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

Primary Aldosteronism

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CONTINUING MEDICAL EDUCATION
DEPARTMENT OF MEDICINE

HARVARD MEDICAL SCHOOL
TEACHING HOSPITAL

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Training

- Harvard Medical School
- IM Residency @BWH
- Endocrinology Fellowship @BWH
- Cardiovascular Endocrinology Research Post-Doc @BWH

Positions

- Associate Professor of Medicine @HMS
- Chief (Interim), Division of Endocrinology, Diabetes, and Hypertension @BWH
- Director, Center for Adrenal Disorders @BWH
- Director, e-Learning Initiative @BWH/@NEJM
- Former Director, Homeostasis II Curriculum @HMS

Key Learning Points

Primary aldosteronism is in your clinic every week, but you are almost never recognizing it

Primary aldosteronism is very common; **most low-renin hypertension is a manifestation of Primary Aldosteronism Pathophysiology**

PARADIGM SHIFT

Primary aldosteronism is not a rare cause of "secondary HTN"; rather it is a highly prevalent and modifiable contributor to HTN and CVD

Case

36-year old woman presents for hypertension management

Age 27: 1st pregnancy, preeclampsia => Persistent HTN => nifedipine

Age 28 (CCB)	Age 32 (no meds)	Age 33 (CCB, ACEi)	Age 36 (CCB, ACEi)
PAC (LC-MS/MS): 8.3 ng/dL (230 pmol/L)	PAC (LC-MS/MS): 6.6 ng/dL (183 pmol/L)	K **3.3 mEq/L**	PAC (LC-MS/MS): 8.1 ng/dL (225 pmol/L)
PRA <0.6 ng/mL/h	PRA <0.6 ng/mL/h		PRA <0.6 ng/mL/h
ARR >14	ARR >11		ARR >14
K 4.1 mEq/L	K 4.4 mEq/L		K 4.3 mEq/L

Does this patient have primary aldosteronism? YES

Is further testing needed to make the diagnosis? NO

Vaidya et al. NEJM 2025

Case

36-y ...nts fo... ...om...

Age ...ecla... ...N

Low-Renin HTN
Renin-Independent
Aldosteronism

Low-Renin HTN
Renin-Independent
Aldosteronism

Hypokalemia
on ACEi?

Renin low on ACEi
ALDO not suppressed
on ACEi

Age 28 (CCB)
PAC (LC-MS/MS): 8.3 ng/dL
(230 pmol/L)
PRA <0.6 ng/mL/h
ARR >14
K 4.1 mEq/L

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(183 pmol/L)
PRA <0.6 ng/mL/h

Age 33 (CCB, ACEi)
K **3.3 mEq/L**

Age 36 (CCB, ACEi)
PAC (LC-MS/MS): 8.1 ng/dL
(225 pmol/L)
PRA <0.6 ng/mL/h
ARR >14
K 4.3 mEq/L

Negative Confirmatory Test PA
(expert opinion)

Oral Sodium Suppression Test:
24h UNa of 190 mEq
24h UAldo 9.7 mcg (>12)

Vaidya et al. NEJM 2025

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ARR >
K 4.4 m

Age 33 (C
K **3.3 m**

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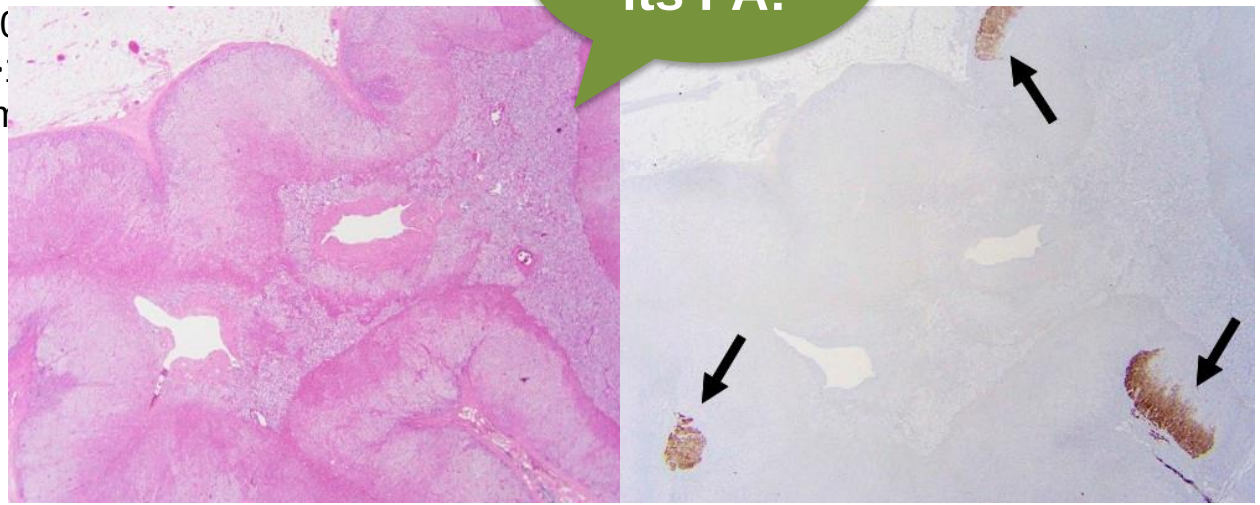
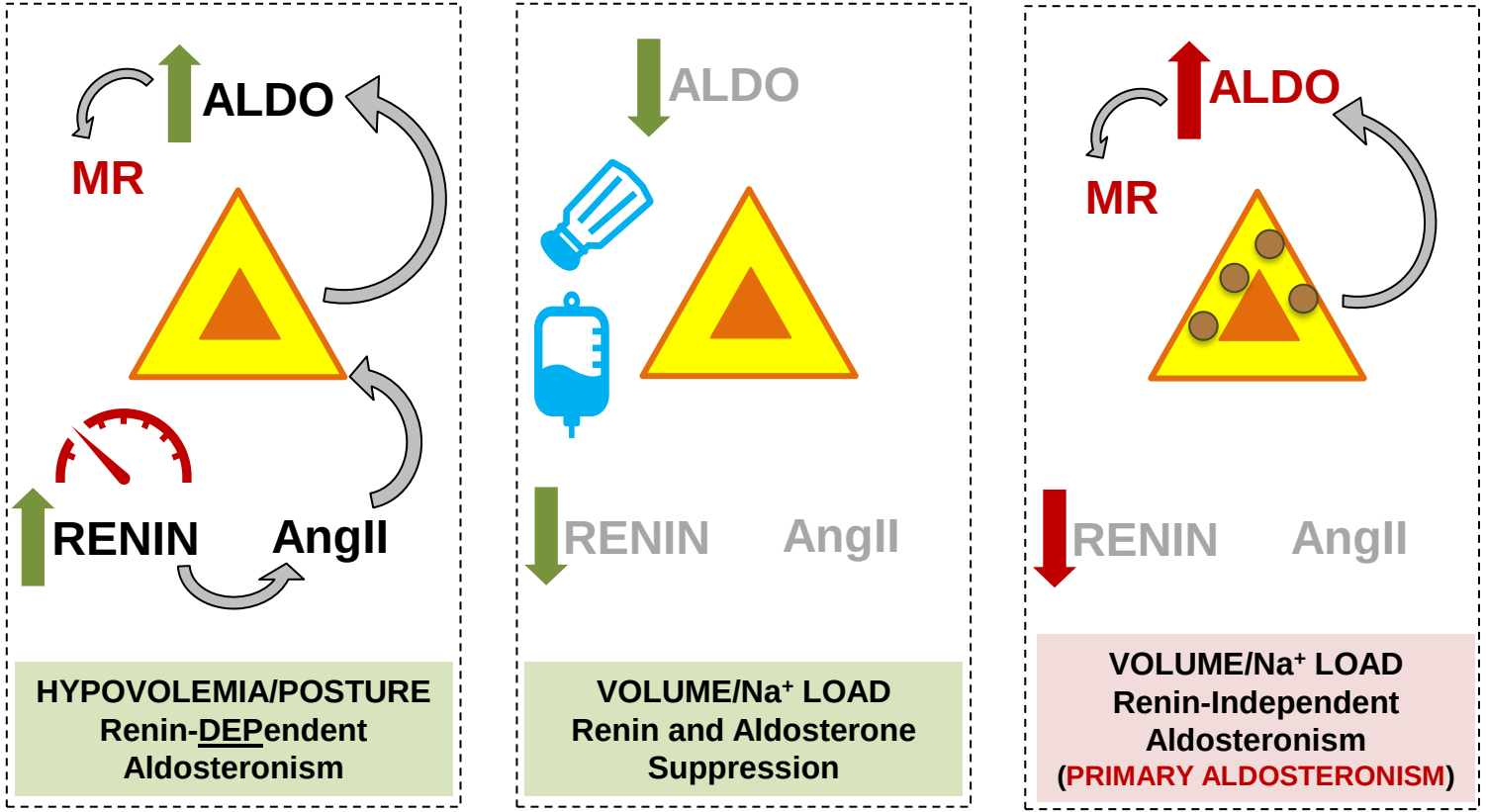


Vaidya et al. NEJM 2025

What is Primary Aldosteronism?

Primary Aldosteronism



What is Primary Aldosteronism?

PATHOPHYSIOLOGIC SYNDROME:

- Inappropriate aldosterone production: **renin-independent aldosterone production, relatively non-suppressible**
- Excessive activation of the MR, vicious cycle of volume expansion, hypertension, CV and Kidney disease *independent of BP*

Clinical Manifestations:

Most patients with PA **do not have** hypokalemia or Resistant HTN

Hallmark Biochemical Diagnostics:

Low or Suppressed Renin

Inappropriate/Dysregulated Production of Aldosterone

Vaidya et al. Endocrine Reviews 2018

Pathogenesis of PA

Key Point: The vast majority of primary aldosteronism is attributable to *diffuse and bilateral* foci of autonomous aldosterone production

Pathogenesis & Morphologies of PA

Early Life

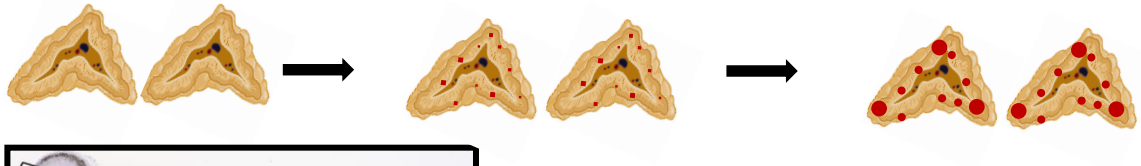
Normal Physiology & Structure

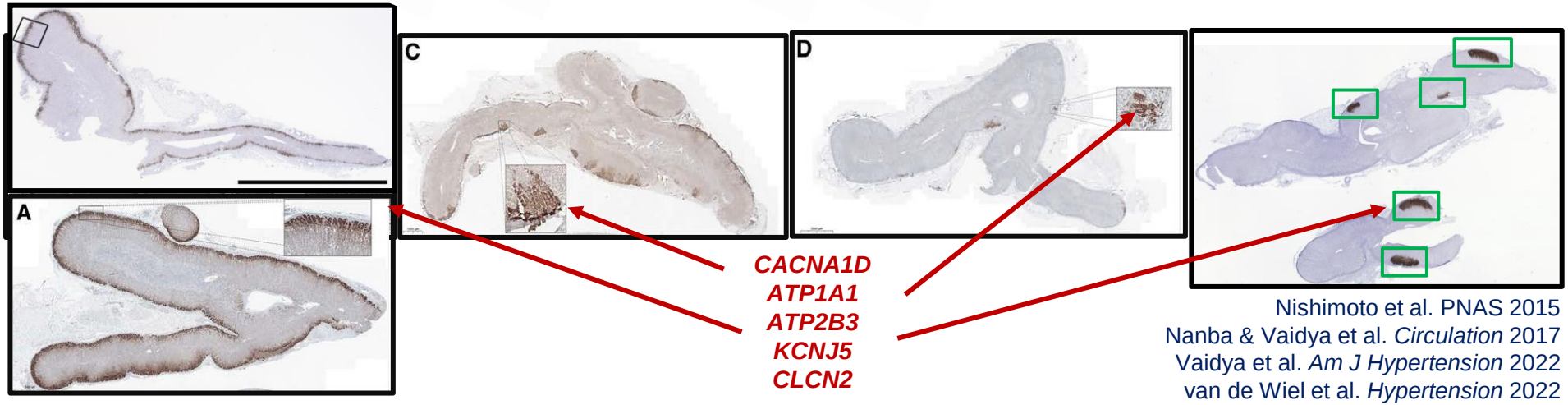
- Continuous CYP11B2 in ZG
- No neoplasia

Progressive Aging

Primary Aldosteronism with Morphologically Normal Adrenal Glands

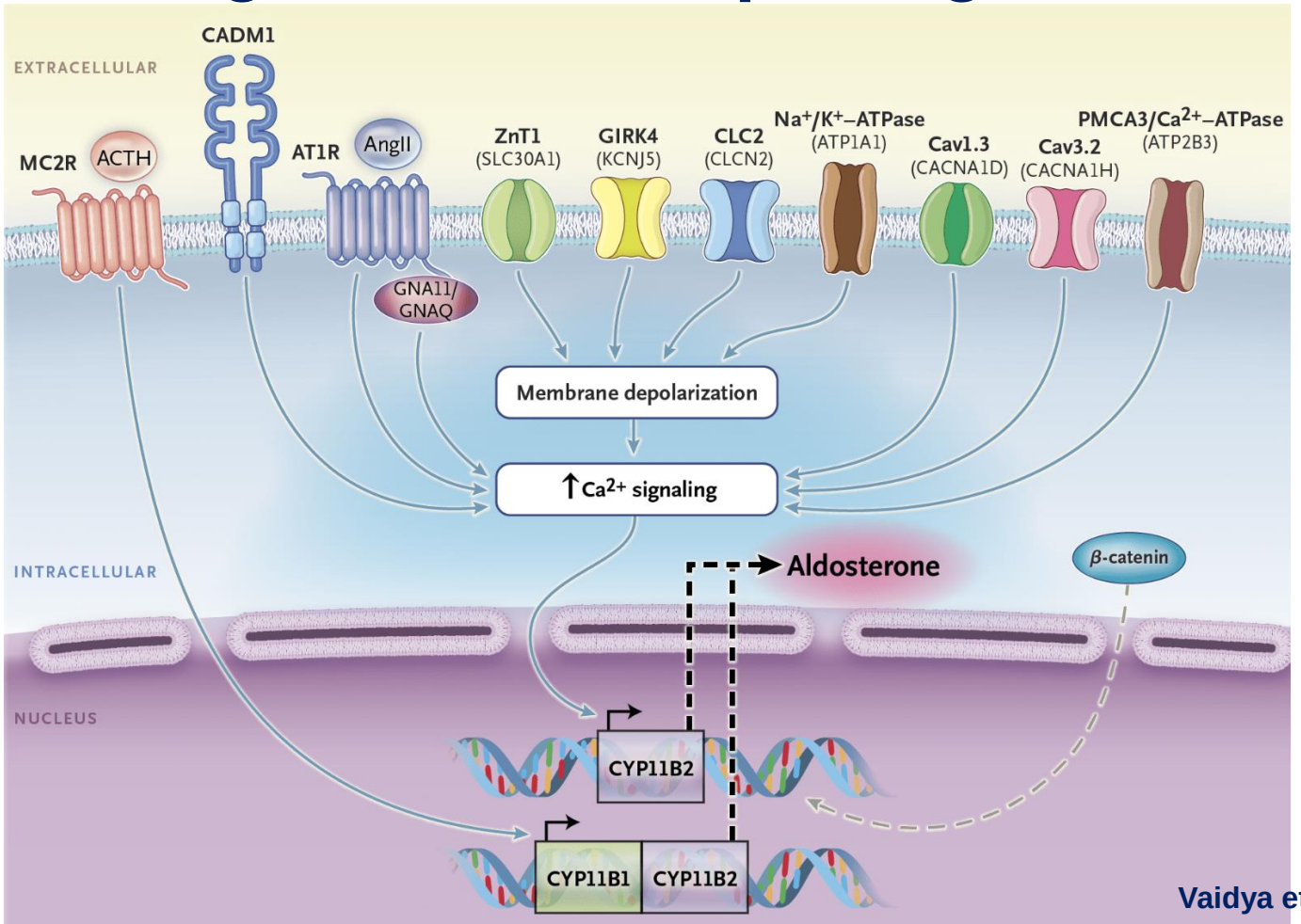
- Progressive loss of continuous ZG
- Emergence of Aldosterone Producing Cell Clusters
- Aldo-Driver somatic mutations within APCCs





Nishimoto et al. PNAS 2015
Nanba & Vaidya et al. *Circulation* 2017
Vaidya et al. *Am J Hypertension* 2022
van de Wiel et al. *Hypertension* 2022

Pathogenesis & Morphologies of PA



Vaidya et al. *NEJM* 2025

Genetic Mutations Affecting *Zona Glomerulosa* Cells to Cause Primary Aldosteronism

Mutation		Inheritance		Consequences
Gene	Protein	Germline	Somatic	
CYP11B1/CYP11B2 hybrid	CYP11B1/CYP11B2 fusion	X		Fusion of *CYP11B1* promoter to the 5'-end of *CYP11B2* such that CYP11B2 expression is regulated by adrenocorticotropic hormone (ACTH). Cause of familial hyperaldosteronism type I.
CLCN2	CLC2	X	X	Increases chloride ion efflux, which results in a lower membrane depolarization threshold. Cause of familial hyperaldosteronism type II when germline variant.
KCNJ5	GIRK4	X	X	Permits sodium ion influx, which results in a lower membrane depolarization threshold. Cause of familial hyperaldosteronism type III when germline variant.
CACNA1D	Cav1.3	X	X	Increases calcium ion influx, which causes membrane depolarization. Cause of familial hyperaldosteronism type IV when germline variant.
ATP1A1	Na^+/K^+–ATPase		X	Increases influx of sodium and hydrogen ions, which results in a lower membrane depolarization threshold.
ATP2B3	PMCA3/Ca^{2+}–ATPase		X	Increases influx of sodium and calcium ions, which results in a lower membrane depolarization threshold.
CACNA1H	Cav3.2		X	Increases calcium ion influx, which causes membrane depolarization.
CTNNB1	β–catenin		X	Increases β–catenin signaling, which in concert with other genomic and nongenomic factors can synergize primary aldosteronism pathophysiology.
GNA11/GNAQ	GNA11/GNAQ		X	Increases G-protein signaling, which in turn increases intracellular calcium signaling.
CADM1	CADM1		X	Inhibits gap-junction permeability.
SLC30A1	ZnT1		X	Increases sodium ion influx, which results in a lower membrane depolarization threshold.

<<1% of all PA is due to inheritable/germline mutations

>95% of all unilateral APA surgical specimens harbor ***pathogenic somatic mutations***

~60% of all bilateral PA surgical specimens harbor ***pathogenic somatic mutation***

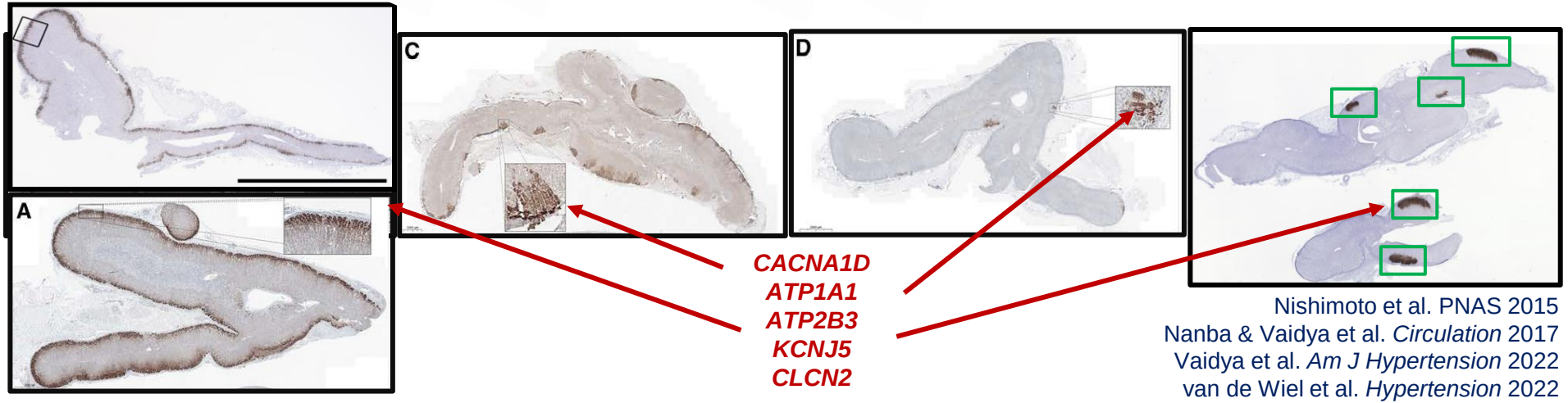
PA is largely a genetic disorder driven by somatic mutagenesis

Vaidya et al. NEJM 2025

Morphologically normal adrenals/no neoplasia

- Time/Age-dependent dysregulation CYP11B2 expression

  (loss of continuous ZG and emergence of APCCs)
- Enriched with Pathogenic Somatic Mutations in Aldosterone Driver Genes
- Bilateral process



Nishimoto et al. PNAS 2015
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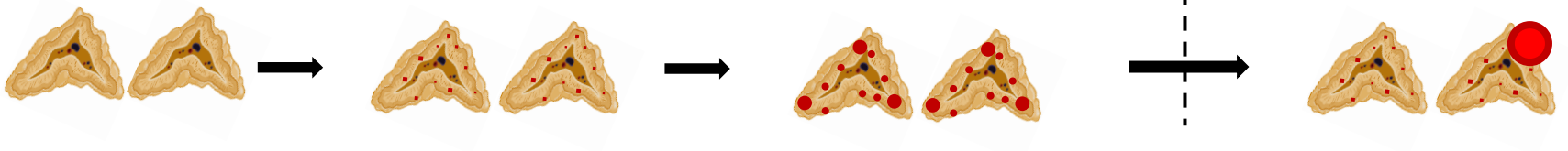
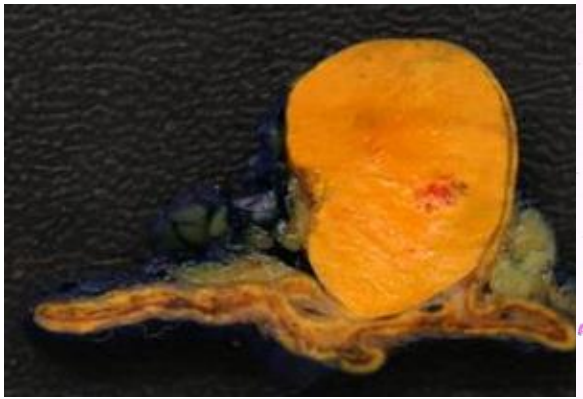
Stochastic Collision/Co-Occurrence

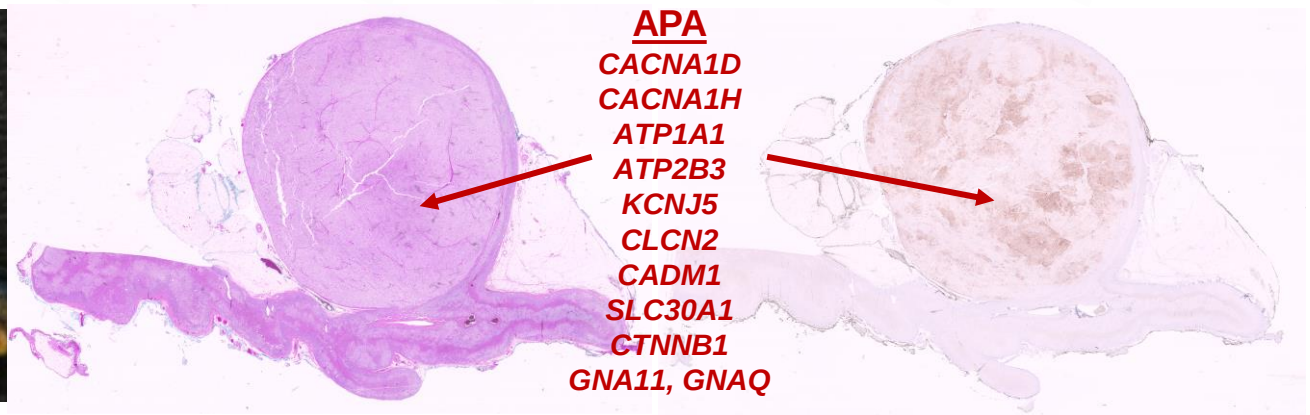
Neoplastic PA

Overt Primary Aldosteronism with Aldosterone Producing Adenoma

- Aldo-Driver somatic mutations *within* adrenocortical neoplasia







APA

CACNA1D
CACNA1H
ATP1A1
ATP2B3
KCNJ5
CLCN2
CADM1
SLC30A1
CTNNB1
GNA11, GNAQ

Vaidya et al. *Am J Hypertension* 2022

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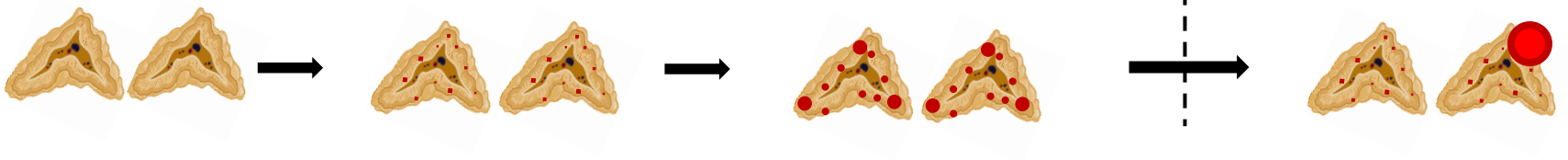


Prevalence

Severity of Primary Aldosteronism

Vaidya et al. ***Am J Hypertension*** 2022

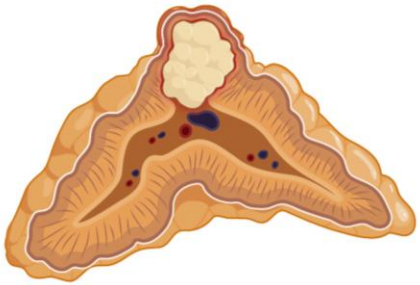
Pathogenesis & Morphology of PA

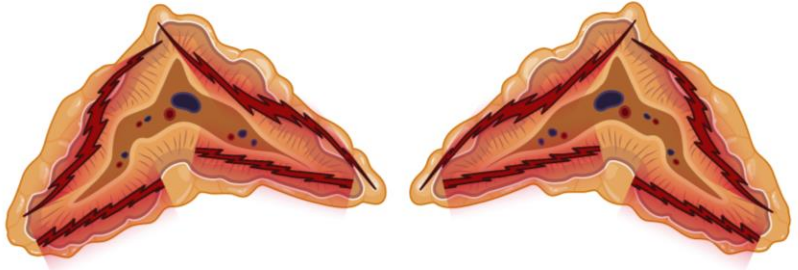
Nearly all PA is caused by *pathogenic somatic mutations* resulting in foci of autonomous aldosterone production

These foci of aldosterone production generally arise *diffusely and bilaterally*, accumulate with age, usually not neoplasia/adenomas, and not seen on standard histopathology (requires IHC for CYP11B2)

The absence of an adrenal mass does not exclude the possibility of PA; the presence of an adrenal mass(es) does not imply that it is the source of PA

Cortisol co-production from adrenal adenomas can occur in PA; all adrenal adenomas should be screened for hypercortisolism





"Unilateral"
"Aldosterone-producing adenoma"

vs.

"Bilateral"
"Adrenal Hyperplasia"

The majority of primary aldosteronism is attributable to diffuse, bilateral, microscopic foci of aldosterone production

Prevalence & Detection

Key Point: Primary Aldosteronism is *very common*
Key Point: The detection of PA is *abysmal*

Conservative Prevalence Estimates

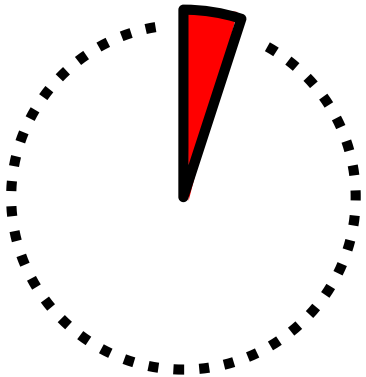
Resistant Hypertension	>25-30%
HTN + Hypokalemia	>30%
Stage I-II Hypertension	>15-20%
Pre-HTN/Normal BP	~10%

Failure to Screen for Primary Aldosteronism

Recommended Indications to Screen

- Resistant HTN
- HTN + HypoK

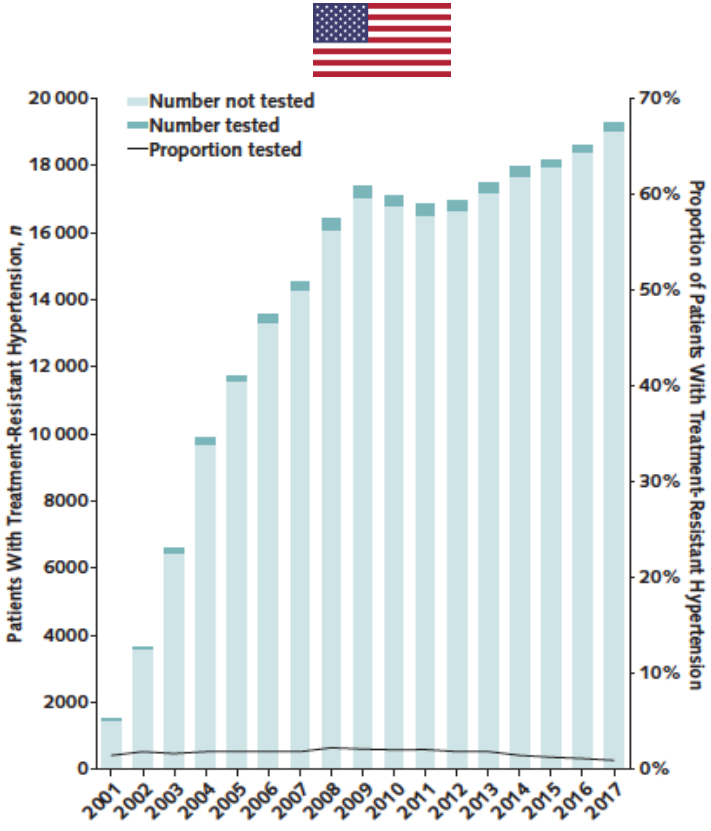
Reality

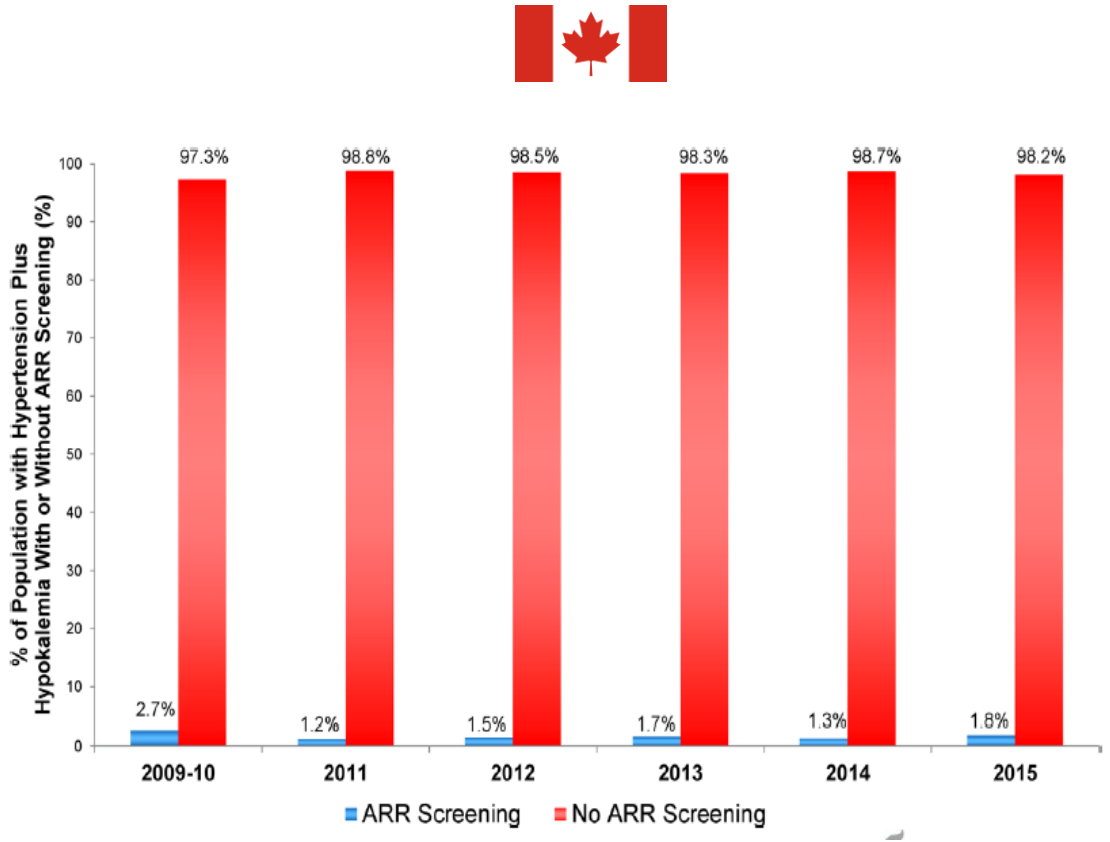


<1-2% !!

Burrello et al. Hypertension 2020; Ruhle et al. 2019; Jaffe et al. 2020; Cohen et al. 2021; Liu et al 2021; Hundemer et al. Hypertension 2021

Failure to Screen for Primary Aldosteronism





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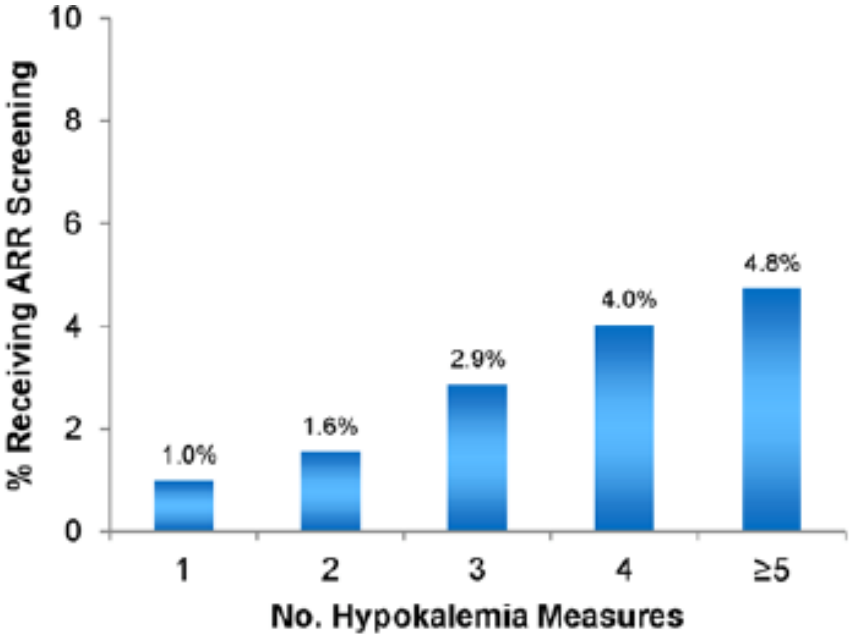
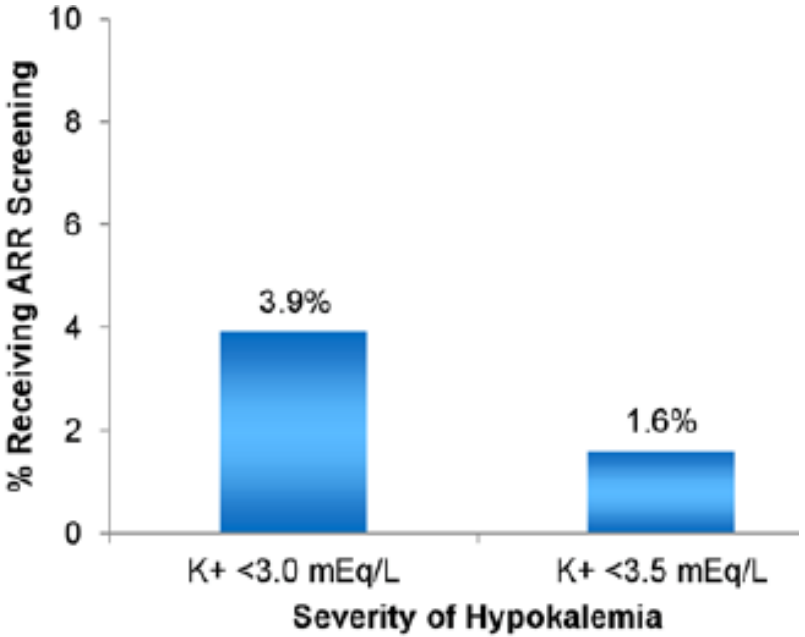




Hundemer et al. Hypertension 2021

Failure to Screen for Primary Aldosteronism

Reasons Why PA is Under-Diagnosed

1) We don’t look for it!
2) When we do look for it, *we often mis-interpret or ignore the results*

Diagnostic Testing Complexity

Key Point: The landscape of PA testing is dominated by *relatively arbitrary* and *unnecessarily complex* practices that rely on *unvalidated* diagnostic thresholds

Diagnostic Testing

There is no reference/gold-standard diagnostic
Diagnostic thresholds are relatively arbitrary and not rigorously validated

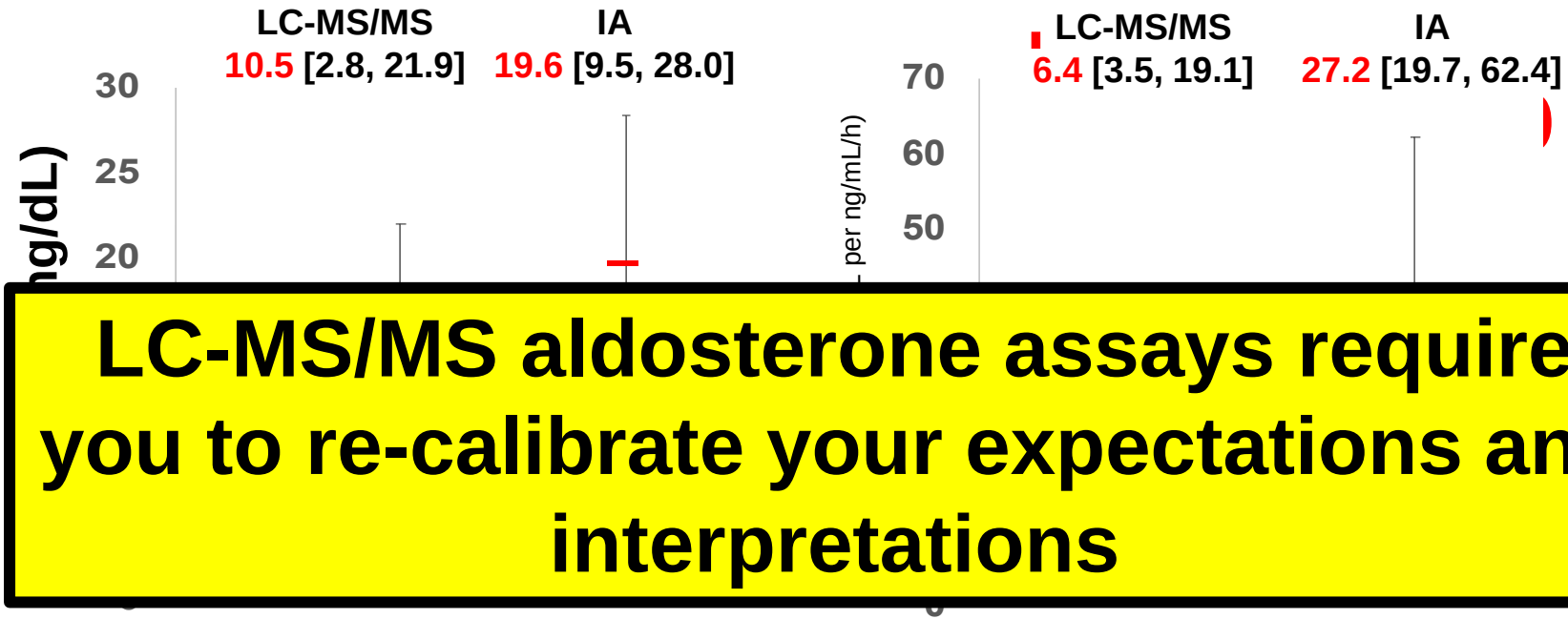
ARR ➡ **Relatively Arbitrary Thresholds**
30, 25, 20, etc.

Aldosterone ➡ **Relatively Arbitrary Thresholds**
20, 15, 10 ng/dL, etc.

Aldosterone Suppression Tests ➡ **Multiple Protocols**
Arbitrary Thresholds

Aldosterone Assays

Immunoassay → LC-MS/MS

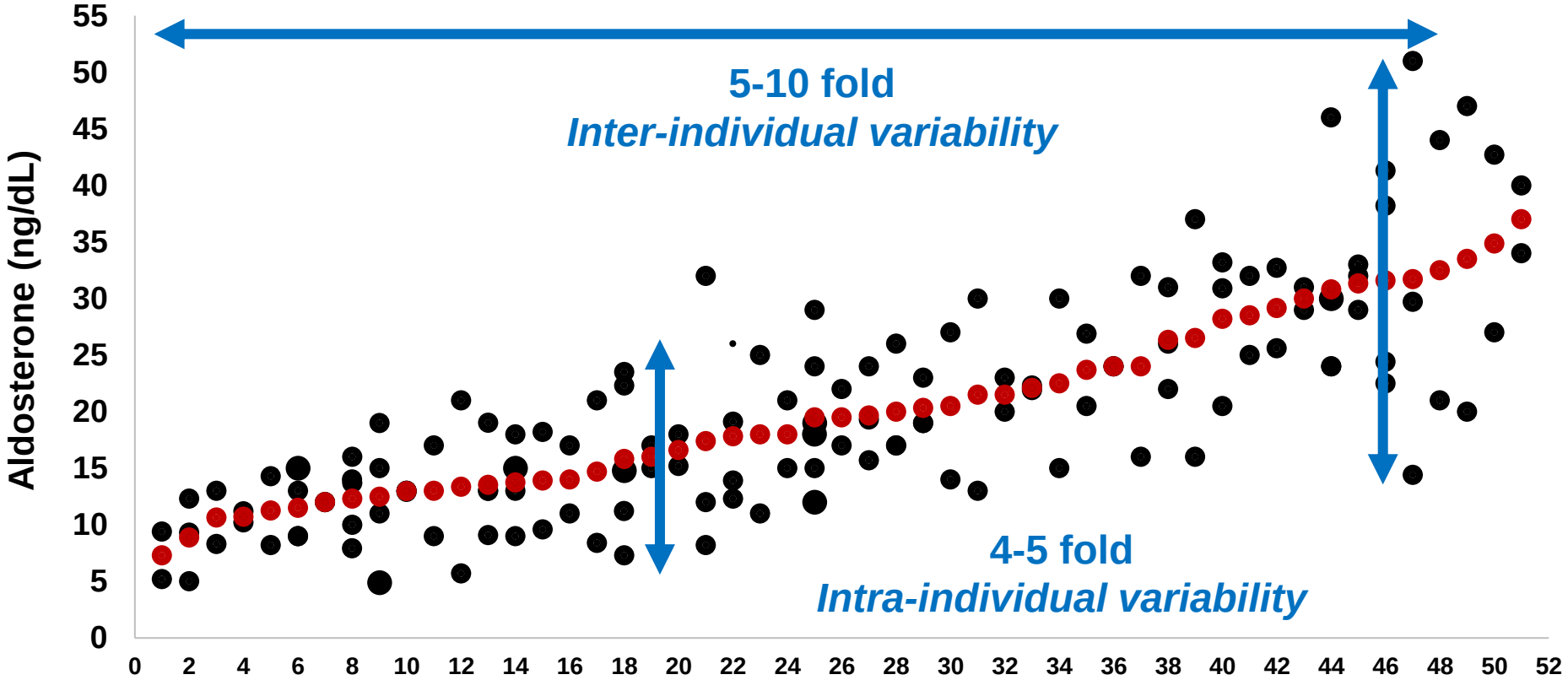


LC-MS/MS aldosterone assays require you to re-calibrate your expectations and interpretations

Brown et al. Journal of the Endocrine Soc 2022

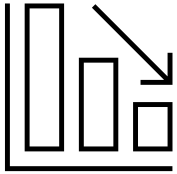
Variability of Aldosterone Production

Variability of Aldosterone Production



Yozamp et al. Hypertension 2021

Variability of Aldosterone Production



ALDO Thresholds

< 15 ng/dL: **49%**

< 10 ng/dL: **29%**

A single aldosterone measurement should not be used to confidently *exclude* the possibility of PA when the pre-test probability is high

Yozamp et al. Hypertension 2021

Over-Reliance on “Confirmatory” Testing

BETTER TERMINOLOGY

“Aldosterone Suppression Tests”

Aldosterone Suppression Tests

1. Oral sodium suppression test
2. Supine Saline suppression test
3. Seated Saline suppression test
4. Captopril challenge test
5. Fludrocortisone suppression test
6. Losartan suppression test
7. Dex-captopril-valsartan suppression test
8. IV Furosemide challenge test
9. Oral furosemide challenge test
10. Upright Posture test
11. ….

Continuum of Primary Aldosteronism

Arbitrary/conventional diagnostic thresholds aside

How common is "*inappropriate, non-suppressible, renin-independent aldosterone production*"
(aka *Primary Aldosteronism Pathophysiology*)?

Aldosterone Suppression Test → **Visualize the spectrum of PA**
(agnostic of conventional thresholds)

Continuum of Primary Aldosteronism

Oral Sodium Suppression Test

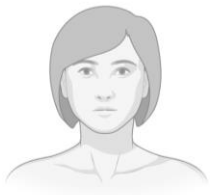
Brown et al. *Annals of Int Med* 2020

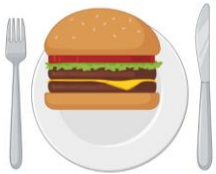
Continuum of Primary Aldosteronism

Oral Sodium Suppression Test

Supplemental Na^+ Intake

~2-4 g/d x 3-4 days





\+

Mean U.S. Dietary Na^+ Intake

~3.5 g/d

Net Summary

~4-6 g/d Na^+ x 3-4d
~1.5 L/d H_2O/NS x 3-4d

Physiologic Expectations

ECV/IVV Expansion
↓Renin
↓AngII
↓**Aldosterone**

Brown et al. *Annals of Int Med* 2020

Continuum of Primary Aldosteronism

CONTINUUM: severity spectrum of non-suppressible, renin/AngII-independent aldosterone production

This is physiologic

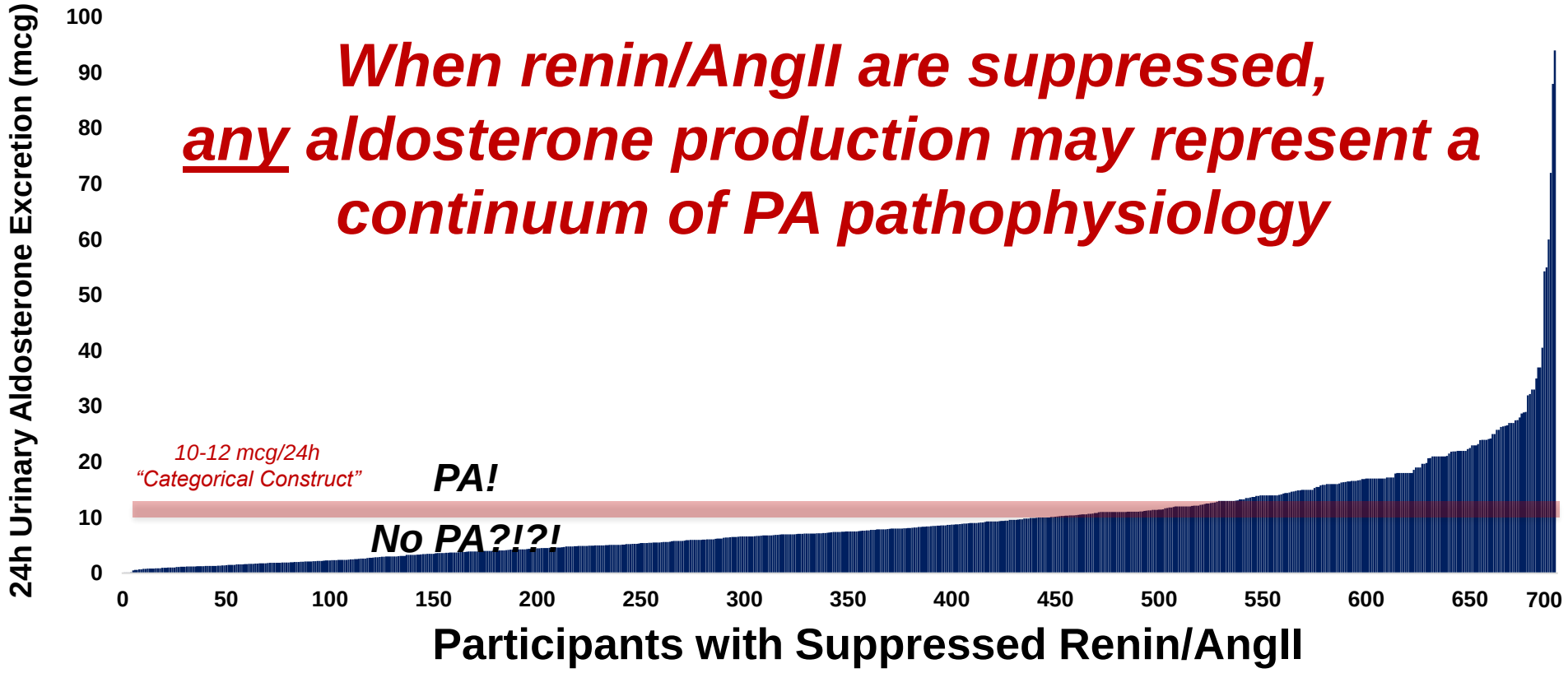
When does physiology end?
When does pathophysiology begin?

This is pathophysiologic (aka overt PA)

Number of Participants with Suppressed Renin/AngII

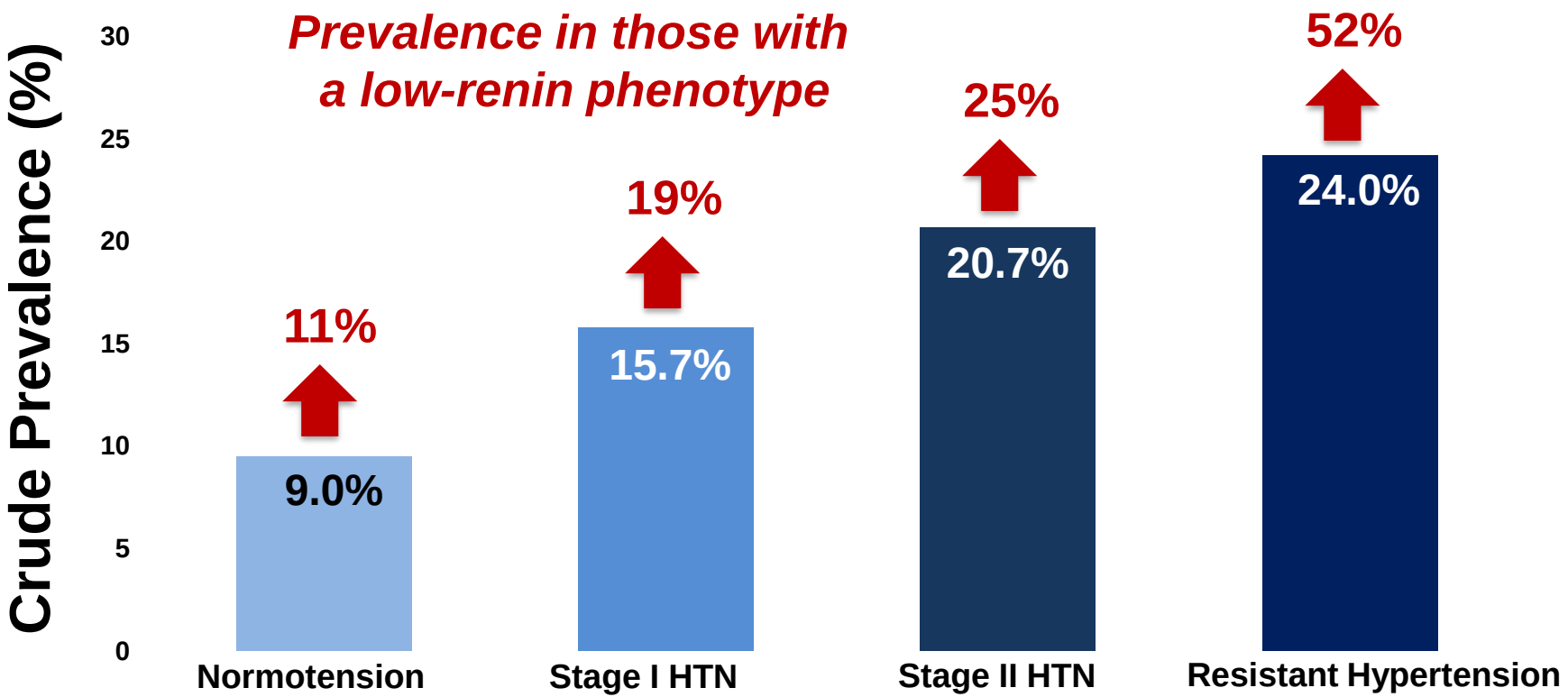
Brown et al. *Annals of Int Med* 2020

Continuum of Primary Aldosteronism



Brown et al. *Annals of Int Med* 2020

The Prevalence of Primary Aldosteronism

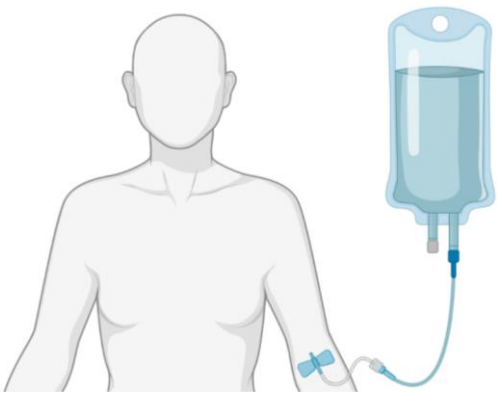


Brown et al. *Annals of Int Med* 2020

Spectrum of PA Pathophysiology

Saline Suppression Test

2L NS (0.9%) over 4 hours



Net Summary

7g Na^+ over 4h (1.8g Na^+/h x 4h)
2L of H_2O

Physiologic Expectations

IV volume Expansion
↓Renin
↓AngII
↓**Aldosterone**

Made with biorender

Parksook et al. JCEM 2024

Spectrum of PA Pathophysiology

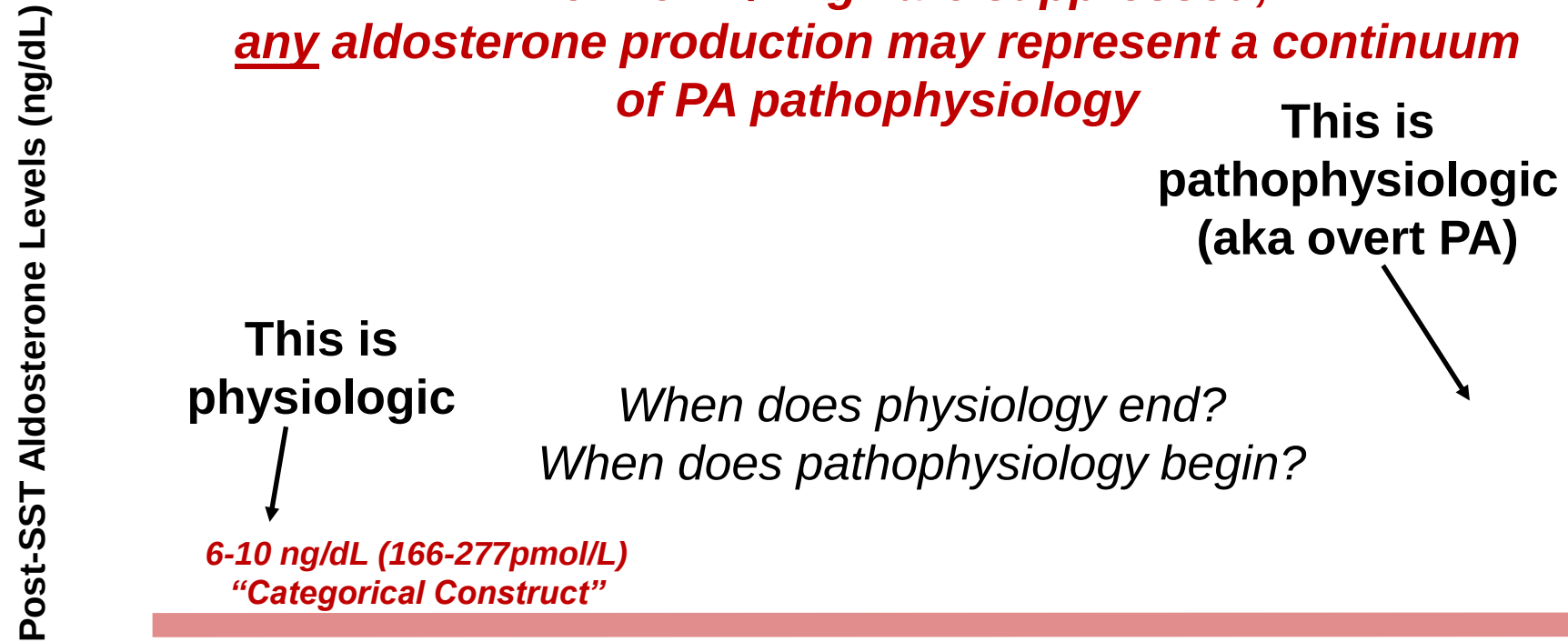
Saline Suppression Test in individuals with a low-renin phenotype

(PRA<1.0 ng/mL/h or DRC<10 mU/L)

Parksook et al. JCEM 2024

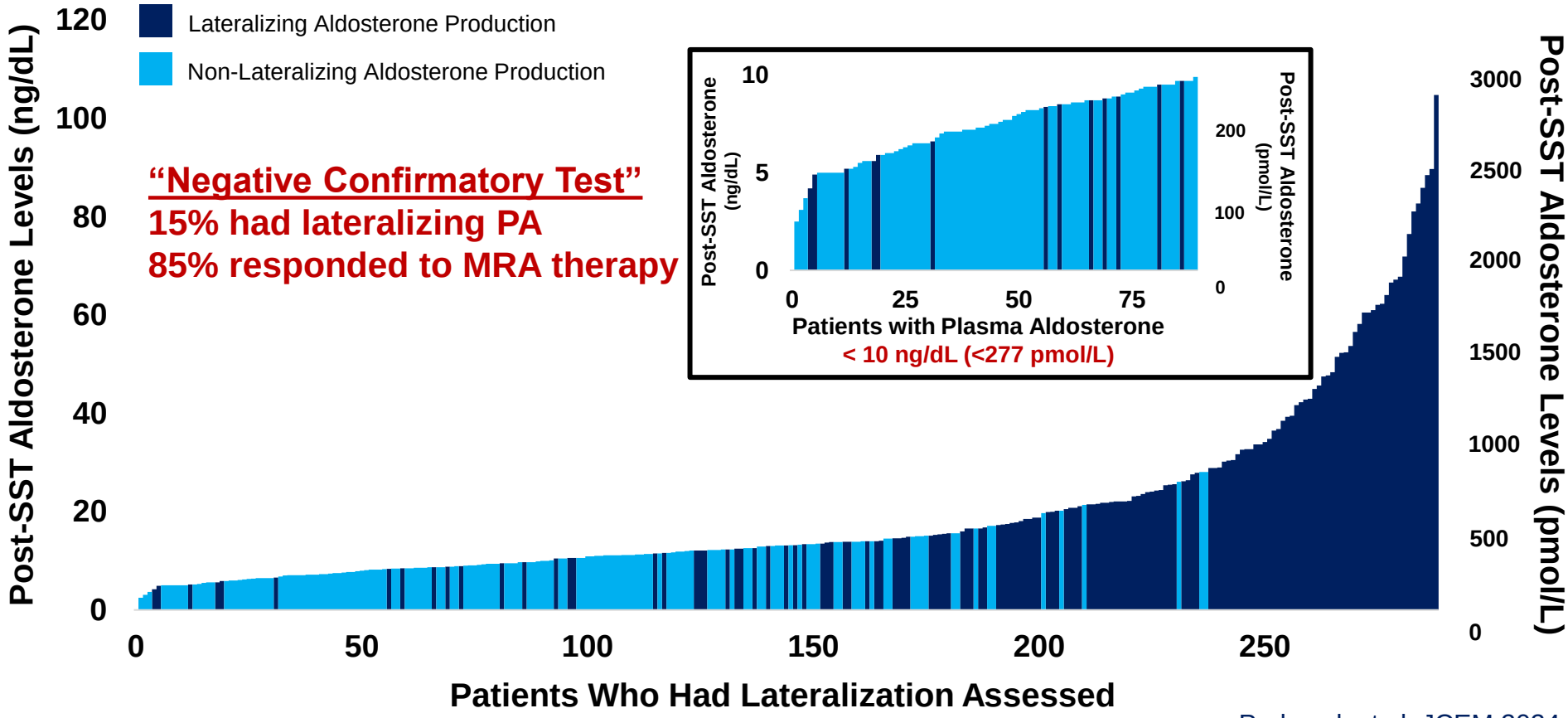
Spectrum of PA Pathophysiology

When renin/AngII are suppressed,
<u>any</u> aldosterone production may represent a continuum
of PA pathophysiology



Parksook et al. JCEM 2024

Spectrum of PA Pathophysiology



Parksook et al. JCEM 2024

Confirmatory Testing Clinical Trial

In a blinded assessment:

"The SSST could not discriminate between treatment response statuses. The positive and negative likelihood ratios were equivocal for aldosterone cutoffs ranging from 140 pmol/L (5 ng/dL) to 300 pmol/L (10 ng/dL)"

"The SSST is associated with a high false-negative rate, and reliance on it may lead to missed opportunities for intervention"

Leung et al. Annals of Int Med 2025

“Confirmatory” Tests

Are better described as “aldosterone suppression tests”

Are not validated or calibrated or reproducible…

They are not accurate diagnostic tests…

They are not accurate at predicting lateralization…

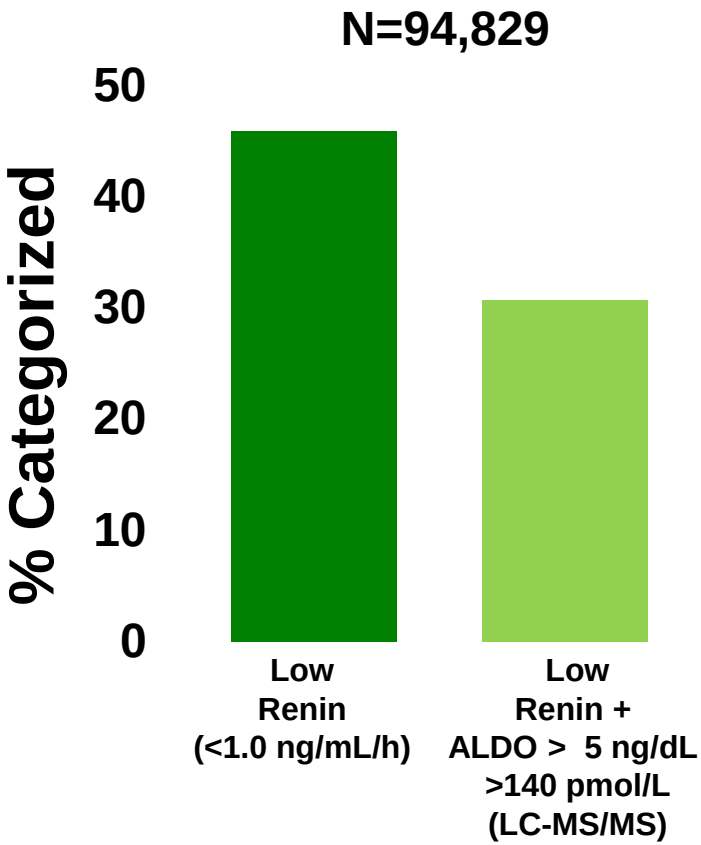
Are labor and resource intensive…

Add little PPV, but contribute to false-negative case detection...

Are NOT necessary or supported by the evidence

Is the continuum of Primary Aldosteronism Pathophysiology clinically relevant?

The Prevalence of Primary Aldosteronism

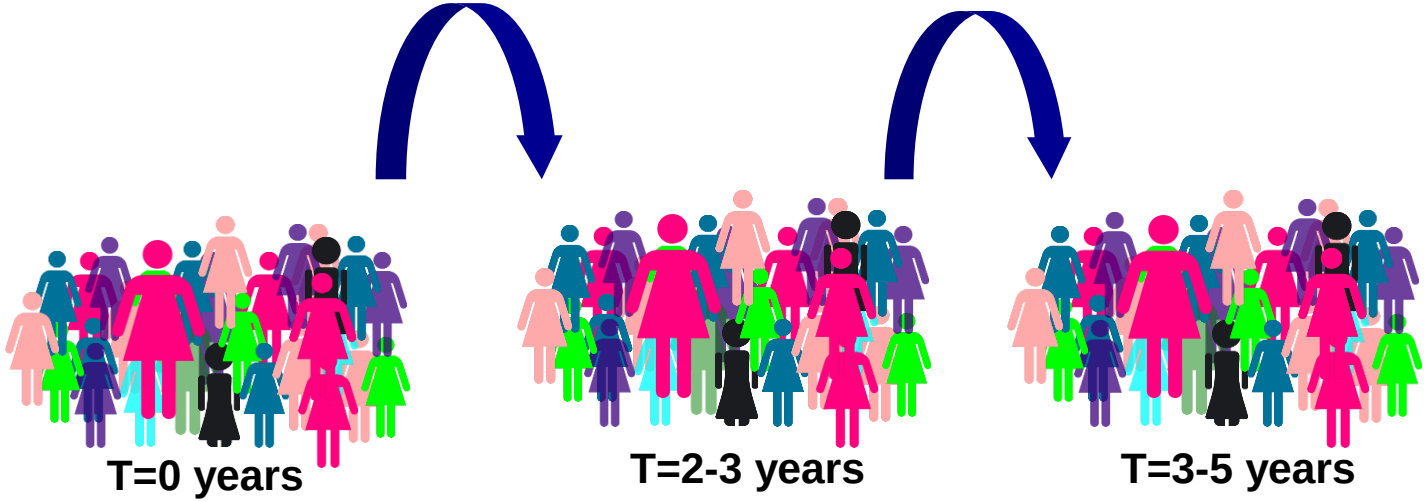


~30% of all hypertensive people have some degree of PA Pathophysiology

(~30% of 1.3 billion)

Marcelli et al. *Hypertension* 2024

Prospective Cohort Studies



Normotensive Cohort Studies

Normotensive

Magnitude of Aldosterone Production when Renin Suppressed

→

Arterial Stiffness
Incident Hypertension
Subclinical Structural CVD
Atrial Fibrillation
MACE

RAINE
Young
Perth, Australia

MESA
Multi-ethnic
Nationwide

FHS
White (mostly)
Framingham, MA

JHS
Black
Jackson, MS

ARIC
Nationwide
USA

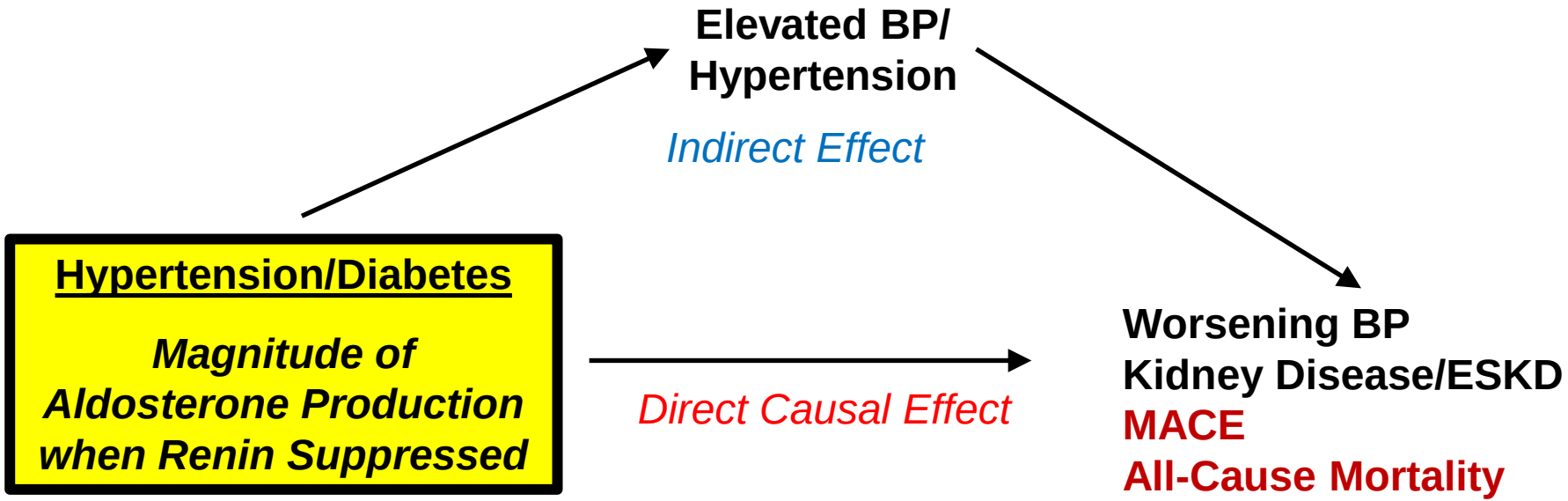
CARTaGENE
Quebec
Canada

Brown et al. *Annals of Internal Medicine* 2017
Brown et al. *Hypertension* 2022

Vasan et al. *NEJM* 2004
Newton-Cheh et al. *Hypertension* 2007

Joseph et al. *Circulation* 2021
Hundemer et al. *Circulation* 2023
Goupil et al. *Circulation* 2025

Hypertensive Cohort Studies



FHS
White (mostly)
Framingham, MA

MESA
Multi-ethnic
USA

JHS
Black
Jackson, MS

CONPASS
Chinese
Chongqing, China

EIMDS
Chinese
Chongqing, China

UK BIOBANK
Nationwide
UK

ARIC
Nationwide
USA

CRIC
Nationwide
USA

CARTaGENE
Quebec
Canada

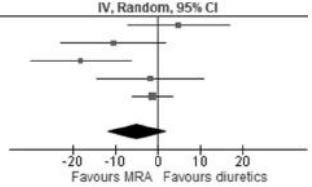
Brown et al. *Annals Int Med* 2017; Inoue et al. *Hypertension* 2020; Joseph et al. *Circulation* 2021; Hu et al. *JAHA* 2021; Brown et al. *Hypertension* 2022; Verma et al *Eur Heart J* 2022; Hu et al Metabolism 2023; Hundemer et al. 2023, Goupil et al. *Circulation* 2025

Meta-Analysis of RCTs in LRH

MRA vs Thiazides (- 4.8 mmHg)

Study or Subgroup	MRA Mean [mmHg]	MRA SD [mmHg]	MRA Total	Diuretic Mean [mmHg]	Diuretic SD [mmHg]	Diuretic Total	Weight	Mean Difference IV, Random, 95% CI	Year
Adlin	-24	15.8	18	-29	15.1	10	17.8%	5.00 [-6.87, 16.87]	1972
Vaughan	-28.3	17.8	21	-17.9	19.7	16	17.2%	-10.40 [-22.69, 1.89]	1973
Spark	-32	11.4	10	-13.9	15.3	10	17.9%	-18.10 [-29.93, -6.27]	1974
Ferguson	-13.1	15.1	11	-11.4	15.1	11	16.8%	-1.70 [-14.32, 10.92]	1977
Hood (bendroflumethiazide)	-11.6	13.8	51	-10.5	10.4	51	30.3%	-1.10 [-5.84, 3.64]	2007
Total (95% CI)			111			98	100.0%	-4.75 [-11.91, 2.40]	

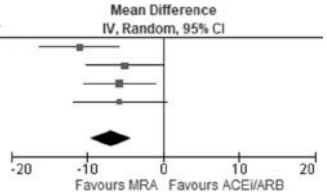
Heterogeneity: Tau² = 38.05; Chi² = 10.05, df = 4 (P = 0.04); I² = 60%
Test for overall effect: Z = 1.30 (P = 0.19)



MRA vs ACEi/ARB (- 6.8 mmHg)

Study or Subgroup	MRA Mean [mmHg]	MRA SD [mmHg]	MRA Total	ACEi/ARB Mean [mmHg]	ACEi/ARB SD [mmHg]	ACEi/ARB Total	Weight	Mean Difference IV, Random, 95% CI	Year
Flack 2003	-16	15.5	60	-5	14.2	62	24.7%	-11.00 [-16.28, -5.72]	2003
Williams 2004	-15.3	15.7	67	-10.3	16	82	26.3%	-5.00 [-10.11, 0.11]	2004
Weinberger 2005	-15.8	15.8	86	-10.1	15.4	82	30.4%	-5.70 [-10.42, -0.98]	2005
Hood 2007 (losartan)	-11.6	13.8	51	-5.9	17.7	51	18.5%	-5.70 [-11.86, 0.46]	2007
Total (95% CI)			264			277	100.0%	-6.83 [-9.56, -4.10]	

Heterogeneity: Tau² = 0.58; Chi² = 3.24, df = 3 (P = 0.36); I² = 7%
Test for overall effect: Z = 4.90 (P < 0.00001)



MRA vs β-blocker (- 4.5 mmHg)
MRA vs α-blocker (- 4.0 mmHg)

Spironolactone versus placebo, bisoprolol, and doxazosin to determine the optimal treatment for drug-resistant hypertension (PATHWAY-2): a randomised, double-blind, crossover trial


ESC European Heart Journal - Cardiovascular Pharmacotherapy ORIGINAL ARTICLE

Spironolactone effect on the blood pressure of patients at risk of developing heart failure: an analysis from the HOMAGE trial

João Pedro Ferreira, Timothy Collier, Andrew L. Clark, Mamas A. Mamas, Hans-Peter Brunner-La Rocca, Stephane Heymans, Arantxa González, Fozia Z. Ahmed, Johannes Petutschnigg, Blerim Mujaj, Joe Cuthbert, Philippe Rouet, Pierpaolo Pellicori, Beatrice Mariottoni, Franco Cosmi, Frank Edelmann, Lutgarde Thijs, Jan A. Staessen, Mark Hazebroek, Job Verdonschot, Patrick Rossignol, Nicolas Girerd, John G. Cleland, and Faiez Zannad

Williams et al. Lancet 2015; Shah et al. JHH 2024

KEY CONCEPT

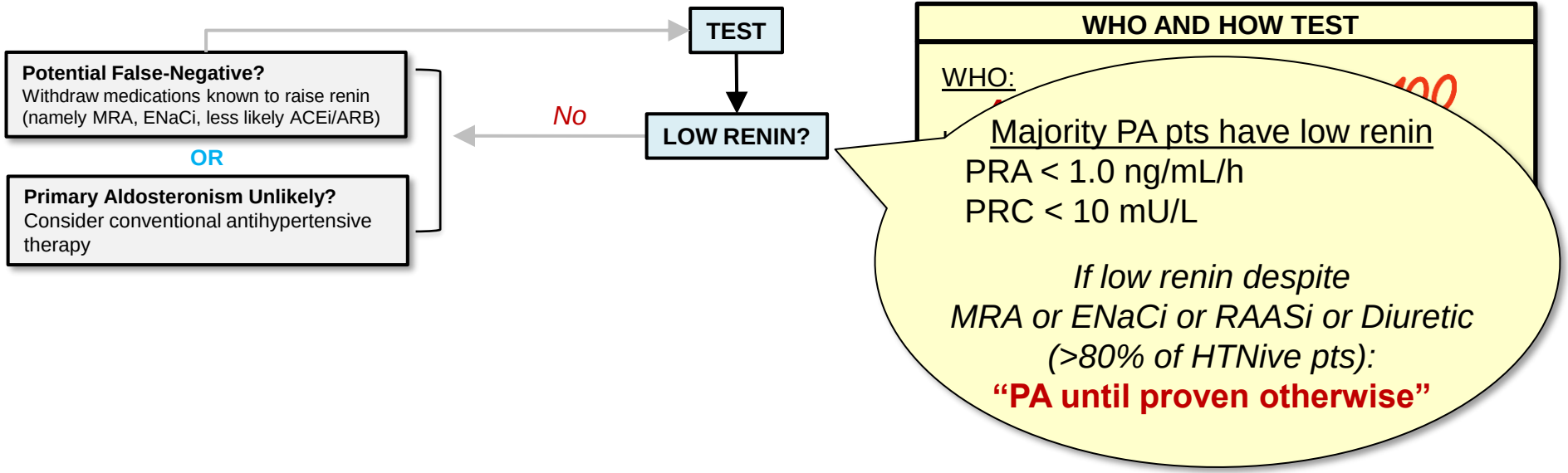
Greater aldosterone production in the context of a low-renin phenotype is associated with progressive risk for CV and kidney disease and responds preferentially to MRA therapy

PA Pathophysiology is *any* aldosterone production when renin is low; there is no lower limit of aldosterone that confidently excludes PA

Re-Calibrating the Diagnostic Approach

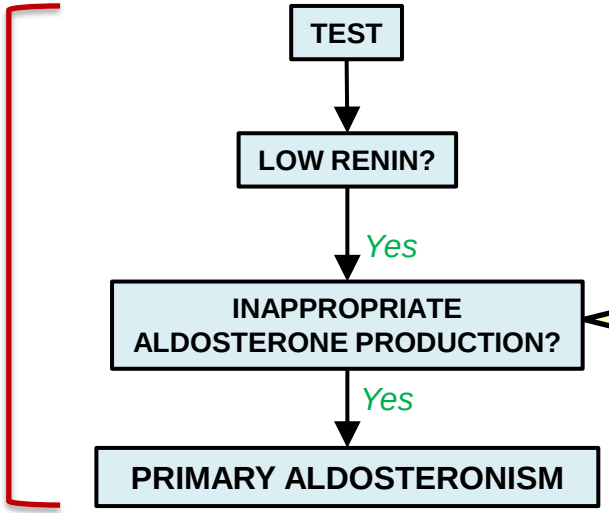
Adopting a new mindset…

"It's Primary Aldosteronism Until Proven Otherwise"



Vaidya et al. NEJM 2025
Adler…Vaidya…et al. JCEM 2025

CENTRAL DOGMA



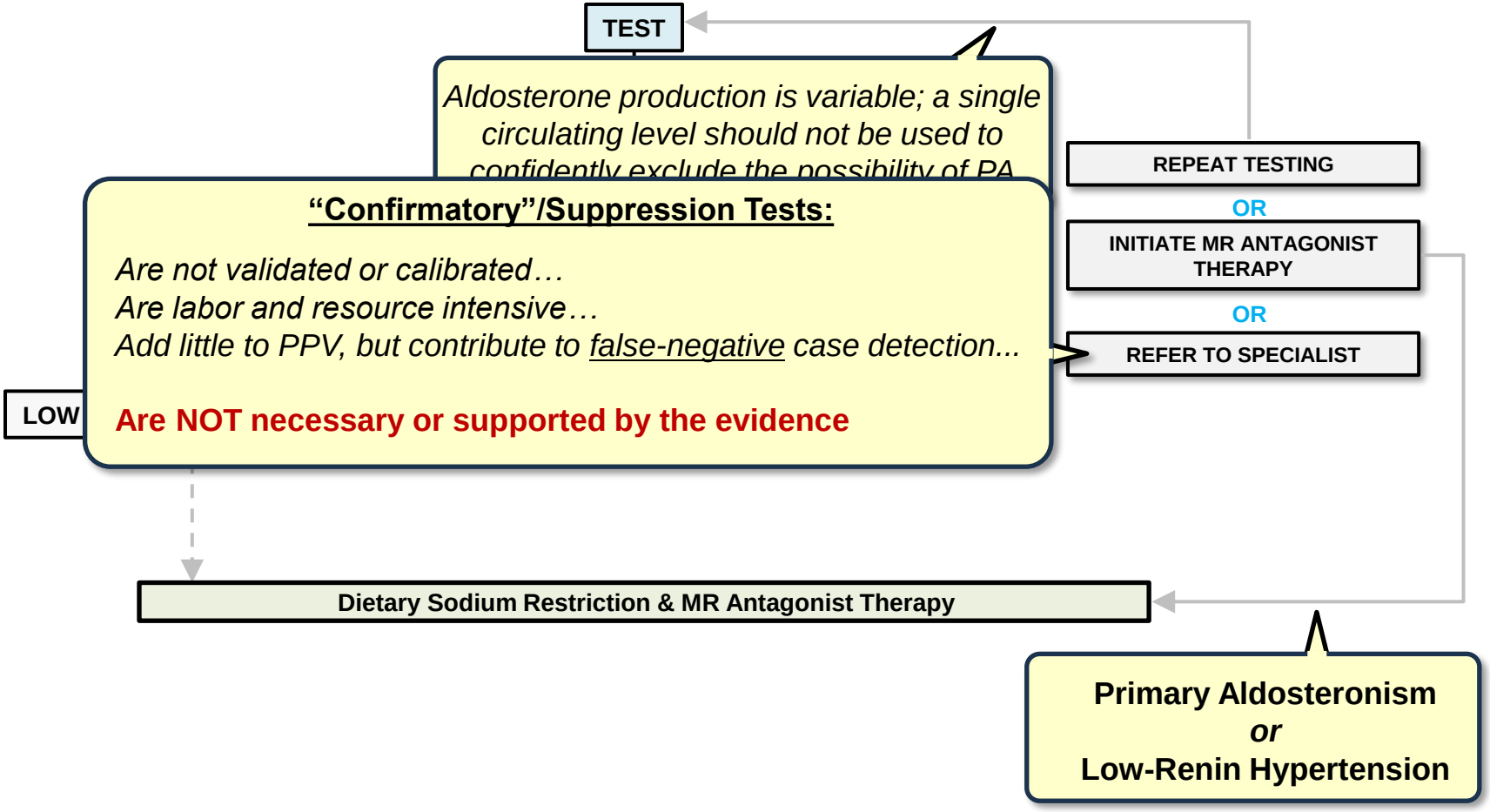
Continuum: *any aldosterone production when renin is low represents PA Pathophysiology amenable to targeted therapy*

Categorical: > 7.5 ng/dL (LC-MS/MS)
> 10 ng/dL (IA)

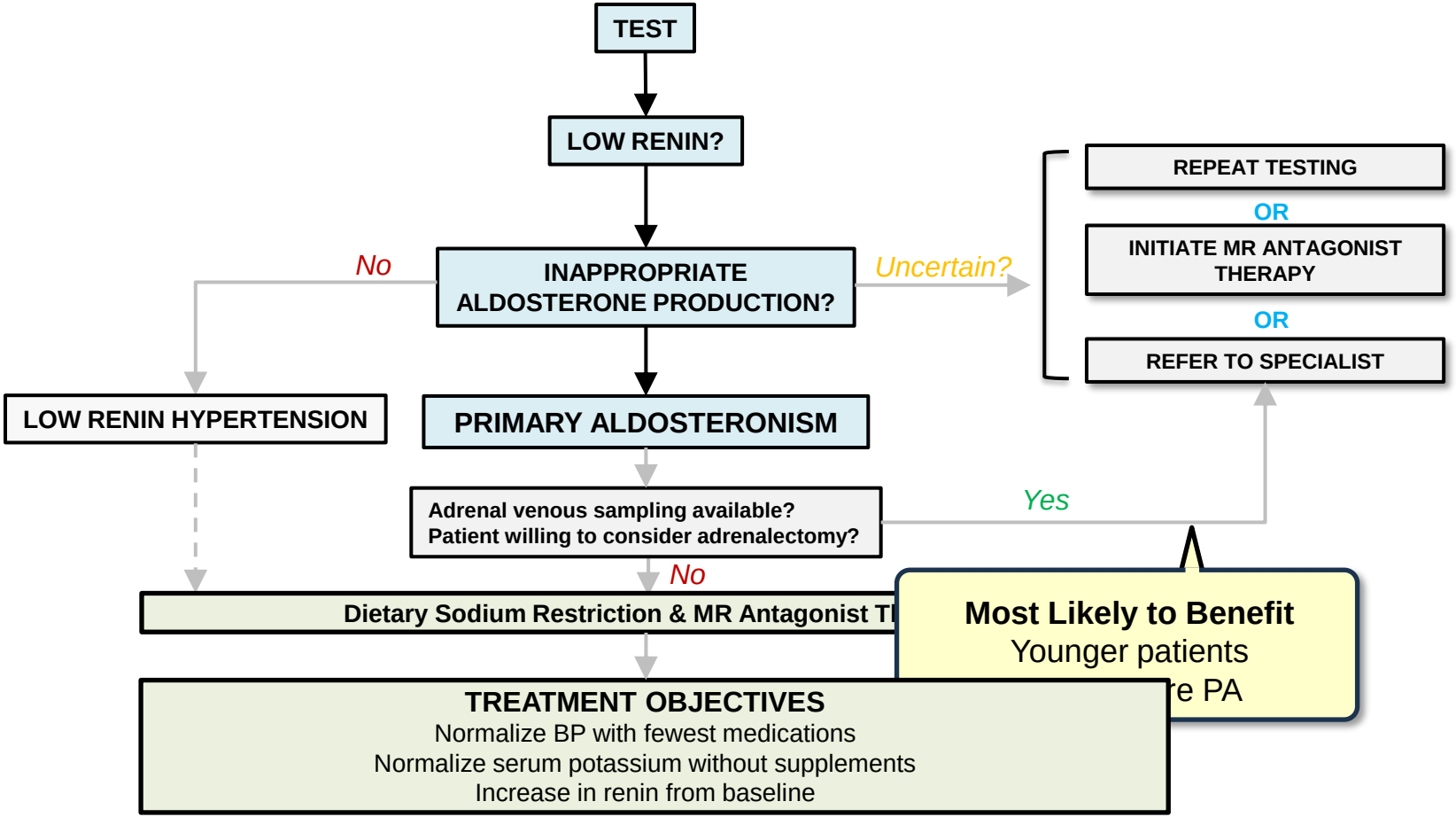
****If on ACEi/ARB (75-80%):*
"PA until proven otherwise"

1. **Liberally test high-risk groups**
2. **Low or persistently suppressed renin is highly indicative**
3. **Any inappropriate aldosterone production when renin is low**

Vaidya et al. NEJM 2025
Adler…Vaidya…et al. JCEM 2025



Vaidya et al. NEJM 2025
Adler…Vaidya…et al. JCEM 2025



Vaidya et al. NEJM 2025
Adler…Vaidya…et al. JCEM 2025

ASIs appear to be a new anti-HTN class, highly effective at lowering aldosterone, and BP in R-HTN, uncontrolled HTN, and PA

They affirm that a large proportion of essential/idiopathic HTN is aldosterone-mediated

"Medical Aldosterone-ectomy" may be the treatment of the future

Krishna Poli, MD; Simon D. Roger, MD; B. T. Slingsby, MD, PhD; Frank Strutz, MD; Liffert Vogt, MD, PhD; Matthew K. Weir, MD; David Rodman, MD;
for the Launch-HTN Investigators

Key Learning Points

Primary aldosteronism is in your clinic every week, but you are almost never recognizing it

Primary aldosteronism is very common; **most low-renin hypertension is a manifestation of Primary Aldosteronism Pathophysiology**

PARADIGM SHIFT

Primary aldosteronism is not a rare cause of "secondary HTN"; rather it is a highly prevalent and modifiable contributor to HTN and CVD

Key Resources

NEJM Primary Aldosteronism Case Overview
Vaidya A, Morlote M, Moussa M, Barletta J, Nehs M.
New England Journal of Medicine 2025; 392(4): e14

2025 Endocrine Society Guidelines on Primary Aldosteronism
Adler G, Stowasser M, Correa R, Khan N, Kline G, McGowan M, Mulatero P, Murad M, Touyz R, Vaidya A, Williams T, Yang J, Young W, Zennaro M, Brito J
JCEM 2025; 110(9): 2453-2495