Brigham and Women's Hospital
Founding Member, Mass General Brigham

Management of Ischemic Heart Disease and Lipids - 2025

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Professor of Medicine, Harvard Medical School

CONTINUING MEDICAL EDUCATION
DEPARTMENT OF MEDICINE

HARVARD MEDICAL SCHOOL
TEACHING HOSPITAL

Benjamin Scirica, MD MPH

- Harvard Medical School
- Residency and Fellowship @ Brigham and Women's Hospital
- MPH @ Harvard School of Public Health
- Professor of Medicine @ Harvard Medical School
  - Clinical focus: Critical Care Cardiology
  - Research focus: Diabetes, Obesity, Care-redesign

Disclosures

Dr. Scirica reports institutional research support to Brigham and Women's Hospital from Amgen, Better Therapeutics, Boehringer Ingelheim, Foresite Labs. Milestone Pharmaceutical, Merck, NovoNordisk, Pfizer, and Verve Therapeutics. Consulting fees from Abbvie (DSMB), Amgen, AstraZeneca (DSMB), Bayer, Boehringer Ingelheim (DSMB), Elsevier Practice Update Cardiology, Hanmi (DSMB), Lexeo (DSMB), NovoNordisk, Verve Therapeutics, and equity in Health at Scale, Arboretum Lifesciences, and AIwithCare.com, and a family member is an employee at Vertex Pharmaceuticals and has stock.

Key Learning Objectives

- Understand what Stable Ischemic Heart Disease means and its clinical implications
- Review the latest data on lipid-lowering therapies
- Identify key questions regarding coronary revascularization.

What is Ischemic Heart Disease

by any other name…

- Chronic stable angina
- ***Chronic coronary syndrome***
- Stable angina pectoris
- Coronary heart disease
- Stable ischemic heart disease
- Stable coronary artery disease
- Non-acute coronary syndrome

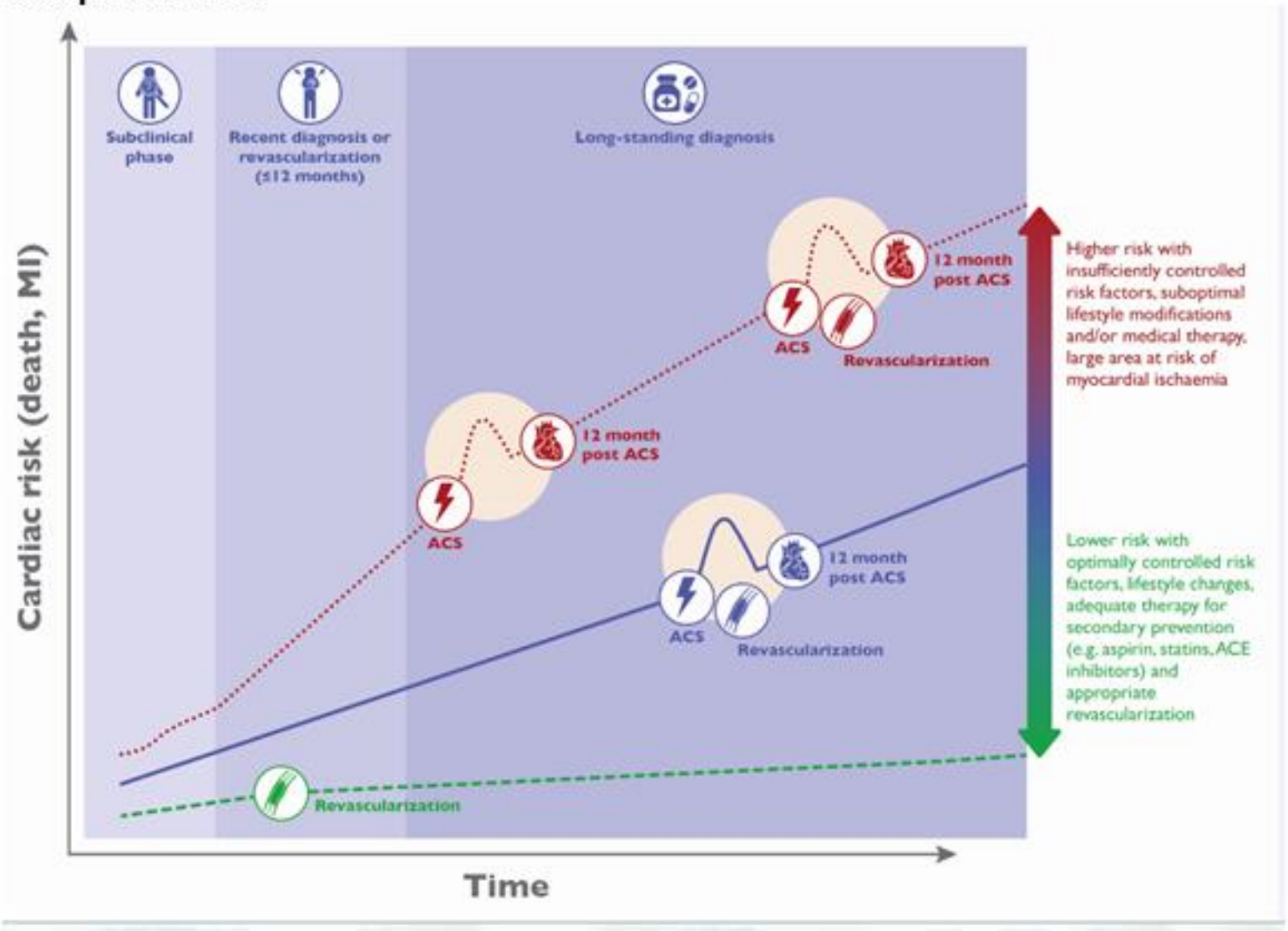
"… angina secondary to stable ischemic heart disease (SIHD) is the most common clinical presentation of cardiovascular disease encountered by general practitioners and cardiologists."

AHA 2023 CHD Statistics

Population group	Prevalence, CHD, 2017–2020, ≥20 y of age
Both sexes	20 500 000 (7.1%) [95% CI, 6.1%–8.3%]
Males	11 700 000 (8.7%)
Females	8 800 000 (5.8%)
NH White males	9.4%
NH White females	5.9%
NH Black males	6.2%
NH Black females	6.3%
Hispanic males	6.8%
Hispanic females	6.1%
NH Asian males	5.2%
NH Asian females	3.9%
NH American Indian or Alaska Native	…

Natural history of chronic coronary syndromes

A dynamic process

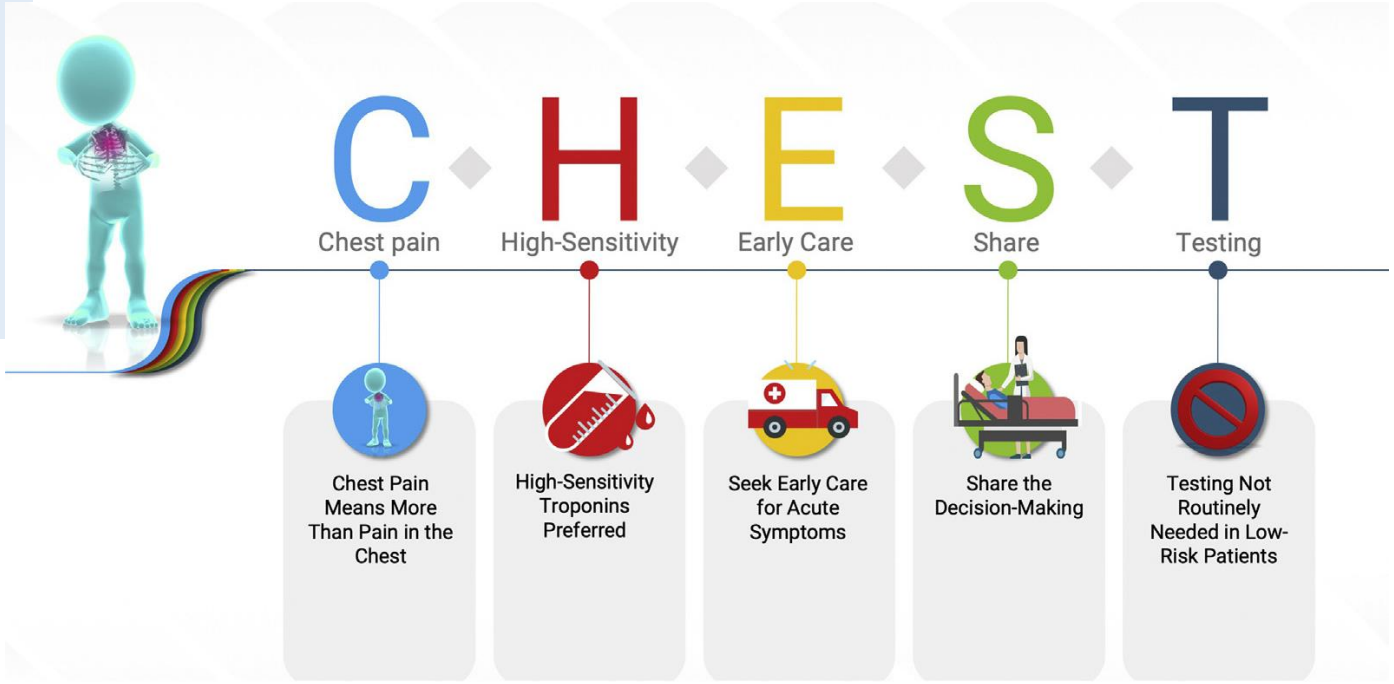


ESC Guidelines on the diagnosis and management of chronic coronary syndromes (European Heart Journal 2019; 10.1093/eurheartj/ehz425)

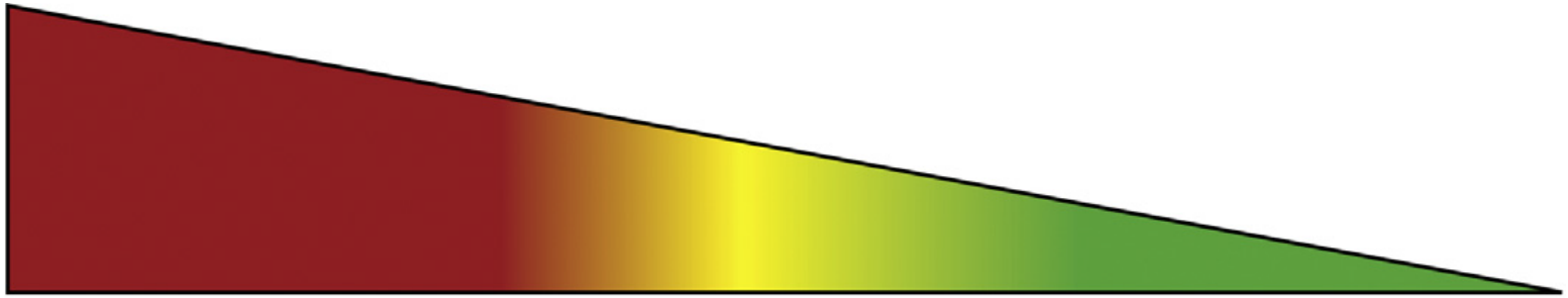
CLINICAL PRACTICE GUIDELINE: FULL TEXT

2021 AHA/ACC/ASE/CHEST/SAEM/ SCCT/SCMR Guideline for the Evaluation and Diagnosis of Chest Pain

AIM: This clinical practice guideline for the evaluation and diagnosis of chest pain provides recommendations and algorithms for clinicians ***to assess and diagnose chest pain in adult patients.***



Index of Suspicion Based on Descriptors



- Central
- Pressure
- Squeezing
- Gripping
- Heaviness
- Tightness
- Exertional/stress-related
- Retrosternal

- Left-sided
- Dull
- Aching

- Stabbing

- Right-sided
- Tearing
- Ripping
- Burning

- Sharp
- Fleeting
- Shifting
- Pleuritic
- Positional

Nomenclature Update

“Noncardiac Is In. Atypical Is Out. “

Noncardiac” should be used if heart disease is not suspected.

“Atypical” is a misleading descriptor of chest pain, and its use is discouraged.

Pre-Test Probability

Light Green – no testing

Green/Orange – testing indicated

Pretest Probabilities of Obstructive CAD in Symptomatic Patients
(A) according to age, sex, and symptoms;
(B) according to age, sex, symptoms, and CAC

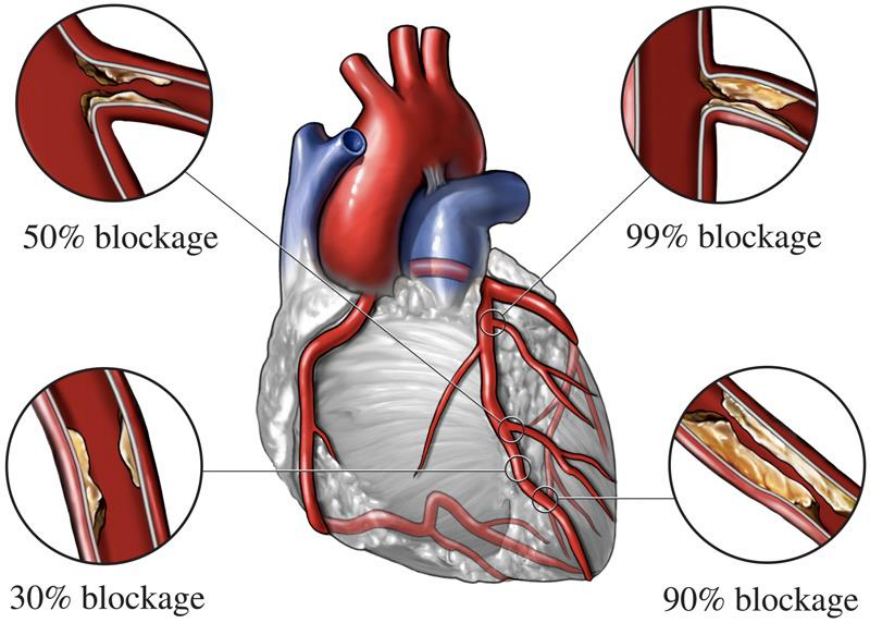
Age, y	Chest Pain		Dyspnea	
	Men	Women	Men	Women
30–39	≤4	≤5	0	3
40–49	≤22	≤10	12	3
50–59	≤32	≤13	20	9
60–69	≤44	≤16	27	14
70+	≤52	≤27	32	12

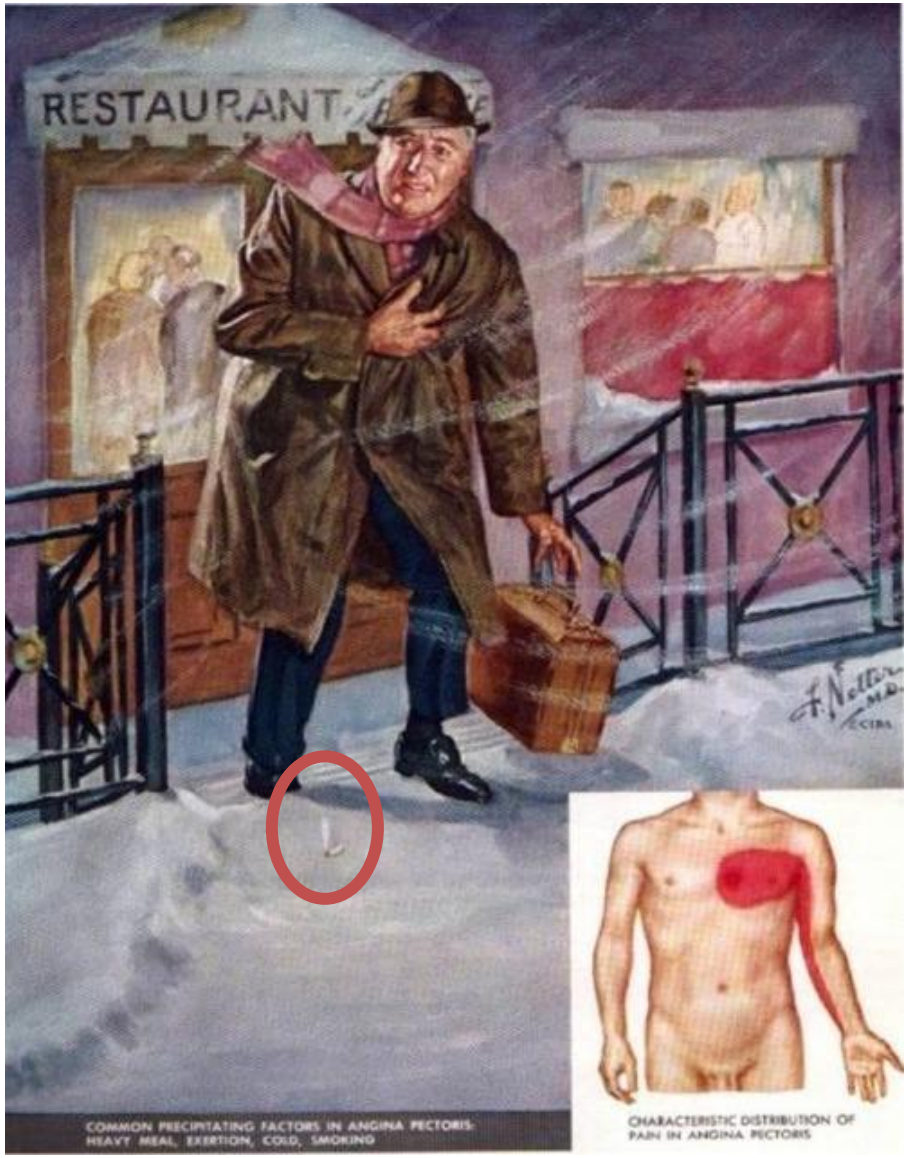
When to Order What Test

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

Stress testing information					
	ETT	**Stress echocardiography**	**SPECT MPI**	**PET MPI**	**Stress CMR MPI**
Patient capable of exercise	✓	✓	✓		
Pharmacologic stress indicated		✓	✓	✓	✓
Quantitative flow				✓	✓
LV dysfunction/scar		✓	✓	✓	✓

Stable Ischemic Heart Disease *(Old School)*

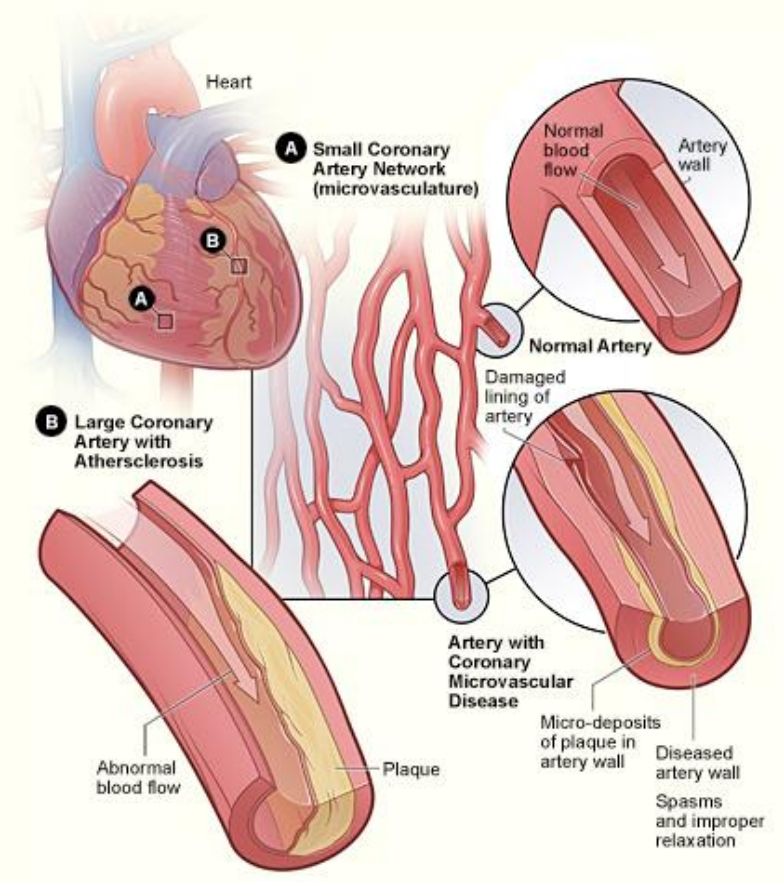


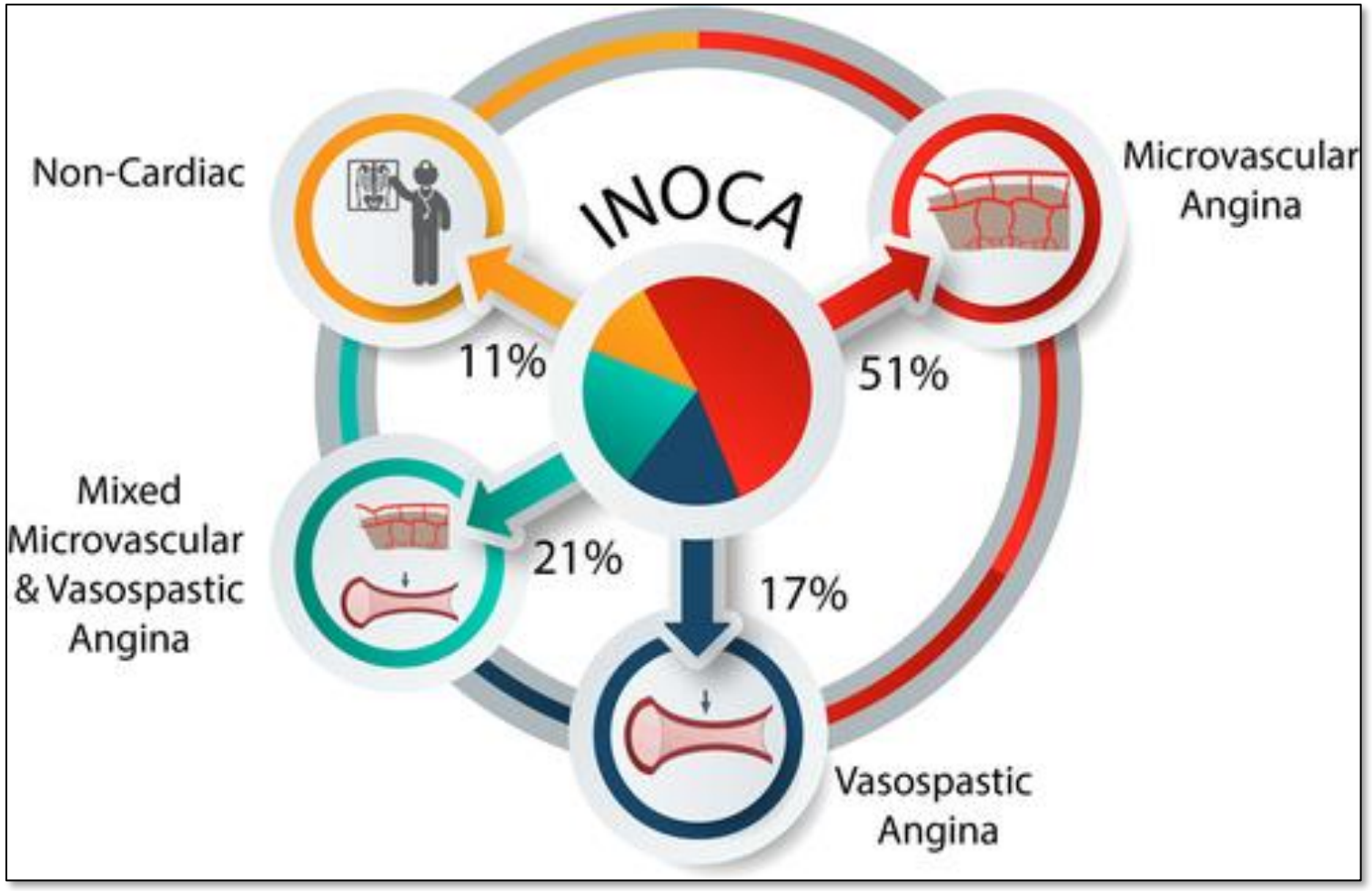


But it's just not that simple

INOCA – A new name for old entity

Ischemia with No Obstructive Coronary Artery Disease



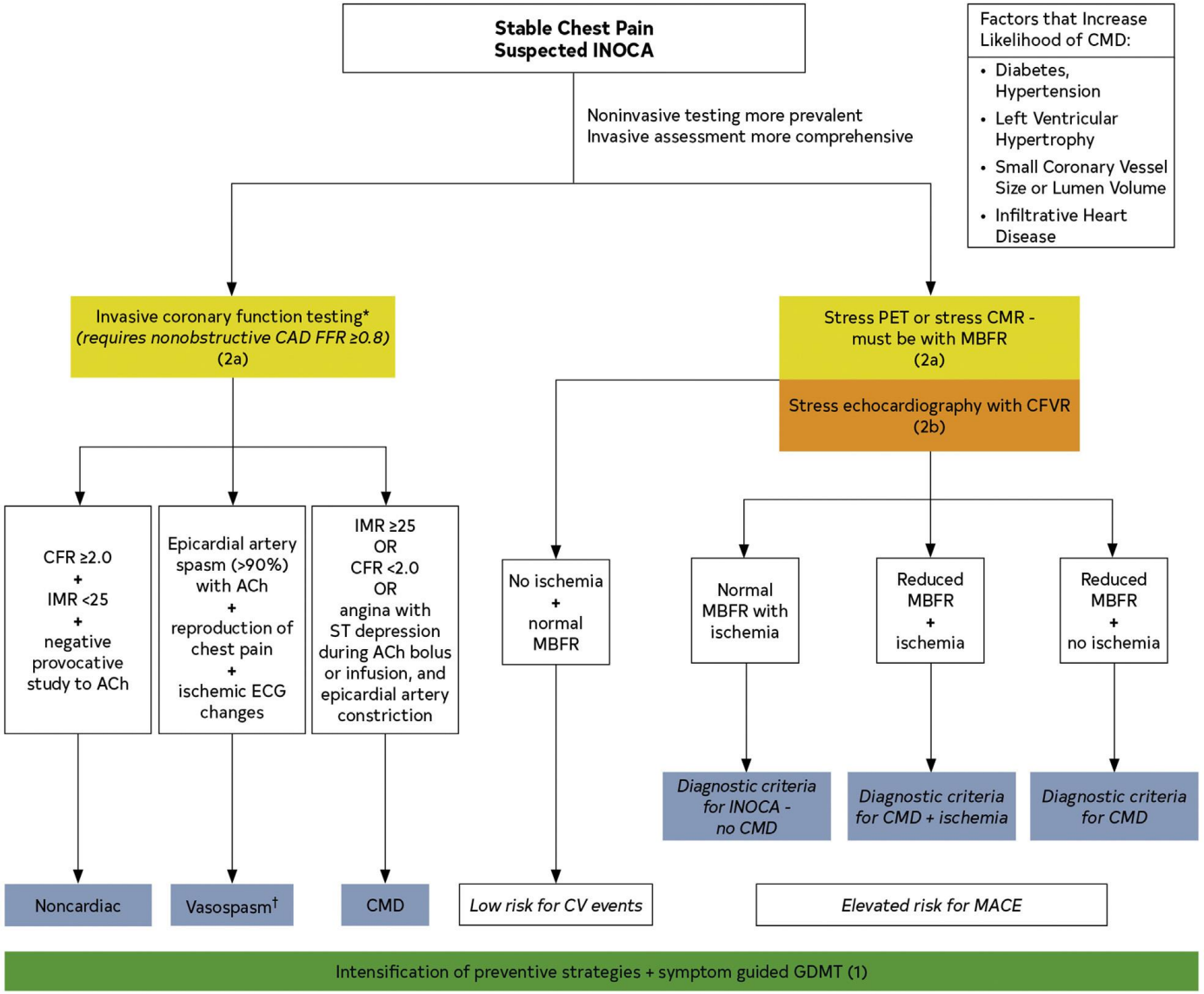


Thomas J. Ford. Circulation: Cardiovascular Interventions., Volume: 12, Issue: 12, DOI: (10.1161/CIRCINTERVENTIONS.119.008126)

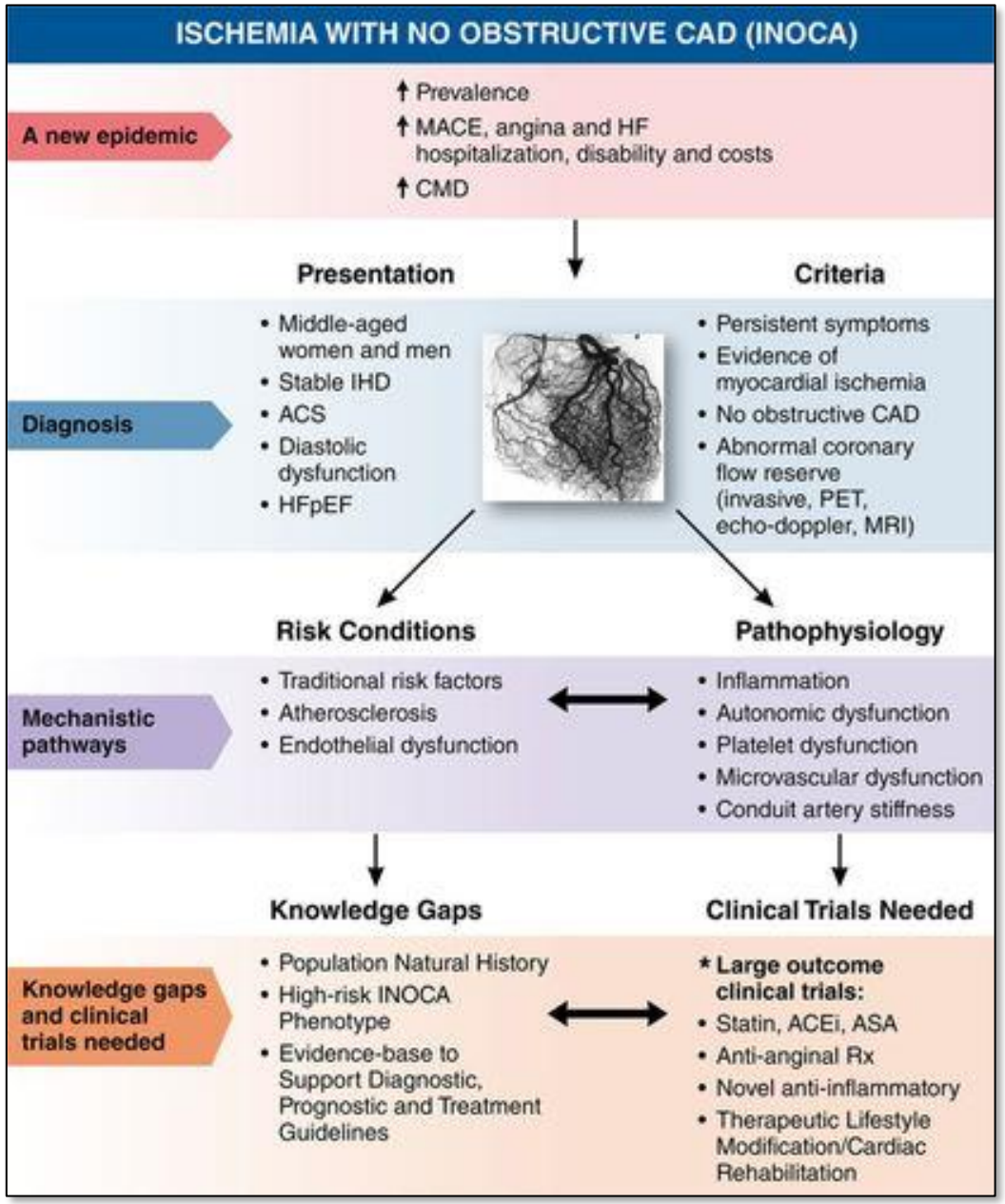
Diagnostic Pathway for Suspected INOCA

In patients with symptoms, don't just stop with a "normal" stress test

Other imaging like **Stress MRI**, **PET**, or **Echo**, and even **coronary angiography** can often provide incremental diagnostic information



INOCA – Management



Potential Therapies for CMD	
Pharmacologic	**Non-Pharmacologic**
• Nitrates • Statins • ACE-I • ACE-I + Aldosterone blockade • Calcium antagonists • Low-dose tricyclic antidepressants • Estrogens • PDE-5 inhibitors • Exercise • L-arginine • Ranolazine • Ivabradine • Ranolazine + Ivabradine • Metformin • Rho-kinase inhibitors • Endothelin receptor blockers	• Exercise • Cognitive behavioral therapy • Transcendental meditation • Transcutaneous electrical nerve stimulation

C. Noel Bairey Merz. Circulation. (INOCA), Volume: 135, Issue: 11, Pages: 1075-1092, DOI: (10.1161/CIRCULATIONAHA.116.024534)

Dual Goals for Management of Stable Ischemic Heart Disease (SIHD)

Prevent MI and Death (Disease Modification)

Improve "Quantity of Life"

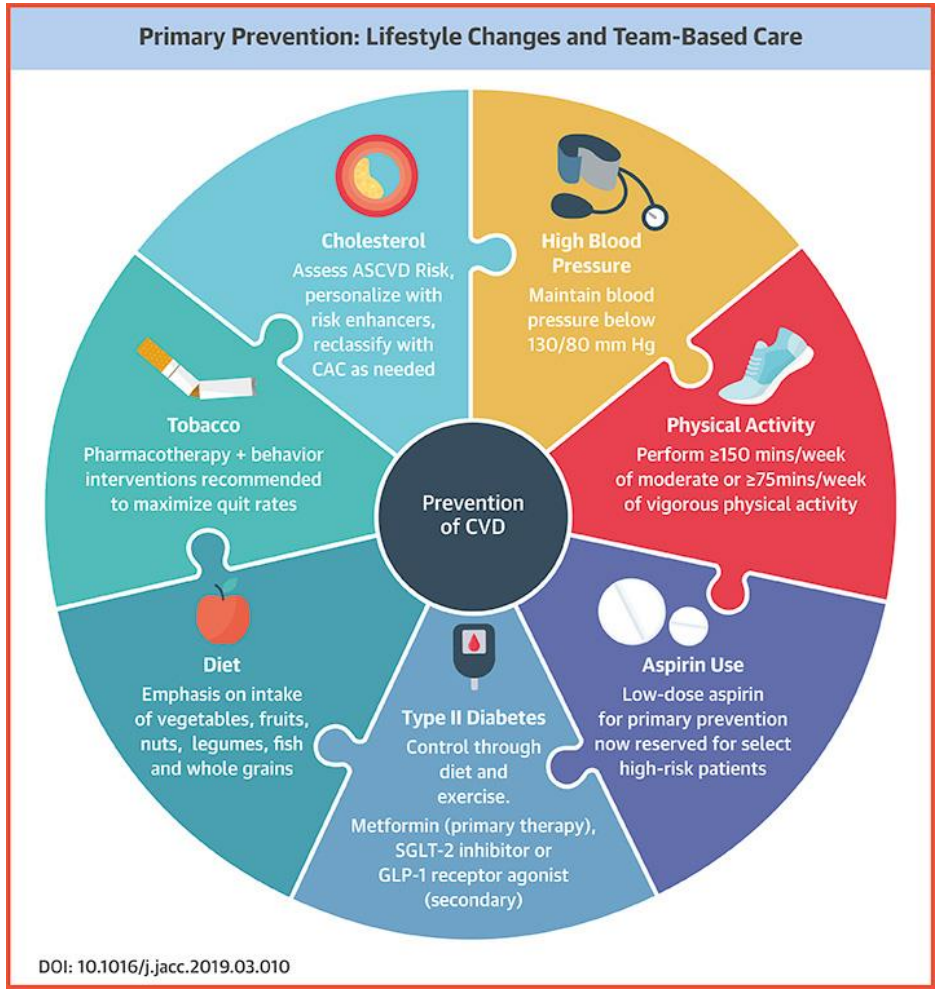
Reduce Ischemia & Relieve Anginal Symptoms

Improve "Quality of Life"

Prevention Goals of Therapy in SIHD

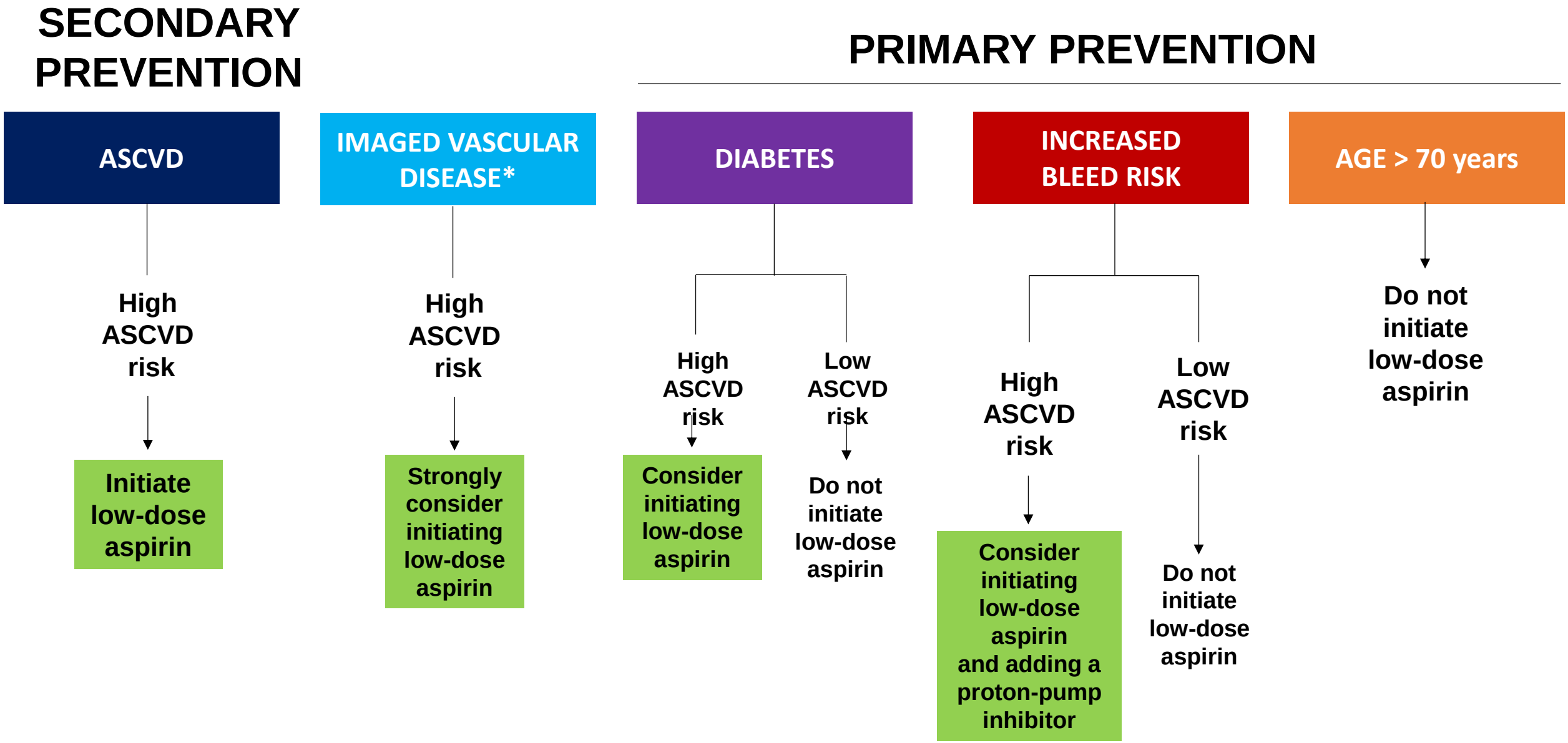
Reduce/stabilize atherosclerotic plaque → ACS/MI/SCD

Primary Prevention



Secondary Prevention

- Antiplatelet Therapy
  - ASA 81 mg or Clopidogrel for life
  - ADP antagonist if recent ACS or stent
- ACEI / ARB (especially if DM, HF, EF <40%, HTN)
- Aggressive Lipid Lowering
- Diabetes Rx with proven CV benefit (GLP1RA and SGLT2i)
- Smoking cessation
- Other Secondary Prevention Measures
  - BP control
  - Weight management
    - Semaglutide for overweight/obese
  - Physical exercise
  - Influenza Vaccine



*Evidence of atherosclerosis on CT scan or vascular ultrasound tests, or an elevated coronary calcium score; ASCVD = atherosclerotic cardiovascular disease.

GLP1RA in Pts with ASCVD, BMI ≥ 27, No Diabetes

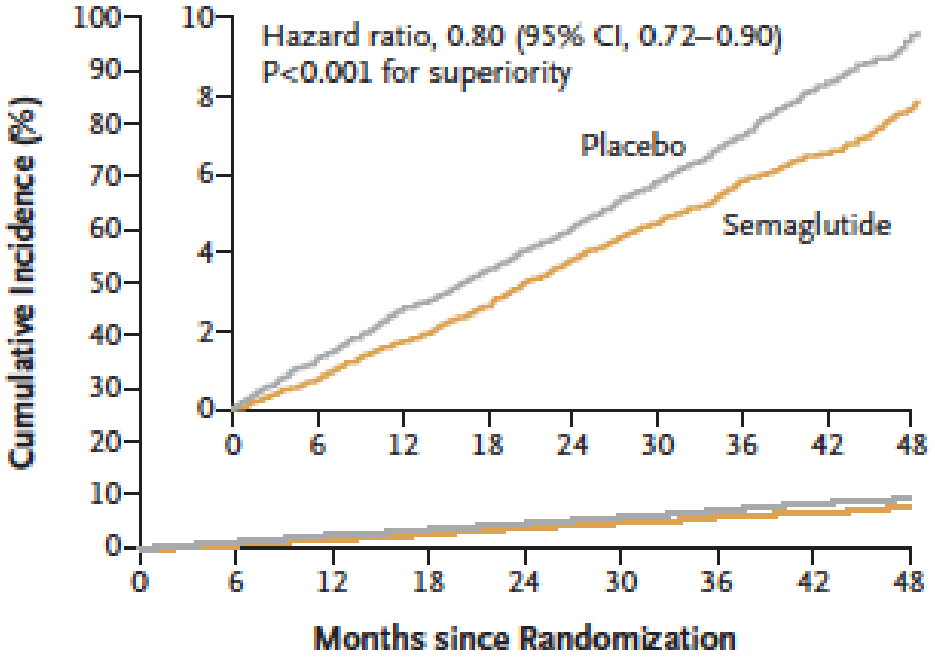
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

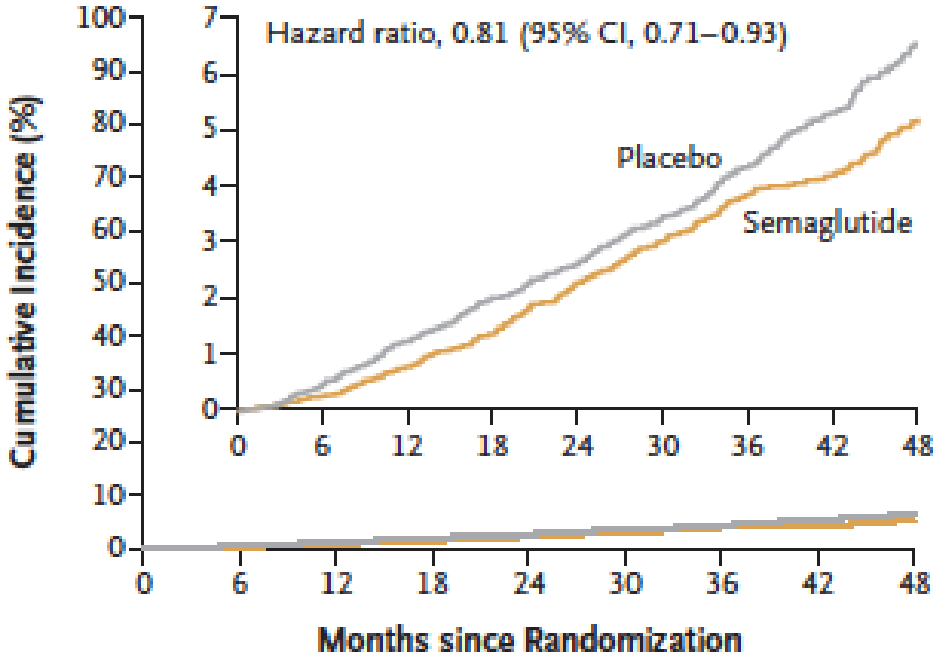
Semaglutide and Cardiovascular Outcomes in Obesity without Diabetes

A. Michael Lincoff, M.D., Kirstine Brown-Frandsen, M.D., Helen M. Colhoun, M.D., John Deanfield, M.D., Scott S. Emerson, M.D., Ph.D., Sille Esbjerg, M.Sc., Søren Hardt-Lindberg, M.D., Ph.D., G. Kees Hovingh, M.D., Ph.D., Steven E. Kahn, M.B., Ch.B., Robert F. Kushner, M.D., Ildiko Lingvay, M.D., M.P.H., Tugce K. Oral, M.D., Marie M. Michelsen, M.D., Ph.D., Jorge Plutzky, M.D., Christoffer W. Tornøe, Ph.D., and Donna H. Ryan, M.D., for the SELECT Trial Investigators*

A Primary Cardiovascular Composite End Point

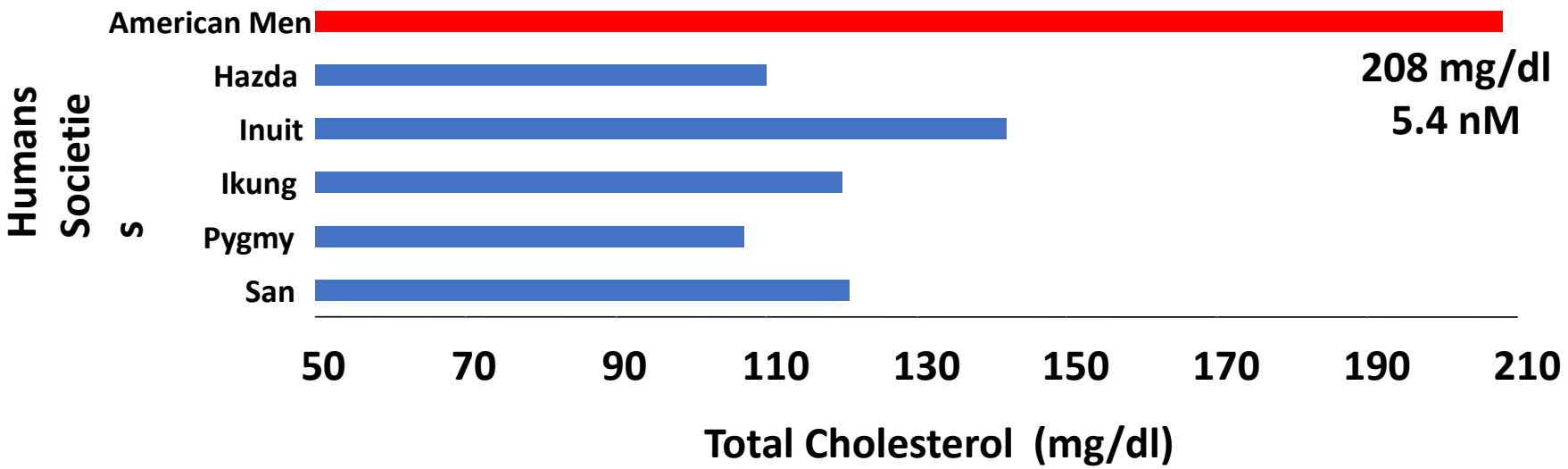


D Death from Any Cause



Cholesterol

First remember that “normal” cholesterol levels are not physiologic



American men

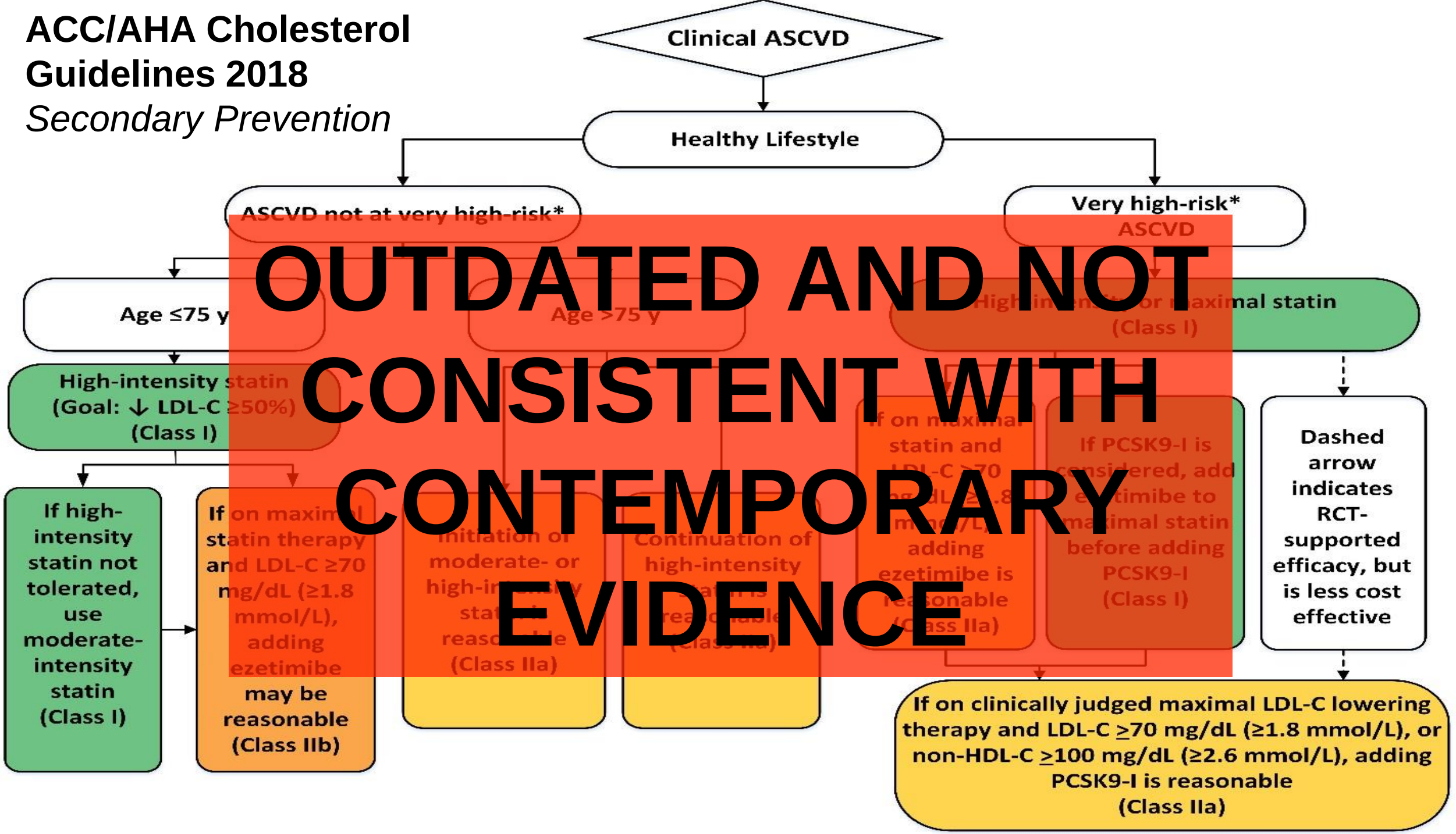
Total Cholesterol levels in small scale subsistence societies only ~100-120 mg/dl

Only modern-day humans have TC >200 mg/dl

Adapted from O’Keefe J. JACC Vol. 43, No. 11, 2004

ACC/AHA Cholesterol Guidelines 2018

Secondary Prevention

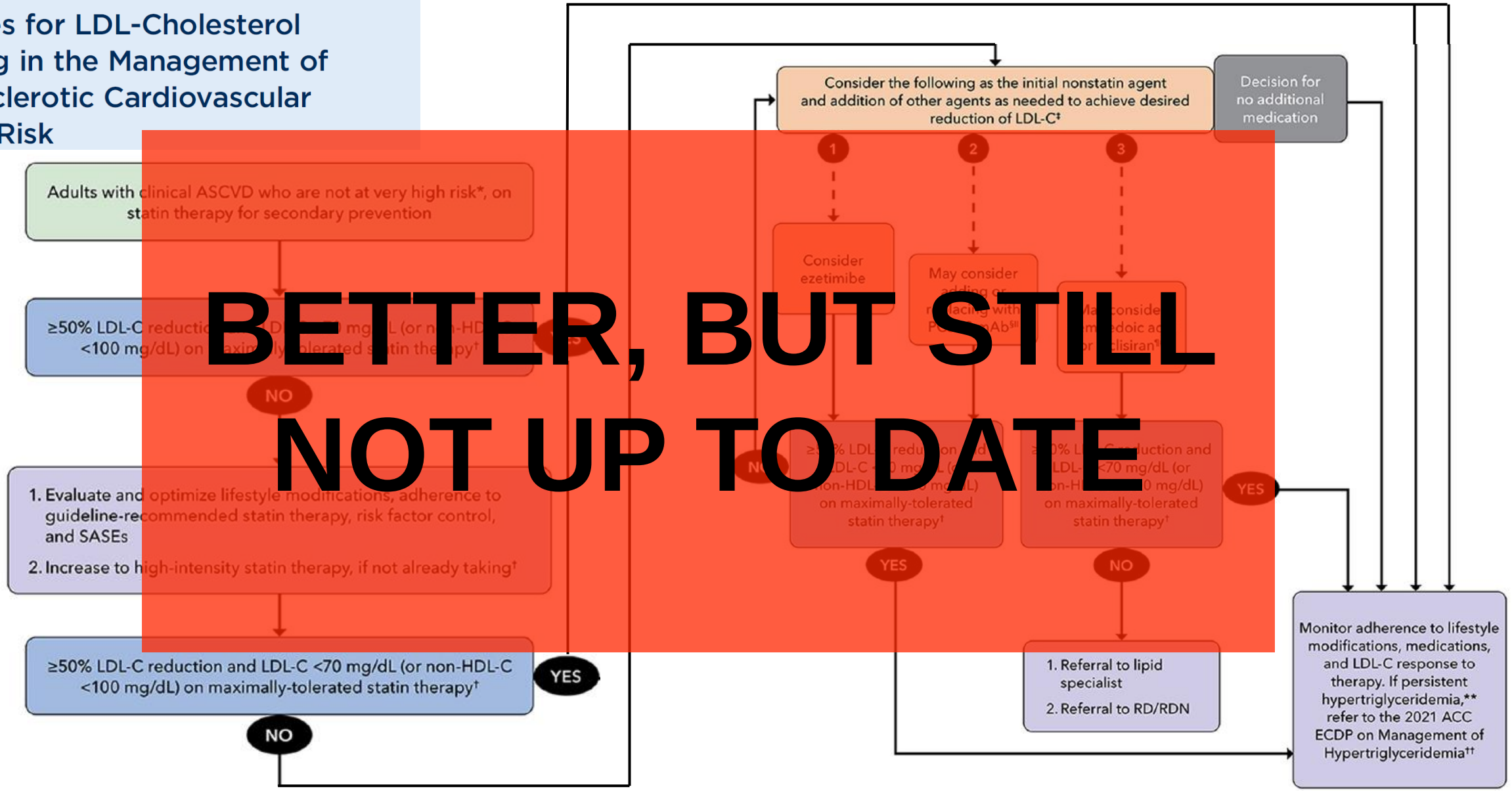


OUTDATED AND NOT CONSISTENT WITH CONTEMPORARY EVIDENCE

EXPERT CONSENSUS DECISION PATHWAY

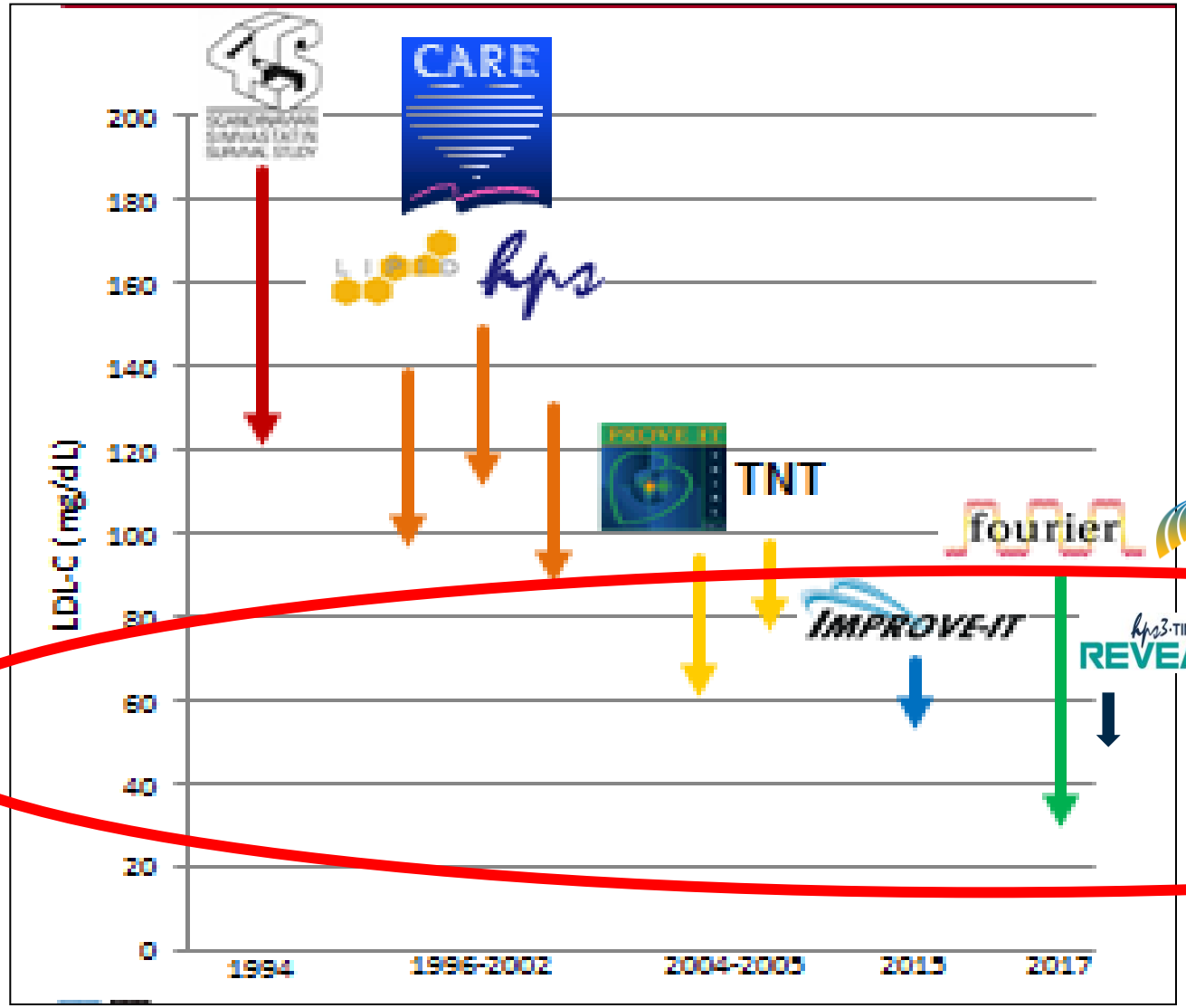
2022 ACC Expert Consensus Decision Pathway on the Role of Nonstatin Therapies for LDL-Cholesterol Lowering in the Management of Atherosclerotic Cardiovascular Disease Risk

ASCVD "Not very high risk" – Goal LDL <70 mg/dl



BETTER, BUT STILL NOT UP TO DATE

LDL-C Levels for Optimal CV Risk Reduction: What We Know Now



High is bad

Average is not good

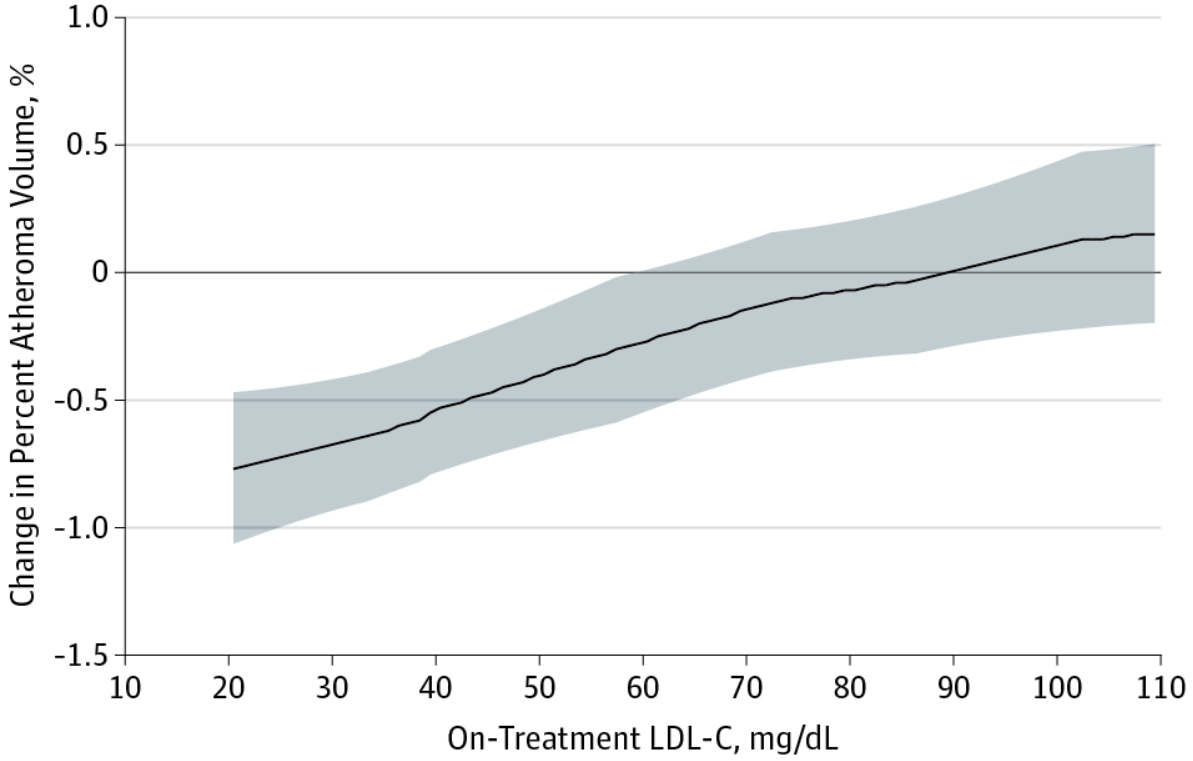
Lower is better

Even lower is even better

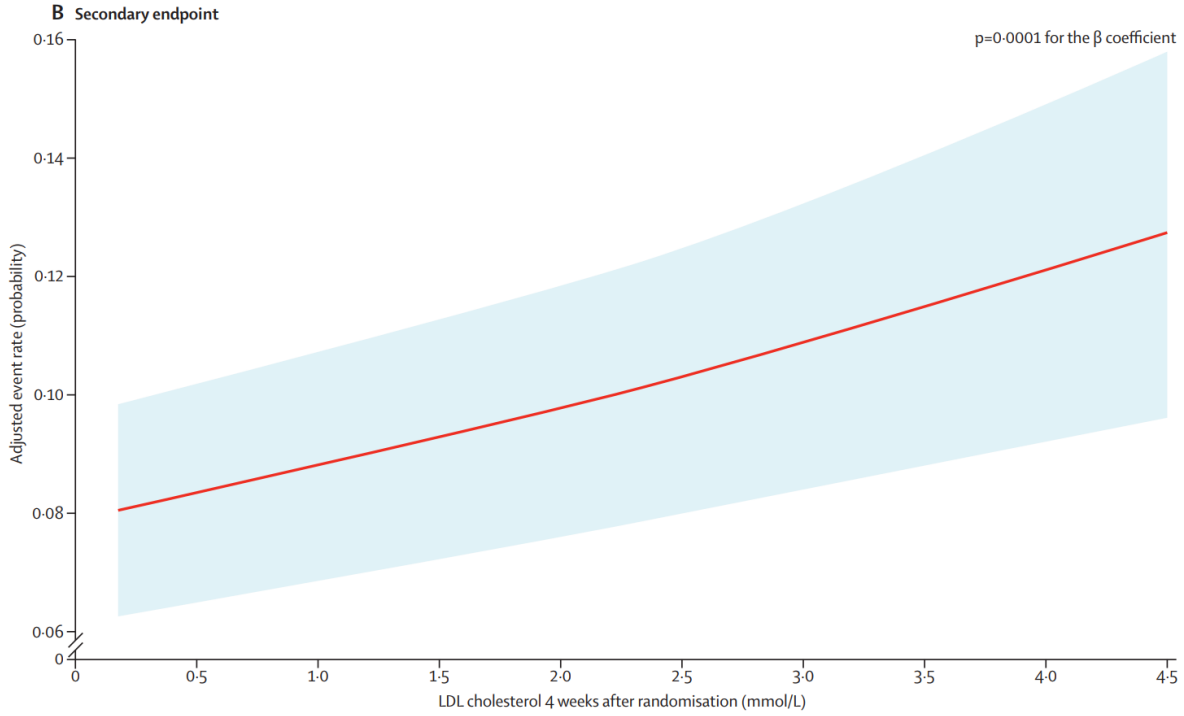
Lowest is best

Achieving Lower LDL-C Modifies Cholesterol Plaques and Clinical Outcomes

Relationship between LDL-C and Percent Atheroma Volume[1]



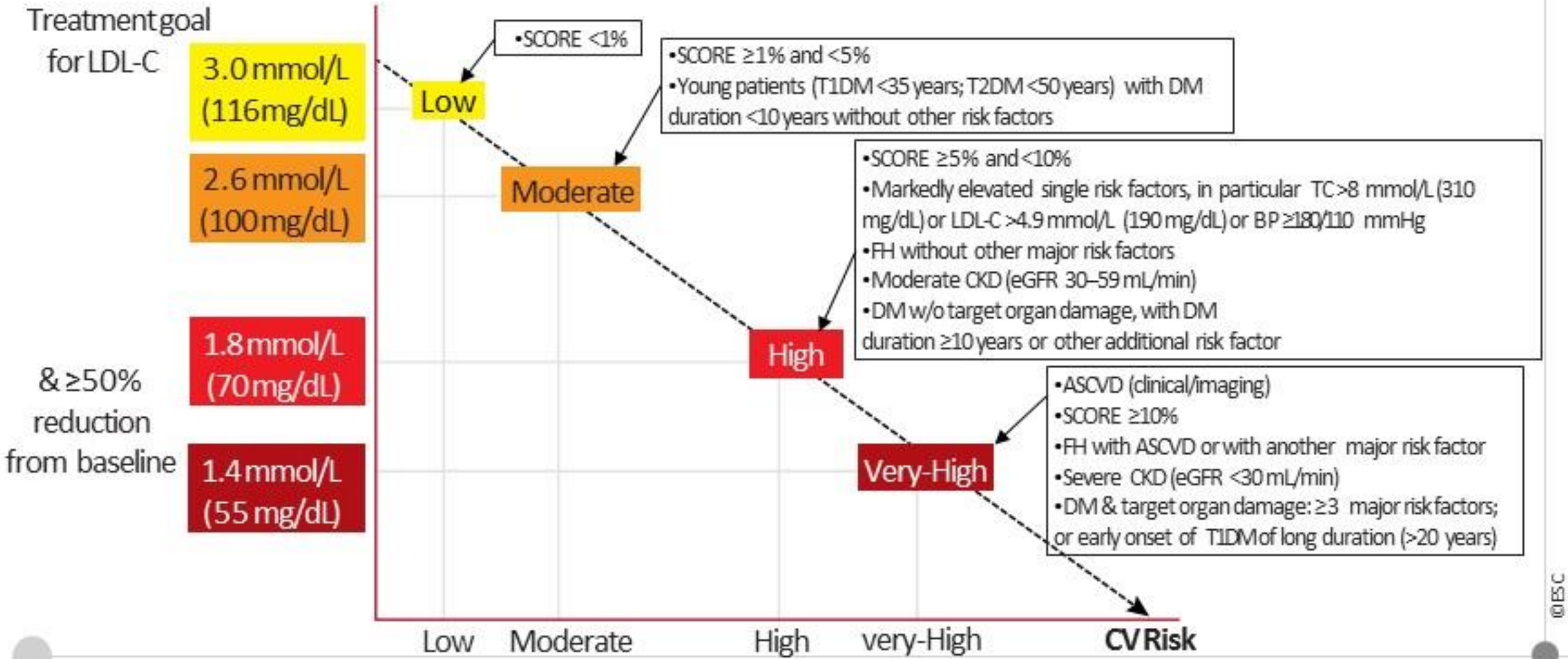
Relationship between LDL-C and Outcomes (CVD, MI, Stroke)[2]



1. *JAMA* 2016;316(22):2373-2384
2. *Lancet* 2017;390: 1962–71

Central Illustration Upper panel Treatment goals for low-density lipoprotein cholesterol (LDL-C) across categories of total cardiovascular disease risk

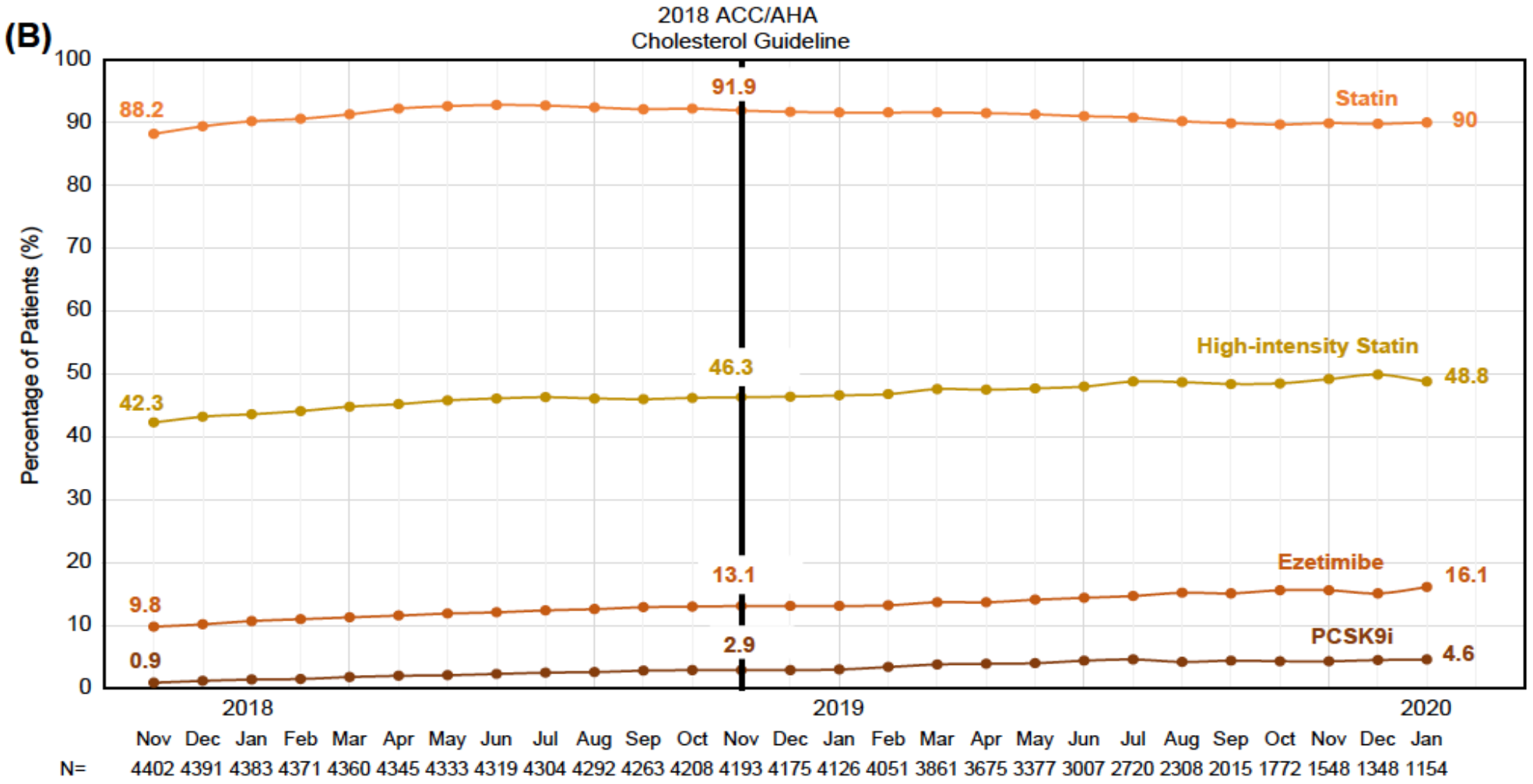
EAS ESC European Society of Cardiology



www.escardio.org/guidelines

2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk (European Heart Journal 2019 -doi: 10.1093/eurheartj/ehz455)

Use of Lipid-Lowering Therapies Over 2 Years in GOULD, a Registry of Patients With Atherosclerotic Cardiovascular Disease in the US



Cannon CP et al. JAMA Cardiol. Published online June 16, 2021. doi:10.1001/jamacardio.2021.1810

Lipids 2025 - “Lower (LDL) is better)

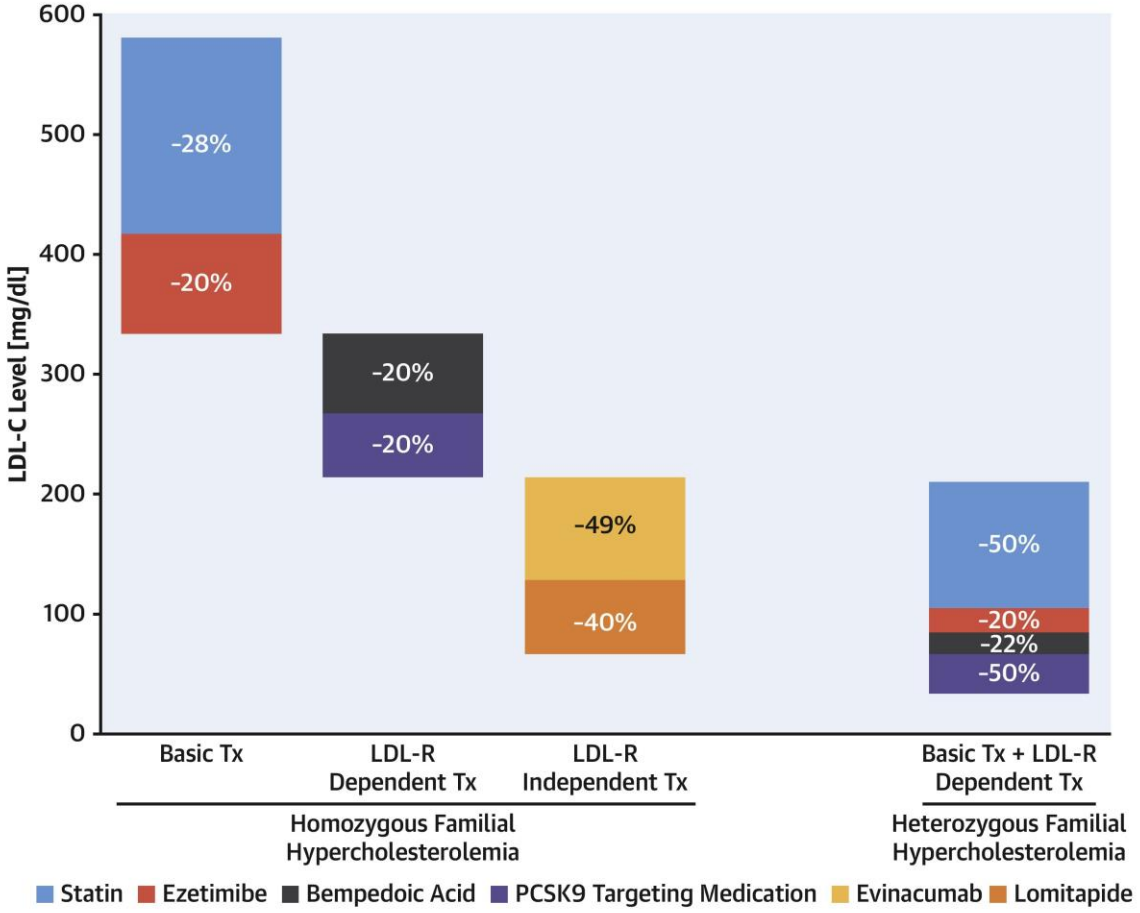
- Primary prevention: statins (or EZE) -> LDL <100 mg/dl
  - Low-Moderate risk → **LDL <100 mg/dl**
  - High risk → **LDL <70 mg/dl**
- SIHD/ASCVD: statin +/- ezetimibe +/- PCSK9 +/- BDA
  - ASCVD → **LDL <55 mg/dl**
- Statin intolerance: rosuva 2.5 tiw, EZE, early PCSK9
- Check Trig and if >135, add icosapent ethyl 4gm daily
- Check Lp(a) –> increased risk and now clinical trials for Lp(a) reduction

My Approach to Elevated Trigs in 2025

- Diet, lifestyle, no EtOH
- Evaluate for MASH
- If DM -> any GLP1RA
- If no DM but overweight/obese -> Sema or Tirzepatide
- If mod-trigs and CAD also consider icosapent ethyl b/c of CV benefit

Beyond Statins

CENTRAL ILLUSTRATION: Simulation of Maximal Pharmacological Low-Density Lipoprotein Cholesterol Reduction



Brandts, J. et al. J Am Coll Cardiol. 2021;78(18):1831-1843.

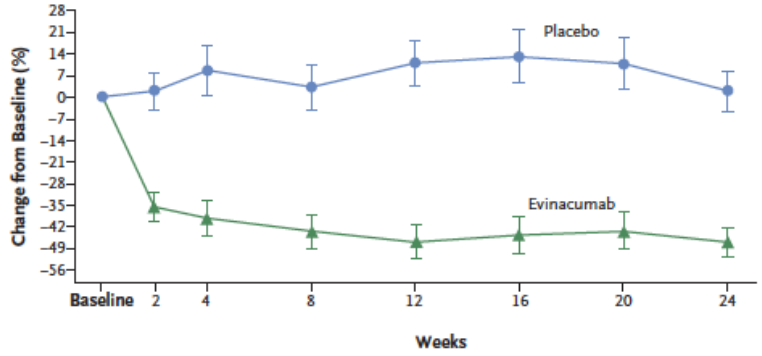
The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 AUGUST 20, 2020 VOL. 383 NO. 8

Evinacumab for Homozygous Familial Hypercholesterolemia

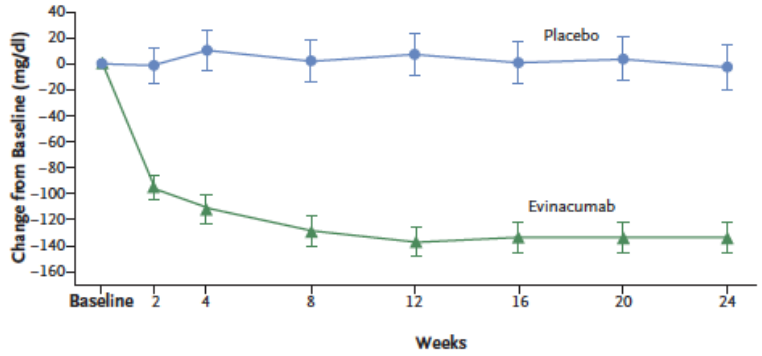
Frederick J. Raal, M.D., Ph.D., Robert S. Rosenson, M.D., Laurens F. Reeskamp, M.D., G. Kees Hovingh, M.D., Ph.D., John J.P. Kastelein, M.D., Ph.D., Paolo Rubba, M.D., Shazia Ali, Pharm.D., Poulabi Banerjee, Ph.D., Kuo-Chen Chan, Ph.D., Daniel A. Gipe, M.D., Nagwa Khilla, M.S., Robert Pordy, M.D., David M. Weinreich, M.D., George D. Yancopoulos, M.D., Ph.D., Yi Zhang, Ph.D., and Daniel Gaudet, M.D., Ph.D., for the ELIPSE HoFH Investigators*

A Percent Change in LDL Levels



No. at Risk								
Placebo	22	19	20	21	20	20	20	21
Evinacumab	43	38	43	42	42	40	43	43

B Absolute Change in LDL Levels



No. at Risk								
Placebo	22	19	20	21	20	20	20	21
Evinacumab	43	38	43	42	42	40	43	43

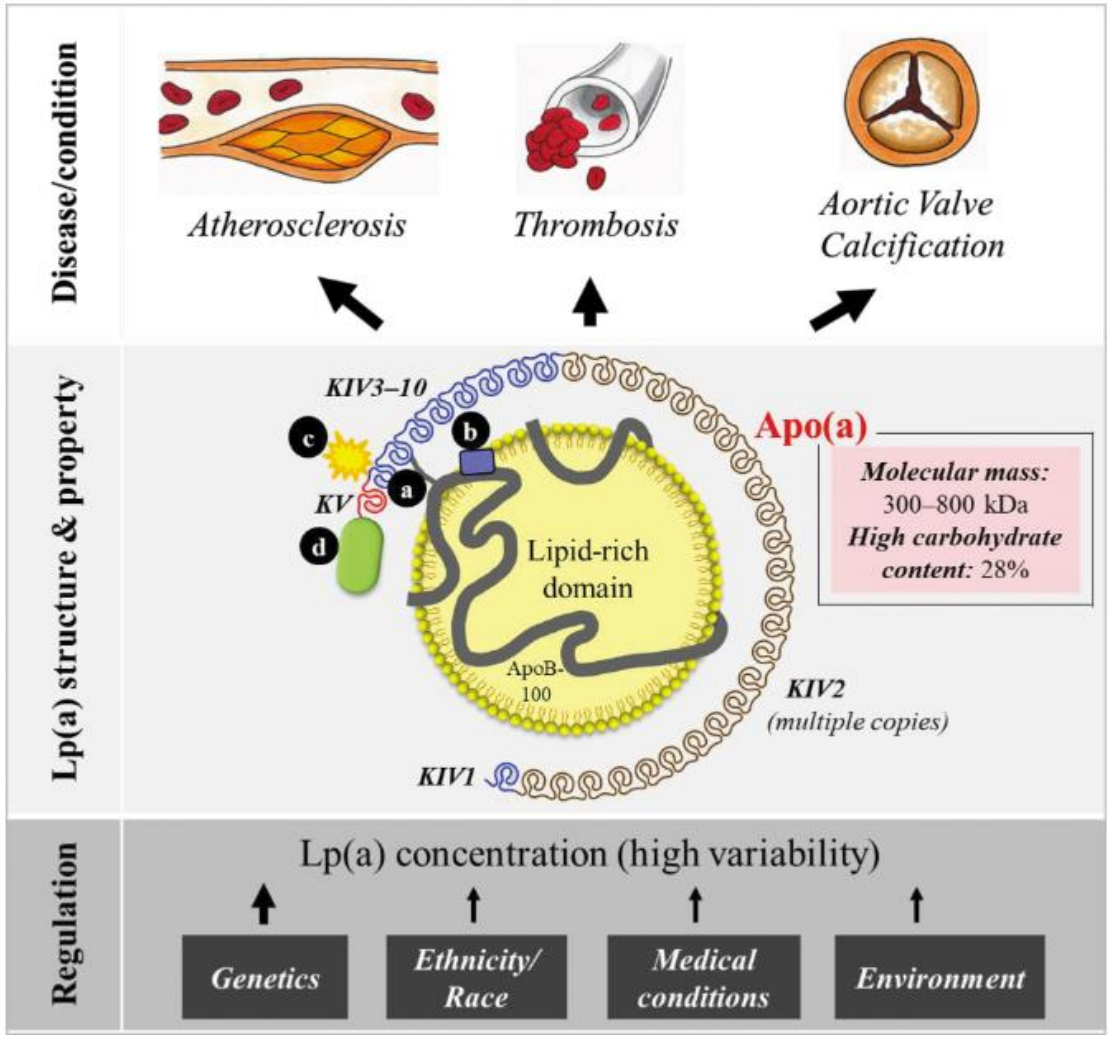
Arteriosclerosis, Thrombosis, and Vascular Biology

AHA SCIENTIFIC STATEMENT

Lipoprotein(a): A Genetically Determined, Causal, and Prevalent Risk Factor for Atherosclerotic Cardiovascular Disease: A Scientific Statement From the American Heart Association

The International Atherosclerosis Society endorses this statement.

- High levels of lipoprotein(a) [Lp(a)], an apoB100-containing lipoprotein, are an independent and causal risk factor for atherosclerotic CV disease through increased atherogenesis, inflammation, and thrombosis.
- Lp(a) is predominantly a monogenic cardiovascular risk determinant, with ≈70% to ≥90% of interindividual heterogeneity in levels being genetically determined.
- The 2 major protein components of Lp(a) particles are apoB100 and apolipoprotein(a).
- Lp(a) remains a risk factor for cardiovascular disease development even in the setting of effective LDL cholesterol reduction.



Lp(a) and CV Risk

Lipoprotein(a) Concentration and the Risk of Coronary Heart Disease, Stroke, and Nonvascular Mortality

JAMA, July 22/29, 2009—Vol 302, No. 4

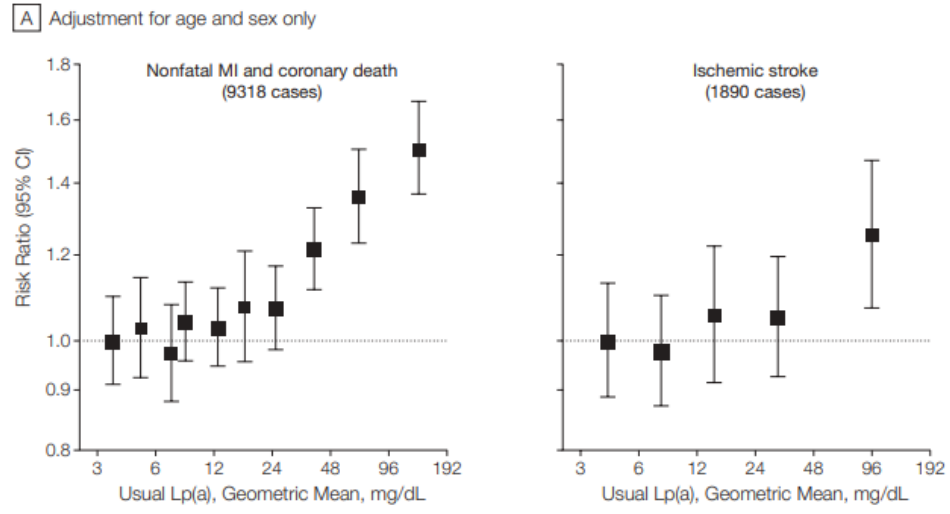
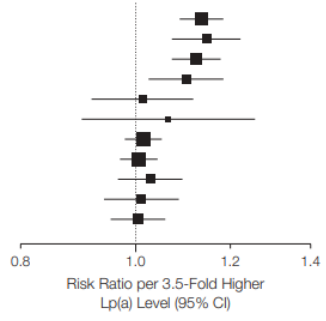


Figure 3. Risk Ratios for Vascular and Nonvascular Outcomes per 3.5-Fold (1-SD) Higher Usual Lp(a) Level, Adjusted for Cardiovascular Risk Factors

Outcome	No. of Studies	No. of Individuals	No. of Cases	Risk Ratio (95% CI)
Nonfatal MI and coronary death	30	106 645	8362	1.13 (1.09-1.18)
Coronary death[a]	24	72 683	2159	1.14 (1.07-1.22)
Nonfatal MI[a]	26	102 221	6045	1.12 (1.07-1.18)
Ischemic stroke	13	69 539	1684	1.10 (1.02-1.18)
Unclassified stroke	12	48 407	680	1.01 (0.92-1.12)
Hemorrhagic stroke	9	56 165	285	1.06 (0.90-1.26)
Nonvascular death	25	102 268	7268	1.01 (0.98-1.05)
All cancer death	20	91 424	3492	1.00 (0.97-1.04)
Smoking-related cancer death	16	63 555	1340	1.03 (0.97-1.09)
Other cancer death	19	91 307	2140	1.01 (0.94-1.08)
Other nonvascular death	22	100 378	3745	1.00 (0.95-1.06)



ORIGINAL ARTICLE

Genetic Variants Associated with Lp(a) Lipoprotein Level and Coronary Disease

Robert Clarke, F.R.C.P., John F. Peden, Ph.D., Jemma C. Hopewell, Ph.D., Theodosios Kyriakou, Ph.D., Anuj Goel, M.Sc., Simon C. Heath, Ph.D., Sarah Parish, D.Phil., Simona Barlera, M.S., Maria Grazia Franzosi, Ph.D., Stephan Rust, Ph.D., Derrick Bennett, Ph.D., Angela Silveira, Ph.D., Anders Malarstig, Ph.D., Fiona R. Green, Ph.D., Mark Lathrop, Ph.D., Bruna Gigante, M.D., Karin Leander, Ph.D., Ulf de Faire, M.D., Udo Seedorf, Ph.D., Anders Hamsten, F.R.C.P., Rory Collins, F.R.C.P., Hugh Watkins, F.R.C.P., and Martin Farrall, F.R.C.Path., for the PROCARDIS Consortium*

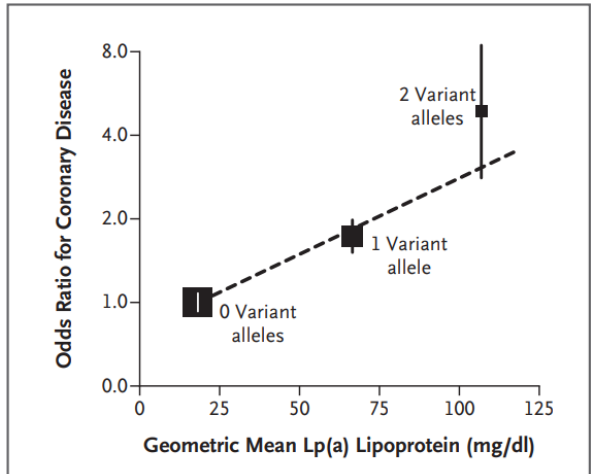
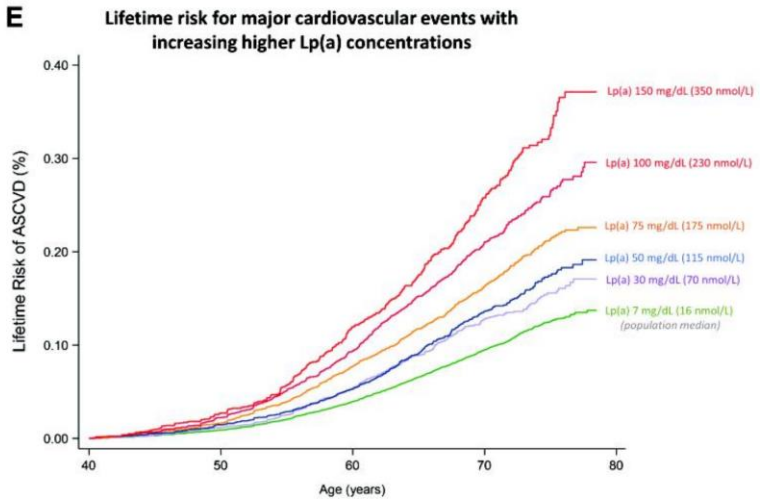


Figure 3. Association of the *LPA* Genotype Score with the Lp(a) Lipoprotein Level and the Risk of Coronary Disease in the PROCARDIS Cohort.

The odds ratios (squares, with the size inversely proportional to the sampling variation) are for the association of the *LPA* genotype score (no variant alleles, one variant allele, or two variant alleles) with the risk of coronary disease, as measured with the use of "floating absolute risks" which summarize the sampling variation for the three genotype scores without the selection of an arbitrary baseline genotype score. The vertical lines indicate 95% confidence intervals.

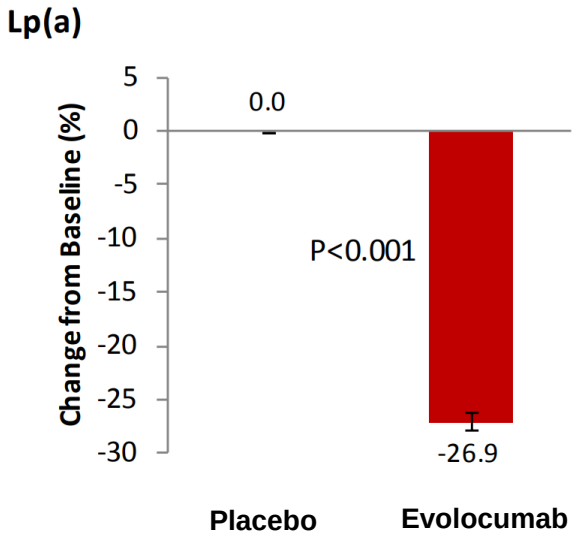
Lipoprotein(a), CV Outcomes, and PCSK9 inhibitors

Lifetime risk of CV Events by Lp(a)



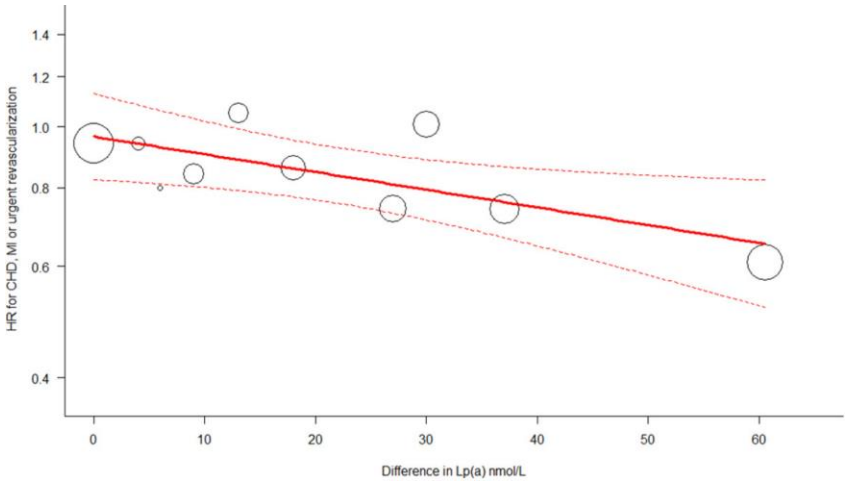
Eur Heart J. 2022;43(39):3925-3946

PCSK9i reduces Lp(a) levels



Circulation. 2019;139:1483–1492

Lower Lp(a) associated with↓ CV events



Circulation. 2019;139:1483–1492

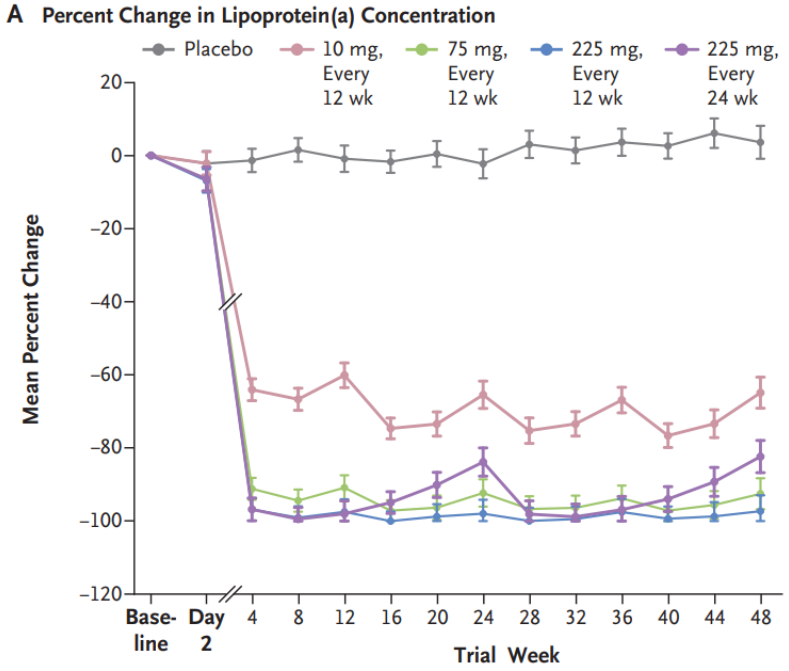
Targeted Lp(a) Therapy

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Small Interfering RNA to Reduce Lipoprotein(a) in Cardiovascular Disease

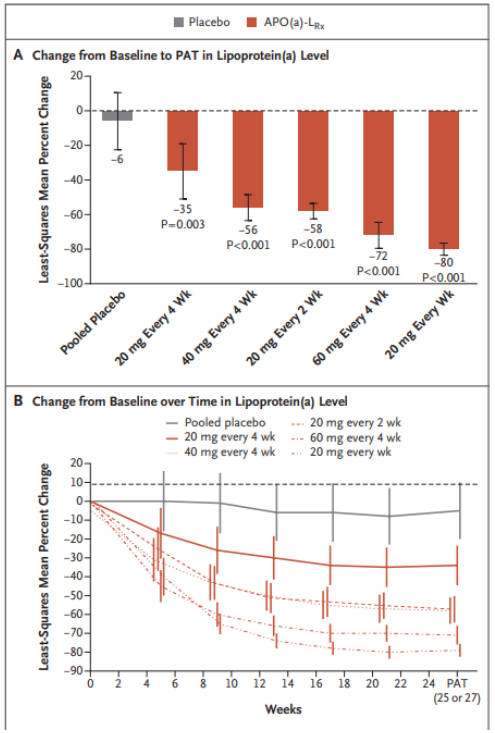
Michelle L. O'Donoghue, M.D., M.P.H., Robert S. Rosenson, M.D., Baris Gencer, M.D., M.P.H., J. Antonio G. López, M.D., Norman E. Lepor, M.D., Seth J. Baum, M.D., Elmer Stout, M.D., Daniel Gaudet, M.D., Ph.D., Beat Knusel, Ph.D., Julia F. Kuder, M.A., Xinhui Ran, M.S., Sabina A. Murphy, M.P.H., Huei Wang, Ph.D., You Wu, Ph.D., Helina Kassahun, M.D., and Marc S. Sabatine, M.D., M.P.H., for the OCEAN(a)-DOSE Trial Investigators*



ORIGINAL ARTICLE

Lipoprotein(a) Reduction in Persons with Cardiovascular Disease

Sotirios Tsimikas, M.D., Ewa Karwatowska-Prokopczuk, M.D., Ph.D., Ioanna Gouni-Berthold, M.D., Jean-Claude Tardif, M.D., Seth J. Baum, M.D., Elizabeth Steinhagen-Thiessen, M.D., Michael D. Shapiro, D.O., Erik S. Stroes, M.D., Patrick M. Moriarty, M.D., Børge G. Nordestgaard, M.D., D.M.Sc., Shuting Xia, M.S., Jonathan Guerriero, M.B.A., Nicholas J. Viney, B.Sc., Louis O'Dea, M.B., B.Ch., B.A.O., and Joseph L. Witztum, M.D., for the AKCEA-APO(a)-L_{Rx} Study Investigators*

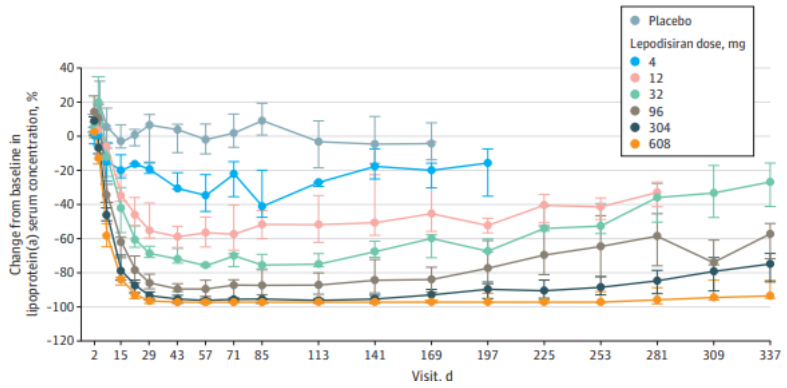


JAMA | Original Investigation

Lepodisiran, an Extended-Duration Short Interfering RNA Targeting Lipoprotein(a)
A Randomized Dose-Ascending Clinical Trial

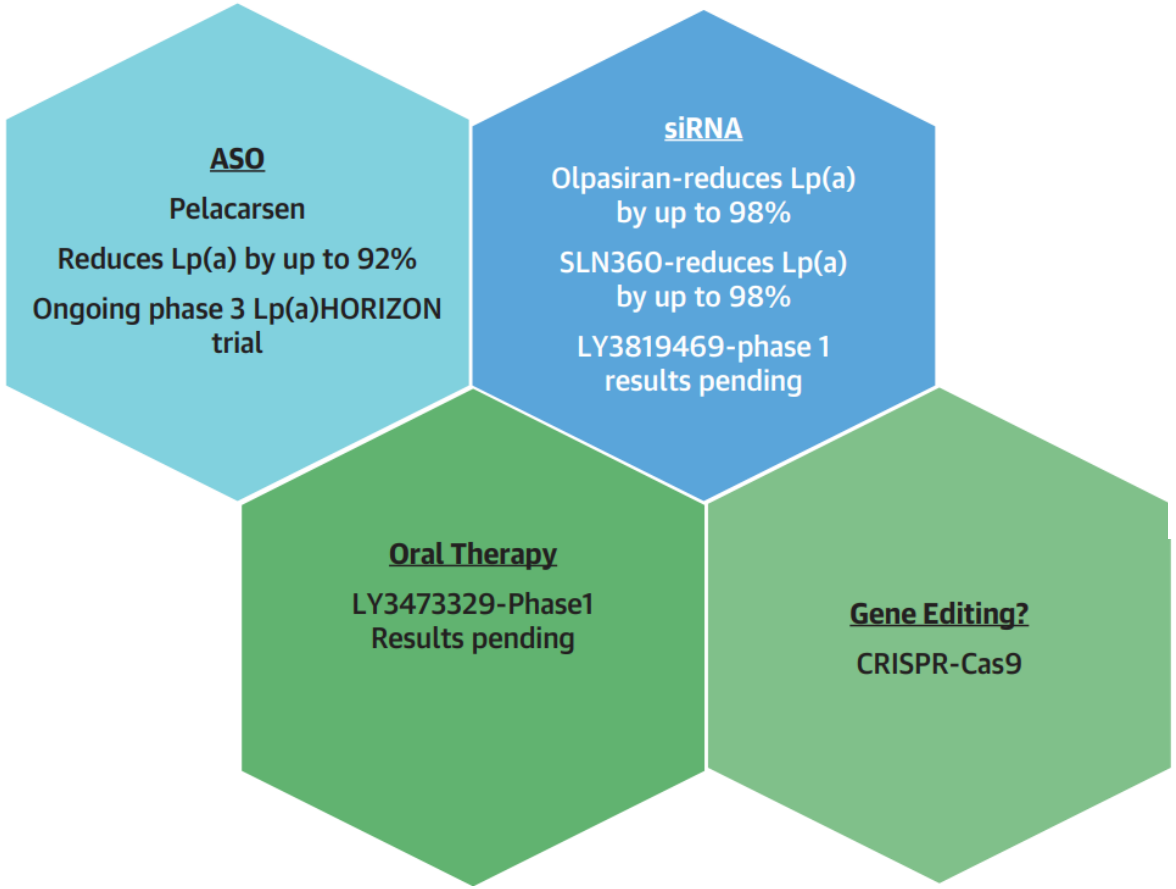
Steven E. Nissen, MD; Helle Linnebjerg, PhD; Xi Shen, PhD; Kathy Wolski, MPH; Xiaosu Ma, PhD; Shufen Lim, PhD; Laura F. Michael, PhD; Giacomo Ruotolo, MD, PhD; Grace Gribble, MS; Ann Marie Navar, MD, PhD; Stephen J. Nicholls, MBBS, PhD

Figure 3. Percentage Change in Levels of Lipoprotein(a) From Baseline to 336 Days (48 Weeks) After Administration



Lp(a) Therapeutic Studies

FIGURE 1 Emerging Therapeutics for Lp(a)



Pelacarsen

Lp(a)HORIZON

- Inclusion Criteria: History of MI, History of ischemic stroke, history of PAD
- Baseline Cut-Off Lp(a) Value: 175 nmol/L
- Primary Outcome: Composite of cardiovascular death, MI, stroke, and urgent coronary revascularization requiring hospitalization

Olpasiran

OCEAN(a)

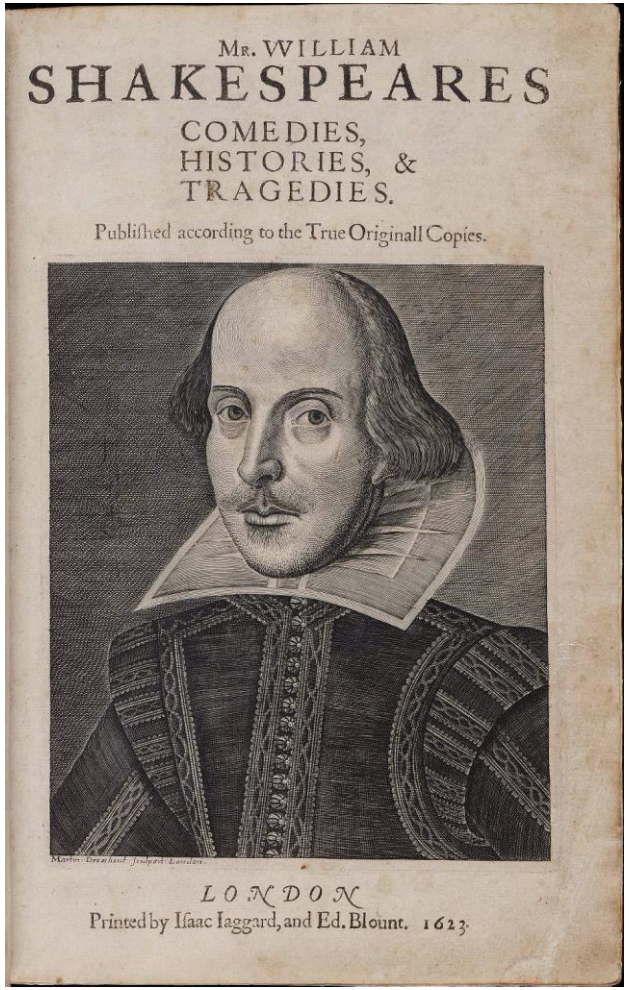
- Inclusion Criteria: History of MI, PCI with a high-risk condition
- Baseline Cut-Off Lp(a) Value: 200 nmol/L
- Primary Outcome: Composite of CHD death, MI, or urgent revascularization

Lepodisiran

ACCLAIM-Lp(a)

- Inclusion Criteria: 1) Established ASCVD with history of an event or revascularization and 2) Individuals 55 years or older who are at risk for a first CV event based on risk factors
- Baseline Cut-off Lp(a) Value: 175 nmol/L
- Primary Outcome: Composite of cardiovascular death, MI., stroke, or urgent revascularization.

The Eternal Question in SIHD



"To cath, or not to cath, that is the question…":

Revascularization - Perception

Make patient live longer

Make patient feel better

Revascularization - Reality

Make patient live longer

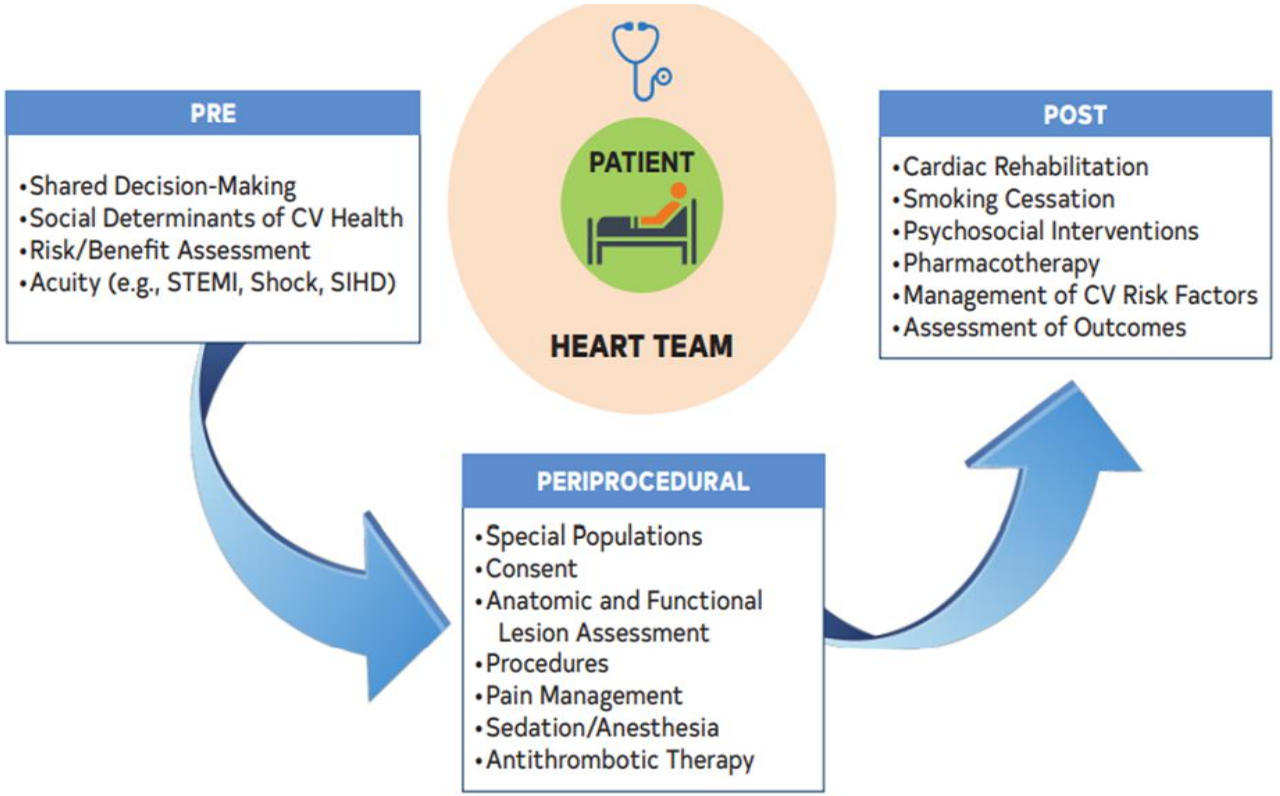
Make patient feel better

Key Questions about Revascularization

- Is angina (or anginal equivalent) refractory to medical therapy?
- Is there Left Main Disease?
- How complex is the CAD (eg, SYNTAX score)
- Diabetes?
- LVEF <50%
- Suitability for PCI or CABG
- Patient preference

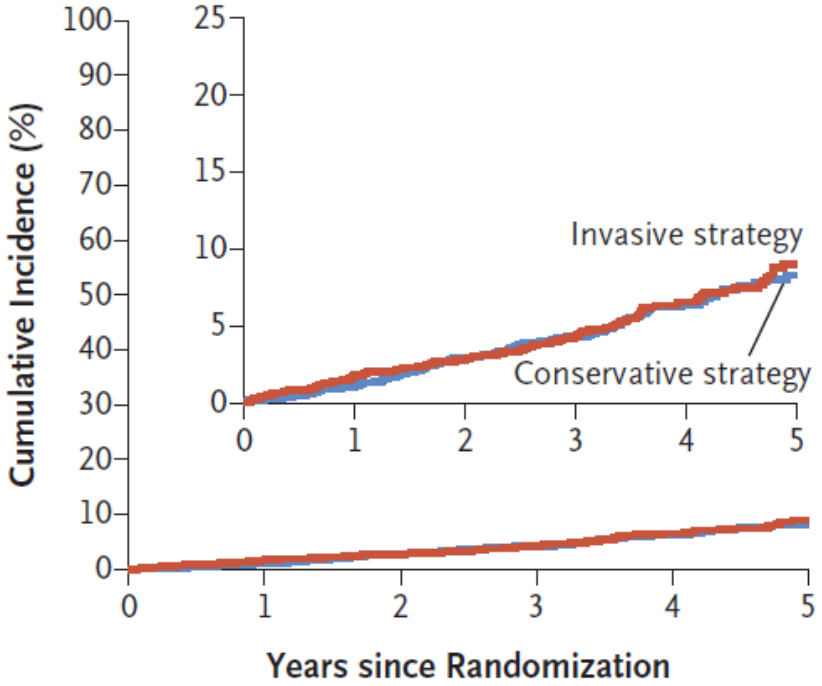
Patient Centered – Heart Team

COR	LOE	RECOMMENDATION
1	B-NR	1. In patients for whom the optimal treatment strategy is unclear, a Heart Team approach that includes representatives from interventional cardiology, cardiac surgery, and clinical cardiology is recommended to improve patient outcomes (1-7).



ISCHEMIA Trial

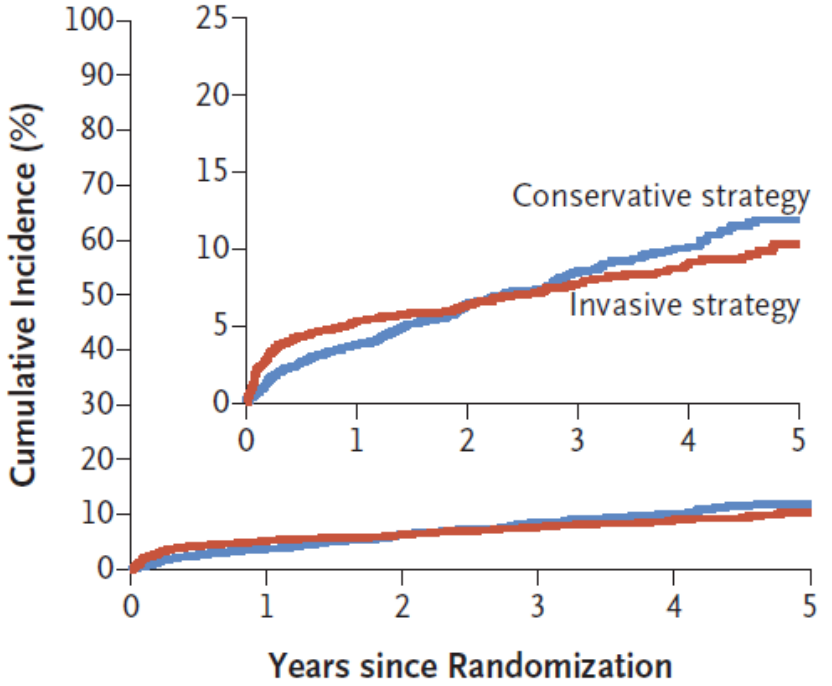
C Death from Any Cause



No. at Risk

Conservative strategy	2591	2548	2065	1445	844	349
Invasive strategy	2588	2518	2061	1431	827	317

D Myocardial Infarction

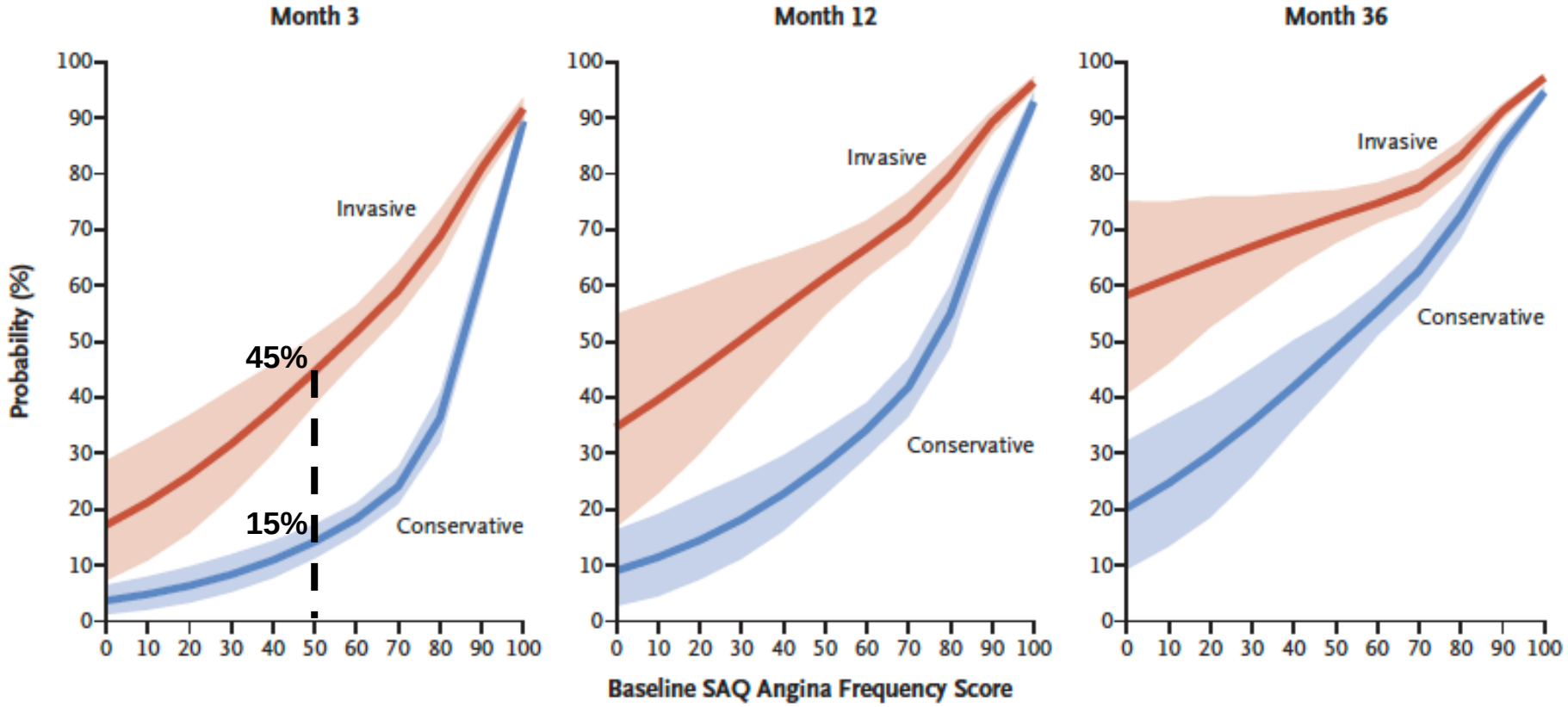


No. at Risk

Conservative strategy	2591	2452	1931	1321	747	298
Invasive strategy	2588	2379	1931	1313	742	283

Maron. NEJM. 2020.

Probability of Being Angina-Free



Maron DJ. NEJM. 2020382:1395-

Summary of "Disease Altering" Interventions in SIHD

- Greatest evidence for life-prolonging or MI-reducing therapy is with optimal medical therapy
- Revascularization is very good for reducing angina and minimizing the need for recurrent coronary interventions.
- But, except in small, high-risk populations, immediate revascularization does not prolong life or reduce the risk of future MI

Key References

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- Lloyd-Jones D, et al. 2022 ACC Expert Consensus Decision Pathway on the Role of Nonstatin Therapies for LDL-Cholesterol Lowering in the Management of Atherosclerotic Cardiovascular Disease Risk. J Am Coll Cardiol. 2022 Oct, 80 (14) 1366–1418.

Question 1

A 56 year-old man presents with symptoms suggestive of exertional angina. Symptoms of chest pressure begin after moderate exertion and resolve with rest and are associated with SOB. There has been no rest pain. He has a history of hypertension and dyslipidemia for which he is taking losartan and simvastatin. His exam does not reveal any signs of volume overload or heart failure. His ECG has mild PR prolongation, LVH with strain pattern but no Q-waves. What is the next most appropriate test?

a) **Echocardiography**
b) **Exercise stress test**
c) **Exercise stress test with radionuclide images**
d) **Pharmacologic stress test**
e) **Invasive coronary angiogram**
f) **No testing needed at this time**

Question 1

Answer: C

This patient has symptoms typical of cardiac ischemia, thus the pre-test probability of CAD is moderate to high, but hr otherwise appears to be at relatively low risk given his co-morbidities. Non-invasive functional imaging for myocardial ischemia or coronary CTA is recommended as the initial test to diagnose CAD in symptomatic patients in whom obstructive CAD cannot be excluded by clinical assessment alone.

Exercise test is attractive because of the important prognostic data regarding exercise capacity, time to symptoms, and HR and BP response. In patients with an uninterpretable ECG, radionuclide imaging is needed. If this patient has documented ischemia on stress testing, then an assessment of left ventricular function is indicated for further risk stratification.