Initial Evaluation of Chest Pain and the Updated Management of ACS

David Prutzman, DO, FACC, FSCAI

Structural Cardiologist

Biltmore Cardiology and Abrazo Health Network

Disclosures

- None

Outline

- Initial evaluation of common causes of chest pain
- Rapid diagnosis and intervention of high risk cardiac patients
- Understanding treatment of ACS patients and relevant guideline updates

- Identifying an ischemic electrocardiogram

“Patient’s don’t read the book.”

Background

- 1 person dies every 33 seconds from cardiovascular disease
- In 2022, heart disease accounted for approximately 702K deaths in the US, or 1 in every 5, and more common than all major causes of cancer combined!
- Coronary artery disease is the leading cause of death worldwide estimated at 9 million deaths per year
- Economic burden of heart disease in the US is estimated around 240 BILLION ANNUALLY
  - If you earned that much money, you would be the 3rd richest person in the world!

Cardiac

- ACS
- Pericarditis
- Aortic Dissection
- Valvular Heart Disease
- Heart Failure
- Arrhythmia

Non-cardiac

- Pulmonary Embolism
- GERD
- Esophageal Spasms
- Costochondritis
- Panic Attacks
- Pleurisy

What is typical chest pain?

Table 1. Clinical Classification of Chest Pain[4]	
Typical angina (definite)	(1) Substernal chest discomfort with a characteristic quality and duration that is (2) provoked by exertion or emotional stress and (3) relieved by rest or nitroglycerin.
Atypical angina (probable)	Meets 2 of the above characteristics.
Noncardiac chest pain	Meets 1 or none of the typical angina characteristics.

Important Considerations

- Age
- Sex
- Risk factors
- Family History
- Timing
- Exacerbating factors
- Recent changes, progression

Initial Examination

- ***History and physical ***
  - Timing, character, frequency, location
  - Cold vs. warm extremities
  - Vitals
  - Cardiac exam (murmur, rub, gallop, etc)
- Electrocardiogram
- Chest X Ray
- CT imaging
- Cardiac enzymes, CBC, CMP, Inflammatory Markers

ECG

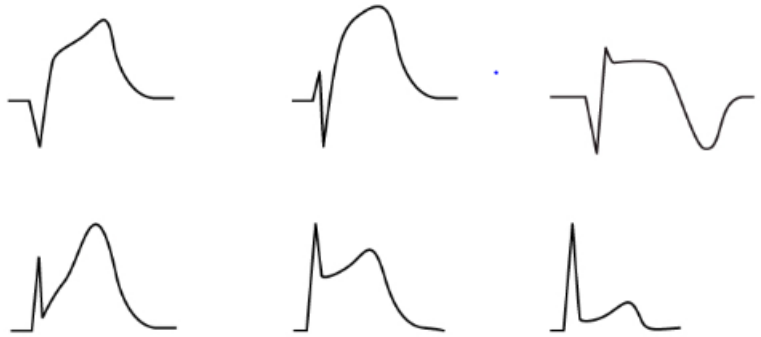
STEMI

STEMI: ST-segment elevations ≥1 mm (0.1 mV) in 2 anatomically contiguous leads or ≥2 mm (0.2 mV) in leads V2 and V3 **or** new left bundle branch block and presentation consistent with ACS. If ECG suspicious but not diagnostic, consult cardiologist early.

A Characteristics of ST-segment elevations caused by ischemia

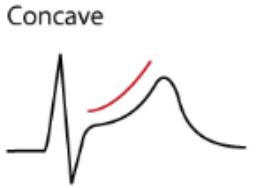
ST-segment elevations caused by ischemia typically displays a convex or straight ST-segment. Such ST-segment elevations in presence of chest discomfort are strongly suggestive of transmural myocardial ischemia. Note that the straight downsloping variant is unusual.

C Examples of ST-segment elevations caused by ischemia



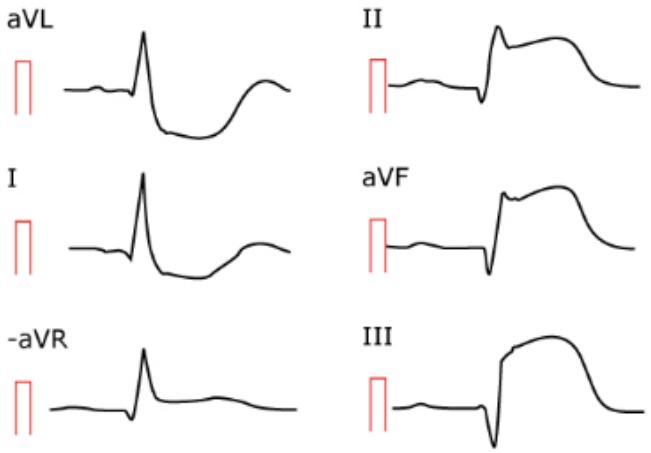
ST-segment elevation can vary markedly in appearance. These six examples were retrieved from six different patients with STEMI.

B Typical non-ischemic ST-segment elevation



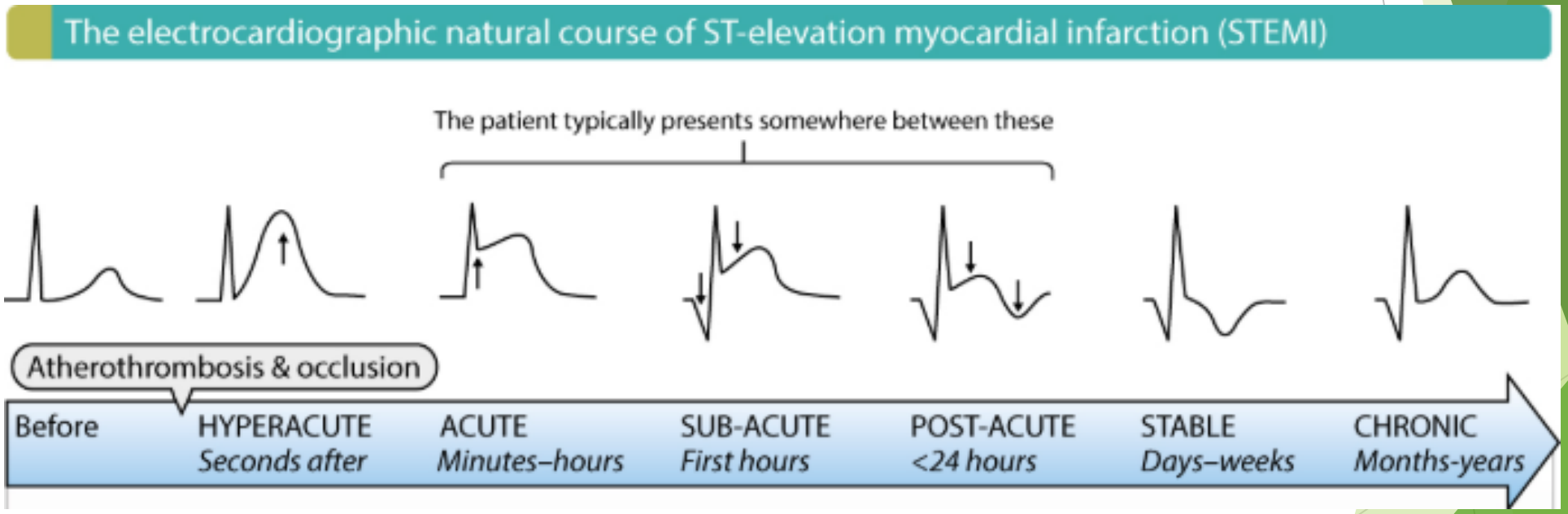
Non-ischemic ST-segment elevations are extremely common in all populations. They are characterized by a concave ST-segment and a greater distance between the J point and the T wave apex.

D Real life example (limb leads shown)



ECG from a male patient (age 61) who experienced chest pain while driving to work. Note ST-segment elevations as well as reciprocal ST-segment depressions. There are also pathological Q-waves (leads III, aVF and perhaps II).

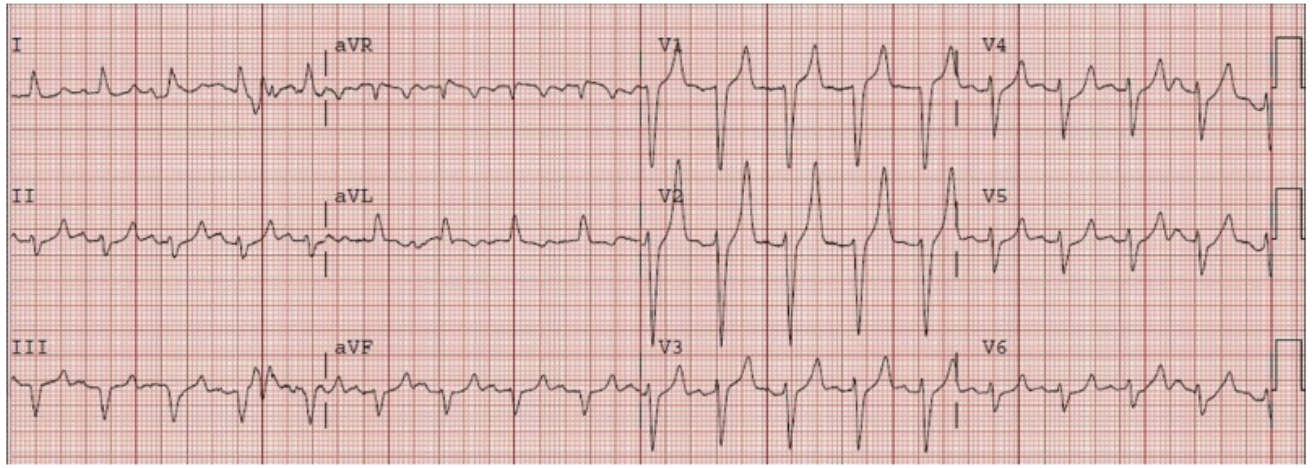
ECG evolution in ACS



Peaked Hyperacute T wave

QRS Widening

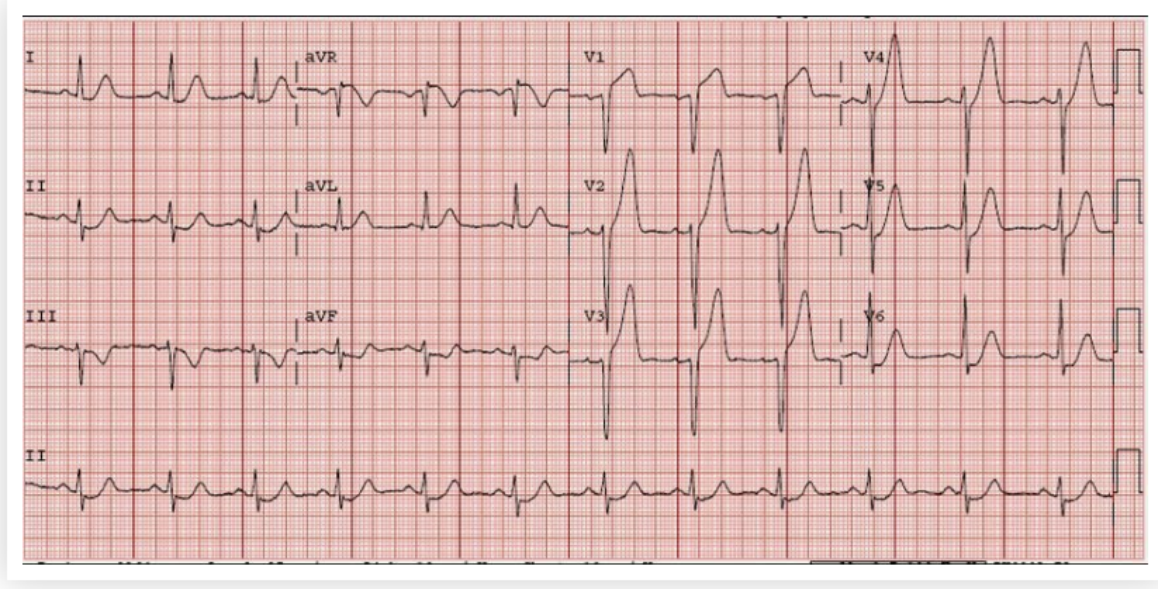
No reciprocal changes



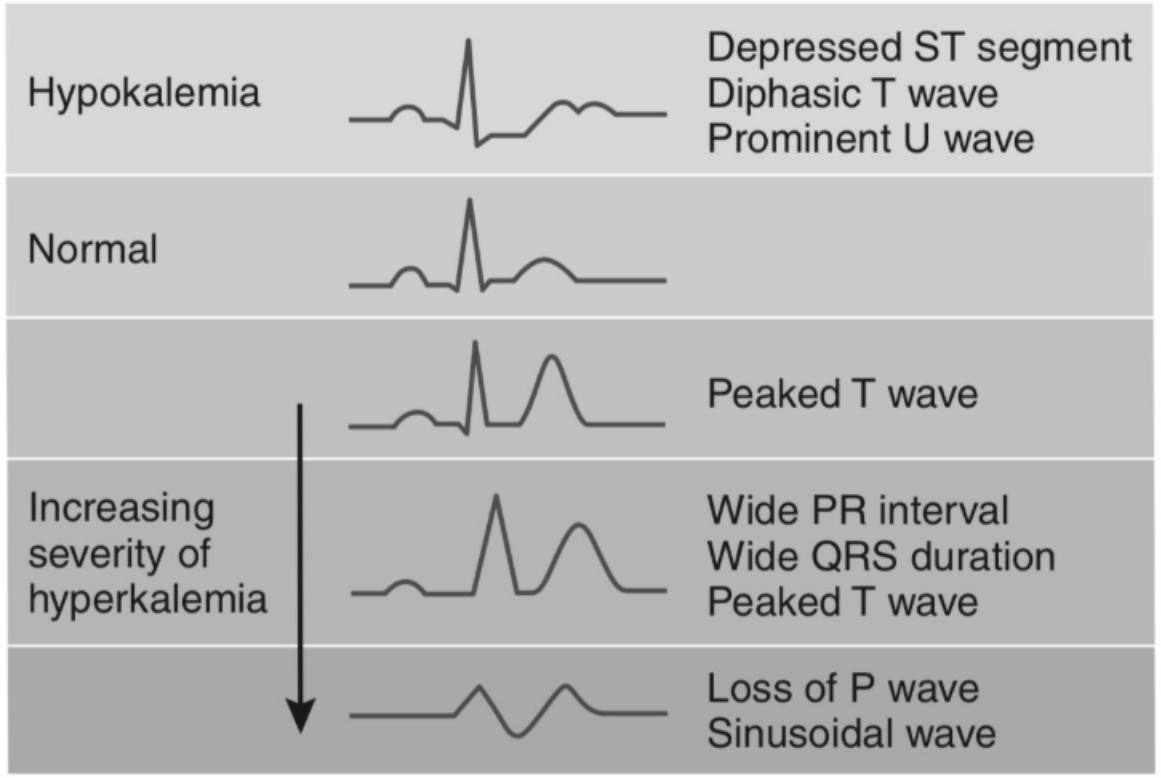
Blunted Hyperacute T wave

Elevation

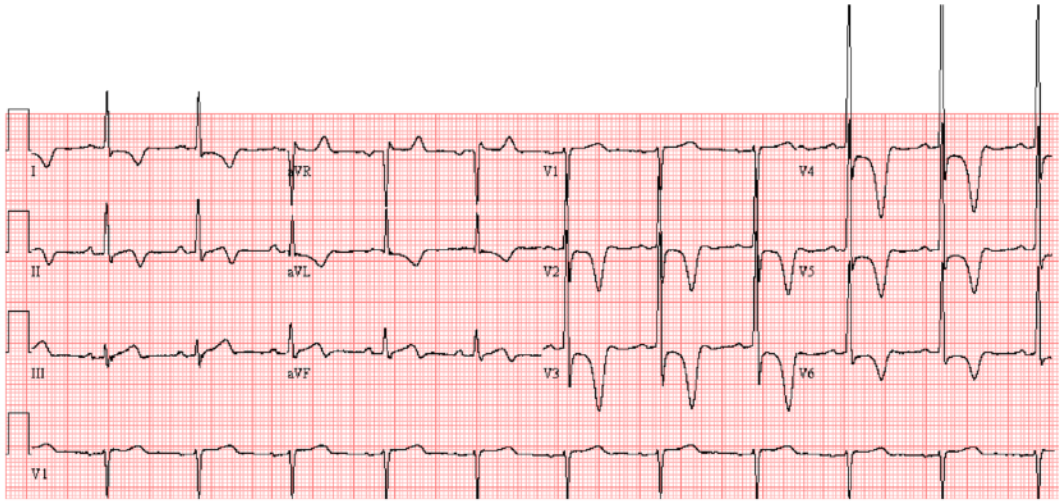
Reciprocal changes



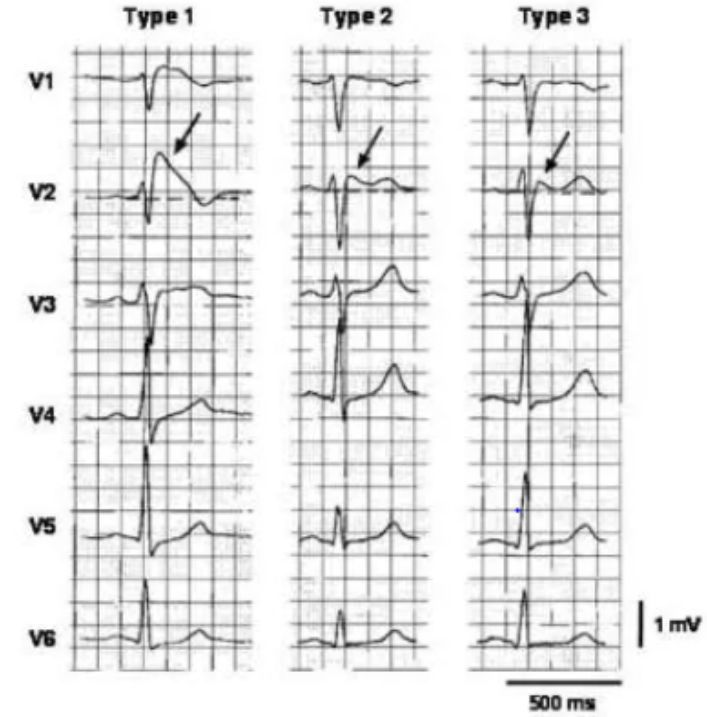
ECG Evolution in Hyperkalemia

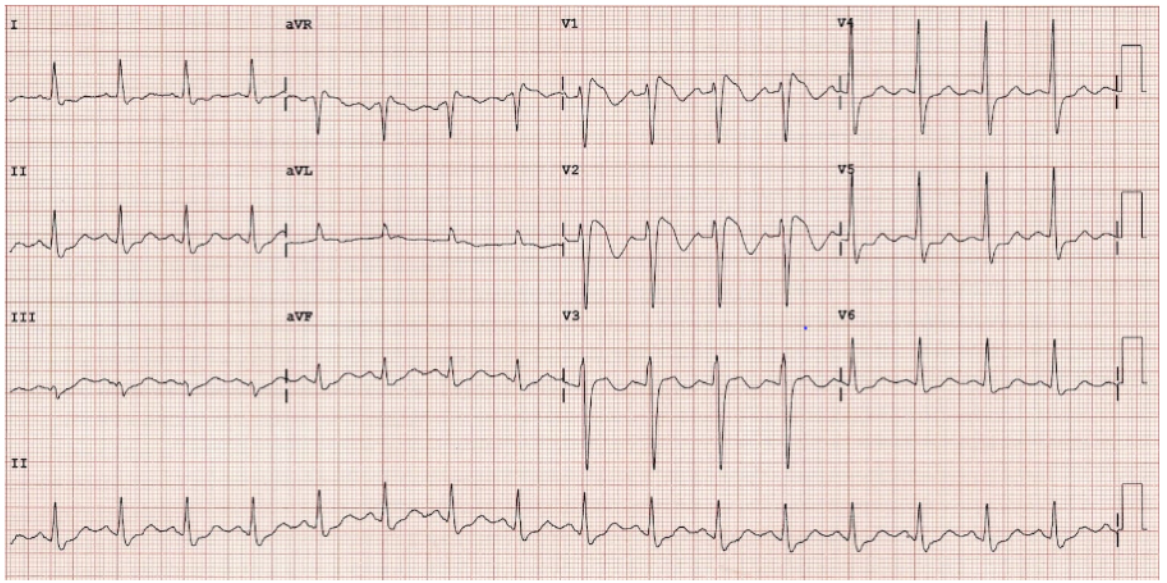


LVH with HOCM



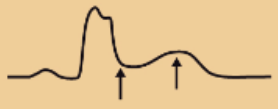
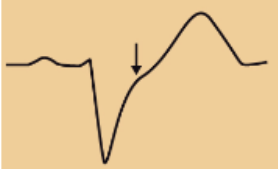
BRUGADA





Sgarbossa Criteria

A Original Sgarbossa criteria for diagnosis of STEMI in the setting of LBBB

CRITERIA	POINTS		Normal LBBB	Ischemic LBBB
ST segment elevation ≥1 mm in any lead with positive QRS (V4, V5, V6, aVL, I) .	5	V4-V6, aVL, I		
ST segment depression ≥1 mm in V1, V2 and/or V3.	3	V1-V3		
ST segment elevation ≥5 mm in any lead with discordant QRS (V1, V2, V3)	2	V1-V3		
			These complexes show the normal (expected) appearance of LBBB.	These complexes show ischemic manifestations in the setting of LBBB.

B Modified Sgarbossa Criteria

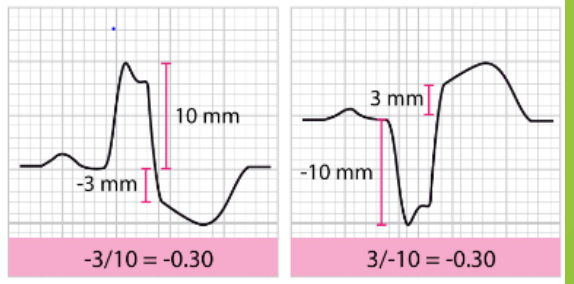
CRITERIA

1. Unchanged

2. Unchanged

3. Abnormal, excessive discordance

≥1 lead with ≥1 mm ST elevation and proportionally excessive discordant ST elevation, as defined by STE ≥ 25% of the depth of the preceding S-wave (an ST / S ratio of ≤ -0.25)



Greater than 3 points specificity of 90-98% for AMI

Musculoskeletal Disorders

- Chest Wall Pain
- Costochondritis
- Trauma

- MC cause of chest pain outpatient
- 33-50%
- Localized muscle tension, stinging pain, reproducible
- Usually self limited
- NSAID/ analgesia therapy
- Does not typical require testing in absence of cardiopulmonary symptoms or risk factors

Pulmonary Disorders

- Infectious/Inflammatory
- COPD
- Asthma

- Clinical syndrome of fever, cough, tachypnea
- Pleuritic chest pain
- Tachypnea
- Physical exam findings
  - Expiratory wheezing
  - Rales
  - Dullness or decreased auscultation
- CXR or CT chest
- Treatment revolving around diagnosis, usually combination of steroids, nebulizers, antibiotics

Gastrointestinal Disorders

- GERD
- Achalasia
- Dysmotility
- Ulceration/AVM
- Gallbladder disease

- Burning retrosternal pain
- Sour/bitter taste in mouth
- Can be worse at night/ lying flat
- Association with food

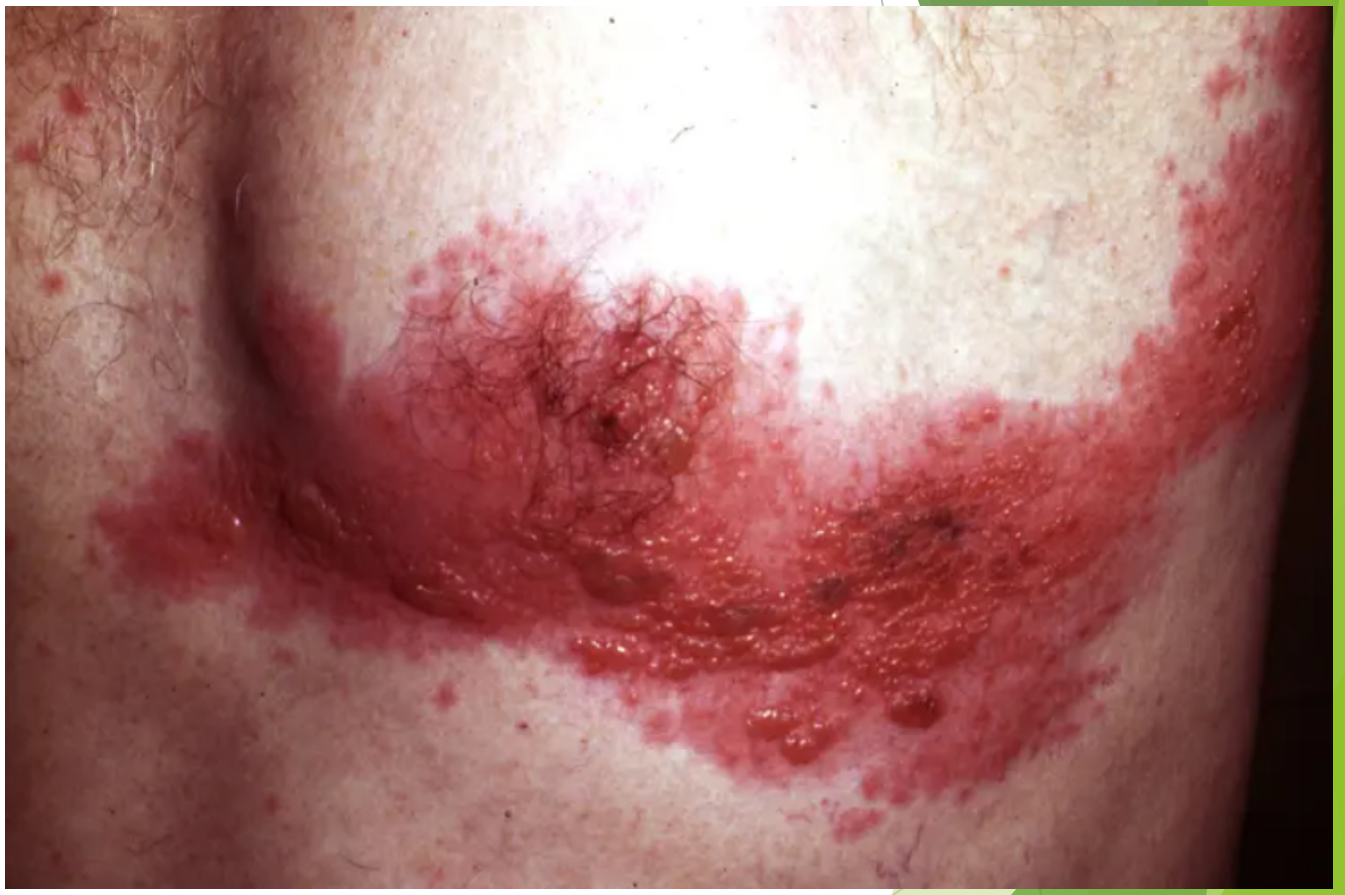
- ECG/cardiac enzymes to evaluate for ischemia
- CBC, stool samples

Dermatologic Disorders

- Shingles
- Psoriasis
- Scleroderma

- Importance of Physical Exam

- Association with Heart Disease



Psychiatric Disorders

- Panic Disorders
- Anxiety
- PTSD

- 1 in 4 people with panic attack with have chest pain or dyspnea
- Often a diagnosis of exclusion
- Consider drug induced chest pain

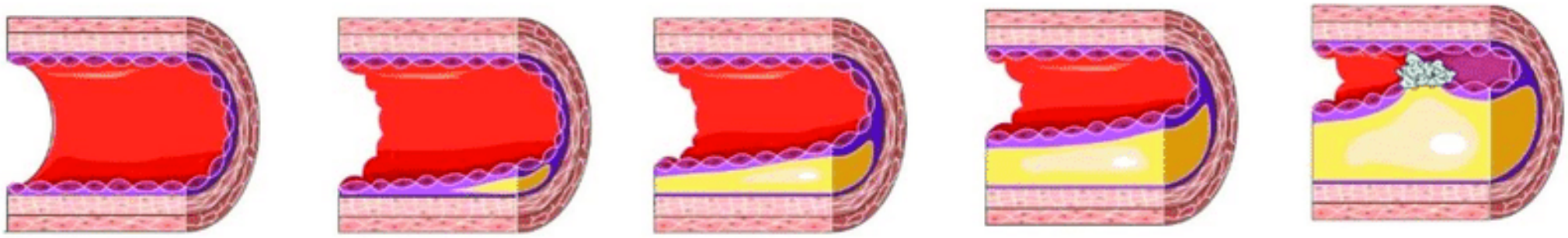
Cardiac

- Coronary artery disease
- Valvular Heart Disease
- Heart Failure
- Arrhythmia disorders; tachycardia or bradycardia
- Aortic pathology
- Inflammatory/ Infectious
  - Pericarditis

- History and physical exam
  - Murmur, Edema, Orthopnea, Wide or narrow Pulse pressure, Cool extremities, Altered mental status, BP difference, etc..

Cardiovascular disease

The Evolution of CAD over Time



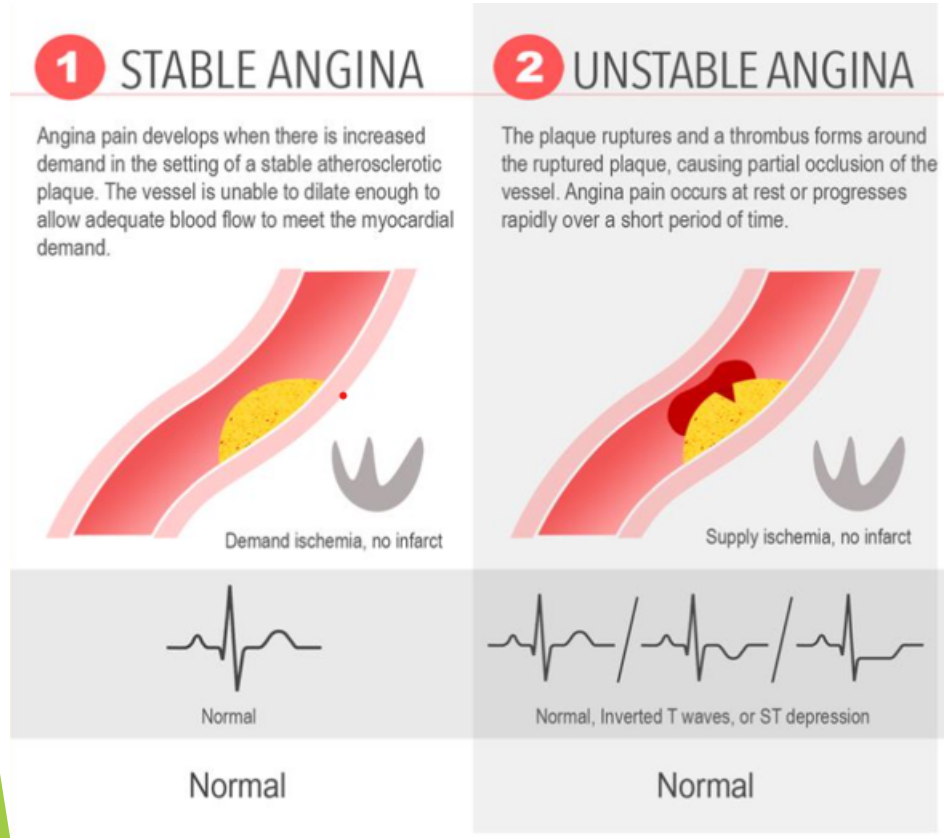
Smoking
High LDL
Low HDL
Hypertension
Genetics
Diabetes
Obesity

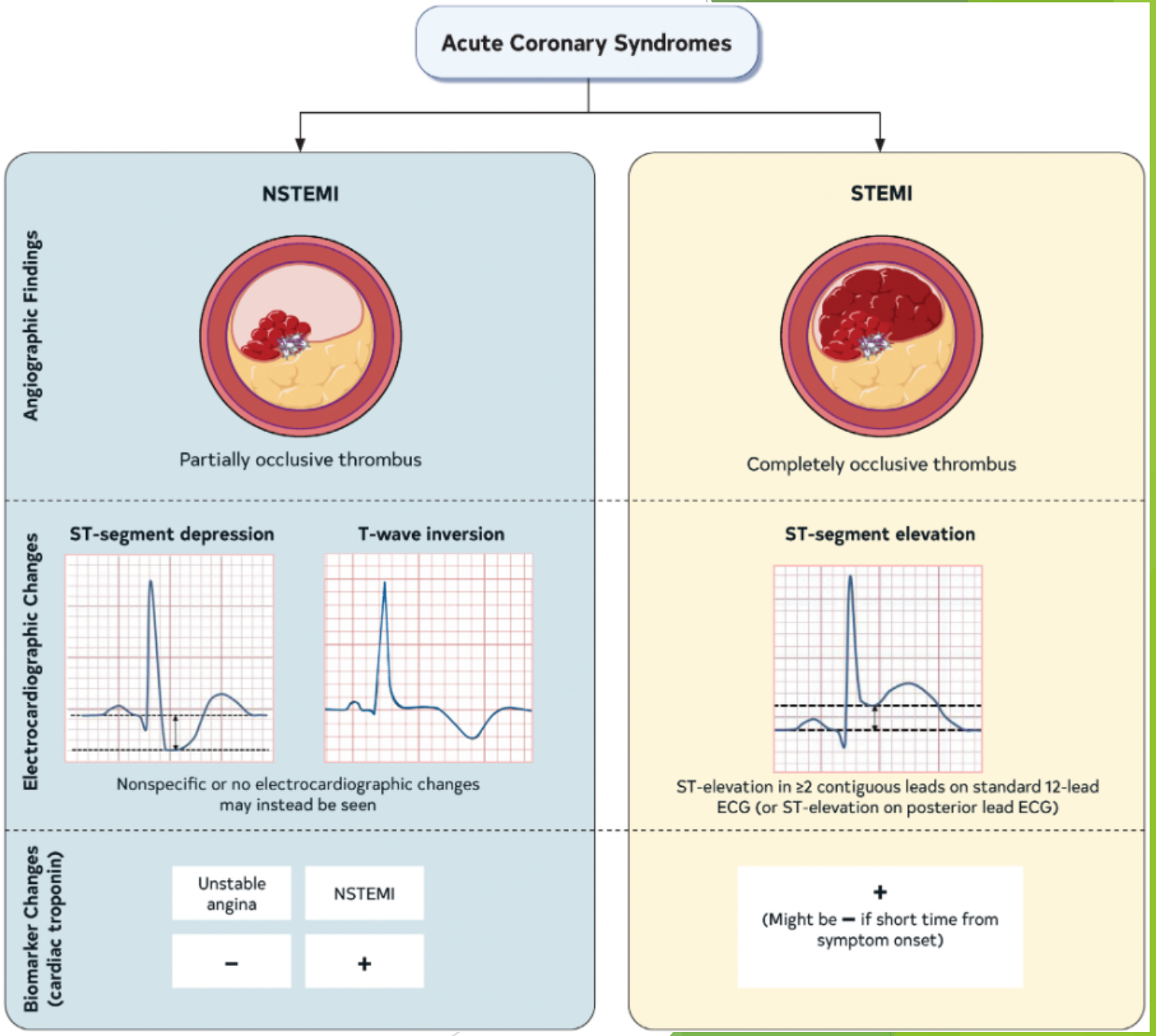
Atherosclerotic plaques
Endothelial dysfunction, SMC proliferation
Inflammation, Foam cell formation
Fibroblast proliferation, Collagen formation

PLAQUE RUPTURE

Clinical event:
- Myocardial infarction
- Unstable angina
- Death

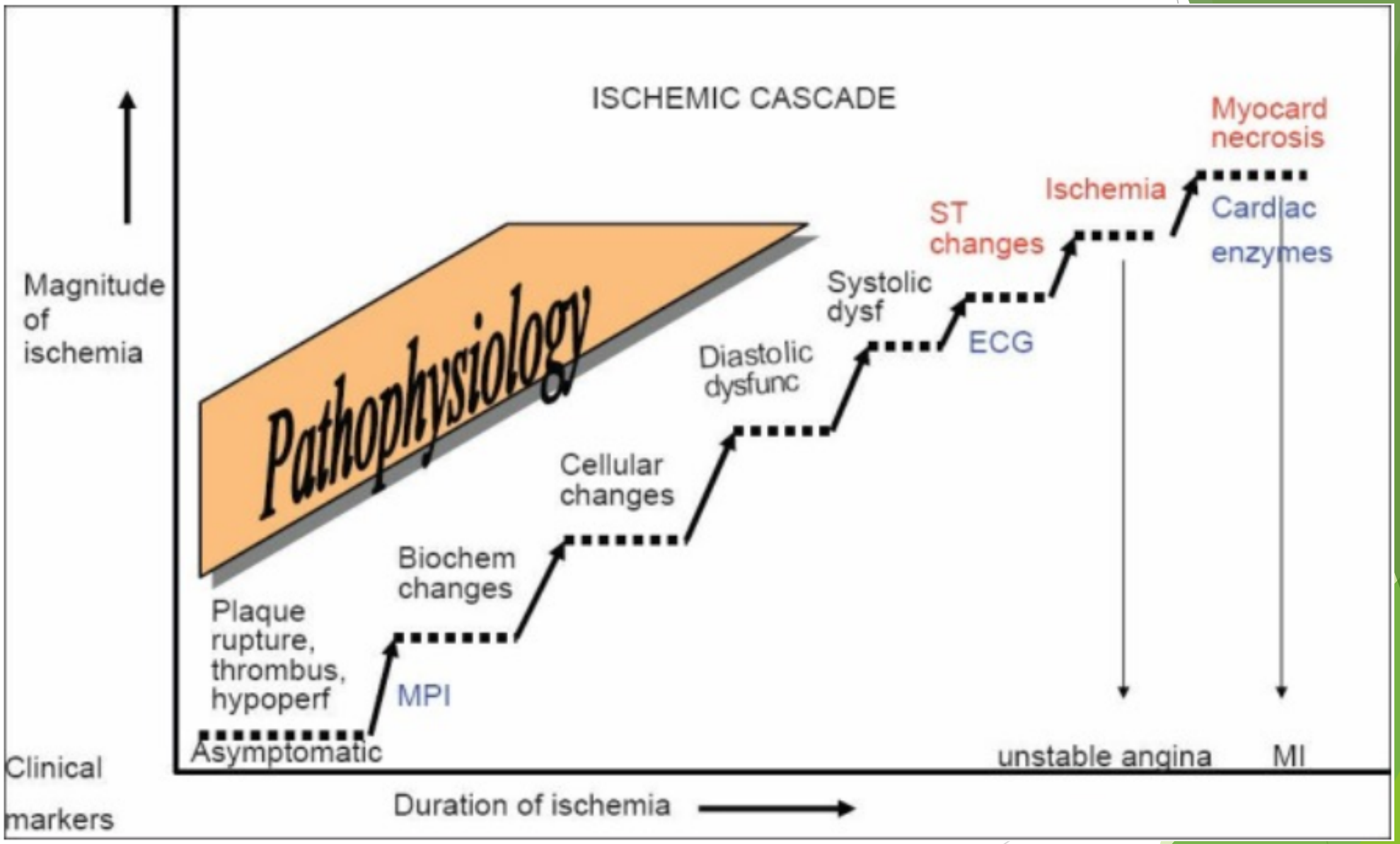
Acute Coronary Syndrome (ACS)





Types of MI by Universal Definition

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.


ISCHEMIC CASCADE
Myocard necrosis
Ischemia
Cardiac enzymes
ST changes
ECG
Systolic dysf
Diastolic dysfunc
Cellular changes
Biochem changes
MPI
Plaque rupture, thrombus, hypoperf
Asymptomatic
Pathophysiology
Magnitude of ischemia
Clinical markers
unstable angina
MI
Duration of ischemia

Treatment:
What do the new Guidelines Say?

2025 ACC/AHA/ACEP/NAEMSP/SCAI Guideline

- DAPT is recommended for patients with ACS, Ticagrelor or Prasugrel > clopidogrel
  - In patient's with NSTEMI with angiogram >24 hours, upstream treatment may be reasonable to reduce MACE
- Recommended for 12 months, but some exceptions
- High- intensity statin for all ACS +/- ezetimibe with goal <70 mg/dL
- Radial > femoral to reduce bleeding, vascular complications, and death.

Highlights Continued

- Complete revascularization recommended in patients with STEMI, pending lesion complexity, and culprit-only in SHOCK patients
- Impella (mechanical support) is reasonable to reduce death in shock related to AMI
- RBC transfusion to maintain hemoglobin of 10g/dL may be reasonable
- After discharge, focus on secondary prevention is fundamental.
  - Lipid panel recommended 4-8 weeks
  - Cardiac rehab

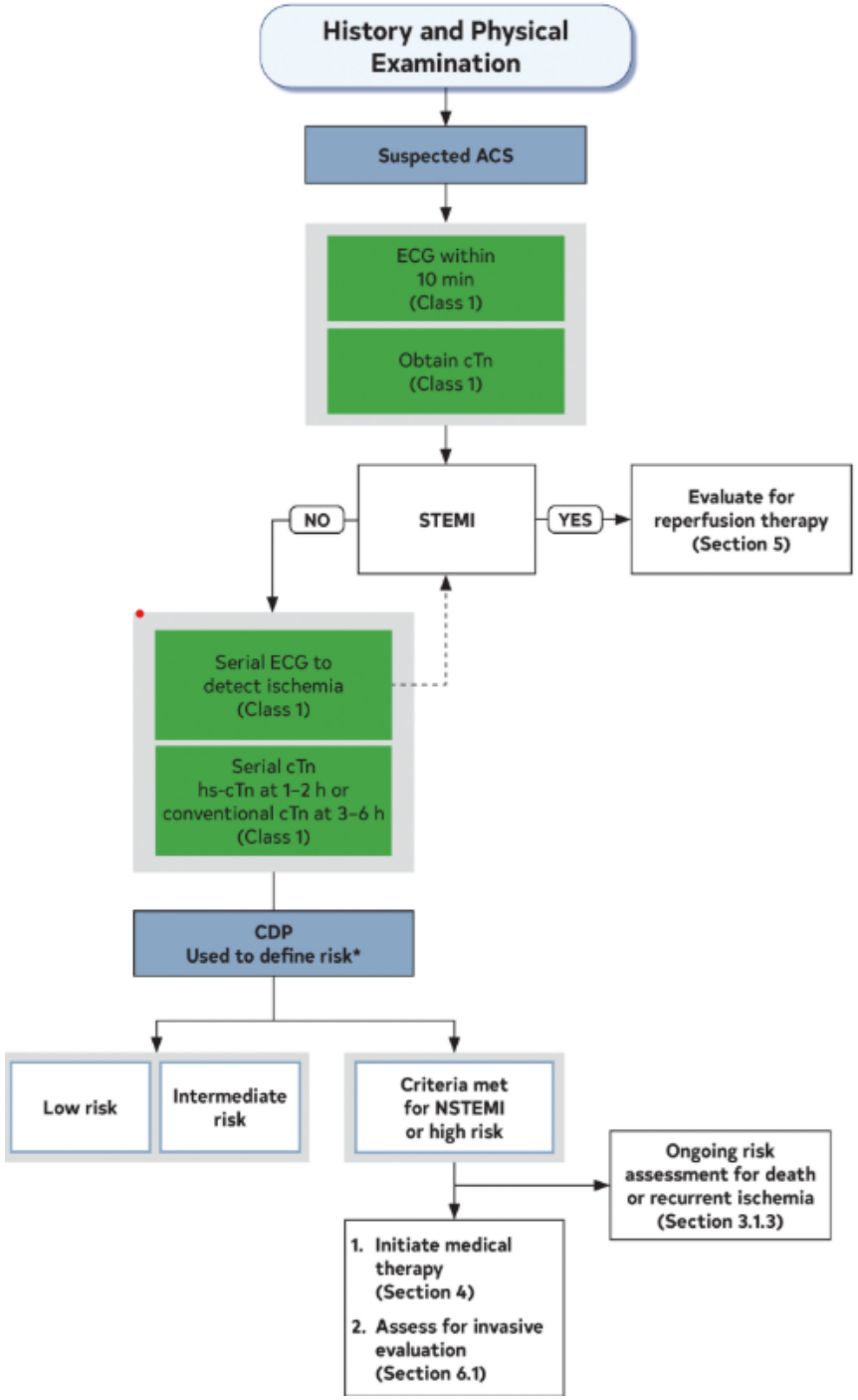
Pre- Hospital Assessment

- A second or third ECG may identify up to an addition 15% of STEMI's
- NCDR
  - 11% had nondiagnostic initial ECG
  - 72.4% had follow up diagnostic within 90 minutes

COR	LOE	Recommendations
Suspected ACS		
1	B-NR	1. In patients with suspected ACS, a 12-lead ECG should be acquired and interpreted within 10 minutes of first medical contact (FMC)* to identify patients with STEMI.[1,2]
1	C-LD	2. In patients with suspected ACS in which the initial ECG is nondiagnostic of STEMI, serial ECGs to detect potential ischemic changes should be performed, especially when clinical suspicion of ACS is high, symptoms are persistent, or the clinical condition deteriorates.†[3,4]
STEMI		
1	B-NR	3. In patients with suspected STEMI, immediate emergency medical services (EMS) transport to a PCI-capable hospital for primary PCI (PPCI‡) is the recommended triage strategy, with an FMC–to-first-device time system goal of ≤90 minutes.[5–7]
1	B-NR	4. In patients with suspected STEMI, early advance notification of the receiving PCI-capable hospital by EMS personnel and activation of the cardiac catheterization team is recommended to reduce time to reperfusion.[1,8,9]

In-Hospital Assessment

COR	LOE	Recommendations
1	B-NR	1. In patients with suspected ACS, acquisition and interpretation of an ECG within 10 minutes is recommended to help guide patient management.*[1,2]
1	B-NR	2. In patients with suspected ACS in whom the initial ECG is nondiagnostic, serial 12-lead ECGs should be performed to detect potential ischemic changes, especially when clinical suspicion of ACS is high, symptoms are persistent, or clinical condition deteriorates.*[3]
1	B-NR	3. In patients with suspected ACS, cTn should be measured as soon as possible, preferably using a high-sensitivity cTn (hs-cTn) assay.*[4–7]
1	B-NR	4. In patients with suspected ACS with an initial hs-cTn or cTn that is nondiagnostic, the recommended time intervals for repeat measurements after the initial sample collection (time zero) are 1 to 2 hours for hs-cTn and 3 to 6 hours for conventional cTn assays.*[8–12]



TIME = MUSCLE

Reperfusion of the infarct related artery is associated with improved survival. Every 30 minutes of delay is associated with relative risk of 1 year mortality by 7.5%!

Important Considerations

- Focused history and rapid evaluation
- Risk stratification Tools with STEMI and NSTE-ACS
  - GRACE, TIMI
- Evidence of heart failure, multi-organ dysfunction and cardiogenic shock
- Importance of stabilization, especially in Cardiac Arrest

- Poor Risk Factors
  - pH levels (<7 even <7.2)
  - Downtime
  - CPR duration (15 min?)
  - Age
  - Male Sex
  - Co-morbidities (renal)
  - Non-shockable rhythm

Recommendations for Management of Patients Presenting With Cardiac Arrest
Referenced studies that support recommendations are summarized in the Evidence Table.

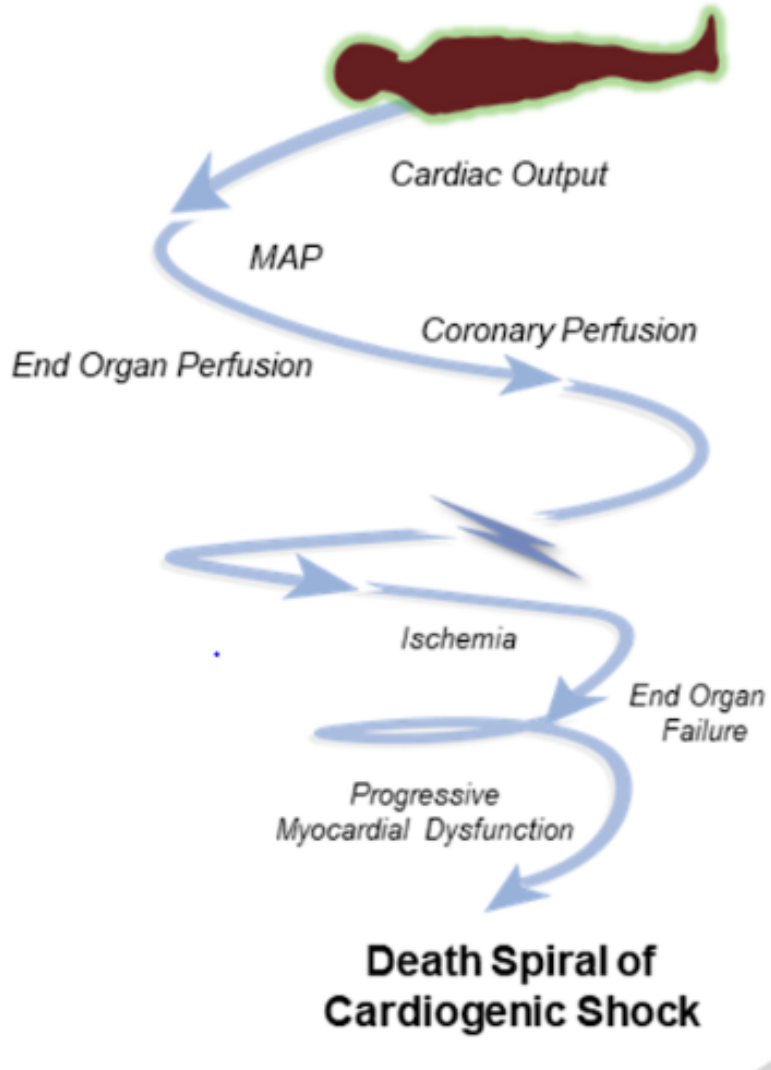
COR	LOE	Recommendations
1	C-LD	1. Patients with cardiac arrest and STEMI who have been resuscitated should preferentially be transferred by EMS to a PPCI-capable center.[1,2]
1	B-NR	2. Patients who have been resuscitated after cardiac arrest and are noncomatose or who are comatose with favorable prognostic features and with evidence of STEMI, should undergo PPCI to improve survival.[3–7]
2b	C-LD	3. In patients with cardiac arrest who are comatose, have unfavorable prognostic features, and evidence of STEMI, PPCI may be reasonable after individualized assessment.[4,7]
3: No Benefit	A	4. In resuscitated patients who are comatose after cardiac arrest, electrically and hemodynamically stable, and without evidence of STEMI, immediate angiography is not recommended due to lack of benefit.[8–14]

Cardiogenic Shock

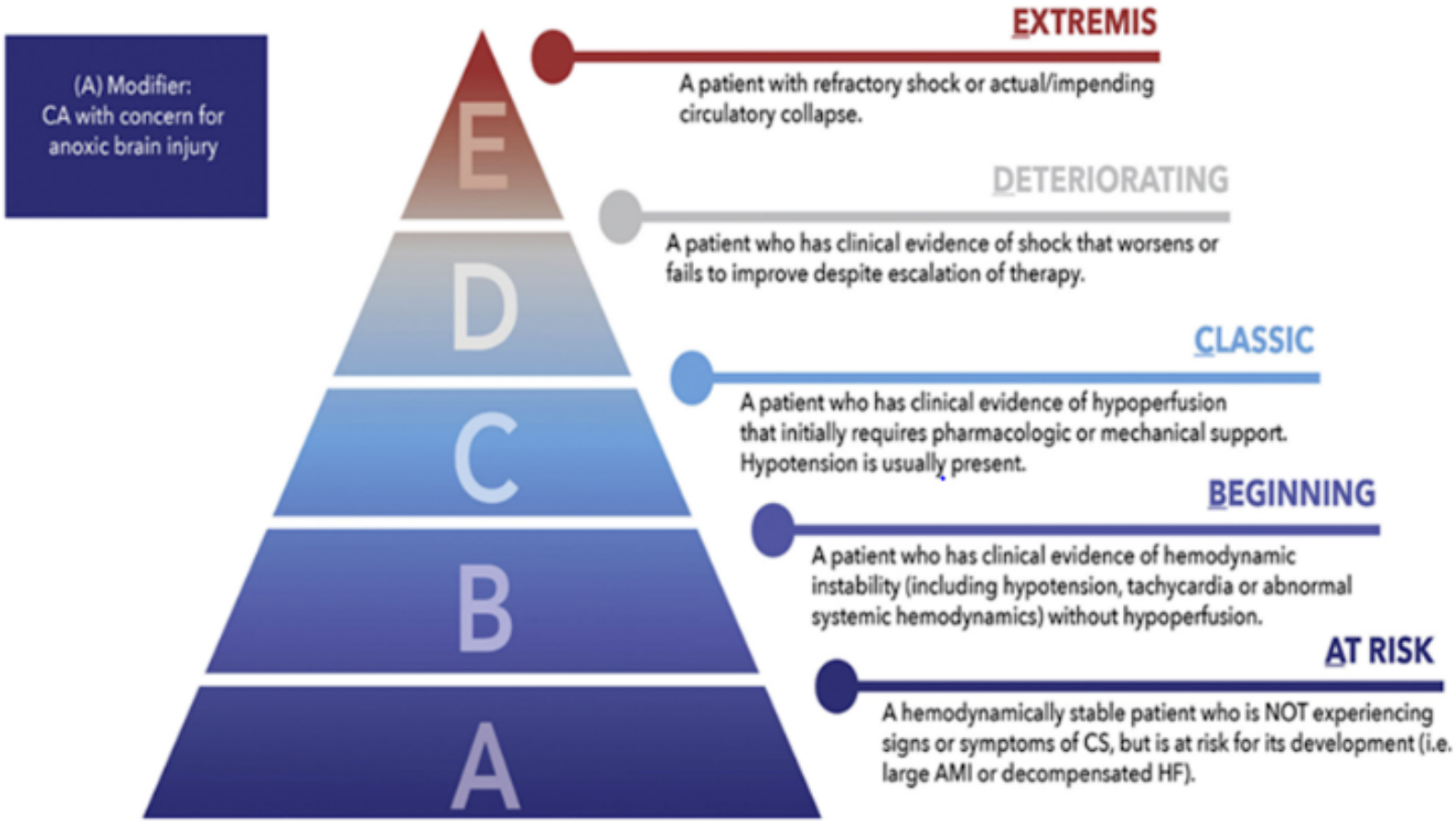
- Inadequate cardiac output to maintain tissue perfusion and body function, due to pump failure
- Most common cause is Acute Myocardial Infarction
- Overall mortality dependent on stage of shock, but overall about 40%
- In hospital mortality has declined due to early revascularization and overall management

CARDIOGENIC SHOCK

- Inadequate tissue perfusion[1]
- Result of cardiac dysfunction[1]
- Leading cause of death in AMI patient[1]
- Wide spectrum of presentation[2]
- No agreed consensus on the criteria[2]
- Almost always fatal if cycle of damage is not interrupted[2]
- Incidence is growing[3]

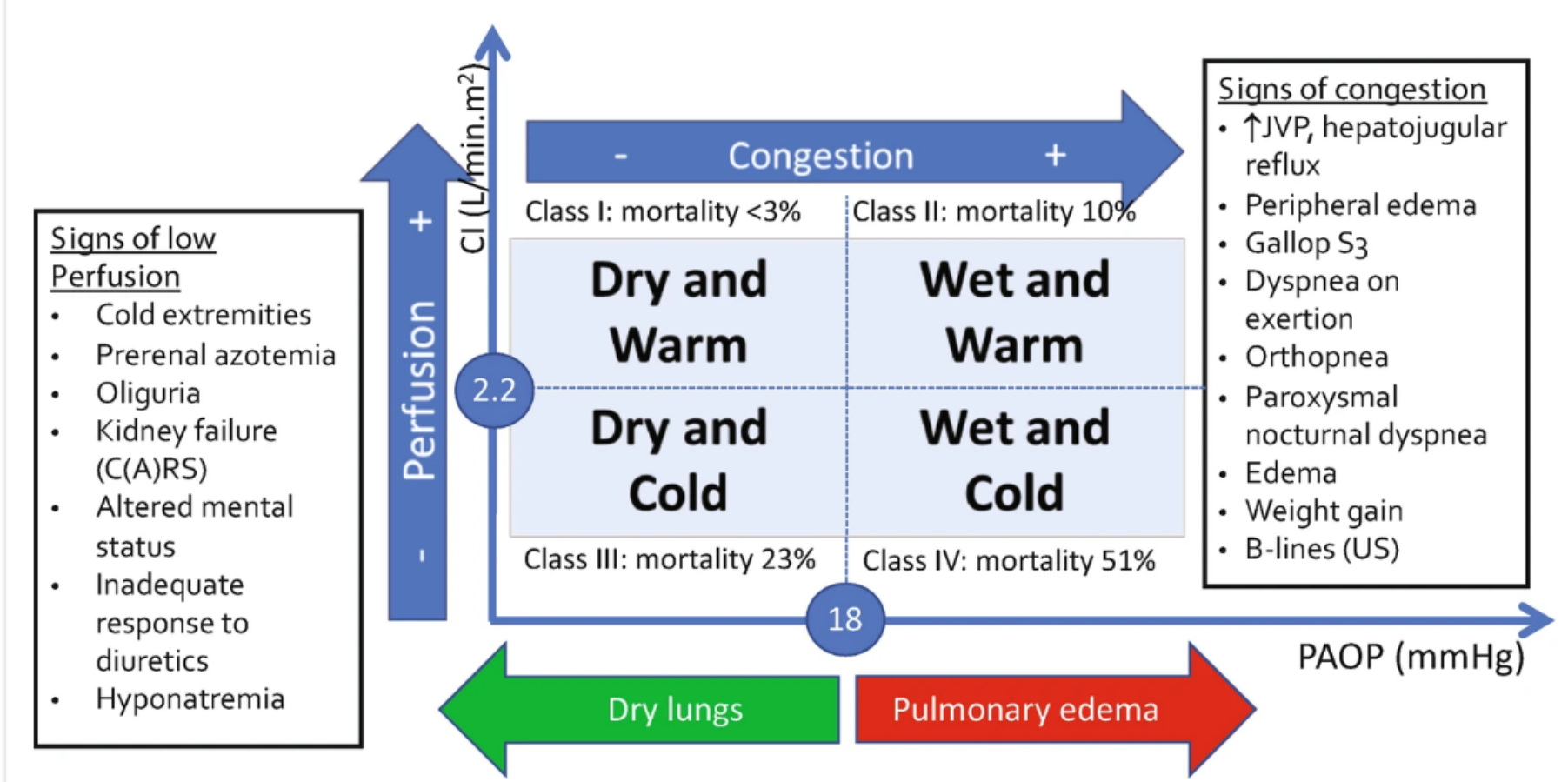


SCAI SHOCK CLASSIFICATION PYRAMID



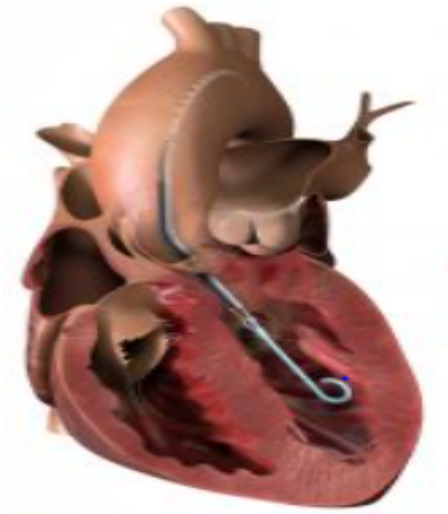
Naidu, S. S., Baran, D. A., Jentzer, J. C., Hollenberg, S. M., van Diepen, S., Basir, M. B., Grines, C. L., Dieroks, D. B., Hall, S., Kapur, N. K., Kent, W., Rao, S. V., Samsky, M. D., Thiele, H., Truesdell, A. G., & Henry, T. D. (2022). SCAI Shock Stage Classification Expert Consensus Update: A review and incorporation of Validation Studies. *Journal of the Society for Cardiovascular Angiography & Interventions, 1*(1), 100008. https://doi.org/10.1016/j.jscai.2021.100008

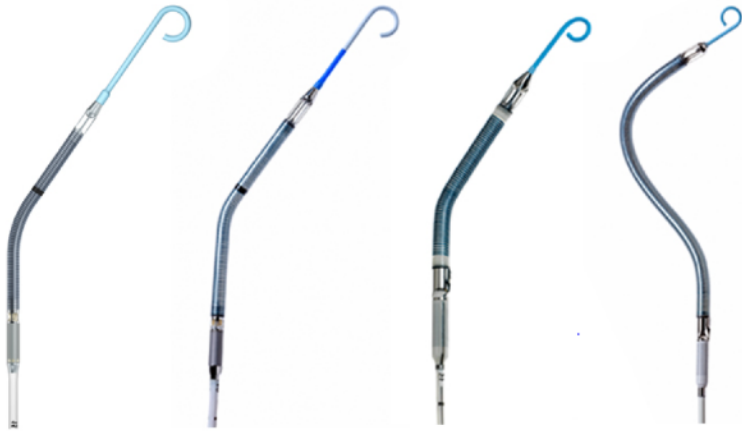
Heart Failure and Cardiogenic Shock

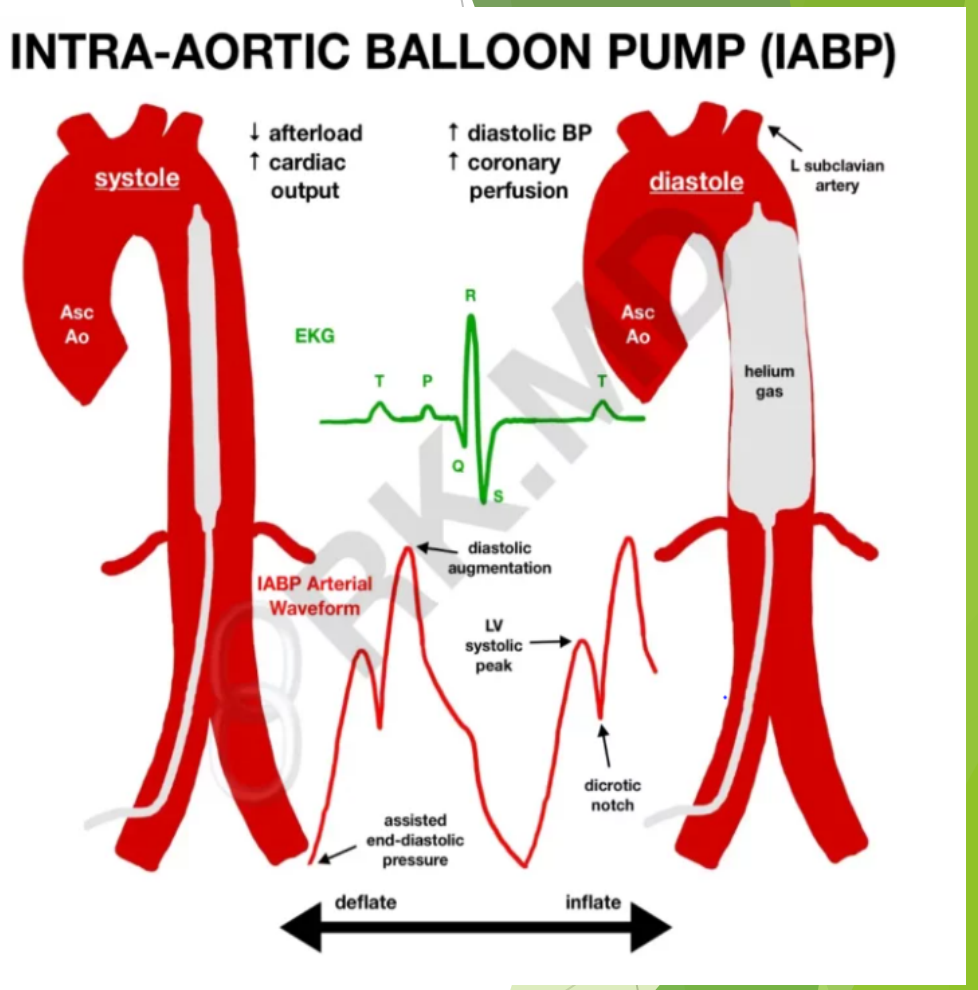


Impella


BLOOD INLET AREA
Catheter Diameter: 9 FR
Flow Rate: Up to 2.5 L/min
BLOOD OUTLET AREA
12 FR PUMP METER




IMPELLA 2.5
IMPELLA CP
IMPELLA 5.0
IMPELLA


INTRA-AORTIC BALLOON PUMP (IABP)
systole
↓ afterload
↑ cardiac output
↑ diastolic BP
↑ coronary perfusion
diastole
L subclavian artery
Asc Ao
Asc Ao
EKG
R
T
P
T
Q
S
helium gas
diastolic augmentation
IABP Arterial Waveform
LV systolic peak
dicrotic notch
assisted end-diastolic pressure
deflate
inflate

ACS: Initial Treatment

- MONA?
  - Morphine: 2-4mg, may repeat Q5-15min up to 10mg
    - May delay absorption of antiplatelets
  - Oxygen
    - Keep >90%
  - Nitrates
    - Hemodynamically stable patients SBP >90mmHg
    - For infusion, start 10ug/min and titrate for pain relief
  - Aspirin
    - All patient's with ACS without absolute contraindication (162-325mg)
    - Low dose aspirin long term
  - Beta Blockers
    - In ACS without contraindications, oral therapy recommended to reduce risk of re-infarction and ventricular arrhythmia

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

Fibrinolytic Therapy

- Reperfusion (PCI vs. fibrinolysis)
  - PCI unavailable within 120minutes of first medical contact, symptoms <12 hours, no contraindications

Recommendations for Reperfusion at Non–PCI-Capable Hospitals
Referenced studies that support recommendations are summarized in the Evidence Table.

COR	LOE	Recommendations
1	A	1. In patients with STEMI and an estimated time from FMC to device activation of ≤120 minutes or those with a contraindication to fibrinolytic therapy, transfer to a PCI-capable hospital for PPCI is recommended to reduce MACE.[1–3]
1	A	2. In patients with STEMI and symptom onset of <12 hours and anticipated delay to PPCI >120 minutes from FMC, fibrinolytic therapy should be administered in patients without contraindication to reduce MACE.[4–10]
2a	B-NR	3. In patients with STEMI and symptom onset of 12 to 24 hours, transfer to a PCI-capable hospital for PPCI is reasonable to reduce infarct size and MACE.[11,12]
3: Harm	B-R	4. In patients with only ST-segment depression, except when true posterior STEMI is suspected, fibrinolytic therapy should not be administered due to risk of hemorrhagic stroke and major noncerebral bleeding.[13]

Anticoagulation

NSTE-ACS: Upstream* Anticoagulant Therapy		
1	B-R	1. In patients with NSTE-ACS, intravenous unfractionated heparin (UFH) is useful to reduce ischemic events.†[1–4]
1	B-R	2. In patients with NSTE-ACS in whom an early invasive approach is not anticipated, either enoxaparin or fondaparinux are recommended alternatives to UFH to reduce ischemic events.[5–11]
Anticoagulant Therapy in Patients Undergoing Coronary Revascularization		
1	C-LD	3. In patients with ACS undergoing coronary revascularization (CABG or PCI) in the same admission, parenteral anticoagulation should be continued until revascularization to reduce ischemic events.[12,13]

Anticoagulant Therapy to Support PCI in ACS (STEMI and NSTE-ACS)		
1	C-EO	4. In patients with ACS undergoing PCI, intravenous UFH is useful to reduce ischemic events.†
1	B-R	5. In patients with STEMI undergoing PCI, bivalirudin is useful as an alternative to UFH to reduce mortality and bleeding.†[14–19]
2b	B-R	6. In patients with NSTE-ACS undergoing PCI, bivalirudin may be reasonable as an alternative to UFH to reduce bleeding.†[16,20–23]
2b	B-R	7. In patients with ACS, intravenous enoxaparin may be considered as an alternative to UFH at the time of PCI to reduce ischemic events.‡[7,8,24–26]
3: Harm	B-R	8. In patients with ACS, fondaparinux should not be used to support PCI because of the risk of catheter thrombosis.[5,27]

STEMI: Anticoagulant Therapy Treated With Fibrinolytic Therapy		
1	A	9. In patients with STEMI treated with fibrinolytic therapy, parenteral anticoagulation should be continued for the duration of the hospital stay (maximum of 8 days) or until revascularization is performed to reduce ischemic events.[9,10,27–29]
1	A	10. In patients with STEMI treated with fibrinolytic therapy who are not intended to undergo an invasive approach, enoxaparin is the recommended anticoagulant to reduce ischemic events.[9,10,28,29]
1	B-R	11. In patients with STEMI treated with fibrinolytic therapy who are not intended to undergo an invasive approach, fondaparinux is a recommended alternative to reduce ischemic events.[27,30]

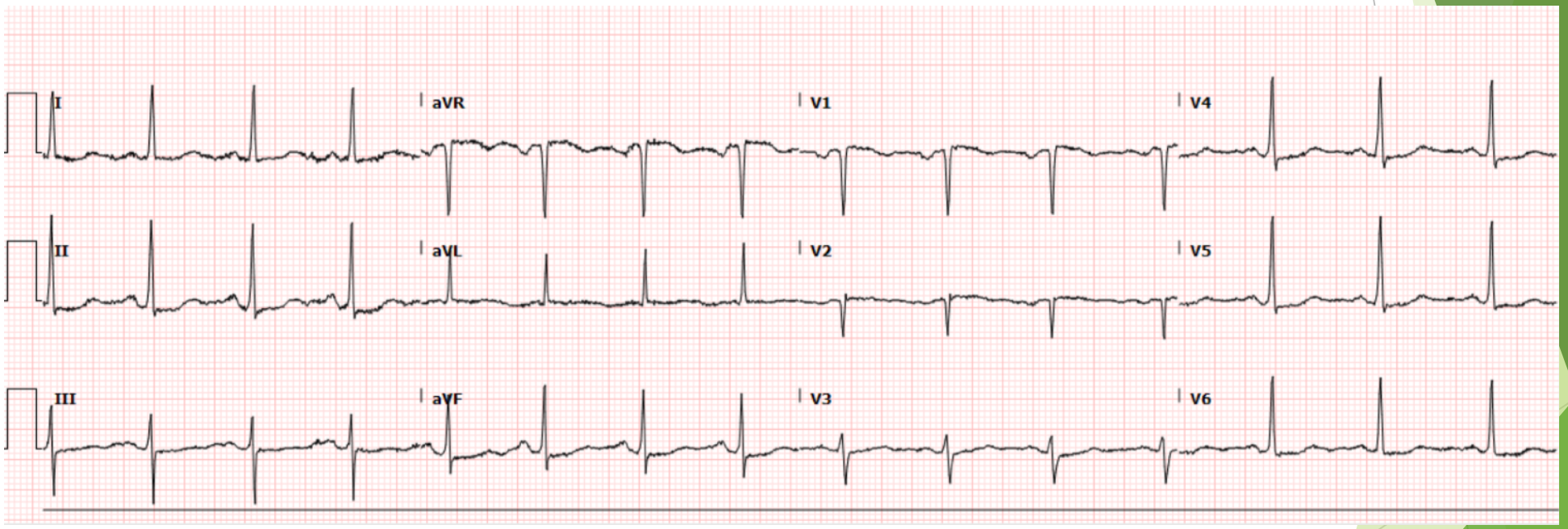
For Your Reference

UFH	**Initial therapy:** Loading dose 60 IU/kg (max 4000 IU), with initial infusion 12 IU/kg per h (max 1000 IU/h) adjusted to therapeutic aPTT range of 60-80 s. **To support PCI:** In patients who have received prior anticoagulant therapy, additional UFH as needed to achieve an ACT 250-300 s. In patients who have not received prior anticoagulant therapy, 70-100 U/kg initial bolus to achieve target ACT of 250-300 s. **With fibrinolytic therapy:** Loading dose 60 IU/kg (maximum 4000 IU) with initial infusion 12 IU/kg per h (maximum 1000 IU/h) adjusted to therapeutic aPTT range.
Bivalirudin	**To support PCI:** 0.75 mg/kg bolus, 1.75 mg/kg per h IV infusion during the PCI procedure. **Post-PCI infusion for PPCI:** 1.75 mg/kg per h for 2-4 h post-PCI. In patients with CrCl <30 mL/min, reduced infusion to 1 mg/kg per h.
Enoxaparin	**Initial therapy:** 1 mg/kg subcutaneous every 12 h. Reduce dose to 1 mg/kg per d subcutaneous if CrCl <30 mL/min. **To support PCI:** For previous treatment with enoxaparin, if the last subcutaneous dose was administered 8-12 h earlier or if only 1 subcutaneous dose of enoxaparin has been administered, an IV dose of 0.3 mg of enoxaparin should be given. If the last dose was administered within the previous 8 h, no additional enoxaparin should be given. For patients who have not received prior anticoagulant therapy, 0.5-0.75 mg/kg IV bolus. **With fibrinolytic therapy:** If age <75 y, 30 mg IV bolus, followed in 15 min by 1 mg/kg subcutaneous every 12 h (maximum 100 mg for the first 2 doses). If age ≥75 y: no bolus, 0.75 mg/kg subcutaneous every 12 h (maximum 75 mg for the first 2 doses). Regardless of age, if CrCl <30 mL/min: 1 mg/kg subcutaneous every 24 h.
Fondaparinux	**Initial therapy:** 2.5 mg subcutaneous daily. **With fibrinolytic therapy:** 2.5 mg IV, then 2.5 mg subcutaneous daily starting the following day. Contraindicated if CrCl <30 mL/min. Fondaparinux should not be used to support PCI because of the risk of catheter thrombosis.

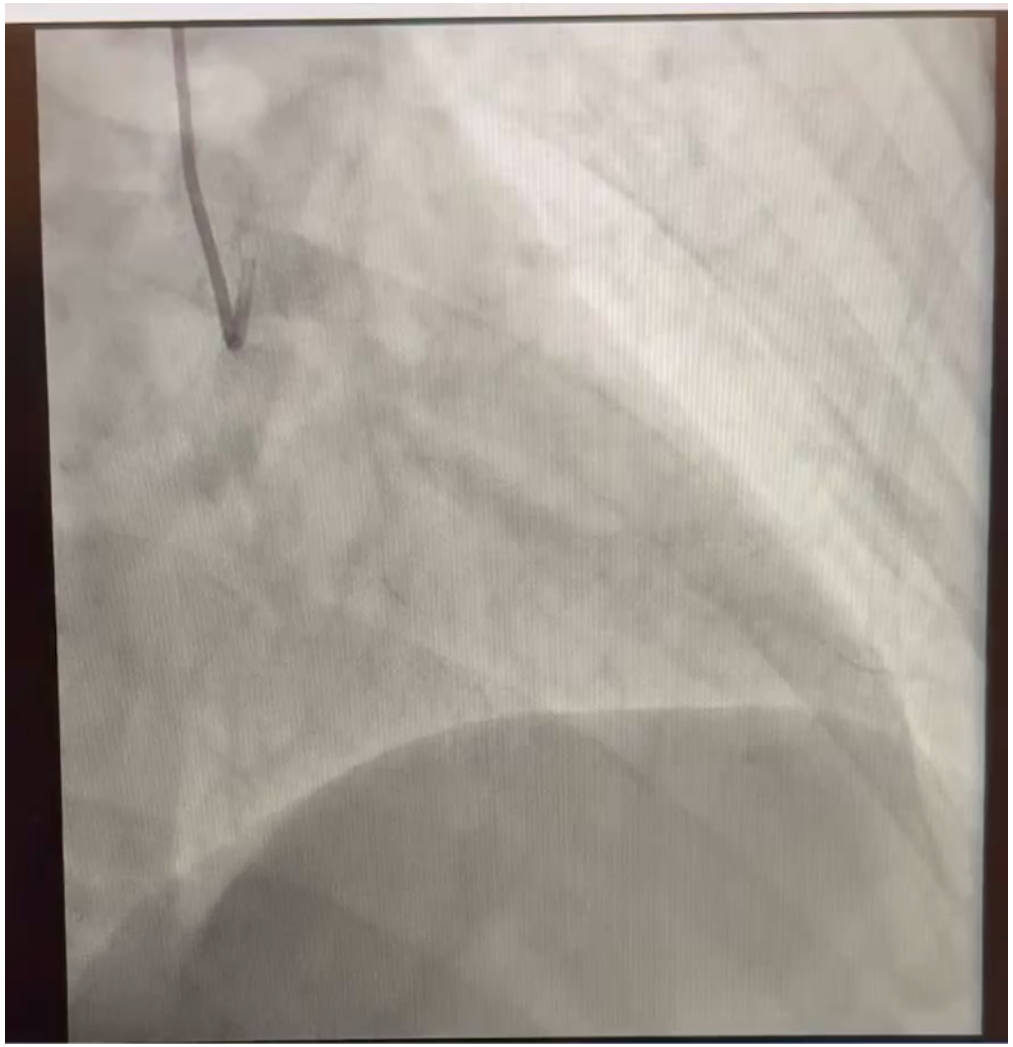
Case Presentation

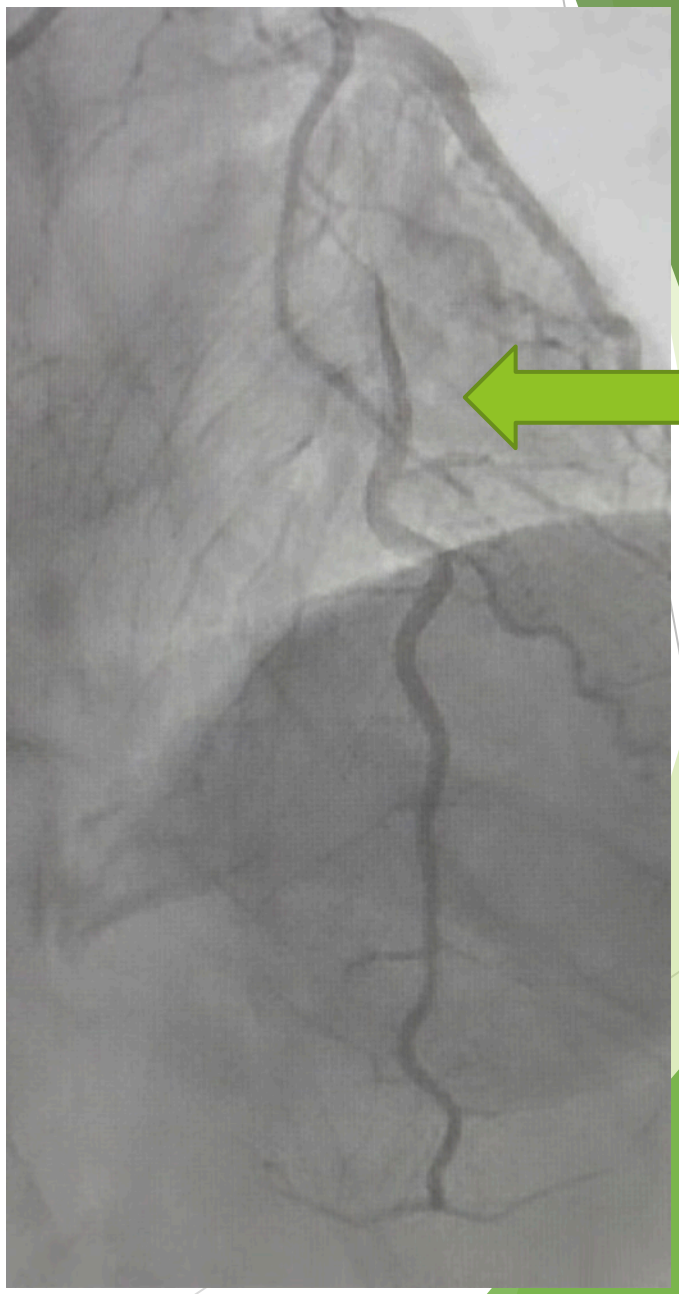
- 51yo female presenting with sudden onset chest pain, bilateral arm paresthesia, dyspnea and diaphoresis
- EMS called, initial ECG showing possible STEMI
- Patient given 3 rounds of nitro with resolution of symptoms; ECG as follows..
- CXR, CT scan no significant findings
- Labs show hs-Trop 29, increase to 357
- Started on ACS protocol and medications

ECG



Cath

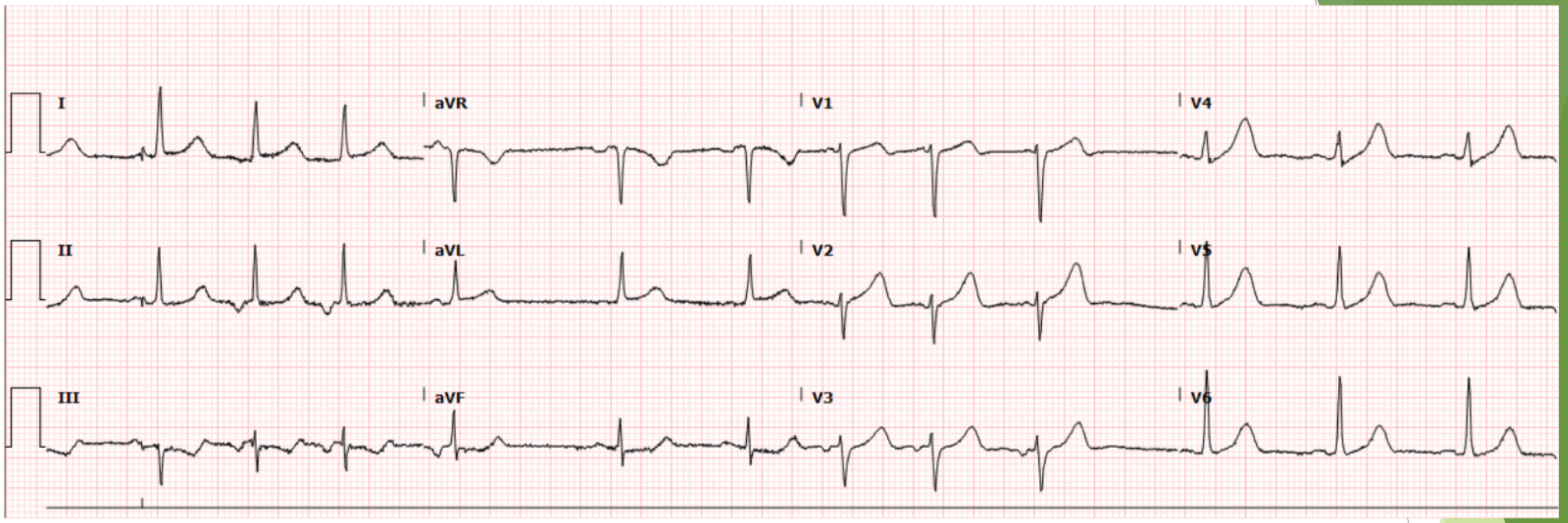




SCAD- Spontaneous Coronary Artery Dissection

- Patient spends several days in the hospital for medical management and observation
- CP symptoms resolve on medical therapy
  - So do ECG changes
- Discharged home on DAPT with scheduled outpatient follow up

- 2 days later presents via EMS to the ED with crushing chest pain..


I
aVR
V1
V4
II
aVL
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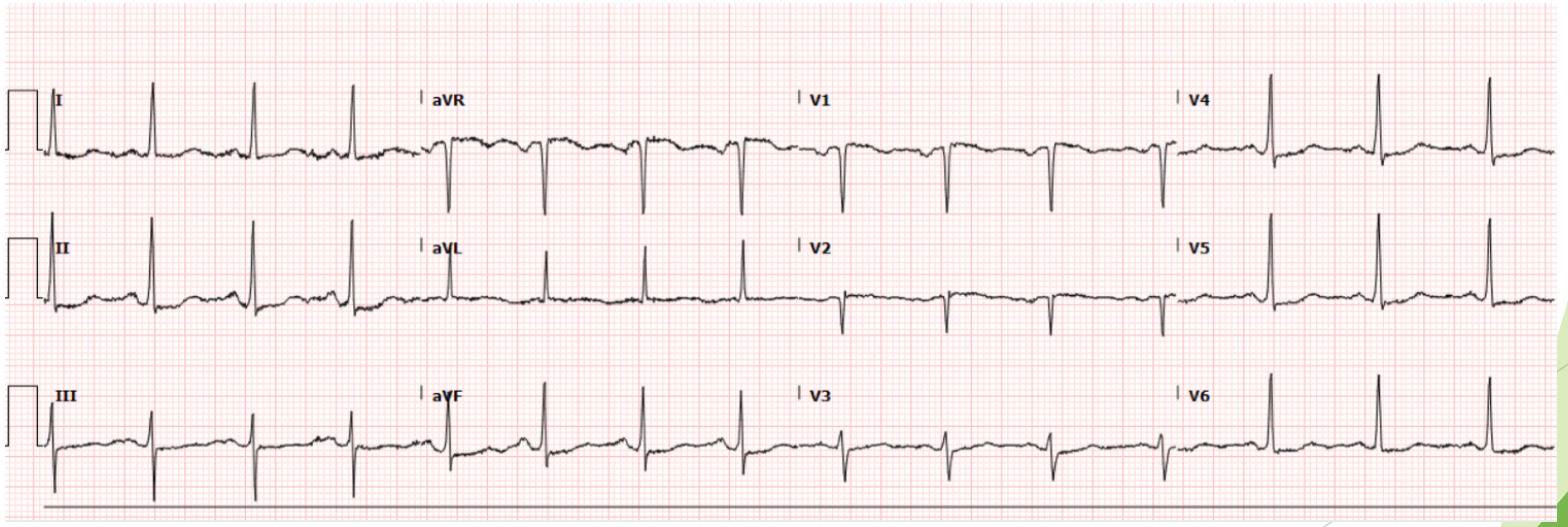
III
aVF
V3
V6


I
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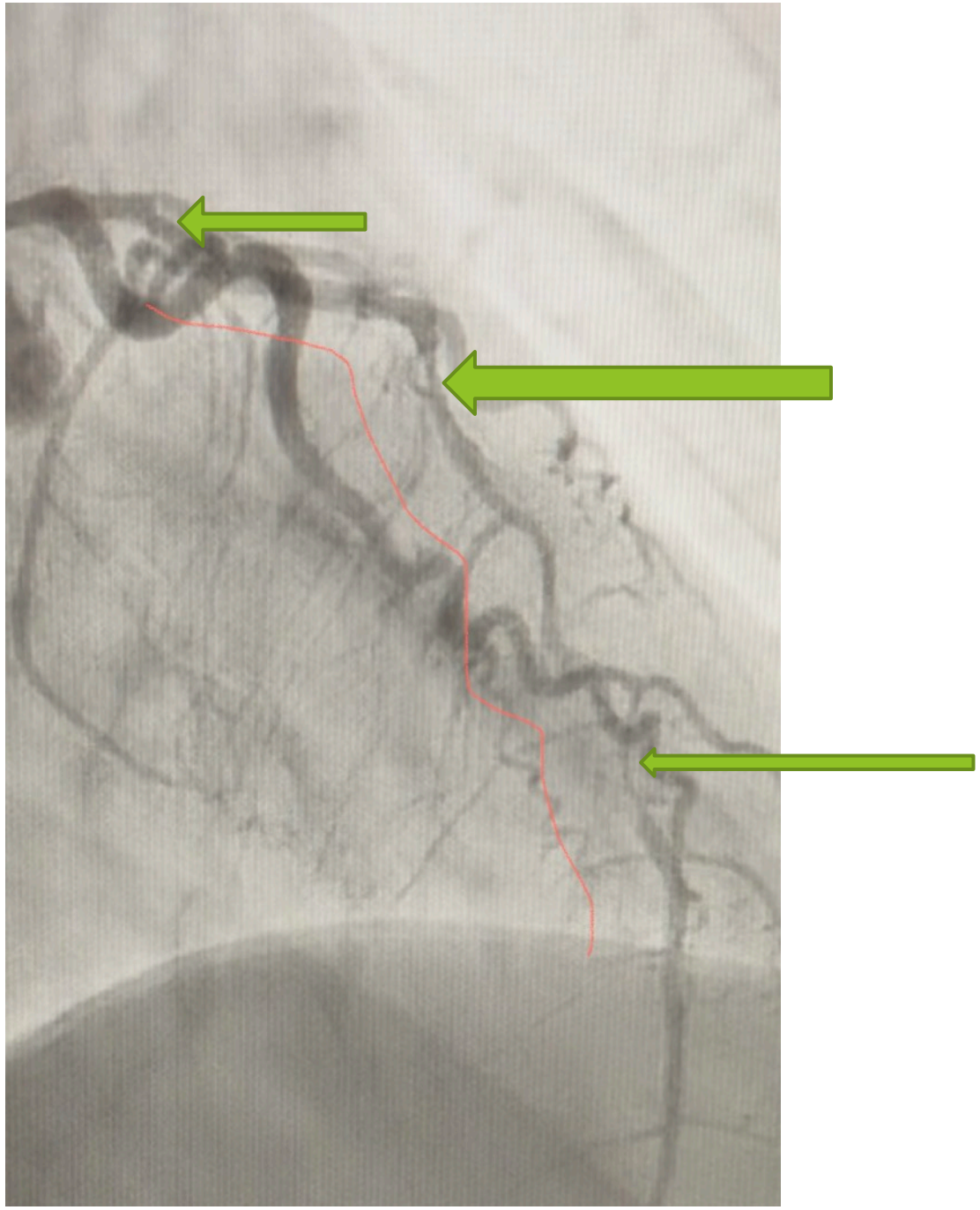

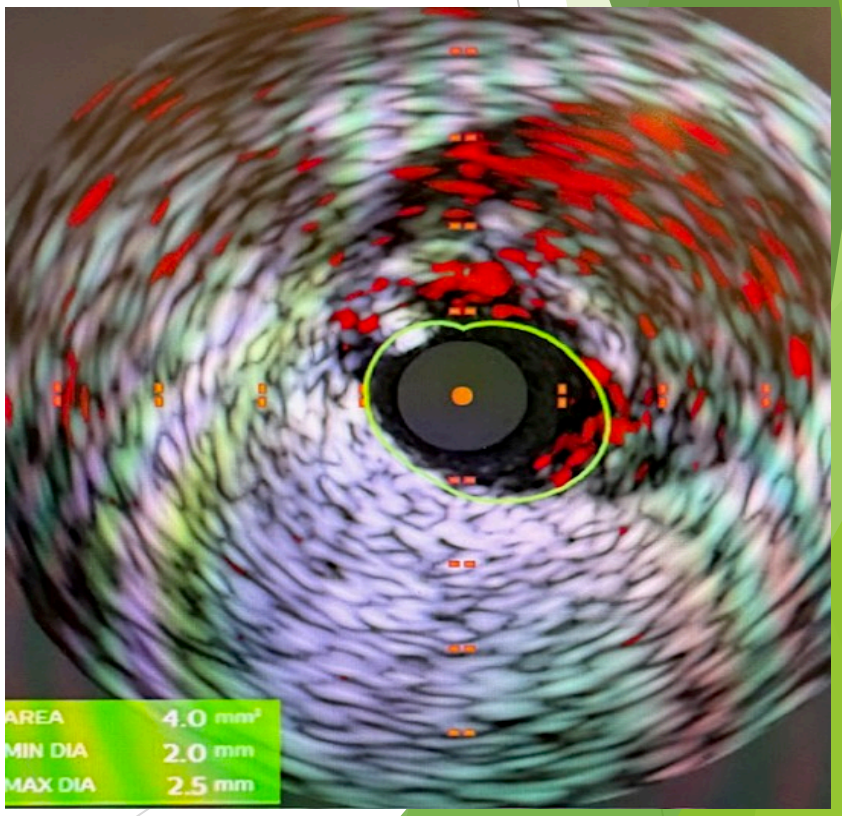
AREA 4.0 mm²
MIN DIA 2.0 mm
MAX DIA 2.5 mm

Follow up

- Patient went for emergent OHS and received CABG x2.
- Ejection fraction has since normalized
- Patient was discharged several days later and is now approximately 2 months post- surgery, Alive and Doing well.

Final Thoughts

- “Patient’s don’t read the book.”
- A good history and physical exam can narrow it down
- Rapid recognition and treatment of patients can literally save lives; especially in ACS and cardiogenic shock
- PCI has changed overall mortality, lowering complications, and vital in patients with ACS presenting via EMS
- Guidelines are guidelines, sometimes deviation is reasonable in a case by case basis

Questions?

- THANK YOU!