2017 ACC/AHA Guideline for High Blood Pressure in Adults

Comprehensive Hypertension Management Study Guide

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Clinical Practice Guideline

2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults

A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines

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ACC/AHA 2017 Hypertension Guideline

CardioDetect Clinical Advisor Module

December 2024

Quick Reference Cheat Sheet

CLINICAL DECISION SUPPORT: Rapid reference for BP categories and pharmacological interventions.

1. Blood Pressure Thresholds & Actions

Category	SBP (mmHg)	DBP (mmHg)	Clinical Action
Normal	<120	<80	Promote optimal lifestyle
Elevated	120-129	<80	Non-pharm therapy
Stage 1 (Low Risk)	130-139	80-89	Non-pharm therapy
Stage 1 (High Risk*)	130-139	80-89	INITIATE MEDICATION (+ Lifestyle)
Stage 2	≥140	≥90	INITIATE MEDICATION (+ Lifestyle)
Hypertensive Crisis	>180	>120	EVALUATE FOR ORGAN DAMAGE

**High Risk = 10-year ASCVD risk ≥10%, CVD, Diabetes, or CKD.*

2. Preferred First-Line Agents

Class	Examples	Primary Indication
Thiazide Diuretics	Chlorthalidone, HCTZ	General pop, Elderly
ACE Inhibitors	Lisinopril, Enalapril	CKD, Diabetes, Heart Failure
ARBs	Losartan, Valsartan	ACEi intolerant, CKD, Diabetes
CCBs	Amlodipine, Nifedipine	General pop, Elderly

Executive Summary

The 2017 ACC/AHA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults provides comprehensive, evidence-based recommendations for blood pressure management. This landmark guideline lowered the threshold for hypertension diagnosis from 140/90 to 130/80 mmHg.

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Preamble

Since 1980, the American College of Cardiology (ACC) and American Heart Association (AHA) have translated scientific evidence into clinical practice guidelines (guidelines) with recommendations to improve cardiovascular health. In 2013, the National Heart, Lung, and Blood Institute (NHLBI) Advisory Council recommended that the NHLBI focus specifically on reviewing the highest-quality evidence and partner with other organizations to develop recommendations.[P-1,P-2] Accordingly, the ACC and AHA collaborated with the NHLBI and stakeholder and professional organizations to complete and publish 4 guidelines (on assessment of cardiovascular risk, lifestyle modifications to reduce cardiovascular risk, management of blood cholesterol in adults, and management of overweight and obesity in adults) to make them available to the widest possible constituency. In 2014, the ACC and AHA, in partnership with several other professional societies, initiated a guideline on the prevention, detection, evaluation, and management of high blood pressure (BP) in adults. Under the management of the ACC/AHA Task Force, a Prevention Subcommittee was appointed to help guide development of the suite of guidelines on prevention of cardiovascular disease (CVD). These guidelines, which are based on systematic methods to evaluate and classify evidence, provide a cornerstone for quality cardiovascular care. The ACC and AHA sponsor the development and publication of guidelines without commercial support, and members of each organization volunteer their time to the writing and review efforts. Guidelines are official policy of the ACC and AHA.

Intended Use

Practice guidelines provide recommendations applicable to patients with or at risk of developing CVD. The focus is on medical practice in the United States, but guidelines developed in collaboration with other organizations can have a global impact. Although guidelines may be used to inform regulatory or payer decisions, they are intended to improve patients' quality of care and align with patients' interests. Guidelines are intended to define practices meeting the needs of patients in most, but not all, circumstances and should not replace clinical judgment.

Clinical Implementation

Management in accordance with guideline recommendations is effective only when followed by both practitioners and patients. Adherence to recommendations can be enhanced by shared decision making between clinicians and patients, with patient engagement in selecting interventions on the basis of individual values, preferences, and associated conditions and comorbidities.

Methodology and Modernization

The ACC/AHA Task Force on Clinical Practice Guidelines (Task Force) continuously reviews, updates, and modifies guideline methodology on the basis of published standards from organizations, including the Institute of Medicine,[P-3,P-4] and on the basis of internal reevaluation. Similarly, the presentation and delivery of guidelines are reevaluated and modified on the basis of evolving technologies and other factors to facilitate optimal dissemination of information to healthcare professionals at the point of care.

Toward this goal, this guideline continues the introduction of an evolved format of presenting guideline recommendations and associated text called the "modular knowledge chunk format." Each modular "chunk" includes a table of related recommendations, a brief synopsis, recommendation-specific supportive text, and when appropriate, flow diagrams or additional tables. References are provided within the modular chunk itself to facilitate quick review. Additionally, this format will facilitate seamless updating of guidelines with focused updates as new evidence is published, as well as content tagging for rapid electronic retrieval of related recommendations on a topic of interest. This evolved approach format was instituted when this guideline was near completion; therefore, the present document represents a transitional format that best suits the text as written. Future guidelines will fully implement this format, including provisions for limiting the amount of text in a guideline.

Recognizing the importance of cost–value considerations in certain guidelines, when appropriate and feasible, an analysis of the value of a drug, device, or intervention may be performed in accordance with the ACC/AHA methodology.[P-5]

To ensure that guideline recommendations remain current, new data are reviewed on an ongoing basis, with full guideline revisions commissioned in approximately 6-year cycles. Publication of new, potentially practice-changing study results that are relevant to an existing or new drug, device, or management strategy will prompt evaluation by the Task Force, in consultation with the relevant guideline writing committee, to determine whether a focused update should be commissioned. For additional information and policies regarding guideline development, we encourage readers to consult the ACC/AHA guideline methodology manual[P-6] and other methodology articles.[P-7–P-10]

Selection of Writing Committee Members

The Task Force strives to avoid bias by selecting experts from a broad array of backgrounds. Writing committee members represent different geographic regions, sexes, ethnicities, races, intellectual perspectives/biases, and scopes

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of clinical practice. The Task Force may also invite organizations and professional societies with related interests and expertise to participate as partners, collaborators, or endorsers.

Relationships With Industry and Other Entities

The ACC and AHA have rigorous policies and methods to ensure that guidelines are developed without bias or improper influence. The complete relationships with industry and other entities (RWI) policy can be found online. Appendix 1 of the present document lists writing committee members' relevant RWI. For the purposes of full transparency, writing committee members' comprehensive disclosure information is available online. Comprehensive disclosure information for the Task Force is available online.

Evidence Review and Evidence Review Committees

In developing recommendations, the writing committee uses evidence-based methodologies that are based on all available data.[P-6–P-9] Literature searches focus on randomized controlled trials (RCTs) but also include registries, nonrandomized comparative and descriptive studies, case series, cohort studies, systematic reviews, and expert opinion. Only key references are cited.

An independent evidence review committee (ERC) is commissioned when there are 1 or more questions deemed of utmost clinical importance that merit formal systematic review. The systematic review will determine which patients are most likely to benefit from a drug, device, or treatment strategy and to what degree. Criteria for commissioning an ERC and formal systematic review include: a) the absence of a current authoritative systematic review, b) the feasibility of defining the benefit and risk in a time frame consistent with the writing of a guideline, c) the relevance to a substantial number of patients, and d) the likelihood that the findings can be translated into actionable recommendations. ERC members may include methodologists, epidemiologists, healthcare providers, and biostatisticians. The recommendations developed by the writing committee on the basis of the systematic review are marked with "SR."

Guideline-Directed Management and Therapy

The term guideline-directed management and therapy (GDMT) encompasses clinical evaluation, diagnostic testing, and pharmacological and procedural treatments. For these and all recommended drug treatment regimens, the reader should confirm the dosage by reviewing product insert material and evaluate the treatment regimen for contraindications and interactions. The recommendations are limited to drugs, devices, and treatments approved for clinical use in the United States.

Class of Recommendation and Level of Evidence

The Class of Recommendation (COR) indicates the strength of the recommendation, encompassing the estimated magnitude and certainty of benefit in proportion to risk. The Level of Evidence (LOE) rates the quality of scientific evidence that supports the intervention on the basis of the type, quantity, and consistency of data from clinical trials and other sources (Table 1).[P-6–P-8]

Glenn N. Levine, MD, FACC, FAHA
Chair, ACC/AHA Task Force on Clinical Practice Guidelines

1. Introduction

As early as the 1920s, and subsequently in the 1959 Build and Blood Pressure Study[S1.5-1] of almost 5 million adults insured between 1934 and 1954, a strong direct relationship was noted between level of BP and risk of clinical complications and death. In the 1960s, these findings were confirmed in a series of reports from the Framingham Heart Study.[S1.5-2] The 1967 and 1970 Veterans Administration Cooperative Study Group reports ushered in the era of effective treatment for high BP.[S1.5-3,S1.5-4] The first comprehensive guideline for detection, evaluation, and management of high BP was published in 1977, under the sponsorship of the NHLBI.[S1.5-5] In subsequent years, a series of Joint National Committee (JNC) BP guidelines were published to assist the practice community and improve prevention, awareness, treatment, and control of high BP.[S1.5-5–S1.5-7] The present guideline updates prior JNC reports.

1.1. Methodology and Evidence Review

An extensive evidence review, which included literature derived from research involving human subjects, published in English, and indexed in MEDLINE (through PubMed), EMBASE, the Cochrane Library, the Agency for Healthcare Research and Quality, and other selected databases relevant to this guideline, was conducted between February and August 2015. Key search words included but were not limited to the following: *adherence; aerobic; alcohol intake; ambulatory care; antihypertensive: agents, drug, medication, therapy; beta adrenergic blockers; blood pressure: arterial, control, determination, devices, goal, high, improve, measurement, monitoring, ambulatory; calcium channel blockers; diet; diuretic agent; drug therapy; heart failure: diastolic, systolic; hypertension: white coat, masked, ambulatory, isolated ambulatory, isolated clinic, diagnosis, reverse white coat, prevention, therapy, treatment, control; intervention; lifestyle: measures, modification; office visits; patient outcome; performance measures; physical activity; potassium intake; protein intake; renin inhibitor; risk reduction: behavior, counseling; screening; sphygmomanometers; spironolactone; therapy; treatment: adherence, compliance, efficacy, outcome, protocol, regimen; weight.* Additional relevant studies published through June 2016, during the guideline writing process, were also considered by the writing committee and added to the evidence tables when appropriate. The final evidence tables included in the Online Data Supplement summarize the evidence used by the writing committee to formulate recommendations.

As noted in the preamble, an independent ERC was commissioned to perform a formal systematic review of 4 critical clinical questions related to hypertension (Table 2), the results of which were considered by the writing committee for incorporation into this guideline. Concurrent with this process, writing committee members evaluated other published data relevant to the guideline. The findings of the ERC and the writing committee members were formally presented and

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Table 1. Applying Class of Recommendation and Level of Evidence to Clinical Strategies, Interventions, Treatments, or Diagnostic Testing in Patient Care* (Updated August 2015)

CLASS (STRENGTH) OF RECOMMENDATION

CLASS I (STRONG) — Benefit >>> Risk

Suggested phrases for writing recommendations:
- Is recommended
- Is indicated/useful/effective/beneficial
- Should be performed/administered/other
- Comparative-Effectiveness Phrases†:
  - Treatment/strategy A is recommended/indicated in preference to treatment B
  - Treatment A should be chosen over treatment B

CLASS IIa (MODERATE) — Benefit >> Risk

Suggested phrases for writing recommendations:
- Is reasonable
- Can be useful/effective/beneficial
- Comparative-Effectiveness Phrases†:
  - Treatment/strategy A is probably recommended/indicated in preference to treatment B
  - It is reasonable to choose treatment A over treatment B

CLASS IIb (WEAK) — Benefit ≥ Risk

Suggested phrases for writing recommendations:
- May/might be reasonable
- May/might be considered
- Usefulness/effectiveness is unknown/unclear/uncertain or not well established

CLASS III: No Benefit (MODERATE) — Benefit = Risk
(Generally, LOE A or B use only)

Suggested phrases for writing recommendations:
- Is not recommended
- Is not indicated/useful/effective/beneficial
- Should not be performed/administered/other

CLASS III: Harm (STRONG) — Risk > Benefit

Suggested phrases for writing recommendations:
- Potentially harmful
- Causes harm
- Associated with excess morbidity/mortality
- Should not be performed/administered/other

LEVEL (QUALITY) OF EVIDENCE‡

LEVEL A
- High-quality evidence‡ from more than 1 RCT
- Meta-analyses of high-quality RCTs
- One or more RCTs corroborated by high-quality registry studies

LEVEL B-R (Randomized)
- Moderate-quality evidence‡ from 1 or more RCTs
- Meta-analyses of moderate-quality RCTs

LEVEL B-NR (Nonrandomized)
- Moderate-quality evidence‡ from 1 or more well-designed, well-executed nonrandomized studies, observational studies, or registry studies
- Meta-analyses of such studies

LEVEL C-LD (Limited Data)
- Randomized or nonrandomized observational or registry studies with limitations of design or execution
- Meta-analyses of such studies
- Physiological or mechanistic studies in human subjects

LEVEL C-EO (Expert Opinion)

Consensus of expert opinion based on clinical experience

COR and LOE are determined independently (any COR may be paired with any LOE).

A recommendation with LOE C does not imply that the recommendation is weak. Many important clinical questions addressed in guidelines do not lend themselves to clinical trials. Although RCTs are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

* The outcome or result of the intervention should be specified (an improved clinical outcome or increased diagnostic accuracy or incremental prognostic information).

† For comparative-effectiveness recommendations (COR I and IIa; LOE A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

‡ The method of assessing quality is evolving, including the application of standardized, widely used, and preferably validated evidence grading tools; and for systematic reviews, the incorporation of an Evidence Review Committee.

COR indicates Class of Recommendation; EO, expert opinion; LD, limited data; LOE, Level of Evidence; NR, nonrandomized; R, randomized; and RCT, randomized controlled trial.

discussed, and then guideline recommendations were developed. The systematic review report, "Systematic Review for the 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults," is published in conjunction with this guideline,[S1.5-8] and its respective data supplements are available online. No writing committee member reported a RWI. Drs. Whelton, Wright, and Williamson had leadership roles in SPRINT (Systolic Blood Pressure Intervention Trial). Dr. Carey chaired committee discussions in which the SPRINT results were considered.

1.2. Organization of the Writing Committee

The writing committee consisted of clinicians, cardiologists, epidemiologists, internists, an endocrinologist, a geriatrician, a nephrologist, a neurologist, a nurse, a pharmacist, a physician assistant, and 2 lay/patient representatives. It

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Table 2. Systematic Review Questions on High BP in Adults

Question Number	Question	Section Number
1	Is there evidence that self-directed monitoring of BP and/or ambulatory BP monitoring are superior to office-based measurement of BP by a healthcare worker for 1) preventing adverse outcomes for which high BP is a risk factor and 2) achieving better BP control?	4.2
2	What is the optimal target for BP lowering during antihypertensive therapy in adults?	8.1.5 9.3 9.6
3	In adults with hypertension, do various antihypertensive drug classes differ in their comparative benefits and harms?	8.1.6 8.2
4	In adults with hypertension, does initiating treatment with antihypertensive pharmacological monotherapy versus initiating treatment with 2 drugs (including fixed-dose combination therapy), either of which may be followed by the addition of sequential drugs, differ in comparative benefits and/or harms on specific health outcomes?	8.1.6.1

BP indicates blood pressure.

included representatives from the ACC, AHA, American Academy of Physician Assistants (AAPA), Association of Black Cardiologists (ABC), American College of Preventive Medicine (ACPM), American Geriatrics Society (AGS), American Pharmacists Association (APhA), American Society of Hypertension (ASH), American Society for Preventive Cardiology (ASPC), National Medical Association (NMA), and Preventive Cardiovascular Nurses Association (PCNA).

1.3. Document Review and Approval

This document was reviewed by 2 official reviewers nominated by the ACC and AHA; 1 reviewer each from the AAPA, ABC, ACPM, AGS, APhA, ASH, ASPC, NMA, and PCNA; and 38 individual content reviewers. Reviewers' RWI information was distributed to the writing committee and is published in this document (Appendix 2).

This document was approved for publication by the governing bodies of the ACC, AHA, AAPA, ABC, ACPM, AGS, APhA, ASH, ASPC, NMA, and PCNA.

1.4. Scope of the Guideline

The present guideline is intended to be a resource for the clinical and public health practice communities. It is designed to be comprehensive but succinct and practical in providing guidance for prevention, detection, evaluation, and management of high BP. It is an update of the NHLBI publication, "The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure" (JNC 7).[S1.5-7] It incorporates new information from studies of office-based BP-related risk of CVD, ambulatory blood pressure monitoring (ABPM), home blood pressure monitoring (HBPM), telemedicine, and various other areas. This guideline does not address the use of BP-lowering medications for the purposes of prevention of recurrent CVD events in patients with stable ischemic heart disease (SIHD) or chronic heart failure (HF) in the absence of hypertension; these topics are the focus of other ACC/AHA guidelines.[S1.5-9,S1.5-10] In developing the present guideline, the writing committee reviewed prior published guidelines, evidence reviews, and related statements. Table 3 contains a list of publications and statements deemed pertinent to this writing effort and is intended for use as a resource, thus obviating the need to repeat existing guideline recommendations.

1.5. Abbreviations and Acronyms

Abbreviation/Acronym	Meaning/Phrase
ABPM	ambulatory blood pressure monitoring
ACE	angiotensin-converting enzyme
AF	atrial fibrillation
ARB	angiotensin receptor blocker
BP	blood pressure
CCB	calcium channel blocker
CHD	coronary heart disease
CKD	chronic kidney disease
CPAP	continuous positive airway pressure
CVD	cardiovascular disease
DBP	diastolic blood pressure
DM	diabetes mellitus
ECG	electrocardiogram
ESRD	end-stage renal disease
GDMT	guideline-directed management and therapy
GFR	glomerular filtration rate
HBPM	home blood pressure monitoring
EHR	electronic health record
HF	heart failure
HF*p*EF	heart failure with preserved ejection fraction
HF*r*EF	heart failure with reduced ejection fraction
ICH	intracerebral hemorrhage
JNC	Joint National Commission
LV	left ventricular
LVH	left ventricular hypertrophy
MI	myocardial infarction
MRI	magnetic resonance imaging
PAD	peripheral artery disease
RAS	renin-angiotensin system
RCT	randomized controlled trial
SBP	systolic blood pressure
SIHD	stable ischemic heart disease
TIA	transient ischemic attack

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Table 3. Associated Guidelines and Statements

Title	Organization	Publication Year
Guidelines		
Lower-extremity peripheral artery disease	AHA/ACC	2016[S1.5-11]
Management of primary aldosteronism: case detection, diagnosis, and treatment	Endocrine Society	2016[S1.5-12]
Stable ischemic heart disease	ACC/AHA/AATS/PCNA/SCAI/STS	2014[S1.5-13]* 2012[S1.5-9]
Pheochromocytoma and paraganglioma	Endocrine Society	2014[S1.5-14]
Atrial fibrillation	AHA/ACC/HRS	2014[S1.5-15]
Valvular heart disease	ACC/AHA	2017[S1.5-16]
Assessment of cardiovascular risk	ACC/AHA	2013[S1.5-17]
Hypertension in pregnancy	ACOG	2013[S1.5-18]
Heart failure	ACC/AHA	2017[S1.5-19] 2013[S1.5-10]
Lifestyle management to reduce cardiovascular risk	AHA/ACC	2013[S1.5-20]
Management of arterial hypertension	ESH/ESC	2013[S1.5-21]
Management of overweight and obesity in adults	AHA/ACC/TOS	2013[S1.5-22]
ST-elevation myocardial infarction	ACC/AHA	2013[S1.5-23]
Treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults	ACC/AHA	2013[S1.5-24]
Cardiovascular diseases during pregnancy	ESC	2011[S1.5-25]
Effectiveness-based guidelines for the prevention of cardiovascular disease in women	AHA/ACC	2011[S1.5-26]
Secondary prevention and risk-reduction therapy for patients with coronary and other atherosclerotic vascular disease	AHA/ACC	2011[S1.5-27]
Assessment of cardiovascular risk in asymptomatic adults	ACC/AHA	2010[S1.5-28]
Thoracic aortic disease	ACC/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM	2010[S1.5-29]
Diagnosis, evaluation, and treatment of high blood pressure in children and adolescents	NHLBI	2004[S1.5-30]
Statements		
Salt sensitivity of blood pressure	AHA	2016[S1.5-31]
Cardiovascular team-based care and the role of advanced practice providers	ACC	2015[S1.5-32]
Treatment of hypertension in patients with coronary artery disease	AHA/ACC/ASH	2015[S1.5-33]
Ambulatory blood pressure monitoring in children and adolescents	AHA	2014[S1.5-34]
An effective approach to high blood pressure control	AHA/ACC/CDC	2014[S1.5-35]
Ambulatory blood pressure monitoring	ESH	2013[S1.5-36]
Performance measures for adults with coronary artery disease and hypertension	ACC/AHA/AMA-PCPI	2011[S1.5-37]
Interventions to promote physical activity and dietary lifestyle changes for cardiovascular risk factor reduction in adults	AHA	2010[S1.5-38]
Resistant hypertension: diagnosis, evaluation, and treatment	AHA	2008[S1.5-39]

*The full-text SIHD guideline is from 2012.[S1.5-9] A focused update was published in 2014.[S1.5-13]

AATS indicates American Association for Thoracic Surgery; ACC, American College of Cardiology; ACOG, American College of Obstetricians and Gynecologists; ACR, American College of Radiology; AHA, American Heart Association; AMA, American Medical Association; ASA, American Stroke Association; ASH, American Society of Hypertension; CDC, Centers for Disease Control and Prevention; ESC, European Society of Cardiology; ESH, European Society of Hypertension; HRS, Heart Rhythm Society; NHLBI, National Heart, Lung, and Blood Institute; PCNA, Preventive Cardiovascular Nurses Association; PCPI, Physician Consortium for Performance Improvement; SCA, Society of Cardiovascular Anesthesiologists; SCAI, Society for Cardiovascular Angiography and Interventions; SIHD, stable ischemic heart disease; SIR, Society of Interventional Radiology; STS, Society of Thoracic Surgeons; SVM, Society for Vascular Medicine; and TOS, The Obesity Society.

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2. BP and CVD Risk

2.1. Observational Relationship

Observational studies have demonstrated graded associations between higher systolic blood pressure (SBP) and diastolic blood pressure (DBP) and increased CVD risk.[S2.1-1,S2.1-2] In a meta-analysis of 61 prospective studies, the risk of CVD increased in a log-linear fashion from SBP levels <115 mm Hg to >180 mm Hg and from DBP levels <75 mm Hg to >105 mm Hg.[S2.1-1] In that analysis, 20 mm Hg higher SBP and

10 mm Hg higher DBP were each associated with a doubling in the risk of death from stroke, heart disease, or other vascular disease. In a separate observational study including >1 million adult patients ≥30 years of age, higher SBP and DBP were associated with increased risk of CVD incidence and angina, myocardial infarction (MI), HF, stroke, peripheral artery disease (PAD), and abdominal aortic aneurysm, each evaluated separately.[S2.1-2] An increased risk of CVD associated with higher SBP and DBP has been reported across a broad age spectrum, from 30 years to >80 years of age. Although the relative risk of incident CVD associated with higher SBP and DBP is smaller at older ages, the corresponding high BP–related increase in absolute risk is larger in older persons (≥65 years) given the higher absolute risk of CVD at an older age.[S2.1-1]

2.2. BP Components

Epidemiological studies have evaluated associations of SBP and DBP, as well as derived components of BP measurements (including pulse pressure, mean BP, and mid-BP), with CVD outcomes (Table 4). When considered separately, higher levels of both SBP and DBP have been associated with increased CVD risk.[S2.2-1,S2.2-2] Higher SBP has consistently been associated with increased CVD risk after adjustment for, or within strata of, DBP.[S2.2-3–S2.2-5] In contrast, after consideration of SBP through adjustment or stratification, DBP has not been consistently associated with CVD risk.[S2.2-6,S2.2-7] Although pulse pressure and mid-BP have been associated with increased CVD risk independent of SBP and DBP in some studies, SBP (especially) and DBP are prioritized in the present document because of the robust evidence base for these measures in both observational studies and clinical trials and because of their ease of measurement in practice settings.[S2.2-8–S2.2-11]

2.3. Population Risk

In 2010, high BP was the leading cause of death and disability-adjusted life years worldwide.[S2.3-1,S2.3-2] In the United States, hypertension (see Section 3.1 for definition) accounted for more CVD deaths than any other modifiable CVD risk factor and was second only to cigarette smoking as a preventable cause of death for any reason.[S2.3-3] In a follow-up study of 23 272 US NHANES (National Health and Nutrition Examination Survey) participants, >50% of deaths from coronary heart disease (CHD) and stroke occurred among individuals with hypertension.[S2.3-4] Because of the high prevalence of hypertension and its associated increased risk of CHD, stroke, and end-stage renal disease (ESRD), the population-attributable risk of these outcomes associated with hypertension is high.[S2.3-4,S2.3-5] In the population-based ARIC (Atherosclerosis Risk in Communities) study, 25% of the cardiovascular events (CHD, coronary revascularization, stroke, or HF) were attributable to hypertension. In the Northern Manhattan study, the percentage of events attributable to hypertension was higher in women (32%) than in men (19%) and higher in blacks (36%) than in whites (21%).[S2.3-6] In 2012, hypertension was the second leading assigned cause of ESRD, behind diabetes mellitus (DM), and accounted for 34% of incident ESRD cases in the US population.[S2.3-7]

Table 4. BP Measurement Definitions

BP Measurement	Definition
SBP	First Korotkoff sound*
DBP	Fifth Korotkoff sound*
Pulse pressure	SBP minus DBP
Mean arterial pressure	DBP plus one third pulse pressure†
Mid-BP	Sum of SBP and DBP, divided by 2

*See Section 4 for a description of Korotkoff sounds.

†Calculation assumes normal heart rate.

BP indicates blood pressure; DBP, diastolic blood pressure; and SBP, systolic blood pressure.

2.4. Coexistence of Hypertension and Related Chronic Conditions

Recommendation for Coexistence of Hypertension and Related Chronic Conditions

References that support the recommendation are summarized in Online Data Supplement 1.

COR	LOE	Recommendation
I	B-NR	1. Screening for and management of other modifiable CVD risk factors are recommended in adults with hypertension.[S2.4-1,S2.4-2]

Synopsis

Many adult patients with hypertension have other CVD risk factors; a list of such modifiable and relatively fixed risk factors is provided in Table 5. Among US adults with hypertension between 2009 and 2012, 15.5% were current smokers, 49.5% were obese, 63.2% had hypercholesterolemia, 27.2% had DM, and 15.8% had chronic kidney disease (CKD; defined as estimated glomerular filtration rate [eGFR] <60 mL/min/1.73 m^2 and/or urine albumin:creatinine ≥300 mg/g).[S2.4-3]

Not only are CVD risk factors common among adults with hypertension, a higher percentage of adults with CVD risk

Table 5. CVD Risk Factors Common in Patients With Hypertension

Modifiable Risk Factors*	Relatively Fixed Risk Factors†
Current cigarette smoking, secondhand smoking	CKD
	Family history
Diabetes mellitus	Increased age
Dyslipidemia/hypercholesterolemia	Low socioeconomic/educational status
Overweight/obesity	Male sex
Physical inactivity/low fitness	Obstructive sleep apnea
Unhealthy diet	Psychosocial stress

*Factors that can be changed and, if changed, may reduce CVD risk.

†Factors that are difficult to change (CKD, low socioeconomic/educational status, obstructive sleep apnea[S2.4-12]), cannot be changed (family history, increased age, male sex), or, if changed through the use of current intervention techniques, may not reduce CVD risk (psychosocial stress).

CKD indicates chronic kidney disease; and CVD, cardiovascular disease.

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factors have hypertension. For example, 71% of US adults with diagnosed DM have hypertension.[S2.4-4] In the Chronic Renal Insufficiency Cohort (CRIC), 86% of the participants had hypertension.[S2.4-5] Also, 28.1% of adults with hypertension and CKD in the population-based REGARDS (Reasons for Geographic and Racial Differences in Stroke) study had apparent resistant hypertension.[S2.4-6] In NHANES 1999–2010, 35.7% of obese individuals had hypertension.[S2.4-7] The presence of multiple CVD risk factors in individuals with hypertension results in high absolute risks for CHD and stroke in this population. For example, among US adults with hypertension between 2009 and 2012, 41.7% had a 10-year CHD risk >20%, 40.9% had a risk of 10% to 20%, and only 18.4% had a risk <10%.[S2.4-3]

Modifiable risk factors for CVD that are common among adults with hypertension include cigarette smoking/tobacco smoke exposure, DM, dyslipidemia (including high levels of low-density lipoprotein cholesterol or hypercholesterolemia, high levels of triglycerides, and low levels of high-density lipoprotein cholesterol), overweight/obesity, physical inactivity/low fitness level, and unhealthy diet.[S2.4-8] The relationship between hypertension and other modifiable risk factors is complex and interdependent, with several sharing mechanisms of action and pathophysiology. CVD risk factors affect BP through over activation of the renin-angiotensin-aldosterone system, activation of the sympathetic nervous system, inhibition of the cardiac natriuretic peptide system, endothelial dysfunction, and other mechanisms.[S2.4-9–S2.4-11] Treating some of the other modifiable risk factors may reduce BP through modification of shared pathology, and CVD risk may be reduced by treating global risk factor burden.

Recommendation-Specific Supportive Text

1. Observational studies have demonstrated that CVD risk factors frequently occur in combination, with ≥3 risk factors present in 17% of patients.[S2.4-1] A meta-analysis from 18 cohort studies involving 257 384 patients identified a lifetime risk of CVD death, nonfatal MI, and fatal or nonfatal stroke that was substantially higher in adults with ≥2 CVD risk factors than in those with only 1 risk factor.[S2.4-1,S2.4-2]

3. Classification of BP

3.1. Definition of High BP

Recommendation for Definition of High BP References that support the recommendation are summarized in Online Data Supplement 2.		
COR	LOE	Recommendation
I	B-NR	1. BP should be categorized as normal, elevated, or stage 1 or 2 hypertension to prevent and treat high BP (Table 6).[S3.1-1–S3.1-20]

Synopsis

Although a continuous association exists between higher BP and increased CVD risk (see Section 2.1), it is useful to categorize BP levels for clinical and public health decision making. In the present document, BP is categorized into 4 levels on the basis of average BP measured in a healthcare setting (office pressures): normal, elevated, and stage 1 or 2 hypertension (Table 6). Online Data Supplement C illustrates schematically the SBP and DBP categories defining normal BP, elevated BP, and stages 1 and 2 hypertension. This categorization differs from that previously recommended in the JNC 7 report, with stage 1 hypertension now defined as an SBP of 130–139 or a DBP of 80–89 mm Hg, and with stage 2 hypertension in the present document corresponding to stages 1 and 2 in the JNC 7 report.[S3.1-21] The rationale for this categorization is based on observational data related to the association between SBP/DBP and CVD risk, RCTs of lifestyle modification to lower BP, and RCTs of treatment with antihypertensive medication to prevent CVD. The increased risk of CVD among adults with stage 2 hypertension is well established. An increasing number of individual studies and meta-analyses of observational data have reported a gradient of progressively higher CVD risk going from normal BP to elevated BP and stage 1 hypertension.[S3.1-4–S3.1-10,S3.1-12,S3.1-13,S3.1-16] In many of these meta-analyses, the hazard ratios for CHD and stroke were between 1.1 and 1.5 for the comparison of SBP/DBP of 120–129/80–84 mm Hg versus <120/80 mm Hg and between 1.5 and 2.0 for the comparison of SBP/DBP of 130–139/85–89 mm Hg versus <120/80 mm Hg. This risk gradient was consistent across subgroups defined by sex and race/ethnicity. The relative increase in CVD risk associated with higher BP was attenuated but still present among older adults.[S3.1-1] The prevalence of severe hypertension has been declining over time, but approximately 12.3% of US adults with hypertension have an average SBP ≥160 mm Hg or average DBP ≥100 mm Hg.[S3.1-22] Lifestyle modification and pharmacological antihypertensive treatment recommendations for individuals with elevated BP and stages 1 and 2 hypertension are provided in Sections 6 and 8, respectively. The relationship of this classification schema with measurements obtained by ambulatory BP recording and home BP measurements is discussed in Section 4.2.

Table 6. Categories of BP in Adults*

BP Category	SBP		DBP
Normal	<120 mm Hg	and	<80 mm Hg
Elevated	120–129 mm Hg	and	<80 mm Hg
Hypertension			
Stage 1	130–139 mm Hg	or	80–89 mm Hg
Stage 2	≥140 mm Hg	or	≥90 mm Hg

*Individuals with SBP and DBP in 2 categories should be designated to the higher BP category.

BP indicates blood pressure (based on an average of ≥2 careful readings obtained on ≥2 occasions, as detailed in Section 4); DBP, diastolic blood pressure; and SBP, systolic blood pressure.

Recommendation-Specific Supportive Text

1. As was the case in previous BP classification systems, the choice and the naming of the categories were based on a pragmatic interpretation of BP-related CVD risk and benefit of BP reduction in clinical trials. Meta-analyses of observational studies have demonstrated that elevated

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BP and hypertension are associated with increased risk of CVD, ESRD, subclinical atherosclerosis, and all-cause death.[S3.1-1–S3.1-17] The recommended BP classification system is most valuable in untreated adults as an aid in decisions about prevention or treatment of high BP. However, it is also useful in assessing the success of interventions to reduce BP.

3.2. Lifetime Risk of Hypertension

Observational studies have documented a relatively high incidence of hypertension over periods of 5 to 10 years of follow-up.[S3.2-1,S3.2-2] Thus, there is a much higher long-term population burden of hypertension as BP progressively increases with age. Several studies have estimated the long-term cumulative incidence of developing hypertension.[S3.2-3,S3.2-4] In an analysis of 1132 white male medical students (mean age: approximately 23 years at baseline) in the Johns Hopkins Precursors study, 0.3%, 6.5%, and 37% developed hypertension at age 25, 45, and 65 years, respectively.[S3.2-5] In MESA (Multi-Ethnic Study of Atherosclerosis), the percentage of the population developing hypertension over their lifetimes was higher for African Americans and Hispanics than for whites and Asians.[S3.2-3] For adults 45 years of age without hypertension, the 40-year risk of developing hypertension was 93% for African-American, 92% for Hispanic, 86% for white, and 84% for Chinese adults.[S3.2-3] In the Framingham Heart Study, approximately 90% of adults free of hypertension at age 55 or 65 years developed hypertension during their lifetimes.[S3.2-4] All of these estimates were based on use of the 140/90–mm Hg cutpoint for recognition of hypertension and would have been higher had the 130/80–mm Hg cutpoint been used.

3.3. Prevalence of High BP

Prevalence estimates are greatly influenced by the choice of cutpoints to categorize high BP, the methods used to establish the diagnosis, and the population studied.[S3.3-1,S3.3-2] Most general population prevalence estimates are derived from national surveys. Table 7 provides estimates for prevalence of hypertension in the US general adult population (≥20 years of age) that are based on the definitions of hypertension recommended in the present guideline and in the JNC 7 report. The prevalence of hypertension among US adults is substantially higher when the definition in the present guideline is used versus the JNC 7 definition (46% versus 32%). However, as described in Section 8.1, nonpharmacological treatment (not antihypertensive medication) is recommended for most US adults who have hypertension as defined in the present guideline but who would not meet the JNC 7 definition for hypertension. As a consequence, the new definition results in only a small increase in the percentage of US adults for whom antihypertensive medication is recommended in conjunction with lifestyle modification.

The prevalence of hypertension rises dramatically with increasing age and is higher in blacks than in whites, Asians, and Hispanic Americans. NHANES estimates of JNC 7–defined hypertension prevalence have remained fairly stable since the early 2000s.[S3.3-1] Most contemporary population surveys, including NHANES, rely on an average of BP measurements obtained at a single visit,[S3.3-2] which is likely to result in an overestimate of hypertension prevalence compared with what would be found by using an average of ≥2 readings taken on ≥2 visits,[S3.3-1] as recommended in current and previous BP guidelines.[S3.3-3–S3.3-5] The extent to which guideline recommendations for use of BP averages from ≥2 occasions is followed in practice is unclear. Adding self-report of previously diagnosed hypertension yields a 5% to 10% higher estimate of prevalence.[S3.3-1,S3.3-6,S3.3-7] Most individuals who were added by use of this expanded definition have been diagnosed as having hypertension by a health professional on >1 occasion, and many have been advised to change their lifestyle.[S3.3-2,S3.3-6]

Table 7. Prevalence of Hypertension Based on 2 SBP/DBP Thresholds*†

	SBP/DBP ≥130/80 mm Hg or Self-Reported Antihypertensive Medication†		SBP/DBP ≥140/90 mm Hg or Self-Reported Antihypertensive Medication‡	
Overall, crude	46%		32%	
	Men (n=4717)	Women (n=4906)	Men (n=4717)	Women (n=4906)
Overall, age-sex adjusted	48%	43%	31%	32%
Age group, y				
20–44	30%	19%	11%	10%
45–54	50%	44%	33%	27%
55–64	70%	63%	53%	52%
65–74	77%	75%	64%	63%
75+	79%	85%	71%	78%
Race-ethnicity§				
Non-Hispanic white	47%	41%	31%	30%
Non-Hispanic black	59%	56%	42%	46%
Non-Hispanic Asian	45%	36%	29%	27%
Hispanic	44%	42%	27%	32%

The prevalence estimates have been rounded to the nearest full percentage.

*130/80 and 140/90 mm Hg in 9623 participants (≥20 years of age) in NHANES 2011–2014.

†BP cutpoints for definition of hypertension in the present guideline.

‡BP cutpoints for definition of hypertension in JNC 7.

§Adjusted to the 2010 age-sex distribution of the US adult population.

BP indicates blood pressure; DBP, diastolic blood pressure; NHANES, National Health and Nutrition Examination Survey; and SBP, systolic blood pressure.

3.4. Awareness, Treatment, and Control

Prevalence estimates for awareness, treatment, and control of hypertension are usually based on self-reports of the hypertension diagnosis (awareness), use of BP-lowering medications in those with hypertension (treatment), and achievement of a satisfactory SBP/DBP during treatment of hypertension (control). Before the present publication, awareness and treatment in adults were based on the SBP/DBP cutpoints of 140/90 mm Hg, and control was based on an SBP/DBP <140/90 mm Hg. In the US general adult population, hypertension awareness, treatment, and control

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have been steadily improving since the 1960s,[S3.4-1–S3.4-4] with NHANES 2009 to 2012 prevalence estimates for men and women, respectively, being 80.2% and 85.4% for awareness, 70.9% and 80.6% for treatment (88.4% and 94.4% in those who were aware), 69.5% and 68.5% for control in those being treated, and 49.3% and 55.2% for overall control in adults with hypertension.[S3.4-5] The NHANES experience may underestimate awareness, treatment, and control of hypertension because it is based on BP estimates derived from an average of readings obtained at a single visit, whereas guidelines recommend use of BP averages of ≥2 readings obtained on ≥2 occasions. In addition, the current definition of control excludes the possibility of control resulting from lifestyle change or nonpharmacological interventions. NHANES hypertension control rates have been consistently higher in women than in men (55.3% versus 38.0% in 2009–2012); in whites than in blacks and Hispanics (41.3% versus 31.1% and 23.6%, respectively, in men, and 57.2% versus 43.2% and 52.9%, respectively, in women, for 2009–2012); and in older than in younger adults (50.5% in adults ≥60 years of age versus 34.4% in patients 18 to 39 years of age for 2011–2012) up to the seventh decade,[S3.4-4,S3.4-5] although control rates are considerably lower for those ≥75 years (46%) and only 39.8% for adults ≥80 years.[S3.4-6] In addition, control rates are higher for persons of higher socioeconomic status (43.2% for adults with an income >400% above the US government poverty line versus 30.2% for those below this line in 2003 to 2006).[S3.4-5] Research studies have repeatedly demonstrated that structured, goal-oriented BP treatment initiatives with feedback and provision of free medication result in a substantial improvement in BP control.[S3.4-7–S3.4-9] Control rates that are much higher than noted in the general population have been reported in care settings where a systems approach (detailed in Sections 12.2 and 12.3) has been implemented for insured adults.[S3.4-10–S3.4-12]

4. Measurement of BP

4.1. Accurate Measurement of BP in the Office

Recommendation for Accurate Measurement of BP in the Office		
COR	LOE	Recommendation
I	C-EO	1. For diagnosis and management of high BP, proper methods are recommended for accurate measurement and documentation of BP (Table 8).

Synopsis

Although measurement of BP in office settings is relatively easy, errors are common and can result in a misleading estimation of an individual's true level of BP. There are various methods for measuring BP in the office. The clinical standard of auscultatory measures calibrated to a column of mercury has given way to oscillometric devices (in part because of toxicological issues with mercury). Oscillometric devices use a sensor that detects oscillations in pulsatile blood volume during cuff inflation and deflation. BP is indirectly calculated from maximum amplitude algorithms that involve population-based data. For this reason, only devices with a

Table 8. Checklist for Accurate Measurement of BP[S4.1-3,S4.1-4]

Key Steps for Proper BP Measurements	Specific Instructions
Step 1: Properly prepare the patient	1. Have the patient relax, sitting in a chair (feet on floor, back supported) for >5 min. 2. The patient should avoid caffeine, exercise, and smoking for at least 30 min before measurement. 3. Ensure patient has emptied his/her bladder. 4. Neither the patient nor the observer should talk during the rest period or during the measurement. 5. Remove all clothing covering the location of cuff placement. 6. Measurements made while the patient is sitting or lying on an examining table do not fulfill these criteria.
Step 2: Use proper technique for BP measurements	1. Use a BP measurement device that has been validated, and ensure that the device is calibrated periodically.* 2. Support the patient's arm (eg, resting on a desk). 3. Position the middle of the cuff on the patient's upper arm at the level of the right atrium (the midpoint of the sternum). 4. Use the correct cuff size, such that the bladder encircles 80% of the arm, and note if a larger- or smaller-than-normal cuff size is used (Table 9). 5. Either the stethoscope diaphragm or bell may be used for auscultatory readings.[S4.1-5,S4.1-6]
Step 3: Take the proper measurements needed for diagnosis and treatment of elevated BP/hypertension	1. At the first visit, record BP in both arms. Use the arm that gives the higher reading for subsequent readings. 2. Separate repeated measurements by 1–2 min. 3. For auscultatory determinations, use a palpated estimate of radial pulse obliteration pressure to estimate SBP. Inflate the cuff 20–30 mm Hg above this level for an auscultatory determination of the BP level. 4. For auscultatory readings, deflate the cuff pressure 2 mm Hg per second, and listen for Korotkoff sounds.
Step 4: Properly document accurate BP readings	1. Record SBP and DBP. If using the auscultatory technique, record SBP and DBP as onset of the first Korotkoff sound and disappearance of all Korotkoff sounds, respectively, using the nearest even number. 2. Note the time of most recent BP medication taken before measurements.
Step 5: Average the readings	Use an average of ≥2 readings obtained on ≥2 occasions to estimate the individual's level of BP.
Step 6: Provide BP readings to patient	Provide patients the SBP/DBP readings both verbally and in writing.

*See Section 4.2 for additional guidance.

Adapted with permission from Mancia et al[S4.1-3] (Oxford University Press), Pickering et al[S4.1-2] (American Heart Association, Inc.), and Weir et al[S4.1-4] (American College of Physicians, Inc.).

BP indicates blood pressure; DBP, diastolic blood pressure; and SBP, systolic blood pressure.

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validated measurement protocol can be recommended for use (see Section 4.2 for additional details). Many of the newer oscillometric devices automatically inflate multiple times (in 1- to 2-minute intervals), allowing patients to be alone and undisturbed during measurement. Although much of the available BP-related risk information and antihypertensive treatment trial experience have been generated by using "traditional" office methods of BP measurement, there is a growing evidence base supporting the use of automated office BP measurements.[S4.1-1]

Recommendation-Specific Supportive Text

1. Accurate measurement and recording of BP are essential to categorize level of BP, ascertain BP-related CVD risk, and guide management of high BP. Most systematic errors in BP measurement can be avoided by following the suggestions provided in Table 8, including having the patient sit quietly for 5 minutes before a reading is taken, supporting the limb used to measure BP, ensuring the BP cuff is at heart level, using the correct cuff size (Table 9), and, for auscultatory readings, deflating the cuff slowly.[S4.1-2] In those who are already taking medication that affects BP, the timing of BP measurements in relation to ingestion of the patient's medication should be standardized. Because individual BP measurements tend to vary in an unpredictable or random fashion, a single reading is inadequate for clinical decision-making. An average of 2 to 3 BP measurements obtained on 2 to 3 separate occasions will minimize random error and provide a more accurate basis for estimation of BP. In addition to clinicians, other caregivers and patients who perform BP self-monitoring should be trained to follow the checklist in Table 8. Common errors in clinical practice that can lead to inaccurate estimation of BP include failure to allow for a rest period and/or talking with the patient during or immediately before the recording, improper patient positioning (eg, sitting or lying on an examination table), rapid cuff deflation (for auscultatory readings), and reliance on BPs measured at a single occasion.

4.2. Out-of-Office and Self-Monitoring of BP

Recommendation for Out-of-Office and Self-Monitoring of BP

References that support the recommendation are summarized in Online Data Supplement 3 and Systematic Review Report.

COR	LOE	Recommendation
I	A[SR]	1. Out-of-office BP measurements are recommended to confirm the diagnosis of hypertension (Table 11) and for titration of BP-lowering medication, in conjunction with telehealth counseling or clinical interventions.[S4.2-1–S4.2-4]

SR indicates systematic review.

Synopsis

Out-of-office measurement of BP can be helpful for confirmation and management of hypertension. Self-monitoring of BP refers to the regular measurement of BP by an individual at home or elsewhere outside the clinic setting. Among individuals with hypertension, self-monitoring of BP, without other interventions, has shown limited evidence for treatment-related BP reduction and achievement of BP control.[S4.2-1,S4.2-5,S4.2-6] However, with the increased recognition of inconsistencies between office and out-of-office BPs (see Section 4.4) and greater reduction in BP being recommended for hypertension control, increased attention is being paid to out-of-office BP readings. Although ABPM is generally accepted as the best out-of-office measurement method, HBPM is often a more practical approach in clinical practice. Recommended procedures for the collection of HBPM data are provided in Table 10. If self-monitoring is used, it is important to ensure that the BP measurement device used has been validated with an internationally accepted protocol and the results have been published in a peer-reviewed journal.[S4.2-7] A guide to the relationship between HBPM BP readings and corresponding readings obtained in the office and by ABPM is presented in Table 11. The precise relationships between office readings, ABPM, and HBPM are unsettled, but there is general agreement that office BPs are often higher than ABPM or HBPM BPs, especially at higher BPs.

Table 9. Selection Criteria for BP Cuff Size for Measurement of BP in Adults

Arm Circumference	Usual Cuff Size
22–26 cm	Small adult
27–34 cm	Adult
35–44 cm	Large adult
45–52 cm	Adult thigh

Adapted with permission from Pickering et al[S4.1-2] (American Heart Association, Inc.).

BP indicates blood pressure.

Recommendation-Specific Supportive Text

1. ABPM is used to obtain out-of-office BP readings at set intervals, usually over a period of 24 hours. HBPM is used to obtain a record of out-of-office BP readings taken by a patient. Both ABPM and HBPM typically provide BP estimates that are based on multiple measurements. A systematic review conducted by the US Preventive Services Task Force reported that ABPM provided a better method to predict long-term CVD outcomes than did office BPs. It incorporates new information from studies of HBPM, ABPM, the relationship of overall CVD risk to the effectiveness of blood pressure lowering, clinical outcomes related to different blood pressure goals, strategies to improve blood pressure control and various other areas. A small body of evidence suggested, but did not confirm, that HBPM could serve as a similar predictor of outcomes.[S4.2-4] Meta-analyses of RCTs have identified clinically useful reductions in SBP and DBP and achievement of BP goals at 6 months and 1 year when self-monitoring of BP has been used in conjunction with other interventions, compared with usual care. Meta-analyses of RCTs have identified only small

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Table 10. Procedures for Use of HBPM[S4.2–5–S4.2–7]

Patient training should occur under medical supervision, including:
Information about hypertension
Selection of equipment
Acknowledgment that individual BP readings may vary substantially
Interpretation of results
Devices:
Verify use of automated validated devices. Use of auscultatory devices (mercury, aneroid, or other) is not generally useful for HBPM because patients rarely master the technique required for measurement of BP with auscultatory devices.
Monitors with provision for storage of readings in memory are preferred.
Verify use of appropriate cuff size to fit the arm (Table 9).
Verify that left/right inter-arm differences are insignificant. If differences are significant, instruct patient to measure BPs in the arm with higher readings.
Instructions on HBPM procedures:
Remain still:
Avoid smoking, caffeinated beverages, or exercise within 30 min before BP measurements.
Ensure ≥5 min of quiet rest before BP measurements.
Sit correctly:
Sit with back straight and supported (on a straight-backed dining chair, for example, rather than a sofa).
Sit with feet flat on the floor and legs uncrossed.
Keep arm supported on a flat surface (such as a table), with the upper arm at heart level.
Bottom of the cuff should be placed directly above the antecubital fossa (bend of the elbow).
Take multiple readings:
Take at least 2 readings 1 min apart in morning before taking medications and in evening before supper. Optimally, measure and record BP daily. Ideally, obtain weekly BP readings beginning 2 weeks after a change in the treatment regimen and during the week before a clinic visit.
Record all readings accurately:
Monitors with built-in memory should be brought to all clinic appointments.
BP should be based on an average of readings on ≥2 occasions for clinical decision making.
The information above may be reinforced with videos available online.

See Table 11 for HBPM targets.
BP indicates blood pressure; and HBPM, home blood pressure monitoring.

net reductions in SBP and DBP at 6 months and 1 year for use of self-monitoring of BP on its own, as compared with usual care.[S4.2-1,S4.2-5,S4.2-6] See Section 4.4 for additional details of diagnostic classification and Section 12 for additional details of telehealth and out-of-office BP measurement for management of high BP.

4.3. Ambulatory BP Monitoring

All of the major RCTs have been based on use of clinic BP readings. However, ABPM is often used to supplement BP readings obtained in office settings.[S4.3-1] The monitors are usually programmed to obtain readings every 15 to 30 minutes throughout the day and every 15 minutes to 1 hour during the night. ABPM is conducted while individuals go about their normal daily activities. ABPM can a) provide estimates of mean BP over the entire monitoring period and separately during nighttime and daytime, b) determine the daytime-to-nighttime BP ratio to identify the extent of nocturnal "dipping," c) identify the early-morning BP surge pattern, d) estimate BP variability, and e) allow for recognition of symptomatic hypotension. The US Centers for Medicaid & Medicare Services and other agencies provide reimbursement for ABPM in patients with suspected white coat hypertension.[S4.3-2] Medicare claims for ABPM between 2007 and 2010 were reimbursed at a median of $52 and were submitted for <1% of beneficiaries.[S4.3-3,S4.3-4] A list of devices validated for ABPM is available.[S4.3-5,S4.3-6]

Table 11. Corresponding Values of SBP/DBP for Clinic, HBPM, Daytime, Nighttime, and 24-Hour ABPM Measurements

Clinic	HBPM	Daytime ABPM	Nighttime ABPM	24-Hour ABPM
120/80	120/80	120/80	100/65	115/75
130/80	130/80	130/80	110/65	125/75
140/90	135/85	135/85	120/70	130/80
160/100	145/90	145/90	140/85	145/90

ABPM indicates ambulatory blood pressure monitoring; BP, blood pressure; DBP, diastolic blood pressure; HBPM, home blood pressure monitoring; and SBP, systolic blood pressure.

ABPM and HBPM definitions of high BP use different BP thresholds than those used by the previously mentioned office-based approach to categorize high BP identified in Section 3.1. Table 11 provides best estimates for corresponding home, daytime, nighttime, and 24-hour ambulatory levels of BP, including the values recommended for identification of hypertension with office measurements. Typically, a clinic BP of 140/90 mm Hg corresponds to home BP values of 135/85 mm Hg and to ABPM values defined as a daytime SBP/DBP of 135/85 mm Hg, a nighttime SBP/DBP of 120/70 mm Hg, and a 24-hour SBP/DBP of 130/80 mm Hg.[S4.3-7,S4.3-8] These thresholds are based on data from European, Australian, and Asian populations, with few data available for establishing appropriate thresholds for US populations.[S4.3-9–S4.3-13] They are provided as a guide but should be interpreted with caution. Higher daytime SBP measurements from ABPM can be associated with an increased risk of CVD and all-cause death independent of clinic-measured BP.[S4.3-14] A meta-analysis of observational studies that included 13 844 individuals suggested nighttime BP is a stronger risk factor for CHD and stroke than either clinic or daytime BP.[S4.3-15]

Methodological issues complicate the interpretation of data from studies that report office and out-of-office BP readings. Definitions and diagnostic methods for identifying white coat hypertension and masked hypertension (see Section 4.4) have not been standardized. The available studies have differed with regard to number of office readings obtained, use of 24-hour ABPM, use of daytime-only ABPM, inclusion of daytime and nighttime BP readings as separate categories, HBPM

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for monitoring out-of-office BP levels, and even the BP thresholds used to define hypertension with ABPM or HBPM readings. In addition, there are few data that address reproducibility of these hypertension profiles over time, with several studies suggesting progression of white coat hypertension and especially of masked hypertension to sustained office-measured hypertension.[S4.3-16–S4.3-22]

4.4. Masked and White Coat Hypertension

Recommendations for Masked and White Coat Hypertension

References that support recommendations are summarized in Online Data Supplements 4, 5, and 6.

COR	LOE	Recommendations
IIa	B-NR	1. In adults with an untreated SBP greater than 130 mm Hg but less than 160 mm Hg or DBP greater than 80 mm Hg but less than 100 mm Hg, it is reasonable to screen for the presence of white coat hypertension by using either daytime ABPM or HBPM before diagnosis of hypertension.[S4.4-1–S4.4-8]
IIa	C-LD	2. In adults with white coat hypertension, periodic monitoring with either ABPM or HBPM is reasonable to detect transition to sustained hypertension (S4.4-2,S4.4-5,S4.4-7).
IIa	C-LD	3. In adults being treated for hypertension with office BP readings not at goal and HBPM readings suggestive of a significant white coat effect, confirmation by ABPM can be useful (S4.4-9,S4.4-10).
IIa	B-NR	4. In adults with untreated office BPs that are consistently between 120 mm Hg and 129 mm Hg for SBP or between 75 mm Hg and 79 mm Hg for DBP, screening for masked hypertension with HBPM (or ABPM) is reasonable (S4.4-3,S4.4-4,S4.4-6,S4.4-8,S4.4-11).
IIb	C-LD	5. In adults on multiple-drug therapies for hypertension and office BPs within 10 mm Hg above goal, it may be reasonable to screen for white coat effect with HBPM (or ABPM) (S4.4-3,S4.4-7,S4.4-12).
IIb	C-EO	6. It may be reasonable to screen for masked uncontrolled hypertension with HBPM in adults being treated for hypertension and office readings at goal, in the presence of target organ damage or increased overall CVD risk.
IIb	C-EO	7. In adults being treated for hypertension with elevated HBPM readings suggestive of masked uncontrolled hypertension, confirmation of the diagnosis by ABPM might be reasonable before intensification of antihypertensive drug treatment.

Synopsis

The availability of noninvasive BP monitoring techniques has resulted in differentiation of hypertension into several clinically useful categories that are based on the place of BP measurement (Table 12).[S4.4-1,S4.4-13,S4.4-14] These include masked hypertension and white coat hypertension, in addition to sustained hypertension. White coat hypertension is characterized by elevated office BP but normal readings when measured outside the office with either ABPM or HBPM. In contrast, masked hypertension is characterized by office readings suggesting normal BP but out-of-office (ABPM/HBPM) readings that are consistently above normal.[S4.4-15] In sustained hypertension, BP readings are elevated in both office and out-of-office settings.

In patients treated for hypertension, both "white coat effect" (higher office BPs than out-of-office BPs) and "masked uncontrolled hypertension" (controlled office BPs but uncontrolled BPs in out-of-office settings) categories have been reported.[S4.4-5,S4.4-15,S4.4-16] The white coat effect (usually considered clinically significant when office SBP/DBPs are >20/10 mm Hg higher than home or ABPM SBP/DBPs) has been implicated in "pseudo-resistant hypertension" (see Section 11.1) and results in an underestimation of office BP control rates.[S4.4-17,S4.4-18] The prevalence of masked hypertension varies from 10% to 26% (mean 13%) in population-based surveys and from 14% to 30% in normotensive clinic populations.[S4.4-6,S4.4-16,S4.4-19–S4.4-21]

The risk of CVD and all-cause mortality in persons with masked hypertension is similar to that noted in those with sustained hypertension and about twice as high as the corresponding risk in their normotensive counterparts.[S4.4-3,S4.4-4,S4.4-6,S4.4-8,S4.4-11] The prevalence of masked hypertension increases with higher office BP readings.[S4.4-20,S4.4-22,S4.4-23]

The prevalence of white coat hypertension is higher with increasing age,[S4.4-24] female versus male sex, nonsmoking versus current smoking status, and routine office measurement of BP by clinician observers versus unattended BP measurements. Many, but not all, studies[S4.4-4,S4.4-6,S4.4-8,S4.4-25,S4.4-26] have identified a minimal increase in risk of CVD complications or all-cause mortality in patients who have white coat hypertension. This has resulted in a recommendation by some panels to screen for white coat hypertension with ABPM (or HBPM) to avoid initiating antihypertensive drug treatment in such individuals.[S4.4-2,S4.4-5,S4.4-27] The white coat effect and masked uncontrolled hypertension appear to follow the risk profiles of their white coat hypertension and masked hypertension counterparts, respectively.[S4.4-3,S4.4-12]

There are no data on the risks and benefits of treating white coat and masked hypertension. Despite these methodological differences, the data are consistent in indicating that

Table 12. BP Patterns Based on Office and Out-of-Office Measurements

	Office/Clinic/ Healthcare Setting	Home/Nonhealthcare/ ABPM Setting
Normotensive	No hypertension	No hypertension
Sustained hypertension	Hypertension	Hypertension
Masked hypertension	No hypertension	Hypertension
White coat hypertension	Hypertension	No hypertension

ABPM indicates ambulatory blood pressure monitoring; and BP, blood pressure.

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masked hypertension and masked uncontrolled hypertension are associated with an increased prevalence of target organ damage and risk of CVD, stroke, and mortality compared with normotensive individuals and those with white coat hypertension.

Figure 1 is an algorithm on the detection of white coat hypertension or masked hypertension in patients not on drug therapy. Figure 2 is an algorithm on detection of white coat effect or masked uncontrolled hypertension in patients on drug therapy. Table 12 is a summary of BP patterns based on office and out-of-office measurements.

Recommendation-Specific Supportive Text

1. White coat hypertension prevalence averages approximately 13% and as high as 35% in some hypertensive populations,[S4.4-1,S4.4-2] and ABPM and HBPM are better predictors of CVD risk due to elevated BP than are office BP measurements, with ABPM being the preferred measurement option. The major clinical relevance of white coat hypertension is that it has typically been associated with a minimal to only slightly increased risk of CVD and all-cause mortality risk.[S4.4-3,S4.4-4,S4.4-7,S4.4-11,S4.4-24] If ABPM resources are not readily available, HBPM provides a reasonable but less desirable alternative to screen for white coat hypertension, although the overlap with ABPM is only 60% to 70% for detection of white coat hypertension.[S4.4-5,S4.4-9,S4.4-27–S4.4-30]
2. The incidence of white coat hypertension converting to sustained hypertension (justifying the addition of antihypertensive drug therapy to lifestyle modification) is 1% to 5% per year by ABPM or HBPM, with a higher incidence of conversion in those with elevated BP, older age, obesity, or black race.[S4.4-2,S4.4-7]
3. The overlap between HBPM and both daytime and 24-hour ABPM in diagnosing white coat hypertension is only 60% to 70%, and the data for prediction of CVD risk are stronger with ABPM than with office measurements.[S4.4-5,S4.4-9,S4.4-27–S4.4-30] Because a diagnosis of white coat hypertension may result in a decision not to treat or intensify treatment in patients with elevated office BP readings, confirmation of BP control by ABPM in addition to HBPM provides added support for this decision.
4. In contrast to white coat hypertension, masked hypertension is associated with a CVD and all-cause mortality risk twice as high as that seen in normotensive individuals, with a risk range similar to that of patients with sustained hypertension.[S4.4-3,S4.4-4,S4.4-6,S4.4-8,S4.4-11,S4.4-31] Therefore, out-of-office readings are reasonable to confirm BP control seen with office readings.
5. The white coat effect has been implicated in office-measured uncontrolled hypertension and pseudo-resistant hypertension, which may result in BP control being underestimated when subsequently assessed by ABPM.[S4.4-17,S4.4-18] The risk of vascular complications in patients with office-measured uncontrolled hypertension with a white coat effect is similar to the risk in those with controlled hypertension.[S4.4-3,S4.4-4,S4.4-7,S4.4-11,S4.4-12] White coat hypertension and white coat effect raise the concern that unnecessary antihypertensive drug therapy may be initiated or intensified. Because a diagnosis of white coat hypertension or white coat effect would result in a decision to not treat elevated office BP readings, confirmation of BP control by HBPM (or ABPM) provides more definitive support for the decision not to initiate antihypertensive drug therapy or accelerate treatment.
6. Analogous to masked hypertension in untreated patients, masked uncontrolled hypertension is defined in treated patients with hypertension by office readings suggesting adequate BP control but out-of-office readings (HBPM) that remain consistently above goal.[S4.4-3,S4.4-15,S4.4-16,S4.4-32,S4.4-33] The CVD risk profile for masked uncontrolled hypertension appears to follow the risk profile for masked hypertension.[S4.4-3,S4.4-12,S4.4-34] Although the evidence is consistent in identifying the increased risk of masked uncontrolled hypertension, evidence is lacking on whether the treatment of masked hypertension or masked uncontrolled hypertension reduces clinical outcomes. A suggestion for assessing CVD risk is provided in Section 8.
7. Although both ABPM and HBPM are better predictors of CVD risk than are office BP readings, ABPM confirmation of elevated BP by HBPM might be reasonable because of the more extensive documentation of CVD risk with ABPM. However, unlike the documentation of a significant white coat effect to justify the decision to not treat an elevated clinic BP, it is not mandatory to confirm masked uncontrolled hypertension determined by HBPM.

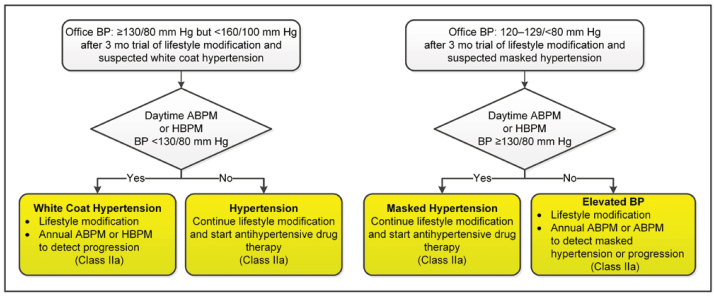

Figure 1. Detection of white coat hypertension or masked hypertension in patients not on drug therapy. Colors correspond to Class of Recommendation in Table 1. ABPM indicates ambulatory blood pressure monitoring; BP, blood pressure; and HBPM, home blood pressure monitoring.

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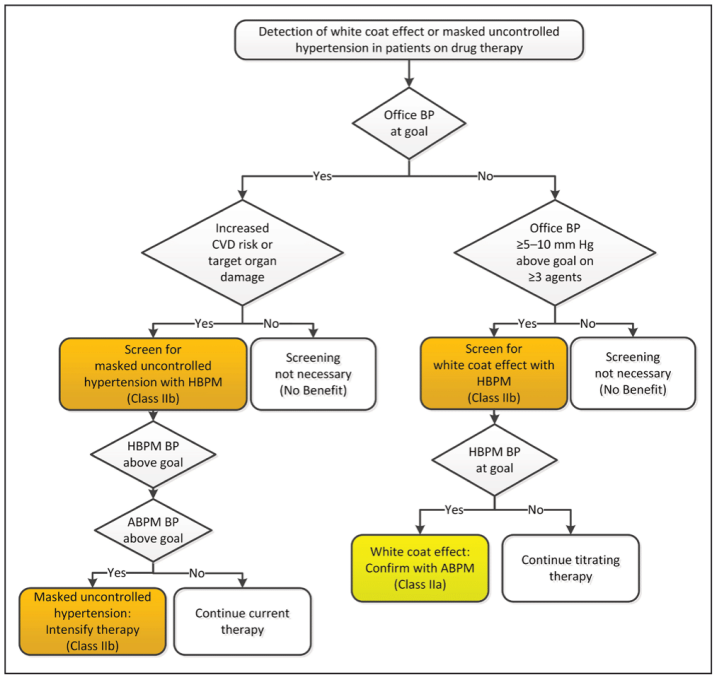

Figure 2. Detection of white coat effect or masked uncontrolled hypertension in patients on drug therapy. Colors correspond to Class of Recommendation in Table 1. See Section 8 for treatment options. ABPM indicates ambulatory blood pressure monitoring; BP, blood pressure; CVD, cardiovascular disease; and HBPM, home blood pressure monitoring.

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5. Causes of Hypertension

5.1. Genetic Predisposition

Hypertension is a complex polygenic disorder in which many genes or gene combinations influence BP.[S5.1-1,S5.1-2] Although several monogenic forms of hypertension have been identified, such as glucocorticoid-remediable aldosteronism, Liddle's syndrome, Gordon's syndrome, and others in which single-gene mutations fully explain the pathophysiology of hypertension, these disorders are rare.[S5.1-3] The current tabulation of known genetic variants contributing to BP and hypertension includes more than 25 rare mutations and 120 single-nucleotide polymorphisms.[S5.1-3,S5.1-4] However, even with the discovery of multiple single-nucleotide polymorphisms influencing control of BP since completion of the Human Genome Project in 2003, the associated variants have only small effects. Indeed, at present, the collective effect of all BP loci identified through genome-wide association studies accounts for only about 3.5% of BP variability.[S5.1-4] The presence of a high number of small-effect alleles associated with higher BP results in a more rapid increase in BP with age.[S5.1-5] Future studies will need to better elucidate genetic expression, epigenetic effects, transcriptomics, and proteomics that link genotypes with underlying pathophysiological mechanisms.

5.2. Environmental Risk Factors

Various environmental exposures, including components of diet, physical activity, and alcohol consumption, influence BP. Many dietary components have been associated with high BP.[S5.2-1,S5.2-2] Some of the diet-related factors associated with high BP include overweight and obesity, excess intake of sodium, and insufficient intake of potassium, calcium, magnesium, protein (especially from vegetables), fiber, and fish fats. Poor diet, physical inactivity, and excess intake of alcohol, alone or in combination, are the underlying cause of a large proportion of hypertension. Gut microbiota have also been linked to hypertension, especially in experimental animals.[S5.2-3] Some of the best-proven environmental relationships with high BP are briefly reviewed below, and nonpharmacological interventions to lower BP are discussed in Section 6.2.

5.2.1. Overweight and Obesity

Insurance industry actuarial reports have identified a striking relationship between body weight and high BP[S5.2.1-1] and a direct relationship between overweight/obesity and hypertension.[S5.2.1-2] Epidemiological studies, including the Framingham Heart Study[S5.2.1-3] and the Nurses' Health Study,[S5.2.1-4] have consistently identified a direct relationship between body mass index and BP that is continuous and almost linear, with no evidence of a threshold.[S5.2.1-5,S5.2.1-6] The relationship with BP is even stronger for waist-to-hip ratio and computed tomographic measures of central fat distribution.[S5.2.1-7] Attributable risk estimates from the Nurses' Health Study suggest that obesity may be responsible for about 40% of hypertension, and in the Framingham Offspring Study, the corresponding estimates were even

higher (78% in men and 65% in women).[S5.2.1-8,S5.2.1-9] The relationship between obesity at a young age and change in obesity status over time is strongly related to future risk of hypertension. In combined data from 4 longitudinal studies begun in adolescence with repeat examination in young adulthood to early middle age, being obese continuously or acquiring obesity was associated with a relative risk of 2.7 for developing hypertension. Becoming normal weight reduced the risk of developing hypertension to a level similar to those who had never been obese.[S5.2.1-10]

5.2.2. Sodium Intake

Sodium intake is positively associated with BP in migrant,[S5.2.2-11] cross-sectional,[S5.2.2-12–S5.2.2-14] and prospective cohort studies[S5.2.2-15] and accounts for much of the age-related increase in BP.[S5.2.2-11,S5.2.2-16] In addition to the well-accepted and important relationship of dietary sodium with BP, excessive consumption of sodium is independently associated with an increased risk of stroke,[S5.2.2-17,S5.2.2-18] CVD,[S5.2.2-19] and other adverse outcomes.[S5.2.2-20] Certain groups with various demographic, physiological, and genetic characteristics tend to be particularly sensitive to the effects of dietary sodium on BP.[S5.2.2-21–S5.2.2-23] Salt sensitivity is a quantitative trait in which an increase in sodium load disproportionately increases BP.[S5.2.2-21,S5.2.2-24] Salt sensitivity is especially common in blacks, older adults, and those with a higher level of BP or comorbidities such as CKD, DM, or the metabolic syndrome.[S5.2.2-25] In aggregate, these groups constitute more than half of all US adults.[S5.2.2-26] Salt sensitivity may be a marker for increased CVD and all-cause mortality risk independently of BP,[S5.2.2-27,S5.2.2-28] and the trait has been demonstrated to be reproducible.[S5.2.2-29] Current techniques for recognition of salt sensitivity are impractical in routine clinical practice, so salt sensitivity is best considered as a group characteristic.

5.2.3. Potassium

Potassium intake is inversely related to BP in migrant,[S5.2.3-30] cross-sectional,[S5.2.2-13,S5.2.2-16,S5.2.3-31,S5.2.3-32] and prospective cohort[S5.2.3-33] studies. It is also inversely related to stroke.[S5.2.3-34–S5.2.3-36] A higher level of potassium seems to blunt the effect of sodium on BP,[S5.2.3-37] with a lower sodium–potassium ratio being associated with a lower level of BP than that noted for corresponding levels of sodium or potassium on their own.[S5.2.3-38] Likewise, epidemiological studies suggest that a lower sodium–potassium ratio may result in a reduced risk of CVD as compared with the pattern for corresponding levels of either cation on its own.[S5.2.3-39]

5.2.4. Physical Fitness

Epidemiological studies have demonstrated an inverse relationship between physical activity and physical fitness and level of BP and hypertension.[S5.2.4-40] Even modest levels of physical activity have been associated with a decrease in the risk of incident hypertension.[S5.2.4-41] In several observational studies, the relationship between physical activity and BP has been most apparent in white men.[S5.2.4-40] With the advent of electronic activity trackers and ABPM, it has become increasingly feasible to conduct studies that relate physical activity and BP.[S5.2.4-42] Physical fitness, measured objectively by graded exercise testing, attenuates the rise of BP with age and prevents the development of hypertension. In the CARDIA (Coronary Artery Risk Development in Young Adults) study,[S5.2.4-43] physical fitness measured at 18 to 30 years of age in the upper 2 deciles of an otherwise healthy population was associated with one third the risk of developing hypertension 15 years later, and one half the risk after adjustment for body mass index, as compared with the lowest quintile. Change in fitness assessed 7 years later further modified risk.[S5.2.4-43] In a cohort of men 20 to 90 years of age who were followed longitudinally for 3 to 28 years, higher physical fitness decreased the rate of rise in SBP over time and delayed the time to onset of hypertension.[S5.2.4-44]

5.2.5. Alcohol

The presence of a direct relationship between alcohol consumption and BP was first reported in 1915[S5.2.5-45] and has been repeatedly identified in contemporary cross-sectional and prospective cohort studies.[S5.2.5-46] Estimates of the contribution of alcohol consumption to population incidence and prevalence of hypertension vary according to level of intake. In the United States, it seems likely that alcohol may account for close to 10% of the population burden of hypertension (higher in men than in women). In contrast to its detrimental effect on BP, alcohol intake is associated with

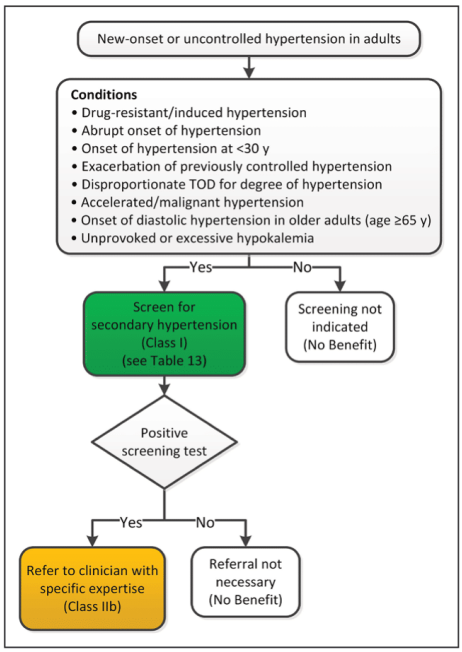

Figure 3. Screening for secondary hypertension. Colors correspond to Class of Recommendation in Table 1. TOD indicates target organ damage (eg, cerebrovascular disease, hypertensive retinopathy, left ventricular hypertrophy, left ventricular dysfunction, heart failure, coronary artery disease, chronic kidney disease, albuminuria, peripheral artery disease).

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Table 13. Causes of Secondary Hypertension With Clinical Indications and Diagnostic Screening Tests

	Prevalence	Clinical Indications	Physical Examination	Screening Tests	Additional/Confirmatory Tests
Common causes					
Renal parenchymal disease[S5.4-1,S5.4-3]	1%–2%	Urinary tract infections; obstruction, hematuria; urinary frequency and nocturia; analgesic abuse; family history of polycystic kidney disease; elevated serum creatinine; abnormal urinalysis	Abdominal mass (polycystic kidney disease); skin pallor	Renal ultrasound	Tests to evaluate cause of renal disease
Renovascular disease[S5.4-4]	5%–34%*	Resistant hypertension; hypertension of abrupt onset or worsening or increasingly difficult to control; flash pulmonary edema (atherosclerotic); early-onset hypertension, especially in women (fibromuscular hyperplasia)	Abdominal systolic-diastolic bruit; bruits over other arteries (carotid – atherosclerotic or fibromuscular dysplasia), femoral	Renal Duplex Doppler ultrasound; MRA; abdominal CT	Bilateral selective renal intra-arterial angiography
Primary aldosteronism[S5.4-5,S5.4-6]	8%–20%†	Resistant hypertension; hypertension with hypokalemia (spontaneous or diuretic induced); hypertension and muscle cramps or weakness; hypertension and incidentally discovered adrenal mass; hypertension and obstructive sleep apnea; hypertension and family history of early-onset hypertension or stroke	Arrhythmias (with hypokalemia); especially atrial fibrillation	Plasma aldosterone/ renin ratio under standardized conditions (correction of hypokalemia and withdrawal of aldosterone antagonists for 4–6 wk)	Oral sodium loading test (with 24-h urine aldosterone) or IV saline infusion test with plasma aldosterone at 4 h of infusion Adrenal CT scan, adrenal vein sampling.
Obstructive sleep apnea[S5.4-7]‡	25%–50%	Resistant hypertension; snoring; fitful sleep; breathing pauses during sleep; daytime sleepiness	Obesity, Mallampati class III–IV; loss of normal nocturnal BP fall	Berlin Questionnaire;[S5.4-8] Epworth Sleepiness Score;[S5.4-9] overnight oximetry	Polysomnography
Drug or alcohol induced[S5.4-10]§	2%–4%	Sodium-containing antacids; caffeine; nicotine (smoking); alcohol; NSAIDs; oral contraceptives; cyclosporine or tacrolimus; sympathomimetics (decongestants, anorectics); cocaine, amphetamines and other illicit drugs; neuropsychiatric agents; erythropoiesis-stimulating agents; clonidine withdrawal; herbal agents (Ma Huang, ephedra)	Fine tremor, tachycardia, sweating (cocaine, ephedrine, MAO inhibitors); acute abdominal pain (cocaine)	Urinary drug screen (illicit drugs)	Response to withdrawal of suspected agent
Uncommon causes					
Pheochromocytoma/paraganglioma[S5.4-11]	0.1%–0.6%	Resistant hypertension; paroxysmal hypertension or crisis superimposed on sustained hypertension; "spells," BP lability, headache, sweating, palpitations, pallor; positive family history of pheochromocytoma/ paraganglioma; adrenal incidentaloma	Skin stigmata of neurofibromatosis (café-au-lait spots; neurofibromas); Orthostatic hypotension	24-h urinary fractionated metanephrines or plasma metanephrines under standard conditions (supine position with indwelling IV cannula)	CT or MRI scan of abdomen/pelvis
Cushing's syndrome[S5.4-12]	<0.1%	Rapid weight gain, especially with central distribution; proximal muscle weakness; depression; hyperglycemia	Central obesity, "moon" face, dorsal and supraclavicular fat pads, wide (1-cm) violaceous striae, hirsutism	Overnight 1-mg dexamethasone suppression test	24-h urinary free cortisol excretion (preferably multiple); midnight salivary cortisol

(*Continued*)

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Table 13. Continued

	Prevalence	Clinical Indications	Physical Examination	Screening Tests	Additional/Confirmatory Tests
Uncommon causes (Continued)					
Hypothyroidism[S5.4-10]	<1%	Dry skin; cold intolerance; constipation; hoarseness; weight gain	Delayed ankle reflex; periorbital puffiness; coarse skin; cold skin; slow movement; goiter	Thyroid-stimulating hormone; free thyroxine	None
Hyperthyroidism[S5.4-10]	<1%	Warm, moist skin; heat intolerance; nervousness; tremulousness; insomnia; weight loss; diarrhea; proximal muscle weakness	Lid lag; fine tremor of the outstretched hands; warm, moist skin	Thyroid-stimulating hormone; free thyroxine	Radioactive iodine uptake and scan
Aortic coarctation (undiagnosed or repaired)[S5.4-13]	0.1%	Young patient with hypertension (<30 y of age)	BP higher in upper extremities than in lower extremities; absent femoral pulses; continuous murmur over patient's back, chest, or abdominal bruit; left thoracotomy scar (postoperative)	Echocardiogram	Thoracic and abdominal CT angiogram or MRA
Primary hyperparathyroidism[S5.4-14]	Rare	Hypercalcemia	Usually none	Serum calcium	Serum parathyroid hormone
Congenital adrenal hyperplasia[S5.4-15]	Rare	Hypertension and hypokalemia; virilization (11-beta-hydroxylase deficiency [11-beta-OH]); incomplete masculinization in males and primary amenorrhea in females (17-alpha-hydroxylase deficiency [17-alpha-OH])	Signs of virilization (11-beta-OH) or incomplete masculinization (17-alpha-OH)	Hypertension and hypokalemia with low or normal aldosterone and renin	11-beta-OH: elevated deoxycorticosterone (DOC), 11-deoxycortisol, and androgens17-alpha-OH; decreased androgens and estrogen; elevated deoxycorticosterone and corticosterone
Mineralocorticoid excess syndromes other than primary aldosteronism[S5.4-15]	Rare	Early-onset hypertension; resistant hypertension; hypokalemia or hyperkalemia	Arrhythmias (with hypokalemia)	Low aldosterone and renin	Urinary cortisol metabolites; genetic testing
Acromegaly[S5.4-16]	Rare	Acral features, enlarging shoe, glove, or hat size; headache, visual disturbances; diabetes mellitus	Acral features; large hands and feet; frontal bossing	Serum growth hormone ≥1 ng/mL during oral glucose load	Elevated age- and sex-matched IGF-1 level; MRI scan of the pituitary

*Depending on the clinical situation (hypertension alone, 5%; hypertension starting dialysis, 22%; hypertension and peripheral vascular disease, 28%; hypertension in the elderly with congestive heart failure, 34%).

†8% in general population with hypertension; up to 20% in patients with resistant hypertension.

‡Although obstructive sleep apnea is listed as a cause of secondary hypertension, RCTs on the effects of continuous positive airway pressure on lowering BP in patients with hypertension have produced mixed results (see Section 5.4.4 for details).

§For a list of frequently used drugs causing hypertension and accompanying evidence, see Table 14.

BP indicates blood pressure; CT, computed tomography; DOC, 11-deoxycorticosterone; IGF-1, insulin-like growth factor-1; IV, intravenous; MAO, monamine oxidase; MRI, magnetic resonance imaging; MRA, magnetic resonance arteriography; NSAIDs, nonsteroidal anti-inflammatory drugs; OH, hydroxylase; and RCT, randomized clinical trial.

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a higher level of high-density lipoprotein cholesterol and, within modest ranges of intake, a lower level of CHD than that associated with abstinence.[S5.2.3-35]

5.3. Childhood Risk Factors and BP Tracking

BP distribution in the general population increases with age. Multiple longitudinal studies have investigated the relationship of childhood BP to adult BP. A meta-analysis of 50 such studies showed correlation coefficients of about 0.38 for SBP and 0.28 for DBP, with BPs in the upper range of the pediatric distribution (particularly BPs obtained in adolescence) predicting hypertension in adulthood.[S5.3-1] Several factors, including genetic factors and development of obesity, increase the likelihood that a high childhood BP will lead to future hypertension.[S5.3-2] Premature birth is associated with a 4–mm Hg higher SBP and a 3–mm Hg higher DBP in adulthood,

with somewhat larger effects in women than in men.[S5.3-3] Low birth weight from other causes also contributes to higher BP in later life.[S5.3-4]

5.4. Secondary Forms of Hypertension

Recommendations for Secondary Forms of Hypertension		
COR	LOE	Recommendations
I	C-EO	1. Screening for specific form(s) of secondary hypertension is recommended when the clinical indications and physical examination findings listed in Table 13 are present or in adults with resistant hypertension.
IIb	C-EO	2. If an adult with sustained hypertension screens positive for a form of secondary hypertension, referral to a physician with expertise in that form of hypertension may be reasonable for diagnostic confirmation and treatment.

Synopsis

A specific, remediable cause of hypertension can be identified in approximately 10% of adult patients with hypertension.[S5.4-1] If a cause can be correctly diagnosed and treated, patients with secondary hypertension can achieve a cure or experience a marked improvement in BP control, with reduction in CVD risk. All new patients with hypertension should be screened with a history, physical examination, and laboratory investigations, as recommended in Section 7, before initiation of treatment.

Secondary hypertension can underlie severe elevation of BP, pharmacologically resistant hypertension, sudden onset of hypertension, increased BP in patients with hypertension previously controlled on drug therapy, onset of diastolic hypertension in older adults, and target organ damage disproportionate to the duration or severity of the hypertension. Although secondary hypertension should be suspected in younger patients (<30 years of age) with elevated BP, it is not uncommon for primary hypertension to manifest at a younger age, especially in blacks,[S5.4-2] and some forms of secondary hypertension, such as renovascular disease, are more common at older age. Many of the causes of secondary hypertension are strongly associated with clinical findings or groups of findings that suggest a specific disorder.

Figure 3 is an algorithm on screening for secondary hypertension. Table 13 is a detailed list of clinical indications and diagnostic screening tests for secondary hypertension, and Table 14 is a list of drugs that can induce secondary hypertension.

Recommendation-Specific Supportive Text

1. The causes of secondary hypertension and recommended screening tests are provided in Table 13, and drugs that can induce secondary hypertension are provided in Table 14.
2. Diagnosis of many of these disorders requires a complex set of measurements, specialized technical expertise, and/or experience in data interpretation. Similarly, specific treatment often requires a level of technical training and experience.

5.4.1. Drugs and Other Substances With Potential to Impair BP Control

Numerous substances, including prescription medications, over-the-counter medications, herbals, and food substances, may affect BP (Table 14).[S5.4.1-1–S5.4.1-6] Changes in BP that occur because of drugs and other agents have been associated with the development of hypertension, worsening control in a patient who already has hypertension, or attenuation of the BP-lowering effects of antihypertensive therapy. A change in BP may also result from drug–drug or drug–food interactions.[S5.4.1-2,S5.4.1-4] In the clinical assessment of hypertension, a careful history should be taken with regard to substances that may impair BP control, with close attention paid to not only prescription medications, but also over-the-counter substances, illicit drugs, and herbal products. When feasible, drugs associated with increased BP should be reduced or discontinued, and alternative agents should be used.

5.4.2. Primary Aldosteronism

Recommendations for Primary Aldosteronism		
COR	LOE	Recommendations
I	C-EO	1. In adults with hypertension, screening for primary aldosteronism is recommended in the presence of any of the following concurrent conditions: resistant hypertension, hypokalemia (spontaneous or substantial, if diuretic induced), incidentally discovered adrenal mass, family history of early-onset hypertension, or stroke at a young age (<40 years).
I	C-LD	2. Use of the plasma aldosterone: renin activity ratio is recommended when adults are screened for primary aldosteronism.[S5.4.2-1]
I	C-EO	3. In adults with hypertension and a positive screening test for primary aldosteronism, referral to a hypertension specialist or endocrinologist is recommended for further evaluation and treatment.

Synopsis

Primary aldosteronism is defined as a group of disorders in which aldosterone production is inappropriately high for sodium status, is relatively autonomous of the major regulators of secretion (angiotensin II and potassium), and cannot be suppressed with sodium loading.[S5.4.2-2,S5.4.2-3] The increased production of aldosterone induces hypertension; cardiovascular and kidney damage; sodium retention; suppressed plasma renin activity; and increased potassium excretion, which, if prolonged and severe, may cause hypokalemia. However, hypokalemia is absent in the majority of cases and has a low negative predictive value for the diagnosis of primary aldosteronism.[S5.4.2-4] In about 50% of the patients, primary aldosteronism is due to increased unilateral aldosterone production (usually aldosterone-producing adenoma or, rarely, unilateral adrenal hyperplasia); in the remaining 50%, primary aldosteronism is due to bilateral adrenal hyperplasia (idiopathic hyperaldosteronism).[S5.4.2-2,S5.4.2-3]

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Table 14. Frequently Used Medications and Other Substances That May Cause Elevated BP*

Agent	Possible Management Strategy
Alcohol	Limit alcohol to ≤1 drink daily for women and ≤2 drinks for men[S5.4.1-7]
Amphetamines (eg, amphetamine, methylphenidate dexmethylphenidate, dextroamphetamine)	Discontinue or decrease dose[S5.4.1-8] Consider behavioral therapies for ADHD[S5.4.1-9]
Antidepressants (eg, MAOIs, SNRIs, TCAs)	Consider alternative agents (eg, SSRIs) depending on indication Avoid tyramine-containing foods with MAOIs
Atypical antipsychotics (eg, clozapine, olanzapine)	Discontinue or limit use when possible Consider behavior therapy where appropriate Recommend lifestyle modification (see Section 6.2) Consider alternative agents associated with lower risk of weight gain, diabetes mellitus, and dyslipidemia (eg, aripiprazole, ziprasidone)[S5.4.1-10,S5.4.1-11]
Caffeine	Generally limit caffeine intake to <300 mg/d Avoid use in patients with uncontrolled hypertension Coffee use in patients with hypertension is associated with acute increases in BP; long-term use is not associated with increased BP or CVD[S5.4.1-12]
Decongestants (eg, phenylephrine, pseudoephedrine)	Use for shortest duration possible, and avoid in severe or uncontrolled hypertension Consider alternative therapies (eg, nasal saline, intranasal corticosteroids, antihistamines) as appropriate
Herbal supplements (eg, Ma Huang [ephedra], St. John's wort [with MAO inhibitors, yohimbine])	Avoid use
Immunosuppressants (eg, cyclosporine)	Consider converting to tacrolimus, which may be associated with fewer effects on BP[S5.4.1-13–S5.4.1-15]
Oral contraceptives	Use low-dose (eg, 20–30 mcg ethinyl estradiol) agents[S5.4.1-16] or a progestin-only form of contraception, or consider alternative forms of birth control where appropriate (eg, barrier, abstinence, IUD) Avoid use in women with uncontrolled hypertension[S5.4.1-16]
NSAIDs	Avoid systemic NSAIDs when possible Consider alternative analgesics (eg, acetaminophen, tramadol, topical NSAIDs), depending on indication and risk
Recreational drugs (eg, "bath salts" [MDPV], cocaine, methamphetamine, etc.)	Discontinue or avoid use
Systemic corticosteroids (eg, dexamethasone, fludrocortisone, methylprednisolone, prednisone, prednisolone)	Avoid or limit use when possible Consider alternative modes of administration (eg, inhaled, topical) when feasible
Angiogenesis inhibitor (eg, bevacizumab) and tyrosine kinase inhibitors (eg, sunitinib, sorafenib)	Initiate or intensify antihypertensive therapy

*List is not all inclusive.

ADHD indicates attention-deficit/hyperactivity disorder; BP, blood pressure; CVD, cardiovascular disease; IUD, intra-uterine device; MAOI, monoamine-oxidase inhibitors; MDPV, methylenedioxypyrovalerone; NSAIDs, nonsteroidal anti-inflammatory drugs; SNRI, serotonin norepinephrine reuptake inhibitor; SSRI, selective serotonin reuptake inhibitor; and TCA, tricyclic antidepressant.

Recommendation-Specific Supportive Text

1. Primary aldosteronism is one of the most frequent disorders (occurring in 5% to 10% of patients with hypertension and 20% of patients with resistant hypertension) that causes secondary hypertension.[S5.4.2-5,S5.4.2-6] The toxic tissue effects of aldosterone induce greater target organ damage in primary aldosteronism than in primary hypertension. Patients with primary aldosteronism have a 3.7-fold increase in HF, a 4.2-fold increase in stroke, a 6.5-fold increase in MI, a 12.1-fold increase in atrial fibrillation (AF), increased left ventricular hypertrophy (LVH) and diastolic dysfunction, increased stiffness of large arteries, widespread tissue fibrosis, increased remodeling of resistance vessels, and increased kidney damage as compared with patients with primary hypertension matched for BP level.[S5.4.2-6–S5.4.2-8] Because the deleterious effects of aldosterone overproduction are often reversible with unilateral laparoscopic adrenalectomy or treatment with mineralocorticoid receptor antagonists (ie, spironolactone or eplerenone), screening of patients with hypertension at increased risk of primary aldosteronism is beneficial.[S5.4.2-2,S5.4.2-3] These include hypertensive patients with adrenal "incidentaloma," an incidentally discovered adrenal lesion on a computed tomography or magnetic resonance imaging (MRI) scan performed for other purposes. Patients with hypertension and a history of early onset hypertension and/or cerebrovascular accident at a young age may have primary aldosteronism due to glucocorticoid-remediable aldosteronism (familial hyperaldosteronism type-1) and therefore warrant screening.[S5.4.2-2,S5.4.2-3]
2. The aldosterone:renin activity ratio is currently the most accurate and reliable means of screening for primary

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aldosteronism.[S5.4.2-1] The most commonly used cutoff value is 30 when plasma aldosterone concentration is reported in nanograms per deciliter (ng/dL) and plasma renin activity in nanograms per milliliter per hour (ng/mL/h).[S5.4.2-3] Because the aldosterone:renin activity ratio can be influenced by the presence of very low renin levels, the plasma aldosterone concentration should be at least 10 ng/dL to interpret the test as positive.[S5.4.2-3] Patients should have unrestricted salt intake, serum potassium in the normal range, and mineralocorticoid receptor antagonists (eg, spironolactone or eplerenone) withdrawn for at least 4 weeks before testing.[S5.4.2-2,S5.4.2-3]

3. The diagnosis of primary aldosteronism generally requires a confirmatory test (intravenous saline suppression test or oral salt-loading test).[S5.4.2-2,S5.4.2-3] If the diagnosis of primary aldosteronism is confirmed (and the patient agrees that surgery would be desirable), the patient is referred for an adrenal venous sampling procedure to determine whether the increased aldosterone production is unilateral or bilateral in origin. If unilateral aldosterone production is documented on adrenal venous sampling, the patient is referred for unilateral laparoscopic adrenalectomy, which improves BP in virtually 100% of patients and results in a complete cure of hypertension in about 50%.[S5.4.2-2,S5.4.2-3] If the patient has bilaterally increased aldosterone secretion on adrenal venous sampling or has a unilateral source of excess aldosterone production but cannot undergo surgery, the patient is treated with spironolactone or eplerenone as agent of choice.[S5.4.2-2,S5.4.2-3] Both adrenalectomy and medical ktherapy are effective in lowering BP and reversing LVH. Treating primary aldosteronism, either by mineralocorticoid receptor antagonists or unilateral adrenalectomy (if indicated), resolves hypokalemia, lowers BP, reduces the number of antihypertensive medications required, and improves parameters of impaired cardiac and kidney function.[S5.4.2-9,S5.4.2-10]

5.4.3. Renal Artery Stenosis

Recommendations for Renal Artery Stenosis

References that support recommendations are summarized in Online Data Supplements 7 and 24.

COR	LOE	Recommendations
I	A	1. Medical therapy is recommended for adults with atherosclerotic renal artery stenosis.[S5.4.3-1,S5.4.3-2]
IIb	C-EO	2. In adults with renal artery stenosis for whom medical management has failed (refractory hypertension, worsening renal function, and/or intractable HF) and those with nonatherosclerotic disease, including fibromuscular dysplasia, it may be reasonable to refer the patient for consideration of revascularization (percutaneous renal artery angioplasty and/or stent placement).

Synopsis

Renal artery stenosis refers to a narrowing of the renal artery that can result in a restriction of blood flow. Atherosclerotic disease (90%) is by far the most common cause of renal artery stenosis, whereas nonatherosclerotic disease (of which fibromuscular dysplasia is the most common) is much less prevalent and tends to occur in younger, healthier patients.[S5.4.3-3] Renal artery stenosis is a common form of secondary hypertension. Relieving ischemia and the ensuing postischemic release of renin by surgical renal artery reconstruction is an invasive strategy with a postoperative mortality as high as 13%.[S5.4.3-4] With the advent of endovascular procedures to restore blood flow, several trials were designed to test the efficacy of these procedures against medical therapy, but they suggested no benefit over medical therapy alone.[S5.4.3-1,S5.4.3-2]

Recommendation-Specific Supportive Text

1. Atherosclerotic disease in the renal arteries represents systemic disease and higher risk of both renal failure and cardiovascular morbidity and mortality. No RCT to date has demonstrated a clinical advantage of renal artery revascularization (with either angioplasty or stenting) over medical therapy.[S5.4.3-2] On the basis of the CORAL (Cardiovascular Outcomes in Renal Atherosclerotic Lesions) trial, the recommended medical approach encompasses optimal management of hypertension with an antihypertensive regimen that includes a renin-angiotensin system (RAS) blocker, in addition to low-density lipoprotein cholesterol reduction with a high-intensity statin, smoking cessation, hemoglobin A1c reduction in patients with DM, and antiplatelet therapy.[S5.4.3-1]
2. Revascularization may be considered for those who do not respond to medical therapy and for those who have nonatherosclerotic disease (eg, Takayasu arteritis in Asian populations, fibromuscular dysplasia in other populations). Fibromuscular dysplasia occurs over the lifespan of women (mean: 53 years of age) with almost equal frequency in the renal and carotid circulations.[S5.4.3-3] Percutaneous transluminal angioplasty alone (without stenting) can improve BP control and even normalize BP, especially in patients with recent onset of hypertension or resistant hypertension.[S5.4.3-5]

5.4.4. Obstructive Sleep Apnea

Recommendation for Obstructive Sleep Apnea

References that support the recommendation are summarized in Online Data Supplement 8.

COR	LOE	Recommendation
IIb	B-R	1. In adults with hypertension and obstructive sleep apnea, the effectiveness of continuous positive airway pressure (CPAP) to reduce BP is not well established.[S5.4.4-1–S5.4.4-5]

Synopsis

Obstructive sleep apnea is a common chronic condition characterized by recurrent collapse of upper airways during sleep, inducing intermittent episodes of apnea/hypopnea, hypoxemia, and sleep disruption.[S5.4.4-6] Obstructive sleep apnea is a risk factor for several CVDs, including hypertension, coronary and cerebrovascular diseases, HF, and AF.[S5.4.4-6–S5.4.4-9] Observational studies have shown that the presence of obstructive sleep apnea is associated with increased risk of incident hypertension.[S5.4.4-10,S5.4.4-11] Obstructive sleep apnea is

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highly prevalent in adults with resistant hypertension (≥80%),[S5.4.4-12,S5.4.4-13] and it has been hypothesized that treatment with CPAP may have more pronounced effects on BP reduction in resistant hypertension.[S5.4.4-6]

Recommendation-Specific Supportive Text

1. CPAP is an efficacious treatment for improving obstructive sleep apnea. However, studies of the effects of CPAP on BP have demonstrated only small effects on BP (eg, 2– to 3–mm Hg reductions), with results dependent on patient compliance with CPAP use, severity of obstructive sleep apnea, and presence of daytime sleepiness in study participants.[S5.4.4-1–S5.4.4-5] Although many RCTs have been reported that address the effects of CPAP on BP in obstructive sleep apnea, most of the patients studied did not have documented hypertension, and the studies were too small and the follow-up period too short to allow for adequate evaluation. In addition, a well-designed RCT demonstrated that CPAP plus usual care, compared with usual care alone, did not prevent cardiovascular events in patients with moderate–severe obstructive sleep apnea and established CVD.[S5.4.4-14]

6. Nonpharmacological Interventions

Correcting the dietary aberrations, physical inactivity, and excessive consumption of alcohol that cause high BP is a fundamentally important approach to prevention and management of high BP, either on their own or in combination with pharmacological therapy. Prevention of hypertension and treatment of established hypertension are complementary approaches to reducing CVD risk in the population, but prevention of hypertension provides the optimal means of reducing risk and avoiding the harmful consequences of hypertension.[S6-1–S6-3] Nonpharmacological therapy alone is especially useful for prevention of hypertension, including in adults with elevated BP, and for management of high BP in adults with milder forms of hypertension.[S6-4,S6-5]

6.1. Strategies

Nonpharmacological interventions can be accomplished by means of behavioral strategies aimed at lifestyle change, prescription of dietary supplements, or implementation of kitchen-based interventions that directly modify elements of the diet. At a societal level, policy changes can enhance the availability of healthy foods and facilitate physical activity. The goal can be to modestly reduce BP in the general population or to undertake more intensive targeted lowering of BP in adults with hypertension or at high risk of developing hypertension.[S6.1-1] The intent of the general population approach is to achieve a small downward shift in the general population distribution of BP, which would be expected to result in substantial health benefits.[S6.1-2] The targeted approach focuses on BP reduction in adults at greatest risk of developing BP-related CVD, including individuals with hypertension, as well as those at increased risk of developing hypertension, especially blacks and adults who are overweight, consume excessive amounts of dietary sodium, have a high intake of alcohol, or are physically inactive. The targeted approach tends to be intensive, with a more ambitious goal for BP reduction. Both approaches are complementary and mutually reinforcing, and modeling studies suggest they are likely to provide similar public health benefit.[S6.1-3,S6.1-4] However, as the precision of risk prediction tools increases, targeted prevention strategies that focus on high-risk individuals seem to become more efficient than population-based strategies.[S6.1-5]

6.2. Nonpharmacological Interventions

Recommendations for Nonpharmacological Interventions

References that support recommendations are summarized in Online Data Supplements 9-21.

COR	LOE	Recommendations
I	A	1. Weight loss is recommended to reduce BP in adults with elevated BP or hypertension who are overweight or obese.[S6.2-1–S6.2-4]
I	A	2. A heart-healthy diet, such as the DASH (Dietary Approaches to Stop Hypertension) diet, that facilitates achieving a desirable weight is recommended for adults with elevated BP or hypertension.[S6.2-5–S6.2-7]
I	A	3. Sodium reduction is recommended for adults with elevated BP or hypertension.[S6.2-8–S6.2-12]
I	A	4. Potassium supplementation, preferably in dietary modification, is recommended for adults with elevated BP or hypertension, unless contraindicated by the presence of CKD or use of drugs that reduce potassium excretion.[S6.2-13–S6.2-17]
I	A	5. Increased physical activity with a structured exercise program is recommended for adults with elevated BP or hypertension.[S6.2-3,S6.2-4,S6.2-12,S6.2-18–S6.2-22]
I	A	6. Adult men and women with elevated BP or hypertension who currently consume alcohol should be advised to drink no more than 2 and 1 standard drinks* per day, respectively[S6.2-23–S6.2-28]

*In the United States, 1 "standard" drink contains roughly 14 g of pure alcohol, which is typically found in 12 oz of regular beer (usually about 5% alcohol), 5 oz of wine (usually about 12% alcohol), and 1.5 oz of distilled spirits (usually about 40% alcohol).[S6.2-29]

Synopsis

Nonpharmacological interventions are effective in lowering BP, with the most important interventions being weight loss,[S6.2-1] the DASH (Dietary Approaches to Stop Hypertension) diet,[S6.2-5–S6.2-7,S6.2-30] sodium reduction,[S6.2-8–S6.2-12] potassium supplementation,[S6.2-13,S6.2-17] increased physical activity,[S6.2-18–S6.2-22,S6.2-31] and a reduction in alcohol consumption.[S6.2-23,S6.2-24] Various other nonpharmacological interventions have been reported to lower BP, but the extent and/or quality of the supporting clinical trial experience is less persuasive. Such interventions include consumption of probiotics;[S6.2-32,S6.2-33,S6.2-34] increased intake of protein,[S6.2-35–S6.2-37] fiber,[S6.2-38,S6.2-39] flaxseed,[S6.2-40] or fish oil;[S6.2-41] supplementation with calcium[S6.2-42,S6.2-43] or magnesium;[S6.2-44,S6.2-45] and use of dietary patterns other than the DASH diet, including

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low-carbohydrate, vegetarian, and Mediterranean diets.[S6.2-46–S6.2-49] Stress reduction is intuitively attractive but insufficiently proved,[S6.2-51] as are several other interventions, including consumption of garlic,[S6.2-52] dark chocolate,[S6.2-53,S6.2-54] tea,[S6.2-55] or coffee.[S6.2-56] Behavioral therapies, including guided breathing, yoga, transcendental meditation, and biofeedback, lack strong evidence for their long-term BP-lowering effect.[S6.2-51,S6.2-57–S6.2-61] The best proven nonpharmacological measures to prevent and treat hypertension are summarized in Table 15.[S6.2-62]

The nonpharmacological interventions presented in Table 15 may be sufficient to prevent hypertension and meet goal BP in managing patients with stage 1 hypertension, and they are an integral part of the management of persons with stage 2 hypertension. To a lesser extent, the Mediterranean diet[S6.2-49,S6.2-63] (which incorporates the basics of healthy eating but emphasizes consumption of legumes and monounsaturated fat, avoidance of red meats, and moderate intake of wine) has been effective in reducing BP, as well as improving lipid profile.

Recommendation-Specific Supportive Text

1. Weight loss is a core recommendation and should be achieved through a combination of reduced calorie intake and increased physical activity.[S6.2-1] The BP-lowering effect of weight loss in patients with elevated BP is consistent with the corresponding effect in patients with established hypertension, with an apparent dose–response relationship of about 1 mm Hg per kilogram of weight loss. Achievement and maintenance of weight loss through behavior change are challenging[S6.2-64–S6.2-66] but feasible over prolonged periods of follow-up.[S6.2-64] For those who do not meet their weight loss goals with nonpharmacological interventions, pharmacotherapy or minimally invasive and bariatric surgical procedures can be considered.[S6.2-67,S6.2-68] Surgical procedures tend to be more effective but are usually reserved for those with more severe and intractable obesity because of the frequency of complications.[S6.2-69]
2. The DASH eating plan is the diet best demonstrated to be effective for lowering BP. Because the DASH diet is high in fruits, vegetables, and low-fat dairy products, it provides a means to enhance intake of potassium, calcium, magnesium, and fiber. In hypertensive and nonhypertensive adults, the DASH diet has produced overall reductions in SBP of approximately 11 mm Hg and 3 mm Hg, respectively,[S6.2-7] and the diet was especially effective in blacks.[S6.2-70] When combined with weight loss[S6.2-6] or a reduction in sodium intake,[S6.2-5,S6.2-30] the effect size was substantially increased. Most of the clinical trial

Table 15. Best Proven Nonpharmacological Interventions for Prevention and Treatment of Hypertension*

	Nonpharmacological Intervention	Dose	Approximate Impact on SBP		
			Hypertension	Normotension	Reference
Weight loss	Weight/body fat	Best goal is ideal body weight, but aim for at least a 1-kg reduction in body weight for most adults who are overweight. Expect about 1 mm Hg for every 1-kg reduction in body weight.	−5 mm Hg	−2/3 mm Hg	S6.2-1
Healthy diet	DASH dietary pattern	Consume a diet rich in fruits, vegetables, whole grains, and low-fat dairy products, with reduced content of saturated and total fat.	−11 mm Hg	−3 mm Hg	S6.2-6,S6.2-7
Reduced intake of dietary sodium	Dietary sodium	Optimal goal is <1500 mg/d, but aim for at least a 1000-mg/d reduction in most adults.	−5/6 mm Hg	−2/3 mm Hg	S6.2-9,S6.2-10
Enhanced intake of dietary potassium	Dietary potassium	Aim for 3500–5000 mg/d, preferably by consumption of a diet rich in potassium.	−4/5 mm Hg	−2 mm Hg	S6.2-13
Physical activity	Aerobic	90–150 min/wk 65%–75% heart rate reserve	−5/8 mm Hg	−2/4 mm Hg	S6.2-18,S6.2-22
	Dynamic resistance	90–150 min/wk 50%–80% 1 rep maximum 6 exercises, 3 sets/exercise, 10 repetitions/set	−4 mm Hg	−2 mm Hg	S6.2-18
	Isometric resistance	4 × 2 min (hand grip), 1 min rest between exercises, 30%–40% maximum voluntary contraction, 3 sessions/wk 8–10 wk	−5 mm Hg	−4 mm Hg	S6.2-19,S6.2-31
Moderation in alcohol intake	Alcohol consumption	In individuals who drink alcohol, reduce alcohol† to: Men: ≤2 drinks daily Women: ≤1 drink daily	−4 mm Hg	−3 mm Hg	S6.2-22—S6.2-24

Resources: Your Guide to Lowering Your Blood Pressure With DASH—How Do I Make the DASH? Available at: https://www.nhlbi.nih.gov/health/resources/heart/hbp-dash-how-to. Accessed September 15, 2017.[S6.2-72]

Top 10 Dash Diet Tips. Available at: http://dashdiet.org/dash_diet_tips.asp. Accessed September 15, 2017.[S6.2-73]

*Type, dose, and expected impact on BP in adults with a normal BP and with hypertension.

†In the United States, one "standard" drink contains roughly 14 g of pure alcohol, which is typically found in 12 oz of regular beer (usually about 5% alcohol), 5 oz of wine (usually about 12% alcohol), and 1.5 oz of distilled spirits (usually about 40% alcohol).[S6.2-29]

DASH indicates Dietary Approaches to Stop Hypertension; and SBP, systolic blood pressure.

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experience comes from short-term feeding studies,[S6.2-7] but lifestyle change with the DASH diet has been successful in at least 2 trials that used a behavioral intervention over a 4-month[S6.2-30] or 6-month[S6.2-6] period of follow-up. Websites and books provide advice on implementation of the DASH diet.[S6.2-13,S6.2-71–S6.2-74] Counseling by a knowledgeable nutritionist can be helpful. Several other diets, including diets that are low in calories from carbohydrates,[S6.2-46] high-protein diets,[S6.2-75] vegetarian diets,[S6.2-48] and a Mediterranean dietary pattern,[S6.2-49,S6.2-63] have been shown to lower BP.

3. Sodium reduction interventions prevent hypertension and lower BP in adults with hypertension, especially in those with higher levels of BP, blacks, older persons, and others who are particularly susceptible to the effects of sodium on BP.[S6.2-8–S6.2-11] Sodium reduction interventions may prevent CVD.[S6.2-76,S6.2-77] Lifestyle change (behavioral) interventions usually reduce sodium intake by about 25% (approximately 1000 mg per day) and result in an average of about a 2–mm Hg to 3–mm Hg reduction in SBP in nonhypertensive individuals, though the reduction can be more than double this in more susceptible individuals, those with hypertension, and those concurrently on the DASH diet[S6.2-5] or receiving a weight loss intervention.[S6.2-12] Sodium reduction in adults with hypertension who are already being treated with BP-lowering medications further reduces SBP by about 3 mm Hg and can facilitate discontinuation of medication, although this requires maintenance of the lifestyle change and warrants careful monitoring.[S6.2-12] When combined with weight loss, the reduction in BP is almost doubled. A reduction in sodium intake may also lower SBP significantly in individuals with resistant hypertension who are taking multiple antihypertensive medications[S6.2-78] (see Section 11.1). Reduced dietary sodium has been reported to augment the BP-lowering effects of RAS blocker therapy.[S6.2-79] Maintenance of the lifestyle changes necessary to reduce sodium intake is challenging,[S6.2-2–S6.2-4,S6.2-12] but even a small decrement in sodium consumption is likely to be safe[S6.2-2,S6.2-4,S6.2-9,S6.2-12,S6.2-80] and beneficial,[S6.2-8,S6.2-81] especially in those whose BP is salt sensitive.[S6.2-82] In the United States, most dietary sodium comes from additions during food processing or during commercial food preparation at sit-down and fast-food restaurants.[S6.2-83,S6.2-84] Person-specific and policy approaches can be used to reduce dietary sodium intake.[S6.2-85,S6.2-86] Individuals can take action to reduce their dietary intake of sodium by choice of fresh foods, use of food labels to choose foods that are lower in sodium content, choice of foods with a "no added sodium" label, judicious use of condiments and sodium-infused foods, use of spices and low-sodium flavorings, careful ordering when eating out, control of food portion size, and avoiding or minimizing use of salt at the table. Dietary counseling by a nutritionist with expertise in behavior modification can be helpful. A reduction in the amount of sodium added during food processing, as well as fast food and restaurant food preparation, has the potential to substantially reduce sodium intake without the need for a conscious change in lifestyle.[S6.2-81,S6.2-85,S6.2-87]
4. Dietary potassium is inversely related to BP and hypertension in migrant studies,[S6.2-88] cross-sectional reports,[S6.2-89–S6.2-91] and prospective cohort studies.[S6.2-92] Likewise, dietary potassium[S6.2-93–S6.2-96] and a high intake of fruits and vegetables are associated with a lower incidence of stroke.[S6.2-97] Potassium interventions have been effective in lowering BP,[S6.2-13,S6.2-14,S6.2-16,S6.2-81] especially in adult patients consuming an excess of sodium[S6.2-13,S6.2-74,S6.2-98] and in blacks.[S6.2-13] The typical BP-lowering effect of a 60-mmol (1380-mg) administration of potassium chloride has been about 2 mm Hg and 4 to 5 mm Hg in adults with normotension and hypertension, respectively, although the response is up to twice as much in persons consuming a high-sodium diet. A reduction in the sodium/potassium index may be more important than the corresponding changes in either electrolyte alone.[S6.2-99] Some but not all studies suggest that the intervention effect may be restricted to adult patients with a low (1500-mg to 2000-mg) daily intake of potassium.[S6.2-92,S6.2-100] Most of the intervention experience comes from trials of relatively short duration (median of 5 to 6 weeks),[S6.2-13,S6.2-14] but the BP-lowering effect of potassium in adult patients consuming a high-sodium diet has been reproduced after an interval of 4.4 years.[S6.2-98] In most trials, potassium supplementation was achieved by administration of potassium chloride pills, but the BP response pattern was similar when dietary modification was used.[S6.2-13] Because potassium-rich diets tend to be heart healthy, they are preferred over use of pills for potassium supplementation. The 2015 Dietary Guidelines for Americans[S6.2-101] encourage a diet rich in potassium and identify the adequate intake level for adult patients as 4700 mg/day.[S6.2-102] The World Health Organization recommends a potassium intake of at least 90 mmol (3510 mg) per day from food for adult patients.[S6.2-15] Good sources of dietary potassium include fruits and vegetables, as well as low-fat dairy products, selected fish and meats, nuts, and soy products. Four to five servings of fruits and vegetables will usually provide 1500 to >3000 mg of potassium. This can be achieved by a diet, such as the DASH diet, that is high in potassium content.[S6.2-7]
5. A BP-lowering effect of increased physical activity has been repeatedly demonstrated in clinical trials, especially during dynamic aerobic exercise,[S6.2-18,S6.2-20,S6.2-22] but also during dynamic resistance training[S6.2-18,S6.2-21] and static isometric exercise.[S6.2-18,S6.2-19,S6.2-31] The average reductions in SBP with aerobic exercise are approximately 2 to 4 mm Hg and 5 to 8 mm Hg in adult patients with normotension and hypertension, respectively.[S6.2-18] Most trials have been of relatively short duration, but increased physical activity has been an intrinsic component of longer-term weight reduction interventions used to reduce BP and prevent hypertension.[S6.2-3,S6.2-4,S6.2-12] BP-lowering effects have been reported with lower- and higher-intensity exercise and with continuous and interval exercise training.[S6.2-18,S6.2-103] Meta-analyses suggest isometric exercise results in substantial lowering of BP.[S6.2-18,S6.2-19,S6.2-31]
6. In observational studies, there is a strong, predictable direct relationship between alcohol consumption and BP,

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especially above an intake of 3 standard drinks per day (approximately 36 ounces of regular beer, 15 ounces of wine, or 4.5 ounces of distilled spirits).[S6.2-29,S6.2-104,S6.2-105] Meta-analyses of RCTs that have studied the effect of reduced alcohol consumption on BP in adults have identified a significant reduction in SBP and DBP.[S6.2-23,S6.2-24] The benefit has seemed to be consistent across trials, but confined to those consuming ≥3 drinks/day, as well as dose dependent, with those consuming ≥6 drinks/day at baseline who reduce their alcohol intake by about 50%, experiencing an average reduction in SBP/DBP of approximately 5.5/4.0 mm Hg.[S6.2-23,S6.2-24] Only limited information is available on the effect of alcohol reduction on BP in blacks.[S6.2-23,S6.2-106] In contrast to its effect on BP, alcohol seems to have a beneficial effect on several biomarkers for CVD risk, including high-density lipoprotein cholesterol.[S6.2-107,S6.2-108] Observational studies have shown a relatively consistent finding of an inverse relationship between alcohol intake and CHD,[S6.2-109,S6.2-110] within a moderate range (approximately 12–14 and ≤9 standard drinks/week for men and women, respectively). On balance, it seems reasonable for those who are consuming moderate quantities of alcohol (≤2 drinks/day) to continue their moderate consumption of alcohol.

7. Patient Evaluation

The patient evaluation is designed to identify target organ damage and possible secondary causes of hypertension and to assist in planning an effective treatment regimen. Historical features are relevant to the evaluation of the patient (Table 16). The pattern of BP measurements and changes over time may differentiate primary from secondary causes of hypertension. A rise in BP associated with weight gain, lifestyle factors (such as a job change requiring travel and meals away from home), reduced frequency or intensity of physical activity, or advancing age in a patient with a strong family history of hypertension would suggest the diagnosis of primary hypertension. An evaluation of the patient's dietary habits, physical activity, alcohol consumption, and tobacco use should be performed, with recommendation of the nonpharmacological interventions detailed in Section 6.2 where appropriate. The history should also include inquiry into possible occurrence of symptoms to indicate a secondary cause (Tables 13 and 16). The patient's treatment goals and risk tolerance should also be elicited. This is especially true for older persons, for whom an assessment of multiple chronic conditions, frailty, and prognosis should be performed, including consideration of the time required to see benefit from intervention, which may not be realized for some individuals.

The physical examination should include accurate measurement of BP (Table 8). Automated oscillometric devices provide an opportunity to obtain repeated measurements without a provider present, thereby minimizing the potential for a white coat effect. Change in BP from seated to standing position should be measured to detect orthostatic hypotension (a decline >20 mm Hg in SBP or >10 mm Hg in DBP after 1 minute is abnormal). For adults ≤30 years of age with elevated brachial BP, a thigh BP measurement is indicated; if the thigh measurement is lower than arm pressures, a diagnosis of coarctation of the aorta should be considered. The physical examination should include assessment of hypertension-related target organ damage. Attention should be paid to physical features that suggest secondary hypertension (Table 13).

Table 16. Historical Features Favoring Hypertension Cause

Primary Hypertension	Secondary Hypertension
Gradual increase in BP, with slow rate of rise in BP	BP lability, episodic pallor and dizziness (pheochromocytoma)
Lifestyle factors that favor higher BP (eg, weight gain, high-sodium diet, decreased physical activity, job change entailing increased travel, excessive consumption of alcohol)	Snoring, hypersomnolence (obstructive sleep apnea)
	Prostatism (chronic kidney disease due to post-renal urinary tract obstruction)
Family history of hypertension	Muscle cramps, weakness (hypokalemia from primary aldosteronism or secondary aldosteronism due to renovascular disease)
	Weight loss, palpitations, heat intolerance (hyperthyroidism)
	Edema, fatigue, frequent urination (kidney disease or failure)
	History of coarctation repair (residual hypertension associated with coarctation)
	Central obesity, facial rounding, easy bruisability (Cushing's syndrome)
	Medication or substance use (eg, alcohol, NSAIDS, cocaine, amphetamines)
	Absence of family history of hypertension

BP indicates blood pressure; and NSAIDs, nonsteroidal anti-inflammatory drugs.

7.1. Laboratory Tests and Other Diagnostic Procedures

Laboratory measurements should be obtained for all patients with a new diagnosis of hypertension to facilitate CVD risk factor profiling, establish a baseline for medication use, and screen for secondary causes of hypertension (Table 17). Optional tests may provide information on target organ damage. Monitoring of serum sodium and potassium levels is helpful during diuretic or RAS blocker titration, as are serum creatinine and urinary albumin as markers of CKD progression.[S7.1-1] Measurement of thyroid-stimulating hormone is a simple test to easily detect hypothyroidism and hyperthyroidism, 2 remediable causes of hypertension. A decision to conduct additional laboratory testing would be appropriate in the context of increased hypertension severity, poor response to standard treatment approaches, a disproportionate severity of target organ damage for the level of BP, or historical or clinical clues that support a secondary cause.

7.2. Cardiovascular Target Organ Damage

Pulse-wave velocity, carotid intima-media thickness, and coronary artery calcium score provide noninvasive estimates

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Table 17. Basic and Optional Laboratory Tests for Primary Hypertension

Basic testing	Fasting blood glucose*
	Complete blood count
	Lipid profile
	Serum creatinine with eGFR*
	Serum sodium, potassium, calcium*
	Thyroid-stimulating hormone
	Urinalysis
	Electrocardiogram
Optional testing	Echocardiogram
	Uric acid
	Urinary albumin to creatinine ratio

*May be included in a comprehensive metabolic panel.
eGFR indicates estimated glomerular filtration rate.

of vascular target organ injury and atherosclerosis.[S7.2-1] High BP readings, especially when obtained several years before a noninvasive measurement, are associated with an increase in subclinical CVD risk.[S7.2-2–S7.2-4] Although carotid intima-media thickness values and coronary artery calcium scores are associated with cardiovascular events, inadequate or absent information on the effect of improvement in these markers on cardiovascular events prevents their routine use as surrogate markers in the treatment of hypertension.

LVH is a secondary manifestation of hypertension and independently predicts future CVD events. LVH is commonly measured by electrocardiography, echocardiography, or MRI.[S7.2-5,S7.2-6] Left ventricular (LV) mass is associated with body size (particularly lean body mass), tobacco use, heart rate (inverse), and long-standing DM.[S7.2-7–S7.2-9] BP lowering leads to a reduction in LV mass. In TOMHS (Treatment of Mild Hypertension Study), the long-acting diuretic chlorthalidone was slightly more effective in reducing LVH than were a calcium channel blocker (CCB) (amlodipine), ACE inhibitor (enalapril), alpha-receptor blocker (doxazosin), or beta-receptor blocker (acebutolol).[S7.2-10] Beta blockers are inferior to angiotensin receptor blockers (ARBs), angiotensin-converting enzyme (ACE) inhibitors, and CCBs in reducing LVH.[S7.2-11]

Hypertension adversely impacts other echocardiographic markers of cardiac structure and function, including left atrial size (both diameter and area; left atrial size is also a precursor of AF); diastolic function (many parameters; a precursor of HF with preserved ejection fraction [HF*p*EF]); cardiac structure; and subclinical markers of LV systolic function, such as myocardial strain assessment with echocardiography and MRI.

Assessment of LVH by means of echocardiography or MRI is not universally recommended during evaluation and management of hypertension in adults because there are limited data on the cost and value of these measures for CVD risk reclassification and changes in type or intensity of treatment. Assessment of LVH is most useful in adults who are young (≤18 years of age) or have evidence of secondary hypertension, chronic uncontrolled hypertension, or history of symptoms of HF. Electrocardiographic criteria for LVH correlate weakly with echocardiographic or MRI definitions of LVH and are less strongly related to CVD outcomes.[S7.2-12–S7.2-15] Imprecision in lead placement accounts, in part, for the poor correlation of electrocardiographic measurements with direct imaging results. However, electrocardiographic LVH has been valuable in predicting CVD risk in some reports.[S7.2-16,S7.2-17] Electrocardiography may also be useful in the assessment of comorbidities, such as rhythm disturbances and prior MI.

LVH, as assessed by electrocardiography, echocardiography, or MRI, is an independent predictor of CVD complications.[S7.2-18,S7.2-19] Reduction in LVH can predict a reduction in CVD risk, independent of change in BP.[S7.2-20] When used in CVD risk predictor models, echocardiographic LVH has a small but significant independent effect on CVD risk in younger patients. At older ages, LVH measured by electrocardiography or MRI provides no independent contribution to prediction of CVD risk.[S7.2-21–S7.2-23] Patients can be classified into 4 groups on the basis of the presence or absence of LVH and a determination of whether the LVH has an eccentric (normal relative wall thickness) or concentric geometry.[S7.2-6,S7.2-22]

8. Treatment of High BP

Clinicians managing adults with high BP should focus on overall patient health, with a particular emphasis on reducing the risk of future adverse CVD outcomes. All patient risk factors need to be managed in an integrated fashion with a comprehensive set of nonpharmacological (see Section 6) and pharmacological strategies. As patient BP and risk of future CVD events increase, BP management should be intensified.

8.1. Pharmacological Treatment

8.1.1. Initiation of Pharmacological BP Treatment in the Context of Overall CVD Risk

For any specific difference in BP, the relative risk of CVD is constant across groups that differ in absolute risk of atherosclerotic CVD,[S8.1.1-1–S8.1.1-4] albeit with some evidence of lesser relative risk but greater excess risk in older than in younger adults.[S8.1.1-5–S8.1.1-8] Thus, there are more potentially preventable CVD events attributable to elevated BP in individuals with higher than with lower risk of CVD and in older than in younger adults. The relative risk reduction for CVD prevention with use of BP-lowering medications is fairly constant for groups that differ in CVD risk across a wide range of estimated absolute risk[S8.1.1-9,S8.1.1-10] and across groups defined by sex, age, body mass index, and the presence or absence of DM, AF, and CKD.[S8.1.1-5,S8.1.1-11–S8.1.1-21] As a consequence, the absolute CVD risk reduction attributable to BP lowering is greater at greater absolute levels of CVD risk.[S8.1.1-9,S8.1.1-10,S8.1.1-12,S8.1.1-15–S8.1.1-19,S8.1.1-22,S8.1.1-23] Put another way, for a given magnitude of BP reduction due to antihypertensive medications, fewer individuals at high CVD risk would need to be treated to prevent a CVD event (ie, lower number needed to treat) than those at low CVD risk.

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8.1.2. BP Treatment Threshold and the Use of CVD Risk Estimation to Guide Drug Treatment of Hypertension

Recommendations for BP Treatment Threshold and Use of Risk Estimation* to Guide Drug Treatment of Hypertension

References that support recommendations are summarized in Online Data Supplement 23.

COR	LOE	Recommendations
I	SBP: A DBP: C-EO	1. Use of BP-lowering medications is recommended for secondary prevention of recurrent CVD events in patients with clinical CVD and an average SBP of 130 mm Hg or higher or an average DBP of 80 mm Hg or higher, and for primary prevention in adults with an estimated 10-year atherosclerotic cardiovascular disease (ASCVD) risk of 10% or higher and an average SBP 130 mm Hg or higher or an average DBP 80 mm Hg or higher.[S8.1.2-1–S8.1.2-9]
I	C-LD	2. Use of BP-lowering medication is recommended for primary prevention of CVD in adults with no history of CVD and with an estimated 10-year ASCVD risk <10% and an SBP of 140 mm Hg or higher or a DBP of 90 mm Hg or higher.[S8.1.2-3,S8.1.2-10–S8.1.2-13]

*ACC/AHA Pooled Cohort Equations (http://tools.acc.org/ASCVD-Risk-Estimator/)[S8.1.2-13a] to estimate 10-year risk of atherosclerotic CVD. ASCVD was defined as a first CHD death, non-fatal MI or fatal or non-fatal stroke.

Synopsis

Whereas treatment of high BP with BP-lowering medications on the basis of BP level alone is considered cost effective,[S8.1.2-14] use of a combination of absolute CVD risk and BP level to guide such treatment is more efficient and cost effective at reducing risk of CVD than is use of BP level alone.[S8.1.2-15–S8.1.2-24] Practical approaches have been developed to translate evidence from RCTs into individual patient treatment recommendations that are based on absolute net benefit for CVD risk,[S8.1.2-25] and several national and international guidelines recommend basing use of BP-lowering medications on a combination of absolute risk of CVD and level of BP instead of relying solely on level of BP.[S8.1.2-26–S8.1.2-31]

Attempts to use absolute risk to guide implementation of pharmacological treatment to prevent CVD have had mixed results, with many reports of improvements in provider prescribing behaviors, patient adherence, and reductions in risk,[S8.1.2-32–S8.1.2-38] but with others showing no impact on provider behaviors.[S8.1.2-39,S8.1.2-40] Use of global CVD risk assessment is infrequent in routine clinical practice,[S8.1.2-41–S8.1.2-46] which suggests that intensive efforts would be required to achieve universal implementation. The choice of specific risk calculators for estimation of risk and risk threshold has been an important source of variability, ambiguity, and controversy.[S8.1.2-47–S8.1.2-54] In addition, implementation of a standard (worldwide) absolute CVD risk threshold for initiating use of BP-lowering medications would result in large variations in medication use at a given level of BP across countries.[S8.1.2-48,S8.1.2-54,S8.1.2-55] Future research in this area should focus on issues related to implementation of a risk-based approach to CVD prevention, including the use of BP-lowering medications. Although several CVD risk assessment tools are available, on the basis of current knowledge, we recommend use of the ACC/AHA Pooled Cohort Equations (http://tools.acc.org/ASCVD-Risk-Estimator/) to estimate 10-year risk of atherosclerotic CVD (ASCVD) to establish the BP threshold for treatment.[S8.1.2-56,S8.1.2-57] It should be kept in mind that the ACC/AHA Pooled Cohort Equations are validated for US adults ages 40 to 79 years in the absence of concurrent statin therapy.[S8.1.2-56] For those >79 years old, the 10-year ASCVD risk is generally >10%, and thus the SBP threshold for antihypertensive drug treatment for patients >79 years old is 130 mm Hg. Two recent reviews have highlighted the importance of using predicted CVD risk together with BP to guide antihypertensive drug therapy.[S8.1.2-22,S8.1.2-23]

Figure 4 is an algorithm on BP thresholds and recommendations for treatment and follow-up.

Recommendation-Specific Supportive Text

1. For the purposes of secondary prevention, clinical CVD is defined as CHD, congestive HF, and stroke. Several meta-analyses of RCTs support the value of using BP-lowering medications, in addition to nonpharmacological treatment, in patients with established CVD in the absence of hypertension, defined previously by an SBP ≥140 mm Hg or a DBP ≥90 mm Hg.[S8.1.2-1,S8.1.2-6,S8.1.2-7,S8.1.2-9] Many RCTs of BP lowering in adults without CVD have used inclusion criteria designed to increase the level of CVD risk in the study populations to increase trial efficiency by facilitating shorter duration and a smaller sample size. As a consequence, few relatively low-risk adults with hypertension have been included in the trials. Trial results provide evidence of CVD prevention from use of BP-lowering medications in adults with an average SBP ≥130 mm Hg or an average DBP ≥80 mm Hg and clinical CVD; 5-year risk of CVD (defined as stroke, CHD, HF, or other CVD death) of approximately 6% to 7%;[S8.1.2-3,S8.1.2-5] an estimated 10-year CVD death rate of approximately 4.5%;[S8.1.2-4] or an annual rate of major CVD events of approximately 0.9% per year.[S8.1.2-7] In the absence of clinical CVD, these risk estimates are roughly equivalent to a 10-year risk of ASCVD exceeding 10% as per the ACC/AHA Pooled Cohort Equations.[S8.1.2-56] SPRINT (Systolic Blood Pressure Intervention Trial) provides additional support for the use of BP-lowering medications in patients without CVD at SBP levels ≥130 mm Hg; however, it is important to note that few SPRINT participants had untreated SBP between 130 mm Hg and 139 mm Hg at baseline. Furthermore, SPRINT used a Framingham 10-year risk of general CVD exceeding 15% to identify increased CVD risk.[S8.1.2-8] Although this level of risk is lower than the levels described previously, being roughly equivalent to a 6% to 7% 10-year ASCVD risk per the ACC/AHA Pooled Cohort Equations, most of the participants in SPRINT had a much higher level of CVD risk. This recommendation differs from JNC 7 in its use of CVD risk, rather than diabetes or CKD, to recognize patients, including older adults, with a SBP/DBP <140/90 mm Hg who are likely to benefit from BP lowering drug therapy in addition to nonpharmacological antihypertensive treatment. In JNC 7, the BP

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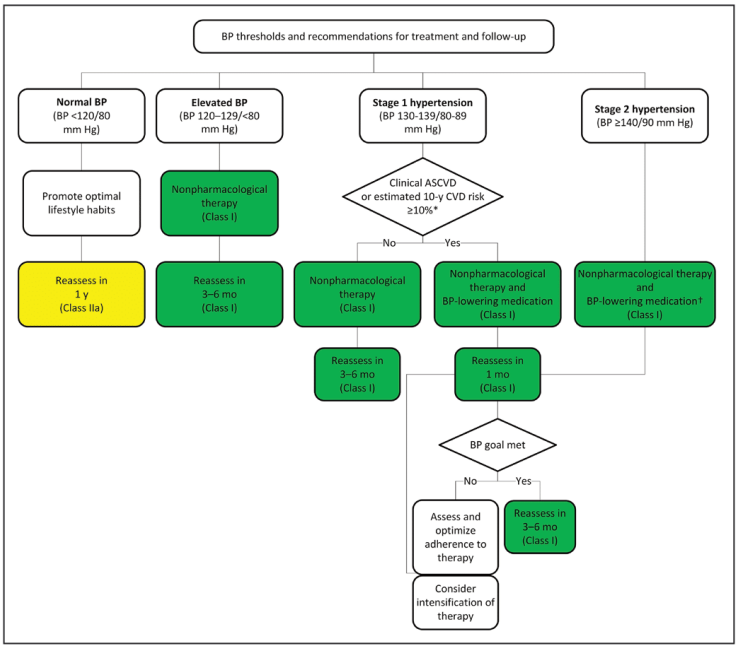

Figure 4. Blood pressure (BP) thresholds and recommendations for treatment and follow-up. Colors correspond to Class of Recommendation in Table 1. *Using the ACC/AHA Pooled Cohort Equations.[S8.1.2-56,S8.1.2-57] Note that patients with DM or CKD are automatically placed in the high-risk category. For initiation of RAS inhibitor or diuretic therapy, assess blood tests for electrolytes and renal function 2 to 4 weeks after initiating therapy. †Consider initiation of pharmacological therapy for stage 2 hypertension with 2 antihypertensive agents of different classes. Patients with stage 2 hypertension and BP ≥160/100 mm Hg should be promptly treated, carefully monitored, and subject to upward medication dose adjustment as necessary to control BP. Reassessment includes BP measurement, detection of orthostatic hypotension in selected patients (eg, older or with postural symptoms), identification of white coat hypertension or a white coat effect, documentation of adherence, monitoring of the response to therapy, reinforcement of the importance of adherence, reinforcement of the importance of treatment, and assistance with treatment to achieve BP target. ACC indicates American College of Cardiology; AHA, American Heart Association; ASCVD, atherosclerotic cardiovascular disease; BP, blood pressure; CKD, chronic kidney disease; DM, diabetes mellitus; and RAS, renin-angiotensin system.

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threshold for initiation of antihypertensive drug therapy was ≥ 140/90 mm Hg for the general adult population and ≥ 130/80 mm Hg for adults with diabetes or CKD. Since the publication of JNC 7 in 2003, we have gained additional experience with risk assessment and new data from randomized trials, observational studies and simulation analyses have demonstrated that antihypertensive drug treatment based on overall ASCVD risk assessment combined with BP levels may prevent more CVD events than treatment based on BP levels alone.[S8.1.2-15–S8.1.2-24] According to an analysis of NHANES 2011-2014, the new definition results in only a small increase in the percentage of US adults for whom antihypertensive medication is recommended in conjunction with lifestyle modification. The previously cited meta-analyses are consistent with the conclusion that lowering of BP results in benefit in higher-risk individuals, regardless of their baseline treated or untreated BP ≥130/80 mm Hg and irrespective of the specific cause of their elevated risk. These analyses indicate that the benefit of treatment outweighs the potential harm at threshold BP ≥130/80 mm Hg.

2. This recommendation is consistent with prior guidelines, such as JNC 7. In addition, for those for whom nonpharmacological therapy has been ineffective, antihypertensive drug treatment should be added in patients with an SBP ≥140 mm Hg or a DBP ≥90 mm Hg, even in adults who are at lower risk than those included in RCTs. The rationale for drug treatment in patients with an SBP ≥140 mm Hg or a DBP ≥90 mm Hg and an estimated 10-year risk of CVD <10% is based on several lines of evidence. First, the relationship of SBP with risk of CVD is known to be continuous across levels of SBP and similar across groups that differ in level of absolute risk.[S8.1.2-10] Second, the relative risk reduction attributable to BP-lowering medication therapy is consistent across the range of absolute risk observed in trials,[S8.1.2-3,S8.1.2-11,S8.1.2-58] supporting the contention that the relative risk reduction may be similar at lower levels of absolute risk. This is the case even in a meta-analysis of trials in adults without clinical CVD and an average SBP/DBP of 146/84 mm Hg.[S8.1.2-5] Finally, modeling studies support the effectiveness and cost-effectiveness of treatment of younger, lower-risk patients

over the course of their life spans.[S8.1.2-12,S8.1.2-13] Although the numbers needed to treat with BP-lowering medications to prevent a CVD event in the short term are greater in younger, lower-risk individuals with hypertension than in older, higher-risk adults with hypertension, the estimated gains in life expectancy attributable to long-term use of BP-lowering medications are correspondingly greater in younger, lower-risk individuals than in older adults with a higher risk of CVD.[S8.1.2-12,S8.1.2-13] Indirect support is also provided by evidence from trials using BP-lowering medications to reduce the risk of developing higher levels of BP[S8.1.2-59–S8.1.2-61] and, in one case, to achieve a reduction in LV mass.[S8.1.2-62] In the HOPE-3 (Heart Outcomes Prevention Evaluation-3) BP Trial, there was no evidence of short-term benefit during treatment of adults (average age 66 years) with a relatively low risk of CVD (3.8% CVD event rate during 5.6 years of follow-up). However, subgroup analysis suggested benefit in those with an average SBP approximately >140 mm Hg (and a CVD risk of 6.5% during the 5.6 years of follow-up).[S8.1.2-63] We acknowledge the importance of excluding white coat hypertension before initiating pharmacological therapy in hypertensive patients with low ASCVD risk. This may be accomplished (as described in Section 4) by HBPM or ABPM as appropriate.

8.1.3. Follow-Up After Initial BP Evaluation

Recommendations for Follow-Up After Initial BP Evaluation

References that support recommendations are summarized in Online Data Supplement 24.

COR	LOE	Recommendations
I	B-R	1. Adults with an elevated BP or stage 1 hypertension who have an estimated 10-year ASCVD risk less than 10% should be managed with nonpharmacological therapy and have a repeat BP evaluation within 3 to 6 months.[S8.1.3-1,S8.1.3-2]
I	B-R	2. Adults with stage 1 hypertension who have an estimated 10-year ASCVD risk of 10% or higher should be managed initially with a combination of nonpharmacological and antihypertensive drug therapy and have a repeat BP evaluation in 1 month.[S8.1.3-1,S8.1.3-2]
I	B-R	3. Adults with stage 2 hypertension should be evaluated by or referred to a primary care provider within 1 month of the initial diagnosis, have a combination of nonpharmacological and antihypertensive drug therapy (with 2 agents of different classes) initiated, and have a repeat BP evaluation in 1 month.[S8.1.3-1,S8.1.3-2]
I	B-R	4. For adults with a very high average BP (eg, SBP ≥180 mm Hg or DBP ≥110 mm Hg), evaluation followed by prompt antihypertensive drug treatment is recommended.[S8.1.3-1,S8.1.3-2]
IIa	C-EO	5. For adults with a normal BP, repeat evaluation every year is reasonable.

Synopsis

An important component of BP management in hypertensive patients is follow-up. Different periods of time for follow-up are recommended depending on the stage of hypertension, the presence or absence of target organ damage, treatment with antihypertensive medications, and the level of BP control. Recommendations for follow-up are summarized in Figure 4.

Recommendation-Specific Supportive Text

1. Nonpharmacological therapy (see Section 6.2) is the preferred therapy for adults with elevated BP and an appropriate first-line therapy for adults with stage 1 hypertension who have an estimated 10-year ASCVD risk of <10%. Adherence to and impact of nonpharmacological therapy should be assessed within 3 to 6 months.
2. Nonpharmacological therapy can help reduce BP in patients with stage 1 hypertension with an estimated 10-year ASCVD risk of ≥10% and should be used in addition to pharmacological therapy as first-line therapy in such patients (see Section 6.2).
3. Prompt evaluation and treatment of patients with stage 2 hypertension with a combination of drug and nonpharmacological therapy are important because of the elevated risk of CVD events in this subgroup, especially those with multiple ASCVD risk factors or target organ damage.[S8.1.3-1,S8.1.3-2]
4. Prompt management of very high BP is important to reduce the risk of target organ damage (see Section 11.2). The rapidity of the treatment needed is dependent on the patient's clinical presentation (presence of new or worsening target organ damage) and presence or absence of CVD complications, but treatment should be initiated within at least 1 week.
5. Given that the lifetime risk of hypertension exceeds 80% in US adults,[S8.1.3-3] it is likely that individuals with a normal BP will develop elevated BP in the future. BP may change over time because of changes in BP-related lifestyle factors, such as degree of sedentary lifestyle, dietary sodium intake, body weight, and alcohol intake. Less commonly, secondary causes of hypertension can occur over time and lead to an increase in BP. Periodic BP screening can identify individuals who develop elevated BP over time. More frequent BP screening may be particularly important for individuals with elevated ASCVD risk.

8.1.4. General Principles of Drug Therapy

Recommendation for General Principle of Drug Therapy

References that support recommendations are summarized in Online Data Supplement 25.

COR	LOE	Recommendation
III: Harm	A	1. Simultaneous use of an ACE inhibitor, ARB, and/or renin inhibitor is potentially harmful and is not recommended to treat adults with hypertension.[S8.1.4-1–S8.1.4-3]

Synopsis

Pharmacological agents, in addition to lifestyle modification (see Section 6.2), provide the primary basis for treatment of high BP. A large number of clinical trials have demonstrated that antihypertensive pharmacotherapy not only lowers BP but reduces the risk of CVD, cerebrovascular events, and death.[S8.1.4-4–S8.1.4-7]

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Numerous classes of antihypertensive agents are available to treat high BP (Table 18). Agents that have been shown to reduce clinical events should be used preferentially. Therefore, the primary agents used in the treatment of hypertension include thiazide diuretics, ACE inhibitors, ARBs, and CCBs[S8.1.4-8–S8.1.4-11] (see Section 8.1.6). Although many other drugs and drug classes are available, either confirmation that these agents decrease clinical outcomes to an extent similar to that of the primary agents is lacking, or safety and tolerability may relegate their role to use as secondary agents. In particular, there is inadequate evidence to support the initial use of beta blockers for hypertension in the absence of specific cardiovascular comorbidities (see Section 9).

When the initial drug treatment of high BP is being considered, several different strategies may be contemplated. Many patients can be started on a single agent, but consideration should be given to starting with 2 drugs of different classes for those with stage 2 hypertension (see Section 8.1.6.1). In addition, other patient-specific factors, such as age, concurrent medications, drug adherence, drug interactions, the overall treatment regimen, out-of-pocket costs, and comorbidities, should be considered. From a societal perspective, total costs must be taken into account. Shared decision making, with the patient influenced by clinician judgment, should drive the ultimate choice of antihypertensive agent(s).

Many patients started on a single agent will subsequently require ≥2 drugs from different pharmacological classes to reach their BP goals.[S8.1.4-12,S8.1.4-13,S8.1.4-14] Knowledge of the pharmacological mechanisms of action of each agent is important. Drug regimens with complementary activity, where a second antihypertensive agent is used to block compensatory responses to the initial agent or affect a different pressor mechanism, can result in additive lowering of BP. For example, thiazide diuretics may stimulate the renin-angiotensin-aldosterone system. By adding an ACE inhibitor or ARB to the thiazide, an additive BP-lowering effect may be obtained.[S8.1.4-13] Use of combination therapy may also improve adherence. Several 2- and 3-fixed-dose drug combinations of antihypertensive drug therapy are available, with complementary mechanisms of action among the components (Online Data Supplement D). However, it should be noted that many triple-dose combinations may contain a lower-than-optimal dose of thiazide diuretic.

Table 18 is a summary of oral antihypertensive drugs.

Recommendation-Specific Supportive Text

1. Drug combinations that have similar mechanisms of action or clinical effects should be avoided. For example, 2 drugs from the same class should not be administered together (eg, 2 different beta blockers, ACE inhibitors, or nondihydropyridine CCBs). Likewise, 2 drugs from classes that target the same BP control system are less effective and potentially harmful when used together (eg, ACE inhibitors, ARBs). Exceptions to this rule include concomitant use of a thiazide diuretic, K-sparing diuretic, and/or loop diuretic in various combinations. Also, dihydropyridine and nondihydropyridine CCBs can be combined. High-quality RCT data demonstrate that simultaneous administration of RAS blockers (ie, ACE inhibitor with ARB; ACE inhibitor or ARB with renin inhibitor aliskiren) increases cardiovascular and renal risk.[S8.1.4-1–S8.1.4-3]

8.1.5. BP Goal for Patients With Hypertension

Recommendations for BP Goal for Patients With Hypertension

References that support recommendations are summarized in Online Data Supplement 26 and Systematic Review Report.

COR	LOE	Recommendations
I	SBP: B-R[SR]	1. For adults with confirmed hypertension and known CVD or 10-year ASCVD event risk of 10% or higher (see Section 8.1.2), a BP target of less than 130/80 mm Hg is recommended.[S8.1.5-1–S8.1.5-5]
	DBP: C-EO	
IIb	SBP: B-NR	2. For adults with confirmed hypertension, without additional markers of increased CVD risk, a BP target of less than 130/80 mm Hg may be reasonable.[S8.1.5-6–S8.1.5-9]
	DBP: C-EO	

SR indicates systematic review.

Synopsis

Refer to the "Systematic Review for the 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults" for the complete systematic evidence review for additional data and analyses.[S8.1.5-10] Several trials have tested whether more intensive BP control improves major CVD outcomes. Meta-analyses and systematic reviews of these trials provide strong support for the more intensive approach, but the data are less clear in identification of a specific optimal BP target.[S8.1.5-1–S8.1.5-5,S8.1.5-7,S8.1.5-11–S8.1.5-13] Recent trials that address optimal BP targets include SPRINT and ACCORD (Action to Control Cardiovascular Risk in Diabetes), with targets for more intensive (SBP <120 mm Hg) and standard (SBP <140 mm Hg) treatment,[S8.1.5-14,S8.1.5-15] and SPS-3, with a more intensive target of <130/80 mm Hg.[S8.1.5-16] These trials yielded mixed results in achieving their primary endpoints. SPRINT was stopped early, after a median follow-up of 3.26 years, when more intensive treatment resulted in a significant reduction in the primary outcome (a CVD composite) and in all-cause mortality rate. In ACCORD, more intensive BP treatment failed to demonstrate a significant reduction in the primary outcome (a CVD composite). However, the incidence of stroke, a component of the primary outcome, was significantly reduced. The standard glycemia subgroup did show significant benefit in ACCORD, and a meta-analysis of the only 2 trials (ACCORD and SPRINT) testing an SBP goal of <120 mm Hg showed significant reduction in CVD events.[S8.1.5-17] SPS-3 failed to demonstrate benefit for the primary endpoint of recurrent stoke (P=0.08) but found a significant reduction in a subgroup with hemorrhagic stroke. Pooling of the experience from 19 trials (excluding SPRINT) that randomly assigned participants to different BP treatment targets identified a significant reduction in CVD events, MI, and stroke in those assigned to a lower (average achieved SBP/DBP was 133/76 mm Hg) versus a higher BP treatment target.[S8.1.5-2] Similar patterns of benefit were reported in 3 other meta-analyses of trials in which participants were randomly

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Table 18. Oral Antihypertensive Drugs

Class	Drug	Usual Dose, Range (mg/d)*	Daily Frequency	Comments
Primary agents				
Thiazide or thiazide-type diuretics	Chlorthalidone	12.5–25	1	Chlorthalidone is preferred on the basis of prolonged half-life and proven trial reduction of CVD. Monitor for hyponatremia and hypokalemia, uric acid and calcium levels. Use with caution in patients with history of acute gout unless patient is on uric acid–lowering therapy.
	Hydrochlorothiazide	25–50	1	
	Indapamide	1.25–2.5	1	
	Metolazone	2.5–5	1	
ACE inhibitors	Benazepril	10–40	1 or 2	Do not use in combination with ARBs or direct renin inhibitor. There is an increased risk of hyperkalemia, especially in patients with CKD or in those on K^+ supplements or K^+-sparing drugs. There is a risk of acute renal failure in patients with severe bilateral renal artery stenosis. Do not use if patient has history of angioedema with ACE inhibitors. Avoid in pregnancy.
	Captopril	12.5–150	2 or 3	
	Enalapril	5–40	1 or 2	
	Fosinopril	10–40	1	
	Lisinopril	10–40	1	
	Moexipril	7.5–30	1 or 2	
	Perindopril	4–16	1	
	Quinapril	10–80	1 or 2	
	Ramipril	2.5–20	1 or 2	
	Trandolapril	1–4	1	
ARBs	Azilsartan	40–80	1	Do not use in combination with ACE inhibitors or direct renin inhibitor. There is an increased risk of hyperkalemia in CKD or in those on K^+ supplements or K^+-sparing drugs. There is a risk of acute renal failure in patients with severe bilateral renal artery stenosis. Do not use if patient has history of angioedema with ARBs. Patients with a history of angioedema with an ACE inhibitor can receive an ARB beginning 6 weeks after ACE inhibitor is discontinued. Avoid in pregnancy.
	Candesartan	8–32	1	
	Eprosartan	600–800	1 or 2	
	Irbesartan	150–300	1	
	Losartan	50–100	1 or 2	
	Olmesartan	20–40	1	
	Telmisartan	20–80	1	
	Valsartan	80–320	1	
CCB—dihydropyridines	Amlodipine	2.5–10	1	Avoid use in patients with HFrEF; amlodipine or felodipine may be used if required. They are associated with dose-related pedal edema, which is more common in women than men.
	Felodipine	2.5–10	1	
	Isradipine	5–10	2	
	Nicardipine SR	60–120	2	
	Nifedipine LA	30–90	1	
	Nisoldipine	17–34	1	
CCB—nondihydropyridines	Diltiazem ER	120–360	1	Avoid routine use with beta blockers because of increased risk of bradycardia and heart block. Do not use in patients with HFrEF. There are drug interactions with diltiazem and verapamil (*CYP3A4* major substrate and moderate inhibitor).
	Verapamil IR	120–360	3	
	Verapamil SR	120–360	1 or 2	
	Verapamil-delayed onset ER	100–300	1 (in the evening)	
Secondary agents				
Diuretics—loop	Bumetanide	0.5–2	2	These are preferred diuretics in patients with symptomatic HF. They are preferred over thiazides in patients with moderate-to-severe CKD (eg, GFR <30 mL/min).
	Furosemide	20–80	2	
	Torsemide	5–10	1	
Diuretics—potassium sparing	Amiloride	5–10	1 or 2	These are monotherapy agents and minimally effective antihypertensive agents. Combination therapy of potassium-sparing diuretic with a thiazide can be considered in patients with hypokalemia on thiazide monotherapy. Avoid in patients with significant CKD (eg, GFR <45 mL/min).
	Triamterene	50–100	1 or 2	

(*Continued*)

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Table 18. Continued

Class	Drug	Usual Dose, Range (mg/d)*	Daily Frequency	Comments
Diuretics—aldosterone antagonists	Eplerenone	50–100	1 or 2	These are preferred agents in primary aldosteronism and resistant hypertension. Spironolactone is associated with greater risk of gynecomastia and impotence as compared with eplerenone. This is common add-on therapy in resistant hypertension. Avoid use with K^+ supplements, other K^+-sparing diuretics, or significant renal dysfunction. Eplerenone often requires twice-daily dosing for adequate BP lowering.
	Spironolactone	25–100	1	
Beta blockers—cardioselective	Atenolol	25–100	2	Beta blockers are not recommended as first-line agents unless the patient has IHD or HF. These are preferred in patients with bronchospastic airway disease requiring a beta blocker. Bisoprolol and metoprolol succinate are preferred in patients with HF/EF. Avoid abrupt cessation.
	Betaxolol	5–20	1	
	Bisoprolol	2.5–10	1	
	Metoprolol tartrate	100–200	2	
	Metoprolol succinate	50–200	1	
Beta blockers—cardioselective and vasodilatory	Nebivolol	5–40	1	Nebivolol induces nitric oxide–induced vasodilation. Avoid abrupt cessation.
Beta blockers—noncardioselective	Nadolol	40–120	1	Avoid in patients with reactive airways disease. Avoid abrupt cessation.
	Propranolol IR	80–160	2	
	Propranolol LA	80–160	1	
Beta blockers—intrinsic sympathomimetic activity	Acebutolol	200–800	2	Generally avoid, especially in patients with IHD or HF. Avoid abrupt cessation.
	Penbutolol	10–40	1	
	Pindolol	10–60	2	
Beta blockers—combined alpha- and beta-receptor	Carvedilol	12.5–50	2	Carvedilol is preferred in patients with HF/EF. Avoid abrupt cessation.
	Carvedilol phosphate	20–80	1	
	Labetalol	200–800	2	
Direct renin inhibitor	Aliskiren	150–300	1	Do not use in combination with ACE inhibitors or ARBs. Aliskiren is very long acting. There is an increased risk of hyperkalemia in CKD or in those on K^+ supplements or K^+-sparing drugs. Aliskiren may cause acute renal failure in patients with severe bilateral renal artery stenosis. Avoid in pregnancy.
Alpha-1 blockers	Doxazosin	1–16	1	These are associated with orthostatic hypotension, especially in older adults. They may be considered as second-line agent in patients with concomitant BPH.
	Prazosin	2–20	2 or 3	
	Terazosin	1–20	1 or 2	
Central alpha$_2$-agonist and other centrally acting drugs	Clonidine oral	0.1–0.8	2	These are generally reserved as last-line because of significant CNS adverse effects, especially in older adults. Avoid abrupt discontinuation of clonidine, which may induce hypertensive crisis; clonidine must be tapered to avoid rebound hypertension.
	Clonidine patch	0.1–0.3	1 weekly	
	Methyldopa	250–1000	2	
	Guanfacine	0.5–2	1	
Direct vasodilators	Hydralazine	100–200	2 or 3	These are associated with sodium and water retention and reflex tachycardia; use with a diuretic and beta blocker. Hydralazine is associated with drug-induced lupus-like syndrome at higher doses. Minoxidil is associated with hirsutism and requires a loop diuretic. Minoxidil can induce pericardial effusion.
	Minoxidil	5–100	1-3	

*Dosages may vary from those listed in the FDA-approved labeling (available at https://dailymed.nlm.nih.gov/dailymed/) From Chobanian et al JNC 7.[S8.1.4-15]

ACE indicates angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BP, blood pressure; BPH, benign prostatic hyperplasia; CCB, calcium channel blocker; CKD, chronic kidney disease; CNS, central nervous system; CVD, cardiovascular disease; ER, extended release; GFR, glomerular filtration rate; HF, heart failure; HF/EF, heart failure with reduced ejection fraction; IHD, ischemic heart disease; IR, immediate release; LA, long-acting; and SR, sustained release.

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assigned to different BP targets[S8.1.5-3–S8.1.5-5] and in larger meta-analyses that additionally included trials that compared different intensities of treatment.[S8.1.5-12] Data from the most recent meta-analysis (42 trials and 144 220 patients)[S8.1.5-5] demonstrate a linear association between mean achieved SBP and risk of CVD mortality with the lowest risk at 120 to 124 mm Hg. The totality of the available information provides evidence that a lower BP target is generally better than a higher BP target and that some patients will benefit from an SBP treatment goal <120 mm Hg, especially those at high risk of CVD.[S8.1.5-15] The specific inclusion and exclusion criteria of any RCT may limit extrapolation to a more general population with hypertension. In addition, all of the relevant trials have been efficacy studies in which BP measurements were more consistent with guideline recommendations than is common in clinical practice, resulting in lower absolute values for SBP. For both of these reasons, the SBP target recommended during BP lowering (<130 mm Hg) is higher than that which was used in SPRINT.

Recommendation-Specific Supportive Text

1. Meta-analysis and systematic review of trials that compare more intensive BP reduction to standard BP reduction report that more intense BP lowering significantly reduces the risk of stroke, coronary events, major cardiovascular events, and cardiovascular mortality.[S8.1.5-1] In a stratified analysis of these data, achieving an additional 10–mm Hg reduction in SBP reduced CVD risk when compared with an average SBP of 158/82 to 143/76 mm Hg, 144/85 to 137/81 mm Hg, and 134/79 to 125/76 mm Hg. Patients with DM and CKD were included in the analysis.[S8.1.5-1,S8.1.5-2,S8.1.5-11–S8.1.5-13,S8.1.5-18] (Specific management details are in Section 9.3 for CKD and Section 9.6 for DM.)
2. The treatment of patients with hypertension without elevated risk has been systematically understudied because lower-risk groups would require prolonged follow-up to have a sufficient number of clinical events to provide useful information. Although there is clinical trial evidence that both drug and nondrug therapy will interrupt the progressive course of hypertension,[S8.1.5-6] there is no trial evidence that this treatment decreases CVD morbidity and mortality. The clinical trial evidence is strongest for a target BP of 140/90 mm Hg in this population. However, observational studies suggest that these individuals often have a high lifetime risk and would benefit from BP control earlier in life.[S8.1.5-19,S8.1.5-20]

8.1.6. Choice of Initial Medication

Recommendation for Choice of Initial Medication

References that support the recommendation are summarized in Online Data Supplement 27 and Systematic Review Report.

COR	LOE	Recommendation
I	A[SR]	1. For initiation of antihypertensive drug therapy, first-line agents include thiazide diuretics, CCBs, and ACE inhibitors or ARBs.[S8.1.6-1,S8.1.6-2]

SR indicates systematic review.

Synopsis

The overwhelming majority of persons with BP sufficiently elevated to warrant pharmacological therapy may be best treated initially with 2 agents (see Section 8.1.6.1). When initiation of pharmacological therapy with a single medication is appropriate, primary consideration should be given to comorbid conditions (eg, HF, CKD) for which specific classes of BP-lowering medication are indicated (see Section 9).[S8.1.6-1] In the largest head-to-head comparison of first-step drug therapy for hypertension,[S8.1.6-3] the thiazide-type diuretic chlorthalidone was superior to the CCB amlodipine and the ACE inhibitor lisinopril in preventing HF, a BP-related outcome of increasing importance in the growing population of older persons with hypertension.[S8.1.6-4–S8.1.6-7] Additionally, ACE inhibitors were less effective than thiazide diuretics and CCBs in lowering BP and in prevention of stroke. For black patients, ACE inhibitors were also notably less effective than CCBs in preventing HF[S8.1.6-8] and in the prevention of stroke[S8.1.6-9] (see Section 10.1). ARBs may be better tolerated than ACE inhibitors in black patients, with less cough and angioedema, but according to the limited available experience they offer no proven advantage over ACE inhibitors in preventing stroke or CVD in this population, making thiazide diuretics (especially chlorthalidone) or CCBs the best initial choice for single-drug therapy. For stroke, in the general population, beta blockers were less effective than CCBs (36% lower risk) and thiazide diuretics (30% lower risk). CCBs have been shown to be as effective as diuretics for reducing all CVD events other than HF, and CCBs are a good alternative choice for initial therapy when thiazide diuretics are not tolerated. Alpha blockers are not used as first-line therapy for hypertension because they are less effective for prevention of CVD than other first-step agents, such as thiazide diuretics.[S8.1.6-3,S8.1.6-10]

Recommendation-Specific Supportive Text

1. The overall goal of treatment should be reduction in BP, in the context of underlying CVD risk. Five drug classes have been shown, in high-quality RCTs, to prevent CVD as compared with placebo (diuretics, ACE inhibitors, ARBs, CCBs, and beta blockers).[S8.1.6-11,S8.1.6-12] In head-to-head comparisons of first-step therapy, different drug classes have been reported to provide somewhat divergent capacity to prevent specific CVD events. Interpretation of meta-analyses comparing agents from different drug classes is challenging because the relevant RCTs were conducted in different time periods, during which concurrent antihypertensive therapy was less or more common, and the efficacy of agents from certain drug classes may have changed. In recognition of this, some[S8.1.6-2] but not all[S8.1.6-11,S8.1.6-12] meta-analyses, as well as the largest individual RCT that compared first-step agents,[S8.1.6-3] have suggested that diuretics, especially the long-acting thiazide-type agent chlorthalidone, may provide an optimal choice for first-step drug therapy of hypertension. In contrast, some meta-analyses have suggested that beta blockers may be less effective, especially for stroke prevention in older adults, but interpretation is hampered by inclusion of RCTs that used beta blockers that are now considered to be inferior for prevention of CVD.[S8.1.6-13,S8.1.6-14] In a systematic review and network meta-analysis conducted for the present guideline, beta blockers were significantly less effective than diuretics

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for prevention of stroke and cardiovascular events.[S8.1.6-1] Diuretics were also significantly better than CCBs for prevention of HF. There were some other nonsignificant differences between diuretics, ACE inhibitors, ARBs, and CCBs, but the general pattern was for similarity in effect. As indicated in Section 8.1.6.1, most adults with hypertension require >1 drug to control their BP. As recommended in Section 10.1, for black adults with hypertension (without HF or CKD), initial antihypertensive treatment should include a thiazide diuretic or CCB.

8.1.6.1. Choice of Initial Monotherapy Versus Initial Combination Drug Therapy

Recommendations for Choice of Initial Monotherapy Versus Initial Combination Drug Therapy*

COR	LOE	Recommendations
I	C-EO	1. Initiation of antihypertensive drug therapy with 2 first-line agents of different classes, either as separate agents or in a fixed-dose combination, is recommended in adults with stage 2 hypertension and an average BP more than 20/10 mm Hg above their BP target.
IIa	C-EO	2. Initiation of antihypertensive drug therapy with a single antihypertensive drug is reasonable in adults with stage 1 hypertension and BP goal <130/80 mm Hg with dosage titration and sequential addition of other agents to achieve the BP target.

*Fixed-dose combination antihypertensive medications are listed in Online Data Supplement D.

Synopsis

Systematic review of the evidence comparing the initiation of antihypertensive treatment with monotherapy and sequential (stepped-care) titration of additional agents versus initiation of treatment with combination therapy (including fixed-dose combinations) did not identify any RCTs meeting the systematic review questions posed in the PICOTS format (P=population, I=intervention, C=comparator, O=outcome, T=timing, S=setting). However, in both ACCORD and SPRINT, 2-drug therapy was recommended for most participants in the intensive- but not standard-therapy groups.

Recommendation-Specific Supportive Text

1. Because most patients with hypertension require multiple agents for control of their BP and those with higher BPs are at greater risk, more rapid titration of antihypertensive medications began to be recommended in patients with BP >20/10 mm Hg above their target, beginning with the JNC 7 report.[S8.1.6.1-1] In these patients, initiation of antihypertensive therapy with 2 agents is recommended. Evidence favoring this approach comes mostly from studies using fixed-dose combination products showing greater BP lowering with fixed-dose combination agents than with single agents, as well as better adherence to therapy.[S8.1.6.1-2,S8.1.6.1-3] The safety and efficacy of this strategy have been demonstrated in adults to reduce BPs to <140/90 mm Hg though not compared with other strategies.[S8.1.6.1-4–S8.1.6.1-6] In general, this approach is reasonable in older adults, those at high CVD risk, or those who have a history of hypotension or drug-associated side effects. However, caution is advised in initiating antihypertensive pharmacotherapy with 2 drugs in older patients because hypotension or orthostatic hypotension may develop in some patients; BP should be carefully monitored.
2. The stepped-care approach defined by the initiation of antihypertensive drug therapy with a single agent followed by the sequential titration of the dose and addition of other agents has been the recommended treatment strategy since the first report of the National High Blood Pressure Education Program.[S8.1.6.1-7] This approach is also reasonable in older adults or those at risk or who have a history of hypotension or drug-associated side effects. This strategy has been used successfully in nearly all hypertension treatment trials but has not been formally tested against other antihypertensive drug treatment strategies for effectiveness in achieving BP control or in preventing adverse outcomes.

8.2. Achieving BP Control in Individual Patients

Recommendations for lifestyle modifications and drug selection are specified in Sections 6.2, 8.1.4, and 8.1.6. Initial drug selections should be based on trial evidence of treatment efficacy, combined with recognition of compelling indications for use of an agent from a specific drug class, as well as the individual patient's lifestyle preferences and traits. For a subset of patients (25% to 50%),[S8.2-1] the initial drug therapy will be well tolerated and effective in achieving the desired level of BP, with only the need for subsequent monitoring (see Section 8.3 for an appropriate follow-up schedule). For others, the initial drug will not be tolerated or will not be effective, requiring either a change in medication or addition of another medication, followed by BP monitoring.[S8.2-2] Approximately 25% of patients will require additional treatment adjustments. In a minority of this group, achievement of goal BP can be challenging.

In patients who do not respond to or do not tolerate treatment with 2 to 3 medications or medication combinations, additional trials of treatment tend to be ineffective or poorly tolerated. Some patients may become disillusioned and lost to follow-up, whereas others will identify an alternative healthcare provider, including nontraditional healers, or will try popular home remedies. Working with this more demanding subset requires provider expertise, patience, and a mechanism to respond efficiently and sensitively to concerns as they arise. In this setting, team-based care (see Section 12) may be effective, encouraging coupling of nonpharmacological and pharmacological treatments, while improving access to and communication with care providers.

In the setting of medication intolerance, consider allowing a defined period of time to evaluate the effects of lifestyle modification in patients with a relatively low CVD risk (10-year risk of ASCVD <10%, based on the ASCVD Risk Estimator [http://tools.acc.org/ASCVD-Risk-Estimator]), with scheduled follow-up visits for assessment of BP levels, including a review of HBPM data, and an appraisal of lifestyle change goal achievements. For patients with a higher level of CVD risk or with significant elevations in BP (SBP or DBP >20 or >10 mm Hg above target, respectively), medication is usually

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started even while the patient is pursuing lifestyle change (see Section 8.1.2).

Consideration of patient comorbidities, lifestyle, and preferences may suggest better tolerance or greater effect from one class of medication versus other classes. For example, if hyponatremia is present, it would be important to avoid or stop thiazide diuretic therapy. In this case, a loop diuretic should be used if a diuretic is required. If hypokalemia is present, primary or secondary aldosteronism should be excluded, after which one should consider a potassium-sparing agent, such as spironolactone, eplerenone, triamterene, or amiloride. In addition, reducing dietary sodium intake will diminish urinary potassium losses. If the patient has chronic cough or a history of ACE inhibitor–induced cough or develops a cough or bronchial responsiveness while on an ACE inhibitor, one should use an ARB in place of an ACE inhibitor. For patients with bronchospastic lung disease, a beta-1-selective blocker (eg, bisoprolol, metoprolol) should be considered if beta-blocker therapy is required. A patient who is already adherent to lifestyle change recommendations, including diligent reduction in sodium intake, may show a greater response to a RAS blocker. Prior patient experience should be considered, as in the case of cough associated with prior use of an ACE inhibitor, which is likely to reoccur if an agent from the same class is prescribed.

8.3. Follow-Up of BP During Antihypertensive Drug Therapy

Appropriate follow-up and monitoring enable assessment of adherence (see Section 12.1) and response to therapy, help identify adverse responses to therapy and target organ damage, and allow assessment of progress toward treatment goals. High-quality RCTs have successfully and safely developed strategies for follow-up, monitoring, and reassessment from which recommendations can be made (Figure 4).[S8.2-1,S8.2-2] A systematic approach to out-of-office BP assessment is an essential part of follow-up and monitoring of BP, to assess response to therapy; check for evidence of white coat hypertension, white coat effect, masked hypertension, or masked uncontrolled hypertension; and help achieve BP targets (see Sections 4 and 12).

8.3.1. Follow-Up After Initiating Antihypertensive Drug Therapy

Recommendation for Follow-Up After Initiating Antihypertensive Drug Therapy

References that support the recommendation are summarized in Online Data Supplement 28.

COR	LOE	Recommendation
I	B-R	1. Adults initiating a new or adjusted drug regimen for hypertension should have a follow-up evaluation of adherence and response to treatment at monthly intervals until control is achieved.[S8.3.1-1–S8.3.1-3]

Recommendation-Specific Supportive Text

1. Components of the follow-up evaluation should include assessment of BP control, as well as evaluation for orthostatic hypotension, adverse effects from medication therapy, adherence to medication and lifestyle therapy, need for adjustment of medication dosage, laboratory testing (including electrolyte and renal function status), and other assessments of target organ damage.[S8.3.1-1–S8.3.1-3]

8.3.2. Monitoring Strategies to Improve Control of BP in Patients on Drug Therapy for High BP

Recommendation for Monitoring Strategies to Improve Control of BP in Patients on Drug Therapy for High BP

References that support the recommendation are summarized in Online Data Supplement 29.

COR	LOE	Recommendation
I	A	1. Follow-up and monitoring after initiation of drug therapy for hypertension control should include systematic strategies to help improve BP, including use of HBPM, team-based care, and telehealth strategies.[S8.3.2-1–S8.3.2-6]

Recommendation-Specific Supportive Text

1. Systematic approaches to follow-up have been shown to improve hypertension control and can be adapted and incorporated into clinical practices according to local needs and resource availability (see Section 8.3.1 for time intervals for treatment follow-up and monitoring and Sections 12.2 and 12.3.2 on systematic strategies to improve BP control).

9. Hypertension in Patients With Comorbidities

Certain comorbidities may affect clinical decision-making in hypertension. These include ischemic heart disease, HF with reduced ejection fraction (HF*r*EF), HF*p*EF, CKD (including renal transplantation), cerebrovascular disease, AF, PAD, DM, and metabolic syndrome.[S9-1] As noted in Section 8.1.2, this guideline generally recommends use of BP-lowering medications for secondary prevention of CVD in patients with clinical CVD (CHD, HF, and stroke) and an average BP ≥130/80 mm Hg and for primary prevention of CVD in adults with an estimated 10-year ASCVD risk of ≥10% and an average SBP ≥130 mm Hg or an average DBP ≥80 mm Hg. Although we recommend use of the ACC/AHA Pooled Cohort Equations (http://tools.acc.org/ASCVD-Risk-Estimator/) to estimate 10-year risk of ASCVD to establish the BP threshold for treatment, the vast majority of adults with a co-morbidity are likely to have a 10-year risk of ASCVD that exceeds 10%. In some instances, clinical trial confirmation of treatment in patients with comorbidities is limited to a target BP of 140/90 mm Hg. In addition, the selection of medications for use in treating high BP in patients with CVD is guided by their use for other compelling indications (eg, beta blockers after MI, ACE inhibitors for HF*r*EF), as discussed in specific guidelines for the clinical condition.[S9-2–S9-4] The present guideline does not address the recommendations for treatment of hypertension occurring with acute coronary syndromes.

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9.1. Stable Ischemic Heart Disease

Recommendations for Treatment of Hypertension in Patients With Stable Ischemic Heart Disease (SIHD)

References that support recommendations are summarized in Online Data Supplements 30-32.

COR	LOE	Recommendations
I	SBP: B-R DBP: C-EO	1. In adults with SIHD and hypertension, a BP target of less than 130/80 mm Hg is recommended.[S9.1-1–S9.1-5]
I	SBP: B-R DBP: C-EO	2. Adults with SIHD and hypertension (BP ≥130/80 mm Hg) should be treated with medications (eg, GDMT[S9.1-6] beta blockers, ACE inhibitors, or ARBs) for compelling indications (eg, previous MI, stable angina) as first-line therapy, with the addition of other drugs (eg, dihydropyridine CCBs, thiazide diuretics, and/or mineralocorticoid receptor antagonists) as needed to further control hypertension.[S9.1-7–S9.1-10]
I	B-NR	3. In adults with SIHD with angina and persistent uncontrolled hypertension, the addition of dihydropyridine CCBs to GDMT[S9.1-6] beta blockers is recommended.[S9.1-8,S9.1-11,S9.1-12]
IIa	B-NR	4. In adults who have had a MI or acute coronary syndrome, it is reasonable to continue GDMT (S9.1-6) beta blockers beyond 3 years as long-term therapy for hypertension.[S9.1-13,S9.1-14]
IIb	C-EO	5. Beta blockers and/or CCBs might be considered to control hypertension in patients with CAD (without HF/EF) who had an MI more than 3 years ago and have angina.

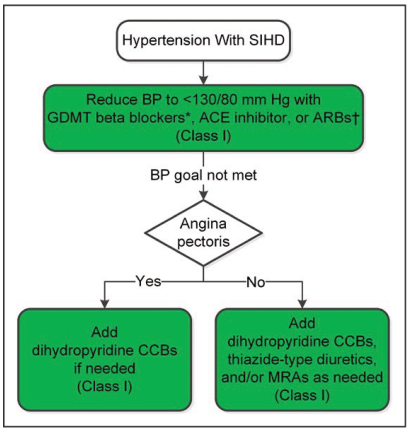

Figure 5. Management of hypertension in patients with SIHD. Colors correspond to Class of Recommendation in Table 1. *GDMT beta blockers for BP control or relief of angina include carvedilol, metoprolol tartrate, metoprolol succinate, nadolol, bisoprolol, propranolol, and timolol. Avoid beta blockers with intrinsic sympathomimetic activity. The beta blocker atenolol should not be used because it is less effective than placebo in reducing cardiovascular events. †If needed for BP control. ACE indicates angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BP, blood pressure; CCB, calcium channel blocker; GDMT, guideline-directed management and therapy; and SIHD, stable ischemic heart disease.

Synopsis

Hypertension is a major risk factor for ischemic heart disease. Numerous RCTs have demonstrated the benefits of antihypertensive drug therapy in reducing the risk of ischemic heart disease. The following recommendations apply only to management of hypertension in patients with SIHD without HF. See Section 9.2 for recommendations for the treatment of patients with SIHD and HF.

Figure 5 is an algorithm on management of hypertension in patients with SIHD.

Recommendation-Specific Supportive Text

1. In patients with increased cardiovascular risk, reduction of SBP to <130/80 mm Hg has been shown to reduce CVD complications by 25% and all-cause mortality by 27%.[S9.1-1]
2. After 5 years of randomized therapy in high-CVD-risk patients, ramipril produced a 22% reduction in MI, stroke, or CVD compared with placebo.[S9.1-10] No added benefit on CVD outcomes was seen when compared with CCBs and diuretics.[S9.1-15,S9.1-16] After 4.2 years of randomized therapy in patients with SIHD, perindopril reduced CVD death, MI, or cardiac arrest by 20% compared with placebo.[S9.1-7] Beta blockers are effective drugs for preventing angina pectoris, improving exercise time until the onset of angina pectoris, reducing exercise-induced ischemic ST-segment depression, and preventing coronary events.[S9.1-8,S9.1-17–S9.1-22] Because of their compelling indications for treatment of SIHD, these drugs are recommended as a first-line therapy in the treatment of hypertension when it occurs in patients with SIHD. GDMT beta blockers for SIHD that are also effective in lowering BP include carvedilol, metoprolol tartrate, metoprolol succinate, nadolol, bisoprolol, propranolol, and timolol. Atenolol is not as effective as other antihypertensive drugs in the treatment of hypertension.[S9.1-23]
3. Dihydropyridine CCBs are effective antianginal drugs that can lower BP and relieve angina pectoris when added to beta blockers in patients in whom hypertension is present and angina pectoris persists despite beta-blocker therapy.[S9.1-8,S9.1-17,S9.1-19–S9.1-22,S9.1-24,S9.1-25] GDMT beta blockers for SIHD that are also effective in lowering BP include carvedilol, metoprolol tartrate, metoprolol succinate, nadolol, bisoprolol, propranolol, and timolol.
4. In randomized long-term trials, use of beta blockers after MI reduced all-cause mortality by 23%.[S9.1-13] Given the established efficacy of beta blockers for treating hypertension and SIHD, their use for treatment continuing beyond 3 years after MI is reasonable.[S9.1-6,S9.1-25]
5. GDMT beta blockers and CCBs are effective antihypertensive and antianginal agents. CCBs include dihydropyridine and nondihydropyridine agents. CCBs can be

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used separately or together with beta blockers beginning 3 years after MI in patients with CAD who have both hypertension and angina.

9.2. Heart Failure

Recommendation for Prevention of HF in Adults With Hypertension

References that support the recommendation are summarized in Online Data Supplement 33.

COR	LOE	Recommendation
I	SBP: B-R DBP: C-EO	1. In adults at increased risk of HF, the optimal BP in those with hypertension should be less than 130/80 mm Hg.[S9.2-1–S9.2-3]

Synopsis

Antecedent hypertension is present in 75% of patients with chronic HF.[S9.2-4] In the Cardiovascular Health Study[S9.2-5] and the Health, Aging and Body Composition Study,[S9.2-6] 11.2% of 4408 persons (53.1% women, with a mean age of 72.8 years, living in the community, and not receiving antihypertensive drugs at baseline) developed HF over 10 years.[S9.2-7] Compared with those with an average SBP <120 mm Hg, the adjusted incidence of HF was increased 1.6, 2.2, and 2.6 times in those with average SBPs between 120 and 139 mm Hg, between 140 and 159 mm Hg, and ≥160 mm Hg, respectively.[S9.2-7]

No RCTs are available that compare one BP-lowering agent to another for the management of patients with HF. The following recommendations for treatment of hypertension in HF are based on use of drugs that lower BP and also have compelling indications for management of HF (with HF*r*EF or HF*p*EF) as recommended in current ACC/AHA guidelines.[S9.2-4,S9.2-8]

Recommendation-Specific Supportive Text

1. In adults with hypertension (SBP ≥130 mm Hg or DBP ≥80 mm Hg) and a high risk of CVD, a strong body of evidence supports treatment with antihypertensive medications (see Section 8.1.2) and more-intensive rather than less-intensive intervention (see Section 8.1.5). In SPRINT, a more intensive intervention that targeted an SBP <120 mm Hg significantly reduced the primary outcome (CVD composite) by about 25%.[S9.2-9] The incidence of HF, a component of the primary outcome, was also substantially decreased (hazard ratio: 0.62; 95% confidence interval: 0.45–0.84). Meta-analyses of clinical trials have identified a similar beneficial effect of more-intensive BP reduction on the incidence of HF,[S9.2-2,S9.2-10] but the body of information from studies confined to trials that randomly assigned participants to different BP targets is more limited and less compelling.[S9.2-3] In addition, the available trials were efficacy studies in which BP measurements were more consistent with guideline recommendations than is common in clinical practice, resulting in lower absolute values for SBP. For both of these reasons, the SBP target recommended during BP lowering (<130 mm Hg) is higher than that used in SPRINT.

9.2.1. Heart Failure With Reduced Ejection Fraction

Recommendations for Treatment of Hypertension in Patients With HF*r*EF

References that support recommendations are summarized in Online Data Supplement 34.

COR	LOE	Recommendations
I	C-EO	1. Adults with HF*r*EF and hypertension should be prescribed GDMT[S9.2.1-2] titrated to attain a BP of less than 130/80 mm Hg.
III: No Benefit	B-R	2. Nondihydropyridine CCBs are not recommended in the treatment of hypertension in adults with HF*r*EF.[S9.2.1-1]

Synopsis

Approximately 50% of patients with HF have HF*r*EF.[S9.2.1-2–S9.2.1-6] Numerous RCTs have shown that treatment of HF*r*EF with GDMT reduces mortality and HF hospitalizations.[S9.2.1-7] Large-scale RCTs have shown that antihypertensive drug therapy reduces the incidence of HF in patients with hypertension.[S9.2.1-8–S9.2.1-11] In ALLHAT (Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial), chlorthalidone reduced the risk of HF*r*EF more than amlodipine and doxazosin but similarly to lisinopril.[S9.2.1-12,S9.2.1-13]

Recommendation-Specific Supportive Text

1. This recommendation is based on guidance in the 2017 ACC/AHA/HFSA guideline focused update on heart failure[S9.2.1-14] (see figure from the HF focused update that is reproduced in Online Data Supplement A). Lifestyle modification, such as weight loss and sodium reduction, may serve as adjunctive measures to help these agents work better. No RCT evidence is available to support the superiority of one BP-lowering medication with compelling indications for treatment of HF*r*EF over another. Medications with compelling indications for HF that may be used as first-line therapy to treat high BP include ACE inhibitors or ARBs, angiotensin receptor–neprilysin inhibitors, mineralocorticoid receptor antagonists, diuretics, and GDMT beta blockers (carvedilol, metoprolol succinate, or bisoprolol).

   Clinical trials evaluating goal BP reduction and optimal BP-lowering agents in the setting of HF*r*EF and concomitant hypertension have not been performed. However, in patients at higher CVD risk, BP lowering is associated with fewer adverse cardiovascular events.[S9.2.1-7] GDMT for HF*r*EF with agents known to lower BP should consider a goal BP reduction consistent with a threshold now associated with improved clinical outcomes but not yet proven by RCTs in an HF population.
2. Nondihydropyridine CCBs (verapamil, diltiazem) have myocardial depressant activity. Several clinical trials have demonstrated either no clinical benefit or even worse outcomes in patients with HF treated with these drugs.[S9.2.1-1] Therefore, nondihydropyridine CCBs are not recommended in patients with hypertension and HF*r*EF.

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9.2.2. Heart Failure With Preserved Ejection Fraction

Recommendations for Treatment of Hypertension in Patients With HF*p*EF

References that support recommendations are summarized in Online Data Supplements 35 and 36.

COR	LOE	Recommendations
I	C-EO	1. In adults with HF*p*EF who present with symptoms of volume overload, diuretics should be prescribed to control hypertension.
I	C-LD	2. Adults with HF*p*EF and persistent hypertension after management of volume overload should be prescribed ACE inhibitors or ARBs and beta blockers titrated to attain SBP of less than 130 mm Hg.[S9.2.2-1–S9.2.2-6]

Synopsis

Approximately 50% of patients with HF have HF*p*EF.[S9.2.2-2,S9.2.2-7–S9.2.2-11] The ejection fraction in these studies has varied from >40% to ≥55%.[S9.2.2-2] Patients with HF*p*EF are usually older women with a history of hypertension. Obesity, CHD, DM, AF, and hyperlipidemia are also highly prevalent in patients with HF*p*EF.[S9.2.2-2,S9.2.2-11,S9.2.2-12] Hypertension is the most important cause of HF*p*EF, with a prevalence of 60% to 89% in large RCTs, epidemiological studies, and HF registries.[S9.2.2-2,S9.2.2-13] Patients with HF*p*EF also have an exaggerated hypertensive response to exercise.[S9.2.2-14] Hypertensive acute pulmonary edema is an expression of HF*p*EF.[S9.2.2-15]

BP control is important for prevention of HF*p*EF in patients with hypertension.[S9.2.2-2,S9.2.2-16–S9.2.2-19] ALLHAT showed that treatment of hypertension with chlorthalidone reduced the risk of HF compared with amlodipine, doxazosin, and lisinopril.[S9.2.2-19,S9.2.2-20] Improved BP control also reduces hospitalization, CVD events, and mortality.[S9.2.2-2,S9.2.2-16–S9.2.2-19]

Recommendation-Specific Supportive Text

1. Diuretics are the only drugs used for the treatment of hypertension and HF that can adequately control the fluid retention of HF. Appropriate use of diuretics is also crucial to the success of other drugs used for the treatment of hypertension in the presence of HF. The use of inappropriately low doses of diuretics can result in fluid retention. Conversely, the use of inappropriately high doses of diuretics can lead to volume contraction, which can increase the risk of hypotension and renal insufficiency. Diuretics should be prescribed to all patients with hypertension and HF*p*EF who have evidence of, and to most patients with a prior history of, fluid retention.
2. In a trial of patients with HF*p*EF and MI, patients randomized to propranolol had at 32-month follow-up a 35% reduction in mortality rate.[S9.2.2-3] After 21 months of treatment in patients with HF*r*EF and HF*p*EF, compared with placebo, those randomized to nebivolol had a 14% reduction in mortality or CVD hospitalization if they had HF*r*EF and a 19% reduction if they had HF*p*EF.[S9.2.2-4] In patients with HF*p*EF, the primary outcome (a composite of CVD death or HF hospitalization) was observed in 22% for candesartan and 24% for placebo (11% reduction), but fewer patients receiving candesartan were hospitalized for HF.[S9.2.2-5] The use of nitrates in the setting of HF*p*EF is associated with a signal of harm and in most situations should be avoided. For many other common antihypertensive agents, including alpha blockers, beta blockers, and calcium channel blockers, limited data exist to guide the choice of antihypertensive therapy in the setting of HF*p*EF.[S9.2.2-21] Renin-angiotensin-aldosterone system inhibition, however, with ACE inhibitor or ARB and especially MRA would represent the preferred choice. A shared decision-making discussion, with the patient influenced by clinician judgment, should drive the ultimate choice of antihypertensive agents.

9.3. Chronic Kidney Disease

Recommendations for Treatment of Hypertension in Patients With CKD

References that support recommendations are summarized in Online Data Supplements 37 and 38 and Systematic Review Report.

COR	LOE	Recommendations
I	SBP: B-R[SR] DBP: C-EO	1. Adults with hypertension and CKD should be treated to a BP goal of less than 130/80 mm Hg.[S9.3-1–S9.3-6]
IIa	B-R	2. In adults with hypertension and CKD (stage 3 or higher or stage 1 or 2 with albuminuria [≥300 mg/d, or ≥300 mg/g albumin-to-creatinine ratio or the equivalent in the first morning void]), treatment with an ACE inhibitor is reasonable to slow kidney disease progression.[S9.3-3,S9.3-7–S9.3-12]
IIb	C-EO	3. In adults with hypertension and CKD (stage 3 or higher or stage 1 or 2 with albuminuria [≥300 mg/d, or ≥300 mg/g albumin-to-creatinine ratio in the first morning void]) (S9.3-7,S9.3-8), treatment with an ARB may be reasonable if an ACE inhibitor is not tolerated.

SR indicates systematic review.

Synopsis

Refer to the "Systematic Review for the 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults" for the complete systematic evidence review for additional data and analyses.[S9.3-13] Hypertension is the most common comorbidity affecting patients with CKD. Hypertension has been reported in 67% to 92% of patients with CKD, with increasing prevalence as kidney function declines.[S9.3-14] Hypertension may occur as a result of kidney disease, yet the presence of hypertension may also accelerate further kidney injury; therefore, treatment is an important means to prevent further kidney functional decline. This tight interaction has led to extensive debate about the optimal BP target for patients with CKD.[S9.3-15–S9.3-18] Masked hypertension may occur in up to 30% of patients with CKD and portends higher risk of CKD

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progression.[S9.3-19–S9.3-23] CKD is an important risk factor for CVD,[S9.3-24] and the coexistence of hypertension and CKD further increases the risk of adverse CVD and cerebrovascular events, particularly when proteinuria is present.[S9.3-25] Even as the importance of hypertension treatment is widely accepted, data supporting BP targets in CKD are limited, as patients with CKD were historically excluded from clinical trials. Furthermore, CKD is not included in the CVD risk calculations used to determine suitability for most clinical trials.[S9.3-26–S9.3-28]

Until publication of the SPRINT results, most guidelines for BP targets in patients with CKD favored treatment to a BP <140/90 mm Hg,[S9.3-15] with consideration of the lower target of <130/80 mm Hg for those with more severe proteinuria (≥300 mg albuminuria in 24 hours or the equivalent), if tolerated.[S9.3-16–S9.3-18] Patients with stage 3 to 4 CKD (eGFR of 20 to <60 mL/minute/1.73 m^2) comprised 28% of the SPRINT study population, and in this group intensive BP management seemed to provide the same benefits for reduction in the CVD composite primary outcome and all-cause mortality as were seen in the full study cohort. Given that most patients with CKD die from CVD complications, this RCT evidence supports a lower target of <130/80 mm Hg for all patients with CKD (Figure 6). It is appropriate to acknowledge that many patients with CKD have additional comorbidities and evidence of frailty that caused them to be excluded from past clinical trials. Observational studies of CKD cohorts indicate a higher risk of mortality at lower systolic pressures and a flat relationship of SBP to event risk in elderly patients with CKD,[S9.3-29,S9.3-30] which supports concerns that these complex patients may be at greater risk of complications from intensive BP treatment and may fail to achieve benefits from lower BP targets. In contrast, in the prespecified subgroup analysis of the elderly cohort in SPRINT, frail elderly patients did sustain benefit from the lower BP target, which supports a lower goal for all patients, including those with CKD.[S9.3-31] In this setting, incremental BP reduction may be appropriate, with careful monitoring of physical and kidney function.

An ACE inhibitor (or an ARB, in case of ACE inhibitor intolerance) is a preferred drug for treatment of hypertension if albuminuria (≥300 mg/day or ≥300 mg/g creatinine by first morning void) is present, although the evidence is mixed[S9.3-10,S9.3-11] (Figure 6). In the course of reducing intraglomerular pressure and thereby reducing albuminuria, serum creatinine may increase up to 30% because of concurrent reduction in GFR.[S9.3-32] Further GFR decline should be investigated and may be related to other factors, including volume contraction, use of nephrotoxic agents, or renovascular disease.[S9.3-33] The combination of an ACE inhibitor and an ARB should be avoided because of reported harms demonstrated in several large cardiology trials[S9.3-34,S9.3-35] and in 1 diabetic nephropathy trial.[S9.3-36] Because of the greater risk of hyperkalemia and hypotension and lack of demonstrated benefit, the combination of an ARB (or ACE inhibitor) and a direct renin inhibitor is also contraindicated during management of patients with CKD.[S9.3-37]

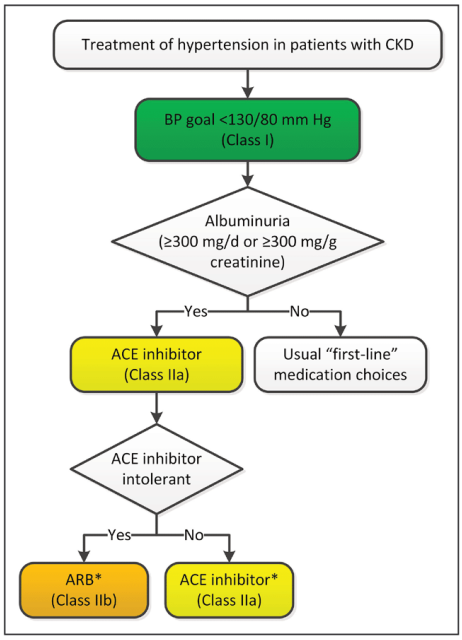

Figure 6. Management of hypertension in patients with CKD. Colors correspond to Class of Recommendation in Table 1. *CKD stage 3 or higher or stage 1 or 2 with albuminuria ≥300 mg/d or ≥300 mg/g creatinine. ACE indicates angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BP blood pressure; and CKD, chronic kidney disease.

Figure 6 is an algorithm on management of hypertension in patients with CKD.

Recommendation-Specific Supportive Text

1. We recommend ASCVD risk assessment in all adults with hypertension, including those with CKD. As a matter of convenience, however, it can be assumed that the vast majority of patients with CKD have a 10-year ASCVD risk ≥10%, placing them in the high risk category that requires initiation of antihypertensive drug therapy at BP ≥130/80 mm Hg (see Section 8.1.2, Figure 4 and Table 23 for BP thresholds for initiating antihypertensive drug treatment). In SPRINT, the participants with CKD who were randomized to intensive antihypertensive therapy (SBP target <120 mm Hg) appeared to derive the same beneficial reduction in CVD events and all-cause mortality that was seen in their counterparts without CKD at baseline. Likewise, intensive therapy was beneficial even in those ≥75 years of age with frailty or the slowest gait speed. There was no difference in the principal kidney outcome (≥50% decline in eGFR or ESRD) between the intensive-and standard-therapy (SBP target <140 mm Hg) groups.[S9.3-26] Three other RCTs[S9.3-1–S9.3-3] have evaluated the effect

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of differing BP goals of <140/90 mm Hg versus 125–130/75–80 mm Hg on CKD progression in patients with CKD. None of these trials demonstrated a benefit for more intensive BP reduction, although post hoc follow-up analyses favored the lower targets in patients with more severe proteinuria,[S9.3-38,S9.3-39] and these trials were underpowered to detect differences in CVD event rates. Recent meta-analyses and systematic reviews that included patients with CKD from SPRINT support more intensive BP treatment[S9.3-40–S9.3-42] to reduce cardiovascular events but do not demonstrate a reduction in the rate of progression of kidney disease (doubling of serum creatinine or reaching ESRD). More intensive BP treatment may result in a modest reduction in GFR, which is thought to be primarily due to a hemodynamic effect and may be reversible. Electrolyte abnormalities are also more likely during intensive BP treatment. More intensive BP lowering in patients with CKD is also supported by a BP Lowering Treatment Trialists' Collaboration meta-analysis of RCTs in patients with CKD.[S9.3-43]

2. Evidence comes from AASK (The African American Study of Kidney Disease and Hypertension), 2 small trials (1 positive, 1 negative), and a meta-analysis.[S9.3-3,S9.3-6,S9.3-10,S9.3-11] Albuminuria is quantified by 24-hour urine collection. A 10% to 25% increase in serum creatinine may occur in some patients with CKD as a result of ACE inhibitor therapy.
3. ARBs were shown to be noninferior to ACE inhibitors in clinical trials in the non-CKD population.[S9.3-35] A 10% to 25% increase in serum creatinine may occur in some patients with CKD as a result of ARB therapy.

9.3.1. Hypertension After Renal Transplantation

Recommendations for Treatment of Hypertension After Renal Transplantation

References that support recommendations are summarized in Online Data Supplements 39 and 40.

COR	LOE	Recommendations
IIa	SBP: B-NR DBP: C-EO	1. After kidney transplantation, it is reasonable to treat patients with hypertension to a BP goal of less than 130/80 mm Hg.[S9.3.1-1]
IIa	B-R	2. After kidney transplantation, it is reasonable to treat patients with hypertension with a calcium antagonist on the basis of improved GFR and kidney survival.[S9.3.1-2]

Synopsis

After kidney transplantation, hypertension is common because of preexisting kidney disease, the effects of immunosuppressive medications, and the presence of allograft pathology.[S9.3.1-3] Transplant recipients frequently harbor multiple CVD risk factors and are at high risk of CVD events. Hypertension may accelerate target organ damage and kidney function decline, particularly when proteinuria is present.[S9.3.1-4–S9.3.1-6]

Use of calcineurin inhibitor–based immunosuppression regimens after transplantation is associated with a high (70% to 90%) prevalence of hypertension.[S9.3.1-7] Hypertension is less common when calcineurin inhibitors have been used without corticosteroids in liver transplantation patients,[S9.3.1-8] although prevalence rates have not differed in steroid minimization trials after kidney transplantation.[S9.3.1-9,S9.3.1-10] Reports from long-term belatacept-based immunosuppression studies indicate higher GFR and preservation of kidney function. However, hypertension was still present in the majority of patients, although fewer agents were needed to achieve BP goals.[S9.3.1-11] Severity of hypertension and intensity of treatment may differ somewhat depending on the type of organ transplanted; however, most concepts relevant to kidney transplant recipients will apply to the other solid organ recipients as well.

BP targets change over time after transplantation. Initially, it is important to maintain ample organ perfusion with less stringent BP targets (<160/90 mm Hg) to avoid hypotension and risk of graft thrombosis. Beyond the first month, BP should be controlled to prevent target organ damage as in the nontransplantation setting.[S9.3.1-12,S9.3.1-13] Hypertension after transplantation is often associated with altered circadian BP rhythm with loss of the normal nocturnal BP fall[S9.3.1-14,S9.3.1-15] and, in some, a nocturnal BP rise. These changes may return to normal after a longer period of follow-up.[S9.3.1-16]

Recommendation-Specific Supportive Text

1. Although treatment targets for hypertension after transplantation should probably be similar to those for other patients with CKD, there are no trials in post-transplantation patients comparing different BP targets. As kidney transplant recipients generally have a single functioning kidney and CKD, BP targets should be similar to those for the general CKD population.
2. Limited studies have compared drug choice for initial antihypertensive therapy in patients after kidney transplantation. On the basis of a Cochrane analysis,[S9.3.1-2] most studies favor CCBs to reduce graft loss and maintain higher GFR, with some evidence suggesting potential harm from ACE inhibitors because of anemia, hyperkalemia, and lower GFR. In recognition of this concern, RAS inhibitors may be reserved for the subset of patients with hypertension and additional comorbidities that support the need for ACE inhibitor therapy (ie, proteinuria or HF after transplantation). With appropriate potassium and creatinine monitoring, this has been demonstrated to be safe.[S9.3.1-17]

9.4. Cerebrovascular Disease

Stroke is a leading cause of death, disability, and dementia.[S9.4-1] Because of its heterogeneous causes and hemodynamic consequences, the management of BP in adults with stroke is complex and challenging.[S9.4-2] To accommodate the variety of important issues pertaining to BP management in the stroke patient, treatment recommendations require recognition of stroke acuity, stroke type, and therapeutic objectives. Future studies should target more narrowly defined questions, such as optimal BP-reduction timing and target, as well as ideal antihypertensive agent therapeutic class by patient type and event type.

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9.4.1. Acute Intracerebral Hemorrhage

Recommendations for Management of Hypertension in Patients With Acute Intracerebral Hemorrhage (ICH)

References that support recommendations are summarized in Online Data Supplement 41.

COR	LOE	Recommendations
IIa	C-EO	1. In adults with ICH who present with SBP greater than 220 mm Hg, it is reasonable to use continuous intravenous drug infusion (Table 19) and close BP monitoring to lower SBP.
III: Harm	A	2. Immediate lowering of SBP (Table 19) to less than 140 mm Hg in adults with spontaneous ICH who present within 6 hours of the acute event and have an SBP between 150 mm Hg and 220 mm Hg is not of benefit to reduce death or severe disability and can be potentially harmful.[S9.4.1-1,S9.4.1-2]

Synopsis

Spontaneous, nontraumatic ICH is a significant global cause of morbidity and mortality.[S9.4.1-3] Elevated BP is highly prevalent in the setting of acute ICH and is linked to greater hematoma expansion, neurological worsening, and death and dependency after ICH.

Figure 7 is an algorithm on management of hypertension in patients with acute ICH.

Recommendation-Specific Supportive Text

1. Information about the safety and effectiveness of early intensive BP-lowering treatment is least well established for patients with markedly elevated BP (sustained SBP >220 mm Hg) on presentation, patients with large and severe ICH, or patients requiring surgical decompression. However, given the consistent nature of the data linking high BP with poor clinical outcomes[S9.4.1-4–S9.4.1-6] and some suggestive data for treatment in patients with modestly high initial SBP levels,[S9.4.1-1,S9.4.1-7] early lowering of SBP in ICH patients with markedly high SBP levels (>220 mm Hg) might be sensible. A secondary endpoint in 1 RCT and an overview of data from 4 RCTs indicate that intensive BP reduction, versus BP-lowering guideline treatment, is associated with greater functional recovery at 3 months.[S9.4.1-1,S9.4.1-7]
2. RCT data have suggested that immediate BP lowering (to <140/90 mm Hg) within 6 hours of an acute ICH was feasible and safe,[S9.4.1-1,S9.4.1-8,S9.4.1-9] may be linked to greater attenuation of absolute hematoma growth at 24 hours,[S9.4.1-7] and might be associated with modestly better functional recovery in survivors.[S9.4.1-1,S9.4.1-7] However, a recent RCT[S9.4.1-2] that examined immediate BP lowering within 4.5 hours of an acute ICH found that treatment to achieve a target SBP of 110 to 139 mm Hg did not lead to a lower rate of death or disability than standard reduction to a target of 140 to 179 mm Hg. Moreover, there were significantly more renal adverse events within 7 days after randomization in the intensive-treatment group than in the standard-treatment group.[S9.4.1-2] Put together, neither of the 2 key trials[S9.4.1-1,S9.4.1-2] evaluating the effect of lowering SBP in the acute period after spontaneous ICH met their primary outcomes of reducing death and severe disability at 3 months.

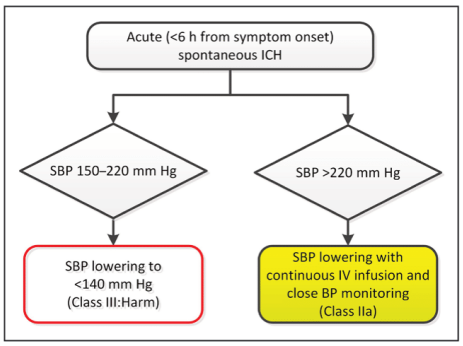

Figure 7. Management of hypertension in patients with acute ICH. Colors correspond to Class of Recommendation in Table 1. BP indicates blood pressure; ICH, intracerebral hemorrhage; IV, intravenous; and SBP, systolic blood pressure.

9.4.2. Acute Ischemic Stroke

Recommendations for Management of Hypertension in Patients With Acute Ischemic Stroke

References that support recommendations are summarized in Online Data Supplement 42.

COR	LOE	Recommendations
I	B-NR	1. Adults with acute ischemic stroke and elevated BP who are eligible for treatment with intravenous tissue plasminogen activator should have their BP slowly lowered to less than 185/110 mm Hg before thrombolytic therapy is initiated.[S9.4.2-1,S9.4.2-2]
I	B-NR	2. In adults with an acute ischemic stroke, BP should be less than 185/110 mm Hg before administration of intravenous tissue plasminogen activator and should be maintained below 180/105 mm Hg for at least the first 24 hours after initiating drug therapy.[S9.4.2-3]
IIa	B-NR	3. Starting or restarting antihypertensive therapy during hospitalization in patients with BP greater than 140/90 mm Hg who are neurologically stable is safe and reasonable to improve long-term BP control, unless contraindicated.[S9.4.2-4,S9.4.2-5]
IIb	C-EO	4. In patients with BP of 220/120 mm Hg or higher who did not receive intravenous alteplase or endovascular treatment and have no comorbid conditions requiring acute antihypertensive treatment, the benefit of initiating or reinitiating treatment of hypertension within the first 48 to 72 hours is uncertain. It might be reasonable to lower BP by 15% during the first 24 hours after onset of stroke.

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Recommendations for Management of Hypertension in Patients With Acute Ischemic Stroke (Continued)		
COR	LOE	Recommendations
III: No Benefit	A	5. In patients with BP less than 220/120 mm Hg who did not receive intravenous thrombolysis or endovascular treatment and do not have a comorbid condition requiring acute antihypertensive treatment, initiating or reinitiating treatment of hypertension within the first 48 to 72 hours after an acute ischemic stroke is not effective to prevent death or dependency.[S9.4.2-4–S9.4.2-9]

Synopsis

Elevated BP is common during acute ischemic stroke (occurring in up to 80% of patients), especially among patients with a history of hypertension.[S9.4.2-10] However, BP often decreases spontaneously during the acute phase of ischemic stroke, as soon as 90 minutes after the onset of symptoms. Countervailing theoretical concerns about arterial hypertension during acute ischemic stroke include aiming to enhance cerebral perfusion of the ischemic tissue while minimizing the exacerbation of brain edema and hemorrhagic transformation of the ischemic tissue.[S9.4.2-11,S9.4.2-12] Some studies have shown a U-shaped relationship between the admission BP and favorable clinical outcomes, with an optimal SBP and DBP ranging from 121 to 200 mm Hg and 81 to 110 mm Hg, respectively.[S9.4.2-13] It is conceivable that an optimal arterial BP range exists during acute ischemic stroke on an individual basis, contingent on the ischemic stroke subtype and other patient-specific comorbidities. Early initiation or resumption of antihypertensive treatment after acute ischemic stroke is indicated only in specific situations: 1) patients treated with tissue-type plasminogen activator,[S9.4.2-1,S9.4.2-2] and 2) patients with SBP >220 mm Hg or DBP >120 mm Hg. For the latter group, it should be kept in mind that cerebral autoregulation in the ischemic penumbra of the stroke is grossly abnormal and that systemic perfusion pressure is needed for blood flow and oxygen delivery. Rapid reduction of BP, even to lower levels within the hypertensive range, can be detrimental. For all other acute ischemic stroke patients, the advantage of lowering BP early to reduce death and dependency is uncertain,[S9.4.2-4–S9.4.2-9] but restarting antihypertensive therapy to improve long-term BP control is reasonable after the first 24 hours for patients who have preexisting hypertension and are neurologically stable.[S9.4.2-4,S9.4.2-5,S9.4.2-14]

Figure 8 is an algorithm on management of hypertension in patients with acute ischemic stroke.

Recommendation-Specific Supportive Text

1. These BP cutoffs correspond to study inclusion criteria in pivotal clinical trials of intravenous thrombolysis for acute ischemic stroke.[S9.4.2-1]
2. In a large observational study of patients with acute ischemic stroke who received intravenous tissue-type plasminogen activator, high BP during the initial 24 hours was linked to greater risk of symptomatic ICH.[S9.4.2-3]
3. For the goal of antihypertensive therapy, see Section 8.1.5.
4. Extreme arterial hypertension is detrimental because it can lead to encephalopathy, cardiac compromise, and

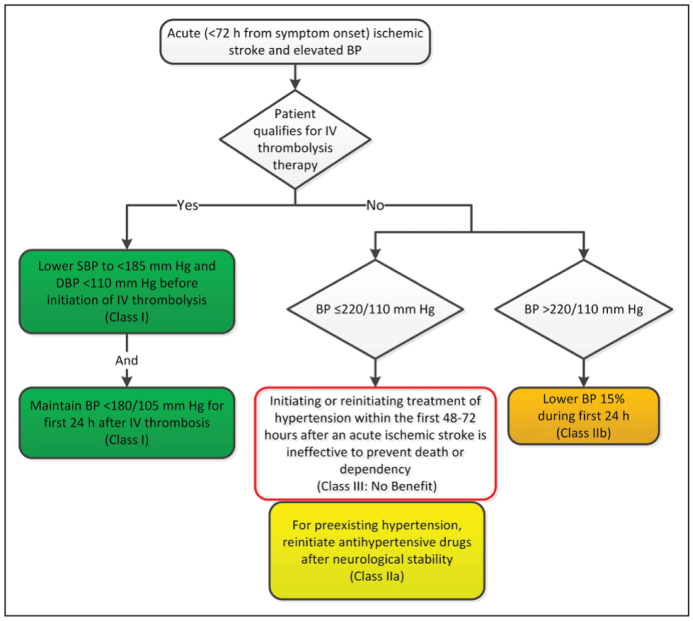

Figure 8. Management of hypertension in patients with acute ischemic stroke. Colors correspond to Class of Recommendation in Table 1. BP indicates blood pressure; DBP, diastolic blood pressure; IV, intravenous; and SBP, systolic blood pressure.

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renal damage. However, hypotension, especially when too rapidly achieved, is potentially harmful because it abruptly reduces perfusion to multiple organs, including the brain.

5. Data from 2 RCTs,[S9.4.2-5,S9.4.2-9] as well as systematic reviews and meta-analyses,[S9.4.2-6–S9.4.2-8] indicate that antihypertensive agents reduce BP during the acute phase of an ischemic stroke but do not confer benefit with regard to short- and long-term dependency and mortality rate. One RCT did not demonstrate a benefit of continuing pre-stroke antihypertensive drugs during the first few days after an acute stroke, but it was substantially underpowered to answer the question.[S9.4.2-4]

9.4.3. Secondary Stroke Prevention

Recommendations for Treatment of Hypertension for Secondary Stroke Prevention

References that support recommendations are summarized in Online Data Supplements 43 and 44.

COR	LOE	Recommendations
I	A	1. Adults with previously treated hypertension who experience a stroke or transient ischemic attack (TIA) should be restarted on antihypertensive treatment after the first few days of the index event to reduce the risk of recurrent stroke and other vascular events.[S9.4.3-1–S9.4.3-3]
I	A	2. For adults who experience a stroke or TIA, treatment with a thiazide diuretic, ACE inhibitor, or ARB, or combination treatment consisting of a thiazide diuretic plus ACE inhibitor, is useful.[S9.4.3-1,S9.4.3-3–S9.4.3-5]
I	B-R	3. Adults not previously treated for hypertension who experience a stroke or TIA and have an established BP of 140/90 mm Hg or higher should be prescribed antihypertensive treatment a few days after the index event to reduce the risk of recurrent stroke and other vascular events.[S9.4.3-1–S9.4.3-3]
I	B-NR	4. For adults who experience a stroke or TIA, selection of specific drugs should be individualized on the basis of patient comorbidities and agent pharmacological class.[S9.4.3-6]
IIb	B-R	5. For adults who experience a stroke or TIA, a BP goal of less than 130/80 mm Hg may be reasonable.[S9.4.3-6,S9.4.3-7]
IIb	B-R	6. For adults with a lacunar stroke, a target SBP goal of less than 130 mm Hg may be reasonable.[S9.4.3-8]
IIb	C-LD	7. In adults previously untreated for hypertension who experience an ischemic stroke or TIA and have a SBP less than 140 mm Hg and a DBP less than 90 mm Hg, the usefulness of initiating antihypertensive treatment is not well established.[S9.4.3-9]

Synopsis

Each year in the United States, >750 000 adult patients experience a stroke, of which up to 25% are recurrent strokes.[S9.4.3-10] For an individual who experiences an initial stroke or TIA, the annual risk of a subsequent or "secondary" stroke is approximately 4%,[S9.4.3-11] and the case mortality rate is 41% after a recurrent stroke versus 22% after an initial stroke.[S9.4.3-12] Among patients with a recent stroke or TIA, the prevalence of premorbid hypertension is approximately 70%.[S9.4.3-13] Risk of recurrent stroke is heightened by presence of elevated BP, and guideline-recommended antihypertensive drug treatment to lower BP has been linked to a reduction in 1-year recurrent stroke risk.[S9.4.3-14] RCT meta-analyses show an approximately 30% decrease in recurrent stroke risk with BP-lowering therapies.[S9.4.3-1–S9.4.3-3] An issue frequently raised by clinicians is whether the presence of clinically asymptomatic cerebral infarction incidentally noted on brain imaging (computed tomography or MRI scan) in patients without a history of or symptoms of a stroke or TIA warrants implementation of secondary stroke prevention measures. Clinically asymptomatic vascular brain injury is increasingly being considered as an entry point for secondary stroke prevention therapies, because these apparently "silent" brain infarctions are associated with typical stroke risk factors, accumulatively lead to subtle neurological impairments, and bolster risk of future symptomatic stroke events.[S9.4.3-15] Although the evidence for using antihypertensive treatment to prevent recurrent stroke in stroke patients with elevated BP is compelling,[S9.4.3-1–S9.4.3-3] questions remain about when precisely after an index stroke to initiate it, what specific agent(s) to use (if any), which therapeutic targets to aim for, and whether the treatment approach should vary by index stroke mechanism and baseline level of BP.[S9.4.3-16]

Figure 9 is an algorithm on management of hypertension in patients with a previous history of stroke (secondary stroke prevention).

Recommendation-Specific Supportive Text

1. Two overviews of RCTs published through 2009 showed that antihypertensive medications lowered the risk of recurrent vascular events in patients with stroke or TIA.[S9.4.3-1–S9.4.3-3]
2. Specific agents that have shown benefit in either dedicated RCTs or systematic reviews of RCT data include diuretics, ACE inhibitors, and ARBs.
3. Support for this recommendation is based on data from 2 dedicated RCTs, as well as a systematic review and meta-analysis, among patients with a history of stroke or TIA.[S9.4.3-1–S9.4.3-3]
4. Reduction in BP appears to be more important than the choice of specific agents used to achieve this goal. Thus, if diuretic and ACE inhibitor or ARB treatment do not achieve BP target, other agents, such as CCB and/or mineralocorticoid receptor antagonist, may be added.
5. An overview of RCTs showed that larger reductions in SBP tended to be associated with greater reduction in risk of recurrent stroke. However, a separate overview of RCTs in patients who experienced a stroke noted that achieving an SBP level <130 mm Hg was not associated with a lower stroke risk, and several observational

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