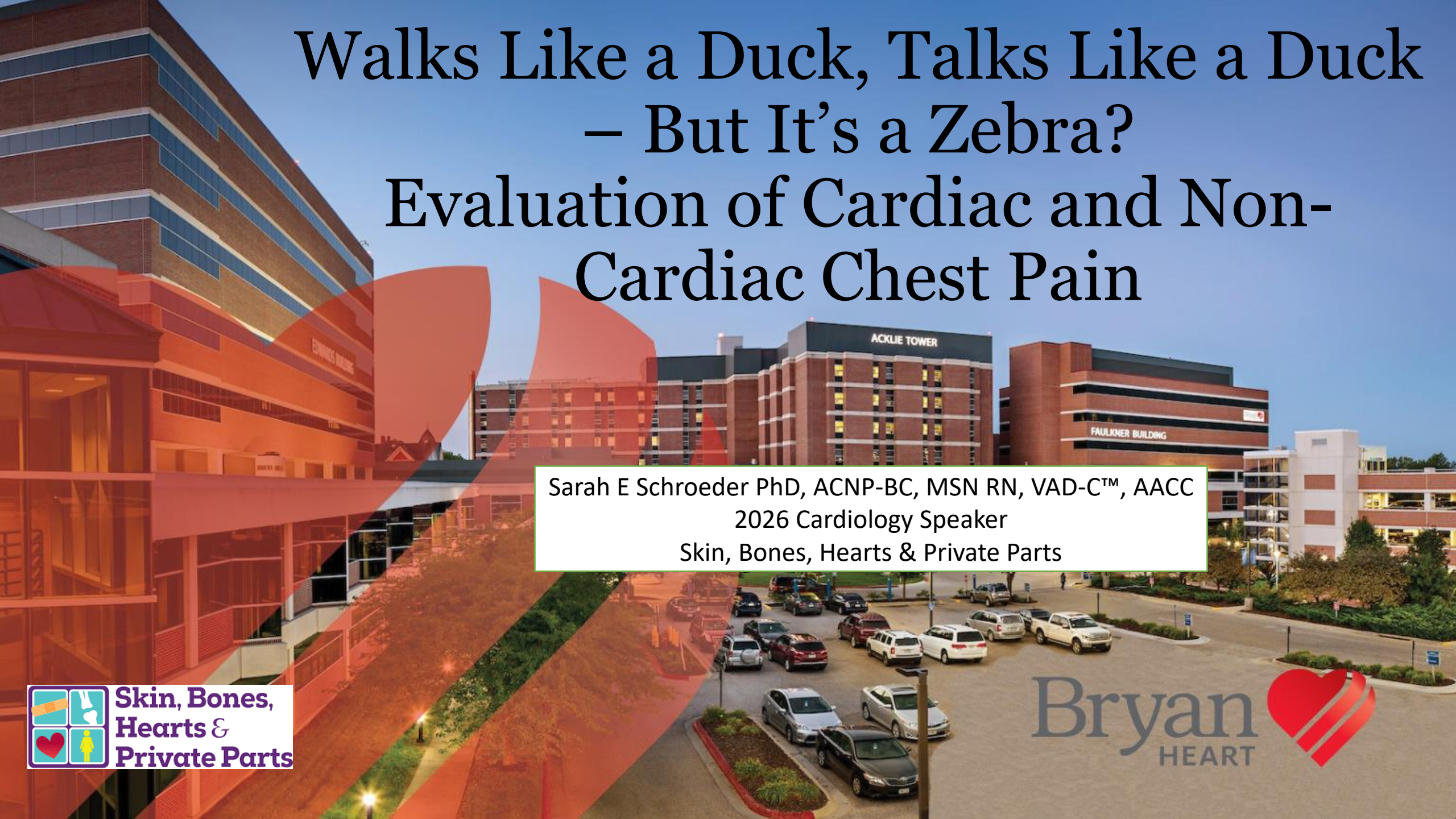
Walks Like a Duck, Talks Like a Duck – But It's a Zebra? Evaluation of Cardiac and Non-Cardiac Chest Pain



Sarah E Schroeder PhD, ACNP-BC, MSN RN, VAD-C™, AACC
2026 Cardiology Speaker
Skin, Bones, Hearts & Private Parts

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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

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I have a paid subscription to Vecteezy

Bryan HEART

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

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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

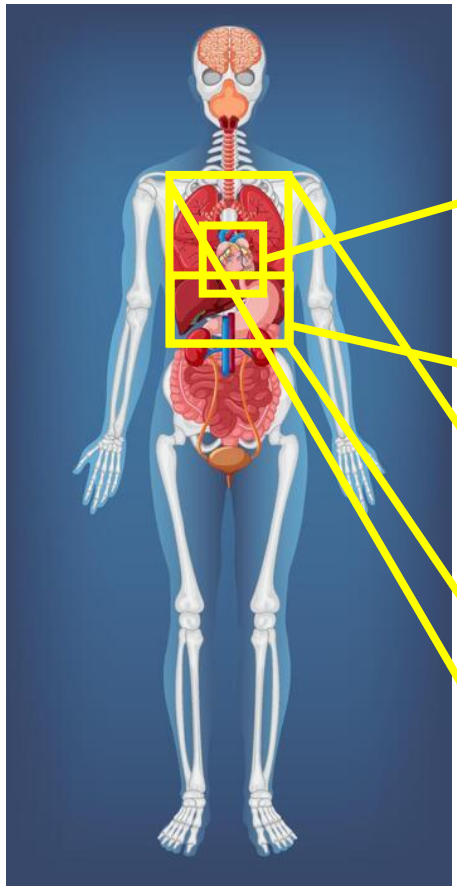
- Poor localization of pain due to lack of sensory fibers in the spinothalamic tracts
- "REFERRED PAIN"

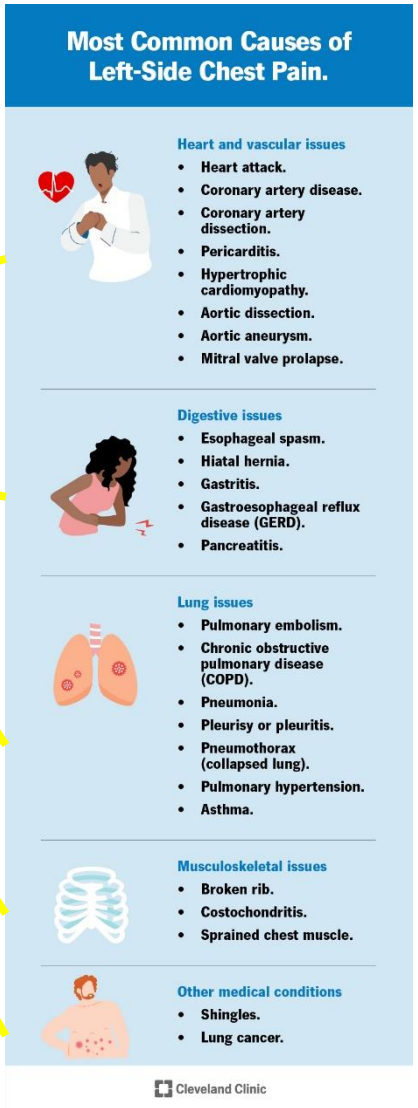


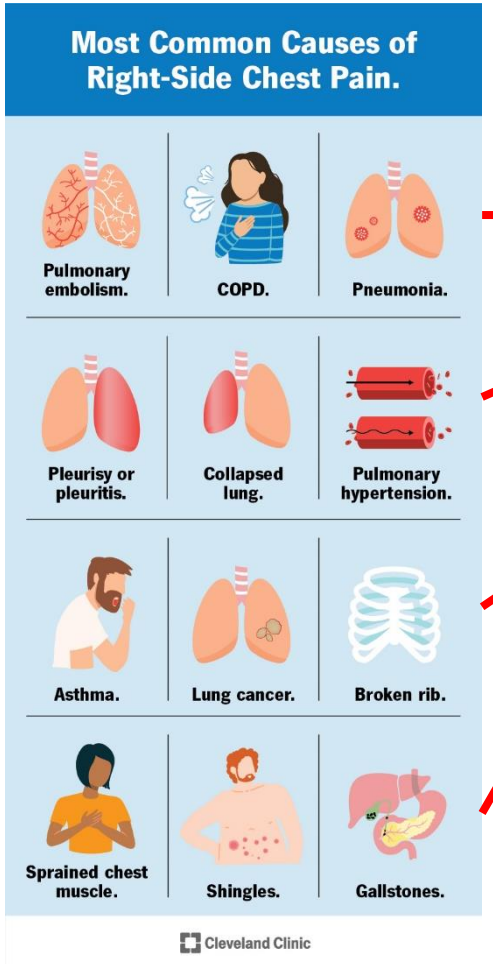
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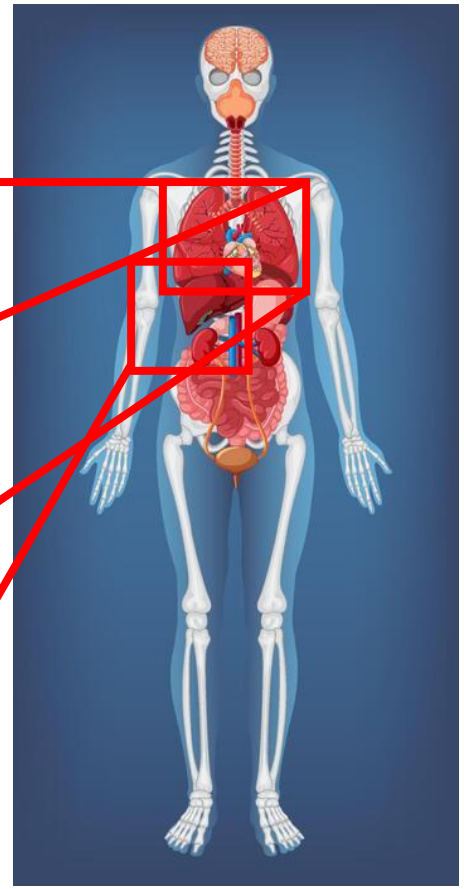
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Causes of Chest Pain









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Cleveland Clinic, 2025. What Can Cause Chest Pain on the Left Side?

Cleveland Clinic, 2025. What Can Cause Chest Pain on the Left Side?

Complex Evaluation and Treatment: Non-Ischemic Chest Pain Scenarios

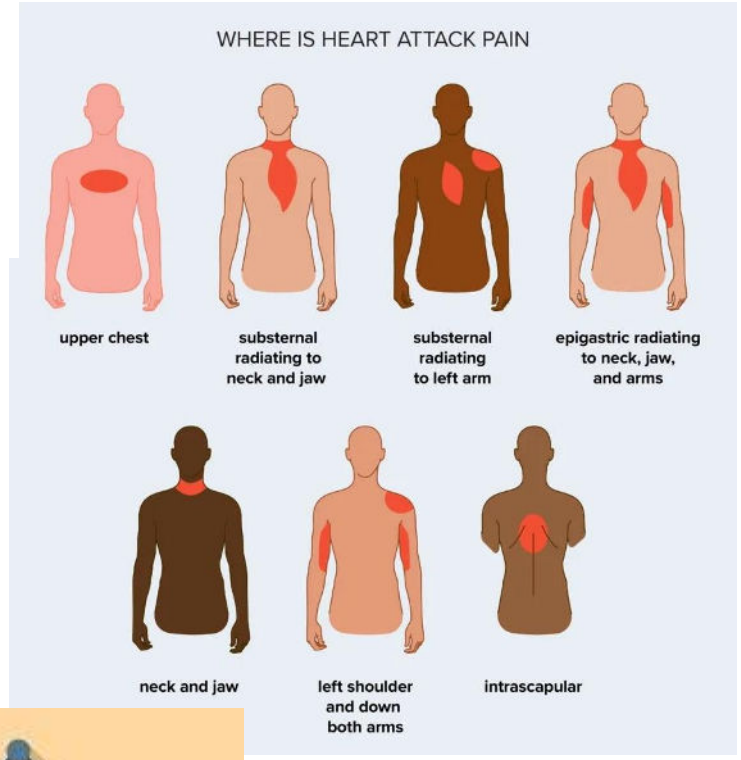
PQRST ASSESSMENT

- **P** — BETTER/WORSE
- **Q** — QUALITY OF PAIN
- **R** — REGION/LOCATION
- **S** — SEVERITY
- **T** — TIMING



PROVOKE







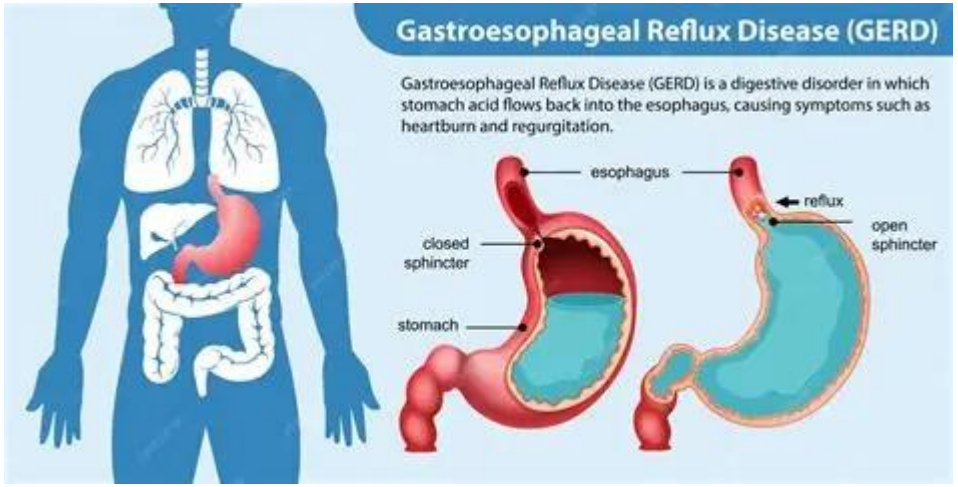


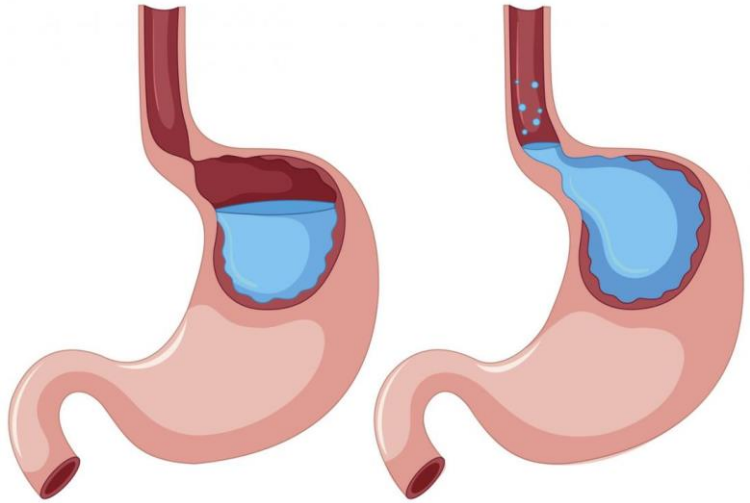
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Gastroesophageal Reflux Disease (GERD)

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

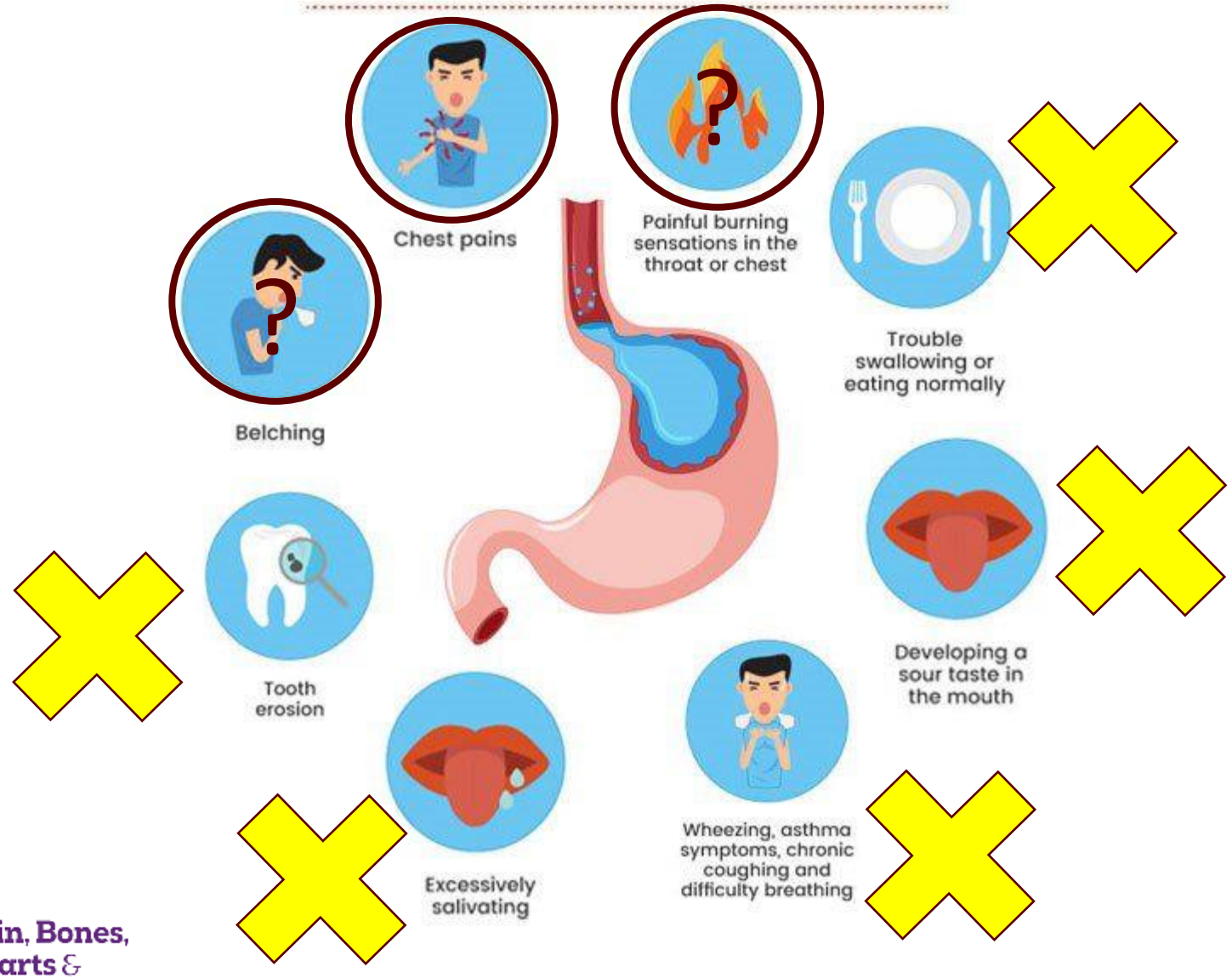




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COMMON SYMPTOMS



Best Treatments:











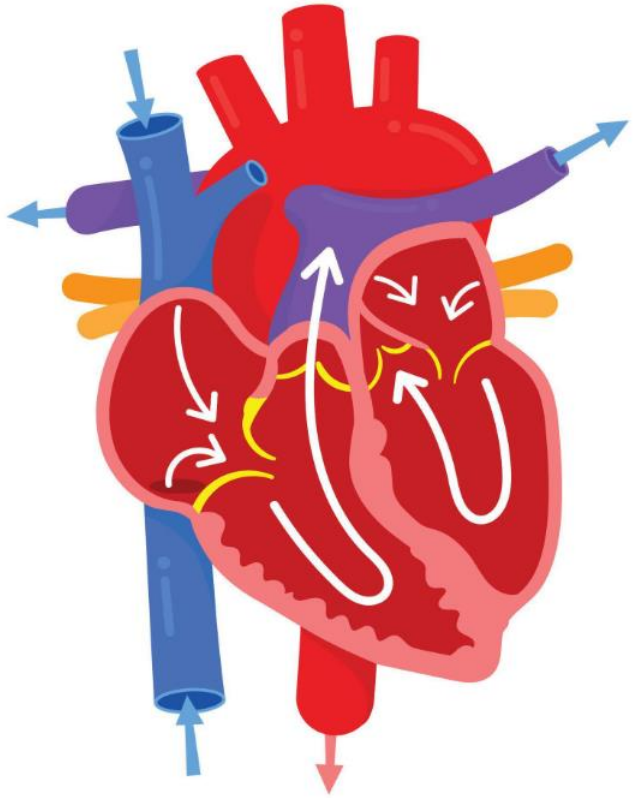


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Acute Pulmonary Embolism (PE)

- 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



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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)"

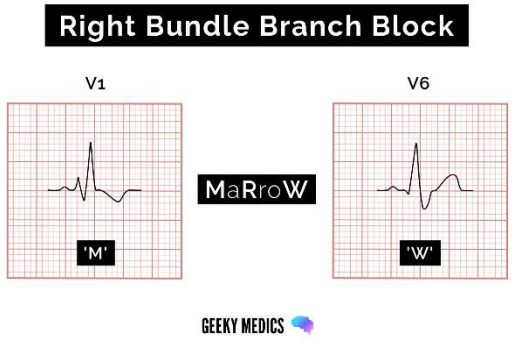
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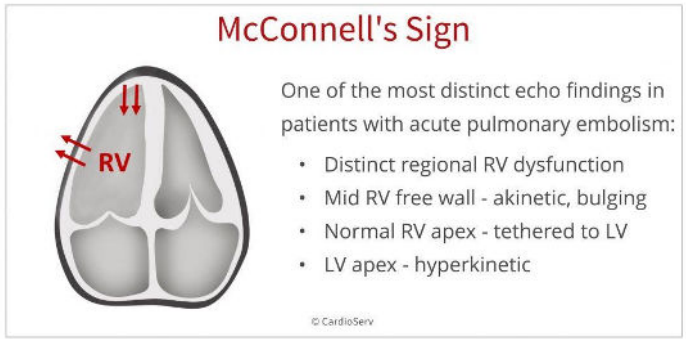
Haase et al., 2019. BMJ doi: https://doi.org/10.1136/bmj.l1945
Mirza & Hashmi, 2023. StatPearls. Lung Ventilation Perfusion Scan (VQ Scan) - StatPearls - NCBI Bookshelf

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 >/=120ms, V1-V2 rSR^1 or rsR^1 ; 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

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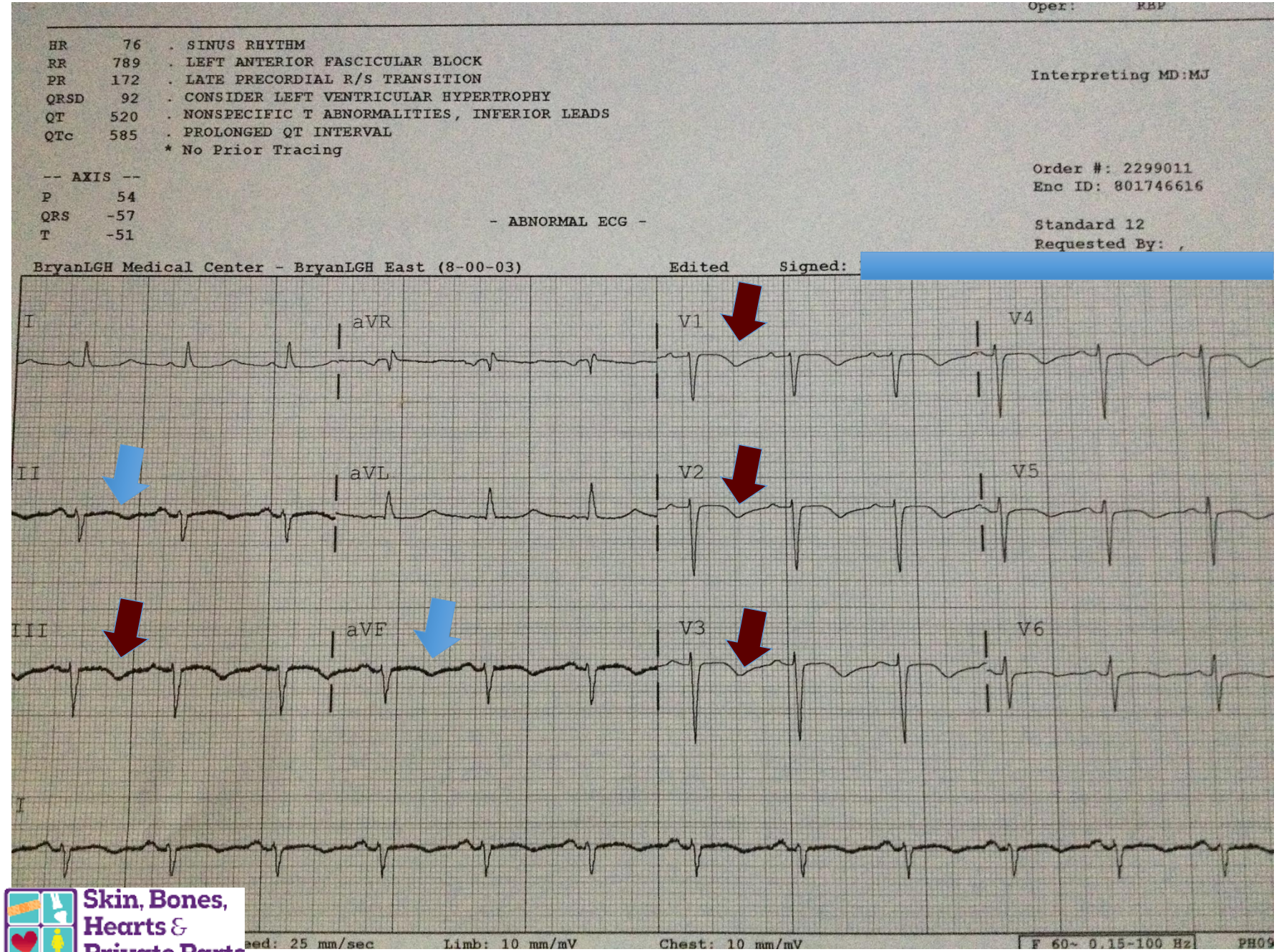
Bundle Branch Block | LBBB | RBBB | Geeky Medics

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 severly hypokinetic and dilated**, Moderate tricuspid regurgitation, **severe pulmonary hypertension (RVSP 72mmhg)**

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

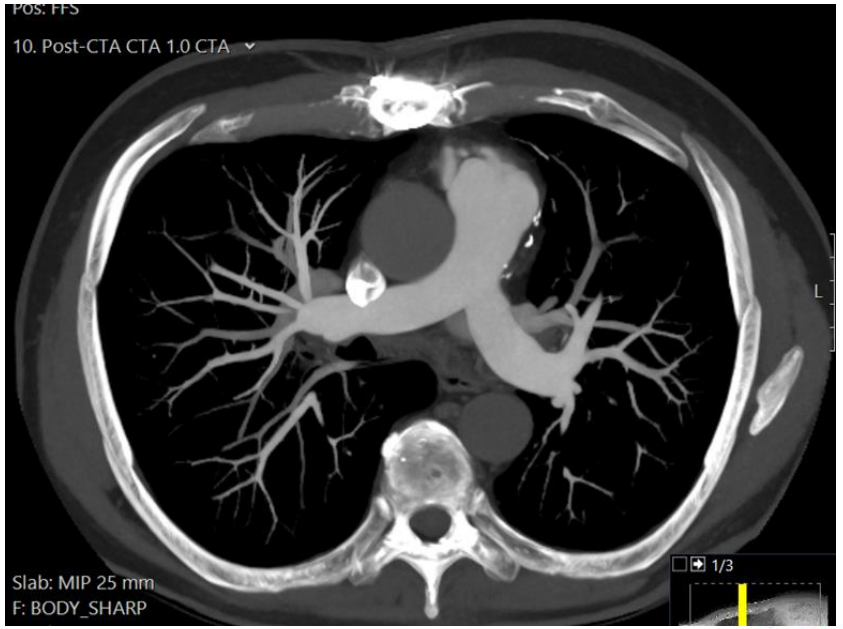
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- D-dimer came back at >20 (measures degradation of fibrinolysis).
- CT angio of chest ordered to look for pulmonary embolism.
- Lots of discussion about if she could have it because her creatinine wasn't to where radiology wanted it at (although normal).
- 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.

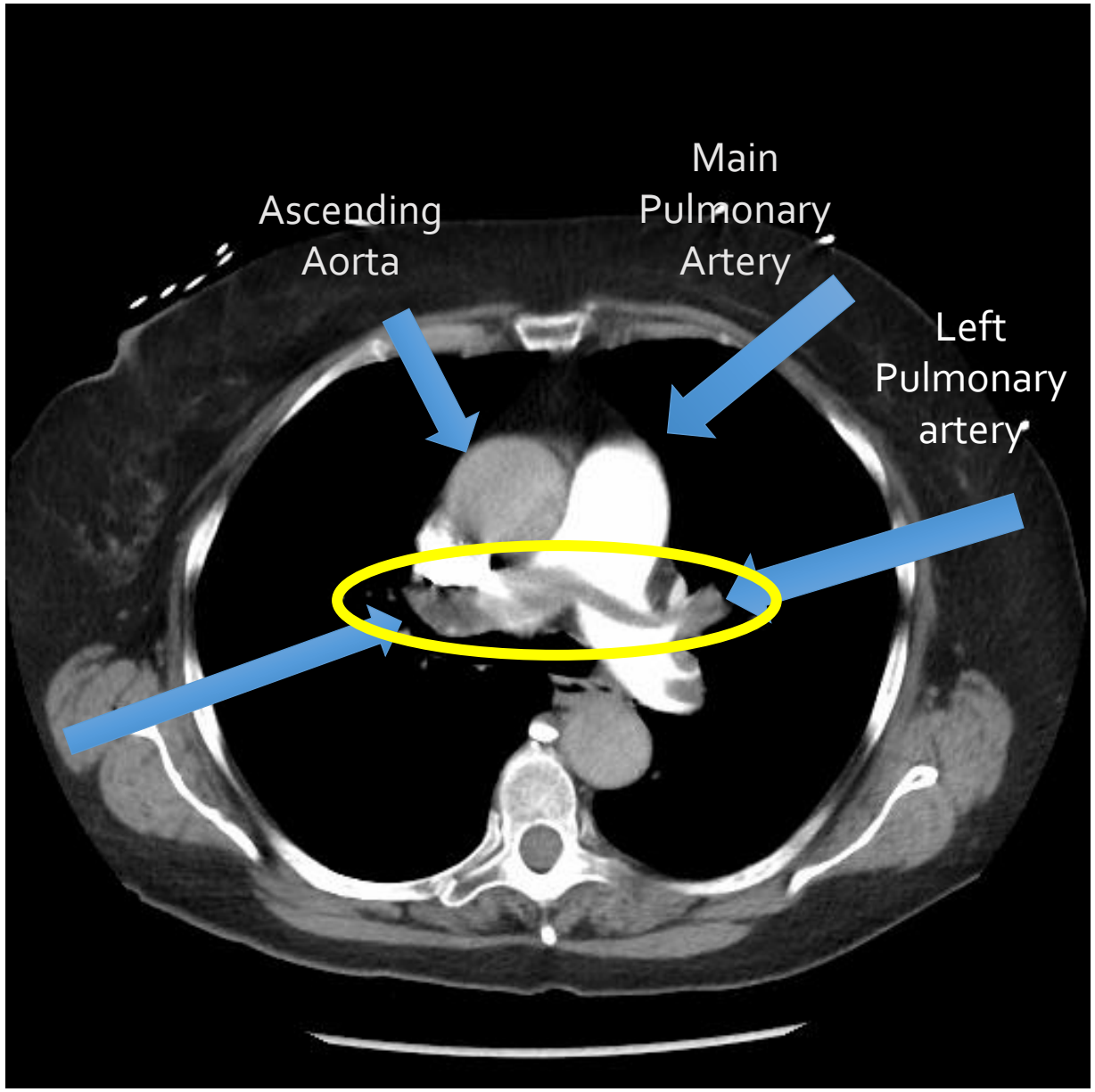
Got a call 10 minutes later that her CT Angio showed:

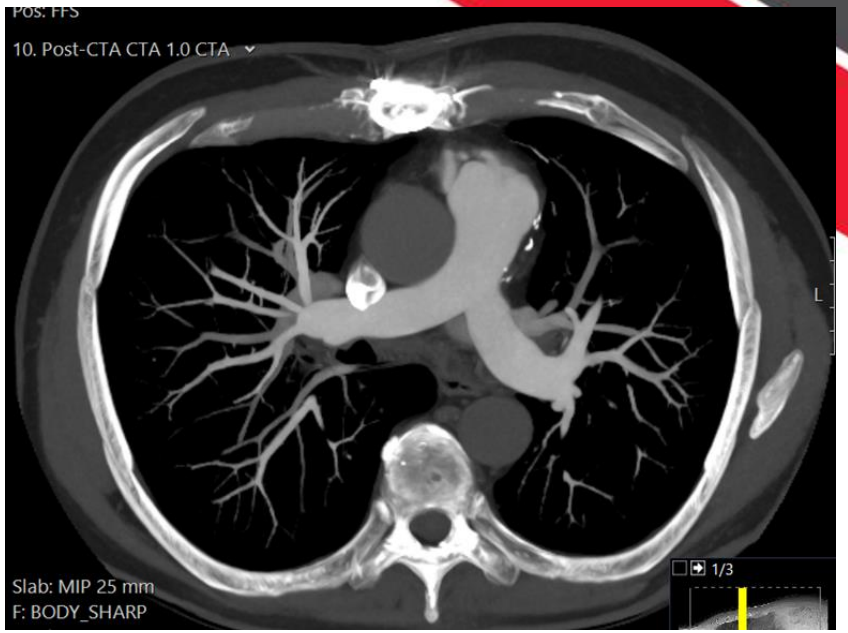
LARGE BILATERAL PULMONARY EMBOLUS IN A SADDLE BAG FASHION-extending into both lower lobes and into the upper lobe as well. High degree of occlusion noted especially on the RIGHT.

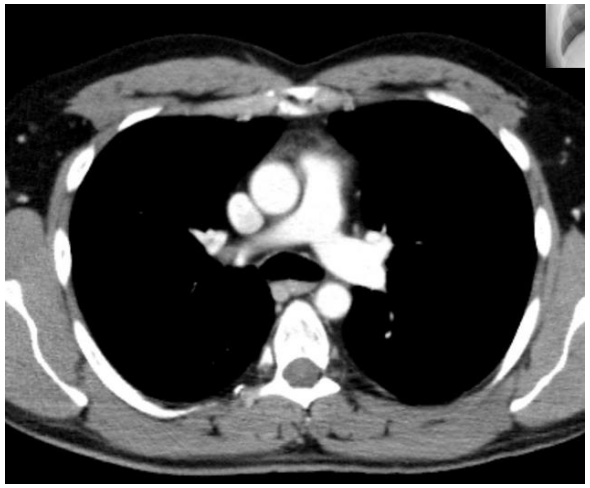


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The Story Goes On…

- 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

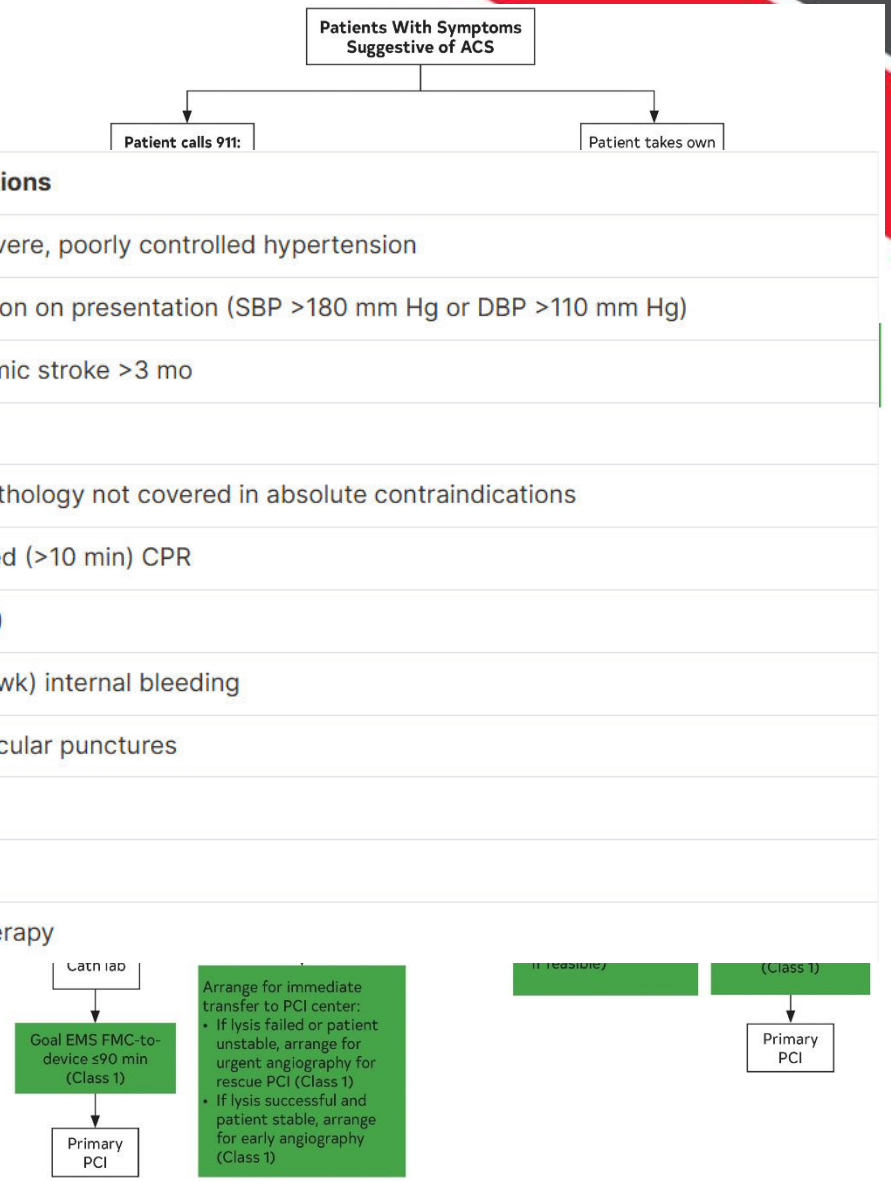
Current Guidelines for tPA Management

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.

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



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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

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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

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Levy et al., 2024. Aortic Dissection - StatPearls - NCBI Bookshelf

Harris et al., 2022. doi:10.1001/jamacardio.2022.2718

- 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

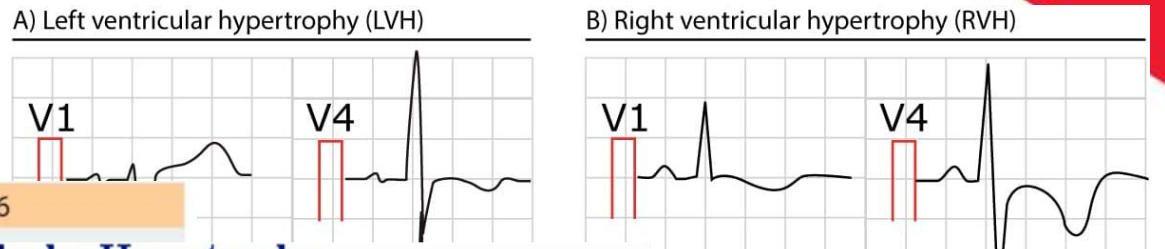
Usually NO EKG changes

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

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Hypertension/Left Ventricular Hypertrophy



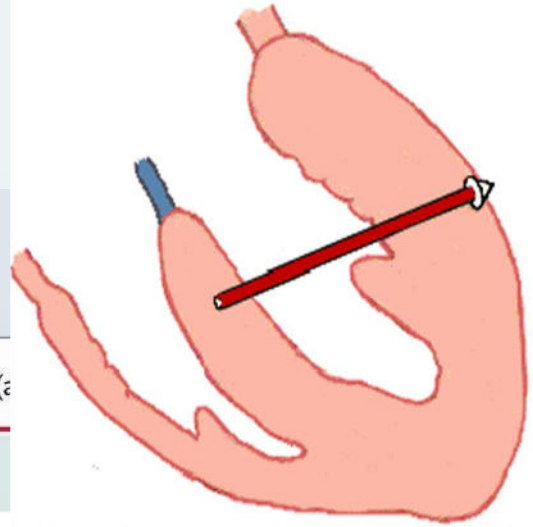
Left ventricular hypertrophy	V1/V2	V5/V6
Sv1		
R v		
Sv3 + RaVL (Cornell voltage)		
Cornell duration product[b]		

Cornell Duration Product = [S(V3) + R(a

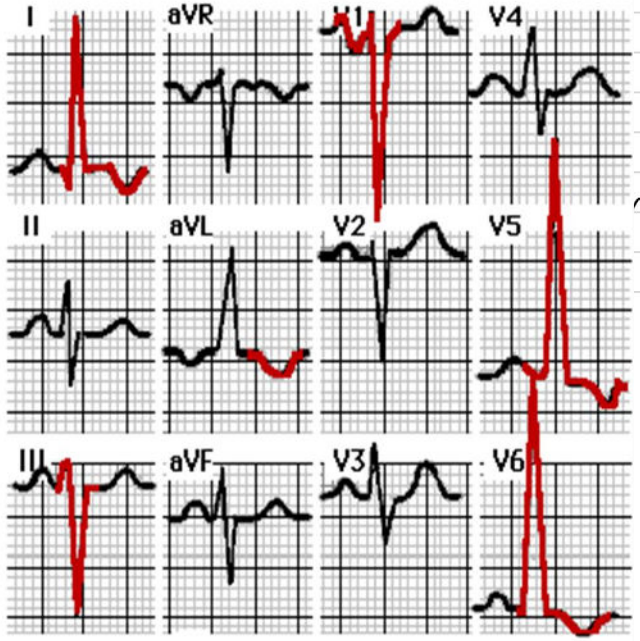


Left Ventricular Hypertrophy

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



Arrow indicates major electrical vector of ventricular depolarization



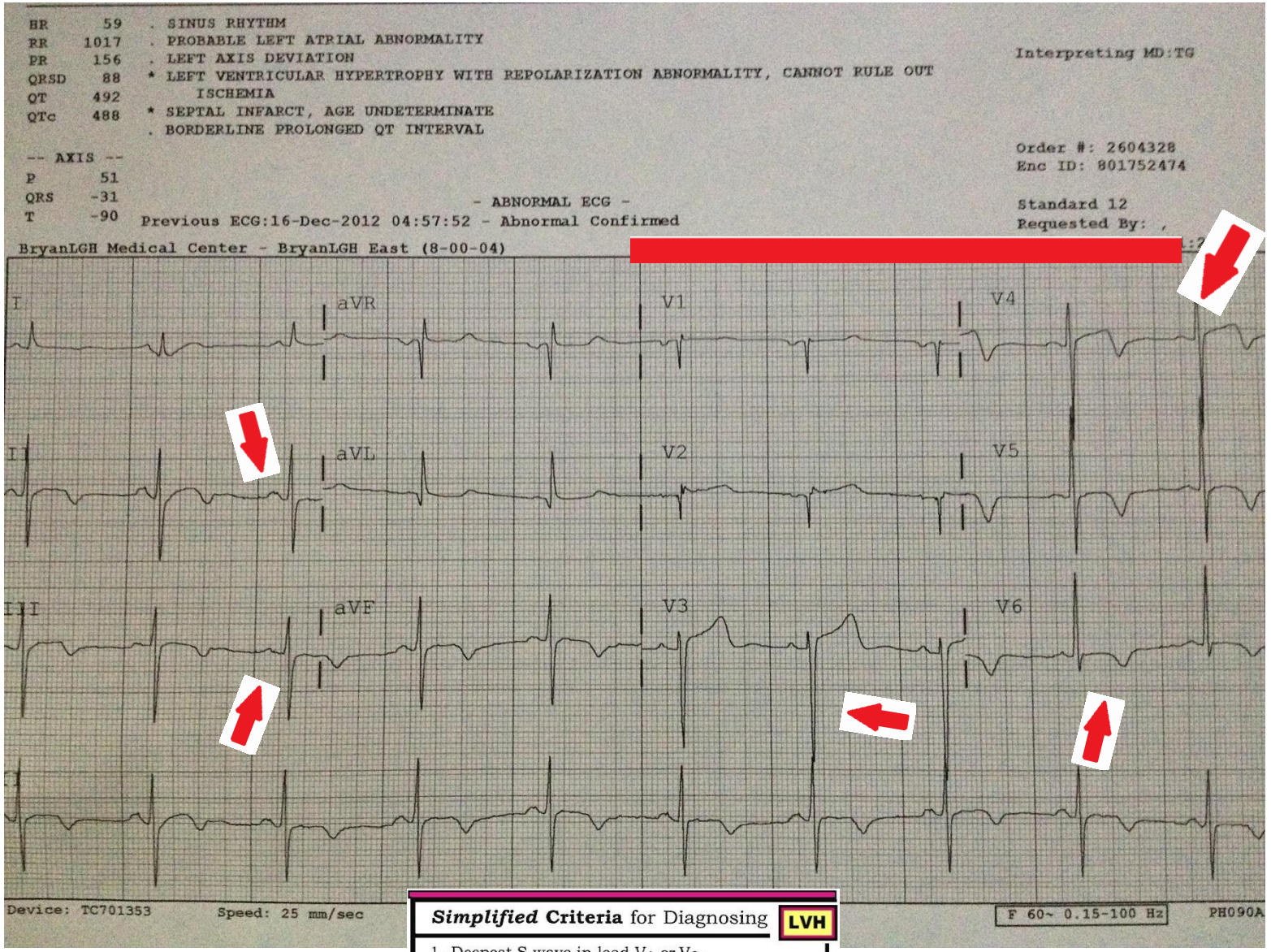
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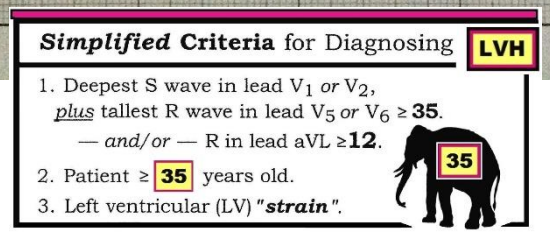
Simple Voltage Criteria for LVH on ECG

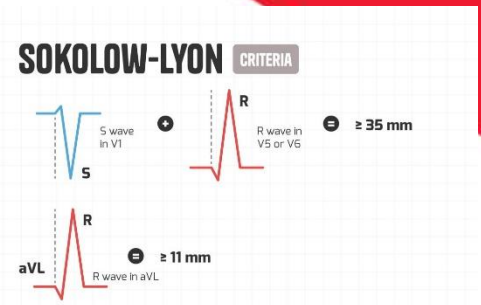
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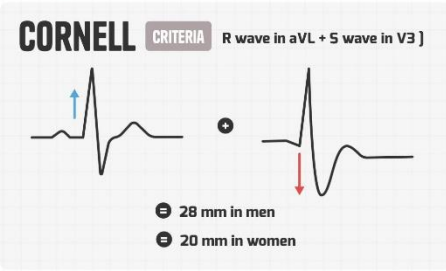
53-Year-Old Male…

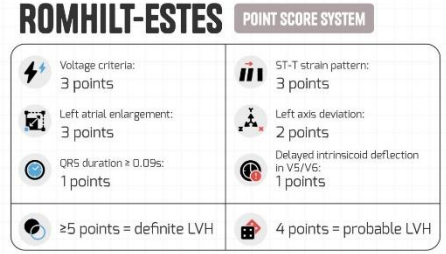
- No history of CAD but does have HTN
- Admitted 2 months before for atypical chest pain
  - Stress at that time showed diaphragmatic attenuation but no ischemia
- Came in as a cardiac alert (at table drinking coffee when he developed **sudden and severe CP**)
- EKG had inconsistent changes because of 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)

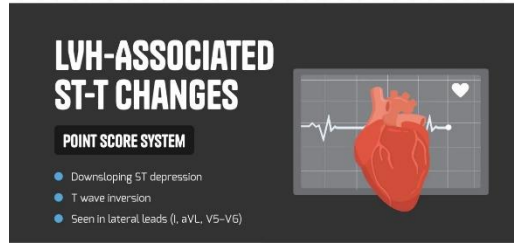












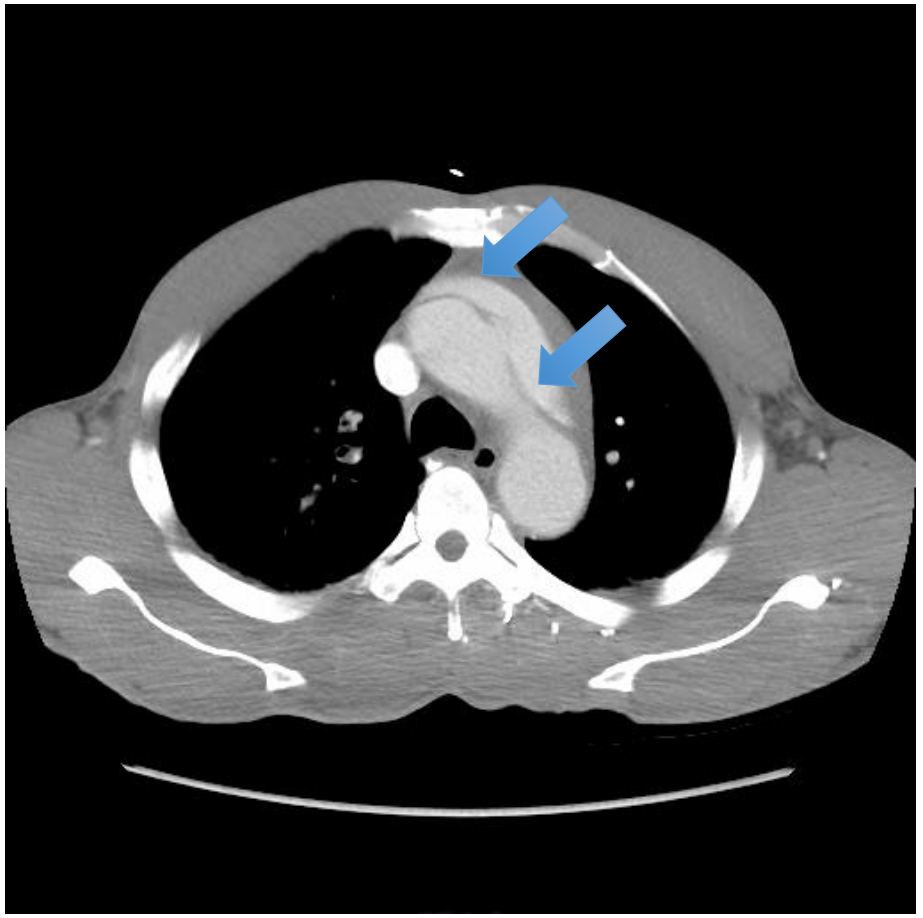
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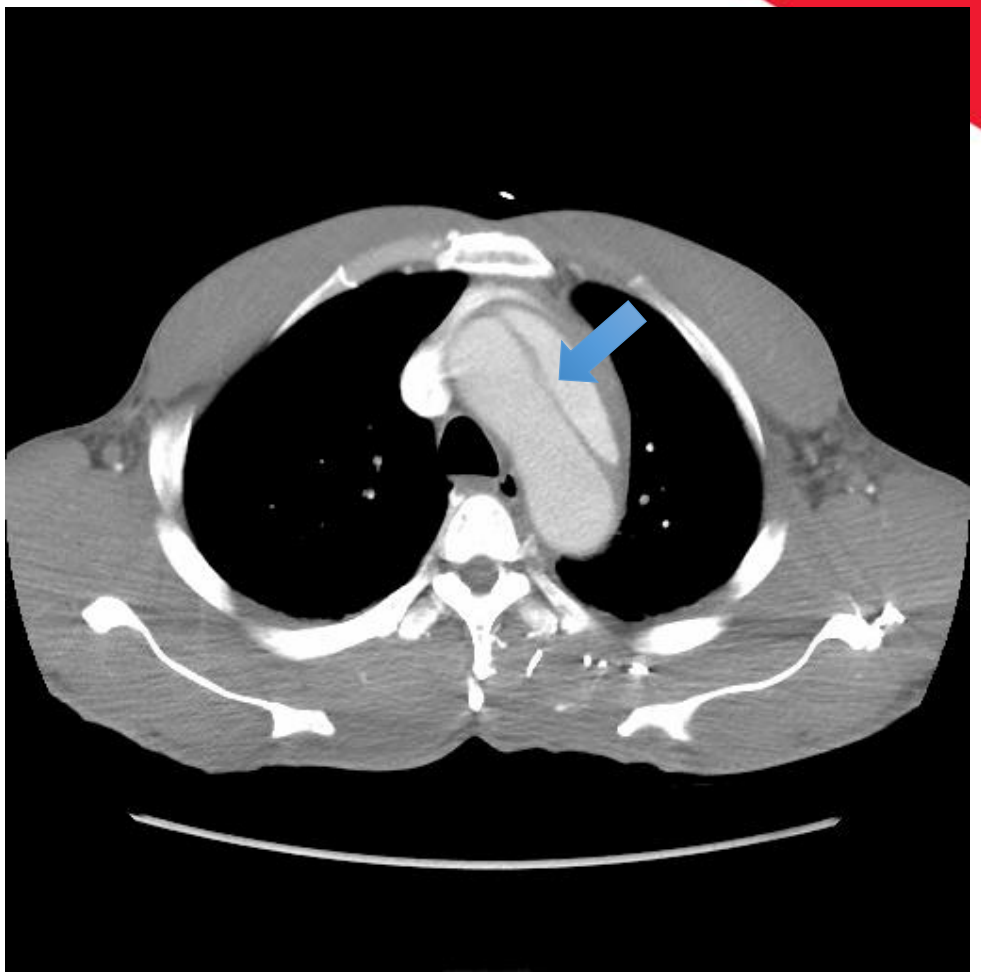
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VisualMed (2025): Left Ventricular Hypertrophy ECG Criteria Infographic – Visualmed: Clinical Trials

- 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.



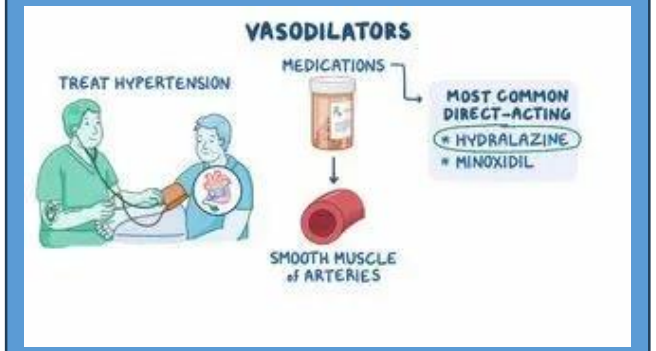


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- Stat CT surgery consult and into surgery with in the hour
- Lengthy hospitalization with acute respiratory failure, acute kidney injury, neurologic changes but able enough to bite through his ET tube three different times; PEG and trach placed but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



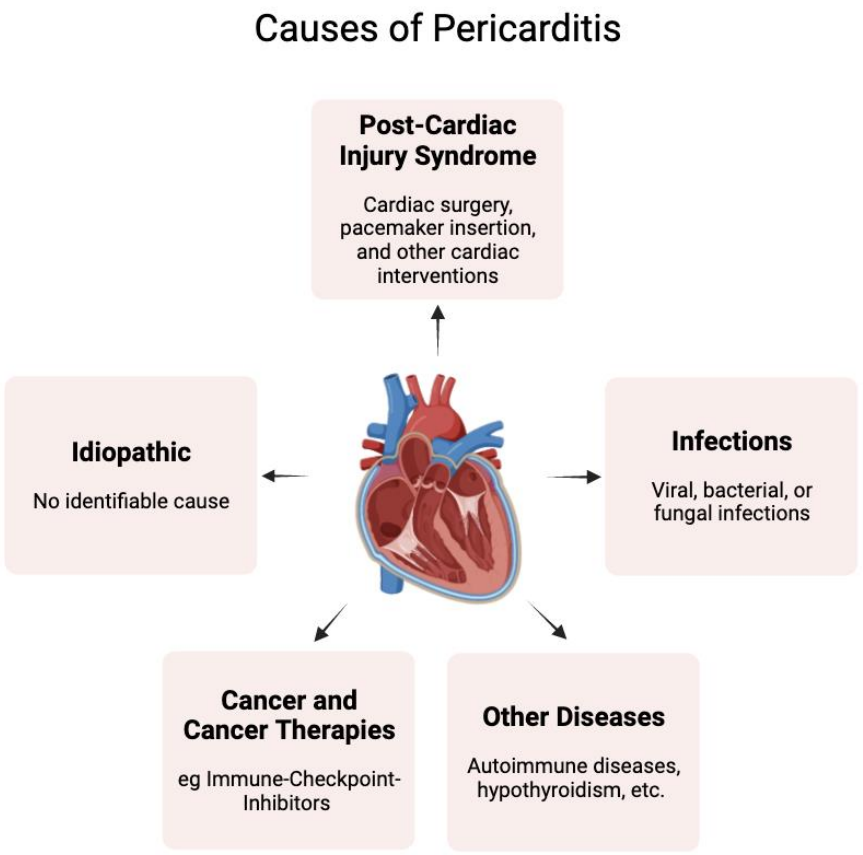




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Pericarditis



Recent Cold?

Recent Surgery?

Recent Surgery?

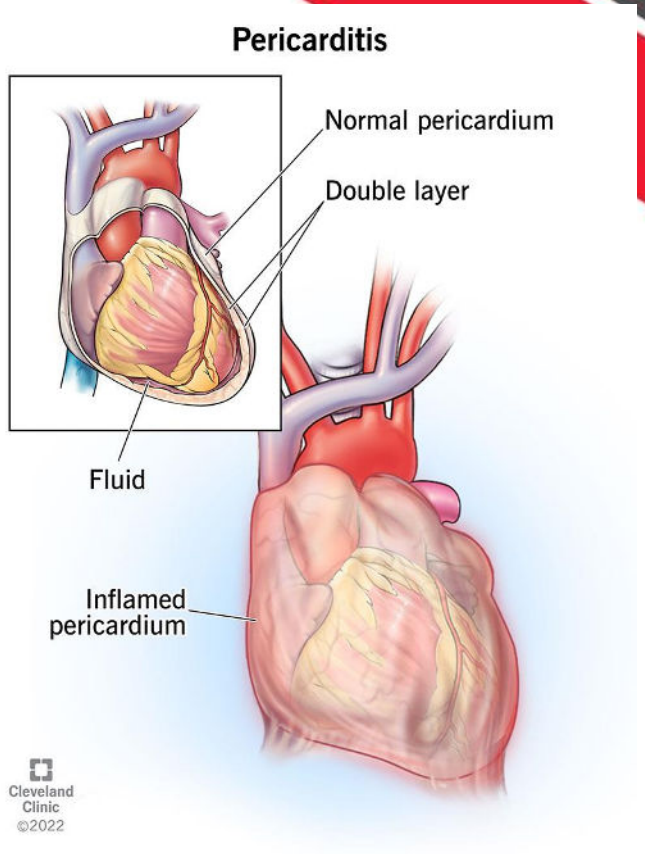


Table 3. Differential Diagnosis of Acute Pericarditis

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.

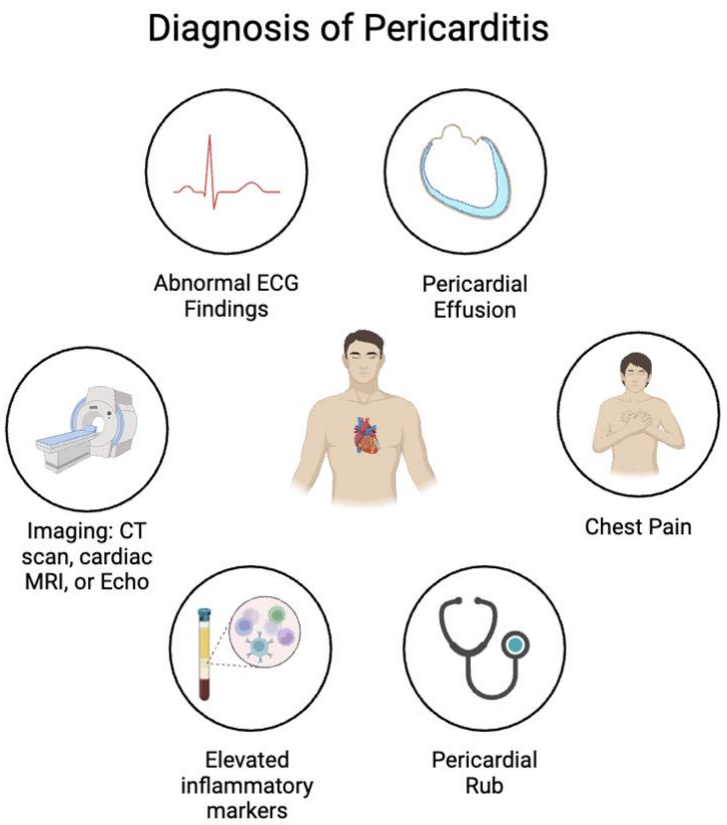
Affects EKG

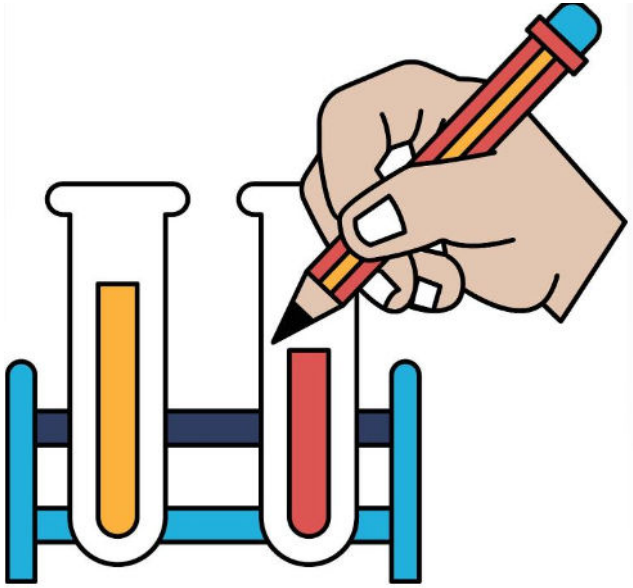
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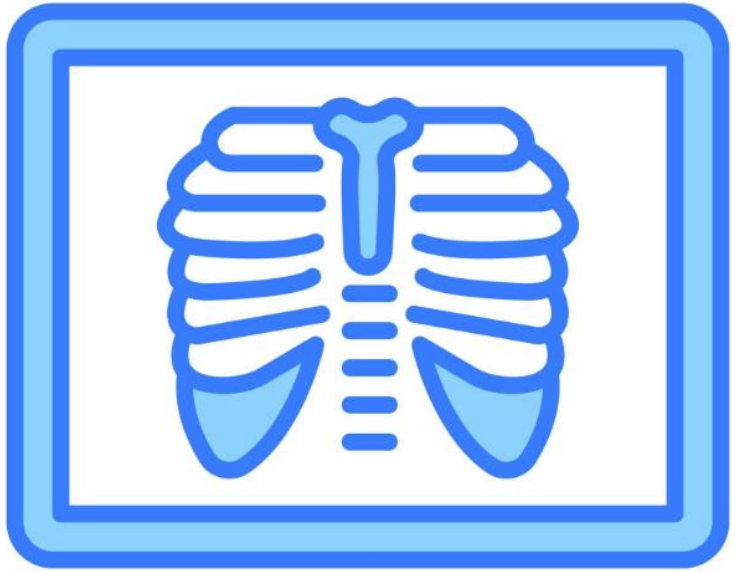
[Pericardial Disease Center | UCSF Cardiology]

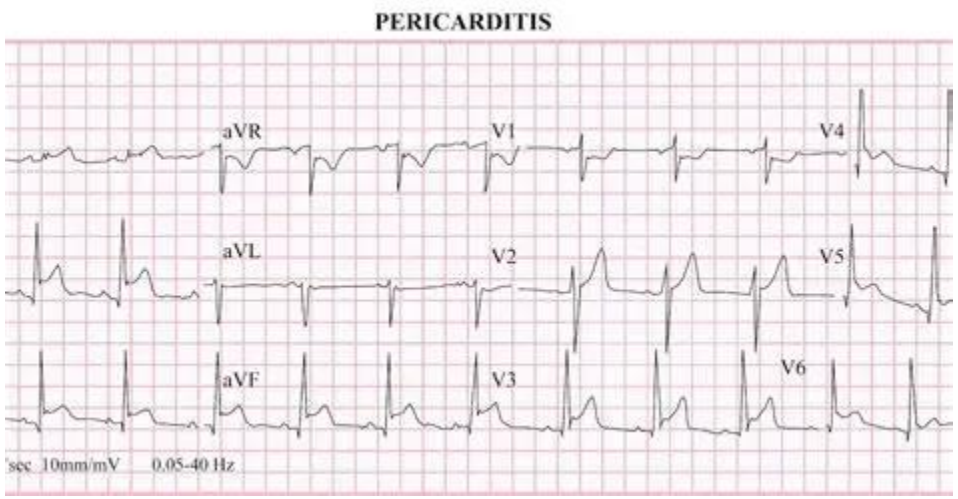
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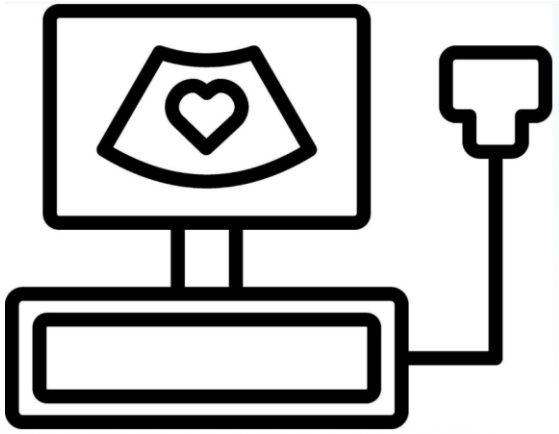
Typical ER Work Up





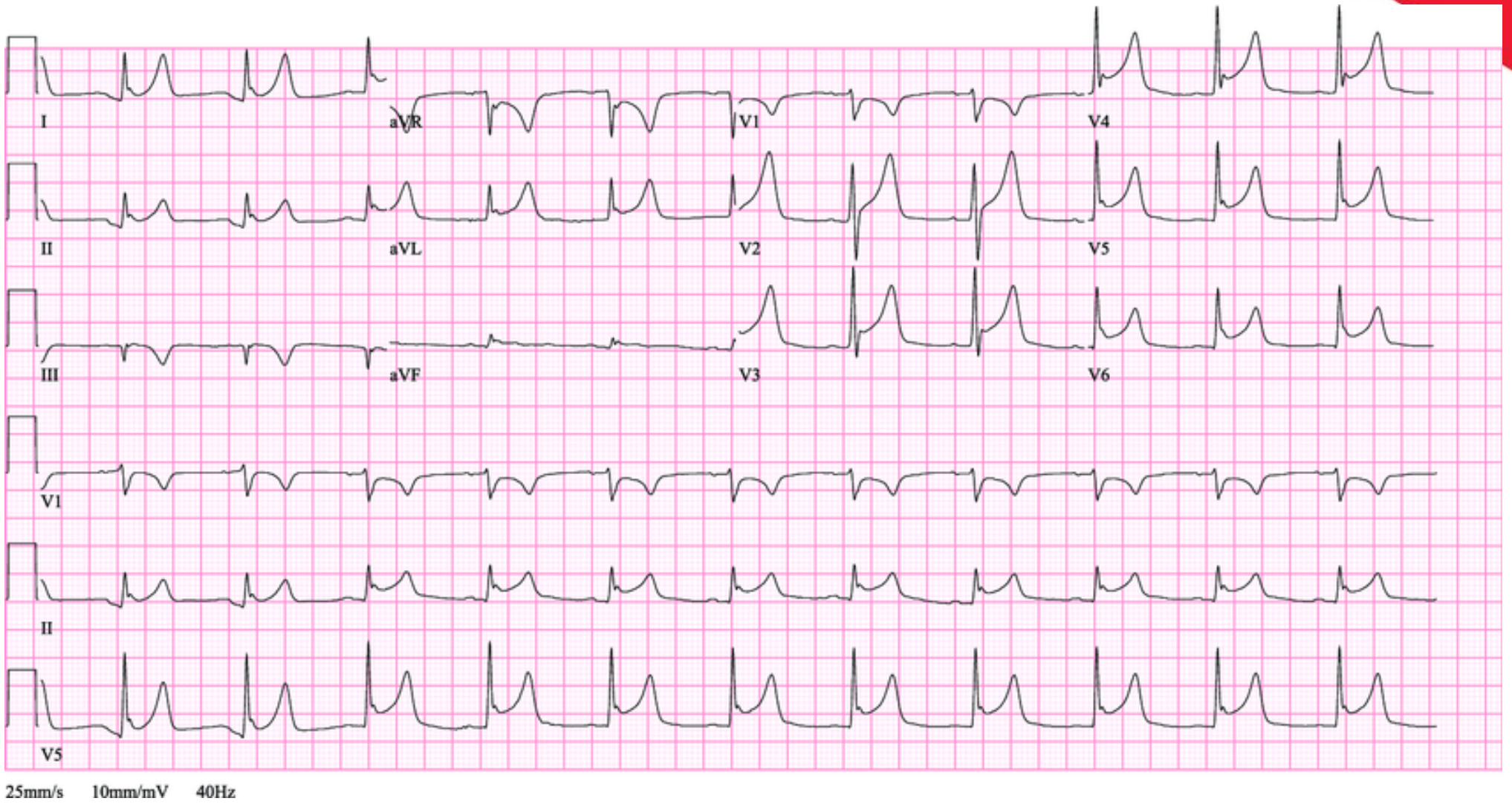






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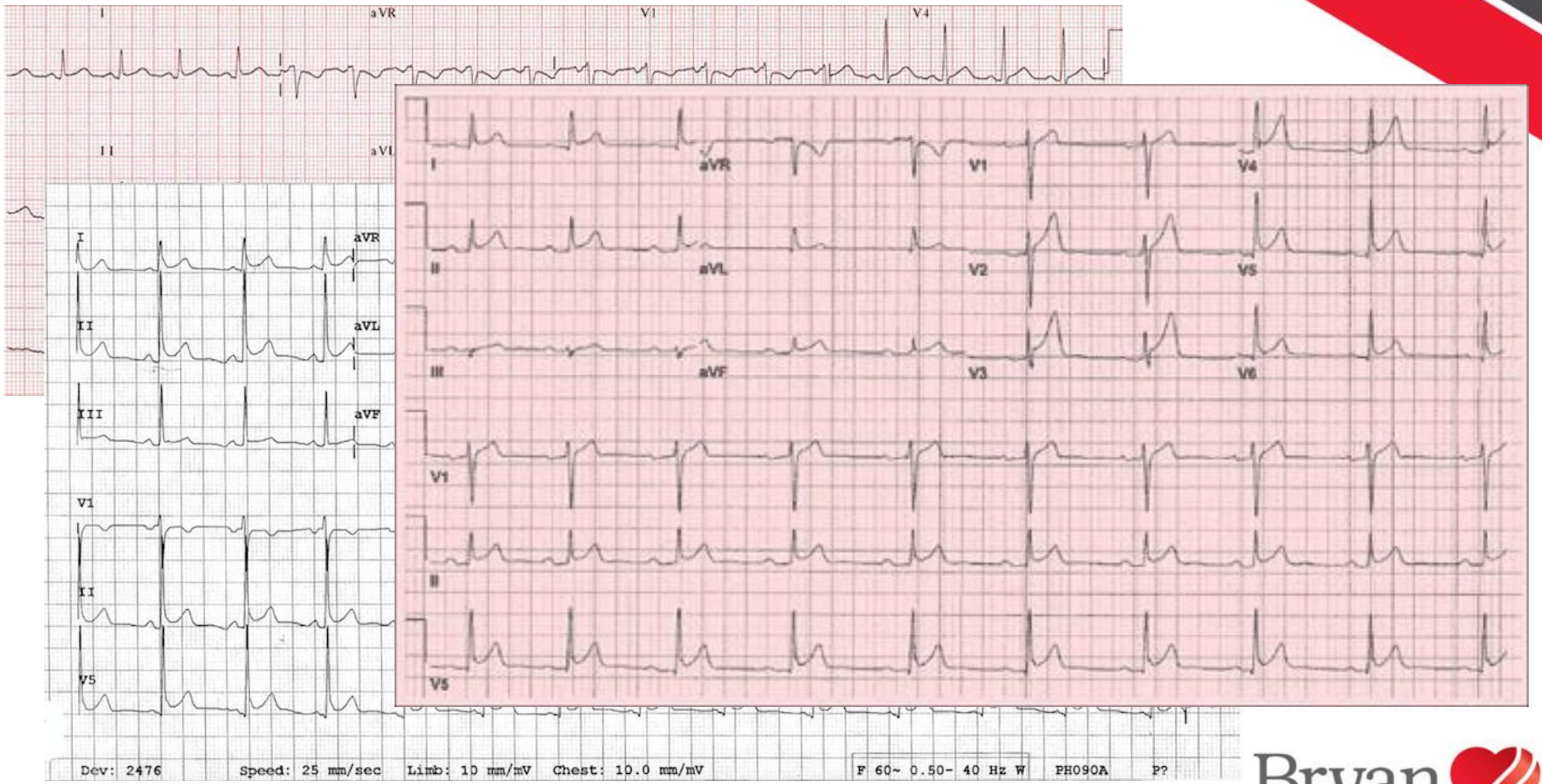
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Case Scenario Discussion

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Typical Treatment of Pericarditis

Management for Pericarditis



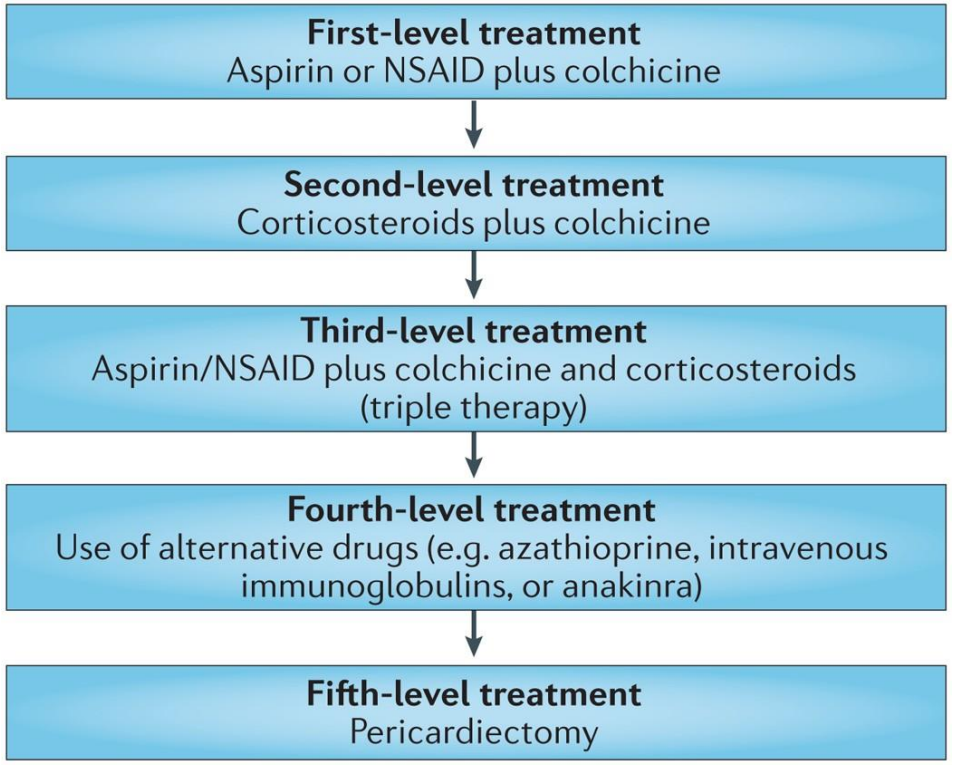
High-dose aspirin or NSAIDs may be given for ~2 weeks as well as GI protective medication.



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



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



Nature Reviews | Cardiology

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Pericardial Disease Center | UCSF Cardiology

Recurrent pericarditis: new and emerging therapeutic options | Nature Reviews Cardiology.

Diagnosis Is Made With Presence of Pleuritic Chest Pain and ≥1 of the Following Criteria:
Friction pericardial rub on auscultation
Electrocardiographic evidence such as classic PR-segment depression or diffusive concave ST-segment elevations, or in the setting of MI, persistent ST-segment elevations or dynamic T-wave changes
New or growing pericardial effusion on echocardiography

Table 19. Additional Therapy for Persistent/Late Pericarditis Symptoms

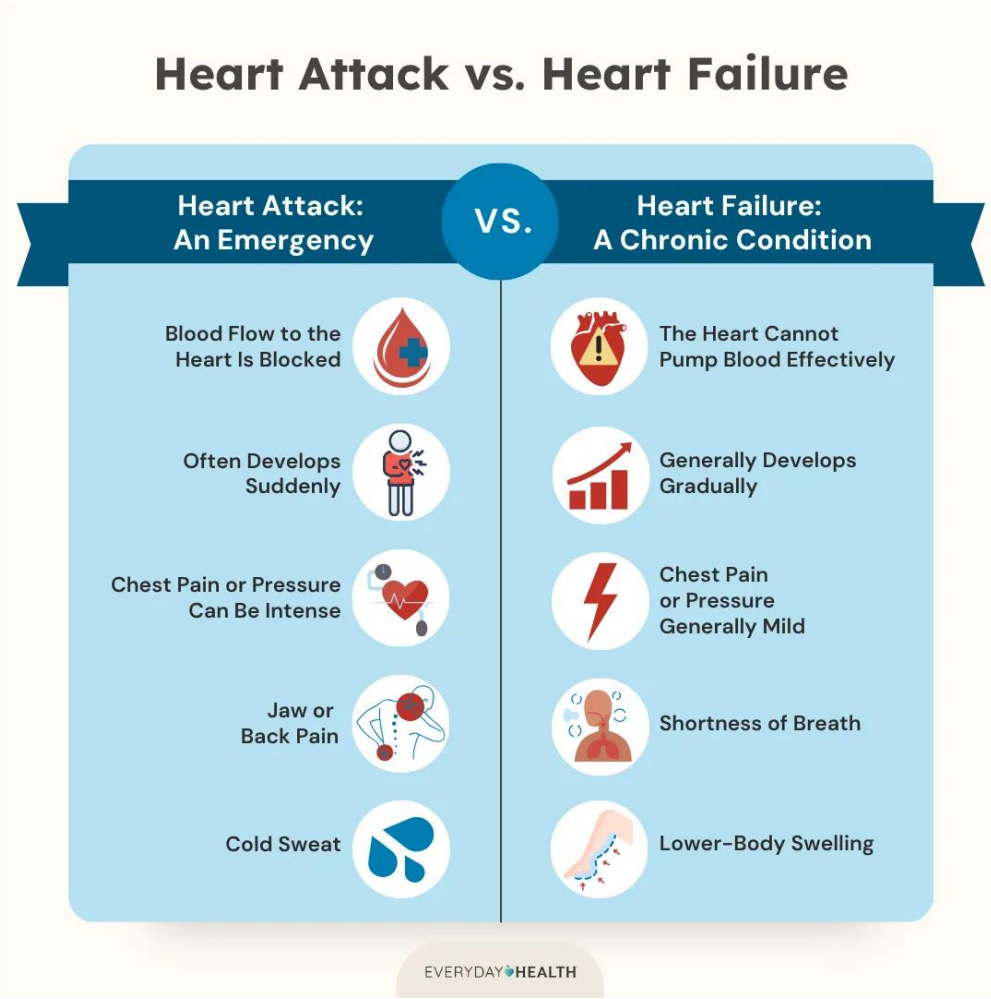
Medical Therapy	Dosing Schedule
High-dose aspirin	500-1000 mg every 6-8 h until symptoms improve
Colchicine	0.5-0.6 mg once* or twice daily for 3 mo

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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

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Acute Heart Failure



JOURNAL ARTICLE

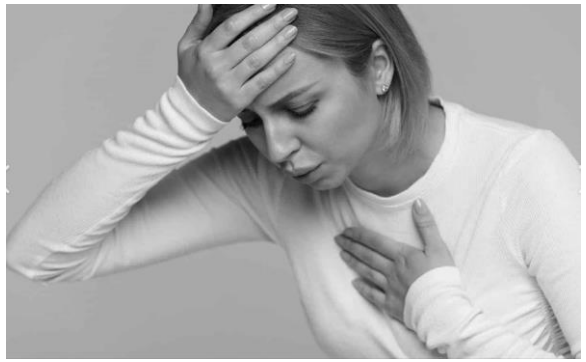
Chest pain in patients with heart failure: why history may matter

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.

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Heart Attack vs. Heart Failure: Symptoms and Differences

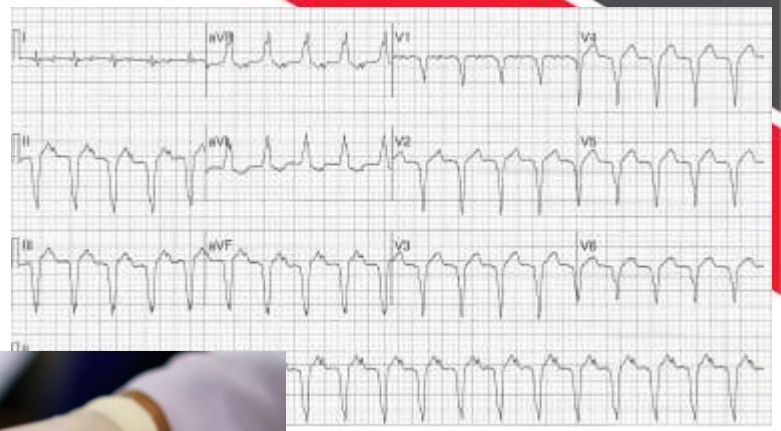
Chest pain in patients with heart failure: why history may matter | European Heart Journal | Oxford Academic

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Evaluation/Diagnosis

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

Heart failure	Alternative causes
Symptoms	Symptoms
Abdominal swelling	Abdominal swelling (liver failure)
Dyspnea on exertion	Anorexia, weight loss (sarcoidosis)
Edema	Chest pain (coronary artery disease)
Exercise intolerance	Claudication (atherosclerotic disease)
Fatigue	Cough (pulmonary disease)
Orthopnea	Diarrhea or skin lesions (amyloidosis)
Paroxysmal nocturnal dyspnea	Dyspnea on exertion (pulmonary disease, valvular disease)
Recent weight gain	Edema (liver or kidney failure)
Physical examination findings	Neurologic problems (sarcoidosis)
Abdomen: hepatojugular reflux, ascites	Palpitations (tachyarrhythmia)
Extremities: cool, dependent edema	Recent fevers, viral infection (endocarditis, myocarditis, infection)
Heart: bradycardia/tachycardia, laterally displaced point of maximal impulse, third heart sound (gallop or murmur)	Syncope (bradycardia, heart block)
Lungs: labored breathing, rales	Physical examination findings
Neck: elevated jugular venous pressure	Abdomen: distended, hepatosplenomegaly, tender, ascites (liver disease)
Skin: cyanosis, pallor	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)







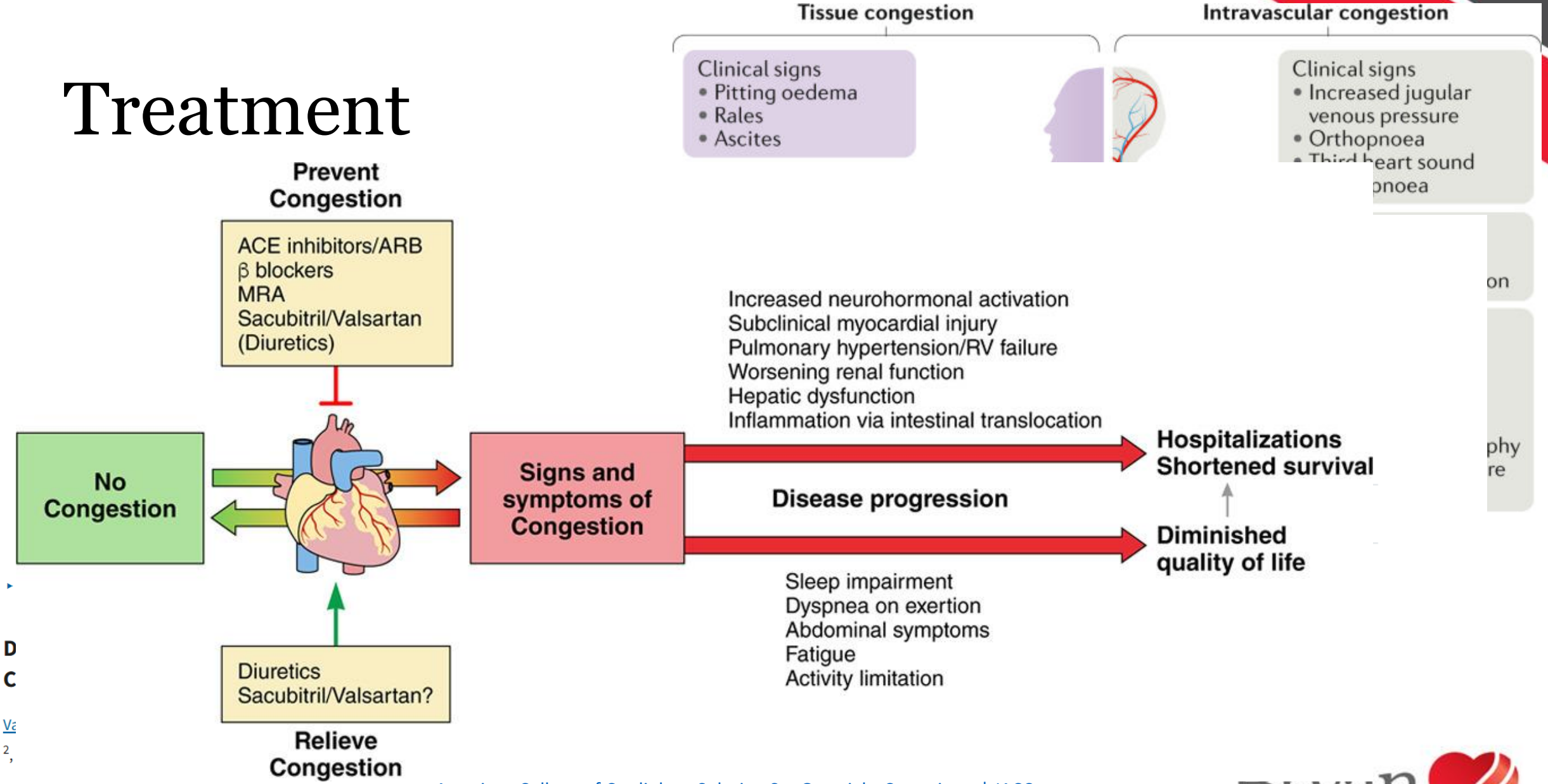




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Treatment



American College of Cardiology Solution Set Oversight Committee | JACC

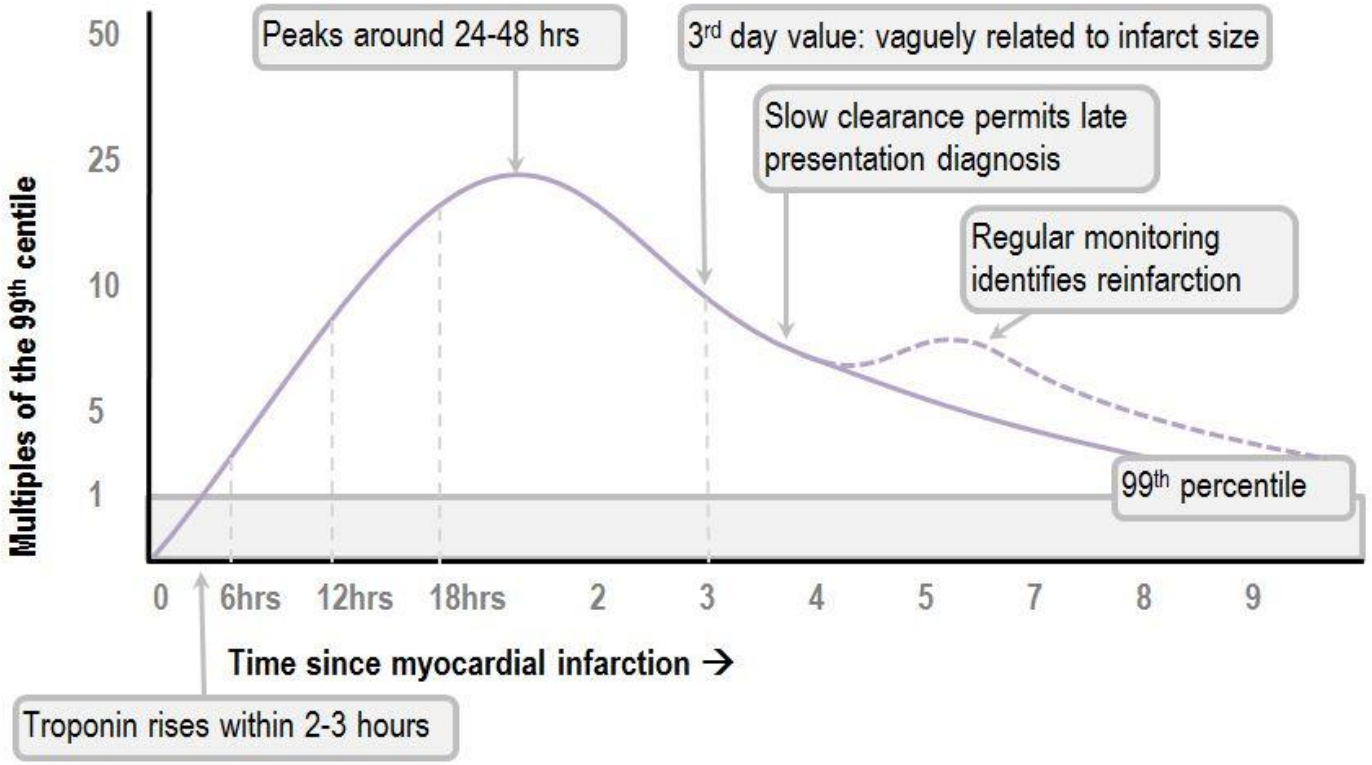
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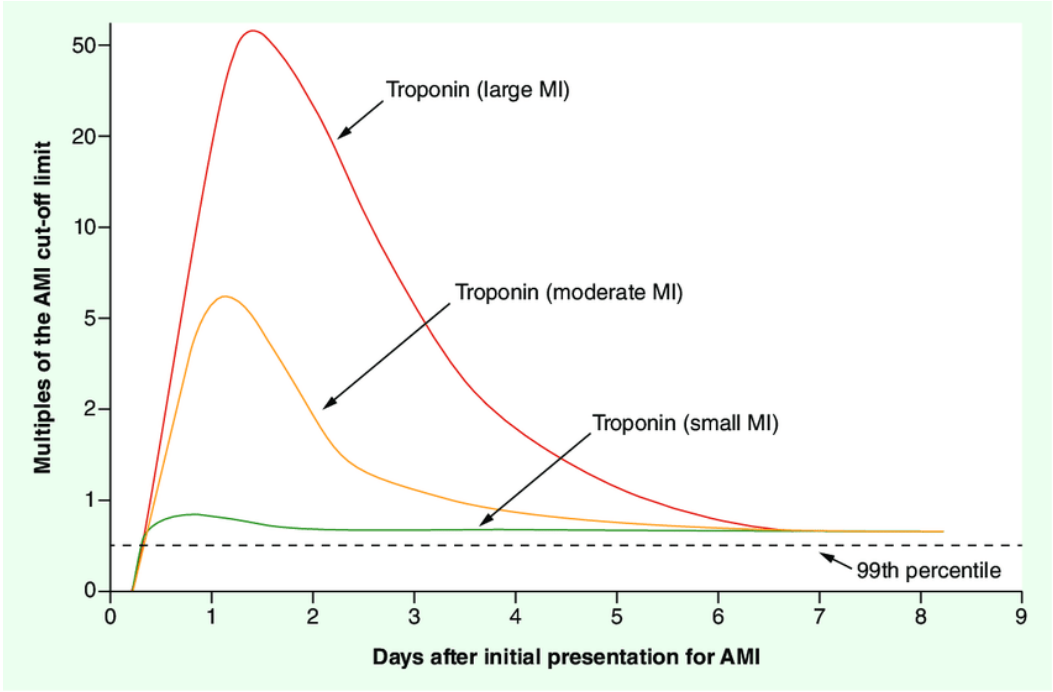
Decongestion in Acute Heart Failure—Time to Rethink and Standardize Current Clinical Practice? - PMC

Congestion in heart failure: a contemporary look at physiology, diagnosis and treatment | Nature Reviews Cardiology

Relief and Prevention of Congestion in Heart Failure Enhance Quality and Length of Life | Circulation

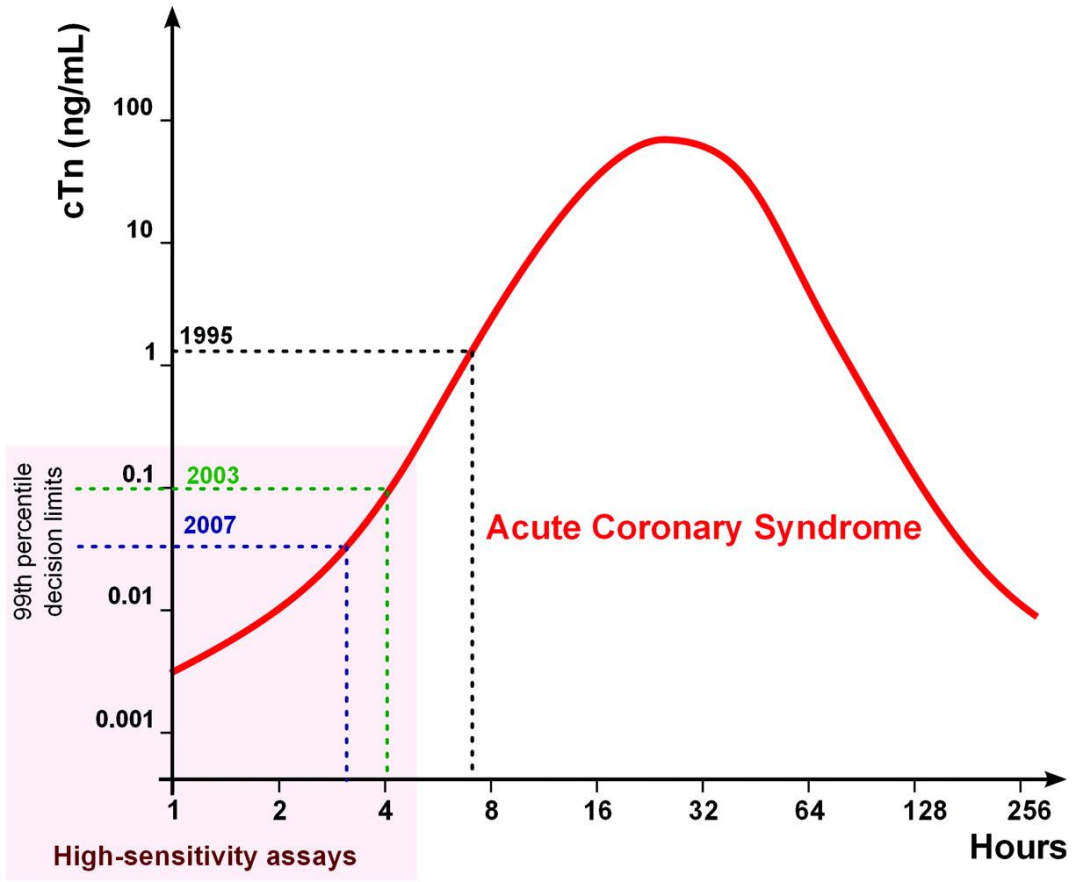
Non-ST Elevated Myocardial Infarction



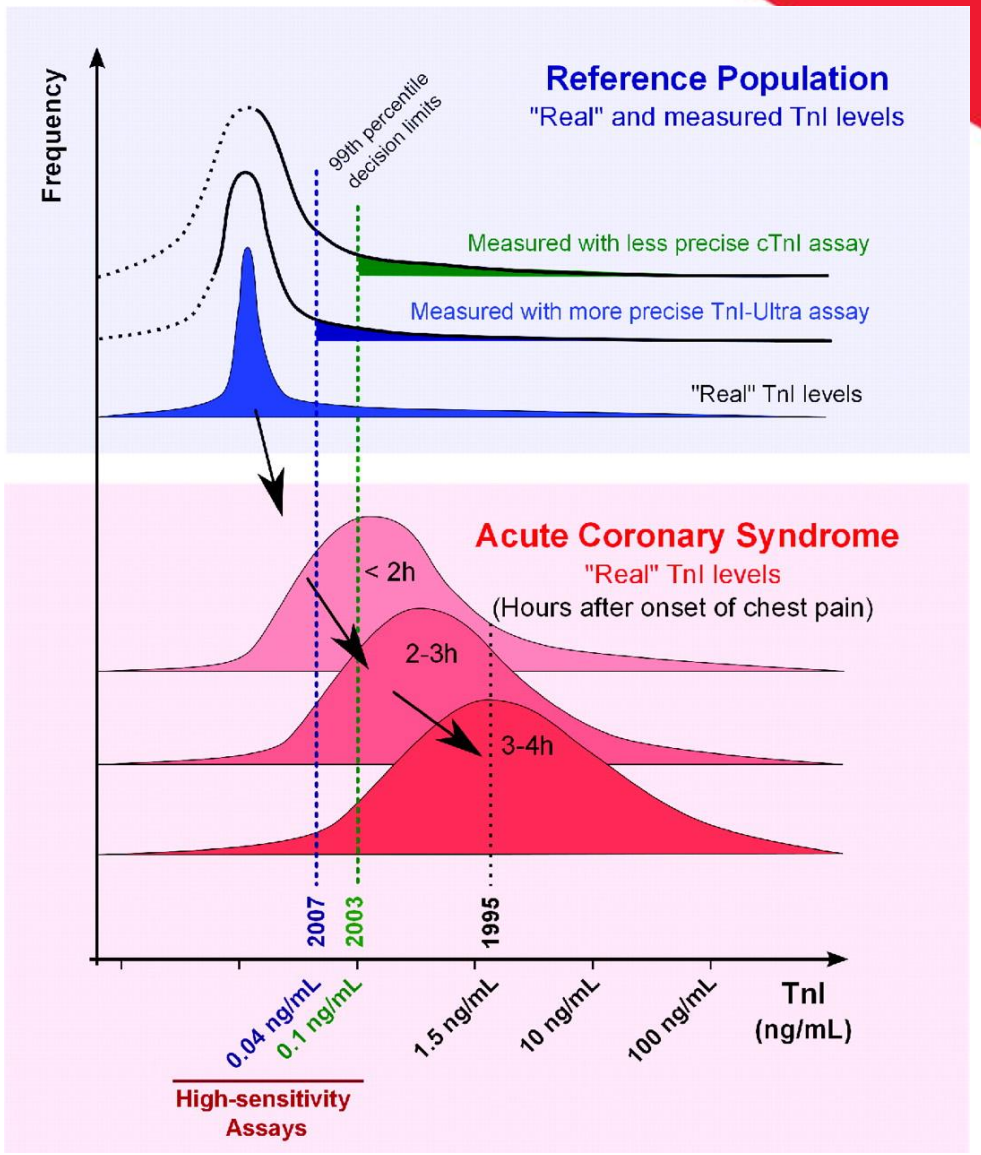


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cTn Assay	Diagnostic cutoff	Implementation
TnI	≥ 1.5 ng/mL	1995
cTnI	> 0.10 ng/mL	2003
TnI-Ultra	> 0.04 ng/mL	2007



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How to Interpret Elevated Cardiac Troponin Levels | Circulation

Type 1*	**Caused by acute coronary atherothrombosis, usually precipitated by atherosclerotic plaque disruption (rupture or erosion) and often associated with partial or complete vessel thrombosis.**
Type 2	Caused by an imbalance between myocardial oxygen supply and demand unrelated to acute coronary atherothrombosis.
Type 3	Cardiac death, with symptoms of myocardial ischemia and presumed ischemic electrocardiographic changes or ventricular arrythmia, before blood samples for cardiac biomarkers can be obtained or increases in cardiac biomarkers can be identified and/or in whom MI is identified by autopsy.
Type 4	4a: Peri-PCI MI caused by a procedural complication and detected ≤48 h after PCI. 4b: Post-PCI MI caused by coronary stent or stent scaffold thrombosis. 4c: Post-PCI MI caused by coronary stent restenosis.
Type 5	Peri-CABG MI caused by a procedural complication detected ≤48 h after CABG surgery.

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[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]

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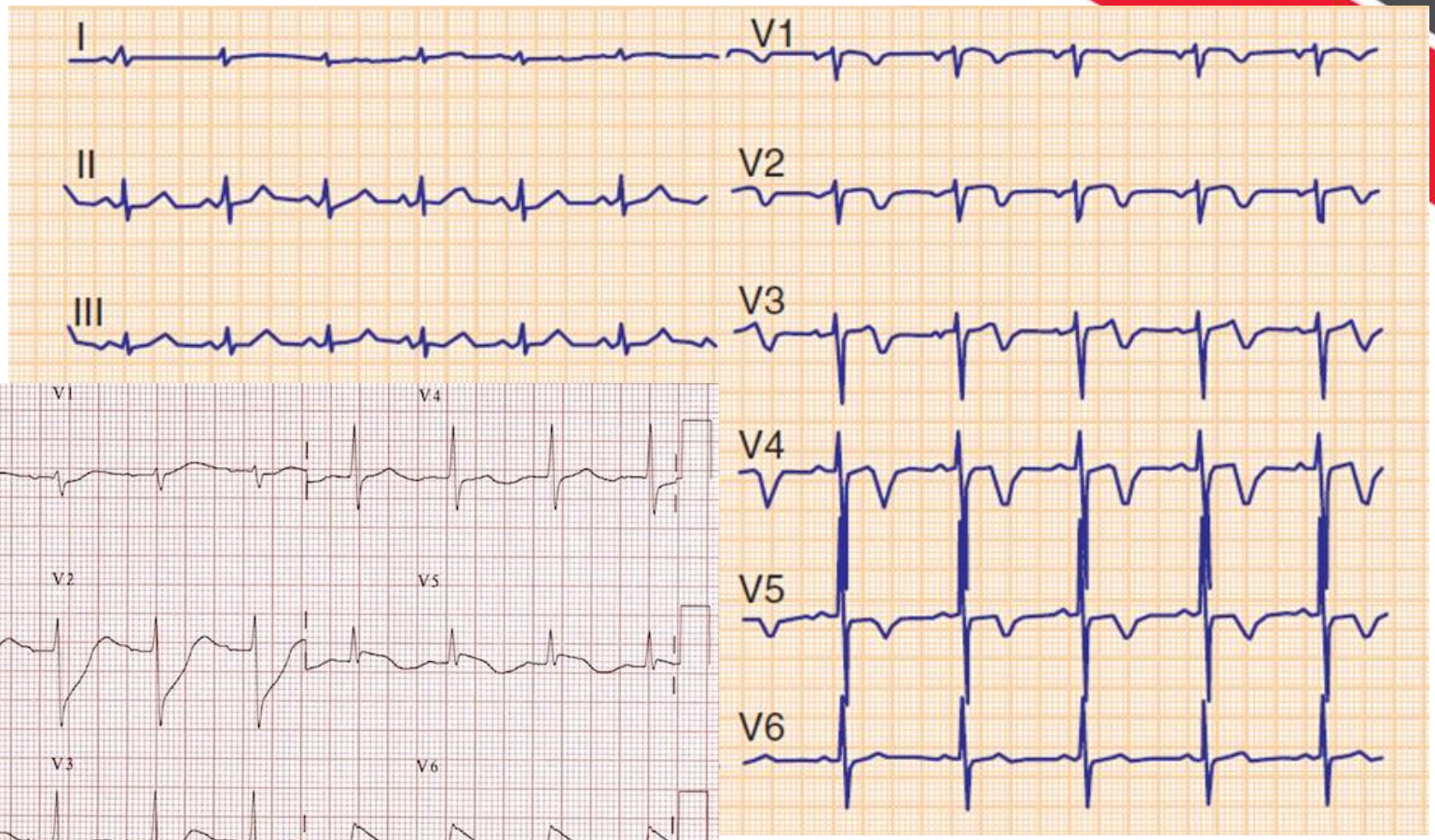
Medication	Route	Suggested Dosing	Considerations
Nitroglycerin*	SL (tablets, spray)	0.3 or 0.4 mg every 5 min as needed up to a total of 3 doses	Use in hemodynamically stable patients with SBP ≥90 mm Hg.
Nitroglycerin*	IV	Start at 10 µg/min and titrate to pain relief and hemodynamic tolerability.	Consider for persistent anginal pain after oral nitrate therapy, or if ACS is accompanied by hypertension or pulmonary edema.[20–22] Avoid use in suspected RV infarction, SBP <90 mm Hg or a change in SBP >30 mm Hg below baseline. Tachyphylaxis may occur after approximately 24 h.
Morphine	IV	2-4 mg; may repeat if needed every 5-15 min. Doses up to 10 mg may be considered.	Use for relief of pain that is resistant to other maximally tolerated anti-ischemic medications. May delay the effects of oral P2Y12 therapy.[7,9–12] Monitor closely for adverse effects.
Fentanyl	IV	25-50 µg; may repeat if needed. Doses up to 100 µg may be considered.	Use for relief of pain that is resistant to other maximally tolerated anti-ischemic medications. May delay the effects of oral P2Y12 therapy.[8] Monitor closely for adverse effects.

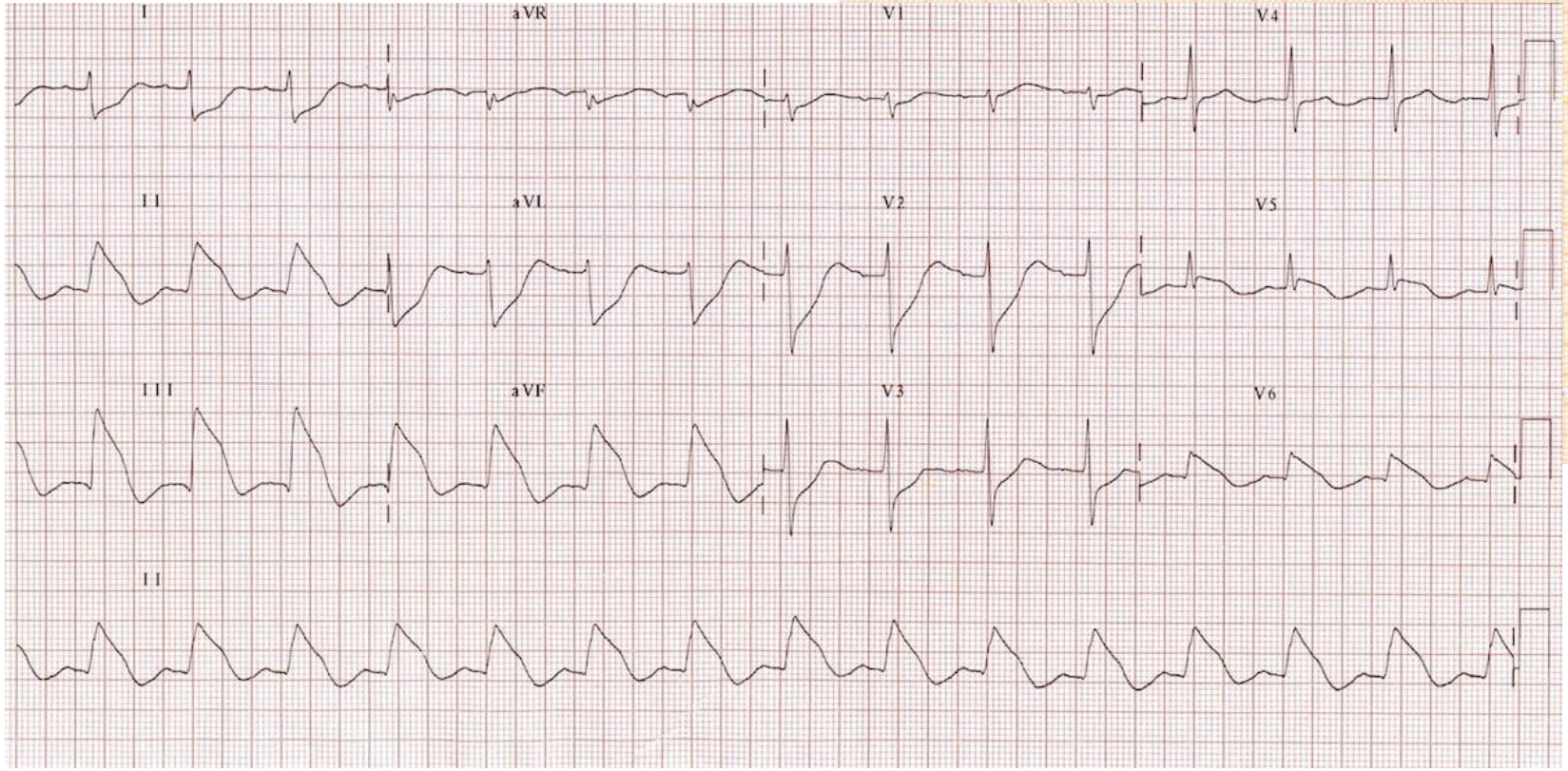
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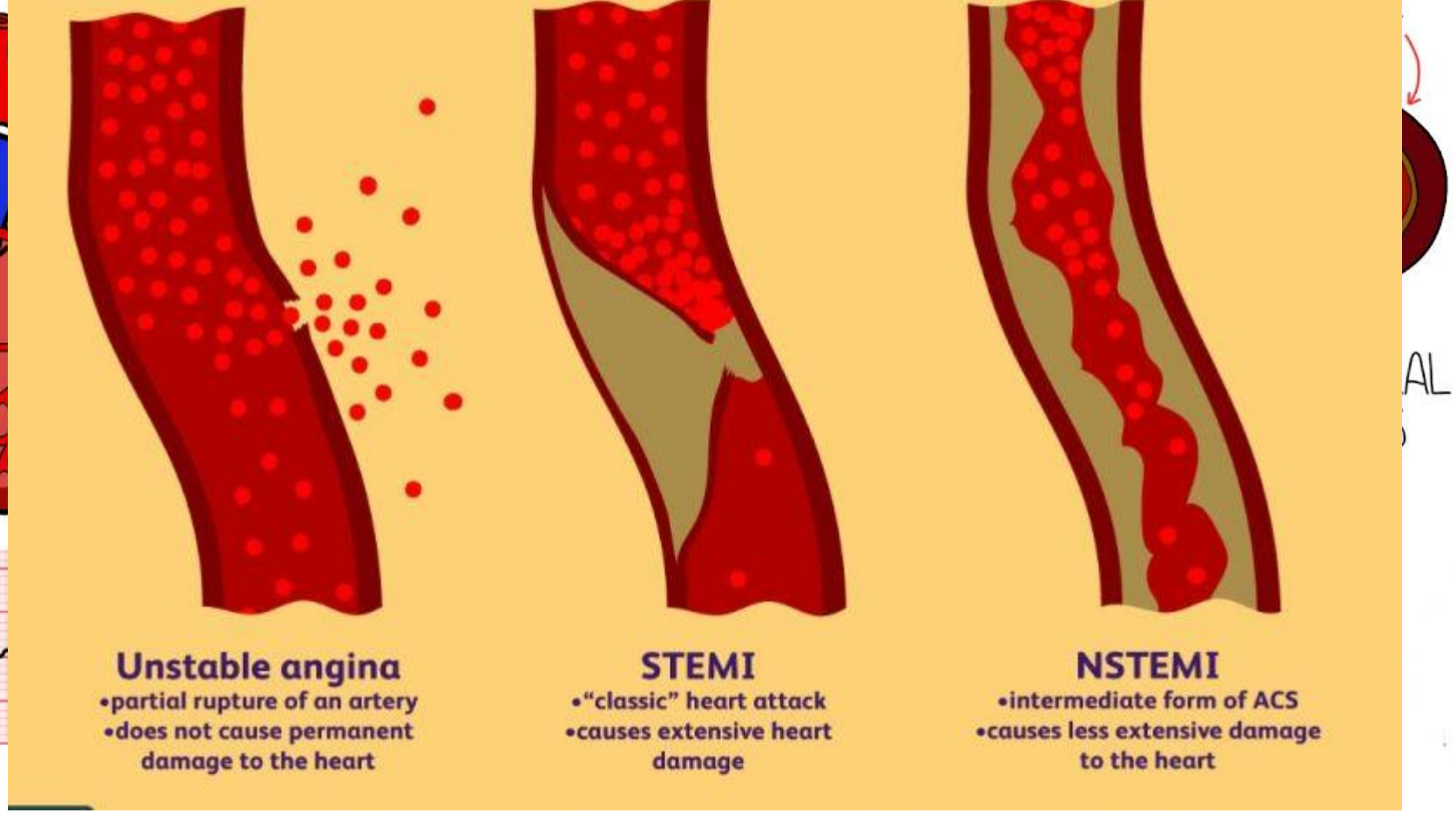
The EKG





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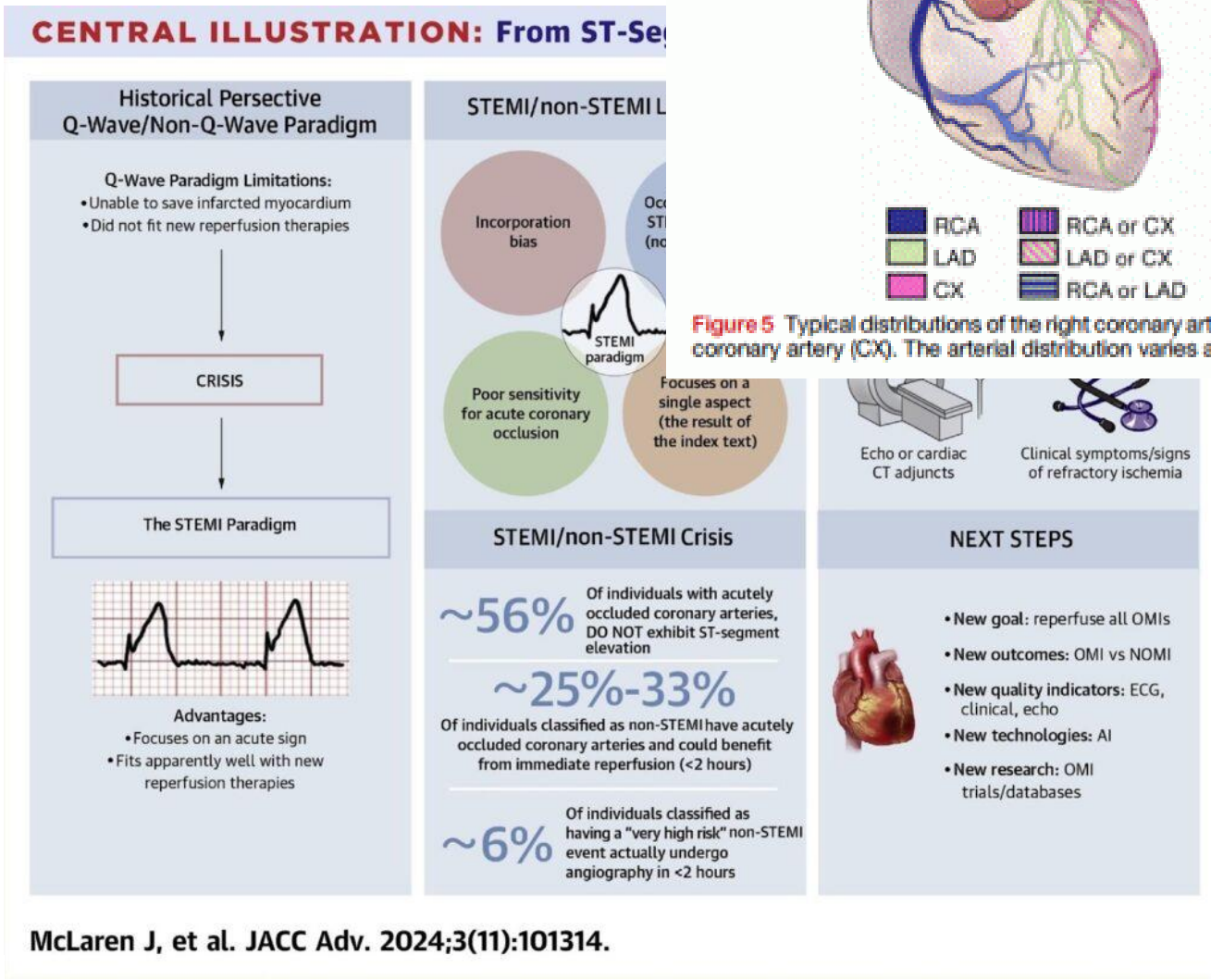
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The Echo



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

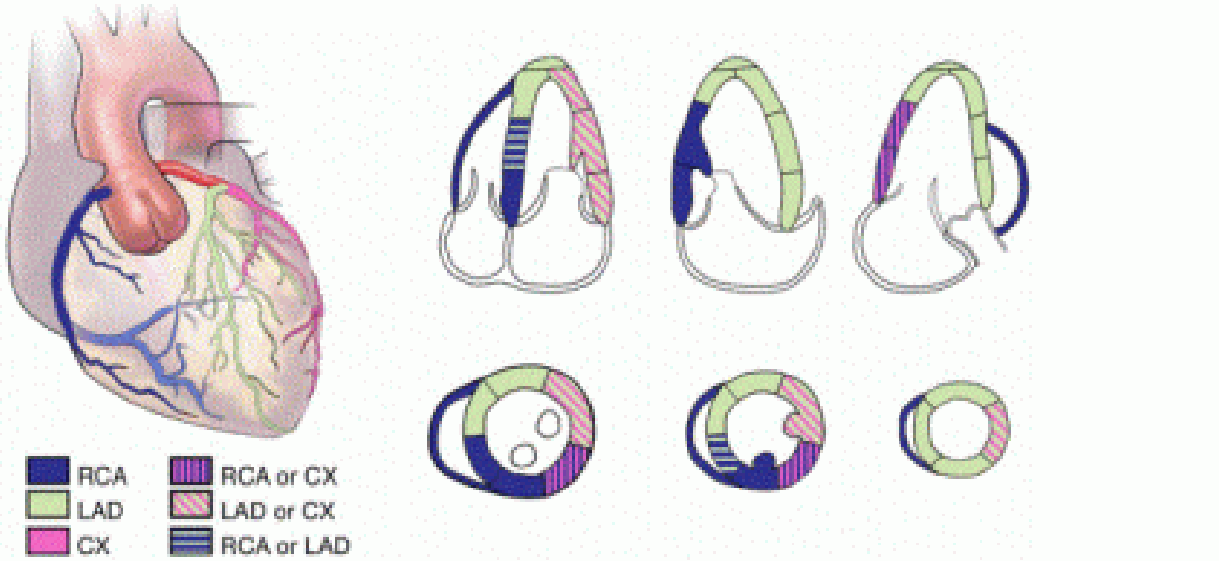


Figure 5 Typical distributions of the right coronary artery (RCA), the left anterior descending coronary artery (LAD), and the circumflex coronary artery (CX). The arterial distribution varies among patients. Some segments have variable coronary perfusion.

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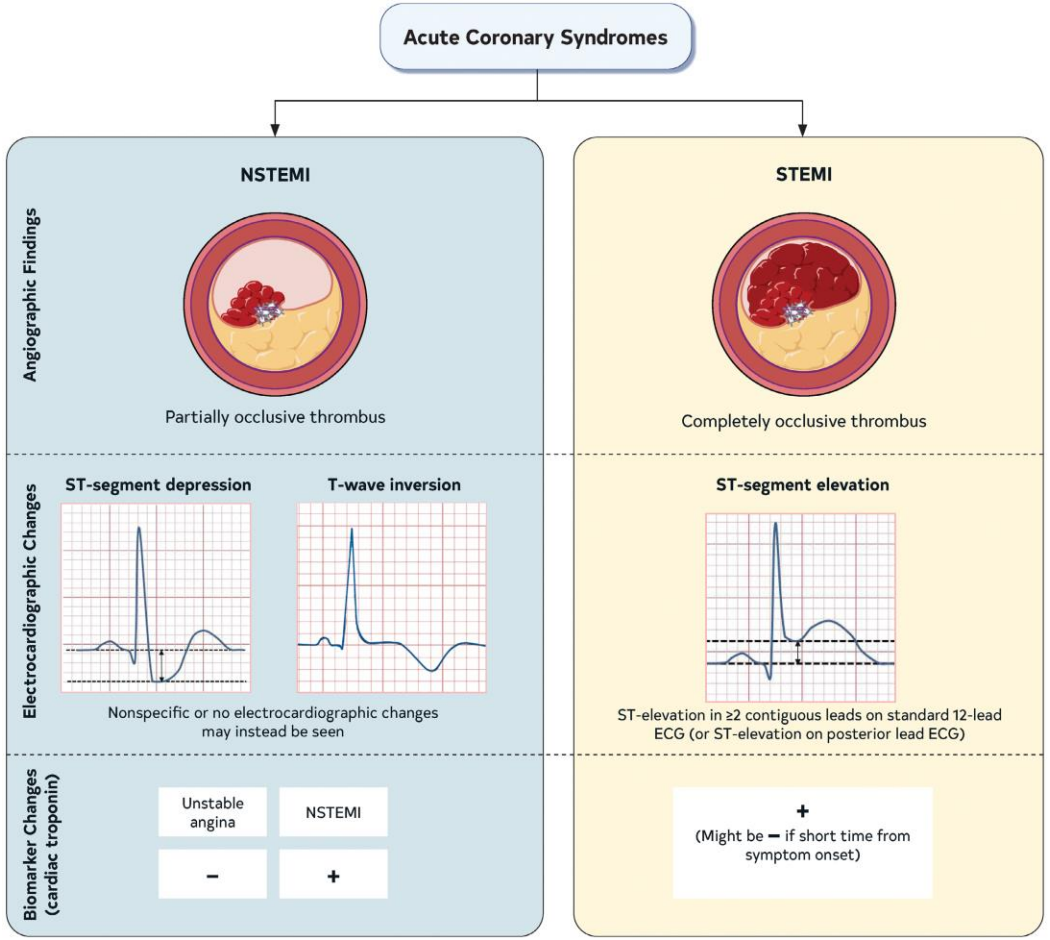
[PMcardio Featured on 'This Week in Cardiology': The Shift from STEMI/NSTEMI to OMI]

[Inferior STEMI : Is it RCA or LCX , and why we need to bother ? | Dr.S.Venkatesan MD]

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.

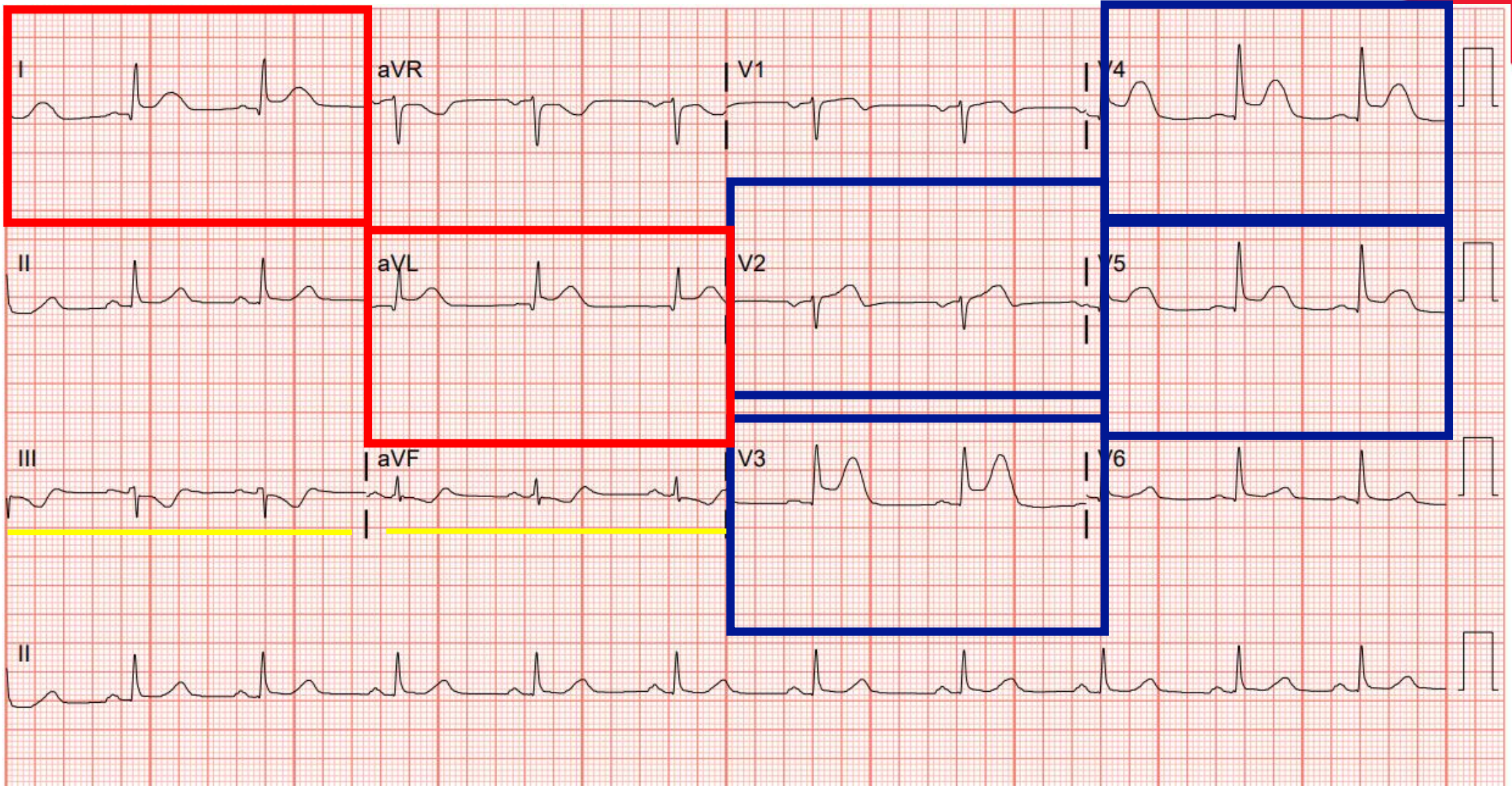
ST Elevated Myocardial Infarction

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



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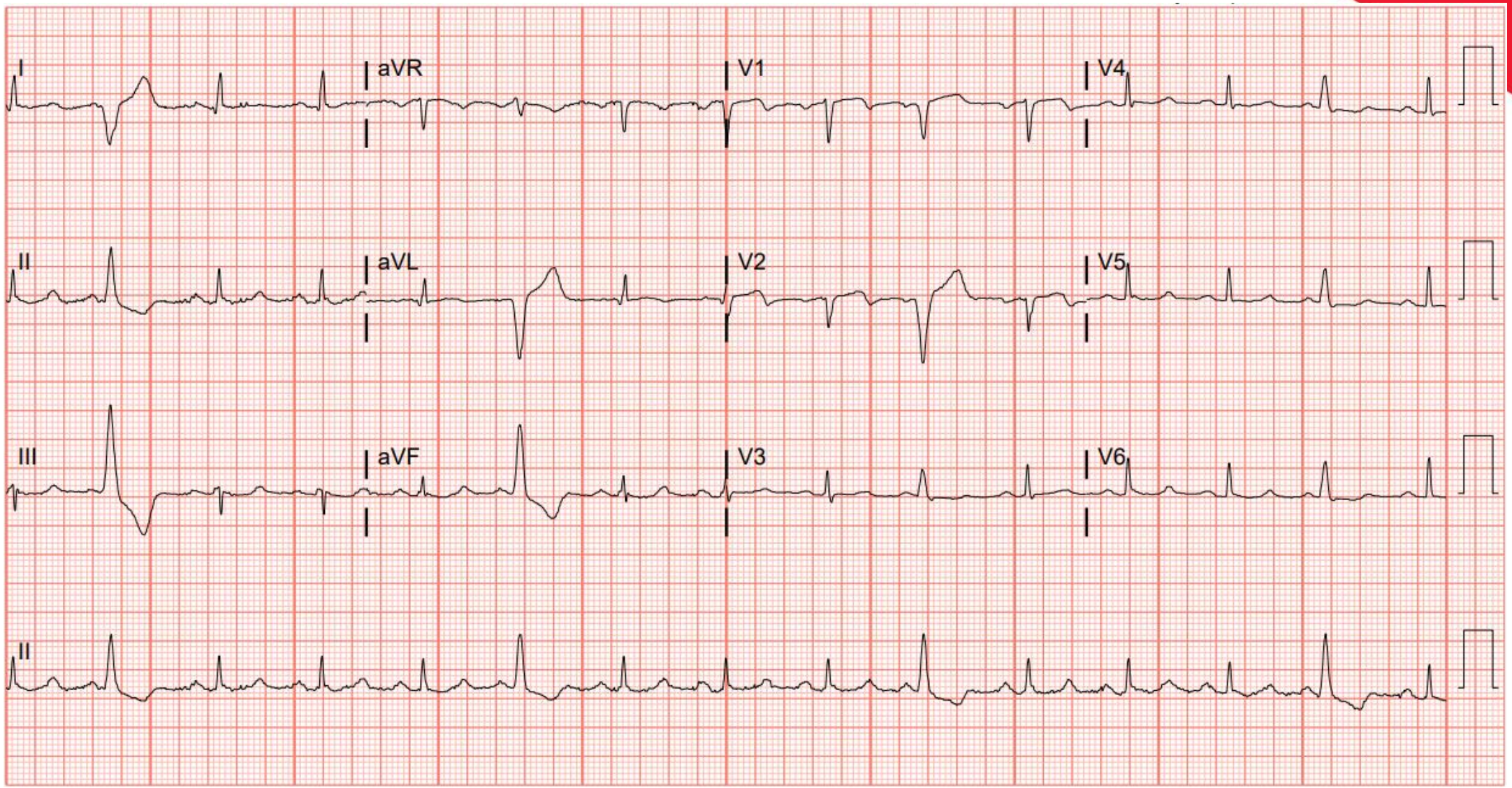
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Case #1: KR

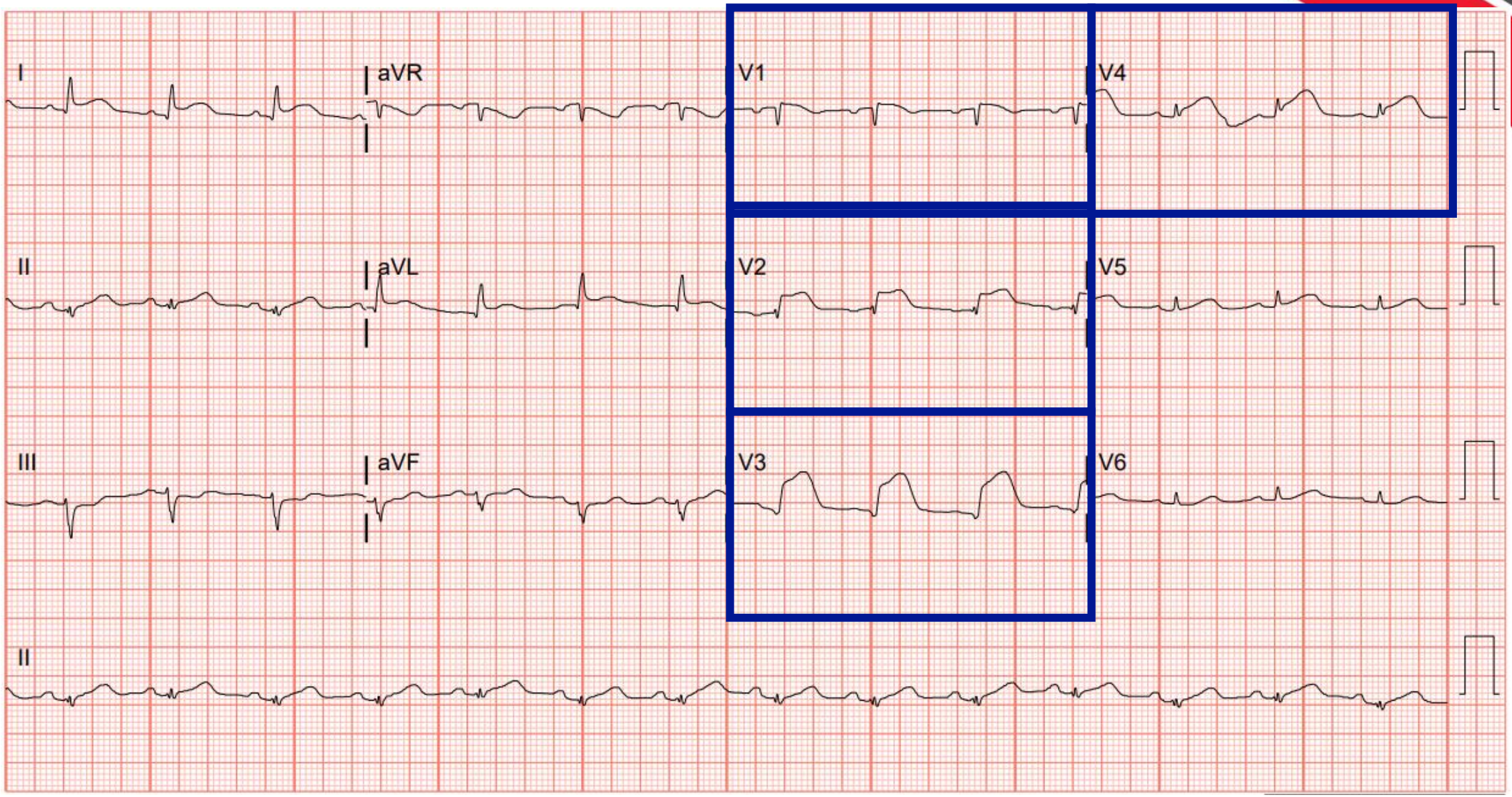
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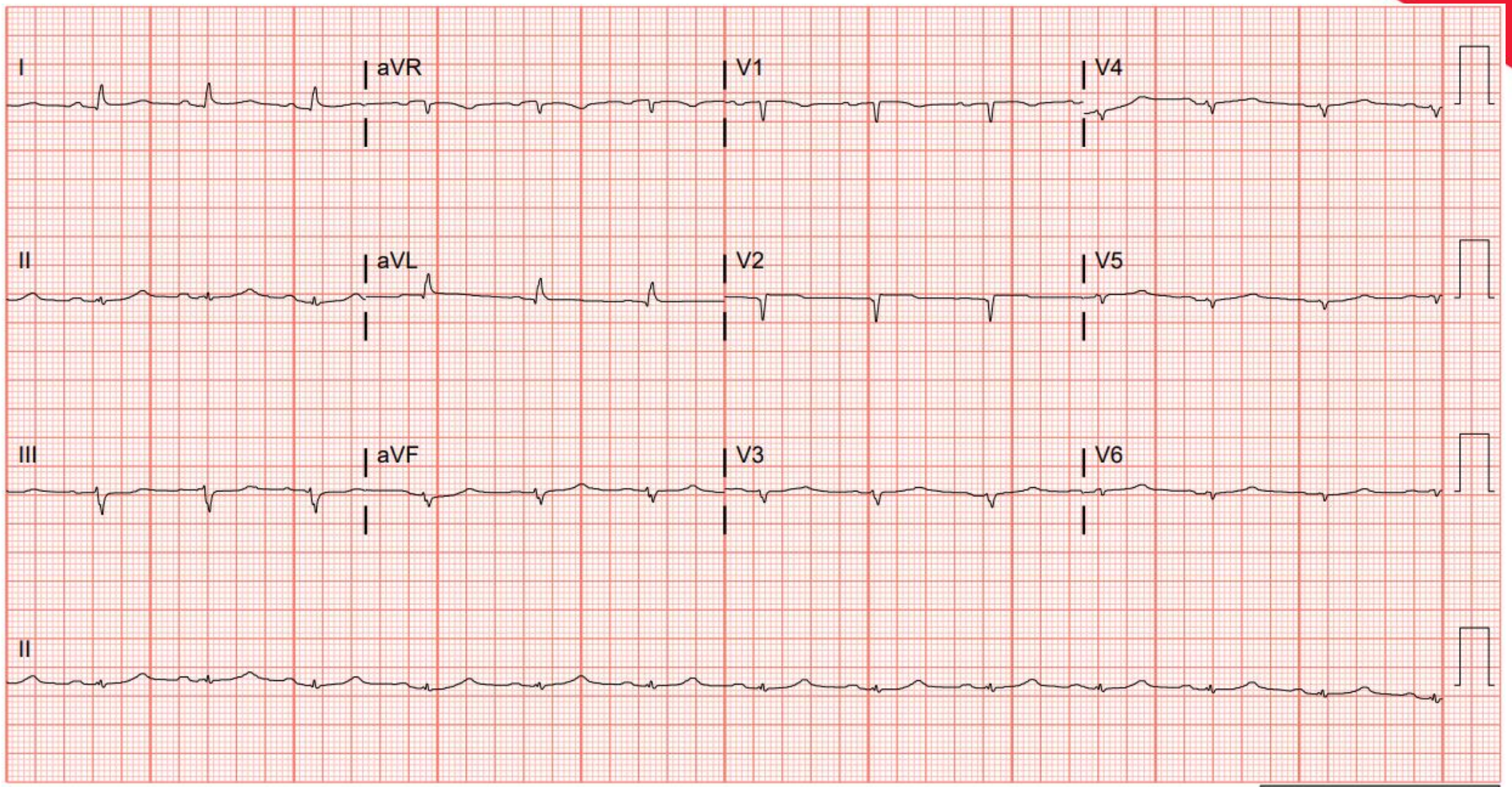


Case #2: CLS

1455 pm

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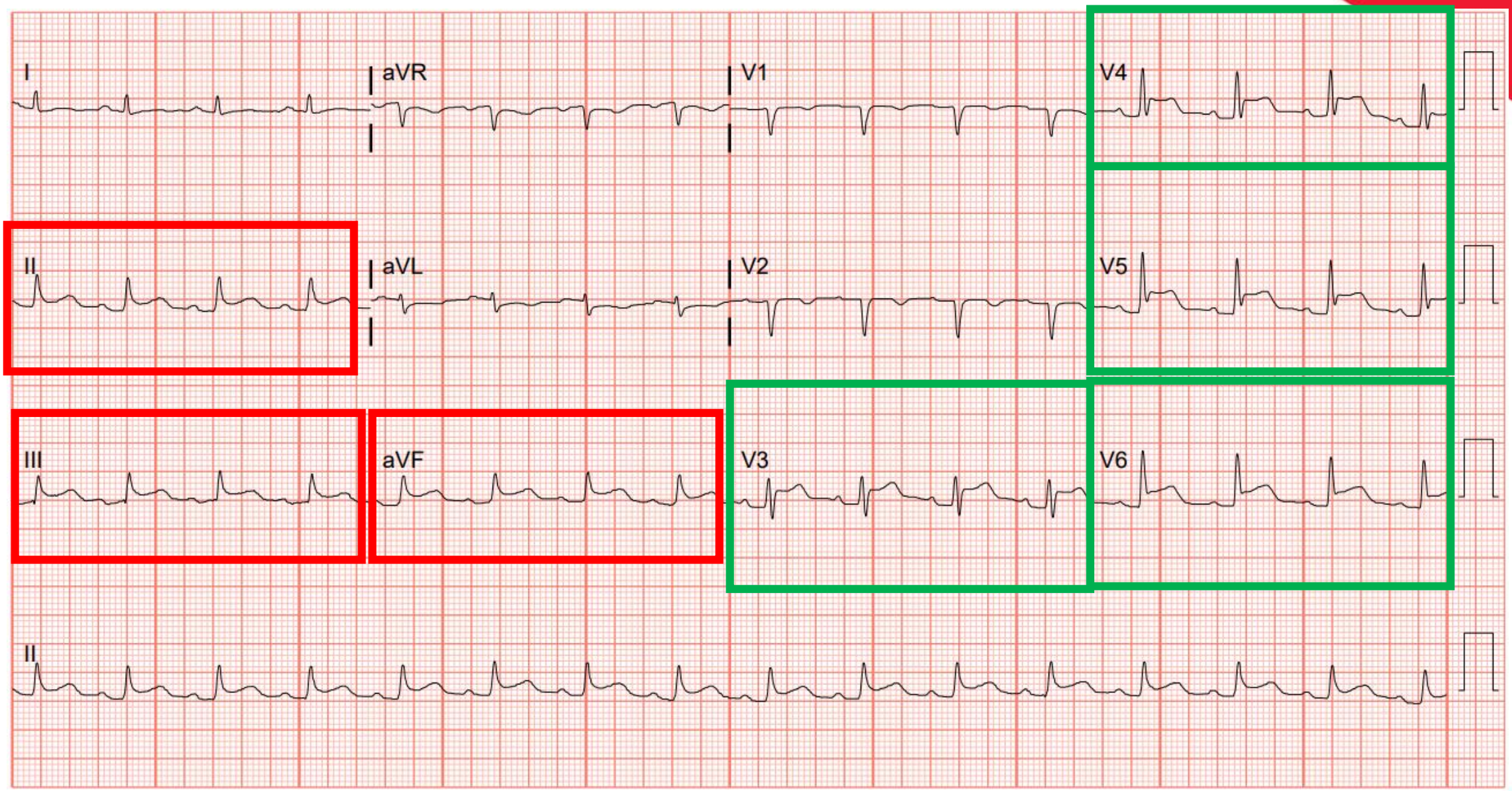
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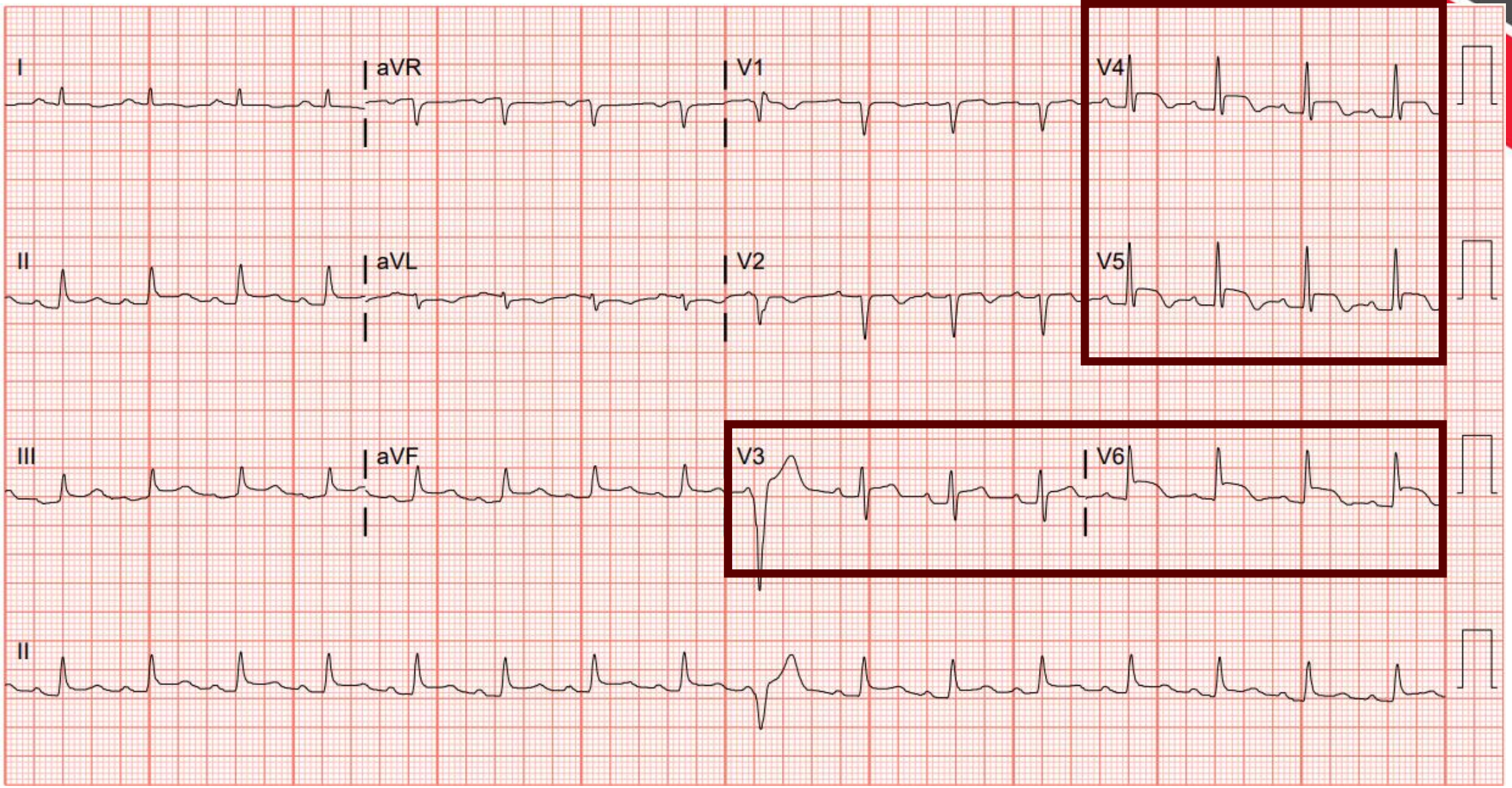
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Case #4: DB

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Treatment Options

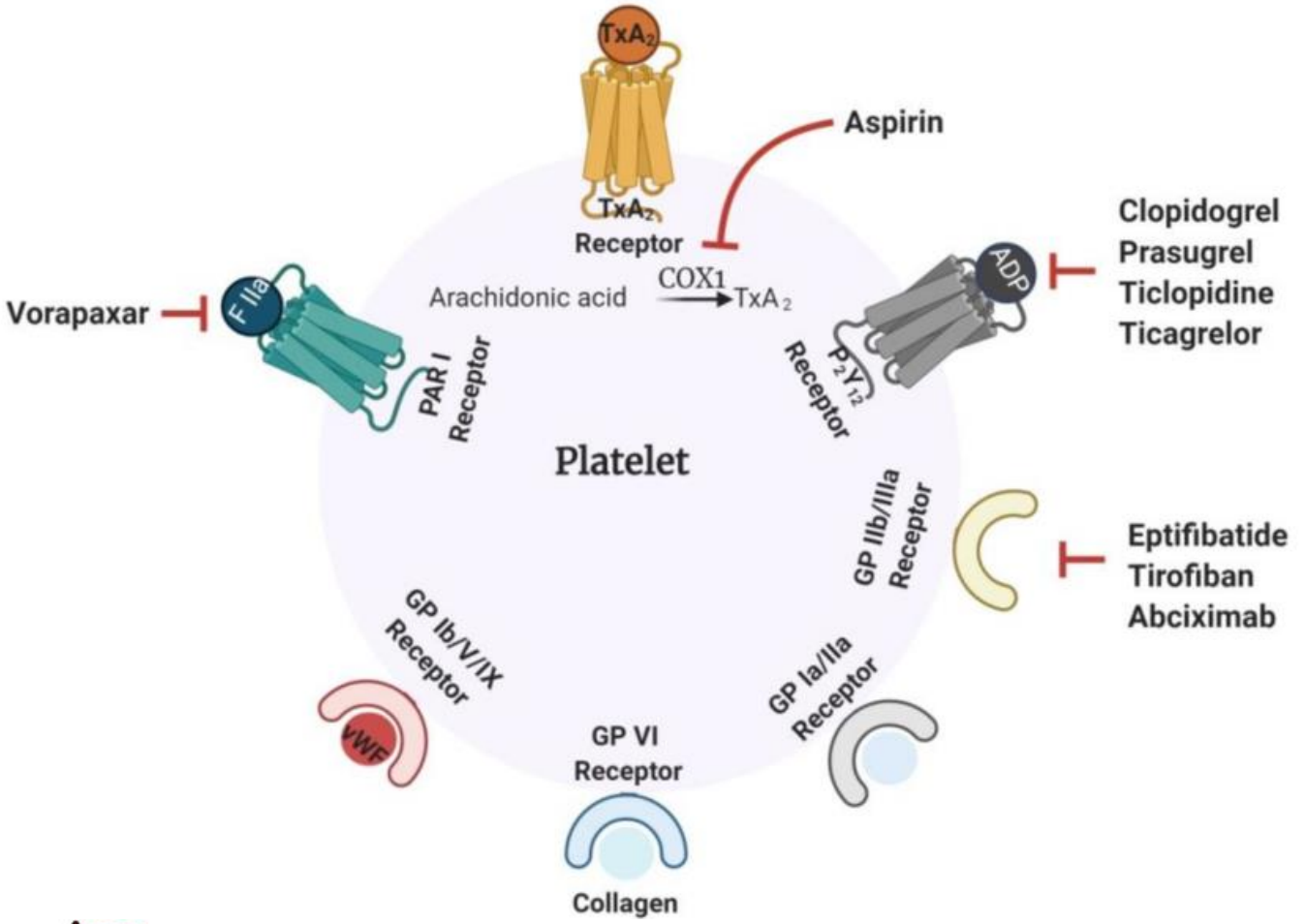
- "Cardiac Alert"
- Cath lab + Stents
- Cath lab – no stents
- Cath lab > CG Shock > devices > OR
- GOAL of door to balloon window
- Talk anti-plt, tPA indications (as they are different than PEs), Risk factor modification (BP, Lipids etc…)

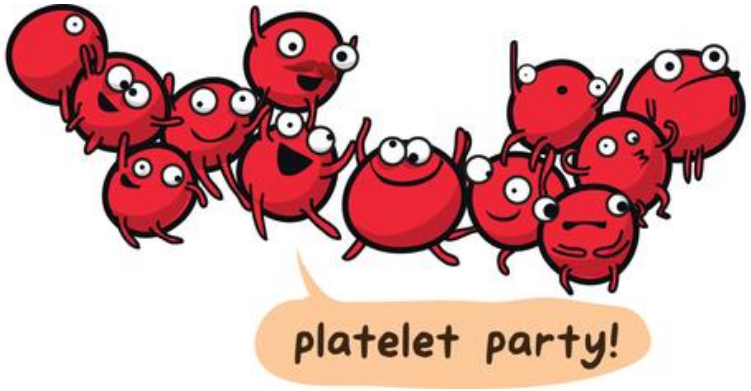
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Treatment Management

- CAD – treat medically vs clot buster vs stents
- CAD- differences between platelet inhibition
- Follow up Recommendations





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Aspirin Mechanism Of Antiplatelet Effect at Michael Schiller blog

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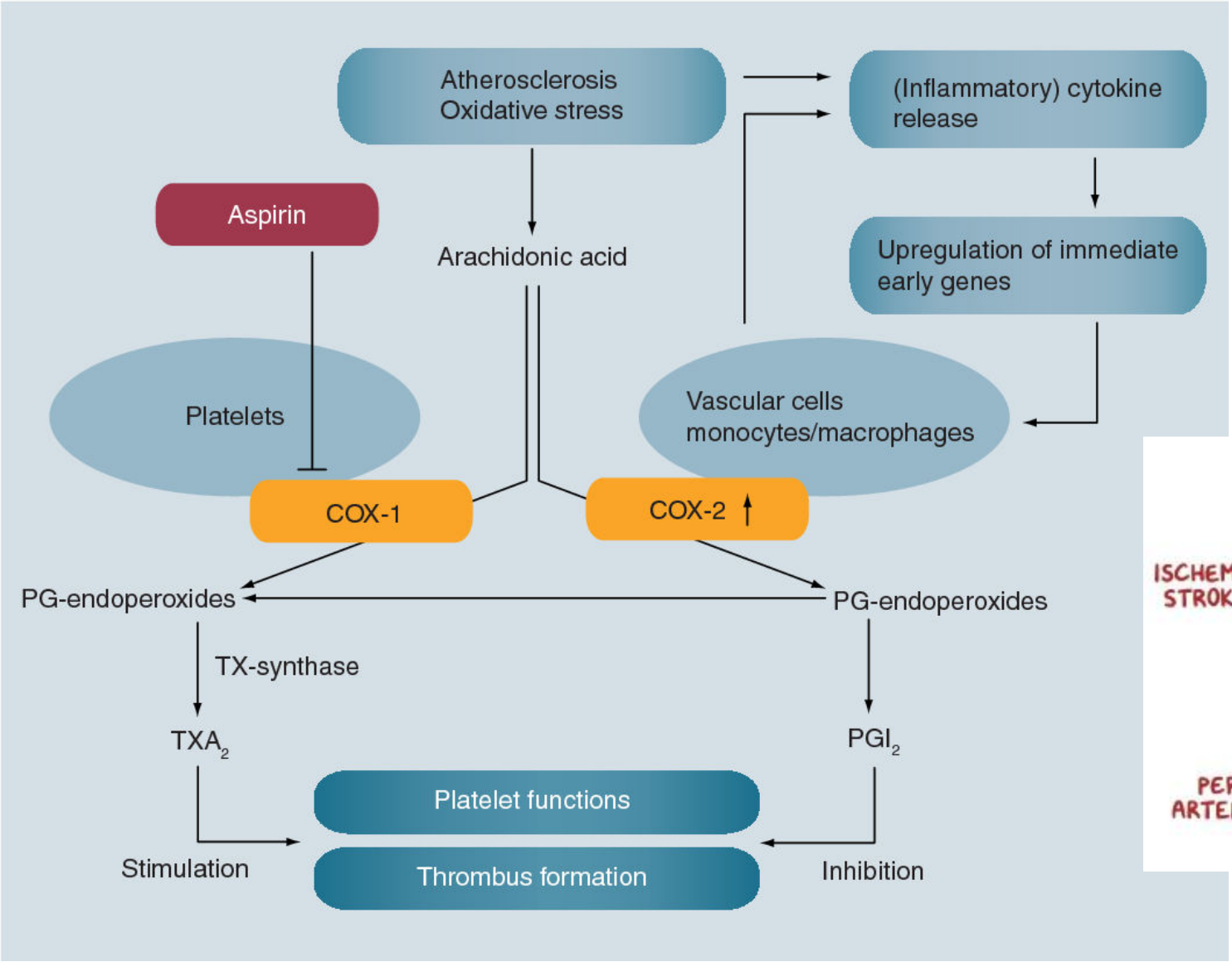
Fibrinolytic Agent	Dose
Tenecteplase (TNK-tPA)	Single IV weight-based bolus*
Reteplase (rPA)	Two 10-unit IV boluses given 30 min apart (administered over 2 min)
Alteplase (tPA)	90-min weight-based infusion†

Streptokinase is no longer available in the United States.

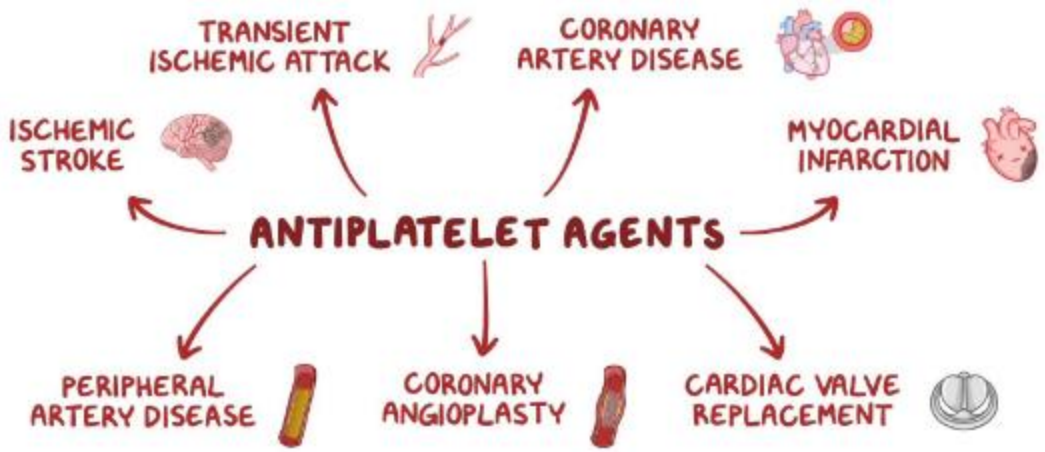
* 30 mg for weight <60 kg; 35 mg for 60-69 kg; 40 mg for 70-79 kg; 45 mg for 80-89 kg; and 50 mg for ≥90 kg.

† Adults ≥67 kg: 100-mg total dosage administered as a 15-mg IV bolus, followed by 50-mg IV infused over 30 min, and then 35-mg IV infused over the next 60 min. Adults <67 kg: 15-mg IV bolus, followed by 0.75 mg/kg IV (not to exceed 50 mg) infused over 30 min, and then 0.5 mg/kg IV (not to exceed 35 mg) over the next 60 min.

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Recommendation for Aspirin Referenced studies that support recommendations are summarized in the Evidence Table.		
COR	**LOE**	**Recommendation**
1	**A**	1. In patients with ACS, an initial oral loading dose of aspirin, followed by daily low-dose aspirin, is recommended to reduce death and MACE.[1–5]



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Aspirin Mechanism Of Antiplatelet Effect at Michael Schiller blog

Anti-Platelet Agent Considerations

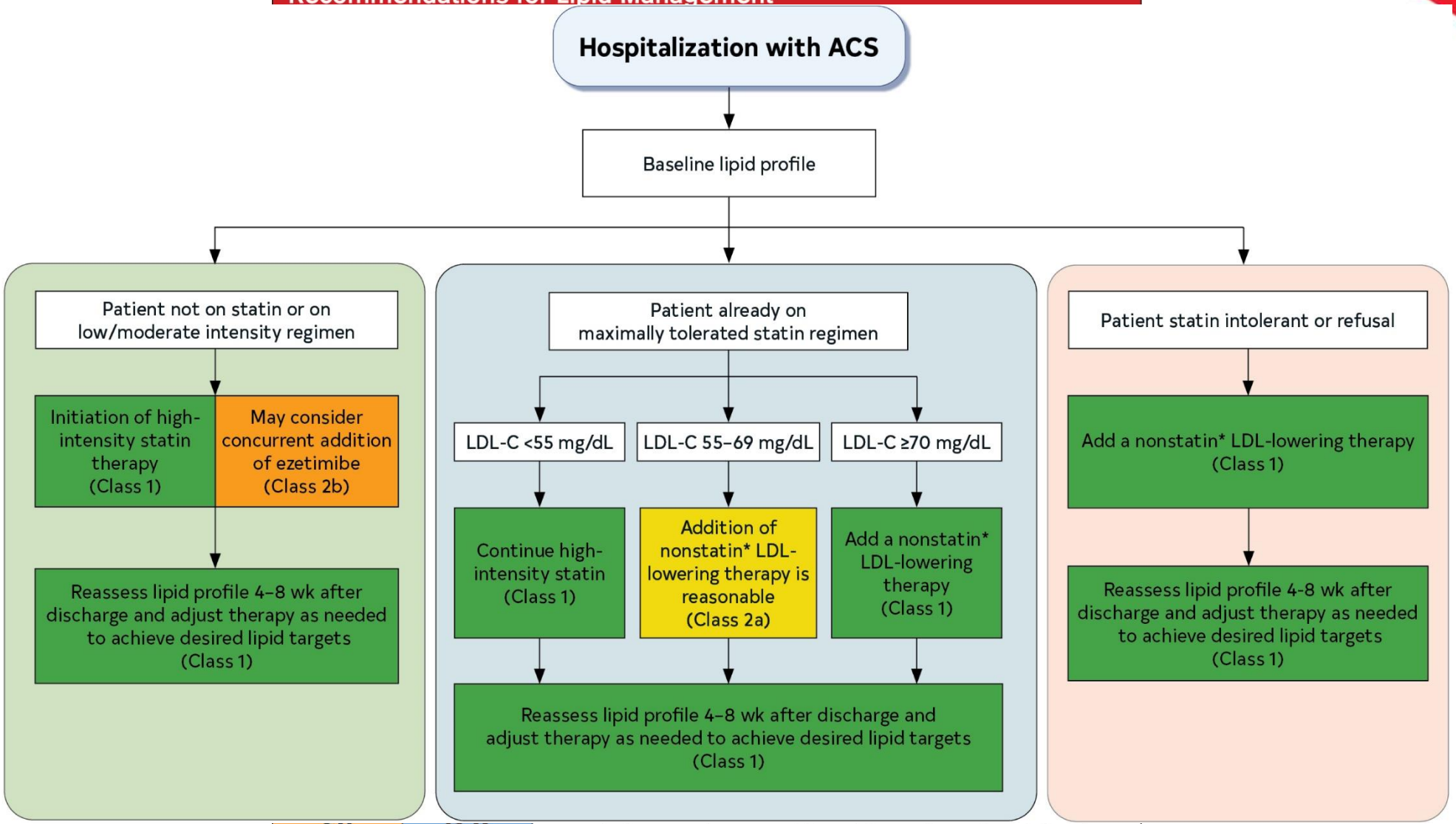
Agent	Setting	Dosing Considerations
Aspirin	NSTE-ACS or STEMI	Loading dose 162-325 mg orally. Aspirin (nonenteric coated) should be chewed, when possible, to achieve faster onset of antiplatelet action. Loading dose should be administered for patients already on aspirin therapy. Maintenance dose 75-100 mg orally daily (nonenteric coated)
Clopidogrel	NSTE-ACS or STEMI without fibrinolytic	Loading dose 300 or 600 mg orally Maintenance 75 mg orally daily
	STEMI with fibrinolytic	Loading dose 300 mg orally if age ≤75 y; Initial dose 75 mg orally if age >75 y Maintenance 75 mg orally daily
Prasugrel	NSTE-ACS or STEMI without fibrinolytic, and undergoing PCI	Loading dose 60 mg orally Maintenance dose 10 mg orally daily if body weight ≥60 kg and age <75 y Maintenance dose 5 mg orally daily if body weight <60 kg or age ≥75 y (use caution)
Ticagrelor	NSTE-ACS or STEMI without fibrinolytic	Loading dose 180 mg orally Maintenance dose 90 mg orally twice daily

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Recommendations for Lipid Management



statin may be considered to reduce the risk of MACE.[5]

*Modified or

†adapted from the "2023 AHA/ACC/ACCP/ASPC/NLA/PCNA Guideline for the Management of Patients With Chronic Coronary Disease."[14]

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Statin Classification by Expected LDL-C Reduction

High-Intensity Therapy*	Moderate-Intensity Therapy†	Low-Intensity Therapy‡
Atorvastatin 40-80 mg Rosuvastatin 20-40 mg	Atorvastatin 10-20 mg Rosuvastatin 5-10 mg Simvastatin 20-40 mg Pravastatin 40-80 mg Lovastatin 40 mg Fluvastatin XL 80 mg Fluvastatin 40 mg BID Pitavastatin 1-4 mg	Simvastatin 10 mg Pravastatin 10-20 mg Lovastatin 20 mg Fluvastatin 20-40 mg

Recommendation for Reassessment of Lipid Levels Postdischarge

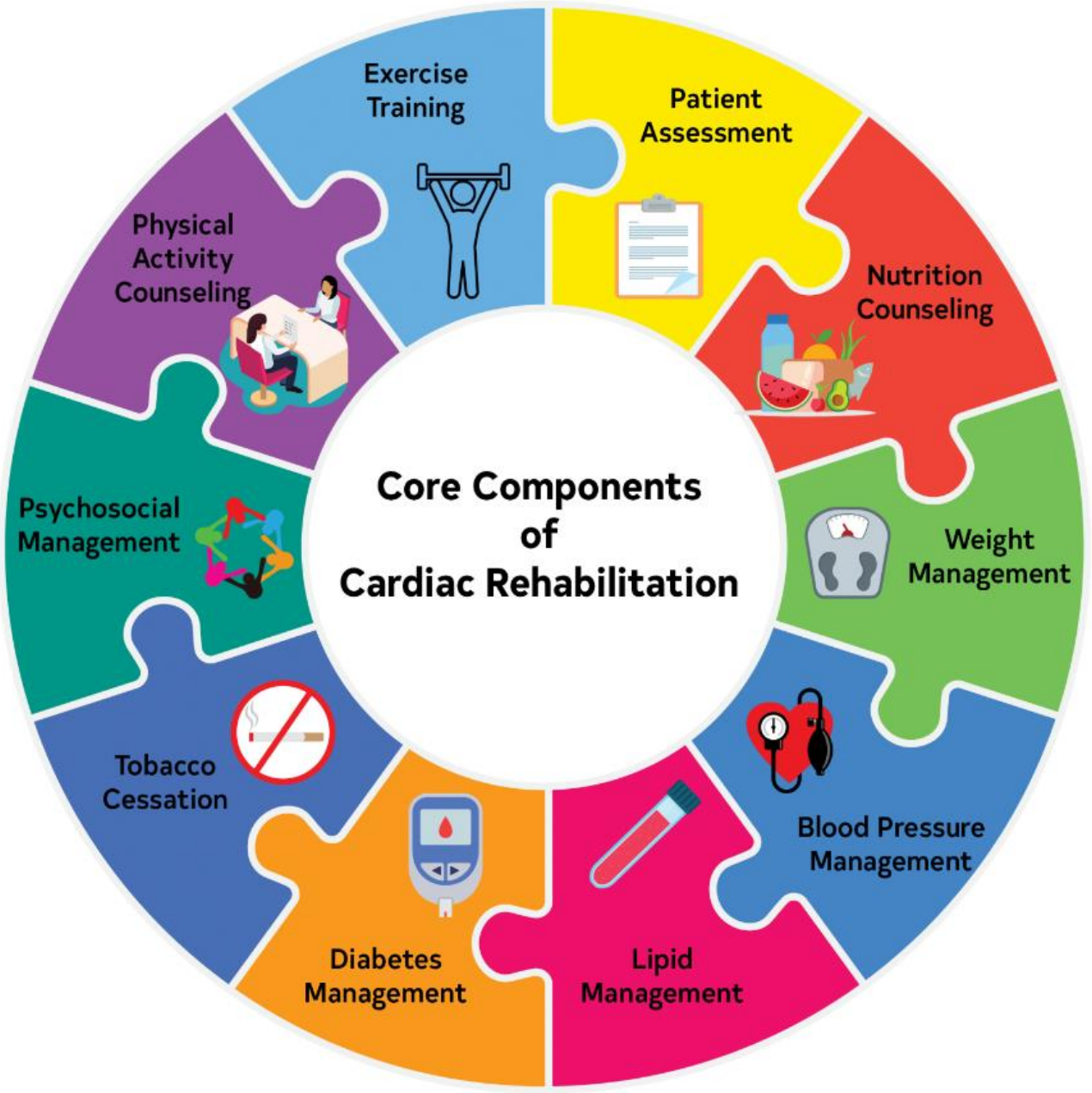
COR	LOE	Recommendation
1	C-LD	1. In patients after ACS, a fasting lipid panel is recommended 4 to 8 weeks after initiation or dose adjustment of lipid-lowering therapy to assess response or adherence to therapy.*[1,2]

*Modified from the "2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Blood Cholesterol."[3]

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Blood Pressure Categories

American Heart Association.

BLOOD PRESSURE CATEGORY	SYSTOLIC mm Hg (upper number)		DIASTOLIC mm Hg (lower number)
NORMAL	LESS THAN 120	and	LESS THAN 80
ELEVATED	120 – 129	and	LESS THAN 80
HIGH BLOOD PRESSURE (HYPERTENSION) STAGE 1	130 – 139	or	80 – 89
HIGH BLOOD PRESSURE (HYPERTENSION) STAGE 2	140 OR HIGHER	or	90 OR HIGHER
HYPERTENSIVE CRISIS (consult your doctor immediately)	HIGHER THAN 180	and/or	HIGHER THAN 120



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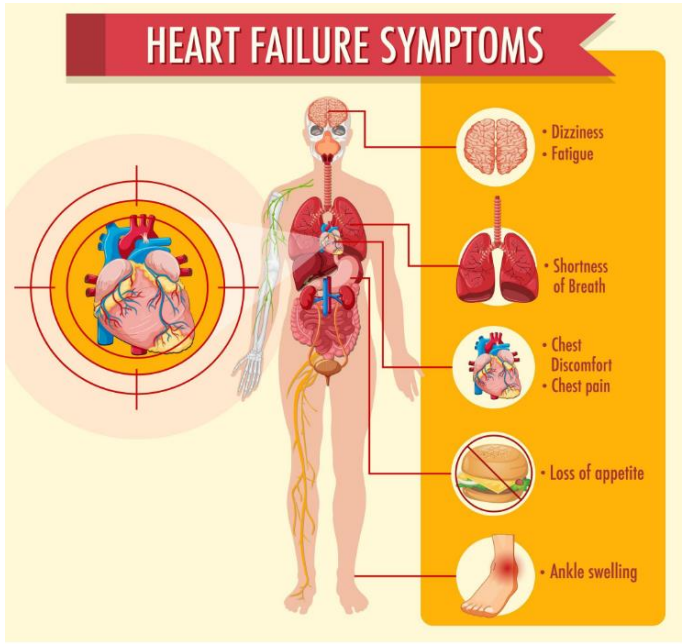
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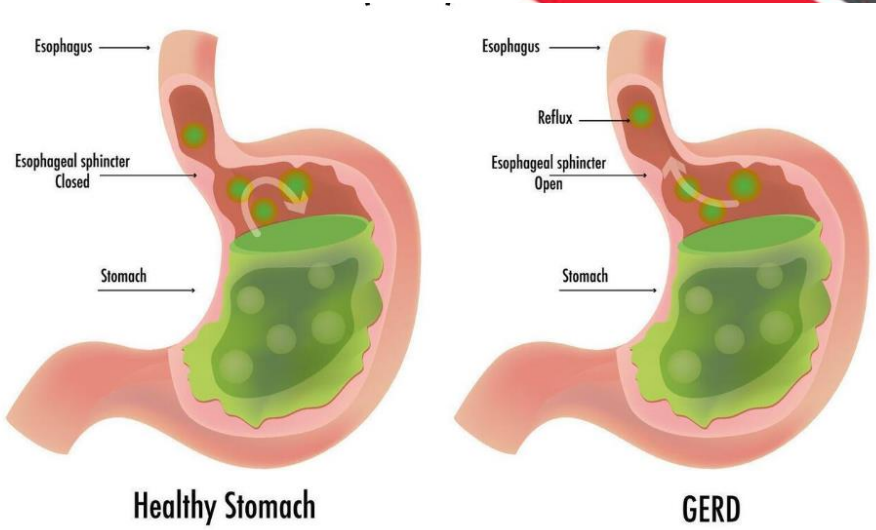
2025 ACC/AHA/ACEP/NAEMSP/SCAI Guideline for the Management of Patients With Acute Coronary Syndromes: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines | Circulation

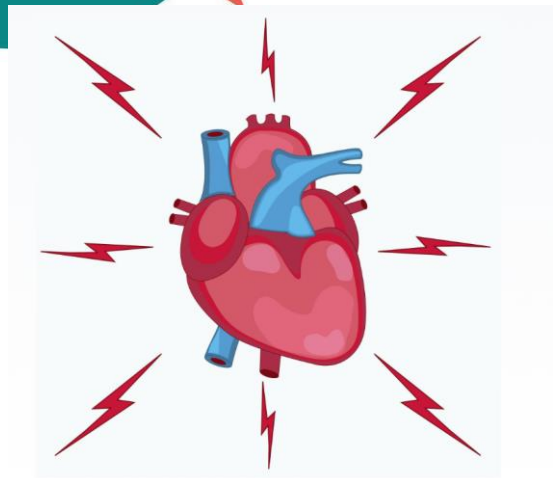
Target: BP™ program helps more than 8 million Americans with hypertension improve heart health | American Heart Association

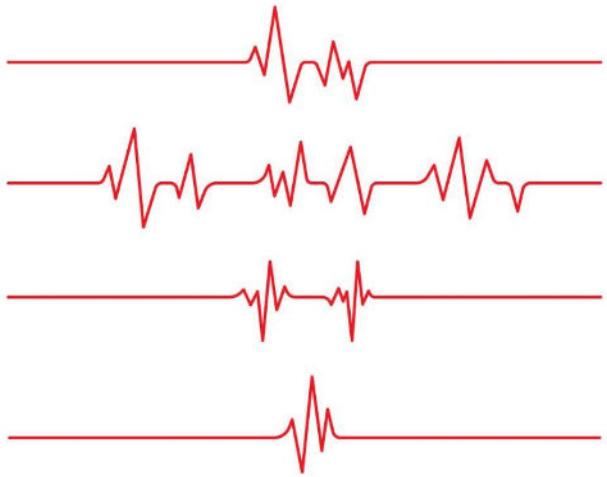
Final PEARLS…

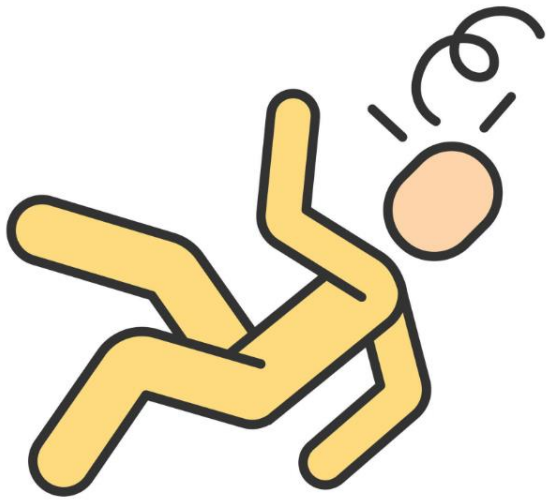












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Questions?



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