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Chronic Hypertension in Pregnancy

Chronic hypertension is present in 0.9–1.5% of pregnant women (1) and may result in significant maternal, fetal, and neonatal morbidity and mortality. The rate of maternal chronic hypertension increased by 67% from 2000 to 2009, with the largest increase (87%) among African American women. This increase is largely secondary to the obesity epidemic and increasing maternal age (1, 2). The trend is expected to continue.

The purpose of this document is to clarify the criteria used to define and diagnose chronic hypertension before or during pregnancy, to review the effects of chronic hypertension on pregnancy and vice versa, and to appraise the available evidence for management options. The purpose of these revised best practice recommendations is to provide a rational approach to chronic hypertension in pregnancy based on new research data and relevant pathophysiologic and pharmacologic considerations.

Background

Chronic Hypertension

Definition and Diagnosis of Chronic Hypertension

Chronic hypertension in pregnancy is defined as hypertension diagnosed or present before pregnancy or before 20 weeks of gestation. Hypertension that is diagnosed for the first time during pregnancy and that does not resolve in the typical postpartum period also is classified as chronic hypertension (3). Traditionally, the criteria for hypertension in pregnancy under this definition are a systolic blood pressure of 140 mm Hg or more, a diastolic blood pressure of 90 mm Hg or more, or both. In general, it is recommended that a diagnosis of hypertension requires at least two determinations at least 4 hours apart, although on occasion, especially when faced with severe hypertension, the diagnosis can be confirmed within a shorter interval (even minutes) to facilitate timely therapy.

Recent recommendations from the American College of Cardiology (ACC) and the American Heart Association (AHA) have changed the criteria for diagnosing hypertension in adults (4). These recommendations include classifying blood pressure into four categories: 1) normal (systolic blood pressure less than 120 mm Hg and diastolic blood pressure less than 80 mm Hg); 2) elevated (systolic blood pressure of 120–129 mm Hg and diastolic blood pressure less than 80 mm Hg); 3) stage 1 hypertension (systolic blood pressure of 130–139 mm Hg or diastolic blood pressure of 80–89 mm Hg); and 4) stage 2 hypertension (systolic blood pressure of 140 mm Hg or more or diastolic blood pressure of 90 mm Hg or more). These changes were made to assist in clinical and public health decision making and reflect data to suggest modifiable long-term cardiovascular risk even in the elevated and stage 1 hypertension ranges (5). Importantly, the recommendations now suggest beginning treatment in nonpregnant adults with risk factors for current or future cardiovascular disease in patients with stage 1 hypertension (systolic blood pressure of 130–139 mm Hg or diastolic blood pressure of 80–89 mm Hg) (6). Thus, obstetric care providers may see an increase in patients classified as hypertensive based on these ACC/AHA definitions. For these patients, it is reasonable to continue to manage the

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patient in pregnancy as chronically hypertensive as specified in this guideline. The effect of the ACC/AHA changes on the diagnosis of hypertension in women of reproductive age, on pregnancy outcomes, and on the use of health care resources for pregnant women is unknown. For instance, it is not clear what should be done with a patient without a prior diagnosis of chronic hypertension who has blood pressures in the stage 1 hypertension range (systolic blood pressure of 130–139 mm Hg or diastolic blood pressure of 80–89 mm Hg) before 20 weeks of gestation. Based on the recommendations in this Practice Bulletin this range would not require initiation of antihypertensive medication. However, a conservative approach of a higher degree of observation may be warranted. A secondary analysis of a randomized trial of low-dose aspirin in the prevention of preeclampsia looked at outcomes in the placebo patients who at the time were diagnosed as normotensive, but according to this new criterion would have stage 1 chronic hypertension. These patients had a higher risk of preeclampsia, gestational diabetes, and indicated preterm birth. However, aspirin, compared with placebo, did not appear to lower the risk of preeclampsia among patients in the stage 1 hypertension group (7). The uncertainty of the new approach to hypertension recommended by the ACA and AHA as applied to the care of pregnant women should be an active area of investigation.

Using the ACC/AHA definition to determine chronic hypertension for pregnant women presents a lower threshold for diagnosis than traditionally used. Some borderline and possibly inconsequential cases of blood pressure elevation, particularly in patients diagnosed with chronic hypertension during pregnancy before 20 weeks of gestation, may mislabel some women as abnormal. In the absence of a preexisting diagnosis, detection of hypertension at any time before 20 weeks of gestation is assumed to indicate chronic hypertension antedating the pregnancy or, much more rarely, is associated with fetoplacental abnormalities such as hydatidiform mole. On the other hand, hypertension detected after 20 weeks of gestation typically is indicative of preeclampsia or gestational hypertension if the woman was normotensive before 20 weeks of gestation. The approach of using more conservative thresholds, like those of the ACC/AHA guidelines, with higher sensitivity admittedly errs on the side of caution. The assumption that the 20-week mark always can discriminate chronic hypertension from pregnancy-related hypertension is not well substantiated by scientific data. For example, a retrospective cohort study found that 46 of 119 (39%) women with a history of obstetric complications and normal blood pressure before pregnancy (as indicated on blood pressure recordings before and during pregnancy) developed nonproteinuric gestational hypertension before 20 weeks of gestation that resolved in the postpartum period (8). The authors of the study concluded that hypertension arising in the first 20 weeks of pregnancy may not necessarily indicate chronic hypertension. Indeed, it defies logic to think that one point in time (eg, 20 weeks of gestation) may always and reliably differentiate disease preceding fertilization from a pregnancy-related condition. The 20-week convention should not be used dogmatically, but rather for orientation while maintaining clinical judgment.

To establish a diagnosis of chronic hypertension, it is ideal to have knowledge of prepregnancy blood pressure values. However, for many women, prepregnancy blood pressures are not known. Moreover, the prevalence of hypertension is underestimated when based on self-reporting as opposed to documented measurements or physician diagnosis (9). In addition, previously undiagnosed chronic hypertension may be masked because of the pregnancy-related hemodynamic changes in the first and second trimesters. The normal physiologic decrease in systemic vascular resistance leads to a decrease in blood pressure, with its nadir at 16–18 weeks of gestation, followed by return to prepregnancy levels by the third trimester. The 30% decrease in systemic vascular resistance that normally occurs early in pregnancy typically generates a decrease in blood pressure of 10% as early as 7 weeks of gestation (and even more by midpregnancy), which results in a potential blood pressure normalization in the absence of any treatment. The decrease in diastolic blood pressure (by as much as 20 mm Hg) is more marked than the decrease in systolic blood pressure. Because blood pressure usually returns to prepregnancy levels in the third trimester, diagnostic confusion is possible, and chronic hypertension may be mislabeled as gestational hypertension or preeclampsia in this setting. Moreover, approximately 11% of women with chronic hypertension have proteinuria (more than 300 mg/day) at baseline (10) because of hypertension-related nephrosclerosis or, less frequently, undiagnosed chronic kidney disease. Therefore, the distinction between chronic hypertension and either gestational hypertension or preeclampsia sometimes can be made only in retrospect, especially among women who initiate prenatal care beyond 20 weeks gestation. It has been suggested that hypertension persisting longer than 12 weeks after delivery may be retrospectively reclassified as chronic. However, the time required for resolution of pregnancy-related hypertension has not been clearly established. For example, a prospective cohort study of 205 preeclamptic women in the Netherlands found that 39% still had hypertension 12 weeks after delivery, and in 50% of these women it took up to 2 years for blood pressure to normalize (11).

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Types of Chronic Hypertension

It is estimated that chronic hypertension antedating pregnancy is essential (ie, of unknown cause) in more than 86–89% of cases of hypertension and secondary (ie, related to underlying renal, endocrine, or vascular conditions) in 11–14% of cases of hypertension (1). A thorough history and physical examination are crucial to detect the rarer secondary forms of hypertension. To help in defining either the hypertensive mechanism or the extent of end-organ damage, directed tests, such as serum electrolytes (specifically potassium), blood urea nitrogen, serum creatinine, a complete blood count, liver function tests, a urinalysis, a toxicology screen, or an electrocardiogram, should be obtained as appropriate.

Hypertension in pregnancy also is classified as severe if at or above cutoff points of 160 mm Hg for systolic blood pressure or 110 mm Hg for diastolic blood pressure, or both (Table 1). Prior publications have suggested a diastolic cutoff of 105 mm Hg based on safety concerns but for consistency and based on the best available evidence the diastolic cutoff of 110 mm Hg is recommended. At least two blood pressure readings at or above the cutoffs of 160 mm Hg (systolic) or 110 mm Hg (diastolic), measured 4 hours apart, are necessary to consider the hypertension diagnosis as severe. However, antihypertensive treatment should not be delayed just for the sake of confirming the nomenclature for the type of hypertension, and the initiation and effectiveness of antihypertensive therapy should be considered when classifying the degree of hypertension. In these cases, the diagnosis may be confirmed within a shorter interval (even minutes) to facilitate timely therapy.

Measuring Blood Pressure

The blood pressure levels that meet the definition criteria should be documented on repeat readings only after the patient has rested (preferably for 10 minutes or more) and is seated with legs uncrossed and back supported. No caffeine or tobacco should have been used for at least 30 minutes before measurement, because these can temporarily elevate blood pressure. An appropriate-sized cuff (eg, one with a length 1.5 times the upper arm circumference or a cuff with a bladder that encircles at least 80% of the arm and a width of at least 40% of arm circumference) positioned at the level of the heart to ensure accurate readings should be used. Appropriate cuff sizes for specific arm circumferences are the following:

- For an arm circumference of 22−26 cm, the cuff should be small adult size: 12 × 22 cm.
- For an arm circumference of 27−34 cm, the cuff should be adult size: 16 × 30 cm.
- For an arm circumference of 35−44 cm, the cuff should be large adult size: 16 × 36 cm.
- For an arm circumference of 45−52 cm, the cuff should be adult thigh size: 16 × 42 cm.

Blood pressure cuffs that are too small will result in an overestimation of actual blood pressure, and an unsupported back, crossed legs, or unsupported arm can cause small overestimations as well. If blood pressure must be taken in a recumbent position, the patient should be placed in a left lateral decubitus position and the cuff should be at the level of the right atrium (12). These details represent the standard for blood pressure assessment. Abnormal values not taken in this manner are sometimes overestimated and, thus, repeat assessment using the best methods is advisable.

Chronic Hypertension With Superimposed Preeclampsia

Preeclampsia is considered superimposed when it complicates preexisting chronic hypertension. Up to 20–50% of women with chronic hypertension may develop

Table 1. American College of Obstetricians and Gynecologists Definitions of Hypertensive Disorders

Disorder	Definition
Hypertension in pregnancy	Systolic blood pressure ≥140 mm Hg or diastolic BP ≥90 mm Hg, or both, measured on two occasions at least 4 hours apart
Severe-range hypertension	Systolic blood pressure ≥160 mm Hg or diastolic BP ≥110 mm Hg, or both, measured on two occasions at least 4 hours apart
Chronic hypertension	Hypertension diagnosed or present before pregnancy or before 20 weeks of gestation; or hypertension that is diagnosed for the first time during pregnancy and that does not resolve it the postpartum period
Chronic hypertension with superimposed preeclampsia	Preeclampsia in a woman with a history of hypertension before pregnancy or before 20 weeks of gestation

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superimposed preeclampsia, an incidence five times or more than that of pregnant women without hypertension (10, 13, 14). In women with end-organ disease or secondary hypertension, the rate of superimposed preeclampsia has been reported to be as high as 75% (15–18). In patients with chronic hypertension, preeclampsia tends to have an earlier onset and to be more severe, and the prognosis for the woman and her fetus is worse than in either condition alone (10, 15). There are currently no useful tools for predicting superimposed preeclampsia, but the risk of superimposed preeclampsia is higher in women who are African American, obese, smoke, have had hypertension for 4 years or more, have a diastolic blood pressure higher than 100 mm Hg at baseline, and have a history of preeclampsia (10, 19).

Superimposed preeclampsia is not always easy to diagnose and is often a diagnosis of exclusion. A sudden increase in baseline hypertension or a sudden increase in proteinuria (above the threshold for normal or a clear change from baseline) should prompt assessment for a possible diagnosis of superimposed preeclampsia and consideration for subspecialty (eg, maternal–fetal medicine) referral. However, it is often difficult to distinguish between worsening of chronic hypertension and chronic hypertension with superimposed preeclampsia. New onset of thrombocytopenia may be helpful because—in contrast to blood pressure elevation and proteinuria—platelet activation, aggregation, and consumption are not present with gestational or chronic hypertension. As with thrombocytopenia, a sudden increase in liver enzymes to abnormal levels or the sudden development of symptoms suggestive of preeclampsia should point to the diagnosis of superimposed preeclampsia. Elevated uric acid levels may be helpful in cases of diagnostic uncertainty as well (20). On the other hand, a thorough evaluation may detect specific circumstances indicative of chronic hypertension aggravation (eg, cocaine or methamphetamine use or nonadherence with treatment) rather than superimposed preeclampsia.

White Coat Hypertension

White coat hypertension, defined as elevated blood pressure primarily in the presence of health care providers, may account for up to 15% of individuals with office hypertension, although the exact prevalence in pregnancy is not known. It must be emphasized that even white coat hypertension should not be considered entirely benign, because 8% and 40% of such cases will progress to preeclampsia and gestational hypertension, respectively, later in pregnancy (21). For women with suspected white coat hypertension, the use of ambulatory blood pressure monitoring may be beneficial to confirm the diagnosis and to assist with decisions for initiation of antihypertensive therapy.

Monitoring Blood Pressure

Good clinical practice dictates increased monitoring for women with elevated blood pressure especially in the second half of pregnancy. Although out-of-office and self-monitoring of blood pressure has not been evaluated in pregnant women with hypertension, the literature examining nonpregnant women with hypertension suggest that it is safe to use in both populations. Presumed advantages of out-of-office and self-monitoring include patient convenience, increased therapeutic adherence, confirmation of white coat hypertension, and assistance with adjusting medications when there is uncertainty. However, many home blood pressure devices are sold without formal validation of accuracy (22). For example, several reports have shown that 25–70% of tested blood pressure devices were not accurate to within 5 mm Hg, a degree of blood pressure difference considered to be clinically important (23, 24). Procedures for the use of home blood pressure monitoring are available and emphasize patient training, use of appropriately validated devices, and clear instructions (4). It may be useful to have a patient bring in her home monitor to compare against measurements done in the office. Home monitoring may reduce the frequency of office visits in cases with marginal blood pressure control.

Effects of Chronic Hypertension on Pregnancy

Maternal Risks

A population study of nearly 30,000 pregnant women with chronic hypertension demonstrated that maternal mortality and the risk of cerebrovascular accidents, pulmonary edema, or renal failure were about fivefold to sixfold higher than in normotensive pregnant women (16). However, the absolute risk of mortality and major maternal morbidity is low in developed countries, and most women with mild essential hypertension will have uncomplicated gestations (25). Only in cases of severe, uncontrolled hypertension does the risk of maternal complications markedly increase (13).

Accelerated hypertension with resultant end-organ damage (heart, brain, kidneys) is extremely uncommon. The diagnosis of superimposed preeclampsia is much more likely when a sudden exacerbation of hypertension, typically with end-organ dysfunction, develops after 20 weeks of gestation requiring an acute escalation of antihypertensive therapies.

Chronic hypertension is associated with an increased risk of gestational diabetes, possibly as a consequence of common risk factors (such as obesity) and shared pathogenic context (such as increased insulin resistance, chronic inflammation, and endothelial dysfunction) (17,

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26, 27). In a prospective cohort study, the incidence of gestational diabetes was 8.1% in women with chronic hypertension compared with 2.3% in those without chronic hypertension (adjusted odds ratio [AOR] 1.6; 95% CI, 1.27–2.05) (28).

In addition, compared with normotensive women, those with uncomplicated chronic hypertension have a 1.8-fold increased risk of planned cesarean delivery before labor and twice the risk of postpartum hemorrhage (18, 28).

Fetal Risks

A large body of evidence indicates that chronic hypertension in pregnancy is associated with poorer perinatal outcomes. A systematic review of 55 studies demonstrated that the pooled incidence for low birth weight was approximately 17%, and the pooled incidence for preterm delivery was 28% (29). The increase in preterm delivery appears to be ascribed to indicated preterm deliveries, without an increase in spontaneous preterm deliveries, according to a prospective cohort study that included 1,417 pregnant women with chronic hypertension (28). In the same cohort, the incidence of fetal growth restriction was twice as high in pregnant women with chronic hypertension compared with those without chronic hypertension (30).

The perinatal mortality rate reported with maternal chronic hypertension is 2–4 times higher than that of the general population (31), and the increased risk of stillbirth or neonatal death appears to be independent of other possible contributors such as superimposed preeclampsia, fetal growth restriction, or gestational diabetes (32). In a Swedish population study, the risk of placental abruption was reported as 1.1% in women with chronic hypertension (AOR 2.3; 95% CI, 1.6–3.4) compared with controls without hypertension (17). The gestational age at stillbirth in pregnancies complicated by chronic hypertension has been noted to be lower than in pregnancies not complicated by chronic hypertension (a median of 28 weeks of gestation versus 35 weeks of gestation, respectively) (28).

The incidence of these adverse perinatal effects appears to be related to the duration and severity of chronic hypertension as possible surrogates for end-organ damage. Consequently, the rate of fetal adverse effects may be correlated with factors such as proteinuria at baseline or maternal cardiac dysfunction (10, 33). Women with severe hypertension, end-organ disease, or secondary hypertension represent the highest risk category, in which the risk of fetal growth restriction increases to 25–40%, preterm delivery to 67%, placental abruption to 8–20%, and perinatal death to 11% (10, 13, 34–36).

In the setting of superimposed preeclampsia, higher rates of adverse maternal and fetal outcomes can be expected. The risk of preterm delivery and placental abruption are further increased (16), and the risk of fetal growth restriction has been reported to be as high as 50% (15). The relative risk of perinatal mortality is 3.6 in women with superimposed preeclampsia compared with those with uncomplicated chronic hypertension (34).

Limited evidence is emerging that patients with chronic hypertension may be at higher risk of fetal congenital malformations. In a systematic review of 16 observational studies, a higher relative risk of congenital heart disease of 1.4 (95% CI, 1.2−1.7) and 2.0 (95% CI, 1.5−2.7) was found among the offspring of women with both untreated and treated chronic hypertension, respectively (37). The specific mechanism involved is unclear, but it does not appear to be simply because of a teratogenic effect from medication. The authors of a case–control study based on registry data confirmed the above findings supporting the hypothesis that physiological changes early in pregnancy among women with

Box 1. Risks of Chronic Hypertension in Pregnancy

Maternal
- Death
- Stroke
- Pulmonary edema
- Renal insufficiency and failure
- Myocardial infarction
- Preeclampsia
- Placental abruption
- Cesarean delivery
- Postpartum hemorrhage
- Gestational diabetes

Fetal and Neonatal
- Stillbirth or perinatal death
- Growth restriction
- Preterm birth
- Congenital anomalies (eg, heart defects, hypospadias, esophageal atresia)

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chronic hypertension may play a role in the etiology of cardiac septal defects, hypospadias, and esophageal atresia (38). The main risks of chronic hypertension in pregnancy are listed in Box 1.

Clinical Considerations and Recommendations

▶ ***What considerations are important for prepregnancy counseling in patients with chronic hypertension?***

A woman with chronic hypertension should be evaluated prepregnancy to identify possible end-organ involvement, to consider evaluation for secondary hypertension, and for the optimization of maternal comorbidities (eg, obesity, diabetes) before pregnancy. Women with modifiable risk factors, such as obesity and poor glycemic control, may benefit from counseling on weight loss, diet, and lifestyle modifications (39–41). Women with chronic hypertension should have their blood pressure optimized before pregnancy and should avoid excessive sodium and caffeine intake as well as smoking. Prepregnancy counseling should include an explanation of the risks associated with chronic hypertension in pregnancy (Box 1).

The medication review should place special emphasis on agents to be avoided, in particular angiotensin-converting enzyme inhibitors and angiotensin receptor blockers. These drugs, active against renin-dependent vasoconstriction, represent the first line of treatment in nonpregnant patients with decompensated heart failure, pulmonary edema, coronary ischemia, proteinuric renal disease, and diabetes (for renal protection). However, they are fetopathic, and exposure in the first trimester should be avoided if possible because of the risk of malformations (eg, renal dysgenesis, calvarial hypoplasia) and fetal growth restriction (42). However, there may be cases of primary or secondary hypertension in pregnancy refractory to other antihypertensive medications and, thus, controlled only by angiotensin-converting enzyme inhibitors. When angiotensin-converting enzyme inhibitor therapy is being considered in pregnancy, a maternal–fetal medicine subspecialty referral is advised.

▶ ***Which clinical tests are useful in the initial evaluation of a pregnant woman with chronic hypertension?***

Specific testing before or at the time of presenting for pregnancy care is used to detect possible end-organ involvement. This baseline evaluation (Box 2) may be influenced by the physiologic changes of pregnancy seen as early as the early first trimester and underscores the importance of prepregnancy testing (43). However, if testing was not done before pregnancy, it should be performed at entry to prenatal care. Box 2 summarizes the tests for baseline evaluation for chronic hypertension in pregnancy. Although uric acid has historically been among these tests and may be helpful in cases of diagnostic uncertainty, it is no longer a recommended routine baseline test.

Box 2. Tests for Baseline Evaluation for Chronic Hypertension in Pregnancy

- Serum aspartate aminotransferase and alanine aminotransferase
- Serum creatinine
- Serum electrolytes (specifically potassium)
- Blood urea nitrogen
- Complete blood count
- Spot urine protein/creatinine ratio or 24-hour urine for total protein and creatinine (to calculate creatinine clearance) as appropriate
- Electrocardiogram or echocardiogram as appropriate

Because the kidneys are usually the first end-organ to be affected by chronic hypertension, baseline renal function assessment commonly includes serum creatinine, a spot urine protein-to-creatinine ratio, and, if needed, a 24-hour urinary collection for total protein and creatinine clearance. The spot urine protein-to-creatinine ratio can be used effectively as a screening test to rule out proteinuria and is a sensible first step. For patients with borderline or abnormal urine protein-to-creatinine ratios or serum creatinine values, a 24-hour urine collection is recommended. A spot urine protein-to-creatinine ratio of less than 0.15 safely indicates a level of proteinuria less than 300 mg for a 24-hour sample and, in the absence of an abnormality in serum creatinine, likely does not warrant further evaluation with a 24-hour urine collection, unless there is a need to assess creatinine clearance. One systematic review from 2008 found that protein-to-creatinine ratio cutoffs of 0.13–0.15 had sensitivities ranging from 90% to 99% to rule out significant proteinuria of 300 mg or more (44). A more recent meta-analysis demonstrated sensitivities of 88% (95% CI, 86−93%) for a protein-to-creatinine ratio cutoff of 0.13 and 88% (95% CI, 85−92%) for a cutoff of 0.15 to detect 300 mg or more of protein in a 24-hour urine sample (45).

There is a relationship between the degree of renal impairment and physiologic adaptation to pregnancy, indicating why assessment of creatinine clearance may be helpful in some cases. With mild renal impairment (serum creatinine between 0.9 mg/dL and 1.4 mg/dL), there is normal intravascular volume expansion and an incremental increase in creatinine clearance although

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somewhat less than in normal pregnancy (46). With moderate impairment (serum creatinine from 1.4 mg/dL to 2.4–2.8 mg/dL), only 50% of women will have the expected increase in creatinine clearance despite a normal blood volume expansion. With severe impairment (serum creatinine more than 2.4–2.8 mg/dL), there is a markedly attenuated increase in blood volume and no increase in creatinine clearance (47). Knowledge of impaired creatinine clearance can assist in managing complications related to changes in normal physiology. Further, patients with impaired creatinine clearance and elevated serum creatinine levels are at risk of worsening of their renal disease during pregnancy and, thus, serial assessment is indicated.

Assessment of kidney function by serum and urinary components can assist counseling and management through pregnancy. Renal dysfunction, when detected, indicates an increased risk of adverse pregnancy outcomes. Baseline proteinuria in women with chronic hypertension significantly increases the risk of superimposed preeclampsia, preterm delivery, fetal growth restriction, and neonatal admission to intensive care (10, 33). Furthermore, a normal baseline renal function assessment, including the absence of proteinuria, will permit subsequent comparisons and will assist in establishing the diagnosis of superimposed preeclampsia if suspected later in pregnancy.

Women who have had poorly controlled hypertension for more than 4 years or those suspected of having long-standing hypertension based on age (older than 30 years) are more likely to have cardiac hypertrophic changes, cardiomegaly, and ischemic heart disease and are candidates for additional diagnostic testing. Detecting these problems is important for risk mitigation in pregnancy, childbirth, and the postpartum period. Good clinical practice suggests assessing cardiac status in women with long-standing hypertension, specifically left ventricular function, with electrocardiography as an acceptable first-line test. Patients with extenuating risk factors or abnormal electrocardiography should be evaluated with echocardiography.

▶ ***When is evaluation for secondary hypertension appropriate, and what is that evaluation?***

In general, patients with primary (essential) hypertension demonstrate gradual increases in blood pressure over time (months to years), lifestyle factors favoring higher blood pressures (obesity, poor diet, lack of physical exercise, smoking, or excessive alcohol use), or a family history (Box 3). Secondary hypertension describes the 10% of adult patients with hypertension due to a specific and remediable cause (4). Hypertension resistant to treatment or diagnosed at a young age (younger than 30 years) warrants consideration and diagnostic workup for secondary (and potentially curable) hypertension unless such evaluations were previously performed (4). A strong family history of kidney disease also may indicate a renal etiology for secondary hypertension. Other signs and symptoms, listed in Box 3, also suggest secondary hypertension (4). When evaluation for secondary hypertension is considered, the work-up should be performed in consultation with a maternal–fetal medicine subspecialist and those in the appropriate medical subspecialty (such as cardiology, nephrology, endocrinology, or pulmonology). Given the complexity and variability in the recommended strategies for diagnosing secondary hypertension, referral to a physician with expertise in treating hypertension is suggested for the work-up of women with features suggestive of secondary hypertension.

▶ ***What treatments should be used for pregnant women with chronic hypertension, and what are the goals of treatment?***

Control of Chronic Hypertension

Controversy exists over the potential benefits and harms of treatment with antihypertensive drugs during pregnancy. Because of the underlying physiology of pregnancy as well as fetal considerations, the antihypertensive protocols used for nonpregnant individuals cannot be extrapolated to pregnant women. The precise blood pressure level at which antihypertensive therapy is indicated during pregnancy in women with chronic hypertension continues to be debated. In nonpregnant adults, therapy is universally recommended at a blood pressure of 140/90 mm Hg or more based on therapeutic benefit (eg, decreased incidence of stroke, myocardial infarction, and heart failure) demonstrated in large clinical trials with long-term blood pressure control (4). Patients with blood pressures of 130–139/80–89 mm Hg and other cardiovascular morbidities and risk factors may benefit from treatment as well (4). Few clinical trials on this topic have been conducted in pregnancy and the evidence is limited. Most of the available studies included women with chronic hypertension and women with pregnancy-related hypertension, making the interpretation of the results less specific (48). Moreover, their design is variable, with antihypertensive drugs being compared to one another, to a placebo, or to no therapy. Demonstrated maternal and perinatal benefits outweighing the theoretical adverse maternal, fetal, and neonatal risks are needed to justify the use of antihypertensive therapy during pregnancy at the same blood pressure threshold as in nonpregnant adults. The benefit of blood pressure treatment for the pregnant woman to achieve

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Box 3. Historical Features Favoring Hypertension Cause

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

Abbreviations: BP, blood pressure; NSAIDs, nonsteroidal antiinflammatory drugs.

Reprinted from: Whelton PK, Carey RM, Aronow WS, Casey DE Jr, Collins KJ, Dennison Himmelfarb C, 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 [published erratum appears in J Am Coll Cardiol 2018;71:2275-9]. J Am Coll Cardiol 2018;71:e127–248.

targets that are recommended for nonpregnant individuals may not be apparent during the short time frame of puerperal treatment (13).

A 2014 Cochrane systematic review of 49 trials (4,723 women) concluded that treatment of mild-to-moderate hypertension reduced the risk of developing severe hypertension but had no effect on the incidence of preeclampsia, preterm birth, fetal death, fetal growth restriction, or any other measured outcome (49). Notably, the theoretical concern of fetal harm (primarily growth restriction) resulting from possible impairment of the uteroplacental blood flow could not be verified in this systematic review. However, in the absence of any improvement in perinatal outcomes, the authors concluded that it remains unclear whether antihypertensive drug therapy for mild-to-moderate hypertension during pregnancy is worthwhile. The 2015 CHIPS trial, an international randomized controlled trial comparing less tight control (ie, target diastolic blood pressure below 100 mm Hg) to tight control (ie, target diastolic blood pressure below 85 mm Hg) in 987 pregnant women, 75% of whom had chronic hypertension, reached similar conclusions (48). Tight control of hypertension conferred no benefit to the fetus and had only marginal effects for the woman, namely reduced frequency of progression to severe hypertension. A large multicenter, randomized study in the United States to evaluate whether a blood pressure treatment strategy during pregnancy aiming to achieve targets that are recommended for nonpregnant reproductive-aged adults (less than 140/90 mm Hg) is safe and effective when compared with no treatment (unless hypertension is severe), is currently ongoing. In the absence of clear evidence supporting the use of antihypertensive therapy for lower blood pressures, initiation of antihypertensive therapy is recommended for persistent chronic hypertension when systolic pressure is 160 mm Hg or more, diastolic pressure is 110 mm Hg or more, or both. In the setting of comorbidities or underlying impaired renal function, treating at lower blood pressure thresholds may be appropriate.

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Severe elevation in blood pressure may be associated with acute maternal cerebrovascular and cardiac events; however, the precise blood pressure level at which the risk of such adverse events increases is not known and is likely dependent on comorbidities and other factors such as the rate of blood pressure increase. A Cochrane systematic review, including 35 trials (3,573 women), evaluating treatment for severe hypertension during pregnancy provided only limited evidence for women with chronic hypertension as they were generally excluded from the individual studies and, when they were included, no subgroup analyses were reported (50). Future placebo-controlled trials addressing treatment of severe hypertension in pregnancy are unlikely and may not be recommended given ethical considerations. Therefore, recommendations for treating women with chronic hypertension in the severe range (ie, systolic blood pressure of 160 mm Hg or more or diastolic blood pressure of 110 mm Hg or more) are based on indirect evidence from treatment of preeclampsia and extrapolation of national guidelines for nonpregnant adults (51, 52). In women with evidence of end-organ damage (such as left ventricular hypertrophy or renal insufficiency) or severe thrombocytopenia, antihypertensive treatment is recommended at even lower thresholds (eg, systolic blood pressure of 150 mm Hg and diastolic blood pressure of 100 mm Hg) to reduce the risks of further end-organ damage or hemorrhagic stroke (53).

There are minimal data to guide decisions regarding continuing or discontinuing therapy for women with chronic hypertension with blood pressures of less than 160 mm Hg systolic or less than 110 mm Hg diastolic, or well-controlled blood pressure, who were receiving antihypertensive medication before pregnancy. Because treatment was initiated before the pregnancy with the understanding that it offers long-term health benefits, some question whether the lack of short-term benefits noted with treatment during pregnancy are a reasonable argument for discontinuation of treatment. Two case–control studies of women in whom medication was reduced or stopped early in pregnancy detected no increase in preeclampsia, placental abruption, or perinatal death (10, 54). However, a prospective observational study, including 222 women with mild chronic hypertension, found that although discontinuation of treatment had no effect on the incidence of preeclampsia, fetal growth restriction, or perinatal death, there was a significantly higher occurrence of severe hypertension, placental abruption, preterm delivery, and neonatal intensive care unit admissions in the group that discontinued treatment (55). With such mixed and limited information, the decision to continue or discontinue must be individualized and guided by an informed discussion with the pregnant woman. There is also the practical aspect requiring close monitoring of blood pressure if medication is withdrawn. Reductions or discontinuations of therapy in these cases should be countered with appropriate surveillance to ensure the patient does not develop blood pressures that do require treatment. Drugs contraindicated in pregnancy should be promptly replaced with other antihypertensive agents and subsequent therapy adjustments should be made based on clinical findings.

There also are limited data to address the ideal target blood pressure for a pregnant woman receiving therapy for chronic hypertension in order to improve maternal and perinatal outcomes. Overly aggressive blood pressure lowering is discouraged because of concerns that uteroplacental blood flow may be compromised. It is recommended to maintain blood pressure levels for pregnant women with chronic hypertension treated with antihypertensive medications at or above 120 mm Hg but below 160 mm Hg systolic and at or above 80 mm Hg but below 110 mm Hg diastolic. For women with comorbid conditions, such as diabetes or renal disease, blood pressure goals should be lower, as noted previously, and optimal management can be achieved in consultation with other subspecialties.

When antihypertensive therapy is used during pregnancy, an important consideration is the context of therapy, which is either 1) chronic treatment to lower blood pressure to maintenance levels, sometimes slowly during 24–48 hours often in the outpatient setting, or 2) acute lowering of critical hypertension in the hospital setting.

For chronic maintenance treatment, the oral agents listed in Table 2 can be considered alone or in combination. The table lists the most commonly used medications in pregnancy and is not meant to be comprehensive. For the long-term treatment of pregnant women who require pharmacologic therapy, labetalol or nifedipine are reasonable options and are recommended above all other antihypertensive drugs. The use of angiotensin-converting-enzyme inhibitors, angiotensin receptor blockers, renin inhibitors, and mineralocorticoid receptor antagonists is generally not recommended. Methyldopa is generally less favored because it appears to be less effective compared with other agents (49) and because of adverse effects. Diuretics, like hydrochlorothiazide, generally are considered second-line agents for the treatment of hypertension in pregnancy (56). Theoretical concern has been raised regarding the potential for diuretics to cause intravascular volume depletion and thereby lead to fetal growth restriction or oligohydramnios; however, this concern is not supported based on data from a meta-analysis of nine randomized trials as well as a systematic review of diuretics for the prevention of preeclampsia (57, 58). Other agents such as clonidine and prazosin have been used but should only be considered with

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Table 2. Common Oral Antihypertensive Agents in Pregnancy

Drug	Dosage	Comments
Labetalol	200–2,400 mg/d orally in two to three divided doses. Commonly initiated at 100–200 mg twice daily	Potential bronchoconstrictive effects. Avoid in women with asthma, preexisting myocardial disease, decompensated cardiac function, and heart block and bradycardia.
Nifedipine	30–120 mg/d orally of an extended-release preparation. Commonly initiated at 30–60 mg once daily (extended-release)	Do not use sublingual form. Immediate-release formulation should generally be reserved for control of severe, acutely elevated blood pressures in hospitalized patients. Should be avoided in tachycardia.
Methyldopa	500–3,000 mg/d orally in two to four divided doses. Commonly initiated at 250 mg twice or three times daily	Safety data up to 7 years of age in offspring. May not be as effective as other medications, especially in control of severe hypertension. Use limited by side effect profile (sedation, depression, dizziness).
Hydrochlorothiazide	12.5–50 mg daily	Second-line or third-line agent

appropriate input from maternal–fetal medicine and cardiology subspecialists. Atenolol, a β-blocker, is not recommended in pregnancy because of the risk of growth restriction and low birth weight (59).

Control of Acute-Onset Severe-Range Hypertension

Because most existing randomized controlled trials of acute treatment of severe-range blood pressure during pregnancy excluded women with chronic hypertension or previous antihypertensive therapy, the available evidence is inadequate to demonstrate a therapeutic advantage of any medication over another. Therefore, drug selection should be individualized depending on potential adverse effects and contraindications. Because of the unique physiologic and fetal considerations of pregnancy, the antihypertensive protocols used for nonpregnant individuals cannot be extrapolated simply to pregnant women.

Table 3. Antihypertensive Agents Used for Urgent Blood Pressure Control in Pregnancy

Drug	Dosage	Comments	Onset of Action
Labetalol	10–20 mg IV, then 20–80 mg every 10–30 minutes to a maximum cumulative dosage of 300 mg; or constant infusion 1–2 mg/min IV	Tachycardia is less common and fewer adverse effects than other agents. Avoid in women with asthma, preexisting myocardial disease, decompensated cardiac function, and heart block and bradycardia.	1–2 minutes
Hydralazine	5 mg IV or IM, then 5–10 mg IV every 20–40 minutes to a maximum cumulative dosage of 20 mg; or constant infusion of 0.5–10 mg/hr	Higher or frequent dosage associated with maternal hypotension, headaches, and abnormal fetal heart rate tracings; may be more common than other agents.	10–20 minutes
Nifedipine (immediate release)	10–20 mg orally, repeat in 20 minutes if needed; then 10–20 mg every 2–6 hours; maximum daily dose is 180 mg	May observe reflex tachycardia and headaches.	5–10 minutes

Abbreviations: IM, intramuscularly; IV, intravenously.

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Although blood pressure levels in the nonpregnant population are not considered the only criteria for hypertensive crisis, in obstetric practice blood pressure levels are traditionally relied upon for diagnostic and management decisions. More conservative thresholds have been established because cerebrovascular accidents or hypertensive encephalopathy can occur in pregnancy at lower blood pressure levels (60). Women with chronic hypertension and preeclampsia have an increased risk of cerebral complications or stroke during pregnancy even without excessive elevations in blood pressure (61). This may be explained by the fact that cerebral autoregulation is impaired in pregnant women with chronic hypertension or preeclampsia (62).

Antihypertensive treatment should be initiated expeditiously for acute-onset, severe hypertension (systolic blood pressure of 160 mm Hg or more or diastolic blood pressure of 110 mm Hg or more, or both) that is confirmed as persistent (15 minutes or more) (See Boxes 4, 5, and 6). The available literature suggests that antihypertensive agents should be administered within 30–60 minutes. However, it is recommended to administer antihypertensive therapy as soon as reasonably possible after the criteria for acute-onset severe hypertension are met (63–66). It is not known how quickly one should lower blood pressure in an acute setting. Mindful that blood pressure control should be expeditious and occur as safely as possible, evidence suggests that therapy should be initiated within 60 minutes and this goal is emerging as an important quality metric in obstetrics (65, 66). If blood pressure remains elevated after initiating antihypertensive therapy or for initial blood pressure elevations of 180 mm Hg or more systolic or 120 mm Hg or more diastolic, this represents further and ongoing risk of severe maternal morbidity and mortality and requires accelerated maternal and obstetric evaluation and decision making by the obstetric care provider. Evidence of acute end-organ damage, usually at blood pressure values exceeding 240/140 mm Hg, identifies the patient as having a genuine hypertensive emergency, and management in an intensive care unit with evaluation by a maternal–fetal medicine or critical care subspecialist, or both is advisable.

A thorough history and physical examination are essential to detect occult, secondary forms of hypertension; however, in a hypertensive crisis, drug therapy is frequently initiated before a final diagnosis is established. Laboratory tests, including serum electrolytes, serum creatinine, complete blood count with platelets, aspartate aminotransferase and alanine aminotransferase, urinalysis, and urine protein-to-creatinine ratio, should be promptly obtained as appropriate to help define both the hypertensive mechanism (superimposed preeclampsia or worsening chronic hypertension) and the extent of end-organ damage. Serum uric acid may have value in the management of specific patients where there is diagnostic uncertainty. Toxicology screen or electrocardiogram, or both may be appropriate in certain clinical situations.

It must be stressed that acute hypertensive episodes in pregnancy typically are approached more seriously than those in nonpregnant patients given the continuum of risk between incremental increases in maternal blood pressure and perinatal morbidity and mortality (67). In cases of acute-onset, severe hypertension, a structured and sequential antihypertensive approach with careful attention to both maternal and fetal conditions is recommended to ensure the best overall outcome. Box 4, Box 5, and Box 6 outline uniform sample order sets for the use of immediate-release oral nifedipine, intravenous (IV) hydralazine, and IV labetalol for the initial management of acute-onset, severe hypertension in women who are pregnant or in the postpartum period. Monitoring of viable fetuses usually is recommended for evaluating maternal hemodynamics and the fetal response to lowering maternal blood pressures.

Overzealous correction of blood pressure can be harmful and can cause hypoperfusion of critical territories, such as the maternal brain and heart or the placenta (68). A drop in diastolic blood pressure below 80 mm Hg may cause fetal heart rate abnormalities because the uteroplacental circulation does not autoregulate blood flow. The influence of concomitant obstetric management tools should not be overlooked either. As an example, epidural anesthesia can by itself lower blood pressure by approximately 15%, frequently minimizing the need for antihypertensive medication when used for typical obstetric indications of pain control (69). However, the effect of magnesium sulfate IV infusion on hypertension is modest if it occurs at all.

A systematic review of 35 randomized trials (3,573 women) examining different agents for acute antihypertensive therapy for severe-range blood pressure (ie, a systolic blood pressure of 160 mm Hg or more and a diastolic blood pressure of 110 mm Hg or more) during pregnancy showed insufficient evidence to conclude that one agent was more effective (50). A randomized trial comparing IV hydralazine to oral nifedipine showed equivalence of the two agents in the time to achieve blood pressure targets (70). No specific recommendations are available regarding the choice of antihypertensive agent based on the suspected hypertensive mechanism (eg, superimposed preeclampsia or worsening chronic hypertension), and the following are the choices of therapeutic modalities for rapid parenteral

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Box 4. Sample Order Set for Severe Intrapartum or Postpartum Hypertension Initial First-line Management With Immediate-Release Oral Nifedipine*†

- Notify physician if systolic blood pressure (BP) is greater than or equal to 160 mm Hg or if diastolic BP is greater than or equal to 110 mm Hg.
- Institute fetal surveillance if undelivered and fetus is viable.
- If severe BP elevations persist for 15 minutes or more, administer immediate-release nifedipine capsules (10 mg orally).‡
- Repeat BP measurement in 20 minutes and record results.
- If either BP threshold is still exceeded, administer immediate-release nifedipine capsules (20 mg orally). If BP is below threshold, continue to monitor BP closely.
- Repeat BP measurement in 20 minutes and record results.
- If either BP threshold is still exceeded, administer nifedipine immediate release capsule (20 mg orally). If BP is below threshold, continue to monitor BP closely.
- Repeat BP measurement in 20 minutes and record results.
- If either BP threshold is still exceeded, administer labetalol (20 mg intravenously for more than 2 minutes) and obtain emergency consultation from maternal–fetal medicine, internal medicine, anesthesia, or critical care subspecialists.
- Give additional antihypertensive medication per specific order.
- Once the aforementioned BP thresholds are achieved, repeat BP measurement every 10 minutes for 1 hour, then every 15 minutes for 1 hour, then every 30 minutes for 1 hour, and then every hour for 4 hours.
- Institute additional BP timing per specific order.

*Please note there may be adverse effects and contraindications.

†When used with magnesium sulfate, facilities should monitor maternal vital signs as described above in reference to blood pressure, with attention to normal heart rate and blood pressure.

‡Capsules should be administered orally and not punctured or otherwise administered sublingually.

Data from Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, National Heart, Lung, and Blood Institute, National High Blood Pressure Education Program Coordinating Committee. Hypertension 2003;42:1206–52; Vermillion ST, Scardo JA, Newman RB, Chauhan SP. A randomized, double-blind trial of oral nifedipine and intravenous labetalol in hypertensive emergencies of pregnancy. Am J Obstet Gynecol 1999;181:858–61; Raheem IA, Saaid R, Omar SZ, Tan PC. Oral nifedipine versus intravenous labetalol for acute blood pressure control in hypertensive emergencies of pregnancy: a randomised trial. BJOG 2012;119:78–85; Shekhar S, Sharma C, Thakur S, Verma S. Oral nifedipine or intravenous labetalol for hypertensive emergency in pregnancy: a randomized controlled trial. Obstet Gynecol 2013;122:1057–63; *and* Duley L, Meher S, Jones L. Drugs for treatment of very high blood pressure during pregnancy. Cochrane Database of Systematic Reviews 2013, Issue 7. Art. No.: CD001449. Available at https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD001449.pub2/media/CDSR/CD001449/CD001449_standard.pdf.

correction of acute, severe hypertension in pregnancy irrespective of etiology.

For acute blood pressure treatment, the agents listed below and in Table 3 can be considered alone or in sequence (as specified in Box 4, Box 5, and Box 6). If a maximum cumulative dose is achieved with any agent, it is recommended to switch to another class of agent.

Labetalol

Labetalol is a mixed α-adrenergic and β-adrenergic blocker and the most common β-blocker used in pregnancy. To avoid the risk of precipitous fall in blood pressure, the initial bolus should not be larger than 20 mg because there is a wide spectrum of individual dose response that is unpredictable based on clinical characteristics (71). If there is no response to the first bolus of 10–20 mg, incremental repeat doses (such as 20 mg, 40 mg, 80 mg, 80 mg, and 80 mg) may be administered every 10–30 minutes up to a maximum cumulative dosage of 300 mg. The onset of action after IV administration of labetalol occurs rapidly (within 5 minutes) with peak effect at 10–20 minutes and a total duration of action up to 6 hours. Labetalol can be administered as a continuous infusion (1 mg/kg), but the bolus IV administration is more frequently used. Labetalol should be avoided in women with preexisting

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Box 5. Sample Order Set for Severe Intrapartum or Postpartum Hypertension Initial First-line Management With Hydralazine*

- Notify physician if systolic BP is 160 mm Hg or more or if diastolic BP is 110 mm Hg or more.
- Institute fetal surveillance if undelivered and fetus is viable.
- If severe BP elevations persist for 15 minutes or more, administer hydralazine (5 mg or 10 mg IV for more than 2 minutes).
- Repeat BP measurement in 20 minutes and record results.
- If either BP threshold is still exceeded, administer hydralazine (10 mg IV for more than 2 minutes). If BP is below threshold, continue to monitor BP closely.
- Repeat BP measurement in 20 minutes and record results.
- If either BP threshold is still exceeded, administer labetalol (20 mg IV for more than 2 minutes). If BP is below threshold, continue to monitor BP closely.
- Repeat BP measurement in 10 minutes and record results.
- If either BP threshold is still exceeded, administer labetalol (40 mg IV for more than 2 minutes) and obtain emergency consultation from maternal–fetal medicine, internal medicine, anesthesia, or critical care subspecialists.
- Give additional antihypertensive medication per specific order.
- Once the aforementioned BP thresholds are achieved, repeat BP measurement every 10 minutes for 1 hour, then every 15 minutes for 1 hour, then every 30 minutes for 1 hour, and then every hour for 4 hours.
- Institute additional BP timing per specific order.

Abbreviations: BP, blood pressure; IV, intravenously.

*Please note there may be adverse effects and contraindications.

Data from Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, National Heart, Lung, and Blood Institute, National High Blood Pressure Education Program Coordinating Committee. Hypertension 2003;42:1206–52.

myocardial disease, decompensated cardiac function, and heart block and bradycardia. Labetalol also should be avoided in individuals with asthma as it can precipitate bronchospasm.

Hydralazine

Hydralazine has been used extensively for severe hypertension in pregnancy for more than 65 years. The onset of action is relatively slow for an IV drug (10–20 minutes) because it must be metabolized after attachment to the vessel wall in order to be effective. Common limiting adverse effects, which are experienced by up to 50% of recipients, include reflex tachycardia, hypotension, headaches, palpitations, flushing, anxiety, tremors, vomiting, epigastric pain, and fluid retention by activation of the renin-angiotensin system. Many of these adverse effects resemble the signs and symptoms of severe preeclampsia, confusing the clinical picture. The hypotensive overshoot that may be associated with hydralazine use may affect uteroplacental blood flow or maternal renal blood flow, which may lead to oliguria, particularly in pregnant women who are volume depleted. Late decelerations that occur after the administration of hydralazine often respond to fluid loading and position changes, but cases with placental abruption or fetal distress requiring emergency cesarean delivery have been reported. Whenever a powerful antihypertensive is administered, prior correction of hypovolemia may be helpful to prevent the hypotensive overshoot.

The hypotensive overshoot with hydralazine is an unpredictable complication that is not always dose related. Because hydralazine has a long duration of action, it can last up to 12 hours (72). Fetal distress secondary to maternal hypotension seems to be more frequent when a continuous infusion of hydralazine is used instead of repeat bolus administrations (73). Furthermore, in a comparative study of hydralazine bolus injection compared with continuous infusion, bolus injections achieved the therapeutic goal significantly

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Box 6. Sample Order Set for Severe Intrapartum or Postpartum Hypertension, Initial First-line Management With Labetalol*

- Notify physician if systolic BP measurement 160 mm Hg or more or if diastolic BP measurement is 110 mm Hg or more.
- Institute fetal surveillance if undelivered and fetus is viable.
- If severe BP elevations persist for 15 minutes or more, administer labetalol (20 mg IV for more than 2 minutes).
- Repeat BP measurement in 10 minutes and record results.
- If either BP threshold is still exceeded, administer labetalol (40 mg IV for more than 2 minutes). If BP is below threshold, continue to monitor BP closely.
- Repeat BP measurement in 10 minutes and record results.
- If either BP threshold is still exceeded, administer labetalol (80 mg IV for more than 2 minutes). If BP is below threshold, continue to monitor BP closely.
- Repeat BP measurement in 10 minutes and record results.
- If either BP threshold is still exceeded, administer hydralazine (10 mg IV for more than 2 minutes). If BP is below threshold, continue to monitor BP closely.
- Repeat BP measurement in 20 minutes and record results.
- If either BP threshold is still exceeded, obtain emergency consultation from maternal–fetal medicine, internal medicine, anesthesia, or critical care subspecialists.
- Give additional antihypertensive medication per specific order.
- Once the aforementioned BP thresholds are achieved, repeat BP measurement every 10 minutes for 1 hour, then every 15 minutes for 1 hour, then every 30 minutes for 1 hour, and then every hour for 4 hours.
- Institute additional BP timing per specific order.

Abbreviations: BP, blood pressure; IV, intravenously.

*Please note there may be adverse effects and contraindications.

Data from Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, National Heart, Lung, and Blood Institute, National High Blood Pressure Education Program Coordinating Committee. Hypertension 2003;42:1206–52.

faster and with smaller total doses (74). Therefore, intermittent bolus is recommended over continuous IV infusion. Repeat IV bolus administration is recommended as 5–10 mg injections every 20–40 minutes to a maximum cumulative dose of 20 mg until blood pressure control is achieved.

In a meta-analysis, hydralazine was more effective than labetalol in lowering severe blood pressure in pregnancy but was associated with more adverse maternal and perinatal events (eg, maternal tachycardia, prolonged hypotension, oliguria, cesarean delivery, placental abruption, nonreassuring fetal heart tracing, and low Apgar scores) (75).

Nifedipine

Immediate-release oral nifedipine, a calcium channel blocker, is an acceptable first-line alternative to hydralazine or labetalol, especially in cases of patients without IV access (50, 76–78). Nifedipine has been associated with an increase in maternal heart rate and has less risk of overshoot hypotension (76). Immediate-release nifedipine should not be given sublingually given the risk of hypotension with this route of administration. One study assessing the feasibility of using extended-release nifedipine tablets instead of immediate-release capsules for the acute lowering of severe hypertension in pregnancy (79) found that absorption for tablets peaked at 60–70 minutes compared with 30 minutes for capsules; moreover, it took 45–90 minutes to achieve adequate lowering of blood pressure, making the performance of nifedipine tablets unacceptable in acute situations.

In the rare circumstance that these first-line agents, used as directed, fail to relieve acute-onset, severe hypertension, emergent consultation with an anesthesiologist, maternal–fetal medicine subspecialist, or critical

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care subspecialist to discuss further interventions is recommended (66). Second-line agents to consider in such emergencies include nicardipine or esmolol by infusion pump or IV enalapril.

Once blood pressure control has been achieved with the acute therapeutic agent, it is important to anticipate the need for further treatment and to initiate oral maintenance. Failure to do so may expose the patient to a recurrent acute situation.

▶ ***What is the role for low-dose aspirin in patients with chronic hypertension in pregnancy?***

Evidence suggesting that an imbalance in prostacyclin and thromboxane A_2 metabolism is involved in the development of preeclampsia prompted interest in the study of aspirin for preeclampsia prevention because of its preferential inhibition of thromboxane at lower doses. Low doses of aspirin also inhibit platelet aggregation (80). The 2014 U.S. Preventive Services Task Force guidelines on low-dose aspirin for the prevention of preeclampsia concluded with moderate certainty that there are benefits with this intervention in women at high risk of preeclampsia without evidence of adverse effects (81).

The recent American College of Obstetricians and Gynecologists' recommendations for women at high risk of preeclampsia are to start treatment with 81 mg daily between 12 weeks and 28 weeks of gestation and to continue until delivery (82). Thus, for women with chronic hypertension, it is recommended to initiate daily low-dose aspirin (81 mg) between 12 weeks and 28 weeks of gestation (optimally before 16 weeks) and to continue this therapy until delivery. Initiating treatment after 28 weeks of gestation is unlikely to be beneficial. Low-dose aspirin should not be used in women with risk factors for gastrointestinal hemorrhage (eg, bleeding disorders or peptic ulcer).

▶ ***Is there a role for fetal surveillance in pregnancies complicated by chronic hypertension?***

Antenatal fetal surveillance may be beneficial in reducing perinatal morbidity and mortality in high-risk pregnancies (83). Therefore, antenatal fetal testing is recommended for women with chronic hypertension complicated by issues such as the need for medication, other underlying medical conditions that affect fetal outcome, any evidence of fetal growth restriction, or superimposed preeclampsia. However, limited data exist regarding the timing and interval of testing. Furthermore, in the absence of randomized trials comparing testing to no testing, it remains unclear whether antenatal fetal testing translates into outcome improvements.

The risks of fetal growth restriction in patients with chronic hypertension warrant third-trimester ultrasound assessment of fetal growth, with subsequent evaluation as appropriate (84). This may be justified because the risk of fetal growth restriction is higher in women with chronic hypertension and the sensitivity of fundal height measurements to detect fetal growth restriction is inadequate for high-risk women including women with hypertension or obesity (85).

The performance of electronic fetal monitoring in labor has been tested predominantly in low-risk populations, and these studies have largely excluded patients at high risk of adverse outcomes. It is recommended that patients with high-risk conditions be monitored with continuous fetal heart rate monitoring in labor (86); thus, patients with chronic hypertension at higher risk of adverse outcomes (eg, those who require any blood pressure control or with other short-term or long-term sequelae), at a minimum, should have continuous fetal monitoring.

▶ ***Are there intrapartum concerns unique to pregnant women with chronic hypertension?***

Women with chronic hypertension who are admitted for delivery should continue their medications. Blood pressure should be monitored at regular intervals and acute elevations should be addressed accounting for transient elevations that may occur in the setting of labor. Women with severe hypertension complicated by cardiovascular or renal disease may present with special problems peripartum and should be collaboratively managed with subspecialists (cardiology, nephrology, maternal–fetal medicine) as appropriate. Assessment of urine for proteinuria should be considered, as the diagnosis of preeclampsia in a patient with chronic hypertension could have important management implications. Urine output monitoring may be appropriate in populations that are susceptible to fluid overload with resultant pulmonary edema.

Results of a retrospective cohort study using data from the Consortium on Safe Labor (which represents 19 hospitals in the United States) seem to indicate that women with chronic hypertension and superimposed preeclampsia have longer first stages of labor, particularly nulliparous patients (87). Allowing a longer first stage of labor in these women may avoid unnecessary cesarean deliveries.

Issues related to analgesia or anesthesia in pregnant women with chronic hypertension have not been studied extensively. Although women with hypertension are more vulnerable to sympathetic blockade, it appears that axial anesthesia is safe even in cases with severe hypertension in labor, and that it has no adverse effects on neonatal outcomes or maternal condition (88). Whenever possible, unless contraindicated by maternal thrombocytopenia, neuraxial anesthesia should be employed

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because general anesthesia may pose a higher risk for pregnant women with severe hypertension. Intubation and extubation may be associated with acute elevations in blood pressure. In these situations, administration of IV antihypertensive agents, such as β-blockers, may be necessary. When possible, severe hypertension should be corrected before intubation to avoid health risks of further increases in acute blood pressure.

▶ ***How is chronic hypertension distinguished from superimposed preeclampsia?***

It often is difficult to distinguish chronic hypertension from superimposed preeclampsia when a patient with chronic hypertension presents with elevated blood pressure later in pregnancy. The clinical work-up should screen for symptoms associated with preeclampsia, and a comprehensive laboratory evaluation may be useful in making a diagnosis. New-onset proteinuria meeting criteria for preeclampsia, for instance, makes the diagnosis simple. Performing a baseline work-up for patients with chronic hypertension at the beginning of pregnancy is important because it can provide a context for comparison. However, in women with chronic hypertension and chronic renal end-organ impairment antedating pregnancy, it may not be possible to distinguish between the two entities. Acute, severe, and persistent elevations in blood pressure, in the absence of other explanations, such as cocaine intoxication, may suggest superimposed preeclampsia. Less commonly, a woman presenting with hypertension late in pregnancy could have systemic lupus erythematosus, primary renal disease, or hyperthyroidism. Although it is not necessary to screen all women with hypertension for these conditions, the possibility should be considered in the clinical context of unclear and severe presentations.

In cases of diagnostic uncertainty in discriminating transient blood pressure increases in chronic hypertension from superimposed preeclampsia, particularly with severe-range blood pressures, initial surveillance in the hospital setting is recommended. Work-up should include evaluation of hematocrit, platelets, creatinine, and liver function tests as well as assessment of new-onset proteinuria. Serum uric acid may be a helpful marker. Elevated hematocrit (indicating hemoconcentration), thrombocytopenia, hyperuricemia, new-onset or worsening proteinuria, elevated serum creatinine, and elevated liver transaminases are more indicative of preeclampsia than chronic hypertension, and, from a practical point of view, the practitioner should think preeclampsia first. Fetal well-being should be assessed as appropriate with fetal heart rate monitoring and sonography. Often, serial blood pressure assessment during 4–8 hours can be helpful in discriminating acute and serious increases in blood pressure from transient hypertension. The assessment for superimposed preeclampsia typically takes a few hours but sometimes requires longer, perhaps up to 48 hours, in challenging cases of patients with comorbidities or end-organ damage that antedate pregnancy.

▶ ***When is delivery of a woman with chronic hypertension indicated?***

There is a paucity of reliable clinical trial data to guide decisions about the timing of delivery in women with chronic hypertension. A small clinical trial has suggested that the risk of adverse perinatal outcomes in women with chronic hypertension without any obstetric complications is similar to the risk among women without chronic hypertension (89). Moreover, a cohort study of women with chronic hypertension found that delivery at 38 0/7–39 6/7 weeks of gestation was optimal for balancing fetal and neonatal risks (90). Other publications also have endorsed delivery from 38 0/7 weeks to 39 6/7 weeks of gestation for women with chronic hypertension who are not prescribed medication or from 37 0/7 weeks to 39 6/7 weeks of gestation for women whose chronic hypertension is well-controlled with medication (91).

Based on this, the earlier gestational age in the suggested range for timing of delivery for women at term depends on the use of maintenance antihypertensive medications. For women with chronic hypertension and with no additional maternal or fetal complications supporting earlier delivery, if not prescribed maintenance antihypertensive medications, delivery before 38 0/7 weeks of gestation is not recommended. For women with chronic hypertension and with no additional maternal or fetal complications supporting earlier delivery, if prescribed maintenance antihypertensive medications, delivery before 37 0/7 weeks of gestation is not recommended. Patients with hypertension that is difficult to control, such as those requiring frequent maintenance medication adjustments or those on maximal doses of maintenance medications, may require earlier delivery, such as in the late preterm period.

The data regarding the later gestational age in the suggested range for timing of delivery is mixed. As previously described, expectant management up to 39 6/7 weeks of gestation may be considered. However, a retrospective cohort study of 683 women found that planned delivery before 39 0/7 weeks of gestation was associated with a decreased risk of preeclampsia with severe features. When compared with patients with a planned delivery at 39 weeks of gestation or more, those delivered before 39 0/7 weeks of gestation had a lower rate of preeclampsia with severe features (10% versus 1% absolute risk) (AOR for severe preeclampsia

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of 0.07; 95% CI, 0.01–0.5) (92). Additionally, a recent retrospective population-based study looked at the practice of routine induction at 38 weeks or 39 weeks of gestation, suggesting that induction of labor at 38 weeks or 39 weeks of gestation may prevent severe hypertensive complications (superimposed preeclampsia and eclampsia) without increasing the risk of cesarean delivery when compared with expectant management (93). Therefore, expectant management beyond 39 0/7 weeks of gestation should only be done after careful consideration of the risks and benefits and with appropriate surveillance.

Delivery timing in preterm pregnancies involves careful consideration of risks and benefits for a woman and fetus. Expectant management before term helps the fetus but risks the health of the woman. Women with severe acute hypertension that is not controlled with traditional chronic antihypertensive regimens or women who develop superimposed preeclampsia with severe features should be delivered upon diagnosis at 34 0/7 weeks of gestation or more. Because of the significant maternal–fetal and maternal–neonatal morbidity, immediate delivery after maternal stabilization is recommended if any of the following are present at any gestational age in women with superimposed preeclampsia: uncontrollable severe hypertension, eclampsia, pulmonary edema, disseminated intravascular coagulation, new or increasing renal insufficiency, placental abruption, or abnormal fetal testing. Women who develop superimposed preeclampsia with severe features before 34 0/7 weeks of gestation may be candidates for expectant management under certain circumstances, although expectant management is not recommended beyond 34 0/7 weeks of gestation. In these cases, inpatient management is recommended and should be undertaken only at facilities with adequate maternal and neonatal intensive care resources. Initiation of antenatal steroids is recommended according to American College of Obstetricians and Gynecologists' guidelines. Even in cases of preterm pregnancies with anticipated imminent delivery, neonates may benefit from exposure to a first dose. A retrospective case series examining a subset of 29 women with superimposed preeclampsia managed expectantly (36) found latency periods similar to women with severe preeclampsia in the absence of chronic hypertension (8.4 days compared with 8.5 days), with no differences in the rates of placental abruption, oliguria, or hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome between the two groups.

In women with superimposed preeclampsia without any severe features and with stable maternal and fetal conditions, expectant management until 37 0/7 weeks of gestation with close maternal and fetal surveillance is suggested. Induction at term for women with mild pregnancy-related hypertensive disease has not been associated with an increased risk of cesarean delivery (94). Given the lack of clinical trials and prospective studies in women with chronic hypertension and superimposed preeclampsia, the aforementioned recommendations for timing of delivery are based on indirect evidence from the management of preeclampsia consistent with published expert opinions (95).

There is a paucity of data to support outpatient management of superimposed preeclampsia. Reasonable and cautious practice would indicate that outpatient management be considered for cases without any severe features (eg, severe-range blood pressures, symptoms, laboratory abnormalities).

▶ *What are the postpartum considerations and recommendations in patients with chronic hypertension?*

Blood pressure control usually continues to be an issue postpartum, and even women who were not treated during pregnancy may require treatment with antihypertensive medication in the postpartum period. This elevation of blood pressure is considered to be related to the mobilization of extravascular fluid with rise in intravascular volume in addition to factors such as pain or anxiety.

After an initial decline immediately after delivery, blood pressure tends to rise. Therefore, a rapid decrease in blood pressure postpartum is more likely to indicate substantial blood loss than a rapid resolution of the disease process. Blood pressure in the postpartum period is often higher compared with antepartum levels, particularly in the first 1–2 weeks postpartum (96). Severe hypertension or superimposed preeclampsia also may develop for the first time in the postpartum period; therefore, women with chronic hypertension should be closely monitored for blood pressure changes and symptoms of severe-range hypertension or superimposed preeclampsia. Early ambulatory visits in the first 1–2 weeks after delivery or home blood pressure monitoring may be prudent surveillance for these postpartum changes (97). Medication in the weeks postpartum should be adjusted to maintain a systolic blood pressure not higher than 150 mm Hg and a diastolic blood pressure not higher than 100 mm Hg (98–100).

Some common medications and substances used in the postpartum period may potentially aggravate hypertension through three major mechanisms: 1) volume retention, 2) sympathomimetic activation, and 3) direct vasoconstriction. Of particular interest are the nonsteroidal anti-inflammatory drugs (NSAIDs) which are

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frequently prescribed as postpartum analgesics. These medications decrease prostaglandins leading to a lack of vasodilation and increased sodium retention. Nonsteroidal antiinflammatory medications should continue to be used preferentially over opioid analgesics; however, women with chronic hypertension theoretically may require intensification of blood pressure monitoring and regimen adjustments when on these medications. Overall, data support the safe use of NSAIDs in postpartum patients with blood pressure issues. In a randomized trial comparing use of ibuprofen to acetaminophen in postpartum patients with preeclampsia with severe features, ibuprofen did not lengthen the duration of severe-range blood pressures (101). In a cohort of 399 patients with preeclampsia with severe features, there was no association of NSAID use with postpartum blood pressure elevations (102). Further, another cohort study of postpartum patients on magnesium for seizure prophylaxis for preeclampsia did not show differences in blood pressure, antihypertensive requirements, or other adverse events for patients managed with NSAIDs in the postpartum period (103).

Antihypertensive medications may be used more liberally in the postpartum period than during pregnancy. Blood pressure parameters to guide medication dosage in the postpartum period should be adjusted given that there are no longer fetal considerations. Therefore, the goal should be a lower blood pressure range. Methyldopa, however, should be avoided because it often can be associated with depression, and the postpartum period is already characterized by increased vulnerability for depression (104). A recent, small (n=50) randomized controlled trial comparing labetalol to extended-release nifedipine in women with persistent postpartum hypertension not previously on medication found that both agents were effective; however, labetalol had fewer adverse effects and achieved control more often with the starting dose (105). Patients with chronic hypertension also may be returned to their prepregnancy regimen in collaboration with their primary care provider or internal medicine provider.

Antihypertensives, in general, can be used in breastfeeding women. Most antihypertensive medications are detectable, albeit at low concentrations, in breast milk thus their use during lactation is not contraindicated (106). Some β-blockers (eg, atenolol and metoprolol) are concentrated in breast milk resulting in higher levels, whereas propranolol and labetalol with their higher plasma protein binding are not concentrated in breast milk and remain at low levels. Thus, propranolol and labetalol are preferred for treatment in breastfeeding women. Angiotensin-converting enzyme inhibitors (eg, enalapril and captopril) concentrations in breast milk are low, and these drugs may be used safely during breastfeeding unless high doses are required. No adverse effects are known to occur with calcium channel blockers during breastfeeding. Although the concentration of diuretics in breast milk is low, these agents may reduce the quantity of milk production.

Clinical Considerations and Recommendations

The following recommendation is based on good and consistent scientific evidence (Level A):

- For women with chronic hypertension, it is recommended to initiate daily low-dose aspirin (81 mg) between 12 weeks and 28 weeks of gestation (optimally before 16 weeks) and to continue this therapy until delivery.

The following recommendations are based on limited or inconsistent scientific evidence (Level B):

- Initiation of antihypertensive therapy is recommended for persistent chronic hypertension when systolic pressure is 160 mm Hg or more, diastolic pressure is 110 mm Hg or more, or both. In the setting of comorbidities or underlying impaired renal function, treating at lower blood pressure thresholds may be appropriate.
- For the long-term treatment of pregnant women who require pharmacologic therapy, labetalol or nifedipine are reasonable options and are recommended above all other antihypertensive drugs. The use of angiotensin-converting-enzyme inhibitors, angiotensin receptor blockers, renin inhibitors, and mineralocorticoid receptor antagonists is generally not recommended.
- Antihypertensive treatment should be initiated expeditiously for acute-onset severe hypertension (systolic blood pressure of 160 mm Hg or more or diastolic blood pressure of 110 mm Hg or more, or both) that is confirmed as persistent (15 minutes or more). The available literature suggests that antihypertensive agents should be administered within 30–60 minutes. However, it is recommended to administer antihypertensive therapy as soon as reasonably possible after the criteria for acute-onset severe hypertension are met.
- For women with chronic hypertension and with no additional maternal or fetal complications supporting earlier delivery,

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• if not prescribed maintenance antihypertensive medications, delivery before 38 0/7 weeks of gestation is not recommended.

• if prescribed maintenance antihypertensive medications, delivery before 37 0/7 weeks of gestation is not recommended.

▶ Women with severe acute hypertension that is not controlled with traditional chronic antihypertensive regimens or women who develop superimposed preeclampsia with severe features should be delivered upon diagnosis at 34 0/7 weeks of gestation or more. Because of the significant maternal–fetal and maternal–neonatal morbidity, immediate delivery after maternal stabilization is recommended if any of the following are present at any gestational age in women with superimposed preeclampsia: uncontrollable severe hypertension, eclampsia, pulmonary edema, disseminated intravascular coagulation, new or increasing renal insufficiency, placental abruption, or abnormal fetal testing.

▶ Women who develop superimposed preeclampsia with severe features before 34 0/7 weeks of gestation may be candidates for expectant management under certain circumstances, although expectant management is not recommended beyond 34 0/7 weeks of gestation. In these cases, inpatient management is recommended and should be undertaken only at facilities with adequate maternal and neonatal intensive care resources.

The following recommendations are based primarily on consensus and expert opinion (Level C):

▶ A woman with chronic hypertension should be evaluated prepregnancy to identify possible end-organ involvement, to consider evaluation for secondary hypertension, and for the optimization of maternal comorbidities (eg, obesity, diabetes) before pregnancy.

▶ It is recommended to maintain blood pressure levels for pregnant women with chronic hypertension treated with antihypertensive medications at or above 120 mm Hg but below 160 mm Hg systolic and at or above 80 mm Hg but below 110 mm Hg diastolic.

▶ Antenatal fetal testing is recommended for women with chronic hypertension complicated by issues such as the need for medication, other underlying medical conditions that affect fetal outcome, any evidence of fetal growth restriction, or superimposed preeclampsia.

▶ The risks of fetal growth restriction in patients with chronic hypertension warrant third-trimester ultrasound assessment of fetal growth, with subsequent evaluation as appropriate.

▶ In cases of diagnostic uncertainty in discriminating transient blood pressure increases in chronic hypertension from superimposed preeclampsia, particularly with severe-range blood pressures, initial surveillance in the hospital setting is recommended. Work-up should include evaluation of hematocrit, platelets, creatinine, and liver function tests as well as assessment of new-onset proteinuria. Serum uric acid may be a helpful marker. Elevated hematocrit (indicating hemoconcentration), thrombocytopenia, hyperuricemia, new-onset or worsening proteinuria, elevated serum creatinine, and elevated liver transaminases are more indicative of preeclampsia than chronic hypertension, and, from a practical point of view, the practitioner should think preeclampsia first. Fetal well-being should be assessed as appropriate with fetal heart rate monitoring and sonography. Often, serial blood pressure assessment during 4–8 hours can be helpful in discriminating acute and serious increases in blood pressure from transient hypertension.

▶ In women with superimposed preeclampsia without severe features and with stable maternal and fetal conditions, expectant management until 37 0/7 weeks of gestation with close maternal and fetal surveillance is suggested.

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The MEDLINE database, the Cochrane Library, and the American College of Obstetricians and Gynecologists' own internal resources and documents were used to conduct a literature search to locate relevant articles published between January 2000–July 2018. The search was restricted to articles published in the English language. Priority was given to articles reporting results of original research, although review articles and commentaries also were consulted. Abstracts of research presented at symposia and scientific conferences were not considered adequate for inclusion in this document. Guidelines published by organizations or institutions such as the National Institutes of Health and the American College of Obstetricians and Gynecologists were reviewed, and additional studies were located by reviewing bibliographies of identified articles. When reliable research was not available, expert opinions from obstetrician–gynecologists were used.

Studies were reviewed and evaluated for quality according to the method outlined by the U.S. Preventive Services Task Force:

I Evidence obtained from at least one properly designed randomized controlled trial.

II-1 Evidence obtained from well-designed controlled trials without randomization.

II-2 Evidence obtained from well-designed cohort or case–control analytic studies, preferably from more than one center or research group.

II-3 Evidence obtained from multiple time series with or without the intervention. Dramatic results in uncontrolled experiments also could be regarded as this type of evidence.

III Opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees.

Based on the highest level of evidence found in the data, recommendations are provided and graded according to the following categories:

Level A—Recommendations are based on good and consistent scientific evidence.

Level B—Recommendations are based on limited or inconsistent scientific evidence.

Level C—Recommendations are based primarily on consensus and expert opinion.

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