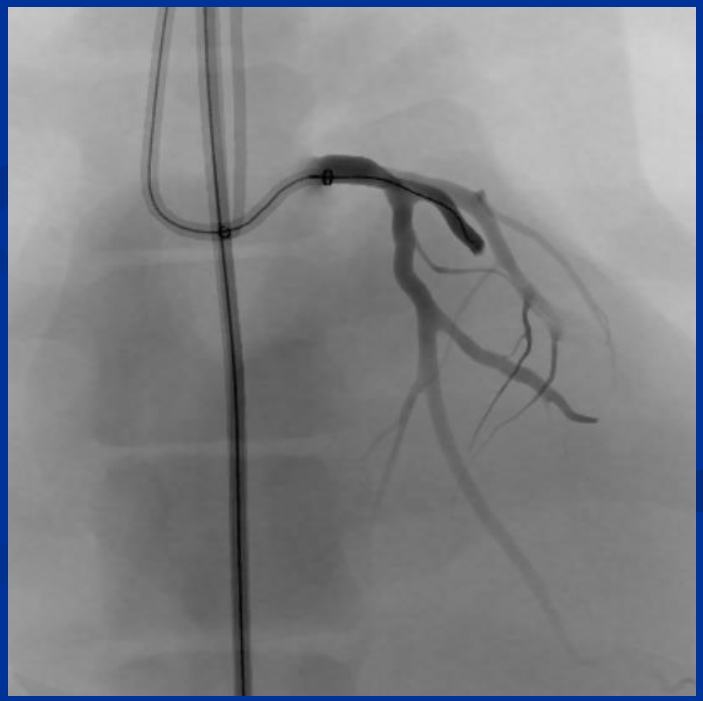
BP 176/100, ST 110, occ. PVCs, CXR COPD & congestion



- "Severe CP" a few weeks prior while snow blowing, made it in the house, laid on the floor, smoked a few cigarettes, finished snow blowing, "just like when I had a heart attack 2 years ago".

Case study 3

- First trop >1000, patient refused urgent angio until his prior interventionalist could perform it
- Treated with ASA, BB, IV heparin, NTG
- Cath showed CTO LAD, severe RCA & Cx disease, EF 10%, sent to higher level of care



File: angiography occlusion
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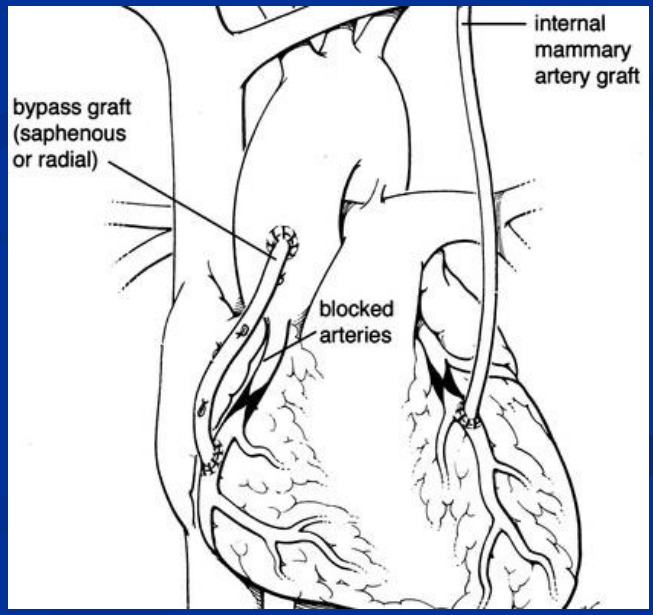
How to treat significant CAD?

- PCI - Balloon angioplasty, laser angioplasty, rotational atherectomy, angioplasty with stenting, protected PCI (use of Impella), high risk PCI
- CABG – bypass surgery
- GDMT – Guideline directed medical therapy



File: heart bypass
Commons.Wikimedia.org

Treatment

- Patients with nstemi, stable VS, no pain, can be managed with DAPT and PCI within 48 hrs of admission – improved in-hospital mortality and decreased LOS
- Patients with nstemi with refractory ischemia, hemodynamic or electrical instability, intractable chest pain – emergent cath/PCI

Treatment goal (STEMI or unstable) – immediate reperfusion

- PCI – give ASA, IV heparin, adenosine diphosphate inhibitor receptors

- Brilinta (ticagrelor) - BID
- Plavix (clopidogrel) – some non-responders (P2Y12)
- Effient (prasugrel) – post-PCI

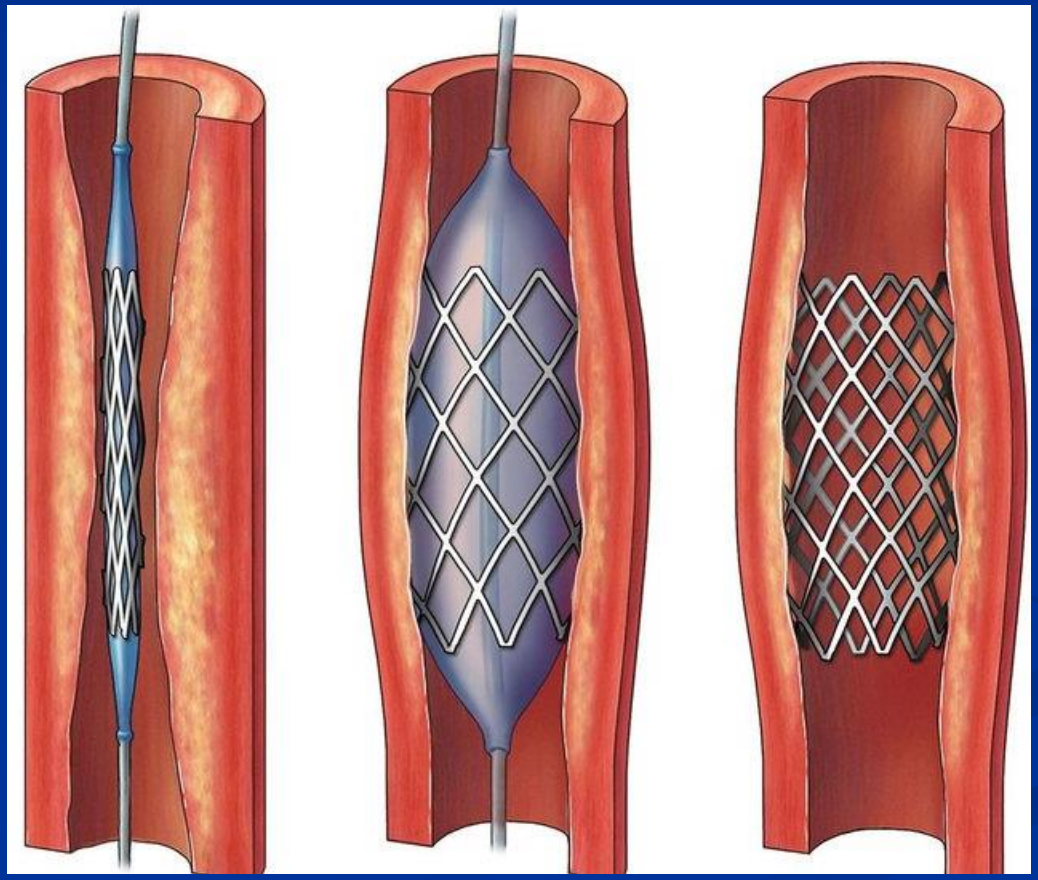
- At the time of PCI:

Glycoprotein IIb/IIIa inhibitor (Integrilin/eptifibatide)

Direct thrombin inhibitor (Angiomax/bivalirudin)

Percutaneous Coronary Intervention (PCI)

If STEMI, "primary PCI"



File: coronary artery stent
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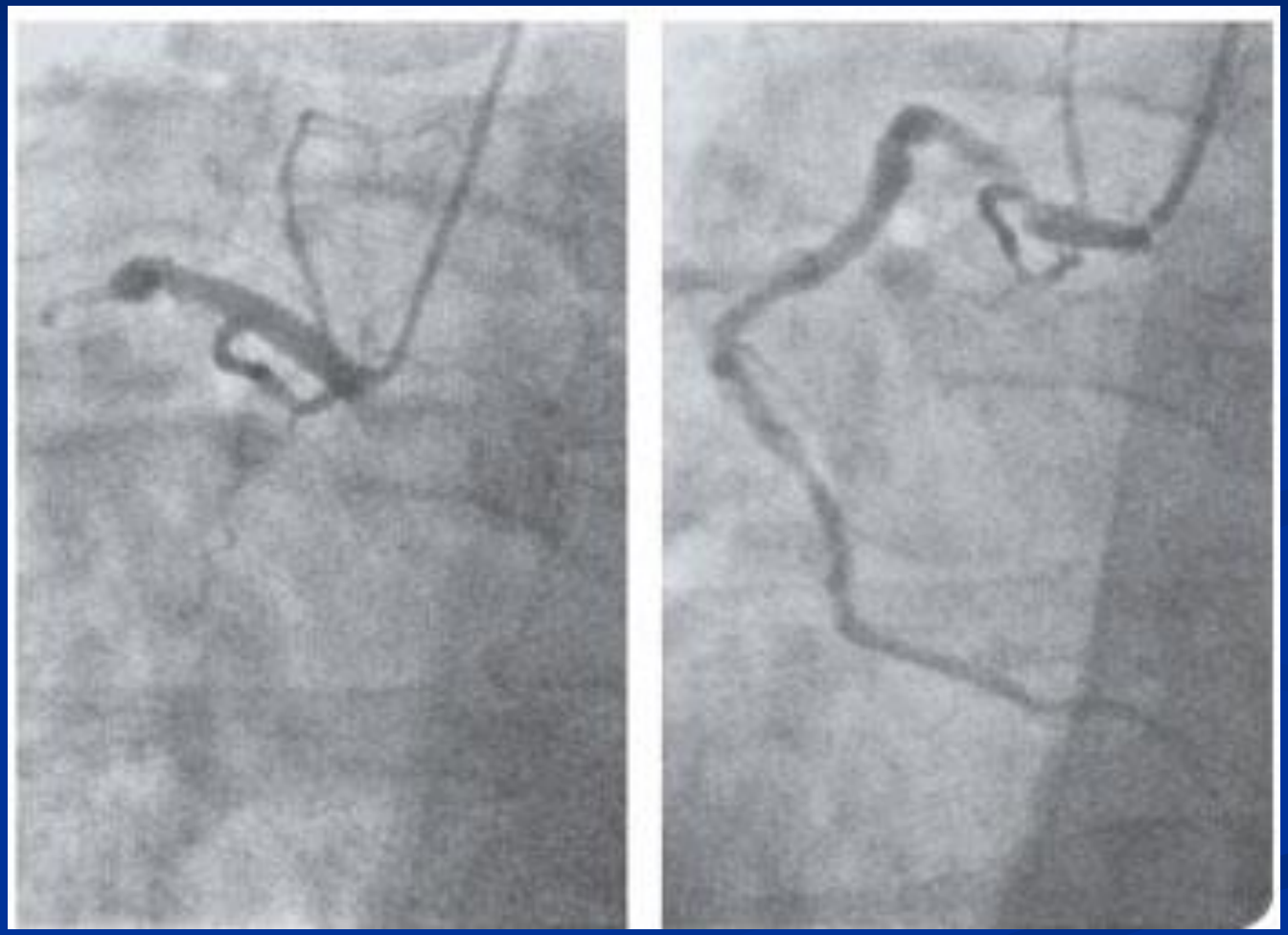
Occluded RCA (Inferior STEMI) Before and after PCI



File: angiography stenting RCA
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Indications for surgery

- Indications for CABG
  - presence of triple-vessel disease
  - severe left main artery stenosis
  - left main equivalent – 70% or > stenosis LAD & proximal circumflex disease
  - unsuccessful PCI
  - incomplete revascularization
- Patients without active ischemia, with complex coronary anatomy, poor LV function, DM, often are individualized decisions

Indications for surgery

- Mechanical complications of AMI requiring surgery – acute mitral regurgitation, rupture of the interventricular septum, LV free wall rupture

- Issue with patients on DAPT needing urgent surgery

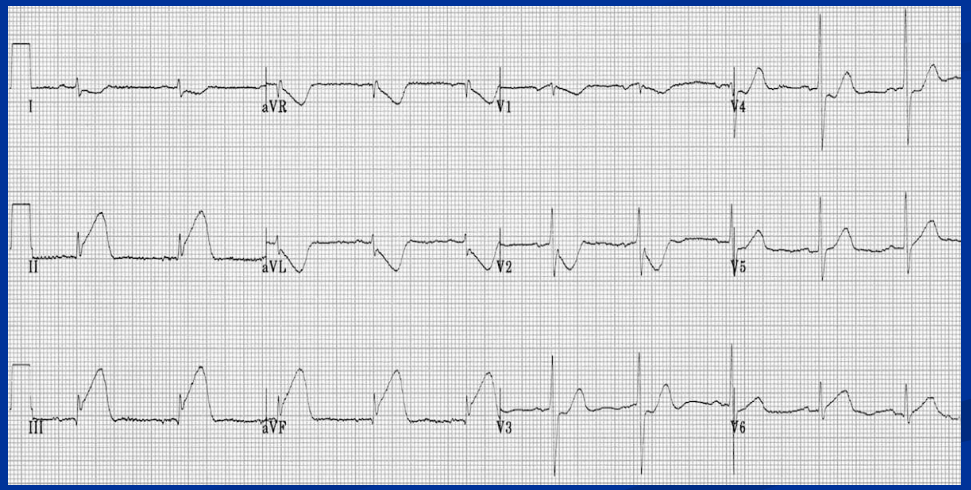
  Risk of ongoing bleeding 6X higher on DAPT

  Washout of 5 or >days is recommended

  Waiting at least 24 hrs if possible (AHA/ACC)

Case study 4

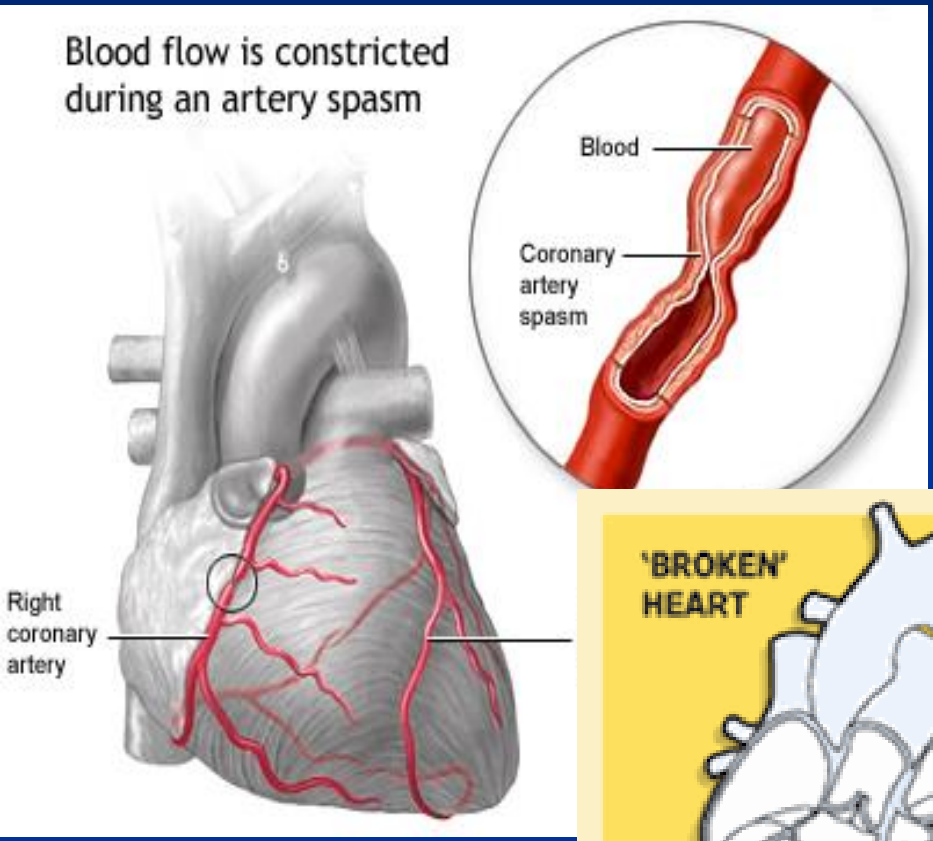
- Dorothy, 85 yo, hx HTN, DM, asthma
- c/o abd pain pointing to epigastric area, BP 140/88, SR 90, EKG non-spec ST-Ts, trop 10
- c/o worsening abd & neck pain, *given morphine*, BP 96/50, HR 60, EKG > Right sided EKG ST Elevation V4, IVFs given, cath lab with PCI prox RCA



Issues causing MI lookalikes

- Cocaine & Methamphetamine users can have coronary vasospasm causing elevated biomarkers and abnormal EKG's, even STEMI's. Cath shows normal coronaries, they usually do not have permanent damage.
- Takotsubo's CM, Broken-Heart Syndrome. Positive enzymes, CP, EKG abnormalities including STEMI. Big heart (CM), apical ballooning, normal coronaries. May be proceeded by a very stressful event. Treat with BB's, ACE's, diuretics if needed. It will usually resolve in 3 mos.

Coronary vasospasm & Takotsubo's Cardiomyopathy



File: heart artery spasm
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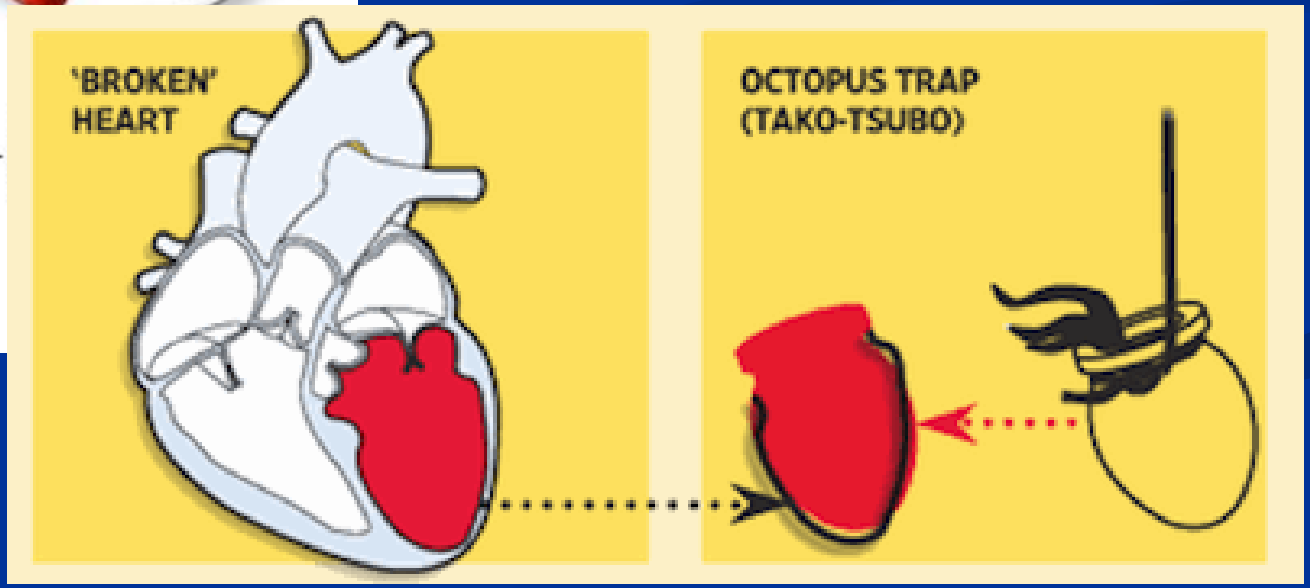

Academic Life in Emergency Medicine (ALiEM), 2013

What do patients need to know? Medications

- ***Medications –*** the **WHY** – take your Brilinta to keep your stent open or risk stroke, MI, death
- The **WHAT** – what the medication does *for them*
- ***Provide practical ways to remember –*** the **HOW** – Brilinta must be taken twice a day so put your pill bottle on your pillow after morning dose
- The **WHEN** – before stopping any meds, speak to your provider (ie. elective colonoscopy – pts are told to stop any blood thinners) Can have catastrophic results

What do patients need to know?

- Reasons for their other medications – BB, ace/arb, statin, etc. Explain the *what, why, when, how*
- Follow-ups: PMD, Cardiologist
- Diet, limiting ETOH
- Exercise/cardiac rehab
- Signs/Symptoms to call provider
- Modification of risk factors – glucose control, etc

If patients have a low LVEF, they will need MORE teaching as they will be on more medications & need further instruction regarding sodium, ETOH, etc

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For your time and attention, I thank you <3

File: heart and ecg
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