Coronary Artery Disease

From Plaque Biology to Clinical Decisions

A practical framework for primary care

Tony Chen, MD

General Cardiologist

Virginia Mason Franciscan Health

Seattle, Issaquah, Bellevue

No disclosures/conflict of interest

How We Will Think About CAD today

- Plaque biology → 2 clinical syndromes
- Myocardial injury vs myocardial infarction
- Ischemia → cardiac stress testing → core pillars of CAD therapy

Anchoring Case: Meet the Patient

- 62-year-old man with HTN, diabetes, hyperlipidemia
- 3 months of exertional chest tightness
- Normal resting EKG

What would you do next?

1. Referral to cardiology.
2. Order a cardiac stress test.
3. Refer to ED for expediated evaluation.
4. Start a baby aspirin, statin, and beta blocker, follow up in 3-4 weeks.

What Is Coronary Artery Disease?

- Chronic inflammatory disease of the coronary arteries
- Initiated by endothelial dysfunction and lipid accumulation
- Progresses silently over decades

From Plaque Biology to Clinical Syndromes

CAD comes in two clinical phenotypes

Stable plaque progression

Chronic Coronary Syndrome

Stable predictable angina

Plaque rupture / erosion

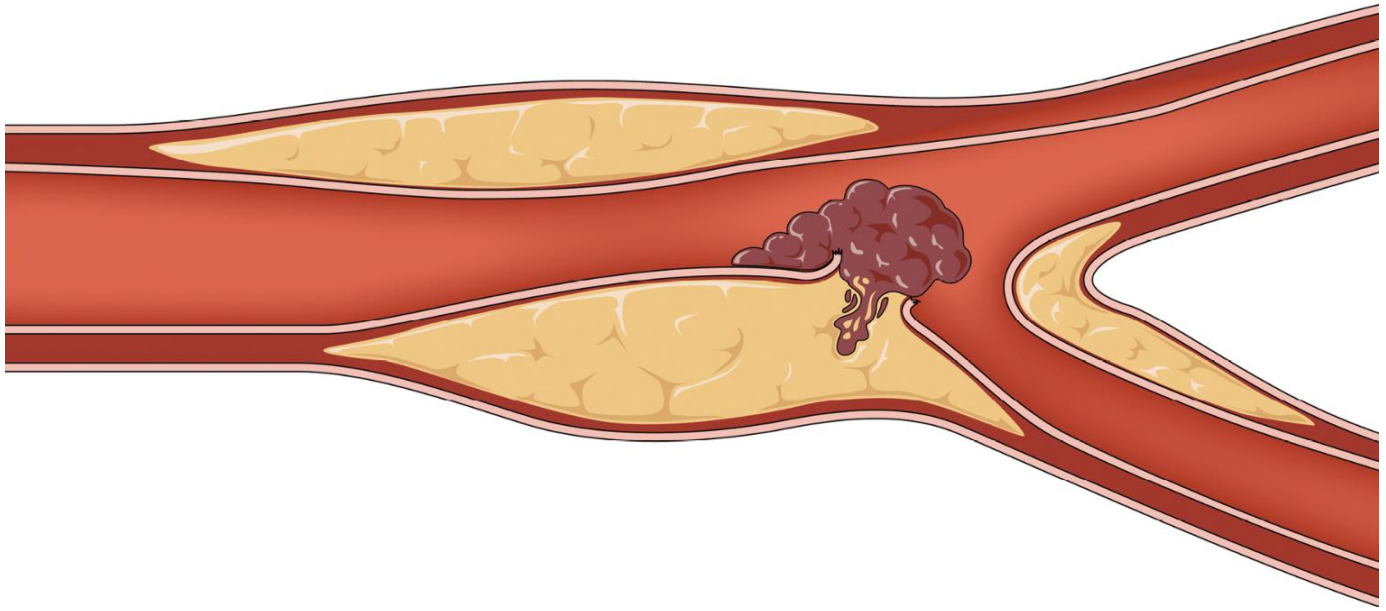
Acute Coronary Syndrome

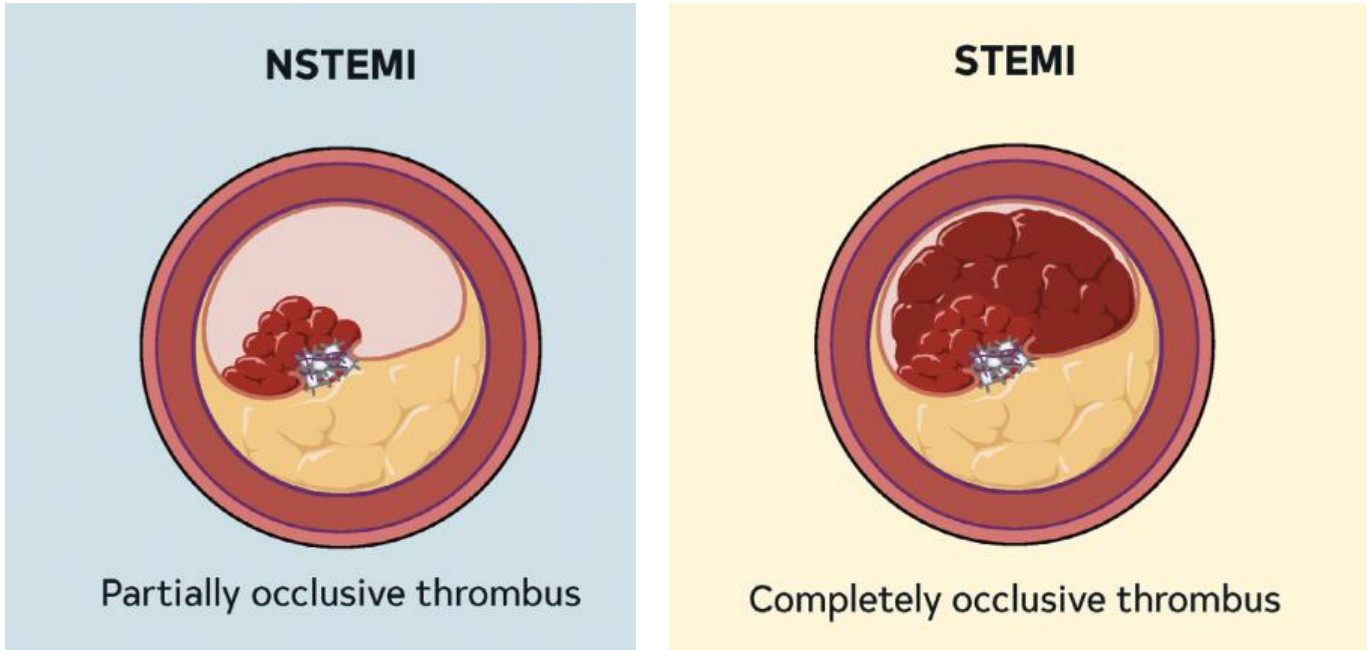
Unstable angina, NSTEMI, STEMI

Chronic Coronary Syndrome

- Most common CAD presentation
- Fixed stenosis → predictable supply–demand ischemia

Acute Coronary Syndrome (ACS)





Anchoring Case: Syndrome Classification

In the emergency room:

- Negative troponin
- EKG without acute ischemic changes

- Is this ACS or CCS?
- Discharged with ASA 81mg qday, high-intensity statin, metopolol succinate 25mg qday, sublinqual nitro prn
- Referral to cardiology

Anchoring Case: Injury or Infarction

The following day:

Sudden onset palpitations, generalized weakness, very slight chest discomfort.

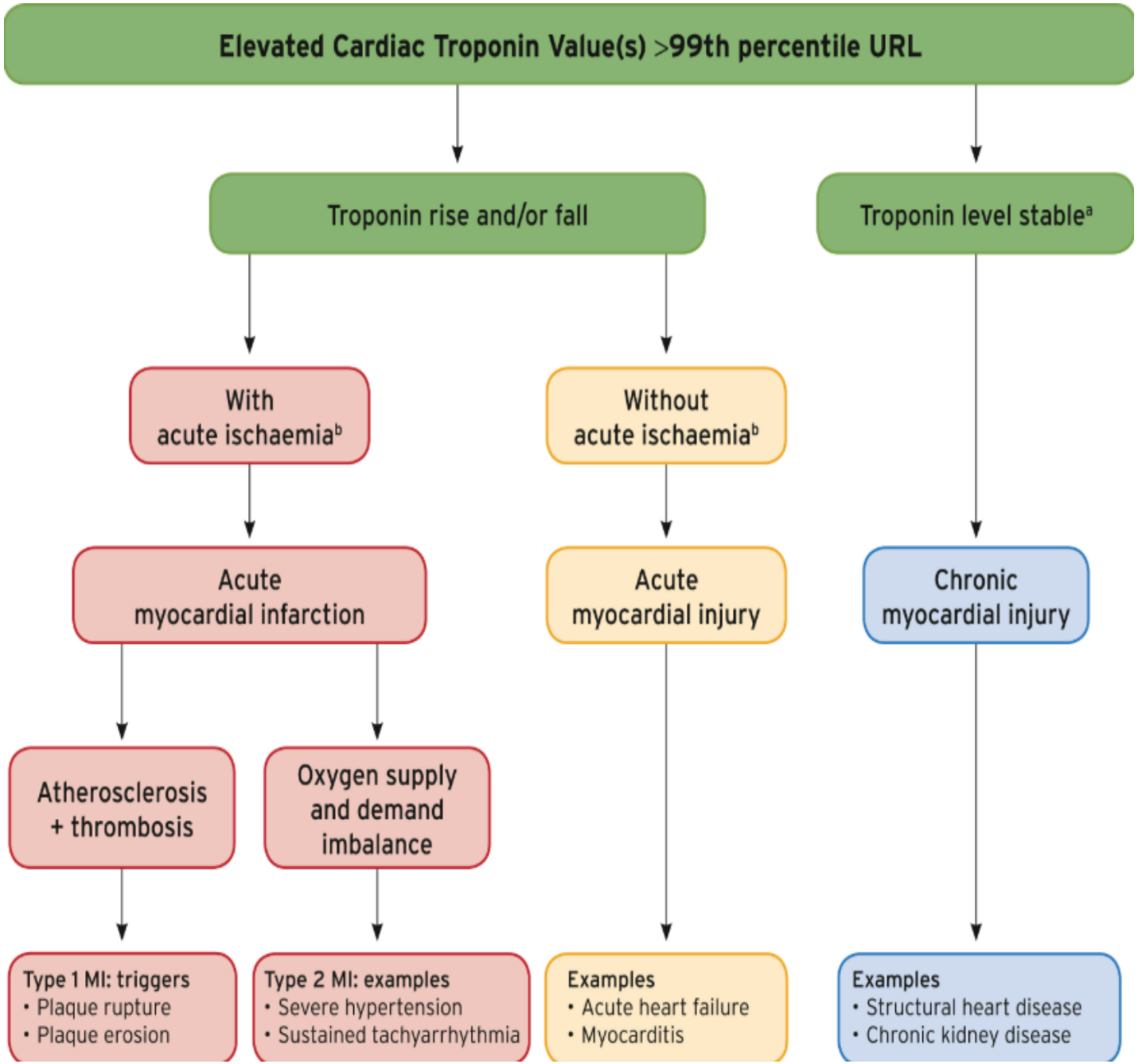
EKG showing atrial fibrillation RVR 130-150 bpm. Stable BP.

In the ED, HS-troponin trend slightly elevated (40 → 50 → 30).

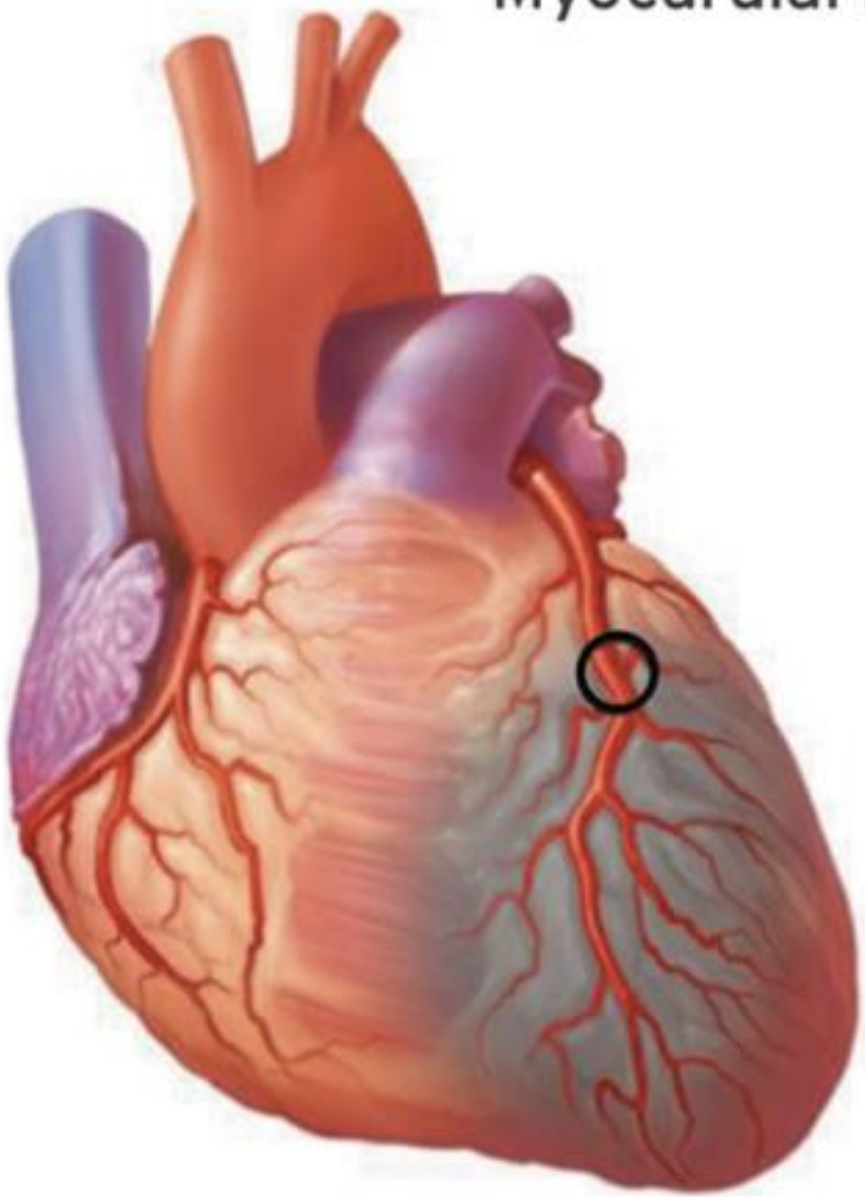
Did the patient have a heart attack?

Troponin Elevation: Injury vs Infarction

- Troponin elevation defines myocardial injury
- Acute injury shows rise and fall
- Myocardial infarction requires ischemia


Elevated Cardiac Troponin Value(s) >99th percentile URL
Troponin rise and/or fall
Troponin level stable[a]
With acute ischaemia[b]
Without acute ischaemia[b]
Acute myocardial infarction
Acute myocardial injury
Chronic myocardial injury
Atherosclerosis + thrombosis
Oxygen supply and demand imbalance
Type 1 MI: triggers
• Plaque rupture
• Plaque erosion
Type 2 MI: examples
• Severe hypertension
• Sustained tachyarrhythmia
Examples
• Acute heart failure
• Myocarditis
Examples
• Structural heart disease
• Chronic kidney disease

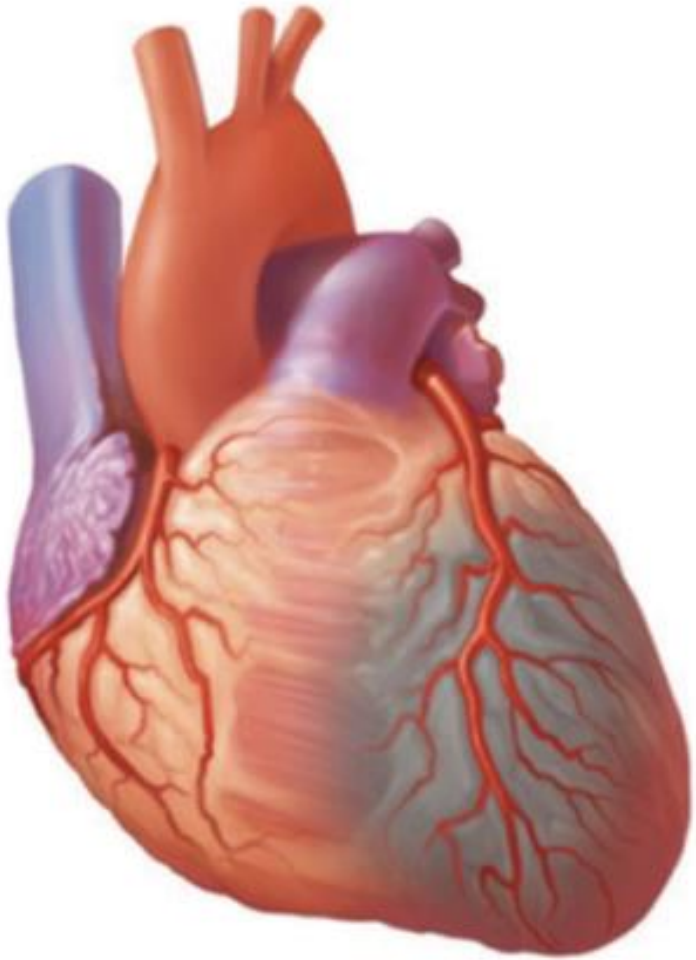
Myocardial Infarction Type 1



Plaque rupture/erosion with occlusive thrombus

Plaque rupture/erosion with non-occlusive thrombus

Myocardial Infarction Type 2

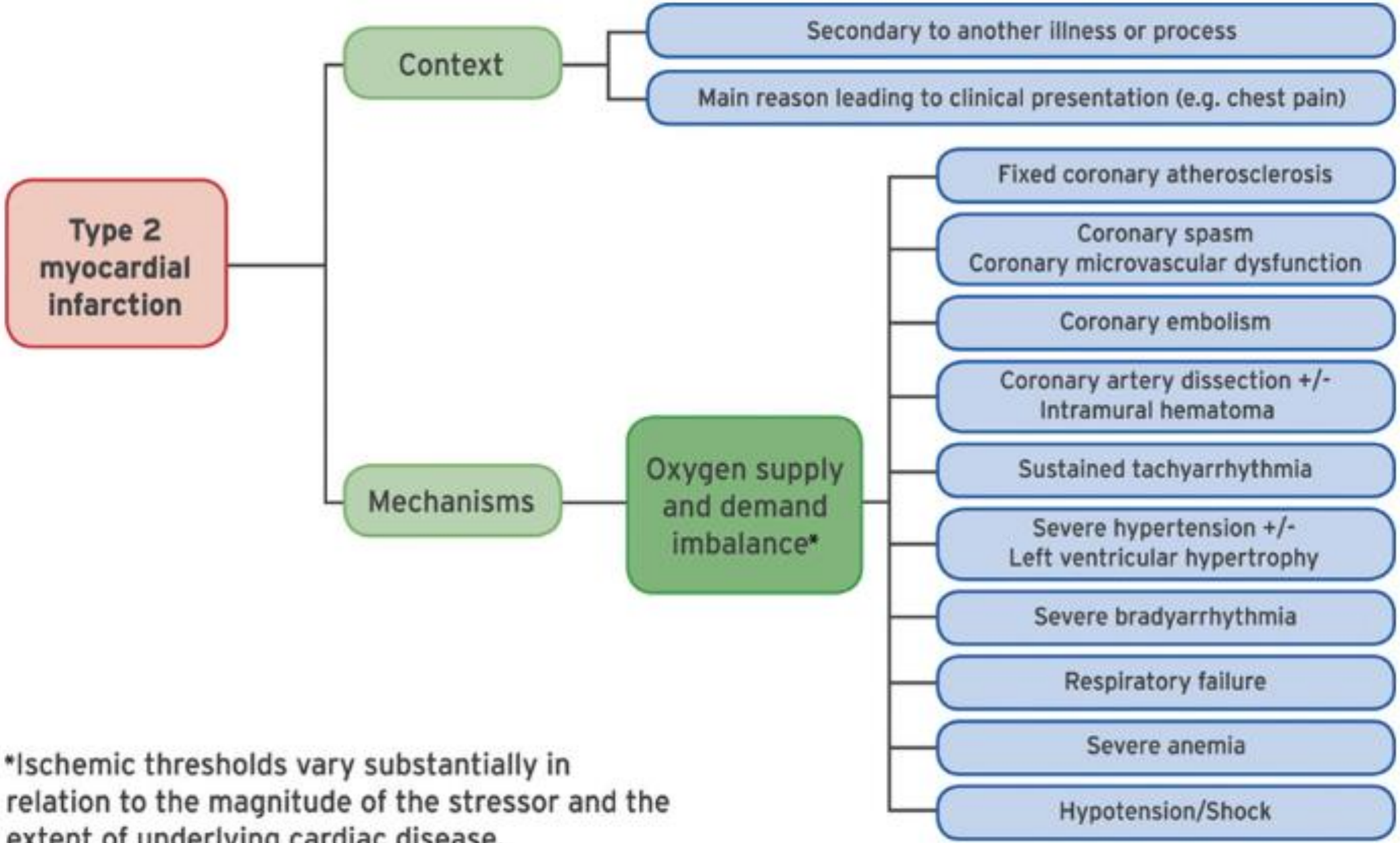


Atherosclerosis and oxygen supply/demand imbalance

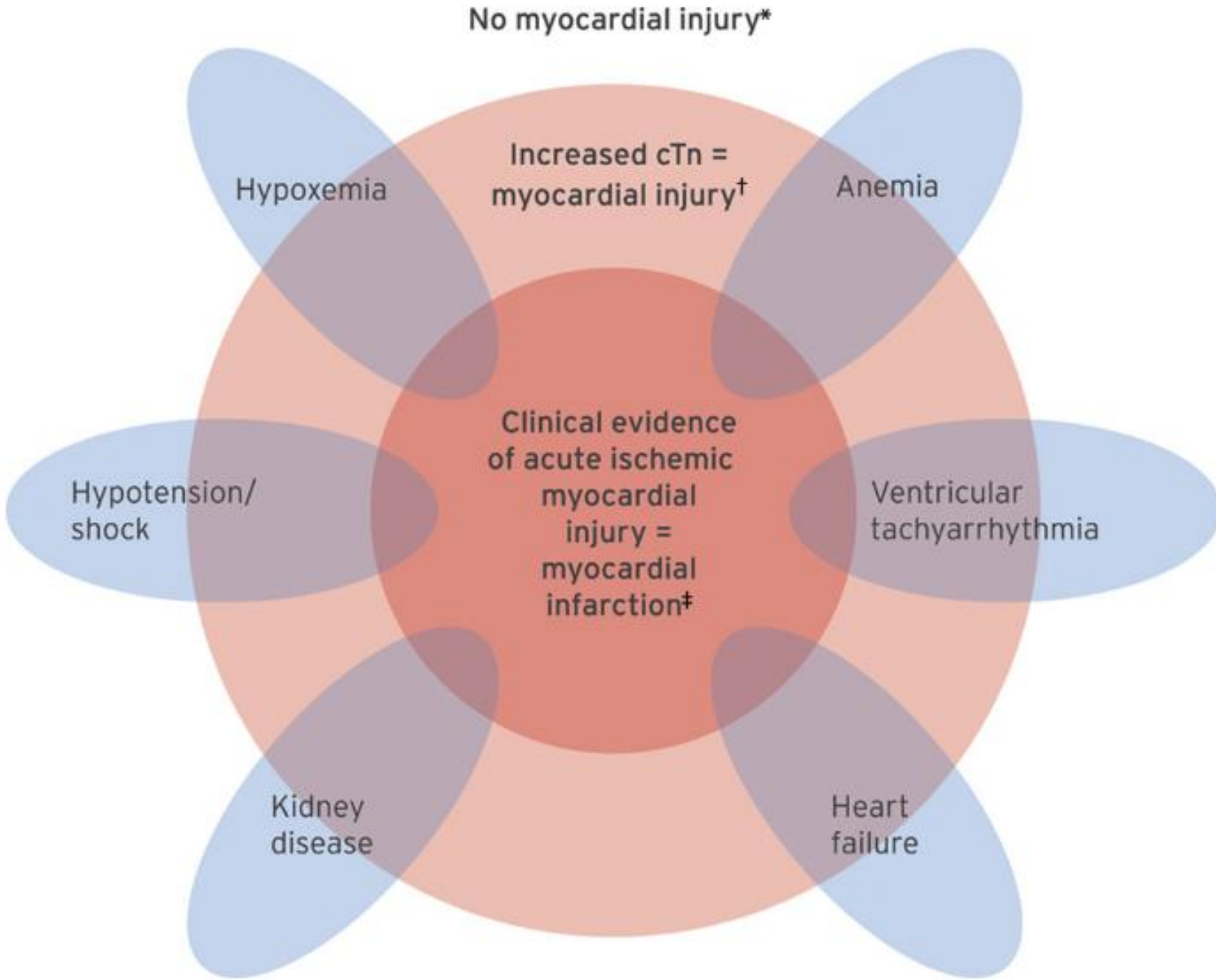
Vasospasm or coronary microvascular dysfunction

Non-atherosclerotic coronary dissection

Oxygen supply/demand imbalance alone



*Ischemic thresholds vary substantially in relation to the magnitude of the stressor and the extent of underlying cardiac disease.

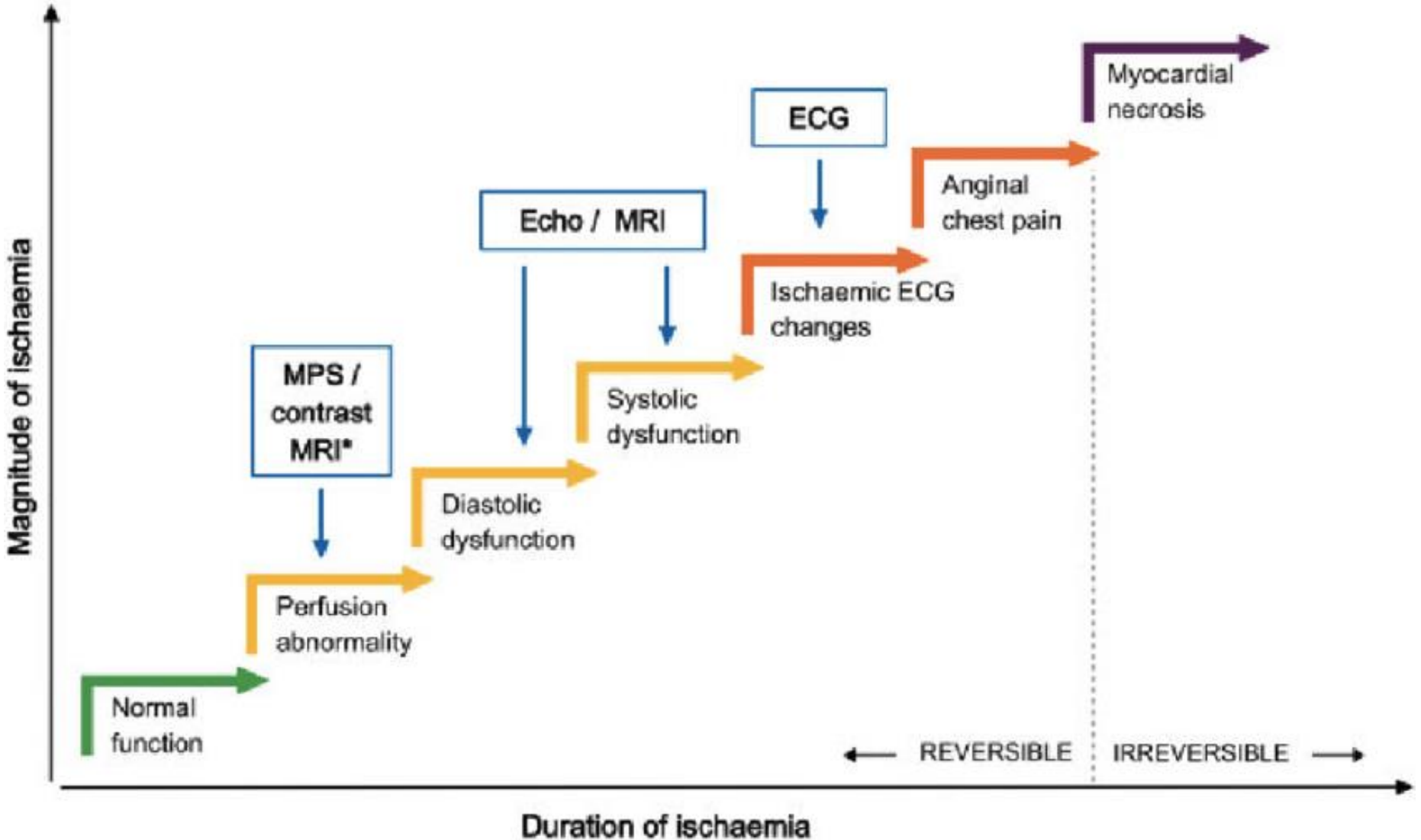

No myocardial injury*
Increased cTn = myocardial injury†
Hypoxemia
Anemia
Clinical evidence of acute ischemic myocardial injury = myocardial infarction‡
Hypotension/ shock
Ventricular tachyarrhythmia
Kidney disease
Heart failure

Anchoring Case: Testing Strategy

Presents to cardiology clinic

- high pretest probability → cath lab
- low → continue to monitor
- Intermediate → stress test

Ischemic Cascade



Choosing a Stress Test

- Intermediate pre-test probability of disease
- Decide how you want to stress the patient
  - Exercise
  - Pharmacologic
- Decide how you want to acquire the stress data
  - EKG only
  - EKG + echocardiogram
  - EKG + nuclear perfusion imaging

Common Options to Consider

- Treadmill stress test

- Exercise stress echo
- Dobutamine stress echo

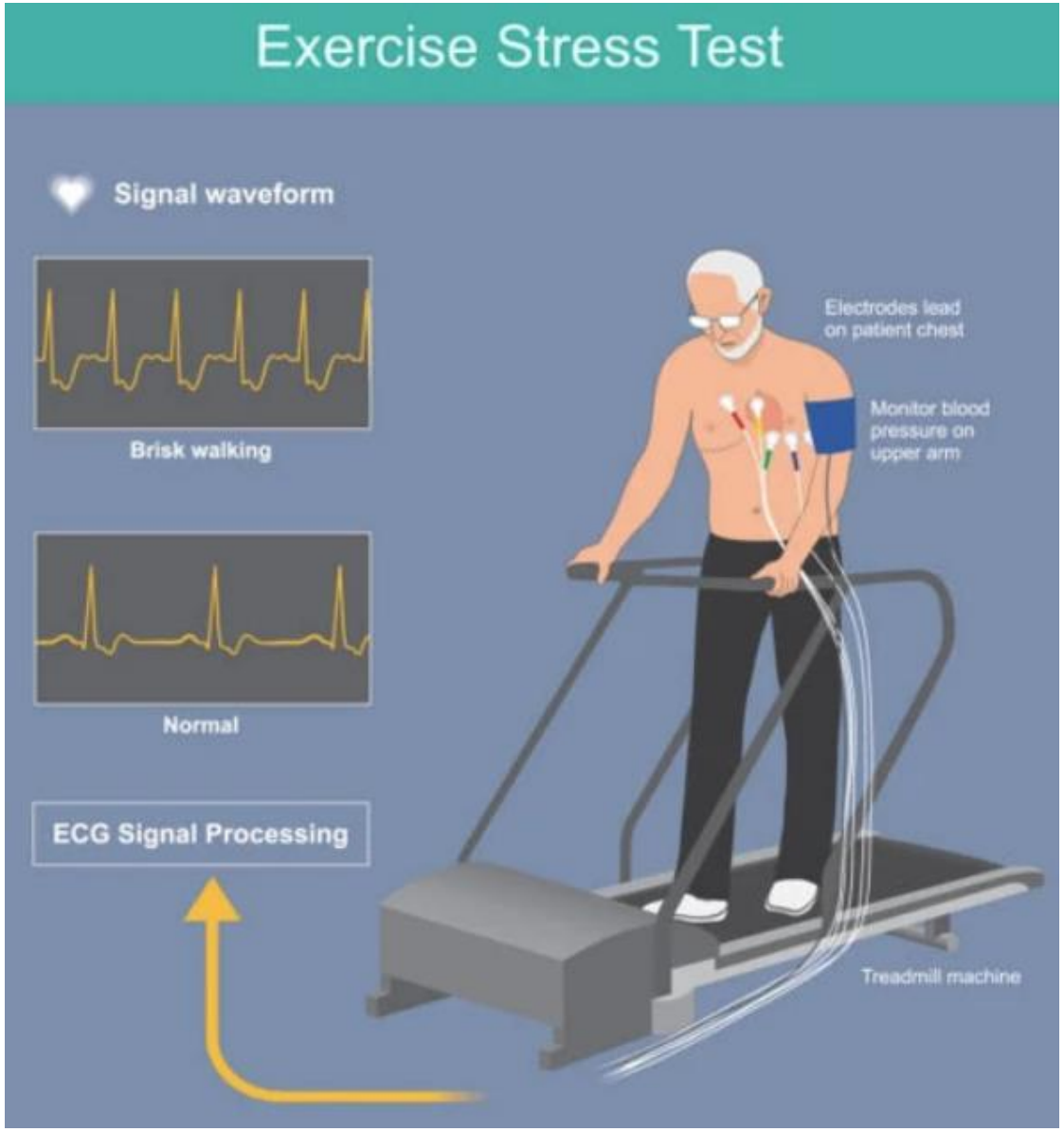
- Exercise myocardial perfusion imaging
- Pharmacologic myocardial perfusion imaging (Lexiscan)

Treadmill stress test

- Interpretable baseline EKG

- Uninterpretable EKG
  - >1mm resting ST depression
  - LBBB
  - Ventricular pacing
  - ST/T changes related to LVH or digoxin

- Sensitivity/specificity: 0.58/0.62

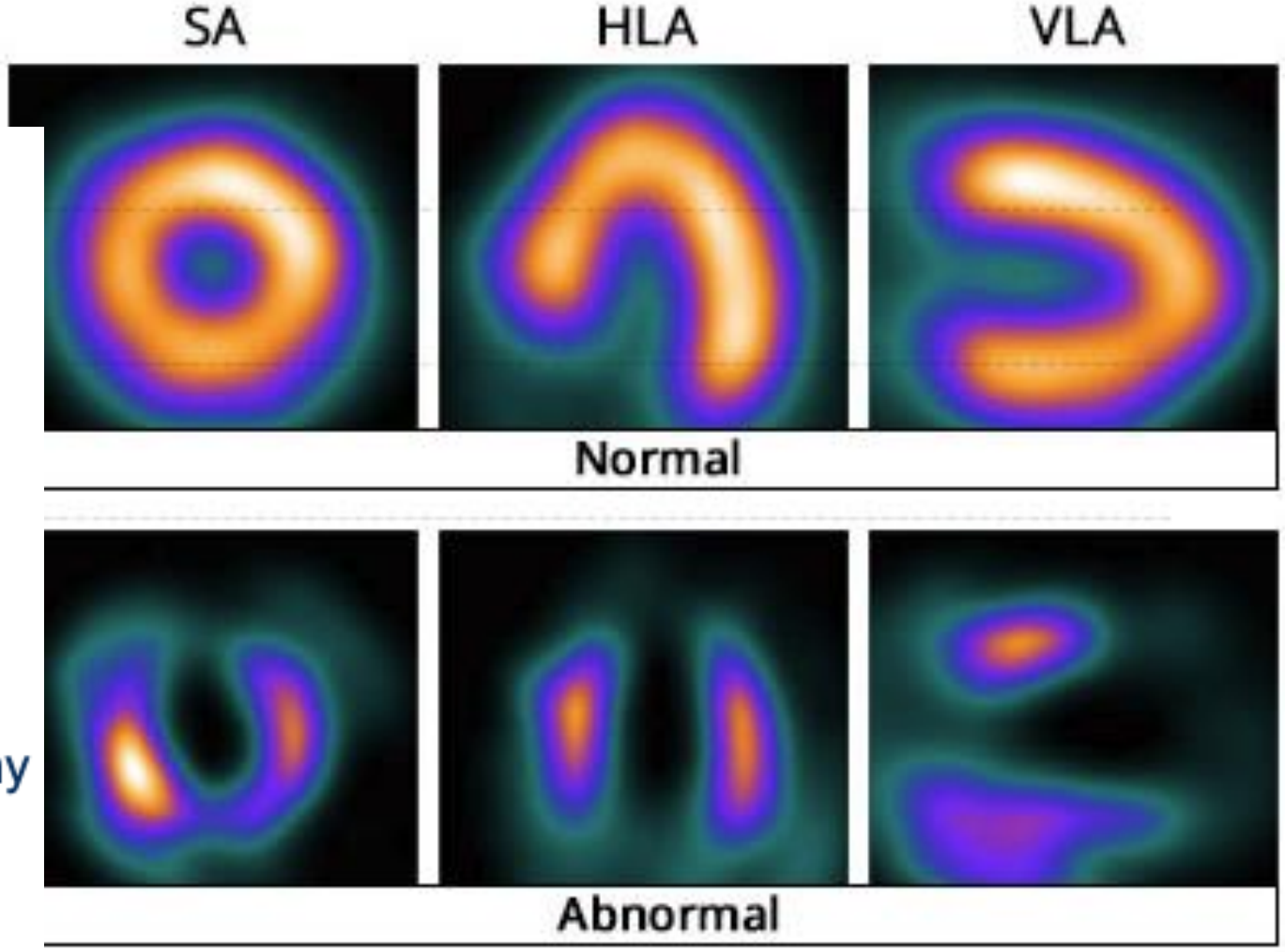


SPECT Myocardial perfusion imaging (nuclear stress test)

Sensitivity
Exercise MPI: 82-88%*
Pharmacologic MPI: 88-91%*

Specificity
Exercise MPI: 70-88%*
Pharmacologic MPI: 75-90%*

Test Characteristics compared to angiography



ACCF/AHA Stable Ischemic Heart Disease Guidelines . JACC 2012

Stress echocardiogram

- Absolute contraindication
- Malignant hypertension
- ACS within 24 hours
- Critical aortic stenosis
- Severe LVOT obstruction
- Severe arrhythmia

Diagnostic Accuracy

Stress Echo

- Sensitivity 88%
- Specificity 83%
- Sensitivity is better for multivessel disease
- Stress echo comparable sensitivity to nuclear but ⬆ specificity
- LM/MV CAD Stress echo ⬆ sensitivity (cannot miss balanced ischemia)
- DSE and Dipyridamole ~ similar sensitivity and specificity

Test	Stress electrocardiography	Stress echocardiography	Stress myocardial perfusion imaging	Coronary computed tomographic angiography	Invasive coronary angiography
Requirements and considerations	Exercise stress on a treadmill Requires interpretable electrocardiogram (eg, no left bundle-branch block or major ST- and T-wave changes) at baseline	Exercise or pharmacological stress (dobutamine [with atropine if necessary to achieve target heart rate] or adenosine derivatives) For patients with poor-quality echocardiographic images, contrast may improve the interpretability of the test	Exercise or pharmacological stress (vasodilator) Higher radiation exposure than other noninvasive tests	β-Blocker to lower heart rate and nitroglycerin for vasodilation Use with caution for patients with kidney impairment	Invasive procedure with bleeding risks (lower with radial vs femoral approach) Use with caution for patients with kidney impairment
Sensitivity	**0.58** (95% CI, 0.46-0.69)	**0.85** (95% CI, 0.80-0.89)	**0.87** (95% CI, 0.83-0.90)	**0.97** (95% CI, 0.93-0.99)	NA
Specificity	**0.62** (95% CI, 0.54-0.69)	**0.82** (95% CI, 0.72-0.89)	**0.70** (95% CI, 0.63-0.76)	**0.78** (95% CI, 0.67-0.86)	NA
Findings indicating high risk	>2-mm ST-segment depressions at low workload ST-segment elevations or ventricular tachycardia or ventricular fibrillation	Decrease in left ventricular ejection fraction (LVEF) >10% or left ventricular (LV) dilation Wall motion abnormalities in multiple coronary territories Baseline LV dysfunction	Decrease in LVEF >10% or LV dilation Perfusion defect in >10% of myocardium Baseline LV dysfunction	Multiple coronary arteries with ≥70% stenosis Left main stenosis ≥50%	Multiple coronary arteries with ≥70% stenosis Left main stenosis ≥50%

Medical Therapy: Core Pillars

- Antiplatelet therapy
  - Aspirin 81mg qday
  - Plavix
  - Dual antiplatelet therapy (DAPT)

- Lipid lowering therapy
  - High intensity statin
  - High intensity statin + ezetimibe
  - PCSK-9 inhibitor
    - alirocumab (Praluent)
    - evolocumab (Repatha)

- Anti-anginal therapy

Why Anti-anginal Therapy Works

- Angina reflects myocardial oxygen supply–demand mismatch
- If supply cannot be increased, reduce demand

- Beta blockers, nitrates, BP control

JACC Vol. 35, No. 5, Suppl B
April 2000;45B–48B

Table 1. Determinants of Myocardial Oxygen Consumption

1	Tension development
2.	Contractile state
3.	Heart rate
4	Basal cost
5.	Depolarization
6.	Direct metabolic effect of catecholamines
7.	Activation
8	Maintenance of active state
9.	Shortening against a load–Fenn effect

Anchoring Case: Treatment strategy

Myocardial perfusion imaging:

- Small size, moderate severity, reversible ischemia in the distal anterior wall. No TID.
- Normal LVEF

What should we do:

1) Continue medical therapy (ASA, statin, BB)
2) Go to the cath lab
3) Coronary CTA to rule out left main disease. If +LM disease → cath lab

Key Takeaways for Primary Care

- CAD presents in 2 clinical expressions: CCS and ACS
- Troponin elevation does not necessarily mean myocardial infarction
- Strategically choose your stress test based on your objective, using the ischemic cascade as a framework
- Known the components of CAD medical therapy: antiplatelet, lipid-lowering, anti-anginal.

Questions