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5 Advances in Cardiac Care that All Internists Should Know

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Associate Professor of Medicine
Director, Nuclear Cardiology
Medical Director, Outpatient Cardiology
Senior Associate Director, Training Program in CV Diseases
Leon H Charney Division of Cardiology,
NYU Grossman School of Medicine

Former Governor, New York Chapter (Manhattan) of the American College of Physicians

October 24, 2026

~~5~~ Lots of Exciting Advances in Cardiac Care to Share With My IM Colleagues ~~that All Internists Should Know~~

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Disclosures

- Novo Nordisk (Consultant – Research Clinical Adjudication Committee)
- Eli Lilly (Consultant – Research Clinical Adjudication Committee)

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Introduction:



Created using ChatGPT

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2026 ACC/AHA Guideline on the Management of Dyslipidemia

- Replaces the 2018 Cholesterol Guidelines.
- Revises and updates tools for risk stratification to personalize risk.
- Adds recommendations on testing and utilization of ApoB and Lp(a).
- Swings the pendulum back to earlier treatment and absolute goals for LDL/non-HDL as well as percent reduction.

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Case #L1:

- 53 year old man without known medical history, presents for cardiac risk assessment
  - Non-smoker
  - Denied family medical history
  - Lives in Albany, NY (Zip code 12208)
  - BP 134/78 mmHg
  - BMI 29 kg/m2
  - Total Chol 220 mg/dL; LDL-C 145 mg/dL; HDL-C 48 mg/dL
  - eGFR 85 ml/min/1.73m2

What do you do next regarding LLT (lipid lowering therapy)?

- ✓ Recommend lifestyle modification alone
- ✓ Perform additional laboratory assessment
- ✓ Perform CV imaging
- ✓ Initiate moderate intensity LLT

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PREVENT-ASCVD

- Goal to estimate 10-year and 30-year risk for atherosclerotic CVD (ASCVD), heart failure (HF) and total cardiovascular disease (CVD).
- Validated form over 6.5 million US adults across multiple datasets.
- Validated in patients ages 30-79 years old.

https://professional.heart.org/en/guidelines-and-statements/prevent-calculator#riskreduction

CVD | **ASCVD** | Heart Failure

Sex* ○ Female ○ Male

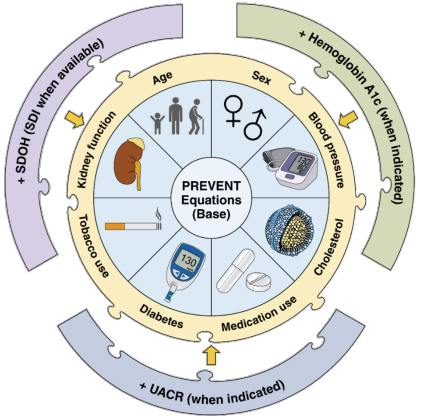
Age (years)*	**SBP (mmHg)***	**Total Cholesterol (mg/dL)***
30-79	90-200	130-320
HDL Cholesterol (mg/dL)*	**eGFR (mL/min/1.73m²)***	**BMI (kg/m²)***
20-100	15-140	18.5-39.9

Diabetes
Any history of diabetes.
● No ○ Yes

Current Smoking
Any cigarette use within the last 30 days
● No ○ Yes

Lipid-lowering medication
Current use of statin medication to lower cholesterol
● No ○ Yes

Anti-hypertensive medication
Current use of any medication for hypertension
● No ○ Yes



Additional data points if available:

- ✓ UACR (urine albumin-to-creatinine ratio) (mg/g)
- ✓ HbA1C
- ✓ Zip Code

Source: https://professional.heart.org/en/guidelines-and-statements/prevent-calculator#riskreduction

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- **53 year old man** without known medical history, presents for cardiac risk assessment
  - Non-smoker
  - Denied family medical history
  - Lives in Albany, NY (Zip code **12208**)
  - BP **134**/78 mmHg
  - BMI **29** kg/m2
  - Total Chol **220** mg/dL; LDL-C 145 mg/dL; HDL-C **48** mg/dL
  - eGFR **85** ml/min/1.73m2

Estimated **10-year** risk of ASCVD

3.8%

See charted results

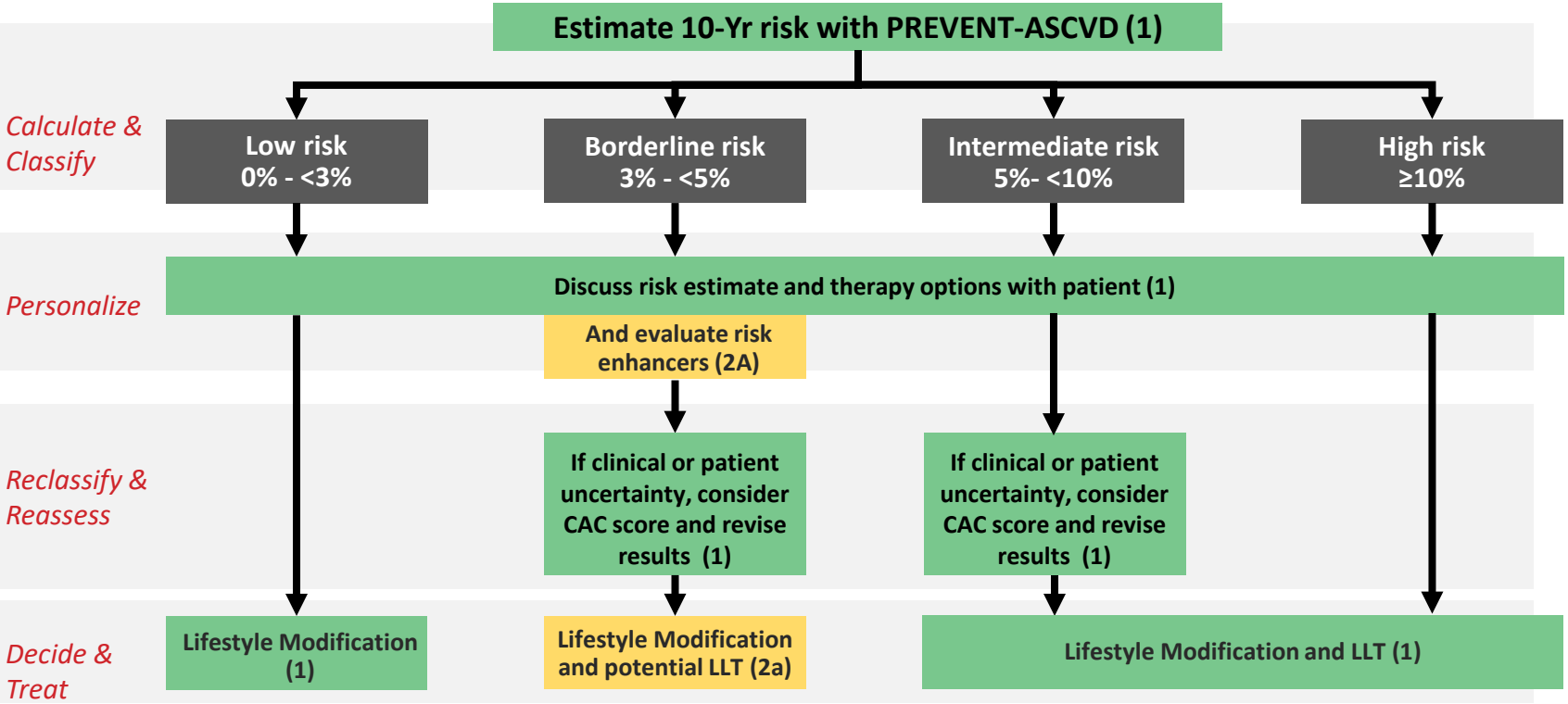
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Approximate Equivalent Ranges of 10-yr ASCVD Risk Estimates

RISK	POOLED COHORT EQUATIONS	PREVENT-ASCVD
Low	<5%	<3%
Borderline	5 - <7.5%	3 to <5%
Intermediate	7.5 - <20%	5 to <10%
High	≥20%	≥10%

Source: Blumenthal RS, et al. J Am Coll Cardiol. 2026;87(19):2624–2757.

Calculate, Personalize, Reclassify (CPR) Framework



Abbreviations: ASCVD indicates atherosclerotic cardiovascular disease; CAC, coronary artery calcium; LLT, lipid-lowering therapy; and PREVENT, Predicting Risk of cardiovascular disease EVENTS.

Blumenthal, R.S., Morris, P.B., et al. 2026 ACC/AHA Guideline on the Management of Dyslipidemia. *Circulation*.

Risk Enhancers

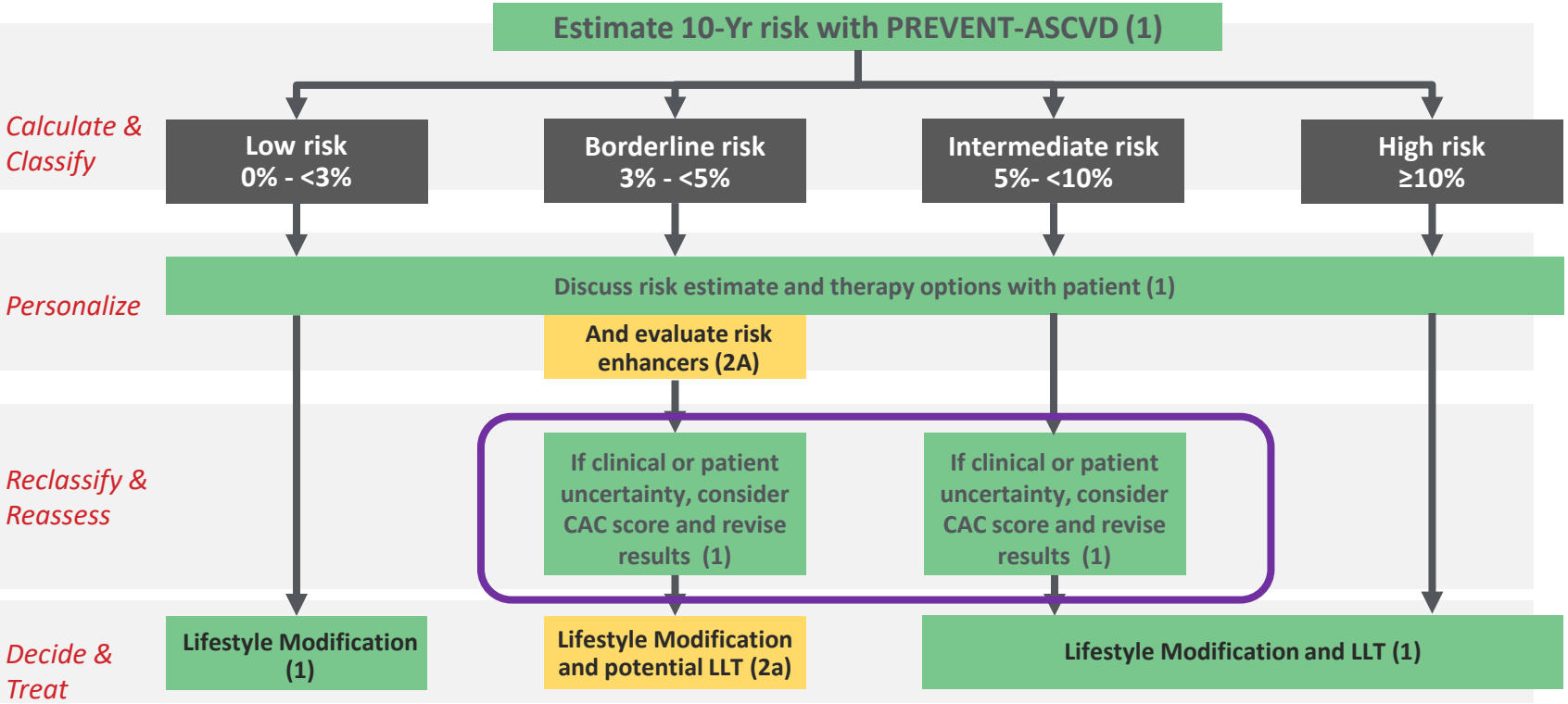
- “In adults without ASCVD with a borderline 10-y ASCVD risk estimate (3% to <5%) by the PREVENT-ASCVD equations, consideration of risk enhancers is reasonable to personalize risk assessment and the potential benefit of initiating LLT as an adjunct to lifestyle management to reduce ASCVD risk”
- Absence of risk enhancers should not be taken to indicate lower ASCVD risk than calculated using PREVENT-ASCVD

Risk Enhancers
History of premature ASCVD in a parent or sibling (onset age <55 y for men, <65 y for women)
Higher risk ancestry (eg, South Asian, Filipino)
High polygenic risk (if measured) (Section 4.2.3.5, “Polygenic Risk Scores”)
Chronic inflammatory diseases (eg, systemic lupus, rheumatoid arthritis, advanced psoriasis, inflammatory arthritis)
Lp(a) ≥125 nmol/L or ≥50 mg/dL
hsCRP ≥2 mg/L on >1 occasion (if measured)
TG persistently ≥175 mg/dL (2 mmol/L) (if nonfasting) and ≥150 mg/dL (1.7 mmol/L) (if fasting)
CKM syndrome
LDL-C persistently ≥160–189 mg/dL (4.1–4.9 mmol/L), non–HDL-C ≥190–219 mg/dL or apoB ≥120 mg/dL*
Reproductive risk markers (premature menopause, preeclampsia, gestational diabetes, gestational hypertension, preterm delivery; Section 4.2.3.4, “Reproductive Risk Marker”)

Source: Blumenthal RS, et al. J Am Coll Cardiol. 2026;87(19):2624–2757.

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American Heart Association.

Blumenthal, R.S., Morris, P.B., et al. 2026 ACC/AHA Guideline on the Management of Dyslipidemia. *Circulation*.

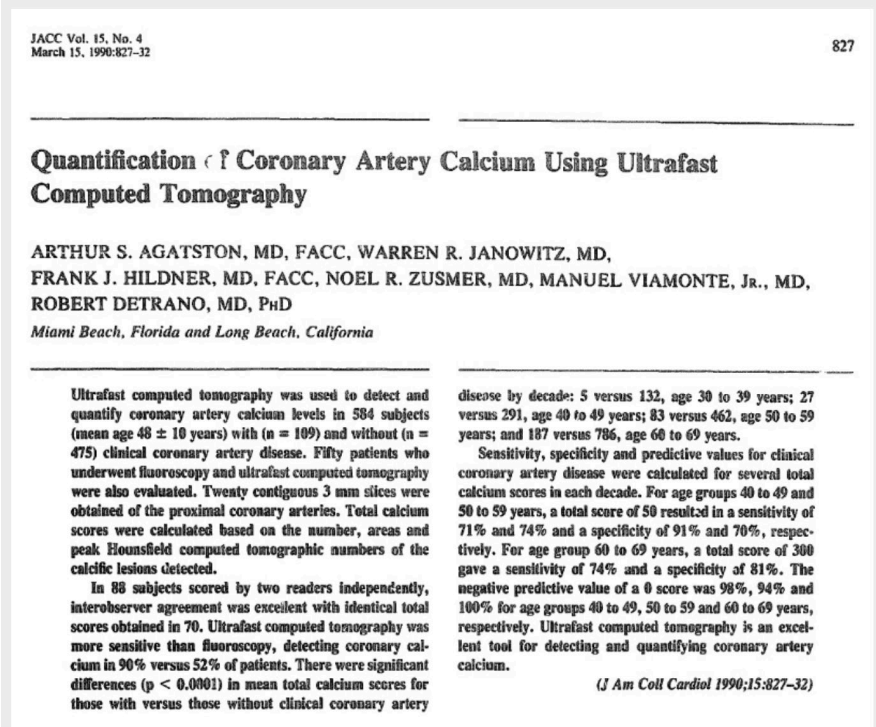
Using Imaging to Reclassify Patients: Coronary Artery Calcium Assessment

- Goal here is assess for subclinical atherosclerosis.
- New guidelines recommend it for patients with borderline/intermediate ASCVD risk though prior guidelines have focused on borderline risk levels.
- Very helpful for patients who are LLT resistant.
- Utilize both score as well as standardize percent
- Remains frequently out of pocket expense.
- Does not:
  - Assess stenosis of coronary arteries
  - Evaluate non-calcified plaque**

CACS	
0	Absent
1-9	Minimal
10-99	Mild
100-299	Moderate
300*-999	Severe
>1000	Extensive

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Agatston Scoring: Reproducible, accurate calculation of CACS



JACC Vol. 15, No. 4
March 15, 1990:827–32

827

Quantification of Coronary Artery Calcium Using Ultrafast Computed Tomography

ARTHUR S. AGATSTON, MD, FACC, WARREN R. JANOWITZ, MD,
FRANK J. HILDNER, MD, FACC, NOEL R. ZUSMER, MD, MANUEL VIAMONTE, JR., MD,
ROBERT DETRANO, MD, PHD

Miami Beach, Florida and Long Beach, California

Ultrafast computed tomography was used to detect and quantify coronary artery calcium levels in 584 subjects (mean age 48 ± 10 years) with (n = 109) and without (n = 475) clinical coronary artery disease. Fifty patients who underwent fluoroscopy and ultrafast computed tomography were also evaluated. Twenty contiguous 3 mm slices were obtained of the proximal coronary arteries. Total calcium scores were calculated based on the number, areas and peak Hounsfield computed tomographic numbers of the calcific lesions detected.

In 88 subjects scored by two readers independently, interobserver agreement was excellent with identical total scores obtained in 70. Ultrafast computed tomography was more sensitive than fluoroscopy, detecting coronary calcium in 90% versus 52% of patients. There were significant differences ($p < 0.0001$) in mean total calcium scores for those with versus those without clinical coronary artery disease by decade: 5 versus 132, age 30 to 39 years; 27 versus 291, age 40 to 49 years; 83 versus 462, age 50 to 59 years; and 187 versus 786, age 60 to 69 years.

Sensitivity, specificity and predictive values for clinical coronary artery disease were calculated for several total calcium scores in each decade. For age groups 40 to 49 and 50 to 59 years, a total score of 50 resulted in a sensitivity of 71% and 74% and a specificity of 91% and 70%, respectively. For age group 60 to 69 years, a total score of 300 gave a sensitivity of 74% and a specificity of 81%. The negative predictive value of a 0 score was 98%, 94% and 100% for age groups 40 to 49, 50 to 59 and 60 to 69 years, respectively. Ultrafast computed tomography is an excellent tool for detecting and quantifying coronary artery calcium.

(J Am Coll Cardiol 1990;15:827–32)

- Cut-off: Threshold of measurement was 130 Hounsfield units with an area > 1mm^2
- Protocol: 20 sequential images obtained, each slice triggered at 80% of the patent's RR interval
  - Region of interest (ROI) identified and created a score based on multiplying the lesion density score and the area.
  - Total CACS calculated by adding all ROI over the 20 slices.

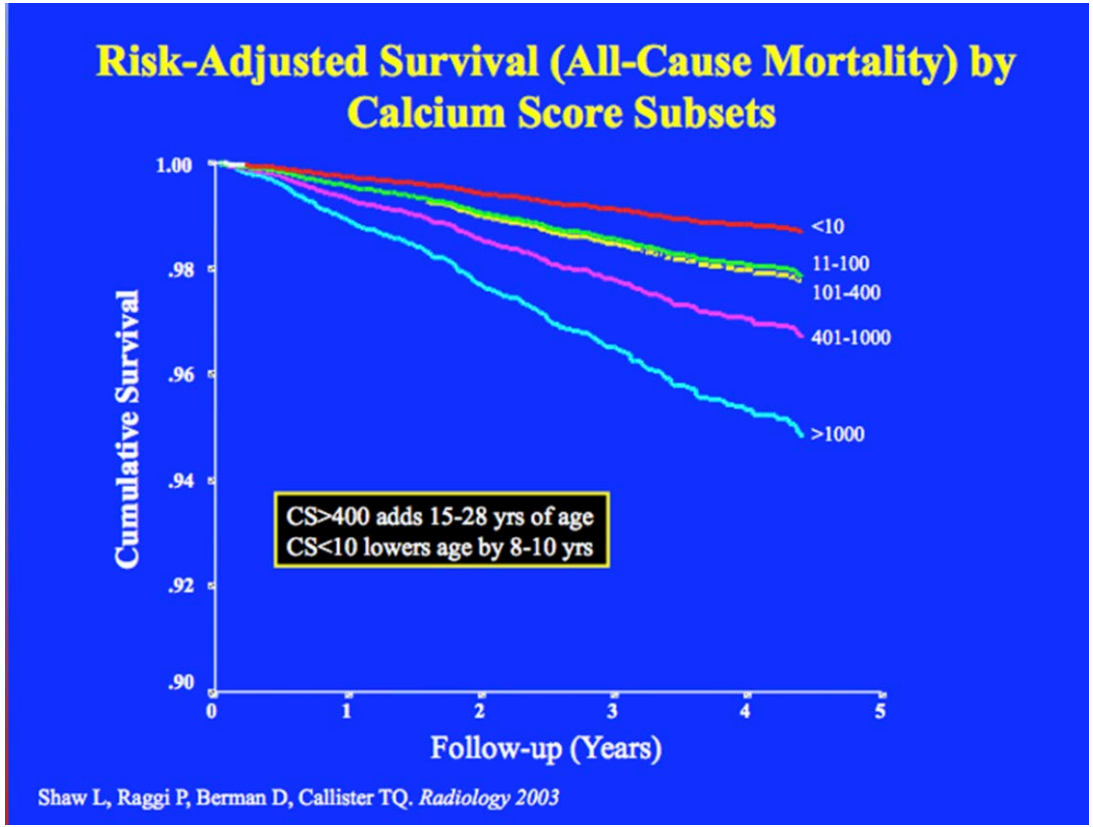
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CAC: “Paint by Numbers” Imaging



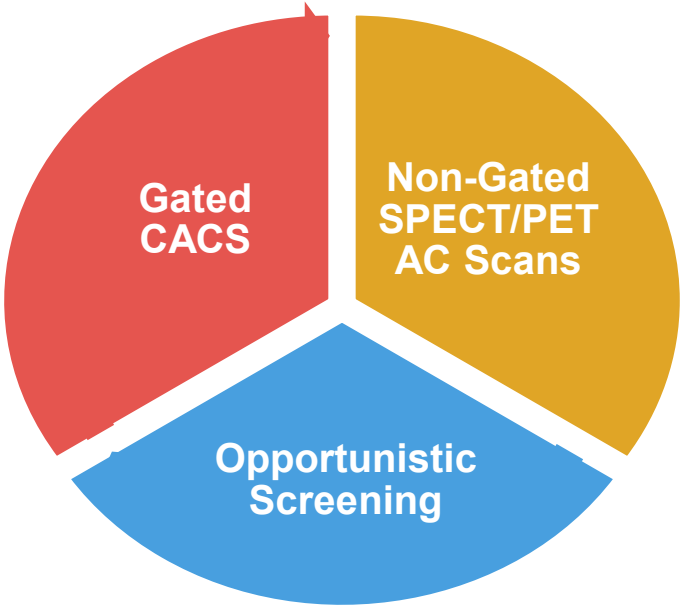
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Higher CACS is associated with worsened survival



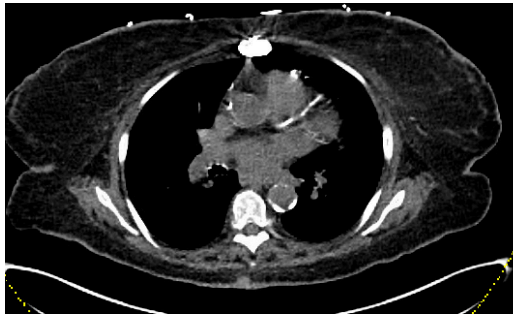
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Pathways to Evaluate Coronary Artery Calcium

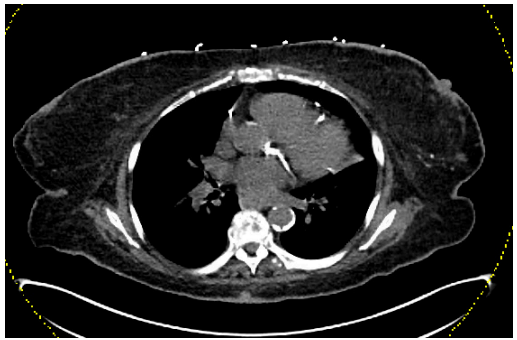


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Non-Gated CT Attenuation Images









N.B. Absence of incidental CAC should not be a justification to withdraw or avoid LLT!

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Lipoprotein Goals are Back

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LDL-C and Non-HDL Goals

Primary Prevention

- Borderline/intermediate PREVENT Risk
  - 10 year risk 3% - < 10%
  - LDL < 100 mg/dL; non-HDL < 130 mg/dL
- High PREVENT Risk
  - 10 year risk > 10%
  - LDL < 70 mg/dL
  - Non-HDL < 100 mg/dL
  - >50% LDL reduction

Clinical ASCVD

- Not very high risk
  - CAC <299; tbc CAC 300—999*
  - LDL < 70; non-HDL < 100
  - > 50% LDL reduction
- Very-High Risk
  - LDL < 55, Non-HDL < 85 mg/L
  - ≧ 2 major ASCVD event: ACS within 12 months; prior MI; ischemic stroke, PAD
  - 1 major ASCVD + ≧ 2 high risk features - >65 yo, PCI/CABG, current smoker/DM, HF, HTN, LDL > 100*

Source: Blumenthal RS, et al. J Am Coll Cardiol. 2026;87(19):2624–2757.

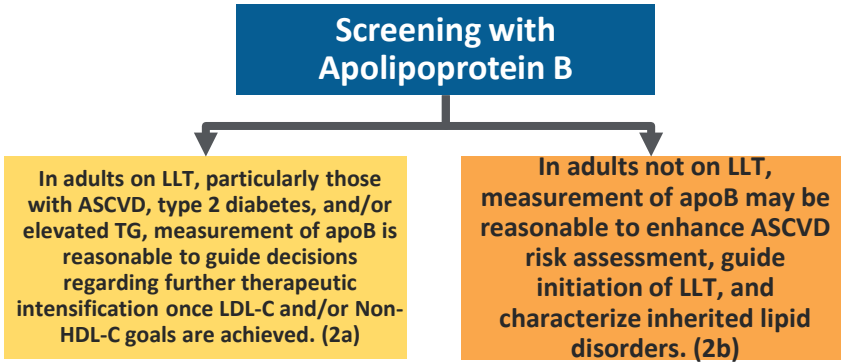
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Apo B and Lp(a)

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Apolipoprotein B

- ApoB predicts ASCVD risk more accurately than LDL.
- Directly quantifies the number of atherogenic lipoproteins
- Identifies residual CVD risk in statin-treated populations.



Particularly for secondary prevention, metabolic syndrome, diabetes, elevated triglyceride

Source: Blumenthal RS, et al. J Am Coll Cardiol. 2026;87(19):2624–2757.

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Lp (a): Screen it once… for now

Screening with Lp(a)

- Measurement of Lp(a) in all adults is recommended at least once for ASCVD risk assessment. (1)
- In those with FH, premature ASCVD, or high Lp(a), cascade testing of 1st-degree relatives is recommended. (1)

Lp (a) in nmol/L	ASCVD relative risk
450	4x
375	3x
250	2x
125	1.4x
75	Reference

Elevated Lp(a) (≥125 nmol/L or ≥50 mg/dL)

- **All Individuals** → Optimal early control of modifiable cardiovascular risk factors
- **Clinical ASCVD** → Achieve LDL-C and non-HDL-C goals on maximally tolerated statin therapy → NOT AT GOAL? → Add PCSK9 mAb

Source: Blumenthal RS, et al. J Am Coll Cardiol. 2026;87(19):2624–2757.

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Per the National Lipid Association

The Lp(a)HORIZON trial is the first of several clinical trials underway to test the impact of lowering Lp(a) on reducing MACE. Other current trials include:

- ACCLAIM-Lp(a) trial by Eli Lilly & Co., evaluating the impact of lepodisiran on the reduction of MACE in adults with elevated Lp(a) who have ASCVD or are at increased ASCVD risk. Lepodisiran is an injectable small interfering RNA (siRNA) therapeutic.
- OCEAN(a)-Outcomes trial by Amgen, evaluating the impact of olpasiran on the risk for coronary heart disease death, myocardial infarction, or urgent coronary revascularization in participants with coronary artery disease and history of myocardial infarction or revascularization and elevated Lp(a). Olpasiran is also an injectable siRNA therapeutic.
- OCEAN(a)-PreEvent trial by Amgen, evaluating whether Lp(a) reduction with olpasiran can prevent a first coronary artery disease event in individuals with elevated Lp(a) at high ASCVD risk.
- MOVE-Lp(a) trial by Eli Lilly & Co., evaluating the impact of muvalaplin on the reduction of MACE in adults with elevated Lp(a) who have experienced a previous ASCVD event or are at high ASCVD risk. Muvalaplin is an oral therapeutic that disrupts assembly of the Lp(a) particle.

Source: https://www.lipid.org

Hot off the Press…

NOVARTIS

Search | Global | en | Menu

Ad hoc announcement pursuant to Art. 53 LR

Basel, September 4, 2026 – Novartis today announced that the pelacarsen phase III Lp(a)HORIZON trial, a pioneering cardiovascular outcomes study, did not meet its primary endpoint of reducing the risk of cardiovascular events, a composite of cardiovascular death, non-fatal myocardial infarction, non-fatal stroke, and urgent coronary revascularization requiring hospitalization, compared to placebo[1]. Lower lipoprotein (a), Lp(a), levels were achieved with pelacarsen in this study population, which was receiving guideline-directed treatments, including lipid lowering and antihypertensive therapies[1,2]. The findings provide important new evidence on the relationship between Lp(a) lowering and cardiovascular outcomes. Elevated Lp(a) is an inherited cardiovascular risk factor affecting approximately one in five people worldwide with no approved targeted treatment[1,3].

"Lp(a)HORIZON was a pioneering cardiovascular outcomes trial designed to answer one of the most important unanswered questions in cardiovascular medicine by evaluating whether lowering Lp(a) can reduce residual cardiovascular risk beyond optimized, guideline-directed care, when other major cardiovascular risk factors are already being managed," said Shreeram Aradhye, President, Development and Chief Medical Officer, Novartis. "Although lower Lp(a) levels were observed with pelacarsen, the findings did not demonstrate that this translated into reduced cardiovascular risk in the overall study population. These are not the results we hoped for, but they provide important evidence that advances scientific understanding of the relationship between Lp(a) lowering and cardiovascular outcomes and may help inform future approaches to cardiovascular risk management. We thank the more than 8,000 trial participants and investigators who made this study possible, and remain committed to driving cardiovascular innovation."

The Lp(a)HORIZON trial data will be presented at an upcoming medical congress.

Medical News in Brief

Lipoprotein(a)-Lowering Agent Fails to Reduce Cardiovascular Events

The experimental drug pelacarsen did not reduce the risk of adverse cardiovascular events in a phase 3 study, despite lowering lipoprotein(a) (Lp[a]) levels, the manufacturer announced.

The 7-year Lp(a)HORIZON trial included about 8300 people with high Lp(a) levels and preexisting heart disease. Participants who received monthly injections of pelacarsen, an investigational antisense oligonucleotide, lowered their Lp(a) levels compared with participants who received a placebo, but the drug did not reduce the risk of cardiovascular events, including urgent coronary revascularization requiring hospitalization, nonfatal myocardial infarction, nonfatal stroke, and cardiovascular death.

Elevated Lp(a) is an inherited cardiovascular risk factor with no approved targeted treatment. It was thought that lowering Lp(a) would reduce residual cardiovascular risk beyond guideline-directed care, the manufacturer said in a press release. Phase 3 clinical trials are ongoing for 2 other Lp(a)-lowering agents, lepodisiran and olpasiran, with a different mechanism of action from pelacarsen. **– Samantha Anderer**

Published Online: September 25, 2026. doi:10.1001/jama.2026.14725

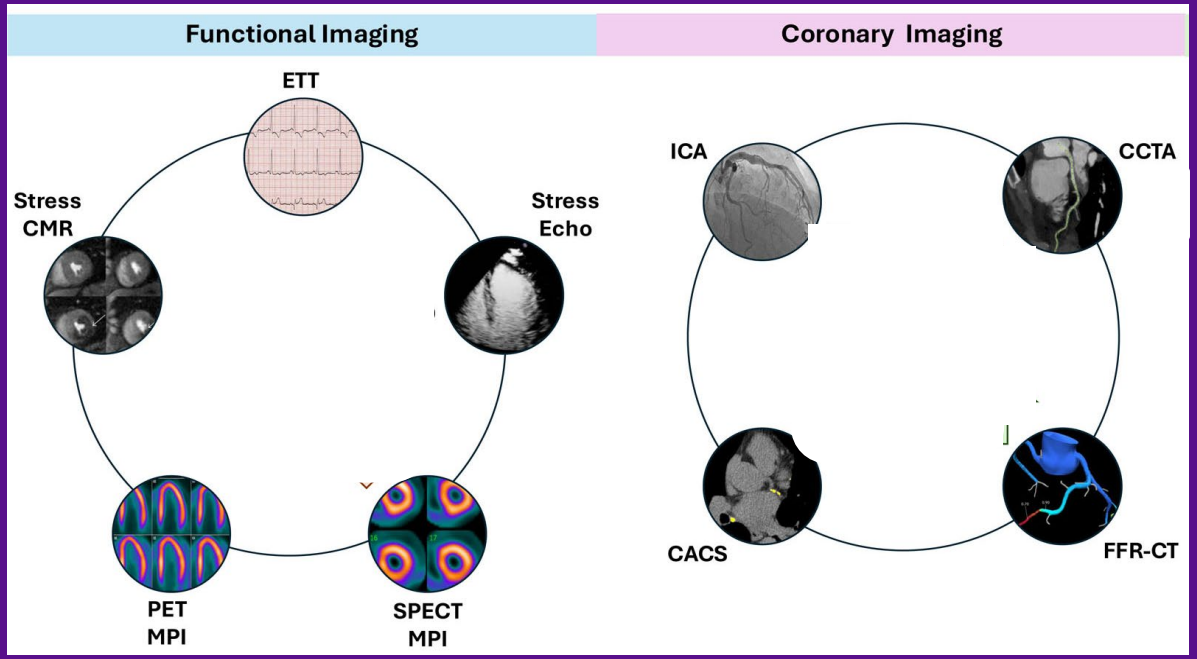
Note: Source references are available through embedded hyperlinks in the article text online.

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Take Home Points

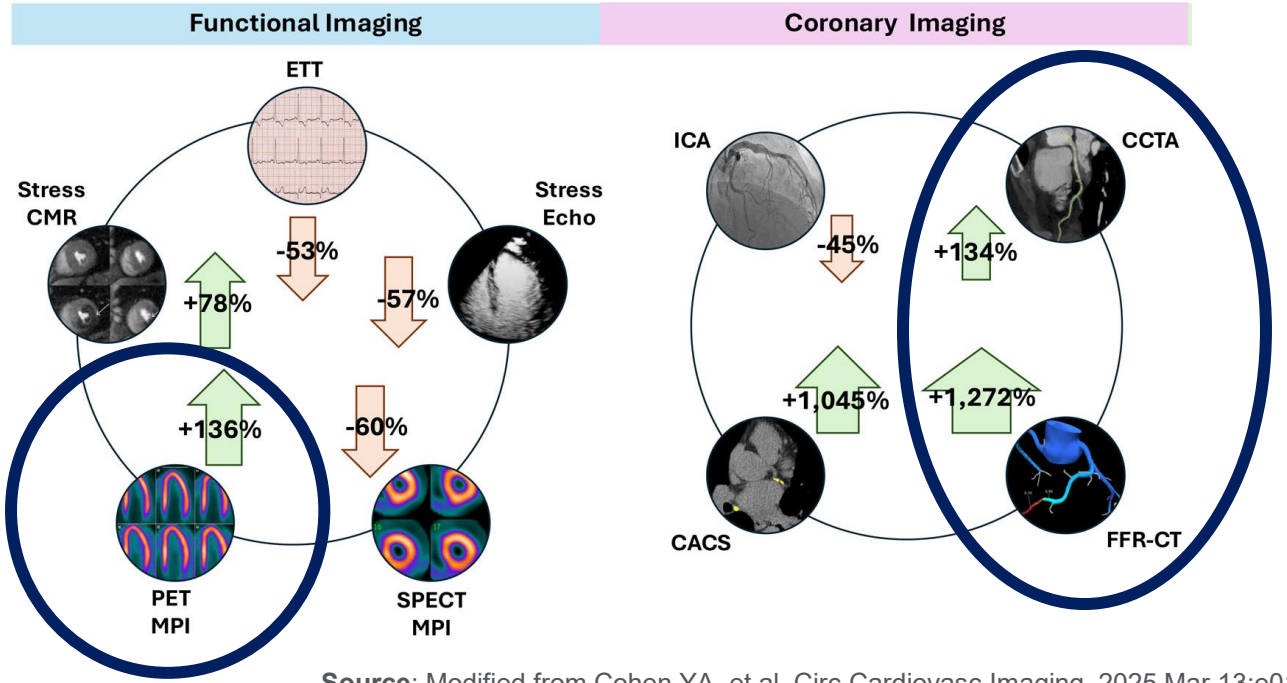
- Use PREVENT-ASCVD to initially predict risk and treatment guidance.
- Explore the CPR framework to calculate, personalize and recalibrate risk.
- ApoB can be helpful to guide reduction further beyond LDL.
- Lp(a) should be checked once for evaluation of genetic risk.
- LDL and non-HDL targets are back.
- Jury is still out on whether there is risk reduction by treating with Lp(a) reducing agents.

Chest Pain Evaluation Utilizing Advances in Cardiac Imaging



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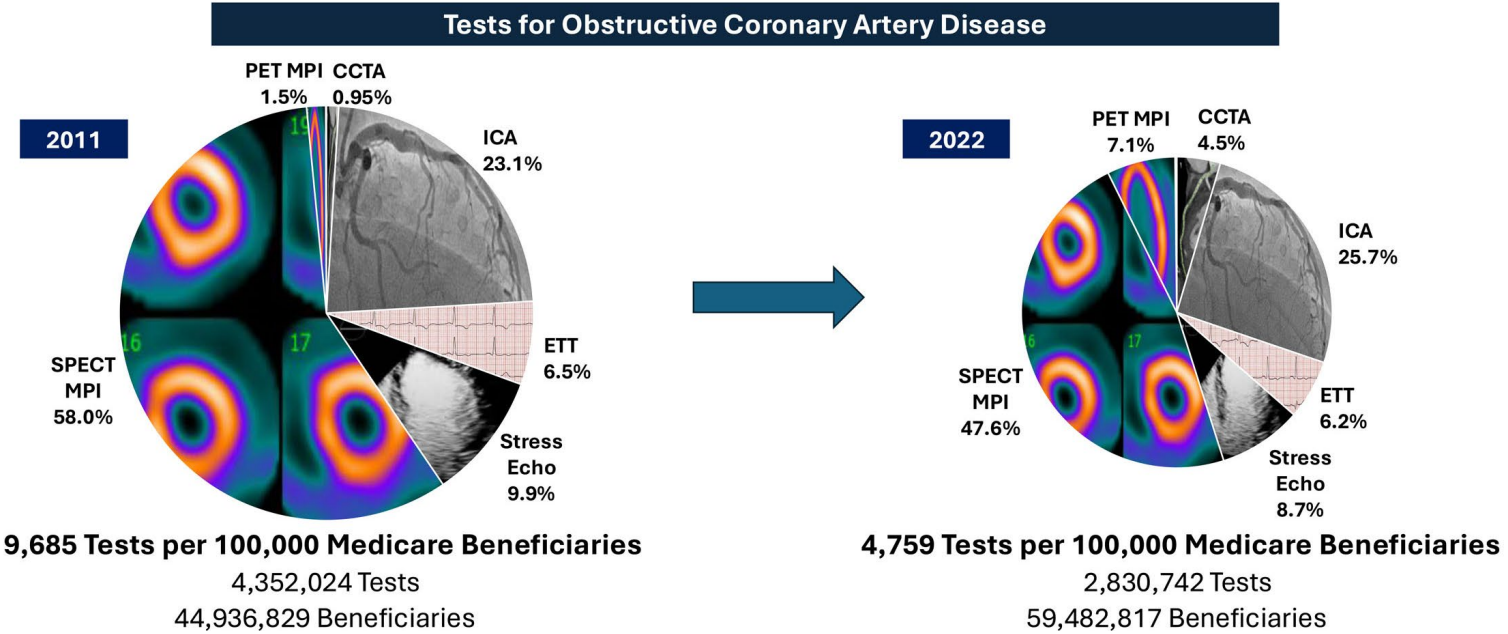
Changes in Medicare Part B Cardiac Testing 2011-2022



Source: Modified from Cohen YA, et al. Circ Cardiovasc Imaging. 2025 Mar 13:e017567.

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Changes in Medicare Part B Cardiac Testing 2011-2022



Source : Modified from Cohen YA, et al. Circ Cardiovasc Imaging. 2025 Mar 13:e017567.

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Case #C1:

- 62 year old man with history of hypertension, hyperlipidemia, OSA, who presents in the outpatient setting of evaluation of chest pain.
  - He notes several episodes of rest chest pain that resolved after 15 minutes.
  - A recent ER evaluation with EKG and labs was non-focal.
  - Baseline EKG: Normal sinus rhythm without ST segment deviation

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What CV imaging would you recommend next for this patient?

1. None
2. Coronary Artery Calcium Score
3. Non-Imaging Treadmill Stress Test
4. Exercise Stress Echocardiogram
5. Pharmacologic Stress Echocardiogram
6. Exercise Stress SPECT
7. Pharmacologic Stress SPECT
8. Pharmacologic Stress PET
9. Pharmacologic Stress MRI
10. Coronary CTA

What is the chest pain patient’s risk?

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How do we predict pretest probability of physiologically significant CAD in symptomatic patients?

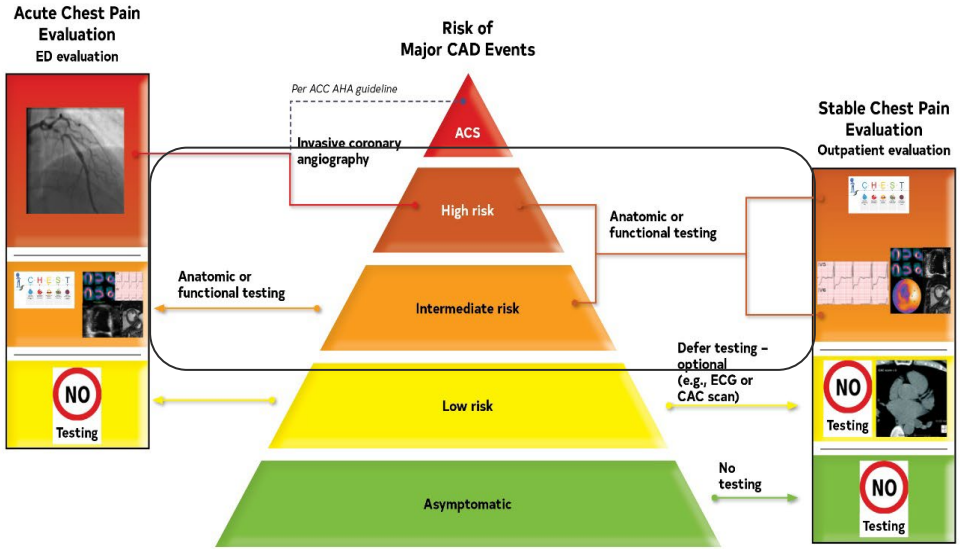
Age, y	Chest Pain		Dyspnea	
	Men	Women	Men	Women
30–39	≤4	≤5	0	3
40–49	≤22	≤10	12	3
50–59	≤32	≤13	20	9
60–69	≤44	≤16	27	14
70+	≤52	≤27	32	12



Source: Gulati M, et al. JACC. 2021 Nov, 78 (22) e187-e285.

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Intermediate Risk Patients



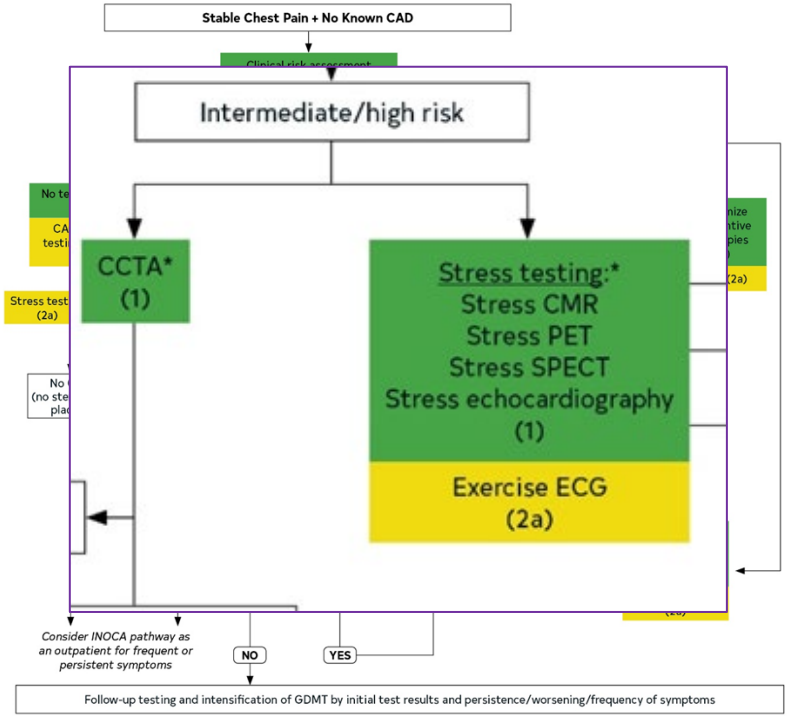
Options include:

- ✓ Stress Echocardiography
- ✓ Nuclear Stress (SPECT)
- ✓ Nuclear Stress (PET)
- ✓ Stress Cardiac MR
- ✓ Coronary CTA
- ✓ Invasive Coronary Angiography

Source: Gulati M, et al. JACC. 2021 Nov, 78 (22) e187-e285.

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ACC/AHA Chest Pain Guideline



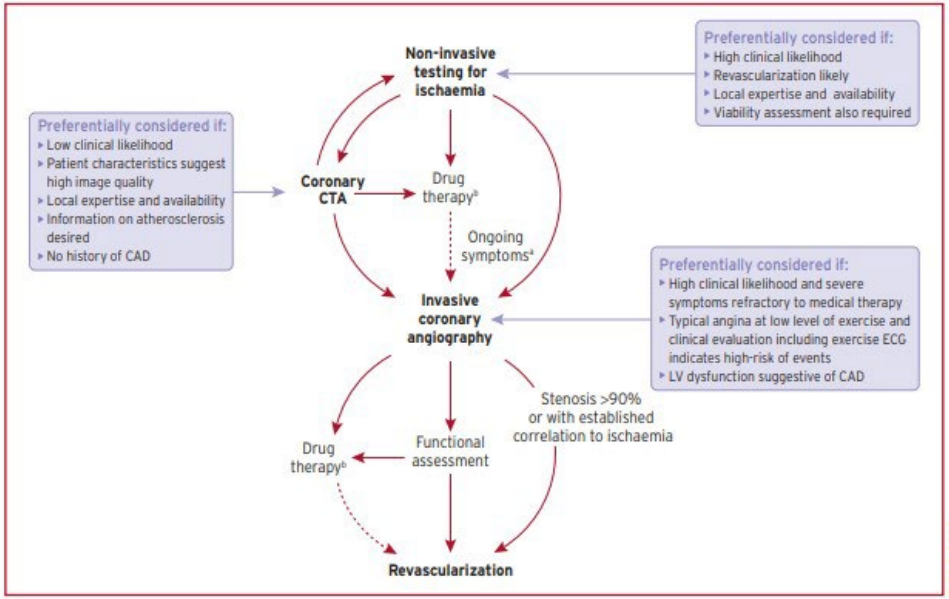
- **KEY POINT!** Class I Indication in intermediate risk patients with Stable Chest Pain & No Known CAD:
  - Stress SPECT
  - Stress PET
  - Stress Echocardiography
  - Stress CMR
  - CCTA

Source: Gulati M, et al. JACC. 2021 Nov, 78 (22) e187-e285.

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Factors to Break the Equipoise: Functional vs. Anatomic

ESC Guidance



US Guidance

Pretest likelihood of CAD	Low	→ No testing necessary	→ Option for CAC for ASCVD risk stratification		
	Intermediate-high	→ Younger patient (<65 y of age)	OR	Less obstructive CAD suspected	→ CCTA favored
	Intermediate-high	→ Older patient (≥65 y of age)	OR	More obstructive CAD suspected	→ Stress testing favored

	Favors use of CCTA	Favors use of stress imaging
Goal	• Rule out obstructive CAD • Detect nonobstructive CAD	• Ischemia-guided management
Availability and expertise	• High-quality imaging and expert interpretation routinely available	• High-quality imaging and expert interpretation routinely available
Likelihood of obstructive CAD	• Age <65 y	• Age ≥65 y
Prior test results	• Prior functional study inconclusive	• Prior CCTA inconclusive
Other compelling indications	• Anomalous coronary arteries • Require evaluation of aorta or pulmonary arteries	• Suspect scar (especially if PET or stress CMR available) • Suspect coronary microvascular dysfunction (when PET or CMR available)

Source: Knuuti J, et al. European Heart Journal (2020) 41, 407-477.
Gulati M, et al. JACC. 2021 Nov, 78 (22) e187-e285.

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Back to our Case #C1:

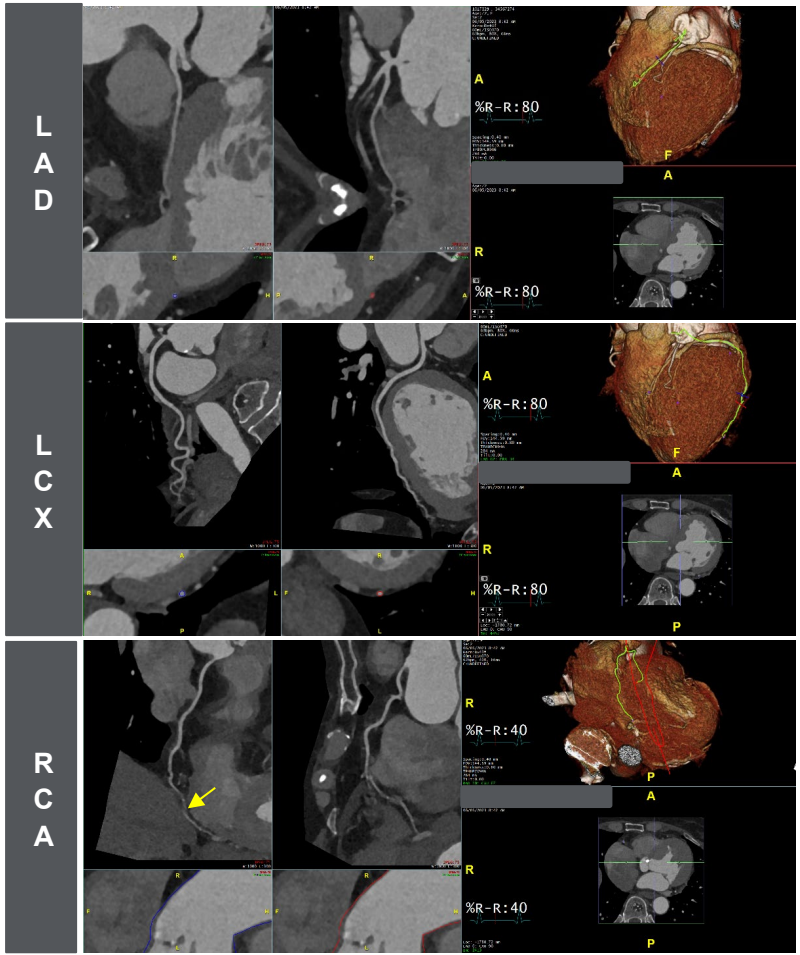
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  - Baseline EKG: Normal sinus rhythm without ST segment deviation

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Coronary CT Angiography

- Anatomic evaluation for obstructive CAD.
- Identifies plaque as well as stenosis.
- High negative predictive value
- Caveats:
  - Radiation exposure
  - Requires a slow heart rate
  - Difficult to evaluate stenosis with heavy calcium burden
  - Requires contrast load

Normal coronary arteries

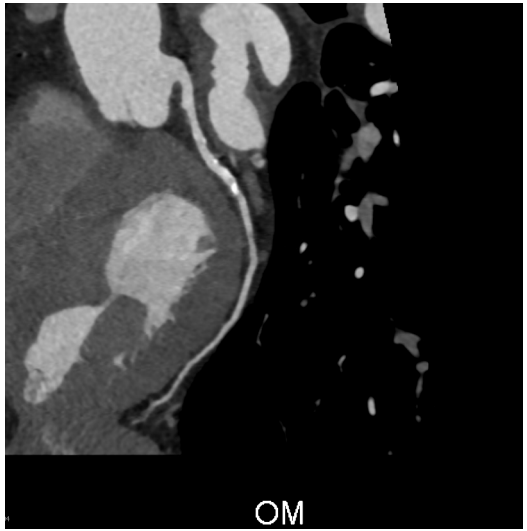


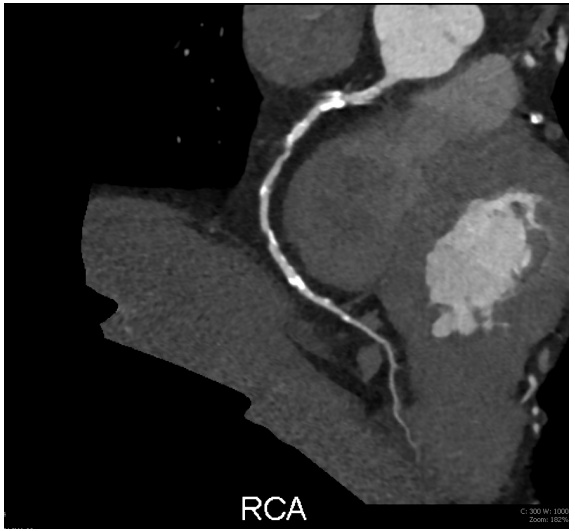
Courtesy of Muhamed Saric, MD

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Case #C1



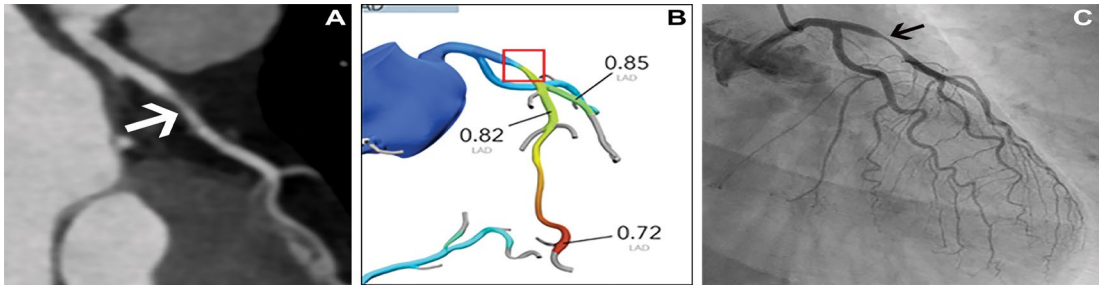




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FFR-CT

- Physiologic simulation modeling coronary blood flow from CTA.
- Determines lesion specific FFR by measuring 2 cm distal to a stenosis.
- > 0.8 is normal (no functional significance of the stenotic lesion)



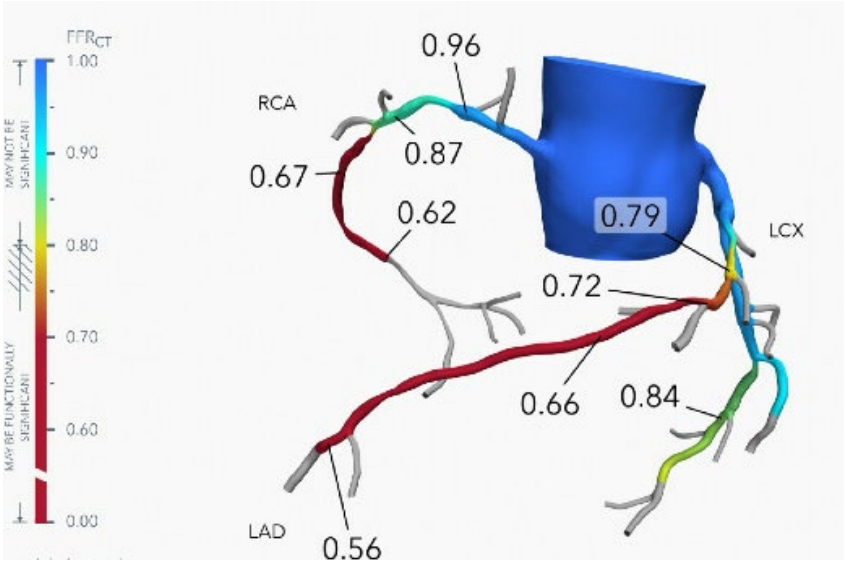
Source: Rajiah P, et al. RadioGraphics 2022; 42:340–358

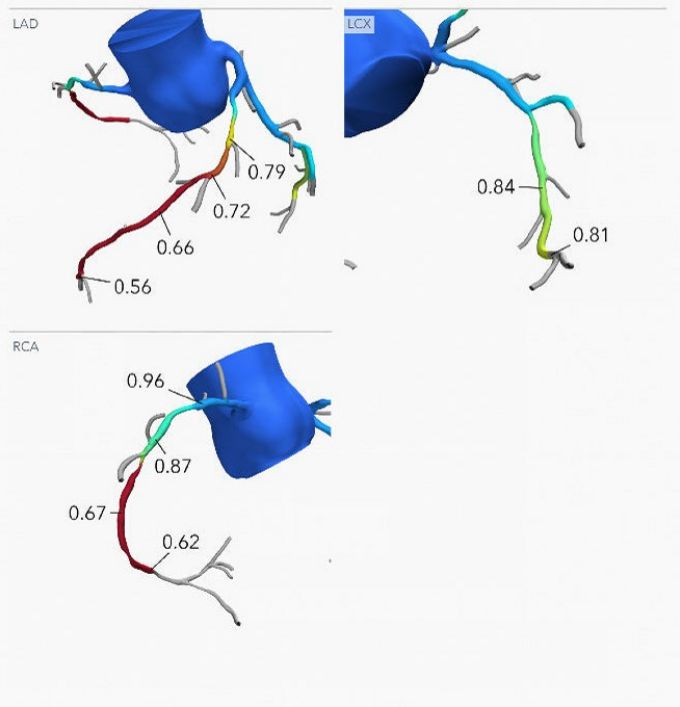
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FFR-CT

- Post-processing of data gathered during a coronary CTA to assess functional significance of coronary artery stenosis.
- Computer software simulates blood flow through the coronary arteries and calculates the ratio of blood flow in preceding normal coronary arteries and stenotic regions.
- AHA/ACC Chest Pain Guideline – CT-FFR can be considered when anatomic stenosis is noted between 40% and 90%.
- Looking for a discrete drop along the coronary vessel instead of slow/steady decrease which could represent diffuse vessel disease

Case #C1 FFR-CT





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Case #C2: 60 year old man with chest pain undergoes CCTA and found to have 65% proximal LAD stenosis.

CT-FFR 0.88 distal to the lesion

- Can strongly consider medical therapy with close clinical follow up.

CT-FFR 0.72 distal to the lesion

- Consider invasive evaluation with coronary angiography
- Note: Anginal presence and clinical story continues to play a significant role in this decision

Limitations and Pitfalls of CT-FFR

- Motion, misregistration, poor contrast and severe calcification impair accuracy
- Can see decreased specificity in stent and bypass anatomy.
- CT-FFR does not allow direct measurement of microvascular function.

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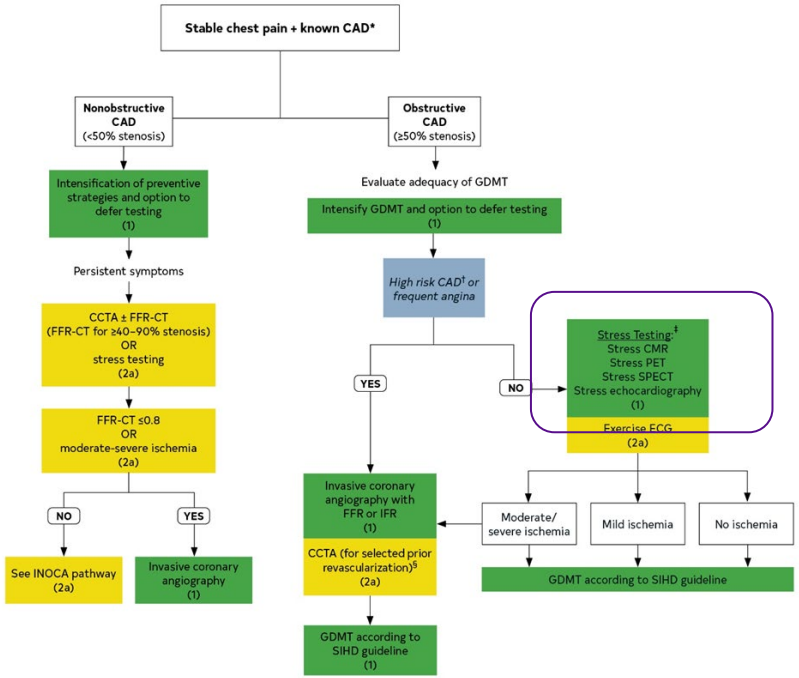
Case #C3:

- 67 year old woman h/o CAD, maximized GDMT, presents to clinic with recent chest pain evaluated in the ED.
  - Prior pLCx PCI
  - Prior pRCA PCI
  - Distant pLAD and mLAD PCI
- Baseline EKG: Normal sinus rhythm

Case prepared by Brian Hsia, MD

Division/Department
NYU Langone Health

Stable Chest Pain & Known CAD



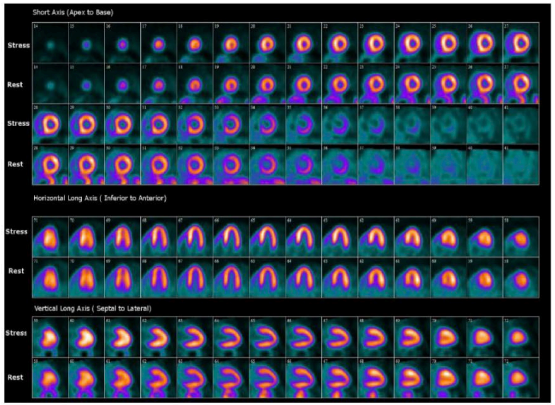
- **KEY POINTS!**
  - In patients with known obstructive CAD, and no high risk CAD nor frequent angina, intensification of GDMT +/- Stress MPI is recommended.
  - In patients with non-obstructive CAD and persistent symptoms, initial equipoise between CCTA and stress MPI.

Source: Gulati M, et al. JACC. 2021 Nov, 78 (22) e187-e285.

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Comprehensive Cardiac PET/CT Stress Test

A



B

	Rest (ml/g/min)	Stress (ml/g/min)	Reserve Stress/Rest
LAD	1.20	4.13	3.44
LCx	1.12	3.40	3.06
RCA	1.13	4.12	3.60
Global	1.16	3.94	3.38

C



D - Rest EF: 62%; Post-Vasodilator: EF: 66%

- ✓ High diagnostic accuracy
- ✓ Consistent high-quality images
- ✓ Low radiation exposure
- ✓ Short acquisition protocols
- ✓ Quantification of myocardial blood flow
- ✓ Strong prognostic power

Source: Modified from Bateman, TM. et al. J Nucl Cardiol. 2021 Apr;28(2):768-787.

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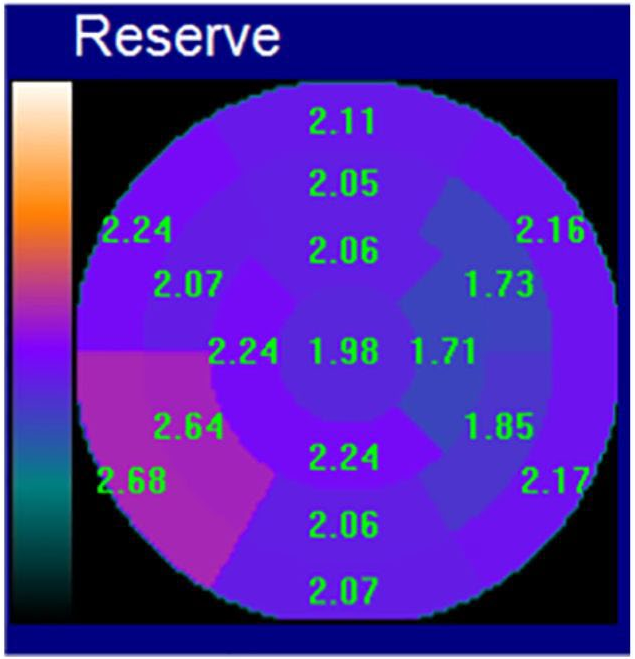
Clinical Value of Myocardial Blood Flow Reserve

No Known CAD
High negative predictive value in combination with normal perfusion
Confirm an abnormality is CAD
Predict more severe disease: e.g., 1-vessel abnormal perfusion, 2-3-vessel abnormal MBFR
Confirm single vessel disease: 1 vessel abnormal perfusion, 1 vessel abnormal MBFR Normal perfusion, abnormal MBFR: identify balanced CAD, microvascular disease
Identify non-responder: all patients
Known CAD
Often abnormal after CABG, CAD history, myocardial infarction
Cardiomyopathy less useful but if normal, helps exclude CAD
Renal failure patients generally abnormal
Post PCI may be abnormal, but most useful if pre-PCI data available
Identify non-responder: all patients

Modified from Bateman TM, et al. J Nucl Med. 2021 Nov;62(11):1599-1615.

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Vessels and Segments and Flow

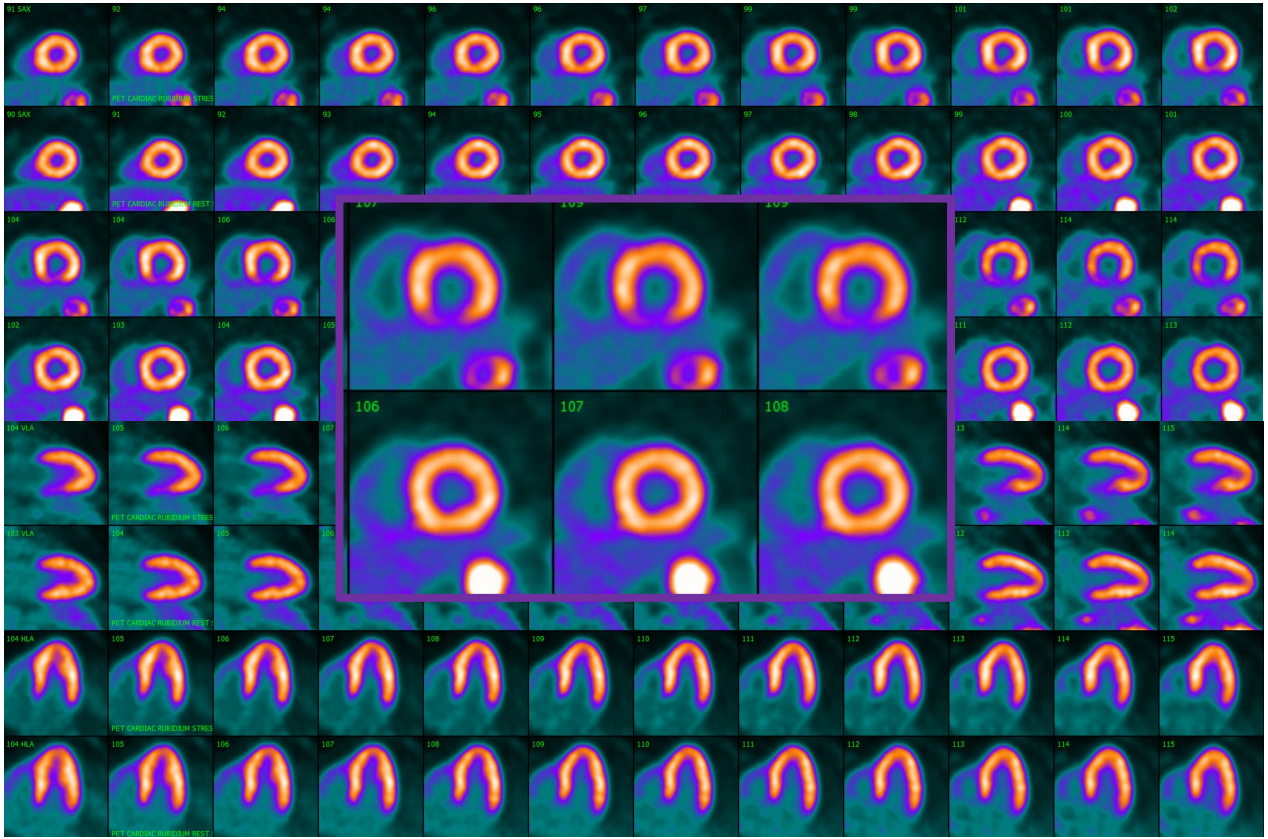


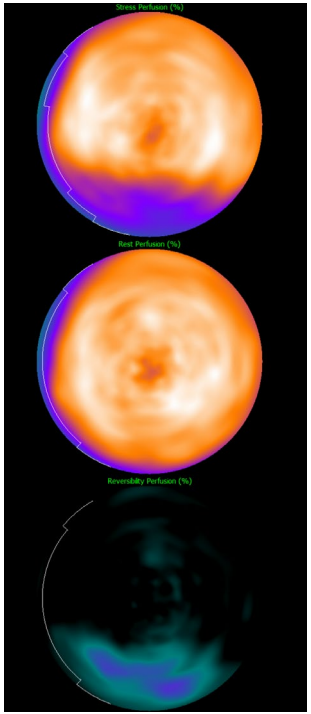
	Rest (mL/g/min)	Stress (mL/g/min)	Reserve (Stress/Rest)
LAD	0.81	1.70	2.10
LCx	0.78	1.65	2.11
RCA	0.79	1.95	2.46
Global	0.80	1.72	2.15

Bateman TM, et al. J Nucl Med. 2021 Nov;62(11):1599-1615.

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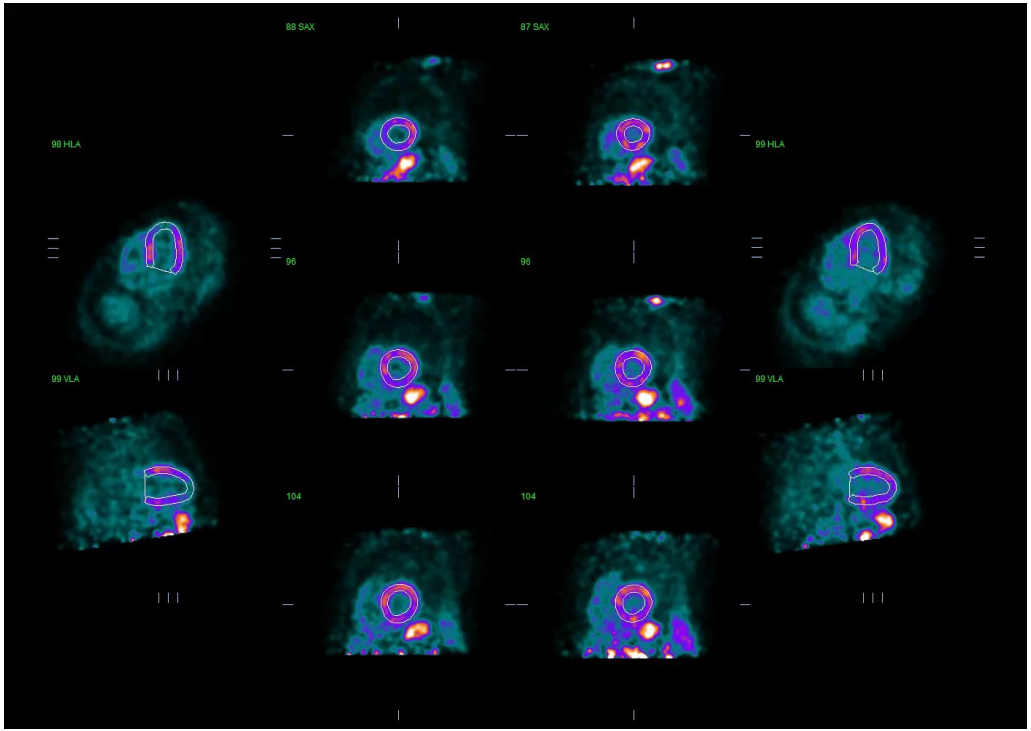
Case #C3: PET MPI





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Case #C3 Gating



Study	PET CT RUBIDIUM MYOCARDIAL STRESS PHARMACOLOGIC
Dataset	CARDIAC GATED STRESS AC Images
Status	QC=6.10, IR=0.39
Volume	19ml [10]
EDV	77ml [16]
ESV	17ml [8]
SV	60ml
EF	78%
Shape	0.67 [SI ED], 0.40 [SI ES], 0.87 [Ecc 10]

Study	PET CT RUBIDIUM MYOCARDIAL STRESS PHARMACOLOGIC
Dataset	CARDIAC GATED REST AC Images
Status	QC=8.72, IR=0.40
Volume	35ml [10]
EDV	65ml [16]
ESV	18ml [7]
SV	47ml
EF	73%
Shape	0.61 [SI ED], 0.39 [SI ES], 0.84 [Ecc 10]

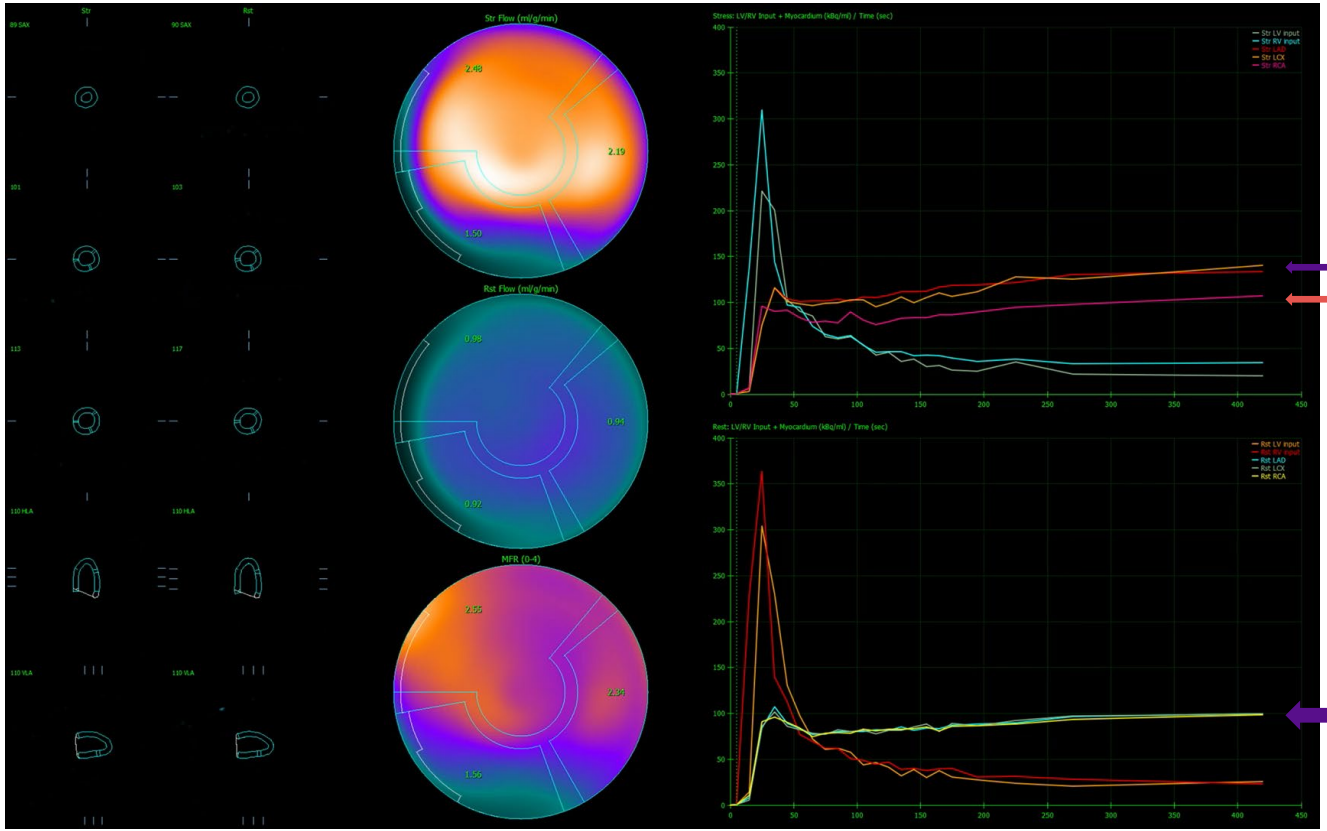
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Case #C3 Attenuation CT



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Case #C3 Kinetic flow analysis



	Stress Flow	Rest Flow	Myocardial Flow Reserve
LAD	2.48	0.98	2.55
LCx	2.19	0.94	2.34
RCA	**1.50**	0.92	1.56
Total	2.24	0.97	2.30

LAD, LCx Stress Flow

RCA Stress Flow

LAD, LCx, RCA Resting Flow

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What is likely to be found on cardiac cath?

a) Obstructive LAD
b) Obstructive RCA
c) Obstructive LCx
d) Multivessel obstructive CAD
e) No obstructive CAD

Case #C4:

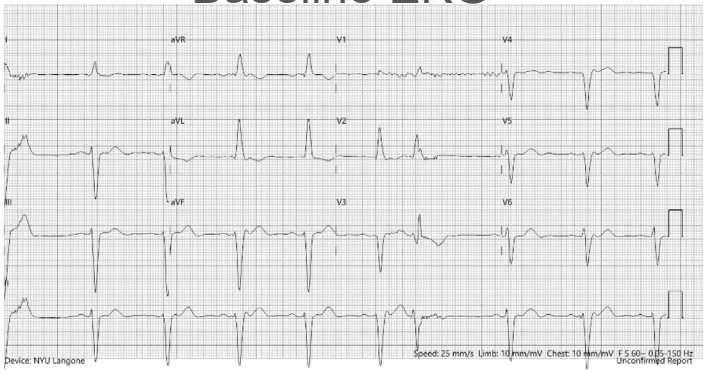
- 81 y.o. woman with history of diabetes, ESRD on HD, CAD s/p PCI to RCA (10 years prior), presents with chest pain symptoms. Sent for a rubidium PET/CT stress test.

Regadenoson Hemodynamics

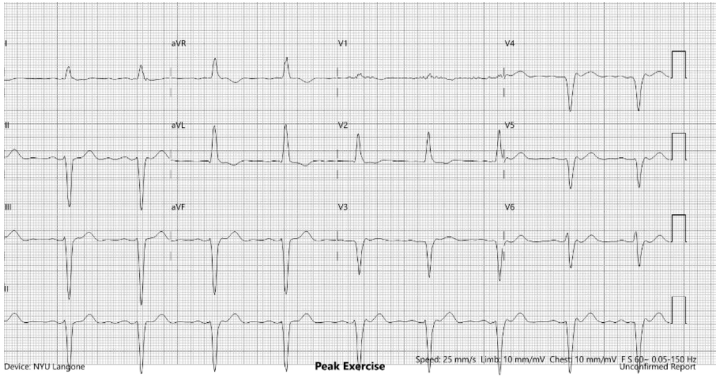
Regadenoson Table

TIME	REGADENOSON INJECTION	HR (BPM)	SYSTOLIC BP(MMHG)	DIASTOLIC BP(MMHG)	O2 SAT %
Pre-Injection	Pre-Injection	58	101	36	100
00:00	Injection Time	56	92	35	100
00:30	Post Injection	60	98	74	100
01:00	Post Injection	62	82	42	100
02:00	Post Injection	63	86	43	100
03:00	Post Injection	63	86	46	100
04:00	Post Injection	63	86	46	100
05:00	Post Injection	62	87	49	100
06:00	Post Injection	63	91	57	100

Baseline EKG

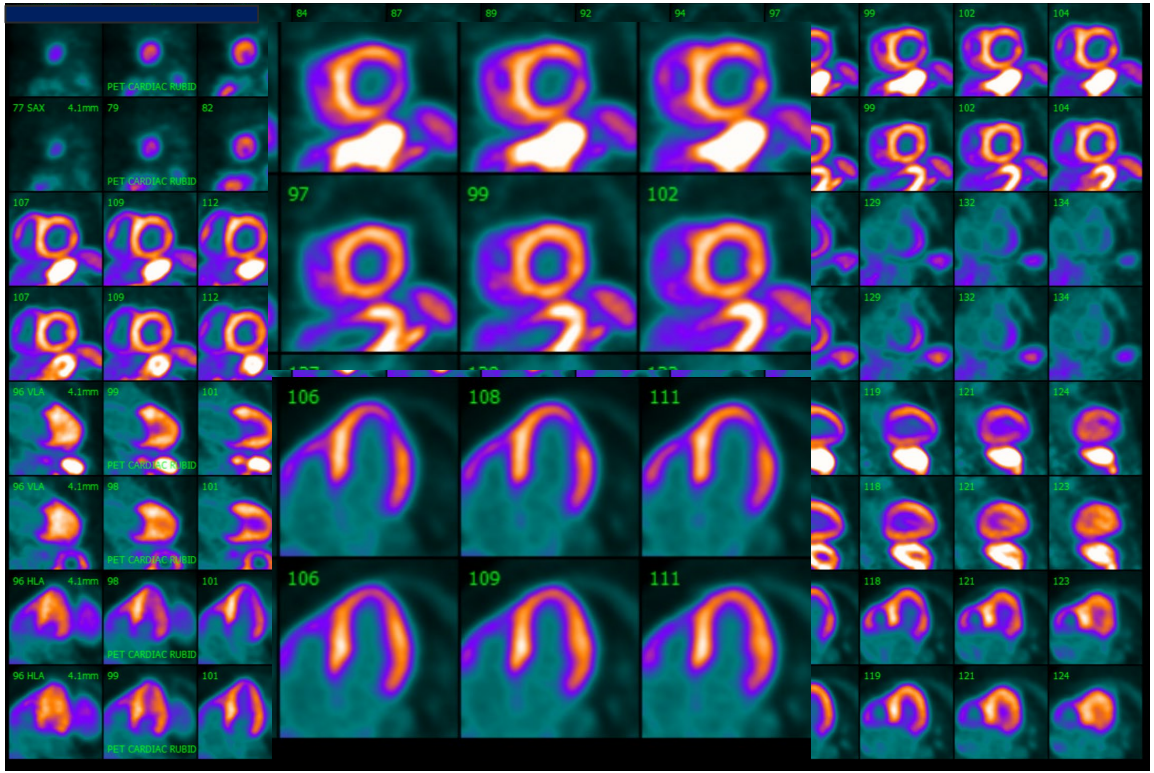


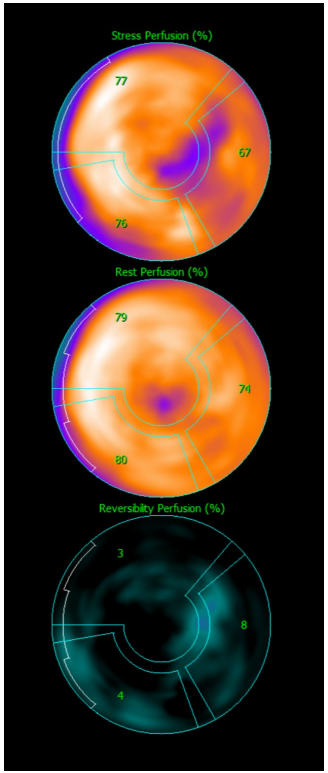
Post-Stress EKG



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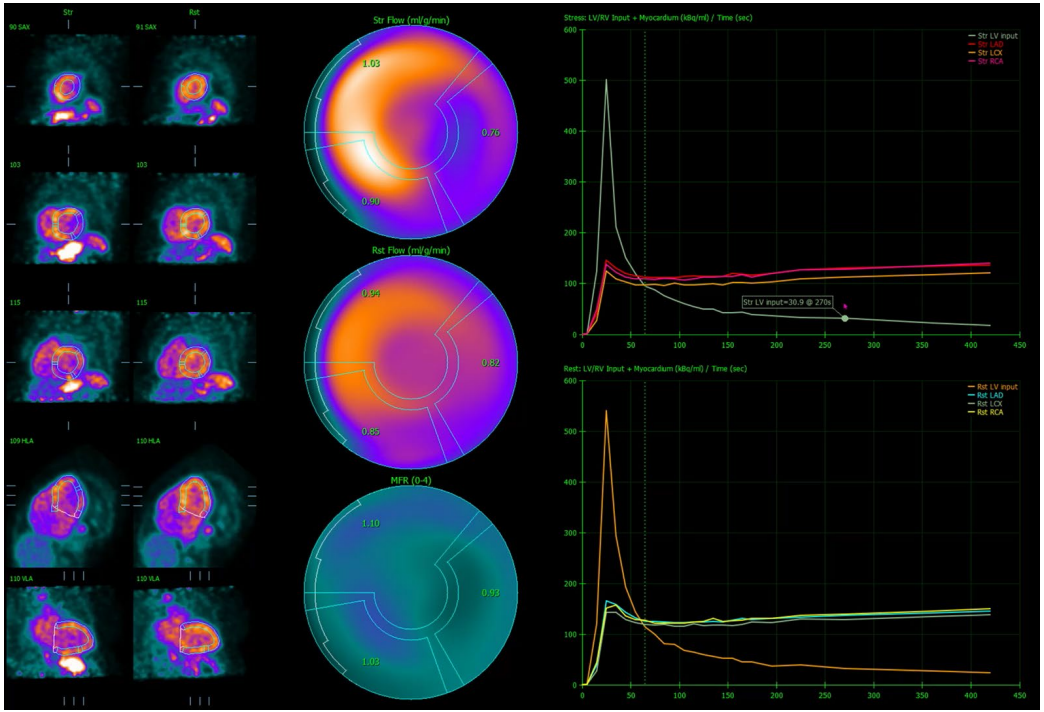
Case #C4: Rubidium PET Rest/Stress Imaging





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Case #C4 Myocardial Blood Flow Assessment



	Stress Flow	Rest Flow	Myocardial Flow Reserve	Stress Spillover	Rest Spillover
LAD	1.03	0.94	1.10	0.31	0.31
LCX	0.76	0.82	0.93	0.30	0.30
RCA	0.90	0.85	1.03	0.30	0.31
TOT	0.94	0.89	1.04	0.31	0.31

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Note markedly abnormal myocardial blood flow

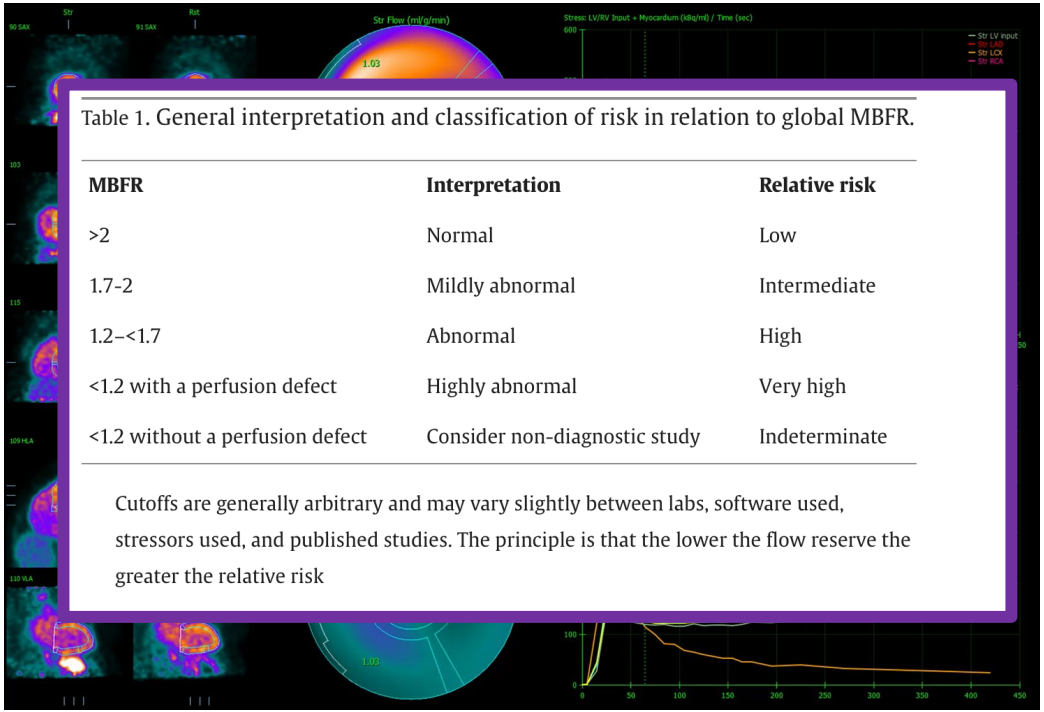


Table 1. General interpretation and classification of risk in relation to global MBFR.

MBFR	Interpretation	Relative risk
>2	Normal	Low
1.7-2	Mildly abnormal	Intermediate
1.2–<1.7	Abnormal	High
<1.2 with a perfusion defect	Highly abnormal	Very high
<1.2 without a perfusion defect	Consider non-diagnostic study	Indeterminate

Cutoffs are generally arbitrary and may vary slightly between labs, software used, stressors used, and published studies. The principle is that the lower the flow reserve the greater the relative risk

	Stress Flow	Rest Flow	Myocardial Flow Reserve	tress illover	Rest Spillover
LAD	1.03	0.94	1.10	.31	0.31
LCX	0.76	0.82	0.93	.30	0.30
RCA	0.90	0.85	1.03	.30	0.31
TOT	0.94	0.89	1.04	.31	0.31

Source: Bateman, TM. et al. J Nucl Cardiol. 2021 Apr;28(2):768-787.

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MBF Assessment continues to have increased value in nuclear cardiology

1. Improved diagnosis of epicardial CAD
2. Improved assessment of extent and severity of epicardial CAD
3. Improved risk stratification
4. Selection of patients for coronary interventions and/ or medical therapy
5. Diagnosis of coronary microvascular disease
6. Confirmation of adequate pharmacologic stress in patients

Source: Bateman, TM. et al. J Nucl Cardiol. 2021 Apr;28(2):768-787.

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Source: Bateman, TM. et al. J Nucl Cardiol. 2021 Apr;28(2):768-787.

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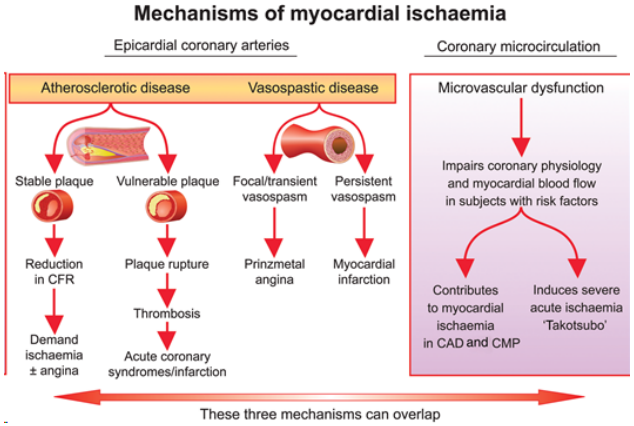
^{18}F Flurpiridaz as a PET Perfusion Tracer

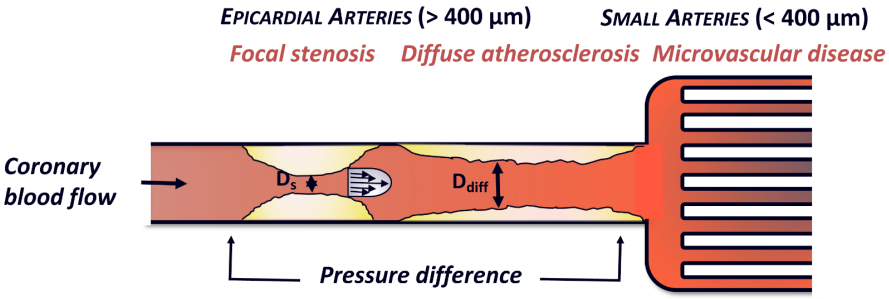
- ✓ **Longer half-life** of ^{18}F (108 minutes) compared to other PET stress tracers.
- ✓ **Unit dose** delivery from a radiopharmacy (via cyclotron)
- ✓ The longer half-life of ^{18}F would facilitate the use of **treadmill exercise** as an alternative to pharmacologic stress for PET MPI (though at a loss of blood flow assessment).

Source: Calnon, D.A. *J. Nucl. Cardiol.* **26**, 2031–2033 (2019)

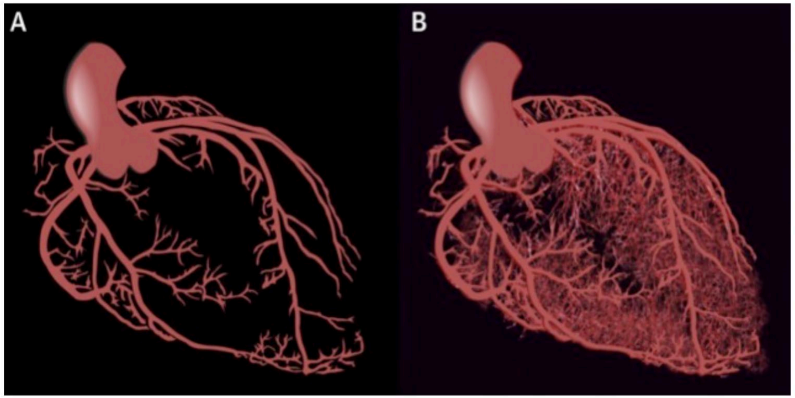
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Evaluating the Entire Coronary Artery Tree





Source: Crea Eur Heart J 2014;35:1101–1111.



Macrocirculation

Macrocirculation & Microcirculation

Source: Taqueti et al. J Am Coll Cardiol 2018;72:2625-41.

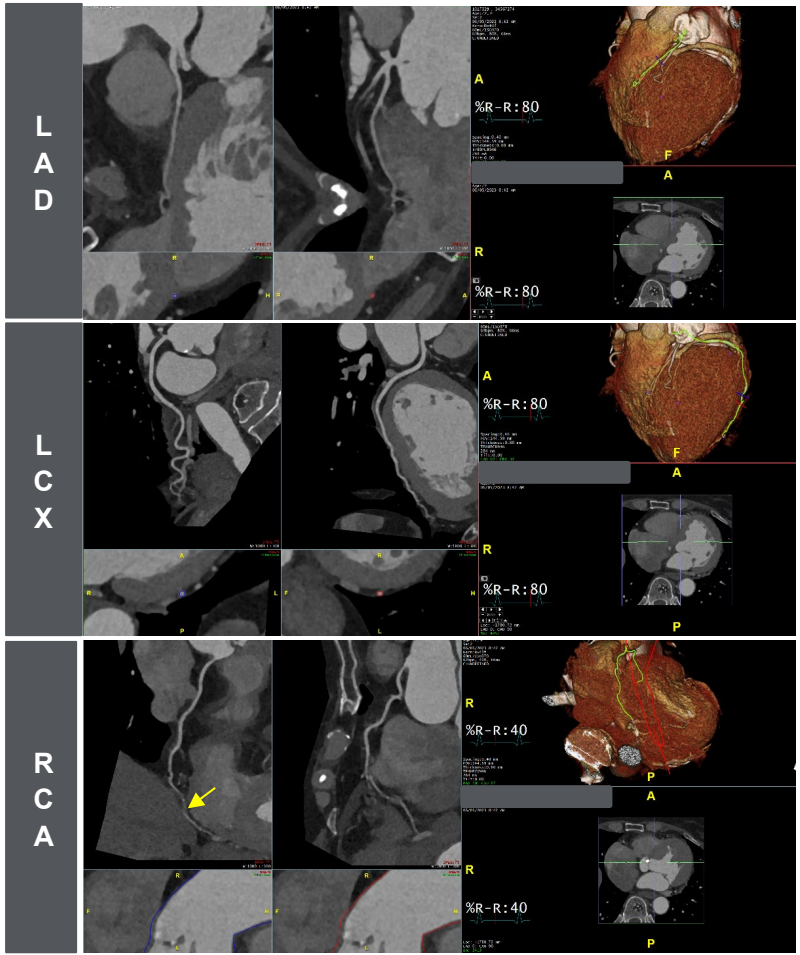
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Case #C5:

- A 62-yo woman h/o HTN, complains of chest pain.
  - Notes symptoms are related to exertion and associated with dyspnea.
  - Normal baseline EKG.
  - Normal echocardiogram with EF of 60% and no regional wall motion abnormalities.

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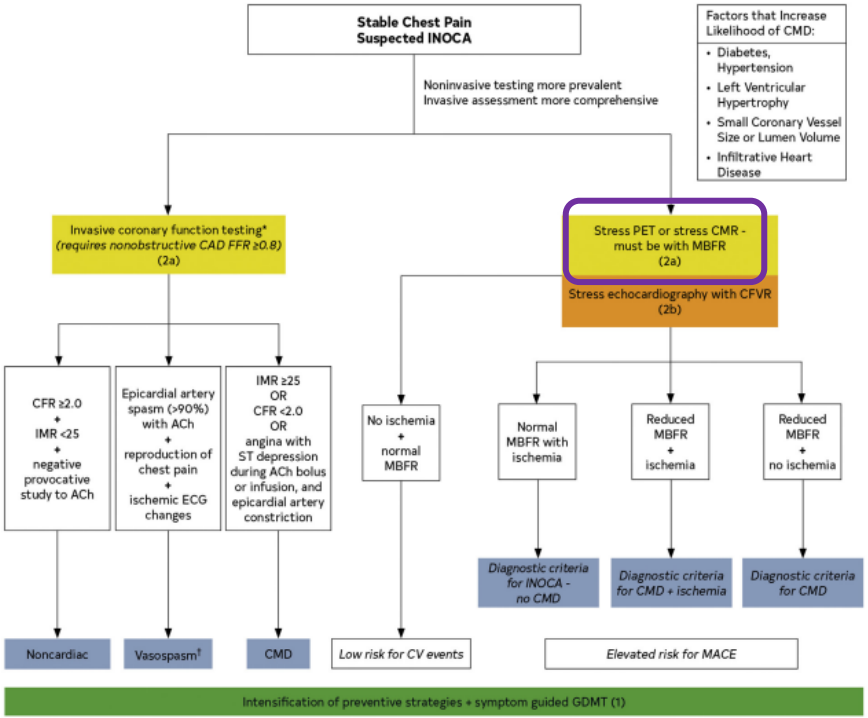
Normal coronary arteries



Courtesy of Muhamed Saric, MD

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Example of Utilization of Newer Technology: Evaluation in Suspected INOCA

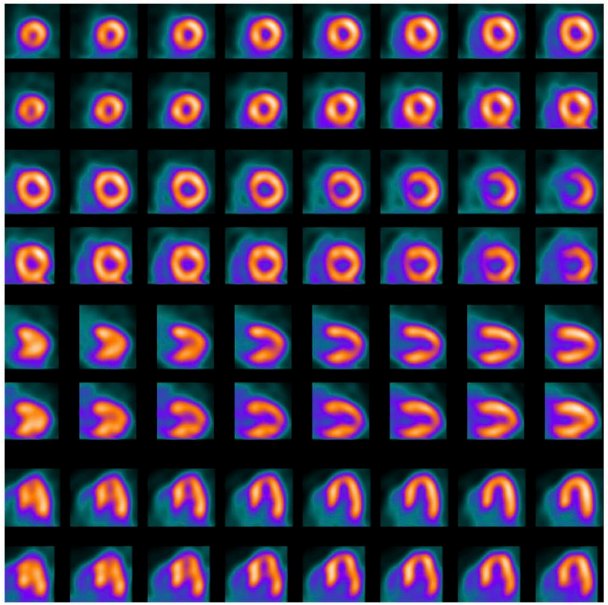


Source: Gulati M, et al. JACC. 2021 Nov, 78 (22) e187-e285.

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Case #C5: A 62-yo woman h/o HTN, complains of chest pain

Coronary microvascular dysfunction



	Rest (ml/g/min)	Stress (ml/g/min)	Reserve (Stress/Rest)
LAD	**0.61**	**0.78**	**1.27**
LCx	**0.59**	**0.65**	**1.09**
RCA	**0.80**	**0.92**	**1.14**
Global	**0.67**	**0.79**	**1.19**

C

	Rest (mg/g/min)	Stress (mg/g/min)	Reserve (Stress/Rest)
LAD	**0.80**	**0.95**	**1.20**
LCx	**0.98**	**1.08**	**1.11**
RCA	**1.05**	**1.32**	**1.26**
Global	**0.91**	**1.09**	**1.20**

Gating:
- Rest LVEF 70%
- Augmented to 74% with vasodilator Stress

CACS: 2

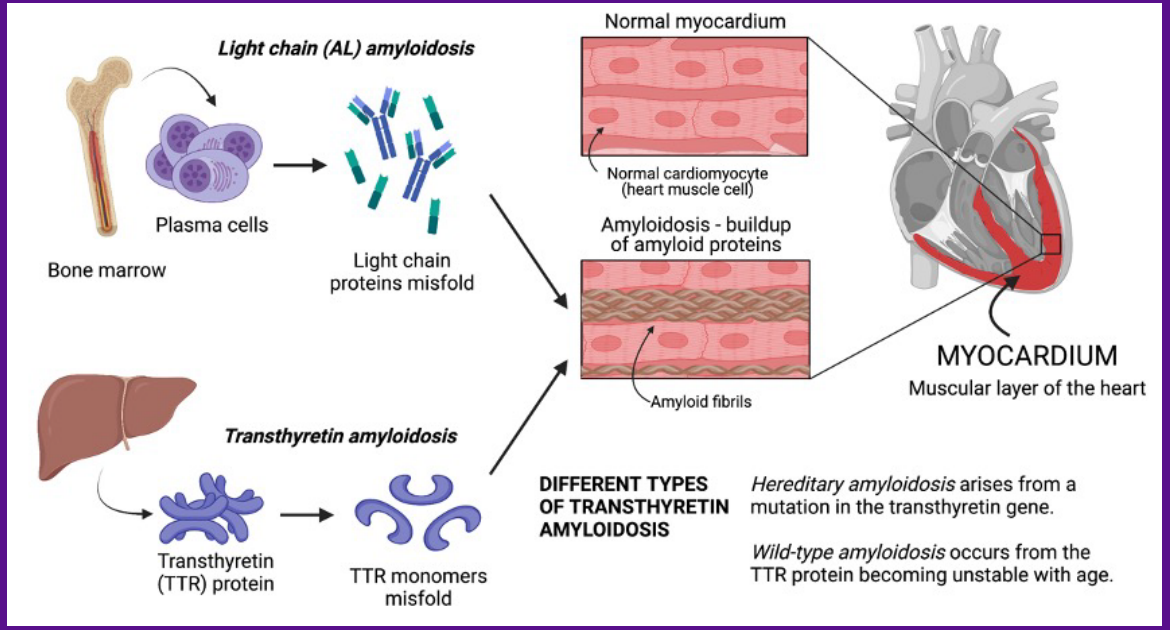
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Take Home Points

- Many patients with chronic chest pain are at lower clinical risk than previously considered.
- Younger patients without known coronary artery disease often do well with initial anatomic testing.
- Older patients and those with known coronary artery disease often do well with initial functional testing.
- CT-FFR allows functional assessment of significant stenotic lesions on CCTA.
- Cardiac PET/CT allows comprehensive assessment of both ischemia and atherosclerosis.
- Myocardial blood flow assessment allows evaluation of macrovascular and microvascular disease.



A Diagnosis Not to Miss: Cardiac Amyloidosis

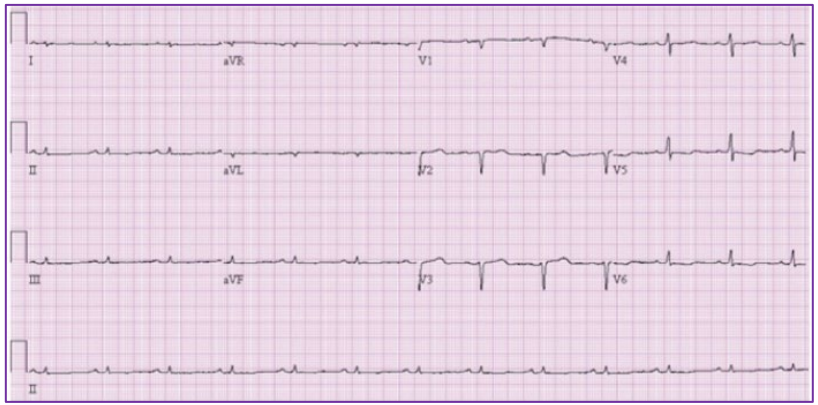


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Source: Modified from https://ucsfhealthcardiology.ucsf.edu/care/clinical/amyloid

Case A1:

- 64 year old woman with bilateral carpal tunnel, presents for evaluation of shortness of breath.
  - Notes progressive decrease in her functional capacity over the past six month.
  - She denies orthopnea or PND.
  - She denies associated chest pain.
  - Echocardiography with evidence of left ventricular hypertrophy



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What's the diagnosis?

Not surprisingly (based on the topic): ATTR-CM

- > 4 year delay between symptoms onset and diagnosis of ATTR
- > 2 year delay between onset of HF and diagnosis of ATTR-CM
- > 6 year delay between carpal tunnel syndrome and diagnosis of ATTR
- > 6 year delay between LVH diagnosis and diagnosis of ATTR-CM

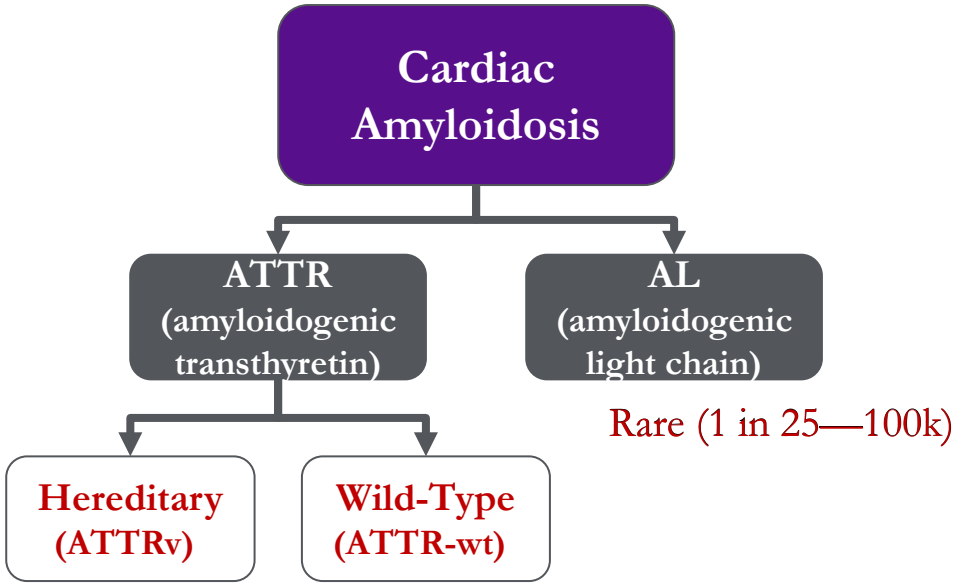
Source: Rozenbaum, MH et al. Cardiol Ther. 2021 Apr 20;10(1):141–159.
Achten, A et al. Heart Vessels. 2024 Oct;39(10):857-866.

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What is Cardiac Amyloidosis?

- Amyloidosis is a family of systemic diseases due to **deposits of misfolded proteins, amyloid fibrils**, in tissues.
- Cardiac amyloidosis refers to when these deposits are made in the heart, specifically the interstitial space between cardiac myocytes, resulting in a thickened, restrictive picture.
- Traditionally considered a “rare disease,” improvements in diagnostic capabilities and awareness of the disease is resulting in higher diagnosed prevalence.
- Now viewed as an “**underdiagnosed**” disease process.

Major Types of Cardiac Amyloidosis



Rare (1 in 25—100k)

- Over 130 genetic variants

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Why focus on earlier diagnosis now?

1. AL amyloidosis is an emergency diagnosis and requires prompt initiation of treatment
2. TTR amyloidosis has seen an explosion in the treatment options.

Treatment	Mechanism	US approval context
Tafamidis	TTR stabilization	Established ATTR-CM therapy
Acoramidis	TTR stabilization	2024 approval
Vutrisiran	RNA silencing	2025 ATTR-CM expansion

CLINICAL PRESENTATIONS

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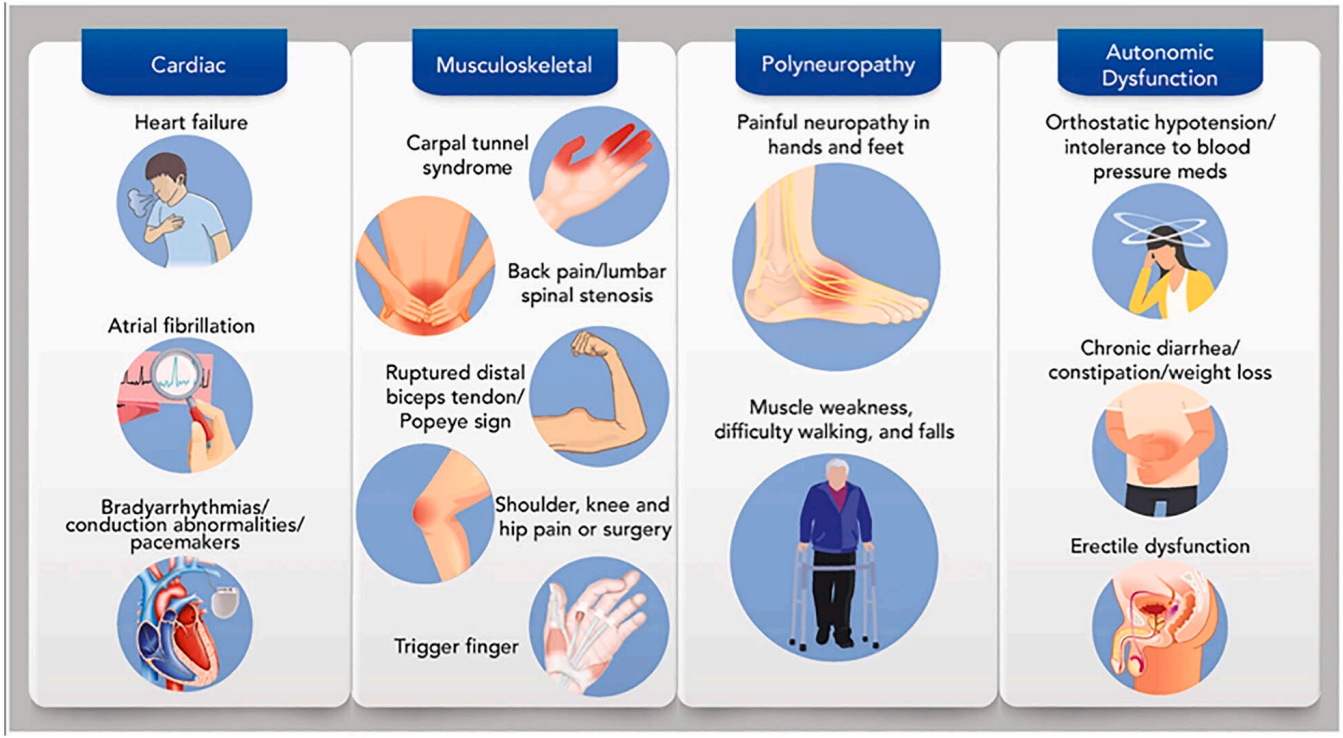
Clinical Presentations

- Age:
  - AL: > 40 years old
  - TTR: 60-70's years old
- Cardiac symptoms:
  - ✓ Fatigue
  - ✓ Dyspnea
  - ✓ Elevated JVP
  - ✓ Hepatic congestion
  - ✓ Lower extremity edema
  - ✓ Orthostasis
  - ✓ Arrhythmias and conduction abnormalities



- Labs
  - ✓ Low level troponin elevation
  - ✓ Elevated BNP

Clinical Presentations Suggestive of Amyloidosis

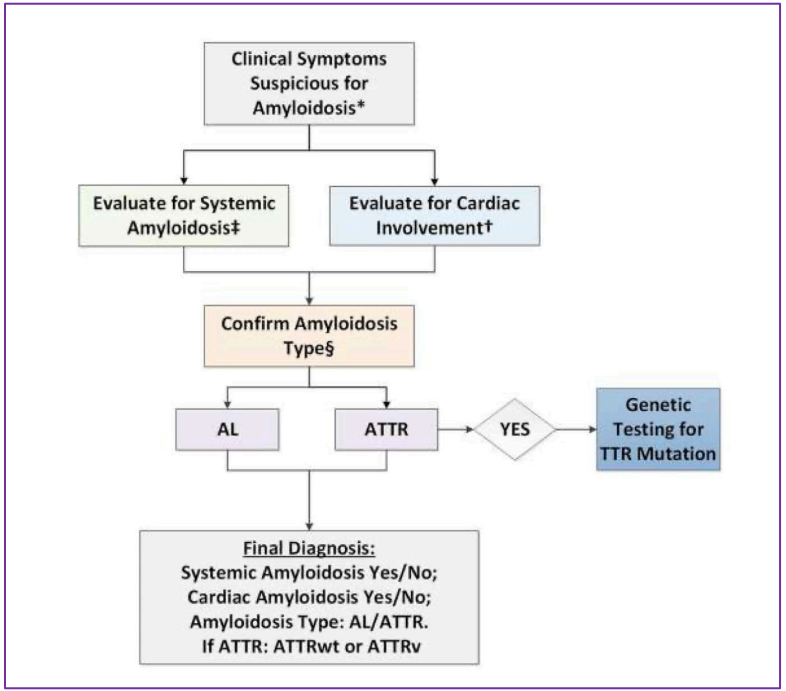


Cardiac TTR Amyloidosis

- 16% patients Aortic stenosis
- 13% patients Heart Failure with Preserved Ejection Fraction

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Systematic Evaluation for Cardiac Amyloidosis



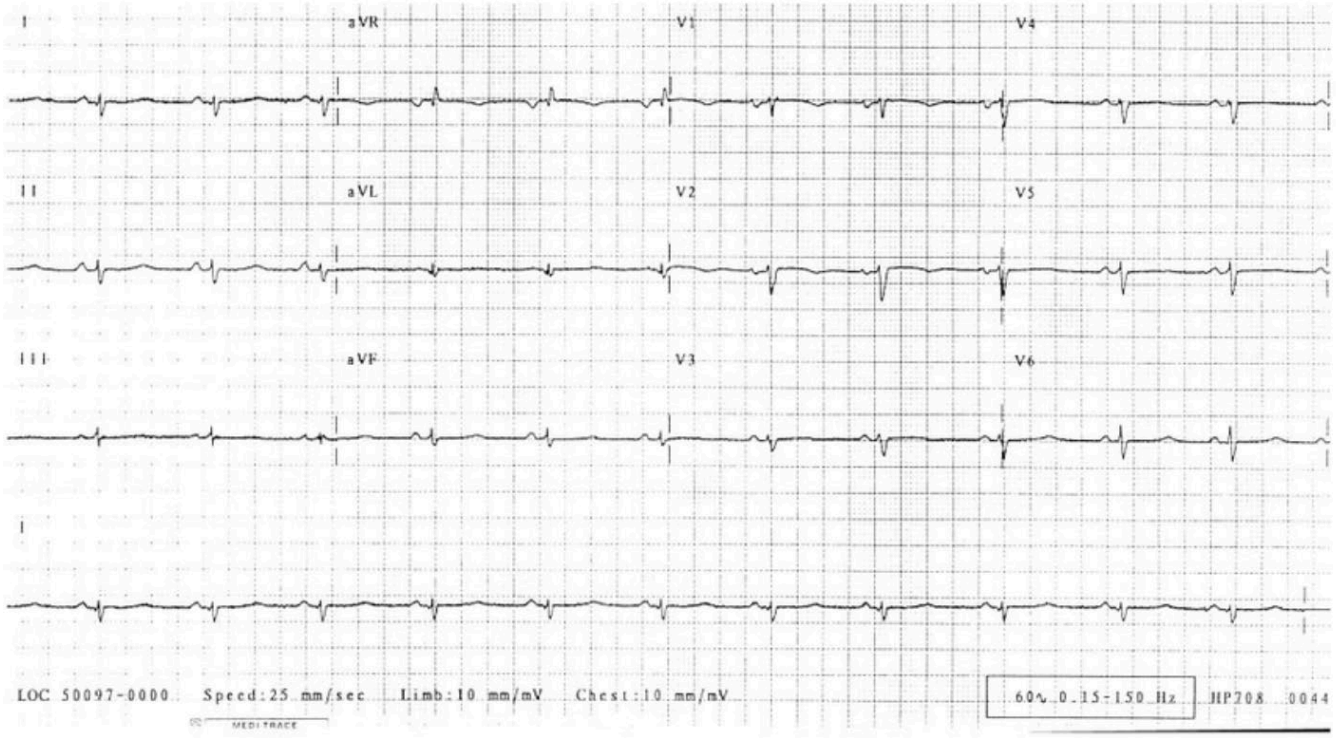
Source: Dorbala S, et al. Nucl Cardiol 2019;26:2065–123

NYU Langone Heart: Cardiac Amyloidosis Order Set



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EKG Findings Associated with Amyloidosis

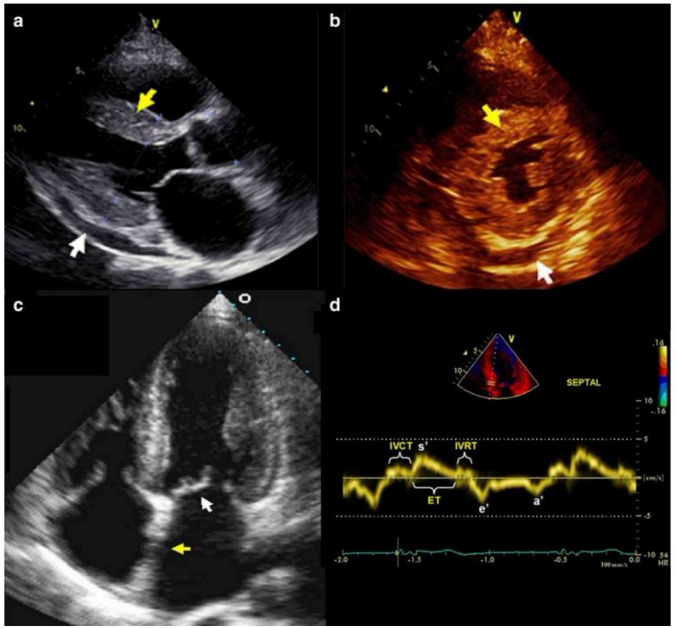


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Echocardiography: *Helps support, but not diagnostic…*

Typical echocardiographic findings:

- ✓ Increased biventricular thickness
- ✓ Speckling pattern of the left ventricle
- ✓ Pericardial effusion
- ✓ Biatrial enlargement
- ✓ Thickened interatrial septum
- ✓ Thickened mitral valve leaflets
- ✓ Low septal mitral annulus tissue doppler imaging velocities
- ✓ Diastolic dysfunction

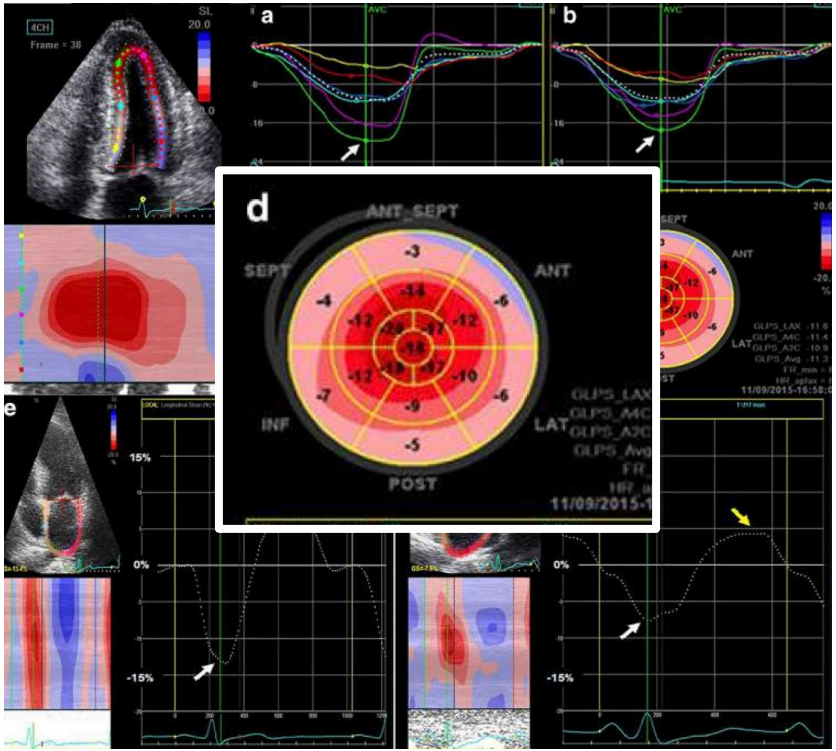


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Echocardiography: Longitudinal LV Strain

- ✓ Decreased global longitudinal LV strain (< -15%)
- ✓ “Cherry-on-top” for bulls-eye view

Note: Although these findings are strongly suggestive of cardiac amyloidosis, they are unable to differentiate AL from ATTR



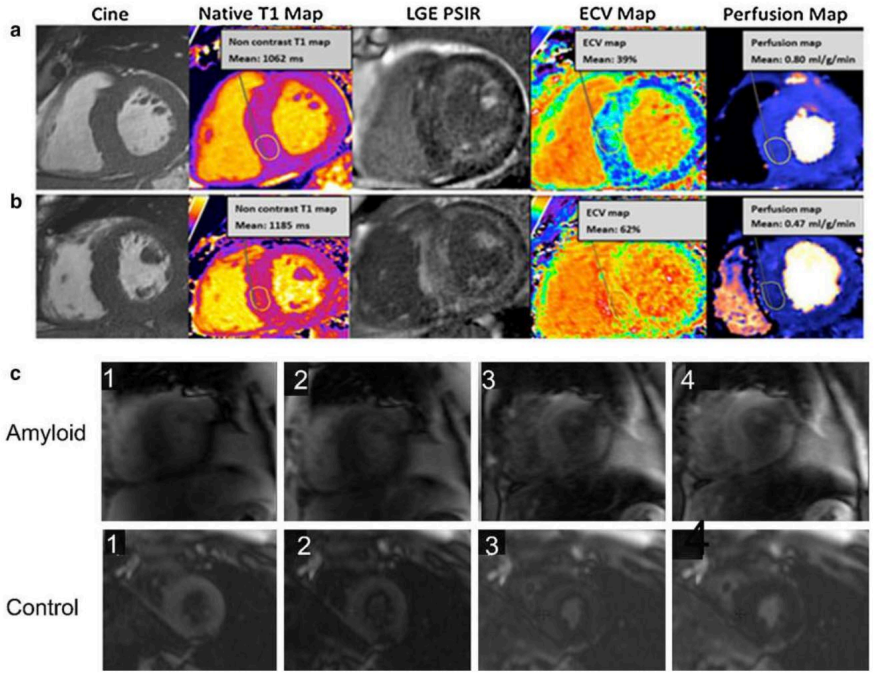
Source: Dorbala S, et al. J Nucl Cardiol 2019;26:2065–123

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Cardiac MRI

- Helpful for differentiation of cardiac amyloidosis from other forms of cardiomyopathy with thickened left ventricular wall.
- Very helpful for its negative predictive value.
- Unable to differentiate AL vs TTR.

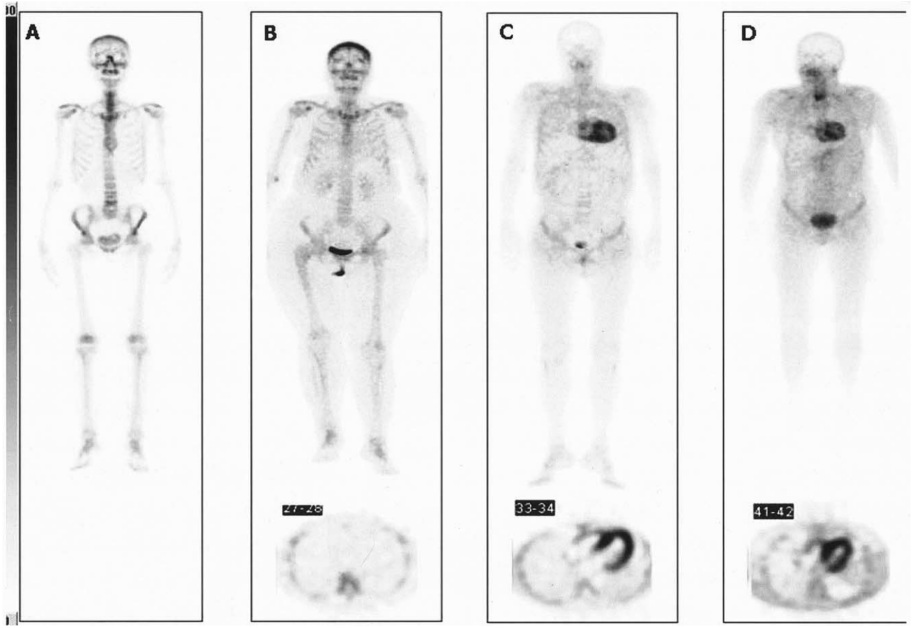
Characteristic CMR Findings
Expanded extracellular volume
Abnormal gadolinium contrast kinetics
Diffuse late gadolinium enhancement



Source: Kittleson, M et al. *JACC.* 2023 Mar, 81 (11) 1076−1126.
Dorbala S, et al. J Nucl Cardiol 2019;26:2065–123

Cardiac Amyloidosis Radionuclide Imaging

Unaffected AL TTR TTR

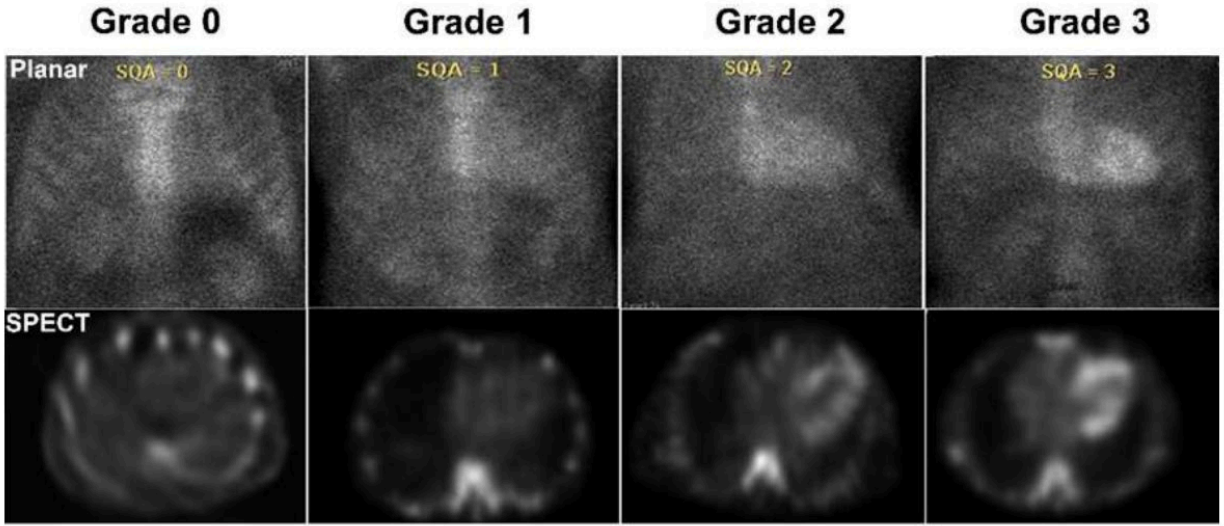


Grade	
0	No cardiac uptake
1	Myocardial uptake is less than rib uptake
2	Myocardial uptake is equal to rib uptake.
3	Myocardial uptake is greater than rib uptake.

Source: Perugini E, et al. J Am Coll Cardiol 2005;46:1076–84

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Nuclear Imaging



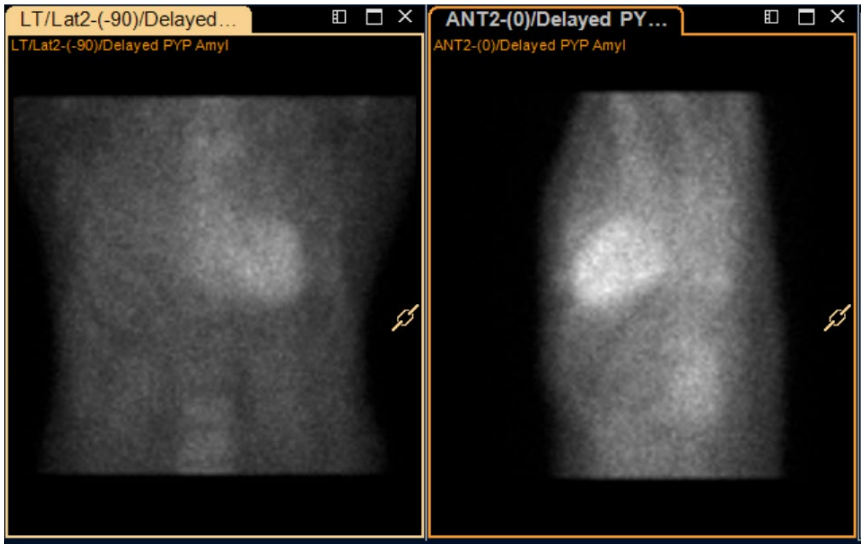
✓ Imaging at 1-3 hours post-injection

Bone Imaging Agents Helpful for TTR

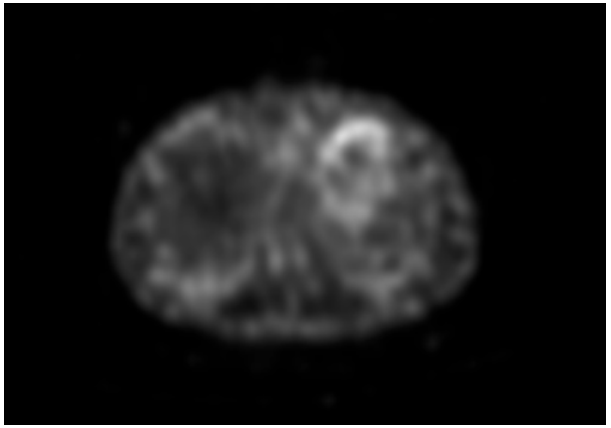
- Tc-PYP: pyrophosphate
- Tc-DPD: diphosphono-1,2-propanodicarboxylic acid
- HMDP (HDP): hydroxymethylene diphosophonate

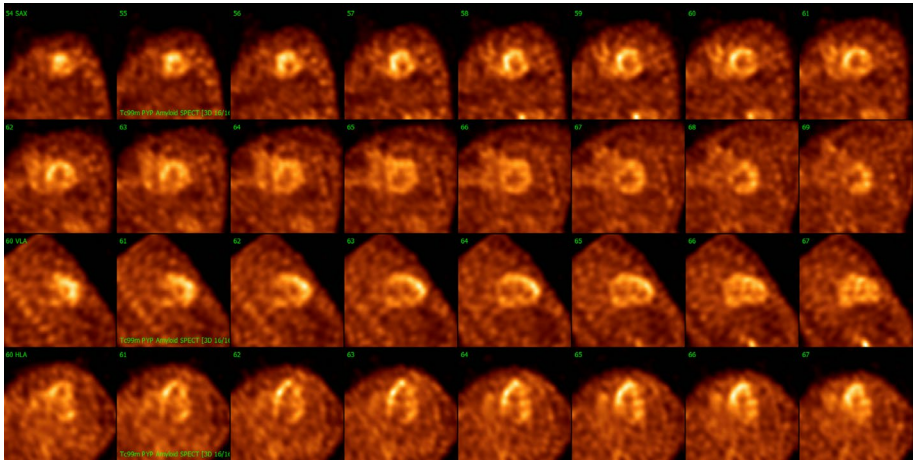
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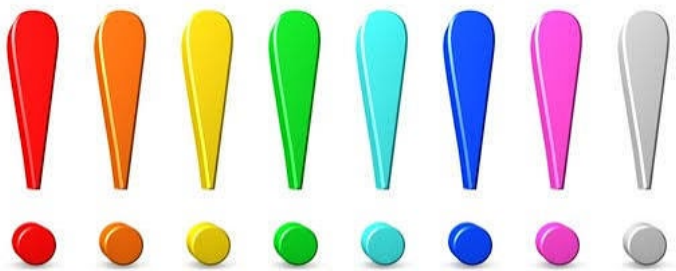
Planar and SPECT imaging



H/C 1.9



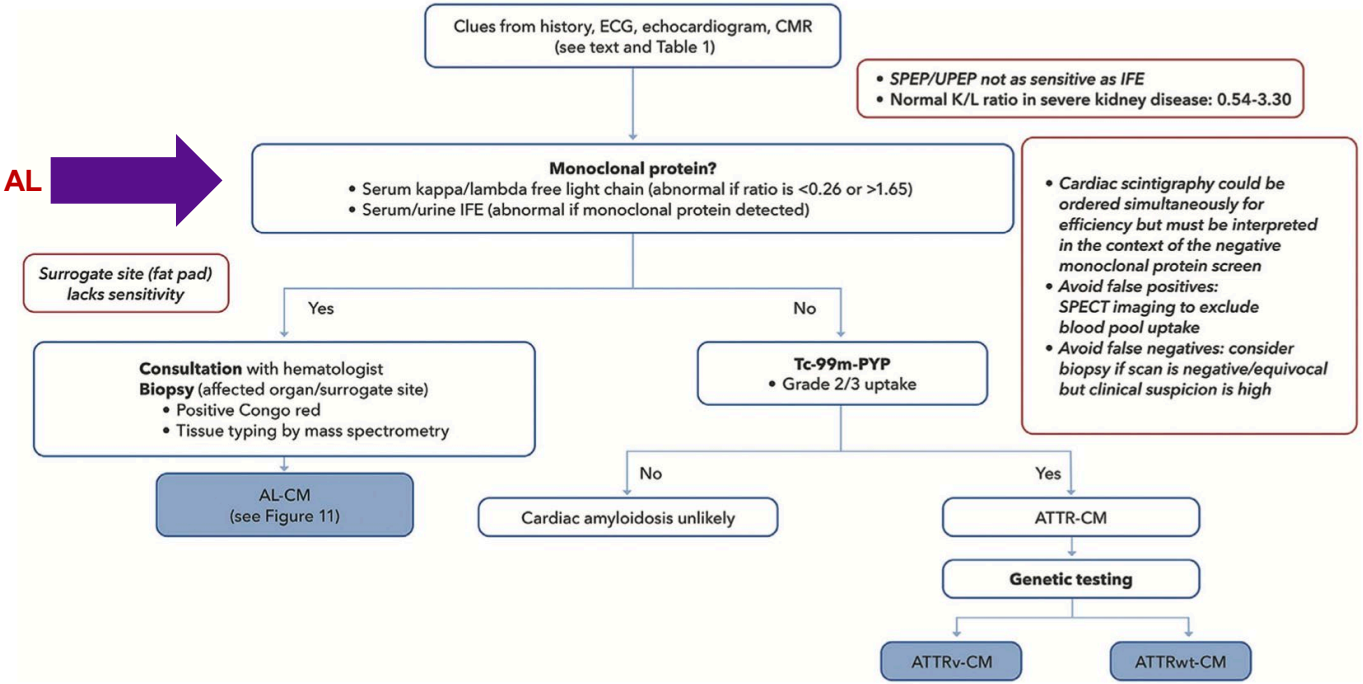




Important: Always start your diagnostic evaluation with testing for a plasma cell disorder and free light chains to rule out AL amyloidosis!

Diagnostic Algorithm for Amyloidosis

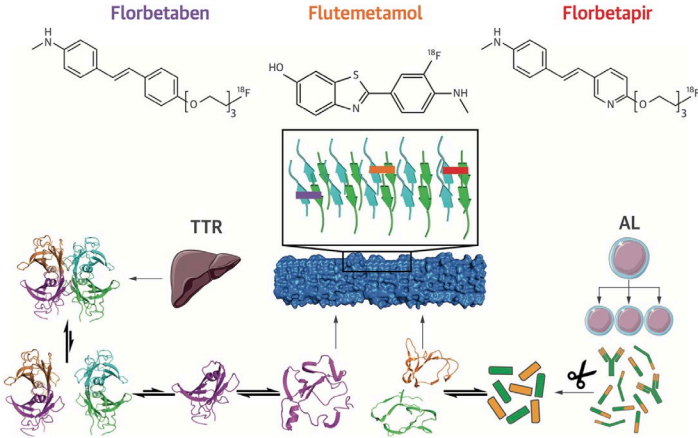
Do not miss AL

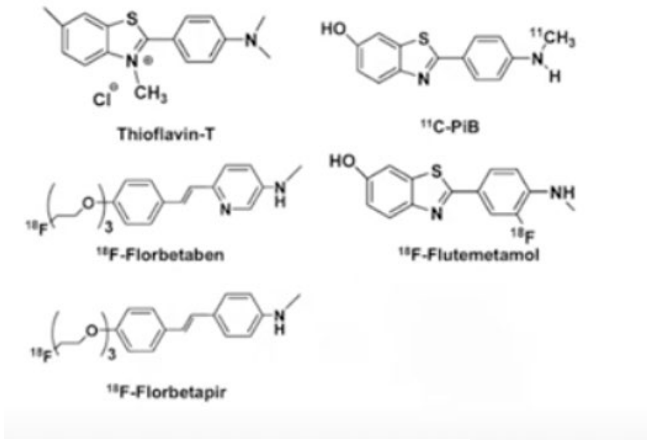


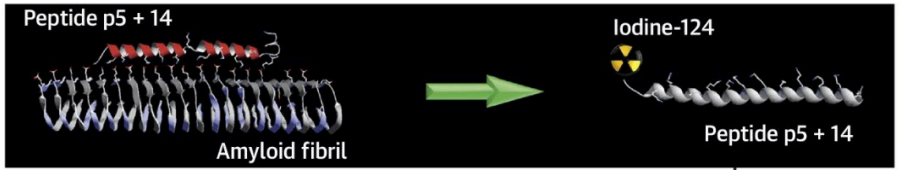
Source: Kittleson MM, et al. J Am Coll Cardiol. 2023;81:1076-1126

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Cardiac PET Allows for Earlier Pan-Amyloid Diagnosis, Extra-cardiac Disease, Differentiation of Subtypes and Possibly Treatment Response







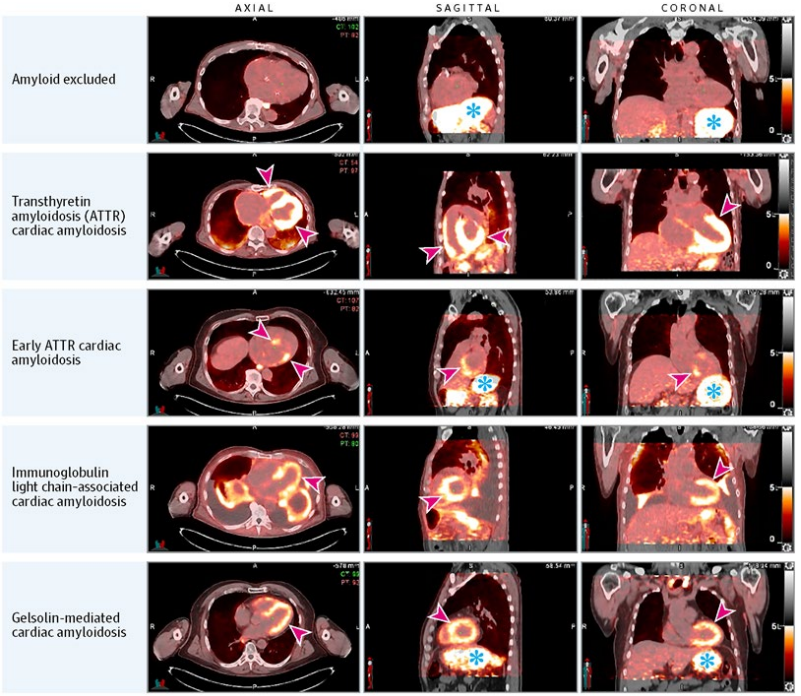
Source: Packard R, et al. JACC Cardiovasc Imaging. 2025
Dorbala S & Kijewski MF. J Nucl Med. 2023 Nov;64(Suppl 2):20S-28S.
Wall JS, et al. J Am Coll Cardiol Img. 2023;16(11):1433-1448

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Hot Off The Press: REVEAL (Research With I-124 Evuzamitide to Elucidate Cardiac Amyloidosis)

- Phase 3, open-label, multicenter study
- 170 patients suspected of having cardiac amyloidosis.

Objectives	Endpoints
Primary	
To evaluate the efficacy of ^{124}I-evuzamitide for diagnosing cardiac amyloidosis.	• The sensitivity of PET/CT imaging with ^{124}I-evuzamitide for the diagnosis of cardiac amyloidosis based on visual scan interpretation. • The specificity of PET/CT imaging with ^{124}I-evuzamitide for the diagnosis of cardiac amyloidosis based on visual scan interpretation.
Secondary	
To evaluate the efficacy of ^{124}I-evuzamitide for diagnosing cardiac amyloidosis from ATTR.	• The sensitivity of PET/CT imaging with ^{124}I-evuzamitide for the diagnosis of ATTR cardiac amyloidosis based on visual scan interpretation. • The specificity of PET/CT imaging with ^{124}I-evuzamitide for the diagnosis of ATTR cardiac amyloidosis based on visual scan interpretation.
To evaluate the efficacy of ^{124}I-evuzamitide for diagnosing cardiac amyloidosis from AL.	• The sensitivity of PET/CT imaging with ^{124}I-evuzamitide for the diagnosis of AL cardiac amyloidosis based on visual scan interpretation. • The specificity of PET/CT imaging with ^{124}I-evuzamitide for the diagnosis of AL cardiac amyloidosis based on visual scan interpretation.



Source: Dorbala S, et al. JAMA. 2026 Aug 30:e2617361

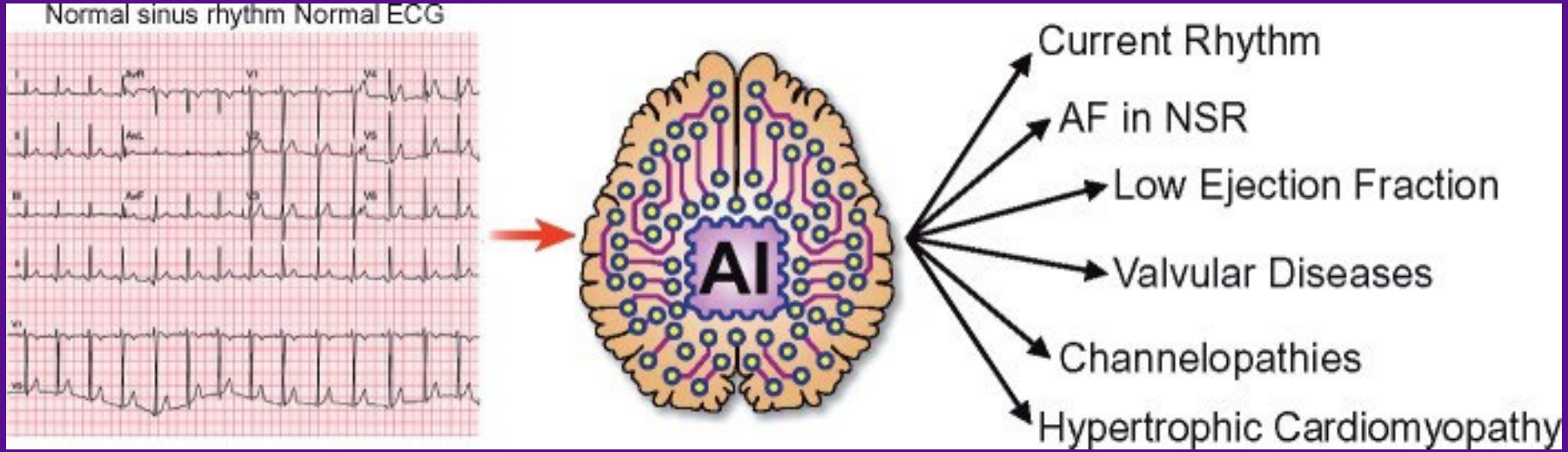
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Key Points for Cardiac Amyloidosis

- ✓ Recognize the patterns
- ✓ Order the complete lab evaluation – monoclonal screen
- ✓ Ensure correct imaging is performed
- ✓ Refer early

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Brief Comment: AI in Cardiology



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Source: Attia ZI, et al. Eur Heart J. 2021 Sep 17:ehab649.

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Lots of Exciting Advances in Cardiac Care to Share With My IM Colleagues

Lawrence Phillips, MD, MASNC, FACC, MACP
Deputy Director, Cardiology
Associate Professor of Medicine
Director, Nuclear Cardiology
Medical Director, Outpatient Cardiology
Senior Associate Director, Training Program in CV Diseases
Leon H Charney Division of Cardiology,
NYU Grossman School of Medicine

Former Governor, New York Chapter (Manhattan) of the American College of Physicians

October 24, 2026