

Walks Like a Duck, Talks Like a Duck – But It's a Zebra? Evaluation of Cardiac and Non- Cardiac Chest Pain

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2026 Cardiology Speaker
Skin, Bones, Hearts & Private Parts



Disclosures

- Investigator-Driven Research Grant, Abbott Labs, 2023-2026
- VAD Coordinator Professional Development Grant, ICCAC, 2024-2026

I will occasionally discuss patient scenarios – any pictures directly of patients are displayed following consent and will be noted

Objectives

- Overarching Theme of Causes of Chest Pain
- Complex Evaluation and Treatment of Non-Ischemic Chest Pain
 - Gastroesophageal Reflux Disease (GERD)
 - Acute Pulmonary Embolus (PE)
 - Acute Aortic Dissection
 - Hypertension/Left Ventricular Hypertrophy
 - Pericarditis
 - Acute Heart Failure
- Complex Evaluation and Treatment of Ischemic Chest Pain
 - NSTEMI
 - STEMI
- Final PEARLS on Distinguishing Differences Between Types of Chest Pain

Chest Pain

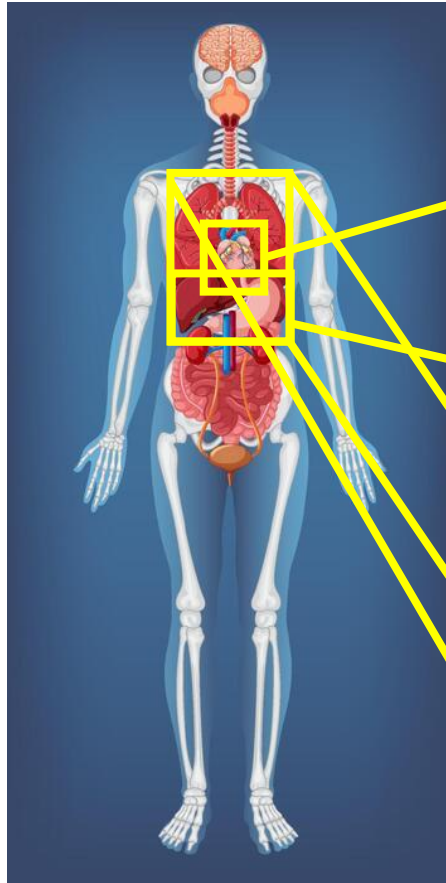
- Chest pain is “discomfort or pain that you feel anywhere along the front of your body between your neck and upper abdomen.”

-*MedlinePlus*

- Poor localization of pain due to lack of sensory fibers in the spinothalamic tracts
- “REFERRED PAIN”



Causes of Chest Pain



Most Common Causes of Left-Side Chest Pain.

Heart and vascular issues

- Heart attack.
- Coronary artery disease.
- Coronary artery dissection.
- Pericarditis.
- Hypertrophic cardiomyopathy.
- Aortic dissection.
- Aortic aneurysm.
- Mitral valve prolapse.

Digestive issues

- Esophageal spasm.
- Hiatal hernia.
- Gastritis.
- Gastroesophageal reflux disease (GERD).
- Pancreatitis.

Lung issues

- Pulmonary embolism.
- Chronic obstructive pulmonary disease (COPD).
- Pneumonia.
- Pleurisy or pleuritis.
- Pneumothorax (collapsed lung).
- Pulmonary hypertension.
- Asthma.

Musculoskeletal issues

- Broken rib.
- Costochondritis.
- Sprained chest muscle.

Other medical conditions

- Shingles.
- Lung cancer.

Cleveland Clinic

Most Common Causes of Right-Side Chest Pain.

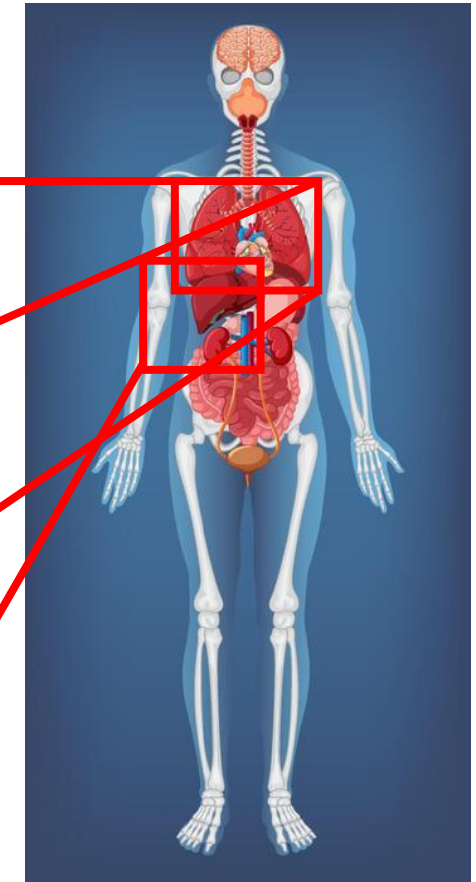
Pulmonary embolism. **COPD.** **Pneumonia.**

Pleurisy or pleuritis. **Collapsed lung.** **Pulmonary hypertension.**

Asthma. **Lung cancer.** **Broken rib.**

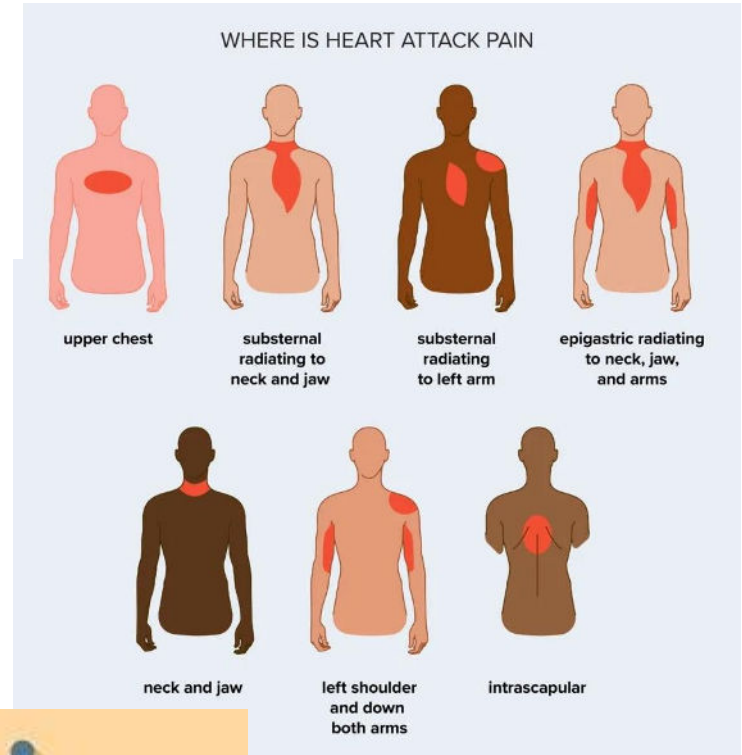
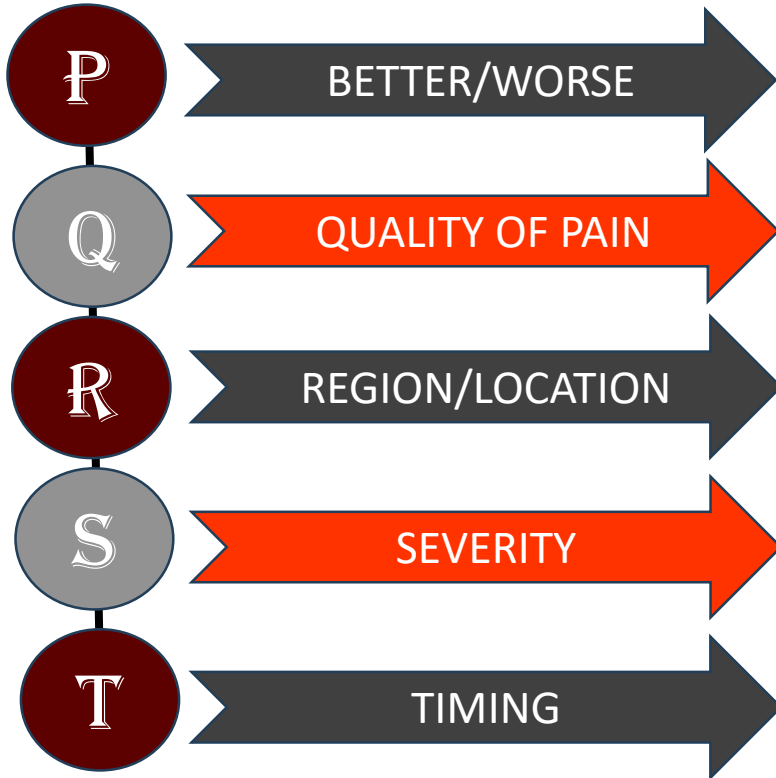
Sprained chest muscle. **Shingles.** **Gallstones.**

Cleveland Clinic

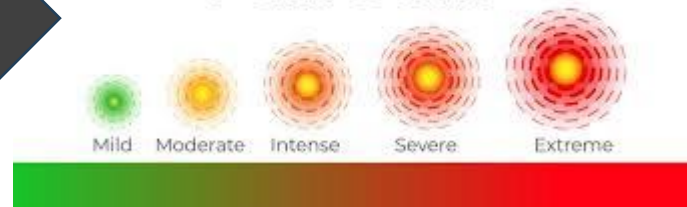


Complex Evaluation and Treatment: Non-Ischemic Chest Pain Scenarios

PQRST ASSESSMENT

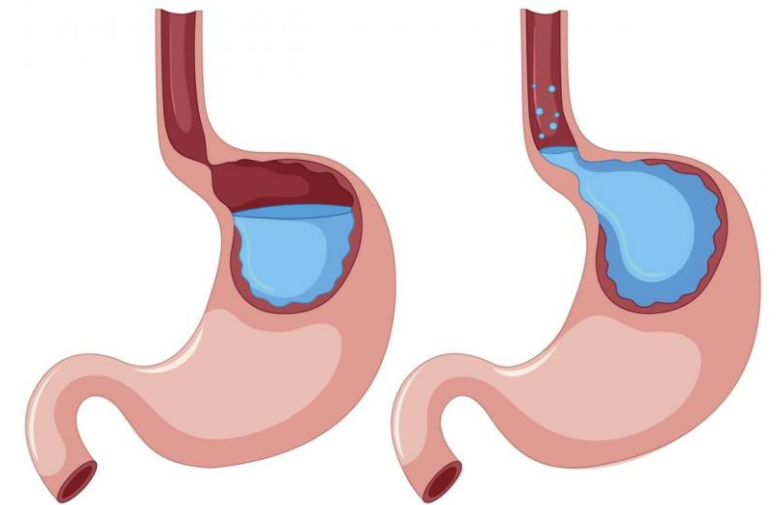


Pain Scale

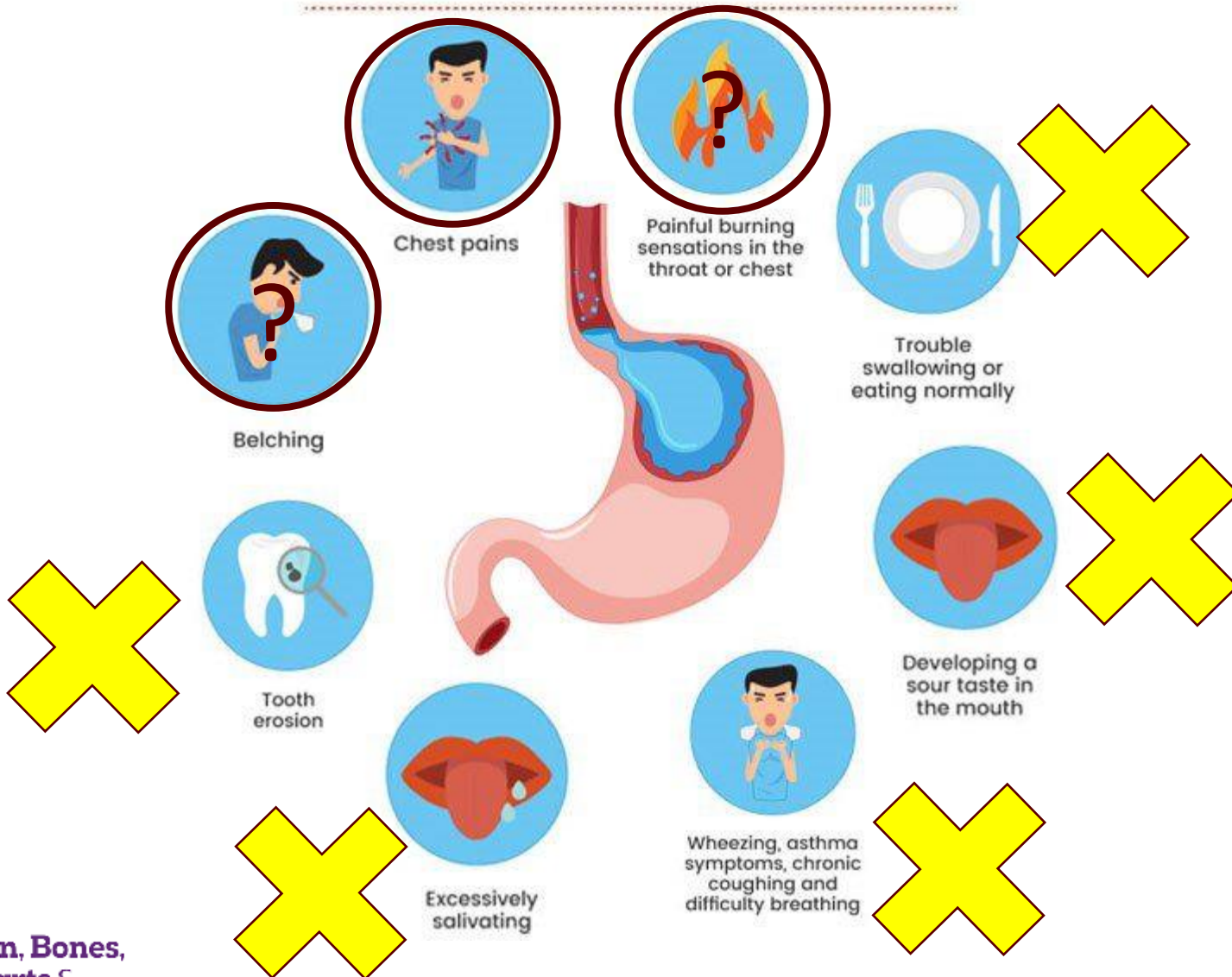


Gastroesophageal Reflux Disease (GERD)

- Stomach contents refluxes back up into the esophagus
- #1 Cause of “Non-Cardiac Chest Pain ER visit”
- Acid Reflux \neq GERD (acute vs chronic)



COMMON SYMPTOMS

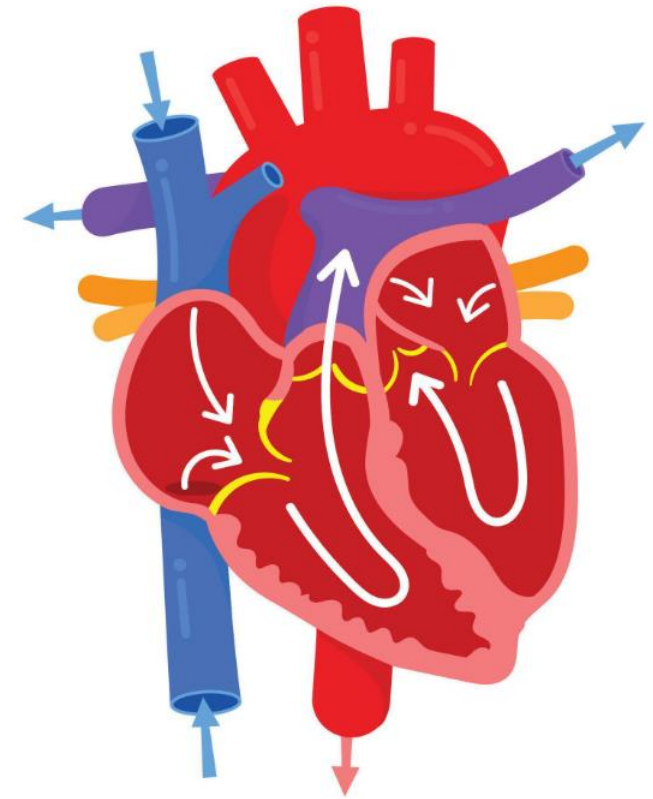


Best Treatments:



Acute Pulmonary Embolism (PE)

- Sudden blockage in a main pulmonary artery
- May or may NOT come from a DVT
- May be proximal or distal (centrally located in main PA or distally in the outward vessels)
 - This is important because of the type of study that needs to be done
- Can be FATAL if symptoms dismissed



Imaging Modalities for PE Evaluation

- **CT Angiogram of the Chest (with Contrast)**
 - 97-100% Sensitivity with 87-94% Specificity
 - Looking for LARGER vessel occlusions
 - Directly visualize the pulmonary arteries and RV dilation
- **VQ Scan**
 - 93% Sensitivity with 85 % Specificity
 - Used in Pregnancy, Renal Dysfunction, Prohibitive Weights and Iodine Allergies
 - Looks for MISMATCHED defect differences in ventilation and perfusion
 - Calculated as “Large (>75% of segment), Moderate (25-75% of segment) and Small (<25% of segment)”

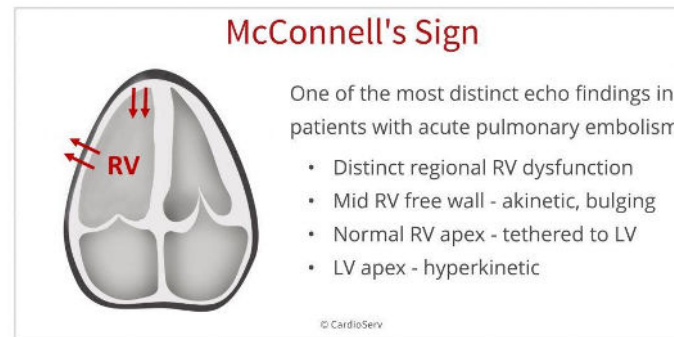
Abnormalities Seen

- **EKG Changes:**

- Sinus Tachycardia
- S₁Q₃T₃=Large S wave in lead I, Q wave and inverted T wave in lead III
- Right Axis Deviation (I negative and AVF positive)
- Right Bundle Branch Block (QRS \geq 120ms, V₁-V₂ rSR¹ or rsR¹; V₆ wide S wave)
- T- Wave inversion V₁-V₄ and Inferior Leads (II, III, AVF)

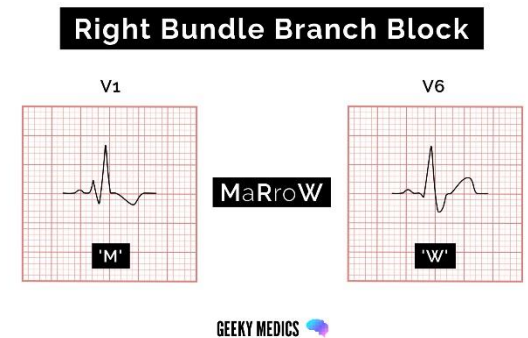
- **ECHO Changes:**

- RV enlargement
- RV dysfunction
- McConnell's Sign:



- **CTA or VQ Scan:**

- Clots are seen on exam, or mismatch between ventilation and perfusion



78-Year-Old Female...

- No history of Coronary Artery Disease (CAD)
- Admitted for elevated Troponin (0.192), Limb Weakness, Paroxysmal Nocturnal Dyspnea (PND), Orthopnea and Dyspnea On Exertion(DOE)
- Had **syncopal** spell in bathroom with severe lightheadness/dizziness
- Cardiology asked to consult for possible “CHF” (weight gain, lower extremity edema, etc...)
- Echo: EF 55%, **right ventricle was severely hypokinetic and dilated**, Moderate tricuspid regurgitation, **severe pulmonary hypertension (RVSP 72mmhg)**

Oper: RBP

HR 76 . SINUS RHYTHM
 RR 789 . LEFT ANTERIOR FASCICULAR BLOCK
 PR 172 . LATE PRECORDIAL R/S TRANSITION
 QRSD 92 . CONSIDER LEFT VENTRICULAR HYPERTROPHY
 QT 520 . NONSPECIFIC T ABNORMALITIES, INFERIOR LEADS
 QTc 585 . PROLONGED QT INTERVAL
 * No Prior Tracing

Interpreting MD: MJ

-- AXIS --
 P 54
 QRS -57
 T -51

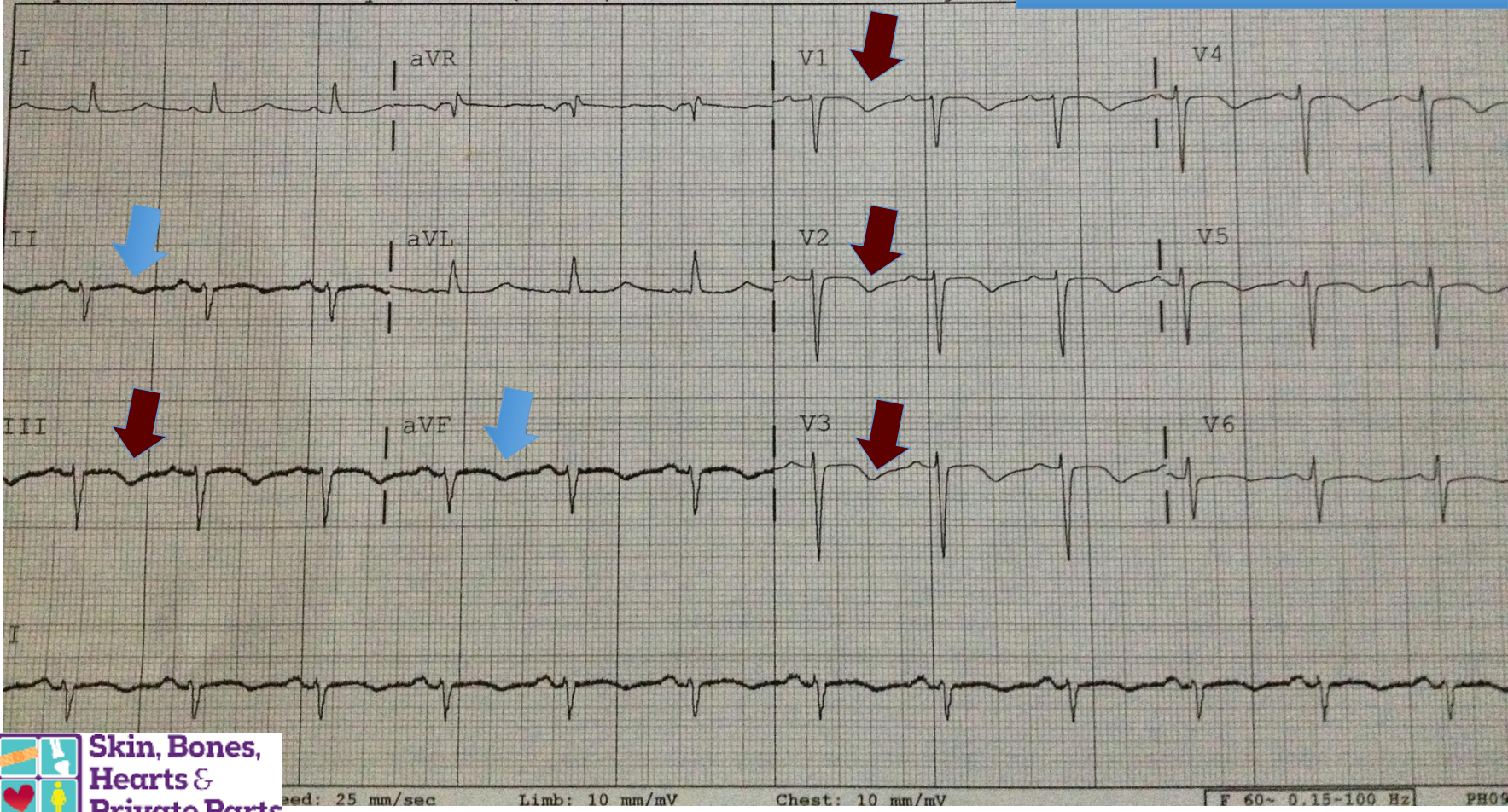
- ABNORMAL ECG -

Order #: 2299011
 Enc ID: 801746616

Standard 12
 Requested By: ,

BryanLGH Medical Center - BryanLGH East (8-00-03)

Edited Signed: [REDACTED]

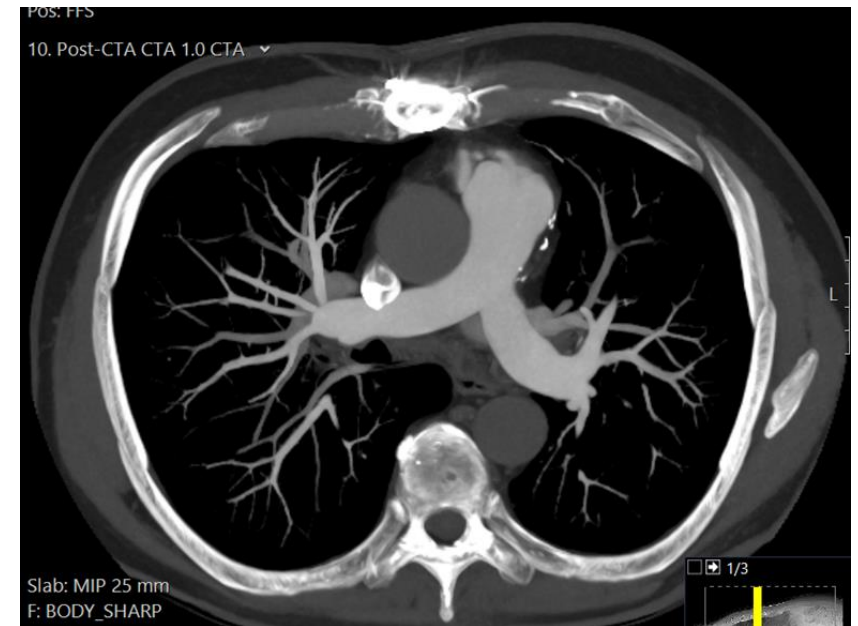


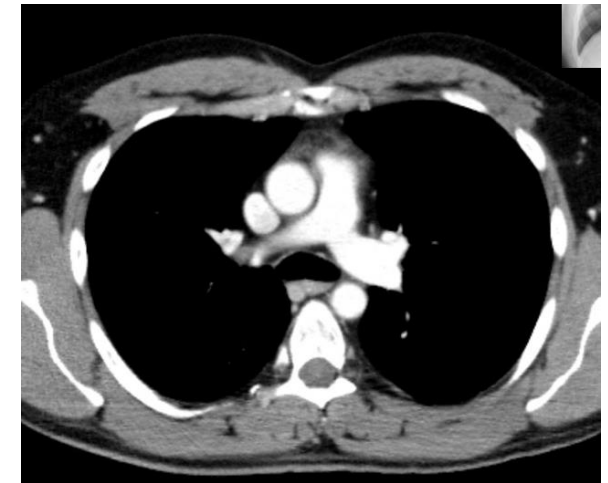
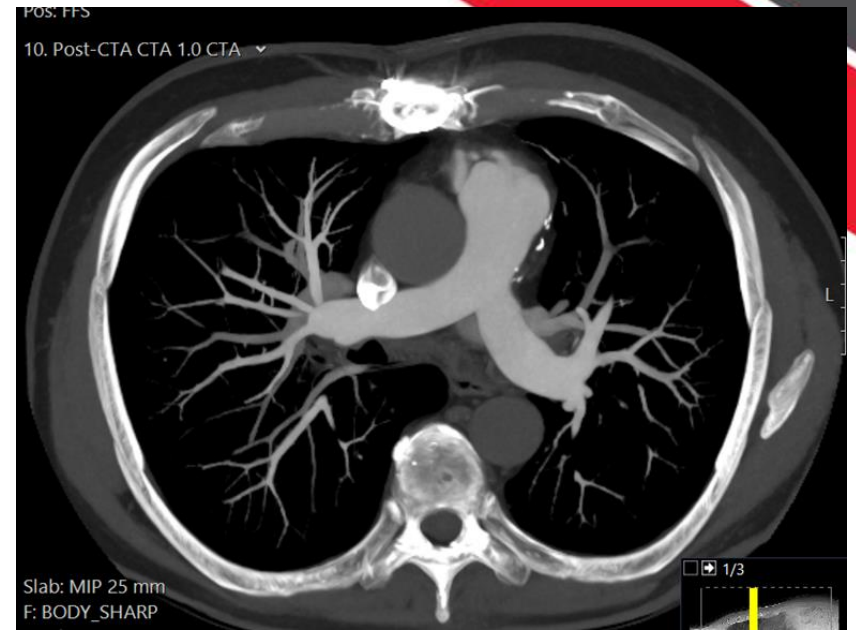
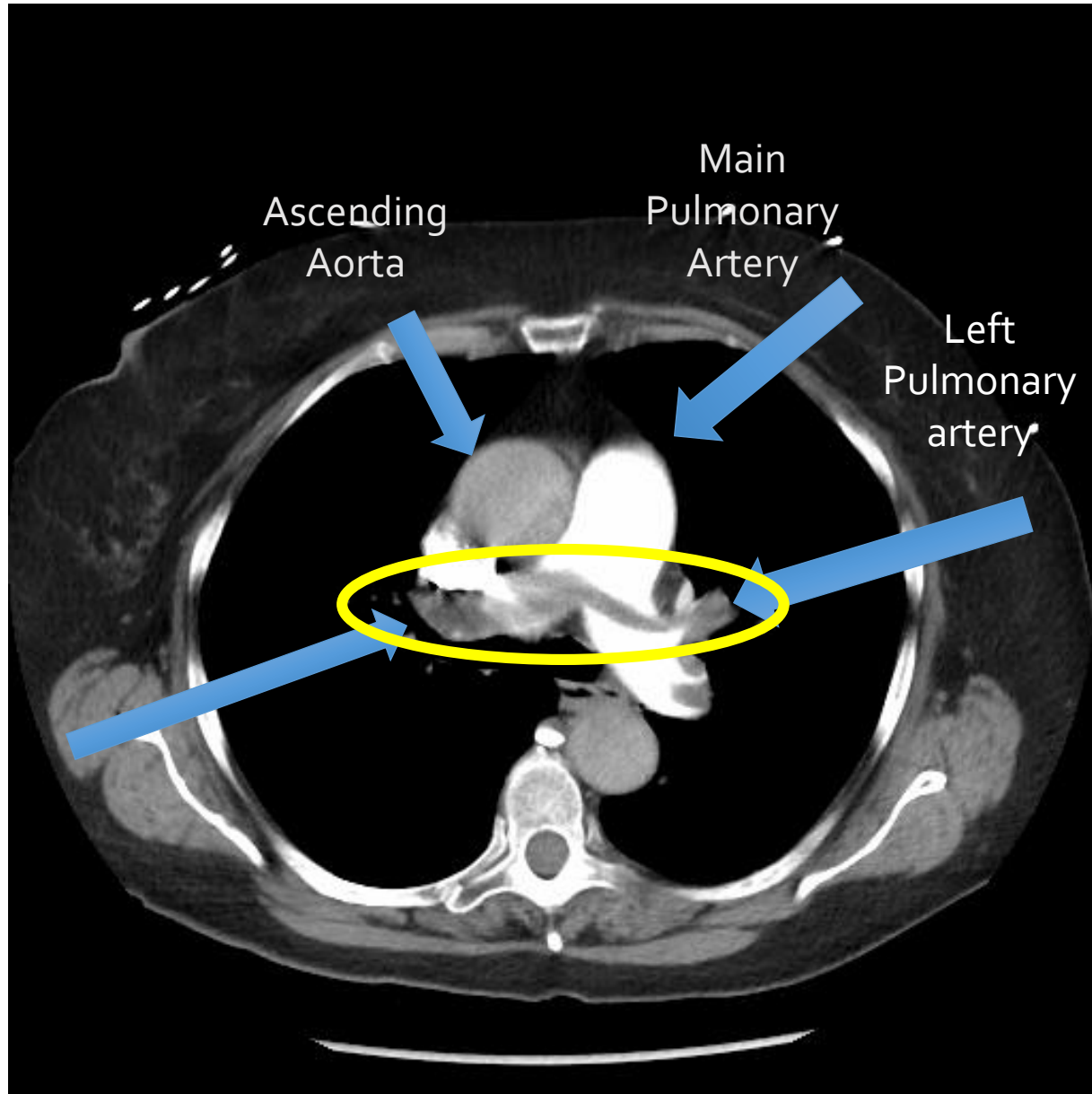
EKG changes:
 Sinus Rhythm, HR 76; inverted T
 waves in II, III, AVF, and V1-V3-
 V6

- D-dimer came back at >20 (measures degradation of fibrinolysis).
- CT angio of chest ordered to look for pulmonary embolism.
- Lots of discussion about if she could have it because her creatinine wasn't to where radiology wanted it at (although normal).
- Had to talk to the radiologist personally to tell the story and basically got told that he would do but would be VERY surprised if it would be abnormal.

Got a call 10 minutes later that her CT Angio showed:

LARGE BILATERAL PULMONARY EMBOLUS IN A SADDLE BAG FASHION-extending into both lower lobes and into the upper lobe as well. High degree of occlusion noted especially on the **RIGHT**.





The Story Goes On...

- After I got called about the embolus, I contacted all of the physicians.
- Talked with CT surgery, they declined to surgically fix it because she was stable and recommended tPA (clot buster)
 - She was not significantly hypoxic and hemodynamically unstable at the time
- She was given tPA in the ICU, started on dobutamine to help her right heart > stayed for another few days
- Scanned her legs (as PE's most often times comes from DVTs): **partial occlusion to the right popliteal vein (acute/subacute) and acute occlusive DVT at both channels in right peroneal veins**
- Discharge ECHO: RV normal in size, EF 45-55%, RV mild to moderate functional reduction and RVSP 37 (mild pHTN)
- **Discharged without any issues on**: Warfarin with goal INR 2-3 with PCP management

Current Guidelines for tPA Management

Patients With Symptoms Suggestive of ACS

Patient calls 911:

Patient takes own:

Relative Contraindications

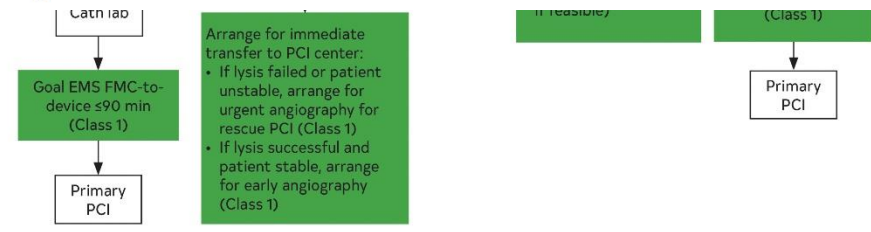
- History of chronic, severe, poorly controlled hypertension
- Significant hypertension on presentation (SBP >180 mm Hg or DBP >110 mm Hg)
- History of prior ischemic stroke >3 mo
- Dementia
- Known intracranial pathology not covered in absolute contraindications
- Traumatic or prolonged (>10 min) CPR
- Major surgery (<3 wk)
- Recent (within 2 to 4 wk) internal bleeding
- Noncompressible vascular punctures
- Pregnancy
- Active peptic ulcer
- Oral anticoagulant therapy

Table 14. Absolute and Relative Contraindications for Fibrinolytic Therapy in STEMI*

Absolute Contraindications

- Any prior ICH
- Known structural cerebral vascular lesion (eg, arteriovenous malformation)
- Known malignant intracranial neoplasm (primary or metastatic)
- Ischemic stroke within 3 mo except acute ischemic stroke†
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed-head or facial trauma within 3 mo
- Intracranial or intraspinal surgery within 2 mo
- Severe uncontrolled hypertension (unresponsive to therapy) (SBP >180 mm Hg or DBP >110 mm Hg)

activator; and tPA, tissue-type plasminogen activator.



UpToDate, 2025. [UpToDate](#)

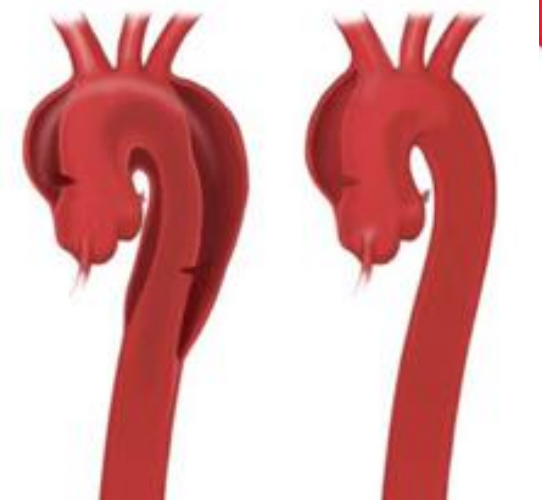
[2025 ACC/AHA/ACEP/NAEMSP/SCAI Guideline for the Management of Patients With Acute Coronary Syndromes: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines | Circulation](#)



Acute Aortic Dissection

- “Occurs when a tear in the intimal layer of the aorta allows blood to flow between the layers of the aortic wall, creating a false lumen between the intima-media and media-adventitia layer”

-Levy et al., 2024



**Mortality Risk: first 48 hours
of 0.5% per hour**

- 33% die within the first 24 hours
- 50% die within the first 48 hours
- 2-week mortality is 75% in undiagnosed patients

30-day Survival After
Repair = 90%

Sudden,
Severe
Onset of
Pain

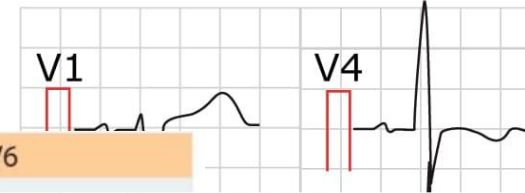
Tearing,
Stabbing
Pain

Usually
NO EKG
changes

More
common in
HTN, Turner
Syndrome,
Marfan's or
Bicuspid
AoV

Hypertension/Left Ventricular Hypertrophy

A) Left ventricular hypertrophy (LVH)



B) Right ventricular hypertrophy (RVH)



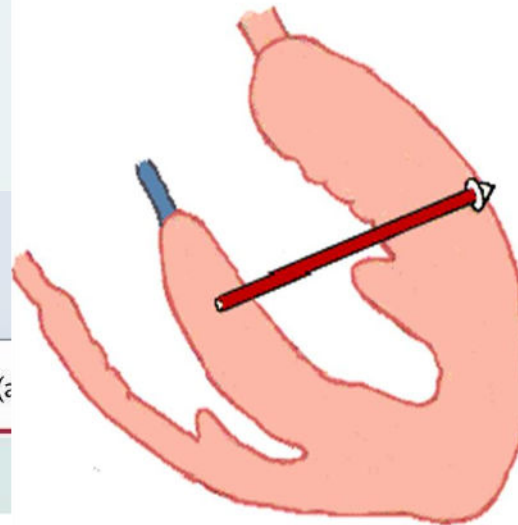
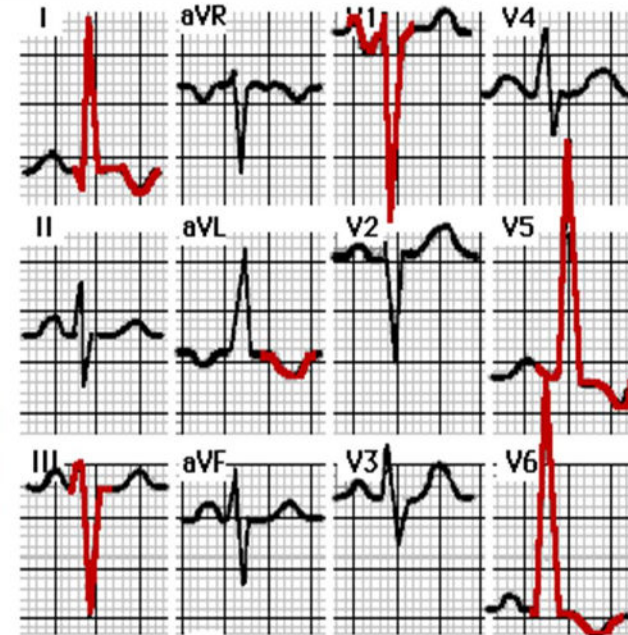
Left ventricular hypertrophy

V1/V2

V5/V6

Left Ventricular Hypertrophy

High voltage in limb leads: $(R I + S III > 25 \text{ mm.})$
 Or precordial leads: $(S V1 + R V5, \text{ or } S V1 + R V6, \geq 35 \text{ mm.})$
 Often, left atrial enlargement. ST - T abnormalities.



Arrow indicates major electrical vector of ventricular depolarization

The cut-ven



Sv

Rv

$S V_3 + R a V_L$ (Cornell voltage)
 Cornell duration product^b

Cornell Duration Product = $[S(V_3) + R(aV_L)] \times \text{duration}$

53-Year-Old Male...

- No history of CAD but does have HTN
- Admitted 2 months before for atypical chest pain
 - Stress at that time showed diaphragmatic attenuation but no ischemia
- Came in as a cardiac alert (at table drinking coffee when he developed **sudden and severe CP**)
- EKG had inconsistent changes because of left ventricular hypertrophy (tall R waves in LV leads or **deep S waves in RV leads, widened QRS or ST-T wave abnormalities, left axis deviation, left atrial enlargement** (double peaked T wave or biphasic P wave))

BR 59 SINUS RHYTHM
 RR 1017 PROBABLE LEFT ATRIAL ABNORMALITY
 PR 156 LEFT AXIS DEVIATION
 QRSD 88 * LEFT VENTRICULAR HYPERTROPHY WITH REPOLARIZATION ABNORMALITY, CANNOT RULE OUT ISCHEMIA
 QT 492
 QTc 488 * SEPTAL INFARCT, AGE UNDETERMINATE
 . BORDERLINE PROLONGED QT INTERVAL

Interpreting MD: TG

Order #: 2604328
 Enc ID: 801752474

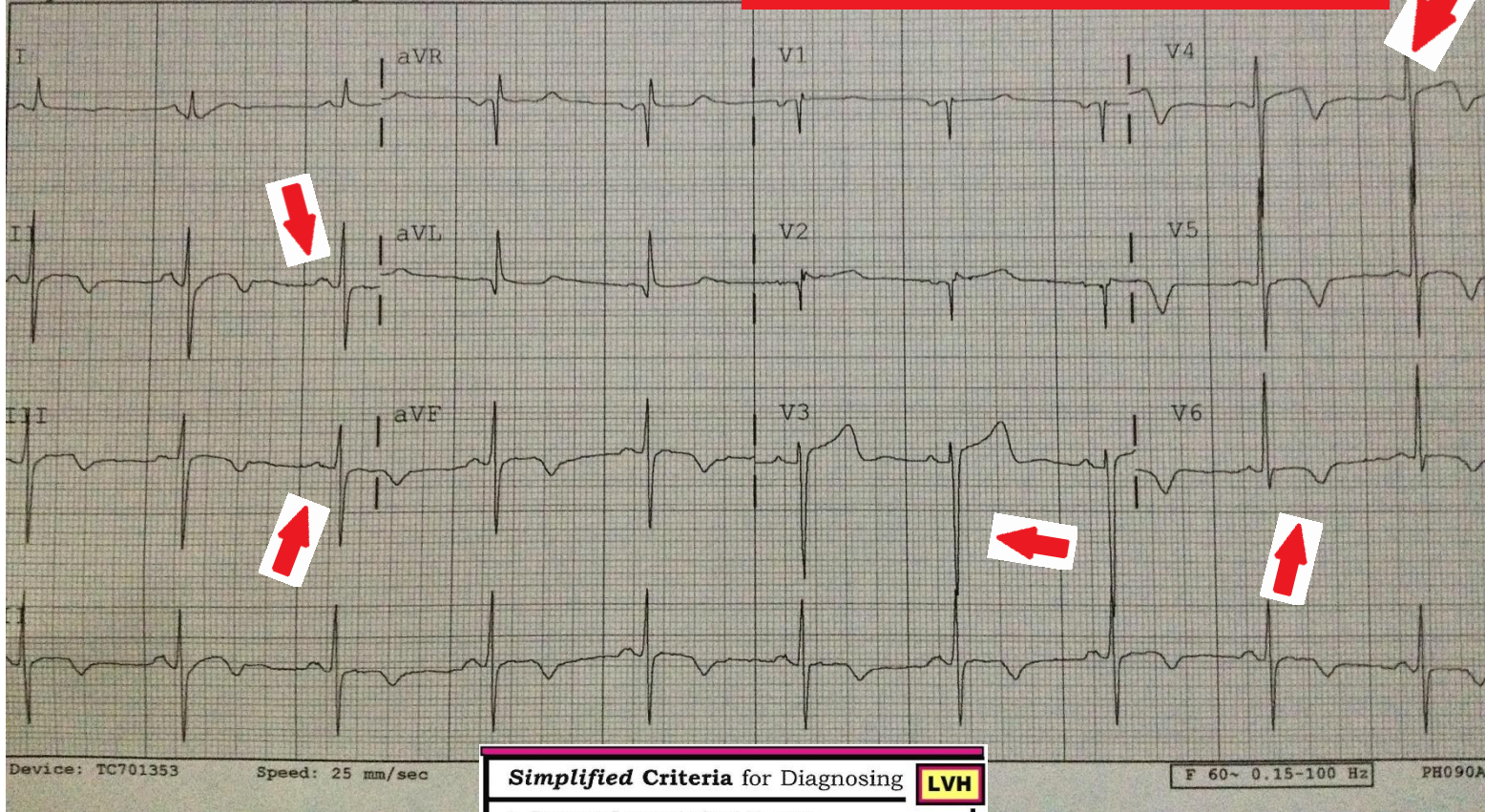
-- AXIS --
 P 51
 QRS -31
 T -90

- ABNORMAL ECG -

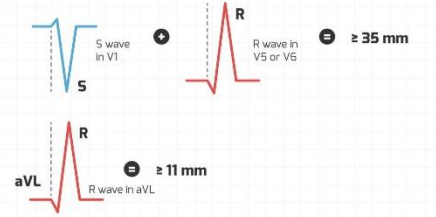
Previous ECG: 16-Dec-2012 04:57:52 - Abnormal Confirmed

Standard 12
 Requested By:

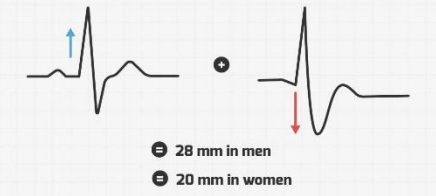
BryanLGH Medical Center - BryanLGH East (8-00-04)



SOKOLOW-LYON CRITERIA



CORNELL CRITERIA R wave in aVL + S wave in V3



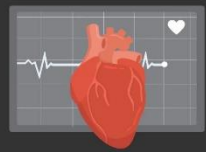
ROMHILT-ESTES POINT SCORE SYSTEM

Voltage criteria: 3 points	ST-T strain pattern: 3 points
Left atrial enlargement: 3 points	Left axis deviation: 2 points
QRS duration > 0.09s: 1 points	Delayed intrinsicoid deflection in V5/V6: 1 points
≥5 points = definite LVH	4 points = probable LVH

LVH-ASSOCIATED ST-T CHANGES

POINT SCORE SYSTEM

- Downsloping ST depression
- T wave inversion
- Seen in lateral leads (I, aVL, V5-V6)



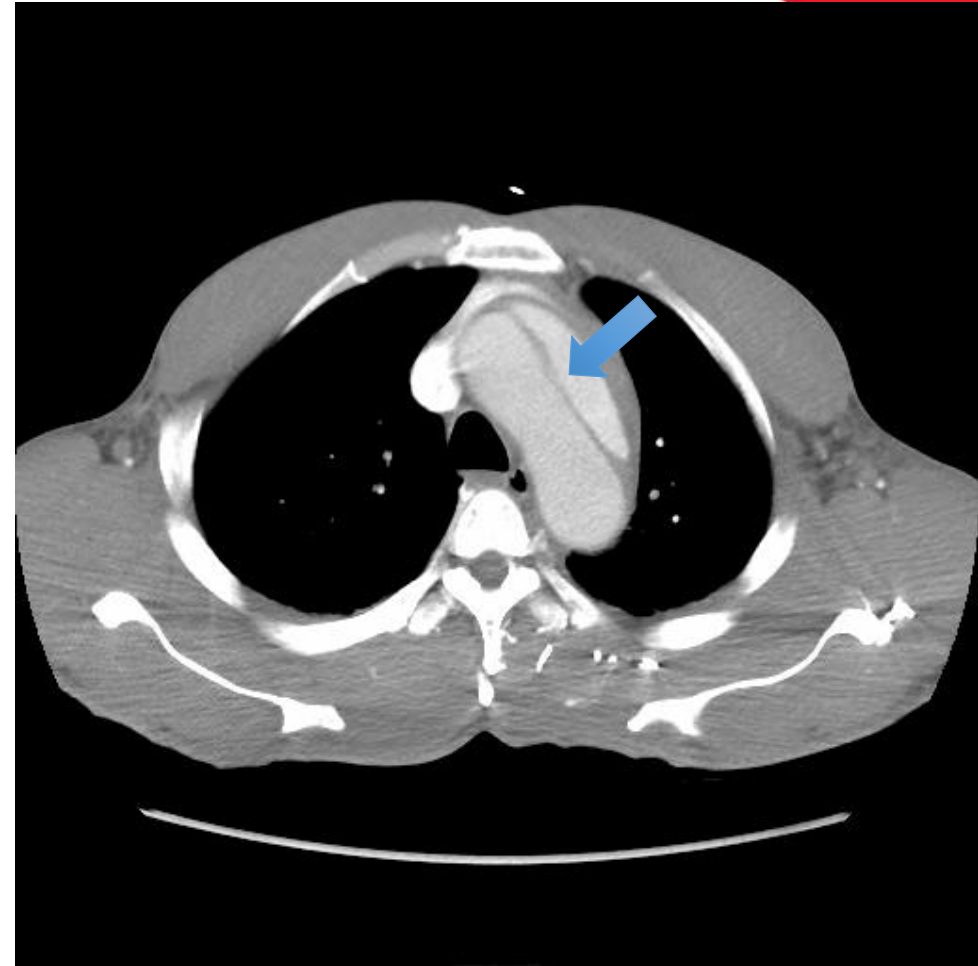
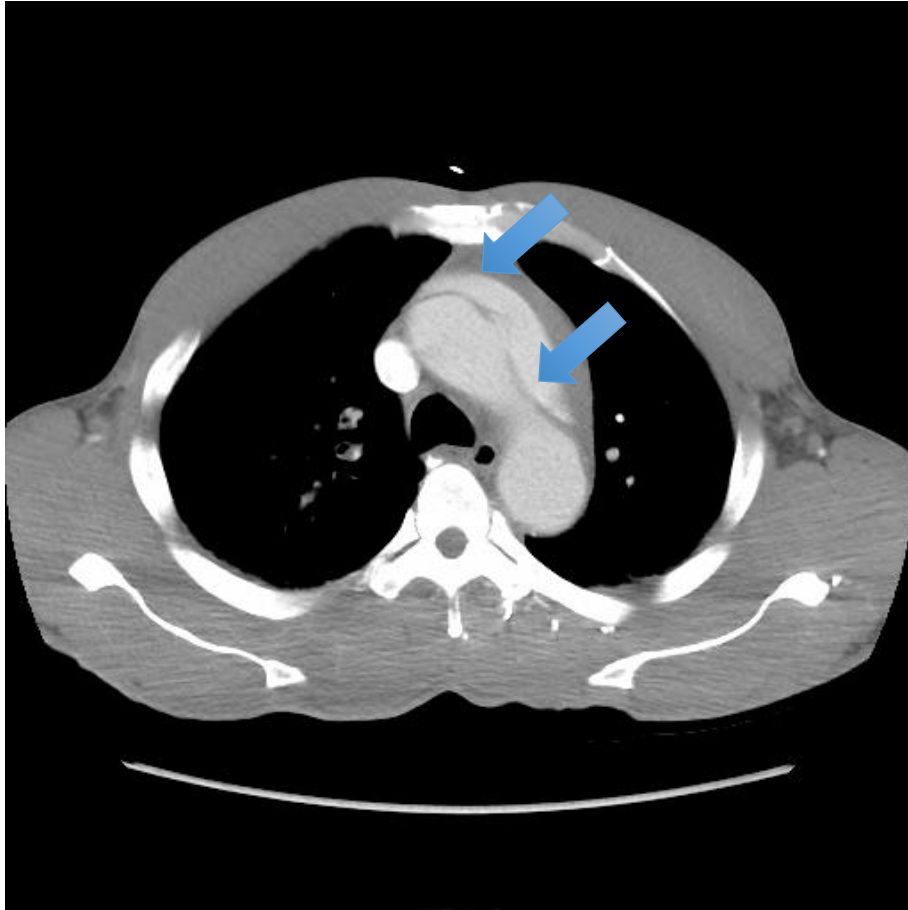
Simplified Criteria for Diagnosing LVH

- Deepest S wave in lead V₁ or V₂, plus tallest R wave in lead V₅ or V₆ ≥ 35.
 — and/or — R in lead aVL ≥ 12.
- Patient ≥ 35 years old.
- Left ventricular (LV) "strain".

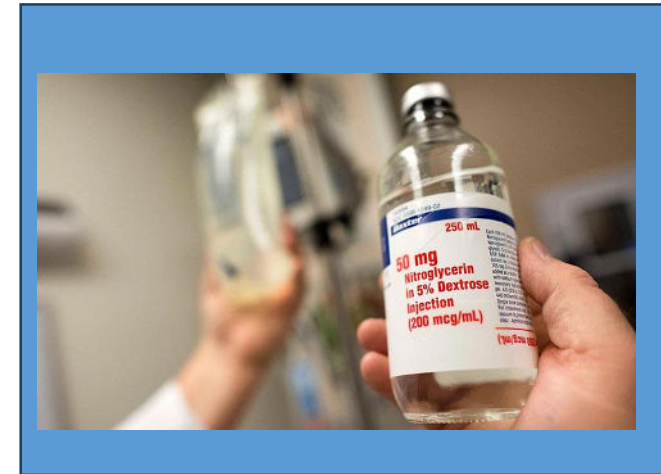
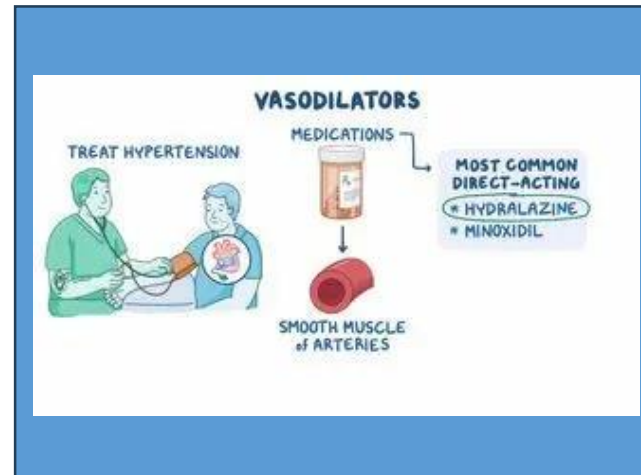


- Went to the Cath lab because of his excruciating pain
- Had normal Coronary Arteries
- Ordered echo before going to cath as MD's typical protocol
- Echo tech called me and said something is significantly wrong with his AORTA (Aortic Root 4.5cm and Proximal Ascending aorta is 5.2 cm with pericardial effusion)
- Sent him for a CT angio dissection protocol of his chest
- Radiologist immediately called to say: Type A dissection of the ascending thoracic aorta and aortic arch; moderate sized pericardial effusion
- **PATIENT STILL HAVING CHEST PAIN BUT NOW MOVING TO HIS BACK**

When there is a dissection, one can see a FLAP... such as in these images.

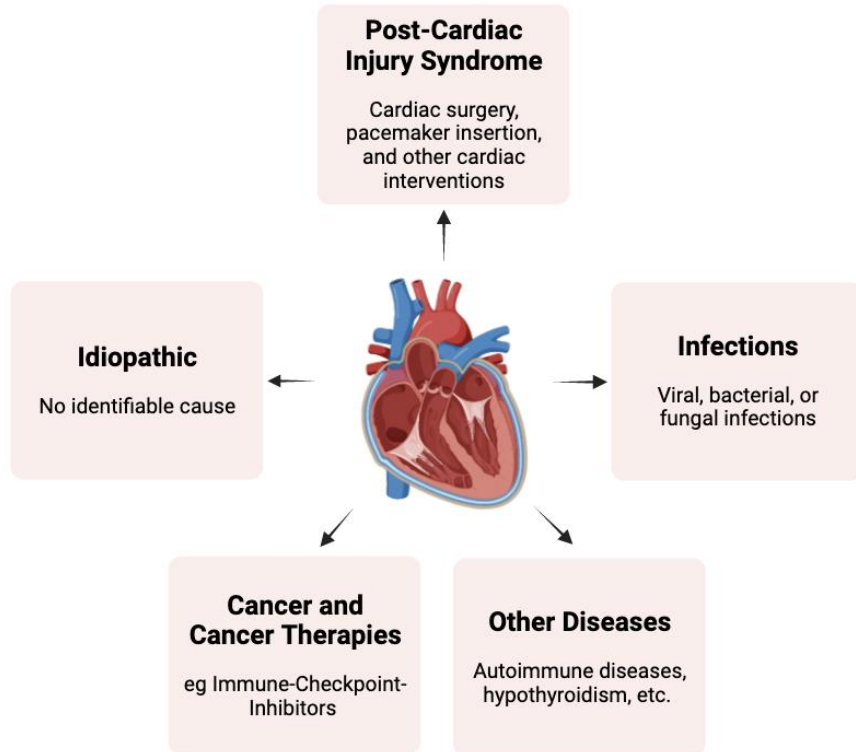


- Stat CT surgery consult and into surgery with in the hour
- Lengthy hospitalization with acute respiratory failure, acute kidney injury, neurologic changes but able enough to bite through his ET tube three different times; PEG and trach placed but ultimately discharged to rehab
- Overall was treated with beta blockers and vasodilators to keep blood pressure in control



Pericarditis

Causes of Pericarditis



Recent Cold?

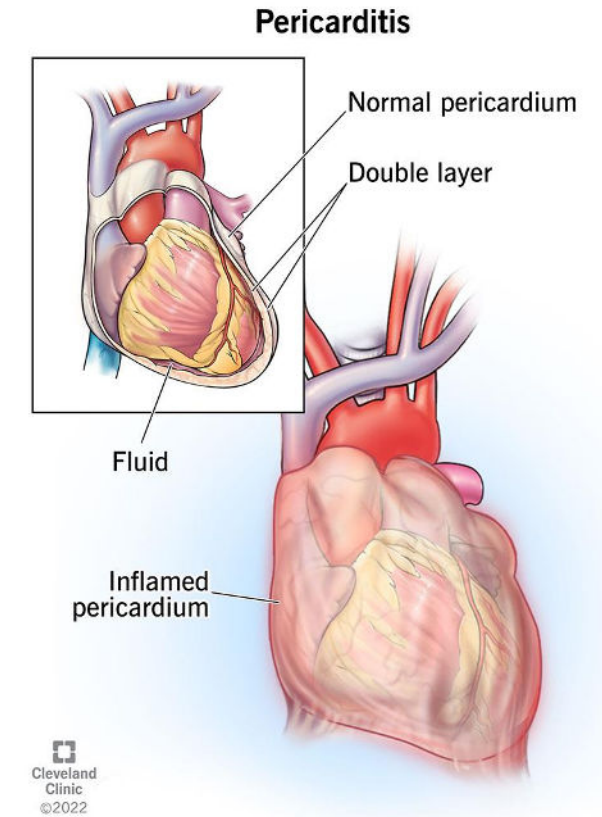
Recent Surgery?

Recent Surgery?

Table 3. Differential Diagnosis of Acute Pericarditis

Common	Less common
Angina pectoris	Esophageal spasm
Esophagitis	Pulmonary embolism
Gastritis (acute)	Tension pneumothorax
Gastroesophageal reflux disease	Rare
Myocardial infarction	Aortic dissection
Myocardial ischemia	Esophageal rupture
Peptic ulcer disease	
Pleuritis	
Pneumonia	

Information from reference 11.



Affects EKG

Typical ER Work Up

Diagnosis of Pericarditis



Abnormal ECG Findings



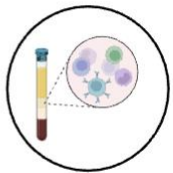
Pericardial Effusion



Imaging: CT scan, cardiac MRI, or Echo



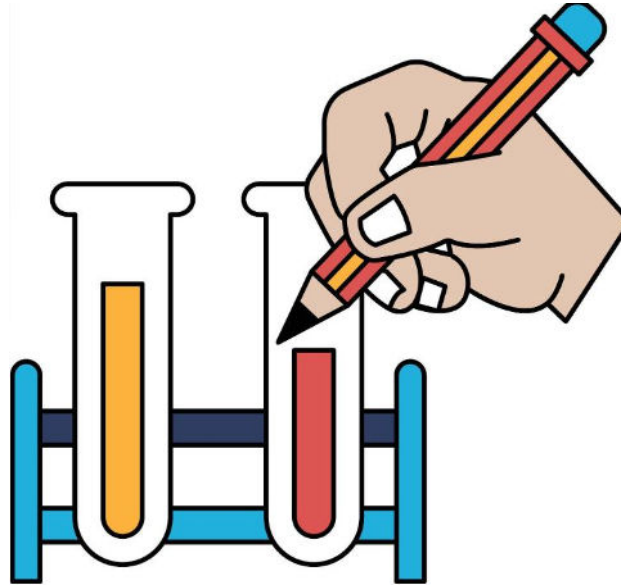
Chest Pain



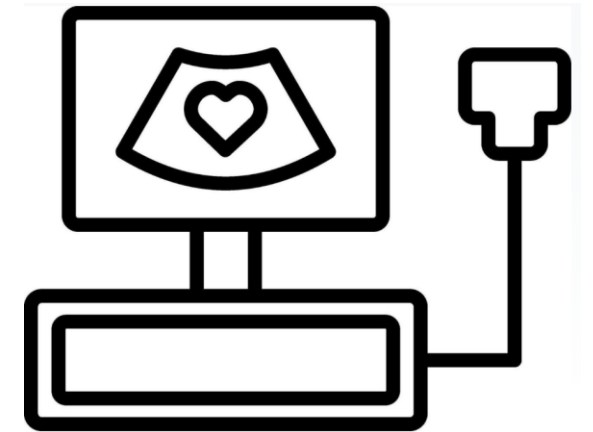
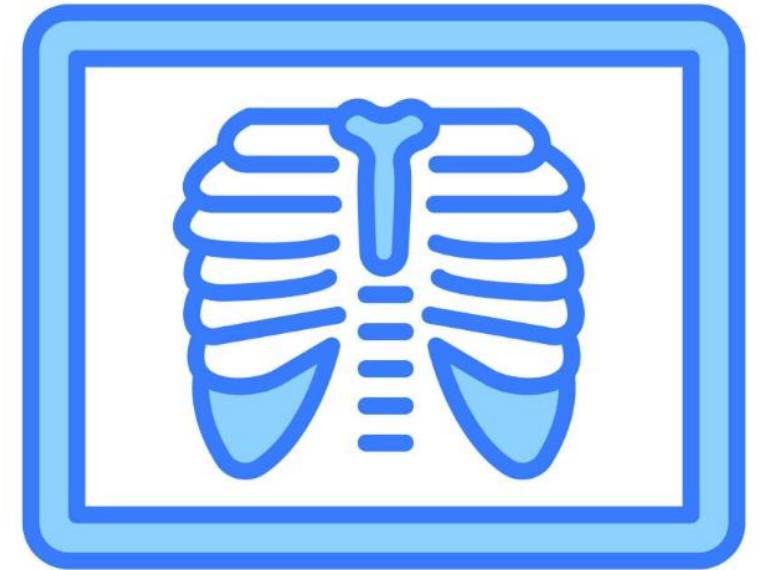
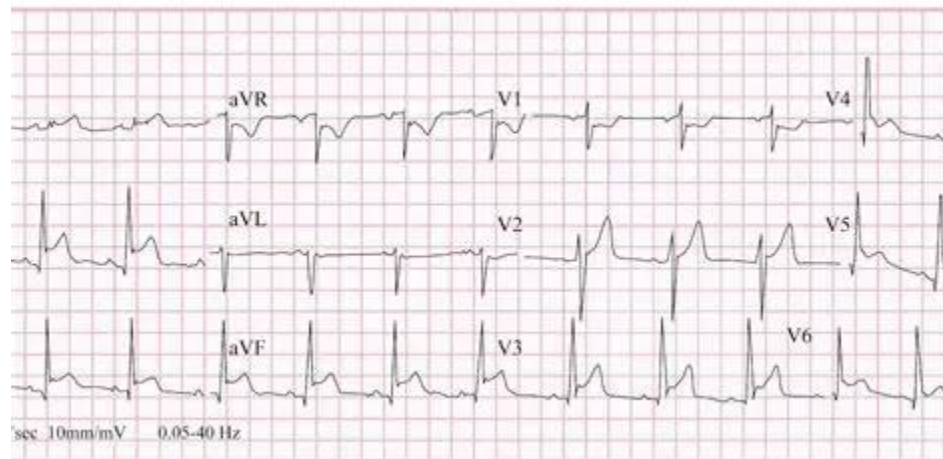
Elevated inflammatory markers

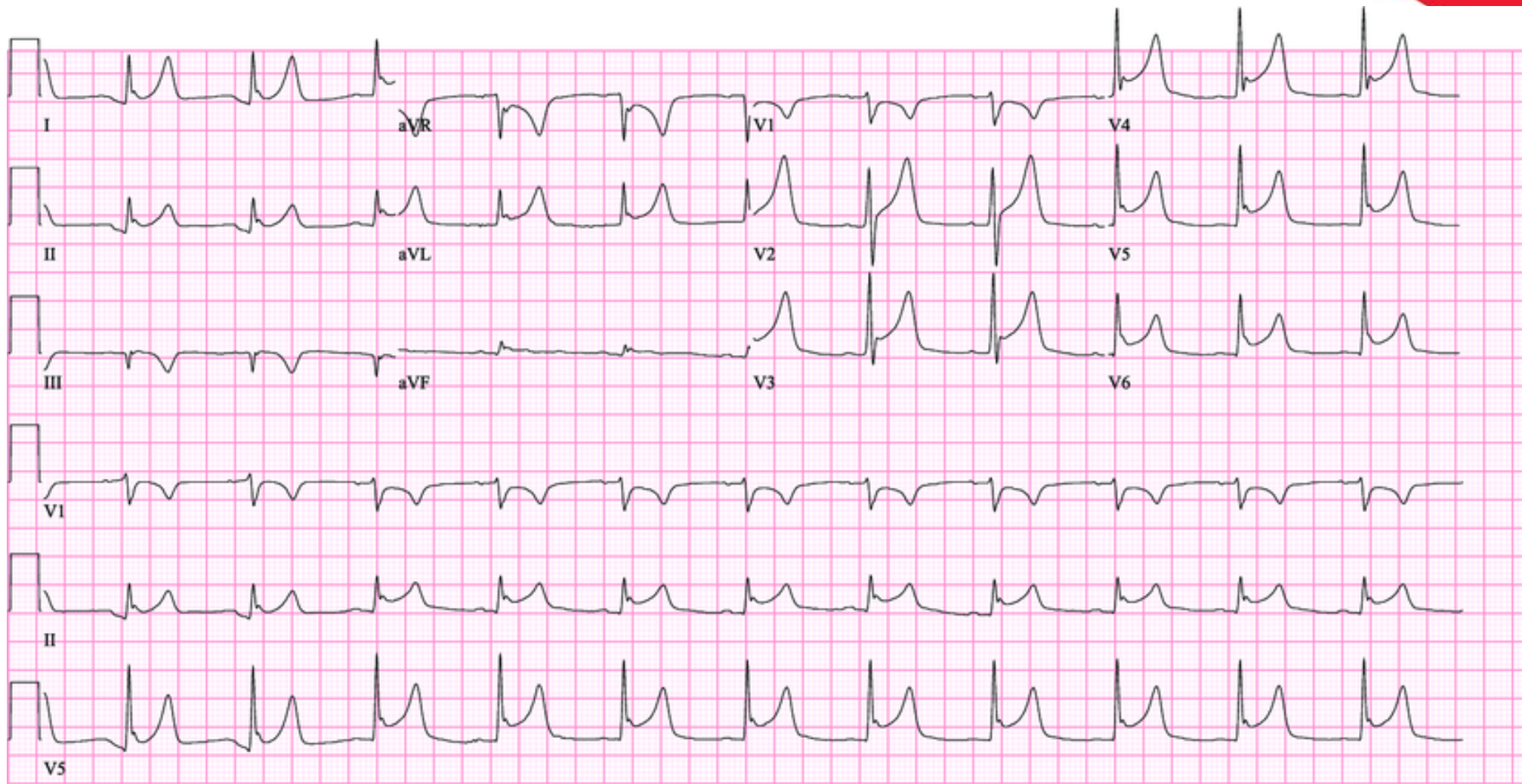


Pericardial Rub



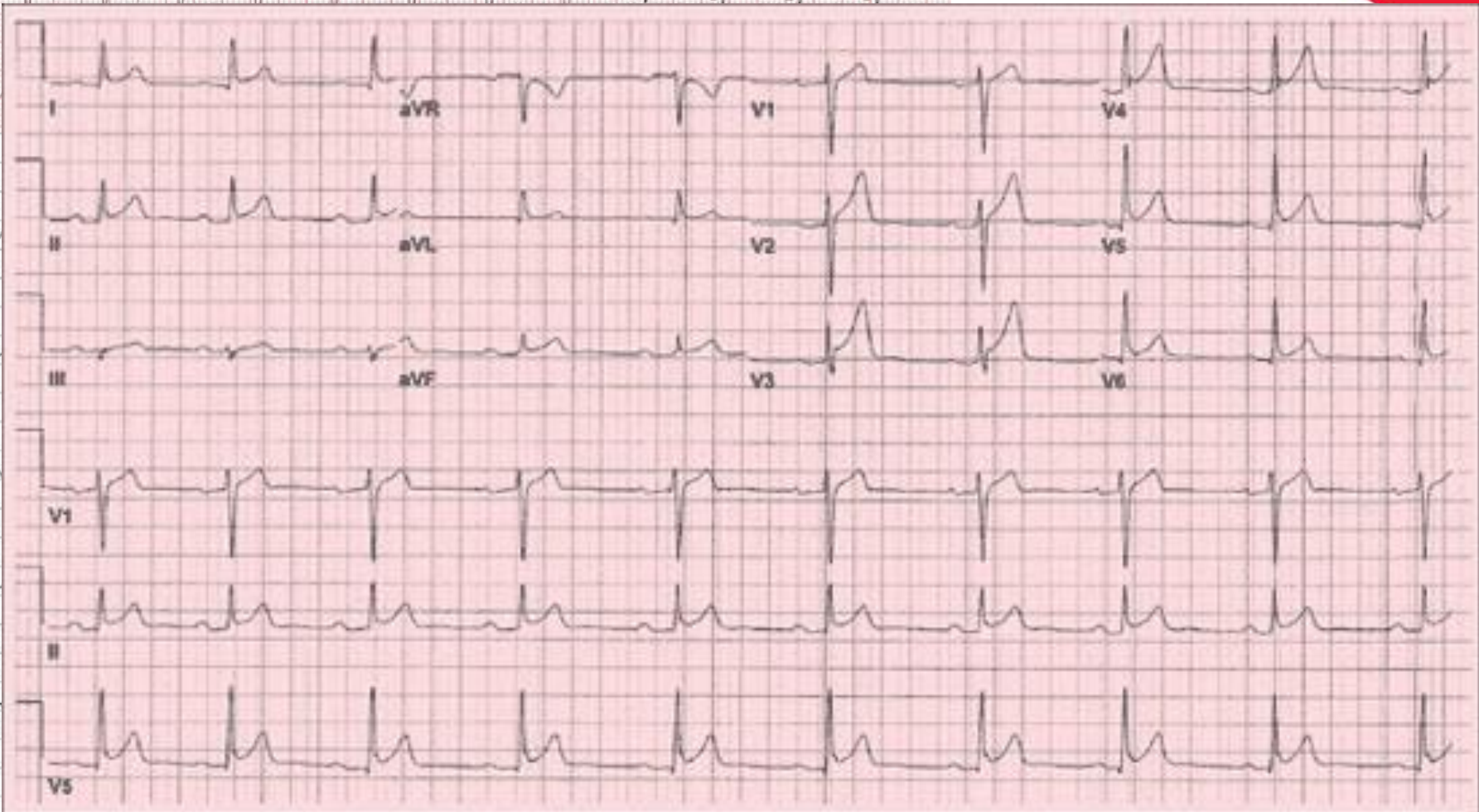
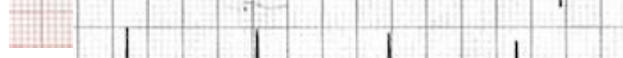
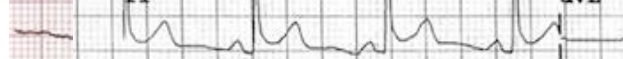
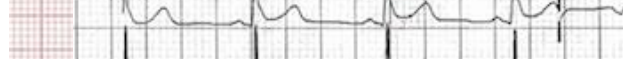
PERICARDITIS





25mm/s 10mm/mV 40Hz

Case Scenario Discussion



Dev: 2476

Speed: 25 mm/sec

Limb: 10 mm/mV

Chest: 10.0 mm/mV

F 60~ 0.50- 40 Hz W

PH090A

P?



**Skin, Bones,
Hearts &
Private Parts**

Bryan
HEART

Typical Treatment of Pericarditis

Management for Pericarditis



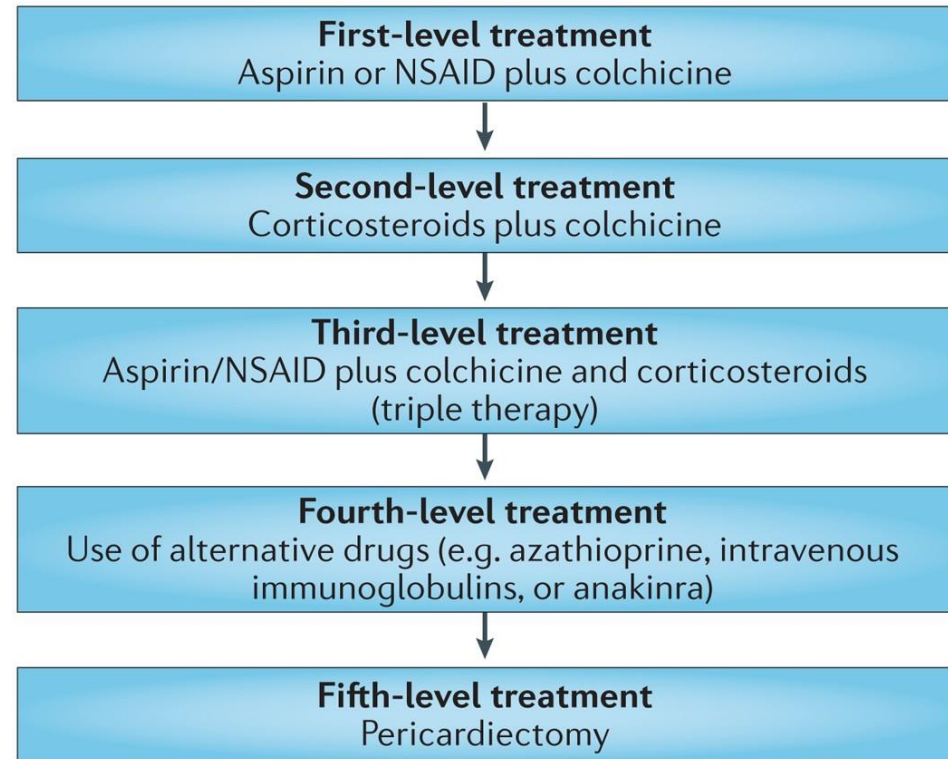
High-dose aspirin or NSAIDs may be given for ~2 weeks as well as GI protective medication.



Colchicine, an anti-inflammatory medication, is recommended to reduce symptoms and recurrence.



Imaging tests that may be used to monitor pericardial disease such as chest x-ray, echocardiography, CT, cardiac MRI, and PET scans.













Nature Reviews | Cardiology



Acute Heart Failure

Heart Attack vs. Heart Failure

Heart Attack: An Emergency	VS.	Heart Failure: A Chronic Condition
Blood Flow to the Heart Is Blocked 		The Heart Cannot Pump Blood Effectively 
Often Develops Suddenly 		Generally Develops Gradually 
Chest Pain or Pressure Can Be Intense 		Chest Pain or Pressure Generally Mild 
Jaw or Back Pain 		Shortness of Breath 
Cold Sweat 		Lower-Body Swelling 

EVERYDAY HEALTH

JOURNAL ARTICLE

Chest pain in patients with heart failure: why history may matter [Get access >](#)

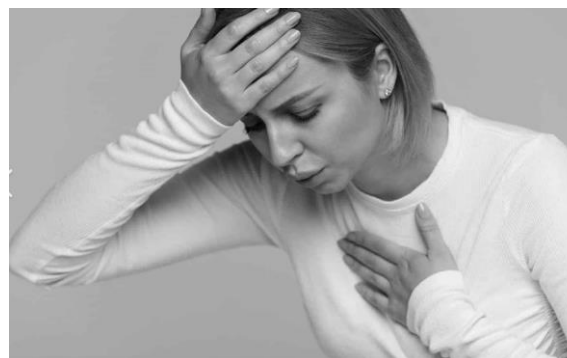
Adam D. DeVore, Adrian F. Hernandez ✉

European Heart Journal, Volume 35, Issue 48, 21 December 2014, Pages 3408–3409,
<https://doi.org/10.1093/eurheartj/ehu408>

Published: 21 October 2014



Chest pain can be a symptom of heart failure, often caused by reduced blood flow to the heart or fluid buildup in the lungs, and may present as pressure, tightness, or discomfort.



[Chest pain in patients with heart failure: why history may matter | European Heart Journal | Oxford Academic](#)



Evaluation/Diagnosis

Table 3. History and Physical Examination Findings for Heart Failure and Selected Alternative Causes

Heart failure

Symptoms

- Abdominal swelling
- Dyspnea on exertion
- Edema
- Exercise intolerance
- Fatigue
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Recent weight gain

Physical examination findings

- Abdomen: hepatjugular reflux, ascites
- Extremities: cool, dependent edema
- Heart: bradycardia/tachycardia, laterally displaced point of maximal impulse, third heart sound (gallop or murmur)
- Lungs: labored breathing, rales
- Neck: elevated jugular venous pressure
- Skin: cyanosis, pallor

Alternative causes

Symptoms

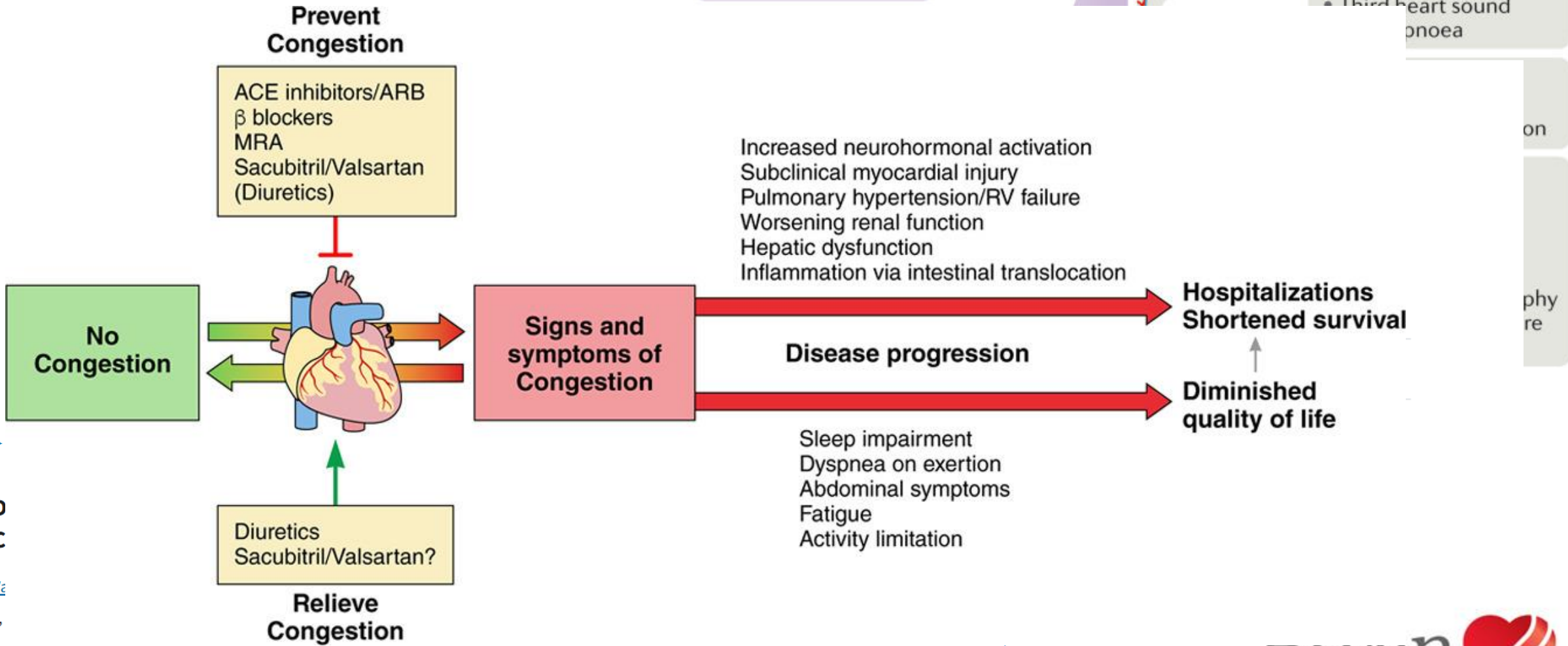
- Abdominal swelling (liver failure)
- Anorexia, weight loss (sarcoidosis)
- Chest pain (coronary artery disease)
- Claudication (atherosclerotic disease)
- Cough (pulmonary disease)
- Diarrhea or skin lesions (amyloidosis)
- Dyspnea on exertion (pulmonary disease, valvular disease)
- Edema (liver or kidney failure)
- Neurologic problems (sarcoidosis)
- Palpitations (tachyarrhythmia)
- Recent fevers, viral infection (endocarditis, myocarditis, infection)
- Syncope (bradycardia, heart block)

Physical examination findings

- Abdomen: distended, hepatosplenomegaly, tender, ascites (liver disease)
- Extremities: joint inflammation/warmth (rheumatologic disease)
- Heart: irregular rate or rhythm (arrhythmia)
- Lungs: wheezing (pulmonary disease)
- Neck: thyromegaly/nodule (thyroid disease)
- Skin: cyanosis (anemia), jaundice (liver failure)



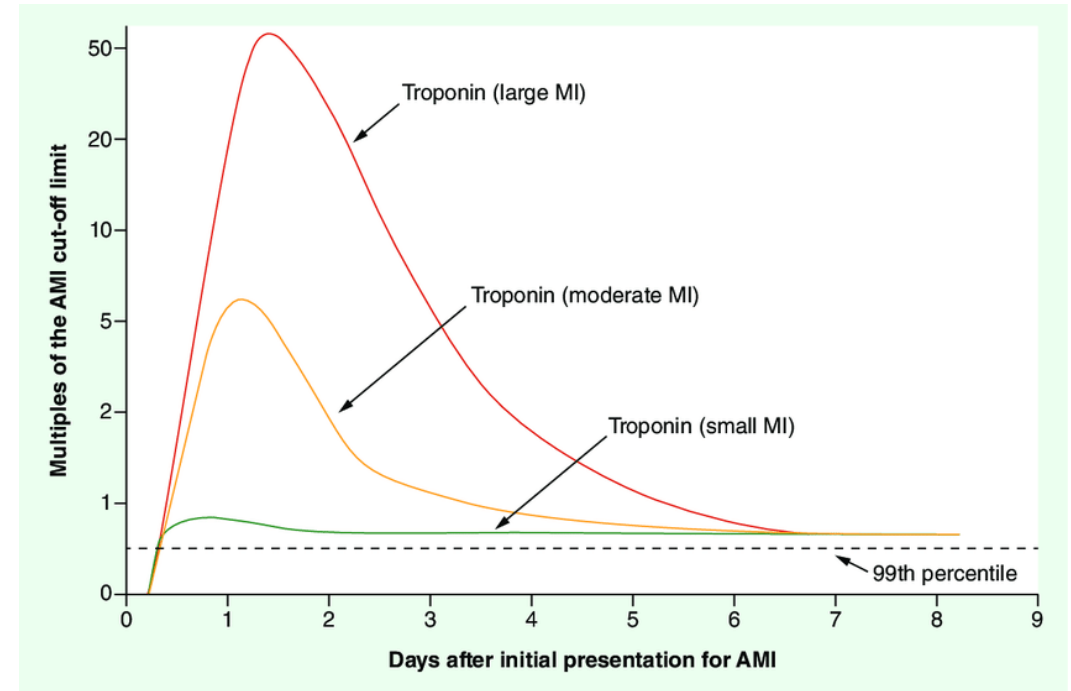
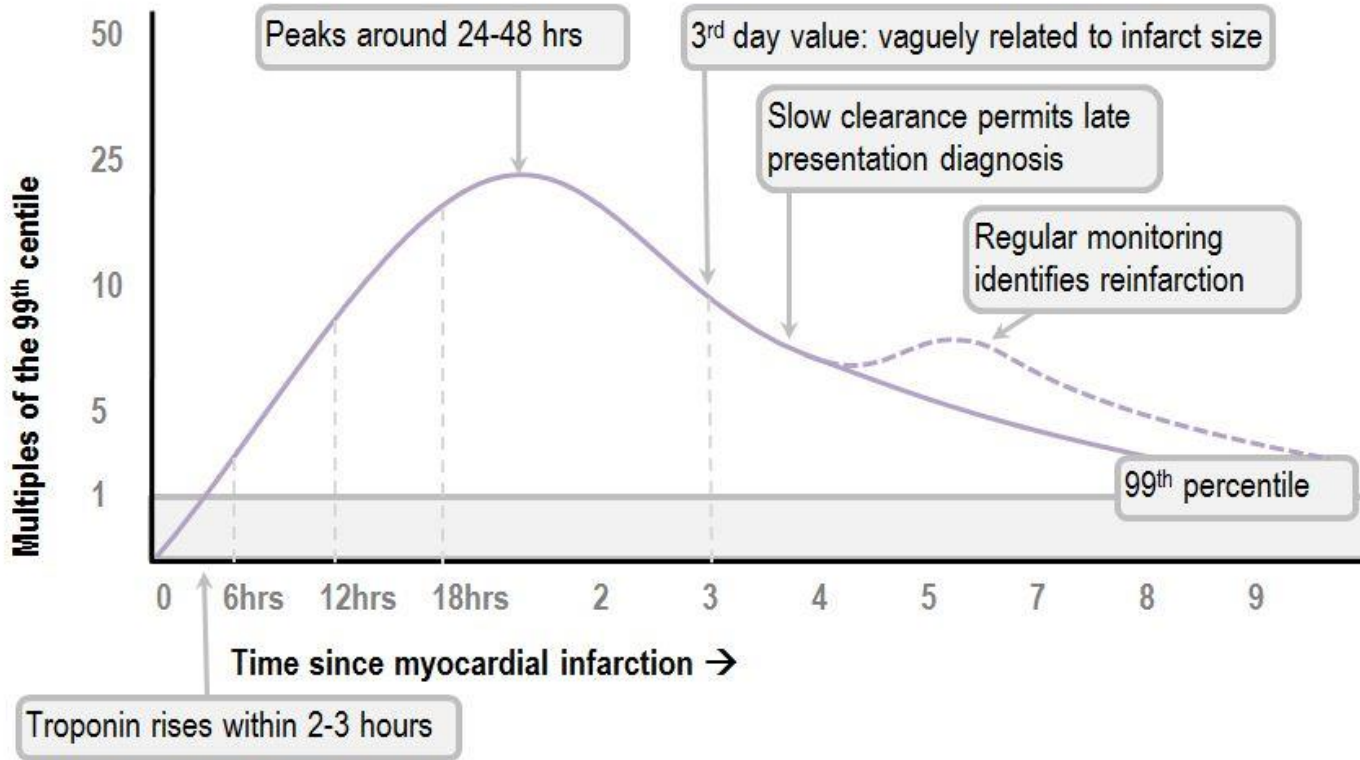
Treatment



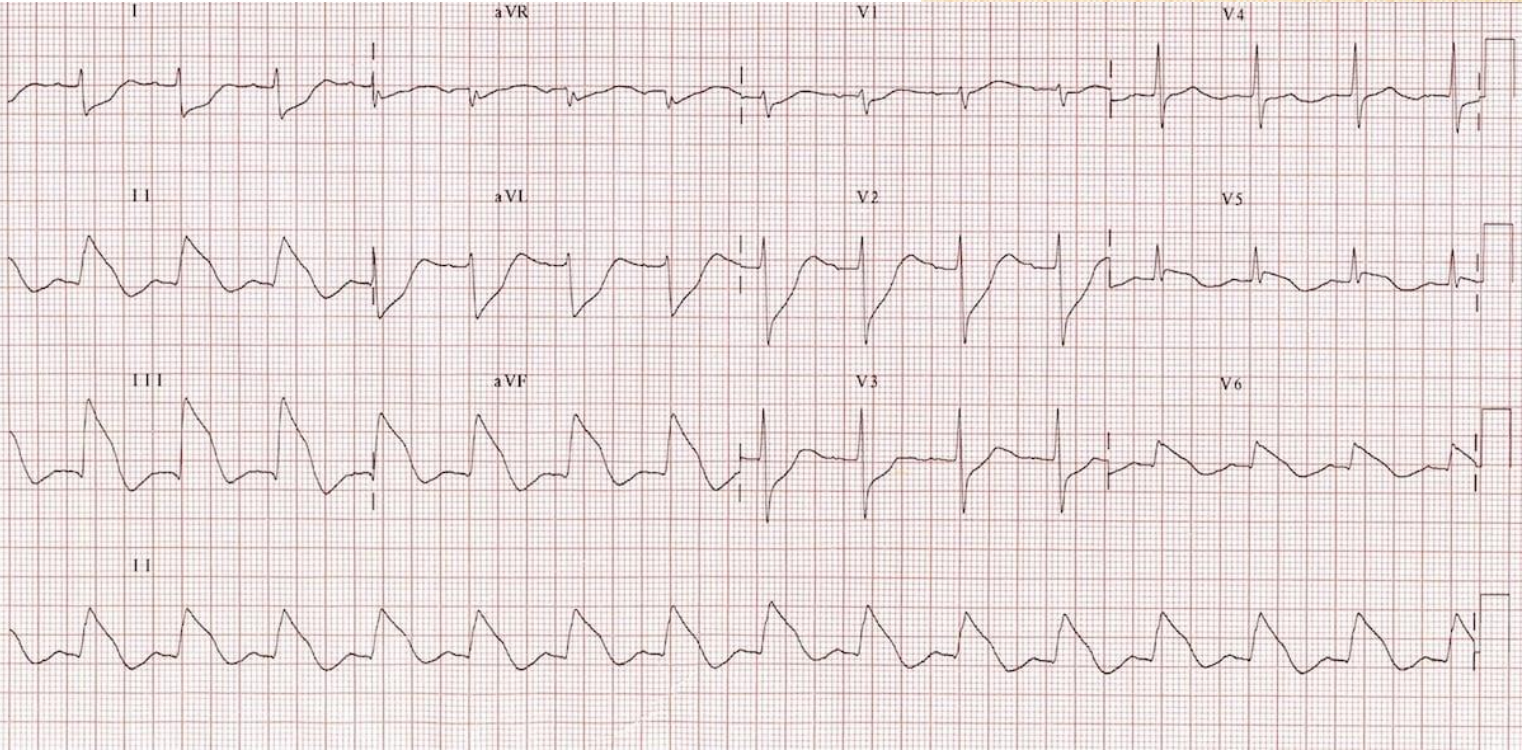
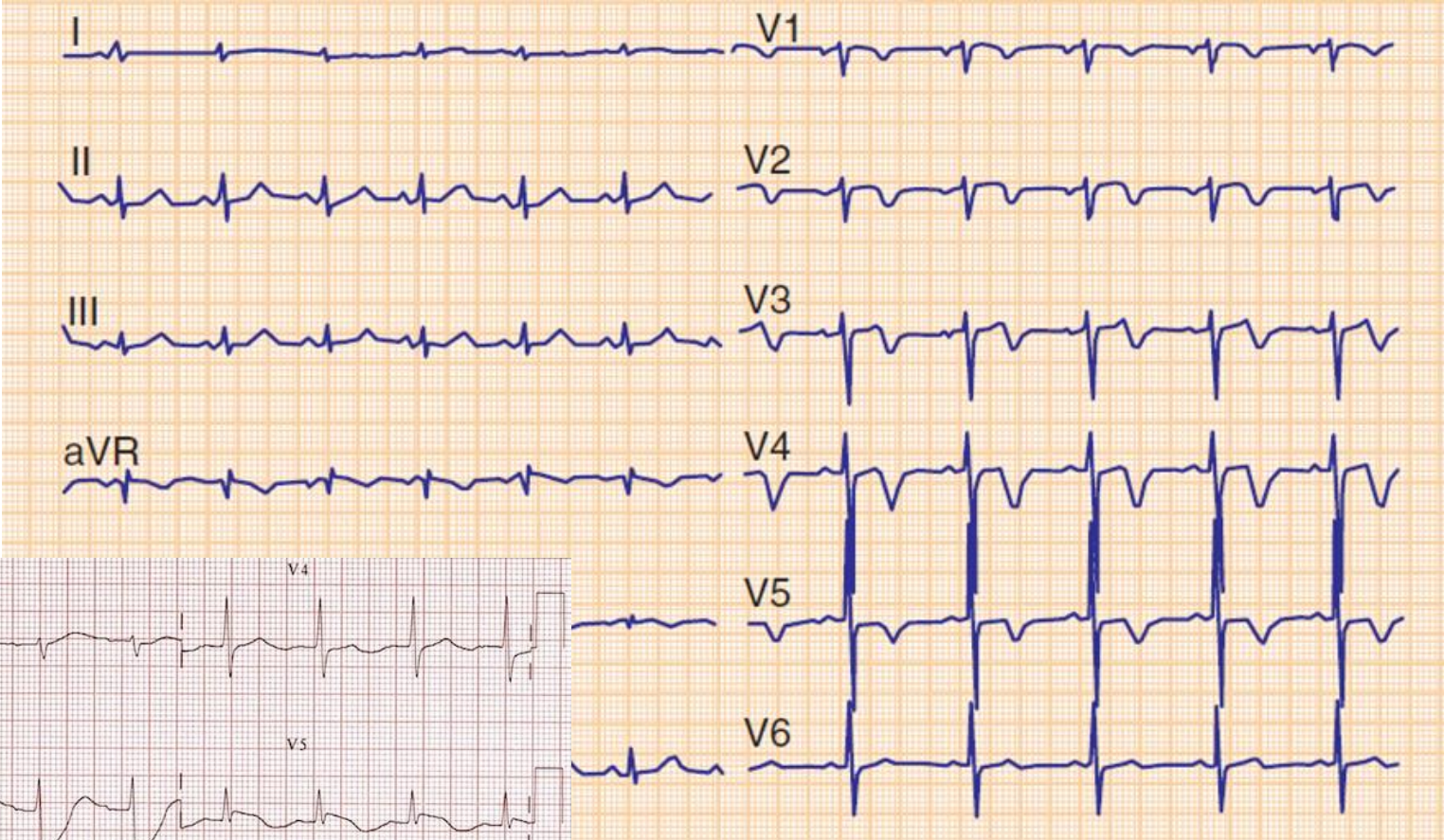
[American College of Cardiology Solution Set Oversight Committee | JACC](#)



Non-ST Elevated Myocardial Infarction



The EKG



UNSTABLE
ANGINA

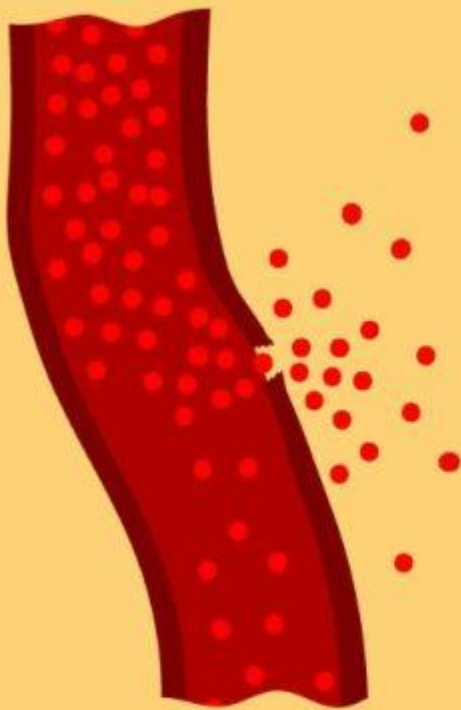
vs

NSTEMI

vs

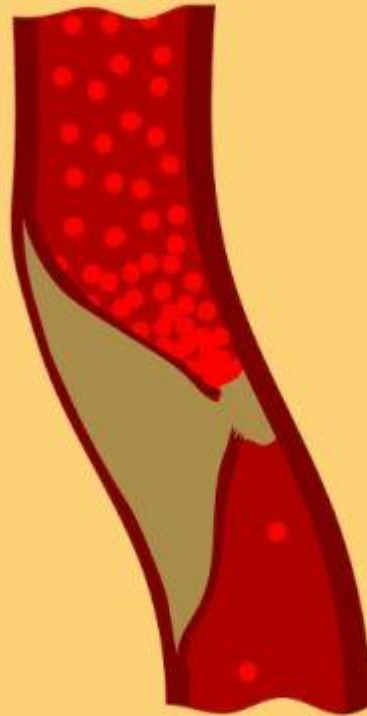
STEMI

Types of Acute Coronary Syndrome



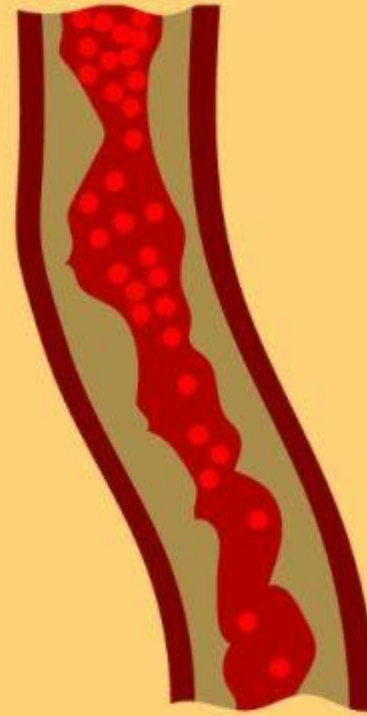
Unstable angina

- partial rupture of an artery
- does not cause permanent damage to the heart



STEMI

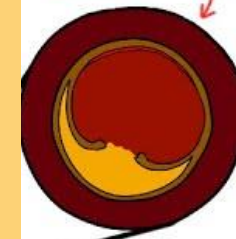
- "classic" heart attack
- causes extensive heart damage



NSTEMI

- intermediate form of ACS
- causes less extensive damage to the heart

COMPLETE
OCCLUSION

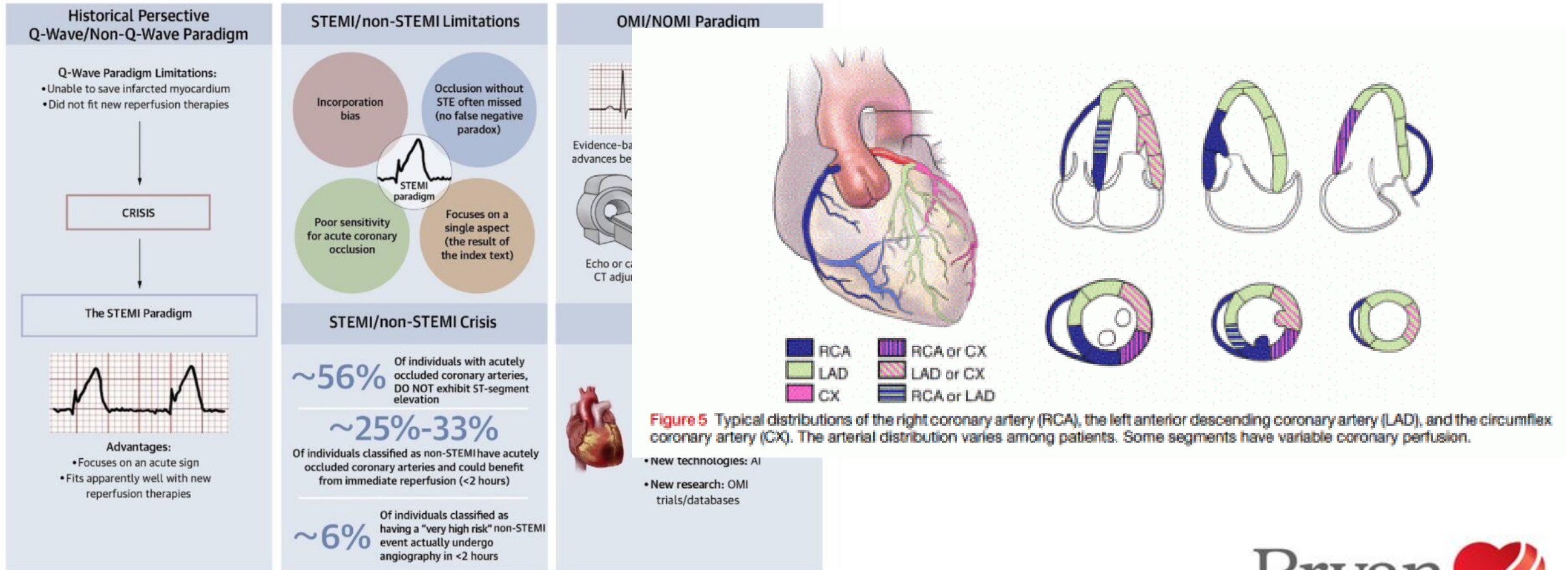


TRANSMURAL
NECROSIS



The Echo

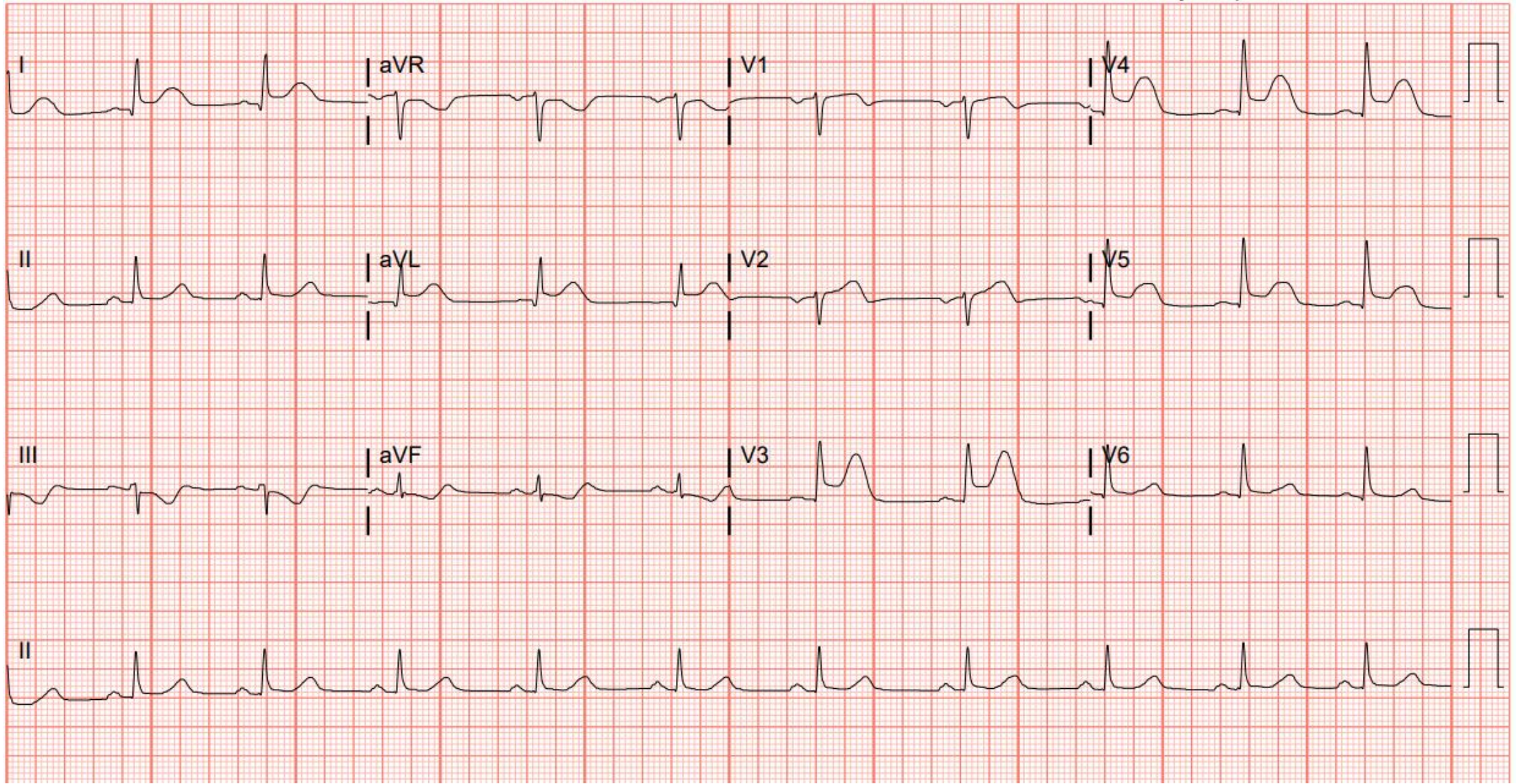
CENTRAL ILLUSTRATION: From ST-Segment Elevation MI to occlusion MI



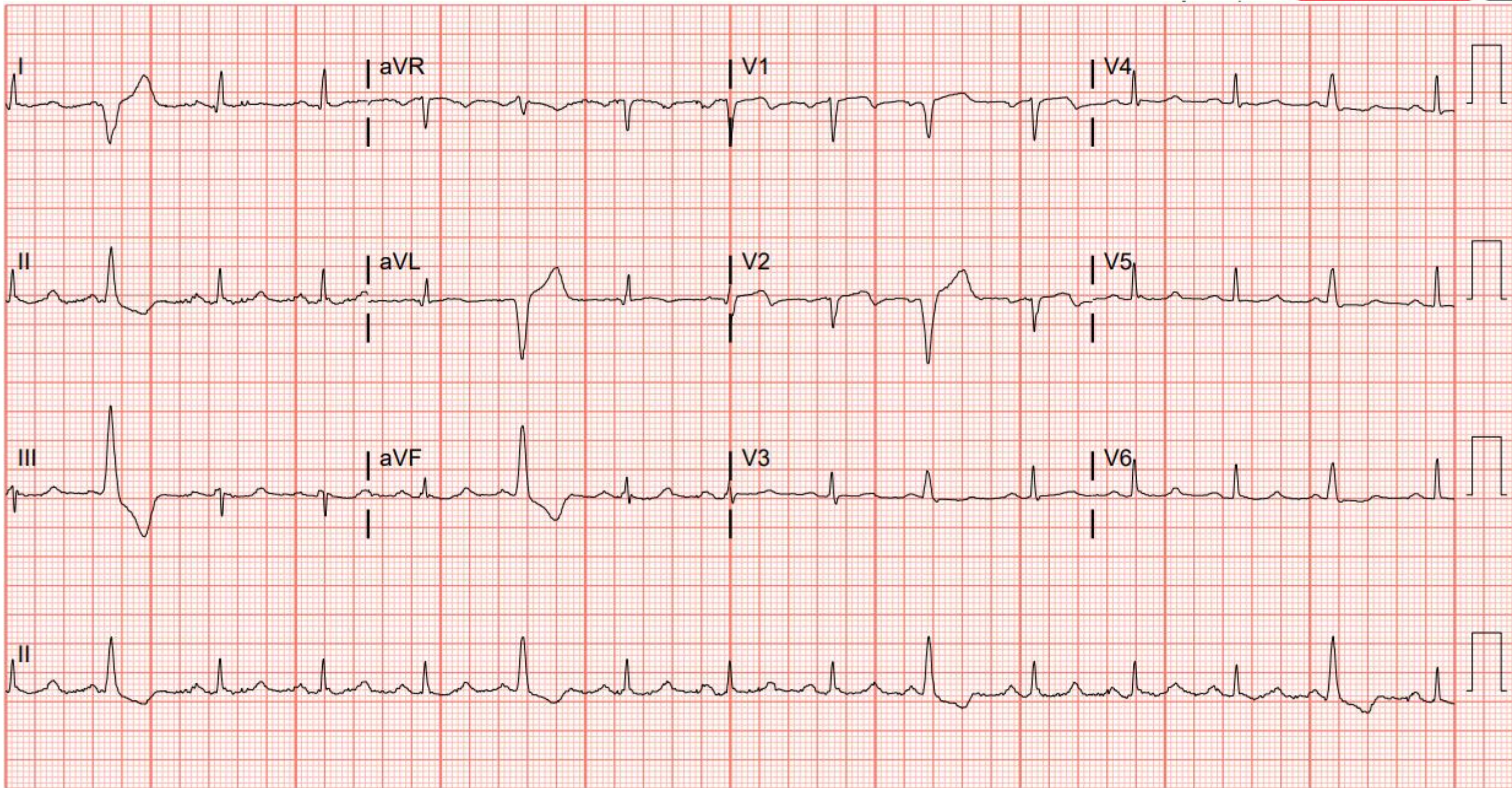
McLaren J, et al. JACC Adv. 2024;3(11):101314.



Aspect	STEMI Prevention	NSTEMI Prevention
Blood Pressure Control	Aggressive blood pressure control. It helps to prevent coronary artery disease and subsequent myocardial infarction.	Blood pressure management is essential to prevent myocardial ischemia. It is vital for reducing the risk of cardiovascular events, too.
Smoking Cessation	Such programs and support mitigate the risk of atherosclerosis and thrombotic events.	Smoking cessation is a crucial component of prevention strategies. They aim to prevent cardiovascular disease.
Physical Activity	Encouragement of regular physical activity to improve cardiovascular health. Also, it helps to prevent atherosclerosis.	Regular exercise aims to improve cardiac function and reduce the risk of myocardial infarction.
Healthy Diet	Promotion of a diet rich in fruits, vegetables, and whole grains. Lean proteins to maintain cardiovascular health.	Dietary modifications include reducing saturated fats and cholesterol intake. They are essential for preventing atherosclerosis and myocardial infarction.



Case #1: KR



Treatment Management

- CAD – treat medically
- CAD- differences between platelet inhibition
- Follow up Recommendations

ST Elevated Myocardial Infarction

- Difference between STEMI vs NSTEMI
- Labs (Troponin, CRP, Enzymes)
- Purpose of EKG
- Purpose of Echo
- Purpose of Trends
- Symptoms
- Urgency to Cath lab

Treatment Options

- “Cardiac Alert”
- Cath lab + Stents
- Cath lab – no stents
- Cath lab > CG Shock > devices > OR
- GOAL of door to balloon window
- Talk anti-plt, tPA indications (as they are different than PEs), Risk factor modification (BP, Lipids etc...)

Final PEARLS...

- GERD-
- PE-
- Aortic Dissection/LVH-
- Pericarditis -
- Acute Heart Failure –
- NSTEMI
- STEMI

Questions?



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- [2024 ACC Expert Consensus Decision Pathway on Clinical Assessment, Management, and Trajectory of Patients Hospitalized With Heart Failure Focused Update: A Report of the American College of Cardiology Solution Set Oversight Committee | JACC](#)
- [All about myocardial infarction: STEMI vs NSTEMI](#)
- [PMcardio Featured on 'This Week in Cardiology': The Shift from STEMI/NSTEMI to OMI](#)