Why *Think Aldo*?

Erika Jones

Associate Professor of Nephrology and Hypertension

University of Cape Town, South Africa

"Aldosterone plays a key role in driving kidney disease, so it's crucial that we understand its impact and actively address it, especially when treating patients with hypertension or existing kidney disease"

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Roland Schmieder

Professor of Internal Medicine, Nephrology and Hypertension

University Hospital Erlangen, Germany

"Over the past two decades, we have focused on the renin–angiotensin system and developed drugs that protect the heart and kidneys. However, one important contributor to damage, aldosterone, has been forgotten"

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Hirotaka Shibata

Professor and Chairman, Department of Endocrinology, Metabolism, Rheumatology and Nephrology, Faculty of Medicine

Oita University, Japan

"In every hypertensive patient, I consider aldosterone. The role of aldosterone in hypertension is more widely distributed than we expect"

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Rui Jin Hospital, Shanghai Jiao Tong University School of Medicine, China

"I think about aldosterone when treating patients because even moderately elevated aldosterone levels can significantly disrupt blood pressure regulation"

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Professor of Kidney Medicine

University College London, United Kingdom

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Professor of Medicine and Head of the Department of Hypertension and Diabetology

Medical University of Gdansk, Poland

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Michael Böhm

Director of the Klinik für Innere Medizin III and Chief of Cardiology

University of Saarland, Germany

"Aldosterone comes to mind when I treat patients with heart failure. Research has shown that aldosterone contributes to fibrosis, disrupted homeostasis, and vascular remodelling in the heart and kidneys"

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Hear from some of our experts on why they think about aldosterone in their research and in clinical practice

Think Aldo

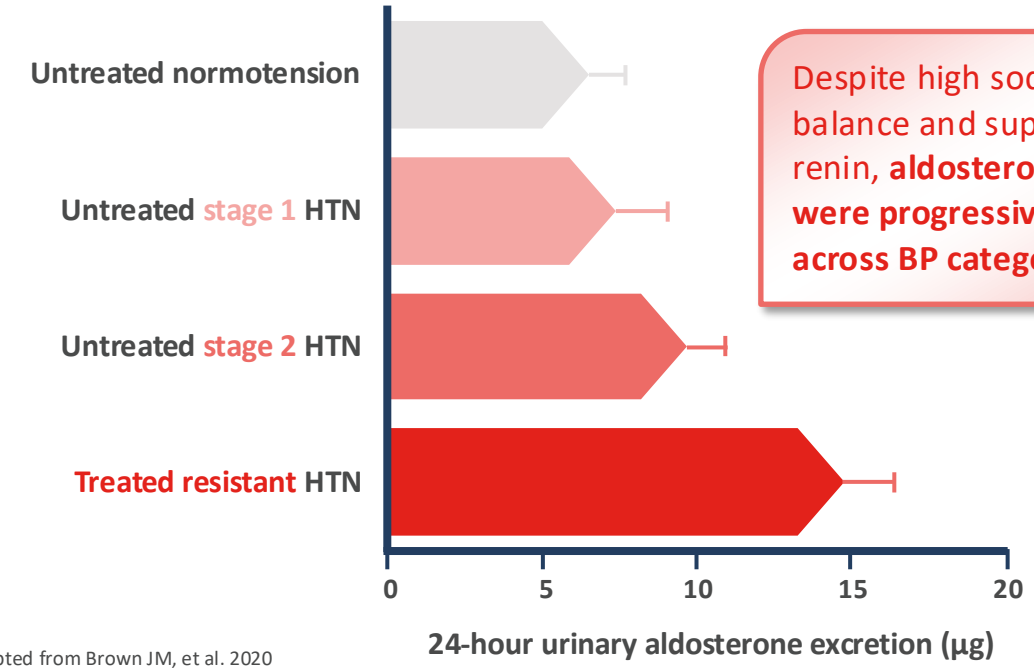
The above comments reflect the personal clinical experience and professional opinion of the healthcare professional. They are provided for educational discussion and should not be interpreted as definitive scientific evidence.

This non-promotional educational infographic has been developed and funded by AstraZeneca, in collaboration with Think Aldo Steering Committee members.
Intended for healthcare professionals outside of the US only.
Veeva ID: Z4-82791 | Date of preparation: April 2026

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker

Why *Think Aldo*?

There is a prevalent continuum of aldosterone dysregulation that parallels the severity of hypertension[a]



Despite high sodium balance and suppressed renin, **aldosterone levels were progressively higher across BP categories**[b]

Figure adapted from Brown JM, et al. 2020

Hirotaka Shibata

Professor and Chairman, Department of Endocrinology, Metabolism, Rheumatology and Nephrology, Faculty of Medicine

Oita University, Japan

"In every hypertensive patient, I consider aldosterone. The role of aldosterone in hypertension is more widely distributed than we expect"

[a]Based on a cross-sectional study that pooled five study protocols from four distinct sites across the United States. Participants with untreated normotension (n=289), untreated stage 1 HTN (n=115), untreated stage 2 HTN (n=203) and treated resistant HTN (n=408); [b]Adjusted for age, BMI, race, sex, history of diabetes and 24-hour urinary sodium excretion

BMI, body mass index; BP, blood pressure; HTN, hypertension

Brown JM, et al. *Ann Intern Med* 2020;173:10–20

Why *Think Aldo*?

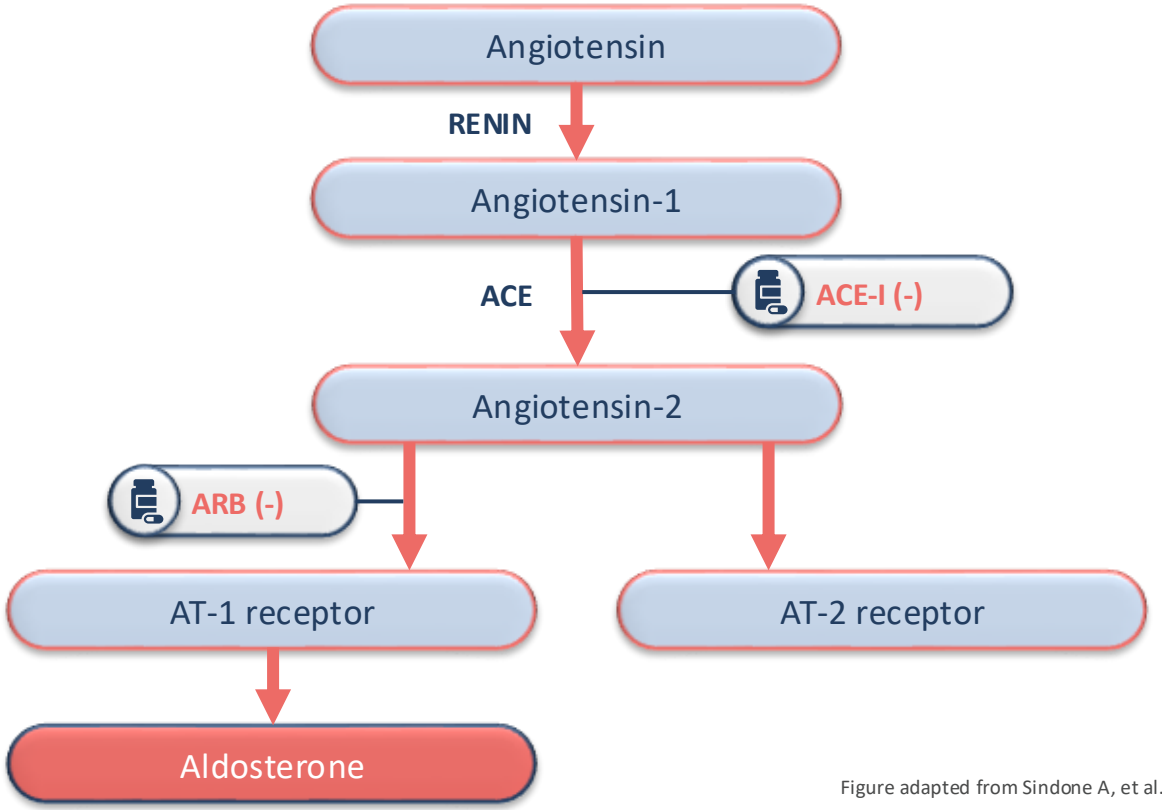


Figure adapted from Sindone A, et al. 2013

David Wheeler

Professor of Kidney Medicine

University College London, United Kingdom

"I think about aldosterone when I prescribe ACEi and ARBs because these medications inhibit the actions of the hormone downstream"

ACE(i), angiotensin-converting enzyme (inhibitor); ARB, angiotensin receptor blocker; AT, angiotensin II; RAAS, renin–angiotensin–aldosterone system

Sindone A, et al. *Aust Fam Physician* 2013;42:634–638

Why *Think Aldo*?

Elevated aldosterone is associated with an increased risk of CKD progression and all-cause mortality

45%
CKD progression[a–d]
Higher risk in patients in the highest vs lowest quartile of serum aldosterone

46%
ESKD development[a,d]
Higher risk in patients in the highest vs lowest quartile of serum aldosterone

22%
All-cause mortality[e]
Higher risk in patients in the highest vs lowest quartile of serum aldosterone

ELEVATED ALDOSTERONE LEVELS

Erika Jones
Associate Professor of Nephrology and Hypertension
University of Cape Town, South Africa

"Aldosterone plays a key role in driving kidney disease, so it's crucial that we understand its impact and actively address it, especially when treating patients with hypertension or existing kidney disease"

[a]Results depicted are from a multicentre, prospective, observational cohort study of 3680 patients with known CKD, defined as eGFR 20–70 mL/min/1.73 m^2, over a median follow-up of 9.6 years. Results were analysed using a multivariable model adjusted for age, sex, race, clinical centre, BMI, diabetes mellitus, SBP, history of CV disease, HbA_{1c}, history of hypercholesterolaemia, serum albumin, serum potassium, ACEi/ARB use, loop diuretic use, statin, smoking, BNP, 24-hour urine sodium, 24-hour urine potassium, 24-hour urine protein and baseline eGFR; [b]CKD progression was defined as the composite of 50% decline in eGFR or incident ESKD; [c]Risk of CKD progression=HR, 1.45; 95% CI, 1.22–1.73 (p=0.001) for those in the highest quartile of serum aldosterone relative to lowest quartile; [d]Risk of developing ESKD=HR, 1.46; 95% CI, 1.19–1.78 (p=0.02) for those in the highest quartile of serum aldosterone relative to lowest quartile; [e]Higher risk of all-cause mortality in those in the highest quartile of aldosterone compared with those in the lowest quartile

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index; BNP, B-type natriuretic peptide; CI, confidence interval; CKD, chronic kidney disease; CV, cardiovascular; eGFR, estimated glomerular filtration rate; ESKD, end-stage kidney disease; HbA_{1c}, glycated haemoglobin; HTN, hypertension; HR, hazard ratio; OR, odds ratio; SBP, systolic blood pressure

Verma A, et al. *Eur Heart J* 2022;43:3781–3791

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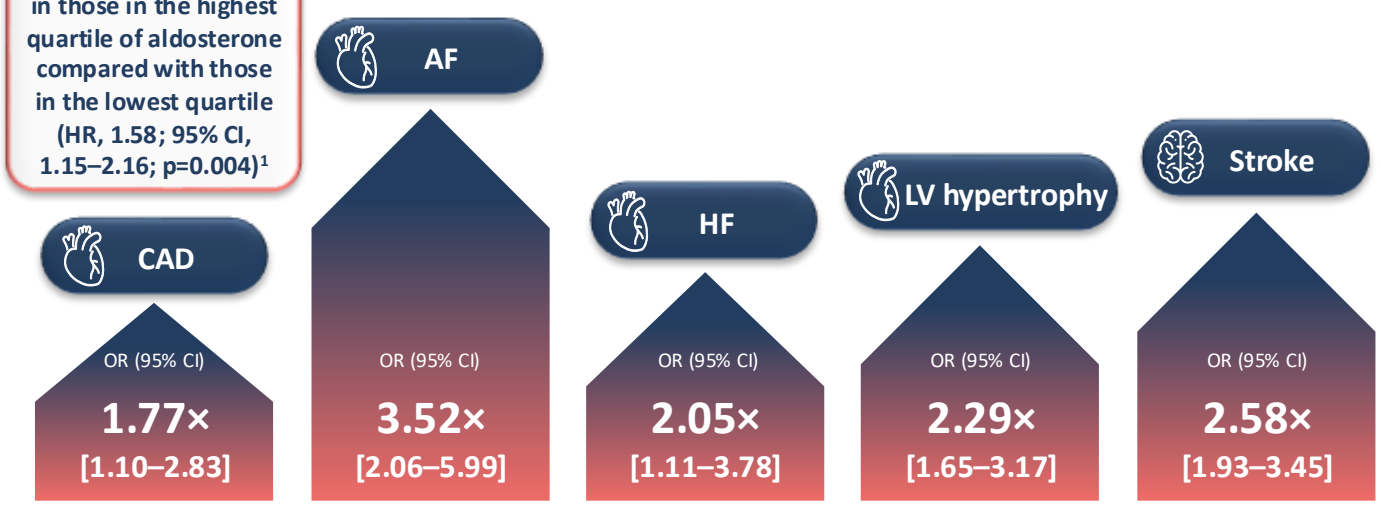
Why *Think Aldo*?

Aldosterone dysregulation is associated with an increased risk of CV disease[a,b]

58% ↑ in risk of CV mortality in those in the highest quartile of aldosterone compared with those in the lowest quartile (HR, 1.58; 95% CI, 1.15–2.16; p=0.004)[1]



ALDOSTERONE DYSREGULATION[2]

Michael Böhm

Director of the Klinik für Innere Medizin III and Chief of Cardiology

University of Saarland, Germany

"Aldosterone comes to mind when I treat patients with heart failure. Research has shown that aldosterone contributes to fibrosis, disrupted homeostasis, and vascular remodelling in the heart and kidneys."

[a]Meta-analysis of 31 studies that evaluated CV risk in patients with primary aldosteronism (n=3838) compared with patients with essential HTN (n=9284). Median duration of HTN was 8.8 years (IQR, 6.2–10.7); [b]OR (95% CI) for the outcomes of coronary artery disease=1.77 (1.10–2.83), atrial fibrillation=3.52 (2.06–5.99), heart failure=2.05 (1.11–3.78) and stroke=2.58 (1.93–3.45)

AF, atrial fibrillation; CAD, coronary artery disease; CI, confidence interval; CV, cardiovascular; HF, heart failure; HR, hazard ratio; HTN, hypertension; IQR, interquartile range; LV, left ventricular; OR, odds ratio

1. Tomaschitz A, et al. *Eur Heart J* 2010;31:1237–1247; 2. Monticone S, et al. *Lancet Diabetes Endocrinol* 2018;6:41–50

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Why *Think Aldo*?

Aldosterone's effects extend beyond electrolyte and BP regulation by causing end-organ damage, which may occur independent of BP

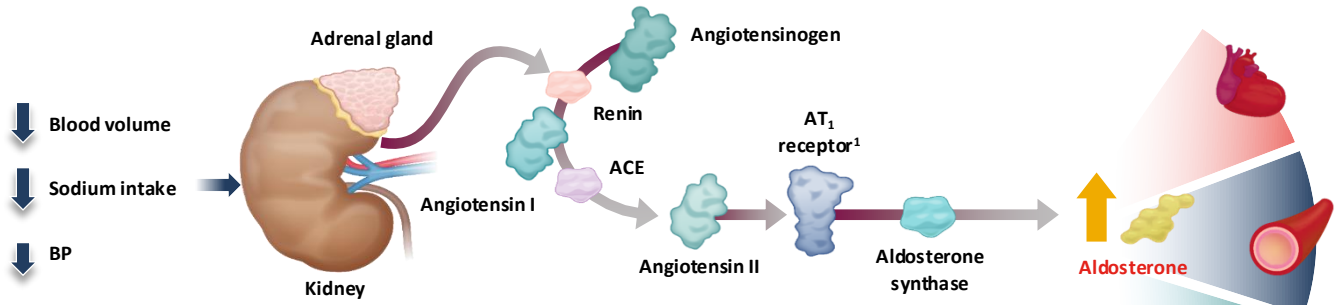

Figure adapted from Ekman N, et al. 2025

Aldosterone-mediated effects:

Heart[2]
- Ischaemia
- Hypertrophy
- Fibrosis
- Heart failure
- Arrythmias

Vasculature[2]
- Fibrosis
- Inflammation
- Necrosis
- Hypertrophy
- Endothelial dysfunction

Kidneys[2]
- Proteinuria
- Fibrosis
- Ischaemia
- Sclerosis
- Chronic renal failure

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ACE, angiotensin-converting enzyme; AT, angiotensin II; BP, blood pressure

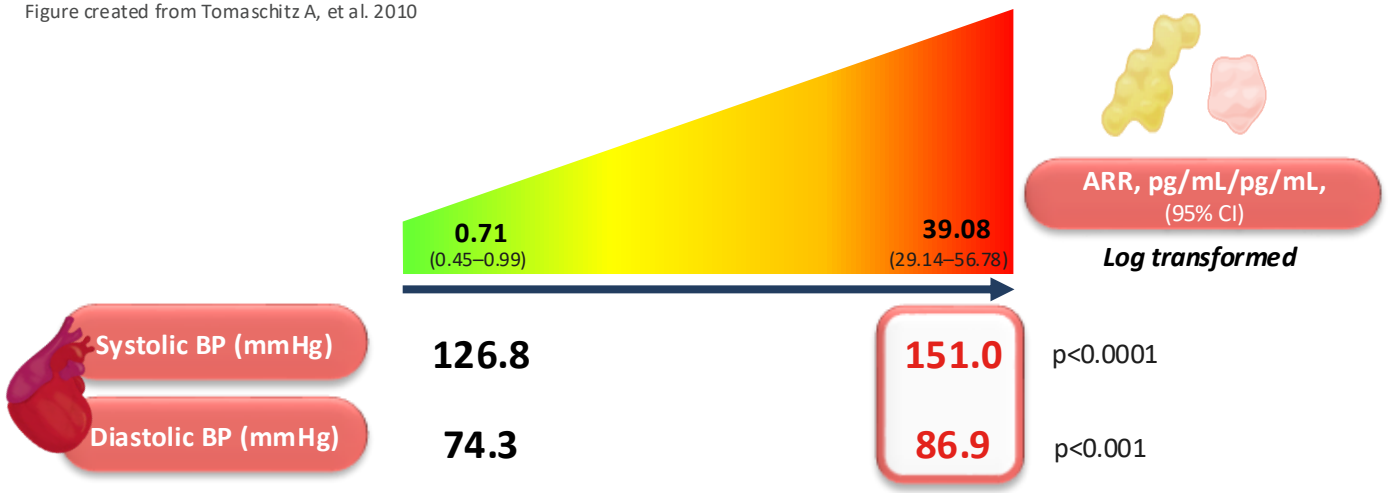
1. Ekman N, et al. *Int J Mol Sci* 2025;26:540; 2. Verhovez A et al. *Curr Signal Transduct Ther* 2012;7(2):132–141

Why *Think Aldo*?

Higher ARR values are paralleled by continuously and progressively higher BP

Inappropriately elevated aldosterone levels exert an effect on BP values and were the most important, and second-most important predictors of SBP and DBP, respectively[a]

Figure created from Tomaschitz A, et al. 2010



ARR affects BP well below a cutoff used for screening for primary aldosteronism

Jiguang Wang
Director of the Department of Hypertension
Rui Jin Hospital, Shanghai Jiao Tong University School of Medicine, China

"I think about aldosterone when treating patients because even moderately elevated aldosterone levels can significantly disrupt blood pressure regulation"

[a]Elevated aldosterone emerged as the most important predictor of SBP and the second-most important predictor of DBP in multivariable models. Continuous variables were log-transformed where appropriate. Variables excluded from the SBP model: aspirin/NSAIDs, beta blockers, cystatin C, plasma cortisol, daily physical activity, current smoking, and serum potassium (R^2 without log ARR = 0.218). Variables excluded from the DBP model: ARBs, beta blockers, aspirin/NSAIDs, nitrates, calcium-channel blockers, cystatin C, serum potassium, plasma cortisol, daily physical activity, and type 2 diabetes mellitus (R^2 without log ARR = 0.147).

ARR, aldosterone:renin ratio; BP, blood pressure; CI, confidence interval; DBP, diastolic blood pressure; SBP, systolic blood pressure

Tomaschitz A, et al. *J Am Coll Cardiol* 2010;55:2171–2180

Why *Think Aldo*?

Emerging evidence reveals that aldosterone dysregulation exists along a broader continuum of renin-independent aldosterone excess[1,2]

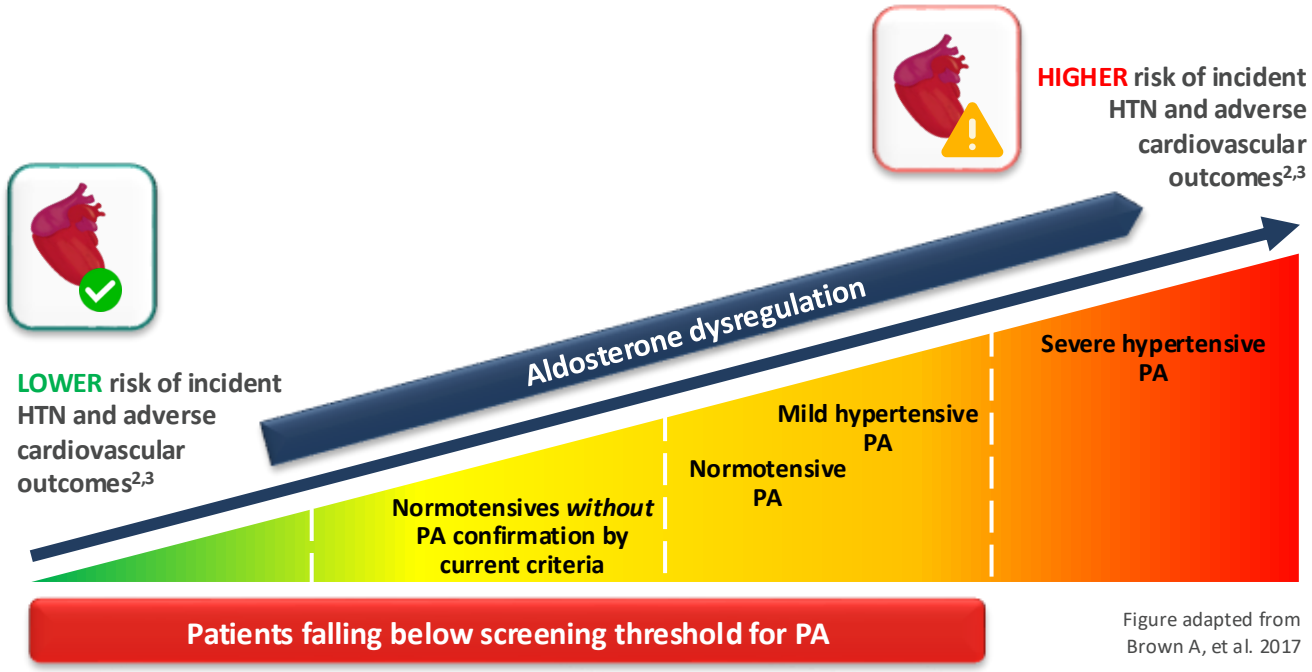


Figure adapted from Brown A, et al. 2017

Krzysztof Narkiewicz

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Medical University of Gdansk, Poland

"Just as we recognise a cardiovascular continuum, there is also a continuum of aldosterone dysregulation that likely plays an important role in many patients"

Figure adapted from Brown JM, et al. 2017
HTN, hypertension; PA, primary aldosteronism

1. Brown JM, et al. *Ann Intern Med* 2017;167:630–641; 2. Brown JM, et al. *Ann Intern Med* 2020;173:10–20;
3. Goupil R, et al. *Circulation* 2025. doi: 10.1161/CIRCULATIONAHA.124.073507 [Online ahead of print]