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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

Writing Committee Members

Paul K. Whelton, MB, MD, MSc, FAHA, *Chair*
Robert M. Carey, MD, FAHA, *Vice Chair*

Wilbert S. Aronow, MD, FACC, FAHA*
Donald E. Casey, Jr, MD, MPH, MBA, FAHA†
Karen J. Collins, MBA‡
Cheryl Dennison Himmelfarb, RN, ANP, PhD, FAHA§
Sondra M. DePalma, MHS, PA-C, CLS, AACC‖
Samuel Gidding, MD, FAHA¶
Kenneth A. Jamerson, MD#
Daniel W. Jones, MD, FAHA†
Eric J. MacLaughlin, PharmD**
Paul Muntner, PhD, FAHA†
Bruce Ovbiagele, MD, MSc, MAS, MBA, FAHA†
Sidney C. Smith, Jr, MD, MACC, FAHA††
Crystal C. Spencer, JD‡
Randall S. Stafford, MD, PhD‡‡
Sandra J. Taler, MD, FAHA§§
Randal J. Thomas, MD, MS, FACC, FAHA‖‖
Kim A. Williams, Sr, MD, MACC, FAHA†
Jeff D. Williamson, MD, MHS¶¶
Jackson T. Wright, Jr, MD, PhD, FAHA##

*American Society for Preventive Cardiology Representative. †ACC/AHA Representative. ‡Lay Volunteer/Patient Representative. §Preventive Cardiovascular Nurses Association Representative. ‖American Academy of Physician Assistants Representative. ¶Task Force Liaison. #Association of Black Cardiologists Representative. **American Pharmacists Association Representative. ††ACC/AHA Prevention Subcommittee Liaison. ‡‡American College of Preventive Medicine Representative. §§American Society of Hypertension Representative. ‖‖Task Force on Performance Measures Liaison. ¶¶American Geriatrics Society Representative. ##National Medical Association Representative.

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ACC/AHA Task Force Members

Glenn N. Levine, MD, FACC, FAHA, *Chair*
Patrick T. O'Gara, MD, MACC, FAHA, *Chair-Elect*
Jonathan L. Halperin, MD, FACC, FAHA, *Immediate Past Chair*

Sana M. Al-Khatib, MD, MHS, FACC, FAHA
Joshua A. Beckman, MD, MS, FAHA
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Laura Mauri, MD, MSc, FAHA
Susan J. Pressler, PhD, RN, FAHA***
Barbara Riegel, PhD, RN, FAHA
Duminda N. Wijeysundera, MD, PhD

***Former Task Force member; current member during the writing effort.

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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 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

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.

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.

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.

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 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 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

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

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.

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).

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.

2.3. 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 U.S. 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 U.S. population (S2.3-7).

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 U.S. 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 factors have hypertension. For example, 71% of U.S. 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 U.S. 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.

TABLE 5 CVD Risk Factors Common in Patients With Hypertension

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

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

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.

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 U.S. 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.

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 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 U.S. 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 U.S. 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 U.S. 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

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 U.S. adult population.

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

definition results in only a small increase in the percentage of U.S. 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).

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 U.S. general adult population, hypertension awareness, treatment, and control 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 U.S.

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 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).

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 (e.g., 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.

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. 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 (e.g., 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.

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 U.S. Preventive Services Task Force reported that ABPM provided a better method to predict long-term CVD outcomes than did office BPs. It incorporates new

TABLE 10 Procedures for Use of HBPM (S4.2-8—S4.2-10)

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.

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.

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 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 U.S. 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).

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 U.S. 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 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.

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.

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.

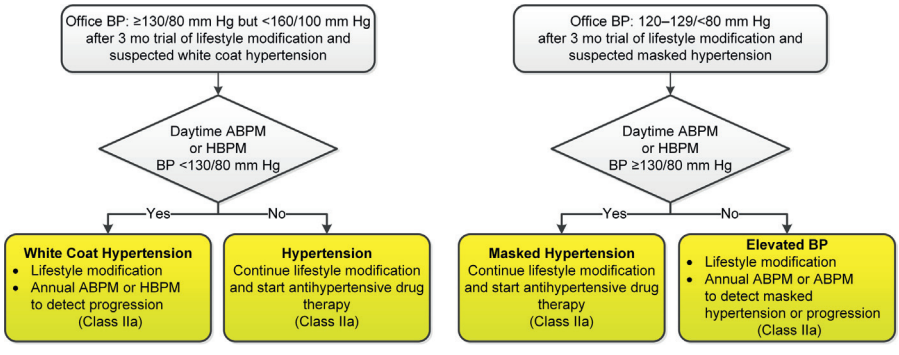
In patients treated for hypertension, both "white coat effect" (higher office BPs than out-of-office BPs) and "masked uncontrolled hypertension" (controlled office BPs but uncontrolled BPs in out-of-office settings) categories have been reported (S4.4-5,S4.4-15,S4.4-16). The white coat effect (usually 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).

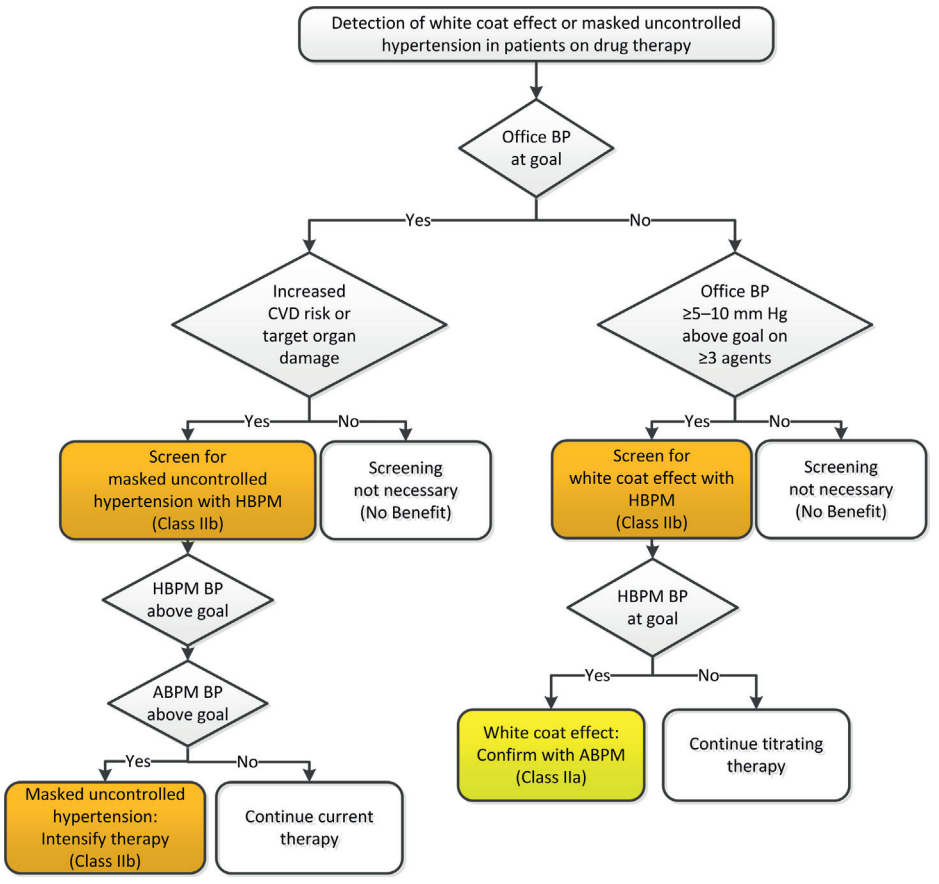
There are no data on the risks and benefits of treating white coat and masked hypertension. Despite these methodological differences, the data are consistent in indicating that masked hypertension and masked uncontrolled hypertension are associated with an increased prevalence of target organ damage and risk of CVD, stroke, and mortality compared with normotensive individuals and those with white coat hypertension.

FIGURE 1 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.

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.

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.

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