



Advancing Maternal Health:

Closing the Gaps in Cardiovascular Care

— A Toolkit for Health Care Professionals

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Disclosures: Nothing to disclose



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Disclosures: Nothing to disclose



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Relationship disclosures within the past 24 months:

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Advancing Maternal Health



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Introduction

Cardiovascular disease (CVD) is the leading cause of death in women globally^{1-F} and the leading cause of pregnancy-related maternal mortality in the United States.^{1-A} Although maternal mortality has fallen globally in recent decades, the proportion of CVD-related maternal mortality is rising, especially in higher income areas. As many as two-thirds of CVD-related pregnancy deaths could be prevented by bridging gaps in care with appropriate surveillance, prevention and treatment strategies from preconception to a reimagined fourth trimester of postpartum care.^{1-F, A; 2-A, B}

CVD presents and is treated differently in men and women due to underlying biologic differences between the sexes and underutilization of evidence-based approaches.^{3-A} Women are at particular risk for CVD during and after pregnancy. Even uncomplicated pregnancies and deliveries are a physical and psychological stress test for every organ system.^{1-B} The maternal mortality rate in the United States is among the highest of all high-income countries, with the largest toll affecting American Indian, Alaska Native and Black women. CVD disproportionately affects these same populations, largely due to the higher prevalence of CVD risk factors.^{4-A}

The normal physiology of pregnancy increases CVD risk. Cardiac output increases by 30% to 50% with increased stroke volume and heart rate.^{1-B} Pregnancy increases the risk of acute myocardial infarction three to fourfold as well as acute cardiac syndrome and cardiomyopathy.^{3-A, B, C} A pregnant person's blood vessels can remodel, and certain measurements can increase, including plasma volume, hypercoagulability, LDL cholesterol, triglycerides, insulin resistance and vascular dysfunction.^{3-C}

The increased CVD risk often presents as one or more **hypertensive disorders of pregnancy (HDP)**, including chronic hypertension, gestational hypertension, preeclampsia/eclampsia, preeclampsia superimposed on chronic hypertension and HELLP (hemolysis, elevated liver enzymes and low platelet count) syndrome. HDP occurs in about 10% of pregnancies^{9-A} and is the second leading cause of maternal mortality after maternal hemorrhage. It is also an important cause of short- and long-term maternal and fetal/offspring morbidity and mortality.^{4-B}

Many women undergo the stress of pregnancy and delivery without significant residual effects, but population-wide increases in underlying risk factors, such as chronic hypertension, obesity, older age at pregnancy, diabetes and tobacco use, can heighten individual risk for **adverse pregnancy outcomes (APOs)**, including preterm birth, gestational diabetes, preeclampsia, gestational hypertension, small-for-gestational-age infant and placental



Cardio-obstetrics — closing the gaps in care between pregnancy, delivery and the remaining decades of life — is the next frontier in CVD prevention and treatment for women.^{1-E, D}

abruption. APOs are associated with adverse maternal events, including arrhythmia, heart failure, peripartum cardiomyopathy, valvular disorders, cerebrovascular accidents, coronary artery disease,^{1-B, C} stroke and chronic kidney disease.^{5-A}

In addition to adverse maternal outcomes, fetal exposure to HDP and/or APOs is associated with increased risk of CVD and CVD mortality,^{6-A, B} hypertension, diabetes and dyslipidemia for offspring later in life.^{6-C; 5-B} Elevated maternal systolic **blood pressure (BP)** during pregnancy, even at levels below accepted thresholds for hypertension, are associated with increased risk for preterm delivery and infants who are small-for-gestational age and low birth weight.^{4-C}



Maternal hypertension develops more frequently following HDP than after normotensive pregnancies. Women with HDP develop hypertension faster and are diagnosed up to 10 years earlier compared to women with normotensive pregnancies. Cardiometabolic risk factors and CVD events develop earlier following HDP, and women with HDP have higher rates of accumulated chronic conditions and comorbidities. HDP may also accelerate maternal aging.^{4-D}

In many cases, HDP can be mitigated or prevented by bridging gaps in care,^{2-B, C} implementing lifestyle changes in line with Life's Essential 8,^{7-A} taking appropriate medications to manage hypertension and other APOs^{8-A} and improving peripartum and postpartum care. Continuing follow-up into the fourth trimester^{2-D} — the first three months following delivery — presents an opportunity to engage women in early identification of CVD and CVD risk factors that may have developed as a result of pregnancy. Early involvement in cardioprotective strategies can delay or prevent CVD later in life.

Hypertensive Disorders of Pregnancy and Cardiovascular Disease

Current guidelines addressing CVD and females may include historical and unsubstantiated perspectives affecting women's health throughout their reproductive lives that should be avoided in future publications.^{4-B} Social determinants of health (SDOH) can also affect the quality, quantity and timing of CVD care.^{3-A; 4-A}

SDOH and ethnic/racial disparities in maternal health play major roles in maternal and offspring outcomes. Maternal mortality in the United States was 18/100,000 live births in 2012, among the highest of all high-income countries. The overall maternal mortality rate masks dramatic variations. The estimated maternal mortality rate in 2016 for white women was 13/100,000 live births compared to 30 for American Indian/Alaska Native women and 41 for Black women.^{4-A}

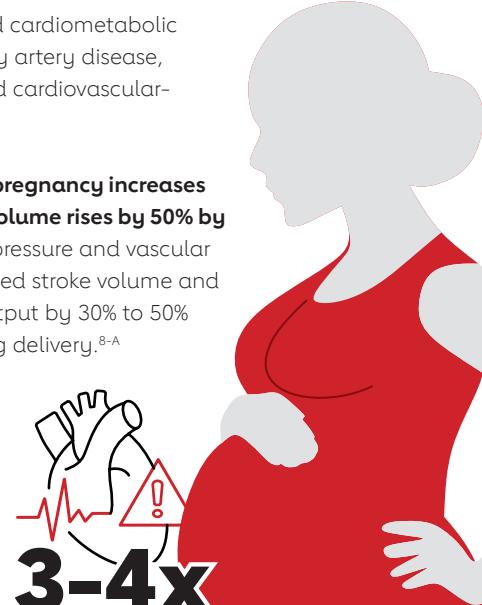
HDP disproportionately affects Black, American Indian and Alaska Native women, largely due to the higher prevalence of CVD risk factors. Severe morbidity and mortality related to preeclampsia are higher for Black women, while Hispanic women tend to have better pregnancy outcomes than either Black or white women with similar risk factors.^{4-A}

CVD outcomes in current or previously pregnant women are driven largely by the **hypertensive disorders of pregnancy (HDP)**. Women with a history of HDP have an elevated risk of

multiple cardiovascular and cardiometabolic diseases, including coronary artery disease, cerebrovascular disease and cardiovascular-related mortality.^{9-A}

The normal physiology of pregnancy increases CVD risk. Maternal blood volume rises by 50% by 32 weeks. Although blood pressure and vascular resistance decrease, increased stroke volume and heart rate boost cardiac output by 30% to 50% with further increases during delivery.^{8-A}

Pregnancy increases the risk of acute myocardial infarction three to fourfold as well as acute cardiac syndrome (ACS) and cardiomyopathy.

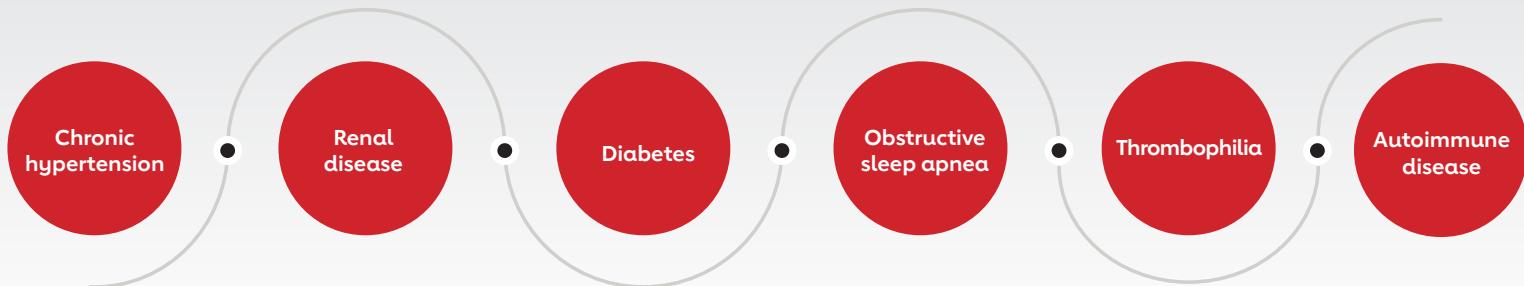


3-4x increased risk

Preeclampsia and eclampsia may contribute to ACS in pregnancy in addition to illegal drugs like cocaine and commonly used peripartum drugs, such as tocolytics and oxytocics.

About 10% of pregnancies are affected by hypertensive disorders of pregnancy.^{9-A}

Preexisting conditions that contribute to uteroplacental blood flow and vascular insufficiency increase risk for HDP, including:



Additional factors that put women at elevated risk for HDP are:

- Prior history of preeclampsia
- HELLP syndrome
- Twin or other multiple pregnancies
- Body mass index greater than 30
- Aged 35 years or more
- First-degree relative who had gestational hypertension.^{11-A}

Physiologic Changes in Pregnancy

Pregnancy and labor are hemodynamically taxing periods with multiple psychologic and physiologic stressors.^{3-A, B, C} Adapting to these changes can contribute to an adverse cardiometabolic risk profile during and after pregnancy.

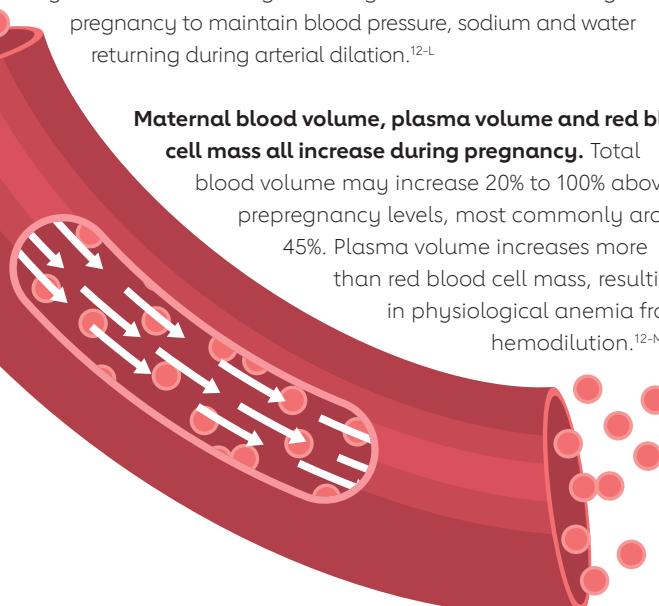


Pregnancy brings significant changes in hormonal levels, including increased estrogen and progesterone, which help to maintain early pregnancy and contribute to peripheral and systemic vasodilation.^{12-K} Systemic vasodilation begins as early as five weeks, and peripheral vascular resistance can fall 35% to 40% from baseline by the middle of the second trimester.^{12-A}



Vasodilation in the kidneys increases renal plasma flow and glomerular filtration rate up to 50% by the end of the first trimester, reducing serum creatinine, urea and uric acid. Levels that appear normal before pregnancy may indicate borderline kidney function during pregnancy.^{13-A;14-M}

Pregnancy activates the **renin-angiotensin-aldosterone system (RAAS)**, with plasma volume increasing as early as five to eight gestational weeks. Angiotensinogen levels increase during pregnancy to maintain blood pressure, sodium and water returning during arterial dilation.^{12-L}

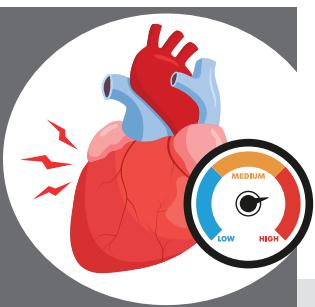


Maternal blood volume, plasma volume and red blood cell mass all increase during pregnancy. Total blood volume may increase 20% to 100% above prepregnancy levels, most commonly around 45%. Plasma volume increases more than red blood cell mass, resulting in physiological anemia from hemodilution.^{12-M, N}

Pregnancy can increase a person's heart rate, stroke volume and cardiac output. Heart rate can be expected to progressively increase 20% to 25% over baseline.^{12-I} Stroke volume increases gradually through the end of the second trimester and may decrease late in pregnancy.^{12-D} The sharpest rise in cardiac output is usually seen by the beginning of the first trimester and continues to increase into the second trimester.

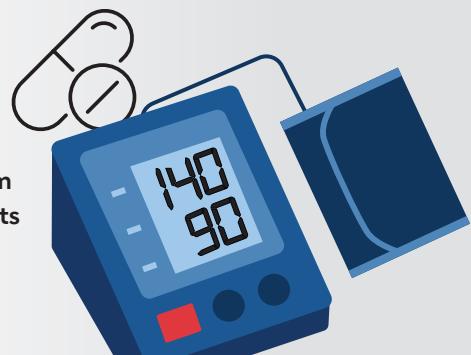


By 24 weeks, cardiac output can be up to 45% higher than prepregnancy levels in a normal, singleton pregnancy.^{12-C}



Both **systolic blood pressure (SBP)** and **diastolic blood pressure (DBP)** fall during pregnancy, with a greater reduction in DBP. Mean arterial pressure can fall 5–10 mmHg below baseline. Arterial pressure begins to increase during the third trimester and should return to near preconception levels postpartum.

Women on antihypertensive medications for any reason require regular BP monitoring to inform medication adjustments during and after pregnancy.^{12-E, 15-Table 4}



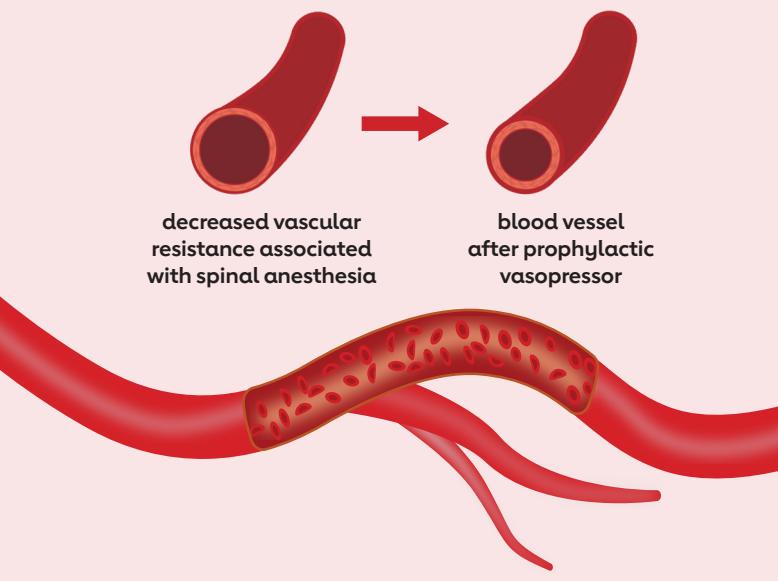


Expect maximum cardiac output during labor and immediately postpartum with 60% to 80% increase over levels before the onset of labor.



The sharply increased output is the result of increased heart rate and preload with the pain of uterine contractions, increased catecholamines and autotransfusion of 300–500 mL of blood from the uterus to systemic circulation with each contraction.^{12-Q}

Spinal anesthesia is associated with decreased vascular resistance with a corresponding increase in heart rate and stroke volume. Prophylactic vasopressors, such as phenylephrine, can prevent hypotension associated with spinal anesthesia.^{12-R}



Supine posture during later pregnancy may lead to compression of the **inferior vena cava (IVC)** by the uterus. Compression of the IVC decreases venous return and stroke volume, which leads to a drop in arterial blood pressure. The heart rate increases to restore cardiac output, which impairs diastolic filling time and may result in inadequate fetal perfusion.



Maternal activities should avoid supine positioning whenever possible, particularly after 20 weeks gestation.^{16-A} Fetal heart monitoring may guide maternal positioning during necessary medical procedures.^{17-A}

Pregnancy creates a **prothrombotic state** with changes in coagulation to support hemostatic control during pregnancy and delivery, when blood loss is expected. The rate and level of thrombin generation increases progressively during pregnancy and plasma fibrinogen increases by about 50%.^{14-A} Fibrin-degradation products also increase with the net changes favoring fibrin formation.

Antithrombin levels, including coagulation inhibitor proteins C and S, decrease throughout pregnancy, then return to baseline within 72 hours postpartum.^{14-A, B, C, D}

The risk of a thrombotic event remains elevated at least 12 weeks after delivery, with the greatest increase in absolute risk seen during the first six weeks.^{18-A}



Postpartum Changes Can Affect CVD Risk



Normal physiologic changes in pregnancy can enhance the pathogenesis of CVD in women with underlying risk factors. These underlying risk factors have become more common in recent years with more women having children later in life, and there are population-wide increases in traditional cardiometabolic risk factors, such as chronic hypertension, obesity or overweight, diabetes and tobacco use.^{1-8, C}

All these risk factors can increase maternal risk for HDP and adverse pregnancy outcomes (APOs), including preterm birth, gestational diabetes, preeclampsia, gestational hypertension, small-for-gestational age neonates and placental abruption.

Both HDP and APOs increase lifetime risk for poor cardiometabolic outcomes for both mother and child.

Below are hazard ratio increases as a result of HDP.^{4-Table 2}

Type 2 diabetes	1.8
Hyperlipemia	1.3
Coronary heart disease	1.7
Heart failure	2.7
Stroke	1.9
Venous thromboembolism	1.5

Women who develop gestational hypertension have:

40% higher risk for CVD

3x risk for vascular dementia

Nearly 4x risk for end-stage kidney disease

The maternal hazard ratio for **people with hypertension** equal to or greater than 140/90 mmHg **increases to 2.3** compared to pregnancy without HDP.



The offspring of women with HDP are at increased risk for CVD, stroke, obesity and hypertension.

Although any HDP increases the likelihood of maternal hypertension in the following years, women with recurrent HDP after an index HDP pregnancy are at higher risk of hypertension and other CVD compared to women with a single episode of HDP. Women may develop hypertension within months to years after HDP.

An episode of HDP can increase the risk of hypertension within two years postpartum more than sixfold. The risk of developing hypertension is highest in the first six months postpartum (18.33-fold), falling to 4.36-fold between six months and one year, then rebounding to 7.24-fold between one and two years postpartum. Women with preeclampsia are at even higher risk of hypertension, 57.08-fold, 4.83-fold and 7.44-fold at the same time periods.^{9-8, C}

HDP is the second leading cause of maternal mortality after maternal hemorrhage.⁴⁻⁸ The incidence of HDP is rising as a result of the increasing prevalence of obesity, advancing age at pregnancy and other cardiometabolic risk factors.⁴⁻⁸

- **CVD** accounts for up to half of all maternal deaths.
- **HDP-associated strokes** have more than doubled compared to non-HDP-associated strokes.
- **Preeclampsia** alone complicates up to 8% of pregnancies globally and an increased 25% of pregnancies in the United States between 1987 and 2004.^{19-A}

In addition to documented adverse maternal effects, HDP also affects offspring. Blood pressure falls during the first trimester of a normal pregnancy, reaching a maximum decline of 10–15 mmHg by mid-pregnancy, then returning to prepregnancy levels by term.^{20-A} Elevated systolic BP during pregnancy is associated with elevated risk of preterm delivery and infants who are small for gestational age and have low birth weight, even if BP is below diagnostic thresholds for hypertension.^{4-C}

Fetal exposure to preeclampsia nearly doubles the risk of hypertension and dyslipidemia and increases the risk of diabetes by more than 50% into early adulthood.^{6-C; 5-B}

HDP may be a useful clinical marker for increased risk of future hypertension for both mother and her children,^{5-C} and early detection and treatment can reduce the toll of maternal, fetal and neonatal mortality and morbidity.^{7-B}

Preventing and Managing HDP

Health care professionals have an expanding toolbox to help prevent HDP by mitigating risk factors before pregnancy and manage HDP after conception using combinations of lifestyle changes and appropriate pharmacotherapy.



Lifestyle changes made before and during pregnancy that reduce blood pressure can modify both maternal and fetal risks.



Dietary changes can reduce gestational weight gain and improve pregnancy outcomes, while exercise may reduce gestational hypertension and preeclampsia by 30% and 40%, respectively.

Canadian guidelines recommend physical activity for all women during pregnancy unless there are preexisting contraindications.^{4-F}

Cardiovascular health is not simply the absence of CVD but a broader concept of specific health factors that are associated with longer CVD-free survival, total longevity and higher quality of life.^{7-B} The American Heart Association's Life's Essential 8™ focuses on:^{7-Figure 1}

- Diet
- Physical activity
- Nicotine exposure
- Sleep health
- Body mass index
- Blood lipids
- Blood glucose
- Blood pressure



Each element represents one or more CVD risk factors that can be modified to improve cardiovascular health preconception, during pregnancy and postpartum. Health care professionals can use motivational interviewing approaches to help patients identify areas that can be improved and practical approaches they can envision themselves taking.^{7-C}

The American College of Obstetricians and Gynecologists (ACOG) classifies HDP into four categories:^{29-A; 8-D}

1 Chronic hypertension, either primary or secondary, predates the pregnancy or is diagnosed before 20 weeks. Chronic hypertension is associated with increased risk of preterm delivery, fetal growth restriction and perinatal death.

2 Gestational hypertension develops at or after 20 weeks without features of preeclampsia or eclampsia. Gestational hypertension increases the risk of preeclampsia.

3 Preeclampsia are multisystem disorders that can feature renal, hematologic, hepatic, neurologic and pulmonary complications. Adverse maternal outcomes can include eclampsia, stroke, multiorgan failure, hemorrhage and death. Fetal complications include growth restriction, preterm delivery, placental abruption and perinatal death.

4 Preeclampsia superimposed on chronic hypertension combines the increased risks and adverse outcomes of hypertension and preeclampsia/eclampsia.^{8-E}

Other publications add eclampsia and HELLP (hemolysis, elevated liver enzymes and low platelet count) syndrome to the list of HDPs.^{9-A}

Starting low-dose aspirin between 12 and 16 weeks reduces the risk for preeclampsia and HDP-associated adverse outcomes, including preterm birth, fetal growth restriction and stillbirth.

When given early in pregnancy, aspirin alters the thromboxane-prostacyclin equilibrium to favor prostacyclin and associated vasodilatory effects while inhibiting platelet aggregation, thought to improve implantation, placentation and vascular remodeling.

The U.S. Preventive Services Task Force and ACOG recommend low-dose aspirin (81 mg daily) for women with one or more major risk factors, including:

- A history of preeclampsia
- Multiple gestation
- Chronic hypertension
- Diabetes
- Kidney disease
- Renal disease
- Autoimmune disorders.^{8-C; 21-A, Table 1}

Risk reduction was estimated at 24% for preeclampsia, 20% for growth restriction and 14% for preterm birth. The benefits of aspirin appear to be dose-related, with suggestions of risk reduction up to 82% for early preeclampsia from 150 mg daily.^{8-C}

Likewise, ACOG recommends daily low-dose aspirin for women with two or more moderate risk factors, including 25 years of age or more, Black race or low socioeconomic status, family history of preeclampsia (mother or sister), history of low birth weight pregnancies, any

APO, more than 10 years between pregnancies, BMI greater than 30 kg per m² or nulliparity.^{21-A, Table 1}

Studies of nutritional supplements such as antioxidant vitamins C and E show no statistically significant reduction in the risk of preeclampsia. Calcium supplementation appears to reduce the risk of preeclampsia in women who are calcium deficient but not in the general U.S. population.^{8-D}

Experimental evidence and clinical studies suggest metformin may reduce the risk of gestational hypertension in women with gestational diabetes and may also prevent preeclampsia.^{4-F}

The U.S. Preventive Services Task Force recommends that all adults be screened for hypertension and concludes that screening has substantial net benefits.^{22-A, B} AHA and the American College of Cardiology (ACC) use a hypertension threshold of 130/80 mmHg for the general population. For people with chronic hypertension before or during pregnancy, the treatment threshold is 140/90 mmHg, as defined by AHA,²⁴ ACOG^{25-A} and most international societies.^{4-I} The updated recommendation by AHA, as of 2025, reflects increasing evidence that pregnant individuals may reduce their risk of serious complications by adhering to stricter blood pressure control.²⁵

ACOG lowered its previous treatment threshold following the Chronic Hypertension and Pregnancy (CHAP) trial,^{24-A} which confirmed better pregnancy outcomes when targeting a BP of

The following agents are generally accepted as safe during pregnancy:^{20-Table2}

Antihypertensive drug	Class/ action	Dose	Adverse effects
labetalol	Beta blocker	100 mg twice a day to 400 mg three times a day	Bradycardia, bronchospasm, headache
nifedipine controlled release	Calcium channel blocker	30 mg daily to 60 mg twice a day	Headache (first dose effect), flushing, tachycardia, peripheral edema
methyldopa	Central acting	250 mg twice a day to 750 mg three times a day	Depression, dry mouth, sedation, rarely hemolysis and hepatitis
hydralazine	Vasodilator	25–50 mg three times a day	Flushing, headache, lupus-like syndrome
prazosin	Alpha blocker	0.5 mg twice a day to 5 mg three times a day	Orthostatic hypotension

Labetalol or methyldopa are widely accepted as first-line hypertension agents during pregnancy.^{4-K} OTIS, the [Organization of Teratology Information Specialists](#) provides up-to-date information on the safety of medications during pregnancy and breastfeeding.^{4-L}

less than 140/90 mmHg compared to reserving hypertension treatment for more severe hypertension with systolic BP equal to or greater than 160 and/or diastolic BP equal to or greater than 105.^{25-A} Women in the active treatment arm of the trial took labetalol or nifedipine and had a lower risk of preeclampsia with severe features, medically indicated preterm birth before 35 weeks, placental abruption or fetal/neonatal death without apparent maternal or fetal/neonatal harm.^{23-Table 2} The trial hypothesis did not address treatment thresholds below 140/90 mmHg.^{23-B}



Women with known hypertension may already be on pharmacotherapy before or at the time of conception.

- Multiple classes of antihypertensives should be avoided during pregnancy due to potential adverse effects of mother, fetus or both.
- ACE inhibitors and angiotensin receptor blockers are contraindicated for teratogenic effects and should be stopped before conception or at the time of pregnancy diagnosis.^{20-B, C}
- Many diuretics, beta blockers (other than labetalol) and calcium channel blockers (other than nifedipine and diltiazem) may need to be avoided for potential maternal and fetal side effects. Alternative antihypertensive agents are frequently recommended.^{8-A, B}

Managing Maternal Health Postpartum

Attention to maternal health cannot lapse after delivery.

The definitive treatment for gestational hypertension, preeclampsia and eclampsia is delivery, and maternal sequelae are expected to resolve rapidly postpartum,^{11-B} but not all ill effects resolve as expected. More than half of maternal deaths occur postpartum.^{26-A}



First 6 months

Hypertension risk highest

↑ **13.39-fold**
with any HDP

↑ **43.95-fold**
with preeclampsia^{9-D}

First 2 years

Hypertension risk elevated by HDP

↑ **6.28-fold**
with any HDP^{9-B}

↑ **7.49-fold**
with preeclampsia^{9-C}

First 3 years

Any HDP doubles the risk for:

- Cardiovascular readmission
- Acute myocardial infarction
- Stroke
- Heart failure^{9-E}

Health care professionals and patients can take positive steps to reduce the risk of pregnancy-related postpartum adverse events. For example, breastfeeding is strongly associated with reduced maternal cardiovascular risk in addition to other recognized benefits for infants and mothers. Children who have been breastfed have reduced risk of death from infectious disease and reduced risk of respiratory infections.^{27-A}

Any HDP should trigger serial BP monitoring in the first months to years following delivery. Patients who have been diagnosed with pregnancy-associated high blood pressure should have their BP checked at least annually²⁵. Lifestyle changes, medical therapy and standard follow-up care are also appropriate.^{9-F}



Following delivery, women who have chronic hypertension can transition back to their usual antihypertensive treatment subject to compatibility with breastfeeding. Antihypertensive agents that are safe during pregnancy are also safe during breastfeeding. ACE inhibitors have low concentrations in breast milk and are often used during the breastfeeding period. Angiotensin receptor blockers are not recommended due to lack of safety information.^{20-E}



Gestational hypertension and preeclampsia contribute to increased risk for subsequent cardiovascular and cardiometabolic disease, including new onset hypertension, stroke, diabetes, venous thromboembolism and chronic kidney disease. Routine follow-up with annual reviews of BP, fasting lipids and blood glucose are recommended.^{20-D} The AHA's Life's Essential 8 may be a useful tool to help optimize cardiovascular health, including a healthy lifestyle and diet, normal BMI, tobacco cessation and regular exercise.^{7- Figure 1}



Lactation may help to reset maternal metabolic homeostasis after pregnancy.

Breastfeeding reduces the risk of:

- Breast cancer
- Ovarian cancer
- Type 2 diabetes^{27-A}
- Hypertension
- Subclinical atherosclerosis^{27-B}

Breastfeeding relatively reduces the risk of:

- CVD (11%)
- Coronary heart disease (14%)
- Stroke (12%)
- Fatal CVD (17%)^{27-C, D}

The Fourth Trimester

All these actions aimed at improving maternal health postpartum — breastfeeding recommendations, monitoring BP and other metabolic parameters, starting or adjusting hypertensive medications as needed and making appropriate lifestyle adjustments — assume clinical follow-up after delivery. Too many women never attend a comprehensive postpartum visit.

The postpartum visit can include multiple types of care, from acute issues arising after pregnancy, delivery or the immediate postpartum period to chronic conditions, infant care and feeding and engaging new mothers in preventative care for themselves and their infants. The reality is that postpartum attendance rates vary widely by geography, population, insurance coverage and data source, ranging from 24.9% to 95% with a mean of 74.2%.^{28-A, B}

A substantial proportion of women who have given birth do not attend at least one postpartum visit, which may contribute to maternal morbidity and mortality.^{28-C, 26-A}

Not only do more than half of all maternal deaths occur postpartum,

40% occur within the first six weeks

19% within one to six days

21% within seven to 42 days

12% within 43 to 365 days.^{26-A}

Postpartum care has traditionally focused on a single clinical visit six to eight weeks after delivery.^{26-C} To optimize the health of women and infants, ACOG has called for a redesign of postpartum care as an ongoing process throughout a fourth trimester of care, with services and support tailored to each woman's individual needs.^{2-A, D}

ACOG recommends all women should have contact with a maternal care provider within the first three weeks postpartum.

This initial exam should be followed by appropriate care as needed to support each woman's individual needs, concluding with a comprehensive postpartum visit no later than 12 weeks after birth.^{2-B, D}



The comprehensive visit should include a full assessment of physical and psychological well-being. Women with pregnancies complicated by HDP, preterm birth or gestational diabetes should be advised that these events are associated with a higher lifetime risk of maternal cardiometabolic disease.^{2-E, 26-B, C}

Women with chronic conditions, such as hypertensive disorders, obesity, diabetes, thyroid disorders, kidney disease, mood disorders or substance use disorders, should receive counseling regarding appropriate medical follow-up with their OB/GYN or primary care provider for continuing coordination of care.^{2-F, 9-G}

Improving coordination between OB/GYNs and primary care providers during the fourth trimester is only one element in the reinvention of maternal health care. **CVD is the leading cause of pregnancy-related mortality and is rising. As many as two-thirds of pregnancy-related CVD deaths may be preventable, identifying an important gap in care and CVD prevention.**

Pregnancy and the fourth trimester can be an opportunity for CVD risk evaluation, stratification and implementation of primary and secondary prevention strategies that are not widely recognized or used at this time.^{1-A, F}

Chronic hypertension, diabetes, dyslipidemia, obesity, tobacco use and sleep apnea are recognized risk factors for CVD. All can be amplified during pregnancy, and all can be modified before, during and after pregnancy with the appropriate prevention strategies.^{1-F} ACOG and the AHA have long advocated the use of multidisciplinary care by cardiologists and obstetricians.^{1-G}

Establishing postpartum follow-up opens the door for continuing interventions to better manage CVD risk factors, including pharmacotherapy, lifestyle management and education.^{1-D, F}



Postpartum visits need not always be in person. Telemedicine can help women maintain breastfeeding, manage postpartum depression and continue BP monitoring, among other benefits. The use of telemedicine may improve access to care, especially for women with time and/or resource constraints.^{1-H} Regardless of the type of visit, continued follow-up during the fourth trimester can improve early identification and intervention for CVD and CVD risk.^{1-D}

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