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Hypertension Management in Women With a Multidisciplinary Approach

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Abstract

Current clinical practice guidelines were established by several organizations to guide the diagnosis and treatment of hypertension in men and women in a similar manner despite data demonstrating differences in underlying mechanisms. Few publications have provided a contemporary and comprehensive review focused on characteristics of hypertension that are unique to women across their life spectrum. We performed a computerized search using PubMed, OVID, EMBASE, and Cochrane library databases between 1995 and 2023 that highlighted relevant clinical studies, challenges to the management of hypertension in women, and multidisciplinary approaches to hypertension control in women, including issues unique to racial and ethnic minority groups. Despite our current understanding of underlying mechanisms and strategies to manage hypertension in women, numerous challenges remain. Here, we discuss potential factors contributing to hypertension in women, differences related to effects of lifestyle modifications and drug therapy between men and women, the impact of sleep, and the importance of recognizing disparities in socioeconomic conditions and access to care. This review outlines several opportunities for future studies to fill gaps in knowledge to achieve optimal control of hypertension in women using a multidisciplinary approach, particularly related to sex-specific treatment approaches while considering socioeconomic conditions and life stages from premenopause through the transition to menopause.

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Hypertension is a significant global health problem and an important cause of morbidity and mortality worldwide, serving as an important risk factor for cardiovascular disease (CVD), chronic kidney disease (CKD), stroke, and dementia. The prevalence of hypertension was estimated to be 1.27 billion globally, with ~626 million women living with hypertension, worldwide.¹ Nearly 1 of 2 adults in the United States has hypertension (116 million), with more than 50% reported to be women (44.9 million women and 40.8 million men).² Optimal management of hypertension can improve both cardiovascular outcomes and mortality. It has become evident that, although hypertension is more prevalent in men,³ the risk factors for developing hypertension and the blood pressure

thresholds for its diagnosis may differ by sex. However, the current guideline does not provide different blood pressure values for men and women.⁴ Specific risk factors related to lifecycle changes are present in women that affect both development and management of hypertension. A multidisciplinary approach may lead to better blood pressure (BP) control in hypertensive females. This review, which was conducted using PubMed, OVID, EMBASE, and Cochrane library databases between 1995 and 2023, provides a comprehensive summary of the presentation and treatment of hypertension as it relates to women across the lifespan and includes reference to specific racial and ethnic minority groups. It describes relevant clinical studies and ongoing challenges and outlines unanswered questions pertinent to



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ARTICLE HIGHLIGHTS

- Hypertension and its sequelae in women should be managed by a multidisciplinary team based on the phase of a woman's life cycle, underlying medical comorbidities, socioeconomic stressors, and access to care.
- Choices of therapy should be made in relation to reproductive, peri-, and post-menopausal phases, with consideration of hormonal alterations and other comorbid factors such as hypertension disorders of pregnancy.
- Researchers are encouraged to incorporate stratification by sex, race, ethnicity, and the phase of the woman's life cycle when designing and analyzing clinical trials focused on dietary patterns, lifestyle modifications, and the efficacy of pharmacotherapy.
- Several challenges and unmet needs to optimize the management of hypertension in women still exist.

the optimal management of hypertension in women.

The 2017 American College of Cardiology/American Heart Association Clinical Practice hypertension guideline recognizes the importance of risk calculators to determine when to initiate antihypertensive therapy. It also includes major changes in BP values, lowering the treatment target from less than 140/90 mm Hg to less than 130/80 mm Hg,⁵ and supporting improved cardiovascular outcomes with treatment aimed at lower targets. These changes were derived from evidence of increased CVD risk associated with BP 130-139/80-89 mm Hg, along with data supporting better associated cardiovascular outcomes.³ According to the new guideline, BP is "normal" if it is less than 120/80 mm Hg. The guideline also categorizes hypertension by stages: patients with systolic BP between 130 and 139 mm Hg or diastolic BP between 80 and 89 mm Hg have stage 1 hypertension, whereas those with a systolic BP greater than or equal to 140 mm Hg or diastolic BP greater than or equal to 90 mm Hg have stage 2 hypertension.^{5,6} The guideline remains consistent for both men and women, targeting BP less than 130/80 mm Hg for patients with coexisting coronary artery disease (CAD),

diabetes mellitus (DM), CKD, peripheral vascular disease, and cerebral vascular accident (CVA) events without consideration for patient sex or physiological state at different life cycles.^{5,6} In this review, several sex-specific strategies to achieve optimal management of hypertension in women using a multidisciplinary approach are discussed. Importantly, socioeconomic conditions and transitions across different life stages from premenopause to menopause are also considered.

RISK OF HYPERTENSION ACROSS THE WOMAN'S LIFESPAN

The prevalence of hypertension increases with age in both sexes, particularly after 40 years of age.⁷ In women, the prevalence is slightly lower than in men between the ages of 40 to 59 years (59.4% in men vs 49.9% in women). However, by 60 years of age, women have nearly caught up with men, with hypertension prevalence of 75.2% in men and 73.9% in women.⁷

The current guideline does not provide specific recommendations related to the management of hypertension by sex. According to all major society guidelines, accurate measurements, diagnosis of hypertension with repeat visits, ambulatory blood pressure monitoring (ABPM), or home BP monitoring via commercial devices are important in the diagnosis of hypertension for both women and men. Home and ABPM measurements are less likely to be confounded by white coat or masked hypertension. Although nearly all major clinical trials use clinic BP readings, ABPM can supplement these readings because data indicate that ABPM is a stronger predictor of clinical outcomes compared with clinic measurements⁸⁻¹⁰ and allows for measurement of nocturnal pressures.¹¹⁻¹³

A 10% to 20% decrease in nighttime BP compared with daytime is expected,^{5,6} and patients who are nighttime "nondippers" or "reverse dippers" have higher risk for stroke, cardiac hypertrophy, and silent CVA events.^{8,11,14,15} Reverse and nondipping BP, for women more than men, increase the risk of cardiovascular events with age.

Middle-aged and postmenopausal women who are nondippers are at highest risk for cardiovascular events, particularly if they present with comorbid conditions.¹⁶ Nondipping is associated with a higher left ventricular mass in both sexes, although this is the case to a greater extent in women.¹⁷

Ambulatory BP monitoring has also allowed for assessment of important changes in BP over the lifetime of males vs females. Using ABPM data in 15,913 women and 14,600 men, diastolic BP was noted to fall at a very early age (22.3 years) in women, whereas in men, diastolic BP begins to fall at age 46.5 years.¹⁸ Finally, there is a greater prevalence of masked hypertension in men than women, as detected by ABPM.¹⁹⁻²¹

HYPERTENSION DURING REPRODUCTIVE AGE

Epidemiological studies have shown associations between clinical BP or diagnosed hypertension in youth with atherosclerotic CVD and premature mortality.²² The menstrual cycle affects arterial compliance and may change systolic and diastolic BP; however, these variations in normotensive women appear to be modest and larger studies are needed.

Although most patients have primary (essential) hypertension, the proportion of individuals affected by secondary hypertension is higher in women of reproductive age, despite a lower prevalence of primary hypertension in this age group. The diagnosis of secondary causes requires a high index of suspicion, and appropriate testing based on clinical presentation. Early detection of secondary hypertension is pivotal because timely diagnosis and management of underlying condition allow for prevention of hypertension-mediated organ damage (HMOD).

Several recognized endocrine causes of secondary hypertension include primary aldosteronism (PA), pheochromocytoma, Cushing syndrome, thyroid disease, acromegaly, and hyperparathyroidism.²³ Primary aldosteronism is the most common cause of secondary hypertension, with a prevalence of ~5% among patients with hypertension, and up to 20% in individuals with resistant hypertension.^{24,25} The most common cause

of PA is bilateral adrenal hyperplasia, followed by aldosterone-producing adrenal adenoma, representing in 60% and 30% of cases, respectively.²⁶ Unilateral PA is more common in men, whereas bilateral adrenal hyperplasia is more common in women.²⁷ Observed differences in incidence are further modified by age, where women diagnosed with unilateral adrenal hyperplasia are more likely to be younger than men. The inverse of this is true for bilateral adrenal hyperplasia, as male patients with this diagnosis tend to be younger than female patients.²⁷ The prevalence of PA is similar in men and women, but estrogen can affect plasma renin concentration (PRC), and may interfere with laboratory investigation of PA. Although there is good correlation between plasma renin activity (PRA) and PRC, estrogen use or preovulatory (luteal phase) estrogen surge in women is associated with false positive case detection testing when using PRC but not PRA. Plasma renin activity may be more suitable for PA case detection in premenopausal women or in those taking oral estrogen.^{26,28}

Salt-sensitive BP characterized by an increase in BP following salt loading or a drop in BP following salt depletion is reported in up to 50% of hypertensive patients.²⁹ This is more common in women than men and increases with age.^{30,31} The mineralocorticoid pathway may be implicated in the mechanism for salt-sensitive hypertension in younger women. Premenopausal women may have heightened aldosterone production to stimulate and expression of endothelial mineralocorticoid receptor (MR) mediated by sex hormones that lead to endothelial dysfunction and hypertension. Therefore, MR antagonism may be preferred in the treatment of salt-sensitive hypertension in premenopausal women.³² The role of MR activation and augmented aldosterone production diminishes after menopause. In postmenopausal women, as in men, dysfunctional renal physiology leading to impaired natriuresis is the main contributor to salt-sensitive hypertension.^{16,32}

Renovascular hypertension is among the most common causes of secondary hypertension, primarily from atherosclerotic lesions (60%-90%) followed by fibromuscular

dysplasia (10%-30%).³³ Fibromuscular dysplasia has a female/male ratio of 84%/16% in the United States and is predominantly diagnosed in middle-aged subjects.³³ It is a noninflammatory disease of medium-size arteries which may lead to stenosis, occlusion, and or dissection.³⁴ Approximately 90% of these patients have hypertension, and a significant proportion of them may present with major vascular events such as CAD, cerebrovascular and renal events including ischemia, infarction, and renal failure. Earlier diagnosis and vascular interventional treatment with angioplasty will improve outcome and adverse events (Table 1).^{33,34}

Preconception Care

Optimizing the care of the pregnant person with hypertension, even before pregnancy, is the ideal. As such, a preconception consultation with a multidisciplinary care team, including maternal-fetal medicine, is recommended in reproductive-age people who desire pregnancy.⁴³ The goal of preconception care is to affirm pregnancy intention, mitigate potential harm, and recognize modifiable risk factors related to pregnancy while stratifying pregnancies on a continuum of low to high risk.⁴³ Although most patients with well-controlled hypertension will have uncomplicated pregnancies, patients with hypertension should have a clear understanding of the possible complications in pregnancy, potential effects of pregnancy on BP, and the need for heightened increased maternal and fetal surveillance. Patients with modifiable risk factors such as obesity, smoking, and poorly controlled diabetes may benefit from a discussion on lifestyle modifications. These include weight loss, diet, exercise, and smoking cessation to improve BP control and decrease rates of hypertension disorders of pregnancy (HDP) and associated adverse maternal and fetal outcomes.^{44,45} All patients in need of renin-angiotensin-aldosterone (RAAS) blockade agents should have a discussion of effective contraceptive options along with negative pregnancy test before initiation of therapy given teratogenicity of these agents including fetal cardiac and kidney defects.⁴⁶

Contraceptives

Drug-induced hypertension by estrogen and progestins is another cause of secondary hypertension among women. The first cases of severe BP elevations after oral contraceptive pills (OCPs) were noted in the 1960s.⁴⁷ A few decades later, changes in the RAAS have been noted in users of OCPs, and cumulative use of OCPs has been found to be associated with a higher risk of hypertension.⁴⁸ Combined hormonal contraceptives have remained a more popular option among users and newer formulations of OCPs containing natural estrogens and progestins with antiminerlocorticoid effects (ie, drospirenone) to decrease or reverse the association between OCP use and BP elevations.⁴⁹ Approximately 10% of reproductive-aged women have systolic BP greater than or equal to 140 mm Hg or diastolic BP greater than or equal to 90 mm Hg, and almost 20% have systolic BP greater than or equal to 130 mm Hg or diastolic BP greater than or equal to 80 mm Hg in the United States.⁵⁰ Therefore, standardized practices to identify contraindications to OCPs are needed to ensure safety of use. Hormonal intrauterine devices which only contain progestin are generally considered safe for individuals with high BP.⁵¹ A pre-existing understanding of the risks and benefits associated with hormonal contraceptive agents is recommended.

HYPERTENSIVE DISORDERS OF PREGNANCY

Hypertensive disorders of pregnancy, including pre-eclampsia, eclampsia, and gestational hypertension affect ~10% of all pregnancies and are associated with increased morbidity and mortality for the mother and the fetus.⁵²⁻⁵⁴ Hypertensive disorders of pregnancy include both pregestational and gestational hypertension, with maternal and neonatal complications.^{55,56} According to the 2019 National Center for Health Statistics data, more than 83,000 pregnant women in the United States had chronic hypertension predating their pregnancies⁵⁷ and ~46,000 maternal deaths were associated with pre-eclampsia or gestational hypertension. The prevalence of HDP

TABLE 1. Secondary Causes of Hypertension and Gender Difference^a

Cause	When to suspect	Laboratory screening	Sex difference
Primary aldosteronism ³⁵	Hypokalemia (28% of cases) Resistant hypertension Age <50 y.	Plasma renin activity (preferred if on estrogen) or plasma renin concentration and plasma aldosterone.	None
Renovascular disease ^{33,34} Fibromuscular dysplasia 10% -30% Atherosclerotic renal artery stenosis 60%-90%	Severe hypertension in young and middle-aged women. It may also occur in men and children with resistant hypertension. Common in patients with atherosclerosis — higher in men older than 45 y.	Renal artery duplex ultrasound and/or CT angiography.	9-fold higher prevalence in women.
Drug-induced hypertension ³⁶	Sympathetic agent NSAIDs Corticosteroids CNS-stimulants Estrogen and progestins Contraceptives Dietary supplements (ginseng, licorice, yohimbine) SNRIS immunosuppressants		Dietary supplement and NSAIDs use are more common among women.
OSA ^{37,38}	Snoring Gaspings Apneas Morning headache Fatigue Depression Women, tend to present in an atypical fashion with less frequent reports of snoring or witnessed apneas.	Polysomnography Home sleep apnea test	OSA is more common in men than women and the prevalence increases throughout life in both sexes.
Endogenous Cushing syndrome ²⁹	Higher degree of suspicion: wide purple striae, thin skin with easy bruising, proximal limb atrophy, and facial plethora. Common yet less specific characteristics: weight gain, excessive abdominal adiposity, fatigue, acne, hirsutism, and menstrual irregularities.	Overnight 1 mg dexamethasone suppression test cortisol 24-h urine free cortisol. Late-night salivary cortisol.	3-fold higher incidence in women.
Pheochromocytomas-parangliomas ³⁹	Adrenal mass Familial genetic syndrome Classic symptoms of headaches, palpitations, pallor, and diaphoresis Nonischemic and nonvalvular cardiomyopathy, including Takotsubo cardiomyopathy Sustained or paroxysmal hypertension Normal BP in 5% to 15% of patients	Plasma free or urinary fractionated metanephrines	None
Severe thyroid dysfunction ⁴⁰	Hyperthyroidism: tachycardia, fine tremor, weight loss, increased bowel movements. Hypothyroidism: bradycardia, weight gain, new onset constipation, fatigue.	TSH, Free T4, and Free T3	10-fold higher prevalence in women
Severe primary hyperparathyroidism ⁴¹	Hypercalcemia Nephrolithiasis Low bone mineral density Neuropsychologic symptoms	PTH and calcium levels	2-3-fold higher incidence and prevalence in women

Continued on next page

TABLE 1. Continued

Cause	When to suspect	Laboratory screening	Sex difference
Acromegaly ⁴²	Enlargement of acral tissues, frontal bossing, and mandibular prognathism Increased sweating Headache Worsening or new onset type 2 diabetes mellitus Obstructive sleep apnea	Screening IGF-I level Confirmatory: growth hormone suppression test to an oral glucose load	None

^aBP, blood pressure; CNS, central nervous system; CT, computed tomography; IGF, insulin-like growth factor; NSAIDs, nonsteroidal anti-inflammatory drug; OSA, obstructive sleep apnea; PTH, parathyroid hormone; SNRIS, serotonin and norepinephrine reuptake inhibitor; TSH, thyroid stimulating hormone.

is expected to continue to rise with the obesity epidemic and increasing maternal age.⁵⁸ Data from a large cohort of 433,430 women in the United States confirmed an increase in maternal CAD, stroke, and mortality within 5 years post-delivery among women with HDP.⁵⁹ It is also associated with peripheral vascular disease and CKD.⁵⁵ Across all ethnicities, HDPs are associated with significantly increased risk for developing CKD and chronic hypertension⁶⁰ with a large increase among black women.⁶¹ Among HDPs, pre-eclampsia has been referred to as “the great obstetrical syndrome,”⁶²⁻⁶⁴ with plausible mechanisms including intravascular inflammation, endothelial cell activation, and syncytiotrophoblast stress.⁶⁵ Imbalances in circulating angiogenic factors which regulate angiogenesis and vessel remodeling play a pathogenic role with increased circulating soluble fms-like tyrosine kinase 1, which binds placental growth factor and vascular endothelial growth factor. This triggers the onset of pre-eclampsia by inducing microangiopathy.⁶⁴⁻⁶⁶ Pre-eclampsia and CVD share the same risk factors including diabetes, hypertension, and obesity with a 30% risk of chronic hypertension in the decade after birth.⁶⁴ The unifying theme touches on a multitude of organ systems, with significant lifelong sequelae for the fetus and the pregnant person.⁶⁷⁻⁶⁹ The risk of complications has been estimated to be double for CVD and five times higher for the development of hypertension. The greatest risk is observed in those with recurrent pre-eclampsia in subsequent pregnancies or

those with early-onset pre-eclampsia requiring delivery before 34 weeks’ gestation.⁷⁰⁻⁷² Establishment of long-term follow-up is encouraged to not only continue to monitor BPs but also assess and treat modifiable cardiometabolic risk factors.

In 2019, updates on the primary prevention of CVD guideline indicated that a history of HDPs was a risk-enhancing factor requiring statin therapy for atherosclerotic CVD prevention.⁷³ As mentioned earlier, the American College of Cardiology/American Heart Association guideline lowered the stage 1 hypertension threshold from 140/90 mm Hg to 130/80 mm Hg.⁷³ As of 2022, the Society for Maternal-Fetal Medicine has revised the recommendation for treatment of chronic hypertension in pregnancy to lower BP to a target goal of less than 140/90 mm Hg.⁷⁴ The National Institute for Health and Care Excellence guideline recommends treatment for greater than or equal to 140/90 mm Hg with a goal of less than 135/85 mm Hg.⁷⁵ The International Society for the Study of Hypertension in Pregnancy recommends treatment to maintain a goal of 110-140/80-85 mm Hg.⁷⁶ Based on available data, BP management in pregnancy indicates the benefits of lower treatment thresholds outside of pregnancy.

Management of Categories of HDP

The categories of HDPs, defined by the American College of Obstetricians and Gynecologists Hypertension in Pregnancy Task Force diagnostic criteria and management are summarized in [Table 2](#).

SELECTION OF ANTIHYPERTENSIVE AGENTS IN PREGNANCY

Specific antihypertensive therapy is required for: (1) chronic treatment to gradually lower BP to maintain goal range, or (2) acute lowering of severe-range BP. The timing and setting of intervention along

with the selection of antihypertensive agents will differ between these scenarios. The most used antihypertensive agents are listed in the [Supplemental Table](#) (available online at <http://www.mayoclinicproceedings.org>). Labetalol or nifedipine extended release are preferred for the

TABLE 2. Hypertensive Disorders of Pregnancy^{a,b}

Condition	Diagnostic criteria	Management
Chronic hypertension	BP: systolic BP ≥ 140 mm Hg or diastolic BP ≥ 90 mm Hg on two occasions at least 4 h apart any time before 20 wks of pregnancy, including outside of pregnancy	Well-controlled, no medication, expectant management 38 0/7- 39 6/7-wks' gestation Well-controlled, with medication, expectant management 37 0/7- 39 6/7-wks' gestation Difficult to control, medication, expectant management 36 0/7- 37 6/7-wks' gestation
Gestational hypertension	BP: systolic BP ≥ 140 mm Hg or diastolic BP ≥ 90 mm Hg on two occasions at least 4 h apart after 20 wks with previously normal BPs	Expectant management up to 37 0/7 wks' gestation or at diagnosis, if later
Preeclampsia without severe features	BP: systolic BP ≥ 140 mm Hg or diastolic BP ≥ 90 mm Hg on two occasions at least 4 h apart after 20 wks with previously normal BPs and Proteinuria: ≥ 300 mg/24-hour urine collection or protein/creatinine ratio ≥ 0.3 or dipstick reading 2+, if quantitative methods are not available	Expectant management up to 37 0/7 wks' gestation or at diagnosis if later
Pre-eclampsia with severe features	BP: systolic BP ≥ 160 mm Hg or diastolic BP ≥ 110 mm Hg on two occasions at least 4 h apart after 20 wks with previously normal BPs or Systolic BP ≥ 140 mm Hg or diastolic BP ≥ 90 mm Hg on two occasions at least 4 h apart after 20 wks with previously normal BPs with evidence of end-organ damage, including: Thrombocytopenia (platelet $< 100 \times 10^9/L$), Renal insufficiency (creatinine > 1.1 mg/dL or doubling in serum creatinine in the absence of other renal disease) Impaired liver function (transaminases twice normal), or severe persistent epigastric pain unresponsive to pain Pulmonary edema New-onset headache unresponsive to medication Visual disturbances	Expectant management up to 34 0/7 wks or at diagnosis if later unless any contraindication to expectant management, including eclampsia, fetal demise, non-reassuring fetal heart tracing, placental abruption, disseminated intravascular coagulation, renal insufficiency, HELLP syndrome, or persistent symptoms of severe pre-eclampsia
HELLP syndrome	May include elevated BPs: systolic BP ≥ 140 mm Hg or diastolic BP ≥ 90 mm Hg. Lactate dehydrogenase ≥ 600 IU/L Transaminases twice normal Platelets count $< 100 \times 10^9/L$	Maternal stabilization Delivery at diagnosis soon after maternal stabilization

^aBP, blood pressure; HELLP, hemolysis, elevated liver enzymes, low platelet count.

^b■■■■

From the American College of Obstetrics and Gynecology.^{55,56}

long-term treatment of hypertension in pregnancy.⁵⁵ Methyldopa, previously considered first-line, has shown lower efficacy with greater adverse effects.⁷⁷ Diuretics can be considered second- or third-line HDP treatment, and their use during pregnancy and breastfeeding is generally tolerated but should be closely monitored.⁷⁸ Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers are avoided due to potential teratogenic risks and associations with fetal growth restriction and neonatal kidney failure.⁷⁹ There is evidence demonstrating the risk of beta-blockers, primarily atenolol, and its association with small for gestational age infants.⁷⁹

For severe hypertension (systolic ≥ 160 mm Hg or diastolic ≥ 110 mm Hg) and if it is persistent (>15 minutes) requires prompt pharmacologic treatment. Therapy should be initiated within 30 minutes of diagnosis to decrease maternal risks. Medications used include intravenous labetalol, hydralazine, or oral immediate-release nifedipine.⁸⁰ Hypertension disorders of pregnancy require timely treatment because CVA and hypertensive encephalopathy occur at lower BPs compared to outside of pregnancy. Pregnancies complicated by HDP should undergo increased antenatal surveillance for maternal health and risks to the neonate, including fetal growth restriction. Delivery timing depends on the underlying HDP diagnosis and the control of the disease process. The delivery mode (ie, vaginal vs cesarean birth) is not dictated by HDPs and is based solely on routine obstetrical indications.⁵²

Postpartum Care for HDP

Blood pressure commonly decreases immediately after birth and declines for a few days postpartum but, after 3 to 5 days, there is a gradual increase in BP that often reaches levels higher than those observed during the antepartum period. The etiology is thought to be due to the mobilization of extravascular fluid leading to an increase in intravascular volume. Because the postpartum period is high-risk for pregnancies

with HDPs, an outpatient follow-up within 1 to 2 weeks is recommended.⁸¹

Antihypertensive medications are titrated more liberally postpartum, with goals similar to those for nonpregnant woman. The majority of antihypertensive medications can be used safely during breastfeeding, with very low concentrations secreted in breastmilk.⁸² Angiotensin-converting enzyme inhibitors, including enalapril and captopril in low doses, have also shown low concentrations in breastmilk and can be considered an additional second- or third-line therapy if necessary as long as higher doses are avoided.⁸² Achieving optimal BP targets over the entire life cycle and establishing long-term follow-up are encouraged to not only continue to monitor BP but also to assess and treat modifiable cardiorenal metabolic risk factors and HMOD.^{55,56} The recommendation to work concomitantly with lactation consultants and maternal-fetal medicine physicians for specific medication and individualized care to the postpartum patient is imperative (Table 2).^{55,56}

HYPERTENSION AND MENOPAUSE

Changes in BP after menopause seem to be related to alterations in estrogen and progesterone levels along with other factors including genetic predisposition, obesity, type 2 DM, endothelial dysfunction, salt sensitivity, and arterial stiffness.⁸³

Estrogen plays a significant role in the observed sex differences in hypertension and CVD.^{16,84} The mechanism for its vascular protective role is complex, and many pathways remain to be clarified. Estrogen contributes to vascular homeostasis by upregulation of endothelial nitric oxide pathway, augmenting prostacyclin release, reducing oxidative stress and fibrosis, and stimulating angiogenesis. Decline in estrogen has been associated with augmentation of ACE and angiotensin II pathways and lower MR expression.^{32,85} Estradiol deficiency results in RAAS system dysregulation with switch to proinflammatory pathways that contributes to impaired immune response and CVD.⁸⁶ Estrogen modulates

sympathetic tone by attenuating α -adrenergic receptor-mediated vasoconstriction, while enhancing β -adrenergic receptor effect. This may be a cause of age-related increase in hypertension among women that is at least in part caused by falling estrogen levels during menopause.^{16,84}

Although observational studies in humans and experimental studies in animals provide evidence that estrogen replacement protects post-menopausal women against CVD, randomized control trials did not support this concept.⁸⁷ The reason for the disparity remains unclear; yet, it is hypothesized that several factors, including the type of estrogen used, interaction with progesterone, women's age, and timing of the treatment, contribute to these results.^{88,89} There is evidence that estrogen therapy may be cardioprotective if started around menopause transition and may be harmful if started >10 years after menopause.⁹⁰ Replacement with endogenous 17 β -estradiol shows superior cardiovascular benefit compared to replacement with oral conjugated equine estrogen, which is associated with a greater risk of hypertension development.^{91,92} Data on possible benefits of hormone replacement therapy on CVD are controversial.^{93–95}

International medical societies, including the American Association of Clinical Endocrinologists, Endocrine Society, American College of Obstetricians and Gynecologists, and North American Menopause Society recommend against menopausal hormone therapy for primary or secondary prevention of CVD, especially for those older than 60 years or more than 10 years after menopause.⁹⁶ Estrogen is a safe option for treatment of menopausal symptoms when initiated in healthy women younger than 60 years of age or within 10 years of menopause onset when it is used at the lowest effective dose and for the shortest total duration based on risk benefit analysis (typically <5-10 years).⁹⁷

However, estrogen may have a plaque-destabilizing effect in the setting of advanced atherosclerosis, favoring thrombosis. Therefore, it is contraindicated in women with

atherosclerotic CVD, BP greater than 180/110 mm Hg, venous thrombosis or pulmonary embolism, cerebrovascular disease, and congenital heart disease.⁹⁸ Selected younger (<60 years of age) women with one or more cardiovascular risk factor including obesity, controlled hypertension, diabetes, and dyslipidemia may use menopausal hormone therapy; however, the transdermal route of estrogen is preferred and there should be an emphasis on optimizing primary prevention efforts. Additionally, cardiovascular risk must be regularly reassessed.^{93–95} Additional studies are needed to better understand the consequences of menopause on CVD and to determine the value, timing, and dosing of hormone replacement therapy on BP.

HYPERTENSION IN TRANSGENDER INDIVIDUALS

Transgender patients receiving gender-affirming hormonal therapy may suffer from higher rates of CVD than cisgender people.⁹⁹ Transgender women are generally prescribed a combination of estrogen and an antiandrogen if they have not had orchiectomy, whereas transgender men are generally only on testosterone. In transgender women, the choice of antiandrogen has a profound effect on BP.¹⁰⁰ For example, decreased BP has been noted with spironolactone and increased BP has been observed with cyproterone acetate.^{100,101} In transgender men, BP increases after initiation of testosterone therapy; however, the incidence of hypertension in younger transgender men is low and comparable to the general population.^{100,101} One study found that transgender men receiving long-term testosterone exhibit higher aging-related arterial stiffness compared to cisgender women and cisgender men, suggesting a potential deleterious effect of testosterone on arterial function.¹⁰² The effect of male sex hormones in arterial vessels may be direct or indirect through estrogen originating from the peripheral aromatization of testosterone. Prospective studies evaluating the long-term effects of gender-affirming therapy on BP and HMOD are needed.

RACIAL AND ETHNIC DIFFERENCES IN WOMEN WITH HYPERTENSION

The prevalence of hypertension in the United States may vary by race and ethnicity. Specifically, the prevalence of hypertension in Black individuals is higher (45.3%) compared to either White individuals (31.4%, $P < .001$), Hispanic individuals (31.6%), or Asian American individuals (31.8%).¹⁰³ The discrepancy may partly be due to differences in lifestyle, genetic influences, and economic status. In addition, differences in prevalent rates may be an underlying contributor to the higher hypertension-related complications in Black persons that include CKD, CVD, stroke, heart failure, and death compared to other racial and ethnic minority groups.^{103,104}

Furthermore, the prevalence of hypertension in Black women is higher than in Black men. Recent studies reported the prevalence of hypertension among Hispanic women as 14.0% and 39.0% among non-Hispanic Black women.¹⁰⁵ Looking at trends in hypertension incidence solely among women, Hispanic women present with the second highest incidence of hypertension after non-Hispanic Black women.¹⁰⁶

Rates of hypertension awareness tend to be higher in White compared to Black patients, but hypertension control rates are lower across all racial and ethnic groups, possibly due to systemic racial discrimination, differences in access to health care, socioeconomic factors, and lack of trust in the health care system.^{103,104} Although Black women have higher hypertension prevalence, they have similar rates of hypertension awareness and control compared to men. Among all racial and ethnic minority women, Hispanic women are reported to achieve the highest hypertension control (61.7%) with medications, followed closely by Non-Hispanic White (59.6%) and Non-Hispanic Black women (41.5%).¹⁰⁵

In Black women, depression is associated with both hypertension and the lack of healthy lifestyle promoting behaviors.¹⁰⁷ Psychosocial factors, such as lower income

level, greater number of comorbidities, lower coping mechanisms and resilience, and poorer medication adherence were significantly associated with higher depression scores, contributing to higher levels of hypertension.^{107,108} Psychological stressors, including discrimination, financial stress, and reduced caregiving are further associated with higher rates of hypertension in Black women.¹⁰⁸ Social determinants of health such as health literacy, patient activation, and financial stress are more prevalent barriers to optimal BP control, particularly in Black women compared to their White counterparts.¹⁰⁹

Future strategies to achieve better control of hypertension among racial and ethnic minority women may begin with appropriate and culturally sensitive screening approaches, increasing awareness, and improving access to appropriate use of antihypertensive medication. Importantly, approaches to achieve more equitable management may include identification of barriers to care, particularly among women of reproductive age.

LIFESTYLE MODIFICATIONS AND HYPERTENSION MANAGEMENT IN WOMEN

Lifestyle modification plays a pivotal role in preventing and managing hypertension. In fact, 24.3 million (21%) adults with hypertension can be treated with lifestyle modifications alone, without the need for antihypertensive medications.¹¹⁰

Nutrition and Hypertension Management in Women

The Dietary Approaches to Stop Hypertension (DASH) diet has emerged as a balanced dietary strategy for preventing and treating hypertension.¹¹¹ This diet emphasizes fruits, vegetables, and low-fat dairy products and recommends reduced amounts of saturated fat, total fat, and cholesterol and is rich in potassium, magnesium, calcium, and fiber. Importantly, the DASH diet lowers BP beyond the level achievable by simply reducing sodium intake. It has been demonstrated that it not only results in the expected lower

BP, but can benefit cardiometabolic health, decreasing body mass index (BMI), body fat content, fasting glucose, insulin, and leptin concentrations.^{112,113}

The consensus from studies on sex differences in dietary patterns and hypertension outcomes remain unclear. One study on sex differences in those between 40 and 69 years of age found that diets higher in the consumption of vegetables, potatoes, fruits, beans, and seaweeds were related to lower BP.¹¹⁴ Another study concluded a diet rich in whole grains and legumes was inversely associated with the risk of hypertension in women, suggesting sex differences in association with diet and hypertension.¹¹⁵ Additional studies are needed to evaluate diet-based recommendations for women in the various stages of life cycle relative to hormonal alterations, aging, sodium retention after menopause and by race and ethnicity.

Impact of Alcohol on Hypertension Management in Women

The effect of chronic alcohol intake on BP in women is understudied. The relationship between alcohol intake and BP is complex and might vary at different stages of a woman's life cycle. In healthy premenopausal women 25 to 49 years of age, a randomized controlled intervention study confirmed dose-dependent effects of alcohol on ambulatory BP.¹¹⁶ These findings verified that regular consumption of alcohol (200-300 mL red wine/d, 146-218 g alcohol/wk) can elevate 24-hour systolic and diastolic BPs. This confirmed that the magnitude of the increase in BP is similar to that previously reported in men.¹¹⁶ Although drinking within "sensible limits" of 1 to 2 drinks/d seems to have no negative impact on chronic hypertension,¹¹⁷ additional studies to investigate the impact of alcohol on women's BP and arterial stiffness in different stages of the life cycle are needed.

Impact of Cigarette Smoking on Hypertension in Women

Smoking remains the single most important preventable cause of cardiovascular morbidity and mortality worldwide. Earlier

data in a large cohort of women smokers suggested a modest association between developing hypertension and smoking, with the strongest effect among women who smoke at least 15 cigarettes per day.¹¹⁸ Smoking seems to be more harmful in women than men, particularly women who use oral contraceptives.¹¹⁹ Results from a European study on life expectancy suggest that women who smoke, like men, have a relatively higher risk of reduced life expectancy.¹²⁰ There is a need for sex-based studies to address the gaps in knowledge and determine whether these differences are rooted in biological factors, linked to variations in smoking behavior, or influenced by factors such as BMI, age, and hormonal status.

Weight and Body Mass and Hypertension in Women

Increased BMI greater than 25 kg/m² is associated with greater risk of hypertension and mortality, whereas weight loss can reduce risk of hypertension.^{121,122} Data support up to a 20-mm Hg drop in BP with 10 kg of weight loss.¹²³ Combined use of DASH diet and weight loss can further lower BP.⁵ Results from a study on women who had a BMI greater than 25 kg/m² at the age of 18 years showed a relative risk of 2.2 for developing hypertension.¹²¹ Recent data indicate that indices of central adiposity including waist circumference (WC), and waist-to-height ratio (WHtR), may have better predictive value for the risk of hypertension in women.¹²⁴ The WC and waist to hip ratio and WHtR may increase with parity and is consistently associated with an increase in WC and a reduction in hip circumference in women 18 to 30 years of age.¹²⁵

Impact of Physical Activity on Hypertension in Women

Approximately 80% of US adults are insufficiently active. Regular, mild-to-moderate aerobic activity can independently decrease BP by 5 to 8 mm Hg in women, regardless of weight loss.^{126,127} Few studies have assessed the combined impact of body

weight and physical activity in relation to hypertension among women. A report studying a large cohort of French women concluded that higher physical activity was associated with a lower risk of hypertension, but only within a BMI range of 22.5 to 25.0 kg/m².¹²⁸ Further studies are needed to provide guidance on the duration and intensity of physical activity on BP reduction and HMOD in women.

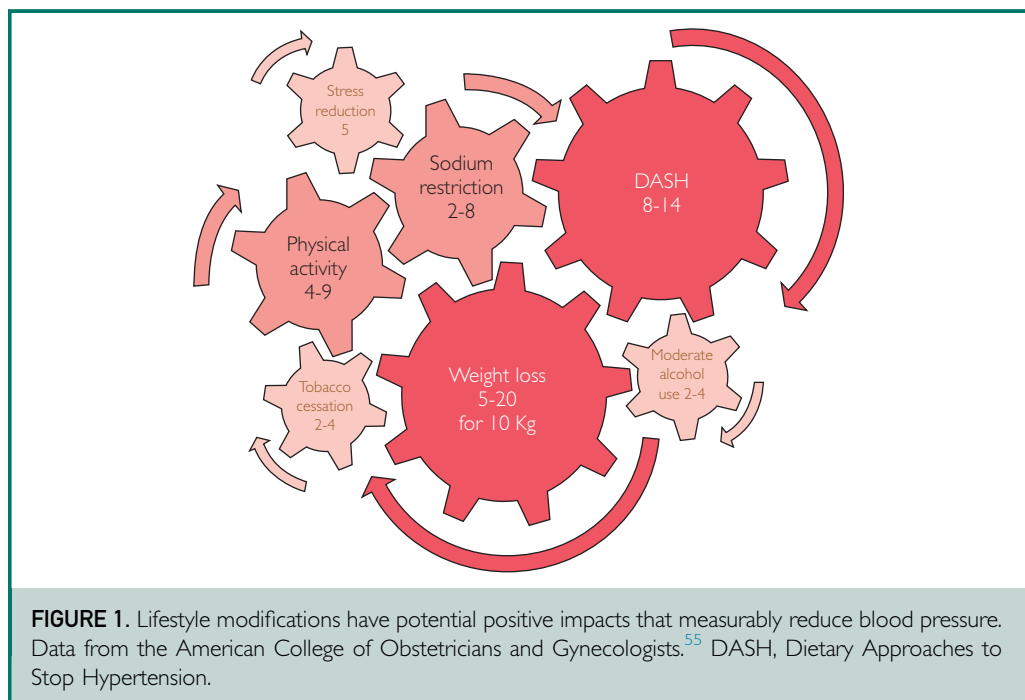
Impact of Sleep on Hypertension in Women

Sleep disturbances are associated with increased risk of morbidity and mortality.¹²⁹ Key sleep disorders that can impact BP are obstructive sleep apnea (OSA), short sleep duration, and poor sleep quality.^{130–132} One study of 277 perimenopausal women with a mean age of 56 years and a mean BMI of 28 kg/m² showed that women with moderate to severe OSA were more likely to be hypertensive, use more medications to reduce BP, and have higher awake and nocturnal BP and increased arterial stiffness.¹³³ Results from the Nurses' Health Study supported the need for sufficient sleep to reduce hypertension incidence and

prevalence.¹³¹ The prevalence of hypertension was significantly higher among women who slept 5 hours or less per night. Studies suggest that night shift work, short sleep duration, or poor sleep with circadian disruption might increase the risk of hypertension because acute sleep restriction has been shown to increase BP and sympathetic nervous system activity.^{131,134} Figure 1 summarizes the effects of lifestyle modifications on BP with the largest impacts being from the DASH diet and weight loss.⁵

PSYCHOLOGICAL PARAMETERS IN WOMEN WITH HYPERTENSION

The association between mental health and development of hypertension is complex.¹³⁵ Presence of depression, anxiety, or chronic stress, and particularly severe mental illness is associated with hypertension and other cardiovascular risk factors.¹³⁶ Psychosocial factors that induce emotional stress can evoke a physiological response mediated by activation of sympathetic nervous system, inflammation, and the hypothalamic–pituitary–adrenal axis.^{137,138} Factors such as hostility and job-related stress have



been found to be associated with higher circulating catecholamines, higher cortisol, and increased BP over time.¹³⁹

Comorbid depression, anxiety, and severe mental illness with hypertension share common risk factors and neurobiological mechanisms such as reduced baroreflex sensitivity due to autonomic dysfunction caused by chronic stress. Cardio- and cerebrovascular risk factors (eg, obesity, smoking, poor sleep, chronic stress, and poor lifestyle habits) are higher in those with mental illness, including depression and anxiety.^{136,140–143} A cohort study of women veterans showed that post-traumatic stress disorder was associated with increased risk of ischemic heart disease in these women.¹⁴⁴ This may have implications for ischemic heart disease risk assessment in vulnerable individuals.

Menopause is the time of a woman’s life cycle recognized by biological and social changes which can impact mental wellbeing and risk of hypertension and CAD.¹⁴⁵ Menopause transition has an additional adverse effect to aging that may demand specific attention to ensure optimal cardiovascular health and quality of life.¹⁴⁵ Stress and depression management can play a complementary role to lifestyle modification to decrease BP.¹⁴⁶ Studies suggest stress-reducing interventions, such as transcendental meditation, decrease diastolic and systolic BPs.^{146,147} Some potential confounders in former studies can be psychosocial determinants of health such as socioeconomic status, access to health care, obesity, history of chronic stress, and trauma.^{140,141} To address race-related stressors, such as racial discrimination, a review from 2015 highlights

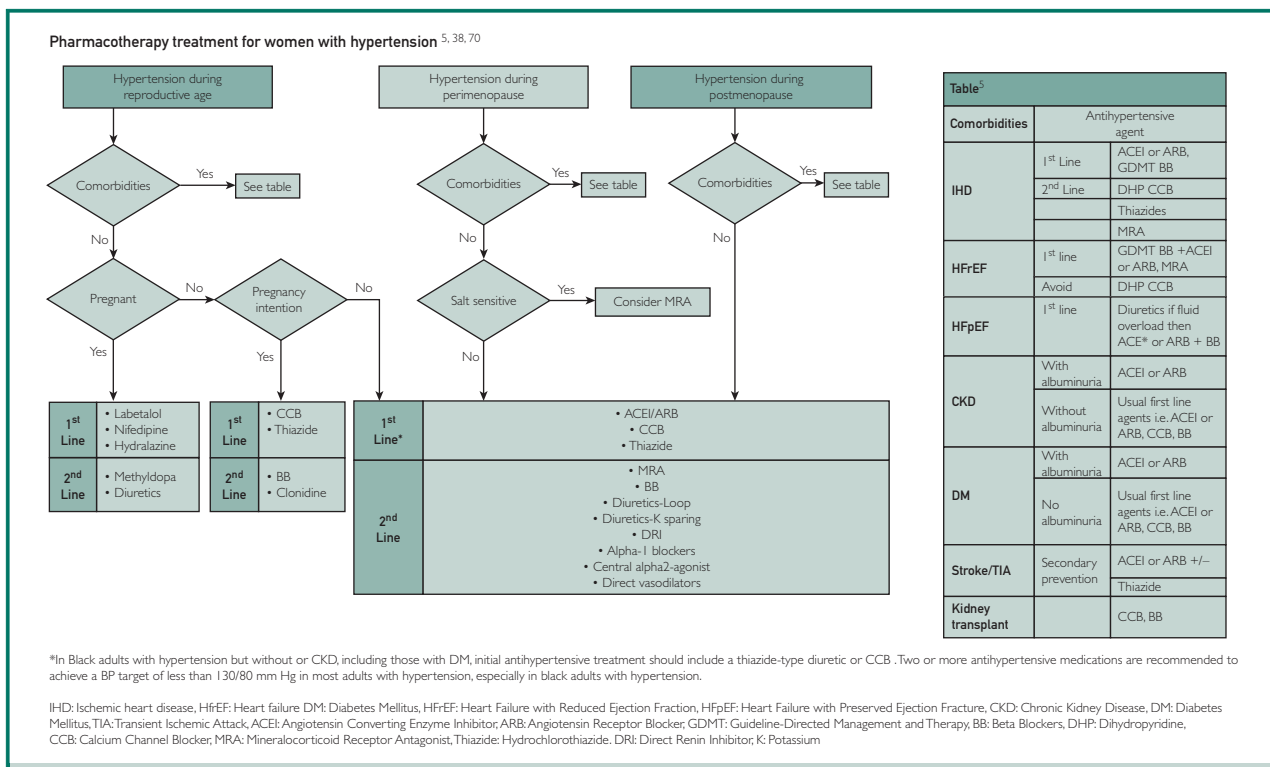


FIGURE 2. Treatment algorithm for antihypertensive medication in women during three phases: reproductive, perimenopause, and post-menopause and relation to comorbidities.^{5,32,55,79} ACE, angiotensin-converting enzyme; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BB, beta-blocker; CCB, calcium channel blocker; DHP, dihydropyridine; GDMT, guideline-directed medical therapy.

promising interventions that can reduce the effects of discrimination on health, such as religious involvement, value affirmation, forgiveness, and racism counter marketing.¹⁴⁸

PHARMACOTHERAPY TREATMENT FOR WOMEN WITH HYPERTENSION

Antihypertensive therapy choices vary among women across different phases of life. Therapy choices are limited during the reproductive age including the preconception phase and during pregnancy given the risk of teratogenicity and the side effects on the fetus. This limits access to RAAS blockade agents for patients with diabetes, CKD, or cardiac disease.^{55,79} Therapy choices during perimenopause and post

menopause are more similar to those with men in relation to comorbidities; there is greater attention to MR antagonists as a preferential treatment for premenopausal women who have been diagnosed with salt-sensitive hypertension with recent evidence that aldosterone production is sex-specifically heightened in salt-sensitive hypertensive women.^{5,32} Figure 2 summarizes pharmacotherapy treatment for women across three phases of life.^{5,32,55,79} Adverse effects of antihypertensive therapy can be higher in women than men with some class of medications such as ACE inhibitor–induced cough or edema with calcium antagonists. Table 3 summarizes sex-specific adverse events of antihypertensive agents.^{5,149,150}

TABLE 3. Sex-Specific Adverse Events of Antihypertensive Agents

Antihypertensive agent	Sex-specific adverse events	Additional benefit
Angiotensin-converting enzyme inhibitor	Potential risk in women in childbearing ages due to risk of fetal abnormalities Three-fold higher cough	Suitable in patients with comorbidities such as chronic kidney disease, coronary artery disease, congestive heart failure, diabetes mellitus, proteinuria
Angiotensin receptor blocker	Potential risk in women in childbearing ages due to risk of fetal abnormalities	Migraine prevention
Dihydropyridine calcium channel blocker	More peripheral edema in younger women	Suitable choice for Raynaud
Non-dihydropyridine calcium channel blocker	More peripheral edema in younger women	Verapamil for prevention of migraine Might improve proteinuria
Thiazide diuretics	Hyponatremia and hypokalemia more common in women Decreases placental perfusion, and plasma volume and association with pre-eclampsia	Reduce risk of osteoporosis Beneficial in calcium containing kidney stones
Mineralocorticoid receptor antagonist	Feminization of male fetus Oligohydramnios Failure of kidney development Ambiguous genitalia in newborns	Resistant hypertension Suitable in patients with comorbidities such as primary hyperaldosteronism, congestive heart failure, potassium wasting tubulopathies. Beneficial in androgen dependent conditions such as acne, hirsutism, polycystic ovarian syndrome Spironolactone reduces testosterone and is useful in male-to-female transgender
β-blockers	Sexual dysfunction	Migraine prevention Beneficial in patients with comorbidities such as ischemic heart diseases, coronary artery disease, congestive heart failure, atrial fibrillation
Minoxidil	Hirsutism	Resistant hypertension

From Whelton et al⁵, Bager et al¹⁴⁹, and Ahmad et al.¹⁵⁰

CONCLUSION

There is a significant clinical need for the diagnosis and optimal management of hypertension in women across the lifespan. With the continued growth of hypertension in women and the anticipated increase in prevalence of HDPs due to the obesity epidemic, increasing maternal age, and the rising prevalence of metabolic syndrome, additional strategies in a multidisciplinary fashion are needed to address unmet needs.

POTENTIAL COMPETING INTERESTS

All authors report no potential competing interests.

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SUPPLEMENTAL ONLINE MATERIAL

Supplemental material can be found online at <http://www.mayoclinicproceedings.org>. Supplemental material attached to journal articles has not been edited, and the authors take responsibility for the accuracy of all data.

Abbreviations and Acronyms: **ABPM**, ambulatory blood pressure monitor; **ACC**, American College of Cardiology; **AHA**, American Heart Association; **BMI**, body mass index; **BP**, blood pressure; **CAD**, coronary artery disease; **CKD**, chronic kidney disease; **CVD**, cardiovascular disease; **DASH**, Dietary Approaches to Stop Hypertension; **HDP**, hypertension disorders of pregnancy; **HMOD**, hypertension-mediated organ damage; **MR**, mineralocorticoid receptor; **OSA**, obstructive sleep apnea; **PA**, primary aldosteronism; **PRA**, plasma renin activity; **PRC**, plasma renin concentration; **RAAS**, renin-angiotensin-aldosterone system; **WC**, waist circumference; **WHtR**, waist-to-height ratio

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REFERENCES

1. NCD Risk Factor Collaboration. Worldwide trends in hypertension prevalence and progress in treatment and control from 1990 to 2019: a pooled analysis of 1201 population-representative studies with 104 million participants. *Lancet*. 2021;398(10304):957-980. [https://doi.org/10.1016/S0140-6736\(21\)01330-1](https://doi.org/10.1016/S0140-6736(21)01330-1)
2. Hearts M. Estimated Hypertension Prevalence, Treatment, and Control Among U.S. Adults. Centers for Disease Control and Prevention. <https://millionhearts.hhs.gov/data-reports/hypertension-prevalence.html>
3. Tsao CW, Aday AW, Almarazooq ZI, et al. Heart disease and stroke statistics-2023 update: a report from the American Heart Association. *Circulation*. 2023;147(8):e93-e621. <https://doi.org/10.1161/CIR.0000000000001123>
4. Connelly PJ, Cumie G, Delles C. Sex differences in the prevalence, outcomes and management of hypertension. *Curr Hypertens Rep*. 2022;24(6):185-192. <https://doi.org/10.1007/s11906-022-01183-8>
5. Whelton PK, Carey RM, Aronow WS, et al. 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: executive summary: a report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines. *Hypertension*. 2018;71(6):1269-1324. <https://doi.org/10.1161/HYP.0000000000000066>
6. Williams B, Mancia G, Spiering W, et al. 2018 Practice guidelines for the management of arterial hypertension of the European Society of Hypertension and the European Society of Cardiology: ESH/ESC task force for the management of arterial hypertension. *J Hypertens*. 2018;36(12):2284-2309. <https://doi.org/10.1097/HJH.0000000000001961>
7. Ostchega YFC, Nwankwo T, Nguyen DT. Hypertension prevalence among adults aged 18 and over: United States, 2017–2018. *NCHS Data Brief*. 2020;(364):1-8.
8. Fagard RH, Celis H, Thijs L, et al. Daytime and nighttime blood pressure as predictors of death and cause-specific cardiovascular events in hypertension. *Hypertension*. 2008;51(1):55-61. <https://doi.org/10.1161/HYPERTENSIONAHA.107.100727>
9. Hansen TW, Kikuya M, Thijs L, et al. Prognostic superiority of daytime ambulatory over conventional blood pressure in four populations: a meta-analysis of 7,030 individuals. *J Hypertens*. 2007;25(8):1554-1564. <https://doi.org/10.1097/HJH.0b013e3281c49da5>
10. Yano Y, Kario K. Nocturnal blood pressure and cardiovascular disease: a review of recent advances. *Hypertens Res*. 2012; 35(7):695-701. <https://doi.org/10.1038/hr.2012.26>
11. Salles GF, Reboldi G, Fagard RH, et al. Prognostic effect of the nocturnal blood pressure fall in hypertensive patients: the Ambulatory Blood Pressure Collaboration in Patients With Hypertension (ABC-H) Meta-Analysis. *Hypertension*. 2016; 67(4):693-700. <https://doi.org/10.1161/HYPERTENSIONAHA.115.06981>
12. Hansen TW, Li Y, Boggia J, Thijs L, Richart T, Staessen JA. Predictive role of the nighttime blood pressure. *Hypertension*. 2011;57(1):3-10. <https://doi.org/10.1161/HYPERTENSIONAHA.109.133900>
13. Yano Y, Tanner RM, Sakhuja S, et al. Association of daytime and nighttime blood pressure with cardiovascular disease events among African American individuals. *JAMA Cardiol*. 2019;4(9):910-917. <https://doi.org/10.1001/jamacardio.2019.2845>
14. Boggia J, Li Y, Thijs L, et al. Prognostic accuracy of day versus night ambulatory blood pressure: a cohort study. *Lancet*. 2007; 370(9594):1219-1229. [https://doi.org/10.1016/S0140-6736\(07\)61538-4](https://doi.org/10.1016/S0140-6736(07)61538-4)
15. Yan B, Peng L, Han D, et al. Blood pressure reverse-dipping is associated with early formation of carotid plaque in senior

- hypertensive patients. *Medicine (Baltimore)*. 2015;94(10):e604. <https://doi.org/10.1097/MD.0000000000000604>
16. Visniauskas B, Kilanowski-Doroh I, Ogola BO, et al. Estrogen-mediated mechanisms in hypertension and other cardiovascular diseases. *J Hum Hypertens*. 2023;37(8):609-618. <https://doi.org/10.1038/s41371-022-00771-0>
 17. Verdecchia P, Schillaci G, Borgioni C, et al. Gender, day-night blood pressure changes, and left ventricular mass in essential hypertension. Dippers and peakers. *Am J Hypertens*. 1995; 8(2):193-196. [https://doi.org/10.1016/0895-7061\(94\)00187-G](https://doi.org/10.1016/0895-7061(94)00187-G)
 18. Matangi MAD, Cases M, Zhu T, Brouillard D. Sex differences in diastolic blood pressure changes with age using 24-hour ABPM in 30,513 patients. *Eur Heart J*. 2020;41(2). <https://doi.org/10.1093/ehjci/ehaa946.3166>
 19. Gorostidi M, Sarafidis PA, de la Sierra A, et al. Differences between office and 24-hour blood pressure control in hypertensive patients with CKD: a 5,693-patient cross-sectional analysis from Spain. *Am J Kidney Dis*. 2013;62(2):285-294. <https://doi.org/10.1053/j.ajkd.2013.03.025>
 20. Pugliese DN, Booth JN 3rd, Deng L, et al. Sex differences in masked hypertension: the Coronary Artery Risk Development in Young Adults study. *J Hypertens*. 2019;37(12):2380-2388. <https://doi.org/10.1097/HJH.0000000000002175>
 21. Wang YC, Shimbo D, Muntner P, Moran AE, Krakoff LR, Schwartz JE. Prevalence of masked hypertension among US adults with nonelevated clinic blood pressure. *Am J Epidemiol*. 2017;185(3):194-202. <https://doi.org/10.1093/aje/kww237>
 22. Sundstrom J, Neovius M, Tynelius P, Rasmussen F. Association of blood pressure in late adolescence with subsequent mortality: cohort study of Swedish male conscripts. *BMJ*. 2011;342: d643. <https://doi.org/10.1136/bmj.d643>
 23. de Silva T, Cosentino G, Ganji S, Riera-Gonzalez A, Hsia DS. Endocrine causes of hypertension. *Curr Hypertens Rep*. 2020; 22(11):97. <https://doi.org/10.1007/s11906-020-01108-3>
 24. Hannemann A, Wallaschofski H. Prevalence of primary aldosteronism in patient's cohorts and in population-based studies — a review of the current literature. *Horm Metab Res*. 2012; 44(3):157-162. <https://doi.org/10.1055/s-0031-1295438>
 25. Douma S, Petidis K, Doumas M, et al. Prevalence of primary hyperaldosteronism in resistant hypertension: a retrospective observational study. *Lancet*. 2008;371(9628):1921-1926. [https://doi.org/10.1016/S0140-6736\(08\)60834-X](https://doi.org/10.1016/S0140-6736(08)60834-X)
 26. Young WF Jr. Diagnosis and treatment of primary aldosteronism: practical clinical perspectives. *J Intern Med*. 2019; 285(2):126-148. <https://doi.org/10.1111/joim.12831>
 27. Akasaka H, Yamamoto K, Rakugi H, et al. Sex difference in the association between subtype distribution and age at diagnosis in patients with primary aldosteronism. *Hypertension*. 2019; 74(2):368-374. <https://doi.org/10.1161/HYPERTENSIONAHA.119.13006>
 28. Pimenta E. Hypertension in women. *Hypertens Res*. Feb 2012; 35(2):148-152. <https://doi.org/10.1038/hr.2011.190>
 29. Broder MS, Neary MP, Chang E, Cherepanov D, Ludlam WH. Incidence of Cushing's syndrome and Cushing's disease in commercially insured patients <65 years old in the United States. *Pituitary*. 2015;18(3):283-289. <https://doi.org/10.1007/s1102-014-0569-6>
 30. Parksook WVV, Williams GH. Challenges and Approach to Identifying Individuals with Salt Sensitivity of Blood Pressure. *Am J Nephrol*. 2022;53(11-12):847-855. <https://doi.org/10.1159/000529057>
 31. He J, Gu D, Chen J, et al. Gender difference in blood pressure responses to dietary sodium intervention in the GenSalt study. *J Hypertens*. 2009;27(1):48-54. <https://doi.org/10.1097/hjh.0b013e328316bb87>
 32. Faulkner JL, Belin de Chantemele EJ. Female sex, a major risk factor for salt-sensitive hypertension. *Curr Hypertens Rep*. 2020;22(12):99. <https://doi.org/10.1007/s11906-020-01113-6>
 33. van Twist DJ, Houben AJ, de Haan MW, de Leeuw PW, Kroon AA. Pathophysiological differences between multifocal fibromuscular dysplasia and atherosclerotic renal artery stenosis. *J Hypertens*. 2017;35(4):845-852. <https://doi.org/10.1097/HJH.0000000000001243>
 34. Olin JW, Froehlich J, Gu X, et al. The United States Registry for Fibromuscular Dysplasia: results in the first 447 patients. *Circulation*. 2012;125(25):3182-3190. <https://doi.org/10.1161/CIRCULATIONAHA.112.091223>
 35. Rossi GP, Bernini G, Caliumi C, et al. A prospective study of the prevalence of primary aldosteronism in 1,125 hypertensive patients. *J Am Coll Cardiol*. 2006;48(11):2293-2300. <https://doi.org/10.1016/j.jacc.2006.07.059>
 36. Darrell Hulisz R, Pham D. Drug-induced hypertension. *US Pharm*. 2008;33(9):HS11-HS20. Accessed April 24, 2024. <https://www.uspharmacist.com/article/drug-induced-hypertension>
 37. Gabbay IE, Lavie P. Age- and gender-related characteristics of obstructive sleep apnea. *Sleep Breath*. 2012;16(2):453-460. <https://doi.org/10.1007/s11325-011-0523-z>
 38. Enright PL, Newman AB, Wahl PW, Manolio TA, Haponik EF, Boyle PJ. Prevalence and correlates of snoring and observed apneas in 5,201 older adults. *Sleep*. 1996;19(7):531-538. <https://doi.org/10.1093/sleep/19.7.531>
 39. Al Subhi AR, Boyle V, Elston MS. Systematic review: incidence of pheochromocytoma and paraganglioma over 70 years. *J Endocr Soc*. 2022;6(9):bvac105. <https://doi.org/10.1210/endo/bvac105>
 40. Vanderpump MP. The epidemiology of thyroid disease. *Br Med Bull*. 2011;99:39-51. <https://doi.org/10.1093/bmb/ldr030>
 41. Yeh MW, Ituarte PH, Zhou HC, et al. Incidence and prevalence of primary hyperparathyroidism in a racially mixed population. *J Clin Endocrinol Metab*. 2013;98(3):1122-1129. <https://doi.org/10.1210/jc.2012-4022>
 42. Burton T, Le Nestour E, Neary M, Ludlam WH. Incidence and prevalence of acromegaly in a large US health plan database. *Pituitary*. 2016;19(3):262-267. <https://doi.org/10.1007/s1102-015-0701-2>
 43. ACOG Committee Opinion No. 762: Prepregnancy Counseling. *Obstet Gynecol*. 2019;133(1):e78-e89. <https://doi.org/10.1097/AOG.0000000000003013>
 44. Magro-Malosso ER, Saccone G, Di Tommaso M, Roman A, Berghella V. Exercise during pregnancy and risk of gestational hypertensive disorders: a systematic review and meta-analysis. *Acta Obstet Gynecol Scand*. Aug 2017;96(8):921-931. <https://doi.org/10.1111/aogs.13151>
 45. Thangaratnam S, Rogozinska E, Jolly K, et al. Effects of interventions in pregnancy on maternal weight and obstetric outcomes: meta-analysis of randomised evidence. *BMJ*. 2012;344: e2088. <https://doi.org/10.1136/bmj.e2088>
 46. Burgner A, Hladunewich MA. Contraception and CKD. *Clin J Am Soc Nephrol*. 2020;15(4):563-565. <https://doi.org/10.2215/CJN.09770819>
 47. Harris PW. Malignant hypertension associated with oral contraceptives. *Lancet*. 1969;2(7618):466-467. [https://doi.org/10.1016/S0140-6736\(69\)90169-x](https://doi.org/10.1016/S0140-6736(69)90169-x)
 48. Kang AK, Duncan JA, Cattran DC, et al. Effect of oral contraceptives on the renin angiotensin system and renal function. *Am J Physiol Regul Integr Comp Physiol*. 2001;280(3):R807-R813. <https://doi.org/10.1152/ajpregu.2001.280.3.R807>
 49. Cameron NA, Blyler CA, Bello NA. Oral contraceptive pills and hypertension: a review of current evidence and recommendations. *Hypertension*. 2023;80(5):924-935. <https://doi.org/10.1161/HYPERTENSIONAHA.122.20018>
 50. Topel ML, Duncan EM, Krishna I, Badell ML, Vaccarino V, Quyyumi AA. Estimated impact of the 2017 American College of Cardiology/American Heart Association Blood Pressure guidelines on reproductive-aged women. *Hypertension*. 2018;72(4):e39-e42. <https://doi.org/10.1161/HYPERTENSIONAHA.118.11660>

51. CDC contraceptive guidance for health care providers—classifications for intrauterine devices. Accessed April. 2024;25. <https://www.cdc.gov/reproductivehealth/contraception/mmw/mec/appendixb.html>
52. ACOG Practice Bulletin No. 202: Gestational Hypertension and Preeclampsia. *Obstet Gynecol.* 2019;133(1):1. <https://doi.org/10.1097/AOG.0000000000003018>
53. Stuart JJ, Tanz LJ, Cook NR, et al. Hypertensive disorders of pregnancy and 10-year cardiovascular risk prediction. *J Am Coll Cardiol.* 2018;72(11):1252-1263. <https://doi.org/10.1016/j.jacc.2018.05.077>
54. Garovic VD, Dechend R, Easterling T, et al. Hypertension in pregnancy: diagnosis, blood pressure goals, and pharmacotherapy: a scientific statement from the American Heart Association. *Hypertension.* 2022;79(2):e21-e41. <https://doi.org/10.1161/HYP.000000000000208>
55. American College of Obstetricians and Gynecologists' Committee on Practice Bulletins—Obstetrics. ACOG practice bulletin no. 203: chronic hypertension in pregnancy. *Obstet Gynecol.* 2019;133(1):e26-e50. <https://doi.org/10.1097/AOG.0000000000003020>
56. Gestational Hypertension and Preeclampsia: ACOG Practice Bulletin. Number 222. *Obstet Gynecol.* 2020;135(6):e237-e260. <https://doi.org/10.1097/AOG.0000000000003891>
57. Tita AT, Szychowski JM, Boggess K, et al. Treatment for mild chronic hypertension during pregnancy. *N Engl J Med.* 2022;386(19):1781-1792. <https://doi.org/10.1056/NEJMoa2201295>
58. Bateman BT, Bansil P, Hernandez-Diaz S, Mhyre JM, Callaghan WM, Kuklina EV. Prevalence, trends, and outcomes of chronic hypertension: a nationwide sample of delivery admissions. *Am J Obstet Gynecol.* 2012;206(2):134.e1-8. <https://doi.org/10.1016/j.ajog.2011.10.878>
59. Malek AM, Wilson DA, Turan TN, Mateus J, Lackland DT, Hunt KJ. Maternal coronary heart disease, stroke, and mortality within 1, 3, and 5 years of delivery among women with hypertensive disorders of pregnancy and pre-pregnancy hypertension. *J Am Heart Assoc.* 2021;10(5):e018155. <https://doi.org/10.1161/JAHA.120.018155>
60. Burger RJ, Gordijn SJ, Bolijn R, et al. Cardiovascular risk profile after a complicated pregnancy across ethnic groups: the HELIUS study. *Eur J Prev Cardiol.* 2022. <https://doi.org/10.1093/eurjpc/zwac307>
61. Teal EN, Appiagyei A, Sheffield-Abdullah K, Manuck TA. Differences in disease severity and delivery gestational age between black and white patients with hypertensive disorders of pregnancy. *Pregnancy Hypertens.* 2022;28:88-93. <https://doi.org/10.1016/j.preghy.2022.03.001>
62. Young J. The etiology of eclampsia and albuminuria and their relation to accidental haemorrhage. *Trans Edinb Obstet Soc.* 1914;39:153-202.
63. McMaster-Fay RA. Uteroplacental vascular syndromes: theories, hypotheses and controversies. *Clin Obstet Gynecol Reprod Med.* 2018;4(6):1-5. <https://doi.org/10.15761/COG.RM.1000239>
64. Melchiorre K, Giorgione V, Thilaganathan B. The placenta and preeclampsia: villain or victim? *Am J Obstet Gynecol.* 2022;226(25):S954-S962. <https://doi.org/10.1016/j.ajog.2020.10.024>
65. Jung E, Romero R, Yeo L, et al. The etiology of preeclampsia. *Am J Obstet Gynecol.* 2022;226(25):S844-S866. <https://doi.org/10.1016/j.ajog.2021.11.1356>
66. Rana S, Burke SD, Karumanchi SA. Imbalances in circulating angiogenic factors in the pathophysiology of preeclampsia and related disorders. *Am J Obstet Gynecol.* 2022;226(25):S1019-S1034. <https://doi.org/10.1016/j.ajog.2020.10.022>
67. Mantel A, Sandstrom A, Faxen J, et al. Pregnancy-induced hypertensive disorder and risks of future ischemic and nonischemic heart failure. *JACC Heart Fail.* 2023;11(9):1228. <https://doi.org/10.1016/j.jchf.2023.03.021>
68. Barrett PM, McCarthy FP, Evans M, et al. Hypertensive disorders of pregnancy and the risk of chronic kidney disease: a Swedish registry-based cohort study. *PLoS Med.* 2020;17(8):e1003255. <https://doi.org/10.1371/journal.pmed.1003255>
69. Ackerman-Banks CM, Lipkind HS, Palmsten K, Ahrens KA. Association between hypertensive disorders of pregnancy and cardiovascular diseases within 24 months after delivery. *Am J Obstet Gynecol.* 2023;229(1):65.e1-65.e15. <https://doi.org/10.1016/j.ajog.2023.04.006>
70. Grandi SM, Filion KB, Yoon S, et al. Cardiovascular disease—related morbidity and mortality in women with a history of pregnancy complications. *Circulation.* 2019;139(8):1069-1079. <https://doi.org/10.1161/CIRCULATIONAHA.118.036748>
71. Cain MA, Salemi JL, Tanner JP, Kirby RS, Salihi HM, Louis JM. Pregnancy as a window to future health: maternal placental syndromes and short-term cardiovascular outcomes. *Am J Obstet Gynecol.* 2016;215(4):484.e1-484.e14. <https://doi.org/10.1016/j.ajog.2016.05.047>
72. Hallum S, Basit S, Kamper-Jorgensen M, Sehested TSG, Boyd HA. Risk and trajectory of premature ischaemic cardiovascular disease in women with a history of pre-eclampsia: a nationwide register-based study. *Eur J Prev Cardiol.* 2023. <https://doi.org/10.1093/eurjpc/zwad003>
73. Whelton PK, Carey RM, Aronow WS, et al. 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. *J Am Coll Cardiol.* 2018;71(19):e127-e248. <https://doi.org/10.1016/j.jacc.2017.11.006>
74. Society for Maternal-Fetal Medicine, Publications Committee. Society for Maternal-Fetal Medicine statement: antihypertensive therapy for mild chronic hypertension in pregnancy — the Chronic Hypertension and Pregnancy trial. *Am J Obstet Gynecol.* 2022;227(2):B24-B27. <https://doi.org/10.1016/j.ajog.2022.04.011>
75. Webster K, Fishburn S, Maresh M, Findlay SC, Chappell LC, Committee G. Diagnosis and management of hypertension in pregnancy: summary of updated NICE guidance. *BMJ.* 2019;366:i5119. <https://doi.org/10.1136/bmj.i5119>
76. Brown MA, Magee LA, Kenny LC, et al. Hypertensive disorders of pregnancy: ISSHP classification, diagnosis, and management recommendations for international practice. *Hypertension.* 2018;72(1):24-43. <https://doi.org/10.1161/HYPERTENSIONAHA.117.10803>
77. Abalos E, Duley L, Steyn DW, Gialdini C. Antihypertensive drug therapy for mild to moderate hypertension during pregnancy. *Cochrane Database Syst Rev.* 2018;10(10):CD002252. <https://doi.org/10.1002/14651858.CD002252.pub4>
78. Halpern DG, Weinberg CR, Pinnelas R, Mehta-Lee S, Economy KE, Valente AM. Use of medication for cardiovascular disease during pregnancy: JACC State-of-the-Art Review. *J Am Coll Cardiol.* 2019;73(4):457-476. <https://doi.org/10.1016/j.jacc.2018.10.075>
79. Mehta LS, Wames CA, Bradley E, et al. Cardiovascular considerations in caring for pregnant patients: a scientific statement from the American Heart Association. *Circulation.* 2020;141(23):e884-e903. <https://doi.org/10.1161/CIR.00000000000000772>
80. Practice CoO. Committee Opinion No. 692: Emergent therapy for acute-onset, severe hypertension during pregnancy and the postpartum period. *Obstet Gynecol.* 2017;129(4):e90-e95. <https://doi.org/10.1097/AOG.0000000000002019>
81. Society for Maternal-Fetal Medicine, Gibson KS, Combs CA, et al. Society for Maternal-Fetal Medicine special statement: quality metric for timely postpartum follow-up after severe hypertension. *Am J Obstet Gynecol.* 2022;227(3):B2-B8. <https://doi.org/10.1016/j.ajog.2022.05.045>
82. Beardmore KS, Morris JM, Gallery ED. Excretion of antihypertensive medication into human breast milk: a systematic review. *Hypertens Pregnancy.* 2002;21(1):85-95. <https://doi.org/10.1081/PRG-120002912>

83. Sabbatini AR, Kararigas G. Estrogen-related mechanisms in sex differences of hypertension and target organ damage. *Biol Sex Differ*. 2020;11(1):31. <https://doi.org/10.1186/s13293-020-00306-7>
84. Srivaratharajah K, Abramson BL. Hypertension in menopausal women: the effect and role of estrogen. *Menopause*. 2019; 26(4):428-430. <https://doi.org/10.1097/GME.00000000000001304>
85. Gallagher PE, Li P, Lenhart JR, Chappell MC, Brosnihan KB. Estrogen regulation of angiotensin-converting enzyme mRNA. *Hypertension*. 1999;33(1 Pt 2):323-328. <https://doi.org/10.1161/01.hyp.33.1.323>
86. Gersh FL, O'Keefe JH, Lavie CJ, Henry BM. The renin-angiotensin-aldosterone system in postmenopausal women: the promise of hormone therapy. *Mayo Clin Proc*. 2021; 96(12):3130-3141. <https://doi.org/10.1016/j.mayocp.2021.08.009>
87. Alhurani RE, Chahal CA, Ahmed AT, Mohamed EA, Miller VM. Sex hormone therapy and progression of cardiovascular disease in menopausal women. *Clin Sci (Lond)*. 2016; 130(13):1065-1074. <https://doi.org/10.1042/CS20160042>
88. Salpeter SR, Walsh JM, Greyber E, Salpeter EE. Brief report: Coronary heart disease events associated with hormone therapy in younger and older women. A meta-analysis. *J Gen Intern Med*. 2006;21(4):363-366. <https://doi.org/10.1111/j.1525-1497.2006.00389.x>
89. Lemaitre RN, Weiss NS, Smith NL, et al. Esterified estrogen and conjugated equine estrogen and the risk of incident myocardial infarction and stroke. *Arch Intern Med*. 2006; 166(4):399-404. <https://doi.org/10.1001/archinte.166.4.399>
90. Yoon BK, Sung J, Song YM, et al. Effects of menopausal hormone therapy on ambulatory blood pressure and arterial stiffness in postmenopausal Korean women with grade 1 hypertension: a randomized, placebo-controlled trial. *Clin Hypertens*. 2021;27(1):18. <https://doi.org/10.1186/s40885-021-00175-1>
91. Lindsey SH. Importance of estrogen metabolites. *Hypertension*. 2014;64(1):21-22. <https://doi.org/10.1161/HYPERTENSIONAHA.114.03382>
92. Pingili AK, Davidge KN, Thirunavukkarasu S, et al. 2-Methoxyestradiol reduces angiotensin ii-induced hypertension and renal dysfunction in ovariectomized female and intact male mice. *Hypertension*. 2017;69(6):1104-1112. <https://doi.org/10.1161/HYPERTENSIONAHA.117.09175>
93. Baber RJ, Panay N, Fenton A, Group IMSW. 2016 IMS Recommendations on women's midlife health and menopause hormone therapy. *Climacteric*. 2016;19(2):109-150. <https://doi.org/10.3109/13697137.2015.1129166>
94. Gartlehner G, Patel SV, Feltner C, et al. Hormone therapy for the primary prevention of chronic conditions in postmenopausal women: evidence report and systematic review for the US Preventive Services Task Force. *JAMA*. 2017;318(22):2234-2249. <https://doi.org/10.1001/jama.2017.16952>
95. ACOG Committee Opinion No. 565: Hormone therapy and heart disease. *Obstet Gynecol*. 2013;121(6):1407-1410. <https://doi.org/10.1097/01.AOG.0000431053.33593.2d>
96. Cho L, Kaunitz AM, Faubion SS, et al. Rethinking menopausal hormone therapy: for whom, what, when, and how long? *Circulation*. 2023;147(7):597-610. <https://doi.org/10.1161/CIRCULATIONAHA.122.061559>
97. Boardman HM, Hartley L, Eisinga A, et al. Hormone therapy for preventing cardiovascular disease in post-menopausal women. *Cochrane Database Syst Rev*. 2015;2015(3):CD002229. <https://doi.org/10.1002/14651858.CD002229.pub4>
98. Stuenkel CA, Davis SR, Gompel A, et al. Treatment of symptoms of the menopause: an Endocrine Society Clinical Practice Guideline. *J Clin Endocrinol Metab*. Nov 2015;100(11):3975-4011. <https://doi.org/10.1210/jc.2015-2236>
99. Karalexi MA, Frisell T, Cnattingius S, et al. Cardiovascular outcomes in transgender individuals in Sweden after initiation of gender-affirming hormone therapy. *Eur J Prev Cardiol*. 2022; 29(15):2017-2026. <https://doi.org/10.1093/eurjpc/zwac133>
100. Martinez-Martin FJ, Kuzior A, Hernandez-Lazaro A, et al. Incidence of hypertension in young transgender people after a 5-year follow-up: association with gender-affirming hormonal therapy. *Hypertens Res*. 2023;46(1):219-225. <https://doi.org/10.1038/s41440-022-01067-z>
101. Banks K, Kyinn M, Leemaqz SY, Sarkodie E, Goldstein D, Irwig MS. Blood pressure effects of gender-affirming hormone therapy in transgender and gender-diverse adults. *Hypertension*. 2021;77(6):2066-2074. <https://doi.org/10.1161/HYPERTENSIONAHA.120.16839>
102. Cunha FS, Bacheга T, Costa EMF, et al. Arterial stiffness in transgender men receiving long-term testosterone therapy. *J Endocr Soc*. 2023;7(5):bvad040. <https://doi.org/10.1210/endo/bvad040>
103. Aggarwal R, Chiu N, Wadhwa RK, et al. Racial/ethnic disparities in hypertension prevalence, awareness, treatment, and control in the United States, 2013 to 2018. *Hypertension*. 2021;78(6):1719-1726. <https://doi.org/10.1161/HYPERTENSIONAHA.121.17570>
104. Egan BM. Editorial commentary: racial and ethnic disparities in hypertension prevalence, awareness, treatment, and control in the United States, 2013 to 2018. *Hypertension*. 2021;78(6):1727-1729. <https://doi.org/10.1161/HYPERTENSIONAHA.121.18023>
105. Ford ND, Robbins CL, Hayes DK, Ko JY, Loustalot F. Prevalence, treatment, and control of hypertension among US women of reproductive age by race/hispanic origin. *Am J Hypertens*. 2022;35(8):723-730. <https://doi.org/10.1093/ajh/hpac053>
106. Fryar CDOY, Hales CM, Zhang G, Kruszon-Moran D. Hypertension prevalence and control among adults: United States, 2015–2016. *NCHS Data Brief*. 2017;(289):1-8.
107. Gabriel A, Zare H, Jones W, et al. Evaluating depressive symptoms among low-socioeconomic-status African American women aged 40 to 75 years with uncontrolled hypertension: a secondary analysis of a randomized clinical trial. *JAMA Psychiatry*. 2021;78(4):426-432. <https://doi.org/10.1001/jamapsychiatry.2020.4622>
108. Kalinowski J, Kaur K, Newsome-Garcia V, et al. Stress interventions and hypertension in Black women. *Womens Health (Lond)*. 2021;17:17455065211009751. <https://doi.org/10.1177/17455065211009751>
109. Milani RV, Price-Haywood EG, Burton JH, Wilt J, Entwisle J, Lavie CJ. Racial differences and social determinants of health in achieving hypertension control. *Mayo Clin Proc*. 2022;97(8):1462-1471. <https://doi.org/10.1016/j.mayocp.2022.01.035>
110. Ritchey MD, Gillespie C, Wozniak G, et al. Potential need for expanded pharmacologic treatment and lifestyle modification services under the 2017 ACC/AHA hypertension guideline. *J Clin Hypertens (Greenwich)*. 2018;20(10):1377-1391. <https://doi.org/10.1111/jch.13364>
111. Appel LJ, Moore TJ, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. *N Engl J Med*. Apr 17 1997;336(16):1117-1124. <https://doi.org/10.1056/nejm199704173361601>
112. Chiavaroli L, Viguiouk E, Nishi SK, et al. DASH dietary pattern and cardiometabolic outcomes: an umbrella review of systematic reviews and meta-analyses. *Nutrients*. 2019;11(2):338. <https://doi.org/10.3390/nu11020338>
113. Kucharska A, Gajewska D, Kiedrowski M, et al. The impact of individualised nutritional therapy according to DASH diet on blood pressure, body mass, and selected biochemical parameters in overweight/obese patients with primary arterial hypertension: a prospective randomised study. *Kardiol Pol*. 2018; 76(1):158-165. <https://doi.org/10.5603/KP.a2017.0184>

114. Sadakane A, Tsutsumi A, Gotoh T, et al. Dietary patterns and levels of blood pressure and serum lipids in a Japanese population. *J Epidemiol*. 2008;18(2):58-67. <https://doi.org/10.2188/jea.18.58>
115. Song S, Kim J, Kim J. Gender differences in the association between dietary pattern and the incidence of hypertension in middle-aged and older adults. *Nutrients*. 2018;10(2):252. <https://doi.org/10.3390/nu10020252>
116. Mori TA, Burke V, Beilin LJ, Puddey IB. Randomized controlled intervention of the effects of alcohol on blood pressure in premenopausal women. *Hypertension*. 2015;66(3):517-523. <https://doi.org/10.1161/HYPERTENSIONAHA.115.05773>
117. Fisher NDL, Orav EJ, Chang G. Effects of alcohol consumption on blood pressure in hypertensive women. *Am J Drug Alcohol Abuse*. 2018;44(2):200-205. <https://doi.org/10.1080/00952990.2017.1355921>
118. Bowman TS, Gaziano JM, Buring JE, Sesso HD. A prospective study of cigarette smoking and risk of incident hypertension in women. *J Am Coll Cardiol*. 2007;50(21):2085-2092. <https://doi.org/10.1016/j.jacc.2007.08.017>
119. Cifkova R, Pitha J, Kraljcoviechova A, Kralikova E. Is the impact of conventional risk factors the same in men and women? Plea for a more gender-specific approach. *Int J Cardiol*. 2019;286:214-219. <https://doi.org/10.1016/j.ijcard.2019.01.039>
120. Prescott E, Osler M, Hein HO, Borch-Johnsen K, Schnohr P, Vestbo J. Life expectancy in Danish women and men related to smoking habits: smoking may affect women more. *J Epidemiol Community Health*. 1998;52(2):131-132. <https://doi.org/10.1136/jech.52.2.131>
121. Huang Z, Willett WC, Manson JE, et al. Body weight, weight change, and risk for hypertension in women. *Ann Intern Med*. 1998;128(2):81-88. <https://doi.org/10.7326/0003-4819-128-2-199801150-00001>
122. Manson JE, Willett WC, Stampfer MJ, et al. Body weight and mortality among women. *N Engl J Med*. 1995;333(11):677-685. <https://doi.org/10.1056/NEJM199509143331101>
123. Chobanian AV, Bakris GL, Black HR, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension*. 2003;42(6):1206-1252. <https://doi.org/10.1161/01.HYP.0000107251.49515.c2>
124. Caminha TC, Ferreira HS, Costa NS, et al. Waist-to-height ratio is the best anthropometric predictor of hypertension: A population-based study with women from a state of northeast of Brazil. *Medicine (Baltimore)*. 2017;96(2):e5874. <https://doi.org/10.1097/MD.0000000000005874>
125. Mansour AA, Ajeel NA. Parity is associated with increased waist circumference and other anthropometric indices of obesity. *Eat Weight Disord*. 2009;14(2-3):e50-e55. <https://doi.org/10.1007/BF03327800>
126. Wenger NK, Arnold A, Bairey Merz CN, et al. Hypertension across a woman's life cycle. *J Am Coll Cardiol*. 2018;71(16):1797-1813. <https://doi.org/10.1016/j.jacc.2018.02.033>
127. Forman JP, Stampfer MJ, Curhan GC. Diet and lifestyle risk factors associated with incident hypertension in women. *JAMA*. 2009;302(4):401-411. <https://doi.org/10.1001/jama.2009.1060>
128. MacDonald CJ, Madika AL, Lajous M, et al. Associations between physical activity and incident hypertension across strata of body mass index: a prospective investigation in a large cohort of French women. *J Am Heart Assoc*. 2020;9(23):e015121. <https://doi.org/10.1161/JAHA.119.015121>
129. https://www.cdc.gov/sleep/about_sleep/key_disorders.html
130. Nieto FJ, Young TB, Lind BK, et al. Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study. Sleep Heart Health Study. *JAMA*. 2000;283(14):1829-1836. <https://doi.org/10.1001/jama.283.14.1829>
131. Gangwisch JE, Feskanich D, Malaspina D, Shen S, Forman JP. Sleep duration and risk for hypertension in women: results from the nurses' health study. *Am J Hypertens*. Jul 2013; 26(7):903-911. <https://doi.org/10.1093/ajh/hpt044>
132. Valero M, Viney TJ, Machold R, et al. Sleep down state-active ID2/Nkx2.1 interneurons in the neocortex. *Nat Neurosci*. 2021;24(3):401-411. <https://doi.org/10.1038/s41593-021-00797-6>
133. Pedrosa RP, Barros IML, Drager LF, et al. OSA is common and independently associated with hypertension and increased arterial stiffness in consecutive perimenopausal women. *Chest*. 2014;146(1):66-72. <https://doi.org/10.1378/chest.14-0097>
134. Yong LC, Li J, Calvert GM. Sleep-related problems in the US working population: prevalence and association with shiftwork status. *Occup Environ Med*. 2017;74(2):93-104. <https://doi.org/10.1136/oemed-2016-103638>
135. Schaere HL, Blochl M, Kumral D, et al. Associations between mental health, blood pressure and the development of hypertension. *Nat Commun*. 2023;14(1):1953. <https://doi.org/10.1038/s41467-023-37579-6>
136. Johnson HM. Anxiety and hypertension: is there a link? a literature review of the comorbidity relationship between anxiety and hypertension. *Curr Hypertens Rep*. 2019;21(9):66. <https://doi.org/10.1007/s11906-019-0972-5>
137. Spruill TM. Chronic psychosocial stress and hypertension. *Curr Hypertens Rep*. 2010;12(1):10-16. <https://doi.org/10.1007/s11906-009-0084-8>
138. Black PH, Garbutt LD. Stress, inflammation and cardiovascular disease. *J Psychosom Res*. 2002;52(1):1-23. [https://doi.org/10.1016/s0022-3999\(01\)00302-6](https://doi.org/10.1016/s0022-3999(01)00302-6)
139. Everson-Rose SA, Lewis TT. Psychosocial factors and cardiovascular diseases. *Annu Rev Public Health*. 2005;26:469-500. <https://doi.org/10.1146/annurev.publhealth.26.021304.144542>
140. Jackson CA, Pathirana T, Gardiner PA. Depression, anxiety and risk of hypertension in mid-aged women: a prospective longitudinal study. *J Hypertens*. 2016;34(10):1959-1966. <https://doi.org/10.1097/HJH.0000000000001030>
141. Ho AK, Thorpe CT, Pandhi N, Palta M, Smith MA, Johnson HM. Association of anxiety and depression with hypertension control: a US multidisciplinary group practice observational study. *J Hypertens*. 2015;33(11):2215-2222. <https://doi.org/10.1097/HJH.0000000000000693>
142. Polanka BM, Bertson J, Vraney EA, Stewart JC. Are cardiovascular risk factors stronger predictors of incident cardiovascular disease in u.s. adults with versus without a history of clinical depression? *Ann Behav Med*. 2018;52(12):1036-1045. <https://doi.org/10.1093/abm/kay007>
143. Watkins DC, Assari S, Johnson-Lawrence V. Race and ethnic group differences in comorbid major depressive disorder, generalized anxiety disorder, and chronic medical conditions. *J Racial Ethn Health Disparities*. 2015;2(3):385-394. <https://doi.org/10.1007/s40615-015-0085-z>
144. Ebrahimi R, Lynch KE, Beckham JC, et al. Association of post-traumatic stress disorder and incident ischemic heart disease in women veterans. *JAMA Cardiol*. 2021;6(6):642-651. <https://doi.org/10.1001/jamacardio.2021.0227>
145. El Khoudary SR, Greendale G, Crawford SL, et al. The menopause transition and women's health at midlife: a progress report from the study of women's health across the nation (SWAN). *Menopause*. 2019;26(10):1213-1227. <https://doi.org/10.1097/GME.0000000000001424>
146. Rainforth MV, Schneider RH, Nidich SI, Gaylord-King C, Salerno JW, Anderson JW. Stress reduction programs in patients with elevated blood pressure: a systematic review and meta-analysis. *Curr Hypertens Rep*. 2007;9(6):520-528. <https://doi.org/10.1007/s11906-007-0094-3>
147. Ludwig J, Sanbonmatsu L, Genetian L, et al. Neighborhoods, obesity, and diabetes — a randomized social experiment. *N Engl J Med*. 2011;365(16):1509-1519. <https://doi.org/10.1056/NEJMsal103216>

148. Lewis TT, Cogburn CD, Williams DR. Self-reported experiences of discrimination and health: scientific advances, ongoing controversies, and emerging issues. *Annu Rev Clin Psychol*. 2015;11:407-440. <https://doi.org/10.1146/annurev-clinpsy-032814-112728>
149. Bager JE, Manhem K, Andersson T, et al. Hypertension: sex-related differences in drug treatment, prevalence and blood pressure control in primary care. *J Hum Hypertens*. 2023;37(8):662-670. <https://doi.org/10.1038/s41371-023-00801-5>
150. Ahmad A, Oparil S. Hypertension in women: recent advances and lingering questions. *Hypertension*. 2017;70(1):19-26. <https://doi.org/10.1161/HYPERTENSIONAHA.117.08317>