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Foundations of Cardiometabolic Health Certification Course

Certified Cardiometabolic Health Professional (CCHP)

Hypertension

Definition/Screening/Diagnosis

Luke J. Laffin, MD FACC

Co-Director, Center for Blood Pressure Disorders

Medical Director of Cardiac Rehabilitation

Cleveland Clinic

Cleveland, Ohio USA

Outline

- Overview
- Epidemiology and Burden of Hypertension
- Pathophysiology of Hypertension
- Risk Factors for Hypertension
- Defining Hypertension
- Blood Pressure Measurement
- Diagnosis of Hypertension (Primary and Secondary)
- Cardiovascular Risk Factors Associated with Hypertension

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Hypertension: A Call to Action

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Cleveland Clinic

Cleveland, Ohio USA

Uncontrolled hypertension is getting worse in the U.S., study finds

High blood pressure can be major risk factor for severe illness from COVID-19

https://www.nbcnews.com/health/heart-health/uncontrolled-hypertension-getting-worse-u-s-study-finds-n1239678

Uncontrolled hypertension is getting worse in the U.S., study finds

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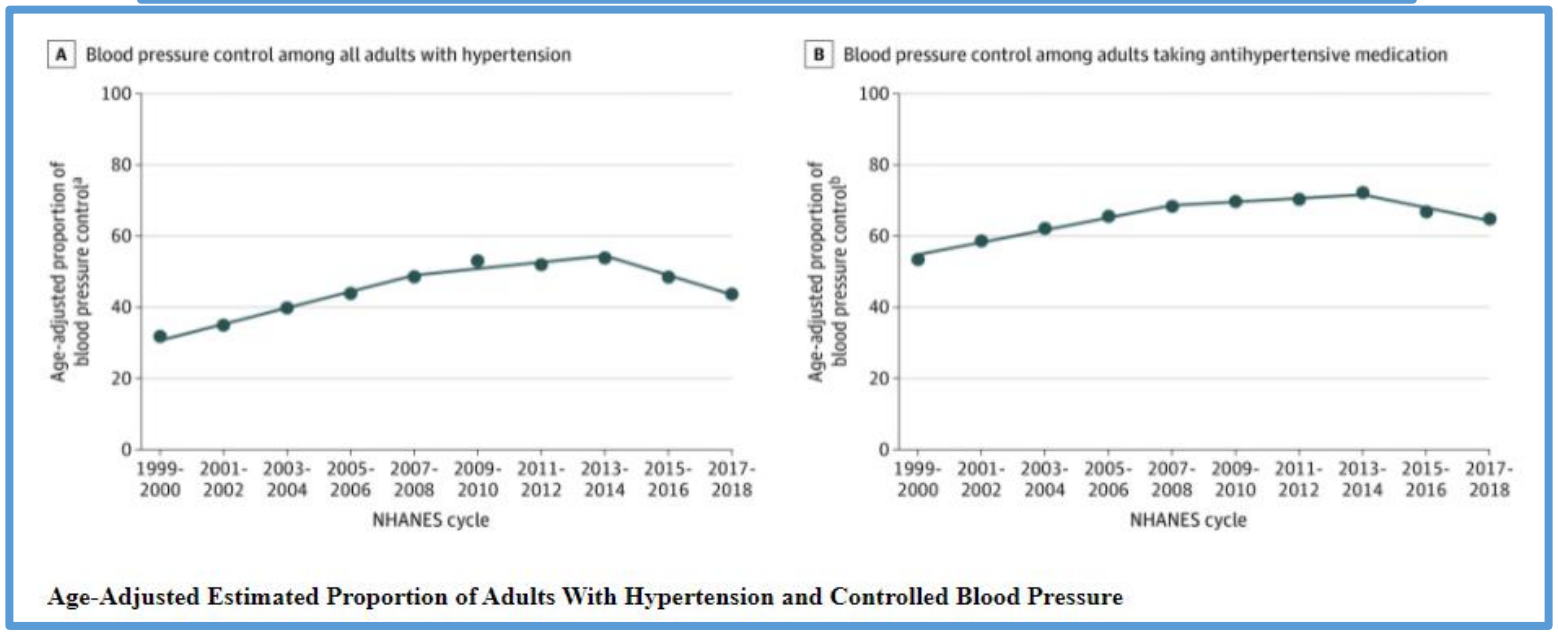
JAMA | Original Investigation

Trends in Blood Pressure Control Among US Adults With Hypertension, 1999-2000 to 2017-2018

Paul Muntner, PhD; Shakia T. Hardy, PhD; Lawrence J. Fine, MD; Byron C. Jaeger, PhD; Gregory Wozniak, PhD; Emily B. Levitan, ScD; Lisandro D. Colantonio, MD, PhD



Age-Adjusted Estimated Proportion of Adults With Hypertension and Controlled Blood Pressure

JAMA. 2020 Sep 22;324(12):1190-1200

https://www.nbcnews.com/health/heart-health/uncontrolled-hypertension-getting-worse-u-s-study-finds-n1239678

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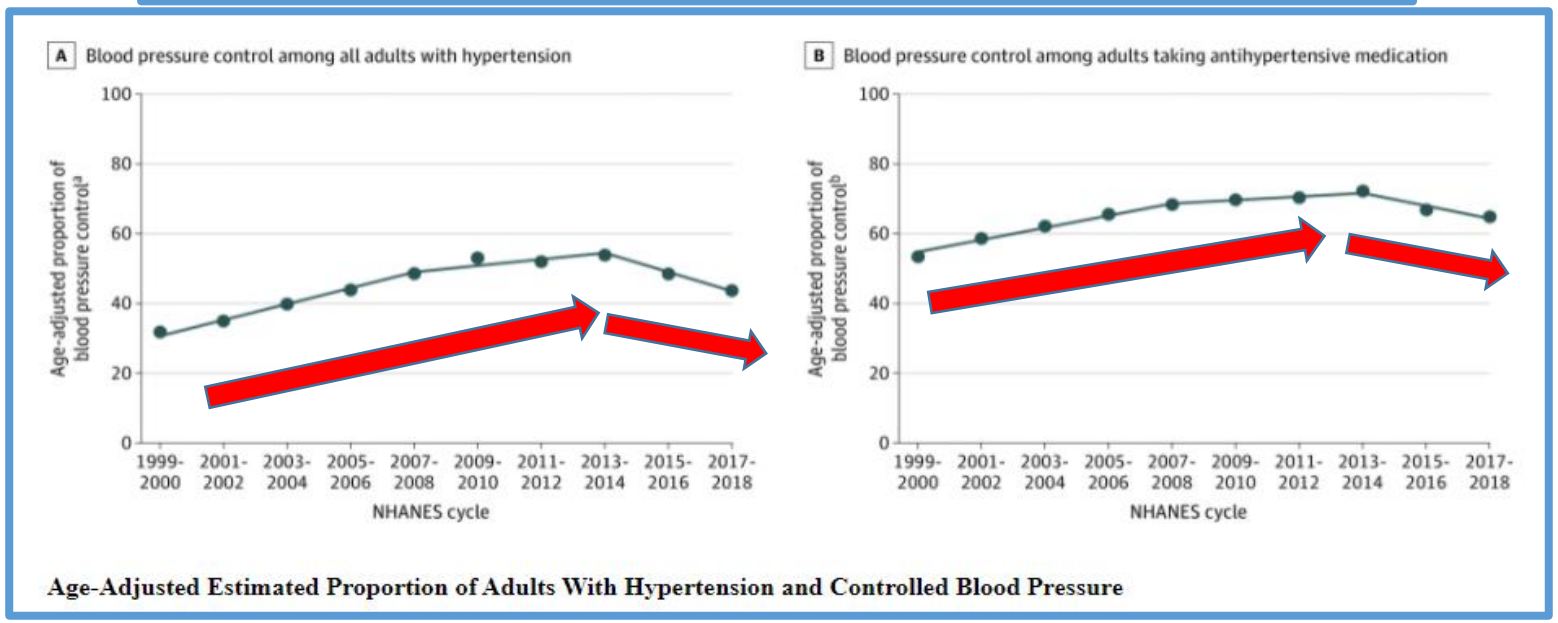

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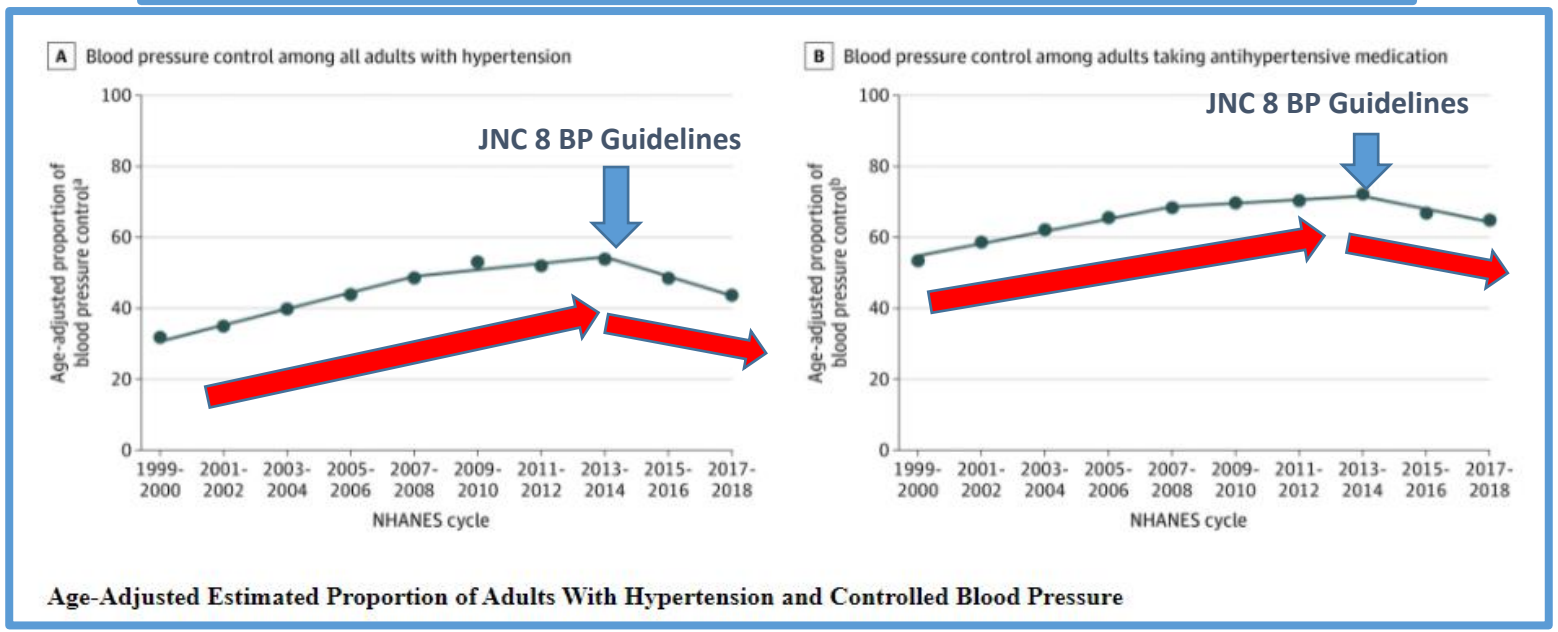

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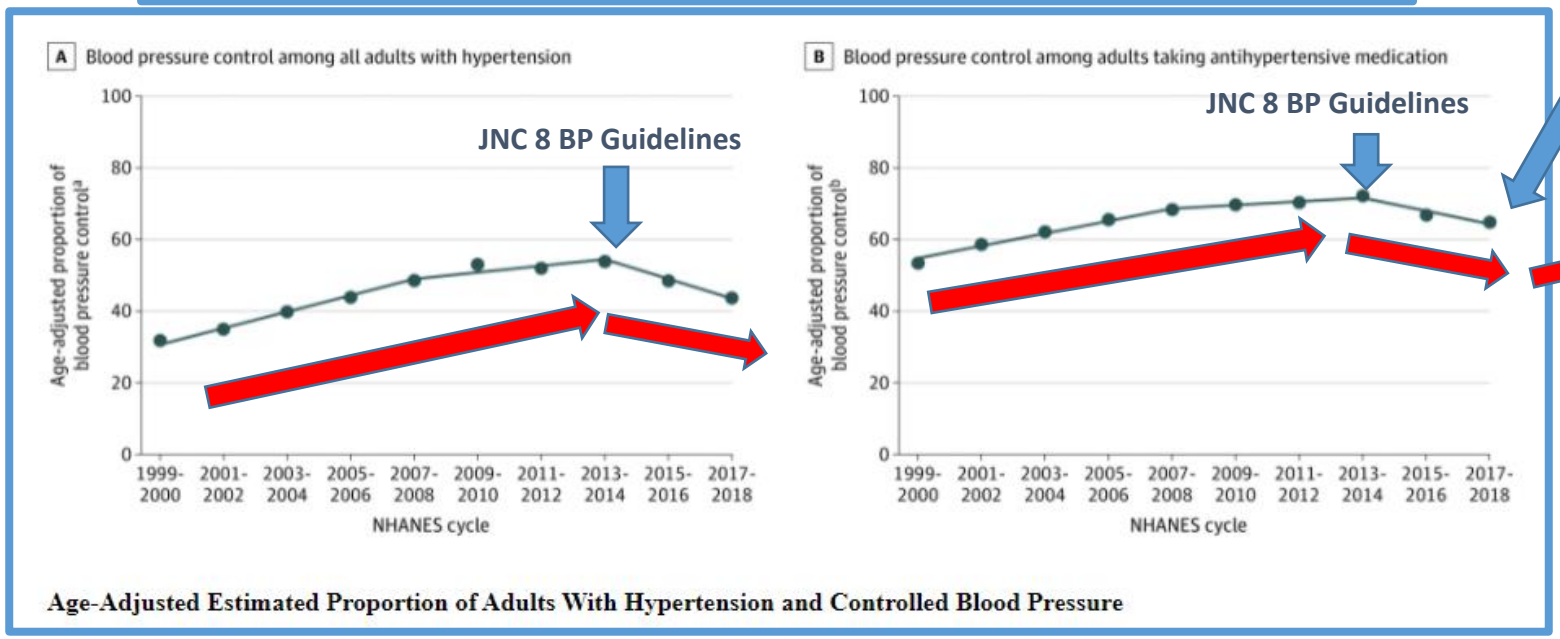

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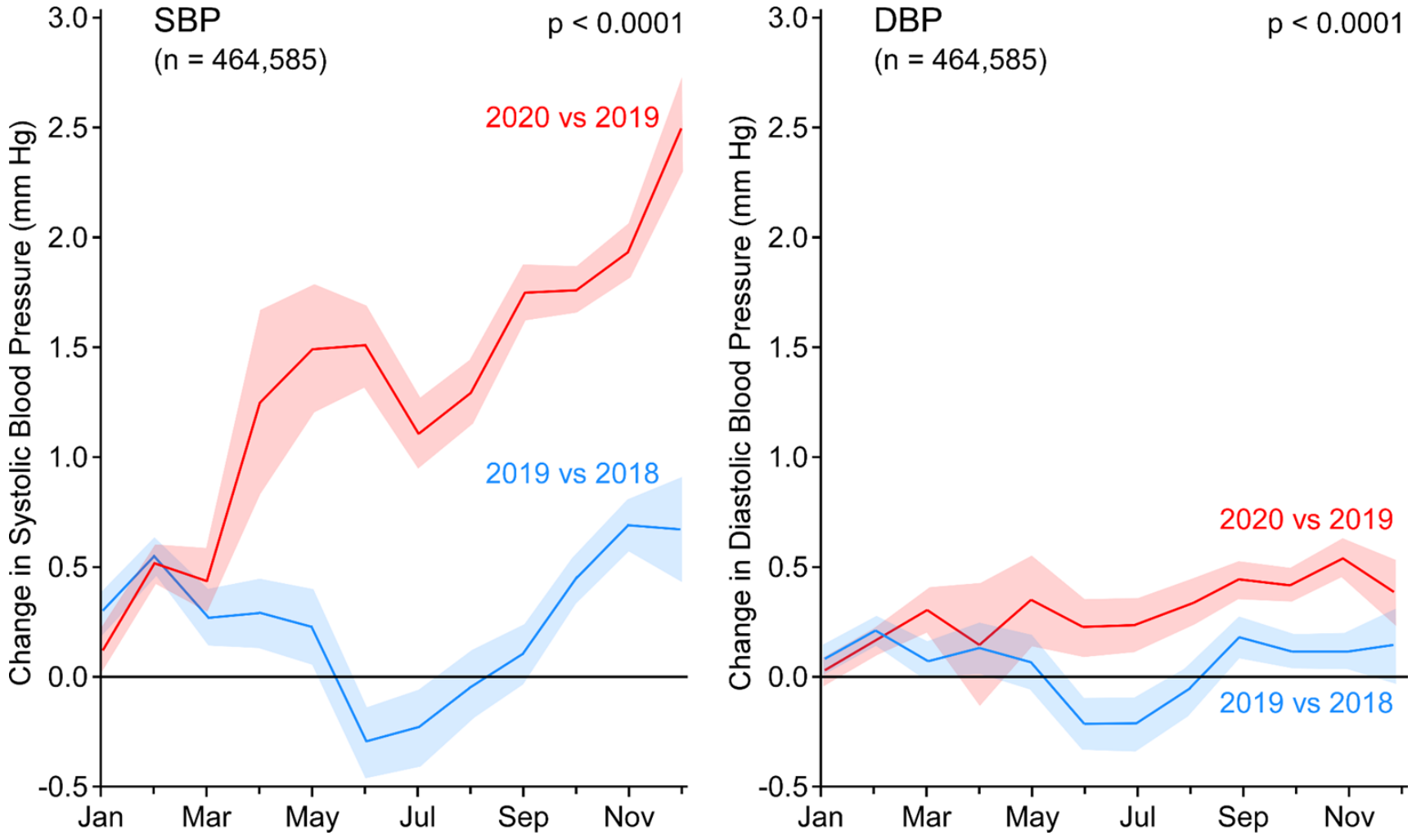
Global pandemic has not helped…



Circulation. 2022 Jan 18;145(3):235-23

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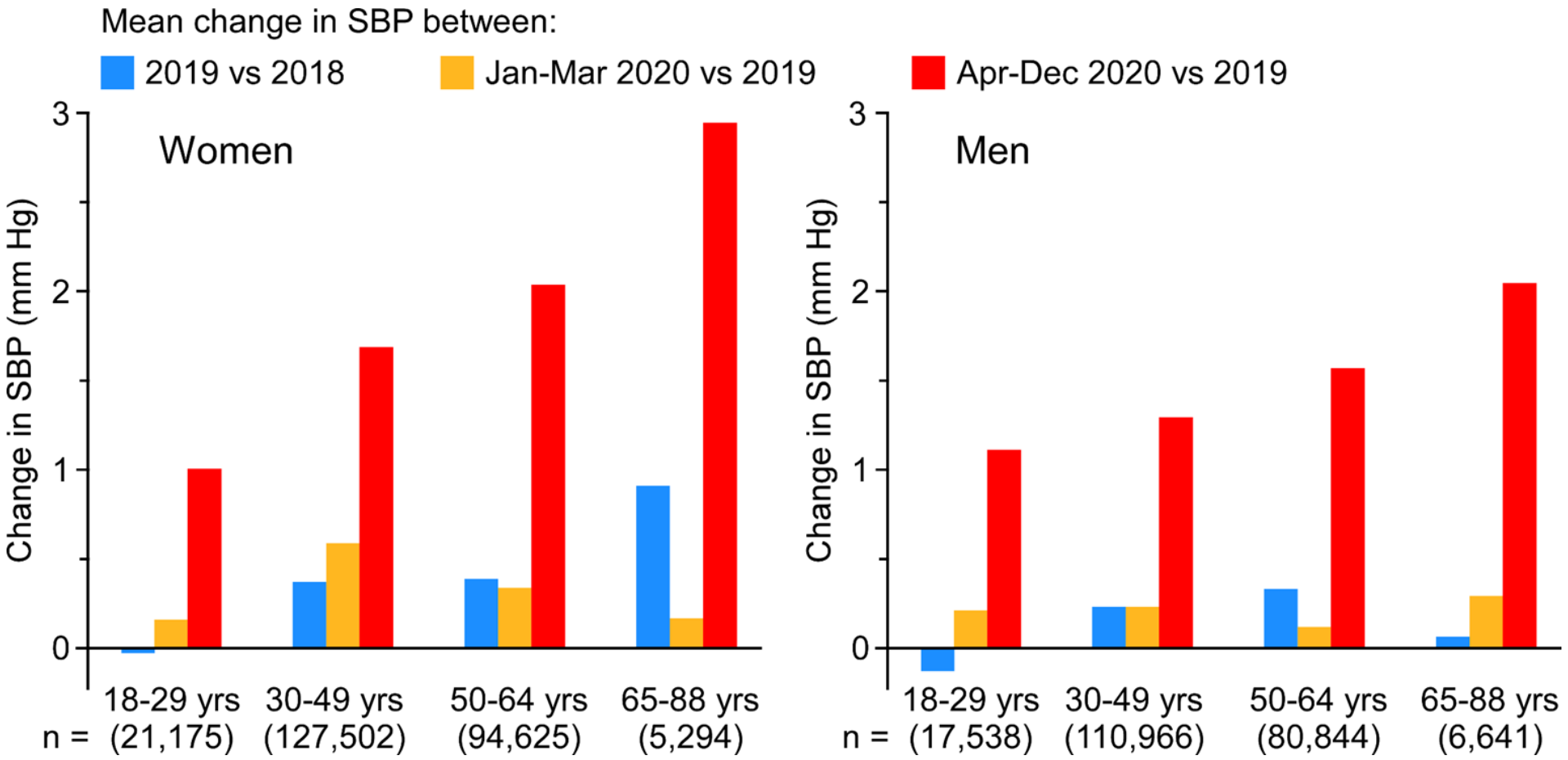
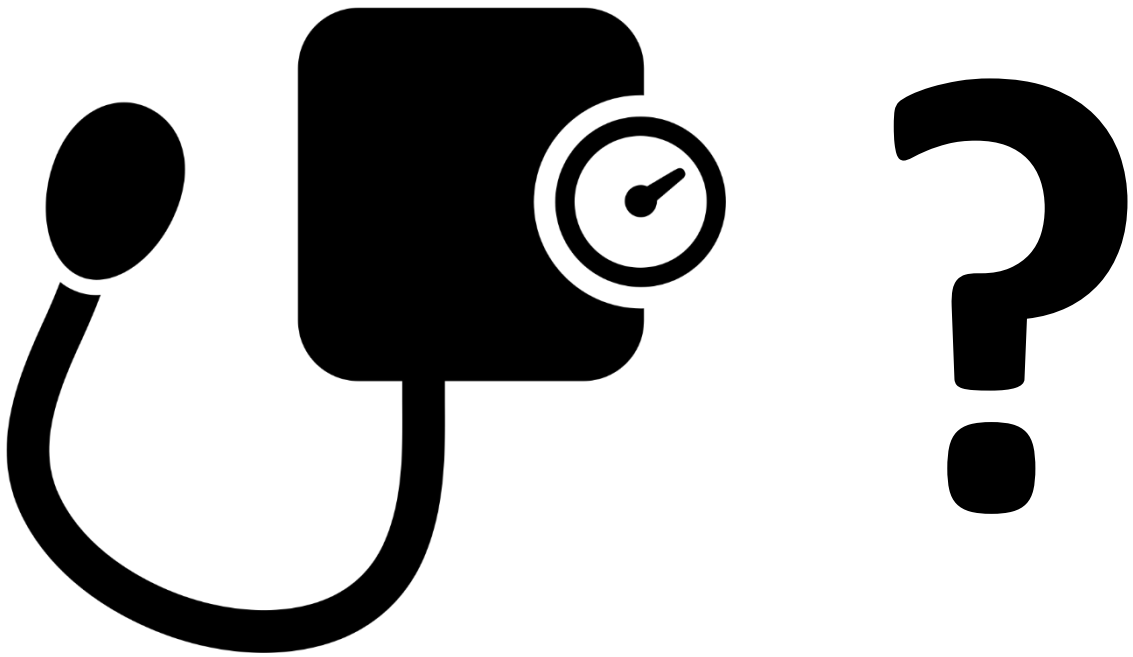


Circulation. 2022 Jan 18;145(3):235-23



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.

Goal #1:

Declare hypertension control a national priority

Goal #2:

Ensuring, encouraging, and utilizing community-level supports for hypertension control

Goal #3:

Achieving optimal clinical care for patients with hypertension

US Department of Health & Human Services. Surgeon General's Call to Action to Control Hypertension. Published October 7, 2020.
JAMA. 2020;324(18):1825-1826.

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Epidemiology and Burden of Hypertension

Luke J. Laffin, MD FACC

Co-Director, Center for Blood Pressure Disorders

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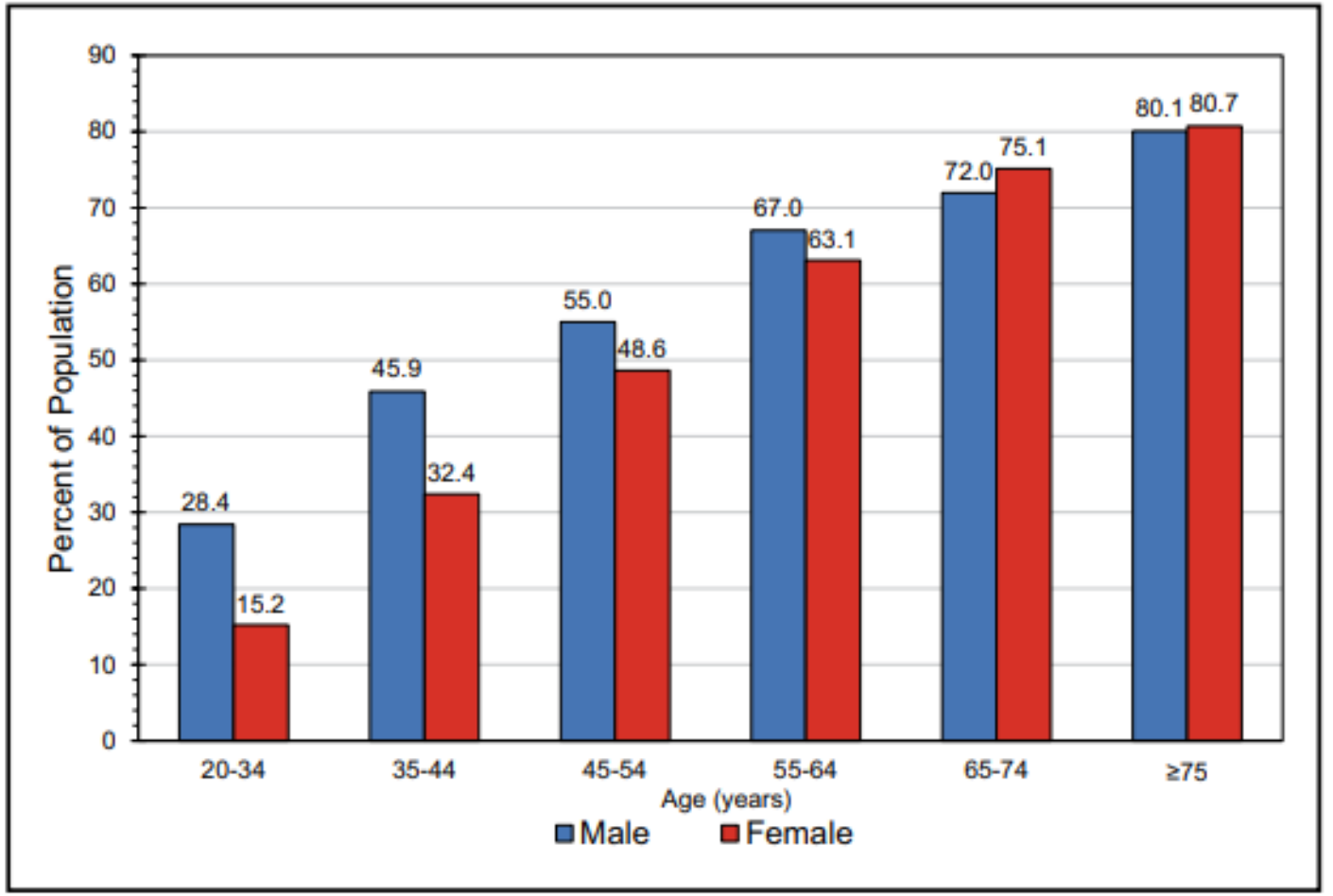
Cleveland Clinic

Cleveland, Ohio USA

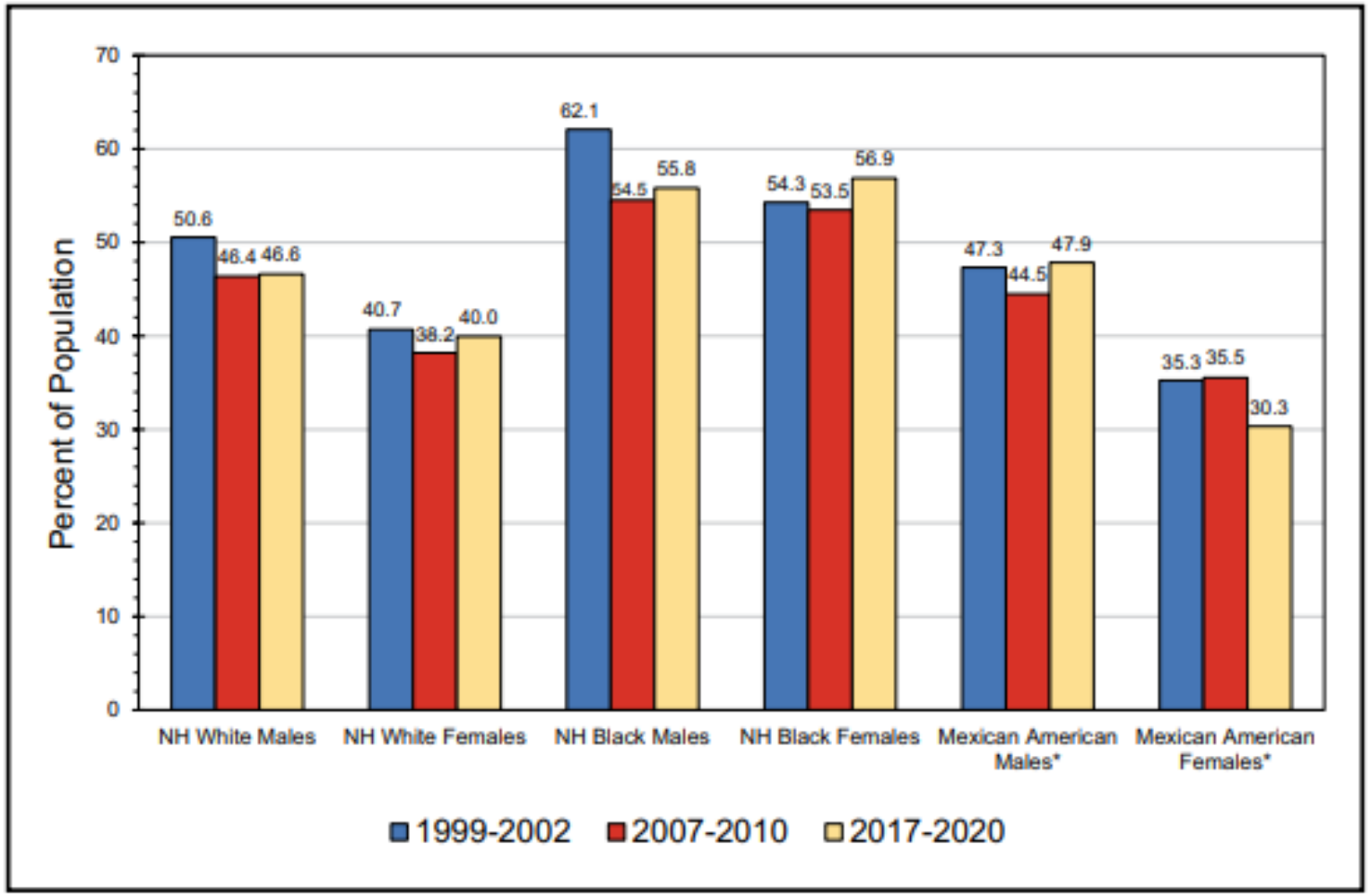
Prevalence of hypertension in US adults >20 years of age by sex and age (NHANES, 2017-2020)



Hypertension is defined in terms of NHANES blood pressure measurements and health interviews. A person was considered to have hypertension if he or she had **SBP ≥130 mm Hg or DBP ≥80 mm Hg**, if he or she said "yes" to taking antihypertensive medication, or if the person was told on 2 occasions that he or she had hypertension.

Circulation. 2023;147:e93–e621.

Age-adjusted prevalence trends for hypertension in US adults >20 years of age by race/ethnicity, sex, and survey year (NHANES 1999-2002, 2007-2010, and 2017-2020)



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Hypertension Awareness, Treatment, and Control: NHANES 1999 to 2002, 2007 to 2010, and 2017 to 2020 Age-Adjusted Percent With Hypertension in US Adults by Sex and Race/Ethnicity

	Awareness, %			Treatment, %			Control, %		
	1999–2002	2007–2010	2017–2020	1999–2002	2007–2010	2017–2020	1999–2002	2007–2010	2017–2020
Overall	48.9	61.2	62.0	37.7	52.5	52.6	12.0	24.1	25.7
NH White males	42.7	58.0	62.0	31.4	48.7	50.4	10.9	22.2	26.7
NH White females	56.7	66.1	62.9	45.9	59.2	56.4	14.8	28.7	27.6
NH Black males	46.0	60.5	61.5	33.0	47.6	48.4	9.1	18.2	17.3
NH Black females	67.7	73.5	71.2	54.9	64.3	61.0	16.4	28.2	25.6
Mexican American males*	25.9	40.6	47.7	14.0	30.5	36.2	4.1	12.7	20.6
Mexican American females*	50.4	55.6	60.5	35.4	49.3	49.9	10.4	21.2	23.9

Values are percentages. Hypertension is defined in terms of NHANES blood pressure measurements and health interviews. A person was considered to have hypertension if he or she had **SBP ≥130 mm Hg or DBP ≥80 mm Hg**, if he or she said “yes” to taking antihypertensive medication, or if the person was told on 2 occasions that he or she had hypertension.

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NH White females	56.7	66.1	62.9	45.9	59.2	56.4	14.8	28.7	27.6 ↓
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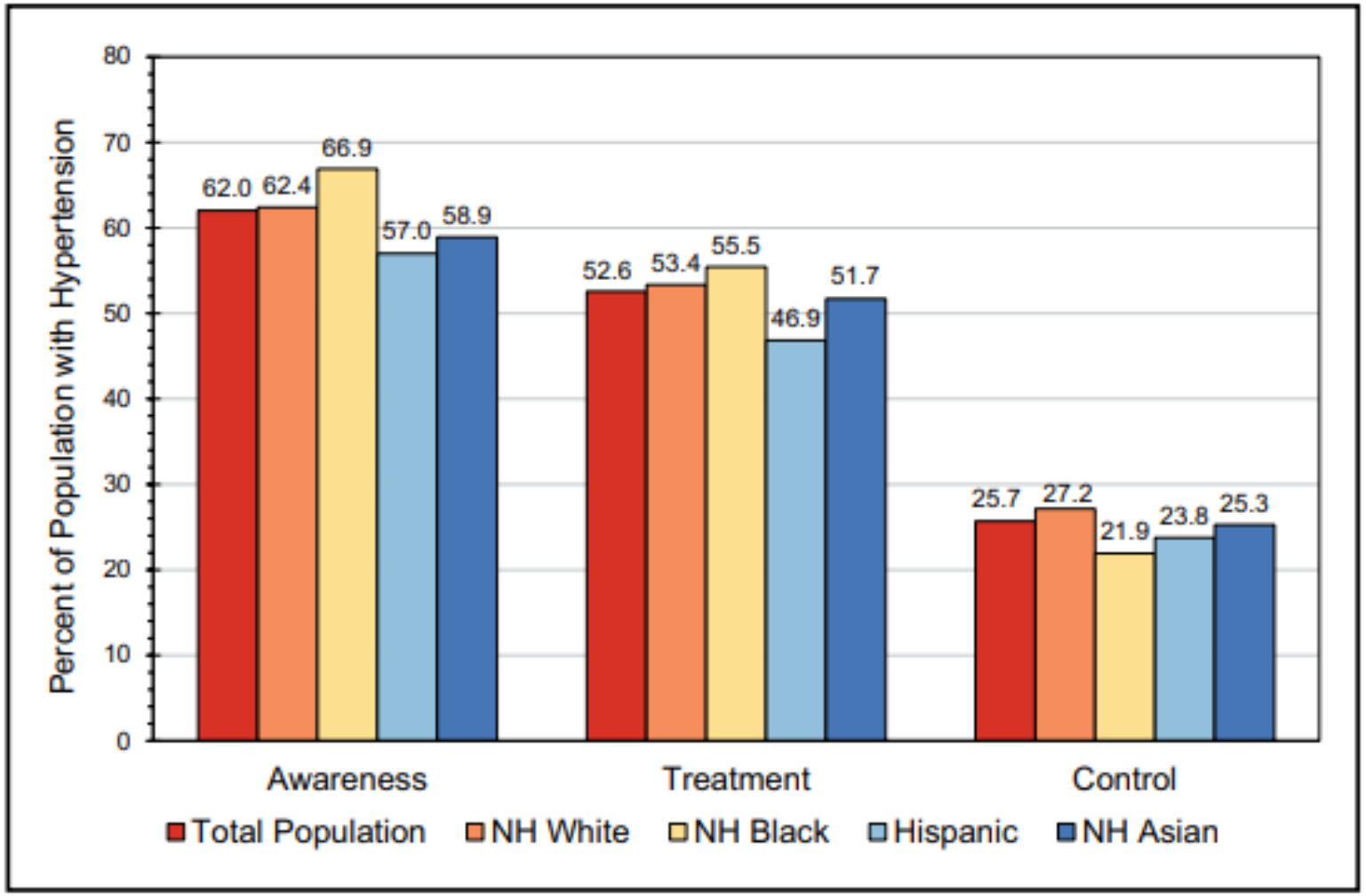
Circulation. 2023;147:e93–e621.

Extent of awareness, treatment, and control of high blood pressure by race/ethnicity and sex, United States (NHANES, 2017-2020)



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Circulation. 2023;147:e93–e621.

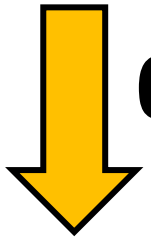
Impact of Blood Pressure Control on Cardiovascular Outcomes

HR per 5 mm Hg reduction in systolic blood pressure

Major Cardiovascular Disease

0.90

Heart Failure



0.87

Cardiovascular Death

0.95

The Blood Pressure Lowering Treatment Trialists' Collaboration

344,716 participants from 48 randomized clinical trials were available for this analysis

Irrespective of previous diagnoses of cardiovascular disease, and even at normal or high–normal blood pressure values

Lancet 2021; 397: 1625–36

$$$ Healthcare Expenditures Related to Hypertension $$$

Individuals with hypertension face nearly **$2000 higher** annual healthcare expenditure compared with their non-hypertensive peers.

National medical costs associated with hypertension account for about **$131 billion**, or **over 3%** of the $3 trillion US national healthcare expenditure

J Am Heart Assoc. 2018;7:e008731

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Pathophysiology of Hypertension

Luke J. Laffin, MD FACC

Co-Director, Center for Blood Pressure Disorders

Medical Director of Cardiac Rehabilitation

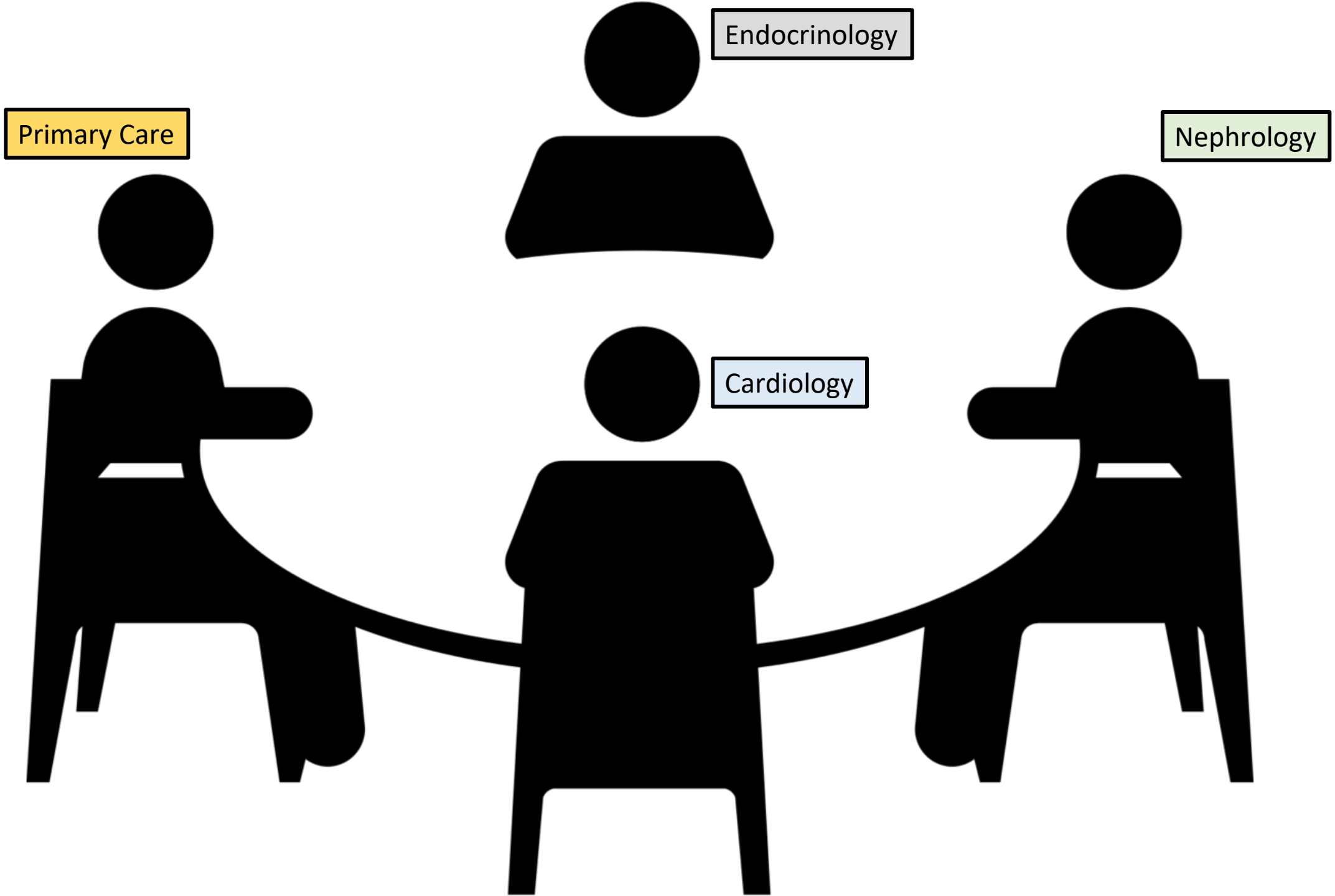
Cleveland Clinic

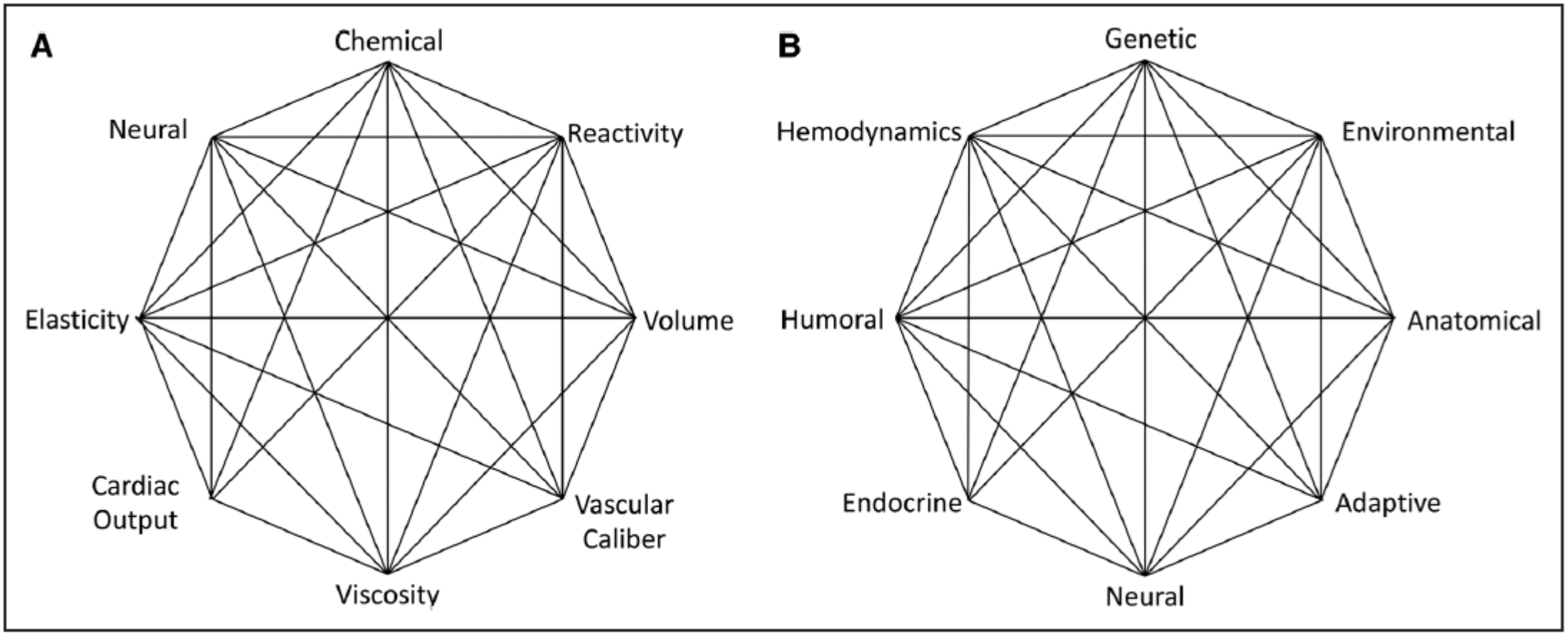
Cleveland, Ohio USA

Primary hypertension (no longer called essential hypertension)

Secondary hypertension (to discuss evaluation later in talk)

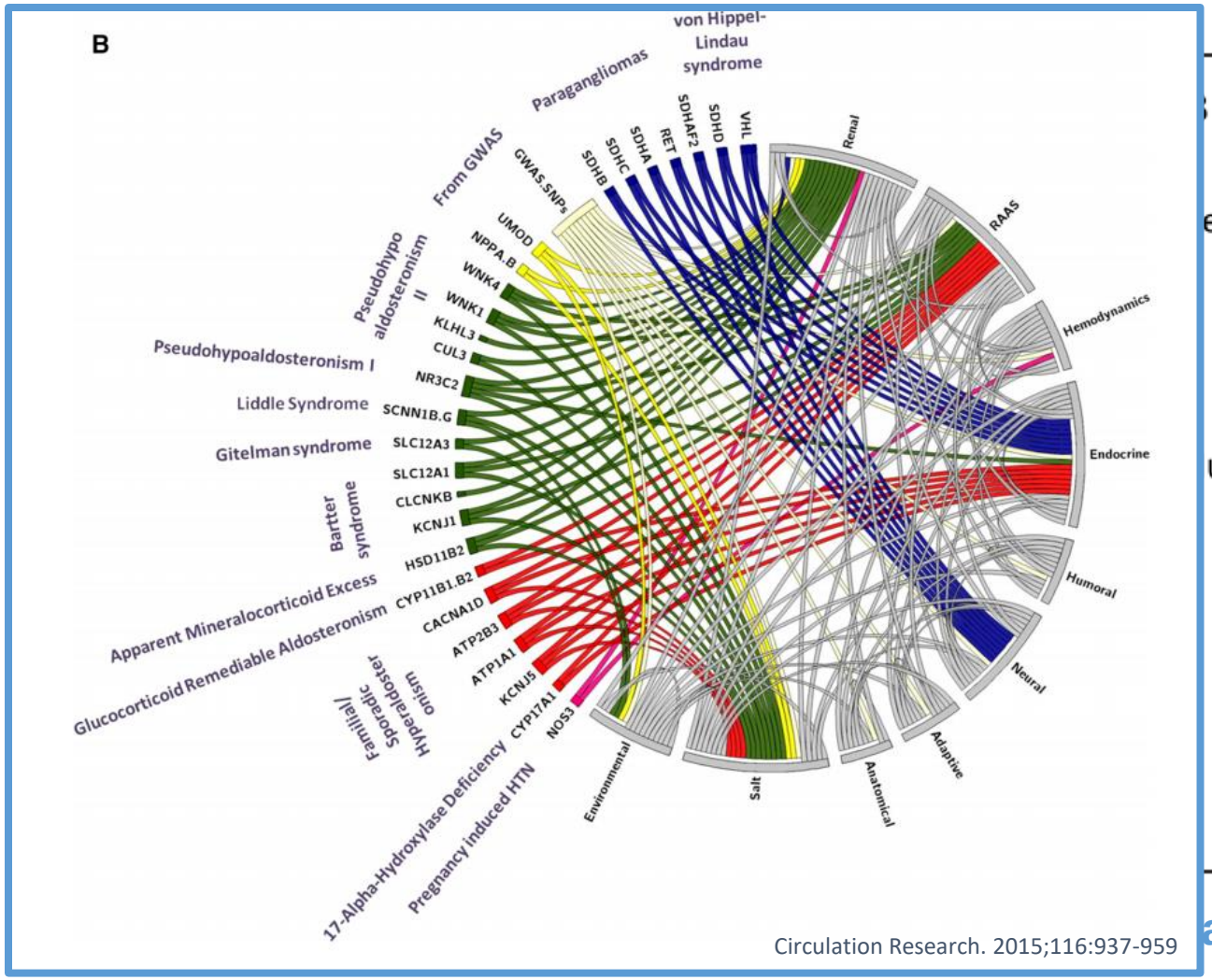
Many factors at play making hypertension a very interesting field but challenging to treat!


Endocrinology
Primary Care
Nephrology
Cardiology

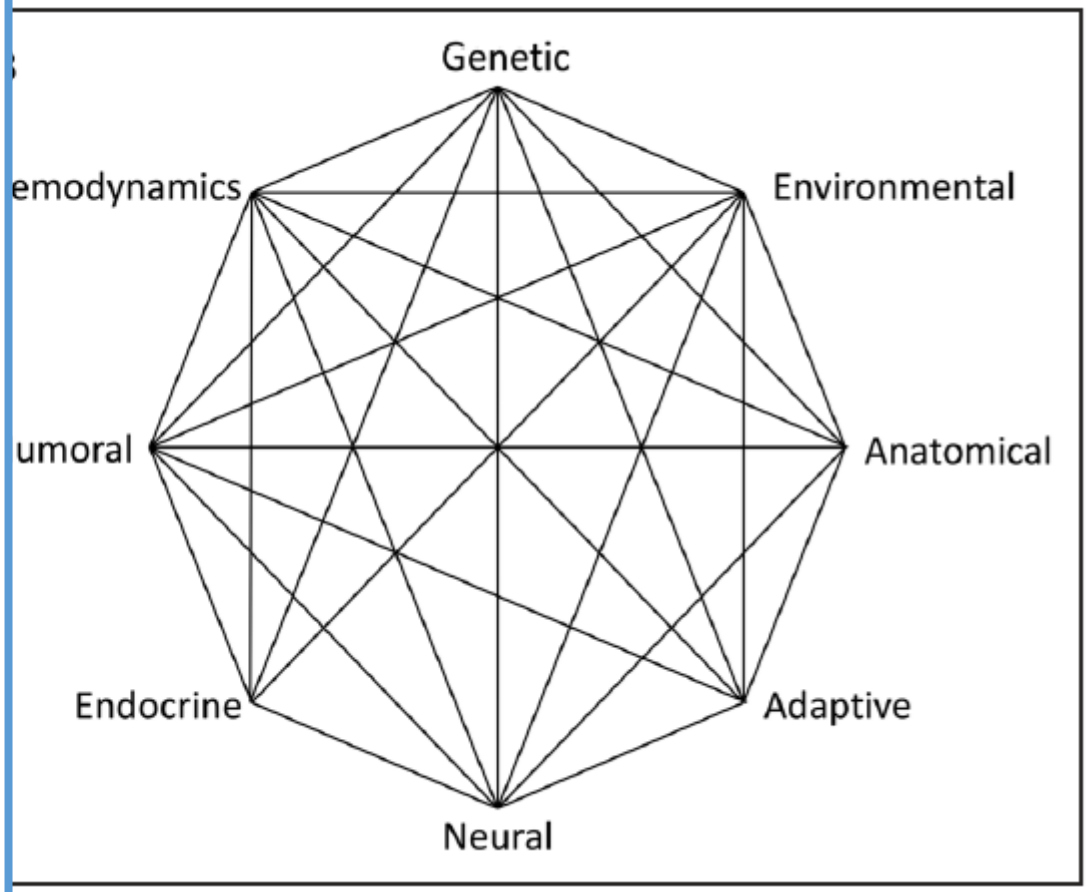


The original (A) and revised (B) Mosaic Theories proposed by Page.

Circulation Research. 2021;128:847–863.
JAMA. 1949; 140(5):451-458.



Circulation Research. 2015;116:937-959



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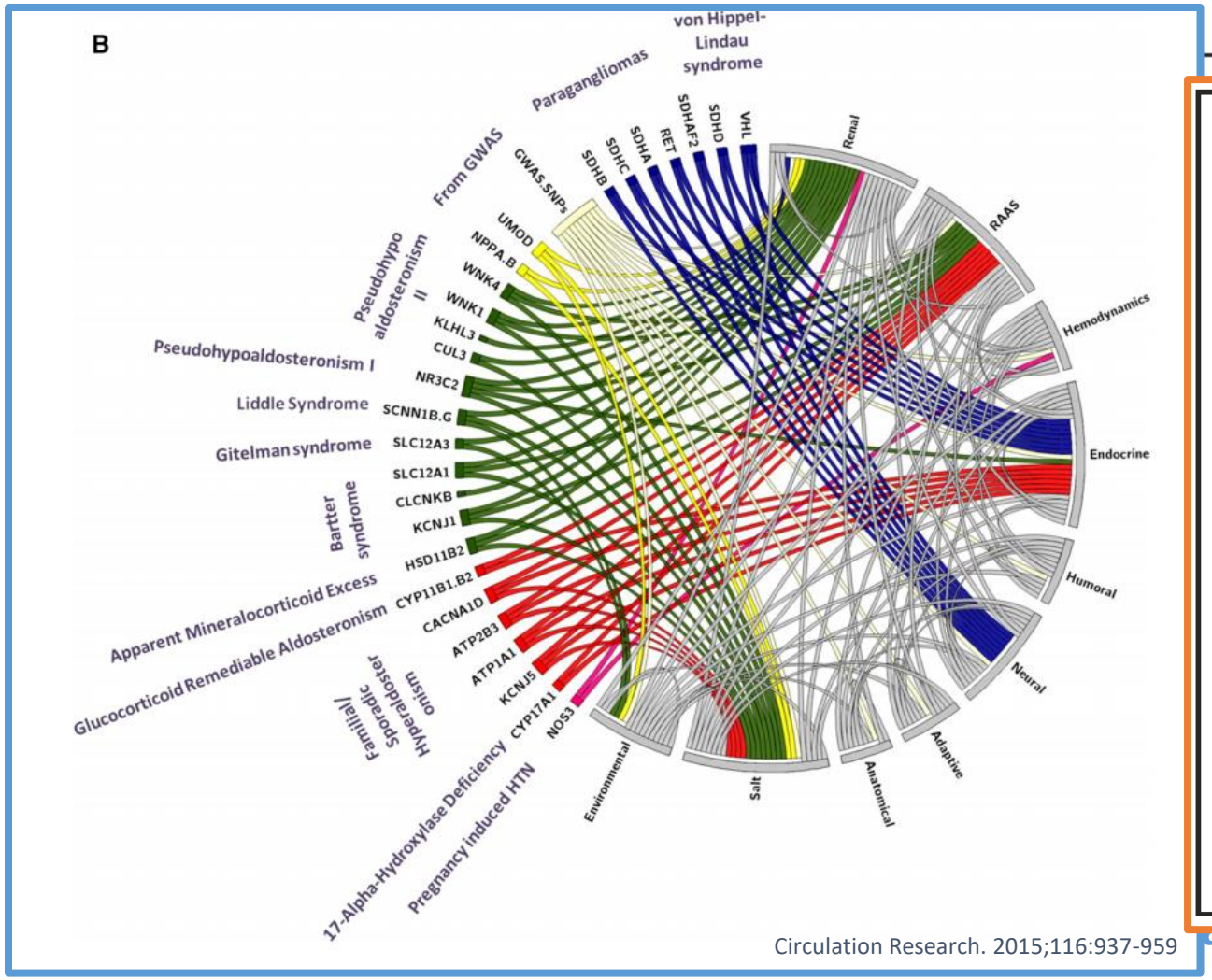

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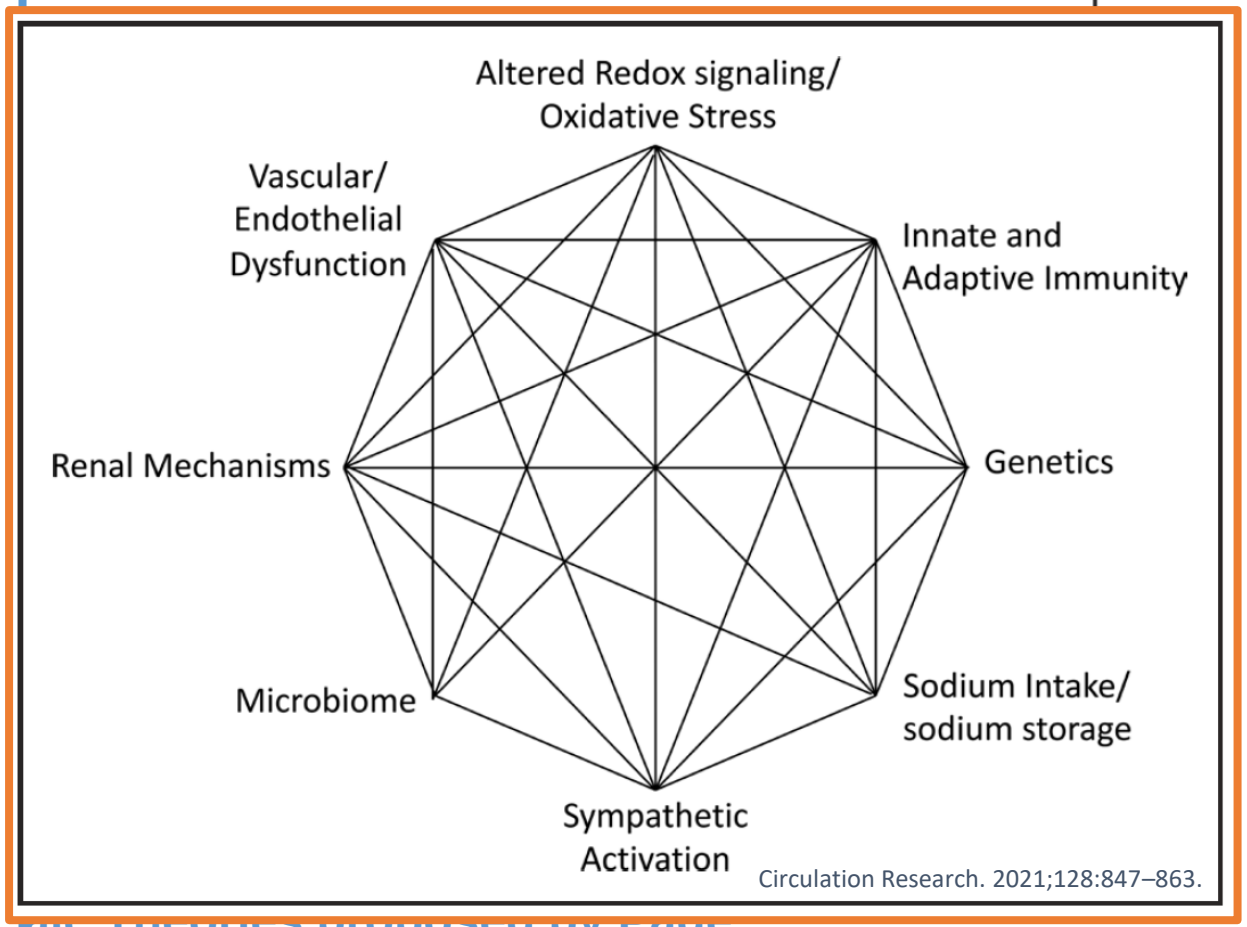

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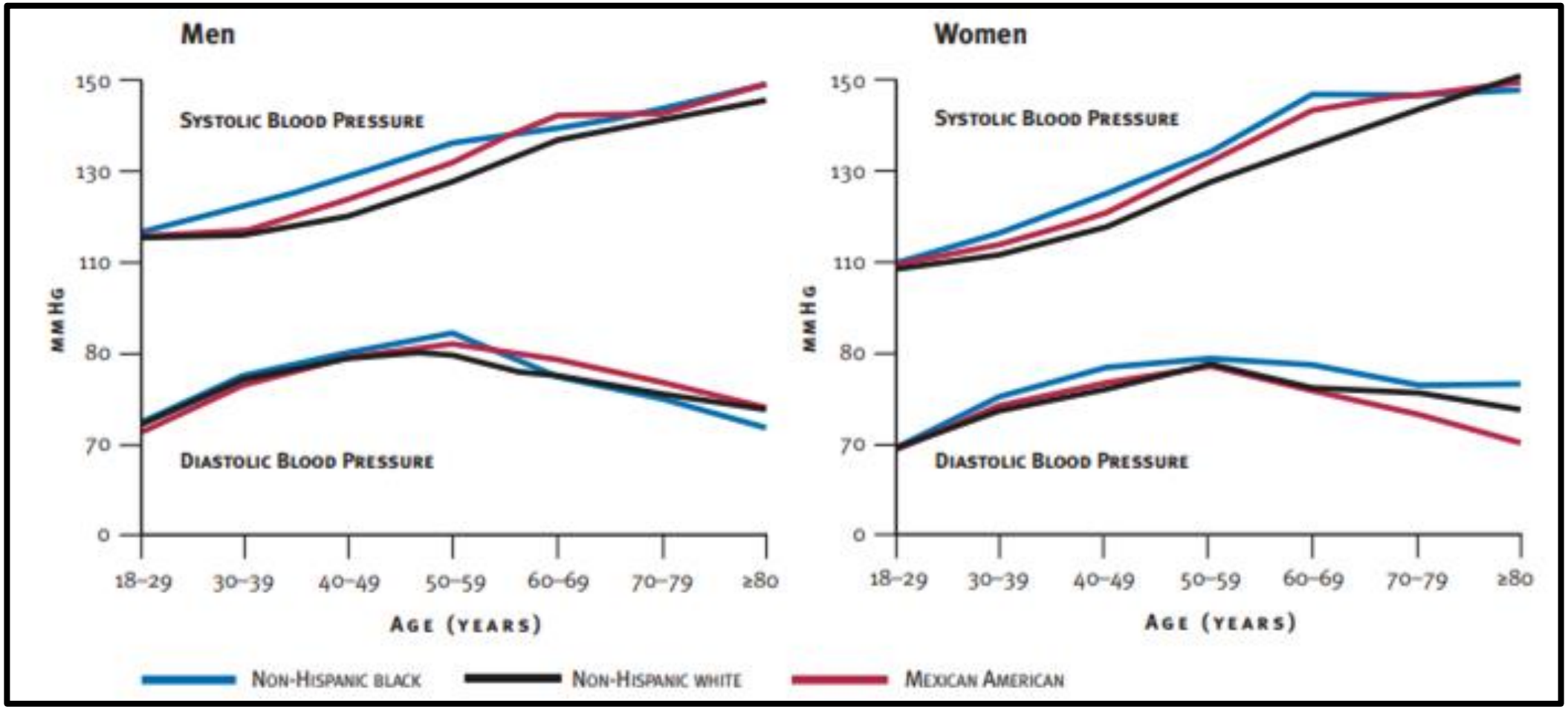
Changes in systolic and diastolic blood pressure with age



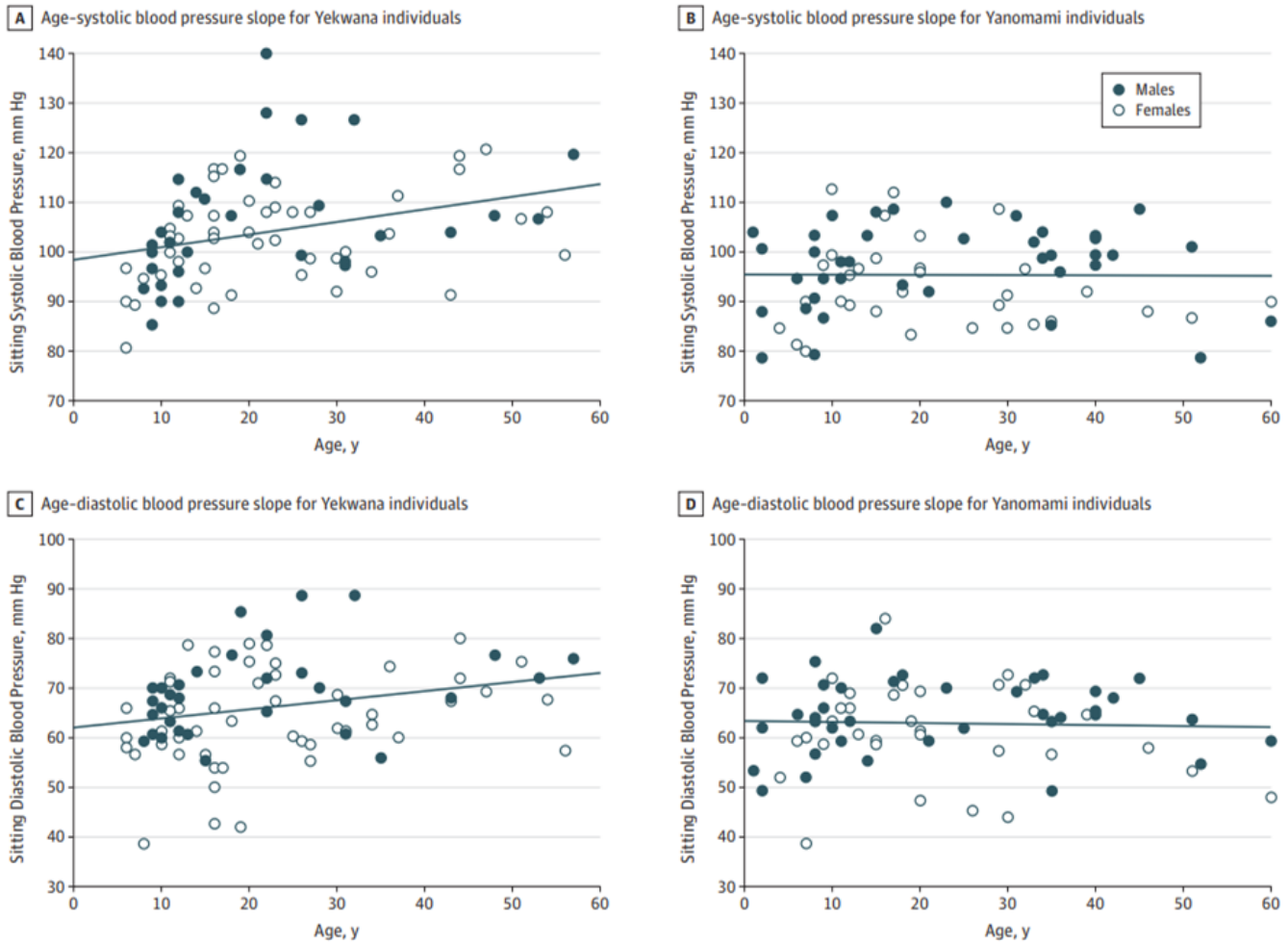
SBP and DBP by age and race or ethnicity for men and women over 18 years of age in the U.S. population. Date from NHANES III, 1988-1991.

Hypertension 1995;25(3):305–13.

Changes in systolic and diastolic blood pressure with age… are not inevitable

Changes in systolic and diastolic blood pressure with age… are not inevitable

Two Amerindian communities, the Yanomami and the Yekwana, from a remote area of the Venezuelan rainforest inaccessible by land



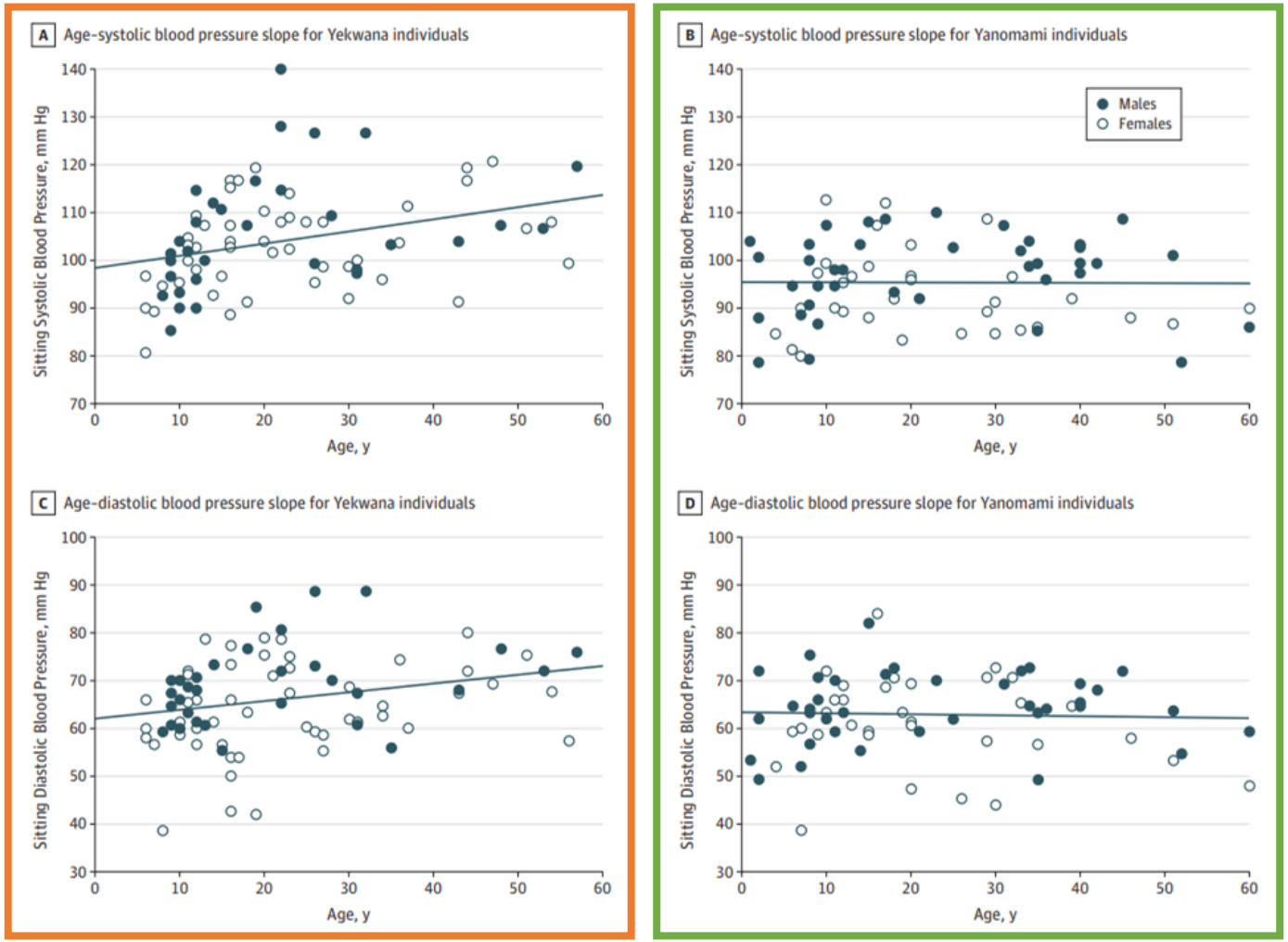
Scatter Plots of Sitting Systolic and Diastolic Blood Pressure Levels by Age in Yekwana and Yanomami Individuals Aged 1 to 60 years

JAMA Cardiol. 2018;3(12):1247-1249.

Changes in systolic and diastolic blood pressure with age… are not inevitable

Two Amerindian communities, the Yanomami and the Yekwana, from a remote area of the Venezuelan rainforest inaccessible by land

The **Yekwana** people live near the Yanomami people, but have been affected by missions and an airstrip for small-engine planes, which has allowed for delivery of medicine and aspects of Western lifestyle, including intermittent **exposure to processed foods and salt**



Yanomami community hunter-gatherer-gardeners who are among the least acculturated peoples in the world and their adults have the lowest known BP measurements

Scatter Plots of Sitting Systolic and Diastolic Blood Pressure Levels by Age in Yekwana and Yanomami Individuals Aged 1 to 60 years

JAMA Cardiol. 2018;3(12):1247-1249.

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Risk Factors for Hypertension

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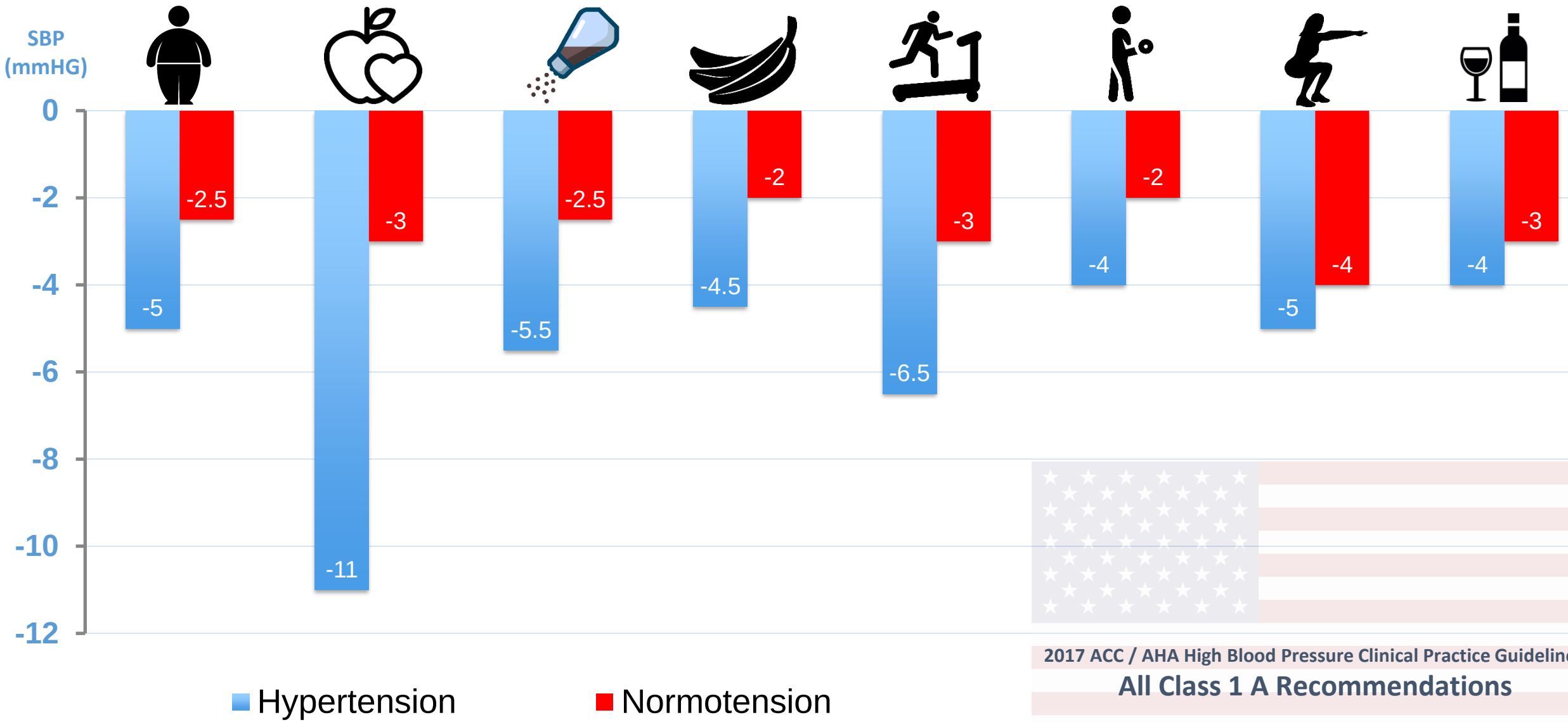
Cleveland, Ohio USA



2017 ACC / AHA High Blood Pressure Clinical Practice Guidelines

All Class 1 A Recommendations

Hypertension. 2018;71:e13–e115

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Properly Defining Hypertension

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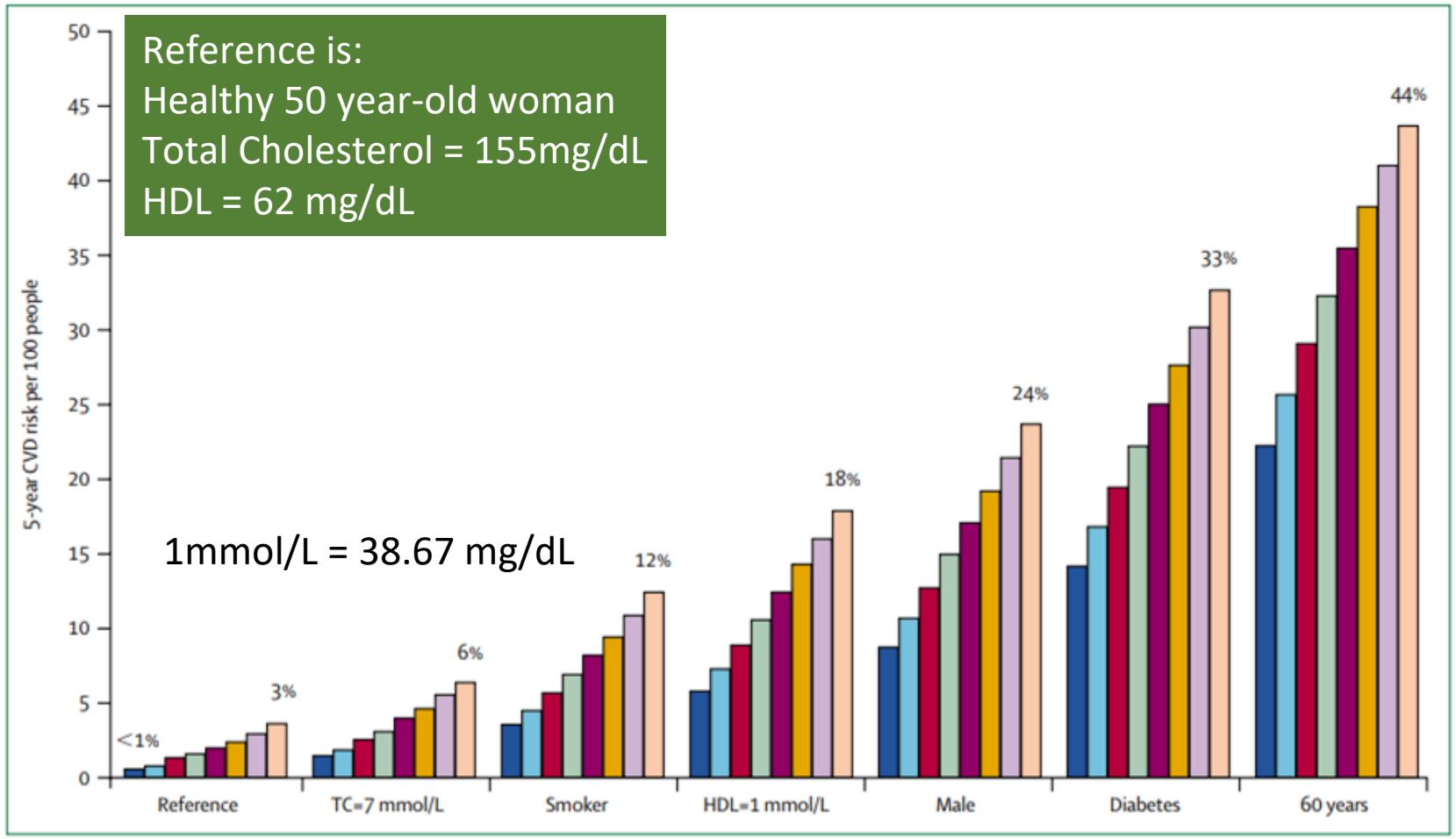
Two key points

1. Elevated blood pressure does necessarily mean someone has hypertension

2. Blood pressure is *not like pregnancy*. Blood pressure is **continuously** related to cardiovascular risk.

Absolute risk of cardiovascular disease over 5 years in patients by systolic blood pressure at specified levels of other risk factors



Risks are given for SBP levels of 110, 120, 130, 140, 150, 160, 170, and 180 mmHg. In the other categories additional risk factors are added consecutively, for example, the diabetes category is a diabetic 50-year old male cigarette smoker with a total cholesterol of 7 mmol/L.

Lancet 2005; 365: 434–41

Defining *"hypertension"* at an absolute threshold is not unimportant (particularly for epidemiological purposes), but I would urge you to think clinically about hypertension as:

"the blood pressure level above which there would be substantial (or clinically significant) benefits from lowering blood pressure"

Benefits can be short term, or long-term.

Similar logic has been applied to defining hyperlipidemia, particularly in more recent American lipid guidelines

Lancet 2005; 365: 434–41

CURRENT DEFINITIONS

Types of Hypertension

Primary	Previously known as essential hypertension (*multifactorial*)
Secondary	Discrete cause apart from primary hypertension (*not necessarily mutually exclusive from primary hypertension*)
Resistant	BP that is elevated despite patient prescribed 3 or more medications, including a diuretic appropriately dosed for kidney function. Also includes *controlled resistant hypertension*; 4 or more medications and controlled
Refractory	BP that is elevated despite patient prescribed ≥ 5 BP medications, which include chlorthalidone and spironolactone
Isolated Systolic Hypertension (ISH)	Elevated systolic BP with normal or low diastolic BP

Most Recent American Heart Association / American College of Cardiology Blood Pressure Guidelines (2017)

SBP (mm Hg)		DBP (mm Hg)	Categories of BP (in Adults)
<120	and	<80	Normal BP
120-129	and	<80	Elevated BP
130-139	or	80-89	Stage 1 Hypertension
140-159	or	90-99	Stage 2 Hypertension
≥160	or	≥100	Stage 2 Hypertension

J Am Coll Cardiol. Sep 2017, 23976

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≥160	or	≥100	Stage 2 Hypertension

These are OFFICE Blood Pressure

J Am Coll Cardiol. Sep 2017, 23976

Corresponding Values of SBP/DBP for Clinic, HBPM, Daytime, Nighttime, and 24-Hour ABPM Measurements

Clinic	HBPM	Daytime ABPM	Nighttime ABPM	24-Hour ABPM
120/80	120/80	120/80	100/65	115/75
130/80	130/80	130/80	110/65	125/75
140/90	135/85	135/85	120/70	130/80
160/100	145/90	145/90	140/85	145/90

ABPM indicates ambulatory blood pressure monitoring; BP, blood pressure; DBP diastolic blood pressure; HBPM, home blood pressure monitoring; and SBP, systolic blood pressure.

J Am Coll Cardiol. Sep 2017, 23976

European Society of Cardiology / European Society of Hypertension Guidelines (2018)

SBP (mm Hg)		DBP (mm Hg)	Categories of BP (in Adults)
<120	and	<80	Optimal
120-129	and/or	80-84	Normal
130-139	and/or	85-89	High Normal
140-159	and/or	90-99	Grade 1 Hypertension
160-179	and/or	100-109	Grade 2 Hypertension
≥ 180	and/or	≥ 110	Grade 3 Hypertension
≥ 140	and	< 90	Isolated Systolic Hypertension

European Heart Journal (2018) 00, 1–98

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Blood Pressure Measurement

Luke J. Laffin, MD FACC
Co-Director, Center for Blood Pressure Disorders
Medical Director of Cardiac Rehabilitation
Cleveland Clinic
Cleveland, Ohio USA

What prompted blood pressure guideline changes?

What prompted blood pressure guideline changes?

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 NOVEMBER 26, 2015 VOL. 373 NO. 22

A Randomized Trial of Intensive versus Standard Blood-Pressure Control

The SPRINT Research Group*

N Engl J Med 2015; 373:2103-2116

What prompted blood pressure guideline changes?

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 NOVEMBER 26, 2015 VOL. 373 NO. 22

Journal article provided to students

A Randomized Trial of Intensive versus Standard Blood-Pressure Control

The SPRINT Research Group*

N Engl J Med 2015; 373:2103-2116

Basics of SPRINT

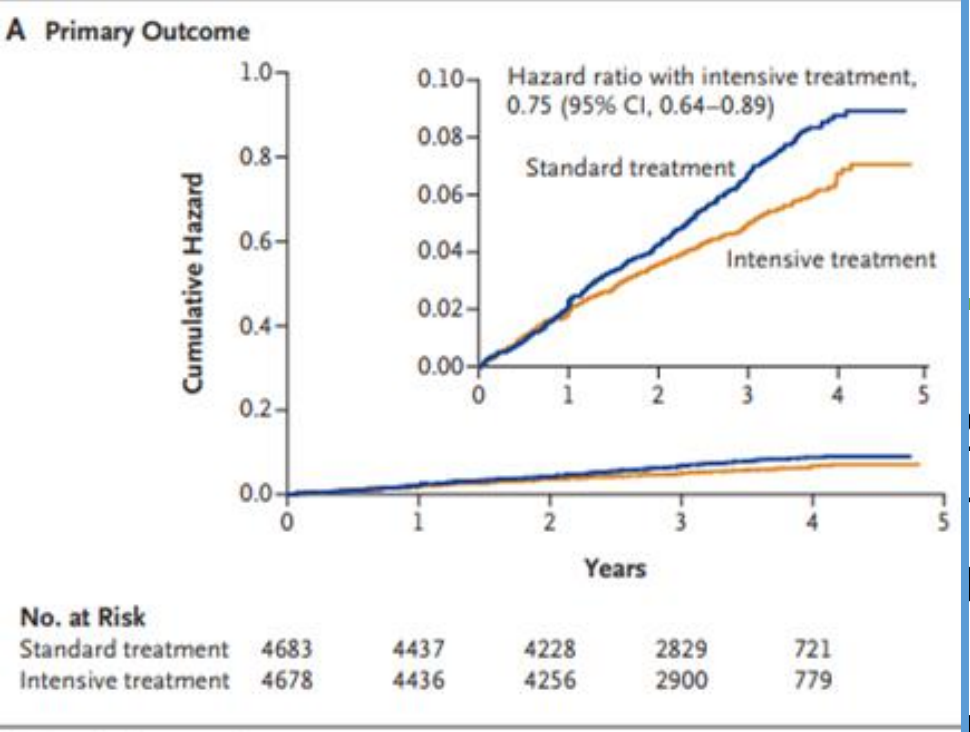
- 9361 participants **without** type 2 diabetes mellitus
- Participants needed to be at high cardiovascular risk or have existing CV disease
- Randomized to a BP goal of <120 mmHg systolic **versus 135-140 mmHg systolic**

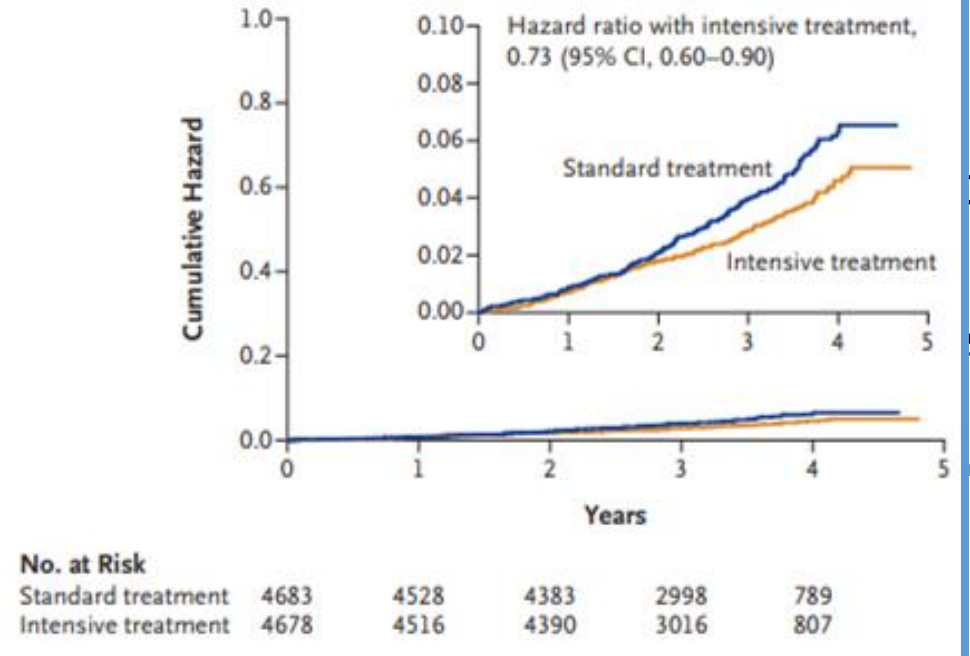
- Intensive control group achieved 121 mmHg systolic
- Standard control group achieved 136 mmHg systolic

- Trial stopped early 3.26 years after initiation due to benefit of intensive BP control

- Primary outcome was composite of nonfatal MI, nonfatal stroke, **heart failure,** or death from CV cause

N Engl J Med 2015; 373:2103-2116





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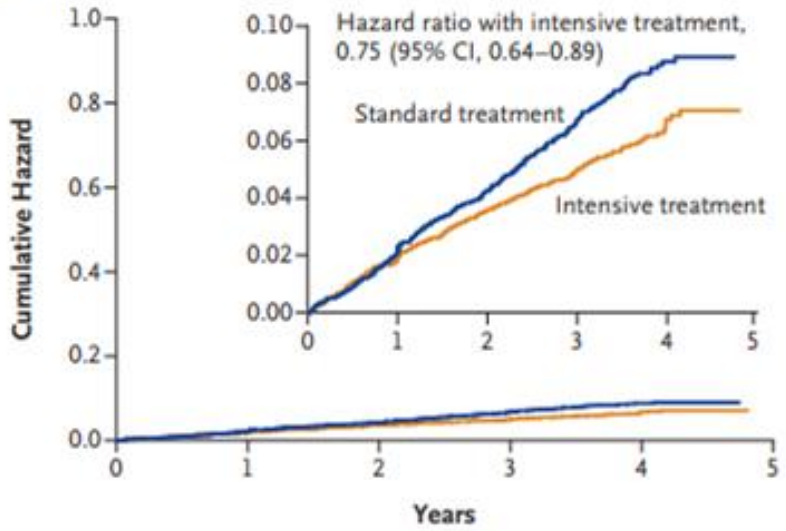
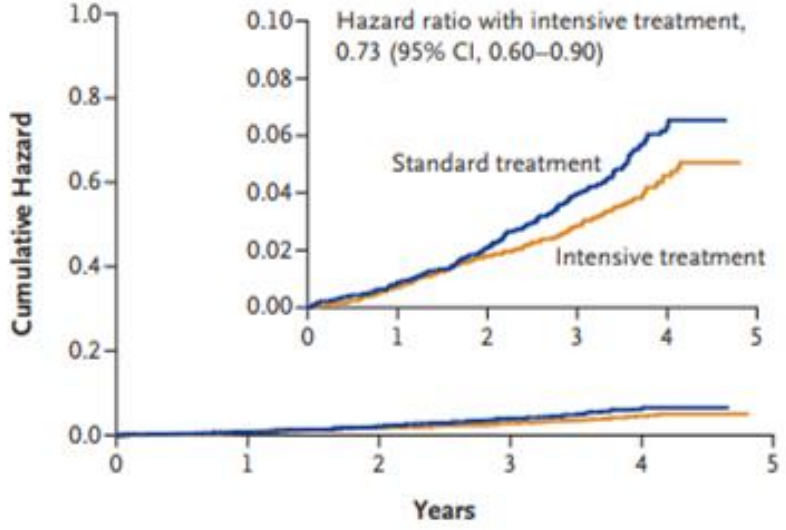
A Primary Outcome



No. at Risk

Standard treatment	4683	4437	4228	2829	721
Intensive treatment	4678	4436	4256	2900	779

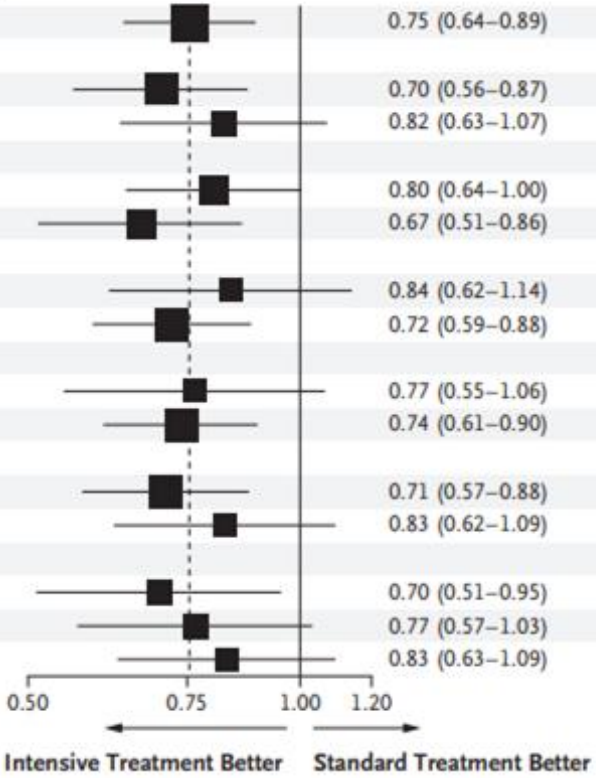
B Death from Any Cause



No. at Risk

Standard treatment	4683	4528	4383	2998	789
Intensive treatment	4678	4516	4390	3016	807

Subgroup	Intensive Treatment	Standard Treatment	Hazard Ratio (95% CI)	P Value for Interaction
	no. of patients with primary outcome/total no. (%)			
Overall	243/4678 (5.2)	319/4683 (6.8)	0.75 (0.64–0.89)	
Previous CKD				0.36
No	135/3348 (4.0)	193/3367 (5.7)	0.70 (0.56–0.87)	
Yes	108/1330 (8.1)	126/1316 (9.6)	0.82 (0.63–1.07)	
Age				0.32
<75 yr	142/3361 (4.2)	175/3364 (5.2)	0.80 (0.64–1.00)	
≥75 yr	101/1317 (7.7)	144/1319 (10.9)	0.67 (0.51–0.86)	
Sex				0.45
Female	77/1684 (4.6)	89/1648 (5.4)	0.84 (0.62–1.14)	
Male	166/2994 (5.5)	230/3035 (7.6)	0.72 (0.59–0.88)	
Race				0.83
Black	62/1454 (4.3)	85/1493 (5.7)	0.77 (0.55–1.06)	
Nonblack	181/3224 (5.6)	234/3190 (7.3)	0.74 (0.61–0.90)	
Previous cardiovascular disease				0.39
No	149/3738 (4.0)	208/3746 (5.6)	0.71 (0.57–0.88)	
Yes	94/940 (10.0)	111/937 (11.8)	0.83 (0.62–1.09)	
Systolic blood pressure				0.77
≤132 mm Hg	71/1583 (4.5)	98/1553 (6.3)	0.70 (0.51–0.95)	
>132 to <145 mm Hg	77/1489 (5.2)	106/1549 (6.8)	0.77 (0.57–1.03)	
≥145 mm Hg	95/1606 (5.9)	115/1581 (7.3)	0.83 (0.63–1.09)	



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Strengths of SPRINT

- Almost double the size of other prominent BP target trial of the decade (ACCORD-BP), i.e. adequately powered
- Included heart failure in the primary end-point
- Effects of more intensive blood pressure control seen across all subgroups
- Enrolled a high risk population

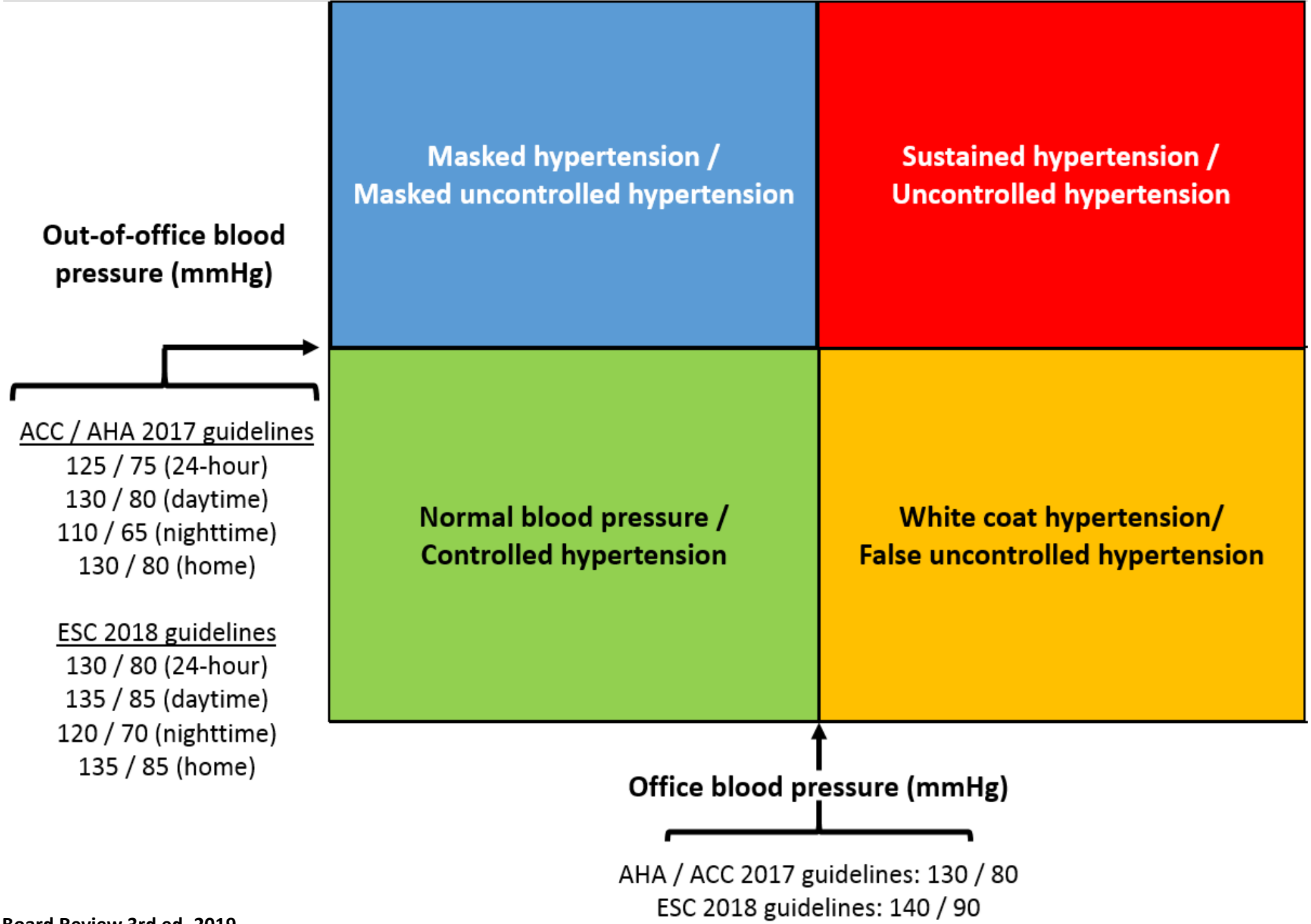
Weaknesses of SPRINT

- Method of blood pressure measurement (AOBP)
- Did not include post-stroke patients (or pts with diabetes, intentional)
- May not have reflected typical clinical practice by withdrawing medications to meet the 135-140 mmHg target

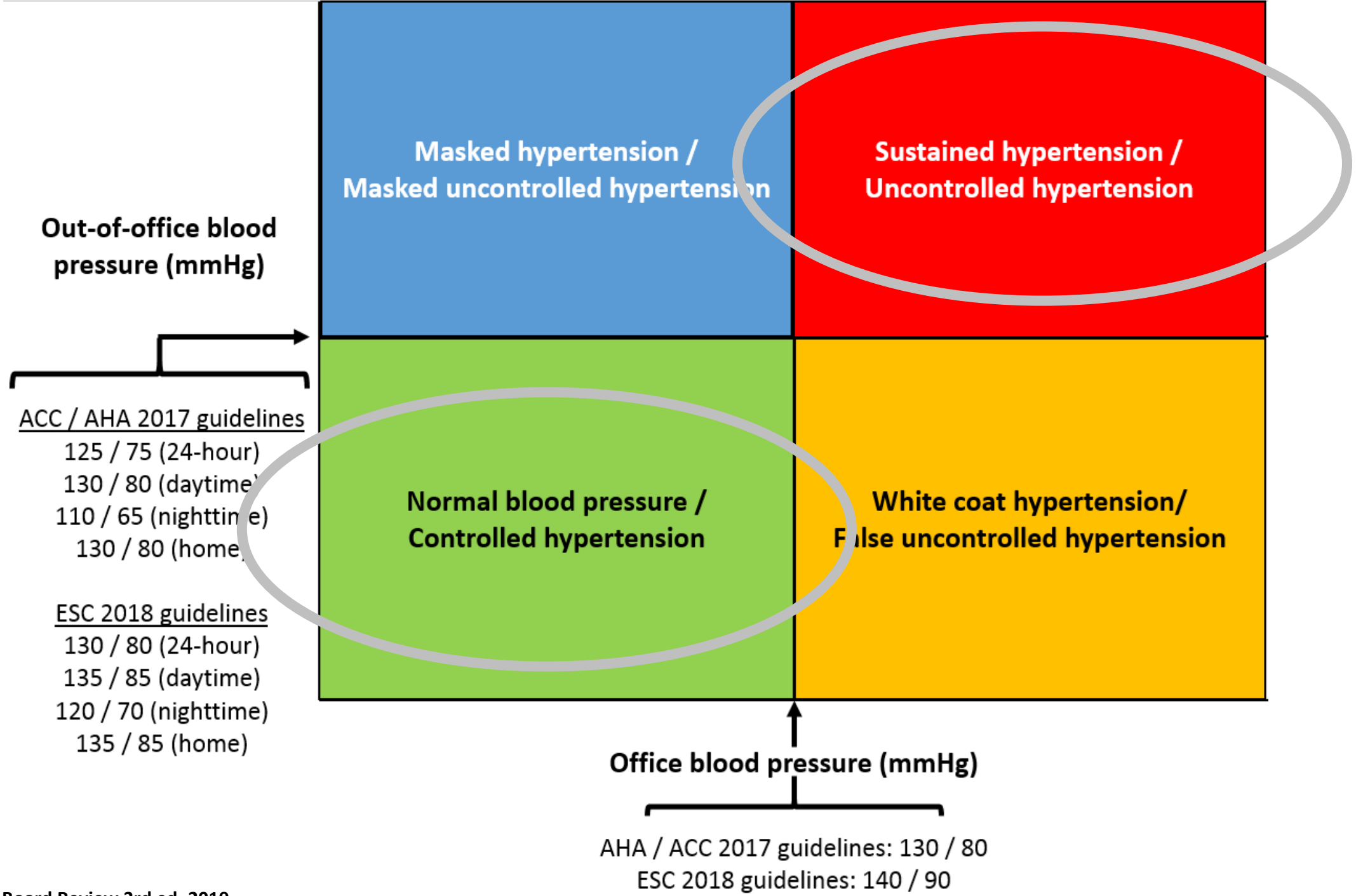
Office blood pressure measurement in 2021 is not good enough

COR	LOE	Recommendation for Out-of-Office and Self-Monitoring of BP
I	A^{SR}	Out-of-office BP measurements are recommended to confirm the diagnosis of hypertension and for titration of BP-lowering medication, in conjunction with telehealth counseling or clinical interventions.

J Am Coll Cardiol. Sep 2017, 23976



Laffin and Cho. CCF Board Review 3rd ed. 2019



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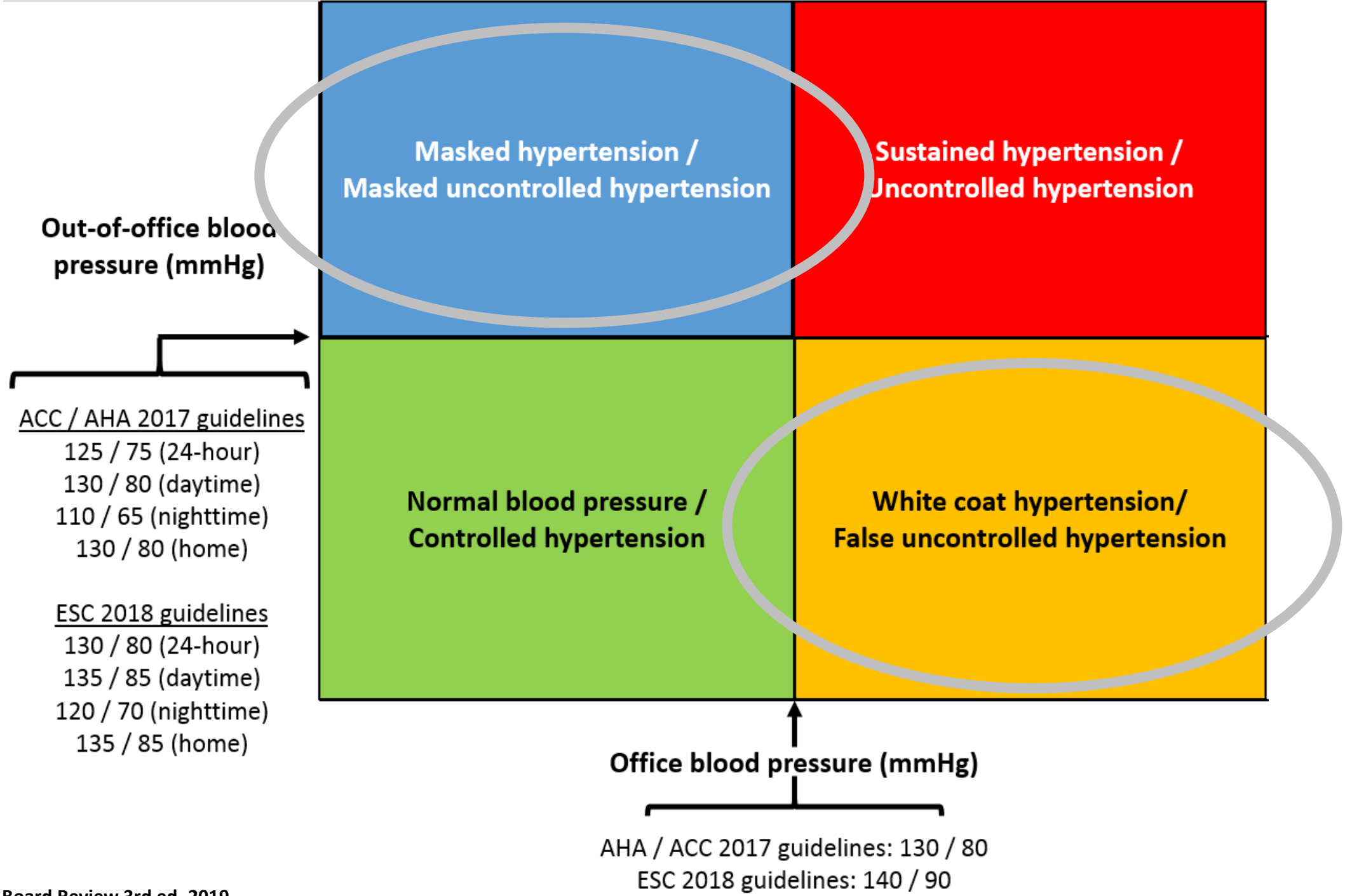

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Out-of-office blood pressure (mmHg)

ACC / AHA 2017 guidelines
125 / 75 (24-hour)
130 / 80 (daytime)
110 / 65 (nighttime)
130 / 80 (home)

ESC 2018 guidelines
130 / 80 (24-hour)
135 / 85 (daytime)
120 / 70 (nighttime)
135 / 85 (home)

Masked
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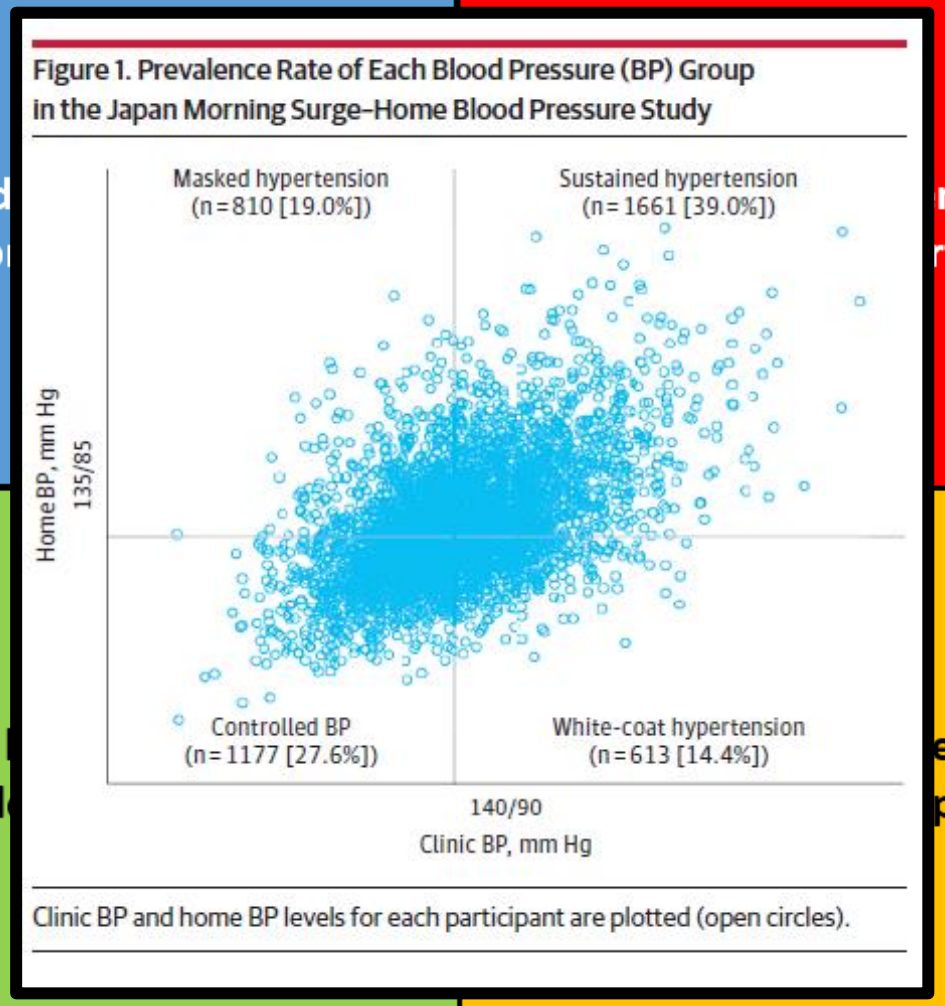


Figure 1. Prevalence Rate of Each Blood Pressure (BP) Group in the Japan Morning Surge–Home Blood Pressure Study

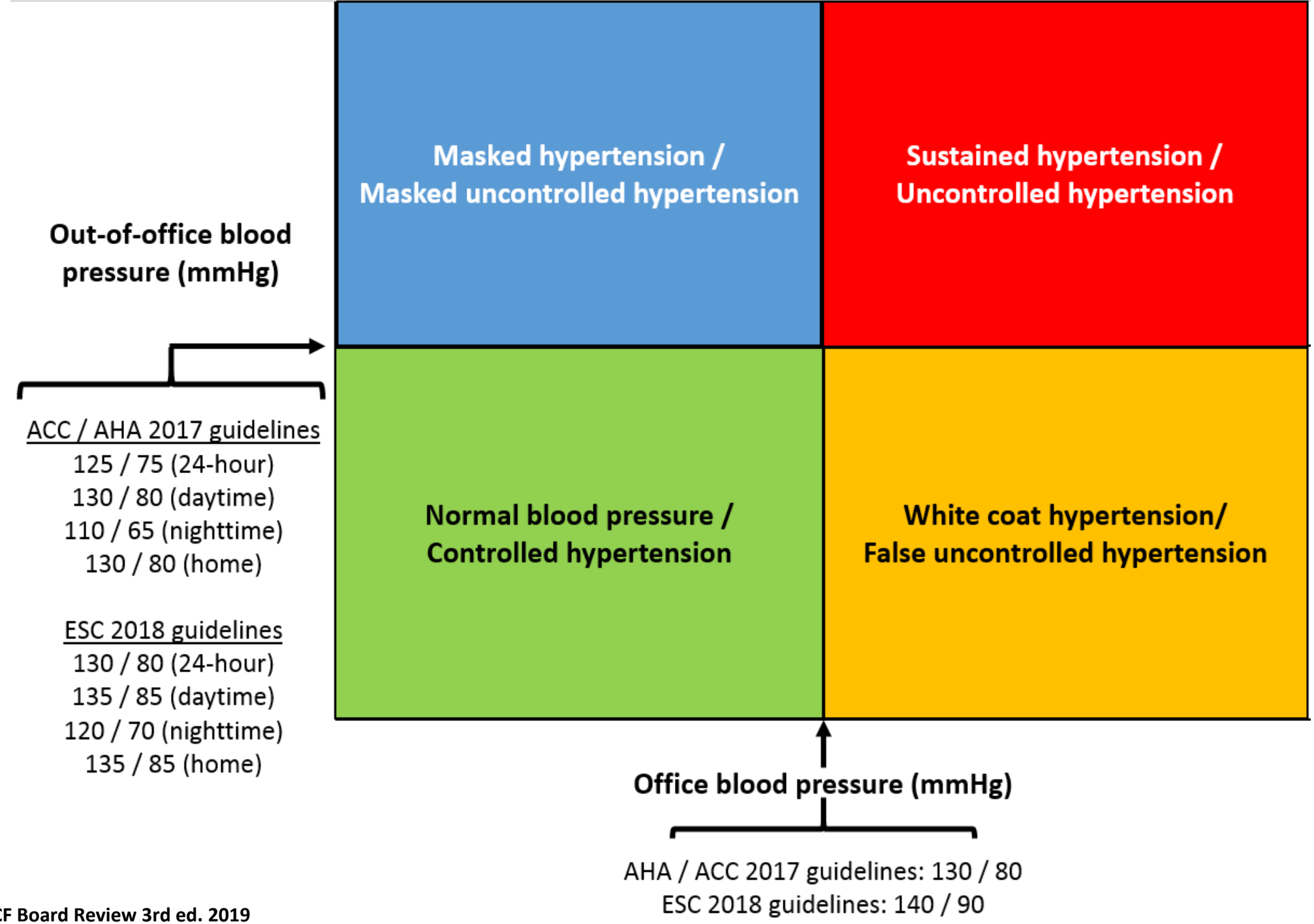
Clinic BP and home BP levels for each participant are plotted (open circles).

Office blood pressure (mmHg)

AHA / ACC 2017 guidelines: 130 / 80
ESC 2018 guidelines: 140 / 90

JAMA Cardiol. 2018;3(7):583-590.

Laffin and Cho. CCF Board Review 3rd ed. 2019



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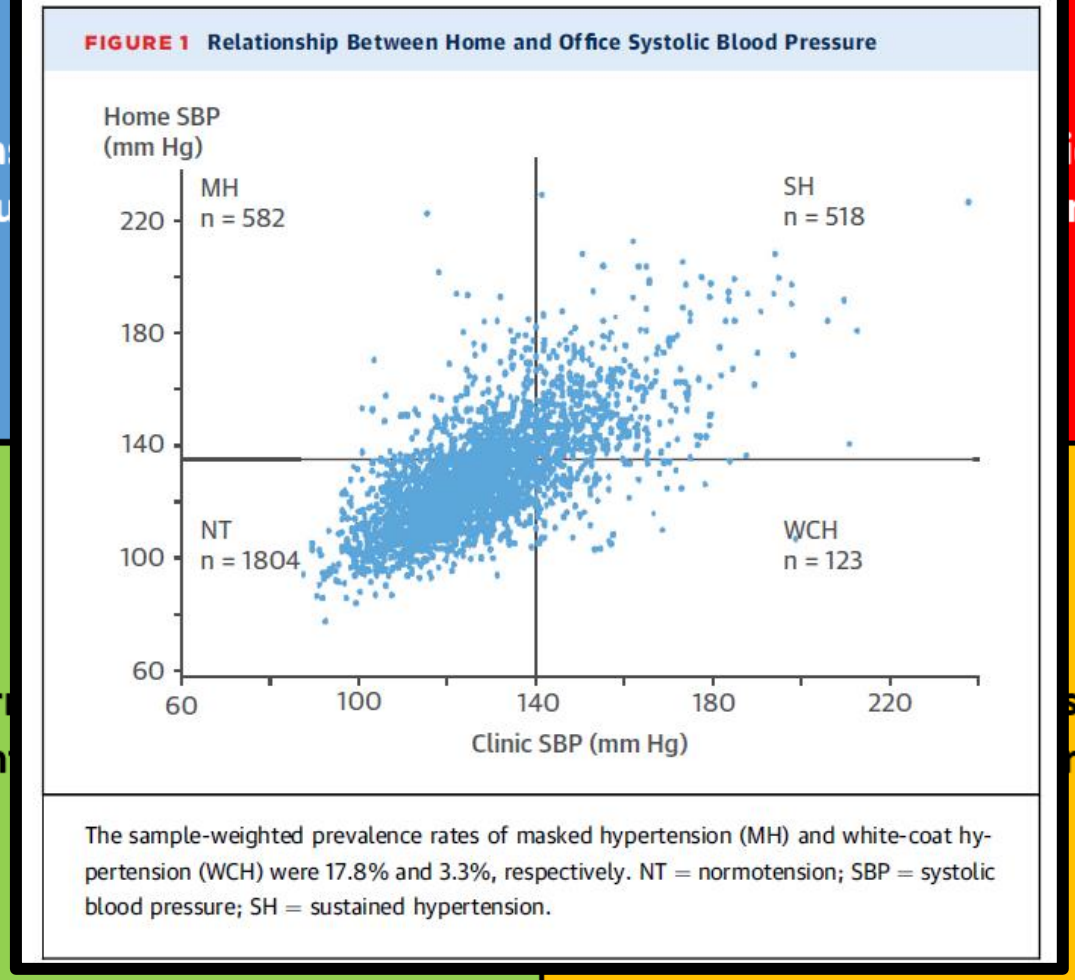

FIGURE 1 Relationship Between Home and Office Systolic Blood Pressure

The sample-weighted prevalence rates of masked hypertension (MH) and white-coat hypertension (WCH) were 17.8% and 3.3%, respectively. NT = normotension; SBP = systolic blood pressure; SH = sustained hypertension.

Office blood pressure (mmHg)

AHA / ACC 2017 guidelines: 130 / 80
ESC 2018 guidelines: 140 / 90

J Am Coll Cardiol 2015;66:2159–69

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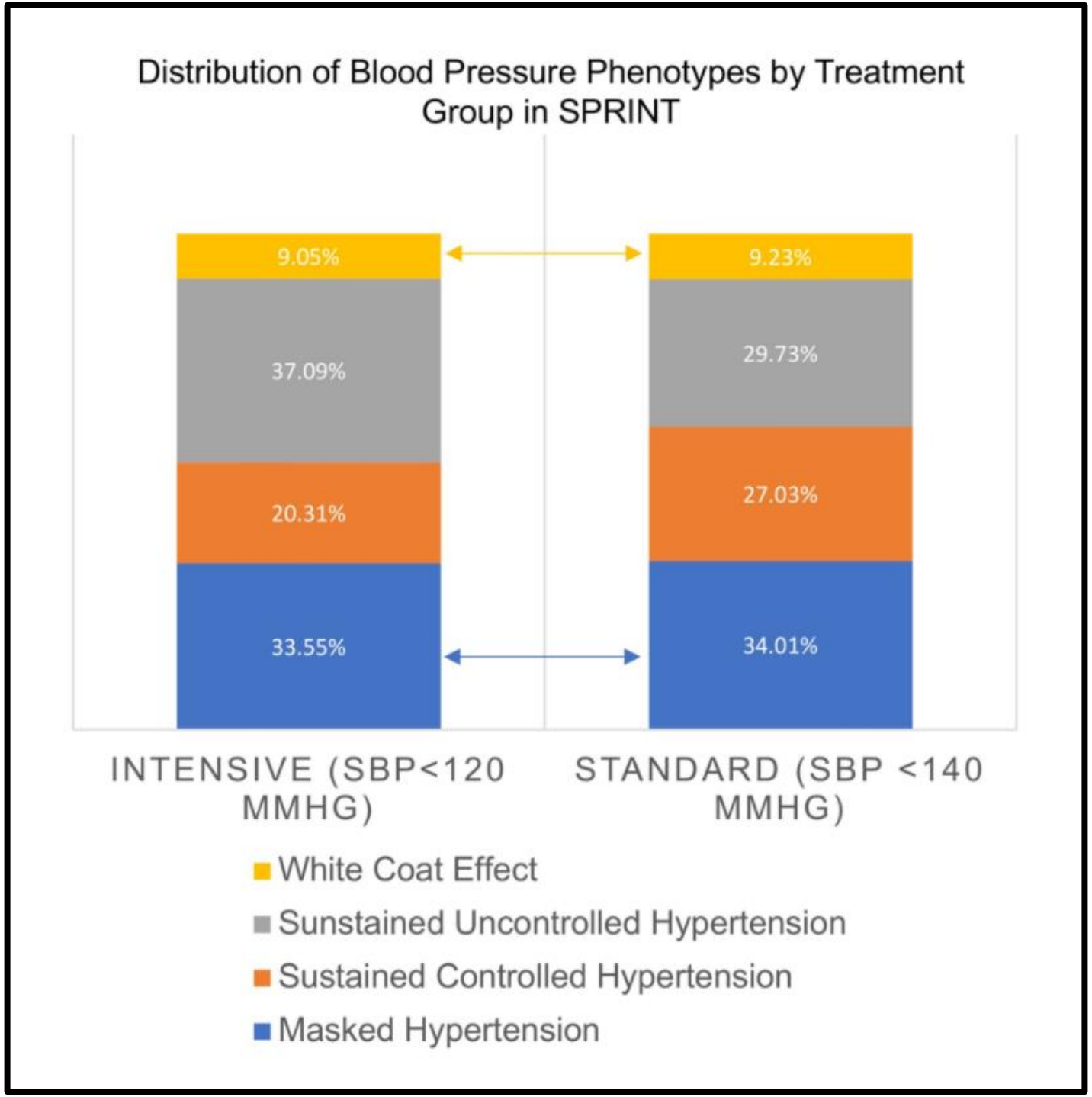

Hypertension. 2020;76:1090-1096

CMHC
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Foundations of Cardiometabolic Health Certification Course

Certified Cardiometabolic Health Professional (CCHP)

Diagnosis of Hypertension (Primary and Secondary)

Luke J. Laffin, MD FACC

Co-Director, Center for Blood Pressure Disorders

Medical Director of Cardiac Rehabilitation

Cleveland Clinic

Cleveland, Ohio USA

Masked and white coat hypertension are not benign phenomena

JAMA Cardiology | Original Investigation

Association of Cardiovascular Outcomes With Masked Hypertension Defined by Home Blood Pressure Monitoring in a Japanese General Practice Population

Takeshi Fujiwara, MD, PhD; Yuichiro Yano, MD, PhD; Satoshi Hoshide, MD, PhD; Hiroshi Kanegae, BSc; Kazuomi Kario, MD, PhD

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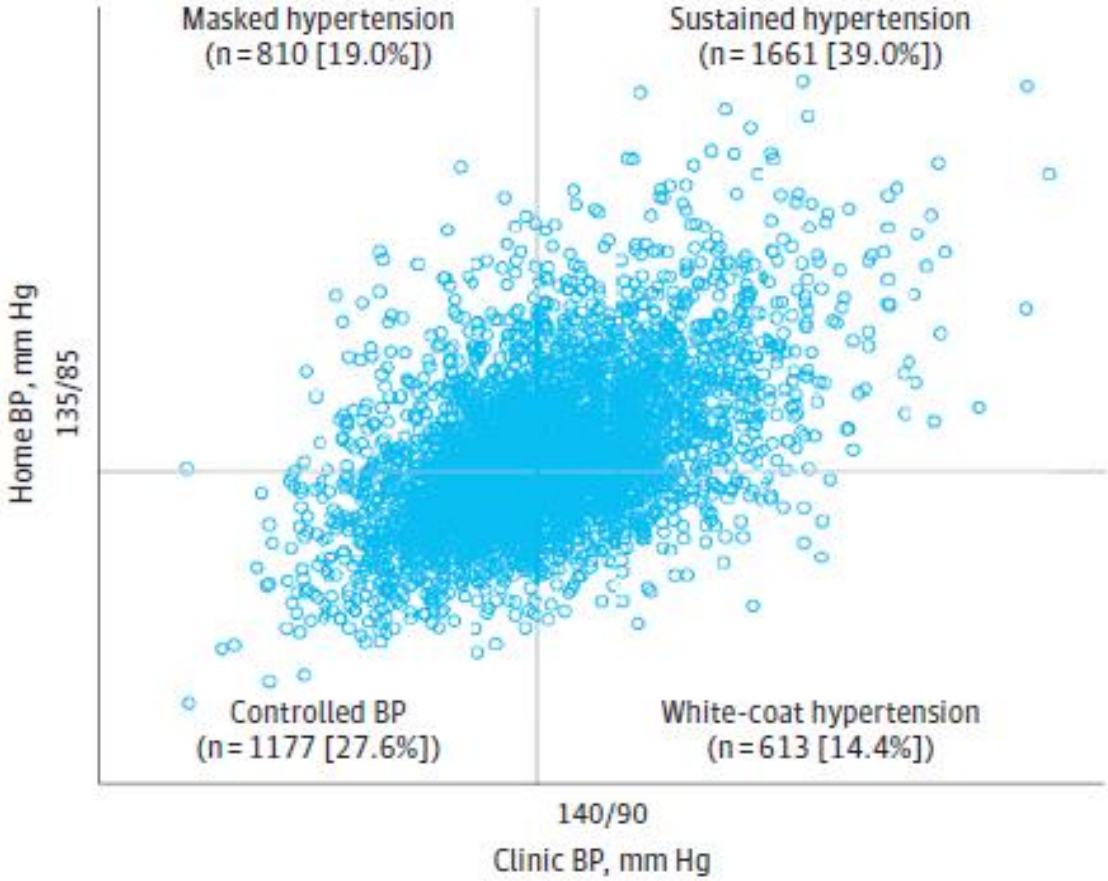

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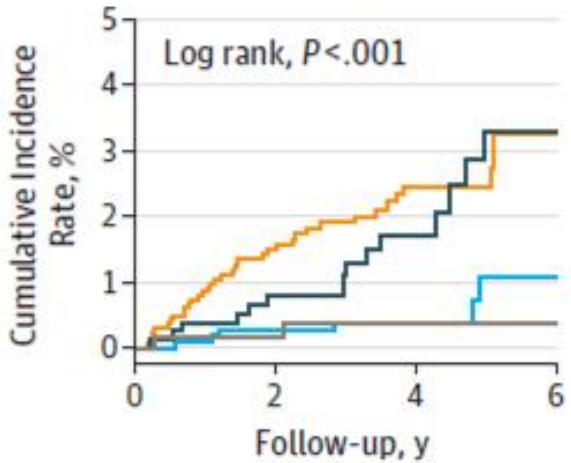
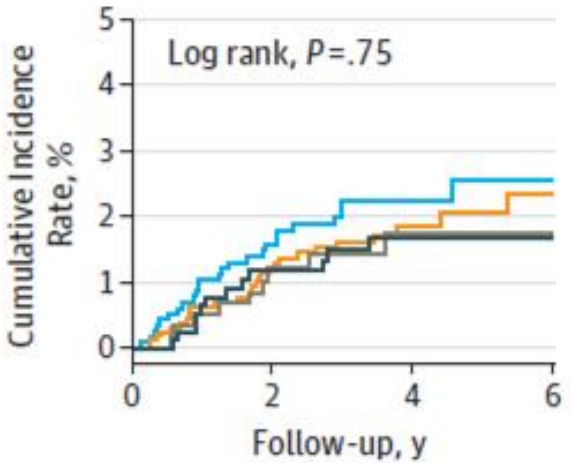
Four Blood Pressure Groups and Cardiovascular Disease Events

A Stroke events



No. at risk				
Sustained hypertension	1661	1373	720	313
Masked hypertension	810	681	394	187
White-coat hypertension	613	516	255	99
Controlled BP	1177	1005	515	231

B CHD events



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Kaplan-Meier curves of the cumulative incidence of stroke (A) or coronary heart disease (CHD) (B) by the 4 BP groups are shown.

An increased stroke risk may be associated with masked hypertension defined by HBPM in a general practice population.

JAMA Cardiol. 2018;3(7):583-590.

Annals of Internal Medicine

REVIEW

Cardiovascular Events and Mortality in White Coat Hypertension

A Systematic Review and Meta-analysis

Jordana B. Cohen, MD, MSCE; Michael J. Lotito; Usha K. Trivedi, BS; Matthew G. Denker, MD, MSCE; Debbie L. Cohen, MD; and Raymond R. Townsend, MD

Ann Intern Med. 2019;170:853-862

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Figure 2. Cardiovascular event risk in WCH and WCE.

Study, Year (Reference)	Total Participants, *n*	HR (95% CI)
Verdecchia et al, 1994 (23)	1392	1.17 (0.25–5.33)
Fagard et al, 2005 (24)	359	1.00 (0.35–2.90)
Pierdomenico et al, 2008 (25)	2037	0.97 (0.38–2.46)
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Asayama et al, 2014 (28)	8237	1.20 (0.93–1.54)
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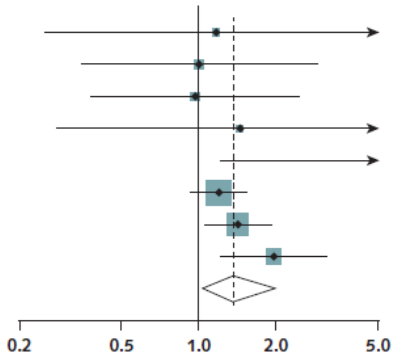
Overall (I^2 = 0.0%; P = 0.379)		1.36 (1.03–2.00)



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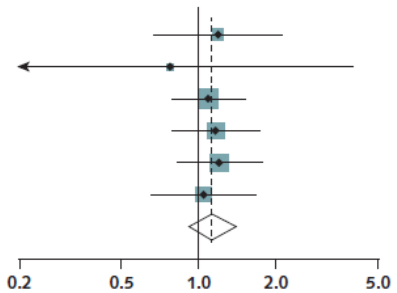
Overall (I^2 = 0.0%; P = 0.992)		1.12 (0.91–1.39)



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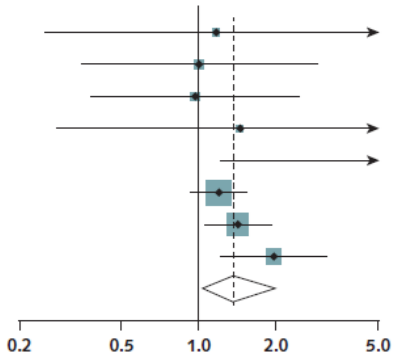

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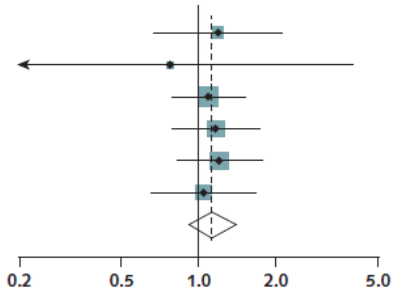


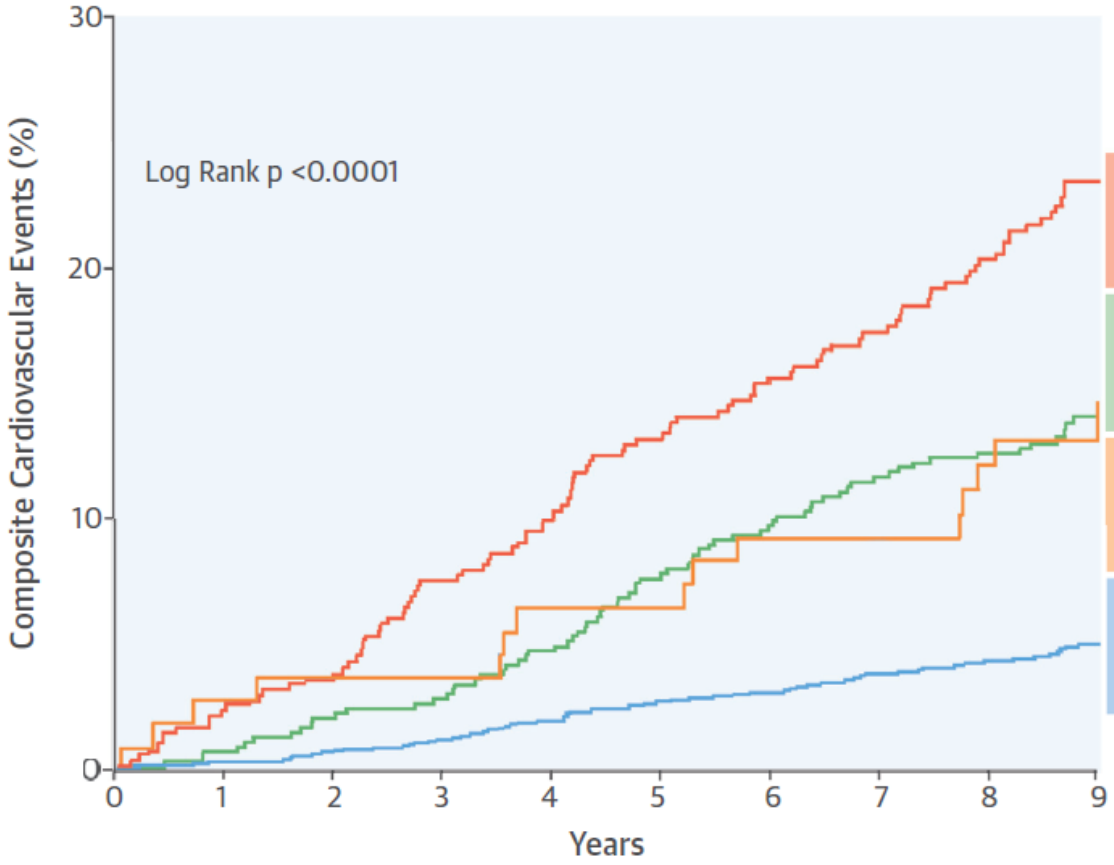
Untreated WCH, **but not treated WCE**, is associated with an increased risk for cardiovascular events and all cause mortality.

Out-of-office BP monitoring is critical in the diagnosis and management of hypertension.

Untreated patients with isolated office hypertension should be monitored closely for transition to sustained hypertension

Ann Intern Med. 2019;170:853-862

CENTRAL ILLUSTRATION **Composite Cardiovascular Events Associated With Masked Hypertension and White-Coat Hypertension**



	Home BP	Office BP
Sustained HTN	↑	↑
Masked HTN	↑	Normal
White Coat HTN	Normal	↑
Normotension	Normal	Normal

Tientcheu, D. et al. J Am Coll Cardiol. 2015; 66(20):2159-69.

Kaplan-Meier curves for the cumulative incidence of composite cardiovascular events among the normotension, white-coat hypertension, masked hypertension, and sustained hypertension groups. BP = blood pressure; HTN = hypertension.

J Am Coll Cardiol 2015;66:2159–69

Office blood pressure measurement in 2021 is not good enough

COR	LOE	Recommendation for Out-of-Office and Self-Monitoring of BP
I	A^{SR}	Out-of-office BP measurements are recommended to confirm the diagnosis of hypertension and for titration of BP-lowering medication, in conjunction with telehealth counseling or clinical interventions.

J Am Coll Cardiol. Sep 2017, 23976

	AMBULATORY BLOOD PRESSURE MONITORING (ABPM)	SELF BLOOD PRESSURE MONITORING
What is it?	Wearable device that records BP over time. Provides a comprehensive record of BP through static/active.	Multiple readings taken with an automatic BP machine by the patient themselves
How is it performed?	BP measured during routine activities. 48-72 readings taken during a 24-hour period.	BP measured at home while seated, resting quietly w/ back supported and feet on the floor
Advantages	**Able to detect white Coat & masked hypertension** **Stronger prognostic evidence of CV outcomes compared to OBP** Extensive info on short-to-long-term BP profile as well as circadian BP variation Abundant info from single measurement session	**Able to detect white coat hypertension +/- masked** Inexpensive & widely available More relaxing setting for patient Easy to repeat to assess day-to-day BP variability
Disadvantages	Expensive and potential limited availability (reimbursement) Possible patient discomfort in wearing	Need motivation from patient to do it Proper technique it needed Lack of nocturnal readings

Comparison of the features of office, ambulatory and home BP measurement modalities

Feature	Office	Ambulatory	Home
Detection of white-coat hypertension	-	++	++
Detection of masked hypertension	-	++	++
Assessment of nighttime BP levels and dip	-	++	+
Assessment of early-morning BP surge	-	++	-
Assessment of morning hypertension	+/-	++	++
Assessment of antihypertensive drug action	+	++	++
Assessment of duration of drug action	+/-	++	+
Long-term follow-up of hypertension	++	+/-	++
Improvement of patients' adherence	+	-	++
Improvement of hypertension control rate	+	-	++
Reproducibility	-	++	++
Prognostic value	+	++	++
Availability	++	-	++
Cost	-	-	++

Adapted from Stergiou at al. J Clin Hypertens (Greenwhich). 2018; 20:116-1121

Feature	Office	Ambulatory	Home
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Assessment of morning hypertension	+/-	++	++
Assessment of antihypertensive drug action	+	++	++
Assessment of duration of drug action	+/-	++	+
Long-term follow-up of hypertension	++	+/-	++
Improvement of patients' adherence	+	-	++
Improvement of hypertension control rate	+	-	++
Reproducibility	-	++	++
Prognostic value	+	++	++
Availability	++	-	++
Cost	-	-	++

Comparison of the features of office, ambulatory and home BP measurement modalities

OFFICE

Adapted from Stergiou at al. J Clin Hypertens (Greenwhich). 2018; 20:116-1121

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Assessment of antihypertensive drug action	+	++	++
Assessment of duration of drug action	+/-	++	+
Long-term follow-up of hypertension	++	+/-	++
Improvement of patients' adherence	+	-	++
Improvement of hypertension control rate	+	-	++
Reproducibility	-	++	++
Prognostic value	+	++	++
Availability	++	-	++
Cost	-	-	++

Comparison of the features of office, ambulatory and home BP measurement modalities

ABPM

Adapted from Stergiou at al. J Clin Hypertens (Greenwhich). 2018; 20:116-1121

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Assessment of morning hypertension	+/-	++	++
Assessment of antihypertensive drug action	+	++	++
Assessment of duration of drug action	+/-	++	+
Long-term follow-up of hypertension	++	+/-	++
Improvement of patients' adherence	+	-	++
Improvement of hypertension control rate	+	-	++
Reproducibility	-	++	++
Prognostic value	+	++	++
Availability	++	-	++
Cost	-	-	++

Comparison of the features of office, ambulatory and home BP measurement modalities

HOME

Adapted from Stergiou at al. J Clin Hypertens (Greenwhich). 2018; 20:116-1121

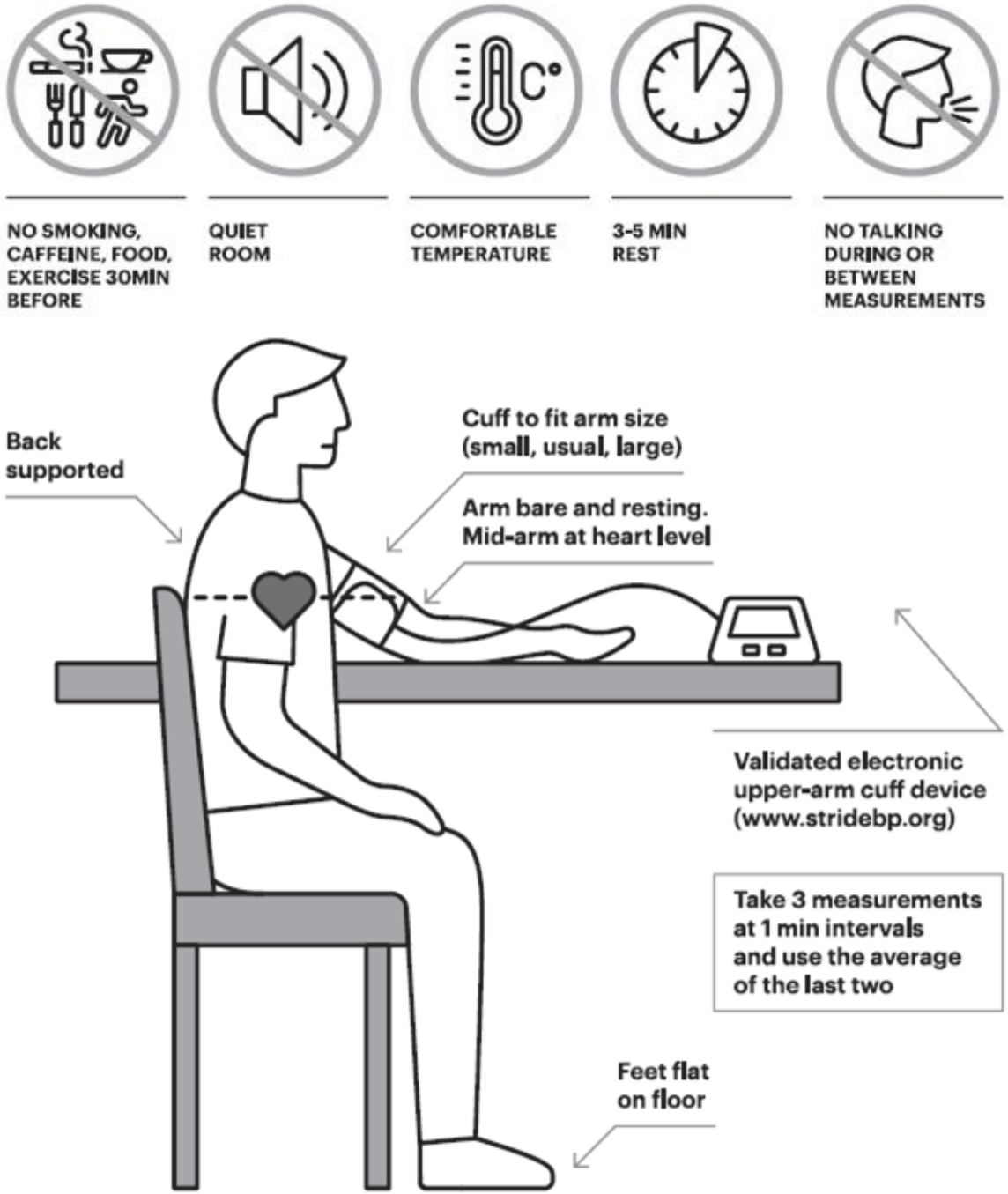
Medicare National Coverage Determination (NCD) – July 2019

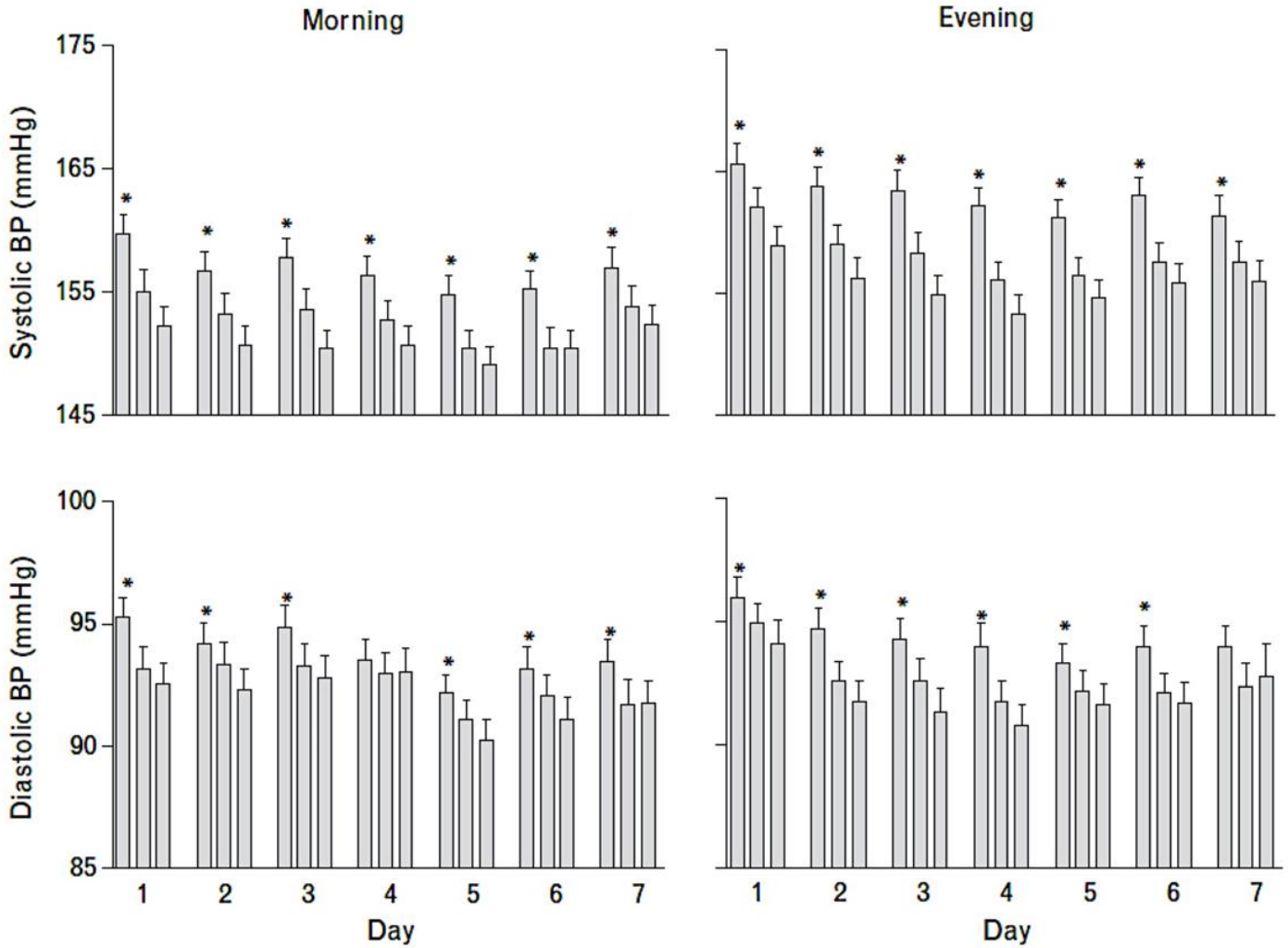
The Centers for Medicare & Medicaid Services (CMS) has determined that the **evidence is sufficient to cover ambulatory blood pressure monitoring (ABPM) for the diagnosis of hypertension in Medicare beneficiaries under the following circumstances:**

1) For beneficiaries with **suspected white coat hypertension**, which is defined as an average office blood pressure of systolic blood pressure greater than 130 mm Hg but less than 160 mm Hg or diastolic blood pressure greater than 80 mm Hg but less than 100 mm Hg on two separate clinic/office visits with at least two separate measurements made at each visit and with at least two blood pressure measurements taken outside the office which are <130/80 mm Hg.

2) For beneficiaries with **suspected masked hypertension**, which is defined as average office blood pressure between 120 mm Hg and 129 mm Hg for systolic blood pressure or between 75 mm Hg and 79 mm Hg for diastolic blood pressure on two separate clinic/office visits with at least two separate measurements made at each visit and with at least two blood pressure measurements taken outside the office which are ≥130/80 mm Hg.

For eligible patients, ABPM is covered once per year.


NO SMOKING, CAFFEINE, FOOD, EXERCISE 30MIN BEFORE
QUIET ROOM
COMFORTABLE TEMPERATURE
3-5 MIN REST
NO TALKING DURING OR BETWEEN MEASUREMENTS
Back supported
Cuff to fit arm size (small, usual, large)
Arm bare and resting. Mid-arm at heart level
Validated electronic upper-arm cuff device (www.stridebp.org)
Take 3 measurements at 1 min intervals and use the average of the last two
Feet flat on floor



Triplicate morning and evening home blood peessure (BP) measurements assessed during a 7-day period in patients without hypertensive treatment. Results are expressed as means +/- SEM; * indicates that measurements are significantly higher than subsequent ones (P <0.001).

Journal of Hypertension 2006, 24:1541–1548

Do we have validated tools patients can use?

AHA POLICY STATEMENT

Self-Measured Blood Pressure Monitoring at Home

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

Upper arm self-measured BP monitoring devices are preferred over wrist devices.

"Some wrist devices have been validated, but most guidelines and scientific statements do not recommend their routine use because of a higher likelihood of errors associated with incorrect positioning.

Wrist devices, however, are useful for individuals whose arms do not fit into available brachial cuff sizes or those who may have issues related to using an upper arm cuff over a long-term period."

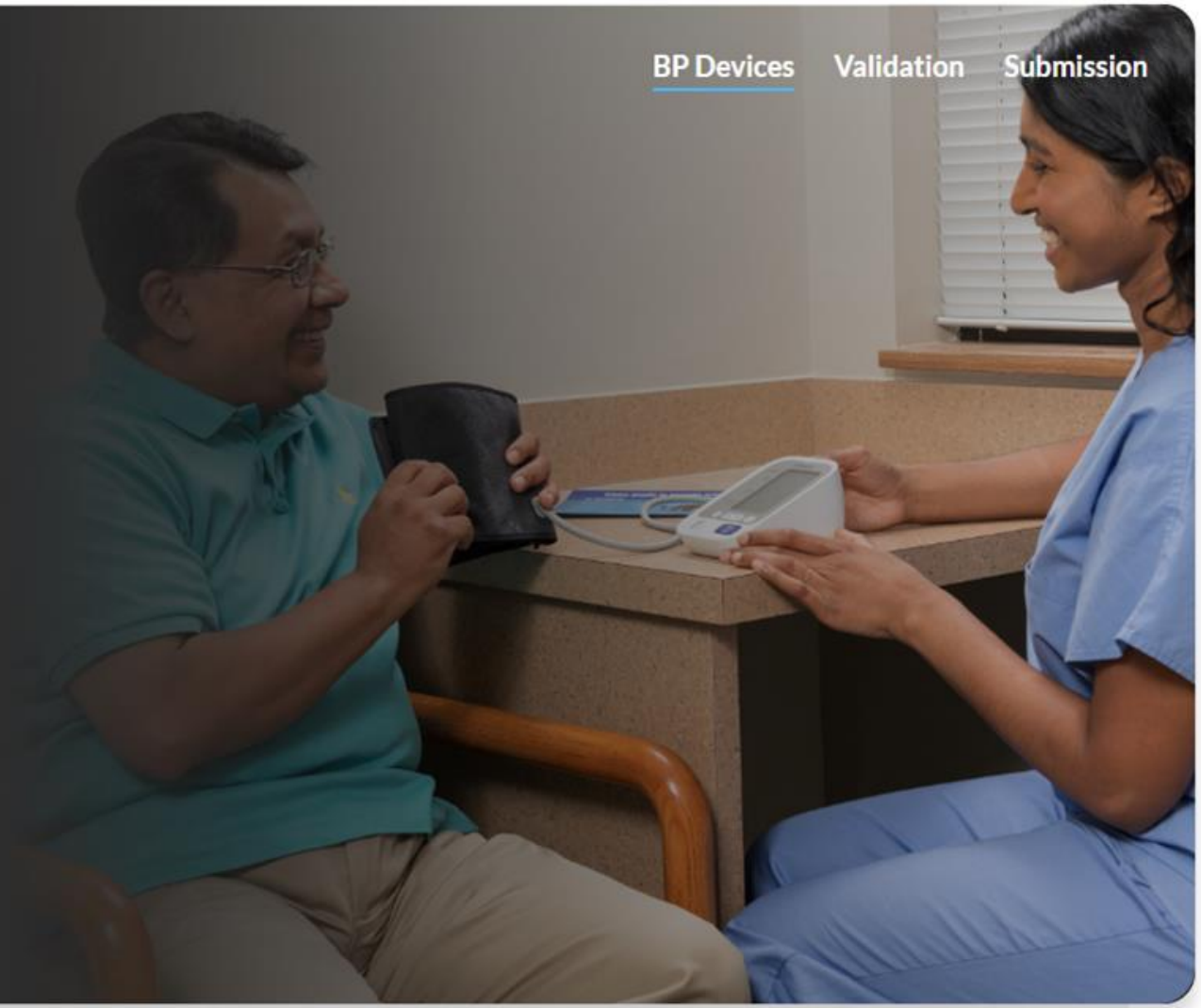
Circulation. 2020;141:00–00.

BP Devices Validation Submission

US BLOOD PRESSURE VALIDATED DEVICE LISTING

Blood pressure measurement devices that have been validated for clinical accuracy as determined through an independent review process.

See more ⊕



Validatebp.org

US Blood Pressure Validated Device Listing (VDL™)

The ultimate judgment regarding whether a BP measurement device meets the requisite VDL Criteria rests with the Independent Review Committee and is not in any way determined or influenced by the AMA. The AMA does not receive funding from any device manufacturer or other third party in relation to the development of the VDL Criteria or VDL process.*

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Patient Case #1

Luke J. Laffin, MD FACC

Co-Director, Center for Blood Pressure Disorders

Medical Director of Cardiac Rehabilitation

Cleveland Clinic

Cleveland, Ohio USA

61 year old woman with GERD and is overweight

Presents to PCP office for annual physical. Her former PCP left office, new patient visit.

Blood pressure checked in office after 5 minutes of waiting, using a brachial cuff
= **168 / 101 mm Hg with a heart rate of 82 bpm**

PCP reviews chart and blood pressure trends over past 4 years that patient has been seen:

145 / 95 mm Hg
138 / 89 mm Hg
151 / 96 mm Hg

Noted by prior treating physician…

“Patient running late, may contribute to elevated office BP reading”

“States she has normal BP at home”

“Encouraged lifestyle modification including low sodium diet”

“Stage 1 Hypertension: Lifestyle modification, reassess in follow-up”

61 year old woman with GERD and is overweight

Presents to PCP office for annual physical. Her former PCP left office, new patient visit.

Blood pressure checked in office after 5 minutes of waiting, using a brachial cuff
= **168 / 101 mm Hg with a heart rate of 82 bpm**

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What do you do?

Noted by prior treating physician…

"Patient running late, may contribute to elevated office BP reading"

"States she has normal BP at home"

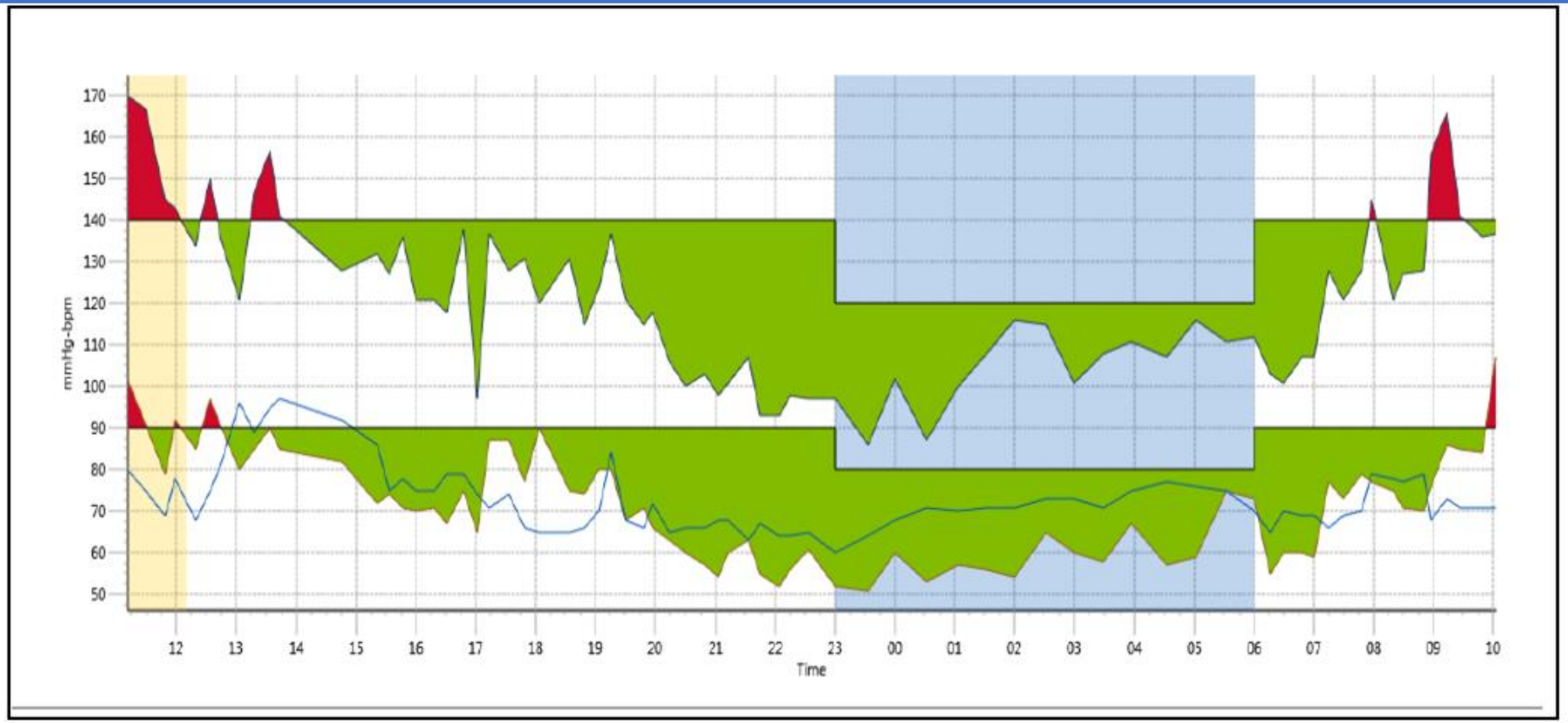
"Encouraged lifestyle modification including low sodium diet"

"Stage 1 Hypertension: Lifestyle modification, reassess in follow-up"


120/80

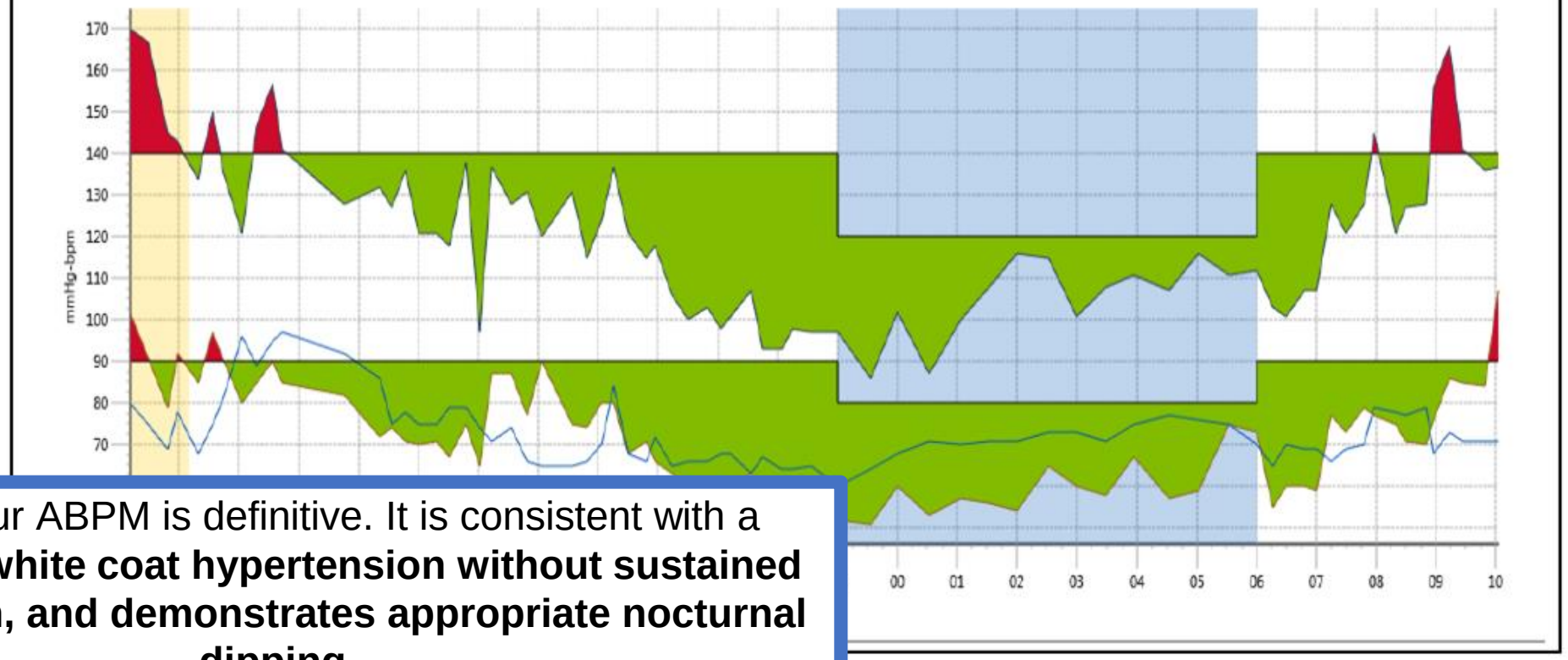

CUFF SIZE INDEX
CE
LATEX FREE
ADULT M L
22-32 cm
8.7-12.6 inches
ERY MARK
01:38
158
96
83
SYS.
mmHg
DIA.
mmHg
PUL.
/min
START
STOP


Reusable ABPM
Blood Pressure Cuff
ADULT
22-32cm



Brachial BP Results

Period	Time	Samples	Mean SYS mmHg (+/- Std.Dev)	Mean DIA mmHg (+/- Std.Dev)	Mean HR BPM (+/- Std.Dev)	BP Load Sys (%)	BP Load Dia (%)
Overall	11:11-10:02 (22:51)	72	121 (+/-19.6)	72 (+/-13.2)	73 (+/-7.8)	17	8
Awake Period	06:00-23:00	58	125 (+/-19.3)	75 (+/-12.6)	73 (+/-8.4)	21	10
Asleep Period	23:00-06:00	14	105 (+/-9.7)	59 (+/-6.5)	71 (+/-4.6)	0	0
White Coat Period	11:11-12:10 (1st Hr.)	4				100	75
Max			170	102	80		
Mean			156	91	76		
Asleep Dip:	SYS = 16.5% DIA =21.1%						



This 24-hour ABPM is definitive. It is consistent with a diagnosis of **white coat hypertension without sustained hypertension, and demonstrates appropriate nocturnal dipping**.

Period	Time	Samples	Mean SYS mmHg (+/- Std.Dev)	Mean DIA mmHg (+/- Std.Dev)	Mean HR BPM (+/- Std.Dev)	BP Load Sys (%)	BP Load Dia (%)
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Certified Cardiometabolic Health Professional (CCHP)

Patient Case #2

Luke J. Laffin, MD FACC

Co-Director, Center for Blood Pressure Disorders

Medical Director of Cardiac Rehabilitation

Cleveland Clinic

Cleveland, Ohio USA

74 year old man with resistant hypertension, dyslipidemia, left ventricular systolic dysfunction, and IgA nephropathy

Office BP 127/57 mm Hg with a heart rate of 66 bpm

Brings with him a number of home BPs that are quite variable

Currently taking for BP:

- Carvedilol 25 mg BID
- Amlodipine 10 mg nightly
- Torsemide 20 mg daily
- Losartan 100 mg daily
- Hydralazine 50 mg TID
- Isosorbide mononitrate 60 mg daily
- Spironolactone 25 mg daily
- Tamsulosin 0.4 mg daily

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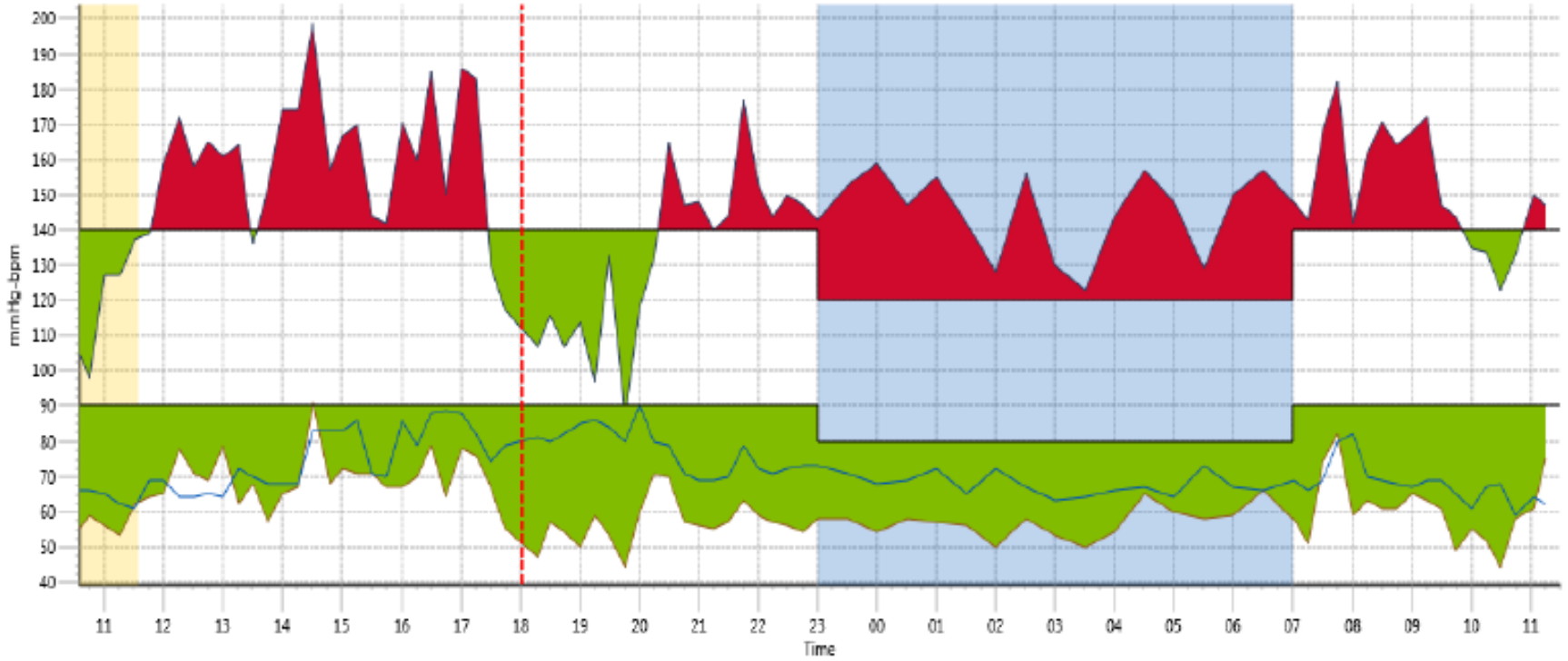
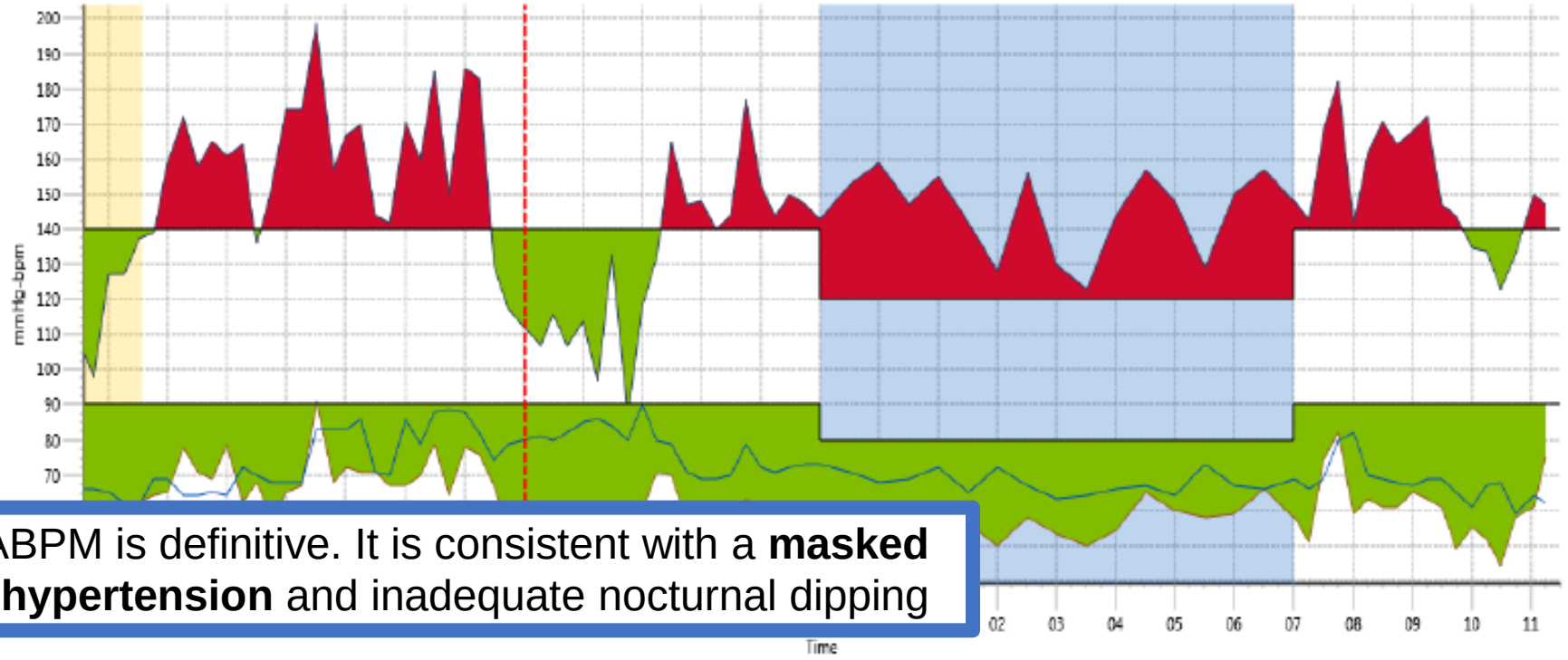
- Tamsulosin 0.4 mg daily

"I'm taking too many meds doc"



Brachial BP Results

Period	Time	Samples	Mean SYS mmHg (+/- Std.Dev)	Mean DIA mmHg (+/- Std.Dev)	Mean HR BPM (+/- Std.Dev)	BP Load Sys (%)	BP Load Dia (%)
Overall	10:34-11:30 (24:56)	83	147 (+/-22)	62 (+/-8.9)	72 (+/-7.8)	72	1
Awake Period	07:00-23:00	67	147 (+/-23.8)	63 (+/-9.4)	73 (+/-8.3)	66	1
Asleep Period	23:00-07:00	16	145 (+/-11.8)	57 (+/-4.4)	68 (+/-3.4)	100	0
White Coat Period	10:34-11:33 (1st Hr.)	5				0	0
Max			137	62	66		
Mean			119	57	64		
Asleep Dip:	SYS = 1.5% DIA =8.8%						



This 24-hour ABPM is definitive. It is consistent with a **masked uncontrolled hypertension** and inadequate nocturnal dipping

Brachial BP Results

Period	Time	Samples	Mean SYS mmHg (+/- Std.Dev)	Mean DIA mmHg (+/- Std.Dev)	Mean HR BPM (+/- Std.Dev)	BP Load Sys (%)	BP Load Dia (%)
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Mean			119	57	64		
Asleep Dip:	SYS = 1.5% DIA =8.8%						

SO YOU HAVE CONFIRMED A DIAGNOSIS OF SUSTAINED HYPERTENSION…

Medications and other substances that cause elevations in BP

AGENT	POSSIBLE MANAGEMENT STRATEGY
Alcohol	Limit alcohol to ≤ 1 drink for women and ≤ 2 drinks for men
Amphetamines	Discontinue or decrease dose Consider behavioral therapies for ADHD
Antidepressants (MAOIs, SNRIs, TCAs)	Consider alternative agents (eg .SSRIs) depending on indication Avoid tyramine-containing foods with MAOIs
Atypical antipsychotics (eg. Clozapine, olanzapine)	Discontinue or limit use Consider behavioral therapy where appropriate Recommend lifestyle modification Consider alternative agents associated with lower risk of weight gain, diabetes mellitus, and dyslipidemia (eg. aripiprazole, ziprasidone)
Caffeine	Generally limit intake to < 300 mg/day Avoid use in patient with uncontrolled hypertension Coffee use in patient with hypertension is associated with acute increase in BP, long-term use is not associated with increased BP or CVD
Decongestants (eg. phenylephrine, pseudoephedrine)	Use for shortest duration possible, and avoid in severe or uncontrolled hypertension Consider alternative therapies (eg. nasal saline, intranasal corticosteroids, antihistamines as appropriate.
Herbal supplements (eg. Ma Huang [ephedra], St. John's wort[with MAO inhibitors, yohimbine]	Avoid use
Immunosuppressants (eg. Cyclosporine)	Consider converting to tacrolimus, which may be associated with fewer effects on BP
Oral contraceptives	Use low dose (eg. 20-30 mcg ethinyl estradiol agents or a progestin only form of contraception, or consider alternative forms of birth control where appropriate (eg. barrier, abstinence, IUD) Avoid use in women with uncontrolled hypertension
NSAIDs	Avoid systemic NSAIDs when possible Consider alternative analgesics (eg. Acetaminophen, tramadol, topical NSAIDs) depending on indication and risk
Recreational drugs (eg. "bath salts [MDPV], cocaine, methamphetamine, etc.)	Discontinue or avoid use
Systemic corticosteroids (eg. dexamethasone, fludrocortisone, methylprednisolone, prednisone, prednisolone)	Avoid or limit use when possible Consider alternative modes of administration (eg. Inhaled or topical) when feasible
Angiogenesis inhibitors (eg. bevacizumab) and tyrosine kinase inhibition (eg. sunitinib, sorafenib)	Initiate or intensify antihypertensive therapy

J Am Coll Cardiol. Sep 2017, 23976

WHAT OTHER TESTING SHOULD BE UNDERTAKEN?

Other evaluation at time of hypertension diagnosis

- Thorough history
- Physical exam
- Fasting blood glucose*
- Complete blood count
- Lipid profile
- Serum creatinine with eGFR*
- Serum sodium, potassium and calcium*
- Thyroid stimulating hormone
- Urinalysis
- Electrocardiogram

* May be included in a complete metabolic panel

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Other evaluation at time of hypertension diagnosis

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- Fasting blood glucose*
- Complete blood count
- Lipid profile
- Serum creatinine with eGFR*
- Serum sodium, potassium and calcium*
- Thyroid stimulating hormone
- Urinalysis
- Electrocardiogram

Optional testing

- Echocardiogram
- Uric acid
- Urinary albumin to creatinine ratio

* May be included in a complete metabolic panel

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WHAT ARE SECONDARY CAUSES OF HYPERTENSION?

WHEN TO SCREEN FOR SECONDARY HYPERTENSION?

Secondary causes of hypertension

COMMON

- Renal parenchymal disease
- Renal vascular disease
- Primary aldosteronism
- Obstructive sleep apnea
- Drug or alcohol induced

UNCOMMON

- Pheochromocytoma and paraganglioma
- Cushing's syndrome
- Hypothyroidism
- Hyperthyroidism
- Aortic coarctation (undiagnosed or repaired)
- Primary hyperparathyroidism
- Congenital adrenal hyperplasia
- Mineralocorticoid excess syndromes other than primary hypertension
- Acromegaly

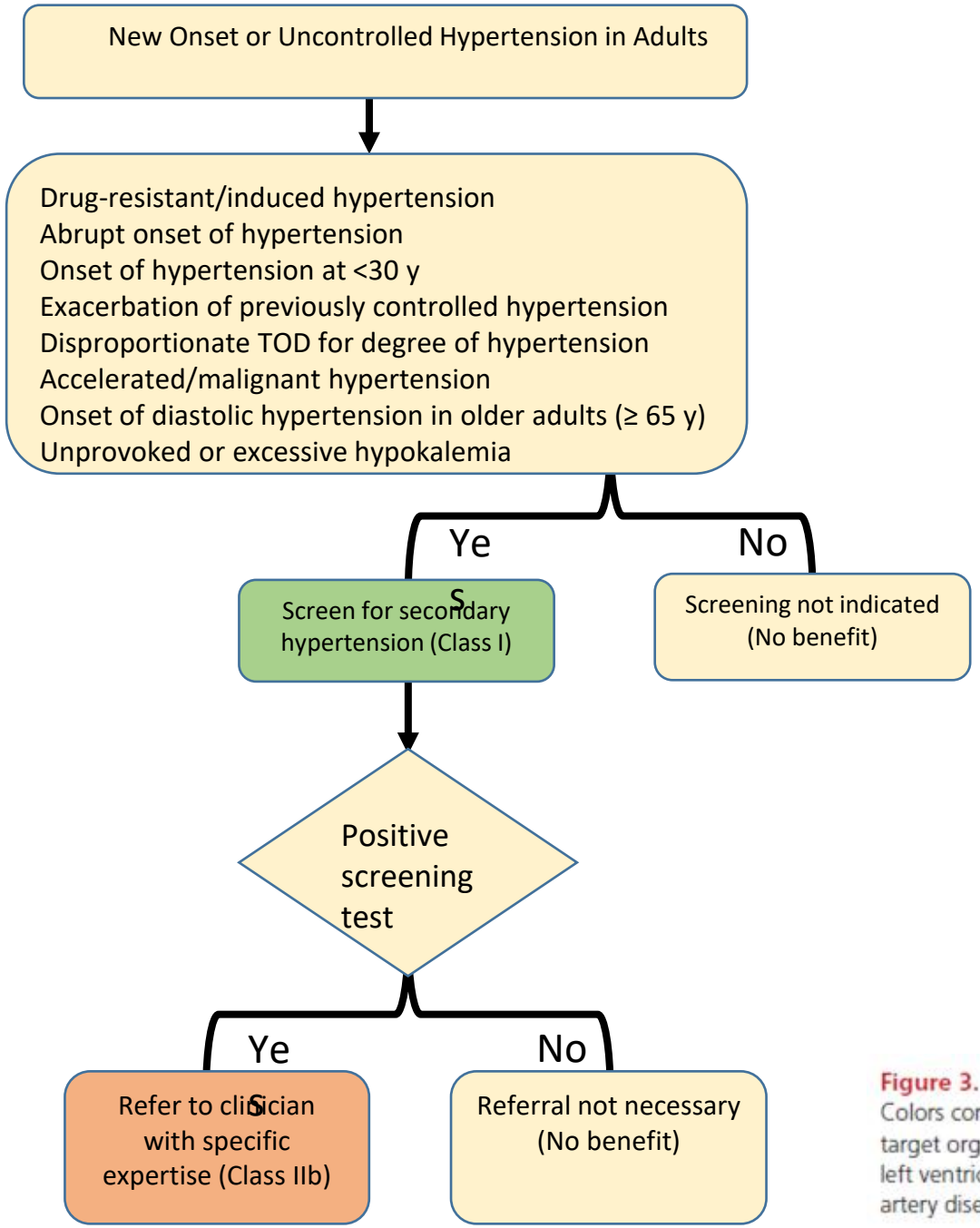

Figure 3. Screening for secondary hypertension.
Colors correspond to Class of Recommendation in Table 1. TOD indicates target organ damage (eg, cerebrovascular disease, hypertensive retinopathy, left ventricular hypertrophy, left ventricular dysfunction, heart failure, coronary artery disease, chronic kidney disease, albuminuria, peripheral artery disease).

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Patient Case #3

Luke J. Laffin, MD FACC

Co-Director, Center for Blood Pressure Disorders

Medical Director of Cardiac Rehabilitation

Cleveland Clinic

Cleveland, Ohio USA

67 year old woman with elevated coronary artery calcium, dyslipidemia, and early family history of ASCVD

Presented to PCP office for exercise treadmill test as part of executive physical.

Noted to have severely elevated BPs (160-180s / 90-100s mmHg). New finding for her.

No history of hypertension previously. Started on lisinopril and amlodipine and told to return to care in one month.

Initial doses of these medications were increased due to inadequate control.

BP ultimately controlled but patient was previously on no medications and wondering why the sudden shift.

67 year old woman with elevated coronary artery calcium, dyslipidemia, and early family history of ASCVD

Presented to PCP office for exercise treadmill test as part of executive physical.

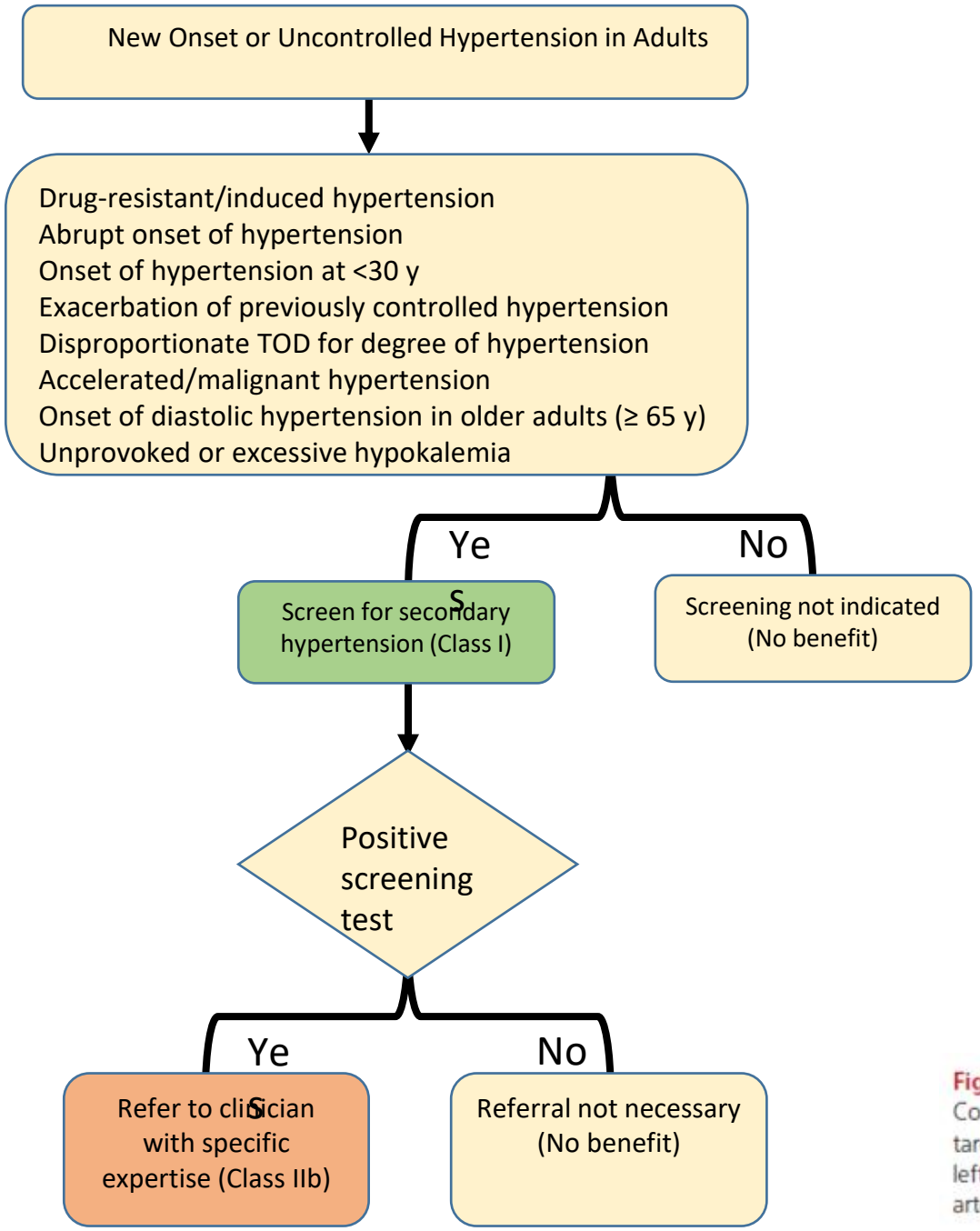
Noted to have severely elevated BPs (160-180s / 90-100s mmHg). New finding for her.

No history of hypertension previously. Started on lisinopril and amlodipine and told to return to care in one month.

Initial doses of these medications were increased due to inadequate control.

BP ultimately controlled but p den shift.

What do you do?



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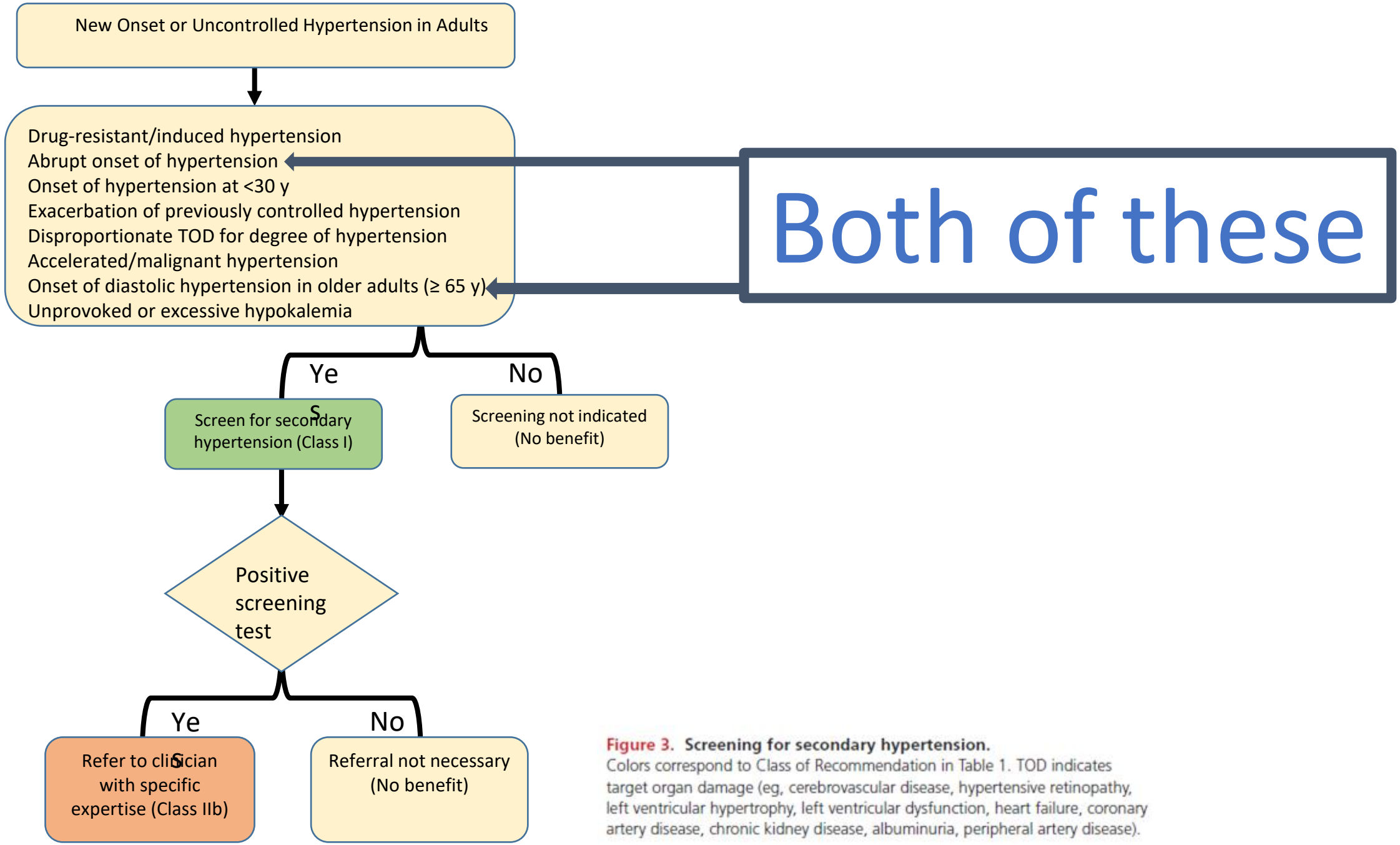

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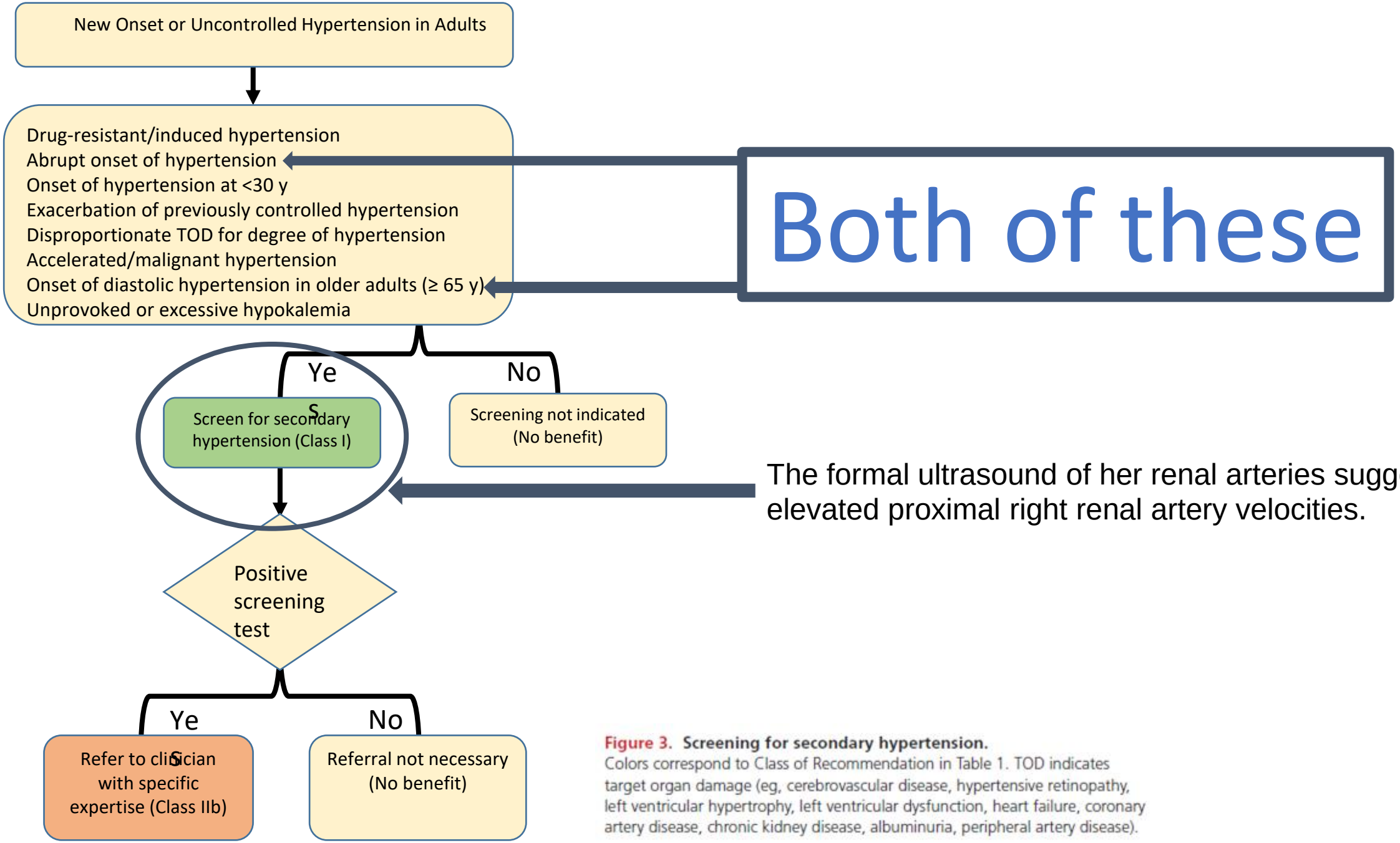

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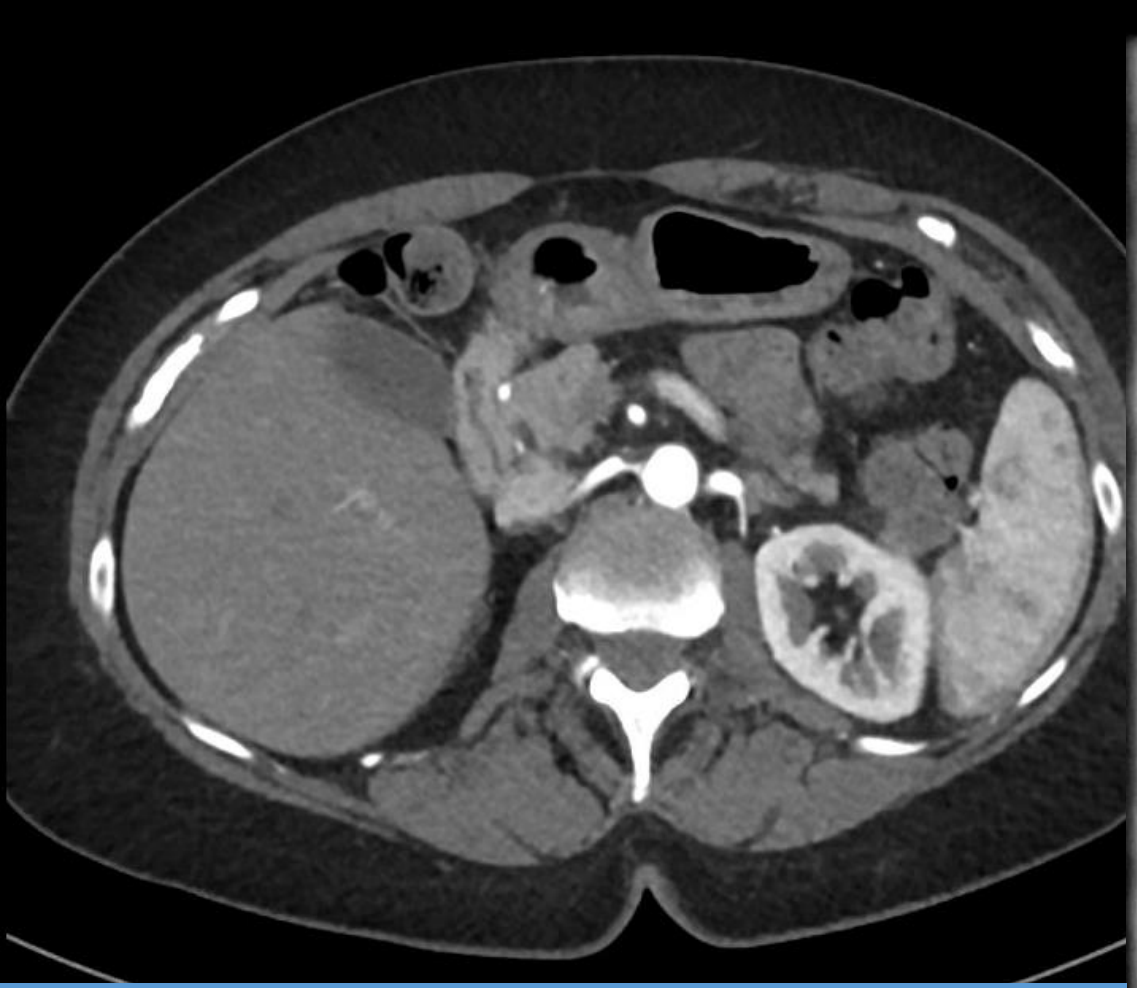

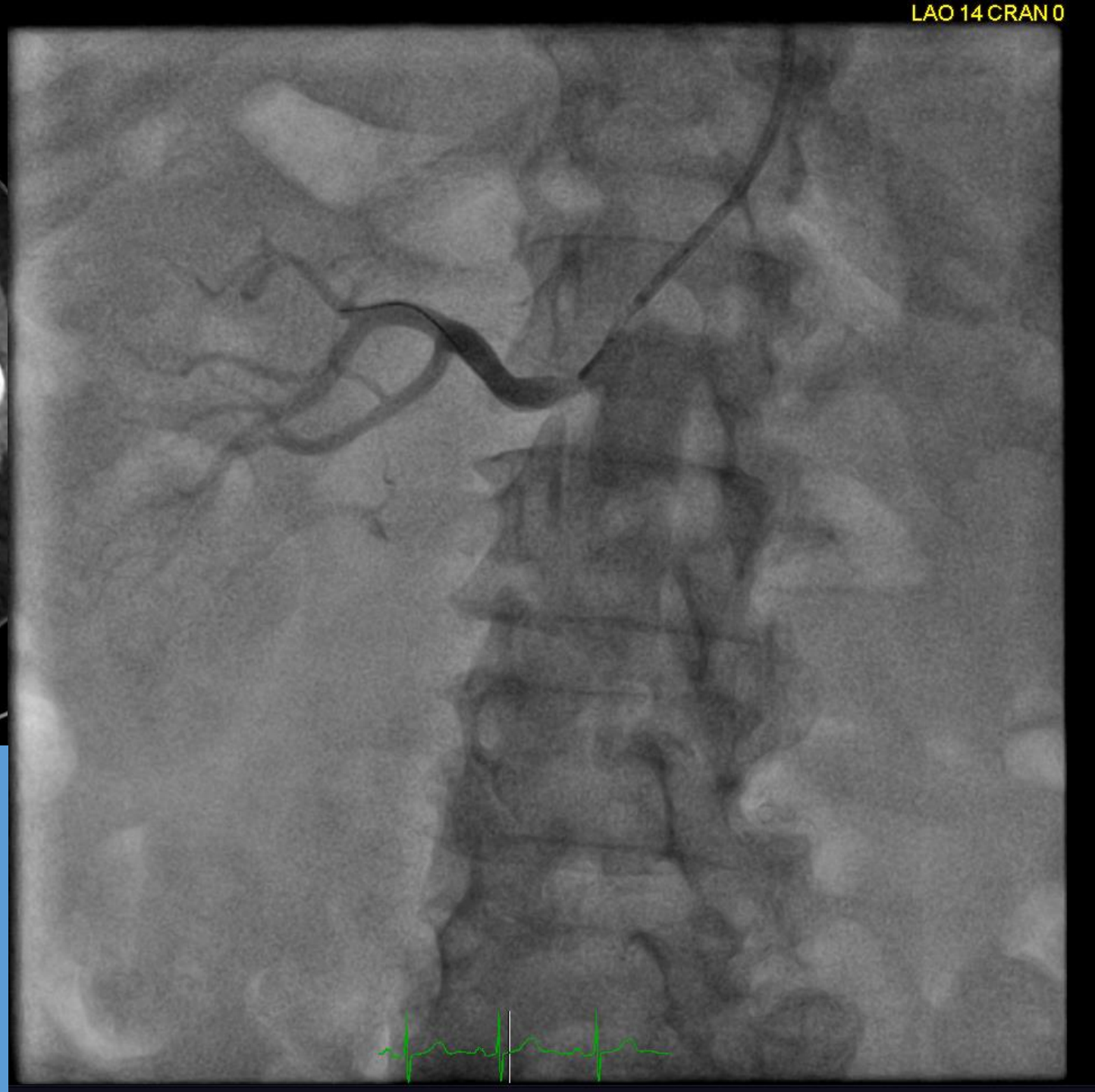

Stent of the right renal artery with a 5.0 x 16 mm Synergy Megatron, post-dilated to 6.0 mm

One month post procedure she had stopped all blood pressure lowering medications and home blood pressures are well controlled

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Patient Case #4

Luke J. Laffin, MD FACC

Co-Director, Center for Blood Pressure Disorders

Medical Director of Cardiac Rehabilitation

Cleveland Clinic

Cleveland, Ohio USA

39 year old man with bicuspid aortic valve, LV dilation and AI, and uncontrolled hypertension

Diagnosis of hypertension for 5+ years.

For his bicuspid AV he had a MRA of the aorta which did not reveal a coarctation.

Renal artery duplex was not suggestive of renal artery stenosis

Hypokalemic on prior BMP, had tried spironolactone in the past but had GI upset with it.

Prior screening for primary aldosteronism only checked a serum aldosterone level, no renin.

Checks home BP with a validated brachial BP machine. Ranges between 160-180 mmHg/80-90 mmHg with a heart rate between 50-60 bpm.

Very physically active, low sodium diet, BMI 24.

Taking hydralazine 50 mg BID, Lisinopril 40 mg daily, and metoprolol succinate 50 mg daily.

Scheduled for AVR and aortic repair in 6 weeks.

39 year old man with bicuspid aortic valve, LV dilation and AI, and uncontrolled hypertension

Diagnosis of hypertension for 5+ years.

For his bicuspid AV he had a MRA of the aorta which did not reveal a coarctation.

Renal artery duplex was not suggestive of renal artery stenosis

Hypokalemic on prior BMP, had tried spironolactone in the past but had GI upset with it.

Prior screening for primary aldosteronism only checked a serum aldosterone level, no renin.

Checks home BP with a validated brachial BP machine. Ranges between 160-180 mmHg/80-90 mmHg with a heart rate between 50-60 bpm

Initially prescribed chlorthalidone 25 mg daily, lisinopril 40 mg daily, carvedilol 12.5 mg BID, and amlodipine 5 mg nightly

Very p

Strongly recommended repeating aldosterone/renin testing

Taking

Aldosterone 24.5 ng/dL

Direct Renin Concentration < 2.1 pg/mL

Serum potassium 3.0 mmol/L

Primary Aldosteronism

Recommendations for Primary Aldosteronism		
COR	LOE	Recommendations
I	C-EO	1. In adults with hypertension, screening for primary aldosteronism is recommended in the presence of any of the following concurrent conditions: resistant hypertension, hypokalemia (spontaneous or substantial, if diuretic induced), incidentally discovered adrenal mass, family history of early-onset HTN, or stroke at a young age (<40 yrs)
I	C-LD	2. Use o plasma aldosterone: renin activity ratio is recommended when adults are screened for primary aldosteronism.
I	C-EO	3. In adults with hypertension and a positive screening test for primary aldosteronism, referral to a HTN specialist or endocrinologist is recommended for further evaluation and treatment.

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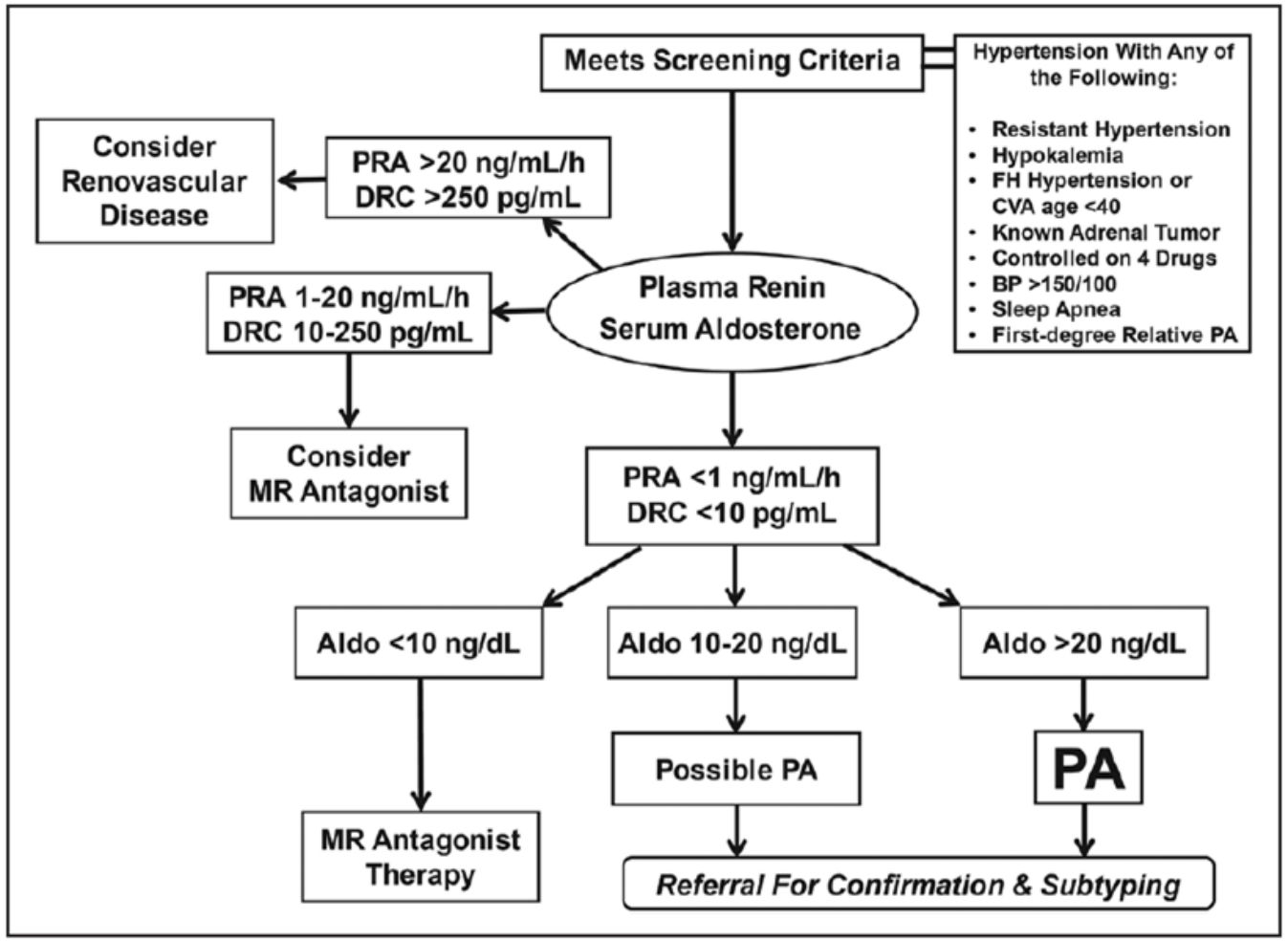
The net effect of normal physiology is that renin and aldosterone should rise and fall in parallel.

Circulation. 2018;138:823–835.

Simplified algorithm for primary aldosteronism (PA) screening and triaging based primarily on plasma renin and secondarily on serum aldosterone



Patient with low renin and aldosterone > 10 ng/dL are referred for confirmatory testing and subtyping; most patients with aldosterone > 20 ng/dL have PA and those with hypokalemia do not require confirmatory testing.

Circulation. 2018;138:823–835.

ORIGINAL RESEARCH

Annals of Internal Medicine

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; and Anand Vaidya, MD, MMSc

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.

Participants: Participants with normotension (n = 289), stage 1 hypertension (n = 115), stage 2 hypertension (n = 203), and resistant hypertension (n = 408).

Measurements: Participants completed an oral sodium suppression test, regardless of aldosterone or renin levels, as a confirmatory diagnostic for primary aldosteronism and to quantify the magnitude of renin-independent aldosterone production. Urinary aldosterone was measured in participants in high sodium balance with suppressed renin activity. Biochemically overt primary aldosteronism was diagnosed when urinary aldosterone levels were higher than 12 μg/24 h.

Results: Every blood pressure category had a continuum of renin-independent aldosterone production, where greater severity of production was associated with higher blood pressure, kaliuresis, and lower serum potassium levels. Mean adjusted levels of urinary aldosterone were 6.5 μg/24 h (95% CI, 5.2 to 7.7 μg/24 h) in normotension, 7.3 μg/24 h (CI, 5.6 to 8.9 μg/24 h) in stage 1 hypertension, 9.5 μg/24 h (CI, 8.2 to 10.8 μg/24 h) in stage 2 hypertension, and 14.6 μg/24 h (CI, 12.9 to 16.2 μg/24 h) in resistant hypertension; corresponding adjusted prevalence estimates for biochemically overt primary aldosteronism were 11.3% (CI, 5.9% to 16.8%), 15.7% (CI, 8.6% to 22.9%), 21.6% (CI, 16.1% to 27.0%), and 22.0% (CI, 17.2% to 26.8%). The aldosterone–renin ratio had poor sensitivity and negative predictive value for detecting biochemically overt primary aldosteronism.

Limitation: Prevalence estimates rely on arbitrary and conventional thresholds, and the study population may not represent nationwide demographics.

Conclusion: The prevalence of primary aldosteronism is high and largely unrecognized. Beyond this categorical definition of primary aldosteronism, there is a prevalent continuum of renin-independent aldosterone production that parallels the severity of hypertension. These findings redefine the primary aldosteronism syndrome and implicate it in the pathogenesis of "essential" hypertension.

Primary Funding Source: National Institutes of Health.

Ann Intern Med. 2020;173:10-20. doi:10.7326/M20-0065 **Annals.org**
For author, article, and disclosure information, see end of text.
This article was published at Annals.org on 26 May 2020.

ORIGINAL RESEARCH

Annals of Internal Medicine

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; and Anand Vaidya, MD, MMSc

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:

Participa

hypertension (n = 115), stage 2 hypertension (n = 203), and resistant hypertension (n = 408).

Measurements: Participants completed an oral sodium suppression test, regardless of aldosterone or renin levels, as a confirmatory diagnostic for primary aldosteronism and to quantify the magnitude of renin-independent aldosterone production. Urinary aldosterone was measured in participants in high sodium balance with suppressed renin activity. Biochemically overt primary aldosteronism was diagnosed when urinary aldosterone levels were higher than 12 µg/24 h.

Results: Every blood pressure category had a continuum of renin-independent aldosterone production, where greater severity of production was associated with higher blood pressure, kaliuresis, and lower serum potassium levels. Mean adjusted levels of urinary aldosterone were 6.5 µg/24 h (95% CI, 5.2 to 7.7 µg/24 h) in normotension, 7.3 µg/24 h (CI, 5.6 to 8.9 µg/24 h) in stage 1 hypertension, 9.5 µg/24 h (CI, 8.2 to 10.8 µg/24 h) in stage 2 hypertension, and 14.6 µg/24 h (CI, 12.9 to 16.2 µg/24 h) in resistant hypertension; corresponding adjusted prevalence estimates for biochemically overt primary aldosteronism were 11.3% (CI, 5.9% to 16.8%), 15.7% (CI, 8.6% to 22.9%), 21.6% (CI, 16.1% to 27.0%), and 22.0% (CI, 17.2% to 26.8%). The aldosterone–renin ra-

detecting

conventional thresholds, and the study population may not represent nationwide demographics.

Conclusion: The prevalence of primary aldosteronism is high and largely unrecognized. Beyond this categorical definition of primary aldosteronism, there is a prevalent continuum of renin-independent aldosterone production that parallels the severity of hypertension. These findings redefine the primary aldosteronism syndrome and implicate it in the pathogenesis of "essential" hypertension.

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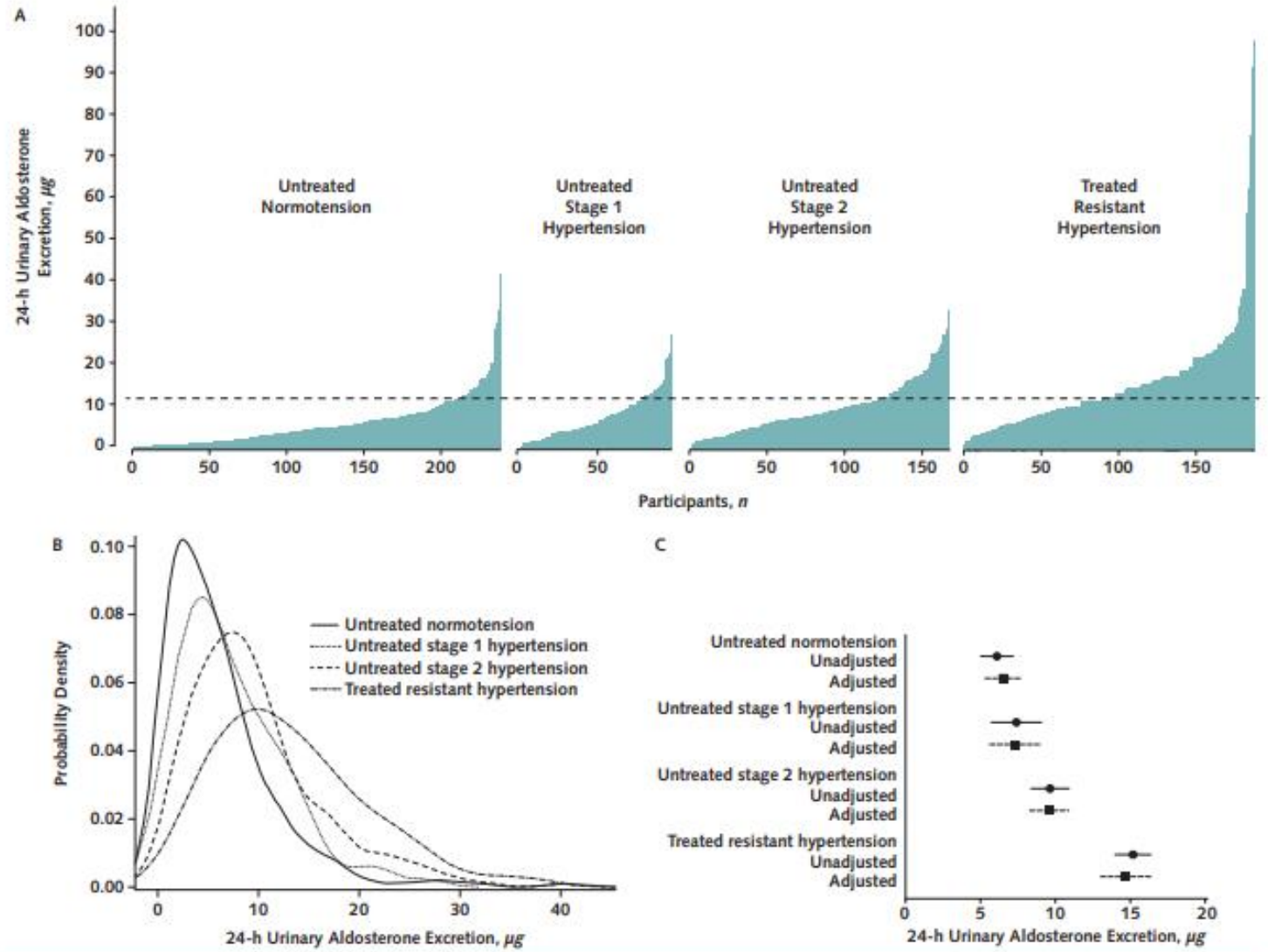
Journal article provided to students

Figure 2. Distribution of renin-independent aldosterone production, by blood pressure category.



A. The unadjusted urinary aldosterone excretion rate in the context of high sodium balance and renin suppression. Vertical bars represent the unadjusted renin-independent aldosterone excretion rate (*y*-axis) for each individual participant, ordered from lowest to highest (*x*-axes). The dashed horizontal line represents the conventional 12 μg/24 h threshold for the diagnosis of biochemically overt primary aldosteronism. B. Unadjusted overlaid density plots depicting the distribution of renin-independent aldosterone production, by blood pressure category (truncated at 45 μg/24 h). The *x*-axis shows the 24-h urinary aldosterone excretion rate. The *y*-axis shows the probability density function (smoothed using a kernel density estimation) per unit on the *x*-axis. C. Mean (95% CI) urinary aldosterone excretion rates for each blood pressure category, unadjusted (*solid lines with circles*) and adjusted (*dotted lines with squares*) for age, body mass index, race, sex, history of diabetes, and 24-h urinary sodium excretion.

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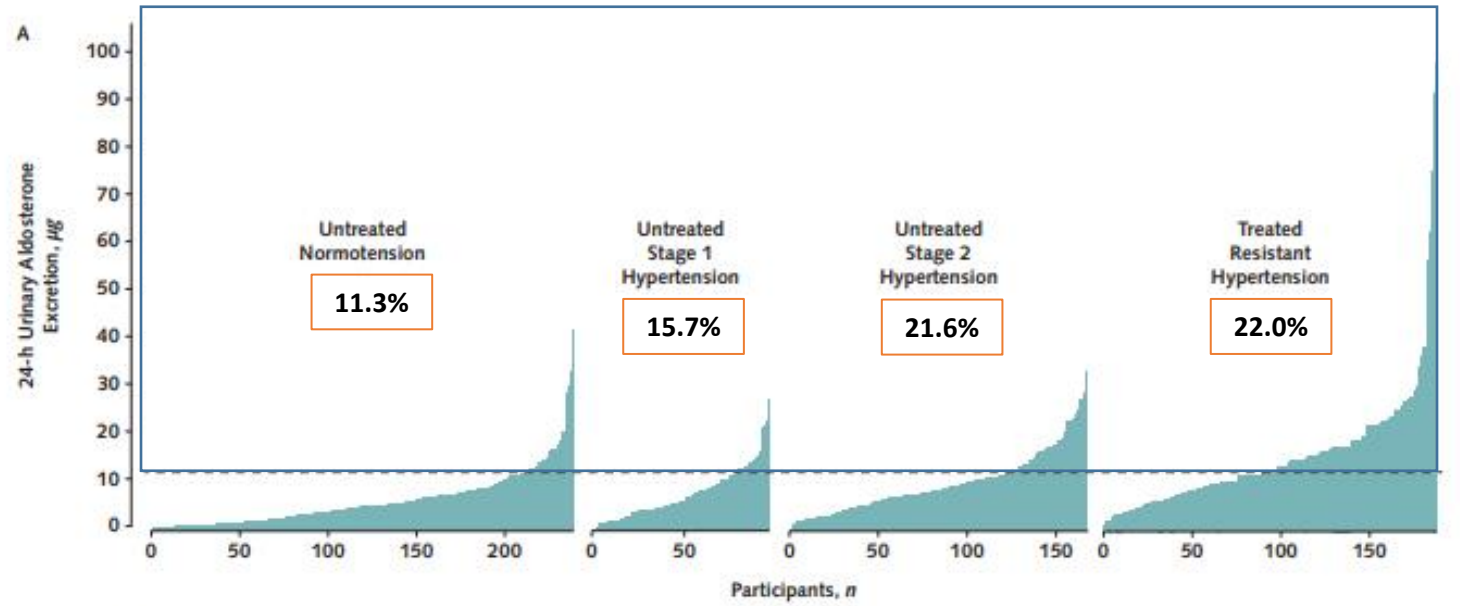
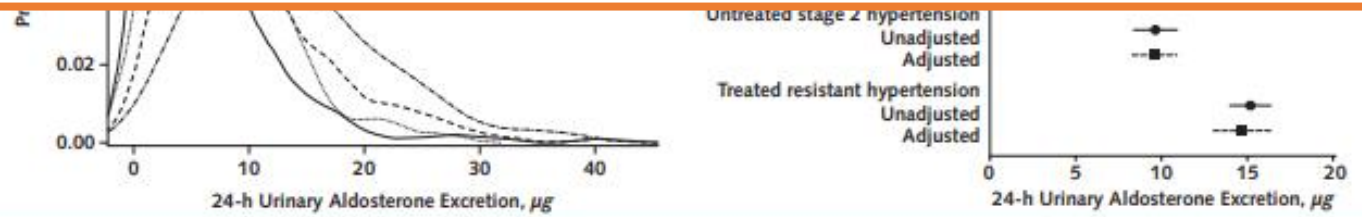


1st step: Identify patients with overt primary aldosteronism



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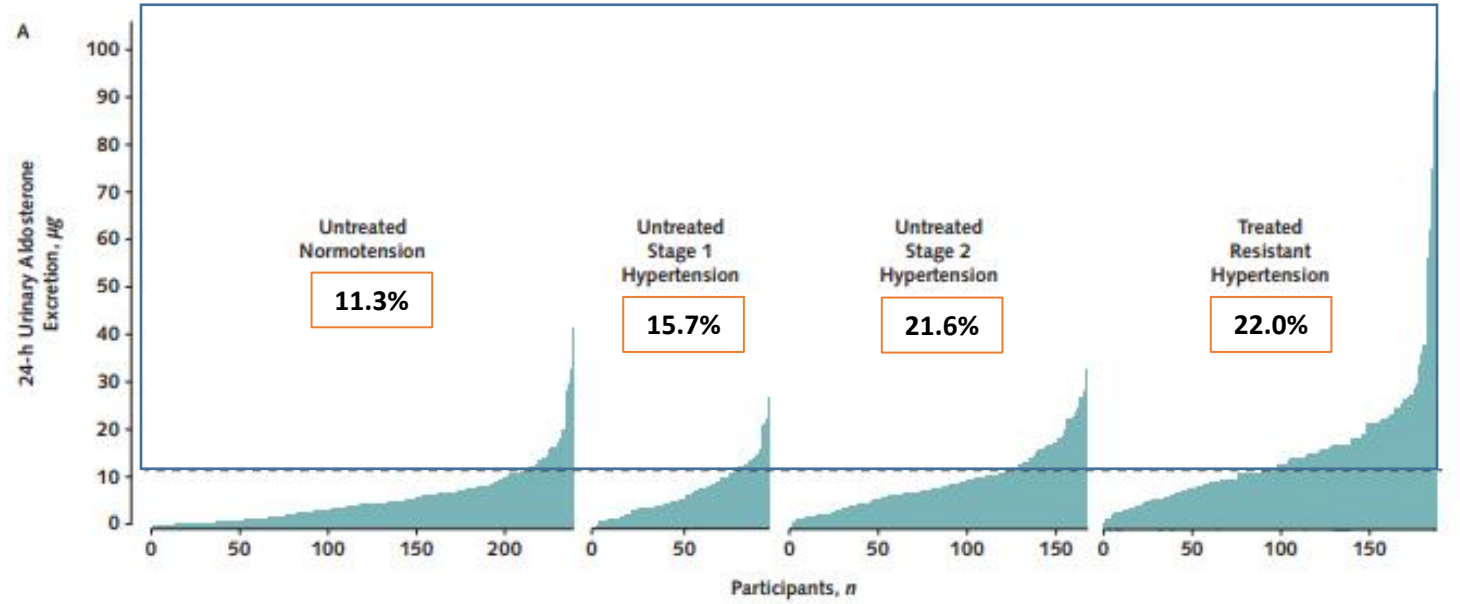

2nd step: Realize that autonomous aldosterone production is a spectrum of disease and is seen in primary hypertension

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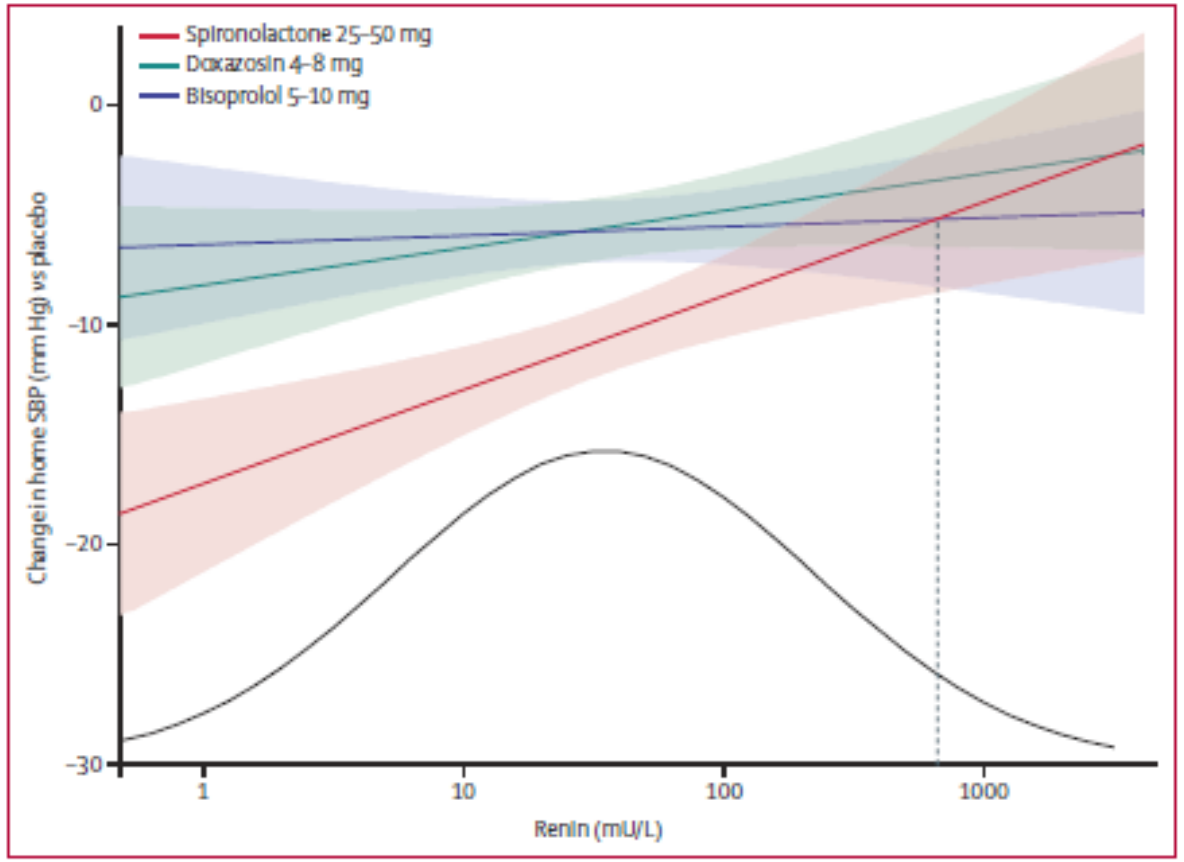
Blood Pressure Response versus Renin in the Pathway-2 Trial



Lancet 2015; 386: 2059–68

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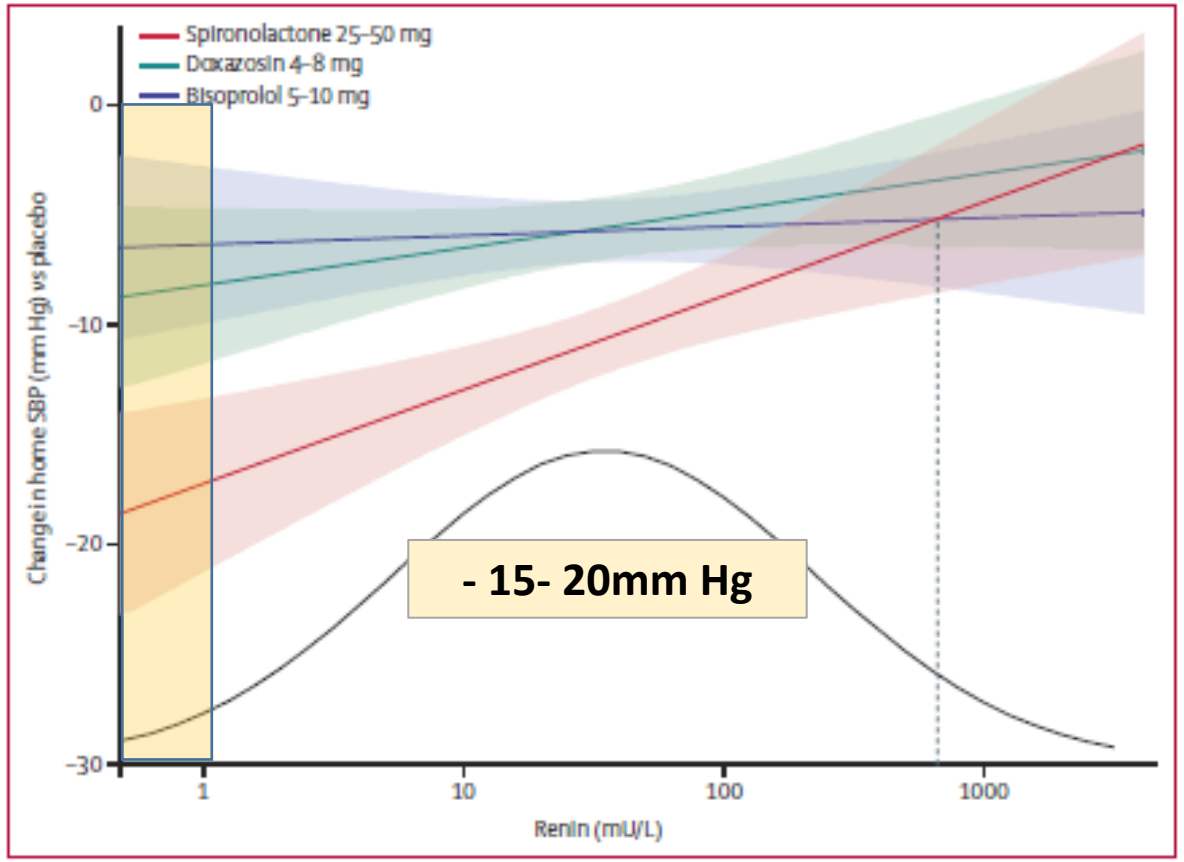


Lancet 2015; 386: 2059–68

When aldosterone production is autonomous….

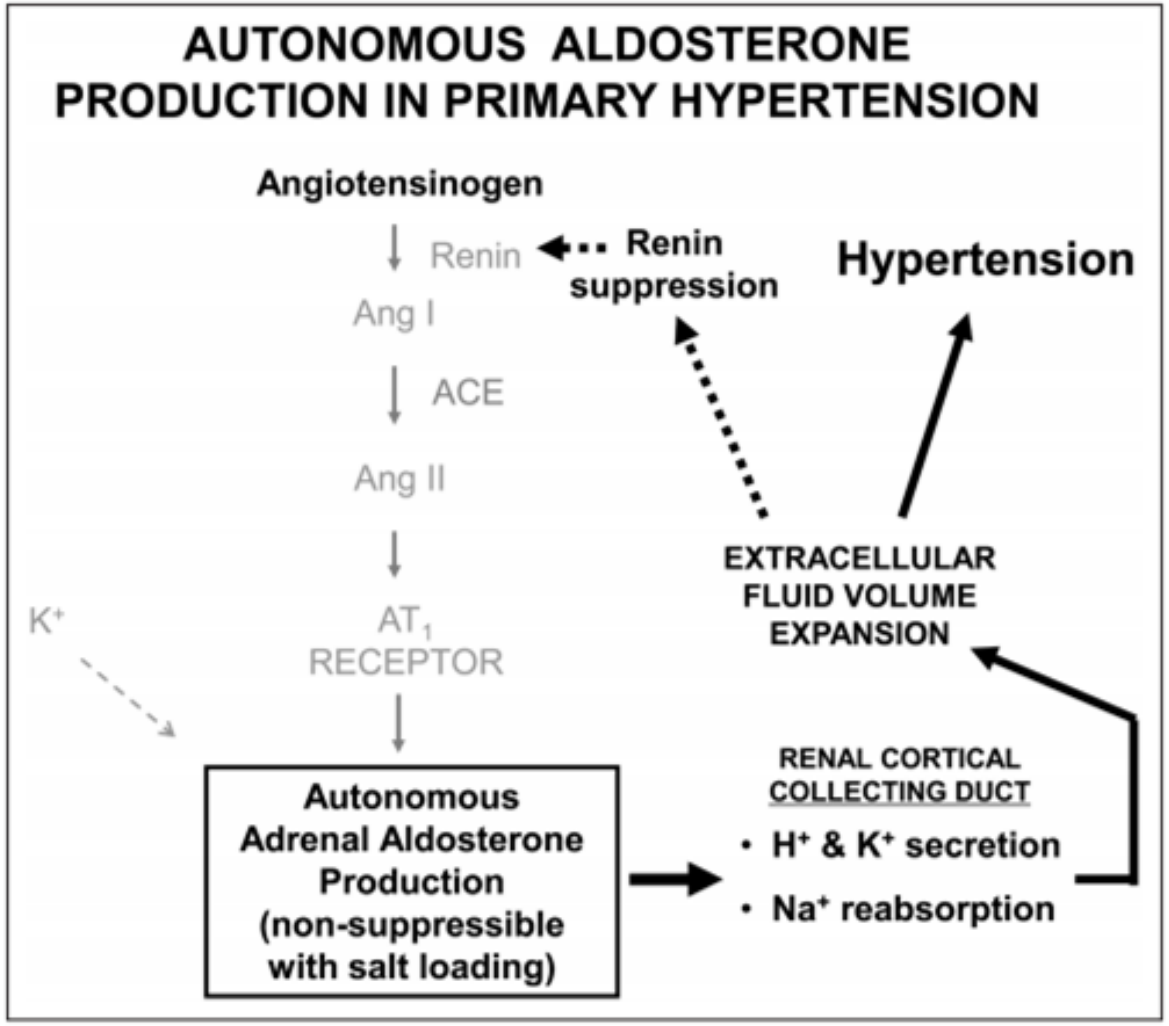
Excess aldosterone

Aldosterone producing adenoma (APA)

Bilateral hyperaldosteronism (BHA)

- **Bilateral adenomas**
- **Unilateral hyperplasia**
- **Micronodules**
- **Microscopic aldosterone-producing cell clusters (APCCs)**

Schematic representation of the mechanisms of autonomous aldosterone production in primary hypertension



Aldosterone production from the adrenal zona glomerulosa is independent of the renin-angiotensin system and is not suppressible with dietary sodium loading. Excess aldosterone expands extracellular fluid volume by augmenting sodium reabsorption in the renal cortical collecting duct. Expanded fluid volume lead to hypertension and suppression of renin and the entire renin-angiotensin cascade. Increased aldosterone production is abnormal in the face of renin suppression but plasma aldosterone concentrations are lower than those of patient with classical overt primary aldosteronism.

Circulation Research. 2021;128:827–846

CARDIOVASCULAR RISK FACTORS ASSOCIAED WITH HYPERTENSION

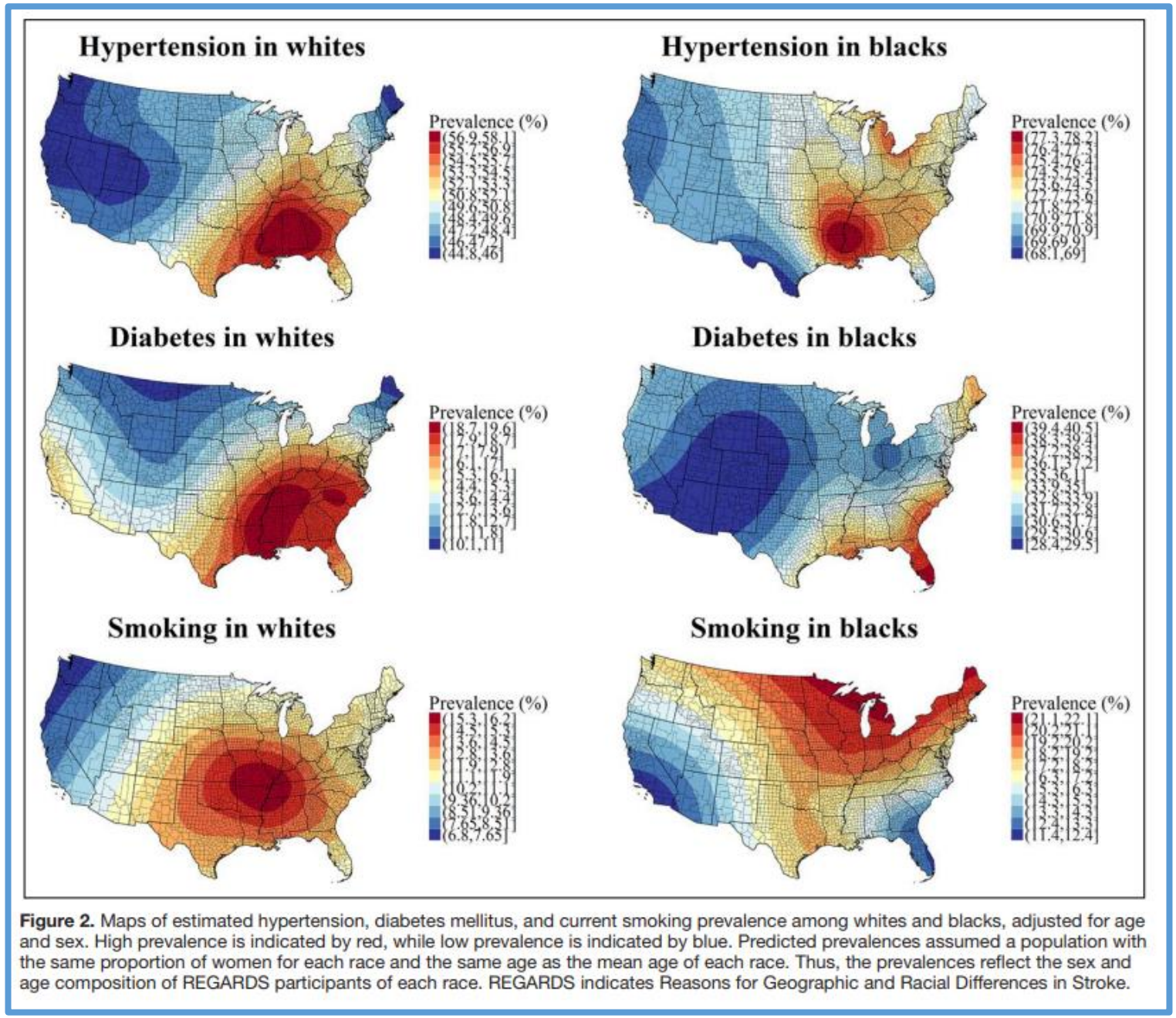


Figure 2. Maps of estimated hypertension, diabetes mellitus, and current smoking prevalence among whites and blacks, adjusted for age and sex. High prevalence is indicated by red, while low prevalence is indicated by blue. Predicted prevalences assumed a population with the same proportion of women for each race and the same age as the mean age of each race. Thus, the prevalences reflect the sex and age composition of REGARDS participants of each race. REGARDS indicates Reasons for Geographic and Racial Differences in Stroke.

Circ Cardiovasc Qual Outcomes. 2017;10:e003350-00.

Cardiovascular Disease Risk Factors Common in Patients with Hypertension

MODIFIABLE RISK FACTORS*

- Smoking
- Diabetes mellitus
- Dyslipidemia
- Overweight / obesity
- Physical inactivity / low fitness
- Unhealthy diet

*Factors that can be changed and, if changed may reduce CVD risk

RELATIVELY FIXED RISK FACTORS+

- Chronic kidney disease
- Family history
- Increased age
- Low socioeconomic status / educational status
- Male sex
- Obstructive sleep apnea
- Psychosocial stress

+Factors that are difficult to change (CKD, socioeconomic status, obstructive sleep apnea), cannot be changed (family history, increased age, male sex), or if changed through the use of current intervention techniques may not reduce CVD risk (psychosocial stress).

Adapted from Whelton et al. J Am Coll Cardiol 2018;71:e127–248