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ESC GUIDELINES

2024 ESC Guidelines for the management of elevated blood pressure and hypertension

Developed by the task force on the management of elevated blood pressure and hypertension of the European Society of Cardiology (ESC) and *endorsed by the European Society of Endocrinology (ESE) and the European Stroke Organisation (ESO)*

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Click here to access the corresponding ESC CardioMed chapters.

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Abbreviations and acronyms

ABI	Ankle–brachial index
ABPM	Ambulatory blood pressure monitoring
ACCORD	Action to Control Cardiovascular Risk in Diabetes
ACE	Angiotensin-converting enzyme
ACR	Albumin:creatinine ratio
AF	Atrial fibrillation
AHI	Apnoea–hypopnoea index
ALARA	As low as reasonably achievable
ALLHAT	Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack
AOBP	Automated office blood pressure (measurement)
ARB	Angiotensin receptor blocker
ARNi	Angiotensin receptor-neprilysin inhibitor
ARR	Aldosterone-to-renin ratio
ASCVD	Atherosclerotic cardiovascular disease
BMI	Body mass index
BP	Blood pressure
BSA	Body surface area
CAC	Coronary artery calcium
CAD	Coronary artery disease
CCB	Calcium channel blocker
CHAP	Chronic Hypertension and Pregnancy
CI	Confidence interval
CKD	Chronic kidney disease
CKD-EPI	Chronic Kidney Disease Epidemiology Collaboration
COVID-19	Coronavirus disease 2019
CPAP	Continuous positive airway pressure
CPG	Clinical Practice Guidelines
CT	Computed tomography
CVD	Cardiovascular disease
DASH	Dietary Approaches to Stop Hypertension
DBP	Diastolic blood pressure
DECIDE-Salt	Diet, ExerCIse and carDiovascular hEalth–Salt
EACTS	European Association for Cardio-Thoracic Surgery
ECG	Electrocardiogram
eGFR	Estimated glomerular filtration rate
EPIC	European Prospective Investigation into Cancer and Nutrition
ESC	European Society of Cardiology
ESH	European Society of Hypertension
ESPRIT	Effects of intensive Systolic blood Pressure lowering treatment in reducing RIsk of vascular evenTs
FMD-RVH	Fibromuscular dysplasia-induced renovascular hypertension
GFR	Glomerular filtration rate
GLP-1	Glucagon-like peptide-1
GP	General practitioner
HbA1c	Glycated haemoglobin
HBPM	Home blood pressure monitoring
HDL	High-density lipoprotein
HFpEF	Heart failure with preserved ejection fraction
HF(m)rEF	Heart failure with (mildly) reduced ejection fraction
HIV	Human immunodeficiency virus
HMOD	Hypertension-mediated organ damage
i.m.	Intramuscular
i.v.	Intravenous
KDIGO	Kidney Disease: Improving Global Outcomes
LA	Left atrial
LDL	Low-density lipoprotein
LV	Left ventricular
LVH	Left ventricular hypertrophy
MRA	Mineralocorticoid receptor antagonist
MRI	Magnetic resonance imaging
NNT	Number needed to treat
NT-proBNP	N-terminal pro-brain natriuretic peptide
OSAS	Obstructive sleep apnoea syndrome
PPGL	Phaeochromocytoma/paraganglioma
PREOP-ACEI	Prospective Randomized Evaluation of Preoperative Angiotensin-Converting Enzyme Inhibition
PREMs	Patient-Reported Experience Measures
PROMS	Patient-Reported Outcome Measures
PTRA	Percutaneous transluminal renal angioplasty
PWV	Pulse wave velocity
RAAS	Renin-angiotensin-aldosterone system
RADIANCE-HTN	A Study of the Recor Medical Paradise System in Clinical Hypertension
RAS	Renin–angiotensin system
RCT	Randomized controlled trial
RVH	Renovascular hypertension
RWT	Relative wall thickness
SBP	Systolic blood pressure
SCORE2	Systematic COronary Risk Evaluation 2
SCORE2-OP	Systematic COronary Risk Evaluation 2–Older Persons
SGLT2	Sodium–glucose co-transporter 2
SNP	Single-nucleotide polymorphism
SNS	Sympathetic nervous system
SPC	Single-pill combination
SPRINT	Systolic Blood Pressure Intervention Trial
SSaSS	Salt Substitute and Stroke Study
STEP	Strategy of Blood Pressure Intervention in Elderly Hypertensive Patients
STEP-1	Semaglutide Treatment Effect in People with Obesity

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TIA	Transient ischaemic attack
TRIUMPH	Treating Resistant Hypertension Using Lifestyle Modification to Promote Health
TSH	Thyroid-stimulating hormone
WHO	World Health Organization
WML	White matter lesion

1. Preamble

Guidelines evaluate and summarize available evidence with the aim of assisting health professionals in proposing the best diagnostic or therapeutic approach for an individual patient with a given condition. Guidelines are intended for use by health professionals and the European Society of Cardiology (ESC) makes its guidelines freely available.

ESC Guidelines do not override the individual responsibility of health professionals to make appropriate and accurate decisions in consideration of each patient's health condition and in consultation with that patient or the patient's caregiver where appropriate and/or necessary. It is also the health professional's responsibility to verify the rules and regulations applicable in each country to drugs and devices at the time of prescription and to respect the ethical rules of their profession.

ESC Guidelines represent the official position of the ESC on a given topic and are regularly updated when warranted by new evidence. ESC Policies and Procedures for formulating and issuing ESC Guidelines can be found on the ESC website (https://www.escardio.org/Guidelines/Clinical-Practice-Guidelines/Guidelines-development/Writing-ESC-Guidelines). This guideline version updates and replaces the previous version from 2018.

The Members of this task force were selected by the ESC to include professionals involved in the medical care of patients with this pathology, as well as patient representatives and methodologists. The selection procedure included an open call for authors and aimed to include members from across the whole of the ESC region and from relevant ESC Subspecialty Communities. Consideration was given to diversity and inclusion, notably with respect to gender and country of origin. The task force performed a critical review and evaluation of the published literature on diagnostic and therapeutic approaches including assessment of the risk-benefit ratio. The strength of every recommendation and the level of evidence supporting them were weighed and scored according to predefined scales as outlined in *Tables 1* and *2* below. Patient-Reported Outcome Measures (PROMs) and Patient-Reported Experience Measures (PREMs) were also evaluated as the basis for recommendations and/or discussion in these guidelines. The task force followed ESC voting procedures and all approved recommendations were subject to a vote and achieved at least 75% agreement among voting members. Members of the task force with declared interests on specific topics were asked to abstain from voting on related recommendations.

The experts of the writing and reviewing panels provided declaration of interest forms for all relationships that might be perceived as real or potential sources of conflicts of interest. Their declarations of interest were reviewed according to the ESC declaration of interest rules, which can be found on the ESC website (http://www.escardio.org/guidelines) and have been compiled in a report published in a supplementary document with the guidelines. Funding for the development of ESC Guidelines is derived entirely from the ESC with no involvement of the healthcare industry.

Table 1 Classes of recommendations

Classes of recommendations	Definition	Wording to use
Class I	Evidence and/or general agreement that a given treatment or procedure is beneficial, useful, effective.	Is recommended or is indicated
Class II	Conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of the given treatment or procedure.	
Class IIa	Weight of evidence/opinion is in favour of usefulness/efficacy.	Should be considered
Class IIb	Usefulness/efficacy is less well established by evidence/opinion.	May be considered
Class III	Evidence or general agreement that the given treatment or procedure is not useful/effective, and in some cases may be harmful.	Is not recommended

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Table 2 **Levels of evidence**

Level of evidence A	Data derived from multiple randomized clinical trials or meta-analyses.
Level of evidence B	Data derived from a single randomized clinical trial or large non-randomized studies.
Level of evidence C	Consensus of opinion of the experts and/or small studies, retrospective studies, registries.

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The ESC Clinical Practice Guidelines (CPG) Committee supervises and co-ordinates the preparation of new guidelines and is responsible for the approval process. In addition to review by the CPG Committee, ESC Guidelines undergo multiple rounds of double-blind peer review by external experts, including members from across the whole of the ESC region, all National Cardiac Societies of the ESC and from relevant ESC Subspecialty Communities. After appropriate revisions, the guidelines are signed off by all the experts in the task force. The finalized document is signed off by the CPG Committee for publication in the *European Heart Journal*.

ESC Guidelines are based on analyses of published evidence, chiefly on clinical trials and meta-analyses of trials, but potentially including other types of studies. Evidence tables summarizing key information from relevant studies are generated early in the guideline development process to facilitate the formulation of recommendations, to enhance comprehension of recommendations after publication, and reinforce transparency in the guidelines development process. The tables are published in their own section of ESC Guidelines and reference specific recommendation tables.

Off-label use of medication may be presented in these guidelines if a sufficient level of evidence shows that it can be considered medically appropriate for a given condition. However, the final decisions concerning an individual patient must be made by the responsible health professional giving special consideration to:

- The specific situation of the patient. Unless otherwise provided for by national regulations, off-label use of medication should be limited to situations where it is in the patient's interest with regard to the quality, safety, and efficacy of care, and only after the patient has been informed and has provided consent;
- Country-specific health regulations, indications by governmental drug regulatory agencies, and the ethical rules to which health professionals are subject, where applicable.

2. Introduction

This 2024 document updates the 2018 ESC/European Society of Hypertension (ESH) Guidelines on the management of arterial hypertension.[1] While the current document builds on prior guidelines, it also incorporates important updates and new recommendations based on current evidence. For example:

(1) The title has changed from 'Guidelines on the management of arterial hypertension' to 'Guidelines on the management of elevated blood pressure and hypertension'. This is based on evidence that the risk for cardiovascular disease (CVD) attributable to blood pressure (BP) is on a continuous exposure scale, not a binary scale of normotension vs. hypertension.[2,3] Updated evidence also increasingly demonstrates the benefit on CVD outcomes of BP-lowering medications among persons with high CVD risk and BP levels that are elevated but that do not meet traditional thresholds used to define hypertension. The term 'arterial' is removed from the title of the 2024 Guidelines, as arterial hypertension can also occur in the pulmonary arteries, which is not a focus here.

(2) The 2024 Guidelines continue to define hypertension as office systolic BP of ≥140 mmHg or diastolic BP of ≥90 mmHg. However, a new BP category called 'Elevated BP' is introduced. Elevated BP is defined as an office systolic BP of 120–139 mmHg or diastolic BP of 70–89 mmHg.

(3) A major, evidence-based change in the 2024 Guidelines is the recommendation to pursue a target systolic BP of 120–129 mmHg among adults receiving BP-lowering medications. There are several important caveats to this recommendation, including: (i) the requirement that treatment to this BP target is well tolerated by the patient, (ii) the fact that more lenient BP targets can be considered in persons with symptomatic orthostatic hypotension, those aged 85 years or over, or those with moderate-to-severe frailty

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or limited life expectancy, and (iii) a strong emphasis on out-of-office BP measurement to confirm the systolic BP target of 120–129 mmHg is achieved. For those selected individual cases where a target systolic BP of 120–129 mmHg is not pursued, either due to intolerance or the existence of conditions that favour a more lenient BP target, we recommend targeting a BP that is as low as reasonably achievable. Personalized clinical decision-making and shared decisions with the patient are also emphasized.

(4) Another important change in the 2024 Guidelines compared with earlier versions is the increased focus on evidence related to fatal and non-fatal CVD outcomes rather than surrogate outcomes such as BP lowering alone. Except for lifestyle interventions and low-risk non-pharmacological interventions aimed at implementation or care delivery, the current guidelines require that, for a Class I recommendation to be made for a drug or procedural intervention, the evidence must show benefit on CVD outcomes and not only BP lowering.

(5) The task force comprised of a balanced representation of males and females.

(6) The present guidelines consider sex and gender as an integral component throughout the document, rather than in a separate section at the end. In this document, sex is the biological condition of being female or male from conception, based on genes, and gender is the socio-cultural dimension of being a woman or a man in a given society, based on gender roles, gender norms, gender identity, and gender relations valid in the respective society at a given timepoint.[4,5]

(7) The 2024 Guidelines are written to make them more 'user friendly'. Input from general practitioners (GPs) was obtained in this regard, and one task force member is a GP. Given the ageing population in Europe, there was also a focus on tailoring treatment with respect to frailty and into older age, which is addressed in multiple sections. Moreover, patient input and their lived experiences are considered throughout. We also now include evidence tables in the Supplementary section to provide improved transparency regarding our recommendations. As appropriate, readers who wish to seek additional details and information are referred to the Supplementary data online and to the ESC CardioMed.[6]

(8) The task force recognized that a major challenge in guideline usage is poor implementation. This likely contributes to suboptimal control of hypertension.[7–9] To address this, a dedicated section on implementation is included in the Supplementary data online. Moreover, through a new initiative, we include information from national societies following a survey on guideline implementation completed during the national society peer review of the guidelines document. It is hoped this information may help inform national societies about potential barriers to implementation.

2.1. What is new

These 2024 Guidelines contain a number of new and revised recommendations, which are summarized in *Tables 3* and *4*, respectively.

Table 3 New recommendations

Recommendations	Class[a]	Level[b]
5. Measuring blood pressure		
It is recommended to measure BP using a validated and calibrated device, to enforce the correct measurement technique, and to apply a consistent approach to BP measurement for each patient.	I	B
Out-of-office BP measurement is recommended for diagnostic purposes, particularly because it can detect both white-coat hypertension and masked hypertension. Where out-of-office measurements are not logistically and/or economically feasible, then it is recommended that the diagnosis be confirmed with a repeat office BP measurement using the correct standardized measurement technique.	I	B
Most automated oscillometric monitors have not been validated for BP measurement in AF; BP measurement should be considered using a manual auscultatory method in these circumstances, where possible.	IIa	C
An assessment for orthostatic hypotension (≥20 systolic BP and/or ≥10 diastolic BP mmHg drop at 1 and/or 3 min after standing) should be considered at least at the initial diagnosis of elevated BP or hypertension and thereafter if suggestive symptoms arise. This should be performed after the patient is first lying or sitting for 5 min.	IIa	C
6. Definition and classification of elevated blood pressure and hypertension, and cardiovascular disease risk assessment		
It is recommended to use a risk-based approach in the treatment of elevated BP, and individuals with moderate or severe CKD, established CVD, HMOD, diabetes mellitus, or familial hypercholesterolaemia are considered at increased risk for CVD events.	I	B
It is recommended that, irrespective of age, individuals with elevated BP and a SCORE2 or SCORE2-OP CVD risk of ≥10% be considered at increased risk for CVD for the purposes of risk-based management of their elevated BP.	I	B
SCORE2-Diabetes should be considered to estimate CVD risk among type 2 diabetes mellitus patients with elevated BP, particularly if they are <60 years of age.	IIa	B
History of pregnancy complications (gestational diabetes, gestational hypertension, pre-term delivery, pre-eclampsia, one or more stillbirths, and recurrent miscarriage) are sex-specific risk modifiers that should be considered to up-classify individuals with elevated BP and borderline increased 10-year CVD risk (5% to <10% risk).	IIa	B
High-risk ethnicity (e.g. South Asian), family history of premature onset atherosclerotic CVD, socio-economic deprivation, auto-immune inflammatory disorders, HIV, and severe mental illness are risk modifiers shared by both sexes that should be considered to up-classify individuals with elevated BP and borderline increased 10-year CVD risk (5% to <10% risk).	IIa	B
After assessing 10-year predicted CVD risk and non-traditional CVD risk modifiers, if a risk-based BP-lowering treatment decision remains uncertain for individuals with elevated BP, measuring CAC score, carotid or femoral plaque using ultrasound, high-sensitivity cardiac troponin or B-type natriuretic peptide biomarkers, or arterial stiffness using pulse wave velocity, may be considered to improve risk stratification among patients with borderline increased 10-year CVD risk (5% to <10% risk) after shared decision-making and considering costs.	IIb	B

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7. Diagnosing hypertension and investigating underlying causes		
Opportunistic screening for elevated BP and hypertension should be considered: • At least every 3 years for adults aged <40 years. • At least annually for adults aged ≥40 years.	IIa	C
In individuals with elevated BP who do not currently meet risk thresholds for BP-lowering treatment, a repeat BP measurement and risk assessment within 1 year should be considered.	IIa	C
Other forms of screening for hypertension (i.e. systematic screening, self-screening, and non-physician screening) may be considered, depending on their feasibility in different countries and healthcare systems.	IIb	B
In individuals with increased CVD risk where their screening office BP is 120–139/70–89 mmHg, it is recommended to measure BP out of office, using ABPM and/or HBPM or, if not logistically feasible, make repeated office BP measurements on more than one visit.	I	B
Objective evaluation of adherence (either directly observed treatment or detecting prescribed drugs in blood or urine samples) should be considered in the clinical work-up of patients with apparent resistant hypertension, if resources allow.	IIa	B
If moderate-to-severe CKD is diagnosed, it is recommended to repeat measurements of serum creatinine, eGFR, and urine ACR at least annually.	I	C
Coronary artery calcium scoring may be considered in patients with elevated BP or hypertension when it is likely to change patient management.	IIb	B
Patients with resistant hypertension should be considered for referral to clinical centres with expertise in hypertension management for further testing.	IIa	B
It is recommended that patients with hypertension presenting with suggestive signs, symptoms, or medical history of secondary hypertension are appropriately screened for secondary hypertension.	I	B
Screening for primary aldosteronism by renin and aldosterone measurements should be considered in all adults with confirmed hypertension (BP ≥ 140/90 mmHg).	IIa	B
8. Preventing and treating elevated blood pressure		
Opportunistic screening with office BP measurements to monitor development of BP during late childhood and adolescence, especially if one or both parents have hypertension, should be considered to better predict development of adult hypertension and associated CVD risk.	IIa	B
It is recommended to restrict free sugar consumption, in particular sugar-sweetened beverages, to a maximum of 10% of energy intake. It is also recommended to discourage consumption of sugar-sweetened beverages, such as soft drinks and fruit juices, starting at young age.	I	B
In patients with hypertension without moderate-to-advanced CKD and with high daily sodium intake, an increase of potassium intake by 0.5–1.0 g/day—for example through sodium substitution with potassium-enriched salt (comprising 75% sodium chloride and 25% potassium chloride) or through diets rich in fruits and vegetables—should be considered.	IIa	A
In patients with CKD or taking potassium-sparing medication, such as some diuretics, ACE inhibitors, ARBs, or spironolactone, monitoring serum levels of potassium should be considered if dietary potassium is being increased.	IIa	C
It is recommended to take medications at the most convenient time of day for the patient, to establish a habitual pattern of medication taking to improve adherence.	I	B
In adults with elevated BP and low/medium CVD risk (<10% over 10 years), BP lowering with lifestyle measures is recommended and can reduce the risk of CVD.	I	B
In adults with elevated BP and sufficiently high CVD risk, after 3 months of lifestyle intervention, BP lowering with pharmacological treatment is recommended for those with confirmed BP ≥130/80 mmHg to reduce CVD risk.	I	A
It is recommended that in hypertensive patients with confirmed BP ≥140/90 mmHg, irrespective of CVD risk, lifestyle measures and pharmacological BP-lowering treatment is initiated promptly to reduce CVD risk.	I	A
It is recommended to maintain BP-lowering drug treatment lifelong, even beyond the age of 85 years, if well tolerated.	I	A
Because the benefit in reducing CVD outcomes is uncertain in these settings, and noting that close monitoring of treatment tolerance is advised, BP-lowering treatment should only be considered from ≥140/90 mmHg (office) among persons meeting the following criteria: • pre-treatment symptomatic orthostatic hypotension; • age ≥85 years; • clinically significant moderate-to-severe frailty; • and/or limited predicted lifespan (<3 years).	IIa	B
In cases where BP-lowering treatment is poorly tolerated and achieving a target systolic of 120–129 mmHg is not possible, it is recommended to target a systolic BP level that is 'as low as reasonably achievable' (ALARA principle).	I	A
Once BP is controlled and stable under BP-lowering therapy, at least a yearly follow-up for BP and other CVD risk factors should be considered.	IIa	C

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9. Managing specific patient groups or circumstances		
Young adults		
Comprehensive screening for the main causes of secondary hypertension is recommended in adults diagnosed with hypertension before the age of 40 years, except for obese young adults where it is recommended to start with an obstructive sleep apnoea evaluation.	I	B
Since SCORE2 has not been validated for individuals <40 years, screening for HMOD may be considered in such young individuals with elevated BP without other increased CVD risk conditions to identify additional individuals for possible medical treatment.	IIb	B
Hypertension in pregnancy		
In consultation with an obstetrician, low- to moderate-intensity exercise is recommended in all pregnant women without contraindications to reduce the risk of gestational hypertension and pre-eclampsia.	I	B
HBPM and ABPM should be considered to exclude white-coat and masked hypertension, which are more common in pregnancy.	IIa	C
Older and frail patients		
It is recommended that treatment of elevated BP and hypertension among older patients aged <85 years who are not moderately to severely frail follows the same guidelines as for younger people, provided BP-lowering treatment is well tolerated.	I	A
When initiating BP-lowering treatment for patients aged ≥85 years, and/or with moderate-to-severe frailty (at any age), long-acting dihydropyridine CCBs or RAS inhibitors should be considered, followed, if necessary, by a low-dose diuretic if tolerated, but preferably not a beta-blocker (unless compelling indications exist) or an alpha-blocker.	IIa	B
As the safety and efficacy of BP treatment is less certain in individuals with moderate or severe frailty, clinicians should consider screening older adults for frailty using validated clinical tests; frail patients' health priorities and a shared-decision approach should be considered when deciding on BP treatments and targets.	IIa	C
If BP drops with progressing frailty, deprescription of BP-lowering medications (and other drugs that can reduce BP, such as sedatives and prostate-specific alpha-blockers) may be considered.	IIb	C
Hypertension and orthostatic hypotension		
Before starting or intensifying BP-lowering medication, it is recommended to test for orthostatic hypotension, by first having the patient sit or lie for 5 min and then measuring BP 1 and/or 3 min after standing.	I	B
It is recommended to pursue non-pharmacological approaches as the first-line treatment of orthostatic hypotension among persons with supine hypertension. For such patients, it is also recommended to switch BP-lowering medications that worsen orthostatic hypotension to an alternative BP-lowering therapy and not to simply de-intensify therapy.	I	A
Chronic kidney disease		
In hypertensive patients with CKD and eGFR >20 mL/min/1.73 m^2, SGLT2 inhibitors are recommended to improve outcomes in the context of their modest BP-lowering properties.	I	A
Other conditions		
BP-lowering drug treatment is recommended for people with pre-diabetes or obesity when confirmed office BP is ≥140/90 mmHg or when office BP is 130–139/80–89 mmHg and the patient is at predicted 10-year risk of CVD ≥10% or with high-risk conditions, despite a maximum of 3 months of lifestyle therapy.	I	A
In patients with a history of aortic valve stenosis and/or regurgitation who require BP-lowering treatment, RAS blockers should be considered as part of that treatment.	IIa	C
In patients with a history of moderate-to-severe mitral valve regurgitation who require BP-lowering treatment, RAS blockers should be considered as part of that treatment.	IIa	C
Renovascular hypertension		
Renal artery angioplasty without stenting should be considered for patients with hypertension and haemodynamically significant renal artery stenosis due to fibromuscular dysplasia.	IIa	C
Renal artery angioplasty and stenting may be considered in patients with haemodynamically significant, atherosclerotic, renal artery stenosis (stenosis of 70%–99%, or 50%–69% with post-stenotic dilatation and/or significant trans-stenotic pressure gradient) with: • Recurrent heart failure, unstable angina, or sudden-onset flash pulmonary oedema despite maximally tolerated medical therapy; • Resistant hypertension; • Hypertension with unexplained unilaterally small kidney or CKD; • Bilateral renal artery stenosis or unilateral renal artery stenosis in a solitary viable kidney.	IIb	C
Renal artery angioplasty is not recommended in patients without confirmed haemodynamically significant renal artery stenosis.	III	A
10. Acute and short-term lowering of blood pressure		
In patients with intracerebral haemorrhage presenting with systolic BP ≥220 mmHg, acute reduction in systolic BP >70 mmHg from initial levels within 1 h of commencing treatment is not recommended.	III	B

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11. Patient-centred care in hypertension		
An informed discussion about CVD risk and treatment benefits tailored to the needs of a patient is recommended as part of hypertension management.	I	C
Motivational interviewing should be considered for patients with hypertension at hospitals and community health centres to assist patients in controlling their BP and to enhance treatment adherence.	IIa	B
Physician–patient web communications are an effective tool that should be considered in primary care, including reporting on home BP readings.	IIa	C
Home BP measurement for managing hypertension by using self-monitored BP is recommended to achieve better BP control.	I	B
Self-measurement, when properly performed, is recommended due to positive effects on the acceptance of a diagnosis of hypertension, patient empowerment, and adherence to treatment.	I	C
Enhanced self-monitoring of BP using a device paired with a connected smartphone application may be considered, though evidence to date suggests that this may be no more effective than standard self-monitoring.	IIb	B
Multidisciplinary approaches in the management of patients with elevated BP and hypertension, including appropriate and safe task-shifting away from physicians are recommended to improve BP control.	I	A

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ABPM, ambulatory blood pressure monitoring; ACE, angiotensin-converting enzyme; ACR, albumin-to-creatinine ratio (urine); AF, atrial fibrillation; ALARA, as low as reasonably achievable; ARB, angiotensin receptor blocker; BP, blood pressure; CAC, coronary artery calcium; CCB, calcium channel blocker; CKD, chronic kidney disease; CVD, cardiovascular disease; eGFR, estimated glomerular filtration rate; HBPM, home blood pressure monitoring; HFpEF, heart failure with preserved ejection fraction; HIV, human immunodeficiency virus; HMOD, hypertension-mediated organ damage; MRA, mineralocorticoid receptor antagonist; RAS, renin–angiotensin system; SCORE2, Systematic COronary Risk Evaluation 2; SCORE2-OP, Systematic COronary Risk Evaluation 2–Older Persons; SGLT2, sodium–glucose co-transporter 2.
[a]Class of recommendation.
[b]Level of evidence.

Table 4 Revised recommendations

Recommendations in 2018 version	Class[a]	Level[b]	Recommendations in 2024 version	Class[a]	Level[b]
6. Definition and classification of elevated blood pressure and hypertension					
It is recommended that BP be classified as optimal, normal, high–normal, or grades 1–3 hypertension, according to office BP.	I	C	It is recommended that BP be categorized as non-elevated BP, elevated BP, and hypertension to aid treatment decisions.	I	B
CV risk assessment with the SCORE system is recommended for hypertensive patients who are not already at high or very high risk due to established CVD, renal disease, or diabetes, a markedly elevated single risk factor (e.g. cholesterol), or hypertensive LVH.	I	B	SCORE2 is recommended for assessing 10-year risk of fatal and non-fatal CVD among individuals aged 40–69 years with elevated BP who are not already considered at increased risk due to moderate or severe CKD, established CVD, HMOD, diabetes mellitus, or familial hypercholesterolaemia.	I	B
			SCORE2-OP is recommended for assessing the 10-year risk of fatal and non-fatal CVD among individuals aged ≥70 years with elevated BP who are not already considered at increased risk due to moderate or severe CKD, established CVD, HMOD, diabetes mellitus, or familial hypercholesterolaemia.	I	B
7. Diagnosing hypertension and investigating underlying causes					
It is recommended that the diagnosis of hypertension should be based on: • Repeated office BP measurements on more than one visit, except when hypertension is severe (e.g. grade 3 and especially in high-risk patients). At each visit, three BP measurements should be recorded, 1–2 min apart, and additional measurements should be performed if the first two readings differ by >10 mmHg. The patient's BP is the average of the last two BP readings. Or • Out-of-office BP measurement with ABPM and/or HBPM, provided that these measurements are logistically and economically feasible.	I	C	Where screening office BP is 140–159/90–99 mmHg, it is recommended that the diagnosis of hypertension should be based on out-of-office BP measurement with ABPM and/or HBPM. If these measurements are not logistically or economically feasible, then diagnosis can be made on repeated office BP measurements on more than one visit.	I	B
			Where screening office BP is ≥160/100 mmHg: • It is recommended that BP 160–179/100–109 mmHg be confirmed as soon as possible (e.g. within 1 month) preferably by either home or ambulatory BP measurements. • It is recommended when BP ≥180/110 mmHg that hypertensive emergency be excluded.	I	C

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Echocardiography is recommended in hypertensive patients when there are ECG abnormalities or signs or symptoms of LV dysfunction.	I	B	Echocardiography is recommended in patients with hypertension and ECG abnormalities, or signs or symptoms of cardiac disease.	I	B
Echocardiography may be considered when the detection of LVH may influence treatment decisions.	IIb	B	Echocardiography may be considered in patients with elevated BP, particularly when it is likely to change patient management.	IIb	B
Ultrasound examination of the carotid arteries may be considered for the detection of asymptomatic atherosclerotic plaques or carotid stenosis in patients with documented vascular disease elsewhere.	IIb	B	Ultrasound examination of the carotid or femoral arteries for detecting plaque may be considered in patients with elevated BP or hypertension when it is likely to change patient management.	IIb	B
Measurement of PWV may be considered for measuring arterial stiffness.	IIb	B	Measurement of PWV may be considered in patients with elevated BP or hypertension when it is likely to change patient management.	IIb	B
8. Preventing and treating elevated blood pressure					
Regular aerobic exercise (e.g. at least 30 min of moderate dynamic exercise on 5–7 days/week) is recommended.	I	A	Moderate intensity aerobic exercise of ≥150 min/week (≥30 min, 5–7 days/week) or alternatively 75 min of vigorous intensity aerobic exercise per week over 3 days are recommended and should be complemented with low- or moderate-intensity dynamic or isometric resistance training (2–3 times/week) to reduce BP and CVD risk.	I	A
Body-weight control is indicated to avoid obesity (BMI >30 kg/m^2 or waist circumference >102 cm in men and >88 cm in women), as is aiming at healthy BMI (about 20–25 kg/m^2) and waist circumference values (<94 cm in men and <80 cm in women) to reduce BP and CV risk.	I	A	It is recommended to aim for a stable and healthy BMI (20–25 kg/m^2) and waist circumference values (<94 cm in men and <80 cm in women) to reduce BP and CVD risk.	I	A
Increased consumption of vegetables, fresh fruits, fish, nuts, and unsaturated fatty acids (olive oil); low consumption of red meat; and consumption of low-fat dairy products are recommended.	I	A	Adopting a healthy and balanced diet, such as the Mediterranean or DASH diets, is recommended to help reduce BP and CVD risk.	I	A
It is recommended to restrict alcohol consumption to: • Less than 14 units/week for men. • Less than 8 units/week for women.	I	A	Men and women are recommended to drink less alcohol than the upper limit, which is about 100 g/week of pure alcohol. How this translates into number of drinks depends on portion size (the standards of which differ per country), but most drinks contain 8–14 g of alcohol per drink. Preferably, it is recommended to avoid alcohol to achieve the best health outcomes.	I	B
Among all anti-hypertensive drugs, ACE inhibitors, ARBs, beta-blockers, CCBs, and diuretics (thiazides and thiazide-like drugs, such as chlorthalidone and indapamide) have demonstrated effective reduction of BP and CV events in RCTs, and thus are indicated as the basis of anti-hypertensive treatment strategies.	I	A	Among all BP-lowering drugs, ACE inhibitors, ARBs, dihydropyridine CCBs, and diuretics (thiazides and thiazide-like drugs such as chlorthalidone and indapamide) have demonstrated the most effective reduction of BP and CVD events, and are therefore recommended as first-line treatments to lower BP.	I	A
It is recommended that if BP is not controlled with a three-drug combination, treatment should be increased by the addition of spironolactone or, if not tolerated, other diuretics such as amiloride or higher doses of other diuretics, a beta-blocker, or an alpha-blocker.	I	B	If BP is not controlled with a three-drug combination and in whom spironolactone is not effective or tolerated, treatment with eplerenone instead of spironolactone, or the addition of a beta-blocker if not already indicated and, next, a centrally acting BP-lowering medication, an alpha-blocker, hydralazine, or a potassium-sparing diuretic should be considered.	IIa	B
8. Preventing and treating elevated blood pressure (blood pressure targets)					
It is recommended that the first objective of treatment should be to lower BP to <140/90 mmHg in all patients and, provided that the treatment is well tolerated, treated BP values should be targeted to 130/80 mmHg or lower in most patients.	I	A	To reduce CVD risk, it is recommended that treated systolic BP values in most adults be targeted to 120–129 mmHg, provided the treatment is well tolerated.	I	A

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A diastolic BP target of <80 mmHg should be considered for all hypertensive patients, independent of the level of risk and comorbidities.	IIa	B	In cases where on-treatment systolic BP is at or below target (120–129 mmHg) but diastolic BP is not at target (≥80 mmHg), intensifying BP-lowering treatment to achieve an on-treatment diastolic BP of 70–79 mmHg may be considered to reduce CVD risk.	IIb	C
In older patients (aged ≥65 years) receiving BP-lowering drugs: • It is recommended that systolic BP should be targeted to a BP range of 130–139 mmHg.	I	A	Because the CVD benefit of an on-treatment systolic BP target of 120–129 mmHg may not generalize to the following specific settings, personalized and more lenient systolic BP targets (e.g. <140 mmHg): should be considered among patients meeting the following criteria: • pre-treatment, symptomatic, orthostatic hypotension; • and/or age ≥85 years.	IIa	C
			Because the CVD benefit of an on-treatment systolic BP target of 120–129 mmHg may not generalize to the following specific settings, personalized and more lenient BP targets (e.g. <140/90 mmHg) may be considered among patients meeting the following criteria: • clinically significant, moderate to severe frailty at any age; • and/or limited predicted lifespan (<3 years).	IIb	C
8. Preventing and treating elevated blood pressure (renal denervation)					
Use of device-based therapies is not recommended for the routine treatment of hypertension, unless in the context of clinical studies and RCTs, until further evidence regarding their safety and efficacy becomes available.	III	B	To reduce BP, and if performed at a medium-to-high volume centre, catheter-based renal denervation may be considered for resistant hypertension patients who have BP that is uncontrolled despite a three BP-lowering drug combination (including a thiazide or thiazide-like diuretic), and who express a preference to undergo renal denervation after a shared risk-benefit discussion and multidisciplinary assessment.	IIb	B
			To reduce BP, and if performed at a medium-to-high volume centre, catheter-based renal denervation may be considered for patients with both increased CVD risk and uncontrolled hypertension on fewer than three drugs, if they express a preference to undergo renal denervation after a shared risk-benefit discussion and multidisciplinary assessment.	IIb	A
			Due to a lack of adequately powered outcomes trials demonstrating its safety and CVD benefits, renal denervation is not recommended as a first-line BP-lowering intervention for hypertension.	III	C
			Renal denervation is not recommended for treating hypertension in patients with moderately to severely impaired renal function (eGFR <40 mL/min/1.73 m^2) or secondary causes of hypertension, until further evidence becomes available.	III	C
9.1. Managing specific patient groups or circumstances					
Hypertension in pregnancy					
In women with gestational hypertension, pre-existing hypertension superimposed by gestational hypertension, or with hypertension and subclinical organ damage or symptoms, initiation of drug treatment is recommended when systolic BP is ≥140 mmHg or diastolic BP ≥90 mmHg.	I	C	In women with gestational hypertension, starting drug treatment is recommended for those with confirmed systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg.	I	B

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In all other cases, initiation of drug treatment is recommended when systolic BP is ≥150 mmHg or diastolic BP is ≥95 mmHg.	I	C	In pregnant women with chronic hypertension, starting drug treatment is recommended for those with confirmed office systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg.	I	B
			In women with chronic and gestational hypertension, it is recommended to lower BP below 140/90 mmHg but not below 80 mmHg for diastolic BP.	I	C
Systolic BP ≥170 mmHg or diastolic BP ≥110 mmHg in a pregnant woman is an emergency, and admission to hospital is recommended.	I	C	Systolic BP ≥160 mmHg or diastolic BP ≥110 mmHg in pregnancy can indicate an emergency, and immediate hospitalization should be considered.	IIa	C
Diabetes					
Antihypertensive drug treatment is recommended for people with diabetes when office BP is ≥140/90 mmHg.	I	A	In most adults with elevated BP and diabetes, after a maximum of 3 months of lifestyle intervention, BP lowering with pharmacological treatment is recommended for those with confirmed office BP ≥130/80 mmHg to reduce CVD risk.	I	A
In people with diabetes receiving BP-lowering drugs it is recommended: • To target SBP to 130 mmHg and <130 mmHg if tolerated, but not <120 mmHg. • In older people (aged ≥65 years aged), to target to an SBP range of 130–139 mmHg.	I	A	In persons with diabetes who are receiving BP-lowering drugs, it is recommended to target systolic BP to 120–129 mmHg, if tolerated.	I	A
Chronic kidney disease					
In patients with diabetic or non-diabetic CKD, it is recommended that an office BP ≥140/90 mmHg be treated with lifestyle advice and BP-lowering medication.	I	A	In patients with diabetic or non-diabetic moderate-to-severe CKD and confirmed BP ≥130/80 mmHg, lifestyle optimization and BP-lowering medication are recommended to reduce CVD risk, provided such treatment is well tolerated.	I	A
In patients with diabetic or non-diabetic CKD:			In adults with moderate-to-severe CKD who are receiving BP-lowering drugs and who have eGFR >30 mL/min/1.73 m^2, it is recommended to target systolic BP to 120–129 mmHg, if tolerated. Individualized BP targets are recommended for those with lower eGFR or renal transplantation.	I	A
• It is recommended to lower systolic BP to a range of 130–139 mmHg.	I	A			
• Individualized treatment should be considered according to its tolerability and impact on renal function and electrolytes.	IIa	C			
RAS blockers are more effective at reducing albuminuria than other antihypertensive agents, and are recommended as part of the treatment strategy in hypertensive patients in the presence of microalbuminuria or proteinuria.	I	A	ACE inhibitors or ARBs are more effective at reducing albuminuria than other BP-lowering agents and should be considered as part of the treatment strategy for patients with hypertension and microalbuminuria or proteinuria.	IIa	B
Heart failure					
In patients with HFrEF, it is recommended that BP-lowering treatment comprises an ACE inhibitor or ARB, a beta-blocker and diuretic and/or MRA if required.	I	A	In patients with symptomatic HFrEF/HFmrEF, the following treatments with BP-lowering effects are recommended to improve outcomes: ACE inhibitors (or ARBs if ACE inhibitors are not tolerated) or ARNi, beta-blocker, MRA, and SGLT2 inhibitors.	I	A
In patients with HFpEF, because no specific drug has proven its superiority, all major agents can be used.	I	C	In hypertensive patients with symptomatic HFpEF, SGLT2 inhibitors are recommended to improve outcomes in the context of their modest BP-lowering properties.	I	A
			In patients with symptomatic HFpEF who have BP above target, ARBs and/or MRAs may be considered to reduce heart failure hospitalizations and reduce BP.	IIb	B

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Stroke					
In all hypertensive patients with ischaemic stroke or TIA, an SBP target range of 120–130 mmHg should be considered.	IIa	B	In patients with confirmed BP ≥130/80 mmHg with a history of TIA or stroke a systolic BP target of 120–129 mmHg is recommended to reduce CVD outcomes, provided treatment is tolerated.	I	A
Different ethnic groups					
In black patients, initial antihypertensive treatment should include a diuretic or a CCB, either in combination or with a RAS blocker.	I	B	In black patients from Sub-Saharan Africa who require BP-lowering treatment, combination therapy including a CCB combined with either a thiazide diuretic or a RAS blocker should be considered.	IIa	B
Resistant hypertension					
Recommended treatment of resistant hypertension is: • Reinforcement of lifestyle measures, especially sodium restriction. • Addition of low-dose spironolactone to existing treatment. • Or the addition of further diuretic therapy if intolerant to spironolactone, with either eplerenone, amiloride, a higher dose thiazide/thiazide-like diuretic, or a loop diuretic. • Or the addition of bisoprolol or doxazosin.	I	B	In patients with resistant hypertension and uncontrolled BP despite use of first-line BP-lowering therapies, the addition of spironolactone to existing treatment should be considered.	IIa	B
			In patients with resistant hypertension in whom spironolactone is not effective or tolerated, treatment with eplerenone instead of spironolactone, or the addition of a beta-blocker if not already indicated, and, next, a centrally acting BP-lowering medication, an alpha-blocker, or hydralazine, or a potassium-sparing diuretic should be considered.	IIa	B
			To reduce BP, and if performed at a medium-to-high volume centre, catheter-based renal denervation may be considered for resistant hypertension patients who have BP that is uncontrolled despite a three BP-lowering drug combination, and who express a preference to undergo renal denervation after a shared risk-benefit discussion and multidisciplinary assessment.	IIb	B
10. Acute and short-term management of blood pressure					
In patients with acute intracerebral haemorrhage: • Immediate BP lowering is not recommended for patients with systolic BP <220 mmHg.	III	A	In patients with intracerebral haemorrhage, immediate BP lowering (within 6 h of symptom onset) should be considered to a systolic target 140–160 mmHg to prevent haematoma expansion and improve functional outcome.	IIa	A
• In patients with systolic BP ≥220 mmHg, careful acute BP lowering with i.v. therapy to <180 mmHg should be considered.	IIa	B			
In hypertensive patients with an acute cerebrovascular event, anti-hypertensive treatment is recommended: • Immediately for TIA.	I	A	For patients with ischaemic stroke or TIA and an indication for blood pressure lowering, it is recommended that BP lowering therapy be commenced before hospital discharge.	I	B
• After several days in ischaemic stroke.	I	A			
In severe hypertension, drug treatment with i.v. labetalol, oral methyldopa, or nifedipine is recommended.	I	C	In severe hypertension in pregnancy, drug treatment with i.v. labetalol, oral methyldopa, or oral nifedipine is recommended. Intravenous hydralazine is a second-line option.	I	C

ABPM, ambulatory blood pressure monitoring; ACE, angiotensin-converting enzyme; ACR, albumin-to-creatinine ratio (urine); ARB, angiotensin receptor blocker; ARNi, angiotensin receptor–neprilysin inhibitor; BMI, body mass index; BP, blood pressure; CCB, calcium channel blocker; CKD, chronic kidney disease; CV, cardiovascular; CVD, cardiovascular disease; DASH, Dietary Approaches to Stop Hypertension; eGFR, estimated glomerular filtration rate; HBPM, home blood pressure monitoring; HFpEF, heart failure with preserved ejection fraction; HF(m)rEF, heart failure with (mildly) reduced ejection fraction; HFrEF, heart failure with reduced ejection fraction; HMOD, hypertension-mediated organ damage; i.v., intravenous; LVH, left ventricular hypertrophy; MRA, mineralocorticoid receptor antagonist; RAS, renin–angiotensin system; RCT, randomized controlled trial; SBP, systolic blood pressure; SCORE2, Systematic COronary Risk Evaluation 2; SCORE2-OP, Systematic COronary Risk Evaluation 2–Older Persons; SGLT2, sodium–glucose co-transporter 2; TIA, transient ischaemic attack.

[a]Class of recommendation.

[b]Level of evidence.

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3. Pathophysiology of elevated blood pressure and hypertension

Persistently high BP in systemic arteries is the hallmark of hypertension, which is the most important modifiable risk factor for all-cause and CVD morbidity and mortality globally.[2] Most patients with hypertension have essential or primary hypertension, where the exact cause remains unknown, while an estimated 10% have secondary hypertension, with an identifiable cause (notably some studies indicate that the prevalence of secondary hypertension may be substantially higher, with modern systematic screening).[10]

The pathophysiology of hypertension involves complex interactions between environmental and behavioural factors, genes, hormonal networks, and multiple organ systems (renal, cardiovascular, and central nervous system[11]) (*Figure 1*). In addition, vascular and immune mechanisms are involved.[12] Dysregulation of these processes leads to hypertension, which if uncontrolled, can lead to hypertension-mediated organ damage (HMOD) and adverse CVD outcomes.

Details on the pathophysiological processes, molecular mechanisms, and environmental and psychosocial elements that underlie hypertension are provided in the Supplementary text (Supplementary data online).

4. Clinical consequences of elevated blood pressure and hypertension

Longstanding hypertension causes organ damage and ultimately leads to cardiovascular, cerebrovascular, and clinical renal disease, which are all major contributors to the global burden of chronic disease

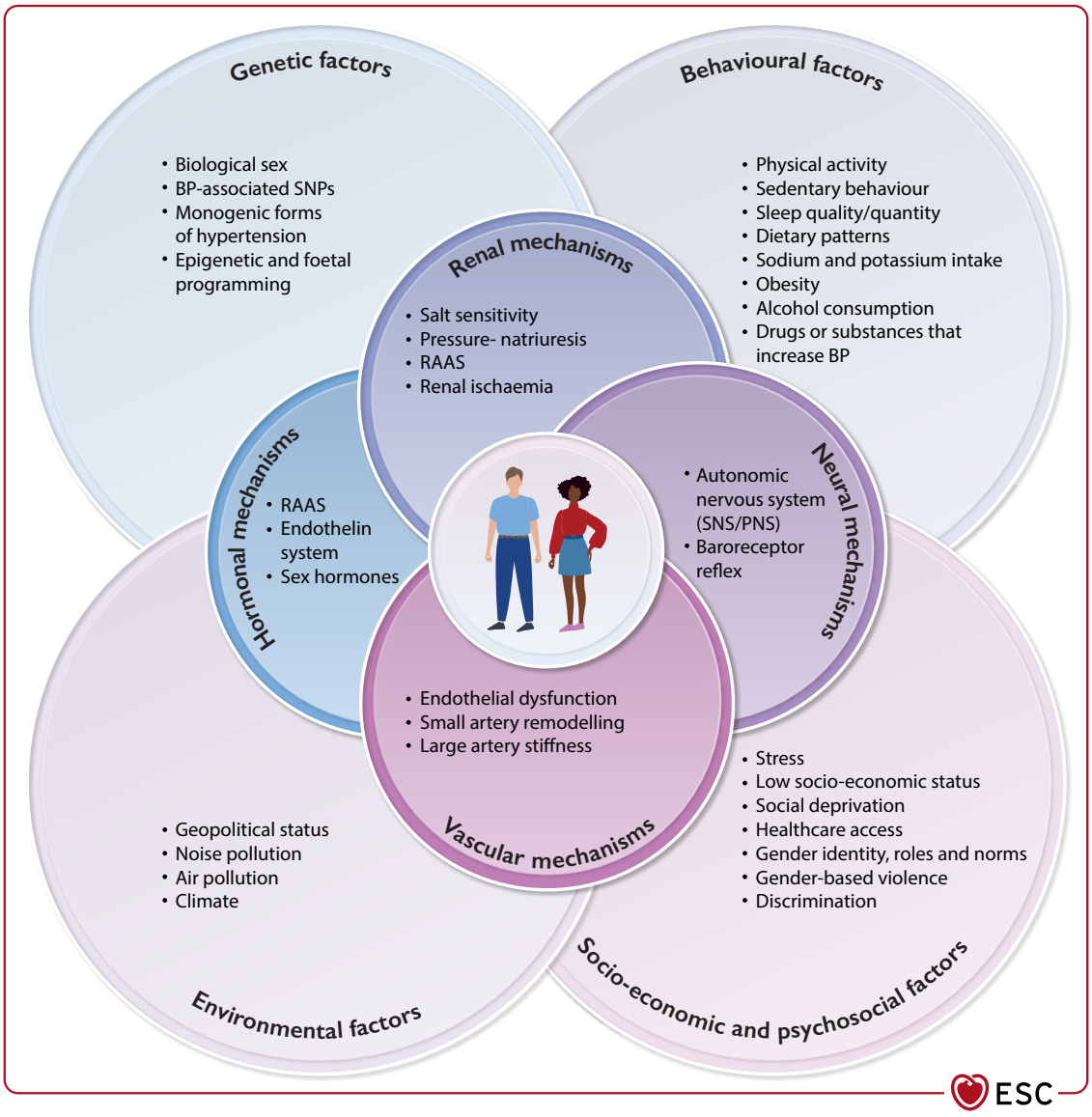

Figure 1 Pathophysiology of elevated blood pressure and hypertension. BP, blood pressure; PNS, parasympathetic nervous system; RAAS, renin-angiotensin-aldosterone system; SNP, single-nucleotide polymorphism; SNS, sympathetic nervous system. Complex interplay between genes, environmental, and behavioural factors, organs, physiological systems, and neurohumoral processes contribute to BP regulation. Dysfunction of these processes leads to hypertension. The contribution of these factors to elevated BP and hypertension may differ among males and females.

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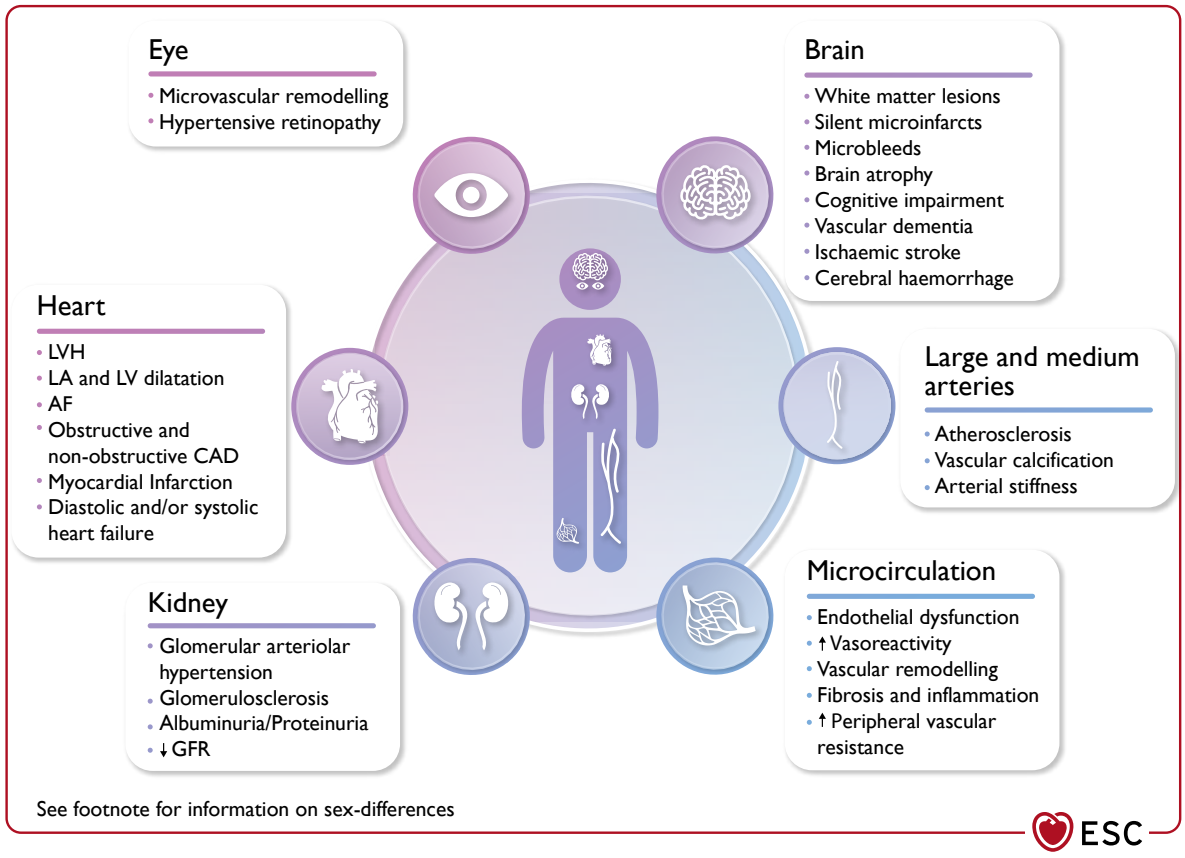

Figure 2 Persistently elevated blood pressure and hypertension lead to hypertension-mediated organ damage and cardiovascular disease. AF, atrial fibrillation; CAD, coronary artery disease; GFR, glomerular filtration rate; LA, left atrial; LV, left ventricular; LVH, left ventricular hypertrophy. See the supplementary data online for detailed information on sex differences.

(*Figure 2*).[2,13–22] Organs adversely affected by elevated BP and hypertension include the heart, brain, kidneys, eyes, and vessels (macrocirculation and microcirculation in organs with low resistance, such as the brain or kidney[23]), which undergo structural and functional changes. Although factors besides BP can contribute to these changes (i.e. dyslipidaemia, hyperglycaemia), we use the term 'hypertension-mediated organ damage' to indicate the presence of subclinical complications of hypertension that indicate high risk for subsequent clinical events. HMOD may have different profiles in men and women; for instance, left ventricular hypertrophy (LVH) and left atrial dilatation are more frequent in women.[24–28]

Evidence of HMOD usually indicates long-standing elevated BP and/or hypertension and confers incremental prognostic information regarding CVD risk in all BP categories.[29–31] Unless treated, HMOD can progress from asymptomatic to symptomatic, ultimately resulting in overt CVD events.[31]

The pathophysiological mechanisms underlying HMOD in the heart, brain, kidneys, vessels, and eyes are detailed in the Supplementary text (Supplementary data online). The clinical consequences of HMOD, especially cerebrovascular disease (stroke and cognitive decline), kidney disease (acute and chronic), and heart disease [heart failure, atrial fibrillation (AF), ischaemic heart disease, and valvular disease] are also discussed in the Supplementary text (Supplementary data online). In addition, the Supplement highlights the impact of different measures of BP on CVD risk, including systolic BP, diastolic BP, pulse pressure, and BP variability.[22,32–36]

5. Measuring blood pressure

5.1. Introduction and pertinent definitions

This section reviews practical aspects of BP measurement, including technique and clinical validation of devices. It also reviews the evidence for the most appropriate BP measurement methods when screening populations for hypertension, diagnosing hypertension, and managing patients receiving BP-lowering interventions. The current guidelines promote use of out-of-office measurement for diagnosis and ongoing management of hypertension, reflecting increasing evidence for the stronger relationship of home and ambulatory monitoring with outcomes, the ability to detect white-coat and masked hypertension, new BP treatment targets as low as 120–129 mmHg systolic (*Table 5*), and evidence supporting enabling patient involvement and shared decision-making.

Definitions:

Systolic BP: arterial BP during systole (maximum arterial pulsatile pressure). This is measured using an auscultatory device at the onset of the first Korotkoff sound. Oscillometric devices estimate systole using an algorithm that imputes from mean arterial pressure.[37]

Diastolic BP: arterial BP during diastole (minimum arterial pulsatile pressure). This is measured using an auscultatory device at the time of complete disappearance of the Korotkoff sounds (fifth sound). If there is no disappearance of sounds (no fifth sound) then the fourth

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Korotkoff sound (muffling) is used to estimate diastolic BP. Oscillometric devices estimate diastole using an algorithm that imputes from mean arterial pressure.[37]

Inter-arm difference: systolic BP difference of >10 mmHg when BP is measured sequentially in each arm.[38]

Postural/orthostatic hypotension: decrement of ≥20 mmHg in systolic BP and/or ≥10 mmHg in diastolic BP when BP is measured in the standing position at 1 and/or 3 min after standing following a 5-min period in the sitting or lying position.

White-coat hypertension: BP that is above the threshold for diagnosing hypertension in the office but below the threshold in home/ambulatory settings, e.g. ≥140/90 mmHg in office but <135/85 mmHg at home/ambulatory daytime (or 24-h BP < 130/80 mmHg).

Masked hypertension: BP that is below the hypertension diagnostic threshold in the office but above the hypertension diagnostic threshold in home/ambulatory settings, e.g. <140/90 mmHg in clinic but ≥135/85 mmHg at home/ambulatory daytime (or 24-h BP ≥130/80 mmHg).

Office BP: also known as clinic BP. The two terms are interchangeable. This guidelines document uses 'office BP' preferentially. Of note, office BP can be measured manually or using an automated device. In addition, automated office BP (AOBP) can be conducted in a setting attended by a healthcare professional or in an unattended fashion. Finally, not all office BP measurements are equal, with some facilities using a standardized method (which is recommended and outlined below) and others unfortunately using suboptimal approaches to office BP measurement.

Home BP measurement (HBPM): an out-of-office approach to measuring BP when the patient measures their own BP at home using a validated monitor (usually an upper-arm oscillometric cuff device).

Ambulatory BP measurement (ABPM): an out-of-office BP measurement that uses a fully automated oscillometric device, usually for a 24-h period, and measures BP at set intervals.

5.2. Practical recommendations for measuring blood pressure

5.2.1. Clinical validation of equipment for measuring blood pressure

A prerequisite of BP measurement is that it must be undertaken using a device that has been clinically validated and confirmed to be accurate. Of the commercially available oscillometric BP measurement devices, as few as 6% have been adequately tested.[39–41] National and international organizations provide lists of validated monitors (e.g. www.stridebp.org, www.validatebp.org).

Since the 2018 ESC/ESH Guidelines on the management of arterial hypertension, three arbiters of device accuracy (the Association for the Advancement of Medical Instrumentation, the ESH, and the International Organization for Standardization) have published a universal standard for validating devices for measuring BP.[42] This standard will likely become widely adopted. Validation standards and methodology need to be developed and implemented for novel BP measurement devices that are non-occlusive and 'cuffless'.[43,44]

5.2.2. Office blood pressure measurement

All BP measurements can be influenced by circumstances of measurement, including position, ambient temperature, the technique of measurement, accuracy of equipment, and physical condition of the patient.[45] For BP measurements in the office, we recommend following a standardized method (*Figure 3*).

Patient preparation: BP should be measured with the patient seated comfortably after 5 min of rest. Patients should avoid exercising and stimulants (caffeine, tobacco) for at least 30 min before measurement. The patient's bladder should be emptied if needed.[46] Patients should be seated with their legs unfolded and their back supported at the time of measurement. The arm should be supported (to avoid isometric exercise-induced increases in BP). Clothing at the location of the cuff placement should be removed; rolling up of shirt sleeves should be avoided as this can result in a tourniquet effect.

BP measurement technique: auscultatory or oscillometric techniques can be used to measure BP non-invasively. The manual auscultatory approach is the traditional method of measuring systolic and diastolic BP at the brachial artery site using a stethoscope. In contrast, oscillometric devices compute mean arterial BP using the oscillation amplitude with cuff deflation (or inflation) and then estimate systolic and diastolic BP. Oscillometric devices can be semi-automated (taking one reading per activation) or fully automated (obtaining multiple readings before averaging them). Oscillometric devices are not typically validated for use in AF, and the manual auscultatory method is preferred in these circumstances when feasible.[47–49]

BP cuff selection and positioning: an appropriately sized cuff should be used, as an under-sized or over-sized cuff will artificially elevate or reduce BP, respectively.[50] The bladder length should be 75%–100% and the width 35%–50% of the arm circumference. The arm circumference can be measured at the mid-point of the olecranon and the acromion but many cuffs include sizing indicators. The cuff should be positioned on the patient's upper arm at the level of the heart with the lower edge of the cuff a few centimetres above the antecubital fossa. The stethoscope should not be placed under the cuff. In those with significant obesity where a correctly fitting upper arm cuff is not available, measurement at the lower arm or wrist can be considered as an alternative.[51]

BP measurement by manual auscultation: three BP measurements should be taken, each 1–2 min apart, and additional measurements only if the readings differ by >10 mmHg (e.g. this may occur with arrhythmias or white-coat effects). The BP recorded should be the average of the last two BP readings.

BP measurement using AOBP measurement: as noted above, AOBP using oscillometric devices may be obtained with (attended) or without (unattended) clinicians or staff present. Clear evidence regarding superiority of unattended vs. attended AOBP in managing BP to reduce rates of CVD is lacking; however, because BP readings may differ for unattended vs. attended measurements,[52] we advise that a consistent approach be used depending on local resource and preference. AOBP monitors typically make three or six readings at 1-min intervals and provide an average. AOBP correlates more closely with mean ABPM than with the manual auscultatory technique and may reduce measurement error and white-coat effects.[53]

Inter-arm BP difference: at the initial visit, BP should be measured in both arms to detect an inter-arm difference. Though devices allowing simultaneous measurement in both arms exist, sequential arm measurement is considered sufficiently reliable.[54,55] Measurement in the contralateral arm should be undertaken once the three measurements in the index arm have been taken, and if a difference is detected, further measurement in the original arm is indicated to ensure the difference is consistent. If systolic BP differs by >10 mmHg between arms, subsequent measurements are obtained using the arm with the higher BP value. Significant inter-arm BP differences may reflect arterial stenosis or

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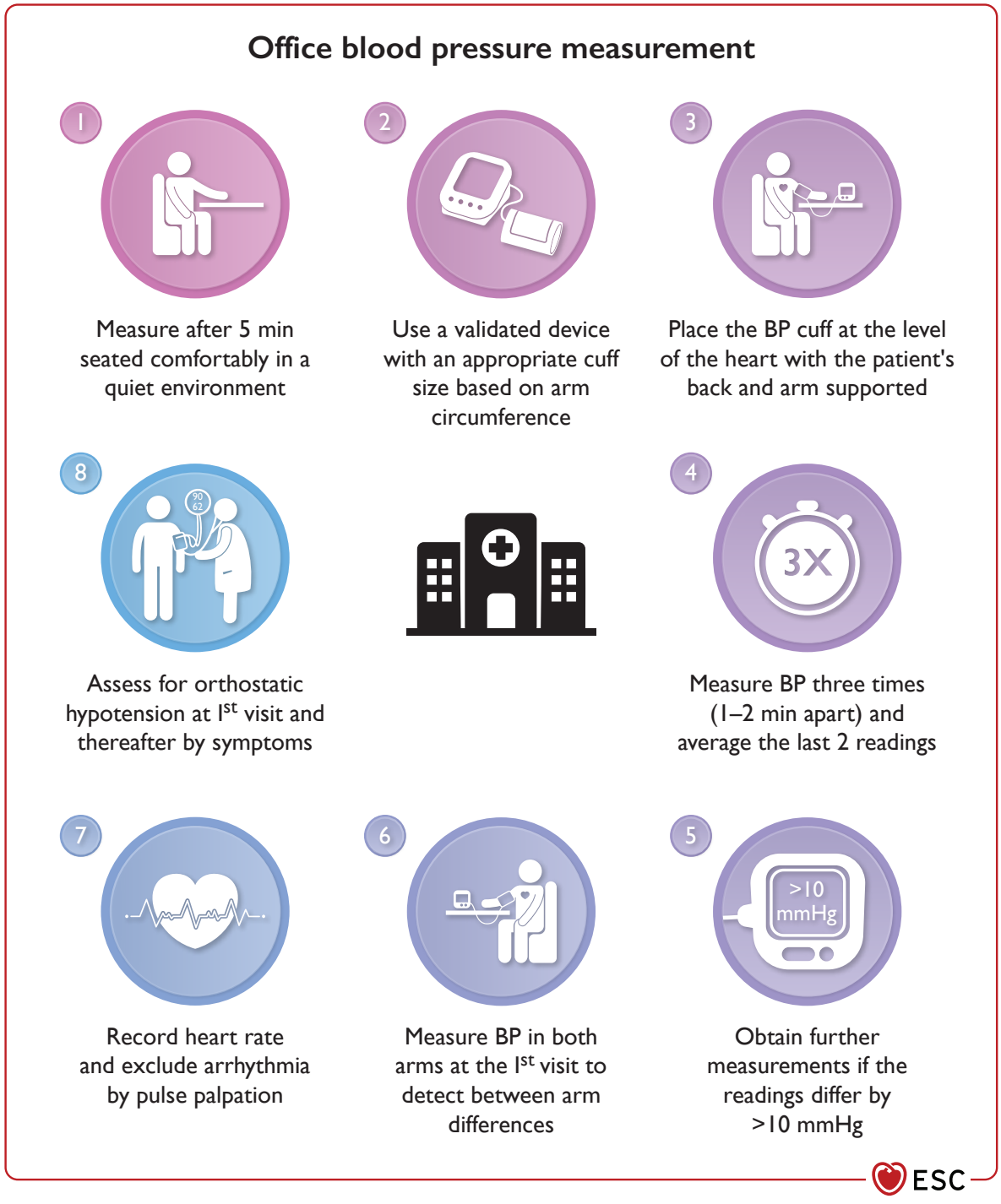

Figure 3 Summary of office blood pressure measurement. BP, blood pressure.

coarctation of the aorta, which may require investigation. Also, of note, in some patients one arm is preferred to the other for routine BP measurement (e.g. to avoid measurement of BP in an arm with an arteriovenous fistula or an arm where axillary lymph node dissection has occurred).

Postural/orthostatic hypotension: patients should be assessed for orthostatic hypotension at the initial visit and if concerning symptoms arise. After 5 min of rest in the sitting or lying position, BP should be measured at 1 min and/or 3 min after standing, with a threshold for orthostatic hypotension of ≥20/10 mmHg (systolic BP/diastolic BP) drop. Measurement after lying may be more sensitive for detecting orthostatic hypotension and may better predict falls but may be less feasible than measurement after sitting in clinical practice.[56]

Pulse assessment: heart rate should be recorded at the initial visit and arrhythmia excluded.

5.2.3. Home blood pressure measurement

HBPM is an out-of-office approach to measuring BP when the patient measures their own BP at home using a validated monitor (usually an upper-arm oscillometric cuff device).[57,58] A consistent approach to

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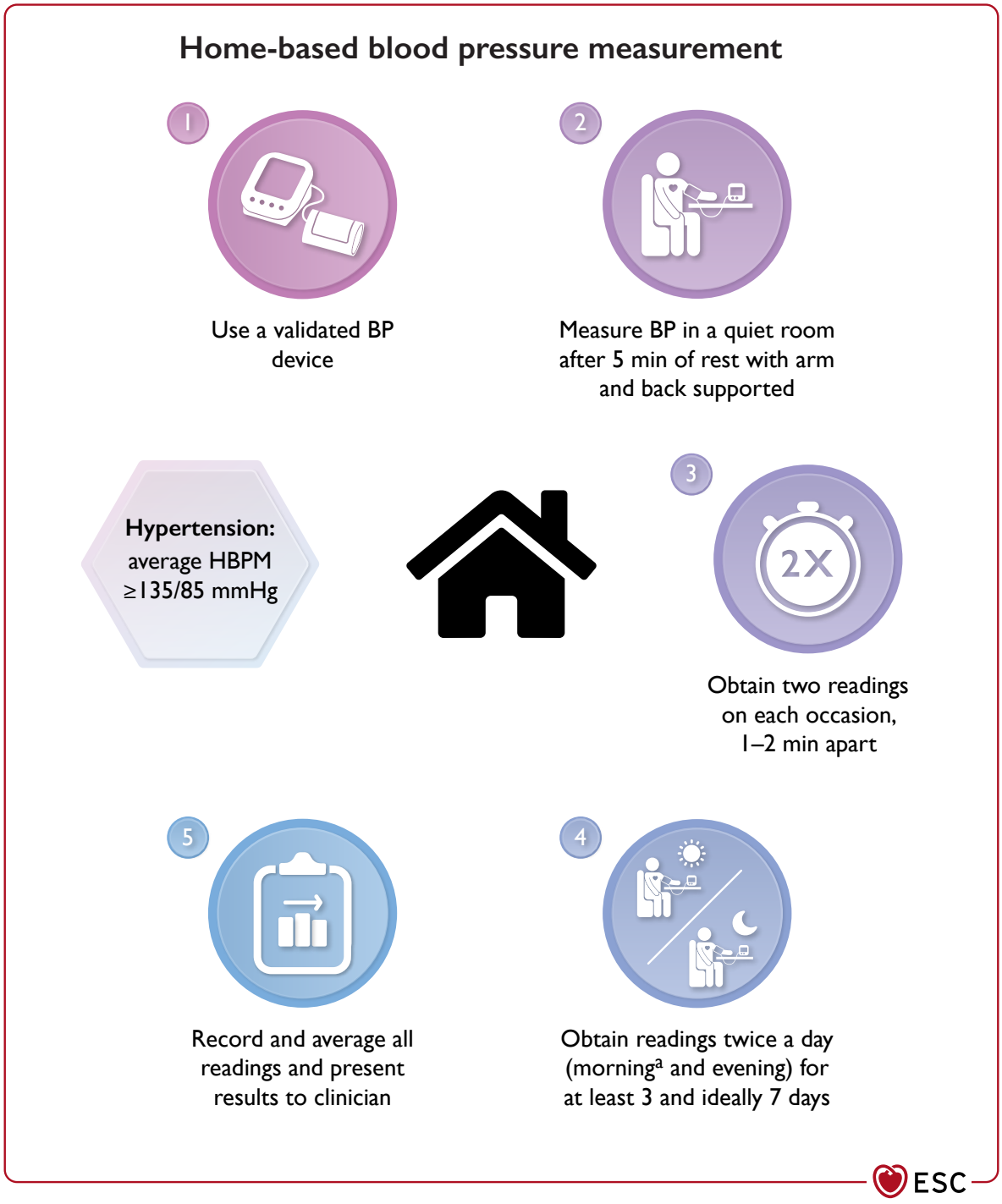

Figure 4 Summary of home blood pressure measurement. BP, blood pressure; HBPM, home blood pressure measurement. [a]Morning HBPM readings should be obtained before breakfast and before intake of medication but not immediately after awakening.

HBPM should be used (*Figure 4*). Patients should be counselled to follow the same preparation steps as used in clinics, which are outlined in *Section 5.2.2*. Two measurements should be taken at each measurement session, performed 1–2 min apart. Measurements should be made twice a day (morning and evening) at the same time for a minimum of 3 days and up to 7 days.[59] At the end of the measurement period, all readings are averaged. If the average after 3 days is close to the treatment threshold, then measurement should continue for the full 7 days. Patients should be informed to keep a record of their home BP values and to ask their healthcare provider that the device accuracy be intermittently checked. Devices older than 4 years may be inaccurate and, if inaccurate, should be replaced.[60]

An average HBPM of ≥135/85 mmHg (equivalent to an office BP of ≥140/90 mmHg) should be used to diagnose hypertension and an average systolic BP of 120–134 mmHg or diastolic BP of 70–84 mmHg should be used to diagnose elevated BP. Of note, we use the same lower BP threshold (120/70 mmHg) for both office and HBPM in defining elevated BP.[61]

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5.2.4. Ambulatory blood pressure measurement

ABPM (summarized in *Figure 5*) is an out-of-office BP measurement that uses a fully automated device, usually for a 24-h period. The devices measure BP by the oscillometric method and are programmed to measure BP at set intervals. Readings are usually obtained at 15–30 min intervals during the day (typically 7 a.m. to 11 p.m.) and 30–60 min intervals at night (typically 11 p.m. to 7 a.m.). The software usually provides average BP measurements for daytime, night-time, and 24 h. A minimum of 70% useable BP recordings is required for a valid measurement session, typically numbering ≥27 measurements over 24 h. Preferably, seven nocturnal readings should also be obtained.[62] However, emerging data indicate that ≥8/≥4 wake/sleep readings may be adequate if more cannot be obtained.[63] Prior to using mean ABPM values (either 24 h, daytime, or night-time) the raw BP values at each measurement should be reviewed for possible outlier or erroneous values. A diary should record activities (e.g. meals and exercise) and sleep time to assist interpretation. The diagnostic thresholds for elevated BP and hypertension using ABPM, and comparison with office BP and HBPM, are provided in *Table 5*.[61,64]

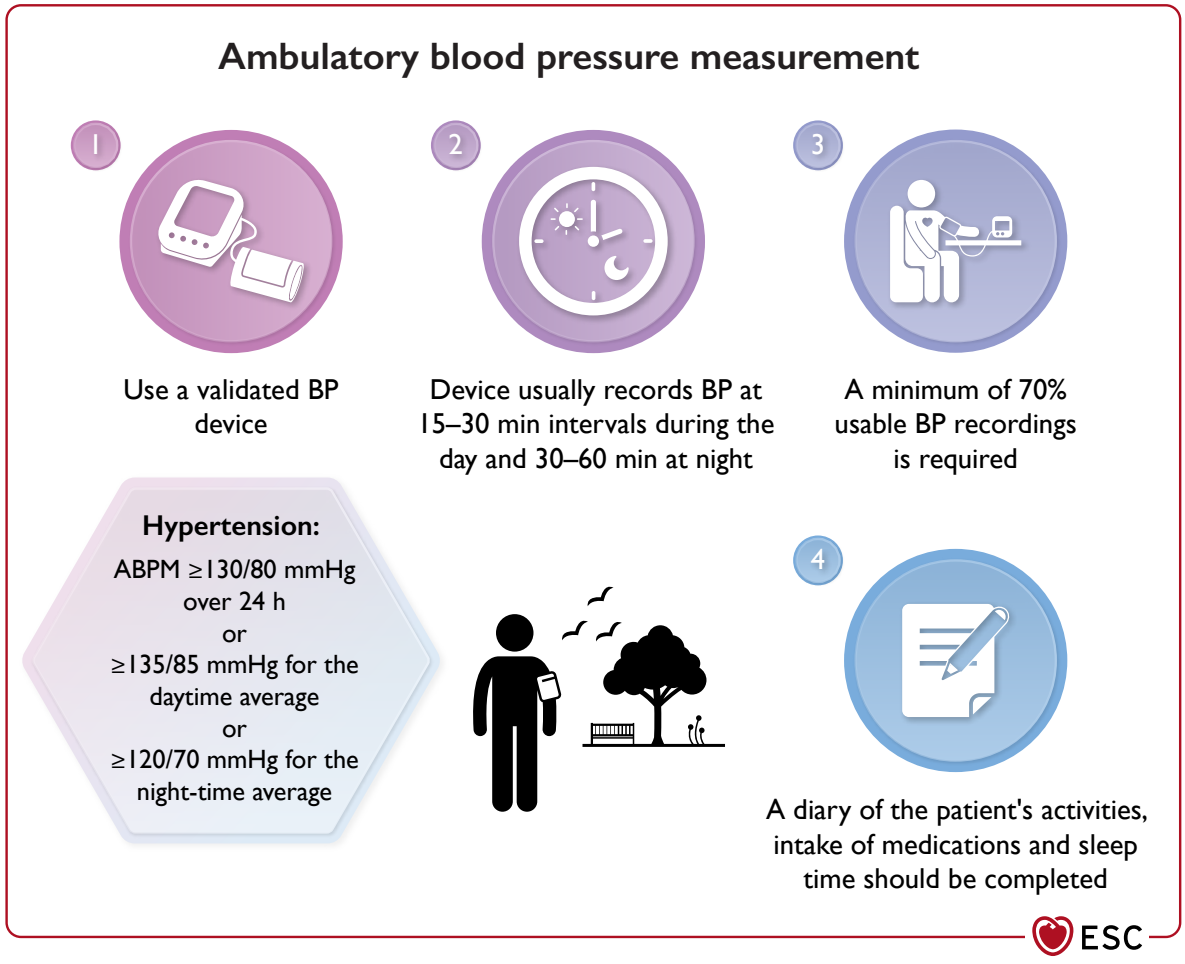

Figure 5 Summary of ambulatory blood pressure measurement. ABPM, ambulatory blood pressure measurement; BP, blood pressure.

Table 5 Comparison of office, home, and ambulatory blood pressure measurement thresholds for elevated blood pressure and hypertension

	Office BP (mmHg)[a]	Home BP (mmHg)	Daytime ABPM (mmHg)	24 h ABPM (mmHg)	Night-time ABPM (mmHg)
Reference					
Non-elevated BP	<120/70	<120/70	<120/70	<115/65	<110/60
Elevated BP	120/70–<140/90	120/70–<135/85	120/70–<135/85	115/65–<130/80	110/60–<120/70
Hypertension	≥140/90	≥135/85	≥135/85	≥130/80	≥120/70

ABPM, ambulatory blood pressure monitoring; BP, blood pressure.
[a]The BP thresholds provided assume that a standardized approach to office BP measurement is performed (*Figure 3*). However, evidence indicates that office BP measurement in routine clinical settings is often not done using a standardized approach and, in this case, the routine office BP value may be 5–10 mmHg higher than it would have been if measured using the recommended standardized approach.[65,66]

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5.2.5. Comparison of home and ambulatory blood pressure monitoring

There is overlap between home and ambulatory monitoring in terms of differentiating between hypertensive phenotypes. However, around 15% of people will have diagnostic disagreement, of whom approximately 50% will represent clinically significant differences of >5 mmHg.[67] The advantages and disadvantages of home and ambulatory monitoring are outlined in *Table 6*.

5.3. What is the best method for measuring blood pressure to diagnose hypertension?

5.3.1. Blood pressure measurement for hypertension screening

Opportunistic screening is typically performed using office BP measurement and is key in detecting possible hypertension. However, a single screening office BP alone does not typically have sufficient diagnostic test performance to establish a diagnosis, especially for BP values close to diagnostic thresholds. Therefore, a single screening office BP requires some form of repeat BP assessment to confirm a diagnosis (preferably out-of-office or repeat office if out-of-office is not available). Relatedly, the BP threshold for acting on a screening office BP by conducting repeat BP assessments should also be lower than the office BP threshold used for diagnosing hypertension. This latter consideration is particularly relevant in the presence of increased CVD risk or markers of HMOD. Also of note, populations where masked hypertension is more prevalent include men, those who smoke, those with excessive alcohol intake, or those with diabetes or obesity.[68,69] While a screening office BP of >160/100 mmHg is almost always consistent with a diagnosis of hypertension, a small proportion of patients will have extreme white-coat effects that motivate prompt repeat BP assessment.[68] Hypertension screening approaches are discussed further in *Section 7.1*.

Table 6 Comparison of ambulatory and home blood pressure monitoring

Ambulatory monitoring
Advantages
• Can identify white-coat and masked hypertension
• Measurement in real-life settings and during usual activities
• Stronger prognostic evidence
• Night-time readings
• Abundant information from a single investigation, including short-term diurnal BP variability
• Additional BP phenotyping (e.g. nocturnal dipping status)
Disadvantages
• Relatively expensive and sometimes limited availability
• Can be uncomfortable and affect sleep
Home monitoring
Advantages
• Identify white-coat and masked hypertension
• Cheap and widely available
• Measurement at home, which may be more relaxed than at doctor's office
• Patient engagement in BP measurement and telemedicine potential
• Easily repeated and used over longer periods to assess day-to-day BP variability
Disadvantages
• Only static BP at rest is typically available
• Potential for measurement error due to improper measurement technique or unvalidated or poorly calibrated device
• Nocturnal readings not usually possible

BP, blood pressure.

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5.3.2. Blood pressure measurement for diagnosing hypertension

After detecting high BP in the office, subsequent BP measurement for diagnosing hypertension depends on the clinical circumstances. Office BP has lower specificity than ABPM for detecting hypertension, so diagnosis based on office BP alone is less desirable unless resources do not allow out-of-office measurements.[70] For screening BP of 160–179 mmHg systolic or 100–109 mmHg diastolic, prompt confirmation (within 1 month) using either office or out-of-office methods is recommended, as delays in treatment are associated with increased CVD event rates.[71] For BP of ≥180/110 mmHg, assessment for hypertensive emergency is recommended. In the setting of hypertensive emergency, immediately commencing BP-lowering treatment is recommended, otherwise, prompt confirmation (preferably within a week) can be considered prior to commencing treatment (*Sections 7 and 10*).

For screening BP of 140–159/90–99 mmHg, out-of-office BP should be measured to confirm the diagnosis.[72] When treatment of elevated BP is being considered (e.g. 120–139/70–89 mmHg) for individuals with high risk CVD conditions or sufficiently high 10-year predicted CVD risk, out-of-office BP measurement is recommended, both to confirm BP and to assess for masked hypertension. Out-of-office measurements may also be helpful for individuals with office BP of 130–139/85–89 mmHg to diagnose masked hypertension. Further details on the diagnostic evaluation of hypertension are provided in *Section 7.2*.

5.4. What is the best method for measuring blood pressure for long-term management of hypertension?

While repeat office measurement of BP remains the commonest approach to long-term management of hypertension, several lines of investigation support augmenting office BP measurements with out-of-office assessment.

5.4.1. Home monitoring

There are over 50 trials of different self-monitoring-based interventions.[73] Self-monitoring is associated with lower mean systolic BP at 12 months [−3.2 mmHg; 95% confidence interval (CI) −4.9 to −1.6 mmHg].[74] Furthermore, there are known benefits of telemonitoring, digital interventions, and mobile health in managing BP.[75–78] Self-monitoring is also likely to be cost-effective.[79] Unfortunately, in clinical practice, some patients may not provide reliable information on their home BP, and both their device and measurement technique need to be checked.

5.4.2. Ambulatory monitoring

ABPM provides a reference BP measurement but repeat ABPM testing is sometimes not practical due to resource constraints and, uncommonly, low patient acceptability.[80] There is a paucity of data on treatment guided by ABPM vs. that of office or HBPM measurements. A trial of treatment guided by HBPM vs. clinic and ambulatory monitoring found equivalence in BP control and HMOD.[81] Other studies reported a non-significant trend to worse BP control with ambulatory vs. office BP monitoring, though the ambulatory group also received fewer

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medications.[82] Potential advantages of ABPM over HBPM include diagnosing nocturnal hypertension or symptomatic transient hypotension or hypertension with exertion. As such, ABPM and HBPM should be considered complementary and additive, rather than competing approaches to long-term BP management.[83]

5.5. Measuring blood pressure in selected groups

5.5.1. Pregnancy

Monitoring BP during pregnancy is typically done at antenatal visits, which vary dependent on trimester (with increasing frequency towards term). BP tends to reach a nadir at 20–30 weeks of pregnancy before increasing towards term at 40 weeks.[84] Only a small number of automated oscillometric BP monitors have been adequately validated in pregnancy and several have failed, usually due to providing BP values that are erroneously high.[85] Auscultatory measurement with sphygmomanometry is consequently the clinical standard in pregnancy.[85] Self-monitoring at home is not yet proven to be effective in gestational hypertension.[86,87] While norms for BP during pregnancy remain unclear, the 2022 Chronic Hypertension and Pregnancy (CHAP) trial indicated benefit of targeting clinic BP below 140/90 mmHg.[88] Consideration of secondary causes of hypertension is important in young women with gestational hypertension. Further details are provided in *Section 9.2* and the 2018 ESC Guideline for the management of cardiovascular disease during pregnancy.[89]

5.5.2. Atrial fibrillation

Hypertension is a risk factor for AF.[90,91] Oscillometric BP monitors are not always accurate in the presence of AF, due to the greater variability of BP beat to beat, so multiple auscultatory measurements are recommended.[48,92,93] Some oscillometric BP monitors include an algorithm for detecting AF, but an electrocardiogram (ECG) is still required to confirm the diagnosis.[49,94]

5.5.3. Orthostatic hypotension

Postural or orthostatic hypotension is common,[95,96] present in approximately 10% of all hypertensive adults and up to 50% of older institutionalized adults.[97,98] Orthostatic hypotension is defined as a BP drop of ≥20/10 mmHg 1 and/or 3 min after standing following a 5-min period in the seated or lying position.[99–101] Diagnosis is made in the office. Routine ABPM is not currently suitable for formally assessing orthostatic hypotension,[102] though it may help in some cases, particularly when accompanied by a patient symptom diary.[103]

5.6. Novel methods of measuring blood pressure

New methods to measure BP are under development. Continuous office and out-of-office BP recordings and ABPM and HBPM have been developed that derive beat-to-beat, reading-to-reading, and day-to-day BP variability. However, there is no agreement on the optimum approach to measuring variability, and there is no trial evidence that reducing BP variability specifically can reduce CVD events.[104] Other emerging technologies include wearable, wrist-based BP measurement devices, devices evaluating central BP, and cuffless devices implementing plethysmographic or other technologies.[105,106] However, there is at present insufficient scientific consensus on the accuracy standards and validation procedures that these cuffless devices must comply with prior to commercialization.[43,44,107–109]

In view of these issues, none of these cuffless measurement modalities are currently recommended for routine clinical use.

Recommendation Table 1 — Recommendations for measuring blood pressure (see Evidence Tables 1–8)

Recommendations	Class[a]	Level[b]
It is recommended to measure BP using a validated and calibrated device, to enforce the correct measurement technique, and to apply a consistent approach to BP measurement for each patient.[41,42]	I	B
All adult patients (≥18 years) are recommended to have their office and/or out-of-office BP measured on an opportunistic basis and recorded in their medical file, and be told what their current BP is.	I	C
Out-of-office BP measurement is recommended for diagnostic purposes, particularly because it can detect both white-coat hypertension and masked hypertension. Where out-of-office measurements are not logistically and/or economically feasible, then it is recommended that the diagnosis be confirmed with a repeat office BP measurement using the correct standardized measurement technique.[70]	I	B
It is recommended that office BP should be measured in both arms at least at the first visit, because a between-arm systolic BP difference of >10 mmHg is associated with an increased CVD risk and may indicate arterial stenosis.[55,110]	I	B
If a between-arm difference of >10 mmHg in systolic BP is recorded, then it is recommended that all subsequent BP readings use the arm with the higher BP reading.[110]	I	B
Out-of-office BP measurement is recommended for ongoing management to quantify the effects of treatment and guide BP-lowering medication titration, and/or identify possible causes of side effects (e.g. symptomatic hypotension). Where out-of-office measurements are not logistically and/or economically feasible, then ongoing management is recommended to be based on repeated office BP measurements using the correct standardized measurement technique.[74,111,112]	I	B
It is recommended that all patients undergoing BP measurement also undergo pulse palpation at rest to determine heart rate and arrhythmias such as AF.[113]	I	C
Most automated oscillometric monitors have not been validated for BP measurement in AF; BP measurement should be considered using a manual auscultatory method in these circumstances where possible.[47–49]	IIa	C

Continued

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An assessment for orthostatic hypotension (≥20 systolic BP and/or ≥10 diastolic BP mmHg drop at 1 and/or 3 min after standing) should be considered at least at the initial diagnosis of elevated BP or hypertension and thereafter if suggestive symptoms arise. This should be performed after the patient is first lying or sitting for 5 min.	IIa	C
Other BP measures and indices (pulse pressure, BP variability, exercise BP) may be considered to provide additional clinical information on CVD risk in some circumstances.	IIb	C

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AF, atrial fibrillation; BP, blood pressure; CVD, cardiovascular disease.
[a]Class of recommendation.
[b]Level of evidence.

6. Definition and classification of elevated blood pressure and hypertension, and cardiovascular disease risk assessment

6.1. Definition and classification of elevated blood pressure and hypertension

Epidemiological studies demonstrate a continuous and log-linear association between BP and adverse CVD outcomes.[22,32,33,114,115] Starting at levels as low as 90 mmHg systolic, the higher the BP the higher the relative risk of CVD including atherosclerosis.[32,114] These observational data are complemented by randomized clinical trials (RCTs),[116] which have provided experimental evidence regarding the BP range for which BP lowering with treatment is proven to reduce CVD events. Of note, some studies suggest a stronger relative risk for CVD for a given BP among females compared with males.[117,118]

A healthy lifestyle should be encouraged for all adults to prevent an increase in BP and development of hypertension.[119,120] To aid pharmacological treatment decisions, the 2024 ESC Guidelines recommend a simplified categorization of adults according to their BP (*Figure 6*). In compiling this categorization, priority was given to evidence from randomized trials over observational data. However, it is important to reiterate that the risk of CVD attributable to BP is continuous and that interpreting randomized trial data is an iterative process involving an element of subjectivity. As such, no categorization of BP can be considered immutable or flawless.

The 2024 Guidelines define hypertension as a confirmed office systolic BP of ≥140 mmHg or diastolic BP of ≥90 mmHg. For this diagnosis to be made, confirmation is recommended with out-of-office measurements (HBPM or ABPM) or at least one repeat office measurement at a subsequent visit, as detailed in *Section 5* and *Section 7.2*. This definition is based on several factors. First, meta-analyses of randomized trials provide evidence among all adults and across various settings for the benefit of BP-lowering therapy among patients with BP above this threshold.[116,121,122] Second, most adults with BP above this threshold are at increased CVD risk, typically with 10-year risk estimates of ≥10% for fatal and non-fatal CVD events.[123–125] The higher the patient's baseline absolute risk for CVD, the greater the net benefit from BP-lowering treatment and, at the population level, the lower the estimated number needed to treat (NNT).[126–128] Third, this more traditional BP threshold for hypertension is already widely used by policymakers to define a disease state, and maintaining this BP threshold to define hypertension (vs. lowering it) does not require most adults to be labelled with what is widely considered a disease.[129]

Here, we introduce a new BP category called 'elevated BP', which is defined as an office systolic BP of 120–139 mmHg or diastolic BP of 70–89 mmHg. Within this BP range, the efficacy of BP-lowering therapy has been established in meta-analyses of RCTs,[116] but average CVD risk in the elevated BP group is not sufficiently high to merit drug treatment in all patients.[123,124,130] Pharmacological treatment initiation is, however, suggested for a subgroup of patients within this BP range who are at increased global risk of CVD as identified by the risk stratification approach outlined in *Sections 6.3*, *6.4*, and *8*.

Non-elevated BP is defined as a systolic BP of <120 mmHg and a diastolic BP of <70 mmHg. Fewer individuals within this BP range are at increased risk of CVD,[124] and evidence for CVD benefit with BP-lowering pharmacological treatment is lacking due to an absence of trials. We use the term 'non-elevated BP' to define this BP category in recognition that these are treatment categories and not prognostic categories. Because the relative risk for CVD starts to increase at BP below this threshold (even as low as 90 mmHg systolic BP), particularly among women,[117,118] we avoid terms like 'normal BP', 'optimal BP', or 'normotension' in defining this category.

Recommendation Table 2 — Recommendations for categorizing blood pressure (see Evidence Table 9)

Recommendation	Class[a]	Level[b]
It is recommended that BP be categorized as non-elevated BP, elevated BP, and hypertension to aid treatment decisions.[116,121,122,131–138]	I	B

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BP, blood pressure.
[a]Class of recommendation.
[b]Level of evidence.

6.2. Principles of a risk-based approach for managing blood pressure and preventing cardiovascular disease

In the context of BP-lowering interventions, randomized trials demonstrate a consistent relative risk reduction in adverse CVD outcomes per unit reduction in BP.[131,139] However, many medical interventions incur costs and have side effects. Therefore, guidance is needed on selecting patients most likely to benefit from BP-lowering treatment. This is especially true among adults with elevated BP (office systolic BP of 120–139 mmHg and/or diastolic BP of 70–89 mmHg). Practical aspects for implementing a risk-based approach are further discussed in *Section 8*.

6.2.1. Role of cardiovascular disease risk assessment

The risk of adverse CVD outcomes increases log-linearly with constant increments in systolic BP and diastolic BP.[22,32,33,114,140] Concurrently, at higher BP, there is clustering of additional CVD risk factors.[141,142] Consequently, many patients with hypertension will have an estimated 10-year risk for CVD events of ≥10%,[116,121,122] which, for the purposes of these guidelines, is considered sufficiently high risk to merit consideration of BP-lowering treatment in the setting of elevated BP.[143]

Using BP thresholds for hypertension alone for allocating treatment would lead to under-treatment of many high-risk patients.[144,145,115] A substantial proportion of excess CVD events attributable to BP occur in patients with BP levels below the traditional threshold for

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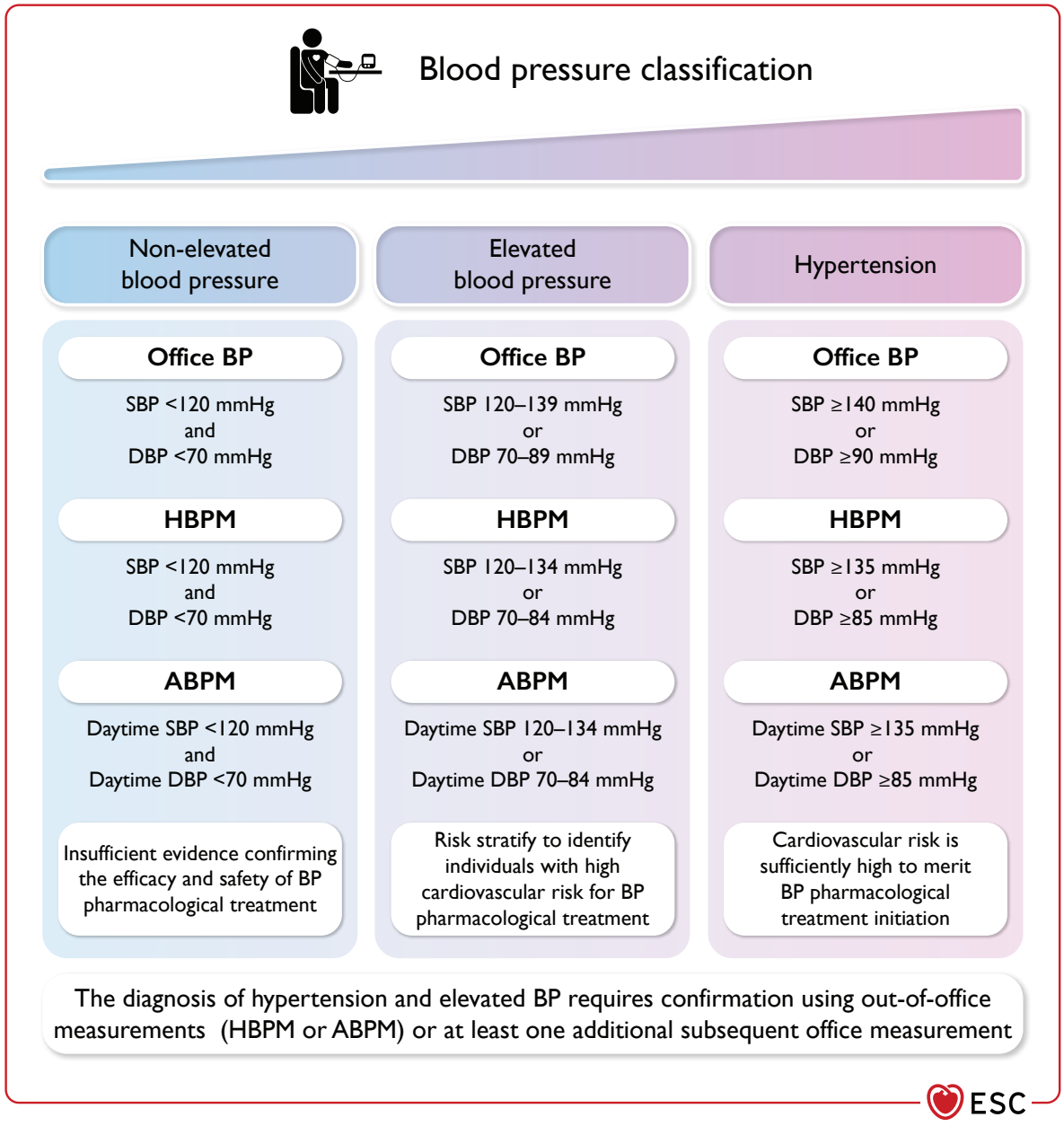

Figure 6 Blood pressure categories. ABPM, ambulatory blood pressure monitoring; BP, blood pressure; DBP, diastolic blood pressure; HBPM, home blood pressure monitoring; SBP, systolic blood pressure. We note that the respective non-daytime ABPM thresholds for elevated BP and hypertension diagnosis are listed in *Section 5* (*Table 5*).

hypertension diagnosis. As the efficacy of BP lowering on preventing CVD events extends down to a systolic BP of 120 mmHg and a diastolic BP of 70 mmHg,[116,135,136,146] patients with elevated BP and increased CVD risk can also derive benefit from BP-lowering treatment.[124,145]

The heterogeneity in CVD risk among adults with elevated BP is larger than in those with hypertension, as such patients tend to be younger, and their absolute CVD risk depends more on the prevalence of concomitant CVD risk factors.[123,147] Consequently, formally estimating the patient's CVD risk, encapsulating demographics and other CVD risk factors, is recommended to guide BP-lowering treatment decisions among patients with elevated BP.[148–151]

6.3. Predicting cardiovascular disease risk

Certain conditions on their own are associated with sufficient CVD risk such that patients with elevated BP alongside these conditions can be considered for BP-lowering therapy (*Figure 7*). These include moderate or severe chronic kidney disease (CKD),[152] established clinical CVD (coronary heart disease, cerebrovascular disease, peripheral arterial disease, or heart failure)[153–158] concomitant HMOD (see *Figure 7*; *Section 7*; Supplementary data online, *Table S1*),[31,159] diabetes mellitus, and familial hypercholesterolaemia (probable or definite).[160–163] Regarding diabetes, some adults aged <60 years with type 2 diabetes and elevated BP have 10-year CVD risk of <10%. Accordingly, the diabetes-specific

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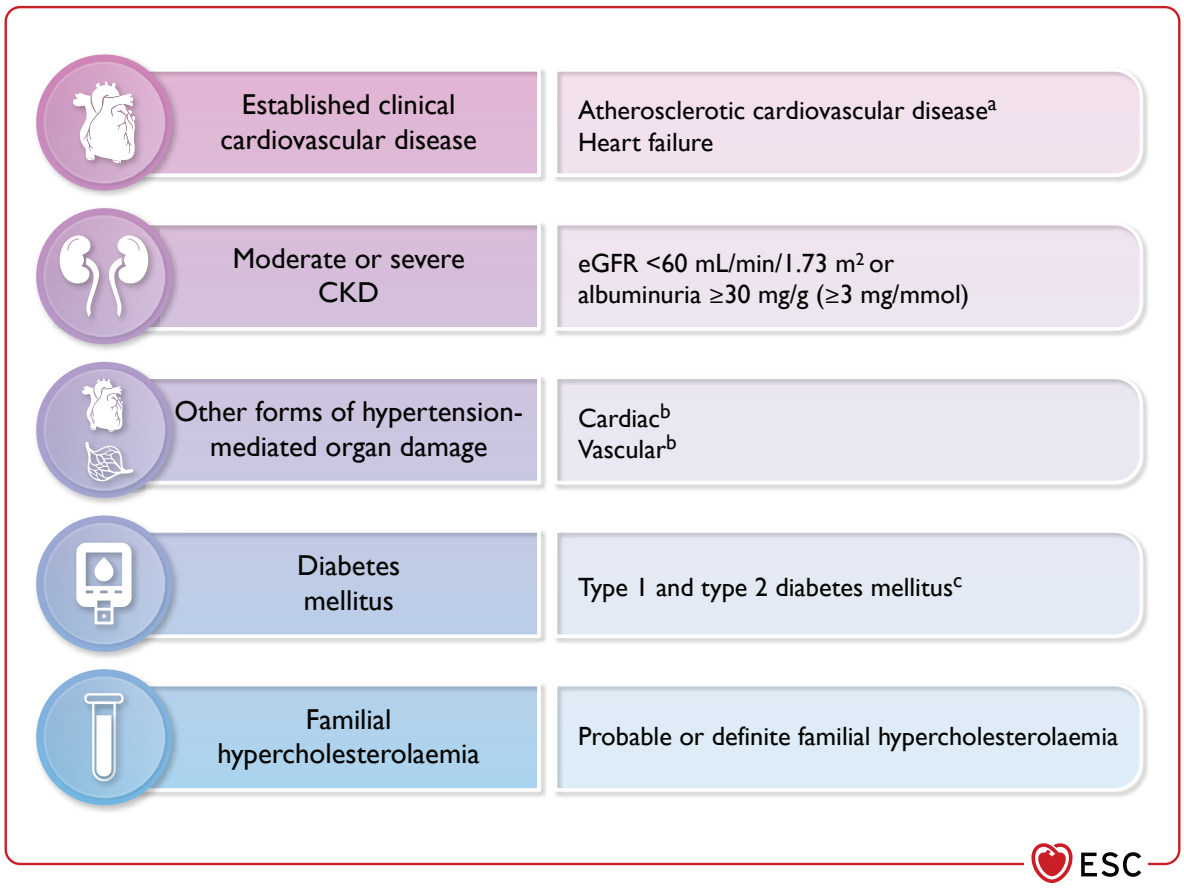

Figure 7 Sufficiently high cardiovascular risk conditions that warrant blood pressure-lowering treatment among adults with elevated blood pressure. CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate. [a]Coronary heart disease, cerebrovascular disease, peripheral arterial disease. [b]See *Section 7*. [c]SCORE2-Diabetes should be considered to identify lower-risk individuals (<10% 10-year CVD risk), who may not require BP-lowering medication, particularly in individuals <60 years.

Systematic COronary Risk Evaluation 2 (SCORE2)-Diabetes risk-prediction model should be considered to confirm CVD risk is sufficiently high (≥10%) among individuals with type 2 diabetes mellitus who are aged <60 years.[164]

In the absence of these sufficiently high-risk conditions, risk-prediction models (SCORE2 and SCORE-OP) have been developed in the general population to predict 10-year risk of CVD.[165,166] In adults with elevated BP without the above sufficiently high-risk conditions, risk-prediction models are recommended to inform BP-lowering treatment decisions. Risk-prediction models are more accurate than clinical judgment or tallying of individual risk factors.[167–169]

6.3.1. 10-year cardiovascular disease risk-prediction models

Prediction models differ in their input variables, predicted endpoints (outputs), and populations in which they were derived and validated. We endorse the use of SCORE2 for individuals aged 40–69 years and SCORE2–Older Persons (SCORE2-OP) for individuals aged ≥70 years for predicting 10-year global risk of fatal and non-fatal CVD events (stroke or myocardial infarction).[165,166] The management of adults aged <40 years is discussed in *Section 9.1*. The SCORE2 and SCORE2-OP models are preferred over other 10-year risk-prediction models, as they predict both fatal and non-fatal CVD events, have been validated and recalibrated to European populations, and because SCORE2-OP is adjusted for the competing risk of non-cardiovascular mortality. Calculating SCORE2 or SCORE2-OP is recommended for individuals with elevated BP who are not already at sufficiently high CVD risk due to established CVD, moderate or severe CKD, probable or definite familial hypercholesterolaemia, diabetes mellitus, or HMOD.[165,166,170]

For the purpose of BP-lowering treatment decisions, individuals with elevated BP and a predicted 10-year CVD risk of ≥10% by SCORE2 or SCORE2-OP are considered in these guidelines to be sufficiently high risk, with details on the choice of lifestyle or drugs to facilitate BP-lowering treatment in this setting provided in *Section 8*.[171,172] A number of considerations influenced our choice to recommend a single risk threshold of ≥10%, vs. the alternative option of using age-specific risk thresholds, such as those provided in the 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice.[170] For example, contemporary data indicate the heightened importance of BP control in older adults due to their higher absolute CVD risk (resulting in a lower NNT) and concomitantly to reduce age-dependent adverse outcomes attributable to increased BP, such as dementia. Recent treat-to-target trials (testing systolic BP targets of approximately 120 mmHg) used a single CVD risk inclusion threshold and were also

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enriched with older adults.[135,136,146] In addition, the average CVD event rate in the control arm of a landmark meta-analysis showing the benefits of more intensive BP-lowering treatment was approximately equivalent to a 10% 10-year risk.[116] Finally, the task force, which included patient members, felt that age-specific risk thresholds could result in BP treatment decisions being made solely based on age, which is difficult to support scientifically or otherwise. To try to avoid any confusion with the 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice, we use the terms 'sufficiently high risk' or 'increased risk' to describe a person with 10-year CVD risk of ≥10% (rather than the terms 'high risk' or 'very high risk').

Recommendation Table 3 — Recommendations for assessing cardiovascular disease risk among individuals with elevated blood pressure (office systolic blood pressure 120–139 mmHg or diastolic blood pressure 70–89 mmHg) (see Evidence Tables 10 and 11)

Recommendations	Class[a]	Level[b]
It is recommended to use a risk-based approach in the treatment of elevated BP, and individuals with moderate or severe CKD, established CVD, HMOD, diabetes mellitus, or familial hypercholesterolaemia are considered at increased risk for CVD events.[31,153–159,161–163,172]	I	B
SCORE2 is recommended for assessing 10-year risk of fatal and non-fatal CVD among individuals aged 40–69 years with elevated BP who are not already considered at increased risk due to moderate or severe CKD, established CVD, HMOD, diabetes mellitus, or familial hypercholesterolaemia.[143,165,172]	I	B
SCORE2-OP is recommended for assessing the 10-year risk of fatal and non-fatal CVD among individuals aged ≥70 years with elevated BP who are not already considered at increased risk due to moderate or severe CKD, established CVD, HMOD, diabetes mellitus, or familial hypercholesterolaemia.[143,166,172]	I	B
It is recommended that, irrespective of age, individuals with elevated BP and a SCORE2 or SCORE2-OP CVD risk of ≥10% be considered at increased risk for CVD for the purposes of risk-based management of their elevated BP.[143,165,166,172]	I	B
SCORE2-Diabetes should be considered to estimate CVD risk among type 2 diabetes mellitus patients with elevated BP, particularly if they are <60 years of age.[164]	IIa	B

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BP, blood pressure; CKD, chronic kidney disease; CVD, cardiovascular disease; HMOD, hypertension-mediated organ damage; SCORE2, Systematic COronary Risk Evaluation 2; SCORE2-OP, Systematic COronary Risk Evaluation 2–Older Persons.
Established CVD: coronary artery disease, cerebrovascular disease, peripheral arterial disease, or heart failure. For details on HMOD see *Section 7*.
[a]Class of recommendation.
[b]Level of evidence.

6.4. Refining cardiovascular disease risk estimation beyond risk models

The SCORE2 and SCORE2-OP risk-prediction models incorporate traditional risk factors such as age, sex, systolic BP, cholesterol values, and smoking status to predict 10-year risk of CVD.[165,166] However, they do not include 'non-traditional' CVD risk factors (detailed below and hereafter termed 'risk modifiers'). Non-traditional CVD risk modifiers can improve the predictive performance (i.e. discrimination) of other CVD risk-prediction models, and may also apply to SCORE2 or SCORE2-OP.[173] For example, among individuals with elevated BP and borderline increased 10-year predicted CVD risk by SCORE2 or SCORE2-OP (estimates of 5% to <10%), these non-traditional CVD risk modifiers may help up-classify the patient's risk and thereby prompt BP-lowering treatment (*Figure 8*).

6.4.1. Sex-specific non-traditional cardiovascular disease risk modifiers

Sex differences in the distribution of traditional and non-traditional CVD risk factors have been documented among patients with hypertension.[174] Although sex itself is included as an input variable in the SCORE2 and SCORE2-OP, and though these models were derived separately in men and women, some sex-specific, non-traditional risk modifiers were not included, and their associated impact on CVD risk may not be fully captured by SCORE2, SCORE2-OP, or SCORE2-Diabetes.

The relationship between BP and overall CVD risk is similar in both sexes, though some studies even suggest a stronger relative risk for CVD for a given BP level among females compared with males.[117] Female-specific, non-traditional CVD risk modifiers often arise at specific times throughout the life course, especially during pregnancy and the peri-partum period. Women with a history of hypertensive disorders of pregnancy, including gestational hypertension and pre-eclampsia, have a two-fold higher long-term risk of CVD vs. women without these pregnancy conditions.[175–177] The relative long-term CVD risk associated with hypertensive disorders of pregnancy may also be higher in younger vs. older pregnant women.[178,179] Most, but not all, of the excess CVD risk associated with hypertensive disorders of pregnancy is captured by conventional CVD risk factors.[176,178] Gestational diabetes is independently associated with an approximately two-fold increase in the long-term relative risk of CVD events.[180] Other complications such as preterm delivery, recurrent miscarriage, and one or more stillbirths are associated with a 40% relative increase in long-term CVD risk.[181–185] Accordingly, a history of specific pregnancy complications, including gestational hypertension, pre-eclampsia, gestational diabetes, pre-term delivery, one or more stillbirths, and recurrent miscarriage, can be considered as non-traditional CVD risk modifiers to up-classify women with elevated BP and borderline increased 10-year predicted CVD risk (5% to <10%) to sufficiently high risk, thereby influencing the risk-based management of their elevated BP.

Evidence whether other female-specific conditions (infertility, polycystic ovary syndrome, and premature menopause) and male-specific conditions (androgenic alopecia and erectile dysfunction) improve prediction of CVD sufficiently to inform risk-based BP-lowering treatment decisions is inconclusive at present.

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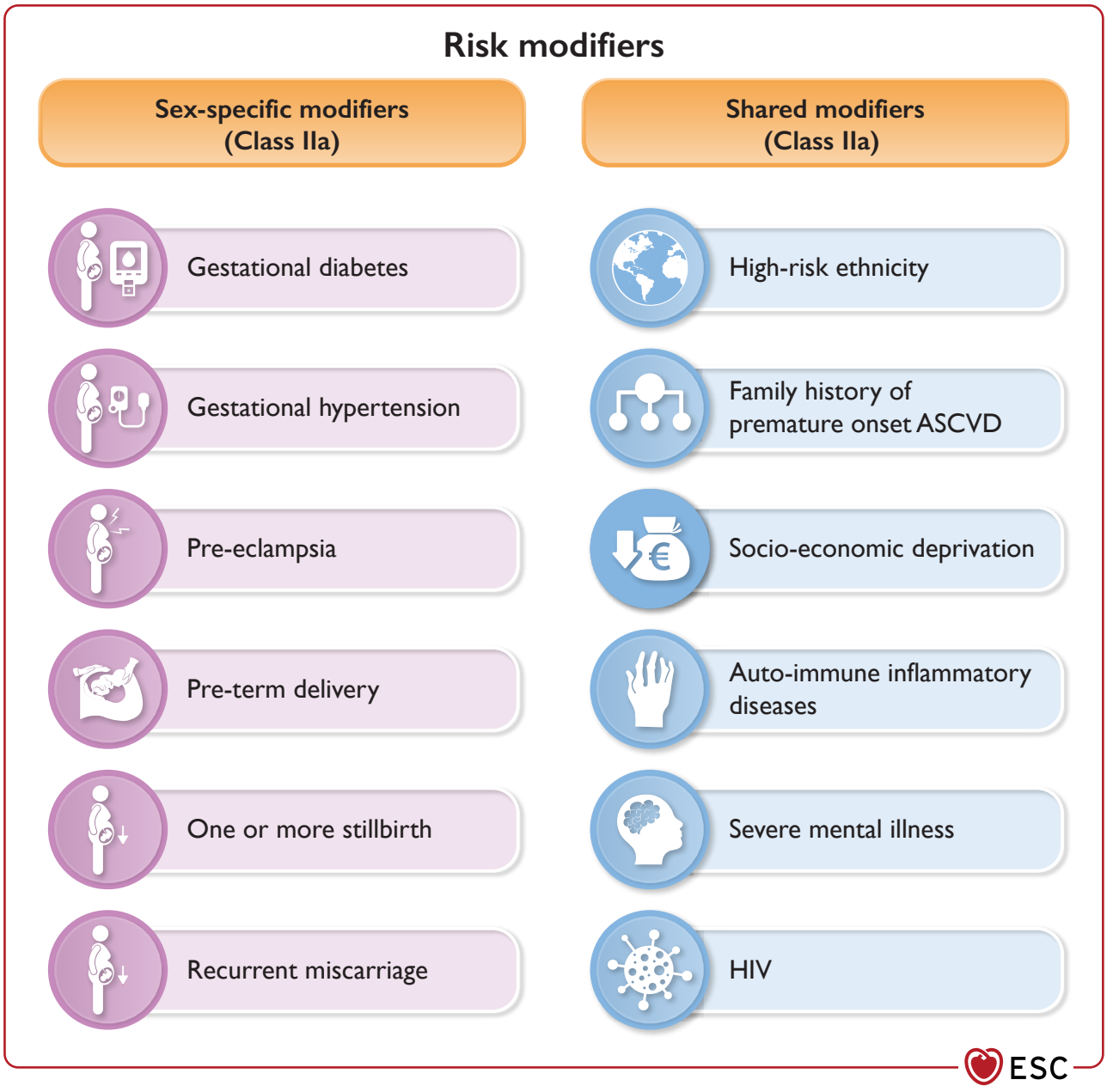

Figure 8 Cardiovascular disease risk modifiers to consider for up-classification of risk. ASCVD, atherosclerotic cardiovascular disease; HIV, human immunodeficiency virus.

6.4.2. Non-traditional cardiovascular disease risk modifiers shared by men and women

In addition to sex-specific risk modifiers, several other non-traditional risk factors are associated with an increased risk of CVD, but few have been shown to improve risk prediction or discrimination beyond traditional CVD risk factors.

We advise considering high-CVD-risk race/ethnicity (e.g. South Asian),[186–188] family history of premature onset atherosclerotic CVD (CVD event in males aged <55 years and/or females <65 years),[189,190] socio-economic deprivation,[191] inflammatory conditions (e.g. systemic lupus erythematosus, rheumatoid arthritis, and psoriasis affecting 10% or more of body surface area or requiring systemic therapy),[192–202] HIV,[203–205] and severe mental illness (major depressive disorder, bipolar disorder, and schizophrenia)[206–208] as shared non-traditional risk modifiers to up-classify the risk of individuals with a borderline increased 10-year predicted risk using SCORE2/SCORE2-OP (5% to <10%) to sufficiently high CVD risk.

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6.4.3. Additional risk decision tests

Coronary artery calcium (CAC) scoring improves CVD risk prediction and reclassifies risk when added to conventional CVD risk factor-based estimation models.[209,210] A CAC score of >100 Agatston units or ≥75th percentile for age, sex, and ethnicity favours up-classification of CVD risk.[127] Internal or external carotid plaque may also improve CVD risk prediction.[211] Similarly, femoral artery plaque detection may improve CVD risk prediction.[212–214] Arterial stiffness, as assessed by pulse wave velocity (PWV), is associated with increased risk of CVD events and improves CVD risk stratification.[215–218] Common arterial stiffness thresholds for increased risk include carotid–femoral PWV of >10 m/s and brachial–ankle PWV of >14 m/s. After assessing 10-year predicted CVD risk and non-traditional risk factors, if a risk-based treatment decision remains uncertain for patients with elevated BP, it is reasonable to measure a CAC score or, alternatively, carotid or femoral plaque, or arterial stiffness; most especially after shared decision-making with the patient and after considering cost (see *Section 7* for more details on these tests). There is also evidence that elevated cardiac biomarker levels (specifically high-sensitivity cardiac troponin and B-type natriuretic peptide/N-terminus B-type natriuretic peptide) are significant and effective risk modifiers,[219,220] with further supportive data from hypertensive participants.[159,221,222] Of note, these cardiac biomarkers can be considered markers of HMOD (*Section 7*); however, we focus on them in this risk modifier section because they may be elevated due to other reasons besides high BP (such as atherosclerosis or heart rhythm disease).

Recommendation Table 4 — Recommendations for refining cardiovascular disease risk (see Evidence Tables 12–14)

Recommendation	Class[a]	Level[b]
History of pregnancy complications (gestational diabetes, gestational hypertension, pre-term delivery, pre-eclampsia, one or more stillbirths, and recurrent miscarriage) are sex-specific risk modifiers that should be considered to up-classify individuals with elevated BP and borderline increased 10-year CVD risk (5% to <10% risk).[183,184,223,224]	IIa	B
High-risk ethnicity (e.g. South Asian), family history of premature onset atherosclerotic CVD, socio-economic deprivation, auto-immune inflammatory disorders, HIV, and severe mental illness are risk modifiers shared by both sexes that should be considered to up-classify individuals with elevated BP and borderline increased 10-year CVD risk (5% to <10% risk).[186–191,193,198,202,204,208]	IIa	B
After assessing 10-year predicted CVD risk and non-traditional CVD risk modifiers, if a risk-based BP-lowering treatment decision remains uncertain for individuals with elevated BP, measuring CAC score, carotid or femoral plaque using ultrasound, high-sensitivity cardiac troponin or B-type natriuretic peptide biomarkers, or arterial stiffness using pulse wave velocity, may be considered to improve risk stratification among patients with borderline increased 10-year CVD risk (5% to <10% risk) after shared decision-making and considering costs.[209–211,215,218,225,226]	IIb	B

BP, blood pressure; CAC, coronary artery calcium; CVD, cardiovascular disease; HIV, human immunodeficiency virus.
[a]Class of recommendation.
[b]Level of evidence.

6.5. Summary of the cardiovascular disease risk stratification approach for allocating blood pressure treatment

Measured BP combined with 10-year CVD risk-prediction models and non-traditional risk modifiers should be used for stratifying risk when allocating BP-lowering treatment for persons with elevated BP (*Figure 9*). It is important to stress here that patients with confirmed hypertension are recommended to receive BP-lowering treatment and no further risk stratification is needed.

For patients with elevated BP, the presence of diabetes, familial hypercholesterolaemia, established CVD (defined as prior acute or chronic coronary syndrome, cerebrovascular disease, symptomatic peripheral arterial disease, or heart failure), moderate or severe CKD, or HMOD confers increased CVD risk. One caveat is that, specifically for individuals with elevated BP and type 2 diabetes mellitus only aged <60 years, SCORE2-Diabetes should be considered to identify lower CVD risk individuals (<10% over 10 years).

Otherwise, for patients without these high-risk conditions, 10-year risk of CVD should be calculated using SCORE2 (if aged 40–69 years) and SCORE2-OP (if aged ≥70 years). Patients with elevated BP and a 10-year predicted risk of CVD events ≥10% are considered sufficiently high risk to warrant BP-lowering treatment (either by lifestyle or drug treatment, see *Section 8*). For patients with elevated BP and borderline increased predicted CVD risk by SCORE2/SCORE2-OP (5% to <10% over 10 years), up-classification of risk may be considered in the presence of sex-specific or shared non-traditional risk modifiers. After considering sex-specific and shared non-traditional risk modifiers, if a risk-based BP-lowering treatment decision remains uncertain, it may be reasonable to measure CAC score, carotid or femoral plaque, high-sensitivity cardiac troponin or B-type natriuretic peptide biomarkers, or arterial stiffness.

Risk stratification for patients with non-elevated BP (systolic BP of <120 mmHg and diastolic BP of <70 mmHg) is not required for the purpose of allocating BP-lowering treatment, as the safety and efficacy of commencing BP-lowering treatment below this threshold is uncertain. Risk assessment may nonetheless be needed in this setting when considering other prevention therapies (e.g. lipid lowering).

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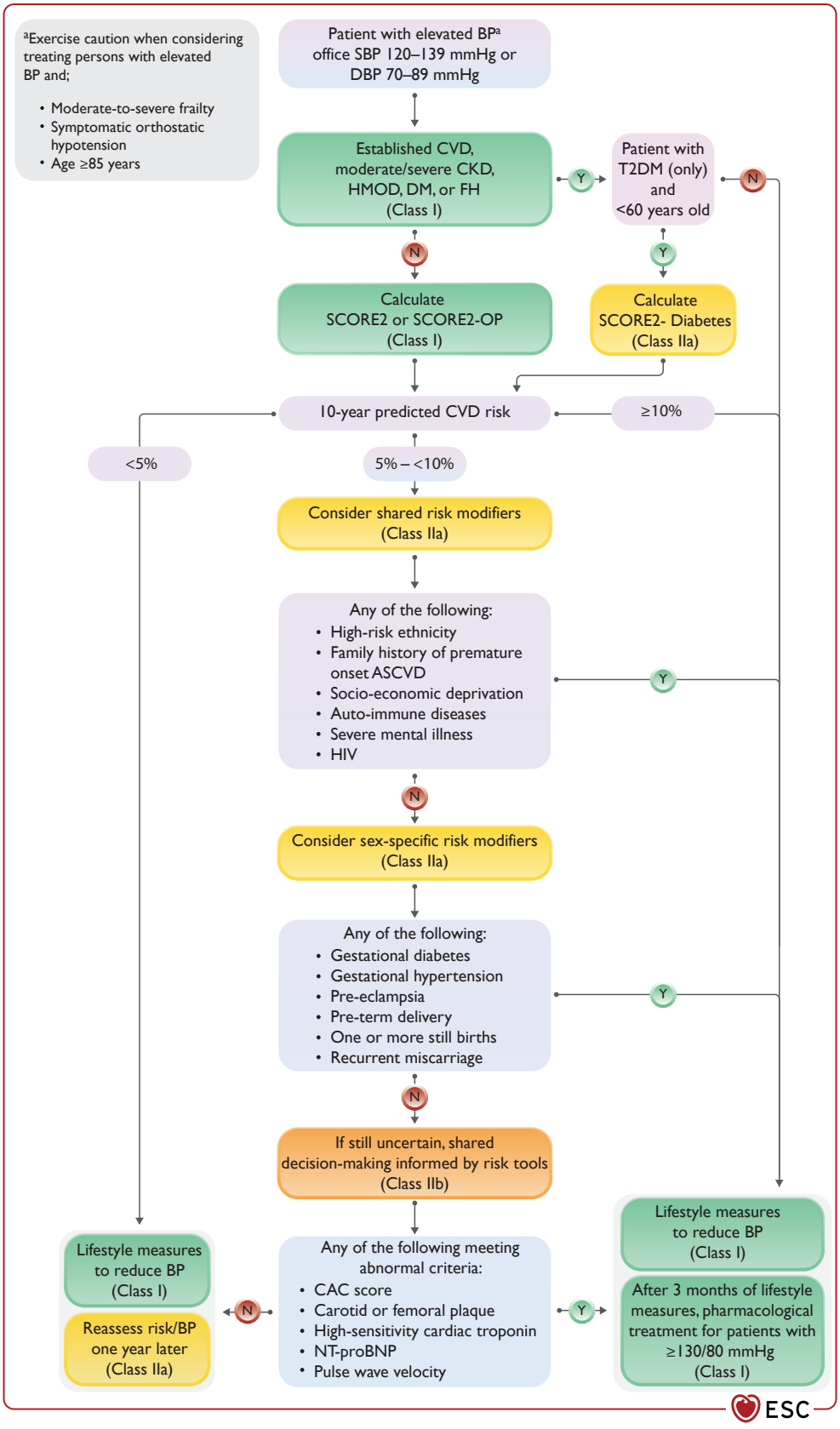

Figure 9 Summary of cardiovascular disease risk-stratification approach for blood pressure treatment in adults with elevated blood pressure. ASCVD, atherosclerotic cardiovascular disease; BP, blood pressure; CAC, coronary artery calcium; CKD, chronic kidney disease; CVD, cardiovascular disease; DBP, diastolic blood pressure; DM, diabetes mellitus; FH, familial hypercholesterolaemia; HMOD, hypertension-mediated organ damage; NT-proBNP, N-terminal pro-brain natriuretic peptide; SBP, systolic blood pressure; SCORE2, Systematic COronary Risk Evaluation 2; SCORE2-OP, Systematic COronary Risk Evaluation 2–Older Persons.

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7. Diagnosing hypertension and investigating underlying causes

7.1. Screening for hypertension

Hypertension is predominantly an asymptomatic condition that is typically detected by systematic or opportunistic screening in a healthcare setting. Systematic screening refers to any process where individuals are identified and invited to a healthcare setting solely to measure their BP and CVD risk profile. Opportunistic screening refers to BP being measured when the patient presents to a healthcare setting for any reason, such as a routine check-up or the treatment of an acute or chronic condition. Self-screening and non-physician screening are also increasingly used.[227–230]

Few data are available on the effectiveness of different hypertension screening strategies to reduce the morbidity and mortality associated with hypertension.[231–233] More evidence is needed before systematic screening programmes with BP measurement can be recommended in all adults to reduce CVD events.[231]

Opportunistic BP screening in a primary care setting appears effective, with an estimated 90% of all adults aged >40 years in the UK having a BP check within a 5-year time period,[234] though these findings may not extrapolate to other countries. When patients provide HBPM recordings, these can also be used as part of an opportunistic screening programme (see *Section 5.2*).[235]

Despite ongoing uncertainty about the effect of hypertension screening programmes on CVD outcomes, many studies have demonstrated that screening (mostly opportunistic screening) increases hypertension detection, and that the benefits of screening likely outweigh harms.[70] Global initiatives to raise BP awareness, such as the May Measurement Month,[228] or targeted initiatives, such as the barbershop health outreach programmes,[229] are successful examples of BP screening campaigns.

Screening for hypertension, like for global CVD risk assessment, should be intermittently repeated, e.g. every 3 years. Considering the rate of progression to hypertension in European population samples,[236] it is reasonable to measure BP at least every 3 years in the case of non-elevated BP and low–moderate CVD risk (i.e. individuals aged <40 years). More frequent BP checks (i.e. yearly) should be considered in individuals 40 years or older and individuals with elevated BP not currently meeting indications for treatment[170] (*Figure 10*).

Recommendation Table 5 — Recommendations for blood pressure screening (see Evidence Table 15)

Recommendation	Class[a]	Level[b]
Opportunistic screening for elevated BP and hypertension should be considered at least every 3 years for adults aged <40 years.[236,237]	IIa	C
Opportunistic screening for elevated BP and hypertension should be considered at least annually for adults aged ≥40 years.[231,237]	IIa	C
In individuals with elevated BP who do not currently meet risk thresholds for BP-lowering treatment, a repeat BP measurement and risk assessment within 1 year should be considered.	IIa	C
Other forms of screening for hypertension (i.e. systematic screening, self-screening, and non-physician screening) may be considered, depending on their feasibility in different countries and healthcare systems.[231–233]	IIb	B

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BP, blood pressure.
[a]Class of recommendation.
[b]Level of evidence.

7.2. Confirming the diagnosis of hypertension

As noted in *Section 5*, assessment at a single visit by office BP has lower specificity compared with ABPM for diagnosing hypertension.[70,238–241] Accordingly, a protocol for confirming the diagnosis of hypertension is proposed (*Figure 10*), with out-of-office BP measurement as the preferred method for confirming cases of elevated BP or hypertension. For initial screening systolic BP of >160 mmHg and/or diastolic BP of >100 mmHg, a prompt re-evaluation (within days to weeks but not >1 month) preferably with ABPM or HBPM is advisable.[71] BP of >180/110 mmHg at screening requires exclusion of hypertensive emergencies, which should be managed as appropriate (see [242] and *Section 10*) with prompt treatment. For individuals with BP of >180/110 mmHg at screening but without hypertensive emergency, prompt confirmation (preferably within a week) can be considered prior to commencing treatment.

Recommendation Table 6 — Recommendations for confirming hypertension diagnosis

Recommendations	Class[a]	Level[b]
In individuals with increased CVD risk where their screening office BP is 120–139/70–89 mmHg, it is recommended to measure BP out of office, using ABPM and/or HBPM or, if not logistically feasible, by making repeated office BP measurements on more than one visit.[70,238–241]	I	B
Where screening office BP is 140–159/90–99 mmHg, it is recommended that the diagnosis of hypertension should be based on out-of-office BP measurement with ABPM and/or HBPM. If these measurements are not logistically or economically feasible, then diagnosis can be made on repeated office BP measurements on more than one visit.[70,238–241]	I	B
Where screening office BP is ≥160/100 mmHg: • It is recommended that BP 160–179/100–109 mmHg be confirmed as soon as possible (e.g. within 1 month) preferably by either home or ambulatory BP measurements; • It is recommended when BP ≥180/110 mmHg that hypertensive emergency be excluded.	I	C

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ABPM, ambulatory blood pressure measurement; BP, blood pressure; CVD, cardiovascular disease; HBPM, home blood pressure measurement.
[a]Class of recommendation.
[b]Level of evidence.

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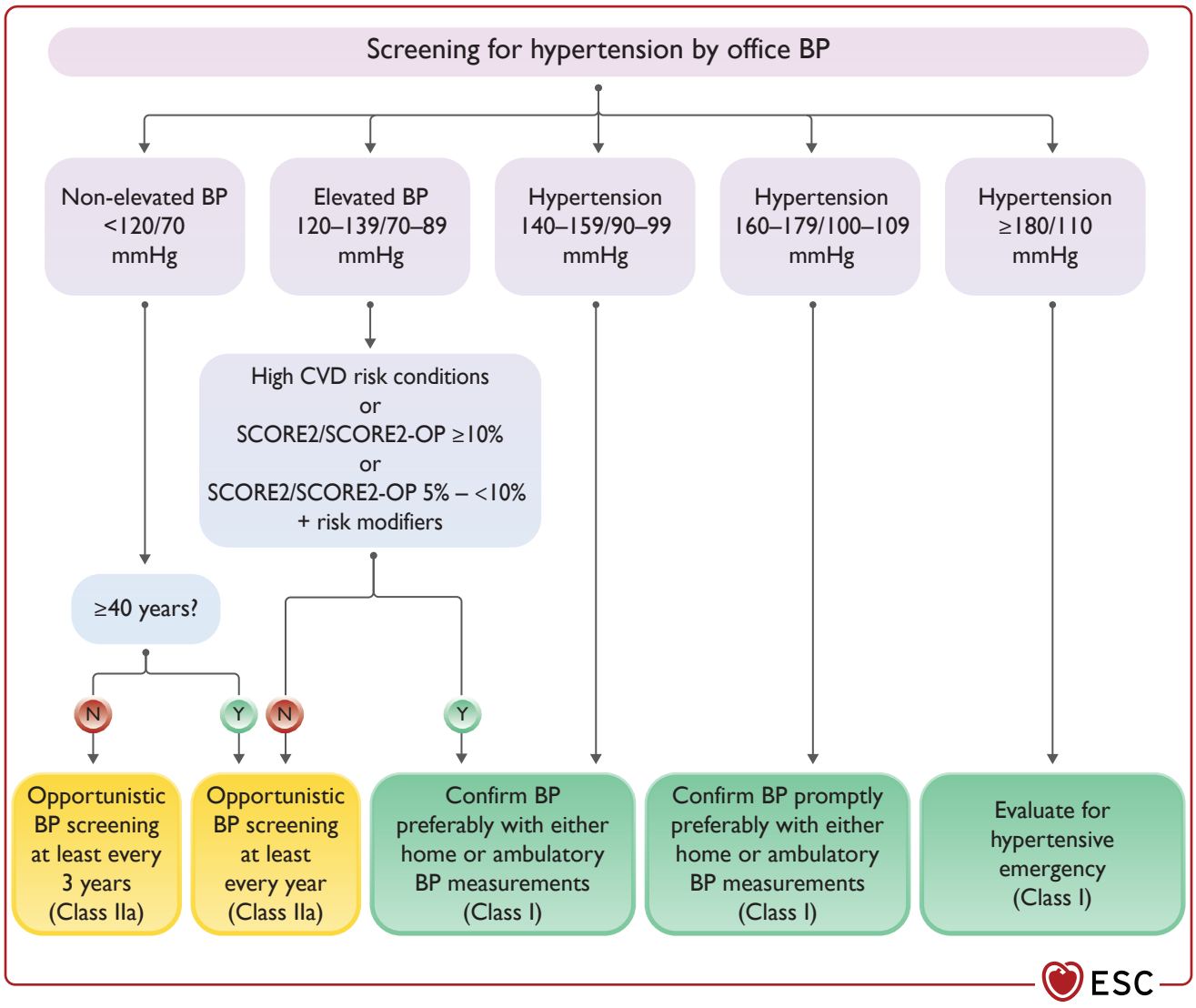

Figure 10 Protocol for confirming hypertension diagnosis. BP, blood pressure; CVD, cardiovascular disease; SCORE2, Systematic COronary Risk Evaluation 2; SCORE2-OP, Systematic COronary Risk Evaluation 2–Older Persons.

7.3. Communicating the diagnosis

Behavioural responses to health-related threats are strongly influenced by five core themes (termed 'illness representations'), which are identity, timeline, cause, consequences, and control/cure.[243,244] These illness representations form the basis of how patients understand a diagnosis, and can influence their responses after being diagnosed with hypertension.[243] This conceptual framework can help guide the clinical communication of a diagnosis of hypertension. For example, patients' understanding of the chronic nature of hypertension (i.e. timeline theme) is key for ensuring long-term engagement with medical treatment.[245] Prior to commencing treatment, it is helpful to understand the extent to which patients believe that medications are necessary and ascertain if they have concerns.[246] The core illness representations and beliefs about medicines for clinicians to consider are included in *Table 7*.

The 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice recommend "an informed discussion about CVD risk and treatment benefits—tailored to the needs of a patient" as part of a diagnosis communication in hypertension.[170] This can be facilitated using an interdisciplinary healthcare-provided approach (see *Section 11*) and by visual information or other more accessible material that might optimally communicate hypertension-related risk.[128] Visualizing risk by medical imaging to motivate risk-reducing behaviour changes may also be beneficial.[247]

7.4. Baseline assessment and diagnostic approach

7.4.1. Medical history, medication history, and physical examination

The purpose of clinical evaluation is to diagnose hypertension, delineate factors potentially contributing to hypertension, identify other CVD risk factors, define relevant comorbidities, screen for potential secondary causes of hypertension (where indicated), and establish whether

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Table 7 Key illness representations and treatment beliefs: how these apply to communicating a hypertension diagnosis to the patient (note that gender influences these representations)

Illness representation	Example patient question	Application to a hypertension diagnosis conversation
Identity	What is the disease/illness label and the related symptoms?	The condition where your systolic BP is ≥140 and/or diastolic BP ≥90 mmHg is called hypertension. We classify systolic BP 120–139 or diastolic BP 70–89 mmHg as elevated BP. For most people, this has no noticeable signs or symptoms, therefore, we need to monitor your BP to assess how medications and behavioural changes are working.
Control	Is the illness controllable through medical intervention or behavioural change?	Hypertension can usually be controlled with medication and behavioural changes such as dietary changes and regular physical activity. For some people we need to try a few different options before we get BP under control.
Timeline	Is this an acute or chronic problem?	This is a serious long-term or chronic condition condition that will require long-term management. This means that it may need to be managed throughout life.
Consequences	What are the physical and psychosocial consequences?	If hypertension is not controlled, then there is a risk of a serious acute cardiovascular disease event such as a stroke or heart attack; however, if it is managed through the right medical intervention and behavioural changes, then this risk can be reduced and the condition will have less consequences for your life.
Causes	What caused the condition?	Multiple factors contribute to someone developing hypertension. These include both non-modifiable factors (e.g. genetics and age) and modifiable factors (e.g. diet, weight, and physical activity). We are best focusing on those things that we can control to reduce your BP.
Treatment beliefs	**Example patient question**	**Application to a hypertension diagnosis communication**
Necessity	To what extent is treatment necessary?	Taking BP-lowering medication every day is necessary to keep your BP under control and to help prevent a more serious health problem developing. Do you think that these medicines will help you?
Concerns	To what extent does treatment cause concern?	Some patients have concerns about taking daily medications throughout their life, e.g. about side effects. Do you have any concerns about taking your BP medications every day?

BP, blood pressure.

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there is evidence of HMOD or existing cardiac, cerebrovascular, or renal disease.

Details on medical history and physical examination steps are summarized in the supplement (see Supplementary data online, *Tables S2 and S3*), as well as drugs or substances that may increase BP (see Supplementary data online, *Table S4*).

7.4.2. Drug adherence and persistence with treatment

Adherence is defined as the extent to which a patient's behaviour, e.g. with respect to taking medication, coincides with agreed recommendations from a healthcare provider. Persistence represents the amount of time from initiation to discontinuation of therapy.[248] Adherence to medical therapies is especially suboptimal in asymptomatic conditions such as hypertension.[249–254] Non-adherence to BP-lowering therapy correlates with a higher risk of CVD events.[255,256] Objective methods to assess adherence, such as detecting prescribed drugs in blood or urine samples and directly observed treatment (witnessed pill intake during ABPM), have demonstrated their potential usefulness, particularly in the setting of apparently resistant hypertension.[257] However, all methods for testing drug adherence have limitations.

Non-adherence to BP-lowering therapy depends on many factors (*Figure 11*).[253] Effective patient–physician communication is crucial to improve adherence.[258,259] Single-pill combinations improve persistence in BP-lowering treatment and are associated with lower all-cause mortality.[260]

Recommendation Table 7 — Recommendations for assessing adherence and persistence with treatment (see Evidence Table 16)

Recommendation	Class[a]	Level[b]
Objective evaluation of adherence (either directly observed treatment or detecting prescribed drugs in blood or urine samples) should be considered in the clinical work-up of patients with apparent resistant hypertension, if resources allow.[261–263]	IIa	B

[a]Class of recommendation.
[b]Level of evidence.

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7.4.3. Routine and optional tests

Routine tests include laboratory and clinical tests to detect increased CVD risk and relevant comorbidities (e.g. hyperlipidaemia and diabetes) (*Table 8*).

Optional tests should be considered in the initial assessment if they are likely to change patient management, with the main rationale being to improve CVD risk stratification.[170] As highlighted in *Section 6*, for adults with elevated BP who also have a 10-year estimated CVD risk of 5% to <10%, optional tests including those for HMOD may be considered if up-classification of risk on the basis of an abnormal test result could prompt initiation of BP-lowering therapy.[31,170] Evidence of subclinical microvascular neurodegeneration and/or lacunar brain disease due to small-vessel pathology may also indicate HMOD.[264]

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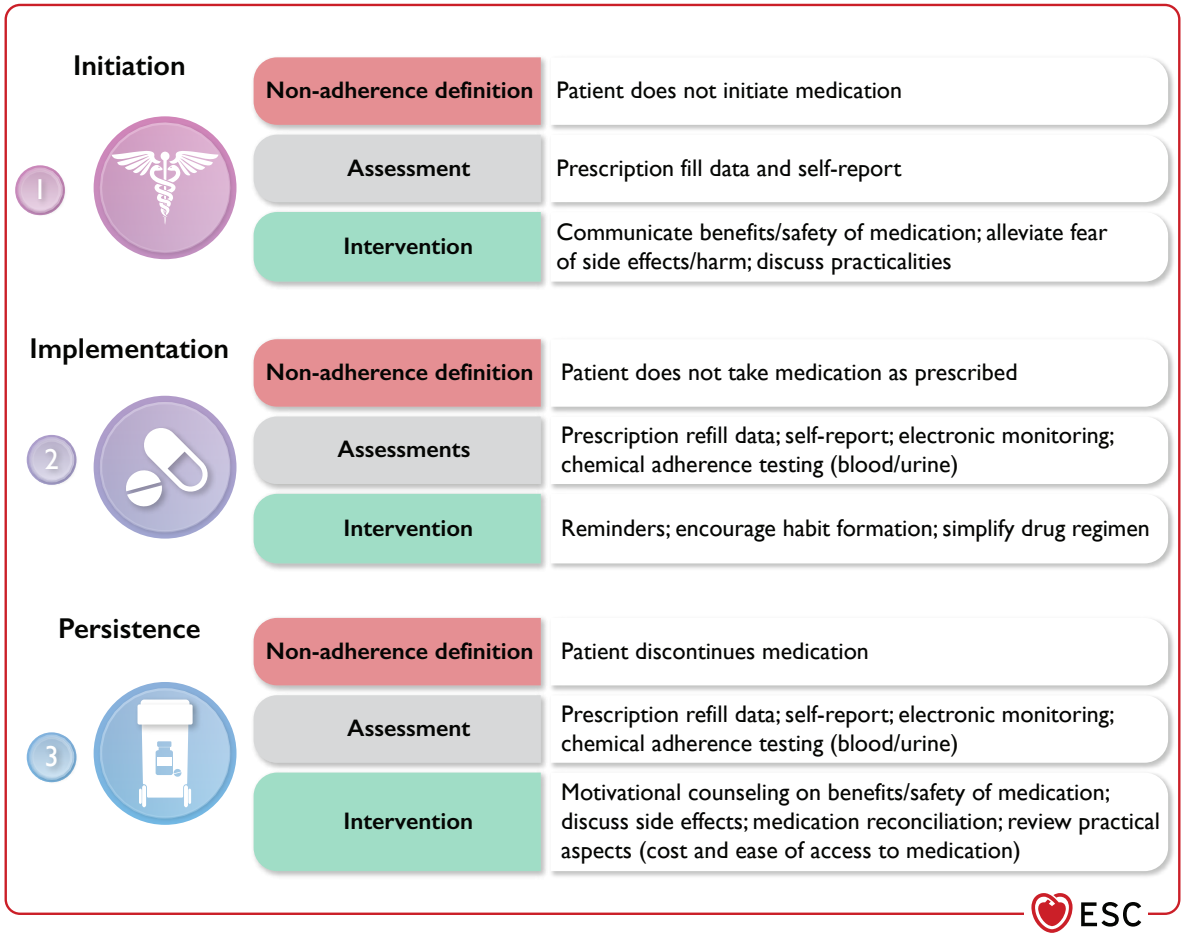

Figure 11 Definitions, assessments, and potential interventions for the three phases of adherence to BP-lowering medications.

Table 8 Routine tests recommended in the initial work-up of a patient with elevated blood pressure or hypertension

Routine test	Clinical utility
Fasting blood glucose (and HbA1c if fasting blood glucose is elevated)	Assessing CVD risk and comorbidities
Serum lipids: total cholesterol, LDL cholesterol, HDL and non-HDL cholesterol, triglycerides	Assessing CVD risk
Blood sodium and potassium, haemoglobin and/or haematocrit, calcium, and TSH	Screening secondary hypertension (primary aldosteronism, Cushing's disease, polycythaemia, hyperparathyroidism, and hyperthyroidism)
Blood creatinine and eGFR; urinalysis and urinary albumin-to-creatinine ratio	Assessing CVD risk and HMOD Guiding treatment choice Screening secondary hypertension (renoparenchymal and renovascular)
12-lead ECG	Assessing HMOD (left atrial enlargement, left ventricular hypertrophy) Assessing irregular pulse and other comorbidities (AF, previous acute myocardial infarction)

AF, atrial fibrillation; CVD, cardiovascular disease; ECG, electrocardiogram; eGFR, estimated glomerular filtration rate; HbA1c, glycated haemoglobin; HDL, high-density lipoprotein; HMOD, hypertension-mediated organ damage; LDL, low-density lipoprotein; TSH, thyroid-stimulating hormone.

While the role of optional tests for HMOD (*Table 9*) in the management of elevated BP is emphasized in these guidelines, we also note that these tests may help to optimize treatment in hypertensive adults with BP of >140/90 mmHg who are prescribed BP-lowering therapy (e.g. by facilitating patient adherence and overcoming clinician inertia in achieving an intensive BP treatment target of as low as 120 mmHg systolic). The role of visualizing HMOD in helping motivate risk-reducing changes in patients and overcome physician inertia has been tested in interventional trials (*Section 7.3*).[247,265–267]

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Table 9 Optional tests that may be used as clinically indicated in the initial work-up of a patient with elevated blood pressure or hypertension to assess hypertension-mediated organ damage or established cardiovascular disease

Optional test	Clinical utility
Echocardiography	Assessing HMOD (hypertensive heart disease) Assessing established CVD (previous acute myocardial infarction, heart failure) Assessing thoracic aorta dilation
CAC by cardiac CT or carotid or femoral artery ultrasound imaging	Assessing HMOD (atherosclerotic plaque)
Large artery stiffness (carotid–femoral or brachial–ankle PWV)	Assessing HMOD (arterial stiffness)
High-sensitivity cardiac troponin and/or NT-proBNP	Assessing HMOD
Ankle–brachial index	Assessing established CVD (lower-extremity arterial disease)
Abdominal ultrasound	Assessing established CVD (abdominal aneurysm)
Fundoscopy	Assessing HMOD (hypertensive retinopathy) Diagnosing hypertensive emergency/malignant hypertension (haemorrhages and exudates, papilloedema)

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CAC, coronary artery calcium; CT, computed tomography; CVD, cardiovascular disease; HMOD, hypertension-mediated organ damage; NT-proBNP, N-terminal pro-brain natriuretic peptide; PWV, pulse wave velocity.

HMOD assessment is also an important way to identify young adults <40 years old who have increased CVD risk, since 10-year estimated CVD risk by SCORE2 cannot be calculated in this age group (see *Section 8.1*). More details on diagnostic thresholds for HMOD by the various assessment options, including important sex differences, are provided in Supplementary data online, *Tables S1 and S5* and *Figure 12*.

Finally, some individuals may be at heightened risk for CVD events when cardiac and vascular HMOD measurements like LVH and increased PWV do not regress over time with appropriate treatment and BP control.[14,268–271]

Investigations aimed at screening for secondary hypertension are additional optional tests and are detailed in *Section 7.6*. Of note, patients with an incidental adrenal nodule or nodules (typically detected on imaging of the abdomen done for other clinical reasons) warrant screening for elevated BP and hypertension. Those with adrenal incidentalomas and hypertension warrant a basic work-up for secondary hypertension to include screening for primary aldosteronism, Cushing's syndrome and phaeochromocytoma.

7.4.3.1. The kidneys

CKD is defined as abnormalities of kidney structure or function, present for at least 3 months with implications for health.[272] Renal function is evaluated initially using serum creatinine and an estimated glomerular filtration rate (eGFR) equation (preferably race-free CKD-EPI) and typically for proteinuria.[273] Our definition of moderate-to-severe CKD requires an eGFR of <60 mL/min/1.73 m^2 or albuminuria of ≥30 mg/g (≥3 mg/mmol). Intensive BP control in patients with CKD reduces rates of CVD events.[274,275] CKD can influence the choice of BP-lowering treatment (*Sections 8* and *9*), as well as newer drugs for cardiovascular prevention, such as sodium–glucose co-transporter 2 (SGLT2) inhibitors and finerenone.

We recommend repeat measurement of eGFR and urine albumin: creatinine ratio (ACR) at least annually if clinically significant CKD is diagnosed. Renal ultrasound and Doppler examination to evaluate causes of CKD and to exclude renoparenchymal and renovascular hypertension (RVH) should also be considered.[276,277]

Recommendation Table 8 — Recommendations for assessing renal hypertension-mediated organ damage

Recommendation	Class[a]	Level[b]
It is recommended to measure serum creatinine, eGFR, and urine ACR in all patients with hypertension.[170,273]	I	A
If moderate-to-severe CKD is diagnosed, it is recommended to repeat measurements of serum creatinine, eGFR, and urine ACR at least annually.[276]	I	C
Renal ultrasound and Doppler examination should be considered in hypertensive patients with CKD to assess kidney structure and determine causes of CKD and to exclude renoparenchymal and renovascular hypertension.[276,277] CT or magnetic resonance renal angiography are alternative testing options.	IIa	C

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ACR, albumin:creatinine ratio; CKD, chronic kidney disease; CT, computed tomography; eGFR, estimated glomerular filtration rate.
[a]Class of recommendation.
[b]Level of evidence.

7.4.3.2. The heart

A 12-lead ECG is a part of the initial routine work-up for all patients with hypertension and should be repeated whenever patients present with an irregular pulse or cardiac symptoms. The ECG should be analysed for LVH (Supplementary data online, *Table S1*) and AF.[31,278–282]

Echocardiography is recommended in patients with hypertension when the ECG is abnormal, murmurs are detected, or there are cardiac symptoms. A full, standardized, two-dimensional echocardiogram should be performed, preferably with tissue Doppler and strain assessment. Echocardiography can be considered for all patients with newly diagnosed hypertension, if local resources and reimbursement policies allow. Over 5 years of follow-up, subclinical left ventricular diastolic dysfunction predicts the incidence of CVD.[283–285] In addition, LVH detected by echocardiography predicts total and cardiovascular mortality and CVD events in the general population,[286,287] including in young adults.[26,268,288,289] Data on the associations with CVD of other metrics for detecting HMOD in the heart are also available.[26,268,290] Since cardiac size and function differ by sex, sex-specific thresholds for detecting HMOD in the heart are used to avoid under-diagnosis in women.[25,174,291]

Recommendation Table 9 — Recommendations for assessing cardiac hypertension-mediated organ damage

Recommendation	Class[a]	Level[b]
A 12-lead ECG is recommended for all patients with hypertension.[31,281]	I	B

Continued

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Echocardiography is recommended in patients with hypertension and ECG abnormalities, or signs or symptoms of cardiac disease.[14,31,292]	I	B
Echocardiography may be considered in patients with elevated BP, particularly when it is likely to change patient management.[31,291]	IIb	B

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BP, blood pressure; ECG, electrocardiogram.
[a]Class of recommendation.
[b]Level of evidence.

7.4.3.3. The arteries

Cardiac computed tomography (CT) may be used to measure CAC and, if intravascular contrast is administered, fully visualize coronary artery disease to improve risk stratification.[211,293] As noted in *Section 6*, CAC scoring can reclassify CVD risk upwards or downwards in addition to conventional risk factors.[127,170,211,233,294] Carotid ultrasound detects presence or absence of carotid plaque (wall thickness ≥ 1.5 mm) and stenosis. Presence of plaque in the carotid or femoral arteries improves risk prediction for CVD events in asymptomatic patients on top of conventional risk-factor assessment.[211,247,265,267,295,296] Systematic use of intima media thickness does not appear to consistently improve prediction of future CVD events.[297] Arterial stiffness is measured as carotid–femoral PWV or brachial–ankle PWV, and can contribute to predictive value and risk reclassification.[28,31,215,216] PWV is currently used mostly for research purposes or in specialist referral centres. Checking for inter-arm BP difference may identify a subclavian stenosis as vascular HMOD.[38] Other tests assessing the vasculature [such as abdominal ultrasound or ankle–brachial index (ABI)] should also be considered in patients with hypertension, when specific cardiovascular complications (abdominal aneurysm, peripheral artery disease) are clinically suspected. Finally, microvascular HMOD can be assessed by fundoscopy. A simplified classification has been proposed and validated.[298] In hypertensive individuals, the presence of mild or moderate hypertensive retinopathy is associated with an increased risk of CVD events.[299] Fundoscopy is recommended also in hypertensive diabetic patients and in the work-up of malignant hypertension and hypertensive emergencies.

Recommendation Table 10 — Recommendations for assessing vascular hypertension-mediated organ damage (see Evidence Table 17)

Recommendations	Class[a]	Level[b]
Fundoscopy is recommended if BP >180/110 mmHg in the work-up of hypertensive emergency and malignant hypertension, as well as in hypertensive patients with diabetes.	I	C
Fundoscopy for detecting hypertensive retinopathy may be considered in patients with elevated BP or hypertension.[299]	IIb	B
Ultrasound examination of the carotid or femoral arteries for detecting plaque may be considered in patients with elevated BP or hypertension when it is likely to change patient management.[211]	IIb	B
Coronary artery calcium scoring may be considered in patients with elevated BP or hypertension when it is likely to change patient management.[127,211]	IIb	B
Measurement of PWV may be considered in patients with elevated BP or hypertension when it is likely to change patient management.[28,31,215,216]	IIb	B

Continued

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BP, blood pressure; PWV, pulse wave velocity.
[a]Class of recommendation.
[b]Level of recommendation.

7.4.4. Genetic testing

Hypertension is considered a complex polygenic disorder, because many genes or gene combinations influence BP.[300,301] However, some well-defined phenotypes relating to single-gene mutations (i.e. monogenic forms of hypertension) have been identified (see Supplementary data online, *Table S6*). These are rare, but knowledge of the genetic defect may allow targeted treatment of the proband and also proper management of the patient's siblings.[302,303] As such, genetic testing should be considered only for those with a high prior probability of having a monogenic condition and such patients should be referred to specialized centres. In most patients with elevated BP or hypertension, routine genetic testing is not recommended. Family history and a pedigree analysis can help to find a heritable pattern of hypertension or hypotension.[304]

Recommendation Table 11 — Recommendations for genetic testing in hypertension management

Recommendations	Class[a]	Level[b]
Genetic testing should be considered in specialist centres for patients suspected to have rare monogenic causes of secondary hypertension or for those with phaeochromocytoma/paraganglioma.[302,305]	IIa	B
Routine genetic testing for hypertension is not recommended.	III	C

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[a]Class of recommendation.
[b]Level of evidence.

7.5. Resistant hypertension: definition and diagnosis

Despite availability and use of multiple BP-lowering medications, many patients worldwide have uncontrolled hypertension.[306–308] Considering this, societies have introduced the term 'drug-resistant hypertension', or 'treatment-resistant hypertension', or 'resistant hypertension',[309] which has been reported in 10%–20% of patients with hypertension.[310,311]

Resistant hypertension is not a disease *per se*. Compared with treated patients who achieve BP control, patients with resistant hypertension (by any definition) have a worse prognosis: risk of myocardial infarction, stroke, end-stage renal disease, and death in these adults may be two- to six-fold higher.[309] Secondary causes of hypertension are also more likely in the presence of resistant hypertension.[312]

All resistant hypertension definitions require a diuretic in the prescribed multiple-drug regimen, because excess salt intake and salt and water retention are key players in resistance to BP-lowering treatments (*Table 10*).[309]

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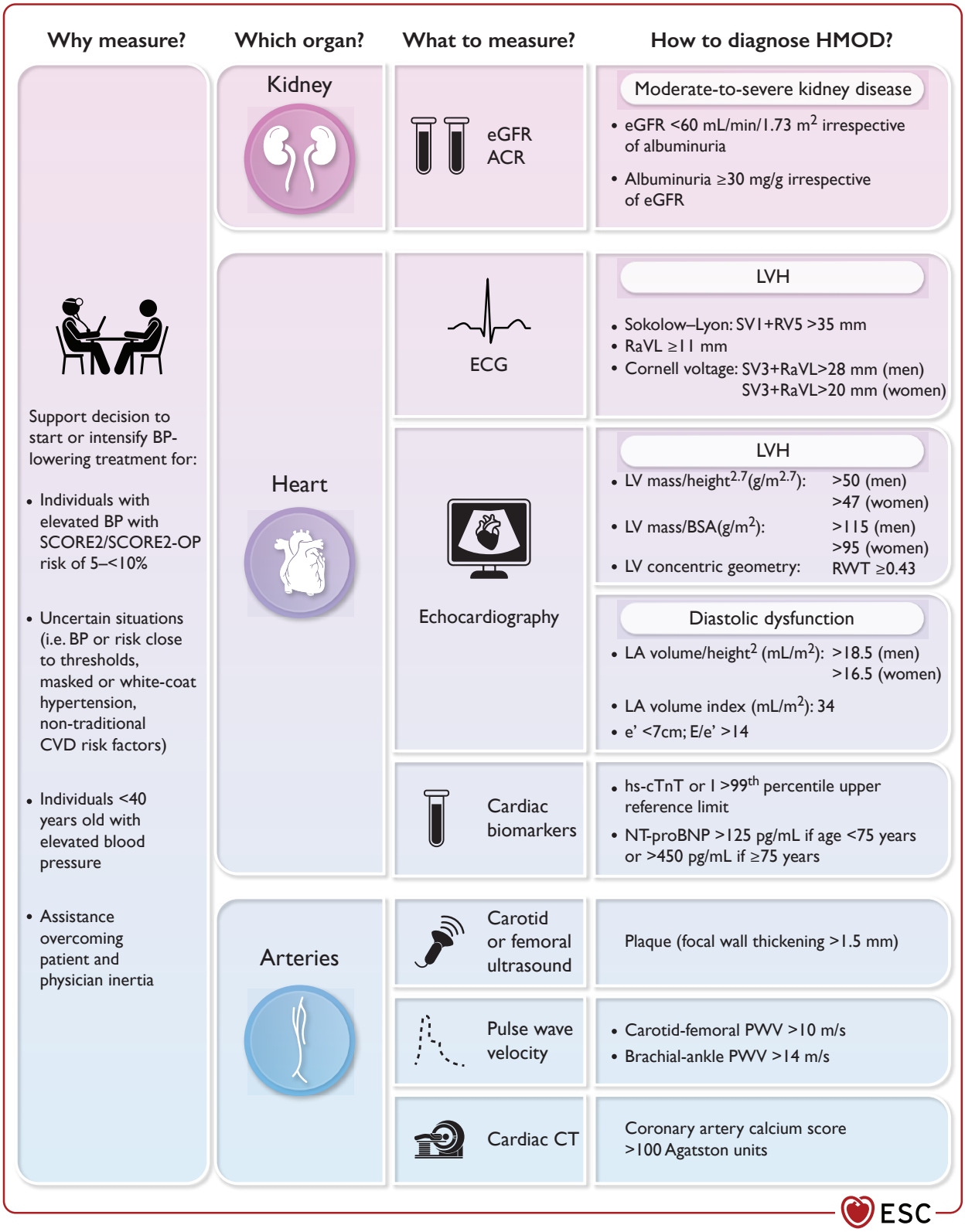

Figure 12 Tests and criteria for defining hypertension-mediated organ damage and considerations for their use in clinical practice. ACR, albumin: creatinine ratio; BP, blood pressure; BSA, body surface area; CT, computed tomography; CVD, cardiovascular disease; ECG, electrocardiogram; eGFR, estimated glomerular filtration rate; HMOD, hypertension-mediated organ damage; hs-cTnT, high-sensitivity cardiac troponin T; LA, left atrial; LV, left ventricular; LVH, left ventricular hypertrophy; NT-proBNP, N-terminal pro-brain natriuretic peptide; PWV, pulse wave velocity; RWT, relative wall thickness; SCORE2, Systematic COronary Risk Evaluation 2; SCORE2-OP, Systematic COronary Risk Evaluation 2–Older Persons. More details and references can be found in the Supplementary data online, *Tables S1 and S5*.

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Table 10 Current definition of resistant hypertension

Definition of resistant hypertension
Hypertension is defined as resistant when a treatment strategy including appropriate lifestyle measures and treatment with maximum or maximally tolerated doses of a diuretic (thiazide or thiazide-like), a RAS blocker, and a calcium channel blocker fail to lower office systolic and diastolic BP values to <140 mmHg and/or <90 mmHg, respectively. These uncontrolled BP values must be confirmed by out-of-office BP measurements (HBPM or ABPM—*Section 5.1* for relevant BP thresholds).
Key considerations
• Resistant hypertension is not a disease, but an indicator that should be used to identify patients at high risk for CVD, in which secondary hypertension is also frequent; • Pseudo-resistant hypertension must be excluded, including that caused by non-adherence to treatment; • In patients with decreased eGFR (i.e. <30 mL/min/1.73 m^2) an adequately up-titrated loop diuretic is necessary to define resistant hypertension; • Patients with suspected resistant hypertension should be referred to specialized centres; • These ESC Guidelines do not include the terms 'controlled resistant hypertension' (BP at target but requiring ≥4 medications) or 'refractory hypertension' (BP not at target despite ≥5 medications).

ABPM, ambulatory blood pressure monitoring; BP, blood pressure; CVD, cardiovascular disease; eGFR, estimated glomerular filtration rate; HBPM, home blood pressure monitoring; RAS, renin–angiotensin system.

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In addition, excluding pseudo-resistance is a prerequisite. Specifically, pseudo-resistance indicates poor adherence to BP-lowering treatment, which should be verified by careful questioning of the patient in the first instance (*Section 7.4.3*).[257] In addition, white-coat hypertension must be excluded.[263] Contributors to pseudo-resistance are listed in *Table 11*. Objective evaluation of adherence (either directly observed treatment or detecting prescribed drugs in blood or urine samples) should also be considered, if resources allow.

The work-up of patients presumed to have resistant hypertension is complex and often requires technologies that are not available to GPs.[257,309] Accordingly, we recommend these patients are referred to specialized centres.

Recommendation Table 12 — Recommendations for resistant hypertension work-up (see Evidence Table 18)

Recommendation	Class[a]	Level[b]
Patients with resistant hypertension should be considered for referral to clinical centres with expertise in hypertension management for further testing.[309,312]	IIa	B

[a]Class of recommendation.
[b]Level of evidence.

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7.6. Secondary hypertension: when to screen/further investigations

7.6.1. General considerations

Secondary hypertension is more prevalent than previously thought (*Figures 13–15*).[312–317] Depending on the definition used and the cohort studied, the prevalence of secondary hypertension is 10%–35% in all hypertensive patients[318,319] and up to 50% of patients with resistant hypertension (though the latter prevalence estimate included persons with eGFR < 40 mL/min/1.73 m^2).[312] Primary aldosteronism is a common cause,[315,320] with, e.g. a high prevalence of hyperaldosteronism (up to 12%) observed in patients with BP of >180/110 mmHg.[316] Despite these numbers, screening rates for primary aldosteronism, even in high-risk groups such as those with resistant hypertension[321] and hypokalaemia,[322] are low (around 2% and 4% of eligible patients, respectively). In most healthcare systems, GPs are typically the 'gatekeeper' of access to specialized care and should be involved in screening patients for common causes of secondary hypertension, especially sleep apnoea and primary aldosteronism (Supplementary data online, *Tables S2 and S3*). Primary aldosteronism is associated with an increased risk of CVD events, which may be partly independent of BP.[323,324]

Table 11 Conditions found to cause pseudo-resistance or resistance to blood pressure-lowering treatment

Causes of pseudo-resistant hypertension
Poor adherence to and persistence with treatment
White-coat phenomenon
Poor BP measurement method
Marked brachial artery calcification (Osler phenomenon)
Clinician inertia (inadequate doses, inappropriate combinations of BP-lowering drugs)
Munchausen syndrome (rare)
Causes of resistant hypertension
Behavioural factors
Overweight/obesity
Physical inactivity
Excess daily dietary sodium
Excess habitual alcohol consumption
Use of drugs or substances that may increase BP
See Supplementary data online, *Table S4*
Undetected secondary hypertension
See *Table 13*

BP, blood pressure.

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7.6.2. Primary aldosteronism

Though spontaneous or diuretic-induced hypokalaemia are strongly suggestive of primary aldosteronism, a history of hypokalaemia is not present in most patients diagnosed with this condition. The aldosterone-to-renin ratio (ARR) is thus recommended for primary aldosteronism screening (see *Figure 13*).[325] This test can easily be done in treatment-naïve patients, though it is far more common for the ARR test to be considered when patients are already being treated for elevated BP or hypertension. This is relevant because ARR can be influenced by the drugs being taken at the time of testing. Accordingly, there are 2 approaches to screen for aldosteronism among patients who are already undergoing treatment for elevated BP or hypertension:

- The first is to conduct ARR testing in treated patients with an indication for aldosteronism screening as efficiently as possible and without changing or stopping their baseline BP-lowering medications, simply to facilitate such testing. The ARR result then needs to be interpreted in the context of the specific medication(s) the patient is taking. Advantages of this approach include reducing barriers to screening and no change in medication in these patients, many of whom do not have BP controlled and in whom further deterioration in their BP control by stopping or changing medication may increase risk of CVD. Disadvantages include the interpretation of the ARR result, which depends on the specific medications taken at the time of testing.[326] Input from a hypertension specialist or endocrinologist may be necessary.

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- To reliably estimate renin and aldosterone status (and therefore ARR), and to facilitate a 'clean' screen for aldosteronism, a second approach is to discontinue drugs that affect these variables whenever feasible before ARR testing (*Table 12*). Such interfering drugs include, beta-blockers, centrally acting drugs (e.g., clonidine and alpha-methyldopa) renin-angiotensin system (RAS) blockers and diuretics.[326] Long-acting calcium channel blockers (CCBs), either dihydropyridine or non-dihydropyridine, and alpha-receptor antagonists do not interfere with the ARR and can be used instead of interfering medications before ARR testing. Should drugs that do not interfere with the ARR be contraindicated or insufficient to control BP, centrally acting sympatholytic drugs can also then be used, but at the risk of slightly more false positives (by renin suppression). Furthermore, when mineralocorticoid receptor antagonists (MRAs) cannot be stopped for safety reasons (i.e. severe hypokalaemia or severe hypertension among patients with severe hyperaldosteronism), recent evidence suggests that the accuracy of ARR testing under this treatment is only marginally impacted, particularly in the presence of florid primary aldosteronism.[327]

Assessing sodium intake (preferably 24 h urinary sodium, or sodium-to-creatinine ratio in the morning urine sample) is also important for interpreting the ARR, as is time in menstrual cycle for females. ARR cut-offs vary depending on unit of measurement and by local laboratory. For detailed information, readers are referred to the latest primary aldosteronism guidelines.[328,329]

7.6.3. Renovascular hypertension

Renovascular hypertension (RVH) defines a condition where renal artery occlusion or stenosis decreases renal perfusion pressure to a level that activates the renin–angiotensin–aldosterone system (RAAS), thereby raising BP. Major causes are atherosclerosis and fibromuscular dysplasia (*Figure 14* and Supplementary data online, *Tables S1* and *S2*). Atherosclerosis is the most common form of RVH, especially in older adults.[318] Fibromuscular dysplasia is a systemic non-atherosclerotic vascular disease involving medium-sized muscular arteries. When renal arteries are involved, fibromuscular dysplasia may induce RVH (FMD-RVH), especially in children and younger women.[330–332]

Though not highly sensitive, very elevated renin levels raise the suspicion for RVH. The work-up of RVH (*Table 13*) is based on imaging tests, such as renal artery Doppler ultrasound, with bilateral assessment of renal arterial resistive index, or abdominal CT angiography, or magnetic resonance imaging (MRI), in line with current ESC Guidelines on the diagnosis and treatment of peripheral arterial diseases,[277] which will be updated in 2024. Of note, bystander renal artery stenosis may be present in patients with essential hypertension, without causing secondary hypertension due to RVH. Since fibromuscular dysplasia is a systemic disease, CT or MRI angiography from head to pelvis is recommended in patients with FMD-RVH.[277,332]

7.6.4. Obstructive sleep apnoea syndrome

Obstructive sleep apnoea syndrome (OSAS) is prevalent in hypertension and particularly in resistant hypertension, with studies indicating that up to 60% of patients with resistant hypertension have features of OSAS.[314] OSAS should be suspected in patients with hypertension and suggestive symptoms (see Supplementary data online, *Table S2*), in all patients with resistant hypertension, and in patients with non-dipping or reverse-dipping pattern at 24 h BP monitoring, especially if obese (*Figure 15*). Using validated questionnaires may help identify patients at high risk of OSAS.[333] Lack of suggestive symptoms does not rule out OSAS. A simplified polysomnogram confirms the diagnosis [apnoea–hypopnoea index (AHI) > 5] and can quantify the severity of OSAS (mild: AHI < 15; moderate: AHI of 15–30; severe: AHI > 30).[334]

Table 12 Drugs and conditions that affect aldosterone, renin, and aldosterone-to-renin ratio

Factor	Effect on plasma aldosterone levels	Effect on renin levels	Effect on ARR
Serum potassium status			
Hypokalaemia	↓	→↑	↓ **(FN)**
Potassium loading	↑	→↓	↑
Sodium restriction	↑	↑↑	↓ **(FN)**
Sodium loading	↓	↓↓	↑ **(FP)**
Drugs			
Beta-adrenergic blockers	↓	↓↓	↑ **(FP)**
Calcium channel blockers (DHPs)	→↓	→↑	→↓ **(FN** with short-acting DHPs**)**
ACE inhibitors	↓	↑↑	↓ **(FN)**
ARBs	↓	↑↑	↓ **(FN)**
Potassium-sparing diuretics	↑	↑↑	↓ **(FN)**
Potassium-wasting diuretics	→↑	↑↑	↓ **(FN)**
Alpha-2 agonists (clonidine, methyldopa)	↓	↓↓	↑ **(FP)**
NSAIDs	↓	↓↓	↑ **(FP)**
Steroids	↓	→↓	↑ **(FP)**
Contraceptive agents (drospirenone)	↑	↑	↑ **(FP)**

↑, raised; ↓, lowered; →, no effect; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; ARR, aldosterone-to-renin ratio; DHPs, dihydropyridines; FN, false negative; FP, false positive; NSAID, non-steroidal anti-inflammatory drug.

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Table 13 Optional tests that should be used to screen for secondary hypertension in the presence of suggestive signs, symptoms, or medical history

Cause of secondary hypertension	Screening test
Primary aldosteronism	Aldosterone-to-renin ratio Helpful information can also be provided by reviewing prior potassium levels (hypokalaemia increases the likelihood of coexistent primary hyperaldosteronism)
Renovascular hypertension	Renal doppler ultrasound Abdominal CT angiogram or MRI
Phaeochromocytoma/paraganglioma	24 h urinary and/or plasma metanephrine and normetanephrine
Obstructive sleep apnoea syndrome	Overnight ambulatory polysomnography
Renal parenchymal disease	Plasma creatinine, sodium, and potassium eGFR Urine dipstick for blood and protein Urinary albumin-to-creatinine ratio Renal ultrasound
Cushing's syndrome	24 h urinary free cortisol Low-dose dexamethasone suppression test
Thyroid disease (hyper- or hypothyroidism)	TSH
Hyperparathyroidism	Parathyroid hormone Calcium and phosphate
Coarctation of the aorta	Echocardiogram Aortic CT angiogram

CT, computed tomography; eGFR, estimated glomerular filtration rate; MRI, magnetic resonance imaging; TSH, thyroid-stimulating hormone.

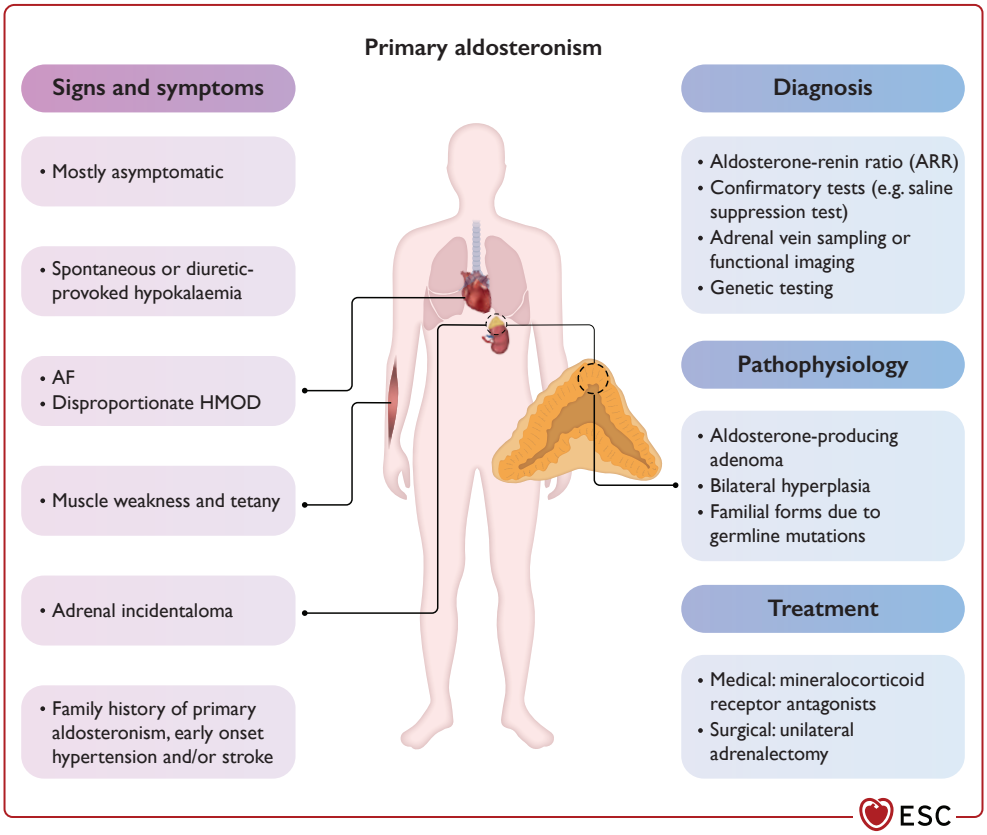

Figure 13 Summary of primary aldosteronism as a common form of secondary hypertension. AF, atrial fibrillation; HMOD, hypertension-mediated organ damage.

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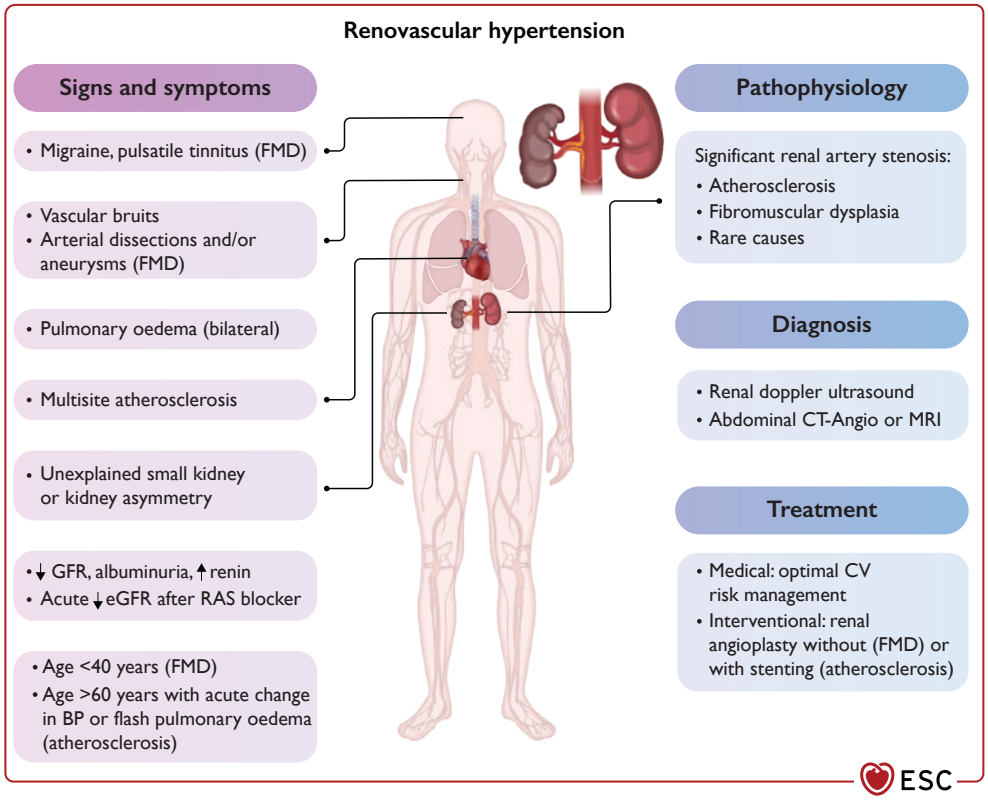

Figure 14 Summary of renovascular disease as a common form of secondary hypertension. CT-Angio, computed tomography angiography; CV, cardiovascular; FMD, fibromuscular dysplasia; GFR, glomerular filtration rate; MRI, magnetic resonance imaging; RAS, renin–angiotensin system.

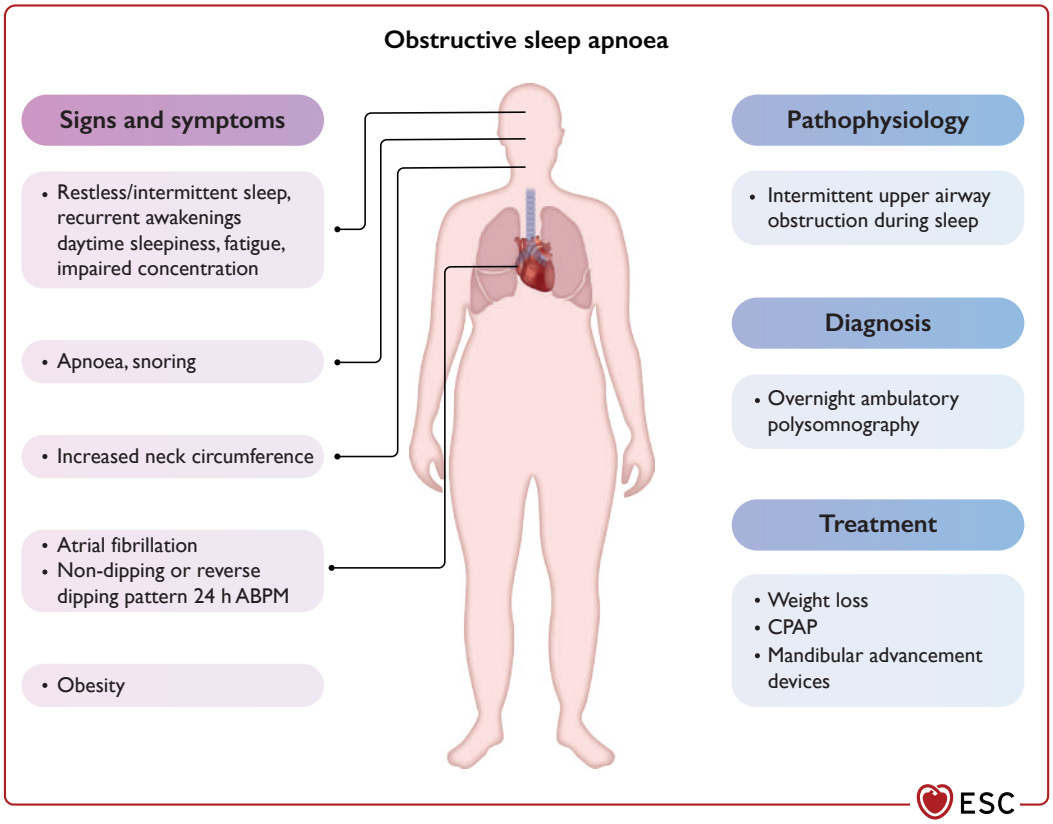

Figure 15 Summary of obstructive sleep apnoea as a common form of secondary hypertension. AF, atrial fibrillation; ABPM, ambulatory blood pressure monitor; CPAP, continuous positive airway pressure.

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7.6.5. Phaeochromocytoma/paraganglioma

Phaeochromocytomas/paragangliomas (PPGLs) are a rare form of secondary hypertension characterized by a highly heterogeneous clinical presentation.[335,336] PPGLs are usually discovered incidentally.[337]

A PPGL should be suspected in the presence of signs and symptoms of catecholamine excess or in syndromic PPGL, in patients with a family history of PPGL, and in carriers of a germline mutation in one of the PPGL-causing genes.[338] Since normetanephrine and metanephrine are secreted constitutively, as opposed to the highly variable nature of catecholamine secretion, they are preferred as screening tests for PPGL (*Table 13*).

Recommendation Table 13 — Recommendations for screening for secondary hypertension (see Evidence Tables 19 and 20)

Recommendations	Class[a]	Level[b]
It is recommended that patients with hypertension presenting with suggestive signs, symptoms or medical history of secondary hypertension are appropriately screened for secondary hypertension.[312,314,315,323,339]	I	B
Screening for primary aldosteronism by renin and aldosterone measurements should be considered in all adults with confirmed hypertension (BP ≥140/90 mmHg).[313,316,323,339]	IIa	B

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BP, blood pressure.
[a]Class of recommendation.
[b]Level of evidence.

8. Preventing and treating elevated blood pressure and hypertension

The ultimate goal of preventing and treating elevated BP and hypertension is to reduce CVD, to improve quality of life, and to prevent premature death. Crucially, besides BP, other CVD risk factors need to be comprehensively addressed (e.g. smoking, glucose, dyslipidaemia) as detailed in the 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice.[170] Indeed, it is worth emphasizing that, when combined, these CVD risk factors have multiplicative (not additive) effects on CVD risk.[340]

8.1. Prevention strategies in early life

Detailed information on this topic is provided in the Supplementary data online. High BP tracks from childhood to adulthood.[341,342] Hypertension in childhood was redefined in a 2022 ESC Consensus Document.[343]

Recommendation Table 14 — Recommendations for screening for hypertension in children and adolescents (see Evidence Table 21)

Recommendation	Class[a]	Level[b]
Opportunistic screening with office BP measurements to monitor development of BP during late childhood and adolescence, especially if one or both parents have hypertension, should be considered to better predict development of adult hypertension and associated CVD risk.[344–346]	IIa	B

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BP, blood pressure; CVD, cardiovascular disease.
[a]Class of recommendation.
[b]Level of evidence.

8.2. Non-pharmacological interventions

A major underlying contributor to elevated BP and hypertension in the general adult population is unhealthy lifestyle, with severe consequences for all-cause and CVD mortality. As such, we give lifestyle interventions to reduce BP a special status in our recommendations throughout these guidelines (*Figures 16* and *17*). This is reflected by a modified approach to the class of recommendations for lifestyle interventions compared with medical interventions (pharmacological or procedural). Given the salutary benefits of healthy lifestyle on a range of outcomes that extend well beyond BP-lowering effects,[347] including broad mental and physical health benefits, we do not require lifestyle interventions to have RCT evidence for efficacy in reducing CVD events through BP lowering to achieve a Class I recommendation. In deciding to give lifestyle interventions this status, the task force also recognizes that: (i) lifestyle interventions are less likely to be subjected to clinical outcomes trials (e.g. due to funding limitations and lack of interest from industry), and (ii) the risks of adverse effects and toxicity relating to healthy lifestyle interventions are low. In contrast, in these guidelines, to achieve a Class I recommendation (irrespective of level of evidence) there needs to be evidence that medical interventions that reduce BP also decrease CVD events by BP lowering.

8.2.1. Dietary sodium and potassium intake

8.2.1.1. Sodium

Reducing dietary salt (sodium chloride) intake in individuals with high baseline intake lowers CVD event rates.[348] Extensive observational studies have reported dose–response associations between high dietary sodium intake and CVD events.[349–351] The potential impact of salt reduction on population health is significant, particularly in countries where the population's average salt intake is high. Pooled data from long-term follow-up salt-reduction trials demonstrate that reducing salt by 2.5 g/day is associated with an approximately 20% reduction in CVD events at the population level.[349]

The health benefits of salt reduction are likely mediated, largely, by BP-lowering effects.[352–354] An almost linear relationship has been described in a dose–response meta-analysis between sodium intake ranging from 0.4 to 7.6 g/day and reduction of systolic and diastolic BP is independent of baseline BP.[355,356] Women appear to be, on average, more sodium sensitive than men,[357] and may have greater outcome benefits when receiving comparable sodium-restricted diets.[358] Trial evidence for the BP-lowering benefits of salt reduction extend down to daily sodium intakes of <1.5 g/day.[356,358–361]

The task force acknowledges that the observational data linking sodium intake to CVD outcomes are mixed and that some studies have not found a link between salt intake and CVD.[362,363] In addition, a potential J-curve exists between sodium intake and CVD events (whereby some analyses suggest that sodium reduction to very low levels could be harmful).[363,364] While there are differences of opinion, the task force agreed that, on balance, (i) observational J-curve data are often due to reverse causality or confounding,[114,348,365,366] (ii) the relationship between dietary sodium and stroke is typically linear in shape, without any J-curve, (iii) if the J-curve were causal, the adverse effect of very low sodium on CVD would have to be mediated by some harmful mechanism that overcomes the expected benefit mediated by BP lowering (which is unlikely), and (iv) estimation of sodium intake using spot-urine sodium testing (which was commonly done in studies reporting a J-curve) may not be as valid as other methods.[367] For example, most (but not all)[362] reports measuring 24 h urine sodium excretion (a surrogate measure of sodium intake) have not reported a J-curve association with CVD.[350,364] Furthermore the causal evidence demonstrating reduced CVD with sodium restriction (using potassium-enriched salt substitutes) in the Salt Substitute and Stroke Study (SSaSS)

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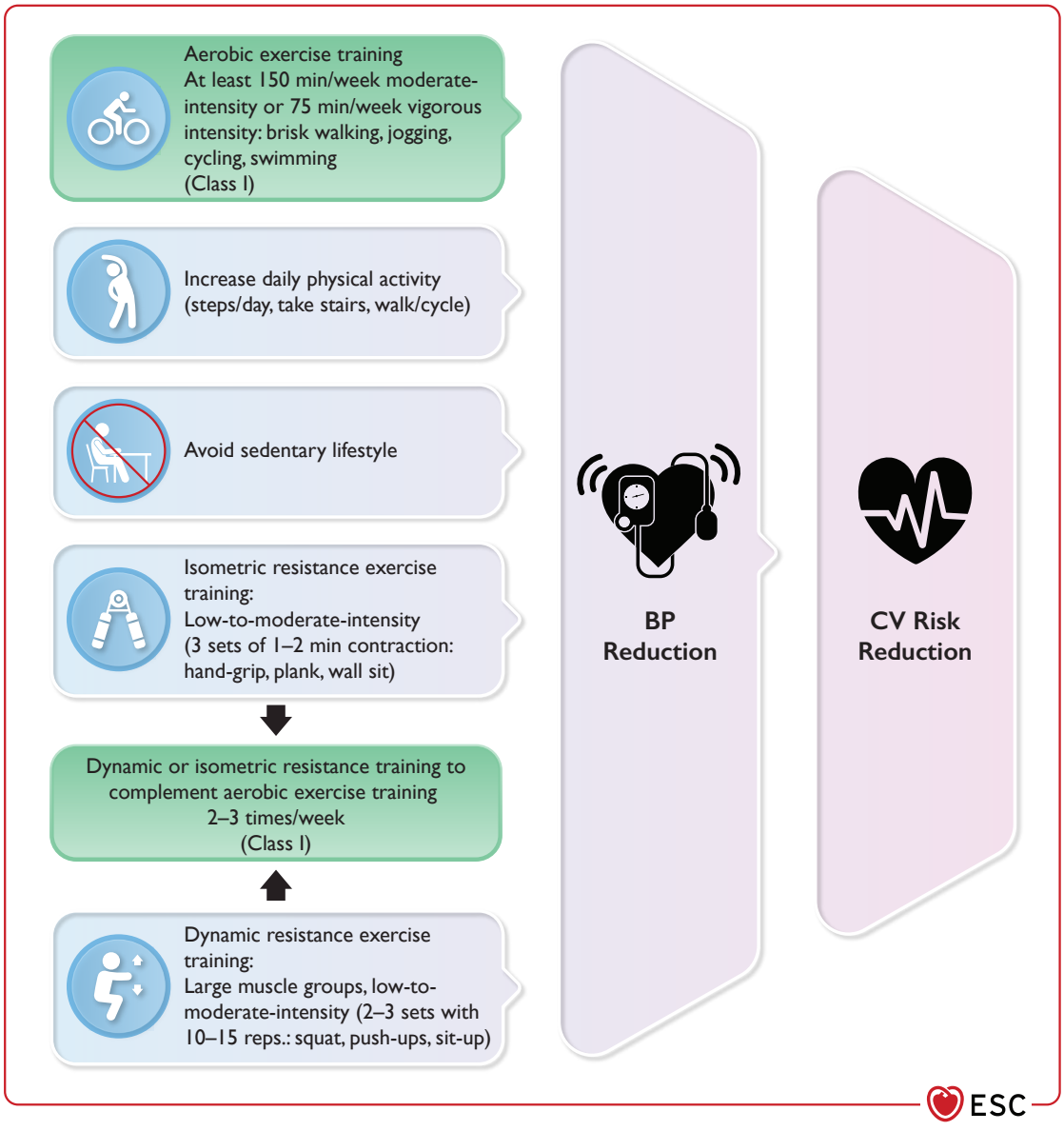

Figure 16 Physical activity according to different types of exercise and reduction of blood pressure and overall cardiovascular disease risk. Priority is given to aerobic exercise training (green). BP, blood pressure; CVD, cardiovascular disease.

and Diet, ExerCIse and carDiovascular hEalth-Salt (DECIDE-Salt) trials was compelling,[348,368] even though sodium restriction in these trials was not below 2 g/day.

It is recommended to restrict total dietary sodium intake to approximately 2 g/day or less (equivalent to approximately 5 g or about a teaspoon of salt per day). This includes added salt and salt already contained in food. While the feasibility of this sodium target can be debated, the evidence for the benefits of this sodium target among patients with elevated BP or hypertension is sufficient, particularly in terms of lowering BP. The optimal sodium intake in the general population with non-elevated BP is less clear (noting also that the BP-lowering effect of salt reduction among patients with non-elevated BP appears lower).[353,354] A more feasible compromise in the general population might be a target sodium intake range of 2–4 g/day.[369,370] It needs to be emphasized that large parts of daily sodium intake occur by means of sodium consumption contained in processed foods.

8.2.1.2. Potassium

Optimal dietary potassium intake, e.g. by consuming diets rich in fruits and vegetables, has BP-lowering effects and may be associated with lower CVD risk.[348,364,368,371–373] The association between potassium intake, systolic BP, and CVD events may be sex-specific, being stronger in women.[374] The World Health Organization (WHO) recommends over 3.5 g/day (~90 mmol/day) of dietary potassium.[375] Excessive potassium supplementation should, however, be avoided[374] and CKD guidelines recommend dietary potassium restriction to <2.4 g/day in persons with advanced CKD (see Supplementary data online).[376]

A lower urinary sodium-to-potassium ratio (Na^+/K^+ ratio; a surrogate for reduced dietary sodium intake complemented by increased potassium intake) has been associated with a greater reduction in systolic and diastolic BP than with a higher ratio.[348,377]

In patients with hypertension and high dietary sodium, increased dietary intake of potassium (in addition to lower dietary sodium) should be considered.[348,350,378] In patients with persistently high sodium intake (>5 g/day)

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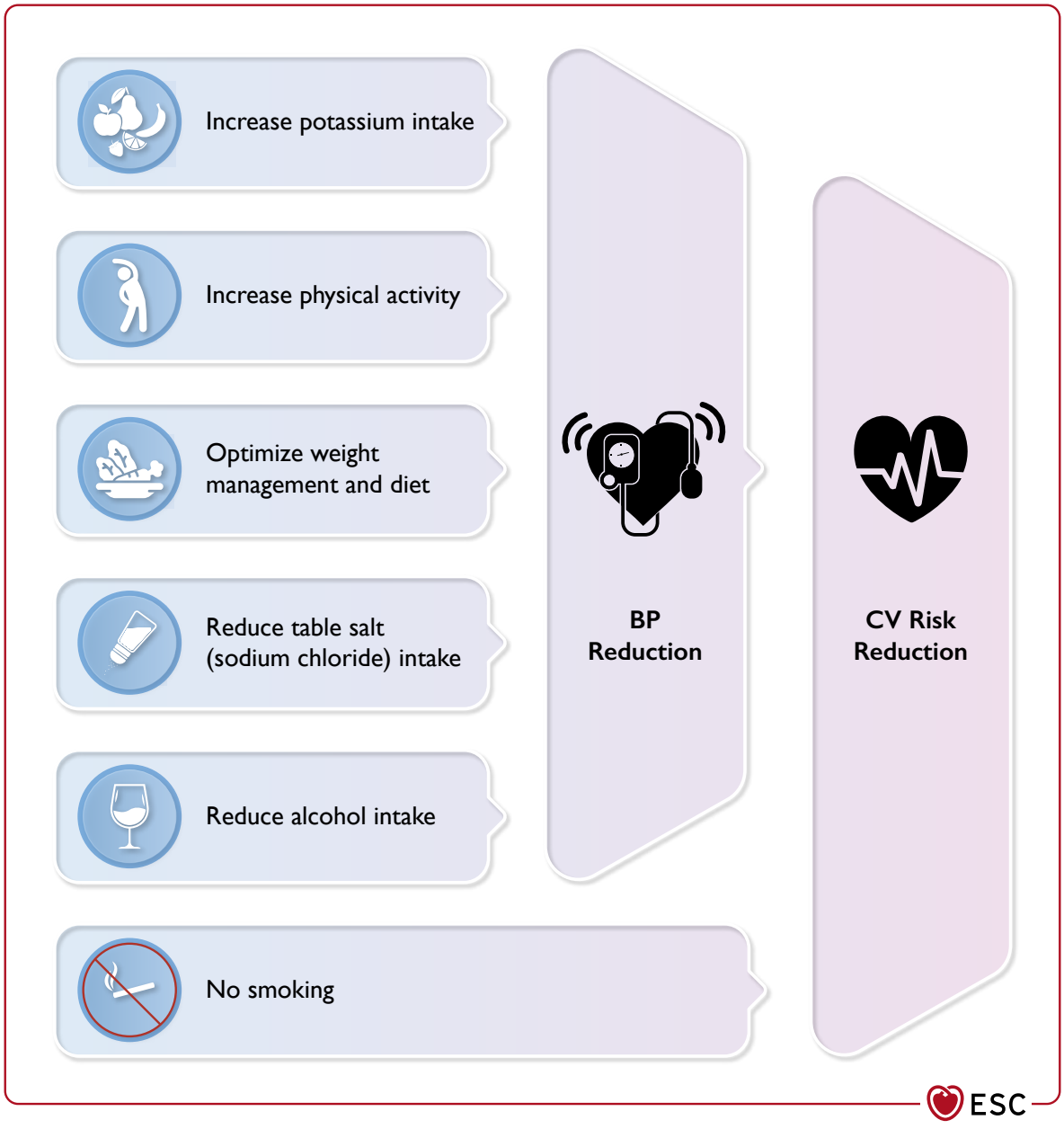

Figure 17 Effects of main lifestyle factors on blood pressure and cardiovascular risk reduction. BP, blood pressure; CV, cardiovascular. Smoking cessation reduces overall cardiovascular risk but not BP (long arrow). Salt reduction reduces BP and (for persons with high baseline intake) reduces cardiovascular risk. Increased potassium intake and higher physical activity, as well as optimized weight management, reduce BP and are associated with lower overall cardiovascular risk (short arrows).

and without moderate-to-advanced CKD, particularly women, an increase in potassium intake by 0.5–1.0 g/day may be considered to achieve a favourable Na^+/K^+ ratio of 1.5–2.0 and to reduce CVD risk. Potassium supplementation can be achieved by substituting sodium using potassium enriched salts (75% sodium chloride and 25% potassium chloride)[368,379,380] or by increasing dietary potassium intake [e.g. a 125 g (medium) banana contains about 450 mg potassium, or unsalted boiled spinach (840 mg/cup) or mashed avocado (710 mg/cup)]. In patients with CKD and/or those taking potassium-sparing medication, such as some diuretics, angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs) or spironolactone, serum levels of potassium should be monitored (noting that phlebotomy recommendations to avoid spuriously high potassium results should be followed).

8.2.2. Physical activity and exercise

In a systematic review and meta-analyses, aerobic (endurance) exercise was suggested as the first-line exercise therapy for reducing BP in patients with elevated BP and hypertension vs. alternative forms of exercise, such as dynamic or isometric resistance training.[381] In patients with hypertension, regular aerobic exercise substantially lowers systolic BP by up to 7–8 mmHg and diastolic BP by up to 4–5 mmHg.[381,382] For non-white patients with hypertension, dynamic resistance training elicits BP reductions that appear comparable to aerobic exercise.[383] Isometric resistance training also achieves clinically relevant BP reductions in patients with hypertension, but results are inconsistent and more data from more high-quality intervention trials are required (see Supplementary data online).[381,384,385] With respect to mode and intensity of aerobic exercise,

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high-intensity interval training elicits comparable BP reductions to moderate continuous exercise, with high-intensity interval training achieving greater improvement in physical fitness.[386]

In patients with known hypertension, engaging in physical activity is associated with reduced CVD mortality risk vs. sedentary patients with hypertension.[387]

An exaggerated BP response to exercise may yield diagnostic merits for predicting incident hypertension and CVD. In a meta-analysis, an exaggerated BP response to exercise was associated with an increased risk for masked hypertension.[388] The risk of coronary heart disease also increases with higher systolic BP during exercise, independent of systolic BP at rest.[389]

Prior recommendations for at least 150 min/week of moderate intensity aerobic exercise (≥30 min, 5–7 days/week) can be maintained.[1,390] Alternatively, 75 min of vigorous-intensity exercise per week over 3 days may be performed, with additional benefits derived by achieving 300 min of moderate-intensity or 150 min of vigorous-intensity aerobic physical activity per week.[390,391] As acute aerobic exercise induces intensity-dependent short-term reductions in ambulatory BP after exercise, patients with elevated BP and hypertension may benefit from daily exercise to improve their 24 h BP profile and avoid BP peaks on sedentary days.[392] Aerobic exercise should be complemented by low- or moderate-intensity resistance training (2–3 times per week), e.g. dynamic resistance, starting at 2–3 sets of 10–15 repetitions at 40%–60% of one-repetition maximum[393] or isometric resistance training with three sets of 1–2 min contractions, such as hand-grip, plank, or wall sit (*Figure 16*).[381,394]

In uncontrolled hypertension at rest, high-intensity exercise should be applied with caution, with resting systolic BP of >200 mmHg and diastolic BP of >110 mmHg indicating relative contraindications.[395] Age, sex, gender,[396] ethnicity, and comorbidities, as well as individual preferences, should be considered for individual exercise prescription. Detailed information on exercise prescription in terms of frequency, intensity, time (duration) and type and progression are available in the 2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease,[390] which include recommendations for pre-participation screening and cardiopulmonary exercise testing.[390]

8.2.3. Weight reduction and diet

Visceral obesity is common and associated with incident hypertension.[397,398] An average weight loss of 5 kg has been associated with an average systolic and diastolic BP reduction of 4.4 and 3.6 mmHg, respectively.[399] Data show that, starting at an index body mass index (BMI) of 40 kg/m^2, a median weight loss of 13% is associated with a 22% lower risk for hypertension.[400,401] Maintaining even moderate weight loss of 5%–10% of initial body weight can improve not only BP, but also glucose and lipid metabolism, and potentially reduce premature all-cause mortality.[402–404] However, achieving long-term effects in patients with hypertension via weight loss is challenging and the magnitude of these effects remains unclear.[405,406] Weight stabilization during middle-age appears to be an important and attainable goal to prevent obesity-related increase in BP later in life.[407]

Evidence-based diets, such as the Mediterranean diet and the Dietary Approaches to Stop Hypertension (DASH) diet, are established interventions in patients with hypertension to reduce their BP and CVD risk.[408,409] Additional information on healthy dietary patterns is provided in the 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice and Supplementary data online.[170]

In combination with weight-loss and exercise interventions[410] and low sodium intake,[411] the DASH diet has added effect on BP reduction.[412] Pharmacological treatment of obesity with orlistat achieved a slight reduction of 2.6 mmHg in systolic BP.[413] The greatest BP-lowering effects of weight-loss medications may be achieved with the glucagon-like peptide 1 (GLP-1) receptor agonists.[414–416] For example, in the Semaglutide Treatment Effect in People with Obesity (STEP-1) trial, the GLP-1 analogue semaglutide resulted in a mean weight reduction of 12.4% and a 5.1 mmHg reduction in systolic BP.[415]

8.2.4. Alcohol, coffee, and soft drinks

In a 2020 Cochrane review, the short-term effects of alcohol on BP were dose dependent; low-dose alcohol (<14 g) did not affect BP within 6 h, medium-dose (14–28 g) decreased both systolic and diastolic BP, and high-dose alcohol (>30 g) first decreased BP up to 12 h and then increased BP following >13 h of consumption by 3.7 mmHg systolic and 2.4 mmHg diastolic.[417] The trials in this Cochrane review included small numbers of women. In the longer term, no evidence has been found for a protective effect of chronic alcohol consumption on hypertension, for either sex. In contrast, even low-dose alcohol consumption (10 g/day) increases chronic risk of hypertension by 14% in men, but not in women.[418] As per the 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice, men and women are recommended to stay within the upper limit of drinking alcoholic beverages (100 g/week of pure alcohol). Defining number of drinks depends on portion size, the standards of which differ per country, but translates to 8–14 g/drink.[170] Emerging data indicate it is likely healthiest to avoid all alcohol, where possible.[419]

Coffee intake is not associated with a higher risk of hypertension in the general population; in fact, higher coffee consumption may be associated with a lower risk for incident hypertension.[420] Data regarding the association between tea drinking and CVD are inconclusive, though mechanistic trials have suggested benefits on BP lowering.[421] In contrast, energy drinks with high concentrations of ingredients such as taurine and caffeine increase BP and may lead to acute or chronic cardiovascular complications in young adults.[422–424]

Consuming two or more servings per day of sugar-sweetened beverages was associated with a 35% higher risk of coronary artery disease in women in the Nurses' Health Study.[425] In the European Prospective Investigation into Cancer and Nutrition (EPIC) cohort, both sugar- and artificially sweetened soft drinks were associated with higher all-cause mortality.[426] In children and adolescents, sugar-sweetened beverages increased systolic BP and the risk for incident hypertension.[427] It is recommended to restrict free sugar consumption, in particular sugar-sweetened beverages, to a maximum of 10% of energy intake.

8.2.5. Smoking

To stop smoking is arguably the most effective measure in preventing major CVD events at the individual level, likely through improved vascular health.[428–431] Estimated health benefits will be even more substantial looking at all-cause morbidity and mortality, e.g. including smoking cessation for cancer prevention.

The effects of electronic cigarettes (e-cigarettes) on BP remain unclear and to date there are no robust outcomes data. However, growing evidence suggests that e-cigarettes can increase BP (see Supplementary data online).[432,433]

Among adults, smoking affects ambulatory BP by raising daily BP,[434] but effects of chronic smoking on office BP appear to be small.[435] Smoking cessation advice helps, but more intensive interventions are superior.[436,437] As recommended by previous ESC Guidelines, smoking cessation is recommended to reduce CVD risk and improve non-CVD health.[1,170]

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Recommendation Table 15 — Recommendations for non-pharmacological treatment of blood pressure and cardiovascular risk reduction (see Evidence Tables 22–26)

Recommendations	Class[a]	Level[b]
Restriction of sodium to approximately 2 g per day is recommended where possible in all adults with elevated BP and hypertension [this is equivalent to about 5 g of salt (sodium chloride) per day or about a teaspoon or less].[353,354]	I	A
Moderate intensity aerobic exercise of ≥150 min/week (≥30 min, 5–7 days/week) or alternatively 75 min of vigorous intensity aerobic exercise per week over 3 days are recommended and should be complemented with low- or moderate-intensity dynamic or isometric resistance training (2–3 times/week) to reduce BP and CVD risk.[1,381,390–393]	I	A
It is recommended to aim for a stable and healthy BMI (e.g. 20–25 kg/m^2) and waist circumference values (e.g. <94 cm in men and <80 cm in women) to reduce BP and CVD risk.[399–401]	I	A
Adopting a healthy and balanced diet, such as the Mediterranean or DASH diets, is recommended to help reduce BP and CVD risk.[412,438,439]	I	A
Men and women are recommended to drink less alcohol than the upper limit, which is about 100 g/week of pure alcohol. How this translates into number of drinks depends on portion size (the standards of which differ per country), but most drinks contain 8–14 g of alcohol per drink. Preferably, it is recommended to avoid alcohol to achieve the best health outcomes.[170,419,440,441]	I	B
It is recommended to restrict free sugar consumption, in particular sugar-sweetened beverages, to a maximum of 10% of energy intake. It is also recommended to discourage consumption of sugar-sweetened beverages, such as soft drinks and fruit juices, starting at a young age.[425–427]	I	B
It is recommended to stop tobacco smoking, initiate supportive care and refer to smoking cessation programmes, as tobacco use strongly and independently causes CVD, CVD events, and all-cause mortality.[428,429,431,437]	I	A
In patients with hypertension without moderate to advanced CKD and with high daily sodium intake, an increase of potassium intake by 0.5–1.0 g/day—for example through sodium substitution with potassium-enriched salt (comprising 75% sodium chloride and 25% potassium chloride) or through diets rich in fruits and vegetables—should be considered.[348,368,373,374,442]	IIa	A
In patients with CKD or taking potassium-sparing medication, such as some diuretics, ACE inhibitors, ARBs, or spironolactone, monitoring serum levels of potassium should be considered if dietary potassium is being increased.	IIa	C

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ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BMI, body mass index; BP, blood pressure; CKD, chronic kidney disease; CVD, cardiovascular disease; DASH, Dietary Approaches to Stop Hypertension.
[a]Class of recommendation.
[b]Level of evidence.

8.3. Pharmacological interventions

8.3.1. Treatment strategy to reduce adverse cardiovascular disease outcomes

The main goal of reducing BP is to prevent adverse CVD outcomes. The relative risk reduction afforded by a fixed degree of BP reduction is largely independent of pre-treatment BP.[116] There is a clear relationship between the intensity of BP lowering and the relative and absolute reduction in risk of CVD events for all adults, regardless of age (at least up to 85 years), sex, prior CVD, diabetes, or AF.[116,131,443–445] With this strong evidence for the 'the lower the better, but within reason' paradigm, decision rules are required for selecting patients most likely to benefit from treatment.[172] In this section, a summary of evidence for BP-lowering drug treatment is provided, followed by strategies for their use for preventing CVD.

8.3.2. Drug classes with evidence on clinical outcomes in the target population

The major drug classes with robust evidence for BP-mediated reduction in CVD events are ACE inhibitors, ARBs, dihydropyridine CCBs, diuretics (thiazides and thiazide-like diuretics such as hydrochlorothiazide, chlorthalidone, and indapamide), and beta-blockers (see Supplementary data online, *Tables S7 and S8*).[122,446,447] The first four are recommended as first-line options for starting hypertension treatment in the general population. Beta-blockers can be added preferentially in circumstances such as in the presence of angina or heart failure, after myocardial infarction, or for controlling heart rate, where they are the cornerstone of therapy.[122,448,449] In such settings, second-generation (cardioselective) and, specifically, third-generation (vasodilating) beta-blockers are preferred.[450] However, beta-blockers are less effective than ACE inhibitors, ARBs, CCBs, or diuretics at preventing stroke, and have a higher discontinuation rate due to side effects.[451,452] Beta-blockers and diuretics, especially when combined, are associated with an increased risk of new-onset diabetes in predisposed patients.[453,454] The effect of RAS blockers and CCBs on preventing progression of HMOD also appears to be superior to beta-blockers.[455–458] Beta-blockers should also be avoided in patients with isolated systolic hypertension or more generally with arterial stiffness, as they increase stroke volume (given the lower heart rate).[218]

When therapy and adherence with the above-mentioned drug classes is optimized but insufficient to reach BP goals, other drug classes can be used for treating hypertension. Of these, spironolactone, an MRA, appears to be the most effective at further lowering BP in resistant hypertension; however, more evidence of CVD risk-lowering effects with MRAs among all hypertensive populations, especially those without resistant hypertension, is needed.[459] Specifically, while use of MRAs in patients with heart failure has provided clinical evidence on the effectiveness of MRAs for preventing CVD events, dedicated outcome trials in patients with primary hypertension without heart failure are lacking. Because the present guidelines require trial evidence for CVD outcome benefit for a BP-lowering drug or procedure to achieve a Class I recommendation, and given no outcome trials of MRAs have been conducted in general samples of patients with primary hypertension, we have given MRAs a Class IIa recommendation (see below). We acknowledge that spironolactone was provided a Class I recommendation in the 2018 ESC/ESH Guidelines on the management of arterial hypertension. However, to be consistent with our requirement for trial evidence for CVD outcomes benefit in patients with hypertension, the task force agreed to provide a Class IIa recommendation for spironolactone in these 2024 Guidelines. Importantly, it was also agreed that a Class IIa recommendation (i.e. should be considered) is an endorsement of MRAs for

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treating resistant hypertension but one that acknowledges some uncertainty of outcomes benefit. Future outcome trials of MRAs, perhaps including finerenone,[460–462] are encouraged in patients with hypertension.

Clinical outcome evidence from trials for other BP-lowering drug classes, such as alpha-blockers, hydralazine, minoxidil, other potassium-sparing diuretics, and centrally acting agents, is less compelling and caution regarding adverse effects is warranted. However, they may be a final addition if all other therapeutic efforts are insufficient to decrease BP. Of note, in the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack (ALLHAT) trial, the alpha-blocker arm was stopped early due to futility of benefit for the CVD outcome.[463]

8.3.3. New therapies with blood pressure-lowering properties that await supportive evidence from cardiovascular outcomes trials prior to guideline endorsement and routine use in hypertension

A few other drug classes with indication in heart failure have emerged that also have BP-lowering properties. For example, the angiotensin receptor–neprilysin inhibitor (ARNi) sacubitril/valsartan, which was initially developed for hypertension,[464] reduces CVD mortality and morbidity in patients with heart failure, an effect that may have been mediated, in part, by superior BP lowering compared with enalapril alone.[465,466] In a *post hoc* subgroup analysis, sacubitril/valsartan lowered BP in adults with both heart failure with preserved ejection fraction (HFpEF) and resistant hypertension.[467] In the context of research studies, sacubitril/valsartan has been used in higher doses (200 mg or 400 mg once daily) for treating hypertension.[464,466,468,469]

SGLT2 inhibitors have shown favourable effects on CVD events and renal haemodynamics in patients with and without type 2 diabetes, and in heart failure trials.[470] In these trials, SGLT2 inhibitors did lower BP, though only modestly.[471] Small trials among adults with hypertension have confirmed the potential for BP lowering with this drug class.[472,473]

Other new drugs with preliminary data include GLP-1 agonists and the new non-steroidal MRAs, e.g. finerenone, in managing hypertension.[415,460,461] In addition, novel aldosterone synthase inhibitors (baxdrostat and lorundrostat) have significantly lowered BP in patients with uncontrolled hypertension in phase 2 trials.[474,475]

The dual endothelin-A and -B receptor antagonist aprocitentan also reduced office and 24 h BP compared with placebo at 4 weeks in patients with resistant hypertension in a phase 3 trial.[476] Zilebesiran, an investigational RNA interference agent administered subcutaneously, inhibits hepatic angiotensinogen synthesis and a single dose reduced 24 h BP over approximately 6 months.[477]

8.3.4. Drug combinations and up-titrating strategies

To treat hypertension, many patients will require more than one BP-lowering medication. Combining drugs from different drug classes can have additive or synergistic effects and lead to greater BP reduction than increasing the dose of one drug.[478–483] The superior BP-lowering efficacy of combination therapy is mediated, at least in part, by the potential of combination therapy to target multiple pathophysiological pathways contributing to perturbed BP in each patient.[484] A further benefit of combination therapy is the potential to use lower doses of each individual BP-lowering agent, which may reduce side effects and improve adherence and persistence,[485] though the evidence for this hypothesis has been questioned.[486]

Upfront low-dose combination therapy is therefore recommended in persons with hypertension, with the potential advantages of fewer side effects and swifter BP control being important for long-term adherence.[487–489] If combination BP-lowering therapy is pursued, single-pill combinations are preferred. For those with elevated BP who have an indication for BP-lowering treatment, monotherapy is recommended in the first instance.

One caveat to combination therapy in hypertension is that patient-level response to individual BP-lowering drug classes can be heterogeneous (suggesting some patients may benefit from more personalized treatment compared with routine combinations).[490] This is relevant also with respect to race/ethnicity (see *Section 9*). Another caveat is that the evidence for reduced CVD outcomes with BP-lowering drugs in combination therapy is based on observational studies.[491–493] There are no outcomes data from prospective trials that prove superiority of upfront combination therapy (either as single-pill combinations or as separate pills) over upfront monotherapy in the isolated treatment of hypertension.[486] Therefore, we considered giving upfront combination therapy (either as separate pills or as single-pill combinations) a Class IIa recommendation in these guidelines. However, given the totality of evidence for outcomes benefit in observational studies, randomized trial data for better BP control and adherence, and importantly, also given CVD outcomes benefit for polypills (a form of single-pill combination) in randomized trials,[494–496] we chose to provide a Class I recommendation for upfront combination therapy in adults with confirmed hypertension, in agreement with 2018 ESC recommendations.

The major four drug classes (ACE inhibitors, ARBs, dihydropyridine CCBs, and thiazide or thiazide-like diuretics) are recommended as first-line BP-lowering medications, either alone or in combination.[122,447,484,497] An exception is the combination of two RAS blockers, which is not recommended.[498–500] For most hypertensive patients, a single-pill combination, initially containing two of these major drug classes, and initially at low dose, is recommended.[489,501,502] Doses of BP-lowering drugs are presented in the Supplementary data online, *Tables S7* and *S8*.

When BP is still uncontrolled under maximally tolerated triple-combination (RAS blocker, CCB, and diuretic) therapy, and after adherence is assessed, the patient should be considered resistant and referred to an expert centre for appropriate work-up (see *Section 7.5*). At the same time, the addition of spironolactone should be considered.[459] If spironolactone is not tolerated, eplerenone or other MRA, or beta-blockers (if not already indicated), should be considered. Eplerenone may need to be dosed higher (50–200 mg) for effective BP lowering. In a meta-analysis, eplerenone 25 mg did not lower BP.[503] Due to the shorter time of action than spironolactone, eplerenone may need to be administered twice daily for treating hypertension. An alternative to MRA as fourth-line treatment for BP lowering is the use of beta-blockers for persons who do not already have a compelling indication. A vasodilating beta-blocker (e.g. labetalol, carvedilol, or nebivolol) is preferred when a beta-blocker is chosen.[504] However, we note that the BP-lowering effects of beta-blockade appears to be less potent than spironolactone in the setting of resistant hypertension.[459]

Only thereafter should hydralazine, other potassium-sparing diuretics (amiloride and triamterene), centrally acting BP-lowering medications, or alpha-blockers be considered. Given multiple side-effects, minoxidil should only be considered if all other pharmacological agents prove ineffective in resistant hypertension.[505]

As noted above, polypills combining fixed doses of BP-lowering treatment, lipid-lowering therapy and, if indicated, aspirin are effective in more general CVD prevention.[496,506–509] However, the polypill is not available for routine clinical use in many European countries.

8.3.5. A practical algorithm for intensive, effective, and tolerable blood pressure lowering with drug therapy, including considerations around single-pill combinations

The aim of the algorithm in *Figure 18* is to introduce a low-dose double- and then triple-combination strategy while monitoring tolerance

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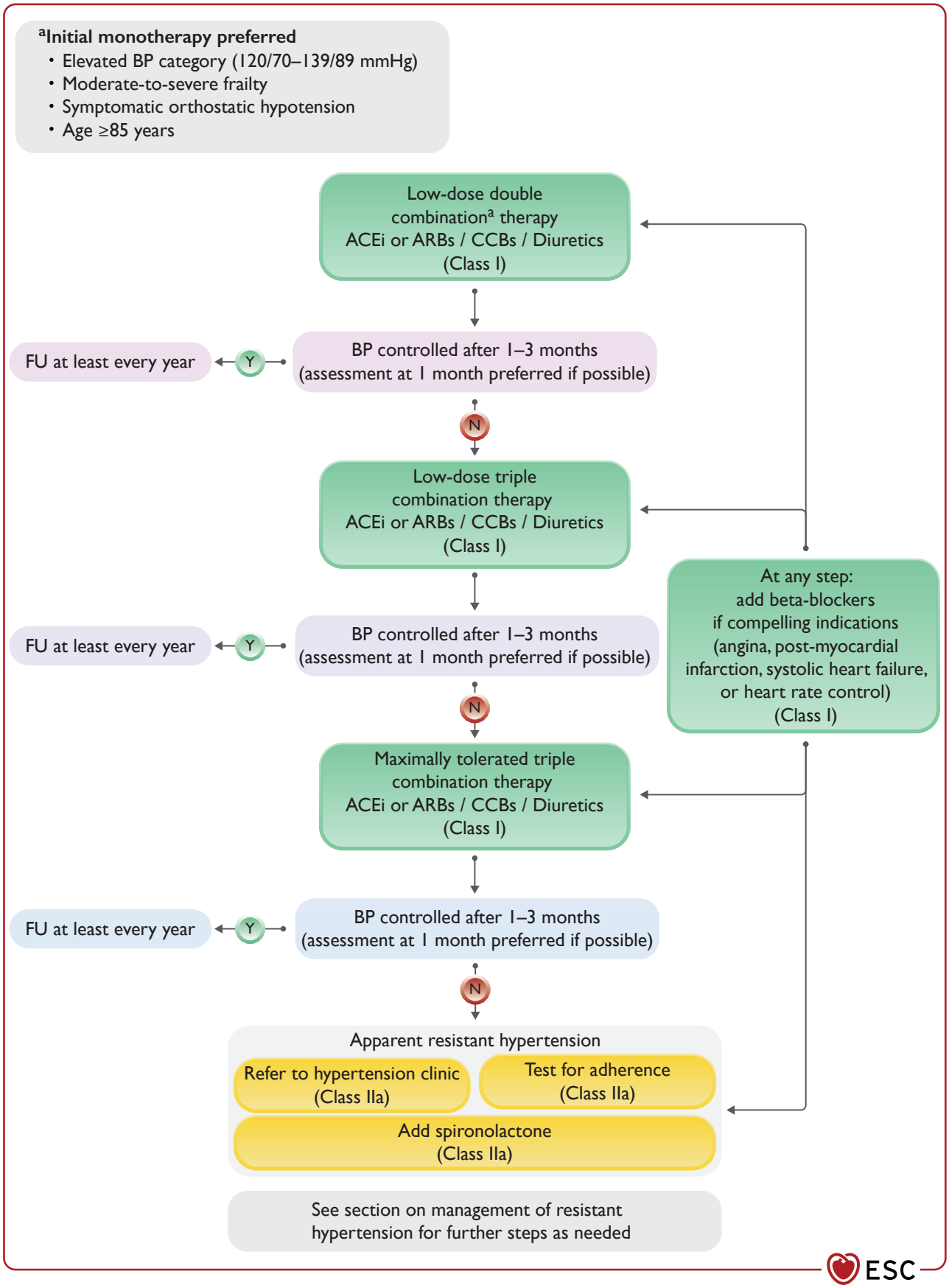

Figure 18 Practical algorithm for pharmacological blood pressure lowering. ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BP, blood pressure; CCB, calcium channel blocker; FU, follow-up.

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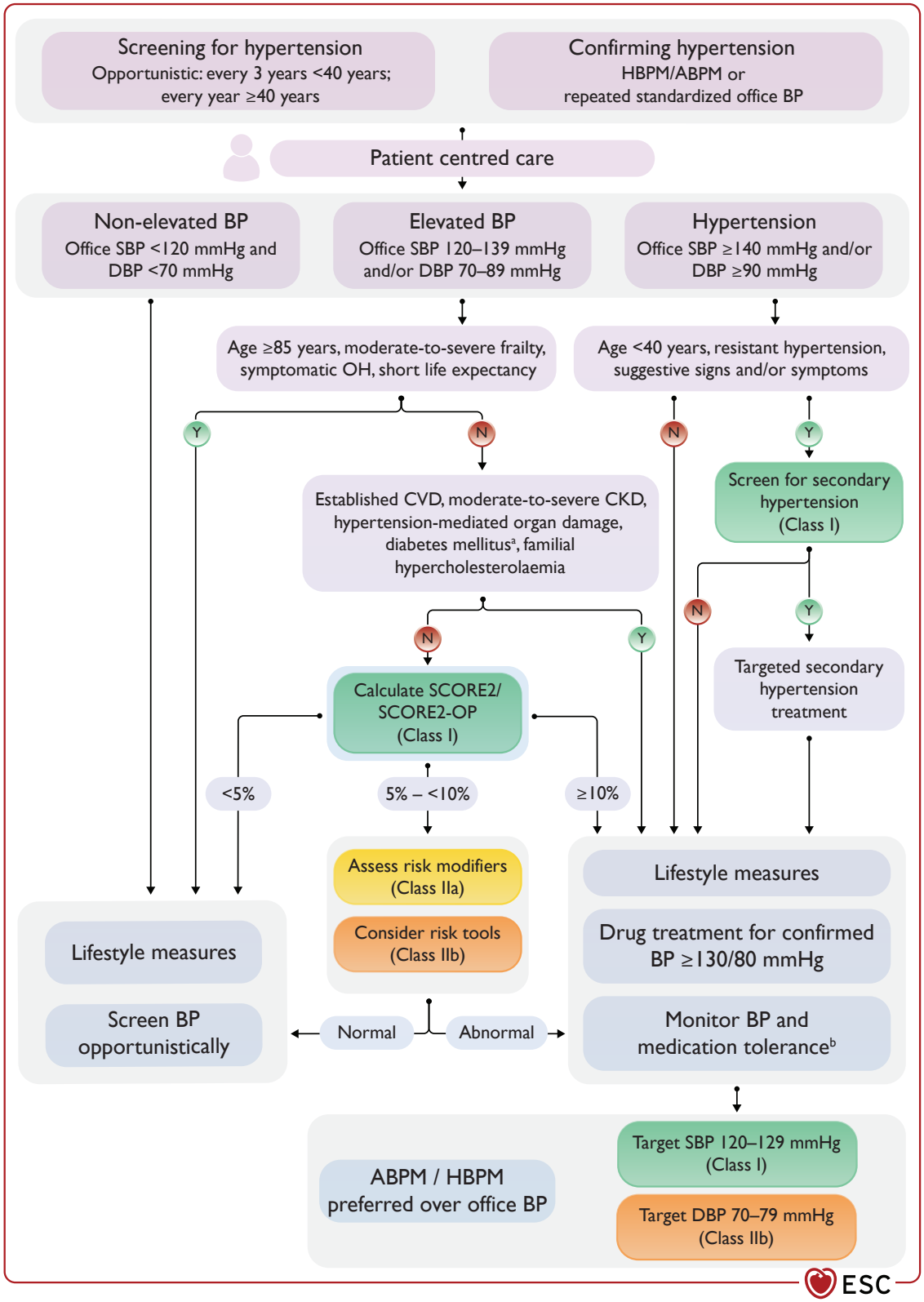

Figure 19 Central Illustration. ABPM, ambulatory blood pressure monitoring; BP, blood pressure; CVD, cardiovascular disease; CKD, chronic kidney disease; DBP, diastolic blood pressure; HBPM, home blood pressure monitoring; OH, orthostatic hypotension; SBP, systolic blood pressure; SCORE2, Systematic COronary Risk Evaluation 2; SCORE2-OP, Systematic COronary Risk Evaluation 2–Older Persons. Summary algorithm for BP classification and management. See *Section 5* for recommendations on out-of-office confirmation of the three BP categories. [a]Adults with type 2 diabetes mellitus only and younger than 60 years should be considered for SCORE2-Diabetes assessment. [b]Though scientific data demonstrate that, under research conditions, the optimal target BP is ≤120/70 mmHg, the target BP recommended by these guidelines in routine practice is 120–129/70–79 mmHg. If achieving this target is not possible, or if treatment is not well tolerated, then BP should be treated to as low as reasonably achievable. For persons with elevated BP, treatment with lifestyle measures for 3 months is first recommended, prior to considering medications.

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among patients with hypertension, and only afterwards to start up-titrating doses to maximum amounts.

Initiation with monotherapy, slower up-titration, and lower dosing should be considered in the setting of elevated BP and increased CVD risk, or in moderate-to-severe frailty, limited life expectancy, symptomatic orthostatic hypotension, or older people (aged ≥85 years). Ideally, BP should be treated to target within 3 months to retain the confidence of the patient, to ensure long-term adherence, and to reduce CVD risk.[71]

An overview of the recommended approach to BP management in all adult patients is provided in *Figure 19* (Central Illustration). Also, of note, teleconsultation, multidisciplinary or nurse-led care, or patient self-monitoring can help with achieving BP control in certain healthcare systems.[75,510,511]

8.3.6. Timing of blood pressure-lowering drug treatment

Current evidence does not show benefit of diurnal timing of BP-lowering drug administration on major CVD outcomes.[512] It is important that medication is taken at the most convenient time of day to improve adherence. Patients should also be encouraged to take medications at the same time each day and in a consistent setting, to help ensure adherence.[246,513]

Recommendation Table 16 — Recommendations for pharmacological treatment of hypertension (see Evidence Tables 27, 28, and 29)

Recommendations	Class[a]	Level[b]
Among all BP-lowering drugs, ACE inhibitors, ARBs, dihydropyridine CCBs, and diuretics (thiazides and thiazide-like drugs such as chlorthalidone and indapamide) have demonstrated the most effective reduction of BP and CVD events, and are therefore recommended as first-line treatments to lower BP.[122,446]	I	A
It is recommended that beta-blockers are combined with any of the other major BP-lowering drug classes when there are other compelling indications for their use, e.g. angina, post-myocardial infarction, heart failure with reduced ejection fraction, or for heart rate control.[122,448–450]	I	A
It is recommended to take medications at the most convenient time of day for the patient to establish a habitual pattern of medication taking to improve adherence.[246,513]	I	B
Given trial evidence for more effective BP control vs. monotherapy, combination BP-lowering treatment is recommended for most patients with confirmed hypertension (BP ≥140/90 mmHg) as initial therapy. Preferred combinations are a RAS blocker (either an ACE inhibitor or an ARB) with a dihydropyridine CCB or diuretic. Exceptions to consider include patients aged ≥85 years, those with symptomatic orthostatic hypotension, moderate-to-severe frailty, or elevated BP (systolic BP 120–139 mmHg or diastolic BP 70–89 mmHg) with a concomitant indication for treatment.[131,480,483,484,489]	I	B
In patients receiving combination BP-lowering treatment, fixed-dose single-pill combination treatment is recommended.[484,489,501,502,514]	I	B
If BP is not controlled with a two-drug combination, increasing to a three-drug combination is recommended, usually a RAS blocker with a dihydropyridine CCB and a thiazide/thiazide-like diuretic, and preferably in a single-pill combination.[489]	I	B
If BP is not controlled with a three-drug combination, adding spironolactone should be considered.[459]	IIa	B
If BP is not controlled with a three-drug combination and in whom spironolactone is not effective or tolerated, treatment with eplerenone instead of spironolactone,[503] or the addition of a beta-blocker if not already indicated[459] and, next, a centrally acting BP-lowering medication,[515] an alpha-blocker,[515] hydralazine, or a potassium-sparing diuretic should be considered.[516]	IIa	B
Combining two RAS blockers (ACE inhibitor and an ARB) is not recommended.[498–500,517]	III	A

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BP, blood pressure; CCB, calcium channel blocker; CVD, cardiovascular disease; RAS, renin–angiotensin system.
[a]Class of recommendation.
[b]Level of evidence.

8.4. Selecting patients for pharmacological blood pressure-lowering treatment

Commencement of BP-lowering treatment is often decided based on office BP measurements but, where possible, the present guidelines strongly recommend using out-of-office BP measurement for confirming elevated BP and hypertension (see *Section 5*). As detailed in *Section 6*, an office BP of <120/70 mmHg is categorized in these guidelines as non-elevated BP.

When a patient is diagnosed with confirmed hypertension (sustained BP ≥ 140/90 mmHg), starting BP-lowering treatment is recommended irrespective of CVD risk, which should consist of a simultaneous combination of lifestyle interventions and pharmacological therapy. Lifestyle interventions are crucial as an initial treatment step, and must be strongly emphasized with the patient, but concurrent pharmacological therapy is recommended. This concurrent initiation of lifestyle and pharmacological therapy should not give patients the impression that lifestyle changes are of lesser importance, and the patient should be counselled that these lifestyle changes may allow subsequent discontinuation or down-titration of medication, which can be used as motivation to persist with lifestyle changes. After treatment initiation, the patient should be seen frequently (e.g. every 1–3 months with a GP or specialist) until BP is controlled. BP should be controlled, preferably within 3 months (see also *Section 6* and algorithm *Figure 18*). If lifestyle changes are effective in BP lowering, pharmacological treatments may subsequently be down-titrated or stopped as appropriate.

When office BP is 120–139/70–89 mmHg, the patient is considered as having elevated BP, and further CVD risk stratification is recommended to guide therapy (*Table 14*).

- In patients with elevated BP who are not at increased risk for CVD (10-year CVD risk <10%) and do not have other high-risk conditions or risk modifiers, BP-lowering lifestyle measures are recommended.

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Table 14 Initiation of blood pressure-lowering treatment based on confirmed blood pressure category and cardiovascular disease risk

Blood pressure (mmHg)	Non-elevated BP (<120/70)	Elevated BP (120/70 to 139/89)		Hypertension (≥140/90)
Risk		(a) All adults with SBP 120–129 mmHg (b) SBP 130–139 AND 10-year estimated CVD risk <10% AND no high-risk conditions or risk modifiers or abnormal risk tool tests	(a) SBP 130–139 AND high-risk conditions (e.g. established CVD, diabetes mellitus, CKD, FH or HMOD) (b) SBP 130–139 AND 10-year estimated CVD risk ≥10% (c) SBP 130–139 AND 10-year estimated CVD risk 5% - <10% AND risk modifiers or abnormal risk tool tests	Assumed all at sufficiently high risk to benefit from pharmacological treatment
Treatment	Lifestyle measures for prevention Screen BP and CVD risk opportunistically	Lifestyle measures for treatment Monitor BP and CVD risk yearly	Lifestyle measures and pharmacological treatment (after 3-month delay). Monitor BP yearly once treatment control is established	Lifestyle measures and pharmacological treatment (immediate) Monitor BP yearly once treatment control is established
Target (mmHg)	**Maintain BP <120/70**	**Aim BP 120–129/70–79 mmHg[a]**		

BP, blood pressure; CKD, chronic kidney disease; CVD, cardiovascular disease; DBP, diastolic blood pressure; FH, familial hypercholesterolaemia; HMOD, hypertension-mediated organ damage; SBP, systolic blood pressure.
[a]Caution in adults with orthostatic hypotension, moderate-to-severe frailty, limited life expectancy, and older patients (aged ≥85 years).

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While there are not enough outcomes data for a drug recommendation in lower CVD risk adults with elevated BP, there is some evidence to suggest that relative treatment effects of BP lowering are similar across a wide range of predicted risk categories, including among those with a <10% risk.[518] Therefore, while we do not provide a formal recommendation for this, if initial lifestyle measures are not successful after 6–12 months, drug treatment might be discussed on an individual basis among lower CVD-risk adults when BP is between 130/80 and <140/90 mmHg.[518]

- In patients with elevated BP (office BP of 120–139/70–89 mmHg) who are at sufficiently high risk for CVD (e.g. 10-year CVD risk ≥ 10%) or in the presence of high-risk conditions or borderline 10-year CVD risk (5% - <10%) combined with risk modifiers or abnormal risk tool tests, BP-lowering lifestyle measures should be initiated for 3 months. Following this, pharmacological therapy is recommended for persons with confirmed BP of ≥130/80 mmHg, when these lifestyle changes have not worked or are not being implemented (*Section 8.2*) Prompt addition of pharmacological therapy, if needed by 3 months, should be emphasized, to avoid therapeutic inertia.[519] For those with BP of 120–129/70–79 mmHg, ongoing and intensified lifestyle intervention is preferred.

The above recommendations apply to all individuals with elevated BP, irrespective of age. However, recognizing the lack of conclusive evidence and added risk of side effects among certain subgroups, the task force also recommends that, among patients with elevated BP, BP-lowering treatment should always be started based on individual clinical judgment and shared decision-making.

In addition, consideration of BP-lowering drug treatment should be deferred until BP is >140/90 mmHg in the following settings: pre-treatment symptomatic orthostatic hypotension, age ≥85 years, clinically significant moderate-to-severe frailty, and/or limited predicted lifespan (<3 years) due to high competing risk (including eGFR < 30 mL/min/1.73 m^2). Patients with elevated BP in these settings are less likely to obtain sufficient net benefit from BP-lowering drug therapy or to tolerate intensive drug therapy. *Section 9* contains more information on the treatment of specific subgroups, including older and frail adults.

Recommendation Table 17 — Recommendations for initiating blood pressure-lowering treatment (see Evidence Tables 30–32)

Recommendations	Class[a]	Level[b]
In adults with elevated BP and low/medium CVD risk (<10% over 10 years), BP lowering with lifestyle measures is recommended and can reduce the risk of CVD.[119,120,348,408,411,520,521]	I	B
In adults with elevated BP and sufficiently high CVD risk[c], after 3 months of lifestyle intervention, BP lowering with pharmacological treatment is recommended for those with confirmed BP ≥130/80 mmHg to reduce CVD risk.[116,522]	I	A
It is recommended that in hypertensive patients with confirmed BP ≥140/90 mmHg, irrespective of CVD risk, lifestyle measures and pharmacological BP-lowering treatment are initiated promptly to reduce CVD risk.[116,121,122]	I	A
It is recommended to maintain BP-lowering drug treatment lifelong, even beyond the age of 85 years, if well tolerated.[523–525]	I	A
Because the benefit in reducing CVD outcomes is uncertain in these settings, and noting that close monitoring of treatment tolerance is advised, BP-lowering treatment should only be considered from ≥140/90 mmHg among persons meeting the following criteria: pre-treatment symptomatic orthostatic hypotension, age ≥85 years, clinically significant moderate-to-severe frailty, and/or limited predicted lifespan (<3 years).[131,524,526,527]	IIa	B

BP, blood pressure; CVD, cardiovascular disease.
[a]Class of recommendation.
[b]Level of evidence.
[c]10-year estimated CVD risk of ≥10%; or 10-year estimated CVD risk of 5% - ≤10% plus risk modifiers or abnormal risk tool tests; or high-risk conditions (e.g. established CVD, diabetes, moderate or severe CKD, familial hypercholesterolaemia, or hypertension-mediated organ damage).

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8.5. Intensity of blood pressure-lowering therapy and ideal treatment targets

8.5.1. Expected degree of blood pressure reduction with approved drugs

The magnitude of BP reduction achieved with the main classes of BP-lowering medications (ACE inhibitors, ARBs, dihydropyridine CCBs, diuretics, and beta-blockers) as monotherapy is similar overall.[528,529] BP reduction with standard doses of any of these five classes can be expected to be approximately 9/5 mmHg with office BP and 5/3 mmHg with ABPM.[478] These BP-lowering effects may attenuate over time.[530] Combination therapy (e.g. with three drugs at half standard dose) over the short term can lower office BP by up to 20/11 mmHg.[478,531] The reason why beta-blockers are not considered first-line BP-lowering medications (outside of compelling indications) is not because of inferior BP-lowering properties (particularly for vasodilating beta-blockers),[532] but because of inferior efficacy in reducing CVD events (particularly stroke) among patients with hypertension, and tolerance issues.[533–536]

The BP-lowering effect of each BP medication class generally increases with the dose administered, though this relationship is not linear.[537] Effects of each medication can also vary at the individual level, sometimes requiring personalization by matching the patient with the best medication for them.[490] The magnitude of BP reduction for any BP-lowering medication may increase as a function of the pre-treatment BP, which is also known as Wilders principle.[538,539]

The BP-lowering effect of pharmacological therapy is typically evident after 1–2 weeks of treatment,[540] but the maximum effect might take longer to manifest. Therefore, the advised follow-up after 1–3 months (1 month preferred with a GP or specialist) allows for assessment of tolerance/safety, but also allows enough time to gauge the full BP-lowering effect of each drug titration (see *Section 8.3.4*).

8.5.2. The ideal target of blood pressure-lowering treatment

As discussed in *Section 6*, optimal control of BP translates into CVD risk reduction, thereby reducing morbidity and mortality in the population.[116,478,541]

BP threshold is defined as the BP at which BP-lowering treatment is initiated, while BP target is the BP goal with treatment.

The BP threshold to initiate BP-lowering therapy is not necessarily the same as the recommended BP target once therapy is commenced (in other words, treatment threshold and treatment target may not be the same for a given patient). Specifically, for hypertensive patients in whom BP-lowering treatment is recommended above a baseline BP of ≥140/90 mmHg, the recommended target of BP-lowering therapy is 120–129/70–79 mmHg, provided treatment is well tolerated (see Supplementary data online). Persons with elevated BP who receive treatment are also recommended to achieve a target of 120–129/70–79 mmHg.

Therefore, the treatment target in the 2024 Guidelines is always 120–129/70–79 mmHg (but only if treatment is tolerated and with certain exceptions where more lenient targets are advised). In contrast, the treatment threshold may differ based on CVD risk, specifically in the elevated BP category. For example, in addition to hypertensive adults with BP ≥140/90 mmHg, there are individuals with an office systolic BP of 130–139 mmHg and/or diastolic BP of 80–89 mmHg who have sufficiently high CVD risk to recommend BP-lowering drug treatment.

The BP target range of 120–129/70–79 mmHg recommended in these guidelines reflects the most current evidence from contemporaneous RCTs[135,136,146,542–545] and from meta-analyses of RCTs.[131] Of note, this treatment target reduces CVD events in older adults[136,523] with evidence for efficacy of more intensive BP-lowering treatment targets established up to age 85 years.[131] Furthermore, research data indicate that, to optimally reduce CVD risk, achieving an on-treatment BP of 120/70 mmHg is the best point on the BP target range provided in our guideline recommendations (*Figure 20*). However, while we strongly considered recommending a treatment target of exactly 120/70 mmHg with out-of-office BP confirmation, we instead chose a target range of 120–129/70–79 mmHg (preferably with out-of-office BP confirmation but also allowing for office BP) for the following reasons: providing flexibility to patients and clinicians; feedback from external peer review; feedback from patients that lifestyle is preferred to medication unless BP is in the hypertensive range; the knowledge that contemporary treat-to-target intensive BP trials included only persons with baseline systolic BP of ≥130 mmHg; and a recognition that the BP values recorded under research conditions using systematic approaches to measurement (while strongly recommended by these guidelines) are not always the same as BP values recorded under routine clinical care, which can be 5–10 mmHg higher.[65,66]

In addition, the trial data confirming efficacy for our recommended treatment target of 120–129/70–79 mmHg do not necessarily apply to moderately-to-severely frail adults who were generally excluded from trials. Furthermore, the data supporting this BP target among adults aged >85 years are inconclusive.[131] Frailty can occur at different ages and is, together with tolerability of BP-lowering treatment, an important characteristic when considering the BP target for a given patient. Accordingly, personalized BP-lowering treatment should be instituted in people aged ≥85 years and/or those with significant frailty. Recommended indicators of frailty in guiding BP-lowering treatment are given in *Section 9*.

Several important nuances are highlighted and warrant consideration prior to implementing the new BP target of 120–129/70–79 mmHg among patients receiving BP-lowering therapy:

- Evidence for a systolic BP-lowering treatment target of 120–129 mmHg is strong (Class I, level of evidence A).
- Evidence for a specific diastolic BP-lowering treatment target is less strong in those who are treated to a systolic target of 120–129 mmHg. While most adults treated to a systolic BP target of 120–129 mmHg will also achieve a diastolic BP of 70–79 mmHg, not all will.[543,546] Furthermore, adults who achieve systolic BP control are generally at low relative risk for CVD, even when diastolic BP is 70–90 mmHg.[547,548] Nonetheless, due in part to the known higher risk of isolated diastolic hypertension among younger adults,[549] the task force agreed that it is reasonable to target an on-treatment diastolic BP of 70–79 mmHg among patients with diastolic BP of ≥80 mmHg who are already at the systolic BP target of 120–129 mmHg (Class IIb, level of evidence C).
- The task force acknowledges the 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice,[170] which take a stepwise approach to their recommendations for BP-lowering treatment. The 2021 Guidelines recommended an on-treatment systolic BP target of 130–139 mmHg as the first step and then—based on patient preferences, risk, and frailty—to aim for a target on-treatment systolic BP of <130 mmHg as the second step. While we recognize the potential value of this two-step approach, which many clinicians may choose to follow, the current guidelines emphasize one on-treatment BP target (120–129/70–79 mmHg, provided treatment is tolerated). This one-step approach is based on the evidence, and motivated to discourage therapeutic inertia around BP lowering. As an illustrative example of the latter concern for therapeutic inertia, an

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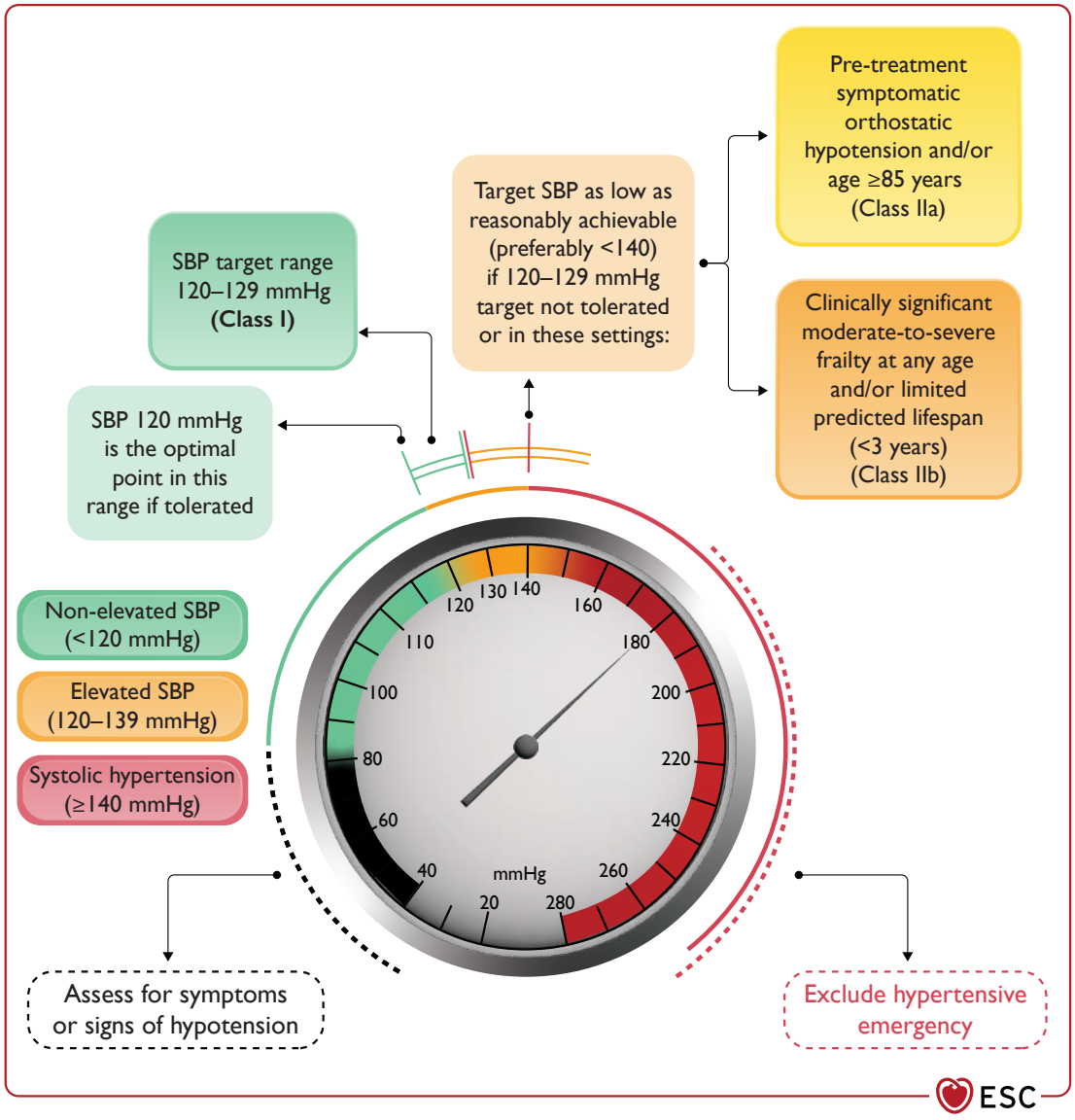

Figure 20 Systolic blood pressure categories and treatment target range. BP, blood pressure; SBP, systolic blood pressure.

on-treatment systolic BP of 135 mmHg (office) may be considered reasonable when reviewing the 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice but, we hope, will not be considered reasonable to those who follow the updated 2024 ESC Guidelines presented here. Specifically, it is well established that an on-treatment systolic BP of 135 mmHg is not optimal relative to more intensive control.[116,131,135,136,445,542,543,545]

- We acknowledge that the results from RCTs cannot always be extrapolated to routine clinical care. In addition, we recognize the increased risk of side effects among patients receiving more intensive BP-lowering treatments, compared with traditional BP targets.[545,550] Accordingly, an important caveat to our treatment target of 120–129/70–79 mmHg is the recommendation to pursue this target only when treatment is well tolerated. In cases where BP-lowering treatment is not well tolerated and a target of 120–129/70–79 mmHg is not possible, it is recommended to follow the 'as low as reasonably achievable' (ALARA) principle, by targeting treatment to a BP that is as low as reasonably achievable.
- In addition to adults with significant frailty and/or who are ≥85 years of age, the evidence for a BP-lowering treatment target of 120–129/70–79 mmHg may also not generalize to patients with: (i) pre-treatment symptomatic orthostatic hypotension, (ii) limited predicted lifespan (e.g. <3 years),[527] and/or (iii) high levels of competing risk for non-CVD death including CKD with eGFR < 30 mL/min/1.73 m^2) (see *Section 9*).

Finally, as outlined in *Section 5*, these guidelines endorse a 'trust but verify' approach to office BP measurements, and, where possible, confirming BP with accurate out-of-office BP measurements (ABPM, HBPM) is recommended prior to starting treatment, to monitor the treatment effect of BP-lowering medication.

8.5.3. Personalizing treatment strategies

Though promising, there is little to no evidence to date from CVD outcome trials to use novel biomarkers for individualizing BP-lowering treatment.[551,552] Nonetheless, different patient groups

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Recommendation Table 18 — Recommendations for blood pressure targets with treatment (see Evidence Table 34)

Recommendations	Class[a]	Level[b]
To reduce CVD risk, it is recommended that treated systolic BP values in most adults be targeted to 120–129 mmHg, provided the treatment is well tolerated.[22,122,131,523,541]	I	A
In cases where BP-lowering treatment is poorly tolerated and achieving a systolic of 120–129 mmHg is not possible, it is recommended to target a systolic BP level that is 'as low as reasonably achievable' (ALARA principle).[22,122,131,523,541]	I	A
Because the CVD benefit of an on-treatment systolic BP target of 120–129 mmHg may not generalize to the following specific settings, personalized and more lenient BP targets (e.g. <140 mmHg) should be considered among patients meeting the following criteria: pre-treatment symptomatic orthostatic hypotension, and/or age ≥85 years.[131]	IIa	C
Because the CVD benefit of an on-treatment systolic BP target of 120–129 mmHg may not generalize to the following specific settings, personalized and more lenient BP targets (e.g. <140/90 mmHg) may be considered among patients meeting the following criteria: clinically significant moderate-to-severe frailty at any age, and/or limited predicted lifespan (<3 years).	IIb	C
In cases where on-treatment systolic BP is at or below target (120–129 mmHg) but diastolic BP is not at target (≥80 mmHg), intensifying BP-lowering treatment to achieve an on-treatment diastolic BP of 70–79 mmHg may be considered to reduce CVD risk.	IIb	C

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ALARA, as low as reasonably achievable; BP, blood pressure; CVD, cardiovascular disease.
[a]Class of recommendation.
[b]Level of evidence.

can be identified, and medication initiation can be tailored to pre-existing conditions, such as diabetes mellitus, CKD, AF, post-myocardial infarction, heart failure, metabolic syndrome, and proteinuria/albuminuria (*Section 9*). History of previous side effects and possible and compelling indications also need to be considered when selecting treatment (see Supplementary data online, *Tables S9 and S10*). Cardioselective beta-blockers may be used in low dose in chronic asthma,[553,554] in line with their use in patients with heart failure with chronic asthma.

For considerations of BP-lowering treatment among specific patient populations of interest, including different racial/ethnic populations, see *Section 9*.

8.5.4. Duration and monitoring of drug therapy

BP-lowering treatment is usually chronic, often lifelong. This raises the question of long-term efficiency, long-term side effects, adherence, and persistence with therapy. While BP-lowering therapies typically provide an overall durable effect, some attenuation of effect may be seen over time.[66,530] First-line BP-lowering medication classes appear to be safe for long-term use.[555–557] Once BP is controlled, at least a yearly follow-up is advised. Because of the known temporal variability in BP[558,559] and medication efficacy in the long term,[530] medication changes may be necessary over time (see Supplementary data online).

Recommendation Table 19 — Recommendations for follow-up in patients with treated hypertension (see Evidence Table 33)

Recommendation	Class[a]	Level[b]
Once BP is controlled and stable under BP-lowering therapy, at least a yearly follow-up for BP and other CVD risk factors should be considered.	IIa	C

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BP, blood pressure; CVD, cardiovascular disease.
[a]Class of recommendation.
[b]Level of evidence.

8.6. Device-based blood pressure lowering

Several device-based therapies designed to lower BP have been investigated.[560,561] To date, the best evidence exists for catheter-based renal denervation.

8.6.1. Catheter-based renal denervation

Sympathetic nervous system overactivity contributes to the development and progression of hypertension.[562] Renal denervation aims to interrupt afferent and efferent sympathetic nerves in the adventitia and perivascular tissue of the renal arteries.[563] The 2018 ESC/ESH Guidelines on the management of arterial hypertension did not recommend the use of device-based therapies for routine treatment of hypertension, unless in the context of clinical studies and RCTs.[1] This was based on negative data using first-generation radiofrequency catheters (see Supplementary data online).

More recent data from sham-controlled trials investigating second-generation radiofrequency and ultrasound catheters demonstrated a BP-lowering efficacy in a broad range of patients, with and without concomitant BP-lowering medications, including those with resistant hypertension.[564–568] Long-term, non-randomized, follow-up data from the Global Symplicity Registry,[569] Symplicity HTN-3 trial,[570] Spyral HTN-ON MED pilot trial,[571] and A Study of the Recor Medical Paradise System in Clinical Hypertension (RADIANCE-HTN) SOLO trial[572] indicate a sustained BP-lowering effect for up to 3 years. A single-centre open-label study suggested sustained BP reductions up to 10 years.[573] These data also highlight a potentially important advantage of renal denervation, namely that the BP-lowering effect of this intervention might be 'always on', making this approach attractive for patients with suboptimal medication adherence.[254] Some patients may prefer a one-off procedure rather than taking daily medications chronically and may request renal denervation.

Of significance, there are no reported procedure-related serious safety signals in the first- and second-generation trials beyond the usual risk of femoral arterial access procedures (noting that most trials to date were not powered for safety outcomes and that the task force could find no published meta-analysis data on exact rates of major bleeding and major femoral artery vascular access complications after renal denervation procedures). However, the rate of major bleeding

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and major femoral artery vascular access complications for coronary angiography using a femoral approach is typically reported as 1%–4%[574,575] but has been reported as 5%–10% in some studies.[575] Trials investigating radial access for renal denervation are currently ongoing (ClinicalTrials.gov identifier: NCT05234788). After renal denervation, there is a 0.25%–0.5% rate of renal artery stenosis/dissection requiring stenting.[576] Long-term follow-up data up to 3 years have not suggested worsening of renal function beyond the expected rates in patients with hypertension with mildly-to-moderately reduced renal function.[569,577] Of note, sham-controlled trials to date excluded patients with severely reduced kidney function at baseline.[564,566–568]

Despite the clear promise of renal denervation in reducing BP, there are some concerns that warrant consideration, as we indicate in the recommendations. First, the effect of current renal denervation catheter technologies on BP lowering is relatively modest for an invasive procedure (meta-analyses report placebo-corrected systolic BP lowering of approximately 6 mmHg on office BP assessment and 4 mmHg on 24 h ABPM).[578] As such, the average BP-lowering effect of renal denervation appears no more than for one standard BP-lowering medication. Accordingly, many adults undergoing renal denervation will likely require ongoing, post-procedural, BP-lowering drugs.

Second, the cost-effectiveness of renal denervation has not been fully established. Since effects of current renal denervation technologies are similar to that of one standard BP-lowering medication, most of which are generic, it is difficult to see a scenario where renal denervation could be proven cost-effective for most patients. An exception might be patients who are at very high risk of CVD events and who have uncontrolled BP due to resistant hypertension (with or without non-adherence).[579,580]

Third, there are no adequately powered outcomes trials demonstrating that renal denervation reduces CVD events and is safe in the long term. While observational reports have suggested associations between renal denervation and reduced risk for CVD events,[581,582] these observational data have major inferential limitations including a significant potential for confounding. While BP lowering is typically a good surrogate for CVD benefit, there is no guarantee that this is true with renal denervation and, furthermore, off-target effects independent of BP could influence CVD and other adverse-event rates after the procedure. Because of the lack of outcomes trials, renal denervation cannot reach the Class I indication threshold set by this task force. Arguments that outcomes trials will not be funded are insufficient to influence guideline recommendations. However, it is hoped that the position of these guidelines will motivate industry to sponsor the necessary renal denervation outcomes trials.

Fourth, related to the lack of outcomes data, the potentially 'always on' effect of renal denervation could backfire if late complications emerge. Medications causing complications or side effects can simply be stopped and replaced with alternative medications when such problems emerge; this is not true with renal denervation.

Fifth, the impact of scaling up renal denervation on usual cardiac department catheterization laboratory workflows is of some concern. Specifically, it is important that renal denervation procedures do not delay timely access to other elective procedures with proven efficacy in reducing CVD outcomes.[583]

Sixth, there is still no direct evidence to gauge whether renal denervation procedures are successful and that the kidneys are denervated and do not reinnervate over time.[584] Relatedly, the concept of responders and non-responders to renal denervation (and the hypothesis that predictors of response might be found to help identify patients most suitable for the procedure) is questioned. Medical interventions, including drugs, are naturally subject to inter-individual variability in response.[567] Additionally, there are few examples in medicine of consistent and clinically useful predictors of treatment response for medical conditions that have complex genetic and environmental underpinnings (i.e. conditions like hypertension).

A multidisciplinary hypertension team, including experts in hypertension and percutaneous cardiovascular interventions, is recommended to evaluate the indication and to perform the procedure.[585] Based on the available evidence, renal denervation may be considered for patients who have uncontrolled, true resistant hypertension with a three-drug combination and who express a preference to undergo renal denervation.[566,568,585] This recommendation is informed, in part, by the higher risk of CVD events in this subgroup, which represents a major unmet clinical need and which also means that cost-effectiveness considerations are likely to be optimal in this setting. In patients who are non-adherent or intolerant to multiple BP-lowering medications, particularly first-line agents, and who have high predicted CVD risk and a BP that is not at target, renal denervation may, for the same reasons, be considered if the patients express a preference to undergo renal denervation after a tailored shared decision-making process. The shared decision-making process requires that the patients are fully informed about the benefits, limitations, and risks associated with renal denervation.

Recommendation Table 20 — Recommendations for device-based treatment of hypertension (see Evidence Table 35)

Recommendations	Class[a]	Level[b]
To reduce BP, and if performed at a medium-to-high volume centre, catheter-based renal denervation may be considered for resistant hypertension patients who have BP that is uncontrolled despite a three BP-lowering drug combination (including a thiazide or thiazide-like diuretic), and who express a preference to undergo renal denervation after a shared risk-benefit discussion and multidisciplinary assessment.[564,566–568,586–590]	IIb	B
To reduce BP, and if performed at a medium-to-high volume centre, catheter-based renal denervation may be considered for patients with both increased CVD risk and uncontrolled hypertension on fewer than three drugs, if they express a preference to undergo renal denervation after a shared risk-benefit discussion and multidisciplinary assessment.[564,566–568,586–590]	IIb	A
Due to a lack of adequately powered outcomes trials demonstrating its safety and CVD benefits, renal denervation is not recommended as a first-line BP-lowering intervention for hypertension.	III	C
Renal denervation is not recommended for treating hypertension in patients with moderate-to-severely impaired renal function (eGFR <40 mL/min/1.73 m^2) or secondary causes of hypertension, until further evidence becomes available.	III	C

BP, blood pressure; CVD, cardiovascular disease; eGFR, estimated glomerular filtration rate.
[a]Class of recommendation.
[b]Level of evidence.

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8.6.2. Other devices

Most device-based therapies investigated for BP-lowering therapy in hypertension have been aimed at modulating the autonomic nervous system activity (baroreflex activation therapy, endovascular baroreflex amplification therapy, and carotid body ablation).[560] Cardiac neuromodulation therapy aims to lower left ventricular preload by variably altering the atrioventricular interval using a dual-chamber, rate-responsive, implantable pulse generator in patients indicated for implantation or replacement of a dual-chamber pacemaker.[591,592] Some, though not all, of these devices have shown promising results in non-randomized, single-arm studies. A sham-controlled trial investigating central iliac arteriovenous stent anastomosis was terminated early after longer-term follow-up data indicated an increase in heart failure in the stent group.[561] Therefore, the use of these device-based therapies is not recommended for routinely treating hypertension until further evidence regarding their safety and efficacy becomes available (see Supplementary data online).

8.7. Unintended and potentially harmful consequences of blood pressure lowering and implications for treatment targets

8.7.1. Adverse effects of blood pressure-lowering medications

8.7.1.1. Symptomatic adverse effects

BP-lowering medications have multiple side effects, which may be more common in females.[536,593,594] Although generally well tolerated, common side effects include headaches, cough, dizziness or light-headedness, diarrhoea or constipation, fatigue, ankle swelling, and erectile problems, depending on the drug class (see Supplementary data online, *Table S9*).[536,550,593–597]

In randomized trials of adults aged >60 years, the overall rate of symptomatic BP-lowering drug withdrawal was higher than the rate of placebo withdrawal (approximately 15% vs. 5%).[593] A systematic review, which included 280 638 participants in 58 RCTs, reported no evidence for an increased relative risk of falls in those taking BP-lowering drugs.[550] There was, however, an increased relative risk of mild hyperkalaemia, acute kidney injury, hypotension, and syncope. Furthermore, very frail adults were excluded from BP-lowering trials, which is relevant because such patients are more prone to adverse effects and polypharmacy (see Supplementary data online).[596]

8.7.1.2. Renal effects

A systematic review reported an increased risk of acute kidney injury and hyperkalaemia associated with BP-lowering treatment.[550] Analyses of outcomes by specific drug class showed that drugs affecting the RAAS were more likely to be associated with acute kidney injury and hyperkalaemia.[550]

Patients with significant CKD tend to be excluded from RCTs.[137,545,598] It is important to remember these exclusion criteria, and that patients with CKD are more likely to suffer from resistant hypertension, when extrapolating the results of more intensive BP lowering to patients with moderate-to-severe CKD (see *Section 9*).[599]

8.7.1.3. Erectile dysfunction

Older classes of BP-lowering drugs (including diuretics, beta-blockers, and centrally acting medications) are associated with erectile dysfunction.[600] However, newer classes have neutral effects.[601] Angiotensin receptor antagonists may have beneficial effect on erectile function.[602]

8.7.2. Pill burden and non-adherence

More intensive treatment of elevated BP and hypertension may be associated with an increased risk of polypharmacy and pill burden, which are themselves associated with non-adherence.[603,604] Single-pill, fixed-dose drug combinations can help to reduce pill burden and are recommended to improve adherence (refer to *Section 8.3.4*).

Increased intensity of BP lowering (while ultimately cost reducing in terms of CVD reduction)[605] can also result in higher upfront direct and indirect healthcare costs, with more people requiring medication and higher demand for technology-based adherence strategies, which can be challenging to implement, especially in resource-poor settings.[604]

8.7.3. Potentially harmful consequences of blood pressure lowering for frail older people

Unintended consequences of BP lowering (hypotension, syncope, falls) can be hazardous for frail older people in particular.[606] Retrospective studies have shown that adults aged >75 years from the general population, who would have met the criteria for inclusion in the Systolic Blood Pressure Intervention Trial (SPRINT), had a rate of injurious falls and syncope that was nearly five times that of the standard care group in the trial. This suggests that healthy participant bias may have contributed to the findings of SPRINT and other similar BP-lowering trials, and that the results may not fully generalize to older adults in more routine clinical care.[607]

Patients' functional ability should be considered in addition to age to help negate any unintended consequences of BP lowering in a frailer cohort. Despite their chronological age, older patients with hypertension who are fit and can independently carry out activities of daily living will benefit from guideline-directed treatment similar to younger cohorts.[131] However, tailoring treatment targets and treatment plans for frail older patients is necessary to avoid unintended consequences. This should include assessing frailty, including cognitive status, risk of falls, propensity for symptomatic orthostatic hypotension, polypharmacy, and other comorbid conditions.[608,609] Of note, and as detailed in *Section 9*, some data indicate a benefit of more intensive BP-lowering on cognitive function.[523,610,611] For those with loss of function but preserved activities of daily living, a more detailed geriatric assessment is required to explore the risks and benefits of treatment, as well as considerations for tailoring therapeutic strategies where appropriate. For patients who are both functionally impaired and unable to carry out activities of daily living, the therapeutic goals of hypertension treatment should be personalized, and medications discontinued where appropriate (see *Section 9.3*).[596]

8.7.4. Clinical inertia in blood pressure lowering

The fear of serious adverse events with BP-lowering medications is often cited as a reason for clinical inertia, although the evidence to date from meta-analyses of RCTs suggests these side-effect concerns may be exaggerated.[550,612] However, RCTs often select populations with less frailty and multimorbidity who are more likely to tolerate treatment.[613] Consequently, fewer adverse effects might be reported than would be expected in the general population. It remains up to individual clinicians to initiate shared decision-making with each patient, especially patients in vulnerable groups and those who have experienced previous adverse events, weighing up potential benefits against risks of treatment.[614,615]

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9. Managing specific patient groups or circumstances

9.1. Young adulthood (18–40 years)

9.1.1. Definition and epidemiology

In the present guidelines 'young adulthood' is defined as age 18–40 years. The prevalence of hypertension in young adults is increasing in men and women.[616–618] Unhealthy lifestyle, gender, obesity, and socio-economic factors contribute.[617,619–621] Hypertension-attributable CVD burden in young adults, evaluated as mortality or years of living with disability, has increased in the last decades, especially in low- and middle-income countries and in men.[622] Hypertension awareness, treatment, and control in young adults is lower than in other age categories, a result driven by worse control in young men.[623]

Systolic and diastolic hypertension and isolated diastolic hypertension are associated with increased CVD risk in the young (see Supplementary data online).[624] Isolated systolic hypertension in the young is discussed in *Section 9.4*.

9.1.2. Secondary hypertension in young adulthood

Secondary hypertension is more frequent in younger than in later-onset hypertension, with a prevalence of 15%–30% in hypertensive young adults reported from some referral centres.[625,626] Major causes of secondary hypertension include drug-induced hypertension (e.g. oestrogen-progesterone oral contraceptives; cold medication) and primary aldosteronism. The use of recreational drugs/substances, as well as supplements and energy drinks should be investigated (see *Section 7*).

Combined oestrogen-progesterone contraceptives are among the most common causes of drug-induced hypertension in young women,[627,628] and should not be used in hypertensive women unless there is no other method available or acceptable to the patient.[629] Conversely, progestin-only contraceptives are generally considered safe in women with hypertension.[89,630,631] Fibromuscular dysplasia should be considered as a cause of secondary hypertension in young women,[626,632] whereas primary aldosteronism, the most common form of secondary hypertension, is equally common in different age classes.[316] Screening for secondary hypertension is thus recommended in young adults with hypertension. However, in obese young adults, primary hypertension is more common, though OSAS should also be considered in this instance.[633]

9.1.3. Measurement and management of blood pressure in young adults

Out-of-office BP measurement is recommended in young adults for confirming diagnosis, since the white-coat phenomenon occurs in the young.[634] Because of the lower absolute CVD risk in this age category compared with older adults, hard-endpoint randomized trials of BP lowering in young adults have not been performed. However, since relative risk reduction by BP-lowering treatment is homogeneous in any age group, including those <55 years old,[131] young adults with suitable indications are also expected to benefit from BP-lowering therapy. The hypertension management algorithm based on CVD risk proposed in *Section 6* is not fully applicable in young adults, since SCORE2 has not been validated for individuals <40 years old. Even risk stratification based on lifetime risk assessment does not apply to very young adults (e.g. 20–30 years of age).[128] In the absence of established CVD, diabetes mellitus, familial hypercholesterolaemia, and moderate or severe CKD a BP-lowering treatment initiation threshold of office 140/90 mmHg is appropriate in most young adults. However, HMOD assessment may be considered in patients aged <40 years to stratify individuals with elevated BP into a higher risk category. For example, arterial stiffness better reclassifies CVD risk in individuals aged <50 years than in older individuals.[28,215] Echocardiographic left ventricular mass also maintains its added reclassification and discrimination on top of risk scores in young adults (see *Section 6* for discussion of risk modifiers).[289]

Irrespective of cardiovascular risk, all young adults with elevated BP are recommended to follow lifestyle guidance for BP lowering. A discussion about family planning should be taken with young women of childbearing potential at each visit.[635,636]

Adherence to treatment is low in young adults, <50% in some studies.[251] Therefore, communicating the importance of adherence, education, and follow-up clinics is important. (see *Sections 7* and *11*).

Recommendation Table 21 — Recommendations for managing hypertension in young adults (see Evidence Tables 36 and 37)

Recommendations	Class[a]	Level[b]
Comprehensive screening for the main causes of secondary hypertension is recommended in adults diagnosed with hypertension before the age of 40 years, except for obese young adults where it is recommended to start with an obstructive sleep apnoea evaluation.[316,626]	I	B
Since SCORE2 has not been validated for individuals <40 years, screening for HMOD may be considered in such young individuals with elevated BP without other increased CVD risk conditions to identify additional individuals for possible medical treatment.[28,215]	IIb	B

BP, blood pressure; CVD, cardiovascular disease; HMOD, hypertension-mediated organ damage; SCORE2, Systematic COronary Risk Evaluation 2.
[a]Class of recommendation.
[b]Level of evidence.

9.2. Pregnancy

9.2.1. Definition and epidemiology

Hypertension in pregnancy is typically defined as systolic BP of ≥140 mmHg and/or diastolic BP of ≥90 mmHg, measured using repeated BP readings in the office or hospital on two separate occasions or ≥15 min apart in severe hypertension (≥160/110 mmHg).[1,637,638]

Hypertension in pregnancy is the second leading cause of maternal death after maternal peri-partum haemorrhage.[639] Approximately 7% of pregnancies are complicated by hypertension, of which 3% are due to pre-eclampsia and around 1% are chronic or pre-existing hypertension.[640] Women with a history of hypertensive disorders during pregnancy are at increased risk of subsequent hypertension and CVD.[640–642]

9.2.2. Classifying hypertension in pregnancy

Hypertension in pregnancy includes:

- **Chronic hypertension:** precedes pregnancy, develops before 20 weeks of gestation, persists for >6 weeks post-partum, and may be associated with proteinuria.
- **Gestational hypertension:** develops after 20 weeks of gestation and usually resolves within 6 weeks post-partum.

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- **Antenatally unclassifiable hypertension:** BP is first recorded after 20 weeks of gestation, and hypertension is diagnosed but it is unclear if chronic or not; reassessment is necessary 6 weeks post-partum.
- **Pre-eclampsia:** gestational hypertension accompanied by new-onset: (i) proteinuria (>0.3 g/day or ≥30 mg/mmol ACR), (ii) other maternal organ dysfunction, including acute kidney injury (serum creatinine ≥ 1 mg/dL), liver dysfunction (elevated transaminases > 40 UI/L with or without right upper quadrant or epigastric abdominal pain), neurological complications (convulsions, altered mental status, blindness, stroke, severe headaches, and persistent visual scotomata), or haematological complications (platelet count < 150 000/μL, disseminated intravascular coagulation, haemolysis), or (iii) uteroplacental dysfunction (such as foetal growth restriction, abnormal umbilical artery Doppler waveform analysis, or stillbirth).[643] The only cure for pre-eclampsia is delivery, which is recommended at 37 weeks' gestation, or earlier in high-risk cases. Of note, proteinuria is not mandatory for diagnosing pre-eclampsia but is present in about 70% of cases.[644] Also, as proteinuria may be a late manifestation of pre-eclampsia, it should be suspected when *de novo* hypertension is accompanied by headache, visual disturbances, abdominal pain, or abnormal laboratory tests, specifically low platelets and/or abnormal liver function.

Other potential causes for high BP, including pain and anxiety, must be excluded when treating hypertension during pregnancy.

9.2.3. Measuring blood pressure in pregnancy

See *Section 5.5.1* for information on BP measurement approaches in pregnancy.[645] It is important to restate here that oscillometric devices tend to under-estimate the true BP and are unreliable in severe pre-eclampsia; only a few have been validated in pregnancy. Importantly, only the relatively few devices validated for measuring BP in pregnancy and pre-eclampsia should be used (https://stridebp.org).

9.2.4. Investigating hypertension in pregnancy

Basic laboratory investigations include urinalysis, blood count, haematocrit, liver enzymes, serum creatinine, and serum uric acid. Serum uric acid is increased in pre-eclampsia and identifies women at increased risk of adverse maternal and foetal outcomes in hypertensive pregnancies.[646]

All pregnant women should be assessed for proteinuria in early pregnancy (e.g. 11–14 weeks' gestation).[647] A dipstick test of ≥1+ should prompt further investigations, including ACR, which can be quickly determined in a single spot-urine sample.[648] An ACR of <30 mg/mmol (<0.3 mg/mg) can rule out proteinuria.[649] Higher values should prompt 24 h urine collection.

In one study, 10% of pregnant women with chronic hypertension had secondary hypertension (estimated to affect 0.24% of all pregnancies).[650] Secondary hypertension during pregnancy is associated with an increased risk of adverse outcomes.[650] The most common cause of secondary hypertension during pregnancy is CKD. The onset of hypertension during the first trimester, at the peak of human chorionic gonadotropin (HCG) secretion, should prompt consideration of primary aldosteronism.[651] Phaeochromocytoma in pregnant women is rare (0.002% of all pregnancies) but highly morbid.[652,653]

9.2.5. Preventing hypertension and pre-eclampsia

Low-to-moderate-intensity exercise, especially if supervised and initiated during the first trimester of pregnancy, decreases the incidence of developing gestational hypertension.[654] As such, after consultation with their obstetrician, all pregnant women should participate in physical activity, unless contraindicated.[655] Factors indicating risk of pre-eclampsia are discussed in the Supplementary data online.

Women at high or moderate risk of pre-eclampsia should be advised to take 100–150 mg of aspirin daily at bedtime from gestational weeks 12–36.[647,656,657]

Oral calcium supplementation of 0.5–2 g daily is recommended for preventing pre-eclampsia in women with low dietary intake of calcium (<600 mg daily).[658,659]

9.2.6. Treatment initiation and blood pressure targets

Acute management of BP in pre-eclampsia and eclampsia is detailed in *Section 10.4*.

Meta-analyses have found no evidence for an increased risk for delivering small-for-gestational-age babies in pregnant women with mild hypertension receiving BP-lowering medications.[660] Despite a historical paucity of trial data, previous European guidelines[1,89] recommended initiating BP-lowering drug treatment (i) in all women with persistently elevated office BP of ≥150/90 mmHg, and (ii) in women with gestational hypertension (with or without proteinuria), pre-existing hypertension with superimposed gestational hypertension, or hypertension with subclinical HMOD, when office BP is >140/90 mmHg.

In the CHAP trial, treating pregnant women with chronic hypertension and BP of ≥140/90 mmHg reduced the occurrence of pre-eclampsia with severe features, and reduced medically indicated pre-term birth <35 weeks, compared with only treating severe hypertension (BP ≥ 160/105 mmHg).[88] Tight BP control (target diastolic BP < 85 mmHg) compared with less-tight BP control (target diastolic BP < 100 mmHg) reduces the incidence of subsequent severe maternal hypertension (BP ≥ 160/110 mmHg), but not foetal or other maternal outcomes in women with mild hypertension at baseline (diastolic BP of 85–105 mmHg).[661]

Treatment with BP-lowering drugs in all pregnant women with confirmed BP of ≥140/90 mmHg is recommended to reduce the progression to severe hypertension and the related risks for adverse pregnancy outcomes.[660,661] In women with pre-existing and gestational hypertension with and without pre-eclampsia, we recommend lowering BP below 140 mmHg for systolic and to 80–90 mmHg for diastolic BP.[661] Evidence to support a BP target as low as 120–129/70–79 mmHg is lacking in pregnancy, though such evidence exists for non-pregnant patients receiving BP-lowering medication.

9.2.7. Managing mild hypertension in pregnancy (office blood pressure 140–159/90–109 mmHg)

RAS inhibitors are not recommended in pregnancy due to adverse foetal and neonatal outcomes. The BP-lowering drugs of choice are: beta-blockers (most data are available for labetalol, a non-selective beta-blocker that also acts as an alpha-blocker in higher doses; metoprolol and bisoprolol are also considered safe), dihydropyridine CCBs (most data are available for nifedipine, which is generally considered first choice, also felodipine, nitrendipine, amlodipine, and isradipine can be used), and methyldopa.[662,663] A meta-analysis suggests that beta-blockers and CCBs are more effective than methyldopa in preventing severe hypertension.[660] Of note, however, atenolol should be avoided, as it is associated with foetal growth restriction.[664,665] Methyldopa has been associated with an increased risk of post-partum depression and caution is therefore advised both intra-partum and post-partum.[637]

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Hydralazine can be particularly effective for severe hypertension in pregnancy and can be administered intravenously in hypertensive admissions (*Section 10*).[666–668] While thiazide diuretics in pregnancy have limited safety data and should be used with caution, other diuretics such as furosemide are not contraindicated and may be necessary in some situations (see Supplementary data online).[669]

9.2.8. Managing severe hypertension in pregnancy (>160/110 mmHg)

Acute onset of severe hypertension persisting for more than 15 min is considered a hypertensive emergency in pregnancy and is covered in *Section 10.4.2*.

9.2.9. Managing blood pressure post-partum

For women with hypertension during pregnancy, BP should be measured within 6 h of delivery and, if possible, daily for at least a week after discharge from the hospital.[637] Post-partum hypertension is common in the first week and associated with prolonged hospitalization.[670]

Women with hypertension in pregnancy are at increased risk of chronic hypertension,[671] CKD,[672] and CVD.[177,223,640] The relative risk of chronic hypertension is highest in the first 6 months following delivery, motivating regular screening in these women.[673] Women with gestational hypertension, especially those with pre-eclampsia, have higher risk of masked hypertension.[674] BP measurements, ideally including out-of-office measurements, urine analyses, and CVD risk assessment, should at least be performed 6–12 weeks, 6 months, and 12 months post-partum and, after that, annually. Recent data indicate the potential utility of self-monitoring of BP during the busy early post-partum period.[675]

All BP-lowering drugs are excreted into breast milk.[637] Except for propranolol, atenolol, acebutolol, and nifedipine, most drugs are excreted in very low concentrations in breast milk (see Supplementary data online, *Table S11*).[637]

9.2.10. Risk of recurrence of hypertensive disorders in a subsequent pregnancy

About 20%–30% of women with hypertensive disorders in a previous pregnancy will experience recurrence in a subsequent pregnancy.[676,677] The earlier the onset of hypertension in the first pregnancy, the higher the risk of recurrence in a subsequent pregnancy.[677]

Further details on managing hypertension and other cardiovascular disorders in pregnancy are available elsewhere.[89,637]

Recommendation Table 22 — Recommendations for managing hypertension in pregnancy (see Evidence Tables 38–40)

Recommendations	Class[a]	Level[b]
In women with gestational hypertension, starting drug treatment is recommended for those with confirmed office systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg.[661]	I	B
In pregnant women with chronic hypertension, starting drug treatment is recommended for those with confirmed office systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg.[88,660,661,678]	I	B
In women with chronic and gestational hypertension, it is recommended to lower BP below 140/90 mmHg but not below 80 mmHg for diastolic BP.	I	C
Dihydropyridine CCBs (preferably extended-release nifedipine), labetalol, and methyldopa are recommended first-line BP-lowering medications for treating hypertension in pregnancy.	I	C
In consultation with an obstetrician, low- to moderate-intensity exercise is recommended in all pregnant women without contraindications to reduce the risk of gestational hypertension and pre-eclampsia.[654,655]	I	B
Systolic BP ≥160 mmHg or diastolic BP ≥110 mmHg in pregnancy can indicate an emergency, and immediate hospitalization should be considered.	IIa	C
HBPM and ABPM should be considered to exclude white-coat and masked hypertension, which are more common in pregnancy.[679]	IIa	C
RAS blockers are not recommended during pregnancy.[680,681]	III	B

ABPM, ambulatory blood pressure monitoring; BP, blood pressure; CCB, calcium channel blocker; HBPM, home blood pressure monitoring; RAS, renin–angiotensin system.
[a]Class of recommendation.
[b]Level of evidence.

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9.3. Very old age (≥85 years), frailty, multimorbidity, and polypharmacy

9.3.1. Definition of frailty

The most common definition of frailty is an age-associated, biological syndrome characterized by decreased biological reserves, due to dysregulation of several physiological systems.[682] This puts an individual at risk when facing physiological stressors, and is associated with poor outcomes, such as disability, hospitalization, and death.[683] The estimated prevalence of frailty in people aged >65 years is 7%–16% and is greater in women than in men.[684,685] Although the main determinant of frailty is age, chronological age must be differentiated from biological age.[686] An older patient can be fit and robust while a multimorbid young patient can be frail. Using multiple drugs may have more unpredictable effects on BP in older patients, because of increased competition for underlying mechanisms responsible for their degradation and elimination, and because the ability of the baro-[687] and chemo-reflex[688] systems in maintaining a steady treated BP level can decline with ageing.

With respect to BP, two issues compound interpretation of the frailty literature. First, frailty on its own is a strong predictor of mortality and cardiovascular complications[689] and is accompanied by a decrease in systolic BP.[690] This raises the issue of the so-called BP J-curve (see *Section 9.8*) and reverse causality, with frailty rather than excessive BP lowering being the root cause of adverse health outcomes. Only properly randomized and controlled clinical trials can differentiate between the effects of frailty vs. overly intensive BP-lowering treatment, but unfortunately, few BP-lowering trials have included a substantial proportion of frail patients. Second, there is no consensus on how to grade frailty in day-to-day clinical practice.[606] Complex frailty scales exist for application in research,[523,691] but unless they are electronically generated,[692] they are typically not practical in routine clinical care. Nonetheless, the clinical frailty scale (*Figure 21*) is intuitive and easy to administer and has been validated against 5-year risk of death.[596,693]

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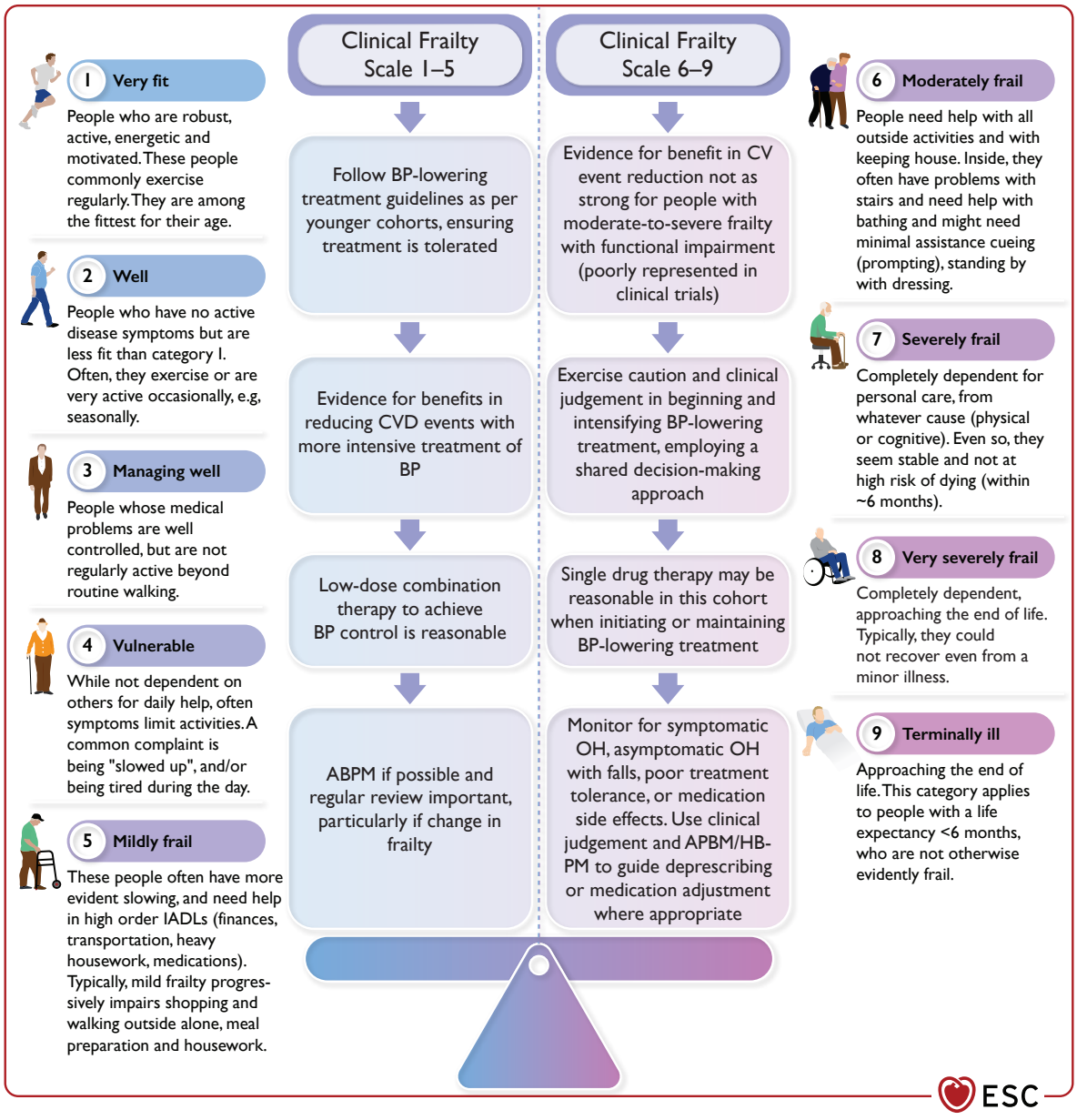

Figure 21 Frailty assessment in the management of blood pressure. ABPM, ambulatory blood pressure monitoring; BP, blood pressure; CV, cardiovascular; CVD, cardiovascular disease; HBPM, home blood pressure monitoring; IADLs, independent activities of daily living; OH, orthostatic hypotension. Adapted from Rockwood *et al.*[693]

9.3.2. Randomized controlled trials of blood pressure lowering in frail older patients

Few adults aged ≥85 years have been included in trials.[131] In addition, generalizing data from RCTs to very frail patients may not be possible.[692,694–697] However, the currently available evidence from RCTs has not demonstrated weakening of the benefits of BP-lowering treatment (i.e. no effect modification) among frailer patients enrolled in these trials, although these participants likely had no more than mild frailty (see Supplementary data online).[523,694,698]

In the absence of robust randomized evidence, several observational studies have suggested that lowering BP might not be warranted or even be harmful in patients with significant frailty or multimorbidity, particularly when BP is not very high. For instance, a systematic review and meta-analysis of non-randomized studies that investigated associations between BP and risk of mortality in older patients found evidence for interaction by frailty status, suggesting that lowering BP might be harmful in this patient group.[699] However, as noted above and in *Section 9.8*, these observational J-curve findings are unreliable when guiding clinical care, as unidentified biases potentially confound the results. For instance, in addition to reverse causality, stiffness of the large arteries is associated with both low diastolic BP and increased mortality.[697] In addition, absolute CVD risk increases with age, indicating that fewer older than younger patients with hypertension may need to be treated to prevent one adverse health outcome.[700]

Therefore, given the totality of evidence from clinical trials,[523,694,701,702] very old and frail patients with hypertension should not be denied the potential benefits of BP-lowering treatment down to a target of 120–129/70–79 mmHg. However, personalized decision-

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making should be a priority in the very old and frail. Together with management of BP, a major consideration should also be whether reversible causes of frailty can be addressed,[609] e.g. by treating underlying comorbidities or undergoing supervised muscle-strengthening physiotherapy or supervised exercise and co-ordination and balance training.[703]

9.3.3. Starting blood pressure-lowering treatment in very old or frail patients

All patients must be fully informed about the benefits and risks of starting BP-lowering treatment, so that their preference is considered. Among 34 hypertension guidelines, 18 recommended 150 mmHg as the systolic goal in frailer and/or older patients, but four endorsed systolic targets <130 mmHg or <120 mmHg.[704] Treatment can be started with a long-acting dihydropyridine calcium channel antagonist.[596,705] To achieve BP control, an ACE inhibitor, or if contraindicated, an ARB, can also be used. Thereafter, low-dose thiazides or thiazide-like diuretics are preferred unless there is a specific contraindication, such as gout, orthostatic hypotension, or disturbed micturition (including micturition syncope).[596,705] Beta-blockers are less desirable as they reduce heart rate, cause fatigue, and increase the systolic pulse wave amplitude, which is insufficiently buffered in stiff central elastic arteries. Vasodilating beta-blockers and direct vasodilators (e.g. hydralazine and minoxidil) are associated with increased risk of orthostasis. Though data are conflicting,[706] alpha-blockers are also considered less desirable as they appear to be associated with an increased risk of orthostasis and falls in very old patients (aged ≥85 years).[707,708] Alpha-1 blockers, such as doxazosin, prazosin, terazosin (also used to treat prostate symptoms), are particularly prone to causing orthostasis.[98] Once the appropriate combination is found, a combination tablet with variable composition of two agents may optimize adherence. Starting with combination therapy is not advised in most very old and/or frail patients, unless BP is very high.

9.3.4. Maintaining blood pressure lowering in very old or frail patients

If very old and frail patients tolerate BP-lowering treatment well, there is no automatic need to deprescribe or discontinue treatment; however, this should be kept under review. In the case of progressive frailty, systolic BP tends to drop,[709] such that deprescription of a BP-lowering drug might become necessary. To identify candidate drugs for deprescribing, a patient's current medications should be reviewed to identify BP-lowering drugs that may have become contraindicated due to concomitant prescriptions or newly developed comorbidities.[705] To help guide deprescription of BP-lowering agents, ABPM can be used to detect orthostatic hypotension or a highly variable BP not buffered by autonomic nervous reflexes.[687,688]

Recommendation Table 23 — Recommendations for managing hypertension in patients who are very old or frail (see Evidence Table 41)

Recommendations	Class[a]	Level[b]
It is recommended that treatment of elevated BP and hypertension among older patients aged <85 years who are not moderately to severely frail follows the same guidelines as for younger people, provided BP-lowering treatment is well tolerated.[131,523,524]	I	A
It is recommended to maintain BP-lowering drug treatment lifelong, even beyond the age of 85 years, if well tolerated.[523–525]	I	A
Because the benefit in reducing CVD outcomes is uncertain in these settings, and noting that close monitoring of treatment tolerance is advised, BP-lowering treatment should only be considered from ≥140/90 mmHg among persons meeting the following criteria: pre-treatment symptomatic orthostatic hypotension, age ≥85 years, clinically significant moderate-to-severe frailty, and/or limited predicted lifespan (<3 years).[131,524,526,527]	IIa	B
As the safety and efficacy of BP treatment is less certain in individuals with moderate or severe frailty, clinicians should consider screening older adults for frailty using validated clinical tests; frail patients' health priorities and a shared-decision approach should be considered when deciding on BP treatments and targets.[523,524,613,710]	IIa	C
When initiating BP-lowering treatment for patients aged ≥85 years, and/or with moderate-to-severe frailty (at any age), long-acting dihydropyridine CCBs or RAS inhibitors should be considered, followed if necessary by low-dose diuretic if tolerated, but preferably not a beta-blocker (unless compelling indications exist) or an alpha-blocker.[711]	IIa	B
If BP drops with progressing frailty, deprescription of BP-lowering medications (and other drugs that can reduce BP, such as sedatives and prostate-specific alpha-blockers) may be considered.[712]	IIb	C

BP, blood pressure; CCB, calcium channel blocker; CVD, cardiovascular disease; RAS, renin–angiotensin system.
[a]Class of recommendation.
[b]Level of evidence.

9.4. Isolated systolic and diastolic hypertension

9.4.1. Definition of isolated systolic hypertension

Isolated systolic hypertension is typically defined as systolic BP of ≥140 mmHg with a diastolic BP of <90 mmHg. While isolated systolic hypertension is uncommon in younger patients,[713] it is the most common type of hypertension in older patients; >80% of untreated patients with hypertension aged >60 years have isolated systolic hypertension.[714]

9.4.2. Isolated systolic hypertension, risk factors, and ageing

Systolic BP increases with age in men and women until the eighth decade of life, while diastolic BP gradually increases up until the fifth or sixth decade of life, after which it either plateaus or decreases. As a result, the pulse pressure (the difference between the systolic and diastolic BP) gradually widens from middle age.[34] These BP changes are related to increased aortic stiffening with age.[715,716]

Since most older patients with hypertension have isolated systolic hypertension, and since with advancing age, risk of CVD events is driven by systolic rather than diastolic BP,[717] management of isolated systolic hypertension in older adults is broadly in line with that of combined systolic-diastolic hypertension seen in younger adults.[718] Early isolated

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systolic hypertension studies used systolic BP treatment targets of 160 or 150 mmHg.[718] However, results from the SPRINT and the Strategy of Blood Pressure Intervention in Elderly Hypertensive Patients (STEP) trials (mean BP at study entry of 140/78 mmHg and 146/82 mmHg, respectively, indicating that many of the patients had isolated systolic hypertension) confirm that lower systolic BP targets are effective in reducing CVD events in patients with isolated systolic hypertension (see Supplementary data online).[135,136]

Since relative risk reduction by BP-lowering treatment is homogeneous in any age group, whereas absolute risk reduction is larger with advancing age,[131] therapeutic inertia in older patients with isolated systolic hypertension should be avoided (see *Section 9.3*). As noted earlier, beta-blockers should be avoided in patients with isolated systolic hypertension or more generally with arterial stiffness, as they increase stroke volume (given the lower heart rate).[218]

9.4.3. Isolated systolic hypertension in young adults

In young adults (<40 years old), the presence of isolated systolic hypertension poses different pathophysiological and clinical considerations. In young patients with isolated systolic hypertension, arterial stiffness[713] and relative risk of CVD events[624] appear to be similar to those without isolated systolic hypertension and lower than young adults with combined systolic-diastolic hypertension and isolated diastolic hypertension. Indeed, younger patients with isolated systolic hypertension appear to comprise a heterogeneous group.[719] For these reasons, it might be reasonable to assess central BP and arterial stiffness in these individuals, as recommended by other scientific societies.[720,721] Out-of-office BP measurement is recommended to exclude white-coat hypertension, which is often associated with isolated systolic hypertension in the young.[634]

9.4.4. Isolated diastolic hypertension

Isolated diastolic hypertension is defined as a systolic BP of <140 mmHg with a diastolic BP of ≥90 mmHg. The isolated diastolic hypertension phenotype is more commonly seen in younger adults and, particularly, younger adults with obesity or other metabolic derangements.[722,723] In older adults with this phenotype, consideration should be given to whether the diastolic BP was accurately measured.[724]

Patients with isolated diastolic hypertension appear to have a slightly increased relative risk for CVD of 5%–30%.[548,723,725] However, because patients with isolated diastolic hypertension are younger, they tend to have few events, and very large samples are required to show this association. Furthermore, because the absolute risk for CVD among these individuals is low (typically <10% over 10 years), it is less clear if isolated diastolic hypertension should prompt initiation of BP-lowering medication, particularly among persons in whom baseline systolic BP is already at the target of 120–129 mmHg.[723] Irrespective, patients with isolated diastolic hypertension should be followed up, as they are at increased risk for systolic hypertension.[723]

Finally, it is also worth noting that when a patient achieves a target systolic BP of 120–129 mmHg with BP-lowering treatment, there is little to no high-quality trial evidence that further intensifying BP-lowering medication to achieve both systolic BP of <120 mmHg and also diastolic BP of <70 mmHg improves CVD prognosis.[547,723]

9.5. Orthostatic hypotension with supine hypertension

Patients with orthostatic hypotension need not be hypotensive and indeed, many have supine elevated BP or supine hypertension. Furthermore, many patients with orthostatic hypotension are asymptomatic. Orthostatic hypotension is present in around 10% of all adults with hypertension and is defined as a drop in BP of ≥20/10 mmHg after rising from either a sitting or lying position to a standing position (see *Section 5.5.3*).[97,99] Assessment for orthostatic hypotension should be timed to occur at 1 and/or 3 min after standing. Because seated to standing assessment can lead to under-detection of orthostatic hypotension, it is preferable, where possible, to test for orthostatic hypotension using a supine (lying) to standing assessment (see *Section 5*).[56,98,726]

Assessing for orthostatic hypotension is important in managing adults with elevated BP or hypertension for several reasons. First, findings of trials linking more intensive BP control to improved outcomes may not generalize to patients with orthostatic hypotension, particularly when it is severe in magnitude (standing systolic BP < 110 mmHg[97]) and/or symptomatic. Second, orthostatic hypotension may be associated with symptoms that may limit the patient's tolerability of more intensive BP-lowering approaches. Third, orthostatic hypotension may be associated with an increased risk of adverse effects commonly co-attributed to pharmacological BP lowering (such as hospitalizations for hypotension).[727] Fourth, orthostatic hypotension is associated with increased risk for CVD.[728]

However, the frequency of orthostatic hypotension is not increased in the more intensive BP-lowering arms of randomized trials compared with the less intensive BP-lowering arms.[726,727,729,730] As such, and in contrast to common belief, it does not appear that more intensive treatment of BP (which almost always requires more BP-lowering medication) worsens orthostatic hypotension. In contrast, there is some evidence that more intensive treatment of hypertension may actually reduce the risk of orthostatic hypotension.[730,731]

The aetiology of orthostatic hypotension may be considered as neurogenic or non-neurogenic, with the latter being far more common.[99] Patients with orthostatic hypotension may have underlying neurodegenerative diseases, diabetes, B12 deficiency,[732] renal failure, dehydration, prolonged recumbency, deconditioning, and triggering medications (like alpha-blockers, beta-blockers, diuretics, nitrates, antidepressants, and antipsychotics). Of note, ACE inhibitors, ARBs, and dihydropyridine CCBs are examples of BP-lowering medications that appear to have less impact on orthostatic hypotension, and their adverse impact, if any, on orthostatic hypotension typically occurs in the first 2 weeks or so after starting or intensifying treatment.[733]

Managing patients with supine hypertension and orthostatic hypotension is a common clinical conundrum. More detailed reviews on this topic are available elsewhere.[98,99] The approach to managing orthostatic hypotension should be non-pharmacological at first. Patients with orthostatic hypotension should be asked to change position slowly, maintain adequate hydration, and avoid alcohol and large meals. Compression stockings, crossing legs while standing, and abdominal binders may also help and should be trialled.[734,735] Abdominal heating pads and a head-up bed position can reduce supine (typically nocturnal) hypertension, which may reduce nocturnal diuresis and daytime orthostatic hypotension.[736]

The treatment of orthostatic hypotension among those with supine hypertension is not to automatically down-titrate BP-lowering medications. Rather, reversible causes should be sought and treated (including discontinuation of offending medications), and patients requiring BP-lowering medication should be switched to BP-lowering medications that are less likely to cause orthostatic hypotension. When symptoms are disabling and the above interventions do not help, particularly in neurogenic orthostatic hypotension, the best evidence exists for midodrine to reverse orthostatic hypotension, and this may be given in conjunction with ongoing BP-lowering medications when supine hypertension exists.[99] An alternative option to midodrine is droxidopa, though this is less readily available. Specialist referral is prudent when persons with supine hypertension are prescribed these orthostatic hypotension treatments, as these agents can increase supine BP more than standing BP.

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Recommendation Table 24 — Recommendations for managing hypertension in patients with orthostatic hypotension

Recommendation	Class[a]	Level[b]
Before starting or intensifying BP-lowering medication, it is recommended to test for orthostatic hypotension, by first having the patient sit or lie for 5 min and then measuring BP 1 and/or 3 min after standing.[97,99]	I	B
It is recommended to pursue non-pharmacological approaches as the first-line treatment of orthostatic hypotension among persons with supine hypertension. For such patients, it is also recommended to switch BP-lowering medications that worsen orthostatic hypotension to an alternative BP-lowering therapy and not to simply de-intensify therapy.[726,727,729,730]	I	A

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BP, blood pressure.
[a]Class of recommendation.
[b]Level of evidence.

9.6. Diabetes

9.6.1. Diabetes and elevated blood pressure/ hypertension

Patients with diabetes (both type 1 and type 2) often have elevated BP or hypertension, and are about twice as likely to suffer a major CVD event over the medium to long term compared with those without diabetes.[737] Diabetes is also a major cause of microvascular events, such as retinopathy and nephropathy.[738,739] Although the risk of CVD in patients with diabetes varies by screening and diagnostic methods,[740,741] as well as with the presence of other CVD risk factors,[740,742] on average, patients with diabetes are at ≥10% 10-year risk for CVD. However, formal risk estimation with the use of SCORE2-Diabetes among type 2 diabetes mellitus patients should be considered if they are aged <60 years (see *Section 6*).[164,739]

9.6.2. J-shaped curve of blood pressure and risk of cardiovascular disease in patients with diabetes

Evidence on the BP threshold and target for treatment in patients with diabetes has been subject to debate. Reports of a J-shaped association between BP and risk of CVD in diabetes,[743] and the lack of a clear benefit of treatment on cardiac outcomes at lower BP in some meta-analyses,[744–746] has led to some cautious recommendations for intensive treatment in this patient population.

An individual patient data meta-analysis by the Blood Pressure Lowering Treatment Trialists' Collaboration tested treatment effects in 103 325 patients with diabetes and provided evidence against effect modification by categories of baseline BP down to a systolic BP of 120 mmHg.[445] BP reduction in patients with diabetes is expected to reduce the risk of diabetes-associated complications including retinopathy, vasculopathy, and nephropathy (albuminuria and end-stage renal disease), which adds weight to the importance of reducing BP in these patients (see Supplementary data online).[745–747] This task force also considered the fact that a proportion of patients with diabetes have orthostatic hypotension due to diabetic neuropathy,[748] which might affect the tolerability of BP lowering.

9.6.3. Managing blood pressure in diabetes

We recommend that all patients with diabetes are offered pharmacological BP-lowering treatment with a BP target of 120–129/70–79 mmHg, if feasible and tolerated.[136,146,445,747,749–752] The task force further sees no strong evidence for a differential BP treatment targets in patients with diabetes and those without.[136,146,445,746,747] While the Action to Control Cardiovascular Risk in Diabetes (ACCORD) trial reported a null primary endpoint for more intensive BP targets in diabetes, stroke was marginally reduced.[137] Furthermore, extended follow-up of ACCORD,[750] as well as analyses of intensive BP reduction in those randomized to the standard glycaemic arm,[749] provide evidence suggesting benefit consistent with the SPRINT, STEP, and Effects of intensive Systolic blood Pressure lowering treatment in reducing RIsk of vascular evenTs (ESPRIT) trials.[136,146,545] Overall, all major BP-lowering medication classes are effective in preventing CVD in people with or without diabetes. Of note, however, albuminuria is more common in diabetes and, for this reason, ACE inhibitors and ARBs have potential advantages that may warrant consideration for BP-lowering in patients with diabetes (see Supplementary data online, *Table S10*).[753]

Evidence for modifying BP-lowering treatment in patients with pre-diabetes is somewhat limited. Furthermore, the relative effect of BP lowering is relatively consistent across different categories of BMI as a measure of obesity.[754,755] It is also noteworthy that elevated BP itself may increase the risk of diabetes,[755] emphasizing the potential role of BP lowering in preventing diabetes in addition to preventing CVD. Among the major classes of BP-lowering drugs, ACE inhibitors and ARBs are effective in preventing new-onset diabetes and can be considered in patients at risk of diabetes and who are indicated for BP-lowering therapy.[164,755]

Recommendation Table 25 — Recommendations for managing hypertension in patients with diabetes

Recommendations	Class[a]	Level[b]
In most adults with elevated BP and diabetes, after a maximum of 3 months of lifestyle intervention, BP lowering with pharmacological treatment is recommended for those with confirmed office BP ≥130/80 mmHg to reduce CVD risk.[445,749]	I	A
BP-lowering drug treatment is recommended for people with pre-diabetes or obesity when confirmed office BP is ≥140/90 mmHg or when office BP is 130–139/80–89 mmHg and the patient is at predicted 10-year risk of CVD ≥10% or with high-risk conditions, despite a maximum of 3 months of lifestyle therapy.[445]	I	A
In persons with diabetes who are receiving BP-lowering drugs, it is recommended to target systolic BP to 120–129 mmHg, if tolerated.[136,146,445,747,749–752]	I	A

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BP, blood pressure; CVD, cardiovascular disease.
[a]Class of recommendation.
[b]Level of evidence.

9.7. Chronic kidney disease

9.7.1. Relationship between hypertension and chronic kidney disease

Approximately 850 million people worldwide have CKD, with >80% of them hypertensive, and the prevalence is expected to rise to 1.56 billion

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by 2025.[756–759] The pathogeneses of hypertension and CKD are closely entwined.[760] Resistant hypertension, masked hypertension, HMOD, and higher night-time BP are common in patients with CKD.[761] CVD is one of the largest contributors to mortality in patients with CKD, with hypertension being a major risk factor.[760,762]

For the purposes of these guidelines, adults with moderate-to-severe CKD and elevated BP are at sufficiently high risk to be considered for BP-lowering drug therapy as outlined in *Section 8* and the Central Illustration (*Figure 19*). We use Kidney Disease: Improving Global Outcomes (KDIGO) categories to define CKD-based risk, and our definition of moderate-to-severe CKD comprises persons with an eGFR of <60 mL/min/1.73 m^2 and/or albuminuria of ≥30 mg/g (≥3 mg/mmol).[19] For persons with mild CKD and elevated BP, a CVD risk assessment should be conducted before deciding on BP-lowering treatment.

9.7.2. Blood pressure lowering in chronic kidney disease

BP lowering in patients with CKD is associated with beneficial effect on CVD events and mortality.[275,763–766] BP lowering reduces progression of CKD and the incidence of end-stage renal disease, but this tends to be only in those with significant proteinuria at baseline.[766,767]

9.7.3. Managing blood pressure in chronic kidney disease

Patients with CKD should receive lifestyle advice, especially regarding reducing sodium intake. Dietary potassium supplementation recommendations are provided in *Section 8*, with caution required among persons with moderate-to-severe CKD. While exercise appears to have little effect on improving BP in patients with CKD[768] or patients on dialysis,[769] those with CKD on ACE inhibitor monotherapy have protection against adverse kidney outcomes, CVD events, cardiovascular death, and all-cause death.[770,771] Both ACE inhibitors and ARBs reduce the risk of CVD events and kidney failure compared with placebo; however, ACE inhibitors appear to do so with higher probability than ARBs.[772,773] Patients with CKD usually require combination therapy, and this should be initiated as a combination of a RAS inhibitor and a CCB or diuretic. In patients with eGFR < 30 mL/min/1.73 m^2, an adequately up-titrated loop diuretic is necessary to define resistant hypertension. Chlorthalidone, typically added to a loop diuretic, also effectively lowers BP and reduces microalbuminuria in patients with resistant hypertension with stage 4 CKD (eGFR of 15–30 mL/min/1.73 m^2).[774] The combination of an ACE inhibitor and an ARB is not recommended in CKD or any other BP-treatment scenario.

9.7.4. Blood pressure targets in chronic kidney disease

Evidence regarding BP targets in patients with CKD is complex and controversial. The 2021 KDIGO Guideline suggested that adults with elevated BP and CKD be treated to a target systolic BP of <120 mmHg, when tolerated, using standardized office BP measurement (Class of Recommendation IIb).[19] This suggestion was based, in part, on the SPRINT trial.[545] It should be noted that patients with 24 h urine protein excretion ≥ 1 g/day or eGFR < 20 mL/min/1.73 m^2 were excluded from SPRINT. In patients with CKD, after a median follow-up of 3.3 years, the hazard ratio for the primary composite cardiovascular outcome was 0.81 (95% CI 0.63–1.05) and for all-cause death it was 0.72 (95% CI 0.53–0.99). Although intensive BP lowering in SPRINT resulted in greater early decline in eGFR, there was no evidence that this reduction in eGFR attenuated the beneficial effects of the SPRINT intervention on CVD events or death.[775]

Several systematic reviews have examined the benefit of intensive BP control in patients with CKD (see Supplementary data online). Some have shown no benefit of intensive BP control on renal outcomes,[764,767] while others showed lower mortality in intensively treated vs. non-intensively treated patients.[275] Highlighting the beneficial effects of SGLT2 inhibitors in persons with CKD[776,777] and finerenone in persons with CKD and diabetes[460–462,778] is relevant, though these drugs are not currently marketed for BP-lowering effects alone.

Recommendation Table 26 — Recommendations for managing hypertension in patients with chronic kidney disease

Recommendations	Class[a]	Level[b]
In patients with diabetic or non-diabetic moderate-to-severe CKD and confirmed BP ≥130/80 mmHg, lifestyle optimization and BP-lowering medication are recommended to reduce CVD risk, provided such treatment is well tolerated.[275,766]	I	A
In adults with moderate-to-severe CKD who are receiving BP-lowering drugs and who have eGFR >30 mL/min/1.73 m^2, it is recommended to target systolic BP to 120–129 mmHg, if tolerated. Individualized BP targets are recommended for those with lower eGFR or renal transplantation.[274,779]	I	A
In hypertensive patients with CKD and eGFR >20 mL/min/1.73 m^2, SGLT2 inhibitors are recommended to improve outcomes in the context of their modest BP-lowering properties.[776,777]	I	A
ACE inhibitors or ARBs are more effective at reducing albuminuria than other BP-lowering agents and should be considered as part of the treatment strategy for patients with hypertension and microalbuminuria or proteinuria.[780–782]	IIa	B

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BP, blood pressure; CKD, chronic kidney disease, eGFR, estimated glomerular filtration rate; SGLT2, sodium–glucose co-transporter 2.
[a]Class of recommendation.
[b]Level of evidence.

9.8. Cardiac disease

9.8.1. Blood pressure thresholds and targets in patients with cardiac disease

Recommended BP thresholds for initiating BP-lowering therapy and recommended BP targets in patients receiving therapy are provided in *Sections 6* and *8*. All patients with a history of CVD (including coronary artery disease) are at increased risk of recurrent CVD. As such, these patients are recommended to be treated with BP-lowering therapy for confirmed baseline BP of ≥130/80 mmHg and the recommended treatment target BP of 120–129/70–79 mmHg, provided treatment is tolerated (see *Sections 6* and *8*). As stated in *Section 8*, it should be remembered that a systolic BP of 120 mmHg (especially by out-of-office assessment) is likely the optimal point in the target range recommended in these guidelines. In addition to considering patients with known CVD at sufficiently high risk for more intensive BP treatment targets, the

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task force considers patients with both severe valvular heart disease and symptomatic heart failure to also be at sufficiently high risk. We also note that, whether used for angina control or BP control, a beta-blocker should not be combined with a non-dihydropyridine CCB.

9.8.2. Coronary artery disease with particular reference to the blood pressure J-curve

Important considerations in patients with coronary artery disease are: (i) the BP J-curve (an observation suggesting that over-intensive BP lowering may increase CVD risk in some patients), and (ii) compelling indications for specific classes of BP-lowering medications.

The J-curve phenomenon describes increased risk for CVD observed among patients with the lowest and highest BP in the dataset, with the best CVD outcome rates typically observed among those with BP in the normal range (e.g. systolic BP of 100–120 mmHg and diastolic BP of 60–80 mmHg). For this reason, the J-curve is sometimes also called the U-curve, with both terms typically used interchangeably.[697,783,784]

However, observational data do not consistently demonstrate a BP J-curve with CVD risk.[697] It is more commonly observed among patients with established clinical CVD, such as those with coronary artery disease, or in secondary prevention cohorts.[785,786] Furthermore, the J-curve is more commonly observed when analysing diastolic BP values vs. systolic BP values, though it has been described for both.[114,697] This stronger relationship with diastolic BP has informed the hypothesis that the J-curve may be caused by reduced perfusion of major organs at low BP, which is particularly operative for diastolic BP in the coronary vasculature when considering ischaemic heart disease events (since coronary blood flow is largely confined to diastole).[786,787]

If excessive lowering of BP causes CVD events, this needs to be addressed in treatment recommendations provided by BP management guidelines. The 2018 ESC/ESH Guidelines on the management of arterial hypertension introduced, for the first time, lower bounds of BP-lowering treatment targets, implying that treatment be de-intensified for patients with on-treatment BP below these bounds (i.e. <120 mmHg systolic or <70 mmHg diastolic).[1] The 2023 ESH document also makes this argument.[788]

However, since 2018, compelling evidence has demonstrated that the BP J-curve evident in observational datasets is highly unlikely to reflect a causal process and can instead be attributed to residual confounding and/or reverse causation.[33,114,115,546,697,789–793]

Accordingly, while low BP can indicate a high-risk state, particularly among older adults and those with comorbidities, there is no evidence that this is a causal phenomenon. Indeed, if there is another indication for BP-lowering therapy (e.g. in a patient with wide pulse pressure and a baseline systolic BP of >140 mmHg but diastolic BP of <60 mmHg), the evidence suggests that such therapy should be provided if tolerated to reduce CVD risk.

There is a limit to how low BP can be treated without potentially tipping the scales in favour of CVD harm vs. CVD benefit. However, it is not clear what that limit is and how much it differs based on comorbidities. Currently, the data do not suggest that risk for CVD can be causally increased by treating any patient to the recommended intensive BP target outlined in these guidelines of as low as 120/70 mmHg. We also do not recommend stopping or de-intensifying BP-lowering medication among asymptomatic patients with on-treatment BP of <120/70 mmHg. It should be recognized, though, that there are no robust data demonstrating that an on-treatment systolic BP of <90 mmHg or an on-treatment diastolic BP of <50 mmHg is safe from a CVD perspective and there is clear potential for harm. Furthermore, it must be emphasized that the above discussion of the BP J-curve relates solely to CVD risk and does not consider the known non-CVD side effects of BP-lowering drugs, like, e.g. orthostatic hypotension, syncope, and renal injury. We do know that patients treated to a more intensive BP target of 120/70 mmHg are at increased risk for these side effects,[550] which is why these guidelines stress that this more intensive target should only be pursued among those in whom treatment is being tolerated (*Section 8*).

The second consideration in managing BP in patients with coronary artery disease is the recommended use of BP-lowering medications with compelling indications based on outcomes trials that demonstrated CVD outcomes benefits in the setting of coronary artery disease. These recommendations are provided in the recommendation table below.

9.8.3. Valvular heart disease

Most patients with both severe heart valve disease and heart failure, defined by the 2021 ESC/European Association for Cardio-Thoracic Surgery (EACTS) Guidelines for the management of valvular heart disease,[794] can be considered at increased risk of CVD. Accordingly, it is recommended they are treated with BP-lowering therapy for confirmed baseline BP of >130/80 mmHg, and their recommended target of treatment is BP of 120–129/70–79 mmHg, provided treatment is tolerated. Persons with mild-to-moderate heart valve disease should have a CVD risk assessment prior to deciding their BP-lowering treatment threshold and target.

Vasodilating ARBs/ACE inhibitors are preferable over vasodilating dihydropyridine CCBs because of the link between valvular heart disease and subsequent heart failure and given the stronger efficacy evidence for ARBs/ACE inhibitors in the setting of heart failure once manifested.[795] In aortic valve stenosis, concomitant hypertension influences both the aortic root, the aortic valve, and the left ventricular structure and function.[796] In this subgroup, treatment preferably with ARBs/ACE inhibitors should be considered. A beta-blocker may be added if BP remains >140/90 mmHg.[796,797]

9.8.4. Heart failure

Patients with symptomatic heart failure are at increased risk of CVD. Therefore, it is recommended that these patients are treated with BP-lowering therapy for confirmed baseline BP of >130/80 mmHg and their recommended treatment target is BP of 120–129/70–79 mmHg, provided treatment is tolerated and with out-of-office confirmation of on-treatment BP. Of note, many patients with systolic heart failure on maximal heart failure therapies have BP of <120/70 mmHg, and we do not recommend de-intensifying such treatment unless indicated by symptomatic side effects. Besides referencing the new evidence for ARNi and SGLT2 inhibitor therapies,[795] our 2024 recommendations for heart failure are largely unchanged from the 2018 ESC/ESH Guidelines on the management of arterial hypertension. Non-dihydropyridine CCBs should not be used in heart failure. Frailty and hypotension risk should be assessed in older heart failure patients being considered for ARNi and SGLT2 inhibitor therapies, and older patients should be closely followed to ensure they are tolerating such treatments. For more information on the management of heart failure, we direct readers to the latest ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure.[798]

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Recommendation Table 27 — Recommendations for managing hypertension in patients with cardiac disease

Recommendations	Class[a]	Level[b]
In patients with a history of myocardial infarction who require BP-lowering treatment, beta-blockers and RAS blockers are recommended as part of that treatment.[538]	I	A
In patients with symptomatic angina who require BP-lowering treatment, beta-blockers and/or CCBs are recommended as part of that treatment.[538]	I	A
In patients with symptomatic HFrEF/HFmrEF, the following treatments with BP-lowering effects are recommended to improve outcomes: ACE inhibitors (or ARBs if ACE inhibitors are not tolerated) or ARNi, beta-blockers, MRAs, and SGLT2 inhibitors.[795]	I	A
In hypertensive patients with symptomatic HFpEF, SGLT2 inhibitors are recommended to improve outcomes in addition to their modest BP-lowering properties.[795]	I	A
In patients with a history of aortic valve stenosis and/or regurgitation who require BP-lowering treatment, RAS blockers should be considered as part of that treatment.[794,796]	IIa	C
In patients with a history of moderate-to-severe mitral valve regurgitation who require BP-lowering treatment, RAS blockers should be considered as part of that treatment.[794]	IIa	C
In patients with symptomatic HFpEF who have BP above target, ARBs and/or MRAs may be considered to reduce heart failure hospitalizations and reduce BP.[795,799,800]	IIb	B

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ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; ARNi, angiotensin receptor–neprilysin inhibitor; BP, blood pressure; CCB, calcium channel blocker; HFpEF, heart failure with preserved ejection fraction; HF(m)rEF, heart failure with (mildly) reduced ejection fraction; MRA, mineralocorticoid receptor antagonist; RAS, renin–angiotensin system; SGLT2, sodium–glucose co-transporter 2.
[a]Class of recommendation.
[b]Level of evidence.

9.8.5. Heart rhythm disease (including AF)

Not all patients with heart rhythm disease, including those with AF, are at increased risk for CVD and, as such, the management of BP among patients with heart rhythm disease should be the same as for the general adult population.[443] However, there is a close relationship between increased BP and AF risk, hence, ensuring good BP control is important.[801,802] Management of heart rhythm disorders should follow recommendations in guidelines specific to these conditions.[803]

9.9. Chronic cerebrovascular disease and/or cognitive impairment

9.9.1. Role of hypertension in chronic cerebrovascular disease

Hypertension is a risk factor for chronic cerebrovascular disease through its direct effects on brain structure and microvasculature. This manifests as transient ischaemic attack (TIA) and stroke in the acute setting, but chronic hypertension can lead to covert stroke and white-matter ischaemic change over time, resulting in cognitive decline and progressive vascular dementia.[804–808] Hypertension is also associated with increased risk of Alzheimer's disease,[809] and is a risk factor for developing AF, heart failure, and CKD, all of which are associated with increased risk of developing cognitive impairment and dementia.[810–813] For the purposes of these guidelines, adults with a history of stroke or TIA and elevated BP are considered at sufficiently high risk to be considered for BP-lowering drug therapy as outlined in *Section 8* and the Central Illustration (*Figure 19*).

9.9.2. Treatment in patients with history of prior stroke or transient ischaemic attack

In patients with a prior history of TIA and ischaemic stroke, BP-lowering treatment reduces the risk of any recurrent stroke by 20%.[814–817] The aetiology of stroke can affect the degree of risk reduction with pharmacological treatment, with greater reductions observed for intracerebral haemorrhage and lacunar ischaemic stroke syndromes.[818–820]

Most prior guidelines recommend an intensive BP target in patients with a prior history of stroke, typically using combination treatment (ACE inhibitor/ARB plus either a calcium channel antagonist or a thiazide/thiazide-like diuretic), with therapy commencing immediately after TIA and within a few days of ischaemic stroke (see Supplementary data online and see *Section 10.3* for acute BP management during hospitalization for stroke).[814,815,821–824]

Regimens containing an ACE inhibitor and thiazide/thiazide-like diuretic may be superior to beta-blockers in terms of stroke risk reduction.[825,826] Regarding intensive BP control after stroke, typically targeting a systolic BP of <130 mmHg, individual trials were somewhat inconclusive, but a meta-analysis showed a reduced risk of recurrent stroke of 22% in the intensive treatment group randomized to a target systolic BP as low as 120 mmHg.[543,824,827,828] Caveats to this recommendation would be for frail patients, who have a much higher rate of stroke and recurrent stroke than the general population, and who are more sensitive to adverse effects of BP-lowering agents (see *Section 9.3*).[596,606,607,829]

9.9.3. Treatment in patients with chronic cerebrovascular disease and cognitive impairment

Treatment of hypertension represents a key mechanism for reducing the global burden of dementia at the population level.[830] Epidemiological studies have reported associations between mid-life hypertension and development of cognitive decline in later life, with, e.g. mid-life hypertension increasing relative risk of lifetime dementia by 20%–54%.[831–837] In one observational meta-analysis, an increased risk for dementia emerged with systolic BP of >130 mmHg.[831]

Evidence for lowering BP to reduce the risk of dementia is limited due to heterogeneity in populations studied, cognitive testing methods used, and the varied use of dementia or cognitive impairment or both as a primary outcome.[838,839] Findings from individual studies have mixed results (see Supplementary data online).[264,839–843] Studies on effects of BP-lowering treatment on white-matter intensities concluded that patients in the intensive-control arm had less white-matter intensity accumulation than in the standard-treatment arm.[841,844] Studies in which people with stroke and TIA were included reported a reduced risk of dementia and cognitive decline for the active-treatment group, but a mixed signal for dementia alone.[841,845] However, individual studies

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may have been under-powered and more recent meta-analyses do convincingly support efficacy in reducing dementia with BP-lowering treatment.[610,611] Indeed, these meta-analyses reported a reduced risk of incident dementia or cognitive impairment with BP lowering of 7%–13%.[610,611] While one trial suggested superiority of long-acting CCBs,[264] it is unclear if any first-line BP-lowering agent is preferable for preventing dementia and cognitive impairment.[846,847]

The role of competing risk mechanisms including orthostatic hypotension[848] and BP variability[849] may be important factors in treatment decisions for people with frailty, multimorbidity, and/or chronic cerebrovascular disease.

Recommendation Table 28 — Recommendations for managing hypertension in patients with chronic cerebrovascular disease and cognitive impairment

Recommendations	Class[a]	Level[b]
It is recommended that the BP-lowering drug treatment strategy for preventing recurrent stroke should comprise a RAS blocker plus a CCB or a thiazide-like diuretic.[820,823,825,826]	I	A
In patients with confirmed BP ≥130/80 mmHg with a history of TIA or stroke a systolic BP target of 120–129 mmHg is recommended to reduce CVD outcomes, provided treatment is tolerated.[824,827,828]	I	A

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BP, blood pressure; CCB, calcium channel blocker; CVD, cardiovascular disease; RAS, renin–angiotensin system; TIA, transient ischaemic attack.
[a]Class of recommendation.
[b]Level of evidence.

9.10. Aortopathy

9.10.1. Coarctation of the aorta

Aortic coarctation is associated with CVD in the long term, even following early surgical or percutaneous treatment. The most common complications are associated with hypertension, which is common in aortic coarctation. When aortic coarctation is not treated, patients often develop severe hypertension and HMOD (especially LVH and left ventricular dysfunction, aortopathy, and cerebrovascular complications).[850–852] No formal RCTs to define optimal medical treatment of hypertension in aortic coarctation have been conducted, therefore, patients not suitable for, or having undergone, intervention should be treated for hypertension following the core algorithm for the general population.

9.10.2. Bicuspid aortic valve-related aortopathy

Bicuspid aortic valve is the most common congenital heart disease and is sometimes associated with aortopathy or aortic coarctation. Bicuspid aortic valve disease is associated with an increased risk of valve malfunction and adverse aortic events.[853,854] This risk is exacerbated by hypertension.

Beyond aortic dilation and aneurysm formation, bicuspid aortic valve disease is also a risk factor for aortic dissection and rupture.[855] Blood pressure should be carefully monitored and controlled.[856]

9.10.3. Preventing aortic dilation and dissection in high-risk patients

A modest dilatation of the ascending aorta or aortic root is often associated with chronic hypertension and HMOD. An additional cause of aortopathy (bicuspid valve, coarctation, Marfan or other syndromes) should be considered in more severe cases.[857] Patients with aortic dilatation should have their BP optimally controlled following the core algorithm for the general hypertension population.

In patients with Marfan syndrome, prophylactic use of ARBs, ACE inhibitors, or beta-blockers may reduce complications or progression of aortic dilation.[857–860] More information is available in the 2024 ESC Guidelines for the management of peripheral arterial and aortic diseases.[861]

9.11. Different ethnic groups

Influx and settlement of migrant populations in Europe have contributed to regional population growth and changes in its composition.[862] Ethnic minority populations are disproportionally affected by hypertension and hypertension-mediated complications, compared with historically native Europeans, with data suggesting migrant women are particularly vulnerable.[536,863] In particular, hypertension is more prevalent in those of African descent.[863,864] The predominant group of European black ethnicity originates from sub-Saharan Africa,[863] but specific studies on the management and control of hypertension in this population are lacking, and data are often extrapolated from studies in the African American population.[864] This assumption requires caution, as differences likely exist between these populations in terms of CVD risk, economic, and sociological status,[865,866] as well as responses to BP-lowering drugs.[867]

Black patients have a greater prevalence of low-renin, salt-sensitive hypertension and may be more predisposed to HMOD than white patients, possibly in part due to increased vascular stiffness.[864,868,869] Salt restriction, thiazide or thiazide-like diuretics, and CCBs appear particularly useful in black patients with hypertension, whereas RAS blocker monotherapy may be less effective.[870–873] If combination therapy is needed, in a recent RCT conducted in sub-Saharan African countries, amlodipine plus either hydrochlorothiazide or perindopril proved to be equally effective and superior, respectively, to hydrochlorothiazide plus perindopril.[874] When RAS blockers are used in combination therapy, ARBs may be preferable to ACE inhibitors, as angioedema appears more common with ACE inhibitors in black patients.

Despite some recent progress,[875] data on hypertension epidemiology and management in European immigrant patients are still lacking.[863,875–877]

Recommendation Table 29 — Recommendations for managing hypertension in different ethnic groups

Recommendation	Class[a]	Level[b]
In black patients from Sub-Saharan Africa who require BP-lowering treatment, combination therapy including a CCB combined with either a thiazide diuretic or a RAS blocker should be considered.[874]	IIa	B

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BP, blood pressure; CCB, calcium channel blocker; RAS, renin–angiotensin system.
[a]Class of recommendation.
[b]Level of evidence.

9.12. Nocturnal hypertension

9.12.1. Definition

Nocturnal hypertension is defined as night-time BP of >120 mmHg systolic and/or >70 mmHg diastolic by 24 h ABPM. Nocturnal hypertension can occur as day–night sustained hypertension or isolated nocturnal hypertension (daytime BP < 135/85 mmHg on 24 h ABPM). Physiologically, BP is

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expected to decrease during sleep by 10%–20% relative to daytime BP.[878] Night-time dipping patterns are classified into four groups:[879,880]

- **Inverse dipping (riser):** nocturnal increase in BP (night-to-day ratio of >1.0).
- **Non-dipper:** reduced night-time BP dip of <10% (or night-to-day ratio of >0.9 and ≤1.0).
- **Normal dipping:** fall in night-time BP of >10% and <20% (or night-to-day ratio of 0.8 to 0.9).
- **Extreme dipping:** marked fall in night-time BP of >20% (or night-to-day ratio of <0.8).

Patients with nocturnal hypertension may be dippers or non-dippers. Of note, the long-term reproducibility of dipping patterns appears to be low.[881,882]

9.12.2. Epidemiology

Nocturnal hypertension has been observed in up to half of patients with hypertension,[883–886] and is associated with increased HMOD,[883] impaired renal function, and diabetes mellitus.[887] Nocturnal hypertension appears to be more prevalent in black[888–890] and Asian[891,892] populations. Masked uncontrolled hypertension, which occurs in 30% of patients treated for hypertension, is more often due to poorly controlled nocturnal BP than daytime BP on ABPM.[893]

Environmental factors, including sleep duration and higher humidity,[894] nocturia,[895] OSAS,[896] obesity, high salt intake in salt-sensitive patients,[897] orthostatic hypotension, autonomic dysfunction, CKD,[898–900] diabetic neuropathy/diabetes,[901] and old age[62] are associated with non-dipping. Moreover, nocturnal hypertension and absent night-time dipping pattern are more common in secondary hypertension.[902,903]

9.12.3. Night-time blood pressure as a cardiovascular disease risk factor

Nocturnal hypertension is a risk factor for adverse CVD events,[904] cerebrovascular disease, including stroke,[905] and cardiovascular mortality.[891,906,907] Night-time BP may provide more prognostic information than daytime BP, perhaps as it is less dependent on physical activities. Non-dipping[908–910] and reverse dipping (nocturnal rise in BP) may also be associated with increased CVD risk.[62,910–913] A nocturnal rise in BP is associated with an increased risk of dementia and Alzheimer's disease in older men.[914] There is also some evidence that extreme dipping, particularly in untreated patients, is associated with an increased risk for CVD events.[35,886]

9.12.4. Treatment of nocturnal hypertension

There is no reliable evidence that BP-lowering medication should be routinely dosed at bedtime. The diurnal timing of drug administration is discussed in *Section 8.3.4*. In patients with secondary hypertension, the underlying cause (OSAS, primary aldosteronism) should be treated as discussed in *Section 9.14*.

9.13. Resistant hypertension

9.13.1. Definition of resistant hypertension

Resistant hypertension is defined as BP remaining above goal despite three or more BP-lowering drugs of different classes at maximally tolerated doses, of which one is a diuretic (*Table 11*).[915] Resistant hypertension should be managed at specialized centres with the expertise and resources to exclude pseudo-resistant hypertension (adherence testing) and causes of secondary hypertension.[916]

9.13.2. Non-pharmacological interventions

The Treating Resistant Hypertension Using Lifestyle Modification to Promote Health (TRIUMPH) trial demonstrated significant clinic and ambulatory BP reductions in patients with resistant hypertension participating in a 4-month lifestyle intervention comprising diet and exercise interventions delivered within a cardiac rehabilitation programme.[917]

9.13.3. Pharmacological interventions

BP-lowering treatment of resistant hypertension with single-pill combinations is recommended to reduce the pill burden, thereby increasing drug adherence and persistence.[492]

As resistant hypertension often, and especially in CKD,[918] represents a state of salt retention and volume expansion secondary to relative aldosterone excess,[516,919,920] BP control may be improved by switching hydrochlorothiazide to long-acting thiazide-like diuretics, such as chlorthalidone.[921,922] However, a recent trial of chlorthalidone vs. hydrochlorothiazide—which probably included a sizeable proportion of adults with resistant hypertension—did not demonstrate any difference in systolic BP or CVD outcomes between the two medications. In the subgroup of patients with prior CVD, there was a strong trend of benefit with chlorthalidone on CVD outcomes.[447] Of note, the risk of hypokalaemia was higher in the chlorthalidone group than in the hydrochlorothiazide group.[447] In patients with eGFR < 30 mL/min/1.73 m^2, an adequately uptitrated loop diuretic is necessary to define resistant hypertension.

Most patients with resistant hypertension require the addition of non-first-line BP-lowering drugs (*Figure 22*). Of these, low-dose spironolactone (25–50 mg daily) should be considered first.[459,515,923–925] In patients with resistant hypertension and type 2 diabetes, spironolactone (25–50 mg daily) reduced BP and albuminuria.[926] The use of spironolactone can be precluded by limited tolerability due to anti-androgenic side effects resulting in breast tenderness or gynaecomastia (in about 6%), impotence in men, and menstrual irregularities in women.[927] The efficacy and safety of spironolactone for treating resistant hypertension have not yet been established in patients with significant renal impairment. Moreover, spironolactone, especially in addition to RAS inhibitors, increases the risk of hyperkalaemia.[927,928] Therefore, spironolactone should be restricted to patients with an eGFR of ≥30 mL/min/1.73 m^2 and a plasma potassium concentration of ≤4.5 mmol/L.[459] Steroidal MRAs are contraindicated in patients with an eGFR of <30 mL/min/1.73 m^2. Serum electrolytes and kidney function should be monitored soon after initiation and frequently thereafter. In patients with resistant hypertension and CKD (eGFR of 25–45 mL/min/1.73 m^2), the oral potassium binder patiromer enabled more patients to continue treatment with spironolactone.[929]

If spironolactone is not tolerated due to anti-androgen side effects, eplerenone may be used. If eplerenone is used, higher doses (i.e. 50–200 mg daily) and twice-daily dosing may be necessary to achieve a BP-lowering effect.[503] Of note, eplerenone is not licensed for hypertension treatment in many countries.

When not already prescribed for a compelling indication, beta-blockers should be considered in the treatment of resistant hypertension, though their BP-lowering effects appear to be less potent than spironolactone in the setting of resistant hypertension.[459]

Amiloride and clonidine have data suggesting they are as effective as spironolactone for BP lowering, though they lack outcomes data. A non-exhaustive list of additional medications sometimes used for BP-lowering purposes includes other centrally acting BP-lowering medications (e.g. methyldopa), hydralazine, aliskiren, minoxidil, triamterene, and loop diuretics (*Figure 22*).[515,516] As noted earlier, minoxidil use is often limited by side effects.

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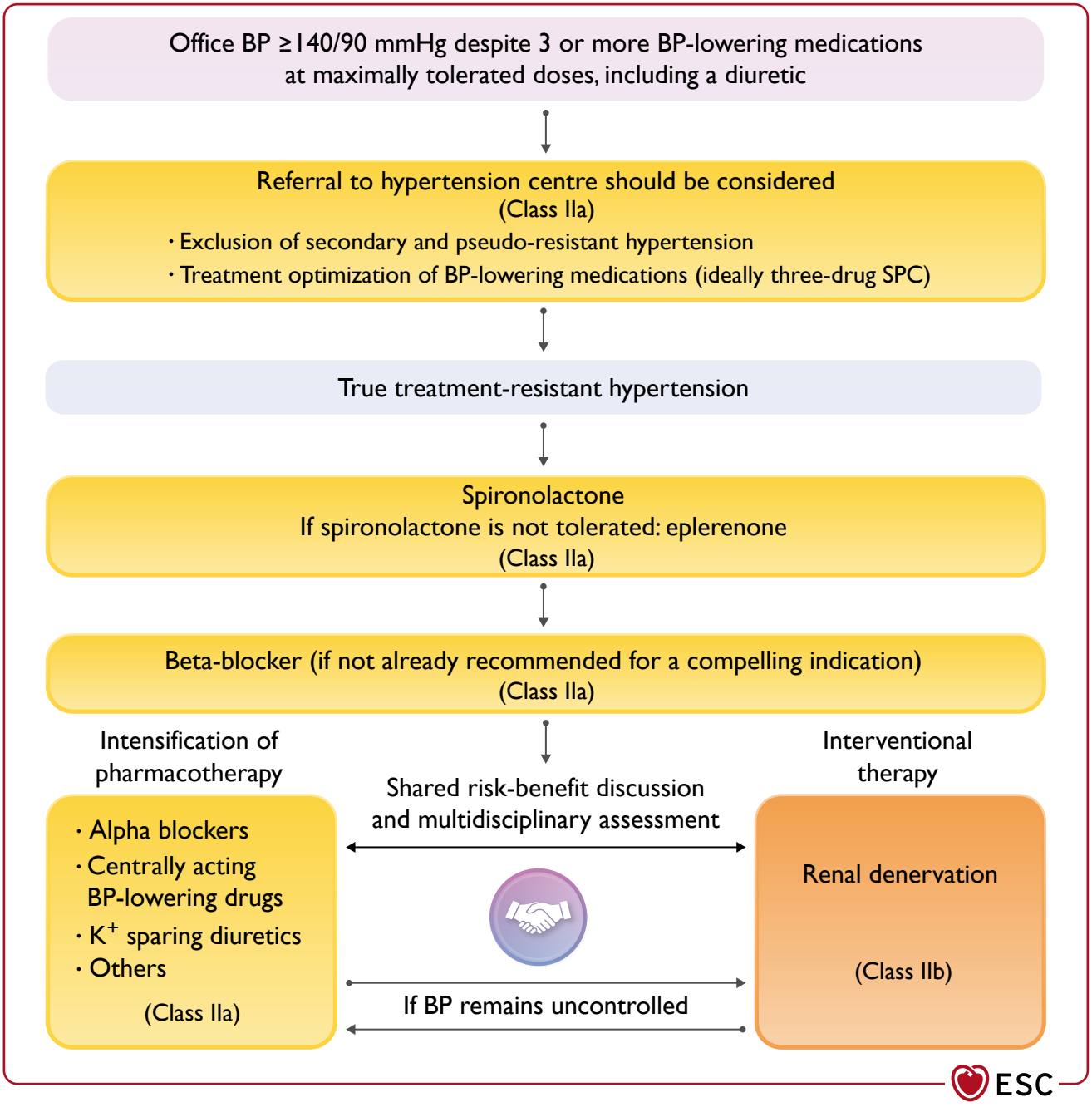

Figure 22 Management of resistant hypertension. BP, blood pressure; K^+, potassium; SPC, single-pill combination.

9.13.4. Devices for blood pressure lowering

Several devices have been investigated for treating resistant hypertension. Of these, the most evidence is available for catheter-based renal denervation. As discussed in *Section 8.6.1*, several randomized, sham-controlled trials have been published, demonstrating a BP-lowering efficacy over 24 h for radiofrequency and ultrasound renal denervation in a broad spectrum of hypertension, including resistant hypertension.[568,585] Other devices are still under investigation and are not recommended for routine use in clinical practice (*Section 8.6.2*).

Recommendation Table 30 — Recommendations for treating resistant hypertension (see Evidence Tables 42 and 43)

Recommendations	Class[a]	Level[b]
In patients with resistant hypertension and uncontrolled BP despite use of first-line BP lowering therapies, the addition of spironolactone to existing treatment should be considered.[459,515]	IIa	B

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In patients with resistant hypertension in whom spironolactone is not effective or tolerated, treatment with eplerenone instead of spironolactone,[503] or the addition of a beta-blocker if not already indicated[459] and, next, a centrally acting BP-lowering medication,[515] an alpha-blocker,[515] hydralazine, or a potassium-sparing diuretic[516] should be considered.	IIa	B
To reduce BP, and if performed at a medium-to-high volume centre, catheter-based renal denervation may be considered for resistant hypertension patients who have BP that is uncontrolled despite a three BP-lowering drug combination, and who express a preference to undergo renal denervation after a shared risk-benefit discussion and multidisciplinary assessment.[564,566–568,586–590]	IIb	B

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BP, blood pressure.
[a]Class of recommendation.
[b]Level of evidence.

9.14. Management of specific causes of secondary hypertension

9.14.1. General considerations

These guidelines will describe only the general principles of managing the most common forms of secondary hypertension. For the rarer forms of secondary hypertension, patients should be referred to specialized hypertension centres.

By definition, secondary hypertension should be, for the most part, cured when the underlying cause has been unambiguously identified and removed. However, in clinical practice, this is not always the case. Vascular remodelling, a common feature of a delayed diagnosis of secondary hypertension, affects renal function and can account for residual high BP in some patients with secondary hypertension. The rate of cure is higher when the diagnosis is made early in the course of the disease. Most common forms of secondary hypertension are listed in *Table 13*.

9.14.2. Primary aldosteronism

Primary aldosteronism (Conn syndrome) is the most common form of secondary hypertension. The management of primary aldosteronism depends on its subtype, particularly on adrenal lesions being unilateral or bilateral, because the unilateral forms are amenable to surgical treatment while the latter require lifelong medical treatment. In sporadic forms, unilateral primary aldosteronism is distinguished from bilateral primary aldosteronism by adrenal vein sampling or functional imaging with radiolabelled tracers.[930–932] In the much less common familial forms (necessitating a family history be taken), genetic testing for germline mutations is necessary.[933]

For unilateral primary aldosteronism, surgical removal of the offending adrenal gland is typically considered, unless the patient is older or has comorbidities of concern. Surgery is not an option for bilateral primary aldosteronism. Medical treatment is currently based on MRAs. Among MRAs, spironolactone is the most widely available. The effective dose, usually 50–100 mg once daily, can be titrated up to 300–400 mg once daily, if necessary. Eplerenone is also used and, despite being less potent than spironolactone and requiring twice-daily administration, it has the advantage of causing less gynaecomastia and erectile dysfunction in men.[934] Newer agents, such as the non-steroidal MRAs finerenone and exarenone, and the aldosterone synthase inhibitor baxdrostat, which lower BP in resistant hypertension,[326,474] are also being tested for treating primary aldosteronism. Of the familial forms, only glucocorticoid-remediable primary aldosteronism, now reclassified as familial hyperaldosteronism type 1, can be corrected with dexamethasone,[935] usually with low doses that are free of glucocorticoid effects and can be safely used during pregnancy.[936] For detailed information, readers are referred to the latest primary aldosteronism guidelines.[328,329]

9.14.3. Renovascular hypertension

Patients with RVH should receive medical therapy to reduce BP in the first instance. Percutaneous transluminal renal angioplasty (PTRA) without stenting is the treatment of choice for fibromuscular dysplasia, and can restore renal perfusion pressure and lower BP.[937] When this is not feasible, RAS blockers are the drugs of choice for treatment, but they require careful monitoring of renal function over time, as they can cause acute renal failure in those with tight bilateral stenoses or a stenosed solitary functioning kidney. Possible involvement of the carotid, coronary, and other major arteries, possibly leading to dissection if BP is not controlled, should also be considered, as fibromuscular dysplasia is now recognized as a systemic disease affecting multiple vascular beds.

Patients with significant atherosclerotic renal artery stenosis are at very high risk of CVD and renal events. It is recommended that PTRA and stenting are performed in experienced centres due to the high risk of restenosis. Unfortunately, though these studies did not solely recruit patients with true significant atherosclerotic RVH, publication of some null trials[938,939] have decreased the enthusiasm for investigating atherosclerotic renal artery stenosis. This could result in more uncontrolled hypertension, recurrent flash pulmonary oedema (Pickering syndrome), and worsening renal function ultimately leading to end-stage renal disease.[940]

Recommendation Table 31 — Recommendations for managing hypertension in patients with renovascular hypertension (see Evidence Tables 44 and 45)

Recommendations	Class[a]	Level[b]
Renal artery angioplasty without stenting should be considered for patients with hypertension and haemodynamically significant renal artery stenosis due to fibromuscular dysplasia.[941]	IIa	C
Renal artery angioplasty and stenting may be considered in patients with haemodynamically significant, atherosclerotic, renal artery stenosis (stenosis of 70%–99%, or 50%–69% with post-stenotic dilatation and/or significant trans-stenotic pressure gradient) with: • Recurrent heart failure, unstable angina, or sudden onset flash pulmonary oedema despite maximally tolerated medical therapy; • Resistant hypertension; • Hypertension with unexplained unilaterally small kidney or CKD; • Bilateral renal artery stenosis or unilateral renal artery stenosis in a solitary viable kidney.[942,943]	IIb	C

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In patients with an indication to renal artery revascularization and technically unfeasible, or failed, renal artery angioplasty and stenting, open surgical revascularization may be considered.	IIb	C
Renal artery angioplasty is not recommended in patients without confirmed haemodynamically significant renal artery stenosis.[c] [938,939]	III	A

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CKD, chronic kidney disease.
[a]Class of recommendation.
[b]Level of evidence.
[c]A haemodynamically relevant stenosis is usually defined by a luminal narrowing of >70% or 50%–70% with post-stenotic dilatation.

9.14.4. Phaeochromocytoma/paraganglioma

Phaeochromocytomas are rare adrenal tumours that secrete catecholamines and are present in <0.2% of patients with hypertension. A small percentage (<10%) of catecholamine-producing tumours are extra-adrenal and are derived from sympathetic and non-sympathetic nerves. More than 35% of the non-syndromic PPGLs are due to germline mutations.[338] These mutations should be screened for because, when found, they can drive management of the proband and the family and also inform the choice of functional imaging. Moreover, some germline mutations, such as those involving succinate dehydrogenase B, carry a risk of malignant adrenal tumours.[301,336]

Sympathetic PPGLs are usually secreting and present with chronic, episodic, or labile hypertension. Adrenergic crises cause hypertensive emergencies and should be treated with an intravenous (i.v.) alpha-1-blocker, such as phentolamine, doxazosin or terazosin, or labetalol. When given i.v. (1–2 mg/kg) twice weekly as a bolus over 1 min followed by a continuous infusion, labetalol also has alpha-blocker properties and has the advantage of allowing titration of the infusion based on the BP response, and avoids tachycardia via beta-blockade.

Identifying a single tumour mandates surgical excision after adequate pharmacological preparation, because secreting PPGLs can cause fatal events with no warning. Administering doxazosin or terazosin, followed by a beta-blocker, usually controls BP and adrenergic crises. As PPGLs are associated with a redistribution of volume from the periphery to the cardiopulmonary system,[944] patients with PPGLs have peripheral hypovolaemia that exposes them to the risk of profound hypotension, particularly right after tumour excision. Therefore, adequate fluid administration should be carefully managed.

9.14.5. Obstructive sleep apnoea syndrome

The management of this prevalent condition should be driven by the result of a polysomnography study, which should provide the value of the AHI (the average number of episodes per hour) and the sleep position in which apnoeic–hypopnoeic episodes occur. For mild OSAS (AHI < 15), weight loss and advice on sleep hygiene are usually sufficient. For moderate (AHI of 15–30) and severe (AHI > 30) OSAS, continuous positive airway pressure (CPAP) is indicated and usually improves BP control and helps to resolve resistant hypertension. If CPAP is not tolerated, the site of upper airway obstruction should be determined by an Ear, Nose, and Throat evaluation with drug-induced sleep endoscopy as a potential step to corrective surgery.

9.14.6. Drug-induced hypertension

Over-the-counter medications, prescribed drugs, and drug abuse (recreational substances and misuse of drugs) can cause hypertension (Supplementary data online, *Table S4*).

9.14.6.1. Anticancer drug-induced hypertension

Growing evidence indicates that, while contemporary anticancer and anti-angiogenic drugs improve cancer survival, they can also cause hypertension (Supplementary data online, *Table S4*). This is especially evident in patients treated with vascular endothelial growth factor inhibitors, in whom BP increases in 80%–90%.[945] Tyrosine kinase inhibitors and proteasome inhibitors also increase BP, as do adjuvant therapies (corticosteroids, calcineurin inhibitors, non-steroidal anti-inflammatory drugs, and anti-androgen hormone therapy). Hypertension caused by anticancer drugs is often dose limiting and may be reversible after therapy interruption or discontinuation. Evidence-based clinical trials specifically addressing patients who develop hypertension due to cancer therapy are lacking. It is recommended that management of hypertension in these patients follows that for the general population.[945,946] Managing these complex patients requires multidisciplinary healthcare involving oncologists, hypertension specialists, cardiologists, and nephrologists,[945,946] as highlighted in the 2022 ESC Guidelines on cardio-oncology.[946]

9.14.7. Other forms of secondary hypertension

Other forms of secondary hypertension, such as genetic causes of hypertension (Liddle's syndrome, glucocorticoid-remediable aldosteronism), excess liquorice, Cushing's syndrome, thyroid disease, hyperparathyroidism, aortic coarctation, and acromegaly are rare. Affected patients should be referred to specialized centres for treatment.

10. Acute and short-term lowering of blood pressure

10.1. Acute blood pressure management in hypertensive emergencies

10.1.1. Definition and characteristics of hypertensive emergencies

Hypertensive emergency is defined as BP of ≥180/110 mmHg (see *Figure 10*) associated with acute HMOD, often in the presence of symptoms. Hypertensive emergencies are potentially life-threatening and require immediate and careful intervention to reduce BP, often with i.v. therapy.

Symptoms of hypertensive emergency depend on the organs affected but may include headache, visual disturbances, chest pain, shortness of breath, dizziness, and other neurological deficits. In patients with hypertensive encephalopathy, somnolence, lethargy, tonic–clonic seizures, and cortical blindness may precede a loss of consciousness; however, focal neurological lesions are rare and should raise the suspicion of stroke.

As outlined in *Section 7*, we define HMOD among patients with chronically elevated BP or hypertension as the presence of specific cardiac, vascular, and renal alterations.[31,159] However, in the setting of hypertensive emergency, more acute manifestations of organ damage are relevant for management.

Acute manifestations of organ damage include:

- Patients with severe acute hypertension associated with other clinical conditions likely to require urgent reduction in BP, e.g. acute onset of aortic dissection, myocardial ischaemia, eclampsia, or heart failure.

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- Malignant hypertension, defined as extreme BP elevations and acute microvascular damage (microangiopathy) affecting various organs.[947] The hallmark of this condition is small-artery fibrinoid necrosis in the kidneys, retina, and brain. The acute microangiopathy is typically characterized clinically by retinopathy (flame haemorrhages, cotton wool spots, and/or papilloedema). Other manifestations of microangiopathy include disseminated intravascular coagulation, encephalopathy (in about 15% of cases), acute heart failure, and acute deterioration in renal function.
- Patients with sudden severe hypertension due to phaeochromocytoma, which can result in severe acute organ damage.

The term 'hypertension urgency' describes severe hypertension in patients without clinical evidence of acute organ damage. While these patients require BP reduction, they do not usually require admission to hospital, and BP reduction is best achieved with oral medication according to the drug treatment algorithm presented in *Section 8*. However, these patients may require more urgent outpatient review to ensure that their BP is controlled.

Acute and severe increases in BP can sometimes be precipitated by sympathomimetics such as methamphetamine or cocaine, when caution around beta-blocker use is also needed. Many patients in an emergency department with acute pain or distress may have acutely elevated BP that will normalize when the pain and distress are relieved, rather than requiring any specific intervention to lower BP.

A diagnostic work-up is necessary for patients with a suspected hypertensive emergency (see Supplementary data online, *Table S12*).

10.1.2. Acute management of hypertensive emergencies

Key considerations in defining treatment are:

(1) Establishing the affected target organ(s) and whether they require any specific interventions other than BP lowering.
(2) Determining whether there is a precipitating cause for the acute rise in BP and/or another concomitant health condition present that might affect the treatment plan (e.g. pregnancy).
(3) The recommended timing and magnitude of BP lowering required for safe BP reduction.

These considerations will inform the type of BP-lowering treatment required. Regarding BP-lowering drugs, i.v. treatment using a short half-life drug is typically ideal to allow careful titration of the BP response to treatment. This requires a higher dependency clinical area with facilities for continuous or near-continuous haemodynamic monitoring. Recommended drug treatments for specific hypertensive emergencies are provided in the Supplementary data online, *Table S13*.

Rapid and uncontrolled or excessive BP lowering is not recommended in hypertensive emergency as this can lead to further complications. Although i.v. drug administration is recommended for most hypertensive emergencies, oral therapy with ACE inhibitors, ARBs, or beta-blockers (shorter-acting formulations like captopril or metoprolol) can also be effective. However, low initial doses should be used because these patients can be very sensitive to these agents, and treatment should take place in hospital. Further comprehensive details on the clinical management of hypertensive emergencies are available elsewhere.[242]

10.1.3. Prognosis and follow-up

The survival of patients with hypertensive emergencies has improved over the past few decades, but these patients remain at high risk and should be screened for secondary hypertension.

10.2. Acute blood pressure management in acute intracerebral haemorrhage

In acute intracerebral haemorrhage, an increased BP is common and is associated with a greater risk of haematoma expansion and death, and a worse prognosis for neurological recovery. In trials testing immediate BP lowering (within <6 h) to a systolic target of <140 mmHg, the achieved systolic BP in the intervention group was typically 140–160 mmHg and was reported to reduce the risk of haematoma expansion.[948,949] Excessive acute drops in systolic BP (>70 mmHg) may be associated with acute renal injury and early neurological deterioration and should be avoided.[950,951]

10.3. Acute blood pressure management in acute ischaemic stroke

The beneficial effects of BP reduction in acute ischaemic stroke remain unclear. In patients not receiving i.v. thrombolysis or mechanical thrombectomy, there is no evidence for actively lowering BP unless it is extremely high (e.g. >220/120 mmHg). If BP is extremely high, an initial moderate relative reduction of 10%–15% over a period of hours may be considered.[952] The reason for a more conservative approach to acute BP management is that cerebral autoregulation may be impaired in acute stroke, and maintaining cerebral perfusion relies on systemic BP.

In contrast, patients who are treated with i.v. thrombolysis or mechanical thrombectomy (or both) should have more proactive management of severe hypertension, because they have an increased risk of reperfusion injury and intracranial haemorrhage. In patients undergoing treatment with i.v. thrombolysis, BP should be lowered to <185/110 mmHg prior to thrombolysis and then maintained at <180/105 mmHg over the following 24 h.[953] In patients undergoing treatment with mechanical thrombectomy (with or without i.v. thrombolysis) there is limited evidence from clinical trials, but BP should also be lowered to <180/105 mmHg prior to thrombectomy and maintained over the next 24 h.[953,954] Therefore, patients with acute ischaemic stroke and a BP of <180/105 mmHg in the first 72 h after stroke do not seem to benefit from the introduction or reintroduction of BP-lowering medication.[955] For stable patients who remain hypertensive (≥140/90 mmHg) ≥3 days after an acute ischaemic stroke, initiation or reintroduction of BP-lowering medication is recommended.

Recommendation Table 32 — Recommendations for acutely managing blood pressure in patients with intracerebral haemorrhage or acute ischaemic stroke

Recommendations	Class[a]	Level[b]
For patients with ischaemic stroke or TIA and an indication for BP lowering, it is recommended that BP-lowering therapy be commenced before hospital discharge.[819,820,823]	I	B
In patients with acute ischaemic stroke, early BP lowering with BP-lowering therapy should be considered in the first 24 h in the following settings:		
• In patients who are eligible for re-perfusion therapy with intravenous thrombolysis or mechanical thrombectomy, BP should be carefully lowered and maintained at <180/105 mmHg for at least the first 24 h after treatment.[956–960]	IIa	B

Continued

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• In patients with ischaemic stroke not receiving re-perfusion treatment and BP of ≥220/110 mmHg, BP should be carefully lowered by approximately 15% during the first 24 h after stroke onset.[956–960]	IIa	C
In patients with intracerebral haemorrhage, immediate BP lowering (within 6 h of symptom onset) should be considered to a systolic target 140–160 mmHg to prevent haematoma expansion and improve functional outcome.[948,949]	IIa	A
In patients with intracerebral haemorrhage presenting with systolic BP ≥220 mmHg, acute reduction in systolic BP >70 mmHg from initial levels within 1 h of commencing treatment is not recommended.[950,951,960–963]	III	B

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BP, blood pressure; TIA, transient ischaemic attack.
[a]Class of recommendation.
[b]Level of evidence.

Recommendation Table 33 — Recommendations for acutely managing blood pressure in patients with severe hypertension in pregnancy and pre-eclampsia (see Evidence Table 46)

Recommendation	Class[a]	Level[b]
In pre-eclampsia or eclampsia with hypertensive crisis, drug treatment with i.v. labetalol or nicardipine and magnesium is recommended.[971]	I	C
In pre-eclampsia or eclampsia associated with pulmonary oedema, nitroglycerin given as an i.v. infusion is recommended.[242]	I	C
In severe hypertension in pregnancy: • drug treatment with i.v. labetalol, oral methyldopa, or oral nifedipine is recommended. Intravenous hydralazine is a second-line option.[666–668,969,971]	I	C

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i.v., intravenous.
[a]Class of recommendation.
[b]Level of evidence.

10.4. Acute blood pressure management in pre-eclampsia and severe hypertension in pregnancy

10.4.1. Pre-eclampsia

Pre-eclampsia is discussed in *Section 9*. Here we focus on its management in the acute setting. Pre-eclampsia is cured by delivery. Most international societies, including the ESC, recommend an intensive approach to BP lowering in pre-eclampsia.[89,964,965] In women with pre-eclampsia and severe hypertension, immediately reducing systolic BP to <160 mmHg and diastolic BP to <105 mmHg using i.v. labetalol or nicardipine (with administration of magnesium sulfate if appropriate and consideration of delivery if appropriate) was recommended in the 2018 ESC/ESH Guidelines on the management of arterial hypertension and the 2022 ESC Guidelines for management of cardiovascular disease in pregnancy.[1,89] The objective of treatment is to lower BP within 150–180 min.

Magnesium sulfate [4 g i.v. over 5 min, then 1 g/h i.v.; or 5 g intramuscularly (i.m.) into each buttock, then 5 g i.m. every 4 h] is recommended for eclampsia treatment but also for women with pre-eclampsia who have severe hypertension and proteinuria or hypertension and neurological symptoms or signs.[966] There is a risk of hypotension when magnesium is given concomitantly with nifedipine.[967] If BP control is not achieved by 360 min despite two medications, consulting critical care is recommended for intensive care unit admission, stabilization, and delivery (if appropriate).[966] Since plasma volume is reduced in pre-eclampsia, diuretic therapy should be avoided.

10.4.2. Severe acute hypertension in pregnancy

Severe hypertension in pregnancy (without pre-eclampsia) may necessitate acute BP-lowering therapies. Severe hypertension in pregnancy is defined in general as systolic BP of >160 mmHg and diastolic BP of >110 mmHg and is associated with adverse maternal and peri-natal outcomes independent of pre-eclampsia and potentially of the same magnitude as eclampsia itself.[89,968]

There are differences in rate of BP control between i.v. labetalol and i.v. hydralazine in severe hypertension in pregnancy.[969] While evidence is conflicting,[667,668] hydralazine may be associated with more peri-natal adverse events than other drugs.[970] Nifedipine seems to provide lower BP with lower rates of neonatal complications than labetalol.[971]

10.5. Peri-operative acute management of elevated blood pressure

Details are provided in the ESC Guidelines on cardiovascular assessment and management of patients undergoing non-cardiac surgery.[972] Peri-operative hypertension, hypotension, and BP variability are associated with haemodynamic instability and poor clinical outcomes for patients undergoing surgery.[973] Pre-operative risk assessment for BP management, therefore, should involve assessing for underlying end-organ damage and comorbidities.[974] Postponing necessary non-cardiac surgery is not usually warranted for patients with minor or moderate elevations in BP, as they are not at higher CVD risk.[130,975]

Avoiding large fluctuations in BP in the peri-operative course is important, and planning a strategy for a patient should account for the baseline office BP.[974–977]

There is insufficient evidence for reduced or increased peri-operative BP targets compared to usual care BP targets to lower peri-operative events.[978] No specific measure of BP appears better than any other for predicting risk of peri-operative events.[975]

10.5.1. Blood pressure-lowering drugs in the peri-operative phase

Routine initiation of a beta-blocker peri-operatively is not necessary.[979]

Pre-operative initiation of beta-blockers in advance of high-risk, non-cardiac surgery may be considered in patients who have known coronary artery disease or myocardial ischaemia[980] or two or more significantly elevated clinical risk factors in order to reduce the incidence of peri-operative myocardial infarction.[979] Peri-operative continuation of beta-blockers is recommended for patients currently taking beta-blockers.[981]

Some studies suggest that continued use of ACE inhibitors is associated with a higher risk of peri-operative hypotension and subsequent end-organ damage including kidney injury, myocardial infarction, and stroke.[982] In the Prospective Randomized Evaluation of Preoperative Angiotensin-Converting Enzyme Inhibition (PREOP-ACEI) trial, transient pre-operative interruption of ACE inhibitor therapy was associated with a decreased risk of intra-operative hypotension.[983] A subsequent systematic review also showed a decreased risk of intra-operative hypotension with withholding ACE inhibitors/ARBs before surgery, but no association

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with decreased mortality or CVD outcomes.[984] On the other hand, vigilance is needed because withholding ACE inhibitors has also been shown to increase post-operative hypertension.[985] In patients with heart failure, loop diuretics can be continued in patients prone to volume overload.[986] CCBs are generally considered safe pre-operatively.

11. Patient-centred care in hypertension

11.1. Definition

Patient-centred care is defined as an attitude of the healthcare professional that closely aligns with the patient's preferences and needs.[987] In the patient-centred approach (*Figure 23*), patients are viewed as active participants in health services, who work as partners alongside healthcare professionals. A patient-centred approach is associated with higher satisfaction rates, better adherence to recommendations and prescriptions, and better treatment, particularly in the management of chronic illness, such as hypertension.[988] While there is limited evidence for the efficacy and effectiveness of specific shared decision-making intervention strategies in hypertension care,[989] it is viewed as an ethical imperative in healthcare practice and health policy, and in clinical guidelines.[130]

11.2. Communicating consequences of treatment

In line with patient-centred care, it is important to assess whether patients understand their hypertension-related risk, the rationale for any hypertension treatment, the benefits and harms of hypertension treatment, and that the treatment plan is also centrally guided by what matters most to the patient. Risk communication is challenging, and providers need to be led by the individual's preferences when presenting more detailed numeric and visual representations of risk and the likely benefits and harms of hypertension treatment. Socio-demographic differences in healthcare need to be considered in patient–provider communication.[990,991]

Standard approaches to communicate consequences of treatment can involve 10-year risk of a CVD event with SCORE2 or SCORE2-OP. Alternatively, individual risk and risk reduction can be communicated in terms of 'risk age' or 'heart age' (*Section 7.3*).

Figure 23 Patient-centred care.

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Recommendation Table 34 — Recommendations for communicating consequences of treatment (see Evidence Table 47)

Recommendation	Class[a]	Level[b]
An informed discussion about CVD risk and treatment benefits tailored to the needs of a patient is recommended as part of hypertension management.[992]	I	C
Motivational interviewing should be considered for patients with hypertension at hospitals and community health centres to assist patients in controlling their BP and to enhance treatment adherence.[993,994]	IIa	B
Physician–patient web communications are an effective tool that should be considered in primary care, including reporting on home BP readings.[995]	IIa	C

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BP, blood pressure; CVD, cardiovascular disease.
[a]Class of recommendation.
[b]Level of evidence.

11.3. Self-measuring and monitoring

Self-care refers to individual responsibility for healthy lifestyle behaviours, as well as the actions required to cope with health conditions.[996,997] In the context of hypertension, it also includes self-management and self-measurement of BP.

Self-management includes lifestyle behaviour (diet, exercise, smoking, alcohol), co-management of medical treatments, and support for adhering to prescribed medication.[998] Self-monitoring allows high BP to be detected early,[999] and enables patients to co-manage medications with their healthcare provider.[1000,1001] Suitably validated and correctly used digital devices have the potential to support co-management,[1002,1003] and facilitate remote monitoring of BP.[76,81,1004]

Recommendation Table 35 — Recommendations for self-measuring and monitoring blood pressure (see Evidence Table 48)

Recommendations	Class[a]	Level[b]
Home BP measurement for managing hypertension by using self-monitored BP is recommended to achieve better BP control.	I	B
Self-measurement, when properly performed, is recommended due to positive effects on the acceptance of a diagnosis of hypertension, patient empowerment, and adherence to treatment.[1001]	I	C
Enhanced self-monitoring of BP using a device paired with a connected smartphone application may be considered, though evidence to date suggests that this may be no more effective than standard self-monitoring.[1005,1006]	IIb	B

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BP, blood pressure.
[a]Class of recommendation.
[b]Level of evidence.

11.4. Facilitating medication adherence and persistence

Adherence (*Figure 24*) to BP-lowering drug regimens in clinical practice is almost always lower than seen in clinical trials.[1007] Most apparent treatment-resistant hypertension is accounted for by non-adherence.[1008] Adherence should always be assessed with a no-blame approach. Various methods are available to assess adherence and, along with details on barriers to adherence, are described in the Supplementary data online and *Table S14*.[1009]

Adherence may also be facilitated by an optimal therapeutic regimen, which can be achieved by medication reviews carried out at appropriate intervals. Several factors should be considered: (i) identifying drug-related adverse events and appropriate dosing levels, (ii) using long-acting drugs that require once-daily dosing (preferably drugs that are long-acting due to pharmacokinetic properties rather than galenic formulation), (iii) avoiding complex dosing schedules, (iv) using single-pill combinations whenever feasible, (v) taking into account the financial capacity of the patient to pay for a given regimen in the longer term, if relevant, or other pertinent aspects of the local or national healthcare systems, and (vi) enlisting support of a family member or other social support to facilitate medication adherence and persistence (see Supplementary data online, *Table S15*).[1010]

While there have been advancements in digital tools to support self-management of chronic illness including hypertension, there is little efficacy evidence evaluating these interventions. Therefore, it is premature to make recommendations about specific digital tools.

11.5. Multidisciplinary management

A collaborative approach to managing hypertension, using team-based care among physicians, nurses, pharmacists, dietitians, and physiotherapists, offers significant benefits over physician-only care. Multidisciplinary care is intended to be collaborative and complementary to regular medical care[1011] and is associated with lower systolic and diastolic BP[227,229,1012,1013] and improved outcomes.[230,1014] Task-shifting away from physicians is necessary to meet the huge need for the management of elevated BP and hypertension in the population.[1015] Prescribing remains a physician duty, but prescribing can be conducted under collaborative practice agreements with the multidisciplinary team in many countries.

Further details on patient-centred care in hypertension is provided in the Supplementary data online.

Recommendation Table 36 — Recommendations for multi/interdisciplinary blood pressure management (see Evidence Table 49)

Recommendation	Class[a]	Level[b]
Multidisciplinary approaches in the management of patients with elevated BP and hypertension, including appropriate and safe task-shifting away from physicians, are recommended to improve BP control.[227,229,230,1012–1014,1016]	I	A

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BP, blood pressure.
[a]Class of recommendation.
[b]Level of evidence.

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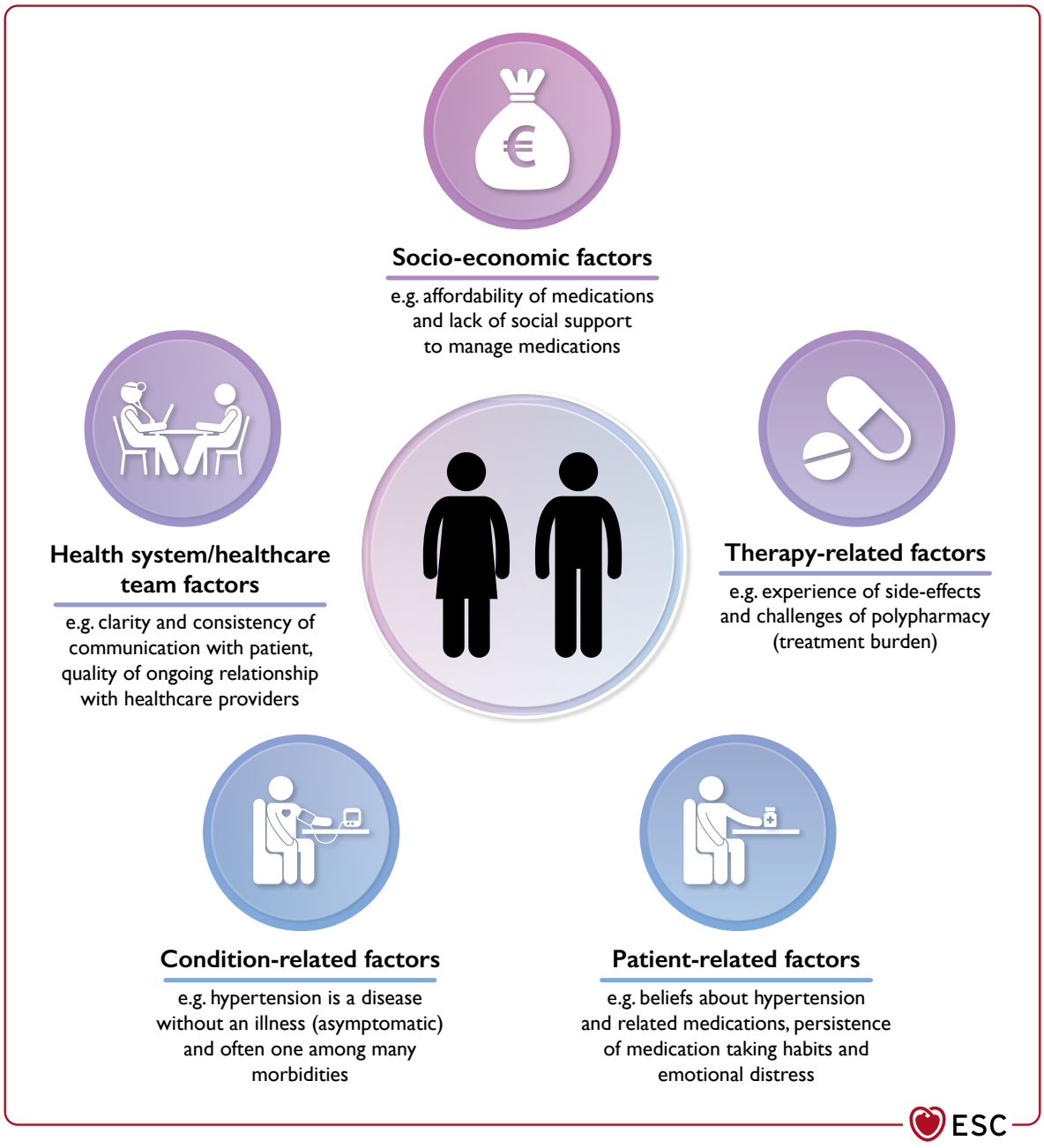

Figure 24 The five dimensions of adherence (WHO, 2003) applied to hypertension.

12. Key messages

(1) Given the demographic transition and the worldwide ageing of populations, the number of individuals with elevated BP or hypertension is increasing worldwide.
(2) The trajectory of BP control appears to be worsening in North America, in some (but not all) European countries, and elsewhere around the world.
(3) The risk for CVD attributable to BP is on a continuous log-linear exposure variable scale, not a binary scale of normotension vs. hypertension.
(4) BP-lowering drugs can reduce CVD risk even among individuals not traditionally classified as hypertensive. Accordingly, a new BP category called 'elevated BP' is introduced. Elevated BP is defined as an office systolic BP of 120–139 mmHg or diastolic BP of 70–89 mmHg. Hypertension remains defined as office BP of ≥140/90 mmHg.
(5) Hypertension in women is under-studied in basic, clinical, and population research.
(6) HMOD suggests long-standing or severe hypertension and is associated with increased CVD risk.
(7) Absolute CVD risk must be considered when assessing and managing elevated BP.
(8) Despite the growing number of hypertension guidelines, the rates of diagnosis, treatment, and control of hypertension (and elevated BP) remain suboptimal. A major factor underlying this is poor

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implementation of evidence-based guidelines in real-world clinical practice.

(9) One of the most important changes in the 2024 Guidelines is the focus on evidence related to CVD outcomes of BP-lowering interventions rather than BP lowering alone.

(10) Irrespective of the threshold BP above which BP-lowering treatment (lifestyle or pharmacological or other treatment) is recommended, the on-treatment BP target is 120–129/70–79 mmHg for all adults, provided this treatment is well tolerated. There are several important exceptions to these targets and individualized decision-making is always the most important priority.

13. Gaps in the evidence

(1) Drivers of worsening trajectories of BP control in women and men.

(2) Need for sex-specific data on epidemiology, risk factors, and pathophysiology of hypertension. Need for more prospective studies to assess women's and men's specific CVD risk factors pertinent to adults with elevated BP and hypertension, due to biological and socio-cultural conditions. This includes sex-specific weighting of traditional risk factors, as well as inclusion of sex-dependent, non-traditional, vascular risk factors such as stress, socio-economic conditions, and others.[1017,1018] We are also lacking data on sex-specific hormonal and genetic mechanisms and pathophysiology in the human.[1019] Another important area in need of investigation is a better understanding of the role of gender in the management of elevated BP and hypertension (including gender-driven barriers in accessing medical care and adherence).

(3) More widespread validation of home BP measuring devices. Validation protocols for cuffless BP measurement devices have just recently been proposed and need to be tested.

(4) Clinical effectiveness of HMOD in directing intensity of care and personalized approaches in managing elevated BP and hypertension.

(5) Best practice to screen and manage primary aldosteronism.

(6) Clinical benefits of treating low CVD-risk individuals with elevated BP and further data strengthening the use of BP-lowering medication among high-risk persons with baseline systolic BP of 120–129 mmHg.

(7) Need for more data on the sex-specific optimal dosing, effects, and adverse effects of BP-lowering drugs,[1020] in particular from specifically planned prospective randomized trials.

(8) More consideration for overall CVD outcomes of BP-lowering interventions.

(9) More European data (RCTs, real life) about the beneficial effect of treating patients with elevated BP and hypertension with polypills (inclusive of non-BP lowering medications).

(10) CVD outcomes-based data on MRAs as add-on therapy solely for resistant hypertension.

(11) Trials on the BP-lowering effects of newer antidiabetic drugs (such as SGLT2 inhibitors and GLP-1 receptor agonists) or drugs that now have indications for other conditions, such as finerenone or sacubitril-valsartan.

(12) Beneficial BP and CVD effects of increasing dietary potassium intake and other lifestyle interventions. Studies to disentangle the effect of sodium reduction vs. the effect of potassium supplementation on BP control and CVD outcomes.

(13) RCTs comparing single-pill combination therapy with fixed doses vs. multiple monotherapies and their effects on CVD outcomes.

(14) Cardiovascular outcomes trials of renal denervation.

(15) BP-lowering treatment RCTs on different ethnic and migrant groups established in Europe.

(16) Pharmacological BP management in young adults (aged <40 years) and better data on the efficacy of a life-course approach for the drug management of BP.[1021]

(17) CVD outcomes in moderately to severely frail and/or very elderly persons where BP medications have been deprescribed, and the impact of competing risks.

(18) Management of renal artery disease with haemodynamically stable but severe stenosis (i.e. without high-risk features).

(19) Need for clinical trials on managing hypertension in patients treated with anticancer drugs or anti-rejection drugs in recipients of an allograft transplant.

(20) Hypertension management in the setting of climate changes, global warming, air and other forms of pollution, pandemics, war zones, and in the context of drug restrictions experienced in some low-to-middle-income countries.

(21) Need to improve implementation of guidelines by healthcare providers.

(22) How to develop sustainable hypertension care at the intersection of growing numbers of patients and limited resources.

(23) Treat-to-target trials specifically testing BP-lowering drugs among drug-naïve persons with baseline BP of 120–129 mmHg and increased CVD risk.

14. 'What to do' and 'what not to do' messages from the guidelines

A selected sample of the main messages from these guidelines are provided in *Table 15*.

Table 15 What to do and what not to do

Recommendations	Class	Level
5. Measuring blood pressure		
It is recommended to measure BP using a validated and calibrated device, to enforce the correct measurement technique, and to apply a consistent approach to BP measurement for each patient.	I	B
All adult patients (≥18 years or older) are recommended to have their office and/or out-of-office BP measured on an opportunistic basis and recorded in their medical file, and be told what their current BP is.	I	C

Continued

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Out-of-office BP measurement is recommended for diagnostic purposes, particularly because it can detect both white-coat hypertension and masked hypertension. Where out-of-office measurements are not logistically and/or economically feasible, then it is recommended that the diagnosis be confirmed with a repeat office BP measurement using the correct standardized measurement technique.	I	B
It is recommended that office BP should be measured in both arms at least at the first visit, because a between-arm systolic BP difference of >10 mmHg is associated with an increased CVD risk and may indicate arterial stenosis.	I	B
If a between-arm difference of >10 mmHg in systolic BP is recorded, then it is recommended that all subsequent BP readings use the arm with the higher BP reading.	I	B
Out-of-office BP measurement is recommended for ongoing management to quantify the effects of treatment and guide BP-lowering medication titration, and/or identify possible causes of side effects (e.g. symptomatic hypotension). Where out-of-office measurements are not logistically and/or economically feasible then ongoing management is recommended to be based on repeated office BP measurements using the correct standardized measurement technique.	I	B
It is recommended that all patients undergoing BP measurement also undergo pulse palpation at rest to determine heart rate and arrhythmias such as AF.	I	C
6. Definition and classification of elevated blood pressure and hypertension		
It is recommended that BP be categorized as non-elevated BP, elevated BP, and hypertension to aid treatment decisions.	I	B
It is recommended to use a risk-based approach in the treatment of elevated BP, and individuals with moderate or severe CKD, established CVD, HMOD, diabetes mellitus, or familial hypercholesterolaemia are considered at increased risk for CVD events.	I	B
SCORE2 is recommended for assessing 10-year risk of fatal and non-fatal CVD among individuals aged 40–69 years with elevated BP who are not already considered at increased risk due to moderate or severe CKD, established CVD, HMOD, diabetes mellitus, or familial hypercholesterolaemia.	I	B
SCORE2-OP is recommended for assessing the 10-year risk of fatal and non-fatal CVD among individuals aged ≥70 years with elevated BP who are not already considered at increased risk due to moderate or severe CKD, established CVD, HMOD, diabetes mellitus, or familial hypercholesterolaemia.	I	B
It is recommended that, irrespective of age, individuals with elevated BP and a SCORE2 or SCORE2-OP CVD risk of ≥10% be considered at increased risk for CVD for the purposes of risk-based management of their elevated BP.	I	B
7. Diagnosing hypertension and investigating underlying causes		
In individuals with increased CVD risk where their screening office BP is 120–139/70–89 mmHg, it is recommended to measure BP out-of-office, using ABPM and/or HBPM or, if not logistically feasible, make repeated office BP measurements on more than one visit.	I	B
Where screening office BP is 140–159/90–99 mmHg, it is recommended that the diagnosis of hypertension should be based on out-of-office BP measurement with ABPM and/or HBPM. If these measurements are not logistically or economically feasible, then diagnosis can be made on repeated office BP measurements on more than one visit.	I	B
Where screening BP is ≥160/100 mmHg: • It is recommended that BP 160–179/100–109 mmHg is confirmed as soon as possible (e.g. within 1 month) preferably by either home or ambulatory BP measurements. • It is recommended when BP is ≥180/110 mmHg that hypertensive emergency be excluded.	I	C
It is recommended to measure serum creatinine, eGFR, and urine ACR in all patients with hypertension.	I	A
If moderate-to-severe CKD is diagnosed, it is recommended to repeat measurements of serum creatinine, eGFR, and urine ACR at least annually.	I	C
A 12-lead ECG is recommended for all patients with hypertension.	I	B
Echocardiography is recommended in patients with hypertension and ECG abnormalities, or signs or symptoms of cardiac disease.	I	B
Fundoscopy is recommended if BP >180/110 mmHg in the work-up of hypertensive emergency and malignant hypertension, as well as in hypertensive patients with diabetes.	I	C
Routine genetic testing for patients with hypertension is not recommended.	III	C
It is recommended that patients with hypertension presenting with suggestive signs, symptoms, or medical history of secondary hypertension are appropriately screened for secondary hypertension.	I	B
8. Preventing and treating elevated blood pressure		
Restriction of sodium to approximately 2 g per day is recommended where possible in all adults with elevated BP and hypertension [this is equivalent to about 5 g of salt (sodium chloride) per day or about a teaspoon or less].	I	A
Moderate-intensity aerobic exercise of ≥150 min/week [moderate aerobic exercise (≥30 min, 5–7 days/week) or alternatively 75 min of vigorous exercise per week over 3 days] is recommended and should be complemented with low- or moderate-intensity dynamic or isometric resistance training (2–3 times/week) to reduce BP and CVD risk.	I	A
It is recommended to aim for a stable and healthy BMI (20–25 kg/m^2) and waist circumference values (<94 cm in men and <80 cm in women) to reduce BP and CVD risk.	I	A

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Adopting a healthy and balanced diet such as the Mediterranean or DASH diets is recommended to help reduce BP and CVD risk.	I	A
Men and women are recommended to drink less alcohol than the upper limit, which is about 100 g/week of pure alcohol. How this translates into number of drinks depends on portion size (the standards of which differ per country), but most drinks contain 8–14 g of alcohol per drink. Preferably, it is recommended to avoid alcohol consumption to achieve best health outcomes.	I	B
It is recommended to restrict free sugar consumption, in particular sugar-sweetened beverages, to a maximum of 10% of energy intake. It is also recommended to discourage consumption of sugar-sweetened beverages, such as soft drinks and fruit juices, starting at young age.	I	B
It is recommended to stop tobacco smoking, initiate supportive care, and refer to smoking cessation programmes, as tobacco use strongly and independently causes CVD, CVD events, and all-cause mortality.	I	A
Among all BP-lowering drugs, ACE inhibitors, ARBs, dihydropyridine CCBs, and diuretics (thiazides and thiazide-like drugs such as chlorthalidone and indapamide) have demonstrated the most effective reduction of BP and CVD events, and are therefore recommended as first-line treatments to lower BP.	I	A
It is recommended that beta-blockers are combined with any of the other major BP-lowering drug classes when there are other compelling indications for their use, e.g. angina, post-myocardial infarction, HFrEF, or for heart rate control.	I	A
It is recommended to take medications at the most convenient time of day for the patient to establish a habitual pattern of medication taking to improve adherence.	I	B
Given trial evidence for more effective BP control vs. monotherapy, combination BP-lowering treatment is recommended for most patients with confirmed hypertension (BP ≥140/90 mmHg) as initial therapy. Preferred combinations are a RAS blocker (either an ACE inhibitor or an ARB) with a dihydropyridine CCB or diuretic. Exceptions to consider include patients aged ≥85 years, symptomatic orthostatic hypotension, moderate-to-severe frailty, and those with elevated BP (systolic BP 120–139 mmHg or diastolic BP 70–89 mmHg) with a concomitant indication for treatment.	I	B
In patients receiving combination BP-lowering treatment, fixed-dose single-pill combination treatment is recommended.	I	B
If BP is not controlled with a two-drug combination, increasing to a three-drug combination is recommended, usually a RAS blocker with a dihydropyridine CCB and a thiazide/thiazide-like diuretic, and preferably in a single-pill combination.	I	B
Combining two RAS blockers (ACE inhibitor and an ARB) is not recommended.	III	A
In adults with elevated BP and low/medium CVD risk (<10% over 10 years), BP lowering with lifestyle measures is recommended and can reduce the risk of CVD.	I	B
In adults with elevated BP and sufficiently high CVD risk, after 3 months of lifestyle intervention, BP lowering with pharmacological treatment is recommended for those with confirmed BP ≥130/80 mmHg to reduce CVD risk.	I	A
It is recommended that in hypertensive patients with confirmed BP ≥140/90 mmHg, irrespective of CVD risk, lifestyle measures and pharmacological BP-lowering treatment is initiated promptly to reduce CVD risk.	I	A
It is recommended to maintain BP-lowering drug treatment lifelong, even beyond the age of 85 years, if well tolerated.	I	A
8. Preventing and treating elevated blood pressure (blood pressure targets)		
To reduce CVD risk, it is recommended that treated systolic BP values in most adults be targeted to 120–129 mmHg, provided the treatment is well tolerated.	I	A
In cases where BP-lowering treatment is poorly tolerated and achieving a target systolic of 120–129 mmHg is not possible, it is recommended to target a systolic BP level that is 'as low as reasonably achievable' (ALARA principle).	I	A
8. Preventing and treating elevated blood pressure (renal denervation)		
Due to a lack of adequately powered outcomes trials demonstrating its safety and CVD benefits, renal denervation is not recommended as a first-line BP-lowering intervention for hypertension.	III	C
Renal denervation is not recommended for treating hypertension in patients with moderate-to-severely impaired renal function (eGFR <40 mL/min/1.73 m^2) or secondary causes of hypertension, until further evidence becomes available.	III	C
9. Managing specific patient groups or circumstances		
Young adults		
Comprehensive screening for the main causes of secondary hypertension is recommended in adults diagnosed with hypertension before the age of 40 years, except for obese young adults where it is recommended to start with an obstructive sleep apnoea evaluation.	I	B
Hypertension in pregnancy		
In women with gestational hypertension, starting drug treatment is recommended for those with confirmed office systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg.	I	B
In pregnant women with chronic hypertension, starting drug treatment is recommended for those with confirmed office systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg.	I	B
In women with chronic and gestational hypertension, it is recommended to lower BP below 140/90 mmHg but not below 80 mmHg for diastolic BP.	I	C

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Dihydropyridine CCBs (preferably extended-release nifedipine), labetalol, and methyldopa are recommended first-line BP-lowering medications for treating hypertension in pregnancy.	I	C
In consultation with an obstetrician, low- to moderate-intensity exercise is recommended in all pregnant women without contraindications to reduce the risk of gestational hypertension and pre-eclampsia.	I	B
RAS blockers are not recommended during pregnancy.	III	B
Very old and frail patients; orthostatic hypotension		
It is recommended that treatment of elevated BP and hypertension among older patients aged <85 years who are not moderately to severely frail follows the same guidelines as for younger people, provided BP-lowering treatment is well tolerated.	I	A
It is recommended to maintain BP-lowering drug treatment lifelong, even beyond the age of 85 years, if well tolerated.	I	A
Before starting or intensifying BP-lowering medication, it is recommended to test for orthostatic hypotension, by first having the patient sit or lie for 5 min and then measuring BP 1 and/or 3 min after standing.	I	B
It is recommended to pursue non-pharmacological approaches as the first-line treatment of orthostatic hypotension among persons with supine hypertension. For such patients, it is also recommended to switch BP-lowering medications that worsen orthostatic hypotension to an alternative BP-lowering therapy and not to simply de-intensify therapy.	I	A
Diabetes		
In most adults with elevated BP and diabetes, after a maximum of 3 months of lifestyle intervention, BP lowering with pharmacological treatment is recommended for those with confirmed BP ≥130/80 mmHg to reduce CVD risk.	I	A
BP-lowering drug treatment is recommended for people with pre-diabetes or obesity when confirmed office BP is ≥140/90 mmHg or when office BP is 130–139/80–89 mmHg and the patient is at predicted 10-year risk of CVD ≥10% or with high-risk conditions, despite a maximum of 3 months of lifestyle therapy.	I	A
In persons with diabetes who are receiving BP-lowering drugs, it is recommended to target systolic BP to 120–129 mmHg, if tolerated.	I	A
Chronic kidney disease		
In patients with diabetic or non-diabetic moderate-to-severe CKD and confirmed BP ≥130/80 mmHg, lifestyle optimization and BP-lowering medication are recommended to reduce CVD risk, provided such treatment is well tolerated.	I	A
In adults with moderate-to-severe CKD who are receiving BP-lowering drugs and who have eGFR >30 mL/min/1.73 m^2, it is recommended to target systolic BP to 120–129 mmHg, if tolerated. Individualized BP targets are recommended for those with lower eGFR or renal transplantation.	I	A
In hypertensive patients with CKD and eGFR >20 mL/min/1.73 m^2, SGLT2 inhibitors are recommended to improve outcomes in the context of their modest BP-lowering properties.	I	A
Cardiac disease		
In patients with a history of myocardial infarction who require BP-lowering treatment, beta-blockers and RAS blockers are recommended as part of that treatment.	I	A
In patients with symptomatic angina who require BP-lowering treatment, beta-blockers and/or CCBs are recommended as part of that treatment.	I	A
In patients with symptomatic HFrEF/HFmrEF, the following treatments with BP-lowering effects are recommended to improve outcomes: ACE inhibitors (or ARBs if ACE inhibitors are not tolerated) or ARNi, beta-blockers, MRAs, and SGLT2 inhibitors.	I	A
In hypertensive patients with symptomatic HFpEF, SGLT2 inhibitors are recommended to improve outcomes in the context of their modest BP-lowering properties.	I	A
Other conditions		
It is recommended that the BP-lowering drug treatment strategy for preventing stroke should comprise a RAS blocker plus a CCB or a thiazide-like diuretic.	I	A
In patients with confirmed BP ≥130/80 mmHg with a history of TIA or stroke a systolic BP target 120–129 mmHg is recommended to reduce CVD outcomes, provided treatment is tolerated.	I	A
Renal artery angioplasty is not recommended in patients without confirmed haemodynamically significant renal artery stenosis.	III	A
10. Acute and short-term lowering of blood pressure		
Intracerebral haemorrhage or acute ischaemic stroke		
For patients with ischaemic stroke or TIA and an indication for BP lowering, it is recommended that BP lowering therapy should be commenced before hospital discharge.	I	B
In patients with intracerebral haemorrhage presenting with systolic BP ≥220 mmHg, acute reduction in systolic BP >70 mmHg from initial levels within 1 h of commencing treatment is not recommended.	III	B

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Severe hypertension in pregnancy and pre-eclampsia		
In pre-eclampsia or eclampsia with hypertensive crisis, drug treatment with i.v. labetalol or nicardipine and magnesium is recommended.	I	C
In pre-eclampsia or eclampsia associated with pulmonary oedema, nitroglycerin given as an i.v. infusion is recommended.	I	C
In severe hypertension in pregnancy: • drug treatment with i.v. labetalol, oral methyldopa, or oral nifedipine is recommended. Intravenous hydralazine is a second-line option.	I	C
11. Patient-centred care in hypertension		
An informed discussion about CVD risk and treatment benefits tailored to the needs of a patient is recommended as part of hypertension management.	I	C
Home BP measurement for managing hypertension by using self-monitored BP is recommended to achieve better BP control.	I	B
Self-measurement, when properly performed, is recommended due to positive effects on the acceptance of a diagnosis of hypertension, patient empowerment, and adherence to treatment.	I	C
Multidisciplinary approaches in the management of patients with elevated BP and hypertension, including appropriate and safe task-shifting away from physicians are recommended to improve BP control.	I	A

ABPM, ambulatory blood pressure monitoring; ACE, angiotensin-converting enzyme; ACR, albumin:creatinine ratio; AF, atrial fibrillation; ALARA, as low as reasonably achievable; ARB, angiotensin receptor blocker; ARNi, angiotensin receptor–neprilysin inhibitor; BMI, body mass index; BP, blood pressure; CCB, calcium channel blocker; CKD, chronic kidney disease; CVD, cardiovascular disease; DASH, Dietary Approaches to Stop Hypertension; ECG, electrocardiogram; eGFR, estimated glomerular filtration rate; ESC, European Society of Cardiology; HBPM, home blood pressure monitoring; HFpEF, heart failure with preserved ejection fraction; HF(m)rEF, heart failure with (mildly) reduced ejection fraction; HMOD, hypertension-mediated organ damage; i.v., intravenous; MRA, mineralocorticoid receptor antagonist; RAS, renin–angiotensin system; SCORE2, Systematic COronary Risk Evaluation 2; SCORE2-OP, Systematic COronary Risk Evaluation 2-Older Persons; SGLT2, sodium–glucose co-transporter 2; TIA, transient ischaemic attack.

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15. Evidence tables

Evidence tables are available at *European Heart Journal* online.

16. Data availability statement

No new data were generated or analysed in support of this research.

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18. Appendix

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