2027 Australian Guideline for the Diagnosis and Management of Elevated Blood Pressure and Hypertension in Adults

PUBLIC CONSULTATION NOTE

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Please read it as a draft in every sense. You will find typographical and spelling errors, abbreviations defined more than once or not at all, inconsistent terminology and headings, incomplete cross-references and reference formatting, and sections still in progress. Tables, figures and graphics are working versions only — all will be rebuilt and professionally designed for publication, so layout, colour and styling are not indicative of the final product.

What we are seeking at this stage is your view on the content: whether the evidence has been accurately and fairly represented, whether the recommendations follow from that evidence, whether anything important is missing, and whether anything could be misread in practice. Feedback of that kind is far more valuable to us than corrections to spelling or formatting, which will be resolved at copyediting and design.

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First Nations peoples is the term used throughout this guideline, based on the guidance of the Heart Foundation's First Nations Heart Health team. However, the Heart Foundation recognises that the preferred term(s) when writing for, and about, First Nations peoples can differ between communities and individuals. No disrespect is intended to First Nations peoples who may identify with an alternative term.

About this guideline

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Disclosures

For a full list of disclosures/conflicts of interest, refer to Supplementary material.

Abbreviations and acronyms

Abbreviation	Full term	Abbreviation	Full term
ABI	Ankle-brachial index	**HbA1c**	Glycated haemoglobin
ABPM	Ambulatory blood pressure monitoring	**HBPM**	Home blood pressure monitoring
ACC	American College of Cardiology	**HDL**	High-density lipoprotein
ACE	Angiotensin-converting enzyme	**HDL-C**	High-density lipoprotein cholesterol
ACEi	Angiotensin-converting enzyme inhibitor	**HFpEF**	Heart failure with preserved ejection fraction
ACR	Albumin–creatinine ratio	**HMOD**	Hypertension-mediated organ damage
AF	Atrial fibrillation	**IRIS**	Indigenous Risk Impact Screen
AGREE II	Appraisal of Guidelines for Research and Evaluation II	**ISH**	Isolated systolic hypertension
AHA	American Heart Association	**LC-MS/MS**	Liquid chromatography–tandem mass spectrometry
AKI	Acute kidney injury	**LDL**	Low-density lipoprotein
ALARA	As low as reasonably achievable	**Lp(a)**	Lipoprotein(a)
AOBP	Automated office blood pressure	**MACE**	Major adverse cardiovascular events
ARB	Angiotensin receptor blocker	**MBS**	Medicare Benefits Schedule
ARR	Aldosterone-renin ratio	**MI**	Myocardial infarction
ASCVD	Atherosclerotic cardiovascular disease	**mm Hg**	Millimetres of mercury
ASI	Aldosterone synthase inhibitor	**MR**	Mineralocorticoid receptor
AUD	Australian dollar	**MRA**	Mineralocorticoid receptor antagonist
AUDIT-C	Alcohol Use Disorders Identification Test-Consumption	**MRI**	Magnetic resonance imaging
BMI	Body mass index	**NICE**	National Institute for Health and Care Excellence

Abbreviation	Full term	Abbreviation	Full term
BP	Blood pressure	**NSAID**	Non-steroidal anti-inflammatory drug
BPLTTC	Blood Pressure Lowering Treatment Trialists' Collaboration	**OSA**	Obstructive sleep apnoea
CAC	Coronary artery calcium	**PA**	Primary aldosteronism
CALD	Culturally and linguistically diverse	**PBS**	Pharmaceutical Benefits Scheme
CBT	Cognitive behavioural therapy	**PCP**	Phencyclidine
CCB	Calcium channel blocker	**PCR**	Protein–creatinine ratio
CHD	Coronary heart disease	**PICO**	Patient, Intervention, Comparison, Outcome
CI	Confidence interval	**PRA**	Plasma renin activity
CKD	Chronic kidney disease	**RAAS**	Renin–angiotensin–aldosterone system
COX-2	Cyclooxygenase-2	**RACGP**	Royal Australian College of General Practitioners
CPAP	Continuous positive airway pressure	**RAS**	Renin–angiotensin system
CT	Computed tomography	**RCT**	Randomised controlled trial
CTCA	CT coronary angiography	**RDN**	Renal denervation
CV	Cardiovascular	**RNA**	Ribonucleic acid
CVD	Cardiovascular disease	**RR**	Relative risk
DASH	Dietary Approaches to Stop Hypertension	**SBP**	Systolic blood pressure
DBP	Diastolic blood pressure	**SGLT2**	Sodium-glucose cotransporter-2
DRC	Direct renin concentration	**SOMANZ**	Society of Obstetric Medicine of Australia and New Zealand
ECG	Electrocardiogram	**SPC**	Single-pill combination
eGFR	Estimated glomerular filtration rate	**SR**	Systematic review
ENaC	Epithelial sodium channel	**T4**	Thyroxine
ESC	European Society of Cardiology	**TC**	Total cholesterol
ESG	Expert Steering Group	**TGA**	Therapeutic Goods Administration
ESH	European Society of Hypertension	**TIA**	Transient ischaemic attack
FEC	Free equivalent combination	**TIS**	Translating and Interpreting Service
FH	Familial hypercholesterolaemia	**TKI**	Tyrosine kinase inhibitor
GDP	Gross domestic product	**TSH**	Thyroid-stimulating hormone

Abbreviation	Full term	Abbreviation	Full term
GIP	Glucose-dependent insulinotropic polypeptide	**uACR**	Urine albumin–creatinine ratio
GLP-1	Glucagon-like peptide-1	**uPCR**	Urine protein–creatinine ratio
GP	General practitioner	**VEGF**	Vascular endothelial growth factor
GRADE	Grading of Recommendations Assessment, Development and Evaluation	**WHO**	World Health Organization
GRADE-ADOLOPMENT	GRADE Adopt, Adapt, or De Novo development framework		

Glossary

Term	Definition
Ambulatory blood pressure monitoring (ABPM)	A method using a fully automated device worn by the patient to record blood pressure automatically at preset intervals (recommended every 15–30 min during daytime and 20–60 min during night-time) over a usual 24-hour period, capturing daytime, night-time and full 24-hour averages. It is described in the guideline as the reference standard for confirming the diagnosis of hypertension because it provides more readings than office measurement, reduces the impact of short-term BP variability, and is the preferred method for detecting white-coat and masked hypertension, nocturnal hypertension, and abnormal nocturnal dipping.
Automated office blood pressure (AOBP)	A standardised method for measuring blood pressure in a clinical setting: an automated, validated device takes multiple readings (typically three at 30-second intervals) after 5 minutes of seated rest while the patient rests alone, ideally unattended, with the average used as the representative reading.
Clinically determined high risk	A classification applied when an individual has a condition that confers high cardiovascular risk in its own right, so that a calculated risk score is not required and comprehensive risk-factor management proceeds directly. In this guideline the qualifying features are established atherosclerotic CVD; heart failure (including HFpEF) or atrial fibrillation; moderate to severe chronic kidney disease; familial hypercholesterolaemia or markedly elevated cholesterol; severe hypertension; hypertension-mediated organ damage; early-onset type 1

	diabetes; and severe obesity with additional cardiometabolic risk factors.
Clinician	Used throughout this guideline as a generic term for any health professional providing care within their scope of practice, including general practitioners, nurses and nurse practitioners, pharmacists, Aboriginal and Torres Strait Islander Health Practitioners and Health Workers, and allied health professionals. It recognises that in many communities, particularly regional, rural and remote areas, these professionals are often the first point of contact.
CVD risk	The estimated probability (expressed as a 5-year percentage) that an individual will experience a cardiovascular event, calculated using a validated multivariable risk calculator (in this guideline, *the Australian CVD Risk Calculator*) rather than any single risk factor in isolation. The guideline stratifies adults into low (<5%), intermediate (5% to <10%), and high (≥10%) 5-year risk categories, which in turn determine blood-pressure treatment thresholds and intensity of risk-factor management.
Deprescribing	A planned, monitored reduction in dose or cessation of antihypertensive medication, considered in frail or older adults when the original rationale for tight blood pressure control no longer clearly outweighs risks such as polypharmacy, falls and hypotension.
Dose titration/escalation	The process of adjusting antihypertensive treatment intensity to reach target blood pressure.
Elevated blood pressure	A standard office blood pressure of 120–139 mm Hg systolic and/or 80–89 mm Hg diastolic. Cardiovascular risk increases progressively across this range rather than beginning at a threshold, and management is determined by overall CVD risk assessment rather than the blood pressure category alone.
GRADE (Grading of Recommendations Assessment, Development and Evaluation)	A structured methodology used to rate the certainty of evidence and formulate recommendations by balancing benefits and harms, patient values and preferences, and resource use; recommendations are labelled strong or conditional depending on the certainty of evidence and the anticipated impact of the intervention.
GRADE-ADOLOPMENT	The specific framework used to develop this guideline, allowing the writing group to adopt, adapt, or create new (de novo)

	recommendations from existing high-quality international guidelines and evidence syntheses, rather than starting evidence review entirely from scratch.
Home blood pressure monitoring (HBPM)	Repeated self-measurement of blood pressure by the patient at home using a validated, automated upper-arm blood pressure device and a standardised protocol.
Hypertension	A standard office blood pressure of ≥140 mm Hg systolic and/or ≥90 mm Hg diastolic, confirmed on averaged readings taken on at least two separate occasions (or a single reading where blood pressure is ≥180/110 mm Hg). The threshold is retained for consistency with national statistics and as the level above which all adults should be considered for pharmacotherapy, but it is an arbitrary point on a continuum of risk, not a level below which no one should be treated.
Hypertension-mediated organ damage (HMOD)	The maladaptive structural and functional changes that sustained high blood pressure cause in target organs — the heart, blood vessels, kidneys and brain, such as left ventricular hypertrophy, albuminuria, hypertensive retinopathy, and cerebral small-vessel disease.
Low-dose	A term used inconsistently across the hypertension literature. In trials of low-dose combination therapy, "low dose" denotes any dose below the standard (full) dose of an agent, most commonly a quarter or half of standard dose. International guidance applies a broader operational definition, encompassing the recommended starting dose or the lowest available formulation of a drug or fixed-dose combination, doses that for many agents are equivalent to standard dose. To avoid ambiguity, this document uses the terms "recommended starting dose" and "lowest available formulation" rather than "low dose." Few dual combinations registered in Australia at the time of writing meet the narrower trial definition.
Masked hypertension	A blood pressure phenotype in which office readings are normal (e.g. <140/90 mm Hg) but out-of-office readings are elevated. Elevation is defined as an average of ≥135/85 mm Hg on home blood pressure monitoring, ≥135/85 mm Hg on the daytime (awake) period of 24-hour ambulatory blood pressure monitoring, or ≥130/80 mm Hg on the 24-hour ambulatory average. An estimated 10–17% of adults are affected.

Optimal blood pressure	A blood pressure reading taken in a clinic that is below 120 over 80 (120/80 mmHg). The chance of having a heart attack or stroke, or of dying early, is lowest in people whose blood pressure sits below about 115/75 — and in the general population there is no sign that going lower than this causes harm.
Orthostatic (postural) hypotension	A sustained fall in blood pressure on standing, commonly defined as a drop in systolic blood pressure of at least 20 mm Hg or diastolic blood pressure of at least 10 mm Hg within three minutes of standing (or head-up tilt). It may be asymptomatic, or it may cause symptoms such as dizziness, light-headedness or falls.
Patient non-adherence	Medication-taking behaviour that does not correspond with the regimen agreed with the healthcare provider, occurring as non-initiation, suboptimal implementation of the dosing regimen, or early discontinuation. It may be intentional or unintentional, and is distinct from clinical inertia, which occurs at the provider level.
Priority populations	Population groups identified in this guideline as experiencing a higher burden of hypertension, poorer rates of detection or control, or greater barriers to accessing care, and for whom tailored guidance is provided throughout: Including but not only First Nations peoples; people living in regional and remote areas; culturally and linguistically diverse communities; older adults; women; and transgender and gender-diverse people.
Secondary hypertension	Hypertension attributable to an identifiable underlying cause, such as primary aldosteronism, renal or renovascular disease, obstructive sleep apnoea, other endocrine conditions, or medications that raise blood pressure. It accounts for approximately 5–10% of all hypertension, rising to around 30% in adults aged 18–40 years. Some causes are reversible or require specific management, and reliable identification requires deliberate screening rather than reliance on clinical suspicion alone.
Shared decision-making	A cooperative process in which clinicians, patients, and families/carers work together to choose a course of treatment that aligns with the patient's values and preferences alongside clinical evidence, involving presentation of options, discussion of benefits and risks, and reaching a shared plan.

Single-pill combination (SPC)	A fixed-dose combination of two or more antihypertensive agents from different classes (e.g. ACE inhibitor/ARB with a calcium channel blocker or thiazide-like diuretic) combined into a single tablet.
Standard office blood pressure	Blood pressure measured in a clinical setting by a trained operator using a validated device and standardised technique, including appropriate cuff size, seated rest before measurement, correct positioning, and the averaging of at least two readings. Unless stated otherwise, all thresholds, categories and targets in this guideline are expressed as standard office blood pressure. Automated office blood pressure, home blood pressure monitoring and ambulatory blood pressure monitoring produce systematically lower values for the same underlying blood pressure, and the equivalent values for those methods should be applied.
Suspected hypertension	Suspected hypertension is identified when initial office screening shows a blood pressure of ≥140 mm Hg systolic and/or ≥90 mm Hg diastolic. A single elevated reading does not confirm a diagnosis. Confirmation requires either a repeat office measurement at a subsequent visit, or ideally out-of-office monitoring — 24-hour ambulatory blood pressure monitoring (ambulatory blood pressure monitoring, average ≥130/80 mm Hg) or home blood pressure monitoring (home blood pressure monitoring, average ≥135/85 mm Hg).
Team-based care	An evidence-based model in which two or more healthcare professionals (e.g. general practitioners, nurses, pharmacists, exercise physiologists) work collaboratively with the patient toward a shared blood pressure goal, with tasks tiered from administrative to advanced clinical duties such as medication titration.
Treatment inertia (clinical/therapeutic inertia)	The absence of treatment initiation or intensification by healthcare providers when clinical guidelines indicate that it is warranted. It is distinct from patient non-adherence.
White-coat effect/white-coat hypertension	The white-coat effect is a transient rise in blood pressure occurring in a clinical setting, typically in the presence of healthcare professionals; when this pattern is persistent alongside normal out-of-office blood pressure, it is termed white-coat hypertension (defined as elevated office blood

	pressure but blood pressure below the hypertension threshold on out-of-office measurement).

1 How to read this guideline

The key conventions used in this guideline are set out below.

‘Clinician’ means any health professional working within their scope of practice

Throughout this guideline, ‘clinician’ is used as a generic term for any health professional providing care within their scope of practice. This includes, but is not limited to, general practitioners, nurses and nurse practitioners, pharmacists, Aboriginal and Torres Strait Islander Health Practitioners and Health Workers, and allied health professionals.

This convention recognises that in many communities, particularly regional, rural and remote areas, these professionals are often the first point of contact.

‘Office blood pressure’ is used in preference to ‘clinic blood pressure’

Australian practice more commonly refers to clinic BP (BP), and the two terms are equivalent. Throughout this guideline, ‘office BP’ is used instead, for consistency with the established international terminology of automated office blood pressure (AOBP) measurement.

This convention keeps terminology uniform across the guideline, so that measurement in a clinical setting is described the same way whether or not an automated device is used and aligns with the terminology of the underpinning evidence base.

A blood pressure value means an average, not a single reading

Unless a single reading is explicitly specified, every BP value in this guideline — for classification, diagnosis, treatment thresholds and targets — refers to an average of at least two standardised measurements, obtained on at least two separate occasions.

A single high reading does not establish a diagnosis. A single reading at target does not establish control. One exception applies: in individuals with a BP ≥180 mm Hg systolic and/or ≥110 mm Hg diastolic and evidence of cardiovascular disease (CVD), provided the reading is confirmed by repeat standardised measurement at that same visit and no reversible cause of acute BP elevation, such as pain, acute anxiety, or recent exertion or stimulant use is identified, a single reading is sufficient to establish the diagnosis.

Numbers are standard office blood pressure unless stated otherwise

Although several methods are available to measure BP, the thresholds, categories and targets in this guideline are expressed according to measurement by standard office blood pressure.

This matters because AOBP, home blood pressure monitoring (HBPM) and ambulatory blood pressure monitoring (ABPM) produce systematically lower values than standardised conventional office measurement for the same underlying BP. Where a method other than standardised conventional office measurement is used, apply the equivalent values given in the relevant section for that method.

A blood pressure category is not a treatment decision

Optimal BP, elevated BP and hypertension define bands of BP measurement and the associated risk. They do not by themselves determine optimal management.

Treatment decisions combine the BP category with calculated cardiovascular risk, generally using the *Australian Guideline for assessing and managing cardiovascular disease risk*.[1] Two people with identical readings may be best managed differently. Some people with BP below the diagnostic threshold for hypertension may need to be treated while others above the threshold may not benefit from intervention.

What we mean by a 'heart-healthy' eating pattern

Throughout this guideline, a 'heart-healthy' eating pattern describes an overall pattern of food chosen regularly over time, not single foods, 'good' and 'bad' foods, or restrictive dieting.

A heart-healthy diet includes a wide variety of foods and is rich in wholegrains, fibre, vitamins, minerals, and healthy fats, while naturally low in unhealthy fats, sodium and added sugar. In practice, this means:

- Plenty of vegetables, fruit and wholegrains.
- A variety of healthy protein-rich foods, favouring legumes, nuts, seeds, fish and seafood, with lean red meat limited to 1–3 times per week.
- Unflavoured milk, yogurt and cheese (low fat if living with coronary heart disease or high cholesterol).
- Healthy fats and oils, such as avocado, olives, nuts, seeds and olive, canola or sunflower oil.
- Herbs, spices, or potassium-enriched salt in place of regular salt for flavour.

It also means limiting ultra-processed foods and sugary drinks, with water as the drink of choice. Sugary drinks include sugar-sweetened beverages, flavoured milks, fruit juices, energy drinks and sports drinks.

Further information about a heart-healthy diet can be found at Heart Foundation – Healthy eating. https://www.heartfoundation.org.au/healthy-living/healthy-eating

2 Recommendations

Recommendation	Strength of recommendation or source guideline class	Certainty of evidence or source guideline level
Identifying hypertension		
We recommend opportunistic office blood pressure measurement to screen for elevated BP and hypertension in all adults aged 18 years or older without known hypertension.	Consensus	
In adults not currently receiving antihypertensive treatment and with a prior optimal reading, we suggest the following rescreening intervals: • at least every 3 years for adults aged 18–39 years at low cardiovascular risk; • at least annually for adults aged 40 years or older; at least annually at any age for adults at increased cardiovascular risk, a family history of early-onset hypertension or with previously elevated BP not currently meeting the threshold for treatment.	Consensus	
Blood pressure measurement		
We recommend automated office blood pressure (AOBP) measurement with a validated upper-arm device as the preferred office method, applying method-specific thresholds. Where AOBP is not available, we recommend standard office blood pressure measurement.	Consensus*	
We recommend that hypertension not be diagnosed on the basis of a single office visit. A diagnostic threshold is only met when the averaged readings across at least two separate, consecutive occasions are at or above the diagnostic thresholds.	Consensus	

Recommendation	Strength of recommendation or source guideline class	Certainty of evidence or source guideline level
Hypertension may be diagnosed at a single visit when office BP is ≥180 mm Hg systolic and/or ≥110 mm Hg diastolic and there is evidence of CVD, provided the reading is confirmed by repeat standardised measurement at that same visit and no reversible cause of acute BP elevation, such as pain, acute anxiety, or recent exertion or stimulant use is identified.	Consensus	
We recommend confirming suspected hypertension with out-of-office measurement using ambulatory (ABPM) or home (HBPM) monitoring, applying method-specific thresholds.	Adopted recommendation AHA/ACC 2025[2] and ESC 2024[3]	
	Class I AHA/ACC 2025[2]; ESC 2024[3]	Level A AHA/ACC 2025[2] Level B ESC 2024[3]
We recommend including HBPM in the long-term follow-up of treated hypertension.	Adopted recommendation AHA/ACC 2025[2]; ESH 2023[4]	
	Class I AHA/ACC 2025;[2] ESH 2023[4]	Level A AHA/ACC 2025[2] Level B ESH 2023[4]
Secondary causes of hypertension – primary aldosteronism		
In all adults with newly diagnosed hypertension, we suggest screening for primary aldosteronism.	Adopted recommendation Primary Aldosteronism: An Endocrine Society Clinical Practice Guideline 2025;[5] ESC 2024[6]	
	Conditional Endocrine Society PA guideline 2025[5] Class IIa ESC 2024[6]	Low Endocrine Society PA guideline 2025[5] Level B ESC 2024[6]
In adults with pre-existing hypertension and any of the following, we suggest screening for primary aldosteronism: • BP above 140/90 mm Hg despite antihypertensive treatment, including resistant hypertension (BP above target on	Consensus	

Recommendation	Strength of recommendation or source guideline class	Certainty of evidence or source guideline level
three or more agents at maximally tolerated doses including a diuretic, or controlled on four or more agents) • spontaneous or diuretic-induced hypokalaemia • atrial fibrillation unexplained by structural heart disease or thyroid disease • an adrenal adenoma • obstructive sleep apnoea • a family history of early-onset hypertension, a family history of stroke before age 40, or a first-degree relative with primary aldosteronism		
Blood pressure categories		
Optimal blood pressure	<120 and <80 mm Hg	
Elevated blood pressure	120–139 and/or 80–89 mm Hg	
Hypertension	≥140 and/or ≥90 mm Hg	
Blood pressure targets		
In adults receiving antihypertensive treatment, we recommend targeting an office blood pressure below 130 mm Hg systolic and below 80 mm Hg diastolic.	Strong	High
Dietary patterns and nutrients		
We recommend the Heart Foundation's heart-healthy eating pattern, adapted to individual cultural food preferences, for all adults to prevent and manage elevated blood pressure, maintained long term and alongside antihypertensive medication where indicated.	Strong	Moderate

Recommendation	Strength of recommendation or source guideline class	Certainty of evidence or source guideline level
We recommend that adults limit sodium intake to less than 2,000 mg per day (equivalent to less than 5 g of salt, or about one teaspoon) from all sources, including salt added during cooking and at the table and salt already present in processed, packaged and takeaway foods.	Adopted recommendation ESC 2024[6]	
	Class I ESC 2024[6]	Level A ESC 2024[6]
In adults with elevated blood pressure or hypertension, we recommend increasing intake of potassium-rich foods, including vegetables, fruit, legumes, nuts and unsalted dairy.	Adopted recommendation AHA/ACC 2025[2]	
	Class I AHA/ACC 2025[2]	Level A AHA/ACC 2025[2]
In adults with elevated blood pressure or hypertension who add salt during cooking or at the table, we suggest replacing regular salt with a potassium-enriched salt substitute.	Conditional	Moderate
Advice to increase dietary potassium intake, including through potassium-rich foods or potassium-enriched salt substitutes, does not apply to adults at increased risk of hyperkalaemia. In these people, increase dietary potassium or introduce a potassium-enriched salt substitute only under clinical supervision and monitoring.	Consensus[†]	
Physical activity		
In adults, we recommend regular physical activity accumulated throughout the day, combined with reducing sedentary behaviour, to prevent and manage elevated blood pressure. Activity should be maintained long term, alongside antihypertensive medication where indicated.	Consensus[‡]	
In adults with elevated BP or hypertension, we recommend a combination of moderate-to-	Adopted recommendation AHA/ACC 2025[2]	

Recommendation	Strength of recommendation or source guideline class	Certainty of evidence or source guideline level
vigorous aerobic activity and resistance training to reduce blood pressure and cardiovascular risk, with the exercise program tailored to the individual's goals and condition.	Class I AHA/ACC 2025[2]	Level A AHA/ACC 2025[2]
Alcohol		
In adults with elevated blood pressure or hypertension who currently consume alcohol, we recommend advising abstinence as the preferred goal. Where abstinence is not achieved, we recommend limiting intake to no more than 1 standard drink per day for women and 2 standard drinks per day for men, and to no more than 10 standard drinks per week overall, with several alcohol-free days each week.*	Adopted recommendation AHA/ACC 2025[2]	
	Class I AHA/ACC 2025[2]	Level A AHA/ACC 2025[2]
Smoking		
In adults with elevated blood pressure or hypertension who smoke, we recommend that smoking cessation be encouraged and supported at every opportunity, including referral to behavioural interventions (such as cognitive behavioural therapy or cessation counselling) and TGA-approved pharmacotherapy where clinically indicated.	Adopted recommendation ESC 2024[6]	
	Class I ESC 2024[6]	Level A ESC 2024[6]
Weight loss		
In adults with elevated blood pressure or hypertension who are living with overweight or obesity, we recommend weight reduction, alongside antihypertensive medication where indicated.	Adopted recommendation‡ AHA/ACC 2025;[2] ESC 2024[6]	
	Class I AHA/ACC 2025;[2] ESC 2024[6]	Level A AHA/ACC 2025;[2] ESC 2024[6]

Recommendation	Strength of recommendation or source guideline class	Certainty of evidence or source guideline level
Pharmacotherapy initiation thresholds		
In adults with low cardiovascular risk (<5% risk over 5 years), we recommend initiating antihypertensive pharmacotherapy alongside healthy lifestyle modification when office blood pressure is ≥140 mm Hg systolic and/or ≥90 mm Hg diastolic.	Strong	High
In adults with intermediate cardiovascular risk (5 to <10% risk over 5 years), we recommend considering antihypertensive pharmacotherapy when office blood pressure is ≥130 mm Hg systolic and/or ≥80 mm Hg diastolic, and initiating pharmacotherapy alongside lifestyle modification in all when office blood pressure is ≥140 mm Hg systolic and/or ≥90 mm Hg diastolic.	Strong	High
In adults with either established cardiovascular disease or high cardiovascular risk (≥10% risk over 5 years), we recommend initiating antihypertensive pharmacotherapy alongside healthy lifestyle modification when office blood pressure is ≥130 mm Hg systolic and/or ≥80 mm Hg diastolic.	Strong	High
In adults with office blood pressure ≥180 mm Hg systolic and/or ≥110 mm Hg diastolic, we recommend initiating antihypertensive pharmacotherapy without delay, alongside healthy lifestyle modification.	Consensus	
Pharmacotherapy management		
In adults with confirmed hypertension (office blood pressure ≥140 mm Hg systolic and/or ≥90 mm Hg diastolic), we recommend initiating treatment with	Strong	High

Recommendation	Strength of recommendation or source guideline class	Certainty of evidence or source guideline level
a lowest available formulation single-pill combination of two first-line antihypertensive agents from different classes (ACE inhibitor or angiotensin receptor blocker, calcium channel blocker, or thiazide/thiazide-like diuretic).		
In adults indicated for pharmacotherapy at systolic blood pressure 130–139 mm Hg or diastolic blood pressure 80–89 mm Hg, we recommend initiating treatment with a lowest available formulation dual single-pill combination of two first-line agents from different classes. We suggest that a single first-line agent (monotherapy) may be considered for selected patients where clinically appropriate.	Strong	High
In adults requiring pharmacotherapy who have a compelling indication for beta-blocker therapy (e.g. heart failure with reduced ejection fraction, or the need for heart rate control), we recommend a beta-blocker in combination with one or more of the major first-line blood pressure–lowering classes.	Strong	High
In adults with hypertension, we recommend against dual renin–angiotensin system blockade with an ACE inhibitor plus an angiotensin receptor blocker. Dual blockade does not reduce all-cause or cardiovascular mortality compared with monotherapy, and increases the risk of hyperkalaemia, hypotension, renal failure and treatment discontinuation.	Strong (against)	High

*Adapted from *Automated office blood pressure measurement: a Hypertension Australia and National Hypertension Taskforce of Australia position statement*.[7]

†Adapted from *Potassium-enriched salt for patients with hypertension: a Hypertension Australia and National Hypertension Taskforce of Australia position statement*.[8]

‡Adopted from the National Heart Foundation of Australia, *Obesity and cardiovascular disease: a clinical consensus statement*, 2026.

3 Introduction and methods

3.1 Introduction

This guideline assists clinicians in diagnosing and managing adults with elevated BP and hypertension. Recommendations are informed by the best available contemporary evidence and support consistent, high-quality, equitable care, complementing rather than replacing clinical judgement. Shared decision-making among healthcare professionals, individuals and their families or carers should reflect individual values, preferences and social or cultural contexts, including the needs of First Nations peoples and those in regional, rural and remote areas.

The guideline was developed collaboratively by the Heart Foundation, Stroke Foundation, and Hypertension Australia, in consultation with the National Hypertension Taskforce, clinical experts, researchers, professional colleges, policymakers, and consumers with lived experience, drawing on diverse geographic, professional and population representation.

3.2 Purpose and objectives

This document replaces the *2016 Guideline for the Diagnosis and Management of Hypertension in Adults*, reflecting new evidence, evolving practice, and national health priorities. It aims to:

- Improve detection of hypertension through enhanced screening, particularly in high-risk and underserved populations, and standardised BP measurement.
- Increase BP control rates by improving treatment effectiveness and adherence.
- Reduce morbidity and mortality from hypertension-related conditions, including cardiovascular disease (CVD), stroke, kidney disease and dementia.
- Provide practical tools to support consistent application of recommendations across healthcare settings.

3.3 Scope

This guideline addresses the assessment, diagnosis and management of elevated blood pressure and hypertension in adults (≥18 years). It includes:

- Diagnostic criteria, risk assessment and evaluation of elevated blood pressure and hypertension.
- Measurement technique and guidance for the diagnosis and ongoing monitoring of blood pressure and hypertension.
- Behavioural and pharmacological management strategies.
- Tailored advice for groups experiencing health inequities or access challenges.

Out of scope: hypertension in children and adolescents, gestational hypertension or pre-eclampsia, hypertensive crisis and emergency, and detailed management of specific comorbidities such as diabetes or chronic kidney disease (CKD).

3.3.1 Recognising a hypertensive emergency

Management of hypertensive emergencies is outside the scope of this guideline, but primary care clinicians should recognise the presentation.

Hypertensive emergency — BP ≥180/120 mm Hg with acute target-organ damage or dysfunction. Recognised manifestations include hypertensive encephalopathy, acute ischaemic or haemorrhagic stroke, papilloedema or retinal haemorrhage, acute heart failure or pulmonary oedema, acute coronary syndrome, acute aortic syndrome, acute kidney injury, and severe pre-eclampsia or eclampsia.

Requires immediate referral for emergency assessment, rather than routine outpatient management or same-day antihypertensive titration. Do not delay referral to repeat the measurement or to commence oral therapy. In-hospital management, including monitoring and parenteral antihypertensive therapy, is outside the scope of this guideline.

Severe asymptomatic hypertension — BP ≥180/110 mm Hg with no symptoms and no clinical features of acute target-organ damage. Diagnosis may be made on a single occasion if there is evidence of CVD, provided the reading is confirmed by repeat standardised measurement at that same visit and no reversible cause of acute BP elevation, such as pain, acute anxiety, or recent exertion or stimulant use is identified. Confirmatory out-of-office measurement is not required before commencing treatment. Assess for target-organ damage and commence or intensify oral antihypertensive therapy.

The two states are distinguished by the presence of acute target-organ damage, not just by the blood pressure level. Symptoms that may signal acute target-organ damage — including severe headache with neurological signs, chest pain, dyspnoea, altered consciousness, visual disturbance, or signs of acute heart failure — warrant urgent assessment to exclude an emergency, regardless of the diastolic value.

3.4 Intended population

This guideline is intended for people aged 18 years and over seen in primary care or other community settings for screening, diagnosis, or ongoing management of blood pressure, including those with optimal BP, confirmed hypertension, and persistently elevated readings not yet meeting diagnostic threshold.

For guidance in those under the age of 18, the *Australian guideline for hypertension in children and adolescents* can be used, available at Hypertension Australia.

This guideline treats ‘sex’ as biological (chromosomal, hormonal, anatomical) and ‘gender’ as social/behavioural (care-seeking, occupational exposure, structural access); both are relevant. ‘Women’ and ‘men’ are used where source data refers to a binary sex variable. Guidance for transgender, intersex and gender diverse populations is provided where possible, noting limited evidence at the time of writing.

3.5 Intended audience

This guideline is intended for all healthcare professionals involved in prevention, detection, diagnosis, and management of elevated blood pressure and hypertension. This includes general practitioners, rural generalists, general physicians, cardiologists, nephrologists, endocrinologists, neurologists, nurses, nurse practitioners, Aboriginal and Torres Strait Islander Health Workers and Practitioners, pharmacists, dietitians, exercise physiologists, and other allied health professionals — as well as health administrators, policymakers, and consumer advocates.

Throughout this guideline, 'clinician' is used as a generic term for any health professional providing care within their scope of practice. For further information see How to read this guideline.

3.6 Method

Recommendations are evidence-based where possible, reflecting the most contemporary, high-quality evidence. A detailed method is documented in the Supplementary document.

3.6.1 Guideline development process

This guideline was developed using the GRADE-ADOLOPMENT framework, which enables guideline groups to adopt, adapt, or develop de novo recommendations from existing high-quality guidelines and evidence syntheses, ensuring recommendations are both rigorous and suited to the Australian context.[9,10] Development was overseen by a multidisciplinary Expert Steering Group (ESG) and Writing Committees (with consumer representation), under Heart Foundation conflict-of-interest policy, with all recommendations formally approved prior to finalisation. Full methodological detail, including the scoping search and AGREE II appraisal process, PICO question development, systematic search and evidence synthesis methods, and committee composition, is provided in the Supplementary document.

3.6.2 Classification of guidance

There are four classifications of guidance used throughout this guideline, arranged in a hierarchy that reflects the strength of the underlying evidence and the context of their application: GRADE recommendations, consensus recommendations and practice points. Further information is in Table 1.

Table 1: Classification of guidance used throughout guideline

Type of guidance	Basis	When used	Strength/format
GRADE recommendation	Grading of Recommendations Assessment, Development and	When the certainty of evidence is sufficient	Strong or conditional (weak), reflecting the certainty of evidence

	Evaluation (GRADE) methodology; balances benefits and harms, incorporates patient values and preferences, and considers resource use	to apply the full GRADE approach	and the intervention's impact
Adopted recommendation	A recommendation taken from a credible existing guideline, appraised for quality, currency and applicability, and used either unmodified or with minor changes for the Australian context, without a new systematic review. The source guideline and any modifications are stated.	When reliable, current guidance on the question already exists and repeating the synthesis was not within scope of the guideline. Adoption reflects the availability of trustworthy guidance, not limited underlying evidence.	Retains the source guideline's strength and certainty of evidence as published, reported using the source's own terminology and cited to the source guideline
Consensus recommendation	Expert opinion, supported by the available evidence and informed by values, preferences, and resource considerations	When the GRADE approach is not applicable, often due to indirect or limited evidence	Provides critical guidance where clinical decisions are necessary despite an incomplete or indirect evidence base
Practice point	Practical, actionable advice to support implementation	Alongside a recommendation, to guide how it is applied in practice	Specifies the who, what, how, and when; may include supplementary detail such as medication dosing or implementation tools. Does not stand alone

GRADE recommendations are the most robust, using the full GRADE methodology.[11] Consensus recommendations are used where GRADE is not applicable, often due to indirect or limited evidence, or where an international recommendation was endorsed by the ESG without an updated systematic review (the source guideline is cited in the recommendation table notes). Practice points are actionable implementation advice that does not stand alone but is essential to applying recommendations effectively, particularly where geographic or resource constraints apply.

4 What's new in this guideline

This 2027 guideline is the first comprehensive revision since 2016, reflecting a decade of accumulated trial evidence, a revised methodology, and a deliberate broadening of scope from a primarily biomedical, single-condition document into one explicitly organised around equity, implementation and the Australian health system.

Blood pressure categories and terminology

The blood pressure classification system has been simplified and re-labelled.

- Optimal blood pressure: <120 mm Hg and <80 mm Hg
- Elevated blood pressure: 120–139 mm Hg and/or 80–89 mm Hg
- Hypertension: ≥140 mm Hg and/or ≥90 mm Hg

The diagnostic threshold for hypertension itself is unchanged at ≥140 mm Hg and/or ≥90 mm Hg.

Diagnostic pathways and CVD risk assessment

The Australian CVD Risk Calculator (aligned to the 2023 *Australian Guideline for assessing and managing cardiovascular disease risk*) is the primary risk-assessment tool for eligible adults. A new 'CVD risk at any age' pathway covers adults outside its eligible age range, with explicit guidance not to withhold treatment from adults aged 80 or over on the basis of age alone. See *Hypertension and CVD risk assessment*.

Secondary causes and primary aldosteronism

ARR testing is now recommended in all adults with confirmed hypertension rather than only those with triggering features, with a targeted fallback where access is limited. Screening-phase medication management is simplified (withhold MRAs where feasible; other agents can generally continue), and a new recommendation covers next steps after a positive result. See Secondary causes of hypertension.

Blood pressure measurement standards

AOBP, ideally unattended, is the recommended office method. Out-of-office confirmation is now indicated at a lower BP than previously — office SBP 130–139 or DBP 80–89 mm Hg. A valid ABPM recording needs at least 20 daytime and 7 night-time readings. See **Blood pressure measurement**.

Blood pressure targets

Target lowered to <130 and <80 mm Hg for confirmed hypertension, consistent across secondary stroke prevention, CKD and type 2 diabetes. Average of ≥2 standardised measurements on ≥2 separate occasions at or below target. See **Treatment targets**.

Treatment thresholds for pharmacotherapy

Pharmacotherapy should be initiated at ≥140/90 mm Hg for adults at low-to-intermediate CV risk and at ≥130/80 mm Hg for those with established CVD or high CV risk; also consider it at the upper end of intermediate risk. Severe elevation (≥180/110 mm Hg) with acute target-organ symptoms is a hypertensive emergency (see Recognising a hypertensive emergency) — refer immediately rather than initiating treatment.

Pharmacotherapy

Initial therapy is a lowest available formulation dual single-pill combination rather than monotherapy with titration, with SPCs preferred at every stage of initiation and escalation; a 2026 PBS change permits dual-therapy SPCs first-line. Beta-blockers are not recommended as routine first-line monotherapy, and should be added instead when there is a compelling indication (e.g. angina, heart failure with reduced ejection fraction, or rate control). TIME trial evidence shows dosing time does not affect cardiovascular outcomes, so it can be individualised. See Pharmacotherapy for blood pressure.

Treatment inertia, adherence and digital health

Treatment (clinical) inertia — clinicians failing to initiate or intensify therapy when indicated — is a key barrier to BP control. Adherence guidance has expanded to cover SPCs and coordinated multidisciplinary care for polypharmacy, alongside a new section on digital health.

Multidisciplinary and team-based care

New material on team-based models in which non-physician team members conduct most follow-up and, in the highest-impact models, titrate medication under standing orders or protocols. See General approaches to care.

5 Preamble

5.1 Hypertension in Australia

5.1.1 Burden and importance

Hypertension, or high BP, is a major public health issue in Australia and is a significant modifiable risk factor for chronic conditions including stroke, coronary heart disease (CHD), heart failure, dementia and chronic kidney disease (CKD).[12] It contributes substantially to morbidity, mortality and health system burden.[12] Despite its well-established links with adverse outcomes, rates of adequate BP control have remained largely unchanged for more than a decade, and around half of affected adults remain undiagnosed.[13]

5.1.2 How high blood pressure causes harm

High BP damages the vasculature directly, weakening arterial walls and promoting aneurysm formation, cerebral small-vessel disease, and atherosclerosis, making it a particularly strong risk factor for haemorrhagic and ischaemic stroke, cardiac decompensation, heart failure and atrial fibrillation. It also contributes to atherosclerotic CVD indirectly, acting alongside other risk factors (see *Hypertension and CVD risk assessment*).

5.1.3 Arterial changes and blood pressure patterns

Hypertension is primarily a small-artery condition (raising diastolic BP via arteriolar narrowing), while systolic BP more often reflects stiffening of larger arteries with age, producing lower diastolic pressure and the common isolated systolic hypertension (ISH) phenotype in older Australians (see *Isolated systolic hypertension*). Risk is similar whether systolic, diastolic, or both are elevated, so this guideline treats these patterns collectively.

5.1.4 Prevalence, trends, outcome and disease burden in Australia

Hypertension (defined in national statistics as BP ≥140/90 mm Hg and/or use of BP-lowering medication) is increasingly common in Australia, rising from 33.7% in 2017–18 to 39.6% in 2022–23, and increasing sharply with age (around 7% at 18–24 years to 85% at 75 and older).[12,14] Population ageing and higher rates of overweight/obesity, diabetes and other cardiometabolic risks are driving this burden.

Hypertension is the leading risk factor for preventable death in Australia, contributing to more than 25,000 deaths each year and a large share of disease burden, including about 43% of CHD, 41% of stroke, 65% of hypertensive heart disease, 38% of CKD, 32% of AF, and 3.6% of dementia cases.[15-17] This burden is not shared equally. Hypertension affects 34% of First Nations peoples, control rates are lower than in the general population (22.2%), and prevalence in those aged 25–34 years is nearly twice that of non-First Nations Australians of the same age.[12,14,18,19] These disparities reflect the ongoing impacts of colonisation and the

social and cultural determinants of health, including access to culturally safe care, rather than any inherent difference in risk.

5.1.5 Gaps in detection and control

BP control remains inadequate. Only about 40% of adults with hypertension achieve recommended targets, and around half (about 3.4 million people) remain undiagnosed, limiting timely management and increasing complication risk.[12,13,17,20-22] Control rates are poor compared with other high-income countries, and although absolute cardiovascular risk-based approaches are increasingly used, fewer than half of those at highest risk receive all recommended therapies, constraining risk reduction.[21,23-25]

In response, the National Hypertension Taskforce was established to increase national blood pressure control rates to at least 70% by 2030. Its roadmap outlines coordinated actions to improve prevention, detection and treatment across the health system, and this guideline is aligned with its key recommendations.[20]

5.1.6 Economic impact and value of effective management

Uncontrolled hypertension costs Australia an estimated $137 billion in lost GDP over a working lifetime through reduced productivity, with $76 billion recoverable through effective treatment and control.[26] Effective management is highly cost-effective: antihypertensives are widely available as low-cost PBS-subsidised generics, and single-pill combinations can reduce patient costs by around 30% compared with equivalent multi-pill regimens, particularly as 60-day scripts, while also improving adherence and BP control.[27-30]

5.2 General approaches to care

5.2.1 Multidisciplinary and team-based care

Primary care is central to hypertension assessment, management and long-term monitoring in Australia, with general practice teams providing first-line, person-centred care, including healthy lifestyle support, pharmacotherapy and referral when required. Team-based care is an evidence-based approach in which two or more healthcare professionals — such as general practitioners, nurses, pharmacists, dietitians, exercise physiologists and other allied health professionals — collaborate with the patient to achieve BP targets. This improves BP control compared with usual care while being clinically effective and cost-effective.[31-36] Tasks are shared across administrative, basic clinical and advanced clinical roles, with non-physician team members commonly leading routine follow-up, monitoring, adherence support and escalation, and achieving the greatest BP reductions when they titrate medication directly under protocol.[34-36] Medicare-subsidised Chronic Condition Management Plans and Home Medicines Reviews support this model of team-based care.

Additionally, pharmacists contribute to hypertension care — in an increasing number of jurisdictions — through assessment, diagnosis and prescribing under structured protocols. Scope of practice differs between states and territories, because authority to prescribe is set

by state and territory medicines and poisons legislation rather than nationally. Clinicians should confirm what is authorised locally.

Where expanded scope applies, pharmacist-led hypertension care operates within defined protocols. Under the Queensland Community Pharmacy Chronic Conditions Management Pilot, trained pharmacists assess absolute cardiovascular risk, measure blood pressure using a standardised technique, prescribe up to two first-line antihypertensive agents, order relevant pathology, review response at 4–6 weeks, and modify or deprescribe therapy. The protocol excludes people with severe hypertension, chronic kidney disease stages 3–5, heart failure, pregnancy, and those aged under 18 or over 79, and specifies referral where target is not reached on two agents at maximum tolerated doses. Comparable programs operate or are in development in the Northern Territory, Victoria and regional New South Wales, and further expansion is expected.

Follow-up should be tailored to BP levels, treatment response and overall cardiovascular risk. Where follow-up is led by nurses or pharmacists, review is typically more frequent than in usual physician-led care, supported by ongoing communication within the team. Culturally appropriate, continuity-promoting models sustain engagement and adherence, and telehealth can further support care access and continuity, particularly in regional and remote areas.

5.2.2 Shared decision-making

Shared decision-making is especially relevant to cardiovascular risk reduction. Blood pressure is one of several modifiable risks to be weighed alongside healthy lifestyle change and treatment burden, rather than treated in isolation.[37,38]

This approach improves engagement, treatment adherence and BP control, and may reduce health inequities.[39,40] However, disparities in participation persist, particularly among women and older adults, highlighting the need for more inclusive approaches.[41]

5.3 Priority populations and models of care in hypertension management

5.3.1 General principles across priority populations

Across all priority groups, hypertension care should be person-centred, culturally safe and tailored to individual clinical circumstances, preferences and goals of care. Shared decision-making should underpin assessment, treatment selection and follow-up, particularly where access barriers, communication needs, competing comorbidities or differing risk profiles affect care choices. Continuity of care, clear communication, interpreters or support workers, and flexible models such as team-based care and telehealth can strengthen engagement and support sustained BP control.

Specific practice points for priority populations are embedded throughout the guideline. Table 2 provides an at-a-glance summary of the population, key considerations and practice tips.

Table 2: Priority populations at a glance

Population	Key consideration	Practice tip
First Nations peoples	34% hypertension prevalence; control rate 22.2% vs 31.1% general population; nearly twice the rate in 25–34-year-olds [12,14,18,19]	Involve First Nations health practitioners and liaison officers early; use cultural competence and yarning resources on the Heart Foundation website[42]
Regional/remote	Similar prevalence to major cities, but access barriers, higher smoking/alcohol use, and higher service costs[12,43,44]	Balance clinical need with the person's preference for local vs travel-based care; telehealth and 60-day dispensing where feasible
Culturally and linguistically diverse communities	32% born overseas, 23% speak a language other than English at home; language and health-literacy barriers[45,46]	Use credentialled interpreters (TIS National provides free telephone/video interpreting for eligible services) rather than relying on bilingual staff
Older adults (≥65 years)	73.5–85.3% prevalence; frailty, comorbidity, polypharmacy; historically underrepresented in trials but HYVET/SPRINT show clear benefit[12,47-51]	Individualise treatment goals and monitoring; don't withhold or under-intensify therapy based on age alone
Women	BP rises steeply during/post-menopause, exceeding male prevalence by the 7th decade[12,52,53]	Consider hormonal contraceptives, hypertensive disorders of pregnancy, and menopausal status in assessment
Transgender and gender-diverse	Limited evidence; hormone therapy affects BP (testosterone may raise, oestrogen/antiandrogens may lower)[54,55]	Integrate BP and CV risk monitoring into gender-affirming care rather than managing separately[56]

Abbreviations: BP, blood pressure; CV, cardiovascular; HYVET, Hypertension in the Very Elderly Trial; SPRINT, Systolic Blood Pressure Intervention Trial; TIS, Translating and Interpreting Service.

Detailed practice points for these populations are embedded throughout the guideline wherever they affect measurement, risk assessment, or management decisions.

6 Detection and confirmation of hypertension

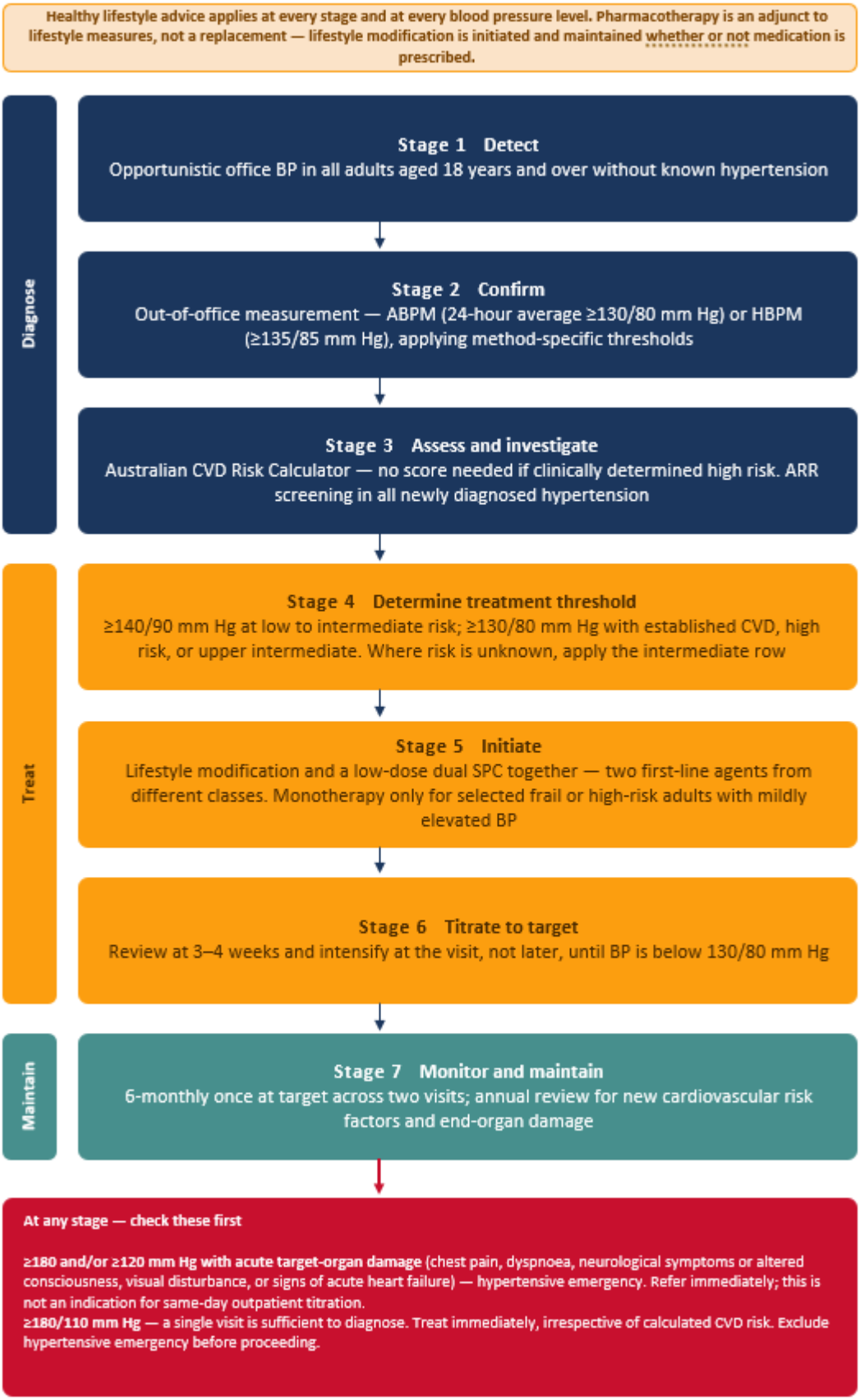


Figure 1: Hypertension management pathway

The hypertension management pathway (Figure 1) summarises the whole patient journey covered by this guideline, from detection through to ongoing monitoring. Each stage below is expanded in the corresponding chapter; the BP classification and treatment escalation diagrams referenced within it provide the detail behind stages 4 and 6.

6.1 Identifying patients at risk of hypertension

Recommendation	Strength of recommendation	Certainty of evidence
We recommend opportunistic office blood pressure measurement to screen for elevated BP and hypertension in all adults aged 18 years or older without known hypertension.	Consensus	
In adults not currently receiving antihypertensive treatment and with a prior optimal reading, we suggest the following rescreening intervals: • at least every 3 years for adults aged 18–39 years at low cardiovascular risk; • at least annually for adults aged 40 years or older; at least annually at any age for adults at increased cardiovascular risk, a family history of early-onset hypertension or with previously elevated BP not currently meeting the threshold for treatment.	Consensus	

Hypertension is a largely asymptomatic condition and is usually detected through systematic or opportunistic screening in healthcare settings.[3] It is not confined to middle age and later life: 7% of people aged 18–24 and 14.2% of those aged 25–34 in Australia have hypertension (13% of First Nations people aged 18–24), and young adult hypertension can cause significant CVD-related morbidity and mortality later in life — underscoring the value of measuring BP at every age, not just at screening intervals tailored to older adults.[12,57] Evidence on optimal screening intervals is limited, so the intervals described reflect ESG judgement rather than direct comparative trial data.

Screening frequency can be guided by age and CV risk:

- Adults under 40: at least every three years.

- Adults 40 and over: annually.
- Increased cardiovascular risk, family history of early-onset hypertension or previously elevated BP: annually regardless of age.

For those under the age of 18, the *Australian guideline for hypertension in children and adolescents* can be used for guidance, available at Hypertension Australia.

For the recommended office measurement modality, see *Office BP measurement*.

6.2 Measurement settings

Clinic or office BP measurement remains the primary detection and diagnostic pathway, as it is the only setting reliably linked to confirmation, risk assessment and follow-up.[58] Measure BP at routine clinical encounters as part of standard care, adjusting frequency for visit patterns and clinical priorities; use telehealth where in-clinic measurement cannot occur. General practice offers the best opportunity for case detection, since around 80% of individuals living in Australia attend a GP each year.[59]

Screening may be opportunistic (BP measured whenever a person presents for any reason) or systematic (a predetermined process of selection, invitation, follow-up and feedback at defined intervals).[3,60] Systematic screening may improve BP control compared with no or opportunistic screening, but current evidence does not show a clear clinical or economic advantage over opportunistic screening.[60]

Community-based measurement, such as in pharmacies, can support detection and engagement, provided it uses validated devices, trained personnel and standardised protocols. It is particularly valuable for reaching people who do not routinely attend health services — in international screening campaigns, up to one quarter of participants report never having had their blood pressure measured before.[61]

The benefit of community-based measurement depends on closing the loop. A person presenting with an elevated community reading should be formally assessed using standardised office or out-of-office measurement, rather than having the screening step repeated. Community readings are a prompt for assessment, not a diagnosis.

Detection alone is insufficient.[62,63] Screening that is not linked to confirmatory assessment, treatment initiation and follow-up has limited impact on long-term control, so community-based measurement should be implemented with defined pathways into ongoing care.

Assessment and diagnosis of hypertension integrate standardised BP measurements with clinical evaluation, including a detailed medical history, physical examination, assessment of CVD risk, evaluation of end-organ damage, and investigation of possible secondary causes where clinically indicated.[64]

The diagnostic process aims to:

- determine BP level and confirm persistent elevation using standardised protocols

- identify modifiable and non-modifiable CV risk factors
- detect end-organ damage or related conditions
- investigate secondary causes when indicated
- guide decisions on timing and type of treatment initiation.

6.3 Blood pressure measurement protocol

Recommendation	Strength of recommendation	Certainty of evidence
We recommend automated office blood pressure (AOBP) measurement with a validated upper-arm device as the preferred office method, applying method-specific thresholds. Where AOBP is not available, we recommend standard office blood pressure measurement.	Consensus*	
We recommend that hypertension not be diagnosed on the basis of a single office visit. A diagnostic threshold is only met when the averaged readings across at least two separate, consecutive occasions are at or above the diagnostic thresholds.	Consensus	
Hypertension may be diagnosed at a single visit when office BP is ≥180 mm Hg systolic and/or ≥110 mm Hg diastolic and there is evidence of CVD, provided the reading is confirmed by repeat standardised measurement at that same visit and no reversible cause of acute BP elevation, such as pain, acute anxiety, or recent exertion or stimulant use is identified.	Consensus	
We recommend confirming suspected hypertension with out-of-office measurement using ambulatory (ABPM) or home (HBPM) monitoring, applying method-specific thresholds.	Adopted recommendation AHA/ACC 2025[2] and ESC 2024[3]	
	Class I AHA/ACC 2025[2]; ESC 2024[3]	Level A AHA/ACC 2025[2] Level B ESC 2024[3]
We recommend including HBPM in the long-term follow-up of treated hypertension.	Adopted recommendation AHA/ACC 2025[2]; ESH 2023[4]	
	Class I AHA/ACC 2025;[2] ESH 2023[4]	Level A AHA/ACC 2025[2]

		Level B ESH 2023[4]

*This recommendation is adapted from *Automated office blood pressure measurement: a Hypertension Australia and National Hypertension Taskforce of Australia position statement*.[7]

6.3.1 Blood pressure measurement

Accurate BP measurement is the essential first step in diagnosing hypertension, and depends on four things every time, in every setting: a validated device, a standardised protocol, trained staff, and multiple readings, averaged — rather than a single reading. Where any of these isn't feasible, document the deviation and treat the result with appropriate caution rather than as definitive. These principles apply throughout this guideline and are not repeated in full at each measurement method below.

BP may be measured in the office using standardised conventional or automated office BP (AOBP), or in out-of-office settings using ambulatory BP monitoring (ABPM) or home BP monitoring (HBPM). The choice should be guided by clinical indication, diagnostic or management goals, availability, cost, and patient preference.

Blood pressure values obtained using different measurement modalities are not directly interchangeable. The method used to measure BP significantly influences the reading obtained, and applying a single universal threshold across all methods risks both over- and under-diagnosis of hypertension. Thresholds differ by measurement modality and should be interpreted accordingly; the equivalent measures are reported in each section. The overall diagnostic and confirmation pathway is illustrated in the BP classification and risk-based treatment pathway diagram in Hypertension and CVD risk assessment.

Key point — what the numbers in this guideline mean

- Every blood pressure value in this guideline is an average of at least two standardised measurements taken on at least two separate occasions, unless a single reading is explicitly specified. A single high reading does not establish a diagnosis; a single reading at target does not establish control.
- Reaching BP target is defined when an average of at least two standardised measurements taken on at least two separate occasions is at or below target

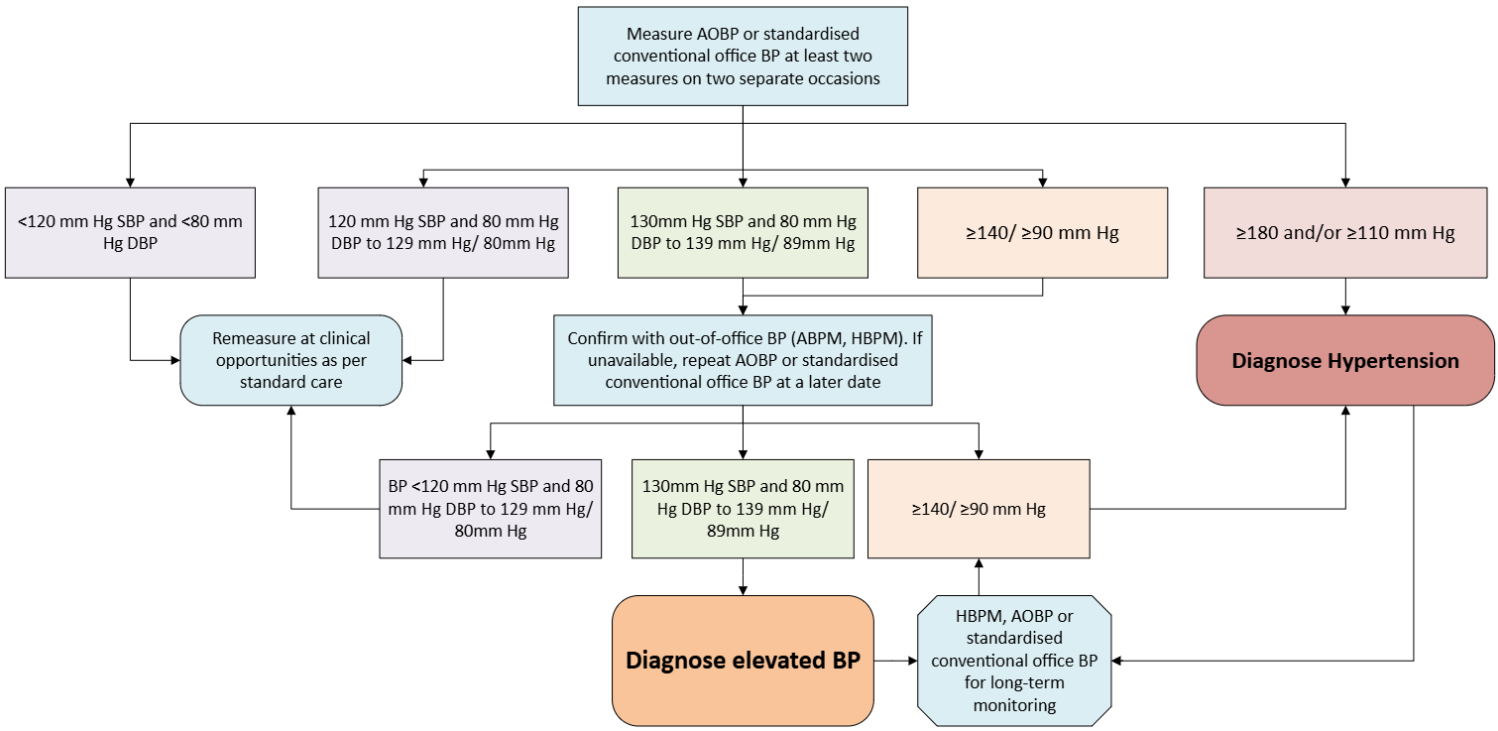


Figure 2: Steps involved in measuring blood pressure and diagnosing hypertension

6.3.2 Office BP measurement

Standardised conventional office (clinic) BP is the most used method for detecting and managing hypertension and is well integrated into Australian primary care. It shows high specificity but only low-to-moderate sensitivity for detecting elevated BP and is less accurate for masked hypertension.[65] Therefore, diagnosis should not rely on a single visit except where BP is severely elevated; confirm with repeated measurements or out-of-office monitoring instead. Globally, only around 10% of devices in use are validated, and this predominantly affects home BP monitoring (HBPM), where patient-owned devices are often unvalidated — confirming device validation status before HBPM is undertaken is therefore an important part of accurate HBPM assessment.[66] Listings are available at www.stridebp.org and www.validatebp.org.[67] Table 3 has the protocol for standard office BP.

Table 3: Protocol for standard office BP measurements

Topic	Practice point	Guidance/key details
Equipment and validation	Use a validated automated electronic device	Registry-listed automated device (e.g. STRIDE BP, ValidateBP); maintain and calibrate per manufacturer instructions; stock a full range of cuff sizes
Automated devices and AF	Check rhythm before AOBP	Palpate radial pulse before measuring; if irregular → 12-lead ECG to confirm AF;[68] most automated devices are not validated if AF is

		present; if readings are inconsistent or implausible, use an AF-validated automated device or manual auscultation.
Cuff selection and placement	Select and position cuff correctly	Select cuff size according to bare mid-arm circumference, following the device manufacturer's cuff-size range; use the cuff whose range includes the measured circumference. Where arm circumference falls outside the range of available cuffs, use a validated alternative (e.g. conical cuff, or a validated wrist device if upper-arm measurement is not feasible). Measure on the bare upper arm, supported at heart level. Centre the bladder over the brachial artery, with the lower edge approximately 2–3 cm above the elbow crease.
Cuffless devices	Avoid cuffless wearables	Do not use rings, wristbands or other cuffless devices to diagnose or manage hypertension; accuracy evidence is insufficient
Patient preparation and positioning	Standardise conditions and posture	Quiet, comfortable area; no smoking, caffeine, food, relevant medications or exercise for ≥30 min where practicable; empty bladder; seated 5 min before first reading, back supported, feet flat on the floor, legs uncrossed; arm bare and supported at heart level; no talking, phone or television during or between readings
Inter-arm difference	Check both arms initially	Measure both arms at first assessment; use the arm with the higher measured BP thereafter; systolic difference >20 mm Hg → consider vascular assessment
Documentation and interpretation	Average repeated readings	Take three consecutive readings; use the average of three, or at least two;[7,64] record averaged BP, arm used and measurement conditions; if readings vary by more than 10 mm Hg systolic or 6 mm Hg diastolic, have the individual rest quietly for 5 minutes then re-measure

Abbreviations: AF, atrial fibrillation; AOBP, automated office blood pressure; BP, blood pressure; ECG, electrocardiogram; mm Hg, millimetres of mercury.

Automated office blood pressure measurement

AOBP, performed attended or unattended (unattended preferred where feasible), is the recommended office-based method: an automated device obtains multiple readings under standardised conditions and averages the results. AOBP requires a device specifically capable of taking and averaging a programmed series of readings while the patient sits quietly; conventional electronic monitors cannot perform this function. Devices available in Australia at the time of writing include:[7]

- Omron HEM-907
- Microlife WatchBP Office
- A&D Medical UM-212 BLE

- Dinamap pro-Care 400

Unattended AOBP modestly reduces the white-coat effect, producing readings approximately 2–3 mm Hg lower than attended measurements, but agreement with 24-hour ABPM is modest, so it should not replace ABPM for diagnostic confirmation where available.[69]

This guideline recommends the AOBP protocol developed by Hypertension Australia and the National Hypertension Taskforce, approximating mean daytime ambulatory BP and removing the need for clinician-obtained readings.[7] Consistent with this equivalence to mean daytime ambulatory BP, the AOBP hypertension diagnostic threshold is lower than the conventional office threshold. The AOBP protocol is presented in Table 4 and Figure 3; the equivalent diagnostic threshold is in Table 5.

Table 4: AOBP protocol

Step	Action	Key details
1. Set up	Prepare the space	• Quiet room or dedicated bay in the waiting area • Ideally performed by a trained operator other than the doctor
	Prepare the individual	• Seat the patient with back supported, legs uncrossed, feet flat on the floor, and the arm supported with the cuff at heart level • Fit a correctly sized cuff over a bare arm (preferred) or thin sleeved clothing — measure mid-arm circumference if cuff size is uncertain • Position the cuff with the lower edge 3 cm above the elbow crease where possible and the bladder centred over the brachial artery • Instruct the patient to sit quietly and remain still throughout, with no distractions (phone off, no talking) • Explain that the device will automatically take three readings at 30-second intervals after a 5-minute rest • Check whether postural hypotension is suspected; if so, prepare for two standing readings at 30-second intervals after 1 minute of standing
2. Start	Brief, then step away	• Confirm the patient understands the instructions • Start the device with a single button press • Operator leaves the area
3. Measure	Unattended recording	• 5-minute rest, patient seated and still • Device records three readings at 30-second intervals • Operator returns for standing readings if required
4. Record	Report the average	• Average of the three seated readings (plus two standing, if taken)

Abbreviations: DBP, diastolic blood pressure; mm Hg, millimetres of mercury; SBP, systolic blood pressure.

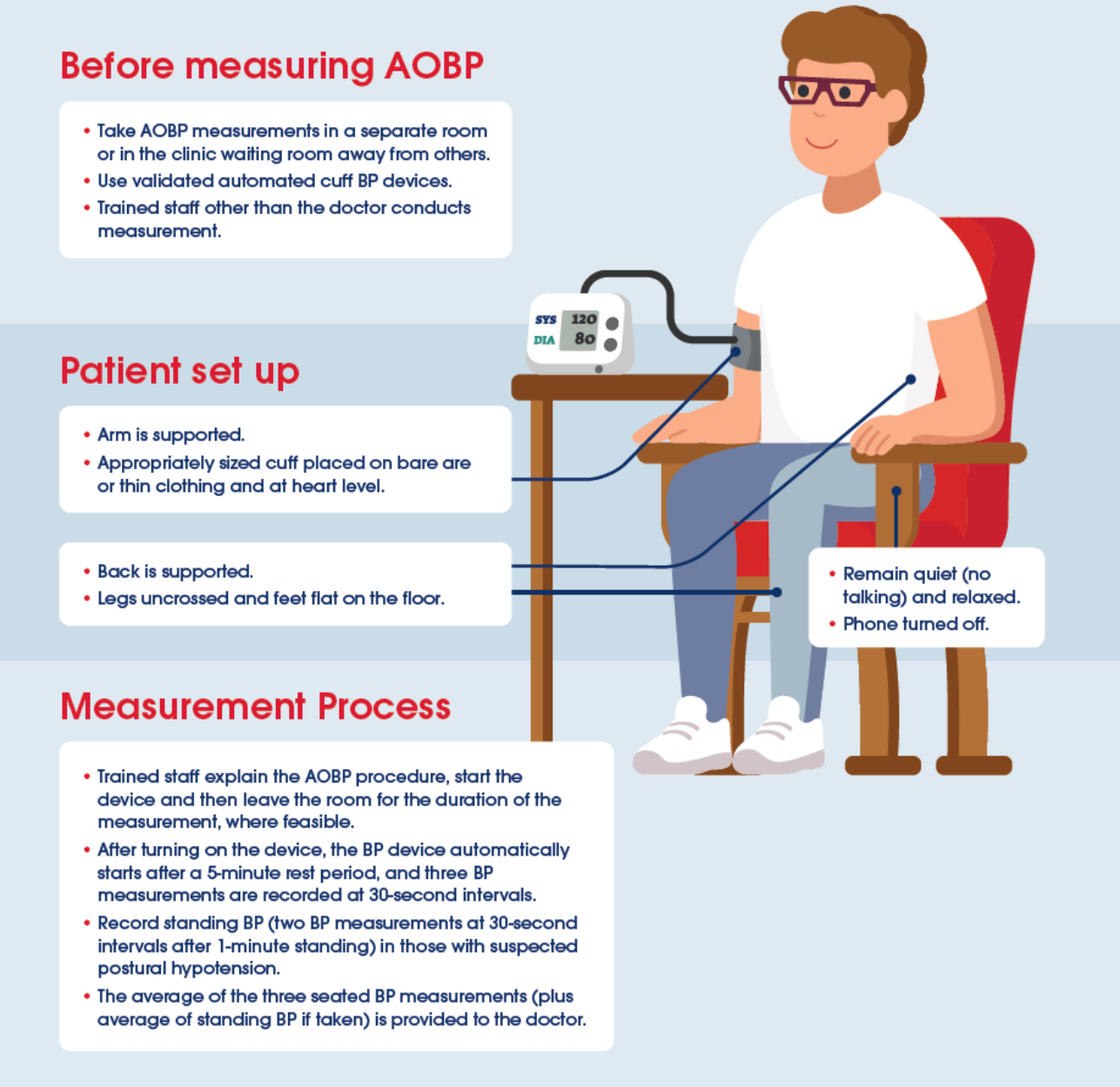


Figure 3: AOBP measurement protocol and diagram

Table 5: AOBP equivalent diagnostic threshold

Measurement method	SBP (mm Hg)	DBP (mm Hg)
Office BP	≥140	≥90
AOBP	≥135	≥85

Abbreviations: AOBP, automated office blood pressure; BP, blood pressure; DBP, diastolic blood pressure; SBP, systolic blood pressure.

Manual auscultatory BP

Manual auscultatory measurement remains an important alternative when automated methods are unavailable, contraindicated, or produce unreliable or 'error' readings, and requires appropriate training and adherence to a standardised protocol (Table 6).

Table 6: Manual auscultatory measurement protocol

Step	Action
1	Estimate systolic pressure by palpating the radial pulse while inflating the cuff, noting the pressure at which it disappears; inflate a further 30 mm Hg above this. Fully deflate, wait 30 seconds, then re-inflate to this level for the true reading.
2	Deflate at 2–3 mm Hg/beat or less while auscultating over the brachial artery in the antecubital fossa.
3	Where BP is measured by auscultation using an aneroid sphygmomanometer, record SBP and DBP to the nearest 2 mm Hg: systolic at phase I (the first of two consecutive Korotkoff sounds, even if they briefly disappear — the auscultatory gap) and diastolic at phase V (disappearance of sound), using phase IV (muffling) only where sound continues to 0 mm Hg.
4	Wait 30 seconds before repeating measurements on the same arm.

Abbreviations: BP, blood pressure; DBP, diastolic blood pressure; mm Hg, millimetres of mercury; SBP, systolic blood pressure.

6.3.3 Out-of-office BP measurement

Out-of-office BP measurement methods include ambulatory BP monitoring (ABPM), home BP monitoring (HBPM), community-based methods such as pharmacy and kiosk readings, and opportunistic screening events. This guideline prioritises ABPM and HBPM for diagnosis and monitoring. Other methods have a growing role, particularly community pharmacy, where expanding scope of practice in some jurisdictions now extends beyond screening (see Multidisciplinary and team-based care).

When available, out-of-office measurement is recommended. It better reflects usual BP, improves diagnostic accuracy and treatment decisions, and is necessary to identify white-coat and masked hypertension, which clinic readings alone cannot reliably detect.[70] See Hypertension-related phenotypes for information on white-coat and masked hypertension. However, feasibility can be a barrier. HBPM may not be feasible for all individuals, with reported barriers including lack of access to validated devices, insufficient knowledge of correct measurement technique, and anxiety associated with frequent monitoring.[71] ABPM is not available in all settings, with access particularly limited in rural and regional practice, and cost matters — patients may face out-of-pocket fees for ABPM. Where out-of-office

measurement genuinely isn't feasible, confirm hypertension through repeated AOBP or standard office BP measurements instead.

Out-of-office measurement is indicated for suspected white-coat or masked hypertension, major differences between office and out-of-office BP, autonomic, postural, post-prandial, or drug-induced hypotension, resistant hypertension, and assessment of nocturnal BP patterns.[64]

For office BP (AOBP or standard office BP) of SBP 130–139 mm Hg and/or DBP 80–89 mm Hg, hypertension should be confirmed with out-of-office monitoring. ABPM is the diagnostic reference standard and preferred method because it also detects patterns such as nocturnal hypertension and transient hypotension.[3] HBPM is an appropriate alternative when ABPM is unavailable, poorly tolerated, or impractical, and supports ongoing monitoring and treatment assessment when performed with a standardised protocol and periodic review. The methods are complementary: both provide multiple readings, reduce short-term variability, and improve diagnostic accuracy.[64] If neither is feasible, diagnosis should rely on repeated AOBP or conventional standardised office measurements. In individuals at high CV risk, treatment should not be delayed if out-of-office measurement cannot be obtained promptly.

Wearable cuffless BP devices, such as wristbands or rings, are increasingly available. However, at the time of writing they lack sufficient validation, so their role in BP assessment for this guideline is unclear and they are not recommended for clinical practice.[72,73]

Ambulatory blood pressure monitoring (ABPM)

ABPM records BP over 24 hours during usual activities and is the reference standard for diagnostic confirmation, capturing both daytime and night-time BP with superior predictive value for CV outcomes. Medicare reimbursement is currently limited to specific indications (it covers confirming the diagnosis in those with an office BP of ≥140 mm Hg SBP and ≥90 mm Hg DBP who have not commenced antihypertensive therapy) — broader use for ongoing management would need expanded Medicare funding.

Access can be limited by equipment availability, trained personnel, referral pathways, cost and patient acceptability (sleep disturbances, anxiety about frequent measurements). Barriers may be greater in rural and remote areas, potentially contributing to inequities in care.

Accurate interpretation requires a standardised protocol, including appropriate measurement frequency and inclusion of both daytime and night-time readings. These are documented in the

Practice points below. It is important to recognise that ABPM typically records lower BP values than standard office measurement. Consequently, lower diagnostic thresholds are used to define hypertension with ABPM (see Table 7).

Table 7: Office BP thresholds and corresponding ambulatory BP monitoring thresholds for hypertension

BP measure	Office BP	24-hour ABPM	ABPM Awake	ABPM Asleep
SBP (mm Hg)	≥140	≥130	≥135	≥120
DBP (mm Hg)	≥90	≥80	≥85	≥70

Practice points

General

- Use validated devices programmed to record BP automatically at preselected intervals for 24 hours; individuals record activities, symptoms, meals, drug intake times, sleep times, or unusual events.
- Balance measurement interval accuracy with tolerability (15–30 minutes daytime, 20–60 minutes night-time).
- Aim for at least 20 daytime and 7 night-time readings.
- Average all measurements (≥20 daytime, ≥7 night-time) to obtain 24-hour, daytime (awake) and night-time (asleep) BP.

Home blood pressure monitoring (HBPM)

HBPM should be offered when ABPM is unavailable, not tolerated, or inaccessible, and may support patient engagement and self-management. Guide patients using the Heart Foundation blood pressure measuring guide and diary.

Compared with office BP, HBPM provides multiple readings over several days, improving reproducibility and prognostic value, and is more effective at identifying phenotypes associated with postural drop, autonomic neuropathy, and diurnal variation.[74] However, agreement with ABPM is moderate and some individuals with white-coat or masked hypertension may remain misclassified, so HBPM complements but does not replace ABPM for diagnostic confirmation.[75] HBPM typically records lower BP values than standard office measurement; corresponding hypertension thresholds are shown in Table 8.

Table 8: Office BP thresholds and corresponding HBPM thresholds for hypertension

Measurement method	SBP (mm Hg)	DBP (mm Hg)
Office BP	≥140	≥90

HBPM	≥135	≥85

Adherence to standardised technique is essential, but many people don't follow recommended procedures, often due to limited education, poor technique, and low use of validated devices (only 26% of devices available in Australian pharmacies are validated, and around 51% of users have a validated device).[76-80] A standardised HBPM protocol is described in Table 9.

EQUIPMENT

- Use a validated automated device with an appropriately sized cuff, preferably with memory storage.

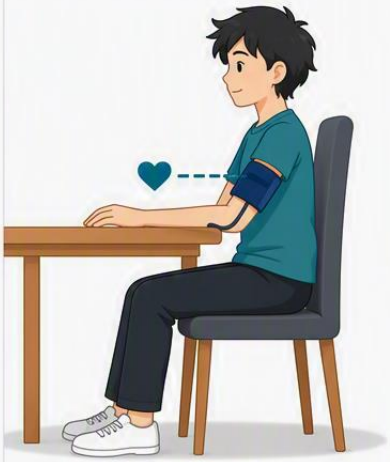
VALIDATED DEVICE

PREPARATION

- Take measurements as advised by the clinician — for example, before a scheduled appointment or after a medication change.
- Measure before taking relevant medications, before food, caffeine, or exercise, and after emptying the bladder.
- Sit quietly for 5 minutes beforehand, with no talking or distractions.

POSITION

- Sit with the back supported, feet flat on the floor, and legs uncrossed.
- Rest the arm supported at heart level, with the cuff on the bare upper arm.



MEASUREMENT SCHEDULE

Measure at approximately the same times each morning and evening.

MORNING EVENING

Take two readings 1 minute apart on each occasion (4 readings per day).

1st reading 2nd reading

7 DAY MONITORING PERIOD

Continue for 7 days (minimum 5 days).

RECORDING

Record all values in a paper diary or electronic spreadsheet, noting anything that may explain unusual readings (for example, caffeine or exercise beforehand).

Date	Time	SBP (mmHg)	DBP (mmHg)	Pulse (/min)	Notes
Day 1	07:00	123	78	72	Slept well
Day 1	07:01	120	76	70	–
Day 1	20:00	128	80	74	Coffee in afternoon
Day 1	20:01	126	78	72	–
...	...	...	...	...	...

INTERPRETATION

1 **Discard all day 1 readings and average the remainder.**
This can be done by the patient or by their clinician.

Average days 2–7 (minimum 5 days)

Table 9: Home blood pressure monitoring standardised protocol

Equipment	• Use a validated automated device with an appropriately sized cuff, preferably with memory storage.
When to monitor	• Monitor over a 7-day period (minimum 5 days), timed as advised by the clinician — for example, in the week before a scheduled appointment or following a medication change.
Before each reading	• Empty the bladder, and avoid food, caffeine and exercise beforehand. • If taking BP medication, measure before the dose is due, then take the medication as usual. • Sit quietly for 5 minutes, with no talking or distractions.
Position	• Sit with the back supported, feet flat on the floor, and legs uncrossed. • Rest the arm supported at heart level, with the upper arm bare.
Measurement schedule	• Measure twice daily, at around the same time each morning and evening. • Take two readings 1 minute apart on each occasion — four readings per day, or 28 over 7 days. • Continue for 7 days (minimum 5 days).
Recording	• Record all values in a paper diary or electronic spreadsheet, noting anything that may explain an unusual reading. • Also record any readings taken while standing or when symptoms of low BP occur, such as dizziness on standing, and note the time.
Interpretation	• Discard all day 1 readings and average the remainder.

Practice points

General

- Using a validated upper-arm device, take two readings one minute apart after sitting quietly for 5 minutes, twice daily (morning and evening) for 7 days (minimum 5); discard all day 1 readings and average the remainder.

Rural, remote and regional

- Community and pharmacy-based BP screening supports opportunistic detection of hypertension when clinical access is limited; elevated readings should be confirmed using AOBP or a standard office BP protocol.
- Where ABPM is unavailable or impractical, HBPM using a validated upper-arm device and standardised protocol is the preferred alternative for confirming diagnosis and monitoring treatment; repeated AOBP can be used if neither is feasible.

- Telehealth and remote monitoring can support BP monitoring, device instruction, and follow-up when in-person or specialist access is limited, but they do not replace an initial face-to-face assessment.

First Nations peoples

- First Nations peoples experience hypertension at disproportionately high rates and at younger ages. Opportunistic and community-led BP screening from the age of 18 is strongly encouraged.[14,81]
- Measure BP in a culturally safe, private setting, ideally with an Aboriginal and Torres Strait Islander Health Practitioner or a health professional of the same cultural background.[82]

Women

- Routine BP measurement on the arm ipsilateral to breast cancer surgery is no longer contraindicated; where feasible, or where repeated monitoring is needed, preferentially use the unaffected arm to minimise discomfort and anxiety.
- For pregnancy and postpartum (out of scope for this guideline), only 7% of BP devices available through Australian pharmacies in 2025 were validated for pregnancy, so antenatal care should use validated devices only.[80] Refer to the 2023 SOMANZ *Hypertension in Pregnancy* guideline for further guidance.[79,80]

Older and frail adults

- Where an older adult cannot perform HBPM independently, HBPM by a trained carer or family member is a valid alternative to ABPM.[83]
- Modify standard seated measurements where needed (e.g. inability to sit for 5 minutes, postural instability), using clinical judgement and documenting any deviation from protocol.

6.4 Medical history

Most people with hypertension are asymptomatic, but certain signs or symptoms may point to secondary hypertension or complications requiring further evaluation. Take a comprehensive medical and family history, focused on BP control, risk factors, end-organ damage, and potential secondary causes.[64] Details of a comprehensive medical history are documented in Table 10.

Table 10: Elements of medical history to assess

Domain	Key elements to assess	Significance
Part 1 – Characterise **Establish the hypertension, the person, and their absolute risk**		

Personal history of hypertension	• Time and circumstances of first diagnosis, including previous medical screening or hospitalisation records • Stability or rapid progression of BP elevation • HBPM or ABPM results (current and past) • Current and previous antihypertensive therapy, including dose, efficacy, side effects, and reasons for discontinuation • Adherence to prescribed treatment and barriers to adherence • Previous hypertension in pregnancy or pre-eclampsia	Establishes chronicity and treatment resistance Prior pre-eclampsia confers lifelong elevated CV risk
Family history	• Hypertension, premature CVD, stroke, kidney disease, or early-onset CHD (<55 years in men, <65 years in women) • Genetic or familial kidney or endocrine disease (e.g. polycystic kidney disease) • Family history of dyslipidaemia or premature death from cardiovascular causes	Family history of premature CVD is absent from the risk equation due to face validity concerns — a well-documented family history is a reclassification factor for consideration of a higher risk category. Familial hypercholesterolaemia is itself a high-risk condition Familial kidney or endocrine disease indicates a heritable secondary cause Familial hypertension or dyslipidaemia indicates familial predisposition to primary hypertension
Assess CVD risk	• Refer to Hypertension and CVD risk assessment	Determines treatment threshold and intensity
Lifestyle and behavioural risk factors	• Smoking history and exposure to environmental tobacco smoke • Alcohol consumption and drinking patterns • Dietary patterns (including sodium and potassium intake, and fruit and vegetable consumption) • Weight history, recent weight gain or loss • Physical activity level and sedentary behaviour • Recreational drug use • Sleep quality, history of snoring or suspected sleep apnoea (including partner's report) •	Identifies modifiable contributors and potential secondary causes. Severe mental illness is a reclassification factor in the *Australian Guideline for assessing and managing cardiovascular disease risk*.
Other medical conditions and medications	• Diabetes, dyslipidaemia, CKD, gout, obstructive sleep apnoea, erectile dysfunction, cancer survivorship	Comorbidities guide drug choice CKD is a reclassification factor in the *Australian Guideline for*

	• Psychosocial stress, depression, anxiety, or social isolation • Use of medicines known to elevate BP (e.g. NSAIDs, COX-2 inhibitors, sympathomimetics, corticosteroids, oestrogens, immunosuppressants, anticancer agents, nasal decongestants, complementary or non-prescription medicines)	*assessing and managing cardiovascular disease risk*. Moderate to severe CKD is a clinically determined high risk.
Part 2 – Determine history, symptoms or signs of HMOD		
Brain and eyes	• Abnormalities of the optic fundi (hypertensive retinopathy [mild: generalised or focal arterial narrowing, arteriovenous nicking, or copper wiring; moderate: retinal haemorrhages, micro-aneurysms, cotton-wool spots, or hard exudates; severe: moderate signs plus swelling of the optic disc][84]; or diabetic retinopathy), morning headache, dizziness, syncope, impaired vision, transient ischaemic attack, ischaemic and haemorrhagic stroke, cognitive impairment, or memory decline	May indicate hypertension-mediated organ damage or established CVD – refer to Assessing CVD risk
Heart	• Chest pain, dyspnoea, palpitations, syncope, oedema, history of MI, coronary or valve interventions, AF, or heart failure	
Kidneys	• Nocturia, haematuria, polyuria, recurrent urinary tract infection	
Peripheral arteries	• Claudication, cold extremities, rest pain, ulcers, or previous revascularisation	
Part 3 – Determine history suggestive of secondary hypertension		
Atypical onset or course	• Early-onset hypertension (<40 years) or sudden/worsening hypertension in older adults • Poorly controlled blood pressure on three or more BP-lowering medications	May indicate secondary cause of hypertension, see Secondary causes of hypertension
Renal	• Recurrent renal or urinary tract disease • Known or familial kidney disease	
Hypokalaemia	• Spontaneous or diuretic-induced hypokalaemia, muscle weakness, or tetany (possible hyperaldosteronism)	
Paroxysmal symptoms	• Episodic severe headache, sweating, palpitations, pallor	

Endocrine features	• Symptoms or findings consistent with thyroid or parathyroid disease • Cushingoid features or long-term corticosteroid use	
Reproductive and hormonal	• Pregnancy history, menopausal status, oral contraceptive or hormone therapy use	

Abbreviations: ABPM, ambulatory blood pressure monitoring; AF, atrial fibrillation; BP, blood pressure; CHD, coronary heart disease; CKD, chronic kidney disease; COX-2, cyclooxygenase-2; CV, cardiovascular; CVD, cardiovascular disease; HBPM, home blood pressure monitoring; HMOD, hypertension-mediated organ damage; MI, myocardial infarction; NSAIDs, non-steroidal anti-inflammatory drugs.

6.4.1 Physical examination and laboratory investigations

Physical examination and laboratory investigations are essential in hypertension evaluation, linking elevated BP readings to end-organ involvement, distinguishing primary from secondary hypertension, and informing individual CVD risk to guide timely, individualised management. Laboratory tests further support diagnosis by uncovering secondary causes and target-organ damage not yet clinically apparent, and by establishing a baseline before starting or intensifying therapy — informing safe medication selection, dosing, and monitoring for adverse effects or disease progression over time. Kidney function, electrolytes, glucose/HbA1c, lipids and urinalysis can reveal renal disease, endocrine disorders, diabetes and dyslipidaemia, all of which influence cardiovascular risk and treatment choices. Guidance on physical examination is presented in Table 11, and recommended laboratory tests in Table 12.

Physical examinations

Table 11: Physical examinations for hypertension-mediated organ damage and secondary hypertension

Physical examination	
Signs of secondary hypertension and/or organ damage	• Pulse rate, rhythm and character • Evidence of cardiac failure • Evidence of arterial disease (e.g. carotid, renal, abdominal or femoral bruits, abdominal aortic aneurysm, absent femoral pulses, radio-femoral delay) • Evidence of brain damage (e.g. ischaemic or haemorrhagic stroke, lacunar infarcts, microbleeds, white matter disease/leukoaraiosis) • Palpation of enlarged kidneys (polycystic kidneys) • Abnormalities of the optic fundi (e.g. hypertensive retinopathy – mild: generalised or focal arterial narrowing, arteriovenous nicking, or copper wiring; moderate: retinal haemorrhages, micro-aneurysms, cotton-wool spots, or hard exudates; severe: moderate signs plus swelling of the optic disc; or diabetic retinopathy)[84] • Evidence of endocrine disease (e.g. Cushing's syndrome, thyroid disease)
Evidence of obesity	• Waist circumference (cm) measured in standing position midway between the lower border of the costal margin and the uppermost border of the iliac crest

	• Calculate BMI: body weight without shoes divided by height^2 (kg/m^2)

Abbreviations: BMI, body mass index.

Table adapted from *Guideline for the diagnosis and management of hypertension in adults 2016*.[64]

Laboratory tests

Table 12: Initial laboratory investigations — all patients

#	Investigation	Who/when	Practice points
Urine testing – stepwise			
1	Urine dipstick (blood and protein)	All patients — initial screen	If abnormal, go to 2. Long-standing diabetes: go to 2 regardless of dipstick result
2	Urine albumin–creatinine ratio (uACR)	Abnormal dipstick, or long-standing diabetes	First-void spot urine preferred; random spot acceptable. Interpret against Table 13. If macroalbuminuric (male >25, female >35 mg/mmol), go to 3
3	Urine protein–creatinine ratio (uPCR)	uACR in macroalbuminuria range	Quantifies proteinuria (>500 mg/day). Also for monitoring where albumin-specific measures unavailable. 24-hour protein only where precise quantification is required
Blood tests and ECG – all performed at baseline, in parallel with urine testing			
HbA1c		All	Screening for diabetes No fasting required
Lipids – serum total cholesterol, LDL, and HDL cholesterol, and triglycerides		All	Non-fasting sample acceptable
Renal function – serum urea, electrolytes and creatinine (with eGFR)		All	Includes potassium, required to interpret the ARR
Aldosterone–renin ratio (ARR)		All adults with confirmed hypertension, ideally before antihypertensives are started	Screen for primary aldosteronism. See Primary aldosteronism
12-lead ECG		All	Detection of AF, left ventricular hypertrophy and evidence of previous ischaemic heart disease

Abbreviations: AF, atrial fibrillation; ARR, aldosterone–renin ratio; ECG, electrocardiogram; eGFR, estimated glomerular filtration rate; HbA1c, glycated haemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; uACR, urine albumin–creatinine ratio; uPCR, urine protein–creatinine ratio.

Table adapted from *Guideline for the diagnosis and management of hypertension in adults 2016*.[64]

Table 13: Approximate equivalents between ACR, PCR, 24-hour protein excretion and reagent strip categories

Category	ACR (mg/mmol)	Albumin excretion (mg/day)	PCR (mg/mmol)	Protein excretion (mg/day)	Reagent strip
Microalbuminuria	Male 2.5–25, Female 3.5–35	30–300	Male 4–40, Female 6–60	50–500	Trace to +1
Macroalbuminuria	Male >25, Female >35	>300	Male >40, Female >60	>500	≥+1

Abbreviations: ACR, albumin–creatinine ratio; PCR, protein–creatinine ratio.

Further diagnostic tests may be performed based on clinical suspicion of organ damage, CVD, or CKD, after a comprehensive medical history and physical examination.[64] Additional diagnostic tests are documented in Table 14.

Table 14: Additional diagnostic tests for selected patients

Domain	Investigation	Indication
CVD	Echocardiography	To diagnose left ventricular hypertrophy, or where left atrial dilation or concomitant heart disease is suspected
CKD	Haemoglobin and/or haematocrit blood tests	Investigation of renovascular causes of hypertension, e.g. fibromuscular dysplasia in young women with hypertension, older patients who may have atherosclerotic renal artery disease, and patients with renal and/or femoral bruit
	Renal artery imaging — duplex ultrasound, nuclear medicine and/or CT angiography	
Cerebrovascular disease	Brain imaging by CT or MRI scan	To assess presence and severity of hypertensive brain disease: e.g. ischaemic or haemorrhagic stroke, lacunar infarcts, microbleeds, white matter disease/leukoaraiosis.
Peripheral artery disease	Ankle–brachial index (ABI)	In those with risk factors for peripheral arterial disease, including people with hypertension and diabetes, vascular bruit, older age and/or smoking. An index ≤0.9 is diagnostic for peripheral arterial disease.[85]

Other	Plasma metanephrines	Preferred screening test for phaeochromocytoma; usually guided by symptoms

Abbreviations: ABI, ankle–brachial index; CKD, chronic kidney disease; CT, computed tomography; CVD, cardiovascular disease; MRI, magnetic resonance imaging.

6.5 Hypertension and CVD risk assessment

Key point — a blood pressure category is not a treatment decision

- Optimal, elevated blood pressure and hypertension define bands of measurement and associated risk. They do not by themselves determine management.
- Treatment decisions combine the blood pressure category with calculated cardiovascular risk. Two people with identical readings may be managed differently: some below the diagnostic threshold need treatment, and some above it will not benefit.
- Both blood pressure and CVD risk are continuous. The categorical boxes in the pathway are decision zones, not absolute cut-offs.

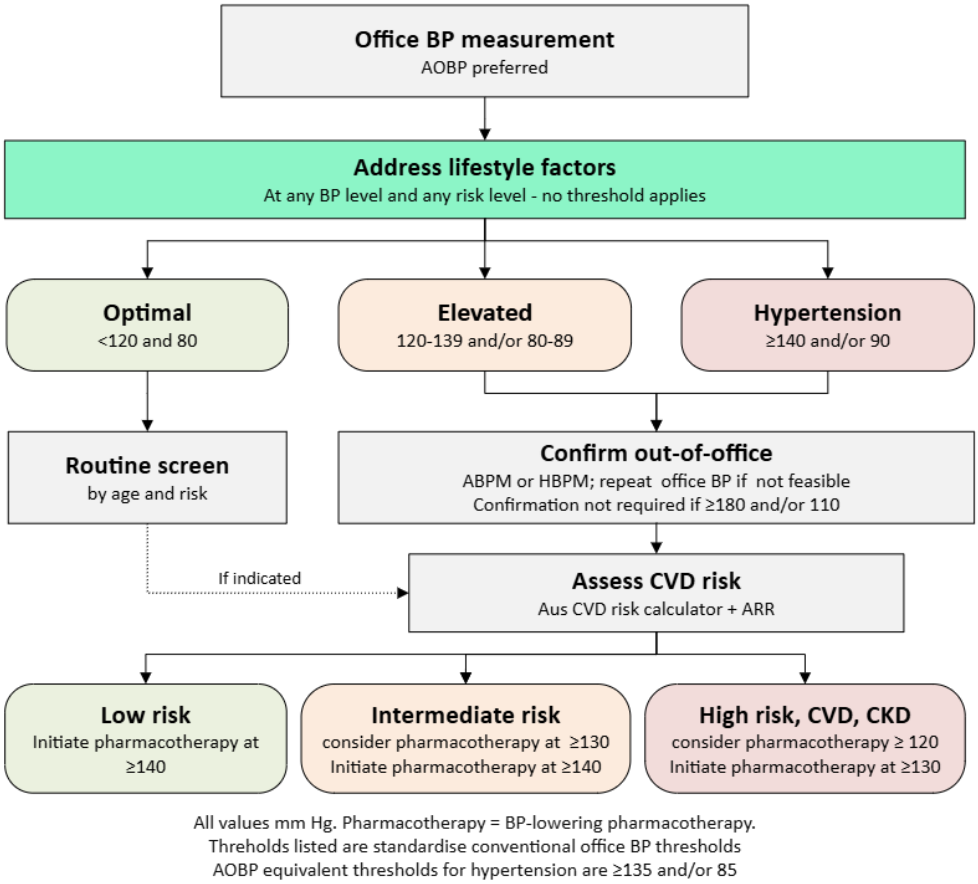


Figure 4: BP classification, confirmation and risk-based treatment pathway

Elevated BP is both a continuous, graded driver of overall CVD risk and a direct cause of hypertension-mediated organ damage (HMOD) — the structural and functional changes it drives in the heart, vasculature, kidneys and brain, underpinning its role in heart failure, atrial fibrillation, CKD and stroke. In adults eligible for primary prevention, BP should be assessed and managed within a risk-based framework using estimated 5-year absolute CVD risk, per the 2023 *Australian Guideline for assessing and managing cardiovascular disease risk* and the Aus CVD Risk calculator, not BP thresholds in isolation. For full risk-assessment methodology, use the Australian guideline and calculator directly.[86]

The pathway above (Figure 4) presents discrete categories to support everyday decision-making; however, both BP and CVD risk are continuous variables, and the categorical boxes represent decision zones rather than absolute cut-offs.

Risk determinants used in this assessment tool include:[87]

- Non-modifiable factors: age, sex, history of atrial fibrillation
- Modifiable factors: systolic blood pressure (SBP), total cholesterol to high-density lipoprotein cholesterol (TC:HDL-C) ratio, diabetes status, use of BP- and lipid-lowering medications, smoking status, and postcode.

Furthermore, reclassification factors may redefine an individual's risk estimate, including ethnicity (up or down), coronary artery calcium (CAC) score (up or down), family history of premature CVD (up), eGFR and uACR (up), and severe mental illness (up).

6.5.1 Assessing CVD risk

#	Step	Detail
1	Confirm eligibility by age/population group	• All adults aged 45–79 years • Aboriginal and Torres Strait Islander adults aged 30–79 years (individual risk-factor assessment recommended from 18–29 years) • People with diabetes aged 35–79 years
2	Check whether calculator-based assessment can be skipped because the person is already at clinically determined high risk	• Do not calculate a score — proceed straight to comprehensive risk-factor management — if the person has any of: ○ Established CVD (prior MI, stroke, TIA, peripheral arterial disease, heart failure, or aortic disease) ○ Chronic kidney disease (eGFR <45 mL/min/1.73 m^2, or persistent uACR >25 mg/mmol in men or >35 mg/mmol in women) ○ Familial hypercholesterolaemia
3	Gather the required inputs for eligible patients	• Comprehensive medical history, fasting lipids, diabetes status, and renal function — and at the same visit, order ARR testing, now recommended in

		all adults with confirmed hypertension (see Secondary causes of hypertension for the testing protocol). • Starting first-line therapy does not need to wait for the ARR result.
4	Enter data into the Aus CVD Risk calculator	At www.cvdcheck.org.au, which aligns with the 2023 *Australian Guideline for assessing and managing cardiovascular disease risk*.[1] Note that this calculator estimates risk over a 5-year horizon, whereas some international guidelines use 10-year or lifetime estimates; a 5-year window can understate risk in younger patients with a high long-term risk-factor burden, so consider a lifetime risk perspective when counselling this group.[88,89]
5	Apply reclassification factors where present	May shift the estimate up or down: • Ethnicity (up or down) • Coronary artery calcium (CAC) score (up or down) • Family history of premature CVD (up) • eGFR and urinary albumin-to-creatinine ratio (uACR) (up) • Severe mental illness (up) • Clinical judgement for First Nations peoples, in whom the calculator may underestimate risk — adjust using the approach in the 2023 *Australian Guideline for assessing and managing cardiovascular disease risk*, factoring in individual clinical, psychosocial and socioeconomic circumstances alongside community CVD prevalence
6	Classify into a risk category using the estimated 5-year risk	• Low: <5% • Intermediate: 5% to <10% • High: ≥10%
7	Use clinical judgement outside the calculator's age range	• Adults <45 years with elevated BP or concerning features, or where risk is likely underestimated within the calculator range: undertake pragmatic risk assessment (BP phenotyping, renal function, lipids, HbA1c, BMI, smoking status, family history, ECG) plus targeted secondary-hypertension screening as clinically indicated. Adults ≥80 years : do not withhold treatment on the basis of age alone — this group carries among the highest absolute CVD/stroke burden, and treatment has shown meaningful benefit even though risk scoring tools were not built for this age band
8	Link the risk category to treatment intensity	• Target BP, lipids and glycaemic parameters together, not BP in isolation, with a pharmacotherapy initiation threshold of ≥130 and/or ≥80 mm Hg for those classified as high risk.

Abbreviations: ARR, aldosterone–renin ratio; BMI, body mass index; BP, blood pressure; CAC, coronary artery calcium; CVD, cardiovascular disease; ECG, electrocardiogram; eGFR, estimated glomerular filtration rate; HbA1c, glycated haemoglobin; MI, myocardial infarction; mm Hg, millimetres of mercury; TIA, transient ischaemic attack; uACR, urine albumin–creatinine ratio.

6.6 CVD risk at any age

The Aus CVD Risk calculator is the recommended first-line tool for assessing CV risk, and where a person is eligible it should be used. Its calibrated age range, however, is 45–79 years (30–79 years for First Nations peoples, 35–79 years for those with diabetes), so a substantial

group of adults sits outside it — and the *2023 Australian Guideline for assessing and managing cardiovascular disease risk* itself provides for clinically determined high risk alongside the calculated estimate.[1] This section sets out how to assess those adults, using standard history, examination and basic investigations, and reserving specialised tests for selected cases where they are not validated for population-level screening.

This approach aligns with the *Obesity and cardiovascular disease: a clinical consensus statement* (National Heart Foundation of Australia, 2026), which defines high atherosclerotic CVD risk through four groups (Figure 5): people with high calculated risk on the Aus CVD Risk calculator (estimated 5-year CVD risk ≥10%); people with clinically determined high risk according to the *2023 Australian Guideline for assessing and managing cardiovascular disease risk*; people with very elevated individual risk factors, such as very high BP, very elevated cholesterol or very high lipoprotein(a) (Lp(a)); and people with subclinical or pre-clinical disease, such as a very high coronary artery calcium (CAC) score.

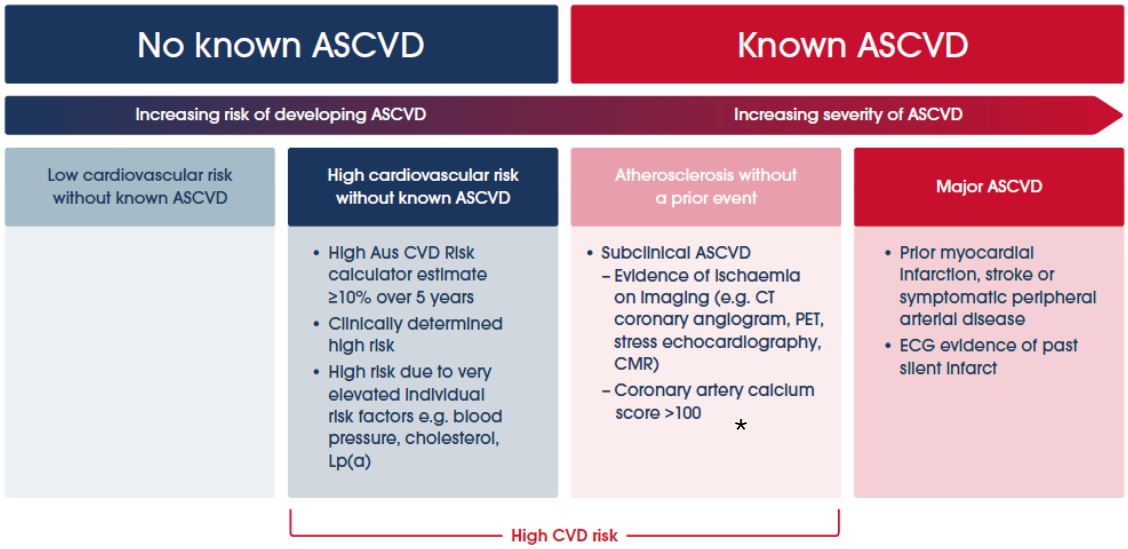


Figure 5: Continuum of atherosclerotic cardiovascular disease risk

*An individual with a CAC score of ≥300 Agatston is suggested to be at equivalent risk of MACE and its components as those with established ASCVD.[90]

6.6.1 Who this pathway applies to

Use this pathway for adults outside the calculator's eligible age range, including those under 45 with elevated BP or concerning features — early-onset hypertension carries higher lifetime CVD risk and mortality. It also applies to eligible adults whose calculated 5-year risk sits at intermediate level (5–<10%) alongside risk-enhancing features, and to adults aged 80 and over, who carry a high absolute CVD and stroke burden and are often undertreated despite safe and meaningful treatment benefit.[43,50,87,91]

Evaluation should include a pragmatic global assessment (BP phenotyping, renal function, lipids, HbA1c, BMI, smoking status, family history, ECG) and, as a first step, targeted testing for secondary causes (see Secondary causes of hypertension).

6.6.2 Features that indicate high risk

The presence of any one of the features in Table 15 is sufficient to classify an adult as clinically determined high risk of CVD and to prompt intensive preventive management, regardless of age or calculator eligibility.

Table 15: Features that indicate high risk

Feature	Detail
Established atherosclerotic CVD	Includes prior myocardial infarction, angina, coronary revascularisation, ischaemic stroke, transient ischaemic attack (TIA), cerebral haemorrhage, peripheral artery disease, or haemodynamically significant stenosis identified on imaging.[92]
Heart failure (including HFpEF) and atrial fibrillation	While not all causes are atherosclerotic in origin, both conditions are associated with elevated CV risk and warrant intensive risk-factor management.[92]
Moderate to severe chronic kidney disease	Defined by a sustained eGFR <45 mL/min/1.73 m^2, or persistent uACR >25 mg/mmol (men) or >35 mg/mmol (women).[87,93]
Familial hypercholesterolaemia, or markedly elevated cholesterol	Includes total cholesterol >7.5 mmol/L. Assessment using the Dutch Lipid Clinic Network Score is recommended. The odds ratio for coronary artery disease in FH ranges from 10.3 (treated) to 13.2 (untreated) compared with non-FH individuals.[94-96]
Severe hypertension	Persistent blood pressure of ≥180/110 mm Hg, which warrants immediate pharmacological treatment irrespective of calculated risk.[2,70,87,97]
Hypertension-mediated organ damage (HMOD)	Includes left ventricular hypertrophy (on ECG or echocardiography), significant albuminuria, hypertensive retinopathy, ischaemic or haemorrhagic stroke, or cerebral small-vessel disease. This is particularly important for younger individuals, where risk calculators may underestimate true individual risk.[92,97]
Early-onset type 1 diabetes	Especially in the presence of long-term hyperglycaemia, diabetic nephropathy and the presence of usual CV risk factors.[98]
Severe obesity with additional cardiometabolic risk factors	A significant direct and indirect driver of premature CVD.

Abbreviations: CV, cardiovascular; CVD, cardiovascular disease; ECG, electrocardiogram; eGFR, estimated glomerular filtration rate; FH, familial hypercholesterolaemia; HFpEF, heart failure with preserved ejection fraction; HMOD, hypertension-mediated organ damage; TIA, transient ischaemic attack; uACR, urine albumin–creatinine ratio.

6.6.3 Advanced testing including the role of Lp(a) and coronary imaging

Lp(a) and coronary imaging (CAC and CTCA) are not BP biomarkers, and evidence linking them to hypertension management specifically is limited. They should not be used routinely to decide when to start or intensify antihypertensive therapy, but can refine risk assessment in selected patients (intermediate risk or strong family history) and support shared decision-

making about lifetime risk and adherence.[99-103] At the time of writing this guideline, neither Lp(a) testing nor CAC/CTCA is rebatable under Medicare.

- Lp(a): reasonable as a once-only measurement in hypertension with a strong family history, or in younger adults with elevated BP. A level ≥50 mg/dL (≥125 nmol/L) indicates higher lifetime CVD risk and supports intensified management of modifiable risk factors.[104-108]
- CAC: most useful at intermediate calculated risk where treatment is uncertain. A score ≥100 Agatston units reclassifies as high risk (≥300 as secondary-prevention level).[90,109] Interpret cautiously in adults under 45, where a zero score can miss soft plaque, and in people on statins, where scores may rise without worse prognosis.
- CTCA: significant coronary artery disease (≥50% luminal stenosis in any major epicardial vessel, including left main) reclassifies as high risk.[110]
- ECG: silent myocardial infarction confers risk equivalent to symptomatic MI and should be managed as established CVD.[111-113]

6.6.4 Practical application

Basic assessment identifies most high-risk individuals; reserve CAC, CTCA, Lp(a) and echocardiography for cases where the result would meaningfully change management. Standard BP thresholds remain the foundation of decision-making. For adults aged 18–44 years with office BP 130–139 mm Hg and/or 80–89 mm Hg, where evidence is limited, use judgement, overall risk profile, and shared decision-making. See Assessing CVD risk for the step-by-step process.

6.7 Secondary causes of hypertension

Secondary hypertension accounts for 5–10% of all hypertension (about 30% in young adults aged 18–40 years), making it an important primary care consideration.[114] It's more commonly suspected with severe, resistant or atypical BP, but these features alone are insufficient — reliably identifying secondary hypertension needs deliberate screening.[115-117] Common causes include primary aldosteronism, renal disease, obstructive sleep apnoea, renovascular, endocrine and medication-related conditions; some are reversible or need specific management, and are more frequent in resistant hypertension.

Evaluation should be guided by clinical features, early or abrupt onset, resistant hypertension, unexpected deterioration, or features suggesting an underlying condition, with history, examination, targeted investigations, and specialist referral where indicated.[2,3,118] Table 16 focuses on recognising key triggers, selecting initial tests, and knowing when to refer.

Table 16: Secondary causes of hypertension – symptoms, signs and investigations

Secondary causes of hypertension	Symptoms	Signs	Investigations

Primary aldosteronism	Often asymptomatic; may report muscle cramps, weakness or headaches.	Hypertension ± spontaneous or diuretic-induced hypokalaemia; treatment-resistant hypertension; incidental adrenal mass.	Measure serum electrolytes, aldosterone, renin and the aldosterone–renin ratio. Refer to specialist services if elevated; or consider trial of MRA treatment.
Renal parenchymal disease	May be asymptomatic; obstructive symptoms, urinary frequency, nocturia, haematuria, family history of polycystic kidney disease, analgesic misuse.	Elevated creatinine or reduced eGFR; abnormal urinalysis (haematuria, proteinuria); kidney structural abnormalities on imaging.	Order serum creatinine, eGFR, electrolytes, urinalysis and urine albumin–creatinine ratio; arrange renal ultrasound when indicated; consider nephrology referral.
Renovascular disease/hypertension	Resistant or abrupt-onset hypertension; possible flash pulmonary oedema, peripheral oedema or worsening renal function.	Abdominal systolic and/or diastolic bruit; asymmetry in kidney size, rise in creatinine after starting an ACE inhibitor or ARB.	Measure serum creatinine and eGFR, perform urinalysis; arrange renal artery imaging (Doppler ultrasound, CT angiography or magnetic resonance angiography) based on clinical suspicion and local availability. Refer to nephrology/vascular specialist if confirmed — revascularisation may benefit fibromuscular dysplasia (especially in younger patients), but is not routinely recommended for atherosclerotic disease over optimal medical therapy.
Obstructive sleep apnoea	Snoring, apnoeic episodes, nocturnal	Obesity, large neck size, Mallampati class 3–4; loss of normal nocturnal BP fall.	Use validated screening tools (e.g. OSA50, Epworth

	choking and gasping, nocturia, daytime sleepiness, fatigue.		Sleepiness Scale, STOP-Bang) to identify high-risk patients; confirm diagnosis with an in-laboratory or home sleep study via GP or specialist referral.
Phaeochromocytoma/paraganglioma	Headaches, palpitations, diaphoresis, anxiety, pallor, abdominal pain, weight loss, hyperglycaemia.	Paroxysmal or labile hypertension; tachycardia or postural BP changes; features of associated genetic syndromes.	Measure plasma free metanephrines; if positive, proceed to cross-sectional imaging (CT or MRI) and refer to an appropriate specialist centre.
Other endocrine causes (e.g. Cushing's syndrome, thyroid or parathyroid disease)	Systemic features such as weight change, heat or cold intolerance, menstrual disturbance, mood or sleep changes, or features of hypercortisolism.	Characteristic body habitus or skin changes in Cushing's syndrome; goitre, tremor or bradycardia in thyroid disease; hypercalcaemia in primary hyperparathyroidism.	Targeted testing guided by clinical suspicion, e.g. TSH and T4 for thyroid disease, late-night or suppression tests for Cushing's syndrome, serum calcium and parathyroid hormone for suspected primary hyperparathyroidism, with specialist referral as needed.
Anticancer therapies (including VEGF pathway inhibitors, some TKIs, calcineurin inhibitors, platinum agents, endocrine therapies)	Often asymptomatic; patients may report headaches or other non-specific symptoms; usually identified on routine BP monitoring.	New-onset or worsening hypertension during or after initiation of cancer therapy; possible rapid rises in BP with some agents (e.g. VEGF pathway inhibitors).	Document baseline BP before starting therapies known to increase BP; arrange regular office or home BP monitoring during treatment, particularly in the first weeks; if marked or persistent elevation occurs, initiate or intensify antihypertensive therapy and liaise with the oncology team.
Medications, alcohol and other substances (e.g. NSAIDs, corticosteroids, oral contraceptives with oestradiol, erythropoietin,	Often asymptomatic; patients may not volunteer use	Temporal relationship between starting or escalating a medication/drug/substanc	Take a detailed medication and substance history (including over-the-

stimulants, recreational drugs, excess alcohol, caffeine)	unless specifically asked.	e and BP rise; resistant hypertension despite appropriate therapy.	counter agents and supplements); where safe and feasible, reduce dose or cease the suspected agent and reassess BP.
Coarctation of the aorta and other structural causes	May be asymptomatic; some patients report exertional leg fatigue, headaches or epistaxis.	Higher BP in upper than lower limbs, radio-femoral delay, weak or absent femoral pulses, murmurs suggestive of aortic pathology.	Measure BP in both arms and at least one leg, and palpate femoral pulses in younger adults with hypertension; if suspected, arrange echocardiography and/or CT or MR angiography and refer to cardiology.

Notes: adapted from Jones (2025), McEvoy (2024) and Rimoldi (2014).[2,3,117]

Abbreviations: ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BP, blood pressure; CT, computed tomography; eGFR, estimated glomerular filtration rate; GP, general practitioner; MR, magnetic resonance; MRA, mineralocorticoid receptor antagonist; MRI, magnetic resonance imaging; NSAIDs, non-steroidal anti-inflammatory drugs; OSA50, obstructive sleep apnoea 50 (screening questionnaire); T4, thyroxine; TKIs, tyrosine kinase inhibitors; TSH, thyroid-stimulating hormone; VEGF, vascular endothelial growth factor.

Once OSA is confirmed, CPAP produces a modest BP reduction (typically 2–3 mm Hg), greatest with good adherence and in patients with resistant or poorly controlled hypertension — reassess BP after CPAP initiation, but continue antihypertensive therapy, as CPAP alone rarely normalises BP.[2,92]

Medications, supplements and alcohol are often-overlooked secondary causes of hypertension and account for approximately 2–20% of diagnoses.[2,3] The mechanisms underlying elevated blood pressure vary, but most relate to alterations in RAAS pathways or sympathetic tone. Medications associated with hypertension are listed in Table 17.

Table 17: Medications associated with hypertension

Class of medication	Medications
Anti-VEGF signalling inhibitors	Bevacizumab Sorafenib Sunitinib
Immunosuppressive agents	Tacrolimus
Non-steroidal anti-inflammatory drugs and paracetamol	-
Steroids	Glucocorticoids, mineralocorticoids, liquorice
Haematological agents	Erythropoietin-stimulating agents
Sex hormones	Oestrogen
Antidepressants and psychotropics	Buspirone Fluoxetine

	Venlafaxine
Recreational drugs	Phencyclidine (PCP), methamphetamine, cocaine
Alcohol	-
Caffeine	-
Stimulants	Methylphenidate Dexmethylphenidate

Abbreviations: PCP, phencyclidine.

6.7.1 Primary aldosteronism

Primary aldosteronism (PA) is a common cause of secondary hypertension, with worse cardiovascular outcomes than primary hypertension at comparable blood pressures.[3,119-123] The ARR is the key screening test: low or suppressed renin with normal or elevated aldosterone concentration and a raised ARR are suggestive of PA.

<table>
<tr><th rowspan="2">Recommendation</th><th>Strength of recommendation
or source guideline class</th><th>Certainty of evidence
or source guideline level</th></tr>
<tr><td rowspan="2">In all adults with newly diagnosed hypertension, we suggest screening for primary aldosteronism.</td><td colspan="2">Adopted recommendation
Primary Aldosteronism: An Endocrine Society Clinical Practice Guideline 2025;[5] ESC 2024[6]</td></tr>
<tr><td>Conditional
Endocrine Society PA guideline 2025[5]
Class IIa
ESC 2024[6]</td><td>Low
Endocrine Society PA guideline 2025[5]
Level B
ESC 2024[6]</td></tr>
<tr><td>In adults with pre-existing hypertension and any of the following, we suggest screening for primary aldosteronism:
• BP above 140/90 mm Hg despite antihypertensive treatment, including resistant hypertension (BP above target on three or more agents at maximally tolerated doses including a diuretic, or controlled on four or more agents)
• spontaneous or diuretic-induced hypokalaemia</td><td colspan="2">Consensus</td></tr>
</table>

• atrial fibrillation unexplained by structural heart disease or thyroid disease • an adrenal adenoma • obstructive sleep apnoea • a family history of early-onset hypertension, a family history of stroke before age 40, or a first-degree relative with primary aldosteronism	

Elevated ARR is common in newly diagnosed Australian hypertension: 25% of 247 treatment-naive adults, with PA confirmed in 14% of those eligible, and 26% in an urban Indigenous cohort of young adults in Darwin.[119,124] PA carries largely preventable excess risk of stroke, coronary artery disease, AF and heart failure, yet remains profoundly under-detected in Australia.[121,125] Testing is associated with more targeted treatment and better longitudinal BP control.[126] These findings support routine PA testing in adults with hypertension, ideally before antihypertensive therapy and in high-risk groups (listed below).

In those with previously diagnosed or treated hypertension, high-risk features of PA include:

- Uncontrolled BP >140/90 mm Hg despite antihypertensive treatment
- Resistant hypertension (BP above target on ≥3 agents including a diuretic, or controlled on ≥4 agents)
- Hypertension with:
  - spontaneous or diuretic-induced hypokalaemia; or
  - atrial fibrillation unexplained by structural heart disease or thyroid disease; or an adrenal adenoma; or
  - obstructive sleep apnoea
- Individuals with a family history of early-onset hypertension or stroke before age 40, or a first-degree relative with PA.[5]

The steps associated with screening for PA are presented in Table 18. Further details of the management of PA are in Primary aldosteronism management.

Table 18: Primary aldosteronism screening steps

Step	Detail
1. Prepare the patient	Morning blood test: plasma/serum aldosterone + plasma renin. No sodium restriction in the days before testing. Check potassium at the same time — hypokalaemia can cause a false negative ARR.
2. Consider medications	Screen before starting antihypertensive pharmacotherapy where possible. Medication interference is not a reason to omit testing. In patients already on treatment, international guidance endorses minimal- or no-withdrawal strategies, with results interpreted in the context of current medications.[5] Where minimal withdrawal is used, stop mineralocorticoid receptor antagonists (MRAs) and epithelial sodium channel (ENaC) inhibitors 4 weeks before testing, and beta-blockers and centrally acting alpha$_2$-agonists 2 weeks before testing.[5] Balance accuracy against safety and practicality and consider switching to non-interfering medications if safe to do so.

3. Positive screen	Criterion 1 – Renin low/suppressed with aldosterone inappropriately high: DRC ≤8.2 mU/L (or PRA ≤1 ng/mL/h) and aldosterone ≥277 pmol/L (immunoassay) or ≥208 pmol/L (LC-MS/MS). Criterion 2 – Elevated ARR: aldosterone (pmol/L) / DRC (mU/L) >70 by immunoassay. Both criteria required in most circumstances.
4. Interpretation caveats	Suppressed renin with low aldosterone concentration may lead to an elevated ARR but is not consistent with PA.[5] Always use the local laboratory's cut-offs and units — these and the lowest reportable renin values vary by assay platform in Australia.[127] No ARR cut-off is definitive: interpret alongside clinical features, pre-test probability, medications and other confounders.[5]
5. When to repeat	Hypokalaemia – Negative screen with low potassium — correct potassium (aim >4.0 mmol/L) and repeat the ARR. Ongoing suspicion – Negative screen but moderate-to-high suspicion (e.g. resistant hypertension, unexplained hypokalaemia) — repeat on another day, after correcting hypokalaemia or adjusting interfering medications where safe.[5] CKD – Renin and aldosterone are harder to interpret — consider repeat testing if hypertension worsens or becomes resistant, or if new hypokalaemia or AF develops without another clear cause.[5]

Abbreviations: AF, atrial fibrillation; ARR, aldosterone–renin ratio; CKD, chronic kidney disease; DRC, direct renin concentration; ENaC, epithelial sodium channel; LC-MS/MS, liquid chromatography–tandem mass spectrometry; MRAs, mineralocorticoid receptor antagonists; PA, primary aldosteronism; PRA, plasma renin activity.

Next steps

Management of PA follows one of two pathways depending on the clinical situation and access to services.

Some individuals should be referred to an endocrinologist or specialist hypertension service. This includes potential candidates for unilateral adrenalectomy, who will typically need confirmatory testing (aldosterone suppression tests) and subtype assessment to distinguish unilateral from bilateral PA. Referral is also appropriate where specialist input is needed to interpret results in the context of interfering medications, chronic kidney disease (CKD) or other complex comorbidities.

In other cases, primary care–led management can be appropriate, and where specialist services are not accessible, it may also be the most practical pathway. See Primary aldosteronism management for further details.

Practice points

Remote and rural pathology logistics for PA

- Aldosterone/renin testing in rural and remote Australia is often processed at central laboratories after long-distance transport, which can delay processing and risk sample degradation or artefactual change (e.g. renin cryoactivation). Practical arrangements for sample collection and transport should therefore be established with local pathology providers.
- Some remote services may not routinely offer the ARR or may batch-send samples — agree practical pathways with the local laboratory in advance, including tube type, transport conditions, and validated cut-offs.

- In a Northern Territory cohort, remote participants had higher measured renin despite similar aldosterone, suggesting possible cryoactivation during chilled transport. This may lead to a false negative ARR due to non-suppressed renin.
- Confirmatory testing and subtyping (e.g. seated saline suppression test, fludrocortisone suppression test, adrenal vein sampling) are often unavailable locally and require travel to tertiary centres. Where saline suppression is impractical, a captopril challenge may be a more feasible option, and in high-suspicion cases empiric mineralocorticoid receptor antagonist therapy (e.g. spironolactone) can be used pragmatically to improve BP and outcomes while awaiting definitive work-up.

7 Definition and classification of hypertension

CVD risk rises progressively across the BP spectrum, from optimal through elevated BP to hypertension.[128,129] BP classification systems are pragmatic tools for clinical decision-making, with the diagnostic threshold grounded in data on the strength of the SBP/DBP-CVD risk association.[128,130-137] The BP categories are described in Table 19.

7.1 Blood pressure categories

Table 19: Classification of office blood pressure levels in adults

Blood pressure categories	
Optimal	<120 and <80 mm Hg
Elevated blood pressure	120–139 and/or 80–89 mm Hg
Hypertension	≥140 and/or ≥90 mm Hg

7.1.1 Optimal: <120 and <80 mm Hg

The lowest risk of CVD events and mortality occurs at SBP <115 mm Hg and DBP <75 mm Hg, with no J-curve effect in the general population.[128,129,138] Clinically, optimal BP is defined as <120 and <80 mm Hg.

7.1.2 Elevated BP: 120–139 and/or 80–89 mm Hg

CVD risk increases progressively from SBP levels as low as 115 mm Hg, reflecting a continuous relationship between BP and risk.[128,129,138 139-141]

Clinical trial and meta-analysis data indicate that BP reduction within this range lowers cardiovascular risk, particularly in higher-risk individuals.[128,129,138 139-141] Mean reductions of approximately 6 mm Hg are associated with reductions in coronary heart disease (14%) and stroke (18%), with substantial trial evidence in the 130–139 mm Hg systolic range.[141] Individuals treated to achieve a mean SBP closer to 120 mm Hg have lower CV risk than those treated to levels just below 130 mm Hg.[139-141]

Individuals with elevated BP should be informed that CV risk increases across this range, and overall CVD risk assessment should guide management and treatment decisions.

7.1.3 Hypertension: ≥140 and/or ≥90 mm Hg

Extensive RCT and meta-analysis evidence shows BP-lowering above this level reduces CV events and mortality.[130,142] The ≥140/90 threshold is retained for consistency with national statistics and as the level above which all adults should be considered for pharmacotherapy — but it is an arbitrary point on a continuum, not a threshold below which no one should be treated.

International definitions vary: the AHA/ACC and Hypertension Canada use a lower diagnostic threshold (>130/80 mm Hg), while the ESC, ESH and NICE retain 140/90 mm Hg — both approaches note the continuum of risk below their threshold.[2,3,67,92,143] This guideline's ≥140/90 definition balances population health benefit against the harms of over-labelling, while still recommending treatment from 130–139 and/or 80–89 mm Hg in adults with CVD or high CV risk (see Treatment thresholds for antihypertensive drug therapy).

7.1.4 Resistant hypertension

Resistant hypertension is defined as office BP above target despite concurrent use of three or more antihypertensive agents at optimal or maximally tolerated doses, from complementary classes (typically including a long-acting CCB, a RAS blocker [ACEi/ARB], and a diuretic), or BP controlled on four or more agents.[2,92] Before labelling hypertension as resistant, exclude pseudo-resistance: confirm technique and standardisation of BP measurement, exclude white-coat effect with out-of-office monitoring, and assess medication adherence.[92] Resistant hypertension carries a higher risk of secondary causes (particularly Primary aldosteronism) and adverse cardiovascular outcomes, and warrants: screening for secondary causes (see Secondary causes of hypertension); addition of a fourth-line agent, typically an MRA such as spironolactone (see Titration and dose optimisation); and specialist referral where BP remains uncontrolled despite optimised therapy.[2,92]

Key point — severe blood pressure

- **≥180/110 mm Hg —** a single reading is sufficient to establish the diagnosis if there is evidence of CVD, provided the reading is confirmed by repeat standardised measurement at that same visit and no reversible cause of acute BP elevation, such as pain, acute anxiety, or recent exertion or stimulant use is identified. ≥180/120 mm Hg **with acute target-organ damage —** hypertensive emergency. Refer immediately for emergency care; this is not an indication for same-day outpatient titration.
- **Sequence:** assess for hypertensive emergency first, before applying either of the treatment actions above.

7.2 Blood pressure categories for differing measurement modalities

Blood pressure values obtained using different measurement modalities are not directly interchangeable. The method used to measure BP significantly influences the reading obtained; applying a single universal threshold across all methods risks both over- and under-diagnosis of hypertension.

Table 20 provides a summary of these equivalent thresholds across standard office BP, AOBP, ABPM (24-hour, daytime, and night-time), and HBPM, based on current Australian guidelines and position statements.

Table 20: Hypertension equivalent thresholds across standard office BP, AOBP, ABPM (24-hour, daytime and night-time) and HBPM

	Office BP	AOBP	HBPM	ABPM Daytime	ABPM Night-time	24-hour ABPM
SBP	≥140	≥135	≥135	≥135	≥120	≥130
and/or						
DBP	≥90	≥85	≥85	≥85	≥70	≥80

Adapted from *Automated office blood pressure measurement: a Hypertension Australia and National Hypertension Taskforce of Australia position statement*.[7]

Practice points

Women

- Use the same diagnostic threshold for women and men. Hypertension prevalence in women overtakes that in men by around age 75, but no randomised trial has tested a sex-specific threshold and the BPLTTC 2023 individual-participant meta-analysis found no difference in the relative benefit of BP lowering between sexes — so while sex differences in BP trajectories remain an active research area, current evidence does not support a different threshold.[12,144,145]

7.3 Hypertension-related phenotypes

7.3.1 Isolated systolic hypertension

Isolated systolic hypertension (ISH) is defined as SBP ≥140 mm Hg with DBP <90 mm Hg.[146] It is uncommon in younger individuals but more prevalent in young men than women in Australia, and is the most common hypertension phenotype in older adults.[3,57,147] Its prevalence is expected to rise with population ageing, reflecting modifiable and hereditary influences alongside age-related vascular changes.[148] Accurate diagnosis requires appropriate BP measurement, including out-of-office monitoring to exclude white-coat hypertension, more frequent in younger individuals with ISH.[3]

7.3.2 Orthostatic (postural) hypotension

Orthostatic (postural) hypotension is a sustained fall in BP that occurs when changing from the supine or seated position to standing.[149] It is commonly defined as a reduction in SBP of at least 20 mm Hg or in DBP of at least 10 mm Hg within three minutes of standing, or of head-up tilt to at least 60° on a tilt table. Orthostatic hypotension is a clinical sign and may be either symptomatic (e.g. dizziness, light-headedness, blurred vision or falls) or asymptomatic. In patients with supine hypertension, a more appropriate criterion may be a larger fall in SBP, such as ≥30 mm Hg, because the magnitude of the BP change is greater with a higher baseline BP.[149]

Orthostatic hypotension should be assessed at the initial clinic visit by measuring BP seated and standing, and reassessed at subsequent visits if suggestive symptoms occur; assessment is particularly important in older adults (generally >70 years), those on medications associated with orthostatic hypotension (e.g. alpha-blockers), and people with diabetes or neurodegenerative disorders. For suspected orthostatic hypotension, standing BP should be measured after seated readings: after at least 5 minutes of rest seated or lying, measure BP one minute after standing and again at three minutes, with a trained operator remaining with the patient to ensure safety, particularly for those at increased fall risk.[3]

7.3.3 White-coat hypertension and masked hypertension

On average, out-of-office measures are about 5 mm Hg lower than those taken in office. In some people the difference is more marked, producing BP phenotypes that cannot be reliably identified from office BP alone, even with repeated office measurements over time:

White-coat effect: a transient elevation in BP occurring in a clinical setting, usually in the presence of healthcare professionals, which overestimates BP relative to usual levels.

White-coat hypertension: BP consistently elevated in the office (e.g. ≥140 and/or 90 mm Hg) and consistently below the hypertension threshold in out-of-office settings (e.g. <135/85 mm Hg at home or ambulatory daytime, or 24-hour BP <130 and/or 80 mm Hg). It affects about 13% of the general population and up to 32% of adults with elevated office BP, [150] and is associated with progression to sustained hypertension and a small increase in CV risk compared with normotensive individuals.[151]

Masked hypertension: office BP consistently below the diagnostic threshold (e.g. <140 and/or 90 mm Hg) with out-of-office BP consistently above it (e.g. ≥135 and/or 85 mm Hg at home or ambulatory daytime, or 24-hour BP ≥130 and/or 80 mm Hg). It affects an estimated 10–17% of adults[150] and carries CV risk comparable to or greater than sustained hypertension.[151] It is more common in people at higher baseline risk (diabetes, CKD, obstructive sleep apnoea, high absolute CVD risk, or target-organ damage such as left ventricular hypertrophy or albuminuria), and should be suspected when office BP is optimal despite unexplained end-organ damage, high estimated CVD risk, or repeated elevated readings in non-clinical settings.[152]

Both phenotypes are clinically important because misclassification can lead to inappropriate treatment or delayed diagnosis.[151] In treated individuals with elevated office BP, out-of-office monitoring can distinguish a white-coat effect from true uncontrolled hypertension. However, in people at high cardiovascular risk, treatment should not be delayed if monitoring is unlikely to alter management; repeat standard office BP assessment and clinical judgement remain paramount.[151]

Practice points

General

- Use out-of-office monitoring to identify white-coat effect/hypertension in adults with elevated but not severely elevated office BP, to avoid unnecessary pharmacological treatment.
- In adults on antihypertensive medication with elevated or apparently uncontrolled office BP, use out-of-office monitoring to distinguish white-coat effect from true uncontrolled hypertension and detect any transition to sustained hypertension.
- Do not delay treatment in adults at high CV risk if out-of-office monitoring is unlikely to change management — a persistently elevated standard office BP (e.g. ≥140/90 mm Hg) is unlikely to reflect optimal out-of-office levels, and a single out-of-office reading is not reliable on its own. Repeat standard office BP and clinical judgement remain paramount.[151]

7.4 Treatment targets

Recommendation	Strength of recommendation	Certainty of evidence
In adults receiving antihypertensive treatment, we recommend targeting an office blood pressure below 130 mm Hg systolic and below 80 mm Hg diastolic.	Strong	High

Supporting text

This target reflects RCT and meta-analysis evidence that <130/80 mm Hg reduces major adverse CV events compared with higher targets such as <140/90 mm Hg.[153-158] More intensive lowering, including systolic BP <120 mm Hg, can provide additional benefit in a broad range of patients, including reductions in MI, stroke, and left ventricular hypertrophy.[153-155,157-160]

Although more intensive treatment is associated with a higher rate of adverse events, the overall benefit–risk balance remains favourable.[153] Tolerability is influenced more by the choice and combination of agents than the absolute degree of BP reduction, and adverse effects can often be mitigated with appropriate therapy selection. Clinical judgement is essential, and treatment should be individualised based on patient risk, comorbidities, and preferences. If BP falls below 130/80 mm Hg and the patient remains asymptomatic and tolerating therapy well, this is generally acceptable and does not require treatment de-escalation.

In specific populations, including those with prior stroke or transient ischaemic attack and those with type 2 diabetes, a target of <130/80 mm Hg remains appropriate and is associated with reduced cardiovascular risk.[161-165] In patients with CKD, targets of <130/80 mm Hg are recommended, with some guidelines supporting systolic BP <120 mm Hg when tolerated.[2,166]

In older and frail adults, the same targets apply, although evidence is more limited. Treatment should be guided by tolerability, functional status and patient preference. A step-down approach in those with systolic BP <130 mm Hg has not been shown to reduce mortality.[167]

8 Prevention, management and follow-up of hypertension

8.1 Non-pharmacological interventions

Healthy lifestyle modifications are central to BP management and to preventing or delaying onset of hypertension. Beyond lowering BP, they reduce overall CV risk and are a beneficial treatment approach for everyone with elevated BP.[2,3,20,86,92] Healthy lifestyle interventions can also enhance pharmacological treatment effects and reduce the medication required to achieve BP control, but should not delay initiation of pharmacotherapy for people at higher CV risk.[168-170]

Key interventions include a heart-healthy eating pattern, reducing sodium and increasing potassium intake, achieving and maintaining a healthy weight, regular physical activity and structured exercise, limiting alcohol consumption, and smoking cessation. Table 21 provides a summary of these. Approaches should be tailored to the individual, considering preferences and cultural, social and economic context, to support engagement and sustained behaviour change.

Table 21: Non-pharmacological interventions summary

Intervention	Advice
Heart-healthy eating pattern	Heart Foundation heart-healthy eating pattern, which is the Australian adaptation of DASH and Mediterranean-style eating — maintained long term
Sodium reduction	<2,000 mg/day (about 5 g salt) from all sources; most dietary sodium comes from processed and packaged foods, so prioritise reducing these before addressing salt added at the table
Potassium increase	Increase potassium-rich foods (vegetables, fruit, legumes)
Potassium-enriched salt	Where salt is used, substitute potassium-enriched salt — this both lowers sodium and raises potassium intake
Fibre increase	Adequate intake 25–30 g/day; target 28–38 g/day
Physical activity	Combination of aerobic and resistance training
Alcohol reduction	Abstinence, or ≤1 drink/day for women and ≤2 for men, and to no more than 10 standard drinks per week overall, with several alcohol-free days each week.
Smoking cessation	Complete cessation, including e-cigarettes and vapes
Weight loss	Achieve and maintain healthy weight

Detail on each intervention, including full rationale, follows below.

8.1.1 General principles for healthy lifestyle interventions

Throughout, tailor advice to the person's preferences, culture and socioeconomic circumstances, and use shared decision-making. These principles apply to every intervention below and are not restated for each one.

Brief advice in a consultation is rarely sufficient on its own for the interventions that demand sustained behaviour change, particularly weight loss and dietary change. Where more intensive support is needed, refer early rather than after repeated unsuccessful attempts:

- accredited practising dietitian — for individualised dietary assessment and support, including translating a heart-healthy eating pattern into practical food choices, sodium reduction, and structured weight management.
- accredited exercise physiologist — for prescription and progression of combined aerobic and resistance training, particularly where comorbidity, deconditioning or musculoskeletal limitation complicates activity advice
- pharmacist — for medication review, adherence support, and home BP monitoring technique

Subsidised access pathways can reduce cost as a barrier. A GP Chronic Condition Management Plan supports referral for Medicare-rebated allied health services, and Group Allied Health Services are available for eligible people with type 2 diabetes. Confirm current eligibility and item numbers before referral, as arrangements change.

Structural and access barriers are often greater in regional and remote areas and for First Nations people, including limited local allied health availability, travel cost and cultural safety. Practice points flag where a specific adaptation is needed beyond this general principle.

8.1.2 Dietary patterns and nutrients

Heart-healthy eating pattern

Recommendation	Strength of recommendation	Certainty of evidence
We recommend the Heart Foundation's heart-healthy eating pattern, adapted to individual cultural food preferences, for all adults to prevent and manage elevated blood pressure, maintained long term and alongside antihypertensive medication where indicated.	Strong	Moderate

Supporting text

Heart-healthy eating patterns, such as the Heart Foundation heart-healthy eating pattern, DASH and Mediterranean-style eating, emphasise overall food patterns over isolated nutrients.

They are naturally low in saturated and trans fats, sodium and added sugar while rich in wholegrains, fibre, potassium and unsaturated fats (monounsaturated, omega-3 and omega-6 polyunsaturated fatty acids), improving BP, lipids, and CVD event and mortality risk.[171-173] Of the dietary patterns studied, DASH and Mediterranean-style eating have the strongest evidence for BP lowering: meta-analyses show DASH reducing SBP/DBP by 3.2/2.5 mm Hg and Mediterranean patterns by 1.5/0.9 mm Hg versus control, with DASH's effect enhanced by weight loss, sodium reduction and physical activity, and greater adherence to either pattern linked to lower all-cause and cardiovascular mortality.[169,173-185] Other patterns (vegetarian, low-carbohydrate, low-fat, low-glycaemic index) also lower BP but appear less effective.[175-177,184]

Practice points

- Focus on the overall pattern, not individual nutrients or foods in isolation.
- For First Nations people, support inclusion of traditional foods where aligned with preferences.[186,187]
- For older adults receiving meals from external providers, it is important to identify when special diets are required and to liaise with providers so meals align with the individual's needs.

Sodium

<table>
<tr><th>Recommendation</th><th>Strength of recommendation</th><th>Certainty of evidence</th></tr>
<tr><td rowspan="2">We recommend that adults limit sodium intake to less than 2,000 mg per day (equivalent to less than 5 g of salt, or about one teaspoon) from all sources, including salt added during cooking and at the table and salt already present in processed, packaged and takeaway foods.</td><td colspan="2">Adopted recommendation
ESC 2024[6]</td></tr>
<tr><td>Class I
ESC 2024[6]</td><td>Level A
ESC 2024[6]</td></tr>
</table>

Supporting text

Reducing dietary sodium intake lowers BP in a dose-response manner, with greater effects observed in people with hypertension or higher baseline BP.[188-191] Sodium reduction is effective regardless of whether a person is taking antihypertensive medication and should be offered to everyone with elevated BP. A meta-analysis of 35 RCTs (2,885 adults on BP-lowering medication) reported that each 100 mmol/day reduction in 24-hour urinary sodium reduced SBP by 6.8 mm Hg (95% CI 5.0–8.7) and DBP by 3.9 mm Hg (95% CI 2.3–5.4); a within-study comparison of factorial trials found no significant difference in this dose–response effect between participants on and not on medication, and the magnitude matches that seen in untreated hypertension.[192] In addition, sodium reduction has additive BP-lowering effects when combined with other dietary interventions, including the DASH diet.[173,193,194] Higher sodium intake has also been associated with increased cardiovascular risk.[195,196]

Most people in Australia exceed the Suggested Dietary Target of 2,000 mg/day, with most sodium coming from packaged and processed foods rather than from discretionary salt (added during cooking, food preparation or at the table), which contributes to a lesser extent.[197,198] These patterns suggest that meaningful population-level reductions in BP and CVD events will require both individual behaviour change and wider structural actions, including food reformulation strategies and policies to reduce sodium across the food supply.[199-203]

Practice points

- Use caution with very low sodium targets (below approximately 1,500 mg/day) in diabetes, heart failure or orthostatic hypotension: observational data show a paradoxical mortality association with very low intake in these groups.[185,204-209] Follow higher limits (<2,300 mg/day) rather than routine restriction below 1,500–2,000 mg/day.[206]
- Advise gradual reduction (taste adapts over time) via fresh, minimally processed foods; herbs/spices/citrus instead of salt; and checking labels for 'low salt' or 'no added salt'.[199]
- Apps that scan barcodes and interpret nutrition labels can help identify lower-sodium products.[210,211]
- Recommend switching regular salt for potassium-enriched salt in those adding salt in the home while cooking or seasoning. See Potassium below.

Potassium

Recommendation	Strength of recommendation or source guideline class	Certainty of evidence or source guideline level
In adults with elevated blood pressure or hypertension, we recommend increasing intake of potassium-rich foods, including vegetables, fruit, legumes, nuts and unsalted dairy.	Adopted recommendation AHA/ACC 2025[2]	
	Class I AHA/ACC 2025[2]	Level A AHA/ACC 2025[2]
In adults with elevated blood pressure or hypertension who add salt during cooking or at the table, we suggest replacing regular salt with a potassium-enriched salt substitute.	Conditional	Moderate
Advice to increase dietary potassium intake, including through potassium-rich foods or	Consensus[†]	

potassium-enriched salt substitutes, does not apply to adults at increased risk of hyperkalaemia. In these people, increase dietary potassium or introduce a potassium-enriched salt substitute only under clinical supervision and monitoring.	

†This recommendation is adapted from *Potassium-enriched salt for patients with hypertension: a Hypertension Australia and National Hypertension Taskforce of Australia position statement*,[8] and is informed by the opinion of the Expert Steering Group and Expert Working Group members, with consideration of the evidence, values, preferences and resource use at the time of writing.

Supporting text

Meta-analyses of RCTs report that increasing potassium intake reduces SBP and DBP, with larger effects in people with hypertension and higher baseline sodium intake.[212-214] Higher potassium intake is also linked to lower stroke and CVD event risk, and a lower urinary sodium-to-potassium ratio is associated with greater BP reduction, lower mortality and reduced CV risk.[195,196,212-218] WHO recommends at least 90 mmol/day (approximately 3,500 mg/day) from food, a level most adults currently fall short of.[212,219] A dose–response meta-analysis found that most of the BP benefit accrues from modest increases toward this level, with diminishing additional benefit beyond it — so the practical priority is closing the gap to 90 mmol/day rather than pursuing higher intakes.

Potassium-enriched salt (where a proportion of sodium chloride is replaced with potassium chloride) reduces SBP by 4.6–5.2 mm Hg and DBP by 1.5–1.6 mm Hg versus regular salt, and is associated with reductions in all-cause mortality (11–12%) and cardiovascular mortality (13–17%).[8,220,221] BP effects have been consistent across diverse populations, though the clinical outcome trials were conducted in China and Taiwan in older, higher-risk adults, so hard outcome data remain largely from those settings.[220]

These recommendations do not apply to adults at increased risk of hyperkalaemia, including those with advanced chronic kidney disease or taking potassium-sparing diuretics, ACE inhibitors, ARBs or mineralocorticoid receptor antagonists. In these people, increase dietary potassium or introduce a potassium-enriched salt substitute only under clinical supervision and monitoring.

Practice points

- Encourage potassium-rich foods (chickpeas, beans, nuts, spinach, tomatoes, potatoes, bananas, apples, dried fruit) unless contraindicated.[8,219]
- For those who add salt during cooking or at the table, recommend a 1:1 switch to potassium-enriched salt (approximately 75% sodium chloride, 25% potassium chloride) in place of regular salt.

- Before recommending it, check kidney function and assess hyperkalaemia risk. Avoid or use only with monitoring in advanced CKD, in people taking potassium-sparing diuretics or mineralocorticoid receptor antagonists, and in those on potassium supplements. ACE inhibitor or ARB use alone is not a contraindication, but combined with reduced kidney function it warrants monitoring of serum potassium.[8,222]
- Choose potassium-enriched salt substitutes that are iodised.[222-224]

Fibre

Higher dietary fibre intake is associated with reduced all-cause mortality (RR 0.85) and coronary heart disease incidence (RR 0.76) and mortality (RR 0.69) in observational data, with RCTs confirming a modest BP-lowering effect that scales with intake — in adults with hypertension specifically, each 5 g/day increase is associated with reductions of approximately 2–3 mm Hg SBP and 2 mm Hg DBP.[225,226]

In Australia, the recommended adequate intake is 25 g/day for women and 30 g/day for men, with a higher Suggested Dietary Target of 28 and 38 g/day respectively — yet only 28.2% and 17% of adults meet these, underscoring the potential for population-level benefit.[227]

Practice points

- Encourage a gradual increase rather than prescribing specific foods: at least five serves of vegetables (including legumes) and two of fruit daily; swap refined grains for wholegrain; choose wholegrain snacks and higher-fibre packaged products.

8.1.3 Physical activity

<table>
<tr><th>Recommendation</th><th>Strength of recommendation
or source guideline class</th><th>Certainty of evidence
or source guideline level</th></tr>
<tr><td>In adults, we recommend regular physical activity accumulated throughout the day, combined with reducing sedentary behaviour, to prevent and manage elevated blood pressure. Activity should be maintained long term, alongside antihypertensive medication where indicated.</td><td colspan="2">Consensus‡</td></tr>
<tr><td rowspan="2">In adults with elevated BP or hypertension, we recommend a combination of moderate-to-vigorous aerobic activity and resistance training to reduce blood pressure and cardiovascular risk,</td><td colspan="2">Adopted recommendation
AHA/ACC 2025[2]</td></tr>
<tr><td>Class I</td><td>Level A</td></tr>
</table>

with the exercise program tailored to the individual's goals and condition.	AHA/ACC 2025[2]	AHA/ACC 2025[2]

‡This consensus recommendation is adopted from the *National Heart Foundation of Australia, Obesity and cardiovascular disease: a clinical consensus statement, 2026 (graded Strong advice)*, and is informed by the opinion of the Expert Steering Group and Expert Working Group members, with consideration of the evidence, values, preferences and resource use at the time of writing.

Supporting text

Regular physical activity plays a key role in preventing and managing hypertension. Meta-analyses consistently show BP-lowering effects across exercise modalities — aerobic (e.g. walking, jogging, cycling, swimming), dynamic resistance (e.g. weightlifting), and isometric (e.g. handgrip) — with larger reductions in people with hypertension than in the general population.[175,228-230] In people with hypertension, aerobic exercise lowers SBP/DBP by up to 7–8/4–5 mm Hg, dynamic resistance training by up to 6/5 mm Hg, and isometric exercise by up to 10/5 mm Hg, with combined aerobic and resistance training also producing meaningful reductions.[228,230,231] Aerobic exercise has been shown to lower BP in a dose-dependent manner, with each additional 30 minutes per week reducing SBP by 1.8 mm Hg (95% CI −2.2 to −1.3) and DBP by 1.2 mm Hg (95% CI −1.5 to −0.9), and the greatest reduction reached at 150 minutes per week (SBP −7.2 mm Hg, 95% CI −9.1 to −5.4; DBP −5.6 mm Hg, 95% CI −6.9 to −4.3), with only trivial additional benefit beyond this.[229,232,233] High-intensity interval training achieves reductions comparable to moderate-intensity continuous exercise. Combining exercise with pharmacotherapy may give greater BP reductions than medication alone.[168]

The health benefits of physical activity are not limited to structured exercise programs. Lower-intensity physical activity such as walking, when interrupting prolonged sedentary time, can also meaningfully reduce BP.[234-236] Being physically active is also associated with decreased risks of CV mortality and incidence and all-cause mortality.[237-242]

Practice points

- Assess current physical activity levels and/or fitness, and sedentary time.
- Any type, amount and intensity of activity is better than none — set small achievable goals, and build volume before intensity.[242,243]
- Encourage activity accumulated across the day (e.g. 'exercise snacking').[242-245]
- Where appropriate and achievable for the individual, work towards approximately 150 minutes per week of moderate-intensity activity.
- Wearables, apps and other digital tools may assist with self-monitoring and motivation, although specific products are not endorsed due to the rapidly evolving evidence base and technology landscape.[246-248]
- Refer to an accredited exercise physiologist or physiotherapist for assessment of aerobic/functional capacity (fitness) and safe prescription where comorbidities or physical limitations need tailored guidance.

- Physical activity advice for First Nations people should be individualised, considering cultural context, preferred language, and capacity to engage.[249,250] Refer to relevant local programs and resources where appropriate.

8.1.4 Alcohol

Recommendation	Strength of recommendation or source guideline class	Certainty of evidence or source guideline level
In adults with elevated blood pressure or hypertension who currently consume alcohol, we recommend advising abstinence as the preferred goal. Where abstinence is not achieved, we recommend limiting intake to no more than 1 standard drink per day for women and 2 standard drinks per day for men, and to no more than 10 standard drinks per week overall, with several alcohol-free days each week.*	Adopted recommendation AHA/ACC 2025[2]	
	Class I AHA/ACC 2025[2]	Level A AHA/ACC 2025[2]

*Drinking the daily maximum of 2 standard drinks every day of the week adds up to 14 standard drinks, which exceeds the weekly limit of 10 standard drinks. Anyone drinking 2 standard drinks a day must therefore include alcohol-free days to stay within the recommended weekly limit.

Supporting text

Alcohol raises BP and hypertension risk in a dose-dependent way: the relative risk of developing hypertension falls to 0.89 with abstinence and rises to 1.11, 1.22 and 1.33 at 24, 36 and 48 g/day respectively compared with a reference intake of 12 g/day (one Australian standard drink is 10g).[251,252] Reducing intake follows the same gradient — a meta-analysis of 36 RCTs found no significant BP benefit from reducing intake at ≤2 drinks/day, a modest benefit at 3–5 drinks/day, and the largest reductions (SBP −5.5, DBP −4.0 mm Hg) in those drinking >6 drinks/day who cut intake by about half, with similar effects in people with hypertension.[253] The absence of measurable BP benefit from reducing intake at low levels does not indicate that low-level drinking is without risk; observational data show hypertension risk rising from around one standard drink per day.[251,252]

Current Australian guidance sets a general population limit of ≤10 standard drinks/week and ≤4 on any one day, but emerging evidence of CV risk at even low intake is driving international guidance toward lower thresholds.[2,254-259] The guidance in this guideline supports brief, structured counselling (standard drinks, alcohol-free days). It should not be used to guide specialised addiction treatment.

Practice points

- Use validated screening tools (e.g. AUDIT-C or IRIS) to assess current intake, then explain that there is no completely safe level of alcohol consumption and that drinking less lowers the risk of cardiovascular disease, alcohol-related harm, and other health problems, with clear information about standard drinks and practical strategies such as regular alcohol-free days to help reduce overall intake.[254,257]
- For First Nations people, deliver advice respectfully and non-judgementally, and link to culturally tailored programs where available.

8.1.5 Smoking cessation

<table>
<tr><th>Recommendation</th><th>Strength of recommendation</th><th>Certainty of evidence</th></tr>
<tr><td rowspan="2">In adults with elevated blood pressure or hypertension who smoke, we recommend that smoking cessation be encouraged and supported at every opportunity, including referral to behavioural interventions (such as cognitive behavioural therapy or cessation counselling) and TGA-approved pharmacotherapy where clinically indicated.</td><td colspan="2">Adopted recommendation
ESC 2024[6]</td></tr>
<tr><td>Class I

ESC 2024[6]</td><td>Level A

ESC 2024[6]</td></tr>
</table>

Supporting text

Smoking is a major modifiable risk factor for elevated BP and CVD, and cessation is one of the most effective measures for reducing CV risk.[260,261] It raises ambulatory BP (more than office BP), increases daytime BP variability via sympathetic activation, and is linked to masked hypertension; even passive exposure elevates 24-hour BP and CV risk.[260-265] E-cigarettes carry similar risks — acute rises in BP, heart rate and arterial stiffness — with long-term CV data still limited.[266]

Combining behavioural support with pharmacotherapy gives the best chance of quitting, though brief advice alone still helps when time is limited; more intensive or prolonged support further improves success.[267]

Practice points

- Offer all adults who smoke brief, structured support (Ask, Advise, Help or RACGP 5As), regardless of age.
- Offer multi-session behavioural support (e.g. Quitline) and TGA-approved pharmacotherapy; encourage e-cigarette users to quit too.[268]
- For people with a mental health condition, see the RACGP smoking cessation guidelines for additional options.[269]

Further guidance on smoking cessation can be found at Quit.

8.1.6 Weight loss

Recommendation	Strength of recommendation or source guideline class	Certainty of evidence or source guideline level
In adults with elevated blood pressure or hypertension who are living with overweight or obesity, we recommend weight reduction, alongside antihypertensive medication where indicated.	Adopted recommendation AHA/ACC 2025;[2] ESC 2024[6]	
	Class I AHA/ACC 2025;[2] ESC 2024[6]	Level A AHA/ACC 2025;[2] ESC 2024[6]

Supporting text

Approximately two-thirds of adults in Australia are currently living with overweight or obesity.[270] Excess adiposity contributes to the development of several well-established risk factors for CVD, including hypertension, dyslipidaemia and type 2 diabetes, and is rarely an isolated finding, with higher BMI categories typically showing clustered cardiometabolic abnormalities.[271] Therefore, assessment and management of overweight and obesity should be considered a routine component of hypertension care.[271] International guidelines consistently recommend weight reduction for management of elevated BP, hypertension and CVD risk;[272,273] even modest weight loss can lead to clinically meaningful BP reductions, with an average 5 kg loss associated with mean reductions of about 4.4 mm Hg in SBP and 3.6 mm Hg in DBP, and further enhancement of the BP-lowering effects of dietary interventions such as the DASH diet.[181,272-274]

Effective management requires a multimodal, staged approach, with sustained behaviour change, addressing nutrition, physical activity, smoking, alcohol use, sleep and psychological wellbeing, in a non-stigmatising, culturally appropriate manner, as a core component that should be maintained across the life course, even once weight goals are achieved.[259,275,276] However, weight regain after behaviour modification alone is common, and repeated cycles of weight loss and regain may adversely affect BP and cardiovascular risk, highlighting the need for additional evidence-based treatment options in some individuals.[277-279]

Pharmacological and surgical interventions play a critical role alongside, not instead of, behaviour modification, particularly for those at high CV risk or with established CVD, or where behavioural interventions alone are insufficient.[280-284] High-quality trials show incretin-based medicines (GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonists) produce clinically significant weight loss and improve cardiometabolic risk factors including BP, with average SBP reductions of around 3 mm Hg for GLP-1 agonists and up to 5–7 mm Hg with newer dual and triple agonists, proportional to weight loss.[282,283,285-288] As weight regain is common after

treatment cessation, long-term treatment may be required.[289] People who do not achieve weight-related health improvements, or cannot sustain weight loss after discontinuation, may require referral to specialised care, including metabolic bariatric surgery, though access and cost remain important barriers to both.[290]

Further reading

Refer to the Heart Foundation's *Obesity and cardiovascular disease: a clinical consensus statement* for evidence-based recommendations, practice points and resources on the assessment, diagnosis and management of adults (people aged 18 years or older) living with overweight or obesity with established CVD or at high risk of CVD, encompassing behaviour modifications, obesity management medications, metabolic bariatric surgery, and lifelong care and follow-up.

8.2 Pharmacotherapy for blood pressure

In adults, clinicians should initiate healthy lifestyle modification and antihypertensive pharmacotherapy concurrently, with adequate follow-up to assess response and adherence. Healthy lifestyle change alone rarely achieves target BP once it's elevated to this level.

8.2.1 Treatment thresholds for antihypertensive drug therapy

Recommendation	Strength of recommendation	Certainty of evidence
In adults with low cardiovascular risk (<5% risk over 5 years), we recommend initiating antihypertensive pharmacotherapy alongside healthy lifestyle modification when office blood pressure is ≥140 mm Hg systolic and/or ≥90 mm Hg diastolic.	Strong	High
In adults with intermediate cardiovascular risk (5 to <10% risk over 5 years), we recommend considering antihypertensive pharmacotherapy when office blood pressure is ≥130 mm Hg systolic and/or ≥80 mm Hg diastolic, and initiating pharmacotherapy alongside healthy lifestyle modification in all when office blood pressure is ≥140 mm Hg systolic and/or ≥90 mm Hg diastolic.	Strong	High
In adults with either established cardiovascular disease or high cardiovascular risk (≥10% risk over 5 years), we recommend initiating antihypertensive pharmacotherapy alongside healthy lifestyle	Strong	High

modification when office blood pressure is ≥130 mm Hg systolic and/or ≥80 mm Hg diastolic.		
In adults with office blood pressure ≥180 mm Hg systolic and/or ≥110 mm Hg diastolic, we recommend initiating antihypertensive pharmacotherapy without delay, alongside healthy lifestyle modification.	Consensus	

Supporting text

The threshold at which antihypertensive pharmacotherapy is started depends on the individual's CV risk, not on BP alone. This guideline uses the three risk categories defined by the 2023 *Australian Guideline for assessing and managing cardiovascular disease risk*: low, intermediate and high.[1] Table 22 shows overarching guidance. Across all risk categories, pharmacotherapy is an adjunct to healthy lifestyle measures; healthy lifestyle modification should be initiated and maintained regardless of whether medication is prescribed.

Table 22: Recommended action by risk category and blood pressure band

		Optimal	Elevated		Hypertension
	SBP/DBP	<120 and <80	120–129 and/or 80–89	130–139 and/or 80–89	≥140 and/or ≥90
	Healthy lifestyle advice for everyone – at every BP, at every risk level				
5-year CVD risk	**High** **≥10% or CVD/CKD**	**Monitor** Routine screen	**Consider** Pharmacotherapy	**Initiate** Pharmacotherapy	**Initiate** Pharmacotherapy
5-year CVD risk	**Intermediate** **5 to <10%**	**Monitor** Routine screen	**Healthy lifestyle** Active measures	**Consider** Pharmacotherapy	**Initiate** Pharmacotherapy
5-year CVD risk	**Low** **<5%**	**Monitor** Routine screen	**Healthy lifestyle** Active measures	**Healthy lifestyle** Active measures	**Initiate** Pharmacotherapy

≥180 mm Hg: initiate pharmacotherapy without needing out-of-office confirmation if there is evidence of CVD, provided the reading is confirmed by repeat standardised measurement at that same visit and no reversible cause of acute BP elevation, such as pain, acute anxiety, or recent exertion or stimulant use is identified. Thresholds shown are conventional office BP; AOBP-equivalent hypertension threshold is ≥135/85 mm Hg.

Where risk is genuinely unknown and cannot be estimated, apply the intermediate row rather than the low-risk row as a precautionary practice point, since under-classifying as low-risk risks foregoing treatment consideration and delaying reassessment, whereas the intermediate row prompts closer follow-up without mandating treatment.

Adults at low CV risk

For adults at low calculated risk (5-year risk <5%), start pharmacotherapy when BP is persistently ≥140 and/or 90 mm Hg, as lowering BP in this range reduces mortality, stroke and coronary events.[130,132,142,145,291,292] Below this level, manage with healthy lifestyle modification and scheduled review.

Adults at intermediate CV risk

The 2023 Aus CVD risk algorithm has been compared with the previous 2012 absolute risk equation. It results in a smaller proportion of people classified in the high-risk category, with proportionately more in the intermediate-risk band.[293]

For these intermediate-risk patients, prioritise treatment action including pharmacotherapy, particularly if both BP and CV risk are at the higher end of the intermediate range. Younger adults warrant particular attention. A 5-year risk estimate is designed to quantify short-term risk and is not intended as a measure of lifetime risk, so in younger people it may not convey the cumulative burden associated with an elevated BP maintained over many years. A person aged 40 or younger with a BP of 130/80 mm Hg or higher may therefore accumulate decades of exposure unless BP is lowered and sustained at a lower level through healthy lifestyle modification, pharmacotherapy, or both.

Adults at high CV risk

For adults with persistent office BP ≥130 and/or 80 mm Hg and established CVD or high CV risk, pharmacotherapy is warranted as the greater absolute benefit in preventing future events outweighs the higher rate of treatment-related adverse events.[294-296] Meta-analyses indicate that each 5 mm Hg SBP reduction yields an approximately 10% relative reduction in major CV events across BP strata, with greater benefit in higher-risk groups.[292] Evidence also suggests that small SBP reductions in individuals with high-optimal BP and established CVD are beneficial, mainly for those at very high risk.[141,145,297-299]

Very high BP

In adults with office blood pressure ≥180 mm Hg systolic and/or ≥110 mm Hg diastolic, before initiating antihypertensive pharmacotherapy, assess for a hypertensive emergency — severely elevated blood pressure accompanied by signs or symptoms of acute target-organ damage. Where a hypertensive emergency is suspected, arrange immediate transfer for emergency assessment and management rather than commencing oral therapy in the community setting (see *Recognising a hypertensive emergency*). In the absence of acute target-organ damage, initiate antihypertensive pharmacotherapy without delay, alongside healthy lifestyle modification, and arrange early review to confirm response and tolerability.

Older adults and frailty

Individualise decisions for older adults, weighing the harms of BP lowering (hypotension, syncope, electrolyte disturbance, AKI, intolerance) against benefit, particularly where baseline BP is low or frailty is present.

Hypertension prevalence rises with age, peaking in adults ≥75.[12] Despite higher adverse-effect rates in this group, net-benefit analyses in those 65–74 and ≥75 show the overall balance still favours treatment, even in frail community-dwelling older adults — because absolute risk reduction stays high (their baseline CVD risk is higher) even as proportional benefit lessens with age.[300,301]

Stroke, cognition and dementia

In the acute phase (first 48–72 hours), BP management should follow stroke-specific protocols, which differ for ischaemic stroke (including patients eligible for thrombolysis or thrombectomy) and intracerebral haemorrhage, and are not detailed in this guideline.

The benefit of BP lowering is particularly strong for stroke prevention.[302-306] Meta-analyses in people with prior stroke or TIA show BP-lowering reduces recurrent stroke by approximately 20%, even from mildly elevated baseline BP; each additional 5 mm Hg SBP reduction cuts recurrent stroke risk by approx. 10%.[304-306] The reduction in stroke risk among people with prior haemorrhagic stroke is especially large, because the absolute risk is high and the risk reduction per mm Hg is about twice as great. Overall, in past trials, intensive BP lowering reduced the risk of stroke by around 40% in people with haemorrhagic stroke.[307,308] Proportional effects are similar across the lifespan, but absolute benefit is greater in older, higher-risk groups — supporting treatment decisions based on overall CV risk rather than BP level alone.[304-306]

Hypertension in mid- and later life also raises the risk of cognitive decline and dementia, including Alzheimer's disease, with untreated hypertension in adults aged ≥60 years carrying a 36–42% higher Alzheimer's risk than normotensive or treated individuals.[309] Treatment reduces this risk: a 10/4 mm Hg reduction is associated with approx. 13% lower odds of incident dementia, and BP-lowering after stroke or TIA may also modestly reduce dementia risk.[302,303] BP management across the life course is therefore a strategy for both stroke and cognitive prevention.

8.2.2 Pharmacotherapy

Recommendation	Strength of recommendation	Certainty of evidence
In adults with confirmed hypertension (office blood pressure ≥140 mm Hg systolic and/or ≥90 mm Hg diastolic), we recommend initiating treatment with a lowest available formulation single-pill combination of two first-line antihypertensive agents from different classes (ACE inhibitor or angiotensin receptor blocker, calcium channel blocker, or thiazide/thiazide-like diuretic).	Strong	High

In adults indicated for pharmacotherapy at systolic blood pressure 130–139 mm Hg or diastolic blood pressure 80–89 mm Hg, we recommend initiating treatment with a lowest available formulation dual single-pill combination of two first-line agents from different classes. We suggest that a single first-line agent (monotherapy) may be considered for selected patients where clinically appropriate.	Strong	High
In adults requiring pharmacotherapy who have a compelling indication for beta-blocker therapy (e.g. heart failure with reduced ejection fraction, or the need for heart rate control), we recommend a beta-blocker in combination with one or more of the major first-line blood pressure–lowering classes.	Strong	High
In adults with hypertension, we recommend against dual renin–angiotensin system blockade with an ACE inhibitor plus an angiotensin receptor blocker. Dual blockade does not reduce all-cause or cardiovascular mortality compared with monotherapy, and increases the risk of hyperkalaemia, hypotension, renal failure and treatment discontinuation.	Strong (against)	High

Supporting text

All major antihypertensive classes — thiazide and thiazide-like diuretics, ACE inhibitors, ARBs, CCBs, centrally acting anti-adrenergics, aldosterone antagonists and beta-blockers — effectively lower BP and reduce CVD risk, with only modest differences between classes by outcome or setting.[130,135,310-318] For most patients, the magnitude of risk reduction is determined more by the degree of BP lowering than by class-specific effects.

First-line therapy

Initial combination therapy is now preferred: two agents together lower BP faster and further than monotherapy (often approximately double the reduction), reach target more often, and don't raise serious adverse events.[301,312,319-324] A RAS blocker (ACEi or ARB) with a CCB or diuretic has the best support for both BP reduction and CV event prevention;[322] ARB-based regimens are best tolerated, with fewer withdrawals than monotherapy or placebo.[325]

Reserve initial monotherapy for selected patients such as the frail.[3] Document the rationale if choosing monotherapy at SBP 130–139 or DBP 80–89 mm Hg (e.g. frailty/falls risk, or steno-occlusive cerebrovascular disease where rapid BP reduction risks cerebral hypoperfusion), with close follow-up to add agents if target isn't reached.[2,115] Patients already on monotherapy

who are not controlled to a mean BP <130/80 mm Hg should generally be switched to combination therapy. Where a comorbid condition provides a compelling indication for a particular agent, this may influence drug selection but not the requirement to reach target BP. See Individualising drug choice for comorbidities.

Single-pill combinations

Combinations should be prescribed as single-pill combinations (SPCs/fixed-dose combinations) in preference to free equivalent combinations (FECs) when suitable products, doses, and PBS listing are available. Meta-analyses and systematic reviews show SPCs improve adherence, persistence, BP control, and cardiovascular outcomes, and are cost-effective, with more patients reaching target and lower medical costs after switching from FECs and no increase in withdrawals.[28,322,325-328]

SPCs should be used proactively to overcome therapeutic inertia — stepping up directly from inadequate monotherapy to dual or triple combination therapy rather than titrating single agents in sequence — minimising pill burden while preserving flexibility for dose adjustment and component changes. Monitor BP and safety as usual, and switch to an alternative SPC or FEC if tolerability, supply, or dose-flexibility issues arise. In 2026, PBS restrictions were relaxed to allow dual-therapy SPCs as first-line treatment, with 30-day items listed as Unrestricted Benefit and 60-day items as Restricted Benefit for stable patients.

Adverse events don't generally require reverting to separate pills: for a component-specific effect, switch to an alternative SPC (e.g. a patient with cough on an ACE inhibitor–CCB combination can switch to an ARB–CCB combination). If the effect relates to the degree of BP lowering (such as dizziness or symptoms of hypotension), treatment can be paused or stepped down to a regimen with fewer classes (triple to dual SPC, dual SPC to monotherapy). This also helps in discussing effects unlikely to be causal, important given the tendency to over-attribute causality, as seen with statins.[329-331]

Escalation of therapy

A significant problem in hypertension management in Australian practice is the lack of escalation of therapy (treatment inertia), as outlined further below in Treatment inertia. If BP targets are not reached, a third or fourth agent should be added (or second agent if on monotherapy), ideally as an SPC; lowest available formulation triple or quadruple SPCs produce greater, more sustained BP reduction and higher rates of target achievement than initial monotherapy or usual care and are associated with better adherence.[322,323,332] Mineralocorticoid receptor antagonists (MRAs) are particularly useful as fourth-line therapy. Dual RAS blockade (e.g. an ACE inhibitor with an ARB) should be avoided as it provides no additional cardiovascular or renal benefit and substantially increases the risk of renal dysfunction, acute kidney injury, hyperkalaemia, and hypotension.[333-335]

Individualising drug choice for comorbidities

Drug selection should reflect each person's comorbidities and tolerability profile. Beta-blockers should be combined with another major class when there is a compelling indication for their use — angina, post-myocardial infarction, heart failure with reduced ejection fraction, or heart-rate control.[130,336,337] Similarly, symptomatic benign prostatic hyperplasia is a compelling indication for a non-uroselective α1-blocker such as doxazosin, which relieves lower urinary tract symptoms [338]while also lowering BP. SGLT2 inhibitors produce a modest additional BP reduction [339]of approximately 3–4/2 mm Hg on ambulatory monitoring, and are increasingly used for their compelling indications (type 2 diabetes, CKD, heart failure). Where already indicated for these conditions this BP benefit is a useful adjunct, but SGLT2 inhibitors are not indicated as antihypertensive agents and are [3,340]not a substitute for standard first-line therapy. Detailed management in diabetes and CKD is out of scope for this guideline.

Why is monotherapy no longer recommended for most patients?

Initial monotherapy has been the mainstay of Australian practice for decades.
Three lines of evidence support the change.

1	**Monotherapy is not better tolerated. A recent comprehensive review** found several dual combinations have better tolerability profiles than monotherapies — and than placebo.[325]
2	**Cost is no longer a barrier. Some combinations were once many times the price of monotherapy**, but there is now little or no differential. Switching from Australia's most common regimen — 30-day dispensing of monotherapy — to 60-day dispensing of a single-pill combination would in fact reduce both patient and government costs.
3	**Monotherapy's low efficacy is masked by BP variability. Before-and-after measurements** in practice can create a false impression of a strong individual response: a patient whose BP falls more than 20/10 mm Hg after starting monotherapy owes most of that change to regression to the mean and measurement variability, not true drug response.[341,342] The converse also holds: an unchanged or higher BP at four weeks does not mean treatment has failed, as randomised trials show initial BP change does not predict true placebo-corrected efficacy. Virtually all monotherapies are low-efficacy, with little variation between patients, doses or classes — and regimens below 10/5 mm Hg have negligible probability of reaching the mean systolic level near 120 mm Hg required for sustained control below 130/80 mm Hg.

Primary aldosteronism management

Management of PA aims to control BP, correct hypokalaemia and reduce the CV and renal risks of aldosterone excess. For individuals not requiring specialist referral, commence a mineralocorticoid receptor antagonist (MRA).[5]

1. Start spironolactone 12.5–25 mg daily. Use lower doses and closer monitoring in frail or medically complex patients and where an ACE inhibitor or ARB is co-prescribed.
2. Review the individual's existing antihypertensives and consider stopping or reducing other agents at MRA initiation, guided by the severity of residual hypertension.
3. Stop potassium supplementation within 2–4 days of starting the MRA in all but the most severely hypokalaemic patients. Potassium usually normalises within 3–5 days. Patients who genuinely need ongoing supplementation will require regular potassium monitoring.
4. Review after 2–3 months, or sooner if clinically indicated. Assess BP, serum potassium, kidney function and renin. If BP remains above target and renin remains suppressed, intensify treatment. Table 23 provides guidance on dose titration and escalation of pharmacotherapy based on BP, biochemical results and treatment response.

Table 23: Primary aldosteronism dose titration and escalation based on primary care review

Review findings	Action
BP uncontrolled, renin suppressed	Increase the MRA dose. Spironolactone in 25–50 mg increments. If spironolactone is not tolerated, suggest eplerenone at twice the dose, taken twice a day, e.g. eplerenone 25 mg twice daily is approximately equivalent to spironolactone 25 mg daily. Titrate at 8–12-week intervals. Most patients de-suppress renin at spironolactone approximately 50 mg/day (or equivalent), higher than the doses typically used as add-on in resistant hypertension. Repeat electrolytes, kidney function and renin concentration 2–3 months after each change. Aim for fully unsuppressed renin, normal serum potassium concentration, and optimal blood pressure.
BP uncontrolled, renin fully unsuppressed	MR blockade is adequate. Add or up-titrate a non-MRA antihypertensive.
BP controlled, renin still suppressed, other antihypertensives in use	Consider increasing the MRA and stopping/reducing the other agents.
BP controlled, renin normal or increased from baseline	Routine follow-up and continue MRA monotherapy.

In more severe PA, the full effect of MRA therapy may take up to 3 months to establish. Once the dose has been optimised, routine long-term follow-up comprises blood pressure monitoring together with annual measurement of serum potassium, kidney function and renin concentration.[5] More frequent biochemistry is warranted in individuals with CKD, or where an MRA is combined with an ACE inhibitor or ARB. Renin measurement need not be repeated at

every review, but including it in the routine follow-up panel alongside BP, potassium and eGFR is often practical. A single blood collection covering all four parameters means the patient returns with the complete information needed for review, avoiding a second visit and additional pathology request if BP or potassium control proves incomplete and the titration algorithm is re-entered. Renin also provides useful information on adherence to therapy.

Practice points

Further practice points on avoiding treatment inertia, including reassessment timing, classifying BP as controlled, and 60-day dispensing, are set out in Treatment inertia, below. Two additional prescribing safety points apply specifically at initiation and escalation:

- Treat a confirmed high reading as real: if an appropriately measured AOBP is high, either initiate/intensify treatment or arrange ABPM/home BP monitoring — don't dismiss a high reading simply because a subsequent reading is lower.
- For women of childbearing age, document discussion of teratogenic risk, advise on contraception, and provide a pregnancy-safe alternative if pregnancy is planned — don't start ACE inhibitors or ARBs in this group without counselling about teratogenicity.

General

- Select therapy based on compelling indications, comorbidities, contraindications, and tolerability, with shared decision-making underpinning all prescribing decisions.

Rural, remote and regional

- Use telehealth for monitoring and titrating therapy where GP/specialist access is limited, with proactively scheduled reviews (e.g. 3–4 weeks after initiation, then 6-monthly if controlled and stable).[343]
- Default to 60-day dispensing for stable patients meeting PBS eligibility, given the reduced travel and cost burden in these settings.[20,30]
- Use structured case conferencing (nurses, nurse practitioners, Aboriginal and/or Torres Strait Islander Health Workers, pharmacists) to support monitoring where in-person access is limited.
- Where connectivity or digital health literacy is a barrier, ensure alternative pathways (phone consultations, Aboriginal community-controlled health organisations) are available.

First Nations peoples

- Follow the general drug-selection recommendations above, modified by comorbidity profile — noting the high prevalence of CKD and type 2 diabetes, and that rheumatic heart disease is concentrated almost entirely in this population (median age at diagnosis 33.3 years vs 65.4 in non-Indigenous Australians).[344]

Women

- Maintain awareness of contraindications in women of reproductive age or during pregnancy (obstetric hypertension is out of scope for this guideline).
- Cease ACE inhibitors, ARBs and direct renin inhibitors before or immediately upon confirmed pregnancy (fetotoxicity risk); avoid spironolactone (theoretical feminisation/growth restriction risk) and atenolol specifically among beta-blockers (growth restriction risk).[2]
- Where pharmacotherapy is needed in pregnancy, [2]prefer labetalol, methyldopa or extended-release nifedipine.[2]
- For women planning pregnancy on a contraindicated agent, review medication and transition to a pregnancy-safe alternative before conception; most antihypertensives (including ACE inhibitors/ARBs) can be resumed after delivery and are compatible with breastfeeding.[2] Monitor BP postpartum, as pre-eclampsia can present up to 6 weeks after delivery, and note that a history of a hypertensive disorder of pregnancy substantially raises lifetime CV risk (see Assessing CVD risk, above) — this history should prompt earlier and more regular CV risk assessment.

Older, very old and frail adults

- Do not stop antihypertensive therapy on the basis of frailty alone — HYVET showed benefit in both fitter and frailer participants aged ≥80.[345,346]
- Do not routinely deprescribe in fit, community-dwelling adults ≥80 with controlled BP.[300] Deprescribing may be reasonable in residential aged care where reducing medication burden is itself a goal, but is not recommended in moderate-to-severe dementia, where trial evidence shows possible harm (more delirium, falls, adverse events).[167,345-347]
- Approach deprescribing cautiously in patients on medications for heart failure, post-MI, AF, or diabetes — this guidance applies only to antihypertensives used for BP-lowering.
- Individualise decisions, weighing CV benefit against hypotension, frailty and polypharmacy risk. Where deprescribing occurs, use a structured taper with BP, falls, delirium and orthostatic monitoring for at least 4 weeks after each step.

Young adults

- Individualise therapy based on CVD risk, BP level, comorbidities and preferences — trial evidence shows clear benefit for those <55 years with no age-related modification of proportional treatment effect, despite sparse RCT data at this age.[292]
- Prioritise adherence support (SPCs, 60-day prescribing, telehealth or digital reminders) to maximise long-term CV protection.

8.3 Treatment algorithms

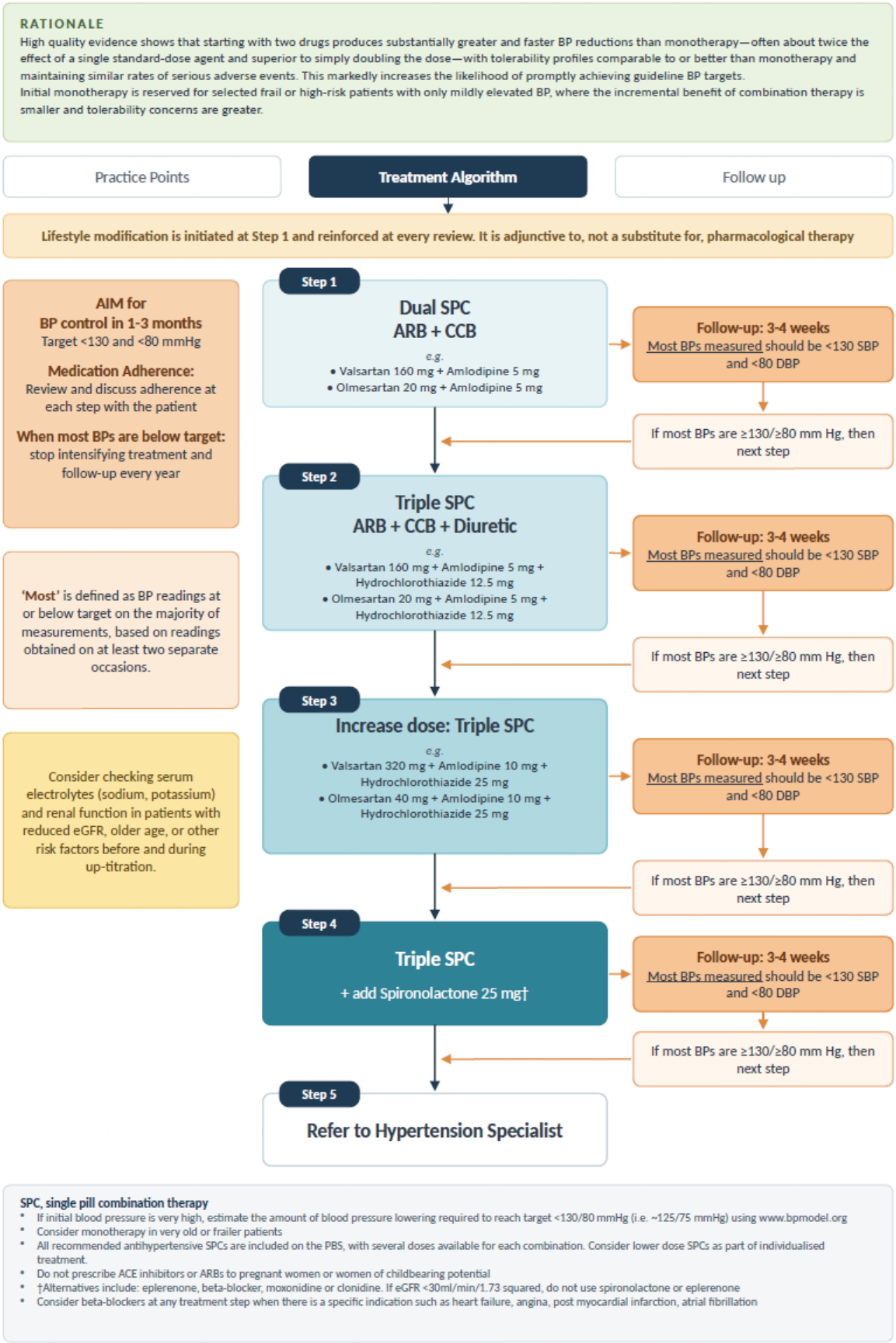


Figure 6: Treatment algorithm one

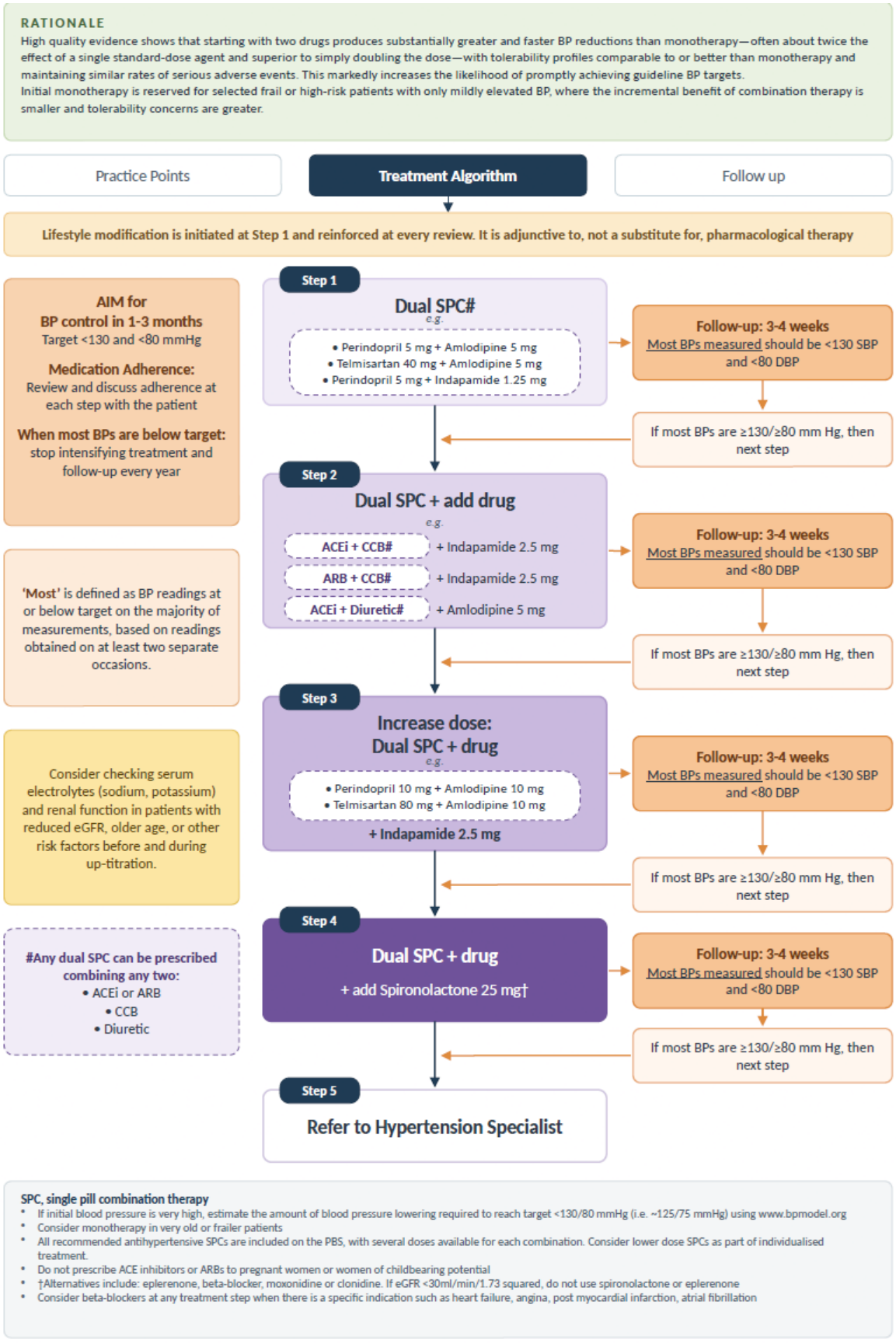


Figure 7: Treatment algorithm two

Figure 6 and Figure 7 present suggested treatment algorithms for individuals requiring pharmacotherapy. Table 24 presents the predicted BP-lowering efficacy of these medications and their combinations.

Table 24: Predicted BP-lowering efficacy for recommended classes and doses

Predicted BP-lowering efficacy for recommended classes and doses			
SPC	**+ free drug**	**SBP/DBP lowering targeting <130 and <80 mm Hg**	**Intensity**
Step 1			
Valsartan 160 mg + Amlodipine 5 mg		15/9 mm Hg	**Moderate**
Olmesartan 20 mg + Amlodipine 5 mg		15/9 mm Hg	**Moderate**
Perindopril 5 mg + Amlodipine 5 mg		15/10 mm Hg	**Moderate**
Telmisartan 40 mg + Amlodipine 5 mg		16/10 mm Hg	**Moderate**
Perindopril 5 mg + Indapamide 1.25 mg		14/9 mm Hg	**Moderate**
Step 2			
Valsartan 160 mg + Amlodipine 5 mg + Hydrochlorothiazide 12.5 mg		19/12 mm Hg	**Moderate**
Olmesartan 20 mg + Amlodipine 5 mg + Hydrochlorothiazide 12.5 mg		19/12 mm Hg	**Moderate**
Perindopril 5 mg + Amlodipine 5 mg	Indapamide 2.5 mg	25/14 mm Hg	**High**
Telmisartan 40 mg + Amlodipine 5 mg	Indapamide 2.5 mg	25/16 mm Hg	**High**
Perindopril 5 mg + Indapamide 1.25 mg	Amlodipine 5 mg	22/13 mm Hg	**High**
Step 3			
Valsartan 320 mg + Amlodipine 10 mg + Hydrochlorothiazide 25 mg		24/14 mm Hg	**High**
Olmesartan 40 mg + Amlodipine 10 mg + Hydrochlorothiazide 25 mg		25/15 mm Hg	**High**
Perindopril 10 mg + Amlodipine 10 mg	Indapamide 2.5 mg	29/17 mm Hg	**High**

Telmisartan 80 mg + Amlodipine 10 mg	Indapamide 2.5 mg	29/17 mm Hg	**High**
Step 4			
Valsartan 320 mg + Amlodipine 10 mg + Hydrochlorothiazide 25 mg	Spironolactone 25 mg	28/17 mm Hg	**High**
Olmesartan 40 mg + Amlodipine 10 mg + Hydrochlorothiazide 25 mg	Spironolactone 25 mg	29/18 mm Hg	**High**
Perindopril 10 mg + Amlodipine 10 mg	Indapamide 2.5 mg Spironolactone 25 mg	33/20 mm Hg	**High**
Telmisartan 80 mg + Amlodipine 10 mg	Indapamide 2.5 mg Spironolactone 25 mg	33/20 mm Hg	**High**

Assuming a baseline BP of 154/100 mm Hg and maximum effect after 4 weeks.

Definition of low, moderate and high intensity: 0–9.9, 10–19.9 and ≥20 mm Hg SBP reduction from a baseline SBP of 154 mm Hg, respectively.

The term "low-dose" is used inconsistently across the hypertension literature. In the low-dose combination trial literature it denotes any dose below the standard dose, which is usually the same as WHO defined daily dose, with a few exceptions – see Wang, Salam [322]. For clarity and to align with international guidance, this document instead uses the terms "recommended starting dose" and "lowest available dose formulation", which for many agents are equivalent to the standard dose. The doses listed in this table are starting or lowest-available doses, which are usually standard doses as few dual combinations registered in Australia at the time of writing have both components at less than standard dose.

Based on www.bpmodel.org Wang, Salam [322]

8.3.1 Titration and dose optimisation

If BP isn't controlled on initial combination therapy at standard doses, add a third drug class (ideally as a triple SPC) rather than escalating existing doses — doubling a single agent's dose adds only approximately 1–2 mm Hg SBP reduction, versus 3–5 times more effect from adding a different class.[3,322,323] The same logic applies to dose escalation within a class more generally, so favour adding or switching to a complementary lowest available formulation agent over pushing doses higher, given the disproportionate rise in dose-dependent adverse events.[135,319,321,322]

If BP remains uncontrolled on maximally tolerated triple SPC therapy, increase to maximum tolerated doses before adding a fourth agent, in line with the ESH 2023/ESC 2024 step-care approach.[3,92] At any step, if a dose increase causes intolerable effects, substitute a lowest available formulation agent from a different class rather than persisting — the ALARA (As Low As Reasonably Achievable) principle supports targeting the lowest effective BP while minimising harm.[3]

8.3.2 Timing of dosing

The large TIME trial found no difference in cardiovascular outcomes between morning and evening dosing, superseding earlier smaller trials that had suggested an evening-dosing benefit.[348] Individualise dosing time for adherence and tolerability instead — e.g. evening

dosing for patients who reliably forget morning doses, or morning dosing to reduce nocturia from diuretics — rather than routinely recommending evening dosing.

8.4 Treatment inertia

Key point — inertia and non-adherence are different problems

- **Treatment inertia** is a clinician-level phenomenon: the absence of treatment initiation or intensification when clinical guidelines indicate it is warranted.
- **Patient non-adherence** is a patient-level phenomenon: medication-taking behaviour that does not correspond with the agreed regimen, whether as non-initiation, suboptimal implementation, or early discontinuation.
- Establish which is operating before acting. The remedies differ, and treating one as the other wastes the consultation.

Treatment (clinical/therapeutic) inertia — clinicians failing to initiate or intensify therapy when guidelines indicate doing so, distinct from patient non-adherence — is historically underemphasised relative to adherence, despite being equally consequential.[349-352] Each visit a patient with uncontrolled hypertension leaves without action is a missed opportunity for population-level control. Standardised protocols, treatment algorithms, SPC therapy and structured follow-up combat this at the point of care; as a general guide, if the proportion of high readings (BP load) exceeds 20%, intensify treatment if clinically appropriate.

8.4.1 Factors associated with treatment inertia

Treatment inertia is multifactorial, operating at clinician, patient, and clinical-complexity levels.[353]

Clinician-related treatment inertia

Failure to acknowledge an elevated reading; diffusing responsibility to another provider without follow-up; unclear roles, time pressure, workload and infrastructure gaps; overestimating one's own care quality; soft deferrals ('wait for next appointment'); and gaps in training on targets, dosing and polypharmacy.[350-353] A common pitfall is 'remeasure to check' — treating a lower follow-up reading as proof the first was false, when it's usually just normal variability. Regard BP as controlled only when the average of ≥2 standardised measurements on ≥2 separate occasions at or below target.

Patient-related treatment inertia

Clinicians may not intensify treatment in response to non-adherence, or defer to patient preference to avoid medication change; patient unawareness, low motivation or fear of adverse effects also contribute.[352,353] Despite common clinician assumptions, physician inertia — not patient refusal — accounts for roughly 75% of failures to intensify.[352] Older age, near-target SBP, and diabetes are associated with higher patient-related inertia.[350,354]

Treatment inertia related to clinical complexity

Competing medical priorities, maintaining current treatment, and — most commonly — uncertainty about whether a reading reflects the patient's true BP.[350,353]

8.4.2 Strategies to overcome treatment inertia

Treatment inertia is a complex phenomenon for which no single solution is sufficient. A systematic review and meta-analysis of 10 RCTs (n=26,871) found that interventional strategies targeting clinical inertia were associated with significantly improved BP control, with all individual intervention types demonstrating benefit in favour of the intervention group.[353] Table 25 has a list of strategies.

Table 25: Strategies to overcome treatment inertia

At this point	Do	Don't
Measuring BP	Use AOBP as standard before treatment decisions	Don't act on an unstandardised reading
BP above target	Intensify same visit; add an agent (about 3 – 5 times more effective than up-titrating monotherapy)	Don't defer without a documented clinical reason
Choosing therapy	Prescribe SPC therapy when two or more agents are indicated	Don't wait for the next GP appointment — refer to multidisciplinary care (practice nurse, pharmacist)
Reassessing	Actively review home BP monitoring results and incorporate into treatment decisions	Don't ignore or simply file home readings
Ending the visit	Explain clearly why treatment is being intensified and what the target is (<130/80 mm Hg)	Don't leave a deferred decision open-ended — document the rationale and schedule a follow-up date

In rural, remote and regional settings, apply the same telehealth, 60-day dispensing and case-conferencing approaches described in Pharmacotherapy for blood pressure, above.

For clinicians who actively address their own clinical inertia, patient-related barriers can become a dominant reason for failure to intensify, underscoring the need for systematic strategies to engage patients, explain the rationale for up-titration, and address concerns about adverse effects. Ensuring that patients are adequately counselled about why treatment intensification is necessary, particularly when there is compelling evidence of undertreatment, may help reduce this form of inertia and improve BP control.

8.4.3 Treatment adherence

Medication adherence is a significant barrier to effective BP control, which contributes to preventable morbidity and mortality.[355] Observational data suggest that approximately half of people prescribed antihypertensives stop treatment within a year, 48% have at least one break from treatment each year, and almost all miss doses, particularly on weekends.[356]

Single-pill combinations

Evidence demonstrates that SPCs improve adherence and persistence compared with the same agents taken separately, with higher proportions of patients achieving good adherence and remaining on therapy over 12 months, accompanied by small but meaningful improvements in systolic BP.[326,327,357] SPCs should therefore be used whenever suitable products and PBS listing are available.

Coordinated care

In adults with polypharmacy (≥5 medicines), coordinated care that combines simplified cardiovascular regimens, prescriber continuity, and harmonised dispensing is associated with significantly better antihypertensive adherence, with the highest adherence seen when all three components are present.[358] Team-based models- involving doctors, nurses and pharmacists are increasingly supported by evidence and should be prioritised where feasible.[358]

Digital health interventions

Digital health interventions can support adherence and BP control. Meta-analysis of randomised trials indicates that mHealth tools (apps, SMS reminders, wearables) improve medication adherence and reduce systolic BP, while telehealth (remote consultations and monitoring) mainly improves BP but has less consistent effects on adherence.[359] Combining mHealth with telehealth appears to produce the largest BP reductions and better BP control, though data on adherence with combined modalities remain limited.[359]

Theory-informed behavioural interventions

Theory-based behavioural- interventions grounded in Social Cognitive Theory, the Transtheoretical Model and Motivational Interviewing can improve antihypertensive adherence, particularly when tailored to individual context and delivered via pharmacists, nurses, digital tools or mixed formats.[360] Several trials show clinically important gains, including pharmacist-led interventions- and SCT-based mobile apps-, reinforcing the value of structured, behaviourally informed support.[360]

Sociodemographic and clinical determinants of adherence

Observational data from over 23.5 million adults in the US identify consistent determinants of adherence: older age, being married, having health insurance and higher education are associated with better adherence, while heavy alcohol use and comorbidities such as depression, diabetes, Alzheimer's disease and CKD are associated with poorer adherence.[361] Sex and BMI are not significantly related to adherence.[361] These findings highlight groups at higher risk of non-adherence, although direct Australian equivalence cannot be assumed.[361]

Practice points

First Nations adherence

- Do not assume lower adherence without evidence — a systematic review found no conclusive evidence of inherently lower adherence, despite clinicians commonly perceiving it as a problem.[362]
- Use dose administration aids, regimen simplification and once-daily dosing; increase Aboriginal and Torres Strait Islander Health Practitioner availability; integrate pharmacists into Aboriginal community-controlled health services; provide health literacy support; and strengthen culturally appropriate self-management.[362]

Culturally and linguistically diverse communities

- Address adherence barriers linked to health literacy, communication and cultural/religious perceptions of medicines using bilingual staff, translated materials, and frameworks such as cognitive behavioural therapy or motivational interviewing — but not bilingual or translated materials alone, which are insufficient.[363-365]
- Favour culturally grounded, co-designed interventions where possible, noting the current evidence gap for this population.

8.5 Device-based therapy for confirmed hypertension

8.5.1 Catheter-based renal denervation

Sustained activation of the sympathetic nervous system is an important contributor to the development and progression of hypertension. Catheter-based renal denervation (RDN) is a minimally invasive approach to selectively ablate the renal nerves travelling to and from the kidneys. Sham-controlled clinical trials have demonstrated a favourable safety profile with significant and clinically meaningful reduction in office and ambulatory BP that was sustained up to at least three years.[366,367] Meta-analysis of sham-controlled trials showed modest but consistent reductions of approximately −4 to −7 mm Hg in ambulatory SBP that were sustained to 36 months.[366,367] Real-world observational data from large clinical registries confirmed the safety, efficacy and durability of the procedure out to approximately 10 years.[368-371] However, RDN has not yet been demonstrated in an adequately powered randomised trial to reduce cardiovascular morbidity or mortality and the individual response to RDN is variable.

In line with international guidelines, catheter-based RDN should be considered as an adjunctive blood pressure-lowering treatment in appropriately selected adults.[2,3,92] These include those with uncontrolled hypertension, particularly those with true resistant hypertension despite guideline-directed combination pharmacotherapy, those with high cardiovascular risk, and selected patients in whom adequate pharmacological blood pressure control cannot be achieved because of intolerance, clinically important adverse effects, or inability to sustain appropriate antihypertensive therapy.

Before renal denervation is considered:

- Uncontrolled hypertension should be confirmed using out-of-office blood pressure measurement, preferably 24-hour ambulatory blood pressure monitoring.

- Pseudo-resistant hypertension should be excluded, including inaccurate blood pressure measurement, white-coat effect, suboptimal treatment, medications or substances that increase blood pressure, and medication non-adherence.
- Secondary causes of hypertension should be systematically investigated and, where identified, appropriately treated.
- Guideline-directed healthy lifestyle and pharmacological treatment should be optimised. In patients with true resistant hypertension, this will generally include a renin–angiotensin system blocker, calcium-channel blocker and thiazide/thiazide-like diuretic, with addition of a mineralocorticoid receptor antagonist when appropriate and tolerated.
- Renal function and renal artery anatomy should be suitable for the procedure. Based on the populations studied in randomised trials and current guideline recommendations, renal denervation should generally be restricted to patients with eGFR ≥40 mL/min/1.73 m^2 and without significant renal artery disease or other anatomical contraindications.

Patient selection should include a shared decision-making process and occur through a multidisciplinary hypertension team, including clinicians experienced in resistant hypertension and an appropriately trained renal denervation interventionalist. The procedure should be performed in an experienced centre with established pathways for patient selection, procedural performance, and follow-up.

Three radio-frequency ablation system renal denervation catheters are TGA-registered for use in Australia, but RDN is only available through self-funded private pathways in the current absence of an MBS item number.

8.6 Emerging therapies

Several novel-mechanism antihypertensives may offer new escalation options for uncontrolled hypertension, but none are yet TGA-registered or PBS-listed — summarised here for awareness, not as current options.

8.6.1 Aldosterone synthase inhibitors

Aldosterone synthase inhibitors (ASIs) lower BP by blocking aldosterone production. In Phase 3 trials, baxdrostat and lorundrostat reduced SBP by roughly 9–12 mm Hg versus placebo at 12 weeks in resistant or uncontrolled hypertension, with 40–44% of patients reaching target — around double the placebo rate.[372,373] The main safety concern is a class-wide increase in hyperkalaemia risk (approximately seven-fold versus placebo), establishing potassium elevation as the principal safety consideration for this therapeutic approach.[374] ASIs have potential as a future escalation option for patients uncontrolled on standard combination therapy.

8.6.2 RNA interference therapy

Zilebesiran lowers BP via RNA interference, with durable effects that could simplify treatment delivery if confirmed in outcomes trials. In Phase 2 trials, monotherapy achieved placebo-

adjusted 24-hour systolic reductions of 14–17 mm Hg over six months, with more variable reductions when added to existing therapy.[375,376] Clinical use remains contingent on Phase 3 outcomes and longer-term safety data.

8.6.3 Dual endothelin receptor antagonists

Aprocitentan blocks endothelin-1, a pathway implicated in salt-sensitive, obesity-related and CKD-associated hypertension. In the Phase 3 PRECISION trial (resistant hypertension on triple therapy), aprocitentan produced modest but sustained BP reductions over 48 weeks, with larger effects in a post-hoc analysis of participants with high-risk CKD.[377-380] It is not registered with the TGA or available in Australia, but is best positioned as a mechanistically distinct add-on for genuinely resistant hypertension, particularly with CKD and albuminuria, should it become available locally.

8.7 Follow-up and monitoring

Following initiation or any change in therapy, review every 3–4 weeks: adherence, BP response (including home readings where available), tolerability, and adverse effects. Review sooner if baseline BP is markedly elevated or comorbidities warrant it. Where BP is not yet at target, intensify at that visit rather than deferring, and add a class in preference to increasing a dose. Home BP monitoring is particularly useful for titration where in-person access is limited, such as regional and remote areas, supported by tele-pharmacy and structured case conferencing.

Check electrolytes and creatinine before starting any ACE inhibitor, ARB or diuretic, and again 1–2 weeks after starting or up-titrating one of these classes — for all patients, not only those already flagged as high risk — to detect hyperkalaemia or significant renal change early. This matters most in older adults and in CKD, diabetes or concurrent NSAID use, but unexpected deterioration can occur in anyone. A small early fall in eGFR after starting an ACE inhibitor or ARB is expected and is not a reason to stop: long-term trials show these agents are renoprotective, and cycling through alternative agents in response to a minor eGFR change delays BP control.[381]

Once BP is at target (<130/80 mm Hg) across two consecutive visits, extend review to 6-monthly. Some variation in BP over time is expected even when controlled; 6-monthly reviews reinforce healthy lifestyle modification, reassess adherence, identify new risk factors, and optimise prescribing — including 60-day dispensing per current PBS policy, noting that uptake remains suboptimal.[30] At least annually, assess for new cardiovascular risk factors and end-organ damage.

8.8 Monitoring schedule

The guidance from across this guideline is summarised in Table 26 and Figure 8.

Table 26: Monitoring and review schedule for hypertension management

Stage	Timing	What to check
Before starting an ACE inhibitor, ARB or diuretic	At prescribing	Electrolytes and creatinine — baseline for all patients, not only those flagged high risk.
After starting or up-titrating an ACE inhibitor, ARB or diuretic	1–2 weeks	Electrolytes and creatinine, to catch hyperkalaemia or significant renal change. Applies to everyone; matters most in older adults, CKD, diabetes, or concurrent NSAID use. A small acute fall (≤30% in eGFR) after starting an ACE inhibitor or ARB is expected and is not a reason to stop or change agent.[381]
After initiating or changing antihypertensive therapy	3–4 weeks	BP response including home readings, adherence, tolerability, adverse effects. Review sooner if baseline BP markedly elevated or comorbidities warrant.
Not yet at target	Every 3–4 weeks until controlled	Intensify at the visit rather than deferring. Add a class in preference to increasing a dose.
At target across two visits	6-monthly	Reinforce healthy lifestyle change, reassess adherence, identify new risk factors, optimise prescribing including 60-day dispensing.
Continuing pharmacotherapy, stable	Annually	New cardiovascular risk factors and end-organ damage.
Primary aldosteronism — after starting an MRA	1–2 weeks at initiation then 2–3 months	BP, serum potassium, kidney function and renin. Full effect may take up to 3 months in more severe PA.
Primary aldosteronism — during titration	Every 8–12 weeks	Repeat electrolytes, kidney function and renin 2–3 months after each dose change. Aim for unsuppressed renin, normal potassium and BP at target.
Primary aldosteronism — dose optimised	Annually	Potassium, kidney function and renin — measured together in a single annual collection. More often with CKD, or where an MRA is combined with an ACE inhibitor or ARB.
Rural and remote, telehealth-supported	3–4 weeks after initiation, then as above	Same content; use tele-pharmacy and structured case conferencing. Default to 60-day dispensing for stable patients meeting PBS eligibility.

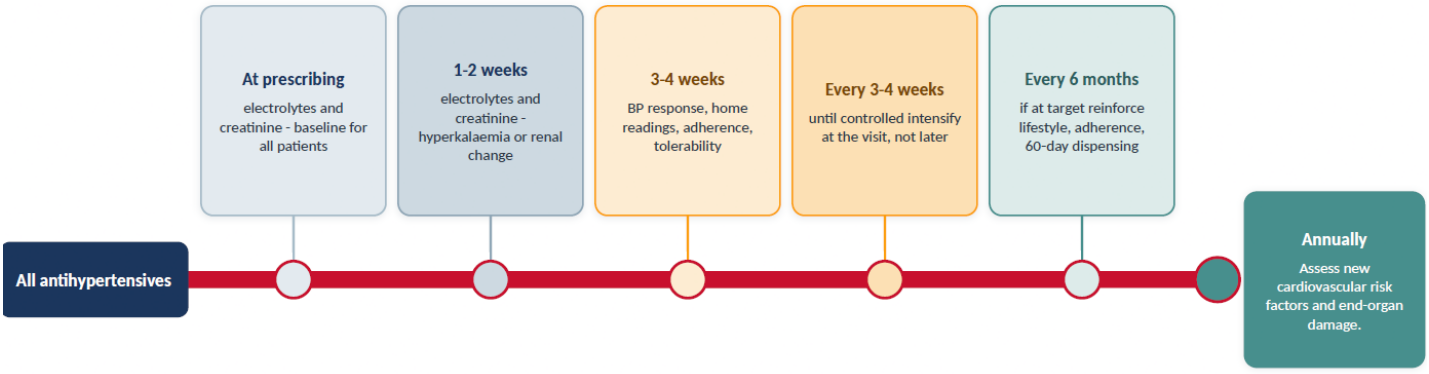


Figure 8: Monitoring schedule

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