INTERVENTIONAL TREATMENT OF STABLE CORONARY DISEASE: TO OPEN OR LEAVE CLOSED

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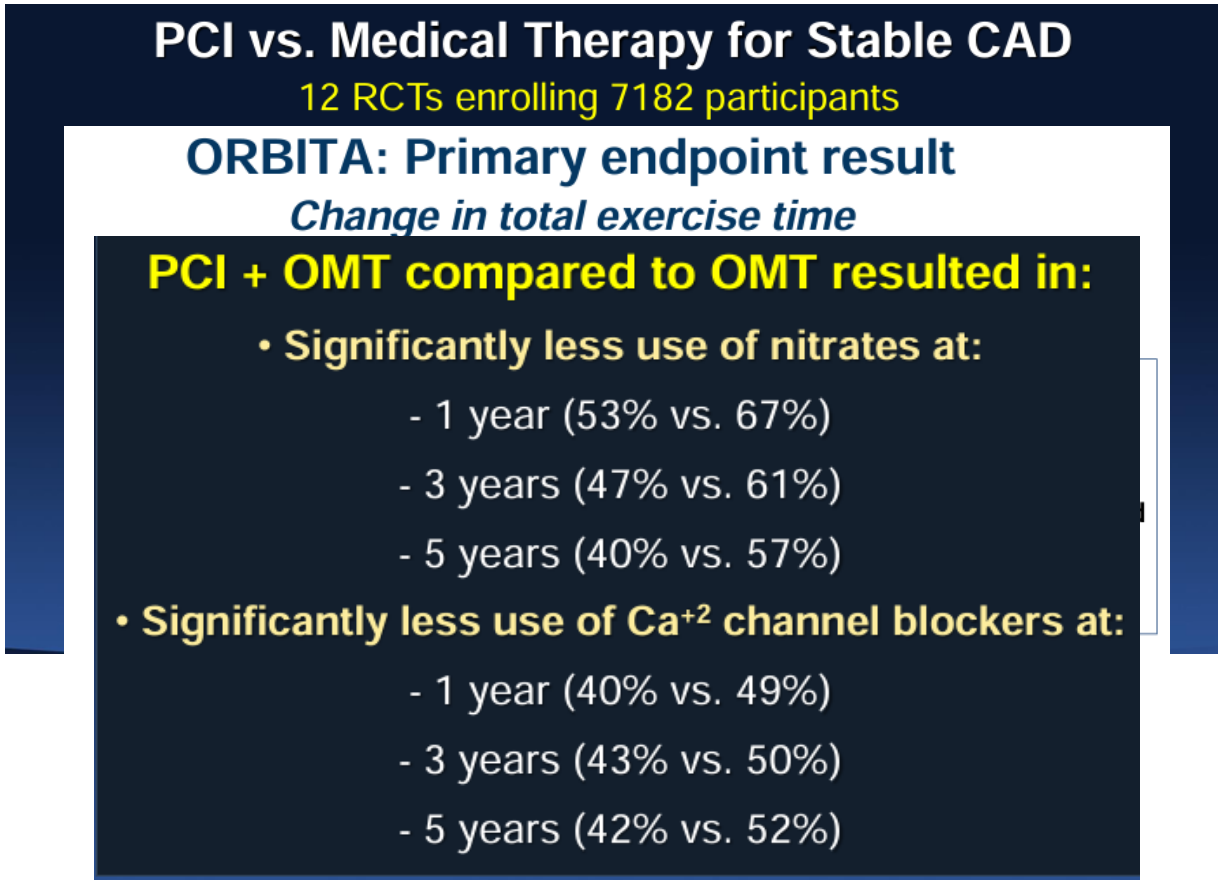
Northwell℠
Cardiovascular Institute

GOALS OF THERAPY IN PATIENTS WITH STABLE CAD

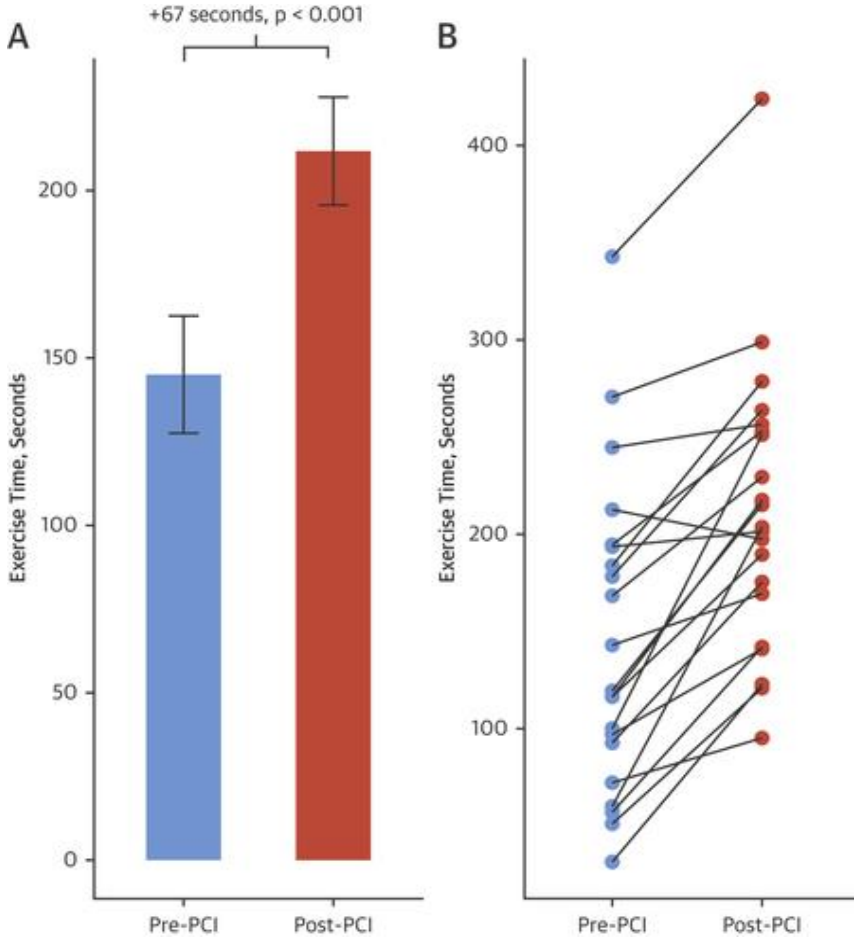
1. Improve Symptoms
2. Improve Prognosis

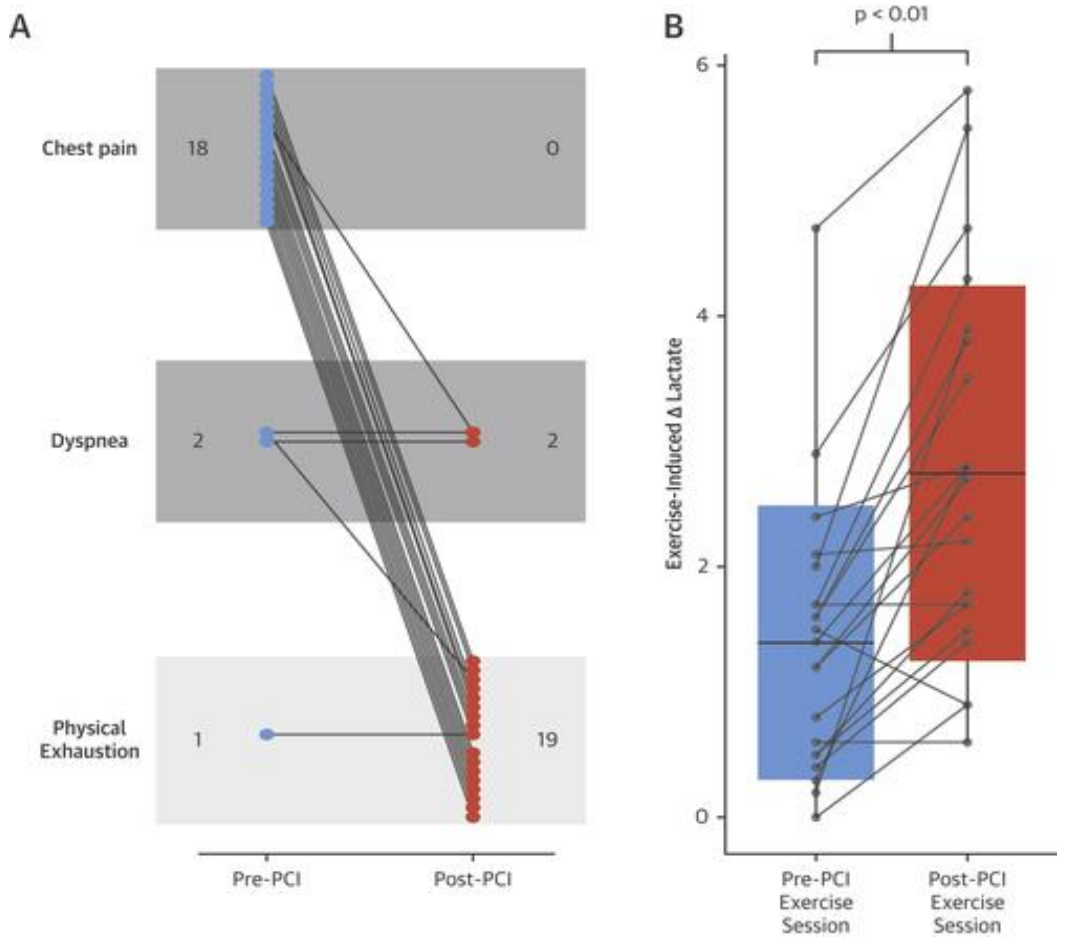


Pursnani et al, circ cv intv 2012

Boden WE, NEJM, 2007

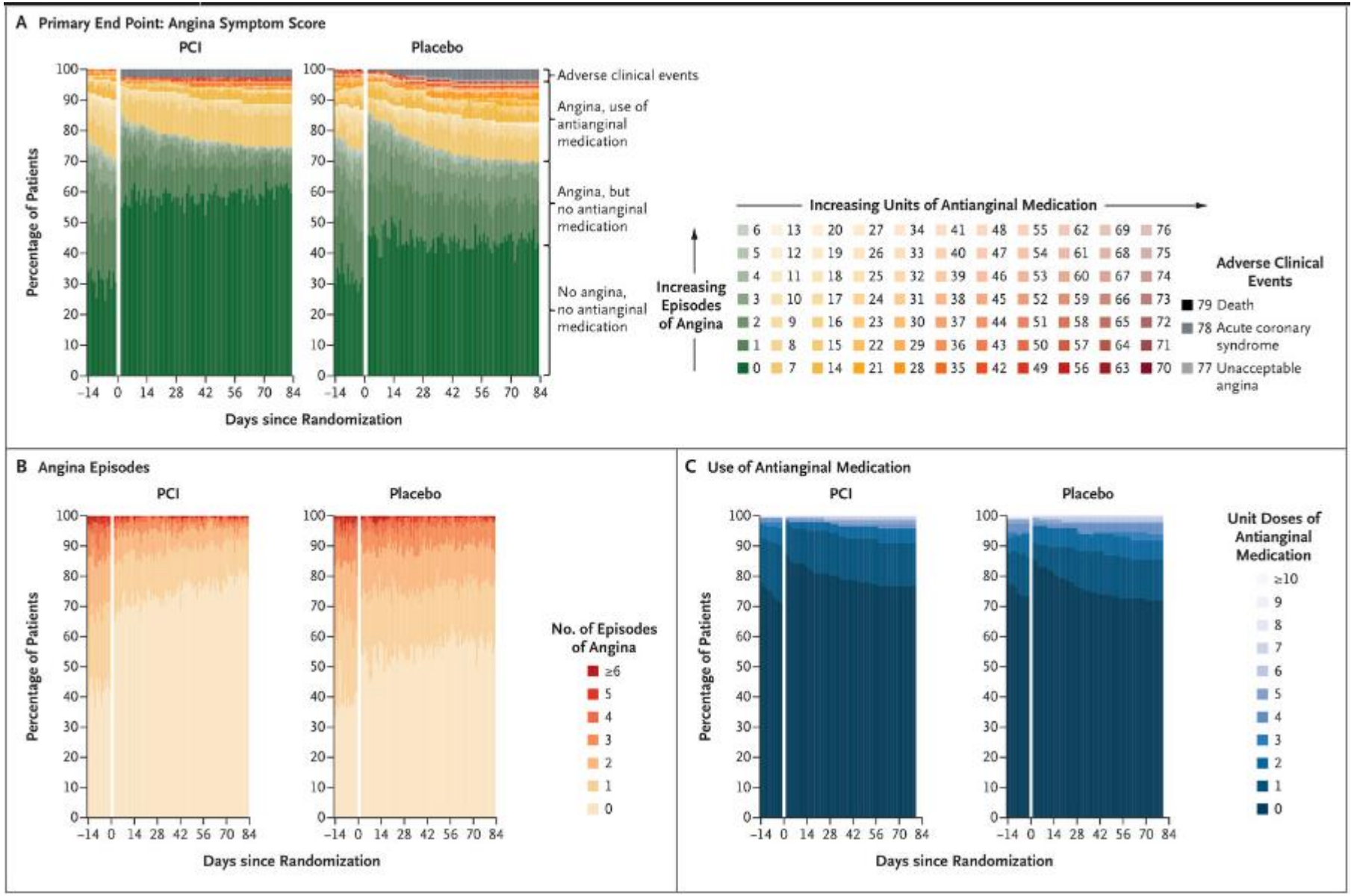
ORBITA





Christopher M. Cook et al. *JACC* 2018; 72:970-983.

ORBITA II

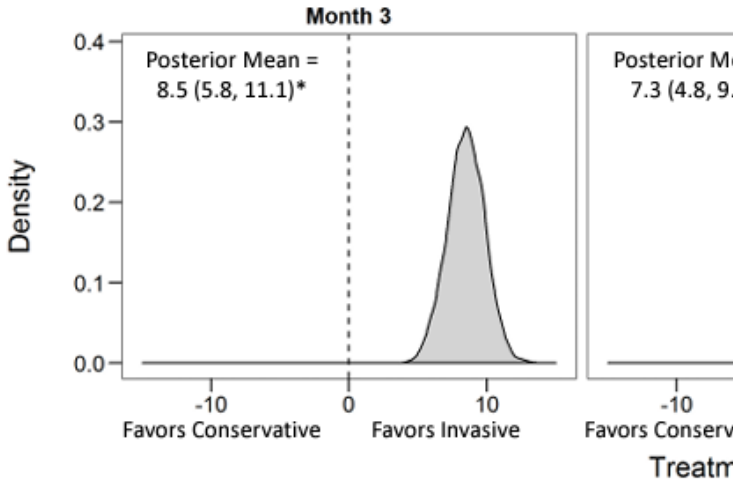


Rajkumar et al, NEJM 2023

EVEN THE ISCHEMIA TRIAL SHOWED BENEFIT FOR PCI

Primary Outcome:
Benefit of Invasive Rx on SAQ Summary Score

Typical Patient with Daily to Weekly Angina

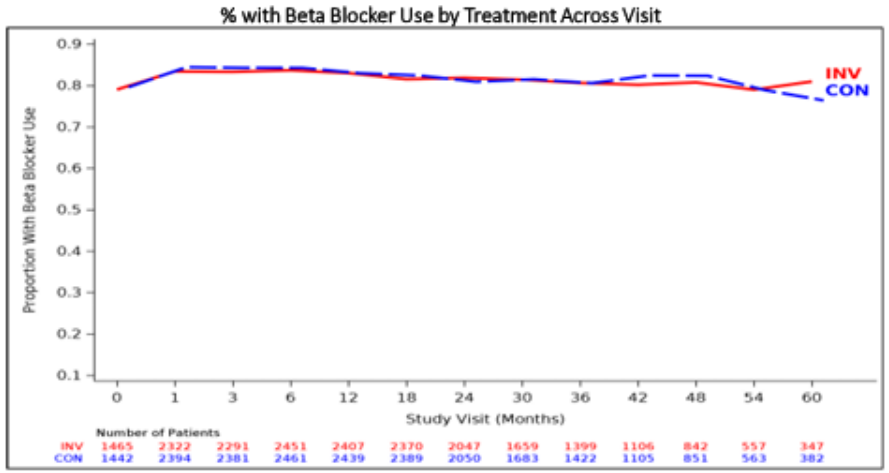


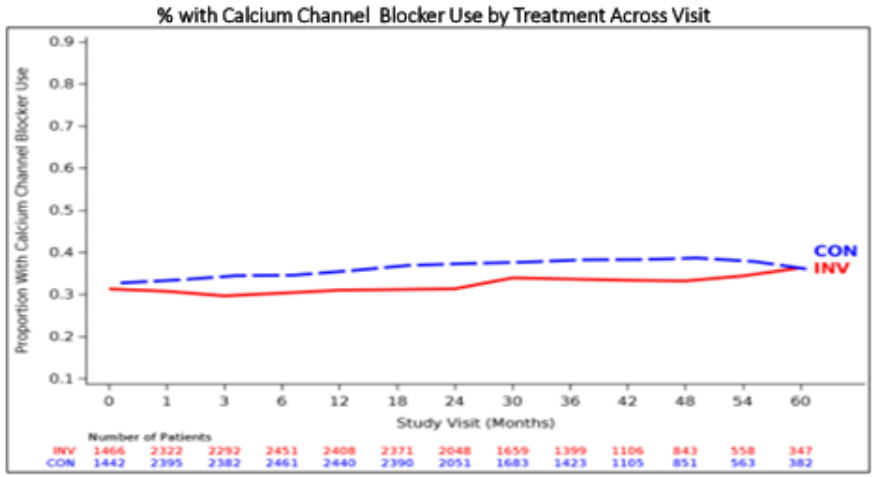
*95% Highest Pos

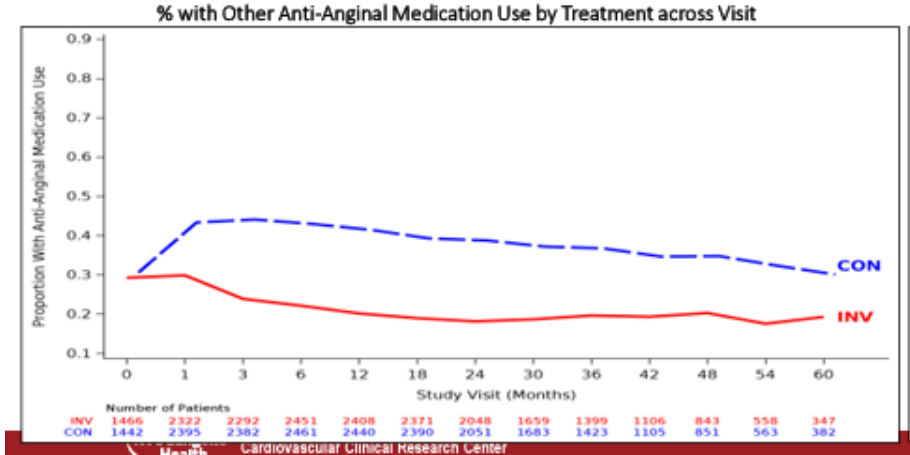
Month 12

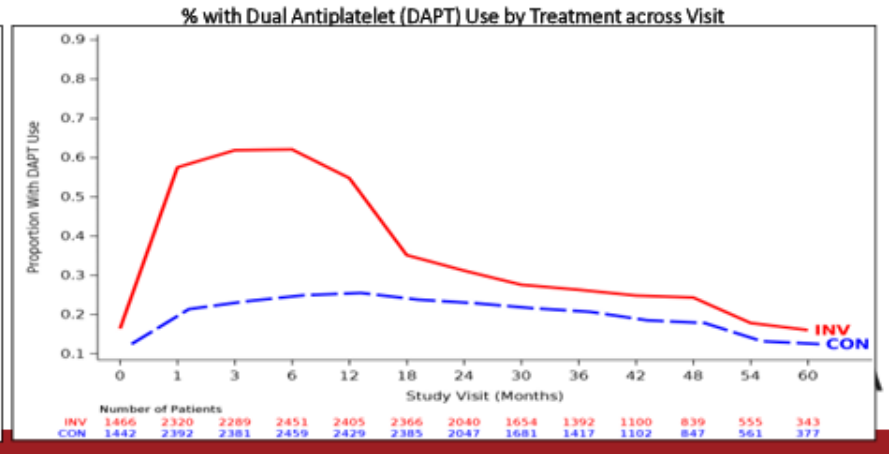
Month 36

Medication Use Over Time



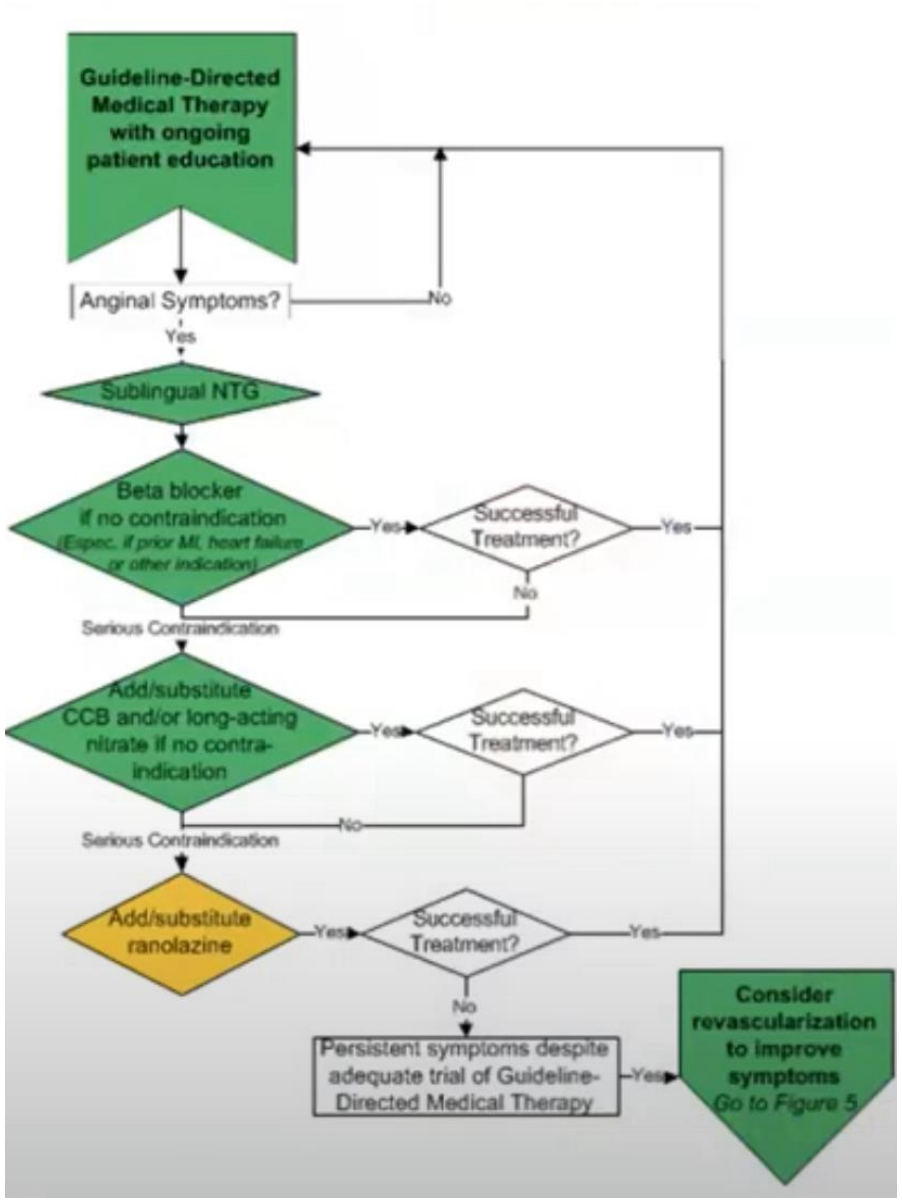






Revascularization to Improve Symptoms in SIHD

1	A	In patients with refractory angina despite medical therapy and with significant coronary artery stenoses amenable to revascularization, revascularization is recommended to improve symptoms.



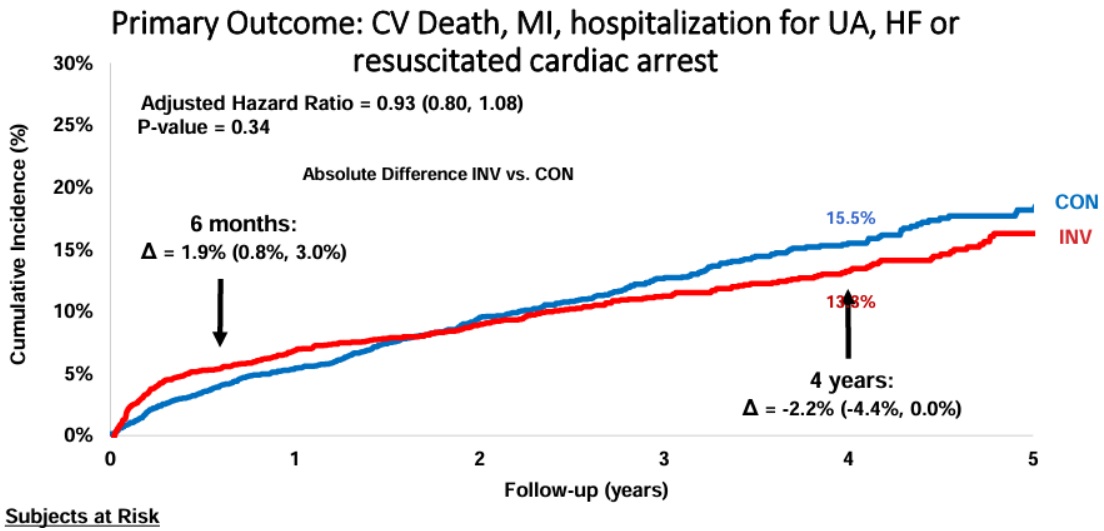
CAN PCI AFFECT PROGNOSIS IN STABLE ISCHEMIC HEART DISEASE?

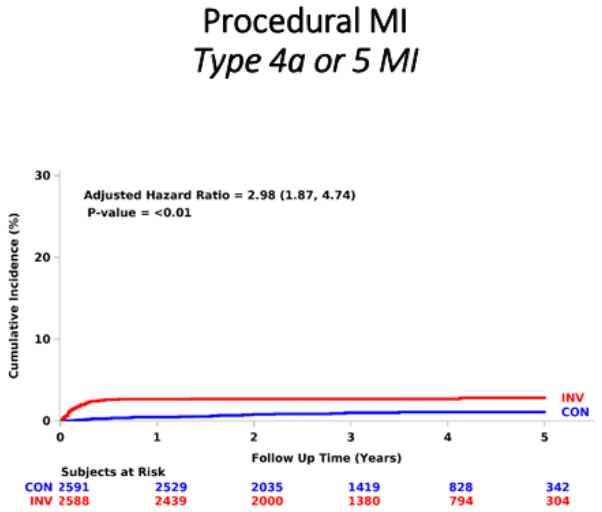
- Depends on the anatomy!
- Danish Cohort Study (2023): CCTA can be used to evaluate anatomy in SHD
  - Highest rate of adverse events with more disease burden on CT
- Extent of Disease matters!

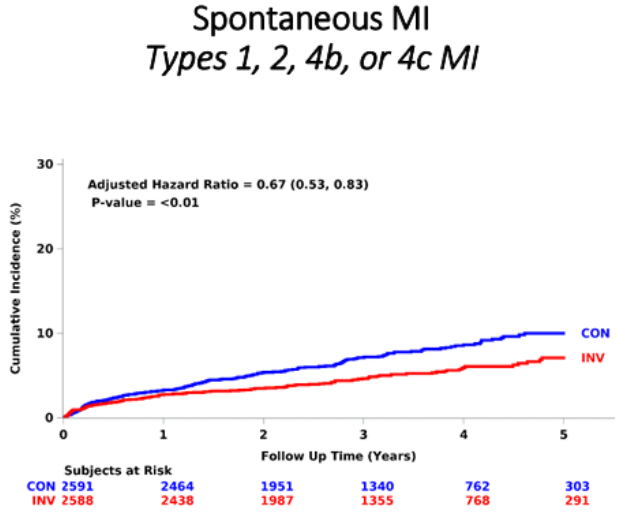
CAD Prognostic Index

Extent of CAD	Prognostic Weight (0–100)	5-Year Survival Rate (%)*
1-vessel disease, 75%	23	93
1-vessel disease, 50% to 74%	23	93
1-vessel disease, ≥95%	32	91
2-vessel disease	37	88
2-vessel disease, both ≥95%	42	86
1-vessel disease, ≥95% proximal LAD artery	48	83
2-vessel disease, ≥95% LAD artery	48	83
2-vessel disease, ≥95% proximal LAD artery	56	79
3-vessel disease	56	79
3-vessel disease, ≥95% in ≥1 vessel	63	73
3-vessel disease, 75% proximal LAD artery	67	67
3-vessel disease, ≥95% proximal LAD artery	74	59

BACK TO ISCHEMIA TRIAL

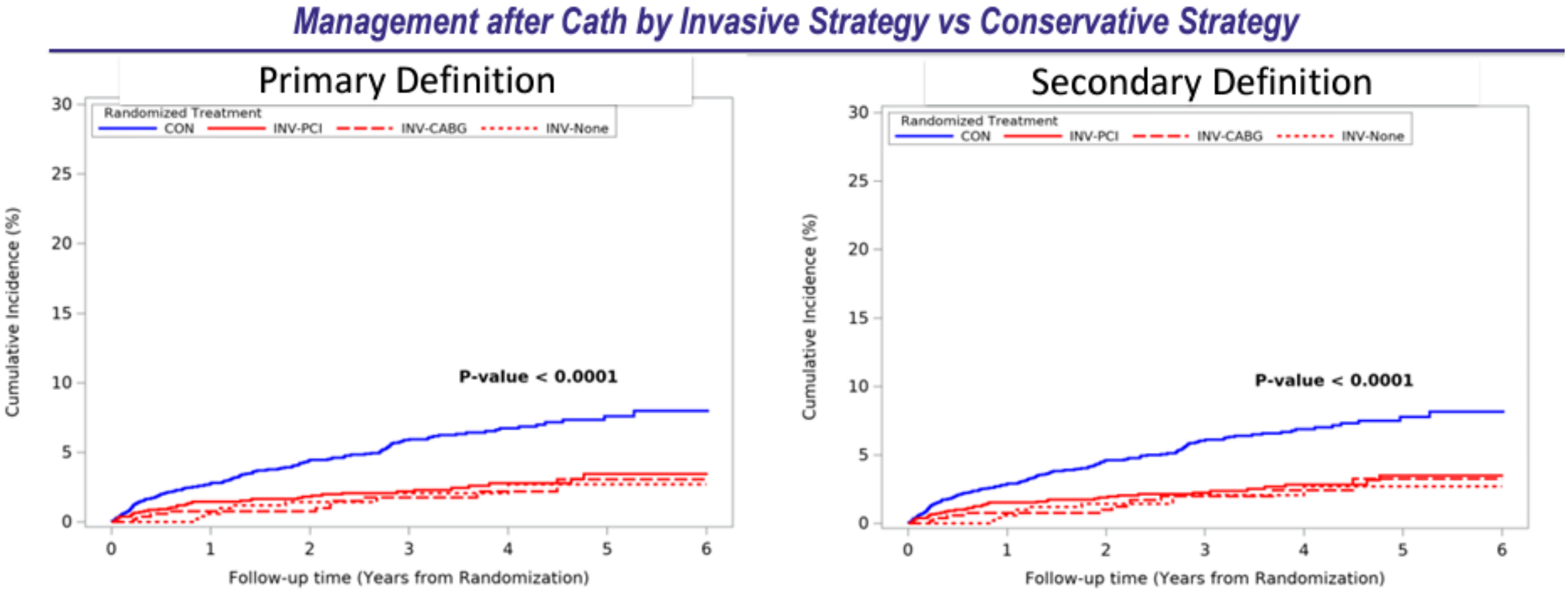






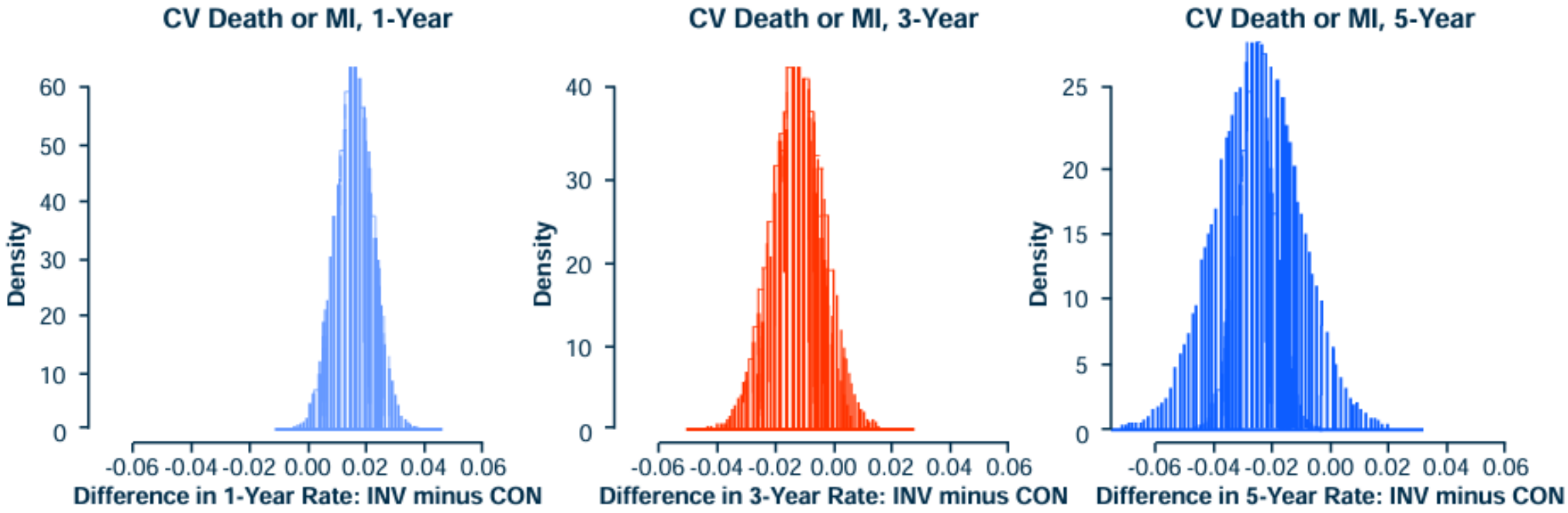
- Significant difference in primary outcome after 4 years

WHAT TYPE OF MI IS THIS?



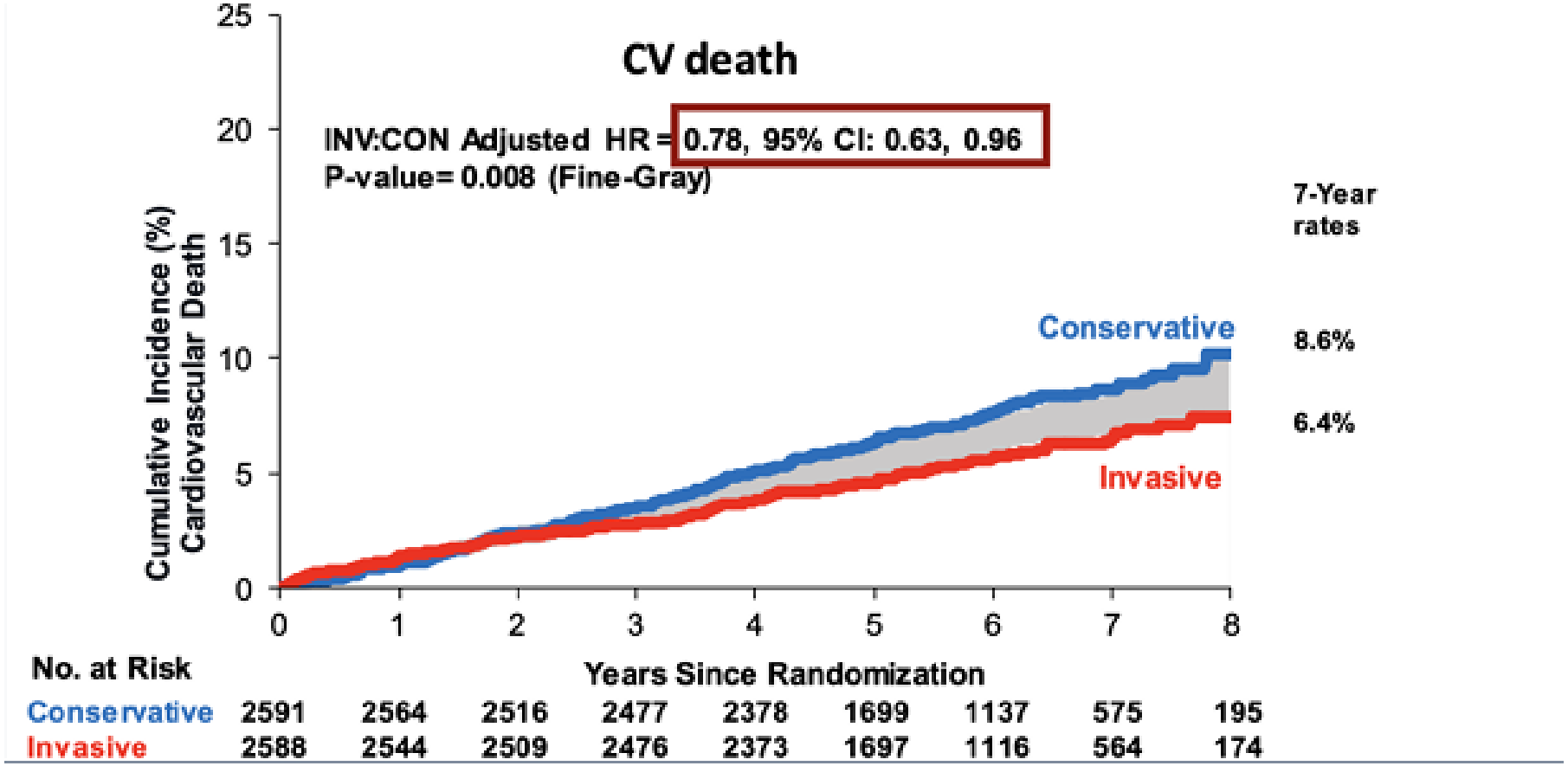
B Chaitman, ACC, 2020

CUMULATIVE EVENT RATE AT 1, 3 AND 5 YEARS



Maron et al. NEJM 2020

ISCHEMIA - EXTENDED



PREVENT

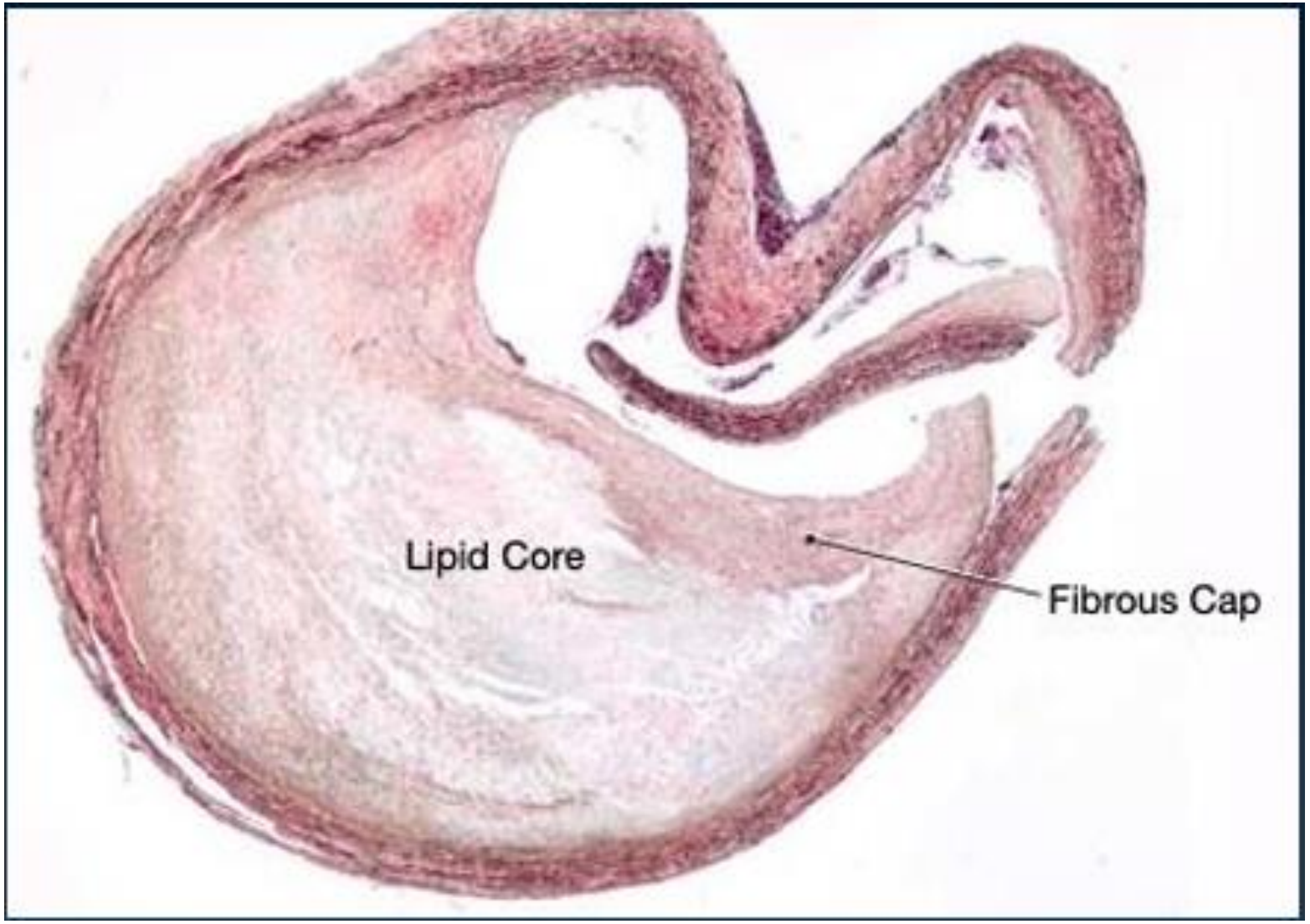
Individual Components of the Primary Composite Outcome PREVENT

Endpoints	Preventive PCI plus OMT (N=803)	OMT alone (N=803)	Difference in event rates (95% CI)	Hazard ratio (95% CI)
Primary composite outcome				**0·54 (0·33 to 0·87)**
At 2 years‡	3 (0·4%)	27 (3·4%)	-3·0 (-4·4 to -1·8)	**0·11 (0·03 to 0·36)**
At 4 years	17 (2·8%)	37 (5·4%)	-2·6 (-4·7 to -0·4)	
At 7 years	26 (6·5%)	47 (9·4%)	-2·9 (-7·3 to 1·5)	
Death from cardiac causes				0·87 (0·31 to 2·39)
At 2 years	1 (0·1%)	6 (0·8%)	-0·6 (-1·3 to	
At 4 years	5 (0·8%)	7 (0·9%)	-0·1 (-1·1 to	
At 7 years	7 (1·4%)	8 (1·3%)	0·1 (-1·4 to	
Target-vessel related MI				
At 2 years	1 (0·1%)	6 (0·8%)	-0·6 (-1·3 to	

Individual Components of the Primary Composite Outcome PREVENT

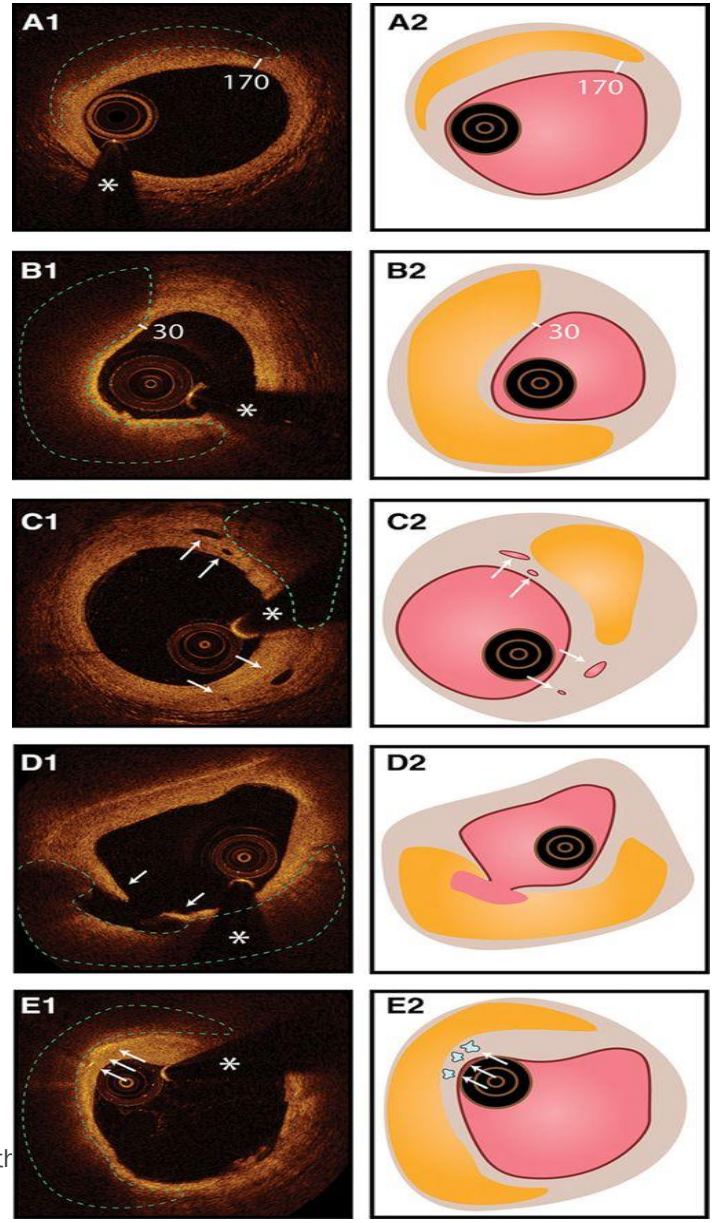
Endpoints	Preventive PCI plus OMT (N=803)	OMT alone (N=803)	Difference in event rates (95% CI)	Hazard ratio (95% CI)
Ischemia-driven target-vessel revascularization				**0·44 (0·25 to 0·77)**
At 2 years	1 (0·1%)	19 (2·4%)	-2·3 (-3·4 to -1·2)	
At 4 years	10 (1·7%)	29 (4·4%)	-2·7 (-4·6 to -0·8)	
At 7 years	17 (4·9%)	38 (8·0%)	-3·2 (-7·4 to 1·1)	
Hospitalization for unstable or progressive angina				**0·19 (0·06 to 0·54)**
At 2 years	1 (0·1%)	12 (1·5%)	-1·4 (-2·3 to -0·5)	
At 4 years	4 (0·7%)	16 (2·4%)	-1·7 (-3·0 to -0·4)	
At 7 years	4 (0·7%)	21 (4·9%)	-4·2 (-7·17 to -1·4)	

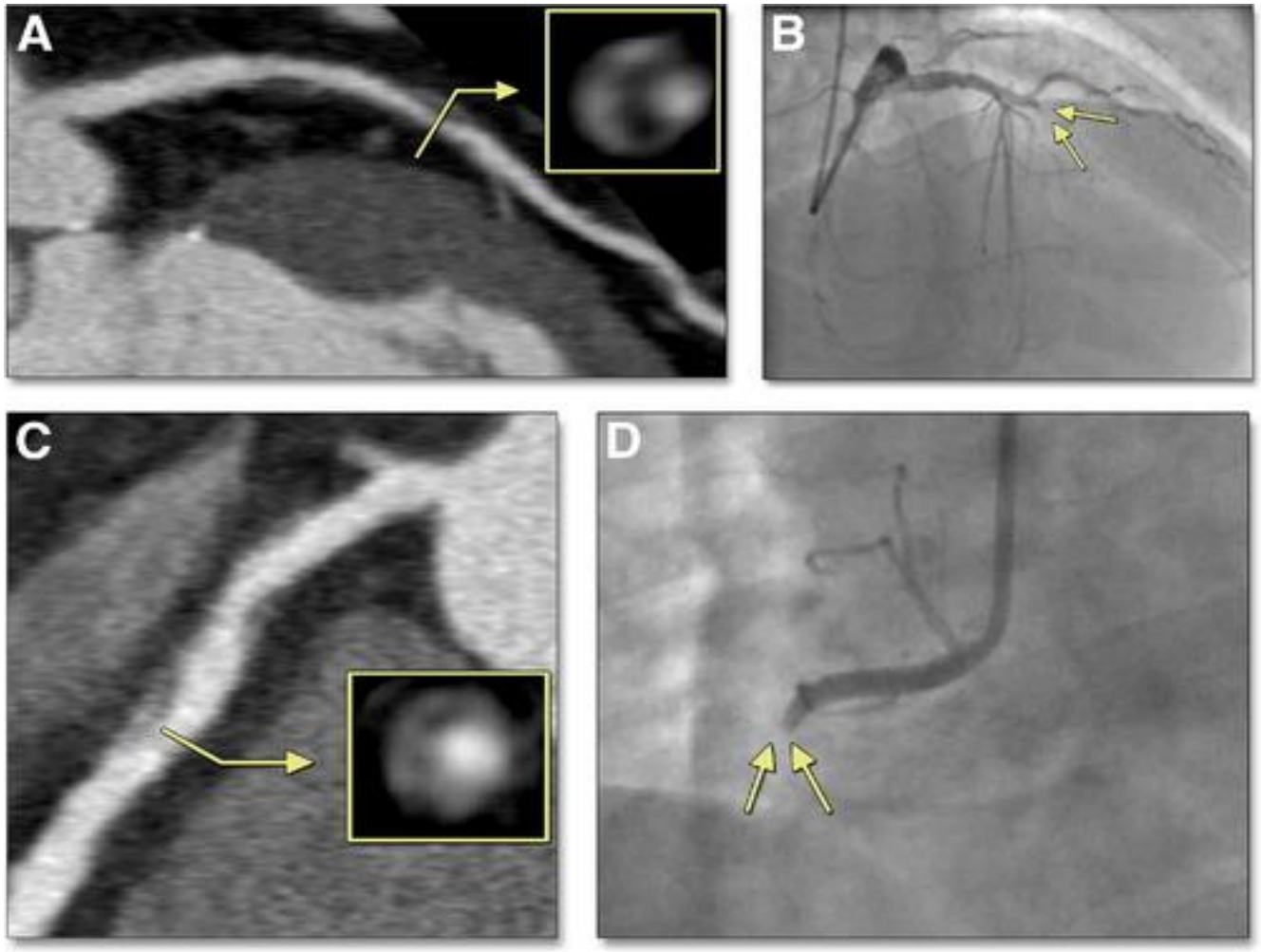
VULNERABLE PLAQUE



Spagnoli et al. JAMA. 2004

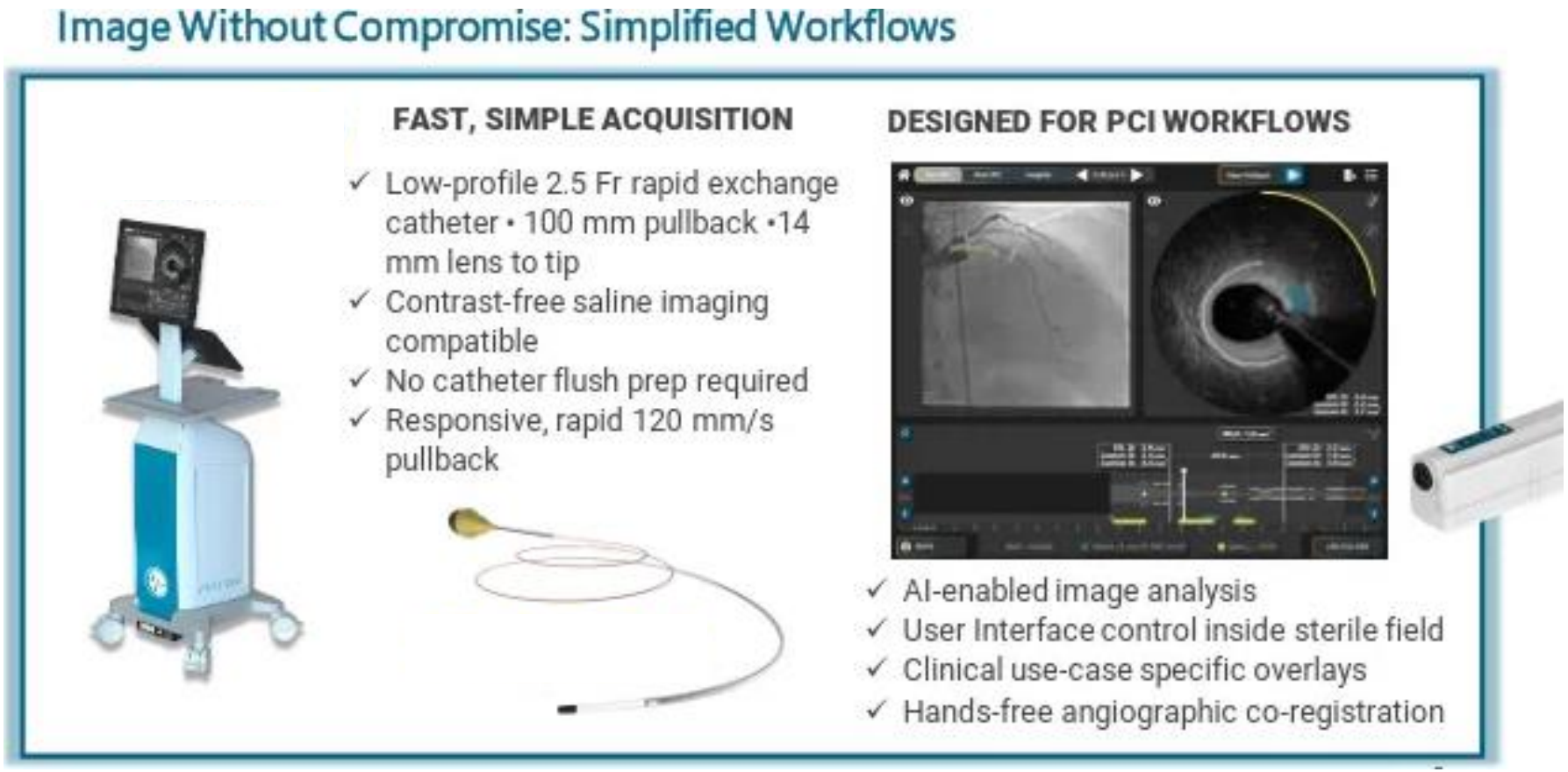
VULNERABLE PLAQUE → PLAQUE DISRUPTION

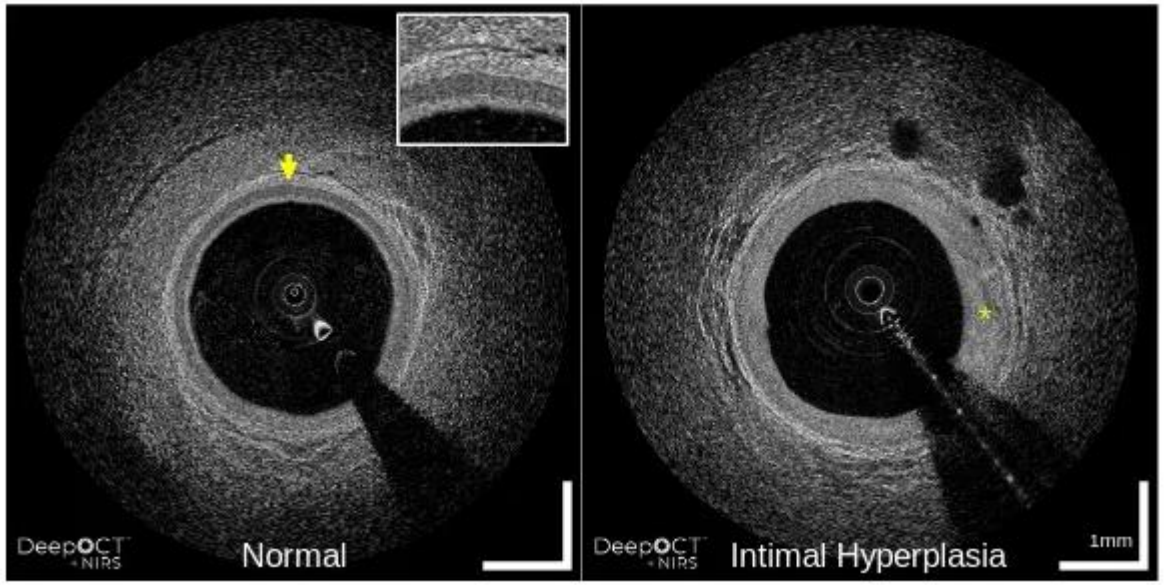


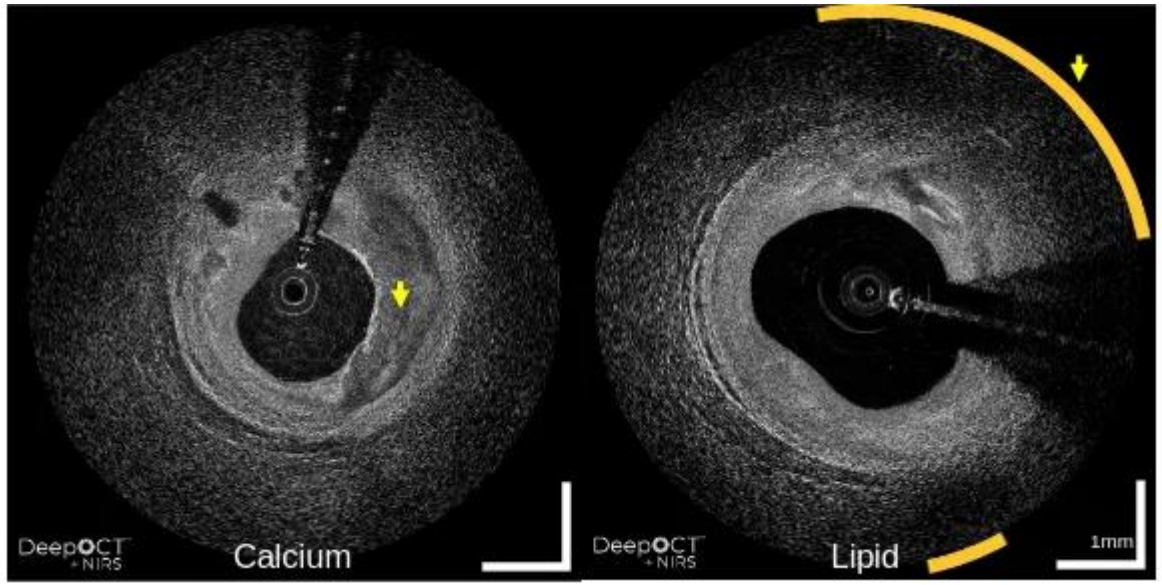


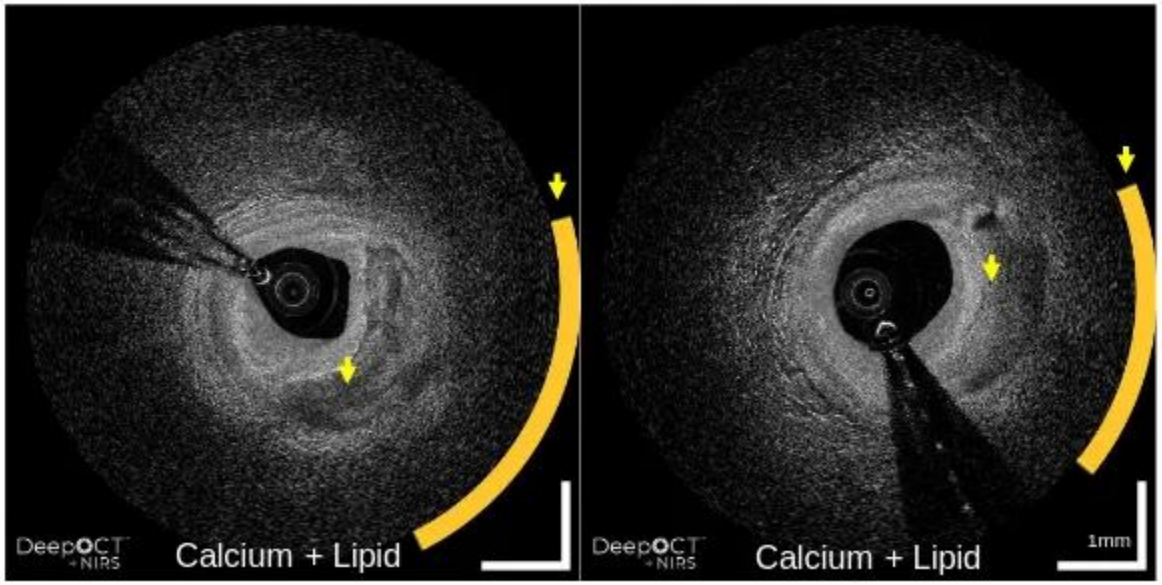
10 months apart

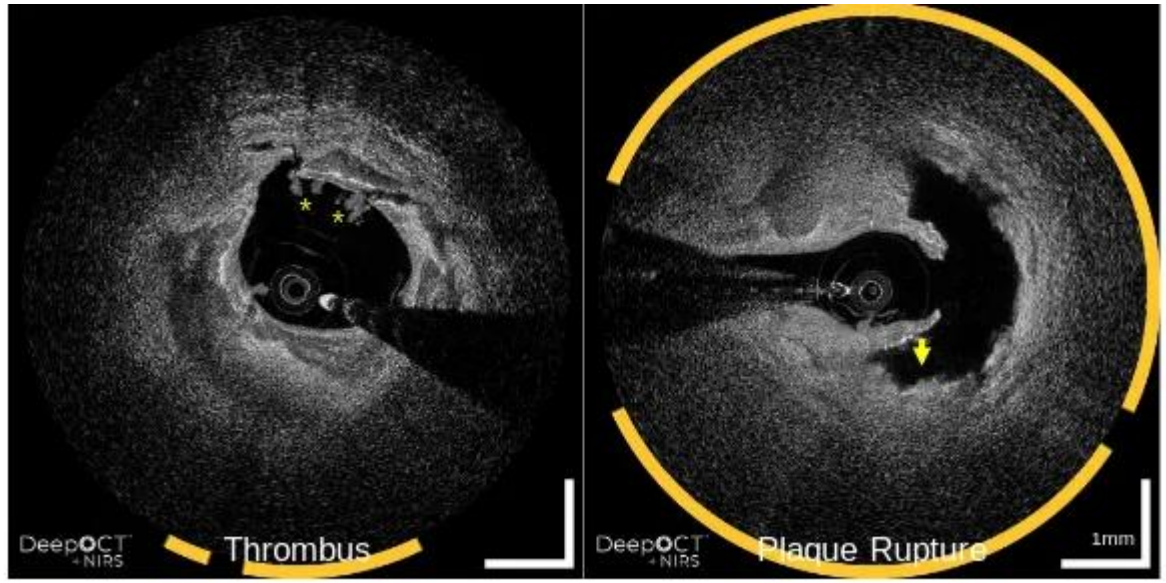
OPTICAL COHERENCE TOMOGRAPHY (OCT) COUPLED WITH NEAR-INFRARED SPECTROSCOPY (NIRS)



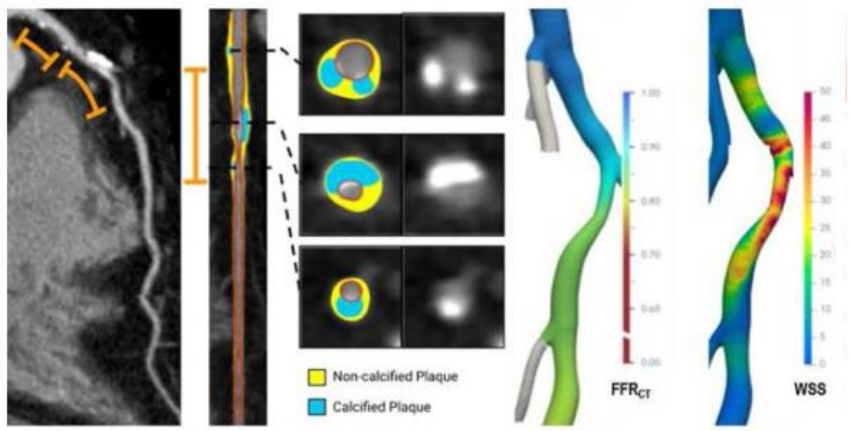

DeepOCT +NIRS
Normal
DeepOCT +NIRS
Intimal Hyperplasia
1mm


DeepOCT +NIRS
Calcium
DeepOCT +NIRS
Lipid
1mm


DeepOCT +NIRS
Calcium + Lipid
DeepOCT +NIRS
Calcium + Lipid
1mm


DeepOCT +NIRS
Thrombus
DeepOCT +NIRS
Plaque Rupture
1mm

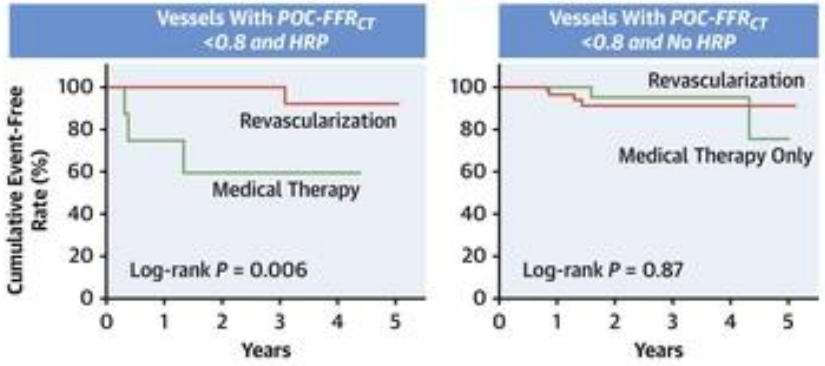
AI- ENABLED QUANTITATIVE PLAQUE AND HEMODYNAMIC ANALYSIS: EMERALD II



1. Percent total myocardial blood flow ≥ 20% (56% of patients) [MBF]
2. Change in FFRCT ≥ 0.05 (42% of patients) [Local Hemodynamic Severity]
3. Percent area stenosis ≥ 65% (40% of patients) [Luminal Stenosis]
4. Noncalcified plaque volume ≥ 72.5 mm3 (39% of patients) [Plaque Morphology]
5. Plaque burden ≥ 85% (35% of patients) [Atherosclerosis Burden]

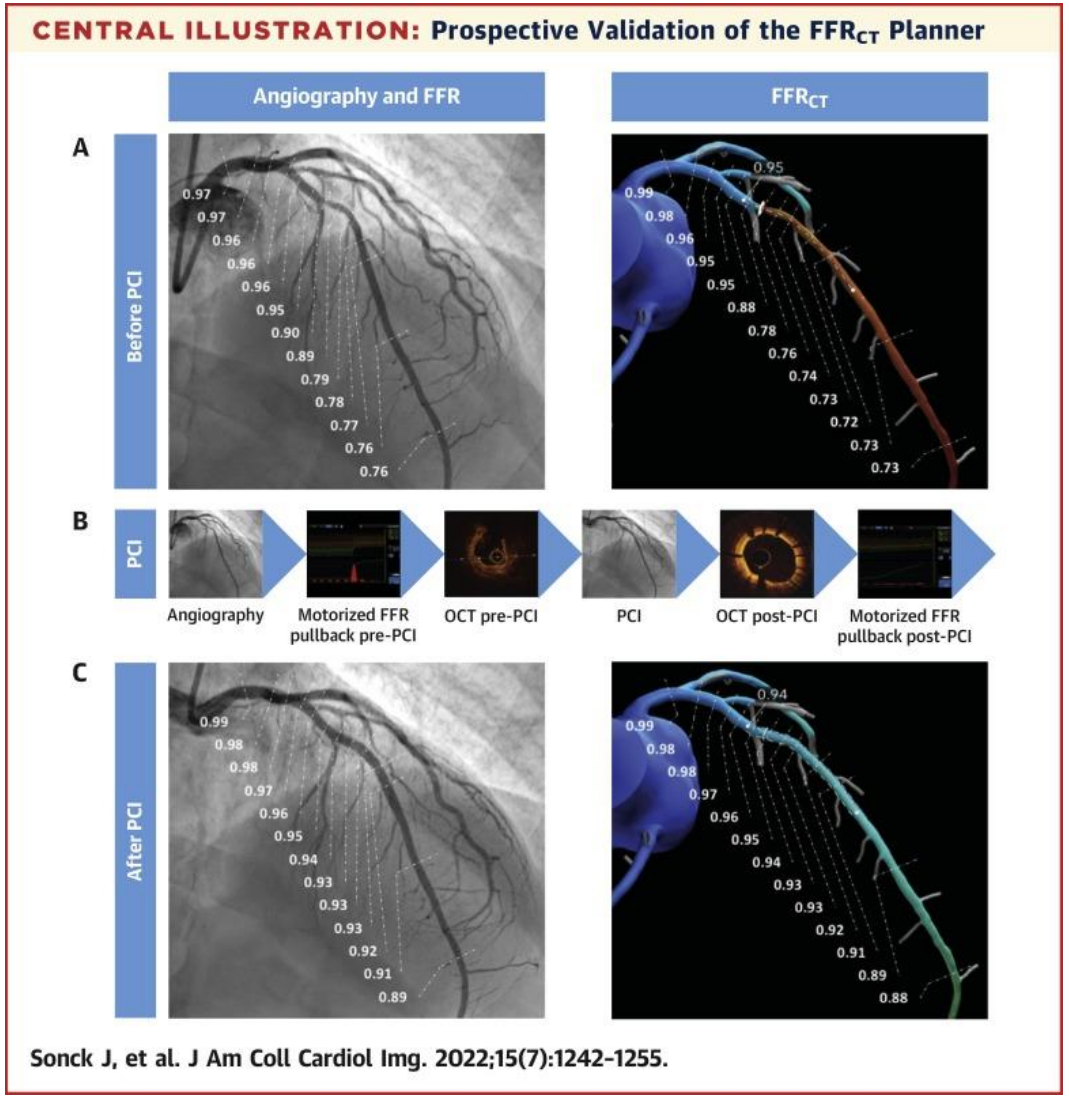
CENTRAL ILLUSTRATION: Revascularization and Plaque Assessment of Point-of-Care Computed Tomography Angiography-Based Fractional Flow Reserve-Positive Lesions

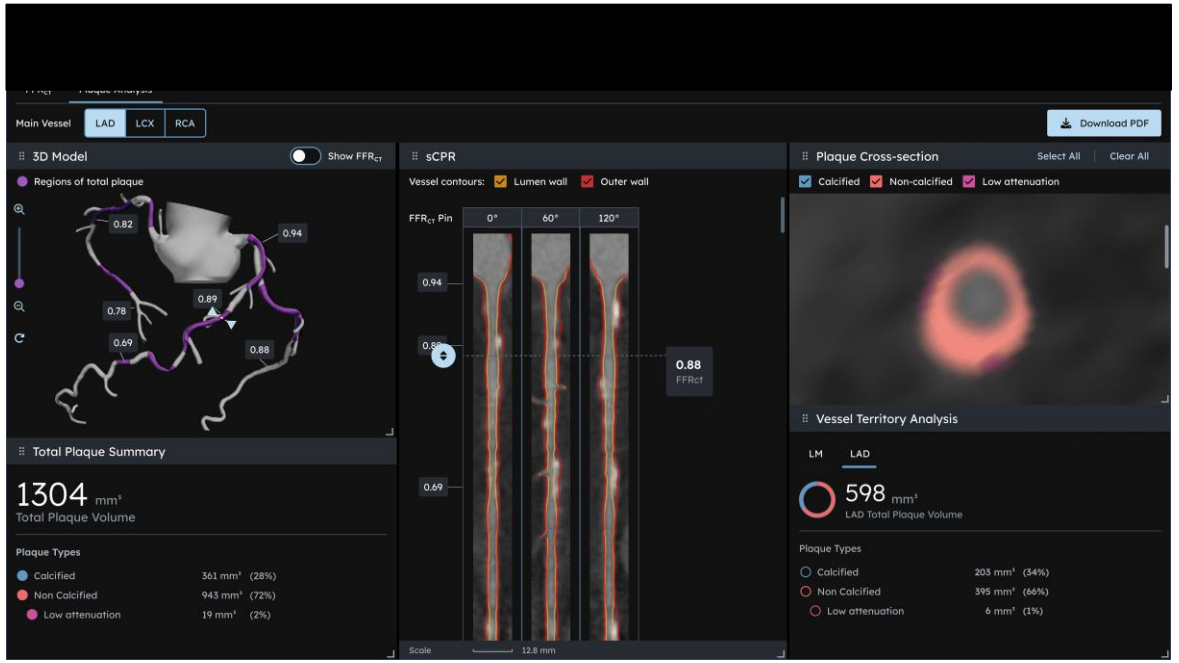
	FFR <0.80	FFR ≥0.80
HRP(+)	Revascularization 1.4 vs no revascularization 16.4 per 100 vessel-years, Log-rank *P* = 0.006 1. Could this represent unsafely deferred group in COURAGE and ISCHEMIA? 2. Could this represent appropriately chosen group for FAME-2 study? 3. Could this have been an alternative population for PROSPECT-II study?	Revascularization 0 vs no revascularization 3.1 per 100 vessel-years, Log-rank *P* = 0.47 1. This represents the PROSPECT-II population. 2. Could this group be treated with aggressive lipid-lowering Rx instead? 3. Do plaques imminently at danger of rupture need better identification?
HRP(−)	Revascularization 2.8 vs no revascularization 2.7 per 100 vessel-years, Log-rank *P* = 0.87 1. Does this group represent true COURAGE or ISCHEMIA proposal? 2. Could this population be safely deferred from the FAME-2 indication?	Revascularization 0 vs no revascularization 0.05 per 100 vessel-years, Log-rank *P* = 0.87 1. Revascularization is unjustified in these patients. 2. Only preventive Rx should be recommended.



Sato Y, et al. J Am Coll Cardiol Img. 2024;17(3):284-297.

PLAQUE ANALYSIS AND PCI PLANNING





CONCLUSION

- PCI plays a role in both reducing symptoms and improving prognosis of patients with stable coronary artery disease
- If you have a SIHD patient with a positive stress test, consider a CT Coronary Study to rule out Left Main disease and to evaluate plaque burden/morphology
- Stay current with the upcoming research in this space — we are finally close to the answer for this question:
  - How can we identify the vulnerable plaque and treat it before the heart attack happens?

THANK YOU