Clinical hypertension

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Abstract

Hypertension is one of the most prevalent modifiable contributors to global morbidity and mortality. In high-income countries such as the UK, it affects nearly 1 in 3 adults and disproportionately impacts individuals of ethnic minority backgrounds, particularly those of African or African–Caribbean heritage, who experience earlier onset, poorer blood pressure (BP) control and higher rates of cardiovascular complications. Despite effective therapies, many patients remain undiagnosed or inadequately treated. Accurate BP measurement is essential for diagnosis and requires confirmation with home or ambulatory BP monitoring in most cases. Evaluation for hypertension-mediated organ damage refines risk assessment and guides treatment intensity. Secondary hypertension accounts for 5–10% of cases, and identification of reversible causes is clinically important as targeted therapy can significantly improve BP control. Management is based on BP severity and overall cardiovascular risk. Lifestyle modification is the foundation of care, but most patients require antihypertensive medication, whether monotherapy or combination therapy. Resistant hypertension remains challenging; once pseudo-resistance and secondary causes are excluded, affected individuals may need intensified pharmacotherapy, and in selected cases specialist interventions such as renal denervation can be considered.

Keywords Ambulatory blood pressure monitoring; antihypertensive medications; blood pressure; cardiovascular risk; hypertension; resistant hypertension; secondary hypertension

Key points

- Hypertension is common and often underdiagnosed, affecting ~1 in 3 UK adults; many remain poorly controlled, contributing to substantial preventable cardiovascular risk
- Accurate diagnosis requires standardized, repeated blood pressure measurements, typically including out-of-office ones
- Assessment of hypertension-mediated organ damage is essential, as it guides treatment strategies particularly in young individuals and in people at low cardiovascular risk
- Lifestyle modification underpins all management, but most patients require pharmacological therapy, often with combination treatment to achieve target blood pressure targets
- Resistant hypertension demands systematic evaluation, including exclusion of pseudo-resistance and secondary causes, with stepwise intensification of therapy and often referral for specialist assessment

Introduction

Hypertension is the single most important modifiable risk factor for cardiovascular (CV) disease and remains a major global public health challenge in both high- and low-to-middle-income countries.[1] For example, in the UK, it affects >10 million people, representing nearly 1 in 3 adults; its prevalence continues to rise partially because of increasing age, obesity and socioeconomic deprivation in the population.

The burden of hypertension is not evenly distributed: individuals from ethnic minority backgrounds, particularly those of African or African–Caribbean heritage, experience earlier onset, poorer blood pressure (BP) control and higher rates of CV complications compared with white individuals living in the Northern hemisphere.

Large-scale clinical trials and meta-analyses have consistently demonstrated that antihypertensive therapy markedly reduces the risk of CV morbidity and mortality, showing a linear relationship between the degree of BP reduction and decreases in adverse CV events.

Despite the availability of treatments, it is estimated that globally up to half of individuals with hypertension remain undiagnosed or untreated, and many of those who are treated fail to achieve optimal BP control. This persistent gap in detection and management places a significant and growing burden on healthcare systems, contributing to preventable hospital admissions each year and substantial costs for individuals and the public.

Epidemiology and pathophysiology

Within populations, BP follows a continuous, approximately normal distribution, meaning that the definition of hypertension is, to some extent, arbitrary. Diagnostic thresholds are based not on natural cut-offs, but on the levels of BP associated with increased CV risk, which explains the variation in criteria used across the world. These differences reflect distinct interpretations of risk–benefit trade-offs in different healthcare contexts.

At the global level, marked geographical variations exist in the prevalence and rate of control of hypertension, reflecting a complex interplay of healthcare-related, environmental, genetic and gene–environment factors.

The prevalence of hypertension increases markedly with age, rising from approximately 4% in children and young adults, to 10% in those <40 years old, and >50% among those ≥60 years. Isolated systolic hypertension is the most common hypertensive phenotype among young men, whereas systolic–diastolic

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hypertension and isolated diastolic hypertension are more frequent in young women. In middle age, systolic–diastolic hypertension predominates, while in elderly individuals, isolated systolic hypertension again becomes the most common phenotype.

The underlying mechanisms probably differ: systo-diastolic hypertension has been attributed to increased peripheral vascular resistance, whereas isolated systolic hypertension appear to be related to an increased cardiac output (in young individuals) and arterial stiffening, and reduced vascular compliance in elderly patients.

Regarding aetiology, hypertension has been divided in two distinct forms: primary (or essential) and secondary. Primary hypertension accounts for most cases. It is characterized by the absence of a clearly identifiable secondary cause and is believed to arise from a complex interaction between environmental and inherited factors.

In contrast, secondary hypertension arises from a specific underlying condition and comprises approximately 5–10% of hypertensive patients. Estimates of the prevalence of secondary hypertension vary according to the age group, population studied and extent of specialist investigations undertaken.

In young adults coarctation of the aorta, phaeochromocytoma, Cushing syndrome and thyroid disease are rare, with a prevalence among people with young-onset hypertension of $\leq$1%. In contrast, primary hyperaldosteronism, renal artery stenosis and renal parenchymal disease are the most common aetiologies of secondary hypertension. Renal artery stenosis in younger adults more commonly results from fibromuscular dysplasia, Takayasu arteritis or neurofibromatosis than atherosclerosis, which is more typically diagnosed in elderly individuals).

Recognition of secondary forms is important because targeted treatment of the root cause can lead to improved BP control and reduced organ damage. A brief overview of common causes of secondary hypertension and recommended investigations is presented in Table 1.

Diagnosis and evaluation

The diagnosis of hypertension should be based on accurate and repeated BP measurements (see BP measurement), taken under standardized conditions using validated equipment.

Most international guidelines recommend that, in conjunction with measurements performed in a clinical setting ('office measurements'), measurements performed out of office (home BP monitoring' (HBPM) and/or ambulatory BP monitoring (ABPM)) are recorded to confirm the diagnosis. In specific circumstances (e.g. individuals with markedly elevated BP values and evidence of organ damage), HBPM/ABPM might not be performed because the clinical situation requires prompt initiation of pharmacological treatment.

Once the diagnosis of hypertension is established, clinicians should undertake a systematic assessment to identify potential secondary causes, evaluate target organ damage and assess the patient's overall CV risk profile. A careful history, physical examination and targeted investigations guide both the identification of underlying causes and the development

Common causes of secondary hypertension

Condition	Clinical suspicion	Investigations
Kidney disease	Usually asymptomatic with only incidental findings of elevated serum creatinine or abnormalities in the urinary sediment. People with diffuse atherosclerotic disease can show a fall in eGFR after treatment with ACEi/ARB	Serum creatinine, urine dipstick, urine sample for albumin:creatinine ratio and protein:creatinine ratio
Primary hyperaldosteronism	Hypokalaemia, adrenal incidentaloma. Most cases are detected in asymptomatic individuals with normokalaemia	Renin/aldosterone ± adrenal imaging (adrenal MRI/HRCT) followed by confirmatory tests and/or adrenal vein sampling
Obstructive sleep apnoea	Snoring, daytime sleepiness, elevated BMI or neck circumference and non-dipping profile on 24-h ABPM	Validated questionnaires such as Epworth Sleepiness scale or STOP-BANG score. Overnight oxygen saturation monitor
Phaeochromocytoma and paraganglioma	Paroxysmal or sustained hypertension, headaches, palpitations, hyperhidrosis, and non-CV symptoms such as weight loss or hypoglycaemia	Plasma free or urinary fractionated metanephrines ± adrenal MRI/HRCT ± functional imaging and/or clonidine suppression test
Cushing syndrome	Truncal obesity, striae, glucose intolerance and repeated infections	Overnight dexamethasone suppression test and 24-h urine free cortisol
Thyroid and parathyroid disorders	Usually asymptomatic: symptoms include palpitations, tiredness, polyuria, muscle weakness, anxiety, tremor and irritability	Evaluation of calcium, PTH, TSH and thyroid hormones. Thyroid and parathyroid ultrasonography
Aortic coarctation	Usually asymptomatic: signs include radiofemoral delay and lower BP in the lower extremities	MRA/CT of the aorta

BMI, body mass index; CT, computed tomography; HRCT, high-resolution computed tomography; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; PTH, parathyroid hormone; TSH, thyroid-stimulating hormone. See text for other abbreviations.

Table 1

of an individualized management plan. Table 2 summarizes the key elements that should be evaluated in the medical history.

Although the physical examination can be completely normal in individuals with hypertension, it remains important as it can provide valuable clues to identify secondary causes of hypertension and other conditions that can influence the overall CV risk assessment. In particular, clinicians should look for signs of Cushing syndrome, acromegaly and thyroid disorders.

A radio-radial or radiofemoral delay, or lower BP in the extremities, can indicate aortic coarctation. Systematic auscultation of the heart for murmurs, and of the carotid, renal and femoral arteries for bruits, should be performed. Finally, fundoscopy (especially in individuals with markedly elevated BP) should be conducted to detect moderate to severe target organ damage.

BP measurement

BP is a dynamic physiological parameter, influenced by a range of internal and external factors, including circadian rhythms, hormonal fluctuations, seasonal variation and immediate environmental or emotional stimuli. Consequently, a single measurement rarely reflects an individual's true or usual BP.

To ensure diagnostic accuracy, it is important to record multiple BP readings during a single consultation and to repeat measurements across several clinical encounters. This approach helps to minimize random variability and regression to the mean, yielding a more reliable estimate of the patient's usual BP. BP should be measured using validated devices with appropriately sized cuffs.

Office BP measurement remains the most accessible and widely used initial screening method for detecting hypertension. Accurate measurement requires adherence to standardized procedures and validated equipment. In the UK, a British and Irish Hypertension Society (BIHS)-approved device should be used, with an appropriately sized cuff whose bladder encircles 80–100% of the upper arm circumference. Recommendations on how to perform BP measurements are summarized in Figure 1.

BP should be measured at least twice (ideally three times), 1–2 minutes apart, and the average of the final two readings recorded. At the first visit, BP should be taken in both arms, with subsequent readings from the arm with the higher value.

In patients with postural symptoms or risk factors for autonomic dysfunction (e.g. older age, diabetes, use of certain medications) BP should also be measured supine and after 1 minute of standing. A fall of ≥20 mmHg systolic or ≥10 mmHg diastolic indicates postural hypotension; in these cases, standing BP should guide hypertension management.

Blood pressure thresholds for diagnosis

In line with clinical practice in the UK, if the average clinic BP is >140/90 but <180/120 mmHg, diagnosis should be confirmed with ABPM/HBPM using a threshold of 135/85 mmHg. In severe hypertension (≥180/120 mmHg), out-of-office testing is not always necessary, as urgent assessment depends on symptoms and evidence of hypertension-mediated organ damage (HMOD), typically evaluated in specialist settings.[2]

ABPM requires at least two readings per hour during waking hours and a minimum of 14 valid readings. HBPM should involve two seated readings, 1 minute apart, taken twice daily for 4–7 days; the first day's measurements should be discarded, and the average of the remaining values used.

Hypertension can be classified into stages based on BP values (Table 3). Combining clinic and out-of-office readings helps identify white-coat hypertension and masked hypertension. Both are relatively common phenotype.

Important elements of the medical history to be considered

Element	Information to gather
History related to hypertension	• Time of first diagnosis of hypertension • Current/past antihypertensive medications including intolerances • Concordance with therapy • History of erectile dysfunction • Previous hypertension in pregnancy, pre-eclampsia, fetal growth restriction
Family history and co-morbidities	• Family history of hypertension, cardiovascular (CV) disease, stroke, kidney disease • Co-morbidities and CV risk factors • Sleep history, snoring, sleep apnoea
Concurrent medications (noting drugs that can increase blood pressure)	• Prescribed medication including but not limited to: combined oral contraceptive pill or implants, hormone substitutes, corticosteroids, non-steroidal anti-inflammatory drugs, selective serotonin–noradrenaline reuptake inhibitors, cancer drugs, dexamphetamine, methylphenidate • Over-the-counter medications, recreational drugs and substance abuse
Lifestyle	• Tobacco smoking history: current, ex or never (pack–years) • Dietary history: fruit and vegetable intake, bread and breakfast cereal intake, salt intake, alcohol consumption, liquorice • Physical activity per week • Weight gain or loss

Table 2

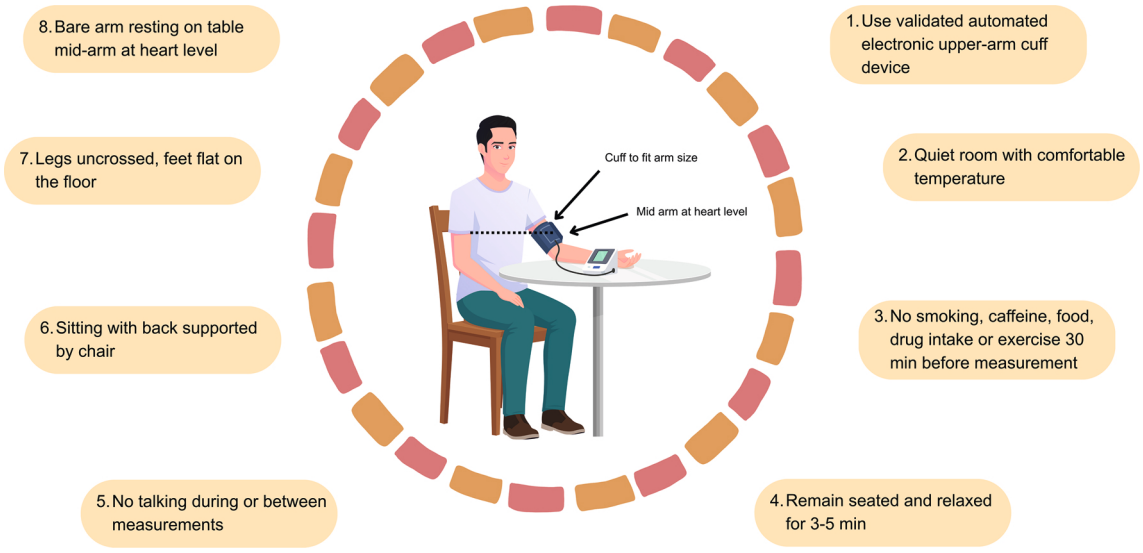


Figure 1 Recommendation for blood pressure measurements. Adapted from Reference 4.

CV risk estimation and assessment of hypertension-mediated organ damage

Evaluating overall CV risk is central to managing hypertension, helping to identify individuals most likely to benefit from early intervention. Risk calculators such as QRISK3 (UK) and SCORE2 (Europe) integrate factors including age, sex, BP, cholesterol concentrations, smoking and diabetes to estimate 10-year CV event risk.

Patients with a high or very high risk are prioritized for intensive lifestyle modification and earlier pharmacological therapy. However, these tools perform less well in younger individuals, whose absolute risk can appear low despite multiple risk factors or early organ damage. Ethnicity is also not consistently incorporated in many CV risk score, despite its independent influence on CV risk.

For these reasons, identifying HMOD is crucial, as it improves CV risk prediction and helps determine treatment intensity. Assessment should include clinical and subclinical markers across several organs (Figure 2). Many forms of HMOD can stabilize or regress with effective BP control, highlighting the importance of periodic reassessment.

Cardiac assessment: left ventricular hypertrophy (LVH) is one of the most important and commonly evaluated forms of HMOD because of its strong prognostic value. In primary care, electrocardiography (ECG) offers a practical screening tool, although it lacks sensitivity. Echocardiography is the reference standard, enabling an assessment of left ventricular mass, diastolic function and left atrial size, helping to distinguish hypertensive remodelling from other cardiomyopathies.

Vascular assessment: vascular HMOD can be assessed using measures of arterial stiffness or structural remodelling. Carotid–femoral pulse wave velocity (PWV) is the gold standard for aortic stiffness assessment and strongly predicts CV events. Brachial–ankle PWV is widely used in some regions but less validated in Europe. Carotid ultrasonography can identify carotid plaque, a robust marker of atherosclerosis, although intima–media thickness is no longer routinely recommended as it has limited predictive value.

Renal assessment: renal damage is evaluated using estimated glomerular filtration rate (eGFR) and urinary albumin: creatinine ratio (ACR). Even mild albuminuria is associated with increased CV risk and faster renal decline. Renal ultrasonography is reserved for suspected secondary causes or structural abnormalities.

Stages of hypertension and their definition according to NICE recommendations[3]

Terminology	Definition
Stage 1 hypertension	Clinic BP ranging from 140/90 to 159/99 mmHg and subsequent average daytime ABPM or average HBPM BP ranging from 135/85 to 149/94 mmHg
Stage 2 hypertension	Clinic BP ≥160/100 but <180/120 mmHg and subsequent ABPM daytime average or HBPM average BP ≥150/95 mmHg
Stage 3 or severe hypertension	Clinic systolic BP ≥180 mmHg or clinic diastolic BP ≥120 mmHg
White-coat hypertension	Clinic BP measurements are elevated (>140/90 mmHg), but BP measurements are normal when taken outside the clinic using average daytime ABPM or average HBPM BP measurements of <135/85 mmHg
Masked hypertension	Clinic BP measurements are normal (<140/90 mmHg), but BP measurements are higher when taken outside the clinic using average daytime ABPM or average HBPM BP measurements

Table 3

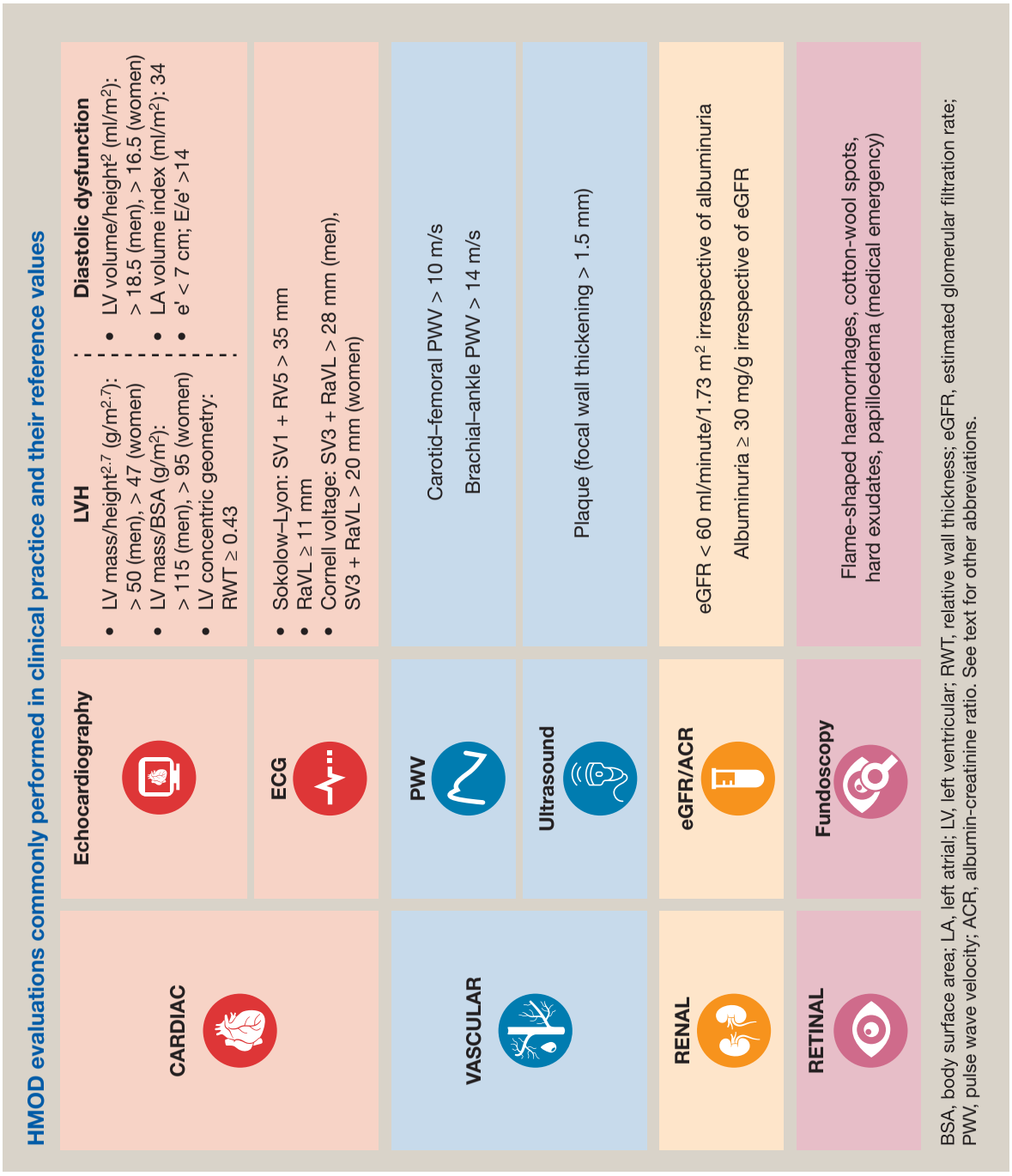


Figure 2

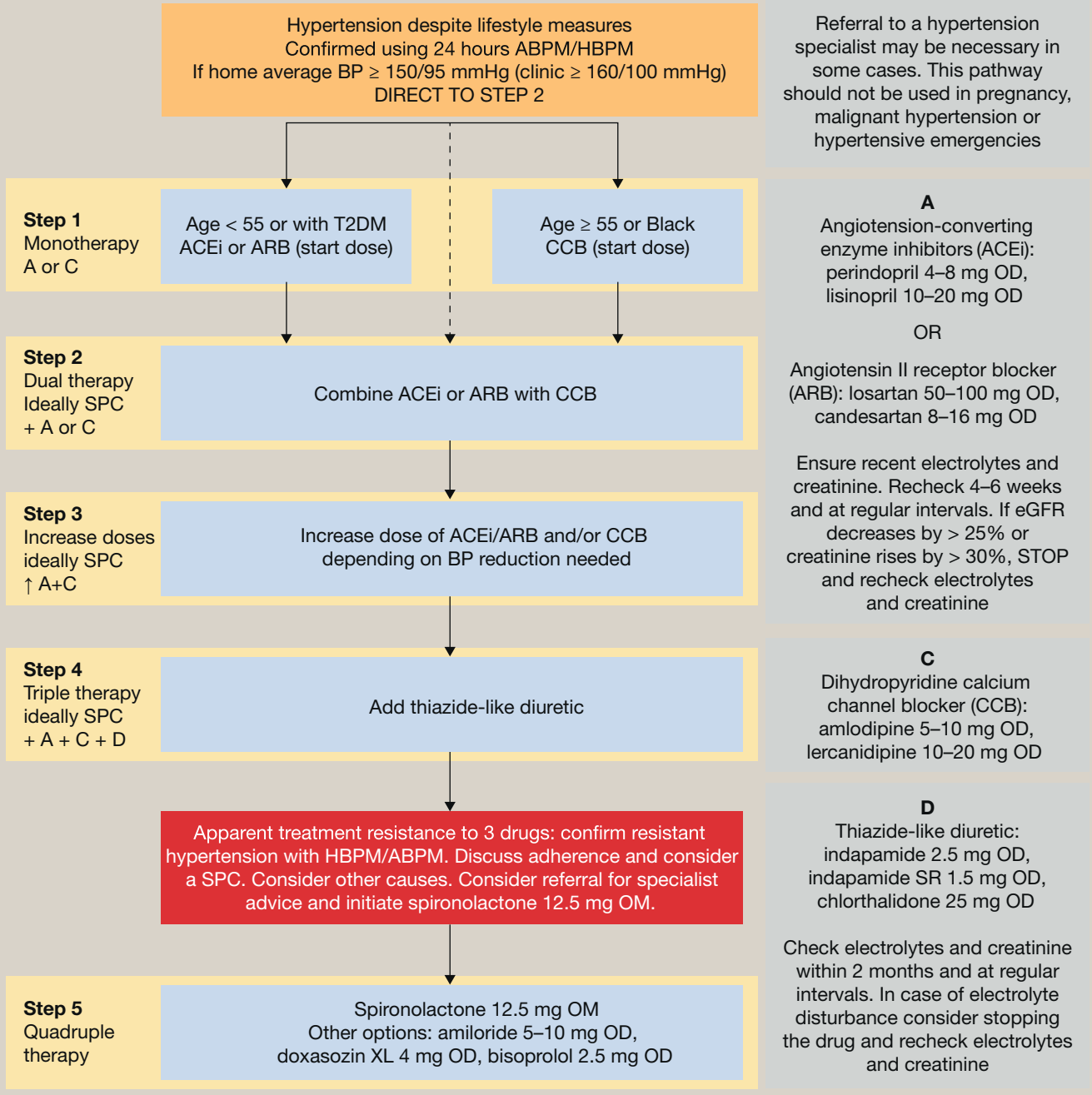
Retinal assessment: retinal microvascular changes offer insight into acute or severe hypertension. Mild changes such as arteriolar narrowing have limited specificity, whereas haemorrhages, cotton wool spots and papilloedema indicate significant more microvascular injury requiring BP optimization.

Management

Lifestyle interventions

Lifestyle interventions are a cornerstone in the prevention and management of hypertension and should be recommended for all individuals with elevated BP values or an established diagnosis

Modified version of the expert recommendations for hypertension management



OD, once daily; OM, every morning; SPC, single-pill combinations; T2DM, type 2 diabetes mellitus.
Adapted from Lewis P, George J, Kapil V, et al. Adult hypertension referral pathway and therapeutic management: British and Irish Hypertension Society position statement. *J Hum Hypertens* 2024; **38**: 3–7.

Figure 3

of hypertension; regular reinforcement should be given as part of continuing care.

Evidence consistently demonstrates that behavioural modification can significantly lower BP and reduce overall CV risk. Key interventions include sodium restriction, weight management (as even modest weight loss in overweight or obese individuals produces substantial BP reductions) and increased physical activity with both aerobic and isometric exercise. Dietary modification, limiting alcohol consumption and smoking cessation, are also crucial.

Importantly, lifestyle interventions are not only effective as stand-alone strategies in individuals with borderline elevated BP values or recently diagnosed with hypertension, but also enhance the efficacy of pharmacological treatment.

Pharmacotherapy

Despite adherence to lifestyle interventions, many individuals still require pharmacological therapy to achieve adequate BP control. The cornerstone of antihypertensive pharmacotherapy consists of drug classes with the strongest evidence for reducing CV morbidity and mortality: angiotensin-converting enzyme inhibitors (ACEi), angiotensin receptor blockers (ARB), calcium channel blockers (CCB) and thiazide or thiazide-like diuretics.

Treatment strategies and BP targets vary across the world. In the UK, therapy is generally initiated with monotherapy (typically an ACEi/ARB or CCB) guided by patient age and ethnicity (a modified version of the BIHS algorithm is shown in Figure 3). By contrast, European[4] and American[5] guidelines often recommend combination therapy from the outset.

In the UK, the National Institute for Health and Care Excellence (NICE) recommends a target of <140/90 mmHg for clinic readings and <135/85 mmHg for out-of-office measurements in most patients. In contrast, European and American guidelines often recommend lower targets and more intensive treatment strategies.

Over the last 15 years, several large outcome trials comparing intensive BP targets (e.g. systolic BP <120 mmHg) versus less intensive targets (e.g. systolic BP <140 mmHg) in individuals with and without pre-existing CV disease or diabetes consistently demonstrate that aiming for lower values results in a greater reduction in major adverse CV events without clear evidence of increasing all-cause mortality, and with no significant difference in serious adverse events.

Based on the available evidence, a very recent BIHS expert consensus recommends a uniform on-treatment BP target of <130/80 mmHg, or as low as reasonably achievable without causing unacceptable adverse effects (see Faconti L, Tantirige N, Poulter NR et al. in Further reading).

Resistant hypertension (RH)

RH is defined as a clinic BP ≥140/90 mmHg, confirmed by elevated out-of-office readings (ABPM/HBPM ≥135/85 mmHg) despite lifestyle modification and optimal or maximally tolerated doses of at least three antihypertensive drugs (typically including an ACEi/ARB, a CCB and a thiazide-like diuretic).

Diagnosis requires excluding non-adherence and secondary hypertension. If these are not excluded, RH cannot be diagnosed and the condition should be defined as apparent RH. Prevalence estimates vary widely (5–30%), although strict definitions suggest rates closer to 5–10%, reflecting cases of pseudo-resistance.

Common causes of pseudo-RH include inaccurate BP measurement, incorrect cuff size, faulty devices, the white-coat effect, non-adherence (affecting up to 50% of individuals with apparent RH), concomitant medications, over-the-counter products and recreational substances. Once pseudo-resistance has been excluded, clinicians should evaluate for secondary hypertension before confirming RH and proceeding with targeted management.

Lifestyle interventions remain foundational in RH management. In particular, obesity is present in over half of individuals with RH. Weight reduction through diet, exercise or pharmacotherapy (e.g. glucagon-like peptide (GLP-1) or GLP-1/glucose-dependent insulinotropic polypeptide (GIP) agonists) can help lower BP, although further evidence regarding the role of anti-obesity drugs is needed.

Pharmacological management in resistant hypertension follows a stepwise approach. After optimization of triple therapy (usually ACEi/ARB + CCB + thiazide diuretic) fourth-line therapy typically involves adding spironolactone (12.5–25 mg, titrated as tolerated), with monitoring of renal function and potassium. Additional agents, including α-adrenoceptor blockers, β-adrenoceptor blockers, centrally acting drugs or vasodilators, can be added based on co-morbidity and tolerability.

Referral to a hypertension specialist is recommended when BP remains uncontrolled after the use of 3–4 agents, to allow a detailed assessment and consideration of advanced therapies.

Renal denervation (RDN) is a device-based treatment available in clinical practice. Early open-label trials showed substantial BP reductions, but later blinded, rigorously designed studies demonstrated more modest overall effects (~5 mmHg). Importantly, responses vary among treated individuals and evidence comparing devices remains limited. Current NICE guidance and BIHS expert consensus support considering RDN only when all other therapies have been exhausted, with full patient understanding of its limitations. Research is ongoing. ◆

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FURTHER READING

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TEST YOURSELF

To test your knowledge based on the article you have just read, please complete the questions below. The answers can be found at the end of the issue or online here.

Question 1

A 52-year-old man was reviewed in the hypertension clinic after persistently elevated clinic blood pressure readings at his general practitioner. He was of African ancestry, and had a 10-year history of hypertension and type 2 diabetes. He was taking an angiotensin receptor blocker, a calcium channel blocker and a thiazide-like diuretic, all at maximum dose. His home blood pressure readings over the previous week had averaged 158/92 mmHg.
On clinical examination, no oedema was noted and heart sounds were normal.

Investigations

- Serum sodium 142 mmol/litre (137–144)
- Serum potassium 3.8 mmol/litre (3.5–4.9)
- Serum creatinine 100 micromol/litre (60–110)

What is the correct definition of this situation?

A. White-coat hypertension
B. Secondary hypertension related to advanced stage chronic kidney disease
C. Masked hypertension
D. Apparent resistant hypertension
E. Resistant hypertension

Question 2

A 34-year-old woman presented after recording home blood pressure readings averaging 168/102 mmHg. She had no medical history and was not taking any medication. She did not smoke and consumed minimal alcohol.
On clinical examination, body mass index was shown to be 24 kg/m^2 and there were no abdominal bruits.

Investigations

- Serum sodium 148 mmol/litre (137–144)
- Serum potassium 3.1 mmol/litre (3.5–4.9)
- Serum creatinine 100 micromol/litre (60–110)

What is the most appropriate next step in her investigation?

A. Overnight dexamethasone suppression test
B. Echocardiography
C. Plasma aldosterone:renin ratio
D. Renal angiography
E. Thyroid function tests

Question 3

A 58-year-old woman was reviewed because of confirmed resistant hypertension on ambulatory monitoring despite treatment with an angiotensin-converting enzyme inhibitor, a calcium channel blocker and a thiazide-like diuretic, all at maximum dose. Secondary causes had been excluded. She was taking her medications, was following dietary advice and had normal renal function and serum potassium.

Investigation

- Echocardiography showed left ventricular hypertrophy

What is the most appropriate next step in her management?

A. Add a β-adrenoceptor blocker
B. Add spironolactone
C. Increase dietary sodium
D. Refer for renal denervation
E. Replace the thiazide-like diuretic with a loop diuretic

