# Reminders for Discussions in Workshop

- 1—In opinions-- focus on favorable and unfavorable features contributing to risk.
- 2—Please refrain from describing exact numeric tables or other ratings or specific company philosophies or decisions or pricing--> instead use: "better than standard risk", "standard risk", "substandard risk", or "decline".
- 3—It is important to couch discussion in educational terms and avoid implications of price fixing.  Every individual company has multiple factors that contribute to their risk tolerance and pricing calculations. (The same label does not = the same risk calculation.)

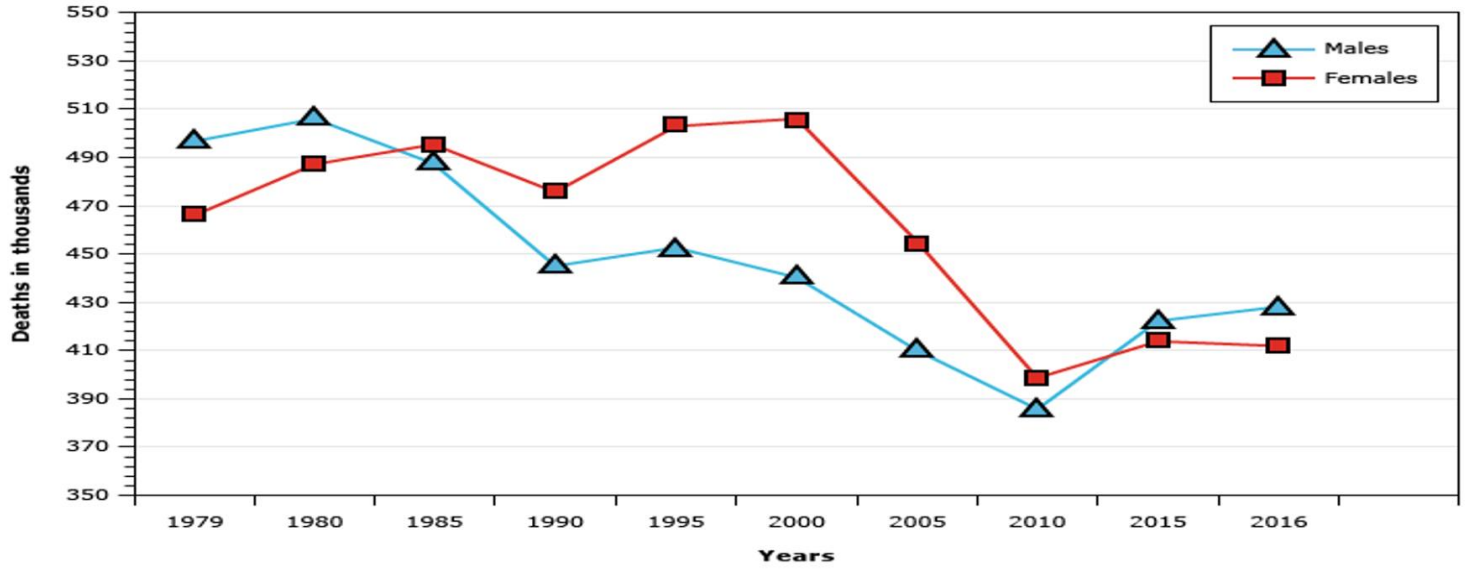
# Objectives for CAD portion of CV Workshop

- **For suspected CAD:**
  - 1—Review trends and risk algorithms (Framingham and others) pros and cons
  - 2—Discuss angiographic options: EBCT, Coronary angiography vs. CCTA and newer plaque options
  - 3—Compare NT-ProBNP vs. ECG screening and mortality risk (this will be in valve dis. portion)
- **For established CAD:**
  - 1—Define and assess “non-obstructive CAD”
  - 2—Discuss plaque morphology and mortality risk
  - 3—Examine co-morbidities and revascularization impacts on mortality

# Part 1—Trends and Background Information

**Are we going in the wrong direction again?**

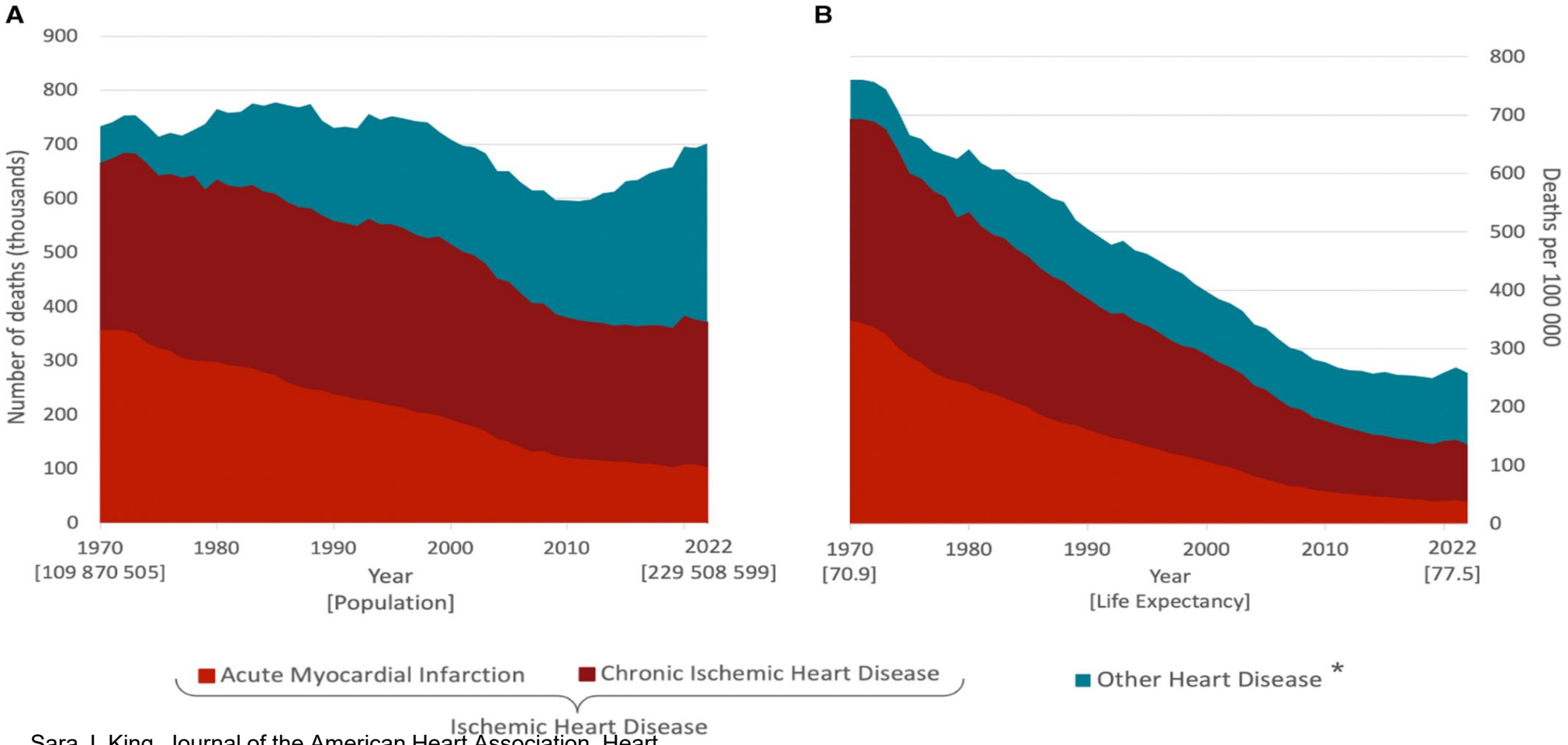
**Cardiovascular disease (CVD) mortality trends for males and females (United States: 1979-2016)**




CVD excludes congenital cardiovascular defects (International Classification of Diseases, 10th Revision [ICD-10] codes I00-I99). The overall comparability for cardiovascular disease between the International Classification of Diseases, 9th Revision (1979-1998) and ICD-10 (1999-2015) is 0.9962. No comparability ratios were applied.

# Yes, CAD but…50 years of change

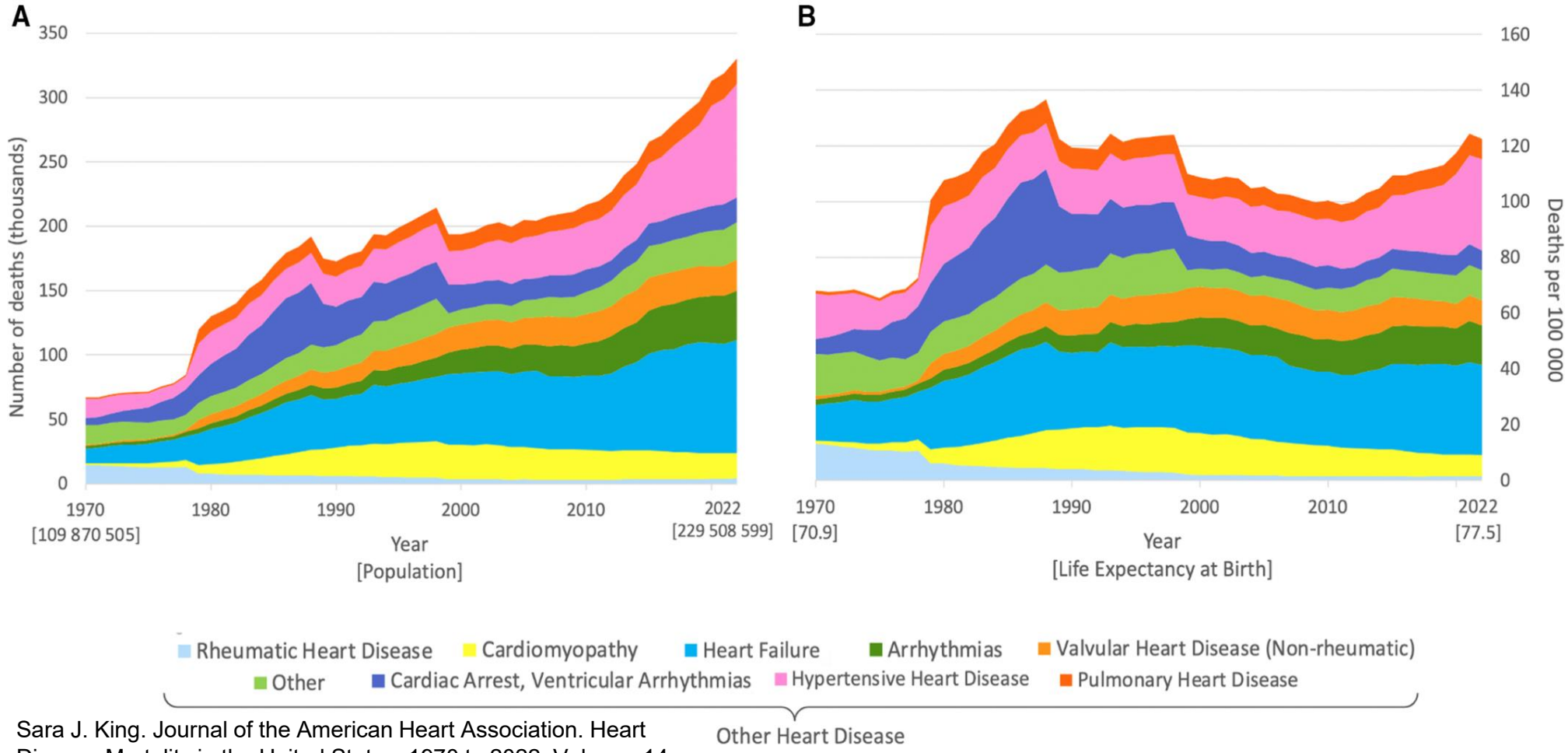
- 89% Acute MI declined
- 81% chr. ischemic declined
- 81% other types increased




Sara J. King. Journal of the American Heart Association. Heart Disease Mortality in the United States, 1970 to 2022, Volume: 14, Issue: 13, DOI: (10.1161/JAHA.124.038644)

# Yes, CAD but…do we need to think more broadly?

- 12% increase in CHF
- 13% increase in bp
- 4% increase in arrhythmia




Sara J. King. Journal of the American Heart Association. Heart Disease Mortality in the United States, 1970 to 2022, Volume: 14, Issue: 13, DOI: (10.1161/JAHA.124.038644)

# Why the 50 Yr. Changes in Mortality-prone CV diseases?

- **The favorables in change:**
- 1—**Effective MI interventions**--> bystander CPR/AEDs/CCU's and CABG; PTCA and stents and thrombolytics; Med. Rx like BB/RAS inhib./statins and lipid drugs/dual antiplatelet Rxs/acute MI cath lab programs/secondary post MI prevention programs
- 2—**Primary prevention (and secondary)** in general—smoking cessation; physical activity increases; bp and lipid goals; CV rehab
- 3—**Earlier Dx testing**

- **The unfavorables in change:**

1--**Obesity**--> 15%-->40% of adults

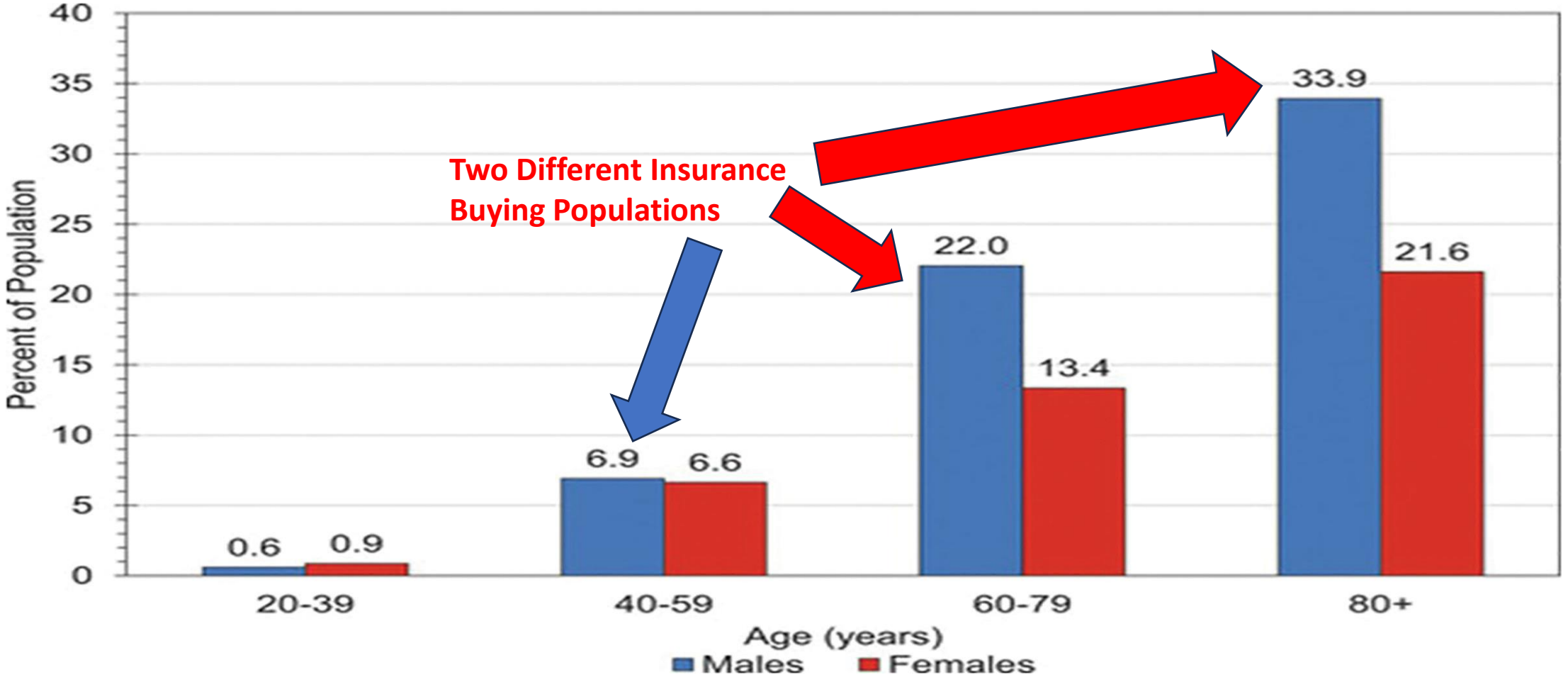
2--**DM and PreDM**--> now 50% adults

3--**High Bp**-->30% -->50% adults

Thus…**CKM** as a **"new disease"** now (Cardio-Kidney-Metabolic)—**PREVENT** predictors

4--**Aging population**

Overall Covid had a bump but didn't change the overall trends


Two Different Insurance Buying Populations
Percent of Population
40
35
30
25
20
15
10
5
0
0.6
0.9
6.9
6.6
22.0
13.4
33.9
21.6
20-39
40-59
60-79
80+
Age (years)
Males
Females

# Part 1—Case 1—CAD Risk calculation with traditional risks and tests—what can help you?

Case 1—39 yo M with smoking, SOB with exertion, creat 1.6, father first MI at 50yo, lipids TC 250 and HDL 40; he appl. for $1M. Term life insurance—**What if this is all you had?**

He has a **Bruce protocol TM** to 7 minutes with peak HR getting to 76% predicted for age, SOB but no CP, and 1.5 mm ST upsloping depression in leads 2,3 and avF and V5-6 at peak—lasting 4 min. into recovery and with "some" PVCs in recovery

He came out with a **Duke TMS** of -0.5 or "moderate" risk.

He had a **resting echo** with LVEF of 48% and an apical area hypokinesis.

He is now on rosuvastatin 5 mg qd and a baby aspirin; he tried a BB and stopped secondary to erectile dysfunction.

1—Vote→(if you only had the TM)
- --Better than std. risk
- --Std. risk
- --Substd. risk
- --Decline

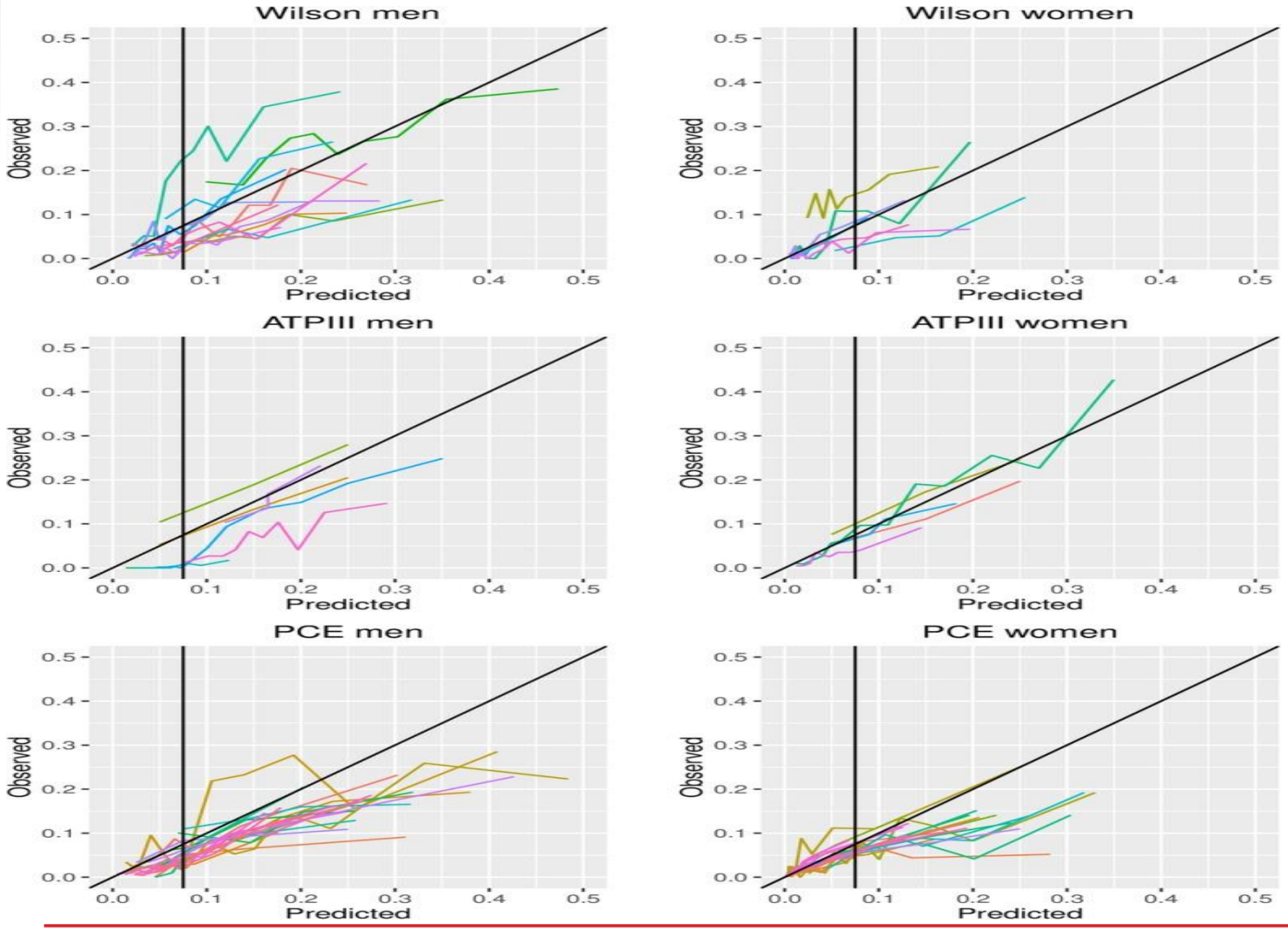
2—Vote→(if you had TM and Duke score)
- --Better than std. risk
- --Std. risk
- --Substd. risk
- --Decline

3—Vote→(if had TM, Duke score, Echo)
- --Better than std. risk
- --Std. risk
- --Substd. risk
- --Decline

| Risk Scores | Measures What? | Validity | # CV Risks | Cons |
|---|---|---|---|---|
| Framingham | 10 yr. --Hard events (MIs, deaths) | Widely valid; long duration; started 1948; publ. 1961 | 7 risks | Overestimates low risk and underestimates high risk –esp. certain populations |
| SCORE | 10 yr. --Risk fatal MI and CVA | European populations | 5 risks | No DM gradations or HDL (SCORE2 is better at those) |
| Reynolds | 10 yr.-- Risk composite (MIs, isch. CVA, cor. revasc., CV deaths) | Less widely used/validated | 8 risks inclu. early age fam hx and hs-CRP | |
| ASCVD risk estimator (PCE = pooled cohort equations) | 10 yr. and lifetime—CHD, CVA, PAD | Newest-2013; shortest track record | 8 risks inclu. race | Overestimates risk in low risk populations |

# Calibration plots of the Framingham Wilson, ATP III and PCE (Pooled Cohort Equation) models

## Calibration plots of the Framingham Wilson, ATP III and PCE models.




Each line represents one external validation. The diagonal line represents perfect agreement between observed and predicted risks.

**All points below that line indicate that more events were predicted than observed (overprediction) and points above the line indicate fewer events were predicted than observed (underprediction).**

The vertical black line represents a treatment threshold of 7.5%

**Is Framingham getting too old for current epidemiology?**

# Why the Framingham (or any) risk score can never be a 'perfect' predictor

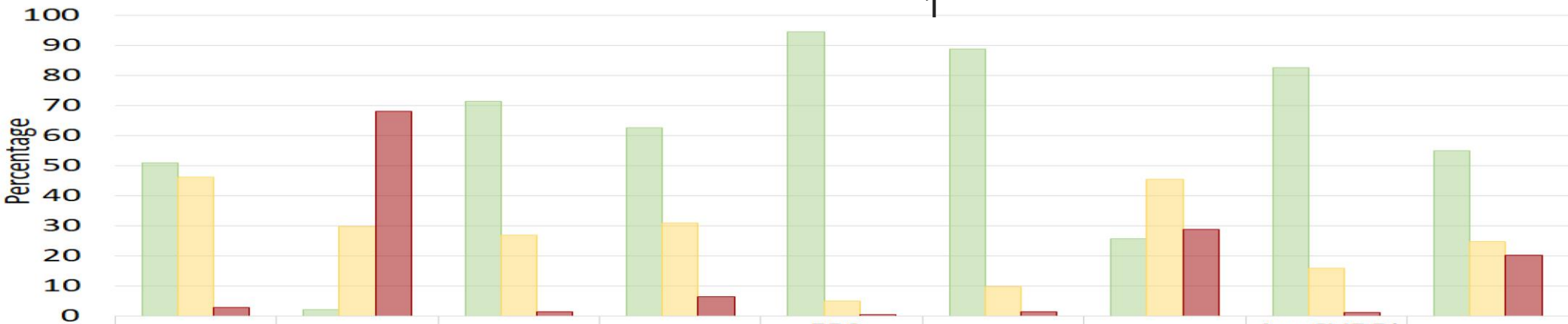
- **1--First, Framingham can never be truly predictive once applied to a different population**
  - its data were obtained from a North-Eastern American, predominantly middle-class, urban, Caucasian population.
- **2--Second, some age–gender groups were rather small**
  - making these underpowered to provide anything like a reliable predictive estimate of cardiovascular risk.
- **3--Third,** with infallible 20:20 hindsight (not available to Framingham's originators), **several risk factors might now reasonably be added to improve 'predictive accuracy'**
  - not least family history, ethnicity, fibrinogen, lipoprotein (a) and socio-economic status.
- **4--Fourth,** Framingham provides an estimate of the absolute risk, that is, the chances of developing cardiovascular disease within 10 years
  - as age is a major contributor to the score, **relative risks are more appropriate in older age.**
- **5--Fifth**, giving the score as a **point estimate is not strictly correct – the score is the median of a range**
  - so some individuals with a specific risk score will do better (and have a lower risk) than the estimated risk and others will do worse (and have a higher risk) than predicted risk.

# PREVENT Equation: The Black Sheep among Cardiovascular Risk Scores? A Comparative Agreement Analysis of Nine Prediction Models in High-Risk Lithuanian Women

The selection of a RPM plays a pivotal role in influencing clinical decisions and managing patient care. In the comparison of cardiovascular risk categorization methods, the **PREVENT model emerged as a balanced option**, steering clear of the extremes seen in both SCORE2 and FRS-hCHD.

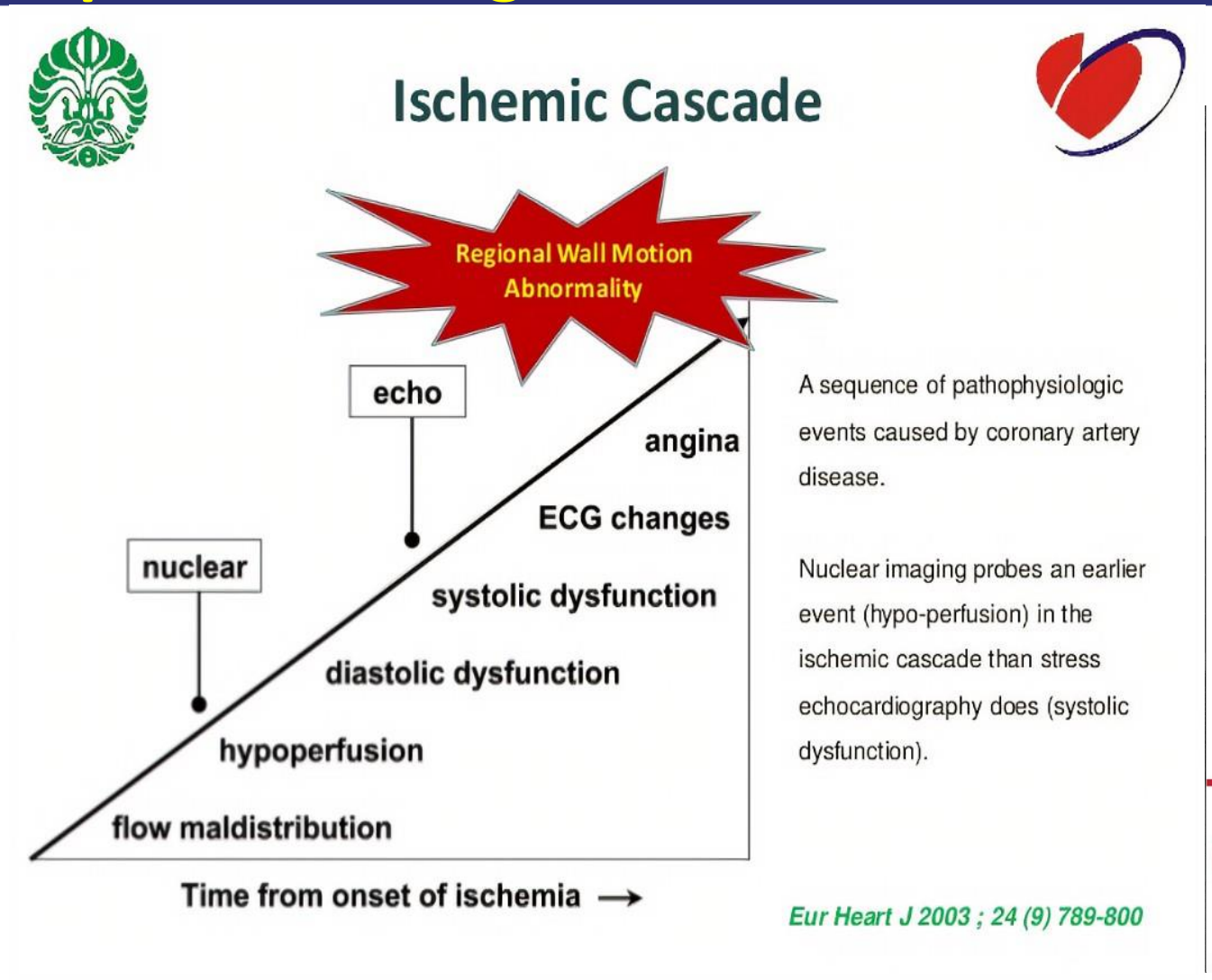
Remarkably, **agreement across all nine models on the same risk category for a patient was rare, occurring in only 1.98% of cases.**

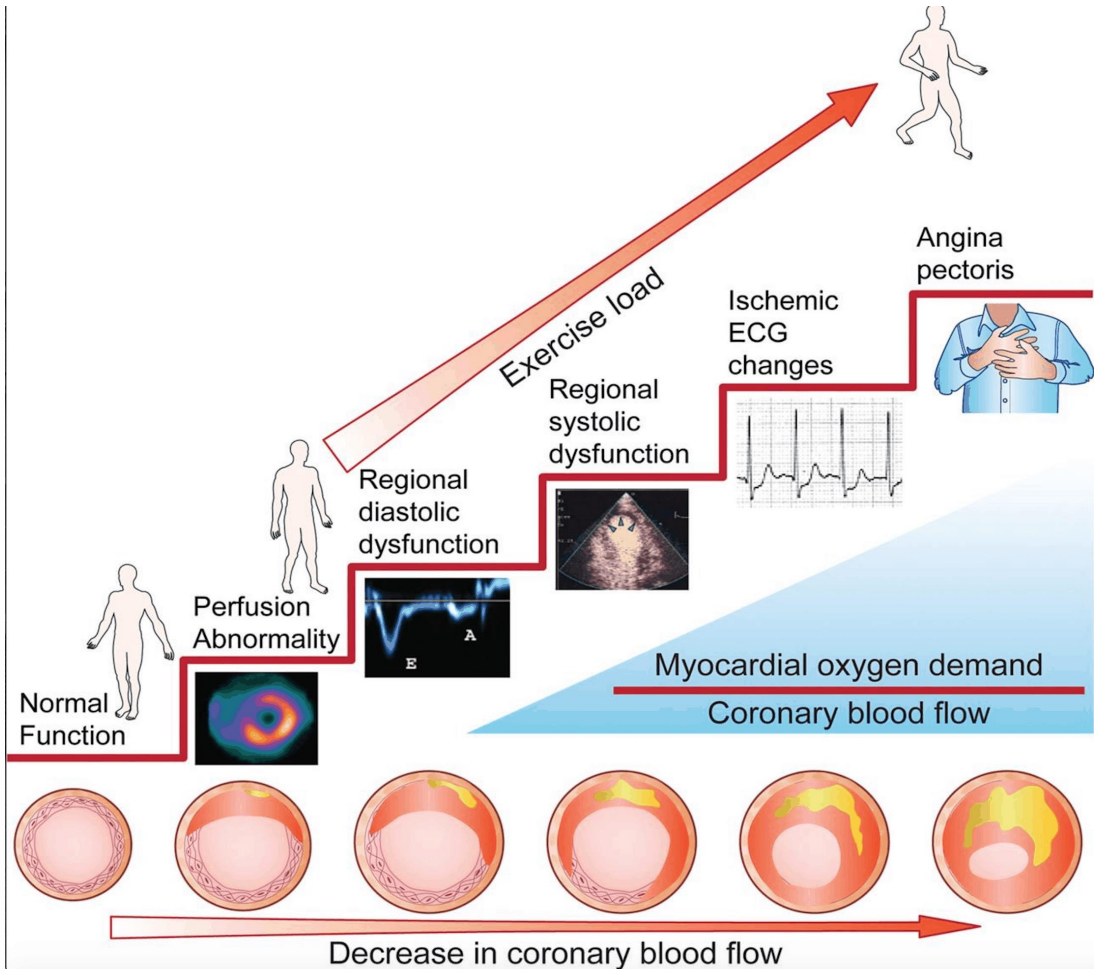
*Medicina*. 2024; 60(9):1511. https://doi.org/10.3390/medicina60091511




| | PREVENT | SCORE2 | PCE | QRISK3 | FRS-hCHD | RRS | ASSIGN | AusCVDRisk | MESA |
|---|---|---|---|---|---|---|---|---|---|
| Low risk (%) | 50.99 | 2.14 | 71.47 | 62.56 | 94.42 | 88.72 | 25.79 | 82.63 | 54.91 |
| Intermediate risk (%) | 46.19 | 29.71 | 27.01 | 30.99 | 5.06 | 9.76 | 45.52 | 16.09 | 24.82 |
| High risk (%) | 2.82 | 68.15 | 1.52 | 6.45 | 0.52 | 1.52 | 28.7 | 1.29 | 20.27 |

# Useful to Understand the Time Course of Ischemia and Impacts on Diagnostic Test Results



# Use your Duke TM scores-- if you have them

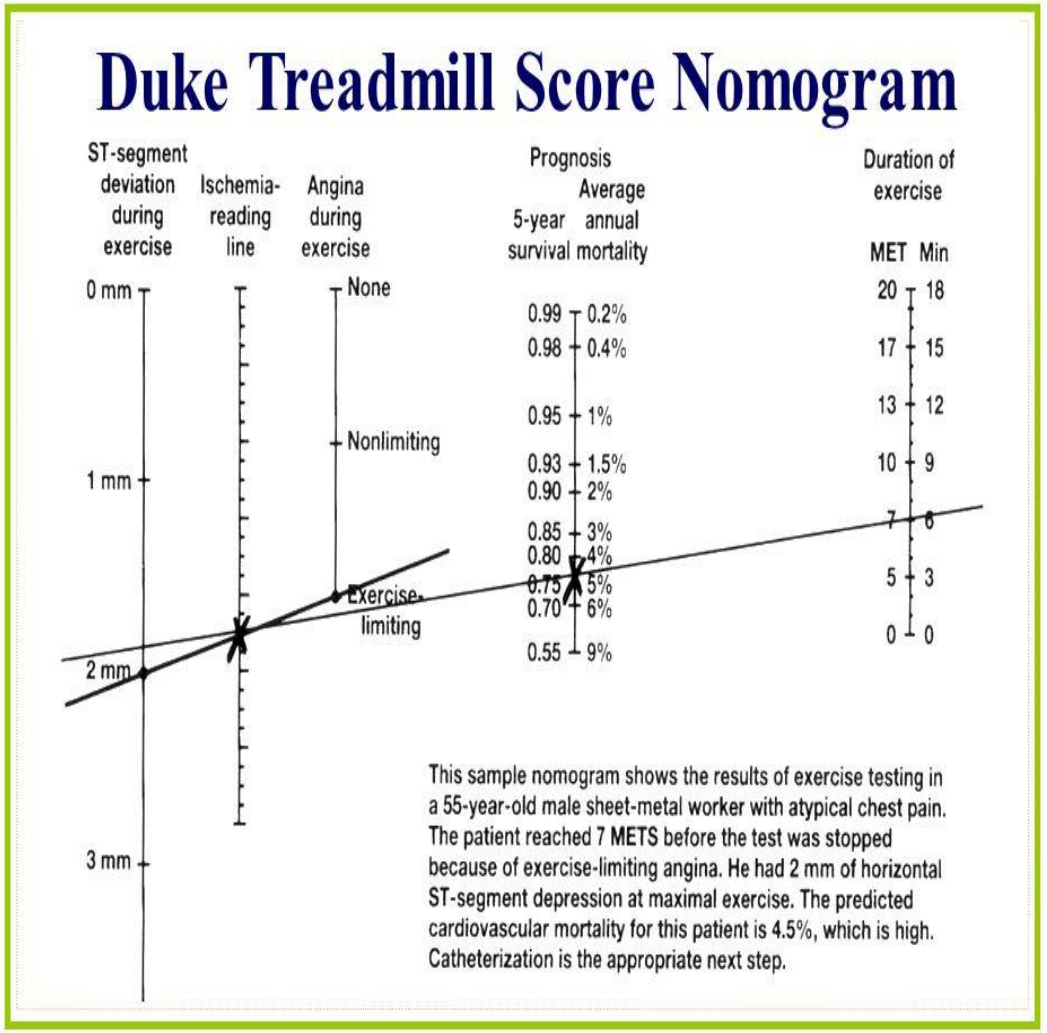
The **Duke treadmill score (DTS)** is calculated from three exercise parameters, where:

- **Exercise time** is based on minutes completed on Bruce protocol (or equivalent to Bruce protocol)
- **ST deviation** is the maximum deviation (in mm) compared with baseline
- **Angina score**: 0 for no pain, 1 for nonlimiting pain, 2 for exercise limiting pain

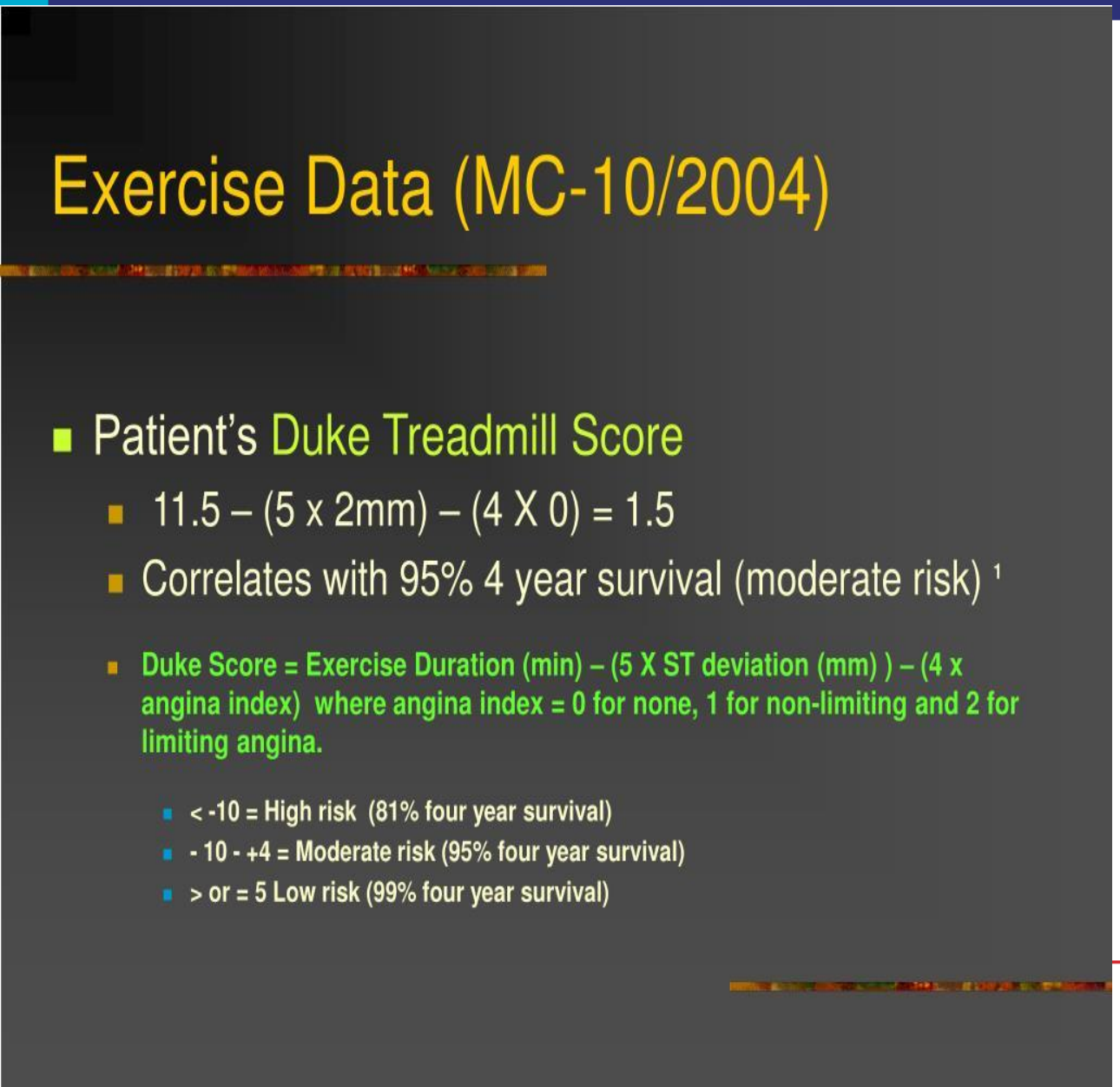
**DTS = Exercise time (minutes) - (5 x ST deviation) - (4 x angina score)**

Patients are classified as low, moderate, or high risk according to the score:

- Low risk – **score ≥+5**
- Moderate risk – **score from -10 to +4**
- High risk – **score ≤-11**



# Duke TM scores Plus Resting Echoes and Annual Mortality % Risks




- **Low scores >/= +5**
  - 1—nml. rest echo--**0.7%**
  - 2—echo w/sgl. territory disease--**1.8%**
  - 3—echo with mult. territory disease--**3%**
- **Moderate scores -10--+4**
  - 1—nml. rest echo--**2.4%**
  - 2—echo w/sgl. territory disease--**3.7%**
  - 3—echo with mult. territory disease--**7%**
- **High Risk scores </= -11**
  - 1—nml. rest echo--**4.6%**
  - 2—echo w/sgl. territory disease--**5%**
  - 3—echo with mult. territory disease--**12%**

# Part 1—Case 1a—CAD Risk calculation with traditional risks and tests—what can help you?

Case 1a—39 yo **F** with smoking, SOB with exertion, creat 1.6, father first MI at 50yo, lipids TC 250 and HDL 40; she appl. for $1M. Term life insurance—**What if this is all you had?**

She has a **Bruce protocol TM** to 7 minutes with peak HR getting to 76% predicted for age, SOB but no CP, and 1.5 mm ST upsloping depression in leads 2,3 and avF and V5-6 at peak—lasting 4 min. into recovery and with "some" PVCs in recovery

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She had a **resting echo** with LVEF of 48% and an apical area hypokinesis-- but **" poor visualization"**

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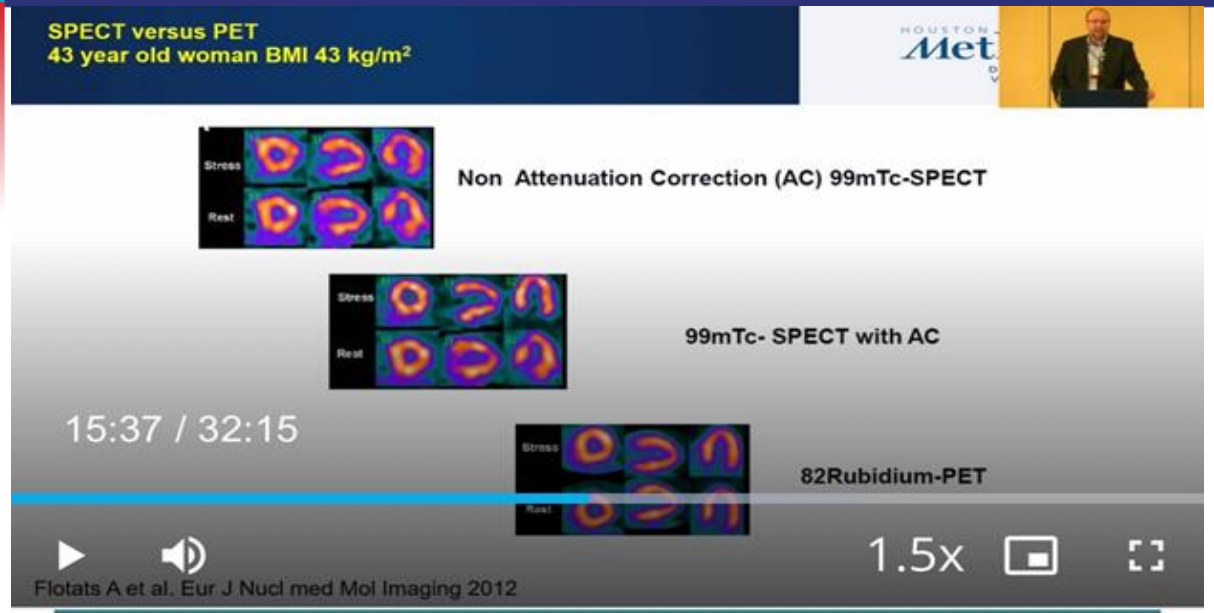
# Because of findings, she gets a nuclear stress test—which kind? Does it help you assess her risk?




EMAIL MOUAZ

Cardiac PET and CTA in the Diagnosis of Coronary Artery Disease in 2025

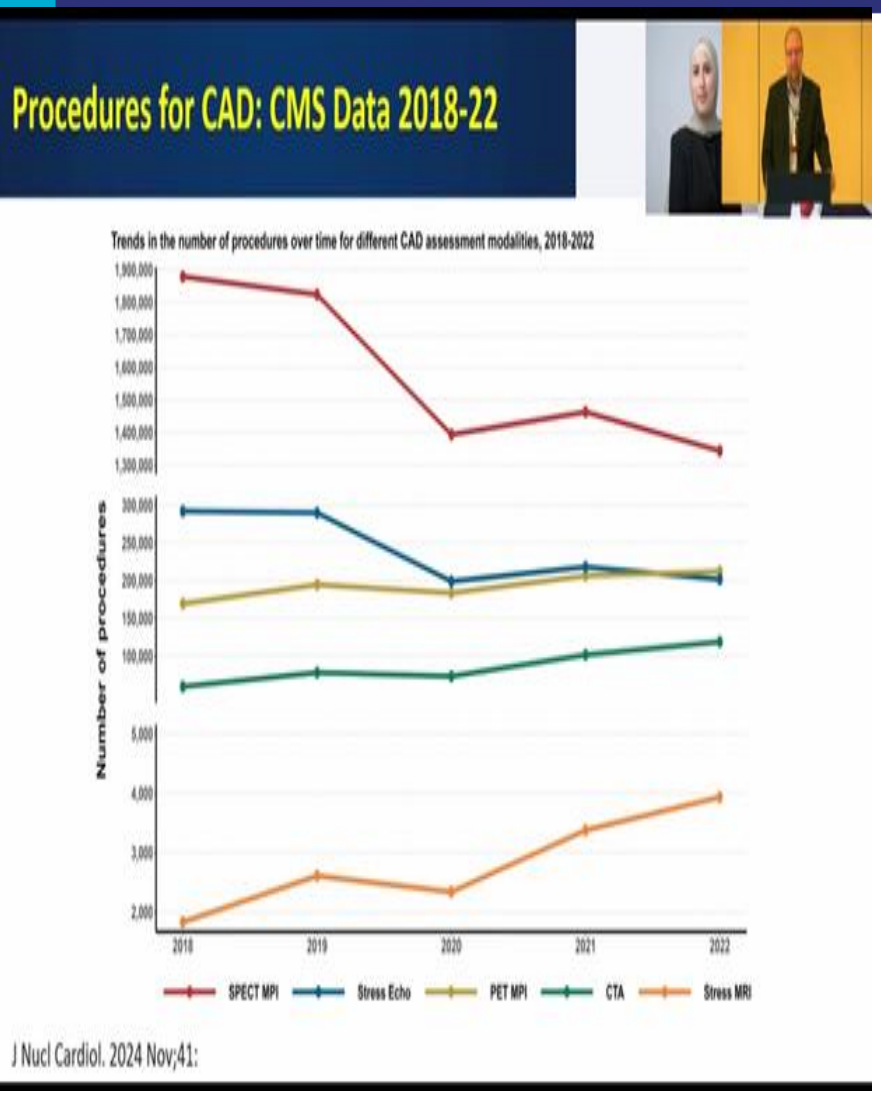
By NORTH FLORIDA CARDIOVASCULAR SYMPOSIUM
FEATURING MOUAZ AL-MALLAH

145 views • July 4, 2025

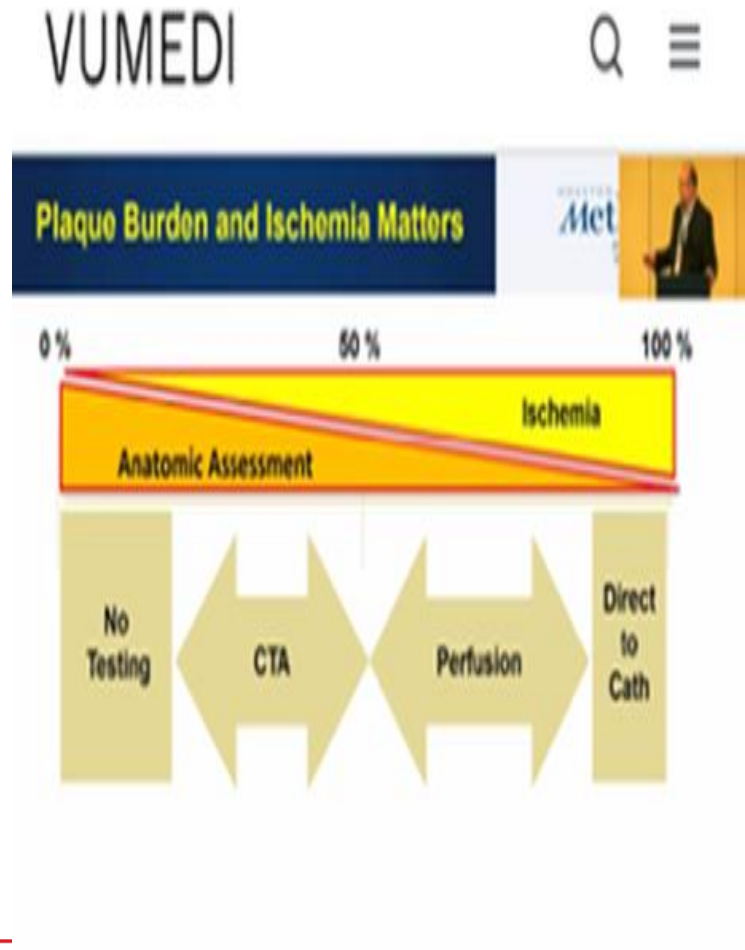
# Things to Look for in Nuclear TMs

- 1—Was this a chemical stress test with Lexiscan (regadenoson) or an exercise test?
- 2—Is attenuation correction used?
- 3—If inferior abnormality, is pt. moved into prone position and reimaged immediately to "see if the defect moves with the diaphragm" instead of being true ischemia or infarct?
- 4—Is quantitation used such as SSS, SRS and SDS?
- 5—Rubidium PET instead of SPECT with thallium or technetium esp. in pts. with high BMI's

# Earlier/Newer Testing—Rapid Changes are Coming




- **Stress nuclear TMs and Stress echoes are decreasing**
- **PET nuclears and CTAs and particularly Stress MRIs are increasing—esp. MBFR (myocardial blood flow reserve)**
- **CTAs used in asymptomatic pts. for screening and Med. Rx mgmt.**
- **CTAs used in ERs as "first test" and identifies diff. "pt. buckets"–**
  - **No dis.**
  - **Mild dis.**
  - **Focal severe dis.**
  - **Complex diffuse dis.**




# Are they really using these? (Note Medicare Data)

## Access to Cardiac PET in the USA

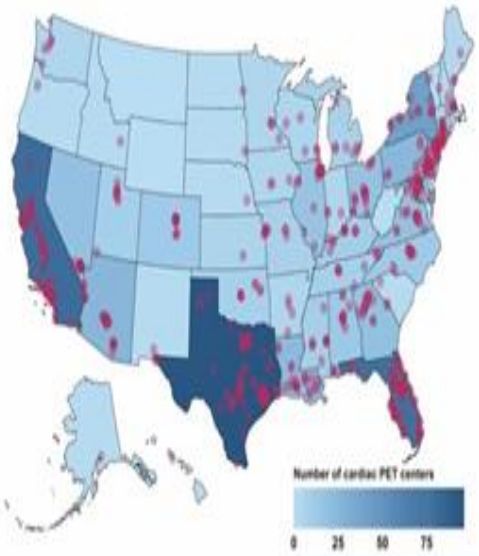
Medicare Data 2021

Geographic Proximity to Cardiac Positron Emission Tomography Facilities Across the United States

Article in Press. Corrected Proof

Ahmed Sayed MBBS and Mouaz Al-Mallah MD

JACC: Cardiovascular Imaging. Copyright © 2024



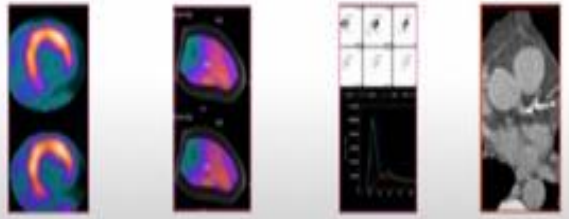

660 unique facilities offered cardiac PET in the US in 2021

Sayyed, Al-Mallah JACC Cardiovasc Imaging. 2024 Aug

## Cardiac PET

1. High diagnostic accuracy
2. Consistent high-quality images
3. Low radiation exposure
4. Short acquisition protocols
5. **Quantification of myocardial blood flow**
6. Strong prognostic power

Relative Perfusion | Rest Stress Gating | Absolute Perfusion | Calcium Score



16:10 / 32:15

1.5x

Al-Mallah, M et al., J Nucl Cardiol 2010;17:498–513

# CAD Test Choices in Dx "Significant" CAD

| Test | Sensitivity | Specificity |
|---|---|---|
| CCTA | 90% | 39% |
| FFR on CCTA | 90% | 71% |
| CV MRI | 90% | 94% |
| SPECT TM | 70% | 79% |
| SE | 77% | 75% |

- Metanalysis 23 studies from 2002-2017
- As compared to gold standard of FFR done invasively in coronary cath lab
- Am Heart J vol. 38, Issue 13, 1 Apr 2017

# Isolated CACS scores—still worth it? Patient subdivisions

Age — CAC is an independent predictor of long-term risk—esp. asymptomatic
- Rotterdam—mean age 69 and Framingham 10-20% risk group--> 52% reclassify high if CACS > 615 or low if CACS score < 50—f/up 9 yrs.

- MESA—CACS works better than Age
- Don't use CACS to "de-risk" younger pt.s with any unexpected plaques

- Females
- Females have less CAD risk than males with = number of risk factors, but CACS still reclassifies correctly

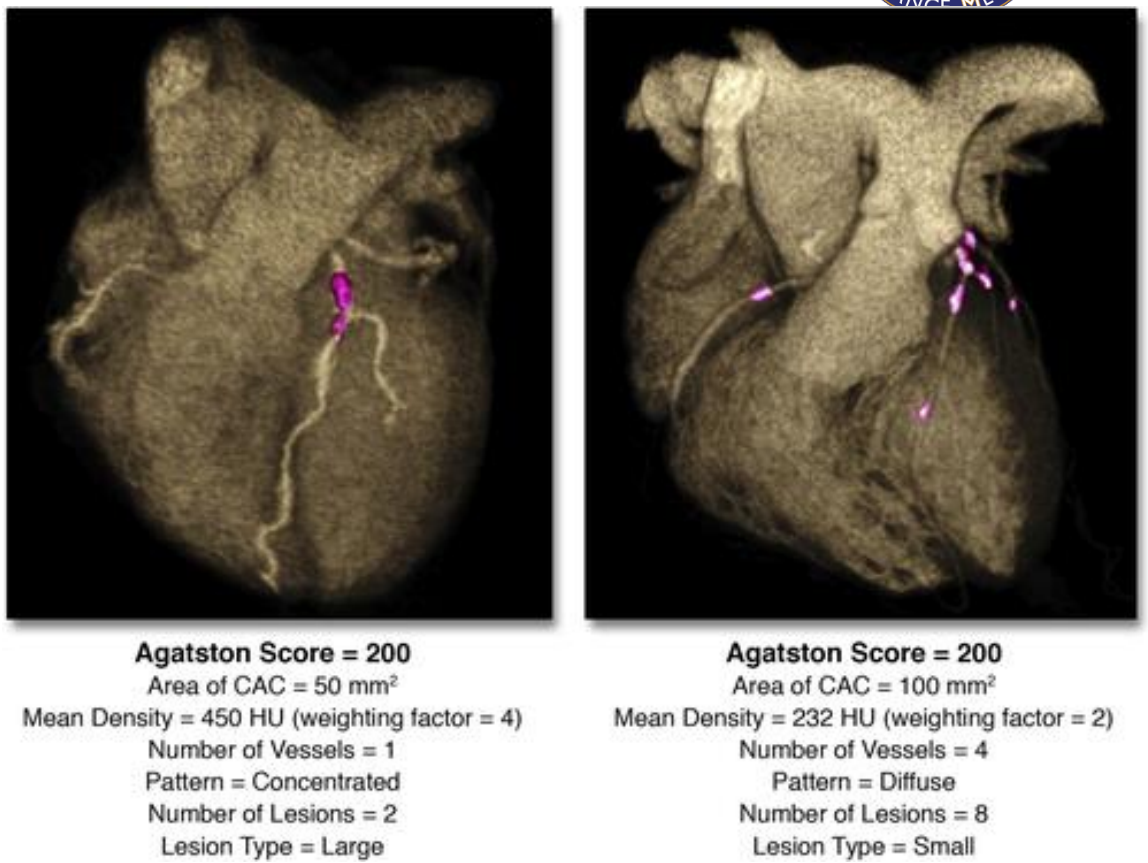
- Metanalysis of 6700 females with 7.5% ten yr. Risk—prevalence of 36% but if CACS> 0 doubled the risk over 7-12 yrs. and CACS reclassifed 20% of all females

- Diabetics
- Study of 22,000 DM pt.s--what age to get first one to make predictive difference?
  - 1—Males w/DM—age 35-38
  - 2—Males w/o DM—age 41-44
  - 3—Females w/DM--age 49-52
  - 4—Females w/o DM—age 56-60

- DM pt.s have more powerful silent plaque progression than other risk factors in pt.s
  - 1—DM pt.s have CAD 6.4 yrs earlier than non DM

2—Smoking, high BP, dyslipidemia, Pos. Fam Hx have CAC 0.3-4.3 yrs earlier than nonsmokers

3—Score of 300+ is equal to prior ASCVD event in terms of risk



**Agatston Score = 200**
Area of CAC = 50 mm²
Mean Density = 450 HU (weighting factor = 4)
Number of Vessels = 1
Pattern = Concentrated
Number of Lesions = 2
Lesion Type = Large

**Agatston Score = 200**
Area of CAC = 100 mm²
Mean Density = 232 HU (weighting factor = 2)
Number of Vessels = 4
Pattern = Diffuse
Number of Lesions = 8
Lesion Type = Small

**But sometimes the CACS number is not enough**

Michael J. Blaha et al. *J Am Coll Cardiol Img* 2017; 10:923-937.

# Part 2—Case 2—CAD Risk calculation with calculator, new tests and mild plaque—what can help you?

Case 2—45 yo M with GERD, on/off smoking, SOB, creat 1.6, father first MI at 52, high bp, BMI> 37, alcohol ++, noncompliance, declines statins re: myalgias, chol 178, TG317, HDL 27,LDL88, applied $1M. Term life

ASCVD Risk Estimator +

ASCVD Risk Estimator Plus at tools.acc.org-->

**Current ten year risk = 4.4%**

**CACS shows one 40% mLAD plaque**

**CCTA with FFR analysis shows distal LAD and diag. have FFR of 0.76** (i.e. less than a 0.80) = they decided no intervention

**Old GL's left statin up in air for shared decisions/**taken off ASA/started on Norvasc

**One month later→ acute ant. STEMI**

He had not taken Rxs—"he thought his scan was ok"

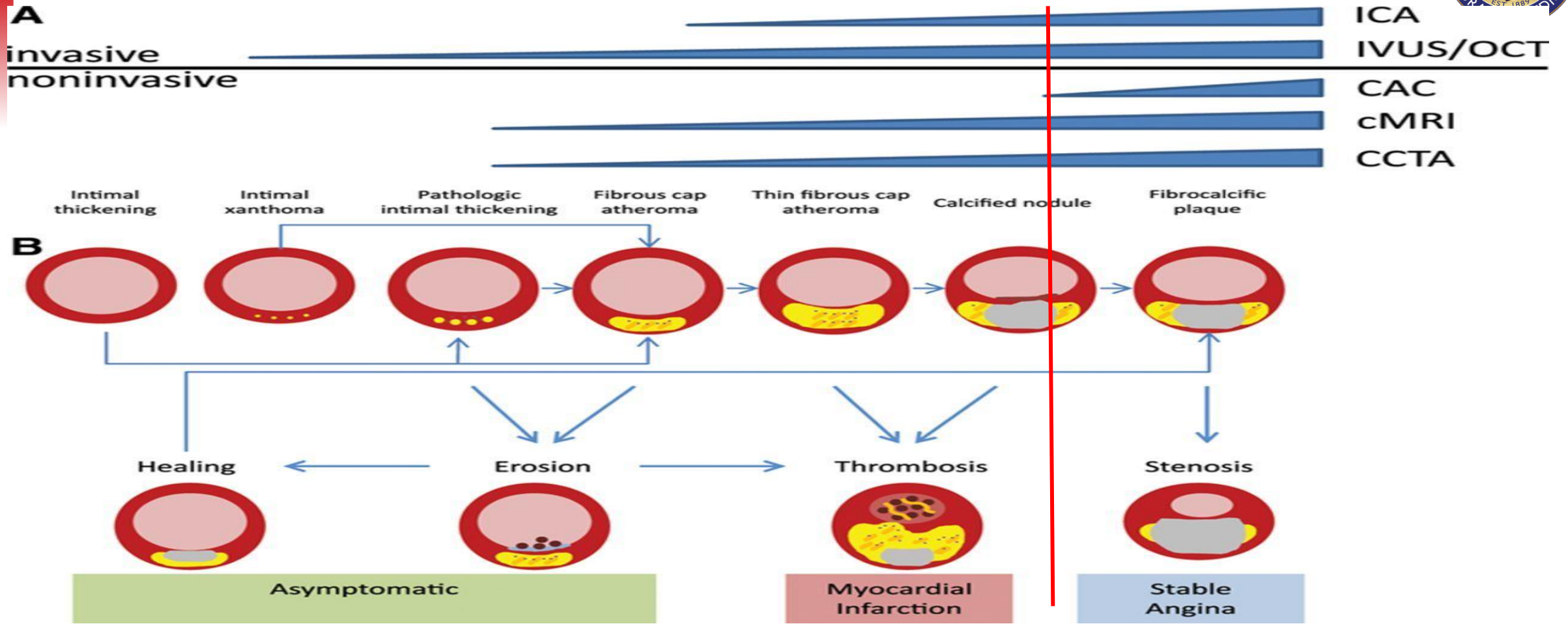
His post PCI status = LAD open but large ant. scar, lower LVEF, on Entresto and SGLP-1 meds and now a "committed pt."

1—Vote→(if you only had the Risk estimator)
- --Better than std. risk
- --Std. risk
- --Substd. risk
- --Decline

2—Vote→(if you had only the CACS score)
- --Better than std. risk
- --Std. risk
- --Substd. risk
- --Decline

3—Vote→(if had risk estimator, CACS score and FFR)
- --Better than std. risk
- --Std. risk
- --Substd. risk
- --Decline

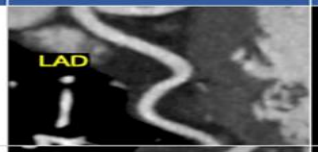
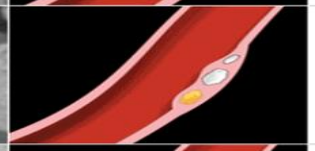
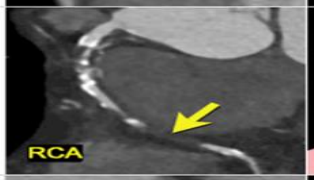
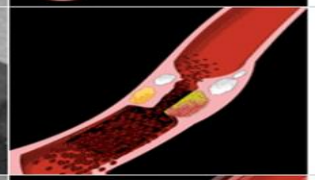
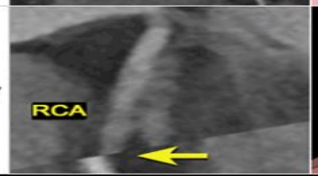
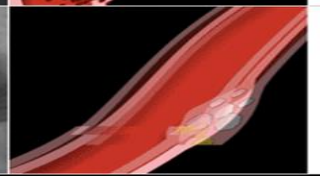
# Look at "how late in time" CAC score finds the risk compared to other tests--> Perhaps it only answers = do I need to go to cath lab soon?



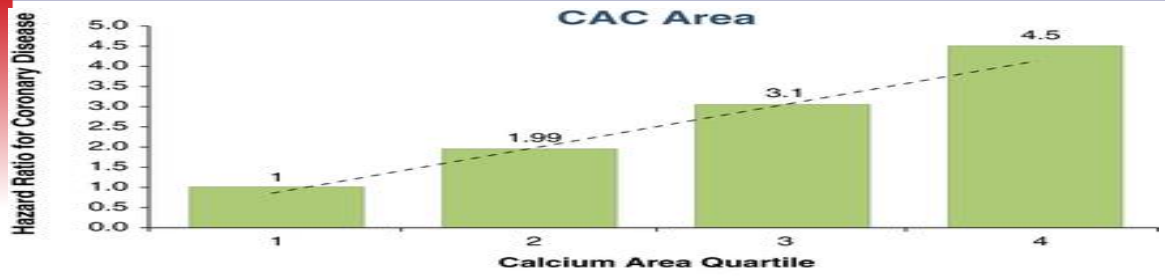

Veit Sandfort. Circulation: Cardiovascular Imaging. Noninvasive Imaging of Atherosclerotic Plaque Progression, Volume: 8, Issue: 7, DOI: (10.1161/CIRCIMAGING.115.003316)

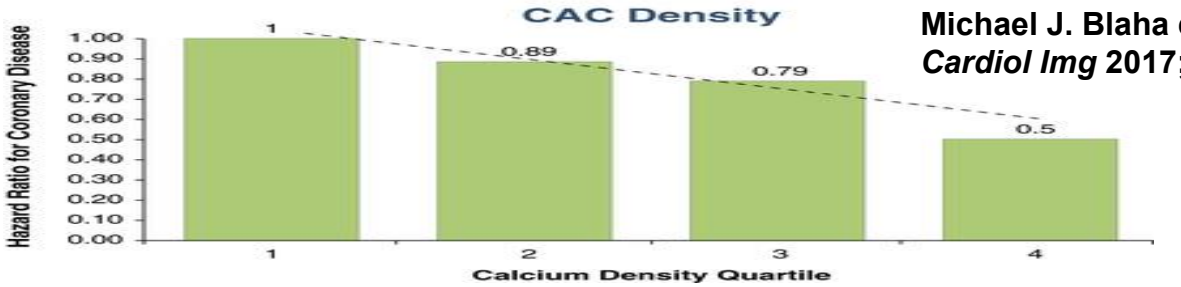
# CCTA vs. coronary angio
# More than the CACS score--> next = RADS score = "single lesion % stenosis"


CAD-RADS0 = no stenosis
CAD-RADS1 = 0-24%
CAD-RADS2 = 25-49%
CAD-RADS3 = 50-69%
CAD-RADS4 = 70%+
CAD-RADS5= 100% occluded

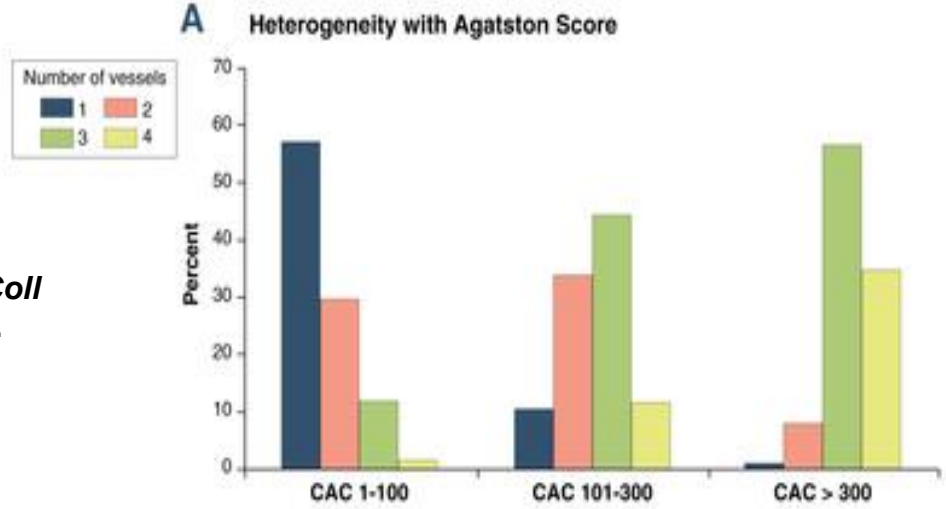
| Cad-Rads | Stenosis | CT imaging | Illustration | Additional Tests |
|---|---|---|---|---|
| Cad-Rads **0** | 0%<br>No stenosis |  |  | None |
| Cad-Rads **1** | 1-24%<br>Minimal stenosis |  |  | None |
| Cad-Rads **2** | 25-49%<br>Mild stenosis |  |  | None |
| Cad-Rads **3** | 50-70%<br>Moderate stenosis |  |  | Consider functional assessment |
| Cad-Rads **4** | A: 70-99% stenosis in 1 or 2 vessels<br>B: >50% stenosis in the left main or >70% stenosis in 3-vessels |  |  | A: Consider functional assessment or ICA<br>B: ICA is recommended |
| Cad-Rads **5** | 100%<br>total occlusion |  |  | ICA and/or viability assessment |
| Cad-Rads **N** | Non-diagnostic study |  |  | Additional evaluation |

# So Maybe Area, Density, Volume, "Diffusivity"-- not just Score



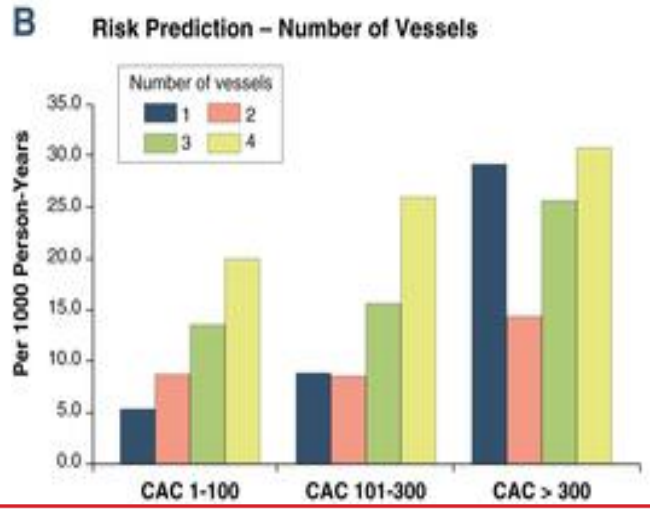



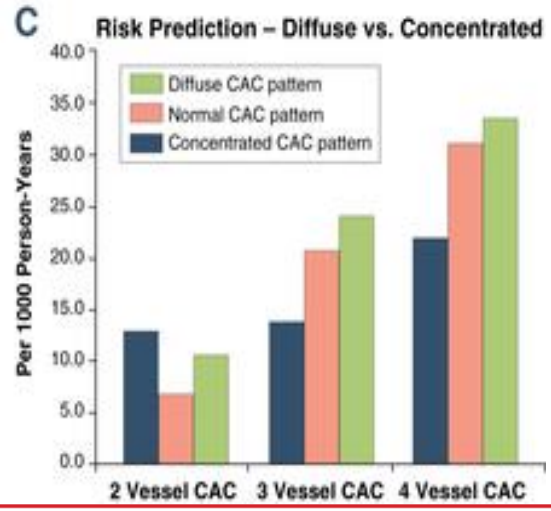

Michael J. Blaha et al. *J Am Coll Cardiol Img* 2017; 10:923-937.




**Increasing vessel involvement and Volume** of plaques signif. increases risk.

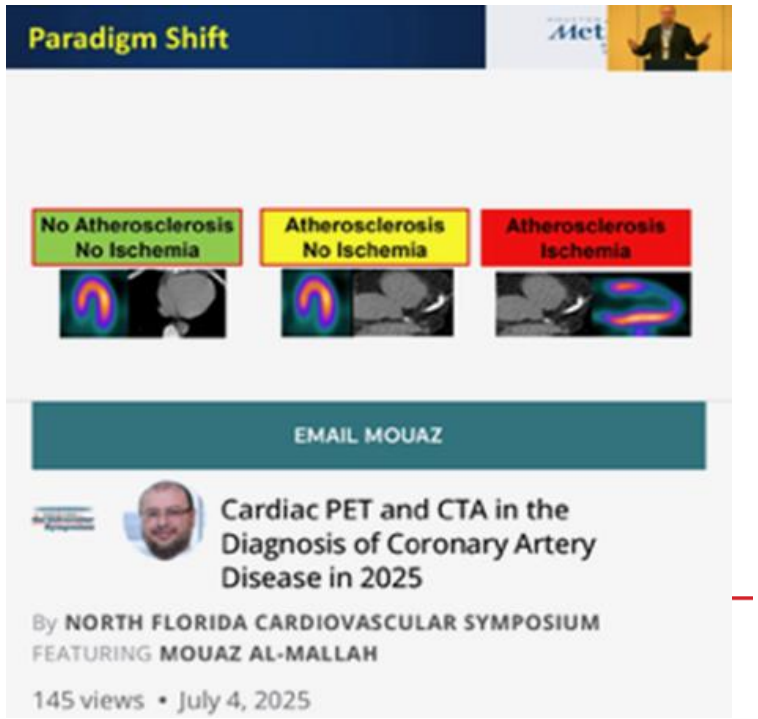
**For every 10% increase in CAC diffusivity, there was a significant 7% increased all-cause mortality risk after adjusting for the Agatston CAC score**. In the MESA study, diffuse CAC, defined as >75% CAC diffusivity, was associated with a **33% increased risk.**

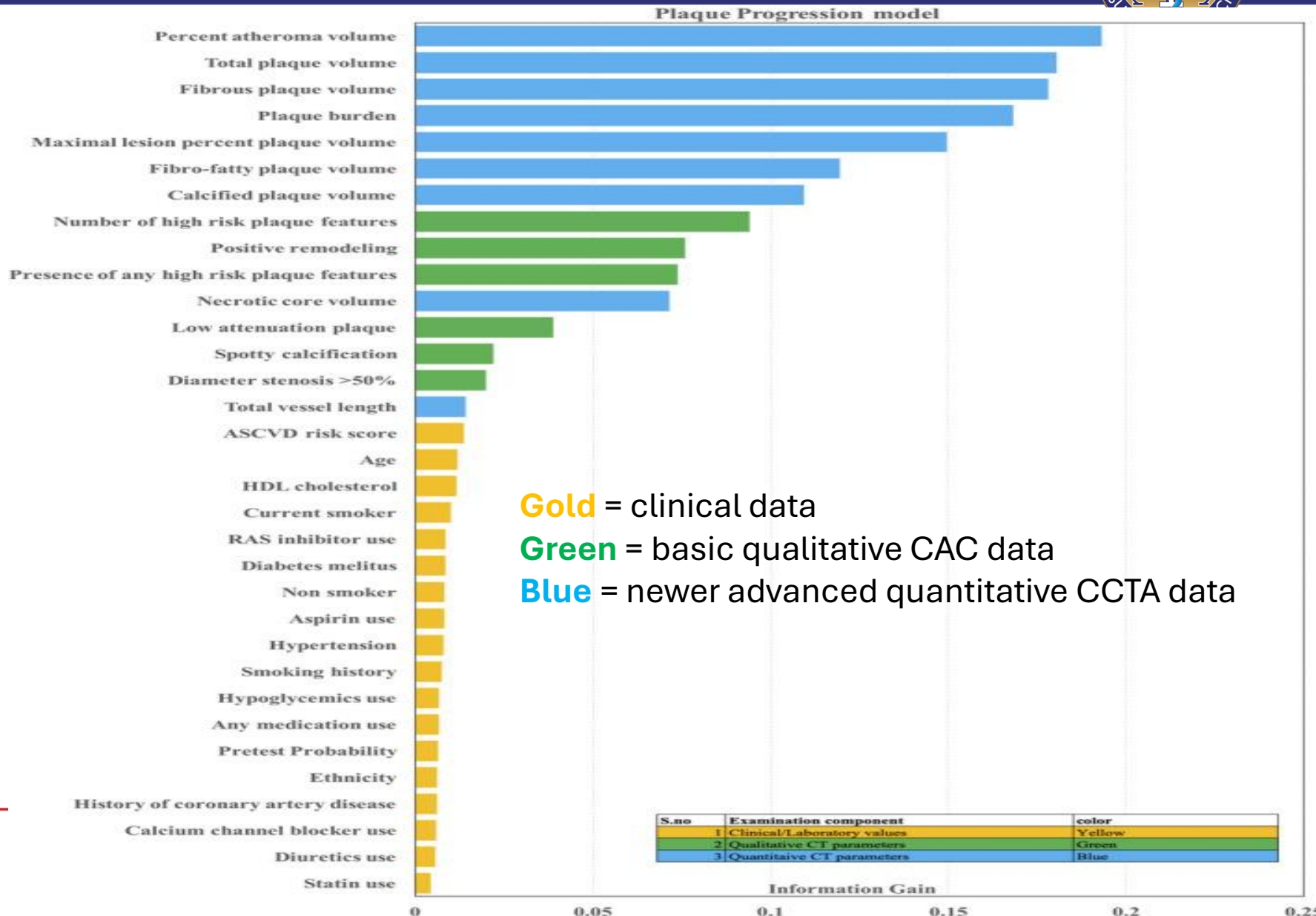


# Old paradigm--> Ischemia present? Yes/No
# New paradigm--> Does pt. have atherosclerosis first and then-- does pt. have ischemia?

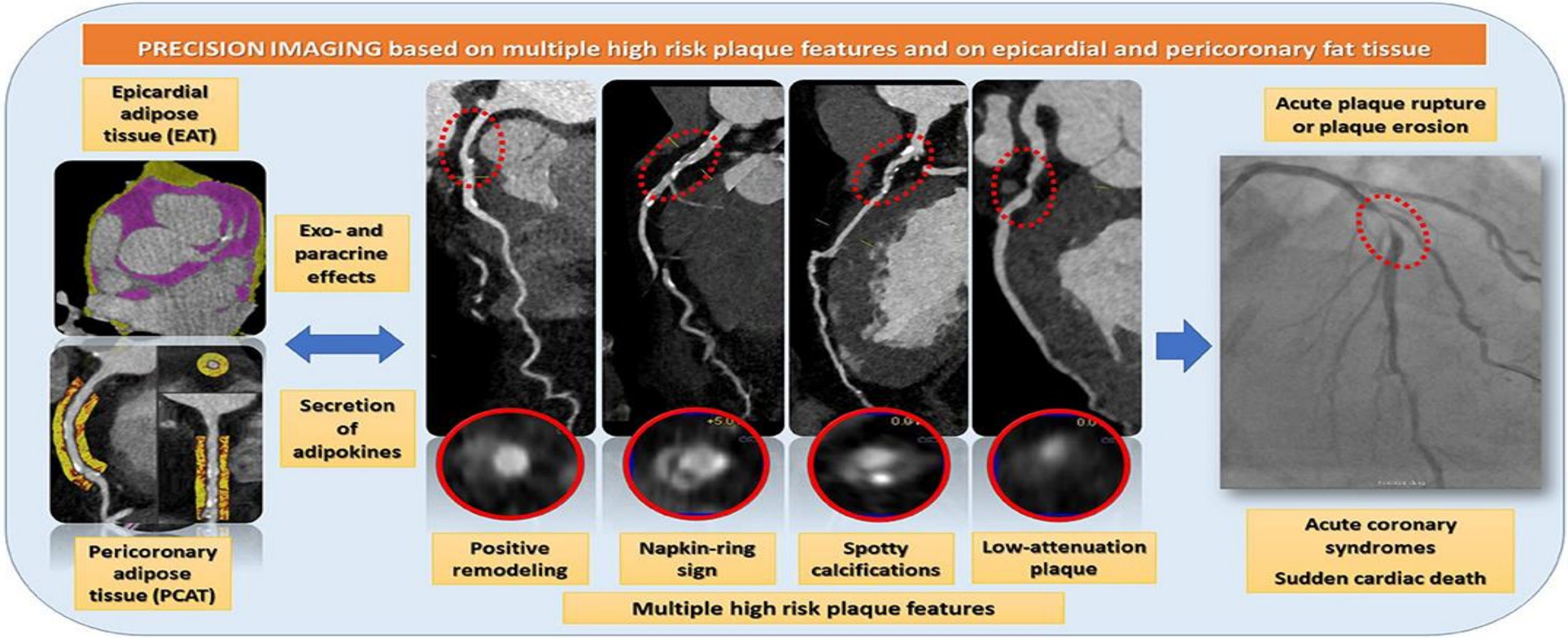
Machine Learning Framework to **Identify Individuals at Risk of Rapid Progression** of Coronary Atherosclerosis: From the PARADIGM Registry | Journal of the American Heart Association



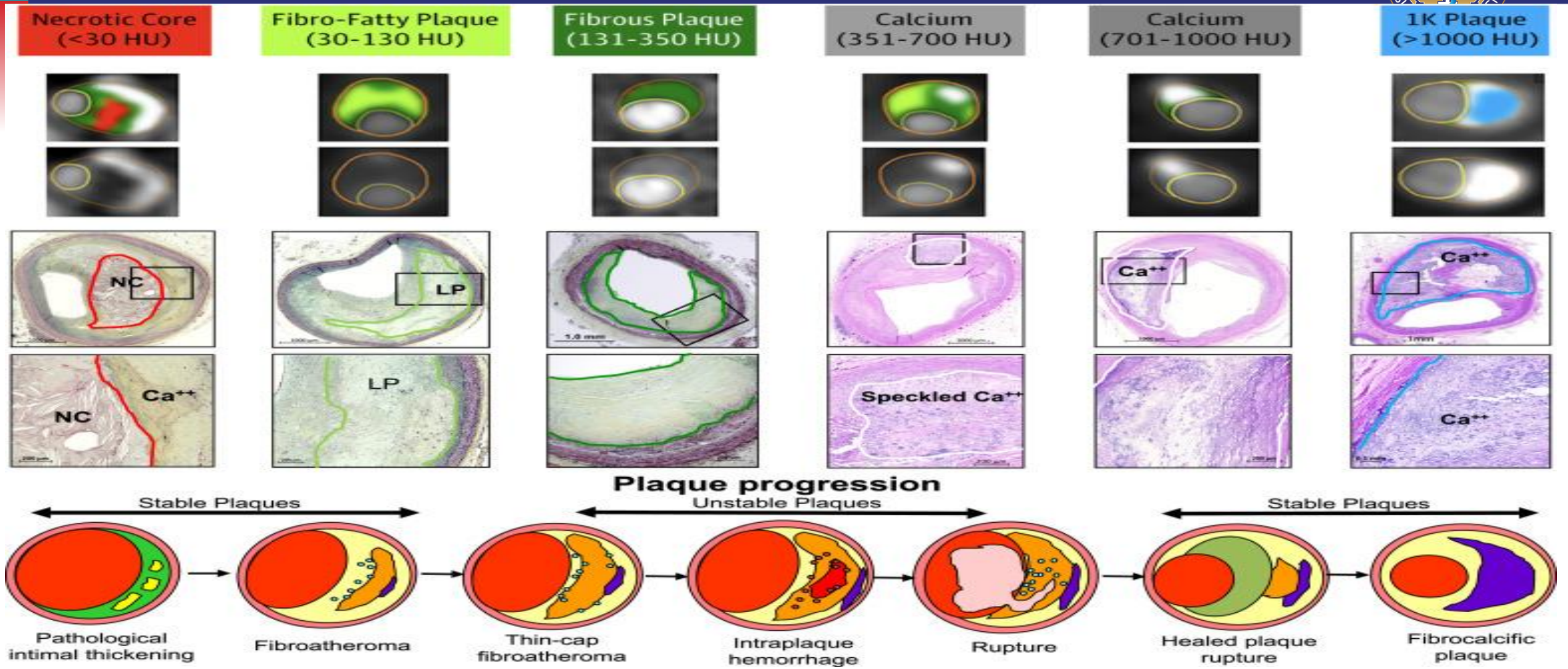




**Gold** = clinical data
**Green** = basic qualitative CAC data
**Blue** = newer advanced quantitative CCTA data

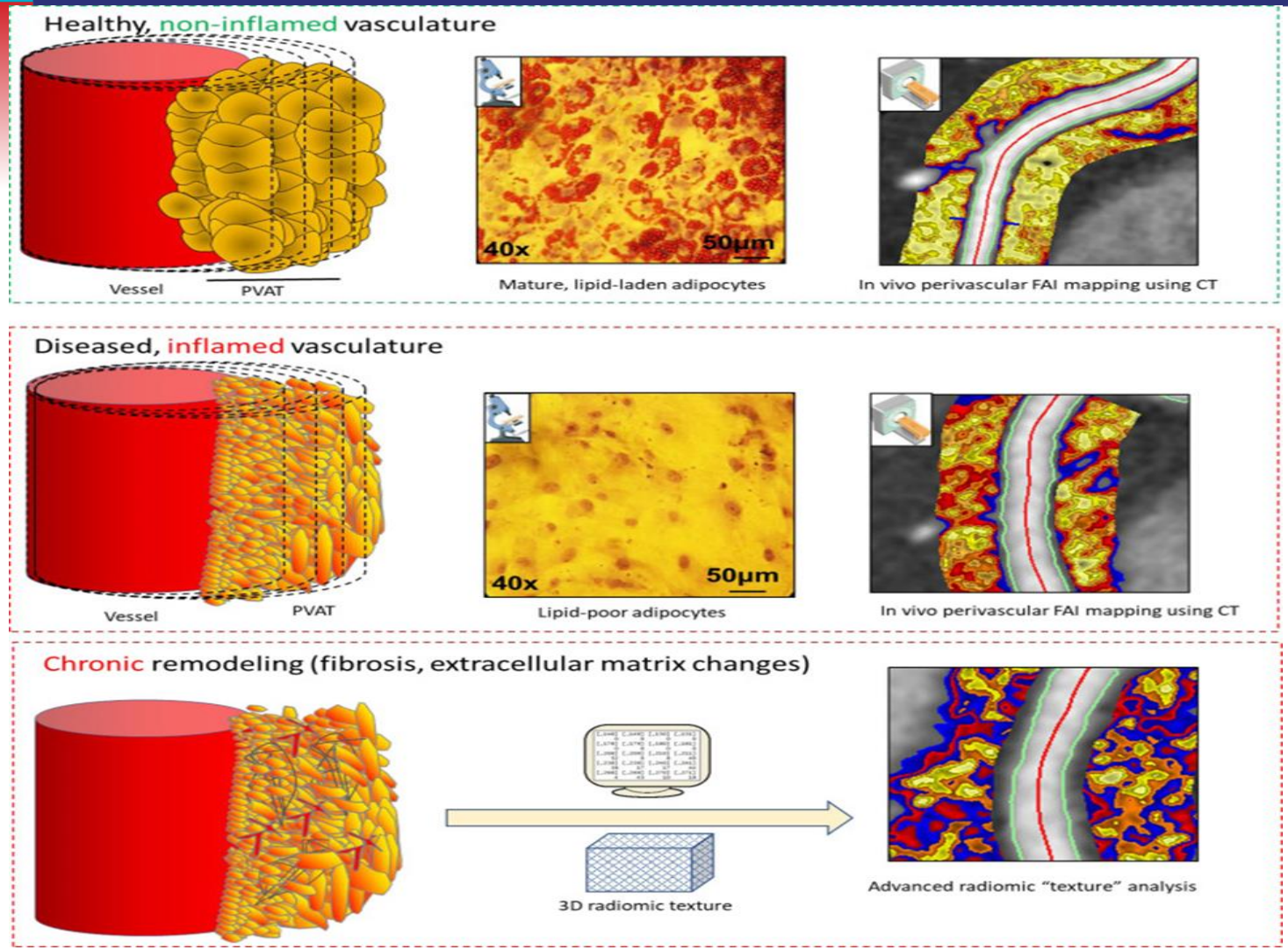
# CCTA--4 "Descriptors" that Add Risk—Also Note Effects of Fat



# Plaque Progressions/Regressions seen on CCTA



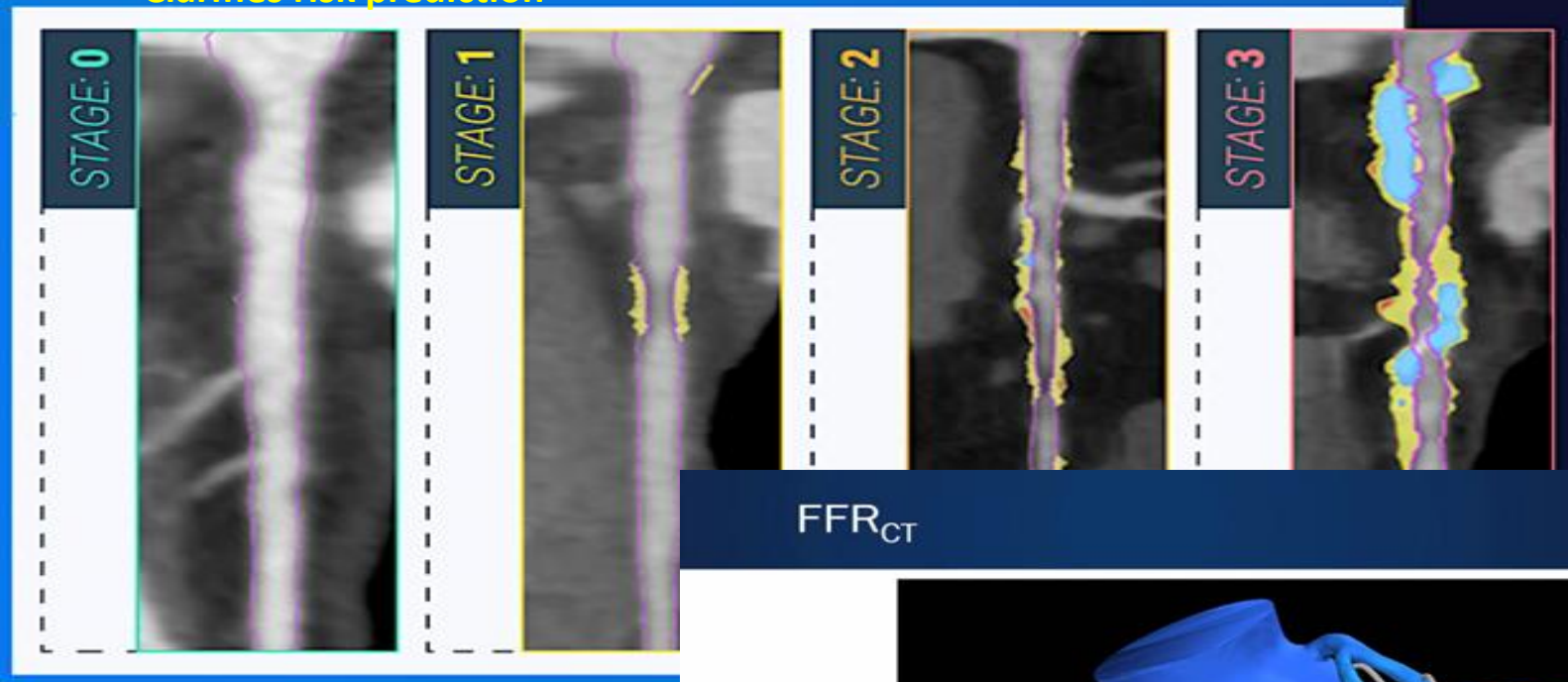
# FAI Scores Add to CACS scores of Risk in ORFAN Study



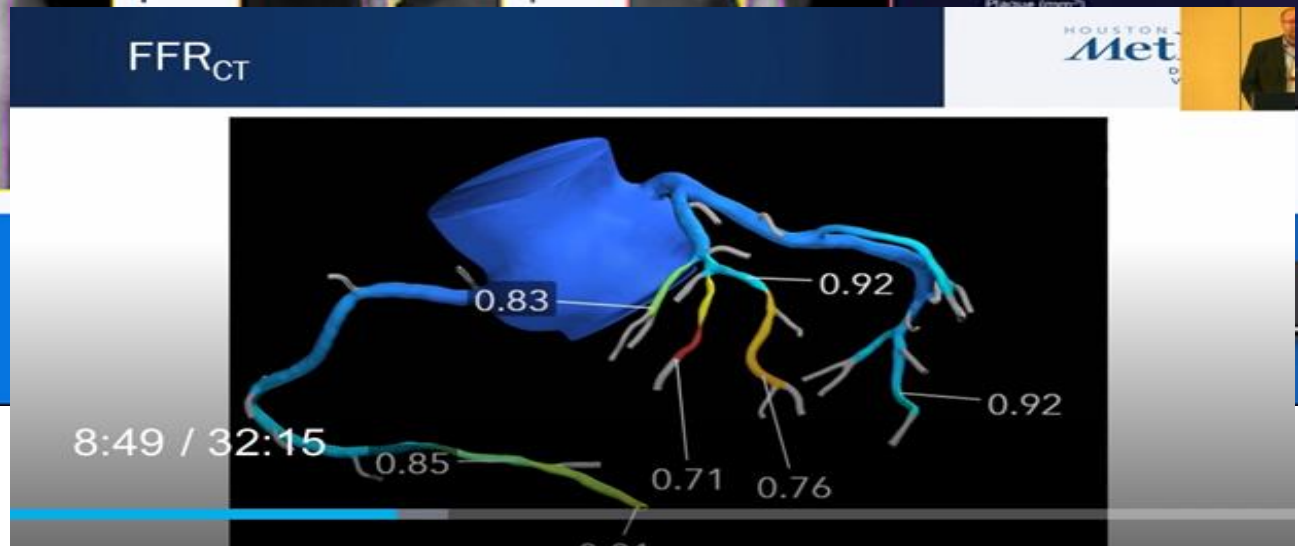

**Results showed that 81.1% (32,533) of patients did not have obstructive CAD and that over the 2.7-year median follow-up they accounted for 66.3% of the total 4,307 MACE and 63.7% of the total cardiac deaths.**

# Example of "Cleerly" images recon from AI on CCTA's

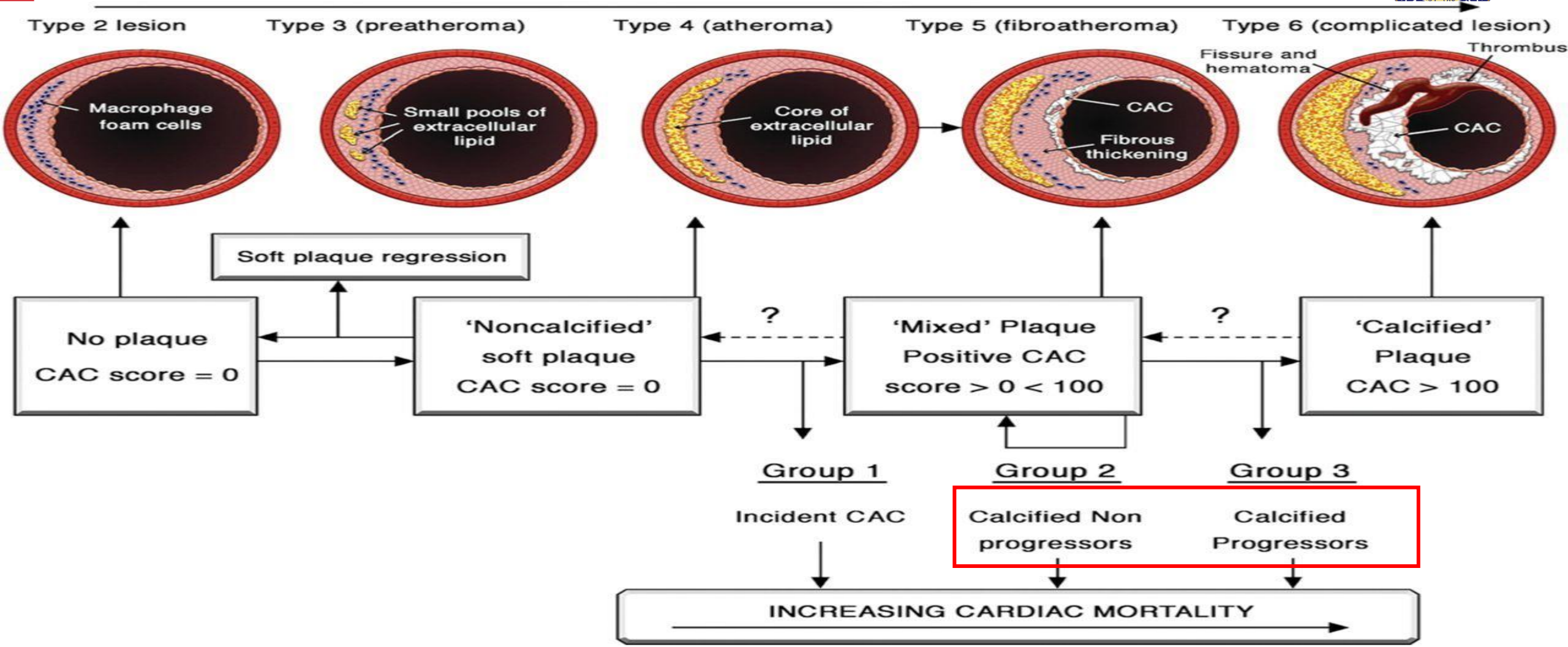
- Analyze/quantify plaque types
- Measures stenosis
- Detects ischemia by flow FFR from CCTA
- Clarifies risk prediction







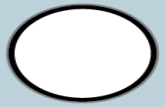
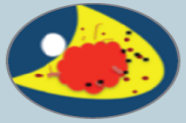
# Can You Medically Impact Plaque Progression?



# Adding Blood Flow/Physiology to CCTA Anatomy-Prevent PCI's?

*JAMA Cardiol.* 2016;1(3):350-357.
doi:10.1001/jamacardio.2016.0263

The FAME 2 trial[3] extended these findings and demonstrated that **deferring PCI in lesions with an abnormal FFR results in high rates of progressive ischemic symptoms, unstable angina, and MI, which require revascularization within 1 to 2 years.** These outcomes could be prevented by PCI.

| Angiographic Diameter Stenosis Severity, % | FFR | No. of Lesions (% in Subgroup) [% in Entire Cohort] | Possible Histologic Feature |
|---|---|---|---|
| Normal | >0.80 | 0 |  |
| 50-70 | >0.80 | 402 (65) [33] |  2FNP with moderate luminal stenosis |
| | ≤0.80 | 218 (35) [18] |  2FPP with moderate luminal stenosis |
| 71-90 | >0.80 | 104 (20) [8] |  2FNP with moderate to severe luminal stenosis |
| | ≤0.80 | 409 (80) [33] |  2FPP with moderate to severe luminal stenosis |
| | | |  2FNP with severe luminal stenosis |

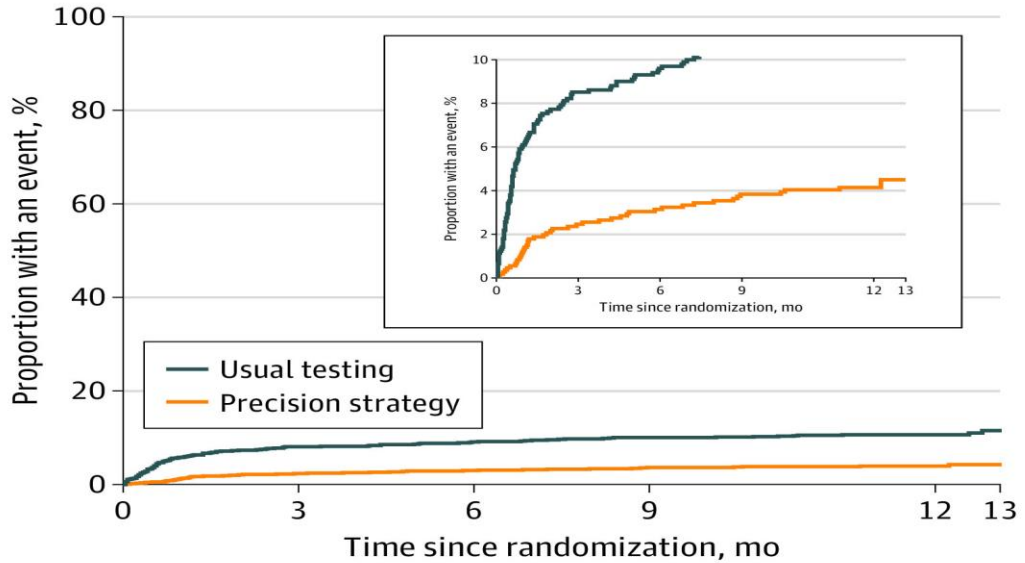
Histological features:
1. Coronary artery lumen
2. Fibrous part of the plaque (entire navy blue area)
3. Necrotic core (entire yellow area) that includes neovascularization (small red lines), red blood cell leakage (red dots), macrophages (black stars), and intraplaque hemorrhage (4)

# PRECISE TRIAL 2023—a "CT First" Pathway-- used CCTA and if 40-90% lesion then had CCTA-FFR done

--Less normal caths in PRECISE group
--Same deaths and MI's in "usual care" group and PRECISE group
--No adverse events in "low risk" untested group
--Usual care group had more caths with minimal disease

2/3 of all CAD pt.s despite knowledge of value of LDL< 70 are actually measured above that—**note in 2022 GL's shifted to "LDL of < 55"—note 100% of "high risk" CAD pts do not reach that target**

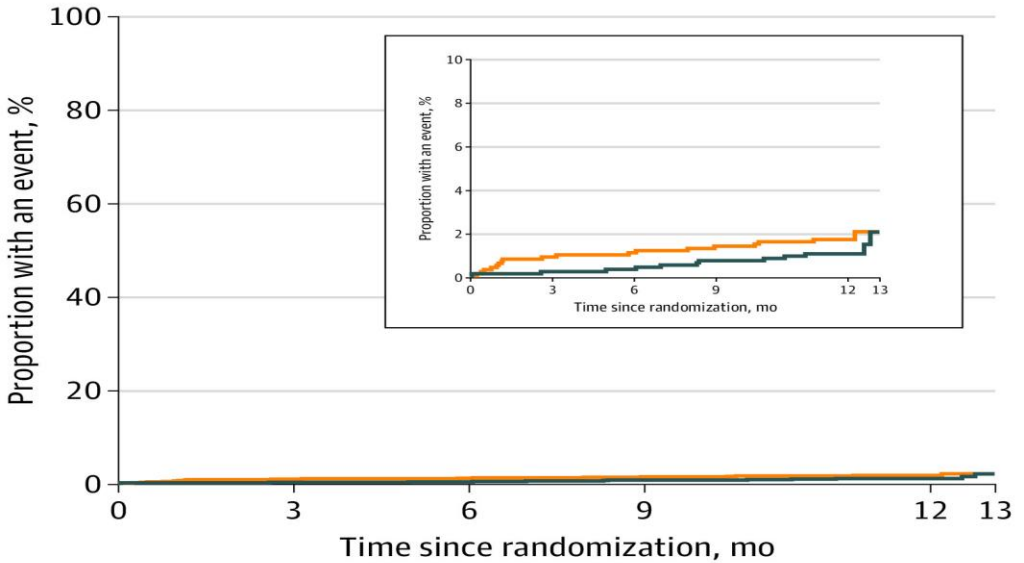
**A** Primary composite end point




No. at risk

| | | | | | | |
|---|---|---|---|---|---|---|
| Precision strategy | 1057 | 997 | 971 | 945 | 431 | 88 |
| Usual testing | 1046 | 922 | 898 | 869 | 421 | 102 |

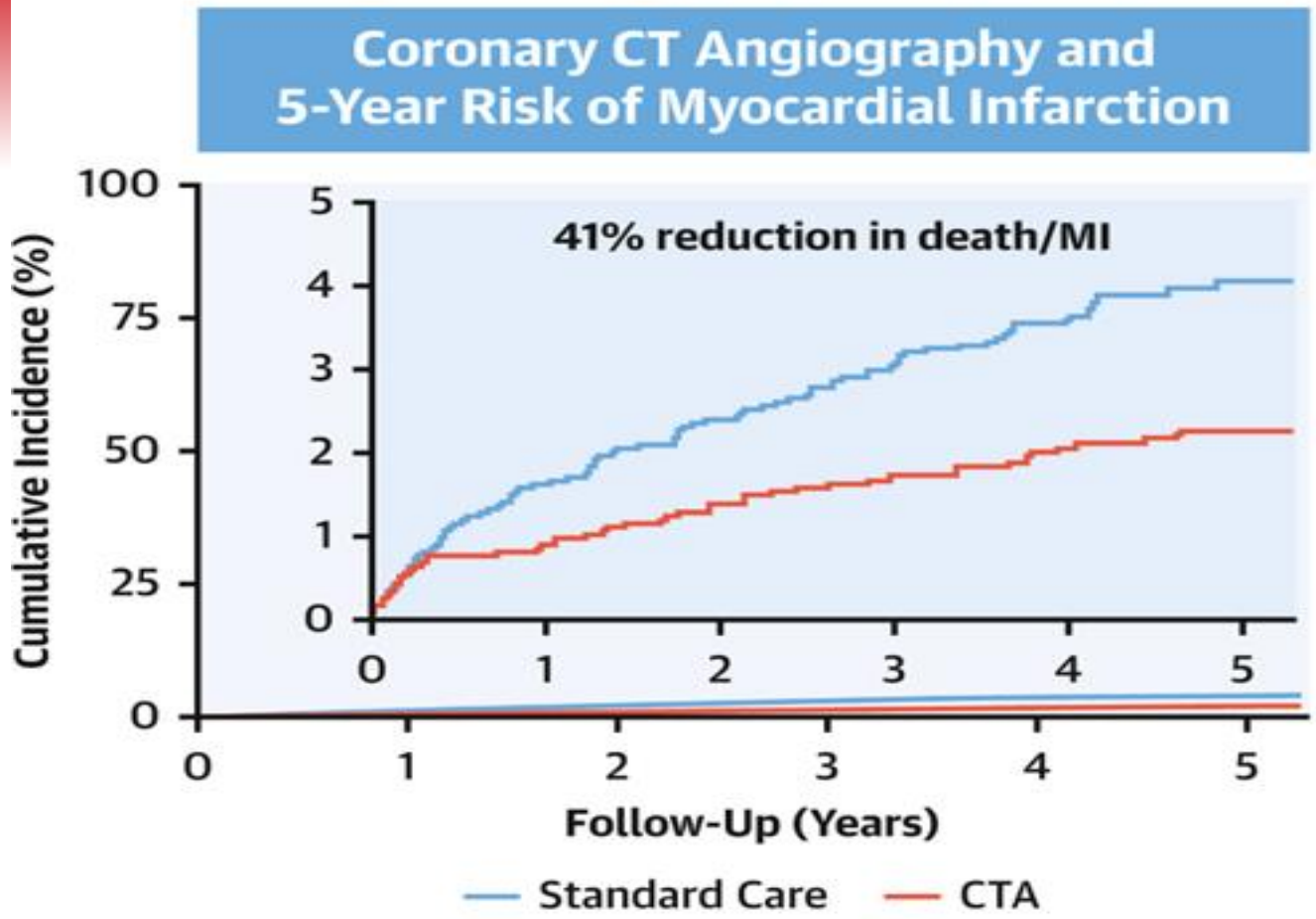
**B** Death or nonfatal MI



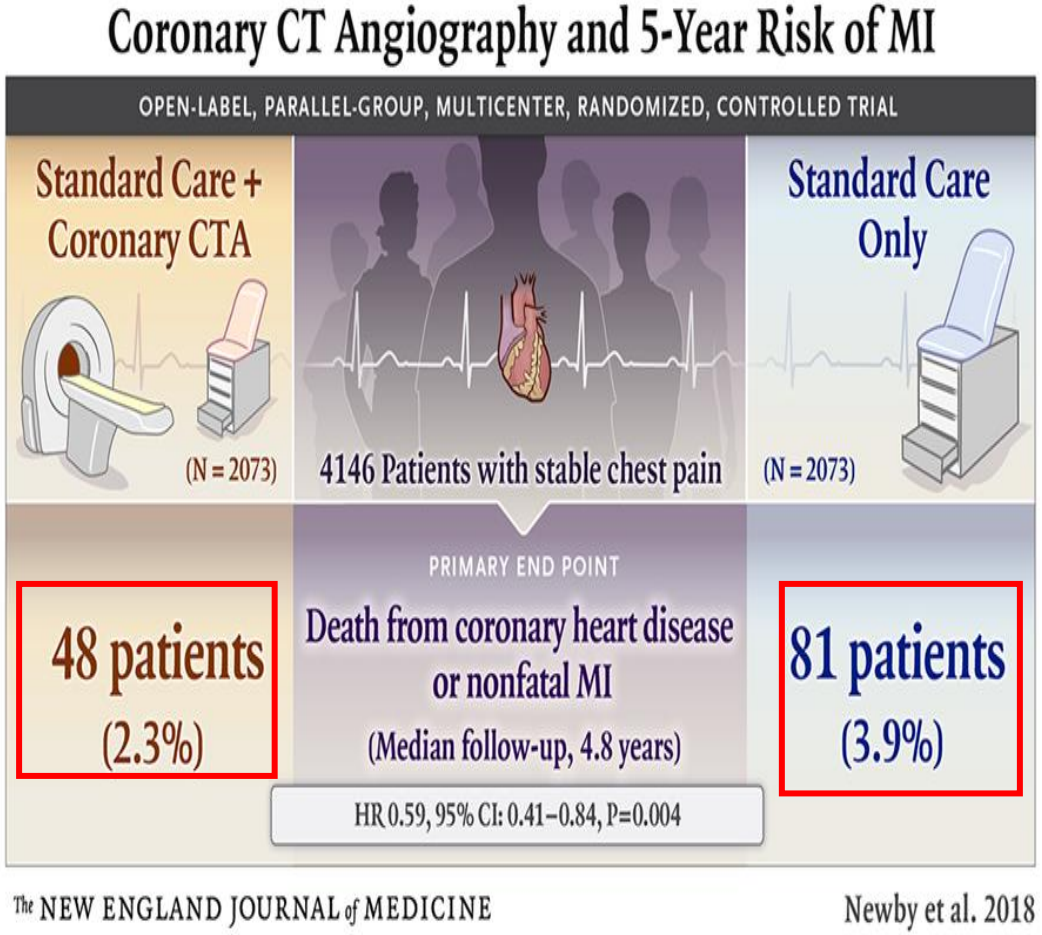

No. at risk

| | | | | | | |
|---|---|---|---|---|---|---|
| Precision strategy | 1057 | 1014 | 993 | 970 | 446 | 89 |
| Usual testing | 1046 | 1009 | 996 | 970 | 468 | 107 |

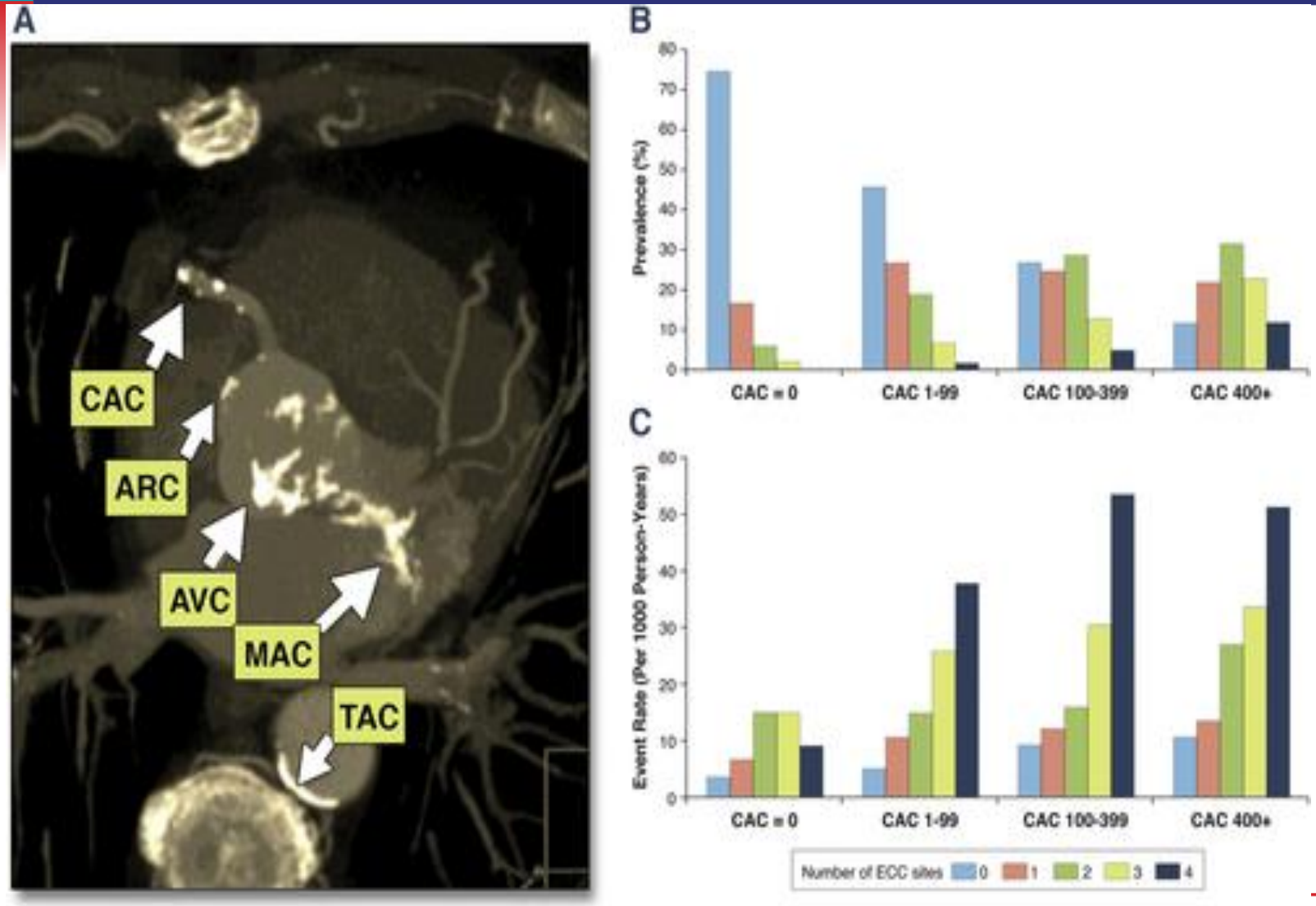
# SCOT-Heart Trials Prospective Atheroma Rx




- 4,146 patients undergoing SOC (n = 2,073) vs. CT (n = 2,073)
- 40% higher preventive therapies in CT arm



# Should we include "outside the coronary" ECC calcifications as part of a score?



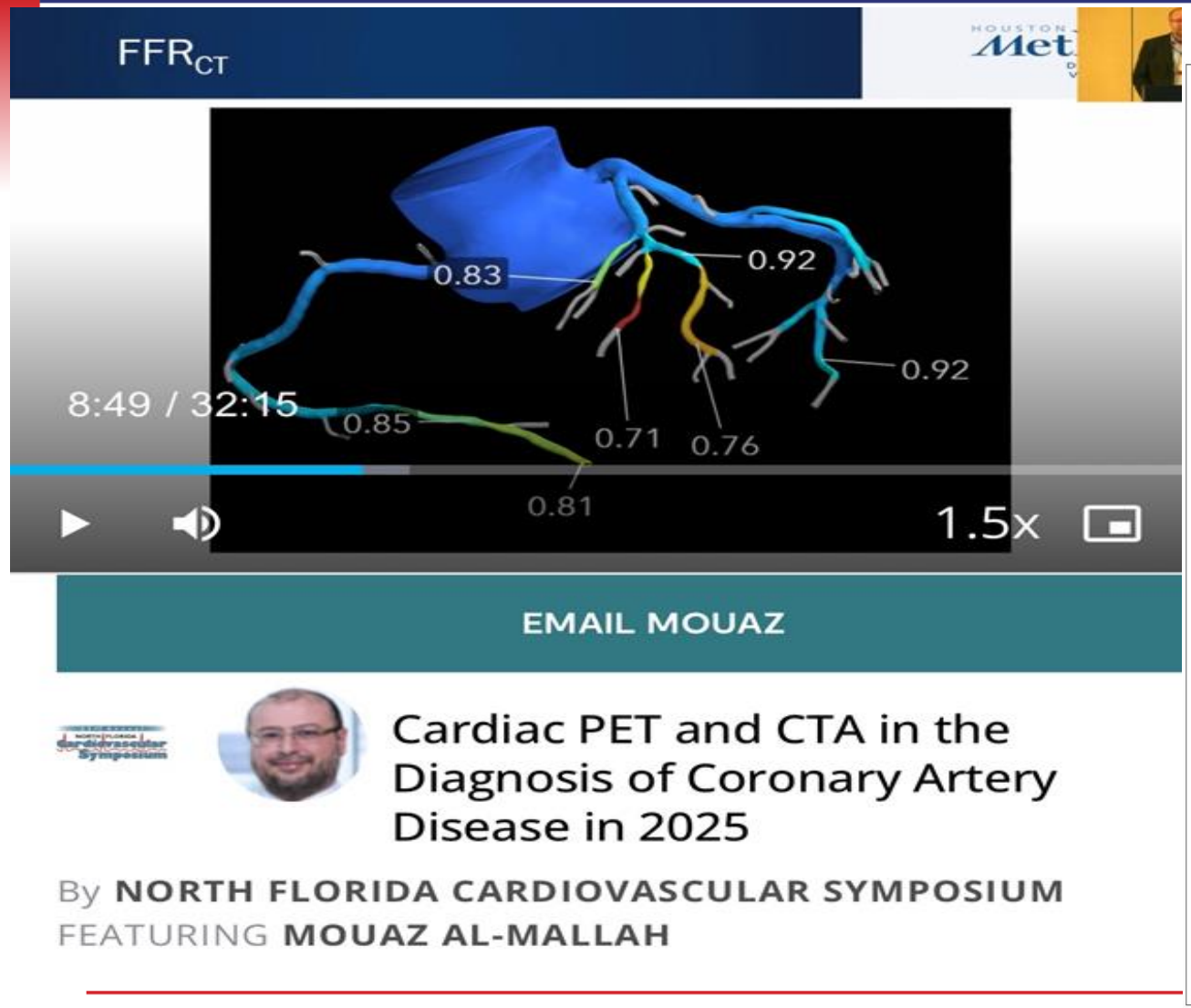

In the MESA study, Tison et al. proposed a **multisite ordinal ECC score**, consisting of a score of 0 to 4 based on cumulative involvement of the **mitral valve, aortic root, aortic valve, and thoracic aorta**.

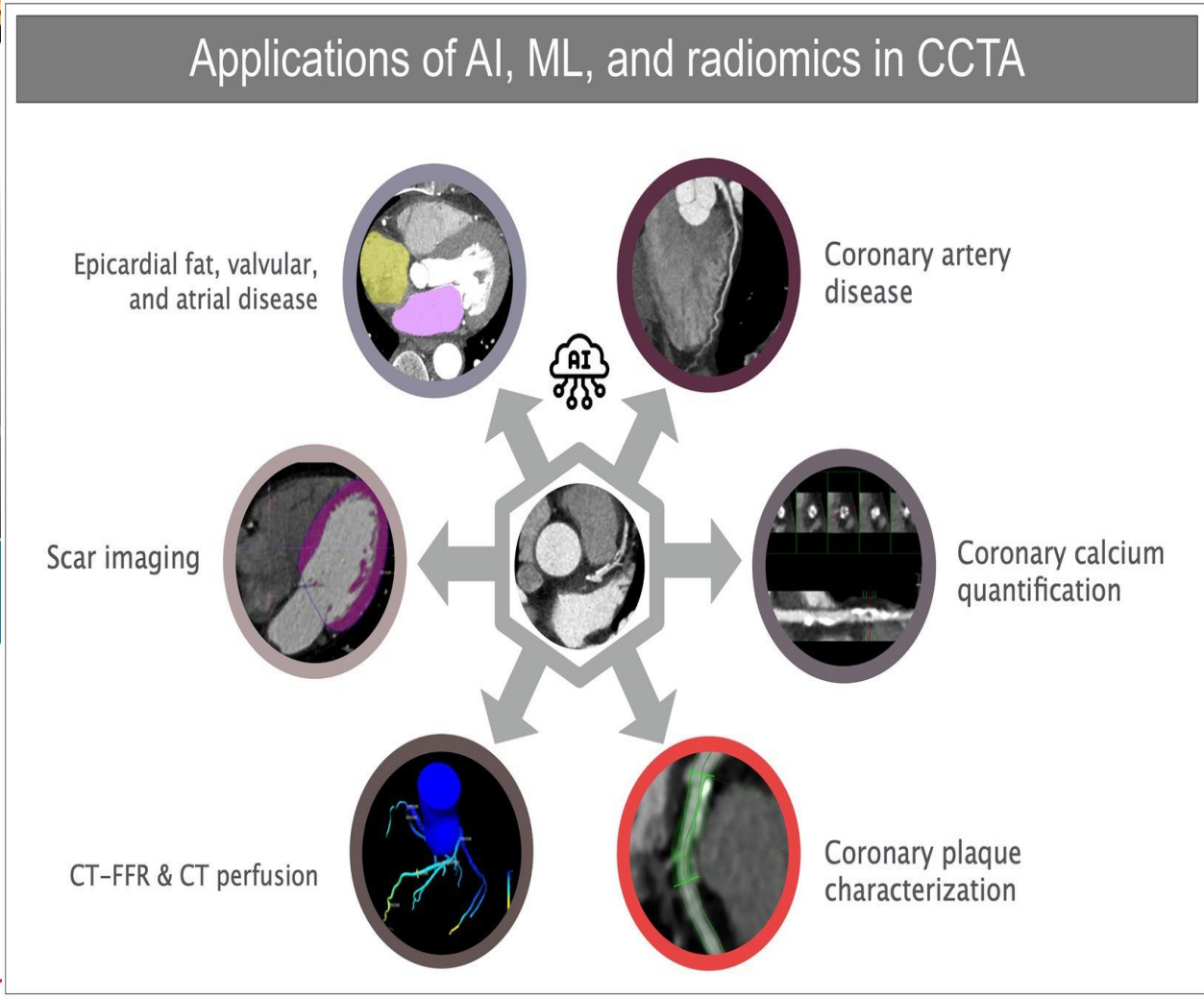
This ordinal score **improved prediction of coronary events, coronary mortality, and total mortality beyond traditional risk factors.**

**After accounting for CAC, the ECC ordinal score remained a significant predictor of total mortality**

Michael J. Blaha et al. *J Am Coll Cardiol Img* 2017; 10:923-937.

# Using AI tools to map coronary flow like FFR on doppler wire but done on reg. CT scans—esp. in mod. lesions



# Will AI allow easy/fast CACS scoring on non-EKG gated chest CT scans?

**The presence and severity of CAC can also be accurately estimated on non-contrast chest computed tomography scans performed for other clinical indications.**

However, the presence of such “incidental” CAC is rarely reported.

**Advances in artificial intelligence have now enabled automatic CAC scoring for both cardiac and non-cardiac CT scans**. Various AI approaches, from rule-based models to machine learning algorithms and deep learning, have been applied to automate CAC scoring. Convolutional neural networks, a deep learning technique, have had the most successful approach, with high agreement with manual scoring demonstrated in

[1] *European Heart Journal*, Volume 45, Issue Supplement_1, October 2024, ehae666.197, https://doi.org/10.1093/eurheartj/ehae666.197

[2] Gupta S, Cardoso R, Blankstein R. Artificial Intelligence in Coronary Artery Calcium Scoring Detection and Quantification. *Diagnostics*. 2024; 14(2):125. https://doi.org/10.3390/diagnostics14020125

- **Mean Pt. Computation time by DL 5.6 sec.**
- **Mean Pt. Computation time by experts 25.7 min.**

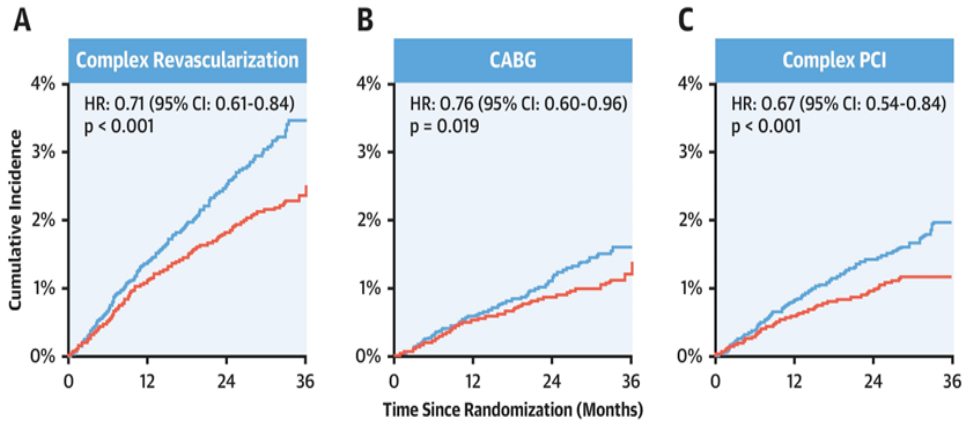
**CAC score risk category concordance (n=90)**

| Cohen’s kappa = 0.80 (p<0.0001) | | Predicted CAC score risk category: contrast-enhanced CT scans - AI model | | | | |
|---|---|---|---|---|---|---|
| | | 0 | 1-100 | 101-300 | >300 | Total |
| Reference CAC score risk category: non-contrast CT scans – expert reader | 0 | 18 | 1 | 0 | 0 | 19 |
| | 1-100 | 3 | 27 | 4 | 1 | 35 |
| | 101-300 | 0 | 1 | 16 | 1 | 18 |
| | >300 | 0 | 1 | 1 | 16 | 18 |
| | Total | 21 | 30 | 21 | 18 | 90 |

Figure 1. Agreement in coronary artery calcification (CAC) score risk categories between AI-model results derived from contrast-enhanced CT scans and manual reference assessment on non-contrast CT scans.

# What if you could "watch" serial Rx effects noninvasively with AI on CTA plaques?

## CENTRAL ILLUSTRATION: Effects of Evolocumab on Complex Revascularization

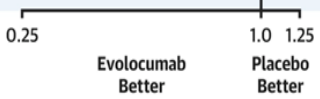



No. at risk

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Placebo | 13,780 | 13,341 | 8,149 | 748 | 13,780 | 13,449 | 8,268 | 765 | 13,780 | 13,414 | 8,241 | 756 |
| Evolocumab | 13,784 | 13,400 | 8,239 | 748 | 13,784 | 13,477 | 8,313 | 751 | 13,784 | 13,469 | 8,307 | 753 |

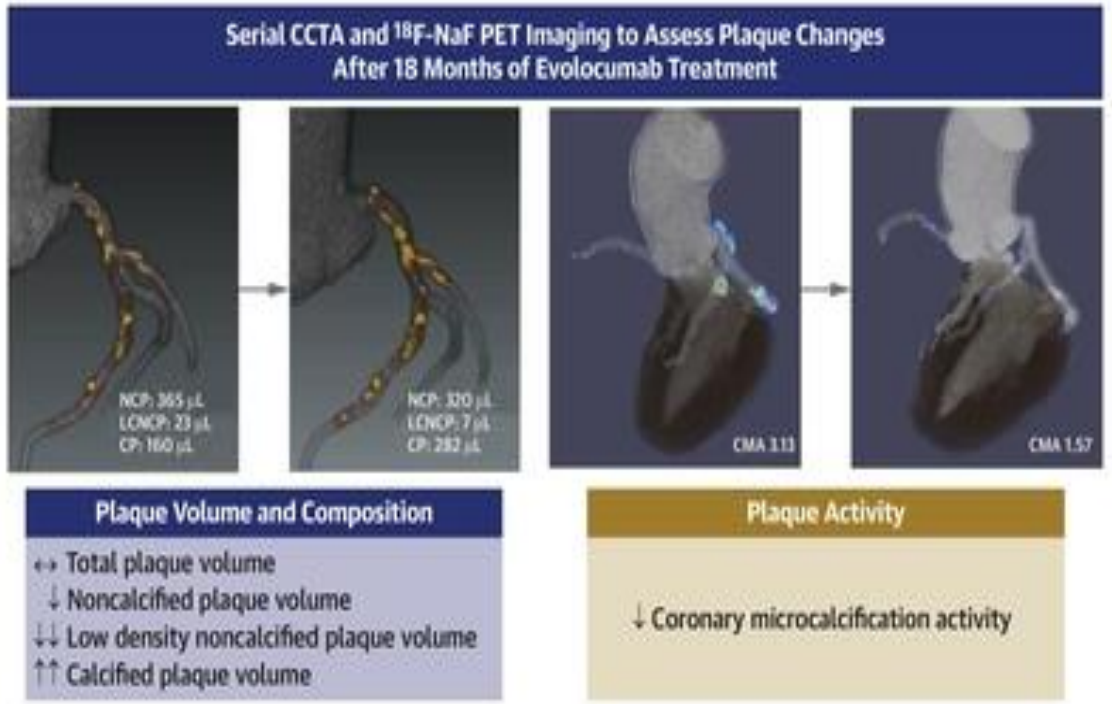
D

| | 3-Year KM Rate (%) Evolocumab | Placebo | HR (95% CI) |
|---|---|---|---|
| Complex revascularization | 2.4 | 3.5 | 0.71 (0.61-0.84) |
| CABG | 1.2 | 1.6 | 0.76 (0.60-0.96) |
| Complex PCI | 1.2 | 2.0 | 0.67 (0.54-0.84) |
| Multivessel PCI | 0.8 | 1.3 | 0.67 (0.51-0.87) |
| ≥3 stents implanted | 0.5 | 0.7 | 0.70 (0.49-0.98) |
| ≥3 lesions treated | 0.3 | 0.6 | 0.59 (0.40-0.87) |
| Bifurcation PCI with ≥2 stents | 0.1 | 0.3 | 0.55 (0.30-0.99) |
| Total stent length >60 mm | 0.3 | 0.5 | 0.70 (0.46-1.06) |




Oyama, K. et al. J Am Coll Cardiol. 2021;77(3):259-67.

## CENTRAL ILLUSTRATION: Effects of Evolocumab on Coronary Plaque and Microcalcification Activity




Han D, et al. JACC Cardiovasc Imaging. 2025;18(5):589-599.

# Can AI Change Primary Prevention?