Update In Preventive Cardiology

R. Todd Hurst, MD, FACC, FASE

Resident Conference

August 17, 2021

Disclosure

Relevant Financial Relationship(s)

- None

Off Label Usage

- None

CDC's National Center for Chronic Disease Prevention and Health Promotion (NCCDPHP)

CHRONIC DISEASES IN AMERICA

6 IN 10

Adults in the US have a **chronic disease**

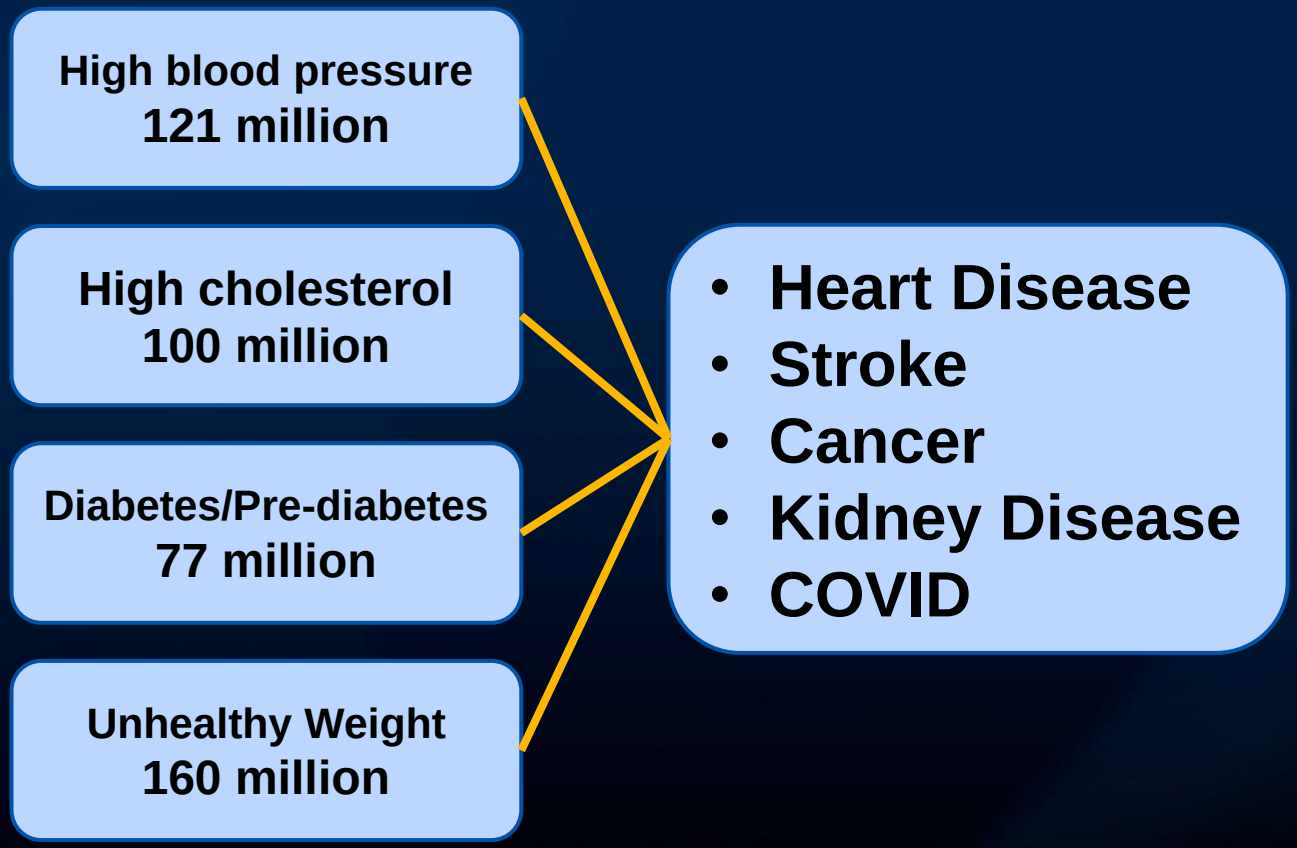
4 IN 10

Adults in the US have **two or more**

THE LEADING CAUSES OF DEATH AND DISABILITY and Leading Drivers of the Nation's **$3.3 Trillion** in Annual Health Care Costs

Cardiometabolic Disease

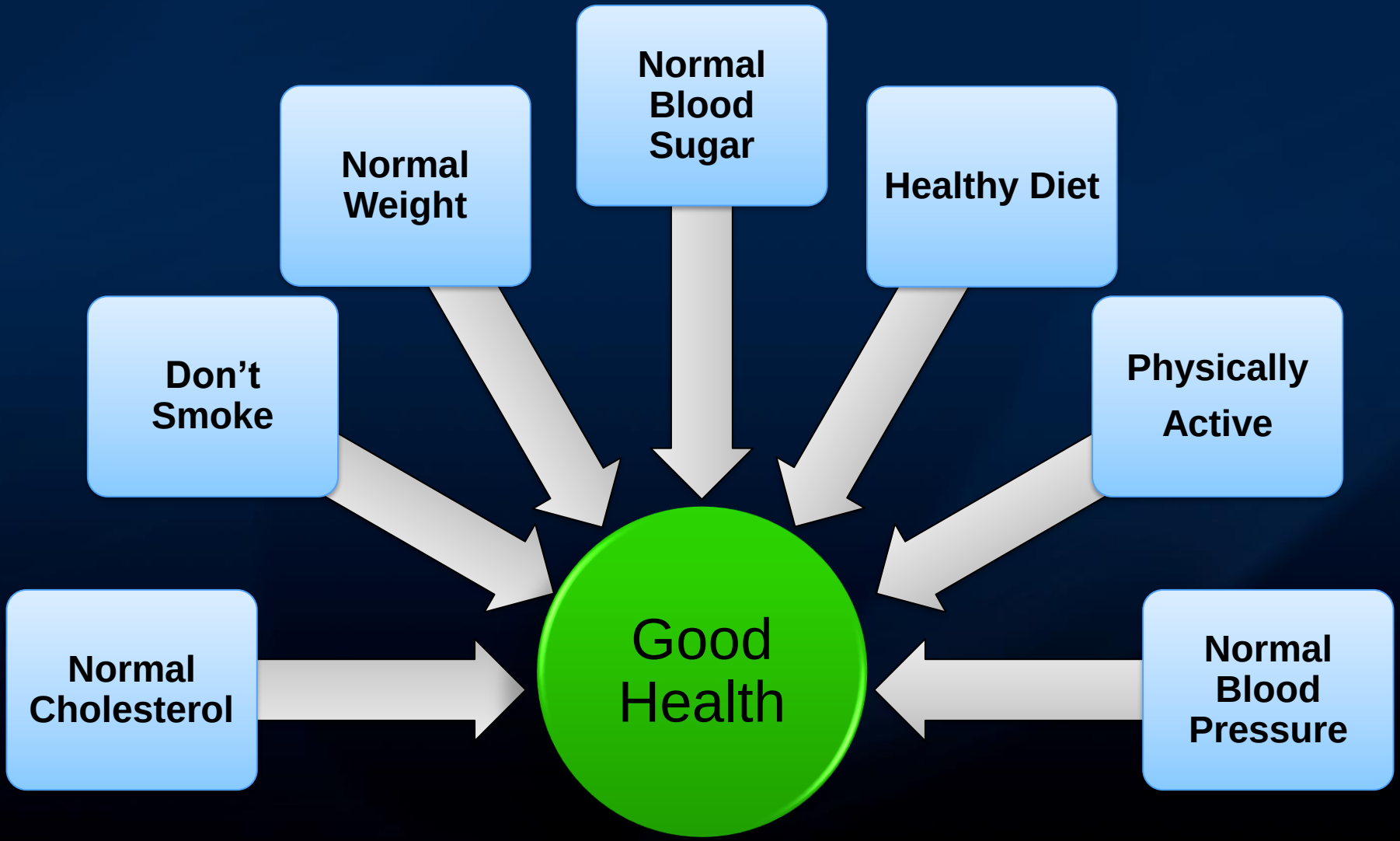
The 21st Century Epidemic



We Already Know What Works

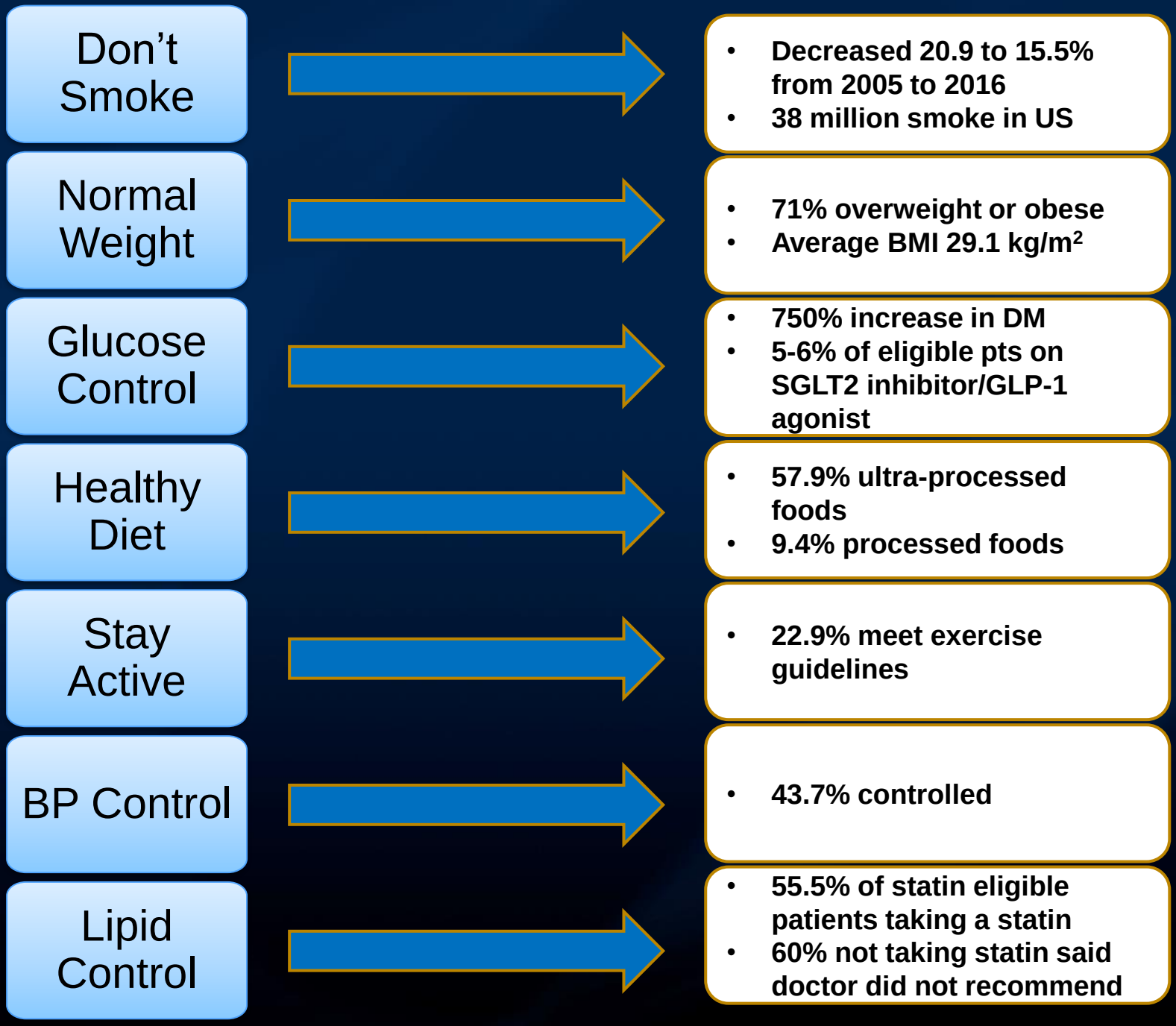
• Decrease MI	80%
• Decrease CKD	62%
• Decrease Stroke	50%
• Decrease Dementia	37%
• Decrease Cancer	33%

Life's Simple 7



We Already Know What Works

• Decrease CAD	80%
• Decrease CKD	62%
• Decrease Stroke	50%
• Decrease Dementia	37%
• Decrease Cancer	33%


Don't Smoke
Decreased 20.9 to 15.5% from 2005 to 2016
38 million smoke in US
Normal Weight
71% overweight or obese
Average BMI 29.1 kg/m2
Glucose Control
750% increase in DM
5-6% of eligible pts on SGLT2 inhibitor/GLP-1 agonist
Healthy Diet
57.9% ultra-processed foods
9.4% processed foods
Stay Active
22.9% meet exercise guidelines
BP Control
43.7% controlled
Lipid Control
55.5% of statin eligible patients taking a statin
60% not taking statin said doctor did not recommend

Total heart disease deaths on the rise

Majority of these deaths are preventable, study authors say

US Deaths From Cardiometabolic Disease on the Rise

Total U.S. deaths from heart disease, stroke, diabetes, and high blood pressure — collectively known as cardiometabolic disease — have been increasing since 2011, thanks in large part to surging obesity rates.

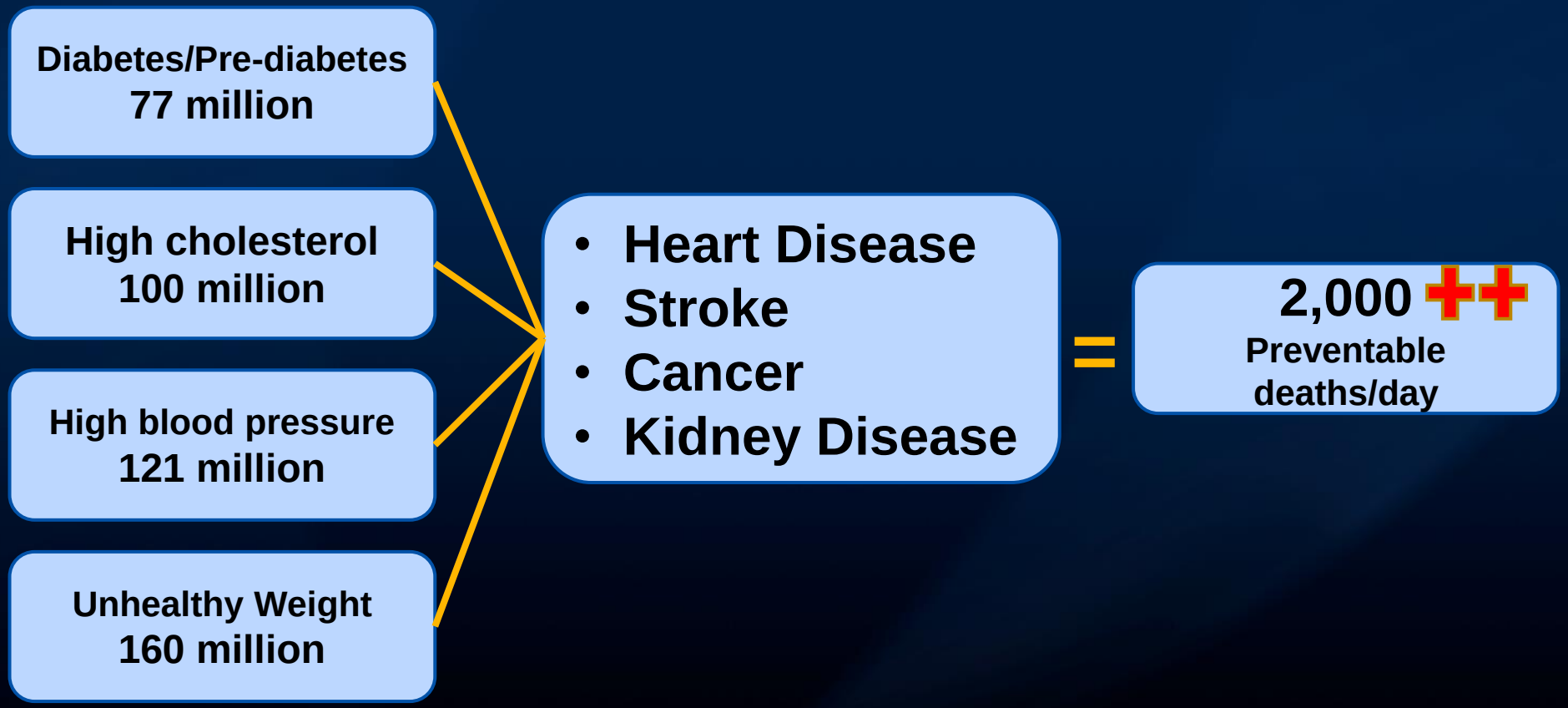
HEALTH

'Deaths of Despair': U.S. Life Expectancy Has Been Falling Since 2014, With Biggest Impacts in Rust Belt and Ohio Valley

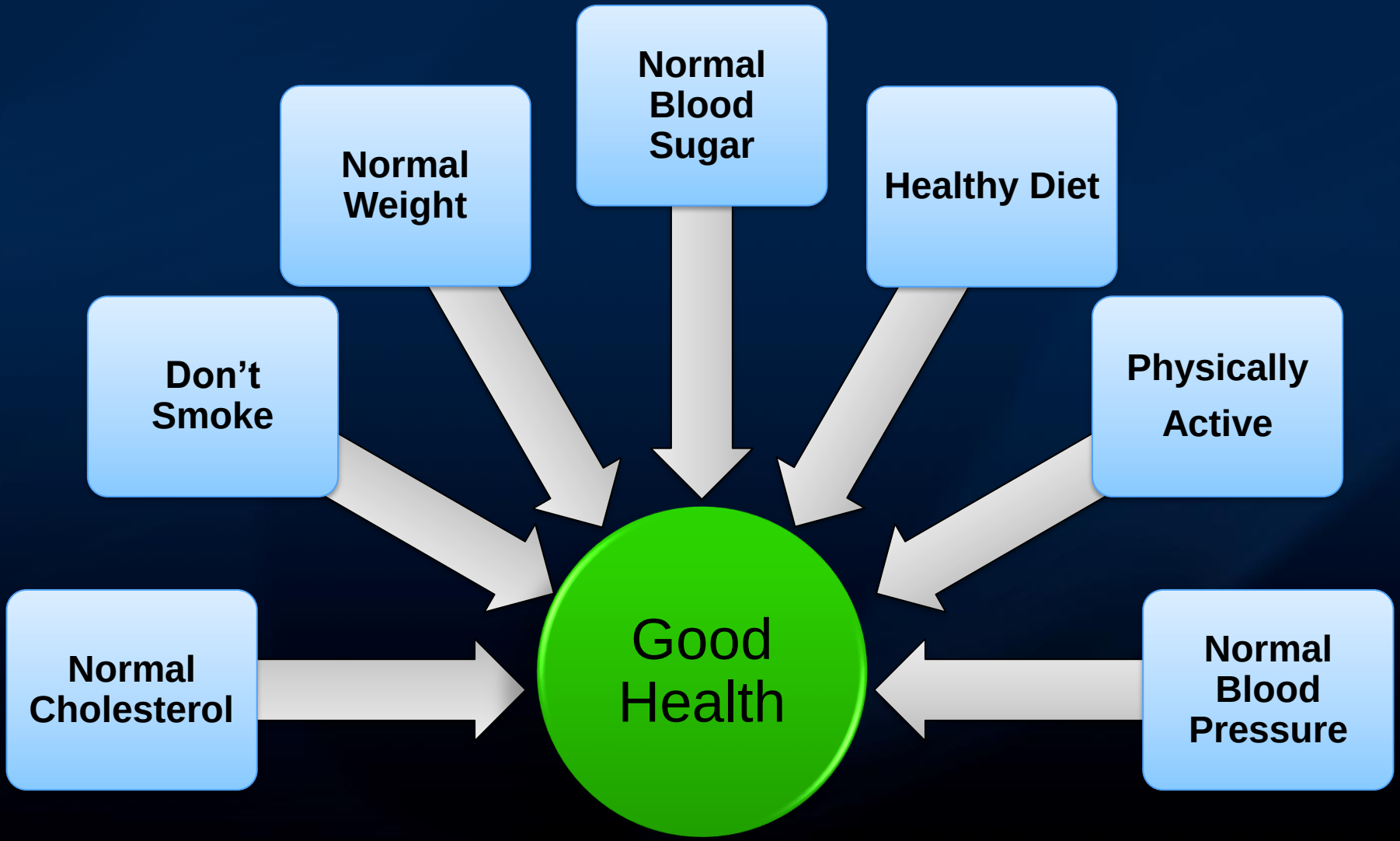
BY **KASHMIRA GANDER** ON 11/26/19 AT 11:26 AM EST

Cardiometabolic Disease

The 21st Century Epidemic



Life's Simple 7



Important Numbers

46% — American adults have hypertension

44% — With hypertension are controlled

1,100 — Deaths per day in the US

Call to Action to Control Hypertension

Surgeon General

Opinion

VIEWPOINT

A National Commitment to Improve the Care of Patients With Hypertension in the US

Jerome M. Adams, MD, MPH
Office of the Surgeon General, Department of Health and Human Services, Washington, DC.

Janet S. Wright, MD
Office of the Surgeon General, Department of Health and Human Services, Washington, DC.

Author Audio Interview

Video and Supplemental content

Hypertension is common, costly, and controllable. Almost 1 in 2 US adults has hypertension, and among those, the estimated rate of controlled blood pressure was only 43.7% in 2017-2018 a decline from 53.8% in 2013-2014.[1] Uncontrolled blood pressure can lead to largely preventable events such as myocardial infarction, stroke, and maternal mortality, as well as debilitating and expensive conditions such as kidney disease, heart failure, and cognitive decline. Hypertensive disorders of pregnancy, which have increased in the US, contribute to adverse maternal and child health outcomes and can increase a woman's lifetime risk of cardiovascular disease. Disparities in blood pressure control and, consequently, in these health outcomes, persist by race and ethnicity, age, and geography. Yet broad and equitable hypertension control is possible, and some health care practices and systems have achieved rates of 80% or higher across a wide spectrum of sites and populations served.[2-5]

These facts about hypertension—a highly prevalent, poorly managed, inequitably experienced, and highly controllable condition—are more than sufficient to merit the *Surgeon General's Call to Action to Control Hypertension*.[6] Some may question the release of this report now, when the challenge of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has become the most pressing health priority in the US. However, it is precisely the effects of the pandemic, and the painful lessons that are being learned, that add even greater urgency to improving hypertension control rates for all US adults.[7] The coronavirus disease 2019 (COVID-19) pandemic has revealed substantial differences in exposure to the SARS-CoV-2 virus and severe outcomes from COVID-19. The higher rates of infection, severe complications, and death among people of color, in particular, are at least in part due to social determinants of health: housing, transportation, education, access to health care, availability of healthy food options, a secure income, and freedom from structural and institutional barriers related to race and bias. These same determinants either support or impede success in controlling high blood pressure. When coupled with widespread implementation of best practices in clinical settings and empowering individuals to actively manage their blood pressure, acknowledging and addressing a community's social conditions may generate sustained improvements in control of both hypertension and COVID-19.

Now is the time to draw attention and drive action to proven strategies that improve blood pressure control. Controlling hypertension requires sustained and specific actions by individuals with and at risk for high blood pressure; health care and public health professionals and the systems in which they operate; and communities. Smart, substantive, and ongoing investments by each of these sectors are needed to achieve better health outcomes in the near term, but also a more resilient, equitable, and prosperous nation in the future.

The goals and strategies presented in the Surgeon General's *Call to Action* provide a national roadmap to drive change (eFigure in the Supplement). The 3 goals are: (1) make hypertension control a national priority; (2) ensure that communities support hypertension control; and (3) optimize patient care for hypertension control. The goals and strategies are grounded in the evidence, informed by experiences of high-performing systems and communities, and adaptable to match the resources available and the populations served.

> Implementing the goals and strategies in the *Call to Action*...could help patients, clinicians, and communities achieve the health, wealth, and equity benefits that national hypertension control can bring.

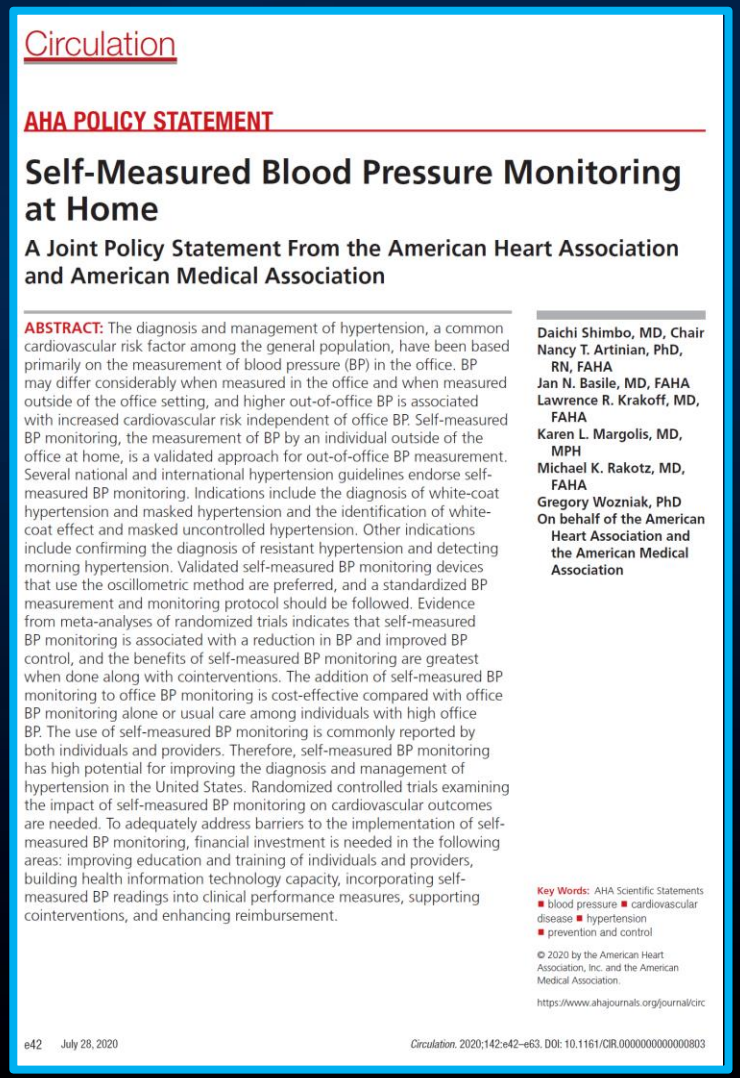
The first major goal is to declare hypertension control a national priority; this is justified by the costs in lives, health, and dollars lost to a largely controllable condition. Generating widespread awareness of the effect of uncontrolled blood pressure on health and the economy is the first step in galvanizing action by the diverse set of sectors outlined in the document. Among the partners essential to achieving a national aim of hypertension control are payers and employers, who, by prioritizing blood pressure control in value-based contracting and incentive programs, could enable practices to invest in the teams and processes proven to achieve high performance over time. Payers and employers also could help individuals manage their hypertension by eliminating cost-sharing for blood pressure monitors and medications. Setting blood pressure control as a population health priority also draws attention to the profound disparities associated with hypertension, with racial and ethnic minority groups experiencing higher rates of hypertension, lower levels of blood pressure control, and greater risk of direct or indirect (eg, COVID-19–related) complications. The reason to establish this goal is not merely to

Corresponding Author: Janet S. Wright, MD, Office of the Surgeon General, Department of Health and Human Services, 200 Independence Ave SW, Washington, DC 20201 (janet.wright@hhs.gov).

- **Highly prevalent**
- **Poorly managed**
- **Inequitably experienced**
- **Highly controllable**

JAMA. 2020;324(18):1825-1826.

Optimize Patient Care
Home Blood Pressure



Circulation

AHA POLICY STATEMENT

Self-Measured Blood Pressure Monitoring at Home

A Joint Policy Statement From the American Heart Association and American Medical Association

ABSTRACT: The diagnosis and management of hypertension, a common cardiovascular risk factor among the general population, have been based primarily on the measurement of blood pressure (BP) in the office. BP may differ considerably when measured in the office and when measured outside of the office setting, and higher out-of-office BP is associated with increased cardiovascular risk independent of office BP. Self-measured BP monitoring, the measurement of BP by an individual outside of the office at home, is a validated approach for out-of-office BP measurement. Several national and international hypertension guidelines endorse self-measured BP monitoring. Indications include the diagnosis of white-coat hypertension and masked hypertension and the identification of white-coat effect and masked uncontrolled hypertension. Other indications include confirming the diagnosis of resistant hypertension and detecting morning hypertension. Validated self-measured BP monitoring devices that use the oscillometric method are preferred, and a standardized BP measurement and monitoring protocol should be followed. Evidence from meta-analyses of randomized trials indicates that self-measured BP monitoring is associated with a reduction in BP and improved BP control, and the benefits of self-measured BP monitoring are greatest when done along with cointerventions. The addition of self-measured BP monitoring to office BP monitoring is cost-effective compared with office BP monitoring alone or usual care among individuals with high office BP. The use of self-measured BP monitoring is commonly reported by both individuals and providers. Therefore, self-measured BP monitoring has high potential for improving the diagnosis and management of hypertension in the United States. Randomized controlled trials examining the impact of self-measured BP monitoring on cardiovascular outcomes are needed. To adequately address barriers to the implementation of self-measured BP monitoring, financial investment is needed in the following areas: improving education and training of individuals and providers, building health information technology capacity, incorporating self-measured BP readings into clinical performance measures, supporting cointerventions, and enhancing reimbursement.

Daichi Shimbo, MD, Chair
Nancy T. Artinian, PhD, RN, FAHA
Jan N. Basile, MD, FAHA
Lawrence R. Krakoff, MD, FAHA
Karen L. Margolis, MD, MPH
Michael K. Rakotz, MD, FAHA
Gregory Wozniak, PhD
On behalf of the American Heart Association and the American Medical Association

Key Words: AHA Scientific Statements ■ blood pressure ■ cardiovascular disease ■ hypertension ■ prevention and control

https://www.ahajournals.org/journal/circ

Circulation. 2020;142:e42–e63. DOI: 10.1161/CIR.0000000000000803

What we can do

- Quit relying on office blood pressure
  - Associated with lower BP and improved control
  - Cost effective

Circulation. 2020;142:e42–e63.

Optimize Patient Care
Use the Right Meds

Opinion

VIEWPOINT

A National Commitment to Improve the Care of Patients With Hypertension in the US

Jerome M. Adams, MD, MPH
Office of the Surgeon General, Department of Health and Human Services, Washington, DC.

Janet S. Wright, MD
Office of the Surgeon General, Department of Health and Human Services, Washington, DC.

Author Audio Interview

Video and Supplemental content

Hypertension is common, costly, and controllable. Almost 1 in 2 US adults has hypertension, and among those, the estimated rate of controlled blood pressure was only 43.7% in 2017-2018 a decline from 53.8% in 2013-2014.[1] Uncontrolled blood pressure can lead to largely preventable events such as myocardial infarction, stroke, and maternal mortality, as well as debilitating and expensive conditions such as kidney disease, heart failure, and cognitive decline. Hypertensive disorders of pregnancy, which have increased in the US, contribute to adverse maternal and child health outcomes and can increase a woman's lifetime risk of cardiovascular disease. Disparities in blood pressure control and, consequently, in these health outcomes, persist by race and ethnicity, age, and geography. Yet broad and equitable hypertension control is possible, and some health care practices and systems have achieved rates of 80% or higher across a wide spectrum of sites and populations served.[2-5]

These facts about hypertension—a highly prevalent, poorly managed, inequitably experienced, and highly controllable condition—are more than sufficient to merit the *Surgeon General's Call to Action to Control Hypertension*.[6] Some may question the release of this report now, when the challenge of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has become the most pressing health priority in the US. However, it is precisely the effects of the pandemic, and the painful lessons that are being learned, that add even greater urgency to improving hypertension control rates for all US adults.[7] The coronavirus disease 2019 (COVID-19) pandemic has revealed substantial differences in exposure to the SARS-CoV-2 virus and severe outcomes from COVID-19. The higher rates of infection, severe complications, and death among people of color, in particular, are at least in part due to social determinants of health: housing, transportation, education, access to health care, availability of healthy food options, a secure income, and freedom from structural and institutional barriers related to race and bias. These same determinants either support or impede success in controlling high blood pressure.

> Implementing the goals and strategies in the *Call to Action*...could help patients, clinicians, and communities achieve the health, wealth, and equity benefits that national hypertension control can bring.

When coupled with widespread implementation of best practices in clinical settings and empowering individuals to actively manage their blood pressure, acknowledging and addressing a community's social conditions may generate sustained improvements in control of both hypertension and COVID-19.

Now is the time to draw attention and drive action to proven strategies that improve blood pressure control. Controlling hypertension requires sustained and specific actions by individuals with and at risk for high blood pressure; health care and public health professionals and the systems in which they operate; and communities. Smart, substantive, and ongoing investments by each of these sectors are needed to achieve better health outcomes in the near term, but also a more resilient, equitable, and prosperous nation in the future.

The goals and strategies presented in the Surgeon General's *Call to Action* provide a national roadmap to drive change (eFigure in the Supplement). The 3 goals are: (1) make hypertension control a national priority; (2) ensure that communities support hypertension control; and (3) optimize patient care for hypertension control. The goals and strategies are grounded in the evidence, informed by experiences of high-performing systems and communities, and adaptable to match the resources available and the populations served.

The first major goal is to declare hypertension control a national priority; this is justified by the costs in lives, health, and dollars lost to a largely controllable condition. Generating widespread awareness of the effect of uncontrolled blood pressure on health and the economy is the first step in galvanizing action by the diverse set of sectors outlined in the document. Among the partners essential to achieving a national aim of hypertension control are payers and employers, who, by prioritizing blood pressure control in value-based contracting and incentive programs, could enable practices to invest in the teams and processes proven to achieve high performance over time. Payers and employers also could help individuals manage their hypertension by eliminating cost-sharing for blood pressure monitors and medications. Setting blood pressure control as a population health priority also draws attention to the profound disparities associated with hypertension, with racial and ethnic minority groups experiencing higher rates of hypertension, lower levels of blood pressure control, and greater risk of direct or indirect (eg, COVID-19–related) complications. The reason to establish this goal is not merely to

Corresponding Author: Janet S. Wright, MD, Office of the Surgeon General, Department of Health and Human Services, 200 Independence Ave SW, Washington, DC 20201 (janet.wright@hhs.gov).

- **What we can do**
  - **Use first line generic meds**
    - **Chlorthalidone or indapamide > HCTZ**
    - **ACEi OR ARB**
    - **Amlodipine**
  - **Screen for sleep apnea**

JAMA. 2020;324(18):1825-1826.

•. 2020 Jul 7;173(1):10-20.
•. 2020 Jul 7;173(1):10-20.

Primary Aldosteronism
Underrecognized?

The Unrecognized Prevalence of Primary Aldosteronism:
A Cross-sectional Study

Jenifer M. Brown, MD, Mohammed Siddiqui, MD, David A. Calhoun, MD, Robert M. Carey, MD, Paul N. Hopkins, MD, MSPH, Gordon H. Williams, MD, Anand Vaidya, MD, MMSc
Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts (J.M.B., G.H.W., A.V.); University of Alabama at Birmingham, Birmingham, Alabama (M.S., D.A.C.); University of Virginia Health System, Charlottesville, Virginia (R.M.C.); and University of Utah School of Medicine, Salt Lake City, Utah (P.N.H.).

Abstract

Background: Primary aldosteronism is a nonsuppressible renin-independent aldosterone production that causes hypertension and cardiovascular disease.

Objective: To characterize the prevalence of nonsuppressible renin-independent aldosterone production, as well as biochemically overt primary aldosteronism, in relation to blood pressure.

Design: Cross-sectional study.

Setting: 4 U.S. academic medical centers.

Corresponding Author: Anand Vaidya, MD, MMSc, Center for Adrenal Disorders, Division of Endocrinology, Diabetes, and Hypertension, Brigham and Women's Hospital, Harvard Medical School, 221 Longwood Avenue, RFB, Boston, MA 02115; anandvaidya@bwh.harvard.edu.
Current Author Addresses: Dr. Brown: Division of Cardiovascular Medicine, Brigham and Women's Hospital, 75 Francis Street, Boston, MA 02115.
Dr. Siddiqui: University of Alabama at Birmingham, 933 19th Street South, Room 115 Community Health Service Building, Birmingham, AL 35209.
Dr. Calhoun: University of Alabama at Birmingham, Division of Cardiology, BMR2 430, Birmingham, AL 35294.
Dr. Carey: PO Box 801414, University of Virginia Health System, Charlottesville, VA 22908.
Dr. Hopkins: Division of Cardiovascular Genetics, University of Utah School of Medicine, 50 North Medical Drive, Room 1160, Salt Lake City, UT 84132.
Drs. Williams and Vaidya: Center for Adrenal Disorders, Division of Endocrinology, Diabetes, and Hypertension, Brigham and Women's Hospital, Harvard Medical School, 221 Longwood Avenue, RFB, Boston, MA 02115.
Author Contributions: Conception and design: J.M. Brown, D.A. Calhoun, R.M. Carey, G.H. Williams, A. Vaidya.
Analysis and interpretation of the data: J.M. Brown, M. Siddiqui, D.A. Calhoun, R.M. Carey, A. Vaidya.
Drafting of the article: J.M. Brown, M. Siddiqui, A. Vaidya. Critical revision of the article for important intellectual content: J.M. Brown, M. Siddiqui, R.M. Carey, P.N. Hopkins, G.H. Williams, A. Vaidya.
Final approval of the article: J.M. Brown, M. Siddiqui, D.A. Calhoun, R.M. Carey, P.N. Hopkins, G.H. Williams, A. Vaidya. Provision of study materials or patients: M. Siddiqui, R.M. Carey, G.H. Williams, A. Vaidya.
Statistical expertise: P.N. Hopkins, A. Vaidya.
Obtaining of funding: D.A. Calhoun, R.M. Carey, A. Vaidya. Administrative, technical, or logistic support: P.N. Hopkins, G.H. Williams, A. Vaidya.
Collection and assembly of data: J.M. Brown, M. Siddiqui, D.A. Calhoun, R.M. Carey, P.N. Hopkins, A. Vaidya.

Disclosures: Dr. Brown reports grants from the National Heart, Lung, and Blood Institute. Dr. Williams reports grants from the National Institutes of Health. Dr. Vaidya reports consultancy for Catalys Pacific, Corcept Therapeutics, HRA Pharma, Orphagen, and Selenity Therapeutics and grants from the National Institutes of Health and Ventus Charitable Foundation. Authors not named here have disclosed no conflicts of interest. Disclosures can also be viewed at www.acponline.org/authors/icmje/ConflictOfInterestForms.do?msNum=M20-0065.

Reproducible Research Statement: *Study protocol:* See the Supplement (available at Annals.org). *Statistical code:* Available from Dr. Vaidya (e-mail, anandvaidya@bwh.harvard.edu). *Data set:* Not available.

Current author addresses and author contributions are available at Annals.org.

What we can do

- **Consider primary aldosteronism?**
  - Up to 20% of resistant hypertensives (1 in 1000 are screened)
  - Strongly consider with hypokalemia, OSA or adrenal mass
  - Secret weapon in resistant HTN
    - Aldactone or eplerenone

Ann Intern Med 2020;173:10-20.

Hygia Chronotherapy

Nighttime meds

ESC European Society of Cardiology

European Heart Journal (2020) **41**, 4565–4576
doi:10.1093/eurheartj/ehz754

CLINICAL RESEARCH
Hypertension

Bedtime hypertension treatment improves cardiovascular risk reduction: the Hygia Chronotherapy Trial

Ramón C. Hermida[1]*, Juan J. Crespo[1,2], Manuel Domínguez-Sardina[2], Alfonso Otero[3], Ana Moyá[4], María T. Ríos[1,2], Elvira Sineiro[1,4], María C. Castiñeira[1,5], Pedro A. Callejas[1,2], Lorenzo Pousa[1,2], José L. Salgado[1,2], Carmen Durán[2], Juan J. Sánchez[1,6], José R. Fernández[1], Artemio Mojón[1], and Diana E. Ayala[1]; for the Hygia Project Investigators[†]

[1]Bioengineering & Chronobiology Laboratories, Atlantic Research Center for Information and Communication Technologies (AtlantTIC), University of Vigo, E.I. Telecomunicación, Campus Universitario, Vigo 36310, Spain; [2]Estructura de Xestión Integrada de Vigo, Servicio Galego de Saúde (SERGAS), Vigo 36214, Spain; [3]Servicio de Nefrología, Complejo Hospitalario Universitario, Estructura de Xestión Integrada de Ourense, Verín e O Barco de Valdeorras, Servicio Galego de Saúde (SERGAS), Ourense 32005, Spain; [4]Estructura de Xerencia Integrada Pontevedra e O Salnés, Servicio Galego de Saúde (SERGAS), Pontevedra 36156, Spain; [5]Estructura de Xestión Integrada de Lugo, Cervo e Monforte de Lemos, Servicio Galego de Saúde (SERGAS), Lugo 27002, Spain; and [6]Estructura de Xestión Integrada de Santiago de Compostela, Servicio Galego de Saúde (SERGAS), Santiago de Compostela 15701, Spain

Received 14 June 2019; revised 26 July 2019; editorial decision 1 October 2019; accepted 1 October 2019; online publish-ahead-of-print 22 October 2019

See page 4577 for the editorial comment on this article (doi: 10.1093/eurheartj/ehz836)

Aims The Hygia Chronotherapy Trial, conducted within the clinical primary care setting, was designed to test whether bedtime in comparison to usual upon awakening hypertension therapy exerts better cardiovascular disease (CVD) risk reduction.

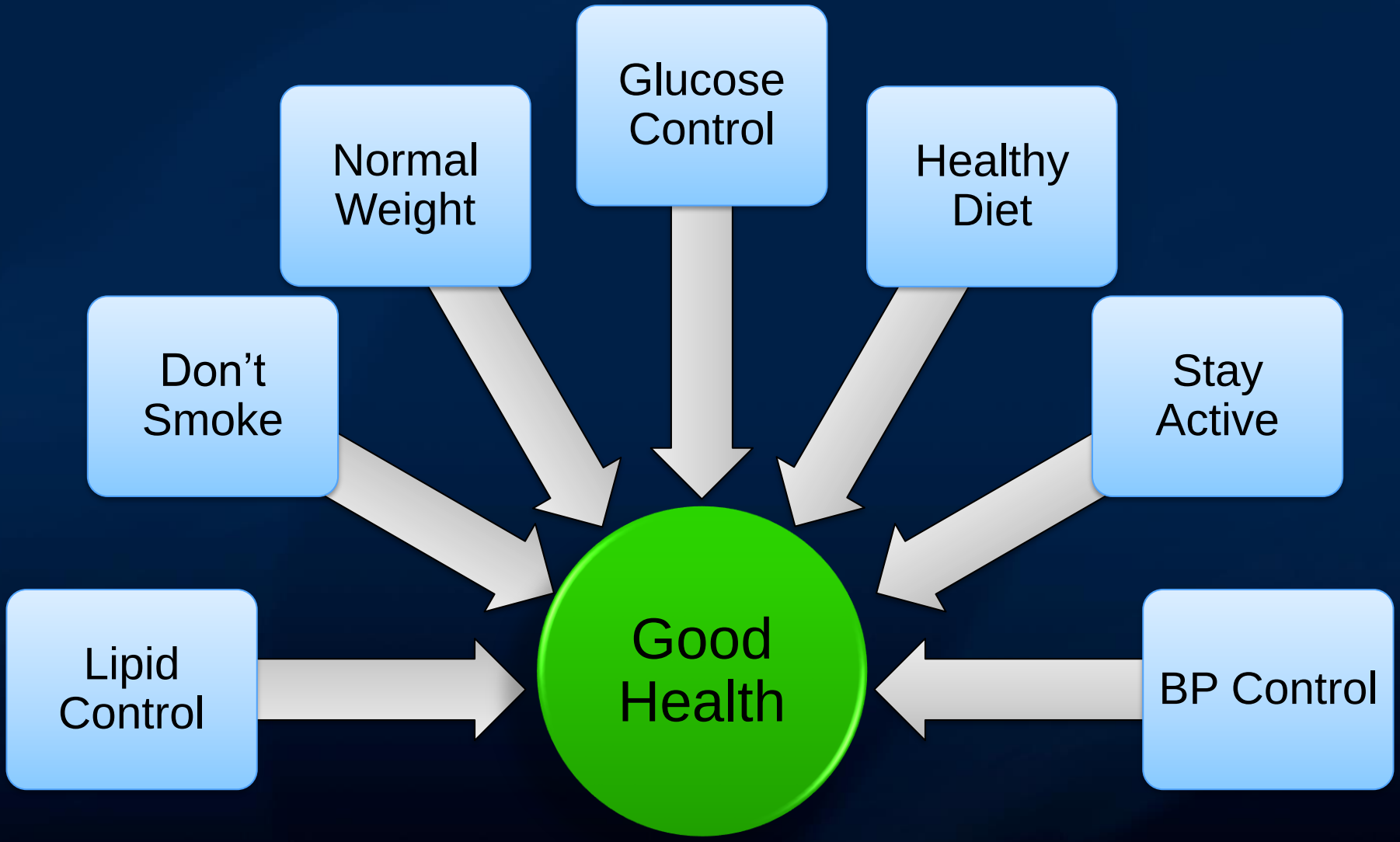
Methods and results In this multicentre, controlled, prospective endpoint trial, 19 084 hypertensive patients (10 614 men/8470 women, 60.5 ± 13.7 years of age) were assigned (1:1) to ingest the entire daily dose of ≥1 hypertension medications at bedtime ($n = 9552$) or all of them upon awakening ($n = 9532$). At inclusion and at every scheduled clinic visit (at least annually) throughout follow-up, ambulatory blood pressure (ABP) monitoring was performed for 48 h. During the 6.3-year median patient follow-up, 1752 participants experienced the primary CVD outcome (CVD death, myocardial infarction, coronary revascularization, heart failure, or stroke). Patients of the bedtime, compared with the upon-waking, treatment-time regimen showed significantly lower hazard ratio—adjusted for significant influential characteristics of age, sex, type 2 diabetes, chronic kidney disease, smoking, HDL cholesterol, asleep systolic blood pressure (BP) mean, sleep-time relative systolic BP decline, and previous CVD event—of the primary CVD outcome [0.55 (95% CI 0.50–0.61), $P < 0.001$] and each of its single components ($P < 0.001$ in all cases), i.e. CVD death [0.44 (0.34–0.56)], myocardial infarction [0.66 (0.52–0.84)], coronary revascularization [0.60 (0.47–0.75)], heart failure [0.58 (0.49–0.70)], and stroke [0.51 (0.41–0.63)].

Conclusion Routine ingestion by hypertensive patients of ≥1 prescribed BP-lowering medications at bedtime, as opposed to upon waking, results in improved ABP control (significantly enhanced decrease in asleep BP and increased sleep-time relative BP decline, i.e. BP dipping) and, most importantly, markedly diminished occurrence of major CVD events.

Trial registration ClinicalTrials.gov, number NCT00741585.

- **n = 19,084, randomized to taking meds at bedtime or on awakening**
- **6.3 year follow-up**
- **Improved BP control**
- **Lower combined primary endpoint, HR 0.55**
  - **CVD death 0.44**
  - **MI 0.66**
  - **CHF 0.58**
  - **Stroke 0.51**
- **Exceptions - diuretics or pts at risk of hypotension**

European Heart Journal 2020;21:3925-3932.


Glucose Control
Normal Weight
Healthy Diet
Don't Smoke
Stay Active
Lipid Control
Good Health
BP Control

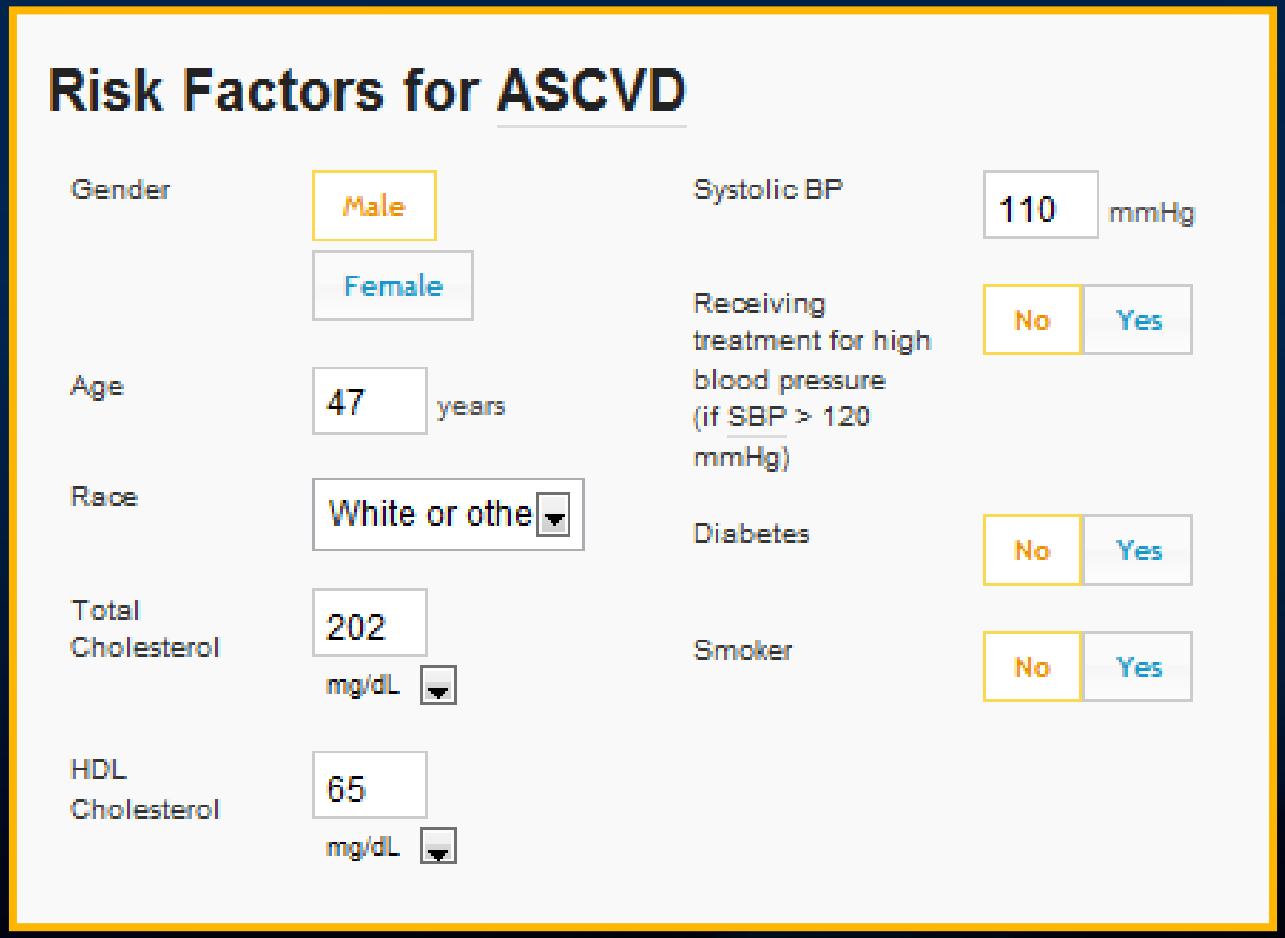
Statin Therapy

- **Age 40-75 “routinely” assess lipids and ASCVD risk**
- **Age 20-39 q 4-6 years**

Clinical CVD	Diabetes Age 40-75, LDL 70-189	No Diabetes Age 40-75, LDL 70-189	LDL ≥190
High intensity statin Add ezetimibe and/or PCSK9 if LDL > 70	Moderate Intensity Statin in all High Intensity Statin with Multiple Risk Factors	Calculate 10-year risk	High intensity statin

Risk < 5% Emphasize lifestyle	**Risk 5-7.5%** If risk enhancers, consider Moderate Intensity Statin	**Risk 7.5 - < 20%** Moderate Intensity Statin	**Risk ≥20%** High Intensity Statin

ASCVD Risk Score



So Many Risk Scores

That aren't used

- **Framingham**
  - **MI and CVD death**
- **Reynold's Risk Score**
  - **Includes CRP and Family history**
  - **MI, CVA, CVD death, revascularization**
- **ASCVD/pooled cohort equation**
  - **MI, CVD death, and CVA**
  - **Includes race**

57-year-old Physician

Concerned About Heart Risk

- **No history or symptoms of CVD**
- **Personal history of high cholesterol (greater than 300 mg/dL during residency)**
- **Family history of CVD**
  - **Father had MI at 76 years**

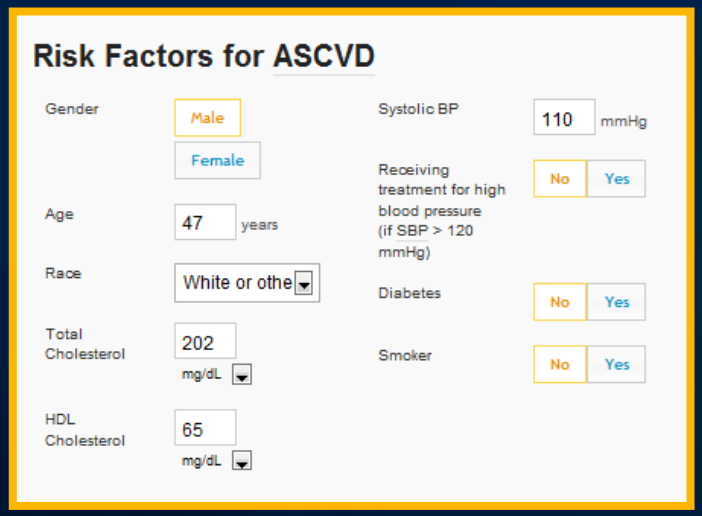
57-year-old Physician

Concerned About Heart Risk

- **Smoked 2 packs for 8 years, quit 30 years ago**
- **No diabetes**
- **BP 116/58 mm Hg**
- **BMI 27 Kg/m2**
- **Exercises regularly**
- **Primarily plant-based diet**

57-year-old Physician

- Outside stress test 2 years ago negative by report
- Currently
  - T Chol 224 mg/dl
  - TG 47 mg/dl
  - HDL 67 mg/dl
  - LDL 128 mg/dl
  - Glucose 106 mg/dl



ASCVD Risk Score

5%

What Would You Do For This Patient?

A) Reassure and congratulate him

B) Start a statin

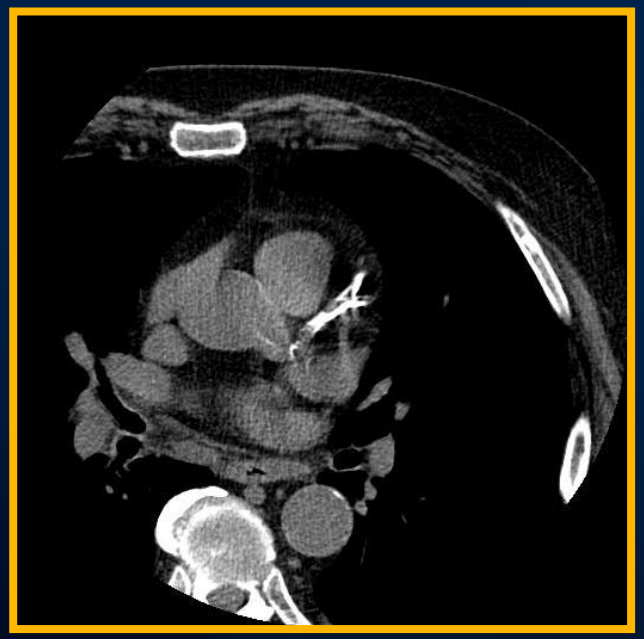
C) Carotid ultrasound

D) Stress test

E) Coronary artery calcium score

CT Coronary Calcium Score

- **Total calcium score**
  4,444 AU
- **99th percentile compared to gender and age matched controls**

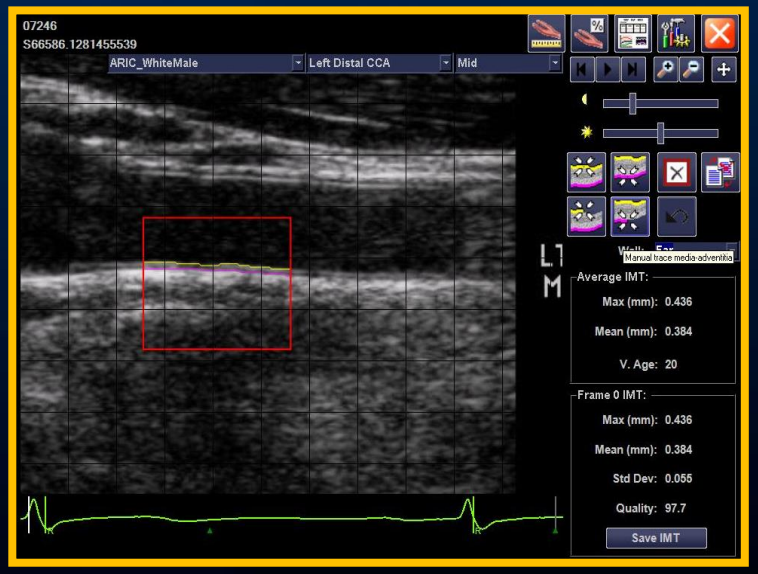


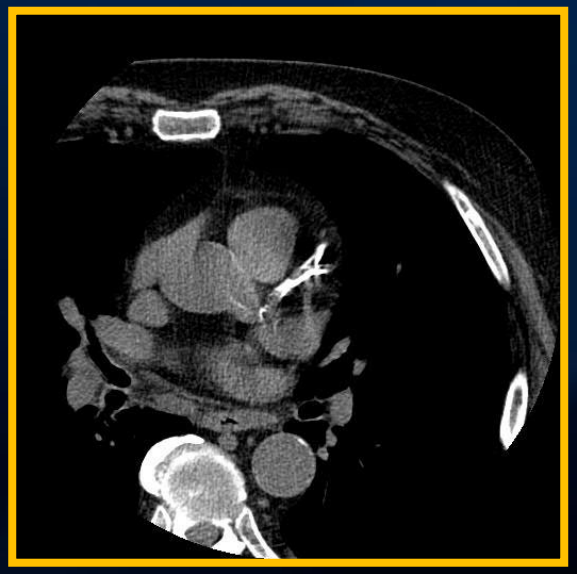
What Would You Do For This Patient?

A) Reassure and congratulate him

B) Start a statin

When do we need additional information to assess CV risk?





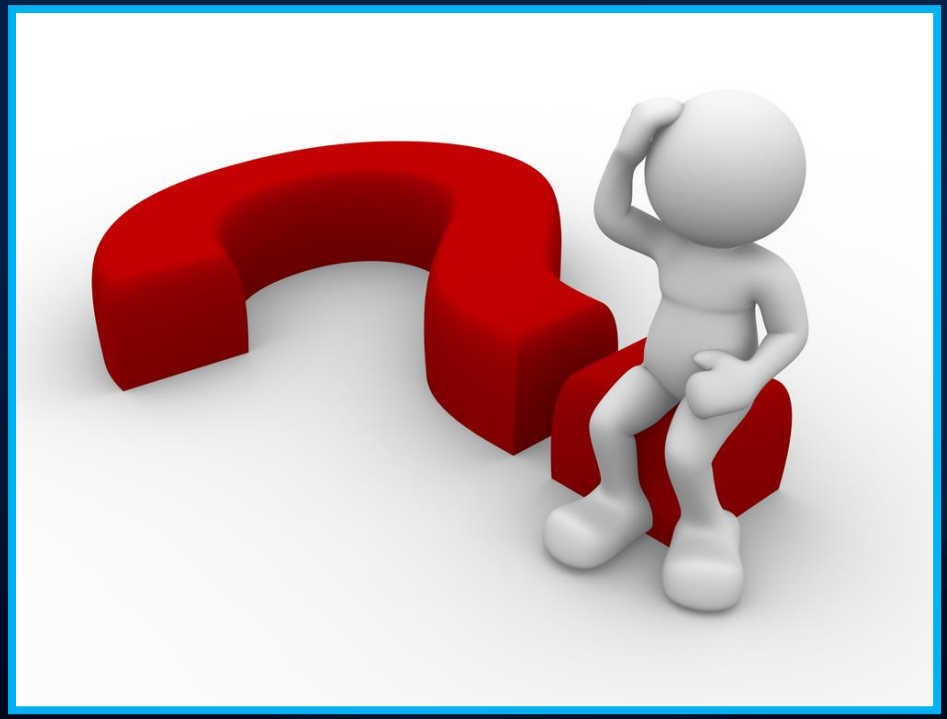
When it changes management


STATIN
Tablets
28
Tablets
STATIN 40 mg
28
Tablets

When to do Additional Testing?

Statin or No Statin?

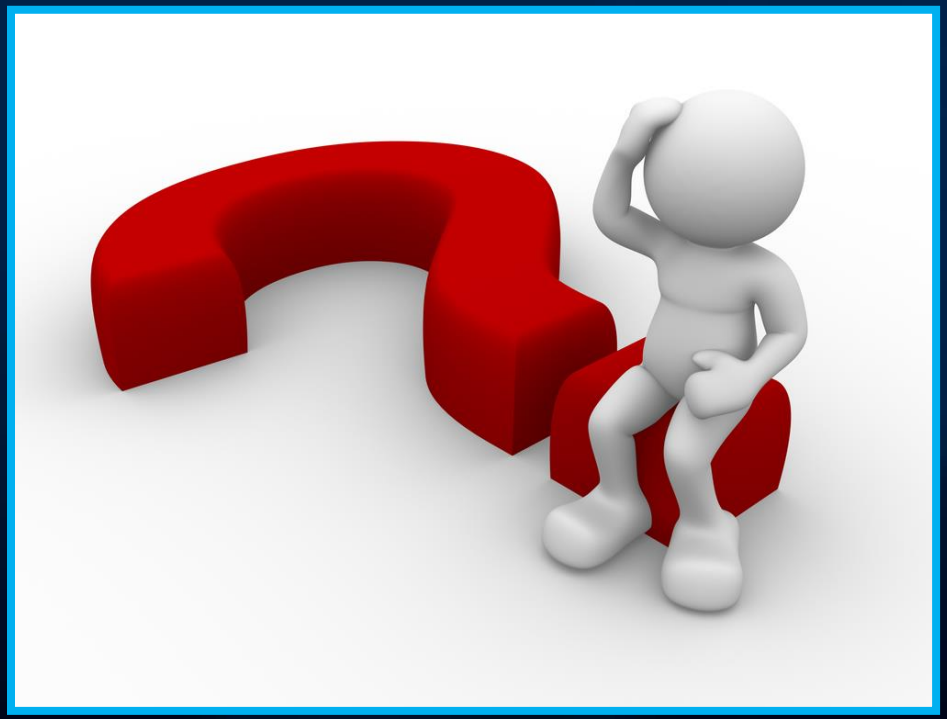
- **Family history of CVD**
- **Striking risk factor in a young person**
- **“Grey zone” ASCVD risk score (5-7.5%)**



When NOT to do Additional Testing?

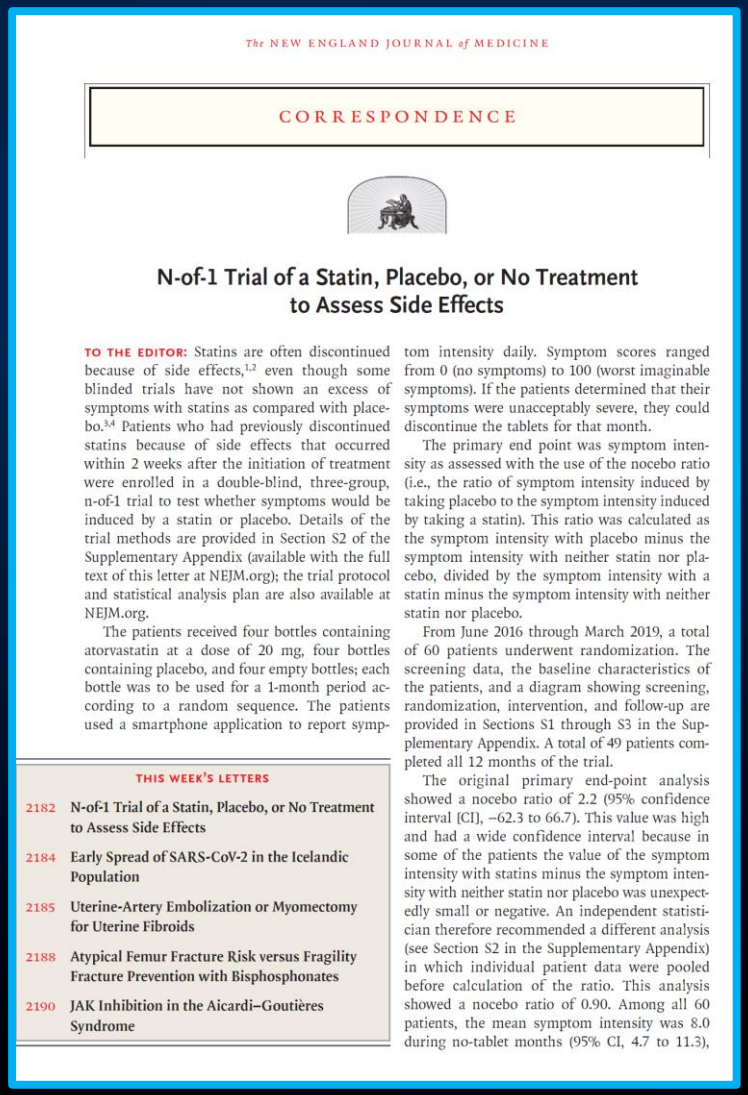
CV Risk Stratification

- **Established CVD**
- **Already on a statin**
- **Patient and provider agree**
- **To assess effectiveness of treatment**



Statins

Are Side Effects Real?



The NEW ENGLAND JOURNAL of MEDICINE

CORRESPONDENCE

N-of-1 Trial of a Statin, Placebo, or No Treatment to Assess Side Effects

TO THE EDITOR: Statins are often discontinued because of side effects,[1,2] even though some blinded trials have not shown an excess of symptoms with statins as compared with placebo.[3,4] Patients who had previously discontinued statins because of side effects that occurred within 2 weeks after the initiation of treatment were enrolled in a double-blind, three-group, n-of-1 trial to test whether symptoms would be induced by a statin or placebo. Details of the trial methods are provided in Section S2 of the Supplementary Appendix (available with the full text of this letter at NEJM.org); the trial protocol and statistical analysis plan are also available at NEJM.org.

The patients received four bottles containing atorvastatin at a dose of 20 mg, four bottles containing placebo, and four empty bottles; each bottle was to be used for a 1-month period according to a random sequence. The patients used a smartphone application to report symptom intensity daily. Symptom scores ranged from 0 (no symptoms) to 100 (worst imaginable symptoms). If the patients determined that their symptoms were unacceptably severe, they could discontinue the tablets for that month.

The primary end point was symptom intensity as assessed with the use of the nocebo ratio (i.e., the ratio of symptom intensity induced by taking placebo to the symptom intensity induced by taking a statin). This ratio was calculated as the symptom intensity with placebo minus the symptom intensity with neither statin nor placebo, divided by the symptom intensity with a statin minus the symptom intensity with neither statin nor placebo.

From June 2016 through March 2019, a total of 60 patients underwent randomization. The screening data, the baseline characteristics of the patients, and a diagram showing screening, randomization, intervention, and follow-up are provided in Sections S1 through S3 in the Supplementary Appendix. A total of 49 patients completed all 12 months of the trial.

The original primary end-point analysis showed a nocebo ratio of 2.2 (95% confidence interval [CI], −62.3 to 66.7). This value was high and had a wide confidence interval because in some of the patients the value of the symptom intensity with statins minus the symptom intensity with neither statin nor placebo was unexpectedly small or negative. An independent statistician therefore recommended a different analysis (see Section S2 in the Supplementary Appendix) in which individual patient data were pooled before calculation of the ratio. This analysis showed a nocebo ratio of 0.90. Among all 60 patients, the mean symptom intensity was 8.0 during no-tablet months (95% CI, 4.7 to 11.3),

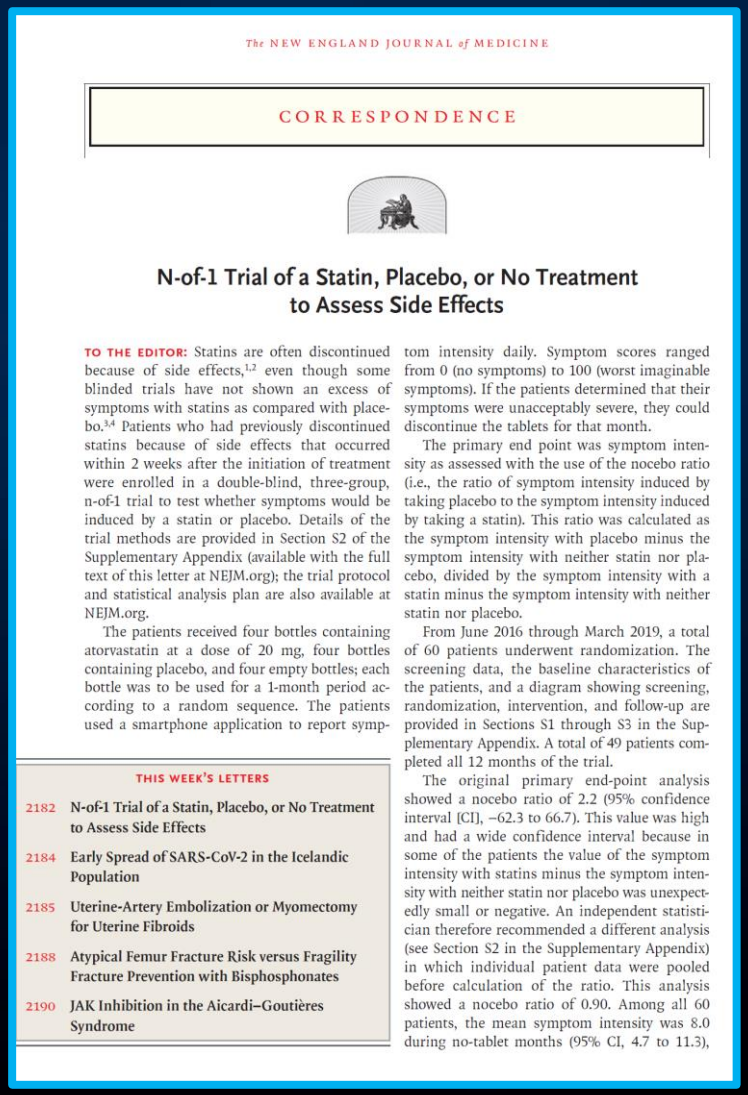
THIS WEEK'S LETTERS

- N-of-1 study design, 60 subjects with statin intolerance
- 4 bottles of atorva 20 mg, placebo, or empty
- Alternate bottles for a month over 1 year
- Symptoms tracked with smart phone app

N Engl J Med 2020;383:epub, Nov 15.

Statins

Are Side Effects Real?



The NEW ENGLAND JOURNAL of MEDICINE

CORRESPONDENCE

N-of-1 Trial of a Statin, Placebo, or No Treatment to Assess Side Effects

TO THE EDITOR: Statins are often discontinued because of side effects,[1,2] even though some blinded trials have not shown an excess of symptoms with statins as compared with placebo.[3,4] Patients who had previously discontinued statins because of side effects that occurred within 2 weeks after the initiation of treatment were enrolled in a double-blind, three-group, n-of-1 trial to test whether symptoms would be induced by a statin or placebo. Details of the trial methods are provided in Section S2 of the Supplementary Appendix (available with the full text of this letter at NEJM.org); the trial protocol and statistical analysis plan are also available at NEJM.org.

The patients received four bottles containing atorvastatin at a dose of 20 mg, four bottles containing placebo, and four empty bottles; each bottle was to be used for a 1-month period according to a random sequence. The patients used a smartphone application to report symptom intensity daily. Symptom scores ranged from 0 (no symptoms) to 100 (worst imaginable symptoms). If the patients determined that their symptoms were unacceptably severe, they could discontinue the tablets for that month.

The primary end point was symptom intensity as assessed with the use of the nocebo ratio (i.e., the ratio of symptom intensity induced by taking placebo to the symptom intensity induced by taking a statin). This ratio was calculated as the symptom intensity with placebo minus the symptom intensity with neither statin nor placebo, divided by the symptom intensity with a statin minus the symptom intensity with neither statin nor placebo.

From June 2016 through March 2019, a total of 60 patients underwent randomization. The screening data, the baseline characteristics of the patients, and a diagram showing screening, randomization, intervention, and follow-up are provided in Sections S1 through S3 in the Supplementary Appendix. A total of 49 patients completed all 12 months of the trial.

The original primary end-point analysis showed a nocebo ratio of 2.2 (95% confidence interval [CI], −62.3 to 66.7). This value was high and had a wide confidence interval because in some of the patients the value of the symptom intensity with statins minus the symptom intensity with neither statin nor placebo was unexpectedly small or negative. An independent statistician therefore recommended a different analysis (see Section S2 in the Supplementary Appendix) in which individual patient data were pooled before calculation of the ratio. This analysis showed a nocebo ratio of 0.90. Among all 60 patients, the mean symptom intensity was 8.0 during no-tablet months (95% CI, 4.7 to 11.3),

THIS WEEK'S LETTERS

- 90% of symptoms while taking atorva also reported in placebo
- No pill bottles ~ half the symptoms
- All subjects shown results at end of trial – 50% restarted statins

N Engl J Med 2020;383:epub, Nov 15.

Statins in Elderly

Meta-analysis


THE LANCET

ARTICLES | VOLUME 396, ISSUE 10263, P1637-1643, NOVEMBER 21, 2020

Efficacy and safety of lowering LDL cholesterol in older patients: a systematic review and meta-analysis of randomised controlled trials

Barış Gencer, MD • Nicholas A Marston, MD • KyungAh Im, PhD • Prof Christopher P Cannon, MD • Prof Peter Sever, FRCP • Prof Anthony Keech, MD • et al. Show all authors

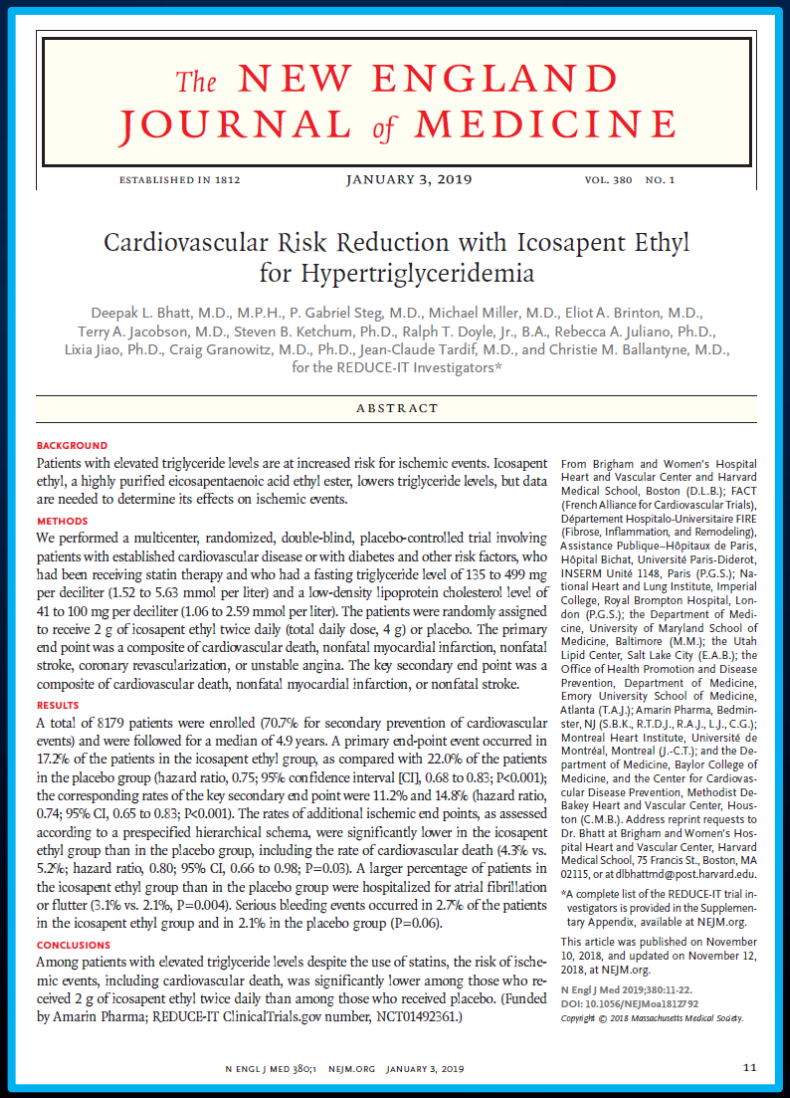
Published: November 10, 2020 • DOI: https://doi.org/10.1016/S0140-6736(20)32332-1 • Check for updates

- n= 244,090, 21,492 over 75 years of age
- **HR 0.74 MACE** in those > 75 years for every 1 mmol/L decrease in LDL
  - **HR 0.85** for death
  - **HR 0.80** for MI
  - **HR 0.73** for stroke
  - **HR 0.80** for revasc

Lancet 2020;396:1637-43.

REDUCE-IT

Icosapent ethyl (Vascepa®)



The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 JANUARY 3, 2019 VOL. 380 NO. 1

Cardiovascular Risk Reduction with Icosapent Ethyl for Hypertriglyceridemia

Deepak L. Bhatt, M.D., M.P.H., P. Gabriel Steg, M.D., Michael Miller, M.D., Eliot A. Brinton, M.D., Terry A. Jacobson, M.D., Steven B. Ketchum, Ph.D., Ralph T. Doyle, Jr., B.A., Rebecca A. Juliano, Ph.D., Lixia Jiao, Ph.D., Craig Granowitz, M.D., Ph.D., Jean-Claude Tardif, M.D., and Christie M. Ballantyne, M.D., for the REDUCE-IT Investigators*

ABSTRACT

BACKGROUND

Patients with elevated triglyceride levels are at increased risk for ischemic events. Icosapent ethyl, a highly purified eicosapentaenoic acid ethyl ester, lowers triglyceride levels, but data are needed to determine its effects on ischemic events.

METHODS

We performed a multicenter, randomized, double-blind, placebo-controlled trial involving patients with established cardiovascular disease or with diabetes and other risk factors, who had been receiving statin therapy and who had a fasting triglyceride level of 135 to 499 mg per deciliter (1.52 to 5.63 mmol per liter) and a low-density lipoprotein cholesterol level of 41 to 100 mg per deciliter (1.06 to 2.59 mmol per liter). The patients were randomly assigned to receive 2 g of icosapent ethyl twice daily (total daily dose, 4 g) or placebo. The primary end point was a composite of cardiovascular death, nonfatal myocardial infarction, nonfatal stroke, coronary revascularization, or unstable angina. The key secondary end point was a composite of cardiovascular death, nonfatal myocardial infarction, or nonfatal stroke.

RESULTS

A total of 8179 patients were enrolled (70.7% for secondary prevention of cardiovascular events) and were followed for a median of 4.9 years. A primary end-point event occurred in 17.2% of the patients in the icosapent ethyl group, as compared with 22.0% of the patients in the placebo group (hazard ratio, 0.75; 95% confidence interval [CI], 0.68 to 0.83; P<0.001); the corresponding rates of the key secondary end point were 11.2% and 14.8% (hazard ratio, 0.74; 95% CI, 0.65 to 0.83; P<0.001). The rates of additional ischemic end points, as assessed according to a prespecified hierarchical schema, were significantly lower in the icosapent ethyl group than in the placebo group, including the rate of cardiovascular death (4.3% vs. 5.2%; hazard ratio, 0.80; 95% CI, 0.66 to 0.98; P=0.03). A larger percentage of patients in the icosapent ethyl group than in the placebo group were hospitalized for atrial fibrillation or flutter (3.1% vs. 2.1%, P=0.004). Serious bleeding events occurred in 2.7% of the patients in the icosapent ethyl group and in 2.1% in the placebo group (P=0.06).

CONCLUSIONS

Among patients with elevated triglyceride levels despite the use of statins, the risk of ischemic events, including cardiovascular death, was significantly lower among those who received 2 g of icosapent ethyl twice daily than among those who received placebo. (Funded by Amarin Pharma; REDUCE-IT ClinicalTrials.gov number, NCT01492361.)

From Brigham and Women's Hospital Heart and Vascular Center and Harvard Medical School, Boston (D.L.B.); FACT (French Alliance for Cardiovascular Trials), Département Hospitalo-Universitaire FIRE (Fibrose, Inflammation, and Remodeling), Assistance Publique–Hôpitaux de Paris, Hôpital Bichat, Université Paris-Diderot, INSERM Unité 1148, Paris (P.G.S.); National Heart and Lung Institute, Imperial College, Royal Brompton Hospital, London (P.G.S.); the Department of Medicine, University of Maryland School of Medicine, Baltimore (M.M.); the Utah Lipid Center, Salt Lake City (E.A.B.); the Office of Health Promotion and Disease Prevention, Department of Medicine, Emory University School of Medicine, Atlanta (T.A.J.); Amarin Pharma, Bedminster, NJ (S.B.K., R.T.D.J., R.A.J., L.J., C.G.); Montreal Heart Institute, Université de Montréal, Montreal (J.-C.T.); and the Department of Medicine, Baylor College of Medicine, and the Center for Cardiovascular Disease Prevention, Methodist DeBakey Heart and Vascular Center, Houston (C.M.B.). Address reprint requests to Dr. Bhatt at Brigham and Women's Hospital Heart and Vascular Center, Harvard Medical School, 75 Francis St., Boston, MA 02115, or at dlbhattmd@post.harvard.edu.

*A complete list of the REDUCE-IT trial investigators is provided in the Supplementary Appendix, available at NEJM.org.

This article was published on November 10, 2018, and updated on November 12, 2018, at NEJM.org.

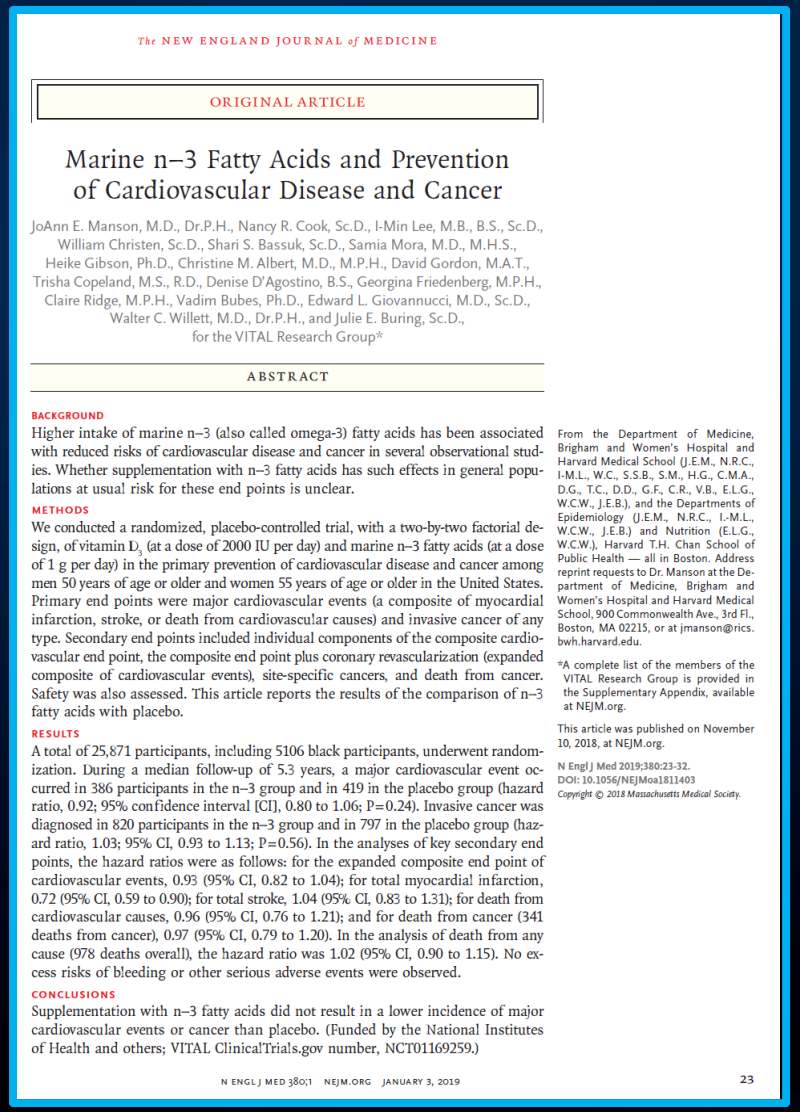
N Engl J Med 2019;380:11-22.
DOI: 10.1056/NEJMoa1812792

- **n = 8179 with CVD or DM and other risk factors on a statin**
- **TG 135-499 mg/dL, LDL 40-100 mg/dL on a statin**
- **CV death reduced 20%**
- **Primary endpoint reduced 25%**

N Engl J Med 2019; 380:11-22

VITAL

Vitamin D and Fish Oil



The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Marine n–3 Fatty Acids and Prevention of Cardiovascular Disease and Cancer

JoAnn E. Manson, M.D., Dr.P.H., Nancy R. Cook, Sc.D., I-Min Lee, M.B., B.S., Sc.D., William Christen, Sc.D., Shari S. Bassuk, Sc.D., Samia Mora, M.D., M.H.S., Heike Gibson, Ph.D., Christine M. Albert, M.D., M.P.H., David Gordon, M.A.T., Trisha Copeland, M.S., R.D., Denise D'Agostino, B.S., Georgina Friedenberg, M.P.H., Claire Ridge, M.P.H., Vadim Bubes, Ph.D., Edward L. Giovannucci, M.D., Sc.D., Walter C. Willett, M.D., Dr.P.H., and Julie E. Buring, Sc.D., for the VITAL Research Group*

ABSTRACT

BACKGROUND

Higher intake of marine n–3 (also called omega-3) fatty acids has been associated with reduced risks of cardiovascular disease and cancer in several observational studies. Whether supplementation with n–3 fatty acids has such effects in general populations at usual risk for these end points is unclear.

METHODS

We conducted a randomized, placebo-controlled trial, with a two-by-two factorial design, of vitamin D_3 (at a dose of 2000 IU per day) and marine n–3 fatty acids (at a dose of 1 g per day) in the primary prevention of cardiovascular disease and cancer among men 50 years of age or older and women 55 years of age or older in the United States. Primary end points were major cardiovascular events (a composite of myocardial infarction, stroke, or death from cardiovascular causes) and invasive cancer of any type. Secondary end points included individual components of the composite cardiovascular end point, the composite end point plus coronary revascularization (expanded composite of cardiovascular events), site-specific cancers, and death from cancer. Safety was also assessed. This article reports the results of the comparison of n–3 fatty acids with placebo.

RESULTS

A total of 25,871 participants, including 5106 black participants, underwent randomization. During a median follow-up of 5.3 years, a major cardiovascular event occurred in 386 participants in the n–3 group and in 419 in the placebo group (hazard ratio, 0.92; 95% confidence interval [CI], 0.80 to 1.06; P=0.24). Invasive cancer was diagnosed in 820 participants in the n–3 group and in 797 in the placebo group (hazard ratio, 1.03; 95% CI, 0.93 to 1.13; P=0.56). In the analyses of key secondary end points, the hazard ratios were as follows: for the expanded composite end point of cardiovascular events, 0.93 (95% CI, 0.82 to 1.04); for total myocardial infarction, 0.72 (95% CI, 0.59 to 0.90); for total stroke, 1.04 (95% CI, 0.83 to 1.31); for death from cardiovascular causes, 0.96 (95% CI, 0.76 to 1.21); and for death from cancer (341 deaths from cancer), 0.97 (95% CI, 0.79 to 1.20). In the analysis of death from any cause (978 deaths overall), the hazard ratio was 1.02 (95% CI, 0.90 to 1.15). No excess risks of bleeding or other serious adverse events were observed.

CONCLUSIONS

Supplementation with n–3 fatty acids did not result in a lower incidence of major cardiovascular events or cancer than placebo. (Funded by the National Institutes of Health and others; VITAL ClinicalTrials.gov number, NCT01169259.)

From the Department of Medicine, Brigham and Women's Hospital and Harvard Medical School (J.E.M., N.R.C., I-M.L., W.C., S.S.B., S.M., H.G., C.M.A., D.G., T.C., D.D., G.F., C.R., V.B., E.L.G., W.C.W., J.E.B.), and the Departments of Epidemiology (J.E.M., N.R.C., I.-M.L., W.C.W., J.E.B.) and Nutrition (E.L.G., W.C.W.), Harvard T.H. Chan School of Public Health — all in Boston. Address reprint requests to Dr. Manson at the Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, 900 Commonwealth Ave., 3rd Fl., Boston, MA 02215, or at jmanson@rics.bwh.harvard.edu.

*A complete list of the members of the VITAL Research Group is provided in the Supplementary Appendix, available at NEJM.org.

This article was published on November 10, 2018, at NEJM.org.

N Engl J Med 2019;380:23-32.
DOI: 10.1056/NEJMoa1811403

- **n = 25,871, primary prevention of CVD and cancer in men >50 and women > 55**
- **Randomized to 2,000 IU Vit D and 1 gm fish oil, FU 5.3 years**
- **NO difference in outcomes**

N Engl J Med 2019; 380:23-32
N Engl J Med 2019; 380:33-44

REDUCE-IT

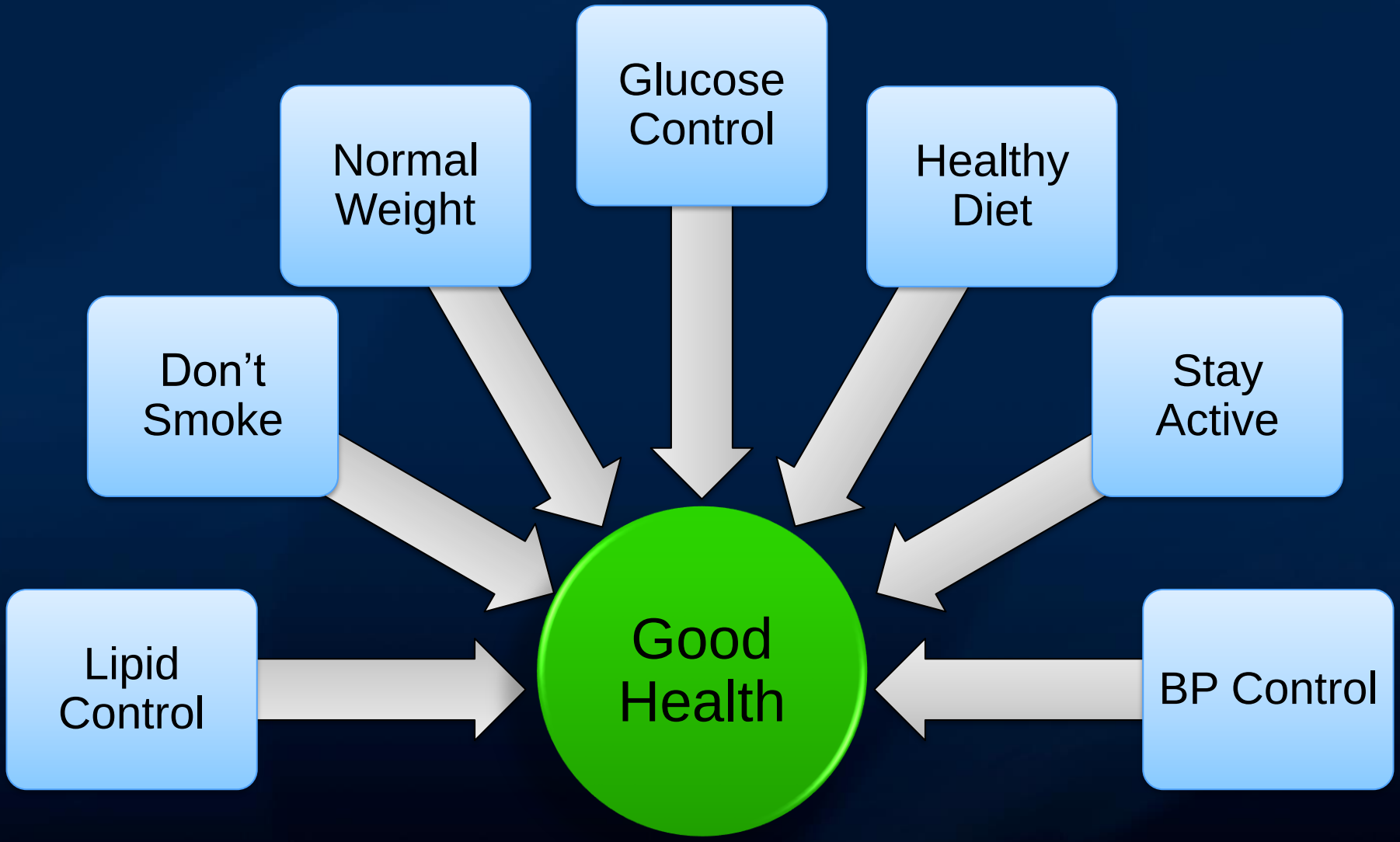
Other Things of Interest

- **Amazing outcome! (Too amazing?)**
- **Outlier result or is it the pure EPA that is important?**
- **Questionable effect of mineral oil placebo**
- **ADA, ALA and ESC have all given strong recommendation to add icosapent ethyl to statin therapy in high-risk pts with TG > 135 mg/dL**

Omega 3 FA

What do we recommend now?

- **As best as I can tell:**
  - **Fish oil (EPA/DHA) at any dose does not change CVD or cancer outcomes and MAY increase Afib and bleeding**
  - **If TG are high, fish oil is reasonable, icosapent ethyl preferred if insurance will cover**


Glucose Control
Normal Weight
Healthy Diet
Don't Smoke
Stay Active
Lipid Control
Good Health
BP Control

SGLT2 Inhibitors

Benefits

- **Death rate** **-30%**
- **CHF** **-25-35%**
- **MI** **-10-15%**
- **ESRD** **-40-50%**
- **Albuminuria** **-25-35%**
- **Weight loss** **-2 Kg**
- **BP** **-4/2 mmHg**
- **HgbA1c** **-0.7-1.0%**

SGLT2 Inhibitors

Adverse effects

- **Ketoacidosis**

  0.3% to 0.6%

- **Diarrhea**

  6% to 8.5%

- **Genital mycotic infection**

  0.9% to 2.4%


life
SMOKING
KILLS

According to a recent Nationwide survey:

MORE DOCTORS SMOKE CAMELS THAN ANY OTHER CIGARETTE

Most Smokers Want to Quit

But it's not easy

76% of smokers want to quit

59% have tried to quit in the last year

6% were successful

Early and Late Benefits

Lots of reasons to quit

- **1 month - lung function improves**
- **1 year – heart attack risk cut in half**
- **10 years – risk of heart disease is same as never smoking**
- **20 years – risk of lung disease, cancer, heart disease same as never smoking**

How much does pharmocotherapy and counseling increase smoking cessation rates?

A) 2 times

B) 5 times

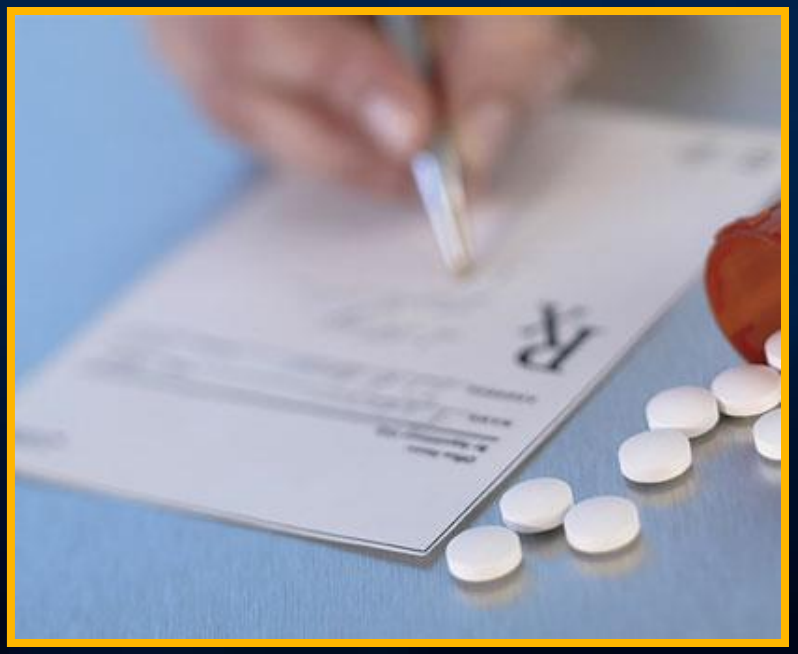
C) 10 times

D) It doesn’t

Counseling AND medications are more effective than either alone.

1-800-QUIT-NOW

It's free. It's personalized.
It's up to you.



For stable CAD, revascularization with PCI has been shown to:

A) Improve mortality

B) Decrease myocardial infarction

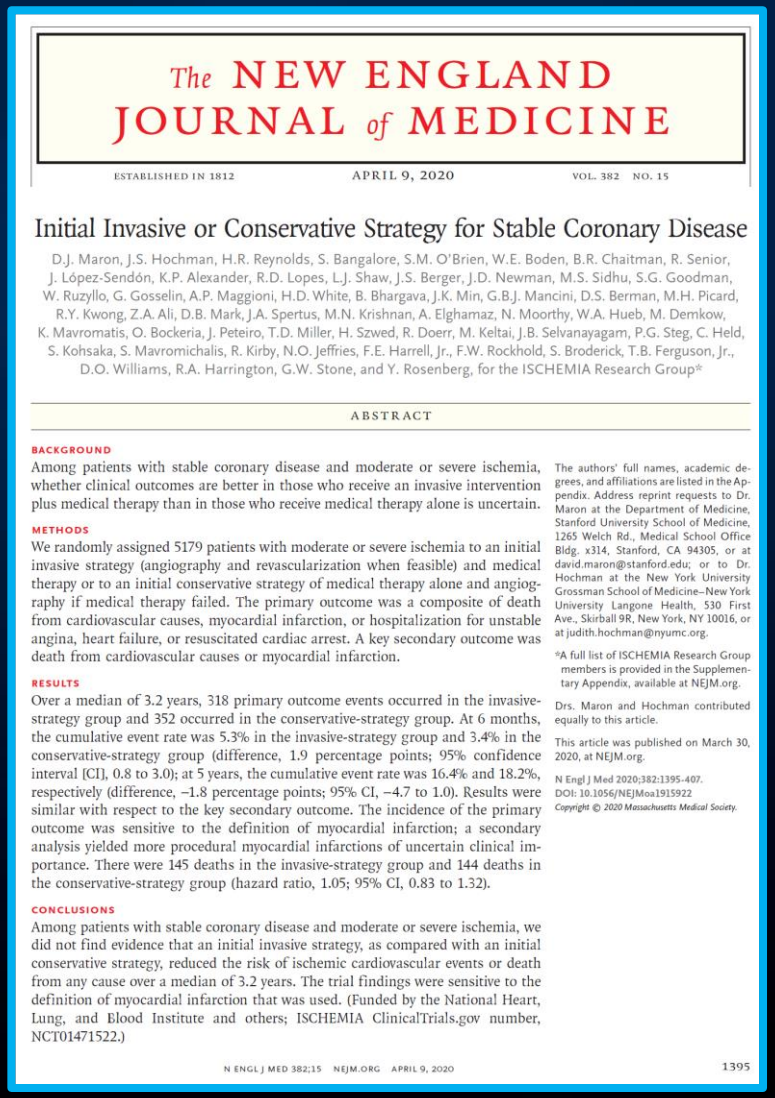
C) Decrease heart failure

D) Decrease angina

E) None of the above

ISCHEMIA

Stable CAD – Invasive or Not?



The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 APRIL 9, 2020 VOL. 382 NO. 15

Initial Invasive or Conservative Strategy for Stable Coronary Disease

D.J. Maron, J.S. Hochman, H.R. Reynolds, S. Bangalore, S.M. O'Brien, W.E. Boden, B.R. Chaitman, R. Senior, J. López-Sendón, K.P. Alexander, R.D. Lopes, L.J. Shaw, J.S. Berger, J.D. Newman, M.S. Sidhu, S.G. Goodman, W. Ruzyllo, G. Gosselin, A.P. Maggioni, H.D. White, B. Bhargava, J.K. Min, G.B.J. Mancini, D.S. Berman, M.H. Picard, R.Y. Kwong, Z.A. Ali, D.B. Mark, J.A. Spertus, M.N. Krishnan, A. Elghamaz, N. Moorthy, W.A. Hueb, M. Demkow, K. Mavromatis, O. Bockeria, J. Peteiro, T.D. Miller, H. Szwed, R. Doerr, M. Keltai, J.B. Selvanayagam, P.G. Steg, C. Held, S. Kohsaka, S. Mavromichalis, R. Kirby, N.O. Jeffries, F.E. Harrell, Jr., F.W. Rockhold, S. Broderick, T.B. Ferguson, Jr., D.O. Williams, R.A. Harrington, G.W. Stone, and Y. Rosenberg, for the ISCHEMIA Research Group*

ABSTRACT

BACKGROUND

Among patients with stable coronary disease and moderate or severe ischemia, whether clinical outcomes are better in those who receive an invasive intervention plus medical therapy than in those who receive medical therapy alone is uncertain.

METHODS

We randomly assigned 5179 patients with moderate or severe ischemia to an initial invasive strategy (angiography and revascularization when feasible) and medical therapy or to an initial conservative strategy of medical therapy alone and angiography if medical therapy failed. The primary outcome was a composite of death from cardiovascular causes, myocardial infarction, or hospitalization for unstable angina, heart failure, or resuscitated cardiac arrest. A key secondary outcome was death from cardiovascular causes or myocardial infarction.

RESULTS

Over a median of 3.2 years, 318 primary outcome events occurred in the invasive-strategy group and 352 occurred in the conservative-strategy group. At 6 months, the cumulative event rate was 5.3% in the invasive-strategy group and 3.4% in the conservative-strategy group (difference, 1.9 percentage points; 95% confidence interval [CI], 0.8 to 3.0); at 5 years, the cumulative event rate was 16.4% and 18.2%, respectively (difference, −1.8 percentage points; 95% CI, −4.7 to 1.0). Results were similar with respect to the key secondary outcome. The incidence of the primary outcome was sensitive to the definition of myocardial infarction; a secondary analysis yielded more procedural myocardial infarctions of uncertain clinical importance. There were 145 deaths in the invasive-strategy group and 144 deaths in the conservative-strategy group (hazard ratio, 1.05; 95% CI, 0.83 to 1.32).

CONCLUSIONS

Among patients with stable coronary disease and moderate or severe ischemia, we did not find evidence that an initial invasive strategy, as compared with an initial conservative strategy, reduced the risk of ischemic cardiovascular events or death from any cause over a median of 3.2 years. The trial findings were sensitive to the definition of myocardial infarction that was used. (Funded by the National Heart, Lung, and Blood Institute and others; ISCHEMIA ClinicalTrials.gov number, NCT01471522.)

The authors' full names, academic degrees, and affiliations are listed in the Appendix. Address reprint requests to Dr. Maron at the Department of Medicine, Stanford University School of Medicine, 1265 Welch Rd., Medical School Office Bldg. x314, Stanford, CA 94305, or at david.maron@stanford.edu; or to Dr. Hochman at the New York University Grossman School of Medicine–New York University Langone Health, 530 First Ave., Skirball 9R, New York, NY 10016, or at judith.hochman@nyumc.org.

*A full list of ISCHEMIA Research Group members is provided in the Supplementary Appendix, available at NEJM.org.

Drs. Maron and Hochman contributed equally to this article.

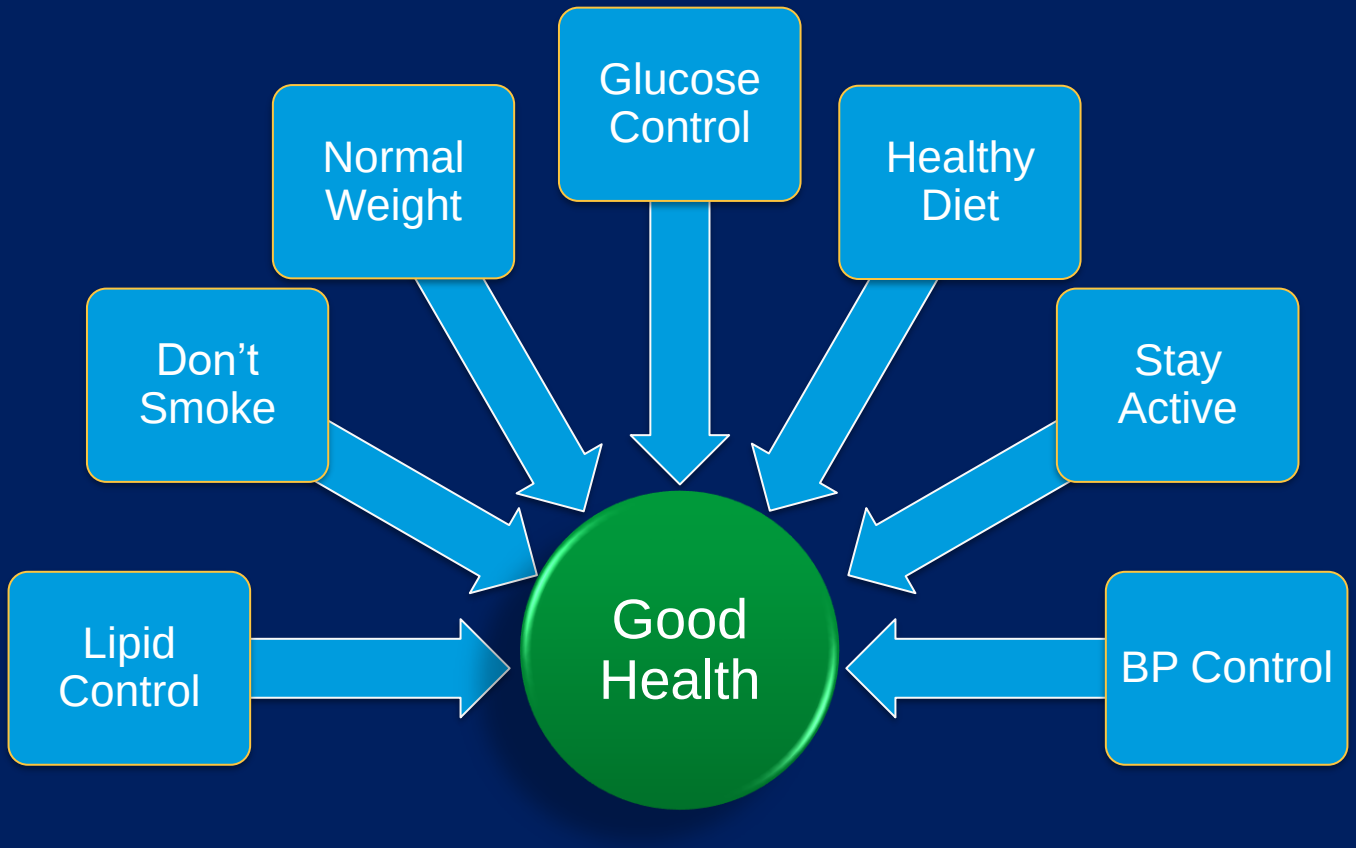
This article was published on March 30, 2020, at NEJM.org.

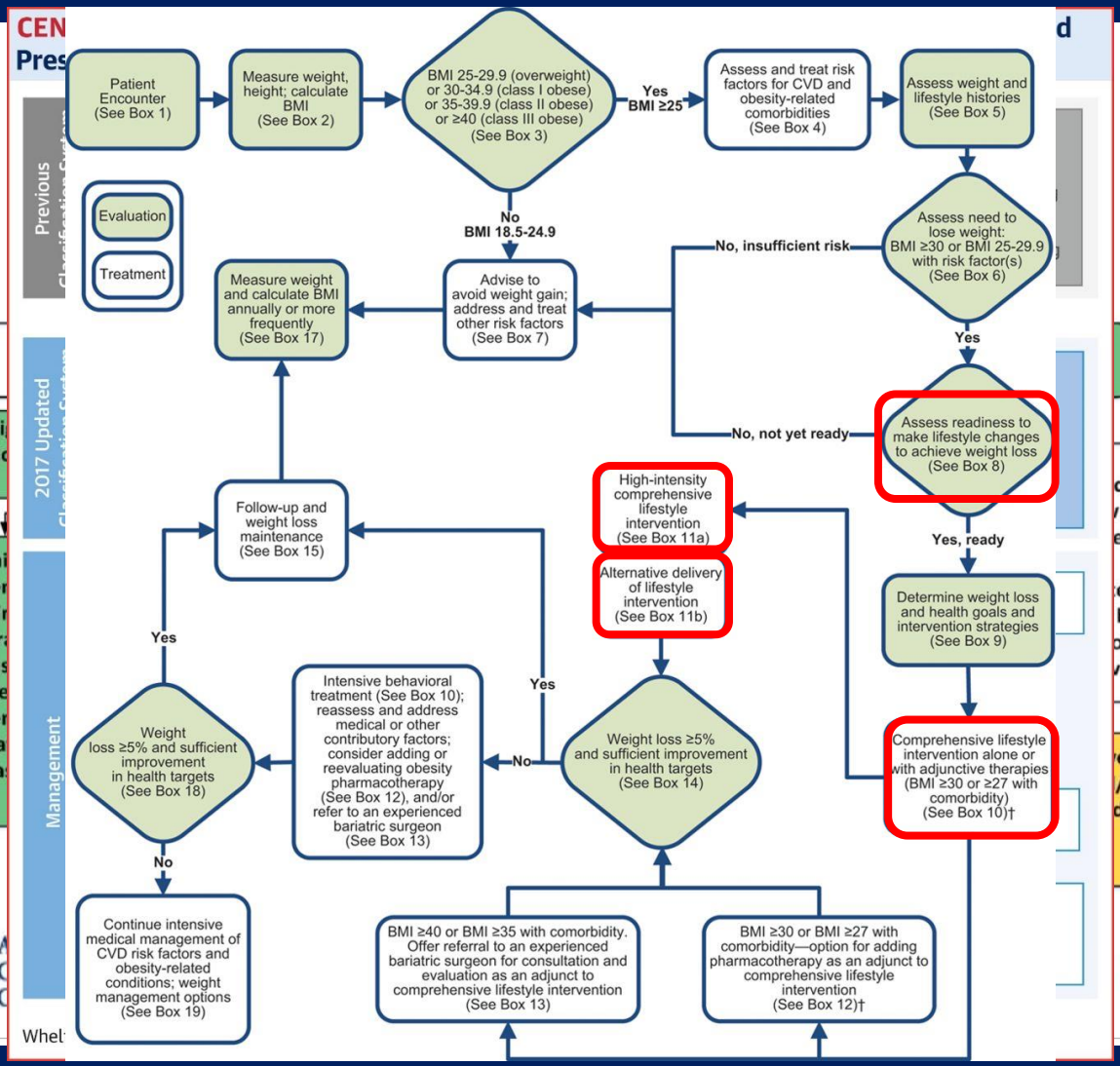
N Engl J Med 2020;382:1395-407.
DOI: 10.1056/NEJMoa1915922

- **n= 5179 subjects**
- **Moderate or severe ischemia**
- **Initial invasive + OMT vs. initial OMT**
- **Initial invasive approach did not change outcomes or death rates.**

N Engl J Med 2020;382:1395-407.

Our Patients Are NOT getting the best care.


Glucose Control
Normal Weight
Healthy Diet
Don’t Smoke
Stay Active
Lipid Control
Good Health
BP Control


Previous Classification System
Evaluation
Treatment
2017 Updated Classification System
Management
Patient Encounter (See Box 1)
Measure weight, height; calculate BMI (See Box 2)
BMI 25-29.9 (overweight) or 30-34.9 (class I obese) or 35-39.9 (class II obese) or ≥40 (class III obese) (See Box 3)
Yes BMI ≥25
Assess and treat risk factors for CVD and obesity-related comorbidities (See Box 4)
Assess weight and lifestyle histories (See Box 5)
No BMI 18.5-24.9
Assess need to lose weight: BMI ≥30 or BMI 25-29.9 with risk factor(s) (See Box 6)
No, insufficient risk
Advise to avoid weight gain; address and treat other risk factors (See Box 7)
Measure weight and calculate BMI annually or more frequently (See Box 17)
Yes
Assess readiness to make lifestyle changes to achieve weight loss (See Box 8)
No, not yet ready
Yes, ready
Determine weight loss and health goals and intervention strategies (See Box 9)
Comprehensive lifestyle intervention alone or with adjunctive therapies (BMI ≥30 or ≥27 with comorbidity) (See Box 10)†
High-intensity comprehensive lifestyle intervention (See Box 11a)
Alternative delivery of lifestyle intervention (See Box 11b)
Weight loss ≥5% and sufficient improvement in health targets (See Box 14)
Yes
No
Intensive behavioral treatment (See Box 10); reassess and address medical or other contributory factors; consider adding or reevaluating obesity pharmacotherapy (See Box 12), and/or refer to an experienced bariatric surgeon (See Box 13)
Weight loss ≥5% and sufficient improvement in health targets (See Box 18)
Yes
Follow-up and weight loss maintenance (See Box 15)
No
Continue intensive medical management of CVD risk factors and obesity-related conditions; weight management options (See Box 19)
BMI ≥40 or BMI ≥35 with comorbidity. Offer referral to an experienced bariatric surgeon for consultation and evaluation as an adjunct to comprehensive lifestyle intervention (See Box 13)
BMI ≥30 or BMI ≥27 with comorbidity—option for adding pharmacotherapy as an adjunct to comprehensive lifestyle intervention (See Box 12)†

MOST EFFECTIVE TREATMENT FOR CARDIOMETABOLIC DISEASE

Expert Medical Care

Treat the Root Causes

What percentage improvement in being free of Afib at 4 years does weight loss and lifestyle change provide for Afib above ablation and medication?

A) 25%

B) 50%

C) 100%

D) 500%

ORIGINAL INVESTIGATIONS

Impact of CARDIOrespiratory FITness on Arrhythmia Recurrence in Obese Individuals With Atrial Fibrillation

The CARDIO-FIT Study

Rajeev K. Pathak, MBBS,* Adrian Elliott, PhD,* Melissa E. Middeldorp,* Megan Meredith,* Abhinav B. Mehta, M Act St,† Rajiv Mahajan, MD, PhD,* Jeroen M.L. Hendriks, PhD,* Darragh Twomey, MBBS,* Jonathan M. Kalman, MBBS, PhD,† Walter P. Abhayaratna, MBBS, PhD,§ Dennis H. Lau, MBBS, PhD,* Prashanthan Sanders, MBBS, PhD*

ABSTRACT

BACKGROUND Obesity begets atrial fibrillation (AF). Although cardiorespiratory fitness is prot[illegible]e against incide[illegible] AF in obese individuals, its effect on AF recurrence or the benefit of cardiorespiratory fitness ga[illegible] unknown.

OBJECTIVES This study sought to evaluate the role of cardiorespiratory fitness and the incremental benefit of cardiorespiratory fitness improvement on rhythm control in obese individuals with AF.

METHODS Of 1,415 consecutive patients with AF, 825 had a body mass index ≥27 kg/m^2 and were offered risk factor management and participation in a tailored exercise program. After exclusions, 308 patients were included in the analysis. Patients underwent exercise stress testing to determine peak metabolic equivalents (METs). To determine a dose response, cardiorespiratory fitness was categorized as: low (<85%), adequate (86% to 100%), and high (>100%). Impact of cardiorespiratory fitness gain was ascertained by the objective gain in fitness at final follow-up (≥2 METs vs. <2 METs). AF rhythm control was determined using 7-day Holter monitoring and AF severity scale questionnaire.

RESULTS There were no differences in baseline characteristics or follow-up duration between the groups defined by cardiorespiratory fitness. Arrhythmia-free survival with and without rhythm control strategies was greatest in patients with high cardiorespiratory fitness compared to adequate or low cardiorespiratory fitness ($p < 0.001$ for both). AF burden and symptom severity decreased significantly in the group with cardiorespiratory fitness gain ≥2 METs as compared to <2 METs group ($p < 0.001$ for all). Arrhythmia-free survival with and without rhythm control strategies was greatest in those with METs gain ≥2 compared to those with METs gain <2 in cardiorespiratory fitness ($p < 0.001$ for both).

CONCLUSIONS Cardiorespiratory fitness predicts arrhythmia recurrence in obese individuals with symptomatic AF. Improvement in cardiorespiratory fitness augments the beneficial effects of weight loss. (Evaluating the Impact of a Weight Loss on the Burden of Atrial Fibrillation [AF] in Obese Patients; ACTRN12614001123639) (J Am Coll Cardiol 2015;66:985-96) © 2015 by the American College of Cardiology Foundation.

Meds and ablation alone

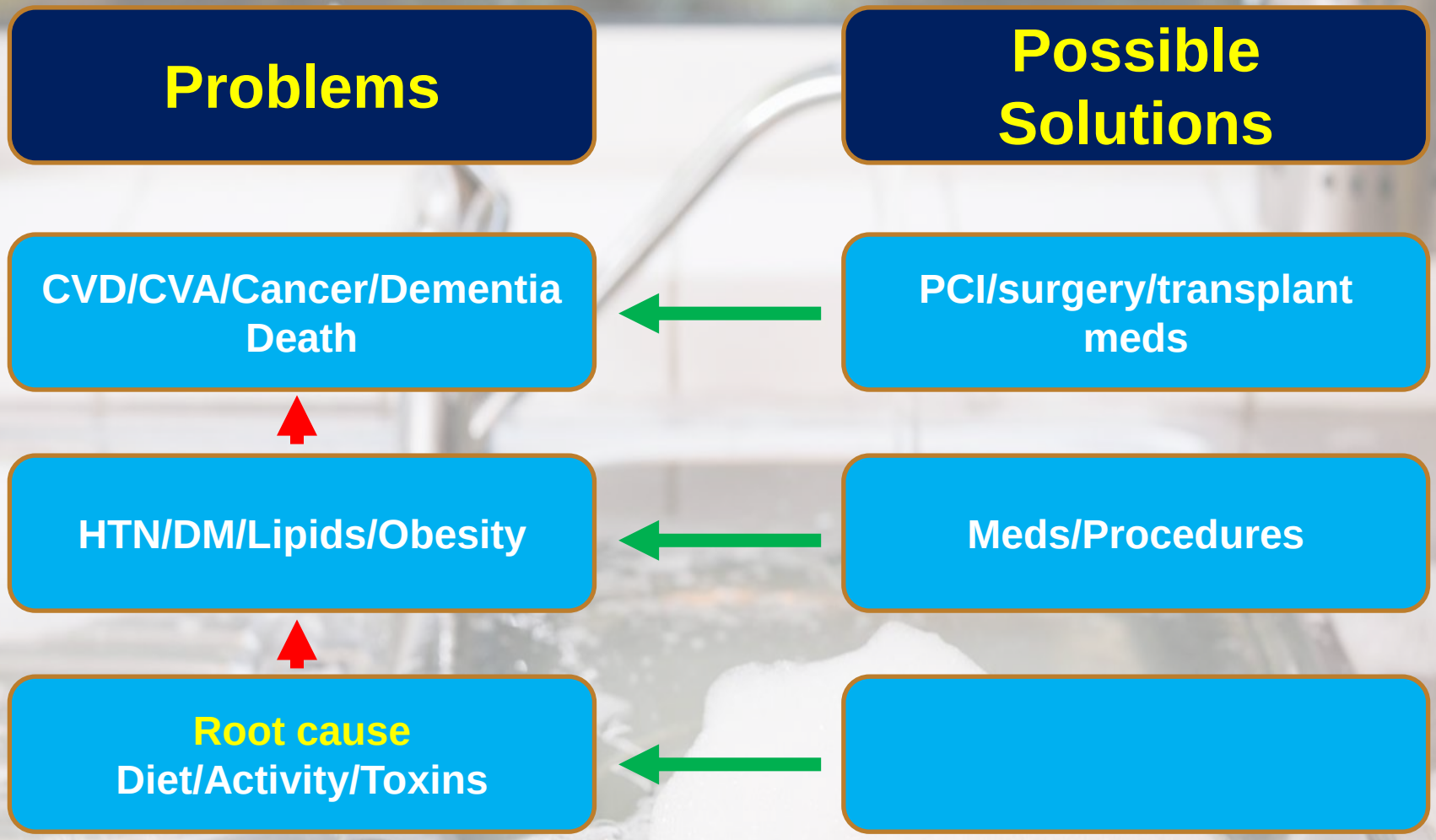
66% had more Afib

11X

Treat the root causes

6% had more Afib

Problems	Possible Solutions
Damage or destroy our house	Replace the floor/baseboards/dry wall Move
Sink overflowing	Mops More drains Bigger sink
Root cause Too much water coming in	Turn the water off (or at least down)

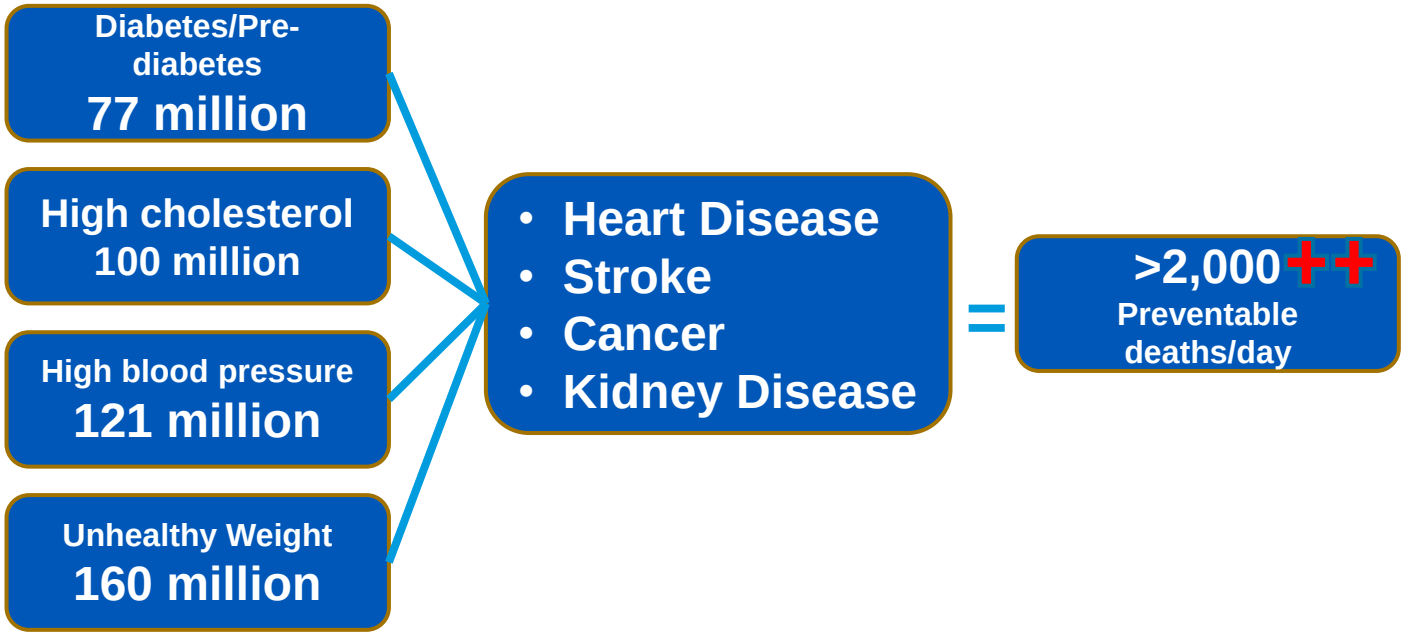

Problems
Possible Solutions
CVD/CVA/Cancer/Dementia Death
PCI/surgery/transplant meds
HTN/DM/Lipids/Obesity
Meds/Procedures
Root cause
Diet/Activity/Toxins

Is Lifestyle/Weight Loss Healthcare's Responsibility?

1. We're not paid for this.
2. Have enough to worry about.
3. Don't have time.

CARDIOMETABOLIC DISEASE

THE 21ST CENTURY EPIDEMIC



Update In Preventive Cardiology

R. Todd Hurst, MD FACC FASE

Email: todd@toddhurstmd.com

LinkedIn