Chest Pain Protocols and Evolving ECG Standards

Eileen Couture, DO, FACEP, RN, MS

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DISCLOSURE

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Chest Pain: What comes next?

Objectives:

- To review pathophysiology and etiology of acute ischemia
- To review current standards for treating acute chest pain regarding concern for acute ischemia
- To review an update on treatment therapies and management of STEMI
- To review EKG changes that could be concerning in a setting a patient with chest pain
- To review other causes of chest pain which demonstrate acute EKG changes

Case 1

- You are making rounds in the infirmary when a 68-year-old patient complains of chest pain. The pain started at 5 am and the night nurse administered one aspirin. She ordered an EKG and took the patients vital signs (normal).
- EKG demonstrates ST depression in leads V2 and V3.
- The patient's pain has now resolved. What comes next?

Case 2

- A 56-year-old female presents and complains of constant pain in the left upper chest for the past two days. Denies dyspnea, diaphoresis or radiation of the pain to her arm.
- An EKG is completed, and it demonstrates ST segment elevation in inferior and lateral leads.
- What should be done next?

Chest Pain

- Failure to recognize MI is dangerous
- Generally, patients do not start seeking healthcare until 1.5 to 2 hours into the process
- Transport to the ED by EMS allows for earlier delivery of ultimate reperfusion (Canto, et. al, 2002; Mathews et al, 2011)
- ACS (acute coronary syndrome) includes:
  - STEMI
  - NSTE ACS (70%)
  - NSTEMI
- Plaques vulnerable to disruption leads to exposure to circulating platelets, platelet adhesion, activation and aggregation

Chest Pain

- With in minutes of the onset of infarction, alterations in the electrical potential of the cardiac myocytes which is see on the EKG.
- Becomes a predictable pattern
- Location of the infarction is noted on the EKG
- Prior to infarction EKG may demonstrate ST depression or T wave inversion…..warning changes

Evaluation

- EKG (10 minutes) + history + physical exam (vital signs)

- More likely: historical features increase likelihood:
  - Radiation to shoulder arms, increase pain with exertion, diaphoresis, vomiting, weakness & dyspnea, syncope

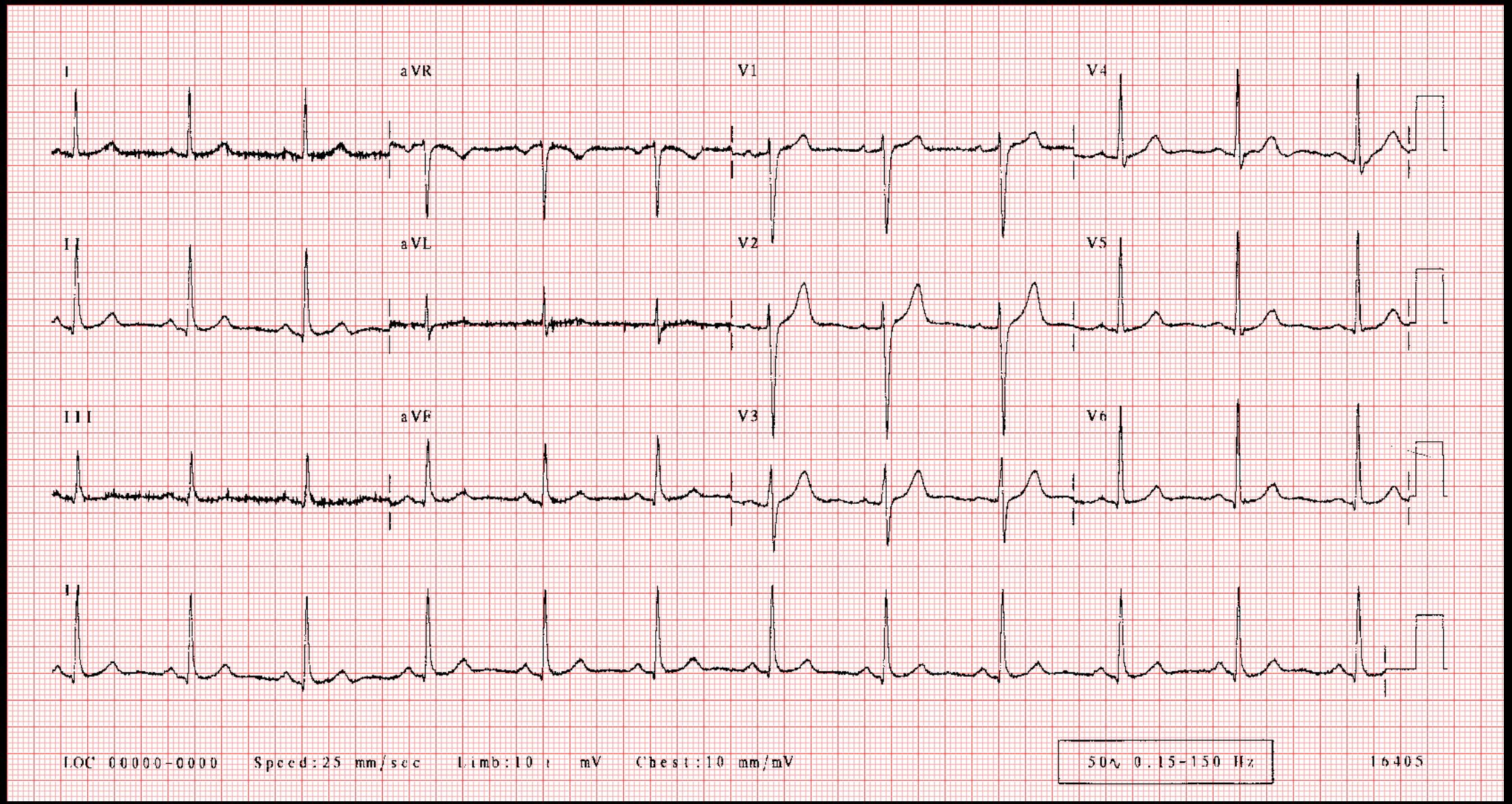
- Thrombolytic therapy option if EMS or transport is not possible
- Management of vital signs: cardiogenic shock
  - Inadequate tissue perfusion, more unstable the patient becomes

Chest pain

- Concern for STEMI, or NSTEMI: load and go
- If the patient stays:
- Monitored
- Serial EKGs **** and troponins
- Chest xray (rule out other causes)
- Aspirin, nitro, hep lock
- Window of suspicion and delay in care, influences mortality

Transport

- Goals: Transport safely by EMS
- Early recognition
- Administer aspirin
- Timely transport to the right center (PTCA) < 30 minutes
- Goal time: < 90 minutes first medical contact to balloon
- Practice of bypassing a non-PCI capable facility is safe and recommended
- Handoff communication: send your EKG


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
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Chest:10 mm/mV
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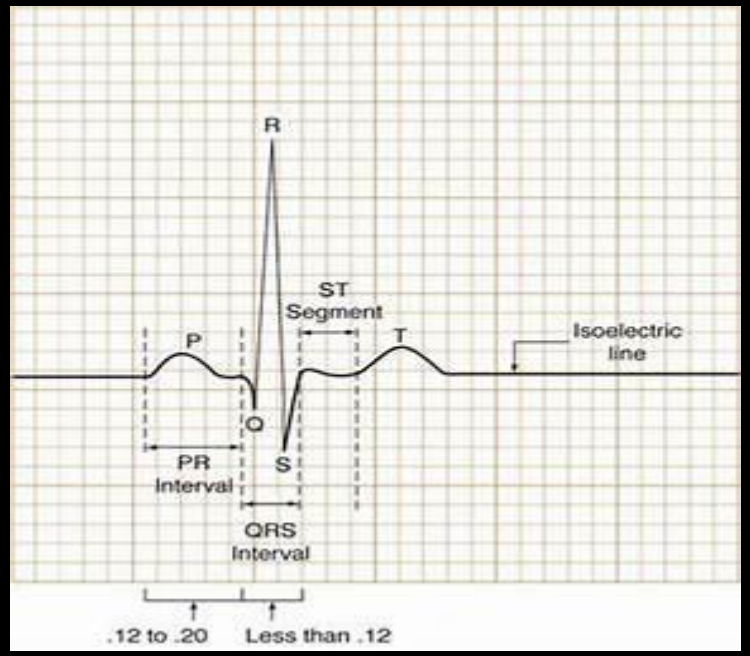
EKG changes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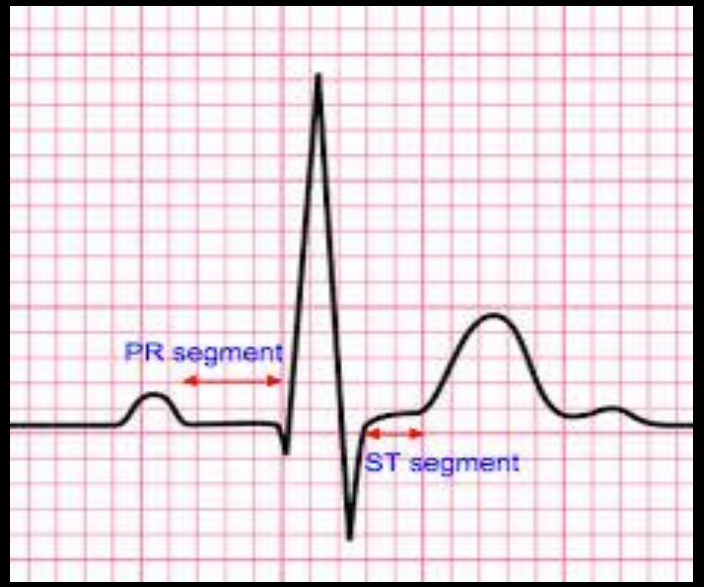
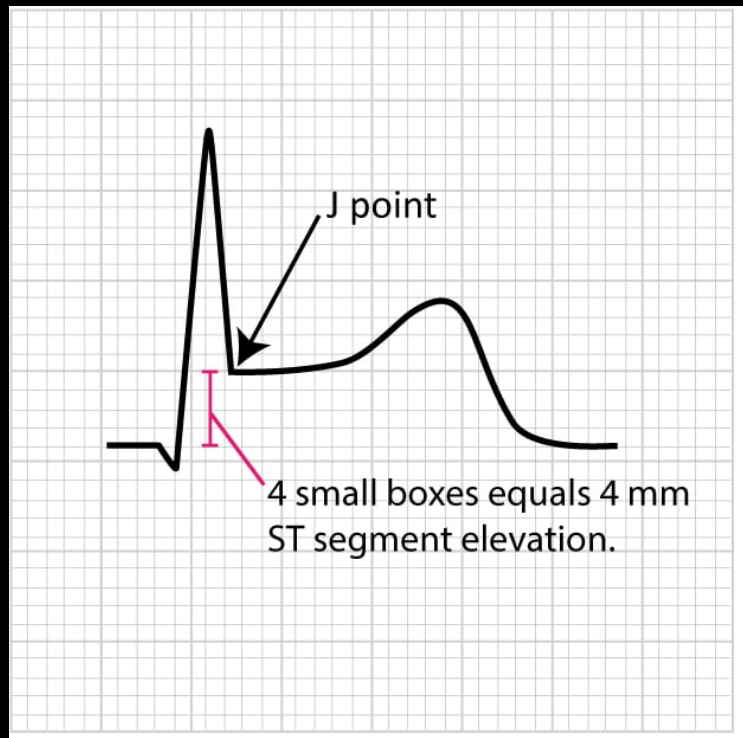
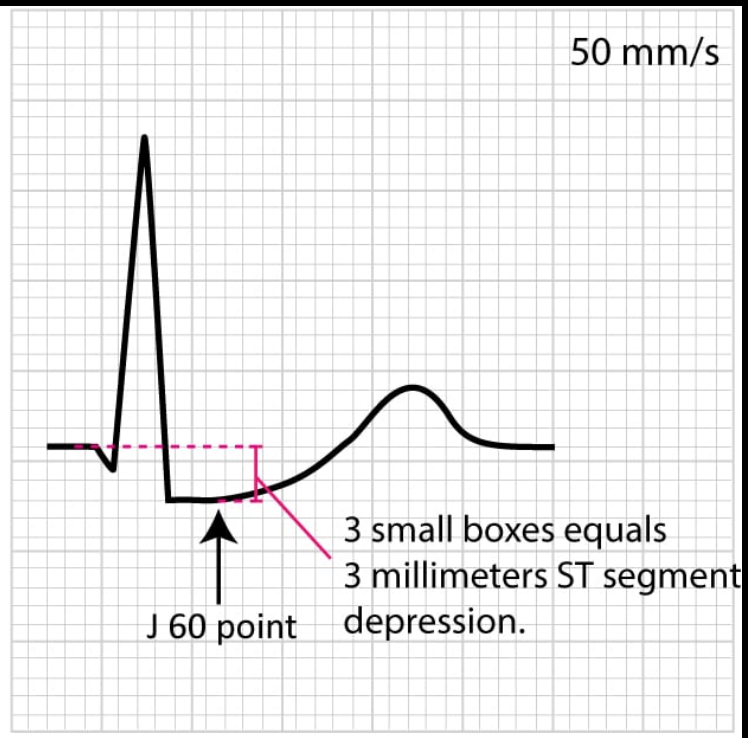

PR segment
ST segment

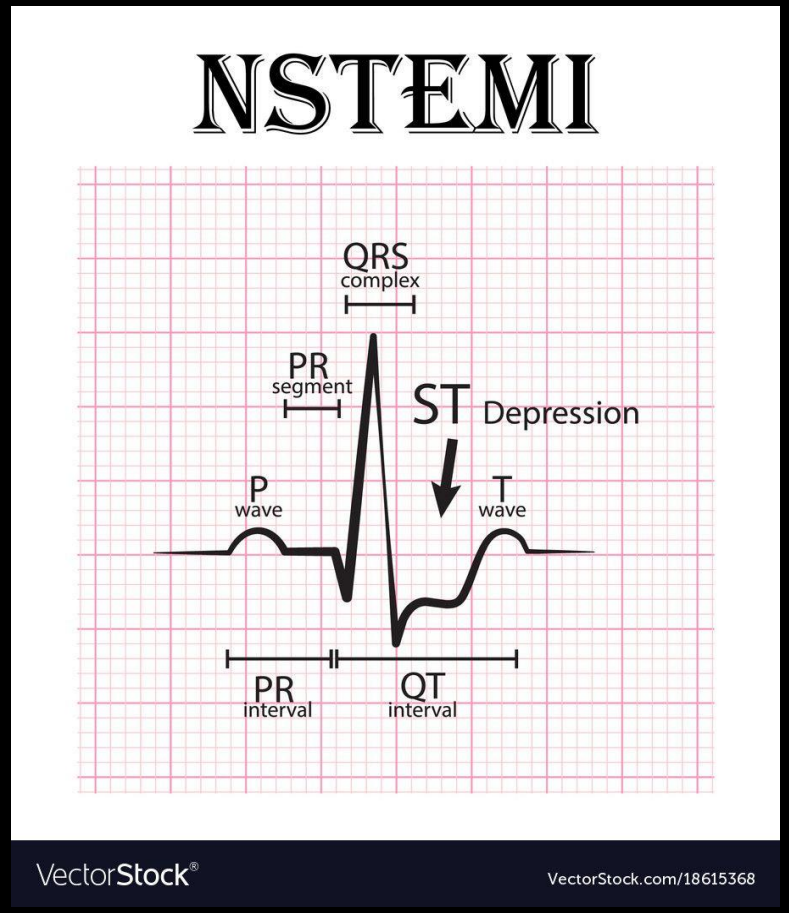
EKG changes





At paper speed 50 mm/s the J 60 point is located 3 small boxes after the J-point. At paper speed 25 mm/s the J 60 point is located 1,5 small boxes after the J point.

EKG changes


NSTEMI
QRS
complex
PR
segment
ST Depression
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wave
PR
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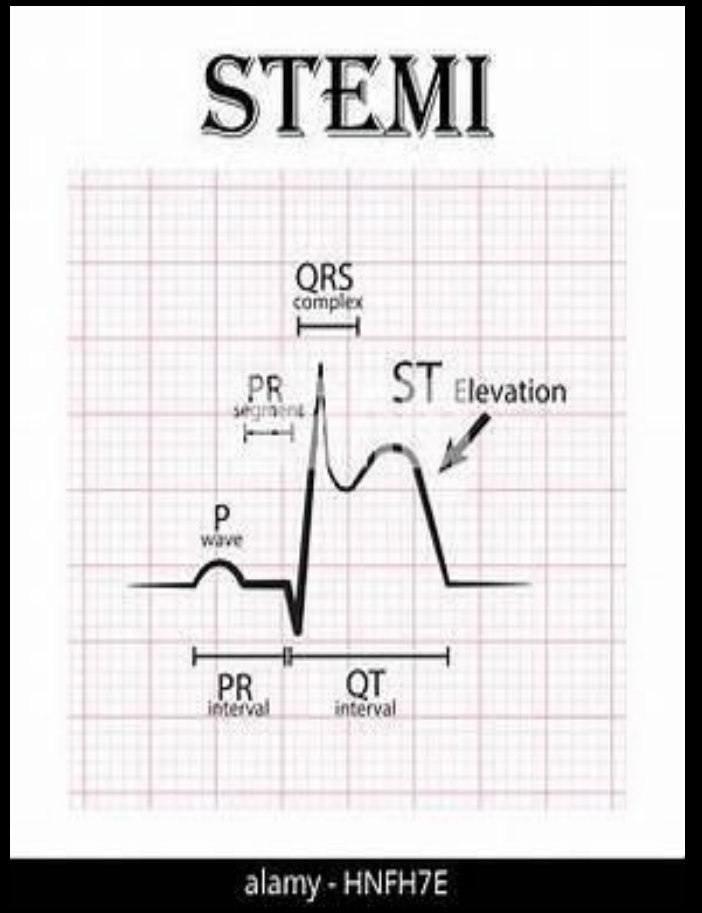

STEMI
QRS
complex
PR
segment
ST Elevation
P
wave
PR
interval
QT
interval
alamy - HNFH7E

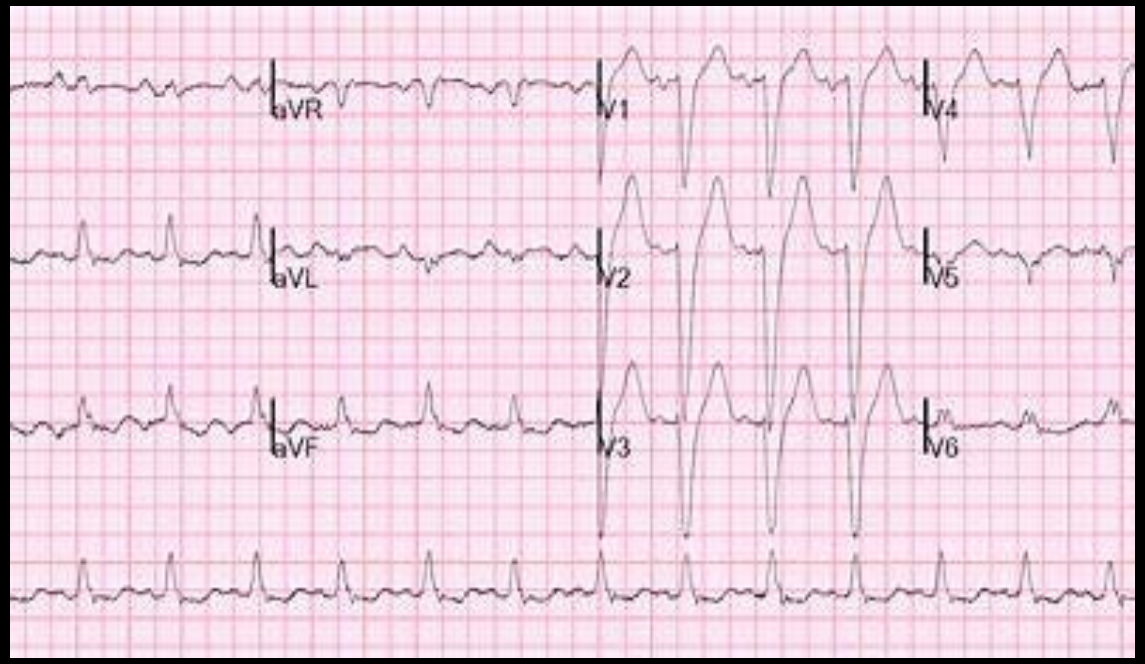
EKG Changes

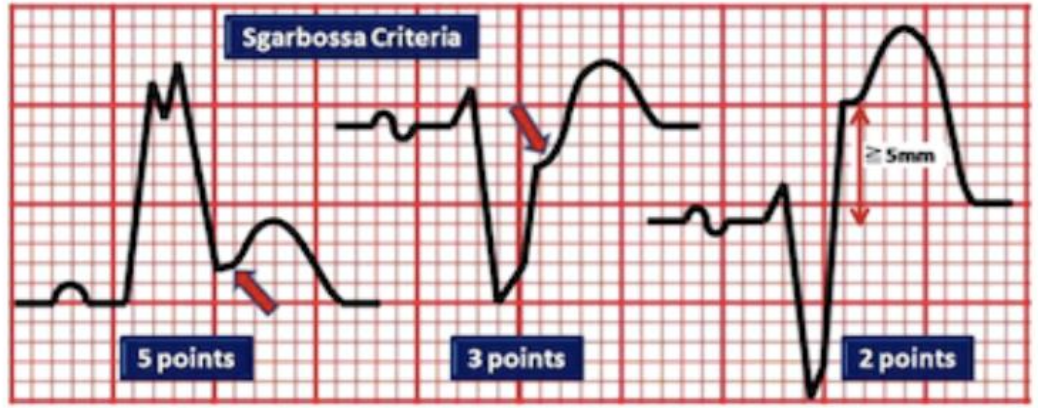
- Get an old EKG when possible

- New ST elevation at the J point in 2 contiguous leads
  - > 2 mm in men > 40 years old
  - >2.5 mm in men <40 years old
  - > 1.5 mm in women of any age
- ST reciprocal changes (PAILS): ST depression that mirror the ST elevation
  - Posterior STEMI-→Anterior changes
  - Anterior STEMI→Inferior changes
  - Inferior STEMI→Lateral changes
  - Lateral STEMI→septal/inferior changes
  - Septal STEMI→posterior changes

- Serial EKGs:
  - May see dynamic changes at 15- & 30-minute intervals for the first 1-2 hours
- Fast and furious:
- ST depression in two leads (not V1 or aVR)
- STE in III is > II
- Horizontal or conves upward of STE
- Absent PR depression

EKG changes: Bundle Branch Block

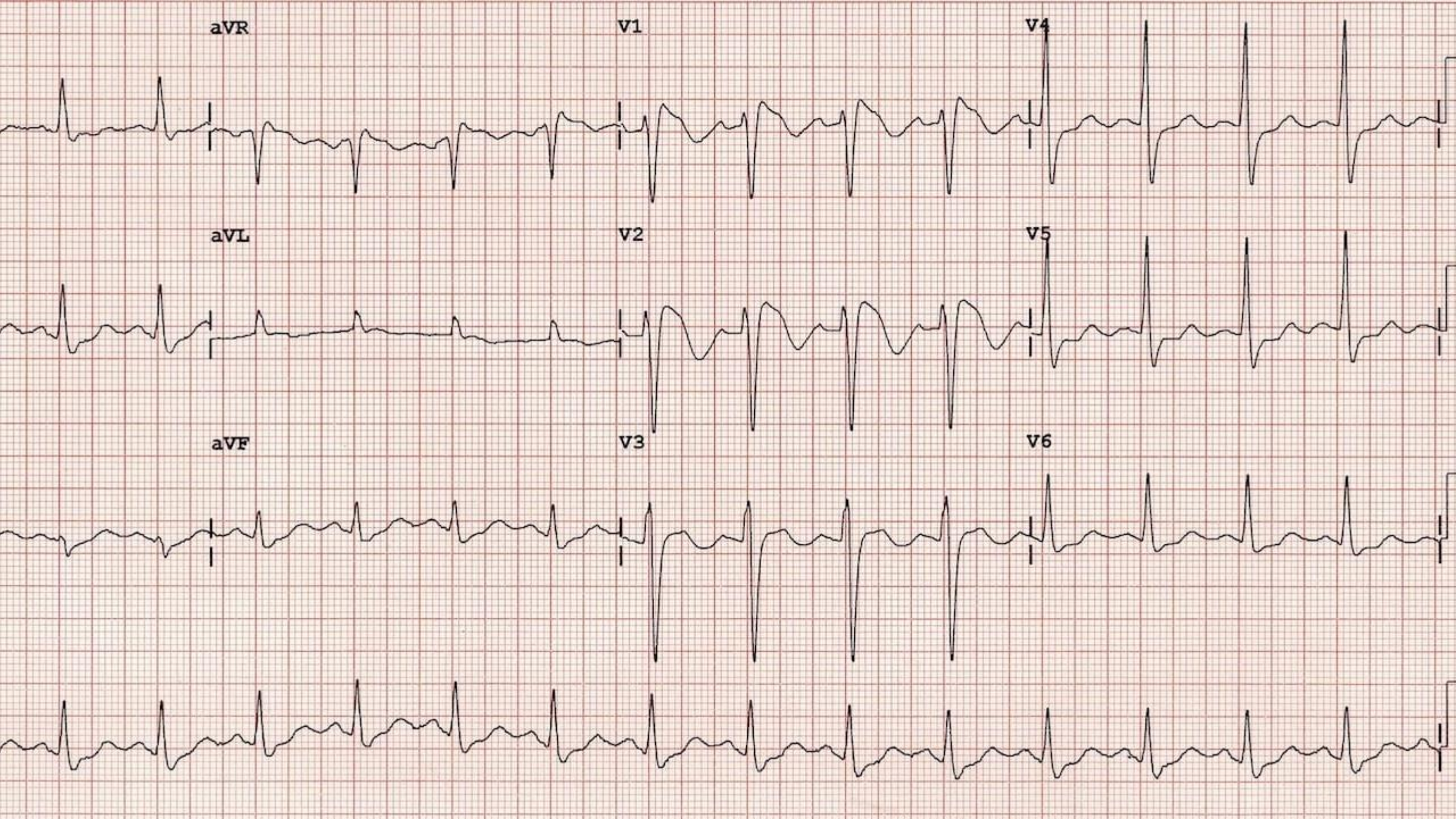




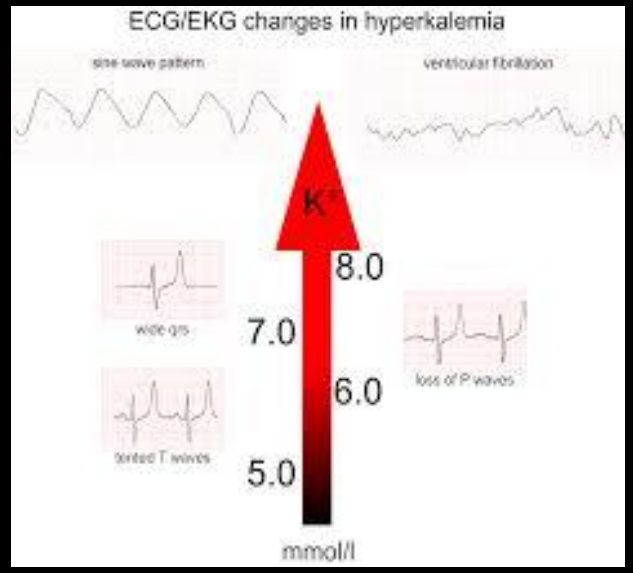
Sgarbossa ECG Criteria for LBBB	
Concordant STE ≥1mm	5 points
STD ≥1mm in V1 – V3	3 points
Discordant STE ≥5mm	2 points

EKG clues to treatment needs

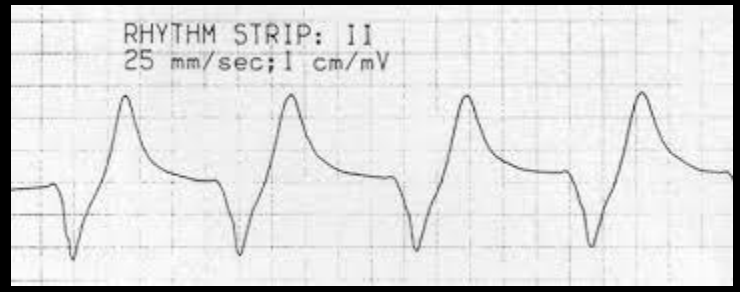
MI	ST elevation in anatomically contiguous leads, with reciprocal changes
Pericarditis	Diffuse ST elevation with concave morphology No ST segment depression or reciprocal changes
Myocarditis	ST changes may resemble acute ischemia
Electrolyte (hyperkalemia)	Tall, Symmetric, peaked T waves
Left bundle branch block	Discordant ST segment elevation typically see in V1-V3
Brugada syndrome	ST segment elevation > 2mm in >1 of V1-3 leads followed by negative T wave inversion
Hypothermia (Osborn waves)	Positive deflection at the J wave, commonly in precordial leads; core temp 30. C
Early repolarization	Notching at the J point


aVR
V1
V4
aVL
V2
V5
aVF
V3
V6

Hyperkalemia

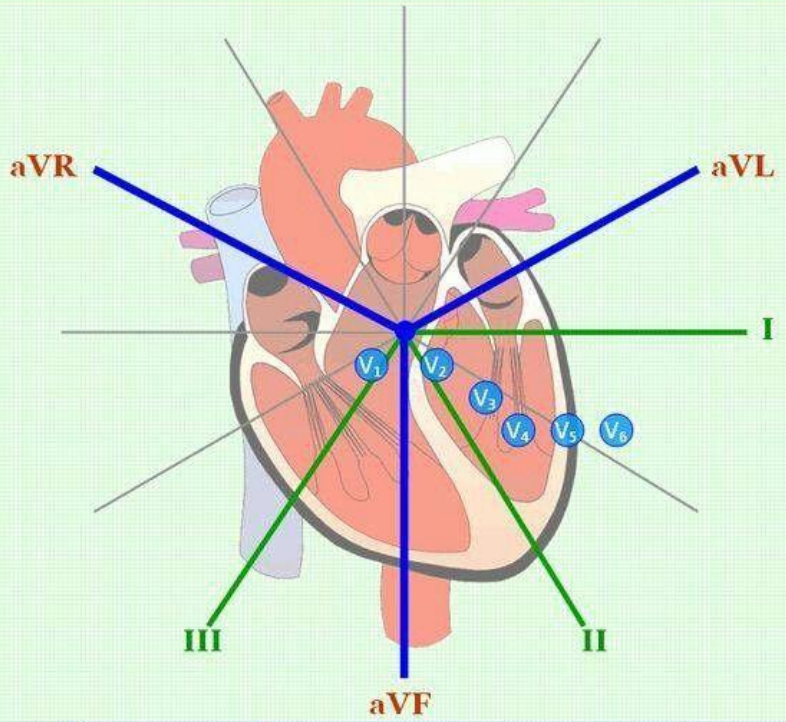


Serum Potassium	Typical ECG Appearance	Possible ECG Abnormalities
Mild (5.5-6.5 mEq/L)		Peaked T waves Prolonged PR segment
Moderate (6.5-8.0 mEq/L)		Loss of P wave Prolonged QRS complex ST-segment elevation Ectopic beats and escape rhythms
Severe (> 8.0 mEq/L)		Progressive widening of QRS complex Sine wave Ventricular fibrillation Asystole Axis deviations Bundle branch blocks Fascicular blocks

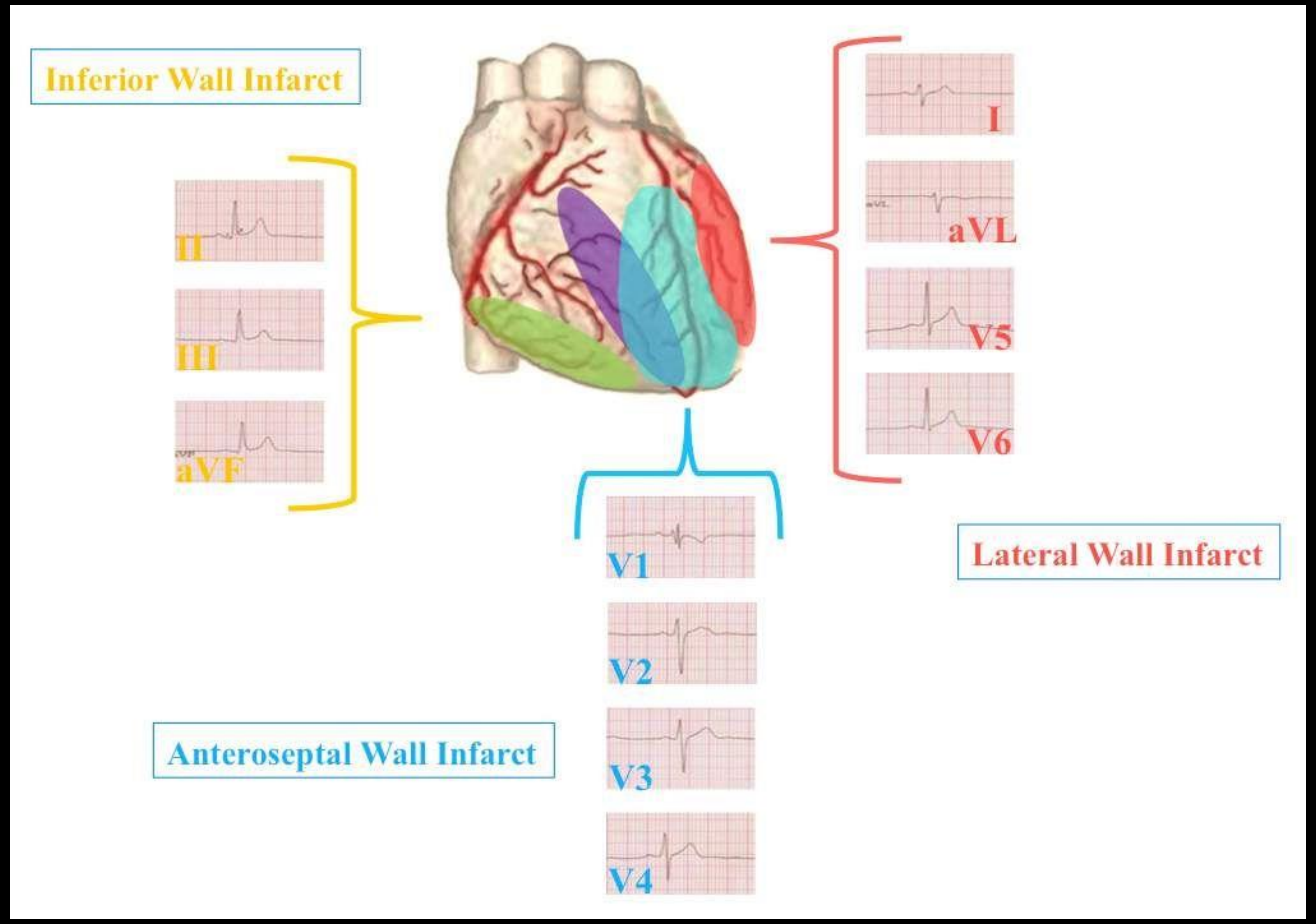


Lead Summary

I Lateral Circumflex Artery	aVR	V1 Septal Left Anterior Descending Artery	V4 Anterior Right Coronary Artery
II Inferior Right Coronary Artery	aVL Lateral Circumflex Artery	V2 Septal Left Anterior Descending Artery	V5 Lateral Circumflex Artery
III Inferior Right Coronary Artery	AVF Inferior Right Coronary Artery	V3 Anterior Right Coronary Artery	V6 Lateral Circumflex Artery



Anterior Wall	V2 to V4	Left Anterior Descending Artery (LAD) –Diagonal branch
Septal Wall	V1, V2	Left Anterior Descending Artery (LAD) –Septal Branch
Lateral Wall	aVL, I, V5, V6	Left Coronary Artery (LCA)–Circumflex Branch
Inferior Wall	II, III, aVF	Right Coronary Artery (RCA)–Posterior Descending Branch
Posterior Wall	V1 to V4	Left Coronary Artery (LCA)–Circumflex Branch Right Coronary Artery (RCA)–Posterior Descending Branch


Inferior Wall Infarct
II
III
aVF
I
aVL
V5
V6
Lateral Wall Infarct
V1
V2
V3
V4
Anteroseptal Wall Infarct

What is next: Treatment

- Oxygen is NO LONGER recommended routinely
- Thought→may increase coronary vascular resistance
- 2018 Meta-analysis demonstrated no benefit (7700 pts)
- 2017 Swedish randomized study did not reduce one year mortality, some demonstrated potential harm
- Recommendation for Oxygen if O2 sat < 90%
- Deliver 2-4 L/nasal cannula

What is next: Treatment

- Morphine was used to reduce pain and anxiety as the standard treatment
  - Goal was to reduce myocyte metabolic demand and decrease tissue injury
  - Safety of morphine has never been established
- 2005 Crusade trial use of morphine was associated with higher mortality
- 2015 retrospective cardiac MRI study demonstrated larger sized infarcts in those who received morphine
- Today, evidence supports that morphine (opioids) interferes with/delays the absorption P2Y12 (ticagrelor and clopidogrel)
- Small trials showed decreased platelet inhibition when IV morphine was also given
- 2017 ACEP clinical policy recommends clinical judgement be used whether to give STEMI patient morphine
- Treatment modalities aimed at resolution of MI (ASA, Nitroglycerin) should be considered first

What is next: Treatment

- Antiplatelet
- Aspirin has the greatest benefit for the reduction of morbidity/mortality when administered early in STEMI
- Most effective dose is unknown, but loading dose, non-enter-coated chewed
- Takes effect within 60 minutes
  - (2013 ACCF/AHA ASA 162-325 mg orally before PCI)

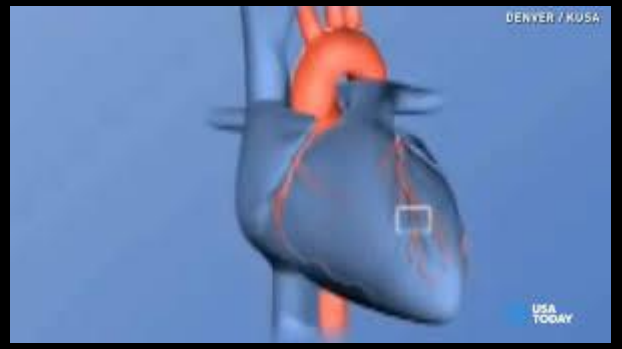
What is next: Treatment

- P2Y12 Inhibitors
- Clopidrel, ticagrelor, prasugrel
- Bind to inhibit platelet activation & aggregation
- Specific drug choice is based on patient specific factors (risk of bleeding)
- Increased risk of bleeding with Prasugrel; Ticagrelor
- Before administering should be discussed with cardiologist

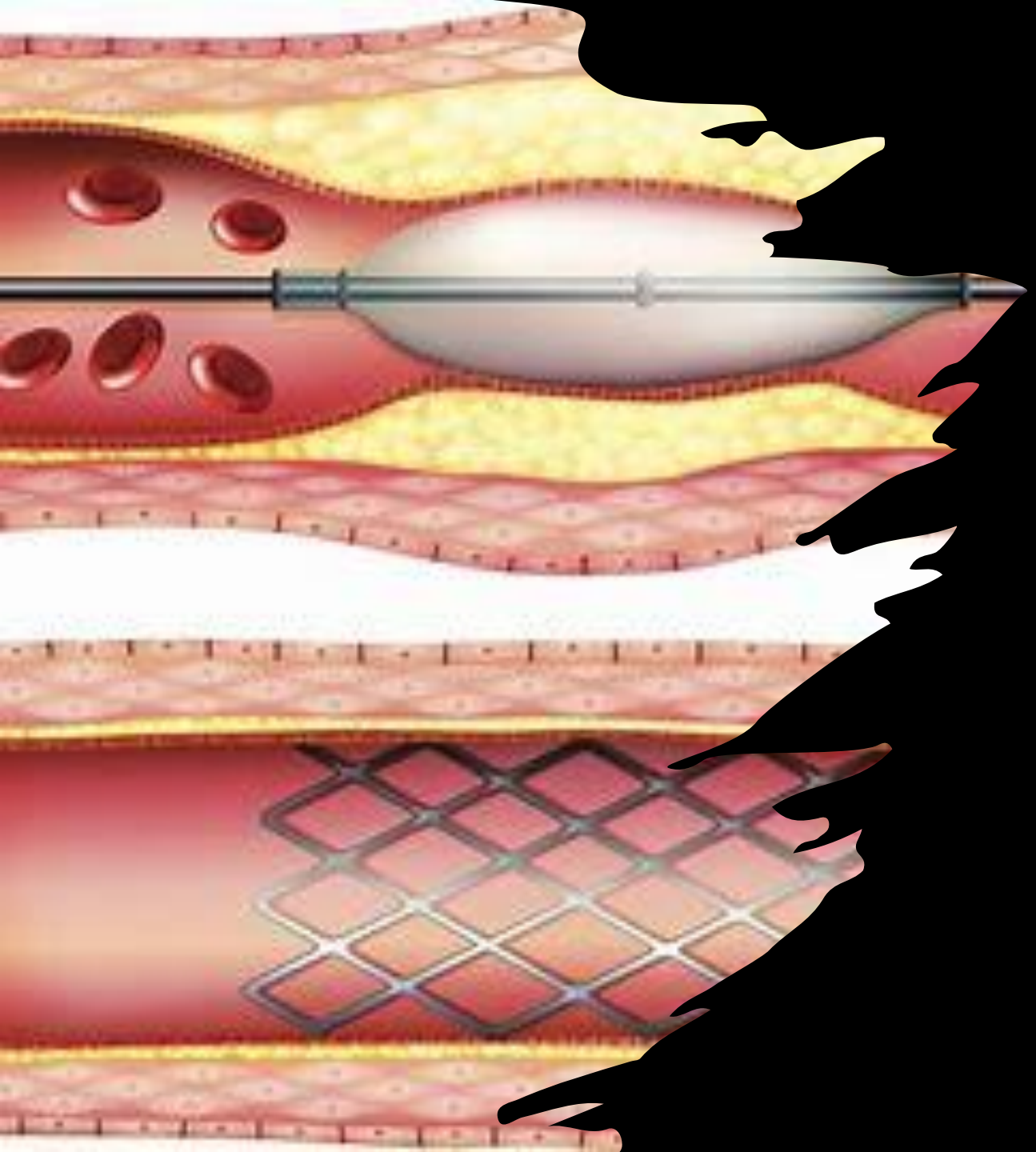
What is next: Treamtment

- Nitroglycerin
  - Reduces left ventricular preload & increases coronary blood flow
  - No evidence that its use reduces myocardial injury in STEMI
  - 2013 ACCF/AHA guidelines may be beneficial to MI with HTN and /heart failure
  - Absorption is variable and can be difficult to titrate effects

What is next : Treatment



- Beta Blockers
- Reduce myocardial oxygen demand→decreasing HR, inotropy, & blood pressure
- Recommended for all patients with STEMI within the first 24 hours
- Initial studies for B blockers were in the 1980's prior to routine use of reperfusion therapies
- Today, with rapid primary PCI (2 studies, AJC (40,873 pts & JACC, 91,895 pts) STEMI patients did not show mortality differences who did and did not receive beta blockers



What is next: Treatment

- PCI (Percutaneous Coronary Intervention)
- In STEMI recommended primary treatment with symptoms < 12 hours
- Between 12 – 24 hours, indicated if there is clinical or EKG evidence of ongoing ischemia
- Also recommended for patients with cardiogenic shock or severe acute heart failure (regardless of when STEMI occurred)
- PCI is superior to thrombolytics (higher rates of artery patency, lower rates of recurrent ischemia, reinfarction, intracranial hemorrhage, death
- Thrombolytics are considered in centers with no PCI
- Heparin is recommended for STEMI patients prior to PCI

What is next: Treatment

- Cocaine chest pain
- STEMI is managed the same with even stronger emphasis on PCI
- Cocaine causes alteration in platelet function and coagulation
- Benzodiazepines can be used initially to relieve chest pain and beta blockers are not recommended in the acute setting of cocaine use

Case 1

- 68-year-old with chest pain and ST depression in V2 and V3. Repeat EKG 15 minutes later demonstrated ST elevation in V3, V4 and V5.
- Although pain may have improved or stable VS, the patient must be transferred ASAP to a chest pain center or closest hospital by EMS. They will inform the hospital to activate the cath lab (team) PTA.

Case 2

- A 56-year-old female presents and complains of constant pain in the left upper chest for the past two days. Denies dyspnea, diaphoresis or radiation of the pain to her arm.
- An EKG is completed, and it demonstrates ST segment elevation in inferior and lateral leads.
- Load and go after aspirin. If there is a delay in transport, start on IV and reassure the patient. Call the hospital ED and communicate clearly about the concerns of STEMI

Take Home Pearls

- Recognize importance a obtaining an EKG with all patient who present with chest pain
- When a suspicion for STEMI is present, consider repeat the EKG if unsure, if consistent with concern transfer early with the EKG
- If concerned administer one aspirin
- Consider IV, nitro and continuous monitoring
- Call the closest ED and give a hands off report
- Return a call to the ED to ask what happened and disposition


Questions?
efcout57@aol.com